5 THE CRANIAL NERVES

CHAPTER OVERVIEW

Of all the systems that a clinician might review, examination of the cranial nerves potentially can be the most accurate in terms of identifying the locus of a lesion in the CNS. As noted in Chapter 4, because of the relative compactness of the nuclei and various nerves and pathways within the brainstem, under certain circumstances it might be possible to localize a brainstem lesion to within 1 cm. Beyond this, the presence or absence of certain cranial nerve deficits might help the clinician differentiate whether certain sensory or motor deficits have their origin above or below the level of the brainstem. The two cranial nerves that lie totally outside the brainstem may help identify lesions lying within any of the four lobes of the brain.

Beyond and perhaps more important than their localizing potential the behavioral deficits associated with lesions of the cranial nerves can have a profound impact on patients' functional capacity. Whether as a direct result of the cranial nerve deficits themselves or as a function of presumed disruption of critical adjacent structures, such deficits may have vocational, social, emotional, or psychiatric implications.

Consistent with the goal of combining anatomical structure with functional relevance, a review of specific behavioral syndromes and their clinical assessment will accompany a review of the gross anatomical and functional correlates of each of the twelve cranial nerves. At the conclusion of this chapter, the reader should be able to identify each of the cranial nerves, their major functions, and how each might be tested as part of the overall clinical examination of a patient. Utilizing the information presented in this chapter, along with that reviewed in Chapters 1, 2 and 4, one hopefully should be able to derive tentative hypotheses regarding the probable level of a lesion producing sensory and/or motor deficits.

INTRODUCTION

A substantial part of the neurological examination is an assessment of the cranial nerves. In addition to providing valuable information regarding the integrity of the brainstem and/or the localization of lesions therein, the discovery of functional disturbances in one or more of the cranial nerves may herald the presence of other intracranial or extracranial pathology. To more fully understand the significance of this portion of the neurological examination, it is necessary to describe the major functional and anatomical features of each of these cranial nerves. However, before doing this a few general facts and characteristics of these nerves and their nuclei will be reviewed.

The cranial nerves are those paired sets of nerves whose constituent fibers enter (or exit) the central nervous system above the level of the foramen magnum, that is, at the level of the brainstem or above (see Figure 5–1). In fact, of the 12 pairs of cranial nerves (CN), all but two—the olfactory (CN I) and optic (CN II)—have their nuclei in the brainstem. The oculomotor (CN III) and trochlear (CN IV) exit from the midbrain. The trigeminal (CN V) enters and leaves at the pontine level. Three of the nerves—the abducens (CN VI), facial (CN VII), and vestibulocochlear (CN VIII)—are found at the pontomedullary junction. The glossopharyngeal (CN IX), vagus (CN X), and hypoglossal (CN XII) are located farther down the medulla. Finally, the spinal accessory (CN XI) has cells of origin both in the medulla and cervical cord. Of the cranial nerves whose nuclei are in the brainstem, all except one (CN IV) are found on the ventral or ventrolateral aspect of the stem (the fourth cranial nerve exits from the dorsal portion of the midbrain). It might be noted that CN IV also is unique in another respect: it is the only nerve to fully cross the midline after leaving its nucleus.

Unlike the spinal nerves that are either sensory or motor, some of the cranial nerves are purely motor (CN III, IV, VI, XI, XII), others are purely sensory (CN I, II, VIII), while others

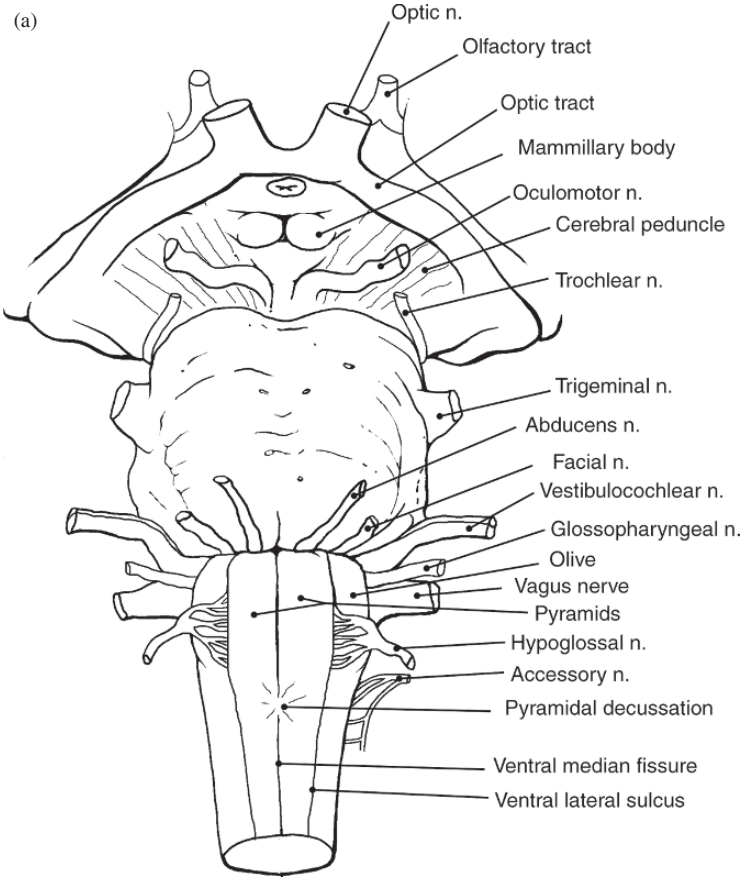

Figure 5–1. (a) Anterior, (b) lateral, and (c) posterior views of brainstem and hypothalamus showing relative location of cranial nerves. Optic nerve exits from the eye, forming optic tract after partial decussation in the optic chiasm. Olfactory nerves synapse in the olfactory bulb (not shown) and second-order fibers form the olfactory tract (see Figure 5–3).

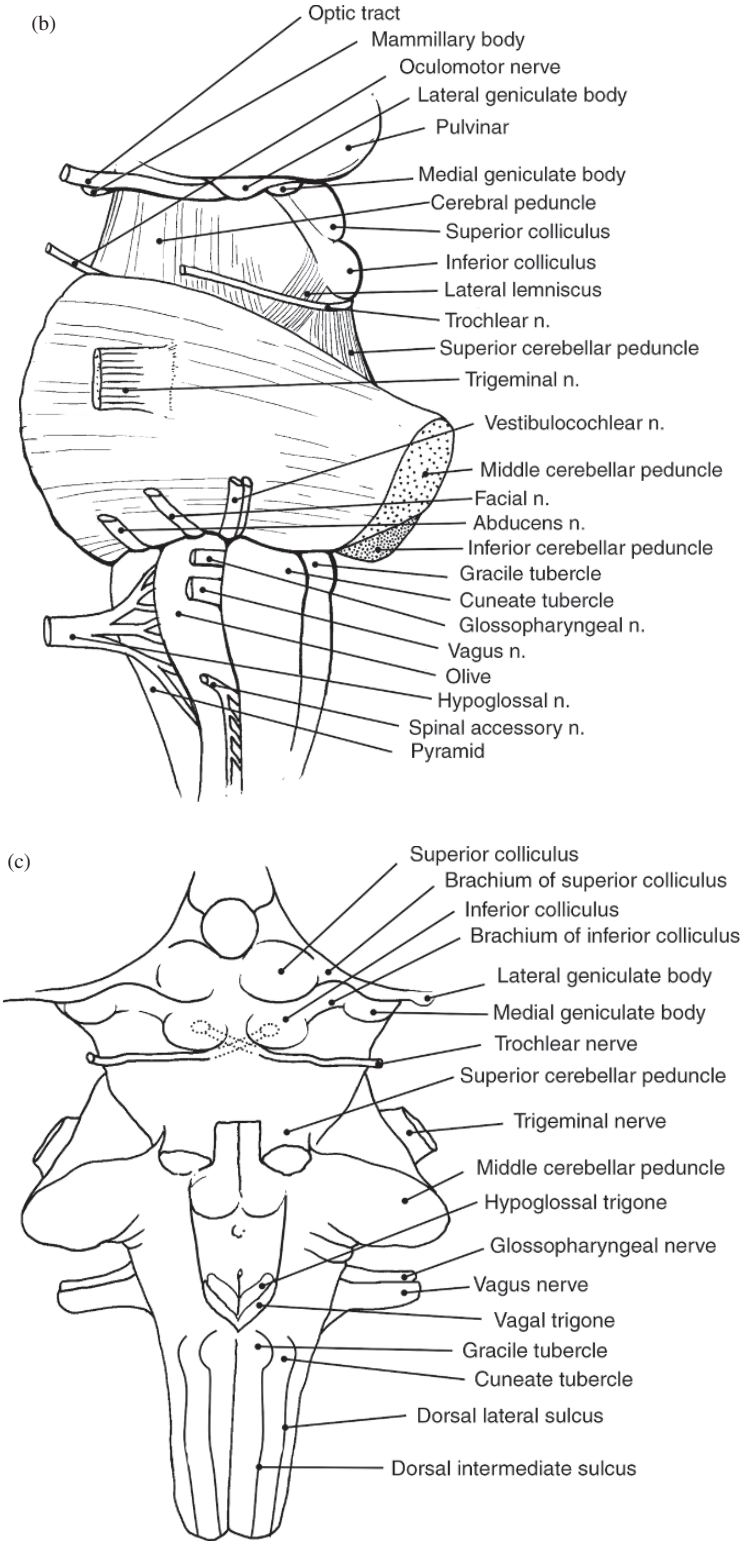

Figure 5–2. *(Continued)*

contain both motor and sensory components (CN V, VII, IX, X). Several of the brainstem nuclei are shared by more than one cranial nerve. For example, the spinal nucleus of the trigeminal nerve is shared by CN V, VI, IX, and X. Another conventional although somewhat more complex way to distinguish individual nerves or their components is based on whether they serve **general somatic functions** or **general visceral functions**. Since these can have either afferent (sensory) or efferent (motor) components, four classifications can be readily described:

1. General somatic efferents (GSE): (CN III, IV, VI, XII)
2. General visceral efferents (GVE): (CN III, VII, IX, X)
3. General somatic afferents (GSA): (CN V, VII, IX, X)
4. General visceral afferents (GVA): (CN IX, X)

The above classifications would apply equally to the spinal nerves. However, the cranial nerves are also responsible for certain special functions that are not found in the spinal nerves. Hence, we have certain "special" cranial nerves or components of those nerves. These are:

5. Special somatic afferents (SSA): (CN II, VIII)
6. Special visceral Aafferents (SVA): (CN I, VII, IX, X)
7. Special visceral efferents (SVE): (CN V, VII, IX, X, XI)[1]

Translating these classifications into more clinically meaningful or descriptive terms, we would have the following:

1. Special somatic afferents (SSA): sensory functions unique to the cranial nerves: vision (CN II), hearing and balance/equilibrium (CN VIII).
2. Special visceral afferents (SVA): taste (CN VII, IX, X) and smell (CN I).
3. General somatic afferent (GSA): perception of touch, pain, temperature, vibration, proprioception, and stereognosis (CN V, VII, IX, X).
4. General visceral afferent (GVA): feedback from such structures as the mucous membranes of the nasal and oral cavities, thoracic and abdominal organs, and the carotid sinus (CN IX, X).
5. General somatic efferents (GSE): control over the extrinsic muscles (movement) of the eyes (CN III, IV, VI), and movement of the tongue (CN XII).
6. General visceral efferents (GVE): control the constriction of the pupils and changes in the shape of the lens of the eye (CN III), lacrimal (CN VII), and salivary (CN VII, IX) glands, and the muscles related to visceral organs such as the heart and diaphragm (CN X).
7. Special visceral efferents (SVE): control over "special" visceral or "brachial" motor functions such as chewing (CN V), facial expressions and tension on the stapedius muscle (CN VII), and tympanic membrane (CN V); the muscles of the larynx and pharynx used in speaking (CN IX, X); and turning of the head (sternocleidomastoid) and shrugging of the shoulders (trapezius) (CN XI).

Following the embryological development of the central nervous system, the somatic and visceral motor nuclei (which develop from the basal plate) tend to be more medially placed in the brainstem tegmentum. In contrast, the brainstem nuclei that represent somatic and visceral afferents (evolving from the alar plate) tend to be more laterally situated. As can be seen in Figure 5–2, the somatic motor nuclei tend to be placed most medially and the somatic sensory nuclei most laterally of the four, with the visceral motor and sensory

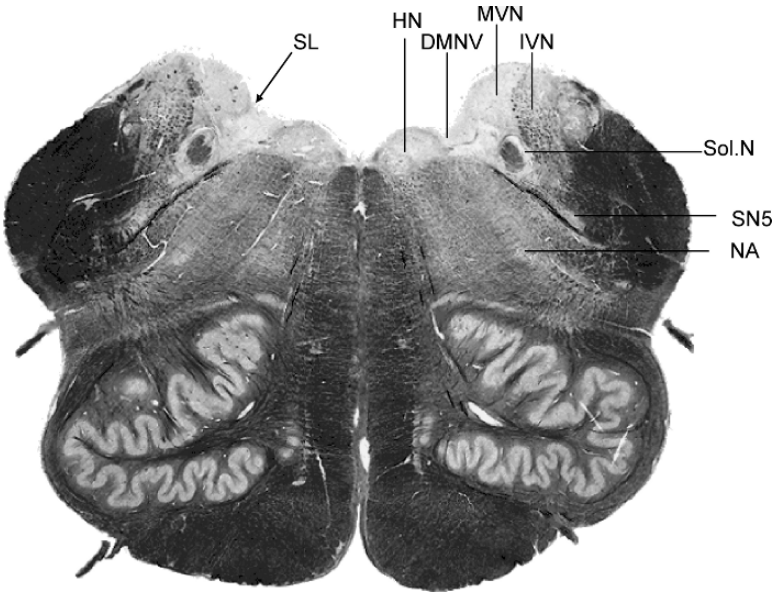

Figure 5–2. Section through the upper medulla representative of the relative arrangement of sensory and motor nuclei in the brainstem. As can be seen in the figure, the sensory nuclei are located lateral and the motor nuclei medial to the sulcus limitans. This reflects a continuation of the dorsal–ventral arrangement seen in the spinal cord as the cord "opens up," forming the brainstem. Abbreviations: DMNV, dorsal motor nucleus of the trigeminal nerve; HN, hypoglossal nucleus; IVN, inferior vestibular nucleus; NA, nucleus ambiguus; MVN, medial vestibular nucleus; SL, sulcus limitans; SN5, spinal nucleus of the trigeminal nerve; Sol.N, solitary nucleus. Brain image was adapted from the *Interactive Brain Atlas* (1994), courtesy of the University of Washington.

occupying the more intermediate positions. There is one notable exception: the special visceral efferent nuclei, for example, the facial nucleus and the nucleus ambiguus have "migrated" to occupy a more ventral and slightly lateral position in the tegmentum of the medulla.

Finally, before proceeding to a more detailed discussion of the individual cranial nerves, it may be useful to commit the names of cranial nerves to memory. One classic mnemonic phrase for recalling the names of the cranial nerves is as follows:

"**O**n	**O**ld	**O**lympus's	**T**owering	**T**ops	**A**	**F**air	**V**irtuous	**G**irl	**V**ends	**S**nowy	**H**ops"
1	2	3	4	5	6	7	8	9	10	11	12

1. **O**lfactory
2. **O**ptic
3. **O**culomotor
4. **T**rochlear
5. **T**rigeminal
6. **A**bducens
7. **F**acial
8. **V**estibulocochlear
9. **G**lossopharyngeal
10. **V**agus
11. **S**pinal accessory
12. **H**ypoglossal

Table 5–1 summarizes the major functions mediated by these nerves. **Note:** The spinal accessory nerve is now more commonly known simply as the accessory nerve. Unfortunately, I am not aware of any mnemonic for remembering all the functions of each, that is simply going to require some good old memorization.

