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Introduction

For more than 60 years, following the work of William Garner Sutherland, the cranium has fascinated the osteopathic world. Being the mechanics that we are, we continue to be captivated by its 22 bones, articulated by sutures and linked through membranes. The fascination that this structure holds for us is perhaps one of the reasons why most cranial techniques address the skull — the container — rather than its contents — the brain.

In our book, *Manual Therapy for the Peripheral Nerves*, we underlined the importance of soliciting the body's proprioceptive responses through manual therapy. This is because all tissues are endowed with a system of mechano-receptors connected to the cerebellum and the thalamus, and it is through their agency that we obtain both local and general results.

Cranial nerves supply sensitivity to the bone, periosteum, sutures and meninges. Thus, central to obtaining an effect on the dura mater, the falx of the brain and the tentorium of the cerebellum, is the treatment of their nervous system. Manipulations of the cranial nerves add to, and prolong, the impact of our techniques for the cranial contents.

This book is dedicated to the most precious and mysterious part of the human being: the brain. The brain has the consistency of an egg custard and, although protected by the cerebrospinal fluid, is very sensitive to all variations in pressure. In this book you will find ways of modifying intracranial pressure.

The brain is shot through with arteries and veins. Intracranial pressure changes have vascular and cerebral consequences. To function at its best, the brain requires malleability and plasticity. By manually modifying intracranial pressure, we produce promising effects on the hydromechanics of the brain.

The central focus of this book is on the practical applications of cranial nerve manipulations. With its wealth of illustrations, it is also an excellent guide with which we can visualize the cranial nerves and come to understand their many functions.

Acknowledgments

What a treasure Jean-Pierre Barral and Alain Croibier have brought to the health-care field of Manual Therapy! In addition to being excellent therapists, inquisitive scientists and creative developers, they are also very prolific in their writing skills. This additional talent, too often lacking in creative therapists, allows them to share their discoveries, ideas and theories with the rest of the world. *Manual Therapy for the Cranial Nerves* is the follow-up to their highly acclaimed and successful *Manual Therapy for the Peripheral Nerves*. Both texts are having a positive influence on hundreds of thousands of lives.

It is truly an honor for me to have had the privilege of helping translate their most recent work into English. As with most projects I've been involved with, this translation required a mix of administrative, technical and supportive efforts. Administratively, I would like to thank Dawn Langnes, whose tireless efforts helped make this whole project happen.

I would also like to thank Elsevier, our publisher, for their continued commitment to sharing this work with therapists throughout the world.

On the technical side, several people should be acknowledged for their tremendous work. Annabel Mackenzie RST tackled the translation of the textbook into English. Susan Railer DO, RMT also assisted with the translation process. Annabel Mackenzie RST, Dawn Langnes and Emmanuel Frantzis, DC, DO edited the manuscript into the form you see in your hands.

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John Matthew Upledger

Some preliminary thoughts

1

Every nerve must be free to act and do its part.

Andrew Taylor Still

Before attempting to manipulate the cranial nerves, the reader is advised to study the properties of nerves by consulting our book, *Manual Therapy for the Peripheral Nerves*.

For readers who have no previous experience with peripheral nerves, we would like to summarize briefly some essential tenets.

1.1 FUNCTIONAL ANATOMY OF NERVES

Nerve fibers are grouped into functional histological units taking the form of fascicles surrounded by layers of connective tissue. Connective tissue comprises 50–90% of the total mass of a peripheral nerve.

1.1.1 Connective tissue of nerves

The connective tissue component of peripheral nerve trunks divides into endoneurium, perineurium and epineurium, according to their topography.

Endoneurium

Endoneurium is the intrafascicular connective tissue. It is composed of several nerve fibers making up a primary fascicle. Glial cells, to which they are attached, provide each nerve with additional reinforcement. The endoneurial tube is a distensible elastic structure composed of a dense matrix of collagen tissue, which has a nutritive and protective function. Endoneurium plays an important role in fluid pressure. By maintaining a light positive pressure in the endoneurial space, it guarantees a constant environment for the nerve.

Perineurium

Perineurium is the connective tissue sheath that surrounds several primary fascicles. When grouped together, clusters of primary fascicles form secondary fascicles. The perineurium comprises 7 or 8 layers that are dense with fibroblast cells. The perineurium serves to:

- protect its contents: the endoneurial tubes
- provide a diffusion barrier to guard against foreign substances
- provide resistance to external forces.

The perineurium is the last connective tissue layer to break when a nerve is subjected to extreme traction.

Epineurium

Epineurium is the interfascicular connective tissue layer. It lies between and around the secondary fascicles and forms a sheath encircling all peripheral nerve trunks. The epineurium is a continuation of the dura mater that extends to the ends of the peripheral nerves. Functionally, the epineurium has a double role:

- The internal epineurium keeps the fascicles apart.
- The external epineurium forms a well-defined sheath around the fascicles.

The epineurium is a vessel carrier, containing the vasa nervorum which constitute the microvascularization of the nerve. The epineurium is also a nerve carrier, containing the nervi nervorum. These nerve fibers extend from the nerves themselves and from the perivascular plexus. They are destined for the epineurium, the perineurium and the endoneurium.

The epineurium is a structure devised for motion. It facilitates gliding between fascicles.

1.1.2 Vascularization of nerves (vasa nervorum)

Within the epineurium the vascular network is very well developed. The majority of arterioles and venules are arranged longitudinally and are widely anastomosed.

Within the perineurium exists a vast plexus of capillaries as well as an important network of arterioles and venules.

Within the endoneurium the capillary vessels are similarly organized in plexuses. The number of venules is, as a rule, slightly greater than the number of arterioles. Empty capillaries are frequently found inside the endoneurial space; their effect is to isolate part of the vascular bed.

These excluded capillaries resume their function as soon as the nerve is lightly mobilized. Our neural manipulations have an immediate vascular effect.

1.1.3 Innervation of nerves (nervi nervorum)

The connective tissue of the peripheral nerves, the nerve roots and the autonomic nervous system possess their own nerve supply. This innervation is provided by the nervi nervorum, which derive from local axon branches. These are the "nerves of the nerves" and ensure intrinsic sensitivity. At the mechanical, metabolic and trophic levels, the state of the nerve is under continuous supervision and control. The nervi nervorum are vital elements in the transmission of information at the level of the medulla and the central nervous system.

The nervi nervorum are made up of an afferent track, which participates in the intrinsic sensitivity of the nerve and is often implicated in any pathological process affecting peripheral nerves. In response to diverse stimuli (chemical, electrical and mechanical), the nervi nervorum behave like primary nociceptors. They release into their environment, prostaglandins and neuropeptides which are involved in the inflammatory process.

When a tissue suffers damage, the nociceptor response is initiated. The damage may not be severe enough to cause identifiable pain, but is nevertheless sufficient to disturb the regional biomechanics and to create a protective response ensuring that the damaged nervous area does not become overloaded.

Note: Nociceptive nerve endings are more responsive to stretching than they are to compression.

The whole art of neural manipulation lies in applying an effective amount of traction without an excess of compression.

There is also an extrinsic vasomotor innervation for the vasa nervorum. It is provided by the fibers that penetrate the nerve at the level of the perivascular plexus. The blood vessels of the perineurium and epineurium are innervated by the sympathetic nervous system.

1.2 MECHANICAL PROPERTIES OF NERVES

1.2.1 Mobility

Nerve tissue is subjected to the numerous demands imposed upon it by movements of the body. According to the circumstances, it can adapt to the constraints in two ways:

- by gliding within its anatomical environment
- by adapting its shape in such way as to diminish itself (by creasing, furrowing or folding) or to allow itself to be stretched.

1.2.2 Visco-elasticity

Visco-elastic materials, when subjected to a strain force, respond by instantly deforming; on returning to their original form, they retain a little of the deformed shape. This differs from elasticity, which is the ability of a material to regain its original shape immediately after the force is removed.

A nerve is a visco-elastic structure. As such, it permanently displays some internal constraint. The level of tension perpetually varies according to the external stress applied to it. When a nerve is subjected to forces of traction, visco-elasticity is its trump card, lending it great adaptability and resistance to being overstretched. On the other hand, a nerve is extremely vulnerable to all forces of compression, even mild ones. In our work we use the quality of visco-elasticity to further slow down the nerve's naturally delayed return to form.

1.2.3 Neural constraints

Intrinsic intraneural pressure

Intrinsic intraneural pressure represents the sum of all intracellular axon pressures, augmented by the intravascular pressure within the vasa nervorum. It is further modulated by intrafascicular pressure. The visco-elastic property of the nerve exposes it to longitudinal, centrifugal and distal tension; any variation in its intensity alters the intrinsic intraneural pressure.

Extrinsic intraneural pressure

A nerve is subject to the pressure surrounding tissues might impose on its various envelopes. If this exogenous pressure is sustained, the extrinsic intraneural pressure rises.

The tension/compression relationship

Pressure imposed on a nerve increases its internal tension level. Inversely, longitudinal tension placed on an already constrained nerve can augment its internal pressure gradient.

1.2.4 Permanent distal tension

Over many years of practicing nerve palpation, we have observed the phenomenon of permanent distal tension. This occurs as the distal part of a nerve seeks to elongate towards the periphery.

The anatomist Brachet (1921) studied the development of nerves, in particular the embryological evolution of the vagus nerve. He stated the following, which confirms our theory:

The part of the vagus nerve that was originally cervicocephalic extends to the viscera and secondarily moves towards the trunk. Extension into lower regions has become a general tendency of nerves.

In the treatment of nerves the aim is always to stretch them as distally as possible in order to achieve an effect on their intrinsic tension.

1.3 FUNCTIONAL UNITY OF THE NERVOUS SYSTEM

It is important to emphasize that the nervous system is an indivisible entity. No other structure in the body possesses the same degree of connectedness and complexity.

This functional unity rests on the following parameters:

- **Electrical**. Neurons are interconnected. Any impulse generated at one end of the system can, in a millisecond, reach the other end.

- **Mechanical**. There is a continuity of tissue envelopes between the central nervous system and the peripheral nervous system.
- **Biomechanical**. The same neurotransmitters are found in the central and peripheral nervous systems. There is centrifugal and centripetal axon–plasmic flux.
- **Electromagnetic**. The perineural system is a carrier of continuous currents, initiated by brain waves, which diffuse through the neural network to the interior of the body. These currents play a key role in tissue repair following an injury. They are important in maintaining homeostasis and in the organization of the scarring process.

In addition, from the electrical or magnetic point of view, nerves are not completely isolated. There are exchanges of information with neighboring tissues, particularly with the fascial system.

1.4 FUNCTIONAL PATHOLOGY OF NERVES

1.4.1 Neural fibrosis

Intraneural movement occurs between neural tissue and connective tissue components. The nerve fibers are able to crease and then unfold themselves in relation to the layer of endoneurium. At the interior of a nerve or nerve root, a fascicle has the ability to glide in relation to another fascicle. Due to this flexibility, the various components are able to withstand external forces without threat of rupture. In a similar manner, the brain can move in relation to the cranial dura mater that surrounds it, and the spinal cord moves in relation to the spinal dura mater.

When the nerve trunk is traumatized, it almost immediately produces an albumin exudate, which circulates at the site of injury. Endoneurial edema increases intravascular pressure, diminishing circulation in the capillary blood stream. Long-standing edema causes endoneurial fibrosis and the formation of intrafascicular scarring. Therefore, all phenomena of fibrosis or edema affect the intrinsic adaptive mechanism of nerves.

1.4.2 Compression

The physiology of compression is complex. The following succession of events may be found with direct compression of nerve fibers and ischemia, and can create a vicious circle:

- compression
- primary ischemia
- transudation
- increase in intraneural pressure
- limitation of lymph and venous drainage
- nerve edema
- increase in intraneural pressure
- blockage of arterial supply
- secondary ischemia
- nerve lesion.

In osteopathy, it has long been thought that only extraneural forces were responsible for compression phenomena. A great many techniques have been developed with the aim of releasing the external forces exerting themselves on a nerve. However, in reality, compression appears to be more complex. Intrinsic pressures, fibrosis and edema of the nerve itself must be addressed. These compressive elements have a direct effect on the nerve and its perineural connective tissue, as well as on its microvascularization and micro-innervation.

1.5 FROM SPECIFIC TO GLOBAL

We unreservedly and enthusiastically support A.T. Still's concept of globality (1899).

All parts of the body play an indispensable role in ensuring homeostasis. The organism is a marvelous machine that requires all of its components to be in good working order. The individual is a whole that must not be compartmentalized. To do so would risk manual therapists being perceived as specializing in a particular part of the body.

We firmly believe that manipulations should be precise. In order to achieve this precision, specific knowledge is necessary. The acquiring of such education demands that the manual therapist be a generalist. For manual therapists to be good general mechanics of the body, they must have the same manual dexterity when working on a foot, an organ, the spinal column or the cranium.

The manipulation of the cranial nerves is an exceptional tool for treating the brain and the rest of the body. Manipulation of the cranial nerves allows us to have an effect on the sutures, the meninges and the encephalon. All local stimulation has as its goal the generation of a whole-body response on the part of the organism: in this case, one induced by the brain. It is in the spirit of A.T. Still that we have written this book.

Characteristics of cranial nerves

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2.1 REVIEW

The core of the central nervous system is connected with the peripheral organs (sensory or motor) by the cerebrospinal nerves, the entire assembly making up the peripheral nervous system. Upon emerging, the peripheral nerves are termed cranial if they arise from the encephalon or the cerebral trunk. They are termed spinal if they issue from the spinal cord.

Like the nerves of the vertebral column, the cranial nerves are symmetrical, corresponding to the cerebral spinal axis. Numbering 12 pairs, they cross the various meningeal layers to exit foramina at the cranial base, just as the spinal nerves emerge on the lateral side of the vertebral column.

2.2 TERMINOLOGY

The nomenclature and numeration of the cranial nerves date from 1664 (Willis). The 12 pairs of cranial nerves are distinguished according to purely descriptive criteria. They are numbered from I to XII anterior to posterior, and named according to their form (trigeminal), their destination (facial, glossopharyngeal) or function (olfactory, optic, auditory). Since the 18th century, comparative embryology and neurology have brought to light certain inaccuracies in these designations. For example:

- The accessory (spinal or XI) and hypoglossal (XII) nerves, although they emerge from the skull, are of medullary origin and can therefore be considered spinal nerves.
- The vagus nerve (X) and the medullary part of the accessory nerve (spinal or XI) constitute one and the same nerve: the vago-accessory.
- The trigeminal (V) nerve in mammals results from the merging of two nerves, distinct in all other vertebrates: the deep ophthalmic and the maxillo-mandibular nerves.

In this book we will endeavor to use the new terminology. For those like us, who learned the cranial nerves by their old designation, we have included a table of corresponding terms (Table 2.1). Some cranial nerve branches and a few ganglions have also had their designation changed (Table 2.2). The classic mnemonic devices of the past will also have to be updated one day!

2.3 EMBRYOLOGICAL FEATURES

As a rule, the spinal nerves have a metameric and symmetrical arrangement, each somite having a corresponding dorsal and ventral nerve. The ventral nerve is a motor nerve, while the dorsal nerve is sensory. The cranial nerves more or less conserve this arrangement of ventral and dorsal nerves, but their metameres often fade away.

At the level of the trunk, the segmental distribution of the vertebral column nerves is

Table 2.1 Corresponding names of cranial nerves

New nomenclature	Old nomenclature	Anatomical name
Olfactory nerve (I)		N. olfactorii
Optic nerve (II)		N. opticus
Oculomotor nerve (III)	Common ocular motor	N. oculomotorius
Trochlear nerve (IV)	Pathetic nerve	N. trochlearis
Trigeminal nerve (V)		N. trigeminus
Abducent nerve (VI)	External ocular motor nerve	N. abducens
Facial nerve (VII)		N. facialis
Vestibulocochlear nerve (VIII)	Acoustic nerve	N. vestibulocochlearis
Glossopharyngeal nerve (IX)		N. glossopharyngeus
Vagus nerve (X)	Pneumogastric nerve	N. vagus
Accessory nerve (XI)	Spinal nerve	N. accessorius
Hypoglossal nerve (XII)	Greater hypoglossal nerve	N. hypoglossus

Table 2.2 New and old nomenclature

New designation	Old designation	Origin	Destination
Communicating branch of lesser petrosal n.	Superficial lesser petrosal n.	VII	Otic ganglion
Greater petrosal n.	Superficial greater petrosal n.	VII	Pterygopalatine ganglion
Lesser petrosal n.	Lesser deep petrosal n.	Tympanic n. (IX)	Otic ganglion
Deep petrosal n.	Greater deep petrosal n.	Carotid plexus	Pterygopalatine ganglion
Tympanic n.	Nerve of Jacobson	IX	Tympanic cavity

relatively simple to imagine. At first glance, the organization of the cranial nerves appears more complicated. A few rudimentaries of embryology will help us to visualize and understand their arrangement more easily.

2.3.1 The neural tube

Near the end of the first month of gestation the neural tube is formed from 3–5 vesicles (Fig. 2.1), which later on fashion the brain and its annexations (the cerebellum and brain stem):

- the pro-encephalon, which subdivides into the diencephalon (endocrine function) and the telencephalon (thought processes)
- the mesencephalon
- the rhombencephalon (hindbrain), which forms the metencephalon (cerebellum) and the myelencephalon (medulla oblongata).

Each of these vesicles is connected to one or more pairs of what will become the cranial nerves, arranged from right to left.
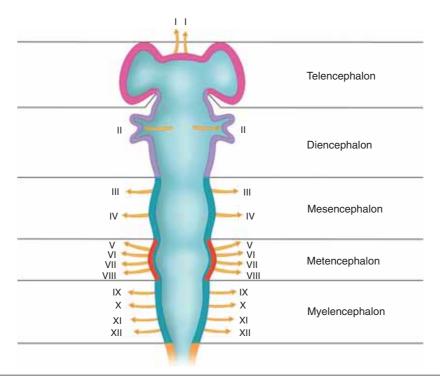
Fig. 2.1 Cerebral vesicles and future cranial nerves.

2.3.2 Sensory organs

The sensory organs form buds at the cephalad extremity of the embryo. The olfactory, optic and otic placodes are three thickenings of ectoderm appearing on each side of the future head (Fig. 2.2). These placodes relate to the olfactory (I), optic (II) and vestibulocochlear (VII) nerves, respectively.

2.3.3 Muscles and other tissues

The future muscles of the cephalic region derive from two mesodermic origins:

- the somitomeres
- the branchial arches.

Somitomeres

At the level of the trunk, the somites are well-differentiated segments of para-axial mesoderm at the origin of the metameres. At the cephalic end, the somites are less well defined and are referred to as diffused somites or somitomeres.

According to their position, two categories can be distinguished:

- The pre-otic somitomeres (in front of the otic placode) form the extrinsic ocular muscles, connected with the oculomotor (III), trochlear (IV) and abducent (VI) nerves.
- The post-otic somitomeres (behind the otic placode) give rise to the muscles of the tongue, governed by the hypoglossal nerve (XII).

The branchial arches

The branchial arches are bulges of mesenchyme which run alongside the primitive intestine of the embryo (Fig. 2.3). Their exterior is lined superficially with ectoderm, while endoderm covers their interior surfaces. The branchial arches make up the major part of the structures of the face and the neck. The nerves of the branchial arches are a mixture of both motor and sensory nerves. They
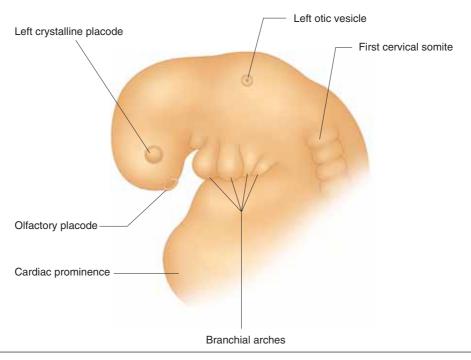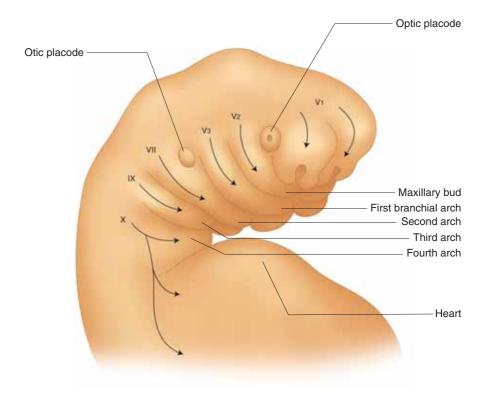
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Fig. 2.2 Placodes.

Fig. 2.3 Branchial arches.

represent the segmental equivalent of the spinal innervation for the rest of the body.

The nerves of a branchial arch contain:

- motor neurons that innervate the striated skeletal muscles; these derive from the arches and are also called branchiomotor neurons
- the sensory neurons for the ectoderm, which line the arches
- the visceral sensory neurons for the endoderm lining the primitive intestine.

The 1st branchial arch is innervated by the trigeminal nerve (V).

The 2nd branchial arch is innervated by the facial nerve (VII).

The 3rd branchial arch is innervated by the glossopharyngeal nerve (IX).

The 4th branchial arch is innervated by the vagus nerve (X).

The 6th branchial arch is innervated by the vagus nerve (X) (Fig. 2.4).

Note: In fish, six branchial arches develop and form the beginnings of the gills and the viscero-cranium. In mammals, there are five numbered branchial arches denoted 1, 2, 3, 4 and 6. The fifth arch does not develop.

2.4 FUNCTIONAL CHARACTERISTICS

Generally speaking, the cranial nerves supply the sensory and motor innervation of the head and the neck, with the exception of the vagus nerve, which also innervates the thorax
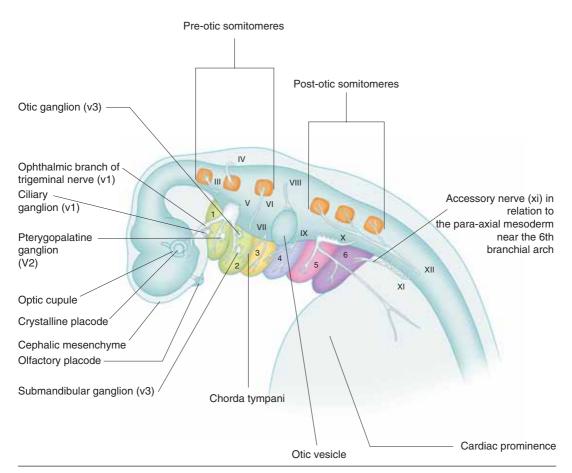

Fig. 2.4 Outline of the cranial nerves.

and the abdomen. In addition, some cranial nerves transmit sensory information coming from the sensory organs. On the motor plane, they convey voluntary and involuntary impulses for muscle control.

Thus the cranial nerves function like modified or somewhat unusual vertebral column nerves. As a group they possess both sensory and motor components. Individually, some are purely sensory or purely motor; others are mixed nerves.

2.4.1 The six impulses

The cranial nerves transmit six different categories of impulse: three sensory and three motor.

Sensory impulses

- General sensitivity: the perception of touch, pain, temperature, pressure, vibration and proprioceptive information.
- Visceral sensitivity: the perception of all sensory data coming from the viscera and the vessels, with the exception of pain of visceral origin.
- Special sensitivity: the sensory perceptions of smell, vision, taste, hearing and equilibrium.

Motor impulses

- Somatomotor impulses: the innervation of the muscles that develop from somites.
- Branchiomotor impulses: the innervation of the muscles that develop from the branchial arches.
- Visceromotor impulses: the innervation of the glands and the smooth muscles of the viscera.

2.4.2 Similarities and differences with the nerves of the vertebral column

Whatever its location, a nerve is a cord of nerve fibers. These fibers are derived from dendrites or from axons. They stem from cellular bodies situated either in the central nervous system or in the peripheral ganglion. The organization of these fibers is, however, very different for the spinal nerves and for the cranial nerves.

The cranial nerves can be divided into two large neurological categories. In one group are those that are analogous to the spinal nerves. These are the motor nerves and the mixed nerves. The other group has special sensory nerves, relating to particular sensory organs of the head (olfactory, optic and vestibulocochlear nerves).

Homologous cranial nerves

The nerves of the vertebral column are mixed nerves resulting from the combination of two medullary roots, one dorsal and one ventral. The homologous cranial nerves correspond to only one of the roots, dorsal or ventral.

Ventral roots

All the motor nerves — oculomotor (III), trochlear (IV), abducent (VI) and hypoglossal (XII) — are homologues of the ventral spinal roots.

With the exception of III, which contains a contingent of visceromotor fibers destined for the smooth muscles of the eye, they are all exclusively somatomotor nerves. They innervate the striated muscles that derive from the cephalic myotomes (motor muscles of the eye, hypobranchial muscles).

Dorsal roots

All the mixed nerves — trigeminal (V), facial (VII), glossopharyngeal (IX) and vagus (X) — are counterparts to the dorsal roots of the vertebral spinal nerves, through their sensory fibers. Like them, they group together sensory fibers issuing from ganglionic neurons located in the cranial ganglions.

These sensory fibers are accompanied by two types of motor fiber:

- Some are visceromotor and innervate the lacrimal and digestive glands, as well as the smooth musculature of the viscera of

the trunk (stomach, intestines, lungs and bronchial tubes). They constitute the essential cranial parasympathetic nervous system.

- Others are somatomotor and innervate particular striated muscles: those of the facial and laryngeal musculature. These muscles are of visceral origin, deriving from the branchiomeric musculature.

Special cranial nerves

Special cranial nerves relate to the cephalic sensory organs: the nose, the eye and the inner ear. The optic "nerve" does not belong to the peripheral nervous system; the retina is a special sensory receptor of cephalic origin. The optic "nerve" represents a single nerve bundle of the neuraxis. The olfactory and vestibulocochlear nerves begin in the sensory placodes, which also give rise to the corresponding sensory organs.

2.4.3 The various roles of the cranial nerves

Sensory role

Meninges
The dura mater, arachnoid, pia mater, falx and tentorium cerebelli receive their sensitivity from the cranial nerves: essentially — as we shall see — from the trigeminal nerve. Others, like the trochlear, make their rather unexpected contributions.

Diploë
The cranial nerves supply the sensitivity of the diploë, which is the spongy part of the cranial bones.

Sutures
Curiously, medical literature is very discreet on the subject of the innervation of sutures. Testut (1896), who is our reference, provides just one piece of information on the topic: the innervation of the lambdoid sutures. He describes this as being due to a nerve filament from the supra-orbital nerve, a branch of the frontal nerve, which itself issues from the trigeminal nerve.

For us, the most important nerve relating to the cranial sutures is certainly the trigeminal because it sends filaments to the coronal suture: the queen of sutures and first in line for absorbing shocks.

Sinuses
The mucosa of the sinuses is very sensitive and reactive. It is the cranial nerves that provide its sensitivity. And here too, without prejudice, we detect the hand of the trigeminal nerve. With its three branches, it innervates the frontal, sphenoidal, ethmoidal and maxillary sinuses.

Skin of the skull and face
The sensitivity of the skin of the face and skull is furnished in large measure, though not exclusively, by the cranial nerves. To give an example, the skin on the skull also receives nerve fibers from the suboccipital nerve of Arnold.[1] It is partly for this reason that we describe its manipulation in this book, even though it does not belong to the cranial nerves.

Arteries
Cranial nerve fibers, combined with those arising from the sympathetic ganglions, deliver arterial sensitivity. Some migraine headaches are attributable to the vasodilatation of the arteries of the skull.

What about the brain?
The brain is not sensitive in and of itself. Intracranial sensitivity is due to the meninges and the vascular system.

Motor role

The muscles of the tongue, the ear and the eye have fibers that come from the cranial

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1. Friedrich Arnold (1803–1890): German anatomist and professor of anatomy in Zurich, Freiberg, Tübingen and Heidelberg.

nerves. So, too, do the muscles of the skull, such as the temporalis, the occipitalis and the frontalis. The oculomotor, trochlear, abducent, facial, accessory and hypoglossal nerves provide the motor function.

Role relating to the special senses

It is via the cranial nerves that the brain receives information which allows us to experience the five senses: to touch, see, hear, smell and taste, and so secondarily to think and to be. The sensory nerves are the olfactory, facial, optic and vestibulocochlear.

Expressive role

It is by virtue of the cranial nerves that the face expresses surprise, joy, sadness, disapproval and fear. It is mainly the facial nerve which provides this function.

Viscerosensory role

The glossopharyngeal, vagus and facial nerves are all viscerosensory.

Glandular role

The salivary and the lacrimal glands are innervated by the cranial nerves.

Anatomical organization of cranial nerves

Like all peripheral nerves, each cranial nerve has a beginning, a trajectory and a termination.

3.1 ORIGIN

We consider the origin of a cranial nerve to be the point at which it emerges from the surface of the encephalon (Fig. 3.1). All the nerves arise from the ventral surface of the encephalon except the trochlear nerve, which arises from the posterior surface.

The most significant concentration of cranial nerves originates in the middle and posterior cerebral fossae.

Here are the localizations, from posterior to anterior:

- medulla:
  — the abducent (VI)
  — the facial (VII)
  — the vestibulocochlear (VIII)
  — the glossopharyngeal (IX)
  — the vagus (X)
  — the accessory (XI) (also at the level of the spinal cord)
  — the hypoglossal (XII)
- protuberance:
  — the trigeminal (V)
- isthmus of the encephalon (superior surface):
  — the trochlear (IV)
- cerebral peduncle:
  — the oculomotor (III)

- chiasm:
  — the optic (II)
- olfactory bulb:
  — the olfactory (1).

> **Note for manual therapists**
>
> Seven cranial nerves are observed to arise from the brain stem. This anatomical arrangement lends great significance to the manipulation of the occipital dura mater and the upper cervicals, and to techniques which open the posterior cranial orifices (foramen magnum, jugular foramen).

3.2 PATHWAY

The pathway of a cranial nerve is intracranial, then cranial, and finally extracranial.

3.2.1 Intracranial pathway

On its intracranial course, the nerve is surrounded by pia mater and is located in the cisterns of the subarachnoid space.

- The optic nerve (II) crosses the chiasmatic cistern.
- The oculomotor nerve (III) is situated in the interpeduncular cistern.
- The trigeminal (V), facial (VII), vestibulocochlear (VIII), glossopharyngeal (IX), vagus (X) and accessory (XI) nerves
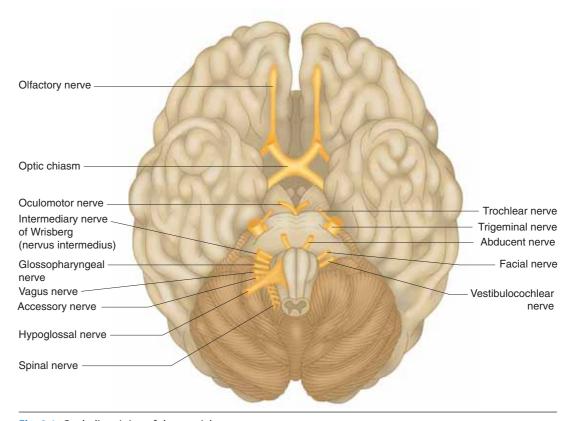
Fig. 3.1 Cephalic origins of the cranial nerves.

are found in the cerebellomedullary cistern.

- The oculomotor (III), trochlear (IV), abducent (VI) and ophthalmic (VI) nerves cross the cavernous sinus.

3.2.2 Pathway through the cranium or orifices

The cranial nerves pass through openings (Fig. 3.2) in the skull to extracranial structures:

- the olfactory foramen for I
- the optic canal for II
- the superior orbital fissure for III, IV, VI and V1 (ophthalmic nerve)
- the foramen rotundum for the maxillary nerve (V2)
- the foramen ovale for the mandibular nerve (V3)

- the internal acoustic meatus for VII and VIII
- the jugular foramen for IX, X and XI
- the hypoglossal canal for XII.

As they exit the cranium, the nerves are enveloped by the pia mater, the arachnoid and the dura mater so that they can pass through the various foramina, fissures and slits of the skull without injury.

Some passages are common to several nerves. This accounts for the possibility of their simultaneous compression by way of tumors or aneurysms, manifesting clinically as the principal basilar syndromes.

3.2.3 Extracranial pathway

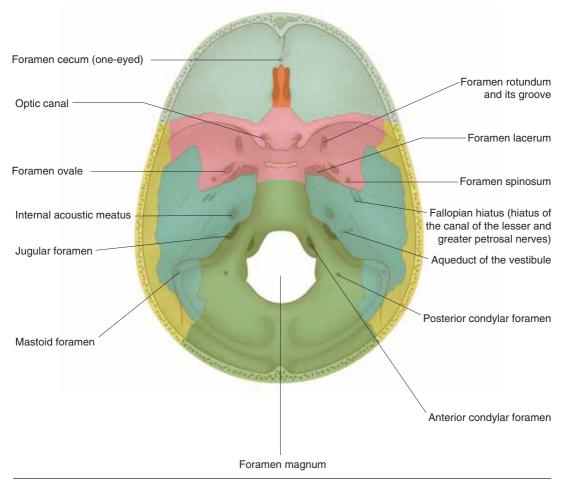The extracranial trajectory may be facial, cervical, or even thoracic and abdominal (Fig. 3.3).

Foramen cecum (one-eyed)

Optic canal

Foramen ovale

Internal acoustic meatus

Jugular foramen

Mastoid foramen

Foramen rotundum and its groove

Foramen lacerum

Foramen spinosum

Fallopian hiatus (hiatus of the canal of the lesser and greater petrosal nerves)

Aqueduct of the vestibule

Posterior condylar foramen

Anterior condylar foramen

Foramen magnum

Fig. 3.2 Endocranial orifices.

3.3 BRANCHES

The terminal and collateral branches are designated to various bony and soft-tissue structures of the head and neck, as well as thoracic and digestive viscera. Peripheral connections are numerous and explain the multiplicity of functions and the many interactions of the cranial nerves.

3.4 GANGLIONS

The sensory ganglions are located on:

- the trigeminal nerve: trigeminal ganglion of Gasser

- the facial nerve: geniculate ganglion
- the acoustic nerve: spiral and vestibular ganglions
- the glossopharyngeal nerve: superior (Ehrenritter[1]) and inferior (Andersch[2]) ganglions
- the vagus nerve: superior (jugular) and inferior (plexiform) ganglions.

Autonomic ganglions, true local plexuses, are appended to certain cranial nerves. For

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1. Johannes Ehrenritter (d. 1790): Austrian anatomist (Vienna).
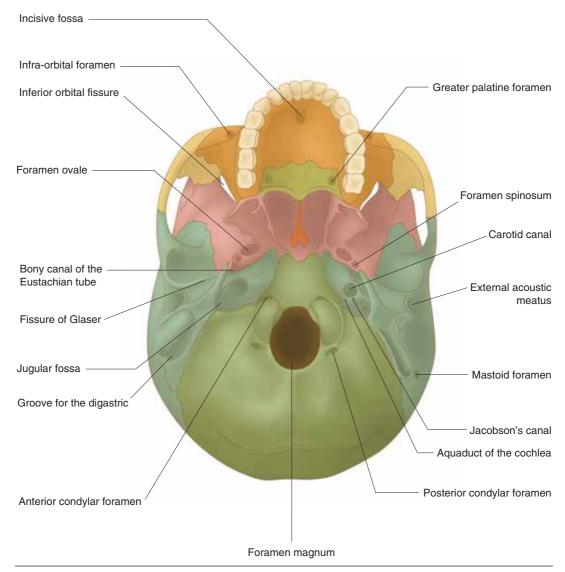2. Carol Samuel Andersch (1732–1777): German physician and anatomist.

Fig. 3.3 Exocranial orifices.

example, the major parasympathetic is supplied by these neurofibers:

- the oculomotor nerve to the ciliary ganglion

- the facial nerve to the pterygopalatine, submandibular and sublingual ganglions
- the glossopharyngeal nerve to the otic ganglion.

Cranial nerves as they emerge from the skull

4

Classically, the skull is anatomically divided into three fossae or cerebral compartments: anterior, medial and posterior.

Moreover, it is composed of two functional parts:

- the neurocranium, for the encephalic brain cavity, comprises the cranial vault and the endocranial surface of the cranial base
- the viscerocranium, for the facial mass, comprises the mandible and all the "visceral" cavities (mouth, nasal fossae, sinuses, pharynx, etc.).

These two parts are of separate embryological origin and are subject to very different mechanical influences:

- Influences of neuromeningeal origin affect the neurocranium.
- Influences of cervico-thoraco-abdominal origin affect the viscerocranium.

We prefer the term "compartment" to that of "fossa" or "*étage*", in order to include the two systems properly. The orifices of the cranial nerves are thus concentrated in three compartments.

4.1 ANTERIOR COMPARTMENT

4.1.1 Cribriform plate of the ethmoid bone

It is through the cribriform plate that the olfactory nerve fibers reach the nasal fossa.

We shall see that it is only indirectly, by way of the nerve endings, that we have an effect on the olfactory nerve; it is too deep to be otherwise fully accessible.

4.1.2 Palatine vault

The palatine vault is perforated by several foramina:

- the anterior palatine foramen: the nasopalatine nerve
- the greater palatine foramen: the anterior palatine nerve
- the accessory palatine foramina: the middle and posterior palatine nerves.

4.1.3 Apertures of the face

The facial foramina are extremely useful for creating an effect on the deepest cranial nerves (Fig. 4.1). Remember that the smallest terminal nerve branches are the most reflexogenic.

Orbital region

The orbital region is rich in orifices that allow access to the cranial nerves of the face. The orbital fissure, the optic canal and the spheno-maxillary fissure bring the skull into communication with the orbital region of the face.

Certain nerves are directly accessible as they exit their orifice, others only indirectly by way of their emerging fibers.
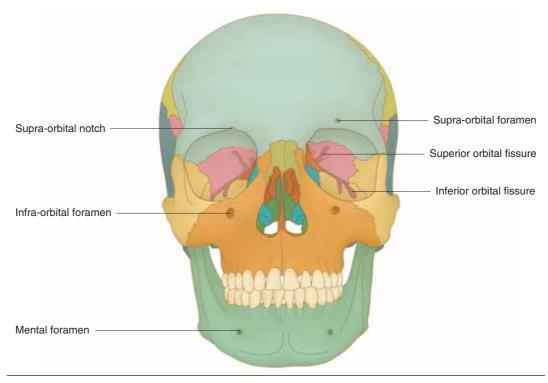
Fig. 4.1 Orifices of the face.

Direct access
- The supra-orbital foramen for the supra-orbital nerve (ophthalmic nerve, VI).
- The infra-orbital foramen for the infra-orbital nerve and its bouquet of nerve fibers (maxillary nerve, V2).
- The zygomatico-orbital foramen for the temporomandibular nerve (maxillary nerve, V2).

Indirect access
- The optic nerve, reached by mobilizing the eyeball.
- The supratrochlear nerve, whose emerging filaments are accessible via the inner corner of the eye, above the trochlea (ophthalmic nerve).
- The infratrochlear nerve, often anastomosed with the supratrochlear nerve and from which it is separated only by the trochlea.

- The lacrimal nerve at the level of the outer corner of the eye (ophthalmic nerve, VI).

Chin region

The mental foramen is the point of exit for the mental nerve, a branch of the mandibular nerve (V3).

4.2 MEDIAL COMPARTMENT

The sphenoid is an extremely important bone in the framework of orifices: key in and of itself, and because it serves as an intermediary for the other bones of the skull.

The superior orbital fissure (sphenoid fissure) brings the cranial cavity into communication with the orbital cavity.

A point to note

The three principal nerve orifices of the face are:

- the supra-orbital notch (sometimes a foramen)
- the infra-orbital foramen
- the mental foramen.

All three are aligned along a vertical line passing 2–3 cm from the median line of the face (Fig. 4.2).
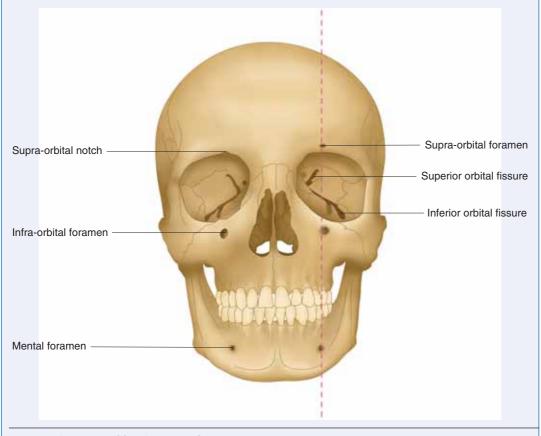
Supra-orbital notch

Infra-orbital foramen

Mental foramen

Supra-orbital foramen

Superior orbital fissure

Inferior orbital fissure

Fig. 4.2 Alignment of facial nerve orifices.

The majority of the cranial nerves and their branches leave the skull by way of the superior orbital fissure (Fig. 4.3):

- the lacrimal nerve (branch of VI)
- the frontal nerve (branch of VI)
- the trochlear nerve (IV)
- the abducent nerve (VI)
- the oculomotor nerve (III)
- the nasociliary nerve (branch of VI).

The optic canal allows the optic nerve to exit. The foramen rotundum affords passage to the maxillary nerve.

The foramen ovale gives passage to:

- the mandibular nerve
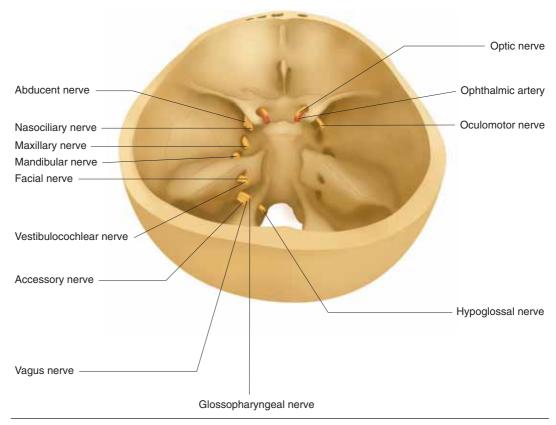- the venous plexus.

The foramen spinosum gives passage to:

21

Fig. 4.3 Cranial nerves and the orifices of the medial compartment.

- the meningeal branch of the mandibular nerve
- the middle meningeal artery.

In the carotid canal, the internal carotid artery, especially the sympathetic carotid plexus, is joined by numerous cranial nerve fibers.

The greater petrosal nerve exits the Fallopian hiatus (hiatus of the canal of the greater petrosal nerve).

The accessory hiatus (hiatus of the canal of the lesser petrosal nerve) gives passage to:

- the lesser petrosal nerve
- the superior tympanic artery.

4.3 POSTERIOR COMPARTMENT

The orifices of the posterior compartment are shown in Figure 4.4.

The foramen magnum is of extreme strategic importance in assuring communication between the medulla oblongata and the encephalon. To underline its tremendous importance we list here other structures which pass through it:

- the two accessory nerves
- the two vertebral arteries
- the two vertebral veins
- the anterior spinal artery
- the two posterior spinal arteries
- the spinal vein.

The hypoglossal nerve emerges from the hypoglossal canal.

After the sphenoid fissure it is the jugular foramen (posterior foramen lacerum) that contains the greatest number of nerve roots:
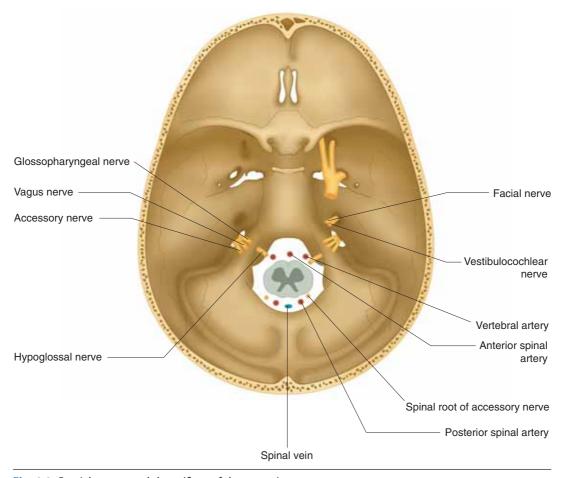
Fig. 4.4 Cranial nerves and the orifices of the posterior compartment.

- the glossopharyngeal nerve
- the vagus nerve
- the accessory nerve (lateral branch).

    Also passing through it are:

- the internal jugular vein
- the posterior meningeal artery.

    The internal acoustic meatus contains:

- the vestibulocochlear nerve
- the facial nerve
- the internal acoustic artery and the internal acoustic vein, which are frequently the cause of tinnitus.

The stylomastoid foramen contains:

- the facial nerve
- the stylomastoid artery.

    The petrotympanic fissure (fissure of Glaser) contains the chorda tympani.

    Note: There appears to be a glaring inequality in the distribution of nerves passing through these apertures. However, one must keep in mind their mechanical, fluid and neural interdependence. A meningeal tension on one small orifice can affect a large fissure through the play of reciprocal tensions.

23

Innervation of cranial dura mater

<div style="text-align: right; font-size: 3em;">5</div>

Many of the results we achieve are probably attributable to the reflexogenic effects of neural manipulations on the dura mater. The cranial dura mater possesses a rich and vast sensory innervation.

5.1 PARIETAL DURA MATER

5.1.1 Dura mater above the tentorium

The three branches of the trigeminal nerve innervate the parietal dura mater of the anterior and medial cerebral compartments (Fig. 5.1):

- the ophthalmic nerve (VI)
- the maxillary nerve (V2)
- the mandibular nerve (V3).

The dura mater of the anterior cerebral compartment is innervated:

- in its medial portion, by the anterior meningeal branches of the ethmoidal nerves, coming from the ophthalmic nerve
- in the area of the lesser wing of the sphenoid, by the meningeal nerve branch of the maxillary nerve
- in its most lateral part, by the mandibular nerve.

The dura mater of the medial cranial compartment is innervated:

- in its anterior portion, corresponding to the region of the superior orbital fissure,

by the middle meningeal portion of the maxillary nerve
- in its posterior and lateral portion, by the nervus spinosum of the mandibular nerve.

5.1.2 Dura mater beneath the tentorium

The innervation of the osseous wall of the posterior cerebral fossa relies on:

- the vagus nerve (X)
- the hypoglossal nerve (XII)
- the sensory branches issuing from the dorsal roots of the first three spinal nerves (C1–C3).

The schematic is further complicated by important anastomoses between these different systems.

5.2 MEMBRANES OF THE DURA MATER

5.2.1 Falx cerebri

In the anterior part of the falx cerebri, innervation is supplied by the anterior meningeal branches of the ethmoidal nerves, which derive from the nasociliary nerve (ophthalmic nerve, VI). Posteriorly, we find the ascending expansions of the tentorial branches, arising from the ophthalmic nerve (providing sensory input), which supply it.
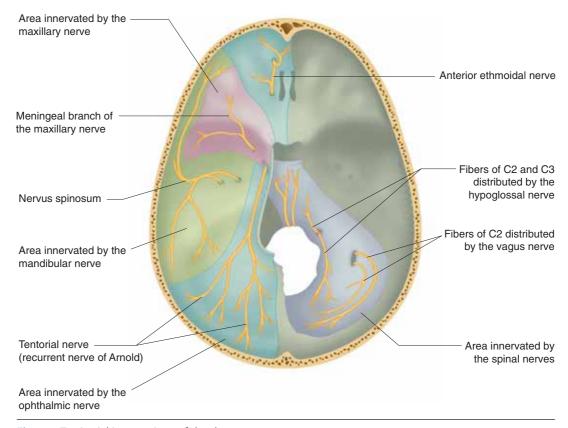
Fig. 5.1 Territorial innervations of the dura mater.

5.2.2 Tentorium cerebelli

The tentorium cerebelli is innervated by the tentorial branches of the recurrent meningeal division of the ophthalmic nerve, or the recurrent nerve of Arnold (Fig. 5.2). The sensory endings are more numerous on each side of the longitudinal superior sinus, and at the level of the tentorium cerebelli, than they are at the level of the cranial base.

5.3 DURAL TUBE SENSITIVITY

Nociceptive fibers are especially numerous:

- in the areas neighboring the venous sinuses and the meningeal arteries

- in the area where the dura mater is perforated by arterial or venous vessels.

The dura mater is very sensitive to pain. Consequently, tugs and tensions at the following places can lead to pain:

- the arteries at the cranial base
- the veins near the vertex or at the cranial base
- the perforation points of the dura mater.

There are multiple causes of cranial pain, which include stretching or distension of the scalp and/or the meningeal vessels. A number of cases of headaches are of dural origin, like those that occur following the drawing of
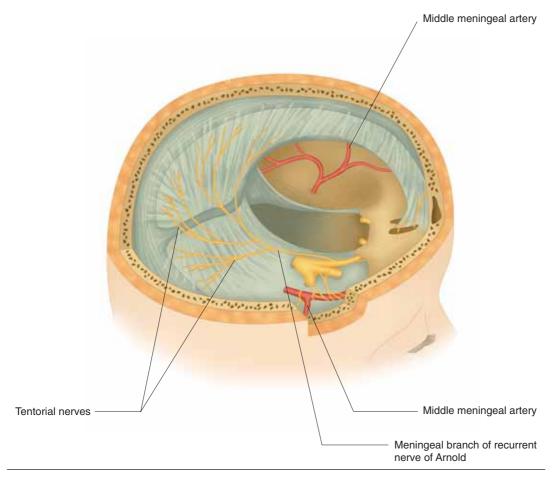
Middle meningeal artery

Tentorial nerves

Middle meningeal artery

Meningeal branch of recurrent nerve of Arnold

Fig. 5.2 Innervations of the reciprocal tension membranes.

cerebrospinal fluid by lumbar puncture. We believe this type of headache results from the stimulation of the sensory terminations of the dura mater and from disturbance to the arterio-venous circulation. When the cerebro-spinal fluid is withdrawn, the effective weight of the brain increases; the encephalon folds in on itself slightly and exerts pressure on the dura mater, causing traction on the arachnoid trabeculae.

Functional anatomy of cranial nerves

The cranial nerves are not, a priori, anatomically different from the peripheral nerves; however, they have distinctive characteristics which must be taken into account if we are to manipulate them in the best possible way.

6.1 NERVE ROOTS

It is not possible to make direct contact with the cranial nerves. It is, however, possible to stretch them through the action of compression/decompression of the skull, combined with rotations of the head.

The optic nerve allows us to have the most direct effect on its roots through the anterior–posterior mobility of the eyeball.

6.2 NERVES

The nerves arise as pairs in the skull, inside of which they are very sensitive to all mechanical forces, particularly variations in pressure. On exiting the cranium, the nerves are surrounded by the pia mater, arachnoid and dura mater, and can thus pass through the various foramina, fissures and slits without risk of injury.

Some nerves are amyelinic, especially at their terminal distribution. It might be thought that this absence of myelin would give these nerves more space and provide improved mobility. This is indeed the case with the optic nerve, for example, which goes from a diameter of 5 mm to 1.5 mm upon reaching the sclera of the eye. We must bear in mind the fact that the optic nerve is made up of more than 800 000 nerve fibers: an extremely high concentration!

6.3 CRANIAL APERTURES

6.3.1 Function of the orifices

At the cranial orifices the nerve roots are well protected by the meninges. This arrangement limits nerve damage in cases of cranial trauma. From the "functional" point of view, these orifices must be as open as possible, to permit the micro-gliding of the structures they contain. The slightest tension on the cranial meninges can affect the structures passing through the orifices.

In their passages, the cranial nerves are almost always accompanied by an artery and a vein. The latter serves as additional mechanical protection.

Because of cranial movement, it seems that these orifices have the capacity to expand very slightly in the expansion phase. The articular motion or play of the cranial orifices must be perfectly free and unimpeded. We will look at the various factors affecting the passage of the cranial nerves.

Gliding within the orifices

When the head changes position and intracranial pressure changes, the cranial orifices allow for microscopic gliding movements of

the structures passing though them. This is made possible through the interaction of the meninges and related orifices.

Remember that tension on an individual orifice has repercussions.

A balance of pressure gradients

The large orifices, like the orbital cavities, the acoustic meatus and the foramen magnum, are able to compensate for large pressure variations. We are all well aware of intracranial pressure changes when coughing or sneezing, especially when we have a headache.

The smaller orifices, like the orbital fissures, the anterior foramina lacerum and the jugular foramina, have the role of harmonizing intracranial pressure when there are small pressure variations.

6.3.2 Conditions for optimal functioning

An optimal opening

The orifice opening may be affected by cranial trauma or fetal malposition, or be the result of slow or difficult labor and delivery. This underlines the importance of early cranial treatment for newborns in these cases.

A restricted orifice can be a predisposing factor in the development of chronic sinusitis or facial paralysis *a frigore.*

An orifice rhythm

We are convinced, but have not yet proven, that the cranial orifices dilate with the cranial expansion phase. To the sceptics we would say that nothing is ever totally frozen or fixed, as they will hopefully have observed. Even the mineral world is subject to the phenomenon of rhythmic expansion. The flow of water that gives us stalactites follows the lunar cycle. The diameter of rock pores varies according to the phases of the moon and influences the quantity of the water running across them.

6.4 EMERGENT CRANIAL NERVES

It is possible to reach the cranial nerves where they emerge on the face and sometimes where they present on the skull or at the neck. The vagus nerve is the exception, surfacing as it does in the thoracic and abdominal regions.

At the point where they emerge, the cranial nerves are surrounded, like the peripheral nerves, by a small fascial "noose". This fascial ring, reinforced by perineural connective tissue, must be released in order to optimize the effects of neural manipulations and remove all external constriction on the nerve.

It is advantageous to mobilize the cutaneous branches due to their effects on the deeper nerve fibers. For this purpose, massage techniques such as skin rolling are useful, particularly in treating the trigeminal and facial nerves.

Functional pathology of cranial nerves

7

In our previous book, *Manual Therapy for the Peripheral Nerves*, we detailed various factors that can negatively influence nerves. This chapter revisits the major causes of peripheral nerve dysfunction. Here we will describe the variables that come into play due to the proximity of the brain and as a result of pressures affecting the endocranial pathway.

7.1 MECHANICAL FUNCTION

From a mechanical point of view, the unit formed by the spinal cord and the spinal nerves is the counterpart of the unit formed by the brain and the cranial nerves.

7.1.1 Tensions

We saw in *Manual Therapy for the Peripheral Nerves* that tensions between the meninges and the medulla rest in part on the tensional equilibrium of the plexuses and on the free movement of the nerves, of which they are an extension.

The same applies at the skull; the balance of tensions between the brain and the meninges depends, to a degree, on the balanced mechanical tension of the 12 pairs of cranial nerves. Intracranial membrane tensions play a considerable role in the modification of intracranial pressure.

7.1.2 Pressures

The basic mechanical model is complicated by the phenomenon of intracranial pressure (ICP). For optimal functioning, the brain requires as constant a pressure as possible. In the skull, cerebrospinal fluid and the intravascular pressures are inversely proportionate.

The arachnoid attachments are the anchors that connect the cranial nerves to the brain.

Nerve tensions affect intracranial pressure, and vice versa.

The brain literally floats in cerebrospinal fluid, thereby lightening its weight considerably. According to the Archimedes principle, a brain weighing 1300 g in the air has an effective weight of just 40 g when suspended in cerebrospinal fluid! Under these physiological conditions, the encephalon exerts only weak traction vis-à-vis its arachnoid attachments.

If the cerebrospinal fluid pressure diminishes, tension on the meningeal connections increases. Remember that this is one of the reasons for the post-lumbar puncture syndrome, where the ensuing headache is due to the increase in the effective weight of the brain.

Inversely, should the cerebrospinal fluid pressure rise, the tension on the arachnoid trabeculations falls. Thus all variations in pressure are linked to variations in the tension of structures moored to the brain.

7.2 VASCULAR FUNCTION

A fundamental cause of pathogenic neuralgias or cranial nerve paralysis is a compromised vascular system (Doyon et al 2002). This may explain some "essential" neuralgias or paralysis *a frigore.*

The vascularization of the cranial nerves derives from very fine branches stemming from the neighboring vessels. They are very sensitive to variations in ICP (see above) and to sympathetic stimulation coming from the pericarotid plexus, as well as to mechanical restrictions at the cranial orifices.

Pathologically and clinically, we can observe ischemic attacks of the cranial nerves in cases of diabetic arterial hypertension, arterial inflammation (viral or other), post-traumatic arterial dissections, arterio-venous fistula, or even after the selective embolization of some arterial branches.

Moreover, some vascular blockages or aneurysms are implicated in cranial nerve compression at the cisterns of the cranial base. Here, classically, we find compression of CN VII (facial) causing hemispasms of the face and compression of CN V (trigeminal) underlying advanced cases of essential neuralgia.

From a functional perspective, a great many disturbances seem to run along the nerve under the effect of local arterio-venous factors. It could be a matter of a defect in the nerve's intrinsic blood supply, or the result of mechanical conflict between the vessel wall and neighboring perineural structures.

These same circumstances affect the peripheral nerves, but their influence is much more pronounced in the cranial nerve domain.

The constancy of the ICP can be a determining factor in pathology. Along their intracranial pathway, the nerves are extremely vulnerable to any and all changes in intrinsic or extrinsic pressure.

Through these neurovascular relationships, important effects can be produced on the microcirculation of the cranial nerves.

7.3 AUTONOMIC NERVOUS SYSTEM FUNCTION

Some cranial and cervical organs are governed by the two great components of the autonomic nervous system: the sympathetic and the parasympathetic. The cranial nerves are connected to the sympathetic cervicals and are borrowed by the parasympathetic afferent and efferent nerve fibers. Accordingly, neural dysfunction can show up as diminished mucosa, as glandular secretions (lacrimal, salivary) or even as vasomotor problems.

7.4 INFORMATION AND PROPRIOCEPTIVE FUNCTION

7.4.1 Central miscommunication

As we explained in *Manual Therapy for the Peripheral Nerves*, the perineural system is abundantly innervated. It contains multimodal receptors and can generate proprioceptive or nociceptive impulses, depending on the situation.

Over time, the malfunction of a nerve does not always mean pain, but often signals miscommunication in the proprioceptors, which activate compensatory mechanisms in the central nervous system. Simply by freeing the nerve of any constraint, we allow it to break the disrupted neurological arc.

7.4.2 Hilton's Law

The nerves supplying a joint also supply the muscles that move the joint and their articular insertions, as well as the skin covering the joint.

Hilton (1860–62, 1981)

The corollary is true. The nerves that innervate the skin over a joint generally provide that same joint's sensitivity.

At the level of the cranium, the concept of joints and muscles is thought of in terms of sutures and membranes.

In this perspective, the manipulation of certain branches of the trigeminal nerve enables an effective release of the osteo-sutural and meningeal mechanics.

7.5 ELECTROMAGNETIC FUNCTION

The perineural system is the center of continuous currents and the place of origin of brain waves.

7.5.1 Perineural currents

In contrast to neurons, which achieve their connections point by point, the perineural

system communicates by criss-crossing the body.

This type of communication relies on the perturbations of a continuous current and is of increasing interest to scientists. It appears that all tissues of the body are bathed by a permanent and continuous electrical influence, generated by the perineural current.

The currents behave like liquid crystals: that is to say, like structures of variable conductivity. According to the orientation of the currents, cells of the same tissue are able to change their polarization.

The perineural current is sensitive to all electromagnetic, telluric, cosmic, barometric and atmospheric changes.

Via the manipulation of certain nerves, we can alleviate pain that started a long time ago. These long-standing discomforts may be worsened before a "change in the weather", as often stated by patients.

7.5.2 Brain waves

Brain waves are propagated along the perineural system and blend with this continuous current. The result is a powerful vector of homeostasis. This balancing force is a key element in tissue repair, and probably in all internal recovery processes. A patient's positive attitude is indispensable to healing. The brain waves he or she generates extend through the perineural system as far as the injured part of the body, wherever this might be. This is one of the physiological justifications for the *placebo effect*.

The manipulations that we direct at freeing the perineural system improve a variety of conditions. Patients often report immediate or delayed reactions that are not explainable by neurophysiology alone.

Nerve manipulation plays a very significant part in all aspects of recovery, rehabilitation, and rebalancing in the entire distribution area of the liberated nerve.

7.5.3 Intention

The work of Sidorov (2002) has shed new light on the electrical elements of the perineural system. Not only do brain waves follow the perineural system to end up in all parts of the body, but they can also *transmit themselves to adjacent organisms*.

This discovery may explain some therapeutic effects, particularly those of touch. To touch is not enough. Therapists must adopt a particular mental attitude. Their gestures must be imprinted with a positive "intention" of well-being.

Our perineural system could be this special link between our brain and our hand.

Is this the vector we call *therapeutic intention*?

