Cranial Nerves

There are 12 pairs of cranial nerves emerging from the brain and radiating from its surface (Fig. 17.1). 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 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 that 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.

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 that 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.

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 that 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.
Although the cranial nerves and their sensory and parasympathetic ganglia (Tables 17.1, 17.2) 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. Another cranial nerve that is

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 following list includes their names and corresponding numbers.

<table>
<thead>
<tr>
<th>Roman Numeral</th>
<th>Cranial Nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Olfactory nerve</td>
</tr>
<tr>
<td>II</td>
<td>Optic nerve</td>
</tr>
<tr>
<td>III</td>
<td>Oculomotor nerve</td>
</tr>
<tr>
<td>IV</td>
<td>Trochlear nerve</td>
</tr>
<tr>
<td>V</td>
<td>Trigeminal nerve</td>
</tr>
<tr>
<td>VI</td>
<td>Abducent nerve</td>
</tr>
<tr>
<td>VII</td>
<td>Facial nerve</td>
</tr>
<tr>
<td>VIII</td>
<td>Vestibulocochlear nerve</td>
</tr>
<tr>
<td>IX</td>
<td>Glossopharyngeal nerve</td>
</tr>
<tr>
<td>X</td>
<td>Vagus nerve</td>
</tr>
<tr>
<td>XI</td>
<td>Spinal accessory nerve</td>
</tr>
<tr>
<td>XII</td>
<td>Hypoglossal nerve</td>
</tr>
</tbody>
</table>

### Table 17.1  
Sensory ganglia of the cranial nerves.

<table>
<thead>
<tr>
<th>Ganglion</th>
<th>Cranial nerve association</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trigeminal (semilunar, Gasserian)</td>
<td>Trigeminal (V)</td>
</tr>
<tr>
<td>Geniculate</td>
<td>Facial (VII)</td>
</tr>
<tr>
<td>Cochlear (spiral)</td>
<td>Cochlear (VIII)</td>
</tr>
<tr>
<td>Vestibular (Scarpa’s)</td>
<td>Vestibular (VIII)</td>
</tr>
<tr>
<td>Superior glossopharyngeal</td>
<td>Glossopharyngeal (IX)</td>
</tr>
<tr>
<td>Inferior glossopharyngeal</td>
<td>Glossopharyngeal (IX)</td>
</tr>
<tr>
<td>Superior vagal (jugular)</td>
<td>Vagus (X)</td>
</tr>
<tr>
<td>Inferior vagal (nodose)</td>
<td>Vagus (X)</td>
</tr>
</tbody>
</table>
atypical is the olfactory nerve. The sensory neurons of the olfactory pathway reside in the olfactory epithelium in the roof of the nasal cavity instead of a ganglion. The axons of these sensory neurons do not collect to form a single bundle of axons, a nerve, but instead form multiple small collections of axons that traverse the foramina of the cribriform plate of the ethmoid bone to terminate in the overlying olfactory bulb in the anterior cranial fossa. A third deviation from the normal cranial nerves is the spinal accessory nerve. Part of the spinal accessory nerve arises from the cervical spinal cord; thus there are only nine pairs of cranial nerves that truly emerge from the brainstem.

The main sensory and motor nuclei of the cranial nerves are shown in Fig. 17.2.

### Table 17.2
Parasympathetic ganglia of the cranial nerves.

<table>
<thead>
<tr>
<th>Ganglion association</th>
<th>Cranial nerve association</th>
<th>Trigeminal Function(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ciliary</td>
<td>Oculomotor (III)</td>
<td>Ophthalmic division</td>
</tr>
<tr>
<td>Pterygopalatine</td>
<td>Facial (VII)</td>
<td>Maxillary division</td>
</tr>
<tr>
<td>Submandibular</td>
<td>Facial (VII)</td>
<td>Medial division</td>
</tr>
<tr>
<td>Otic</td>
<td>Glossopharyngeal (IX)</td>
<td>Salivation (submandibular and sublingual glands)</td>
</tr>
<tr>
<td>Intramural</td>
<td>Vagus (X)</td>
<td>Salivation (parotid gland)</td>
</tr>
</tbody>
</table>

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 special visceral efferent to striated muscles derived from the pharyngeal 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 17.3).

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

3 \textbf{General visceral afferent} (GVA). General sensation from the viscera is transmitted by the facial, glossopharyngeal, and vagus nerves.

4 \textbf{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 \textbf{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 \textbf{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 \textbf{Special visceral efferent} (SVE). These motor fibers are special because they supply motor innervation to skeletal muscles of branchiomeric origin: mandibular, hyoid, 3rd, 4th, and 6th pharyngeal arches.

Table 17.4 summarizes the modalities, nuclei, ganglia, and functions of the cranial nerves.

