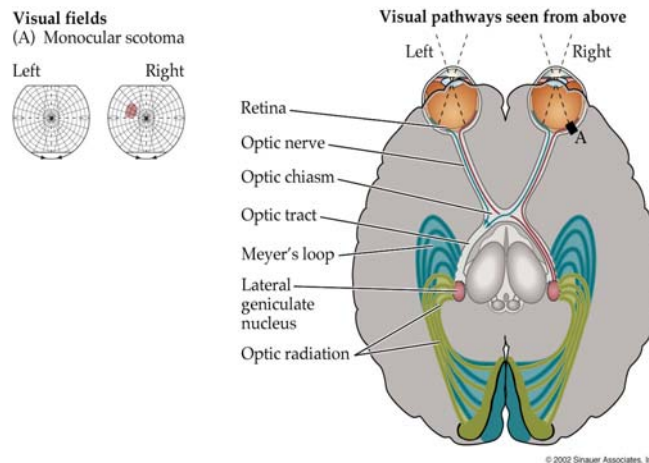


MODULE 5: VISUAL SYSTEM AND CRANIAL NERVES

This module will summarize some of the most important anatomical and key clinical concepts from Chapters 11 and 12 from the book, and thus, you will need to read more from the book than in previous modules to solidify the material in your mind. The first part of Module 5 will review the visual pathways from the retina to visual cortex, examine the retinotopic organization of these tracts, discuss the major types of visual field defects, and present one of the clinical cases from Chapter 11.

The second part of this module is taken from Chapter 12. We will review the functions and course of the cranial nerves, briefly discuss the primary deficits associated with lesions of each cranial nerve, and present an exemplary case from the Chapter as an example.

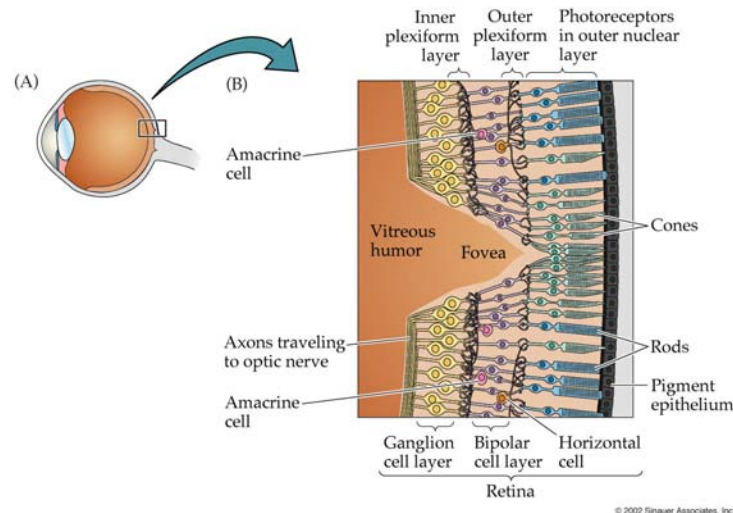
THE VISUAL SYSTEM. The visual pathways begin in the retina, leave the eye as the optic nerve which crosses at the optic chiasm, and then becomes the optic tract. The optic tract travels to the lateral geniculate body of the thalamus, leaves the thalamus to form the optic radiations which go to the visual cortex in the occipital lobes (see figure below).



Eyes and Retina. There are two classes of photoreceptors in the retina: rods and cones. **Rods** are more numerous than cones by a ratio of about 20:1, have relatively poor spatial and temporal resolution of visual stimuli, and do not detect color. The main function of rods is for vision in low-level lighting conditions, where they are far more sensitive than cones.

Cones are less numerous than rods overall, but they are more highly represented in the fovea, where visual acuity is the highest. Cones have high spatial and temporal resolution and detect colors.

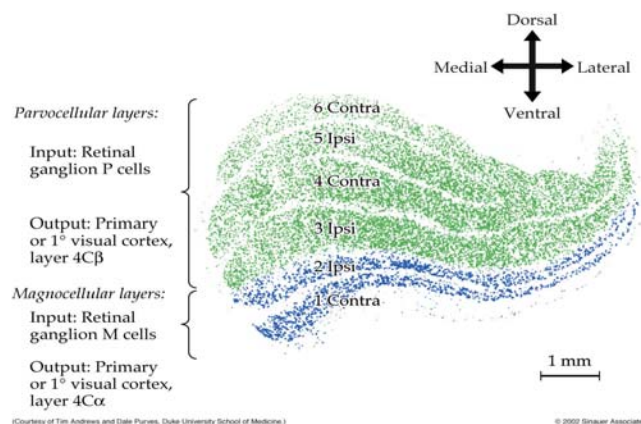
Rods and cones form the outermost layer (farthest from the lens). They respond to light and from excitatory or inhibitory synapses onto **bipolar cells**. Bipolar cells in turn synapse onto **ganglion cells** which send their axons into the optic nerve. The three major layers of the retina are shown in the figure below.



