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A 41-year-old woman noticed that she could not hear anything from the phone receiver when it was on her left ear. Also, over the previous year, she sometimes felt as though the room was spinning slightly when she moved her head. She developed some pain on the left side of her face and decreased taste on the left side of her tongue, as well as a decreased corneal reflex on the left side.

This patient illustrates the complex variety of abnormalities that patients with cranial nerve disorders may experience. In this chapter, we will learn about the origins of cranial nerves in the brainstem, their courses to the periphery, and their various functions.
Located at the base of the cerebral hemispheres, the brainstem is a compact, stalklike structure that carries nearly all information between the brain and the remainder of the body (Figure 12.1). This tight space is the corridor to all major sensory, motor, cerebellar, and cranial nerve pathways. However, the brainstem is not simply a conduit for information. It also contains numerous important nuclei of its own, which control the cranial nerves; level of consciousness; cerebellar circuits; muscle tone; posture; and cardiac, respiratory, and numerous other essential functions. If the brain were a city, then the brainstem would be both the Grand Central Station and Central Power Supply packed into one location. Thus, small lesions in the brainstem can result in substantial deficits, often involving multiple motor, sensory, and neuroregulatory modalities.

An intimate knowledge of brainstem anatomy is a powerful clinical tool. Armed with an understanding of brainstem nuclei and pathways, the clinician can intelligently decide on appropriate diagnostic and therapeutic measures for patients with brainstem disorders. Brainstem anatomy is so complex yet so clinically relevant that we devote three full chapters (Chapters 12–14) to understanding it in detail. Thus, in this chapter we will first review the surface features of the brainstem and then discuss the course and functions of each cranial nerve. Next, in Chapter 13 we will focus in greater detail on the cranial nerves and central pathways mediating eye movements and pupillary control. Finally, in Chapter 14 we will study the vascular supply and internal structures of the brainstem, including the major ascending and descending tracts, reticular formation, and other important brainstem nuclei.
Learning the cranial nerves initially requires some memorization. Over time, however, they become very familiar because of their important clinical relevance. The numbers, names, and main functions of the cranial nerves are listed in Table 12.1. Note that the cranial nerves have both sensory and motor functions. To learn the cranial nerves and their functions, two different review strategies are useful. In one, the cranial nerves are listed in numerical sequence and the sensory and motor functions of each nerve are discussed (see Table 12.4). In the second, the different sensory and motor cranial nerve nuclei are listed, and the functions and cranial nerves subserved by each nucleus are discussed (see Table 12.3). Both approaches are clinically relevant, and we will use both strategies at various points in these brainstem chapters to integrate knowledge of the peripheral and central course of the cranial nerves.

### Surface Features of the Brainstem

The brainstem consists of the midbrain, pons, and medulla (see Figure 12.1). It lies within the posterior fossa of the cranial cavity. The rostral limit of the brainstem is the midbrain–diencephalic junction (see Figure 12.1). Here the brainstem meets thalamus and hypothalamus at the level of the tentorium cerebelli. Midbrain joins pons at the pontomesencephalic junction, and pons meets medulla at the pontomedullary junction. The caudal limit of the brainstem is the cervicomедullary junction, at the level of the foramen magnum and pyramidal decussation (see Figure 12.1, Figure 12.2A; see also Figure 6.8). The cerebellum is attached to the dorsal surface of the pons and upper medulla (see Figure 12.1). Although some authors have included the cerebellum or thalamus in the term “brainstem,” we adopt common clinical usage here and take brainstem to imply only midbrain, pons, and medulla. We discuss the thalamus and cerebellum at greater length elsewhere (see Chapters 7 and 15).

On the dorsal surface of the midbrain are two pairs of bumps called the superior colliculi and inferior colliculi (Figure 12.2B). Together, these form the tec-
Chapter 12

(A) Interpeduncular fossa

- Oculomotor nerve (CN III)
- Trochlear nerve (CN IV)
- Trigeminal nerve (CN V)
- Abducens nerve (CN VI)
- Facial nerve (CN VII)

(B) Cerebral peduncle

- Vagus (CN X)

- Facial colliculus
- Hypoglossal trigone
- Vagal trigone

- Middle cerebellar peduncle
- Inferior cerebellar peduncle

- Dorsal (posterior) columns:
  - Fasciculus gracilis
  - Fasciculus cuneatus

- Dorsal (posterior) column tubercles:
  - Nucleus cuneatus
  - Nucleus gracilis

- Superior colliculus
- Inferior colliculus
- Anterior medullary velum

- Superior cerebellar peduncle
- Middle cerebellar peduncle
- Inferior cerebellar peduncle

- Obex

- Brachium of superior colliculus
- Brachium of inferior colliculus
- Trochlear nerve (CN IV)
- Facial colliculus

- Hypoglossal trigone
- Glossopharyngeal nerve (CN IX)

- Vagus (CN X)

- Vagus nerve (CN X)
- Hypoglossal nerve (CN XII)
- Spinal accessory nerve (CN XI)

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**Brainstem I: Surface Anatomy and Cranial Nerves**

**tum** (meaning “roof”) of the midbrain. The ventral surface of the midbrain is formed by the cerebral peduncles, between which lies the interpeduncular fossa (see Figure 12.2A; see also Figure 5.6). The pons is limited dorsally by the fourth ventricle (see Figure 12.1). More dorsolaterally, the pons is attached to the cerebellum by large white matter tracts called the superior, middle, and inferior cerebellar peduncles (see Figure 12.2B). On the ventral surface of the medulla, the pyramids can be seen descending from the pontomedullary junction to the pyramidal decussation (see Figure 12.2A). It is often useful to divide the medulla into a rostral portion and a caudal portion. In the rostral medulla the prominent bulges of the inferior olivary nuclei can be seen just lateral to the pyramids (see Figure 12.2A). In the caudal medulla the inferior olivary nuclei are no longer seen, but the posterior columns and posterior column nuclei are visible on the dorsal surface (see Figure 12.2B).

The floor of the fourth ventricle extends from the pons to the rostral half of the medulla. Along the floor of the fourth ventricle, several bumps are visible. These include the facial colliculi, formed by the abducens nuclei and fibers of the facial nerve (see Figure 12.2B; see also Figure 14.1C). The hypoglossal trigone and vagal trigone (see Figure 12.2B) are formed by the hypoglossal nucleus (CN XII) and the dorsal motor nucleus of CN X, respectively. Recall that rostrally, the fourth ventricle joins the cerebral aqueduct, which runs through the midbrain (see Figure 12.1). Caudally, the fourth ventricle drains into the subarachnoid space via the foramina of Luschka (located laterally) and foramen of Magendie (located in the midline). The fourth ventricle ends caudally at the obex (see Figure 12.2B), marking the entry to the spinal cord central canal, which in adults is normally closed.

---

**FIGURE 12.2 Surface Anatomy of the Brainstem and Cranial Nerves**
(A) Ventral view with cerebral hemispheres removed. (B) Dorsal view with cerebellum removed, exposing floor of the fourth ventricle. (C) Lateral view.
For those who are verbally inclined, several mnemonics exist for the cranial nerve names and numbers (see Table 12.1). However, the best visual mnemonic for the cranial nerves is the brainstem itself, since the cranial nerves emerge roughly in numerical sequence from I through XII proceeding from rostral to caudal (see Figure 12.2). The first two cranial nerves do not emerge from the brainstem, but rather connect directly to the forebrain. The olfactory nerves (CN I) enter the olfactory bulbs and olfactory tracts, which run along the ventral surface of the frontal lobes in the olfactory sulci (see Figures 18.5 and 18.6). The optic nerves (CN II) meet at the optic chiasm, forming the optic tracts, which wrap laterally around the midbrain to enter the lateral geniculate nuclei of the thalamus (see Figures 11.6 and 11.15).

Cranial nerves III–XII exit the brainstem either ventrally or ventrolaterally (see Figure 12.2A and Figure 12.2C). The one exception is CN IV, which exits from the dorsal midbrain (see Figure 12.2B). We will see shortly that CN III, VI, and XII, which exit ventrally near the midline, together with CN IV, which exits dorsally, form a distinct functional group innervating somatic motor structures.

The oculomotor nerves (CN III) emerge ventrally from the interpeduncular fossa of the midbrain (see Figure 12.2A). Note that the oculomotor nerve usually passes between the posterior cerebral artery and the superior cerebellar artery (see Figure 14.18A). As we just mentioned, the trochlear nerve (CN IV) is exceptional in exiting dorsally from the midbrain (see Figure 12.2B). The fibers of CN IV cross over as they emerge, an arrangement that is also unique to this cranial nerve. The trigeminal nerve (CN V) exits from the ventrolateral pons (see Figure 12.2A,C). The abducens nerve (CN VI) exits ventrally, at the pontomedullary junction (see Figure 12.2A,C). Then, proceeding in sequence, the facial nerve (CN VII), vestibulocochlear nerve (CN VIII), glossopharyngeal nerve (CN IX), and vagus nerve (CN X), exit ventrolaterally from the pontomedullary junction and rostral medulla. The region where CN VII, VIII, and IX exit the brainstem is called the cerebellopontine angle. The spinal accessory nerve (also known as the accessory spinal nerve; CN XI) arises laterally from multiple rootlets along the upper cervical spinal cord. The hypoglossal nerve (CN XII) exits the medulla ventrally, between the pyramids and inferior olivary nuclei (see Figure 12.2A).

Skull Foramina and Cranial Nerve Exit Points

When we discuss each cranial nerve in the sections that follow, we will describe its course in detail. For now, we will simply introduce the foramina through which the cranial nerves exit the skull (Figure 12.3; Table 12.2).

The olfactory nerves exit via the cribriform plate, and the optic nerve via the optic canal (see Figure 12.3; Table 12.2). The superior orbital fissure transmits several nerves (CN III, IV, VI, and V₁) into the orbit (see Figure 12.3A, C). CN III, IV, and VI mediate eye movements. The ophthalmic division of CN V (CN V₁) conveys sensation for the eye and upper face. The maxillary (CN V₂) and mandibular (CN V₃) divisions of the trigeminal nerve exit via the foramen rotundum and foramen ovale, respectively, providing sensation to the remainder of the face (see Figure 12.7). CN VII and VIII both exit the cranial cavity via the internal auditory meatus to enter the auditory canal. CN VIII innervates the inner ear deep within the temporal bone. CN VII exits the skull to reach the muscles of facial expression via the stylomastoid foramen (see Figure 12.3B). The jugular foramen transmits CN IX, X, and XI (see Figure 12.3A,B). Finally, the hypoglossal nerve (CN XII), controlling tongue movements, exits the skull via its own foramen, the hypoglossal canal, which lies just in front of the foramen magnum.
FIGURE 12.3  Skull Foramina Serving as Cranial Nerve Exit Points  (A) Inside view of the base of the skull, seen from above, with cranial nerves shown on the right and exit foramina shown on the left. (B) View of the base of the skull, seen from below. (C) Anterior view of the skull and foramina.
**Chapter 12**

**Sensory and Motor Organization of the Cranial Nerves**

The cranial nerves are analogous in some ways to the spinal nerves, having both sensory and motor functions. Also, like the spinal cord, motor cranial nerve nuclei are located more ventrally, while sensory cranial nerve nuclei are located more dorsally (Figure 12.4). However, cranial nerve sensory and motor functions are more specialized because of the unique anatomy of the head and neck. During embryological development, the cranial nerve nuclei lie adjacent to the ventricular system (see Figure 12.4A). As the nervous sys-

**TABLE 12.2 Cranial Nerve Exit Foramina**

<table>
<thead>
<tr>
<th>CN</th>
<th>NAME</th>
<th>EXIT FORAMEN</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN I</td>
<td>Olfactory nerves</td>
<td>Cribriform plate</td>
</tr>
<tr>
<td>CN II</td>
<td>Optic nerve</td>
<td>Optic canal</td>
</tr>
<tr>
<td>CN III</td>
<td>Oculomotor nerve</td>
<td>Superior orbital fissure</td>
</tr>
<tr>
<td>CN IV</td>
<td>Trochlear nerve</td>
<td>Superior orbital fissure</td>
</tr>
<tr>
<td>CN V</td>
<td>Trigeminal nerve</td>
<td>V₁: Superior orbital fissure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>V₂: Foramen rotundum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>V₃: Foramen ovale</td>
</tr>
<tr>
<td>CN VI</td>
<td>Abducens nerve</td>
<td>Superior orbital fissure a</td>
</tr>
<tr>
<td>CN VII</td>
<td>Facial nerve</td>
<td>Auditory canal (stylomastoid foramen)</td>
</tr>
<tr>
<td>CN VIII</td>
<td>Vestibulocochlear nerve</td>
<td>Auditory canal</td>
</tr>
<tr>
<td>CN IX</td>
<td>Glossopharyngeal nerve</td>
<td>Jugular foramen</td>
</tr>
<tr>
<td>CN X</td>
<td>Vagus nerve</td>
<td>Jugular foramen</td>
</tr>
<tr>
<td>CN XI</td>
<td>Spinal accessory nerve</td>
<td>Jugular foramen (enters skull via foramen magnum)</td>
</tr>
<tr>
<td>CN XII</td>
<td>Hypoglossal nerve</td>
<td>Hypoglossal foramen (canal)</td>
</tr>
</tbody>
</table>

*The abducens nerve first exits the dura through Dorello’s canal (see Figure 12.3) and then travels a long distance before exiting the skull at the superior orbital fissure.*

**Sensory and Motor Organization of the Cranial Nerves**

The cranial nerves are analogous in some ways to the spinal nerves, having both sensory and motor functions. Also, like the spinal cord, motor cranial nerve nuclei are located more ventrally, while sensory cranial nerve nuclei are located more dorsally (Figure 12.4). However, cranial nerve sensory and motor functions are more specialized because of the unique anatomy of the head and neck. During embryological development, the cranial nerve nuclei lie adjacent to the ventricular system (see Figure 12.4A). As the nervous sys-

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Brainstem I: Surface Anatomy and Cranial Nerves 501

tem matures, three motor columns and three sensory columns of cranial nerve nuclei develop that run in an interrupted fashion through the length of the brainstem (see Figure 12.4 and Figure 12.5). Each column subserves a different motor or sensory cranial nerve function, which can be classified as shown in Table 12.3. The color codes for each column used in Figures 12.4 and 12.5 and in Table 12.3 will remain constant throughout this chapter. In another set of terminology described toward the end of this section (and listed in the figures and tables) each column can also be described as general vs. special, somatic vs. visceral, and afferent vs. efferent. Let’s review each of these columns in more detail, moving from medial to lateral.

**Motor nuclei**
- Edinger–Westphal nucleus (GVE: CN III)
- Oculomotor nucleus (GSE: CN III)
- Trochlear nucleus (GSE: CN IV)
- Trigeminal motor nucleus (SVE: CN V)
- Superior salivatory nucleus (GVE: CN VII)
- Abducens nucleus (GSE: CN VI)
- Inferior salivatory nucleus (GVE: CN IX)
- Nucleus ambiguus (SVE: CN IX, X)
- Hypoglossal nucleus (GSE: CN XII)
- Dorsal motor nucleus of CN X (GVE: CN X)
- Spinal accessory nucleus (SVE: CN XI)

**Sensory nuclei**
- Trigeminal nuclei (GSA: CN V, VII, IX, X):
  - Mesencephalic nucleus of CN V
  - Chief sensory nucleus of CN V
  - Spinal trigeminal nucleus
- Vestibular nuclei (SSA: CN VIII)
- Dorsal and ventral cochlear nuclei (SSA: CN VIII)
- Nucleus solitarius, rostral portion (SVA: CN VII, IX, X)
- Nucleus solitarius, caudal portion (GVA: CN IX, X)

**Figure 12.5 Functional Columns of Brainstem Sensory and Motor Cranial Nerve Nuclei**
Longitudinal schematic. GSA, general somatic afferent; GSE, general somatic efferent; GVA, general visceral afferent; GVE, general visceral efferent; SSA, special somatic afferent; SVA, special visceral afferent; SVE, special visceral efferent.

Branchial motor column = SVE
Parasympathetic column = GVE
Somatic motor column = GSE
Visceral sensory column = SVA and GVA

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The somatic motor nuclei are the oculomotor (CN III), trochlear (CN IV), abducens (CN VI), and hypoglossal (CN XII) nuclei, all of which remain adjacent to the midline (see Figures 12.4 and 12.5; Table 12.3). These nuclei send their fibers to exit the brainstem close to the midline as well (see Figure 12.2). The somatic motor nuclei innervate the extraocular and intrinsic tongue muscles, which are derived embryologically from the occipital somites.

The visceral motor nuclei (see Figure 12.4A) separate into two different columns of nuclei: branchial motor nuclei and parasympathetic nuclei (see Table 12.3). The branchial motor nuclei are the trigeminal motor nucleus (CN V), facial nucleus (CN VII), nucleus ambiguus (CN IX, X), and spinal accessory nucleus (CN XI) (see Table 12.3). During development, the branchial motor nuclei initially lie just lateral to the somatic motor nuclei (see Figure 12.4A), but they gradually migrate ventrolaterally into the tegmentum (see Figures 12.4B and 12.5). Both the somatic and branchial motor nuclei innervate striated muscles. However, unlike the somatic motor nuclei, the branchial motor nuclei innervate muscles derived from the branchial arches, including the muscles of mastication, facial expression, middle ear, pharynx, and larynx. Based on the fact that the sternomastoid and upper portion of the trapezius (innervated by CN XI) may derive from somites rather than the branchial arches, some classify the spinal accessory nucleus as somatic or mixed somatic and branchial motor. However, for simplicity, and since the spinal accessory nucleus is located laterally in approximate continuity with the nucleus ambiguus, we will consider the spinal accessory nucleus to be part of the branchial motor column.

The next column comprises the parasympathetic nuclei (see Figure 12.5). These are the Edinger–Westphal nucleus (CN III), superior (CN VII) and infer-
rior (CN IX) salivatory nuclei, and the dorsal motor nucleus of the vagus (CN X) (see Table 12.3; see also Figure 12.6). The parasympathetic nuclei do not innervate striated muscle. They provide preganglionic parasympathetic fibers innervating glands, smooth muscle, and cardiac muscle of the head, heart, lungs, and digestive tract above the splenic flexure (see also Figure 6.13).

Continuing laterally, the sensory nuclei also form three columns (see Figures 12.4 and 12.5). The visceral sensory column contains a single nucleus, the nucleus solitarius, which has two parts. The rostral nucleus solitarius, or gustatory nucleus, receives taste inputs primarily from CN VII, but also from CN IX and X. The caudal nucleus solitarius, or cardiorespiratory nucleus, receives inputs for regulation of cardiac, respiratory, and gastrointestinal function from CN IX and X (see Table 12.3). As discussed in Chapter 14, the nucleus solitarius has other functions as well, such as sleep regulation.

