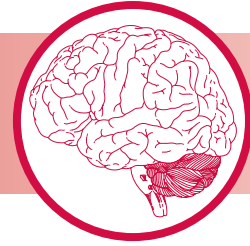
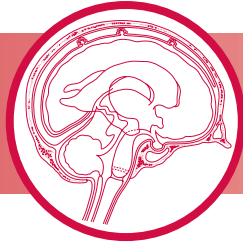


CHAPTER 15



Cranial Nerves

CLINICAL CASE

OLFACTORY NERVE (CN I)

OPTIC NERVE (CN II)

OCULOMOTOR NERVE (CN III)

TROCHLEAR NERVE (CN IV)

TRIGEMINAL NERVE (CN V)

ABDUCENT NERVE (CN VI)

FACIAL NERVE (CN VII)

VESTIBULOCOCHLEAR NERVE (CN VIII)

GLOSSOPHARYNGEAL NERVE (CN IX)

VAGUS NERVE (CN X)

SPINAL ACCESSORY NERVE (CN XI)

HYPOGLOSSAL NERVE (CN XII)

SYNONYMS AND EPONYMS

FOLLOW-UP TO CLINICAL CASE

QUESTIONS TO PONDER



CLINICAL CASE

A 70-year-old male has excruciating pain in the lower left part of his face. This began 1 month ago. He describes it as being like a jolt of lightning that radiates from his left ear, down to his jaw, and to the side of his mouth. These jolts of pain occur numerous times each day. Between attacks his face seems normal. He denies any numbness or tingling sensations. There is no hearing abnormality. The pain is triggered by talking, chewing, or touch of the lower left part of his face. He is unable to eat or brush his teeth, particularly on the left

side, since he fears triggering another painful attack. He can only drink his meals through a straw and cannot lie in bed on his left side. He had the same symptoms about 2 years ago. At that time he was treated with a medication which helped; symptoms subsided, but he stopped taking the medicine. The pain is so distressing that the patient admits to contemplating suicide.

The general and neurologic exam is normal, except that he withdraws and will not let anyone touch the left side of his face.

There are 12 pairs of cranial nerves emerging from the brain and radiating from its surface (Fig. 15.1). They pass through skull foramina, fissures, or canals to exit the cranial vault and then distribute their innervation to their respective structures in the head and neck. One of the cranial nerves, the vagus (L.,

“wanderer”) continues into the trunk where it innervates various thoracic and abdominal organs.

In addition to being named, the cranial nerves are numbered sequentially with Roman numerals in the order in which they arise from the brain, rostrally to caudally. The

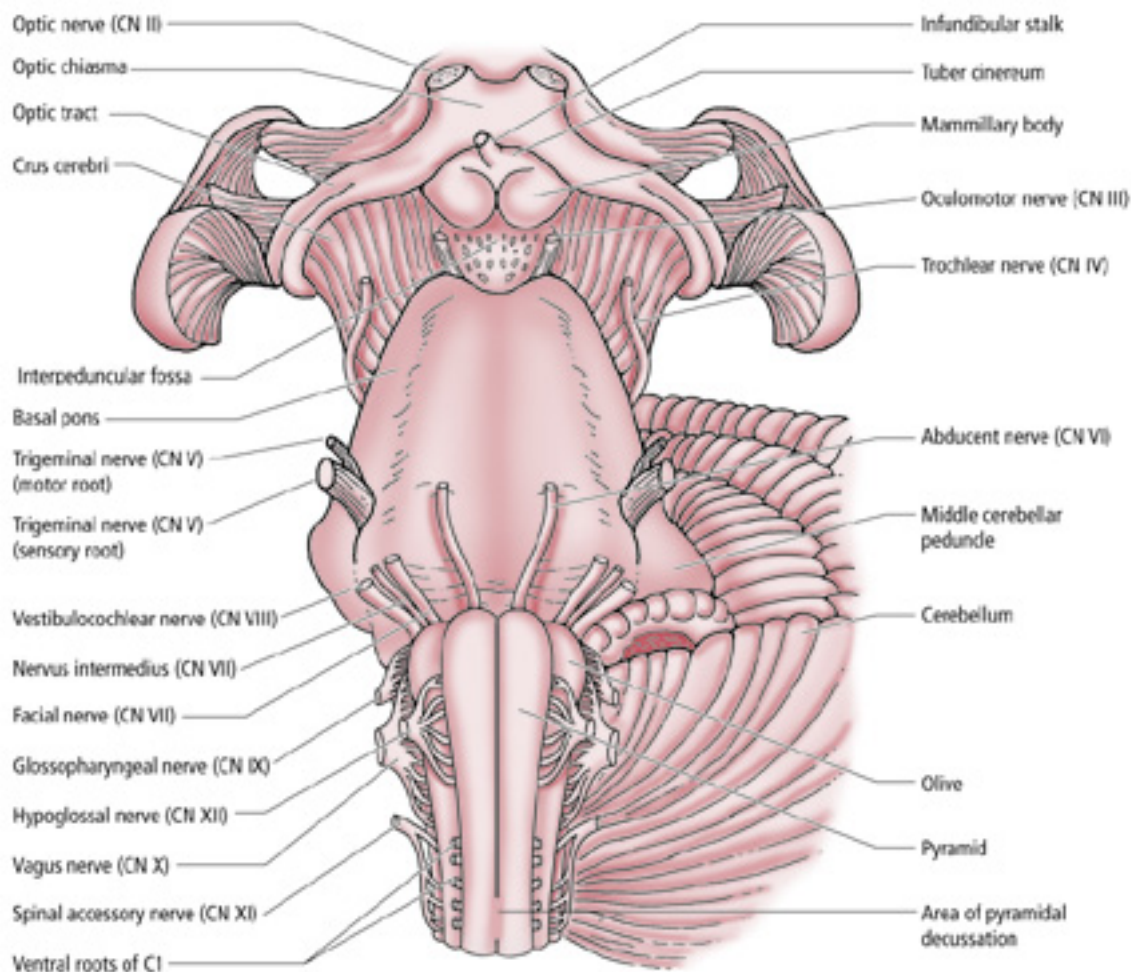


Figure 15.1 ● Ventral view of the brainstem showing the cranial nerves.

following list includes their names and corresponding numbers.

- I Olfactory nerve.
- II Optic nerve.
- III Oculomotor nerve.
- IV Trochlear nerve.
- V Trigeminal nerve.
- VI Abducent nerve.
- VII Facial nerve.
- VIII Vestibulocochlear nerve.
- IX Glossopharyngeal nerve.
- X Vagus nerve.
- XI Spinal accessory nerve.
- XII Hypoglossal nerve.

Although the cranial nerves and their sensory and parasympathetic ganglia (Tables 15.1, 15.2) form part of the peripheral nervous system, the optic nerve is really an outgrowth of the brain that emerges from the prosencephalon (not the brainstem as other cranial nerves), and is therefore not a typical cranial nerve. Moreover, part of the spinal accessory nerve arises from the cervical spinal cord; thus

Table 15.1 ● Sensory ganglia of the cranial nerves

Ganglion	Cranial nerve association
Trigeminal (semilunar, Gasserian)	Trigeminal (V)
Geniculate	Facial (VII)
Cochlear (spiral)	Cochlear (VIII)
Vestibular (Scarpa's)	Vestibular (VIII)
Superior glossopharyngeal	Glossopharyngeal (IX)
Inferior glossopharyngeal	Glossopharyngeal (IX)
Superior vagal	Vagus (X)
Inferior vagal (nodose)	Vagus (X)

there are only nine pairs of cranial nerves that emerge from the brainstem.

The main sensory and motor nuclei of the cranial nerves are shown in Fig. 15.2.

In describing the various functional components (modalities) of the cranial nerves, the definition of the following terms should be kept in mind: *afferent* is sensory input; *efferent* is motor output that may be *somatic* to skeletal muscles or *visceral* to smooth muscle, cardiac muscle, and glands, and

Ganglion association	Cranial nerve association	Trigeminal	Function(s)
Ciliary	Oculomotor (III)	Ophthalmic division	Constricts pupil; lens accommodation
Pterygopalatine	Facial (VII)	Maxillary division	Lacrimation; nasal gland, and minor salivary gland secretion
Submandibular	Facial (VII)	Mandibular division	Salivation (parotid gland)
Otic	Glossopharyngeal (IX)	Mandibular division	Salivation (submandibular and sublingual glands)
Intramural	Vagus (X)		Gland secretion; peristalsis

Table 15.2 • Parasympathetic ganglia of the cranial nerves.

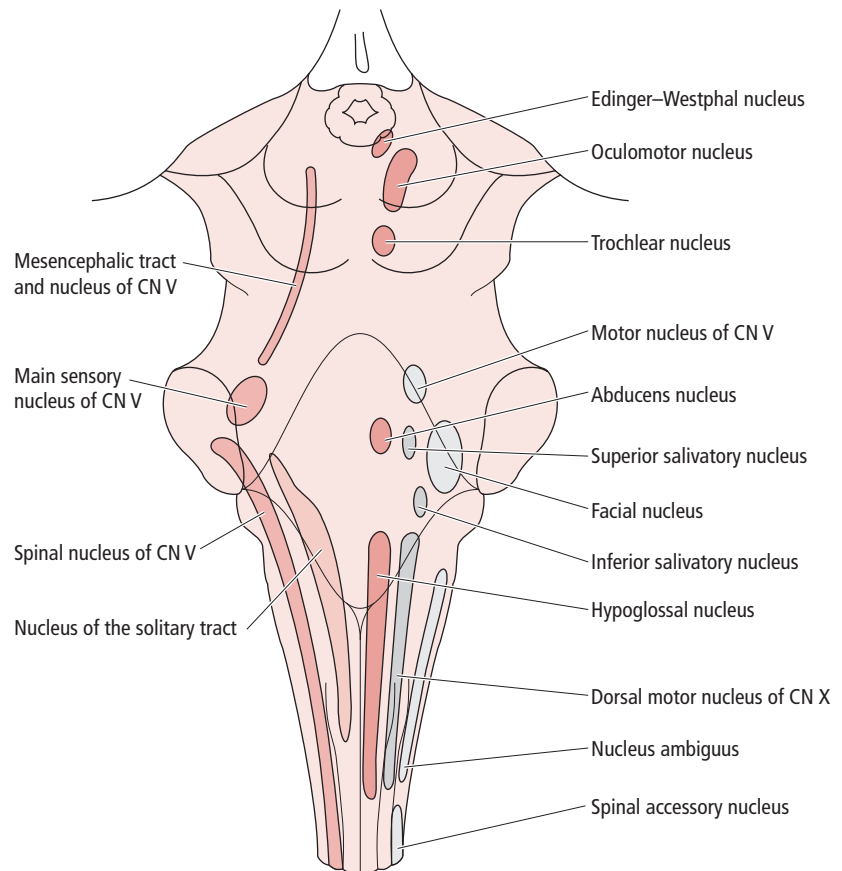


Figure 15.2 • The nuclei of the cranial nerves. The sensory nuclei are illustrated on the left, and the motor nuclei on the right.

special visceral efferent to striated muscles derived from the branchial arches; *general* refers to those components that may be carried by cranial nerves as well as spinal nerves; *special* refers to functional components that are carried by cranial nerves only. The following categories describe the functional components carried by the various cranial nerves (Table 15.3).

1 General somatic afferent (GSA). These fibers carry general sensation (touch, pressure, pain, and temperature)

from cutaneous structures and mucous membranes of the head, and **general proprioception (GP)** from somatic structures such as muscles, tendons, and joints of the head and neck. The trigeminal, facial, glossopharyngeal, and vagus nerves transmit GSA input to the spinal nucleus of the trigeminal nerve.

2 General somatic efferent (GSE). These fibers provide general motor innervation to skeletal muscles derived from embryonic somites. The oculomotor, trochlear, and abducent nerves innervate the extraocular muscles that

Modality	Cranial nerves	Function(s)
General somatic afferent (GSA)	V, VII, IX, X	General sensation and general proprioception
General somatic efferent (GSE)	III, IV, VI XII	Motor supply to extraocular muscles Motor supply to tongue
General visceral afferent (GVA)	VII, IX, X	General sensation from viscera
General visceral efferent (GVE)	III, VII, IX, X	Parasympathetic fibers to viscera
Special somatic afferent (SSA)	II VIII	Special sensory input from retina Special sensory input from vestibulocochlear apparatus
Special visceral afferent (SVA)	I VII, IX, X	Special sense of smell Special sense of taste
Special visceral efferent (SVE)	V, VII, IX, X	Motor innervation to muscles of branchiomeric origin: mandibular, hyoid, 3rd, 4th, and 6th branchial arches

Table 15.3 ● Cranial nerve functional components.

control eye movements, whereas the hypoglossal nerve supplies motor innervation to the muscles of the tongue, mediating movement of the tongue.

- 3 General visceral afferent (GVA).** General sensation from the viscera is transmitted by the facial, glossopharyngeal, and vagus nerves.
- 4 General visceral efferent (GVE).** These fibers provide visceral motor (parasympathetic) innervation to the viscera. The only cranial nerves that transmit parasympathetic fibers are the oculomotor, facial, glossopharyngeal, and vagus nerves.
- 5 Special somatic afferent (SSA).** These fibers carry special sensory input from the eye (retina), for vision, and from the ear (vestibular apparatus for equilibrium, and cochlea for hearing). The only nerves transmitting this component are the optic and vestibulocochlear nerves.
- 6 Special visceral afferent (SVA).** These are special sensory fibers from the viscera. These fibers convey the special sense of smell transmitted by the olfactory nerve and the special sense of taste transmitted by the facial, glossopharyngeal, and vagus nerves.
- 7 Special visceral efferent (SVE).** These motor fibers are special because they supply motor innervation to skeletal muscles of branchial arch origin. These fibers are carried by the nerves of the branchial arches, which are the trigeminal, facial, glossopharyngeal, and vagus nerves.

Table 15.4 summarizes the modalities, nuclei, ganglia, and functions of the cranial nerves.

OLFACTORY NERVE (CN I)

The olfactory receptor cells reside in the olfactory epithelium, and not in a sensory ganglion as is typical of other cranial nerves

The **bipolar olfactory receptor cells** (first order sensory neurons) of the olfactory apparatus reside not in a sensory ganglion,

but instead in the **olfactory epithelium (neuroepithelium)** of the modified nasal mucosa lining the roof and adjacent upper walls of the nasal cavities (see Fig. 19.1). The axons of these bipolar neurons are **SVA fibers** transmitting *olfactory* sensation. These axons assemble to form bundles, the **olfactory fila** (L., “threads”), which collectively form **cranial nerve I**. The olfactory fila traverse the fenestrations of the cribriform plate of the ethmoid bone to terminate in the olfactory bulb where they synapse with **second order relay neurons** and **inter-neurons** (see Chapter 19).

OPTIC NERVE (CN II)

The optic nerve consists of the myelinated axons of the retinal ganglion cells

The **optic nerve** mediates the special sense of *vision* via its **SSA fibers**. Light entering the eye activates cells known

as rods and cones, the photoreceptors of the retina. Electrical signals generated by the photoreceptors are transmitted to other cells of the retina that process and integrate sensory input. The **first order sensory bipolar neurons** of the visual pathway reside in the retina and transmit electrical signals of visual sensory input to the **multipolar second order ganglion cells** of the retina. The ganglion cells give rise to unmyelinated axons that converge at the optic disc and traverse the lamina cribrosa, a sieve-like perforated area of the sclera, to emerge from the back of the eyeball. At this point, the ganglion cell axons acquire a myelin sheath and assemble to form the optic nerve. This nerve, an outgrowth of the diencephalon, leaves the orbit via the optic canal to enter the middle cranial fossa. There, the optic nerves of the right and left sides join each other to form the **optic chiasma** (G., “optic crossing”) where partial decussation of the optic nerve fibers of the two sides takes place. All ganglion cell axons arising from the **nasal half** of the retina *decussate* (through the central region of the chiasma) to the opposite optic tract. All ganglion cell axons arising from the **temporal half** of the

Table 15.4 ● Sensory receptors.

