

## CRANIAL NERVE EXAMINATION

Part of "Chapter 7 - The Neurological Physical Examination"

The next part of the neurological examination focuses on the CNS. There are twelve pairs of CNs. Each CN has a left and a right nerve; each side must therefore be evaluated separately. There may be dysfunction on one side only or both sides. Findings from each side are compared for symmetry. This chapter is organized to include details of testing, discussion of findings, and abnormal findings. See Chapter 5 for a review of the anatomy and physiology of cranial nerves.

### **Olfactory Nerve (Sensory) (CN I)**

Testing of the olfactory nerve is often deferred unless an anterior fossa mass is suspected. The sense of smell is tested by obstructing one nostril while testing the other. A piece of cotton that has been saturated with a common, odoriferous, nonirritating substance is placed under the unobstructed nostril. Cinnamon, cloves, coffee, or peppermint are possible odors that patients should be able to identify if CN I is intact.

### **Abnormalities**

**Anosmia** is an inability to smell. It is an early sign in the diagnosis and localization of certain intracranial neoplasms. There are several causes of anosmia, some of which are attributable to neuropathologic conditions such as meningiomas of the sphenoid ridge and olfactory groove, gliomas of the frontal lobe, and parasellar lesions with pressure on the olfactory bulbs or tracts. Common non-neurological causes of anosmia include the common cold, sinusitis, or inflammation of the nasal cavity. The typical syndrome with *sphenoidal ridge meningioma* is one of unilateral optic atrophy or papilledema, exophthalmos, and ipsilateral anosmia. A *meningioma of the olfactory groove or cribriform plate* includes unilateral anosmia with retrobulbar neuritis or optic atrophy with progression to bilateral anosmia. Another possible presentation is the **Foster-Kennedy syndrome**, which includes optic atrophy and anosmia on the side of the lesion and papilledema on the other side.<sup>6</sup>

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### **Optic Nerve (Sensory) (CN II)**

The optic nerve is the only CN that can be examined directly. Testing encompasses an evaluation of visual acuity, visual fields, and an ophthalmoscopic examination. Each eye is evaluated individually while the other eye is covered.

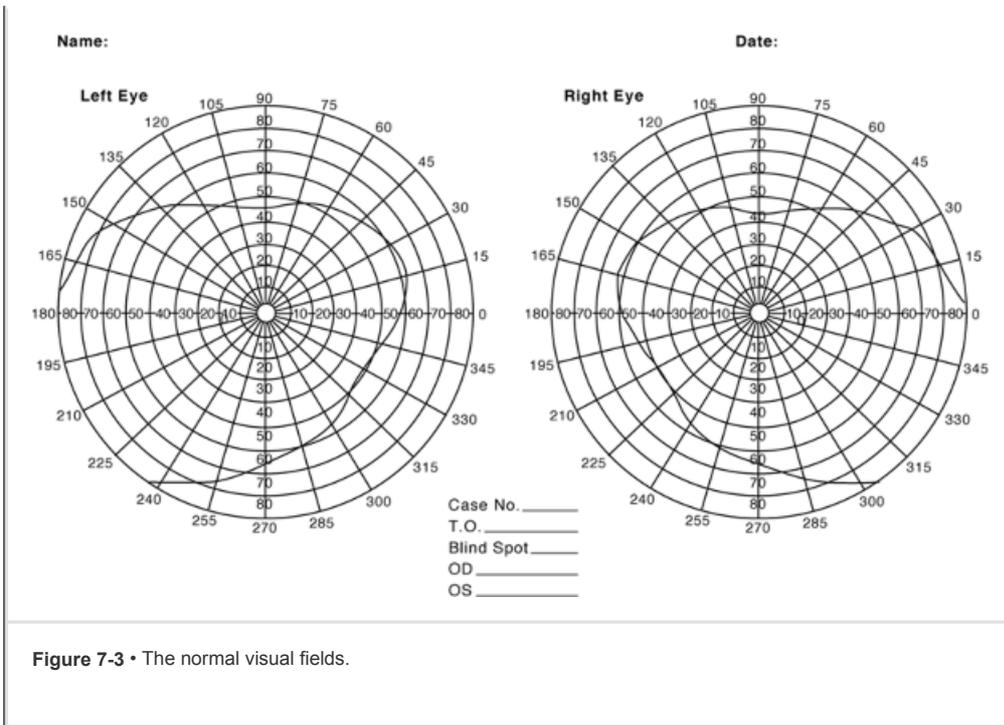
### **Visual Acuity**

**Visual acuity** can be evaluated informally by asking the patient to read from printed material, such as a newspaper. A standard Snellen chart is available in office or clinic settings for more formal testing. Each eye is checked individually. The patient is asked to read the line on the chart with the smallest letters that he or she is able to read at a distance of 20 ft. The number beside each line of letters signifies the number of feet at which letters can be read by a person with normal vision. This becomes the denominator in recording vision. Normal vision is 20/20. When vision is defective, the patient may only be able to see the larger letters at 20 feet. For example, persons with 20/40 vision are able to see at 20 feet what those with normal vision can see at 40 feet.

An adaptation of the Snellen chart, the Rosenbaum Pocket Vision Screener, is designed for quick bedside use. The chart is held at a distance of 14 inches from the patient. Normal vision in this instance is 14/14. The ratio is calculated in the same manner as for the Snellen test.

### **Visual Fields**

A **visual field** is the area of vision normally seen with one eye. Each visual field extends 60 degrees on the nasal side, 100 degrees on the temporal side, and 135 degrees vertically (60 degrees superiorly and 75 degrees). Figure 7-3 displays normal visual fields. The **confrontation test** provides a rough estimate of the scope of each visual field. This is a test in which the examiner "confronts" the patient by sitting 2 feet in front of him or her at eye level. The patient is asked to cover one eye lightly and looks at the examiner's eye directly opposite (i.e., the patient's left eye will be looking at the examiner's right eye). The examiner then closes one eye, the one the visual field of which is *not* being currently used, thus isolating the visual field in the other eye and superimposing it on that of the patient's visual field. A pencil or a moving finger is then introduced from the periphery into the patient's field of vision; each quadrant (upper and lower) is checked individually. The patient is asked to indicate when the object is first seen. The examiner uses his or her own visual field as the norm for comparison to the patient's visual field.

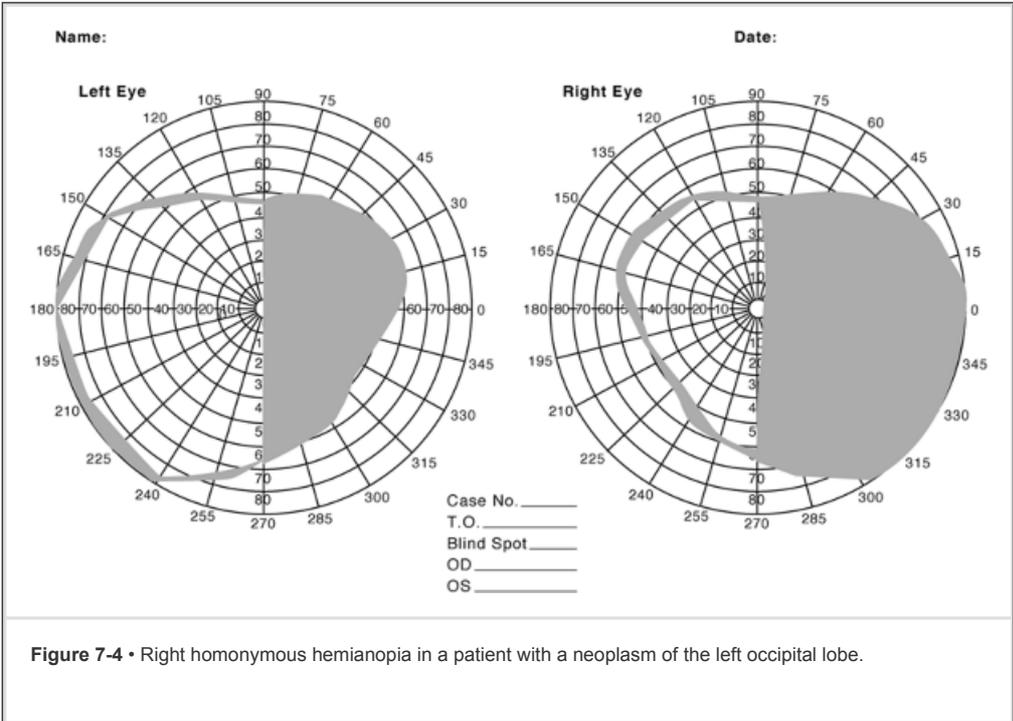


The confrontation test is designed to reveal only gross defects of the visual fields. If any defects are found, the visual fields should be plotted by an ophthalmologist using the standard and more sensitive **perimetric test** or a **tangent screen**. Visual field testing can reveal field cuts related to characteristic deficits along the visual pathway (i.e., optic nerve, optic tract, lateral geniculate body, geniculocalcarine tract, or occipital lobe). Chart 7-1 shows specific field cuts; Figure 7-4 shows field cuts in a person with right homonymous hemianopia.