Table 5–1. Summary of Cranial Nerves

Nerve	*Nuclei of Origin or Termination*	*Function*	*Deficit*
I Olfactory	SVA Medial temporal, medial frontal cortical areas	Smell	Unilat. anosmia
II Optic	SSA Lat geniculates	Vision	Visual loss (nerve) Field cut (tract) ↓ Optic reflexes
III Oculomotor	GSE Oculomotor n.	Eye movements (Sup Inf & Med rectus, and Inf oblique);	Diff. looking up, down, medially Diplopia
		Elevate lid	Ptosis
	GVE Edinger–Westphal	Constrict pupil; Adjust lens.	Dilated pupil Blurred near vision
IV Trochlear	GSE Trochlear n.	Eye movement (Sup oblique)	Diff. looking down when adducted
V Trigeminal	GSA Main sensory n.	Discrimination Proprioception	↓ Discrim. and prop. of facial m.
	GSA Spinal sensory	Touch, pain & temperature; face, cornea, teeth, tongue (ant. 2/3rds)	↓ Touch, pain & temperature on face & tongue; ↓ Corneal reflex; Neuralgia
	GSA Mesencephalic n.	Proprioception: (jaw muscles, eye movements)	↓ Jaw jerk; loss of sensory limb of reflex arc
	SVE Motor n. of V	Muscles of mastication, tensor tympani	Jaw weak, (deviates to side of lesion) ↓ Jaw jerk; (motor limb)
VI Abducens	GSE Abducens n.	Abducts the eye (Lateral rectus)	Nerve: ↓ Abduction; Diplopia Nucl: Lateral gaze palsy; Diplopia
VII Facial	SVE Facial n.	Facial expression; Closes eyelid Stapedius m.	Facial weakness; Diff. closing eyelid Hyperacousis
	GVE Sup salivatory	Salivary and lacrimal glands	Dry eye
	SVA Solitary n.	Taste (anterior 2/3 of tongue)	↓ Taste (anterior tongue)
	GSA Spinal n. of V	Tactile (ear)	
VIII Vestibulo-cochlear	SSA Vestibular	Equilibrium	Vertigo, dizziness
	SSA Cochlear	Hearing	Loss of hearing, Tinnitus

(Continued)

Table 5–1. *(Continued)*

Nerve	*Nuclei of Origin or Termination*	*Function*	*Deficit*
IX Glossopharyngeal	SVE N. ambiguus	M. of pharynx, stylopharyngeus	(see CN X)
	GVE Inf Salivatory	Parotid gland	
	SVA Solitary n. (rostral)	Taste, post. 1/3 of tongue	Taste on (back of tongue)
	GVA Solitary n. (caudal)	Visceral sensation: Carotid body, carotid sinus	
	GSA Spinal n. of V	Tactile sensation: Posterior tongue, upper pharynx, middle ear	Afferent side of gag reflex
X Vagus	SVE N. ambiguus	Muscles of soft palate, pharynx & larynx	Hoarseness, Dysphagia (mild) ↓ Gag reflex ↓ Elev. of palate
	GVE Dorsal Motor n.	Abdominal & thoracic viscera	Parasympathetic control
	GSA Spinal n. of V	Tactile: ear, pharynx	
	SVA Solitary n.	Taste: epiglottis	
	GVA Solitary n.	Sensations from viscera, larynx, trachea	
XI Accessory	SVE Accessory n.	Sternocleidomastoid, trapezius	Turning head against resistance Shrug shoulder
XII Hypoglossal	GSE Hypoglossal n.	M. of tongue	Atrophy, weakness of tongue

Notes: GSA connections are presumed to be present in III, IV, VI, XI, and XII for proprioceptive feedback, but pathways not established.

Not all authors agree on all designations and assignments presented above, but the data presented in the table appear most representative.

Clinically, IX and X are difficult to separate; hence are often combined, e.g., IX mediating the sensory & X the motor side of the gag reflex.

CRANIAL NERVE I (OLFACTORY)

Major Function: Sense of Smell
Classification: Special Visceral Afferent (SVA)

The olfactory nerve (CN I) is somewhat unique in several respects. First, like the optic nerve (CN II), it does not have a nucleus within the brainstem. In fact, for this reason it has been suggested that perhaps it is not truly a "cranial nerve," but simply represents a fiber tract of the brain. Although other sensory systems may respond to chemical stimulation or biochemical changes within the body, olfaction along with taste represent the two major sensory systems through which we are consciously informed about our environment via

chemoreceptors. Unlike the other sensory systems that project first to the thalamus and then to the cortex, the nerve fibers carrying olfactory information project initially and primarily to some of the more primitive parts of the limbic system in the basal frontal and mesial temporal cortex.

It is these rather direct connections with the limbic system that would appear to account for another somewhat unique feature of the olfactory system, namely, that *emotional or affective valences are often associated with the sense of smell.* Any stimulus, regardless of the modality of input, can elicit certain affectively charged responses through learning or experience. However, the senses of smell and to some extent taste have a more direct innate association with emotion and affective responses. Furthermore, these associations very commonly involve behaviors that are basic to survival, either of the individual or the species, for example, feeding, procreation, or defense. Examples range from the feeding frenzy that can be elicited by the smell of blood in the water by the shark, to the panic of herding animals elicited by the smell of a mountain lion, to the marking of territorial boundaries with urine by the wolf. Probably more common to our experience is the "sexual frenzy" witnessed in a male dog when there is a female dog in heat anywhere in the neighborhood. In comparison to most of our mammalian ancestors, humans have a poorly developed sense of smell, although we too evidence a close association between smell and strong, primitive drive states. Regardless of past personal experiences, almost everyone is repelled by putrid odors and attracted to the fragrance of the rose or a freshly baked apple pie. Certain odors, both manufactured and natural, manifest their ability to arouse sexual feelings. In order to have maximal survival value, it is important that such affective valences, be they positive or negative, be well retained in memory. Most of us likely have had the experience of encountering an odor that immediately triggers a host of long-forgotten memories, typically affectively tinged and usually in a manner that is quite distinct from the type of memory elicited by other stimulus modalities. Again, it probably is no coincidence that the limbic system is very closely and intimately connected with the mesial temporal and other areas so critical for memory. We will revisit these topics in Chapter 8, which covers the limbic system, but this brief preview may help explain the connections of the olfactory system to which we will now proceed.

Anatomy

The end organs of the olfactory system are the epithelial cells in the upper part of the nasal mucosa. The primary neurons of these epithelial cells pass through the cribriform plate of the ethmoid bone to the ipsilateral olfactory bulb where they make synaptic connections with second-order neurons (Figure 5–3). The two olfactory bulbs and the anterior portion of the olfactory tracts lie parallel to each other on the basal–medial aspect of the frontal lobes. The nerve cells in the bulbs (primarily mitral cells) give rise to the olfactory tracts. The positioning of the bulbs (and their tracts) and their connection with the cells of the nasal cavity makes this system particularly sensitive to certain types of tumors and trauma as will be discussed below.

As the olfactory tracts proceed posteriorly, one smaller pathway branches off the main olfactory tract, sending fibers via interneurons to the opposite bulb. The main part of the tract then proceeds as the **lateral olfactory stria** (Figure 5–4a).[2] The fibers of the lateral olfactory stria terminate in the anteromedial portion of the temporal lobe, an area that in general is referred to as the primary olfactory cortex. While there are some inconsistencies among different authors as to the exact region(s) that receives these direct inputs, the basal frontal cortex in the region of the anterior perforated substance, corticomedial portions of the amygdaloid complex, the prepyriform and pyriform cortex, and the more rostral portions of the entorhinal cortex most commonly are cited (Figure 5–4b).

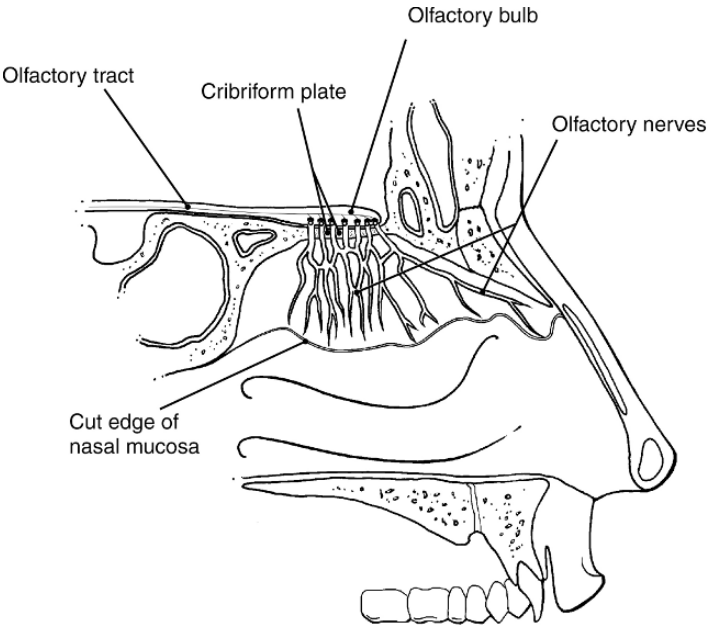

Figure 5–3. "Primary" (nerves) and "secondary" (tract) olfactory neurons synapsing in the bulb.

What is important to remember is that from these sites of primary projection substantial secondary connections are made with other limbic structures in the immediate area, including the lateral portions of the amygdala and hypothalamus (via the medial forebrain bundle). Additional indirect connections also are made with the thalamus, brainstem, and hippocampal formation. Thus, as was suggested above, the olfactory system is intimately connected with some of the more primitive brain structures, which in turn are involved in a variety of emotionally charged or affectively driven behaviors that are often critical for survival.

Because of the location of the olfactory bulbs at the base of the orbital frontal cortex the sense of smell is particularly vulnerable to lesions affecting the base of the frontal lobes. One type of lesion in particular deserves special mention: meningiomas. These very slow-growing tumors often originate from the falx cerebri (the dura lying between the two cerebral hemispheres) or from the base of the skull in the basal frontal regions and may reach considerable size before being detected clinically. However, as they begin to gradually enlarge they may impinge on one of the olfactory bulbs and/or tracts, disrupting their function. Thus, unilateral loss of the sense of smell, without other peripheral explanations (e.g., clogged nasal passage), may be the first clinical manifestation of such a lesion. Aneurysms in the area of the anterior communicating artery similarly could produce such a mass effect. While bleeds in this area also might compromise the olfactory system, other symptoms typically would predominate such as headache and possible mental status changes. Closed-head injury (CHI), particularly from decelerating types of injuries, also can result in anosmia (loss of smell). In this case, the mechanics of the pathology is different. As the brain continues to move forward relative to the skull, the nasal nerve fibers penetrating the cribriform on their way to the olfactory bulb may suffer the shearing effect of this relative motion. Contusions of orbitofrontal cortex also may occur following CHI, resulting in various other clinical syndromes, including behavioral disinhibition and seizures.

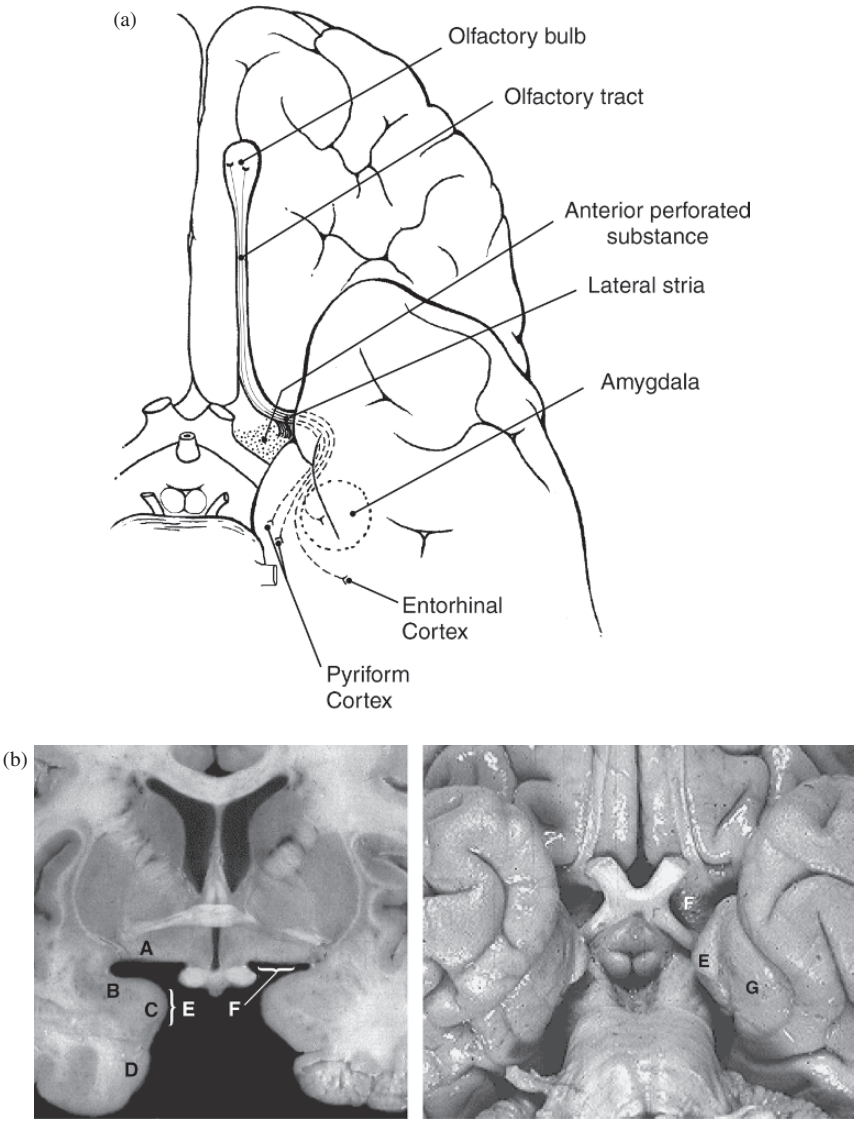

Figure 5–4. (a) Projections of olfactory tract to "olfactory cortices." (b) Primary olfactory projection areas. Abbreviations: A, basal nuclei; B, corticomedial amygdaloid nuclei; C, pyriform cortex; D, entorhinal cortex; E, uncus; F, anterior perforated substance; G, parahippocampal gyrus. **Note**: The basal nuclei, pyriform cortex, and entorhinal cortex, respectively, underlie anterior perforated substance and the uncus, which are essentially surface features. The entorhinal cortex is contained within the parahippocampal gyrus. Brain images were adapted from the *Interactive Brain Atlas* (1994), courtesy of the University of Washington.

Testing for Dysfunction

If it were not for the perception of taste, the assessment of the integrity of the olfactory nerve undoubtedly would be the most frequently neglected aspect of most routine neurological exams. The exam itself is simple enough; the problem arises (as with the assessment of taste) in the easy accessibility of adequate stimuli. A few years back when the "Scratch and Sniff" stickers were a big hit with kids, a ready and convenient supply of "odors" was easy to

obtain. Some neurologists may carry small vials of aromatic substances and routinely assess the patient's sense of smell, but often this portion of the examination is omitted. Systematic examination for anosmia is more likely to occur when specific orbital frontal lesions are suspected. Testing typically entails having the patient smell a variety of easily identifiable substances or essences. Astringent substances such as ammonia, vinegar, or rubbing alcohol should be avoided as these tend to stimulate the trigeminal (CN V) nerve, rather than the olfactory (CN I). Each nostril should be tested independently, since as was noted above in the case of mass lesions only one bulb or tract may be affected. The fact that the patient may not be able to name the substance is not necessarily a pathological sign (he or she may not be quite able to place the smell or may have an aphasic or naming problem). What may be more critical to determine is whether the substance can be identified as having a distinct aroma or smell and differentiated from other substances. Before a diagnosis is made of either a unilateral or bilateral deficit, care should be taken to note whether the patient's nostrils are clear and uncongested.[3]

CRANIAL NERVE II (OPTIC)

Major Function: Sense of Vision
Classification: Special Somatic Afferent (SSA)

If to the poet, the eyes are "windows to the soul," then to the neurologist they also might be thought of as "windows to the nervous system." Through an examination of the eyes, it is possible to assess the integrity of numerous portions of the central and peripheral nervous system. This includes cortical and subcortical sites and pathways, portions of the brainstem and cerebellum, peripheral (cranial) nerves, and the autonomic nervous system, including spinal components (e.g., sympathetic fibers from the superior cervical ganglion that are responsible for dilating the pupil and assist in elevating the eyelid). While cranial nerve II relates only to vision and the afferent link of certain visual reflexes (e.g., blink, light, tracking, and accommodation), the visual pathways extend from the eyes to the thalamus (with some branching to the mesencephalon) and eventually to the posterior cortex. This extensive network creates ample opportunity for a variety of cortical or subcortical lesions to affect the visual system. In addition, movements of the eyes themselves are mediated by yet other parts of the CNS. These include such regions or structures as the frontal eye fields (Brodmann's area 8), cranial nerves III, IV, and VI, as well as other brainstem nuclei and pathways (e.g., the superior colliculi, vestibular nuclei, vestibulocerebellar pathways, the paramedian pontine reticular formation, and the medial longitudinal fasciculus). Cranial nerve V and VII likewise are involved in the tactile afferent and motoric aspects of blinking, respectively. Parasympathetic fibers of cranial nerve III (via the Edinger–Westphal nuclei) are responsible for the constriction of the pupil, while the sympathetic fibers tend to dilate it. Cranial nerve III (with contributions from the sympathetic system) elevates the eyelids, while cranial nerve VII closes the eyelids. Finally, other structures of the midbrain, including the pretectal areas and superior colliculi, also are involved in some of the above-mentioned visual reflexes. In this section, however, the primary emphasis will be on the optic nerves themselves, their pathways, and their influence on vision and certain visual reflexes. Eye movements and other related ocular phenomena (including control of the eyelids) will be covered in greater detail as the relevant cranial nerves are discussed.

Anatomy

As with the olfactory nerve, it has been suggested that the optic nerve perhaps should not be considered truly a nerve, but rather a fiber tract of the brain. However, as with the former,

convention usually prevails and it will most likely continue to be referred to as cranial nerve II. The end organs for the optic nerve are the photoreceptive cells of the retina, the rods, and cones. The **rods**, which are found with relative greater frequency in the peripheral parts of the retina, are better adapted to low-intensity light, such as night vision. The **cones**, which tend to be concentrated in the **macula,** particularly in the center of the macula or **fovea**, are better adapted to the perception of color and the sharper point-to-point vision necessary for fine discriminations (foveal vision). While other cells (horizontal cells) serve as interneurons, it is basically the rods and cones that transmit visual input to the bipolar cells that lie within the retina. These in turn synapse with the ganglion cells also within the retina. The axons of these ganglion cells exit the eye through the optic disk to form the optic nerve. Visualization of the optic disk, which also contains blood vessels entering and exiting the globe, is the closest one can come to directly seeing the nervous system in the intact patient without intrusive or radiographic procedures. In fact, well before the advent of the CT scan or MRI, the presence of **papilledema** (a swelling or "choking" of the optic disk) was used as a sign of increased intracranial pressure, such as might be present with mass effect lesions or inflammatory conditions of the brain.

