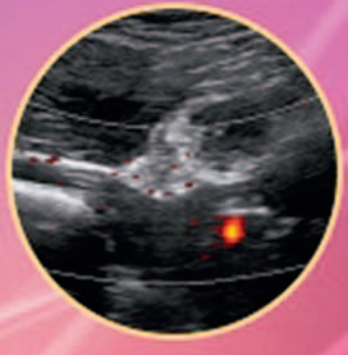
SECOND EDITION

PAIN REVIEW

STEVEN D. WALDMAN



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Dedication

Every long journey begins
with a first step.
—CONFUCIUS

To my children—David Mayo, Corey, Jennifer, and Reid—all of whom are sick of hearing me invoke the above quote . . . but who have nevertheless steadfastly followed its timeless wisdom in their daily lives!

Preface

Hypnopedia: *the art or process of learning while asleep by means of lessons recorded on disk or tapes*

As a child, I was always fascinated by the advertisements on the back of the comic books that my brother Howard and I avidly read. Among the many ads for a myriad of amazing items and services was one featuring a picture of a white-bearded Russian scientist standing next to a sleeping woman, touting that for just $19.95 you could purchase lessons that could teach you to *Learn While You Sleep*. Given that the Russians had just launched Sputnik and had supposedly detonated a hydrogen bomb, I was completely convinced that this was something I could not live without. I must admit that part of my desire to buy *Learn While You Sleep* was that I hated school and was always looking for an easier way to complete my lessons.

While I was never able to con my parents into spending the $19.95 for the *Learn While You Sleep* lessons, they did buy me a pair of the x-ray vision glasses for the then princely sum of $1.99. Needless to say, they didn't work nearly as well as I had hoped, and I began to wonder if the other things advertised on the back pages of my comics were as bogus. I didn't have to wonder too long as the full-size replica of a Sherman tank that my brother had ordered off the back of a Superman comic turned out to be little more than a big orange cardboard box. So much for *Learn While You Sleep*!

At this point, the reader might ask, "What does an old comic book ad for *Learn While You Sleep* have to do with a review text for pain management?" Well, as my brother Howard, with whom I have practiced pain management for the past 26 years, will tell you, I am still and always looking for an easier way to do things. When I started studying for my American Board of Anesthesiology recertification examination in pain management, there were no texts written to specifically help one review pain management in an organized and time-efficient manner, and I approached my publishers with the concept of creating such a review text. The result of our efforts is *Pain Review*.

In writing *Pain Review*, it was my goal to create a text that not only contained all of the material needed to review the specialty of pain management but also to organize that material into small, concise, easy-to-read chapters.

I believe that by breaking up the overwhelming amount of knowledge related to pain management into smaller and more manageable packets of information, the task of reviewing the entire specialty becomes much less daunting. I have also made liberal use of illustrations, as in many chapters a picture is the best way to convey a concept or technique.

Whether you are getting ready to take your certification or recertification examination in pain management or simply want to learn more about the specialty, I hope that *Pain Review* will serve your needs and help with your studies.

Steven D. Waldman, MD, JD

Contents

SECTION 1 ANATOMY

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SECTION 1 Anatomy

CHAPTER 1

Overview of the Cranial Nerves

Abnormal cranial nerve examination should alert the clinician to the possibility of not only central nervous system disease but also significant systemic illness. For this reason, a careful examination of the cranial nerves should be carried out in all patients suffering from unexplained pain. Abnormalities of the cranial nerves may affect one or more of the cranial nerves, and identification of these abnormalities may aid in the localization of a central nervous system lesion or may suggest a more diffuse process such as meningitis, pseudotumor cerebri, or the presence of systemic disease such as diabetes, sarcoidosis, botulism, myasthenia gravis, Guillain-Barré, vasculitis, and others. Common causes of specific cranial nerve abnormalities are listed in respective chapters that discuss each of the 12 cranial nerves. The 12 cranial nerves are listed here in Table 1-1. The classic acrostic, *On Old Olympia's Towering Top A Finn And German Vault And Hop*, has been augmented by the use of a novel clockface-based paradigm to help learners memorize the names and functions of the cranial nerves (Fig. 1-1). This clockface paradigm will be presented in each chapter describing the individual cranial nerves.

To best understand cranial nerve abnormalities, it is useful to think about them in the context of their anatomy. Although the anatomy of the specific cranial nerves will be discussed in the individual chapters covering each cranial nerve, the following schema may be applied to all of the 12 cranial nerves. The efferent fibers of the cranial nerves arise deep within the brain in localized anatomic areas called the *nuclei of origin*. These nerves exit the brain and brainstem at points known as the superficial origins (Fig. 1-2). The afferent fibers of the cranial nerves arise outside the brain and may take the form of either specialized fibers that are grouped together in a sense organ (e.g., the eye or nose) or grouped together within the trunk of the nerve to form ganglia. The fibers enter the brain to coalesce to form the nuclei of termination. Lesions that affect the peripheral portion or trunks of the cranial nerves are called *infranuclear lesions*. Lesions that affect the nuclei of the cranial nerves are called *nuclear lesions*. Lesions that affect the central connections of the cranial nerves are called *supranuclear lesions*. When evaluating a patient presenting with a cranial nerve abnormality, it is also helpful for the clinician to remember that the first two cranial nerves, the olfactory and the optic, are intimately associated with the quite specialized anatomic structures of the nose and eye and are subject to myriad diseases that may present as a cranial nerve lesion. The remaining 10 cranial nerves are much more analogous in structure and function to the spinal nerves and thus more subject to entrapment and/or compression from extrinsic processes such as a tumor, an aneurysm, or an aberrant blood vessel rather than primary disease processes.

Table 1-1 The Cranial Nerves

- 1st—Olfactory
- 2nd—Optic
- 3rd—Oculomotor
- 4th—Trochlear
- 5th—Trigeminal
- 6th—Abducens
- 7th—Facial
- 8th—Acoustic/auditory/vestibulocochlear
- 9th—Glossopharyngeal
- 10th—Vagus
- 11th—Spinal accessory
- 12th—Hypoglossal

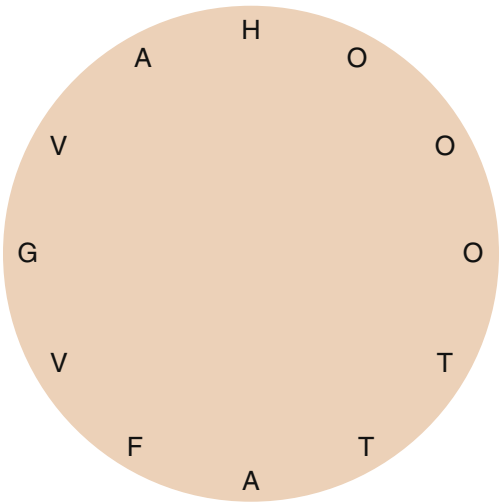

Fig. 1-1 The clockface paradigm for the twelve cranial nerves. (Modified from Weiss, KL, Eldevik, OP Bieliauskas, L, et al: Cranial nerve clock: Part I. A declarative memory paradigm. *Acad Radiol* 2001; 8[12]: 1215–1222.)

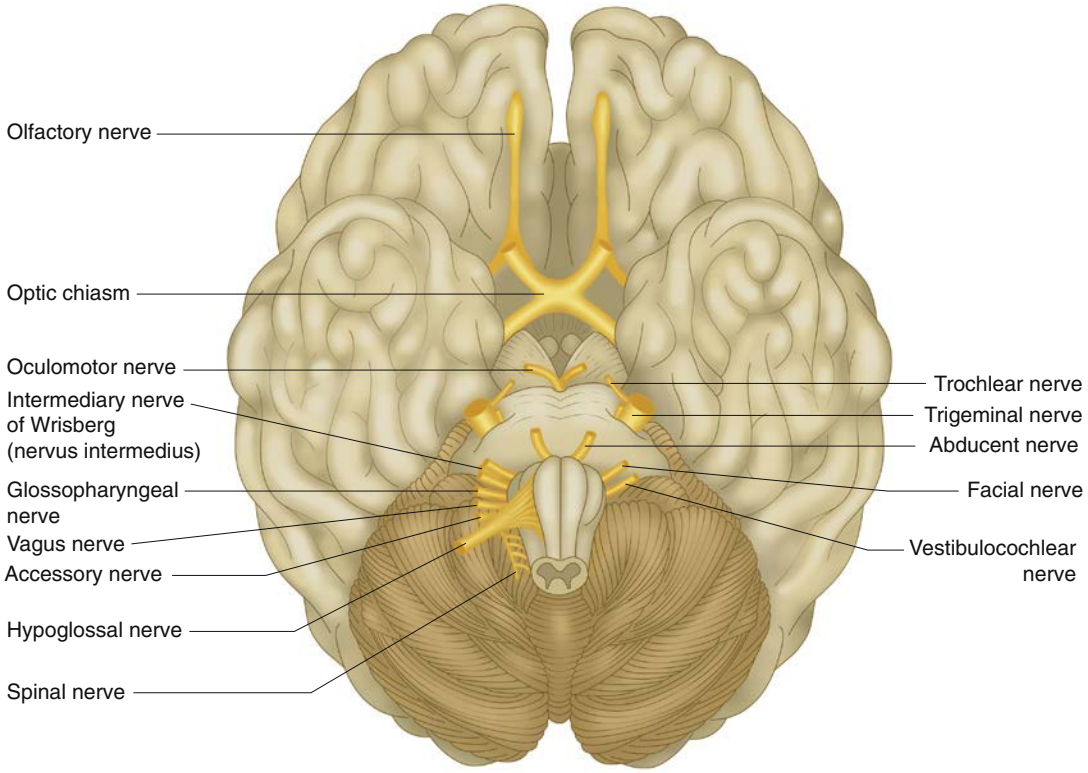

Fig. 1-2 The superficial origin of the cranial nerves. (From Barral JP, Croibier A: Anatomical organization of cranial nerves. In: Barral JP, Croibier A (eds): Manual Therapy for the Cranial Nerves, Edinburgh, Churchill Livingstone, 2009, pp. 15-18.)