**CHART 7-1 • Visual Field Defects Produced by Selected Lesions in the Visual Pathways**

Visual Pathways	Visual Fields	Blackened Field Indicates Area of No Vision
	<p><b>Blind Right Eye (right optic nerve)</b>                      A lesion of the optic nerve and, of course, of the eye itself, produces unilateral blindness.</p>	<p>1 </p>
	<p><b>Bitemporal Hemianopia (optic chiasm)</b>                      A lesion at the optic chiasm may involve only those fibers that cross over to the opposite side. Because these fibers originate in the nasal half of each retina, visual loss involves the temporal half of each field.</p>	<p>2 </p>

	<p><b>Left Homonymous Hemianopia (right optic tract)</b>                  A lesion of the optic tract interrupts fibers originating on the same side of both eyes. Visual loss in the eyes is therefore similar (homonymous) and involves half of each field (hemianopia).</p>	<p>3 </p>
	<p><b>Left Homonymous Quadrantic Defect (optic radiation, partial)</b>                  A partial lesion of the optic radiation may involve only a portion of the nerve fibers, producing, for example, a homonymous quadrantic defect.</p>	<p>4 </p> <p>5 </p>



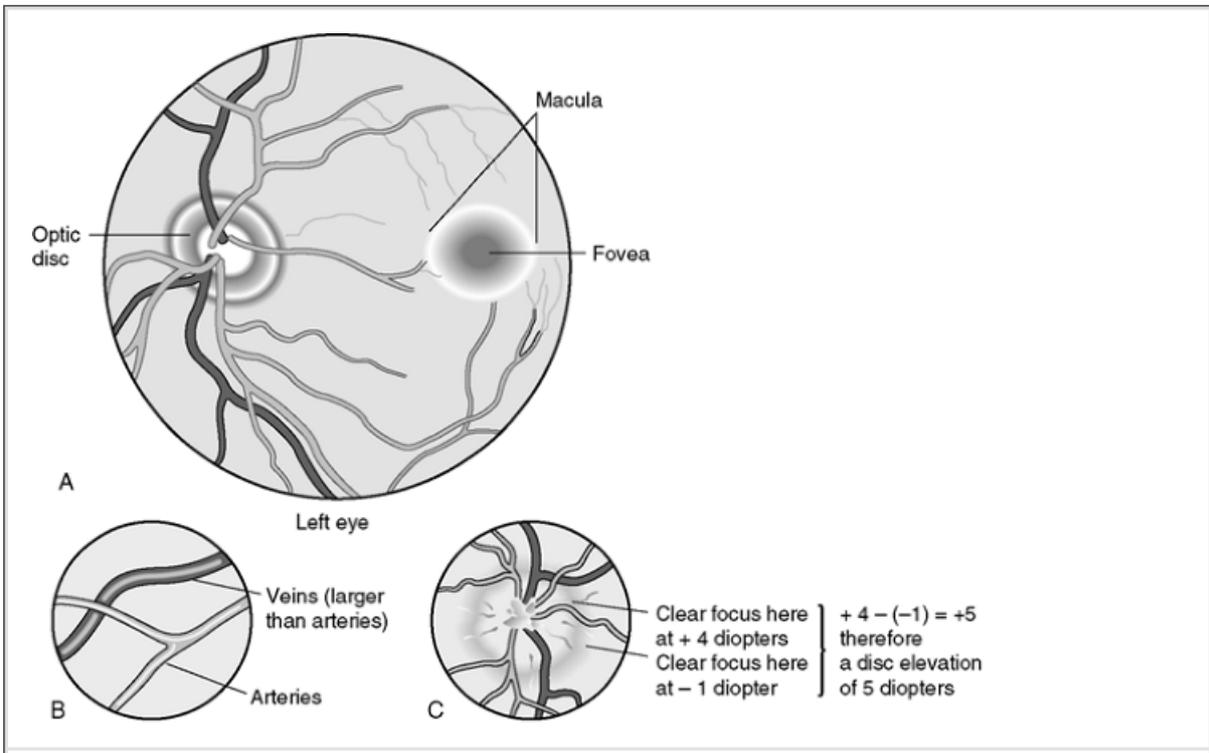
Cortical blindness with complete or severe loss of vision is called **Anton's syndrome**. Often the patient is unaware or denies the blindness. Normal pupillary responses remain intact. The most common cause of cortical blindness is bilateral occipital lobe infarctions.

### Ophthalmoscopic Examination

The **ophthalmoscopic examination** is conducted with an ophthalmoscope, which contains a special lens that is used to visualize the retina by shining a beam of light directly into the eye. The room must be darkened and the examiner must sit directly opposite the subject. To examine the patient's right eye, the examiner holds the ophthalmoscope in the right hand and looks through the instrument with the right eye while the patient focuses on an object straight ahead (Fig. 7-5). The diopter is adjusted to visualize the optic disc (fundus), macula, and blood vessels (Fig. 7-6). The termination of the optic nerve, visible as a prominent, tubelike structure at the back of the eyeball, is called the **optic disc**. As one views the optic disc, small blood vessels can be visualized as they exit and enter the eye. The normal disc is round or slightly oval with sharply defined margins. The outer portions of the disc are elevated slightly above the center, or physiologic cup.



**Figure 7-5** • Technique for the proper use of the ophthalmoscope. The examiner holds the ophthalmoscope with the **right** hand and uses the **right** eye to look into the patient's eye. The index finger is used to adjust the lens for proper focus. For a **myopic** (nearsighted) patient, turn the lens control counterclockwise to the minus diopter; for a **hyperopic** (farsighted) patient, turn the lens control clockwise to the plus diopter.



**Figure 7-6** • The retina as seen through the ophthalmoscope. The ophthalmoscope is capable of visualizing *only a portion of the retina* at any one time. In examining the retina (A) identify the disc; ascertain the sharpness of the disc margins; identify the macula and fovea (bright reflection in the center); (B) compare the diameters of the veins to the arteries; follow the vessels in each quadrant from the disc to the periphery; and note any exudate, hemorrhagic areas, or abnormalities present. **Measurements within the eye.** Lesions are recorded in relationship to the optic disc as **disc diameters**. For example, in A, the macula is about 2 disc diameters from the disc. (C) For an elevated optic disc due to papilledema, note the **differences in diopters** measurement of the two lenses used to focus clearly on the disc and on the uninvolved retina.