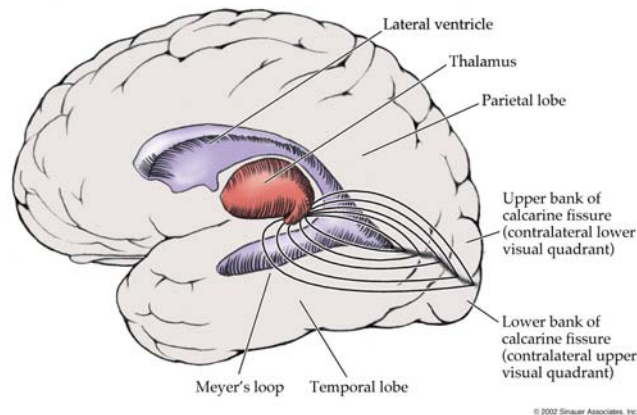
Retinal ganglion cells can be classified as ***M cells***, which have large receptive fields and respond best to gross stimulus features, spatial analysis, and movement, and ***P cells***, which have small receptive fields, are more numerous, and are sensitive to fine visual detail and to colors. M cells are called “M” because they project to the ***magnocellular layer*** in the lateral geniculate body of the thalamus. P cells are named for the ***parvocellular layer*** of the lateral geniculate body of thalamus where they first synapse.

Optic Chiasm. The optic nerve exists at the back of the eye in the region of the optic disc. There is a partial crossing of fibers in the ***optic chiasm***. Thus, fibers from the left hemiretinas of both eyes end up in the left optic tract, while fibers from the right hemiretinas end up in the right optic tract. To accomplish this, the nasal (medial) retinal fibers for each eye (which respond to the temporal [lateral] hemifields, cross over in the optic chiasm (see the figure above on page 1).

Lateral Geniculate Nucleus (or Body). The axons of retinal ganglion cells in the optic tracts from synapses on the lateral geniculate nucleus or body (LGN) of the thalamus. The LGN has 6 layers. From ventral to dorsal, the first (bottom) two are the ***magnocellular layers*** which relay information from M cells. The top four layers of the LGN are the ***parvocellular layers*** which relay information from P cells. These are shown in the figure below.



Optic Radiations to Primary Visual Cortex. The axons leaving the LGN fan out across a wide area of white matter, forming the **optic radiations**. Fibers of the inferior optic radiations arc forward into the temporal lobes, forming **Meyer's loop**. Because Meyer's loop fibers carrying information from the inferior retina (and superior visual field), temporal lobe lesions can cause a **contralateral homonymous superior quadrantanopia**. Conversely, the upper optic radiations travel in the white matter underlying parietal cortex. Therefore, parietal lobe lesions can sometimes cause a **contralateral homonymous inferior quadrantanopia**. Primary visual cortex lies on the banks of the **calcarine fissure** in the occipital lobe (see figure below).

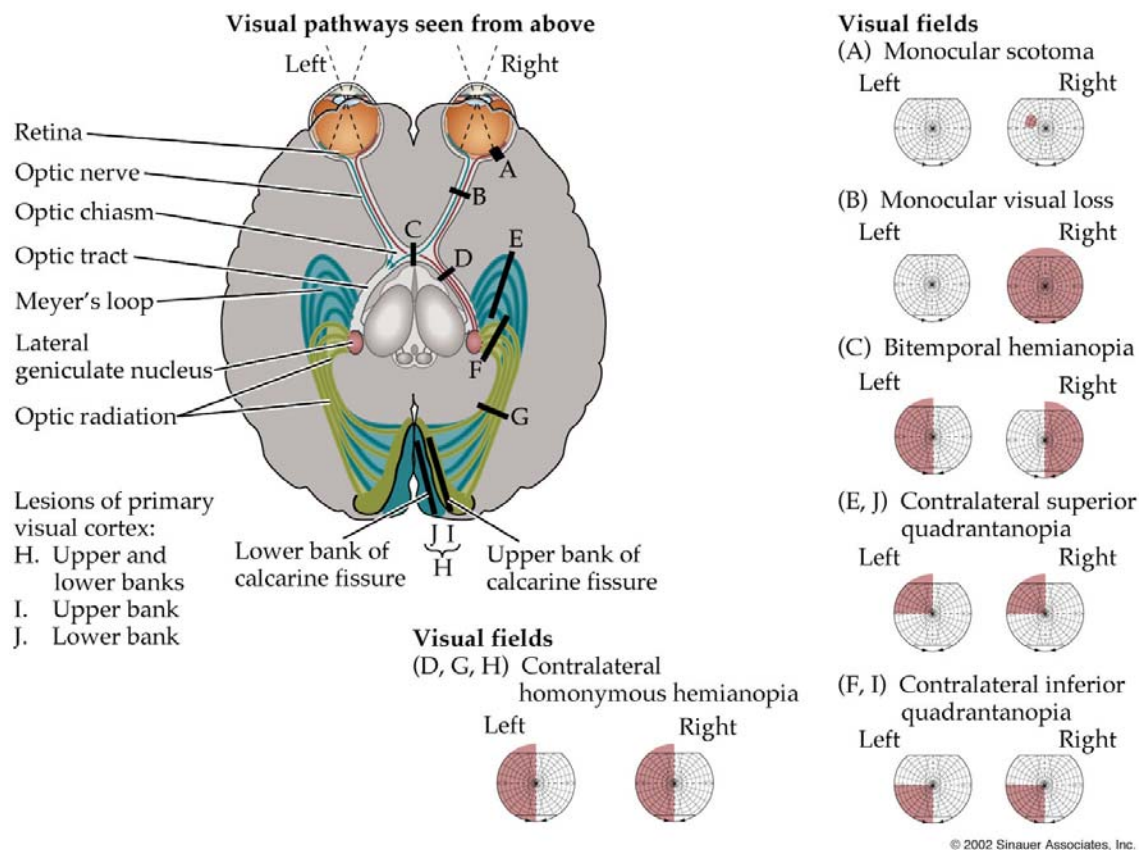


Primary visual cortex, like other parts of the visual system, is **retinotopically** (or visuotopically) **organized**. The region of the fovea is represented near the occipital pole, while more peripheral regions are represented more anteriorly along the calcarine fissure. Because it is the region of highest visual acuity, the fovea has a large cortical representation, occupying about 50% of the primary visual cortex.