Cranial nerve	Functional component (modality)	Nucleus	Location of cranial nerve nuclei	Ganglion	Distribution	Function(s)
I Olfactory	SVA	–	Telencephalon	–	Olfactory mucosa	Smell
II Optic	SSA	–	Diencephalon	–	Ganglion cells of retina	Vision
III Oculomotor	GSE	Oculomotor	Mesencephalon (tegmentum)	–	All extraocular muscles except the lateral rectus and superior oblique	Eye movement
	GVE (parasympathetic)	Edinger–Westphal	Mesencephalon (tegmentum)	Ciliary (parasympathetic)	Sphincter pupillae muscle Ciliary muscle	Pupillary constriction Lens accommodation
	GP	Mesencephalic nucleus of the trigeminal	Mesencephalon	–	All extraocular muscles except the lateral rectus and superior oblique	Kinesthetic sense
IV Trochlear	GSE	Trochlear	Mesencephalon (tegmentum)	–	Superior oblique	Eye movement
	GP	Mesencephalic nucleus of the trigeminal	Mesencephalon	–	Superior oblique	Kinesthetic sense
V Trigeminal	SVE	Motor nucleus of the trigeminal	Metencephalon	–	Muscles of mastication: temporalis masseter medial pterygoid lateral pterygoid Mylohyoid, anterior belly of the digastric Tensor tympani	Chewing Tenses tympanic membrane
			Metencephalon (pons)	Trigeminal	Tens veli palatini Scalp, anterior two-thirds of the dura, cornea, conjunctiva, face, paranasal sinuses, teeth, gingiva, and anterior two-thirds of the tongue	Tenses soft palate General sensation
	GSA	Main (chief, principal) nucleus of the trigeminal Spinal nucleus of the trigeminal	Metencephalon (pons)	Trigeminal	Muscles of mastication Periodontal ligament	Muscle stretch sensation Pressure sensation
			Metencephalon (pons to C3)	–	–	–
VI Abducent	GSE	Abducens	Metencephalon (pons)	–	Lateral rectus	Eye movement
	GP	Mesencephalic nucleus of the trigeminal	Mesencephalon	–	Superior oblique	Kinesthetic sense
VII Facial	SVE	Facial	Metencephalon (pons)	–	Muscles of facial expression, platysma, posterior belly of the digastric, and stylohyoid	Facial expression
			Myelencephalon	Pterygopalatine (parasympathetic)	Stapedius Lacrimal gland Glands of the nasal cavity and palate	Tension on stapes Lacrimation Mucous secretion
	GVA	Solitarius	Myelencephalon	Submandibular (parasympathetic)	Submandibular and sublingual glands	Salivation
			Myelencephalon	Geniculate	Anterior two-thirds of the tongue	Taste
GVA	Solitarius	Myelencephalon	Geniculate	Middle ear, nasal cavity, and soft palate	Visceral sensation	

Table 15.4 ● Continued.

Cranial nerve	Functional component (modality)	Nucleus	Location of cranial nerve nuclei	Ganglion	Distribution	Function(s)
VIII Vestibulocochlear	GSA	Spinal nucleus of the trigeminal	Metencephalon (pons)	Geniculate	External auditory meatus and area posterior to ear	General sensation
	Cochlear SSA	Dorsal and ventral cochlear	Myelencephalon	Spiral	Organ of Corti (inner ear)	Hearing
	Vestibular SSA	Vestibular complex	Myelencephalon	Vestibular	Utricle Saccule Semicircular canal ampullae (inner ear)	Equilibrium
X Glossopharyngeal	SVE	Ambiguous	Myelencephalon	–	Stylopharyngeus and pharyngeal constrictors	Swallowing
	GVE (parasympathetic) SVA	Inferior salivatory Solitarius	Myelencephalon Myelencephalon	Otic (parasympathetic) Inferior ganglion of the glossopharyngeal	Parotid gland Posterior one-third of the tongue and adjacent pharyngeal wall	Salivation Taste
	GVA	Ambiguous Solitarius	Myelencephalon	Inferior ganglion of the glossopharyngeal	Middle ear, pharynx, tongue, carotid sinus	Visceral sensation
	GSA	Spinal nucleus of the trigeminal	Myelencephalon	Superior ganglion of the glossopharyngeal	Posterior one-third of the tongue, soft palate, upper pharynx, and auditory tube	General sensation
X Vagus	GVE (parasympathetic)	Dorsal motor nucleus of the vagus	Myelencephalon	Thoracic and abdominal submucosal and myenteric autonomic plexuses	Thoracic and abdominal viscera	Gland secretion, peristalsis
	SVE	Ambiguous	Myelencephalon	–	Muscles of the larynx and pharynx	Phonation
	SVA	Solitarius	Myelencephalon	Inferior (nodose)	Epiglottis	Taste
	GVA	Solitarius	Myelencephalon	Inferior (nodose)	Thoracic and abdominal viscera Carotid body	Visceral sensation
	GSA	Spinal nucleus of the trigeminal	Myelencephalon	Superior (jugular)	Area posterior to the ear, external acoustic meatus, and posterior part of meninges	General sensation
XI Spinal accessory	SVE	Ambiguous	Myelencephalon	–	Laryngeal muscles To sternocleidomastoid and trapezius	Phonation Head and shoulder movements
XII Hypoglossal	GSE	Hypoglossal	Myelencephalon	–	Muscles of the tongue	Tongue movement

retina proceed (through the lateral aspect of the chiasma) *without decussating* and join the optic tract of the same side. The ganglion cell axons coursing in each optic tract curve around the cerebral peduncle to terminate and relay visual input in one of the following four regions of the brain: the **lateral geniculate nucleus**, a thalamic relay station for vision; the **superior colliculus**, a mesencephalic relay station for vision associated with somatic reflexes; the **pretectal area**, a mesencephalic region associated with autonomic reflexes; and the **hypothalamus** (see Figs 16.5, 16.7, 16.9).

OCULOMOTOR NERVE (CN III)

The oculomotor nerve provides motor innervation to four of the six extraocular muscles and the levator palpebrae superioris, and parasympathetic innervation to the sphincter pupillae and ciliary muscles

The oculomotor nerve supplies skeletal motor (somatomotor) innervation to the superior rectus, medial rectus, inferior rectus, and inferior oblique muscles (which move the bulb of the eye) and the levator palpebrae superioris muscle (which elevates the upper eyelid). It also provides parasympathetic

(visceromotor) innervation to the ciliary and sphincter pupillae muscles, two intrinsic smooth muscles of the eye.

The triangular-shaped **oculomotor nuclear complex** is located in the mesencephalon. It is situated ventral to the periaqueductal gray, adjacent to the midline at the level of the superior colliculus. The oculomotor nucleus consists of several **subnuclei** representing each of the extraocular muscles. These subnuclei are composed of groups of nerve cell bodies of the **GSE neurons** that innervate the listed extraocular muscles and the levator palpebrae superioris muscle. The cell group innervating the levator palpebrae superioris is located in the midline, sending motor fibers to this muscle bilaterally (both right and left upper eyelids). The cell group innervating the superior rectus sends projections to the opposite side; whereas the cell group innervating the medial rectus, inferior oblique, and inferior rectus sends projections to the same side.

The **Edinger–Westphal nucleus**, a subnucleus of the oculomotor nuclear complex is located dorsally, medially, and rostral to the GSE nuclear complex. It contains the cell bodies of **GVE preganglionic parasympathetic neurons** whose axons join the GSE fibers as they converge and pass ventrally in the midbrain to emerge from the ventral aspect of the brainstem in the interpeduncular fossa as the oculomotor nerve.

The oculomotor nerve proceeds anteriorly within the cranial vault, travels within the cavernous sinus, and by passing through the superior orbital fissure, enters the ipsilateral orbit. Within the orbit, the oculomotor nerve gives rise to

branches carrying the GSE fibers that innervate the levator palpebrae superioris muscle and all but two of the extraocular muscles. The preganglionic parasympathetic fibers of the oculomotor nerve terminate in the **ciliary ganglion** where they synapse with postganglionic parasympathetic nerve cell bodies. Postganglionic parasympathetic fibers exit the ganglion and reach the sphincter pupillae and ciliary muscles via the short ciliary nerves to provide them with parasympathetic innervation. The parasympathetic fibers, when stimulated, cause contraction of the sphincter pupillae muscle, which results in constriction of the pupil. Pupillary constriction reduces the amount of light that impinges on the retina. Stimulation of the parasympathetic nervous system causes pupillary constriction (whereas stimulation of the sympathetic nervous system, which innervates the dilator pupillae muscle, causes pupillary dilation). Ciliary muscle contraction releases the tension on the suspensory ligaments of the lens, changing its thickness to become more convex. This accommodates the lens for near vision.

GSA pseudounipolar neurons, whose cell bodies reside within the **mesencephalic nucleus** of the trigeminal nerve, send their peripheral processes to terminate in the muscle spindles of the extraocular muscles. These fibers travel via the branches of the ophthalmic division of the trigeminal nerve. GSA (GP) sensory input is transmitted from the muscle spindles via the spindle afferents centrally to the trigeminal nuclear complex, mediating coordinated and synchronized eye movements by reflex and voluntary control of muscles.

CLINICAL CONSIDERATIONS

Unilateral damage to the oculomotor nerve results in deficits in the *ipsilateral* eye. The following ipsilateral muscles will be paralyzed: the levator palpebrae superioris, resulting in **ptosis** (G., “drooping”) of the upper eyelid; the superior and inferior recti, resulting in an inability to move the eye vertically; and the medial rectus, resulting in an inability to move the eye medially. The eye deviates laterally (due to the unopposed lateral rectus), resulting in **lateral strabismus**. This causes the eyes to become misaligned as one eye deviates from the midline, resulting in **horizontal diplopia** (double vision). The inferior oblique is also paralyzed. Since the innervation to the lateral rectus (CN VI) and superior oblique (CN IV) muscles is intact and these two muscles are functional, the eye ipsilateral to the lesion deviates inferiorly and laterally (Fig. 15.3).

The sphincter pupillae muscle becomes nonfunctional due to interruption of its parasympathetic innervation. The pupil ipsilateral to the lesion will remain dilated (**mydriasis**) and does not respond (constrict) to a flash of light. This may be the first clinical sign of intracranial pressure on the GVE fibers of the oculomotor nerve. The ciliary muscle is also nonfunctional due to interruption of its parasympathetic innervation, and cannot accommodate the lens for near vision (that is, cannot focus on near objects).

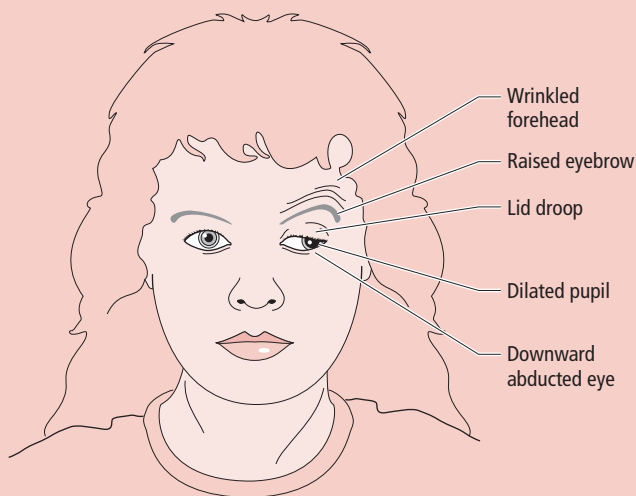


Figure 15.3 • A lesion involving the left oculomotor nerve results in the following symptoms ipsilateral to the side of the lesion: (i) lateral strabismus, (ii) ptosis (drooping of the upper eye lid), (iii) pupillary dilation, (iv) loss of accommodation of the lens, and (v) downward and outward deviation of the eye.

TROCHLEAR NERVE (CN IV)

The trochlear nerve is the smallest (thinnest) cranial nerve and the only one whose fibers originate totally from the contralateral nucleus

The trochlear nerve provides motor innervation to only one of the extraocular muscles of the eye, the superior oblique muscle (a

common mnemonic is SO₄).

The nerve cell bodies of GSE neurons reside in the trochlear nucleus, which lies adjacent to the midline in the tegmentum of the caudal midbrain. Fibers arising from this nucleus initially descend for a short distance in the brainstem and then course dorsally in the periaqueductal gray matter. The fibers *decussate* posteriorly and emerge from the

brainstem at the junction of the pons and midbrain, just below the inferior colliculus.

The trochlear nerve is unique because it is the only cranial nerve whose fibers originate totally from the contralateral nucleus, it surfaces on the *dorsal* aspect of the brainstem, and it is the smallest (thinnest) of the cranial nerves. As the trochlear nerve emerges from the brainstem, it curves around the cerebral peduncle and proceeds anteriorly within the cavernous sinus to pass into the orbit via the superior orbital fissure. Consequently, this cranial nerve has the longest intracranial course and is highly susceptible to increased intracranial pressure.

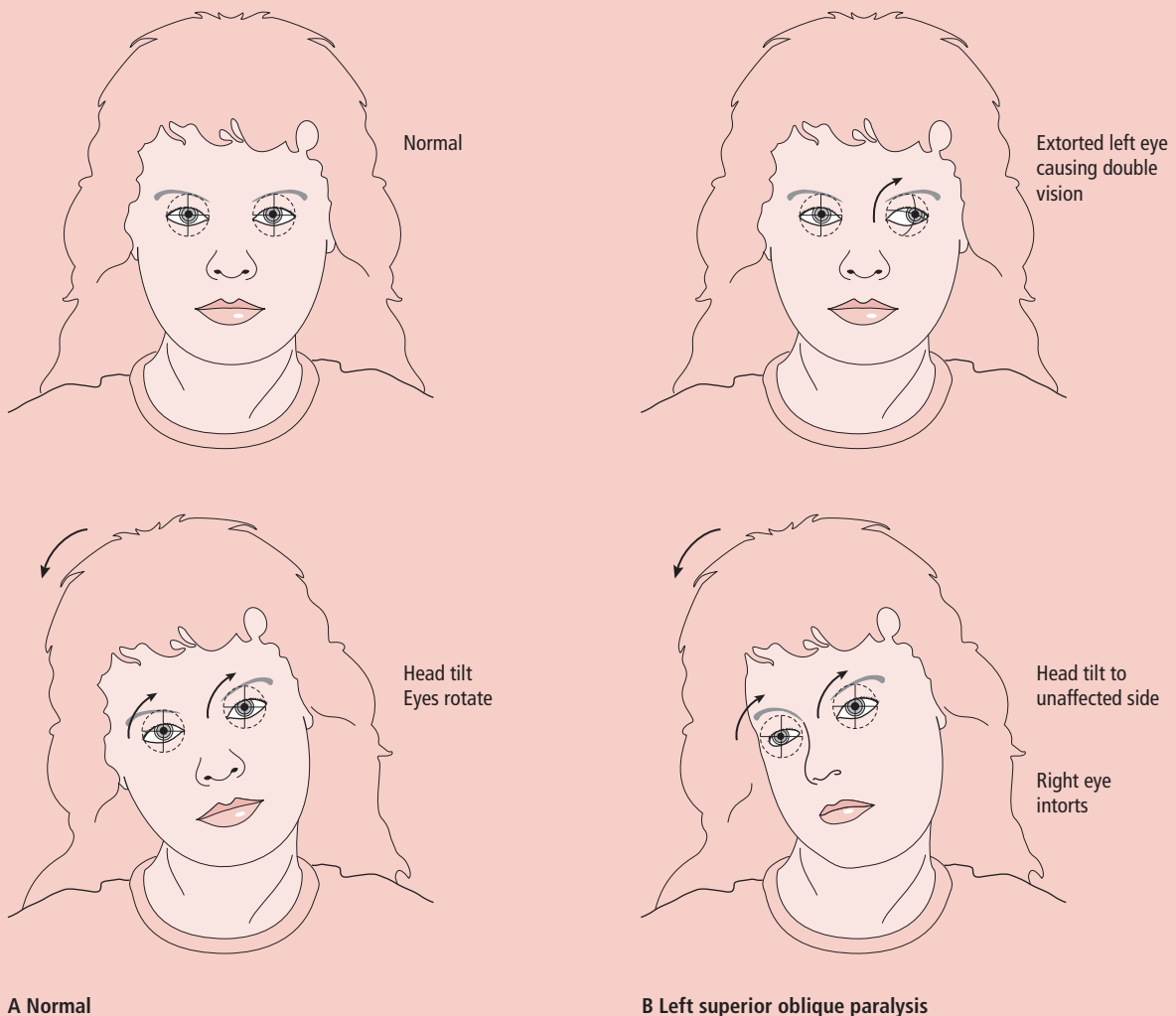
CLINICAL CONSIDERATIONS

Figure 15.4 ● (A) Normal: When the head is tilted, the eyes rotate in the opposite direction. (B) Left superior oblique paralysis following a lesion to the trochlear nerve: the affected eye becomes extorted with consequent double vision. To minimize the double vision, the individual tilts her head toward the unaffected side which intorts the normal eye.

CLINICAL CONSIDERATIONS (continued)

Damage to the trochlear nucleus results in **paralysis** or **paresis** of the *contralateral* superior oblique muscle, whereas damage to the **trochlear nerve** results in the same deficits but in the *ipsilateral* muscle.

Normally, contraction of the superior oblique muscle causes the eye to *intort* (rotate inward) accompanied by simultaneous *depression* (downward) and *lateral* (outward) movement of the bulb of the eye. This is sometimes referred to as the “Salvation Army muscle” (“down and out”). Intorsion of the eyeball is the turning of the eyeball around its axis, so that the superior pole of the eyeball turns inward. Imagine that extreme intorsion (which we really cannot do) will bring the superior pole of the eye facing the medial wall of the orbit. When the superior oblique muscle is paralyzed, the *ipsilateral* eye will *extort* (rotate outward) accompanied by simultaneous *upward* and *outward* movement of the eye (Fig. 15.4B). This is caused by the unopposed inferior oblique muscle and results in **external strabismus**.