7.6 CHEMICAL AND HORMONAL FUNCTION

Metabolically, the brain secretes neurotransmitters and hormones. Any disturbance of the ICP will change brain activity, with possible consequences for major functions of the organism. For example, we can witness changes in the levels of serotonin and gonadotrophin following trauma.

7.7 ETIOLOGY OF NEURAL FIXATIONS

While reflecting on many cases of essential neuralgias and paralysis — that is, those without explanation — we have sought to understand the causes of cranial nerve dysfunctions.

7.7.1 Constraints of the orifices

"Natural" asymmetry

The old anatomists measured the cranial orifices, particularly the jugular foramen, to determine whether there was a natural asymmetry between the two sides. According to their studies, the left jugular foramen is naturally smaller than the right. This holds true for other orifices, which are more complicated to measure because of their irregularity and sinuous configuration.

In referencing different anatomical works, there is a tendency to consider such asymmetries as "natural". However, these analyses are based on observing dry bone. In fact, dry

bone bears the imprint of all of the mechanical constraints it was subjected to in life. Also, the asymmetry in the diameter of the foramina could be understood as the reflection of restricted movement in the area.

Certain asymmetries are "natural". For example, the right hemiface is never comparable to the left hemiface. The position the fetus adopts *in utero*, as well as the way in which the baby presents at delivery (breech birth, etc.), may result in a dominant side, which can explain asymmetrical cranial lesions. These differences are common, and are sometimes genetic and sometimes acquired.

Acquired asymmetry

Fetal malposition
Fetal malposition commonly creates cranial restriction, which can have repercussions for the orifices.

Constraints/restrictions in utero
Constraints *in utero* can be the result of uterine spasms, sometimes due to the mother's position. The baby may be positioned too low, or may enter the birth canal too early, as a result of the mother sitting or lying down for an extended period of time.

Working mothers-to-be often try to work as late as possible into their third trimester so they can take more time off after delivery. The infant is literally compressed against the pelvis, with the head rotated to one side, leaving one side of the head more compressed than the other. Certain orifices would, as a result, be smaller or simply fixated, no longer subject to the pressure–decompression phases of the cranial movements.

Caesareans
Caesarean section spares the baby's cranium from the impact of labor and delivery.

Although it is invaluable at times, caesarean section has the tremendous disadvantage of depriving the cranium of the chance to be "molded" by the natural compressive forces of the birth canal. The cranium is therefore left in the same state as it was in the uterus;

this is not always desirable, especially in the presence of cranial deformations.

Forceps and suction
We know that forceps and suction are inevitable when the life of the infant or the mother is at stake. Nevertheless, this procedure can create numerous and significant osteocartilaginous restrictions.

7.7.2 Cranial and facial trauma

Of course, direct trauma to the cranium has an effect; however, some falls that have no direct impact on the skull can nevertheless affect it through the action of collision forces.

Medical imaging is becoming more impressive and high-resolution, but it does not always reveal functional problems that the hand is able to detect.

7.7.3 After-effects of infection

Meningitis affects the substance and the extensibility of the dura mater, the pia mater and the arachnoid. These changes can also be observed following a high fever or, more rarely, after encephalitis.

When the dura mater is fixed, it puts mechanical tension on the nerve and impedes normal fluid circulation.

7.7.4 After-effects of craniofacial surgery

All surgery creates imbalance at the tissue level and for the ICP. Although sometimes remaining asymptomatic for many years, these discrepancies can be the origin of neural tensions generating pain and dysfunction.

7.7.5 Dental apparatus

All dental devices create restriction in the cranium and the face.

Of course, in orthodontics this is the desired effect. Appliance tolerance varies from one individual to another. It also depends on age and on type of apparatus, as well as on the progression and duration of treatment.

There can also be mechanical distortion linked to wear and tear with dentures and other dental prosthetics. The apparatus might originally have been well fitted but has become pathogenic after a few years.

7.7.6 Inflammatory reactions

Allergies

Due to their congestive effect, especially on the facial sinuses, allergies can irritate the nerves in their orifice passage and where they come into contact with mucous membranes.

Tumors

Tumors augment intracranial pressure. They are sometimes unfortunately situated and inoperable. We have achieved some good results, notably with meningiomas that had been causing headaches because of mechanical constraint on the nerve roots.

Migraines and head pain

The cranial nerves can be the origin or the sustainers of some migraines and headaches. They are, in any case, the vehicle.

Cranial nerves can be irritated by migraines and headaches stemming from the visceral, immune and circulatory systems. They can also be the cause of headache, specifically after craniofacial trauma.

Headaches of mechanical origin typically start at the posterior cervical spine or the back of the skull and radiate forward. Headaches of visceral origin begin, for the most part, in the frontal region of the cranium and then radiate towards the back.

Evaluation and treatment of cranial nerves

<div style="text-align:right">8</div>

8.1 TERMINOLOGY

As with all professions, it is important in osteopathy to understand the technical terms involved. Some basic definitions, upon which this book is based, are given below.

8.1.1 Tissue evaluation

Tissue evaluation is done via the hand. Placed on a part of the body, the hand is sensitive to the various tissue tensions transmitted by the organism.

Tissue fixation

For a tissue, fixation is the loss of a part or all of its qualities.

As a consequence it loses its normal mobility and motility, with possible repercussions for other structures and other systems of the body.

Listening

Listening is a test not a treatment, performed with the hand placed upon the body. The palm of the hand is drawn towards the area of fixation and stops at that level.

It is a topographical assessment; we can distinguish between general listening, which gives a global indication, and local listening, which is topographically more precise.

Neural fixation

A nerve is made up of various connective tissues: endoneurium, epineurium, perineu-rium and mesoneurium. These connective tissues can lose their qualities of elasticity, visco-elasticity and extensibility, and become fibrosed to the point of abnormally stimulating the nervi nervorum.

The tensions exerted on the nervi nervorum will have consequences for the arterial, venous and lymphatic systems of the nerve.

Nerve bud

Mechanical tensions can arrange themselves within various connective tissue structures, to the point of creating a sort of protuberance.

The fixated nerve experiences a rise in intraneural pressure and creates a small, hard, sensitive protrusion (*bud*).

Intraneural pressure concentrates on the weak point of the nerve, creating these small hernias or bulges. Nerve buds can be made to disappear very quickly by regulating the intraneural pressure.

With the cranial nerves, it is only at their points of emergence at the skin that we find nerve buds.

Distal neural tension

Distal neural tension is the intrinsic tension of a nerve, which enables it to lengthen constantly to the periphery.

The vascular and neural forces of turgor create a bridge with the surrounding connective tissues.

To treat a nerve, we work to restore the forces of expansion by stretching the nerve and connective tissue fibers in a distal

direction. This expansion could be a relic of the incredible forces that develop during embryogenesis. After the third month of life *in utero*, *as many as 5000 neurons are produced every second*, and their axons grow in the direction of the periphery.

Neural visco-elasticity

When tissue is compressed, its elasticity restores it quickly to its initial shape or position. Visco-elasticity allows the tissue to regain, little by little, its original shape or position. It is thus a deferred and mediated return.

A nerve requires good visco-elasticity in order for its intraneural components not to be compressed.

Cranial movement

The brilliant Dr Sutherland

We would like to express our profound admiration for William Garner Sutherland, who discovered cranial movement. No one before him had felt it.

Just as Hahnemann (1806) pushed the boundaries of professional conscience in homeopathy, Sutherland (1939) went so far as to mechanically bind down certain parts of his own skull. We firmly believe in the motions of flexion and extension that he described.

Phases of cranial expansion and retraction

From the courses we teach around the world, we are aware that disparities exist on the subject of cranial mobility, especially when it comes to the neuromeninges. In our teachings and in this text, we use the terms "expansion" for the cranial dilatation phase — which corresponds to flexion — and "retraction" to designate the return phase — corresponding to extension.

8.1.2 Neural treatment

Direct treatment

The treatment consists of entraining the nerve in the direction of the correction which is away from the fixation. The hand directly engages the fixation.

This technique is used particularly for perineural adhesions at the exit sites of cranial nerves.

Indirect treatment

The hand will entrain the nerve in the direction of the fixation, which is in the direction of ease.

Induction

The hand follows the nerve in the direction indicated by the "listening". It is not the therapist who chooses the direction; it is the tissues.

The hand exerts an actual pressure. This neural technique is the most effective and the least forceful.

Note: Induction can take the nerve in the same direction as the indirect technique, but does not necessarily do so.

Visco-elastic treatment

Visco-elastic treatment "plays" on the caliber of the nerve by alternately applying compression and decompression. The finger exerts gentle pressure on the neural bud, or on the indurated area of the nerve. Releasing its pressure slowly, the finger gently prevents the nerve from instantly regaining its initial dimension.

This technique is very appropriate for nerve tissue; the ideal would be to combine the visco-elastic technique with induction.

Combined treatment

Combined treatment combines neural manipulation with manipulation of another part of the body, such as a joint, an organ, a ligament or another nerve.

8.2 EVALUATION

8.2.1 Global cranial listening

Global cranial listening is an indispensable evaluation tool for quickly locating exo- and endocranial fixations (Figs 8.1 and 8.2). It is

Fig. 8.1 Global cranial listening.

Fig. 8.2 Global cranial listening.

very precise, and has great subtlety and unequalled speed.

First, cranial listening allows the therapist to localize a part of the cranium or its contents for treatment.

Feel first, think later. Let the tissues speak to your fingers.

Secondly, it is possible to determine which structure is creating the "listening". The problem is knowing if the fixation is:

- osseous
- sutural
- membranous
- nervous
- encephalic (brain).

Position of the patient

The subject is in the supine position, legs extended and hands on the abdomen in order to release the fascial tension of the upper extremities. The therapist is seated behind.

For those who are right-handed, place the palm of the left hand under the occiput, the palm of the right hand at the level of the parietals, and the middle finger along the sagittal suture.

The occipital hand can vary the position of the head in order to locate the fixation precisely and to establish its depth.

Listening in three dimensions

To feel in three dimensions, imagine the skull is a balloon with something inside. Never forget that the most important aspect of the skull is not the bone but the brain that it protects. In skull fractures, it is not the osseous lesion that matters, but the area and extent of the brain damage.

Listening with the palm of the hand

The palm of the hand allows us to feel the fixations with precision. It is the palm that indicates the fixation. If your fingers are attracted to the left and your palm to the right, the fixation is located on the right.

We feel the palm glide in the direction of the fixation, and then sink to the level of the fixation.

Evaluation

Osseous fixation

The palm glides and stops rather suddenly without going deep. It is as if it were glued to the spot (Fig. 8.3). It is possible to confuse this type of listening with listening to the cranial integuments, but simple palpation allows us to tell the difference.

Compress the area of bone that is restricted, in order to assess its degree of compressibility. An osseous fixation feels harder than the other bony areas nearby.

Sutural fixation

The palm glides and stops at a specific place, giving the impression of wanting to sink down a little lower (Fig. 8.4). Experience has shown us that the coronal suture bears witness to all sutural fixations. For example, a fixation on the right side of the coronal suture can be the consequence of and witness to a fixation of the right occipital–mastoid suture.

Membranous fixation
Parietal dura mater

The palm glides and travels more or less horizontally without stopping clearly and precisely, as it does with an osseous fixation (Fig. 8.5).

Falx cerebri and tentorium cerebelli

The palm glides and is pulled rather deep, pauses momentarily and then continues vertically, without a distinct stop. For the falx, the movement is very vertical, whereas for the tentorium, the movement is more oblique.

Brain fixations
Structural lesions

The palm glides and then gives the impression of wanting to go deeper, but in a precise manner — the inverse of membranous listening (Fig. 8.6).
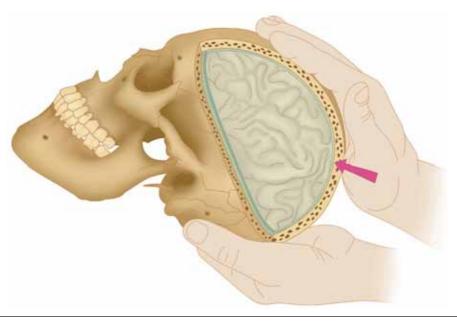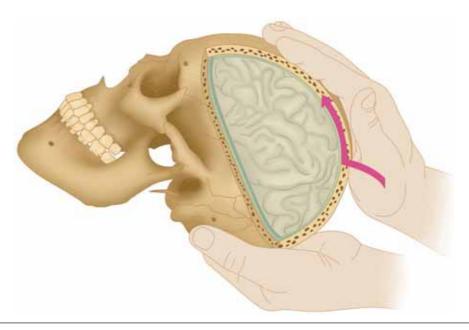
Fig. 8.3 Type of listening for osseous fixation.

Fig. 8.4 Type of listening for sutural fixation.
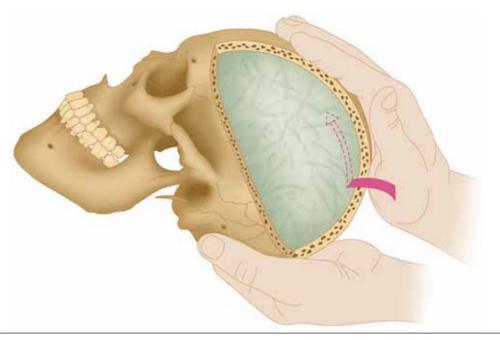
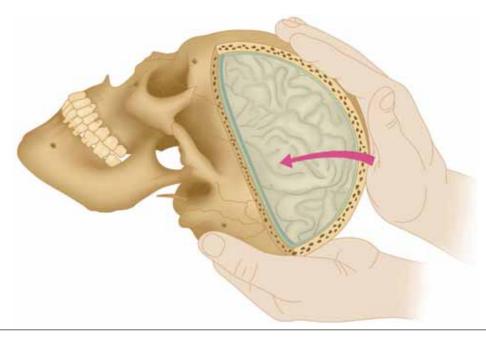**Fig. 8.5** Type of listening in the case of fixation in the cranial membranes.

Fig. 8.6 Type of listening in the case of fixation in the brain of structural origin.

Often, the palm does not remain flat; it tilts on the hypothenar eminence. In analyzing this, one has the impression that the palm follows a small and very narrow shaft.

We have (sadly) been the first to discover 20 or so brain tumors, which had not been revealed by medical imaging. This underlines the fact that the hand is able to detect areas of high density, though without giving information as to the severity of the lesion.

Above all, never imagine that the hand can tell whether a tumor is benign or malignant.

Dysfunctional zones

The palm glides and sinks deep, but in a less precise manner than for structural problems (Fig. 8.7). It gives the impression of floating *entre deux eaux*.

We can easily feel dysfunctional areas at the occiput and temporal lobes when there is a problem with hearing or vision.

Neural fixations

Neural fixations are subtle to feel. Happily, they can be revealed through cranial listening, as well as symptomatology (Fig. 8.8).

Initially, the palm glides and sinks somewhat deeply; secondarily, it is drawn very precisely and comes to a rather abrupt stop.

8.2.2 Exocranial tests (testing the external surface of the cranium)

Via the patient interview, the symptoms and cranial listening, it is possible to verify whether there is a problem with a cranial nerve by assessing the sensitivity of its emerging filaments.

Orifice tests

By testing the cranial apertures, it is possible to assess the sensitivity of the cranial nerve and also to feel it gliding within its canals, and sometimes its serrated conduits. It is always a good idea to compare a cranial nerve with its contralateral counterpart.

Compress the nerve at the exit of the cranial orifice and allow it to return spontaneously in order to assess two qualities:
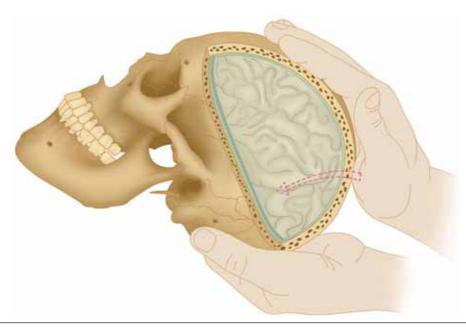

Fig. 8.7 Type of listening in the case of fixation in the brain of functional origin.
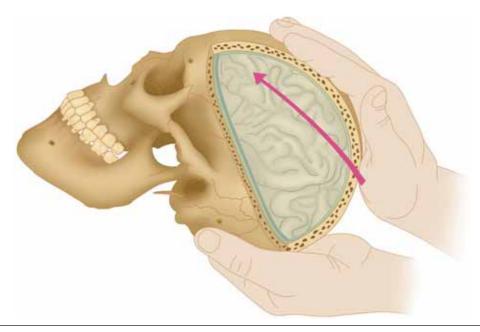
Fig. 8.8 Type of listening in the case of neural fixation.

- **Firmness**. Compress the nerve towards the interior of the orifice. When you compress a nerve and there is a hardness, the fixation is internal.
- **Immediacy of return**. Compress the nerve in its orifice and evaluate its return. It should immediately regain its original position. This test lets us know how freely the nerve is able to move within its cranial aperture and within the distal part of its canal. Of course, we are also evaluating the nerve's visco-elasticity.

Intrategumentary and intramuscular tests

These tests consist of making the terminal nerve branches glide at the level of the scalp, the face and the neck, in order to assess:

- their freedom of movement; these branches should be extensible and elastic
- their degree of firmness
- their sensitivity.

To carry out this test, one finger creates a fixed point on the nerve and the other stretches it. For the cheek, one finger is placed in the mouth, and the other externally on the face. The two fingers meet on the nerve to evaluate its distensibility and sensitivity.

Some of the fibers of the facial nerve can be tested with the muscles of the skull. Differentiate between the nerve fibers and the muscle fibers by their consistency. Nerve fibers are thin, resilient, not very compressible and quite sensitive.

Neural digital listening

In principle, a nerve that is free and without fixation does not attract your finger when listening. If your finger is attracted in any direction, there is a good chance the nerve is fixated. The restriction could be intraneural or in the environment of the nerve.

If your finger is attracted deeply at the orifice, the fixation is either in the conduit itself or within the skull.

We always compare this local test with the results of global cranial listening.

Testing of emerging branches

It is possible to determine which cranial nerve has a problem by analyzing the sensitivity of

its neurofibers as they emerge at the level of the face, the neck and mouth.

The easiest to analyze (which suits us well!) is the trigeminal nerve, which presents three terminal branches in the face. These emerge at the supra-orbital notch and the infra-orbital and mental foramina.

8.3 TREATMENT

8.3.1 Principles of cranial nerve manipulation

In our book *Manual Therapy for the Peripheral Nerves* (Barral and Croibier 2004) we provide a detailed description of nerve components: their Schwann cells, myelin sheath, nervi nervorum and vasa nervorum.

The nerve is a special tissue requiring much care and delicacy when treated manually. Manipulation that is too forceful or too insistent can provoke significant and long-lasting pain.

The main points to respect are described below.

Gentleness

It is important not to press forcefully on a nerve. We must know how to "speak" kindly to it. All mobilization is done with respect for its extraordinary sensitivity.

Minimal compression

All initial contact with a nerve requires "delicate digital anchoring" to establish a purchase. The degree of compression must be as light as possible, but effective in establishing proper contact with the nerve. The degree of compression must be sufficient to stimulate the nervi nervorum without activating the nociceptive impulses.

Induction

All treatment of the cranial nerves (exceptions are rare) is done by induction: that is to say, you magnify the listening.

Induction is not an intention but an actual movement that can be carried out via traction, rotation and sometimes compression.

Because of its terrific reactivity the slightest stimulus to a nerve will immediately send billions of pieces of information shooting towards the central nervous system — and all at the rate of a high-speed train. The brain responds locally or generally to this stimulation.

Visco-elasticity

When we manipulate a nerve, we release the compression gradually while maintaining a light hold, until the nerve regains its normal volume.

Visco-elasticity technique produces an effect on all intraneural elements: arteries, veins, axons and connective tissues.

Distal stretching

A cranial nerve is treated in the same way as a peripheral nerve, by always stretching it distally.

In the case of the peripheral nerves, induction naturally pulls the fingers in the distal direction.

For those cranial nerves that are very short as they exit the skull, things are more difficult. The distal stretching technique may be used for nerves such as the cervicofacial and temporofacial branches of the facial nerve, whose course covers several centimeters.

Preliminary release of the dura mater

All dura mater tensions can have an effect on the perimeter of the orifices. Even excellent cranial nerve manipulation will have little effect if the dura mater that surrounds it is tight.

The best technique we know consists of releasing the dura mater tensions during the cranial expansion phase.

Preliminary distal stimulation

Peripheral nerve manipulations begin by first addressing the mechanoreceptors. These are constituted in the nervi nervorum and the small sensitive filaments of the fascial rings that surround the cranial nerves at the point where they emerge on the face or in the

cervical region. Intracranial nerves are treated with induction. To refine and focus our action on the cranial nerves, it is useful to begin their treatment by stimulating distal branches as they emerge from the skull.

To feel the difference this makes, listen to the cranium before and after preliminary distal stimulation.

Homolateral viscera

Usually, organs react homolaterally to cranial nerve fixations. Of course, there are exceptions, notably in the case of the gall bladder. For example, at the beginning of a biliary attack, it is the left frontal nerve that is the most sensitive.

Techniques for the cranial orifices

These techniques are suited to nerves whose length on issuing from an orifice is minimal. Suction and visco-elasticity are used to achieve an effect.

Depression–aspiration techniques

The finger is placed on the nerve's orifice as if wanting to penetrate several times, and is then released. The nerve fibers, because of their elasticity, tend to emerge a little further, benefiting from the rebound effect.

This greatly resembles the visco-elasticity technique, the difference being that here we also have an effect on the meningeal envelopes that surround the nerve in its passage.

This is the principle used to unblock a sink with the flat of a hand. Pressure is created and then released, creating a lower pressure which aspirates the contents of the siphon towards the surface.

Intraneural release technique

This technique is a variation of the preceding maneuver; its action goes much deeper. Imagine an elastic hose obstructed in its center by an inaccessible plug. In order to unblock it, you can stretch and release the tube several times, or compress and release it at the level of the obstacle. The play of apply-ing and letting off pressure can free the obstacle through the effect of aspiration.

We can use the same system for a nerve; little by little, the hardened intraneural zone area is released.

Musculo-tegumentary techniques

The musculo-tegumentary techniques consist of mobilizing the cranial nerve ending in relation to the integuments and muscles that protect it and within which it lies.

This type of manipulation is useful, for example, for the infra-orbital nerve (branch of the trigeminal nerve), which surfaces just below the cheekbone. One finger is intra-oral and the other is positioned externally on the skin, facing towards the finger in the mouth. The nerve is glided and gently played in the thickness of the muscle and skin tissues between your fingers.

At the scalp, we can either glide or play the nerves of the cranial vault in the thickness of the integument.

Cutaneous techniques

The skin near a fixated nerve is often hypersensitive, and may even be painful. This is due to the cutaneous sensory filaments.

Using a massage technique of skin rolling, the pain can be relieved and a reflexogenic effect can be produced at the same time.

Unpleating vascular ripples

The majority of the cranial nerves are accompanied by arteries and veins as they cross their cranial orifice.

Whenever we manipulate the nerves we achieve a concomitant vascular effect.

Some nerves are accompanied by arterioles over a fairly long trajectory. They do not follow a straight course, but rather wrap and surround the nerve in a vascular web. The manipulation of the nerve then has the effect of smoothing out the vessels.

The best example of this arrangement is the ophthalmic artery, which surrounds the optic nerve. Its various branches deliver

vessels to the ethmoid, ciliary body and muscles of the eye.

Neuro-encephalic techniques

These techniques consist of stimulating a cranial nerve at its point of emergence in order to feel, via cranial listening, to which central zone it is connected:

- Either we feel its encephalic location: its anatomical junction with the central nervous system.
- Or we perceive its functional connections: areas of cortical projection where sequelae linked to physical or psychological trauma are stored.

Cerebral visco-elasticity techniques

These techniques consist of increasing and decreasing the intracranial pressure (ICP) to play on cerebral visco-elasticity. The areas with the least visco-elasticity correspond to zones of dysfunction.

These are the same techniques we apply to the solid visceral organs.

Teaching the patient to self-treat

In chronic cases, results can take a long time to achieve and are not long-lasting. It is a good idea to teach patients how to treat themselves.

The cranial nerve fibers, as they emerge on the face, are easy enough to find and treat. Some patients manage to stop or diminish the intensity of their migraines or their sinusitis, thanks to these points.

8.3.2 Effects of cranial nerve manipulations

Local effects

The effects on the nerve itself are as follows:

- diminished intraneural pressure
- reduced nociceptive response of the nervi nervorum
- effects on the small vessels inside the nerves (vasa nervorum)
- restoration of the distal expansion force.

Regional effects

Regional effects relate to:

- the meninges
- the sutures
- the intraneural pressure; we are not able to provide proof but, subjectively, we feel a general intracranial release, as if the skull "softens"
- the brain: areas of high density and areas of venous congestion
- the cranial nerves: their roots and intracranial course.

Effects on the meninges
All abnormal tensions of the dura mater, the falx cerebri and the tentorium cerebelli can have an effect on the ICP and the cerebral venous circulation. The cranial nerves supply the sensory innervations to these structures.

Thus, manipulation of the supratrochlear nerve at the supra-orbital level can have an effect on the membranes of the cranium.

Effects on the sutures
The manipulation of the supra-orbital nerve has effects on the lambdoidal suture. This manipulation doubles its effects via its influence on the frontal sinus. The results of our manipulations are not limited to the intended area.

Vascular effects
The manipulation of the cranial nerves has an effect on the intrinsic arterial microsystem (vasa nervorum) and on the external arterioles that accompany them. We exert a direct mechanical action by unfolding and unpleating the arteries, and/or an indirect vasomotor action via the autonomic nervous system.

Let us not forget that the veins, in addition to transporting blood, also play a role in providing dynamic and static protection for neighboring tissues.

Central effects

At the sensory level
It is with vision and hearing capacity in particular that patients quickly feel a difference.

They sometimes report a change after just one session.

Sensory organs are interdependent. When, for example, people suffer from anosmia (impairment of the sense of smell), the other senses are influenced; they often lose their sense of taste as well.

Some experiments, observed via positron emission tomography (PET) scans on the blind, show that, little by little, the occipital visual center activates itself to perform other functions: the sense of touch or smell, for example (Impact Médécin 2004). This perhaps partially explains the manual abilities of the blind and the sharpness of their hearing.

We sometimes hear people with poor eyesight say things like, "Hand me my glasses so I can hear you better"!

On the emotional level

Patients react in different ways. They may have dreams of certain events or simply recall them.

On a proprioceptive level

The experiments we have carried out with SPECT (single proton emission computer tomography) have shown that our techniques activate the cerebellum, then the thymus, and finally the various brain centers of the limbic system (Amen et al, 2004).

Reflexogenic effect

The cranial nerves have an undeniable effect on the visceral system. The influence occurs via their connections with the vagus nerve and the sympathetic nervous system.

We have already stated that it is mainly by way of their emerging branches that we obtain an effect on the cranial nerves. As is the case with the peripheral nerves, it is often the distal part of the cranial nerves that is the most reflexogenic. We have no physiological explanation for this phenomenon, only our clinical findings.

In our profession, we know that it is not the visible part of the iceberg that is the most important; the same holds true for the cranial

nerves, whose emerging filaments are the witnesses to the 90% that is inaccessible.

We are especially fond of this Chinese proverb: "The tree which falls makes more noise than the whole forest growing." It is necessary to know how to listen to life and hear the forest quivering.

8.3.3 Indications and contraindications

Indications

Often indications are closely linked to the etiology of cranial fixations. We will see this in detail as we look at each nerve.

- Dura mater tensions.
- Sutural and diploic fixations.
- Venous congestion, characteristic of headaches where the heads feel heavy and which dissipate progressively during the course of the day.
- Migraines and headaches with a vasculo-neuro-trigeminal component.
- Vertigo and instability.
- Sinusitis.
- Dental prostheses and orthodontics.
- Following:
  — trauma
  — surgery
  — otitis
  — facial paralysis
  — hemiplegia
- Snoring.
- When the sensory system has been affected.
- When the central nervous has been affected.

Contraindications

As with indications, contraindications will be covered in more depth in the remaining chapters.

Always remember that an atypical pain, for which there is no logical explanation, or one that is very intense should make us cautious. What we know is infinitesimal in relation to what we have yet to learn. We can only deal

with what we know. If you as a therapist have the smallest doubt, it is best to refer the patient to a specialist.

Keep in mind these major contraindications:

- intracranial hypertension
- severe arterial hypertension: decompensated or malignant

- after-effects of intracranial hemorrhage
- severe diabetes
- intracranial aneurysm.

This is not an exhaustive list. We leave it to our readers to judge what may appear to them to be a contraindication.

Manipulation of the plurineural orifices

9

Some cranial orifices provide a passage for several cranial nerves. To avoid the repetition of techniques common to a number of nerves, we will look at three highly strategic areas:

- superior orbital fissure
- jugular foramen
- foramen magnum.

9.1 PURPOSE OF TECHNIQUES

Although we might certainly influence the diameter of a cranial aperture by a few microns, our goal here is to have an effect on the meningeal coverings of the cranial nerves. Due to our action on the membrane lining the orifices, we influence communication between the endocranial and exocranial vascular systems.

The primary respiratory mechanism is felt at these orifices. This is why the manipulations are performed during the expansion phase of cranial rhythm.

To work specifically on a given nerve, one can:

- slightly modify the direction of the technique
- utilize the principle of preliminary distal stimulation.

9.2 SUPERIOR ORBITAL FISSURE

9.2.1 Anatomy

The superior orbital fissure can truly be described as a crossroads of the utmost importance. It is contained between the medial border of the greater wing of the sphenoid and the caudal surface of the lesser wing. It is much larger at its medial part. It provides a passage for numerous structures, listed here from lateral to medial:

- superior ophthalmic vein
- lacrimal nerve
- frontal nerve
- trochlear nerve
- abducent nerve
- oculomotor nerve
- nasociliary nerve.

We should point out the following:

- The foramen rotundum, through which the maxillary nerve passes, is separated from the orbital fissure by a space of just 1–3 mm.
- The foramen ovale, through which the mandibular nerve transits, is only 1 cm from the foramen rotundum.
- The foramen spinosum is situated a mere 3–4 mm from the foramen ovale.

External acoustic meatus

Palatine process of the maxilla
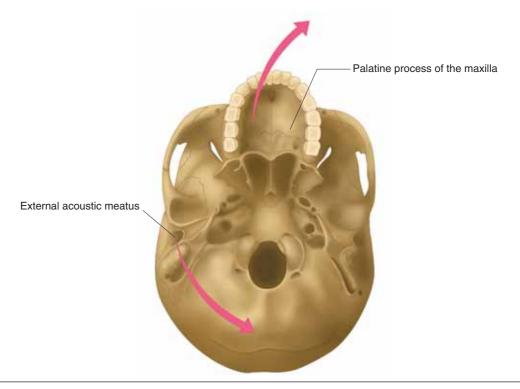
Fig. 9.1 Manipulation of the superior orbital fissure.

The effects of the superior orbital fissure technique have repercussions on all these apertures.

9.2.2 Technique

The patient is supine, with the head rotated to the side opposite the fissure being manipulated (Figs 9.1 and 9.2).

Step 1

Place the index finger in the patient's mouth on the homolateral palatine process of the maxilla. Position it close to the palatine suture. Depending upon the shape of the maxilla, it is sometimes necessary to place the finger a little further back, on the horizontal lamina of the palatine.

Step 2

Place the thumb or index finger of the other hand in the external acoustic meatus on the

Fig. 9.2 Manipulation of the superior orbital fissure.

posterior part of the tympanic plate and the anterior part of the mastoid. The other fingers are placed on the squamous part of the temporal bone and on the sphenoid.

Step 3

The intra-oral finger draws the maxilla anteriorly and medially while at the same time the finger in the acoustic meatus draws the skull in a posterior medial direction.

Detail

For the nerves situated laterally in the orbital fissure, try to create a convexity of the skull. To affect structures located medially, create a concavity. It is best to stretch the skull in both directions.

Note: It is possible to increase the mechanical tension exerted on the superior orbital fissure by slightly increasing the rotation of the head in the final part of the maneuver during the end of the expansion phase of cranial rhythm.

9.3 JUGULAR FORAMEN

9.3.1 Anatomy

The jugular foramen is a large opening between the jugular process of the occiput and the petrosal portion of the temporal bone, set at the posterior end of the petro-occipital suture. In it are joined the sigmoid and the inferior petrosal sinuses. It is called the "posterior foramen lacerum" because of its very irregular contour.

The jugular foramen is divided in two parts:

- an anterior part, for the glossopharyngeal nerve
- a posterior part, for the vagus and accessory nerves, as well as the internal jugular vein, which is the continuation of the sigmoid sinus.

Generally, the right jugular foramen is a little larger than the left. Is this why more problems occur with the left vagus nerve?

9.3.2 Technique

The patient is supine, the head turned slightly to the side of the jugular foramen that is to be treated (Figs 9.3 and 9.4).

Step 1

Place a finger in the anterior part of the external acoustic meatus. For example, for the right jugular foramen, slide the left hand under the skull, placing the left index finger in the external acoustic meatus and drawing it anteriorly and medially.

Step 2

Position the index and middle fingers of the right hand on the jugular process of the occiput and draw it posteriorly and medially. Finally, place the side of the skull you are treating in convexity. To augment the stretch effect, the patient's head can be turned to the same side.

9.4 FORAMEN MAGNUM

9.4.1 Anatomy

The foramen magnum has an oval shape with a large anterior–posterior diameter of about 3.5 cm and a width of 3 cm. It provides a wide communication between the cranial cavity and the vertebral canal.

It contains:

- lower end of the medulla oblongata
- two accessory nerves (spinal roots)
- two hypoglossal nerves
- two vertebral arteries and veins
- anterior spinal artery
- two posterior spinal arteries
- spinal vein.

Note that, in adapting to humans' upright posture, the foramen magnum moved itself forward. This evolutionary process occurred over millions of years.

The technique for the foramen magnum has more of an effect on the vascular contents than on the nerves. This maneuver is indicated for all vertebrobasilar circulatory
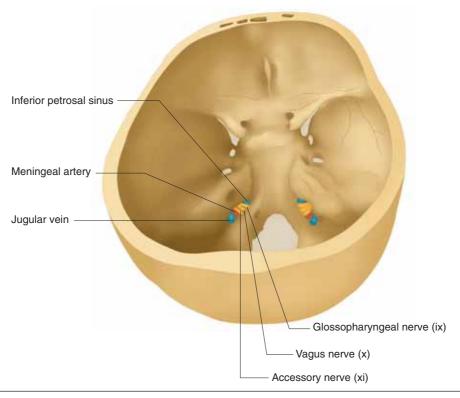
Inferior petrosal sinus

Meningeal artery

Jugular vein

Glossopharyngeal nerve (ix)

Vagus nerve (x)

Accessory nerve (xi)

Fig. 9.3 Manipulation of the jugular foramen.

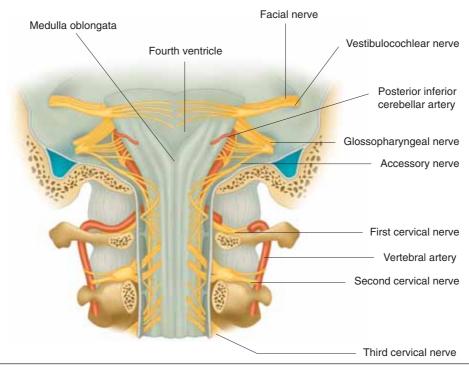**Fig. 9.4** Manipulation of the jugular foramen.

Fig. 9.5 Manipulation of the foramen magnum.

insufficiencies. We also use it for circulatory problems of the inner ear.

9.4.2 Technique

The patient is seated in front of you, facing away (Figs 9.5 and 9.6). Place your two hands on the lateral skull in such a way as to position your thenar eminences under the mastoid processes and the jugular processes of the occiput. To avoid any uncomfortable pressure, ensure that the entire weight of the skull rests securely on your palms.

Your elbows are supported on the lateral aspect of the patient's shoulders. During the expansion phase, press the skull cephalad simultaneously with both hands; repeat this about ten times. To increase the pressure without using more force, ask the patient to lean back against you.
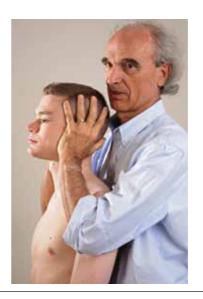
Fig. 9.6 Manipulation of the foramen magnum.

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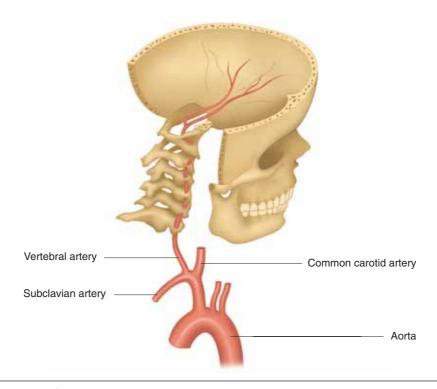
Fig. 9.7 Trajectory of the vertebral arteries.

The effects of this technique are not limited to the foramen magnum. It puts the two vertebral arteries under tension, along with their vertebral trajectory, and does the same for the spinal dura mater.

Vertebral artery technique

This technique follows the one above but is more specific to the curves of the vertebral artery (Figs 9.7 and 9.8). It also has effects on the entire contents of the posterior cranial compartment, notably cranial nerves VII–XII.

Employ the same approach as for the foramen magnum, varying the lateral inclinations and rotation of the cervical column. At the end of axial traction, tilt the cervical column contralaterally and bring it into homolateral rotation.

This technique is most effective when carried out during the cranial expansion phase.

Fig. 9.8 Manipulation of the vertebral artery.

Always remember that there is a privileged relationship between the dura mater and the vertebral artery. We must also keep the peri-arterial sympathetic plexus in mind, as it accompanies the vertebral artery from the stellate ganglion as far as the ends of its terminal branches.

The goal of this technique is to allow the artery some play along its intra-transversary course and as its bends to negotiate a path between the first two cervical vertebrae and the occiput. As a rule, this technique is performed 4–5 times.

Olfactory nerve

10

The olfactory nerve (I) is a sensory nerve that conveys the olfactory impulse, serving the sense of smell.

It comprises four parts:

- the olfactory striae or olfactory roots
- the olfactory tract
- the olfactory bulb
- the olfactory nerves proper.

The first three parts are the central olfactory formations outside of the brain. They attach to the base of the forebrain. The olfactory nerves proper run from the olfactory bulb to the mucosa of the superior nasal fossae.

10.1 ANATOMY

10.1.1 Olfactory nerve

The olfactory nerve is shown in Figure 10.1.

Origin

The olfactory nerve originates on the caudal surface of the olfactory bulb and crosses the cribriform plate of the ethmoid bone from one part of the crista galli process to the other, in order to reach the olfactory region of the nasal cavity.

Caudally, the olfactory nerve roots are located superior to the origin of the optic nerve and the anterior cerebral artery.

Pathway

The axons are collected into small bundles that make their way through the subarach-noid space. Sheathed in pia mater, they cross the foramina of the cribriform plate.

The most common cause of injury to the nerves at this level is a fracture of the anterior cranial fossa or the development of an olfactory meningioma.

Terminal branches

The olfactory nerves terminate in the nasal mucosa, which occupies a small surface on the roof of the nasal cavity.

- The lateral olfactory nerves (12–20) are found in the superior nasal concha.
- The medial olfactory nerves (12–16) descend along the nasal septum.

One of these medial nerves, the terminal (vomeronasal) nerve, descends obliquely in front of the incisor canal to the vomeronasal organ, a little-known structure to which we will return later.

Note: The olfactory nerves are non-myelinated and consist of bundles of slender fibers held together by thin strips of connective tissue.

10.1.2 Olfactory epithelium

The olfactory epithelium is located high in the nasal cavity (Fig. 10.2). It has a yellowish coloring, due to the presence of pigment.

The epithelium contains three types of cell:

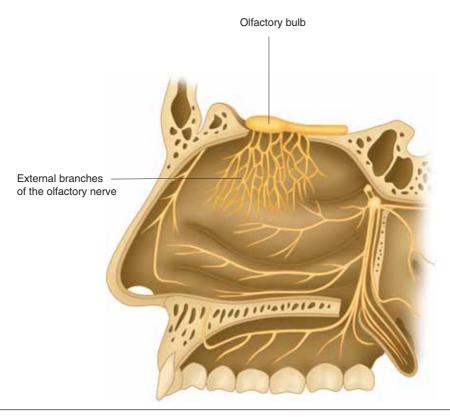- olfactory receptor cells, bipolar neurons that have a single fine dendrite terminated by a bulge at the surface of

Fig. 10.1 Olfactory nerve.

the epithelium, and a tiny non-myelinated axon, which runs with other axons to form the olfactory nerve
- supporting cells, comparable to glial cells
- basal cells, which contribute to the origin of new receptor cells.

The olfactory receptor cells, like the taste receptors, have a continuous turn over, dying and regenerating on a cycle of 4–8 weeks. In fact, the olfactory receptor cells are the only neurons in the nervous system that are regularly replaced in the course of a lifetime!

Characteristics of olfactory cells

The nerve cells of the olfactory system continually regenerate, whether at the level of the olfactory epithelium or that of the two olfactory bulbs.

In mice, 80 000 of these neurons are produced each day: that is, 1% of the neurons of the olfactory system. In 3 months, the cells of the olfactory epithelium are entirely renewed.

The glial cells surround the axons of the olfactory nerves. They provide them with nutritive and anti-adhesive elements that allow the axons to grow. This growth takes place in an orderly and precise fashion to restore olfactory function.

A Spanish research group documented in the journal *Neuron* that they were able to re-establish the sensory and motor functions in rats whose spinal cord had been severed. The protocol consisted of transplanting olfactory glial cells at the level of the medullary lesion. Researchers at University College London, are attempting these transplant grafts in humans (Li et al 2004).

The surface of the olfactory epithelium is an indicator of a species' olfactory acuity. Its surface in humans is about 10 cm^2. By comparison, in dogs it is about 170 cm^2, with 100
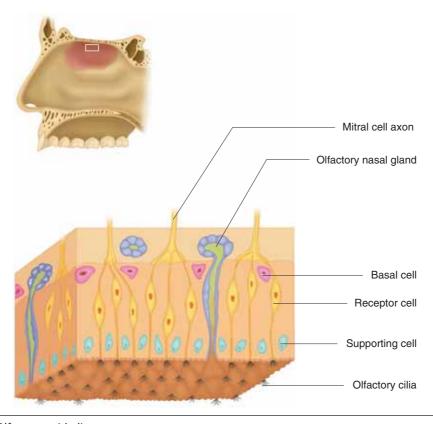
Fig. 10.2 Olfactory epithelium.

times more receptors per square centimeter than in humans.

10.1.3 Central olfactory formations

Olfactory bulb

The olfactory bulb is an oval-shaped bulge 8 mm in length and 4 mm wide. It contains the mitral cell bodies with which the neurofibers synapse in the olfactory tract, and whose axons proceed into the olfactory tract. The two olfactory bulbs nearly touch each other.

Olfactory tract

The olfactory tract (formerly known as the olfactory band) is a narrow band measuring 35 mm in length, on the transversal plane; it is triangular in shape, and located in the olfactory sulcus at the caudal surface of the frontal lobe. It is made up of a neuroglial

axon surrounded by neurofibers of the mitral cells.

Its posterior section becomes wider and flattens, forming the olfactory trigone, which divides into medial, lateral and intermediate olfactory striae.

Rhinencephalon

The encephalic center corresponds to the olfactory part of the brain or the rhinencephalon (Fig. 10.3).

The so-called hippocampic fibers join the lateral olfactory area located at the anterior extremity of the hippocampal convolutions of the hippocampus (5th temporal convolution).

The rhinencephalon is part of the archaic brain that has passed down instinctive and emotional behavior, as well as the sense of smell. In humans, the rhinencephalon is not very well developed, unlike in some animals
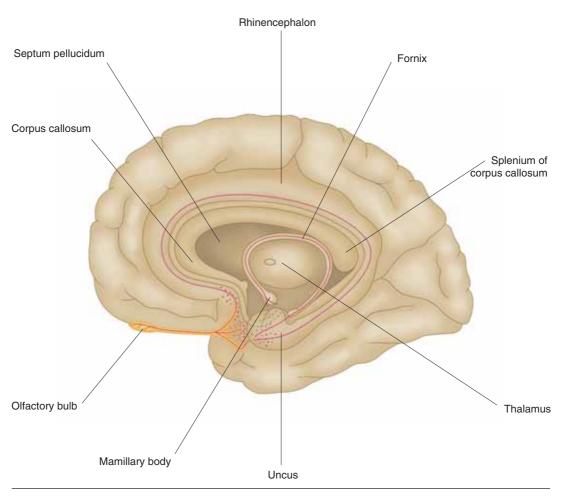
Fig. 10.3 Rhinencephalon.

whose sense of smell is very well known. For example, a polar bear can detect a smell from 6 km away. A retriever dog recognizes smells several days after the stimulant has gone. The male butterfly picks up the scent of a female butterfly from a distance of more than 10 km.

In the animal kingdom, the sense of smell has many functions. Different smells — food, fellow creatures or enemies — produce very different behaviors. For example, the presence of enemies or predators elicits a fight or flight response. The sense of smell is implicated in the subject of life and death of the individual or of its species.

Animals rely considerably on their sense of smell. While this is less true for humans, the olfactory afferents nevertheless influence behavior around sexuality and food. These sensory afferents lie at the origin of the mechanisms that regulate eating and determine satiety.

Brain connections

From the olfactory area, fibers lead towards the visceral centers such as the salivary nuclei, the dorsal nucleus of the vagus, the hypothalamus and the epithalamus.

Olfactory stimulation generates visceral responses, such as salivation in response to pleasant smells, nausea in response to disagreeable odors, or even the acceleration of peristalsis and increases in gastric secretions.

10.2 PHYSIOLOGY AND PATHOLOGY

10.2.1 Olfactory functions

Sense of smell

The sense of smell is the result of physico-chemical stimulation of the olfactory cells.

Little by little, we have lost a large part of our olfactory acuity. Modern life, pollution and the city environment seem to be the causes.

Some animals have a sense of smell that is almost beyond belief; salmon, eels and sharks travel hundreds of kilometers following their sense of smell. It is said that it is the nose rather than the taste buds that allows the gastronome to appreciate the good things in life. The sense of smell is more subtle than the sense of taste.

The olfactory nerves and the olfactory pathways transmit the olfactory message conveyed by the sense of smell. The behavioral and emotional reactions originally associated with the rhinencephalon are relayed by the limbic system.

Behaviors

The secondary olfactory neurons run directly from the olfactory bulb to the olfactory cortex without any relays. The olfactory impulses thus have a direct connection with the limbic system.

On account of these close links, the olfactory tract figures very largely in the autonomic nervous system, emotion, behaviors and their motor implications.

The limbic system plays an important role in the formation and anchoring of memories. It is this very close association with the limbic system which explains why certain smells are so evocative of memories and emotions.

Olfactory disorders

The olfactory nerve is rarely the seat of pathological processes, but it is frequently implicated by pathologies and traumas of adjacent structures. Olfactory disturbances take many forms.

Dysosmias

Anosmia is the loss or impairment of the sense of smell. This dysfunction is sometimes discrete and temporary, and it rarely recurs.

The two nostrils must be tested individually, occluding each in turn. Use non-irritant and familiar substances such as clove oil, turpentine, coffee, vanilla or ether. Avoid the use of substances that are irritating (to the trigeminal nerve), like ammonia or vinegar, as they can be detected even when the sense of smell is lost.

Parosmia is an olfactory perception that does not correspond to the stimulation. This confusion of smells occurs mainly with chronic nasal or sinus discharge.

Olfactory hallucinations or *fantosmia* are perceptions of smells that have no object. They almost always consist of unpleasant smells: rotten fish or eggs, gasoline, excrement, and so on. Their origin is generally central. They are found:

- with some mental illnesses: paranoid schizophrenia, chronic alcoholic psychosis
- with irritating lesions of the cortical center of olfaction at the tip of the 5th temporal convolution (uncus of the hippocampus). The uncinate fit is an epileptic crisis that begins with an olfactory aura.

Etiology

Various ailments can underlie these disturbances:

- loco-regional lesions:
  — nasal polyps
  — rhinitis, coryza
  — congenital malformations
  — sinusitis
  — allergies
  — tumors
  — viral attacks
- intoxicants:
  — tobacco
  — cocaine
  — neurotoxic medications
  — alcohol

- trauma:
  - — fractures of the anterior skull
  - — sagittal trauma to the skull where the anterior–posterior movement of the brain severs the olfactory nerves at the cribriform plate
  - — posterior blows (contre-coup lesions)
  - — falls on the coccyx
  - — whiplash
- tumors:
  - — those affecting the anterior brain. Tumors of the anterior fossa (meningiomas) often manifest as unilateral or bilateral anosmia, associated with visual disturbances and cognitive changes. Tumors of the hypophyses (pituitary tumors) do not affect the olfactory pathways, except when they spread beyond the sella turcica
- infections:
  - — meningitis
  - — frontal lobe abscess; anosmia must be sought out systematically in the subject who presents with progressive behavioral changes
- endocrinopathy:
  - — diabetes
  - — hypothyroidism
  - — Cushing's syndrome
  - — renal insufficiency
- congenital malformations:
  - — agenesis of the olfactory nerves
  - — albinism
- lesions of the central nervous system:
  - — major neurological syndromes
  - — multiple sclerosis (before the age of 40)
  - — Parkinson's disease
  - — Alzheimer's disease (after the age of 40)
  - — epilepsy.

10.3 MANIPULATION

10.3.1 Techniques

The techniques for manipulating the olfactory nerve are carried out via the endonasal route using a Q-tip 10–15 cm long (Fig. 10.4). The cotton swab should be the thinnest possible and can be dampened in serum or in sweet almond oil to facilitate passage into the nostrils. While this technique is sometimes unpleasant, it should never be painful.

Although some doctors, particularly at the beginning of the 20th century, employed nasosympaticotherapy techniques our practice is based not on their work and evidence but rather on our own experience. The same holds true for our work on the peripheral nerves.

Fig. 10.4 Materials for endonasal manipulation.

Generally, we use two manipulation sites:

- the vomeronasal organ and the terminal nerve
- the olfactory epithelium and the olfactory nerves.

Vomeronasal organ

Presentation

The terminal nerves are a specialized bundle of nerves within the olfactory tracts. It arises from a bulge of the olfactory bulb called the accessory olfactory bulb, passes through the cribriform plate, and descends obliquely in an anterior and caudal direction on the partition. It ends up at the vomeronasal organ (organ of Jacobson[1]), a 2–7 mm conduit situated a short distance from the opening of the

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1. Ludwig Levin Jacobson (1783–1843): Danish anatomist and physician (Copenhagen).

nostrils, on the anterocaudal aspect of the septum (Fig. 10.5).

The vomeronasal organ is a diverticulum of the olfactory organ. It is an isolated part of the sensory epithelium made up of typical olfactory cells.

Roughly speaking, the vomeronasal is a small circular or oval depression on the anterior part of the septal cartilage (Fig. 10.6). It can be unilateral or bilateral. A recent study suggested it is more often bilateral in men than in women (Besli et al 2004). It varies in size from 0.2 to 2 mm.

It plays a vasomotor and vasosensory role and participates in the sense of smell. It is also a vestige of our animal life when the sense of smell was essential in the detection of both enemies and sexual partners.

The vomeronasal organ is present in the embryo. For a long time it was thought that the vomeronasal was absent or vistigial and not used in humans. However, recent research has indicated that it does indeed exist in the

Olfactory bulb

Vomeronasal organ

Nasopalatine nerve
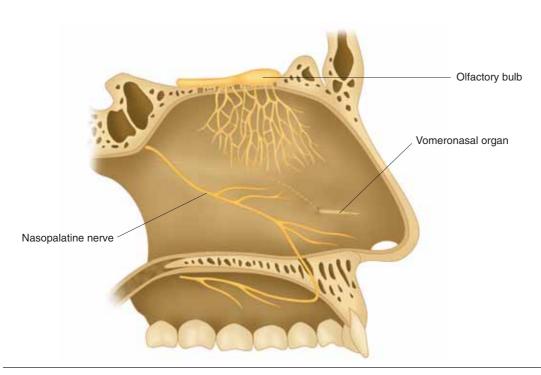
Fig. 10.5 Vomeronasal organ and the terminal nerve.

Vomeronasal organ

Fig. 10.6 Vomeronasal organ.

adult. In 1998, Gaafar et al examined the nasal septum in 200 adults. Instances of the vomeronasal organ varied according to examination technique. With the use of an anterior rhinoscope, the vomeronasal organ was perfectly visible in 32 subjects (16%), whereas with nasal endoscopy, the organ was found in 152 subjects (76%).

The exact function of the vomeronasal organ is not clear, but it does contain pheromone receptors. Pheromones are odorous secretions that play an essential role in sexual attraction and are important for animals in territory recognition. The work of McClintock (1971) has shown that pheromones play a role in the synchronization of the menstrual cycles of cohabiting women (convent, boarding school, prison, and so on).

Cotton swab technique
Caution: As with all internal techniques, extreme care must be taken. The structures of mechanical protection are very fragile. At all times we must be in control of our movements.

To reach the vomeronasal organ, the axis of penetration must be parallel to the floor of the nasal cavity when advancing the swab into the nostril. The therapist should "aim" horizontally at the level of the external auditory meatus (Fig. 10.7).

The target is situated against the nasal septum, at the anterior part of the vomer where the bone meets the cartilage. It is located in the anterior third of the nasal septum at a distance of 1.5–2 cm behind the posterior margin of the nostrils and about 0.5–1 cm above the floor of the nasal cavity.

The fingers delicately control the endonasal tissues, through the cotton swab.

Look for an area of increased density; more often than not it is depressed, but is sometimes faintly bulging and is generally quite sensitive for the patient. To start with, take

Fig. 10.7 Technique for manipulating the vomeronasal organ.

advantage of this sensitivity to locate the area of manipulation (Fig. 10.8).

When the nasal orifice is sufficiently open, it is possible to observe the vomeronasal organ by the light of an otoscope or a simple flashlight. Visually, in the rhinoscope, the vomeronasal organ resembles a small, well-delineated ulcer and cannot be confused with a septal ulcer, which has irregular contours.

Apply the dampened cotton end of the swab against the small induration that you have located, and press it lightly against the nasal septum. Release the pressure just barely and follow the listening of the tissues, making small axial rotations. Follow the tissue attraction by carrying out induction.

Once the listening stops, the technique is finished. Repeat the same technique in the other nostril.

Manipulation of the epithelium and the olfactory nerves

When the therapist enters the nostril to reach the olfactory epithelium, the axis of the cotton
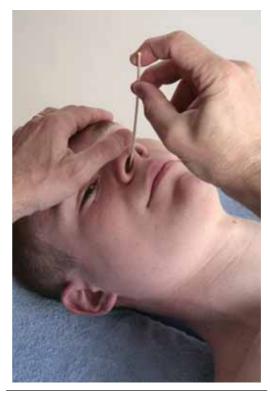
Fig. 10.8 Manipulation of the vomeronasal organ.

swab is parallel with the bridge of the nose. Aim for the inner corner of the eye (Figs 10.9 and 10.10).
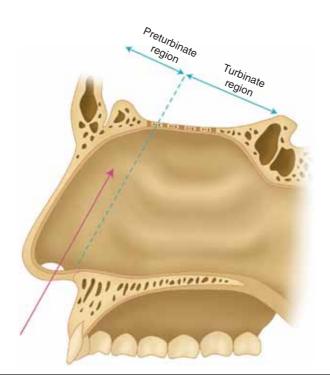
The cotton swab is moved slowly in a cranial direction towards the preturbinate region (in front of the turbinate bones) while remaining in close proximity to the septum.

With the thumb and index finger of your free hand, exert anterior traction on the cartilage of the nose. This facilitates orientation of the penetration of the swab and creates sensory distraction. Ask the patient not to make any sudden movements of the head or to brush away your hand in a gesture of protection.

When you are first learning this technique, progress slowly and respectfully with the swab in order to memorize the pathway. The olfactory area is situated at least 5 or 6 cm from the opening of the nostril.

Fig. 10.9 Axis of nasal penetration.

Preturbinate region

Turbinate region

Fig. 10.10 Endonasal route.

To reduce the sneezing reflex advise the patient:

- to sweep the palate strongly with the tongue
- to breathe in and out slowly through the nose.

With experience you will be able to accelerate the progression of the swab, and then the speed of the maneuver together with adherence to the advice noted above, will ensure that sneezes are very rare. Some patients may experience watering of the eyes, but this should only be a slight bother for them. Have tissues available.

Caution: Encountering an obstacle during the technique constitutes a contraindication. It could be a polyp, but is more likely to be a deviation of the nasal septum that reduces the passage between the concha and the septum. If it is a deviated septum, it is generally possible to perform the technique on the opposite side, and we will have to be satisfied with this unilateral approach.

With the cotton swab pushed high into the nostril, lightly tip it up towards the back while carefully drawing the stick towards you. Once it is in place, proceed with small strokes on the epithelium and the nerves. Do this two or three times on different areas, moving closer to the narrow part of the passage.

This is a delicate sensory area that should be manipulated without over-compressing. Always be very gentle and remain mindful of the highly reactive anatomy. The olfactory tissues and the respiratory mucous membranes react rather like the eyeball and the eyelids when an insect lands in the eye. When possible, always manipulate both sides (Fig. 10.11).

Neuro-encephalic manipulation

The cotton swab is positioned in the nose, creating mechanical tension on the olfactory nerves, thereby affecting the encephalon and the limbic centers.

Cranial listening allows you to feel these areas. Using induction, focus the primary respiratory mechanism on these locations, becoming more and more precise with each phase.

Finish with the visco-elasticity technique at the location in the brain to which your listening hand was attracted.

Emotional discharge

For those who practice the technique called "emotional discharge", try doing this while

Fig. 10.11 Manipulation of the olfactory nerve endings.

leaving the two cotton swabs in place in the nostrils (Fig. 10.12).

To concentrate more on the emotional rather than the physical aspect, your dominant hand is placed as for cranial listening, with a very light touch, just at the limit of contact with the skull. Placed very lightly, your hand will find the location where the brain has stored the greatest psycho-emotional tensions, more precisely in relation to the olfactory system.

With the same very light touch, carry out induction until the listening comes to an end. The goal is to discharge excess tension, often at the origin of an emotional hyper-reactivity with risk of decompensation for the patient. This technique allows the hyper-reactivity to be diminished, while respecting the patient's protective barriers.

10.3.2 Precautions and contraindications

The precautions and contraindications are as follows:

- fragility of the mucous membranes or of the vessels of the nose
- local hemorrhages
- encountering an obstacle during penetration.

10.3.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- cranial dura mater: the falx of the brain
- sacrococcygeal dura mater
- fronto-ethmoidal suture
- vomer
- sphenobasilar synchondrosis
- synchondrosis between the pre- and post-sphenoid.

10.3.4 Indications

There is a wide range of applications for the olfactory nerve technique. It is difficult to make an exhaustive list, but here are the main indications.

Effect on the mucous membranes and sinus pressure

It is not directly through the medium of the olfactory nerve, but rather through the maxillary nerve that an effect is produced on the nasal mucous membrane. This technique has a positive effect on frontal, maxillary and sphenoidal sinus pressures.

Fig. 10.12 Emotional discharge.

Post-traumatic anosmia

Caution: This is not developmental anosmia. Sudden anosmia is generally a sign of a rupture of the nerve fibers at the cribriform plate of the ethmoid. However, there are cases where olfactory acuity declines progressively following trauma. Classically, it is "*advanced meningeal fibrosis*" that constricts the olfactory nerve and decreases conduction. This phenomenon can also be observed after neurosurgery. Manipulations of the olfactory nerve, coupled with craniosacral manipulations, are very effective for this type of problem.

Psycho-emotional problems

Numerous psycho-emotional dysfunctions appear to respond favorably to this type of manipulation. Some emotional problems, like depression, have their roots in old emotional conflicts. It seems that, via manipulation of the olfactory nerve, we are able to help the patient to "discharge" a certain amount of stored emotion.

Eating disorders

Bulimia and anorexia are the two extremes of eating disorder behavior. The rhinencephalon is heavily involved in the neurobiological activity of eating and of satiation.

Eating disorders are difficult to make sense of and psychotherapy is often indicated. They can have a neurological foundation through the role of the rhinencephalon. Even years after the apparent disappearance of symptoms, food and the partaking of it can still be recurring problems. Manipulation of the olfactory nerves is a very good adjunct to psychotherapy treatment.