The four main pairs of blood vessels exiting and entering the optic disc are examined to compare the diameters of the arteries and veins and to determine whether the veins are tortuous. Normally, the diameter of the veins is about 30% greater than the diameter of arteries in a ratio varying from 2:3 to 4:5. Dilated veins are present with increased intracranial pressure. Another area of the retina is the **macula lutea**, which has the highest density of visual receptors. The center of the macula, called the **fovea**, represents the point of greatest visual acuity. As the retina is examined, any abnormalities, such as hemorrhage, swelling, and exudate, should

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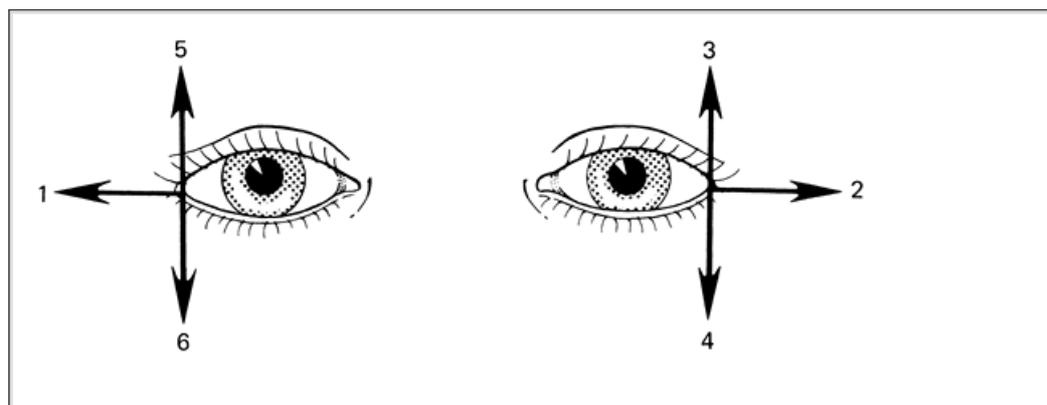
be noted. Table 7-7 summarizes common ophthalmological findings.

TYPE	DESCRIPTION	CAUSES
Papilledema (choked disc)	Margin of the disc is distorted and swollen; the disc has a reddish hue because of congestion	Increased intracranial pressure; pseudotumor cerebri
Optic atrophy	Paleness (the disc is light pink, white, or gray owing to decreased blood supply); visual acuity is decreased	Primary causes: tabes dorsalis and multiple sclerosis Secondary causes: neuritis or prolonged increased intracranial pressure
Retrobulbar neuritis	Inflammatory lesion of the posterior portion of the optic nerve; no swelling of disc	Hemorrhage, diabetes, or retinitis
Optic neuritis	Inflammation of the disc; associated with loss of vision secondary to a central scotoma	Inflammatory process
Cotton wool patches (seen during fundus examination)	Whitish or grayish oval lesions that look like cotton balls; smaller than disc	Hypertension

**Table 7-7** • COMMON ABNORMAL OPTIC DISC FINDINGS

**Oculomotor (CN III), Trochlear (CN IV), and Abducens (CN VI) Nerves (All Motor)**

Cranial nerves III, IV, and VI are tested together because all three supply the extraocular eye muscles (Fig. 7-7). In addition, the oculomotor nerve controls the levator palpebrae superioris muscle, which raises the upper eyelid, and the parasympathetic innervation to the pupil (which causes constriction of the pupil).



**Figure 7-7** • Sequence of eye-testing movements. (Note: Numbers indicate the appropriate sequence of movement.)

Injury to an extraocular muscle compromises the corresponding extraocular movement (EOM). Injury to the oculomotor nerve can result in an inability to focus the eyes medially on the horizontal plane, upward and outward, downward and outward, and upward and inward. A damaged trochlear nerve results in compromised downward and inward movement of the eye, whereas injury to the abducens nerve causes loss of lateral ocular movement on the horizontal plane. Trochlear nerve dysfunction is rare. The abducens nerve has the longest intracranial course and is frequently involved

with neurological disease.

## Observations Related to the Eye and Eye Movement

In assessing CNs III, IV, and VI, begin with focused observations on the position of each orbit, upper eyelid, and pupil. Next, examine EOMs for gaze palsy, internuclear ophthalmoplegia, diplopia, and nystagmus. **Gaze** is the act of focusing in a particular direction. It includes coordinated eye and head movements and is the result of complex reflexes of the visual and vestibular systems and the cerebral cortex. Terms related to the eyes are defined in a later section.

### Position of Eyes, Eyelids, and Pupils

The **position of the eyes** is noted by looking at the position of the eyeball from frontal and lateral views, as well as by looking down from above the patient's head. Abnormal protrusion of

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one or both eyeballs is termed **proptosis** or **exophthalmos**. Abnormal recession of an eyeball within the orbit is termed **enophthalmos**. In assessing the **upper eyelid**, the patient is asked to look straight ahead. The width of the palpebral fissure in each eye is noted and a comparison is made. The **palpebral fissure** is the space between the upper and lower eyelids. One lid may droop compared with the other. The term for a drooping upper eyelid is **ptosis**. Note also edema of the eyelid, if present. Edema can result from trauma to the eye or orbit and may occur in the upper eyelid, the lower eyelid, or both. Next, the position of the eyelids in relation to the pupil and the iris in each eye is noted and compared. This also helps the examiner to note a slight ptosis.

### EOM Evaluation

The six EOM muscles are evaluated next. Ask the patient to follow the examiner's finger or a pencil through the six cardinal directions of gaze in an "H" sequencing (see Fig. 7-7). Observe for full, symmetrical movement of both eyes. See subsequent section for related terms.

### Diplopia

In double vision (**diplopia**), when the eyes move in a particular direction (left or right, or up or down), visual images fall on the retina in different points, rather than on the same point on each retina. The lack of parallelism can cause double vision when the patient focuses in a particular direction. The patient is asked whether diplopia is present. If the answer is yes, is the double image side by side or one on top of the other? If the image is one on top of the other, CN III (oculomotor) or CN VI (trochlear) is involved. If the images are side by side, the examiner needs to determine whether a dysfunction in CN III or VI (abducens) is the cause. Have the patient follow your finger to the left and right on the horizontal plane. Is the diplopia worse when the patient turns to the left or the right? The deficit is on the side where the diplopia is

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worse. If the images are one on top of the other, the examiner needs to determine if a dysfunction in CN III or IV is the cause. Have the patient follow your finger up and down both on the inward (inner aspect of the eye) and outward (outer aspect of the eye) on the vertical plane. Is the diplopia worse when the patient looks up down? The deficit is in the direction that is worse. Note that trochlear involvement is uncommon.

### Pontine and Frontal Gaze Centers

When considering abnormalities of eye movement, differentiate between pontine and frontal gaze palsy. Acute damage in the **frontal gaze center** causes the eyes to **deviate toward the side of the lesion** (away from an accompanying hemiplegia). The palsy is usually temporary and tends to resolve in minutes to hours. **Pontine gaze palsies** are usually bilateral and symmetrical. They cause eye **deviation away from the side of the lesion** and last much longer than frontal gaze palsies.

**Internuclear ophthalmoplegia (INO)** is due to a lesion of the medial longitudinal fasciculus resulting in impairment of adduction in the ipsilateral eye and nystagmus in the contralateral eye on gaze away from the side of involvement. The lesion lies between the nuclei of CN VI and opposite CN III. Convergence is intact, thus ruling out a lesion of CN III. There is no diplopia. The most common cause in the elderly is brainstem infarction and multiple sclerosis in younger people. Other causes include trauma and neoplasms. Occasionally INO is associated with metabolic disorders such as hepatic encephalopathy and may be seen in myasthenia gravis. Table 7-8 summarizes other eye movement abnormalities.



ABNORMALITY	DESCRIPTION	CAUSE/LESION
Wall-eyed bilateral internuclear ophthalmoplegia (INO) "One-and-a-half" syndrome	Bilateral outward eye deviation that occurs with bilateral INO	Pontine infarction or demyelination
Ocular bobbing	Impairment of all horizontal conjugate eye movements, except for abduction of the contralateral eye	Parapontine reticular formation (PPRF)
Skewed deviation	Episodic, intermittent, usually conjugate, downward, brisk eye movement followed by a return to the resting position by a "bobbing action"	Severe destructive lower pontine lesions, pontine hemorrhage, or infection
Parinaud's syndrome	Misalignment of the eyes observed in the vertical plane that is not due to a lesion of the third or fourth cranial nerve. Vertical diplopia results	Pons, usually on side of the higher eye, but may be in the cerebellum
	Paralysis of conjugate upward gaze; also includes paresis of convergence, convergence-retraction nystagmus, pupillary hyporeactivity, and light-near dissociation	Midbrain tectal area; often caused by a pinealoma, a vascular lesion, or hydrocephalus