Visual Processing in Neocortex. M cells, vital for motion and spatial analysis, and P cells, important for form and color detection, remain segregated in primary visual cortex. Information processing by visual association cortex (Brodmann's areas 18 and 19) and higher-order association cortex (multimodal) takes place via a **dorsal "where?" stream** dedicated to analysis of motion and spatial relations and a **ventral "what?" stream** involved in the analysis of form and color.

Visual Field Defects. Lesions in the primary visual pathways produce characteristic visual field defects (see figure below).

Lesions anterior to the optic chiasm produce monocular defects (see A & B below in the figure).



In the optic chiasm, the nasal fibers for each eye, carrying visual information from the lateral (temporal) visual hemifields, cross to the opposite side. Therefore, visual pathways posterior to the optic chiasm carry information from both eyes corresponding to the contralateral visual field (see lesions/visual fields D – H in figure above).

Lesions of the optic tract, lateral geniculate nucleus, optic radiations, or primary visual cortex therefore cause contralateral homonymous (“same named”) visual field defects affecting both eyes.

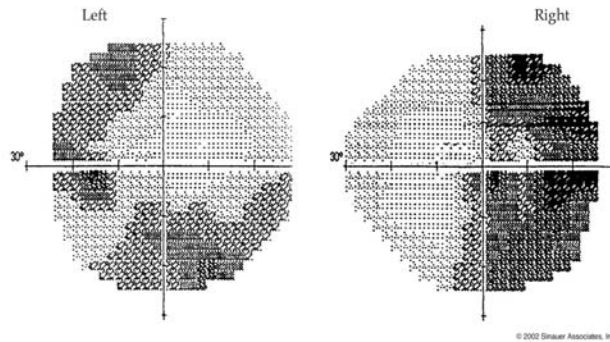
Lesions of the optic chiasm itself produce bitemporal (bilateral lateral) visual field defects (see C in figure above).

Understanding the basic anatomy of visual pathways is essential in clinical diagnosis and differentiation of eye disorders and lesions of the central nervous system.

VISUAL SYSTEM – CLINICAL CASE. A 50 year-old woman came to the ophthalmologist because of worsening vision that began to interfere with her ability to drive.

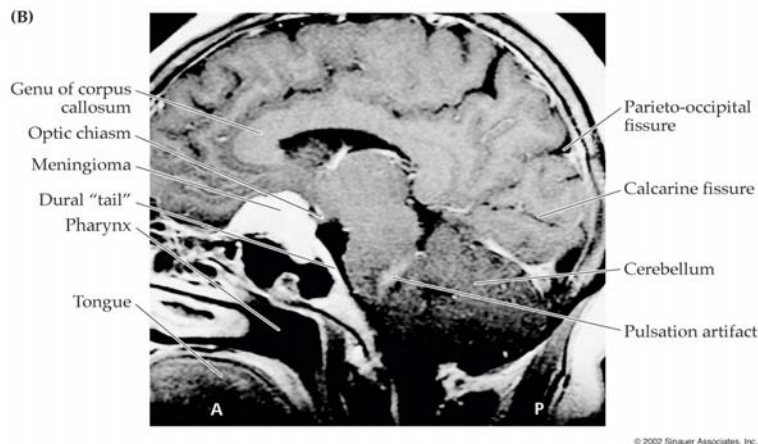
Past history was notable for long-standing menstrual irregularity and infertility. Examination was normal except for decreased vision primarily in the temporal portions of the visual fields bilaterally.

Visual fields were formally tested using automated computerized perimetry which showed bilateral visual field defects conforming most closely to a bitemporal hemianopia (see perimetry results below).



Bitemporal hemianopia is usually caused by lesions of the optic chiasm. Similar to most cases, the hemianopia is not perfectly symmetrical in this patient. The history of menstrual irregularity and infertility suggests an endocrine disorder, which suggests a lesion in the vicinity of both the pituitary gland and the optic chiasm. The most likely diagnosis is a pituitary adenoma although other tumors or masses in the sellar and suprasellar region could also produce these abnormalities including a meningioma, craniopharyngioma, or hypothalamic glioma.

Brain MRI revealed an enhancing mass lesion in the suprasellar region, compressing the optic chiasm (see MRI below).



Note the mass in the MRI above showed a “dural tail” suggesting it arose from the meninges.

The patient was referred to neurosurgery where she underwent resection of the mass. Later pathological analysis revealed the tumor was a meningioma.

Following surgery, the patient made an excellent recovery. There was a near complete return of vision in her lateral fields over the course of the next 6 to 7 months.

CRANIAL NERVES. This section summarizes some of the more important points taken from Chapter 12 of your text. Read Chapter 12 for a more detailed and complete discussion of the cranial nerves and brainstem anatomy. Cranial nerve anatomy and function is one of the more difficult topics in neuroanatomy and will initially require some memorization. Later, as you begin to use your knowledge of the nerves in clinical cases, you will become familiar with them.

The cranial nerves are listed by number, name, and main functions in the table below.