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|}
\hline
\textbf{Modality} & \textbf{Cranial nerves} & \textbf{Function(s)} \\
\hline
General somatic afferent (GSA) & V, VII, IX, X & General sensation and general proprioception \\
General somatic efferent (GSE) & III, IV, VI, XII & Motor supply to extracocular 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 & Special sensory input from retina \\
& VIII & Special sensory input from vestibulocochlear apparatus \\
Special visceral afferent (SVA) & I & Special sense of smell \\
& VII, IX, X & 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 pharyngeal arches \\
\hline
\end{tabular}
\caption{Cranial nerve functional components.}
\end{table}
<table>
<thead>
<tr>
<th>Cranial nerve</th>
<th>Functional component (modality)</th>
<th>Nucleus/ ganglia</th>
<th>Location of cranial nerve nuclei</th>
<th>Ganglion</th>
<th>Distribution</th>
<th>Function(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>V Trigeminal</td>
<td>SVE</td>
<td>Motor nucleus of the trigeminal</td>
<td>Metencephalon (pons)</td>
<td>–</td>
<td>Muscles of mastication: temporalis, masseter, medial pterygoid, lateral pterygoid, Mylohyoid, anterior belly of the digastric</td>
<td>Chewing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trigeminal ganglion</td>
<td>Metencephalon (pons to C3)</td>
<td>Trigeminal</td>
<td>Tense tympanic membrane</td>
<td>Tenses soft palate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Tensor veli palatini</td>
<td>General sensation</td>
</tr>
<tr>
<td>GSA</td>
<td></td>
<td>Main (chief, principal) nucleus of the trigeminal</td>
<td>Metencephalon (pons)</td>
<td>–</td>
<td>Extraocular muscles-proprioception, TMJ, PDL</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trigeminal ganglion</td>
<td>Metencephalon (pons to C3)</td>
<td>Trigeminal</td>
<td>Tensor veli palatini</td>
<td>General sensation</td>
</tr>
<tr>
<td>GP</td>
<td></td>
<td>Mesencephalic nucleus of the trigeminal</td>
<td>Trigeminal</td>
<td>–</td>
<td>Muscles of mastication</td>
<td>Muscle stretch</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trigeminal ganglion</td>
<td>Trigeminal</td>
<td></td>
<td>Periodontal ligament</td>
<td>Pressure sensation</td>
</tr>
<tr>
<td>VI Abducents</td>
<td>GSE</td>
<td>Abducens</td>
<td>Metencephalon (pons)</td>
<td>–</td>
<td>Lateral rectus</td>
<td>Eye movement</td>
</tr>
<tr>
<td>VII Facial</td>
<td>SVE</td>
<td>Facial</td>
<td>Metencephalon (pons)</td>
<td>–</td>
<td>Muscles of facial expression, platysma, posterior belly of the digastric, and stylhyoid</td>
<td>Facial expression</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Stapedius</td>
<td>Tension on stapes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lacrimal gland</td>
<td>Lacrimation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Glands of the nasal cavity and palate</td>
<td></td>
</tr>
<tr>
<td>GVE (parasympathetic)</td>
<td>Superior salivatory</td>
<td>Myelencephalon</td>
<td>Pterygopalatine (parasympathetic)</td>
<td>–</td>
<td>Submandibular and sublingual glands</td>
<td>Salivation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Submandibular (parasympathetic)</td>
<td></td>
<td>Submandibular and sublingual glands</td>
<td></td>
</tr>
<tr>
<td>SVA</td>
<td>Solitarius</td>
<td>Myelencephalon</td>
<td>Geniculate</td>
<td></td>
<td>Anterior two-thirds of the tongue</td>
<td>Taste</td>
</tr>
<tr>
<td>GVA</td>
<td>Solitarius</td>
<td>Myelencephalon</td>
<td>Geniculate</td>
<td></td>
<td>Middle ear, nasal cavity, and soft palate</td>
<td>Visceral sensation</td>
</tr>
<tr>
<td>GSA</td>
<td>Spinal nucleus of the trigeminal</td>
<td>Metencephalon (pons)</td>
<td>Geniculate</td>
<td></td>
<td>External auditory meatus and area posterior to ear</td>
<td>General sensation</td>
</tr>
<tr>
<td>VIII Vestibulocochlear</td>
<td>Cochlear</td>
<td>Dorsal and ventral cochlear</td>
<td>Myelencephalon</td>
<td>–</td>
<td>Organ of Corti (inner ear)</td>
<td>Hearing</td>
</tr>
<tr>
<td>Cranial nerve</td>
<td>Functional component (modality)</td>
<td>Nucleus/ ganglia</td>
<td>Location of cranial nerve nuclei</td>
<td>Ganglion</td>
<td>Distribution</td>
<td>Function(s)</td>
</tr>
<tr>
<td>--------------</td>
<td>---------------------------------</td>
<td>-----------------</td>
<td>---------------------------------</td>
<td>----------</td>
<td>-------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Vestibular</td>
<td>SSA</td>
<td>Vestibular complex</td>
<td>Myelencephalon</td>
<td>Vestibular</td>
<td>Vestibular</td>
<td>Equilibrium</td>
</tr>
<tr>
<td>X Glossopharyngeal</td>
<td>SVE</td>
<td>Ambiguus</td>
<td>Myelencephalon</td>
<td>–</td>
<td>Otic (parasympathetic)</td>
<td>Stylopharyngeus, Parotid gland</td>
</tr>
<tr>
<td></td>
<td>GVE (parasympathetic)</td>
<td>Solitarius</td>
<td>Myelencephalon</td>
<td>Inferior ganglion of the glossopharyngeal</td>
<td>Posterior one-third of the tongue and adjacent pharyngeal wall</td>
<td>Swallowing, Salivation</td>
</tr>
<tr>
<td></td>
<td>SVA</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Taste</td>
</tr>
<tr>
<td></td>
<td>GVA</td>
<td>Ambiguus Solitarius</td>
<td>Myelencephalon</td>
<td>Inferior ganglion of the glossopharyngeal</td>
<td>Middle ear, pharynx, tongue, carotid sinus</td>
<td>Visceral sensation</td>
</tr>
<tr>
<td></td>
<td>GSA</td>
<td>Spinal nucleus of the trigeminal</td>
<td>Myelencephalon</td>
<td>Superior ganglion of the glossopharyngeal</td>
<td>Posterior one-third of the tongue, soft palate, upper pharynx, and auditory tube</td>
<td>General sensation</td>
</tr>
<tr>
<td>X Vagus</td>
<td>GVE (parasympathetic)</td>
<td>Dorsal motor nucleus of the vagus</td>
<td>Myelencephalon</td>
<td>Thoracic and abdominal submucosal and myenteric autonomic plexuses</td>
<td>Thoracic and abdominal viscera</td>
<td>Gland secretion, peristalsis</td>
</tr>
<tr>
<td></td>
<td>SVE</td>
<td>Ambiguus</td>
<td>Myelencephalon</td>
<td>–</td>
<td>–</td>
<td>Phonation</td>
</tr>
<tr>
<td></td>
<td>SVA</td>
<td>Solitarius</td>
<td>Myelencephalon</td>
<td>Inferior (nodose)</td>
<td>Epiglottis</td>
<td>Taste</td>
</tr>
<tr>
<td></td>
<td>GVA</td>
<td>Solitarius</td>
<td>Myelencephalon</td>
<td>Inferior (nodose)</td>
<td>Thoracic and abdominal viscera</td>
<td>Visceral sensation</td>
</tr>
<tr>
<td></td>
<td>GSA</td>
<td>Spinal nucleus of the trigeminal</td>
<td>Myelencephalon</td>
<td>Superior (jugular)</td>
<td>Carotid body</td>
<td>General sensation</td>
</tr>
<tr>
<td>XI Spinal accessory</td>
<td>SVE</td>
<td>accessory</td>
<td>spinal cord C1–C5(C6)</td>
<td>–</td>
<td>Area posterior to the ear, external acoustic meatus, and posterior part of meninges</td>
<td>To sternocleidomastoid and trapezius</td>
</tr>
<tr>
<td>XII Hypoglossal</td>
<td>GSE</td>
<td>Hypoglossal</td>
<td>Myelencephalon</td>
<td>–</td>
<td>–</td>
<td>Muscles of the tongue</td>
</tr>
</tbody>
</table>

**OLFACTORY NERVE (CN I)**

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. 21.1). The axons of these bipolar neurons are SVA fibers transmitting olfactory sensation. These axons assemble to form multiple (15–20) and separate thin bundles of axons, the olfactory fila (L., “threads”), which although collectively form what is considered to be cranial nerve I, they do not form a single nerve trunk as do the conventional cranial nerves. 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 interneurons (see Chapter 21).

**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 second order multipolar ganglion cells of the retina. The ganglion cells give rise to unmyelinated axons that converge posteriorly at the optic disc and traverse the lamina cribrosa, a sieve-like perforated area of the sclera, to emerge from the posterior pole 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, becomes invested by extensions of the three meninges that cover the brain and then leaves the orbit via the optic canal to enter the middle cranial fossa. There, the optic nerves of the right and left sides converge and 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 each retina decussate (through the central region of the chiasma) to the opposite optic tract. All ganglion cell axons arising from the temporal half of each 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 of the midbrain 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 nucleus of the visual system associated with somatic reflexes; the pretectal area, a midbrain region associated with autonomic reflexes; and the hypothalamus (see Figs 18.5, 18.7, 18.9).

**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 (somaticotor) 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 rostral midbrain tegmentum. It is partly embedded within, and is situated ventral to the periaqueductal gray (PAG) matter, adjacent to the midline at the level of the superior colliculus. The oculomotor nucleus consists of several subnuclei representing each of the extraocular muscles and levator palpebrae superioris muscle. 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 central caudate nucleus, the cell group innervating the levator palpebrae superioris, is located in the midline, sending motor fibers to this muscle bilaterally (to both right and left upper eyelids). The medial column, the cell group innervating the superior rectus, sends projections to the contralateral side; whereas the cell group innervating the medial rectus, inferior oblique, and inferior rectus sends projections to the ipsilateral side. Axons that cross, do so within the oculomotor nucleus. As these axons (that have already crossed) exit the oculomotor nucleus, they join the oculomotor nerve root fascicles of the opposite side. Although the oculomotor nerve is unique since it is the only cranial nerve that consists of a mix of crossed and uncrossed fibers, this is of little clinical significance. For example, a lesion that damages the entire (right) oculomotor nucleus that would result in paralysis of the superior rectus of one (left) eye and paralysis of the remaining extraocular muscles innervated by the oculomotor nerve in the other (right) eye, is rare. Even if this occurs, the actions of the functional extraocular muscles with intact innervation compensate for the deficits of the paralyzed (left) superior rectus muscle of the same eye. All of the axons that exit the oculomotor nucleus on each side, course ventrally as numerous fascicles, curving medially, pass through the red nucleus and the medial aspect of the substantia nigra, and then gather to form a bundle that exits the brainstem between the two cerebral peduncles at the interpeduncular fossa.