Sympathetic dystonia

The amygdala and the hippocampus play a major part in the regulation of the hypothalamus–pituitary axis. The rhinencephalon and the limbic system are thus a very interesting port of entry through which to reach the thalamus, which controls, among other things, the tone of the autonomic nervous system.

Manipulation of the olfactory nerves serves to ameliorate a great many autonomic imbalances. Some long-standing situations connected to the causes of emotional conflict or resulting from trauma have been improved by this technique.

Pathological heavy sweating has an important emotional component. It also responds well to this type of manipulation.

Some forms of anxiety

Anxiety, hypersensitivity to psychological stress and post-traumatic stress are forms of pathological expressions of fear. A great many stimuli can trigger anxiety. The olfactory system is certainly implicated in the storage of smells present at accidents.

Panic attacks, especially agoraphobia, respond well to manipulations of the vomeronasal organ and treatment of the olfactory epithelium. We have helped patients who suffer from panic attacks, tightness of the chest, fear of death, fear of loss of self-control, or loss of the sense of personal identity.

Connection to the outside world through odors and the messages relayed by the pheromones is of great importance in the phenomenon of anxiety.

Visceral dysfunctions

Some esophageal and gastric attacks, especially after stress, shock and emotional conflicts or after giving up smoking, are frequently accompanied by allergic rhinitis.

Generally, patients report alternating episodes of stomach pain and rhinitis. When the stomach pain improves, the rhinitis reappears, and vice versa. It is precisely in these cases that manipulation of the olfactory nerve is beneficial. The local action on the nasal mucosa and on the terminal branches of the olfactory nerves doubles the limbic system feedback, bringing about often spectacular improvements to this alternation of symptoms.

Low libido

We have performed these manipulations in the case of patients with low libido, usually patients between the ages of 45 and 60 with no detectable medical problems. While not impotent, they have low sex drive.

It seems that manipulation of the vomeronasal organ is able to produce astonishing results in this department. Is this achieved through the reinforcement of pheromone sensitivity?

Attempts to stimulate the vomeronasal organ using steroids cause elevated testosterone levels (Monti-Bloch et al 1998). Could this be the same mechanism behind the effectiveness of our own manipulations?

Menstrual problems

In numerous cases urogenital manipulations combined with cranial manipulations directed at the hypothalamus–pituitary–ovarian axis produce excellent results with regard to menstrual pathologies (irregular periods, amenorrhea, dysmenorrhea, and so on).

When the results are incomplete or difficult to maintain, manipulation of the vomeronasal organ enables the hormonal system to achieve a new and more lasting equilibrium.

Irregular menstrual cycles are a complex phenomenon with numerous causes. In medicine, there is a syndrome known as Morsier–Kallman's *olfactory–genital syndrome*, in which anosmia is linked to amenorrhoea. This pathological entity is a good illustration of the connections that exist between the olfactory system and the hypothalamus–pituitary axis.

The work of Stern and McClintock (1998) also demonstrates the role that an individual's sensitivity, particularly to pheromones, can play in the regulation of ovulation.

Optic nerve

The optic nerve (II) is the sensory nerve of sight. It is formed by axons of the retinal ganglion cells. With a diameter of 44 mm, the optic nerve is made up of about a million largely myelinated axons. To understand the function of the optic nerve properly, we will look briefly at the anatomy of the eye.

11.1 ANATOMY

11.1.1 The optic nerve

The optic nerve is shown in Figure 11.1.

Origin

The optic nerve begins at the anterolateral corner of the optic chiasm.

Pathway and relationships

The optic nerve, about 5 cm long, is directed anteriorly and laterally, and runs through the optic canal to reach the orbit. It can be described as having four sections: intracranial, intracanal, intra-orbital and intrabulbar.

Intracranial section

Located in the middle cranial fossa, the nerve runs along the chiasmic cistern. In the body of the sphenoid the optic chiasm rests on the diaphragma sellae (tentorium of hypophysis) and the lateral aspect of the groove of the sphenoid bone. Somewhat flattened, it measures 5 mm in width and 3 mm in height.

Intracanal section

Here the nerve is located in the optic canal between the cranial and orbital cavities. Its flattened shape rounds out; its width is now about 3 mm. The optic nerve is fastened to the wall of the optic canal by the dura mater. It is accompanied by the ophthalmic artery.

Intra-orbital section

At the exit of the optic canal, the optic nerve crosses the common tendinous ring (annulus of Zinn), to which are attached the four rectus muscles of the eye. The nerve occupies the center axis of the musculofascial cone of the bulb, in the middle of the orbital fat body. It is surrounded by three sheaths, which are extensions of the cranial meninges.

Curves of the optic nerve

The curves are important to know because they permit the optic nerve to adapt to movements of the eye. Also, we believe that the narrowing of the nerve where it meets the eyeball (from about 5 mm to 1.5 mm) is another factor that assists the mobility of the eyeball.

- The first curve is posterior, with a caudal and medial concavity.
- The second curve is anterior, with a lateral concavity.

These two contours allow the optic nerve to be much longer than it would be if it were straight. The extra available length affords the eyeball even greater mobility.

Optic nerve

Ophthalmic artery

Zygomatic bone

Optic chiasm

Nasociliary nerve
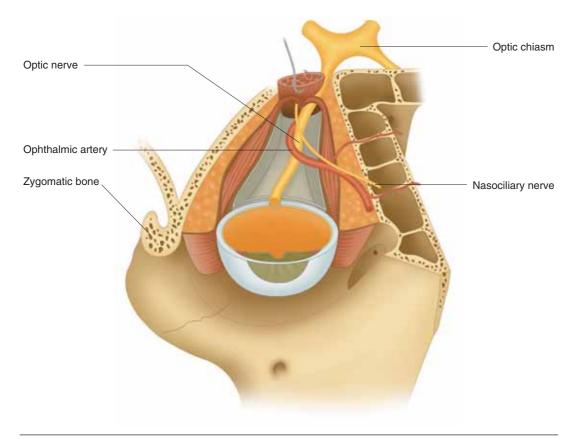
Fig. 11.1 Optic nerve.

Note for manual therapists

Mechanically, the curves of the optic nerve are important in facilitating the movement of the eye. Remember that it is the medial curve that is of particular interest in our manipulations. The techniques of compression–decompression of the eyeball are addressed to the optic nerve and its sheaths. During the decompression phase, a subtle sinuous movement can be perceived, which is probably due to these curves.

Dural sheath of the optic nerve

The dural sheath is quite thick and fibrous. It is continuous with the cranial dura mater (Fig. 11.2), and this feature allows us to have a general effect on the dura mater when we manipulate the optic nerve. The adherence of the optic nerve to the walls of the optic canal, through the intermediary of the meninges,

implicates the nerve in the case of skull fractures and sinus infections.

Useful relationships

As it crosses the orbit, the optic nerve relates to:

- the ophthalmic artery, which goes around its lateral aspect, becoming superior to it, and the posterior ciliary arteries, which surround it
- the superior and inferior ophthalmic veins
- the long ciliary nerves and the ciliary ganglion located on its lateral aspect, where the middle third runs into the posterior third.

Intrabulbar section

Here the optic nerve is unmyelinated, and has narrowed to a width of 1.5 mm. It relates to

Central retinal artery

Central retinal vein

Pia mater sheath

Arachnoid sheath

Subarachnoid space

Dura mater sheath
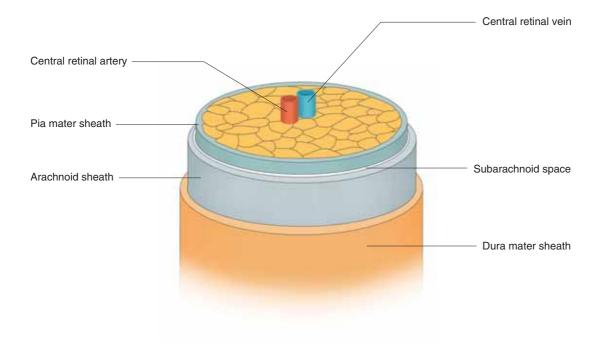
Fig. 11.2 Dural sheaths and the optic nerve.

the sclera and the choroid coat. It penetrates the eyeball about 3 mm to the nasal side of center and 1 mm below the posterior pole of the eyeball. It is important for therapists to locate the insertion of the nerve on the eye in order to optimize our effect on the dura mater (Fig. 11.3).

Terminal part of the optic nerve

The neurofibers shed their myelin sheaths when they cross the lamina cribrosa sclera to arrive at the retina.

The dural sheath bends laterally at an angle of about 100°, to continue with the sclera.

On a mechanical level, this dural obliquity is well suited to provide for ample lateral movement of the eye.

Vascularization

Arteries

The optic nerve has close connections with the sinuous ophthalmic artery and its branches, which wind around it.

The arterial vascularization is supplied by the vascular pia mater network, which derives from the short posterior ciliary arteries, and by an axial network formed from the central retinal artery. Near the eyeball, this artery penetrates the optic nerve, accompanied by its vein.

The ciliary arteries irrigate the remainder of the intra-orbital portion of the nerve. The cranial and intracanal sections are fed from the arterioles of the anterior cerebral artery and from a branch of the hypophysial artery.

Note for manual therapists

Due to the sinuous course of the ophthalmic artery, its branches and the central retinal artery, which is contained in the nerve, all manipulations of the optic nerve have a vascular effect, both mechanically and reflexogenically.

We also believe that it is possible to have an effect on the pituitary via the tubero-hypophysial artery and because the dural sheath of the optic nerve is contiguous with the tent of the diaphragma sellae.
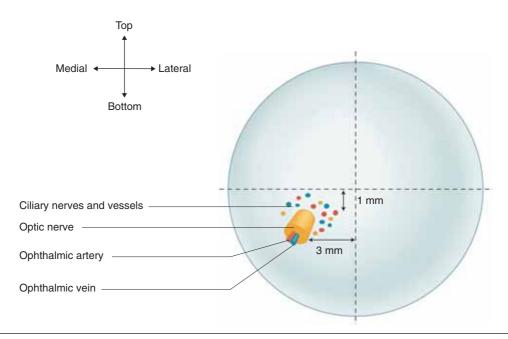
Fig. 11.3 Ocular junction of the optic nerve.

Veins

The veins drain into the central retinal vein, which in turn drains into the cavernous sinus.

Lymphatics

The optic nerve has a sizeable lymphatic system, which is not the case with all nerves.

Innervation

Historically, it was on the optic nerve that Sappey (1867) did the original work that led to his discoveries relating to the nervi nervorum.

The optic nerve is richly innervated. It is because of this intrinsic innervation that many pathologies of the optic nerve are generally very painful.

In cases of neural dysfunction, considerable proprioceptive misinformation is generated by the optic nerve.

11.1.2 The eye

Knowing that the optic nerve adheres to the sclera, we shall look briefly at a few essentials pertaining to the anatomy of the eye.

Eyeball

The eyeball measures about 2.5 cm in diameter. Its wall is opaque and very strong (Fig. 11.4).

Wall of the eye

The wall of the eye is made up of three structures: the sclera, the uvea and the retina.

Sclera

The sclera measures 1 mm in thickness. It is a tough membrane, white in color when healthy (white of the eye). The central part is translucent; this is the cornea. At the periphery of the cornea is a small canal (of Schlemm) that allows the aqueous humor to fill the anterior chamber and escape into the ciliary venous system.

Uvea

The uveal tract is a continuous musculovascular membranous structure. Its anterior part consists of the iris and the ciliary body. Its
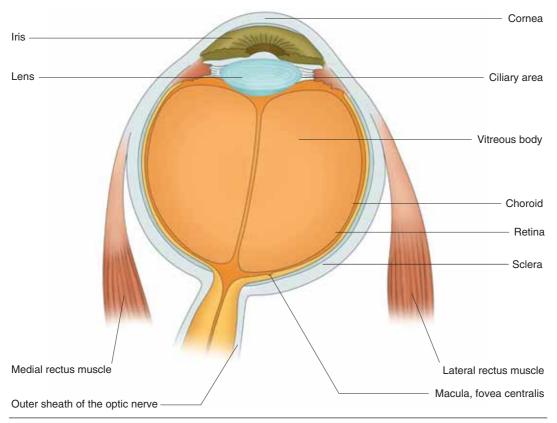
Fig. 11.4 Horizontal section of the eyeball.

posterior part covers the inner surface of the sclera and is known as the choroid.

- The *iris* is a taut membrane behind the cornea. The color of the iris varies, depending on the individual. Near its center is the round aperture of the pupil, which dilates and constricts by the action of the pupillary muscles.
- The *ciliary body* consists of the ciliary muscle that modifies the curvature of the lens in accommodation, and of the ciliary processes that radiate around the lens.
- The *choroid* lines the sclera of the middle and posterior parts of the eye. It is its very dark brown pigmentation that gives the eye its "darkroom", which helps limit reflections within the eye that would potentially result in the perception of confusing images.

Retina

The retina lines the interior of the "darkroom". Through its cones and rods it receives all the visual light rays. Posteriorly, there are two markings: the macula lutea and the optic disc.

Optic system

The optic system is formed by the clear structures of the eye: the cornea, the anterior and posterior chambers, the lens and the vitreous body (Fig. 11.5).

Cornea

The cornea is located at the central anterior aspect of the eyeball. It is clear, circular and projecting. Its curvature is greater than that of the rest of the eyeball. It can be likened to the glass of a watch.
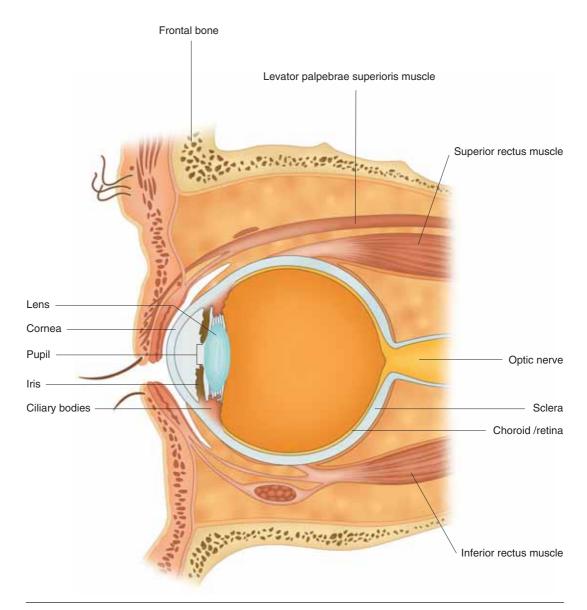
Fig. 11.5 Sagittal view of the optic system.

Anterior chamber
The anterior chamber is contained between the cornea and the iris. It is filled with the aqueous humor secreted by the ciliary processes.

Posterior chamber
The posterior chamber is located between the iris and the lens.

Lens
The biconvex lens is situated between the iris and the vitreous body. It is closely surrounded by its capsule, an elastic and transparent membrane.

Vitreous body
The vitreous body is of gelatinous consistency and fills the interior of the eyeball. It is

enveloped by the hyaloid membrane, which holds it against the retina.

Innervation of the eye

Sensitivity innervation
The ophthalmic branch of the trigeminal nerve and a sympathetic branch of the internal carotid plexus provide the eye's sensitivity.

Sensory innervation
The optic nerve and its 800 000 nerve fibers carry information to the cortico-occipital visual areas.

Intrinsic motor innervation
It is the nervous system of the pupil that controls the:

- dilatation of the pupil: sympathetic fibers from the internal carotid plexus
- contraction of the pupil: the oculomotor nerve.

Movement of the eyelids is provided by the oculomotor and facial nerves.

Pupillary reflexes
The pupillary light reflexes are controlled by the sympathetic cervical plexus and the oculomotor nerve. They regulate the penetration of light into the ocular chamber.

11.2 PHYSIOLOGY AND PATHOLOGY

11.2.1 Functions of the optic tracts
The visual fields have a "reversed" projection on to the retina, vertically as well as laterally. What is high is projected downwards, and vice versa. Objects to the right are projected on the left, and vice versa (Fig. 11.6). Consequently, damage to the right side of the retinas, for example, or to the neurons that gather impulses, causes flawed perception in the left visual fields. Similarly, a lesion of the superior half of the retina or of the neurons that receive the impulses produces faulty per-

ception in the inferior part of the visual field.

Optic pathways
In the optic chiasm the anterior nerve fibers coming from the retina undergo partial decussation, about half of them crossing over to the optic tract of the opposite side. Thus the neurons of the left half of each retina go to the left external geniculate body, and those of the right half of each retina form the optic tract to the right geniculate body.

From the geniculate bodies arise the posterior neurons that transmit light to the visual cortices of the occipital lobes of the brain. It is therefore the right hemisphere that perceives the left visual field, and the left that sees the field on the right.

Note for manual therapists

When we find tension at the eye and the optic nerve, it is worthwhile establishing whether there is also tension at the occiput, and whether it is homolateral or contralateral:

- Homolateral tension signifies mechanical stress (dura mater, optic nerve).
- Contralateral tension indicates a functional visual problem.

11.2.2 Clinical notes
Testing of the eye and of the optic tracts includes the following procedures:

- the eye examination
- visualizing the depth of the eye
- measuring visual acuity
- observation of the pupillary reflex
- observation of the visual fields.

Examining the back of the eye and the visual acuity require at the very least an ophthalmoscope and a Snellen chart. However, these tests remain at the margins of our discipline. If in doubt, never hesitate to refer the patient to a specialist.

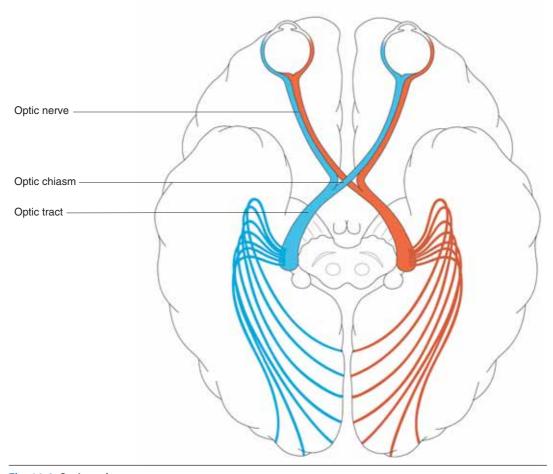Other tests are clinically simpler to implement, and so we describe them here.

Optic nerve

Optic chiasm

Optic tract

Fig. 11.6 Optic pathways.

Eye examination

Abnormalities of the eyeball
Abnormalities of the eye are legion and some are well beyond our scope of practice. It is nevertheless wise to be acquainted with a few simple abnormalities of the eyeball, as we use it as an intermediary to treat the optic nerve. Also, there are some contraindications relating to abnormalities of volume and tension.

Abnormalities of volume
• **Bilateral exophthalmia**. The eyes are normal in size but protrude markedly forwards. This is found with endocrine problems, notably Basedow's goiter which involves a moderate goiter, weight loss, nervousness and palpitations.
• **Unilateral exophthalmia**. This could be a sign of:
  — a tumor
  — a vascular problem
  — an inflammatory condition.
• **Bilateral enophthalmos**. The eyeballs are abnormally displaced backwards in the eye sockets. This is found in cases of poor general health and nutrition (malnourishment, vitamin deficiency, dehydration, periodontal infection, and so on).
• **Unilateral enophthalmos**. This condition suggests Horner's syndrome (miosis, drooping of the eyelid and homolateral facial vasodilatation).

Abnormalities of consistency

If the eye seems very *hard* on palpation, the following should be suspected:

- **Acute glaucoma**. This is accompanied by:
  — opacity of the cornea
  — mydriasis (prolonged or excessive dilatation of the pupil)
  — a very hard eyeball
  — violent headache
  — epiphora (an excessive flow of tears)
  — loss of visual acuity.

In the presence of the above signs, send the patient immediately to a physician.

- **Iridocyclitis**, if the pupil is dilated. This is inflammation of the iris, the cornea and the ciliary bodies.
- **Chronic glaucoma**, whose principal signs are:
  — glaucous eye (glaucoma comes from *glaukos*, meaning gleaming or gray)
  — abnormally elevated eye pressure
  — hard eyeball
  — gradual loss of visual acuity
  — dull pain around the eye: frontal, suborbital and temporal.
- **Trauma to the eye**. For example, vitreous hemorrhage or traumatic cataract (loss of transparency of the lens).

When the eye is *soft*, it feels like touching a deflated balloon. Any of the following can be the cause:

- With brutal trauma the eye can be blinded.
- If the trauma is less sudden, iridocyclitis can result.
- If the occurrence is spontaneous and does not follow trauma, suspect a detached retina.

Coloration of the white of the eye

Ecchymoses (the extravasation of blood into the skin of the conjunctiva) are red or purplish, and can be caused by the spontaneous rupture of blood vessels. Consider arterial hypertension and post-traumatic arteriosclerosis from a direct blow to the eye or fracture at the base of the skull.

Variation in pupil diameter

Parasympathetic fibers that innervate the intrinsic muscle of the eye are conveyed by the oculomotor nerve. The sympathetic fibers for the same muscles come from the internal carotid plexus.

Miosis

Miosis is a persistent constriction of the pupil due to excessive constriction of the sphincter muscle of the iris, or paralysis of the dilator pupillae muscle.

Among the many causes, consider:

- irritation of the sympathetic nerve that innervates the longitudinal fibers of the iris
- a lesion of the trigeminal nerve, in connection with the sympathetic system of the iris
- a central nervous system problem: medullary or cortical.

Paralytic miosis, a condition of the sympathetic nervous system and associated with Horner's syndrome, presents with drooping of the eyelid and sinking of the eyeball into the orbital cavity. The length of the course of the sympathetic fibers from the thalamus to the long ciliary nerves explains the multiplicity of possible causes for this syndrome.

Mydriasis

Mydriasis is abnormal and persistent dilatation of the pupil. It results from a contracture of the dilator pupillae muscle or from paralysis of the constrictor pupillae muscle. It is found with:

- glaucoma
- ocular trauma
- displacement of the lens
- blindness
- intoxication (alcohol, cocaine, mushrooms, belladonna, and so on)
- lesions of the oculomotor nerve.

Mydriasis can be the first sign of a problem with the oculomotor nerve (III), even before the appearance of paralysis of the intrinsic musculature of the eye.

Anisocoria

Anisocoria is an inequality in the size of the pupils. One is more open or more closed than the other. This is of interest to practitioners because it often corresponds to a fixation of the cervical spine or to a sympaticovagal disequilibrium.

When anisocoria is of cervical origin, it is generally on the side of the cervical fixation that the diameter of the pupil is smaller.

Note: With anisotension (inequality of the systolic arterial tension in the arms) the contraction of the pupil corresponds to the side of lowest tension. Remember that (apart from in major vascular disorders) anisotension is due to arterial vasoconstriction on the problem side. It is a local sympaticotonic problem of cervical or upper thoracic origin.

Pupillary reflexes

The photomotor reflex

Too strong a light can damage the retina. The photomotor reflex is a mechanism that controls the quantity of light penetrating the eye.

The afferent tract of this reflex is provided by the optic nerve (II), which conveys impulses to the Edinger–Westphal nucleus. At this point, light stimulation activates the parasympathetic visceromotor neurons, which send fibers through the oculomotor nerve and reach the eyeball by way of the ciliary nerves.

It is possible to test the photomotor reflex with a flashlight. Shine it directly into the eye being examined while protecting the other eye from the light. This provokes abrupt and forceful constriction of the pupil (direct reflex) followed by a slow dilatation. This unilateral stimulation also causes contraction of the untested eye (consensual reflex).

Remember that the sphincter pupillae muscle is innervated by fibers of the parasympathetic nervous system. Interruption of these fibers causes the pupils to dilate because of the unopposed action of the dilator pupillae muscle.

Absence of this reflex can occur with lesions of:

- the optic nerve
- the cerebral trunk
- the oculomotor nerve (the slowness of the pupillary response to light is the first sign of compression of the oculomotor nerve).

The accommodation reflex

For close vision, bilateral pupillary constriction accompanies the convergence of the eyeballs and the ciliaris contracts to modify the shape of the lens. It is synchronic and not a true reflex.

Defects of the visual fields

Visual field testing detects lesions of the visual pathway (Fig. 11.7). Patients may not be aware of changes in their visual fields until late in the course of a disease because lesions of the visual pathway often develop insidiously.

Abnormalities of the visual fields result from lesions that can affect various parts of the visual pathway. The type of defect depends on where the pathway is interrupted.

- Severing of the right *optic nerve* leads to *blindness/blinding* of the right temporal and nasal visual fields.
- Severing of the *optic chiasm* reduces the peripheral vision by creating *bitemporal hemianopia* (loss of half of the visual field of both eyes).
- Severing of the right *optic tract* eliminates vision from the left temporal and right nasal visual fields.

A lesion of the optic tract causes a contralateral *homonymous hemianopia* (indicating that the visual loss is in similar fields). This abnormality is the most common form of visual field loss.

Visual defects due to compression of the optic chiasm can result from a pituitary tumor or from an aneurysm of the internal carotid artery or of the pre-commissural part of the anterior cerebral artery.
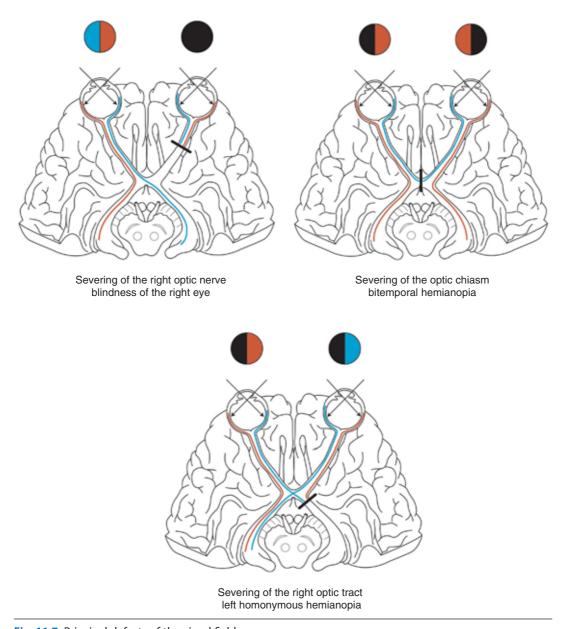
Severing of the right optic nerve
blindness of the right eye

Severing of the optic chiasm
bitemporal hemianopia

Severing of the right optic tract
left homonymous hemianopia

Fig. 11.7 Principal defects of the visual field.

11.2.3 **Defects of the optic nerve**

Optic nerve lesions cause total or partial loss of visual acuity in the corresponding eye. Optic neuritis is rarely painful. It manifests as various degrees of blurred vision.

Blindness is complete loss of sight, whatever the cause of the lesion (occipital cortex, optic pathways or the eyeball).

Amaurosis is a complete loss of sight resulting from an extra-ocular cause.

Amblyopia refers to reduced vision in one eye with no detectable structural cause.

Lesions involving only a subgroup of axons in the visual pathways produce scotomas, a defect of vision in a defined area in one or both eyes. Scotomas are also called *blind spots.*

83

Optical neuropathologies show up as a partial or complete loss of vision. They can be due to:

- ischemia (arteriosclerosis, embolism, and so on); diabetes is the premier cause of blindness in France
- inflammation or infection
- toxicity: alcohol, tobacco, botulism
- trauma resulting in compression of the optic nerve.

11.2.4 Optic nerve lesions

Problems with the optic nerve and visual function can have numerous causes:

- toxicity: tobacco, alcohol, diabetes
- diseases of the nervous system: multiple sclerosis, encephalitis, arachnoiditis, sphenoidal or ethmoidal sinusitis, meningitis, uveitis, sarcoidosis, toxoplasmosis, tuberculosis, and so on
- compression: tumors (glioma, perioptic meningioma), abscess, intracranial hypertension, hematoma, hydrocephalus, thrombophlebitis, ischemia, and so on)
- trauma: orbital fractures, trauma to the optic canal
- a defect of the visual cortex: homolateral hemianopia, quadrantanopia
- all progressive optic neuropathy.

11.3 MANIPULATION

11.3.1 Optic chiasm

The optic nerve runs obliquely towards the chiasm and is concave on its medial aspect.

We will use the same technique as for the orbital fissure, adding an active eyeball component.

Manipulation

The patient is supine, head turned to the side. For example, for the right side of the optic chiasm, place the left index finger on the homolateral palatine process of the patient's maxilla, and the right middle finger in the homolateral external meatus of the ear canal.

During the expansion phase, traction the maxilla anteriorly and draw the mastoid posteriorly while turning the patient's head slightly to the side being treated.

Ask the patient to look left and then right, without moving the head. According to the resulting increase in cranial tension that you feel, choose the direction of the patient's gaze and have him or her turn the eyes that way during the expansion phase.

Sometimes, because of the medial concavity of the optic nerve, we ask the patient to turn the head to the opposite side. Ultimately, it is the level of cranial tension perceived that determines the most effective direction.

Indications

Indications for treating the optic chiasm are as follows:

- dura mater tensions, owing to the intimate relationship of the dura mater and the optic nerve; we can also have an effect on the optic canal, on the osteodural plane
- mechanical pathologies of the eye (strabismus, poor coordination)
- sinusitis: an indirect effect through the frontal nerve
- circulatory problems of the eye and the face
- twitches, whether painful or not.

11.3.2 Optic nerve

Details

It is indirectly, through the eyeball, that we can have an effect on the optic nerve. The optic nerve has a winding course that allows the eye to move in virtually every direction without over-stretching. In restoring full movement to the optic nerve we also have an effect on the arterio-venous system that surrounds it and on its intraneural pressure.

The mobilization of the eyeball and the optic nerve brings about a thorough unfolding of the vasculature, and this release seems to play a significant role in the equilibrium of intracranial pressures.

Pathway of the optic nerve

It is important to emphasize that the optic nerve does not follow a straight course; its sinuous shape allows the nerve to adapt to the mobility of the eyeball and provides it with some extra length. Remember particularly that the nerve is also concave medially.

Ocular insertion of the optic nerve

The optic nerve inserts very close to the horizontal median line of the eyeball, slightly caudal and medial (see Fig. 11.3 above).

To have a more specific effect on the optic nerve, it is necessary to make contact with the caudal and medial quadrant of the eyeball and then to compress it posteriorly.

Intraneural pressure

The action of playing on the length of the optic nerve involves the principle of intraneural compression and decompression.

As we saw in the opening chapters of this book, pressure and tension are related. Stretching a nerve increases its pressure and with the release of the stretch (tension) pressure diminishes.

Pressing the eyeball posteriorly causes a reduction in the intraneural pressure, while on its return to the surface pressure is increased.

Some diseases like diabetes, or even poor nutrition, alcohol, smoking and certain medications, can affect the elasticity of the optic nerve and intraneural pressure.

Accompanying vascular structures

A great many arteries and veins accompany the optic nerve:

- central retinal artery
- supratrochlear artery
- ophthalmic artery
- posterior ciliary arteries
- lacrimal artery
- ethmoid artery
- superior and inferior ophthalmic veins.

The vascular smoothing out that accompanies the mobilization of the optic nerve has a more general ocular and circulatory effect.

We emphasize the importance of the ophthalmic vein, which has anastomoses with the veins of the face and empties into the cavernous sinus. An infection located in the face can have repercussions for the cavernous sinus.

Tests

Note: For all manipulations carried out via the medium of ocular pressure, be very careful with patients who have severe myopia. The retina of these patients is very fragile and too much pressure can cause damage. While these cases do not represent an outright contraindication, great gentleness is called for.

The "return" test

The patient is supine, with the eyes closed. Advise the patient that you will be pressing down on the eyeballs, without causing pain. If this creates trepidation, have patients place their index fingers on their eye and compress the eyeball by placing your thumb on their finger (Figs 11.8 and 11.9).

Note: This technique cannot be carried out on patients wearing hard contact lenses; they must remove them beforehand. Soft contact lenses are generally not a problem.

The thumbs make contact with the caudal and medial quadrant of the eyeball. Slowly and without causing pain, gradually compress both eyeballs.

When you feel you cannot compress any further, slowly release the pressure and analyze the course of the eyeball's return. It should take a curvilinear direction. The fixation is on the side where the eyeball has more difficulty returning to its original position.

The direct test

The direct test consists of directly stretching the optic nerve by mobilizing the eyeball laterally and cephalad. This is done by placing the medial rectus and superior oblique muscles under tension. To make this test more specific, first compress the eyeball posteriorly to release these muscles, and then draw the eye laterally and cephalad. The
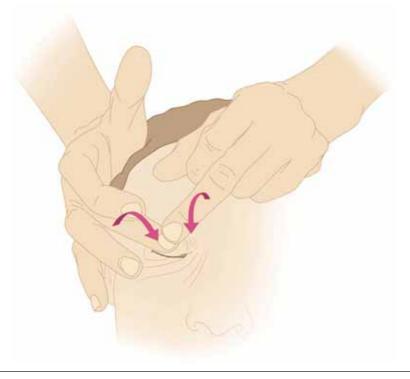
Fig. 11.8 Test of the optic nerve "in return" .

Fig. 11.9 Demonstration of the test "in return" .

fixation is on the side that offers the strongest resistance to the mobility test.

Manipulation

Stretching the optic nerve

Stretching the optic nerve consists of slowing down the return of the eye, as we do in all techniques of visco-elasticity.

Compress the eyeball in a posterior, medial and slightly caudal direction.

Allow the eyeball to return very gradually while drawing it laterally and cephalad to increase the tension on the optic nerve.

Perform the same technique using induction.

Oculo-occipital technique

The vision centers are in the occipital cortex. We like to combine the technique for the optic nerve with occipital compression–decompression (Fig. 11.10).

The patient is supine. Place the thumb of one hand on the eyeball and the palm of the other hand under the homolateral or contralateral occiput in accordance with the movement you feel.

During compression of the eyeball – during the retraction phase – the palm in contact with the occiput will feel reflected into it, little by little, this transmission of pressure exerted on it.

The thumb and hand work in concert, treating by induction during the expansion phase.

Indications

Strabismus
General points

Strabismus has many causes, which we will summarize. When in doubt, it is certainly best to refer the patient to a specialist.

Strabismus is characterized by a deviation in the visual axes (cross-eyes) and by difficulty with binocular vision. It is a defect of convergence of the two visual axes. The eye that looks straight is the fixing eye, and the other eye deviates towards it. Three percent of the population is affected.

There are various forms of strabismus:

- **Absolute strabismus**. This exists whatever the distance of the fixed point.
- **Accommodating strabismus**. This is due to excessive accommodation (hypermetropia).
- **Alternate strabismus**. One eye and then the other are affected.
- **Concomitant strabismus**. The deviation remains constant; the affected eye follows the movement of the other eye.

Fig. 11.10 Oculo-occipital technique.

- **Vertical strabismus**. The visual axis of the affected eye is deviated towards the top.
- **Convergent strabismus** (esotropia). The eye deviates medially.
- **Divergent strabismus** (exotropia). The eye deviates laterally.

In all cases of strabismus, it is important to consider the following two distinctions:

- **Banal strabismus** is a defect in the position of the two eyes in relation to each other. The deviation is permanent, constant and independent of the gaze. It is not accompanied by any limitation in movement of the affected eye. It is a divergent squint or convergent strabismus, without functional disturbance. These are the cases we most commonly see in our offices. They are frequently the result of constraints *in utero*, fetal malposition, and the use of suction or forceps at delivery.
- **Paralytic strabismus** is the inability of the ocular muscles to move the eye because of muscular paralysis.

Strabismus is accompanied by diplopia (double vision) for which the subject compensates by closing the eyelid of the paralyzed eye or by turning the head in order to position the eye better.

Note: Always beware of strabismus that appears suddenly. Suspect a local or central nervous system tumor, an opaque cornea, a cataract, a vitreous abnormality or a retinopathy.

Infant strabismus

We often see young children, even infants, who squint. Consider two possible situations:

- **Permanent strabismus during all movements**. There is no limitation of movement of the deviated eye. This is convergent or divergent banal strabismus, often resulting from abnormal compression of the skull *in utero* or during delivery. It does not involve significant functional problems.
- **Strabismus with limitation of movement of the eyeball to the opposite side**. This type involves paralysis of the ocular muscles. In this case the antagonistic muscle cannot compensate the paralyzed muscle, resulting in an unopposed muscle action. It is accompanied by diplopia (the subject has double vision, resulting from an affliction of the oculomotor nerve) and generally affects both eyes.

A patient presenting, for example, with a paralysis of the right lateral rectus muscle of the right eye was able to look left because of the antagonistic muscle. In looking straight ahead, he had a right convergent strabismus, and on looking to the right, bilateral strabismus. The patient attempted to correct his diplopia by closing the affected eye or by turning his head to a better position.

Trauma

Mobilization of the optic nerve, using the eyeball as intermediary, allows us to have an effect on the cranial dura mater.

Hormonal imbalance

This indication is subtle. We surmise that any effect we have is due to the close anatomical relationships between the dura mater and the pituitary gland.

Vascular problems of the eye

It is advisable to pay attention to the vascular fragilities found in diabetics and patients taking long-term anticoagulant medication, among others.

Disorders of the central nervous system

Multiple sclerosis, Parkinson's disease and other disorders fall into this category. We have no effect on the illnesses themselves but can achieve results on some of their consequences. Patients with this type of disease appreciate all the help we can give them.

Other effects
In the optic canal

The dura mater attaches to the periosteum and accompanies the optic nerve as far as the orbit. It forms a recess, which is concave

posteriorly, called the tent of the optic nerve, running from the sphenoidal limbus to the posterior clinoid process.

The dural, pia and arachnoid sheaths of the optic nerve are continuous with the sclera of the eye.

With trauma or infection, the dura mater can become fixated and fibrosed within the optic canal, and therefore limited in its physiological extensibility.

On the common tendinous ring (annulus of Zinn)

The common tendinous ring is a cone shape, with an anterior base and a posterior summit formed by the oculomotor muscles.

The optic nerve is surrounded by dura mater (and the other meningeal layers) and crosses the cephalic and medial part of the tendon between the superior and medial rectus muscles.

Any manipulation of the optic nerve has an effect on the meninges and on the common tendinous ring, which is particularly helpful in cases of strabismus.

Summary
In summary, we manipulate the optic nerve to have an effect upon:

- the dura mater
- the microvascular system of the eye
- mechanical ocular problems
- visual disturbances
- the equilibrium of intracranial pressure.

11.3.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- peri-optic dura mater: tentorium cerebelli
- sphenoid (lesser wing)
- bony orbit.

Oculomotor nerve

12

The oculomotor nerve (III) is the largest of the motor nerves of the eye. It innervates all the muscles of the orbit, with the exception of the lateral rectus and the superior oblique. Within it are carried parasympathetic fibers destined for the pupillary sphincter and the ciliary muscles.

12.1 ANATOMY

12.1.1 Origin

The fibers of the oculomotor nerve arise from the medial part of the cerebral peduncle. We can distinguish a medial or interpeduncular group of fibers, and a lateral group of fibers that emerge from the anterior surface of the peduncle. These fibers unite to form a nerve cord whose course we will examine.

12.1.2 Pathway

On leaving the peduncle, the oculomotor nerve runs forward, laterally and very slightly cephalad towards the lateral side of the posterior clinoid process (Fig. 12.1). Before the posterior clinoid process, it crosses the dura mater to enter the lateral wall of the cavernous sinus. From there, it penetrates the superior orbital fissure.

12.1.3 Useful relationships

With the basilar arterial system

The basilar trunk separates the two oculomotor nerves, which then pass between the posterior cerebral and superior cerebellar arteries.

In the cavernous sinus

The oculomotor nerve lies on the most cephalic part of the wall of the cavernous sinus.

In the superior orbital fissure

The oculomotor nerve crosses this fissure at its widest part and pierces the orbit, crossing the common tendinous ring (annulus of Zinn) formed by the two tendons of the lateral rectus muscles (Fig. 12.2).

12.1.4 Anastomoses and connections

The oculomotor nerve has some anastomoses and connections:

- with the ophthalmic nerve (branch of the trigeminal nerve)
- with the sympathetic fibers, principally branches arising from the carotid plexus.

12.1.5 Distribution

The oculomotor nerve separates into two branches either on entering the orbit or just before (Fig. 12.3). It divides into a superior branch (cephalic branch) and an inferior branch (caudal branch). Here the nasociliary nerve (branch of the ophthalmic nerve) is found between the two divisions.

Cephalad branches

The superior branch ascends the lateral aspect of the optic nerve and meets the superior
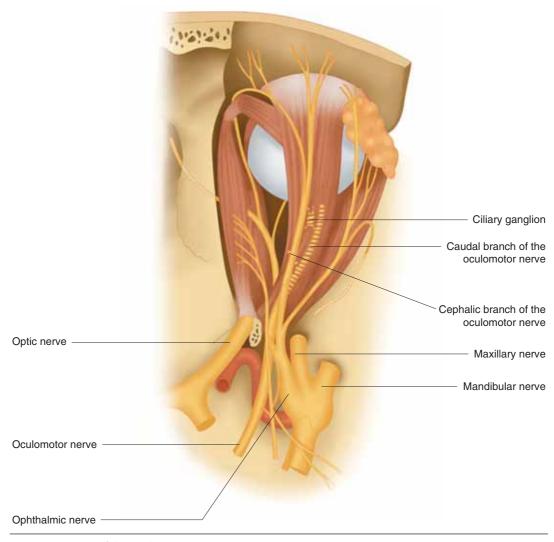
Fig. 12.1 Course of the oculomotor nerve (superior view).

rectus muscle of the eye. A small branch goes to innervate the levator palpebrae superioris.

Caudad branches

The caudal branch is more important than the smaller cephalic branch. It divides into three filaments:

- a medial filament, supplying the medial rectus muscle
- a caudal filament, supplying the inferior rectus muscle
- an anterior filament, supplying the inferior oblique muscle.

It furnishes the oculomotor root of the ciliary ganglion.

12.1.6 **Ciliary ganglion**

The ciliary ganglion is situated against the lateral surface of the optic nerve. It receives:

- the sympathetic root from the carotid plexus
- the nasociliary root from V
- the parasympathetic oculomotor root from III.

Chiasm

Oculomotor nerve

Ophthalmic veins

Frontal nerve
Lacrimal nerve
Nasociliary nerve
Ophthalmic nerve

Maxillary nerve

Abducent nerve

Trigeminal ganglion

Trochlear nerve

A

Trochlear nerve

Superior orbital fissure

Levator palpebrae superioris

Superior ophthalmic vein

Superior rectus muscle
Optic nerve

Lacrimal nerve
Frontal nerve

Cephalic branch of the oculomotor nerve
Lateral rectus muscle

Superior oblique muscle

Medial rectus muscle

Abducent nerve

Ophthalmic artery

Nasociliary nerve

Inferior rectus muscle

Caudal branch of the oculomotor nerve

Inferior ophthalmic vein

B

Inferior orbital fissure

Fig. 12.2 The oculomotor nerve in the superior orbital fissure. A: Intracranial view. B: Frontal section.
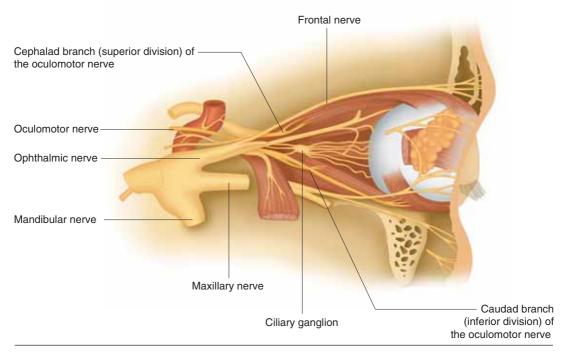
Fig. 12.3 The oculomotor nerve (lateral view).

The short ciliary nerves emerge from the ciliary ganglion.

12.2 PHYSIOLOGY AND PATHOLOGY

12.2.1 Functions

Oculomotor function

The nerve controls adduction (medial rectus muscle), depression (inferior rectus muscle), elevation (superior rectus muscle) and lateral rotation (inferior oblique muscle) of the eye (Fig. 12.4).

Muscle paralysis leads to strabismus.

Palpebral motor function

The oculomotor nerve lifts the upper eyelid.

Visceromotor function

This function is fulfilled by the parasympathetic efferents, which convey the oculomotor nerve.

The cellular bodies of the visceral motor neurons of III (oculomotor nerve) are located in the Edinger–Westphal nucleus. The pre-ganglionic neurons leave the brain stem with the neurons destined for the somatic, to make up the IIIrd cranial nerve. These parasympathetic neurons are located superficially on the nerve. As a result, in cases of compression of the nerve, they are the first to lose function. They leave the nerve, destined for the inferior oblique muscle, and end in the ciliary ganglion.

The short ciliary nerves, 6–10 in number, are branches of the ciliary ganglion. They emerge from the ganglion and penetrate the eyeball through its posterior surface, close to the insertion of the optic nerve.

In the eyeball, the nerves converge towards the front, between the choroid and the sclera where they are distributed to the ciliary bodies and the constrictor muscle of the pupil. By controlling the tone of these two muscles, the visceromotor fibers determine the diameter of the pupil and the shape of the lens.
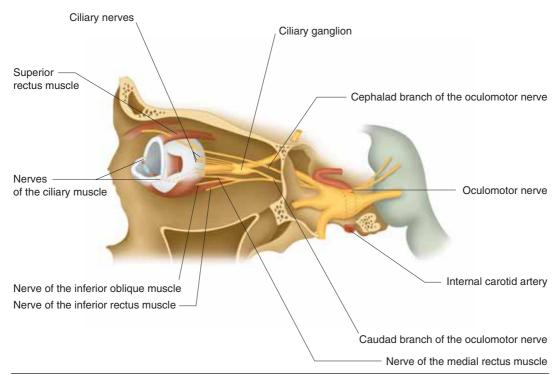
Fig. 12.4 Diagram of the oculomotor nerve.

Pupillary light reflex

In Chapter 11 we described in detail the mechanism of this reflex. Remember that the light impulse is transmitted by the optic nerve at the pretectal nucleus, which projects the signal to the accessory oculomotor nucleus (Edinger–Westphal), causing constriction of the pupils (miosis).

Accommodation reflex

The accommodation reflex is an adjustment of the eye for near vision (Fig. 12.5). Three phenomena are involved:

- **Increased convexity of the lens**. The suspensory ligament of the lens inserts peripherally into it. At rest, the ligament maintains tension on the periphery of the lens, such that it remains flat. During the process of accommodation the parasympathetic neurons of the Edinger–Westphal nucleus govern the contraction of the ciliary muscle (diminishing the distance a–b shown in Fig. 12.5), which relaxes some of the tension on the ligament, modifying the shape of the lens.
- **Pupillary constriction**. Parasympathetic fibers convey the impulse for the contraction of the sphincter pupillae. The pupil constricts and thereby increases the depth of focus.
- **Convergence of the eyes**. The oculomotor nuclei send the impulses for contraction of both medial rectus muscles, causing the eyes to converge.

12.2.2 Paralysis

To understand the role of the oculomotor nerve properly, let us look at what occurs in the case of total paralysis of the nerve (Fig. 12.6). The subject has:

- a lateral strabismus (abduction of the eye)
- an inability to move the eye cephalad, caudad and medial

95

Ciliary muscle

a ← → b

A   Distance vision   Near vision

Oculomotor nerve

Caudad branch of the oculomotor nerve

Optic chiasm

B   Medial rectus muscle
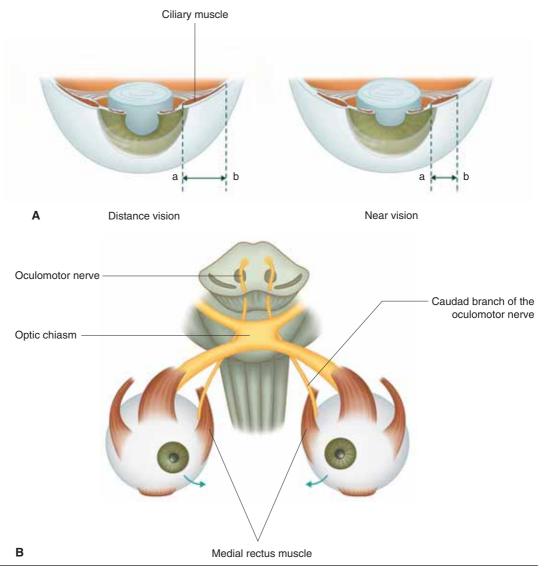
Fig. 12.5 The accommodation reflex. A: Horizontal section. B: Frontal view.

- ptosis
- mydriasis (dilation of the pupil) resulting from the interruption in the parasympathetic fibers destined for the iris
- an absence of the pupillary reflex (no constriction of the pupil in the presence of strong light)
- a loss of distance accommodation (no increase in the convexity of the lens permitting near vision) following upon paralysis of the ciliary muscle.

Etiology

A rapid increase in intracranial pressure, such as occurs with a subdural hematoma, for example, often compresses the IIIrd cranial nerve against the crest of the petrous part of the temporal bone. Owing to their superficial location, the parasympathetic nerve fibers are the first to be affected. As a result, the pupil gradually dilates towards the side of the lesion.

The first sign of compression of the oculomotor nerve is therefore the ipsilateral slowing
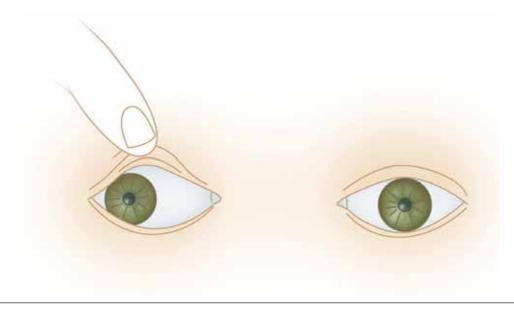
Fig. 12.6 Paralysis of the oculomotor nerve.

of pupillary constriction in the presence of light.

Equally, an aneurysm of either the posterior cerebral or the superior cerebellar artery can put pressure on the oculomotor nerve that passes between these two vessels.

The oculomotor nerve runs in the lateral wall of the cavernous sinus; it can therefore also be injured in cases of infection or trauma to the sinus.

An interesting clinical finding

The oculomotor nerve crosses the large circumference of the tentorium cerebelli and passes to the lateral aspect of the posterior clinoid process. This positioning may explain why, according to Lazorthes (1953), the nerve becomes compressed in cases of a transverse blow to the skull, and also temporal cerebral hernia in cases of cranial hypertension.

12.3 MANIPULATION

12.3.1 Technique for the superior orbital fissure

In the superior orbital fissure the oculomotor nerve runs very medial, accompanied later-ally by the abducent nerve and medially by the nasociliary nerve. Because of this, the emphasis of the technique is on the medial aspect of the fissure, while turning the patient's head to the side of the fissure being treated. This maneuver also has an effect on the foramen rotundum, through which the maxillary nerve passes. This technique should be applied by rotating the head to both sides, as other fixations are sometimes found. From theory to practice!

12.3.2 Technique for the eyeball

The muscles innervated by the oculomotor nerve are shown in Figure 12.7.

The superior rectus muscle of the eye

The superior division of the oculomotor nerve goes principally to the superior rectus muscle of the eye. This nerve may be stretched by mobilizing the superior aspect of the eyeball in an essentially caudad direction, during the cranial expansion phase. This technique is not specific to the muscle; it can also produce an effect on the frontal nerve, ciliary nerve, ciliary ganglion and ophthalmic artery, to mention just a few.
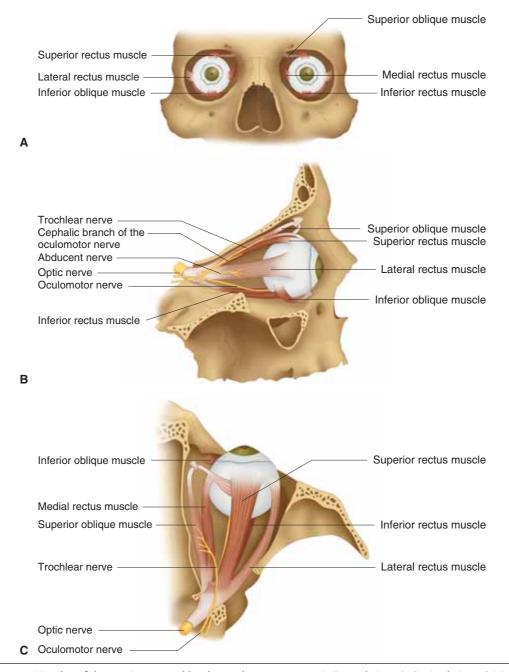
Fig. 12.7 Muscles of the eye innervated by the oculomotor nerve. A: Frontal view. B: Sagittal view. C: View from above.

Medial rectus muscle

The nerve of the medial rectus muscle comes from the caudal branch (inferior division) of the oculomotor nerve. To effect a stretch, draw the eyeball laterally during the expansion phase.

Inferior rectus muscle

The nerve to the inferior rectus muscle also stems from the caudad branch of the oculomotor nerve. To effect a stretch, draw the eyeball cephalad during the cranial expansion phase.

Figure 12.8 Manipulation of the eyeball.

Inferior oblique muscle

The inferior oblique muscle originates in the medial part of the caudad border of the orbit and directs the eyeball laterally. Its innervation also derives from the inferior division of the oculomotor nerve. To effect a stretch, draw the eyeball lateral and cephalad during the cranial expansion phase (Fig. 12.8).

12.3.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- the dura mater: intra-orbital lining, tentorium cerebelli

- the common tendinous ring
- the sphenoid: superior orbital fissure (lesser wing/greater wing)
- the frontosphenoidal suture (horizontal plate/lesser wing)
- the petrosphenoidal suture
- the wall of the cavernous sinus: tentorium cerebelli and intracavernous pressure.

12.3.4 Indications

The indications for manipulation of the oculomotor nerve are as follows:

- strabismus
- poor accommodation of the eye
- visual dysfunction
- problems with proprioception.

Trochlear nerve

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13

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The trochlear nerve (IV; formerly called the pathetic nerve) has two interesting distinctions. It is the most slender of the cranial nerves and also the one that follows the longest course.

It is solely a motor nerve and supplies just one muscle: the superior oblique muscle of the eye.

13.1 ANATOMY

13.1.1 Origin

The trochlear nerve originates caudal to the oculomotor nucleus at the level of the inferior colliculus. It emerges from the dorsal surface of the brain.

13.1.2 Pathway

After leaving the nucleus, the trochlear nerve emerges from the dorsal surface of the midbrain, winds around the brain stem and crosses the dura mater immediately below the free border of the tentorium cerebelli (Fig. 13.1). It passes forwards in the wall of the cavernous sinus and reaches the orbit by way of the superior orbital fissure.

13.1.3 Useful relationships

At the base of the encephalon

The trochlear nerve is located between the oculomotor nerve medially and the trigeminal nerve laterally.

In the cavernous sinus

The trochlear nerve runs above the ophthalmic nerve (VI) and below the oculomotor nerve. It crosses the oculomotor nerve behind the orbital fissure (Fig. 13.2).

In the superior orbital fissure

The trochlear nerve passes through the orbital fissure at its superomedial part outside the common tendinous ring (annulus of Zinn).

13.1.4 Anastomoses

The trochlear nerve has anastomoses:

- with the sympathetic fibers of the carotid plexus
- with fibers of the ophthalmic nerve (VI), generally via two branches:
  - the recurrent nerve of the tentorium cerebelli (or recurrent nerve of Arnold), which sends many filaments to the tentorium cerebelli at the caudal aspect of the falx cerebri
  - the lacrimal nerve.

13.1.5 Distribution

Dura mater

The trochlear nerve participates, along with the recurrent nerve of Arnold, in the sensitivity of the dura mater in the cranial region through which it passes.
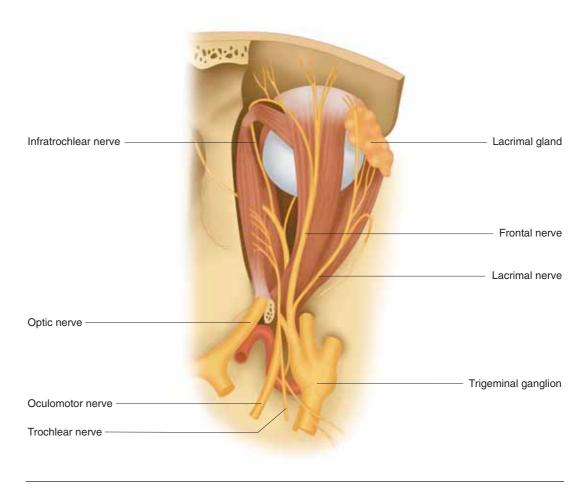
Fig. 13.1 Course of the trochlear nerve.

Orbital

The trochlear nerve crosses the cephalic branch of the oculomotor nerve, passes above the levator palpebrae superioris and the superior rectus muscle of the eye, and finally enters the orbital surface of the superior oblique muscle of the eye to supply its innervation.

13.2 PHYSIOLOGY AND PATHOLOGY

13.2.1 Functions

The trochlear nerve abducts, depresses and medially rotates the eyeball. The pupil moves inferiorly and lateral.

Deficit

A lesion of the trochlear nerve can lead to (Fig. 13.3):

- deviation of the eyeball towards the top
- inability to turn the eye inferior and lateral
- homonymous diplopia (to counteract this, the patient inclines the head to the opposite side).

Trochlear nerve damage is often combined with damage to nerves III and VI along their trajectory in the wall of the cavernous sinus (Foix syndrome), in the superior orbital fissure or even at the orbital apex (Rollet's apex syndrome).
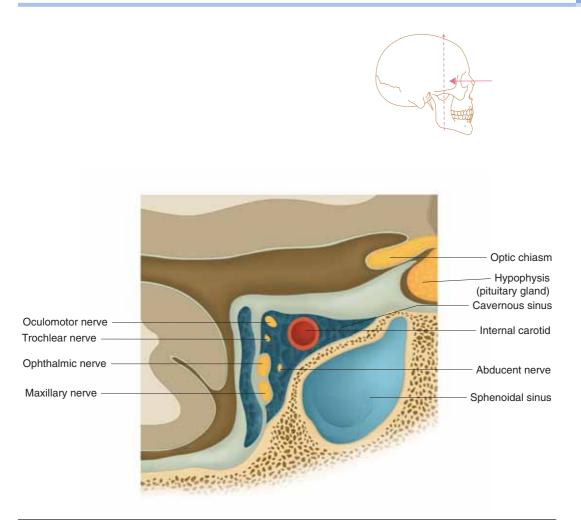
Fig. 13.2 Trochlear nerve and the cavernous sinus.

Optic chiasm

Hypophysis
(pituitary gland)

Cavernous sinus

Internal carotid

Abducent nerve

Sphenoidal sinus

Oculomotor nerve

Trochlear nerve

Ophthalmic nerve

Maxillary nerve
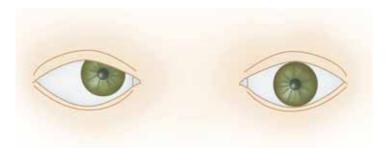
Fig. 13.3 Paralysis of the trochlear nerve.

13.3 MANIPULATION

13.3.1 The superior orbital fissure

The trochlear nerve occupies the medial aspect of the superior orbital fissure, where it is fairly large (Fig. 13.4). It is surrounded laterally by the frontal nerve and medially by the abducent.

This technique is an osteomeningeal maneuver performed during the expansion phase (see Ch. 9). It is indispensible for the

Trochlear nerve

A

Trochlear nerve
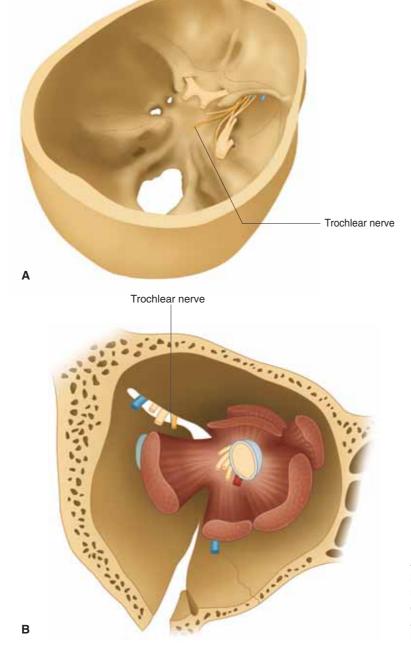
Fig. 13.4 The trochlear nerve in the superior orbital fissure.
A: Intracranial view.
B: Frontal section.

B

trochlear nerve when carried out together with the maneuver for the superior oblique muscle.

13.3.2 The eyeball

The trochlear nerve can be stretched by using the superior oblique muscle as intermediary. Draw the eyeball in a caudad and medial direction during the cranial expansion phase (Fig. 13.5).