Before proceeding to trace the optic pathways and identify specific syndromes produced by various lesions impacting on different parts of these pathways, it is essential to first understand some properties of the retinal image. As can be seen from the accompanying diagrams, as the light rays enter the pupil and fall on the retina they cross. As shown in the two figures below, it is important to remember that the light rays cross along both the horizontal and vertical planes. Visual input representing light rays striking the left half of each eye (the temporal field of the left eye and the nasal field of the right eye) come from the right external hemispace. The reverse is true of images that lie in the observer's left visual field (Figure 5–5). In addition to the reversal of the visual field from left to right along the vertical midline as the image proceeds from the visual field to the retinal field, there is a reversal of superior–inferior along the horizontal axis. That is, input from the superior portion of the external visual field projects to the inferior parts of the retina (Figure 5–6). This organization continues in this manner to the calcarine cortex, so that the right and left as well as the superior and inferior visual fields are "reversed" as they project to the visual cortex.[4]

After converging on the bipolar and then the ganglionic cells in the retina, the visual fibers exit the eye as the **optic nerve**. The two optic nerves (each of which carries information from both the nasal and temporal halves of the eye from which it emanated) proceed to the **optic chiasm**. Upon reaching the chiasm (or "crossing"), which is located above the pituitary gland along the ventral base of the frontal lobes, those fibers in each nerve that represent the visual receptors in the nasal half of each eye decussate, while the temporal fibers of each eye remain on the same side. Thus, in the chiasm, the uncrossed temporal fibers are in the lateral or outside portions of the chiasm, with the crossed nasal fibers from both eyes making up the medial or central portion of the chiasm. This is important to keep in mind when considering the possible effects of lesions affecting the chiasm itself. Because of their position at the base of the frontal lobes, the optic nerves, the optic chiasm, and the optic tracts are particularly vulnerable to encroachment by meningiomas or pituitary tumors growing in this region.

Upon exiting the chiasm, these postganglionic visual pathways are now referred to as the **optic tracts**. However, after the partial crossing that took place in the chiasm, each optic tract now consists of fibers originating from both eyes. The left optic tract is composed of the temporal fibers of the left eye and the nasal fibers of the right eye, while the right optic tract is formed from the nerve processes emanating from the temporal half of the right eye and the nasal half of the left eye. This arrangement basically continues throughout the remainder of the visual pathways all the way to the cortex. This organization enables the

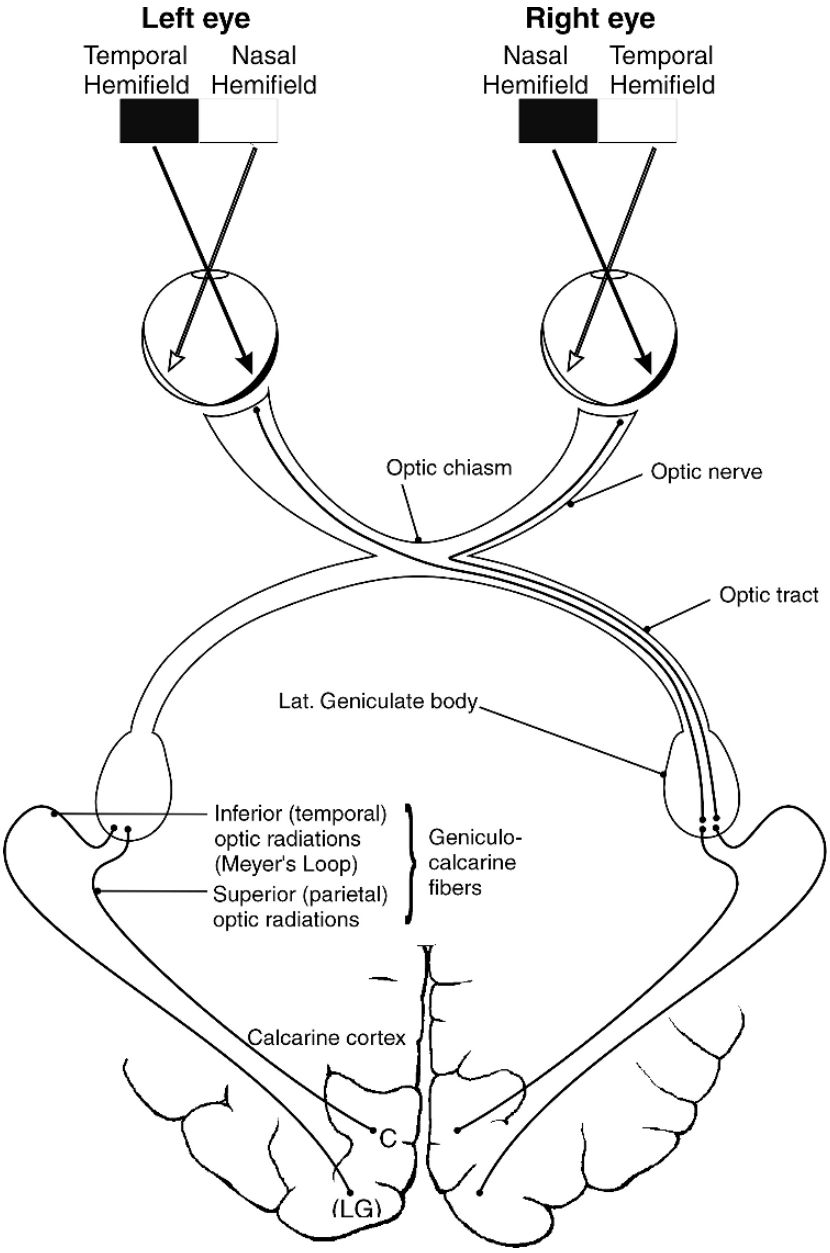

Figure 5–5. Left visual field projections.

right side of the brain to initially process information coming in from the left visual field and the left brain to respond to right visual field stimuli.

The bulk of the fibers of the optic tracts then proceed to the **lateral geniculate nuclei** of the thalamus (located just dorsal and lateral to the rostral midbrain) where they are arranged in a very precise, retinotopic manner. A few collateral fibers exit from the optic tract shortly before the lateral geniculates and proceed to the pretectal area and to the superior colliculi. The former (pretectal) connections, which synapse with the

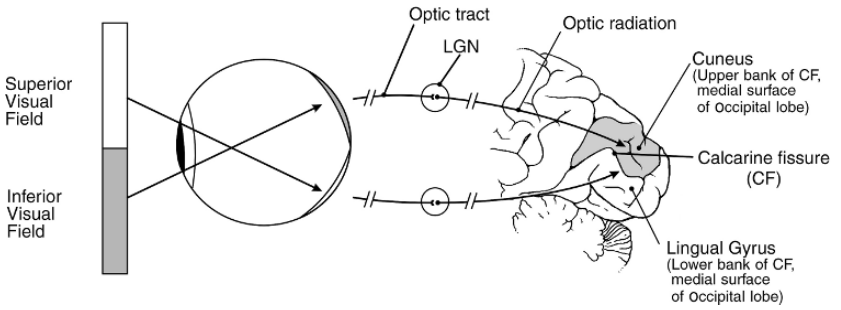

Figure 5–6. Superior–Inferior visual field projections.

Edinger–Westphal nuclei, are thought to be important for simple and consensual pupillary reflexes to light, and the latter (to the superior colliculi) for oculoskeletal reflexes (e.g., orienting responses to visual or auditory stimuli) and visual tracking, especially for novel stimuli. Additional visual reflexes such as fixating on a moving object (or stationary object if the subject is moving), convergence, accommodation, or pupillary changes to psychologically meaningful stimuli, likely are in large part mediated by pathways originating in the occipital cortex.

The lateral geniculate nuclei in turn give rise to the **optic radiations** or *geniculocalarine tracts* that project to the calcarine cortex (Brodmann's area 17) in the medial aspect of the occipital poles. It is here in the calcarine cortex that the visual stimuli begin to be processed into conscious perceptions (additional discussions on the topic of visual perception can be found in Chapter 9, this volume). As the fiber tracts that make up the optic radiations leave the lateral geniculate nuclei via the retrolenticular and sublenticular portions of the internal capsule, they become organized into superior (parietal) and inferior (temporal) pathways. The inferior fibers loop forward into the temporal lobe (Meyer's loop) before turning back toward their destination in the occipital cortex. This is particularly noticeable in those that proceed from the more lateral aspects of the nucleus. As the optic radiations fan out, these latter fibers maintain a more ventral position and synapse in the more ventral portion of the occipital cortex: the lingual gyrus that lies below the calcarine fissure. The fibers from the more medial aspects of the lateral geniculate nuclei maintain a more dorsal position, ending up in the cuneus, which is the gyrus above the calcarine fissure. These more dorsal fibers (to the cuneus in area 17) consist of third-order fibers whose antecedents originated in the dorsal halves of each retina, with the ventral fibers (and lingual gyrus) being related to the ventral parts of each retina. As a consequence of this arrangement, a lesion that might affect, for example, the more temporal–inferior (ventral) portions of the left optic radiations (or the left lingual gyrus) will produce a right superior quadrant defect (right upper quadrant field cut). Conversely, a more dorsal lesion of the superior optic radiations (e.g., deep parietal) or one involving the cuneus will result in a inferior field cut or quadrantanopia on the side opposite the lesion.

Testing for Dysfunction

Testing the integrity of the optic nerve and its central pathways involves three major areas of inquiry: (1) visual acuity or perception, (2) examination of the patient's visual fields, and (3) assessing the integrity of light reflexes. **Reduced visual acuity** can result from damage to the visual pathways, but caution must be observed as it often results from peripheral problems in the eye itself, including diseases affecting the retina, lens, cornea, or vitreous humor, or may simply reflect a refractory problem. Problems of visual perception, that is,

not simply the ability to see but the ability to "recognize" what one sees (visual agnosias) typically do not result from lesions of the primary visual pathways. Rather, problems of **visual perception** generally are thought to result from lesions affecting the visual association cortices or a disconnection of the primary visual cortical areas from these association areas (see Chapter 9). In addition to their possible direct diagnostic significance, a gross assessment of basic acuity and perception (as well as visual neglect: see below) should be an integral part of any comprehensive mental status examination, since these examinations often require attention to and interpretation of visually presented stimuli.

Assessment of the patient's visual fields is a standard and important part of both neurological and neuropsychological examinations. However, before discussing visual field exams, two additional diagnostic complications should be considered. On occasion, patients may report to be "blind" or experience "tunnel vision" when in fact their vision is neurologically intact (e.g., conversion reaction). Demonstrating the presence of optokinetic nystagmus may support a diagnosis of hysterical blindness. Under proper conditions, individuals with intact vision reflexively will respond to a vertically striped stimulus passed in front of them. In these circumstances, the eyes will slowly track in the direction that the stimulus is moving and then alternately quickly shift in the opposite direction. The ability of the patient to adequately navigate their environment suggests the absence of significant tunnel vision on a neurological basis. An opposite problem is that patients can report normal vision when in fact they are totally or nearly totally blind (**Anton's syndrome**). This syndrome is most commonly the result of vascular lesions involving the calcarine cortex bilaterally (basilar artery or both posterior cerebral arteries), although it can result from other etiologies. Since the denial of cortical blindness often is associated with other cognitive deficits, including disturbances of memory, disorientation, or mental confusion, extension of the lesion to the medial temporal cortices and/or the thalamus is suspected in most cases. The presence of cortical blindness with denial of visual loss typically is easily established by asking the patient to identify specific stimuli clearly within his or her visual field.

A gross but clinically useful and fairly reliable assessment of visual fields can be accomplished via a routine confrontation examination. Initially, this can be done with both eyes open, but eventually each eye should be tested individually. There are several methods by which this is done; however, they generally all involve having the patient fixate on the nose of the examiner while the right, left, or bilateral peripheral fields are randomly stimulated. The upper, lower, and middle fields are tested in this manner while the examiner notes any tendency to ignore, suppress, or otherwise fail to report stimuli presented to a particular field. It should be noted whether failure to report such stimuli occurs only under conditions of bilateral simultaneous stimulation (suppression) or consistently even under conditions of unilateral stimulation (suggesting a true field cut). In the latter instance, the examiner may change the instructions, telling the patient that the stimulus *will* occur in the affected field, the patient's job being to report not where but *when* the stimulus occurs. If performance improves under the latter situation (assuming that movements of the eye cannot account for the enhanced reporting), it may suggest that severe neglect rather than a complete visual field cut may be present.

The accompanying diagrams (Figure 5–7) illustrate the visual field defects associated with lesions to different sites along the visual pathways.

The localization of pathology with regard to visual field cuts as illustrated in Figure 5–7, can be broadly summarized as follows:

1. Lesions of the **optic nerve** will result in blindness in that eye. Because no visual input can reach the pretectal areas from that eye (the afferent limb of the visual reflex), there will be both a loss of direct light response to that eye as well as a consensual

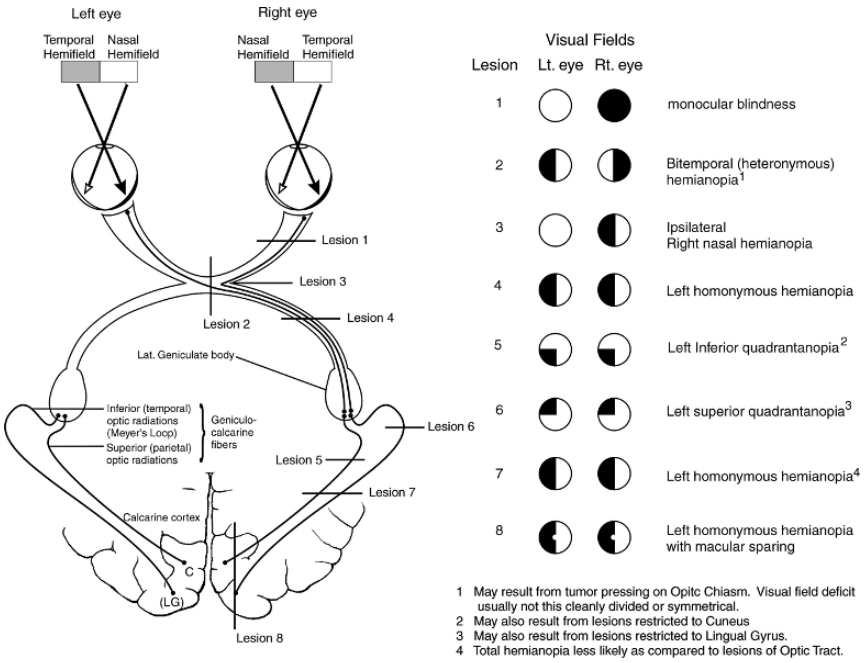

Figure 5–7. Lesions of visual system.

reflex response in the other eye. However, since the efferent limb of the reflex is unaffected, a consensual pupillary response should be present from stimulation of the intact eye and from accommodation (the convergence and pupillary constriction that takes place when attempting to focus on a near object).

2. A **bitemporal hemianopia** can result from a pituitary tumor impinging upon the medial aspect of the chiasm and/or optic tracts that more or less selectively disrupts the crossing fibers from the nasal portions of both retinas.[5]
3. A **homonymous hemianopia** (loss of comparable halves of the visual fields of both eyes) can result from either a lesion of one of the optic tracts, a complete disruption of one of the lateral geniculates or optic radiations, or a complete lesion of one of the occipital poles (area 17). In the latter case, macular sparing may be observed. If the lesion is anterior to the geniculates (e.g., optic tract), the pupillary response to direct visual stimulation (and consensual response in the opposite eye) may be absent if the light source to the eye is limited to the affected field. However, in practice it is exceedingly difficult to reliably maintain such control over the area of the retina being stimulated. In postgeniculate lesions, the pupillary responses will be intact, since the fibers mediating the affective limb of the pupillary response already have left the tract on their way to the pretectal area.
4. A **quadrantic field cut** (*inferior or superior homonymous quadrantanopia* or other partial field cuts) may result from:

   (a) An incomplete lesion of the optic radiations (e.g., deep parietal or temporal lesions producing inferior and superior field cuts, respectively).
   (b) Lesions restricted to the superior (*cuneus*) or inferior (*lingual gyrus*) bank of the calcarine fissure of the occipital pole (producing inferior and superior visual field defects respectively).

Visual Reflexes

As with all reflexes, the visual reflexes require both afferent and efferent loops. However, there are a variety of reflexes involving the eyes that utilize various sensory and motor pathways. The afferent component of the light reflex is carried by the optic nerve (CN II). The motor response—a diminution or constriction of the pupil to excessive light—is carried out by the parasympathetic fibers of cranial nerve III. The opposite response— an enlargement or dilation of the pupils in dim light—is the function of the superior cervical ganglion of the sympathetic nervous system. As noted above, fibers mediating the light reflex travel with the optic tract and detour to the pretectal area at the level of the lateral geniculate body. The pretectal area is just rostral to the superior colliculi. Connections are made with the contralateral pretectal area via the posterior commissure. From the pretectal area the Edinger–Westphal nuclei of the oculomotor nuclear complex then receives both ipsilateral and contralateral inputs (which is why shining a light in one eye will normally cause both pupils to react). These nuclei then send out parasympathetic fibers to the muscles that constrict the pupil as well as adjust the diameter of the lens. The pathway just described is active in the reflex pupillary constriction to light presented to the retina.