CN	NAME	MAIN FUNCTION(S)
CN I	Olfactory nerve	Olfaction
CN II	Optic nerve	Vision
CN III	Oculomotor nerve	Eye movements; pupil constriction
CN IV	Trochlear nerve	Eye movements
CN V	Trigeminal nerve	Facial sensation; muscles of mastication
CN VI	Abducens nerve	Eye movements
CN VII	Facial nerve	Muscles of facial expression; taste; lacrimation; salivation
CN VIII	Vestibulocochlear nerve	Hearing; equilibrium sense
CN IX	Glossopharyngeal nerve	Pharyngeal muscles; carotid body reflexes; salivation
CN X	Vagus nerve	Parasympathetics to most organs; laryngeal muscles (voice); pharyngeal muscles; aortic arch reflexes
CN XI	Spinal accessory nerve	Head turning (trapezius and sternomastoid muscles)
CN XII	Hypoglossal nerve	Tongue movement

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For individuals who like mnemonics, memorization of the names of the cranial nerves may be enhanced by pairing them with the following rhyme.

■ On	Olfactory	I
■ Old	Optic	II
■ Olympus's	Oculomotor	III
■ Towering	Trochlear	IV
■ Top	Trigeminal	V
■ A	Abducens	VI
■ Fine	Facial	VII
■ Vain	Vestibulocochlear	VIII
■ German	Glossopharyngeal	IX
■ Viewed	Vagus	X
■ A	Accessory	XI
■ Horse	Hypoglossal	XII

Cranial Nerve Brainstem Anatomy. From rostral to caudal (or in humans from the top down), the main parts of the brainstem are the midbrain (a.k.a., mesencephalon), pons, and medulla. The cranial nerves exit the brainstem roughly in numerical sequence from rostral to caudal, except for CN I (Olfactory) and CN II (Optic) which arise from the forebrain. Each cranial nerve exits the skull through a specific foramen.

Similar to the spinal cord gray matter, the cranial nerve nuclei for motor functions are located more ventrally in the brainstem, and those for sensory functions are located more dorsally. Some cranial nerves are **purely motor** (CN III, IV, VI, XI, XII), some are **purely sensory** (CN I, II, VIII), and some have **both motor and sensory** functions (CN V, VII, IX, X).

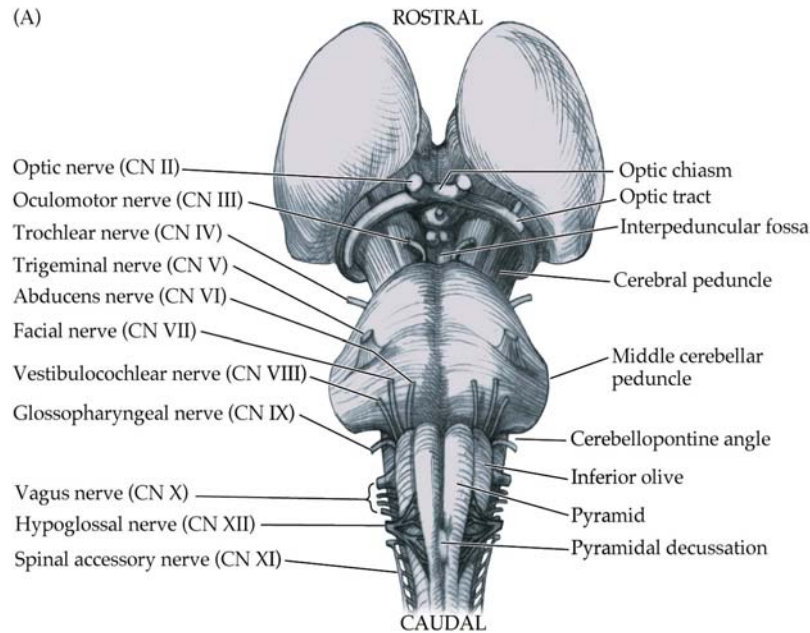
For those who are aided by mnemonics, memorization of the following rhyme may be used to assist in determining whether the specific cranial nerve's function is Sensory, Motor or Both.

■ Some	Olfactory	I
■ Say	Optic	II
■ Marilyn	Oculomotor	III
■ Monroe	Trochlear	IV
■ But	Trigeminal	V
■ My	Abducens	VI
■ Brother	Facial	VII
■ Says	Vestibulocochlear	VIII
■ Bridget	Glossopharyngeal	IX
■ Bardo	Vagus	X
■ Mmmh	Accessory	XI
■ Mmmh	Hypoglossal	XII

Olfactory Nerve (CN I) (Sensory) enters the skull via the cribriform plate to synapse in the olfactory bulb. Information regarding smell then travels to the olfactory cortex in the peri-rhinal area of the anterior temporal lobe via the olfactory tracts. Damage to CN I causes **anosmia**, or olfactory loss.

Optic Nerve (CN II) (Sensory) enters the cranial cavity via the optic canal through the skull. The optic nerve carries visual information from the retina, crosses at the chiasm where it becomes the optic tract, and then synapses in the lateral geniculate nucleus (LGN) of the thalamus. These fibers leave the thalamus and become the optic radiations which fan out and then synapse in primary visual cortex in the occipital lobe. The optic radiations are also called the **geniculocalcarine tract**.

Lesions within the optic tracts cause specific visual disturbances depending upon where the lesion occurs. These visual field defects were reviewed in the first section of this module (see above). The optic nerve, chiasm, and tract are shown below.