Suggested Readings

Fisch A: Clinical examination of the cranial nerves. In Tubbs RS, Rizk E, Shoja MM, et al (eds): Nerves and Nerve Injuries, San Diego, Academic Press, 2015, pp 195–225.

Weiss K, Eldevik OP, Bieliauskas L, et al: Cranial nerve clock: Part I. A declarative memory paradigm, Academic Radiology 8:1215–1222, 2001.

CHAPTER 2

The Olfactory Nerve—Cranial Nerve I

The first cranial nerve is known as the olfactory nerve and is denoted by the Roman numeral I. It is composed of special afferent nerve fibers that are responsible for our sense of smell (Fig. 2-1). The olfactory nerve and associated structures include the chemoreceptors known as the *olfactory receptor cells*, which are located in the epithelium covering the roof, septum, and superior conchae of the nasal cavity (Fig. 2-2). Inhaled substances dissolve in the moist atmosphere of the nasal cavity and stimulate its chemoreceptors. If a firing threshold is reached, these chemoreceptors initiate action potentials that fire in proportion to the intensity of the stimulus. These stimuli are transmitted via fibers of the olfactory nerve that traverse the cribriform plate to impinge on the olfactory bulb, which contains the cell bodies of the secondary sensory neurons that make up the olfactory tract.

The olfactory tract projects into the cerebral cortex to areas known as the *lateral, intermediate,* and *medial olfactory areas*. The lateral olfactory area is most important to humans' sense of smell, with the intermediate area less so. The medial olfactory area, via its interconnections with the limbic system, serves to help mediate humans' emotional response to smell. Collectively, the olfactory receptor cells, epithelium, and bulb tracts and areas are known as the *rhinencephalon* (Fig. 2-3).

All three olfactory areas interact with a number of autonomic centers via a network of interconnected fibers. The medial forebrain bundle carries information from all three olfactory areas to the hypothalamus, while the stria terminalis carries olfactory information from the amygdala to the preoptic region of the cerebral cortex. The stria medullaris carries olfactory information to the habenular nucleus, which along with the hypothalamus interfaces with a number of cranial nerves to mediate humans' visceral responses associated with smell. Examples of such visceral responses include the dorsal motor nuclei of the vagus nerve (10th cranial nerve), which can modulate

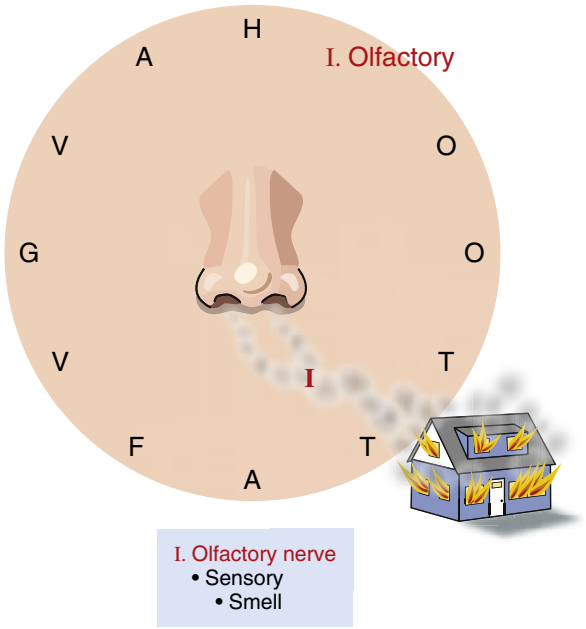

Fig. 2-1 Olfactory nerve I.

nausea and vomiting and changes in gastrointestinal motility, as well as the superior and inferior salivatory nuclei, which modulate salivation.

Abnormalities of the olfactory nerve may result in a condition known as *anosmia*, or the inability to smell. A simple approach to the testing of smell is outlined in Table 2-1. Anosmia can be permanent or temporary like that occurring with bad allergies or colds. It may be congenital or acquired; the most common causes of anosmia are listed in Table 2-2. Although anosmia might seem at first glance to be of little consequence, the lack of smell is associated with significant morbidity and mortality because of impairment of the extremely important warning function that olfaction plays in activities of daily living. The ingestion of spoiled foods, the inability to smell toxic gases such as the mercaptan in natural gas, or the inability to smell the smoke of a house fire are just a few examples of how the inability to smell can harm.

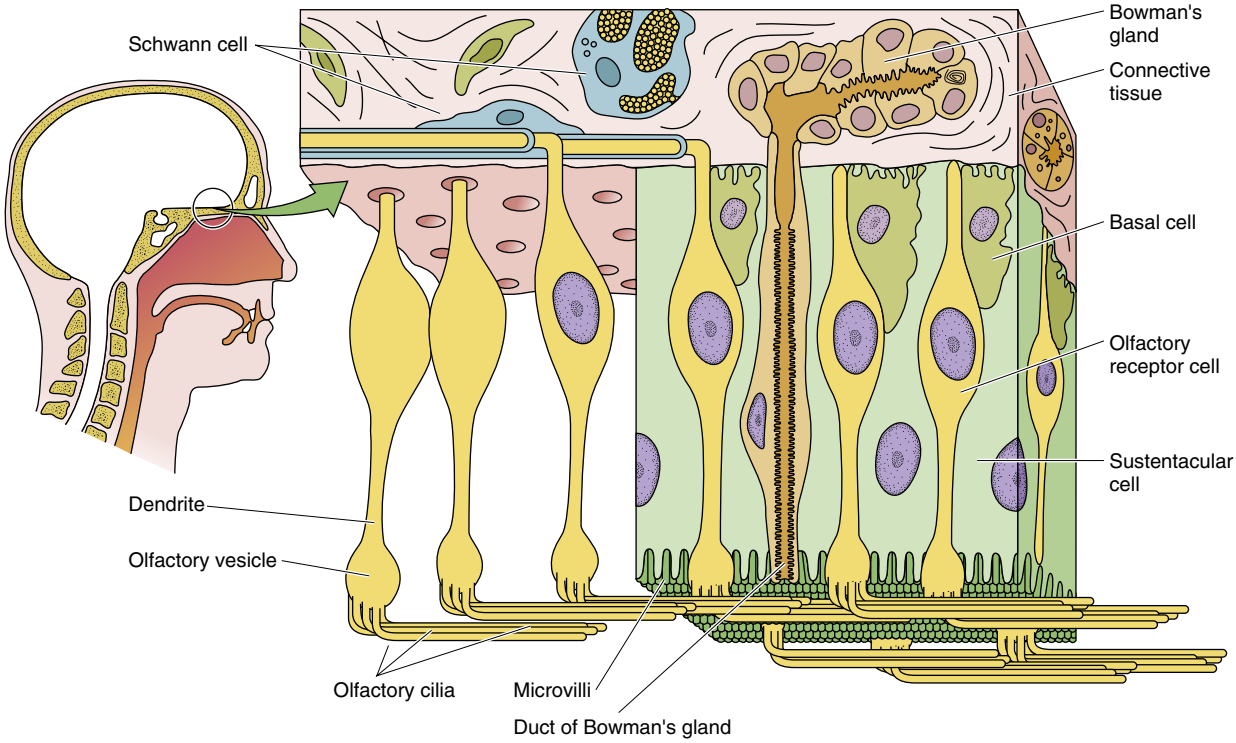

Fig. 2-2 The anatomy of the olfactory epithelium. (From Gartner LP, Hiatt, JL: Color Textbook of Histology. Philadelphia, Saunders, 2007.)

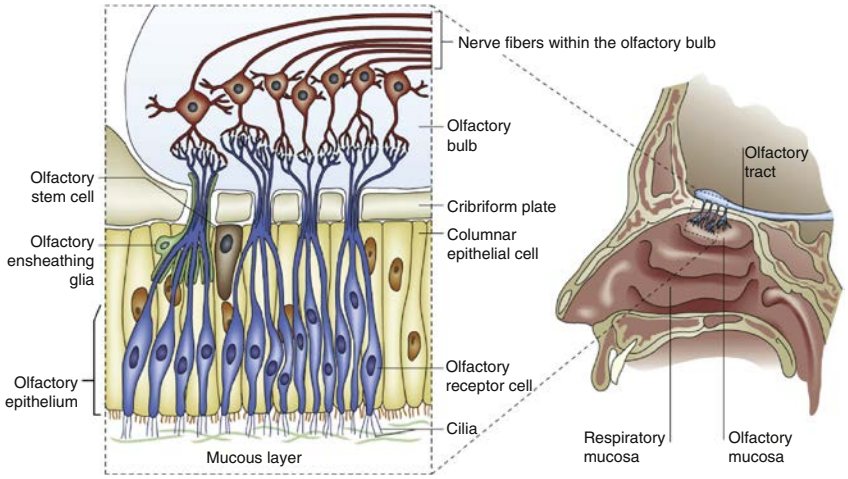

Fig. 2-3 The olfactory bulb, tract, and areas. (Reprinted with permission from Thuret S, Moon LDF, Gage FH: Therapeutic interventions after spinal cord injury. Nat Rev Neurosci 2006; 7:628-643.)

Table 2-1 How to Test Function of the Olfactory Nerve

1. Ascertain that the nasal passages are open.
2. Have the patient close his or her eyes.
3. Occlude one nostril.
4. Place a vial of nonirritating test substance (e.g., fresh ground coffee or oil of lemon) near the open nostril
   Note: Avoid irritating substances such as oil of peppermint that may stimulate the peripheral endings of the trigeminal nerve of the nasal mucosa.
5. Have the patient inhale forcibly.
6. Ascertain whether the patient can perceive an odor.
   Note: The ability to identify what the odor is requires higher cerebral function, and it is the perception of odor or lack thereof rather than its identification that is important.
7. Repeat the above process with the ipsilateral nostril.