Table 7-8 • EYE MOVEMENT ABNORMALITIES WITH POSTERIOR FOSSA LESIONS

### Terms Related to Eye Movement

- **Conjugate gaze:** both eyes move in the same direction, at the same speed, and in constant alignment
- **Dysconjugate gaze:** lack of alignment between the two visual axes
- **Strabismus:** deviation of one's eye from its proper direction so that the visual axes of the two eyes cannot both be directed simultaneously on an object (lack of alignment of ocular axes)
- **Oculocephalic reflex** (doll's eye response) and the **oculovestibular reflex** (caloric stimulation) are used in the unconscious patient and provide information about the presence or absence of brainstem reflexes. Both are discussed later in this chapter.
- **Ophthalmoplegia:** paralysis of the eye muscles; Table 7-9 summarizes types of ophthalmoplegias

TYPE OF OPHTHALMOPLEGIA	DESCRIPTION
External	Paralysis of one or more of the extraocular muscles
Internal	Paralysis of one or more intraocular muscles
Nuclear	Paralysis caused by a lesion involving the nuclei of the motor nerves of the eye (cranial nerve III, IV, or VI)
Internuclear	Paralysis caused by injury or a lesion of the medial longitudinal fasciculus located within the brain stem
Supranuclear	Paralysis caused by injury or a lesion to the conjugate eye movement centers located in the frontal, frontal-parietal, and occipital lobes
Parinaud's syndrome or dorsal midbrain syndrome	A type of supranuclear ophthalmoplegia; conjugate upward gaze paralysis; caused by a lesion in the dorsal midbrain

Table 7-9 • TYPES OF OPHTHALMOPLEGIAS

- **Diplopia:** double vision; seeing two separate images of the same object in visual space, with one of the images displaced from the other; the images can be side by side or one on top of the other; diplopia is usually preceded by blurring of vision.
- **Nystagmus:** a common, involuntary, rhythmic to-and-fro oscillation of the eyes that may be horizontal, vertical, rotary, or mixed in direction. The tempo of the movements can be regular, rhythmic, pendular, or jerky, with a noted fast and slow movement component. True nystagmus

is a pathologic condition caused by a lesion in the central or peripheral vestibular pathways or their cerebellar connections as well as toxic-metabolic disorders, weakness of gaze, and blindness (Table 7-10).

TYPE OF NYSTAGMUS	DESCRIPTION	LOCATION OF LESION	COMMENT
Gaze-evoked nystagmus	With normal eye movement, there may be three or fewer beats at the extremes of eye position; fast phase in direction of gaze	—	Normal finding
Optokinetic nystagmus	Rapid, alternating motion of the eyes normally noted when eyes try to fixate on a moving target; presence indicates physiologic continuity of the optic pathways from the retina to the occipital cortex. Because it is involuntary, a positive response provides reliable verification of intact vision in a patient feigning blindness.	—	Normal finding
Retraction nystagmus	Irregular jerks of the eyes backward into the orbit, precipitated by upward gaze	Midbrain tegmentum	—
Convergence nystagmus	Slow, spontaneous, drifting, ocular divergence with a final quick, convergent jerk	Midbrain	—
See-saw nystagmus	Rapid, pendular, dysconjugate see-saw movement accompanied by deficits of visual fields and visual acuity	Proximal optic chiasm	—
Downbeat nystagmus	Irregular jerks precipitated by downward gaze	Lower medulla	—
Vestibular nystagmus	Mixed nystagmus that can be horizontal, rotational, or both, resulting from vestibular disease	Vestibular pathway	—
Toxic nystagmus	Multidirectional gaze-evoked nystagmus; most often drug induced with toxic levels of phenytoin (Dilantin), barbiturates, and bromides; can also result from cerebellar or central vestibular dysfunction	—	Usually due to drug toxicity

Table 7-10 • COMMON TYPES OF NYSTAGMUS

## Other Findings

Trauma to the peripheral portion of the CN can result in nerve fiber degeneration, followed by unpredictable regeneration. Because of the proximity of other CNs to each other, the regenerating fibers from one nerve can be misdirected to a nearby cranial nerve, forming connections with adjacent nerves. Such an atypical regeneration is called a **misdirection syndrome**. Two are mentioned—the pseudo-Graefe lid sign and Gunn's syndrome. The **pseudo-Graefe syndrome** is an atypical connection between CNs III and IV. Any attempt to look downward is followed by upper eyelid retraction, medial rotation of the eye, and constriction of the pupil. **Gunn's syndrome** involves CNs III and V. As the mouth is opened and the jaw moves to one side, ptosis of the eyelid changes to lid retraction.

## Examination of the Pupils

To assess the pupils, the patient should be directed to focus on a distant object located straight ahead. (With a comatose patient, the pupils are examined however they are found.) The pupils are examined for size, shape, and equality. The normal diameter of a pupil is 2 to 6 mm, with an average diameter of 3.5 mm. When the pupils are compared with each other, their diameters should be equal; however, about 12% to 17% of the normal population has discernibly unequal pupil size (**anisocoria**) in the absence of a pathological condition.

The shape of the pupils is also noted and compared. Normally, the pupils are round; however, in patients who have had cataract surgery, the pupils assume a keyhole shape. An ovoid pupil indicates pupillary dysfunction; it may be seen in early uncal herniation.

Sensitivity to light or **photophobia** may be noted when checking the pupils, or the patient may complain of this problem. The etiology is unclear, but the finding is associated with conditions such as meningitis.

## Pupillary Reflexes

A few important reflexes relating to pupillary responses and eye movement can be tested.

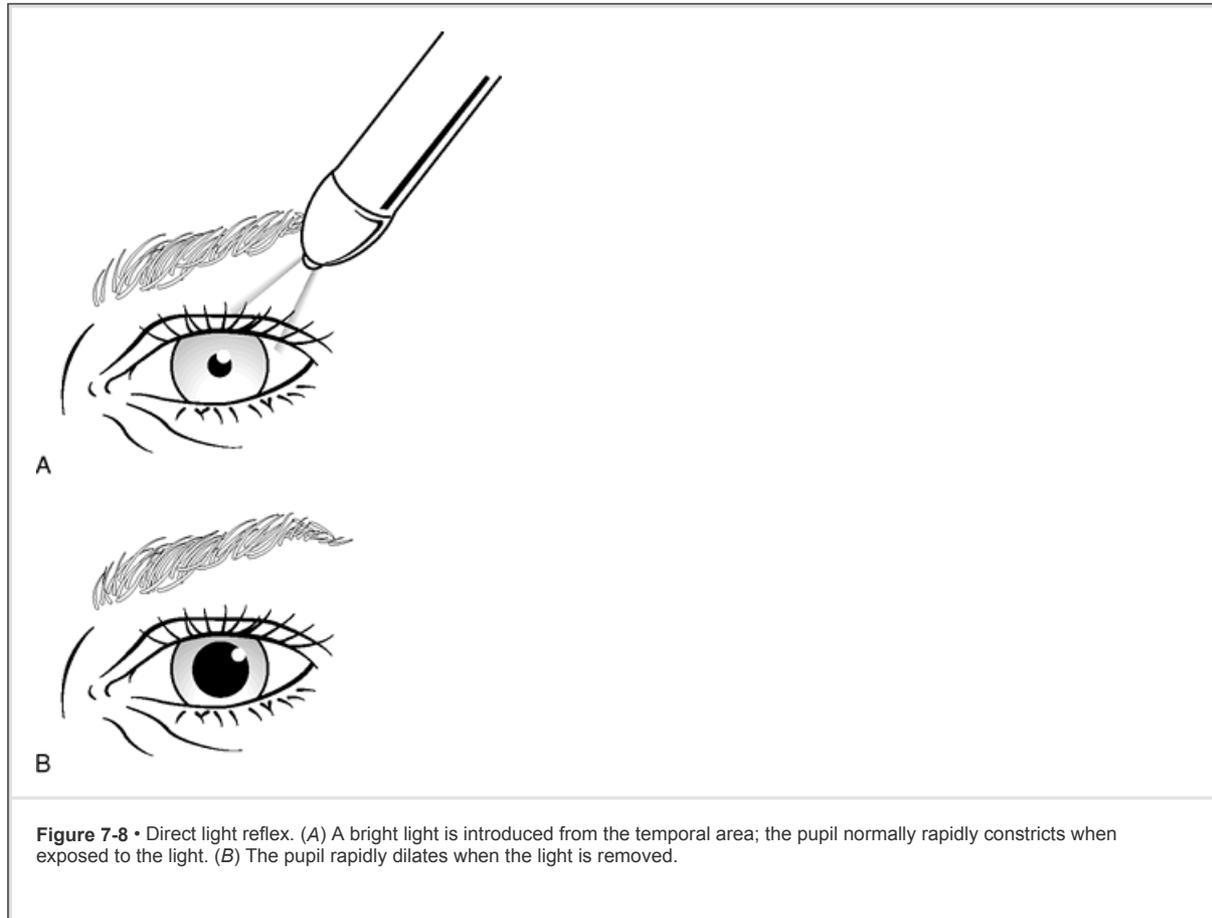
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### Direct Light Reflex