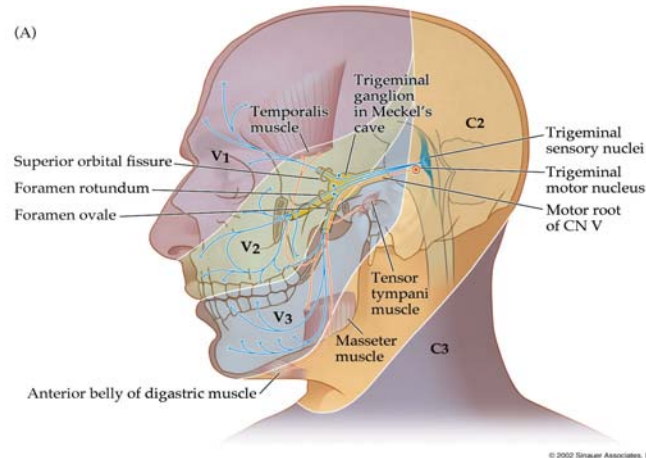
Oculomotor Nerve (CN III), Trochlear Nerve (CN IV), & Abducens Nerve (CN VI) (All three nerves Motor only). These three cranial nerves control eye movements by controlling the extraocular muscles. There are six extraocular muscles for each eye. The **medial** and **lateral rectus** muscles move the eye medially and laterally, respectively. The **superior** and **inferior rectus** and **superior** and **inferior oblique** muscles are involved in vertical and torsional eye movements.

The **oculomotor nerve (CN III)** supplies all the extraocular muscles except the lateral rectus and superior oblique. In addition it supplies the **levator palpebrae** muscle, which elevates the upper eyelid.

The **trochlear nerve (CN IV)** innervates the superior oblique muscle, and the **abducens nerve (CN VI)** innervates the lateral rectus. The extraocular muscles, their functions, and the cranial nerve that innervates them are listed in the table below.

MUSCLE	MAIN ACTIONS ON THE EYE	COMMENTS	INNERVATION
Lateral rectus	Abduction	Abduction = temporal (lateral) movement of eye	Abducens nerve (CN VI)
Medial rectus	Adduction	Adduction = nasal (medial) movement of eye	Oculomotor nerve (CN III)
Superior rectus	Elevation and intorsion	Elevation increases with abduction; intorsion increases with adduction	Oculomotor nerve (CN III)
Inferior rectus	Depression and extorsion	Depression increases with abduction; extorsion increases with adduction	Oculomotor nerve (CN III)
Inferior oblique	Elevation and extorsion	Elevation increases with adduction; extorsion increases with abduction	Oculomotor nerve (CN III)
Superior oblique	Depression and intorsion	Depression increases with adduction; intorsion increases with abduction	Trochlear nerve (CN IV)

Trigeminal nerve (CN V) (both sensory and motor) provides sensation for the face, mouth, and meninges surrounding brain (but not spinal cord) via three major branches: the **ophthalmic division** (V₁), **maxillary division** (V₂), and **mandibular division** (V₃) (see figure below).

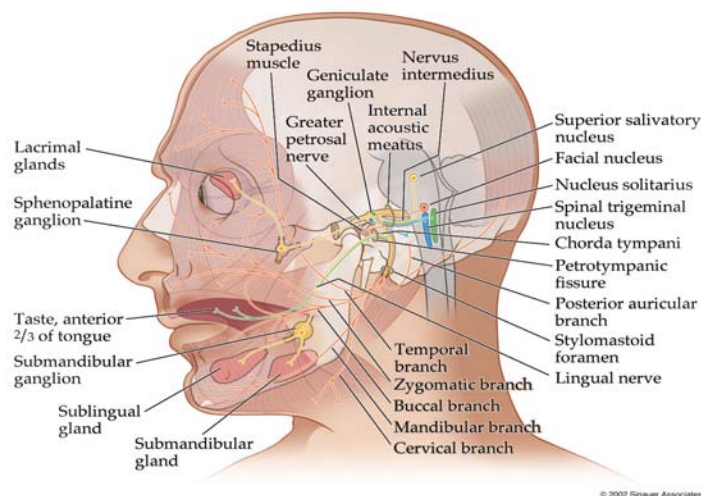


The trigeminal sensory nuclei include the **mesencephalic trigeminal nucleus** mediating proprioception, the **chief sensory nucleus** mediating discriminative touch, and the **spinal nucleus of V** (or spinal trigeminal nucleus) mediating pain and temperature.

Trigeminal sensory information travels to cortex via the **trigeminal lemniscus** and **trigeminothalamic tract** with a relay in **VPM** (ventral posterior medial) nucleus of the thalamus.

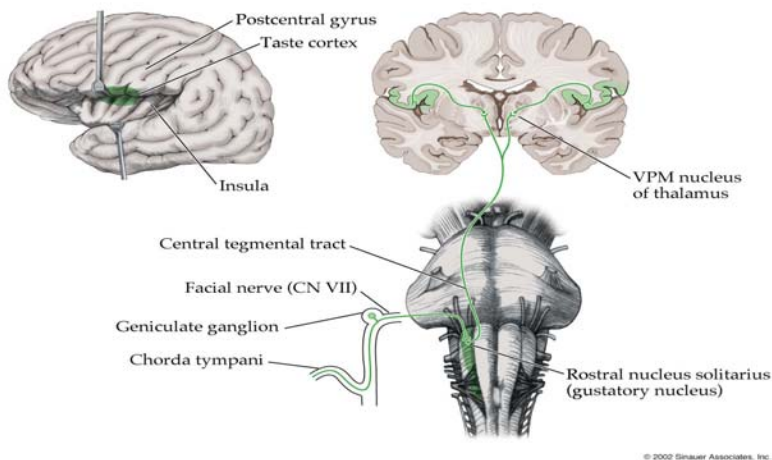
The trigeminal nerve also has a small **motor root** that travels with the V₃ division of the nerve, supplying the **muscles of mastication**.