Since the eyes become misaligned following such a lesion, an individual with **trochlear nerve palsy** experiences **vertical diplopia** (double vision), accompanied by weakness of downward movement of the eye, most notably in an effort to adduct the eye (turn medially). The diplopia is most apparent to the individual when descending stairs or while reading (looking down and inward). To counteract the diplopia and to restore proper eye alignment, the individual realizes that the diplopia is reduced as he tilts his head towards the side of the unaffected eye (Fig. 15.4B). Normally, tilting of the head to one side elicits a reflex rotation about the anteroposterior axis of the eyes in the opposite direction (Fig. 15.4A), so that the image of an object will remain fixed on the retina. Tilting of the head toward the *unaffected* side causes the unaffected eye to rotate inward and become aligned with the affected eye which is rotated outward. Also, pointing the chin downward (“chin tuck”) rolls the normal eye upward.

TRIGEMINAL NERVE (CN V)

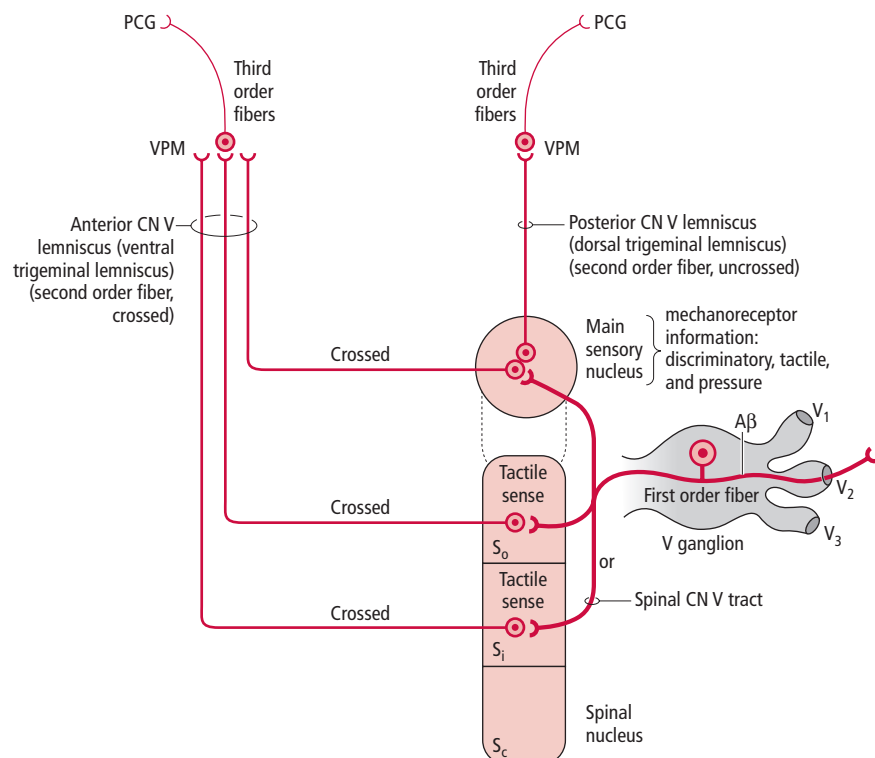
The trigeminal nerve, the largest of the cranial nerves, provides the major general sensory innervation to part of the scalp, most of the dura mater, and the orofacial structures

The trigeminal system consists of the trigeminal nerve, ganglion, nuclei, tracts, and central pathways. The trigeminal sensory pathway, which transmits *touch*, *nociception*, and *thermal sensation*, consists of a three neuron sequence (first, second, and third order neurons) from the periphery to the cerebral cortex respectively (Figs 15.5, 15.6).

The peripheral processes of the first order neurons radiating from the trigeminal ganglion gather to form three separate

nerves, the three divisions of the trigeminal nerve whose peripheral endings terminate in sensory receptors of the orofacial region. Their cell bodies are housed in the trigeminal ganglion. The central processes of these neurons enter the pons, join the spinal tract of the trigeminal, and terminate in the trigeminal nuclei where they establish synaptic contacts with second order neurons housed in these nuclei. The trigeminal nuclei, with the exception of the mesencephalic nucleus, contain second order neurons as well as interneurons. The second order neurons give rise to fibers that may or may not decussate in the brainstem and join the ventral or dorsal trigeminal lemnisci. These lemnisci ascend to relay

Figure 15.5 • The trigeminal pathway for touch and pressure. Touch and pressure sensation from the orofacial structures is transmitted to the brainstem trigeminal nuclei, the main sensory nucleus, and the spinal nucleus via the central processes of first order pseudounipolar neurons whose cell bodies are located in the trigeminal ganglion. Second order neurons in these nuclei form the posterior and anterior trigeminal lemnisci which terminate in the ventral posterior medial nucleus of the thalamus (VPM). Third order neurons in the thalamus project to the postcentral gyrus. PCG, postcentral gyrus; S_c , subnucleus caudalis; S_i , subnucleus interpolaris; S_o , subnucleus oralis; V_1 , ophthalmic division of the trigeminal nerve; V_2 , maxillary division of the trigeminal nerve; V_3 , mandibular division of the trigeminal nerve.



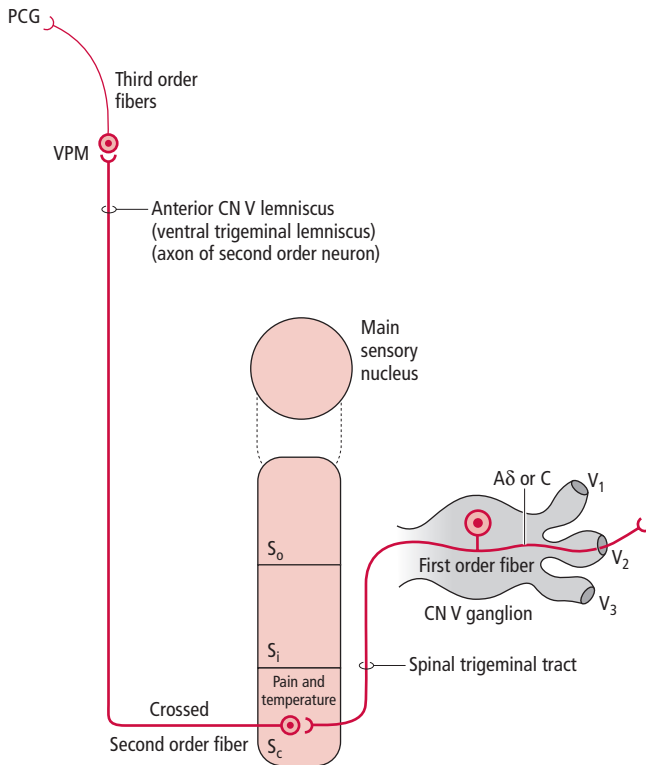


Figure 15.6 ● The trigeminal pathway for pain and temperature. Pain and temperature sensation from the orofacial structures is transmitted to the brainstem subnucleus caudalis (S_c) of the spinal trigeminal nucleus via the central processes of first order pseudounipolar neurons whose cell bodies are located in the trigeminal ganglion. Second order neurons from the subnucleus caudalis join the anterior trigeminal lemniscus to terminate in the ventral posterior medial nucleus of the thalamus (VPM). Third order neurons from the VPM terminate in the postcentral gyrus (PCG). For other abbreviations, see Fig. 15.5.

trigeminal sensory input to the ventral posterior medial (VPM) nucleus of the thalamus, where they synapse with third order neurons. The third order neurons then relay sensory information to the postcentral gyrus (somesthetic cortex) of the cerebral cortex for further processing.

The **trigeminal nerve** is the largest cranial nerve. It provides the major **GSA** innervation (touch, pressure, nociception, and thermal sense) to part of the scalp, most of the dura mater, the conjunctiva and cornea of the eye, the face, nasal cavities, paranasal sinuses, palate, temporomandibular joint, lower jaw, oral cavity, and teeth. It also provides **SVE (branchiomotor)** innervation to the muscles of mastication (temporalis, masseter, medial pterygoid, lateral pterygoid), and the mylohyoid, anterior belly of the digastric, tensor tympani, and tensor veli palatini muscles.

The trigeminal nerve is the only cranial nerve whose sensory root enters and motor root exits at the ventrolateral aspect of the pons (see Fig. 15.1). The larger, **sensory root** consists of the central processes (axons) of the pseudounipolar sensory neurons of the trigeminal ganglion. These axons enter the pons to terminate in the trigeminal sensory nuclear complex of the brainstem. The **motor root** is smaller and consists of the axons of motor (branchiomotor) neurons exiting

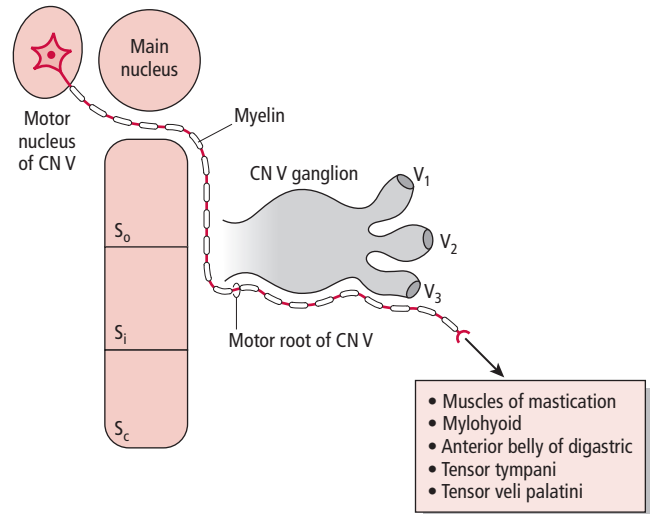


Figure 15.7 ● Branchiomotor innervation of the trigeminal nerve. The motor nucleus of the trigeminal nerve contains the motoneurons whose axons assemble to form the motor root of the trigeminal nerve. The motor root exits the pons and joins the mandibular division of the trigeminal nerve and distributes to the muscles of mastication, the mylohyoid, the anterior belly of the digastric, the tensor tympani, and the tensor veli palatini muscles to provide them with motor innervation. For abbreviations, see Fig. 15.5.

the pons (Fig. 15.7). The motor root joins the sensory portion of the mandibular division of the trigeminal nerve just outside the skull, to form the mandibular trunk. Before the motor root joins it, the trigeminal nerve displays a swelling, the **trigeminal ganglion**, which lies in a bony depression of the petrous temporal bone on the floor of the middle cranial fossa. Since this is a sensory ganglion there are no synapses occurring here. As the peripheral processes of the pseudounipolar neurons exit the ganglion, they form three divisions (hence “trigeminal,” meaning the “three twins”). These divisions traverse the foramina of the skull to exit the cranial vault on their way to reach the structures they innervate. The **ophthalmic division** is purely sensory and innervates the upper part of the face; the **maxillary division** is also purely sensory (although there may be some exceptions) and innervates the middle part of the face. The **mandibular division** is mixed, that is it carries sensory innervation to the lower face and branchiomotor innervation to the muscles listed above.

Trigeminal nuclei

The trigeminal system includes four nuclei: one *motor* nucleus, the motor nucleus of the trigeminal; and three *sensory* nuclei, the main (chief, principal) sensory nucleus of the trigeminal, the mesencephalic nucleus of the trigeminal, and the spinal nucleus of the trigeminal (see Fig. 15.2; Table 15.5).

Motor nucleus

The motor nucleus of the trigeminal nerve contains the cell bodies whose axons form the motor root of the trigeminal nerve, which provides motor innervation to the muscles of mastication

Table 15.5 • The trigeminal nuclei.

Motor nucleus
Sensory nuclei:
• Main (chief, principal) nucleus of the trigeminal
• Mesencephalic nucleus of the trigeminal
• Spinal nucleus of the trigeminal:
Subnucleus oralis
Subnucleus interpolaris
Subnucleus caudalis

The **motor nucleus of the trigeminal** is located at the midpontine levels, medial to the main sensory nucleus. It contains interneurons and the cell bodies of **multipolar** alpha and gamma motor (**branchiomotor**) neurons whose axons form the **motor root** of the trigeminal nerve as they exit the pons. The branchiomotor fibers join the mandibular division of the trigeminal nerve and are distributed to the muscles of mastication as well as to the mylohyoid, anterior belly of the digastric, tensor tympani, and tensor veli palatini muscles.

Sensory nuclei

The sensory nuclei of the trigeminal nerve transmit sensory information from the orofacial structures to the thalamus

The **sensory nuclei** consist of a long cylinder of cells, which extends from the mesencephalon to the first few cervical spinal cord levels. Two of these nuclei—the main sensory nucleus and the spinal nucleus of the trigeminal—receive the first order afferent terminals of pseudounipolar neurons whose cell bodies are housed in the trigeminal ganglion. These nuclei serve as the **first sensory relay station** of the trigeminal system.

The **main** (chief, principal) **sensory nucleus of the trigeminal nerve** is located in the midpons. Based on its anatomical and functional characteristics, it is homologous to the nucleus gracilis and nucleus cuneatus. It is associated with the transmission of mechanoreceptor information for *discriminatory (fine) tactile* and *pressure sense*.

The **mesencephalic nucleus of the trigeminal** is unique, since it is a true “sensory ganglion” (and not a nucleus), containing cells that are both structurally and functionally ganglion cells. During development, neural crest cells are believed to become embedded within the CNS, instead of becoming part of the peripheral nervous system, as other sensory ganglia. This nucleus houses the cell bodies of **sensory** (first order) **pseudounipolar neurons**, thus there are *no synapses* in the mesencephalic nucleus. The peripheral large-diameter myelinated processes of these neurons convey **GP** input from the muscles innervated by the trigeminal nerve and the extraocular muscles, as well as from the periodontal ligament of the teeth.

The **spinal nucleus of the trigeminal** is the largest nucleus of the three nuclei. It extends from the midpontine region

to level C3 of the spinal cord, and is continuous inferiorly with the dorsal-most laminae (substantia gelatinosa) of the dorsal horn of the spinal cord. This nucleus consists of three subnuclei: the rostral-most subnucleus oralis (pars oralis), the caudal-most subnucleus caudalis (pars caudalis), and the intermediate subnucleus interpolaris (pars interpolaris).

The **subnucleus oralis** merges with the main sensory nucleus superiorly and extends to the pontomedullary junction inferiorly. It is associated with the transmission of discriminative (fine) tactile sense from the orofacial region.

The **subnucleus interpolaris** is also associated with the transmission of tactile sense, as well as dental pain, whereas the **subnucleus caudalis** is associated with the transmission of nociception and thermal sensations from the head. The subnucleus caudalis extends from the level of the obex (medulla) to the C3 level of the spinal cord. It is the homologue of the substantia gelatinosa since their neurons have similar cellular morphology, synaptic connections, and functions. Since the subnucleus caudalis lies immediately superior to the substantia gelatinosa of the cervical spinal cord levels, it is also referred to as the “**medullary dorsal horn**.”

The trigeminal nerve does not have any parasympathetic nuclei in the CNS, or parasympathetic ganglia in the peripheral nervous system. However, it is anatomically associated with the parasympathetic ganglia of other cranial nerves (oculomotor, facial, and glossopharyngeal) and carries their autonomic “hitchhikers” to their destination.

Trigeminal tracts

The trigeminal system includes three tracts: the spinal tract of the trigeminal, the ventral trigeminal lemniscus, and the dorsal trigeminal lemniscus

The **spinal tract of the trigeminal nerve** consists of ipsilateral first order afferent fibers of sensory trigeminal ganglion neurons and mediates *tactile, thermal, and nociceptive sensibility* from the orofacial region to the spinal nucleus of the trigeminal. The spinal tract of the trigeminal also carries first order sensory axons of the facial, glossopharyngeal, and vagus nerves. These nerves terminate in the spinal trigeminal nucleus, conveying GVA or GSA sensory input from their respective areas of innervation to be processed by the trigeminal system. The spinal tract descends lateral to the **spinal nucleus of the trigeminal**, its fibers synapsing with neurons at various levels along the extent of this nucleus. Inferiorly this tract overlaps the dorsolateral fasciculus of Lissauer at upper cervical spinal cord levels.

The **ventral trigeminal lemniscus (ventral trigeminothalamic tract)** consists of mainly crossed nerve fibers from the main sensory and spinal nuclei of the trigeminal. This tract relays mechanoreceptor input for *discriminatory tactile* and *pressure sense* (from the main nucleus) as well as *sharp, well-localized pain* and *temperature* and *nondiscriminatory (crude) touch* sensation (from the spinal nucleus) to the contralateral **ventral posterior medial (VPM) nucleus** of the thalamus.

The **dorsal trigeminal lemniscus (dorsal trigeminothalamic tract)** carries uncrossed nerve fibers from the main sensory nucleus of the trigeminal, relaying *discriminatory*

tactile and *pressure sense* information to the ipsilateral VPM nucleus of the thalamus.

The thalamus also receives indirect trigeminal nociceptive (dull, aching pain) input via the reticular formation (reticulothalamic projections).