The oculomotor nerve root fascicles that emerge from the oculomotor nucleus and course ventrally in the midbrain tegmentum to exit at the interpeduncular fossa, and the oculomotor nerve that exits the midbrain, contain fibers that have already crossed. Thus, a lesion involving the oculomotor root fascicles sweeping ventrally in the midbrain tegmentum, or the oculomotor nerve anywhere along its course after it exits the midbrain, would result in deficits in the ipsilateral eye only.

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 two distinct populations of neurons. One group consists of the cell bodies of Edinger–Westphal preganglionic parasympathetic (EWpg) neurons whose axons join the oculomotor nerve and course to the orbit where they synapse in the ciliary ganglion. This group of fibers is involved in two autonomic functions, pupillary constriction and accommodation of the lens. They do that by providing parasympathetic innervation to two of the three smooth muscles of the eye, the sphincter pupillae and ciliary muscles, respectively. The other group of neurons within the Edinger-Westphal nucleus is the Edinger–Westphal centrally projecting (EWcp) neurons that send their axons to various brainstem nuclei including the gracile, cuneate, spinal trigeminal, parabrachial and inferior olivary nuclei. Other fibers descend to the spinal cord. These projections play a role in diverse behaviors such as eating, drinking and stress-related functions.

Several small nuclei that control eye movements reside near the oculomotor nucleus. They are the (1) nucleus of Darkschewitsch located in the ventrolateral margin of the PAG,
rostral to the oculomotor nucleus, the (2) interstitial nucleus of Cajal, located in the dorsomedial region of the rostral midbrain tegmentum, next to the medial longitudinal fasciculus (MLF), and the (3) nucleus of the posterior commissure (a pretectal nucleus). The nucleus of Cajal and the nucleus of the posterior commissure play a role in the coordination of head and eye movements, especially in vertical gaze.

As the oculomotor nerve exits the midbrain, it immediately passes between the posterior cerebral artery (the terminal branch of the basilar artery) and the superior cerebellar artery (a branch of the basilar artery). The oculomotor nerves close proximity to these vessels makes it susceptible to lesions of vascular origin (compression of nerve by calcifications or aneurisms in the vessels wall). The oculomotor nerve then enters the interpeduncular cistern (a dilated region of the subarachnoid space between the cerebral peduncles), passes under the posterior communicating artery and pierces the dura to enter the cavernous sinus. It courses anteriorly in the lateral wall of the cavernous sinus where it branches into superior and inferior divisions. It then exits the cavernous sinus and passes through the superior orbital fissure to enter the orbit. The superior division gives rise to branches that innervate the levator palpebrae superioris and the superior rectus muscles. The inferior division gives rise to branches that innervate the medial rectus, the inferior rectus and the inferior oblique muscles. The levator palpebrae superioris muscle elevates the upper eyelid; the superior and inferior rectus muscles elevate and depress the eye, respectively; the medial rectus muscle adducts the eye; and the inferior oblique muscle elevates the eye when it is in the medial position. 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 focus.

GSA pseudounipolar neurons, whose cell bodies reside within the trigeminal ganglion 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.

Unilateral damage to the oculomotor nerve results in deficits in the ipsilateral eye. The affected individual cannot elevate the upper eyelid; is unable to move the eye medially or vertically; the pupil is dilated, and the lens is flat, and cannot accommodate for near focus. 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 inferior oblique, is also paralyzed. The innervation of the lateral rectus (CN VI) is intact, and since this muscle normally abducts the eye, it pulls the eye laterally due to the unopposed action of its paralyzed antagonist, the medial rectus. The innervation of the superior oblique (CN IV) is also intact and since it normally intorts, depresses and abducts the eye, it turns the eye “down and out” due to the unopposed actions of the paralyzed, fasciculus superior, medial and inferior rectus, and inferior oblique muscles. The lateral deviation of the eye is referred to as lateral strabismus. (Fig. 17.3).

This causes the visual axes of the two eyes to become misaligned as one eye deviates from the midline, resulting in diplopia (double vision). Since the affected eye is deviated “down and out,” the patient complains of a mixed horizontal and vertical binocular diplopia.

The sphincter pupillae muscle becomes nonfunctional due to interruption of its parasympathetic innervation. The pupil ipsilateral to the lesion will remain in fixed dilatation (mydriasis) and does not respond (constrict) to a flash of light. This may be the first clinical sign of intracranial pressure on the respective muscle. The pupil contralateral to the lesion will react normally. The following symptoms ipsilateral to the side of the lesion: (i) ptosis (drooping of the upper eyelid), (ii) downward and outward deviation of the eye, (iii) lateral strabismus, (iv) pupillary dilation, and (v) loss of accommodation of the lens.
The trochlear nerve is unique because it is the only cranial nerve 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 ‘SO’). Since the superior oblique’s tendon threads through the trochlea (L., “pulley”), a fibrous loop attached to the medial superior surface of the orbital margin, the nerve innervating it is called the “trochlear nerve.”

Normally, contraction of the superior oblique muscle causes the eye to intort (around an anteroposterior axis), accompanied by simultaneous depression (downward movement, about a horizontal axis) and abduction (outward movement, about a vertical axis) of the bulb of the eye. This muscle is sometimes referred to as the “Salvation Army muscle” (since when it contracts, its actions cause the eye to turn inferolaterally (“down and out”). Intorsion of the eyeball is the slight turning of the eye about its anteroposterior axis. Imagine that extreme intorsion (which we really cannot do) will bring the superior pole/surface of the eye to face the medial wall of the orbit.

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 around the periaqueductal gray (PAG) matter toward the tectum. The fibers decussate posteriorly before emerging from the brainstem at the junction of the pons and midbrain, just below the inferior colliculus.

The trochlear nerve is the thinnest cranial nerve and the only one whose fibers originate totally from the contralateral nucleus. Fibers arising from this cranial nerve and the only one that accompanies a cranial nerve are the oculomotor nerve. The trochlear nerve arises from the contralateral superior oblique muscle, whereas damage to the trochlear nerve results in the same deficits, but in the ipsilateral muscle.

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

When the superior oblique muscle is paralyzed, the individual develops trochlear nerve palsy, characterized by the following: the ipsilateral (affected) eye becomes extorted about its anteroposterior axis so that the superior surface of the eyeball will approach the lateral wall of the orbit, accompanied by simultaneous elevation (upward deviation of the eye known as hypertropia), and slight abduction (lateral, or outward) deviation of the eye (Fig. 17.4B, 17.5). The new resting position of the eye is caused by the actions of the unopposed inferior oblique (which extorts, elevates, and abducts the eye).

Paralysis of the superior oblique muscle causes the visual axes of the two eyes to become misaligned and consequently, the brain receives two non-overlapping images. This is what causes a patient with trochlear nerve palsy to complain of double vision (diplopia). An individual with trochlear nerve palsy experiences vertical diplopia.

Normally, downward gaze is executed by the joint effort of the superior oblique and inferior rectus muscles. When the superior oblique is paralyzed, the patient’s ability to depress the affected eye is impaired (i.e., the patient
experiences weakness — not total inability — of downward gaze with the affected eye. Consequently, the eye drifts upward caused by the unopposed action of the inferior oblique that extorts, elevates, and abducts the eye.