13.3.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral

Superior oblique muscle

A

Superior oblique muscle

Trochlear nerve

B

Superior oblique muscle

Trochlear nerve

C
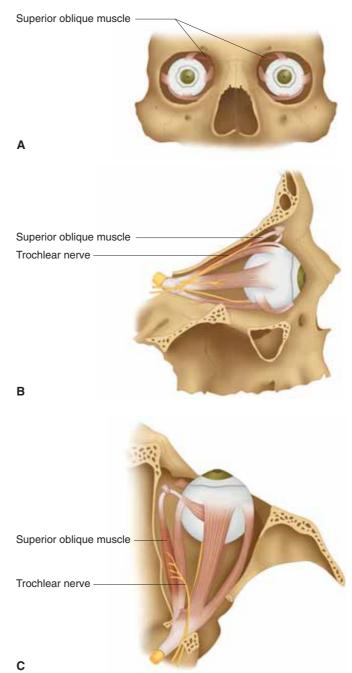
Fig. 13.5 Muscles of the eye innervated by the trochlear nerve. A: Facial view. B: Sagittal view. C: View from above.

mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- the dura mater: intra-orbital lining, tentorium cerebelli
- the common tendinous ring
- the sphenoid: superior orbital fissure (lesser wing/greater wing)
- the frontosphenoidal suture (horizontal plate/lesser wing)
- the petrosphenoidal suture
- the wall of the cavernous sinus: tentorium cerebelli and intracavernous pressure.

13.3.4 Indications

Convergent strabismus

Treatment of the trochlear nerve is most effective for lateral strabismus. As a general rule, the technique is applied to both eyes, even the one which appears to have no strabismus.

Cranial trauma

The trochlear nerve forms an anastomosis with the ophthalmic branch of the trigeminal nerve, which provides the sensitivity of the tentorium cerebelli via the recurrent nerve of Arnold. The trochlear nerve sends small branches to the dura mater, which surround it and join other nerve fibers.

Manipulation of the trochlear nerve is effective following cranial trauma, cranial surgery and paralysis.

Neurological sequelae

Consider the trochlear nerve in cases of paralysis and for neurological lesions originating in the central nervous system.

Trigeminal nerve

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The trigeminal nerve (V) is a mixed nerve: sensory for regions of the face and motor for the muscles of mastication. The sensory root is large, while the motor root is more slender. By way of its connections, the trigeminal nerve has sensory and secretory fibers.

Along the course of the sensory root lies the trigeminal ganglion (of Gasser).

Here the nerve divides into three branches: ophthalmic (V1), maxillary (V2) and mandibular (V3). The mandibular nerve is a mixed nerve, while the other two branches are sensory.

14.1 ANATOMY

14.1.1 Origin

The origin of the trigeminal nerve is the annular protuberance at the limit of the cerebellar peduncles. It emerges from the pons by two roots of unequal size: a small motor root and a large sensory root (Fig. 14.1).

- The large sensory root is made up of about 50 fascicles.
- The small root, the motor root of Wrisberg, is composed of six or seven fascicles.

14.1.2 Pathway

From the annular protuberance the two roots course anteriorly, laterally and cephalad towards the fibrous trigeminal cave (of Meckel) (Fig. 14.2). The cave is a recess between two layers of the dura mater of the middle cranial fossa lodged near the apex of the petrous part of the temporal bone. This recess envelops the roots of the trigeminal nerve and the posterior part of its sensory ganglion.

14.1.3 Trigeminal ganglion

Formerly known as the ganglion of Gasser, the trigeminal ganglion nestles in the trigeminal cave, where it divides into three branches (Fig. 14.3):

- the ophthalmic nerve (VI), the most slender
- the maxillary nerve (V2)
- the mandibular nerve (V3), the most substantial.

The small motor root extends along the medial side of the large sensory root as far as the trigeminal cave. It then runs under the trigeminal ganglion and merges with the mandibular nerve.

The trigeminal ganglion is shaped rather like a very flat bean. Its anterolateral surface is intimately linked to the dura mater, to which it strongly adheres. This anatomical connection is important for manipulations. We will look at a technique aimed at the ganglion where mechanical tension is created through the dura mater.

The trigeminal ganglion sends fibers to the dura mater, the sphenotemporal region and the petrosal sinus.

Ophthalmic nerve

Small trigeminal nerve root
(motor)

Large trigeminal nerve root
(sensory)

Maxillary nerve

Dura mater

Mandibular nerve

Trigeminal ganglion
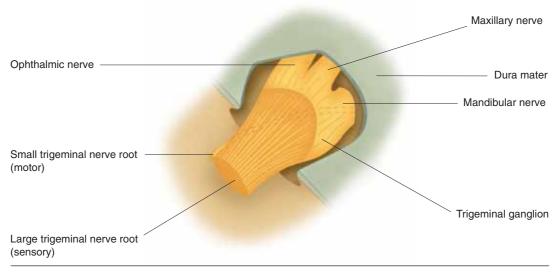
Fig. 14.1 Roots of the trigeminal nerve.

Falx cerebri

Optic chiasm

Ophthalmic nerve

Maxillary nerve

Mandibular nerve

Trigeminal ganglion

Tentorium cerebelli
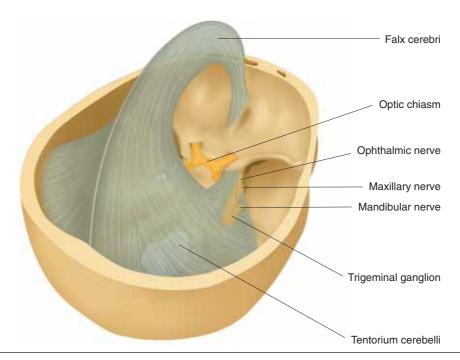
Fig. 14.2 The trigeminal cave.
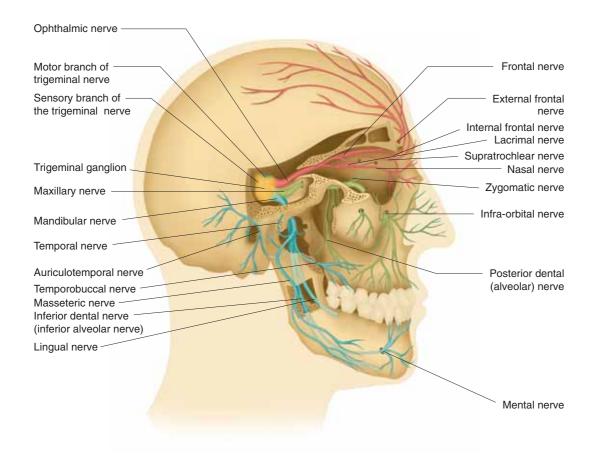
Fig. 14.3 Branches of the trigeminal nerve.

Important relationships

At its medial extremity, the trigeminal ganglion relates to the carotid artery; a single fibrous plate separates them. The ganglion receives sympathetic filaments from the carotid plexus.

Terminal branches

The terminal branches of the trigeminal ganglion are:

- the opthalmic nerve
- the maxillary nerve
- the mandibular nerve.

The trigeminal ganglion also has some fibers that go to the dura mater in the spheno-temporal region and to the petrosal sinuses.

14.2 PHYSIOLOGY AND PATHOLOGY

14.2.1 Functions

The trigeminal nerve is the great sensory nerve of the face (Fig. 14.4). Its territory is delimited by the coronal line passing through the vertex, the tragus and the inferior border of the mandible.

Sensory function

- The supra-ocular area is innervated by the ophthalmic nerve.
- The inter-oculo-buccal area is innervated by the maxillary nerve.
- The infrabuccal area is innervated by the mandibular nerve.

109
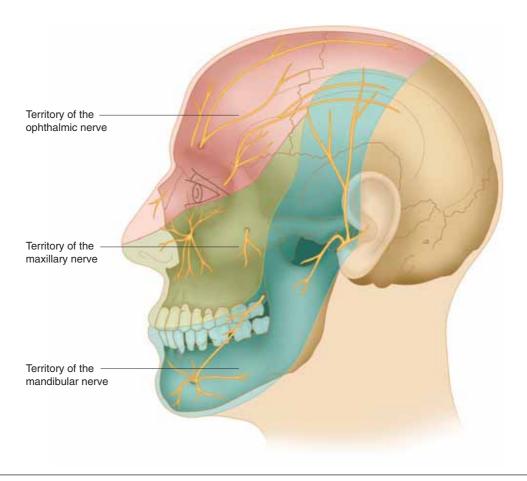
Fig. 14.4 Distribution of the trigeminal nerve.

Motor function

The trigeminal nerve is the motor nerve of the muscles of mastication.

14.2.2 Clinical notes

Multiple sclerosis

According to the French neurologists Doyon and Marsot-Dupuch (2002), 40% of patients suffering from multiple sclerosis have neuropathy of the trigeminal nerve; in 10% of cases this is the first symptom. Multiple sclerosis is difficult to diagnose in its early stages.

Neuralgia

Trigeminal or facial neuralgia can be induced or aggravated by palpating the point on the face where the nerves V1, V2 and V3 emerge.

Damage to the nerve trunk manifests as anesthesia or hypo-aesthesia of the nerve where it perforates the chin. Anesthesia or hypo-esthesia of the chin is always an indication of a problem somewhere along the course of V3.

Neuralgia of the face

Sometimes referred to as prosopalgia, neuralgias of the face exhibit the extreme intensity that is a hallmark of neural pain. Despite

being called "facial" neuralgias, these disorders are the province of the trigeminal nerve.

Its implication in these painful facial conditions and in migraines underlines the nociceptive importance of the trigeminal nerve.

Essential facial neuralgia

Essential facial neuralgia is also called malady of Trousseau. In medicine the term "essential" is used when the cause of an illness is unknown. However, we find this type of neuralgia is often related to mechanical nerve compression at the cranial orifice or canal.

Essential facial neuralgia is often triggered by a stimulus, even a light one, to the *trigger zones*, in particular those places where the trigeminal nerve emerges on the face. Sometimes laughter, chewing, grimacing, emotions, and so on are enough to set off the neuralgia.

It is typically of short duration, no more than 30 seconds. At the beginning, it is unilateral. The pain is sharp and intense, sometimes giving rise to *tic douloureux*. Pain does not persist once the crisis is past. There are no lasting sensory disturbances.

Secondary neuralgia

The characteristic signs of secondary neuralgias are as follows:

- Pain sometimes dies down by half between episodes.
- The sensitivity of the integuments is affected.
- Several branches of the trigeminal nerve are affected at the same time.

Neuralgia that occurs as the result of dental or auricular problems can be aggravated by pressure applied to the painful zones. Damage to the brain stem nucleus is seen with multiple sclerosis, glioma or metastasis, rhombencephalitis (herpes zoster), syringomyelocele or vascular pathologies (arteriovenous malformation, ischemic attack, dissection and cavernoma).

The absence of the corneal reflex is a sure sign of damage to the trigeminal nerve, the cornea being innervated by VI.

Unilateral paralysis does not involve functional disturbance due to the compensating action of the contralateral muscles.

The masseter reflex, contraction of the muscle when percussed, is absent in cases of paralysis.

14.3 MANIPULATION

14.3.1 Trigeminal ganglion

It is interesting to think that rotation of the head influences the mechanical tension of the trigeminal nerve (Breig 1978).

This mechanical action is confirmed clinically. In the condition known as *tic douloureux*, the simple act of turning the head can set off the tic. In effect, the trigeminal ganglion and its roots are being pulled upon when the head is in contralateral rotation. Tension on the dura mater increases nerve irritation.

Remember that the trigeminal ganglion and the roots of the trigeminal nerve are enveloped by the dura mater. Any fixation of the dural membrane can hamper the intraforaminal and intradural gliding of the trigeminal ganglion at its roots.

Technique

This technique plays upon the trigeminal ganglion, the trigeminal nerve roots and the dura mater of the trigeminal cave, as well as the recurrent branch of Arnold (V1) (Fig. 14.5).

Our goal is to influence these structures within the trigeminal cave, as well as the foramen ovale and the foramen rotundum.

The patient is supine, with the head resting in the palm of the practitioner's hand. The practitioner's other palm is placed on the patient's skull, anterior to the coronal suture, near the sagittal suture and focused on the direction of the trigeminal ganglion.

Step 1

Ask the patient to take a breath in during the cranial expansion phase and to hold it for a moment. When you sense that the

Fig. 14.5 Manipulation of the trigeminal ganglion.

intracranial pressure is at its maximum, gradually turn the patient's head to the side opposite the ganglion being treated. Return the head to the original position during the cranial retraction phase and while the patient exhales. Repeat the maneuver several times.

You can ask patients to rotate their head themselves while you bring the cervical spine into flexion to increase the intracranial pressure.

Step 2

Maintaining the head in rotation during the phases of cranial retraction and expiration, compress your anterior palm in the direction of the trigeminal ganglion.

Note: To have a more specific effect on the mandibular nerve, which is more vertical, add a component of cephalad axial traction during inspiration. This will direct the focus to the foramen ovale.

14.3.2 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- the dura mater: trigeminal cave, which is a doubling of the tentorium cerebelli
- temporal: the posterosuperior border of the petrous part of the temporal bone.

Indications

Indications for treatment of the trigeminal nerve are:

- anterolateral fixations of the intracranial dura mater, of either surgical or traumatic origin
- unilateral fixations of the tentorium cerebelli
- trigeminal neuralgia
- facial paralysis (because the trigeminal nerve forms an anastomosis with the facial nerve).

Contraindications

There are numerous contraindications. Care must be taken with the following conditions:

- arterial hypertension
- intracranial arterial pathologies
- intracranial hypertension.

14.4 TRIGEMINAL NERVE AND MIGRAINES

For a long time, migraines were thought to result entirely from cerebral vascular problems. The theory was that a phase of vasoconstriction provoked oligemia (a condition of reduced circulating intravenous volume), succeeded by a phase of vasodilatation (hyperemia, or increased blood flow). New PET scan research has led to the discovery of other more subtle contributing factors. The challenge is to understand why the vascular system is initially underactive and becomes subsequently overactive. We refer to the research done by Gilles Géraud, Chief of Neurology at the University Hospital of Toulouse-Rangueil (2003), as well as to our own clinical experience.

14.4.1 Implicating the nervous system

At the onset of a migraine attack, there is neuronal activation within the brain stem and the hypothalamus. Neurophysiologists term this activity cortical spreading depression or invasive cortical depression, occurring at the same time as migraine auras appear. It affects 20% of migraine sufferers. A migraine aura manifests visual symptoms like scintillating scotoma.

14.4.2 Cortical spreading depression

The massive transitory depolarization of visual cortex neurons generates an electrical signal. This impulse is the result of the extensive migration of sodium and potassium ions, spreading to the surface of the cortex at the speed of 2–4 mm per minute. Following this electrical discharge, the neurons cease all signal transmission and recuperate.

Gilles Géraud puts forward two hypotheses:

- The hypoperfusion could be the consequence of neuronal depression.
- The hypoperfusion could be due to the activation of vasoconstrictor neurons in the brain stem, the resulting oligemia triggering cortical spreading depression.

14.4.3 Trigeminal cervical system

The vessels of the cortex, the pia mater and the dura mater all receive their sensory fibers, for the most part, from the trigeminal ganglion. Fibers coming from other cranial nerves, from the medulla oblongata and the upper cervical region also contribute to this innervation. We call this arrangement the trigeminal-cervical system.

In rats, electrical stimulation of the trigeminal ganglion releases algogenic substances (neuropeptides, substance P and neurokinines A). These cause inflammation of the meningeal vessels, leading to vasodilatation and plasma leakage. Afterwards, a significant amount of serotonin is found in the blood, and this is a powerful factor in vasoconstriction.

14.4.4 Physiology of the migraine attack

Many factors (genetic, hormonal, digestive, sensory, psychological, climatic and electromagnetic) can overactivate the trigeminal ganglion. Vasodilatation of the intracranial arteries results, and this event is transmitted to the trigeminal neurons, which deliver an activating signal to the brain stem.

This activation implicates the peripheral nervous system, the thalamus and the cerebral cortex. The nociceptive circuits become hyperactive. Their stimulation threshold is lowered, such that any overly intense stimulus can bring on a migraine.

Note for manual therapists

In our view, the trigeminal nerve is one of the most important cranial nerves. It supplies sensitivity to the meninges, as well as to the meningeal and cortical arteries. Its role in migraine headaches is undeniable.

Neural manipulation of the trigeminal nerve has an immediate vascular effect. Some patients are instinctively aware of the trigger points for the trigeminal nerve. By massaging these points themselves, they are able to diminish the intensity of the migraine attack.

It is difficult to prove that our manipulations have an effect on electrical nerve conduction. In any case, experience shows us that mobilization of intraneural structures does not entirely explain our results.

We obtain our best results with migraines that begin in the occipito-cervical region and radiate forward. Remember that the suboccipital nerve (of Arnold) exchanges fibers with the frontal branch of the ophthalmic nerve, itself arising from the trigeminal ganglion. Generally, migraines are more complex to analyze and treat than headaches.

Ophthalmic nerve

15

15.1 ANATOMY

15.1.1 Origin

The ophthalmic nerve (V1) arises from the anteromedial part of the trigeminal ganglion (of Gasser).

15.1.2 Pathway

The ophthalmic nerve travels forward obliquely and superomedially towards the superior orbital fissure of the sphenoid (Fig. 15.1).

15.1.3 Useful relationships

The ophthalmic nerve runs below the trochlear nerve. It is joined by filaments of the carotid plexus and communicates with the oculomotor nerve.

15.1.4 Anastomoses

The ophthalmic nerve exchanges nerve fibers with the three motor nerves of the eye:

- the trochlear nerve (IV)
- the oculomotor nerve (III)
- the abducent nerve (VI).

15.1.5 Collateral branches

The ophthalmic nerve sends an important sensory branch to the tentorium cerebelli and to the posterior part of the falx. This is known as the *recurrent meningeal nerve of Arnold* (Fig. 15.2). This nerve allows us to have an effect upon the intracranial membranes.

15.1.6 Terminal branches

The terminal branches of the ophthalmic nerve are the nasociliary, frontal and lacrimal nerves (Fig. 15.3).

Nasociliary nerve

The nasociliary nerve passes through the superomedial aspect of the sphenoid fissure, within the common tendinous ring (annulus of Zinn). It runs towards the medial part of the orbital cavity to end at the medial anterior orbital foramen.

Collateral branches
The collateral branches of the nasociliary nerve are:

- the ophthalmic ganglion
- the ciliary nerves
- the spheno-ethmoidal branch (the posterior ethmoidal nerve), which supplies the sphenoidal and ethmoidal sinuses.

Terminal branches
The terminal branches of the nasociliary nerve (Fig. 15.4) are:

- The infratrochlear nerve (*external nasal*), a branch of which goes to the lacrimal canal and the medial eyelid. It can be palpated below the trochlea.
- The supratrochlear nerve. This branch is also called the ethmoidal filament for the cribriform plate, over which it passes.
- A branch supplies the frontal dura mater.
- Nasal branches.
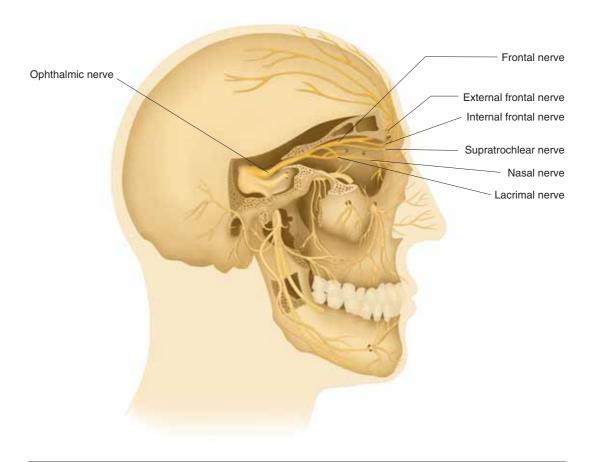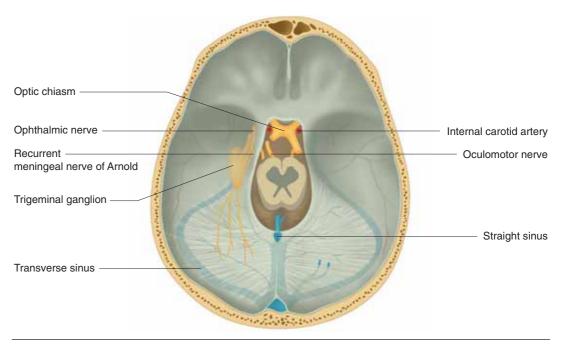
Fig. 15.1 Course of the ophthalmic nerve.

Fig. 15.2 Recurrent meningeal nerve of Arnold.

Frontal nerve
Lacrimal nerve
Nasociliary nerve
Ophthalmic nerve

A

Lacrimal nerve

Frontal nerve

Nasociliary nerve

B
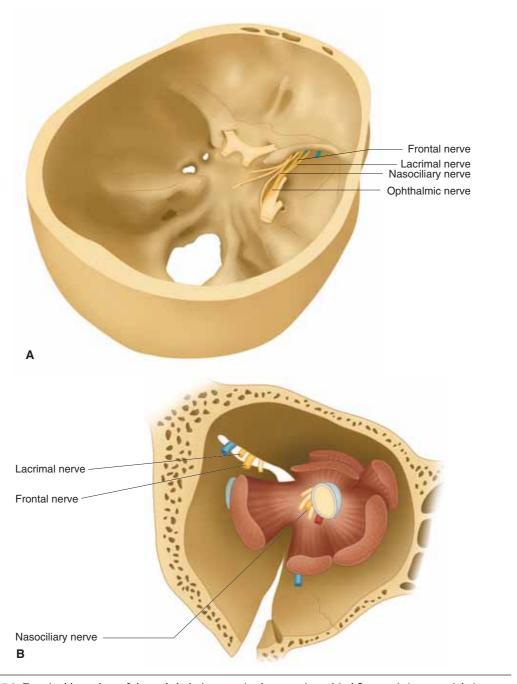
Fig. 15.3 Terminal branches of the ophthalmic nerve in the superior orbital fissure. A: Intracranial view. B: Frontal section.
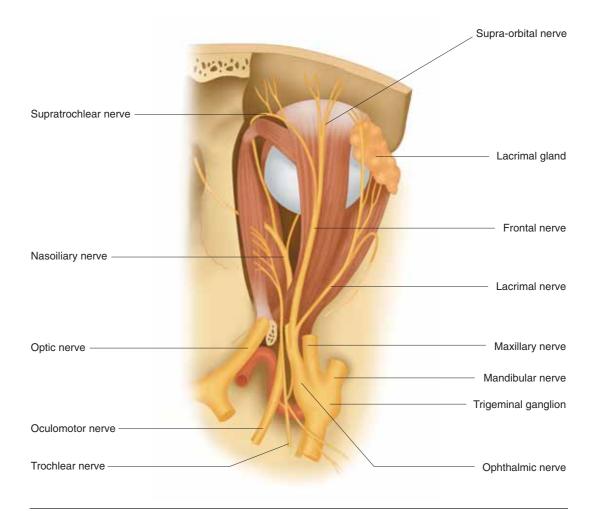
Fig. 15.4 Terminal branches of the ophthalmic nerve in the orbit.

Note for manual therapists

Through the branches of the nasociliary nerve, it is possible to have an effect on the ethmoidal and sphenoidal sinuses, the frontal dura mater and the lacrimal canal.

Frontal nerve

The frontal nerve enters the orbit through the superomedial aspect of the superior orbital fissure, without passing through the common tendinous ring.

Terminal branches

The frontal nerve divides into two branches:

- The supratrochlear nerve (internal frontal) runs towards the inner corner of the eye. It gives off filaments supplying the frontal periosteum, the skin of the lower part of the forehead, and the upper eyelid and its conjunctiva.
- The supra-orbital nerve (external frontal) passes through the supra-orbital notch, or the supra-orbital foramen. It innervates the upper eyelid, the conjunctiva and the skin of the forehead. Small branches perforate the occipitofrontalis muscle to supply the lambdoid suture, the diploë of the frontal bone and the mucosa of the frontal sinus, and join several cutaneous branches of the greater occipital nerve (C2) and the nerve of Arnold.

Note for manual therapists

Treatment of the frontal bone can affect the frontal sinus and the lambdoid suture. Anatomical treatises are sketchy on the subject of the innervation of the cranial sutures. We believe that the sutures derive their innervation largely from the trigeminal nerve.

The lambdoid sutures are impacted by backward falls, such as when the head hits the ground or a car headrest. Following this type of trauma, it is important to manipulate both the trigeminal ganglion and the frontal nerve.

In addition, treat the frontal branch of the supratrochlear nerve, which has anastomoses with branches of the nerve of Arnold. The nerve of Arnold has to be released in order to liberate the supratrochlear nerve.

Lacrimal nerve

The lacrimal nerve is the smallest of the main branches of the ophthalmic nerve. It enters the orbit through the narrowest part of the superior orbital fissure, to reach the lacrimal gland.

Anastomoses

The lacrimal nerve anastomoses with the trochlear nerve and the orbital branch of the maxillary nerve. Some of these terminal branches enter the lacrimal gland; others extend to the lateral aspect of the superior eyelid.

Note for manual therapists

The lacrimal nerve can be reached at the lacrimal gland and more easily at the level of the eyelid. By treating the frontal nerve and its branches, we can have an effect on the frontal, sphenoidal and ethmoidal sinuses, and also the frontal and occipital dura mater.

Ciliary ganglion

Formerly called the ophthalmic ganglion, the ciliary ganglion is located to the lateral side of the optic nerve (Fig. 15.5). It receives:

- sensory efferent branches of the nasociliary nerves
- motor branches issuing from the oculomotor nerve
- a sympathetic branch from the internal carotid plexus.

The efferent branches are made up of the ciliary nerves that supply filaments to the dural sheath of the optic nerve and to the ophthalmic artery and its branches, as well as to the eye and its constituents: the sclera, the choroid, the ciliary muscles, the iris and the cornea. The ciliary ganglion is the center from which impulses arise to modify intra-ocular pressure.

Note for manual therapists

By using the supratrochlear nerve (terminal branch of the nasociliary) as intermediary, we can hope to have an effect on the eye and its circulation.

15.2 MANIPULATION

Treatment of the frontal and nasal nerves affects the ophthalmic nerve.

15.2.1 Frontal nerve and suboccipital nerve of Arnold

As previously described, the supratrochlear nerve (branch of the frontal) exchanges nerve fibers with the suboccipital nerve of Arnold. These are small filaments that travel in the integumentary from the back of the head, over the top of skull, to join with several sensory fibers of the frontal nerve (Fig. 15.6).

These filaments supply the sensitivity of the scalp and, thanks to the frontal nerve, are connected to the frontal sinus and the diploë.

The frontal nerve is a terminal branch of the ophthalmic nerve. It is a recurrent nerve, called the recurrent nerve of Arnold, with distribution to the falx and the tentorium. Even though it is named after Arnold, it has
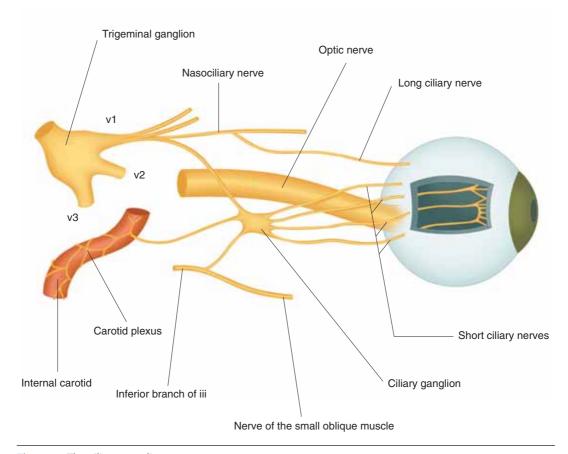
Fig. 15.5 The ciliary ganglion.

nothing to do with the suboccipital nerve of the same name.

We think that it is because of the anastomosis with the supra-orbital nerve that, in treating the Arnold nerve, we have a profound (and not just surface) effect on the intracranial membranes. Before any manipulation of the frontal and trigeminal nerves in general, it is advisable to release the Arnold nerve. We explained this technique in our book, *Manual Therapy for the Peripheral Nerves* (Barral and Croibier 2004), but will describe it again here.

Location of the suboccipital Arnold nerve

The suboccipital Arnold nerve emerges between C1 and C2, three finger widths below the external occipital protuberance and about one finger width lateral to the spinous process of the axis (Fig. 15.7).

Palpation of the suboccipital Arnold nerve

The patient is supine, with the cervical spine placed in very slight extension (Fig. 15.8). With your index finger, search for a small sensitive area, a *bud* of about 2 mm. The easiest way to explore this area is by gliding the finger along the cervical column towards the suboccipital region, meticulously palpating the spaces between the lamina.

Manipulation

Either the index finger pad can be placed directly on the sensitive bud of the nerve, or the middle finger of the opposite hand can be
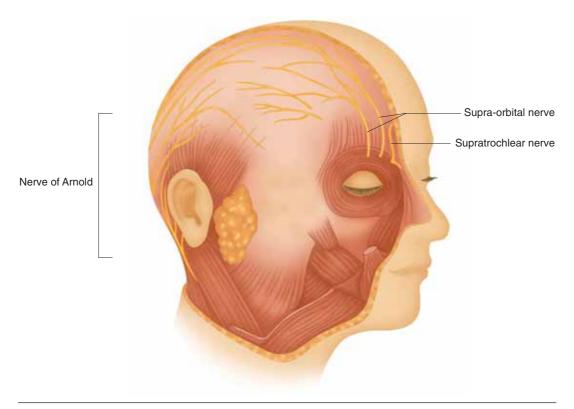
Fig. 15.6 Anastomoses of the frontal and Arnold nerves.

interposed between the bud and the index finger. Indirect contact helps to avoid irritating the nerve root. Gently compress the suboccipital nerve several times in order to wake up the proprioceptors and then treat with induction. In most cases induction will take the nerve caudad or cephalad (Figs 15.9 and 15.10).

Noteworthy area

Experience has shown us that it is around the coronal suture (the most important suture of the skull) that the majority of frontal and suboccipital nerve fixations are found. In this same region we find the nerve filaments that appear to give sensitivity to the coronal suture.

Dimples of the scalp

The fixations of these nerve filaments are often located on the areas of the integumen-

tary dimples. Typically, when we are palpating the skull, it feels like a rather uniform and smooth convexity. These dimples form a sort of miniature bowl whose center is very sensitive, and this is where the integuments adhere more closely.

These small indentations are most frequent near the coronal suture.

Dimple techniques

With the patient in the supine position, place the palm of your non-dominant hand under the occiput. With the thumb of the other hand search the area for dimples (Fig. 15.11). Make contact with the most sensitive part of the dimple, and mobilize using induction until the pain disappears.

15.2.2 Supra-orbital nerve

The supra-orbital nerve passes through the supra-orbital notch or the infra-orbital

121

Greater occipital
nerve

Trapezius muscle

Rectus capitis
posterior major

Vertebral artery

Suboccipital
nerve of Arnold

Rectus capitis
superior minor

Obliquus capitis superior

Obliquus capitis inferior

Cervical plexus
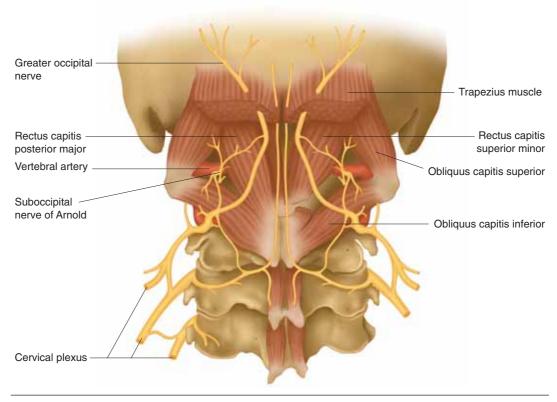
Fig. 15.7 Location of the suboccipital nerve of Arnold.

Fig. 15.8 Palpation of sub-occipital nerve of Arnold.
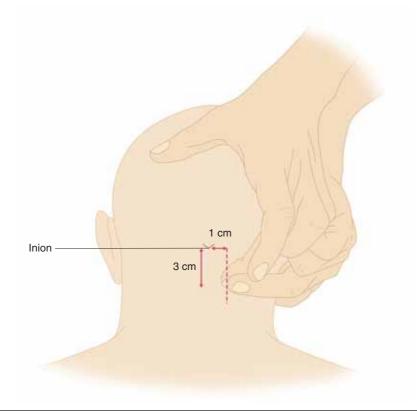
Fig. 15.9 Finger contact for manipulation of the suboccipital nerve of Arnold.

Fig. 15.10 Manipulation of the suboccipital nerve of Arnold.

Fig. 15.11 Manipulation of the dimples of the scalp.

foramen, which is found about 2–3 cm from the vertical median line that divides the face in two (Fig. 15.12).

Glide the index finger in a clockwise and then a counterclockwise direction along the orbital rim until you feel a sensitive puncti-form spot, which is sometimes a little hard-ened. Always compare both sides. These tests evaluate the nerve's consistency and sensitivity, and thereby tell you whether or not this cranial nerve should be manipulated.

Induction technique

Induction technique consists of following the direction the nerve takes and encouraging this movement until the pain disappears and the nerve is no longer hardened. When the supra-orbital nerve merits treatment, you will feel a small sensitive bud against the superior orbital wall.

Cutaneous technique

Taking the skin near the supra-orbital notch between your thumb and index fingers, gently roll the cutaneous and subcutaneous tissues between your fingers, Ask your patients to massage themselves in this way on the days following their appointment.

Locating the ophthalmic nerve branches

The superior medial aspect of the orbital rim is especially rich in nerve filaments. It is quite simple to find the various branches.

The *lateral frontal nerve* leaves the skull accompanied by the artery of the same name, through the supra-orbital notch, situated 3 cm from the median line.

The *medial frontal nerve* exits the supra-orbital notch, together with the medial vessels.

At the junction of the medial and superior edges of the orbital crest, it is easy to palpate the trochlea (pulley of the superior oblique muscle):

- Feel for the fibers of the supratrochlear nerve above the trochlea.
- Fibers of the infratrochlear nerve are found below the trochlea.

15.2.3 Supratrochlear and infratrochlear nerves

Remember that the trochlea is the reflexion pulley for the large superior oblique muscle. The supratrochlear and infratrochlear nerves are found on either side of the trochlea (Fig. 15.13).
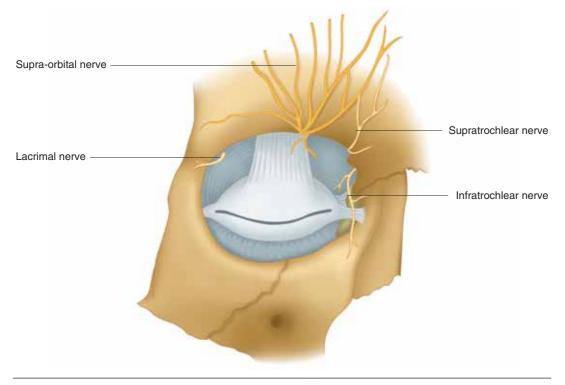
Fig. 15.12 The supra-orbital nerve.
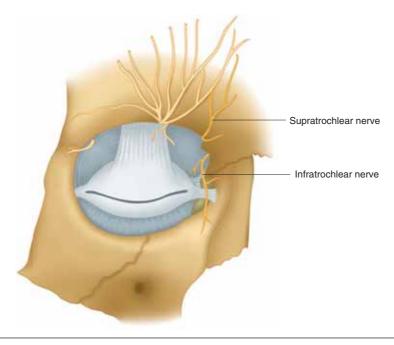
Fig. 15.13 The supratrochlear and infratrochlear nerves.

Following the same protocol as for the supra-orbital nerve, glide your index finger over the medial orbital rim to search for sensitive points and treat them with induction.

15.2.4 Lacrimal nerve

This nerve is a little more difficult to locate and to treat. It is situated superolaterally in the orbit (Fig. 15.14). It innervates the skin of the outer corner of the eye.

Roll the skin between your fingers as you search for small, hardened, sensitive nerve filaments suitable for induction.

15.2.5 Upper eyelid and orbital septum

The upper eyelid is innervated by the supra-orbital, supratrochlear and infratrochlear, and lacrimal nerves and a branch of the maxillary nerve. The orbital septum is a membranous sheath separating the superficial orbital structures from those of the orbit itself.

Technique

Taking the eyelid and the septum between your thumb and index finger, glide the structures between your fingers (Fig. 15.15).

Look for sensitive areas, accompanied by firm nerve fibers. Treat with induction.

15.2.6 Indications

The ophthalmic nerve and its terminal branches are manipulated in cases of:

- headache
- migraine
- craniofacial trauma
- sinusitis
- sutural fixations
- intracranial membranous fixations
- plagiocephaly
- facial paralysis (anastomosis with the facial nerve)
- snoring
- permeability problems of the lacrimal canal.

Fig. 15.14 The lacrimal nerve.
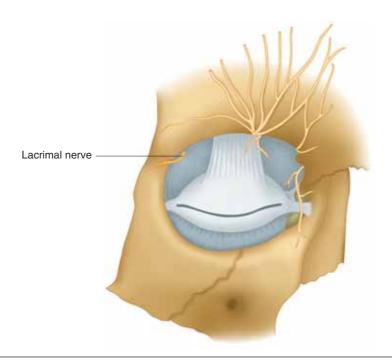
Fig. 15.15 Manipulation of the palpebral branches.

15.2.7 Remarks

By treating the suboccipital nerve of Arnold and the frontal nerves as intermediaries, it is possible to release sutural and cervical fixations. While the anastomoses of cranial nerves are multiple and certainly have not yet all been cataloged, they are well known among manual therapists and anatomists.

By treating the nerves, one can affect the organs. People suffering from biliary dysfunction of the liver or gall bladder have been heard to say, "My hair hurts today." This type of scalp pain is theoretically due to the sensory fibers coming from the suboccipital Arnold and frontal nerves.

It is both difficult and too simple to find a logical explanation for such symptoms. Everyone can find a connection: facial, neural, fluid or emotional. We base our conclusions on what we observe on a daily basis in our clinical work. The principle of neurovisceral homolaterality is not entirely valid at the onset of a gall bladder attack. At first it is the left side that is primarily irritated, then the right, and finally both sides are involved.

Note: Teach your patients to treat themselves. They can massage the skin where the nerve emerges on the orbit. This self-help is particularly valuable for people who suffer from migraine, headache and sinusitis.

15.2.8 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- the dura mater (falx cerebri and tentorium cerebelli)
- the osseous orbit: frontal, sphenoid, maxilla, zygoma, palatine, lacrimal and ethmoid bones
- the superior orbital fissure.

15.2.9 Combined maneuvers

With the cervical nerves

We can combine the Arnold nerve technique with treatment of the frontal nerve filaments in the dimples of the scalp and with treatment of the supra-orbital nerve (Fig. 15.16) at the orbit. This is a strongly recommended method.
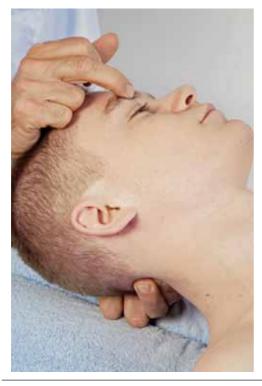
Fig. 15.16 Combined manipulation of the ophthalmic and cervical nerves.

As a general rule, when the supra-orbital nerve is sensitive, you will also find the infra-orbital and mental nerve branches to be sensitive, as they appear on the face. Integrate their treatment with the above points.

With the optic nerve

The ophthalmic nerve follows approximately the same direction as the optic nerve. All the techniques that address the optic nerve have an effect on the ophthalmic nerve. Combine treatment of the two nerves for greater effectiveness.

Maxillary nerve

16

16.1 ANATOMY

16.1.1 Origin

The maxillary nerve (V2) begins at the middle of the trigeminal ganglion and is intermediate between the ophthalmic and mandibular nerves.

16.1.2 Pathway

The maxillary nerve leaves the skull through the foramen rotundum and arrives in the superior part of the pterygopalatine fossa (Fig. 16.1). It enters the inferior orbital fissure, crosses the infra-orbital groove, and appears on the face through the infra-orbital foramen. At its origin it is surrounded by a double layer of dura mater.

16.1.3 Collateral branches

The maxillary nerve has the following collateral branches (Fig. 16.2):

- cranial branch: the middle meningeal branch
- four exocranial branches:
  — orbital branch
  — branches of the sphenopalatine ganglion
  — posterior alveolar (dental) branches
  — anterior superior alveolar (dental) branches.

Middle meningeal branch (nervus meningeus medius)

The middle meningeal branch supplies dura mater neighboring the petrous temporal bone, the sphenoid and the orbit.

Orbital branches

Lacrimal palpebral branch
The lacrimal branch anastomoses with the ophthalmic nerve. The palpebral portion goes to the upper eyelid to innervate it, along with the supra-orbital, supratrochlear, infratrochlear and lacrimal nerves.

Zygomaticotemporal branch
The zygomaticotemporal branch penetrates the zygomaticotemporal foramen and divides into a zygomatic branch, palpable on the external surface of the cheekbone, and a temporal filament.

Pterygopalatine branches

The pterygopalatine ganglion is suspended from the maxillary nerve, which contributes fibers to it. It is found in the pterygopalatine fossa. We will look more closely at this ganglion later.
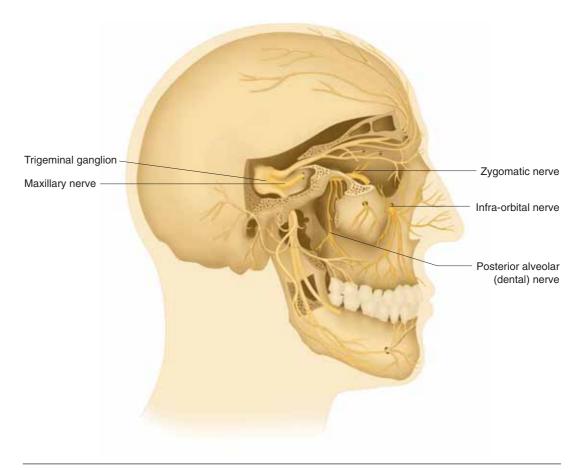
Fig. 16.1 Trajectory of the maxillary nerve.

Posterior superior alveolar (dental) branches

The posterior dental branches arise from the maxillary nerve just in front of the infra-orbital groove (Fig. 16.3).

These branches supply the molar teeth, before issuing branches to the upper gum and the adjoining part of the cheek. There are nerve fibers for:

- the molar and premolar teeth
- the alveoli
- the maxillary sinus
- the maxilla bone.

Anterior superior alveolar (dental) branches

The anterior dental branch originates about 1 cm behind the infra-orbital foramen. It follows a minute osseous canal bringing filaments for:

- the nasal mucous membrane
- the incisor and canine teeth
- the maxilla.

16.1.4 Termination

The maxillary nerve terminates as the infra-orbital nerve. Testut (1896) calls this branch

Right orbit

Infra-orbital "bouquet"

Anterior dental nerve

Middle dental nerve

Lacrimal gland

Temporo-zygomatic nerve

Vomer

Nasopalatine nerve

Anastomosis with the lacrimal nerve

Orbital floor

External spheno-palatine nerve

Infra-orbital nerve

Anterior palatine nerve

Orbital branch

Maxillary sinus

Middle palatine nerve

Posterior alveolar (dental) nerve

Posterior palatine nerve

Internal maxillary artery

Inferior nasal nerve

Right nasal fossa

Orbital branch

Maxillary nerve

Pterygopalatine nerve

Pterygoid process

Pterygopalatine ganglion

Vidian nerve

Fig. 16.2 Collateral branches of the maxillary nerve.

of the nerve the "infra-orbital bouquet", as it appears on the face. The infra-orbital foramen, through which it exits, is located 5 mm from the caudal orbital rim and 3 cm from the median axis.

The infra-orbital nerve supplies nerve fibers for:

- the eyelids
- the upper lips
- the nose.

Note for manual therapists

The maxillary nerve can be palpated at:

- the lower eyelid
- the cheekbone
- the upper lip
- the infra-orbital foramen.
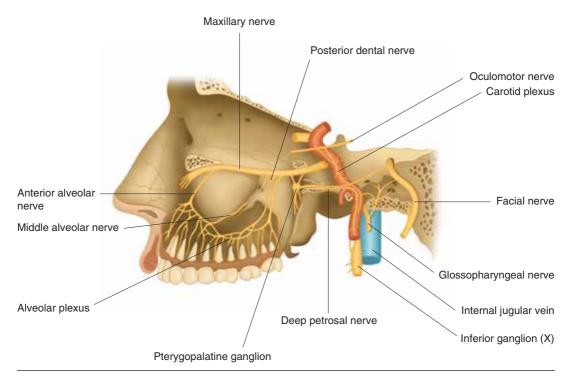
Fig. 16.3 Dental branches.

16.1.5 Pterygopalatine ganglion

Formerly known as the sphenopalatine ganglion, the pterygopalatine ganglion is the size of a lentil. It lies in the pterygopalatine fossa (Fig. 16.4).

Afferent branches

The pterygoid ganglion receives fibers from the maxillary nerve. In addition, it is partly formed by three other roots that unite in a nerve called the vidian nerve.

Vidian nerve
The vidian nerve joins the posterior part of the pterygopalatine ganglion. It runs through the vidian (pterygoid) canal, from which it takes its name, and forms a carotid branch and a cranial branch.

The cranial branch is composed of:

- the greater petrosal nerve, which comes from the facial nerve and runs through the hiatus on the anterior surface of the petrous portion of the temporal bone

- a communicating branch with the greater petrosal nerve, which comes from the tympanic cavity, having originated at the glossopharyngeal nerve.

> **Note for manual therapists**
>
> Via the intermediary of the pterygoid ganglion, it is possible to have an effect on the facial nerve, the glossopharyngeal nerve and the sympathetic carotid plexus.

Efferent branches

The efferent branches of the pterygoid ganglion are:

- the pharyngeal and nasal branch
- the orbital branch
- the pterygopalatine nerve.

We will describe the pterygopalatine and the palatine nerves, which are the most interesting for us because they are accessible to palpation.
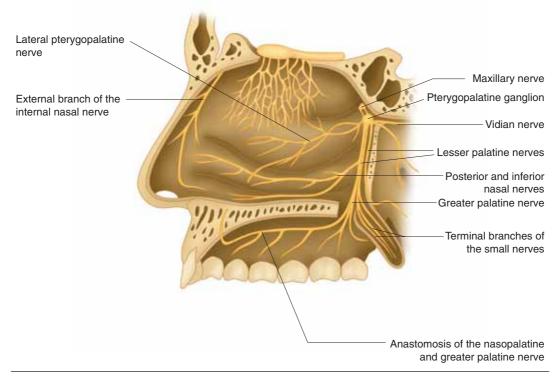
Fig. 16.4 Pterygopalatine nerves.

Pterygopalatine nerve

The pterygopalatine nerve arises from the medial part of the pterygopalatine ganglion, runs in the nasal fossa and crosses the sphenopalatine foramen.

It divides into two branches:

- the lateral pterygopalatine nerve, which goes to the nasal fossa
- the nasopalatine nerve, which runs inferiorly and anteriorly on the posterior nasal septum, then passes through the palatine canal to reach the palatine arch.

Palatine nerves

The palatine nerves originate from the caudad part of the pterygopalatine ganglion and rejoin caudally in the osseous canals of the palatine arch (Fig. 16.5).

Greater palatine nerve

Formerly known as the anterior palatine, this is the largest of the palatine nerves. It supplies fibers to the soft palate, the gums, the palatine arch, the maxillary sinus and the nasal fossa. It anastomoses with the nasopalatine nerve.

Lesser palatine nerve

Formerly designated as the middle and posterior palatine nerves, this nerve is made up of two slender branches that reach the hard palate through the two posterior osseous conduits. They supply sensory filaments to the soft palate and motor fibers to the internal peristaphylin and palatostaphylin muscles of the uvula.

Note for manual therapists

The maxillary nerve can be palpated:

- where it emerges from the zygomatic canal
- on the arch of the palatine, near the molar and incisor teeth
- at the infra-orbital foramen; several nerve fibers can be palpated here
- on the internal surface of the cheek.
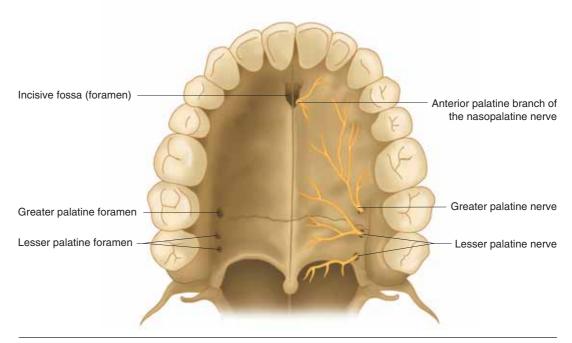
Fig. 16.5 Palatine nerves.

16.1.6 Functions of the maxillary nerve

The sensory fibers of the maxillary nerve go to:

- the cheek
- the inferior eyelid
- the ala of the nose
- the upper lip
- the posterior inferior nasal fossa
- the soft palate
- the teeth and gums of the maxilla.

Together with the facial and glossopharyngeal nerves, as well as their relays with the sympathetic system (pterygopalatine ganglion), the maxillary nerve provides the secretory innervation of the lacrimal and nasal glands, and vasomotor innervation to the nasal fossae.

16.2 MANIPULATION

We begin with the infra-orbital "bouquet", which, owing to its many terminal branches, allows us to have an effect on numerous intracranial and extracranial structures.

16.2.1 Infra-orbital "bouquet"

The infra-orbital foramen is located above the zygomatic process of the maxilla, about 2–3 cm from the median vertical line of the face. This orifice is more important than the supra-orbital foramen (Fig. 16.6).

The infra-orbital technique is performed externally on the face; and some fibers are accessed intra-orally on the interior of the cheek.

Location of the infra-orbital foramen

This foramen is situated 5 mm from the inferior rim of the orbit, on the same vertical line as the supra-orbital foramen. Frequently sensitive, it is located at the medial two-thirds of the inferior border of the orbit, about 3 cm from the median line.

Its cephalad border is distinct, concave caudally and medially. Its caudal border is less well defined and extends by a groove towards the canine fossa. The maxillary nerve opens out into terminal branches (the "bouquet") on a plane overlying that of the canine muscle.
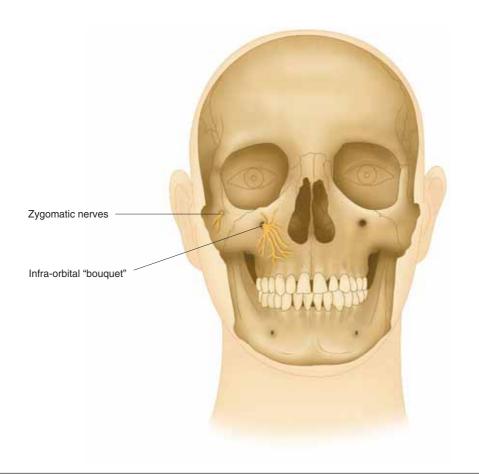
Fig. 16.6 The infra-orbital "bouquet".

External technique

With the patient supine, position yourself either directly behind or to the side of the patient (Fig. 16.7).

Locate the inferior orbital rim and delicately push the soft tissue caudad.

First explore the circumference of the orbital foramen to feel for a small ring of connective tissue. Using induction, aim to release the most hardened parts of this ring. In the infra-orbital foramen feel for a bud of 2–3 mm, and treat it with induction.

To determine which nerve fibers to treat, do a compression/decompression listening test.

As with all orifices, it is important in this case to feel for the play of the fibers just as they emerge from the infra-orbital foramen. You must have the impression that they are able to move freely: to move in and out of the orifice unhindered.

Treat the infra-orbital bouquet with induction. Initially the movement will be quite sagittal, becoming progressively more oblique. Always compare both sides.

Cutaneous technique

Gently grasp the skin between your thumb and forefinger feeling for the infra-orbital filaments. Massage by rolling the cutaneous tissue over the sensitive zones. Ask patients to perform this technique themselves, gently rolling the tissues until the pain vanishes.

135
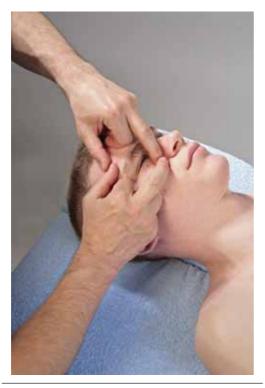
Fig. 16.7 External manipulation of the infra-orbital bouquet.

Internal technique

For the internal technique, place the thumb of one hand on the internal surface of the cheek, and the index finger or thumb of the other hand on the face, angled towards the infra-orbital foramen (Fig. 16.8).

Carrying out double listening with induction, attempt to bring your internal and external fingers as close together as possible.

16.2.2 Foramen in the zygomatic bone

The zygomatic foramen provides access to the zygomaticotemporal nerve. Located on the zygoma at the lateral inferior border of the orbit, this nerve branch is often sensitive. It is much smaller than the infra-orbital bouquet. Treat with induction; as with all the orifices, it is important to check both sides.

16.2.3 Eyelid

The lower eyelid enables us to release the lacrimapalpebral nerve. Gently roll the eyelid between your thumb and index finger. Lightly

Fig. 16.8 Internal manipulation of the infra-orbital bouquet.

Fig. 16.9 Manipulation of the eyelid.

compress your fingers at the sensitive areas and, using induction, encourage the tissues to glide (Fig. 16.9).

This technique, which we also apply to the ophthalmic branches, addresses the supratrochlear and infratrochlear nerves, as well as the lacrimal nerve.

16.2.4 Nasopalatine nerve

The palatine processes of the maxilla form the bony palate. Anterior to the palatine suture is found the incisive fossa, where the nasopalatine nerve is located.

Place your index finger at the junction of both incisive bones, about 1 cm behind the teeth. Look for a sensitive point and treat with compression–induction.

This manipulation is not easy to do.

16.2.5 Palatine nerves

The greater palatine foramen provides a passage for the greater palatine nerve. The lesser palatine nerves pass through the lesser palatine foramina, which are situated just behind the greater palatine foramen (see Fig. 16.5 above).

It is often difficult to differentiate between these foramina, which are very close together.

The listening test will direct you to the nerve to treat.

The patient is supine, hands placed on the abdomen. Explain that you will be placing your finger on the upper palate, and that there is no risk of suffocation. Sometimes patients have a bad memory of an unpleasant throat examination from childhood.

Ask the patient to open his or her mouth and glide your finger directly against the palate (Fig. 16.10). Ask the patient to bite down lightly on your finger a few times in order to release the tension in the muscles of the jaw.

Follow the medial alveolar border as far back as the molars. You will feel a sensitive zone, the principal palatine foramen. Compress and decompress the fibers several times and treat them with viscosity–induction until the pain disappears from the hardened area.

This technique achieves better results than treatment of the nasopalatine nerve.

The incisive foramen is located on the anterior part of the palatine arch. It delivers the right and left nasopalatine nerves. It is possible to locate it visually by asking patients to open their mouth. The canal is situated at the center of the star formed by the folds in the palatine mucosa, just behind the central incisors.
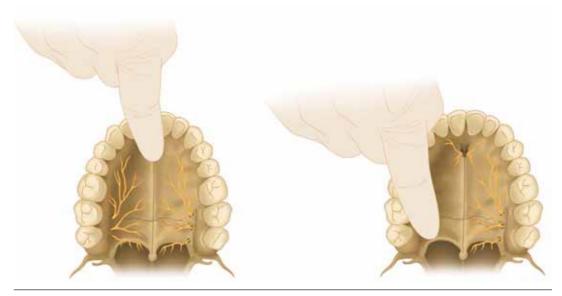
Fig. 16.10 Manipulation of the palatine nerves.

16.2.6 Indications

Indications for manipulation of the maxillary nerve are as follows:

- fixation of the dura mater of the middle cranial fossa
- craniofacial trauma
- gum and dental problems
- allergy
- facial paralysis
- sinusitis (maxillary sinus)
- snoring, sleep apnea
- following maxillofacial surgery.

16.2.7 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- the dura mater
- the maxilla, palatine, sphenoid (greater wing), zygoma and temporal bones
- the inferior orbital fissure.

Be careful when working with problems relating to upper dentures and maxillary sinusitis.

16.2.8 Combined technique

It is possible to combine manipulations of the supra-orbital, infra-orbital and mental foramens.

Mandibular nerve

17

17.1 ANATOMY

17.1.1 Origin

The mandibular nerve (V3) proceeds from the most lateral part of the trigeminal ganglion. It is the most vertical of the trigeminal nerve branches.

The mandibular nerve is made up of two roots: a sensory root and a small motor root.

17.1.2 Pathway

The two roots of the mandibular nerve are sheathed in a doubling of dura mater. Thus coupled, they pass through the foramen ovale, separating once they emerge (Fig. 17.1).

17.1.3 Collateral branches

The collateral branches of the mandibular nerve are:

- the deep temporal nerves
- the masseteric nerve
- the buccal nerve
- the lateral and medial pterygoid nerves
- the meningeal branch
- the auriculotemporal nerve.

Deep temporal nerves

From the foramen ovale, the deep temporal nerves pass through the zygomatic fossa, then through the temporal fossa to enter the deep surface of the temporal muscle.

Masseteric nerve

From the zygomatic fossa, the masseter nerve crosses the mandibular incisor to terminate in the masseter muscle and the temporomandibular joint.

Its anastomoses are as follows:

- the middle temporal nerve
- a sensory filament to the temporomandibular joint
- the posterior deep temporal nerve supplying the temporal muscle.

Buccal nerve

The buccal nerve passes forward between the coronoid process of the mandible and the tuberosity of the maxilla to supply the buccinator muscle.

It supplies:

- a branch to the lateral pterygoid muscle
- a filament to the anterior deep temporal nerve for the temporalis muscle.

The terminal branches supply the cheeks and the mucosa lining their inner surface.

Lateral and medial pterygoid nerves

These nerves pass through the otic ganglion and innervate the lateral and medial pterygoid muscles. The medial pterygoid nerve supplies the tensor veli palatini and the tensor tympani muscles.

Trigeminal ganglion

Mandibular nerve

Temporal nerve

Auriculotemporal nerve

Temporobuccal nerve

Masseteric nerve

Inferior (alveolar) dental nerve

Lingual nerve

Mental nerve
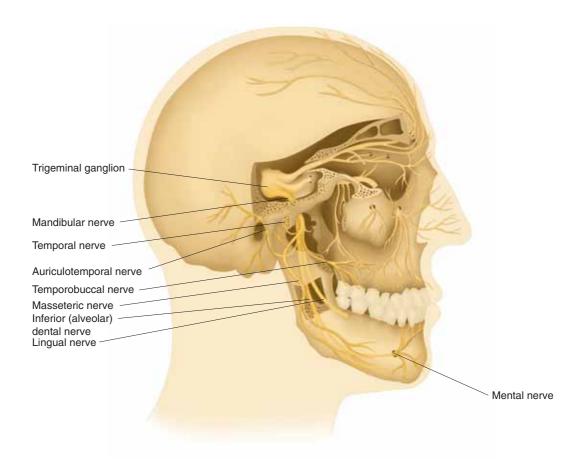
Fig. 17.1 Course of the mandibular nerve.

Meningeal branch

The meningeal branch is the first collateral of the mandibular branch to emerge immediately beyond the foramen ovale. It enters the skull through the foramen spinosum with the middle meningeal artery to innervate the dura mater of the middle cranial fossa.

Auriculotemporal nerve

The auriculotemporal nerve runs behind and laterally towards the neck of the mandibular condyle, which it goes around to pass between the articular (zygomatic) tubercle and the external acoustic meatus, terminating in the temporal region.

It supplies:

- the nerve of the external acoustic meatus
- the anterior auricular nerves for the tragus and the auricle
- a branch to the tympanic membrane
- the parotid branches.

It forms an anastomosis via a communicating branch with the facial nerve (temporofacial branch) to supply the parotid branches.

17.1.4 **Terminal branches**

The mandibular nerve has two terminal branches: the inferior alveolar (dental) nerve and the lingual nerve.

Inferior alveolar (dental) nerve

The inferior dental nerve is the largest terminal branch of the mandibular nerve. It travels

in the mandibular canal as far as the mental foramen.

Along its pathway the inferior dental nerve gives off collaterals: the mylohyoid and the dental branches (for the molars, the gums and the bone).

The terminal branches are: the incisive nerve (supplying the canine and the two incisor teeth) and the mental nerve.

The inferior dental nerve emerges from the mental foramen in a bouquet of nerve fibers which supply the skin of the chin and the lower lip, as well as the labial mucosa. This bouquet is less important than its infra-orbital counterpart.