The sensory receptors for the light reflex are the rods and cones of the retina. Afferent impulses follow the normal visual pathway as far as the lateral geniculate bodies. Rather than entering the geniculate body, sensory impulses enter the pretectal area (near the superior colliculus). Connecting neurons synapse in the **Edinger-Westphal nucleus** (oculomotor nucleus) located in the midbrain. From here, the parasympathetic fibers proceed to the ciliary ganglion to the pupilloconstrictor fibers of the iris, causing the pupil to constrict. When the light is withdrawn, the pupil normally dilates because of sympathetic stimulation. A three-neuron chain begins in the posterior hypothalamus. The first neuron descends dorsally in the brainstem and cervical spinal cord to synapse in the **intermediolateral cells** at the C7 to C12 levels. The second neuron leaves the nervous system through the ventral spinal roots and ascends in the sympathetic chain and synapses in the superior cervical ganglion (at level of carotid bifurcation). The third neuron reaches the iris and Müller's muscles by ascending along the internal carotid artery, then traverses the cavernous sinus and the ciliary ganglion as the long ciliary nerves. The postganglionic sympathetic fibers synapse with the pupillodilator muscles, resulting in pupillary dilation.

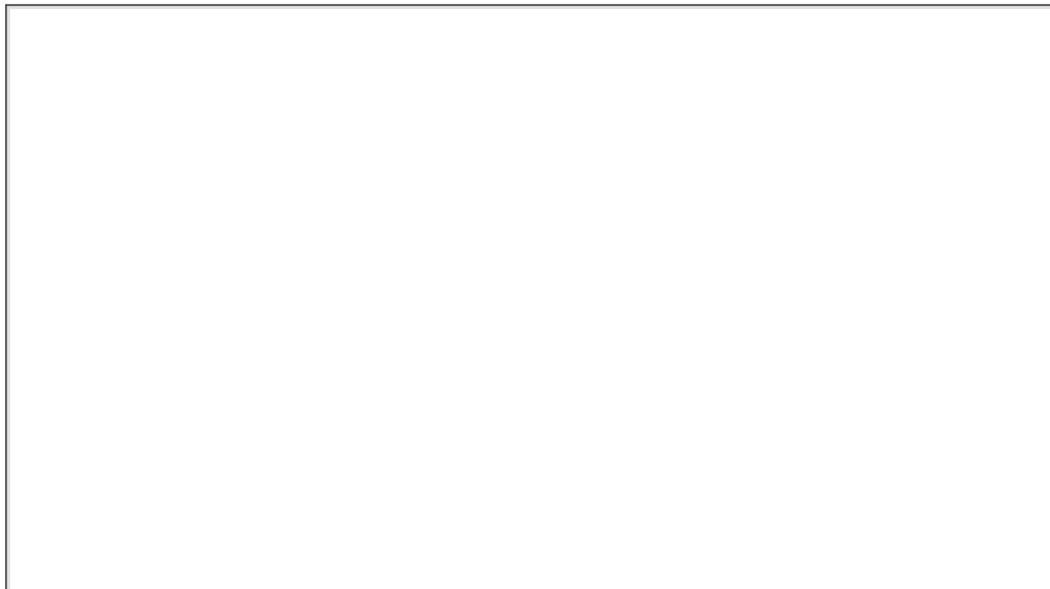
The direct light reflex refers to the constriction and dilation of the pupil when a light is shone into that pupil and

withdrawn (Fig. 7-8). Each pupil is tested individually and the results are compared. Response to light is recorded as:



- **Brisk:** very rapid constriction when light is introduced
- **Sluggish:** constriction occurs but more slowly than expected
- **Nonreactive or fixed:** no constriction or dilation is noted

Normally, pupillary constriction is brisk, although age may affect the briskness of reaction. Pupils in younger people tend to be larger and more responsive to light than those in older people. In addition to the constriction of the pupil that occurs with direct light stimulation (direct light response), there is a somewhat weaker constriction of the nonstimulated pupil. This is called the **consensual light reflex**, which occurs as a result of fibers crossing from each side that cross both the optic chiasm and the posterior commissure of the midbrain. See Table 7-11 for pupillary abnormalities.





**Table 7-11 • PUPILLARY ABNORMALITIES CLASSIFIED BY AFFERENT AND EFFERENT PUPILLARY DEFECTS**

### ***Near-Point Reaction***

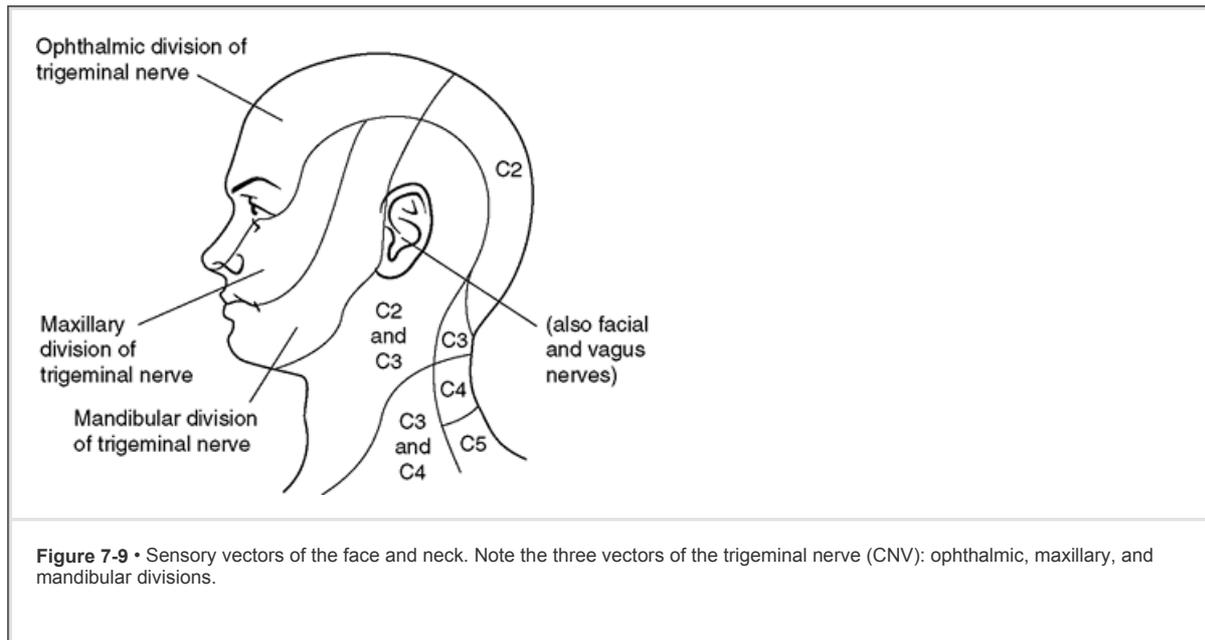
In testing the near-point reaction, patients are asked to focus on the examiner's finger, which is positioned 2 to 3 feet directly ahead of them, and to follow it with their eyes as it is rapidly moved closer to their face. Three reflex responses normally occur:

- **Convergence:** the medial recti muscles contract, thus directing both eyes toward the midline. This occurs so that the image in each eye will remain focused on the fovea; without this reflex, diplopia would occur.
- **Accommodation:** to sharply focus the image on the fovea, the lenses thicken as a result of tension in the ciliary muscles; the ciliary muscles are innervated by the postganglionic parasympathetic neurons in the ciliary ganglion.
- **Pupillary constriction:** the pupils constrict as an optic adjustment to regulate depth of focus. (This pupillary constriction does not depend on light and is regulated separately from the light reflex.)