Table 2-2 Causes of Anosmia

- Congenital
- Upper respiratory tract infections
- Nasal sprays containing zinc
- Facial and nasal trauma
- Prolonged exposure to tobacco smoke
- Enlarged adenoids
- Nasal polyps
- Paranasal sinusitis
- Head trauma damaging the cribriform plate or olfactory areas of the cerebral cortex
- Cerebrovascular accident
- Tumors involving the
  - Paranasal sinuses
  - Pituitary gland
  - Cranial vault, including gliomas, meningiomas, and neuroblastomas

Suggested Readings

Fisch A: Clinical examination of the cranial nerves. In: Tubbs RS (ed): Nerves and Nerve Injuries, Elsevier Science, London, Volume 2, 2015, pp 195–225.

Shipley MT, Puche AC: Olfactory nerve (cranial nerve I). In: Daroff RB, Aminoff MJ (eds): Encyclopedia of the Neurological Sciences, San Diego, Academic Press, 2014, pp 638–642.

CHAPTER 3

The Optic Nerve—Cranial Nerve II

Functional Anatomy of the Optic Nerve

The second cranial nerve is known as the optic nerve and is denoted by the Roman numeral II. Its special afferent sensory fibers carry visual information from the retina to the cerebral cortex for processing and interpretation (Fig. 3-1). In order to best understand abnormalities of vision, it is helpful for the clinician to think about these abnormalities in the context of the functional anatomy of the optic nerve. Light enters the eye in the form of photons, which pass through the cornea, aqueous humor, pupil, lens, and vitreous humor to reach the retina (Fig. 3-1). Special photoreceptor cells known as the *rods and cones*, which are located in the deep layers of the retina, begin the conversion of the photons into electrical signals. As these photoreceptor cells are stimulated, they become hyperpolarized and produce either depolarization (stimulation) or hyperpolarization (inhibition) of the bipolar cells, which are the primary sensory neurons of the visual pathway.

The bipolar cells synapse with and either stimulate or inhibit the ganglion cells that are the secondary sensory neurons of the visual pathway. The axons of the ganglion cells converge at the optic disk near the center of the retina. These axons then exit the posterior aspect of the eye as the optic nerve (cranial nerve II) (Fig. 3-2). Exiting the orbit via the optic canal, the optic nerve enters the middle cranial fossa to join the ipsilateral optic nerve to form the optic chiasm. Fibers from each optic nerve cross the midline to exit the chiasm together as the opposite optic tract (Fig. 3-3).

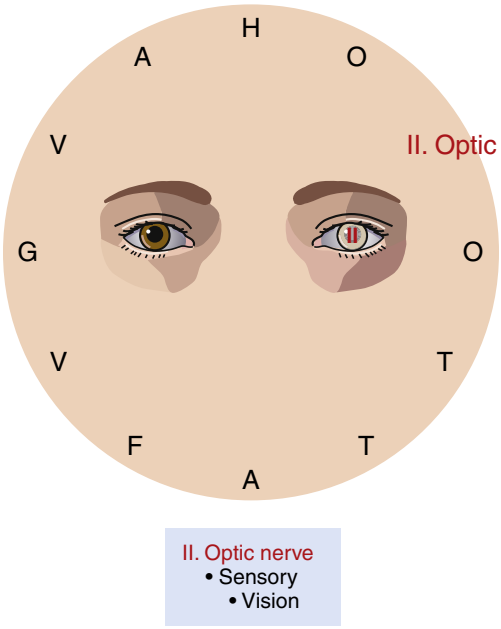

Fig. 3-1 The second cranial nerve is known as the optic nerve and is denoted by the Roman numeral II.

The optic tracts containing fibers from both optic nerves travel posteriorly, passing around the cerebral peduncles of the midbrain. Most of the fibers of the optic tracts synapse with the tertiary sensory neurons of the lateral geniculate nucleus within their contralateral thalamus (see Fig. 3-3). A few optic tract fibers travel to the pretectal region of the midbrain and provide necessary information for the pupillary light reflex. Via the optic radiations, the tertiary sensory neurons of the lateral geniculate nuclei project to the primary visual cortex, which is located in the occipital lobe (Fig. 3-4).

The Visual Field Pathways

The entire area that is seen by the eye when it is focused on a central point is called the *visual field* of that eye. It must be remembered that the photons entering the cornea converge and pass through the narrow pupil, with the entire visual field being projected on the retina in a reversed and upside down orientation (see Fig. 3-3). This means that the upper half of the retina is stimulated with photons from the lower half of the visual field and the lower half of the retina is stimulated with photons from the upper half of the visual field. Furthermore, the right half of the retina receives stimuli from the left visual field, and the left half of the retina receives stimuli from the right half of the visual field.

Given the consistent way that the ganglion cells from the retina group together to form the optic nerve and carry information to the primary visual cortex, the clinician may find it useful to divide the visual field of each eye into four quadrants: (1) the nasal hemiretina, which lies medial to the fovea; (2) the temporal hemiretina, which lies lateral to the fovea; (3) the superior hemiretina, which lies superior to the fovea; and (4) the inferior hemiretina, which lies inferior to the fovea (see Fig. 3-3). The axons of the ganglion cells of the nasal hemiretina decussate at the optic chiasm and travel on to project onto the contralateral lateral geniculate nucleus and midbrain. The axons of the ganglion cells of the temporal hemiretina remain ipsilateral through their course and project onto the ipsilateral lateral geniculate nucleus and midbrain (see Fig. 3-4). The axons of the ganglion cells of the

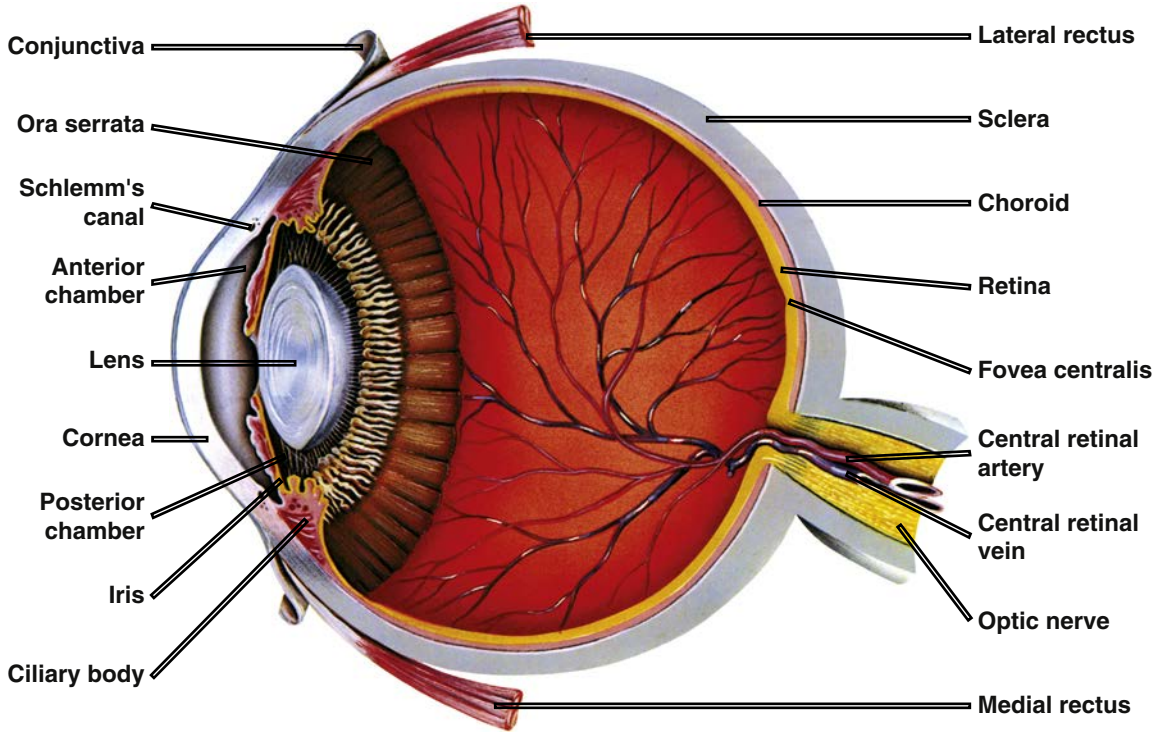

Fig. 3-2 The path of light though the eye. (From Aaron M, Solley WA, Broocker G: Chapter 1 - General Eye Examination. In: Palay DA, Krachmer JH [eds]: Primary Care Ophthalmology, ed 2. Philadelphia, Mosby, 2005, pp 1-23.)