Lingual nerve

The lingual nerve runs to the tip of the tongue, and has four anastomoses:

- the inferior dental nerve
- the chorda tympani (from the facial nerve)
- the hypoglossal nerve
- the mylohyoid nerve.

The lingual nerve sends filaments to the tongue, the soft palate, the gums, the floor of the mouth and the submaxillary and sublingual ganglions.

17.1.5 Sympathetic ganglions

The sympathetic ganglions are the submaxillary and the otic ganglions (Fig. 17.2).

Submaxillary ganglion

The submaxillary ganglion receives fibers from the lingual nerve and from its anastomosis with the chorda tympani. The ganglion lies on the hyoglossal muscle.

Otic ganglion

The otic ganglion is found at the medial part of the mandibular nerve, just below the foramen ovale.
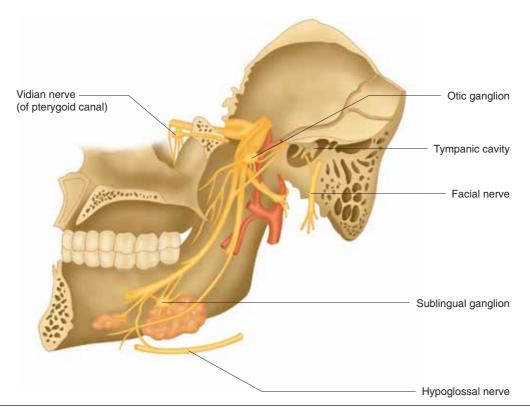
Fig. 17.2 Ganglions annexed to the mandibular nerve.

It receives:

- branches of the maxillary nerve
- a motor root, formed by a communicating branch of the lesser petrosal nerve, a branch of the facial nerve
- a sensory root, the lesser petrosal nerve of the tympanic, a branch of the glossopharyngeal nerve
- a sensory root supplied by the sympathetic plexus surrounding the middle meningeal artery.

It supplies:

- a motor branch for the medial pterygoid muscles and the external peristaphylin muscle of the malleus
- sensory filaments for the auriculotemporal nerve and the tympanic cavity.

17.2 MANIPULATION

17.2.1 External techniques

For the two nerve roots

Refer to the technique for manipulation of the trigeminal ganglion (Ch. 14). As previ-

ously noted, the mandibular nerve roots are located on the most lateral aspect of the trigeminal ganglion. Accordingly, they are placed under most tension during the treatment phase of cervical rotation, accompanied by inspiration and a little vertical axial traction.

Auriculotemporal nerve

The auriculotemporal nerve is extremely important in manipulation (Fig. 17.4). Remember it provides the innervation for:

- the angle of the mandible
- the concha of the auricle
- the external acoustic meatus
- the tympanic membrane
- the posterior temporal region.

This nerve can be manipulated in the temporal region, just in front of the ear, in the external acoustic meatus, the auricle and the tragus of the ear.

Temporal region

The auriculomotor nerve is superficial. Glide your finger in the subcutaneous tissue of the temporal region, searching for small, hardened filaments, and treat them with induction.

Note for manual therapists

- **The foramen ovale**. To affect the mandibular nerve, the technique that we have described for the trigeminal ganglion can be employed. As the mandibular nerve passes laterally through the foramen ovale, the movement of rotation of the head will have an effect on the foramen. Owing to the verticality of the mandibular nerve root, the trigeminal technique would be carried out in gentle axial traction.
- **The auriculotemporal nerve**. The auricular nerve can be accessed through the temporalis muscle. The nerve fibers can be located in the posterior superficial aspect of the muscle.
- **The ear**. The auriculotemporal nerve fibers can be reached at the external auditory canal, the tragus and the anterior part of the auricle.
- **The mandible**. The mandibular foramen is located on the medial surface of the

mandible. It is the external opening of the inferior dental canal. It is partly covered by the spine of Spix. Here the inferior dental nerve is found, which is also accessible at the mental foramen.
- **The mylohyoid muscle**. The lingual nerve lies against the deep surface of the mandible on the medial side of the mylohyoid muscle. The lingual nerve is connected to the sympathetic submandibular ganglion. The hypoglossal nerve can be accessed just posterior to the lingual nerve. The two are easily confused with each other.
- **The mental foramen**. The mental foramen, through which the inferior dental nerve emerges, opens on the external surface of the mandible below the second premolar (Fig. 17.3).
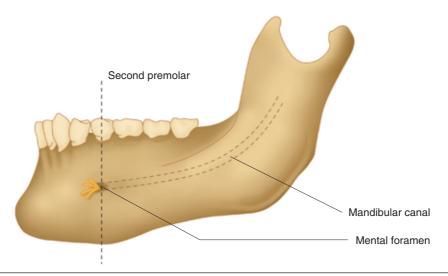
Fig. 17.3 Mental foramen.
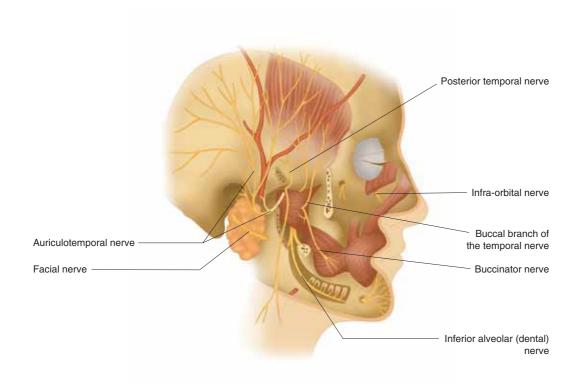
Fig. 17.4 Auriculotemporal nerve.

Search for sensitive filaments going to the superficial temporal artery by feeling for the arterial pulse. This nerve is easily perceived in the temporal region, close to the front of the ear.

Ear

Manipulations for the ear are described in Chapter 25. Sensory fibers of the auriculotemporal nerve are located mainly on the tragus and the ascending part of the helix.

External acoustic meatus

This canal is innervated by the auriculotemporal nerves, the auricular branch of the cervical plexus, and the vagus nerve issuing from the tympanomastoid fissure.

It is practically impossible to distinguish these nerves with your finger. It is the topography that allows them to be differentiated. For the most part, the auriculotemporal nerve, together with the facial, innervates the anterocephalic region of the external acoustic meatus.

Mylohyoid branch

The mylohyoid branch is located in the submandibular lodge. It is formed by:

- the body of the mandible
- the anterior belly of the digastric muscle
- the mandibular band.

Search in the mylohyoid muscle by gliding your finger along the caudal border of the mandible. You will feel a space into which your finger easily sinks. The nerve is sensitive here. By progressing a little caudad and posterior, you will come to the mandibular ganglion and behind it lies the hypoglossal nerve.

Manipulation is done with a light touch in induction (Fig. 17.5).

Mental foramen

The mental foramen is found 2–3 cm lateral to the median vertical line.

Compare both sides. The aim of the technique is to act on the visco-elasticity of the orifice and to treat the external fibers with induction. It is also possible to treat the sensitive cutaneous filaments by applying the skin-rolling massage technique.

> **Locating the mental foramen**
>
> The mental foramen is located 3 cm from the median line, at the height of the first premolar tooth (see Fig. 17.3 above).
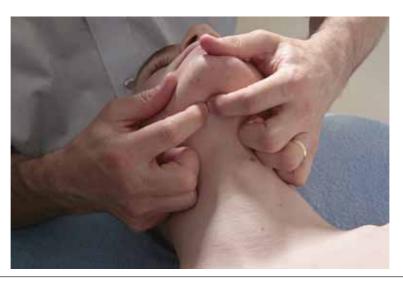
Fig. 17.5 Manipulation of the mylohyoid branch.

Locating the mandibular foramen

The mandibular foramen is located between the anterior and posterior borders of the ascending branch of the mandible (Fig. 17.6):

- 4 cm above the inferior border of the ramus
- 1 cm above the molars.

Inferior dental (alveolar) nerve

Mylohyoid nerve

1 cm

4 cm

Mandibular foramen

Incisive nerve
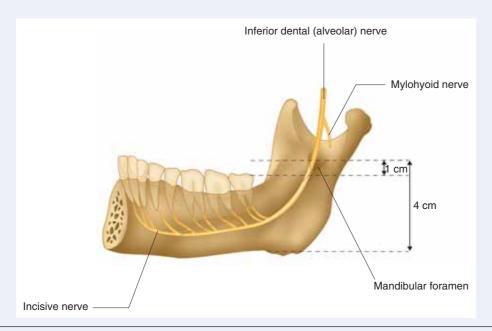
Fig. 17.6 Mandibular foramen (spine of Spix).

17.2.2 Internal technique

Run your finger over the medial surface of the ascending branch of the mandible until you feel the small osseous plate of the spine of Spix. This manipulation of the inferior dental nerve is not pleasant for the patient and does not yield as good results as the technique for the mental foramen. It is nevertheless important for a full understanding the mandibular foramen.

17.2.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- the dura mater of the middle cranial fossa
- the sphenoid
- the lateral pterygoid muscle
- the temporomandibular joint.

17.2.4 Combined techniques

With the supra-orbital and infra-orbital nerves

The supra-orbital and infra-orbital nerves are on the same line as the mental nerve. Treat them in pairs when working with the orifices of the face (Fig. 17.7).

With the facial nerve

It is particularly effective to treat the mandibular nerve in combination with the

Fig. 17.7 Combined manipulation of the mandibular nerve with the supra-orbital and infra-orbital nerves.

posterior auricular branch of the facial nerve and its various filaments on the face.

With the superficial cervical plexus

Manipulation of the mandibular nerve should be combined with the auricular branch of the superficial cervical plexus, which forms anastomoses with the trigeminal and facial nerves.

All of these combined maneuvers are carried out during the cranial expansion phase.

17.2.5 Indications

Indications for manipulation of the mandibular nerve are as follows:

- sinusitis (maxillary)
- gum problems
- facial paralysis (anastomosis with VII)
- craniofacial trauma
- maxillofacial surgery.

Abducent nerve

The abducent nerve (VI, motor nerve of the lateral eye) is a small cranial nerve supplying one extra-ocular muscle, the lateral rectus muscle of the eyeball.

18.1 ANATOMY

18.1.1 Origin

The abducent nerve emerges from the anterior surface of the brain stem in the pontine cistern above the pyramid of the medulla oblongata, medial to the facial nerve (VII).

18.1.2 Pathway

From the brain stem it runs towards the quadrilateral plate of the sphenoid. It pierces the dura mater and enters the cavernous sinus, coursing through it to reach the superior orbital fissure (Fig. 18.1).

18.1.3 Useful relationships

- In the cavernous sinus the abducent nerve is situated between the ophthalmic nerve, which is lateral, and the internal carotid artery, which is medial.
- In the superior orbital fissure the nerve passes through the largest part of the fissure, accompanied by the oculomotor and nasal nerves, as well as the ophthalmic vein.

18.1.4 Anastomoses

The abducent nerve forms an anastomosis with:

- the ophthalmic nerve
- the sympathetic system: filaments coming from the carotid plexus.

The abducent nerve begins as solely motor but, through the addition of sensory and sympathetic fibers, becomes sensory.

18.1.5 Distribution

In the orbit the nerve moves forward and laterally, and sinks into the medial ocular surface of the lateral rectus muscle, its only destination.

18.2 PHYSIOLOGY AND PATHOLOGY

18.2.1 Functions

The abducent nerve abducts the eyeball.

18.2.2 Clinical notes

Due to its long intracranial pathway, the abducent nerve is very sensitive to any increase in intracranial pressure.

Paralysis of this nerve is the most common of all ocular paralyses (Fig. 18.2). It involves:

- Loss of abduction. The eyeball cannot deviate laterally from the medial line.
- Convergent strabismus under the action of the medial rectus muscle (III).
- Homonymic diplopia (double vision). To reduce this diplopia, the subject rotates the head to the side of the lesion.
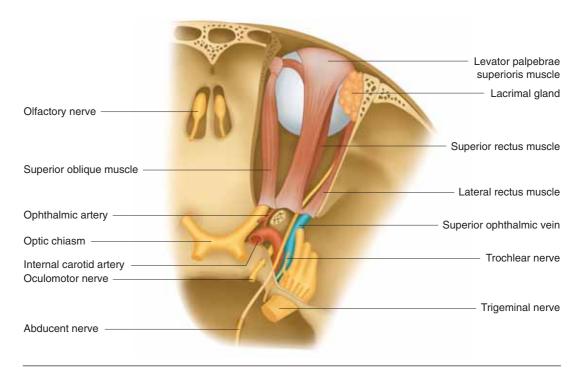
Fig. 18.1 Course of the abducent nerve.
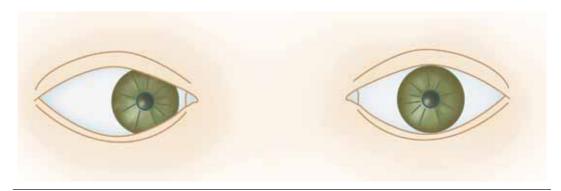
Fig. 18.2 Paralysis of the abducent nerve.

18.3 MANIPULATION

18.3.1 Superior orbital fissure

The abducent nerve lies in the medial aspect of the superior orbital fissure between the oculomotor nerve medially and the trochlear nerve laterally (Fig. 18.3).

We apply the technique of opening the osteodura of the fissure, focusing the action on the medial part of the fissure. The skull is stretched in such a way as to place the medial part of the fissure in convexity.

18.3.2 Eyeball

The abducent nerve supplies only the lateral rectus muscle of the eye (Fig. 18.4). Draw the eyeball medially during the cranial expansion

A

Abducent nerve

Abducent nerve

B
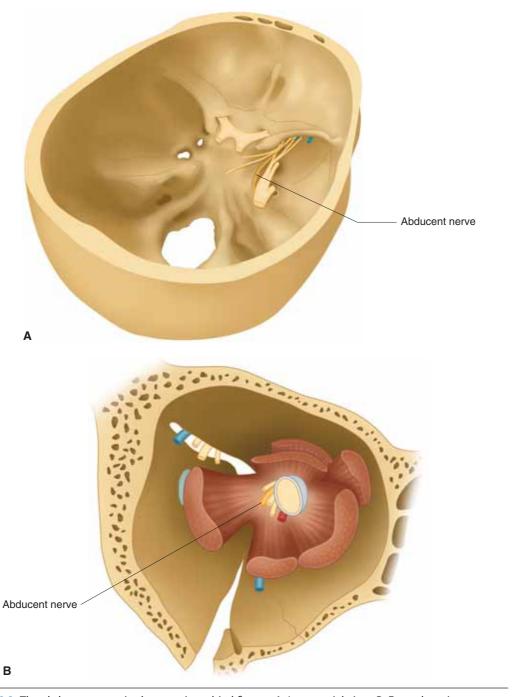
Fig. 18.3 The abducent nerve in the superior orbital fissure. A: Intracranial view. B: Frontal section.

Lateral rectus muscle — Lateral rectus muscle

A

Abducent nerve — Lateral rectus muscle

B

Lateral rectus muscle
Abducent nerve

C
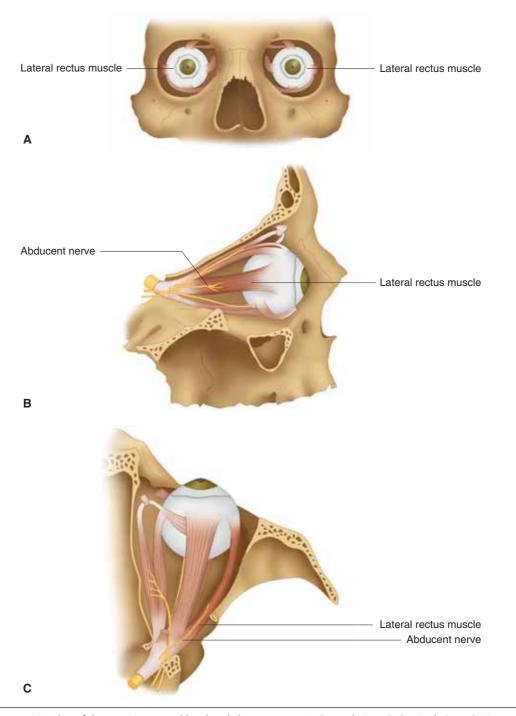
Fig. 18.4 Muscles of the eye innervated by the abducent nerve. A: Frontal view. B: Sagittal view. C: View from above.

Fig. 18.5 Manipulation of the lateral rectus muscle.

phase. Maintain the eyeball on stretch for just a moment before the retraction phase begins (Fig. 18.5).

18.3.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- the dura mater: infra-orbital lining, tentorium cerebelli
- the common tendinous ring (annulus of Zinn)
- the sphenoid: superior orbital fissure (lesser wing/greater wing)
- the frontosphenoidal suture (horizontal plate/lesser wing)

- the sphenosquamosal suture
- the petrosphenoidal ligament (Gruber's ligament)
- the wall of the cavernous sinus: tentorium cerebelli and intracavernous pressure.

18.3.4 Indications

The indications for treating the abducent nerve are as follows:

- Divergent strabismus, if the lateral rectus muscle is hypertonic or in spasm.
- Convergent strabismus, if the lateral rectus muscle is hypotonic or paralyzed.
- Vasomotor problems of the face: the abducent nerve exchanges fibers with the sympathetic system, conferring some autonomic function on it.

Manipulation of the oculomotor nerves

All nerves to the eye muscles have specific actions but, rather than limiting treatment to the muscles a particular nerve innervates, we take a broader approach for the following reasons:

- The nerves have numerous anastomoses with adjacent nerves. For example, the abducent nerve is devoted to the lateral rectus muscle, but nevertheless exchanges fibers with the trigeminal nerve, using the ophthalmic nerve as intermediary.
- The nerves send fibers to the neighboring dura mater. The fibers of these small nerves do not have well-defined territories, and for this reason we must expand our field of action when treating them.
- They anastomose with the sympathetic system either directly by joining fibers or through their ganglia. Relationships with the carotid plexus are frequently found.
- By way of sympathetic and parasympathetic connections, manipulation of these nerves has an undeniable effect on the vasculature. The eyes have a very close relationship with cardiac rhythm and arterial tension. For example, compression of the eyes slows down cardiac rhythm.
- Their sensory role, notably on the face, is not the province of a particular nerve, but rather of both the oculomotor and ocular nerves, together with their anastomoses with the sympathetic and parasympathetic systems.

In our global approach to treatment of the nerves of the eye, we search systematically for all fixations, whether they be neural, muscular or facial.

Happily, thanks to the skill of local listening, the tissues speak to our fingers and we are prevented from focusing too much on one element. It is a good idea, in the beginning, to look for specific fixations; with more experience, we can allow our fingers to go where the tissues lead.

Facial nerve

Early anatomists called the facial nerve (VII) the *expressive nerve*. Certainly, its motor role for the muscles of expression allows us to show our emotions. It also innervates the small muscles of the auditory ossicles and some of the soft palate.

19.1 ANATOMY

19.1.1 Origin

The facial nerve originates from the junction of the pons and the medulla.

It possesses a medial and a lateral root:

- The medial root is larger and constitutes the facial nerve proper.
- The smaller lateral root is located between the medial root and the vestibulocochlear nerve (VIII); this is the intermediate nerve of Wrisberg.

19.1.2 Pathway

From the pons the facial nerve travels laterally and anteriorly with the vestibulocochlear and the intermediate nerve of Wrisberg (Fig. 19.1), and enters the facial canal (the Fallopian aqueduct).

As it runs to the stylomastoid foramen, the facial nerve is divided into three parts by three changes of direction:

- a short horizontal part (a few millimeters) at the bottom of the internal acoustic meatus
- a transverse part (1 cm) located between the two bends

- a vertical part (a little larger than 1 cm) where it descends to reach the stylomastoid foramen.

On emerging from the stylomastoid foramen, the facial nerve innervates the parotid gland and divides into the cervicofacial and temporofacial branches.

Note for manual therapists

For intracranial manipulations directed towards the canal of the facial nerve, two directions are observed:

- sagittal, for the first two parts
- vertical and caudal, for the last part.

19.1.3 Intermediate nerve of Wrisberg

The intermediate nerve of Wrisberg accompanies the facial nerve as far as the genu and ends in the geniculate ganglion ("geniculate" meaning bend or knee).

19.1.4 Geniculate ganglion

The geniculate ganglion is located at the first turn of the facial nerve (Fig. 19.2). Some of its fibers go to the intermediate nerve of Wrisberg and others contribute to the facial nerve as it enters the chorda tympani. The ganglion connects the facial nerve with the sympathetic system.
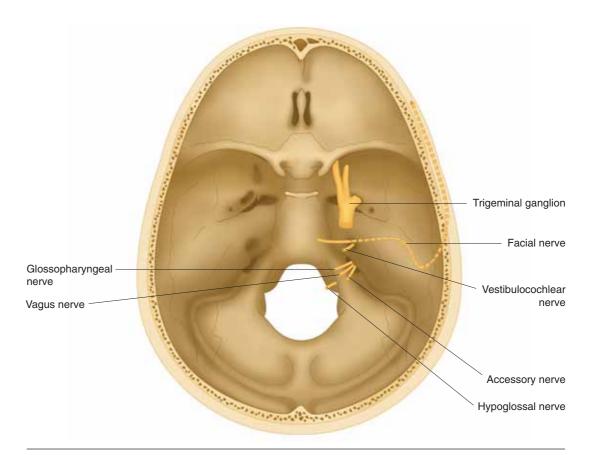
Fig. 19.1 Pathway of the facial nerve.

19.1.5 Useful relationships

In the internal acoustic meatus, the facial nerve, vestibulocochlear nerve and the intermediate nerve of Wrisberg follow the same trajectory. Furthermore, they share a common arachnoid sheath.

In the Fallopian aqueduct the facial nerve, accompanied by the stylomastoid artery, fills the osseous canal.

19.1.6 Anastomoses

The anastomoses of the facial nerve are as follows:

- inside the internal acoustic meatus:
  — the vestibulocochlear nerve
  — the intermediate nerve of Wrisberg
- outside the acoustic meatus:
  — the glossopharyngeal nerve
  — the superficial cervical plexus.

19.1.7 Intrapetrous collateral branches

Within the facial canal the facial nerve gives rise to five intrapetrous branches (Fig. 19.3).

- the greater petrosal nerve
- the communicating branch with the lesser petrosal nerve
- the nerve to stapedius (stirrup muscle)
- the chorda tympani nerve
- the auricular branch (vagal anastomosis).

Greater petrosal nerve

Formerly known as the greater superficial petrosal nerve, the greater petrosal nerve exits the temporal bone by the Fallopian hiatus and runs through an osseous conduit, which brings it to the foramen lacerum.
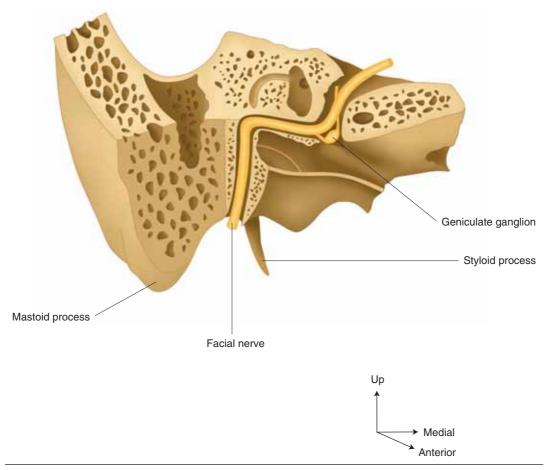
Fig. 19.2 The geniculate ganglion.

At the anterior face of the petrous pyramid it receives sensory fibers from the glossopharyngeal nerve.

In the foramen lacerum, it joins a branch of the carotid plexus to form the vidian nerve and terminates in the pterygopalatine ganglion.

Communicating branch with the lesser petrosal nerve

Formerly known as the superficial lesser petrosal nerve, this small communicating nerve runs to join the lesser petrosal nerve. It follows an osseous canal close to the Fallopian hiatus and emerges from the skull through a small orifice between the foramen ovale and the foramen spinosum. It consti-

tutes the vasomotor component of the lesser petrosal nerve. The secretory component issues from the hypoglossal nerve.

The lesser petrosal nerve also forms a connection between the otic ganglion (where it terminates), the facial nerve and the glossopharyngeal nerve.

Facial nerve branch to stapedius

This nerve is slender and makes its way in the middle ear to meet the stapedius muscle.

Chorda tympani nerve

The chorda tympani nerve arises from the facial nerve a few millimeters above the stylomastoid foramen. It is directed superior

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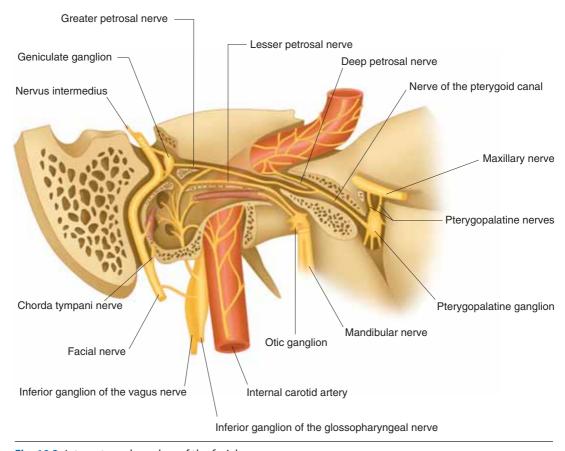
Fig. 19.3 Intrapetrous branches of the facial nerve.

and anterior, and perforates the tympanic cavity. It enters the posterior canaliculus and then descends near the spine of the sphenoid bone. It merges with the lingual nerve, a branch of the maxillary nerve (V3).

This nerve transports the nerves of taste for the anterior two-thirds of the tongue and contains secretory fibers for the sublingual and submaxillary glands. In addition, it sends a branch to the auditory tube.

Auricular branch

The auricular branch forms an anastomosis with the auricular branch of the vagus nerve. It arises just beneath the stylomastoid foramen, level with the chorda tympani. It runs towards the jugular fossa, reaches the superior vagal ganglion and then passes towards the Fallopian aqueduct to meet up with the facial nerve, to which it gives a branch.

On leaving the Fallopian aqueduct, the nerve travels in a small osseous canal. It appears at the base of the skull between the external acoustic meatus and the mastoid process.

It divides into two branches of interest to us:

- The first forms an anastomosis with the posterior auricular nerve.
- The second supplies the medial surface of the auricle and the posterocaudal wall of the acoustic meatus.

It innervates the skin of the external acoustic meatus, the auricle and the lateral surface of the tympanic membrane.

19.1.8 Extrapetrous collateral branches

There are five of these:

- the rami communicantes with the glossopharyngeal and the vagus nerves
- the posterior auricular nerve
- the digastric branch
- the stylohyoid branch
- the lingual branch.

Branches communicating with the glossopharyngeal and vagus nerves

Located immediately at the exit of the stylomastoid foramen, these branches form an important anastomosis with the extracranial part of the glossopharyngeal and vagus nerves.

Posterior auricular nerve

The posterior auricular nerve separates from the facial nerve close to the stylomastoid foramen. It runs anteriorly and laterally in front of the mastoid process. It then turns around the edge of the bone to reach the superior insertion of the sternocleidomastoid muscle (or sterno-cleido-occipito-mastoid).

It receives an anastomosis from the auricular branch of the superficial cervical plexus and divides into:

- a branch to the posterior and superior auricular muscles of the auricle of the ear
- a branch to the occipital muscle.

Digastric branch

The digastric branch arises close to the stylomastoid foramen and supplies the posterior belly of the digastric muscle.

Stylohyoid branch

The stylohyoid branch also arises close to the stylomastoid foramen to enter into the middle part of the stylohyoid muscle.

Lingual branch

The lingual branch passes from the stylomastoid foramen to the base of the tongue. It joins with filaments of the glossopharyngeal nerve in the mucosa of the tongue.

19.1.9 Terminal branches

The terminal branches of the facial nerve are the temporofacial and the cervicofacial divisions (Fig. 19.4).

Temporofacial branch (temporal and zygomatic)

Initially, the temporofacial branch is engulfed by the parotid gland and runs towards the neck of the mandibular condyle. At this level, it receives anastomoses for the auriculotemporal nerve branch of the maxillary nerve (V3).

It divides into filaments supplying:

- the temporal, for the temple and the anterior auricular muscle
- the palpebrae
- the nasal and suborbital branches for the muscles of the face, in particular the nose.

Frontalis belly, occipitofrontalis muscle

Epicranial aponeurosis

Temporal branch

Zygomatic branch

Occipital belly, occipitofrontalis muscle

Buccal branch

Posterior auricular branch

Parotid gland

Cervical branch

Sublingual gland

Marginal mandibular branch

Submandibular gland

Platysma muscle
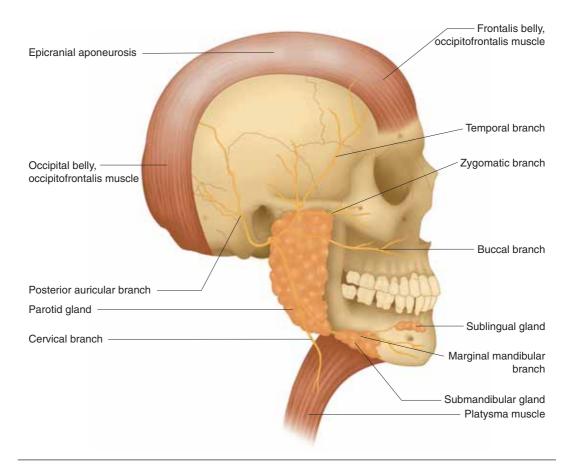
Fig. 19.4 Terminal branches of the facial nerve.

Cervicofacial branch (buccal, marginal mandibular and cervical)

This branch issues from the inferior border of the parotid gland and communicates with the auricular branch of the superficial cervical plexus.

At the angle of the mandible, it divides into filaments:

- the buccal branch, supplying the muscles of the upper lip
- the marginal mandibular branch, some fibers of which anastomose with the inferior dental plexus (mandibular nerve, V3)
- the cervical branch, which goes to the superficial muscle of the neck.

Some fibers form an anastomosis with the superficial cervical plexus.

> **Note for manual therapists**
>
> The terminal branches may be reached at these areas:
>
> - at the temple, where the temporal branches can be mistaken for fibers of the auriculotemporal nerve
> - at the anterior surface of the auricle of the ear
> - surrounding the mental foramen.

19.2 PHYSIOLOGY AND PATHOLOGY

19.2.1 Functions

Motor function

Facial expression

The facial nerve is the nerve of facial expression. All our numerous facial expressions

result from the contraction of subcutaneous muscles. They move the skin and change facial expression to convey mood. The facial nerve innervates all the superficial muscles of the face and the scalp.

Voluntary motor signals derive from the motor cortex. However, a large part of facial expression is involuntary and escapes the control of the superior centers.

In all cultures the face expresses a range of fundamental emotions:

- anger
- pain and disgust
- attention
- contempt
- fear
- joy
- sadness
- surprise or astonishment.

Even people who are blind from birth produce these same expressions. It would seem that these emotional manifestations are not simply acquired, but are innate.

Stapedius muscle
In a less obvious way, the facial nerve intervenes in hearing, innervating the stapedius muscle (stirrup muscle). This muscle diminishes the oscillation of the ossicles of the middle ear, thereby diminishing the vibrations caused by loud noises.

The muscle also contracts as a reflex action on hearing the spoken word. Consequently, a lesion of the nerve branch to the stapedius muscle results in hyperacusis, in which the person has excessive auditory acuity on the injured side.

Evaluation
Tests
Apart from observing the subject's spontaneous facial expressions, we can ask him or her to do the following:

- Raise the eyebrows to assess the symmetry of the frontal muscle.
- Squeeze the eyelids together. If the orbicularis oculi muscle is functional, it is impossible to open the patient's eyelids when he or she resists.

- Purse the lips. If the buccinator and the orbicularis oris muscles are functional, it is impossible to open the patient's lips when he or she resists.

Reflexes
The corneal reflex depends on the facial nerve by way of its motor arc.

The nasopalpebral reflex consists of bilateral contraction of the orbicularis oculi muscles when the bridge of the nose is percussed. However, a preferable way to evaluate this reflex is to hold the patient's eyes closed.

Sensitivity function

The facial nerve conveys general sensation from a small area around the external acoustic meatus, from the eardrum and the concha (area of Ramsay Hunt[1] conjoined with X).

Sensory function

The sensory root (nervus intermedius*) conveys the taste fibers for the anterior two-thirds of the tongue.

Visceromotor function

The parasympathetic component of the facial nerve is important. It controls the lacrimal, submandibular and sublingual salivary glands, as well as the pharyngeal, nasal and palatine glands.

The facial nerve innervates all the major glands of the face, except the integumentary (skin) glands and the parotid.

The cell groups of these neurons are located in the superior salivary nucleus.

This nucleus is connected to the hypothalamus, the center of integration and control of the autonomic nervous system. Nerve impulses coming from the limbic (emotions) system and from the olfactory areas connect to the hypothalamus, and are able to evoke reflex activities like salivation in response to certain smells, or tears in response to certain emotional states.

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1. Ramsay Hunt (1872–1937): British neurophysiologist.

Furthermore, there are connections with the eye, such that an ocular irritant can lead to tearing.

The efferent fibers of the superior salivary nucleus are transported by the intermediary nerve (nervus intermedius). At the geniculate ganglion, in the facial canal, they divide into two groups to become:

- **the greater petrosal nerve**, which relays fibers to the lacrimal and nasal glands
- **a portion of the chorda tympani nerve**, through which fibers are distributed to the submandibular and sublingual glands, as well as for the taste region of the tongue.

19.2.2 Clinical notes

Facial paralysis

Peripheral paralysis
Peripheral facial paralysis, due to a lesion of the trunk or nucleus of VII, produces paralysis of the facial muscles on the ipsilateral side. Complete peripheral facial paralysis results in facial asymmetry. At rest, the paralyzed side is smooth, with:

- an absence of wrinkles on the forehead
- flabby cheeks that flutter during respiration
- deviation of the mouth on the healthy side, causing saliva to run from the drooping corners of the mouth on the injured side
- loss of tone of the orbicularis oculi, causing the lower lid to fall away from the surface of the eye and resulting in tearing
- a positive sign for Bell's palsy: inability to close the eyelid on the paralyzed side, with deviation of the eye towards the superior eyelid
- lack of nasopalpebral and corneal reflexes.

This asymmetry is increased by spontaneous or voluntary facial expression. Wrinkling the forehead, frowning, whistling and blowing are impaired. Traction of the tongue deviates to the healthy side on account of the deformation of the paralyzed corner of the mouth.

Peripheral facial paralysis can be broad. A careful search must be made for signs, predominantly at the eyelids, like asymmetry of blinking and winking or Souques' sign (the eyelashes appear longer on the paralyzed side when the eyelids are closed).

There are many causes of facial paralysis:

- intracranial: tumors of the brain or skull, polyencephalitis
- intrapetrous (temporal bone): fracture, otitis, mastoiditis
- extracranial: parotid gland tumor.

Facial paralysis a frigore is a type of peripheral paralysis caused by arterial vasoconstriction. It brings about a loss of conductivity due to ischemia, worsened by compression (and therefore swelling) of the nerve in the facial canal, especially near the stylomastoid foramen. Viral infection may also be implicated.

Central paralysis
Central facial paralysis, due to a supranuclear lesion, manifests as paralysis of the muscles in the lower face on the contralateral side. Consequently, forehead wrinkling is not impaired. The dissociation occurs because innervation to the upper quadrant of the face is delivered directly by corticonuclear fibers. Central paralysis is often associated with other important neurological deficits (hemiplegia, brachial monoplegia, and so on); it is seldom seen in our clinic.

Facial spasm
Facial spasm is distinct from tics.

Tics are sudden contractions, often bilateral, reproducing certain expressions which have an intentional aspect (winking, grimacing, and so on). With will power and vigilance, these can be toned down.

A facial spasm is, by contrast, unilateral, inexpressive, unsightly, out of place, paradoxical and unmodified by conscious will. The spasm can be total or localized: e.g. blepharospasm (involuntary contraction of the eyelid muscles). Between episodes, motor reactions are normal. Some spasms have a precise etiology, such as cortical epilepsy.

Others are secondary to an old facial paralysis. Most are of unknown origin.

19.3 MANIPULATION

19.3.1 Intrapetrous portion of the facial nerve

As we have seen, from the internal acoustic meatus (internal acoustic pore) the facial nerve runs in the osseous canal, whose route assumes different directions. By following these contours we can carry out specific techniques on the skull. As a rule, manipulations of nerves VII and VIII within the internal acoustic meatus are performed together (Fig. 19.5).

The goal is to have an effect on the nerve through its osteodural environment, especially following cranial trauma and in cases of congenital cranial asymmetries.

Area of the facial nerve

Superior vestibule

End of the internal acoustic meatus

Inferior vestibule

Tractus spiralis foraminosus

Apex of the petrous part of the temporal bone

Internal acoustic meatus
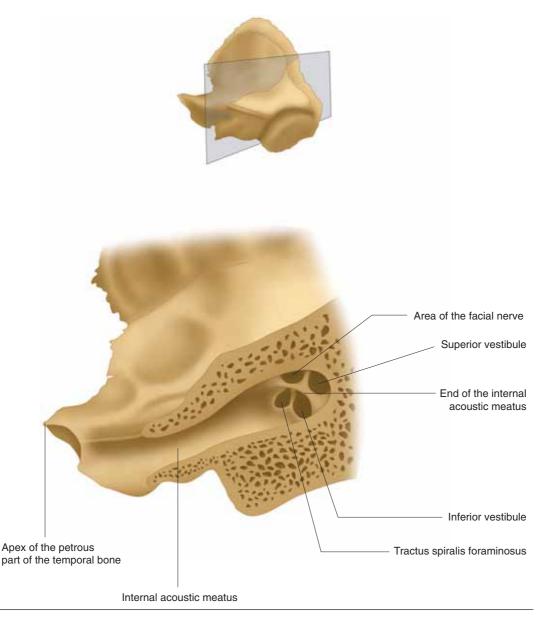
Fig. 19.5 Internal acoustic meatus.

Note that the internal acoustic meatus and its canal also carry the internal auditory artery and vein (branches of the vertebral artery and vein), as well as the vestibulocochlear nerve. It is therefore a highly strategic region for auditory disturbances, problems with balance and tinnitus (noises in the ears).

Unilateral maneuvers

The patient is supine, head turned slightly to the side opposite the facial (and vestibulocochlear) nerves to be treated (Fig. 19.6).

For the right side, for example, the patient turns the head to the left. Place the index finger of your left hand against the anterior rim of the tympanic plate of the temporal bone and its squamous portion. The index and middle fingers make contact with the mastoid process of the right hand.
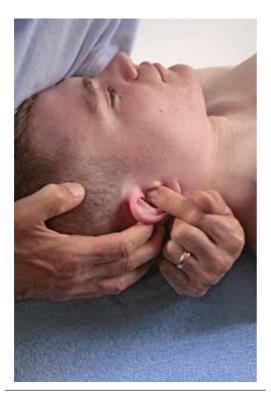
Fig. 19.6 Unilateral manipulation of the intrapetrous part of the facial nerve.

Adapt and modify the rotation of the head, as well as the traction exerted by your fingers, according to the curves and turns of the osseous conduit. As a rule, manipulate by following the various directions in sequence (one after the other) and complete the treatment with a more general induction.

The horizontal conduit
The index finger is placed on the tympanic and squamous part of the temporal bone and points directly anterior, while the fingers traction the mastoid process in a posterior direction.

Note: As with all osseous cranial passages, it is best to execute the manipulations during the cranial expansion phase. The meningeal coating that lines the conduits is especially influenced by cranial movements.

The vertical conduit
With the index finger placed in the opening of the external acoustic meatus, exert traction in a caudad and slightly anteromedial direction, while the fingers on the mastoid work in a caudad and slightly posterior direction.

Bilateral maneuver

The patient is supine, with the head straight. Place your two thumbs in the external auditory pores, deep enough to be in contact with the tympanic part of the temporal bone (a curved plate lying below the squamous part and in front of the mastoid process) (Fig. 19.7).

Begin by pushing the thumbs in an anterior direction. Most of the time, the problem area resides where resistance is greatest. In an arbitrary manner, push your thumbs several times in an anterior then lateral and finally caudad direction in order to stimulate all the mechanoreceptors within the canal. Perform a bilateral induction. The two thumbs can go in the same direction or, sometimes, in opposite directions. Make the movement quite firm.

It seems that this technique is able to balance the bilateral pressures between the

Fig. 19.7 Bilateral manipulation of the intrapetrous part of the facial nerve.

two acoustic meatuses. This is particularly helpful in the amelioration of tinnitus.

19.3.2 Stylomastoid foramen

Anatomy

The trunk of the facial nerve passes through the stylomastoid foramen. It is located between the styloid and mastoid processes (at the inferior surface of the temporal bone, between the tympanic bone in front and the petrous mass behind).

Technique

The patient is supine, the head turned slightly to the side of the stylomastoid foramen to be treated. This technique differs from the usual occipito-mastoid suture technique in that both fingers are placed exclusively on the temporal bone (Fig. 19.8).

Step 1

Place one finger in the anterior part of the external acoustic opening. For example, to make contact with the right stylomastoid foramen, pass your arm under the skull and with your left index finger push the external acoustic opening anterior and medially.

Step 2

Position the middle and index fingers of your right hand against the anterior edge of the mastoid tip to draw it towards the back and medially (see Fig. 19.8).

Working within the elasticity of the osseous tissue of the temporal bone has an effect on the terminal part of the facial canal.

19.3.3 Terminal branches

The facial nerve as it emerges on the face

The facial nerve can be felt a few millimeters after its emergence from the stylomastoid foramen.

Place your index finger behind the ramus of the mandible. Ask the patient to open the mouth and leave it slightly ajar. This opens the retromandibular space (Fig. 19.9).

Move your index finger (sometimes the little finger) as far cephalad as possible, until you feel a sensitive spot. (Try this on yourself; the nerve is easy to find because it can be extremely sensitive.)

From our experience, we prefer to employ the technique for the posterior auricular nerve. It is easier to accomplish, is painless and produces better results.

Fig. 19.8 Manipulation of the facial nerve at the stylomastoid foramen.

Fig. 19.9 Manipulation of the emergence of the facial nerve.

Posterior auricular nerve

The posterior auricular nerve can be reached at the level of its occipital branch and the posterior muscles of the ear. The patient is supine, with the head turned slightly away from the side being treated (Fig. 19.10).

The external acoustic meatus

The techniques for the external acoustic meatus have a major effect on the facial nerve and its anastomoses with the vagus and trigeminal nerves, as well as with the superficial cervical plexus. This technique is described in more detail in Chapter 25.

Fig. 19.10 Manipulation of the posterior auricular nerve.

Occipital branches
Against the mastoid
Place your index finger beneath the external acoustic meatus, directly against the anterior part of the mastoid. Glide the finger cephalad and caudad, forward and backward, in order to find a filament — sometimes two — of the posterior auricular nerve. This technique is that of gliding induction. This facilitates better results than the one used to treat the facial nerve behind the condyle.

In the occipital muscle
Around the curved line of the superior occipital muscles there are many nerve filaments that are valuable for manipulation. Systematically compare the two sides of the anterior surface of the mastoid and the caudal part of the two occipital muscles.

Retro-auricular branches
Glide the index finger behind the ear to meet the small sensitive nerve fibers. The auricle of the ear can be grasped between the index and the middle fingers to make better contact with individual hardened (fibrosed), sensitive nerves.

It is difficult to differentiate the auricular branches of the facial nerve from those of the superficial cervical plexus. This retro-auricular region is very important for manipulation. Within a few square centimeters several anastomoses between various nerves are found. Therefore an effect can be produced upon the following:

- trigeminal nerve
- facial nerve
- suboccipital nerve (of Arnold)
- lesser occipital nerve
- superficial cervical plexus (auricular and mastoid branches).

Temporofacial branch

With the thumb and the index finger, gently palpate the cutaneous tissues, over the temporal bone region, searching for nerve filaments that are painful or sensitive. Treat them with gentle superficial rolling massage.

This technique is especially good for migraines. Do not hesitate to go as far as the area of capillary implantation.

19.3.4 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- dura mater (posterior cerebral fossa, tentorium cerebelli)
- temporal: petrous pyramid, internal acoustic meatus
- temporo-occipital suture
- temporomandibular joint.

19.3.5 Indications

The indications for manipulation of the facial nerve are as follows:

- craniofacial trauma
- following maxillofacial surgery
- facial paralysis
- hearing disorders
- headaches, migraines
- tinnitus
- dental problems (mandibular).

Vestibulocochlear nerve

20

The vestibulocochlear (VIII) nerve is a sensory nerve complex comprised of two distinct nerves. It is concerned with the reception of sound and the maintenance of balance.

The *cochlear nerve*, or auditory nerve, properly called, comes from the organ of hearing (spiral ganglion of the cochlear) and transports auditory sensations. The *vestibular nerve,* or nerve of equilibrium, issues from the organ of equilibrium (vestibular ganglion) and gathers messages that maintain balance, in association with our visual and proprioceptive pathways.

Arising from different parts of the inner ear, these two nerves are separate at their periphery. Later they merge and travel together towards the brain, where they end up in distinct centers.

20.1 ANATOMY

20.1.1 Origin

The vestibulocochlear nerve originates between the pons and the medulla oblongata, by two roots, vestibular and cochlear, emerging behind the facial nerve (VII) and in front of the inferior cerebellar peduncle.

20.1.2 Useful relationships

In the posterior skull

Situated as it is in the groove between the pons and the medulla, the vestibulocochlear nerve makes up part of the acousticofacial bundle. This group is composed of the facial nerve, the intermediary nerve of Wrisberg, the cochlear nerve and the vestibular nerve (Fig. 20.1). Each nerve is surrounded by its own sheath of pia mater as it crosses the cistern of the cerebellopontine angle.

The acousticofacial bundle is situated above the petro-occipital fissure, the inferior petrosal sinus and the posterior surface of the petrous part of the temporal bone. On its course from the pontomedullary junction to the internal acoustic meatus, the nerve runs laterally, slightly anterior and cephalad.

> **Note for manual therapists**
>
> It is important to follow the nerve's trajectory when carrying out treatment.

In the internal acoustic meatus

The facial nerve, and the vestibular and cochlear divisions of the vestibulocochlear nerve cross the internal acoustic meatus together (Fig. 20.2).

The pia mater forms a sheath for each nerve. The arachnoid constitutes a common sheath and the dura mater merges with the periosteum.

The internal auditory (labyrinthine) artery, which may arise from the basilar artery but more often from the anterior inferior cerebellar artery, accompanies these nerves. It distributes to the inner ear and to the facial, vestibulocochlear, cochlear and vestibular nerves.
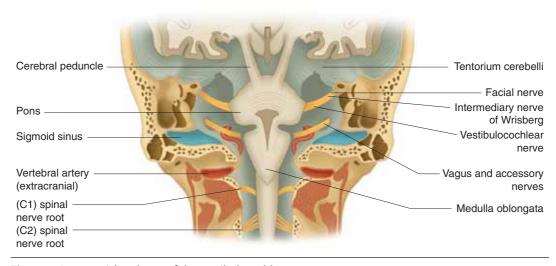
Fig. 20.1 Intracranial pathway of the vestibulocochlear nerve.

Cerebral peduncle

Pons

Sigmoid sinus

Vertebral artery
(extracranial)

(C1) spinal
nerve root
(C2) spinal
nerve root

Tentorium cerebelli

Facial nerve

Intermediary nerve
of Wrisberg

Vestibulocochlear
nerve

Vagus and accessory
nerves

Medulla oblongata

Vestibulocochlear nerve

Cochlea

Vestibule
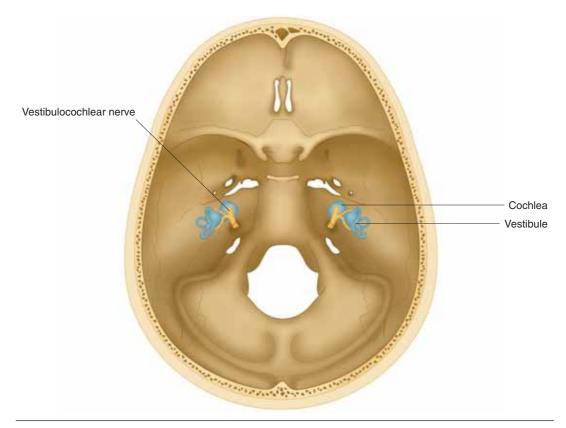
Fig. 20.2 The vestibulocochlear nerve in the internal acoustic meatus.

Note for manual therapists

All the techniques addressed to the thoracic inlet, the subclavian artery, the vertebrobasilar trunk and the foramen magnum play an important role in terms of the entire vascularization of the internal ear. At the bottom of the internal acoustic meatus, the nerve distributes into its terminal branches.

Internal acoustic meatus

The internal acoustic opening is located on the posterior or cerebellar surface of the petrous part of the temporal bone. The posterior aspect of the crest (arcuate eminence) separates the middle cranial fossa from the posterior cranial fossa. This orifice has an oval shape and is about 5 mm high and 8 mm long.

The internal acoustic meatus brings the labyrinth into communication with the posterior cerebral fossa (Fig. 20.3). Nestled inside the petrous pyramid, it forms a short passage between the cochlea medially and the vestibule laterally. It contains the facial nerve, the vestibulocochlear nerve and the labyrinthine artery (the acousticofacial bundle).

The axis runs transversely (practically on a bi-auricular line) in a lateral and very slightly anterior direction. Keep this orientation in mind when directing your technique at the vestibulocochlear nerve. The lateral end, or fundus, of the internal acoustic meatus is separated from the inner ear by a vertical plate that is divided by a transverse crest.

- Superior to the crest (cephalad level) lay the facial and intermediary nerves, and posteriorly the superior vestibular area is found.
- The caudad level is occupied anteriorly by the tractus spiralis foraminosus and posteriorly by the inferior vestibular area.
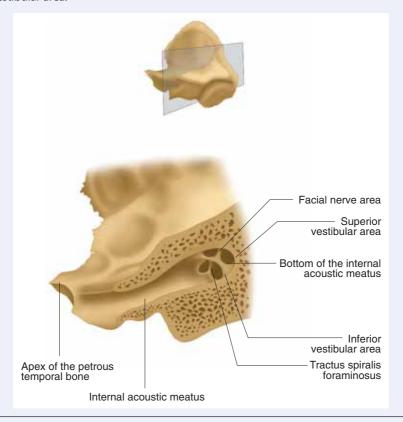
Fig. 20.3 The internal acoustic meatus.

20.1.3 Terminal branches

The cochlear nerve runs towards the cochlea and coils up on itself in the manner of a volute (spiral). The nerve fibers (about 40 000 of them) cross the foramina in the tractus spiralis foraminosus and end at the cochlear ganglion.

Neurofibers leave the cochlear ganglion destined for the inner hair cells, and a small contingent end in relation to the outer hair cells of the spiral organ of Corti.

Note: Testut (1948) notes that these nerve fibers terminate in similar fashion to those of the olfactory nerve: across a cribriform plate. The fibers arising from the spiral ganglion lose their myelin sheath as they pass though the foramina — about 2500 perforations — in the cochlear canal.

The vestibular nerve goes to the vestibule. A little after its separation from the cochlear nerve, it presents the bulge of the ganglion of Scarpa, and immediately after divides into two parts:

- The cephalad part crosses the superior vestibule and subdivides into:
  — the utriculo-ampullary nerve, which gives off:
    — the utricular nerve for the macula of the utricle
    — the anterior and lateral ampullary nerves for the corresponding ampullary crests
  — the superior saccular nerve, for the macula of the saccule.
- The caudal part furnishes:
  — the saccular nerve for the macula of the saccule
  — the posterior ampullary nerve for the posterior ampullary crest.

20.2 PHYSIOLOGY AND PATHOLOGY

20.2.1 Functional anatomy

Labyrinth

The labyrinth, or internal ear, is so termed because of its complex and winding appearance. Deeply set in the temporal bone, it is composed of two parts (Fig. 20.4):

- **The osseous labyrinth** is a whole series of chambers and ducts that communicate with each other. The walls (otic capsule) are bordered by bone, in the form of a shell.
- **The membranous labyrinth**, upon which the bony labyrinth is molded, is contained by simple epithelial membranes. Within the labyrinth is a viscous fluid called endolymph, whose composition is close to that of intracellular fluid.

The osseous labyrinth and the membranous labyrinth are separated by the perilymphatic space, containing perilymph, a clear liquid similar to cerebrospinal fluid (Fig. 20.5).

The labyrinth has two divisions:

- the posterior vestibular labyrinth
- the anterior cochlear labyrinth.

Vestibular labyrinth

The vestibular nerve and the vestibule play a key role in equilibration and in upright posture, by providing awareness of the position of the head in space. In association with visual and proprioceptive pathways, it is one of the three posture regulators.

The vestibular labyrinth is divided from back to front into three parts (Fig. 20.6):

- three semicircular canals
- a vestibule that comprises two membranous sacs: the utricle and the saccule
- the internal acoustic meatus.

Static labyrinth

The utricle and the saccule each contain a macula (receptor) of 3 mm^2. These two maculae are sensory receptors that signal the position of the head.

The macula of the utricle is relatively horizontal, whereas the macula of the saccule is vertically placed. The hair cells of the maculae are covered with a gelatinous matrix in which
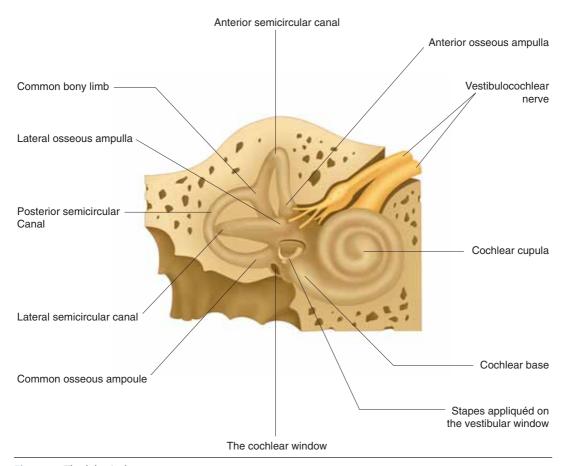
Fig. 20.4 The labyrinth.

minute crystalline bodies are imbedded; these are made up of calcite and associated proteins. These bodies are called otoconia — "sand of the ear".[1]

The otoconia exert traction upon the hair cells (cilia) in response to gravity. As a result, the hair cells bend, and this stimulates the vestibular nerve. Thus the maculae respond to the linear acceleration of the head, on the horizontal plane (while walking, for example) or on the vertical plane (with the acceleration of gravity).

The principal function of the static labyrinth is to indicate the position of the head in relation to the trunk. In response to this signal, the vestibular nucleus organizes compensatory movements for balance. These movements have the effect of maintaining the center of gravity between the feet while standing (static equilibrium) or just in front of the feet during locomotion (dynamic equilibrium). In addition, they allow the head to stay vertical.

The static labyrinth controls balance via the lateral and medial vestibulospinal fascicles. This system functions in association with the plantar proprioceptors and the retina, for the maintenance of upright posture.

Note: The static labyrinth contributes to segmental awareness: that is, the sense of knowing the position of the body in space. This true *sixth sense* is normally provided by three systems: the visual system, the proprioceptive system and the vestibular system. If deprived of one of these systems, a person is

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1. The French term "otolithes" (otoliths) refers to "small stones of the ear" in reptiles. In France, the term is currently used in place of "otoconies" (otoconia).
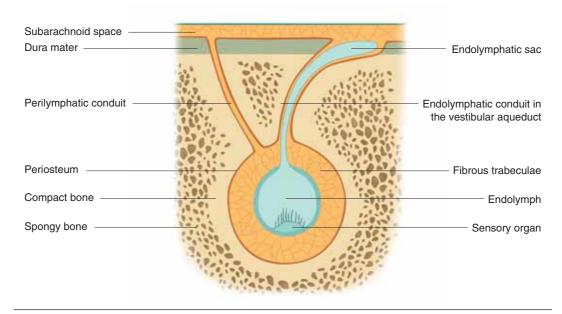
Fig. 20.5 Schematic organization of the labyrinth fluids.

Subarachnoid space
Dura mater
Perilymphatic conduit
Periosteum
Compact bone
Spongy bone

Endolymphatic sac
Endolymphatic conduit in the vestibular aqueduct
Fibrous trabeculae
Endolymph
Sensory organ

Scarpa's ganglion

Vestibular nerve

Auditory nerve

Cochlea

Semicircular canals

Utricle   Saccule

Organs with otolithes
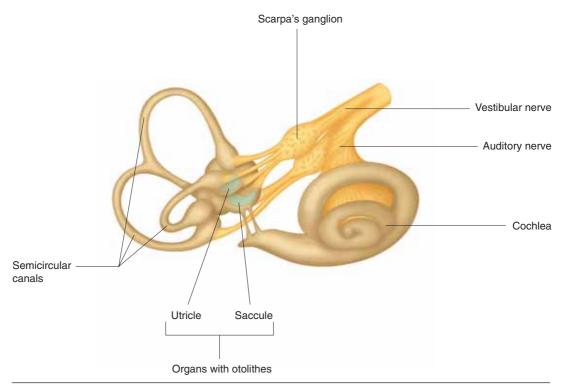
Fig. 20.6 The vestibular labyrinth.

able to remain standing and move about using the awareness provided by the two other systems. For example, after loss of vision, the subject can still walk and remain upright, despite the constraints of blindness.

After the loss of conscious proprioception, people utilize vision as a substitute. In these circumstances, closing their eyes "disables" them (ataxia). Likewise, if the static labyrinths are inactive, simply closing your eyes could cause a heavy fall.

Dynamic labyrinth

Each of the semicircular canals has an ampulla at one end, containing a sensory area: the ampullary crest. These perpendicular crests, found at the axis of the canal, are the receptors of the dynamic labyrinth. The crests are sensors for recording movements of endolymph in the ampullae resulting from rotation of the head in the plane of the duct.

The main function of the dynamic labyrinth is to signal movements of the head, prompting compensatory ocular movements in response.

The vestibulo-ocular reflexes use the pathways uniting the vestibular nuclei with the motor nuclei of the ocular muscles. They have the effect of keeping the gaze on a selected target while the head moves. Thus, the two eyes move together in parallel fashion.

Cochlear labyrinth

The cochlear nerve provides hearing by transmitting the sound impulses coming from the cochlea (Fig. 20.7).

The main part of the cochlear nerve derives from the central processes of the 30 000–40 000 large bipolar neurons occupying the spiral ganglion.
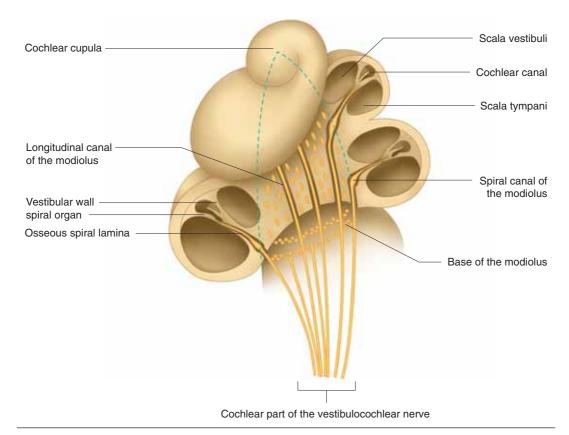
Fig. 20.7 The cochlear labyrinth.

The cochlea is part of the anterior labyrinth and resembles the shell of a snail. It forms a hollow tube, wound up on itself two and a half times. The spiral plate separates two independent compartments:

- the cochlear duct filled with endolymph (similar to intracellular fluid)
- a duct filled with perilymph (similar to cerebrospinal or extracellular fluid).

Acoustic waves are received by the tympanic membrane, then amplified and transmitted to the oval window of the vestibule. Waves of pressure are created in the perilymph of the vestibule by vibrations at the base of the stapes and these ascend to the apex of the cochlea by a channel called the scala vestibuli. Because the perilymph and endolymph are incompressible, the vibrations create pressure variations that are transmitted from the scala vestibuli towards the scala tympani. They pass through the helicotrema, mobilizing the endolymphatic fluid of the cochlear canal.

These movements of fluid are transmitted to the hair cells of the organ of Corti, contained in the cochlear duct. The impulse thus generated is then transmitted to the cochlear nerve. The apex of the cochlea encodes low frequencies and the basal part encodes higher frequencies.

20.2.2 Clinical notes

Although the vestibular and the cochlear nerves are independent, their peripheral lesions often have similar clinical consequences due to their close relationship. Thus lesions of VIII can give rise to ringing in the ears, vertigo, and a disturbance or loss of hearing.