### ***Trigeminal Nerve (Mixed) (CN V)***

The trigeminal nerve is composed of both sensory and motor components. To assess the sensory component, the three

sensory vectors of the face are tested (Fig. 7-9). The **ophthalmic division** innervates the frontal sinuses, the conjunctiva and cornea, the upper lid, the bridge of the nose, the forehead, and the scalp as far as the vertex of the skull. The **maxillary division** innervates the cheek, the maxillary sinus, the lateral aspects of the nose, the upper teeth, the nasal pharynx, the hard palate, and the uvula. The **mandibular division** innervates the chin, the lower jaw, the anterior two thirds of the tongue, the gums and floor of the mouth, and the buccal mucosa of the cheek.



With the patient's eyes closed, ability to appreciate **light touch** is tested by touching the forehead, cheek, and jaw with a wisp of cotton. The patient is instructed to respond every time the skin is touched. **Pain perception** is evaluated with the "picky" (broken) and dull ends of a wooden applicator or by the pinprick method. After demonstrating the difference between sharp and dull, the skin is touched with the sharp end and occasionally with the dull end. The patient is asked to

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respond "sharp" or "dull." Accuracy of response and a comparison between findings on each side of the face is made.

Next, the motor component (i.e., the muscles of mastication) is evaluated. The strength of the masseter and temporal muscles is evaluated by palpating them when the jaw is clenched and opened. Differences in muscle tone or atrophy should be noted.

### **Facial Nerve (Mixed) (CN VII)**

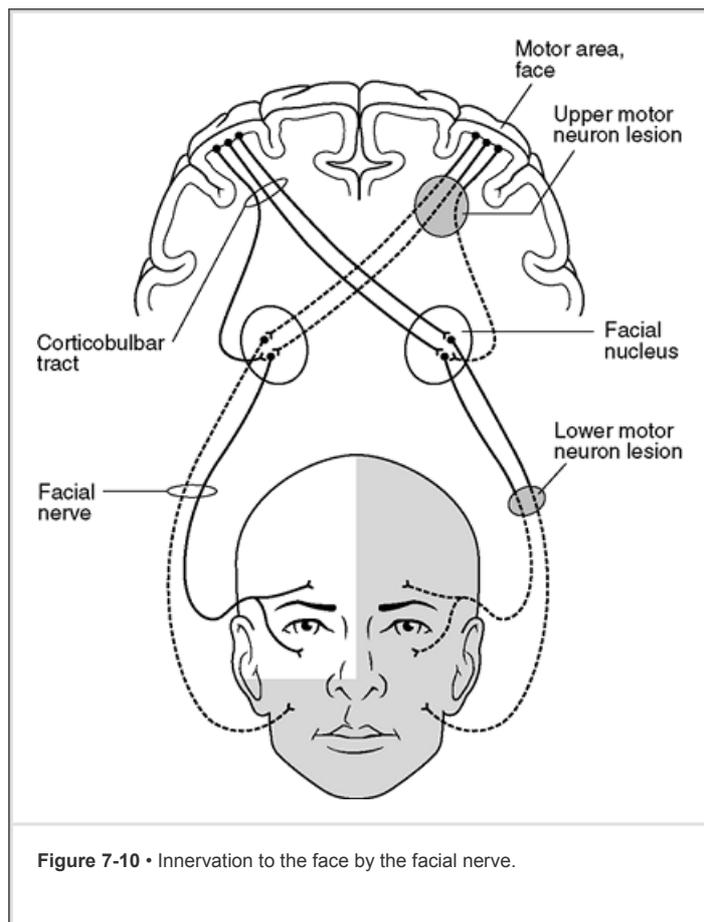
The facial nerve has both sensory and motor components. The sensory component includes the sense of taste on the anterior two thirds of the tongue. Testing of this function is often deferred. However, if tested, each side of the protruded tongue is tested separately. There are four basic modalities of taste: sweet (tip of tongue), sour (sides of tongue), salty (over most of tongue but concentrated on the sides), and bitter (back of tongue, controlled by CN IX). The patient is asked to identify the taste of sugar placed on the tip of the tongue, after which a sip of water is given. The same procedure is used with sour and salty substances. If bitter is tested on the posterior third of the tongue, it should be recognized that it is innervated by the glossopharyngeal nerve. Sensation to the external ear is also supplied by the facial nerve.

The motor component is tested by observing the symmetry of the face at rest and during deliberate facial movements, such as smiling, showing the teeth, whistling, pursing the lips, blowing air into the cheeks, wrinkling the nose and forehead, and raising the eyebrows. Note the nasolabial folds for symmetry. In addition, ask the patient to close the eyes tightly. The examiner should not be able to open the patient's eyes. The facial nerve also controls tearing and salivation.

When weakness is noted, it is important to observe whether the entire side of the face or just the lower face (below the eyes) is affected.

There are two types of facial weakness (Fig. 7-10). If the lower portion of one side of the face is involved, the lesion is said to be caused by a central (involves the central nervous system) problem, an **upper motor neuron lesion** (as seen in association with stroke). This involves the corticobulbar tracts, resulting in contralateral weakness of the *lower face* with normal function of the upper face. Wrinkling of the forehead is left intact because of bilateral innervation of the upper face from the corticobulbar fibers. The lower face, by contrast, has only unilateral contralateral cortical innervation so that retraction of the corner of the mouth to smile is compromised. The second type of facial weakness

involves the *total side face* with ipsilateral facial muscle involvement. The lesion is said to be peripheral (involves the peripheral nervous system) or a **lower motor neuron lesion**. Specifically, the condition is called **Bell's palsy**.



In addition to muscle weakness, note any evidence of spasms, atrophy, or tremors of the facial muscles.

### **Acoustic Nerve (Sensory) (CN VIII)**

The acoustic nerve is a pure sensory nerve and is divided into two branches, the **cochlear nerve** for hearing and the **vestibular nerve**, which contributes to equilibrium, coordination, and orientation in space.