Facial nerve (CN VII) (both sensory and motor) controls the muscles of facial expression (as opposed to the trigeminal nerve which mediates facial sensation) via fibers that arise from the **facial nucleus** in the pons (see figure below).

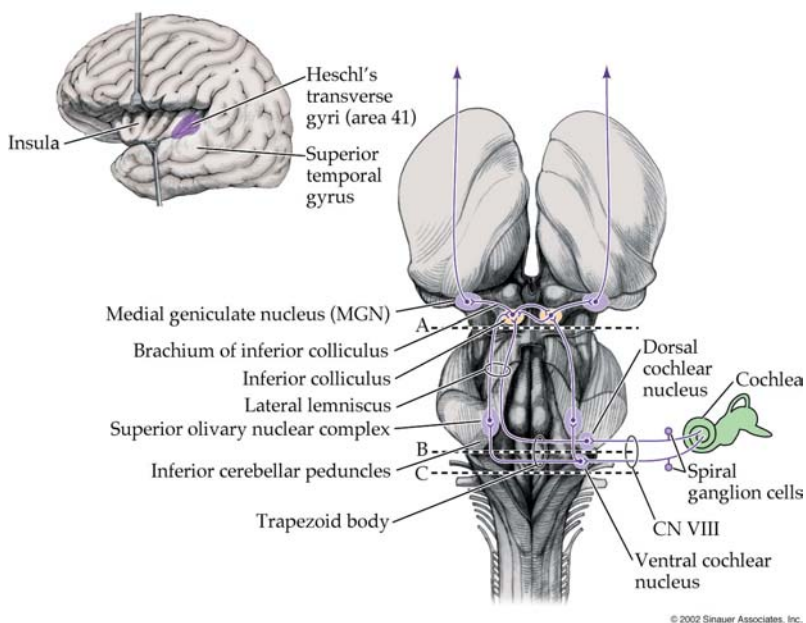


Upper motor neuron control of the facial nucleus is **bilateral for the upper parts of the face**, so in unilateral upper motor neuron lesions the contralateral side can compensate, resulting in sparing of the upper face muscles.

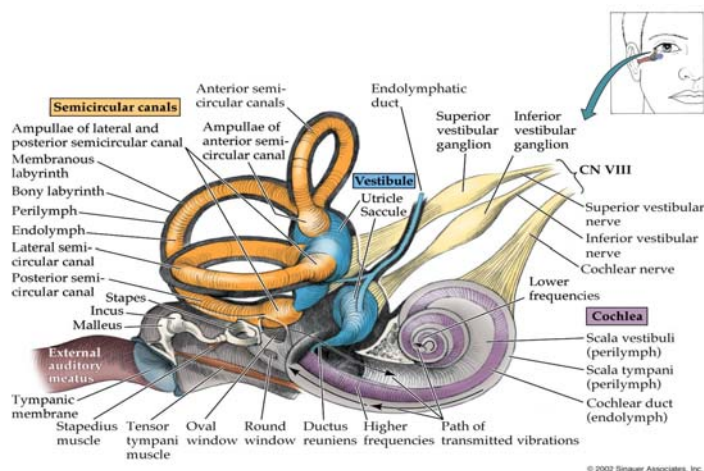
The facial nerve also has sensory fibers that provide **taste sensation** for the anterior 2/3rds of the tongue reaching the **solitarius nucleus** in the brainstem. Pathways for taste sensation are shown below.



Vestibulocochlear nerve (CN VIII) (sensory) carries auditory information from the cochlea to the **dorsal and ventral cochlear nuclei** (see figure below). The primary sensory cell bodies lie in the spiral ganglion. Central auditory pathways cross the midline multiple times, so unilateral lesions in the CNS do not cause clinically significant hearing loss.

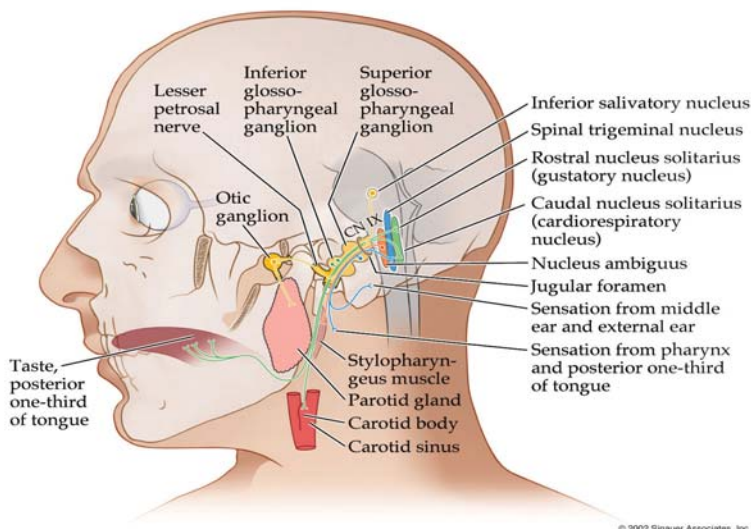


Information about head position and acceleration is carried by the vestibular portions of CN VIII from the **semicircular canals** and **otolith organs** (see figure below).