The general somatosensory nuclei, or trigeminal nuclei (see Table 12.3; Figures 12.4 and 12.5), mediate touch, pain, temperature, position, and vibration sense for the face, sinuses, and meninges. As we will see, sensory inputs to the trigeminal sensory nuclei are mostly from the trigeminal nerve (CN V), but there are also smaller sensory inputs from CN VII, IX, and X (see Figure 12.7B).

By definition, the special senses are olfaction, vision, hearing, vestibular sense, and taste. Olfaction and vision do not have their primary sensory nuclei in the brainstem. The brainstem special somatic sensory nuclei are the cochlear nuclei mediating hearing (CN VIII) and the vestibular nuclei mediating positional equilibrium (CN VIII) (see Figures 12.4 and 12.5; Table 12.3). Note that although taste is one of the special senses, it has been classified here in the visceral sensory column (nucleus solitarius).

As we mentioned earlier, in another commonly used classification scheme, cranial nerve functions are referred to as either general or special, somatic or visceral, and afferent or efferent. Combinations of these terms result in a total of eight possible categories, such as general somatic efferent (GSE), special visceral afferent (SVA), and so on. However, this scheme still results in the same six longitudinal columns of nuclei (three motor, three sensory) described above (see Figure 12.5; Table 12.3). The reasons are that (1) the somatic motor column is not divided into special and general categories, and (2) the nucleus solitarius contains both the special and general visceral afferents.

Note that most cranial nerve nuclei project to or receive inputs from predominantly one cranial nerve (see Figure 12.5). The three exceptions can be remembered with the mnemonic SAT for Solitarius, Ambiguus, and Trigeminal, all of which are long nuclei extending into the medulla (see Figure 12.5).

### Functions and Course of the Cranial Nerves

In the sections that follow we will review each of the cranial nerves and their functions in detail. Table 12.4 lists the motor and sensory functions of each cranial nerve. Note that some cranial nerves are purely motor (CN III, IV, VI, XI, XII), some are purely sensory (CN I, II, VIII), and some have both motor and sensory functions (CN V, VII, IX, X). The information contained in Table 12.4 is of central importance to understanding the cranial nerves and should be very familiar to you by the time you complete this chapter. The relevant portions of Table 12.4 will be repeated as we introduce each cranial nerve in the sections that follow.

In addition to describing sensory and motor functions, we will review the course of each cranial nerve from brainstem nuclei to peripheral terminations, including the intracranial course of each cranial nerve, skull exit points (see Table 12.2), cranial nerve branches, and peripheral sensory or parasympathetic ganglia (Table 12.5). As we discuss each cranial nerve, we will also review common clinical disorders associated with it.
<table>
<thead>
<tr>
<th>NERVE</th>
<th>NAME</th>
<th>FUNCTIONAL CATEGORIES</th>
<th>FUNCTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN I</td>
<td>Olfactory nerve</td>
<td>Special somatic sensory</td>
<td>Olfaction</td>
</tr>
<tr>
<td>CN II</td>
<td>Optic nerve</td>
<td>Special somatic sensory</td>
<td>Vision</td>
</tr>
<tr>
<td>CN III</td>
<td>Oculomotor nerve</td>
<td>Somatic motor</td>
<td>Levator palpebrae superior and all extracocular muscles, except for superior oblique and lateral rectus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parasympathetic</td>
<td>Parasympathetics to pupil constrictor and ciliary muscles for near vision</td>
</tr>
<tr>
<td>CN IV</td>
<td>Trochlear nerve</td>
<td>Somatic motor</td>
<td>Superior oblique muscle; causes depression and intorsion of the eye</td>
</tr>
<tr>
<td>CN V</td>
<td>Trigeminal nerve</td>
<td>General somatic sensory</td>
<td>Sensations of touch, pain, temperature, joint position, and vibration for the face, mouth, anterior two-thirds of tongue, nasal sinuses, and meninges</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Branchial motor</td>
<td>Muscles of mastication and tensor tympani muscle</td>
</tr>
<tr>
<td>CN VI</td>
<td>Abducens nerve</td>
<td>Somatic motor</td>
<td>Lateral rectus muscle; causes abduction of the eye</td>
</tr>
<tr>
<td>CN VII</td>
<td>Facial nerve</td>
<td>Branchial motor</td>
<td>Muscles of facial expression, stapedius muscle, and part of digastic muscle</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parasympathetic</td>
<td>Parasympathetics to lacrimal glands, and to sublingual, submandibular, and all other salivary glands except parotid</td>
</tr>
<tr>
<td>CN VIII</td>
<td>Vestibulocochlear nerve</td>
<td>Special somatic sensory</td>
<td>Hearing and vestibular sensation</td>
</tr>
<tr>
<td>CN IX</td>
<td>Glossopharyngeal nerve</td>
<td>Branchial motor</td>
<td>Stylopharyngeal muscle</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parasympathetic</td>
<td>Parasympathetics to parotid gland</td>
</tr>
<tr>
<td>CN X</td>
<td>Vagus nerve</td>
<td>General somatic sensory</td>
<td>Sensation from middle ear, region near the external auditory meatus, pharynx, and posterior one-third of tongue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral sensory (special)</td>
<td>Taste from posterior one-third of tongue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral sensory (general)</td>
<td>Chemo- and baroreceptors of carotid body</td>
</tr>
<tr>
<td>CN XI</td>
<td>Spinal accessory nerve</td>
<td>Branchial motor</td>
<td>Pharyngeal muscles (swallowing) and laryngeal muscles (voice box)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parasympathetic</td>
<td>Parasympathetics to heart, lungs, and digestive tract down to the splenic flexure</td>
</tr>
<tr>
<td>CN XII</td>
<td>Hypoglossal nerve</td>
<td>General somatic sensory</td>
<td>Sensation from pharynx, meninges, and a small region near the external auditory meatus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral sensory (special)</td>
<td>Taste from epiglottis and pharynx</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral sensory (general)</td>
<td>Chemo- and baroreceptors of the aortic arch</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Branchial motor</td>
<td>Sternomastoid and upper part of trapezius muscle</td>
</tr>
</tbody>
</table>

Note: See Table 12.3 and Figure 12.5 for nuclei.
CN I: Olfactory Nerve

**TABLE 12.5** Cranial Nerves: Peripheral Sensory and Parasympathetic Ganglia

<table>
<thead>
<tr>
<th>NERVE</th>
<th>NAME</th>
<th>PERIPHERAL GANGLIA</th>
<th>FUNCTION(S) OF GANGLIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN I</td>
<td>Olfactory nerve</td>
<td>None</td>
<td>—</td>
</tr>
<tr>
<td>CN II</td>
<td>Optic nerve</td>
<td>None (retina)</td>
<td>—</td>
</tr>
<tr>
<td>CN III</td>
<td>Oculomotor nerve</td>
<td>Ciliary ganglion</td>
<td>Parasympathetics to iris and ciliary muscle</td>
</tr>
<tr>
<td>CN IV</td>
<td>Trochlear nerve</td>
<td>None</td>
<td>—</td>
</tr>
<tr>
<td>CN V</td>
<td>Trigeminal nerve</td>
<td>Trigeminal ganglion (semitunlar or gasserian ganglion)</td>
<td>Primary sensory neuron cell bodies for sensation in the face, mouth, sinuses, and supratentorial meninges</td>
</tr>
<tr>
<td>CN VI</td>
<td>Abducens nerve</td>
<td>None</td>
<td>—</td>
</tr>
<tr>
<td>CN VII</td>
<td>Facial nerve</td>
<td>Sphenopalatine ganglion</td>
<td>Parasympathetics to lacrimal glands and nasal mucosa</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(pterygopalatine ganglion)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Submandibular ganglion</td>
<td>Parasympathetics to submandibular and sublingual salivary glands</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Geniculate ganglion</td>
<td>Primary sensory neuron cell bodies for taste sensation in anterior two-thirds of tongue, and for sensation near outer ear</td>
</tr>
<tr>
<td>CN VIII</td>
<td>Vestibulocochlear nerve</td>
<td>Spiralganglion</td>
<td>Primary sensory neuron cell bodies for hearing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Scarpa’s vestibular ganglion</td>
<td>Primary sensory neuron cell bodies for vestibular sensation</td>
</tr>
<tr>
<td>CN IX</td>
<td>Glossopharyngeal nerve</td>
<td>Otic ganglion</td>
<td>Parasympathetics to parotid gland</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Superior (jugular) glossopharyngeal ganglion</td>
<td>Primary sensory neuron cell bodies for sensation from middle ear, external auditory meatus, pharynx, and posterior one-third of tongue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inferior (petrosal) glossopharyngeal ganglion</td>
<td>Primary sensory neuron cell bodies for sensation from middle ear, external auditory meatus, pharynx, posterior one-third of tongue, for taste from posterior tongue, and for carotid body inputs</td>
</tr>
<tr>
<td>CN X</td>
<td>Vagus nerve</td>
<td>Parasympathetic ganglia in end organs</td>
<td>Parasympathetics to heart, lungs, and digestive tract to level of splenic flexure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Superior (jugular) vagal ganglion</td>
<td>Primary sensory neuron cell bodies for sensation from pharynx, outer ear, and infratentorial meninges</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inferior (nodose) vagal ganglion</td>
<td>Primary sensory neuron cell bodies for laryngeal sensation, for taste from epiglottis, and for reflex inputs from aortic arch receptors and other thoracoabdominal viscera</td>
</tr>
<tr>
<td>CN XI</td>
<td>Spinal accessory nerve</td>
<td>None</td>
<td>—</td>
</tr>
<tr>
<td>CN XII</td>
<td>Hypoglossal nerve</td>
<td>None</td>
<td>—</td>
</tr>
</tbody>
</table>

**Functional Category**

<table>
<thead>
<tr>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Special somatic sensory</td>
</tr>
<tr>
<td>Olfaction</td>
</tr>
</tbody>
</table>

Olfactory stimuli are detected by specialized chemoreceptors on bipolar primary sensory neurons in the olfactory neuroepithelium of the upper nasal cavities. Axons of these neurons travel via short **olfactory nerves** that traverse the **cribriform plate** of the ethmoid bone (see Figure 12.3A; Table 12.2) to synapse in the **olfactory bulbs** (see Figures 18.5 and 18.6). From the olfactory bulbs, information travels via the **olfactory tracts**, which run in the olfactory sulcus between the gyrus rectus and orbital frontal gyri to reach olfactory processing areas, as discussed in Chapter 18. Note that although the olfactory bulbs and tracts are sometimes called CN I, these structures are actually not nerves, but are part of the central nervous system.

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KEY CLINICAL CONCEPT

12.1 ANOSMIA (CN I)

Patients with unilateral anosmia, or olfactory loss, are rarely aware of the deficit because olfaction in the contralateral nostril can compensate. Therefore, when testing olfaction, the examiner must test each nostril separately (see neuroexam.com Video 24). Patients are often aware of bilateral anosmia and may complain of decreased taste because of the important contribution of olfaction to the perception of flavor.

Loss of the sense of smell can be caused by head trauma, which damages the olfactory nerves as they penetrate the cribriform plate of the ethmoid. In addition, viral infections can damage the olfactory neuroepithelium. Obstruction of the nasal passages can impair olfaction. Bilateral anosmia is also common in patients with certain neurodegenerative conditions such as Parkinson’s disease and Alzheimer’s disease.

Intracranial lesions that occur at the base of the frontal lobes near the olfactory sulci can interfere with olfaction. Possible lesions in this location include meningioma, metastases, basal meningitis or less commonly, sarcoidosis, a granulomatous inflammatory disorder that occasionally involves the nervous system, often causing cranial neuropathies. As we will discuss in KCC 19.11, frontal lobe deficits are often difficult to detect clinically, especially with small lesions. Therefore, lesions at the base of the frontal lobes can sometimes grow to a very large size, causing little obvious dysfunction other than anosmia. Large lesions of the olfactory sulcus region (typically meningiomas) can also sometimes produce a condition called Foster Kennedy syndrome, in which there is anosmia together with optic atrophy in one eye (caused by ipsilateral tumor compression) and papilledema in the other eye (caused by elevated intracranial pressure).

CN II: Optic Nerve

<table>
<thead>
<tr>
<th>FUNCTIONAL CATEGORY</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Special somatic sensory</td>
<td>Vision</td>
</tr>
</tbody>
</table>

As we discussed in Chapter 11, the optic nerve carries visual information from the retina to the lateral geniculate nucleus of the thalamus and to the extrageniculate pathways (see Figures 11.6, 11.15, and 12.2A). The retinal ganglion cells are actually part of the central nervous system, so the optic nerves are, strictly speaking, tracts and not nerves. Nevertheless, by widely accepted convention the portion of the visual pathway in front of the optic chiasm is called the optic nerve, and beyond this point it is referred to as the optic tract. The optic nerves travel from the orbit to the intracranial cavity via the optic canal (see Figure 12.3A,C; Table 12.2). The anatomy and disorders of visual pathways are discussed in greater detail in Chapter 11.

CN III, IV, and VI: Oculomotor, Trochlear, and Abducens Nerves

<table>
<thead>
<tr>
<th>NERVE</th>
<th>FUNCTIONAL CATEGORY</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN III</td>
<td>Somatic motor</td>
<td>Levator palpebrae superior and all extraocular muscles, except for superior oblique and lateral rectus</td>
</tr>
<tr>
<td></td>
<td>Parasympathetic</td>
<td>Parasympathetics to pupil constrictor and ciliary muscles for near vision</td>
</tr>
<tr>
<td>CN IV</td>
<td>Somatic motor</td>
<td>Superior oblique muscle; causes depression motor and intorsion of the eye</td>
</tr>
<tr>
<td>CN VI</td>
<td>Somatic motor</td>
<td>Lateral rectus muscle; causes abduction of the eye</td>
</tr>
</tbody>
</table>
These nerves, which are responsible for controlling the extraocular muscles, will be discussed in detail in Chapter 13. Briefly, CN VI abducts the eye laterally in the horizontal direction; CN IV acts through a trochlea, or pulley-like, structure in the orbit, to rotate the top of the eye medially and move it downward; and CN III subserves all other eye movements. The oculomotor (CN III) and trochlear (CN IV) nuclei are located in the midbrain, and the abducens (CN VI) nucleus is in the pons (see Figures 12.5, 14.3, and 14.4C). Recall that CN III exits the brainstem ventrally in the interpeduncular fossa, CN IV exits dorsally from the inferior tectum, and CN VI exits ventrally at the pontomedullary junction (see Figure 12.2). CN III, IV, and VI then traverse the cavernous sinus (see Figure 13.11), and exit the skull via the superior orbital fissure (see Figure 12.3A,C; Table 12.2) to reach the muscles of the orbit. CN III also carries parasympathetics to the pupillary constrictor and to the ciliary muscle of the lens. The preganglionic parasympathetic neurons are located in the Edinger–Westphal nucleus in the midbrain (see Figure 12.5). They synapse in the ciliary ganglion located in the orbit (Figure 12.6). Postganglionic parasympathetic fibers then continue to the pupillary constrictor and ciliary muscles.

Other cranial nerve parasympathetics are also summarized in Figure 12.6.
The name “trigeminal” was given to this nerve because it has three major branches: the opthalmic division \((V_1)\), maxillary division \((V_2)\), and mandibular division \((V_3)\) (Figure 12.7). The trigeminal nerve provides sensory innervation to the face and should be distinguished from the facial nerve, which controls the muscles of facial expression. The trigeminal nerve also has a small
branchial motor root (see Figure 12.7), which travels with the mandibular division and is responsible for controlling the muscles of mastication and some other smaller muscles.

The trigeminal nerve exits the brainstem from the ventrolateral pons (see Figure 12.2A,C). It then enters a small fossa just posterior and inferolateral to the cavernous sinus called Meckel’s cave. The trigeminal ganglion, also known as the semilunar or gasserian ganglion, lies in Meckel’s cave and is the sensory ganglion of the trigeminal nerve (see Figure 12.7; Table 12.5). The ophthalmic division (V₁) travels through the inferior part of the cavernous sinus to exit the skull via the superior orbital fissure (see Figures 12.3A,C, 12.7A; Table 12.2; see also Figure 13.11). The maxillary division (V₂) exits via the foramen rotundum and the mandibular division (V₃) via the foramen ovale. A mnemonic for the exit points of these three branches is Single Room Occupancy, or SRO (for Superior, Rotundum, Ovale). The sensory territories of V₁, V₂, and V₃ are shown in Figure 12.7B. Recall that sensation to the occiput is conveyed by C₂ (see Case 8.2). The trigeminal nerve also provides touch and pain sensation for the nasal sinuses, inside of the nose, mouth, and anterior two-thirds of the tongue. In addition, pain sensation for the supratentorial dura mater is supplied by the trigeminal nerve, while the dura of the posterior fossa is innervated by CN X and upper cervical nerve roots.

Trigeminal Somatic Sensory Functions

The trigeminal nuclei (Figure 12.8 and Figure 12.9) receive general somatic sensory inputs from CN V and other cranial nerves (see Table 12.3). The main inputs are carried by CN V and, as we just mentioned, provide sensation for the face, mouth, anterior two-thirds of the tongue, nasal sinuses, and supratentorial dura. Smaller inputs from CN VII, IX, and X provide sensation for part of the external ear (see Figure 12.7B; Table 12.4). In addition, CN IX provides sensation to the middle ear, posterior one-third of the tongue, and pharynx. CN X additionally provides sensation for the infratentorial dura and probably also contributes to pharyngeal sensation (see Table 12.4). As we will now see, the trigeminal sensory systems are analogous to the posterior column–medial lemniscal system and anterolateral systems of the spinal cord (Table 12.6).

The trigeminal nuclear complex runs from the midbrain to the upper cervical spinal cord (see Figure 12.8) and consists of three nuclei: the mesencephalic, chief sensory, and spinal trigeminal nuclei (see Table 12.6). The chief (main or principal) trigeminal sensory nucleus and the spinal trigeminal nucleus provide sensory systems for the face and head that are analogous to the posterior columns and anterolateral systems, respectively (compare Figures 7.1, 12.8, and 12.9). The trigeminal lemniscus and spinothalamic tract are shown in Figure 12.10.

