

## **PART VIII. DISEASES OF THE NERVOUS SYSTEM.**

### **I. DISEASES OF THE PERIPHERAL NERVES.**

#### **NEURITIS.**

**Definition.**—An inflammation of a nerve or its fibrous covering. Neuritis may be confined to a single nerve, or involve a number of nerves, and may therefore be considered under separate headings,—local, or focal neuritis, and multiple neuritis.

#### **LOCALIZED NEURITIS.**

**Synonym.**—Focal Neuritis.

**Etiology.**—Cold is a common cause, involving especially the facial and sciatic nerves, and gives rise to the so-called rheumatic neuritis.

Trauma is perhaps the most frequent cause and may be due to stretching or tearing that so often accompanies fractures and dislocations, or it may be due to severe muscular exertion, or to wounds, contusions, or hypodermic injections, or the continued strain on certain muscles, as seen in professional palsies.

The various toxins found in the infectious diseases may act upon a single nerve, though usually they produce multiple neuritis. A nerve may be involved by an extension of an inflammation from some neighboring part.

**Pathology.**—The inflammation may be confined to the peri-neurium, the nerve-sheath, or extend to the deeper tissues—interstitial—or involve the axis cylinder—parenchymatous. Where the sheath is involved, the nerve becomes swollen, hyperemic, and infiltrated with leukocytes.

Where the axis cylinder is affected—parenchymatous neuritis—degenerative changes take place, the nuclei of the nerve-cells consisting of oily-looking globules, a fatty degeneration.

**Symptoms.**—These depend somewhat upon the functions of the nerve involved. If a sensory nerve be the seat of the lesion, the pain is intense, and is of a burning, boring, aching, or shooting character. There is also tenderness on pressure, or when the muscles are moved. Although exquisitely painful, tactile sensation is materially lessened.

If a motor nerve be affected, there will be twitching of the muscles, supplied by the nerve, and if it assumes a chronic character, there will be paralysis and atrophy of the muscles.

Frequently both sensory and motor nerves are involved with a combination of symptoms.

Where the disease assumes a chronic form, trophic changes are manifest. There is a loss of faradic irritability, the skin becomes glossy and edematous, the nails become impaired, localized sweatings may arise, the surface temperature is sometimes increased, and there may be effusion into the joints.

**Diagnosis.**—We diagnose neuritis from neuralgia, the only lesion with which it might be mistaken, by the continuous character of the pain, which is increased by pressure. Altered sensation would also suggest neuritis.

**Prognosis.**—This is favorable in mild cases, the disease yielding within ten days or two weeks. If it assumes a chronic form, it may persist for months or years. If the continuity of the nerve be preserved, recovery will take place.

**Treatment.**—The part should have rest and support; where the pain is intense, a hypodermic of morphia may be required to relieve the pain. Hot applications generally afford more relief than cold ones. After tenderness disappears, much benefit will be derived from galvanism and massage.

When due to cold—rheumatic neuritis—the antirheumatics will be found efficient. Bryonia, apocynum, macrotys, rhamnus Californica, and the salicylates will be among the best.

If the skin is red, glossy, and edematous, apocynum is a good remedy.

Jaborandi sometimes gives marked relief.

## **MULTIPLE NEURITIS.**

**Synonyms.**—Polyneuritis; Peripheral Neuritis.

**Etiology.**—Insufficient and improper food, and exposure, are predisposing causes, and it is said to be more prevalent in females than in males.

Toxic agents are most frequently responsible for neuritis, alcohol heading the list, and while it occurs in persons using strong liquors, it may follow the use of malt drinks.

Of the metallic poisons, lead is the most frequent cause, phosphorus, arsenic, mercury, copper, and zinc following.

Of chemical poisons, bisulphide of carbon, coal gas, ether, naphtha, anilin, ergot, and morphia may be named.

Of the infectious diseases, diphtheria is the most frequent cause, typhoid fever, leprosy, scarlet fever, beri-beri, small-pox, influenza, and tuberculosis being less frequently the cause. Cachectic conditions, such as anemia, cancer, and tuberculosis may be responsible for it, and it sometimes follows rheumatism, gout, the puerperal state, and diabetes.

**Pathology.**—The pathology of multiple neuritis does not differ materially from that of neuritis occurring in isolated nerves. The lesion may be that of an interstitial neuritis, or of a parenchymatous neuritis, or belli combined. Generally, the peripheral parts suffer more than the central parts.

The motor nerves alone may be involved as in lead-poisoning, or the sensory nerves may alone be affected as in coal-gas poisoning, or both may be involved as in the neuritis due to alcohol and the infectious diseases.

Changes in the spinal cord, such as meningitis, may occur.

**Symptoms.**—Since there are so many phases of multiple neuritis, it

will simplify the description of their many phenomena by describing separately their principal forms.

*Acute Febrile Polyneuritis.*—The onset is sudden, and frequently resembles the acute infectious fevers. There is a chill followed by febrile reaction, the temperature rapidly rising to 103° or 104° ; there is some tenderness in the spleen, and not infrequently some slight jaundice. There is pain in the head, back, and limbs, and sometimes swelling of the articulations, resembling rheumatic fever. Tingling, numbness, and muscular cramps frequently precede loss of power in certain muscles. The extensors of the hands and feet are most commonly involved, giving rise to wrist and foot drop.

Paralysis rapidly extends up the extremities, and sometimes involves the trunk and face. Faradic irritability is lost, and the muscles rapidly waste. Death may occur in from seven to twenty-one days. In milder cases, after a few weeks of suffering, the patient gradually shows improvement, but a complete cure may not take place short of one or two years.

*Alcoholic Neuritis.*—This is the most common form of the disease, and occurs more frequently in women than in men. Its onset is usually slow, being preceded by impaired digestion, catarrhal gastritis, sleeplessness, irregular or feeble heart's action, and, as the patient expresses it, his feet and hands “go to sleep.” There is tenderness along the course of the nerve-trunks, cramp in the muscles, with loss of power and paralysis of the extensor muscles of the upper and lower extremities, giving rise to the characteristic wrist and foot drop. Neuralgic pains, sensory disturbances, delirium and convulsions not infrequently occur, and sometimes result in insanity. Loss of control of the sphincters of the bladder and rectum sometimes occur. Atrophy of the affected muscles usually takes place. Anesthesia of the skin and hyperesthesia of the muscles is a characteristic condition.

Owing to paralysis of the extensor muscles, the “stoppage gait” (lifting the foot high in the air and swinging the foot forward to avoid striking the toes on the ground) is a prominent symptom. There is loss of the superficial and deep reflexes. In some cases the patient passes into a low ataxic or typhoid condition.

*Lead Paralysis.*—This form is not attended by sensory disturbances and

is usually preceded by anemia, lead-colic, obstinate constipation, and a characteristic blue line on the margin of the gums. The paralysis usually develops gradually, though in exceptional cases the onset is sudden.

The muscles supplied by the musculo-spiral nerve are more frequently involved than those of the lower extremities; hence the characteristic wrist-droop in lead paralysis. When the lower limbs are involved, the peroneal muscles are the first to be attacked.

The prognosis is generally favorable, and results in recovery in from four to six months.

*Arsenical Paralysis.*—This form is more apt to affect the lower extremities, and atrophy of the muscles is more rapid than in lead paralysis: Formication and numbness are the chief sensory symptoms. The “stoppage gait” is generally well marked.

The condition of the reflexes and their behavior to electric stimulation are similar to those of lead-poisoning.

*Carbon-bisulfid Neuritis.*—In this form of neuritis there is intense pain in the head, dizziness, muscular cramps, and sometimes convulsions.

*Coal-gas neuritis* involves only the sensory nerves, is usually mild in character, though numbness may persist in the hands and feet for a long time.

*Beri-beri, Kakke, or Endemic Neuritis.*—This disease occurs. endemically in Japan, China, the Philippines, and Northern Brazil. Its etiology is in doubt, and though many regard it as being due to micro-organisms, its specific character has not yet been determined. Predisposing causes are such as impair the vitality and lower the quality of the blood. Foreigners visiting or residing in endemic localities are apt to be exempt.

**Symptoms.**—The first evidence of the disease is a sense of weariness or weakness in the lower extremities. Soon a fever develops, the patient grows anemic, and edema develops, which is followed by general anasarca. There is painful atrophy of the muscles, and paralysis of the arms and legs occur. Vomiting sometimes occurs and the urine is usually scanty though not albuminous. If the phrenic nerve be affected, paralysis of the diaphragm may result in death.

**Diagnosis.**—A well-marked case of neuritis is not usually readily mistaken, but since a multiple neuritis frequently simulates poliomyelitis anterior, locomotor ataxia, and diffuse myelitis, the differential diagnosis may be readily understood by examining the table used by Ranney, and found in his work on Diseases of the Nervous System.

**Prognosis.**—The prognosis is generally favorable, though much depends upon the cause or rather form of the disease. When due to alcoholism, diphtheria, or beri-beri, the diagnosis should be guarded, since these are grave forms of the disease.

**Treatment.**—The treatment depends upon the wrongs present, and does not differ materially from that of other diseased conditions. Where possible, the cause should be removed; if due to alcoholism, drink should be restricted, and if from lead, arsenic, bisulphid of carbon, etc., the patient should be removed from the exciting cause, and, as far as possible, endeavor to eliminate the poison. When due to infection, the antiseptics will be of much benefit.

Rest in bed, with support to affected parts, will afford much relief. When fever attends, the special sedatives are to be judiciously used. If the pain is intense, morphia hypodermically should be used, though it should not be continued indefinitely for fear of the morphine habit. When due to sepsis, echinacea, the sulphites, chlorates, and mineral acids will be called for. If due to absorption of lead, sulphur baths, and potassium iodid internally, and galvanism will give good results.

Rhus, bryonia, avena, rhamnus Cal., and melilotus will be useful agents under certain conditions.

The warm baths and massage are not to be overlooked.

## NEUROMATA.

**Definition.**—Tumors of nerves, which are divided into true and false neuromata.

True neuromata are made up of *nerve-fibers*, and, in rare cases, of

*ganglion cells.*

False neuromata are devoid of *nerve-tissue*, and are composed of fibrous, gliomatous, myxomatous, or sarcomatous tissue, and situated on or within the nerve-sheath.

**Etiology.**—Neuromata, when single, are generally due to traumatism, either surgical, as when nerves are divided, or accidental, as from puncture, as from the hypodermic needle, or other penetrating instruments.

When multiple, they are due to hereditary or some diathetic disease, as leprosy.

**Pathology.**—All neuromata are made up of a mass of nerve-tissue and are classified as—(1) Stump or bulbous neuromata, developing on the severed ends of nerves. (2) Subcutaneous neuromata, or “*tubercula dolorosa*,” painful nerve-tumors lying just beneath the skin. (3) Nerve-trunk neuromata, usually multiple. (4) Plexiform neuromata, consisting of an interlacing of neural cords, beady or tortuous in character.

**Symptoms.**—Neuromata, as a rule, are not painful, and rarely cause symptoms, if we except the subcutaneous, or those in an amputation stump. Occasionally motor symptoms are present, and are manifested by frequent or constant twitchings. That epilepsy may result from these growths is shown by a cessation of the attacks upon a removal of the neuromata.

**Treatment.**—This is surgical, and in the subcutaneous form gives permanent relief. In the bulbar form the relief may be only temporary, the growths frequently recurring.

In false neuromata, especially when due to syphilis, the iodids, echinacea, and other antisyphilitics will be indicated.

## NEURALGIA.

**Definition.**—Neuralgia is a term used to express a pain of varying character in the course of a nerve or its branches, in which there is no structural change. The pain is intermittent or remittent in character,

and tends to shift from place to place. There are frequently painful points (points douloureux) in the course of the nerve.

**Etiology.**—*Predisposing Causes.*—Neuralgia is a disease of middle life, being very rare in children or persons of extreme age, and occurs in men more frequently than in women, though females of a neurotic temperament are quite often affected. Heredity seems to have some influence in giving rise to neuralgia, it frequently occurring in hysterical and epileptic families; in fact, the general physical condition predisposes to neuralgia, and persons reduced by sickness, severe physical or mental exertion, anemia, and mal-nutrition, are prone to this affection. Workers in paints and metal-workers are also quite likely to attacks of neuralgia, while rheumatism, gout, chronic nephritis, diabetes, and the infectious fevers favor neuralgia. Exposure to cold and wet is a frequent exciting cause, while pressure from morbid growths or inflammation of near-by tissues are not to be overlooked as causal factors.

Reflex conditions must not be overlooked, for some of the most severe forms of neuralgia are due to wrongs of the uterus, ovaries, rectum, or urethra, or to disease of the teeth, nose, middle ear, sinuses, or antrum and to eyestrain.

**Symptoms.**—Pain is the most characteristic symptom, and is usually located in the course of a nerve. The attack may come on abruptly or be preceded by prodromal symptoms, such as chilly sensations, mental depression, uneasiness in the part, or a stinging, tingling, or burning sensation. When fully ushered in, the attack seems unbearable; the pain is lancinating, stabbing, burning, or boring in character, localized or darting to neighboring nerves.

The skin of the affected part may be quite sensitive, and painful spots can be detected., especially where the nerve becomes superficial. Sometimes the skin of the affected area becomes anesthetic, and may remain so for some little time after the attack ceases. Twitching or spasms of the muscles may occur during a paroxysm. Herpes often follows an attack.

Neuralgic paroxysms exhibit a marked tendency to periodicity. A paroxysm may last for a few minutes or for several hours.



*Clinical Varieties.*—*Trifacial* neuralgia (tic douloureux). In this form the pain is felt in one or more branches of the fifth nerve, more frequently the ophthalmic division.

The symptoms vary, from an occasional paroxysm of a mild type, to paroxysms of such excruciating intensity, and occurring so frequently, as to cause the patient to take his own life. Hyperesthesia of the skin of the affected part is common, and vaso-motor phenomena frequently are present, such as flushing, sweating, increased lachrymation, and nasal secretion and salivation.

The pain is of a rending or boring character, and may be so severe as to cause great prostration. Spasms of the muscles may occur. The painful points are: the supraorbital foramen, when the first branch is involved; the infraorbital foramen, when the second branch is affected; and when the third branch is the seat of the lesion, the mental foramen will be the painful spot.

When the pain is long continued, trophic changes, such as erythema, formation of ulcers, induration or drying of the skin, loss of hair or local grayness take place.

*Neuralgia of the Neck and Trunk.*—*Cervico-occipital Neuralgia.*—Exposure to cold, or cervical caries, is the most frequent cause of this form of neuralgia. The pain is localized over the occipital and posterior parietal regions, the most painful spot being located between the mastoid process and the upper cervical nerve, where the great occipital nerve becomes superficial. Hypresthesia of the scalp frequently occurs, and loss of hair is not uncommon.

*Phrenic Neuralgia.*—The pain is in the lower anterior thoracic region, at or near the insertion of the diaphragm. It is a rare condition.

*Intercostal Neuralgia.*—This is a common form of neuralgia, especially in anemic and hysterical women, the middle intercostal nerves of the left side suffering more frequently than the right. There is commonly a dull pain, with paroxysms of a stabbing character at intervals. Painful points are located under the angle of the scapula, beside the vertebra, and at the middle of the ribs.

Expansion of the chest increases the pain; hence respiration is shallow.

Where the attack is severe and persistent, herpes zoster may appear, though this is usually associated with neuritis.

*Neuralgia of the Extremities; Cervico-brachial Neuralgia.*— In this form the sensory nerves of the brachial plexes are involved, the radial and ulnar nerves being affected more frequently than the median.

The pain is most often located in the axilla, along the course of the ulnar nerve, and when the circumflex nerve is involved, in the deltoid muscle. Tender points are found in the axilla, in the posterior border of the deltoid, the superior ulnar behind the elbow, the inferior ulnar in front of wrist, and the musculo-spiral nerve at the bend of the elbow.

*Obturator neuralgia* is frequent in women suffering with uterine or ovarian lesions, the pain extending along the inner side of the thigh as low as the knee.

*Lumbar neuralgia* gives rise to pain in the lumbar region, along the crest of the ilium, in the inguinal and femoral regions, and in the spermatic cord, scrotum, or vulva. If severe and located in the testicles, it may be attended by syncope.

*Sciatica.*—Next to facial neuralgia, sciatica is the most common, and affects males more than females. The pain may be uniformly distributed along the entire length of the nerve, though more frequently the paroxysm is most severe in the gluteal and popliteal regions; other painful spots are the middle of the thigh, below the head of the fibula, behind the external malleolus and the back of the foot.

The pain may be constant, though, usually paroxysms of intense pain, of a shooting, stabbing, or boring character, occur at irregular intervals. Damp weather seems to aggravate the attacks.

The pain is increased by walking or motions of the limb. In walking the knee is flexed and the patient throws his weight upon his toes, to diminish the tension on the nerve. Tremors or spasm of the muscles may be present, and when the disease is long continued there may be atrophy, of the muscles.

*Neuralgia of the Genitalia and Rectum.*—Of all neuralgias, none are more severe or harder to bear than neuralgia of the rectum and

genitals. Coccygodynia is a common affection in women, and is usually associated with other nervous affections. It is aggravated by the sitting posture. This form is often very intractable, resection of the coccyx having been resorted to in some very severe cases, though, unfortunately, not always attended by relief.

*Visceral Neuralgia.*—In persons of nervous habits, and subject to neuralgias, it is not uncommon to find a sudden attack of severe pain in certain of the viscera, such as kidney, liver, stomach, bowel, and spleen, and is referred to as neuralgia of the kidney, liver, etc.

**Treatment.**—In the treatment of neuralgia, much depends upon our ability to remove the underlying cause, which may be local, general, or reflex. Thus when it is local, such as a neuroma, caries, aneurism, or cicatrix, we would not expect much benefit from medication, but surgical measures would effect a cure. Where the cause is general, our attention would be directed toward improving the general health by correcting septic processes, establishing the secretions, and improving nutrition. When due to reflex causes, a careful search must be instituted for the irritant part, a correction of which is followed by relief. It may be due to uterine, ovarian, rectal, or urethral lesions, and until this is overcome, but little relief can be obtained by the administration of medicines.

An illustration of this may be seen in one of the most intractable facial neuralgias I ever encountered. The patient, aged about seventy years, had suffered for eighteen months with the most intense pain, the paroxysms occurring every few minutes day and night. He had had heroic dosing and infinitesimal medication, but, worn in body and mind, was growing worse. My first examination of the patient revealed hemorrhoids, pockets, and redundant tissue of the rectum, and a bad stricture of the urethra; the correction of which gave the patient the first relief in eighteen months.

In the most severe paroxysms, the physician may have to resort to morphia hypodermically, though it should be used at long intervals, lest the morphine habit be established.

Remedies that influence neuralgic conditions are the following :

*Facial Neuralgia.*—Aconite, gelsemium, plantago, passiflora, piper methysticum, and chamomilla.

*Cervico-occipital and Cervico-brachial.*—Aconite, macrotys, rhamnus Californica, gelsemium, and sticta pulmonaria.

*Intercostal.*—Bryonia, asclepias, lobelia, and macrotys.

*Lumbar and Sciatic.*—Macrotys, rhamnus Californica, bryonia, collinsonia, sticta, apocynum, aesculus, quebracho, and the salicylates. Aritikamnia, phenacetin, and like remedies, should be used with care. Where the neuralgia shows periodicity, quinine should be administered. Counter-irritants will be found useful in many cases.

## **DISEASES OF THE CRANIAL NERVES.**

### **THE OLFACTORY NERVE.**

The olfactory nerve may be affected in its nasal origin, in the mucous membrane, by a disturbance at the bulb, at some point in the course of the tract, or at its origin in the brain. The result is an increased, diminished, or perverted function of the sense of smell.

*Hyperosmia* is an abnormally acute sense of smell, and is generally found in hysteria, neurasthenia, and insanity.

*Parosmia.*—Perversion of the sense of smell may be confined to one or many odors, and, like hyperosmia, occurs most frequently in neurasthenia and in the insane.

*Anosmia.*—Loss of the sense of smell, may be due to acute or chronic rhinitis, bone disease, or lesions of the brain.

**Treatment.**—The treatment depends entirely upon the cause. When due to organic changes, medication is unsatisfactory.

### **THE RETINA AND OPTIC NERVE.**

**Retina.**—Hemorrhage into the retina may arise from a number of causes, thus it is found in leukemia, the pernicious anemias, purpura, and scurvy, and is often the first evidence of chronic nephritis. It may

take place during parturition, though it most frequently occurs after the menopause. Vision is more or less impaired, and if the hemorrhage be superficial, an ophthalmo-scopic examination reveals redness and swelling of the eye-ground, while deeper hues reveal the characteristic flame-shaped redness. White opacities are due to fatty degeneration of the retina or to extravasation of leukocytes.

**Retinitis.**—There are three principal forms,—(1) albumin-uric, (2) syphilitic, and (3) pigmentary.

*Albuminuric retinitis* is found in from fifteen to twenty-five per cent of all cases of chronic nephritis, especially in the interstitial form. Not infrequently retinal changes occur before albumin is present to suggest nephritis. According to Gowers, three forms exist,—a degenerative, a hemorrhagic, and an inflammatory form. In the first, degeneration with retinal changes, there may be but slight alteration in the disk, or white patches of fatty degeneration are dotted over the fundus; in the hemorrhagic form, there may be but slight evidence of inflammation, and the hemorrhage assumes a striated or feathery aspect; in the inflammatory form, there is much swelling of the retina and obscuration of the disk.

*Syphilitic Retinitis.*—This form of retinitis occurs occasionally in the late stages of acquired syphilis, and is not so frequently seen as choroiditis or chorio-retinitis.

Opalescent patches are seen upon the retina, and the vitreous humor assumes a turbid condition. Failing vision is the first symptom to attract attention to this condition.

*Pigmentary Retinitis.*—This is usually seen in young adults and suggests inherited syphilis. Not infrequently two or more members of a family are thus afflicted. Pigmentary material is deposited along the course of the retinal arteries; as the deposit increases, there is a progressive loss of vision.

**Treatment.**—The general practitioner will not have many cases to treat, as the patient will consult an oculist. The few that he will treat, should be handled in the same rational manner as characterize diseases in general; namely, meet the conditions, as far as possible, by specific remedies.

Belladonna for evidence of congestion of the retina; gelsemium where there is determination of blood to the eye, and it is in an active excited condition. Antisyphilitics when indicated, such as phytolacca, echinacea, stillingia, the iodids, and kindred remedies. Although we are not to expect too much in the curative action of internal medication, we may at least retard the progressive course of the disease.

## **THE OPTIC NERVE.**

Disease may occur in the nerve, in the chiasm, or in the tract, and is always serious in character, suggesting tumors, syphilis, meningitis, or hemorrhages.

### **Optic Neuritis.**

**Etiology.**—Optic neuritis is generally secondary, and may be regarded as a symptom of a serious cerebral disturbance. While in rare instances it may be primary, the result of cold and exposure, Gowers and Bramwell claim that eighty per cent are associated with cerebral tumors.

It sometimes occurs in the course of certain infectious fevers, notably scarlet fever, measles, enteric fever, and syphilis, and is also found in Bright's disease, leukemia, and diabetes.

**Pathology.**—Congestion of the disk (choked disk, papillitis), with its accompanying blurred or hazy condition, is seen in the early stage of optic neuritis; as the disease progresses the swelling increases and hemorrhages are not uncommon. If the inflammation be slight, resolution and restoration of the nerve take place, but more frequently it results in complete atrophy of the nerve. The retina may become involved, giving rise to neuro-retinitis.

**Symptoms.**—There are no characteristic symptoms, that can be recognized, if we except an ophthalmoscopic examination, and the latter must invariably be made before we can be certain of our diagnosis.

**Prognosis.**—The prognosis is not very favorable, for while mild cases may recover, they usually terminate with partial loss of vision, and the severer cases result in total blindness.

**Treatment.**—Where the cause is specific, potassium iodid, echinacea, stillingia, phytolacca, Donovan's solution of arsenic, and kindred remedies will be given; otherwise the treatment is symptomatic.

## **THE MOTOR NERVES OF THE EYEBALL.**

### **Third, Fourth, and Sixth.**

Diseases belonging to the motor nerves (motor oculi, patheticus, and abducens), properly belong to ophthalmology, and need not be considered here.

### **The Fifth Nerve (Trifacial).**

The fifth nerve is a compound nerve, being the great sensory nerve of the head and face, and the motor nerve of the muscles of mastication.

The *ophthalmic* and *superior maxillary* divisions are entirely sensory; the *inferior* maxillary, the third division, is partly sensory and partly motor.

The *ophthalmic* division supplies the eyeball, the lachrymal gland, the mucous lining of the eye and nasal fossae, and the integument of the eyebrow, forehead, and the nose.

The *superior maxillary division* supplies the infraorbital region, the skin over the malar bone, the root of the nose, the upper lip, a large part of the nasal mucous membrane, the palate, the upper part of the pharynx, the teeth in the upper jaw, and the integument covering the temple and side of the forehead.

The *inferior maxillary division* supplies the teeth and gums of the lower jaw, the integument of the temple and external ear, the lower part of the face, and the lower lip, the tongue, and the muscles of mastication.

Lesions of the fifth nerve may be due to pontine hemorrhage or tumors of the pons, to softening or sclerosis; meningitis; injury at the base of the brain; gummata; caries of bone; or it may be due to injuries or disease of the branches after passing out, as a neuritis, or from pressure by

growths.

**Symptoms.**—*Sensory Portion.*—In the early stage, pains of a shooting, boring, or burning character are observed, with tenderness along the course of the nerve, and certain areas where there is hyperesthesia; this is followed later by anesthesia of the skin of the face and head, the conjunctiva, the mucosa of the tongue, lips, nose, and of the soft and hard palate. The muscles of the face become slower in their action, the senses of smell and taste are impaired, and the salivary, lachrymal, and buccal secretions are diminished.

*Motor Portion: Paralysis.*—The temporal and masseter muscles. may be partially or entirely paralyzed, the jaw being drawn to the affected side.

*Spasm* of the masticatory muscles—spasm of Romberg—may be tonic or clonic. In the tonic spasms the jaws are firmly locked (locked jaw), rigid, and frequently painful.

Clonic spasms may occur as a symptom of chorea, hysteria, and sometimes in women, late in life.

**Treatment.**—This -will d'epend upon the conditions present. For the sensory wrongs, when not due to organic lesions, much relief may be secured from specific plantago major, gelsemium, mellilotus, and piper methysticum. Where the pain is intense, morphia may have to be used. When due to specific causes, the iodids and similar remedies will be required.

Galvanism and Faradism will be useful in some cases.

## THE SEVENTH OR FACIAL NERVE.

**Synonyms.**—Facial Paralysis; Bell's Palsy.

**Etiology.**—Paralysis of the seventh or facial nerve may be supranuclear, nuclear, or peripheral in origin.

*Supranuclear Paralysis.*—This form is due to lesions of the cortex, corona radiata, or internal capsule, and is usually associated with hemiplegia.