Trigeminal pathways

Touch and pressure sense

Nearly half of the sensory fibers in the trigeminal nerve are A β myelinated discriminatory touch fibers. As the central processes of pseudounipolar (first order) neurons enter the pons, they bifurcate into short ascending fibers, which synapse in the **main sensory nucleus**, and long descending fibers, which terminate and synapse mainly in the **subnucleus oralis** and less frequently in the **subnucleus interpolaris** of the spinal nucleus of the trigeminal. These fibers descend in the spinal trigeminal tract to reach their target subnuclei. Some second order fibers from the main sensory nucleus cross the midline and join the ventral trigeminal lemniscus to ascend and terminate in the contralateral VPM nucleus of the thalamus. Other second order fibers from the main sensory nucleus do not cross. They form the dorsal trigeminal lemniscus, and then ascend and terminate in the ipsilateral VPM nucleus of the thalamus. Descending fibers terminating in the subnucleus oralis or interpolaris synapse with second order neurons whose fibers cross the midline and ascend in the ventral trigeminal lemniscus to the contralateral VPM nucleus of the thalamus. The VPM nucleus of the thalamus houses third order neurons that give rise to fibers relaying touch and pressure information to the postcentral gyrus of the cerebral cortex.

Pain and thermal sense

The subnucleus caudalis is involved in the transmission of pain and thermal sensation from orofacial structures

The remaining half of the sensory fibers in the trigeminal nerve are similar to the A δ and C nociceptive and temperature fibers of the spinal nerves. As the central processes of pseudounipolar neurons enter the pons, they descend in the **spinal tract** of the trigeminal and most of them synapse in the **subnucleus caudalis** of the spinal nucleus of the trigeminal. Nociceptive sensory input relayed in the subnucleus caudalis is modified, filtered, and integrated prior to its transmission to higher brain centers.

Interneurons located in the subnucleus caudalis project superiorly to the subnucleus oralis and interpolaris of the spinal nucleus and to the main sensory nucleus of the trigeminal, where they modulate the synaptic activity and relay of sensory input from all of these nuclei to higher brain centers. Furthermore, interneurons residing in the subnucleus oralis and interpolaris project to the subnucleus caudalis where they may in turn modulate the neural activity there.

Most of the second order fibers from the subnucleus caudalis cross the midline and join the contralateral ventral

trigeminal lemniscus, whereas others join the ipsilateral ventral trigeminal lemniscus. All the fibers ascend to the VPM nucleus of the thalamus where they synapse with third order neurons in that nucleus. The fibers of third order neurons ascend in the posterior limb of the internal capsule to relay somatosensory information from the trigeminal system to the **postcentral gyrus** of the somatosensory cortex for further processing.

Electrophysiological observations have indicated that electrical stimulation of the midbrain periaqueductal gray matter, the medullary raphe nuclei, or the reticular nuclei, has an inhibitory effect on the nociceptive neurons of the subnucleus caudalis.

Substance P, a peptide in the axon terminals of small-diameter first order neurons, has been associated with the transmission of nociceptive impulses. A large number of substance P axon terminals have been located in the subnucleus caudalis. Opiate receptors have also been found in the subnucleus caudalis, which can be blocked by opiate antagonists. These findings indicate that there may be an endogenous opiate analgesic system that could modulate the transmission of nociceptive input from the subnucleus caudalis to higher brain centers.

Motor pathway

The motor root fibers of the trigeminal nerve innervate the muscles of mastication

Branchiomotor neurons housed in the **motor nucleus of the trigeminal** give rise to fibers which, upon exiting the pons, form the **motor root** of the trigeminal nerve (see Fig. 15.7). This short root joins the sensory fibers of the mandibular division of the trigeminal nerve outside the skull. Motor fibers are distributed peripherally via the motor branches of the mandibular division, providing motor innervation to the muscles of mastication (temporalis, masseter, medial pterygoid, lateral pterygoid) and the mylohyoid, anterior belly of the digastric, tensor tympani, and tensor veli palatini muscles.

Mesencephalic neural connections

Pseudounipolar neurons of the mesencephalic nucleus transmit general proprioception input to the main sensory and motor nuclei of the trigeminal and reticular formation

The peripheral processes of the pseudounipolar neurons housed in the **mesencephalic nucleus of the trigeminal** accompany the motor root of the trigeminal as they both exit the pons. These peripheral processes follow: (i) the motor branches of the mandibular division to the muscle spindles of the muscles of mastication; (ii) the orbital branches of the ophthalmic division to the muscle spindles of the extraocular muscles; and (iii) the dental branches of the maxillary and mandibular divisions to the sensory receptors of the periodontal ligament of the maxillary and mandibular teeth, respectively. The central processes of the neurons

transmitting general proprioceptive input from all the muscles and from the periodontal ligament synapse in the **main sensory nucleus** and in the **motor nucleus** of the trigeminal, as well as in the reticular formation to mediate reflex responses.

Jaw jerk (masseteric) reflex

The afferent and efferent limbs of the jaw jerk reflex are formed by the branches of the trigeminal nerve

The **jaw jerk reflex** is a monosynaptic, myotatic (G., “muscle stretch”) reflex for the masseter and temporalis muscles. A hammer gently tapped on the chin causes the intrafusal muscle fibers within the muscle spindles of the (relaxed) masseter and temporalis muscles to stretch, which stimulate the sensory nerve fibers innervating them. The cell bodies of these sensory pseudounipolar neurons are located in the **mesencephalic nucleus of the trigeminal** (Fig. 15.8). Their peripheral processes, which terminate in the muscle spindles (and are carried by branches of the trigeminal mandibular division), form the afferent limb of the reflex arc. The central processes of these neurons synapse in the motor nucleus of the trigeminal bilaterally, as well as in the main sensory nucleus and the reticular formation. The efferent limb of this reflex arc is formed by the motoneuron fibers traveling to the masseter and temporalis muscles (bilaterally, via motor branches of the trigeminal mandibular division) to cause them to contract and compensate for the stretch.

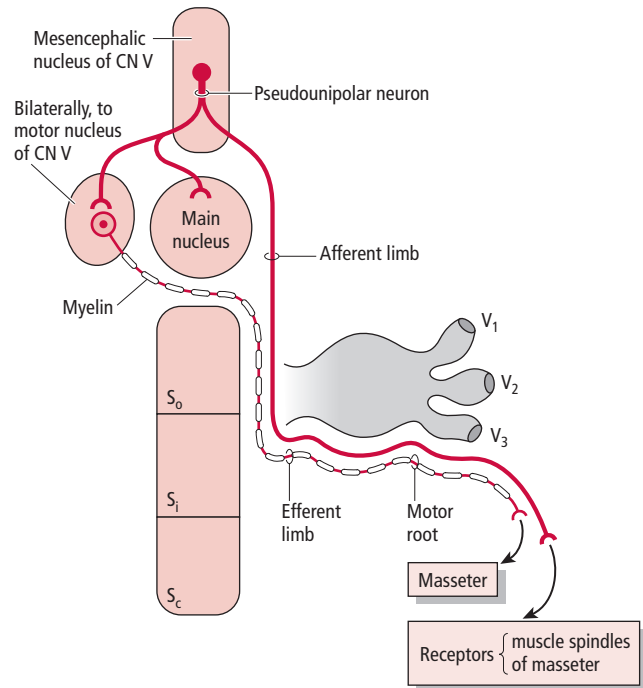


Figure 15.8 • The jaw jerk reflex. The mesencephalic nucleus of the trigeminal nerve contains the nerve cell bodies of pseudounipolar neurons whose peripheral processes terminate in the muscle spindles of the masseter muscle. Sensory information (about muscle stretch) is carried by the central processes of these neurons to the ipsilateral main sensory and bilaterally to the motor nucleus of the trigeminal. The motor neurons innervating the masseter muscle cause its contraction. For abbreviations, see Fig. 15.5.

CLINICAL CONSIDERATIONS

Skull fractures may cause a unilateral lesion of the branchiomotor fibers to the muscles of mastication, which will result in a **flaccid paralysis** or **paresis** with subsequent muscle **atrophy** of the ipsilateral muscles of mastication. This becomes apparent upon muscle palpation when the patient is asked to clench his jaw. When depressing the lower jaw it deviates *towards* the affected side (weak side) primarily due to the unopposed action of the lateral pterygoid muscle of the unaffected side. This impairs chewing on the lesion side due to muscle paralysis.

Damage to the fibers innervating the tensor tympani muscle results in **hyperacusis** (acute sense of hearing) and impaired hearing on the ipsilateral side.

Damage to the GSA fibers of the mandibular division will result in loss of sensation from the areas supplied by the branches of this division. Although the trigeminal nerve has an extensive distribution in the head, there is minimal overlapping of the areas innervated by its three divisions, especially in the central region of the face. Lesions in the peripheral branches of the trigeminal nerve can be located by testing for sensory deficits in the areas that are innervated by each of the three trigeminal divisions. If a lesion is located distal to the joining of the autonomic fibers that hitchhike with the trigeminal branches to the lacrimal gland or the salivary glands, then both sensory and autonomic innervation are interrupted.

Infection of the trigeminal ganglion by herpes zoster virus (known as shingles) causes a significant amount of pain as well as damage to the sensory

fibers of the three trigeminal divisions (the ophthalmic division is most commonly infected). This results in loss of sensation on the affected side. Damage to the sensory fibers innervating the cornea (via the ophthalmic division) results in a loss of the corneal reflex when the ipsilateral eye is stimulated (afferent limb damage of the corneal reflex).

Trigeminal neuralgia (trigeminal nerve pain, tic douloureux)

A common clinical concern regarding the trigeminal nerve is **trigeminal neuralgia**. This condition results from idiopathic etiology (unknown cause) and is manifested as intense, sudden onset, and recurrent unilateral pain in the distribution of one of the three divisions of the trigeminal nerve, most commonly the maxillary division. There may be a trigger zone in the distribution of the affected trigeminal division, and if it is stimulated it may trigger an attack that usually lasts for less than a minute. This condition may be treated pharmacologically or surgically. Surgical treatment includes sectioning of the affected trigeminal division as it emerges from the trigeminal ganglion or producing a lesion in the trigeminal ganglion. Although these procedures may alleviate the excruciating pain experienced by patients, they also abolish tactile sensation from the affected area. Sectioning of the descending spinal trigeminal tract proximal to its termination in the subnucleus caudalis selectively obliterates the afferents relaying nociception but spares the fibers relaying tactile sensation from the orofacial region.



Note that the clinical case at the beginning of the chapter refers to a patient suffering from intermittent excruciating unilateral pain in the lower half of the left side of his face.

- 1 Which cranial nerve provides sensory innervation to the lower half of the face?
- 2 Pain sensation from the lower half of the face is relayed to the brainstem by sensory neurons whose cell bodies are located in which ganglion?
- 3 In which brainstem nucleus is pain sensation from the lower half of the face relayed to?
- 4 Name the thalamic nucleus where pain sensation from the lower half of the face is relayed to.

ABDUCENT NERVE (CN VI)

The abducent nerve innervates only one extraocular muscle, the lateral rectus

The abducent nerve supplies motor innervation to the lateral rectus muscle, which abducts the eye (a common mnemonic is LR₆). The abducent nerve exits the brainstem at the pontomedullary junction, then courses anteriorly, traverses the cavernous sinus, and upon leaving the sinus it passes via the superior orbital fissure into the orbital fossa where it innervates the ipsilateral lateral rectus muscle.

Normally, both eyes move together regardless of the direction of gaze. This is achieved by precise coordinated action of all the extraocular muscles of both eyes. The oculomotor, trochlear, and abducens nuclei are interconnected and are controlled by higher brain centers of the cerebral cortex as well as by the brainstem. During horizontal gaze, when looking to one side, the lateral rectus muscle of one side and the medial rectus muscle of the contralateral side contract simultaneously.

Abducens nucleus

The abducens nucleus mediates conjugate horizontal movement of the eyes

The abducens nucleus and the internal genu of the facial nerve form an elevation, known as the facial colliculus (L., “little hill”) in the floor of the fourth ventricle. Axons emerging from the abducens nucleus belong to GSE nerve cell bodies. The axons course ventrally in the pontine tegmentum to exit in the ventral aspect of the brainstem at the pontomedullary junction.

The abducens nucleus contains two different populations of neurons (Fig. 15.9). One group (which makes up 70% of the nucleus neurons) consists of the GSE motoneurons, whose axons form the abducent nerve and project to the ipsilateral

lateral rectus muscle. The second group consists of internuclear neurons. Their axons emerge from the nucleus, immediately decussate and project via the contralateral medial longitudinal fasciculus (MLF) to the contralateral oculomotor nucleus. There the internuclear neuron terminals synapse with motoneurons that project to and innervate the medial rectus muscle. The MLF interconnects the abducens, trochlear, and oculomotor nuclei so that the two eyes move in unison. Thus the abducens nucleus mediates conjugate horizontal movement of the eyes.

When higher brain centers stimulate the abducens nucleus the following occur simultaneously:

- 1 Stimulation of the GSE motoneurons of the abducens nucleus that cause the ipsilateral lateral rectus muscle to contract, causing the eye to *abduct*.
- 2 Stimulation of the internuclear neurons of the same abducens nucleus that project, via the contralateral MLF, to the contralateral oculomotor nucleus. Here they form excitatory synapses with the motoneurons projecting to the contralateral medial rectus muscle causing it to contract so that the opposite eye *adducts*, resulting in coordinated lateral gaze.

GSA input from the lateral rectus muscle is transmitted centrally to the trigeminal nuclear complex via the processes of pseudounipolar neurons whose cell bodies are believed to reside in the mesencephalic nucleus of the trigeminal nerve.

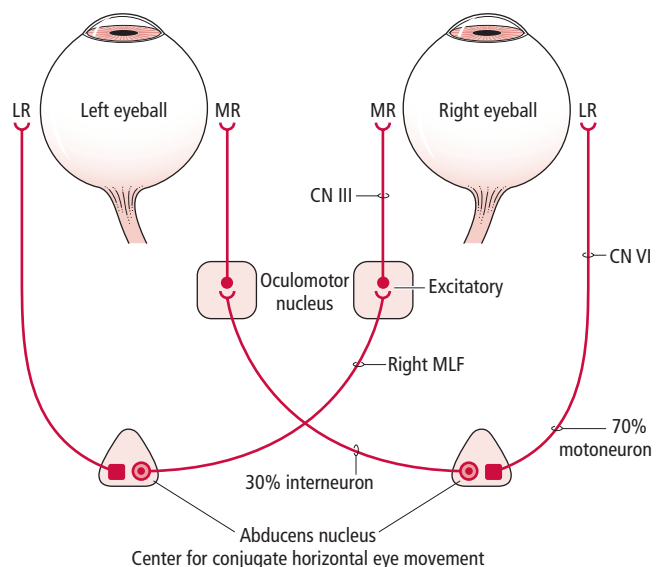


Figure 15.9 ● The connections of the abducens nucleus with the oculomotor nucleus. Note that the abducens nucleus is the center for conjugate horizontal eye movement. It contains two populations of neurons: (i) lower motoneurons whose axons form the abducent nerve that innervates the lateral rectus muscle (LR); and (ii) interneurons whose axons cross the midline and join the contralateral medial longitudinal fasciculus (MLF) to synapse in the oculomotor nucleus with the motoneurons that innervate the medial rectus muscle (MR).

CLINICAL CONSIDERATIONS

Abducent nerve lesion

A lesion in the abducent nerve causes paralysis of the lateral rectus muscle, resulting in medial strabismus and horizontal diplopia

A lesion in the abducent nerve (GSE, motor fibers) results in **paralysis** of the lateral rectus muscle that normally *abducts* the eye. The eye will then deviate medially as a result of the unopposed action of the medial rectus (Fig. 15.10). The individual can turn the ipsilateral eye from its medial position to the center (looking straight ahead), but not beyond it. This paralysis results in **medial strabismus (convergent, internal strabismus, esotropia)**. Since the eyes become misaligned, the individual experiences **horizontal diplopia** (double vision; i.e., a single object is perceived as two separate objects next to each other). The diplopia is greatest in an effort to look toward the side of the lesion and it is reduced by looking towards the unaffected side since the visual axes become parallel. The individual realizes that the diplopia is reduced by turning his head slightly so that his chin is pointing toward the side of the lesion. Bilateral abducent nerve lesion results in the individual becoming "cross-eyed."

Abducens nucleus lesion

A lesion involving the abducens nucleus results in medial strabismus, horizontal diplopia, and lateral gaze paralysis

A lesion involving the abducens nucleus (Fig. 15.11) results in the same deficiency as a lesion to the abducent nerve, with the addition of the inability to turn the opposite eye medially as the individual attempts to gaze toward the side of the lesion. This condition, referred to as **lateral gaze paralysis**, occurs because the damaged abducens nucleus no longer provides excitatory input to the opposite oculomotor nucleus neurons that innervate the medial rectus muscle.

Unilateral medial longitudinal fasciculus lesion: internuclear ophthalmoplegia

A lesion to one MLF results in internuclear ophthalmoplegia

If the oculomotor, trochlear, and abducent nerves and their nuclei are intact, but there is a **unilateral MLF lesion**, eye movements in all directions are possible. However, since the connections between the nuclei of these nerves are interrupted, horizontal ocular movements will not occur in a conjugate fashion.