Opposing (and "balancing") the parasympathetic constrictor actions of cranial nerve III are the sympathetic inputs from the cord. These sympathetic inputs dilate the pupil (and also produce a tonic elevation of the eyelid). However, the latter response is more complex and can involve more than one afferent pathway. The sympathetic action typically predominates when there is minimal light striking the retina; in this instance, the afferent portion of the reflex arc is carried by the optic nerve. However, pupillary dilation also is known to occur in situations of perceived threat to the organism, in response to pain or any intense sudden and unexpected stimulation, and even in response to visual stimuli that elicit strong, positive affective responses. In the latter cases, the afferent connections mediating the reflex action are more extensive, likely involving the cortex, limbic system, and other brainstem structures. When lesions affect this latter sympathetic system, a **Horner's syndrome** may be present that includes:

1. Constricted (miotic) pupil on the affected side (resulting from the unopposed action of the parasympathetic system).
2. Mild ptosis that can be voluntarily overcome.
3. Dry (anhidrosis), warm (vasodilatation) face on the affected side.

In examining the light reflex, the basic procedure is to have the patient in a dimly lit room and to shine a light in one eye at a time. The examiner notes whether there is a direct (in the eye being stimulated) and a consensual (in the opposite eye) pupillary constriction. Normally, both should be present. If a direct response is absent but a consensual response is present, this would suggest that the afferent side of the reflex is intact but the efferent side is not functioning. A special syndrome—the **Argyll Robertson pupil**—produces dissociation between pupillary constriction from the light reflex and pupillary constriction with accommodation. In this syndrome, the pupil normally is somewhat constricted but fails to constrict further in response to light, either directly or consensually. It also typically fails to dilate in response to darkness. It also fails to respond to mydriatic (pupillodilator) drugs. However, the pupil does show the normal pattern of constriction with accommodation (see below for additional detail). The most common etiology for this syndrome is neurosyphilis and the critical lesion is thought to affect the more dorsal pathways between the pretectal area and the Edinger–Westphal nucleus.

A second afferent pathway, involving corticotectal connections (probably mostly from the occipital cortices), also is involved in pupillary constriction. This second reflex arc (for

near vision) uses the same effector system for pupillary constriction to light. In this case, however, it occurs when the occipital cortex recognizes that the visual image is out of focus (e.g., when an object is presented close to the eyes). The subsequent adjustment made by the eyes is called **accommodation**. Impulses are sent again to the pretectal area. From there, connections are again made with the Edinger–Westphal nucleus of cranial nerve III. The fibers from the pretectal area occupy a slightly more ventral position than those mediating the light reflex. This difference in the position of the pretectal-Edinger–Westphal connections is what makes the Argyll Robertson pupil possible. The connections from the Edinger–Westphal nuclei to the sphincter muscles of the iris are identical for both the light reflex and the changes associated with accommodation. In accommodation, three changes take place simultaneously: (1) the lens is thickened by changes in tension of the ciliary muscles, (2) the eyes converge (via the medial rectus muscles of both eyes to prevent double vision), and (3) the pupils constrict. While this process takes place voluntarily when we choose to focus on a near object, it also occurs reflexively as an object is drawn toward the eyes.

If an object is quickly propelled toward the eye such that it might threaten the integrity of the eye itself, another reflex takes over. Instead of accommodation, the eyelids reflexively close. Here the efferent limb of the reflex arc is cranial nerve VII. This same effector response also can be brought about by other stimuli, including loud noise (CN VIII), physical contact with the cornea (CN V), or excessively bright light (CN II). The presence of recurrent stimuli passing across one's visual field with a critical frequency (CN II) can result in involuntary tracking motions of the eyes or optokinetic nystagmus (CN III, IV, and VI). Other automatic or reflex adjustments in the movement of the eyes also can take place given certain types of stimulation to the vestibular system (CN VIII). The external muscles of the eyes and cranial nerve VIII are linked in another reflex response: head (CN XI) and eye (CN III and VI) turning in reaction to a loud, unexpected sound (CN VIII). There are additional reflex changes that affect the intraocular and external muscles of the eye, but the above reflexes are the most common and provide an appreciation of the complex interactions that take place among various systems.

THE EXTRAOCULAR MUSCLES: CONTROL OF EYE MOVEMENTS

Before discussing cranial nerves III, IV, and VI, a brief understanding of the anatomy and functions of the extraocular muscles as a group might be helpful. There are six muscles controlling the movements of each eye. These are the **lateral rectus**, **medial rectus**, **superior rectus**, **inferior rectus**, **superior oblique**, and the **inferior oblique**. The lateral rectus is controlled by cranial nerve VI, the superior oblique by cranial nerve IV, and cranial nerve III controls the superior, medial, and inferior rectus and the inferior oblique. The nerves mediating the superior oblique (CN IV) and that portion of cranial nerve III that supplies the superior rectus cross the midline from their respective nuclei, whereas the inferior rectus, medial rectus, and inferior oblique (CN III) and the lateral rectus (CN VI) are all innervated by ipsilateral nuclei.

Actions of the Lateral and Medial Rectus Muscles

Two of the muscles—the lateral rectus and the medial rectus—have only a single action or effect on the eye: they rotate the eye around the vertical axis (Figure 5–8). The lateral rectus inserts on the temporal or lateral side of each eyeball and, when contracted, it rotates the eye outward or abducts the eye (abducens nerve abducts). The medial rectus inserts on the medial aspect of each eye and, when contracted, it turns the eye toward the midline or adducts the eye. The remaining muscles all have more than one potential or effective impact on eye

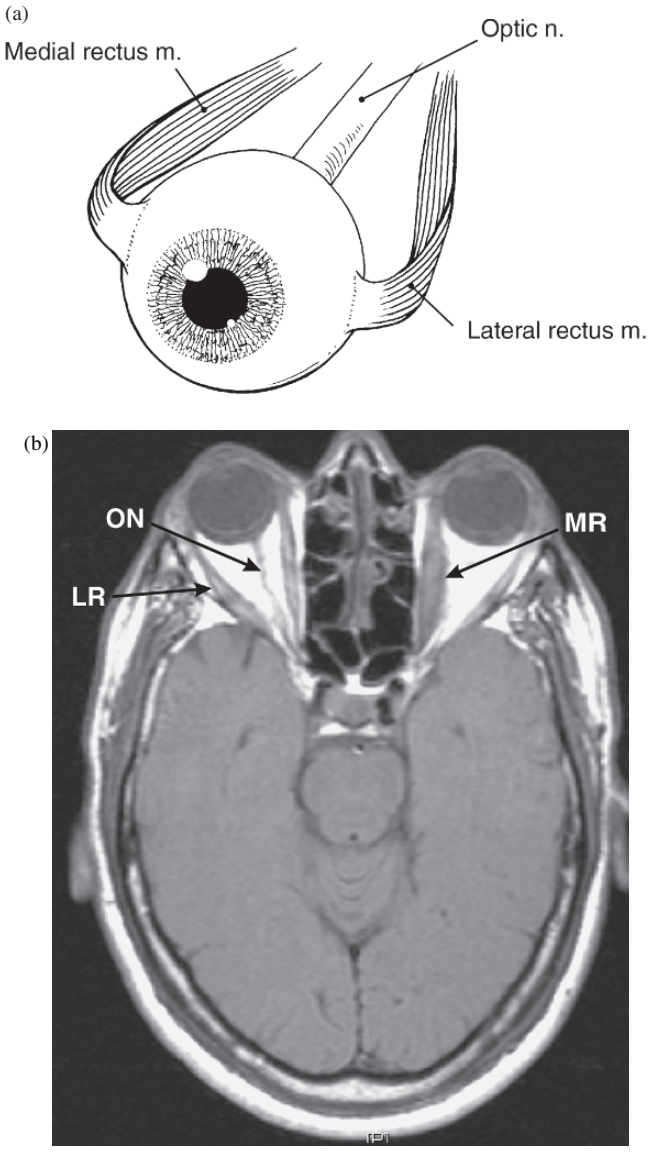

Figure 5–8. (a) Muscles for horizontal movements of the eye. (b) Lateral (LR) and medial (MR) recti muscles, and optic nerve (ON) as seen on MRI.

movement. Thus, these latter muscles have a primary as well as a secondary and a tertiary action. This is a result of the fact that the point of insertion of the remaining muscles are all slightly off center in relation to the vertical axis of the eye (Figure 5–9). The tertiary actions all involve the rotation of the eye around its anterior–posterior axis (intorsion or extorsion). But to try and keep it simple, only the primary and secondary actions will be considered here.

Actions of the Superior and Inferior Rectus Muscles

The superior and inferior rectus, which both attach toward the front of the globe from behind, are actually displaced slightly medial to the vertical axis of the eye (see accompanying

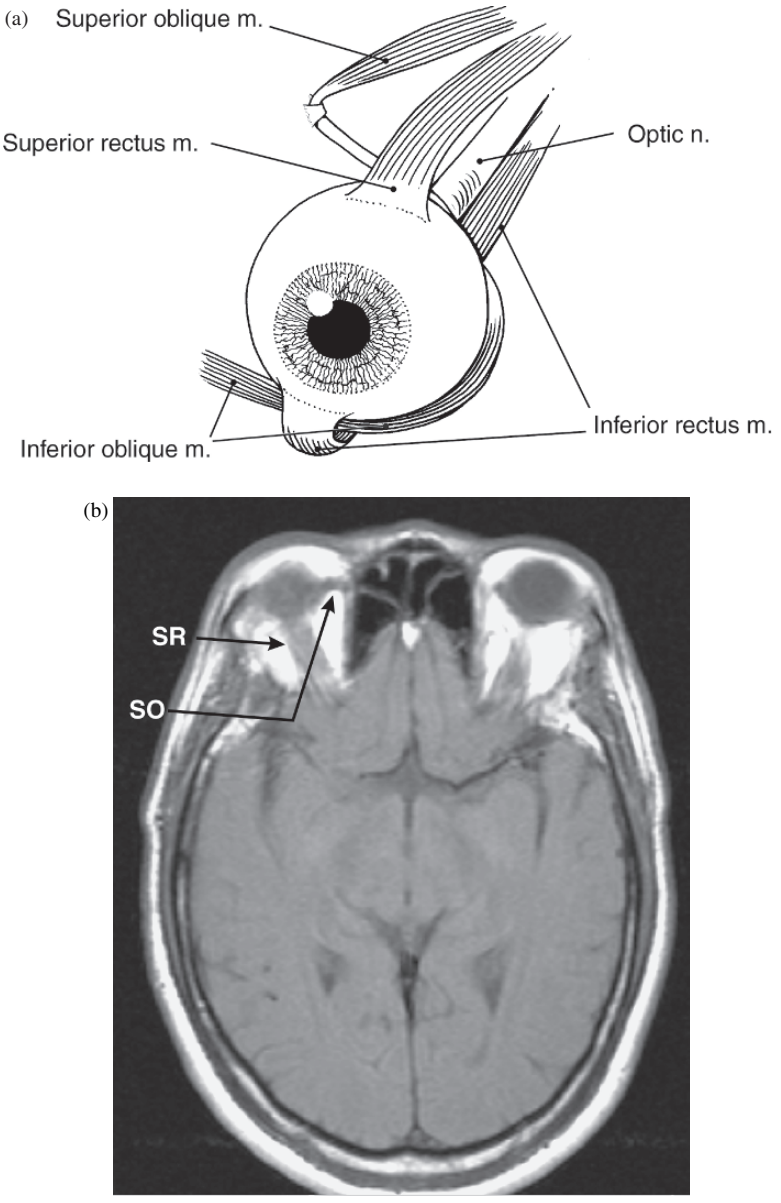

Figure 5–9. (a) Muscles for vertical and rotational movements of the eye. (b) Superior rectus (SR) and superior oblique (SO) as seen on MRI.

figures). This means that, when contracted, they tend to not only respectively elevate and depress the eye, but also tend to rotate the eye (medially) on its vertical axis. Because of this off-center attachment, the strongest elevation effect for the superior rectus will be when the eye is abducted, or looking to the outside or laterally. The same holds true for the inferior rectus.

Actions of the Superior and Inferior Oblique Muscles

Unlike the four recti muscles that pull the front of the globe up, down, or to the side from attachments behind the eye socket, the two obliques (superior and inferior) are unique in that they exert their pulling action from the back of the globe toward the front of the eye. This results from the fact that the base attachment for the obliques are either in the ventral anteromedial part of the eye socket itself (inferior oblique) or run through a pulley (*trochea*) located in the dorsal anteromedial portion of the socket. In both instances, the muscles proceed around the globe to attach more posteriorly. As is the case with the superior and inferior recti, the obliques run medially to the vertical axis of the globe. Thus when looking straight ahead, contraction of the superior oblique will tend not only to depress the eye (primary action), but also to rotate the eye outward on its vertical axis (secondary action). Thus the strongest depressing force of the superior oblique will be when the eye is turned inward or medially (adducted) when the line of force and the line of muscular attachment tend to be parallel. The same is true for the inferior obliques, except that they tend to elevate rather than depress the eye.

Summary of Actions of Extraocular Muscles

Muscle	Primary Action	Secondary Action	Maximal Effect
Medial R.	Turn eye in	(none)	
Lateral R.	Turn eye out	(none)	
Superior R.	Elevation	Adduction	Elevation when abducted
Inferior R.	Depression	Adduction	Depression when abducted
Superior O.	Depression	Abduction	Depression when adducted
Inferior O.	Elevation	Abduction	Elevation when adducted

Note: It is only when looking medially or laterally on a horizontal plane that a single muscle is involved. In looking either up or down, at least two muscles contribute to the action. Hence, from an analysis of the above it can be demonstrated that the maximal strength of each muscle (the direction patient should be told to look when testing each muscle) is as follows:

- **Lateral rectus:** looking to the outside
- **Medial rectus:** looking toward the midline
- **Superior rectus:** looking up and out
- **Inferior rectus:** looking down and out
- **Superior oblique:** looking down and toward the midline
- **Inferior oblique:** looking up and toward the midline

Conjugate Gaze and Convergence

In man, unlike certain lizards, the eyes do not move independently, but jointly. In most instances, the two eyes mirror one another: if one moves up and to the right, the other will do the same. This working together of the eyes in a complementary fashion is called **conjugate eye movement**. Thus, if patient is told to:

	Right eye uses	Left eye uses
"Look to the left"	Medial rectus	Lateral rectus
"Look up and to the left"	Inferior oblique	Superior rectus
"Look down and to the left	Superior oblique	Inferior rectus

There is one instance to which we already have alluded where the two eyes do not move in the same direction at the same time. This is when the individual needs to bring both eyes in toward the center of the visual fields to look at an object that is relatively close to the face. This is known as **convergence** (the two eyes "converge" toward the center). This action involves the simultaneous contraction of both medial recti muscles. Actually, several things take place simultaneously in this process. In addition to the convergence of the eyes, the ciliary muscles of the eyes change the shape of the lens and the pupils constrict; the two latter actions help to bring the image into sharper focus. This entire process, as we have noted earlier, is known as **accommodation**.

Although briefly reviewed in the previous chapter, it may be useful to review the basic neuroanatomical substrates of conjugate eye movements, focusing on lateral conjugate gaze, which requires the coordination of the lateral rectus muscle of one eye in concert with the

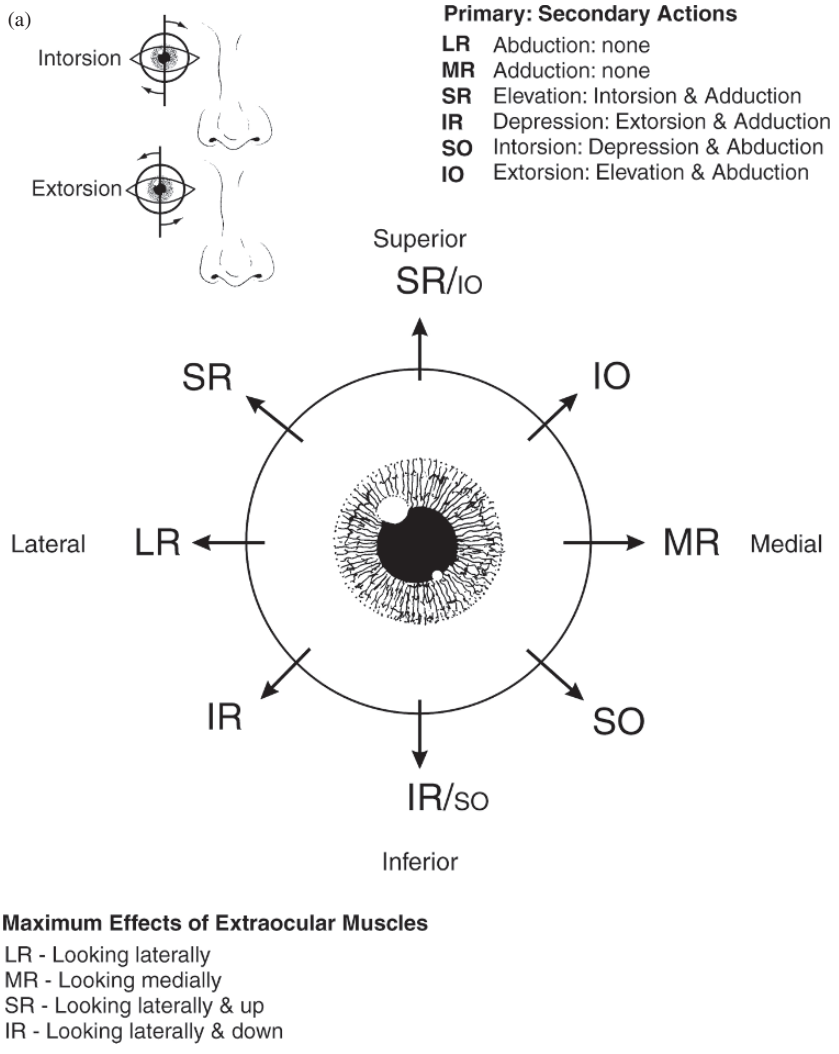

Figure 5–10. (a) Compound (primary and secondary) eye movements. (b) Directional movements of individual muscles of the eye. Abbreviations: IO, inferior oblique; IR, inferior rectus; LR, lateral rectus; MR, medial rectus; SO, superior oblique; SR, superior rectus.