<table>
<thead>
<tr>
<th>TABLE 12.6</th>
<th>Analogous Trigeminal and Spinal Somatosensory Systems</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NUCLEUS</strong></td>
<td><strong>SENSORY MODALITIES</strong></td>
</tr>
<tr>
<td><strong>TRIGEMINAL SENSORY SYSTEMS</strong></td>
<td></td>
</tr>
<tr>
<td>Mesencephalic trigeminal nucleus</td>
<td>Proprioception</td>
</tr>
<tr>
<td>Chief trigeminal sensory nucleus</td>
<td>Fine touch; dental pressure</td>
</tr>
<tr>
<td>Spinal trigeminal nucleus</td>
<td>Crude touch; pain; temperature</td>
</tr>
<tr>
<td><strong>SPINAL SENSORY SYSTEMS</strong></td>
<td></td>
</tr>
<tr>
<td>Posterior column nuclei</td>
<td>Fine touch; proprioception</td>
</tr>
<tr>
<td>Dorsal horn</td>
<td>Crude touch; pain; temperature</td>
</tr>
</tbody>
</table>

*VPL, ventral posterior lateral nucleus; VPM, ventral posterior medial nucleus.
Chapter 12

7.2, and 12.8; see Table 12.6). The primary sensory neurons for these trigeminal nuclei lie mainly in the trigeminal ganglion (see Figure 12.8). Some also lie in the peripheral sensory ganglia of CN VII, IX, and X (see Table 12.5).

The chief trigeminal sensory nucleus, located in the lateral pons, receives synaptic inputs from large-diameter primary sensory neuron fibers mediating fine touch and dental pressure (see Figure 12.8; see also Figure 14.4B). This nucleus is similar in structure and function to the dorsal column nuclei (see Figures 7.1 and 14.5B). The trigeminal lemniscus crosses to the opposite side of the brainstem to ascend with the medial lemniscus toward the thalamus (see Figure 14.3). While the medial lemniscus travels to the ventral posterior lateral nucleus (VPL), the trigeminal lemniscus travels to the ventral posterior medial nucleus (VPM). From there, tertiary sensory neurons travel to the face area of the primary somatosensory cortex. A second, smaller pathway, called the dorsal trigeminal tract (or dorsal trigeminonothalamic tract), travels from the chief trigeminal sensory nucleus to the ipsilateral VPM, without crossing. This pathway appears to convey touch and pressure sensation from the oral cavity, including the teeth.
The spinal trigeminal nucleus is located in the lateral pons and medulla, extending down to the upper cervical spine (see Figure 12.8). Medium- and small-diameter primary sensory fibers conveying crude touch, pain, and temperature sensation enter the lateral pons with the trigeminal nerve and descend in the spinal trigeminal tract to synapse in the spinal trigeminal nucleus. Examine the spinal trigeminal nucleus and tract in the serial sections shown in Figures 14.4C and 14.5A–C. It should be clear from these sections that the spinal trigeminal nucleus is the rostral extension of the dorsal horn. Similarly, the spinal trigeminal tract is analogous to Lissauer’s tract (see Figures 6.4 and 7.2). Secondary sensory neurons from the spinal trigeminal nucleus cross the brainstem to ascend as the trigeminothalamic tract (or ventral trigeminothalamic tract). The trigeminothalamic tract is analogous to the spinothalamic tract (see Table 12.6), and the pathways travel together to the thalamus (see Figures 12.8 and 14.3). Trigeminothalamic tract fibers synapse in the thalamic ventral posterior medial nucleus (VPM), and tertiary sensory neurons then travel in the internal capsule to the primary somatosensory cortex. Like the anterolateral systems in the spinal cord, there are also pathways from the spinal trigeminal nucleus to intralaminar thalamic nuclei, the reticular formation, and other areas, to mediate the affective and arousal aspects of facial pain.

The spinal trigeminal tract and nucleus are somatotopically organized, with the mandibular division represented dorsally, the ophthalmic division represented ventrally, and the maxillary division in between (see Figure 12.9). In addition, concentric rings form an “onion skin”-like representation, with perioral areas represented more rostrally in the nucleus, and areas more removed from the mouth represented more caudally.

The mesencephalic trigeminal nucleus and tract run along the lateral edge of the periaqeductal gray matter of the midbrain (see Figure 14.3A,B) and mediate proprioception (see Table 12.6). The neurons of the mesencephalic trigeminal nucleus are the only case in which primary sensory neurons lie within the central nervous system instead of in peripheral ganglia (see Figure 12.8). The peripheral processes of these neurons convey proprioceptive input from the muscles of mastication and probably also from the tongue, and from the extraocular muscles. In the monosynaptic jaw jerk reflex (see KCC 12.4; neuroexam.com Video 39), processes of mesencephalic trigeminal neurons descend to the pons and synapse in the motor trigeminal nucleus (see Figure 12.7A). Ascending and descending fibers form the mesencephalic trigeminal tract (see Figure 12.8; see also Figure 14.3A,B). Other central pathways of the mesencephalic trigeminal nucleus are still under investigation.

### REVIEW EXERCISE

1. Cover the second column from the left in Table 12.6. For each nucleus in the left column, provide the sensory modalities served and the location of the nucleus (see Figure 12.8).

2. Which thalamic nucleus is most important for relaying somatosensory information from the face, and which is most important for relaying somatosensory information from the rest of the body to the cortex?
Trigeminal Branchial Motor Functions

The trigeminal motor nucleus mediates the branchial motor functions of the trigeminal nerve (see Figure 12.7). This nucleus is located in the upper-to-mid pons (see Figures 12.5 and 14.4B), near the level where the trigeminal nerve exits the brainstem. The branchial motor root of the trigeminal nerve runs inferomedial to the trigeminal ganglion along the floor of Meckel’s cave and then joins V3 to exit via the foramen ovale (see Figure 12.3A). It then supplies the muscles of mastication (see neuroexam.com Video 38), including the masseter, temporalis, and medial and lateral pterygoid muscles, as well as several smaller muscles, such as the tensor tympani, tensor veli palatini, mylohyoid, and anterior belly of the digastric. The upper motor neuron control reaching the trigeminal motor nucleus is predominantly bilateral, so unilateral lesions in the motor cortex or corticobulbar tract usually cause no deficit in jaw movement. Bilateral upper motor neuron lesions, however, can cause hyperreflexia manifested in a brisk jaw jerk reflex (see KCC 12.4).

KEY CLINICAL CONCEPT

12.2 TRIGEMINAL NERVE DISORDERS (CN V)

Disorders of the trigeminal nerve are relatively uncommon, except for trigeminal neuralgia (tic douloureux). In this condition, patients experience recurrent episodes of brief severe pain lasting from seconds to a few minutes, most often in the distribution of V2 or V3. Attacks usually begin after age 35. Painful episodes are often provoked by chewing, shaving, or touching a specific trigger point on the face. Neurologic exam, including facial sensation, is normal. The cause of trigeminal neuralgia in most cases is unknown. In some cases, compression of the trigeminal nerve by an aberrant vessel has been demonstrated, but the significance of this finding is uncertain. It is important to perform an MRI scan to exclude tumor or other lesions in the region of the trigeminal nerve as the cause. Trigeminal neuralgia can also occur in multiple sclerosis (see KCC 6.6), possibly caused by demyelination in the trigeminal nerve entry zone of the brainstem. Initial treatment of trigeminal neuralgia is with carbamazepine, and alternatives include oxcarbazepine, baclofen, lamotrigine, or pimozide. Refractory cases have been successfully treated with various procedures, including radiofrequency ablation of the Gasserian ganglion, gamma knife, CyberKnife (see KCC 16.4), or surgical microvascular decompression of the trigeminal nerve. Sensory loss in the distribution of the trigeminal nerve or its branches (see neuroexam.com Video 36) can be caused by trauma, metastatic disease—especially in isolated chin or jaw numbness, herpes zoster (see KCC 8.3), aneurysms of the petrous portion of the internal carotid artery (see Figures 4.16C and 12.3A), cavernous sinus or orbital apex disorders (see KCC 13.7), trigeminal or vestibular schwannoma (see KCC 12.5), or sphenoid wing meningioma (see KCC 5.8). Lesions of the trigeminal nuclei in the brainstem cause ipsilateral loss of facial sensation to pain and temperature because the primary sensory fibers do not cross before entering the nucleus (see Figure 12.8). Common causes include infarcts (see Chapter 14), demyelination, or other brainstem lesions. Often lesions of the trigeminal nucleus in the pons or medulla also involve the nearby spinothalamic tract (see Figures 7.2, 14.4C, and 14.5A,B). This combination of spinal trigeminal and spinothalamic tract involvement in lateral brainstem lesions leads to a well-recognized pattern with sensory loss to pain and temperature in the face ipsilateral to the lesion, but in the body contralateral to the lesion (see KCC 7.3; Figure 7.9B).
CN VII: Facial Nerve

<table>
<thead>
<tr>
<th>FUNCTIONAL CATEGORY</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Branchial motor</td>
<td>Muscles of facial expression, stapedius muscle, and part of digastric muscle</td>
</tr>
<tr>
<td>Parasympathetic</td>
<td>Parasympathetics to lacrimal glands, and to sublingual, submandibular, and all other salivary glands except parotid</td>
</tr>
<tr>
<td>Visceral sensory (special)</td>
<td>Taste from anterior two-thirds of tongue</td>
</tr>
<tr>
<td>General somatic sensory</td>
<td>Sensation from a small region near the external auditory meatus</td>
</tr>
</tbody>
</table>

The main function of the facial nerve is to control the muscles of facial expression (see neuroexam.com Video 40); however, it has several other important functions as well. The main nerve trunk carries the branchial motor fibers controlling facial expression, while a smaller branch called the nervus intermedius carries fibers for the parasympathetic (tears and salivation), visceral sensory (taste), and general somatosensory functions (Figure 12.10; see also Figures 12.6 and 12.14).

The facial nucleus is located in the branchial motor column, more caudally in the pons than the trigeminal motor nucleus (see Figure 12.5; see also Figure 14.4B,C). The fascicles of the facial nerve loop dorsally around the abducens nucleus, forming the facial colliculus on the floor of the fourth ventricle (see Figure 12.2B and Figure 12.11). The nerve then exits the brainstem ventrolaterally at the pontomedullary junction (see Figure 12.2A,C). Upper motor neuron control of the facial nucleus is discussed in KCC 12.3 (see Figure 12.13). Briefly, lesions in the cortex or corticobulbar tracts cause contralateral face weakness that spares the forehead, while lesions of the facial nucleus, nerve fascicles in the brainstem, or peripheral nerve cause ipsilateral weakness of the entire face.

The facial nerve exits the brainstem ventrolaterally at the pontomedullary junction, lateral to CN VI in a region called the cerebellopontine angle (see Figure 12.2A,C). It then traverses the subarachnoid space and enters the internal auditory meatus (see Figure 12.3A; see also Figure 4.13C) to travel in the auditory canal of the petrous temporal bone together with the vestibulocochlear nerve (see Figure 12.14). At the genu of the facial nerve, the nerve takes a turn posteriorly and inferiorty in the temporal bone to run in the facial canal, just medial to the middle ear (see Figures 12.10 and 12.14). The geniculate ganglion lies in the genu and contains primary sensory neurons for taste sensation in the anterior two-thirds of the tongue, and for general somatic sensation in a region near the external auditory meatus (see Table 12.5; Figure 12.7B). The main portion of the facial nerve exits the skull at the stylomastoid foramen (see Figures 12.3B and 12.10). It then passes through the parotid gland and divides into five major branchial motor branches to control the muscles of facial expression: the temporal, zygomatic, buccal, mandibular, and cervical branches (see Figure 12.10). Other smaller branchial motor branches innervate the stapedius (see Figures 12.10 and 12.15), occipitalis, posterior belly of the digastric, and stylohyoid muscles. The cranial nerves controlling the middle ear muscles can be recalled by the mnemonic Trigeminal for Tensor Tympani and Seventh for Stapedius. Both the tensor tympani and the stapedius dampen movements of the middle ear ossicles (see the section on CN VIII later in this chapter), providing feedback modulation of acoustic signal intensity.

The preganglionic parasympathetic fibers of the facial nerve originate in the superior salivatory nucleus (see Figure 12.10) and are carried by two small branches off the main trunk of the facial nerve. The greater petrosal nerve takes off at the genu of the facial nerve (see Figure 12.14) to reach the sphenopalatine
(pterygopalatine) ganglion, where postganglionic parasympathetic cells project to the lacrimal glands and nasal mucosa (see Figure 12.10). The chorda tympani leaves the facial nerve just before the stylomastoid foramen and travels back upward to traverse the middle ear cavity before exiting the skull at the petrotympanic fissure (see Figures 12.3B and 12.10), just medial and posterior to the temporomandibular joint. The chorda tympani then joins the lingual nerve (a branch of CN V₃) to reach the submandibular ganglion (also called the submaxillary ganglion), where postganglionic parasympathetics arise to supply the submandibular (submaxillary) and sublingual salivary glands as well as other minor salivary glands aside from the parotid. Note that the majority (~70%) of saliva production arises from the submandibular salivary glands.

The lingual nerve and chorda tympani also carry special visceral sensory fibers mediating taste sensation (see neuroexam.com Video 41) for the anterior two-thirds of the tongue (see Figure 12.10). The primary sensory taste fibers have their cell bodies in the geniculate ganglion (Figure 12.12; see also Figure 12.14 and Table 12.5). These cells synapse onto secondary sensory
**FIGURE 12.11** Axial Section of the Mid-to-Lower Pons, Showing the Facial Colliculus Fibers of the facial nerve (CN VII) loop around the abducens nucleus (CN VI) before exiting the brainstem. (Brainstem section modified from Martin JH. 1996. *Neuroanatomy: Text and Atlas*, 2nd Ed. McGraw-Hill, New York.)

**FIGURE 12.12** Central Taste Pathways

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neurons in the **rostral nucleus solitarius**, also known as the gustatory nucleus. The taste functions of the nucleus solitarius can be remembered since this nucleus looks like a delicious doughnut with cell bodies surrounding a myelinated center in cross sections (see Figure 14.5A). There are also taste inputs for the posterior tongue and pharynx that travel via CN IX and X to enter the rostral nucleus solitarius. Ascending projections continue via the **central tegmental tract** (see Figure 12.12; see also Figures 14.3 and 14.4) to reach tertiary sensory neurons in the **ventral posterior medial nucleus** (**VPM**) of the thalamus. Thalamic neurons from the VPM, in turn, project to the cortical taste area, which lies at the inferior margin of the postcentral gyrus adjacent to the tongue somatosensory area and extends into the fronto-parietal operculum and insula (see Figure 12.12). Taste pathways ascend ipsilaterally and likely project to bilateral thalamus and cortex, but the laterality of cortical taste pathways in humans is still under investigation.

Finally, a small branch of the facial nerve provides **general somatic sensation** for a region near the external auditory meatus that lies adjacent to similar regions supplied by CN IX and X (see Figure 12.7B). The somatosensory fibers for CN V, VII, IX, and X all synapse in the trigeminal nuclei (see Figure 12.5; Table 12.3).

**KEY CLINICAL CONCEPT**

**12.3 FACIAL NERVE LESIONS (CN VII)**

As we discussed briefly in KCC 6.3, it is clinically important to distinguish between facial weakness caused by upper motor neuron lesions and facial weakness caused by lower motor neuron lesions. Upper motor neurons in the face area of the primary motor cortex control lower motor neurons in the contralateral facial nucleus of the pons (Figure 12.13). In addition, for the superior portions of the face, projections descend from the ipsilateral motor cortex as well as from the contralateral motor cortex. Thus, the lower motor neurons supplying the forehead and part of the orbicularis oculi receive upper motor neuron inputs from bilateral motor cortices. As a result, unilateral **upper motor neuron lesions** tend to spare the forehead and cause only mild contralateral orbicularis oculi weakness resulting in a slightly widened palpebral fissure, or inability to fully bury the eyelash on forced eye closure. In upper motor neuron lesions the weakness affects mainly the inferior portions of the contralateral face (see Figure 12.13, Lesion A). **Lower motor neuron lesions**, in contrast, affect the entire half of the face and do not spare the forehead (see Figure 12.13, Lesion B). Additional clues sometimes present in upper motor neuron–type weakness include neighborhood effects such as hand or arm weakness, sensory loss, aphasia, or dysarthria, none of which are present in lower motor neuron lesions. The connections shown in Figure 12.13 are somewhat oversimplified; in reality, the upper motor neuron corticobulbar fibers controlling the facial nucleus project mainly to pontine interneurons that project, in turn, to lower motor neurons in the facial nucleus.

The most common facial nerve disorder by far is **Bell’s palsy**, in which all divisions of the facial nerve are impaired within a few hours or days and then gradually recover. The cause is unknown, although viral or inflammatory mechanisms have been suggested. The most striking feature is unilateral facial weakness of the lower motor neuron type, which can be mild but is often severe (see Figure 12.13, Lesion B). Diagnosis is based on clinical history and exam (see neuroexam.com Videos 40 and 41). Patients often initially complain of some retroauricular pain, likely caused by involvement of the general somatosensory component of CN VII (see Figure 12.7B). Hyperacusis can occur because of stapedius muscle weakness (see Figures 12.10 and 12.15). In addi-
tion, patients may suffer from “dry eye,” resulting from decreased lacrimation with parasympathetic involvement (see Figure 12.10). Neurologic examination is notable for unilateral lower motor neuron–type facial weakness, sometimes associated with loss of taste on the ipsilateral tongue (test with mustard or sugar applied with a cotton swab; see neuroexam.com Video 41). The remainder of the exam should be normal in Bell’s palsy. The presence of hand weakness, sensory loss, dysarthria, or aphasia suggests an upper motor neuron lesion. In clinically typical cases, imaging studies are usually normal, however, most practitioners will order an MRI scan to exclude a structural lesion and blood studies including a blood count, glucose, and Lyme titer.

Treatment of Bell’s palsy has been controversial, but recent evidence suggests that a 10-day course of oral steroids started soon after onset improves chances for full recovery. The possible role of antiviral agents in treating Bell’s palsy remains uncertain. Incomplete eye closure and decreased tearing can cause corneal ulcerations. Therefore, patients should be given lubricating eye-drops and instructions to tape the eye shut at night. About 80% of patients recover fully from Bell’s palsy within 3 weeks, although some are left with variable degrees of residual weakness. During recovery, regenerating facial nerve
fibers sometimes reach the wrong target. For example, aberrant regeneration of parasympathetic fibers (see Figure 12.6) can result in the phenomenon of “crocodile tears,” in which patients experience lacrimation instead of salivation when they see food. Aberrant regeneration of different motor branches of the facial nerve sometimes results in synkinesis, meaning abnormal movement together. For example, if the patient is asked to close one eye, the ipsilateral platysma muscle may contract slightly, along with the orbicularis oculi.