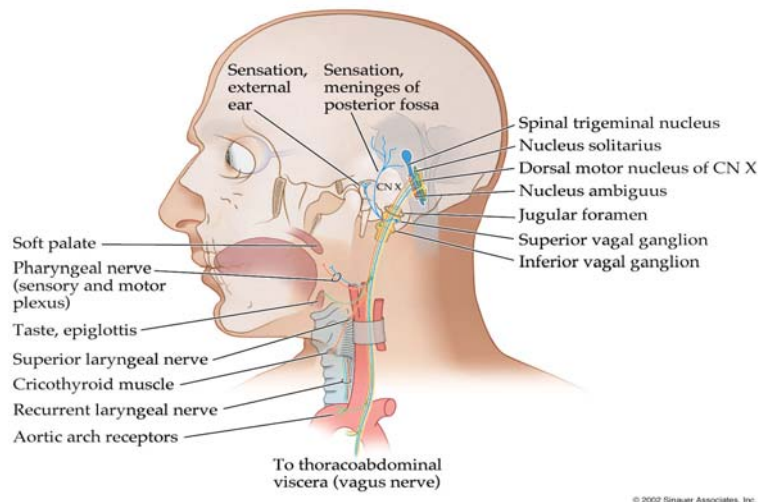


Primary cell bodies are located in the vestibular ganglia, and this information travels to the **vestibular nuclei** in the brainstem (in the pontomedullary junction) to influence unconscious posture and balance, eye movements, and conscious perception of movement through multiple pathways.

Glossopharyngeal nerve (CN IX) (both sensory and motor) was named for role in sensation for the posterior tongue and pharynx; however, it has additional functions as well. The sensory component provides general sensation and **taste** from the **posterior one-third of the tongue**, and part of the skin of the external ear. The motor component serves to control the **stylopharyngeus muscle**, which elevates the **pharynx during swallowing and speech**. Also, part of the motor component serves to innervate the **parotid gland**, an important salivary gland.



Vagus nerve (CN X) (both sensory and motor) also has multiple functions. It provides parasympathetic innervation for the viscera arising from the **dorsal motor nucleus of CN X** (see figure below).



In addition, motor fibers of the vagus arising from the **nucleus ambiguus** supply the pharynx (swallowing) and larynx (voice). Sensory fibers from the aortic arch (near heart) travel to the caudal nucleus solitarius. Sensory fibers for the pharynx, larynx, outer ear, and meninges of the posterior fossa travel to the trigeminal nuclei (CN V).

Spinal accessory nerve (CN XI) (motor) arises from the spinal accessory nucleus and innervates the sternomastoid and upper portions of the trapezius muscles. Because of the mechanical attachments of the sternomastoid muscle, lesions of CN XI cause weakness of head turning to the side opposite the lesion.

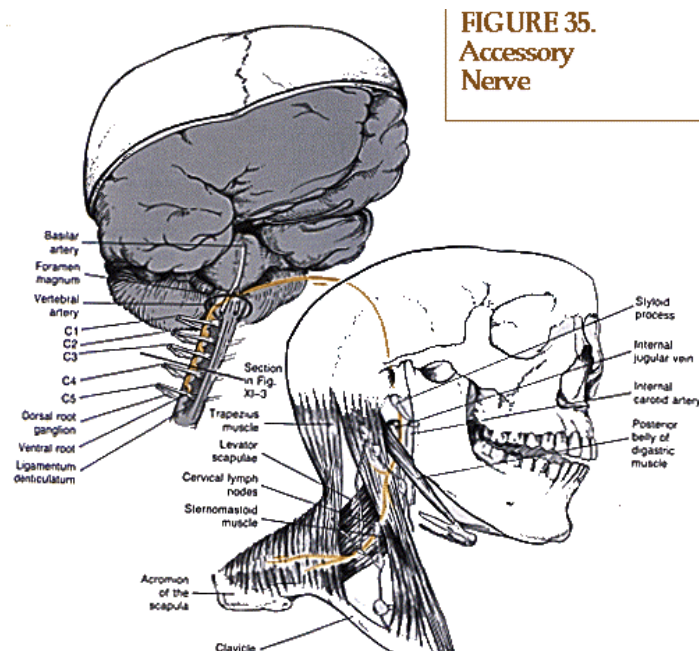
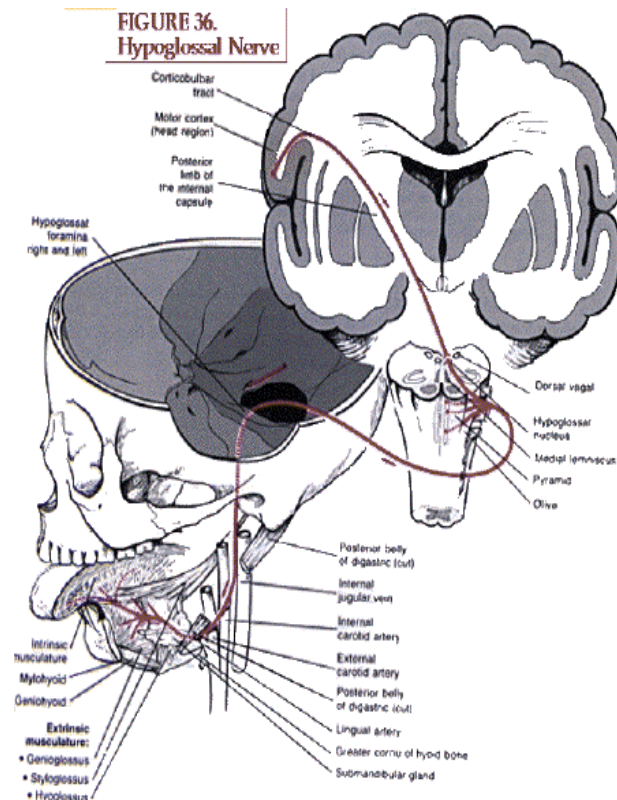


FIGURE 35.
Accessory
Nerve

Hypoglossal nerve (CN XII) (motor) arises from the hypoglossal nucleus and supplies the intrinsic tongue muscles. Hypoglossal nerve lesions cause the tongue to deviate toward the side of the lesion when the tongue is protruded.