Tumors, cerebral softening, hemorrhage, and abscess or chronic inflammation, may be responsible for this lesion.

It differs from the peripheral form in that the upper branches of the facial nerve are intact, enabling the patient to wink and wrinkle the forehead. The normal electrical excitability of both nerves and muscles are also preserved.

The emotional movements are not so impaired in this form of paralysis as the voluntary.

*Nuclear Paralysis.*—This form is more rare and may be due to tumors, hemorrhages, or softening, affecting the nucleus. This center has also been involved in diphtheria and anterior poliomyelitis.

The symptoms are those of the peripheral type.

*Peripheral or Intranuclear Paralysis.*—When the nerve trunk is involved within the pons, it is usually due to hemorrhage, a tumor, or softening. When affected at its point of exit, it may be due to syphilis, meningitis, tumors, or fractures at the base of the skull. Caries of the bone, or disease of the middle ear, may affect the nerve in the Fallopian tube, and at its emergence from the styloid foramen it may be influenced by pressure, as from tumors of the parotid gland, or from forceps in instrumental delivery.

**Symptoms.**—In facial paralysis, the appearance of the affected side presents a picture that is characteristic. The tissues are lax and immobile, the natural lines and furrows are diminished or entirely effaced, especially noticeable in elderly people, the skin of the forehead being smooth and without wrinkles. The lower lid droops, and the patient is unable to close the eye, owing to paralysis of the orbicularis palpebrarum; the eye waters, and the affected eye remains open during sleep.

Voluntary and emotional movements are lost, the corner of the mouth drops and the mouth is drawn to the affected side. In protruding the tongue it appears as though it were drawn to the affected side, but examination reveals the tongue in normal position, the distortion being due to the drawing of the mouth away from the affected side. The

sagging of the mouth causes the saliva to dribble. The patient can not whistle, whisper, or lift the angle of his mouth. In drinking, the lips are not perfectly closed and the fluid is apt to escape from the mouth. Owing to paralysis of the buccinator, the food collects in' the teeth and cheek of the affected side. The patient is unable to sniff, owing to paralysis of the nasal muscles.

Where the nerve is involved within the canal between the geniculate ganglion and the region of the chorda tympani, taste is lost upon the anterior two-thirds of the tongue.

There is often abnormal sensitiveness to sound, though if there has been disease of the middle ear, sensitiveness is lost or materially lessened.

Spasmodic twitching of the affected muscles may occur late in the disease.

Where the paralysis is due to cold, the facial muscles alone are involved, hearing and taste being preserved, and electrical reactions remain good. Recovery takes place in from one to four weeks.

**Diagnosis.**—The diagnosis is readily made by noting the characteristic picture as given above.

**Prognosis.**—The prognosis is generally favorable, though it may last several months. If due to traumatism, tumors, or softening, it will most likely be permanent.

**Treatment.**—The treatment will depend upon the cause giving rise to the lesion. If due to cerebral tumors, softening, and like causes, but little result will be obtained from medication. If the result of middle-ear diseases, thorough drainage must be secured. If due to syphilis, potassium iodid, echinacea, berberis aquifolium, corydalis formosa, thuja, and Donovan's solution will give good results.

If due to cold, gelsemium, bryonia, macrotys, rhamnus Californica, and other anti-rheumatics will be suggested.

Galvanism should receive a thorough trial.

## THE AUDITORY NERVE.

Diseases of the auditory nerve may be due to tumors, softening, syphilis, chronic inflammation, meningitis, aneurism, otitis media, and labyrinthine disturbances. Quinine and the salicylates also affect the hearing.

Hyperacusis (hyperesthesia) is that condition when certain or all sounds are abnormally increased. It sometimes occurs in hysteria and in the course of cerebral diseases. Paralysis of the stapedius is also followed by an abnormally acute hearing.

*Dysacusis*, difficult hearing, may be due to any of the above-mentioned causes that affect the auditory nerve, but more frequently to middle-ear diseases.

Tinnitus aurium is that condition where abnormal sounds occur, such as ringing, crackling, buzzing, whirring, or tickling sensations, and may be due to accumulations of cerumen, labyrinthine disturbances, otitis media, anemia, aneurism, and sometimes it occurs as an aura in epilepsy. These abnormal sounds may be more or less constant or exaggerated at night and when the system is depressed or impoverished.

**Treatment.**—In the treatment of diseases of the auditory nerve, a careful and patient study must be made to determine its cause, and, if possible, remove it. The general health is to be considered and placed in the best possible condition. Where due to specific causes, the antisiphilitis will be used. Galvanism should be tried. This lesion is for the specialist rather than the general practitioner, and should be referred to the aurist.

### **Ménière's Disease: Auditory or Labyrinthine Vertigo,**

In 1861 Ménière described an aural vertigo, where the attack came on suddenly and occurred as a paroxysmal affection, the characteristic symptoms being vertigo, deafness, and tinnitus aurium.

**Etiology.**—The disease occurs more frequently in men than in women, and generally after the age of thirty-five. It is probably due to

labyrinthine disturbances.

**Pathology.**—But little is known of its true pathology, and whether the lesion be in the semicircular canals or in the cerebral centers, is not known.

**Symptoms.**—The attack comes on suddenly, with the sensation of being struck. With the dizziness, there comes a sense of nausea, which rapidly increases, ending in vomiting of bile. In mild cases, the patient, though quite dizzy, retains consciousness, but when severe, he may fall unconscious. Deafness and tinnitus usually are present during an attack.

The attacks recur at irregular intervals, varying from a day to several months.

**Prognosis.**—The prognosis is uncertain, some cases recovering, while others grow progressively worse until deafness is confirmed.

**Treatment.**—The treatment is not very satisfactory. Charcot recommended quinine in twenty-grain doses daily, to be continued for several weeks. Gowers advises sodium silicylate in five-grain doses, while the bromids are suggested by others.

## **THE GLOSSOPHARYNGEAL NERVE.**

The glossopharyngeal nerve is so intimately connected with the trigeminus, the facial, the pneumogastric and sympathetic nerves, that its function is not very well understood; hence very little is known as to lesions of this nerve. Difficulty in swallowing, and loss of sensation in the roof and walls of the pharynx, are most likely due to paralysis of the ninth nerve.

## **THE PNEUMOGASTRIC NERVE.**

The pneumogastric nerve has a longer course and a wider distribution than any of the cranial nerves, being distributed to the pharynx, esophagus, larynx, lungs, heart, stomach, intestines, and spleen.

**Etiology.**—The nucleus may be involved by hemorrhage, softening, tumors, or by degeneration, as seen in bulbar-paralysis.

The nerve-root within the skull may be disturbed by meningitis, by morbid growths, or by aneurism of the vertebral artery.

In the neck it may be disturbed by pressure from tumors, or it may be ligated in tying the carotid artery, or injured in surgical operations or by punctured or incised wounds. Neuritis from exposure or toxemia is quite rare.

*Pharyngeal Branches.*—Functional disturbances of the pharyngeal branches results in spasm of the muscles and mucous membrane of the larynx, as seen in nervous individuals, and generally occurs in hysterical patients, and is known as globus hystericus.

Paralysis of the branches may follow diphtheria or accompany bulbar paralysis, and causes difficulty in swallowing, the food being inclined to enter the larynx, where it gives rise to coughing or to severe choking. If the soft palate be involved, the food is regurgitated into-the nose.

*Laryngeal Branches.*—Functional disturbances of the laryngeal branches gives rise to spasm of the adductors—laryngismus stridulus—not infrequent in children, but rare in adults. Paralysis of the branches gives rise to stridulous respiration, cough, and hoarseness, or complete aphonia, with more or less dyspnea. It may result from diphtheria, or from pressure upon the recurrent laryngeal, from aneurisms, goitre, or morbid growths.

*Pulmonary Branches.*—Since the bronchial muscles are supplied by these branches, their disturbance gives rise to spasm of the intrinsic muscles, as seen in bronchial asthma.

Where paralysis occurs, respiration is slow and sometimes labored, and accompanied by cardiac disturbances.

*Cardiac Branches.*—The cardiac branches are motor, sensory, and trophic; hence the variety of cardiac phenomena displayed by disturbance of these branches.

*Irritation of the motor branches* slows the action of the heart

—bradycardia—and may be due to a pure neurosis or to compression from tumors.

Paralysis of the vagus abolishes inhibitory action, when the accelerators are unrestrained, and the heart fairly runs away— *tachycardia*. This may follow diphtheria, wounds, accidental ligature of the nerve, or pressure from growths.

When the *sensory* branches are disturbed, palpitation and pain are experienced, as witnessed in angina and other painful cardiac affections.

*Trophic* disturbance is seen in fatty degeneration of the heart, following injury to the vagus.

*Esophageal Branches.*—Functional disturbances occur more frequently than paralysis; spasm of the esophagus, occurring in hysterical patients, is attended by difficulty in swallowing. Paralysis of the esophagus results in difficulty in swallowing.

*Gastric Branches.*—These are both motor and sensory. A disturbance of the sensory branches gives rise to pain, as seen in nervous dyspepsia and other gastric disturbances. Hunger is probably a pneumogastric sensation.

A section, or paralysis of the nerve, lessens the contractile power of the stomach, though it does not destroy it entirely. The vomiting occurring in cerebral disturbances is most likely due to irritation of these branches.

Irritation of the intestinal branches accelerates the action of the intestines, though intestinal disturbances from lesions of the vagus are so rare that little has been learned regarding them.

**Treatment.**—The treatment must be symptomatic. When due to specific causes, the antisyphilitics will be used. Electricity and massage should be tried in other cases. Where the lesion is functional, spasm resulting, the antispasmodics will be given, such as gelsemium, lobelia, the bromids, chloral, and like remedies.

## THE SPINAL ACCESSORY NERVE.

The spinal accessory nerve consists of two parts, an internal or accessory portion, and an external or spinal portion.

The accessory part forms the motor portion of the pneumogastric and is distributed to the pharyngeal and laryngeal muscles, lesions of which have been considered in lesions of the vagus.

Lesions of the spinal part may result in spasm or paralysis. Torticollis—wry neck—may be congenital, fixed wry-neck, or spasmodic, acquired torticollis. This may be due to injury at birth, or to some abnormal intra-uterine condition, and results in shortening and atrophy of the sterno-mastoid muscle. The right side is almost exclusively affected. The development of the face on the affected side is slower than that of its fellow, hence facial asymmetry results.

The symptoms are not usually noticed for several months, owing to shortness of the baby's neck.

**Treatment.**—This is surgical, tenectomy relieving the deformity.

*Spasmodic Wry-neck.*—This form may be either clonic or tonic or a combination of the two. Males are more frequently affected than females, and it usually occurs between the ages of thirty and fifty. When it occurs in females, it is usually found in those of a hysterical nature and under thirty years of age. There is generally a neurotic family history. Cold may be an exciting cause, especially in persons inclined to rheumatism.

**Symptoms.**—While spasm may be the first symptom, it is often preceded by a sharp neuralgic pain or one of a dull character, or it may be that a sense of stiffness is the first premonitory warn-The spasm often comes on gradually, involving the sterno-mastoid alone, or it may include the trapezius.

The occiput is rotated toward the shoulder of the affected side, while the chin is elevated and the face turned to the sound side. The facial nerve, as well as the brachial plexus, may become involved, giving rise to a combined spasm of the muscles supplied.

The spasm is usually in abeyance during sleep. Clonic spasms are apt to be more painful than tonic spasms, the latter exhibiting more fatigue of the muscles than actual pain.

**Prognosis.**—The disease is apt to be chronic, though, after months or years, it may cease to progress, and improvement begin. If recovery takes place, recurrences are frequent.

**Treatment.**—Where functional, the antispasmodics and anti-rheumatics should be given a thorough trial. Such remedies as gelsemium, passiflora, scutellaria, plantago major, hyoscyamus, and the bromids of the former class, and macrotys, bryonia, and rhamnus Californica of the latter. Where pain is intense, a hypodermic of morphia may be necessary to overcome the spasm and relieve the pain. Galvanism is sometimes useful, and nerve-stretching has afforded relief in some cases.

*Paralysis of the Spinal Accessory Nerve.*—The same causes may be active in affecting the muscles and nerve-trunk that were seen in lesions of the pneumogastric; namely, degenerations, morbid growths, meningitis, or toxemia.

In paralysis of the spinal portion there is atrophy of the sterno-mastoid on the affected side, impairing the power of rotating the head toward the opposite side, and partial paralysis of the trapezius, which interferes with lifting the arm. The shoulder drops, and the supra-clavicular depression is increased.

Where there is bilateral paralysis of the sterno-mastoid with atrophy, the head falls backward, but if the trapezii are affected the head falls forward.

**Treatment.**—The cause must be carefully sought for, and, when a removal is possible, a cure may be effected. If the lesion be nuclear, but little may be expected from medication.

Where due to pressure, surgery may afford relief. Electricity promises well in some cases.



## THE HYPOGLOSSAL NERVE.

The hypoglossal nerve is the motor nerve of the tongue, and, like the preceding nerves, is affected by degenerative changes, injury, meningitis, tumors, syphilis, and toxemia.

Nuclear lesions, usually bilateral, are frequently associated with locomotor ataxia; cortical lesions with hemiplegia. Spasm or paralysis may attend disease of this nerve.

*Spasm.*—Spasm of the tongue may result from reflex irritation of the fifth nerve, or it may be a part of a general spasm, such as epilepsy or chorea. It sometimes occurs in hysteria and in some forms of stuttering. A rare and peculiar form of clonic spasm is where the tongue is thrust in and out with great rapidity.

*Paralysis.*—When a complete bilateral paralysis occurs, the tongue lies motionless on the floor of the mouth, and speech, mastication, and deglutition are difficult, but taste and touch are not impaired.

When the paralysis is unilateral, the tongue deviates to the affected side when protruded, unless the lesion is within the medulla, when it turns toward the sound side.

**Prognosis.**—It is generally unfavorable.

**Treatment.**—This is symptomatic, and consists of removing the causes, where possible, and building up the general health. The judicious use of tonics and alteratives and the indicated remedy will form the wisest form of medication. Electricity will deserve a trial.

## II. DISEASES OF THE SPINAL CORD.

### DISEASES OF THE SPINAL DURA MATER.

#### PACHYMENINGITIS.

**Definition.**—Pachymeningitis is an inflammation of the outer or inner surface of the dura mater (*pachymeningitis externa or interna*).

Pachymeningitis externa is more of a primary lesion, it always being the result of pressure from morbid growths, caries of the vertebra, or syphilitic deposits. It may be acute or chronic.

In the *acute form*, the inflammation is generally sero-purulent, the symptoms being those of compression-myelitis. The *chronic form* is generally due to tuberculosis of the vertebrae. (Pott's disease.) The external layer is rough, thickened, and covered with a cheesy material.

**Symptoms.**—There may be hyperesthesia and motor spasms, to be followed later by anesthesia, paralysis, atrophy of the muscles, and loss of reflexes—Pachymeningitis interna. Hyper-trophica or cervicalis hypertrophica, is of an obscure origin. It was first described by Charcot in 1871. It is generally limited to the cervical region. The dura mater is much thickened, which destroys the nerve-roots and causes compression of the cord.

In the early stages of the disease neuralgic pains are experienced in the occipital region and in the upper extremities. There may be hyperesthesia, and sometimes a herpetic eruption is present. As the disease progresses, compression of the cord increases, which results in atrophic paralysis, and which gives rise to hand deformity—claw-hand.

**Prognosis.**—The disease is progressive and terminates in death.

**Treatment.**—The treatment recommended, the iodids and electricity, has not been attended with very satisfactory results.

## **DISEASES OF THE SPINAL PIA MATER.**

### **ACUTE LEPTOMENINGITIS.**

**Synonym.**—Acute Spinal Meningitis.

**Definition.**—Leptomeningitis is an inflammation of the pia mater, and may be acute or chronic.

**Etiology.**—The exciting cause is not positively known, though it is now generally accepted as being due to infection from pyogenic cocci, the most frequently seen being the pneumo-coccus, the meningo-coccus

following next.

Exposure to wet and cold, as well as direct or indirect injury to the spinal column, such as fractures, wounds, or surgical operations, has been followed by meningitis, though these may have been only preparatory means for the entrance of the infecting' bacteria.

It is often associated with tuberculosis and the infectious fevers, particularly croupous pneumonia, typhoid fever, scarlet fever, diphtheria, pyemia, and septicemia. It may also be due to an extension of inflammation of the meninges of the brain.

**Pathology.**—The changes in the membranes of the cord are those peculiar to inflammation. First, hyperemia, which may be diffused throughout the length of the cord or localized. This is followed by, an exudation of fibrinous material containing red blood-curpuscles and leukocytes, which later changes to pus. The nerve-roots may also be involved. Owing to the dorsal position assumed, the exudate is more profuse on the posterior portions of the pia mater, due to gravitation. Similar lesions are found in the cerebral meninges, in the majority of cases.

**Symptoms.**—The disease generally begins with a well-marked chill followed by febrile reaction, which early may assume a septic type. Where the cerebral meninges are involved, the early symptoms, flushed face, bright eyes, contracted pupils, and restless condition, may for a time obscure the main lesion. Soon, however, the intense pain in the cervical region, the marked hyperesthesia, and the tonic or clonic spasm of the muscles drawing the head backwards, removes all doubt as to the seat of the lesion.

There is marked tenderness along the spine, and when the spasms are intense there may be opisthotonos. There may be retention of urine, due to reflex spasm of the bladder. The reflexes are all exaggerated.

As the acute inflammation subsides, anesthesia replaces the hyperesthesia, paralysis follows, and the reflexes disappear.

**Diagnosis.**—The diagnosis should not be difficult. The tenderness along the cervical region, the intense pain in the back, radiating to the upper and lower extremities, retraction of the head, hyperesthesia,

rigidity of the muscles, and fever of a septic type, can not be mistaken for any other lesion.

**Prognosis.**—This is a grave lesion, and the prognosis must be guarded. Death may result in forty-eight hours, or be prolonged for two or three weeks.

**Treatment.**—Aconite in the small dose, and gelsemium and echinacea in full doses, will form a good treatment, thus:

Spec. Aconite	5 drops.
Spec. Echinacea	2 drams.
Spec. Gelsemium	1/2 dram.
Water	4 ounces. M.

Sig. Teaspoonful every hour.

Rhus tox., macrotys, and bryonia should be given as may be indicated, and the sulphites, chlorates, and mineral acids where sepsis indicates their use. Great care must be exercised during convalescence, that the patient avoid everything that tends to excitation.

## CHRONIC LEPTOMENINGITIS.

**Synonym.**—Chronic Spinal Meningitis.

**Definition.**—Chronic leptomeningitis is a chronic inflammation of the meninges of the cord.

**Etiology.**—This may follow the acute form, though generally the result of syphilis, alcoholism, and disease of adjacent bone, which may be tubercular carcinomatous, or sarcomatous. It may be the result of concussion of the spine.

**Pathology.**—The pia mater is injected, swollen, edematous, and infiltrated with cells. Adhesion of the pia mater and the dura mater often occurs, or they may be welded together. The ventricles may contain serum, and the ependyma lining them is frequently thickened. There may be hemorrhages into the membrane.

**Symptoms.**—The symptoms are not well denned, and are those of slow compression. In some cases they are negative, while in others, where the nerve-roots are involved, there will be hyperesthesia, radiating pains, stiffness, tremors, and sometimes paralysis. The symptoms are progressive and may extend over years.

**Prognosis.**—Although the disease is not rapid, it generally terminates in death.

**Treatment.**—Diet, hygienic measures, and remedies that improve the general health are to be considered in the treatment. Open air and an equable climate and easily digested food should be recommended.

### **HEMORRHAGE INTO THE SPINAL MEMBRANE.**

**Synonyms.**—Meningeal Apoplexy; Hematorrachis.

**Definition.**—Extramenigeal hemorrhage is where the blood is between the dura mater and the spinal canal. Intramenigeal hemorrhage, is where the bleeding takes place between the dura mater and the pia mater.

**Etiology.**—Most menigeal hemorrhages are due to violence, resulting in injuries, as from blows or falls. The rupture of an aneurism is sometimes responsible for this condition. Cancer may give rise to hemorrhage by erosion of the blood-vessels. Diseases attended by severe convulsion have also figured as causal factors. It may occur as a complication of any of the infectious fevers. Cerebral hemorrhage may result in extravasation of blood into the membranes of the cord. Males are more apt to suffer than females, no doubt owing to greater exposure.

**Pathology.**—The peridural space accommodates quite a large amount of blood without resulting in pressure, hence large hemorrhages are found in the extramenigeal form.

In the intramenigeal form the hemorrhages are small and scattered.

**Symptoms.**—If the hemorrhage be small the symptoms are negative, but if large enough to produce pressure, are apoplectiform in character although consciousness is retained.

The first effect is irritation and hyperesthesia, muscular irritability, radiating neuralgic pains, and herpes follow. Later, however, the pain ceases, and anesthesia and paralysis follow. The bladder and rectum may share in the lesion, incontinence being the distressing feature. Where the hemorrhage is high, respiration is involved.

**Diagnosis.**—This is generally difficult, a positive diagnosis being seldom made.

**Prognosis.**—This depends largely upon the amount of the hemorrhage and the causes giving rise to it. If the hemorrhage be small and the general health good, absorption rapidly takes place, with complete-recovery.

**Treatment.**—The treatment will be that used for hemorrhage of any part or organ. Rest in bed, cold applications, and the internal administration of gallic acid, ergot, the oils of cinnamon and erigeron, mangifera indica, and others of like character. Massage, electricity, and the iodids will be used to promote absorption.

## **HEMORRHAGE INTO THE SPINAL CORD.**

**Synonyms.**—Spinal Apoplexy; Hematomyelia.

**Definition.**—A hemorrhage directly into the substance of the cord.

**Etiology.**—Spinal apoplexy may be due to traumatism, to changes in the coats of the blood-vessels, or to excessive blood-pressure.

**Pathology.**—The clot may be as large as an almond, seldom larger, the appearance of which depends upon its age; if recent, it will be red or dark in color, and changes to brown or yellow with age; still later it may become encapsuled by a fibrous deposit. There may be gliomatous tumors or acute myelitis.

**Symptoms.**—The symptoms depend somewhat upon the seat of the lesion and the extent of the hemorrhage. Where the hemorrhage is large, severe shooting pains occur in the back and limbs, with muscular twitchings, cramps, and rigidity. Usually the pain in the back is severe,

subsiding within twenty-four hours. Paraplegia is sudden, with loss of sensation and the reflexes, and not infrequently loss of control of the bladder and bowels.

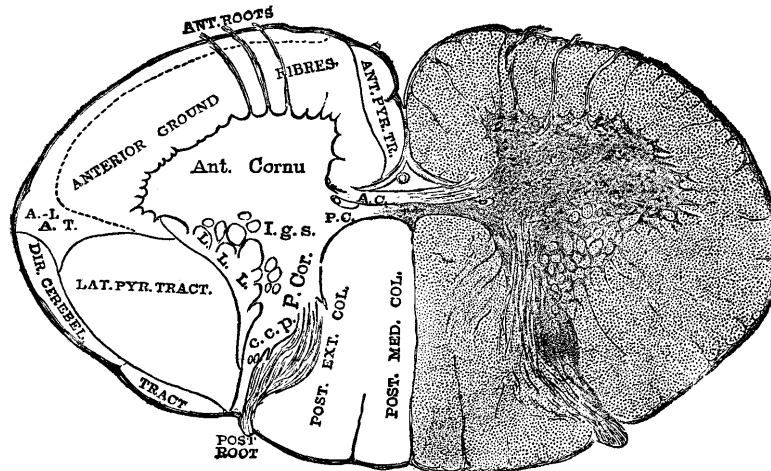


FIGURE 37. SECTION OF A NORMAL SPINAL CORD, IN THE CERVICAL REGION.—(After Cowers.)—(Tyson.)

A.C. Anterior Commissure. P.C. Posterior Commissure. I.g.s. Intermediary gray substance, the gray matter between the two horns. P.cor. Posterior Cornu. C.C.p. Caput Cornu posterioris. L.L.L. Lateral limiting layer. A.L.A.T. Anterio-lateral ascending tract of Gowers, which extends along the periphery of the cord.

Unless the lesion is near the medulla, consciousness is retained. A slight fever may follow after twenty-four or forty-eight hours.

The type of paralysis may be monoplegia, hemiplegia, or paraplegia. Disturbance of the sensory functions may give rise to hyperesthesia, anesthesia, paresthesia, etc.

**Diagnosis.**—The diagnosis is always difficult, the difference between the symptoms of hematomyelia and hematorrachis being so slight as to render a positive diagnosis almost impossible.

**Prognosis.**—This depends upon the extent and location of the clot. Thus hemorrhage into the dorsal segments is less serious than in the cervical or lumbar segments, respiration being disturbed in the former through interference with the phrenic nerve, and the vesical and rectal sphincters in the latter.

If the hemorrhage be small in quantity, absorption may speedily take

place with complete recovery. The rapid development of bed-sores is an unfavorable symptom.

**Treatment.**—The same line of treatment as used in spinal-meningeal hemorrhage, can be carried out in this lesion.

### ACUTE MYELITIS.

**Synonyms.**—Myelitis; Acute Diffuse Myelitis; Transverse Myelitis; Spinal Malacia.

**Definition.**—An acute inflammation of the spinal cord, extending transversely or longitudinally, and attended by softening or sclerosis of the cord.

**Etiology.**—The disease occurs more frequently in men than in women, probably due to greater exposure of the former. Exposure to wet and cold, especially in alcoholics, may be followed by myelitis, and it frequently follows the infectious fevers, especially small-pox, typhus fever, and measles. Traumatism of the spine, caries of the bones, great emotional excitement, and certain mineral poisons, especially lead, arsenic, mercury, and phosphorus, may induce it. Gout, rheumatism, and syphilis not infrequently precede myelitis, though syphilis is more apt to give rise to the chronic form,

**Pathology.**—The lesion is most frequently found in the upper dorsal region, next in the cervical, and rarely in the lumbar region.

The disease may be limited to a small vertical area extending entirely across the cord—*transverse myelitis*—or a large portion may be affected—*diffuse myelitis*; again several areas may be involved in different parts of the cord, when it is termed *disseminated myelitis*, and is known as *central myelitis* when only the gray matter is involved.

Upon ocular inspection the cord may present no visible changes, or it may show extreme softening, almost diffluent, or it may be sclerotic, owing to proliferation of interstitial connective tissue.

The cord may be swollen, and red, gray, or yellow in color, according to the amount of blood, connective tissue, or fat contained.



**Symptoms.**—The symptoms will naturally depend upon the location and extent of the lesion. The most frequent seat of election is the dorsal region, and transverse myelitis is the most common type.