When there is a lesion of the right MLF, and the individual attempts to gaze to the right, the lesion is not apparent, since both eyes can move simultaneously to the right. However, when attempting to gaze to the left, the right eye cannot move inward (medially beyond the midline) but the left eye, which should move outward (laterally) in this lateral gaze, does since it is not affected. If you ask this same individual to look at a near object placed directly in front of him, which necessitates that both eyes adduct (converge), he is able to do so. This indicates that: (i) both oculomotor nerves (which innervate the medial recti) are intact; and (ii) the upper motoneurons arising from the motor cortex (which stimulate the motoneurons of the oculomotor nuclei) are also intact. Therefore, a unilateral lesion of the MLF becomes apparent only during conjugate horizontal eye movement, when gazing away from the side of the lesion.

"One-and-a-half"

A rare condition resulting from a lesion near the abducens nucleus, involving the ipsilateral abducens nucleus and decussating MLF fibers arising from the contralateral abducens nucleus

A rare condition referred to as "one-and-a-half" results following a lesion in the vicinity of the abducens nucleus, which involves the entire ipsilateral

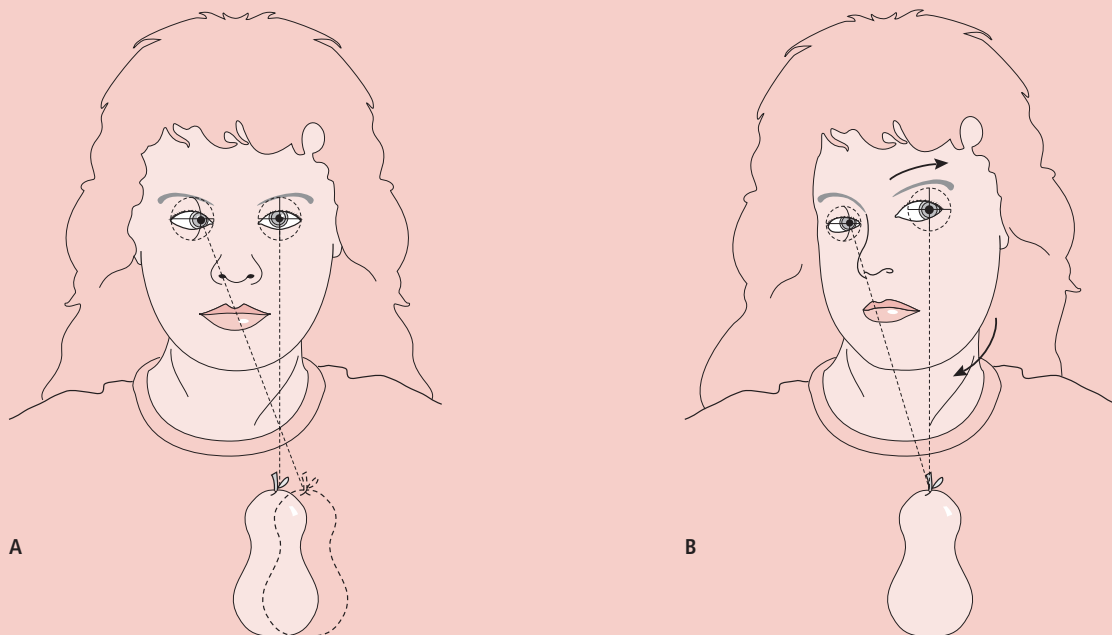


Figure 15.10 • (A) Medial strabismus of the right eye due to paralysis of the lateral rectus muscle, resulting in diplopia (double vision). (B) To minimize the diplopia, the individual turns her head toward the side of the lesion, which abducts the normal eye.

CLINICAL CONSIDERATIONS (continued)

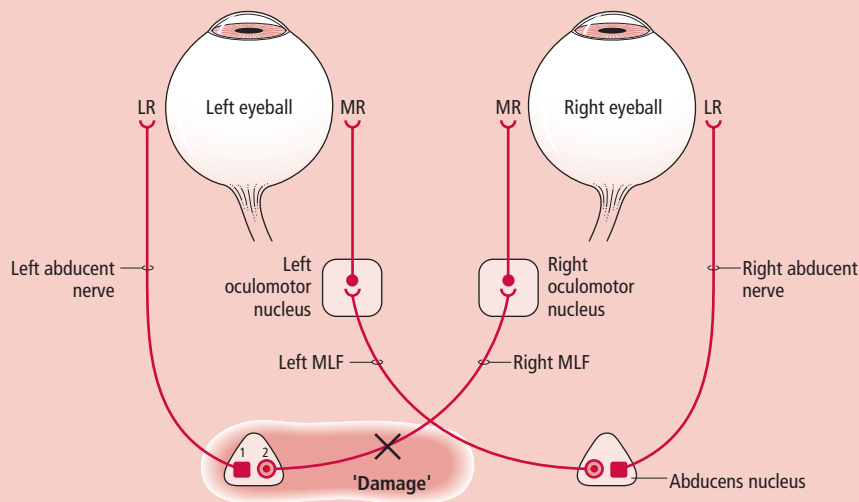


Figure 15.11 ● A lesion of the left abducens nucleus will damage: (i) the lower motoneurons of the abducent nerve, paralyzing the left lateral rectus muscle (LR); and (ii) the interneurons that synapse with the lower motoneurons of the oculomotor nucleus that innervate the right medial rectus muscle (MR). The affected individual is unable to gaze to the side of the lesion (left) during conjugate horizontal eye movement. MLF, medial longitudinal fasciculus.

abducens nucleus as well as the decussating MLF fibers arising from the contralateral abducens nucleus. If a lesion is present in the vicinity of the left abducens nucleus the following things happen:

- 1 The **GSE motoneurons**, whose axons form the left abducent nerve innervating the left lateral rectus, are damaged. Therefore, the left lateral rectus muscle is paralyzed.
- 2 The **internuclear neurons** housed in the left abducens nucleus are also damaged. Their crossing fibers (coursing in the right MLF) do not, therefore, form excitatory synapses with the motoneurons of the contralateral oculomotor nucleus that innervate the right medial rectus muscle.
- 3 The crossing fibers of the internuclear neurons arising from the contralateral (right) abducens nucleus are also damaged; thus they do not

form excitatory synapses with the motoneurons of the left oculomotor nucleus that innervate the left medial rectus.

Therefore, when attempting to gaze to the left, the left eye will not abduct and the right eye will not adduct during conjugate horizontal gaze to the left. When attempting to gaze to the right, the right eye responds normally, that is it is able to abduct, whereas the left eye will not be able to adduct during conjugate horizontal gaze to the right. It is important to note that the innervation to all the extraocular muscles of both eyes is intact, except one—the left lateral rectus. If you ask this individual to look at a near object placed directly in front of him, both eyes will converge, since both medial recti and their innervation (branches of the oculomotor nerve) are intact. Thus this type of lesion becomes apparent only during conjugate horizontal eye movement.

FACIAL NERVE (CN VII)

The facial nerve provides motor innervation to the muscles of facial expression

platysma, the posterior belly of the digastric muscle, the stylohyoid muscle, and the stapedius muscle. It also transmits *taste sensation* from the anterior two-thirds of the tongue, as well as **parasympathetic** (secretomotor) innervation to the lacrimal, submandibular, and sublingual glands. Additionally, it provides *general sensation* to the back of the ear, pinna, and external auditory meatus, as well as *visceral sensation* from the nasal cavity and the soft palate.

The facial nerve consists of two parts: the **facial nerve proper** and the **nervus intermedius**. The facial nerve proper is the motor root of the facial nerve consisting of the axons of **SVE (branchiomotor) neurons** whose cell bodies reside in the **facial nucleus**. This nucleus contains subnuclei, each supplying specific muscles or groups of muscles. The nervus

intermedius is sometimes referred to as the “sensory root,” which is a misnomer since in addition to sensory fibers it also carries parasympathetic fibers. The nervus intermedius consists of the axons of the **GVE (secretomotor) parasympathetic neurons**, whose cell bodies reside in the superior salivatory nucleus. It also contains the central processes of first order, sensory pseudounipolar neurons whose cell bodies are housed in the **geniculate** (L., “bent like a knee”) **ganglion**, the only sensory ganglion of the facial nerve. Some of these pseudounipolar neurons transmit SVA (taste) sensation from the anterior two-thirds of the tongue, others convey GSA sensation from the area posterior to the ear, whereas others carry GVA sensation from the nasal cavity and soft palate.

Both nerve roots (motor root and nervus intermedius) emerge from the brainstem at the cerebellopontine angle. Near their exit from the brainstem, the two roots of the facial nerve accompany one another to the internal acoustic meatus of the petrous portion of the temporal bone and

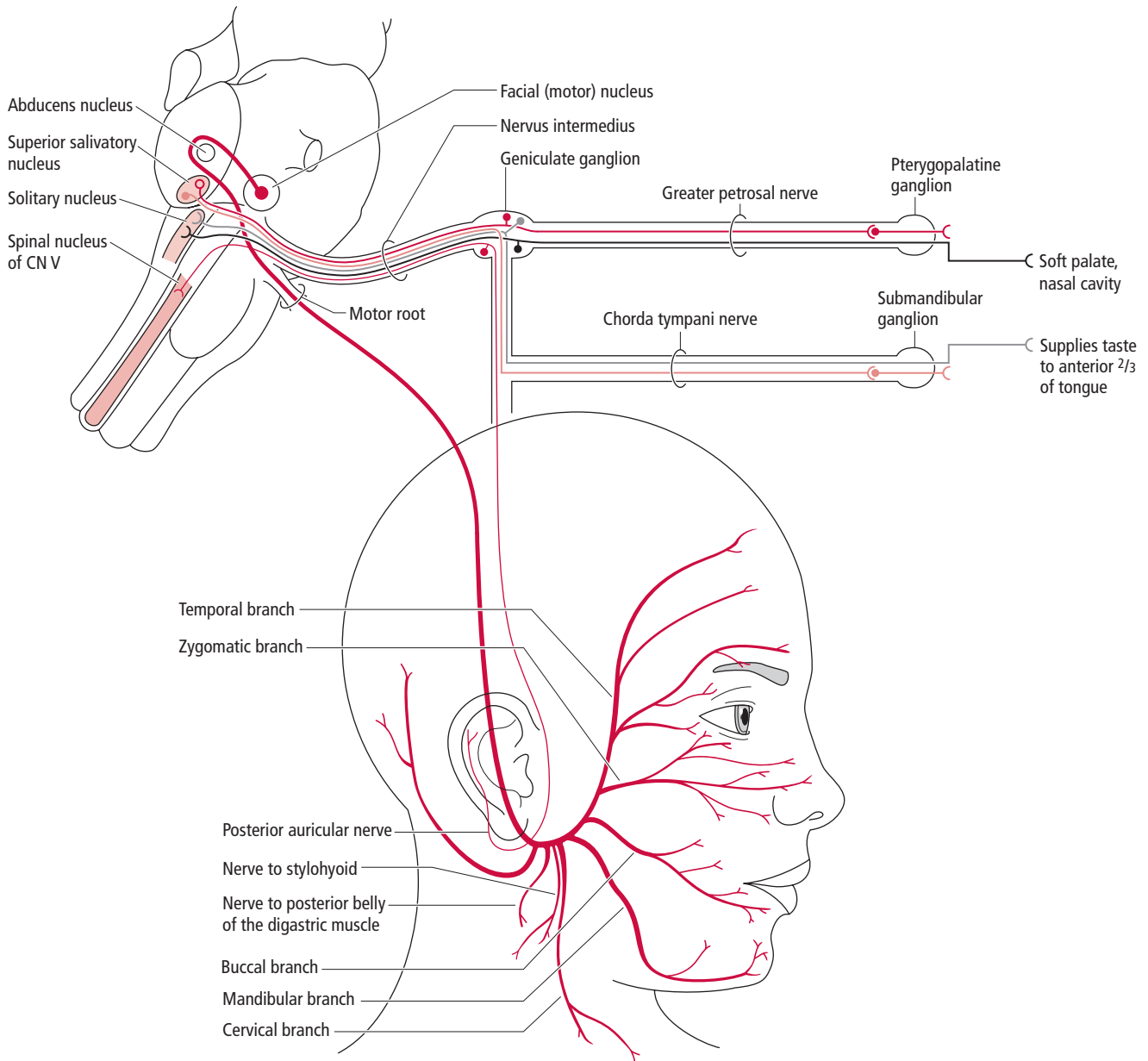


Figure 15.12 • The origin and distribution of the facial nerve and its major branches.

proceed to the facial canal where the nervus intermedius presents a swelling—the **geniculate ganglion**.

The facial nerve gives rise to three of its branches in the facial canal: the greater petrosal nerve, the nerve to the stapedius muscle (which innervates the stapedius muscle in the middle ear), and the chorda tympani nerve. The facial nerve exits the facial canal via the stylomastoid foramen and courses to the parotid bed where its main trunk gives rise to numerous muscular branches, which radiate from within the substance of the gland to innervate their respective muscles (muscles of facial expression, platysma, posterior belly of the digastric, and stylohyoid muscles).

The **superior salivatory nucleus** contains GVE preganglionic parasympathetic nerve cell bodies (Figs 15.12, 15.13)

whose axons leave the brainstem via the nervus intermedius. These preganglionic fibers are distributed by the greater petrosal and chorda tympani nerves. The fibers in the greater petrosal nerve subsequently join the nerve of the pterygoid canal to enter the pterygopalatine fossa where they terminate and synapse in the **pterygopalatine ganglion**, one of the two parasympathetic ganglia of the facial nerve. Postganglionic parasympathetic fibers from this ganglion are distributed to the lacrimal gland and the glands of the nasal and oral cavity to provide them with secretomotor innervation. The **chorda tympani nerve** joins the lingual nerve, a branch of the mandibular division of the trigeminal nerve. The chorda tympani carries preganglionic parasympathetic fibers to the **submandibular ganglion** (the second parasympathetic

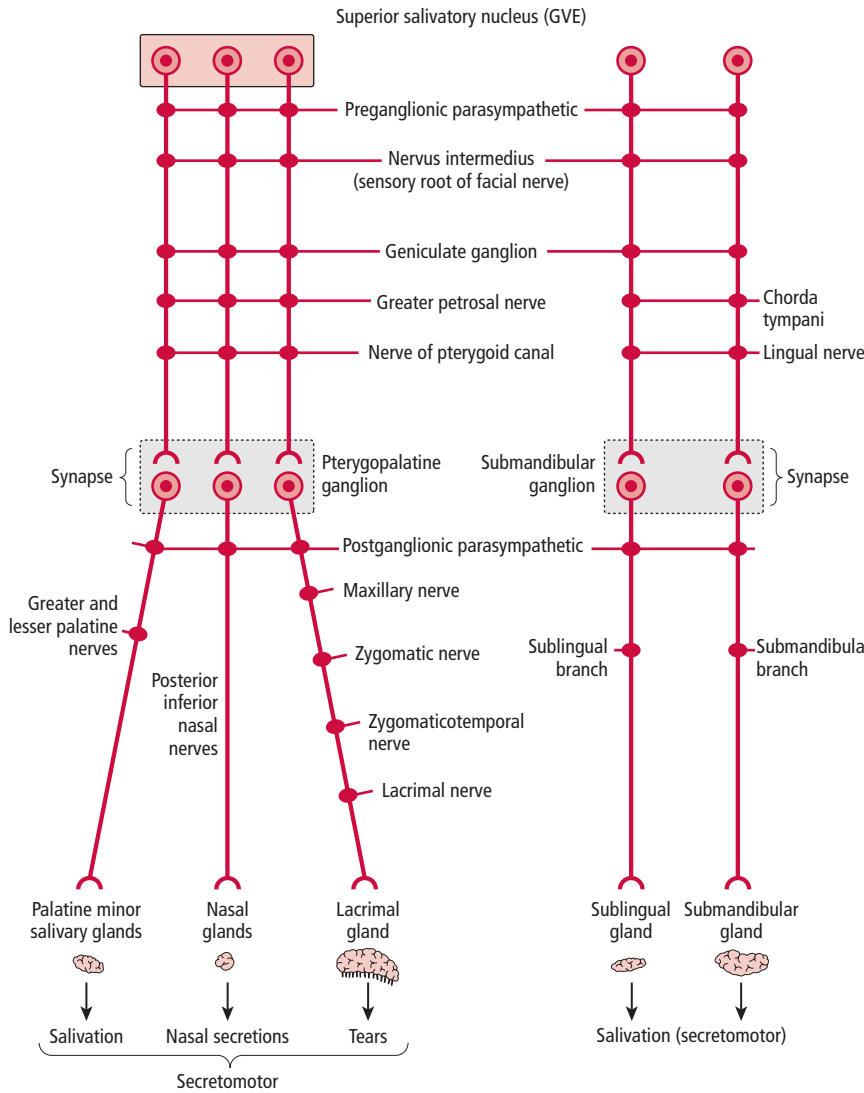


Figure 15.13 ● Parasympathetic innervation of the facial nerve. GVE, general visceral efferent.

ganglion of the facial nerve), where the fibers synapse with its postganglionic parasympathetic neurons. The postganglionic parasympathetic fibers from this ganglion course to the submandibular and sublingual glands providing them with secretomotor innervation.