In trochlear nerve palsy, the hypertropia of the affected eye (and therefore, the vertical diplopia), is most apparent to the individual (and becomes worse) in (1) attempted downward gaze (when descending stairs); in (2) attempted medial gaze (when the affected eye tries to look down and in toward the nose, as in reading a book); and (3) when tilting the head toward the affected side (Fig. 17.6). Because the normal eye becomes depressed, but the affected eye is partially depressed only by the action of the inferior rectus (since the superior oblique is paralyzed and cannot contribute to depressing the eye), it causes the eyes to become misaligned resulting in vertical diplopia. When a patient who has vertical diplopia is descending steps, each (real) step appears to have an overlapping (diplopic, that is an apparent false) step above it. Also when reading, a sentence appears to have a diplopic, overlapping sentence above the real sentence.

To counteract the diplopia and to restore nearly horizontal proper eye alignment, the affected individual realizes that the diplopia is minimized by tilting the head toward the normal side (Fig. 17.4B, 17.7). Normally, tilting of the head to one side elicits a reflex rotation about the anteroposterior axis of the eyes in the opposite direction (Fig. 17.4A), so that the image of an object will remain fixed on the retina.

Tilting of the head to the normal side, causes the normal eye to intort and become aligned with the affected, extorted eye. This head tilt, compensates for the extorsion of the affected eye. Pointing of the chin downward (“chin tuck”) rolls the normal eye upward. The chin tuck compensates for the hypertropia of the affected eye. The diplopia is eliminated when the individual closes either eye because only one image reaches the brain.

**Figure 17.4** (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.
CRANIAL NERVES

Figure 17.5 ● Trochlear nerve paralysis/palsy of the left eye. The affected eye (A) has hypertropia (looks “up”) since the superior oblique’s ability to depress the eye is impaired, and (B) is extorted (looks “out”) since the superior obliques ability to intort the eye is impaired.

Figure 17.6 ● Trochlear nerve paralysis/palsy of the left eye. The affected eye has hypertropia (is deviated upward) and is slightly extorted. Hypertropia is due to the impaired ability to depress the affected eye. When the head is tilted toward the side of the palsy (as seen here), the hypertropia and extorsion become worse. That is, weakness of the superior oblique’s depressor and intorsion functions intensify.

Figure 17.7 ● Trochlear nerve paralysis/palsy of the left eye. By titling the head toward the normal side, the normal eye intorts to align it with the affected eye, which is extorted. “Chin tuck” would elevate the normal eye to become closely aligned with the affected (hypertropic) eye.

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 including the motor innervation to the muscles of mastication and cornea of the eye, the face, mucous membranes of the nasal cavities, paranasal sinuses, oral cavity, and hard palate, the temporomandibular joint, lower jaw, teeth and periodontal ligaments (PDL), the collagenous connective tissue lining the tooth sockets that suspends the teeth in their alveolus. 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 its motor root exits at the ventrolateral aspect of the pons (see Fig. 17.1). 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 the pons (Fig. 17.10). The motor root joins the sensory portion of the mandibular division of the trigeminal nerve just outside the skull, in the infratemporal fossa, to form the mandibular trunk. Before the motor root joins it, the trigeminal nerve displays a swelling, the trigeminal ganglion. The ganglion is enclosed in Meckel’s cave, a dural pouch that lies in a bony depression, the trigeminal impression 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 previously.

The trigeminal system

The trigeminal system is collectively formed by the trigeminal nerve, ganglion, nuclei, and its central pathways to the ascending sensory pathways from the body (dorsal column-medial lemniscus and the spinothalamic tracts) that relay touch, proprioception, nociception, thermal sense, and itch to consciousness, the trigeminal sensory pathways transmit the same type of sensory information, to consciousness, but from structures of the head to the cerebral cortex. Additionally, like the ascending sensory pathways from the body, each of the trigeminal sensory pathways consists of a three neuron sequence (first, second, and third order neurons) from the periphery to the cerebral cortex, respectively (Figs 17.8, 17.9).

Although most of the cell bodies of the first order sensory neurons of the trigeminal pathways for touch, nociception, thermal sense, and itch sensations from the head reside in the trigeminal ganglion, there is an unusual nucleus, the mesencephalic nucleus of the trigeminal (discussed later), that also contains pseudounipolar sensory neurons but they transmit sensory input related to stretch and proprioception from the muscles of mastication and most proprioceptive input from the periodontal ligaments surrounding the roots of the teeth. In contrast, the pseudounipolar sensory neurons that transmit sensory input related to stretch and proprioception from the extraocular muscles, the TMJ and some periodontal ligaments reside in the trigeminal ganglion.

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. The same type of sensory receptors located in the skin, skeletal muscles, tendons, mucous membranes, and other visceral structures in the body, are also

![Figure 17.8](image-url)
CraniAl Nerves

327

Trigeminal nuclei

The trigeminal system includes four nuclei: three sensory nuclei, the mesencephalic nucleus of the trigeminal, the principal (main, chief) sensory nucleus of the trigeminal, and the spinal nucleus of the trigeminal and one motor nucleus, the motor nucleus of the trigeminal; (see Fig. 17.2; Table 17.5). The sensory nuclei form a continuous, longitudinal column of cells that extends from the rostral midbrain rostrally, to the upper two to three cervical spinal cord levels, caudally.

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 principal 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.

Mesencephalic nucleus

The mesencephalic nucleus contains the sensory pseudounipolar neurons that transmit proprioception from the muscles of mastication and periodontal ligaments (PDLs) to the motor and sensory nuclei (principal and spinal) of the trigeminal, the reticular formation and the cerebellum involved in reflexes and to modulate and coordinate muscle activity during chewing

The mesencephalic nucleus of the trigeminal extends from the rostral pons to the rostral midbrain. This nucleus 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 found associated with the peripheral processes of trigeminal sensory neurons that terminate in the head region. The sensory receptors at the peripheral endings of the trigeminal sensory neurons transduce tactile, nociceptive, thermal, or itch sensory stimuli to nerve impulses that are relayed to the brainstem. The central processes of trigeminal sensory neurons conveying these impulses enter the pons as part of the nerve’s sensory root, join the spinal tract of the trigeminal, and terminate in the trigeminal sensory nuclei where they establish synaptic contacts with second order neurons housed in these nuclei. The trigeminal sensory 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 depending on their nuclear origin, join the ventral or dorsal trigeminomotor tracts. These tracts ascend to relay 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 (primary somesthetic cortex) of the cerebral cortex for conscious awareness of the sensation and for further processing.

Table 17.5: The trigeminal nuclei.

<table>
<thead>
<tr>
<th>Motor nucleus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory nuclei:</td>
</tr>
<tr>
<td>Principal (chief, main) nucleus of the trigeminal</td>
</tr>
<tr>
<td>Mesencephalic nucleus of the trigeminal</td>
</tr>
<tr>
<td>Spinal nucleus of the trigeminal:</td>
</tr>
<tr>
<td>Subnucleus oralis</td>
</tr>
<tr>
<td>Subnucleus interpolaris</td>
</tr>
<tr>
<td>Subnucleus caudalis</td>
</tr>
</tbody>
</table>

Figure 17.9: The trigeminal pathway for pain and temperature. Pain and temperature sensation from the orofacial structures is transmitted to the brainstem subnucleus caudalis (Sc) of the spinal nucleus of the trigeminal 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 trigeminomotor tracts to terminate in the VPM nucleus of the thalamus. Third order neurons from the VPM terminate in the postcentral gyrus (PCG). For other abbreviations, see Fig. 17.8.
no synapses in the mesencephalic nucleus. The peripheral large-diameter myelinated processes of these neurons convey proprioceptive (GP) input from the muscle spindles of the muscles of mastication. The pseudounipolar neurons of the mesencephalic nucleus transmit GP input from peripheral structures in the orofacial region to the trigeminal motor nucleus, sensory nuclei (principal and spinal), and to extrinsic sites such as the reticular formation, and the cerebellum, which are involved in reflexes, oral stereognosis, and coordination of voluntary muscle activity (during chewing, swallowing and speaking).