SOME KEY CLINICAL CONCEPTS

Disorders of the **trigeminal nerve (CN V)** are relatively uncommon, except for ***trigeminal neuralgia*** (tic douloureux). In this condition, patients experience recurrent episodes of brief severe pain lasting from a few seconds to minutes. Attacks are most common in individuals after age 35. The cause is unknown in most cases. Painful episodes may be provoked by chewing, shaving, or touching a trigger point on the face. After possible detectable causes (e.g., tumor or other lesions in regions of trigeminal nerve) have been ruled-out treatment is with carbamazepine (Tegretol) or baclofen.

The most common **facial nerve (CN VII)** disorder is ***Bell's palsy***, in which all divisions of the facial nerve are impaired within a few hours or days and then gradually recover. This results in unilateral facial weakness of the lower motor neuron type (see below). Cause is unknown although viral or inflammatory mechanisms are likely. Treatment is controversial but steroids are used in some cases. **Upper motor neuron** lesions, above the level of CN VII nucleus in the pons, will result in mild contralateral lower facial weakness because innervation to the muscles of the lower face is contralateral while innervation to the muscles of the upper portion of the face is bilateral.

CLINICAL CASE

Background. A 27 year-old, right-handed male came to the emergency room because of worsening dysarthria (disorder of vocal articulation), dysphagia (disorder of swallowing), left-sided weakness, and episodes of uncontrollable laughter. The problems began 2 ½ years ago with episodes of left face and

mouth pain precipitated by chewing (suggesting a trigeminal nerve lesion). One year ago, he started having episodes of uncontrollable laughter not accompanied by any corresponding feeling state (pseudobulbar palsy). He then began to notice slurred speech and occasional choking on his food. He also had an unstable gait, bumping into things on his left side and difficulty buttoning his shirt with his left hand.

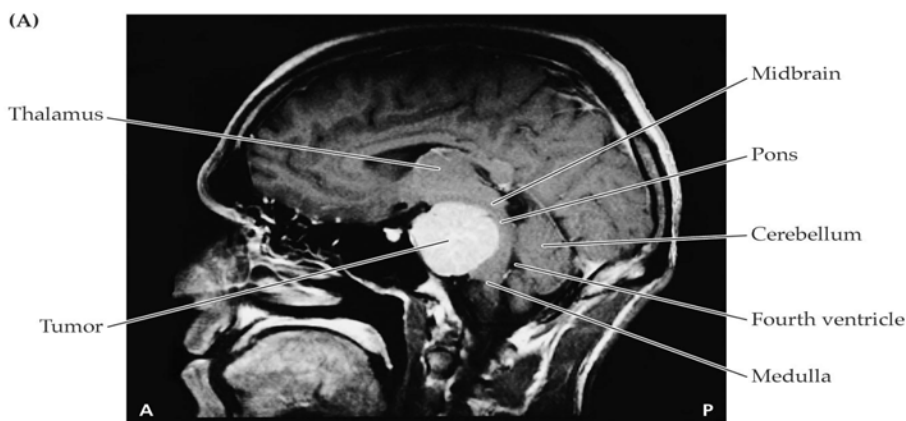
Neurologic examination. Cranial nerve exam revealed mildly decreased left nasolabial fold (left lower facial weakness = CN VII), absent gag reflex, mildly dysarthric speech, bouts of uncontrollable laughter about once per minute without accompanying emotion, and mild weakness of the head turning to the left (absent gag, dysarthria, dysphagia = CNs IX, X). Pseudobulbar palsy suggests dysfunction of the descending corticobulbar pathways in the subcortical white matter above the level of cranial nerve nuclei.

Motor exam revealed mild left pronator drift, slowed finger tapping with the left hand, and slightly increased tone in the left lower extremity. Reflexes were slightly increased in the left upper and lower extremity and there was a left Babinski's sign. Gait was slightly unsteady with a stiff left lower extremity.

Remainder of the physical and neurological examination was normal.

Clinical discussion. A lesion in the region of the pons and medulla could affect these multiple brainstem structures. Possibilities include multiple sclerosis, brainstem vascular malformation, a granulomatous disorder such as sarcoidosis, or a slow-growing tumor such as a brainstem glioma or a meningioma.

The patient underwent a brain MRI which showed a large mass lying outside of the brain adjacent to the dura and enhancing uniformly with gadolinium, consistent with a meningioma (see MRI below).



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The mass may be seen to cause severe compression and distortion of the pons and left middle cerebellar peduncle. The patient underwent a multi-stage resection, involving preoperative embolization by interventional radiology, and two operations involving teams of neurosurgeons and otolaryngologists. Follow-up exam one year postoperatively showed good recovery with rare episodes of inappropriate laughter and mild diplopia, but otherwise he was normal.