The geniculate ganglion houses the cell bodies of the SVA neurons, which are responsible for transmission of taste sensation from the anterior two-thirds of the tongue (Fig. 15.14). The peripheral processes of these neurons run in the chorda tympani, and reach the tongue via the lingual nerve of the mandibular division of the trigeminal nerve. The central processes of the SVA neurons enter the brainstem via the nervus intermedius to join the ipsilateral **solitary tract** and terminate in the **solitary nucleus**.

Other pseudounipolar neurons of the geniculate ganglion mediate GVA sensation. Their peripheral processes run in the greater petrosal nerve and terminate in the nasal cavity and the soft palate. Their central processes course in the nervus intermedius, join the ipsilateral solitary tract, and terminate in the solitary nucleus.

Still other pseudounipolar neurons of the geniculate ganglion are responsible for pain, temperature, and touch sensation from the pinna and the external auditory meatus (GSA fibers). The peripheral processes of these neurons terminate in the pinna and the external auditory meatus. Their central processes course in the nervus intermedius and join the **spinal tract of the trigeminal**, and terminate to synapse in the **spinal nucleus of the trigeminal**.

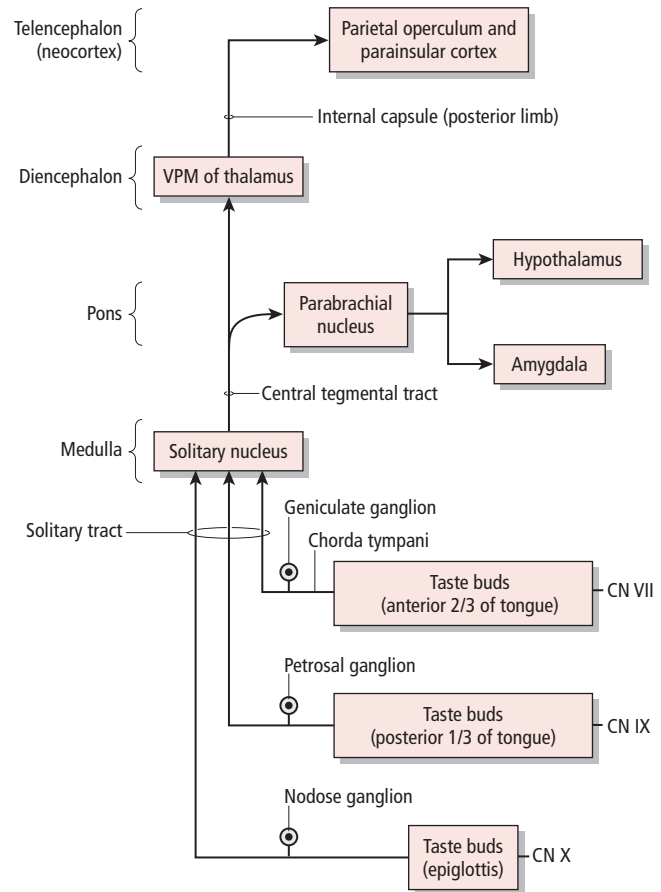


Figure 15.14 • The gustatory pathway. Taste sensation is transmitted by cranial nerves VII (from the anterior two-thirds of the tongue), IX (from the posterior one-third of the tongue), and X (from the epiglottis). Taste sensation is relayed via the solitary tract to the solitary nucleus. The central tegmental tract arising from the solitary nucleus projects to the parabrachial nucleus and to the ventral posterior medial (VPM) nucleus of the thalamus, hypothalamus, and amygdala. The VPM nucleus of the thalamus projects to the gustatory cortex residing in the parietal operculum and the parainsular cortex. (Modified from Fix, JD (1995) *Neuroanatomy*. Williams & Wilkins, Media; fig. 20.2.)

CLINICAL CONSIDERATIONS

A lesion to the facial nerve within the facial canal or near its exit from the stylomastoid foramen causes Bell's palsy

A unilateral lesion of the facial nerve near its root or in the facial canal prior to giving off any of its branches (thus damaging all of its fibers), results in the following conditions *ipsilateral* to the lesion: damage to the SVE (branchiomotor fibers), results in a **flaccid paralysis** or **paresis** (impairment) of the muscles of facial expression, the platysma, stylohyoid, and posterior belly of the digastric muscles with subsequent muscle **atrophy**. The stapedius muscle will also be **paralyzed** and the individual will experience **hyperacusis** (an acute sense of hearing). Usually the stapedius muscle dampens vibrations of the ossicles, but when it is paralyzed, vibrations from the tympanic membrane are transmitted to the ossicles and subsequently to the inner ear receptors for hearing. Furthermore, damage of the SVA fibers relaying taste results in a **loss of taste** from the anterior two-thirds of the tongue. Damage of the GVE parasympathetic fibers causes **decreased salivary secretion** from the submandibular and sublingual glands. Since both parotid glands (innervated by a different cranial nerve) and the contralateral sublingual and submandibular glands remain functional, it is difficult to determine from salivary action alone

whether there is an interruption of the parasympathetic innervation to the ipsilateral submandibular and sublingual glands. In addition, the efferent limb of the corneal blink reflex will be damaged.

Bell's palsy may be idiopathic, or result following trauma or viral infection of the facial nerve within the facial canal or near its exit from the stylomastoid foramen. This condition is characterized by a paresis or paralysis of the muscles of facial expression ipsilateral to the lesion. **Bell's phenomenon** is exhibited by individuals with a Bell's palsy. As the individual attempts to close the eyes, the eye on the affected side deviates up and out.

A unilateral lesion of the facial nerve proximal to the geniculate ganglion causes loss of tear formation by the ipsilateral lacrimal gland. A condition referred to as "**crocodile tear syndrome**" (lacrimation while eating) may result as follows. As the preganglionic parasympathetic ("salivation") fibers originating from the superior salivatory nucleus are regenerating, they may be unsuccessful at finding their way to their intended destination, the submandibular ganglion, and instead take a wrong route to terminate in the pterygopalatine ganglion. The fibers then establish inappropriate synaptic contacts with postganglionic ("lacrimation") neurons whose fibers project to the lacrimal gland.

VESTIBULOCOCHLEAR NERVE (CN VIII)

The vestibular division of CN VIII transmits information about position sense and balance, whereas the cochlear division mediates the sense of hearing

The vestibulocochlear nerve consists of two distinct and separate nerves enclosed within one connective tissue sheath, the **vestibular nerve** (concerned with *position sense* and *balance*) and the **cochlear nerve** (concerned with *hearing*). Both nerves transmit **SSA** information from specialized peripheral ciliated mechanoreceptors (“hair cells”).

The vestibular nerve is the only cranial nerve to send the central processes of some of its first order neurons to synapse directly in the cerebellum

The cell bodies of the **sensory first order bipolar neurons** of the **vestibular nerve** reside within the **vestibular ganglion of Scarpa** (see Fig. 18.6). Their peripheral processes terminate in special receptors, the **cristae** in the ampullae of the semi-circular ducts and the **maculae** of the utricle and saccule, housed within the petrous temporal bone (see Figs 18.2–18.4). The central processes of these neurons enter the brainstem to synapse not only in the vestibular nuclear complex, where they synapse with **second order neurons** of the vestibular pathway, but also in the cerebellum (see Fig. 18.6). The vestibular nerve is unique since it is the only cranial nerve that sends the central processes of some of its first order neurons to synapse *directly* in the cerebellum.

The cell bodies of the sensory first order bipolar neurons of the **cochlear nerve** are housed within the **spiral (cochlear) ganglion** (see Fig. 17.3). Their peripheral processes terminate and synapse in the organ of Corti, containing the special receptors that transduce sound waves into electric impulses. The spiral ganglion and the organ of Corti lie within the cochlea, a snailshell-shaped structure of the inner ear, embedded within the petrous temporal bone (see Fig. 17.3). The central processes of these neurons accompany the seventh vestibular nerve to synapse in the **cochlear nuclei** in the brainstem with second order neurons of the auditory pathway (see Fig. 17.4).

GLOSSOPHARYNGEAL NERVE (CN IX)

The glossopharyngeal nerve provides parasympathetic innervation to the parotid gland

The **glossopharyngeal nerve**, one of the smallest cranial nerves, carries five functional components. These are: (i) **SVA** (taste) and (ii) **GVA** sensation from the posterior one-third of the tongue, the adjacent pharyngeal wall, and the carotid sinus (a baroreceptor or blood pressure receptor located near the bifurcation of the common carotid artery), (iii) **GSA** sensation from the external ear, (iv) **SVE** (branchiomotor) innervation to the stylopharyngeus muscle, and (v) **GVE** parasympathetic innervation to the parotid gland.

The glossopharyngeal nerve exits the brainstem as a group of rootlets posterior to the olive in the dorsolateral sulcus. These rootlets immediately collect to form the main trunk of the glossopharyngeal nerve, which shortly exits the cranial vault via the jugular foramen where it presents two

swellings, its **superior** and **inferior ganglia** (Fig. 15.15). The superior ganglion contains **GSA**, and the inferior ganglion contains **GVA** and **SVA**, cell bodies of **first order pseudounipolar** neurons.

The inferior ganglion of the glossopharyngeal nerve houses the cell bodies of the **SVA (taste)** neurons. Their peripheral processes course with the trunk of the glossopharyngeal nerve to the tongue where they supply the posterior one-third of the tongue and adjacent pharyngeal wall with taste sensation. The central processes of the **SVA** neurons pass into the brainstem via the glossopharyngeal nerve root, join the **solitary tract** and terminate in the **solitary nucleus** (Fig. 15.16A).

GVA first order nerve cell bodies reside in the **inferior ganglion** of the glossopharyngeal nerve. Their peripheral processes terminate in the mucosa of the posterior one-third of the tongue, tonsil and adjacent pharyngeal wall, tympanic cavity, and auditory tube (Fig. 15.16B). Unilateral stimulation of the pharyngeal wall elicits a bilateral contraction of the pharyngeal muscles and soft palate (**gag reflex**). The glossopharyngeal nerve serves as the afferent limb (**GVA** peripheral fibers whose cell bodies are housed in the inferior ganglion), whereas the vagus nerve provides the efferent limb of the reflex arc. The central processes of the afferent fibers enter the solitary tract and synapse in the **nucleus ambiguus**. The nucleus ambiguus sends motor fibers via the vagus nerve to the muscles of the palate and pharynx. Many individuals in the general population do not have a gag reflex.

Baroreceptor fibers terminate in the carotid body and sinus, which form the afferent limb of the reflex arc that controls blood pressure. The central processes of the **GVA** neurons enter the brainstem via the glossopharyngeal nerve root, join the solitary tract and terminate in the solitary nucleus. The solitary nucleus relays sensory input to the reticular formation, the brainstem **GVE** (autonomic) motor nuclei, and the intermediolateral horn (containing preganglionic sympathetic neurons) of the spinal cord for reflex activity related to the control of arterial lumen diameter and blood pressure.

The glossopharyngeal nerve also provides **GSA** touch, pain, and temperature innervation to the pinna of the ear and the external auditory meatus. The cell bodies of these sensory neurons are located in the superior ganglion of the glossopharyngeal nerve. The central processes of these neurons course in the glossopharyngeal nerve root, enter the brainstem, and join the **spinal tract of the trigeminal nerve** to terminate and synapse in the **spinal nucleus of the trigeminal nerve**. Recent clinical evidence supports that fibers transmitting nociceptive sensory input from the pharyngeal wall and posterior one-third of the tongue enter the brainstem and descend in the spinal tract of the trigeminal and terminate in the spinal nucleus of the trigeminal. Furthermore, sensation from oral structures is transmitted via the glossopharyngeal afferent terminals to the **main sensory nucleus** of the trigeminal.

The nucleus ambiguus contains the **SVE branchiomotor** nerve cell bodies whose axons emerge from the brainstem along with rootlets of the glossopharyngeal nerve, and course with the trunk of the glossopharyngeal nerve (Fig. 15.16C). These axons then leave the glossopharyngeal nerve as the

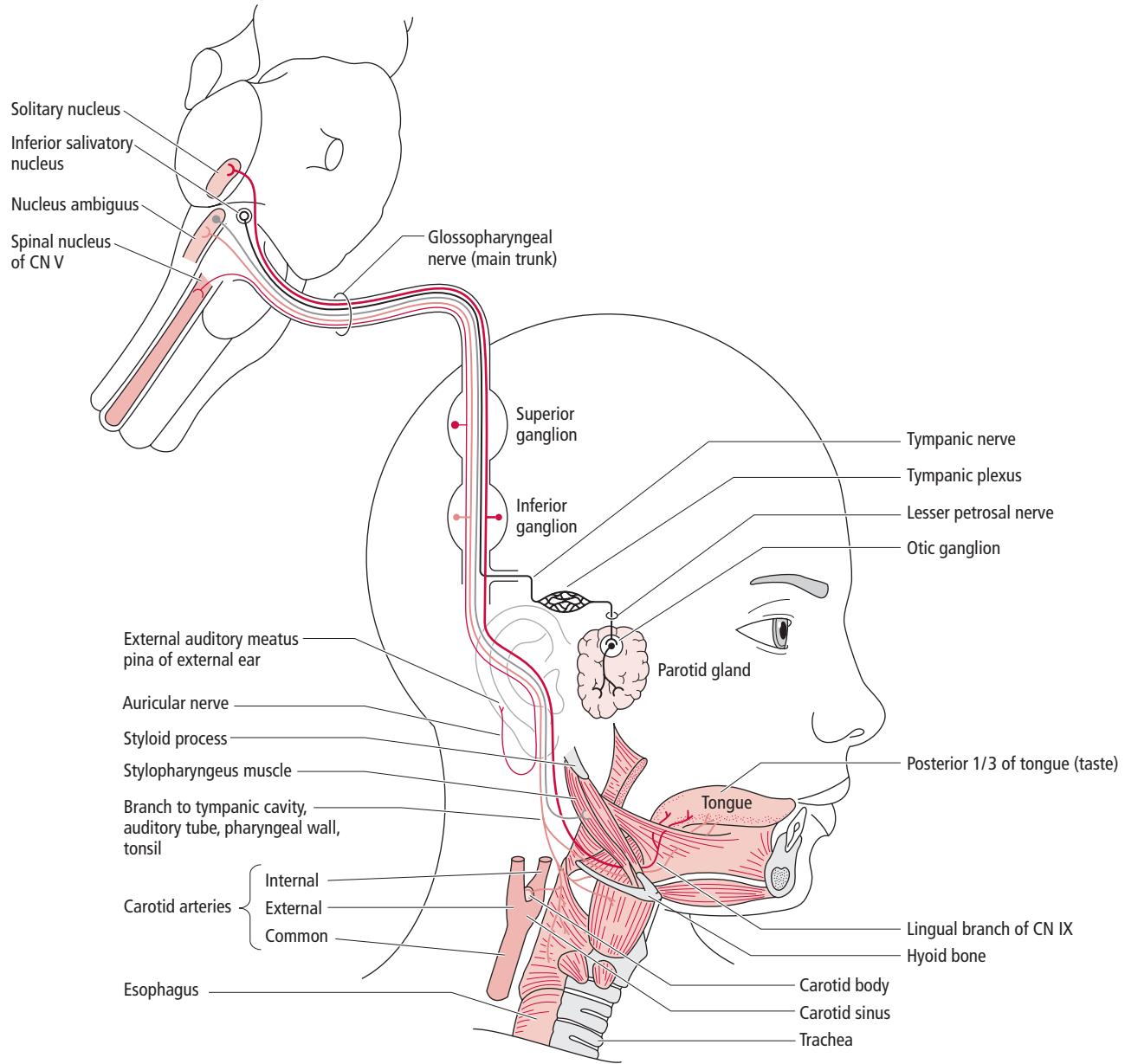


Figure 15.15 • The origin and distribution of the glossopharyngeal nerve and its major branches.

nerve to the stylopharyngeus muscle, the only muscle innervated by the glossopharyngeal nerve.

The **inferior salivatory nucleus**, located in the medulla, contains the **GVE** cell bodies of preganglionic parasympathetic neurons whose axons exit the brainstem as part of the glossopharyngeal nerve (Fig. 15.16D). These fibers then branch off as the tympanic nerve and subsequently spread out to form the tympanic plexus in the tympanic cavity. The preganglionic parasympathetic fibers course to the **otic ganglion**, the parasympathetic ganglion of the glossopharyngeal nerve (located in the infratemporal fossa), where they synapse with postganglionic parasympathetic neurons whose fibers join the auriculotemporal branch of the trigeminal nerve to reach the parotid gland, providing it with secretomotor innervation.