Vestibular nerve

Vestibular syndrome
Injury to the vestibular function simultaneously affects ocular and postural movements. It is responsible for vertigo, balance problems and nystagmus (rhythmic deviation of the eyeballs). Accordingly, nerve and vestibular function can be evaluated by observing postural balance and eye movements.

Vertigo
Definition
Vertigo is a hallucinatory movement involving the patient or the patient's environment. It is a false sensation of the displacement of the subject with regard to surrounding objects, or of the objects in relation to the subject. Most commonly of rotational form, vertigo has three components:

- cerebral: dizziness, a whirling sensation, or a feeling of swaying forward and back, or falling, anxiety
- unsteadiness, a consequence of standing at a dizzying height
- autonomic and vasomotor: due to repercussions on the nucleus at the pons.

Vertigo is often accompanied by a vestibulo-ocular nystagmus.

Vertigo points to a disorder of the vestibular system: of the posterior labyrinth, vestibular nerve and centers of integration, and signal processing. Disturbance of this system prompts the sending of erroneous information to the centers of equilibrium — messages that contradict other sources of information. Vertigo arises from this sensory conflict and is often associated with nausea and vomiting.

The major causes of vestibular vertigo
Benign positional vertigo
This is the most frequent cause; vertigo is provoked solely by a change in position of the head. The attack is brief, lasting just 30–60 seconds, but the nausea can be lasting. The cause can be a blockage of otoliths which have become stuck in one of the semicircular canals.

The mechanism corresponds to cupulolithiasis of the posterior semicircular canal (the deposit of otolithic particles from the utricular macula on the cupula of the posterior semicircular canal); or to canalolithiasis (otoliths moving in the endolymph of the posterior semicircular canal). On turning or elevating the head, the displacement of otoliths stimulates certain receptors, thus sending a false message to the brain. The classic medical treatment is the Epley maneuver, which moves the otoliths out of the canal.

In osteopathy, we find many other causes of benign positional vertigo: for example, problems of the thoracic or vertebral artery in the vertebral canal.

Neuronitis or vestibular neuritis

Fortunately, this type of vertigo is less frequent as it confines the patient to bed. The duration of the attack can be from 2 days to 2 weeks. The etiology is unknown, but we suspect a viral attack on the vestibular nerve. Labyrinthitis can affect not only the vestibular nerve, but also the entire inner ear. Look for hypoacusis. Though patients generally have just a single attack of neuronitis in a lifetime, cases of recurring neuronitis have been reported.

A vestibulocochlear neuritis can be due to mechanical problems or even to medication or alcohol intoxication.

Labyrinthitis often presents a clinical picture similar to auditory dysfunctions. It can be due to a simple otitis.

Ménière's disease or Ménière's vertigo

This is a particular variety of vertigo characterized by recurrent attacks. Progressive hearing loss and tinnitus result, sometimes accompanied by headache. Often there is the sensation of a "plugged" ear.

These episodes are intense (paroxysmal) and can occasionally cause falls. They are worsened by sudden head movements. The attacks are frequent at the beginning, then spread out over time. The crisis can last from a few moments to several hours. Between bouts, the patient is generally asymptomatic.

After the vertigo has passed, hypoacusis or even deafness sets in. In our practice, we often see patients with so-called Ménière's syndrome where there is no loss of hearing. The etiology is unknown, but it is likened to "acute vestibular glaucoma".

There will be an increase in the endolymphatic fluid (endolymphatic hydrops) in the membranous labyrinth of the inner ear, giving rise to dilatation of the cochlear duct, utricle and saccule. This heightened pressure, often attributable to occlusion of the cochlear aqueduct, develops through recurrent episodes of vertigo with varying degrees of time lag between them. Some authors have hypothesized allergy factors as a cause.

Acoustic neuroma

There will be vertigo and disequilibrium, but the principal symptom is hearing loss in one ear. The duration of the vertigo is variable, but it would seem to be triggered by changes of position or postural readjustments. The etiology is a slow-growing benign tumor beginning in the vestibulocochlear nerve.

Note: Vertigo can also be a sign of other disorders, some of them serious:

- vertebrobasilar insufficiency
- compression of the subclavian artery
- disruptions in cardiac rhythm, arterial hypo- or hypertension, cardiac insufficiency, anemia, the effects of medications, poor eyesight
- labyrinthian ischemia can be found with atherosclerosis (or arteriosclerosis) causing diminished blood flow through the vertebral arteries, and thereby influencing circulation to the internal acoustic (labyrinthian) arteries
- anomalies at the craniospinal junction (associated with problems of the last pair of cranial nerves, cervical pain and headache, triggered by maneuvers which increase cerebrospinal fluid pressure)
- following head trauma, contusions or fractures of the temporal bone or the bony labyrinth. Most means of investigating the skull fail to find the many fixations detectable by the manual therapist's hand
- multiple sclerosis (vertigo syndrome is the first symptom in 5–7% of cases)
- ischemic accident of the cerebral trunk and the posterior cerebral fossa (cerebellar syndrome)
- intra- or extracerebral expansive processes, especially at the cerebellopontine angle
- cochlear otosclerosis (otospongiosis), fibrous dysplasia, Lobstein's disease and Paget's disease, which can all involve vertigo syndrome with tinnitus and deafness.

Nystagmus

The semicircular canals are stimulated by the movement of endolymphatic fluid, subject to movements of acceleration or deceleration. In the case of a lesion, among the visible changes arising are those affecting the ocular muscles, called nystagmus.

Nystagmus is characterized by a rhythmic trembling of the eyeballs; involuntary oscillations may be horizontal, vertical, rotary or multidirectional. Vestibular nystagmus is most frequently rotary.

Disequilibrium

Equilibrium disorders are the result of vestibular ataxia. They are characterized by an impaired ability to coordinate movement, resulting in a staggering gait and postural imbalance.

With a unilateral vestibular disorder, patients cannot walk in a straight line, as they feel the urge to move laterally towards the damaged labyrinth. Subjects tend voluntarily to overcorrect this deviation, resulting in a characteristic zigzag gait. If you ask them to walk with their eyes closed, alternately forwards and backwards, you would observe Babinski's reflex in the uneven gait.

In cases of bilateral vestibular disorder, there are no gait problems. The clinical examination utilizes behavior tests or tests that involve shifting, moving and transferring weight rapidly in order to uncover a vestibular deficit.

Clinical tests of vestibular function

Romberg sign

Patients cannot keep their balance when standing erect with their feet together and eyes closed. Patients are examined in the standing position, with bare feet, heels together and feet 30° apart. Position yourself behind patients to be ready in case they fall when they close their eyes.

Romberg sign indicates ataxia whose origin can stem from various sources. In vestibular cases, once past the acute phase, Romberg sign is called "lateralized", exhibiting a tendency to fall to the afflicted side.

Note: Romberg sign must not be confused with Romberg's postural test, which evaluates fine tactical postural changes when subjects close their eyes.

Walking test

Ask the patient to walk in a specific direction. In cases of deviation or staggering, suspect a labyrinthine problem.

Index finger test

With patients seated, ask them to use their index finger to touch an object displayed in front of them. In patients with a labyrinthine disorder, the finger will deviate.

Spontaneous ocular nystagmus

With patients seated, ask them to fix their gaze on your finger as you slowly move it up, down and laterally. Place your finger about 30 cm from the patient's face. With labyrinthine dysfunction, the eyeballs will make involuntary oscillating movements. As a rule, you will witness faster movements in one direction than in the other.

Test with arms outstretched

Observing the same conditions as for Romberg sign, ask the subject to extend the arms in front horizontally, with the hands together at their radial edge. Observe the position of the index fingers by placing your own on the surface of the patient's. If a vestibular disorder exists, a deviation of the index finger (by rotation of the trunk and the scapula) will occur on the side of the vestibular lesion.

Fukuda's test

This test consists of marching on the spot, eyes closed and arms outstretched, taking 30 steps in 30 seconds and lifting the knees to a 45° angle. Medically, this test is considered positive for a vestibular problem if the deviation in position reaches 30°.

This test is also used when studying posture, as the slightest deviations can show a functional problem in postural regulation.

The cochlear nerve

Damage to the cochlear nerve manifests either as a loss of auditory acuity (deafness, hypoacusis) or as a subjective disorder (symptoms like buzzing and a noise in the ears).

Subjective disorders: noise in the ears

Buzzing, ringing and the sound of bells ringing or of paper rustling are all called tinnitus. They signal irritation of the acoustic formations, particularly of the inner ear and the cochlear nerve.

Pulsating buzzing (droning)

Patients hear a rhythmic vascular-type noise. They say that they hear their own pulse in their ears. These droning noises can signify otitis, simple ear congestion, aneurysm, the sequelae of cranial trauma or arterial hypertension.

They could also indicate a lower cervical fixation or, especially, a fixation of the first rib because of its proximity to the stellate ganglion. Note that the first rib is often a witness of an adjacent visceral restriction, notably of the pleural dome, the lung or the homolateral subdiaphragmatic organs.

Other tinnitus

Tinnitus can be found when there is irritation of the vestibulocochlear nerve and its branches. It has numerous characteristics: the sound of paper rustling, continuous humming or purring, whistling, the sound of a conch shell placed against the ear, a foghorn, and so on.

It manifests as:

- problems of the external ear: a plug of earwax, water in the ear, and so on
- lesions of the tympanic membrane: otitis, otosclerosis, tubal catarrh; for the most part, the patient hears low deep sounds
- lesions of the inner ear: hypertension, increased hydraulic pressure of the endolymph, ischemia; the patient tends to hear high-pitched sounds.

It is important to distinguish between:

- intracranial blowing murmurs, sometimes audible with auscultation, due to intracranial arteriovenous aneurysms
- auditory hallucinations caused by irritation of the cortical auditory center.

Objective disorders: deafness

Deafness is a partial or complete loss of hearing. Hearing analysis should be quantitative (degree of deafness), as well as qualitative (type of deafness). All forms of hearing loss can be grouped into two categories:

- Conductive deafness (also called transmission deafness) indicates that there is an obstacle to sound transmission between the middle ear and the cochlea.
- Sensorineural deafness (perception deafness) signals a deterioration of the auditory pathway from the cochlea to the auditory cortex of the brain.

Conductive deafness can be due to:

- An accumulation of earwax in the external acoustic meatus.
- Water in the ear (after shampooing, for example).
- Otitis media, inflammation of the middle ear.
- A pierced eardrum (an intense explosion, deep-sea diving).
- Otospongiosis, which is irregular ossification of the inner ear where the capsule of the synovial joint between the stapes and the labyrinth is progressively replaced by bone. Little by little the stapes is immobilized, causing a severe loss of hearing in all tonal ranges.

Sensorineural deafness usually originates from disease in the cochlea. The principal types are the following:

- **Loss of hearing in the elderly**. Loss of high-pitched sounds (presbycusis) is most frequent. It results from deterioration of the spiral organ of Corti. The elderly can have difficulty distinguishing consonants of high

frequency (d, s, t,). However, vowels, which are of low frequency, are clearly audible. It is therefore better to speak distinctly to older listeners rather than in a loud voice.

- **Work-related deafness**. This type occurs in a noisy work environment. A persistent noise can bring about eventual degeneration of the organ of Corti in the region that corresponds to the particular frequency solicited.
- **Acoustic trauma**. Chance exposure to a loud noise of great intensity can temporarily disturb hearing and cause transitory deafness.
- **Cranial trauma**. Temporal bone fractures or violent contre-coup lesions due to whiplash can definitively damage the cochlear nerve and the cochlea.
- **Cerebrovascular accidents**. A transient ischemic attack or thrombosis of the labyrinthine artery can engender hearing loss with or without vertigo.
- **Ototoxic vertigo**. Medications can have a harmful effect on the organs of hearing and balance: streptomycin, neomycin, quinine and even aspirin, for example.
- **Infectious deafness**. This can be due to the more or less complete destruction of the cochlea by the mumps virus or congenital rubella.

Mixed forms of deafness have a progressive evolution. They affect both ears and are often a familial trait. Women are more frequently affected, in synchrony with major hormonal changes (puberty, pregnancy, menopause, and so on).

Acoustic neuroma is a benign tumor of VIII. It is an important cause of sensorineural deafness in adults. As well as affecting the vestibular and cochlear nerves, this neuroma can impact the facial and trigeminal nerves.

20.3 MANIPULATION

20.3.1 Traction–listening techniques

With the hand on the side opposite the manipulation, place the thumb on the greater wing of the sphenoid and the other fingers under the occiput (Fig. 20.8). On the treatment side grasp the ear between your thumb on one side and your index and middle fingers on the other side (Fig. 20.9).

Step 1

In order to open the sphenobasilar junction on the side you are treating, approximate your thumb (on the greater wing of the sphenoid) and fingers (on the occiput) on the opposite side. Proceed delicately so as not to irritate the temple region, which is very sensitive.

Step 2

Gently grasping the tragus, exert mild progressive traction on the ear and on its posterior aspect where it joins the skull. Draw the ear in a lateral, slightly cranial and anterior direction. Roughly speaking, traction the ear (the tragus) away from the mastoid tip of the opposite ear.

Step 3

Once you perceive the intracranial tissues to be under tension, do not increase your traction. The temporal bone will itself oscillate around an axis of tension produced by the vestibulocochlear nerve and the labyrinth.

Carry out induction of the various movements that arise until a point of equilibrium is reached. Then very slightly increase your traction two or three times, precisely following the axis of the nerve, which can be clearly perceived. Afterwards, release the ear and allow the spheno-occipital to return to its neutral position.

Note: This technique is interesting, not only because of its effects on the vestibulocochlear nerve, but also because of its influence on the internal acoustic artery (labyrinthine) which accompanies it.

The vascularization of the labyrinth is terminal, meaning that the inner ear is very sensitive to ischemia, as well as to lowered pressure or blood deficit. This technique also improves perfusion of the vestibule and the cochlea.

External acoustic meatus

Internal acoustic meatus
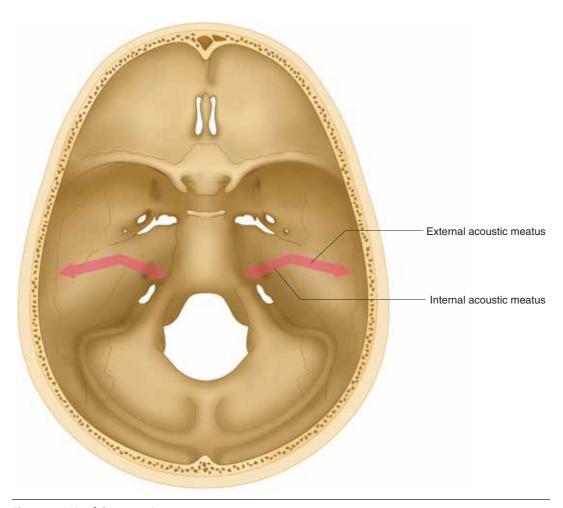
Fig. 20.8 Axis of the acoustic meatus.

Fig. 20.9 Traction — listening technique for the vestibulocochlear nerve.

20.3.2 Globalization

Generally, treatment of the vestibulocochlear nerve is combined with that of the facial nerve:

- in the intrapetrous pathway
- at the posterior auricular nerve
- in the external acoustic meatus.

20.3.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- dura mater (posterior cerebral fossa, tentorium cerebelli)
- temporal: petrous pyramid, internal acoustic meatus
- occipito-temporal suture
- auditory tube (Eustachian).

20.3.4 Indications

The indications for manipulation of the vestibulocochlear nerve are as follows:

- Vertigo, Ménière's disease, disequilibrium. The following are necessary for good equilibrium:
  — An appropriate pressure gradient of the fluids of the inner ear
  — Integrity of the locoregional and central nervous systems, notably the connections of the vestibular nerves to the cerebellum
  — A skull free of all osteo-sutural and membranous fixations
  — The facial nerve, intermediary nerve of Wrisberg and the vestibulocochlear nerve free of constraint
  — Good perfusion pressure to the internal acoustic artery
- Motion sickness
- Hypoacusis
- Proprioceptive problems
- Recurrent postural imbalance
- Recurrent cervical pain.

Glossopharyngeal nerve

21

The glossopharyngeal nerve (IX) contains both sensory and motor fibers. It plays a motor role for the pharynx and the soft palate, while sensory fibers supply the mucosa of the tongue and the ear.

21.1 ANATOMY

21.1.1 Origin

The hypoglossal nerve emerges laterally from the medulla, below the vestibulocochlear nerve and above the vagus nerve.

21.1.2 Pathway

From the medulla, the glossopharyngeal nerve runs laterally and slightly anteriorly towards the base of the skull. It bends at right angles and descends vertically to reach the jugular foramen, through which it passes to arrive at the posterior part of the tongue and then make its way to the cervical level (Fig. 21.1).

21.1.3 Useful relationships

Within the cranium

The glossopharyngeal nerve is set in a shared arachnoid sheath with the vagus and the accessory nerves.

In the osseous foramen

The glossopharyngeal nerve occupies the most anterior and medial aspect of the jugular foramen. Fibrocartilagenous tissue separates it from the vagus nerve, the accessory nerve and the jugular vein (Fig. 21.2).

In the cervical region

Emerging from the jugular foramen, the glossopharyngeal nerve curves forward towards the tongue. It is accompanied medially by the carotid artery and laterally by the jugular vein. More caudally, it descends in front of the carotid and courses in the space contained between the stylopharyngeus and styloglossus muscles. Finally, it distributes to the lingual mucosa.

21.1.4 Ganglions

The ganglions contain the cell bodies of the afferent fibers of the glossopharyngeal nerve.

Superior ganglion (of Ehrenritter)

Of less importance than the inferior ganglion, the superior ganglion is found just before the glossopharyngeal nerve exits the jugular foramen.

Inferior ganglion (of Andersch)

The inferior ganglion is situated on the nerve as it exits the jugular foramen. It relates to the middle part of the posterior border of the petrous portion of the temporal bone.

21.1.5 Anastomoses

The glossopharyngeal nerve forms anastomoses with four nerves:

- **Vagus**. A slender branch joins the glossopharyngeal to the vagus nerve, just beneath the jugular foramen at the level of the inferior ganglion.
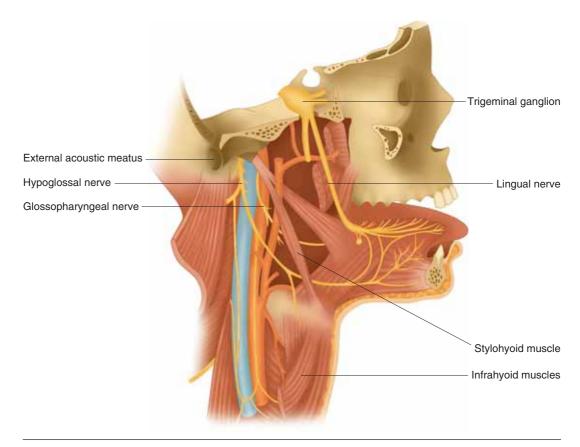
Fig. 21.1 Pathway of the glossopharyngeal nerve.

- **Facial**. A branch of the glossopharyngeal joins the two nerves just below the inferior ganglion. This branch detaches from the facial nerve underneath the stylomastoid foramen. The tympanic nerve stems from a collateral of the glossopharyngeal.
- **Sympathetic**. This anastomosis is made via a branch issuing most commonly from the inferior ganglion. It joins the carotid branch of the superior cervical ganglion.
- **Trigeminal**. This union is made in the tongue via the terminal lingual branches.

21.1.6 Collateral branches

Tympanic nerve

The tympanic nerve was formerly called the nerve of Jacobson, after the Danish physician who first discovered it (Fig. 21.3).

It arises from the anterolateral aspect of the inferior ganglion. It runs in an osseous canal at the posterior caudal surface of the petrous part of the temporal bone. It ascends in a vertical groove to reach the tympanic cavity, where it divides into branches that form the tympanic plexus. The tympanic plexus innervates:

- the mucosa lining the tympanic cavity
- the mucosa of the auditory (Eustachian) tube.

From the tympanic plexus issue:

- the carotid branch, which forms an anastomosis with the carotid plexus
- the lesser petrosal nerve, which receives a connecting branch from the facial nerve and then joins the otic ganglion.
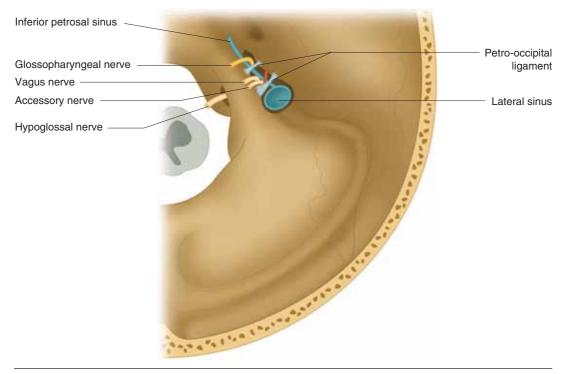
Fig. 21.2 The glossopharyngeal nerve in the jugular foramen (viewed from above).

Other branches

The glossopharyngeal nerve supplies:

- A branch arising from the inferior ganglion, which communicates with the auricular branch of the vagus nerve.
- The carotid branch, which descends on the internal carotid artery and runs close by it to distribute to the carotid sinus and the carotid body. It joins with fibers of the vagus nerve and of the superior cervical ganglion to form the intercarotid plexus.
- The pharyngeal branches, which associate with filaments of the vagus nerve and of the superficial cervical ganglion to supply motor filaments to the superior constrictor muscles of the pharynx, sensory filaments to the pharyngeal mucosa and vascular filaments to the pharynx.
- The muscular branch, to supply the stylopharyngeus muscle.

- The tonsillar branches for the amygdala tonsil and the soft palate.
- The nerves to the styloglossus and glossopharyngeal muscles.

21.1.7 **Terminal branches**

The glossopharyngeal nerve ends in the lingual branches of the mucosa of the posterior tongue, behind the transverse sulcus (V lingual), the vallate papillae (taste buds), the epiglottis and the lateral glosso-epiglottic folds (Fig. 21.4).

21.2 **PHYSIOLOGY AND PATHOLOGY**

21.2.1 **Functions**

Motor function

This function concerns the elevation of the pharynx when swallowing. Accordingly, the glossopharyngeal supplies these muscles:
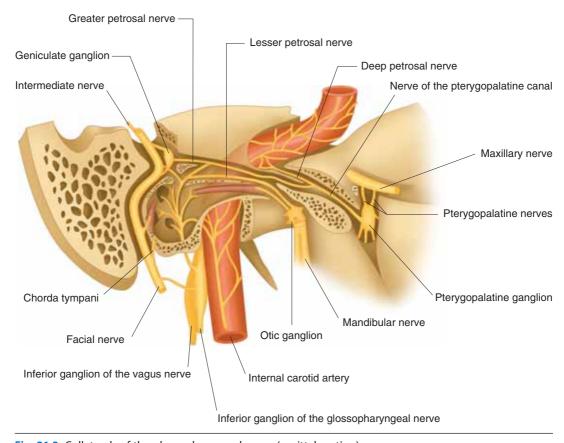
Fig. 21.3 Collaterals of the glossopharyngeal nerve (sagittal section).

- stylopharyngeus
- staphylopharyngeus
- superior constrictor muscle of the pharynx.

Sensory function

The glossopharyngeal nerve supplies branches to the mucosa (Fig. 21.5) of:

- the nasopharynx
- the Eustachian tube
- the tympanic cavity
- the oropharynx
- the amygdala tonsil
- the posterior third of the tongue
- the glosso-epiglottic sulcus.

The gag reflex depends on the excitation of the pharyngeal mucosa, and is carried along the tracts of the glossopharyngeal nerve.

Sense of taste function (special sensory)

The glossopharyngeal nerve supplies the special sense of taste to the posterior third of the tongue, the epiglottic sulcus and the isthmus of the throat. Ageusia is a loss or impairment of the sense of taste.

Autonomic function

Secretory function
The glossopharyngeal nerve innervates the parotid gland and participates in the salivary reflex during mastication. Note that stimulation to the external acoustic meatus causes a salivatory reflex at the tympanic nerve.

Pressure regulation role
The nerve of the carotid sinus (of Hering) is a pressure regulator. The carotid reflex is char-
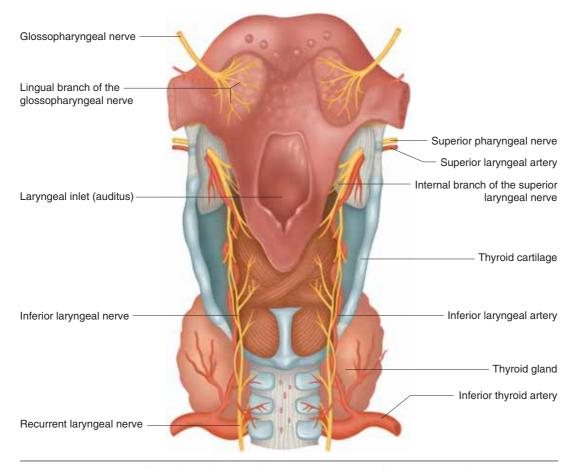
Fig. 21.4 Terminations of the glossopharyngeal nerve (posterior view of the larynx and the tongue).

acterized by hypotension and a slowing of the heart rate provoked by pressure at the carotid. For this reason, a blow to the neck, at the place where the ascending carotid artery forks, can cause syncope.

21.2.2 Clinical notes

Paralysis

Isolated lesions of the glossopharyngeal nerve or its nuclei are rare and do not cause handicap. The associated paralysis manifests as temporary impairment of the sense of taste. There is loss of taste of the posterior third of the tongue, and the gag reflex is absent on the side of the lesion. However, isolated ageusia is ignored by the patient and it is only when both IV and X are affected that lasting dysfunction arises.

Lesions of the glossopharyngeal nerve are usually accompanied by signs revealing the involvement of adjacent nerves (vagus and accessory). This is known as jugular foramen syndrome.

Neuralgia of the glossopharyngeal nerve

Glossopharyngeal neuralgia, or tic douloureux, is uncommon and its cause is mysterious. Pain is experienced in the amygdala tonsil region, in the Eustachian tube and in the ear (external acoustic meatus). The sudden intensification of pain is of a burning or lancinating nature. These paroxysms of pain are often initiated by swallowing, coughing, talking, protruding the tongue, and contact with the palatine tonsil, often during eating.

185
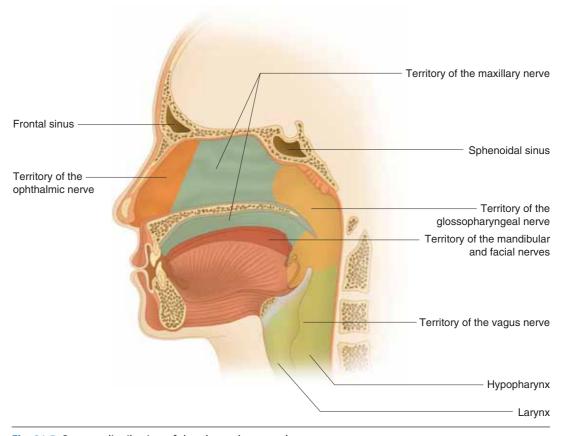
Fig. 21.5 Sensory distribution of the glossopharyngeal nerve.

Notes on the tongue

- **Flavor**. We read that the sense of taste is the simplest of our five senses, as if flavors were only sweet or bitter (Fig. 21.6). We know that the tongue detects innumerable flavors as a function of a person's innate and acquired taste aptitudes. As with all the senses, the sense of taste is dependent on the others, particularly that of smell, and it is simplistic to over-individualize its function.
- **Muscles**. Our tongue is extremely agile and mobile. Seventeen muscles give it this marvelous mobility — it's a record! There is no other organ in the human body capable of such movement, in all directions.
- **Nerves**. Innervation comes from the trigeminal (lingual nerve), glossopharyngeal, vagus and hypoglossal nerves. They supply

fibers for motor function, as well as for vasomotor function. Branches from the sympathetic cervical system are adjoined to these nerves. All these nerves allow the tongue to recognize the tastes and textures of food. In addition to sensory receptors, the corpuscles of Langerhans, Pacini and Krause are found. The tongue has the ability to judge the consistency, temperature and shape of everything it finds in the mouth.
- **Erogenous zone**. For both animals and humans, the tongue has an erogenous role in the transmission and reception of sexual information. The tongue's sensory receptors are linked directly to the limbic centers and the hypothalamo-pituitary axis.

Epiglottis

Lingual tonsil

Palatine tonsil

Bitter

Tart

Salty

Sweet

Vagus nerve

Glossopharyngeal nerve

Lingual branch of the trigeminal nerve

Chorda tympani (VII)

A

Mandibular nerve

Geniculate ganglion

Facial nerve

Chorda tympani

Lingual nerve

Glossopharyngeal nerve

B
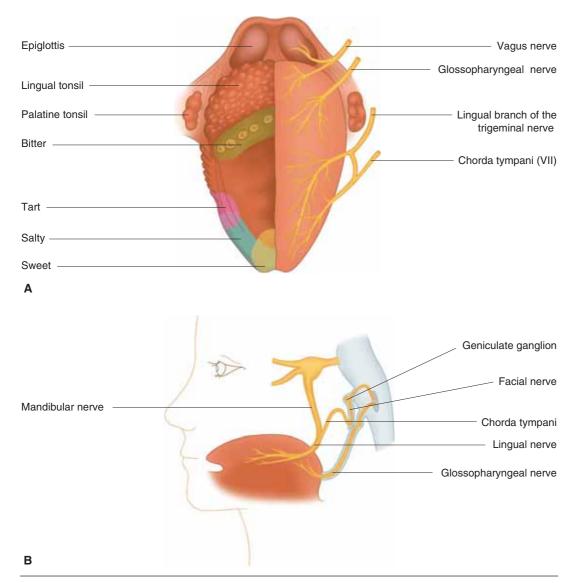
Fig. 21.6 Different taste locations and nerves.
A: View from above. B: Sagittal section.

21.3 MANIPULATION

21.3.1 Jugular foramen

Remember that the glossopharyngeal nerve is the most anterior and medial of the nerves transiting through the jugular foramen. Therefore, by employing the cranial technique for opening the jugular foramen, is it possible to have an effect on the inferior ganglion of the glossopharyngeal nerve, which is situated just at the exit of the jugular foramen.

21.3.2 Lingual manipulation

This technique is very promising in cases of glossopharyngeal neuralgia. Ask the patient to stick out the tongue and grasp it using a tissue, so the tongue does not slip from your fingers (Fig. 21.7).
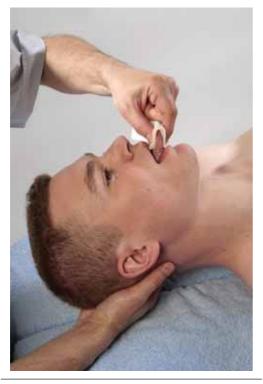
Fig. 21.7 Tongue manipulation technique.

Following the "listening", gently and progressively traction the tongue along the lingual axis. If there is a restriction, the point of the tongue will veer to the affected side. Slightly increase your traction and closely follow the tongue towards the restriction, until there is no more "listening".

21.3.3 At the neck

The glossopharyngeal nerve can be palpated in the middle of the curve (concave anteriorly and cephalic) it follows on exiting the jugular foramen (Fig. 21.8). The nerve is easiest to locate here where it passes anteriorly between the internal carotid artery and the jugular vein, and posterior to the stylopharyngeus muscle. Reference these vessels as landmarks in finding the nerve.

All cranial nerve techniques at the neck must be performed extremely gently. The patient is supine, head turned slightly to the side opposite the nerve to be treated. Palpate the area between the jugular vein and the

Fig. 21.8 Technique for manipulation of the glossopharyngeal nerve at the neck.

internal carotid until you find a small sensitive nerve filament. Using a small superficial sweeping motion, look for the small, somewhat hardened nerve filament and treat it with induction. It is also possible to stretch the nerve by placing it between your two fingers, one cephalad and the other caudal of the nerve.

It is difficult to differentiate the glossopharyngeal nerve from other nerves in this region: for example, the sympathetic, the anastomosis with the facial nerve, the vagus, and the lingual branch of the facial. In any case, when a nerve filament is sensitive and hardened, it must be treated.

21.3.4 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- dura mater (posterior cerebral fossa, tentorium cerebelli)
- temporo-occipital suture
- jugular foramen (posterior foramen lacerum).

21.3.5 Indications

The indications for treating the glossopharyngeal nerve are as follows:

- loss of taste following trauma
- rhinitis, tracheitis
- facial paralysis
- lymphatic problems in the neck
- hypertension (anastomoses with the carotid plexus)
- speech impairment (the tongue plays an important role in elocution)
- libido dysfunction, in association with the olfactory nerve.

Vagus nerve

22

The vagus nerve (X) is by far the longest cranial nerve and covers the most surface area. It sends fibers to the viscera of the neck, thorax and abdomen.

The vagus nerve derives its name from the Latin word *vagus*, meaning vagrant or wanderer. It is practically impossible to follow all of its nerve fibers, and its function remains imprecise and theoretical.

22.1 ANATOMY

22.1.1 Origin

The vagus nerve emerges via eight or ten rootlets from the dorsolateral sulcus of the medulla oblongata, below the glossopharyngeal nerve and above the accessory nerve.

22.1.2 Pathway and useful relationships

In the posterior cranial fossa

The vagus nerve runs laterally and horizontally towards the jugular foramen. It shares the arachnoid sheath with the glossopharyngeal and accessory nerves.

In the jugular foramen

In the jugular foramen the vagus nerve presents an enlargement named the superior ganglion (once known as the jugular ganglion). It crosses the middle part of the foramen, accompanied by the accessory nerve and the posterior meningeal artery (Fig. 22.1).

It is situated:

- behind the glossopharyngeal nerve
- in front of the accessory nerve and the internal jugular vein.

It is separated from the glossopharyngeal nerve by a small fibrous septum called the jugular ligament.

Note: The jugular foramen is a gap set at the posterior end of the petro-occipital suture. It separates into three compartments. The glossopharyngeal lies in the anterior part. The vagus and accessory nerves transmit through the middle compartment. The jugular vein occupies the posterior portion.

In the lateropharyngeal space

After passing through the jugular foramen, the vagus nerve enlarges to form the inferior ganglion (formerly the plexiform ganglion). It then crosses the styloid process and its muscles, medially (Fig. 22.2).

It descends to the root of the neck within the carotid sheath between the internal carotid artery and the internal jugular vein.

In the retrostyloid region it relates:

- anteriorly, to the glossopharyngeal nerve
- posteriorly, to the hypoglossal nerve, the accessory nerve and the superior cervical ganglion
- anteriorly and laterally, to the superficial neck muscles which are contained in the superficial cervical fascia
- posteriorly, to the cardiac nerves and the sympathetic cervical trunk, located on the prevertebral lamina of the cervical fascia
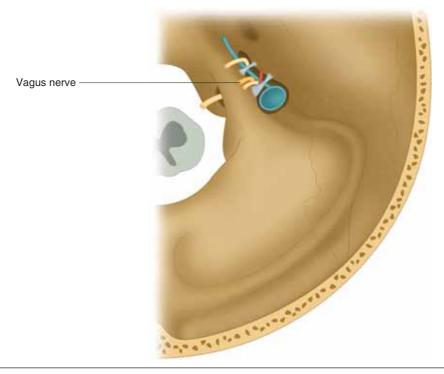
Fig. 22.1 The vagus nerve in the jugular foramen.

- medially, to the visceral compartment of the neck, specifically the lateral lobe of the thyroid gland.

> **Note for manual therapists**
>
> As it is difficult to isolate, accessing the vagus nerve is rather complex and its manipulation requires a great deal of finesse and manual skill. Fortunately, the superior laryngeal branch is easy enough to make contact with and responds well to our techniques.

In the thoracic inlet

The vagus nerve relates:

- anteriorly, to the right brachiocephalic vein
- laterally, to the pleural dome and the right phrenic nerve
- posteriorly, to the subclavian artery as it surrounds the subclavian ansa
- medially, to the common carotid artery.

On the left side, the vagus nerve has an additional relationship with the posterior thoracic duct.

In the thorax

The two vagus nerves descend through the posterior mediastinum. The left runs down the anterior side of the esophagus. In the esophageal hiatus (septum transversum), the right vagus is flattened against the posterior surface of the esophagus.

> **Note for manual therapists**
>
> In their passage through the diaphragm, the two vagus nerves are connected directly to the diaphragm and esophagus by connective tissue filaments that sometimes include small muscular fibers. These fibers can become fibrosed and thereby create vagal irritations of mechanical origin. We believe that this explains the excellent results obtained with manipulations of the hiatal region.

External branch of the accessory nerve

Brachial plexus

Sympathetic thoracic nerve

Parotid gland

Facial nerve

Cervical part of the vagus

Primitive carotid

Bronchus and its divisions

Thoracic part of the vagus nerve

Esophagus

Thoracic artery

Stomach
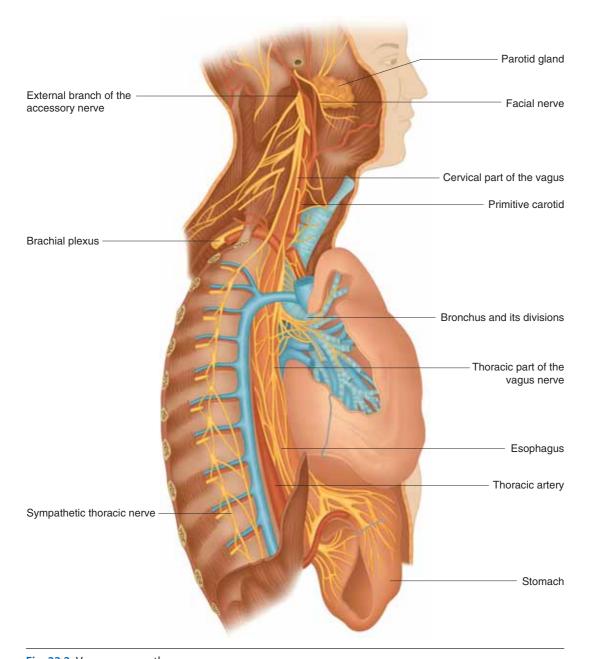
Fig. 22.2 Vagus nerve pathways.

In the abdomen

The right vagus nerve is located behind the esophagus and the stomach where it distributes four or five posterior gastric branches. It sends many branches to the solar plexus and terminates in the right semilunar ganglion.

The left vagus nerve, positioned in front of the esophagus, follows the right border of the cardia of the stomach, supplying branches to the lesser curvature.

Note for manual therapists

- **On the left**. It is through the hiatal zone of the lesser curvature of the stomach that we obtain our best results on the esophago-gastro-vesicular system. We believe that, along with our effect on the contractile fibers of the hiatus, we have an influence on the vagus nerve, particularly the left vagus.
- **On the right**. The right vagus nerve branches to other organs of the abdomen, notably to all the parenchymatous (solid) organs such as the liver, the spleen, the kidneys and even the intestines. Note that the nerve has a privileged and particular connection with the solar plexus.

22.1.3 Collateral branches

Meningeal branch

The meningeal branch arises from the superior ganglion, in the jugular fossa. It is destined for the dura mater of the posterior cerebral compartment. This branch contains fibers from the upper cervical spinal nerves of C1 and C2. Stimulation of the dura mater in the posterior cerebral fossa can cause referred pain in areas innervated by C1 and C2.

Auricular branch

The auricular branch arises from the superior ganglion. It is joined by communicating branches of the glossopharyngeal (IX) and then enters the mastoid canaliculus of the jugular fossa (Fig. 22.3).

It passes through the tympanomastoid fissure to innervate the cranial surface of the ear, the floor of the external acoustic meatus and the adjacent part of the tympanic membrane.

Pharyngeal branch

The pharyngeal branch arises from the upper part of the inferior ganglion. It is made up of filaments from the cranial root of the accessory nerve.

It passes between the internal and external carotid arteries and divides into numerous filaments, which join branches from the sympathetic cervical trunk and the glossopharyngeal nerve to form the pharyngeal plexus.

This plexus, situated on the middle constrictor muscle of the pharynx, innervates the muscles of the pharynx (with the exception of the stylopharyngeus) and the muscles of

Facial nerve
Glossopharyngeal nerve
Vagus nerve
Inferior ganglion of X (jugular)
Spinal nerve
Inferior ganglion of IX (Andersch)
Internal carotid
Internal jugular vein

Facial nerve
Auricular branch of the vagus nerve
Anastomosis uniting the auricular branch of the vagus nerve and homonym branch of the facial nerve
Auricular branch of the facial nerve
Occipital artery
Sympathetic nerve

Fig. 22.3 Auricular branches of the vagus nerve (frontal section, posterior view).

the soft palate (except the tensor muscles of the soft palate).

Carotid sinus branch

This branch arises from the inferior ganglion and, together with rami from the glossopharyngeal nerve and from the sympathetic cervical trunk, forms a plexus going to the carotid body.

Superior laryngeal nerve

The superior laryngeal nerve arises from the inferior ganglion and descends against the lateral wall of the pharynx, along the posterior and then medial surface of the internal carotid artery. It divides into the external and internal laryngeal nerve branches (Fig. 22.4).

The internal branch pierces the thyroid membrane, above the superior laryngeal artery. It is sensory to the laryngeal, the back of the tongue, the epiglottis and the vocal cords. A communicating branch unites with the inferior laryngeal nerve.

The external laryngeal nerve is a mixed nerve and the smaller of the two. It descends with the superior thyroid artery to supply the cricothyroid muscle, the inferior constrictor muscle and the neighboring laryngeal mucosa.

Cardiac branch

One to three superior cervical cardiac branches emerge underneath the inferior ganglion of the vagus nerve and proceed to the cardiac plexus along the length of the internal and common carotid arteries.

One or two inferior cervical cardiac branches emerge at the base of the neck, to the right of the recurrent laryngeal nerve and to the left of the vagal trunk. Branches from the right vagus pass in front of the brachiocephalic artery, while from the left, branches run across the arch of the aorta.

Superior laryngeal nerve (x)

Superior thyroid artery

2.5 cm
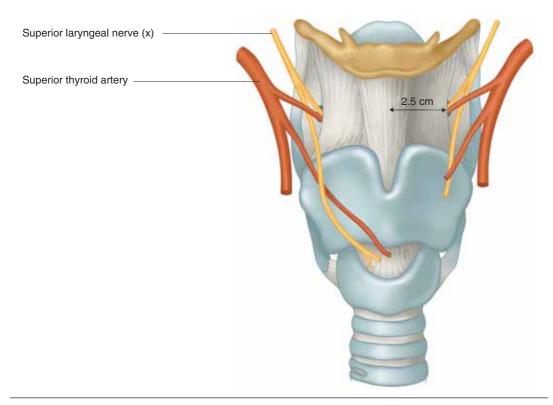
Fig. 22.4 Superior laryngeal nerve.

Recurrent laryngeal nerve

Origin
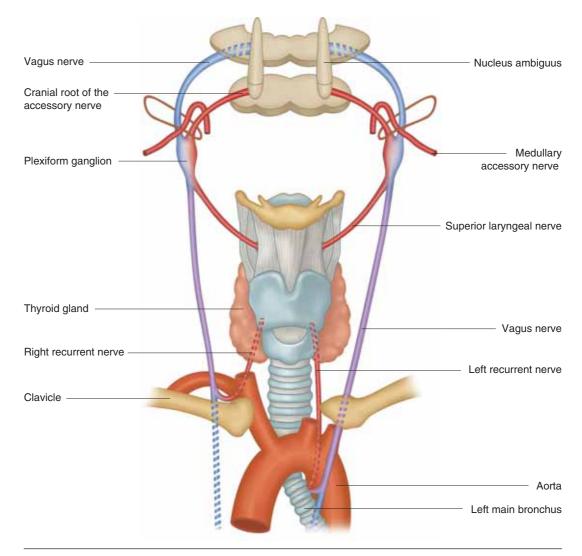
The right recurrent laryngeal nerve branches off from the vagus in front of the subclavian artery. It winds inferiorly around this artery, then posterior to the vessel, and ascends obliquely posterior to the common carotid artery to supply the larynx (Fig. 22.5).

The left recurrent laryngeal nerve arises from the vagus to the left of the arch of the aorta. It curves inferior to the aortic arch and ascends in the groove between the trachea and the esophagus. It relates to the medial surface of the lobe of the thyroid.

In the course of thyroidectomies, risk to the recurrent laryngeal nerves is significant. Such damage can cause aphonia (voice abnormality) and respiratory distress.

Termination

Each recurrent laryngeal nerve ends in the inferior laryngeal nerve. The inferior laryngeal nerve penetrates the larynx, behind the articulation of the thyroid and cricoid carti-

Fig. 22.5 Recurrent laryngeal nerve.

lages. It anastomoses with the internal branch of the superior laryngeal nerve and innervates the laryngeal mucosa underlying the vocal cords.

Collateral branches
The collateral branches of the vagus nerve are as follows:

- tracheal branches
- esophageal branches
- muscular branches for the inferior constrictor muscle of the pharynx and all laryngeal muscles, with the exception of the cricothyroid muscle
- thoracic cardiac branches arising from the right trunk.

Collateral thoracic branches

The vagus nerve distributes to pulmonary and esophageal branches.

Note: The two vagus nerves give off numerous filaments which exchange fibers with each other, and which join with fibers from the sympathetic trunk to form the pulmonary plexus.

From this plexus small branches are distributed to the trachea, the esophagus, the pericardium and the lungs.

Abdominal thoracic branches

Left vagus nerve
Located in front of the cardia, the left vagus nerve distributes fibers to:

- the anterior surface of the stomach
- the lesser omentum
- the liver and its hilum
- the pylorus.

Right vagus nerve
Located behind the esophagus and the cardia, the right vagus sends out fibers to:

- the posterior surface of the stomach
- the solar plexus
- the liver
- the greater splanchnic nerve
- the pancreas
- the duodenum

- the small intestine
- the ascending colon and the hepatic flexure
- the descending colon.

22.1.4 **Connections**

The vagus nerve exchanges fibers with:

- the accessory nerve, in the jugular foramen by way of the jugular ganglion
- the glossopharyngeal nerve, via the intermediary of its inferior ganglion
- the facial nerve, through the auricular branch of the vagus nerve, which can be reached at the posterior inferior wall of the external auditory canal and on the mastoid process
- the hypoglossal nerve, by way of the inferior vagal ganglion
- the large sympathetic, by way of the numerous small filaments, especially for the right vagus nerve
- the two first spinal nerves; this anastomosis is made by the inferior vagal ganglion
- the phrenic nerve, whose anastomosis is mentioned by both Testut (1896) and Lazorthes (1981).

22.2 **PHYSIOLOGY AND PATHOLOGY**

22.2.1 **Functions**

Motor function

- **The pharynx**. The vagus nerve innervates the middle and inferior constrictor muscles, and thus permits the passage of the alimentary bolus. Along with the glossopharyngeal nerve, it controls the swallowing reflex.
- **The soft palate**. The vagus nerve supplies its tone, contributes to suction and prevents fluids from refluxing into the nose.
- **The larynx**. The role of the vagus nerve is paradoxical here; some fibers widen the glottis while others close it. It innervates the motor muscle of the vocal cords, whose subtle interplay is vital to phonation.

Sensory function

- **The auricle of the ear** (posterior part).
- **The external acoustic meatus** (posterocaudal part).
- **The tympanic membrane**. Something touching this can provoke an "ear cough" — a plug of wax irritates filaments of the auricular nerve, causing persistent cough. Ear wax can also be responsible for vomiting or lipothymia (fainting).
- **The mucosa of the pharynx and larynx**. It is here that the cough reflex — whose purpose is to protect the respiratory passages — is initiated.

Autonomic function

Motor role

- The smooth muscles of the alimentary canal, with the exception of the left colon and the rectum, which are innervated by the sacral parasympathetics.
- The smooth muscles of respiration.
- The smooth muscles of glandular tissues.
- The cardiac muscle of the heart.

Interception role

The autonomic nervous system, like the somatic, is a mixed system composed of motor and sensory fibers.

Contrary to accepted opinion, sensory fibers outnumber motor fibers in the vagus nerve. Sensory fibers make up as much as 70–80% of the cervical vagus nerve.

These fibers supply interceptors whose receptors respond to information, often unconscious, about the body's internal environment. Coenesthesia is the conscious sensation of our bodily organs. However, visceral sensations that reach the conscious level — when we feel the organ — indicate that there is a problem.

The neurons supplying the viscera have their cellular bodies in the inferior ganglion of the vagus nerve (plexiform ganglion). Motor fibers do not mingle with sensory fibers in this ganglion.

Note for manual therapists

There is great value in manipulating the jugular foramen, immediately below the location of the plexiform ganglion.

The vagus nerve serves the greater part of the digestive tube, from the upper esophagus to the colon. It shares sensory innervation territory with the splanchnic nerves. Recent studies demonstrate that the sensory vagal fibers terminate in the muscle layers and their mucosa, whereas the splanchnic sensory fibers predominantly innervate the serous layer (Mei, 1998).

Interceptors

There is an abundance of sensory innervation in the walls of the viscera and all layers of their walls. A number of fiber terminations are found:

- the corpuscles of Pacini in the mesentery
- chemoreceptors, such as those in the carotid and aortic bodies
- free terminal arborizations
- baroreceptors.

Physiological signals

Visceral sensory neurons transmit a myriad of physiological signals. These signals are widely diverse, linked as they are to the multiplicity of visceral functions and stimuli.

Many types of information come from the viscera: mechanosensory, thermosensory and chemosensory responses. This rich visceral sensory information arises in different functional receptors: mechanoreceptors, thermoreceptors and chemoreceptors, in particular.

General informational role

The vagus nerves control the majority of visceral activities and also inform the higher centers.

Digestive activity

Numerous activities are capable of generating sensory messages:

- peristalsis
- the opening and closing of sphincters
- segmental filling and expansion

- cytoprotection of the mucosa, vasodilatation
- evacuation
- absorption
- secretion
- immune protection.

Respiratory activity

The nerves are indispensable in the harmonization of intrapulmonary pressures. They provide:

- stretch receptors
- irritation receptors
- juxtapulmonary receptors (sensitive to water pressure in the interstitial pulmonary tissue and to hypercapnia).

Cardiovascular activity

- **Arterial baroreceptors**. These are found in the carotid sinus, in the arch of the aorta, in the common carotid and at the bifurcation of the subclavian artery. They are sensitive to arterial pressure variations. Some have a very low threshold of just 1 mm of mercury! Along with the glossopharyngeal nerve, the vagus serves baroreceptivity of the heart and the great vessels (nerve of Hering). This role is important in controlling arterial pressure.
- **Arterial chemoreceptors**. These are located in the carotid body, the arch of the aorta, the right subclavian artery, the descending aorta and the pulmonary artery. They are sensitive to any fall in blood oxygen levels or a rise in blood carbon dioxide levels.
- **Cardiac receptors**. These are endocavitary baroreceptors and volumetric receptors.

Renal activity

- The arterial mechanoreceptors are sensitive to renal arterial pressure.
- The venous mechanoreceptors are sensitive to renal venous pressure.
- The pyelic mechanoreceptors are sensitive to pressure and the accumulation of urine at the renal pelvis.

- The chemoreceptors are sensitive to:
  — metabolic changes associated with ischemia
  — the chemical composition of urine in the renal pelvis, notably the concentration of ions like sodium or chloride and molecules like urea.

Hepatovesicular activity

- Here the mechanoreceptors are located in the portal circulation; they are sensitive to intraportal venous pressure.
- The glucoreceptors are sensitive to the concentration of glucose in the portal circulation.
- The osmoreceptors are sensitive to osmotic pressure values in the hepatic circulation.
- The gall bladder receptors are mechanoreceptors with a low threshold, especially when responding to even moderate distension of the vesical walls.

Physiopathology of the vagal interceptors

Physiological mechanisms

Interceptors are very much involved in all the physiological mechanisms that ensure visceral function:

- Control of the motor activity of the internal organs (maintenance of muscle tone, peristaltic wave activity and regulation of the exocrine secretions of the alimentary canal).
- Control of endocrine secretions of the viscera and the central nervous system and neurohormonal coordination.
- Coordination of visceral and somatic activities by way of the sympathetic, parasympathetic or sympatico-parasympathetic reflexes. Listed here are several examples:
  — Moderate distension of the intestine induces cardiovascular changes manifesting as increases in heart rate, arterial pressure and volume of inspiration.
  — Stimulation of the visceral afferents can modify somatic muscle activity.

— Distension of the rectum or the vagina provokes contraction of the diaphragm and of the abdominal muscles.
— Stimulation of the vagus nerve influences the amplitude of the digastric and masseteric somatic reflexes.

- Harmonization of the various visceral functions with other bodily functions.
- Postural influence of a greater importance than was once thought. Vagal afferents reach the cerebellum.
- Influence upon higher functions, notably behavior, vigilance, sleep and emotions.

Nociception

Visceral pain is very specific and differs from somatic discomfort. It is vague and diffuse, and can refer out to the somatic body. Visceral nociceptors possess a chemosensitivity vis-à-vis various algogenic substances. Their response to irritation is where the pain messages begin.

Defense phenomena

Visceral afferents are also significantly involved in the manifold mechanisms, conscious and unconscious, which protect the integrity of the organism. Certain familiar reactions like coughing or vomiting serve to preserve an individual's vital functions. The vomiting reflex can come from stimulation originating in the pharynx, the esophagus, the stomach and, more rarely, the intestines.

Other mechanisms have more recently come to light:

- **Protection of the mucous membranes of the tissues**. The vagal afferents play a very important role in cytoprotection of the gastrointestinal mucosa, vis-à-vis hydrochloric acid or alcohol, by intervening with a secretory reflex involving bicarbonate and water.
- **Immune defense**. In the digestive system the vagus nerve constitutes an indispensable link between the local immune response and ensuing central nervous system reactions. There are close relationships between vagal nerve formations and the immune formations (lymph and mastocyte tissues) inside the digestive mucosa. Vagal afferents are very much involved in neuro-immune responses.
- **Trophic function**. Vagal visceral afferents contribute to maintaining the integrity of tissues innervated by them. This trophic function manifests when the afferent fibers are over-stimulated or whenever they function less well or not at all (severance or lesion).

22.2.2 Clinical notes

Peripheral lesions of the vagus nerve

Isolated peripheral lesions of the vagus nerve are rare. They are interesting to look at because they demonstrate the functional complexity of the vagus nerve. Such lesions occur with fractures, invasive tumors, aneurysms, surgery to the neck and carotid dissection.

A lesion of the pharyngeal branches of the vagus nerve results in dysphagia (difficulty in swallowing).

A lesion of the superior laryngeal nerve causes anesthesia of the superior larynx and paralysis of the cricothyroid muscle. The voice is weak and tires easily.

A lesion in one recurrent laryngeal nerve involves the paralysis of the vocal cord. It is responsible for hoarseness and dysphonia (difficulty in speaking). Unilateral paralysis of the recurrent laryngeal nerve is characterized by a sharp bitonal voice, with the vocal cord in the intermediate position.

Bilateral paralysis of the recurrent laryngeal nerves produces a subdued voice, raspy or hoarse, with the vocal cords brought closer together. This aphonia mimics *inspirational stridor* (a harsh high-pitched breathing sound). Because of its longer course, lesions of the left recurrent laryngeal nerve are more frequent than those of the right.

Deficit symptoms of the vagus nerve are often entwined with those of the glossopharyngeal and accessory nerves. At the jugular

foramen, Vernet's syndrome associates with paralysis of the three nerves, resulting in:

- unilateral flaccid paralysis of the palatal, pharyngeal and intrinsic pharyngeal muscles
- hemi-anesthesia of the tongue
- paralysis of the sternocleidomastoid and trapezius muscles.

Bilateral severance of the vagus nerve in the cervical region is quickly fatal due to cardiac arrhythmia and dyspnea. In the thoracic or abdominal regions, by contrast, there are no serious consequences.

Anesthesia of the vagal nucleus results in swallowing difficulties, leading to the aspiration of fluid into the tracheobronchial tree (passageway of the lungs), which is known as Mendelson's syndrome. This is relevant to tracheal intubation during the course of surgical operation.

Vagal disequilibrium

Instabilities come from both the vagal and the sympathetic systems, and it is not always easy to separate them. The principal signs we see during our consultations are indicated below. Some vagal signs are, paradoxically, common to those of the sympathetic system. Who has not seen patients or friends suffer a vagal attack with sudden intense pallor, just before a severe fall? More often than not, it is the fall that causes a problem, rather than the vagal attack.

Here is a summary of the principal signs of vagotonia:

- vasomotor:
  — facial pallor
  — abundant cold sweating
  — cold sensation
  — cyanosis
- psycho-emotional
  — despondency, lassitude, exhaustion
  — sadness, even melancholy
  — withdrawal
  — feeling discouraged
  — tendency towards depression
  — lack of will
  — hypochondria

- digestive:
  — over-production of hydrochloric acid
  — gastro-esophageal reflux
  — nausea
  — colonic spasm
  — sialorrhea
- circulatory:
  — low blood pressure
  — disturbance of cardiac rhythm
  — lipothymia (fainting)
- ocular:
  — miosis
- respiratory:
  — pseudocardiac signs, sensations of stabbing intrathoracic pain, precordial pain, thoracic oppression
  — bronchospasm.

22.3 MANIPULATION

22.3.1 At the ear

Refer to Chapter 25, which is devoted to the ear, for techniques for the auricle (cartilaginous pavilion) and the external acoustic meatus. The ear is a key place for vagus nerve manipulation, especially because of the effects on the superior ganglion and tension on the dura mater of the posterior cerebral fossa.

22.3.2 At the neck

In the carotid trigone

There are so many neurovascular structures in the neck that we will try to simplify the approach to the vagus nerve. It is in the carotid trigone that we can most easily contact and manipulate the nerve (Figs 22.6 and 22.7).

The nerve is found in company with its superior laryngeal branch, between the internal carotid artery and the internal jugular vein. Note that, at the lateral part of the base of the triangle, one can also palpate the accessory nerve. This technique involves very light gliding induction. It is much more of a caress than a compression. This highly reactive area requires the utmost finesse (Fig. 22.8).

Always compare the two sides and apply the technique to the most sensitive zone,
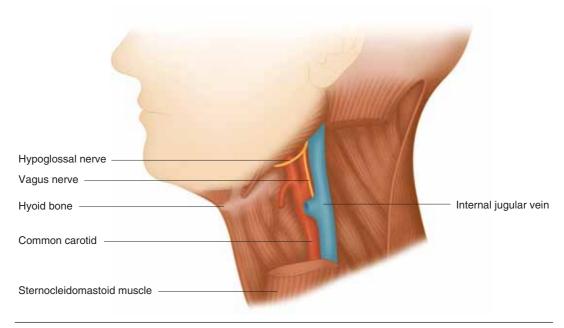
Fig. 22.6 The vagus nerve in the carotid trigone.

feeling for the visco-elasticity of the nerve and modulating the "return" of the nerve during manipulation.

Superior laryngeal nerve

The superior laryngeal nerve is an important collateral of the vagus nerve (Fig. 22.9). From a mechanical perspective, it is directly connected to the inferior vagal ganglion (plexiform ganglion).

Search for the nerve in the lateral thyrohyoid region, where it follows a slightly descending horizontal course. It pierces the thyrohyoid membrane about 1 cm in front of the tubercle of the greater horn of the hyoid bone, lying about 2.5 cm from the median line, to reach the interior of the larynx.

Position yourself at your patient's head, moving just slightly forward of the side opposite the nerve to be treated. With the thumb of your caudal hand, push the hyoid bone in the opposite direction (Fig. 22.10).

With the index finger of your cranial hand, gently examine the tissues along the greater horn of the hyoid bone, searching for the nerve in the thyrohyoid space. The superior laryngeal nerve feels like a small cord parallel, or sometimes slightly caudal, to the greater horn. It is always a little sensitive and occasionally triggers a small cough reflex.

Manipulate the nerve where it perforates the thyrohyoid membrane, a little caudal and in front of the tubercle of the greater horn of the hyoid. Work in induction without overcompressing the nerve. At the end of the maneuver, you may gently stretch the nerve in a caudal direction.

Carotid trigone

The carotid trigone (see Fig. 22.7) is represented by a triangle formed:

- laterally, by the sternocleidomastoid muscle
- medially, by the omohyoid muscle
- cephalically, by the digastric muscle.

The trigone has a caudal summit and a cephalic base. The structures within the trigone are:

- laterally, the internal jugular vein
- centrally, the common carotid artery and its two internal and external branches
- medially, the thyroid body.