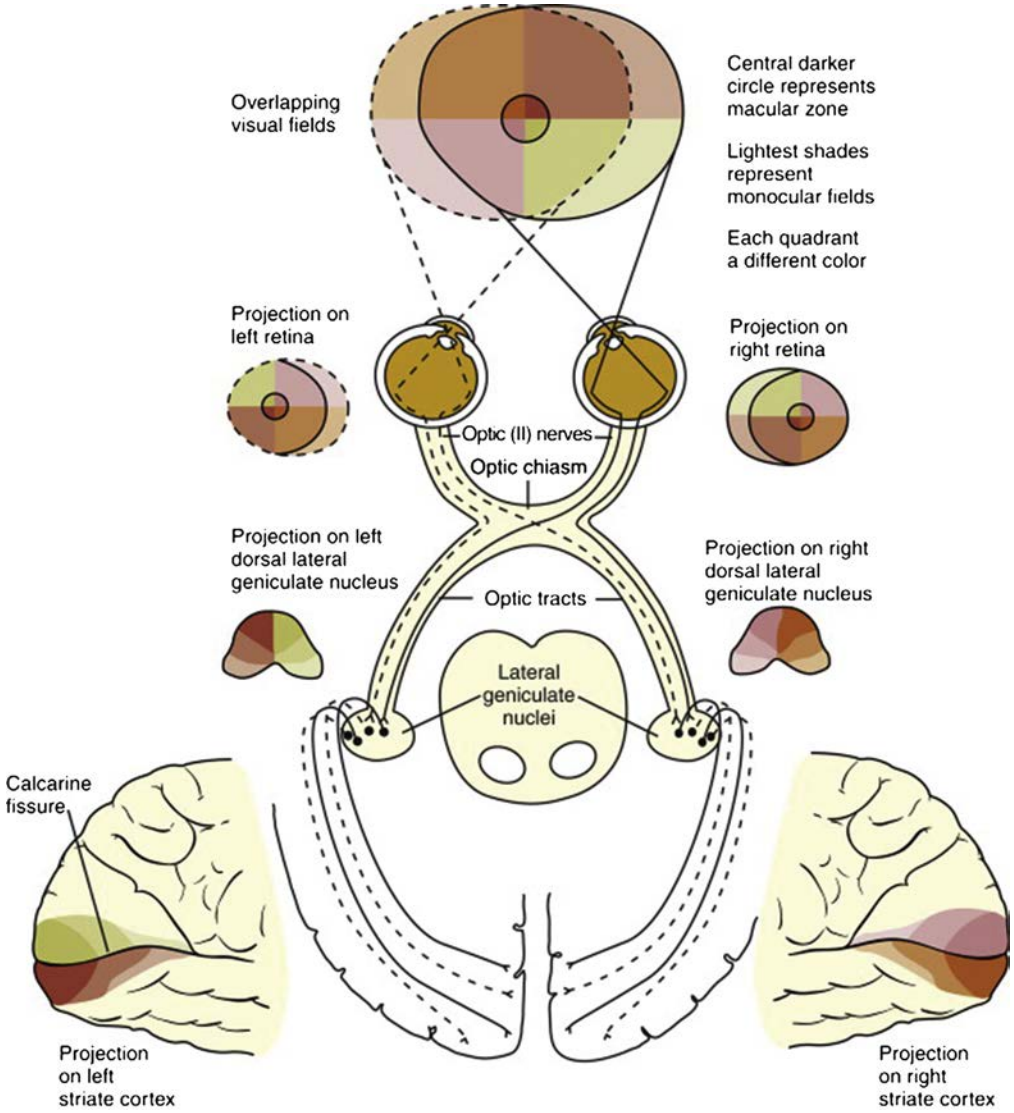

Fig. 3-3 The visual pathway. (From Remington LA: Chapter 13 - Visual Pathway. In: Remington LA (ed): Clinical Anatomy and Physiology of the Visual System, ed 3. St. Louis, Butterworth-Heinemann, 2012, pp 233-252.)

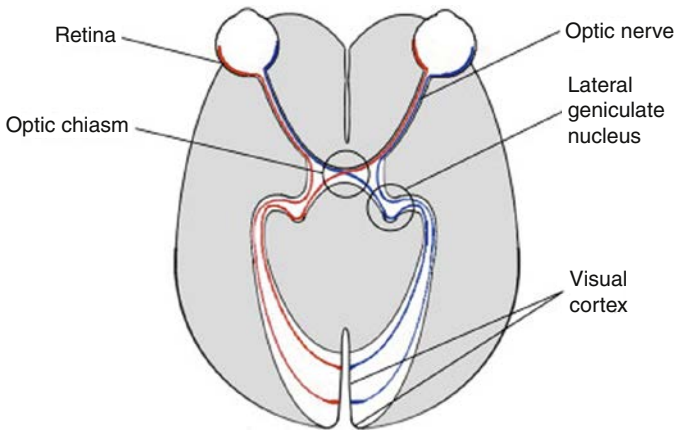

Fig. 3-4 Visual field pathways. The visual pathway begins with the retinas in both eyes and depart from the eyes through the optic nerves. All information from the left of the visual field travels through the optic chiasm and continues to the right lateral geniculate nucleus (LGN). The converse occurs for information from the right side of the visual field. This necessitates that information from both eyes crosses at the optic chiasm. From the LGNs, visual information proceeds to the visual cortex of the respective cerebral hemisphere. (From Escobar A: Qualia as the fundamental nature of visual awareness. J Theor Biol 2011; 279[1]:172-176.)

superior hemiretina carrying images from the inferior visual field project via the parietal lobe portion of the optic radiations to the portion of the primary visual cortex located above the calcarine fissure (see Figs. 3-4 and 3-5). The axons of the ganglion cells of the inferior hemiretina carrying images from the superior visual field project via the temporal lobe portion of the optic radiations to the portion of the primary visual cortex located below the calcarine fissure (see Figs. 3-4 and 3-5). Axons of the ganglion cells from the center of the retina or fovea project onto the tip of the occipital pole. Armed with the above knowledge of the functional anatomy of the visual pathway and the optic nerve, based on the patient's symptoms and visual abnormalities, the clinician can reliably predict what portion of the visual pathway is affected.

Clinical Evaluation of the Optic Nerve and Visual Pathway

Evaluation of optic nerve function also by necessity includes evaluation of retinal function. The clinician examines each of the patient's eyes individually and begins the examination with an assessment of visual acuity. Distant vision is tested using a standard Snellen test chart, and near vision is tested by having the patient read the smallest type possible from a Jaeger reading test card placed 14 inches from the eye being tested. Color blindness, which

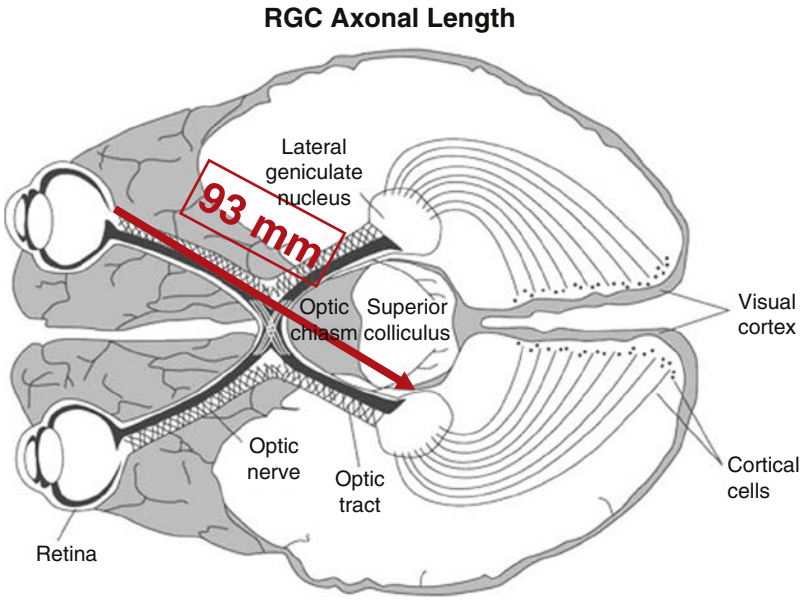

Fig. 3-5 The optic tract and radiations to and from the visual cortex. (From Yücel Y, Gupta N: Glaucoma of the brain: a disease model for the study of transsynaptic neural degeneration. In: Nucci C, Cerulli L, Osborne NN, Bagetta G [eds]: Progress in Brain Research, San Diego, Elsevier, 2008, Volume 173, pp 465-478.)

Fig. 3-6 Ishihara color blindness test showing (left to right) plate nos. 4 and 6 (1st row) and plate nos. 10 and 16 (2nd row) (From Kumar A, Choudhury R: Chapter 5 - Unusual visual phenomena and colour blindness. In: Kumar A, Choudhury R [eds]: Principles of Colour and Appearance Measurement, Woodhead Publishing, Cambridge 2014, pp 185-208.)

Fig. 3-7 Confrontation method of visual field testing. (From Aaron M, Solley WA, Broocker G: Chapter 1 - General Eye Examination. In: Palay DA, Krachmer JH [eds]: Primary Care Ophthalmology, ed 2. Philadelphia, Mosby, 2005, pp 1-23.)

occurs in approximately 3% to 4% of males and 0.3% of women, can be tested by having the patient read isochromatic plates such as the Ishihara plates, with an inability to read the embedded numbers in the presence of normal visual acuity highly suggestive of color blindness (Fig. 3-6).

The next step in evaluation of the optic nerve and associated structures of the visual pathway is examination of the visual fields. Although there is intrapatient variation in visual fields due to the patient's facial characteristics and shape of the globe and orbit, the following general observations can be made. In health, a person is able to see laterally approximately 90 to 100 degrees and medially approximately 60 degrees. The patient can see upward approximately 50 to 60 degrees and downward 60 to 70 degrees with the eye fixed in the midline. The easiest test for evaluation for significant visual field loss is the confrontation test. The confrontation test is performed with the clinician using his or her own visual fields as a control. To perform the confrontation test for visual fields, the examiner and patient both cover opposite eyes, and with the examiner standing approximately 3 feet in front of the patient, the examiner slowly brings his or her finger into each quadrant of the visual field. The patient is instructed to inform the examiner the second the examiner's finger is seen, with the examiner comparing his or her own response with that of the patient's (Fig. 3-7). While beyond the scope of this review, the clinician should be aware that specific patterns of visual field loss are associated with specific clinical abnormalities of the optic nerve and visual pathways, such as homonymous hemianopia, which is often associated with occipital lobe neoplasms or stroke; bitemporal hemianopia, which is often associated with pituitary adenomas; and so on (Fig. 3-8).