The attack may resemble the first stage of rheumatic fever, beginning with a well-marked chill, rapidly followed by febrile reaction; there is headache, backache with general muscular soreness, nausea, vomiting, and sometimes convulsions. In a few days, however, the spinal symptoms appear, paralysis of motion with complete loss of sensation below the site of the lesion, rapidly develop, incontinence of the sphincters of the bladder and rectum follow, and bed-sores over the sacrum and hips appear.

When the cervical region is involved, the onset may occur suddenly, as noted in the dorsal region, or the attack comes on more insidiously. The upper extremities are now involved, and respiration is disturbed. If very high up, death early ensues owing to failure of respiration.

The sensory symptoms are at first a tingling, crawling, or burning sensation, to be followed shortly by entire loss of sensation. The reflexes may be permanently lost, or they may return in an exaggerated form. Superficial ulceration occurs most frequently when the lumbar region is involved. The course of the disease depends upon the extent and cause of the disease.

*Complications and Sequela.*—Cystitis occurs as the most frequent complication, and is usually due to retention of urine. Pulmonary and renal complications are common, and amyloid degeneration of the kidneys is sometimes found.

*Diagnosis.*—The sudden onset of the disease, with early development of paraplegia, paralysis of the sphincters, loss of sensation, absence of muscular pains, and the rapid trophic changes, make the diagnosis comparatively easy.

**Prognosis.**—Since secondary changes are very likely to occur in the cord, *complete* recovery is rare, more or less paraplegia remaining. Where trophic changes are marked, the outlook is unfavorable, and, in some severe, acute cases, death may take place in three or four days. Prolonged high temperature is also unfavorable. When syphilis is

responsible for the lesion, the prognosis is more favorable.

**Treatment.**—In the acute state, treatment will be the same as that for acute meningitis; namely, aconite and gelsemium for the febrile condition and irritation of the cord. Rhus tox. will also be found useful, the indications being the same as for other lesions; namely, *sharp stroke* of the pulse, starting in the sleep, and general restlessness.

For severe sacral or lumbar pains, with muscular twitchings, full doses of gelsemium with rhamnus Californica or macrotys should be given, thus:

Gelsemium.	
Rhamnus Cal.	
Macrotys	1 dram each.
Water	4 ounces. M.

Sig. Teaspoonful every hour.

Owing to the tendency to trophic changes, care must be taken to allow no local applications to be made along the spine, after the acute stage has passed, that would tend to injury of the skin. In fact, even in the acute stage, local measures should be confined to soothing measures rather than irritants. The bladder should be catheterized at regular intervals where there is loss of control of the sphincters. Care should be taken to have the catheter aseptic.

After the acute stage has subsided, massage, electricity, and a general tonic treatment will be found useful. Strychnia, iron, and the hypophosphites, or the old Aitkin Comp. tonic mixture—the triple phosphate of iron, quinia, and strychnia—can be used with advantage.

Specific belladonna must not be overlooked, where there is capillary congestion of the cord. It will be given in the usual small dose, ten drops to four ounces of water, a teaspoonful every one, two, or three hours.

## CHRONIC MYELITIS.

**Etiology.**—Chronic myelitis may follow the termination of an acute attack, or it may be due to trauma, hemorrhage, tumors, or caries of the

vertebra.

The disease may come on insidiously, the result of syphilis or from the toxin of some of the infectious fevers. In rare cases, it may be due to cold from exposure.

**Pathology.**—While it may be confined to a single focus, the lesions are most often disseminated or diffuse. Sclerosis is the most pronounced lesion, even the membranes, in some cases, sharing in the sclerosis. The ascending and descending tracts may be involved in the same process. The blood-vessels are thickened; hence there is an absence of recent hemorrhage. The pia may be thickened and adherent. The nerve-cells in the gray matter become atrophied or they may entirely disappear.

**Symptoms.**—The symptoms are about the same as in the acute form, save the fact that they come on more insidiously, and are not so well defined, several months elapsing before sufficiently pronounced to consult a physician.

There may be perverted sensation, such as numbness, tingling, crawling, etc., though complete loss of sensation is rare.

The motor symptoms are gradual in their onset, and irregular in their development, especially where the lesion is disseminated or diffuse. The patient notices a weakness in a part, as the hand or arm, which is followed soon by impaired sensation; then a foot or leg develops weakness, to be followed by disturbed sensation, and finally paralysis.

Where there is transverse myelitis of the lumbar or sacral region, there is paresis of the lower extremities, with involvement of the sphincters. Atrophy of the muscles is more pronounced, when the lesion is of the cervical or dorsal region. The degree of trophic changes depends upon destruction of the cells in the anterior horn of the spinal cord.

**Diagnosis.**—The diagnosis is not always readily made, though when the symptoms enumerated are well defined, there should be little difficulty.

**Prognosis.**—The disease has progressed to such an extent that degenerative changes have generally taken place before the physician is consulted; hence the prognosis is necessarily grave, though recovery

may possibly take place in rare cases. The disease, however, may extend over a period of years before it terminates fatally by exhaustion.

**Treatment.**—In the early stages, rest in bed, associated with spongbaths and massage, is very beneficial. As the disease becomes more chronic, our attention will be directed to maintaining as good a condition of the general health as possible. With the elaboration of a good blood, degenerative changes are stayed, and all the symptoms modified. To accomplish this end, hygienic and dietetic methods will be important adjuncts to the treatment. Change of air and climate, having the patient much in the open air, is very desirable.

Digestion will be improved by the bitter tonics, and secretion from the skin, kidneys, and bowels should be encouraged. For the motor disturbances, physostigma in from one to three drop doses is recommended by Goss. Phosphide of zinc in one-tenth grain doses three or four times a day has also been used. Electricity will have its place, though too much dependence must not be placed upon remedies or methods.

## **ANTERIOR POLIOMYELITIS.**

**Synonyms.**—Atrophic Spinal Paralysis; Infantile Spinal Paralysis.

**Definition.**—Anterior poliomyelitis is an inflammation of the anterior horn, occurring most frequently in children under three or four years of age, though occasionally found in adults, and characterized by paralysis of one or more of the extremities, followed by atrophy of the muscles, but without loss of sensation.

**Etiology.**—The exciting cause is unknown; it usually occurs in children under three or four years of age, sex having no influence. Dentition, cold, injuries, mental or physical exertion, menstrual suppression, sexual excesses, syphilis, and various dissipations have been regarded by various writers as possible causes, though, most likely, the only influence they exert is predisposing, by lowering the vitality. It occurs most frequently during the summer months.

It may be epidemic in character. Dr. Caverly reporting one hundred and twenty-six cases in Otter Creek Valley, Vermont, during the summer of

1894. Other epidemics have been reported in Norway, Sweden, and in various parts of Europe and America. These epidemics suggest the lesion to be the result of microorganisms.

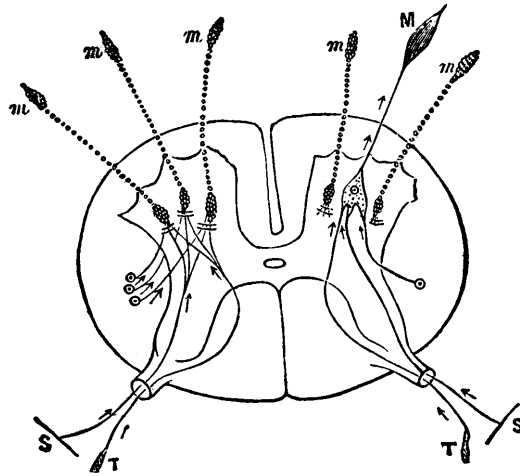


FIG. 38. Diagrammatic representation of the symptoms that result from acute destruction of the anterior cornua of the spinal cord.—(Bram-well.) On the left side the destruction of the nerve-cells is complete; the anterior nerve-roots, motor nerve-fibers, and the muscles which they supply are all degenerated; there is a total “block” to the passage of voluntary motor and reflex motor impulses. On the right side two-thirds of the motor cells are destroyed; two-thirds of the muscular area connected with the right anterior cornu are degenerated and atrophied: one-third (M) remains healthy, and can be made to contract by voluntary or reflex motor impulses. (Lockwood.)

**Pathology.**—The chief lesion is an acute hemorrhagic focal myelitis in the cervical or lumbar enlargement, and is generally unilateral. There is congestion, followed by degeneration of the motor cells of the anterior horn. During the early stage, there is infiltration of leukocytes, blood-cells, and small round cells, into the gray matter about the motor cells. This is followed later by a growth of sclerotic tissue. Since the anterior motor cells preside over the nutrition of the anterior nerves and the muscles of which they are distributed, granular or fatty degeneration and atrophy of the nerve and muscles follow the destruction of the motor cells, and, as a result, the affected limb or part has an imperfect growth, becomes cyanotic, and frequently has a lower temperature.

**Symptoms.**—The disease generally begins abruptly with chills and convulsions, followed by fever, the temperature reaching 102° and 103°. There may be pain in the head, back, and limbs, twitching of the muscles, vomiting, and diarrhea.

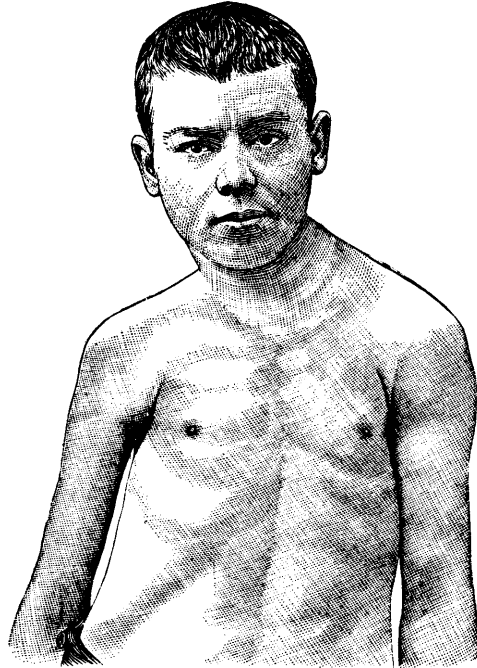


FIG. 39. POLIOMYELITIS ANTERIOR (ACUTE) FOLLOWED BY EXTENSIVE ATROPHY, CHIEFLY OF THE RIGHT SIDE. (*Ranney.*)

In twenty-four or forty-eight hours, paralysis appears, the location of which depends upon the situation of the lesion. The legs are more frequently affected than the arms, about four to one. The type is generally paraplegic.

In some cases the child goes to bed in his usual good health, to awaken in the morning with paralysis. The paralysis reaches its fullest extent the first week, remains stationary from two to eight or ten weeks, when improvement begins, the last muscle affected being the first to show improvement. In a few weeks, however, what seemed an encouraging condition, ceases, and permanent paralysis results.

The muscles of the paralyzed limb soon atrophy, the circulation is impaired, the limb becomes cold, blue, and the muscles flabby. As a result of impaired nutrition in the bone, permanent shortening of the limb occurs. The reflexes, both superficial and deep, are lost.

**Diagnosis.**—The diagnosis, after the first few days, is not difficult, the inability to use the affected limb, together with atrophy of the muscles, renders the diagnosis plain.

**Prognosis.**—The prognosis is favorable as to life, and though some improvement always takes place, permanent paralysis in arm or leg generally remains. When the muscles respond early in the disease to the faradic current, recovery is likely to take place. The prognosis is more favorable where the disease begins as an acute fever, than where it comes on suddenly during the night, the child having been previously in good health.

**Treatment.**—In the acute febrile stage, the special sedatives, frequent sponging of the patient, and absolute rest in bed, will give the best results. Later such hygienic and dietetic measures as will best improve the general condition should be carried out.

Nux vomica and strychnia will be useful after the acute symptoms have subsided. Ergot has been recommended in the early stage of paralysis.

The affected parts should be carefully massaged two or three times a day, and the faradic current applied daily to such muscles as respond, and the galvanic current to the paralyzed group.

Orthopedic appliances may assist materially in deformities of the limbs.

## **ACUTE ASCENDING PARALYSIS.**

**Synonym.**—Landry's Paralysis.

**Definition.**—An acute paralysis beginning in the lower extremities, and extending rapidly upward to the trunk and to the upper extremities, neck, and face, and finally involving the medulla. Its course is rapid and generally terminates fatally.

**Etiology.**—The disease occurs more often in men than in women, and between the ages of twenty and thirty. The disease is of unknown origin, and while it has followed cold, malaria, syphilis, and other infectious diseases, it is more likely a coincidence than a result.

**Pathology.**—There are no characteristic pathological changes found. In some cases an interstitial neuritis has been found, while in others the lesion has been that of myelitis. While the microscope fails to reveal any

morbid changes in many cases, in others there is softening and extravasation of blood into the gray substance.

**Symptoms.**—The first evidence of the disease is a sense of weakness in the lower extremities which soon develops into paralysis. Beginning in the toes, it rapidly extends up the legs and thighs to the muscles of the trunk, arms, and neck, involving respiration, deglutition, and articulation. The muscles do not waste, electrical reactions are maintained, though the reflexes are lost.

The bladder and rectum are not involved, and bed-sores are rarely developed. There may be paresthesia, though sensory symptoms are not constant. The disease may terminate fatally in forty-eight hours, or be postponed for one or two weeks.

**Diagnosis.**—Weakness of the limbs, rapidly followed by paralysis, beginning in the feet and rapidly ascending to the trunk, neck, and face; impaired respiration, relaxation of muscles, with but little atrophy; loss of the reflexes; absence of electric changes, and absence of sensory symptoms, make the diagnosis comparatively easy.

**Prognosis.**—Recovery occurs only in rare cases.

**Treatment.**—The treatment will be symptomatic, and similar to that for any acute disease of the cord.

When following an infectious disease, the antiseptics might be given with benefit, notably echinacea.

After the acute symptoms subside, electricity and massage should be tried.

## **LOCOMOTOR ATAXIA.**

**Synonyms.**—Tabes Dorsalis; Posterior Sclerosis.

**Definition.**—A chronic disease characterized by degeneration and sclerosis of the afferent tract and posterior columns of the cord, and which results in muscular inco-ordination, sensory and trophic disturbances, loss of knee-jerk, and the Argyle-Robertson pupil.



**Etiology.**—The predisposing causes are age, sex, and race. The disease is rarely seen under twenty-five years of age, being most common between the ages of thirty and forty, and more frequently in men than in women, the ratio being about ten to one. It is much more common in white races than in the colored, and Jews are less seldom affected than Gentiles.

Syphilis bears a very important relation to tabes and is undoubtedly the most important of all predisposing causes. There is a history of syphilis in from seventy-five to ninety per cent of all tabetic cases. Thus Erb reports that eighty-nine per cent of three hundred cases in private practice, had a history of syphilis, while Mobius believes that tabes never exists without syphilis.

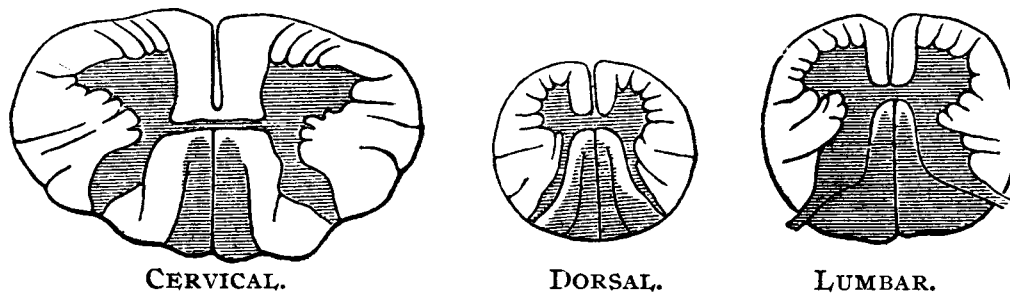


FIG. 40. POSITION OF LESIONS OF LOCOMOTOR ATAXIA IN DIFFERENT PARTS OF THE SPINAL CORD.—(Taylor.)

Among the more common exciting causes may be mentioned sexual excesses, great physical exertion, and exposure to wet and cold. It is more common in cities than in the country.

**Pathology.**—The basal or pathological lesion underlying tabes is a parenchymatous degeneration, resulting in sclerosis, and involving principally the sensory neurons, though the peripheral motor neurons are also not infrequently affected; in fact, although the principal lesion is in the posterior columns of the spinal cord, tabes may be said to be degenerative conditions affecting various parts of the entire nervous system. These degenerative changes begin in the posterior root-zones, advance into the tract of Lissaur and the columns of Burdach, Clark, and Gall, connective tissues ultimately largely replacing the nerve-fiber, the contraction of which causes compression of the cord. There may also be cerebral and medullary changes, as well as changes in the cranial nerves and their nuclei, and the peripheral nerves in the extremities.

Blindness may result from gray degeneration of the optic nerve.

In some cases, trophic changes are seen in the bones, especially their articular surfaces.

**Symptoms.**—It is customary to divide the symptoms into three stages, though in some cases it is hard to separate the one from the other. They are the preataxic, the ataxic, and the paralytic.

1. THE PREATAXIC STAGE.—Although all tabetic cases do not begin in the same way, the most frequent and characteristic symptoms are as follows:

*Pains.*—Pains of lightning-like character occur in ninety per cent of the cases, and are sharp, stabbing, piercing, or cutting in character; they are usually in the lower extremities, though they may occur in the upper extremities, the face, stomach, or rectum.

They last only for a second or two and may be associated with a burning sensation. Occasionally herpes develops at the seat of pain. These attacks occur irregularly, and are apt to follow excesses, cold, and damp weather. In a few cases pain is absent.

*Paresthesia.*—Perverted sensation is often experienced; the patient complains of a numb or tingling sensation in the legs or feet, or an absence of normal sensation in the feet makes the patient feel as though walking on air-cushions or cotton.

The girdle pain, or sense of constriction about some part of the trunk, is not uncommon.

*Loss of Knee-jerk (Westphal's Symptom).*—This early and important symptom usually comes on gradually, and when associated with ocular symptoms and the lightning pains, is positively diagnostic.

*Ocular Symptoms*—These may appear very early in the disease, such as atrophy of the optic nerve resulting in impaired vision and frequently in blindness; ptosis—drooping of the eyelid; diplopia—double vision; the Argyll-Robertson pupil, a failure to contract to light, but contracts to accommodation, and the small contracted pupils, “pin points,” spinal myosis.

*Bladder Symptoms.*—In some cases the earliest symptom noted is difficulty in emptying the bladder, the desire to micturate being frequent but unsatisfactory; as a result, there is a partial retention of urine, often resulting in cystitis. Incontinence of urine appears late in the disease. Loss of sexual desire and impotency not infrequently appear in the early stage.

2. ATAXIC STAGE.—The symptoms of the preataxic stage persist, though new symptoms appear. The lightning-like pains, however, tend to become less severe and recur at longer intervals. The *ataxic symptoms*, the inco-ordination of movement or loss of the muscle sense, is characteristic. The symptoms are first noticed in walking or standing. Thus, if while standing the patient closes his eyes, he can not maintain the erect position without swaying, or if he places the feet close together and closes the eyes, he is in danger of toppling over. (Romberg's Symptom.) In walking, the body is bent slightly forward, the legs are farther apart than normal, and he lifts the advancing foot, throws it outward and forward with a jerk, and brings it down with a slap or stamp. The patient depends for assistance upon a cane, and, later, needs one in each hand. When sitting or lying the patient can not place his heel upon his knee or describe a circle with his foot.

The incoordination may extend to the upper extremities, and is recognized by inability of the patient to execute the finer movements of the hands; this is first noticed in dressing, the patient laboring awkwardly in buttoning the collar or other clothing, or in attempting to pick up a pin or other small objects. If asked to touch his nose or ear quickly, especially with the eyes shut, or to bring the tips of the fingers together, he finds it impossible.

*Sensory Symptoms.*—In addition to the pains already described, areas of hyperesthesia or anesthesia appear, especially in the lower extremities.

*Visceral crises* are characterized by paroxysmal pains in the stomach, larynx, kidney, heart, bladder, rectum, and genitals. The gastric and laryngeal being the most common.

A gastric crisis, the most frequent, consists of paroxysms of pain and vomiting, with hyperacidity. Hematemesis may also occur. Paroxysmal pains occur in the other viscera above named.

*Trophic Changes.*—Of these changes, the most serious are the joint lesions, and are known as Charcot's joints. The knees are the most often affected, and resemble those of arthritis deformans. Serous effusion rapi'dly takes place, the ligaments soften, the articular ends of the bones become absorbed, and dislocations or fractures sometimes follow. Suppuration of the joint may occur. Herpes, local sweating, and perforating ulcers may also occur. A favorite location for an ulcer is on the foot back. of the big toe, on the heel, and in the rectum. Loss of the nails and hair may occur. Atrophy of the muscles does not occur till late in the disease.

3. PARALYTIC STAGE.—This is the culmination of the second stage. The patient is unable to walk, and is confined to his bed or chair. There is a loss of control of the sphincters of the bladder and rectum, the muscles waste, bed-sores are present, and in some cases the patient is blind and deaf.

**Diagnosis.**—The diagnosis is not difficult, no other lesion having so many characteristic symptoms, assimilating tabes. Thus, the loss of the knee-jerk, the inco-ordination as shown in the walk, the lightning-like pains, the various crises, and the Argyll-Robertson pupil, make a group of symptoms that can not be mistaken.

**Prognosis and Course.**—After the disease has become well established and the cord becomes sclerotic, recovery is not to be expected. The disease, however, is slowly progressive, and the patient may live for years, dying of some intercurrent affection, as pneumonia, tuberculosis, etc. The preataxic stage may last for several years, especially if atrophy of the optic nerve occurs; the stage of ataxia in such cases being postponed indefinitely, or, if present, the further course may be arrested. The disease runs from ten to thirty years, though, in rare cases, it lasts but a few months or years.

**Treatment.**—The treatment for tabes has not been attended with very great success. Rest in bed in the early stage when the pains are intense is to be advised, and antikamnia, phenacetin, and in extreme cases morphia, are to be used to allay' the pain. The antisiphilitics have not resulted in much benefit. Hygienic measures that tend to improve the general health, with outdoor life in an equable climate, give the best results. Massage and electricity are of some benefit. When the stage of

loss of control of the bladder is reached, the patient should be instructed in the use of the catheter, and the means of keeping the instrument aseptic.

Echinacea may be tried as an agent to arrest degenerative processes.

Hydrastis, avena, phosphoric acid, camphor, chlorid of gold and sodium may be thoroughly tested according to the conditions presenting.

The diet should be generous and nutritious, as much food being allowed as can be digested.

### HEREDITARY ATAXIA.

**Synonym.**—Friedreich's Disease.

**Definition.**—A hereditary disease, appearing between the third and fifteenth years, and characterized by ataxia, impaired speech, nystagmus or paraplegia, and accompanied by changes in the lateral and posterior columns of the spinal cord. Fried-reich first described the disease in 1861.

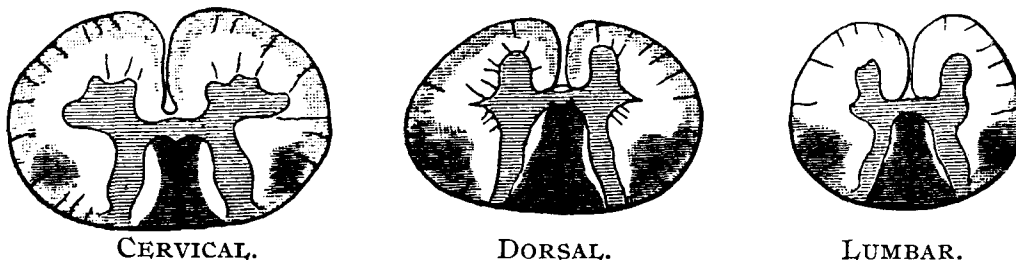


FIG. 41. SECTIONS OF SPINAL CORD FROM CASE OF FRIEDERICH'S ATAXIA. (*Pitt.*)

**Etiology.**—*Heredity.*—Although a single case may occur in a family, the occurrence of two or more in the same family is characteristic, and ten cases have been reported in a single family. There is generally a history of consanguinity, nervous disorders, inebriety, or nervous irritability, and is probably due to defective or feeble development. The infectious fevers have preceded the disease, though most likely a coincidence rather than a possible cause.

**Pathology.**—The most characteristic lesion is the diminution in size of the spinal cord. There is extensive sclerosis in the posterior and lateral, columns of the cord, and degeneration to slight extent in the anterior columns. Extending upwards the sclerosis may involve the medulla.

**Symptoms.**—The first ataxic symptoms develop in the lower extremities, though the gait is somewhat different from locomotor ataxia. There is more of a swaying, staggering, or irregular gait, and less of a stamping character. When the upper extremities are involved, the movements are irregular and jerky, resembling chorea. Irregular movements may occur in arms or legs even when the patient is at rest. There may also be irregular swaying of the head.

Impairment of speech early develops. At first it may be difficulty in enunciation, as in stuttering, or a syllable or word is missed, until finally an unintelligible mumble results. Although the face is more or less expressionless, the intellect remains unaffected, though late in the disease there may be mental impairment. Romberg's symptom is generally present, and a peculiar deformity of the foot develops, known as the "pes cavus," a stumpy foot with the arch exaggerated, and the toes extended, the great toe being abnormally prominent. There may also be deformity of the hand.

Spinal curvature may also follow. The sphincters are not involved; there are no trophic changes in skin or joints, no optic neuritis, nor crises. Late in the disease paralysis appears.

**Diagnosis.**—Usually there is no difficulty in recognizing the disease, there generally being two or more cases in the same family; the age of the patient, between three and fifteen, the irregular shuffling gait, the deformity of the foot, and the imperfect speech, being characteristic.

**Prognosis.**—This is unfavorable as to cure, though the life may be prolonged twenty, thirty, or more years.

**Treatment.**—But little can be hoped for in treating this disease, other than in maintaining the general health. Hygienic measures, fresh air, and a liberal diet are to be secured.

## SPASTIC PARAPLEGIA.

**Synonyms.**—Erb's Palsy; Primary Lateral Sclerosis; Spastic Spinal Paralysis.

**Definition.**—A chronic disease of the spinal cord, due to sclerosis of the descending fibers of the crossed pyramidal tracts, and characterized by loss of power, contractures, exaggerated reflexes, and a peculiar gait.

**Etiology.**—The disease generally occurs in neurotic families and between the ages of twenty-five and forty, males being more frequently affected. Sexual excesses, syphilis, exposure, traumatism, and lead-poisoning have been looked upon as causal factors.

**Pathology.**—Sclerotic changes are found in the crossed pyramidal tracts. In primary lesions of the brain, the anterior median columns may be involved in the sclerotic process. The axis-cylinders of the nerve-fibers may be destroyed and disappear.

**Symptoms.**—The disease comes on slowly, the first evidence of any abnormal condition, being an undue weariness on walking, attended by stiffness and rigidity of the muscles. As the disease progresses, it is with difficulty that the patient can walk, the limbs are drawn close together, the knees touch or overlap in walking, the toes drag, and when the patient steps on the ball of the foot, there is clonic spasm of the muscles. As a result of the contractions of the calf-muscles, the body is thrown forward and the patient walks with the crutches or cane thrown in advance of the body.