In cases of bilateral lower motor neuron–type facial weakness, or if a patient experiences a second episode, a more thorough investigation is warranted. This should include an MRI scan with contrast to look for tumor or other infiltrative disorders, lumbar puncture (see KCC 5.10), and tests for Lyme disease, sarcoidosis, and HIV. In addition to the causes already mentioned, facial nerve injury can occur in head trauma, particularly with fractures of the petrous temporal bone. Facial weakness caused by upper motor neuron lesions is discussed in KCC 6.3. In addition, brainstem lesions can occasionally involve the facial nucleus or exiting nerve fascicles (see Figure 12.11; see also KCC 14.3).

**KEY CLINICAL CONCEPT**

### 12.4 CORNEAL REFLEX AND JAW JERK REFLEX (CN V, VII)

The corneal reflex is elicited by gentle stroking of each cornea with a cotton swab; the response is eye closure (see neuroexam.com Video 37). This reflex is mediated by both monosynaptic and polysynaptic pathways. The afferent limb is conveyed by the ophthalmic division of the trigeminal nerve to the chief sensory and spinal trigeminal nuclei. The efferent limb is then carried by the facial nerve to reach the orbicularis oculi muscles causing eye closure. A lesion of the trigeminal sensory pathways, the facial nerve, or their connections causes a decreased corneal reflex in the ipsilateral eye. The corneal reflex is also modulated by inputs from higher centers. Therefore, lesions of sensorimotor cortex and its connections can cause a diminished corneal reflex in the eye contralateral to the lesion.

Since an eye blink response can also be elicited by an object moving toward the eye, when eliciting the corneal response the examiner must take care to ensure that the blink has been elicited by touch rather than a sudden movement toward the eye. In blink to threat (see neuroexam.com Video 28) the afferent limb of the reflex is carried by the optic nerve (CN II), while in the corneal reflex, the afferent limb is carried by the trigeminal nerve (CN V).

The jaw jerk reflex is elicited by tapping on the chin with the mouth slightly open; the jaw jerks forward in response. The monosynaptic pathway for this reflex consists of primary sensory neurons in the mesencephalic trigeminal nucleus (see Figure 12.8), which send axons to the pons to synapse in the motor trigeminal nucleus. In normal individuals the jaw jerk reflex is minimal or absent (see neuroexam.com Video 39). In bilateral upper motor neuron lesions, such as amyotrophic lateral sclerosis (see KCC 6.7) or diffuse white matter disease, the jaw jerk reflex may be brisk.

### CN VIII: Vestibulocochlear Nerve

<table>
<thead>
<tr>
<th>FUNCTIONAL CATEGORY</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Special somatic sensory</td>
<td>Hearing and vestibular sensation</td>
</tr>
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</table>

This nerve carries the special somatic sensory functions of hearing and vestibular sense from the structures of the inner ear. The vestibulocochlear nerve exits the brainstem at the pontomedullary junction just lateral to the facial nerve, in a region called the cerebellopontine angle (see Figure 12.2A,C). It then tra-
verses the subarachnoid space to enter the **internal auditory meatus** (see Figure 12.3A) together with the facial nerve and travels in the **auditory canal** of the petrous temporal bone to reach the cochlea and vestibular organs (Figure 12.14; see also Figure 4.13C). In the subsections that follow we will discuss the auditory and vestibular functions of CN VIII, in turn.

**Auditory Pathways**

Sound waves are transmitted by the **tympanic membrane** and amplified by the middle ear ossicles—the **malleus**, **incus**, and **stapes**—to reach the **oval window** (Figure 12.15). The movements of the malleus are dampened by the tensor tympani muscle, and movements of the stapes are dampened by the stapedius in response to loud sounds. From the oval window, vibrations reach the inner ear structures. The **inner ear**, or **labyrinth**, consists of the **cochlea**, **vestibule**, and **semicircular canals** (see Figure 12.15). The labyrinth is composed of a **bony labyrinth**, which is lined with compact bone and contains the **membranous labyrinth**. The bony labyrinth is filled with fluid called **perilymph**, within which the structures of the membranous labyrinth are suspended. Interestingly, perilymph communicates with the subarachnoid space through a small perilymphatic duct (not shown). The membranous labyrinth, in turn, is filled with a fluid of slightly different ionic composition, called **endolymph**. The membranous labyrinth includes the **cochlear duct**, **utricle**, **saccule**, and **semicircular canals** (see Figure 12.15).

Acoustic vibrations from the oval window reach the **scala vestibuli** and are transmitted along the snail-shaped **cochlea** to the end, where it joins the **scala tympani**, and the pressure waves are ultimately relieved at the **round window** back in the wall of the middle ear. The vibrations also reach the **cochlear duct** (scala media) (see Figure 12.15, lower right inset), where mechanoreceptor cilia on the **hair cells** are activated by movement of the **basilar membrane** relative to the stiff **tectorial membrane**. The hair cells form excitatory synapses onto the terminals of primary sensory neurons. These bipolar sensory neurons have their cell bodies in the **spiral ganglion**, located along the central rim of the cochlea, and send their axons into the cochlear
Sound waves enter the external auditory meatus, are transmitted mechanically by the middle ear to the cochlea, and are transduced by hair cells to neural signals carried centrally by the cochlear nerve. The ampullae of the semicircular canals detect angular acceleration, and the maculae of the otolith organs (utricle and saccule) detect linear acceleration and head tilt and transmit this information to the vestibular nerves. (Insets from Rosenzweig MR, Breedlove SM, Leiman AL. 2002. *Biological Psychology*, 3rd Ed. Sinauer, Sunderland, MA.)

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nerve (see Figures 12.14 and 12.15). The hair cells of the cochlea, together with their supporting cells, are called the organ of Corti. There is a tonotopic representation determined by structural width and stiffness along the length of the organ of Corti such that higher-frequency sounds activate hair cells near the oval window, while lower-frequency sounds activate hair cells near the apex of the cochlea (see Figure 12.15).

Let’s follow the pathways for hearing centrally, from the cochlear nuclei to the primary auditory cortex (Figure 12.16 and Figure 12.17). Auditory information throughout these pathways is tonotopically organized. Primary sensory neurons in the spiral ganglion send their axons in the cochlear division of CN VIII to reach the dorsal and ventral cochlear nuclei, which are wrapped around the lateral aspect of the inferior cerebellar peduncle at the pontomedullary junction (see Figures 12.16 and 12.17C). The hearing pathways then ascend through
the brainstem bilaterally through a series of relays to reach the inferior colliculi, medial geniculate nuclei, and, ultimately, the auditory cortex. Because auditory information from each ear ascends bilaterally in the brainstem, with decussations occurring at multiple levels, unilateral hearing loss is not seen in lesions in the central nervous system proximal to the cochlear nuclei.

Fibers from the dorsal cochlear nucleus pass dorsal to the inferior cerebellar peduncle, cross the pontine tegmentum, and ascend in the contralateral lateral lemniscus (see Figures 12.16 and 12.17A,B). The lateral lemniscus is an important ascending auditory pathway in the pons and lower midbrain that terminates in the inferior colliculus. Many fibers of the ventral cochlear nucleus pass ventral to the inferior cerebellar peduncle to synapse bilaterally in the superior olivary nuclear complex of the pons (see Figures 12.16 and 12.17B). The superior olivary nuclei appear to function in localizing sounds horizontally in space. Crossing auditory fibers at this level form a white matter structure called the trapezoid body (see Figures 12.16 and 12.17B). The trapezoid body is traversed at right angles by the medial lemniscus (see Figure 14.4C).

From the superior olivary nuclear complex, fibers ascend bilaterally in the lateral lemniscus to reach the inferior colliculi of the midbrain (see Figures 12.16 and 12.17A). Decussating fibers at the level of the inferior colliculi pass both dorsal and ventral to the cerebral aqueduct. From the inferior colliculi, fibers ascend via the brachium of the inferior colliculi to the medial geniculate nuclei of the thalamus, which are located just lateral to the superior colliculi of the midbrain (see Figures 11.6, 12.16, and 14.3A). From this thalamic relay, information continues in the auditory radiations (see Figure 6.9B) to the primary auditory cortex. The primary auditory cortex (Brodmann’s area 41) lies on Heschl’s transverse gyri. We can see these straight, fingerlike gyri in brain specimens by opening the Sylvian fissure and looking at the superior surface of the temporal lobe, just medial to the superior temporal gyrus (see Figure 12.16; see also Figure 4.15D). The nearby areas of cortex in the temporal and parietal lobes are auditory association cortex, including Wernicke’s area, which will be discussed in Chapter 19. In addition to the nuclei already mentioned, there are several smaller nuclei in the hearing pathway, including the nuclei of the trapezoid body and the nuclei of the lateral lemniscus.

As noted already, lesions in the central nervous system proximal to the cochlear nuclei do not cause unilateral hearing loss because auditory information crosses bilater-
ally at multiple points in the brainstem. However, auditory information ascending through the brainstem and thalamus to the auditory cortex does contain a relatively greater contribution from the contralateral ear. In auditory seizures, caused by abnormal electrical discharges in the auditory cortex, patients often perceive a tone or roaring sound like an airplane or a train coming from the side opposite the auditory cortex involved. Bilateral damage to the auditory cortex causes cortical deafness (see KCC 19.7).

Efferent feedback pathways from the brainstem to the cochlea in the vestibulocochlear nerve modulate the sensitivity of the hair cells in response to sounds of varying intensities. Similar pathways exist for modulation of vestibular hair cells. Because of these small efferent pathways, some might not consider CN VIII a purely sensory nerve. In addition, reflex pathways from the ventral cochlear nuclei reach the facial and trigeminal motor nuclei to contract the stapedius and tensor tympani muscles. These muscles dampen the response of the middle ear to loud sounds.

**Vestibular Pathways**

The **vestibular nuclei** are important for adjustment of posture, muscle tone, and eye position in response to movements of the head in space. Not surprisingly, therefore, the vestibular nuclei have intimate connections with the cerebellum, and with the brainstem motor and extraocular systems. In addition, an ascending pathway through the thalamus to the cortex provides an awareness of head position that is integrated with visual and tactile spatial information in the parietal association cortex.

The **semicircular canals** (see Figures 12.14 and 12.15) detect angular acceleration around three orthogonal axes. The spatial orientation of the three semicircular canals can be remembered by imagining the arms of a bodybuilder in three poses (Figure 12.18).

**REVIEW EXERCISE**

Follow the auditory pathways from the cochlear nerve to the auditory cortex in Figures 12.16 and 12.17.

**FIGURE 12.18 Orientation of Semicircular Canals** Images of a bodybuilder in three poses can be helpful to remember the spatial orientations of the semicircular canals.
Rotation of the head around any of these axes causes movement of endolymph through the **ampullae** (see Figure 12.15, upper right inset). This flow deforms the gelatinous **cupula**, within which the mechanoreceptor cilia of hair cells are embedded. The hair cells are located in a ridge within each ampulla, called the **crista ampullaris**. The hair cells activate terminals of bipolar primary sensory neurons that have their cell bodies in the **vestibular ganglia of Scarpa** and send axons into the vestibular nerves (see Figure 12.15). The utricle and saccule contain structures called **maculae** that resemble the cristae ampullaris, but rather than angular acceleration, they detect linear acceleration and head tilt (see Figure 12.15, lower left inset). The macula consists of calcified crystals called **otoliths** sitting on a gelatinous layer within which mechanoreceptor hair cells are embedded. Gravity or other causes of linear acceleration pull on the crystals and activate these hair cells. The **superior vestibular ganglion** receives input from the utricle, anterior saccule, and anterior and lateral semicircular canals. The **inferior vestibular ganglion** receives input from the posterior saccule and posterior semicircular canal.

Primary sensory neurons in the vestibular ganglia (see Figure 12.15) convey information about angular and linear acceleration from the semicircular canals and otolith organs, respectively, through the vestibular division of CN VIII to the vestibular nuclei. There are **four vestibular nuclei** on each side of the brainstem, lying on the lateral floor of the fourth ventricle in the pons and rostral medulla (Figure 12.19). These nuclei can also be seen in the...
myelin sections in Figures 14.4C and 14.5A. The lateral vestibular nucleus gives rise to the lateral vestibulospinal tract, which, despite its name, is part of the medial descending motor systems (see Table 6.3). The lateral vestibulospinal tract extends throughout the length of the spinal cord and is important in maintaining balance and extensor tone (see Figure 6.11D). The medial vestibulospinal tract arises from the medial vestibular nucleus, with additional contributions primarily from the inferior vestibular nucleus. The medial vestibulospinal tract is also a medial descending motor system, but it extends only to the cervical spine and is important in controlling neck and head position. The medial vestibular nucleus is the largest of the vestibular nuclei. The inferior vestibular nucleus (also called Deiters’ nucleus) is relatively easy to identify on myelin-stained sections because fibers of the lateral vestibular nucleus traverse the inferior vestibular nucleus as they descend toward the spinal cord (see Figure 12.19), giving the inferior vestibular nucleus a characteristic “checkerboard” appearance (see Figure 14.5A).

The medial longitudinal fasciculus (MLF) is an important pathway that connects the nuclei involved in eye movements to each other and to the vestibular nuclei (see Figure 12.19). The MLF can be identified in the sections in Figures 14.3–14.5 as a heavily myelinated tract running near the midline on each side, just under the oculomotor and trochlear nuclei in the midbrain, and just under the floor of the fourth ventricle in the midline of the pons. Fibers arising from the medial vestibular nucleus, with additional contributions mainly from the superior vestibular nucleus, ascend in the MLF to the oculomotor, trochlear, and abducens nuclei. This pathway mediates the vestibulo-ocular reflex, in which eye movements are adjusted for changes in head position (see neuroexam.com Video 35). The function of the MLF in interconnecting the abducens and oculomotor nuclei is discussed in Chapter 13. In another commonly used nomenclature, what we have called the MLF is referred to as the ascending MLF, and the medial vestibulospinal tract is referred to as the descending MLF (see Figure 12.19).

The vestibular nuclei have numerous important reciprocal connections with the cerebellum. As we will discuss in Chapter 15, vestibular-cerebellar connections occur mainly with the flocculonodular lobes and cerebellar vermis. These regions of the cerebellum are often called the vestibulocerebellum. A small number of primary vestibular sensory neurons bypass the vestibular nuclei and project directly to the vestibulocerebellum.

Ascending pathways from the vestibular nuclei relay in the ventral posterior nucleus of the thalamus to reach the cerebral cortex. These pathways are still being investigated in humans; however, one important cortical region for vestibular sensation appears to lie in the parietal association cortex, possibly in Brodmann’s area 5, or in the lateral temporoparietal junction and posterior insula.

**KEY CLINICAL CONCEPT**

**12.5 HEARING LOSS (CN VIII)**

Unilateral hearing loss can be caused by disorders of the external auditory canal, middle ear, cochlea, eighth nerve, or cochlear nuclei (see Figures 12.14 and 12.15). As we have emphasized already, once the auditory pathways enter the brainstem, information immediately crosses bilaterally at multiple levels (see Figure 12.16). Therefore, unilateral hearing loss is not caused by lesions in the central nervous system proximal to the cochlear nuclei. (Disturbances of higher-order auditory processing and auditory hallucinations are described in KCC 19.7 and 19.13.)
Impaired hearing is usually divided into **conductive hearing loss**, caused by abnormalities of the external auditory canal or middle ear, and **sensorineural hearing loss**, usually caused by disorders of the cochlea or eighth nerve. When evaluating a patient for hearing loss, the practitioner should first examine the ears with an otoscope. Hearing can be tested with sounds of different frequencies, such as finger rubbing, whispering, or a ticking watch (see neuroexam.com Video 42). Conductive and sensorineural hearing loss can often be distinguished with a simple 256 Hz or 512 Hz tuning fork (some argue that only 512 Hz or higher is useful for testing hearing). In the **Rinne test**, air conduction is compared to bone conduction for each ear. We measure **air conduction** by holding a vibrating tuning fork just outside each ear, and **bone conduction** by placing a tuning fork handle on each mastoid process (see neuroexam.com Video 42). Normal individuals hear the tone better by air conduction. In conductive hearing loss, bone conduction is greater than air conduction because bone conduction bypasses problems in the external or middle ear. In sensorineural hearing loss, air conduction is greater than bone conduction in both ears (as in normal hearing); however, hearing is decreased in the affected ear. In the **Weber test** the tuning fork is placed on the vertex of the skull in the midline, and the patient is asked to report the side where the tone sounds louder (see neuroexam.com Video 42). Normally, the tone sounds equal on both sides. In sensorineural hearing loss, the tone is quieter on the affected side. In conductive hearing loss, the tone is louder on the affected side, because compensatory neural mechanisms or mechanical factors increase the perceived volume on the side of conduction problem. You can verify this on yourself by producing temporary unilateral conductive hearing loss by closing each ear alternately. If you then hum, the tone should be louder on the occluded side.

Other tests that can help localize the cause of hearing loss include audiology and brainstem auditory evoked potentials. An MRI scan with fine cuts through the auditory canal should be performed when disorders of the eighth nerve are suspected. Common causes of conductive hearing loss include cerumen in the external auditory canal, otitis, tympanic membrane perforation, and sclerosis of the middle ear ossicles. Causes of sensorineural hearing loss include exposure to loud sounds, meningitis, ototoxic drugs, head trauma, viral infections, aging, Meniere’s disease (see KCC 12.6), cerebellopontine angle tumors, and, rarely, internal auditory artery infarct (see KCC 14.3).

Cerebellopontine angle tumors include acoustic neuroma (vestibular schwannoma), meningioma, cerebellar astrocytoma, epidermoid, glomus jugulare, and metastases. The most common tumor by far in this location is **acoustic neuroma**, accounting for about 9% of intracranial neoplasms (see Table 5.6). Mean age of onset is 50 years, and the tumor is nearly always unilateral. The exception is in neurofibromatosis type 2, in which the tumors are bilateral and usually occur by adolescence or early adulthood. This slow-growing tumor develops at the transitional zone between Schwann cells and oligodendrocytes, which occurs at the point where CN VIII enters the internal auditory meatus (see Figures 12.3A and 12.14). The term “acoustic neuroma” is a misnomer because the tumor is actually a **schwannoma**, not a neuroma, and it nearly always arises from the vestibular, not acoustic, division of the eighth nerve. Initially the tumor grows within the bony auditory canal, but then it expands into the cerebellopontine angle (see Figure 12.2A,C). Common early symptoms are unilateral hearing loss, **tinnitus** (ringing in the ear), and unsteadiness. The next cranial nerve to be affected is usually the nearby trigeminal nerve, with facial pain and sensory loss. Often the first sign of trigeminal involvement is a subtle decrease in the corneal re-
flex (see KCC 12.4). Interestingly, although the vestibular and facial nerves are compressed within the auditory canal, true vertigo is not usually a prominent symptom (although some unsteadiness is common), and the facial nerve does not usually become involved until the tumor is quite large. Eventually there is facial weakness, sometimes with decreased taste sensation on the side of the tumor.