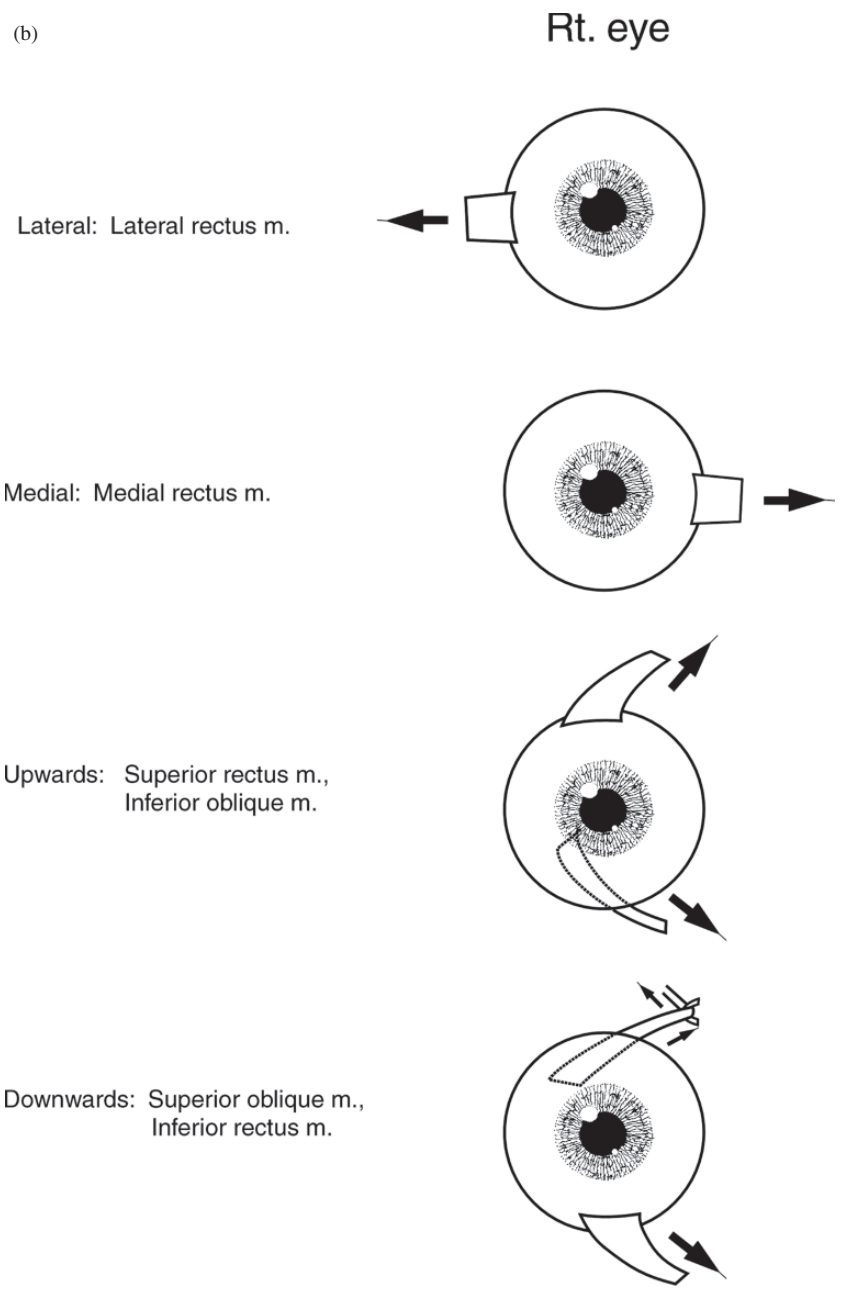

Figure 5–10. *(Continued)*

medial rectus of the other. The cortical center mediating lateral conjugate gaze is located in the posterior part of the frontal cortex (Brodmann's area 8 or the frontal eye fields). From there, fibers pass directly to the pontine brainstem where they decussate and synapse in an area adjacent to the nucleus of cranial nerve VI, which serves as the center for coordinating lateral conjugate gaze in the brainstem [the paramedian pontine reticular formation (PPRF)].

From the PPRF, connections are made between the abducens nucleus (CN VI) and the contralateral oculomotor nucleus via the medial longitudinal fasciculus (MLF).

Thus, if one wants to look to the left, the command is likely initiated in the right frontal eye fields (the right hemisphere is primarily concerned with the left hemispace). The message then is sent to the left PPRF nuclei and then to the left sixth cranial nerve nucleus. The left abducens nerve (CN VI) pulls the left eye laterally (to look to the left). From internuclear cells in the abducens nucleus, a signal also is sent to the right oculomotor nucleus via the MLF commanding the medial rectus of the right eye to contract, thus pulling the right eye toward the left for conjugate left horizontal gaze.

Before looking at the effects of lesions to this system, one must consider that the muscles of the eye act reciprocally in an agonist–antagonist fashion. When, for example, we stare straight ahead, this is accomplished in part by the equal pull or influence of the lateral and medial recti in each eye. What then might be expected to happen if one of those opposing influences were negated? Specifically what would happen, for example, if the lateral rectus muscle in the left eye were to lose its capacity to contract as a result of a lesion to cranial nerve VI or nucleus? In this case, the now unopposed pull of the medial rectus in the left eye would deviate the pupil of the left eye slightly toward the nose, while the pupil of the right eye would remain in its normal midline resting position. This deviation of one eye as a result of weakness or paralysis of one or more of the extrinsic muscles of the eye is referred to as **strabismus**, in this case medial strabismus of the left eye. As might be expected, this would tend to result in double vision (side by side), which then would be exacerbated if the patient were asked to look to his or her left.

As can be seen in Figure 5–11, if cranial nerve VI alone is affected, the patient would

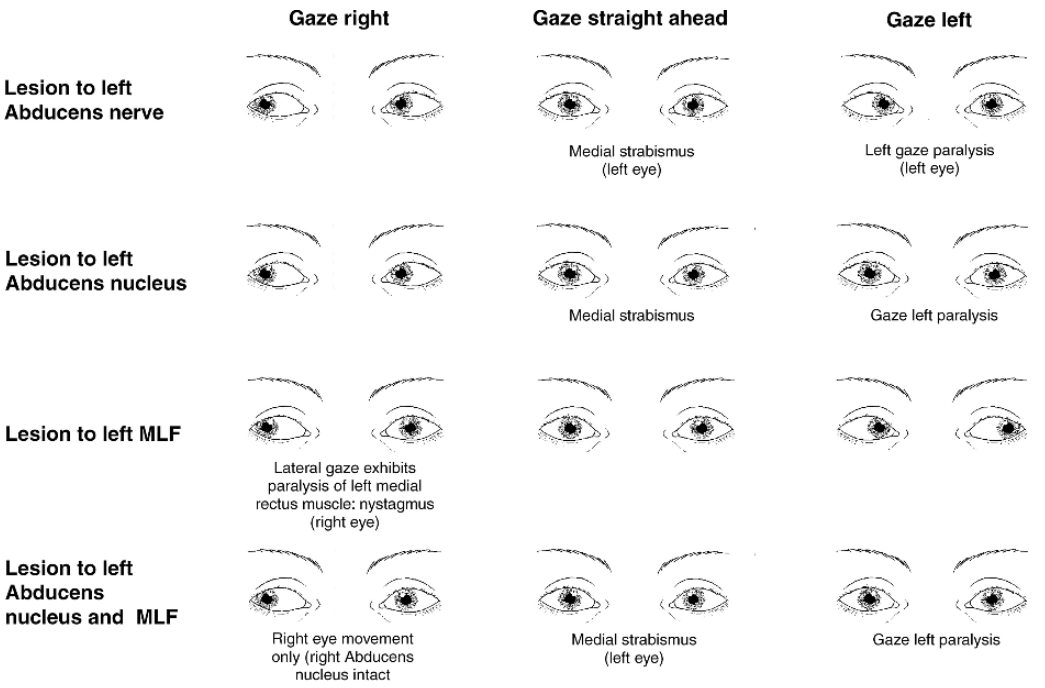

Figure 5–11. Eye deviations on clinical exam with lesions affecting various extraocular muscles. Adapted from Nolte, J (1993). *The human brain: An Introduction to its functional anatomy.* St Louis: Mosby Year Book.

be unable to abduct the eye on the side of the lesion (i.e., unable to look toward the side of the lesion with the eye on the side of the lesion). When attempting to look straight ahead, the eye on the side of the lesion would be slightly deviated toward the midline (as a result of the unopposed action of the medial rectus). However, if there is damage to the nucleus of cranial nerve VI, a slightly different pattern of deficits emerges. While strabismus is still present when the eyes are at rest (i.e., looking straight ahead), when attempting to look toward the side of the lesion not only will there be a failure of abduction, but in addition the eye opposite the lesion will not adduct. This phenomenon is referred to as **lateral gaze palsy** (to the side of the lesion). This results from the fact that not only is the lateral rectus muscle ipsilateral to the lesion affected by the lesion to the nucleus of cranial nerve VI, but the fibers from the nucleus of cranial nerve VI that cross over to supply the medial rectus of cranial nerve III via the MLF also are affected. If the lesion were to spare the nucleus of cranial nerve VI but disrupt the MLF, yet another pattern of deficit known as **internuclear ophthalmoplegia** would be found. In this case, both eyes could abduct on lateral gaze since both the nerves and nuclei of cranial nerve VI are intact. However, since the MLF represents crossed ascending fibers from the nuclei of cranial nerve VI to the oculomotor nuclei (CN III), when attempting lateral gaze, the eye on the side of lesion will not adduct (medial rectus) when attempting to look to the side opposite the lesion. Additionally, nystagmus typically will be present in the abducting (CN VI) eye. In the two latter cases there is a disruption of the function of the medial rectus muscle of one eye when attempting lateral gaze. However, during accommodation the medial rectus, which did not function on attempted lateral gaze, now performs normally, indicating the problem lies in the nuclei responsible for lateral gaze or in the MLF rather than in the oculomotor nerve or its nuclei.

Role of the Vestibular Nuclei in Vision

The vestibular nuclei also are closely associated with eye movements. When the position of the head changes or rotates in space, the eyes typically compensate by conjugate movements in the opposite direction, thus permitting the fixation of gaze to be maintained. Of course, these are reflex responses and can be overcome volitionally. Neuropathologically, the presence of these connections can offer some clinically important information. For example, if the vestibular nuclei and their connections with the centers for lateral gaze are intact, the sharp movement of the head of a coma patient to one side should elicit a conjugate movement of the eyes in the opposite direction (i.e., the eyes should keep "looking" in the same direction they were before the passive movement of the head). This is known as the **oculocephalic** (*doll's head*) **response**. If the eyes stay in a fixed position relative to the head upon passive movement of the head, this suggests a dysfunction at the pontine level (absent doll's head). Another test of related phenomenon could be accomplished by putting cold water in the patient's ear, stimulating the vestibular nerve [**oculovestibular** (*caloric*) **reflex**]. If the vestibular system and its connections with the PPRF and cranial nerve VI nucleus are intact, it should produce nystagmus with the slow component being toward the side being stimulated (see below). Finally, other disruptions of the vestibular system and its connections with the cerebellum or certain other brainstem nuclei can produce abnormal patterns of nystagmus in the conscious patient.

Cortical Control of Eye Movements

All voluntary eye muscles are ultimately under the control of the cortex, particularly Brodmann's area 8 (frontal eye fields) in the frontal lobes that work together to produce smooth pursuit eye movements. Thus lateral gaze palsy also can result from lesions to these

regions of the cortex. With unilateral destructive lesions of area 8, not only may the patient be unable to voluntarily gaze to the contralateral field, but as a result of the unopposed action of the unaffected hemisphere the eyes may tend to be slightly deviated toward the side of the lesion. Conversely, if there is an irritative focus to the lesion (e.g., seizure activity), during the phases of electrical stimulation (seizure) the eyes will be "driven" to look to the side away from the lesion.

Nystagmus

Nystagmus, a rhythmic, involuntary oscillation of the eyes, is yet another phenomenon that can be of diagnostic value. Nystagmus is normal under certain circumstances, for example, watching the fence posts go by when riding in a car at a certain rate of speed. It also can be demonstrated to a slight degree in normal persons by asking the individual to deviate the eyes markedly to one side. A more pronounced effect will be seen when caloric testing is carried out (putting cold water in the ear). Pathological nystagmus can take various forms with multiple etiologies. Perhaps most commonly it is evident as excessive nystagmus on lateral gaze or any evidence of nystagmus on upward gaze. Nystagmus is typically described as either **pendular** (where the to-and-fro motions are equivalent) or **jerk** (where they are unequal, consisting of a fast component in one direction followed by a slow component in the opposite direction). In jerk nystagmus, the fast component classifies its directionality. Pendular nystagmus suggests cerebellar involvement, but it can occur in more benign forms. Jerk nystagmus, when pathological, suggests disturbances in the cerebellum, vestibular system, MLF, or other oculomotor pathways. On the other hand, a diminished, or worse, an absent response (nystagmus) to caloric stimulation implies impaired brainstem function. As noted above the latter finding makes this a valuable test to assess the integrity of the nervous system of the patient in a coma.

CRANIAL NERVE III (OCULOMOTOR)

Major Functions: Move the Eyes
Constrict the Pupils
Adjust the Lens
Elevate the Eyelid
Classification: General Somatic Efferent
General Visceral Efferent
Nuclei: Oculomotor (GSE)
Edinger–Westphal (GVE)

The oculomotor nerve is primarily a motor nerve with both somatic and visceral components. Along with cranial nerves IV and VI, it is responsible for movements of the eye (somatic efferents). Of the six extraocular muscles, the oculomotor nerve controls four. These include the:

1. **Medial rectus,** which adducts the eye (pulls it toward the midline).
2. **Superior rectus,** which, along with the inferior oblique, elevates the eye.
3. **Inferior rectus,** which, along with the superior oblique (CN IV), "depresses" the eye (when one is depressed they might say, "things are looking down").
4. **Inferior oblique,** which assists in elevating the eye, particularly when the eye is turned inward toward the midline (i.e., the right inferior rectus comes maximally into play when one looks up and to the left).

These functions are controlled by the oculomotor nuclei, which are located along the midline in the upper mesencephalon. These nuclei (and the oculomotor nerve) also innervate the levator palpebrae, which are the muscles that retract or open the eyelids (the eyelids are closed by CN VII). It should be noted that the elevation of the eyelid also is partially accomplished by the sympathetic system. Thus partial ptosis (drooping of the eyelid) may not always reflect damage to cranial nerve III (see Horner's syndrome).

In addition to these extraocular muscles, cranial nerve III also exerts parasympathetic action (visceral efferents) on the intraocular muscles. These are the **ciliary muscles,** which affect the shape of the lens, and the sphincter muscles of the iris, which affect the size of the pupil (how much light gets into the eye). Normally the ligaments that are attached to the lens are under tension, keeping the lens more or less flat. When near vision is required, the ciliary muscles contract, reducing the tension on these ligaments, which results in a thickening of the lens, which brings the near object into clear focus.[6] As was seen earlier, the pupils act reflexively to various situations, including the amount of ambient light and accommodation. The third cranial nerve is responsible for the **constriction of the pupil** (pupillary dilation is a function of sympathetic activity carried by nerves derived from the superior cervical ganglion). These latter activities are under the control of the Edinger–Westphal nuclei that lie adjacent to the oculomotor nuclei. The Edinger–Westphal nuclei are the autonomic components of the oculomotor nuclear complex.

There are no identified sensory components to the oculomotor nerves. However, it appears likely that some type of proprioceptive feedback loop exists for the extraocular muscles. Cranial nerve V is the only one of the cranial nerves for which proprioceptive feedback is typically defined. It is well known that the trigeminal nerve (CN V) mediates cutaneous feedback from the face as well as proprioceptive feedback from the muscles of mastication (the latter via the mesencephalic nucleus of V). However, it is quite likely that cranial nerve V also mediates proprioceptive feedback for the muscular activities mediated by other cranial nerves, such as the muscles of facial expression (CN VII) and tongue movements (CN XII). Except for the muscles of mastication, these proprioceptive feedback loops are likely mediated by the chief sensory nucleus of CN V.

Lesions of the Oculomotor Nerve

The classic symptoms of a complete oculomotor nerve palsy include diplopia with strabismus, failure of adduction, inability to elevate the eye, ptosis, dilated pupil, and difficulty focusing on near objects. Because of the unopposed action of the lateral rectus and superior oblique, the affected eye will be shifted slightly laterally downward at rest (strabismus), and as a result of the lack of congruent fixation the patient will experience diplopia (double vision). The diplopia is exacerbated when the patient is asked to focus on near objects or look to the side opposite the lesion. The patient will not be able to adduct (move toward midline) the affected eye because of the involvement of the medial rectus, either on command or with convergence (focusing on near object). Involvement of the superior rectus and inferior oblique interferes with upward gaze in the affected eye [remember that the superior obliques (CN IV) assist downward movements]. Because the fibers of the superior rectus cross the midline in the vicinity of the nuclei of cranial nerve III, and indeed appear to pass through the nuclei, a nuclear lesion may result in bilateral weakness of the superior rectus muscles.[7] Other lesions that are thought to affect the connections between the pretectal region and the oculomotor nuclei also may produce upward gaze palsy (without the other symptoms of third nerve involvement. **Ptosis** or drooping of the eyelid will be present on the affected side because the levator palpebrae superioris muscle that elevates the eyelid is supplied by cranial nerve III.