CLINICAL CONSIDERATIONS

A unilateral lesion to the glossopharyngeal nerve near its exit from the brainstem, damaging all of its fibers, will result in damage to the SVA fibers relaying taste sensation and will cause **ipsilateral loss of taste sensation** from the posterior one-third of the tongue. Damage to the GVE parasympathetic fibers will cause a **reduction in salivary secretion** of the parotid gland; and damage to the GVA fibers will result in **diminished visceral sensation** from the pharyngeal mucous membrane, **loss of the gag reflex** (due to damage of the afferent limb of the reflex arc), and **loss of the carotid sinus reflex**. The stylopharyngeus muscle, which elevates the pharynx during swallowing, will be paralyzed.

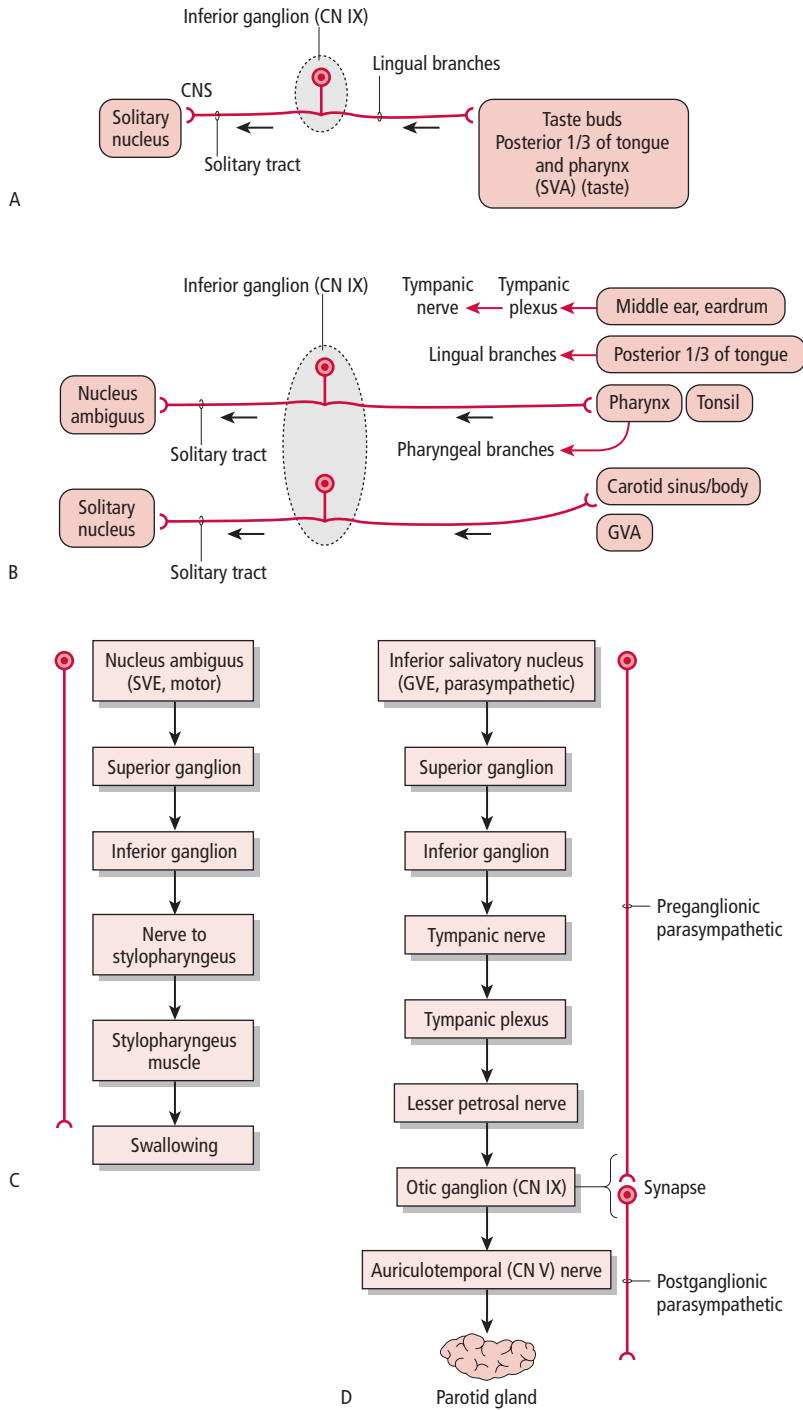


Figure 15.16 ● Innervation by the glossopharyngeal nerve: (A) special visceral afferent (SVA; taste); (B) general visceral afferent (GVA); (C) special visceral efferent (SVE; skeletal motor); and (D) general visceral efferent (GVE; parasympathetic).

VAGUS NERVE (CN X)

The vagus nerve has the most extensive distribution in the body, innervating structures in the head but also the neck, thorax, and abdomen

The vagus nerve carries five functional components: (i) SVA; (ii) GVA; (iii) GSA; (iv) SVE; and (v) GVE (the

The **vagus** (L., “wanderer”) **nerve** (Fig. 15.17) is a large cranial nerve that has the most extensive distribution in the body. Although it is a

same functional components carried by the facial and glossopharyngeal nerves). A group of fine rootlets surface in the medulla in the dorsolateral sulcus, inferior to the glossopharyngeal nerve and superior to the spinal accessory nerve. The rootlets join to form two distinct bundles—a smaller inferior and a larger superior that collectively form the vagus nerve. The inferior bundle joins the spinal accessory nerve and accompanies it for a short distance, but then the two diverge to go their separate ways. The smaller vagal bundle joins the main trunk of the vagus to exit the cranial vault via the

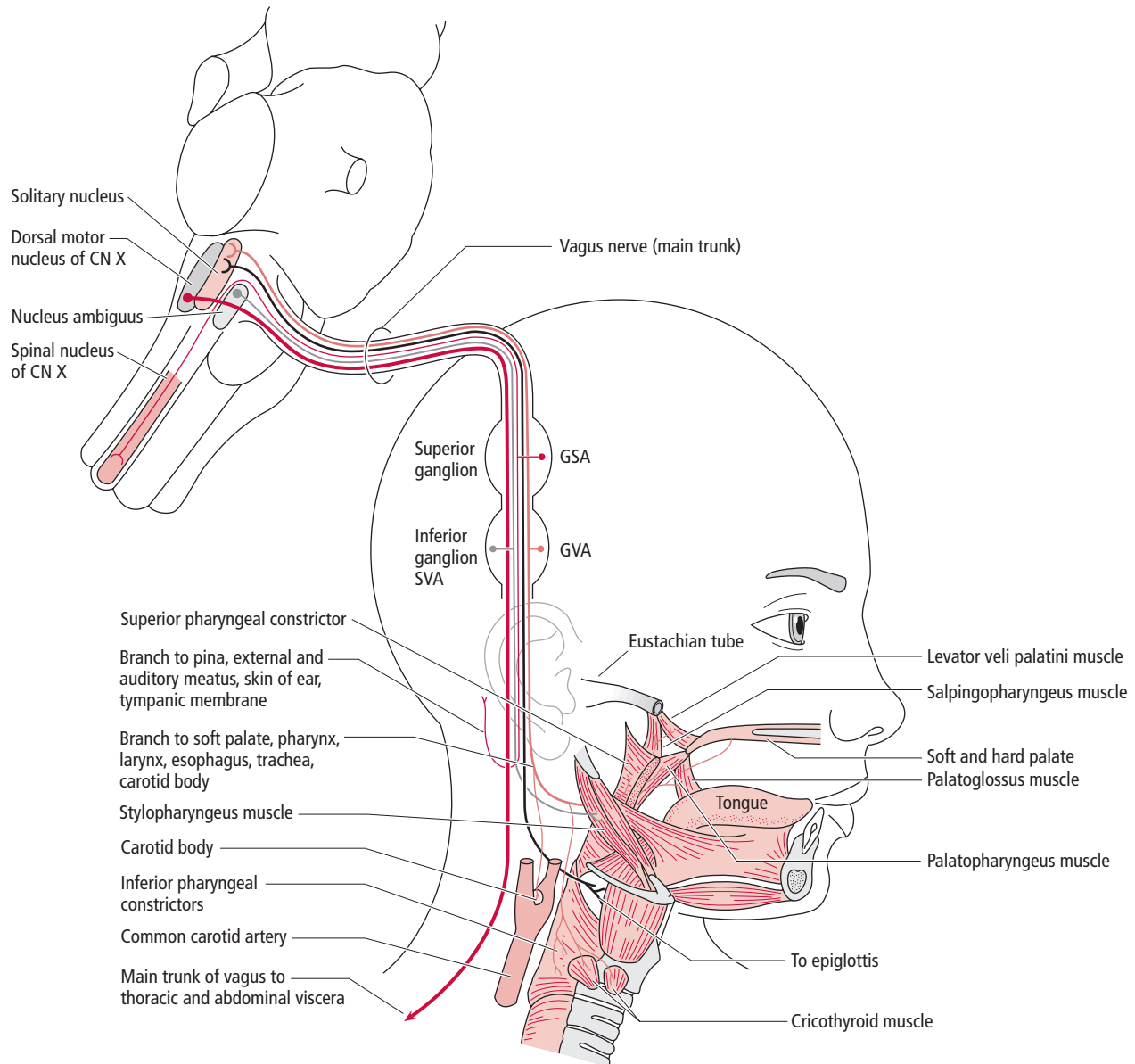


Figure 15.17 • The origin and distribution of the vagus nerve and its major branches. GSA, general somatic afferent; GVA, general visceral afferent; SVA, special visceral afferent.

jugular foramen. Inferior to the jugular foramen, the vagus nerve displays two swellings, the superior (jugular) and inferior (nodose) ganglia. The **superior ganglion** houses the cell bodies of pseudounipolar first order sensory neurons carrying GSA information from the pinna of the ear and external auditory meatus and the dura of the posterior cranial fossa. The **inferior ganglion** contains the pseudounipolar first order nerve cell bodies transmitting GVA sensory innervation from the mucosa of the soft palate, pharynx, and larynx, and a minor SVA (taste) sensation from the epiglottis.

SVA (taste) pseudounipolar neuron cell bodies located in the inferior ganglion of the vagus nerve send their peripheral fibers to terminate in the scant taste buds of the epiglottis. Their central processes enter the brainstem along with the

other vagal fibers to terminate in the **solitary nucleus** (Fig. 15.18A).

GVA pseudounipolar neuron cell bodies housed in the inferior ganglion distribute their peripheral processes in the mucous membranes of the soft palate, and those lining the pharynx, larynx, esophagus, and trachea. Chemoreceptor fibers (GVA also) terminate in the carotid body where they monitor blood carbon dioxide concentration. The central processes of all of the GVA neurons enter the brainstem, course in the **solitary tract** and terminate in the solitary nucleus (Fig. 15.18A).

GSA pseudounipolar neuron cell bodies conveying pain, temperature, and touch sensation reside in the superior ganglion and send their peripheral processes to the pinna,

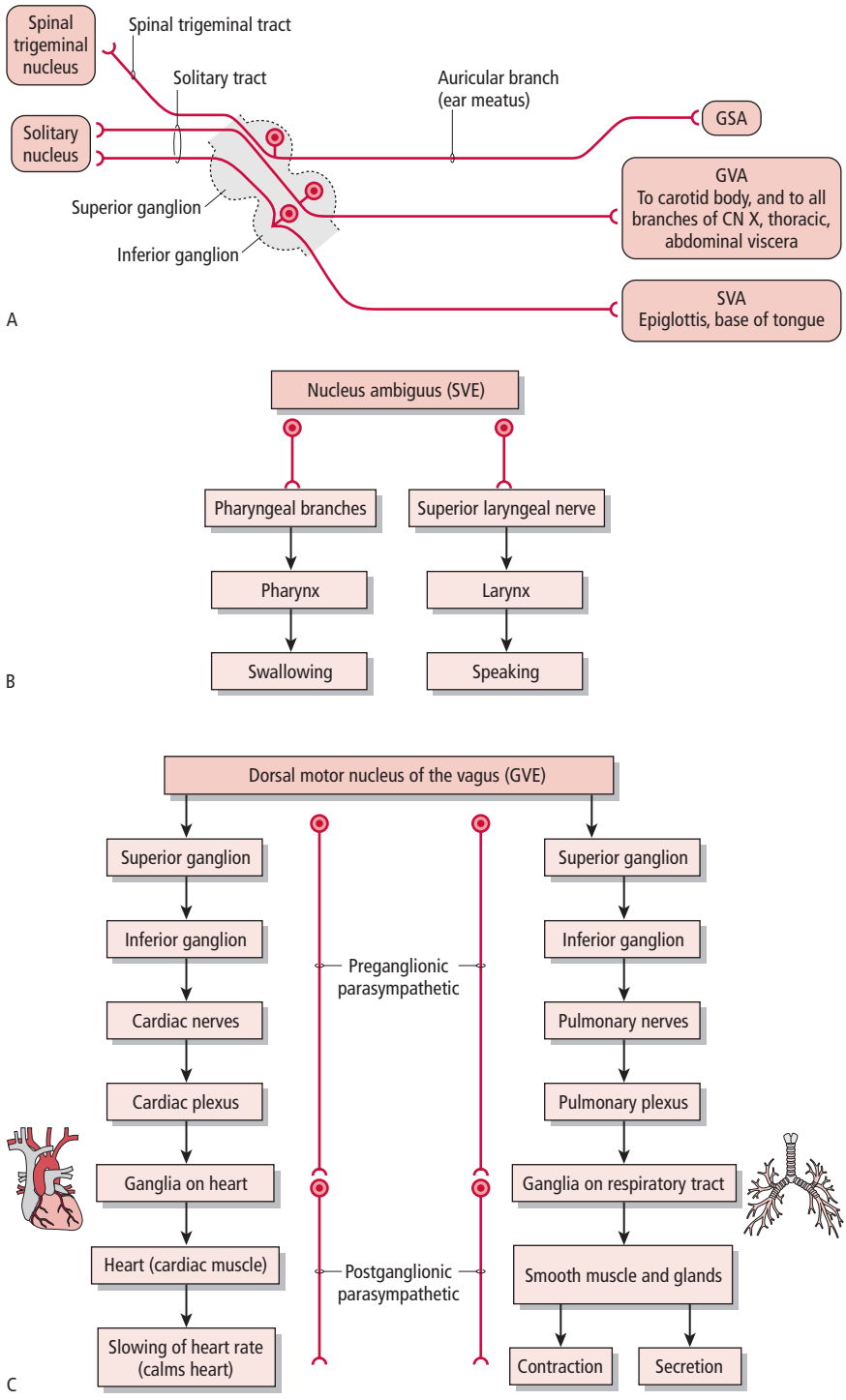


Figure 15.18 ● Innervation by the vagus nerve: (A) general somatic afferent (GSA), general visceral afferent (GVA), and special visceral afferent (SVA; taste); (B) special visceral efferent (SVE; skeletal motor); and (C) general visceral efferent (GVE; parasympathetic).

external auditory meatus, skin of the ear, and tympanic membrane. Their central processes enter the brainstem, join the **spinal tract of the trigeminal** and terminate in the **spinal nucleus of the trigeminal** (Fig. 15.18A).

The cell bodies of the **SVE branchiomotor neurons** are located in the **nucleus ambiguus**. The fibers of these neurons innervate all of the laryngeal and pharyngeal muscles with the exception of the stylopharyngeus and the tensor veli palatini muscles (Fig. 15.18B).

The vagus nerve has a very extensive **GVE** distribution. It supplies parasympathetic innervation to the laryngeal mucous glands and all of the thoracic and most of the abdominal organs. The **dorsal motor nucleus of the vagus** houses the nerve cell bodies of preganglionic parasympathetic neurons whose fibers accompany the other vagal fibers upon their exit from the brainstem. These fibers run in the main trunk of the vagus into the thorax where they leave the main trunk and join the autonomic plexuses scattered throughout

the thoracic and abdominal cavities. The preganglionic fibers terminate and synapse in the terminal parasympathetic ganglia or ganglia near or within the viscera. Parasympathetic innervation decreases the heart rate (calms the heart), reduces adrenal gland secretion, activates peristalsis, and stimulates glandular activity of various organs (Fig. 15.18C).

CLINICAL CONSIDERATIONS

Unilateral damage of the vagus nerve near its emergence from the brainstem results in a number of deficiencies on the ipsilateral side. Damage to the SVE branchiomotor fibers will cause **flaccid paralysis** or **weakness** of: (i) the pharyngeal muscles and levator veli palatini of the soft palate, resulting in **dysphagia** (difficulty swallowing); (ii) the laryngeal muscles, resulting in **dysphonia** (hoarseness) and **dyspnea** (difficulty breathing); and (iii) **loss of the gag reflex** (efferent limb). Damage to the GVA fibers will cause **loss of general sensation** from the soft palate, pharynx, larynx, esophagus, and trachea. Damage to the GVE fibers will cause **cardiac arrhythmias**.

A bilateral lesion of the vagus nerve is incompatible with life, due to the interruption of parasympathetic innervation to the heart.

SPINAL ACCESSORY NERVE (CN XI)

The spinal accessory nerve supplies motor innervation to the sternocleidomastoid, trapezius, and many of the intrinsic laryngeal muscles

The **spinal accessory nerve** (Fig. 15.19) supplies motor innervation to the sternocleidomastoid, trapezius, and many of the intrinsic laryngeal muscles.