With large tumors, cerebellar and corticospinal pathways are compressed, causing ipsilateral ataxia and contralateral hemiparesis. Impairment of swallowing and the gag reflex (CN IX and X) and unilateral impaired eye movements (CN III and VI) occur only in very large tumors. Ultimately, if left untreated, the tumor will compress the fourth ventricle, causing CSF outflow obstruction, hydrocephalus, herniation, and death. With appropriate clinical evaluation and MRI scanning, acoustic nerve tumors can be detected at an early stage, when they still lie entirely within the auditory canal. Treatment has traditionally been by surgical excision, but more recently there is a shift towards stereotactic radiosurgery (see KCC 16.4) with gamma knife or CyberKnife. Some smaller tumors may be monitored by MRI, especially in older patients, and factors such as age, position, size, hearing and patient preference help determine the choice between radiosurgery vs. surgical excision. Conventional surgery requires a posterior fossa approach, often involving collaboration between a neurosurgeon and otolaryngologist. Surgeons strive to spare facial nerve function during the procedure, and with smaller tumors some hearing may even be spared in the affected ear. Schwannomas can occur on other cranial nerves, as well as on spinal nerve roots, causing radiculopathy or spinal cord compression. 

**KEY CLINICAL CONCEPT**

**12.6 DIZZINESS AND VERTIGO (CN VIII)**

“Dizziness” is a vague term used by patients to describe many different abnormal sensations. In taking the history, the examiner should clarify whether the patient is referring to true vertigo, meaning a spinning sensation of movement, or one of the other meanings of dizziness. Other uses of “dizziness” include light-headedness or faintness, nausea, and unsteadiness on one’s feet. True vertigo is more suggestive of vestibular disease than these other symptoms are. However, the situation is complicated by the fact that the other sensations listed here often accompany vertigo, and in some cases they may be the only presenting symptoms of vestibular disease.

Vertigo can be caused by lesions anywhere in the vestibular pathway, from labyrinth, to vestibular nerve, to vestibular nuclei and cerebellum, to parietal cortex. Most cases of vertigo are caused by peripheral disorders involving the inner ear, with central disorders of the brainstem or cerebellum being less common. It is essential to distinguish these possibilities because some central causes of vertigo, such as incipient brainstem stroke or posterior fossa hemorrhage, require emergency treatment to prevent serious sequelae. In taking the history of a patient with vertigo, it is therefore crucial to ask whether any other symptoms suggestive of posterior fossa disease, such as diplopia, other visual changes, somatosensory changes, weakness, dysarthria, incoordination, or impaired consciousness, are present (see Table 14.6). Patients with any of these abnormalities accompanying vertigo should be considered to have posterior fossa disease until proven otherwise and should be treated on an urgent basis. The general physical exam should always include orthostatic measure-
ments of blood pressure and pulse in the supine and sitting or standing positions. Normally the systolic blood pressure drops by only about 10 millimeters of mercury, and the pulse increases by about 10 beats per minute when measured a few minutes after going from supine to a seated position with the legs dangling. Substantially greater changes suggest that the patient’s symptoms may be caused by hypovolemia, antihypertensive medications, or cardiovascular or autonomic disorders, rather than by a vestibular lesion. In addition, during the general exam the tympanic membranes should be examined with an otoscope. A careful neurologic exam should be done to detect any abnormalities that may suggest a central cause for the vertigo.

Dix–Hallpike (or Nylén– Bárány) positional testing is a useful part of the exam that can help distinguish peripheral from central causes of vertigo (see neuroexam.com Video 43). The patient sits on the bed or examining table. The examiner then supports the head of the patient as the patient lies back with the head turned so that one ear is down and the head extends over the edge of the table. This maneuver should be done rapidly but gently. The patient is asked to keep their eyes open and report any sensations of vertigo, while the examiner looks for nystagmus. This change of position causes maximal stimulation of the posterior semicircular canal of the ear that is down (the anterior semicircular canal of the ear that is up is probably also stimulated) (see Figure 12.18). The maneuver is then repeated with the other ear down.

With peripheral lesions affecting the inner ear there is usually a delay of 2 to 5 seconds before the onset of nystagmus and vertigo (Table 12.7). The nystagmus is horizontal or rotatory and does not change directions while the patient remains in the same position. Nystagmus and vertigo then fade away within about 30 seconds. If the same maneuver is repeated, there is often adaptation (also called habituation or fatiguing), so that the nystagmus and vertigo are briefer and less intense each time. In contrast, with central lesions the nystagmus and vertigo may begin immediately, and there tends to be no adaptation (see Table 12.7). Horizontal or rotatory nystagmus can also be seen with central lesions. However, vertical nystagmus, nystagmus that changes directions while remaining in the same position, or prominent nystagmus in the absence of vertigo is seen only in central, and not in peripheral, lesions.

Let’s briefly review a few specific peripheral and central causes of vertigo. Benign paroxysmal positional vertigo is possibly the most common cause of true vertigo. Patients experience brief episodes of vertigo lasting for a few seconds and occurring with change of position. When the symptom first occurs, the patient may be dizzy for several hours. However, after the first episode it is usually brief and occurs only with change of position. In some cases, the vertigo may be so intense that patients cannot walk. The proposed

<table>
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<tr>
<th>TYPE OF LESION</th>
<th>ONSET OF NYSTAGMUS</th>
<th>ADAPTATION (HABITUATION)</th>
<th>CHARACTERISTICS OF NYSTAGMUS AND VERTIGO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral (inner ear)</td>
<td>Delayed</td>
<td>Yes</td>
<td>Horizontal or rotatory, not vertical; does not change directions; prominent nystagmus only if vertigo is present as well.</td>
</tr>
<tr>
<td>Central (brainstem or cerebellum)</td>
<td>Immediate or delayed</td>
<td>No</td>
<td>Horizontal, rotatory, or vertical; may change directions; prominent nystagmus may occur in the absence of vertigo.</td>
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mechanism for this disorder is the presence of pieces of otolithic debris called otoconia in the semicircular canals (especially the posterior canal) that push against the cupula (see Figure 12.15, inset). Symptoms occur especially when the patient attempts to sleep and lies with the affected ear down, or if the patient turns to the affected side; however, if the patient remains still, the dizziness typically abates. Turning away from the affected ear or sitting up may also provoke symptoms. Treatment by **canalith repositioning maneuvers** to dislodge otolithic debris (Epley maneuver, or Semont liberatory maneuver) is beneficial in most patients. Symptoms may also be improved by adaptation exercises including the Brandt–Daroff procedure (patient sits on edge of bed, lies down sideways with the left ear down until vertigo subsides, and then repeats this ten times on each side) or other forms of vestibular rehabilitation therapy.

Viral infections or idiopathic inflammation of the vestibular ganglia or nerve may cause **vestibular neuritis**, a monophasic illness resulting in several days of intense vertigo and sometimes a feeling of unsteadiness that can last from weeks to months. In **Meniere’s disease**, patients have recurrent episodes of vertigo, accompanied by fluctuating and sometimes stepwise, progressive hearing loss and tinnitus. Patients with Meniere’s disease also often complain of a full feeling in the ear. The etiology is thought to be excess fluid and pressure in the endolymphatic system (see Figures 12.14 and 12.15). Meniere’s disease is most frequently treated with salt restriction and diuretics, although there have been no controlled studies of those therapies. There are multiple surgical procedures that have been effective in some patients; these include vestibular nerve section, labrinthectomy, endolymphatic saculotomy (decompression), and transtympanic gentamycin (to cause permanent loss of vestibular function on the affected side). **Autoimmune inner ear disease**, another important cause of vertigo, can produce symptoms resembling Meniere’s disease. **Acoustic neuroma** (vestibular schwannoma) is another cause of hearing loss and tinnitus that can be associated with vertigo (see KCC 12.5). However, unlike Meniere’s disease, patients with acoustic neuroma often complain of unsteadiness rather than true vertigo, and they do not usually have discrete episodes.

Common central causes of vertigo include **vertebrobasilar ischemia or infarct**. Involvement of the vestibular nuclei or cerebellum causes vertigo, often with other symptoms and signs of vertebrobasilar disease (see KCC 14.3; Table 14.6). It is essential to recognize this entity so that treatment is not delayed. Similarly, a small **hemorrhage** in the cerebellum or, rarely, in the brainstem may initially present mainly with vertigo and should be treated as soon as possible to prevent catastrophe. Cerebellar hemorrhage that presents initially with some nausea and dizziness, only to rebleed a few hours later, has been called “fatal gastroenteritis.” **Encephalitis**, **tumors**, or **demyelination** in the posterior fossa can cause vertigo. In addition, numerous **drugs and toxins**, including alcohol and anticonvulsant medications, cause dysfunction of the vestibular nuclei and cerebellum, producing vertigo along with other symptoms. Ototoxic drugs such as gentamicin cause **bilateral vestibular dysfunction**, which is why they cause unsteadiness of gait and oscillopsia (perception of oscillating vision) rather than true spinning vertigo. Anemia and thyroid disorders can also produce dizziness and should be tested for in all patients complaining of vertigo where etiology is unclear. Other disorders occasionally associated with vertigo include atypical migraines, Lyme disease, and syphilis. Finally, epileptic seizures (see KCC 18.2) are uncommon as a cause of vertigo without other symptoms; however, patients with seizures involving the parietal regions responsible for motion perception may report vertigo as one manifestation of their seizures.
CN IX: Glossopharyngeal Nerve

<table>
<thead>
<tr>
<th>FUNCTIONAL CATEGORY</th>
<th>FUNCTION</th>
</tr>
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<tbody>
<tr>
<td>Branchial motor</td>
<td>Stylopharyngeus muscle</td>
</tr>
<tr>
<td>Parasympathetic</td>
<td>Parasympathetics to parotid gland</td>
</tr>
<tr>
<td>General somatic sensory</td>
<td>Sensation from middle ear, region near the external auditory meatus, pharynx, and posterior one-third of tongue</td>
</tr>
<tr>
<td>Visceral sensory (special)</td>
<td>Taste from posterior one-third of tongue</td>
</tr>
<tr>
<td>Visceral sensory (general)</td>
<td>Chemoreceptors and baroreceptors of carotid body</td>
</tr>
</tbody>
</table>

The glossopharyngeal nerve was named for its role in sensation for the posterior tongue and pharynx; however, it has additional functions as well. It exits the brainstem as several rootlets along the upper ventrolateral medulla, just below the pontomedullary junction and just below CN VIII, between the inferior olive and the inferior cerebellar peduncle (see Figure 12.2A,C). The nerve traverses the subarachnoid space to exit the skull via the jugular foramen (see Figure 12.3A,B; Table 12.2).

The branchial motor portion of the nerve supplies one muscle, the stylopharyngeus (Figure 12.20), which elevates the pharynx during talking and swallowing and contributes (with CN X) to the gag reflex. There is evidence that the glossopharyngeal nerve may provide some innervation to other pharyngeal muscles; however, most pharyngeal muscles are supplied primarily by the vagus (see the next section). The branchial motor component of CN IX arises from the nucleus ambiguus in the medulla (see Figure 12.20). “Ambiguus” is Latin for “ambiguous,” and this name can be remembered because the nucleus is difficult to discern on conventional stained sections (see Figure 14.5A,B). Parasympathetic preganglionic fibers in the glossopharyngeal nerve arise from the inferior salivatory nucleus in the pons (see Figure 12.20). These parasympathetic fibers leave the glossopharyngeal nerve via the tympanic nerve and then join the lesser petrosal nerve to synapse in the otic ganglion, providing postganglionic parasympathetics to the parotid gland.

The general visceral sensory portion of the glossopharyngeal nerve conveys inputs from baroreceptors and chemoreceptors in the carotid body. These afferents travel to the caudal nucleus solitarius of the medulla, also known as the cardiorespiratory nucleus (see Figure 12.20). Glossopharyngeal special visceral sensation mediates taste for the posterior one-third of the tongue, which reaches the rostral nucleus solitarius, or gustatory nucleus (see Figures 12.5, 12.12, and 12.20). General somatic sensory functions of CN IX are the sensation of touch, pain, and temperature from the posterior one-third of the tongue, pharynx, middle ear, and a region near the external auditory meatus (see Figure 12.7B). The glossopharyngeal nerve has two sensory ganglia located within or just below the jugular foramen (see Table 12.5). General and special visceral sensation are conveyed by primary sensory neurons in the inferior (petrosal) glossopharyngeal ganglion. General somatic sensation is conveyed by primary sensory neurons in both the inferior and superior (jugular) glossopharyngeal ganglion.
FIGURE 12.20 Glossopharyngeal Nerve (CN IX)
Summary of glossopharyngeal nerve sensory and motor pathways.
The vagus nerve derives its name from the wandering course it takes in providing parasympathetic innervation to organs throughout the body (“vagus” means “wandering” in Latin). Other important functions are also served by the vagus, as we will discuss here. The vagus nerve exits the ventrolateral medulla as several rootlets just below CN IX, between the inferior olive and the inferior cerebellar peduncle (see Figure 12.2A,C). It crosses the subarachnoid space and then leaves the cranial cavity via the jugular foramen (see Figures 12.3A,B and 12.21).

The largest part of the vagus nerve provides parasympathetic innervation to the heart, lungs, and digestive tract, extending nearly to the splenic flexure (see Figures 6.13 and 12.21). Parasympathetic preganglionic fibers arise from the dorsal motor nucleus of CN X, which runs from the rostral to the caudal medulla (see Figure 14.5A,B). The dorsal motor nucleus of CN X forms the vagal trigone on the floor of the fourth ventricle, just lateral to the hypoglossal trigone, near the obex (see Figure 12.2B). Postganglionic parasympathetic neurons innervated by the vagus are found in terminal ganglia located within or near the effector organs. Recall that parasympathetics to the gastrointestinal tract beyond the splenic flexure—and to the urogenital system—are provided by parasympathetic nuclei in the sacral spinal cord (see Figure 6.13). The branchial motor component of the vagus (Figure 12.21) controls nearly all pharyngeal and upper esophageal muscles (swallowing and gag reflex) and the muscles of the larynx (voice box). The nucleus ambiguus supplies branchial motor fibers that travel in the vagus nerve to the muscles of the palate, pharynx, upper esophagus, and larynx, and in the glossopharyngeal nerve (CN IX) to the stylopharyngeus (see Figure 12.20).

A branch of the vagus called the recurrent laryngeal nerve (see Figure 12.21) loops back upward from the thoracic cavity to control all intrinsic laryngeal muscles except for the cricothyroid, which is innervated by another branch of the vagus, the superior laryngeal nerve. The fibers in the recurrent laryngeal nerve arise from the caudal portion of the nucleus ambiguus. After they exit the brainstem, these fibers travel briefly with CN XI before joining CN X (see the next section). Some texts consider these caudal fibers of the nucleus ambiguus to be part of CN XI and refer to the caudal nucleus ambiguus as the cranial nucleus of CN XI. However, we include these fibers with CN X because they spend the majority of their course traveling with CN X, not CN XI. Upper motor neuron innervation to the nucleus ambiguus controlling the voice and voluntary swallowing is from bilateral motor cortex (see Figure 6.2), except for the palate, which receives unilateral innervation from the contralateral cortex (for example, see Case 6.5).

General somatic sensory fibers of the vagus (see Figure 12.21) supply the pharynx, larynx, meninges of the posterior fossa, and a small region near the external auditory meatus (see Figure 12.7B). Note that below the larynx and pharynx, conscious (general somatic) sensation from the viscera is carried by spinal, not cranial, nerves. However, unconscious, general visceral sensation from chemoreceptors and baroreceptors of the aortic arch, cardiorespiratory system, and digestive tract is carried to the brainstem by the vagus nerve.
Many of these general visceral afferents reach the **caudal nucleus solitarius** (cardiorespiratory nucleus; see Figures 12.5 and 14.5B). The vagus nerve also contains a small number of **special visceral sensory** fibers that carry **taste** sensation from the epiglottis and posterior pharynx to the **rostral nucleus solitarius** (gustatory nucleus) (see Figures 12.5 and 14.5A).

The primary sensory neuron cell bodies for CN X general and special visceral sensation are located in the **inferior (nodose) vagal ganglion** (Table 12.5), located just below the jugular foramen. Cell bodies for general somatic sensation are located in both the inferior vagal ganglion and the **superior (jugular) vagal ganglion**, which lies within or just below the jugular foramen.
CN XI: Spinal Accessory Nerve

**FUNCTIONAL CATEGORY** | **FUNCTION**
--- | ---
Branchial motor | Sternomastoid and upper part of trapezius muscle

As its name implies, this nerve does not arise from the brainstem, but rather from the upper five or six segments of the cervical spinal cord (see Figure 12.2). The *spinal accessory nucleus* (also known as the accessory spinal nucleus) protrudes laterally between the dorsal and ventral horns of the spinal cord central gray matter (see Figure 14.5D), providing *branchial motor*^*'* fibers to this nerve. Nerve rootlets leave the spinal accessory nucleus and exit the lateral aspect of the spinal cord between the dorsal and ventral nerve roots just dorsal to the dentate ligament and ascend through the foramen magnum to reach the intracranial cavity (see Figures 12.2A and 12.3A,B). CN XI then exits the cranium again via the *jugular foramen* to supply the *sternomastoid* and upper portions of the *trapezius muscle*. The sternomastoid muscle turns the head toward the opposite side, and the trapezius is involved in elevating the shoulder (see neuroexam.com Video 46). The lower portions of the trapezius are usually supplied mainly by cervical nerve roots C3 and C4.

Note that the left sternomastoid turns the head to the right, and vice versa. Therefore, *lower motor neuron lesions* of CN XI may cause some ipsilateral weakness of shoulder shrug or arm elevation, and weakness of head turning away from the lesion. In turning the head, other neck muscles can sometimes compensate for the sternomastoid; therefore, in subtle cases, it is best to palpate the sternomastoid with one hand for contractions while the patient attempts turning their head against resistance offered by the examiner’s other hand. *Upper motor neuron lesions* can also cause deficits of head turning, toward the side opposite the lesion. Therefore, it is thought that the central pathways for head turning project to the *ipsilateral* spinal accessory nucleus. However, the deficit in head turning to the side opposite the lesion in cortical lesions is often more of a gaze preference than true weakness. With upper motor neuron lesions causing contralateral hemiparesis, the shoulder shrug is also often weak on the side of the hemiparesis.