Fundoscopic examination of the retina and the optic disk is an essential part of the evaluation of the optic nerve. The optic disk, which is located just medial and slightly above the center of the fundus, should appear oval in shape and pale pink in color. The margin of the optic disk should be clearly defined with the margins slightly elevated (Fig. 3-9). A pale or poorly

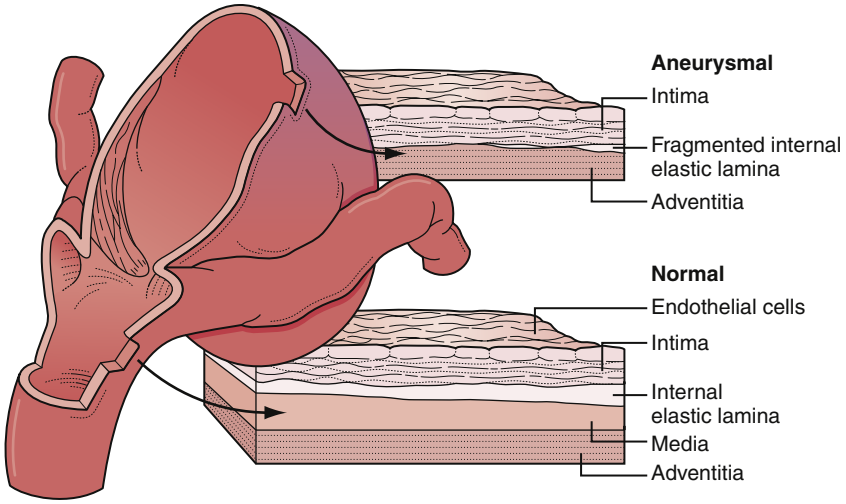

Fig. 3-8 Specific patterns of visual field loss are associated with specific clinical abnormalities of the optic nerve and visual pathways, such as homonymous hemianopia, which is often associated with occipital lobe neoplasms or stroke; bitemporal hemianopia, which is often associated with pituitary adenomas and aneurysms. (From Grant GA, Ellenbogen RG: Chapter 2 - Clinical Evaluation of the Nervous System. In: Principles of Neurological Surgery, ed 3. Philadelphia, Saunders, 2012, pp 37-52.)

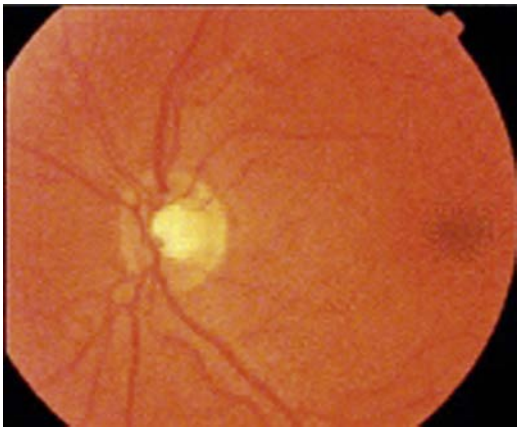

Fig. 3-9 The normal optic disk. (From Fingeret M, Medeiros FA, Susanna R Jr, Weinreb RN: Five rules to evaluate the optic disk and retinal nerve fiber layer for glaucoma. Optometry 2005; 76[11]:661-668.)

defined optic disk is highly suggestive of pathology of the optic nerve, as is a swollen head of the optic nerve, which is called *papilledema*. Papilledema is pathognomonic for increased intracranial pressure (Fig. 3-10). It should be noted that optic neuritis associated with multiple sclerosis may resemble papilledema and confuse the diagnosis.

Abnormalities of the retinal vessels seen on fundoscopic examination may also provide the clinician with useful diagnostic information. Occlusion of the central retinal artery can result in sudden visual loss and is associated with a pale, edematous optic disk and thin arteries, which can only be followed outward a short distance from the disk. Atherosclerosis can be identified by noting a silver wire appearance of the retinal arteries. Systemic hypertension can result in arterial narrowing and cotton wool patches that appear stuck onto the retina. Common abnormalities of the optic nerve and visual pathways are listed in Table 3-1.

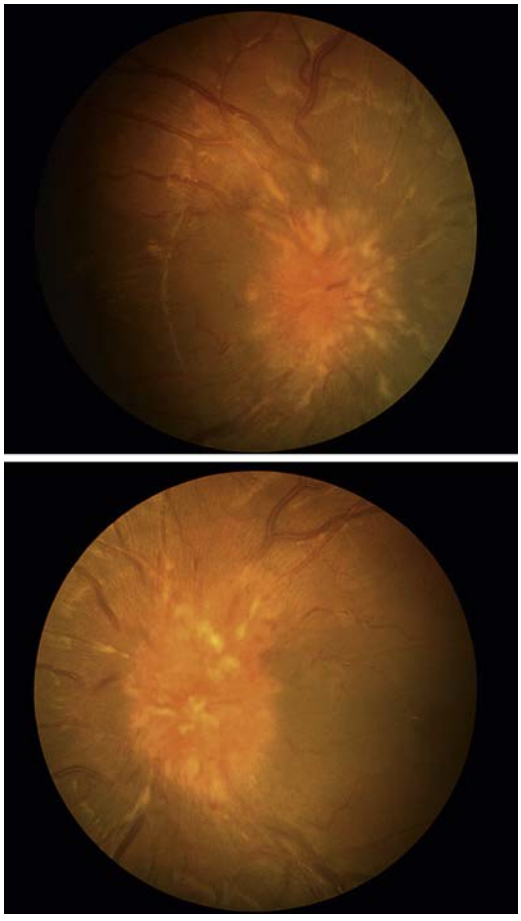

Fig. 3-10 Florid papilledema. (From Rogers DL: A review of pediatric idiopathic intracranial hypertension. Pediatr Clin North Am 2014; 61[3]:579-590.)

Table 3-1 Common Diseases That Result in Visual Impairment

Systemic Diseases
• Diabetes mellitus • Hypertension • Vitamin A deficiency • Vitamin B_{12} deficiency • Lead poisoning • Migraine with aura • Graves' disease • Sarcoidosis • Collagen vascular diseases • Atherosclerosis and stroke • Sickle cell disease • Multiple sclerosis • Refsum's disease • Tay-Sachs disease
Infection
• HIV-associated infections including cytomegalovirus • Trachoma • Bacterial infections including gonococcal infections • Parasitic infections including onchocerciasis • Spirochete infections including syphilis • Viral infections • Leprosy
Eye Diseases
• Macular degeneration • Glaucoma • Cataracts • Retinitis pigmentosa • Rod and cone dystrophy • Best disease, also known as vitelliform macular dystrophy
Trauma
• Burns • Projectile injuries • Side effects of medications • Bungee cord and rubber band injuries • Fish hook injuries • Fireworks injuries • Sports injuries • Complications of eye surgery
Neoplasms
• Optic gliomas • Melanoma • Pituitary adenoma

Suggested Readings

Fingeret M, Medeiros FA, Susanna Jr R, Weinreb RN: Five rules to evaluate the optic disk and retinal nerve fiber layer for glaucoma, Optometry - Journal of the American Optometric Association 76(11):661–668, 2005 Nov.

Fisch A: Clinical examination of the cranial nerves. In: Tubbs RS, et al (eds): Nerves and Nerve Injuries, San Diego, Academic Press, 2015,pp 195–225.

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Waldman SD: Migraine headache. In: Waldman SD (ed): Atlas of Common Pain Syndromes, ed 3. Philadelphia, Saunders, 2015.

CHAPTER 4

The Oculomotor Nerve—Cranial Nerve III

The oculomotor nerve is the third cranial nerve and is denoted by the Roman numeral III. It is made up of both general somatic efferent and general visceral efferent fibers, which serve two distinct functions. The general somatic efferent fibers of the oculomotor nerve provide motor innervation to four of the six extraocular muscles: (1) the ipsilateral inferior rectus muscle, (2) the ipsilateral inferior oblique muscle, (3) the ipsilateral medial rectus muscle, and (4) the contralateral superior rectus muscle (Fig. 4-1). The superior oblique muscles are innervated by the trochlear nerve (cranial nerve IV), and the lateral rectus muscles are innervated by the abducens nerve (cranial nerve VI) (see Chapters 5 and 7). The actions of the six extraocular muscles are summarized in Table 4-1. The general somatic efferent fibers of the oculomotor nerve also provide motor in-

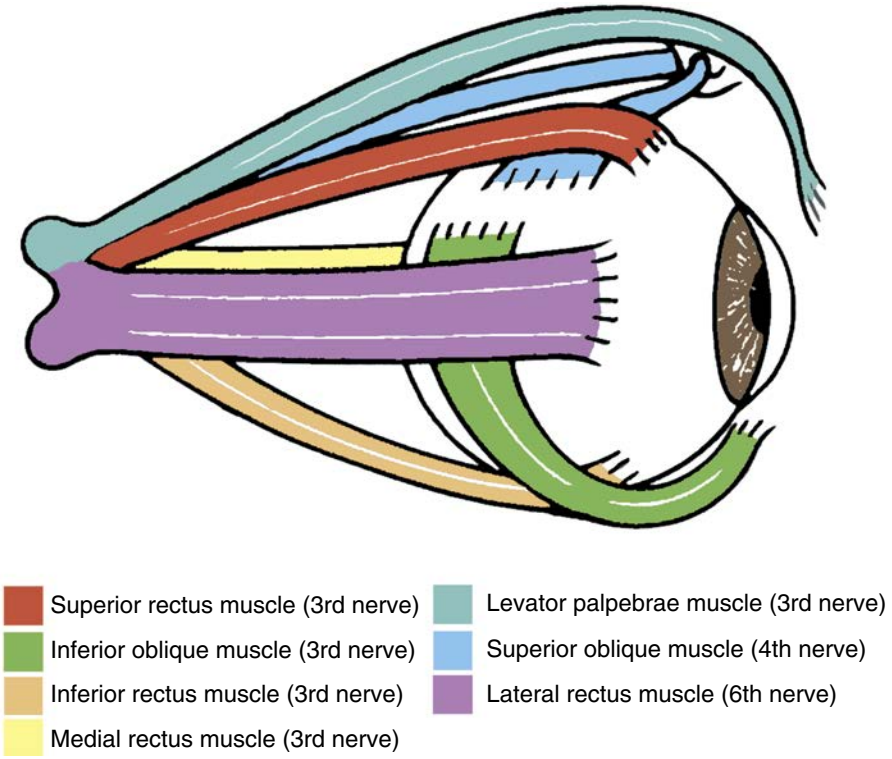

Fig. 4-1 The extraocular muscles. (From Wojno TH: Orbital Disease. In: Palay DA, Krachmer JH [eds], Primary Care Ophthalmology, ed 2. Philadelphia, Mosby, 2005, pp 275-292.)