The central and peripheral processes of the mesencephalic pseudounipolar neurons form the **mesencephalic tract** of the trigeminal nerve which is located immediately lateral to the **mesencephalic nucleus**. The central processes of the mesencephalic pseudounipolar neurons usually branch in the vicinity of the motor nucleus of the trigeminal to send collaterals that terminate in the motor nucleus. Some of these mesencephalic sensory sensory neurons whose central processes synapse in the motor nucleus form the sensory, **afferent limb** of the myotatic jaw jerk reflex. Other neurons of the mesencephalic nucleus which send their peripheral processes to the PDL of the teeth relay proprioceptive input to the motor nucleus, reticular formation and cerebellum to modulate and coordinate the activity of the muscles of mastication during chewing.

The **jaw proprioception** pathway consists of sensory pseudounipolar neurons whose cell bodies are located in the **mesencephalic nucleus**. Their peripheral processes consist of stretch receptors that terminate in the muscles of mastication. Their central processes bifurcate to send a branch to the principal sensory nucleus and another branch to the rostral portions of the spinal nucleus. Second order neurons from these nuclei project to the VPM nucleus of the thalamus which in turn projects to Brodmann’s area 3a on the medial surface of the primary somatosensory cortex in the parietal lobe where position sense of the mandible enters conscious awareness.

Although proprioceptive information is generally processed by the mesencephalic neurons, their receptors, and their central connections, some proprioception is relayed from the temporomandibular joint (TMJ), extraocular muscle spindles and some of the PDLs by the sensory pseudounipolar neurons residing in the trigeminal ganglion to the principal sensory and spinal nuclei of the trigeminal.

**Principal (main, chief) nucleus**

The principal nucleus processes discriminative tactile sensation from the orofacial region.

The principal (main, chief) **sensory nucleus of the trigeminal nerve** is located in the midpons and is continuous superiorly with the mesencephalic nucleus of the trigeminal, and inferiorly with the spinal nucleus of the trigeminal. Based on the pattern of its afferent projections, this nucleus consists of two divisions, a dorsomedial division and a ventrolateral division. Both divisions receive the central processes of first order neurons whose cell bodies reside in the trigeminal ganglion.

The **dorsomedial division** receives the central processes of first order neurons whose peripheral processes collect sensory input originating in the oral cavity (teeth and soft tissues). In contrast, the **ventrolateral division** receives the central processes of first order neurons whose peripheral processes relay sensory input originating from a wide range of structures in the trigeminal nerve’s entire sensory territory, innervated by its three divisions.

Axons of second order neurons arising from the **dorsomedial division** of the principal sensory nucleus form the smaller **ipsilateral posterior (dorsal) trigeminothalamic tract**, which ascends to terminate in the VPM nucleus of the thalamus. Most of the second order neuron axons emerging from the principal sensory nucleus arise from the **ventrolateral division** of the principal sensory nucleus, and cross the midline to join the **contralateral anterior (ventral) trigeminothalamic tract**. This tract, which also carries a small number of ascending axons of second order neurons whose cell bodies reside in the spinal nucleus of the trigeminal relaying tactile, nociceptive and thermal sensation from the orofacial region, ascends to the VPM nucleus of the thalamus.

Based on its anatomical and functional characteristics, the principal sensory nucleus is homologous to the nucleus gracilis and nucleus cuneatus of the dorsal column-medial lemniscus pathway relaying discriminative touch, vibratory sense and proprioception from the body. The principal sensory nucleus is associated with the transmission of mechanoreceptor information for discriminative (fine) tactile, pressure, and some proprioceptive sense from the head.

**Spinal nucleus**

The spinal nucleus is involved in the processing of mechanical, nociceptive, thermal, and itch sensation from the oral and facial regions.

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, oral nucleus), the caudal-most subnucleus caudalis (pars caudalis, caudal nucleus), and the intermediate subnucleus interpolaris (pars interpolaris, interpolar nucleus).

The spinal nucleus of the trigeminal system is involved in the processing of mechanical, nociceptive, thermal, and itch sensation from the oral and facial regions. Although this nucleus processes mechanical stimuli, it is mainly concerned with nociception and thermal sensations. The trigeminal nerve is the only cranial nerve that has a nucleus that processes nociception and thermal sense. Sensory information is relayed to this nucleus via the central processes of sensory pseudounipolar neurons whose cell bodies reside in the trigeminal ganglion and the mesencephalic nucleus. The two rostral subnuclei are also involved in reflex activity and project to the cerebellum, which functions in the coordination of the muscles that move the lower jaw. The spinal nucleus contains the cell bodies of second order neurons whose axons project via
the trigeminothalamic tracts to the contralateral VPM and the dorsomedial (DM) nuclei of the thalamus. The VPM projects to the primary somatosensory cortex residing in the postcentral gyrus, involved in conscious awareness of pain, temperature, and itch sensation. The DM nucleus projects to the anterior cingulate gyrus. The anterior cingulate gyrus is associated with the affective and motivational component of pain, temperature, and itch sensation.

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

The subnucleus interpolaris extends from the rostral extent of the hypoglossal nucleus to the obex and is also associated with the transmission of tactile sense, as well as dental pain.

The subnucleus caudalis extends from the level of the obex (medulla) to the C3 level of the spinal cord. It is associated with the transmission of nociception and thermal sensations from the head. 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." It is the homologue of the substantia gelatinosa since their neurons have similar cellular morphology, synaptic connections, and functions. Based on its afferent projections the subnucleus caudalis itself consists of three regions: rostral, intermediate, and caudal regions. The rostral portion of the subnucleus caudalis processes sensory input from the teeth and other tissues of the oral cavity. Since the trigeminal nerve is the only cranial nerve that has a nucleus that processes nociception, the central processes of sensory neurons residing in the sensory ganglia of the facial, glosopharyngeal and vagus nerves converge to terminate in this rostral region within the subnucleus caudalis. The intermediate component receives sensory input from the perioral region, nose and anterior cheek, whereas its caudal region receives input from the lateral aspect of the face and part of the ear.

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.

The motor nucleus of the trigeminal is located at the midpons, medial to the principal 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.

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 glosopharyngeal) and carries their autonomic “hitchhikers” to their destination.

**Trigeminal tracts**

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

The spinal tract of the trigeminal nerve consists of ipsilateral first order afferent fibers of sensory trigeminal ganglion neurons and mediates tactile, thermal, nociceptive, and itch sensations 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, glosopharyngeal, and vagus nerves. These axons terminate in the spinal trigeminal nucleus, conveying 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 rostrocaudal extent of this nucleus. Inferiorly the spinal tract overlaps the dorsolateral fasciculus of Lissauer (its homologue transmitting similar sensory input from the body), at upper cervical spinal cord levels.

The second order neuron axons emerging from the spinal nucleus form the ventral trigeminothalamic tract.

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

The dorsal trigeminothalamic tract carries uncrossed nerve fibers from the dorsomedial division of the principal sensory nucleus of the trigeminal, relaying discriminative 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).

The mesencephalic tract of the trigeminal is formed by the central and peripheral processes of those pseudounipolar neurons whose cell bodies are in the mesencephalic nucleus of the trigeminal.