Before it exits the cranium, the spinal accessory nerve is briefly joined by some fibers arising from the caudal nucleus ambiguous that exit from the lateral medulla adjacent to the vagus nerve. These fibers rejoin the vagus within a few centimeters and ultimately form the recurrent laryngeal nerve. As noted in the previous section, because these fibers travel briefly with CN XI, some textbooks refer to them as the *cranial root of CN XI*. Despite this name, the recurrent laryngeal nerve fibers spend the majority of their course traveling with CN X and can functionally be considered part of the vagus.

CN XII: Hypoglossal Nerve

**FUNCTIONAL CATEGORY** | **FUNCTION**
--- | ---
Somatic motor | Intrinsic muscles of the tongue

The hypoglossal nerve exits the ventral medulla as multiple rootlets between the pyramid and inferior olivary nucleus (see Figure 12.2A,C). This

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^*As we have already discussed, some consider the spinal accessory nerve to be somatic or mixed somatic and branchial rather than purely branchial motor, since the sternomastoid and upper trapezius muscles may have somatic embryological origins. For simplicity we have kept CN XI in the branchial motor category, since the spinal accessory nucleus is located laterally, in continuity with the branchial motor column (see Figure 12.5).
nerve exits through its own foramen, the hypoglossal foramen (see Figure 12.3A,B), and provides somatic motor innervation to all intrinsic and extrinsic tongue muscles except for the palatoglossus, which is supplied by CN X (see neuroexam.com Video 47). The hypoglossal nucleus is located near the midline on the floor of the fourth ventricle in the medulla (see Figure 14.5A,B), forming the hypoglossal trigone, just medial to the dorsal nucleus of CN X (see Figures 12.2B, 12.4B, and 12.5).

Upper motor neurons for tongue movement arise from the tongue region of the primary motor cortex (see Figure 6.2) and travel in corticobulbar pathways that decussate before reaching the hypoglossal nuclei. Therefore, lesions in the primary motor cortex or internal capsule will cause contralateral tongue weakness, while lesions of the hypoglossal nucleus, exiting fascicles, or nerve cause ipsilateral tongue weakness. Note that unilateral tongue weakness causes the tongue to deviate toward the weak side when it is protruded. Thus, a lesion of the hypoglossal nerve will cause the tongue to deviate toward the side of the lesion.

KEY CLINICAL CONCEPT

12.7 DISORDERS OF CN IX, X, XI, AND XII

Peripheral lesions of the lower cranial nerves are relatively uncommon. Most disorders of these cranial nerves are associated with central lesions (see Chapter 14). Like all other nerves, however, the lower cranial nerves are occasionally affected by diabetic neuropathy; demyelination; motor neuron disease; and traumatic, inflammatory, neoplastic, toxic, or infectious conditions. Let’s briefly discuss a few disorders of the lower cranial nerves.

Glossopharyngeal neuralgia is clinically similar to trigeminal neuralgia but involves the sensory distribution of CN IX, causing episodes of severe throat and ear pain. Injury to the recurrent laryngeal nerve (a branch of CN X), which can occur during surgery of the neck (such as carotid endarterectomy, cervical disc surgery, or thyroid surgery) or during cardiac surgery as the left nerve loops around the aorta (see Figure 12.21), produces unilateral vocal cord paralysis and hoarseness (see KCC 12.8). The recurrent laryngeal nerve can also be infiltrated by apical lung tumors during its looping course through the upper thoracic cavity, which produces hoarseness as part of Pancoast’s syndrome (see KCC 9.1). Glomus tumors are a rare disorder that can affect the lower cranial nerves. Glomus bodies are normal, small epithelioid structures that resemble the carotid bodies histologically, but whose function is unknown. Like the carotid bodies, they are richly innervated by CN IX but are located adjacent to the jugular foramen and along branches of CN IX leading to the middle ear cavity. Tumors arising from the glomus bodies are known by a variety of names, including glomus tumor and glomus jugulare. Patients with glomus jugulare often present with impairments of CN IX, X, and XI, resulting from compression of these nerves in the jugular foramen. In addition, the tumor frequently extends to the nearby CN XII and can grow upward to affect CN VIII and VII in the temporal bone. When the tumor grows into the middle ear, it can sometimes be seen on otoscopic exam as a fleshy vascular mass. Treatment is by resection, although radiation therapy is also used in some cases.

Clinical examination of CN IX through XII and the functional effects of lesions of these cranial nerves was discussed in the previous sections and is discussed in further detail in KCC 12.8.

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KEY CLINICAL CONCEPT

12.8 HOARSENESS, DYSARTHRIA, DYSPHAGIA, AND PSEUDOBULBAR AFFECT

Disorders of speech and swallowing can be very disabling, or even fatal in some cases. Causes of these disorders can range from upper motor neuron lesions (corticobulbar pathways) to lower motor neuron lesions to disorders of the neuromuscular junction or muscles themselves, and they can also result from cerebellar or basal ganglia dysfunction. Let’s discuss some of the more common causes of each of these disorders.

Voice disorders occur when the larynx and vocal cords (more correctly called the vocal folds, or true vocal cords) are not functioning properly. Such malfunction can occur as the result of mechanical factors or neural or muscular disorders. **Hoarseness** of the voice usually results from disorders of the vocal cords causing asynchronous vibratory patterns. Hoarseness is often caused by mechanical factors such as swelling, nodules, polyps, or neoplasms of the vocal cords. **Breathiness** of the voice is caused by paralysis or paresis of the vocal cord(s), which results in incomplete adduction of one or both of the vocal cords and an air leak at the glottis. In common language, breathiness is often called “hoarseness,” although this is not strictly correct. Recall that the muscles of the larynx are innervated by the branchial motor portion of CN X. As the recurrent laryngeal nerve loops through the upper thoracic cavity (see Figure 12.21) it can be injured during surgery in the neck or chest, or it can be compressed by apical lung cancer (Pancoast syndrome; see KCC 9.1). Voice disorders can also occur from lesions of CN X as it exits the brainstem, such as glomus jugulare (see KCC 12.7), or of the nucleus ambiguus in the medulla (see Figures 12.20 and 12.21). The most common lesion of the medulla affecting the nucleus ambiguus is a lateral medullary infarct (see Figure 14.21D; Table 14.7). An abnormal, gravelly sounding voice can also occur in Parkinson’s disease and other related movement disorders (see KCC 16.2). Spasmodic dysphonia is an uncommon form of dystonia (see KCC 16.1) involving the larynx, presumably resulting from dysfunction of basal ganglia circuitry. Vocal cord lesions or abnormal vocal cord movements can best be evaluated by **fiberoptic laryngoscopy**, in which a flexible scope is used to directly visualize the vocal cords during speech.

Dysarthria is abnormal articulation of speech (see neuroexam.com Video 45). Dysarthria should be distinguished from aphasia (see KCC 19.2). Whereas dysarthria is a motor articulatory disorder, aphasia is a disorder of higher cognitive functioning in which language formulation or comprehension is abnormal. Depending on the lesion, aphasia and dysarthria can occur together, or one can occur without the other. Dysarthria can range in severity from mild slurring to unintelligible speech. It can occur in lesions involving the muscles of articulation (jaw, lips, palate, pharynx, and tongue), the neuromuscular junction, or the peripheral or central portions of CN V, VII, IX, X, or XII. In addition, speech articulation can be abnormal because of dysfunction of the motor cortex face area (see Figure 6.2), cerebellum, basal ganglia, or descending corticobulbar pathways to the brainstem. Common causes of dysarthria include infarcts, multiple sclerosis, or other lesions affecting corticobulbar pathways (see KCC 6.3); brainstem lesions; lesions of cerebellar pathways or basal ganglia; toxins (e.g., alcohol); other causes of diffuse encephalopathy; myasthenia gravis; and other disorders of neuromuscular junction, muscle, or peripheral nerves. A few other important but less common specific causes of dysarthria to be aware of include amyotrophic lateral sclerosis (see KCC 6.7), botulism, and Wilson’s disease.
Dysphagia is impaired swallowing. Dysphagia can be caused by esophageal strictures, neoplasms, or other local lesions, or it may have a neural or neuromuscular basis. When dysphagia is caused by neural or neuromuscular disorders, it often has the same causes as, and occurs together with, dysarthria (although dysarthria and dysphagia can occur independently as well). Swallowing is classically divided into four phases: the oral preparatory phase (preparation of the food bolus for swallowing by mastication); oral phase (movement of the bolus in an anterior–posterior direction by the oral tongue); pharyngeal phase (propulsion of the bolus through the pharynx by base-of-tongue driving force, aided by anterior–superior movement of the larynx); and the esophageal phase (opening of the upper esophageal sphincter; esophageal peristalsis; and emptying into the stomach). Thus, dysphagia can be caused by dysfunction of muscles of the tongue, palate, pharynx, epiglottis, larynx, or esophagus; by lesions of CN IX, X, XII, or their nuclei; or by dysfunction at the neuromuscular junction or in the descending corticobulbar pathways.

Impaired oral and pharyngeal swallowing function and impaired reflex closure of the entrance to the trachea by the epiglottis and laryngeal muscles can lead to aspiration of food, and esophageal reflux can lead to aspiration of gastric secretions into the lungs. Aspiration pneumonia, caused by impaired swallowing function, is difficult to treat and is a common cause of death in disorders of the nervous system. Pharyngeal reflexes can be tested by the gag reflex. This reflex is elicited by stroking of the posterior pharynx with a cotton-tipped swab. The gag reflex is mediated by sensory and motor fibers from both CN IX and X, although CN IX may be more important for the afferent limb, while CN X provides primarily the efferent limb. Although an impaired gag reflex may be indicative of impaired motor or sensory function of the pharynx, its absence or presence is not absolutely predictive of aspiration risk.

A simple way to assess soft palate function is to observe palate elevation with a penlight while asking the patient to say, “Aah” (see neuroexam.com Video 44). In unilateral lesions of CN X or of the nucleus ambiguus, the uvula and soft palate will deviate toward the normal side, while the soft palate on the abnormal side hangs abnormally low, producing the stage curtain sign.

Brainstem nuclei involved in laughing and crying include CN VII, IX, X, and XII. Lesions of corticobulbar pathways in the subcortical white matter or brainstem can occasionally produce a bizarre syndrome called pseudobulbar affect. Patients with this syndrome exhibit uncontrollable bouts of laughter or crying without feeling the usual associated emotions of mirth or sadness. Pseudobulbar affect may be likened to an “upper motor neuron” disorder in which there is abnormal reflex activation of laughing and crying circuits in the brainstem, leading to emotional incontinence. The term pseudobulbar palsy is sometimes used to describe dysarthria and dysphagia caused by lesions not of the brainstem (bulb), but rather of the upper motor neuron fibers in the corticobulbar pathways (hence “pseudo”). Another neurologic cause of episodes of inappropriate laughter is a rare seizure disorder called gelastic epilepsy, which is usually associated with hypothalamic lesions (hypothalamic hamartoma) but can occasionally be seen in temporal lobe seizures (see KCC 18.2).

Review: Cranial Nerve Combinations

The preceding material is quite detailed, yet, as we will see, it has numerous important clinical applications. Let’s review several functional combinations to help consolidate the details of cranial nerve anatomy and to clarify some regional aspects of sensory and motor function. Functional combinations involving the eye muscles will be discussed in Chapter 13.
1. **Sensory and motor innervation of the face:** Sensation is provided by the trigeminal nerve (CN V), while movement of the muscles of facial expression is provided by the facial nerve (CN VII).

2. **Taste and other sensorimotor functions of the tongue and mouth:** The anterior two-thirds and posterior one-third of the tongue are derived from different branchial arches and therefore have different innervation. For the anterior two-thirds of the tongue, taste is provided by the facial nerve (CN VII, chorda tympani), while general somatic sensation is provided by the trigeminal nerve (V₃, mandibular division). For the posterior one-third of the tongue, both taste and general somatic sensation are provided by the glossopharyngeal nerve (CN IX). Taste for the epiglottis and posterior pharynx is provided by the vagus nerve (CN X). General sensation for the teeth, nasal sinuses, and inside of the mouth, above the pharynx and above the posterior one-third of the tongue, is provided by the trigeminal nerve (CN V).

3. **Sensory and motor innervation of the pharynx and larynx:** For the pharyngeal gag reflex, general somatic sensation is provided by both the glossopharyngeal and the vagus nerves (CN IX and X), but branchial motor innervation is provided mainly by the vagus (CN X). For the larynx, the vagus provides both sensory and motor innervation. General somatic sensation for organs below the level of the larynx is provided by the spinal nerves.

4. **Sensory and motor innervation of the ear:** General somatic sensation for the middle ear and inner tympanic membrane is provided by the glossopharyngeal nerve (CN IX), while sensation for the external ear and outer surface of the tympanic membrane is provided by the trigeminal (V₃, mandibular branch), facial (CN VII), glossopharyngeal (CN IX), and vagus (CN X) nerves (see Figure 12.7B). Hearing and vestibular sense travel in the vestibulocochlear nerve (CN VIII). Branchial motor innervation for the tensor tympani comes from the trigeminal nerve (CN V), while innervation for the stapedius comes from the facial nerve (CN VII). An aid to remembering this information is the fact that “tensor tympani” and “trigeminal” start with the letter T, while “stapedius” and cranial nerve “seven” start with S. Similarly, the tensor veli palatini is supplied by the trigeminal nerve, while all other muscles of the soft palate are supplied by the vagus.

5. **Sensation from the meninges:** Sensation from the supratentorial dura mater is carried by the trigeminal nerve (CN V), while the dura of the posterior cranial fossa is supplied by the vagus (CN X) and by the upper cervical nerve roots.

6. **General visceral sensation:** Unconscious, general visceral sensation from baroreceptors and chemoreceptors is carried by the glossopharyngeal nerve (CN IX) for the carotid body and sinus and by the vagus nerve (CN X) for the aortic arch and other thoracoabdominal viscera.

7. **Effects of unilateral cortical lesions:** The lower portions of the face (CN VII), soft palate (CN V, X), upper trapezius muscle (CN XI), and tongue (CN XII) receive mainly contralateral input, so they show weakness on the side opposite to cortical or corticobulbar lesions. Effects of unilateral upper motor neuron (UMN) lesions on eye movements are discussed in Chapter 13 (see KCC 13.10). Other cranial nerves do not typically show unilateral deficits with unilateral UMN lesions, although, interestingly, unilateral cortical or corticobulbar tract lesions can cause nonlateralized dysfunction in articulation (dysarthria) and swallowing (dysphagia).

Additional understanding of cranial nerve combinations will be gained through clinical practice.
Discussion

1. The key symptoms and signs in this case are:
   - Bilateral anosmia
   - Difficulty reading and left decreased visual acuity

   The anosmia could be caused by bilateral lesions of the olfactory mucosa or the olfactory nerves, bulbs, or tracts (see KCC 12.1). Decreased acuity in the left eye is consistent with a disorder in the left eye or the left optic nerve (see KCC 11.2). Deficits of CN I and CN II together suggest a lesion at the base of the frontal lobes, where these two cranial nerves briefly run in proximity (see Figure 18.6) before CN II exits the cranium via the optic canal (see Figure 12.3A). It is also possible that the anosmia is an unrelated incidental finding.

   The most likely clinical localization is bilateral orbital frontal areas.

2. Given the prolonged course, a slow-growing tumor at the base of the frontal lobes such as a meningioma should be suspected. Other tumors or chronic inflammatory disorders in this region are also possible, but less likely.

Clinical Course and Neuroimaging

The patient underwent a brain MRI (Image 12.1B,C, pages 540–541). For comparison, Image 12.1A (page 540) shows a normal MRI from another patient demonstrating the anatomical structures at the base of the frontal lobes. With the labels covered, identify the olfactory bulbs, olfactory sulcus, gyrus rectus, and cribriform plate. The images of the patient in this case, taken with gadolinium enhancement, are shown in Image 12.1B and C. An enhancing mass at the base of the frontal lobes extends along the dural surface in the region of the olfactory bulbs and erodes through the cribriform plate into the upper nasal passages (see Image 12.1B). The mass also extends back to encase the left optic nerve (see Image 12.1C). On the basis of its appearance and the patient’s history, it was felt that this was most likely a meningioma, although the irregular borders of the lesion that appeared to infiltrate adjacent structures were somewhat unusual for a meningioma. A biopsy of the mass was performed through the nose, via a transethmoidal approach. Interestingly, pathology revealed noncaseating granulomas consistent with sarcoidosis (see KCC 12.1). Additional workup supported the diagnosis of sarcoidosis confined to the nervous system. The patient was treated with steroids, and his vision improved in the left eye, but he remained unable to smell.

*This patient was described previously as a case report in The New England Journal of Medicine. 1996. 335: 1668–1674.
CASE 12.1 ANOSMIA AND VISUAL IMPAIRMENT

IMAGE 12.1A–C Mass in Orbital Frontal Region  T1-weighted MRI images with intravenous gadolinium enhancement. (A) Coronal image from a normal patient showing relation of olfactory bulbs to the frontal lobes and cribriform plate. (B,C) Coronal images from patient in Case 12.1, with B and C progressing from anterior to posterior.
MINICASE

A 51-year-old woman saw an ophthalmologist because she noticed that her left eye seemed to be bulging out increasingly for the past 3 to 4 years, and she had recently developed left-sided headaches. Exam was normal except for an outward bulging of her left eye (proptosis) and decreased sensation to touch and pinprick over her left cheek.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

1. Which division of which cranial nerve provides sensation to the cheek? Where does this nerve exit the skull?
2. What diagnosis is suggested by the slowly developing left proptosis over the course of several years, together with the cheek sensory deficit and left-sided headache?

CASE 12.2 CHEEK NUMBNESS AND A BULGING EYE

Discussion

1. The key symptoms and signs in this case are:
   - Left-sided headaches
   - Left proptosis
   - Decreased sensation to touch and pinprick over the left cheek

   The maxillary division of the trigeminal nerve (CN V2) provides sensation to the cheek (see Figure 12.7). This branch of the trigeminal nerve exits the skull through the foramen rotundum (see Figures 12.3, 12.7; Table 12.2).