Table 4-1 Actions of the Extraocular Muscles

Muscle	Innervation	Primary Action	Secondary Action	Tertiary Action
Superior rectus	CN III	Elevation	Intorsion	Adduction
Medial rectus	CN III	Adduction	...	...
Inferior rectus	CN III	Depression	Extorsion	Adduction
Inferior oblique	CN III	Extorsion	Elevation	Abduction
Superior oblique	CN IV	Intorsion	Depression	Abduction
Lateral rectus	CN VI	Abduction	...	...

CN, cranial nerve.

nervation to levator palpebrae superioris muscles bilaterally, which elevate the upper eyelids (Fig. 4-2).

The general somatic efferent fibers of the oculomotor nerve that provide motor innervation to four of the six extraocular muscles originate from the oculomotor nucleus located near the midline just ventral to the cerebral aqueduct in the rostral midbrain at the level of the superior colliculus. The oculomotor nucleus is bordered medially by the Edinger-Westphal nucleus (see later). Efferent general somatic fibers exit the oculomotor nucleus and pass ventrally in the tegmentum of the midbrain, passing through the red nucleus and medial portion of the cerebral peduncle to emerge in the interpeduncular fossa at the junction of the midbrain and pons.

Exiting the brainstem, the oculomotor nerve (cranial nerve III) passes between the posterior cerebral and superior cerebellar arteries and then passes through the dura mater to enter the cavernous sinus. The nerve runs along the lateral wall of the cavernous sinus just superior to the trochlear nerve (cranial nerve IV) and enters the orbit via the superior orbital fissure (Fig. 4-3). After entering the orbit, the oculomotor nerve passes through the tendinous ring of the extraocular muscles and then divides into the superior and inferior divisions. The superior division travels superiorly just lateral to the optic nerve to innervate both the superior rectus and levator palpebrae superioris muscles. The inferior division of oculomotor nerve divides into three branches to innervate the medial rectus, inferior rectus, and inferior oblique muscles (see Fig. 4-2).

The general visceral efferent motor fibers of the oculomotor nerve mediate the eye's accommodation and pupillary light reflexes by providing parasympathetic innervation of the constrictor pupillae and ciliary muscles of the eye (see Fig. 4-2). After entering the orbit, preganglionic parasympathetic fibers leave the inferior division of the oculomotor nerve to synapse in the ciliary ganglion, which lies deep to the superior rectus muscle near the tendinous ring of the extraocular muscles (see Fig. 4-2). Postganglionic fibers exit the ciliary ganglion via the short ciliary nerves, which enter the posterior aspect of the globe at a point near the spot where the optic nerve exits the eye. Traveling anteriorly

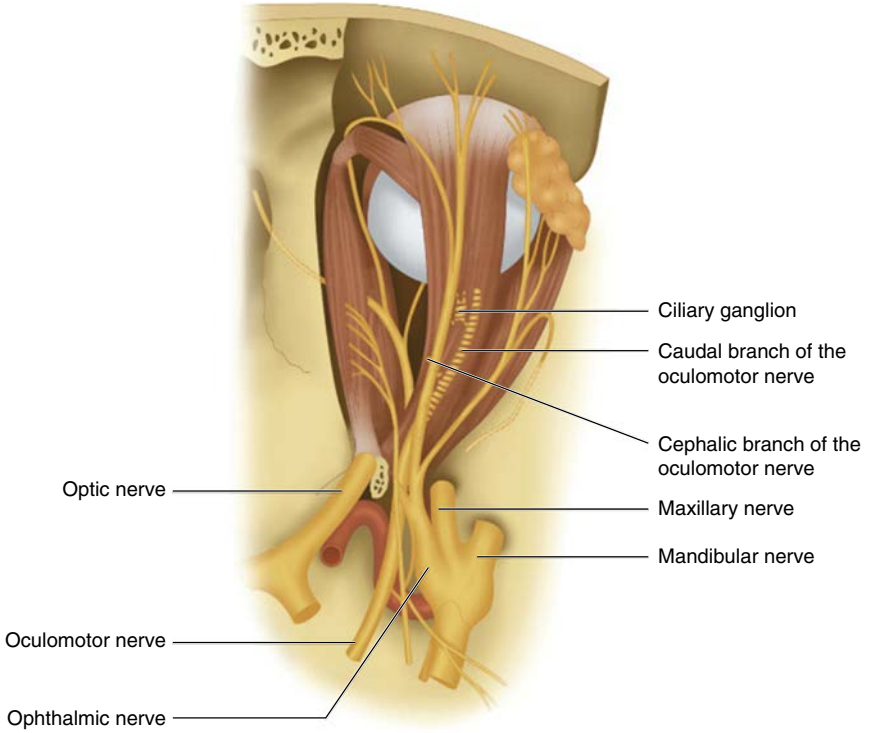

Fig. 4-2 The oculomotor nerve. (From Jean-Pierre Barral: Manual Therapy for the Cranial Nerves. Edinburgh, Churchill Livingstone, 2009; Fig. 12-1.)

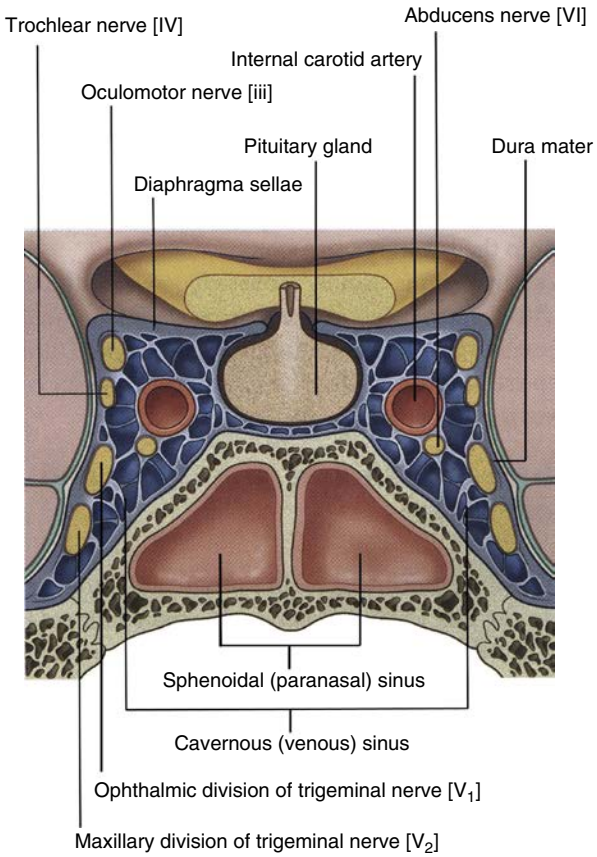

Fig. 4-3 Exiting the brainstem, the oculomotor nerve (cranial nerve III) passes between the posterior cerebral and superior cerebellar arteries and then passes through the dura mater to enter the cavernous sinus. The nerve runs along the lateral wall of the cavernous sinus just superior to the trochlear nerve (cranial nerve IV) and enters the orbit via the superior orbital fissure. (Reprinted from Drake R, Vogl W, Mitchell A: Gray's Anatomy for Students, ed 2. London, Churchill Livingstone, 2010; with permission.)

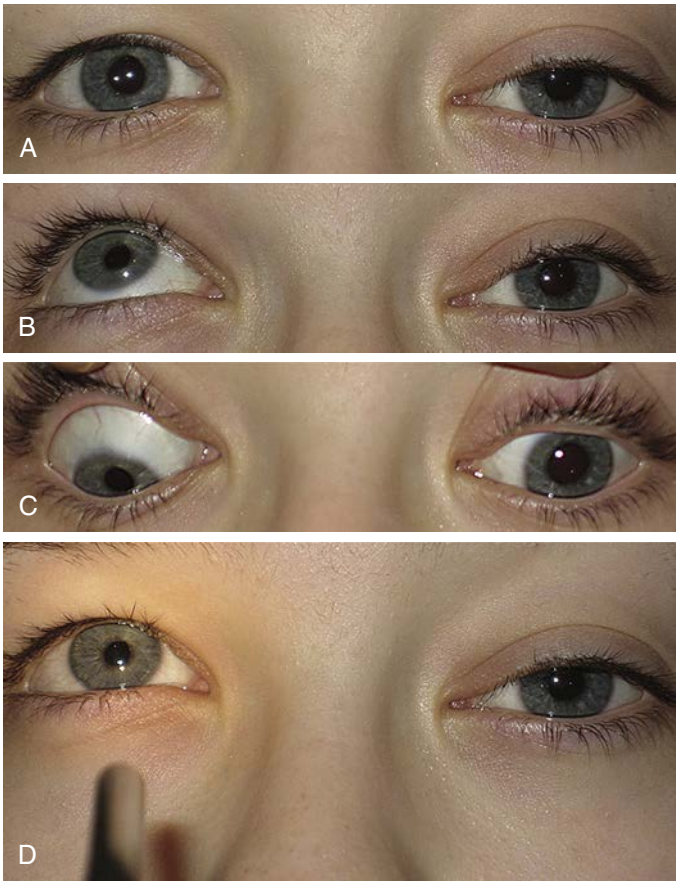

Fig. 4-4 A, B, C, D In almost all disorders of the oculomotor nerve, symptoms will take the form of either a palsy of the extraocular muscles presenting as diplopia, strabismus, or an inability to look upward or downward or by a ptosis of the eyelids. (From Prasad S, Volpe NJ: Paralytic strabismus: third, fourth, and sixth nerve palsy. Neurol Clin 2010; 28[3] pp 803-833.)

between the choroid and the sclera, these postganglionic fibers innervate the ciliary muscles, which alter the shape of the lens, as well as the constrictor muscle of the iris, which constricts the aperture of the iris (see Fig. 4-2).

Disorders of the oculomotor nerve can be caused by central lesions that affect the oculomotor or Edinger-Westphal nuclei such as stroke or space-occupying lesions such as tumor, abscess, or aneurysm. Increased intracranial pressure due to subdural hematoma, sagittal sinus thrombosis, or abscess can compromise the nuclei and/or the efferent fibers of the oculomotor nerve as they exit the brainstem and travel toward the orbit, with resultant abnormal nerve function. Traction on the oculomotor nerve due to loss of cerebrospinal fluid has also been implicated in cranial nerve III palsy. Small vessel disease due to diabetes or vasculitis associated with temporal arteritis may cause ischemia and even infarction of the oculomotor nerve with resultant pathologic symptoms.