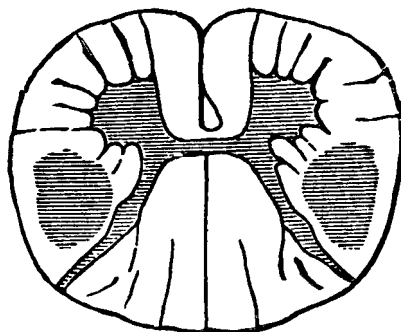


FIG. 42. POSITION OF LESION IN PRIMARY SPASTIC PARAPLEGIA.—  
(Taylor.)

When the patient is in the recumbent position, clonic spasms may cause the legs to twitch or jerk.

The power of locomotion becomes more difficult with the advance of the disease, and is finally lost.

The knee-jerk is excessive and ankle-clonus is easily obtained. In the advanced stage, if the disease has extended high up the cord, the upper extremities become involved.

Ocular symptoms are rare, and the sphincters are not involved till late in the disease. With the exception of dull pains or fleeting pains in the back, there is an absence of sensory symptoms.

*Of the secondary form*, the symptoms depend upon the lesion, whether one or both motor tracts are involved, and are bilateral or unilateral. As in the primary form, the symptoms generally appear gradually, there is weakness in the limbs, rigidity of the muscles, and increase of the reflexes, unless the lesion is total, when the reflexes are lost, and there is a flaccid paralysis.

*Spastic paraplegia of infants*, or congenital spastic paraplegia, is generally due to injury during child-birth, meningeal hemorrhage attending the manipulation of delivery. It may possibly be due, in some cases, to arrested development of the pyramidal tracts. The symptoms are not different from those already described, and may involve the upper as well as the lower extremities.

There may be imbecility, idiocy, or other mental defects.

*Hysterical spastic paraplegia* simulates the genuine type, though there is not the marked rigidity, especially upon extension. It develops suddenly and there is a history of hysterical attacks.

## **ATAXIC PARAPLEGIA.**

**Synonym.**—Progressive Spastic Ataxia.

**Definition.**—A sclerosis of the posterior and lateral columns of the cord, in which the posterior root-zones escape, and the reflexes are retained.



**Etiology.**—The etiology is not well understood. It occurs in males between the ages of thirty and fifty. It may follow exposure to cold or sexual excesses, or follow an injury.

**Pathology.**—This is a combined sclerosis of the posterior and lateral columns, beginning in the lumbar region. The nerve-roots, however are not involved, differing from locomotor ataxia.

**Symptoms.**—The symptoms embrace those of spastic paraplegia and locomotor ataxia, and develop slowly. All the symptoms of spastic paraplegia are present, though the rigidity is not so marked. In addition, in the early stage, the power of locomotion is somewhat impaired; especially if he attempts to turn quickly, he will stumble, or if he attempts to stand with the feet close together, his body will sway, and in the dark or with the eyes shut, he walks with difficulty. As the disease progresses the gait takes on the characteristics of locomotor ataxia, though the stamp is not so forcible.

The reflexes are increased, the knee-jerk being exaggerated, and ankle clonus is present.

Sensory symptoms, especially the lightning pains and crises, are absent, though a dull pain may be experienced in the sacral region.

The Argyll-Robertson pupil is generally absent, and optic atrophy rarely ever occurs.

The sphincters are not usually involved, though menstruation is sometimes difficult.

The ataxia may extend to the upper extremities, and in some cases mental disturbance is pronounced.

**Diagnosis.**—The diagnosis is generally easy, the inco-ordination on the one hand, and the exaggerated knee-jerk on the other, show the existence of a combined disorder.

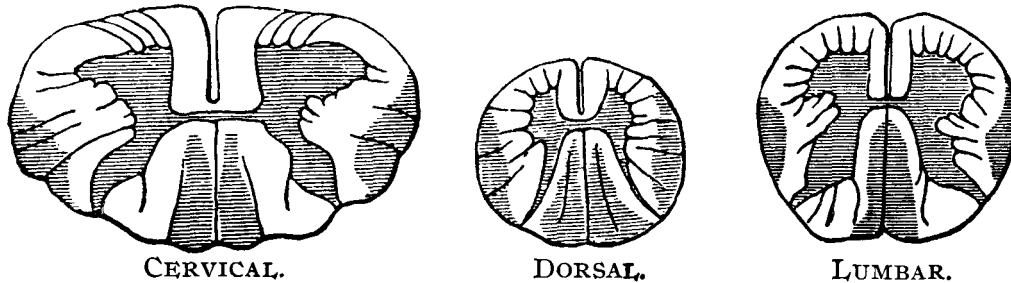


FIG 43. POSITION OF LESION OF ATAXIC PARAPLEGIA. (Taylor.)

**Prognosis.**—The disease being progressive, the prognosis is unfavorable as to cure, though the patient may live for years, and death generally results from intercurrent diseases. In some cases the disease is arrested for a time.

**Treatment.**—The treatment will be along the same line as that for locomotor ataxia.

### MULTIPLE SCLEROSIS.

**Synonyms.**—Insular Sclerosis; Disseminated Sclerosis; Cerebro-Spinal Sclerosis.

**Definition.**—A disease characterized by the development of chronic inflammatory spots, or sclerotic patches of the central nervous system, and may occur either in the brain or cord, or brain and cord combined.

**Etiology.**—The specific or exciting- cause is unknown. Among the predisposing- causes may be named age, it being a disease of early life, and rarely occurs after the age of forty. Hysteria, trauma, exposure, cold, and the infectious diseases may be causal factors. There is no preference for sex, if we except the neurotic state in certain females. Heredity is also a predisposing factor.

**Pathology.**—Prof. Taylor, an English authority, clearly describes the anatomical changes that take place. “The surface of the spinal cord, medulla oblongata, pons varolii, and base of the brain, present a number of irregular patches of pinkish-gray color, rather sharply outlined, and contrasting with the natural white color of the medulla, pons and crura. On section, the discoloration is found to extend inwards so far as to form deposits of a round or oval shape, ranging in size from

that of a pea to that of a hazel-nut, generally harder than that of the normal nervous tissue, and even leathery or cartilaginous, sometimes projecting above the level of the section, sometimes sunken below it. Recent patches are dark gray, older patches more yellowish gray, and less translucent. They affect the white matter more than the gray matter; thus in the spinal cord the greater part of the cornea is unaffected, and in the cerebrum they are best seen on section of the hemispheres, which are dotted with gray areas, and the walls of the lateral ventricles are often invaded. They are not frequent in the cerebellum; but the sclerosis may invade the olfactory bulbs, and the spinal and cranial nerve-roots.

“Under the microscope the outline of the patch or nodule is much less distinctly marked than it appears to the naked eye. The nodule consists chiefly of fibrous or finely fibrillated tissue, developed by overgrowth of the neuroglia; within this area the nerve fibers have lost their myelin sheaths, but great numbers of axis cylinders persist. Nerve-cells are very little involved unless late in the disease. There may be some thickening- of the vessels, but it is rarely pronounced.”

**Symptoms.**—The disease may begin insidiously, the patient simply noticing that he is growing weaker, tremulous, or spastic, or there may be a more rapid beginning, the patient behaving as though affected by hysteria. Thus the knees suddenly give way, or there is a sudden weakness of one arm or leg; this may soon improve temporarily, and then the same or another limb becomes paretic. When the disease is fully developed, however, there are three prominent and characteristic symptoms present:

1. *Tremor of muscles on attempting motion.*—The tremor is volitional, there being no abnormal movement when the patient is at rest; hence the term, intentional tremor. It is best noticed in the hand and arm in attempting to take hold of an object, the limb trembles or oscillates from side to side, or up and down, with regard to the object aimed at, the deviation often being quite marked. When standing, the body swings to and fro, and there is a nodding motion of the head. On attempting to walk, there is a trembling motion of the legs. The tendon reflexes are increased and ankle clonus may be present.

When the patient is quiet in bed or sitting in a chair with the back, head, and arms supported, there is an absence of tremors.

2. *Syllabic or Scanning Speech*.—In talking, the speech is at first only slow or drawling, but later each syllable is uttered slowly and distinctly, with a slight rising and falling inflection, and is known as scanning.

3. *Nystagmus*.—Oscillation of the eyeballs, when the eyes are fixed upon some object or when they are much turned to one side, being the result of voluntary movements as seen in the limbs. Optic-nerve atrophy is not infrequent. Sensory symptoms are not pronounced, and consist of tingling or numbness of the limbs and hyperesthetic areas that are transient in character. There is no atrophy of the muscles nor trophic changes. Vertigo is often present. The function of the bladder, rectum, and sexual organs may be retained, though involuntary expulsion or retention of urine is not uncommon.

There is generally impairment of the mental faculties, at first hysterical in character, but later maniacal tendencies may develop, or dementia result. Epileptiform or apoplectiform attacks may occur, though usually rare.

**Diagnosis.**—The diagnosis is usually easy after the disease is well established, the three characteristic symptoms, *tremors, syllabic or scanning speech, and nystagmus, being easily recognized.*

**Prognosis.**—The disease may extend over a period of years, from five to twenty, death occurring from some intercurrent disease. Death rarely occurs from a convulsion or apoplectiform attack. The prognosis as to cure is always unfavorable.

**Treatment.**—The treatment is mainly symptomatic, and along the same lines as other forms of sclerosis.

## **PROGRESSIVE MUSCULAR ATROPHY.**

**Synonyms.**—Poliomyelitis Anterior Chronica; Wasting Palsy.

**Definition.**—A chronic degeneration of the motor tract and characterized by a progressive wasting of the muscles. The disease is one of adult life, and occurs generally between the age of thirty and fifty years. It affects males more frequently than females.



FIG. 44. TWO VIEWS OF A HAND OF A PATIENT SUFFERING FROM PROGRESSIVE MUSCULAR ATROPHY. (*Ranney.*)

**Etiology.**—The cause is unknown, though quite a variety of causes have been mentioned by various writers, the most important being the following: Heredity, excessive muscular effort, exposure to cold, traumatic injuries of peripheral parts of the body, the *infections fevers*, typhoid fever, influenza, diphtheria, measles, rheumatism, etc., and excessive venery and masturbation.

**Pathology.**—A slow degeneration of the ganglionic cells resulting in their partial or complete destruction. “The neuroglia is excessively developed,” while the anterior nerve-roots passing from the horns are *atrophied*. There is an abnormal dilatation of the vessels of the cord, and sclerosis of the arterioles.

The muscles become shrunken and pale in color to the unaided eye, and the microscope “reveals a disappearance of the transverse striae in the fibrille,” while fat connective tissues replace the muscular fiber in the advanced stage. “As to the relation of the nervous changes to the muscular atrophy, the conspicuous symptoms of the disease, there is more than one possible explanation. As in bulbar palsy, according to one view, the atrophy of the anterior cornua, is primary, the result of chronic poliomyelitis anterior, the degeneration of the peripheral nerves and muscles being secondary to it.

“According to another view, the muscular atrophy is primary, possibly

due, as Friedreich sought to prove, to a myositis, followed by fatty metamorphosis of the sarcous substance and subsequent absorption of fat, or to a simple primary fatty meta-phorosis." (Tyson.)

**Symptoms.**—The disease comes on insidiously, the characteristic feature of which is the development of extensive and progressive atrophy of certain muscles. The disease generally begins in the upper extremities, though the lower may be involved first.

The patient's attention is called to the wasting of certain muscles, by the loss of power in the affected part, this being usually manifested in the hand and shoulder. Beginning with the short muscles of the thumb, there is soon flattening of the ball and the simultaneous wasting of the interossei results in depressions between the metacarpal bones. On account of a predominance of power in the extensors and abductors of the hand, the "ape-hand" becomes characteristic.

As progressive changes take place and the interossei become more affected, the hand assumes greater deformity and we have the "*claw-hand*." With atrophy of the lumbricales, there is flattening of the palms. From the hand the wasting attacks the muscles of the forearm and thence to the arm and shoulder. "In the forearm, the extensor muscles undergo atrophy more frequently than the flexors."

When the shoulder becomes involved, the deltoid becomes flattened and the movements of the arm are more or less impaired. The atrophy generally attacks the right hand before involving the left. With marked atrophy of the arms, the process extends to the muscles of the scapula and trunk.

Late in the disease, the muscles of the lower extremities become involved, the flexor muscles being the first to be attacked.

When the diaphragm undergoes atrophy—a rare case—respiration becomes greatly impaired.

In proportion to the wasting, the electrical excitability fails, faradic and galvanic diminishing together. The reflexes are generally lost, the knee-jerk remaining longest since the lower extremities are among the last to be involved.

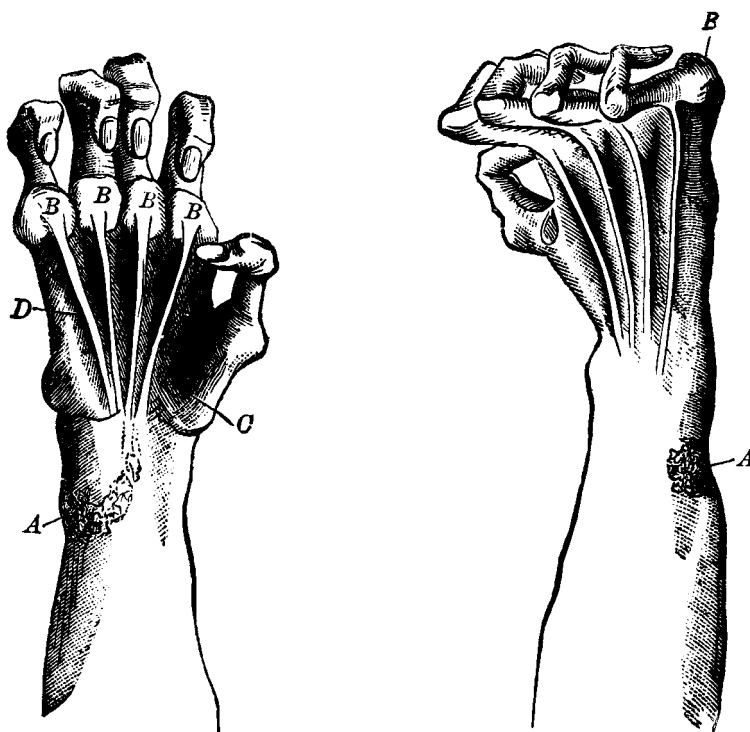


FIG. 45. POSITION OF HANDS AND LINGERS IN ULNAR PARALYSIS OF LONG STANDING: BIRD-CLAW HAND; "MAIN EN GRIFFE."  
—(Duchenne.)—(Tyson.)

A. A. Wound of the ulner nerve. B. B. B. B. B. Ends of the metacarpal bones. D. Tendons of the flexor sublimis digitorum. C. Muscles of the ball of the thumb.

Fibrillary contractions are constant and characteristic symptoms. Sensation is not impaired, and the bladder and rectum are not involved.

The disease is slow in its course, the patient frequently dying of some intercurrent disease, as pneumonia, tuberculosis, etc.

**Diagnosis.**— "Progressive muscular atrophy has to be distinguished from all other diseases accompanied by atrophy of muscles. The important feature is the slow commencement by atrophy and weakness together, without pain, spasm, or sensory troubles. This distinguishes it from *tumor* and *meningitis*, which may cause muscular atrophy. In acute *poliomyelitis* of children and adults, the history is completely different. In the typical cases of *amyotrophic lateral sclerosis* the course is more rapid, and the reflexes are early increased. When the atrophy

affects the hand alone, the deformity resembles somewhat the result of lesion of the ulnar nerve; but in this last the ulnar half of the hand is more decidedly affected (the radial lumbricales being supplied by the media nerve), and anesthesia and trophic changes occur; in traumatic cases, the history of injury will, of course, help. *Lead paralysis* is recognized by the extensors being first, and generally alone, affected; by the blue line on the gum, the detection of lead in the urine, and perhaps by the occupation, and preceding attacks of colic. *Multiple neuritis* is distinguished by the rapid onset, the wide extent of the parts affected, the numbness of anesthesia, and the tenderness of the muscles. *Pseudo-hypertrophic paralysis* must be recognized by the enlargement of muscles, and its development in childhood; *idiopathic muscular atrophy*, by the atrophy beginning differently; e. g., in the face or legs." Taylor.

**Prognosis.**—This is unfavorable as to cure, though in rare cases the progress of the atrophy is arrested.

**Treatment.**—Medication has but little effect upon the progress of the disease. The patient's general health will be improved by the use of the bitter tonics and by hygienic and dietetic measures. Arsenic and strychnia have been extolled as influencing the diseased processes, though this is doubtful. Massage and electricity should be faithfully tried. When possible, the patient should seek an equable climate where he could be much in the open air- and sunshine, and free himself from mental worry. Gradually, but surely, however, the disease advances, finally resulting in death.

## **BULBAR PARALYSIS.**

**Synonyms.**—Glosso-labio-laryngeal Paralysis; Duchenne's Disease.

**Definition.**—An acute or chronic disease of middle age, due to involvement of the motor nuclei of the medulla oblongata, and characterized by paralysis of the lips, tongue, larynx, and pharynx, resulting in impairment of speech, phonation, mastication, and deglutition. Two forms of bulbar paralysis are recognized, the acute and the chronic.

**Etiology.**—Bulbar paralysis is a disease of middle and advanced life,



between the ages of forty-five and seventy, and is more frequent in men than in women. It may be due to hemorrhage, embolism or softening, exposure to cold, trauma, or diphtheria. Lead-poisoning and syphilis have also been named as possible causes.

**Pathology.**—The most pronounced changes are found in the nerve-roots proceeding from the medulla; those of the hypo-glossal, glossopharyngeal, vagus, facial, the motor nucleus of the fifth, and spinal accessory, showing distinct atrophy. They are of a gray or grayish-red color, and much shrunken. The microscope reveals degeneration of the nuclei or complete disappearance of the nerve-cells, some increase of the neuroglia, and thickening of the vessel-walls.

Muscular atrophy is limited to the lips, tongue, palate, and muscles of the larynx, and if spinal atrophy be associated, the muscles of the neck, shoulders, or arms are involved in the process.

**Symptoms.**—The onset is sudden, and may be attended by nausea and vomiting. Articulation is indistinct, especially in the use of the linguals, labials, and dentals: l, m, p, b, t, d, etc.

The lower lip being affected, it drops and there is dribbling of saliva. Deglutition is difficult, with frequent attacks of choking. The mind is impaired, the patient becomes quite emotional and neurasthenic. There may be hemiplegia or crossed facial paralysis.

*Chronic Form.*—The symptoms of this form are very gradual in their development, the first noticeable feature being a difficulty in controlling the tongue and articulating sounds that depend upon it,—thus words containing e, r, s, l, k, g, d, t, n, and sh. As the disease progresses, the paralysis of the tongue becomes more pronounced; it can not be protruded, and lies on the floor of the mouth. Next in order, the lips become involved, and the labials p, b, f, v, m, and o are pronounced with difficulty, and blowing- and whistling are impossible. The lips atrophy and drop, exposing the teeth, and the saliva dribbles away.

Following the lips, the palate becomes paralyzed, and liquids are regurgitated through the nose, and the voice becomes nasal. Increased difficulty in swallowing is now experienced, for in addition to the inability of the tongue to carry the bolus backward, there is paralysis of the pharynx. In time the laryngeal muscles become involved, resulting

in hoarseness and, finally complete aphonia. In swallowing, food is apt to enter the larynx, producing attacks of choking, and not infrequently particles find their way into the bronchi or lung, giving rise to bronchitis and deglutition pneumonia. At the last the patient's condition is most deplorable and disgusting, for, with mind impaired, he resembles a "driveling idiot." He can not talk, swallow, or close his mouth, and the saliva and particles of food dribble away.

There are no sensory symptoms, and the taste remains normal.

**Diagnosis.**—This is generally quite easy, the above symptoms being so striking and characteristic that no one need make a mistake in the diagnosis.

**Prognosis.**—This is unfavorable, though the disease may last for several years. Death usually results from exhaustion, inanition, choking, inspiration pneumonia, or circulatory disturbances.

**Treatment.**—Since the disease is incurable the treatment will be symptomatic. The general health is to be maintained and the patient instructed in the introduction of the esophageal tube, for feeding will have to be conducted in this way in the last stage of the disease. Electricity is of doubtful utility.

## **SYRINGOMYELIA.**

**Synonym.**—Gliosis Spinalis.

**Definition.**—A disease of the spinal cord due to a new growth in the gray substance about the central canal, which results in the formation of cavities.

**Etiology.**—Nothing definite is known as to the exciting cause, though trauma, syphilis, and the infectious diseases, especially typhoid fever, have been suggested as etiological factors. It occurs between the ages of fifteen and thirty years.

**Pathology.**—The disease is generally regarded as a gliosis with degeneration of the central portion of the cord, resulting in the formation of small cavities of various shapes. They are more frequently

found in the dorsal and cervical regions, though the entire length of the cord may be involved. The posterior and posterolateral tracts are the most often involved; though generally independent of the central canal of the cord, they usually communicate with it.

“The wall of these cavities is generally composed of a firm fibrous tissue, or of myxomatous tissue. Its inner surface may or may not be lined with epithelial cells of the cylindrical variety. They usually contain a serous or hemorrhagic fluid, and occasionally a hyaline material.” (Ranney.)

**Symptoms.**—There are three characteristic symptom groups:

1. *Modified sensibility*, especially to *pain, temperature*, and to a certain degree, *touch*.
2. *Progressive muscular atrophy*, with paralysis.
3. *Trophic* disturbance, in the skin, muscles, bones, or joints. With these symptoms, there may be associated, as the disease progresses, spastic paraplegia, symptoms of transverse myelitis, or those due to involvement of the lateral, posterior, or in fact all the columns of the spinal cord.

The symptoms develop gradually, and usually make their appearance about the period of adolescence. As the cavity is most frequently located in the cervico-thoracic region, the arms and neck are the first to be affected. There are aching pains in the neck and arms, with numbness in the hands and loss of the pain and temperature senses. Weakness and atrophy of the muscles follow, attended by trophic changes, such as ulcers, brittleness of the nails, and painless felons.

Later, with the extension of the disease, spastic paresis of the legs follows, and the bladder and the rectum may be involved. From the involvement of the spinal muscles, curvature of the spine often accompanies the disease. With the progress of the disease the symptoms become more general, larger areas of modified sensibility take place, and more extensive trophic changes are noted. Charcot's joint, shoulder, or elbow, edema of the fingers, bullae, ulcers, felons, loss of nails, and brittleness of bones are noted.

If the posterior columns are involved, incoordination of the muscles

occurs similar to that of locomotor ataxia.

The reflexes vary, and may be either lost or exaggerated. Where the disease extends to the medulla, the cranial nerves become involved, giving rise to bulbar symptoms.

**Diagnosis.**—The three characteristic and typical features— modified sensibility to pain and temperature, extreme muscular atrophy, with paralysis and trophic disturbances, to which may be added spasticity of the lower extremities—make the diagnosis comparatively easy.

**Prognosis.**—Although the disease may extend over a period of years, the prognosis is always bad, though death usually results from intercurrent diseases.

**Treatment.**—The treatment can only be symptomatic. The means to preserve the general health, climatic, hygienic, and dietetic, must be observed and conditions met as they arise.

### **AMYOTROPIC LATERAL SCLEROSIS.**

**Synonyms.**—Charcot's Disease; Wasting Palsy; Spinal Muscular Atrophy.

**Definition.**—A degeneration of the motor tract of the cord, and attended by progressive muscular atrophy, loss of power, and spastic paraplegia.

**Etiology.**—Charcot's disease is of unknown etiology, occurring between the ages of twenty-five and fifty, usually after thirty, and is found more frequently in women than in men. It is closely allied to progressive muscular atrophy.

**Pathology.**— “A sclerosis of the crossed pyramidal tracts in the two lateral columns, and the direct pyramidal tracts in the anterior columns, is essential to the morbid anatomy in a typical case. As important is atrophy of the corresponding large ganglion cells in the anterior cornua and medulla. The degeneration has been traced in the pyramidal tracts from the sacral cord upward to the pyramids in the medulla oblongata, sometimes even through the pons and crura into the internal capsule

and center convolutions, in which, too, the large ganglion cells have been found atrophied." (Tyson.)

**Symptoms.**—Charcot's division of this disease into three stages is recognized in all typical cases, and are as follows:

1. In the first stage the upper extremities only are involved.
2. In the second stage the lower extremities are attacked.
3. In the third stage, bulbar symptoms are present, the medulla being attacked.

There rarely occur atypical cases, in which the third stage becomes the first, the medulla being first attacked, the disease gradually extending downwards; or the lower extremities may be first involved, the disease gradually extending upwards.

*First Stage.*—This stage begins with weakness of the upper arms and a gradual wasting of the muscles, and fibrillary twitching, to be followed by paralysis. The atrophy soon spreads, and may involve the whole upper extremity, though the characteristic deformity of the disease is seen in the hand and wrist, and is known as the "claw-hand," the rigidity being due to contraction of the atrophied muscles. "The upper arm lies close to the chest, the forearm is semi-flexed and pronated, whilst the wrist is strongly flexed, and the fingers are bent into the palm."

The tendon reflexes are exaggerated, as may be demonstrated by striking the tendons of the biceps and triceps, or the lower ends of the ulna and radius.

Occasionally the muscles of the neck and jaw share in the spasm, and may remain rigid for a long time, but this gradually disappears, with marked contractions of the hand.

The duration of the first stage varies from four to twelve months.

*Second Stage.*—After the disease has progressed for some months, the muscles of the lower limbs become involved, and tonic or clonic spasms may develop. The knee-jerk is increased, and ankle clonus can be

obtained. Rigidity and contractions are seen in some of the muscles, though sooner or later this gives way to atrophy and fibrillary twitchings.

Sensation is usually not disturbed, nor is there loss of the use of the sphincters.

*Third Stage.*—As soon as the disease extends to the medulla the third stage is ushered in, and bulbar symptoms appear. Paralysis of the lower part of the face occurs, the mouth is not completely closed, the saliva dribbles and articulation and deglutition are greatly impaired.

When paralysis of the pneumogastric nerve occurs, serious disturbances of the circulation and respiration follow, and sometimes cause death.

The mental faculties are but slightly affected, though the emotions are often perverted, the patient laughing or crying without cause.

**Diagnosis.**—The diagnosis is readily made if we keep in mind the characteristic stages and symptoms of each. Beginning with weakness in the arms, there soon follows atrophy of the muscles, and paralysis with contractions, giving the “claw-hand;” this in turn is followed by spastic paralysis of the lower extremities, which, in turn, is followed by the bulbar symptoms.