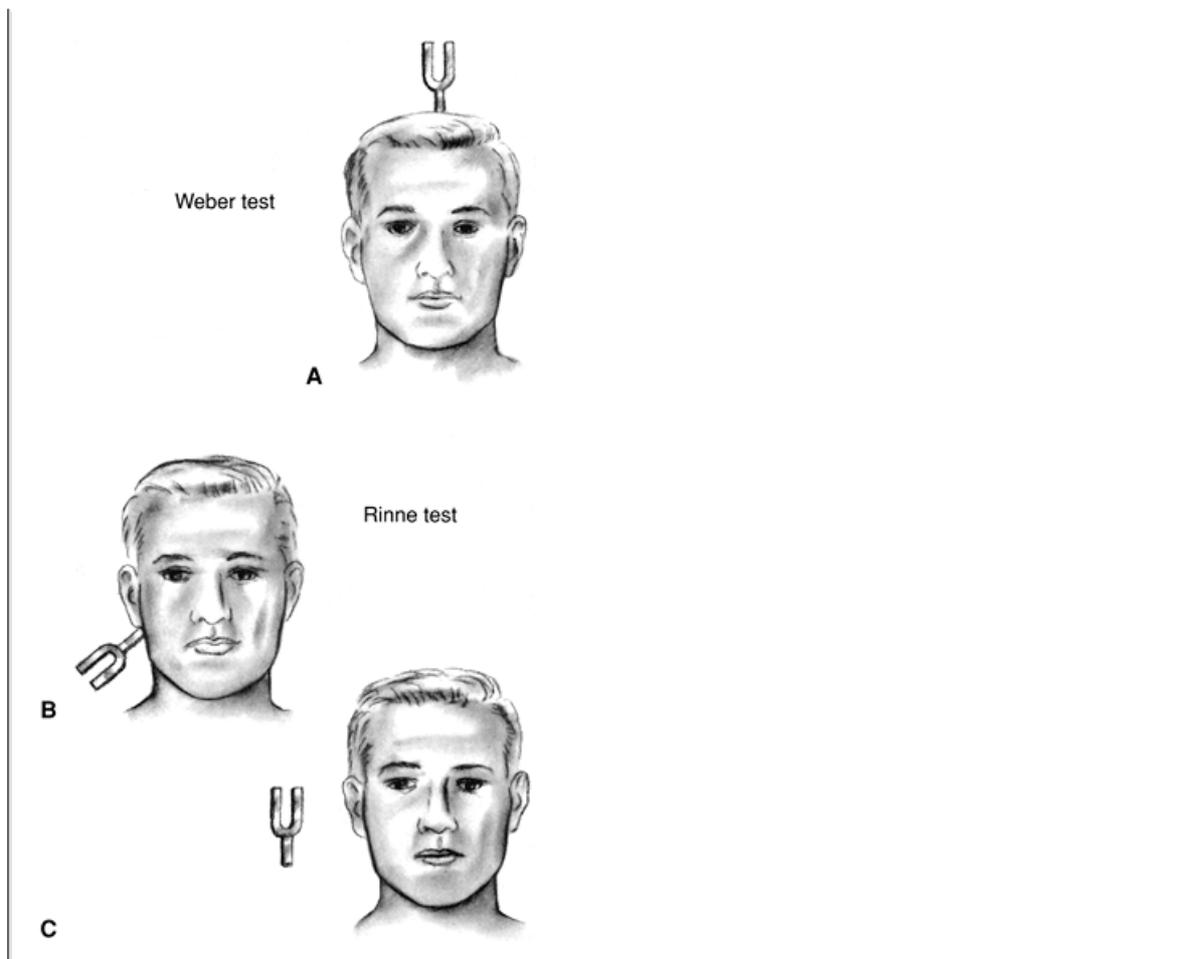
Hearing is evaluated in several ways. Much information can be collected in observing the patient. Ability to understand soft and loud tones and low and high pitches is noted. Signs of hearing deficit include inability to hear high or low tones, turning the head toward the speaker when listening, or lip reading. Certain sounds are heard more loudly and at a greater distance. For example, *a*, *e*, and *i* are heard at a greater distance than consonants such as *l*, *m*, and *r* and vowels such as *o* and *u*. "Seventy-six" and "sixty-seven" can be heard at a greater distance than "ninety-nine." To test **hearing**, direct the patient to cover one ear. Standing on the opposite side, 1 to 2 feet away from the ear, whisper a few numbers or a word. An alternative is to rub your fingers

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together. The patient should be able to hear it. Test the other ear. Hearing should be equal in both ears.

Next, check for **lateralization** and **air** and **bone conduction**. **Weber's test** is used to evaluate lateralization, whereas **Rinne's test** evaluates air and bone conduction. Both tests require the use of a 512-Hz tuning fork (Fig. 7-11). For **Weber's test**, place a lightly vibrating tuning fork firmly vertex of the patient's head or in the middle of the forehead. Inquire whether the patient hears the vibration more on the left side, the right side, or in the middle. Normally, the sound is heard in the middle or equally in both ears. If one ear is occluded, the vibration is heard better or more loudly in the occluded ear.





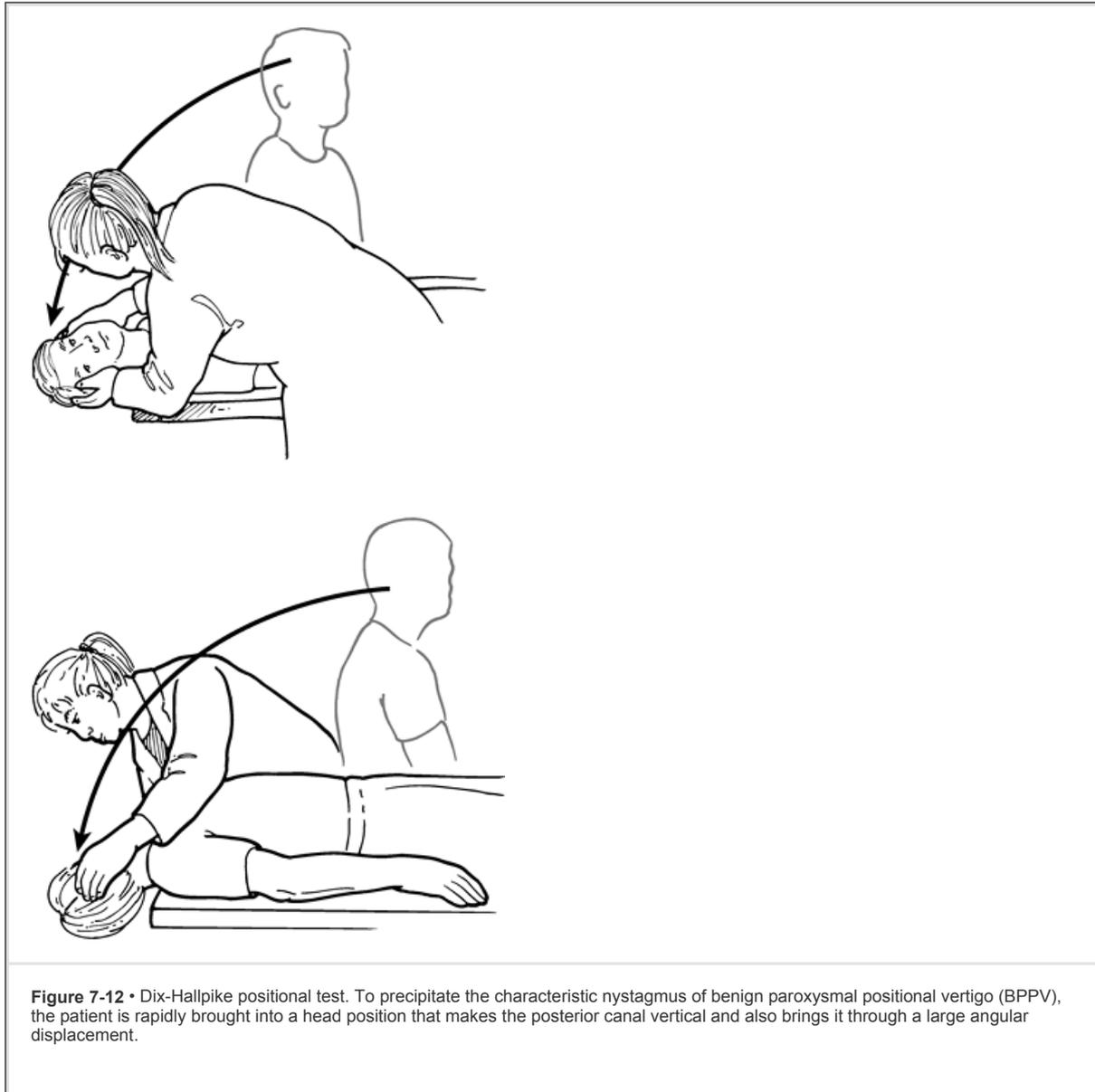
**Figure 7-11** • Weber test and Rinne test. Note the placement of a vibrating tuning fork (512 Hz) for each test. (A) Weber test for lateralization. Place the vibrating fork firmly on the top of the head or on the middle of the forehead. Normally the sound is perceived midline. Rinne test for comparing bone and air conduction: Place the base of the vibrating tuning fork on the mastoid process until the patient can no longer hear it (B); then, quickly place the fork near the external auditory canal, with one side toward the ear (C). Normally, the sound can be heard longer through air than through bone (AC > BC).

In **Rinne's test**, air conduction and bone conduction are compared. Place the base of a lightly vibrating tuning fork firmly on the mastoid process. Ask the patient to inform you when the vibration is no longer heard. Quickly place the vibrating fork near the ear canal with the vibrating portion toward the ear. Ask the patient to inform you when it can no longer be heard. Normally, the sound should be heard longer through air than through bone (AC greater than BC). In evaluating hearing acuity, it is important to differentiate between **conduction loss** and **sensorineural loss**. The findings for unilateral loss for each are as follows:

TYPE OF DEAFNESS		
TYPE OF DEAFNESS	RINNE	WEBER
Conduction deafness	BC > AC	Lateralizes <b>to</b> deafer ear
Sensorineural deafness	AC > BC	Lateralizes <b>away from</b> deafer ear

Abnormal findings from either test warrant further investigation with more sensitive testing.