In almost all disorders of the oculomotor nerve, symptoms will take the form of either a palsy of the extraocular muscles presenting as diplopia, strabismus, or an inability to look upward or downward or by a ptosis of the eyelids (Fig. 4-4). Compromise of the visceral fibers of the oculomotor nerve can result in anisocoria, the loss of the direct or consensual light reflex, and/or the loss of accommodation. Examples of these abnormalities include the Argyll Robertson pupil most frequently associated with syphilis, Adie's pupil, and the Marcus Gunn pupil.

Suggested Readings

Brazis PW: Isolated palsies of cranial nerves III, IV, and VI, Seminars in Neurology, 29 (2009), pp 14–28.

Prasad S, Volpe NJ: Paralytic strabismus: third, fourth, and sixth nerve palsy, Neurologic Clinics Volume 28(3):803–833, 2010 August.

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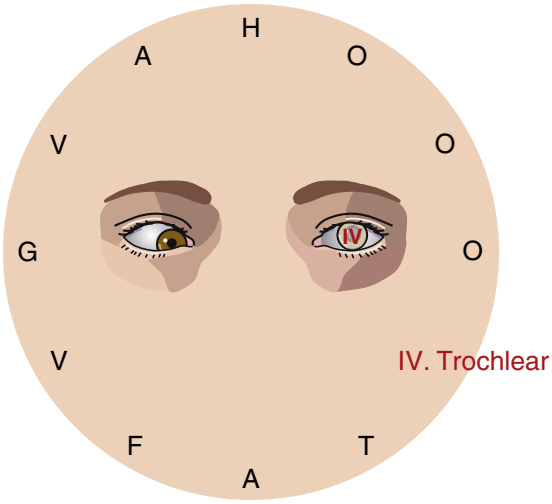
Waldman SD: Post-dural puncture headache. In: Waldman SD (ed): Atlas of Uncommon Pain Syndromes, ed 3. Philadelphia, Saunders, 2015.

CHAPTER 5
The Trochlear Nerve—Cranial Nerve IV

The trochlear nerve (cranial nerve IV) is composed of somatic general efferent motor fibers and is denoted by the Roman numeral IV. It innervates the superior oblique extraocular muscle of the contralateral orbit (Fig. 5-1). Contraction of the superior oblique extraocular muscle intorts (rotates inward), depresses, and abducts the globe. As outlined in Chapter 4, the superior oblique extraocular muscles work in concert with the five other extraocular muscles to allow the eye to perform its essential functions of tracking and fixation on objects.

The fibers of the trochlear nerve originate from the trochlear nucleus, which is just ventral to the cerebral aqueduct in the tegmentum of the midbrain at the level of the inferior colliculus. As the trochlear nerve leaves the trochlear nucleus, it travels dorsally, wrapping itself around the cerebral aqueduct to then decussate in the superior medullary velum. The decussated fibers of the trochlear nerve then exit the dorsal surface of the brainstem just below the contralateral inferior colliculus, where they then curve around the brainstem, leaving the subarachnoid space along with the oculomotor nerve (cranial nerve III) between the superior cerebellar and posterior cerebral arteries (Fig. 5-2). The trochlear nerve then enters the cavernous sinus and runs anteriorly along the lateral wall of the sinus with the oculomotor (cranial nerve III), trigeminal (cranial nerve V), and abducens (cranial nerve VI) nerves.

Exiting the cavernous sinus, the trochlear nerve enters the orbit via the superior orbital fissure. Unlike the oculomotor nerve, the trochlear nerve does not pass through the tendinous ring of the extraocular muscles but passes just above the ring (Fig. 5-3). The trochlear nerve then crosses medially along the roof of the orbit above the levator palpebrae and superior rectus muscles to innervate the superior oblique muscle (see Fig. 5-2).



IV. Trochlear nerve
- Motor
  - Eye movement
  - Superior oblique (S.O.) muscle

Fig. 5-1 The trochlear nerve (cranial nerve IV) is composed of somatic general efferent motor fibers and is denoted by the Roman numeral IV. It innervates the superior oblique extraocular muscle of the contralateral orbit.

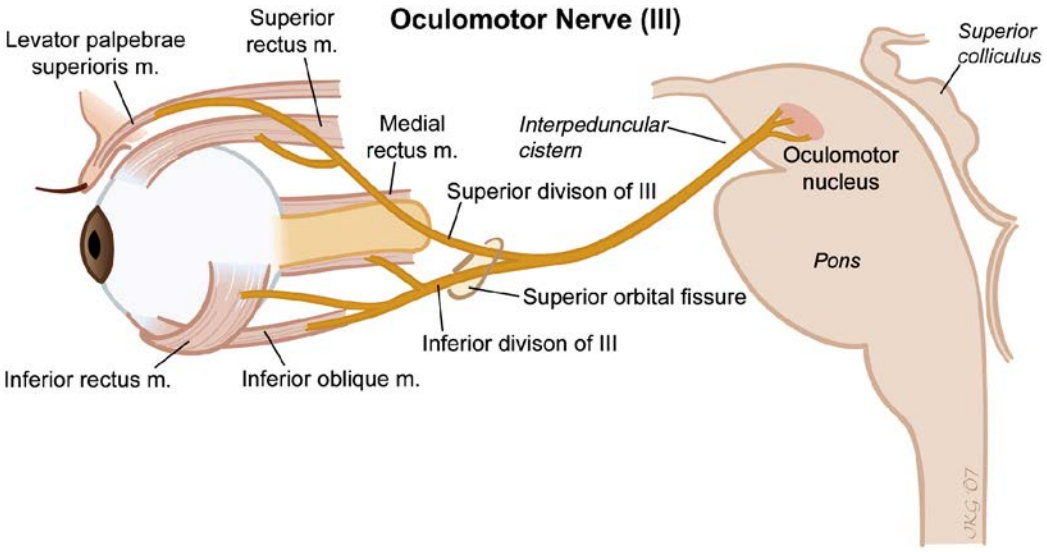


Fig. 5-2 The relationship of the trochlear nerve and the superior oblique extraocular muscle. (From Smoker WRK, Reede DL: Denervation atrophy of motor cranial nerves. Neuroimaging Clinics of North America 2008 May; 18[2]:387-411.)

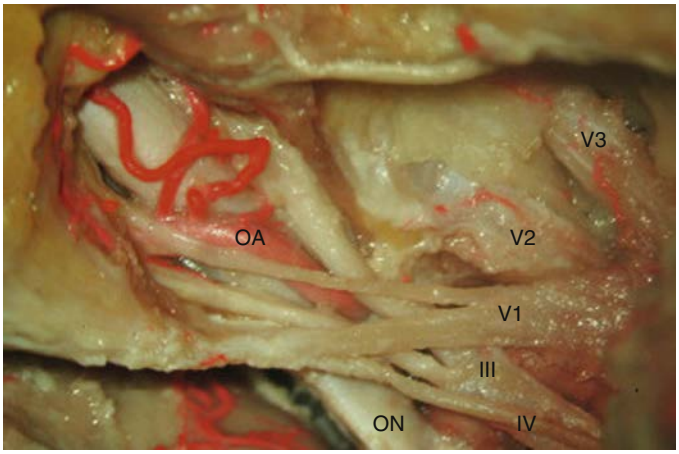


Fig. 5-3 The course of the trochlear nerve. (From Iaconetta G, Notaris MD Galino AP: Chapter 21 - Anatomy of the Trochlear Nerve. In: Tubbs RS, Rizk E, Shoja MM, et al [eds]. Nerves and Nerve Injuries. San Diego, Academic Press, 2015, pp 311-317.)

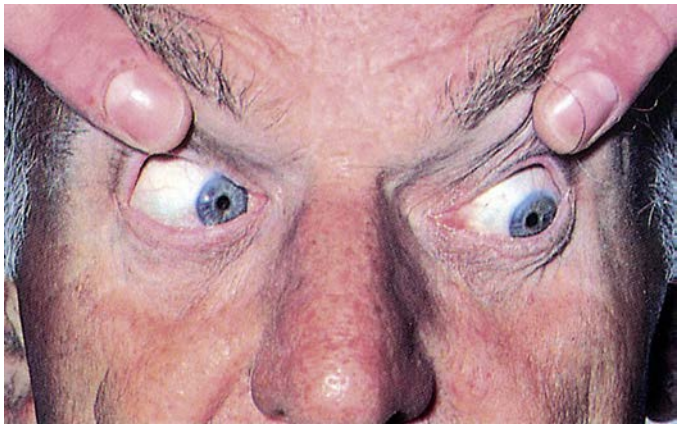


Fig. 5-5 Clinical examination: right fourth nerve palsy in a 65-year-old man with a 6-month history of vertical image separation. Note reduced downward and inward gaze of the right eye. (From Smoker WRK, Reede DL: Denervation atrophy of motor cranial nerves. Neuroimag Clin N Am 2008 May; 18[2]: 387-411.)

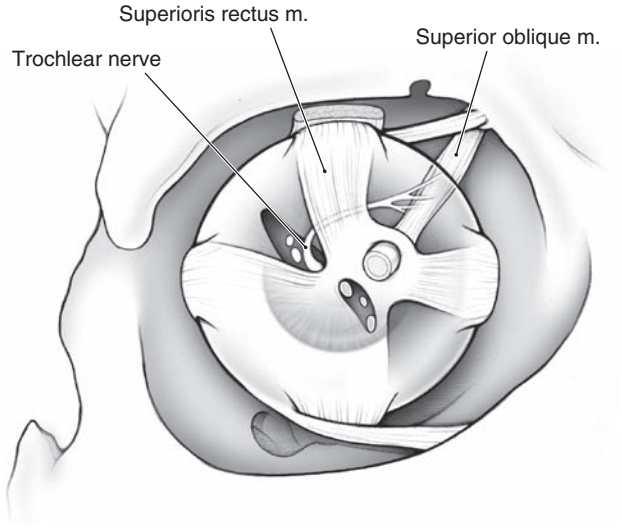


Fig. 5-4 The relationship of the terminal trochlear nerve to the orbit and tendinous ring of the extraocular muscles.