**Trigeminal pathways**

Discriminative tactile and pressure sensation

Nearly half of the sensory fibers in the trigeminal nerve are Aβ myelinated discriminative touch fibers. The central processes of some first order neurons terminate in the principal sensory nucleus (without bifurcating). Most of the central processes of pseudounipolar (first order) neurons
enter the pons, they bifurcate into short ascending fibers, which synapse in the principal 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 (descending) tract of the trigeminal to reach their target subnuclei. Some second order fibers from the principal 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 principal 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 primary somatosensory cortex for conscious awareness and 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. 17.10). This short root joins the sensory fibers of the mandibular division of the trigeminal nerve outside the skull, in the infratemporal fossa. 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, and 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 proprioceptive input to the sensory and motor nuclei of the trigeminal, the reticular formation, and cerebellum.

The peripheral processes of the pseudounipolar neurons housed in the mesencephalic nucleus of the trigeminal
CRANIAL NERVES  ●  ●  ●  331

Figure 17.10  ●  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. 17.8.

accompany the motor root of the trigeminal as they both exit the pons. These peripheral processes follow: (1) the motor branches of the mandibular division to the muscle spindles of the muscles of mastication; (2) 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 ligaments synapse in the principal sensory nucleus, the rostral subnuclei of the spinal nucleus and in the motor nucleus of the trigeminal. These central processes also terminate in the reticular formation to mediate reflex responses and the cerebellum which functions to coordinate movement of the mandible during chewing. The principal sensory and rostral spinal nuclei give rise to axons that ascend in the ventral trigeminothalamic tract to terminate in the contralateral VPM nucleus of the thalamus as part of the jaw proprioception pathway.

The jaw proprioception pathway relays proprioceptive input from the mandible to the primary somatosensory cortex where position sense of the mandible enters conscious awareness. In contrast, the jaw jerk reflex (see next) is a protective reflex elicited by muscle stretch (of the temporalis and masseter muscles) and causes the reflex muscle contraction (of the same muscles) to compensate for the muscle stretch. This reflex is limited/restricted to the brainstem and does not reach the somatosensory cortex or conscious awareness.

Jaw jerk (massetric) 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. 17.11). 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 the mesencephalic pseudounipolar neurons usually branch in the vicinity of the motor nucleus of the trigeminal to send collaterals that terminate in the motor nucleus. The central processes of these neurons synapse not only in the motor nucleus bilaterally, but also in the principal sensory nucleus and the reticular formation as well. 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 17.11  ●  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 principal sensory and bilaterally to the motor nucleus of the trigeminal nerve. The motor neurons innervating the masseter muscle cause its contraction. For abbreviations, see Fig. 17.8.
**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. Normally, contraction of one lateral pterygoid muscle swings the mandible to the opposite side. When both lateral pterygoid muscles contract simultaneously, the mandible protrudes forward. With a lesion that damages the motor root of the trigeminal nerve, 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.

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 abducens nerve supplies motor innervation to the lateral rectus muscle, which abducts the eye (a common mnemonic is LR). As the abducens nerve root fascicles exit the abducens nucleus they course ventrolaterally in the caudal pontine tegmentum to exit the brainstem at the pontomedullary junction. The nerve then passes through the space between the posterior inferior cerebellar artery (PICA) and the anterior inferior cerebellar artery (AICA) to enter and ascend in the pontine subarachnoid cistern, next to the basilar artery. It continues anteriorly, traverses the cavernous sinus where it lies next to the internal carotid artery, 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 as a pair and in unison 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 (frontal and parietal motor eye fields) as well as by the brainstem (vestibular nuclei, MLF and the reticular formation). 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 is located in the medulla oblongata (L. “little hill”) in the floor of the fourth ventricle. A group of root fascicles 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.

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.

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.
medial rectus muscle (MR).

The abducens nucleus contains two different populations of neurons (Fig. 17.12). One group (which makes up about 70% of the nucleus neurons) consists of the GSE motoneurons, whose axons form the abducent nerve that innervates the lateral rectus muscle (LR); and (ii) internuclear neurons whose axons cross the midline and join the contralateral MLF to synapse in the oculomotor nucleus with the motoneurons that innervate the medial rectus muscle (MR).

The abducens nucleus is the center for conjugate horizontal eye movement. 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 only with the motoneurons projecting to the contralateral medial rectus muscle causing it to contract so that the opposite eye adducts.

Thus, horizontal gaze to one side is evoked ipsilaterally to the stimulated abducens nucleus, which involves the coordinated and simultaneous contraction of the lateral rectus muscle of the ipsilateral eye and the medial rectus muscle of the contralateral eye. When the right abducens nucleus is stimulated, gaze will be to the right. The simultaneous movement of the two eyes to one side (to the right in this case), is referred to as conjugate horizontal eye movement (eyes move in unison, horizontally), so they both look in the same direction (to the right) and at the same object. Note that the abducens nucleus and the MLF are next to one another and both are adjacent to the midline. Thus, internuclear neuron axons leaving the right abducens nucleus cross immediately to enter the contralateral (left) MLF. The left MLF carries the axons to the left oculomotor nucleus. It is the left MLF that connects the right abducens nucleus to the left oculomotor nucleus.

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 trigeminal ganglion.

**Abducent nerve lesion**

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

A lesion of the abducent nerve (GSE, motor fibers) results in paralysis of the ipsilateral lateral rectus muscle that normally abducts the eye. Since the paralyzed lateral rectus muscle becomes flaccid and lacks muscle tone, the eye will deviate medially as a result of the unopposed action of its antagonist, the medial rectus muscle (Fig. 17.13). The medial deviation of the affected eye, the new resting position of the eye, is referred to as medial strabismus (internal strabismus, esotropia). The individual can turn the ipsilateral (affected) eye from its medial position to the center (to look straight ahead), but not beyond it. Since the normal eye looks straight ahead and the affected eye is medially deviated (looks toward the nose), the eyes become misaligned, causing double vision, horizontal diplopia (double vision; i.e., a single object is perceived as two separate objects next to each other). The diplopia is greatest when attempting to gaze toward the side of the lesion and it is minimized by looking towards the unaffected side since the visual axes become parallel. The individual realizes that the diplopia is minimized by turning his/her head slightly so that the chin is pointing toward the side of the lesion. This would keep the affected eye adducted, and abduct the normal eye, aligning the eyes (eyes become parallel), minimizing the diplopia.

Bilateral abducent nerve lesion results in the individual becoming “cross-eyed.”

When we fix our gaze at an object and then move our head either in the horizontal (or vertical) plane, our eyes reflexly roll away from the direction of
CHAPTER 17

the head movement to keep the image (of the object) on the retina. This is a function of the vestibular nuclear projections to the motor nuclei innervating the extraocular muscles, coordinating head and eye movements.

Abducens nucleus lesion

A lesion involving the abducens nucleus or PPRF results in medial strabismus, horizontal diplopia, and ipsilateral lateral gaze paralysis. A lesion involving the abducens nucleus (Fig. 17.14) or the pontine paramedian reticular formation (PPRF) 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 (MLF) lesion: internuclear ophthalmoplegia (INO)

A lesion to one MLF results in internuclear ophthalmoplegia (INO). If the oculomotor, trochlear, and abducent nerves and their nuclei are intact, but there is a unilateral MLF lesion, most eye movements are possible. Normally, the eyes move as a pair. However, when there is a lesion to one MLF, the abducent and oculomotor nuclei which are connected by the MLF, become

**Figure 17.14** 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.
CLINICAL CONSIDERATIONS (continued)

Figure 17.15  •  Lesion to the right MLF. (A) Normal primary position. (B) Normal gaze to the right. (C) Adduction deficit of right eye, due to lesion in ipsilateral MLF. Abduction of left eye with nystagmus. (D) Vergence of the eyes is normal.