**Prognosis.**—The prognosis is unfavorable both as to life and improvement of the wasted and paralyzed muscles. Death generally follows in from one to three years, due to impairment of the circulatory and respiratory functions, the result of an extension of the morbid changes to the nuclei of the medulla.

**Treatment.**—But very little can be suggested in the way of cure, or even to retarding its progress. Ranney claims to have relieved the contractions of the muscles and checked the progress of the disease for many months, by employing static sparks daily to the spine and limbs, while Gowers favors the injection of nitrate of strychnia into the muscles, beginning with the minimum and rapidly increasing to the maximum dose. Massage will afford some relief to the contracted muscles. Good hygienic surroundings should be encouraged, and the patient rendered as comfortable as possible.

## COMPRESSION OF THE SPINAL CORD.

**Synonym.**—Compression Myelitis.

**Definition.**—An impairment of function of the cord, due to gradual compression.

**Etiology.**—According to Taylor, of Guy's Hospital, the most common cause of compression of the cord is caries of the spine, not, as he says, from “angular curvature,” which the caries produces, but from the inflammatory or caseous products, which form between the diseased bone and the external surface of the dura mater, destroying the posterior common ligament, and setting up an external pachymeningitis.

Of less frequent occurrence, acting as causes, may be named tumors, carcinomatous and sarcomatous growths, and aneurisms.

**Pathology.**—The alteration in the shape of the cord depends upon the amount of compression. The cord is flattened and may be narrowed, to one-half or one-third its natural diameter; myelitis follows, and the cord, in the early stage, shows some en-gorgement and softening, but later sclerosis follows, attended by “degeneration of the posterior columns above the lesion, and in the pyramidal tracts below the lesion.”

The microscopical changes are those peculiar to myelitis. While the nerve-roots will show more or less impairment, many nerve-fibers will remain intact.

**Symptoms.**—In typical cases there are two groups of symptoms, one due to pressure upon the nerve-roots, the other to involvement of the cord itself. Pain, neuralgic in character, and darting along the course of the nerve, is characteristic of the first. There will be areas of anesthesia, “*anesthesia dolorosa*,” and muscular spasms, followed by paralysis, loss of the reflexes, and atrophy of the muscles.

Areas of *hyperesthesia* frequently accompany the anesthesia. Occasionally, trophic disorders of the skin are present, zona, bullæ, or eschars.

“The symptoms due to direct compression of the cord are those with

which we are familiar in transverse lesions: paralysis, anesthesia, or other modifications of sensation, increased reflexes, often some vesical trouble, and generally spastic rigidity of the paralyzed muscles. The relation of anesthesia to paralysis varies much in different cases, and in the same case at different times. Loss of motion is, as a rule, the most prominent symptom, and anesthesia may be entirely absent. The activity of the reflexes is often in excess of the motor paralysis. It is another important feature, when the compression results from caries, that recovery may take place completely, or improvement may again be followed by relapse. The site of the compression, of course, determines some differences in the symptoms. Compression *limited to one side* will cause the pains to be unilateral, and the paralysis may be on the same side, the anesthesia on the opposite, as has been stated to be the result of strictly one-sided lesions. Cervical compression may be accompanied by alterations of the pupil, especially dilatation from irritation of the ciliospinal center, by cough and dyspnea, dysphagia, vomiting, or very slow pulse. The distribution of the paralysis is also sometimes striking: all four limbs may be paralyzed, the upper limbs being wasted, with diminished reflexes, as a result of compression of the nerve-roots or their centers. But the arms may be paralyzed as a result of compression above the origin of their nerves, and the muscles will then preserve their volume and their electrical reactions, while the reflexes are increased. In some such cases the legs remain unaffected. The distinctive features of compression of the lumbar region are paralysis, with flaccidity, wasting, diminution of the reflexes, paralysis of the sphincters, and tendency to bed-sores." (Taylor.) '

**Diagnosis.**—If caries of the vertebra be present and spinal symptoms appear within a few years after the removal of cancer of the breast, the diagnosis will be comparatively easy; if, however, compression of the cord occurs from the exudate before the evidence of caries appears, the diagnosis may be very difficult, especially if the root symptoms be absent.

**Prognosis.**—The prognosis is more favorable when due to caries than from other causes, though years may elapse before a cure is effected.

**Treatment.**—When, due to caries, some one of the many devices now in use should be selected to produce extension. It may be suspension, a favorite method in use a few years ago, or a plaster cast, or some mechanical appliance that secures hyperextension while in the



recumbent state. Mere rest in bed has proven of much benefit, where it can be maintained for weeks or months. Where the disease is due to other causes, the treatment is generally unavailing. In general the treatment will be that used in tuberculosis.

The patient should be much in the open air, the diet should be easily digested, but nourishing. Arsenic, Howe's acid solution of iron, nuxvomica, hydrastin, echinacea, and remedies of like character, will be used. Massage and electricity should be tried. Work along the line of official treatment, often does more for the patient than medication.

### **III. DISEASES OF THE BRAIN AND ITS MENINGES.**

#### **DISEASES OF THE DURA MATER.**

##### **EXTERNAL PACHYMENINGITIS**

**Synonym.**—Simple Meningitis.

**Definition.**—An inflammation of the external layer of the dura mater of the brain.

**Etiology.**—The most frequent cause is fracture of the skull, with its consequent extravasation of blood. Next in order is caries of the middle ear or of the frontal or ethmoidal sinuses. Syphilis, erysipelas, or carbuncle may also be the exciting-cause.

**Pathology.**—The dura is thickened, due to infiltration of blood and pus, and these products, collecting between the dura and the skull, are apt to give rise to localized abscess. Where due to syphilis, there is generally marked thickening of the inner table and much pus between the dura and the bone. Where there is an infiltration of pus between the two layers of the dura, dura-arachnitis may follow.

**Symptoms.**—The symptoms are not well defined in mild cases, and are apt to be overlooked, headache being the only evidence of the lesion. In the more severe types there will be a chill, followed by fever, headache, more or less dullness, which in turn is followed by stupor. Paralysis and

convulsions occur in rare cases. Should rigors occur, they would suggest pyemia. There is more or less tenderness over the part affected.

**Treatment.**—The treatment is mostly surgical, and consists in trephining for the liberation of pus. In the milder cases, counter-irritation, and the properly selected sedative should be used. When due to syphilis, echinacea, Donovan's solution, berberis aquifolium, corydalis formosa, or stillingia will be found useful as well as potassium iodid.

## INTERNAL PACHYMENINGITIS.

**Synonyms.**—Hemorrhagic Pachymeningitis; Hematoma of the Dura Mater.

**Definition.**—Extravasation of blood into the inner membrane of the dura mater.

**Etiology.**—This is a secondary lesion, following inflammation of the external membrane of the dura mater, or occurring as the result of tuberculosis, Bright's disease, leukemia, erysipelas, pyemia, puerperal fever, syphilis, or any other disease causing degeneration of the blood-vessels.

**Pathology.**—On the inner surface of the dura, a fibrous exudate is found which develops into fibrous connective tissue in which one or more slight, rarely profuse, hemorrhages occur, and as a result a quantity of blood accumulates between the dura and the arachnoid; hence the term hematoma is applied to it. A favorite location is beneath the parietal bone. It may be bilateral, though usually but one side is affected.

**Symptoms.**—These are not characteristic; in fact, they may be so obscured by the primary disease giving rise to it as to pass unnoticed. Pressure symptoms are frequently present, and monoplegia or hemiplegia may occur. Headache is perhaps the most constant symptom, and convulsions are not unfrequent.

**Diagnosis.**—This is extremely difficult, the clinical symptoms not being sufficiently constant to suggest the lesion.

**Prognosis.**—This is unfavorable, there always being a tendency to meningitis and danger from thrombosis.

**Treatment.**—This will always be along the line of meningitis, and when a large hemorrhage is recognized, surgical measures will be necessary.

## **DISEASES OF THE PIA MATER.**

### **ACUTE LEPTOMENINGITIS.**

**Definition.**—An inflammation of the pia and arachnoid membranes of the brain.

**Etiology.**—This is generally, if not always, a secondary lesion, the most frequent primary lesion being disease of the sinuses, the nose, or the middle ear. Being strictly an infectious disease, a great variety of bacteria have been found, besides the staphylo-cocci and streptococci, those peculiar to the infectious fevers in general, but especially pneumonia, influenza, erysipelas, enteric fever, measles, scarlet fever, endocarditis, septicemia, diphtheria, and tuberculosis. Of the more chronic diseases that may precede it, may be mentioned Bright's disease, rheumatism, arteriosclerosis, and gout.

The disease is found more frequently between the ages of thirty and fifty, though when due to tuberculosis it is most common in children.

**Pathology.**—The locality, extent, and degree of tissue-changes vary; thus if due to middle-ear disease, the lesion will be unilateral and over the temporo-sphenoidal lobe; if due to pneumonia, endocarditis, or any infectious disease, the process will generally be bilateral and limited to the cortex, while at times the base alone is involved. The exudate varies from a fibrous exudate to a purulent or hemorrhagic infiltrate. The ventricles may be dilated, especially in children, and contain a turbulent fluid. When the exudate becomes purulent, the various microorganisms peculiar to septic processes and infectious diseases, are found in the fluid.

**Symptoms.**—These naturally vary, depending upon the location of the lesion, the extent of the inflammatory process, and the producing cause.

When due to middle-ear lesions, the symptoms are those of well-defined meningitis, either tubercular or those of the epidemic form.

If the meningitis be a complication of any of the severe infectious diseases, the symptoms of the primary disease may obscure those of the local affection.

In the greater number of cases, however, there will be a train of symptoms quite characteristic. Headache, severe and protracted, is always present; delirium, followed by coma,, soon appears in many cases. The pulse is often slow, though in children it may be very rapid. Vomiting is present in a great many cases.

Photosbacia, intolerance to sound or irritation of the head, are seen in all severe types. Where the base is involved, the cranial nerves are affected, and strabismus, ptosis, or facial paralysis follow, and if the fifth nerve be involved, trophic changes are common symptoms. As the disease progresses, the spine becomes rigid, there is great retraction of the head, and opisthotonos may be pronounced. Convulsions, in children of nervous temperament, are apt to occur.

The suppurative process is announced by chills, and fever of an irregular or septic type.

**Diagnosis.**—The primary affection may so obscure the meningeal lesion for a few days as to make an early diagnosis quite difficult, if not impossible; sooner or later, however, characteristic symptoms develop, and there should be few mistakes as to the disease.

We must differentiate the tubercular from the non-tubercular form, and, if attention be paid to the following well-marked characteristics of the tubercular type, the difference can be readily seen. In tubercular meningitis there is a *history* of tuberculosis and the *forming* stage is of *long* duration. There is no *apparent* cause for the meningeal lesion. Generally there is the presence of *tuberculosis* in the lungs, and the meningeal lesion runs a more protracted course.

**Prognosis.**—Although a grave disease, it is not necessarily fatal.

**Treatment.**—The treatment does not differ materially from that used in epidemic cerebro-spinal meningitis. When due to middle-ear disease,

operative measures afford some relief, and pave the way to recovery.

## **CEREBRAL LOCALIZATION.**

That man is the most wonderful piece of mechanism in the universe is conceded by all, and each individual part of this composite being must be thoroughly understood by the medical man, if he is to successfully combat the countless functional and organic lesions that daily beset him.

Life is too short, however, for any one man to master all, or even one important part, of every organ. The medical man of the present day has come to understand this as never before, and has divided his labor by selecting some special part for his life-work. Thus we have the specialist on the circulatory system, the respiratory apparatus, the blood, the skin, the eye, the ear, etc.

What is true of the body at large, is especially true of the brain. The study of this composite organ during the last few years has completely revolutionized the theories of this most wonderful organ, the seat of all the higher mental faculties and the director of all the voluntary motions, which distinguishes man from the entire animal world, and places him in a class by himself.

Much has been learned by vivisection on the lower animals, much by accidents and disease of the brain, and much by the pathologist after death; and from the combined experience of the experimentalist, the physiologist, the pathologist, and the clinician, the architecture and physiology of the brain has made wonderful advances during the last few years.

The study of the brain is a life-study, and in a work on general practice we can but glance at some of the more important lesions, referring the student for further research to special works upon the brain and nervous system. The general practitioner, however, should understand that the brain must be regarded as a *composite organ*; that each of its parts has some special function that, to some extent, is independent of its fellow-member.

Thus one part, the *medulla oblongata*, is essential to vital processes, and the slightest injury, even the thrust of a needle, may result in

instant death. Another presides over the various movements of the body, and an injury or disease of this part is followed by paralysis of motion; or it may be that the part that presides over sight, or hearing, or taste, or smell is involved, and the function of the eye, or the ear, or taste, or smell is impaired.

Through the researches of Meynert, Luciani, Charcot, Exner, Nothnagle, Ferrier, Flechsig, Wernicke, Munk, and others, the topography of the brain has been so well outlined that the skilled neurologist of to-day can, by carefully noting the symptoms, locate the diseased condition, and not infrequently cure the disease by removing the cause by operative measures. Thus epilepsy has been overcome, when due to pressure upon some part, by removing the source of irritation. Aphasia, or loss of speech, has been restored by opening and draining an abscess of the third frontal convolution, the seat of speech centers.

The topography of the cerebral cortex is now so well understood that it is possible to map, with great accuracy, the various regions in which motor impulses originate.

The cerebrum is divided into two hemispheres, each of which consists of five lobes,—the *frontal*, the *parietal*, the *occipital*, the *temporo-sphenoidal*, and the *central*, or island of Reil. These are divided or separated by three fissures,—the *fissure of Rolando*, the *fissure of Sylvias*, and the *parieto-occipital fissure*.

The frontal lobe consists of *three convolutions*,—the *superior frontal*, the *middle frontal*, the *inferior frontal*, and the *ascending frontal*.

The parietal lobe consists of three convolutions,—the *ascending parietal* (posterior central) convolution, the *superior parietal*, and the *inferior parietal*.

The occipital lobe consists of three convolutions,—the *superior*, the *middle*, and the *inferior* convolutions.

The temporo-sphenoidal lobe consists of three convolutions,— the *superior*, the *middle*, and the *inferior* convolutions.

The central lobe, or island of Reil, consists of six.



FIG. 46. A diagram designed to illustrate the probable functions of different areas of the cerebral cortex.—(Ranney.)

“The surface of the brain is the seat of all conscious mental action. It is the receptacle of all the impressions made upon the organs of sight, smell, taste, hearing, and the tactile organs of the skin. Here, and only here, do such impressions become transformed into conscious appreciation of external objects.”

By examining diagrams, one gets a clearer idea of the areas that control certain functions. Thus the higher mental faculties, reason, will, judgment, etc., are the result of cell activity in the frontal lobe, while the power of speech resides in the inferior frontal convolution of the same lobe. A part of the frontal and parietal (central convolution) presides over all motor acts of the limbs and body. The upper parts control the legs chiefly, the middle part governs the hand and arm, while the lower part presides over the complex movements of the tongue and lips necessary to speech.

The parietal lobes, not occupied by special centers of motion, are centers of touch, pain, and temperature.

The temporal lobes preside over sounds, odors, and taste.

The occipital lobes preside over sight.

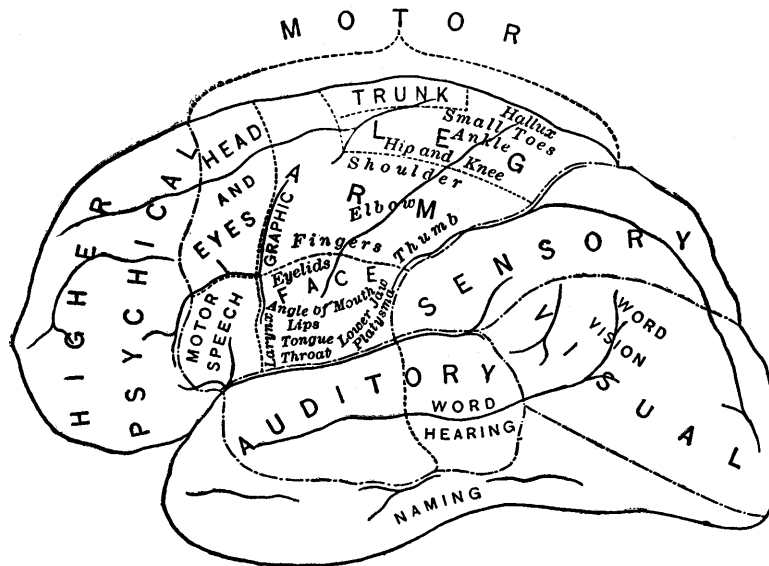


FIG. 47. Diagram of the cortical centers and areas of representation on the lateral aspect of the hemiserebrum (Mills). (Anders.)

## AFFECTIONS OF THE CEREBRAL BLOOD-VESSELS.

### HYPEREMIA.

**Definition.**—Hyperemia of the brain is an abnormal amount of blood in the cerebral capillaries, and may be active or passive.

*Active Hyperemia.*—This probably attends any increase in the general circulation, and is found in greater or less degree in all inflammatory diseases. It may follow prolonged exposure to the sun, to the ingestion of certain drugs, notably nitroglycerin, amyl nitrate, and alcohol. Excessive brain-work will give rise to cerebral hyperemia, while not infrequently the cause is reflex. The infections that are attended by restlessness, delirium, or other cerebral disturbances, also give rise to it.

*Passive Hyperemia.*—Any cause that obstructs the cerebral sinuses or veins, retarding the free flow of blood from the brain, will give rise to passive hyperemia. Of the most common are pressure upon the superior vena cava and jugular veins by tumors or aneurisms, though valvular



lesions of the right heart, emphysema, and asthma bear an almost equal share as causal factors.

**Pathology.**—There are no characteristic changes from hyperemia, either active or passive. In the active form there may be a slight increase in the puncta vasculosa and a darkening of the white substance, while the veins and sinuses may be filled with dark blood in passive congestions, and there is more or less edema of the tissues.

**Symptoms.**—*Active hyperemia* is characterized by the flushed face, bright eyes, contracted pupils, and throbbing of the carotids. There is more or less headache of a throbbing character; the patient is irritable and restless, with more or less insomnia. In the rare cases there will be delirium or convulsions. There is hyperesthesia of the special senses.

In *passive hyperemia* there is dullness of the intellect, the pupils are dilated, and the patient lies passive. If there be headache, it is dull in character. The face is pale or dusky in color. The pulse is small, the tissues relaxed, and the extremities are cold.

**Treatment.**—In gelsemium we have a specific for active hyperemia of the brain. It acts nicely with either aconite or veratrum, or it may be given alone. Add from twenty to sixty drops of the specific tincture (according to the age of the patient and the irritability of the nervous system) to four ounces of water, and give a teaspoonful every one, two, or three hours, as may be indicated.

*Passiflora.*—In nervous children, with insomnia as the chief symptom, add from one to four drams of passiflora to four ounces of water, and give a teaspoonful every one or two hours. When very wakeful, we may give the agent in still larger doses. Thus—

Passiflora.

Simple Syrup

1 ounce each.

Sig. A teaspoonful every two or three hours.

*Rhus Tox.*—Where the child is restless and there is a sharp stroke to the pulse, and the child startles at the slightest sounds, or, if asleep, awakens with a sharp cry, ten drops of specific rhus to a half a glass of water, and a teaspoonful every hour, will be found an efficient agent.

A saline cathartic and a hot foot-bath will materially assist the above medication.

In passive hyperemia, where possible, remove the cause of the obstruction, and the hyperemia will disappear. If the congestion be not due to pressure upon the veins, specific belladonna will be the specific that will give relief. Ten drops of the agent to four ounces of water, and a teaspoonful every hour, will not disappoint.

### **CEREBRAL ANEMIA.**

**Definition.**—An insufficient amount of blood circulating in the capillaries.

**Etiology.**—This may be due to general or local causes. Thus cerebral anemia may attend the general anemia coming from a severe hemorrhage or from profuse diarrhea, or from pernicious anemia, leukemia, and various cachexias, or it may be due to the accumulation of a large quantity of blood in the peritoneal cavity following the removal of ascitic fluid. A more serious condition would be anemia due to aortic stenosis. Among local causes may be mentioned obliterative endarteritis, compression of the brain from tumors, or a partial destruction of the circle of Willis.

**Pathology.**—The puncta vasculosa are so lessened many times as to escape detection, while there may be an increase in the cerebro-spinal fluid. The gray matter assumes a characteristic pallor.

**Symptoms.**—These are due to profuse, exhausting, diarrheal discharges, and, especially where due to hemorrhage, the symptoms are characteristic; there is dizziness, faintness, pallor of face, ringing in the ears, confusion of ideas, and marked dyspnea, and frequently terminating in a “dead faint.” Where the anemia is sudden and intense, the syncopal attack may terminate fatally.

Where the anemia is more chronic in character, there is pallor of face, the skin is cool, and the pulse is feeble and irritable. The patient is listless and stupid, though we may meet cases with general irritability. There is a dull headache, vertigo, buzzing in the ears, spots before the

eyes, and weakness of the muscles.

The hydrocephaloid symptoms, occurring in young children, so named by Marshal Hall, are pallor, dullness, contracted pupils, and depressed fontanels.

**Treatment.**—This will depend upon the cause giving rise to it. Where the attack is acute and sudden, the patient should be placed in the recumbent position, with the head depressed. A diffuse stimulant, like ammonium carbonate, gives prompt results. In severe cases, where there has been excessive hemorrhage, a subcutaneous injection of normal saline solution should be used. In the more chronic form, and when secondary, the treatment suggested for general anemia will be followed with nourishing food and gentle exercise in the open air.

## CEREBRAL HEMORRHAGE.

**Synonym.**—Apoplexy.

**Definition.**—Hemorrhage in the brain or its meninges, usually from the arteries and capillaries, though occasionally from the veins.

**Etiology.**—Eighty per cent of cerebral hemorrhages occur after the age of forty, though it has taken place in a child of nine. It is more frequent in men than women, no doubt from greater dissipations and more severe physical exertions of the former, which tend to diseases of the blood-vessels. A feeble condition of the cerebral vessels, due to disease of their walls, is essential for the lesion, when a variety of causes may be responsible for the accident.

In certain families there is a tendency to degeneration of the arteries, hence *heredity* must figure as a predisposing cause. A large number of cases are preceded by some lesion of the heart. It may be endocarditis with its accompanying- hypertrophy, especially where the hemorrhage occurs in young persons, or it may be due to endarteritis or atheromatous condition of the vessels; again, it may be due to rupture of small aneurisms (miliary) of the branches of the cerebral arteries.

Another common condition is nephritis and diabetes, while alcohol, syphilis, rheumatism, and gout are not infrequent factors in the case.

Certain infectious diseases, especially ulcerative endocarditis and diphtheria, may be mentioned, while leukemia and anemia are not to be overlooked as causal conditions.

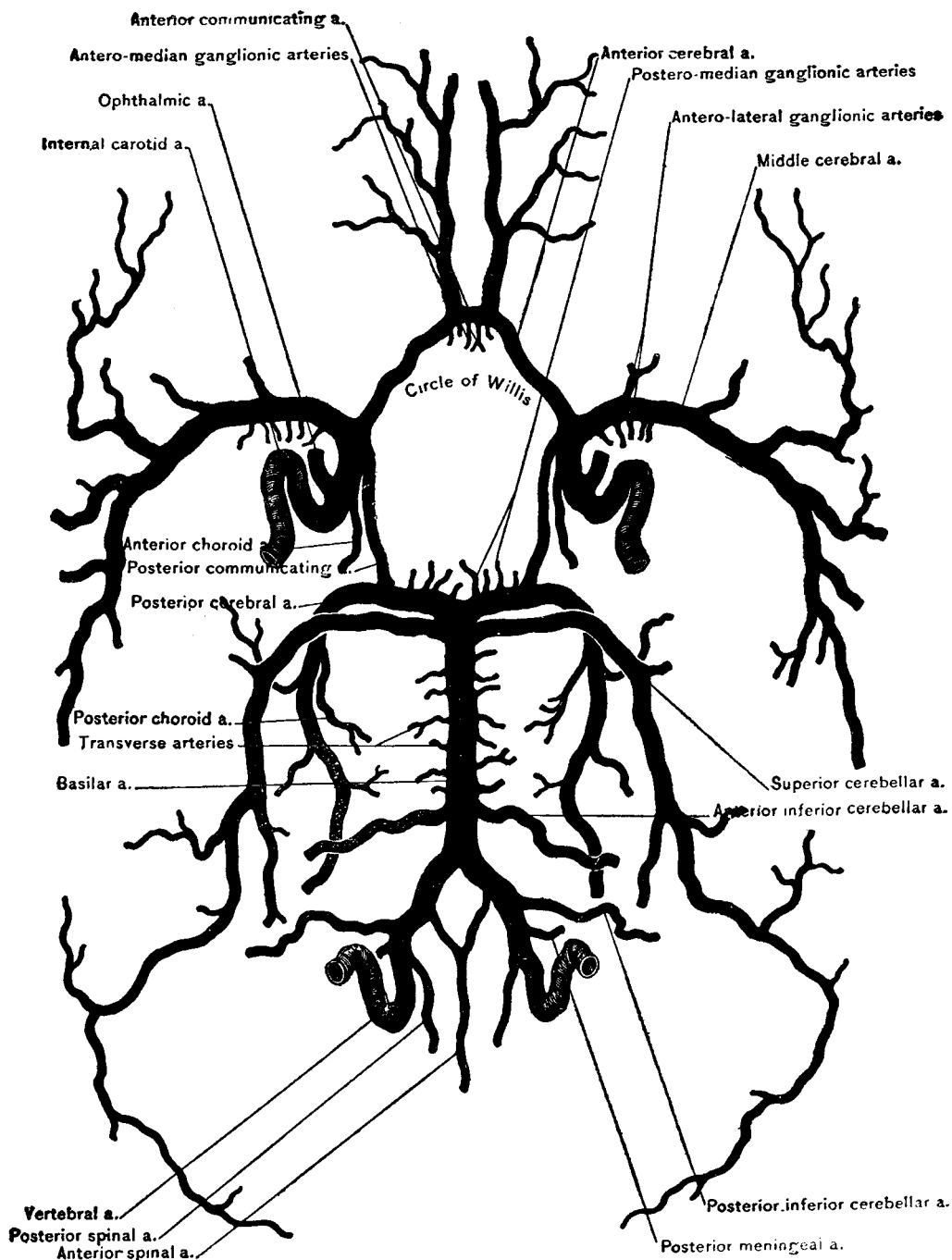


FIG. 48. Circle of Willis and Arteries of Brain.—(Deaver.)—(Tyson.)

**Pathology.**—While any part of the brain may be the seat of hemorrhage, the most frequent site is the base in the neighborhood of the corpus striatum and optic thalamus, since their chief supply comes from the branches of the cerebral artery. The majority of the severe hemorrhages of the brain occur in the internal capsule and lenticular nucleus, by the rupture of the lenticulo-striate artery; so frequently is this artery ruptured that Charcot has termed it the “artery of cerebral hemorrhage.”