The sphincter muscles that control the pupil are supplied by both parasympathetic and sympathetic fibers, working in an agonist and antagonist manner. Since cranial nerve III carries the parasympathetic fibers responsible for pupillary constriction, complete lesions of the oculomotor nerve will leave the sympathetic influences unopposed. This results in a dilated pupil that will respond neither to direct or consensual light reflex nor to accommodation. Because the ciliary muscle controlling the shape of the lens is affected, the patient will experience difficulty focusing on near objects with that eye.

Before leaving the symptoms of the oculomotor nerve, it may be well to recall **Horner's syndrome**, which previously was discussed. Recall that this syndrome results from a disruption of the sympathetic pathways and produces unequal pupils (*anisocoria*) and a mild ptosis. However, in this case, the affected pupil will be constricted as a result of the compromise of the dilator function of the sympathetic system. Also the ptosis, unlike that resulting from CN III, can be voluntarily overcome and will be on the side of the small, rather than the dilated pupil. Infarctions of the lateral medulla are one common cause of this syndrome.

CRANIAL NERVE IV (TROCHLEAR)

Major Function: Eye Movement (Superior oblique)
Classification: General Somatic Efferent
Nucleus: Trochlear (GSE)

The trochlear nerve is considered a pure motor nerve. It mediates a single ocular motor muscle: the superior oblique. As previously noted, it is somewhat unique in that it is the only cranial nerve to exit dorsally from the brainstem and it is the only cranial nerve that fully crosses the midline (decussates) after leaving its nucleus. Though it exits dorsally, it curves around the mesencephalon and appears anteriorly in the lower midbrain just above the pons.

Lesions of the Trochlear Nerve

The primary action of the superior oblique is to depress the eye. It is maximally effective in this capacity when the eye is adducted (turned medially). Hence, the critical test for its function is to ask the patient to adduct the eye and at the same time to look down. Since the affected eye cannot fully carry out this motion, the patient may complain of double vision in situations requiring similar movements, such as when descending stairs or buttoning one's shirt. Clinically, it may be noted that patients with cranial nerve IV palsy often exhibit a head tilt toward the unaffected side in order to compensate for the diplopia produced by the extortion (outward rotation) of the eye on the affected side.

CRANIAL NERVE VI (ABDUCENS)

Major Function: Eye Movement (lateral rectus)
Classification: General Somatic Efferent
Nuclei: Abducens (GSE)

Like the oculomotor and trochlear nerves, the abducens is considered a pure motor nerve and innervates a single eye muscle: the lateral rectus. The function of the lateral rectus is to abduct the eye or to pull the eye laterally or temporally (as opposed to medially or toward the nose). The nerve exits the brainstem near the midline at the pontomedullary junction.

Lesions of the Abducens Nerve

Since the primary action of the lateral rectus is to pull the eye temporally, a complete lesion of cranial nerve VI will result in the affected eye not moving laterally beyond the midline. Also, given the unopposed force of the medial rectus at rest, the eye will normally show a slight deviation toward the midline (medial strabismus) and the patient may complain of double vision (horizontal). This diplopia will intensify if the patient is requested to look toward the side of the lesion. As discussed earlier, if the nucleus of cranial nerve VI is involved, the patient will likely experience lateral gaze palsy in which both the ipsilateral lateral rectus and the contralateral medial rectus will demonstrate deficits when attempting to look to the side of the lesion. *(Remember that the medial rectus in the opposite eye will retain the capacity to adduct with convergence.)* Since the facial nerve (CN VII) loops around the nucleus of cranial nerve VI, a brainstem lesion that affects the abducens nucleus also may produce ipsilateral lower motor neuron seventh nerve palsy.

CRANIAL NERVE V (TRIGEMINAL)

Major Functions: Somatosensory (face and forehead)
Motor Control of the Muscles of Mastication
Classification: General Somatic Afferent
Special Visceral Efferent
Nuclei: Main (chief) sensory n. of V. (GSA)
Spinal sensory n. of V. (GSA)
Mesencephalic n. of V. (GSA)
Motor n. of V. (GVE)

Sensory Components

The trigeminal nerve supplies the somatosensory feedback for most of the face and front part of the head, including the meninges, teeth, tongue, cornea, sinuses, nasal and oral cavities, and skin (the skin of the rest of the head is supplied by the spinal nerves). The trigeminal nerve is the only cranial nerve that enters and exits at the level of the pons. As in the case of the spinal sensory nerves, most of the sensory fibers of the trigeminal nerve have their cell bodies in an external ganglion (the trigeminal ganglion). The one exception is the fibers mediating proprioception for the muscles of the jaw that have their cell bodies in the mesencephalic nucleus of cranial nerve V. Distal to the trigeminal ganglion, the trigeminal nerve divides into three branches. The **ophthalmic** (CN V1) branch or nerve that provides sensory input from the upper part of the face and scalp, the eyes (e.g., the cornea), upper portion of the nose, part of the nasal mucosa and frontal sinuses, and the meninges (all three divisions may supply the meninges). The **maxillary** (CN V2) branch transmits sensory information from the upper jaw, teeth, lip, hard palate, maxillary sinuses, and part of the nasal mucosa. The **mandibular** (CN V3) division has both sensory and motor components. It mediates the somatosensory feedback from the lower jaw, teeth, lips, buccal mucosa, anterior two-thirds of the tongue (touch, not taste), and part of the external ear and auditory meatus. Its motor component innervates the muscles of mastication (see Figure 5–12).

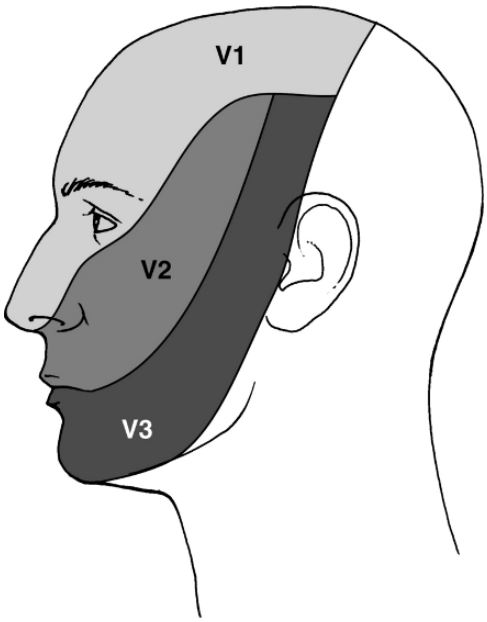

Figure 5–12. Sensory divisions of the trigeminal nerve: V1, ophthalmic; V2, maxillary; V3, mandibular.

The trigeminal nerve basically mediates all the same aspects of somatosensory sensation that are found in the dorsal roots of the spinal nerves: light touch, pain, temperature, stereognosis (fine discriminative touch), vibration, and proprioception. There are three separate nuclei that are responsible for sensory perception associated with the trigeminal nerve: (1) the **mesencephalic**, (2) **chief** (or *main*), and (3) **spinal nuclei** of cranial nerve V. The mesencephalic nucleus of cranial nerve V is the source of fibers that transmit proprioceptive information obtained primarily from stretch receptors located in the muscles of mastication. The sensory fibers synapsing in the main ("chief" or "principle") sensory nucleus of cranial nerve V, which is located in the pons, are comparable to the posterior columns of the spinal cord, mediating stereognosis, vibration, and possibly proprioception. It has been suggested that the mesencephalic nucleus largely may contribute "unconscious" proprioceptive feedback to the cerebellum, while the main sensory nucleus is associated with "conscious" proprioception. However, some authors suggest that the mesencephalic nucleus is responsible for most proprioceptive feedback, both conscious and unconscious, while some authors fail to mention proprioception at all in connection with the main sensory nucleus. Until more consistent data emerge, it seems likely that both nuclei mediate proprioception, but perhaps the mesencephalic nucleus primarily is responsible for feedback that controls mastication. While the pathways or connections have not been definitively established, one or both of these nuclei are probably also responsible for proprioceptive feedback for the muscles of the eyes, tongue, and muscles of facial expression. Lastly, the fibers derived from the spinal nucleus of cranial nerve V can be viewed as being comparable to those of the ventral and lateral spinothalamic tracts in that they appear to carry information for light touch, pain, and temperature. This latter nucleus is very long, extending from the pons into the spinal cord. As we shall see, several other cranial nerves also utilize this nucleus.

Whereas the sensory feedback from the spinal nerves travels to the contralateral ventral posterolateral (VPL) nuclei of the thalamus, the majority of the fibers from the trigeminal sensory nuclei travel to the contralateral **ventral posteromedial (VPM) nucleus** via the **ventral trigeminothalamic tracts**. The VPM nuclei, in turn, project to the more ventral portions of the sensorimotor cortices. There are a few fibers, from the main sensory nucleus in particular, that travel ipsilaterally in the dorsal trigeminothalamic tracts. Since the majority of these fibers cross, lesions affecting these tracts (as well as the VPM nucleus or somatosensory cortex) will result in contralateral sensory deficits in the face, whereas lesions of the trigeminal nuclei or nerve will produce ipsilateral deficits. There likely also are connections with the reticular nuclei, particularly from the spinal nucleus of cranial nerve V, as is the case with the spinal nerves.

Motor Components

The main motor function served by the trigeminal nerve is to innervate the muscles of mastication or the lower jaw (the masseter, temporal, internal and external pterygoids). The nerve fibers emanate from the motor nuclei of V and travel to the periphery with the mandibular branch (CN V3) of the trigeminal nerve. Similar to other muscles that act in a symmetrical fashion, the muscles of mastication receive bilateral cortical input; hence, upper motor neuron (supranuclear) lesions will have minimal effect on the patient's ability to chew. The trigeminal nerve provides sensory feedback from the ear (as do CN VII, VIII, IX, and X), but along with cranial nerve VII, it also provides motor control to the middle ear. Cranial nerve V adjusts the tension on the tympanic membrane.

Lesions of the Trigeminal Nerve

Depending on the type and location of damage to the trigeminal nerve, several different syndromes might result. Clearly, if a lesion destroyed the main branch of the trigeminal nerve, one would expect to find hemianesthesia of the face and weakness of the muscles of mastication on the same side. Commonly, however, the lesion may affect a portion of the nerve after it splits into its three divisions, thus producing reduced sensations in only one of its areas of distribution. Similarly, if nuclear lesions are involved, then only specific aspects of sensory stimulation may be affected. Even more distressing for patients is **trigeminal neuralgia** (*tic douloureux*), which although of unclear etiology, affects one or more branches of the trigeminal nerve. This latter syndrome consists of sharp, paroxysmal pain in one or more of the distributions of the trigeminal nerve. Testing for damage to the trigeminal nerve involves both motor and sensory examinations. To assess the muscles of mastication, the examiner has the patient open his or her jaw. Because of the way the muscles are attached, weakness on one side may result in a deviation of the jaw to the side of the lesion. Similarly, the patient may be asked to move the jaw laterally against resistance. There will be weakness when attempting to move the jaw opposite the side of the lesion. In lower motor neuron lesions (lesion of the nerve or motor nucleus) one might discern atrophy of the temporalis muscle on palpitation. With lesions of the mandibular branch or of the mesencephalic nucleus (afferent limb), the jaw jerk (reflex) should be diminished. The sensory exam consists of comparing light touch, pinprick (pain), two-point discrimination (stereognosis), or temperature on both sides of the face in all three trigeminal divisions. In cases of psychogenic hemianesthesia the patient may report the failure to perceive the vibrations of a tuning fork placed on the forehead of the "affected" side (in true neurological disease the sensation, which travels by bone to both sides of the forehead, is likely to be perceived bilaterally). Corneal reflexes normally are tested by touching the cornea with a wisp of cotton or tissue. Since the normal response is for both

eyes to blink in response to unilateral stimulation, it may be possible to differentiate between diminished or absent sensation in the cornea (CN V) and a diminished motor response (CN VII).

CRANIAL NERVE VII (FACIAL)

Major Functions: Muscles of Facial Expression
Taste
Salivation
Classification: Special Visceral Efferent
General Visceral Efferent
Special Visceral Afferent
General Somatic Afferent
Nuclei: Facial (SVE)
Superior salivatory (GVE)
Solitary (taste) (SVA)
Spinal nucleus of V. (ear) (GSA)

Sensory Components

The facial nerve, which loops posteriorly around the nucleus of CN VI before exiting from the anterolateral portion of the brainstem at the pontomedullary junction, has both motor and sensory components. Its main afferent function is to carry **taste sensations** from the anterior two-thirds of the tongue and the palate. Each nerve supplies the ipsilateral half of the tongue. The rostral portions of the solitary nuclei in the medulla mediate the sense of taste. From the solitary nuclei, the fibers conveying information about taste travel rostrally in the central tegmental tract where they synapse in the ventral posteromedial (VPM) nuclei of the thalamus. From there the information is carried to the opercular portions of the frontal and parietal lobes and the anterior portions of the insular cortices. Cranial nerve VII also carries **somatosensory information** from the auditory meatus, tympanic membrane, and a small section of skin behind the ear. The sensations from the ear synapse in the spinal nuclei of the trigeminal nerve.

Motor Components

The major motor functions of CN VII include:

1. Innervation of the superficial muscles of the face (the muscles of facial expression, including closure of the eyelid).
2. Stimulation of the lacrimal (tearing of the eye) and submandibular and sublingual (salivation) glands and nasal mucosa.
3. Control of the stapedius muscle (regulates the stapes: one of the ossicles of the middle ear).

The seventh cranial nerve via the facial nucleus innervates all ipsilateral facial muscles (muscles of facial expression) over which we have voluntary control, with the exception of the muscles involved in chewing (CN V). Among some of the functions or expressions mediated by cranial nerve VII include furrowing the brow, closing or winking the eye, smiling, whistling, and puffing out the cheeks. There is one aspect of the

neuroanatomical connections to the facial nuclei from its corresponding motor cortex (ventral portion of the precentral gyrus) that deserves special mention because of its important clinical considerations. The corticobulbar fibers, which control the muscles of the lower face, decussate and supply only the contralateral facial nucleus. However, the fibers that will eventually influence the muscles of the upper face (forehead and brow) split in the brainstem and terminate on both the ipsilateral and contralateral facial nuclei (bilateral innervation). Thus, while there is only contralateral innervation of the muscles of expression in the lower face, the muscles of the upper face receive bilateral input from the cortex.

The parasympathetic functions mediated by the salivatory nucleus of the medulla are to enhance the production of saliva (submandibular gland) and tears (lacrimal glands). As is the case with most other aspects of autonomic function, hypothalamic and limbic structures heavily influence these activities. Finally, the facial nerve exerts control of the stapedius muscle in the middle ear, which controls the action of the stapes, one of ossicles that transfers vibrations from the tympanic membrane to the oval window of the inner ear. Contraction of this muscle helps to attenuate loud sounds.

Lesions of the Facial Nerve

Lesions of the seventh cranial nerve will result in diminished or absent taste perception on the anterior two thirds of the tongue. This loss of taste should be limited to that half of the tongue that is ipsilateral to the lesion. If the branches of the nerve mediating the salivary glands are affected, the patient may complain of increased dryness of the mouth. Clinically, the most obvious effects of seventh cranial nerve involvement will center on facial motor symptoms. First, in simply observing the patient, one may note facial asymmetry. This might include a drooping of the mouth on the affected side, a smoothing of the nasal–labial fold, and an incomplete closure of the eyelid. If the patient is observed while eating, one may witness a tendency for food to collect in the cheek pouch on the affected side. There will be a decreased blink reflex to corneal stimulation, regardless of which cornea is stimulated. In fact, the eyelid may never fully close. This latter phenomenon, in conjunction with the decreased tearing in that eye from involvement of the lacrimal gland, may lead to an irritating dryness to the eye, possibly necessitating an eye patch and frequent bathing of the eye with saline solutions.

In cases where the facial weakness is less obvious, the patient may be asked to close the eyes tightly (with the examiner checking the degree of resistance to attempt to passively open them), smile, show his or her teeth, or puff out the cheeks (holding them out against resistance). The patient is then asked to furrow his/her brow (raise their eyebrows). Any significant asymmetry in the contracture of the muscles across the brow is noted. If weakness is noted in these upper facial muscles, along with signs of lower facial weakness, this implies a lesion of the facial nerve or its motor nucleus (**lower motor neuron lesion**). However, if a lower facial weakness is present, but the muscles of the forehead are intact bilaterally, an **upper motor neuron** or "*supranuclear*" **lesion** is suspected (see Figure 5–13). Remember, the upper part of the face (motor) is bilaterally innervated so only a lesion of the nerve or complete nuclear lesion will result in a total hemifacial paresis. With upper motor neuron lesions, there often will be associated weakness of the limbs, most commonly the upper extremity when the lesion involves the cerebral cortex.