“disconnected,” and conjugate horizontal ocular movement will not occur to one side (Fig. 17.15).

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, abducts with nystagmus. This deficit is referred to as internuclear ophthalmoplegia (INO). INO is a horizontal eye movement disorder produced by a lesion to the MLF. The function of the MLF comes into play only during conjugate horizontal eye movement. An individual who has a lesion to the MLF cannot gaze away from the side of the lesion. If you ask this same individual to look at a near object (for example a pencil) placed directly in front of his nose, which necessitates that both eyes adduct (converge), he is able to do so. This indicates that: (1) both oculomotor nerves (which innervate the medial recti) are intact; (2) the cortical projections from the frontal eye field and parietal motor eye fields to the midbrain reticular formation centers that control eye movements are intact; and (3) the projections from the midbrain reticular formation to the motor nucleus of the oculomotor nerve are intact. Therefore with a lesion to the right MLF the right eye will have an adduction deficit (only during conjugate horizontal eye movement) to the left (when gazing away from the side of the lesion), but not during convergence of the eyes. The reason for this is because during conjugate horizontal eye movement the motor neurons that innervate the medial rectus muscle are stimulated by the MLF, but during convergence of the eyes, the motor neurons to the medial rectus are stimulated by the frontal and parietal motor eye fields via the midbrain reticular formation.

“One-and-a-half”

“One-and-a-half” is a rare condition resulting from a lesion in the vicinity of 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 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 happen:

1. The GSE motoneurons, whose axons form the left abducens 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. This is referred to as ipsilateral lateral (horizontal) gaze palsy, when looking to the side of the lesion. When attempting to gaze to the right, the right eye abducts with nystagmus, whereas the left eye will not be able to adduct during conjugate horizontal gaze to the right. Thus “one-and-a-half” is characterized by a lesion involving a combination of “one” abducens nucleus and a “half,” the axons of the internuclear neurons arising from the opposite abducens nucleus, ascending in the MLF. That is, the eye ipsilateral to the lesion has no horizontal movement and is medially-deviated. The opposite eye can only abduct with nystagmus. 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.

Syndrome of trochlear nerve palsy with internuclear ophthalmoplegia

The trochlear nucleus is located immediately dorsal to the MLF, in the caudal midbrain. A lesion in the vicinity of the trochlear nucleus may also affect the near-by MLF. The close proximity and combined damage of the two structures makes them susceptible to a syndrome of trochlear nerve palsy with internuclear ophthalmoplegia (INO). The ascending MLF contains fibers
that interconnect the abducens, trochlear, and oculomotor nuclei as well as vestibular fibers that project to these nuclei. The MLF contains the axons of internuclear neurons (interneurons) whose cell bodies reside in the abducens nucleus in the caudal pons.

The abducens nucleus receives cortical input from the contralateral frontal eye field (Brodmann’s area 8) relaying motor messages that stimulate the neurons of this nucleus. The abducens nucleus contains two distinct populations of neurons: motor neurons and interneurons. The motor neurons give rise to axons that exit the brainstem to form the abducens nerve which courses to the orbit where it provides motor innervation to a single extraocular muscle, the lateral rectus. This muscle mediates abduction (lateral rotation) of the eye. The interneurons connect the abducens nucleus to the contralateral oculomotor nucleus as follows: The interneurons give rise to axons that leave the abducens nucleus and immediately cross the midline to join the contralateral (ascending) MLF to ascend to the rostral midbrain where they terminate in the contralateral oculomotor nucleus and synapse specifically with the motor neurons that innervate the medial rectus muscle. The abducens nucleus is the center for conjugate horizontal eye movement, thus simultaneous stimulation of both populations of neurons of this nucleus (via the descending axons of neurons residing in Brodmann’s area 8) normally mediates (1) abduction of the ipsilateral eye (via the abducens nerve) and (2) adduction of the contralateral eye (via the interneuron axons running in the MLF, and the oculomotor nucleus) to execute lateral gaze ipsilateral to the side of the stimulated abducens nucleus. As mentioned previously, a lesion in the caudal midbrain, damaging the trochlear nucleus will most likely also damage the ipsilateral MLF immediately adjacent to it, containing the ascending axons of the interneurons whose cell bodies are housed in the abducens nucleus and whose axons are in route to ascend further up to the rostral midbrain to terminate in the oculomotor nucleus.

The MLF located next to the trochlear nucleus at the level of the caudal midbrain, contains internuclear neuron axons that already crossed down below in the brainstem, at the level of the abducens nucleus which resides in the caudal pons. Thus if the right MLF (which projects to the right oculomotor nucleus) is damaged at the level of the caudal midbrain, the right medial rectus will not contract (adduct) during conjugate horizontal eye movement in gaze to the left.

If the (right) trochlear nucleus and the (right) MLF are damaged at the level of the caudal midbrain (when they are next to each other), the superior oblique palsy will show up in the left eye (because the axons leaving the right trochlear nucleus will cross in the midbrain tectum before the nerve emerges from the brainstem as the left trochlear nerve), and the INO will become evident when the individual attempts to gaze to the left (when the right eye is supposed to adduct, but can’t, due to the disconnection of the left abducens nucleus and the right oculomotor nucleus).

**FACIAL NERVE (CN VII)**

The facial nerve provides motor innervation to the muscles of facial expression, taste sensation to the anterior two-thirds of the tongue, and parasympathetic innervation to the lacrimal, sublingual, and submandibular glands. 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 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 then immediately gives rise to the sensory posterior auricular nerve, and the motor branches to the auricularis and occipitalis muscles. The main trunk of the facial nerve then enters the parotid bed where it 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 17.16, 17.17) 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 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. 17.18). 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 nerve, and terminate to synapse in the spinal nucleus of the trigeminal nerve.

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 cell bodies of the first order sensory bipolar neurons of the vestibular nerve reside within the
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 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.)

vendibular ganglion (of Scarpa) (see Fig. 20.6). Their peripheral processes terminate in special receptors, the cristae in the ampullae of the semicircular ducts and the maculae of the utricle and saccule, housed within the petrous temporal bone (see Figs 20.2–20.4). 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. 20.6). 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 first order sensory bipolar neurons of the cochlear nerve are housed within the spiral (cochlear) ganglion (see Fig. 19.3). 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. 19.3). The central processes of these neurons accompany the vestibular nerve to synapse in the cochlear nuclei in the brainstem with second order neurons of the auditory pathway (see Fig. 19.4).

**GLOSSOPHARYNGEAL NERVE (CN IX)**

The glossopharyngeal nerve, one of the smallest cranial nerves, carries five functional components. These are: (1) SVA (taste) from the posterior one-third of the tongue and the adjacent pharyngeal wall, (2) 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), (3) GSA sensation from the external ear, (4) SVE (branchiomotor) innervation to the stylopharyngeus

**CLINICAL CONSIDERATIONS**

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

Compression or damage of the facial nerve results in ipsilateral paralysis of the muscles of facial expression, the most commonly-occurring muscle paralysis in the head. The functional deficits vary and depend on the specific location of the lesion along the course of the facial nerve to its targeted structures.

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. A viral infection that affects the facial nerve’s connective tissue coverings causes inflammation (neuritis) and edema. The swelling compresses the enclosed nerve fibers as they course through the facial canal, a narrow channel, in the petrous portion of the temporal bone and as they exit from the stylomastoid foramen. Compression of the nerve results in ischemia and compromised conduction of the facial nerve fibers. 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 pterygopatine ganglion. The fibers then establish inappropriate synaptic contacts with postganglionic (“lacrimation”) neurons whose fibers project to the lacrimal gland. When the individual is eating, tears are produced by the lacrimal gland ipsilateral to the side of the lesion.