“Under different circumstances the blood effused may be small in quantity, or amount to several ounces. In the latter case it tears up the brain-tissue, destroying, for instance, the great ganglia, and the internal capsule, and extending thence into the centrum ovale; or it may burst through the optic thalamus or caudate nucleus into the lateral ventricle. Thence the blood flows by the aqueduct of Sylvius into the fourth ventricle. Such cases are rapidly fatal, and post-mortem examination reveals a mass of black clot, filling the ventricle, and occupying much of the hemisphere, surrounded by brain-tissue, which is ragged and discolored by blood. The pressure exerted by the clot is shown by one, or even both, hemispheres being enlarged, with flattened convolutions and closed sulci. In cases which have lasted a few days, there is the same black-red clot, and the brain-tissue around is soft and discolored yellow, from absorption of hemoglobin. In later stages, the clot becomes brown, or brownish-yellow, consisting of disintegrated blood and nerve-tissue; and the surrounding tissue is frequently softened (white softening), and contains granule-corpuscles. Finally, in patients that survive, the blood becomes absorbed, and leaves a tawny or orange-colored spot, in which crystals of hematoidin can be found; or a cyst may remain, containing serous fluid; or a distinct, tough, fibrous scar, discolored also by the remains of blood-pigment.

“*Secondary Degeneration.*—Permanent lesions of the pyramidal tract, or of the cortical motor area, are followed by descending secondary degenerations, like those which occur in disease of the spinal cord. Such degenerations follow the course of the pyramidal fibers below the lesion; thus, a lesion of the internal capsule (and it is to be observed that lesions of the corpus striatum and optic thalamus alone are not followed by secondary degeneration) cause's this change to take place in the middle third of the crus cerebri, in the anterior part of the pons, in the pyramid of the medulla oblongata on the same side, in the column of Ttirck, also on the same side, but in the posterior part of the lateral

column of the spinal cord for its whole length on the opposite side.” (Taylor.)

**Symptoms.**—Although unusual, premonitory symptoms may be present, and consist of headache, more or less dullness, ringing in the ears, vertigo, and sometimes choreiform movements. There also may be motor impairment, as seen in the slow step or movement of the arm.

Generally, with the rupture of the vessel, comes the *apoplectic stroke* or *apoplectic shock*, and the patient drops unconscious on the ground. The face is dusky or ashen gray; the pulse, at first small, soon becomes full, slow, and of increased tension; the breathing is noisy, stertorous, and the cheek is blown out at each expiration. Sometimes there is the Cheyne-Stoke respiration.

There is usually complete relaxation on the paralyzed side, though there may be twitching of the muscles or slight convulsive movements for a time. Soon, however, these cease, and there is present the characteristic flaccidity and loss of muscular control. This may be seen by lifting the affected member, when it drops heavily to one side.

The pupils vary, usually dilated, though when the hemorrhage is in the pons or ventricles, irritating the nucleus of the third nerve, the pupils are contracted. The temperature is not, infrequently, slightly subnormal, though when the hemorrhage is of the base of the brain, there is apt to be a high temperature. There is loss of control of the sphincters, and the feces and urine are passed involuntarily.

Where the disease does not terminate fatally in twenty-four or forty-eight hours, the breathing becomes less labored, and the patient gradually regains consciousness. The eye is turned towards the affected side or away from the paralyzed side. Hemiplegia, paralysis of one entire side, is the rule.

Sometimes the symptoms come on gradually, when it is termed *ingravescent apoplexy*. Here the patient feels dull, there is heaviness in the head, moves slowly and with difficulty, and it is several hours before the patient loses consciousness and the loss of voluntary motion, and sometimes consciousness is retained till after the paralysis appears, or, with the onset of motor paralysis, the patient is assisted to bed, drops asleep, and passes into a comatose condition, the failure to waken the

patient being the warning note of his true condition.

The evidence of hemiplegia, before consciousness returns, may be overlooked; but if we note carefully the face, we will notice a slight dropping of the angles of the mouth on the affected side, and the muscles of the extremities are flaccid and baggy. In rare cases the muscles are *rigid* on the affected side.

Although the tongue may not be paralyzed, it goes to the affected side when protruded. When it is paralyzed there is great difficulty in articulation.

Sensation may be but slightly impaired, and only for the first few days, but early disappears.

Although the *tendon reflexes* are generally *abolished* in the early stage, they are nearly always increased on the affected side later in the disease. A continued failure in the reflexes should be regarded as an unfavorable sign.

Trophic symptoms are seen in an increased temperature, puffiness of the eyelids and hands, coolness and moisture of the feet, and, in some cases, rapid necrosis and gangrene of the tissues over the sacrum, the "*acute malignant decubitus*" of Charcot.

General nutrition is usually maintained, though, in rare cases, there is general atrophy.

Where partial recovery takes place, the mental faculties, as a rule, remain unimpaired.

**Diagnosis.**—The diagnosis is many times extremely difficult and often it is impossible to differentiate between cerebral hemorrhage, embolism and thrombosis. Care must also be observed, or it may be mistaken for diabetic coma, uremic poisoning, epilepsy, opium poisoning, or alcoholism.

The most characteristic signs of apoplexy are sudden *unconsciousness*, deep, heavy *stertorous* breathing, a full oppressed pulse, and *hemiplegia*, which can be determined, even during unconsciousness, by the flaccidity of the muscles.

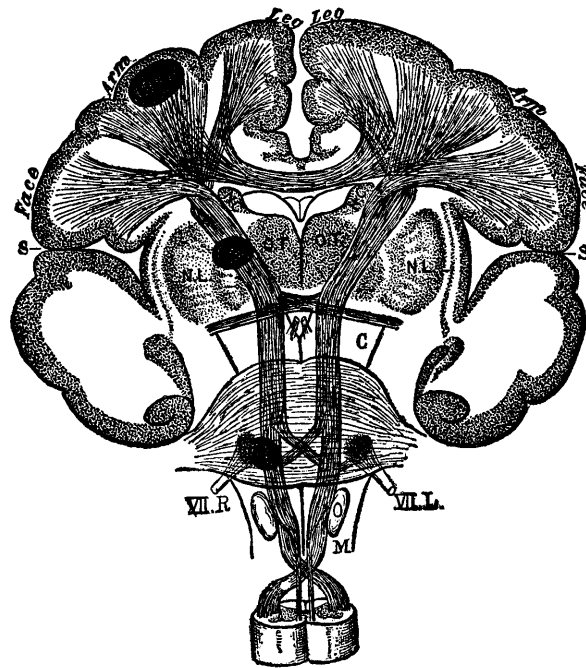


FIG. 49. The motor tract (Starr): *S*, fissure of Sylvius; *NL*, lenticular nucleus; *OT*, optic thalamus; *NC*, caudate nucleus; *C*, crus; *P*, pons; *M*, medulla; *O*, olivary body. The tracts for face, arms, and legs gather from the lower, middle, and upper thirds of the motor area, pass into the capsule, and through the crus and pons, where the face-fibers cross to the opposite VII. N. nucleus, while the others pass on to the lower medulla, where they partially decussate to enter the lateral column of the cord, the non-decussating fibers passing into the ant. median columns. Lesion in cortex causes monoplegia; in capsule, hemiplegia; in pons, alternating paralysis. (Lockwood.)

In a case of drunk, the patient can usually be aroused if only for a moment, and the breathing is *not stertorous*; the breath may be of but little significance, for apoplexy frequently occurs among alcoholics. In epilepsy there is generally the telltale frothing at the mouth, and a history of previous attacks. In uremic coma, the urine should be examined for albumin and casts, and for sugar in diabetic coma. Opium poisoning comes on slowly, and the patient can usually be aroused if but for a moment, and the breathing, while slow and labored, does not give the characteristic stertor. The head should be examined carefully for injuries, that concussion of the brain may be excluded.

**Prognosis.**—This depends largely upon the location and extent of the lesion. Unless the hemorrhage is severe in the pons, death is not likely to occur suddenly. Usually, where the hemorrhage is extensive, the patient will live ten, twelve, or more hours. Where slight, the patient



may recover within a few weeks. Should the patient fail to show improvement in three months, the outlook is unfavorable.

The muscles of the face are the first to show improvement; then the patient will be able to move the toes, and later the limbs may be flexed, till finally he can step and bear his weight. At first his feet seem too heavy to lift, the patient shuffling or dragging the foot, though in time there may be but little use of a cane. The muscles of the hand respond slowly, and complete recovery may not take place, the hand being puffy, bluish, and cold.

Where the coma deepens the second or third day after the attack, or the temperature rapidly increases within the first forty-eight hours, the prognosis is decidedly unfavorable.

Treatment.—The clothing about the neck should be loosened, and the head slightly elevated. If the pulse be *full* and *strong*, veratrum should be given in full doses, say one dram to four ounces of water, and a teaspoonful given every one, two, or three hours. A brisk cathartic should be given early. Sinapisms may be applied to the spine, hot applications to the feet, and the head sponged with hot water, while an assistant uses the fan. Some prefer the ice-bag to the head.

When consciousness returns, the patient should be kept perfectly quiet, all efforts at conversation prohibited for a few days, and liquid nourishment given.

Where the capillary circulation is feeble, belladonna will be found useful. Massage and electricity will be found efficient in restoring tone to the muscles during convalescence.

## **EMBOLISM AND THROMBOSIS.**

**Synonym.**—Cerebral Softening.

### **EMBOLISM.**

**Definition.**—The obstruction of the cerebral arteries or capillaries by material (embolus) floated, by the blood-stream, from some distant part.

**Etiology.**—Nearly ninety per cent of all cases of embolism occur as the result of valvular disease of the left heart. It may be a vegetation of an acute endocarditis, though more frequently the result of chronic inflammation of the valves or the ulceration of their segments. In rarer cases it occurs from a portion of a clot from the auricle, from aneurism or atheroma of the aorta, or from the great vessels of the neck or of the lungs.

In certain infectious diseases, as pneumonia, diphtheria, puerperal fever, septicemia, and kindred affections, heart-clots may form and portions be washed into the cerebral vessels, giving rise to embolism. Since the embolus more frequently enters the left carotid, the left middle cerebral artery is most often the seat of the disease.

The lesion occurs more frequently among young adults, and in females, than in males.

## **THROMBOSIS.**

**Definition.**—Clotting of blood in the cerebral arteries, veins, or sinuses.

**Etiology.**—This is most frequently due to disease of the walls of the blood-vessels, whereby their surface is roughened, favoring the deposit of fibrin. This may arise from endarteritis, atheroma, or syphilitic endarteritis, or from weakening of the circulation from diphtheria, typhoid fever, tuberculosis, cancer, and such diseases as impair the integrity of the blood. It may result from aneurisms, and it has followed the ligation of the carotid artery. In fact any condition that affects the vessel wall, or obstructs the blood-current, or increases its coagulability, is a possible cause of thrombosis. The thrombosis occurs most frequently in the middle cerebral and in the basilar arteries.

**Pathology.**—Taylor thus describes the anatomical changes: “Embolism and thrombosis, by obstructing” the circulation of the blood, alike lead to *softening* of the districts of the brain to which the vessels correspond, unless the vascular supply is maintained by means of anastomoses. These are not abundant in the case of the cerebral vessels, and, indeed, the vessels going to the central ganglia are really terminal vessels, while those going to the cortex of the brain do anastomose more or less. At least, this is true of the distribution of the middle cerebral artery—the

vessel most often obstructed. A part of the brain in which softening has taken place has generally lost the smooth, glistening surface of a normal brain-section, is more opaque, or gray, or speckled; it breaks down readily under a stream of water; or it is milky, or diffluent. It shows under the microscope drops of myelin, portions of nerve-fibers, granule-corpuses, and free fat-globules. It sometimes has a yellowish or brownish color from blood-pigment; or minute extravasations may be present in cases of sudden obstruction, and a form of *red softening* results. In cases of rapid death after embolism, the brain substance may look perfectly healthy, as there has not been time for any changes visible to the naked eye to take place. Occasionally an embolus sets up inflammatory changes in its neighborhood; sometimes it leads to aneurism and cerebral hemorrhage. Rarely actual infarcts are formed. The later stages of softening consist in the absorption of the disintegrated tissue, and the formation of a cyst; or, if the softening is small, a cicatrix may be produced.

“Embolic lesions, involving the motor tract, are followed by the same secondary changes (descending sclerosis) as are hemorrhagic lesions. A persistent lesion of the brain, whether embolic or hemorrhagic, causing hemiplegia in infancy or early childhood, has the remarkable effect of checking the growth of one-half of the brain, or it may be of other parts of the central organs, so that years after it is smaller than the other half, and is described as atrophied (*cerebral hemiatrophy, unilateral atrophy*). If the lesion is in the motor cortex, the hemisphere is atrophied on that side, and there is sclerosis of the pyramidal tract; if it is in the basal ganglia, there is in addition atrophy of the middle fillet in the pons medulla, and of the antero-lateral region of the spinal cord on the *same* side; and atrophy of the cerebellum, superior cerebellar peduncle and dentate nucleus on the *opposite* side (Mott and Tredgold).”

**Symptoms.**—Naturally, the symptoms will depend upon the location and extent of the lesion. They may be so slight as to escape detection, and only be discovered during an autopsy, or they may be so severe as to destroy life in a few hours. The shock from cerebral embolism may be so similar to cerebral hemorrhage as to be almost indistinguishable.

A very important factor in determining the symptoms is whether the embolism be in a small artery or in a large one, and if it be located in the hemispheres or toward the base of the brain.

In embolism, the onset is almost invariably sudden, there being no premonitory symptoms, the patient suddenly losing consciousness. Usually this is not so severe as in cerebral hemorrhage, and the patient soon regains consciousness, though, when very severe, coma becomes pronounced, and the case terminates fatally. Convulsions may occur when the motor regions are involved.

If the anterior cerebral is the vessel involved, the symptoms are often negative, since branches of the middle cerebral will supply about the same area. Apathy and dullness are sometimes present.

When the middle cerebral is involved, the one most frequently affected, hemiplegia follows, and is permanent or transient according to the location of the plug. Thus if the vessel be blocked before the central arteries are given off, it is permanent, while if beyond this point, the arm and face suffer, but it is generally temporary.

If the left side be involved, there is aphasia. If the trunk be spared and the branches are involved, the symptoms will vary according to the part affected. These branches supply the inferior frontal, the anterior and posterior central gyri, the supramarginal, angular, and temporal gyri.

The different types of aphasia are thus explained: *Motor or ataxic aphasia*, when the patient remembers the words, but can not articulate them: here the lesion is in the third left frontal convolution. *Sensory aphasia*, where the patient fails to comprehend the meaning of spoken words (word-deafness), is due to lesions of the first and second temporo-sphenoidal convolutions. If the angular gyrus be involved, *word-blindness* follows.

The posterior cerebral vessels supply the occipital and temporo-sphenoidal lobes, and when involved—a rare case—there is hemianesthesia due to softening of the internal capsule, and hemianopsia due to softening of the cuneus. Sometimes there is complete loss of sight, though, generally, but temporary.

*Involvement of the Internal Carotid.*—If the circulation be by the communicating vessels of the circle of Willis, there will be an absence of symptoms; but if these vessels are small or absent, the circulation is arrested, and permanent paraplegia and death are apt to follow in a few days.

*Basilar Artery.*—Where this is occluded, bilateral paralysis, from involvement of both motor tracts, is apt to follow. In these cases the temperature rapidly rises to 107°, 108° or 109°, or even higher, the pulse is rapid and irregular, and convulsions may occur. Bulbar symptoms are frequently present.

*Vertebral Artery.*—The left is the more frequently involved, and usually in connection with the basilar. The nuclei in the medulla are affected, and attended by symptoms of acute bulbar paralysis.

*Cerebellar Arteries.*—Incoordinations of movement have been recorded as a result of cerebellar softening, though the lesion is rare.

*Thrombosis*, though not so rapid in its development, is followed by the same results, apoplexy and hemiplegia. The disease comes on more gradually; there is frequent and persistent headache, more or less dizziness, a gradual loss of the mental powers, and a perverted sensibility manifested by a numb, tingling, or creeping sensation in arm or leg. These symptoms, especially in elderly people, gradually increase till the mental faculties are destroyed, and we have the “softening of the brain” of old people.

**Diagnosis.**—It is sometimes almost impossible to differentiate between embolism and cerebral hemorrhage during the first few days; generally, however, they can be recognized by the following conditions: A history of rheumatism or endocarditis usually precedes embolism. Unconsciousness is not so prolonged nor coma so marked. The face is pale, not flushed, nor is there stertorous breathing.

Thrombosis comes on gradually, preceded by dizziness, headache, and perverted sensibility.

**Prognosis.**—The prognosis from embolism is slightly more favorable than from hemorrhage; the location is also a determining factor in the recovery. Paralysis is more apt to be permanent in embolism. In thrombosis, cerebral softening frequently follows, the prognosis in such cases being unfavorable.

**Treatment.**—In the administration of remedies we will be guided entirely by the conditions present. The immediate treatment will be rest

in bed, and, if shock be present, stimulants administered. Later, in those cases where irritation occurs, sedative agents will be used. The antisyphilitics will be used where the lesion has been preceded by syphilis.

In cerebral softening, but little can be done save improving the general health. Hygienic and dietetic measures will be an important part of the treatment.

## APHASIA.

**Definition.**—Aphasia is that condition due to cerebral lesions, whereby there is an inability to comprehend words properly and use them correctly, or a total suppression of the power of speech. It must not be confused with—

(1) Anarthria, a defect of articulation due to lesions of the medulla oblongata (bulbar paralysis).

(2) Mental aberrations independent of demonstrable lesions of the cerebral centers.

The language or speech centers are found in the third or inferior *left* frontal convolution (Broca's convolution), and when associated with hemiplegia—and it is in most cases—it is with a *right* hemiplegia.

There are various phases and degrees of aphasia, according to the extent of the lesion and the center involved; thus a patient may be able to, say only one or two words, as yes or no, and repeats these to every question, or he may not be able to speak a single word. Others have a larger vocabulary, but are unconscious of the *fitness* of words to express their ideas, while others recognize their unfitness after speaking.

“One may be unable (a) to *hear* words spoken; (b) to *understand* words spoken; (c) to *see* words written and printed; (d) to *understand* words written and printed; (e) to *speak* from memory; (f) to *repeat* words; (g) to *read aloud*, i. e., to *speak* from *sight*; (h) to *write* from *memory*; (i) to *write* from *dictation*, i. e., from words heard; (k) to write from a copy, i. e., from words seen.”

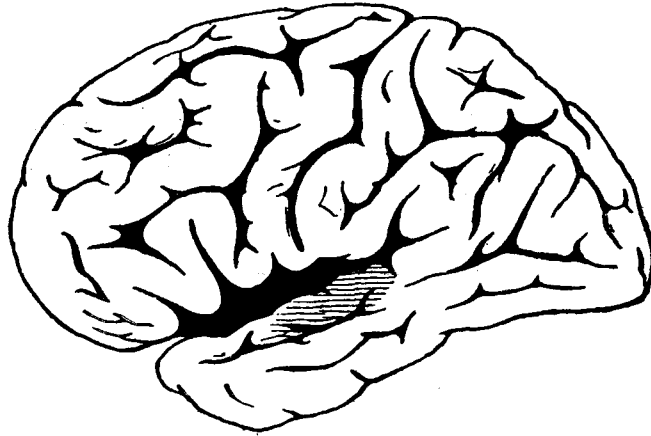


FIG. 50. Situation of lesion in the first left temporal convolution, causing Word-deafness.—(After *Charcot*.)

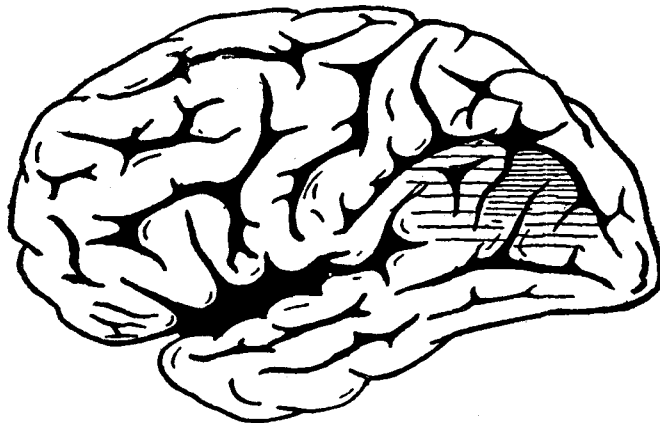


FIG. 51. Situation of lesion causing Word-blindness.  
(After *Charcot*.)

“Speech not only depends upon perfect co-ordinating and motor processes, but also upon the senses of hearing- and sight. In aphasia therefore certain centers are involved, and are as follows: (1) The auditory speech center, in the upper-extremity of the left temporo-sphenoidal convolutions; (2) The visual-speech center, in the left angular gyrus and supramarginal gyrus; (3) The motor-speech center, in the posterior part of the third left frontal convolution, or Broca's convolution; (4) The motor-writing center, probably in the posterior part of the second left frontal convolution, related to the muscles of the hand; (5) Commissural fibers between these.”

Lesions of the first four centers of the commissural fibers between them will cause some form of aphasia or agraphia.

Where the motor-speech center is destroyed there is *motor aphasia*, and where the visual-speech center is involved there is *sensory aphasia*, and an involvement of two or more centers gives rise to the various combinations of aphasia.

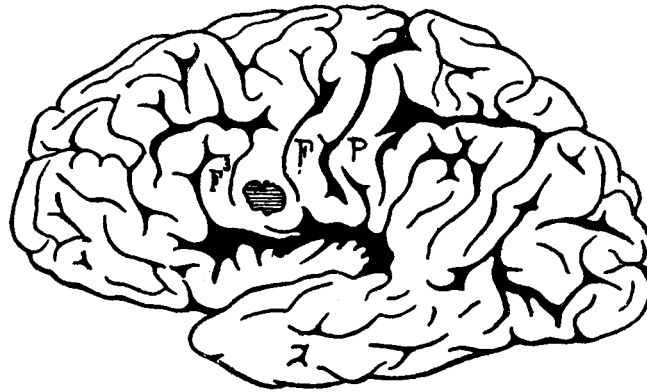


FIG. 52. Situation of the lesion in the case of Motor Aphasia.  
C. Ascending frontal convolution. F<sup>3</sup>. Third frontal convolution. P. Ascending parietal convolution.  
(After Charcot.)

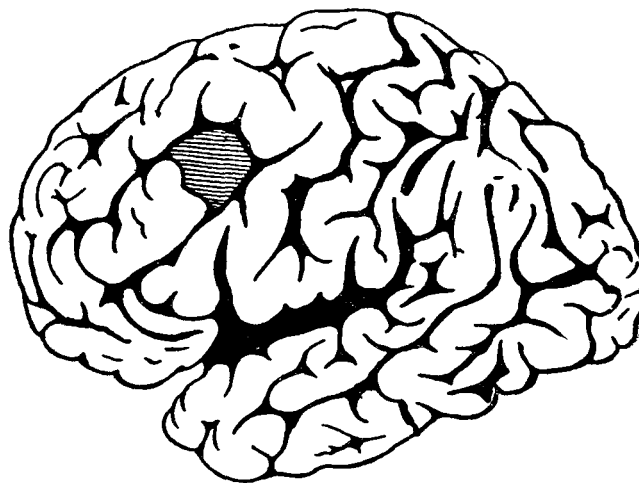


FIG. 53. Situation of lesion in the case of Agraphia. (After Charcot.)

**Etiology.**—Since almost the entire cerebral centers for language are in the cortex or immediately beneath it, any organic or functional disease that interferes with this portion of the brain is a cause of aphasia.

The most common causes are embolism and thrombosis, with their consequent softening. Hemorrhage is also a not infrequent cause.



Severe congestion, without hemorrhage or softening, may give rise to a more transient aphasia. Trauma, as fractures of the skull; meningitis by impairing the cortex; degenerative diseases, and toxemias form cerebral abscesses; or the severe infectious diseases, may also be responsible for aphasia.

**Symptoms.**—*Motor Aphasia.*—The inability to articulate, or even gesture, usually comes on suddenly, and at first may be complete, though a limited power is usually retained or early regained. The patient may be able to say only one or two syllables or words, and repeats them on all occasions regardless of their propriety. Sometimes it will be an exclamatory oath, used automatically. Sometimes the words will consist of some part of a sentence that he was about to utter just before the attack rendered him speechless.

His sensory centers being unimpaired, he can understand what is said to him and appreciate his own errors, but is unable to correct them.

His ability to write is also affected (agraphia), and he finds it impossible to write even the few words that he can articulate, though he may be able to sign his name. Along with this inability to articulate and write there is frequently lost the power of understanding printed or written words.

*Sensory Aphasia.*—In this form there is a disturbance of the auditory and visual speech centers; *word-deafness* in lesion of the auditory speech-center, and *word-blindness* in lesion of the visual speech-center.

In word-deafness, while the patient can hear ordinary sounds. he is unable to recognize and understand spoken words, and can not repeat words or write from dictation.

In word-blindness the patient can not see or understand words printed or written, can not read aloud, and can not write spontaneously from dictation or copy, as writing is simulated directly from the visual center, but he may be able to talk well.

Two other conditions may be associated with sensory aphasia: *mind-deafness*, or inability to recognize the meaning of any kind of sound; and *mind-blindness*, or inability to recognize the meaning or use of any kind of object.

**Prognosis.**—This depends largely upon the cause and extent of the lesion. Where it is functional, and due to congestion, inflammation, or toxemia, the aphasia is transient and disappears with a correction of the exciting cause.

If in children, although the speech centers be destroyed, patient and persistent instruction may result in the .education of the speech centers in the opposite hemispheres. If due to tumors, bone-pressure, or abscess, relief may follow operative measures.

**Treatment.**—Save in functional aphasia, but little may be expected from medication.

When due to pressure from any source, the only promise of relief is at the hands of the surgeon. For special instruction in developing- speech centers, one should consult those who make it their life-work.

## ENCEPHALITIS.

**Definition.**—This, in the true sense, is an inflammation of the brain-tissue, a cerebritis, and usually, if not always, implies an inflammation of the meninges as well, the symptoms of which are those of meningitis aggravated or intensified. Encephalitis, as now *generally used*, is a synonym for abscess of the brain— Suppuration Encephalitis.

## ABSCESS OF THE BRAIN.

**Etiology.**—This lesion is usually found between the ages of twenty and forty, and more frequently in males than in females. It is generally, if not always, secondary, and may arise from an extension of suppurative inflammation of the ear, nose, or orbit, especially where the bones are involved; from head injuries as fracture of the skull, or from a punctured wound; from pyemia, septicemia, gangrene of the lung, ulcerative endocarditis, and necrosis of bones; from intercranial tumors, and from the specific infectious fevers. It is more apt to occur among the poorer classes, especially where due to middle-ear diseases following scarlet fever, and where the child has not had the proper care.