An interesting phenomenon is sometimes present when a facial paresis is secondary to an upper motor neuron lesion (e.g., cortical lesion). Voluntary actions such as asking the patient to smile will reveal a unilateral facial weakness; however, if respond to a joke a spontaneous smile, no facial asymmetry is evident. This clinical feature suggests that different pathways

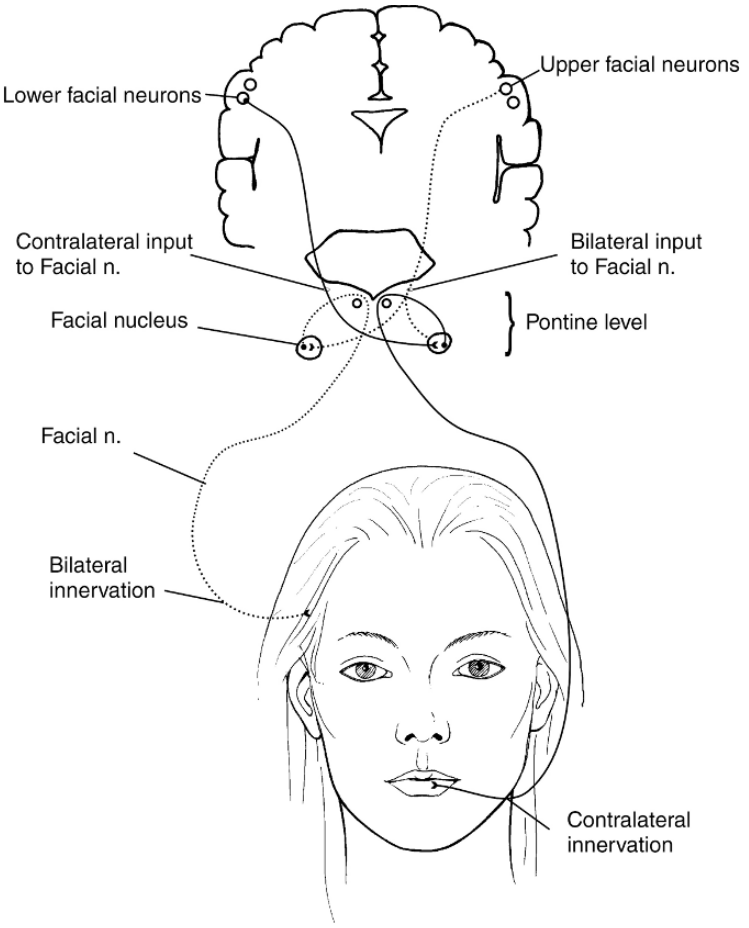

Figure 5–13. Differential innervation to upper and lower face. As shown, corticobulbar fibers that will eventually be responsible for innervation of the muscles of the lower face synapse only in the contralateral facial nucleus in the pons. Conversely, those cortical fibers destined for the upper portion of the face have inputs to both facial nuclei, from where they innervate the upper face bilaterally (not shown).

emerge for certain volitional versus emotionally generated motor responses. In addition to the deficits described above, the patient may report intolerance to high-amplitude auditory stimulation (hyperacousis) due to the impairment of the stapedius muscle.

While seventh cranial nerve paresis or paralysis may result from various conditions, including tumors and strokes, **Bell's palsy** represents perhaps the most common form of this disorder. Thought to be viral in origin, the classic symptoms of a lower motor seventh cranial nerve paralysis, as described above, typically evolve over a few hours to a few days. Fortunately, while most individuals who suffer from this condition recover within a few weeks to months, in some cases complete recovery may take much longer, if indeed it occurs at all. Finally, as in the case of the trigeminal nerve, the facial nerve has many divisions or branches after leaving the brainstem. Lesions that may affect one or more of these distal branches may produce a clinical picture of an "incomplete" seventh cranial nerve palsy.

CRANIAL NERVE VIII (VESTIBULOCOCHLEAR)

Major Function: Sense of Hearing; Balance, and Equilibrium
Classification: Special Somatic Afferent
Nuclei: Vestibular (SSA)
Cochlear (SSA)

The vestibulocochlear nerve is the last of the three purely sensory cranial nerves. As indicated by its name, it serves two functions: auditory and vestibular. It is responsible for carrying information from the vestibular and auditory systems, whose end organs are located in the inner ear, to nuclei located in the brainstem. Both systems respond to stimuli by the mechanical displacement or bending of hair cells in a fluid medium. Like cranial nerves VI and VII, cranial nerve VIII enters the brainstem as a double nerve just posterior to cranial nerve VII and just anterior to the cerebellum at the pontomedullary junction.

Cochlear Division

The cochlear portion of cranial nerve VIII is responsible for carrying auditory information from the hair cells of the organ of Corti, which is housed in a fluid-filled, spiral bony labyrinth called the cochlea in the inner ear, to the brainstem. The energy from the airwaves picked up by the tympanic membrane (eardrum) is mechanically magnified by the ossicles of the middle ear and transferred to the fluid of the cochlea. The hair cells within the organ of Corti are tontopically organized. This tonotopic organization is maintained beginning with the first synaptic connections in the dorsal and ventral cochlear nuclei in the brainstem all the way to the primary cortical projection area (Heschl's gyrus).

Two major pathways—a dorsal and a ventral system—exit from the dorsal and ventral cochlear nuclei, respectively. All of the postsynaptic fibers from the dorsal cochlear nuclei cross the midline as the **dorsal acoustic stria** and the majority ascends directly to the contralateral inferior colliculi via the **lateral lemniscus** (auditory pathway in the brainstem). A few fibers synapse in the contralateral superior olivary nucleus before ascending in the lateral lemniscus. Postsynaptic fibers leaving the ventral cochlear nuclei take a somewhat different course, although most eventually also will make their way to the inferior colliculi. Two fiber tracts leave the ventral cochlear nucleus. One, the **intermediate acoustic stria**, like the pathway from the dorsal cochlear nucleus, curves around the dorsal aspect of the inferior cerebellar peduncle, crosses the midline, and enters the contralateral lateral lemniscus. The **ventral acoustic stria**, the largest of the three acoustic stria, follows a more ventral path around the inferior cerebellar peduncle as it exits from the ventral cochlear nucleus. As shown in Figure 5–14, fibers from the ventral acoustic stria basically take one of three courses. They may:

1. Synapse in the ipsilateral superior olivary nuclei, which in turn may send tertiary fibers to the inferior colliculi via either the ipsilateral or contralateral lateral lemniscus.
2. Send fibers to the contralateral superior olivary nucleus, which similarly projects to the inferior colliculi.
3. Send fibers directly to the contralateral inferior colliculi, bypassing the superior olivary nuclei.

These crossing fibers of the ventral acoustic stria (those either directly entering the opposite lateral lemniscus or crossing before or after synapsing in the contralateral or ipsilateral superior olivary nuclei) make up what is known as the **trapezoid body,** which is seen in the pontine tegmentum. Because of the incomplete crossing at this elementary level,

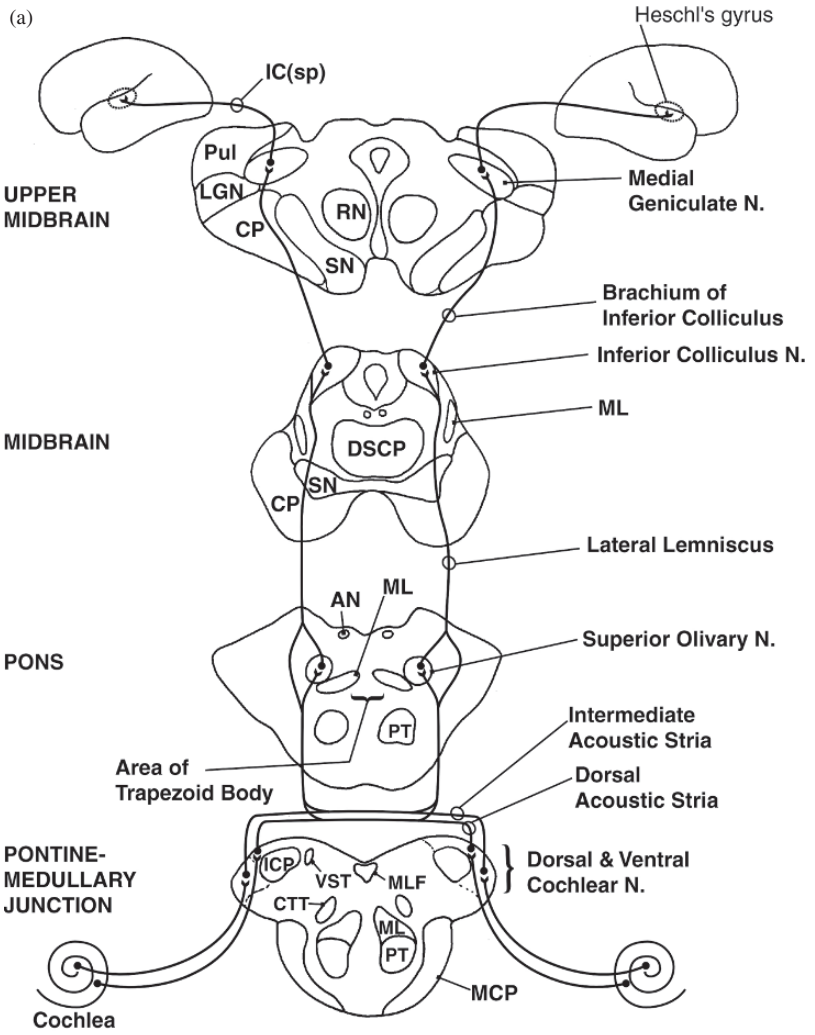

Figure 5–14. Auditory Pathways. (a) A schematic representation of the dorsal acoustic stria, which emanate from the dorsal cochlear nuclei, and the intermediate acoustic stria, which derive from the ventral cochlear nuclei. (b) In order to simplify the illustration, the ventral acoustic stria (also derived from the ventral cochlear nuclei) is shown separately. Note that the dorsal and intermediate stria both cross the midline before entering the lateral lemniscus, while the ventral stria have multiple inputs to the lateral lemniscus, both ipsilateral and contralaterally.

AN, abducens nucleus
CP, cerebral peduncle
CTT, central tegmental tract
DSCP, decussation of the superior cerebellar peduncle
IC(SP), internal capsule, sublenticular portion
ICP, inferior cerebellar peduncle
LGN lateral geniculate nucleus
MCP, middle cerebellar peduncle
ML, medial lemniscus
MLF, medial longitudinal fasciculus
PT, pyramidal tract
Pul, pulvinar
RN, red nucleus
SN, substantial nigra
VCN, vestibulocochlear nerve
VST, vestibulospinal tract

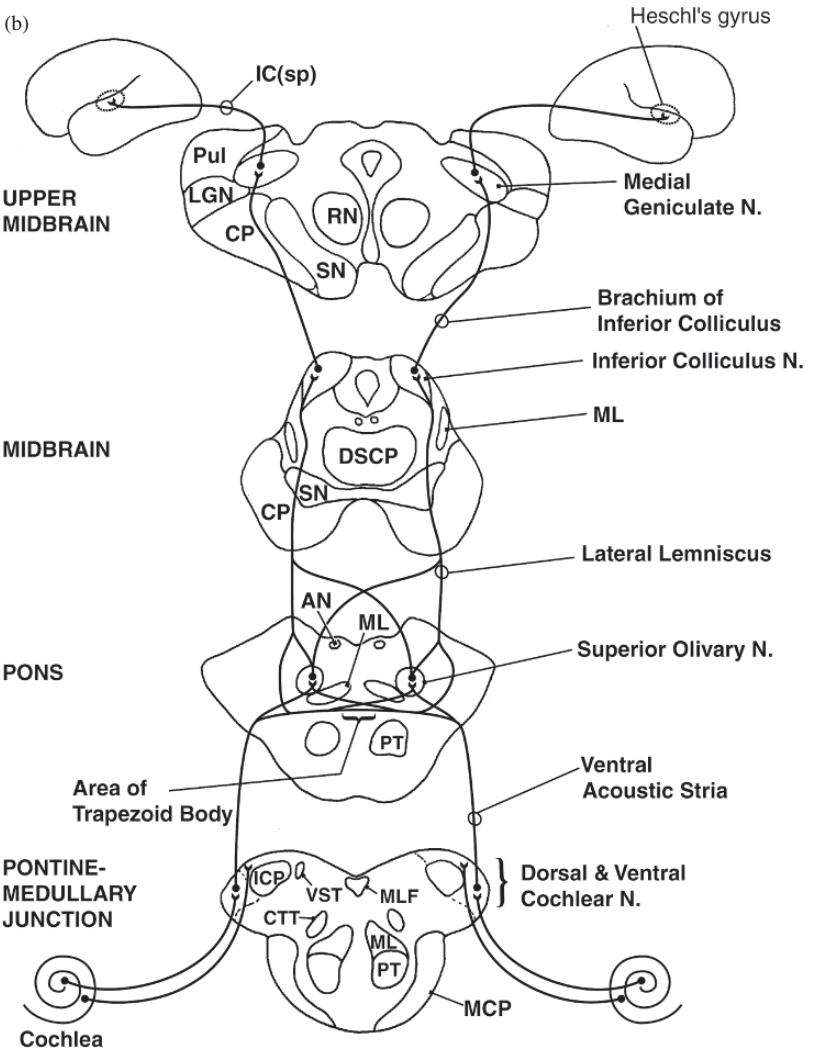

Figure 5–14. *(Continued)*

it is thought that the superior olivary nuclei may be important in the localization of sound, apparently responding to the slight time differential it takes for sound to arrive at the two ears. However, this asymmetry of input is preserved all the way to the cortex, thus also potentially allowing for some mediation of this effect at higher levels.

The above description fails to capture the full complexity of the auditory pathways at the brainstem level. A few additional considerations may help. Although the eighth cranial nerve generally is considered to be a pure sensory nerve (hearing and vestibular sense), there are several motor components that can affect hearing. The nuclear complex surrounding the superior olivary nuclei appears to be the source of an olivocochlear pathway that can modulate sound by altering the sensitivity of hair cells in the organ of Corti. The ear's sensitivity can also be altered by the respective actions of cranial nerves V and VII via differential tension on muscles controlling the tympanic membrane and stapes of the middle ear. Also, although the pathways are not well defined, some auditory input probably reaches the ascending reticular formation and those nuclei responsible for eye movements, such as CN III and VI (probably via the MLF), and movements of the head or neck (colliculi

and CN XI). The former likely serves a general alerting function, while the latter probably establish the anatomical bases for various reflexes, such as orienting the eyes and head to auditory stimuli, especially when they are loud or unexpected.

As previously noted, the major proportion of fibers leaving the cochlear nuclei ends up ascending in the lateral lemniscus to the inferior colliculi. At this level there is some additional crossing of the fibers between the two colliculi. From each of the inferior colliculi, ipsilateral connections are established with the medial geniculate nuclei via the brachium of the inferior colliculi. In turn, the medial geniculate nuclei send projections (via the sublenticular portion of the internal capsule) to the primary auditory cortex (Heschl's gyrus) located in the recesses of the lateral fissure in the temporal lobe. Unlike the visual, motor, and somatosensory systems that are primarily crossed, the auditory system from the superior olives to the cortex has a more extensive bilateral representation. The left auditory cortex, however, still receives most of its input from the right ear; estimates of up to 40% of its input is from the ipsilateral ear. This redundancy in the system, or the bilateral input to the primary auditory cortex, assures that unilateral injury to **Heschl's gyrus** produces minimal clinical deficits. In order for cortical lesions to produce significant clinical deficits, bilateral lesions to Heschl's gyrus or a lesion that involves Heschl's gyrus unilaterally with extension to white matter connections to the contralateral Heschl's gyrus are necessary.

Vestibular Division

The vestibular system provides two general types of information: (1) the orientation of the head in space and (2) the movement of one's body (head) through space (both the direction of movement, as well as the sense of movement). As we have noted in earlier chapters, the integrity of the vestibular system is critical to maintain normal motor functions and for coordination, balance, or maintaining one's equilibrium. The vestibular system apparently provides highly reliable and accurate information regarding spatial orientation and direction of movement, but is still probably very dependent on collateral inputs from other sensory systems. One fairly common illustration of this fact can occur in scuba diving. In very deep dives where neither the surface nor the bottom can be visualized and there are no perceptual forces of gravity operating, it is extremely easy (and very disconcerting) to become "disoriented" and literally not know which way is up. Equally distressing is to unknowingly be diving along an incline, thinking it is a horizontal plane, then discover that one's bubbles are not "rising" but appear to be floating off in what now appears to be a horizontal direction. A diver in such situations may experience "dizziness" or other signs associated with vestibular disturbances.

The sensory organs for the vestibular system, located in the inner ear, include the three semicircular canals, the utricle, and the saccule. The semicircular canals represent three different planes or orientations in space and respond to angular acceleration and deceleration. The utricle responds to gravitational forces and to horizontal linear acceleration. The saccule responds to linear acceleration in the dorsal–ventral plane. As the fibers for the vestibular system enter the brainstem, a few course directly to the flocculonodular lobe of the cerebellum, while most synapse in the vestibular nuclei (superior, inferior, medial, and lateral).

The vestibular nuclei give rise to both the ascending and descending MLF. The former important in ensuring that changes in the position of the head will result in equal compensatory movements of the eyes in order to maintain stability of the visual image despite movement of the head in space. The connections between the vestibular nuclei, the cerebellum and the spinal cord (via the descending MLF) are reciprocal. It has been noted that the vestibular nuclei play a major role in maintaining body posture and equilibrium. While many motor responses to vestibular stimulation are reflexive in nature (e.g., excitation of antigravity muscles

to maintain balance), the vestibular system also likely plays a significant role in the coordination of conscious motor activities. Finally, projections to the cortex may serve two (probably more) basic functions.Direct connections with the visual system (e.g., eye movements through the MLF) ensure that changes in the position of the head will result in equal compensatory movements of the eyes in order to maintain stability of the visual image despite movement of the head in space. Finally, projections to the cortex may serve two (probably more) basic functions. The vestibulocortical connections provide for conscious awareness of the orientation of one's body in space or movement through space, and thus allow for the conscious adjustment of skilled movements (or perhaps, the conscious inhibition of certain reflex righting movements) during the execution of particular activities.