A unilateral lesion damaging the facial nerve after it gives off its branches in the facial canal (that is if only its motor fibers are damaged), it results in paralysis of the muscles of facial expression, ipsilateral to the side of the lesion.

Many individuals in the general population do not have a gag reflex. Baroreceptor fibers terminate in the carotid 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. 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 sensory nucleus of the trigeminal.

The nucleus ambiguus contains the SVE branchiotor 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. 17.20C). These axons then leave the glossopharyngeal...
nerve as the 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. 17.20D). 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.

A unilateral lesion to the glossopharyngeal nerve near its exit from the brainstem, damaging all of its fibers, will include 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, sensory limb of the reflex arc), and *loss of the carotid sinus reflex*. The stylopharyngeus muscle, which elevates the pharynx during swallowing, will be paralyzed.
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. Although it is a cranial nerve, its innervation is not limited to the structures in the head, but also extends into the neck, thorax, and abdomen. The vagus nerve carries five functional components: (1) SVA; (2) GVA; (3) SVE; (4) GVE; and (5) GVE (the 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

![Diagram of Vagus Nerve (CN X)]
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 jugular foramen. Inferior to the jugular foramen, the vagus nerve displays two swellings, the superior (jugular) and inferior (nodose) ganglia of the vagus nerve. 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, larynx, and carotid body, 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, and course in the solitary tract to terminate in the solitary nucleus (Fig. 17.22A).

**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. 17.22A).

GSA pseudounipolar neuron cell bodies conveying pain, temperature, and touch sensation reside in the superior ganglion and send their peripheral processes to the pinna, external auditory meatus, skin of the ear, and tympanic membrane. Their central processes enter the brainstem, join the spinal tract of the trigeminal nerve and terminate in the spinal nucleus of the trigeminal nerve (Fig. 17.22A).

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. 17.22B).

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. 17.22B).

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: (1) the pharyngeal muscles and levator veli palatini of the soft palate, resulting in dysphagia (difficulty swallowing); (2) the laryngeal muscles, resulting in dysphonia (hoarseness) and dyspnea (difficulty breathing); and (3) loss of the gag reflex (efferent, motor 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 (Fig. 17.23) supplies motor innervation to the sternocleidomastoid and trapezius muscles. The spinal accessory nerve is a direct ramification of 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. Slight winging of the scapula is noticeable when the arms are positioned lateral to the trunk, and becomes more obvious during abduction of the affected arm. There is also slight drooping of the ipsilateral shoulder, accompanied by a weakening of the shoulder when attempting to raise it and weakness in abduction of the arm above the horizontal plane. A lesion involving the “cranial root” (aberrant vagal fibers) of the accessory nerve results in paralysis of the laryngeal muscles.
The hypoglossal nerve (Fig. 17.24) 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 dorsomedial aspect of the caudal 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 ventrolaterally in the medullary tegmentum 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 courses to the submandibular region to serve the ipsilateral side of the tongue. The hypoglossal nerve innervates the tongue’s intrinsic muscles (transverse, longitudinals, and vertical), which alter its shape, and all the extrinsic muscles of the tongue (styloglossus, hyoglossus, and genioglossus), with the exception of the palatoglossus, which alter its shape and position. 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.

**HYPOGLOSSAL NERVE (CN XII)**

The hypoglossal nerve provides motor innervation to the muscles of the tongue.
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. Hemiparesis of the tongue causes creasing (wrinkling) of the dorsal surface of the tongue ipsilateral to the lesion. Normally, the simultaneous contraction of the paired genioglossi 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.
### SYNONYMS AND EPONYMS OF THE CRANIAL NERVES

<table>
<thead>
<tr>
<th>Name of structure or term</th>
<th>Synonym(s)/eponym(s)</th>
<th>Name of structure or term</th>
<th>Synonym(s)/eponym(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cochlear ganglion</td>
<td>Spiral ganglion</td>
<td>Postcentral gyrus</td>
<td>Primary sensory cortex (S–I)</td>
</tr>
<tr>
<td>Discriminatory tactile sense</td>
<td>Fine touch sensation</td>
<td></td>
<td>Primary somatosensory cortex</td>
</tr>
<tr>
<td>Dorsal trigeminal lemniscus</td>
<td>Dorsal trigeminotalhamic tract</td>
<td></td>
<td>Primary somesthetic cortex</td>
</tr>
<tr>
<td>Functional components of cranial nerves</td>
<td>Modalities of cranial nerves</td>
<td></td>
<td>Brodmann’s areas 3, 1, and 2</td>
</tr>
<tr>
<td>General somatic efferent (GSE)</td>
<td>Somatic motor</td>
<td></td>
<td>Unipolar neuron</td>
</tr>
<tr>
<td></td>
<td>Somatomotor</td>
<td></td>
<td>Branchiomotor</td>
</tr>
<tr>
<td>General visceral efferent (GVE)</td>
<td>Visceral motor</td>
<td>Spinal nucleus of the trigeminal</td>
<td>Descending nucleus of the trigeminal</td>
</tr>
<tr>
<td></td>
<td>Visceromotor</td>
<td>Spinal tract of the trigeminal</td>
<td>Descending tract of the trigeminal</td>
</tr>
<tr>
<td></td>
<td>Secretomotor</td>
<td>Subnucleus caudalis of the spinal trigeminal nucleus</td>
<td>Pars caudalis of the spinal trigeminal nucleus</td>
</tr>
<tr>
<td>Inferior ganglion of the vagus nerve</td>
<td>Nodose ganglion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internuclear neurons</td>
<td>Interneurons</td>
<td>Subnucleus interpolaris of the spinal trigeminal nucleus</td>
<td>Pars interpolaris of the spinal trigeminal nucleus</td>
</tr>
<tr>
<td>Main nucleus of the trigeminal</td>
<td>Chief nucleus of the trigeminal</td>
<td>Subnucleus oralis of the spinal trigeminal nucleus</td>
<td>Pars oralis of the spinal trigeminal nucleus</td>
</tr>
<tr>
<td>Mandibular division of the trigeminal nerve</td>
<td>Mandibular nerve</td>
<td>Superior ganglion of the vagus nerve</td>
<td>Jugular ganglion</td>
</tr>
<tr>
<td>Maxillary division of the trigeminal nerve</td>
<td>Maxillary nerve</td>
<td>Trigeminal neuralgia</td>
<td>Trigeminal nerve pain</td>
</tr>
<tr>
<td>Medial strabismus</td>
<td>Internal strabismus</td>
<td>Ventral trigeminal lemniscus</td>
<td>Ventral trigeminotalhamic tract</td>
</tr>
<tr>
<td></td>
<td>Convergent strabismus</td>
<td>Vestibular ganglion</td>
<td>Vestibular ganglion of Scarpa</td>
</tr>
<tr>
<td></td>
<td>Esotropia</td>
<td>Vestibulocochlear nerve</td>
<td>Acoustic nerve (older term)</td>
</tr>
<tr>
<td>Ophthalmic division of the trigeminal nerve</td>
<td>Ophthalmic nerve</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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 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. Multiple sclerosis is an autoimmune disease that affects only the myelin sheaths of axons in the central nervous system (CNS). Affected individuals display an autoimmune reaction that kills oligodendrocytes, the glial cells that myelinate axons in the CNS. Demyelination of axons follows with subsequent formation of sclerotic plaques, which have an adverse effect on nerve impulse transmission.

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.

**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 are some treatment options for trigeminal neuralgia?

6. What eye movement deficits result following a lesion in the vicinity of the abducens nucleus?

7. What is the cause of the “crocodile tear syndrome”?

8. 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.