In the early literature this nerve was described as consisting of two distinct parts: a cranial (bulbar) and a spinal root. It is now understood that the “cranial root” of the accessory nerve is composed of *aberrant vagal fibers* arising from the **nucleus ambiguus** in the medulla. These vagal fibers collectively form a distinct root as they emerge from the brainstem. On the other hand, the **spinal accessory nerve** derives its fibers from the **spinal accessory nucleus** residing in the posterolateral aspect of the ventral horns of cervical spinal cord levels C2–C5 (or C6). This nucleus is continuous superiorly with the nucleus ambiguus of the medulla. Delicate rootlets emerging from the surface of the lateral funiculus of the spinal cord (interposed between the dorsal and ventral spinal roots) converge and assemble to form the spinal accessory nerve. This nerve trunk ascends, enters the cranial vault through the foramen magnum, and proceeds on the lateral aspect of the medulla to join the aberrant vagal fibers as they emerge from the medulla. The two groups of fibers accompany one another for a short distance but then diverge to go their separate ways. The aberrant vagal fibers join the main trunk of the vagus nerve and follow those fibers of the vagus that are destined to supply most of the intrinsic laryngeal muscles. The spinal accessory nerve exits the cranial vault via the jugular foramen. It courses inferiorly to the

deep surface of the sternocleidomastoid muscle providing it with motor innervation. It continues its inferior course to the posterior triangle of the neck and then proceeds to the deep aspect of the upper part of the trapezius muscle to supply it with motor innervation. In view of its origin, many neuroanatomists no longer consider the accessory nerve to be a true cranial nerve, but instead a unique type of spinal nerve.

Additionally, there are differences of opinion relating to the classification of the functional components of the spinal accessory nerve. Some authors consider that this nerve carries **branchiomotor SVE** fibers since neurons of the spinal accessory nucleus develop in a manner characteristic of SVE, not GSE, neurons; whereas others believe that they are **somatomotor**, that is GSE.

Recent literature supports that **GSA** proprioceptive fibers are carried by the spinal accessory nerve from the upper cervical spinal cord levels to the structures it innervates, but questions the branchial arch origins of the trapezius and sternocleidomastoid muscles.

CLINICAL CONSIDERATIONS

A unilateral lesion confined to the spinal accessory nucleus or the nerve proximal to its muscular distribution results in an **ipsilateral flaccid paralysis** and subsequent **atrophy** of the sternocleidomastoid and upper part of the trapezius muscles. An individual with such a lesion is unable to turn his or her head away from the lesion. Normally, unilateral contraction of the sternocleidomastoid muscle draws the mastoid process inferiorly, bending the head sideways (approximating the ear to the shoulder), which is accompanied by an upward turning of the chin towards the opposite side. If the upper part of the trapezius is paralyzed, the upper border of the scapula is rotated laterally and inferiorly with its inferior angle pointing towards the spine. This results in slight drooping of the ipsilateral shoulder, accompanied by a weakening of the shoulder when attempting to raise it.

HYPOGLOSSAL NERVE (CN XII)

The hypoglossal nerve provides motor innervation to the muscles of the tongue

The **hypoglossal nerve** (Fig. 15.20) provides motor innervation to the muscles of the tongue. The cell bodies of the

GSE lower motoneurons of the hypoglossal nerve reside in the **hypoglossal nucleus**, a cell column in the medulla. This nucleus, located ventral to the floor of the fourth ventricle near the midline, forms a triangular elevation—the **hypoglossal trigone**—in the floor of the midline of the ventricle. The nerve cell bodies of the hypoglossal nucleus give rise to axons that course ventrally to arise as a series of tiny rootlets on the ventral surface of the medulla in the sulcus separating the pyramid and the olive. These rootlets collect to form the hypoglossal nerve, which exits the cranial vault through the hypoglossal foramen. The nerve then

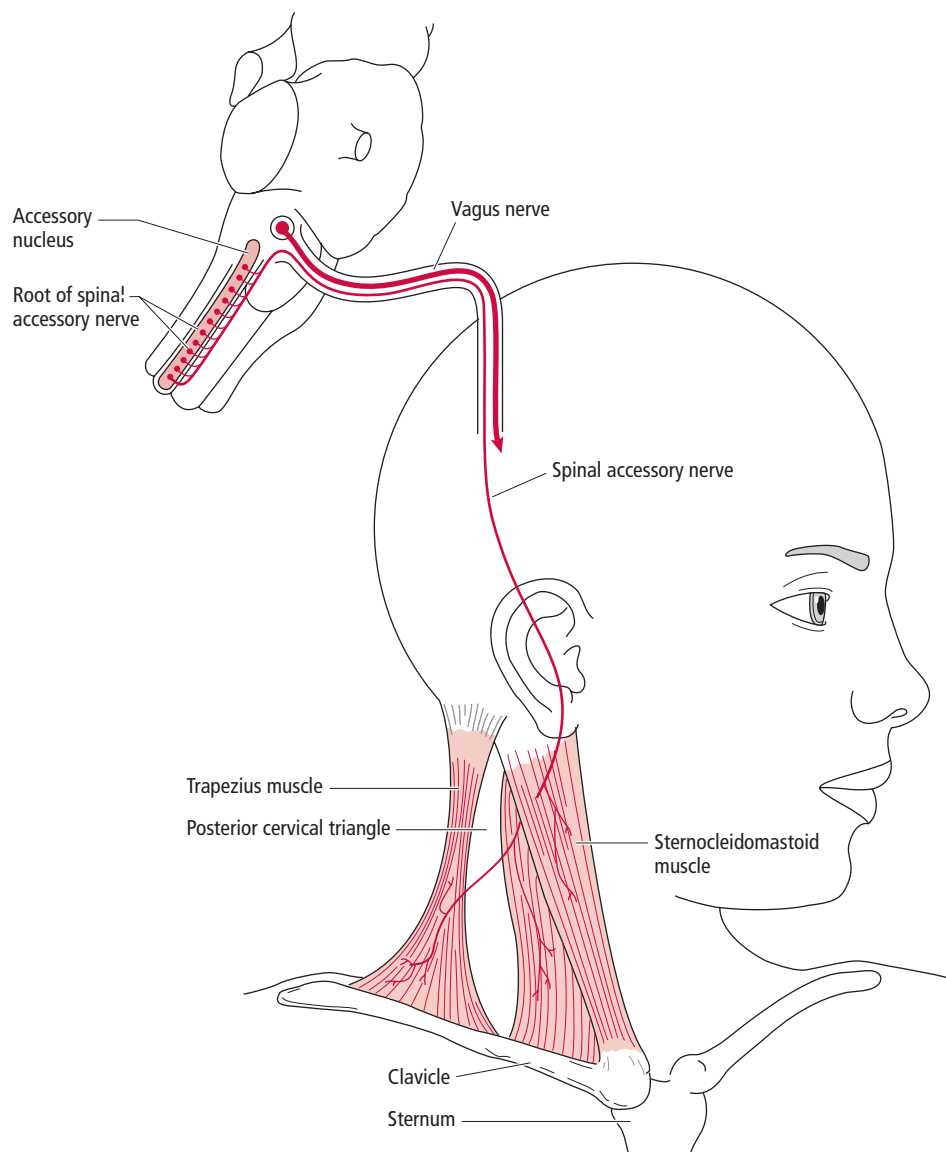


Figure 15.19 ● The origin and distribution of the spinal accessory nerve and its major branches.

courses to the submandibular region to serve the *ipsilateral* side of the tongue. The hypoglossal nerve innervates the intrinsic muscles (transverse, longitudinals, and vertical) and all the extrinsic muscles of the tongue (styloglossus, hyoglossus, and genioglossus) with the exception of the palatoglossus. Recent studies indicate that **GSA** fibers terminating in muscle spindles of the tongue musculature transmit proprioceptive sensation to the trigeminal system involved in reflex activity of mastication. Some investigators believe that the cell bodies of these GSA pseudounipolar neurons are located in the mesencephalic nucleus of the trigeminal nerve, whereas others maintain that they are dispersed along the hypoglossal nerve.

CLINICAL CONSIDERATIONS

A unilateral lesion of the hypoglossal nerve will cause the tongue to deviate toward the side of the lesion (impaired side)

A lesion in the hypoglossal nucleus or nerve results in **flaccid paralysis** and subsequent **atrophy** of the ipsilateral tongue musculature. **Hemiparalysis** of the tongue causes creasing (wrinkling) of the dorsal surface of the tongue ipsilateral to the lesion. Normally, the simultaneous contraction of the paired genioglossus muscles causes the tongue to protrude straightforward. During examination of the patient it is important to remember that a unilateral lesion of the hypoglossal nerve will cause the tongue to deviate towards the *side of the lesion* (impaired side) since the functional genioglossus on the intact side is unopposed by the paralyzed, inactive genioglossus on the lesion side.

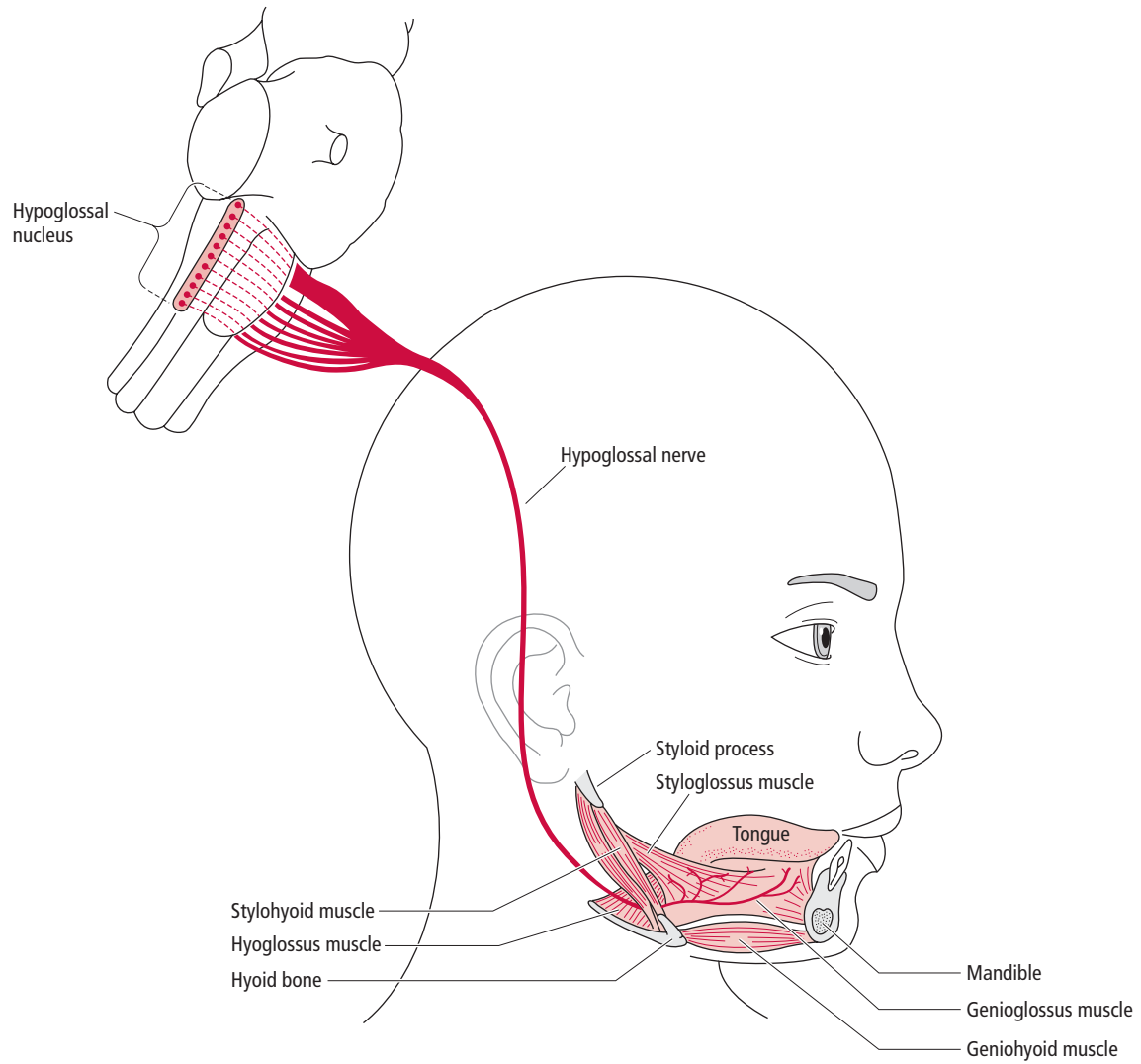


Figure 15.20 ● The origin and distribution of the hypoglossal nerve.

SYNONYMS AND EPONYMS OF THE CRANIAL NERVES

Name of structure or term	Synonym(s)/eponym(s)	Name of structure or term	Synonym(s)/eponym(s)
Cochlear ganglion	Spiral ganglion	Postcentral gyrus	Primary sensory cortex (S-I)
Discriminatory tactile sense	Fine touch sensation		Primary somatosensory cortex
Dorsal trigeminal lemniscus	Dorsal trigeminothalamic tract		Primary somesthetic cortex
Functional components of cranial nerves	Modalities of cranial nerves	Pseudounipolar neurons	Brodmann's areas 3, 1, and 2
General somatic efferent (GSE)	Somatic motor	Special visceral efferent (SVE)	Unipolar neuron
	Somatomotor	Sphincter pupillae muscle	Branchiomotor
General visceral efferent (GVE)	Visceral motor	Spinal nucleus of the trigeminal	Constrictor pupillae muscle
	Visceromotor	Spinal tract of the trigeminal	Descending nucleus of the trigeminal
	Secretomotor	Subnucleus caudalis of the spinal trigeminal nucleus	Descending tract of the trigeminal
Inferior ganglion of the vagus nerve	Nodose ganglion	Subnucleus interpolaris of the spinal trigeminal nucleus	Pars caudalis of the spinal trigeminal nucleus
Internuclear neurons	Interneurons	Subnucleus oralis of the spinal trigeminal nucleus	Pars interpolaris of the spinal trigeminal nucleus
Main nucleus of the trigeminal	Chief nucleus of the trigeminal	Superior ganglion of the vagus nerve	Pars oralis of the spinal trigeminal nucleus
	Principal nucleus of the trigeminal	Trigeminal neuralgia	Jugular ganglion
Mandibular division of the trigeminal nerve	Mandibular nerve		Trigeminal nerve pain
Maxillary division of the trigeminal nerve	Maxillary nerve	Ventral trigeminal lemniscus	Tic douloureux
Medial strabismus	Internal strabismus	Vestibular ganglion	Ventral trigeminothalamic tract
	Convergent strabismus	Vestibulocochlear nerve	Vestibular ganglion of Scarpa
	Esotropia		Acoustic nerve (older term)
Ophthalmic division of the trigeminal nerve	Ophthalmic nerve		



FOLLOW-UP TO CLINICAL CASE

This patient has **trigeminal neuralgia**, also called **tic douloureux**. This is a purely clinical diagnosis, and tests are usually normal. This is a very common disorder, and this is a typical presentation. The pain is indeed excruciating and should be taken very seriously, as suicide is not an uncommon result!

This condition is most common in the elderly, though it can occur in younger age groups. The common etiology is thought to be from ephaptic transmission of nerve impulses, or in other words "short circuiting." The most common cause is from vascular "loops" that develop and surround one of the divisions of the trigeminal nerve at its root, most commonly affecting the ophthalmic and sometimes the maxillary divisions. This causes compression of the nerve, demyelination, and ephaptic

transmission. This condition is usually spontaneous and comes "out of the blue." Trigeminal neuralgia in a young person brings up the specter of multiple sclerosis. Multiple sclerosis is a disease of the central nervous system, and can be a cause of trigeminal neuralgia. This is thought to result from demyelination of the trigeminal nerve root as it enters the brainstem.

There are effective treatments for this condition. Carbamazepine, an antiseizure medication, is the most effective. Other antiseizure medications have been used. Trigeminal neuralgia from vascular loops can be treated, if refractory to medications, by microvascular decompression surgery. This surgical procedure generated much skepticism when it was first introduced, but has produced excellent results for refractory cases and has now become widely accepted.



5 What are some treatment options for trigeminal neuralgia?

QUESTIONS TO PONDER

1. What is the first clinical sign of intracranial pressure on the GVE (parasympathetic fibers) of the oculomotor nerve?
2. What are the functional deficits caused by a lesion to the trochlear nucleus or to the trochlear nerve?
3. What are the functional deficits following a lesion to the right abducens nucleus?
4. What eye movement functional deficits result following a lesion to one medial longitudinal fasciculus?
5. What eye movement deficits result following a lesion in the vicinity of the abducens nucleus?
6. What is the cause of the "crocodile tear syndrome"?
7. Which cranial nerves are likely to be damaged from a growing pituitary tumor?
8. Name the cranial nerves that are susceptible to damage from a tumor growing in the vicinity of the cerebellopontine angle.