Lesions of the Vestibulocochlear Nerve

Cochlear Division

The most common symptoms of damage or irritation to the auditory system are tinnitus and hearing loss. Tinnitus is the perception of "ringing" in one's ear, which may be characterized by either high- or low-pitch sounds. A relatively common and benign cause of tinnitus is medication (e.g., large doses of aspirin). Hearing is one of human's most acute senses, both in terms of absolute thresholds and in discriminating discrete, successive stimuli. Different forms of auditory impairment can result from lesions to the central auditory pathways (brainstem, thalamus, cerebral cortex) and peripheral systems. The peripheral auditory system can be impaired due to buildup of wax in the outer ear canal, damage to the eardrum, changes in the ossicles of the middle ear, and problems with the hair cells of the inner ear. Despite its sensitivity, the eighth cranial nerve probably is the one cranial nerve most likely to require specialty examination (by an audiologist) for more definitive diagnoses. There are, however, several routine procedures that can be carried out at bedside that can provide good preliminary hypotheses about the relative integrity of various parts of this system.

The first step is to obtain some measure of absolute thresholds of hearing sensitivity in each ear. This can be assessed in each ear by close, low-level stimulation that normally could not be picked up by the opposite ear, for example, the ticking of a watch or by rubbing two fingers together. Not uncommonly, the patient may have relative loss for certain frequencies, but this probably is best examined by an audiologist. Certain types of higher-level (e.g., cortical) hearing problems such as discrimination or perception of sounds and unilateral neglect will be covered in Chapter 9. If on routine clinical exam a hearing loss is suspected, it is important to determine whether the deficit is due to nerve damage or is the result of some type of conduction (air or bone) loss.

Two tests performed with a 512 Hz tuning fork (lower vibrations can be misinterpreted as "sound") can help make this distinction. The first test (**Weber test**) involves placing the base of a vibrating tuning fork on the forehead (centered) or on the vertex of the skull. Normally the sound is perceived as being equal in both ears or as coming from the center of the head. If the sound localizes to one ear more than the other, it will suggest either a conduction loss on the side to which the sound localizes or a neural loss in the opposite ear.

In the second (the **Rinne test**), the base of the tuning fork is placed on the mastoid process immediately behind the ear (bone conduction). When the patient reports no longer hearing the sound, the tuning fork (vibrating end) is held near the auditory canal (air conduction). Normally, the perception should increase during the second step, as air conduction is more efficient than bone conduction. If the patient hears better with the tuning fork pressed against the mastoid process than when the tuning fork is held adjacent to the ear, the patient likely suffers from an air conduction deficit. If the sound is reduced with either placement, a sensorineural hearing loss is more likely.

Vestibular Division

The most common symptoms of damage or irritation to the vestibular system (diseases of the labyrinth or neural pathways) are nystagmus and vertigo, dizziness, and nausea or vomiting. Nystagmus may be more or less constant or may be brought on by certain maneuvers (e.g., positional nystagmus). Causes of nystagmus are multiple and its vestibular origin perhaps is most clearly established when it is accompanied by vertigo. With regard to the latter, it is important to note whether the patient has any subjective symptoms of dizziness or vertigo (the room or occasionally the patient himself or herself is perceived to be spinning or the normal visual planes tend to be abnormally tilted). If present, these symptoms often are associated with nausea and vomiting. If vestibular dysfunction is suspected, caloric testing can be done, comparing the response obtained in the two ears. This test was explored earlier in our discussion of eye movements and how it could be used to test the integrity of the caudal pons or brainstem in the coma patient. In addition to providing a test of the integrity of the vestibular portion of the eighth cranial nerve and vestibular nuclei (and their connections with the centers for lateral gaze), the caloric test assesses the integrity of the peripheral mechanisms (labyrinthine complex). If the system is intact, one should produce nystagmus with the fast component to the side opposite the cold water stimulation. Although a valuable diagnostic test, cold calorics generally are not performed on a conscious patient due to the unpleasant side effects (e.g., nausea and vomiting). Although a variety of lesions can affect cranial nerve VIII and its peripheral organs, acoustic neuromas (cerebellopontine angle tumors involving the vestibulocochlear nerve) are one cause of slowly progressive hearing loss associated with tinnitus and vestibular symptoms.

CRANIAL NERVE IX (GLOSSOPHARYNGEAL)

Major Functions: Muscles of Pharynx
Sensation to Throat
Taste

Classification: Special Visceral Efferent
General Visceral Efferent
Special Visceral Afferent
General Visceral Afferent
General Somatic Afferent

Nuclei: Nucleus ambiguus (SVE)
Inferior salivatory (GVE)
Solitary (SVA)
Solitary (GVA)
Spinal n. of V. (GSA)

The glossopharyngeal nerve, as its name implies, primarily is related to functions of the tongue and pharynx. While it has both sensory and motor components, for all practical purposes it is generally regarded as a sensory nerve, since it supplies only one small muscle that has little clinical significance when lesioned (the stylopharyngeus muscle). While it is possible to isolate some specific functions of cranial nerve IX, such as touch and taste on the posterior one-third of the tongue, in practice cranial nerves IX and X typically are examined as a unit by assessing the gag reflex. Cranial nerve IX supplies the afferent limb and cranial nerve X supplies the efferent limb of this reflex response. The glossopharyngeal nerve enters (and exits) the anterolateral portions of the rostral medulla just below cranial nerve VIII.

Sensory Components

Cranial nerve IX mediates the sensation of taste from the posterior third of the tongue and the pharynx. These fibers synapse in the rostral portion of the nucleus solitarius before ascending (see cranial nerve VII). The more caudal portion of the nucleus solitarius receives afferent fibers that monitor blood oxygen levels (from carotid body) and arterial blood pressure (from carotid sinus), which are relayed to the hypothalamus. General somatic sensations from the posterior part of the oral cavity (e.g., posterior third of the tongue, upper pharynx) are mediate via glossopharyngeal connections with the spinal nucleus of the trigeminal nerve. Other GSA fibers carrying tactile information from the ear also arrive at the spinal nuclei of cranial nerve V by way of the ninth cranial nerve.

Motor Components

There are two targets for efferent motor pathway associated with cranial nerve IX. First, one group of fibers innervates one of the muscles of the pharynx (the stylopharyngeus m.), which assists in the elevation of the pharynx (although this also is accomplished to a somewhat greater degree by the vagus nerve). The nucleus ambiguus is the source of these special visceral efferents to the stylopharyngeus muscle. Second, the remaining efferent fibers of cranial nerve IX innervate the parotid gland via the inferior salivatory nuclei. Thus cranial nerves VII and IX share two common functions: (1) taste to the tongue (CN X may mediate taste to the epiglottis) and (2) control of the salivary glands.

Lesions to the Glossopharyngeal Nerve

Touch and taste to the posterior third of the tongue can be used to assess the integrity of cranial nerve IX. However, as the procedure is somewhat awkward and inconvenient, this test typically is not done as part of most neurological exams. Therefore, most commonly cranial nerve IX and X are tested together by checking the presence of the gag reflex, which assesses the afferent input necessary for this response to occur (see Vagus Nerve).

CRANIAL NERVE X (VAGUS)

Major Functions: Muscles of Pharynx and Larynx
Parasympathetic Control of Abdominal and Thoracic Viscera
Sensation to the Throat

Classification: Special Visceral Efferent
General Visceral Efferent
Special Visceral Afferent
General Visceral Afferent
General Somatic Afferent

Nuclei: Nucleus ambiguus (SVE)
Dorsal motor nucleus of X (GVE)
Solitary nucleus (rostral) (SVA)
Solitary nucleus (caudal) (GVA)
Spinal nucleus of V (GSA)

Like the glossopharyngeal nerve, the vagus nerve is a mixed sensory and motor nerve. From a clinical viewpoint, cranial nerve IX is considered more sensory and cranial nerve X is considered more motor. Cranial nerve X is larger than cranial nerve IX and exits just below the glossopharyngeal nerve in the rostral medulla. Although cranial nerves III, VII, IX,

and X all carry parasympathetic fibers, the vagus nerve is the only cranial nerve mediating parasympathetic functions that has target organs that lie outside of the cranial vault.

Sensory Components

The rostral portion of the solitary nucleus (n. solitarius) receives fibers from cranial nerve X, which carry information about taste from the epiglottis. As with cranial nerve IX, the caudal portions of the solitary nuclei also receive visceral sensory input from the trachea and larynx via the vagus nerve, as well as from some of the viscera to which it supplies efferent connections (e.g., lungs, esophagus, stomach, and intestines). Some general sensory fibers of cranial nerve X carry tactile information from the oral cavity and from the ear. This sensory input is conveyed to the spinal nucleus of cranial nerve V via the vagus nerve.

Motor Functions

The vagus nerve also supplies the muscles of the soft palate and pharynx that mediate swallowing. However, as in the case of its sensory input, the vagus nerve is the only cranial nerve that supplies motor fibers to the larynx. All these efferent fibers, which are critical for swallowing and motor speech, originate in the nucleus ambiguus. The other major efferent pathway of the vagus nerve originates from the dorsal motor nuclei of cranial nerve X that lie in the medulla. These fibers constitute the parasympathetic branches that supply the thoracic and abdominal viscera. Some of the preganglionic parasympathetic fibers that supply the heart appear to come from the nucleus ambiguus. These parasympathetic inputs reduce the heart rate, constrict the size of the bronchioles, and stimulate peristalsis as well as gastric, hepatic, and pancreatic activity.

Lesions of the Vagus Nerve

Since the vagus nerve provides most of the motor control to the muscles of the pharynx, soft palate, and larynx, the integrity of cranial nerve X can be assessed by testing and observing activities that directly require the use of those muscles. Hoarseness or dysphonia may suggest difficulties with the larynx. Dysphagia (difficulty swallowing) may suggest difficulties with the muscles of the soft palate and pharynx (in eating or drinking the patient may complain that the food or water gets up into the nasal passages). One simple direct test is to have the patient say, "Ahhh." The examiner notes whether the palate elevates symmetrically and whether the uvula deviates. With lesions affecting the tenth cranial nerve, the palate will show reduced elevation (or fail to elevate) on the affected side, causing the uvula to deviate to the unaffected side. Another direct test is to stimulate each side of the palate and observe the gag reflex. Remember, the afferent side of the "gag reflex" is mediated by the glossopharyngeal nerve, and the motor side by the vagus nerve. Thus, an absent or diminished gag reflex does not indicate whether cranial nerve IX or X is involved. If the gag reflex is intact, however, this argues for the integrity of both cranial nerves IX and X.

Cranial Nerves and Speech Production

While discussing motor functions of the larynx, pharynx, soft palate, tongue, and lips, a few general comments about motor speech function are warranted. Speech requires the integrity of various cranial nerves that mediate movement of the jaw (CN V), the lips (CN VII), the tongue (CN XII), the soft palate and the pharynx (CN IX and X), and the larynx (CN X). All of these motor systems must be intact for well-articulated speech. The afferent feedback from these muscles also must be intact. Ultimately, the motor programming for the execution of speech is derived from higher cortical centers, but these cortical commands must be executed

at the level of the cranial nerves. To vocalize certain guttural sounds such as hard K or G sounds or "Ahhh," the soft palate must elevate and close off the nasal–pharyngeal passageway. Failure of the palate to elevate produces a nasal quality to the voice. Phonation is carried out by the larynx and articulation is carried out by pharynx, soft palate, tongue, lips, and to a lesser extent the mandible. Lingual sounds are those that require maximal deviation of the tongue, such as "D," "L," and "T." Labial sounds are those that emphasize the actions of the lips, such as "B," "M," and "P." Thus, to test for proper elevation of the palate, the patient can say "Ahhh"; to test for proper coordination of soft palate, tongue, and lips in speech production, the patient may be asked to repeat "kuh, tuh, buh," either singly or as a group in sequence. In general, it is important to remember is that disturbances of speech do not always translate into "cortical lesions"; disturbances at the level of the brainstem (or cerebellum) also significantly can impact motor speech production (i.e., dysarthria).

CRANIAL NERVE XI (SPINAL ACCESSORY)

Major Functions: Turn Head
Lift Shoulders
Classification: Special Visceral Efferent (SVE)
Nucleus: Accessory (SVE)

The accessory (also known as "*spinal accessory*") nerve is considered a pure motor nerve. It supplies the sternocleidomastoid muscle, which assists in turning the head to the contralateral side, and the upper trapezius muscle, which allows for lifting or shrugging of the shoulders. Despite its apparent simplicity, this nerve has several unusual features. First, it is the only cranial nerve that has its major nucleus in the spinal cord and whose fibers arise at the spinal level. The accessory nucleus consists of cells in the lateral portion of the anterior horn of the spinal cord at the level of C-1 through C-5. The rootlets emerge from the cord at these levels, but then ascend through the foramen magnum before exiting as the 11th cranial nerve. The cerebral source of activation for the trapezius is in the contralateral precentral gyrus. The tracts for the cortical innervation of the spinal nuclei responsible for the contraction of the sternocleidomastoid muscles have not been well defined. However, it has been suggested that these connections are predominately ipsilateral. There is certain logic to this, since the activation of this muscle causes the head to turn to face the contralateral hemispace. It also is unclear whether a small group of fibers that originate in the caudal sections of the nucleus ambiguus that supply the larynx should be considered part of the accessory nerve. These fibers travel a very short distance with cranial nerve XI before joining the vagus nerve to the larynx. Some authors believe that these fibers more properly belong to the tenth cranial nerve.

Lesions of the Spinal Accessory Nerve

Sternocleidomastoid

The normal contraction of the sternocleidomastoid muscle results in the turning of the head to the opposite side. Hence, in testing the integrity of the muscle (nerve), the patient is asked to turn his or her head against resistance to each side. Weakness when attempting to turn the head to one side suggests a lesion of the contralateral 11th cranial nerve.

Trapezius

The upper trapezius serves to support the shoulders and upper back. Lesions affecting this part of the nerve may result in a slight drooping of the shoulder. In formal examination,

the patient is asked to shrug the shoulders against resistance, observing for asymmetries in strength or changes in the position of the scapula (downward and outward rotation).

CRANIAL NERVE XII (HYPOGLOSSAL)

Major Function: Movement of the Tongue
Classification: General Somatic Efferent
Nucleus: Hypoglossal (GSE)

The hypoglossal nerve is considered to be a pure motor nerve. Like cranial nerve XI, the hypoglossal nerve consists of multiple rootlets. The hypoglossal nerve is found in the rostral medulla just a little below the abducens nerve (CN VI). It exits the medulla in the anterolateral sulcus (between the pyramids and the olives), thus placing the hypoglossal rootlets more anteriorly in the brainstem than cranial nerves IX, X, and XI, all of which exit lateral to the olive.

The solitary function of cranial nerve XII is to control movements of the tongue. A unilateral lesion involving the nucleus or nerve (i.e., a LMN lesion) will result in atrophy on the side of the lesion and weakness of the muscles on that side. Because of the manner in which the muscles operate the tongue when protruded, weakness on one side will result in the tip of the tongue being deviated to the same side as the lesion when the tongue is protruded. This is because as the tongue is pushed forward, the muscle on the intact side of the tongue is unopposed by the muscle on the affected side.

Lesions of the Hypoglossal Nerve

The first step in attempting to identify a possible dysfunction of the hypoglossal nerve is to simply observe the tongue for deviation (toward side of the lesion) on protrusion or for atrophy or fasciculations. One also can test the strength of the tongue when pressed against the inside wall of the cheek. Here one would look for weakness when asked to press on the cheek opposite the lesion. Consistent with the findings in the peripheral musculature, atrophy of the tongue and fasciculations are associated with lesions of the nucleus or nerve (lower motor neuron lesion). In lower motor neuron lesion the tongue will deviate toward the side of the lesions, whereas in an upper motor neuron lesion (because of the contralateral representation) the deviation will be to the side opposite the lesion.

Endnotes

1. These components are sometimes referred to as “branchial motor” since they are derived from the branchial arches: if we were fish, these would have developed into gills.
2. While a medial and (less frequently) an intermediate olfactory stria also are described, most authors seem to agree that the lateral olfactory stria represent the major cortical pathways for olfactory input. The medial and intermediate olfactory stria may have very limited olfactory input, if any, to the subcallosal or septal area and to the region of the anterior perforated substance.
3. Although perhaps one of the less frequently tested cranial nerves, detecting the presence of anosmia may offer important clinical insights, especially following closed-head injuries. Nils Varney (1988) reported that in one sample of head injury victims, the presence of anosmia was very highly correlated with failure to maintain steady employment, despite the relative absence of deficits on formal neuropsychological

measures. Of course, the lack of vocational success within this group (along with their anosmia) was likely due to lesions of the orbital frontal cortices.

4. Why is it that when you look in a mirror, the image appears reversed right to left, but not up and down?
5. A lesion that selectively affects the lateral aspect of the chiasm (i.e., the fibers deriving from the temporal portion of the retina) may produce a nasal field cut in the ipsilateral eye. An internal carotid artery aneurysm that puts pressure on the lateral portion of the chiasm might produce such a finding.
6. As an individual ages, the capacity of the lens to make this adaptation diminishes; hence, the frequent need for bifocals beginning in the fourth or fifth decades.
7. Although anatomical documentation could not be found, we suspect that this crossing of superior rectus fibers allows for synapses within the contralateral nucleus, thus establishing a mechanism for conjugate upward gaze.

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