**Pathology.**—The abscess varies as to size and character of its contents. It may be single or multiple, usually the former. Pus of varying consistency, blood, and pyogenic organisms make up its contents. It is most frequently found in the, temporo-sphenoidal lobe, owing to its relation to the middle ear. It is rarely found in the cerebellum, and still less so in the pons and medulla. The neighboring- brain-substance is generally softened and reveals the change due to inflammation. Where of long standing, a thick layer of fibrous tissue is found walling off the abscess.

**Symptoms.**—These vary, according to size, location, cause, and character of the inflammation. Where there are severe complications, as head injuries or meningitis, the symptoms may be so masked as to escape detection. They may appear quite rapidly, or come on insidiously. Where acute, the symptoms are of acute septic infection; there is a chill, followed by rise of temperature, intense headache, vertigo, vomiting, mental dullness, or delirium and convulsions.

When it comes on slowly, being secondary to septicemia or ulcerative endocarditis, in addition to symptoms of the primary lesions, there will be chills, irregular fever, sometimes the temperature being subnormal, nausea, vomiting, headache, and convulsions. The motor and sensory symptoms depend upon the extent and location of the abscess. There may be aphasia, hemiplegia, clonic spasms, or hemianopsia. The reflexes are generally much exaggerated. Choked disks are rare, though congestion of the eye-grounds is common. Respiration is usually slow, from ten to fifteen per minute, while the Cheyne-Stokes respiration is sometimes observed.

In rare cases the abscess ruptures into the ear or nose, and relief is experienced by drainage from those channels. If the abscess be in the “silent regions,” and small in size, the symptoms may be so slight as to escape detection.

**Diagnosis.**—In acute cases preceded by head injuries, there will be but little difficulty in determining the lesion. With a history of injury followed by intense headache, fever, delirium, optic neuritis, and convulsions, the diagnosis would be easy. In the more chronic cases, an accurate history and a careful examination of the patient should be made for endocarditis, septic lung lesions, bone necrosis, etc., and should these be followed by irregular fever, nausea, vomiting, headache, coma,

convulsions, motor and sensory symptoms, such as aphasia, hemiplegia, etc., the diagnosis should not be difficult.

**Prognosis.**—This is always grave, though if free drainage be secured, either by spontaneous rupture into the nasal or aural opening, or by surgical measures, the patient will frequently recover.

**Treatment.**—Though the antiseptics would be indicated in all cases of sepsis, the treatment is surgical, and consists of early evacuation and thorough drainage.

## **HYDROCEPHALUS.**

**Synonym.**—Dropsy of the Brain.

**Definition.**—An accumulation of fluid within the ventricles or subarachnoid spaces of the brain.

**Etiology.**—Hydrocephalus may be either congenital or acquired. Although heredity has been assigned as influencing the congenital form, several children in a family being affected, the true cause is unknown. The acquired form may result from some impairment of the circulation in the straight sinus, from brain tumor, or from meningitis.

In the congenital form, the enlargement may be so marked as to render labor extremely difficult, and sometimes necessitate craniotomy.

**Pathology.**—In the congenital form, the head is noticed to be unusually large at birth, or very soon develops after coming into the world. The fontanelles are bulging and the sutures separated. On opening the skull, the bones are found to be thin, the dura usually unchanged, though it may be thickened, while the brain-substance is slightly softened. The ventricles, especially the lateral, may be enormously distended, while the third and fourth show less disturbance.

The ependyma may be thickened and slightly roughened, or it may remain unchanged. Where the child survives a few years, the sutures are closed by Wormian bones. Sometimes the head reaches an enormous size.

In the acquired form, the head does not show such marked enlargement, and the ventricles are but moderately distended. The brain shows some softening.

**Symptoms.**—In congenital hydrocephalus the most striking feature is the large head, with sutures separated and fontanelles open and bulging. As the head enlarges, the face appears unusually small, and the eyes, owing to the pressure, are peculiarly prominent. The child is unable to walk, and is soon unable even to sit up and support the great weight of the head. The child lies upon its back with the limbs flexed. Convulsions are quite common. The intellect is generally impaired, or slow in development. In rare cases the intellect is well preserved. The child is generally listless and dull. Nutrition is more or less disturbed, and the puny body bears a striking contrast to the enormously developed head. Fortunately these children do not often survive childhood, though a rare case reaches maturity.

In the acquired form, there may be headache, dimness of the sight, progressive optic neuritis, and irregular gait.

**Diagnosis.**—There is generally no difficulty in diagnosing a case of congenital hydrocephalus. The picture is so characteristic that it is difficult to make a mistake. In the acquired form the symptoms may be obscured and the diagnosis rendered more difficult.

**Prognosis.**—A child rarely lives much beyond the fifth year, though he may survive till adult years are reached.

**Treatment.**—Medication offers but little encouragement in hydrocephalus, and operative measures have not brought brilliant results and are always attended by considerable danger.

## **CEREBRAL PALSIES OF CHILDHOOD.**

**Synonym.**—Infantile Cerebral Paralysis.

**Definition.**—This includes two varieties: one which is noticed at birth, hence is termed birth-palsy, and usually involves both legs and arms; and one which takes place during the first two or three years of life, and is generally hemiplegic.

## BIRTH-PALSY.

**Etiology.**—This occurs most frequently in premature births and in difficult labors, especially in instrumental deliveries, and which are attended by meningeal hemorrhage.

Abnormal conditions during intra-uterine life, not readily accounted for, may be responsible for this condition. Syphilis, heredity, shock, and great mental excitement may have some etiological significance.

**Pathology.**—The most constant lesion is the meningeal hemorrhage, which impairs the cerebral cortex, affecting various brain centers. Thus where the pressure takes place in the third frontal convolution, the speech centers are involved, and when the center controlling the eye movement is involved, the child squints.

Where forces have been at work prior to birth, and therefore are not mechanical, a loss of cerebral substances takes place, *porencephalous* cysts sometimes replacing the brain-tissue.

**Symptoms.**—Even at birth, rigidity of the muscles may be noticed, while attempting to dress the infant, or it may not develop for some weeks, when the legs are inclined to cross. In other cases nothing unusual is noticed until the child attempts to walk or use its hands. The intellect is usually feeble, especially when the cause is due to loss of cerebral substances.

When the child attempts to stand, the weight is thrown upon the inner side of the feet, the knees are placed together or even crossed, and if able to walk, only one foot is advanced, the other being dragged after its fellow. Choreiform movements and various grades of spasmodic inco-ordination are seen, and convulsions are not uncommon. Sensation is generally normal, though the *reflexes* are increased.

**Diagnosis.**—The rigidity of the limbs, the choreiform movements and spasmodic inco-ordination of the muscles, the peculiar position of the feet and limbs on standing, and the unnatural gait on attempting to walk, together with deficient intellectual features, render the diagnosis comparatively easy.

**Prognosis.**—This depends, to some extent, upon the mental condition and the degree of paralysis, though the prognosis is grave in all cases.

**Treatment.**—In addition to hygienic and dietetic measures to improve the general health, massage, and faradization should be given a thorough trial. A careful and systematic course of training should be carried on as long as there is the least encouragement. The medication will be entirely symptomatic.

### **INFANTILE HEMIPLEGIA.**

**Etiology.**—Although cerebral traumatism and the infectious fevers have been regarded by some as being responsible for this condition, the specific cause is unknown. Some cases are congenital, though a very large majority of cases develop during the first three or four years of life.

**Pathology.**—In the majority of cases, the nature of the primary lesion is unknown. In an analysis of ninety autopsies made from the literature on this subject, Osler found the following:

(a) In sixteen cases there was embolism, thrombosis, or hemorrhage. In seven of these a Sylvian artery was occluded; in nine there was hemorrhage. It is interesting to know that in ten of these cases the disease set in in children over six years of age.

(b) Atrophy and sclerosis, which are the common conditions in the majority of cases. The wasting is either of groups of convolutions, an entire lobe, or one hemisphere. The meninges may look normal, though more commonly they are tightly adherent and the brain-substance tears. The convolutions are shrunken, firm, and hard. In some instances there is a remarkable unilateral atrophy, in which the brain-tissue is a mere shell over the greatly dilated ventricle. Thus, in one of my cases, the atrophied hemisphere weighed 169 grammes and the normal 553 grammes.

(c) Porencephalus was present in twenty-four of the ninety autopsies. This term was applied by Heschi to a loss of substance in the form of cavities or cysts at the surface of the brain communicating with the arachnoid space, and in some cases passing deeply into the hemisphere,

reaching even to the ventricle. Of 103 cases of porencephalus analyzed by Audry, hemiplegia was present in 68. (Pepper's "Practice.")

**Symptoms.**—The onset is not infrequently attended by general malaise and slight fever, the temperature rarely exceeding 102°. This is followed in twenty-four or forty-eight hours by convulsions, generally on the side which is afterwards paralyzed. The convulsions may occur at slight intervals, the patient losing consciousness.

Many times the disease comes on suddenly without a single premonitory symptom, the patient suddenly becoming unconscious. and on the return to consciousness it. is found that the child is paralyzed, the right side being the most frequently involved and the arm more seriously than the leg. Atrophy of the muscles, especially of the arm, is quite marked.

Epilepsy is a frequent result, especially when the hemiplegia is from birth.

Generally there is *arrested* or retarded mental development, and idiots or imbeciles are not uncommon among this class of unfortunates.

The knee-jerk and ankle clonus may be present.

Post-hemiplegic movements consist of tremors, choreiform movements, and athetosis.

**Diagnosis.**—This is readily made from the characteristic symptoms that accompany hemiplegia.

**Prognosis.**—As regards life, the prognosis is good, though mental impairment and some degree of paralysis remain. Aphasia is rarely ever permanent.

**Treatment.**—Aside from the treatment for the convulsions, and these should be controlled as soon as possible, the treatment will be symptomatic, and means and methods used for improving the general health.

A nutritious and easily digested diet, plenty of fresh air and sunshine, massage and faradization, and patient and systematic education, will be



important factors in the treatment.

## IV. DISEASES OF UNKNOWN PATHOLOGY.

### EPILEPSY.

**Synonym.**—Falling Sickness.

**Definition.**—A condition in which there is a sudden, though temporary, loss of consciousness, with or without convulsions. When the attack is but momentary and without convulsions, or with but slight tremors, it is termed *petit mal*. Where the attack is severe, unconsciousness is prolonged, and there is severe general convulsions, it is known as *grand mal*.

**Etiology.**—The causes may be properly divided into predisposing and exciting.

*Predisposing.*—*Heredity*, according to Fere, is a strong predisposing factor, in that a marked neurotic taint is to be found in the lineage of epileptics. Not that there is a direct transmission from parent to child, for this is rare, but where not due to pressure, a history of insanity, hysteria, paralysis, insomnia, suicide, chorea, puerperal eclampsia, and other vices of the nervous system can be traced to one or both parents.

*Consanguinity* has been considered by some as favoring epilepsy, but in all probability such cases were coincidences rather than predisposing factors.

*Age.*—One-third of the cases occur before the thirteenth year, two-thirds before the nineteenth year, and the remaining third before the thirtieth year.

*Alcoholism.*—The fact that nearly one-half of all epileptics recorded are children of parents addicted to drink, is a significant factor.

*Scarlet Fever and Puerperal Eclampsia.*—Gowers states that scarlet fever is the disease that is most frequently followed by epilepsy, while Fere has observed epilepsy following puerperal eclampsia. All these conditions, however, only prepare the soil for some one of the many

exciting causes.

*Exciting Causes.*—Favorable conditions being present, a variety of exciting causes may precipitate an attack. Thus traumatism, syphilitic neoplasms in brain or cord, meningeal irritation, great emotion, nervous shock, acute alcoholism, toxic substances in the blood, an overloaded stomach, masturbation, an elongated or adherent prepuce, an adherent clitoris, scar tissue in the uterine cervix, teething, intestinal parasites, etc., may be mentioned.

One peculiar feature of the disease is that the epileptic habit having once been firmly established, the disease is prone to continue, even though the cause is seemingly removed.

**Pathology.**—There is no characteristic pathological condition found that is constant. In some there will be organic changes, such as tumors, meningeal changes, pressure from bone, or from syphilitic deposits, and other demonstrable wrongs; but these same conditions may be present in patients that never suffer from epileptic seizures. Of the idiopathic variety no pathological wrongs can be found. When due to reflex causes, tissue changes are frequently present at the source of irritation, as of the rectum, uterine cervix, urethra, ovary, perineum, etc.

Chaslin claims to have found a characteristic non-inflammatory degeneration, in which the neuroglia of the brain is transformed into an abnormal tissue, and that this degeneration is a result of a vice in development. The finding of this degeneration in all cases, however, has not been corroborated.

**Symptoms.**—*Grand Mal.*—Prodromal symptoms, or aura, frequently occur in this form, and may last but a few moments or occur a few days before the onset of an attack. Where of long duration, the patient becomes morose, cross, sullen, irritable, or melancholy. Where brief, it may be a sense of fear, an idea of impending danger, or there may be flashes of light, or loss of hearing, or a perverted hearing, such as whistling, "buzzing, musical sounds, or hissing; again, disagreeable odors, or a peculiar taste; or there may be discomfort in the epigastric region; sometimes it takes the form of numbness or tingling in the extremities, as the foot or hand, gradually extending to the head. Whether or not the attack is preceded by aura, the *attack* is sudden.

*The Attack.*—A sudden pallor of the face, a frightened look, followed by a wild piercing scream, known as the epileptic cry, and the patient falls unconscious in the throes of a convulsion. Many times there is not a sound or warning symptom, the patient falling unconscious in a spasm.

With the first unconsciousness, tonic spasms come on, and all the muscles of the body are in a tetanic rigidity. The face is congested and distorted by the facial spasm, the head thrown backward or rotated to one side, and the jaws are locked. The phalanges are flexed, the thumbs drawn into the palm, the wrists flexed, and the arm at the elbow. The legs are extended and the feet everted. The circulation is sometimes so greatly impeded as to cause rupture of the vessels, and hemorrhage from the nose, ears, bronchi, and sometimes into the cerebrum, takes place. This stage lasts from a few seconds to one or two minutes, to be succeeded by the stage of clonic spasm.

During this stage the whole body becomes agitated by the alternate contraction and relaxation of the muscles. The eyes twitch or roll, the muscles of the face incessantly move, which gives the features a hideous expression; the jaws open and close; there is grinding of the teeth, and biting of the tongue; there is a hurried, jerky respiration; a bloody, frothy foam escapes from the lips, and there is jerking of the limbs and body. Not infrequently the feces and urine are passed during the convulsive action. The pupils are usually very much dilated during this stage, and the return to the normal may be the first evidence of a cessation of the attack. The respiration during this convulsive period is slow, stertorous, and irregular. Where the convulsions are long continued, the temperature may reach 105°, 106°, or more.

This stage lasts from three to five minutes, when the patient recovers consciousness, or falls into a heavy sleep, lasting for a few minutes or hours. On awakening, the patient complains of muscular soreness and headache.

*Nocturnal Epilepsy.*—The attack occurs in the night, and may be unknown to patient or friends. When a patient of rather dull mind complains at intervals of feeling tired, has a headache, in fact there is general muscular soreness, and the patient seems dazed and confused, and if, in addition, he has involuntarily wet the bed, the probability is that of epilepsy, and if the pillow shows some blood-stains, there can be little doubt as to the cause.

*Petit Mal*.—In this form there is no convulsion, and the patient does not fall. The attack comes on suddenly without any premonitory warning; the patient suddenly loses consciousness, the speech, or whatever he was doing, being suddenly and momentarily stopped, to be resumed again in a few seconds. If talking, he resumes the broken sentence as though nothing had occurred, the patient being rarely aware of his condition. In rare cases he may fall, but there is no convulsion. In some cases the patient appears a little dazed for a few moments after the attack.

*Jacksonian Epilepsy*.—This is characterized by spasms of a local character, though, as the disease advances, they may become general. Tingling and other sensory sensations may precede an attack.

Consciousness, as a rule, is maintained. This is sometimes called cortical epilepsy, and is due to irritation of the cortical motor centers of the brain.

**Diagnosis**.—A typical case of epilepsy can hardly be mistaken for any other lesion. The picture is so striking and horrible that a novice in medicine will recognize it. The sudden attack, the patient falling in a convulsive fit, the tonic, followed by the clonic spasms, the frothing at the mouth, and the coma or deep sleep following, make the diagnosis very easy. In *petit mal* it may not be so easy to recognize unless we have the history of previous attacks.

Hysterical convulsions will be recognized by the more regular rhythm of the spasms and the general history.

**Prognosis**.—This is unfavorable in idiopathic cases, though, where symptomatic, a cure may be effected where the cause can be removed. However, the habit once acquired and continued for some months, is one of the most difficult and stubborn lesions to overcome.

Epilepsy seldom proves fatal, but with the advance of years the intellect becomes more feeble, sometimes resulting in insanity or imbecility.

**Treatment**.—A careful search must be instituted for the exciting cause, and since this is often found to arise at some distant point, like the rectum, the uterus, the ovaries, the urethra, or the stomach, all wrongs

of this character should be corrected. An adherent foreskin should be released, and, where elongated or constricted, circumcision be performed.

All wrongs of the stomach should be corrected, and the patient placed on a nourishing but easily digested diet: It should consist largely of fruits, cereals, and vegetables, meats being allowed sparingly. The bowels should receive careful attention, constipation being avoided. Cold sponge-baths once or twice daily are also beneficial. Where the disease has followed injuries to the head sufficient to produce scalp wounds, scar tissue should be removed, and if there be any depressions causing irritation, surgical measures must be used before any relief can be promised. If pressure result from a tumor or abscess, the skull should be trephined and the pressure overcome.

During an attack the patient should be placed in a horizontal position, the clothing loosened about the throat, and cork, rubber, or a soft piece of wood placed between the teeth to preserve the tongue from injury. Where the convulsions are very intense, chloroform should be used till the throes subside, when the patient should be allowed to sleep till spontaneous awakening occurs.

The medical treatment for epilepsy has not been attended with very satisfactory results. The bromids are used probably more frequently than all other remedies combined, yet few, if any, recoveries can be recorded from its use. That it is a good nervine sedative, however, none will deny, and some relief may be obtained from its administration.

Nitrate of amyli inhaled during the aura has prevented the attack.

*Oenanthe crocata* and *solanum carolinensis* have proven useful in my hands. *Gelsemium* must not be overlooked when selecting agents for this lesion. Each case needs careful study and the various conditions met as they arise,

## **INFANTILE CONVULSIONS.**

**Synonym.**—Eclampsia.

**Definition.**—Convulsions in children, similar to those of epilepsy, are of

frequent occurrence, but differ from the latter in that when the cause is removed, the convulsions cease.

**Etiology.**—The causes giving rise to convulsions are many, and present a wide range in variety; one of the most common being gastrointestinal irritability, either as a result of inflammation or overeating, especially unripe fruit or other indigestible substances. Dentition is another fruitful source of infantile eclampsia, especially when occurring in nervous children. Intestinal parasites not infrequently are the exciting cause, the *as-caris lumbricoides* being peculiarly exciting. An exciting cause, frequently overlooked, is an elongated or adherent foreskin, or the clitoris may be the source of the irritation.

The infectious diseases are often announced by a convulsion, cerebro-spinal fever, measles, scarlet fever, small-pox, and pneumonia particularly. Brain lesions, either organic or functional, are commonly preceded or accompanied by convulsions. Spasms occur in many rickety children. Following scarlet fever, uremic convulsions are occasionally seen.

In peculiarly nervous children, a severe fright is sufficient to bring on an attack. Convulsions occasionally occur in the new-born, especially where the delivery has been instrumental.

**Pathology.**—Unless due to meningeal hemorrhage, tumors, exudates, or hydrocephalus, there are no anatomical changes.

**Symptoms.**—Frequently there are premonitory symptoms, such as flushed face, general irritability, gritting of the teeth, twitching of the muscles, and *closing the fingers over the thumb*; or the attack may come on suddenly without any warning. It frequently begins in the right hand, to be followed by general convulsive action. At first there is rigidity, the eyes stare and are fixed, the body is stiff, or the head drawn backward and the respiration almost suspended, the child assuming a cyanotic appearance. This stage is but momentary, when clonic spasms follow, all the voluntary muscles being involved. There is rolling of the eyes, jerking of the limbs, and more or less contortion of the face. During the convulsion, the tongue is frequently bitten, and a bloody froth collects upon the lips. Sometimes the urine and feces are passed involuntarily. The attack usually lasts but for a moment or two, but to the frightened mother it seems an hour. The subsidence of the

attack is followed by a sound sleep, or the child passes into coma.

**Diagnosis.**—This is very easy ; in fact, is generally diagnosed by the mother, and announced to the physician by the frightened messenger who is hastily started for the doctor.

**Prognosis.**—This depends upon the cause. Unless due to organic changes, or occurring after the child has been sick for some time, as in cholera infantum, the prognosis is favorable. A child in good health, taken with convulsion rarely ever dies from an attack.

**Treatment.**—To overcome the convulsion, a few inhalations of chloroform is the most reliable agent that can be employed. As soon as the convulsion subsides, we commence the administration of remedies to prevent a return of the spasms. If called shortly after a meal, notwithstanding the protestations of the mother that the child has had nothing to eat, it is a good plan to administer an emetic, for it rarely ever fails to give relief. When. due to an overloaded bowel, a dose of castor-oil and a large enema will be followed by good results. When not due to either of the above causes, a careful search must be made for the exciting cause. Gelsemium, lobelia, and passiflora will be the remedies most frequently used when called to a child in convulsions. Where a convulsion announces the onset of a fever or one of the infectious diseases, there is generally no special treatment needed, for the convulsion is rarely repeated.

### **ACUTE CHOREA.**

**Synonyms.**—St. Vitus's or St. Anthony's Dance; Sydenham's Chorea.

**Definition.**—An acute disease affecting children mostly, and characterized by irregular, involuntary, and clonic spasms of the muscles, a tendency to endocarditis, a variable amount of psychical disturbance, and a liability to recurrence.

**Etiology.**—Among predisposing causes may be mentioned age, sex, and race.

**Age.**—The disease occurs most frequently between the ages of five and fifteen, puberty being especially favorable to its development.

*Sex.*—Of five hundred and fifty-four cases reported by Osler, seventy-one per cent were in females, and twenty-four per cent in males, the ratio rising still higher in females after puberty.

*Race.*—It is peculiarly a disease of the white races, rarely ever occurring among the colored races.

*Heredity.*—An effort has been made by some to show a hereditary tendency in the disease, but the fact that only about ten per cent of all choreic patients can show a family history of chorea, makes this quite a doubtful etiological factor.

*Rheumatism.*—The same may be said of rheumatism, and though English statistics show quite a large per cent of rheumatism in chorea, the history of other countries does not bear out the same conclusions, and it is probable that where the two are found in the same patient, it is a coincident.

*Endocarditis.*—Some observers claim endocarditis as a cause, due to the lodgment of vegetations from the valves in the cerebral vessels. In the large majority of cases, however, it is the result rather than the cause.

*Infectious Diseases.*—Scarlet fever, whooping-cough, pyemia, puerperal fever, and gonorrhoea have preceded the disease, but it is very doubtful if they have any significance as an etiological factor.

*Reflex irritation,* while a possible cause, has probably been overrated, though, in one of my cases, the release of an adherent clitoris was followed by a rapid cure, where medication had failed.

*Pregnancy* frequently is the exciting- cause, in one of a neuropathic temperament.

The cause, then, is elusive, and in children of a nervous temperament may arise from fright, shock, mental or emotional excitement.

**Pathology.**—No constant or characteristic lesion has been found, the pathology still being obscure. Some believe a functional disturbance of the centers that control the motor apparatus is the chief lesion, while others believe the lesion to be embolic, and still others the result of infection.



**Symptoms.**—In the beginning the affection is generally unilateral, though both sides are soon involved. It usually begins with slight involuntary twitching of the muscles of the hands or face. It is noticed that the child spills liquids when attempting to drink or pass them at the table, or that he lets articles drop, and this being attributed to carelessness, he is often scolded or punished, which only aggravates the disease. This twitching or rather jerking of the muscles, extends to the shoulders, which are twisted or raised in a jerky manner. The involuntary movements may extend to the leg, making locomotion in severe cases quite difficult.

In the more severe cases, the child presents a pitiable appearance; there is batting of the eyes and jerking of the mouth, the hands are almost in a constant spasm, the legs jerk irregularly, and the child is unable to feed or dress himself, and many times is even unable to walk. The child's disposition seems completely changed, becoming cross, peevish, and with sudden outbursts of anger, especially if corrected for his seeming carelessness.

In severe cases his speech is affected, articulation being so difficult that often no attempt is made to talk. Generally the involuntary movements cease during sleep, though in the most pronounced cases the rest is disturbed even in sleep. Occasionally nocturnal incontinence occurs.

The appetite becoming impaired, the child frequently becomes weak and anemic, and where endocarditis occurs, a blowing murmur may be heard in the apex region.

As a rule, there is no fever unless complications exist.

**Diagnosis.**—Few diseases present more characteristic symptoms than chorea, and the laity generally diagnose the disease even before the physician is consulted. The peculiar twitching and jerking of the muscles of the face and hands, extending to other parts of the body, is apparent to every one, and the diagnosis becomes very easy.

**Prognosis.**—This is favorable in the large majority of cases, though there is a tendency to one or more relapses. The disease runs from four to ten weeks, though sometimes it may linger three or four months.

**Treatment.**—The child should be dealt with kindly, and little attention paid to his contortions; for this reason he should be removed from school as soon as recognized, that he may be saved the pain of thoughtless laughter and remarks by fellow playmates.

The diet should be nourishing though easily digested.

In the way of medication, there are a few remedies that have proven of great value.

*Macrotys.*—Where there is muscular soreness, whether due to acute rheumatism or muscular contraction, macrotys will give good results. From a half to one dram of the specific medicine added to four ounces of water, and a teaspoonful given every two or three hours, will not disappoint.

*Gelsemium.*—Where the child is extremely nervous, gelsemium will be the better agent.

*Cypripedium.*—This agent has been used quite successfully by Eclectics for this troublesome affection. A broad, puffy tongue is the special symptom. It should be given in quite tangible doses, ten to fifteen drops every two hours.

Physostigma, scutellaria, avena, cannabis, hyoscyamus, and like remedies, should be used.

Dr. Webster speaks highly of the use of the tonic faradic or secondary current of electricity.

Should there be no improvement in a carefully selected medication, the clitoris should be examined for adhesions, and released when present. I seriously object to subjecting young girls to any unnecessary examinations, and since many of these cases yield to medication, I should only resort to this measure when other means failed. After treating a severe case of chorea in a girl of twelve, for two weeks without any improvement—in fact, there was rather an increase in the severity of the symptoms—I found a *very tightly adherent clitoris*, the release of which was followed by speedy relief. One could hardly believe so great a change as took place within twenty-four hours; but since these are exceptional cases, do not examine every girl that has chorea,

till other measures fail.