Stylohyoid muscle

Posterior belly of the Digastric muscle

Sternal head of the sternocleidomastoid muscle

Clavicular head of the sternocleidomastoid muscle

Anterior belly of the digastric muscle

Anterior belly of the omohyoid muscle

Inferior constrictor muscle of the pharynx

Sternocleidomastoid muscle
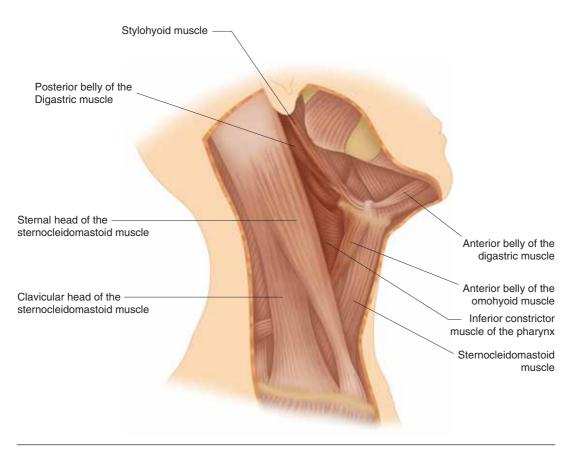
Fig. 22.7 Carotid trigone.
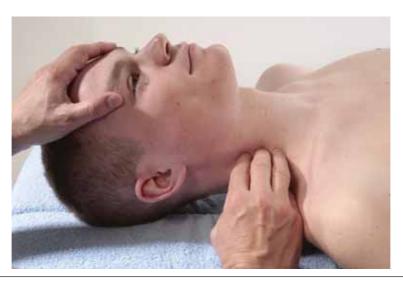
Fig. 22.8 Manipulation of the vagus nerve in the carotid trigone.
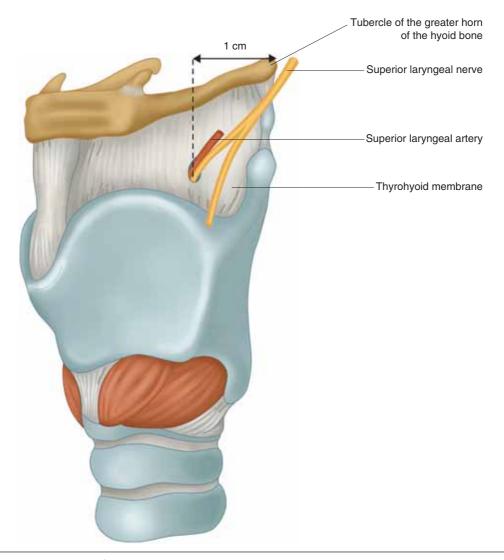
Fig. 22.9 Localization of the superior laryngeal nerve.

Indications

In addition to the general indications for the vagus nerve, its manipulation at the neck addresses these structures in particular:

- pharynx
- trachea
- esophagus
- heart.

We treat many problems of the esophago-cardio-tuberosity junction. In such cases we combine treatment of the vagus nerve at the neck with subcostal mobilization of the hiatal area.

22.3.3 At the hiatal area

As we have seen, the vagus nerves pass through the esophageal aperture of the diaphragm, on either side of the esophagus. Remember that the left-hand side of the liver lies in front of the gastro-esophageal junction. We can obtain an effect on the vagus nerve by mobilizing either the liver or the esophago-cardio-tuberosity junction.

Fig. 22.10 Manipulation of the superior laryngeal nerve.

Liver mobilization

Mobilization of the liver is an indispensable tool. The patient is seated in front of you, in slight kyphosis. Contact the bottom ribs with your palms and draw them anterior and medially in order to release the muscular tensions of the abdomen (Fig. 22.11).

The fingers of your left hand border the left side of the liver, midway between the xyphoid process and a vertical line below the nipple. The fingers of your right hand are placed inside the right phrenico-colic attachment.

Lift the liver several times to stimulate the mechanoreceptors of the coronary and triangular ligaments. Carry out an induction, which virtually always mobilizes the liver in left and right horizontal rotation. Be sure to properly accentuate the movements with your induction.

Hiatal mobilization

Place your middle and index fingers about 5–6 cm below the xyphoid process (Fig. 22.12).

Fig. 22.11 Subhepatic mobilization with a vagal focus.

205

Fig. 22.12 Hiatal mobilization with a vagal focus.

Step 1
Bend the patient forward to gain access to the abdomen without causing discomfort. Direct your fingers posteriorly without attempting to reach the hiatal region.

Step 2
At maximum abdominal penetration achieved without causing discomfort, bring your fingers in the direction of the hiatus (cranial and slightly left), and then draw the lesser curvature of the stomach in a caudal direction.

Note: As with all visceral manipulations, the fingers never travel in a straight line. They should follow, go around and adapt to the various tissues.

As in surgery, there is a true path or approach to the organs, imprinted with finesse and respect for the tissues.

Indications

Gastralgia and ulcers
In combination with visceral manipulations, manipulations of the vagus nerve bring relief to patients who suffer from stomach ailments. However, we must guard against an overly simplistic view of things, as several past experiences have shown us how complex vagal organization is. It is interesting to note some disappointing results following vagal neurotomies.

Ulcer surgery
Very popular in the past, ulcer surgery is now performed less frequently due to the progress made by the pharmaceutical industry and also because of its lack of positive long-term results.

The vagus nerves were more or less resected at the level of the cardia to obtain a decrease in hydrochloric acid. Even if the effect on the pain was very rapid, such resection caused considerable digestive problems. In addition, due to the interaction of nerve filament anastomoses, the hyperacidity gradually reappeared.

In any case, surgical resection of the vagus nerve does not resolve the psycho-emotional conflicts experienced by the patient; nor does it erase the cortical representation of the organ — if it were only that simple!

Helicobacter pylori
Two Australian researchers received the Nobel prize for proving the role of *Helicobacter pylori* in stomach ailments (2005). For a long time, physicians had been noticing a reduction in gastric pain following courses of antibiotics prescribed for other reasons.

Without denying the role of *Helicobacter*, we might still ask the following question: Why do these bacteria appear and proliferate in certain individuals? Simplistically, we know that some mushrooms grow in certain parts of the forest and others grow nearby. It is certainly the same for pathogenic germs that require a particular genetic, metabolic, hormonal and emotional terrain.

We do not deny the value of medical treatment, but it is certainly not the only approach to the problem.

Autonomic equilibrium
For too many people analyzing the nervous system have attempted to separate the sym-

pathetic and parasympathetic systems into a stimulating or a calming category. This simplistic division is not possible, as, interestingly enough, their actions are often opposing. For example, the sympathetic system stimulates the heart and relaxes the stomach, whereas the vagus nerve does the inverse.

However, it is through the *balance* of their actions that the organism is able to function harmoniously. When one system dominates or is dominated by the other, dysfunction and sometimes illness appear.

Our role is not to try to have a precise effect on one or the other, but to provide information to the nervous system in order that it may self-regulate. We return to Andrew Taylor Still's concept, according to which the organism is well able to care for itself. Our first responsibility is to free it of all mechanical constraints, whether these be in the skull, the neck, the thorax or the abdomen. It is through the central nervous system and the reflexes which follow our treatment, that our actions have an effect.

22.3.4 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- dura mater (posterior cerebral fossa, tentorium cerebelli)
- occipitotemporal suture
- jugular foramen.

Accessory nerve

23

The accessory nerve (XI) is a motor nerve whose roots arise from the medulla and the cervical column, and pass together through the jugular foramen. The nerve terminates in the vagus nerve, and the sternocleidomastoid and trapezius muscles.

23.1 ANATOMY

23.1.1 Origin

The accessory nerve has a double origin, being formed from the union of medullary and spinal cord roots:

- the lateral side of the medulla
- the lateral side of the spinal cord in front of the posterior spinal roots.

23.1.2 Pathway

Cranial root

Four or five small rootlets emerge from the lateral side of the medulla and run anteriorly and laterally to the jugular foramen.

Spinal root

The caudal fibers of spinal origin emerge superior to the fourth posterior cervical root, while the cephalad fibers emerge inferior to the first posterior cervical root (Fig. 23.1).

These fibers unite to form a nerve that enters the posterior skull through the foramen magnum.

Common trunk

The common trunk of the accessory nerve passes through the jugular foramen and runs in the retrostyloid space.

It divides into two terminal branches:

- a medial branch which joins the vagus nerve
- a lateral branch which descends posteriorly to pierce the sternocleidomastoid muscle, crosses the posterior triangle of the neck and terminates in the trapezius muscle.

23.1.3 Useful relationships

In the foramen magnum

The accessory nerve enters the lateral part of the foramen magnum, posterior to the hypoglossal nerve and the vertebral artery, and inferior and anterior to the cerebellum (Fig. 23.2).

> **Note for manual therapists**
>
> The vertebral artery technique, performed in the sitting position, allows us to have a promising effect on the accessory nerve. The spinal root of the latter crosses the vertebral artery as it ascends towards the foramen magnum in a cephalad, anterior and medial direction.
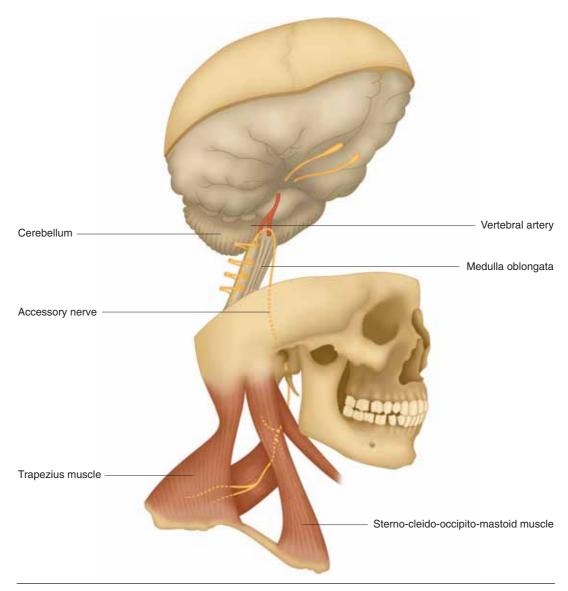
Fig. 23.1 Pathway of the accessory nerve.

In the jugular foramen

In the jugular foramen the accessory nerve is located behind the superior ganglion of the vagus nerve, to whose surface it is closely bound (Fig. 23.3). It lies in front of the lateral sinus, and on crossing the jugular foramen, forms the internal jugular vein.

23.1.4 **Anastomoses**

With the first two cervical nerves

The accessory nerve exchanges fibers with the first two cervical nerves (principally the first). Most authors allow that these fibers unite for a short distance, and that this is more like a brief adherence than a true anastomosis (Fig. 23.4).
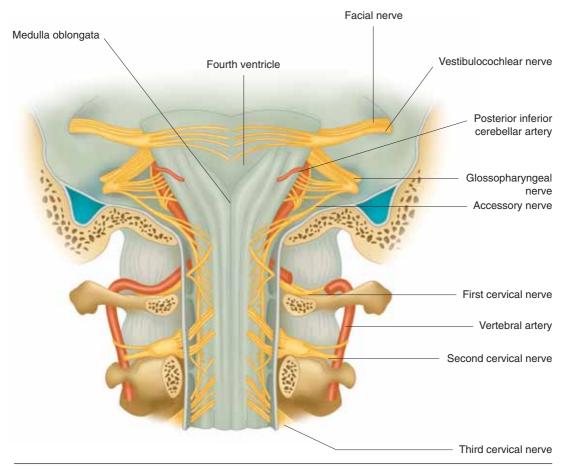
Fig. 23.2 The accessory nerves in the foramen magnum.

Note for manual therapists

In treating the suboccipital nerves, we also affect the accessory nerve.

With the vagus nerve

In the jugular foramen, the accessory nerve is connected by one or two filaments with the superior ganglion of the vagus nerve.

23.1.5 **Terminal branches**

As it exits the jugular foramen, the accessory nerve separates into medial and lateral accessory branches.

Medial branch

Short in length, the medial branch is formed from fibers of the cranial root. It runs anteriorly and medially along the lateral and cephalad surface of the inferior vagal ganglion. It distributes to:

- the superior constrictor muscle of the pharynx via the pharyngeal branch of the vagus nerve
- the cardiac plexus and the heart via the cardiac branch of the vagus nerve.

Lateral branch

More important than the medial, the lateral branch is formed by the fibers of the spinal

Accessory nerve
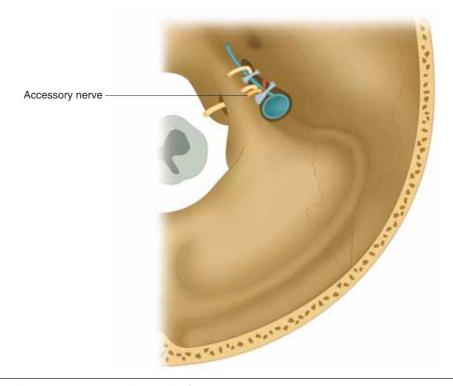
Fig. 23.3 The accessory nerve in the jugular foramen.

root. At its exit from the jugular foramen it runs in a caudal, posterior and lateral direction. It perforates the sternocleidomastoid muscle, crossing the posterior triangle of the neck to terminate at the trapezius.

> **Interesting note**
>
> Before penetrating the sternocleidomastoid muscle, the accessory nerve, together with fibers of the second and third cervical nerves, forms a small plexus within the thickness of the muscle. Accessory filaments interchange fibers with the rhomboid and the angular nerve.

23.1.6 Distribution

Nerves to the sternocleidomastoid

The sternocleidomastoid muscle receives filaments from the accessory nerve, the third cervical nerve and sometimes from the second cervical nerve.

> **Note for manual therapists**
>
> With congenital torticollis in newborns, the entry and exit points of the accessory nerve in the sternocleidomastoid muscle must be sought and treated. These are two excellent trigger points.

Nerves of the trapezius

The accessory nerve gives off two or three branches to the trapezius muscle. This muscle also receives filaments from the fourth cervical nerve, and from the arch uniting the third and fourth cervical nerves.

23.1.7 Key topographical points

Trapezius

The accessory nerve reaches the trapezius muscle at:

- its anterior edge, *or*
- its deep surface, three finger widths behind the clavicle, between the

Auricular nerve

Glossopharyngeal nerve

Vagus nerve

Accessory nerve

Pharyngeal nerve

Laryngeal nerve

Anastomosis towards the hypoglossal nerve
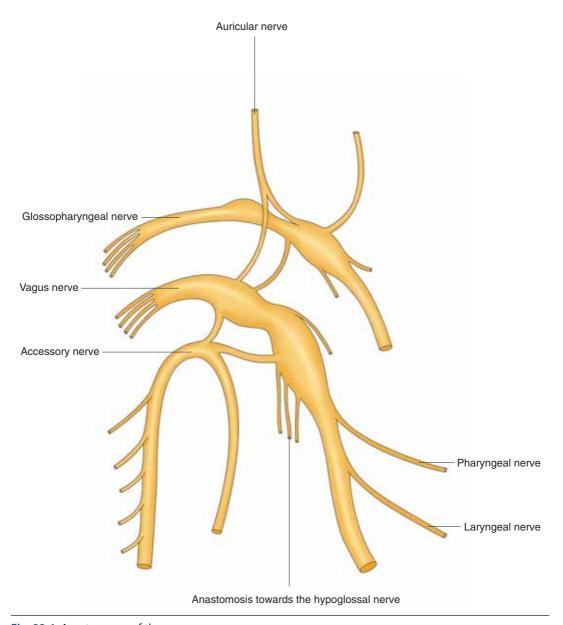
Fig. 23.4 Anastomoses of the accessory nerve.

acromioclavicular joint and the lateral side of the neck (Fig. 23.5).

Sternocleidomastoid

The accessory nerve pierces the upper part of the sternocleidomastoid muscle 3 cm from the mastoid, at the angle of the mandible and at the height of the third cervical (Fig. 23.6).

It exits at the posterior border, 5 cm below the mastoid, on a horizontal line passing through the hyoid bone at the level of the fourth cervical.

Retrostyloid space

The accessory nerve penetrates the sterno-cleidomastoid muscle between the transverse

213
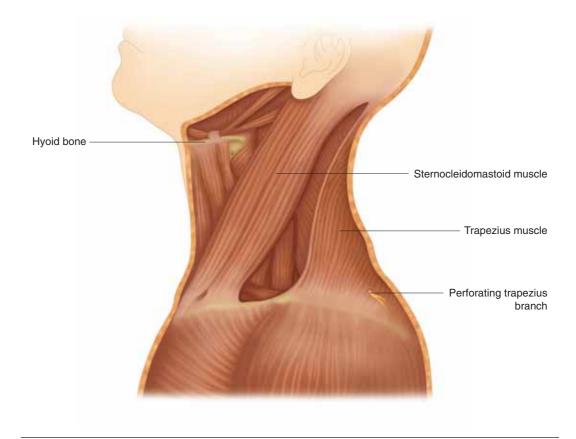
Fig. 23.5 The trapezius muscle: key point of the accessory nerve.

process of the atlas (3 finger widths below the mastoid tip) and the posterior belly of the digastric muscle.

23.2 PHYSIOLOGY AND PATHOLOGY

23.2.1 Functions

Accessory nerve: cranial root

The motor functions of the cranial accessory nerve root are mingled with those of the vagus with regard to motor innervation of the pharynx, the palate and the larynx.

Accessory nerve: spinal root

The spinal accessory nerve root is the principal nerve for rotation of the head, the sterno-cleidomastoid and trapezius muscles receiving fiber contributions from the second, third and fourth cervical nerves.

Note that the two sternocleidomastoid muscles serve as accessory muscles of respiration.

23.2.2 Clinical notes

Peripheral lesions

Lesions of the cranial root cause swallowing and speech problems.

Lesions of the spinal root cause paralysis of the sternocleidomastoid and trapezius muscles.

- Paralysis of the sternocleidomastoid muscle is evident from the absence of the normal ridge shape in the neck and produces an impairment of rotary
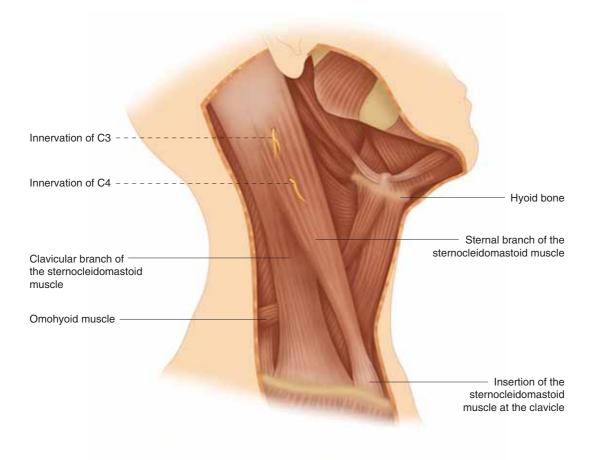
Fig. 23.6 The sternocleidomastoid muscle: key point of the accessory nerve.

movements of the neck to the healthy side.

- Paralysis of the trapezius produces weakness in shrugging movements of the shoulders, a lateral winging of the scapula, and a forward projection of the clavicle.

Avellis syndrome

This syndrome involves injury to the medial branch of the accessory nerve and to the nucleus ambiguus of the vagus nerve. It presents as:

- hemiparalysis and anesthesia of the laryngopharynx on the side of the lesion

- dysarthria
- dysphagia.

23.3 MANIPULATION

23.3.1 Nerves perforating the trapezius muscle

Locate the perforating branch of the accessory nerve, about 2 cm from the anterior border of the trapezius muscle, midway between the acromioclavicular joint and the vertical cervical part of trapezius.

This point measures 2–3 mm in diameter, and is hard and sensitive, sometimes even hypersensitive. Its palpation can produce

215

what is called in medicine an exquisite pain!

Technique

The patient is supine. Place the palm of one hand under the patient's occiput to stabilize the neck. Place the thumb of the other hand on top of the patient's shoulder, on the zone of the perforating nerve. The palm covers the top of the shoulder (Fig. 23.7).

Step 1

Delicately compress the painful point, release a little and carry out the induction technique.

Step 2

While maintaining contact with the painful point, as above, gradually push the top of the shoulder laterally and caudally. This keeps the perforating branch of the accessory nerve under perpetual tension. Ideally, perform this maneuver during the cranial expansion phase.

Note: This technique should "flirt" progressively with the pain. Too strong a touch will increase pain and inhibit your effect.

23.3.2 Nerves perforating the sternocleidomastoid muscle

There are two important points to manipulate on the sternocleidomastoid muscle:

- one at the level of the mandibular angle (facing C3)
- one at the level of a horizontal line passing through the hyoid bone (facing C4).

These two points are sensitive, but less so than the one at the trapezius.

Technique

Step 1

Place your thumbs on either side of the sensitive point. Exert light traction and follow the "listening" (Fig. 23.8).

Step 2

After carrying out the first phase, perform the induction technique directly on the sensitive emerging nerves.

23.3.3 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mech-

Fig. 23.7 Manipulation of the perforating nerve at the trapezius.
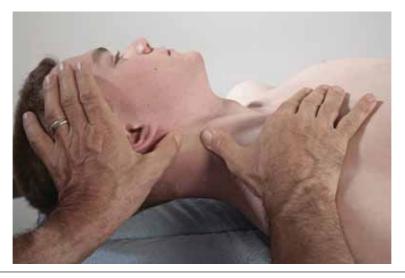
Fig. 23.8 Manipulation of the nerves at the sternocleidomastoid muscle.

anism and the elements of the primary respiratory mechanism, especially in these areas:

- dura mater (posterior cerebral fossa, tentorium cerebelli)
- temporo-occipital suture
- jugular foramen.

23.3.4 Significance of accessory nerve manipulations

Who has not had a tight trapezius muscle? In addition to general techniques on the trapezius that soothe and comfort, we are able to make contact with the accessory nerve and precisely release these perforating points.

We will look at some indications for manipulating the accessory nerve that go beyond the simple indications for contraction of the trapezius and sternocleidomastoid muscles.

As we are treating emerging branches of a cranial nerve, the manipulations have an effect on intracranial structures and on the primary respiratory mechanism.

As described below, the trapezius muscle has subtle implications beyond its familiar motor function.

Erect posture

Because of our vertical stance, we humans are able hold our head up, eyes horizontal, and can "face up" to what is happening in front of us. The accessory nerve plays, albeit indirectly, a central proprioception role. It is thanks to this nerve that we can keep our head up while simultaneously coordinating the movements of our eyes and head.

Territoriality

Individuals in good shape carry themselves erect and face the world head-on. This position allows us to occupy our space in relation to ourselves, to others and to our environment. In the upright position our sensory receptors are perfectly oriented and able to make sense of what is happening around us.

For our forebears, this capability was vital: a question of life and death with regard to the dangers that threatened them. Today, we no longer experience the same dangers, but we have created a difficult society in which we must keep our place at all costs. The accessory

nerve is an indispensable element in the healthy maintenance of our territory, through the role it plays in the carriage of the head and the consequences this has for the proprioceptive system.

A reflection of the soul

Upon release of tension on the accessory nerve, and with it trapezius and sternocleidomastoid muscle strain, we feel an immediate sense of well-being. Subsequently, this allows us to lay down the burden we perennially carry on our shoulders, which is sometimes quite heavy! The giant Atlas carried the world on his head; in our case, we often carry the weight of our little world on our shoulders . . .

Autonomic system

The accessory nerve forms an anastomosis with the vagus nerve, more precisely with its superior ganglion, and via this intermediary connects with the entire autonomic system. Thus manipulation of the accessory nerve allows us to have an effect on the organs.

This is a reciprocal relationship. An organ in difficulty affects the accessory nerve and the accessory nerve has an effect on the organ.

The pharynx, the larynx and the heart all receive fibers from the inferior vagal ganglion, which itself receives a contribution from the medial branch of the accessory nerve. Manipulation of the accessory nerve is particularly indicated for all functional problems involving the organs of the neck and the thorax.

Hypoglossal nerve

24

The hypoglossal nerve is the motor nerve to the tongue and muscles of the infrahyoid region. It is an important nerve for mastication, suction, swallowing and speech.

24.1 ANATOMY

24.1.1 Origin

The hypoglossal nerve arises as a series of a dozen or so rootlets from the anterior surface of the medulla oblongata.

24.1.2 Pathway

Cranial pathway

Several rootlets pass towards the anterior condylar foramen (hypoglossal canal) and collect into two groups: one cephalad and the other caudad. They perforate the dura mater opposite the hypoglossal canal and join together after passing through it. Sometimes the canal is divided into two apertures and sometimes not.

Extracranial pathway

After emerging from the canal, the hypoglossal nerve passes laterally with a caudal inclination and crosses the sternocleidomastoid muscle (Fig. 24.1). It then inclines superiorly along the posterior border of the mylohyoid muscle and continues anteriorly to enter the tongue (Fig. 24.2).

24.1.3 Useful relationships

In the cranium

The hypoglossal rootlets run between the vertebral artery and the posterior inferior cerebellar artery. They are contiguous at the dural orifice of the vertebral artery.

> **Note for manual therapists**
>
> Our treatment of the vertebral artery has an effect on the hypoglossal nerve in two ways: through the direction of the nerve fibers themselves and via the dura mater as intermediary.

At the hyoid

The hypoglossal nerve is located between the greater horn of the hyoid bone below, and the intermediate tendon of the digastric muscle above. It then ascends towards the caudal surface of the tongue between the mylohyoid laterally, and the hyoglossus and genioglossus muscles medially.

24.1.4 Anastomoses

The hypoglossal nerve communicates with many nerves (Fig. 24.3):

Facial artery and vein

Mylohyoid muscle

Lingual nerve

Stylohyoid muscle
Digastric muscle
Hypoglossus nerve

Internal carotid

Sublingual gland

Hyoglossus muscle

Mylohyoid muscle

Descending branch of
the hypoglossal nerve

Common trunk receiving
thyroid, tongue and facial veins
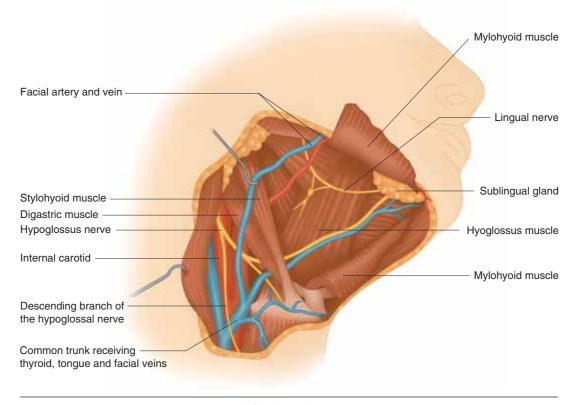
Fig. 24.1 The hypoglossal nerve at the level of the hyoid.

Mylohyoid nerve

Mylohyoid muscle

Hyoid bone

Anterior belly of the
digastric muscle

Mandibular angle

Hypoglossal nerve
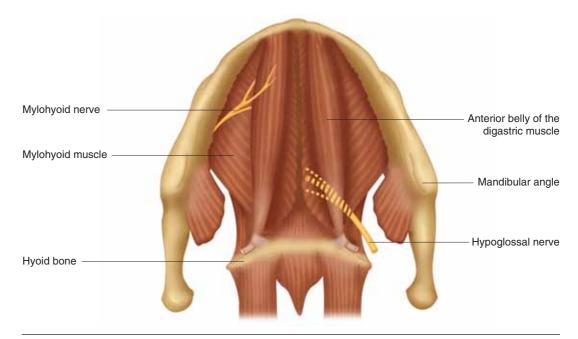
Fig. 24.2 The hypoglossal nerve on the floor of the mouth.
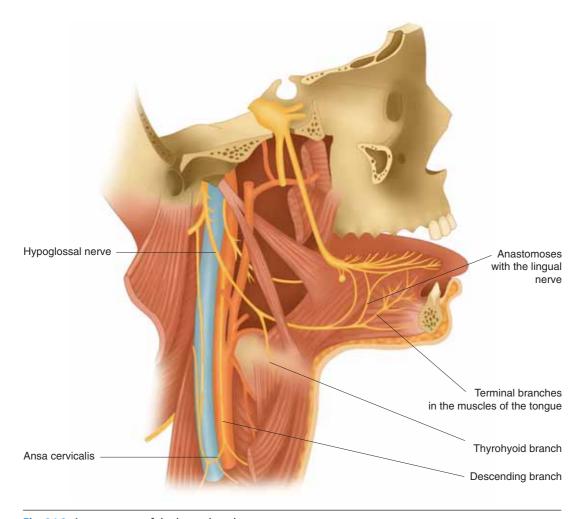
Fig. 24.3 Anastomoses of the hypoglossal nerve.

- **Vagus**: Several filaments pass between the hypoglossal nerve and the inferior ganglion of the vagus nerve.
- **Sympathetic trunk**: Via a filament at the exit of the condylar foramen destined either for the superior cervical ganglion or for the carotid filament of the same ganglion.
- **Cervical**: Opposite the atlas, the nerve is joined by a filament coming from the loop connecting the first and second cervical nerves.
- **Trigeminal** (lingual nerve): Via a nerve loop formed by the lingual and hypoglossal nerves.

24.1.5 **Collateral branches**

Meningeal branch

The meningeal branch leaves the hypoglossal nerve as it passes through the anterior condylar canal (hypoglossal canal). These fibers distribute to the occipital dura mater and the posterior occipital sinus. They often exchange fibers with the vagus, the trigeminal (lingual nerve) and the first cervical nerve.

Descending branch

The descending branch divides from the hypoglossal nerve where the latter crosses the

221

carotid artery. Here it sends an anastomosis to the cervical plexus and forms the ansa hypoglossi.

From this loop, fibers leave for the omohyoid, sternohyoid and sternothyroid muscles.

From the sternohyoid muscle, another filament has been described as descending into the thorax and joining the phrenic nerve and cardiac plexus.

Terminal branches

The terminal branches leave from the anterior border of the hyoglossus muscle to surface on the intrinsic muscles of the tongue.

24.2 PHYSIOLOGY AND PATHOLOGY

24.2.1 Functions

The hypoglossal nerve ensures the mobility of the tongue. It supplies all of the intrinsic muscles, and three of the four extrinsic muscles (genioglossus, styloglossus and hyoglossus).

24.2.2 Clinical notes

Injury to the hypoglossal nerve paralyzes the ipsilateral half of the tongue. Particularly with peripheral lesions, the tongue atrophies over time and appears shrivelled and wrinkled. The patient has trouble with mastication, swallowing is difficult and speaking is slow.

- Ask the patient to stick out the tongue; observe that the tip deviates laterally. When protruded, the tongue deviates:
  — to the side opposite the lesion when there are supranuclear problems
  — to the side of the lesion in cases of peripheral or nuclear lesions.
- Ask the patient to push the tongue laterally against the inside of the cheek while resisting this action with your fingers against the outside of the cheek.

24.3 MANIPULATION

24.3.1 Submandibular

Location

Follow the anterior border of the sternocleidomastoid muscle up to the angle of the mandible. After moving the muscle posteriorly, you will find the hypoglossal nerve inside the external carotid artery.

We can follow the nerve as far as the greater horn of the hyoid bone, being careful not to mistake it for the lingual nerve (trigeminal, V3).

Direct technique

Ask the patient to turn the head to the side opposite the nerve being treated. With the index finger of your chin-contact hand, locate the nerve in the posterior part of the floor of the mouth, just in front of the mandibular angle and behind the mylohyoid muscle (Fig. 24.4).

With the index finger of your occipital hand, make contact with the nerve just posterior to the mandibular angle.

Place the nerve under slight tension between your two finger contacts and follow the "listening" of the tissues, until you obtain a release of the nerve.

Indirect technique

The hypoglossal nerve reacts just as well to glide–induction techniques as it does to compression–inductions.

Make light contact with a sensitive point along the hypoglossal nerve and work the sensitive area superficially with induction (Fig. 24.5).

The manipulation we have learned for the vertebral artery can also be performed, as by it we can achieve an effect on the glossopharyngeal, vagus and accessory nerves (see Ch. 9).

Combined technique

Due to its connections with the ansa cervicalis, the hypoglossal nerve is important to manipulate in combination with the cervical

Fig. 24.4 Direct manipulation of the hypoglossal nerve.

Fig. 24.5 Indirect manipulation of the hypoglossal nerve.

spine to treat fixations of the dorsal branches of C2 and C3.

Ask the patient to turn the head slightly to the side opposite the nerve you are treating. With the index finger of the contralateral hand, locate a sensitive bud on the posterior cervical branch and exert very light compression on it (Fig. 24.6).

With your other hand, make contact with the hypoglossal nerve, just outside the greater horn of the hyoid bone. Reinforce with the index finger and contact with the middle finger.

Treat by performing double induction on each point of contact until you perceive a complete release of the two contacts.

Fig. 24.6 Combined manipulation of the hypoglossal nerve.

24.3.2 Key points for the craniosacral mechanism

To make your treatment more global in its effects, always evaluate the craniosacral mechanism and the elements of the primary respiratory mechanism, especially in these areas:

- dura mater (posterior cerebral fossa, tentorium cerebelli)
- occiput: occipital condyle, foramen magnum
- atlanto-occipital and atlanto-axial articulations.

24.3.3 Indications

Motor control of the tongue

The hypoglossal nerve is the principal motor nerve of the tongue. Remember that the tongue moves through the combined action of 17 muscles, whose balance and coordination must be perfect to guarantee the harmony of the mouth and teeth.

Look for abnormal tension on the hypoglossal nerve in cases of:

- prognathism
- malocclusion
- difficulty swallowing
- elocution dysfunction.

It is a good idea to check the hypoglossal nerve during the course of orthodontic treatments of long duration, or when there is delayed progress.

Functional dysphonia

The tongue is the key structure for speech articulation. Posteriorly, it is attached to the hyoid bone and the upper part of the larynx. All disturbances of tongue mobility, whether of psychological or motor origin, can have repercussions for phonation and vocal expression.

Surgical sequelae

Be on the lookout for tension of the hypoglossal nerve following neck surgery, especially in areas of scarring.

Sequelae to trauma

Evaluate the hypoglossal nerve following cranial and cervical trauma.

Fixations of the dura mater at the posterior cranial fossa and the vertebral canal

We refer to indications regarding:

- fixations of the dura mater at the cranial base around the foramen magnum
- severe fixations of the occipital condyles in infants and children
- vertebrobasilar insufficiency.

Vertigo and instability

Due to the sensory role played by the hypoglossal nerve with regard to the meninges of the posterior cerebral fossa and on the basilar arteriovenous system, we can have an influence on problems of vertigo and loss of balance.

Fixations of the upper cervical nerve roots

In a severe fixation at the first three cervical nerve roots (C1, C2 and C3) manipulation of the hypoglossal nerve is of great significance. The ansa cervicalis represents an important connection between the cervical plexus and the hypoglossal nerve.

Ear

We will endeavor to simplify the complex anatomy of the ear, while retaining what is important for its physical exploration and osteopathic treatment.

For the inner ear, refer to Chapter 20, which deals with the vestibulocochlear nerve.

The ear is described in three parts:

- the external ear, which receives sounds
- the middle ear, which transmits and amplifies sounds
- the internal ear, an organ of reception, perception and bodily equilibrium.

25.1 EXTERNAL EAR

25.1.1 Auricle (pinna)

The auricle collects, directs and channels sounds towards the auditory canal (Fig. 25.1).

Medial surface

The elevations on the medial surface of the auricle are clearly less remarkable than those of its lateral aspect. The convexity of the concha is its salient feature. It is separated from the cranium by the cephalo-auricular sulcus.

Lateral surface

The particular shape of the auricle is what allows it to fulfill its role of catching sound. We will look in some detail at the irregular form of the cartilage, in whose eminences and depressions we are able to manipulate the terminal branches of some cranial nerves.

At the auricle one finds the helix, the antihelix, the groove of the antihelix, the tragus, the antitragus and the concha. The lobule (ear lobe) is devoid of cartilage, which is perhaps why it is less interesting to manipulate.

- The helix begins in the concavity of the concha at the crux of the helix. Below the helix, formed by the curling of the outer rim itself, is the groove of the helix.
- The antihelix is located between the helix and the concha. At its cephalad part, the antihelix divides and forms the scaphoid fossa (navicular fossette).
- The tragus is a cartilaginous flap projecting at the anterior concha. It is separated from the helix by the anterior groove of the ear.
- The antitragus is opposite the tragus. Anteriorly and caudally, it is separated from the side of the tragus by an indentation of the concha.
- The lobule has no cartilage; it is soft.
- The concha is a trumpet shape; its bottom, directed inside the ear, is continuous with the external acoustic meatus. All around the concha are the four prominences: the helix, the antihelix, the tragus and the antitragus.

Auricular muscles (motor)

Extrinsic muscles

The extrinsic muscles of the auricle are important to localize as they are innervated by the facial nerve. They are three in number: the auricularis superior, auricularis anterior and auricularis posterior (Fig. 25.2).

Helix

Scaphoid fossa
(navicular fossette)

Auricular tubercle (of Darwin)

Anterior incisura of the ear

Antihelix

Concha

Anterior notch

Antitragus

Tragus

Intertragic incisura

Lobule
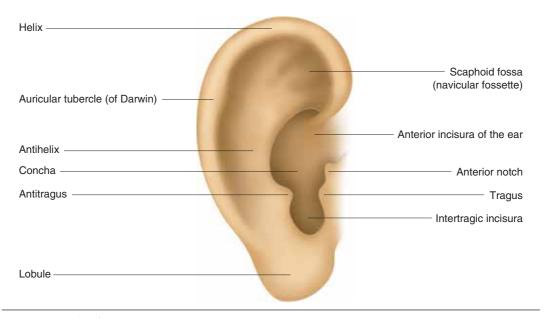
Fig. 25.1 Auricle of the ear.

Superior auricular

Anterior auricular

Helicis major

Helicis minor

Posterior auricular

Antitragus

Tragus
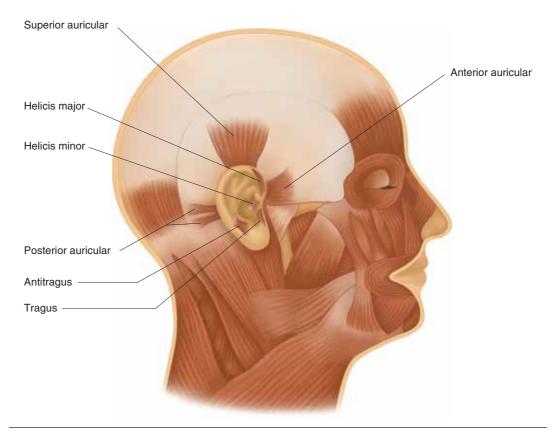
Fig. 25.2 The auricular motor muscles.

Auricularis superior

This muscle is the most developed. Its fibers arise from the epicranial aponeurosis and extend to the medial surface of the auricle, at the convexity of the scaphoid fossa.

Auricularis anterior

This muscle originates at the lateral edge of the epicranial aponeurosis, just superior to the zygomatic arch. Its fibers run posteriorly to insert into the helix and the anterior part of the concha.

Auricularis posterior

This muscle arises from the base of the mastoid part of the temporal bone as two or three fasciculi, which go to the medial part of the concha.

Intrinsic muscles

The intrinsic muscles of the auricle are so named because their insertions extend from one part of the auricle to another. They are six in number:

- helicis major muscle
- helicis minor muscle
- tragus muscle
- antitragus muscle
- transverse muscle
- oblique auricular muscle.

These muscles sometimes exchange fibers with the extrinsic muscles.

Note for manual therapists

In today's world, these muscles have little to do; however, they persist because of the once indispensable role they played in protecting human beings in their environment. Though largely vestigial, they are probably linked on the emotional plane to the fear of being wounded or of dying.

Manipulation of these muscles is valuable, as the small nerve filaments contained in them continue to have a significant influence on the body through reflexogenic connections. Indeed, it is the presence of the auricular muscles that helps explain the highly reflexogenic role of the ear.

Nerves of the auricle

Both motor and sensory nerves supply the auricle (Fig. 25.3).

Motor nerves

The motor nerves to the auricle are as follows:

- It is notably the facial nerve that supplies all the extrinsic and intrinsic muscles. Its ascending posterior auricular branch supplies the posterior and superior auricular muscles.
- The temporofacial branch of the facial nerve receives an anastomosis from the auriculotemporal nerve (V3, mandibular nerve), and supplies temporal filaments to the anterior auricular muscle.

Sensory nerves

Via its auricular branch — and, more exactly, its sensory branch — the vagus nerve passes through the mastoid canal (canaliculus mastoideus) to exit the skull, between the external acoustic pore of the ear and the mastoid process.

This branch divides in two filaments; one forms an anastomosis with the posterior auricular nerve, and the other distributes nerve filaments to the medial surface of the auricle and to the posterocaudal wall of the acoustic meatus (Fig. 25.4).

Detail

The facial nerve is reputed to be solely motor; nevertheless, it receives numerous small sensory filaments by way of anastomosis.

25.1.2 External acoustic meatus

The external acoustic meatus is a canal measuring 25 mm in depth and 6–8 mm in diameter. It extends from the concha to the tympanic membrane. In its walls are numerous secretory ceruminous glands. It is its direction that interests us most, as certain techniques are carried out within this canal.

Auriculotemporal nerve (belonging to the mandibular nerve)

Ramsay Hunt's zone

Auricular branch of the vagus nerve

Posterior auricular nerve (belonging to the facial nerve)

Facial nerve

Great auricular nerve (cervical plexus)
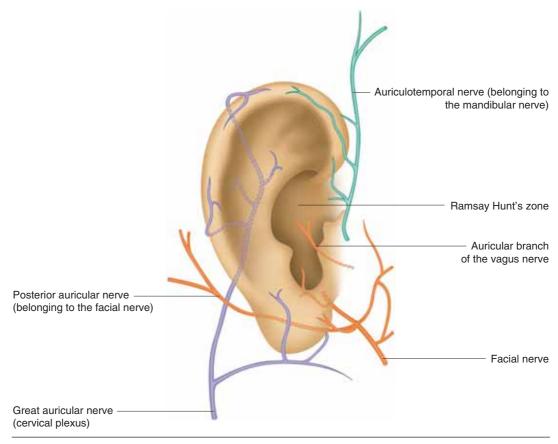
Fig. 25.3 Sensory nerves of the auricle.

Territory of the mandibular nerve

Territory of the vagus and facial nerves

Territory of the cervical plexus
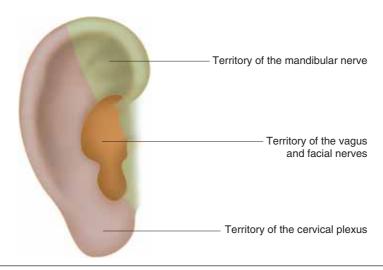
Fig. 25.4 Sensory innervation of the ear.

Direction

The external acoustic meatus is directed in a horizontal oblique, medial and slightly anterior direction. The conduit is not straight but rather sinuous; exploring it in horizontal and vertical sections will give us a better understanding of it.

Horizontal section

The canal leads first anteriorly, then posteriorly and at last anteriorly (Fig. 25.5).

It has two curves and three parts:

- a lateral part, very oblique medially and anteriorly

- a median part, very oblique medially and posteriorly
- a medial part, longer and slightly oblique medially and anteriorly.

The first two of these parts are cartilaginous and the third is carried in the osseous canal of the ear. The cartilaginous part of the meatus measures 8 mm in length and its osseous portion 16 mm.

Frontal section

As far as its middle part, the canal is horizontal; then it follows a long curve which is concave caudally (Fig. 25.6).
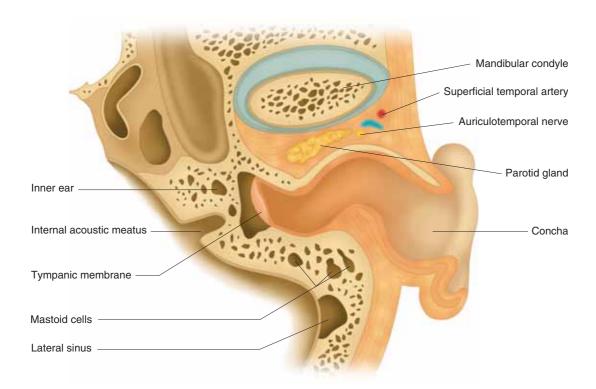
Fig. 25.5 External acoustic meatus (horizontal section).

Squamous part of the temporal bone

Glandular layer

Helix

Auricular cartilage

Scaphoid fossa

Antihelix

Antitragus

External acoustic pore (aperture)

Lobule

Epitympanic recess

Inner ear

Internal acoustic meatus

Round (cochlear) window

Chain of ossicles

Tympanic cavity

Osseous part of the acoustic meatus
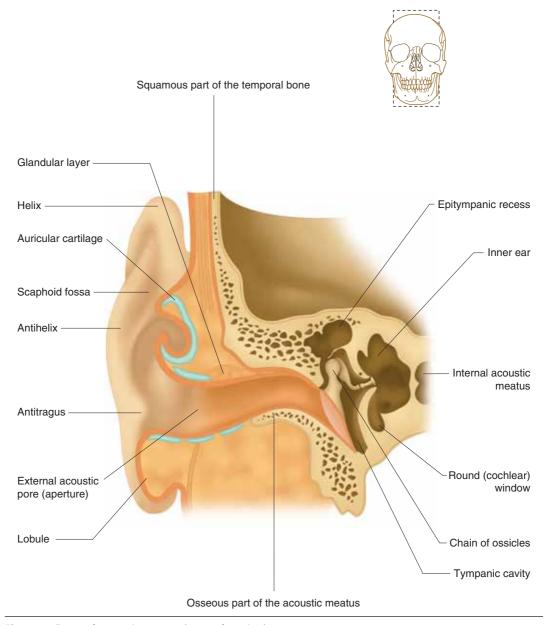
Fig. 25.6 External acoustic meatus (coronal section).

Note for manual therapists

To make penetration of the cotton swab easier, the auricle can be pulled in a cephalad direction and the tragus anteriorly. Because of the obliquity of its plane, the caudal wall is the longest. It is on the surface of the posterior caudal wall of the meatus that we specifically treat the vagus nerve.

Diameter of the meatus

The caliber of the external acoustic meatus is not even. It is narrowest at the union of the external three-quarters and the internal quarter. This region is called the isthmus of the canal. Further inside, it enlarges until it reaches the tympanic membrane.

Important relationships

The anterior wall of the external acoustic meatus relates to the temporomandibular joint, especially the condylar process of the mandible. A thin layer of connective tissue separates them.

Nerves of the meatus

The external acoustic meatus is very sensitive, but less so than the tympanic membrane.
It receives nerve fibers from:

- The auriculotemporal nerve, which issues from the mandibular branch of the trigeminal nerve (V3).
- The vagus nerve, by its auricular branch that separates from it just beneath the cranial base. It then runs in an osseous canal nestled in the petrous part of the temporal bone (mastoid canaliculus) and gives off a small branch to the facial nerve. It passes through the tympanomastoid fissure and distributes to the skin of the osseous part of the external acoustic meatus and to the surface of the tympanic membrane (Fig. 25.7).

> **Note for manual therapists**
>
> Through the agency of the sensory filaments distributed to the posterocaudal wall of the auditory canal, we can have an effect on the tympanic membrane. This feature considerably broadens the scope of the indications for manipulation of the external acoustic meatus.

25.2 MIDDLE EAR

The tympanic membrane separates the tympanic cavity of the middle ear from the external ear.

The middle ear communicates with the nasopharynx by way of the Eustachian tube, and is connected to the oval and round windows through the intermediary of the auditus ad antrum.

25.2.1 Tympanic membrane

Pearly-gray in color, the tympanic membrane forms a partition between the external acoustic meatus and the inner ear. With a thickness of just 0.1 mm, it is both resistant and sensitive. It tilts in a caudal and medial direction,

Territory innervated by the facial nerve

Territory innervated by the vagus nerve

Territory innervated by the trigeminal nerve
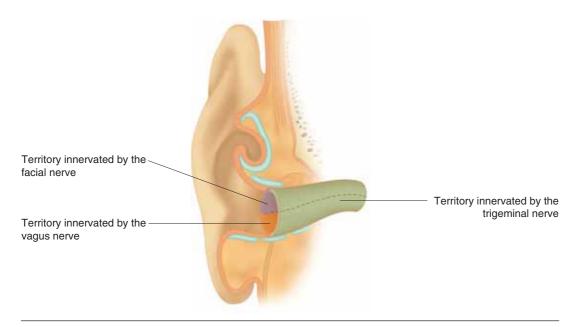
Fig. 25.7 Innervation of the external acoustic meatus and the middle ear.

like a satellite dish ready to pick up sounds. The handle of the malleus is firmly attached to the transparent inner surface, giving the membrane a concavity towards the external acoustic meatus. Above the malleus the membrane is thin (pars flaccida), while the remainder is taut (pars tensa).

We note that its tremendous sensitivity is due to the vagus nerve. Some anatomists insist that a more vertically inclined membrane is a sign of a musical ear.

25.2.2 Tympanic cavity

The tympanic cavity is an air chamber; it contains a chain of movable bones which transmit the vibrations of the tympanic membrane across the cavity to the middle ear. Shaped like a narrow box, its axis has an oblique medial and caudal orientation. It is lodged in the middle region of the petrous part of the temporal bone.

Chain of ossicles

The chain of auditory ossicles contained in the tympanic cavity is comprised of the malleus, the incus and the stapes.

Orifices of the tympanic cavity

- The Eustachian tube is a canal measuring 30 mm in length; it brings the tympanic cavity into communication with the nasopharynx.
- The aditus ad antrum is the tympanomastoid canal. With a length of several millimeters, it links the tympanic cavity to the most important of the pneumatic cells of the mastoid: the mastoid antrum (10 mm high and 6 mm wide).
- The fenestra vestibuli connects the tympanic cavity to the inner ear. The stapes is stituated next to the fenestra vestibuli.
- The fenestra cochleae also connects the tympanic cavity to the inner ear. This orifice is closed by the secondary tympanic membrane.

The tympanic cavity is lined with mucous membrane, which is continuous with the lining of the pharyngotympanic (Eustachian) tube.

25.3 PHYSICAL EXPLORATION OF THE EAR

25.3.1 Auricle

The auricle possesses great sensitivity, toughness and resilience. We are especially interested in its hypersensitive areas; they correspond to superficial sensory filaments of the vagus, trigeminal and facial nerves. Motor fibers of the facial nerve can also be quite sensitive.

We should treat sensitive fibers when we find them, as long as no swelling or infection of the ear is present.

25.3.2 Pre-auricular regions

Observe the ear and surrounding area for signs of lymphangitis, adenitis or swelling (tumefaction).

Behind the ear and towards the mastoid, note any swelling. Check particularly for any possible bone pain with pressure. This could indicate mastoiditis, usually an extension of a middle ear infection (otitis).

While in that area, palpate the parotid gland and the temporomandibular joint.

Angulomaxillary adenopathies will present with angina. Do not forget to consider dental problems, which can have incredibly far-reaching deleterious effects on the whole organism. We have seen sciatica disappear as a result of cavities being treated!

25.3.3 External acoustic meatus

Pull the auricle cephalad and posterior. If this movement is painful, it is a sign of infection in the canal.

By determining which direction of traction provokes the pain, we can establish which part of the conduit is involved.

- If pain results from traction in a cephalad direction, infection of the superior part of the conduit is indicated.

- If pain results from traction in a caudad direction, infection of the inferior wall is likely.
- If pain results from traction on the tragus, it reveals an infection of the anterior wall.

25.3.4 Tympanic membrane

Few manual therapists use a speculum or an otoscope. We suggest that, when in any doubt, it is best to refer the patient to a general physician or a specialist. Nevertheless, it is a good idea to know the Valsalva maneuver.

The Valsalva maneuver

Ask the patient to breathe out through the nose, pinching the nostrils between his or her fingers. This technique is commonly used to restore ear pressure during rapid changes of altitude. In cases of perforation or infection, the air pressure penetrating the Eustachian tube in the tympanic cavity will cause pain. It can also provoke a flow or discharge in the case of perforation.

25.4 MANIPULATION

This section discusses manipulations of the auricle of the ear and the external auditory canal. In previous chapters we described tech-niques for the facial and vestibulocochlear nerves, which address the endocranial osseous canals. Here we concern ourselves with tech-niques for the cartilage, muscles and nerves of the auricle.

25.4.1 Auricle

Cartilage

Cartilage test

With the exception of the lobule, the ear is composed of fibrocartilage that should be supple and painless when folded.

Place the auricle between the thumb and index finger of one or two hands and bend the ear back and forth over its whole area.

If bending the ear is difficult or sensitive, you have located a fibrocartilaginous fixation.

Cartilage technique

Using two hands, take the dense or hard part of the cartilage between your thumbs and index fingers. Give the cartilage a good twist in the direction of the "listening". Treat with induction, being sure to keep the cartilage under traction (Fig. 25.8).

The technique is complete when the sensi-tivity disappears and the cartilage is more supple.

Fig. 25.8 Neurocartilaginous manipulation.

Muscles of the auricle

As previously mentioned, the involuntary muscles of the auricle were essential for the survival of the species. To this day they retain links to the limbic system deep within our brain.

Extrinsic muscles

The superior and posterior auricular muscles, innervated by the facial nerve, are most important to our work.

Superior auricular muscle

Glide your index finger along the posterocephalad aspect of the temporal region. Feel for a sensitive thickening of the epicranial aponeurosis and manipulate it using the compression–stretching and induction techniques.

Search specifically for fixations on the convexity of the scaphoid fossa of the antihelix.

Posterior auricular muscle

Following the same procedure as above, move from the mastoid process along the convexity of the concha, searching for a somewhat hardened sensitive area, and manipulate with induction.

Intrinsic muscles
Helicis major muscle

This muscle sometimes joins the superior auricular muscle. It is a vertical band located on the ascending helix. Look for an area that is sensitive when flattened against the helix. Treat with compression–induction using the tip of the index finger against the posterior helix.

Helicis minor muscle

This muscle is situated obliquely on the crux of the helix, between its horizontal and ascending parts, and is well worth manipulating. Do not hesitate to place the tip of your index finger deeply into it. This is where the most important fixations are found.

Effects

In addition to the distant reflexogenic effects described in greater depth at the end of this chapter, manipulations of the auricular muscles have an effect on circulation to the face. They are used in cases of congestive facial disorders, in facial paralysis, and following hemiplegia. It is due to connections with the sympathetic carotid plexus that the nervous system of the cartilage and the muscles of the ear produce these vascular effects.

Nerves of the auricle

Sensory innervation

Sensory innervation derives from:

- the auriculotemporal nerve that supplies the tragus, crux and the ascending helix
- the auricular branch of the superficial cervical plexus that supplies the lobe, and both posterior parts of the helix and the antihelix
- the vagus nerve anastomosed with the facial nerve that supplies the concha (Ramsay Hunt's zone).

Note that the various innervations intersect, and that it is not always simple to differentiate them.

Technique

Glide and roll the auricle between your fingers, seeking out sensitive, dense or hard nerve filaments. These can be extremely fine. It is only when they are very sensitive that they should be treated. Do so with induction and stop when the pain ceases.

25.4.2 External acoustic meatus

We have seen that the innervation of the external acoustic meatus comes essentially from the facial nerve, with anastomotic branches of the vagus nerve and the auricular branch of the cervical plexus.

The anastomosis with the facial nerve is made by the auricular branch of the vagus nerve. At the Fallopian aqueduct it gives off a small filament to the facial nerve. At the cranial base, it divides into two branches: one for the posterior auricular nerve, and the other for the posterior surface of the auricle and the posterocaudal wall of the canal.

Moreover, the vagus nerve furnishes sensory filaments to the tympanic membrane, whose extreme sensitivity is well known.

Manipulation of the meatus

The most external part of the auricle can be manipulated with the medial tip of the index or little finger, and the deeper part with a dampened cotton swab (Fig. 25.9).

Most external part of the meatus

The indentation of the concha and the external cartilage of the acoustic meatus are accessible. With your finger, locate the most sensitive area; most often it is found at the caudal anterior aspect. Release this sensitive zone using compression–induction.

Deep part of the meatus

Take a cotton swab and dampen it just a little; the "dry rustle" of the swab is not well tolerated by some patients.

Direct the swab initially cephalad then caudal, all the while gently turning it against all the contours of the canal: the cephalad, caudal, medial and lateral canal walls. Progress forwards, maintaining pressure against the contour of the walls; as you come to each wall in turn, ask the patient if the compression is causing pain or discomfort. Above all, take your time; continue gently turning along the axis of the canal as you penetrate deeper.

If there is the slightest pain, retreat carefully and change the orientation of the swab ever so slightly in order to progress further.

With practice you will be able to feel the painful areas with the cotton swab because they are slightly roughened or hardened. You can feel the tissue "listening", as the cotton swab is just an extension of your fingers.

At the sensitive zone, apply compression–induction to release the painful part. This is certainly the most effective treatment we know for the vagus nerve, though it does require a degree of experience.

Recommendation

Do not attempt to apply this technique to the tympanic membrane. It can be very painful and cause severe vagotonia with lipothymia (fainting). The eardrum is a delicate, fragile structure possessing extreme sensitivity. If you touch it with the swab, you will cause an excruciatingly sharp pain.

Always keep the cotton swab pressed against the walls of the meatus for support. This ensures that you progress slowly and

Fig. 25.9 Neural manipulation within the external acoustic meatus.

avoid direct contact with the tympanic membrane. When sensitivity increases markedly, you are very close to the insertion of the tympanic membrane.

Reflex coughing

Reflex coughing is due to stimulation of the vagus nerve. Generally, it is provoked by lightly brushing against the tympanic membrane, but it can also be triggered when touching a deep part of the external acoustic meatus. It indicates that the cotton swab is in the right place.

25.4.3 Indications

It is difficult to be all-encompassing when it comes to indications for manipulation of the ear. This is because of the wide and complex distribution — and imprecise limits — of the vagus nerve. Some of these indications relate to the facial nerve and others to the vagus nerve:

- facial paralysis
- vascular algia of the face
- tinnitus (noises in the ear)
- sinusitis
- post-traumatic deafness
- fibrosis of the tympanic membrane by the intermediary of the wall of the external acoustic meatus

- otalgia (ear pain) without infectious cause
- cranial fixations. The effect results from regulation of intracranial pressure and from the vagal innervation of the meninges of the posterior cerebral fossa
- visceral pain, often ipsilateral. For example, in order to stimulate the liver or the right lung, the technique would be performed on the right ear canal
- vagotonia. For vagal attacks, the patient or his/her family and friends can be taught a maneuver that consists of placing the index or little finger in the external auditory canal every hour and rotating it several times. Another technique consists of biting the middle phalanx of the left little finger
- ear, nose and throat, following surgery, infection or trauma.

25.4.4 Precautions

It is the exploration of the meatus with the cotton swab that indicates whether manipulation is appropriate. You should be extremely circumspect if there is any pain or excessive sensitivity.

Manipulation of the brain

26

26.1 MECHANICAL CHARACTERISTICS

26.1.1 Cerebral visco-elasticity

The brain has the consistency of an egg custard wrapped in a firmer covering, giving it both viscous and elastic properties. Bathed as it is in cerebrospinal fluid, the encephalon is very sensitive to any and all pressure variations. All it takes to feel an immediate increase in intracranial tension is to incline one's head forward.

We will see in this chapter that, by altering intracranial pressure, it is possible both to evaluate and to treat the brain itself.

26.1.2 Mechanical sensitivity of the brain

Owing to its malleable and compressible texture, the brain is sensitive to the slightest pressure variation. Numerous factors — climatic, barometric, hygrometric, mechanical, digestive, hormonal, psychological and emotional — figure in its compressibility. According to the intensity of the stimulus, the brain responds with various symptoms ranging from a simple headache to an epileptic fit.

Manipulation of cerebral visco-elasticity, in addition to the classic cranial techniques, reduces the brain's susceptibility to unfavorable pressure gradients.

The brain and its glial cells, in particular, produce an electromagnetic field. We believe that our manipulations, through their action on intracranial pressure, influence these electromagnetic fields in such a way as to harmonize them.

26.1.3 Modifications of intracranial pressure

Intracranial pressure can be changed using various body positions, respiration, and compression of the abdomen, the eyes and the ears.

General rules

- With inspiration, intracranial pressure increases.
- With forced expiration, pressure tends to diminish.
- Bringing the knees up to the chest augments intracranial pressure.
- In prone position, with the head lowered, the pressure rises.
- Compression of the eyes and the acoustic canals increases pressure.

Global cranial "listening" with inspiration

We have discussed global cranial listening and its possible findings. Suppose, in your evaluation, that you are hesitating between a membranous and a brain fixation. The patient is supine. You have one hand placed under the occiput and the other on the skull, with the middle finger along the axis of the sagittal suture. Ask the patient to breathe in and hold his/her breath for several seconds. The

intracranial tensions will be emphasized as a result of the increased pressure.

In the case of membranous tension, your palm will glide, this time more clearly, towards the area of fixated dura mater.