In considering the vestibular nerve, certain tests of vestibular function are closely related to the examination of other portions of the nervous system (i.e. motor system and cerebellar testing). However, all patients with a major complaint of vertigo should receive a **Dix-Hallpike positional test**. It consists of seating the patient on the edge of an examining table and moving him backward rapidly so that the head is hanging backward while the eyes remain open. If no nystagmus or vertigo develops in 20 seconds, the patient is returned to the sitting position. The head is repositioned to the right, and the downward procedure repeated. If no nystagmus or vertigo develops in 20 seconds, return patient to the sitting position. Reposition the head to the left and repeat the procedure (Fig. 7-12). Note the onset, duration, and direction of the nystagmus. In benign paroxysmal positional vertigo (BPPV) nystagmus is noted when the head is turned to either side. The nystagmus beats upward and also has a rotatory component so that the top part of the eye beats toward the down ear. The characteristics of the nystagmus are a latency of 2 to 5 seconds, a duration of 5 to 60 seconds, and it is followed by a downbeating nystagmus when the patient is placed upright in the sitting position.<sup>7</sup> Further evaluation will be required with persistent vertigo.



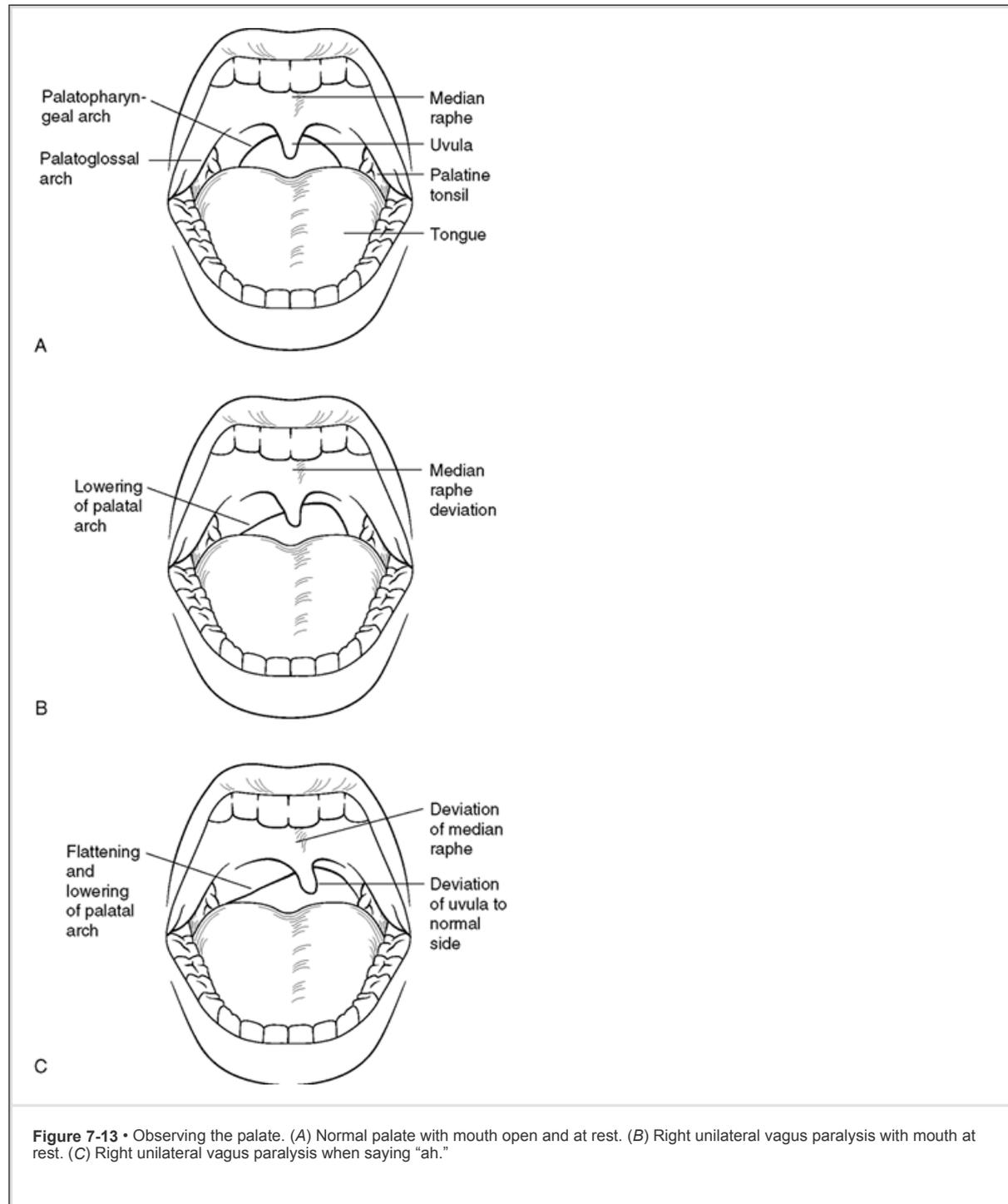
### ***Glossopharyngeal (CN IX) and Vagus (CN X) Nerves (Both Mixed)***

The glossopharyngeal and vagus nerves are tested together because their intimate association of function in the pharynx.

The glossopharyngeal nerve supplies sensory components to the pharynx, tonsils, soft palate, tympanic membrane, posterior third of the tongue, and secretory fibers of the parotid gland. It also supplies motor fibers to the stylopharyngeal muscle of the pharynx, whose role is to elevate the pharynx. The patient is asked to open the mouth and say "ah." Upward movement of the soft palate and uvula should be noted. The vagus nerve provides

parasympathetic fibers to the viscera of the chest and abdomen. Sensory fibers innervate the external ear canal, pharynx, larynx, and viscera of the chest and abdomen. The vagus also provides motor control to the soft palate, larynx, and pharynx.

The soft palate is examined with the patient's mouth open. Normally, when the patient says "ah," the palate elevates and the uvula remains midline. Check to see whether the uvula deviates to one side or the other (Fig. 7-13). The gag reflex is innervated by CNs IX and X. After warning the patient that you are going to check the gag reflex, touch the posterior pharyngeal wall with a tongue blade or applicator first on the left side and next on the right side. A gag response should be stimulated on each side.



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Voice quality is evaluated by listening to the patient speak and noting hoarseness; a soft, whispery voice; or no voice. Difficulty in speaking, such as hoarseness or speaking in a whisper, is termed **dysphonia**. Both are due to paralysis of the soft palate (**palatal paralysis**) and can result in nasal-sounding speech. **Dysarthria** refers to defective articulation that may be caused by a motor deficit of the tongue or speech muscles. Slurred speech should be noted, as should difficulty in pronouncing the letters *m*, *b*, *p*, *t*, and *d* and the number *1*.

### ***Spinal Accessory Nerve (Motor) (CN XI)***

The accessory nerve, as it is sometimes called, is tested in two segments. First, the trapezius muscle is palpated and its strength evaluated while the patient shrugs the shoulders against resistance provided by the examiner's hands. Second, the patient is asked to turn his or her head to one side and push the chin against the examiner's hand, thereby allowing the sternocleidomastoid muscle to be palpated and its strength evaluated. The same procedure is then repeated on the other side. The symmetry of the trapezius and sternocleidomastoid muscles is noted, along with any muscle wasting or spasm.

### ***Hypoglossal Nerve (Motor) (CN XII)***

The patient is asked to open his or her mouth. The tongue is first inspected as it lies on the floor of the oral cavity. Note any atrophy or fasciculation of the tongue. Ask the patient to

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protrude the tongue. Note asymmetry, atrophy, or deviation from midline. Next, direct the patient to move the tongue from side to side. Note the symmetry of movement. Finally, ask the patient to push the tongue against the inside of each cheek as the examiner palpates externally on the cheek for strength and equality.

A common cause of dysfunction is found in patients who have had a carotid endarterectomy. Surgery under the angle of the jaw can result in stretching or injury to the hypoglossal nerve. On examination, weakness is noted toward the side of the surgery. If the nerve has been stretched, recovery is expected. If the nerve has been cut, no recovery is expected and atrophy of one side of the tongue will occur.<sup>8</sup>