## **PARALYSIS AGITANS.**

**Synonyms.**—Shaking Palsy; Parkinson's Disease.

**Definition.**—A chronic disease of the nervous system, characterized by rhythmical contractions of the muscles of the limbs, associated with weakness and rigidity.

**Etiology.**—This is a disease of adult life, rarely ever occurring before forty, the largest number appearing between the ages of forty and sixty. It occurs more frequently in men than women, the ratio being about two to one. Heredity seems to bear no causal relation other than to furnish a neurotic temperament. Acute diseases, cold, fright, great emotion, worry, mental strain, and injuries have been considered as producing causes, though no definite cause is known.

**Pathology.**—No characteristic lesions are found, though, in all probability, the disturbance is in the cerebral cortex. Some regard the degeneration of a senile type. Certain sclerotic changes have been described in the spinal cord, starting from the neighborhood of the vessels. Nothing, however, is positively known of the nature of the lesions.

**Symptoms.**—Except where the result of fright, the disease generally comes on so insidiously that the patient is unable to say when the first symptom began. The first evidence is usually a slight tremor in the muscles of the fingers and hand, gradually extending to the arm and leg of the same side. The right side is the first to be affected as a rule, then both become involved. The motions of thumb, fingers, and wrist are characteristic, the forefinger sliding over the ball of the thumb, while the wrist is semi-rotated. If the head be involved, there is a constant nodding motion. The tremors are usually suspended during voluntary motion, at once to resume when such action ceases. The tremors are constant during waking hours, save in the exception just noted. During sleep there is a suspension of the tremblings.

It is noticed as the disease progresses, that voluntary movements are slow and awkward, the muscles appearing stiff, another characteristic of

the lesion.

The *attitude* and *gait* of the patient is most striking. The head is thrown excessively forward, the eyes straight away or slightly elevated, and the face passive. In walking, the patient takes very short steps, and *trots* along, hurrying one foot after the other, seemingly to prevent pitching forward. The knees are apt to touch or rub, while the feet may be slightly everted, or they may cross.

The muscles show no evidence of atrophy or trophic changes, even in the advanced stages. The electrical irritability of the muscles is retained, and the reflexes remain normal. The bladder and rectum are usually unimpaired.

The *facial expression* of victims of “shaking palsy” is also characteristic, and, because of so little play of the features, is as though the patient wore a mask. In some cases the mouth is kept open, the saliva dropping constantly from the mouth. The speech is usually slow, monotonous, and the voice tremulous and high-pitched.

**Diagnosis.**—The symptoms are so characteristic that one can scarcely be mistaken in the diagnosis. We recognize it from *multiple sclerosis* in that the tremors are not increased by voluntary movements, that it usually begins in the upper extremities, that there is no oscillation of the eyeballs, and that there are no sensory disturbances. From *chorea*, by the marked regularity of the tremors, from four to six per second, by their persistency even during sleep, and that the tremulous condition of the muscles is not increased by voluntary movements.

**Prognosis.**—The disease is incurable, though there is no immediate danger to life, the patient living for years. There may occasionally be intermissions of the tremors, though generally there is a slow advance in the disease. Death usually occurs from some intercurrent disease.

**Treatment.**—Fowler's solution, hyoscyamus, and phosphid of zinc have each had their advocates as beneficial remedies in this disease, though their influence is questionable. The patient should avoid, as far as possible, all mental excitement and excessive fatigue of the muscles. The diet should be as nourishing as possible, though one that is easily digested, and the general health improved when impaired.

## MIGRAINE.

**Synonyms.**—Hemicrania; Sick Headache.

**Definition.**—This is a neurosis characterized by severe paroxysmal attacks of headache, and generally attended by nausea and vomiting.

**Etiology.**—Females are more frequently affected than males, and those of a nervous temperament rather than those of a sanguine or lymphatic type. The disease shows strong hereditary tendencies, usually on the mother's side. It seems to be closely associated with wrongs of the female reproductive apparatus; thus menstrual derangements and ovarian disturbances go hand in hand with migraine.

Among the exciting causes may be mentioned gastro-intestinal disturbances, naso-pharyngeal wrongs, dental irritation, emotional excitement, grief, shock, eye-strain, or anything that tends to impair the nervous system of those predisposed. An important and often overlooked cause is peripheral irritation, as seen in rectal diseases, urethral lesions, and the nerve waste that follows lacerations of the uterine cervix and perineum.

**Pathology.**—There is no characteristic lesion found, and the pathology is therefore obscure. Various theories have been advanced, but none proven. Vaso-motor disturbances are thought to be the chief wrongs, while arterio-sclerosis has been found present in others.

**Symptoms.**—There, are generally premonitory symptoms that give the warning note. These vary, and may consist of malaise, chilliness and flashes of heat, or dizziness. Sometimes there is mental depression and confusion of ideas, or the opposite condition, that of excitation. Not infrequently there will be perverted vision, as flashes of light, zigzag lines, balls of fire, or visions of animals. Numbness of face and tongue, followed by pallor, may usher in an attack.

The pain is generally unilateral, the left side being more frequently affected, though, if long continued, the whole head becomes involved. A favorite starting point is over the eye, gradually extending as the disease grows more severe. There may be tenderness over the affected region, and sometimes twitching of the muscles. In most cases the

headache is accompanied by gastric disturbances. It may be extreme nausea without vomiting, or there may be persistent emesis. Not infrequently a short period of unconsciousness supervenes. Permanent local gray-ness of the hair is not uncommon where the paroxysms have been of unusual severity.

The duration of the attack varies, usually subsiding in twenty-four hours, though it may last two or three days. An attack causes great prostration, which soon passes away after the paroxysm is over.

The disease continues for years, and sometimes for life, though it is apt to disappear after the age of fifty.

**Diagnosis.**—This is readily made by the history, of the case and the symptoms already described.

**Prognosis.**—So far as life is concerned, the prognosis is favorable, and where due to reflex causes and they can be removed, many will permanently recover.

**Treatment.**—In all cases of migraine, a careful search should be instituted for the cause. It so frequently arises or is continued by irritation at the various orifices, that the rectum, urethra, vagina, uterine cervix, and nasal passages should be examined. Any wrongs must be corrected if we are to expect any permanent benefit.

The patient should be placed on a dry diet, which should consist principally of fruits, cereals, and vegetables. Constipation should be overcome.

For the attack, where the pain is intense and the heart's action good, a five-grain antikamnia powder will usually give relief. I have one patient that only obtains relief by a hypodermic of a quarter grain of morphia. Passiflora in half-teaspoonful doses will sometimes give relief. In some of these patients there seems to be a rheumatic or uric-acid diathesis, and the salicylates and anti-rheumatics will be indicated. Where due to malaria, arsenate of quinia, gelsemium, chionanthus, and uvedalia should be tried. Dr. Webster speaks highly of melilotus in such cases. With the first prodromal symptoms, a thorough washing out of the stomach will frequently abort the attack or materially lessen its force. A thorough emetic is the best means of accomplishing this object.

## HYSTERIA.

**Definition.**—A functional disturbance of the general nervous system in which there is “an abnormal susceptibility to external impressions, and a deficient power of the will to restrain its manifestations,” and characterized by a multitude of symptoms that simulate many diseases.

**Etiology.**—Among the predisposing and exciting causes may be mentioned:

*Sex.*—About nine hundred and fifty cases out of every thousand occur in females.

*Heredity.*—A very large per cent of all cases of hysteria occur in families of neurotic tendencies, such as epilepsy, insanity, chorea, sick headache, neuralgias, and allied conditions.

*Age.*—Hysteria usually develops between the age of puberty and twenty-five, few cases occurring after the age of forty.

*Psychical Influences.*—In neuropathically inclined patients, fright, especially of a severe kind, like railroad accidents and fire, great shock, fear, love, jealousy, disappointments, anxiety, melancholy and remorse, are apt to be followed by hysteria.

*Environments.*—Girls reared in luxurious city homes, with but little responsibility, and who are petted, pampered, and reared amid excitement and dissipation, are far more subject to hysteria than those reared in the country, and who are taught to be useful and share some of the responsibilities of life.

Sexual derangements are found to underlie many cases of hysteria. Irregular menstruation, uterine displacements, ovarian irritation, ulceration of the cervix, an adherent clitoris, a vaginitis leucorrhœa, and not infrequently masturbation, are found on examination.

Ranney believes eye-defects and muscular insufficiencies in the orbit attend nearly all hysterical patients.

**Pathology.**—Hysteria is a purely functional disturbance of the

nervous system, no anatomical lesions being found in this disease.

**Symptoms.**—The clinical picture of hysteria embraces such a varied and complex list of symptoms that it will be impossible to name them all. For convenience of description the symptoms may be divided into mental, sensory, motor, and visceral.

*Mental Condition.*—One of the characteristic conditions of hysteria is a defect of the will and an excess of the emotions. These patients crave sympathy, and are peculiarly self-conscious. They readily give way to joy or grief, the effects of which, laughter and tears, are indulged in to excess. Uncontrollable laughing or crying for hours is sometimes witnessed. Such patients, when sick, make no effort to get well, preferring the sympathy of friends to convalescence, and not infrequently so skillfully magnifying their symptoms that they not only succeed in deceiving their friends as to their true condition, but the physician as well. Sometimes they will produce artificial lesions by the application of irritants producing various eruptions, or, as in one case that came under my observation, the patient had produced red spots on the palms of the hands by the use of a red rose on her hat, and so skillfully did she hide her work and so persistently did she deny all knowledge of the cause, that her parents sent her to Seton Hospital for treatment.

Finding, however, that the physicians were not as gullible as her parents, and that the treatment suggested was not to her liking, she confessed to the part she had played in the deception. Some patients, with a perverted sense of delicacy, show concretions or bodies, and insist that they have passed the same from the bladder or rectum.

*Sensory Symptoms.*—Hyperesthesia may manifest itself simply by tenderness on pressure over sensitive points, or it may arise as spontaneous pain. While hyperesthetic spots may be found in any part of the body, they are rare on the extremities. A favorite location is the vortex, where the pain is frequently agonizing in character, and is known as *clavus hystericus*. Other favorite locations are over the sternum, under the mammae, over the ovaries, and along the spinal column. These sensitive spots or zones are known as "*hysterogenic points*." These painful points may involve the entire spinal column or only a single point, and a simple touch may cause exquisite pain. Abdominal hyperesthesia may resemble gastric ulcer, appendicitis, or



peritonitis. The hysterical breast is recognized by the great tenderness, by constant variations in the swelling, and by “the recurrence of the symptoms at the usual period or after exceptional excitement or fatigue.”

The special senses may be affected, as hyperesthesia of the eye or ear, or sight may be limited or even lost for certain colors, while the loss of the sense of taste, smell, or hearing is not infrequent, or the opposite condition, extreme sensitiveness to sound, taste, and smell may be present.

Anesthesia, or loss of sensation in a limb or entire side is known as *hemianesthesia*, and is quite significant as a diagnostic factor. “Sensation has sometimes been restored to a part by the local application of metal plates, especially iron, copper, zinc, or gold (*metallotherapy*), and under these circumstances anesthesia may be found at the corresponding spot on the previous healthy opposite side of the body.”

*Motor Symptoms.*— “*Paralysis.*—Hysterical aphonia is not uncommon ; it results from paralysis of the adductors of the vocal cords. It is important to note that adductor paralysis, without abductor paralysis, is always a functional disorder. On the other hand, abductor paralysis, existing alone, is mostly due to organic disease, and very rarely the result of hysteria; it causes stridor and dyspnea, and may even in hysteria lead to dangerous asphyxia. Dysphagia may arise from functional paralysis of the pharyngeal muscles. Ptosis also occurs as a hysterical symptom; it may be single, or double. Paralysis of the limbs occurs in the form of paraplegia or hemiplegia, or all the limbs may be paralyzed together. The paralysis in these cases is often not complete, and if the patient makes an effort to move the limb in a particular direction, it may be seen that some antagonistic muscles contract. The patient may assert that she is unable to lift the arm, yet if it is raised by any one else she will often keep it supported or let it drop only half-way, showing that the muscles believed to be paralyzed have still a considerable amount of power. Also, if the attention is directed to other things the patient may unconsciously move the supposed useless limb. If one lower extremity is alone affected, on attempting to walk the patient drags the paralyzed limb behind her, making no effort to bring it forward, but only hopping along on the sound limb. The nutrition of the muscles and the electrical reactions are generally normal, but wasting of muscles is sometimes observed. Knee-jerks are generally normal, and there is no true

continuous ankle-clonus, but there is often a clonus lasting only a few seconds, and in some cases the knee-jerks are excessive. In paraplegia the legs can often be moved in bed, but the patient is quite unable to stand, and there is never incontinence of urine or feces; in hemiplegia the leg is sometimes worse than the arm, and the face and tongue are always spared. Paralysis is sometimes, but not always, accompanied by anesthesia. In a rare form of hysteria, every attempt to move, or contract a muscle, is painful (*akinesia algera*). Another form of hysteria is a disorder called *astasia-abasia*, in which the patient can neither stand nor walk, though he can move the legs in bed, and there is neither inco-ordination nor sensory failure." (Taylor.)

*Convulsive Seizures.*—Following excitation of the emotions the patient becomes hysterical, laughs or cries, and complains of a ball in the throat (*globus hystericus*), which in turn is followed by convulsions. The muscular spasms are clonic and irregular. Gradually the attack subsides, with the passage of a large quantity of limpid urine. The convulsions sometimes simulate true epileptic attacks, though the tongue is not bitten. "The convulsive seizure is usually followed by emotional displays, by cataleptic poses, by opisthotonos or other distortions, and by attitudes and grimaces expressive of the deepest emotions." Following this, the patient may go into a trance, or hallucinations and delirium may follow.

*Contractures and Spasms.*—Tonic contractions may affect one side, hemiplegic in character, or from the waist down (paraplegic), or be confined to a single part (monoplegic), as in hysterical contraction of the jaw (trismus). Where one side is affected, the arm is generally flexed at the elbow, while the leg is rigidly extended. The contractions usually disappear during sleep; but in exaggerated cases, only the deepest chloroform narcosis is sufficient to produce relaxation.

Phantom tumors, simulating pregnancy, are sometimes seen, and are due to contraction of certain abdominal muscles and relaxation of the recti, permitting the inflation of the intestines with gas.

Clonic contractions are generally rhythmic in character, and known as "rhythmic chorea" ("hysterical chorea").

*Visceral Symptoms.*—*Globus hystericus*, the sensation of a ball rising in the throat, is often associated with spasm of the pharynx and

esophagus, rendering deglutition difficult or impossible. Gastric disturbances are quite common, and furnish a variety of symptoms. In one the food is regurgitated soon after eating, there being little or no nausea; this may continue for months with but little emaciation; in another there will be hysterical anorexia, the patient persistently refusing food for days, and growing quite emaciated. In these cases, however, there is nearly always deception practiced, the patient eating clandestinely. Another will have a perverted or depraved appetite, eating undesirable and unwholesome food. In rare cases there is reversed peristalsis, the patient vomiting rectal enemas, or there is peristaltic unrest. While diarrhea may be present, constipation of a most persistent type is the rule, and it is not infrequent to find such patient going a week or ten days without stool.

*Respiratory symptoms* are quite frequent, the patient breathing from forty to one hundred per minute, without change in the frequency of the pulse or evidence of dyspnea. A dry, spasmodic, or barking cough is a not infrequent symptom.

*Circulatory disturbances*, such as the “irritable heart” and palpitation, are exceedingly common, while pallor, local flushings, and pseudo-angina occasionally are seen.

*Urinary symptoms* are nearly always present. After an attack of hysteria the urine is abundant, limpid, and of low specific gravity. Suppression, hysterical anuria, may last for several days, yet uremia fail to appear.

“*Trance or lethargy* is, like catalepsy, sometimes the result of hysteria, or of exhausting illness, or of hypnotism. The patient is in a peculiar condition resembling sleep, and may remain so for days or even weeks. The face is pale, the limbs relaxed and the eyelids resist efforts to open them. The pupils are moderately contracted or dilated, and react to light. The pulse is small, the heart-sounds are feeble or inaudible, and the breathing- is extremely quiet, so that occasionally the patient has been thought to be dead. In prolonged cases there are remissions in which the patient may take food, relapsing again into stupor. Most cases recover. *Double consciousness and somnambulism are other developments of hysteria.*” (Taylor.)

**Diagnosis.**—The family history should throw some light upon the case.

Thus, if similar conditions have occurred in the family or near relatives, or they have had insanity, chorea, epilepsy, sick headache, or neurasthenia, and if the patient has been excitable from early childhood, and but little effort has been used to control excessive emotions,—if with such a history the various symptoms above noted be present, there can be but little doubt left as to the true condition.

**Prognosis.**—This is always favorable as to life, and nearly always as to a cure of the disease, though years may elapse before this is accomplished. Usually after the menopause, the disease disappears.

**Treatment.**—There is no class of patients that receive as little sympathy as the victims of hysteria, and an important part of the treatment is patience on the part of the attending physician. While they are not to be humored—in fact, the physician should exercise firmness with his patients—they should not be allowed to observe annoyance or disgust or disinterestedness on the part of the medical adviser. Confidence in the physician must not be shaken. Hypnotism or suggestion will prove more or less beneficial in nearly all cases. Where possible, the patient's environments should be changed and travel, with congenial company, often works wonders in the way of a cure. The patient's mind must be gotten away from self, and anything that will accomplish this will be beneficial. A visit of several weeks' duration will often answer the same purpose.

The excitement and dissipations of city life also tend to aggravate hysteria, and a change should be made from the city to the country, where quiet, fresh air, plenty of sunshine, a nourishing diet, and regular hours can be secured.

The exciting cause must be determined and removed before much benefit can be expected, and a most thorough examination should be made of the rectum, where pockets, papillae, fissures, ulcers, hemorrhoids, undue contraction of the sphincters, or redundant and prolapsed tissue may be found responsible for the lesion. The uterus, both body and cervix, the vagina, the perineum, the ovaries, and the urethra should be carefully inspected; for the *cause* of the trouble *very often* is found at these parts, a correction of which often gives instant and permanent relief.

In the way of medication the treatment will be symptomatic, meeting

the various conditions as they arise. The remedies most frequently indicated are pulsatilla, passiflora, viburnum, gossypium, gelsemium, ammoniated tincture of valerian, scutellaria, cypripedium, and like remedies, which should be studied with reference to this condition.

The rest-cure will be highly beneficial in many cases, while faradic and static electricity is followed by good results. Sometimes the dread of unpleasant medication is sufficient to act as the restraining- cause, till a cure is effected. Thus one cure comes to my mind where my preceptor, with the old lobelia emetic, effected a cure. At the first call the emetic was given for its full effect, two hours or more being required in its administration. A second attack was treated in the same way, and the interval between attacks lengthened. At the third call, while getting ready the nauseating mixture, the patient, with a woe-be-gone disgusted look, said, "Doctor, don't you give nothing but pukes?" "Nothing else for your kind of sickness," said the doctor ; "but I want to assure you that you *will never have to take another*, for this is the *last attack* you will ever have." The cure was complete. The dread of the emetic, together with the mental impression, "*You will never have to take another*," was sufficient to arrest the disease in this case.

In nervous children the treatment should be prophylactic. They should not be advanced too rapidly in their school-work, should be taught self-restraint, not allowed to have every whim gratified, should have but few cares during the stage of puberty, and all dissipations of city life carefully avoided till full maturity, and but few cases will occur.

## NEURASTHENIA.

**Synonym.**—Nervous Prostration.

**Definition.**—A chronic functional disease of the nervous system, characterized by mental and physical exhaustion.

**Etiology.**—Like most functional nervous lesions, certain predisposing factors play an important part, chief of which may be mentioned heredity, occupation, age, and sex.

**Heredity.**—All parents suffering, not only from nervous derangements, such as irritability, degenerations, etc., but also from syphilis,

tuberculosis, rheumatism, and kindred affections, bequeath to their offspring a feeble vitality and feeble resisting power which are favorable to neurasthenia.

*Occupation.*—Any profession or calling that entails great mental strain, and where large responsibilities are assumed, favors neurasthenia.

*Age.*—Neurasthenia comes with maturity, when the cares and anxieties of life press heavy; hence is rarely seen before twenty nor after the age of fifty-five or sixty, when the burdens are shifted to younger shoulders.

*Sex.*—Unlike hysteria, the majority of neurasthenics are found among men.

*Exciting Causes.*—Overwork exhausts not only physical but mental power as well, and the great competition and tension that prevails in all lines of work and business, necessitates a great tax on the mental power, and often results in overwork. Whenever a man takes his business cares home with him, dines with them and sleeps with them, he is on the road that leads to neurasthenia. Sexual excesses and masturbation exhaust nerve force, and are a fruitful cause of the disease.

Orificial irritation is one of the most common exciting causes, and one that is perhaps the most frequently overlooked. Rectal lesions are prolific causes of nerve waste, while urinary wrongs sap the vitality and give rise to prostration.

Unhappy marriages are not an infrequent cause of neurasthenia in both sexes, while fear of infectious diseases may so wear upon the nervous system as to give rise to the disease. Various accidents, especially those occurring on railroads and on the water, may cause the disease, and is known as traumatic neurasthenia.

**Pathology.**—There are no characteristic pathological changes in this disease, and about all that can be said of the lesion is, that it consists of irritation of the nerve centers, followed by more or less weakness.

**Symptoms.**—The symptoms are so varied and complex that it will simplify their narration to group the cases according to the predominance of certain phenomena; thus we have the cerebral, the

spinal, the gastro-intestinal, the cardiac, the urinary, and the sexual varieties.

The general symptoms are great irritability, marked despondency, great prostration, both physical and mental, without sufficient cause, and loss of weight. So many are the symptoms that the patient, fearing that some may be forgotten, presents a long list of symptoms carefully written down.

*The Cerebral Variety.*—Headache is a prominent symptom, there frequently being tenderness of the scalp. Insomnia is quite characteristic, and the patient arises unrefreshed; there is more or less despondency; the patient is anxious, worried, and fearful of some impending danger. There is impairment of memory, and reading seems to tire the patient; in fact, a continued tired feeling is generally present.

*The Spinal Variety.*—Great weakness and prostration is a common symptom, the patient complaining of feeling sore and stiff on rising. Backache, with tenderness along the spine, is characteristic, while there is perverted sensibility, manifested by a tingling, crawling, or burning sensation, or certain parts will feel hot or cold; sometimes there will be lightning pains, simulating locomotor ataxia. Ankle clonus is sometimes present, while the reflexes are exaggerated.

*Gastro-intestinal Variety.*—Gastric disturbances, with their attendant symptoms, are the chief characteristics of this form. Hyperacidity, waterbrash, nausea, retching, and vomiting, with more or less headache, are present. The patient sleeps poorly, has unpleasant dreams, and develops an irritable disposition. There is flatulency, rumbling of the bowels, constipation alternated with diarrhea, and a sense of weight or soreness over the abdomen.

*The Cardiac Variety.*—While there is an absence of organic lesions, there is palpitation on slight exertion, precordial distress, and sometimes sharp pain as in angina. Throbbing of the abdominal aorta is a distressing symptom and one that causes much anxiety on the patient's part. In some cases a capillary pulse may be detected. This group of symptoms causes the patient to become fearful of a sudden termination of life. Loss of flesh is apt to accompany this form.

*Urinary Variety.*—The quantity of urine voided is usually small, and

albuminuria, oxaluria, and glycosuria may be present. The patient becomes irritable, and there is a dull headache.

*Sexual Variety.*—A victim of this variety is often an object of pity. The fear of becoming impotent preys upon the mind, the patient is melancholy, sleeps poorly, has nocturnal emissions, complains of pain or crawling sensations in the testicles, has perverted sexual desires, and frequently masturbates. The patient, worried and distracted, leads a miserable existence.

**Diagnosis.**—This is not often difficult. The physical and mental prostration without undue exertion, the multiplicity of symptoms, with due prominence of some one of the above groups, makes the diagnosis one of but little difficulty.

**Prognosis.**—Where the patient has not become the victim of morphia, chloral, or alcohol, and where a rational treatment is early observed, the prognosis is favorable. Where the patient's mind can be centered on something" besides self, and where hope can be aroused, the patient will recover.

**Treatment.**—A careful search for the cause must be made, the removal of which starts the patient, many times., to a speedy recovery, the history of the following case being a good illustration:

Mr. A, aged forty-five, a prominent business man, had suffered many things, chief of which was a constant headache at the base of the brain. Failing to get relief from medication, galvanism, and massage, he was placed on the rest-cure as outlined by Weir Mitchell. He gave up his business for six months and devoted his time to the rest-cure. After six months of this treatment the pain in the head subsided, and he returned to his business, only to find a return of the pain in the occipital region. At this time I saw the patient, and obtained the above history. On examining his rectum (he was not aware of any trouble in this part of his anatomy), I found a rectal fissure, the cauterization of which not only stopped the headache, but effected a permanent cure. After a failure of six months' treatment by diet and rest, a single application of nitric acid to the anal fissure effected a cure.

Where orificial irritation is the cause of the lesion, and it is a very frequent cause, nothing in the way of rest or medication can take the



place of a removal of the cause.

The same may be said of overwork of an organ; it must have rest, and if it be the brain, but little result may be looked for unless mental rest is secured. Where sexual excesses are draining the system, the patient can not hope for relief if he continue his dissipations.

Many times a sea voyage gives the best of results, securing not only an enforced bodily rest, but the constant change (where the voyage is along the coast with frequent ports) furnishes an agreeable rest for the mind as well.

The rest-cure, as first introduced by Weir Mitchell, will many times be the only effective treatment. This may be modified to suit the individual case. The treatment consists in absolute rest (the worst cases not being allowed to get up to void urine, the feces, or even turn in bed without assistance), passive exercise, massage, diet, and, electricity. As the patient improves, baths are to be added. The treatment is to be continued from one to three months. A favorite diet in the beginning" is one glass of milk every two hours, gradually increasing the quantity till lie is taking a pint or more at each feeding. After two or three weeks, solids are added, which consist of fruits and vegetables, and finally chops, steak, and roast-beef. The faradic and static currents give the best results.

Where the patient is not very sensitive to shock, cold shower or sponge baths are attended by good results. As the patient grows stronger and takes on flesh, outdoor exercise should be taken, care being exercised not to carry it to exhaustion. Tennis, golf, rowing, and surf-bathing will prove of great benefit in stimulating the appetite, improving digestion, and establishing the excretions.

The patient should have cheerful surroundings, agreeable companions, and his reading should be of a light vein and a humorous style, especially avoiding the tragic and all that tends to excite the mind. In the way of medication, the treatment will be entirely symptomatic, only giving remedies when specially indicated. Avena, passiflora, rhus, and the compound tonic mixture will be frequently called for, while chronic constipation must be corrected by appropriate medication before much relief will be obtained.