In the case of a cerebral problem, the palm of your hand will dive deep, in a precise linear manner, towards the brain, where it will come to a more abrupt stop than it would in the case of a membranous restriction.

Inspiration intensifies intracranial fixations, allowing you to be more specific.

26.2 VISCO-ELASTIC CEREBRAL MANIPULATION

Visco-elastic cerebral manipulation consists of interacting with the intracranial pressure to affect the brain and the origins of the cranial nerves.

The hands placed on the skull use the pressure inside the brain to release fixations revealed by the listening evaluation.

26.2.1 Respiration

The patient is supine. Always begin by placing one palm under the occiput and the other on the skull, with the middle finger oriented along the sagittal suture.

- Listen globally to locate the fixated areas.
- Ask the patient to breathe in and hold his/her breath for a few seconds.
- Move your palms so that they are placed on either side of the fixated area, determined by the listening.
- Turn the patient's head so that you have an optimal hold that allows your palms to exert pressure in concert.
- Ask the patient to breathe out fully (the intracranial pressure will slacken). Then ask him to breathe in fully (the pressure will increase) so that you can locate the fixation more precisely.
- At maximum inspiration, ask the patient to breathe out fully. During this expiration phase, simultaneously compress the skull between your palms

in the direction of the listening. Do not hesitate to apply true gradual compression.

This interplay of pressure–decompression works on the brain fixation and on the entire fluid system of the encephalon. It affects the venous system, which has a very important role in diminishing the effective weight of the brain.

Remember that, with inspiration, negative intrathoracic pressure reverberates across the vertebrobasilar veins as far as the cerebellum and the posterior brain. It is one of the factors that diminishes the effective weight of the encephalon. We are always astounded by the fact that the brain, which weighs 1.3 kg when it is measured on a scale, has an effective weight of no more than 40 g in situ.

26.2.2 Respiration and cranial movement

Following exactly the same technique outlined above, coordinate the expiration phase with the retraction phase of the cranium, and inspiration with the cranial expansion phase.

This is an excellent technique, and the patient is able feel the variations in pressure.

26.2.3 Lower extremities

Here we combine the classic visco-elasticity of the brain technique with movement of the knees to the chest.

Following the protocol outlined above, in the inspiration phase, ask the patient to bring the knees up to the chest using both hands. This position will increase intracranial pressure. The pressure will diminish the moment expiration begins, at which point the patient releases the knees.

Finally, the phase of inspiration serves to place the cerebral tissue and its coverings under tension. On exhalation, the release of this tension benefits the elasticity of the return and enables you to work on the denser zones.

26.2.4 Compression of the eyes and the acoustic meatus

With the eyes

In the same position, ask the patient to compress the eyes. This has the effect of augmenting both anterior intracranial membranous tensions and intracranial pressure.

Compression combined with respiration
The patient gradually breathes in fully while compressing the eyeballs (without discomfort).

The use of pressure inside the brain affords a more precise evaluation of the fixation, as well as benefiting from the brain's elasticity.

In expiration
During the retraction phase of cranial rhythm, ask the patient to blow out gradually but completely while simultaneously releasing the compression on the eyes.

Locating and treating areas of cerebral dysfunction
Before treating your patient, perform a global cranial listening.

Suppose that you find an area on the right parietal of a patient suffering from right knee pain. Stimulate the musculo-ligamentous-capsular proprioceptors of the knee by stretching them, after which repeat the global cranial listening. If your palm is drawn to the occipital area or left frontal zone, for example, this attraction is to the part of the brain that is connected to the knee problem. Occasionally, of course, this second evaluation corresponds to the original listening, but this is rare.

The area can be treated using the cerebral visco-elasticity technique described above. This technique permits the release of the tissue density where the brain has stored the (knee) problem.

This technique can be applied to all parts of the body, particularly the viscera.

The acoustic meatus

Follow the same protocol as for compression of the eyes. Compressing the ears is most useful if the listening takes you posterior, as its greatest effect is on the middle and posterior compartments of the brain.

26.2.5 Indications and contraindications

Indications

We expect our techniques to have an effect at the level of the origins of the cranial nerves and also on the attachments of the dura mater to the cranial orifices. It is even possible to imagine that the smaller the orifice and the more sensitive it is to variations in pressure, the more pathogenic its fixation.

The indications for manipulation are as follows:

- variations in brain density (demyelination, protein plaque, micro-scars)
- sequelae of cranial trauma
- Parkinson's disease
- multiple sclerosis
- Alzheimer's disease
- cerebral circulatory insufficiency.

Obviously, we do not have a direct effect on these illnesses but rather on their functional consequences.

Contraindications

Contraindications are as follows:

- arterial hypertension
- intracranial aneurysm
- diabetes
- sequelae of a ruptured aneurysm.

Pathologies of cranial nerves

27

In the manual approach to cranial nerves we are sometimes confronted with serious pathologies. This chapter may serve to help you avoid some pitfalls. In addition, it may help you understand the relevance and effects — local and general — of certain manipulations.

27.1 PATHOLOGIES OF INDIVIDUAL CRANIAL NERVES

The cranial nerves are subject to more or less the same diseases as the peripheral nerves. However, their diagnosis is more complex and requires a neurologist's opinion.

A neurologist can be more specific about the characteristics of the lesion:

- its location
- its pathophysiology
- its etiology
- its remote repercussions.

Brain imaging has considerably refined diagnosis, but often a diagnosis is made a posteriori, after symptoms have appeared.

Table 27.1 shows that each cranial nerve has its own symptomology and contributes to a more complex clinical picture.

27.2 PATHOLOGIES INVOLVING MULTIPLE CRANIAL NERVES

The pathologies that affect several cranial nerves at a time are listed in Table 27.2.

Table 27.1 Principal clinical signs and symptoms of cranial nerves (after Doyon et al 2002)

Nerve	Function	Clinical symptoms and signs
Olfactory nerve (I)	Sensory	Anosmia or hyposmia
Optic nerve (II)	Sensory	Unilateral loss of visual acuity, edema or discoloration of the eyelid, scotoma of the visual field (bitemporal hemianopia with lesions of the chiasm, lateral homonymous hemianopia with lesions of the optic tracts or optic radiations (often in quadrants))
Oculomotor nerve (III)	Motor and autonomic	Diplopia (vertical, horizontal or oblique), ptosis, divergent strabismus, limited adduction, elevation and lowering of the eye, paralytic mydriasis, accommodation paralysis
Trochlear nerve (IV)	Motor	Vertical diplopia looking down and to the healthy side, inclination and rotation of the head to the side opposite the lesion
Trigeminal nerve (V)	Mixed	Paresthesia or neuralgia of the hemiface, abolition of the corneal reflex, deficit in the muscles of mastication, drooping in the corners of the mouth
Abducent nerve (VI)	Motor	Horizontal diplopia, convergent strabismus, inability to abduct the eye
Facial nerve (VII)	Mixed	Peripheral facial paralysis (cephalad and caudal territory of the facial nerve) Hypoesthesia of the Ramsay Hunt's zone, loss of taste to the anterior two-thirds of the tongue, reduced lacrimal and salivary secretions
Acoustic nerve (VIII)	Sensory	Tinnitus (noises in the ear), hypoacusis or deafness (cochlear nerve) Vestibular syndrome (vestibular nerve)
Glossopharyngeal nerve (IX)	Mixed	Neuralgia, hypoesthesia or anesthesia of the pharynx and posterior third of the tongue, loss of taste to the posterior tongue, paralysis of the velo-pharyngo-laryngeal junction, decreased salivary secretions
Vagus nerve (X)	Mixed	Hypoesthesia or anesthesia of the pharynx and larynx, paralysis of the velo-pharyngo-laryngeal junction, autonomic signs
Accessory nerve (XI)	Motor	Paralysis of sternocleidomastoid and trapezius muscles
Hypoglossal nerve (XII)	Motor	Paralysis of the hemitongue (deviation of the tongue to the affected side when protracted)

Table 27.2 Principal topographical syndromes of cranial nerve problems at the cranial base (after Doyon et al 2002).

Syndrome	Cranial nerves involved	Principal etiology
Of the superior orbital fissure	III, IV, VI, ophthalmic branch of V, sometimes II (if the lesion is localized at the orbital apex)	Tumors, sphenoid sinus incursions, aneurysms
Of the lateral wall of the cavernous sinus	III, IV, VI, ophthalmic branch of V, often exopthalmic	Aneurysms in the cavernous sinus, thrombosis of the cavernous sinus, tumors of the sella turcica or cranial sinuses
Of the sphenobasilar junction	II, III, IV, V (neuralgia), VI	Tumors (large) of the middle angle of the skull
Of the mastoid tip	V (neuralgia), VI	Otitis with petrositis, temporal bone tumors
Of the internal acoustic meatus	VII, VIII	Tumors, infectious processes
Of the pontocerebellar cistern	V sensory, VII, VIII (cochlear and vestibular)	Acoustic neuroma, meningioma
Of the jugular foramen (posterior foramen lacerum)	IX, X, XI	Tumors, aneurysms, carotid dissection
Of the jugulo-hypoglossal junction (hypoglossal canal)	IX, X, XI, XII	Tumors, aneurysm, carotid dissection
Of Garcin	Unilateral extradural attack on all the cranial nerves	Oropharyngeal cancers, metastases to the cranial base

Adenopathies of the neck and face

28

When we are exploring the emerging branches of the cranial nerves, it is important to know how to interpret the significance of the various ganglions we might find in the neck.

As a rule, all infections should make us very cautious, and patients should be directed to a physician.

Some ganglions are of little significance and present as a matter of course in young children or adolescents. They signal lymphatic activity in response to the demands of the immune system or to general fatigue.

28.1 TUMEFACTIONS (SWELLINGS) OF THE FACE

- Submaxillary and submental adenopathies signify lesions of the mouth, the tongue and the floor of the mouth.
- Subangulo-maxillary adenopathies occur in connection with tonsillar and lingual infections.
- Submaxillary adenopathies can occur after a lithiasis (calculus) in a salivatory gland.
- Parotid inflammations:
  — Acute parotitis: on palpation the swellings are sensitive behind the maxilla and under the ear.
  — Parotid tumors: on palpation, behind the ascending branch of the maxilla, a hard, mobile, rounded nodule can be felt, which causes discomfort on chewing and speaking.

Caution: Facial paresis, neuralgic pain, and a tumor adhered to the skin and at deeper levels should make you strongly suspect cancer of the parotid gland.

28.2 LATERAL TUMEFACTIONS (SWELLINGS) OF THE NECK

It is important to know how to palpate and analyze lateral swellings in the neck. We repeat: it is quite common to find an adenopathy in this region, signaling normal immune reactions or local infections.

As a rule, we find ganglions that are oval, small in volume, smooth, clearly defined, and about the size of a grain of rice or a small pea. They are generally firm and resilient. They can be easily mobilized in relation to the adjacent tissues.

Look out for these less innocuous signs:

- thickened and coalescent ganglions
- ganglions of sizeable volume
- ganglions of a soft and fluctuating consistency
- overly hardened ganglions which adhere to neighboring tissues.

Note: We have stated this already but it is important to emphasize that it is extremely ill advised to perform joint mobilization techniques on the cervical column in the presence of ganglions on the neck or face! Adenopathies themselves often generate

joint pains or dislocations. Their presence always indicates an immune system that is dealing with infection, inflammation or a tumor.

We have seen a number of diseases whose initial symptoms were cervical pain. The eval-uation trap was that the pain had sometimes been triggered by exertion or poor body posi-tion. In all such cases, patients were convinced that the problem was purely mechanical. Such patient assurances could lead you to a faulty determination or prognosis.

Cervical pain of cranial origin

29

The cervical column is a favored area for pain projecting from the cranium. The example of a stiff neck in cases of meningeal syndrome illustrates this phenomenon well. Meningeal irritation, especially in the posterior cerebral compartment, sends pain messages to C1 and C2 spinal segments. To review the innervation of the dura mater, refer to Chapter 5, which goes into some detail about the relationship between the cervical spine and the posterior cranial fossa.

Cranial nerve manipulations are a valuable treatment approach in response to cervical neuralgia of functional origin. However, it is vital to know the symptoms of more involved cases so as to be able to direct patients towards the correct care.

29.1 INTRACRANIAL HYPERTENSION

Increased intracranial pressure is most often due to an expansion process going on in the skull.

29.1.1 Symptomatology

Some symptoms are so obvious that these patients are unlikely to come to see us; other symptoms are more subtle.

Symptoms in the cases of mild hypertension include:

- visual disturbances
- tension behind the eyes
- malaise, with apathy, sleepiness or torpor
- a slackened pulse

- papillary stasis
- intermittent headache.

The headache is significant but inconsistent. It often wakes the sufferer in the second part of the night, mimics pseudo-migraine syndrome and is accompanied by vomiting.

More severe cases of hypertension involve:

- involuntary projectile vomiting without effort and with no alimentary cause
- papillary edema: this begins with slight venous dilatation but no visual disturbance. Little by little the symptoms worsen, to the point of abducent nerve paralysis resulting in double vision
- convulsive fits
- obsessive state
- cerebral hernias: these are caused by mechanical deformation of the encephalon, rather than by an increase in pressure. The consequences of these cerebral hernias are to be feared, as they cause dysfunction of the part of the encephalon that is squeezed, and vasoneural compression.

Signs of intracranial hypertension and, above all, signs of cerebral hernia are a medical emergency.

29.1.2 Chief causes of intracranial hypertension

The principal causes of intracranial hypertension are anything that can increase cerebrospinal fluid pressure: trauma, meningitis, meningeal hemorrhage, tumor or encephalitis.

Benign intracranial hypertension, with normal CT scan, can occur in connection with:

- severe arterial hypertension
- cerebral venous thrombosis (a complication of otitis)
- Behçet's disease
- certain medications: corticoids, antibiotics and vitamin A.

29.1.3 Topographical clinical forms

Here are the main signs of tumors according to location:

- frontal: psychic problems with euphoric dementia (moria)
- posterior fossa: headaches due to increasing neck stiffness; cerebellar problems
- temporoparietal: signs of cortical excitement, either motor (Bravais–Jackson type) or sensory, sometimes accompanied by homolateral hemianopia (defective vision)
- pontocerebellar: hearing dysfunction, facial paralysis, trigeminal neuralgia
- third ventricle and pituitary region: hypothalamopituitary dysfunction
- sphenoidal: visual problems with enophthalmos (backward displacement of the eye in the bony socket).

29.2 MENINGEAL SYNDROME

Meningeal syndrome groups together symptoms connected with any pathological irritation of the meningeal envelopes and the cerebrospinal fluid. It accompanies biological changes in these components of the nervous system.

29.2.1 Symptoms

The symptoms of meningeal syndrome constitute a classic triad.

Headache

Headache is the most common, most consistent and earliest symptom. It is a consequence of either intracranial hypertension or inflammation of structures of the cranial base.

This type of headache can be very intense, violent, diffuse (sometimes predominantly frontal) and constant (with paroxysms) and can prevent sleep.

It is worsened by:

- noise (phonophobia)
- light (photophobia)
- coughing
- abdominal pressure
- flexion of the neck.

It is not relieved by the usual analgesics, and is often accompanied by vertebral column pain as well as cutaneous hyperesthesia.

In cases of meningeal hemorrhage, the headache can be localized at the start. In subdural hematoma, it is often associated with pain provoked by pressure at the temporal fossa.

Vomiting

This type of vomiting is inconsistent but precocious. It is known as 'cerebral' vomiting, it is projectile, unrelated to meals, frequently spontaneous or occurring when the patient changes position.

Constipation

Constipation is the third element in the classic meningeal triad. It is a rather irregular symptom and of limited practical interest.

29.2.2 Signs

Clinical examination seeks to identify the meningeal syndrome.

Meningeal stiffness

Meningeal tightness is a contracture of the paravertebral muscles, a defense against the secondary pain stemming from inflammation of the meninges.

Painful and permanent, it sometimes presents with the subject lying down, curled up with his or her back to the light, head back, and extremities half-bent. All attempts to flex

the head provoke insurmountable and painful resistance. There is extreme neck stiffness; rotational and side-to-side movements are possible, but aggravate the headache.

Clinical tests

Two maneuvers confirm meningeal stiffness and it is useful to be familiar with these. These signs are especially useful to know when the diagnosis is not immediately evident; we have experienced this on several occasions.

Kernig's sign

This is the inability of a patient who is seated or lying down to extend the leg, without bending the knee, when the thigh is flexed on the abdomen.

Brudzinski's sign

This is an involuntary flexion of the lower extremities when the neck is passively flexed. Hyperflexion of the hip causes the following reaction in the other lower limb:

- flexion, if the lower extremity was initially in extension
- extension, if the lower extremity was initially in flexion.

Accompanying signs

Among the signs accompanying meningeal rigidity are the following:

- a slowing of the cardiac rhythm
- cutaneous hypersensitivity
- the meningeal mark of Trousseau: if a line is traced on the skin with a fingernail, a mark appears; initially white, it reddens and then slowly disappears

- fever, often elevated and dissociated from the pulse.

  In severe cases the following are seen:

- neurological signs caused by irritation to the underlying nervous system: epileptic seizure, diverse forms of paralysis (notably oculomotor paralysis), psychic disorders
- meningeal irritation, which can also involve pyramidal signs in the form of a briskness of osteotendinous reflexes.

Clinical forms

Meningeal syndrome can have two causes: meningeal hemorrhage or meningitis.

The signs suggesting meningeal hemorrhage are:

- sudden onset
- absence of fever despite a possible temperature elevation to 38°C after a few hours.

  The signs pointing to meningitis are:

- more gradual onset
- fever of 39–40°C with shivering, sweating and myalgia (muscle pain)
- possibility of an epidemic (contagion) or other signs of infection: rhinopharyngitis, diarrhea, rash.

The febrile nature of meningeal syndrome is often evident, except where antipyretics have been given. Even aspirin, for example, masks the elevation in temperature.

Sometimes, other causes of fever are associated — pneumopathy, sinusitis or otitis, for example — and are in fact the portal for meningitis.

Sinusitis

30

30.1 PNEUMATIC ROLE OF THE SINUSES

The pneumatization of the bones of the face is linked to the evolution of the skull. It lightens the weight of the facial mass, thereby diminishing the pressure it exerts at the cranial base.

The sinuses play a considerable pneumatic role in harmonizing intracranial and craniofacial pressures, lightening the skull and its contents. Those who have ever experienced sinusitis know how disagreeable and incapacitating the symptoms can be. A few drops of pus are sufficient to increase intrasinous pressures and provoke very strong local (congestion) and regional (headache) reactions.

Some patients have sinusitis with no apparent cause. These are patients with very sensitive mucous membranes, who sometimes have an allergy of which they are unaware. Other patients have an asymptomatic dental problem, which causes irritation of the trigeminal nerve to radiate to the sinuses. In addition, some mechanical imbalances of the skull, frequently of traumatic origin, can cause sinus irritation and inflammation.

30.2 DEFINITION

Sinusitis is the inflammation and infection of the sinuses of the face. Infection may spread from the nasal cavities or from dental infection, or more rarely from a secondary infection following a general infection such as influenza or scarlet fever.

In manual medicine, we see many cases of non-infectious sinus inflammation.

30.3 SYMPTOMS

Symptoms of sinusitis are essentially an acute or dull headache, sometimes of migraine or neuralgia type. Sinus inflammations often entail purulent mucous discharge.

Patients sometimes complain of malaise and of feeling as if their head is in a vice. Their distress is worsened by pressure variations occurring with changes in position (flexion of the head), coughing and sneezing.

Filaments of the trigeminal nerve emerging on the face are very sensitive to the touch.

30.4 LOCALIZATION

30.4.1 Frontal sinusitis

The frontal sinuses produce suborbital and frontal head pain. These headaches are regular in occurrence. The orbital rim and the superior medial corner of the orbit are sensitive to the touch (supra-orbital nerve), as is the root of the nose. Patients have the impression of their head being in a vice.

30.4.2 Maxillary sinusitis

This form of sinusitis creates suborbital pain radiating to the teeth. Pressure on the infra-orbital nerve "bouquet" worsens the pain. Episodes occur regularly.

30.4.3 Ethmoidal sinusitis

Ethmoidal sinusitis is characterized by dull anterior head pain behind the nose. With anterior ethmoiditis, purulent discharge drains into the middle meatus of the nasal fossa. With posterior ethmoiditis, mucous discharge drains into the olfactory fissure and the superior meatus. Chronic episodes contribute to the formation of nasal polyps.

30.4.4 Sphenoidal sinusitis

With sphenoidal sinusitis, headaches are deeper, often projecting to the occiput. Purulent discharge drains into the rhinopharynx, leaving a bad taste in the mouth. In contrast to other types of sinusitis, patients seldom blow their nose.

30.4.5 Combined sinusitis

Sinusitis can occur in combination: fronto-maxillary, fronto-ethmoidal or ethmoido-sphenoidal.

30.5 ETIOLOGY

Various factors create a susceptibility to sinusitis:

- **Digestive**: Examples include gastro-esophageal reflux and hepatic dysfunctions.
- **Hormonal**: There is a particular sensitivity to estrogens.
- **Allergic**: Direct heightened sensitivity can be due to breathing in irritants, as well as reactions from the metabolism of certain substances in the blood (chocolate, sulfites, alcohol, drugs, paint, varnish, solvents, etc.).
- **Toxic**: Air pollution and tobacco smoke are the principal vectors. The mere odor of tobacco on the clothes can cause sinus irritation.

Autonomic nervous system

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31

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The autonomic nervous system is one of the key reasons why our treatments have the effects that they do. In the preceding chapters we have dealt with each cranial nerve individually. Even though we have already mentioned their numerous anastomoses and connections, it is also important to consider the autonomic components of the cranial nerves as a whole. The routes nerve fibers adopt are not always direct, and the effects of our manipulations go well beyond the local.

The rich fiber exchange between the cranial nerves and the sympathetic cervical system explains the conjoint relationship of the head and the neck. It is sometimes hard to differentiate symptoms arising from the neck from those which originate in the head, in so far as their nervous systems are interdependent.

31.1 SYMPATHETIC AND PARASYMPATHETIC SYSTEMS

The two components of the autonomic nervous system are too often considered to be antagonistic. While their actions on various organs may appear to be in opposition, the two systems are, in fact, complementary.

The sympathetic system readies the individual for action. We call this function *ergotropic*. Classically, it is assumed to implement the fight or flight response. This system predominates during the day and in wakefulness.

The parasympathetic system functions to protect and restore the internal environment.

This is termed *endophylactic*. It counterbalances the effects of the sympathetic system. This system predominates at night and during sleep.

The anatomical distinction between the two systems is based primarily on the location of their pre-ganglionic neurons:

- a thoracolumbar location for the sympathetic system
- a craniosacral location for the parasympathetic system.

We will look at the cervicocephalic part of the sympathetic and parasympathetic divisions of the autonomic nervous system. These two systems are complementary and function alternately most of the time.

The autonomic nervous system has a subtlety that is sometimes difficult to grasp. Sympathetic and parasympathetic often affect the same structures, upon which they have different (usually contrasting) but coordinated effects. We will be better able to understand these workings through the examples of the glossopharyngeal and vagus nerves.

31.2 GLOSSOPHARYNGEAL AND VAGUS NERVES

31.2.1 Viscerosensory innervation of the glossopharyngeal nerve

The glossopharyngeal nerve transports the unconscious sensations coming from the carotid body (chemoreceptor) and the carotid sinus (baroreceptor).

The chemoreceptors of the carotid body register oxygen levels, carbon dioxide levels, and the acid–base balance of the arterial circulation. The baroreceptors of the carotid sinus are pressure receptors that register arterial pressure, and therefore are sensitive to the stretching of the artery wall.

These sensations are relayed by the carotid branch of the glossopharyngeal nerve towards its inferior ganglion (of Andersch), where the neuronal cell bodies are located. They send information to the reticular formation and to the hypothalamus to bring about appropriate modification reflexes for the regulation of respiration, arterial pressure and cardiac output.

Carotid body

The carotid body, or carotid corpuscle, is a small chemoreceptor measuring 3 mm × 6 mm, located at the carotid bifurcation (Fig. 31.1).
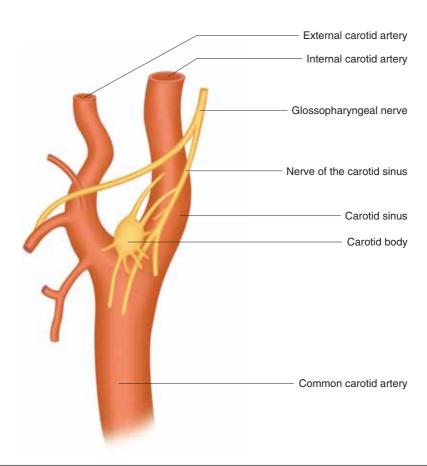
It is very sensitive to:

- hypoxia (inadequate oxygen)
- hypercapnia (greater than normal levels of CO_2 in the blood)
- acidosis (low blood pH).

In response to these changes in the blood, the carotid body relays signals via the carotid branch of the glossopharyngeal nerve.

Carotid sinus

A little beyond the division of the common carotid artery, the carotid sinus shows as a dilatation of the internal carotid vessel. Here the tunica media is thinner, while the tunica adventitia is relatively thick, owing to the presence of stretch receptors.

External carotid artery

Internal carotid artery

Glossopharyngeal nerve

Nerve of the carotid sinus

Carotid sinus

Carotid body

Common carotid artery

Fig. 31.1 The carotid body.

These receptors are connected to glosso-pharyngeal sensory nerve endings. They respond to elevations in arterial pressure in the sinus and generate impulses which initiate the reflexogenic lowering of arterial blood pressure.

31.2.2 Parasympathetic fibers of the vagus nerve

Visceromotor innervation of the vagus nerve

The pre-ganglionic neuronal cell bodies are situated in the dorsal vagal nucleus and in the medial part of the nucleus ambiguus. The dorsal vagal nucleus is located in the floor of the fourth ventricle and lies in the central gray matter of the nearby medulla oblongata. The pre-ganglionic fibers of the nucleus cross the spinal nucleus of the trigeminal nerve, emerging at the lateral border of the medulla oblongata, and course in the vagus nerve.

The neurons of the *dorsal nucleus* innervate the intestinal ganglions and their derivatives (lungs, liver, pancreas). The neurons of the *nucleus ambiguus* supply the ganglions of the cardiac plexus.

The two nuclei are influenced by signals coming from the hypothalamus, the olfactory system, the reticular formation and the nucleus of the tractus solitarius.

In the internal pharynx and the larynx, pre-ganglionic axons activate the ganglionic neurons, which are secretory-motor for the pharyngeal and laryngeal mucosa.

- **For the thorax**: The vagus nerves descend by different routes with each fragmenting into many branches, which join the plexus surrounding the large vessels at the base of the heart. The pulmonary branches provoke broncho constriction. The esophageal branches activate esophageal peristalsis by stimulating the smooth muscles lining the esophageal walls.
- **For the heart**: The cell bodies of the pre-ganglionic axons are located in the nucleus ambiguus. Their axons terminate in the small ganglions

connected to the heart and are responsible for slowing the cardiac cycle.
- **For the stomach**: The right and left gastric nerves emerge from the esophageal plexus. These nerves are secretory-motor to the glands, and motor to the smooth muscles of the stomach.
- **For the intestine**: The intestinal branches have a corresponding action on the small intestine, cecum, appendix, ascending colon and most of the transverse colon, being secretory-motor to the glands and motor to the muscular coats.

Viscerosensory innervation of the vagus nerve

At the conscious level, visceral sensations relayed by the vagus nerve translate as "feeling well" or "feeling poorly". Pain sensations of visceral origin are conveyed by the sympathetic system.

Issuing from the perivisceral plexus, the visceral sensory fibers converge towards the right and left gastric nerves. These nerves pass through the esophageal hiatus of the diaphragm to blend with the peri-esophageal plexus.

The sensory fibers from the cardiac and pulmonary plexus unite and continue their passage upwards within the right and left vagus nerves.

The two vagus nerves are joined by nerves conveying visceral information arising from:

- the aortic arch, from baroreceptors
- the aortic bodies, from chemoreceptors
- the lower larynx, via the recurrent laryngeal nerve
- the superior larynx, via the internal laryngeal nerve
- the mucosa of the epiglottis, the base of the tongue and the aryepiglottic folds by way of the pharyngeal plexus.

The cell bodies of the viscerosensory neurons are located in the inferior ganglion of the vagus nerve (plexiform ganglion). They are connected to the reticular formation and

the hypothalamus for the modification of the reflexes controlling cardiovascular, respiratory and gastrointestinal activities.

The connections via the reticulobulbar tracts (between the reticular formation and the cranial nerve nuclei in the brain stem) with the dorsal vagal nucleus (motor) allow for reflex responses from the vagus nerve itself.

> **Note for manual therapists**
>
> Vagovagal reflexes are of the utmost importance to our work because they allow us to interact with the autoregulation of the vagus nerve in its entirety.

31.3 CERVICOCEPHALIC SYMPATHETIC SYSTEM

The cervical portion of the sympathetic system extends from the base of the skull to the superior thorax. It distributes collaterals to the head, the neck, the upper limbs and the anterior mediastinum.

The sympathetic cervical chain is located anterolateral to the vertebral column, behind the vasoneural pedicle of the neck (Fig. 31.2).

It is composed of three ganglions, distinguished according to their positions (superior, middle and inferior) and connected by intervening cords.

The pre-ganglionic fibers destined for the head and neck stem from the first three or four thoracic segments, and ascend towards the stellate ganglion (inferior cervical ganglion).

31.3.1 Sympathetic cervical system

Superior cervical ganglion

The superior cervical ganglion is located in the retrostyloid space, limited:

- laterally, by the posterior belly of the digastric muscle and the sternocleidomastoid muscle
- posteriorly, by the transverse processes of the second and third cervical vertebrae

and the prevertebral muscles (longus colli and anterior rectus); the ganglion lies against these structures

- anteriorly, by the styloid "curtain" muscles and the jugular carotid neurovisceral bundle
- medially, by the pharynx and sagittal plate.

The superior cervical ganglion is fusiform, shaped like a spindle, and extends from the cranial base to the fourth cervical vertebra.

Inside the ganglion:

- some fibers synapse and go to the common carotid, subclavian and vertebral arteries
- other fibers cross without synapsing and reach underlying ganglions.

Middle cervical ganglion

The middle cervical ganglion is present in 50% of people. Variable in location, shape and size, it usually assumes the form of a large pea. Generally, it is located inside the anterior tubercle of the transverse process of the sixth cervical vertebra. Protruding somewhat, this tubercle is also referred to as the tubercle of Chassaignac.

The middle cervical ganglion relays fibers to the tongue, larynx and thyroid body.

Inferior cervical ganglion

The inferior cervical ganglion is situated at the crossroads of the neck, the thorax and the upper limb. It is formed by the coalescence of 4–6 cervical ganglions, and one or two thoracic ganglions. It is located in the fossa inferior and posterior to the pleura (fossa of Sebileau), and limited:

- in front, by the posterior slope of the pleural dome
- posteriorly, by the transverse process of C7 and by the neck of the first rib
- laterally, by the transverse pleural and costal pleural ligaments
- medially, by the pleural vertebral ligament.

Carotid nerve

Ansa of the atlas

Superior cervical ganglion

Communicating branch of C2

Third cervical nerve

Fourth cervical nerve

Middle cervical ganglion

Anterior scalene muscle

Vertebral artery

Inferior cervical ganglion

Roots of the brachial plexus

Ansa subclavia
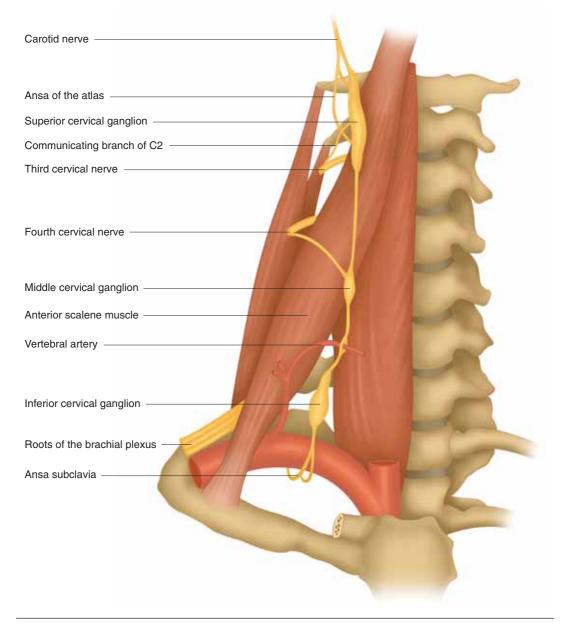
Fig. 31.2 Sympathetic cervical chain.

The ganglion is found on the posterior surface of the subclavian artery and often surrounds the vertebral artery.

From the superior cervical ganglion, postganglionic fibers arise, which divide into:

- anastomoses with the lower cranial nerves and with the first four cervical nerves, via gray rami communicantes

- the peri-arterial plexus of the external and internal carotids, to distribute to the head and neck along their plexus.

The very rich peri-arterial plexuses of the external carotid and its collaterals distribute to the tissues of the face and the extracranial surfaces. They also follow the meningeal arteries and continue to the meninges.

259

The internal pericarotid (Fig. 31.3) and the vertebrobasilar plexuses follow the internal carotid and vertebral arteries into the cranial cavity and continue on the cerebral arteries.

31.3.2 Distribution

From these ganglions there emerge branches that vary in nature and destination.
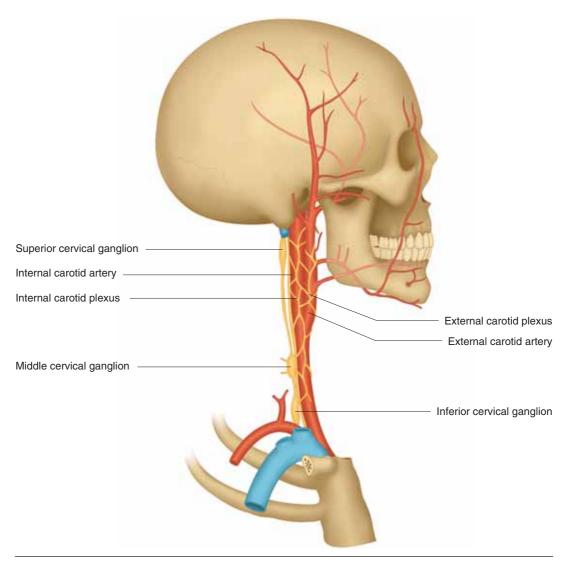
We can distinguish several:

- rami communicantes
- vascular collaterals
- musculoskeletal collaterals
- visceral collaterals
- anastomotic branches.

Communicating branches (rami communicantes)

- From the superior cervical ganglion, branches arise that are destined for the first four cervical nerves, which comprise the cervical plexus. The filaments reach the ansa of the atlas, the second and

Superior cervical ganglion

Internal carotid artery

Internal carotid plexus

Middle cervical ganglion

External carotid plexus

External carotid artery

Inferior cervical ganglion

Fig. 31.3 Pericarotid plexus.

third cervical nerves and, in some people, the fourth cervical nerve.
- The middle cervical ganglion gives off filaments to the fourth and fifth cervical nerves.
- From the inferior cervical ganglion, filaments branch off to the nerves composing the brachial plexus (fifth, sixth, seventh and eighth cervical nerves and first thoracic nerve).

Vascular collaterals

Superior cervical ganglion
Carotid artery and internal carotid plexus

The carotid nerve extends the cephalad pole of the superior cervical ganglion along the internal carotid artery (Fig. 31.4). It carries vasomotor fibers for the brain and the retina, as well as for the control of the iris muscle. At the entrance to the carotid canal, it divides into a medial and a lateral branch, which anastomose to form the carotid plexus.

The *lateral branch* supplies:

- The caroticotympanic nerve. This penetrates the tympanic cavity and unites with a branch of the tympanic nerve (IX, nerve of Jacobson).
- The deep petrosal nerve. At the exit of the carotid canal, this crosses the foramen lacerum and joins the greater petrosal nerve of VII to constitute the vidian nerve, destined for the pterygopalatine ganglion.
- A filament exchanges fibers with the abducent nerve.

The *medial branch* supplies:

- Anastomotic filaments to:
  — the oculomotor, trochlear and abducent nerves
  — the trigeminal ganglion
  — the ophthalmic nerve.

These last filaments control the iris muscle, which they reach by way of the nasal nerve, the ciliary ganglion and the short ciliary nerves. These are the iris dilator fibers.
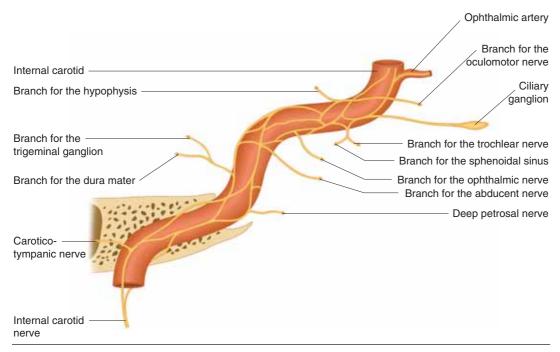
Fig. 31.4 Branches of the internal carotid plexus.

- Filaments for:
  — the hypophyses (pituitary)
  — the dura mater of the sella turcica and of the basilar plate
  — the mucosa of the sphenoidal sinus

At the exit of the cavernous sinus, the two branches terminate in the vascular plexus that surrounds the internal carotid and its branches.

Intercarotid nerves

These are the nerves to the carotid bifurcation. Four to seven in number, the intercarotid nerves constitute, together with nerves issuing from the vagus and glossopharyngeal nerves, the intercarotid plexus. This plexus innervates the carotid bifurcation and the carotid body. From this plexus numerous filaments spring to form a rich peri-arterial plexus on the external carotid. The prolongations of the plexus accompany all the collaterals and terminals of the artery: the facial, superior thyroid, superficial temporal, internal maxillary and middle meningeal arteries.

Middle cervical ganglion

From this ganglion, there arise filaments for the common carotid and particularly for the inferior thyroid artery (inferior thyroid plexus).

Inferior cervical ganglion
Vertebral nerve and vertebrobasilar plexus

From the inferior cervical ganglion come two vertebral nerves, one anterior and the other posterior, which constitute a plexus surrounding the vertebral artery (Fig. 31.5).

The posterior nerve gives off the *deep rami communicantes* into the fifth, sixth and seventh cervical nerves.

At each corresponding intervertebral foramen, the plexus emits a sympathetic root to the *sinuvertebral nerve*, which supplies the spinal dura mater and the intervertebral discs.

Before entering the skull, the perivertebral plexus is reinforced by filaments issuing from the middle cervical ganglion and from the first two or three cervical nerves (Fig. 31.6). It ascends the intracranial vertebral artery, on the basilar trunk and its collaterals. Here it is known as the vertebrobasilar nerve, forming an anastomosis with the pericarotid plexus.

Nerves of the subclavian artery

Some nerves of the subclavian artery derive from the inferior cervical ganglion, either directly or via the ansa subclavia (ansa of Vieussens), and extend to the anterior surface of the artery and its collaterals, particularly the vertebral, internal thoracic and inferior thyroid arteries.

Musculoskeletal collaterals

Slender filaments, arising from the posterior surface of the ganglions, go to the vertebral bodies and the prevertebral muscles (longus colli and anterior rectus).

Visceral collaterals

Among the visceral collaterals, those of the inferior cervical ganglion are of greatest importance. They explain the astonishing results that can be achieved on the cranial vascular system.

In treating the thoracic inlet with manual therapy, one can free up:

- The pleural filaments going to the pleura
- The slender short esophageal and tracheal filaments
- The inferior cardiac nerve, which on both sides terminates behind the arch of the aorta, in the deep cardiac plexus. Along its course it forms anastomoses with the vagus and recurrent nerves, and supplies the vascular nerves to the subclavius, the carotid and the arch of the aorta. It supplies the thymic and pulmonary nerves, as well.
- The anastomosis with the phrenic nerve, formed when an extension of the subclavian plexus is joined by a branch from the phrenic nerve. On the right, an anastomosis joins the ansa subclavia to reach the vagus and recurrent nerves.
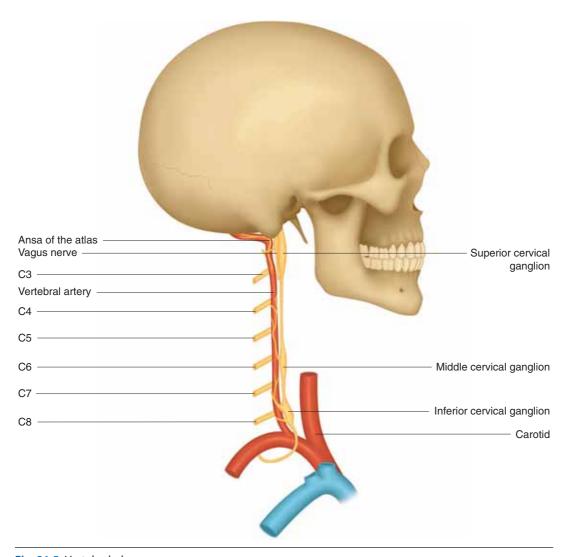
Fig. 31.5 Vertebral plexus.

Ansa of the atlas
Vagus nerve
C3
Vertebral artery
C4
C5
C6
C7
C8

Superior cervical ganglion
Middle cervical ganglion
Inferior cervical ganglion
Carotid

Anastomotic branches

- From the superior cervical ganglion, the jugular nerve ascends towards the jugular foramen. It ends in the inferior ganglions of the glossopharyngeal and vagus nerves. The anastomosis is formed by several filaments.
- From the middle cervical ganglion, an anastomosis is sent to the recurrent nerve of the vagus.
- The inferior cervical ganglion and the phrenic nerve have interconnections

which are continuous, but variable in number and development.

31.4 SYMPATICOTONIA

Just as we described vagotonia, here is a summary of the features of sympaticotonia:

- nervousness
- highly emotional state
- irritability
- pessimism

263
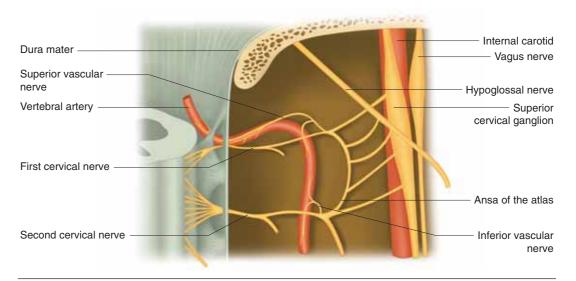
Fig. 31.6 Innervation of the vertebral artery.

- violence, contained to a greater or lesser degree
- insomnia
- weight loss
- exaggerated tendinous and pilomotor reflexes
- mydriasis
- possible enophthalmos
- tachycardia
- elevated systolic pressure
- slow digestion
- postprandial pain
- meteorism and constipation.

Neuroglia

32

32.1 GENERAL POINTS

Glial cells occupy more than 50% of the cerebral volume. They were discovered in 1856 by the German physician, Rudolf Virchow. He grouped them generically as glia, which literally means "glue".

From that time on, they have been the "forgotten ones" of neuroscience. They were once considered to be rudimentary cells, composed of an amorphous substance, simply content to occupy the empty cerebral space and passively insulate neurons. A little later they were considered to be, at best, cells assuming the function of connective tissue: support, exchange, resorption and scar formation.

However, the latest research shows that, in fact, they play a major role in the activities of the nervous system. Some have the role of sentry or protector. Others participate in the processing of information in the brain. They have the capacity to decipher information and transmit it from one end of the brain to the other, forming a communication network to supplement that constituted by the neurons.

32.2 GLIAL CELLS

32.2.1 Oligodendrocytes

Oligodendrocytes fabricate the myelin sheath, an insulator that protects and facilitates the circulation of nerve impulses. They are responsible for laying down myelin in the central nervous system and are thus counter-parts of the peripheral myelinating Schwann cells. However, whereas each Schwann cell myelinates just a single axon, the oligodendrocytes may enclose several neighboring axons in separate myelin sheaths.

32.2.2 Microglia

Microglia, the true sentries of the brain are glial cells equipped with "arms" which they use to explore surrounding areas. The foundation of the cerebral immune system, these glia defend the central nervous system from attack. They also work as phagocytes to clear cellular debris left by the neurons and degenerating glial cells.

32.2.3 Astrocytes

The most numerous glial cells are the astrocytes, so called because of their star shape. These cells were long thought to be simple cellular deposits filling in the space between the neurons.

The astrocytes participate in regulating the composition of the extracellular fluid. Forming a sort of envelope around the synaptic junction, they contribute to reducing the diffusion of the neurotransmitters that have been released. They are capable of capturing the neurotransmitters and molecules that are active in the synaptic space.

They play a role:

- in regulating the rate of synaptic neurotransmission

- in controlling the extracellular concentration of harmful substances for optimal neural function.

32.3 GLIAL MOTILITY

Manual therapists were interested in glial cells long before the scientific community rediscovered them. Since W.G. Sutherland, several authors have seen these cells as very probably the "motor" of the primary respiratory mechanism.

Numerous investigations have demonstrated different neuroglial cellular activity. Histological cell cultures reveal that:

- Astrocytes show only weak mobility and represent the stable tissue element. However, according to Bear et al (2002), "the expansion and contraction of the neurites" (axons and dendrites) observed by certain authors are closely dependent on the astrocytes.
- Microgliocytes move rapidly between fine foliate extensions, continually changing shape.
- Oligodendrocytes are "animated by a pulse that contracts and dilates the cell body according to a regular rhythm" (Kahle, Leonhardt and Platzer (1979), citing Lumsden and Pomerat (1951)).

If it were established that the activity of certain groups of cells operate in concert, the existence could then be affirmed of a generator of the cranial movement perceived by the hand of the manual therapist.

32.4 FUNCTIONAL ROLE

32.4.1 Astrocyte communication

Astrocytes have long arms that encircle the junctions between neurons to form a protective coating. When the nerve impulse arrives at the synapse, it triggers the flow of neurotransmitters into the synaptic space. Due to their privileged location around the synapse, astrocytes are able to facilitate, slow down or even block the flow of information. Equally, they are capable of transmitting information from one synapse to another. Thus neurons are not the only ones capable of information exchange.

In the brain, groups of astrocytes form communication assemblies called *astrocyte domains*. These collectives work specifically with certain neurons.

The spreading of information by astrocytes is not electrical, as it is with neurons, but rather propagated through chemical pathways. There is a considerable output of calcium ions, which move back and forward like a wave from astrocyte to astrocyte. Gliologists speak of a *calcium wave*. The communications transported along this pathway move very slowly. On the nerve pathway *information circulates 100 000 times faster than on the astrocyte pathway*. Having two networks, one rapid and the other slow, probably allows the brain to coordinate its activity more efficiently.

Neuronal organization

Astrocytes are a major component of the cerebral scaffolding and have been shown to be indispensable to the creation of new neurons and new synapses.

The birth of neurons throughout our lifetime is also under astrocyte control. Even into adulthood, the brain is able to make new nerve cells from stem cells, particularly in the hippocampus. Substances released by the astrocytes facilitate the transformation from stem cell to neuron.

Brain plasticity, key to learning and remembering, turns out to be dependent on chemical factors released by astrocytes.

The learning process

Glial cells play a determining role in the intelligence of the human species, which possesses the largest proportion of glial cells in the animal kingdom.

From the neuronal point of view, Albert Einstein's brain, autopsied in the 1980s, was found to be no different from those of the layman. The number and appearance of his neurons were nothing out of the ordinary. Only those areas of the brain dedicated to the

most complex tasks were shown to have an incredibly high proportion of glial cells.

The cortex tends to increase in volume in cerebral zones that have undergone an intense learning process. In these same areas, neuron density tends to diminish. It appears that the glial cells form special contact zones for the neurons. The more complex the task, the more astrocytes appear to intervene in the communication between neurons. In other words, faced with a new or complex situation, the astrocytes also adapt.

Memory formation

Astrocytes appear to play a facilitating role in the formation of memories.

32.5 PATHOGENIC ROLE

Glial cells are increasingly revealing themselves to be involved in pathologies of the central nervous system. Some may even hold the key to brain diseases.

In the biological scenario that results in Alzheimer's disease, astrocytes have a dual personality. Their role in the inflammatory processes accompanying amyloid plaque deposits in the brain has for a long time made them the "bad guys". These collections of proteins accumulate in the brain, causing neuron death and finally dementia. Yet recent studies are beginning to show that astrocytes also have the capacity to break down these amyloid plaque formations. This clearing up of affected areas could slow down, or even stop, the appearance of lesions.

This feature of playing both good and bad roles in the course of a single disease also appears with pathologies like Parkinson's disease, Huntington's disease, amyotrophic lateral sclerosis, epilepsy and cerebrovascular accidents (ischemia, infarct).

We believe that manipulation of the brain has a great impact on the glial cells, certainly much more important than its effect on the neurons themselves.

Conclusion

Cranial nerve manipulations are an undeniable "plus" in improving the effects of many osteopathic techniques. Their effect on the dura mater, the cranial sutures, the brain and indeed the entire organism go a long way towards helping patients.

Osteopathy is a complex science and a difficult art. It is not enough to be satisfied with a catalogue of isolated techniques. With a holistic view of the body, the manual therapist respects even the tiniest structures, each one playing its part under the orchestration of the brain.

The objective of osteopathic treatment is to address by mechanical means the most dysfunctional part of the organism. It seeks to achieve a local effect and also to inform the brain of the changes made. The brain's informational role is considerable. It handles millions of messages transmitted and received by the tissues.

The integration of these messages following our treatment is part of the subtle healing processes that take place in an individual. In reorienting the body's working structure and the mechanisms of homeostasis, the brain creates new conditions that will permit individuals to recover, little by little, a physiology in keeping with their expectations. It is largely the 100 000 km of our peripheral nervous system that permits these amazing exchanges.

Neurons have their own mechanical pathology, namely exogenic constraint, edema or endoneural fibrosis. Our hands can help them regain optimal function and thus improve the essential mind–body relationship.

Manipulation of the cranial nerves is a precious tool for attaining greater refinement of our understanding of the unity of the human body. We must tirelessly defend and affirm the concept and the philosophy of osteopathy.

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Glossary

Acoustic pore formerly the auditory hole, orifice of the acoustic meatus.

A frigore "by chilling". Term used for facial paralysis, and sometimes for pleurisy or nephritis. It is just one parameter of these diseases.

Ageusia total or partial loss of taste.

Agoraphobia fear of wide-open spaces or large groups of people.

Amaurosis blindness, without any apparent lesion in the eye.

Amblyopia reduced vision in an eye that appears to be structurally normal.

Andersch's ganglion a ganglion situated inferior to the glossopharyngeal nerve, caudal to the petrous part of the temporal bone, furnishing the tympanic nerve (Jacobson's nerve).

Anisocoria inequality of the diameter of the two pupils.

Anisotension unequal arterial tension measured at the limbs.

Anosmia loss of the sense of smell.

Anterior foramen condyle hypoglossal canal.

Antrotympanic communication canal between the tympanic membrane and the surface of the mastoid.

Arnold (nerve of) greater suboccipital nerve, emerging between atlas and axis.

Arnold (recurrent nerve of) recurrent meningeal branch of the ophthalmic (VI) innervating the tentorium cerebelli.

Ataxia impaired ability to coordinate movement; a staggering gait.

Aura sensations preceding paroxysms of a disease. Can be motor, sensitivity, sensory, vasomotor, secretory or psychic.

Avellis syndrome homolateral paralysis of the upper palate, the pharynx and the vocal cords due to an affliction of the accessory (XI) and vagus (X).

Behçet's syndrome associated with many symptoms: ocular lesions (uveitis, chorioretinitis), joint pain (cervical), digestive, vascular and nervous.

Bell's palsy facial paralysis, lateral cephalic rotation of the eyeballs to the affected side and inability to close the eye.

Blastoderm portion of the egg that mammals develop into an embryo; it consists of two sheets, ectoderm and endoderm.

Blepharospasm spasm of the orbicular muscles of the eyelid.

Bravais–Jackson disease form of localized epilepsy; the person remains conscious until the convulsion attacks the face. The convulsions are localized to a single limb or half of the body. It is often due to cortical irritation caused by trauma, a tumor, sclerosis, a vascular problem, etc.

Brudzinski's sign involuntary flexion of the lower extremities during passive flexion of the neck, in the course of meningeal syndrome.

Carotid trigone triangular space formed laterally by the sterno-cleido-occipito-mastoid muscle, medially by the omohyoid muscle, and cranially by the digastric muscle.

Cataract opacity of the lens.

Celiac ganglion small nerve plexus, situated around the celiac trunk, that contributes to the formation of the solar plexus.

Chassaignac (tubercle of) tubercle on the anterior surface of the transverse process of C6 that serves as a location point for the carotid.

Cholesteatoma benign tumor formed by epithelial cells and cholesterol that is found in the middle ear as a complication of otitis.

Ciliary ganglion formerly ophthalmic ganglion. Nerve plexus situated against the lateral surface of the optic nerve, from which the short ciliary nerves arise.

Coalescence adherences that form between similar tissue types, e.g. ganglions.

Coenesthesia an often uncomfortable sensation coming from the interior of the body.

Conjunctiva thin mucous membrane covering the internal surface of the eyelid and the anterior surface of the ocular globe. This is what allows them to glide on each other.

Cushing's disease hyperplasia of the adrenal gland with excessive secretions of cortico-adrenal hormone.

Dermatome a cutaneous area that is innervated by the corresponding sensory spinal nerve.

Diplopia double vision. Symptom occurring with paralysis of the oculomotor nerve.

Ectoderm embryonic germ layer forming the epidermis of the skin, the sensory organs and the nervous system.

Edinger–Westphal nucleus synonym: papillary nucleus. The motor nucleus of the intrinsic musculature of the eye, belonging to the oculomotor nerve.

Ehrenritter's ganglion synonym: superior ganglion of IX. The ganglion to the glossopharyngeal nerve situated above the jugular foramen.

Endoderm (or endoblast) embryonic germ layer forming the lining of the digestive tube and its associated structures.

Enophthalmos abnormal sinking of the ocular globe into the orbit. It is the opposite of exophthalmos, protrusion of the ocular globe from its orbit.

Epley's maneuver passive and rapid mobilization of the patient, from a vertical to a horizontal position, to unblock otoliths from the labyrinth of the inner ear.

Ergotrophy reaction to non-specific stimuli.

Garcin (syndrome) unilateral paralysis of the cranial nerves caused by a tumor at the base of the skull.

Geniculate body one of two small eminences located at the posterior thalamus. The medial geniculate body is the relay for the auditory pathways. The lateral geniculate body is the relay for the visual pathways.

Geniculate ganglion ganglion of the facial nerve, situated in the Fallopian aqueduct.

Glaucous bluish-green in color.

Hemianopia partial or complete loss of vision in the visual field of one or both eyes.

Hering's nerve branch of the glossopharyngeal nerve that innervates the carotid sinus.

Hydrops (anasarca) formerly "dropsy". General edema resulting from an accumulation of liquid in the cellular tissues or in the cavity of an organ.

Hyperacusis excessive acoustic sensitivity with pain on hearing certain sounds.

Hypermetropia difficulty in seeing objects that are close.

Induction manually encouraging the tissue in the direction of the "listening".

Inferior ganglion of the vagus nerve formerly plexiform ganglion. The vagus ganglion is situated slightly below the jugular foramen.

Iridocyclitis inflammation of the iris and the ciliary body.

Jacobson's nerve new nomenclature for the tympanic nerve which issues from the glossopharyngeal nerve.

Jacobson's organ (vomeronasal organ) diverticulum of the nasal fossa, situated on each side of the median partition.

Kernig's sign inability to bend at the hip without bending the knees, in a sitting or supine position. Elevation of the lower limb causes lumbar pain. This sign is present in meningeal syndrome.

Lipothymia malaise, excessive sweating, nausea, shallow breathing, muscle weakness, visual disturbance, but no loss of consciousness.

Lobstein's disease genetic bone fragility that becomes apparent late in life with deformation of the cranium and blots on the sclera.

Meatus formerly canal. Designates the conduit itself, not just its orifice.

Meckel cavum trigeminal cavity. Doubling of the dura mater creating a cavity at the anterior surface of the petrous portion of the temporal bone, which contains the trigeminal ganglion.

Ménière's disease vestibulocochlear syndrome with prostrating vertigo, tinnitus and deafness.

Mesoderm primary germ layer that forms the skeleton and muscles of the body: conjunctive tissue, bone, cartilage, blood, lymphatic, pleura, pericardium, peritoneum, synovial capsule, kidneys and sexual organs.

Moria excessive euphoria and joviality.

Myotome synonym: myomere, muscular segment. Series of embryonic symmetrical segments, whose origin is mesoderm, laid down the length of the medullary tube. Each segment gives rise to a group of striated muscle fibers corresponding to a metamere.

Nervi nervorum intrinsic micro-nervous system of a nerve.

Neural bud a small hard raised sensitive bulge, which can be felt with the hands on a nerve whose mobility is restricted.

Neurokinine polypeptide that is secreted by the nervous system in vasodilatation.

Neuropeptide chemical mediator of a peptide nature, secreted by the central nervous system.

Nucleus ambiguus bulbar motor nucleus common to the glossopharyngeal and vagus nerves.

Nystagmus involuntary ocular movement; eyes jerking in an opposite direction.

Otic ganglion nerve plexus appended to the mandibular nerve, situated below the foramen ovale.

Otolith formation rich in calcium crystal, which covers certain parts of the labyrinth and serves to balance the body.

Otosclerosis sclerotic tissue of the middle and internal eye.

Paget's disease chronic disorder of the adult skeleton in which localized areas of bone become hyperactive, replacing the normal matrix with softened and enlarged bone.

Parosmia imaginary olfactory perception.

Petrositis osteitis of the petrous pyramid.

Placode thickening of the ectoderm forming the beginnings of sensory organs and nerve ganglions.

Presbyacusis loss of hearing because of ageing.

Pterygoid in the shape of a wing.

Pterygopalatine ganglion formerly sphenopalatine ganglion. Nerve plexus appended to the maxillary nerve, situated in the pterygopalatine fossa.

Ptosis drooping of the eyelid. The subject cannot lift the upper eyelid due to paralysis of the muscle or a congenital cause.

Quadrantanopia loss of a quarter of the field of vision.

Ramsay Hunt's neuralgia Neuralgia of the geniculate ganglion (ganglion of the facial nerve in the Fallopian aqueduct). The pain is localized to the auditory canal in the middle ear.

Renitency firm consistency, resistant to pressure of the finger.

Rhinencephalon part of the brain specific to the olfactory senses. It is made up of the olfactory bulb, olfactory tract and the limbic system.

Scarpa's ganglion nervous ganglion of the vestibular nerve.

Scotoma blind spots. Lacuna in the visual field with small insensitive points on the retina.

Septum a partition that separates an organ, a cavity or any other anatomical organ into two parts.

Sinuvertebral nerve recurrent nerve (nervus spinosum) that innervates the meninges of the spinal nerves.

Somite one of the 42 paired, metamerically arranged cell masses formed in the early embryonic para-axial mesoderm.

Stapedius nerve the nerve destined for the stirrup muscle.

Superior ganglion of the vagus nerve formerly jugular ganglion, situated in the jugular foramen.

Tic douloureux produced in trigeminal neuralgia (facial neuralgia), often from a mechanical stimulus: rotation of the head, compression of the trigger points of the face, etc.

Tract a collection of nerve fibers in the central nervous system having the same origin, termination and function.

Trigeminal ganglion formerly ganglion of Gasser, situated at the origin of the three branches of the trigeminal nerve (ophthalmic, mandibular, maxillary).

Utricle small cavity situated in the cephalic part of the vestibular membrane of the inner ear.

Uveal tract a vascular tunica of the eye, comprised of the iris, ciliary body and choroids.

Valsalva maneuver expiration from the nose, with the mouth closed and the nostrils pinched, to increase the pressure in the Eustachian tube.

Vasa nervorum intrinsic microvascular system of a nerve.

Velum palatinum mobile musculomembranous partition. It is a caudal and posterior prolongation of the roof of the palate, and separates the nasal from the buccal part of the pharynx.

Vestibule middle cavity of the labyrinth of the inner ear.

Vieussens' ansa (ansa subclavia) loop extending from the third cervical ganglion and surrounding the subclavian artery.

Vomeronasal organ Jacobson's organ. Diverticulum of the endonasal olfactory organ, sensitive to pheromones.

Wrisberg (intermediary nerve of) sensitive nerve root of the facial nerve.

Zinn (annulus of; common tendinous ring) tendon formed by the oculomotor muscle surrounding the optic nerve.

Index

283