2. The history and exam findings suggest a slow-growing mass lesion such as a meningioma (see KCC 5.8) involving the left foramen rotundum area, causing V2 sensory loss, and extending into the left orbit, causing proptosis.

Clinical Course and Neuroimaging

The patient underwent an MRI scan with gadolinium enhancement (Image 12.2, page 544) that revealed an enhancing mass lying outside the brain in
the region of the left foramen rotundum (compare Figure 12.3A) and extending into the left orbit. This appearance was felt to be consistent with a meningioma (see KCC 5.8). Because of concern that the mass would soon lead to impaired vision in the left eye, the patient was referred to a neurosurgeon. A left frontotemporal craniotomy was performed (see KCC 5.11), and a firm, grayish-reddish mass was carefully dissected off the sphenoid wing and removed from the orbit. Pathologic examination confirmed the diagnosis of meningioma. Postoperatively the patient did well, and she had no further problems.

**CASE 12.3 JAW NUMBNESS AND EPISODES OF LOSS OF CONSCIOUSNESS**

**MINICASE**

A 24-year-old woman was admitted to the cardiology service after an episode of syncope. Upon further probing, it was found that the patient had had three prior episodes of loss of consciousness over recent years, during which she was **unresponsive for a few minutes**, had some poorly described **shaking movements**, and was then **confused for up to a half an hour**. On review of systems, the patient described a patch of numbness over her left jaw that had been present for approximately 2 years. Exam was normal except for **decreased light touch, pinprick, and temperature sensation in the left jaw and lower face** (Figure 12.22).

**LOCALIZATION AND DIFFERENTIAL DIAGNOSIS**

1. On the basis of the symptoms and signs shown in **bold** above, where is the lesion?

2. What is the most likely diagnosis, and what are some other possibilities?

**DISCUSSION**

1. The key symptoms and signs in this case are:
   - **Decreased light touch, pinprick, and temperature sensation in the left jaw and lower face**
   - **Episodes of unresponsiveness lasting for a few minutes, with shaking movements, followed by confusion for up to a half an hour**

   The patient’s sensory loss was in the distribution of the mandibular division of the trigeminal nerve (CN V3; see Figure 12.7B). A lesion near the foramen ovale or of the mandibular division of CN V could, therefore, explain this deficit (see Figures 12.3A, 12.7A). Brief episodes of unresponsiveness can have numerous causes (see KCC 10.3; Table 10.2). Over 90% of cases are non-neurologic in origin and are caused by transient hypotension (vasovagal syncope), cardiac arrhythmias, or other medical conditions. However, patients with cardiogenic syncope typically recover immediately after the episode ends. Persistent deficits, such as the confusion seen in our patient, suggest a neurologic cause such as seizures (see KCC 18.2), vertebrobasilar transient ischemic attack (see KCC 10.3, 14.3), or vertebrobasilar migraine (see KCC 5.1). The shaking reported in this patient is suggestive of seizures, although a better description would have been helpful. One way to unify this patient’s findings into a single diagnosis would be to postulate a mass lesion near the left foramen ovale that extends to the adjacent left medial temporal lobe, causing seizures. We will see in Chapter 18 that the limbic structures of the temporal lobes are especially prone to epileptic seizures.
2. Possible causes of a lesion in the vicinity of the foramen ovale and medial temporal lobe include metastases, meningioma, trigeminal neuroma, aneurysm of the petrous segment of the internal carotid artery, or sarcoidosis (see KCC 12.1, 12.2).

Clinical Course and Neuroimaging
The patient underwent both an MRI and a CT scan of the head (Image 12.3A–C, pages 545–546). Image 12.3A is an axial proton density–weighted MRI image showing a roundish mass compressing the left medial temporal lobe, lying in the path of CN V in Meckel's cave. Image 12.3B is a coronal T1-weighted MRI image with gadolinium showing enhancement of the mass and extension downward through the foramen ovale. The “dumbbell” shape of this mass, extending through a bony foramen, is typical of a schwannoma (see KCC 12.5). Image 12.3C is an axial CT scan image, using bone windows to demonstrate erosion of the mass through the temporal bone in the region of the left foramen ovale. The mass appeared to lie outside the substance of the brain and was felt to represent a schwannoma (trigeminal neuroma), meningioma, or giant aneurysm. The patient was started on anticonvulsant medications and an angiogram was done, but no aneurysm was visualized. Therefore, she underwent a left frontotemporal craniotomy, and a tannish white mass was identified under the left temporal lobe. The tumor was carefully removed in a 10-hour operation, with care taken not to damage adjacent cranial nerves or blood vessels. Pathologic examination was consistent with a schwannoma. Postoperatively the patient made an excellent recovery, with no further seizures, but she had persistent numbness of the left jaw.

CASE 12.4 ISOLATED FACIAL WEAKNESS

MINICASE
A 26-year-old woman developed pain behind her left ear one evening. When she looked in the mirror the next morning, she noticed that her left face was drooping. In addition, her left ear was sensitive to loud sounds. She saw her physician, who gave her some medication for the pain, but over the next 2 days her left eye developed a “scratchy” painful sensation, so she came to the emergency room. Exam was notable for marked left facial weakness, including the forehead. Taste was not tested. The remainder of the exam was normal.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS
1. On the basis of the symptoms and signs shown in bold above, where is the lesion?
2. What is the most likely diagnosis, and what are some other possibilities?

Discussion
1. The key symptoms and signs in this case are:
   - Left retroauricular pain, hyperacusis, and facial weakness including the forehead
   - Painful, scratchy sensation in left eye

   This patient had lower motor neuron–type facial weakness (see Figure 12.13), together with hyperacusis and retroauricular pain on the left side. These findings are compatible with a lesion of the left facial nerve affecting branchial motor and general somatic sensory function (see Table 12.4; Figures 12.7B, 12.10).

   The painful, scratchy eye is a bit of a puzzle. However, patients with a facial nerve lesion may have parasympathetic involvement (see Figure 12.10) causing decreased lacrimation; also, they are often not able to completely close the affected eye, especially while they are sleeping, which can lead to corneal desiccation and corneal ulcers.
2. The time course, retroauricular pain, and lack of other medical problems or other findings on exam make Bell’s palsy the most likely diagnosis. For some other, less likely possibilities, see KCC 12.3.

Clinical Course
The patient was examined by an ophthalmologist but did not have corneal damage. She was given lubricating eyedrops and instructed to tape her left eyelid shut at night. In addition, she was treated with a brief course of oral steroids. Lyme titer, antinuclear antibody (ANA), and venereal disease research laboratory (VDRL) tests were undertaken and were negative. When the patient was seen in follow-up 1 month later, her facial weakness had completely resolved. She also no longer had ear pain or hyperacusis.

RELATED CASE. A CT scan from another patient with left facial weakness including the forehead is shown in Image 12.4A–D (page 547). This patient was a 19-year-old woman who fell off the back of a pickup truck, striking her occiput on the pavement without loss of consciousness. In addition to left lower motor neuron–type facial weakness, her exam was notable for left hemotympanum (see Table 3.9) and decreased taste on the left side of the tongue (tested by use of a cotton swab and mustard; see neuroexam.com Video 41). Image 12.4A–D shows CT scan sagittal reconstructions, which allow the course of CN VII to be followed through the temporal bone from medial to lateral. Note the presence of blood in the middle ear, and several fractures of the temporal bone. At the time of discharge, this patient’s facial weakness was unchanged, and she did not return for follow-up.

CASE 12.2 CHEEK NUMBNESS AND A BULGING EYE

IMAGE 12.2 Menigioma in Region of Left Foramen Rotundum  Axial T1-weighted MRI with intravenous gadolinium contrast enhancement.
**CASE 12.3** JAW NUMBNESS AND EPISODES OF LOSS OF CONSCIOUSNESS

IMAGE 12.3A–C  Trigeminal Schwannoma Eroding through Left Foramen Oval (A) Axial proton density–weighted MRI image. (B) Coronal T1-weighted image with intravenous gadolinium. (C) Axial CT scan image using bone windows.

(continued on p. 546)
CASE 12.3 (continued)

Erosion of mass through foramen ovale

Foramen ovale

Foramen spinosum

Foramen magnum
CASE 12.4 RELATED CASE

IMAGE 12.4A–D  Left Temporal Bone Fracture in Region of Facial Canal

Reconstructed sagittal CT scan images through the left temporal bone, with (A) through (D) progressing from medial to lateral.

(A) Cochlea  Auditory canal (CN VII, VIII)

(B) Region of genu of CN VII  Semicircular canals

(C) Blood in tympanic cavity (middle ear)  Facial canal (CN VII)

(D) External auditory meatus  Facial canal (CN VII)

Temporomandibular joint  Stylomastoid foramen
CASE 12.5 HEARING LOSS AND DIZZINESS

CHIEF COMPLAINT
A 41-year-old woman was referred to an otolaryngologist for dizziness and progressive hearing loss in the left ear.

HISTORY
One year ago the patient began having episodes of mild dizziness, which felt like the room was spinning when she moved her head. Two months ago she noticed greatly reduced hearing in her left ear, making it impossible to use the telephone receiver unless it was on her right ear. In addition, she had some left facial pain and decreased taste on the left side of her tongue. Past medical history was notable for a melanoma resected from the right hip region 6 months previously, with one positive lymph node.

PHYSICAL EXAMINATION
Ears: Normal otoscopic exam of the external auditory canals and tympanic membranes.
Neck: Supple.
Lungs: Clear.
Heart: Regular rate with no murmurs or gallops.
Abdomen: Benign.
Extremities: No edema.
Dermatologic: No skin lesions.
Neurologic exam:
MENTAL STATUS: Alert and oriented x 3. Mildly anxious, but otherwise normal.
CRANIAL NERVES: Pupils equal round and reactive to light. Normal fundi. Visual fields full. Extraocular movements intact. Facial sensation intact to light touch, but decreased corneal reflex on the left. Face symmetrical. Hearing greatly diminished to finger rub or whispering in the left ear. A vibrating tuning fork sounded louder when held just outside the left ear than when the handle was touched to the left mastoid process (air conduction greater than bone conduction). Taste was not tested. Voice and palate elevation normal. Sternomastoid strength normal. Tongue midline.
MOTOR: Normal tone. 5/5 power throughout.
REFLEXES:
COORDINATION: Normal on finger-to-nose and heel-to-shin testing.
GAIT: Normal.
SENSORY: Intact pinprick, vibration, and joint position sense.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS
1. On the basis of the symptoms and signs shown in bold above, where is the lesion?
2. What is the most likely diagnosis, and what are some other possibilities?

Discussion
1. The key symptoms and signs in this case are:
   - Decreased hearing in the left ear, with air conduction greater than bone conduction
   - Episodes of mild dizziness
   - Left facial pain and decreased left corneal reflex
   - Decreased taste on the left side of the tongue

   The patient had hearing loss with a sensorineural pattern that localized to the left cochlea or the left vestibulocochlear nerve (see KCC 12.5). Episodic dizziness can be caused by dysfunction anywhere in the pathways of vestibular sensation, including the labyrinth, vestibular ganglia, CN VIII, vestibular nuclei, cerebellum, or parietal cortex (see KCC 12.6). Given the sensorineural hearing loss, however, the dizziness is probably also caused by a problem in the left inner ear or CN VIII. Similarly, left facial pain (CN V), decreased corneal reflex (CN V1 or CN VII; see KCC 12.4), and decreased taste (CN VII) could each result from peripheral lesions of the respective cranial nerves or from lesions in the left brainstem. Since unilateral hearing loss must

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be caused by a lesion outside of the brainstem (see KCC 12.5), the most parsimonious explanation is a lesion in the left cerebellopontine angle, where CN V, VII, and VIII all lie in close proximity (see Figure 12.2A,C).

The most likely clinical localization is CN V, VII, and VIII in the left cerebellopontine angle.

2. The most common lesion of the cerebellopontine angle is acoustic neuroma (see KCC 12.5). Our patient in this case recently had a melanoma, so metastases should also be considered, especially since melanoma often metastasizes to the brain. Other, less likely possibilities include meningioma, epidermoid, and glioma. Meniere’s disease (see KCC 12.6) could account for hearing loss and dizziness, but not for this patient’s abnormalities of CN V and VII.

Clinical Course and Neuroimaging

The otolaryngologist ordered a brain MRI with gadolinium and special thin cuts through the region of the internal auditory canal (Image 12.5A,B, page 551). In these T1-weighted images, an enhancing mass can be seen in the left cerebellopontine angle. The mass appears to lie entirely outside of the brainstem and has a lateral knob extending into the left internal auditory meatus in the petrous portion of the temporal bone. These findings are highly suggestive of an acoustic neuroma (vestibular schwannoma; see KCC 12.5).

The patient was referred to a neurosurgeon and admitted for removal of the tumor. As is often the case with this kind of surgery, the procedure was a collaboration between neurosurgery and otolaryngology. The left occipital bone was opened behind the transverse sinus, the dura was opened, and the left cerebellar hemisphere was gently retracted to reveal the tumor. The tumor was carefully dissected away from the adjacent cerebellum; pons; CN V, VII, IX, and X; and branches of the posterior inferior cerebellar artery (see Figure 15.2). The functioning of the facial nerve was monitored continuously during the resection by use of a stimulating electrode placed on CN VII and by EMG (electromyography; see KCC 9.2) leads placed in the orbicularis oculi and labial muscles. Thus, although the facial nerve was severely distorted by the tumor, its function was preserved. CN VIII, however, was sacrificed because it was completely encapsulated by tumor, resulting in unilateral deafness. The pathology report confirmed schwannoma. Post-operatively the patient suffered from vertigo (see KCC 12.6) and had nystagmus at rest for 1 to 2 days, which then resolved. She also had complete left facial paralysis that resolved over the course of several months, and she subsequently did well.

CASE 12.6  HOARSE VOICE FOLLOWING CERVICAL DISC SURGERY

MINICASE

A 38-year-old saleswoman developed left neck and shoulder pain and evaluation revealed a cervical disc herniation, for which she underwent a discectomy and fusion via an anterior approach through the neck (see KCC 8.5). Her symptoms of cervical radiculopathy resolved. However, in the recovery room following surgery she noticed a marked change in her voice, which now had a breathy, “hoarse” quality. She was reassured that this was a temporary effect of the intubation. Nevertheless, over the next 2 months she continued to have severe breathiness of her voice, making it difficult for her to do her work as a personal shopper. She was referred to an otolaryngologist for evaluation. Conventional neurological exam was normal, aside from the breathiness. Her voice had a soft breathy quality suggesting that an air leak was present in her larynx.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS

On the basis of the symptoms and signs shown in bold above, where is the lesion, and what is the most likely cause?
Discussion

The key symptoms and signs in this case are:

- **Breathy, “hoarse” voice**

  Breathiness of the voice (often called hoarseness, although this is not strictly accurate) can be caused by any disorder that prevents complete closure of the vocal folds (true vocal cords) during phonation (see KCC 12.8). Incomplete vocal cord closure can be caused by lesions anywhere in the pathway from the nucleus ambiguus, to the vagus nerve (CN X), to the recurrent laryngeal nerve, to the muscles of the larynx (see Figure 12.21). Given this patient’s history of surgery on the left side of her neck, the most likely diagnosis is stretch injury or laceration of the left recurrent laryngeal nerve.

  Note that injury to the superior laryngeal nerve does not usually cause noticeable deficits, since it only supplies the cricothyroid muscle (a subtle deficit in reaching high notes is occasionally noted by professional singers), and injury of the vagus itself is uncommon during neck surgery because of its deeper location.

Clinical Course and Videostroboscopic Imaging

To confirm the diagnosis, the otolaryngologist performed fiberoptic video imaging of the larynx using a laryngoscope, which can be inserted through the nose or mouth (Image 12.6A–J, page 552). The process of stroboscopy matches the phonatory frequency to a strobe light, and by offsetting the phase slightly, it gives the illusion of slow-motion vibratory cycles of the true vocal cords. This process demonstrated normal movement of the right cord during phonation and during breathing. However, the left cord was paralyzed, and remained in an abducted position. Thus, her left-cord paralysis resulted in incomplete closure of the glottis during phonation and caused this patient’s breathiness.

Although recurrent laryngeal nerve injuries sometimes recover over time, this patient was eager to have the problem fixed immediately because of the severity of her deficit, its duration, and the importance of her voice for her work. Therefore, she underwent a procedure in which a precisely carved silastic insert was placed into the left paraglottic space. The insertion was performed while visualizing the cords and testing voice quality until the left cord was restored to a sufficiently medial position to allow normal approximation of the cords during phonation. Follow-up over time showed that her left recurrent laryngeal nerve injury was indeed permanent. However, the procedure enabled an immediate and complete recovery of her normal voice.
**CASE 12.5  HEARING LOSS AND DIZZINESS**

**IMAGE 12.5A,B  Left Acoustic Neuroma (Vestibular Schwannoma)**  Axial T1-weighted MRI images with intravenous gadolinium contrast. (A) and (B) are adjacent sections progressing from inferior to superior.
IMAGE 12.6A–J Left Vocal Cord Paralysis  Videostroboscopic images through the laryngoscope during a cycle of phonation (adduction) and inspiration (abduction).

(A–G) During phonation, the right cord adducts medially, while the left cord remains immobile, causing an air leak.

(H–J) During inspiration, the right cord abducts laterally, while the left cord remains immobile. (Courtesy of Michael Goldrich, Robert Wood Johnson Medical School, University of Medicine and Dentistry of New Jersey.)
CASE 12.7 HOARSENESS, WITH UNILATERAL WASTING OF THE NECK AND TONGUE MUSCLES

CHIEF COMPLAINT
A 34-year-old man was referred to an otolaryngologist for progressive hoarseness, dysphagia, and weakness of the left sternomastoid and tongue.

HISTORY
Four months prior to presentation, the patient developed a persistent cough and respiratory infection that did not resolve. Soon afterward he noticed difficulty swallowing thick foods, and his voice gradually became hoarse. Three weeks prior to presentation, he began to have decreased hearing in the left ear, some alteration in taste, and mild left-sided headache. He lost 40 pounds during the 4 months since developing symptoms.

PHYSICAL EXAMINATION
Vital signs: T = 98.1°F, P = 84, BP = 118/86, R = 18.
Neck: Supple; no adenopathy or palpable masses.
Lungs: Clear.
Heart: Regular rate with no gallops or murmurs.
Abdomen: Soft, nontender.
Extremities: Normal.
Neurologic exam:
CRANIAL NERVES: Pupils 4 mm, constricting to 2 mm bilaterally. Visual fields full. Normal optic discs. Extraocular movements intact. Facial sensation intact to light touch and pinprick. Intact corneal reflexes. Mildly decreased left nasolabial fold. Decreased hearing to finger rub on the left. Gag intact. Uvula deviated to the right with palate elevation. Voice hoarse and breathy in quality. Left trapezius and sternomastoid muscles had fasciculations and strength of 4/5. Tongue had marked asymmetrical atrophy and fasciculations of the left side, with tongue deviating to the left on protrusion. On laryngoscopic examination, the left vocal cord was paralyzed (see Case 12.6).
MOTOR: No pronator drift. 5/5 power throughout.
REFLEXES:
COORDINATION: Normal on finger-to-nose and heel-to-shin testing.
GAIT: Normal.
SENSORY: Intact light touch, pinprick, and joint position sense.

LOCALIZATION AND DIFFERENTIAL DIAGNOSIS
1. On the basis of the symptoms and signs shown in bold above, where is the lesion?
2. What are some possible lesions in this location?

Discussion
1. The key symptoms and signs in this case are:
   - Difficulty swallowing, decreased left palate movement, hoarseness, and left vocal cord paralysis
   - Left trapezius and sternomastoid weakness and fasciculations
   - Left tongue deviation, atrophy, and fasciculations
   - Decreased hearing in the left ear
   - Mildly decreased left nasolabial fold
   - Alteration in taste
   - Left-sided headache

   This patient has multiple abnormalities of the cranial nerves on the left side of the head. Although each individual abnormality could be explained by a small brainstem lesion, all of the relevant nuclei could not be involved together without also involving other nearby structures, such as the anterolateral system, inferior cerebellar peduncle, and descending sympathetic pathway (see Figure 14.21D). In addition, as in Case 12.5, the unilateral hearing loss suggests that the lesion lies outside of the brainstem (see KCC 12.5).
Taking each of the above deficits in turn, the swallowing muscles of the pharynx and the left palate are innervated by the left CN X, although CN IX may contribute to the gag reflex as well. A lesion of the left CN X could also explain hoarseness (breathiness) and left vocal cord paralysis, since the larynx is innervated by the vagus as well. Left trapezius and sternomastoid weakness and fasciculations suggest a lower motor neuron lesion (see KCC 6.1) of the left spinal accessory nerve (CN XI). Similarly, deviation of the tongue to the left, together with atrophy and fasciculations, suggests a lower motor neuron lesion of the left hypoglossal nerve (CN XII). Decreased hearing in the left ear can be caused by a lesion in the left external auditory canal, middle ear, cochlea, or vestibulocochlear nerve (CN VIII). Although a decreased left nasolabial fold could be caused by upper motor neuron– or mild lower motor neuron–type weakness, given the other findings, a peripheral lesion of the left facial nerve (CN VII) is more likely. A facial nerve lesion could also explain the alteration in taste. Unilateral headaches can have many causes (see KCC 5.1), but in this setting they support the presence of an intracranial lesion on the left side of the head.

To summarize, this lesion involves CN VII, VIII, IX, X, XI, and XII on the left side. These cranial nerves exit the left lower brainstem and leave the cranium via the internal auditory meatus, jugular foramen, and hypoglossal canal (see Figures 12.2A,C; 12.3A,B; Table 12.2). Note that large lesions of the cerebellopontine angle usually involve CN V (see KCC 12.5 and Case 12.5). Since CN V was spared in this case, it suggests that the lesion lies farther down.

The most likely clinical localization is a large lesion lying just outside of the left ventrolateral medulla or in the vicinity of the left internal auditory meatus, jugular foramen, and hypoglossal canal.

2. Possible lesions in this location include meningioma, schwannoma, metastases, granulomatous disease, and glomus tumors (see KCC 12.7).

Clinical Course and Neuroimaging

A brain MRI with gadolinium was ordered (Image 12.7A,B, page 557). Image 12.7A shows an axial T1-weighted image demonstrating an enhancing mass filling the left jugular foramen, just behind the left internal carotid artery. Note that the mass extends into the posterior fossa near the region where the left hypoglossal nerve exits the medulla to traverse the subarachnoid space. Image 12.7B is a coronal T2-weighted image revealing that the mass extends up into the left petrous temporal bone to reach the vicinity of the left seventh and eighth cranial nerves. These findings are compatible with a left glomus jugulare tumor (see KCC 12.7).

The patient was initially treated with radiation therapy, resulting in no progression of his deficits. He was actively involved in a speech therapy program to help him work on his voice and swallowing. Four years later, however, he began to have worsening hoarseness, severe left facial weakness, and left mastoid pain. Since his tumor appeared to be growing, surgery was planned. Because glomus tumors tend to be very vascular and can bleed profusely during surgery, he had a preoperative angiogram during which an interventional neuroradiologist (see Chapter 4) embolized as much of the tumor as possible. This procedure was followed by a prolonged operation involving collaboration between two teams of neurosurgeons and otolaryngologists, who accomplished a complete removal of all visible tumor. Unfortunately, on pathologic analysis, rather than the usual benign appearance of glomus tumors, this lesion had malignant features including mitotic figures (indicating active cellular proliferation) and necrosis. He remained
stable until 1 year later, when he had worsening left ear pain and an MRI showed recurrence of the tumor. Again, he underwent embolization followed by surgery. However, soon afterward he developed aspiration pneumonia with overwhelming sepsis, and he died five and a half years after the onset of his initial symptoms.

**CASE 12.8 UNCONTROLLABLE LAUGHTER, DYSARTHRIA, DYSPHAGIA, AND LEFT-SIDED WEAKNESS**

**CHIEF COMPLAINT**
A 27-year-old male saxophone player came to the emergency room because of worsening dysarthria, dysphagia, left-sided weakness, and episodes of uncontrolable laughter.

**HISTORY**
Two and a half years prior to presentation, the patient developed episodes of left face and mouth pain precipitated by chewing. One year prior to presentation, he started having episodes of uncontrollable laughter, not accompanied by appropriate affect. When he persisted in laughing repeatedly at his girlfriend’s father’s wake, he was referred to a psychiatrist, who tried behavior modification therapy without benefit. Two to 3 months prior to presentation, he developed increasing difficulty playing the saxophone, and he noticed slurred speech and occasional choking on his food. He found he also had an unstable gait, difficulty buttoning his shirt with his left hand, and urinary urgency and difficulty initiating urination.

**PHYSICAL EXAMINATION**

- **Vital signs:** T = 98°F, P = 72, BP = 130/70, R = 12.
- **Neck:** Supple with no bruits.
- **Lungs:** Clear.
- **Heart:** Regular rate with no gallops or murmurs (but difficult exam because of frequent laughter).
- **Abdomen:** Soft, nontender.
- **Extremities:** Normal.
- **Neurologic exam:**
  - **MOTOR:** Mild left pronator drift. Slowed finger tapping in the left hand. Tone slightly increased in left lower extremity. Power 4/5 in left deltoid, triceps, wrist extensors, finger extensors, iliopsoas, hamstrings, tibialis anterior, and extensor hallucis longus, but otherwise 5/5 throughout.
  - **REFLEXES:**
  - **COORDINATION:** Finger-to-nose and heel-to-shin testing slowed, but without ataxia.
  - **GAIT:** Slightly unsteady, with stiff left lower extremity.
  - **SENSORY:** Intact light touch, pinprick, temperature, vibration, and joint position senses and graphesthesia.

**LOCALIZATION AND DIFFERENTIAL DIAGNOSIS**
1. On the basis of the symptoms and signs shown in bold above, which nerves (see Table 12.4) and which long tracts (see Chapters 6 and 7) are affected by the lesion?
2. In what general region of the nervous system can a single lesion produce all of these findings?
3. What are some possible lesions in this location?

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**Discussion**

1. The key symptoms and signs in this case are:
   - Episodes of left face and mouth pain
   - Episodes of uncontrollable laughter not accompanied by appropriate affect
   - Dysarthria, dysphagia, absent gag reflex

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• Mild weakness of head turning to the left
• Left face arm and leg weakness, with increased tone, hyperreflexia, and gait unsteadiness
• Urinary urgency and difficulty with initiation

The episodes of left face and mouth pain precipitated by chewing were initially suggestive of a trigeminal nerve (CN V) disorder, such as trigeminal neuralgia (see KCC 12.2). However, later findings suggest a lesion affecting central nervous system pathways. These include the development of pseudo-bulbar affect, suggesting a lesion of the corticobulbar pathways (see KCC 12.8), left hemiparesis with upper motor neuron signs compatible with corticobulbar and corticospinal dysfunction (see KCC 6.3), and urinary dysfunction, also compatible with impairment of descending pathways controlling micturition (see KCC 7.5). Dysarthria, dysphagia, and absent gag reflex (CN IX, X) in this patient, along with impaired CN XI function, further support a combination of cranial nerve dysfunction along with involvement of long tracts. These findings suggest a lesion affecting CN V, IX, X, and XI, as well as corticobulbar, corticospinal, and descending sphincteric pathways.

2. A lesion of the brainstem in the region of the pons and medulla could affect these multiple cranial nerves and long tracts. A lesion affecting this many brainstem structures while preserving other brainstem nuclei and pathways would have to be fairly extensive, yet cause patchy involvement.

3. Given the gradual onset of symptoms over several years involving multiple brainstem structures, one possibility would be multiple sclerosis (see KCC 6.6) affecting primarily the brainstem. Other possibilities include a brainstem vascular malformation (see KCC 5.6), a granulomatous disorder such as sarcoidosis (see KCC 12.1), or a slow-growing tumor such as a brainstem glioma or meningioma (see KCC 5.8).

Clinical Course and Neuroimaging

The patient underwent a head CT scan in the emergency room revealing a mass lesion, which was better visualized by MRI scan (Image 12.8A,B, page 558). Note the presence of a large mass lying outside of the brain adjacent to the dura and enhancing uniformly with gadolinium, consistent with a meningioma (see KCC 5.8). The mass could be seen to cause severe compression and distortion of the pons and left middle cerebellar peduncle (see Image 12.8A). The patient’s relatively mild deficits given this degree of distortion attest to the chronic nature of this lesion. The mass could also be seen to extend into the region of Meckel’s cave adjacent to the left cavernous sinus (see Image 12.8B), possibly explaining the patient’s early symptoms of left facial pain. The patient underwent a multistage resection, involving preoperative embolization by interventional radiology, and two collaborative operations involving teams of neurosurgeons and otolaryngologists. He made an excellent recovery with minimal deficits. On follow-up examination 1 year later (Image 12.8C, page 559), he still had rare episodes of inappropriate laughter, and he had some mild diplopia that he had developed following surgery, but he was otherwise without deficits. Repeat MRI scan showed near complete removal of the tumor (see Image 12.8C), with only a small portion left where it was adherent to CN IV.
**CASE 12.7  HOARSENESS, WITH UNILATERAL WASTING OF THE NECK AND TONGUE MUSCLES**

**IMAGE 12.7A,B  Left Glomus Jugulare Tumor** (A) Axial T1-weighted MRI image with gadolinium. (B) Coronal T2-weighted MRI image.
CASE 12.8 UNCONTROLLABLE LAUGHTER, DYSAVRTHRIA, DYSPHAGIA, AND LEFT-SIDED WEAKNESS

IMAGE 12.8A–C Meningioma Compressing the Pons T1-weighted MRI images with intravenous gadolinium enhancement. (A) Sagittal view. (B) Axial view. (C) Follow-up MRI axial view 1 year after surgery.
Additional Cases

Related cases can be found in other chapters for: upper or lower motor neuron cranial nerve disorders (Cases 5.2, 5.3, 5.7, 5.8, 6.3, 6.5, 10.4, 10.5, 10.11, 11.1, 11.2, 13.1–13.3, 13.5, 14.1, 14.4, 14.7, 15.4, 17.2). Other relevant cases can be found using the Case Index located at the end of the book, and new cases are also available through the Online Review and Study Guide.

Brief Anatomical Study Guide

1. The main parts of the brainstem are the midbrain, pons, and medulla, as shown in Figure 12.1. The cranial nerves (see Table 12.1) exit the brainstem roughly in numerical sequence from rostral to caudal (see Figure 12.2), except for CN I and CN II, which arise from the forebrain. Each cranial nerve exits the skull through a specific foramen, as summarized in Table 12.2 and Figure 12.3.

2. As in the spinal cord gray matter, the cranial nerve nuclei for motor functions are located more ventrally in the brainstem, and those for sensory functions are located more dorsally (see Figure 12.4).

3. Along the long axis of the brainstem are three motor columns and three sensory columns of cranial nerve nuclei (see Figures 12.4 and 12.5). The functions, nuclei, and cranial nerves for each of these columns are summarized in Table 12.3. Note that each nucleus can be involved in the motor or sensory functions of one or more cranial nerves. Similarly, each cranial nerve can have both sensory and motor functions, as well as connections with one or more cranial nerve nuclei (see Table 12.4).
Brief Anatomical Study Guide (continued)

4. Some cranial nerves have peripheral ganglia that contain either primary sensory neurons or parasympathetic postganglionic neurons (see Table 12.5; Figure 12.6).

5. The olfactory nerves (CN I) (see Figures 18.5 and 18.6) traverse the cribiform plate of the ethmoid bone to synapse in the olfactory bulbs (see Figure 12.3A). Olfactory information then travels to the olfactory cortex (see Chapter 18) via the olfactory tracts.

6. The optic nerve (CN II) enters the cranial cavity via the optic canal (see Figure 12.3A,C), carrying visual information from the retina to the lateral geniculate nucleus and extrageniculate pathways (see Chapter 11).

7. The oculomotor (CN III), trochlear (CN IV), and abducens (CN VI) nerves are involved in eye movements and pupillary control (CN III) and are discussed at length in Chapter 13.

8. The trigeminal nerve (CN V) provides sensation for the face, mouth, and meninges of the supratentorial cranial cavity via three major branches: the ophthalmic division (V1), maxillary division (V2), and mandibular division (V3) (see Figure 12.7). These branches enter the skull via the superior orbital fissure, foramen rotundum, and foramen ovale, respectively (see Figures 12.3A, 12.7), with primary sensory neurons located in the trigeminal ganglion. The trigeminal sensory nuclei include the mesencephalic trigeminal nucleus mediating proprioception, the chief trigeminal sensory nucleus mediating discriminative touch, and the spinal trigeminal nucleus mediating pain and temperature sensation (see Figures 12.5, 12.8; Table 12.6). Trigeminal sensory information travels to the cortex via the trigeminal lemniscus and trigeminothalamic tract, with a relay in the VPM of the thalamus (see Figure 12.8; Table 12.6). The trigeminal nerve also has a small motor root that travels with CN V3, supplying the muscles of mastication (see Figure 12.7).

9. The facial nerve (CN VII) controls the muscles of facial expression (to be distinguished from the trigeminal nerve that mediates facial sensation) via fibers that arise from the facial nucleus in the pons (see Figure 12.11). The facial nerve travels along with CN VIII in the auditory canal and then exits the skull via the stylomastoid foramen (see Figures 12.3B and 12.10). Upper motor neuron control of the facial nucleus is bilateral for the upper parts of the face, so in unilateral upper motor lesions the contralateral side can compensate, resulting in sparing of the upper face muscles (see Figure 12.13). The facial nerve also has sensory fibers that provide taste sensation for the anterior two-thirds of the tongue reaching the nucleus solitarius (see Figure 12.12) and somatic sensation fibers for a region around the outer ear traveling to the trigeminal nuclei (see Figure 12.7B). Sensory cell bodies lie in the geniculate ganglion. Parasympathetics arising from the superior salivatory nucleus travel in CN VII via the sphenopalatine ganglion and submandibular ganglion, respectively, to the lacrimal glands and salivary glands (see Figure 12.6).

10. The vestibulocochlear nerve (CN VIII) carries auditory information from the cochlea to the dorsal and ventral cochlear nuclei (see Figures 12.15–12.17). The primary sensory cell bodies lie in the spiral ganglion. Central auditory pathways cross multiple times, so unilateral lesions in the
central nervous system do not cause clinically significant unilateral hearing loss (see Figure 12.16). Information about head position and acceleration is carried by the vestibular portions of CN VIII arising from the **semicircular canals and otolith organs** (see Figure 12.15). Primary cell bodies are in the vestibular ganglia, and this information travels to the **vestibular nuclei** in the brainstem to influence unconscious posture and balance, eye movements, and conscious perception of movement through multiple pathways (see, e.g., Figure 12.19).

11. The **glossopharyngeal nerve** (CN IX) exits the skull via the jugular foramen (see Figures 12.3A,B, 12.20; Table 12.2). Motor fibers arising from the **nucleus ambiguus** provide innervation of the stylopharyngeus muscle, important for pharynx elevation during speech and swallowing. Sensory fibers from chemoreceptors and baroreceptors in the carotid body reach the caudal nucleus solitarius (cardiorespiratory nucleus). Taste sensory fibers from the posterior one-third of the tongue travel to the rostral nucleus solitarius (gustatory nucleus). Somatic sensation from the posterior tongue, pharynx, middle ear, and external ear travels via CN IX to the trigeminal nuclei. Finally, parasympathetics arising from the inferior salivatory nucleus activate the parotid salivary gland via the otic ganglion.

12. The **vagus nerve** (CN X) also has multiple functions, providing parasympathetic innervation for the viscera arising from the **dorsal motor nucleus of CN X** (see Figures 12.5 and 12.21; see also Figure 14.5A,B). In addition, motor fibers of the vagus arising from the **nucleus ambiguus** supply the pharynx (swallowing) and larynx (voice). Sensory fibers from the aortic arch travel to the caudal nucleus solitarius. Sensory fibers for the pharynx, larynx, outer ear, and meninges of the posterior fossa travel to the trigeminal nuclei.

13. The **spinal accessory nerve** (CN XI) (see Figure 12.2A,C) arises from the **spinal accessory nucleus** (see Figure 12.5) and innervates the sternomastoid and upper portions of the trapezius muscles. Because of the mechanical attachments of the sternomastoid muscle, lesions of CN XI cause weakness of head turning to the side opposite the lesion.

14. The **hypoglossal nerve** (CN XII) (see Figure 12.2A,C) arises from the hypoglossal nucleus (see Figures 12.4 and 12.5) and supplies intrinsic tongue muscles. Hypoglossal nerve lesions cause the tongue to deviate toward the side of the lesion when the tongue is protruded.

References

**General References**


**Cribriform and Suprasellar Meningiomas**


**Central Nervous System Sarcoidosis**


**Trigeminal Nerve Lesions**


**Facial Nerve Lesions**


**Dizziness and Vertigo**


**Acoustic Neuroma**


**Glomus Jugulare**