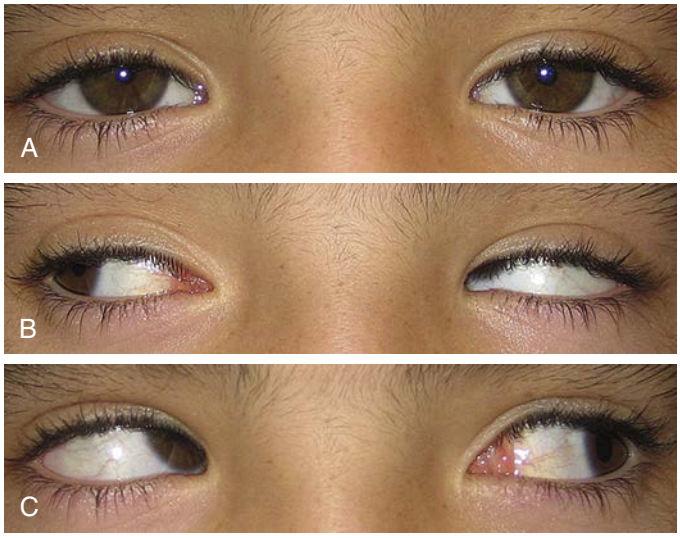


Fig. 5-6 A 7-year-old girl with bilateral congenital fourth nerve palsy. Brain MRI was normal. (A) Normal alignment in primary gaze. (B) Left hypertropia in right gaze, with left inferior oblique overaction. (C) Right hypertropia in left gaze, with right inferior oblique overaction. (From Prasad S, Volpe NJ: Paralytic strabismus: third, fourth, and sixth nerve palsy. Neurol Clin 2010 Aug; 28[3]:803-833.)

Disorders of the trochlear nerve can be caused by central lesions that affect the trochlear nucleus such as stroke or space-occupying lesions such as tumor, abscess, or aneurysm. Increased intracranial pressure due to subdural hematoma, sagittal sinus thrombosis, or abscess can compromise the nucleus and/or the efferent fibers of the trochlear nerve as they exit the brainstem and travel toward the orbit, with resultant abnormal nerve function. Traction on the trochlear nerve due to loss of cerebrospinal fluid has also been implicated in cranial nerve IV palsy. Small vessel disease due to diabetes or vasculitis associated with temporal arteritis may cause ischemia and even infarction of the trochlear nerve, with resultant pathologic symptoms.

In almost all disorders of the trochlear nerve, symptoms will take the form of a palsy of the superior oblique muscle, most commonly presenting as the inability to look inward and downward (Fig. 5-5). Often, the patient will complain of the difficulty in walking down stairs because of the inability to depress the affected eye or eyes. On physical examination, the clinician may note extorsion (outward rotation) of the affected eye because of the unopposed action of the inferior oblique muscle (Fig. 5-6). In an effort to compensate, the patient may deviate his or her face forward and downward with the chin rotated toward the affected side in order to look downward.

Suggested Readings

Brazis PW: Isolated palsies of cranial nerves III, IV, and VI, Seminars in Neurology 29:14-28, 2009.

Iaconetta G, Notaris M: Galino, AP: Anatomy of the trochlear nerve. In: Tubbs RS, Rizk E, Shoja MM, Loukas M, Barbaro N (eds): Nerves and Nerve Injuries, San Diego, Academic Press, 2015, pp 311–317.

Prasad S, Volpe NJ: Paralytic strabismus: third, fourth, and sixth nerve palsy, Neurologic Clinics 28:803–833, 2010 August.

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Waldman SD: Post-dural puncture headache. In: Waldman SD (ed): Atlas of Uncommon Pain Syndromes, ed 3. Philadelphia, Saunders, 2015.

CHAPTER 6
The Trigeminal Nerve—Cranial Nerve V

The trigeminal nerve is the fifth cranial nerve and is denoted by the Roman numeral V. The trigeminal nerve has three divisions and provides sensory innervation for the forehead and eye (ophthalmic V_1), cheek (maxillary V_2), and lower face and jaw (mandibular V_3), as well as motor innervation for the muscles of mastication (Fig. 6-1). The fibers of the trigeminal nerve arise in the trigeminal nerve nucleus, which is the largest of the cranial nerve nuclei. Extending from the midbrain to the upper cervical spinal cord, the trigeminal nerve nucleus is divided into three parts: (1) the mesencephalic trigeminal nucleus, which receives proprioceptive and mechanoreceptor fibers from the mandible and teeth; (2) the main trigeminal nucleus, which receives the majority of the touch and position fibers; and (3) the spinal trigeminal nucleus, which receives pain and temperature fibers.

The sensory fibers of the trigeminal nerve exit the brainstem at the level of the mid-pons with a smaller motor root emerging from the mid-pons at the same level. These roots pass in a forward and lateral direction in the posterior cranial fossa across the border of the petrous bone. They then enter a recess called *Meckel's cave*, which is formed by an invagination of the surrounding dura mater into the middle cranial fossa. The dural pouch that lies just behind the ganglion is called the *trigeminal cistern* and contains cerebrospinal fluid.

The gasserian ganglion is canoe shaped, with the three sensory divisions: (1) the ophthalmic division (V_1), which exits the cranium via the superior orbital fissure; (2) the maxillary division (V_2), which exits the cranium via the foramen rotundum into the pterygopalatine fossa where it travels anteriorly to enter the infraorbital canal to exit through the infraorbital foramen; and the mandibular division (V_3), which exits the cranium via the foramen ovale anterior convex aspect of the ganglion (Fig. 6-2). A small motor root joins the mandibular division as it exits the cranial cavity via the foramen ovale.

Three major branches emerge from the trigeminal ganglion (Fig. 6-3). Each branch innervates a different dermatome. Each branch exits the cranium through a different site. The first division (V_1; ophthalmic nerve) exits the cranium through the superior orbital fissure, entering the orbit to innervate the globe and skin in the area above the eye and forehead.

The second division, V_2, maxillary nerve, exits through a round hole, the foramen rotundum, into a space posterior to the orbit, the pterygopalatine fossa. It then reenters a canal running inferior to the orbit, the infraorbital canal, and exits through a small hole, the infraorbital foramen, to innervate the skin below the eye and above the mouth. The third division, V_3, mandibular nerve, exits the cranium through an oval hole, the foramen ovale. Sensory fibers of the third division either travel directly to their target tissues or reenter the mental canal to innervate the teeth, with the terminal branches of this division exiting anteriorly via the mental foramen to provide sensory cutaneous innervation to the skin overlying the mandible.

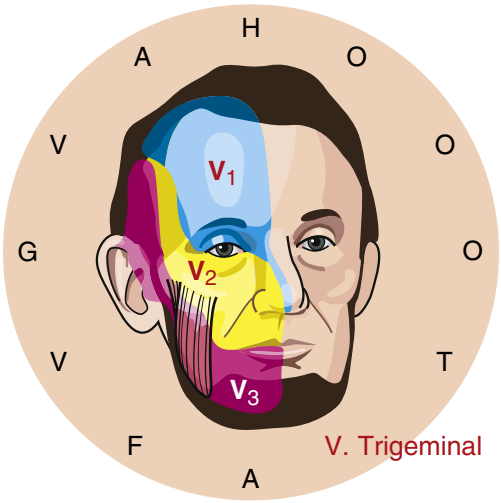


Fig. 6-1 The trigeminal nerve is the fifth cranial nerve and is denoted by the Roman numeral V. The trigeminal nerve has three divisions and provides sensory innervation for the forehead and eye (ophthalmic V_1), cheek (maxillary V_2), and lower face and jaw (mandibular V_3), as well as motor innervation for the muscles of mastication.

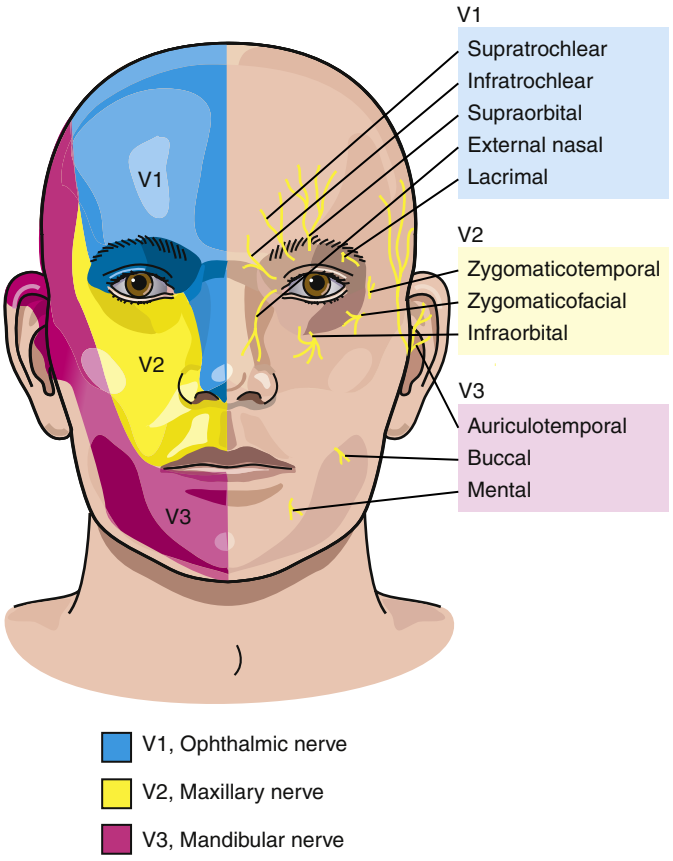


Fig. 6-2 The sensory divisions and peripheral branches of the trigeminal nerve. (From Waldman SD: Atlas of Interventional Pain Management, ed 4. Philadelphia, Saunders, 2015; Fig. 16-1.)

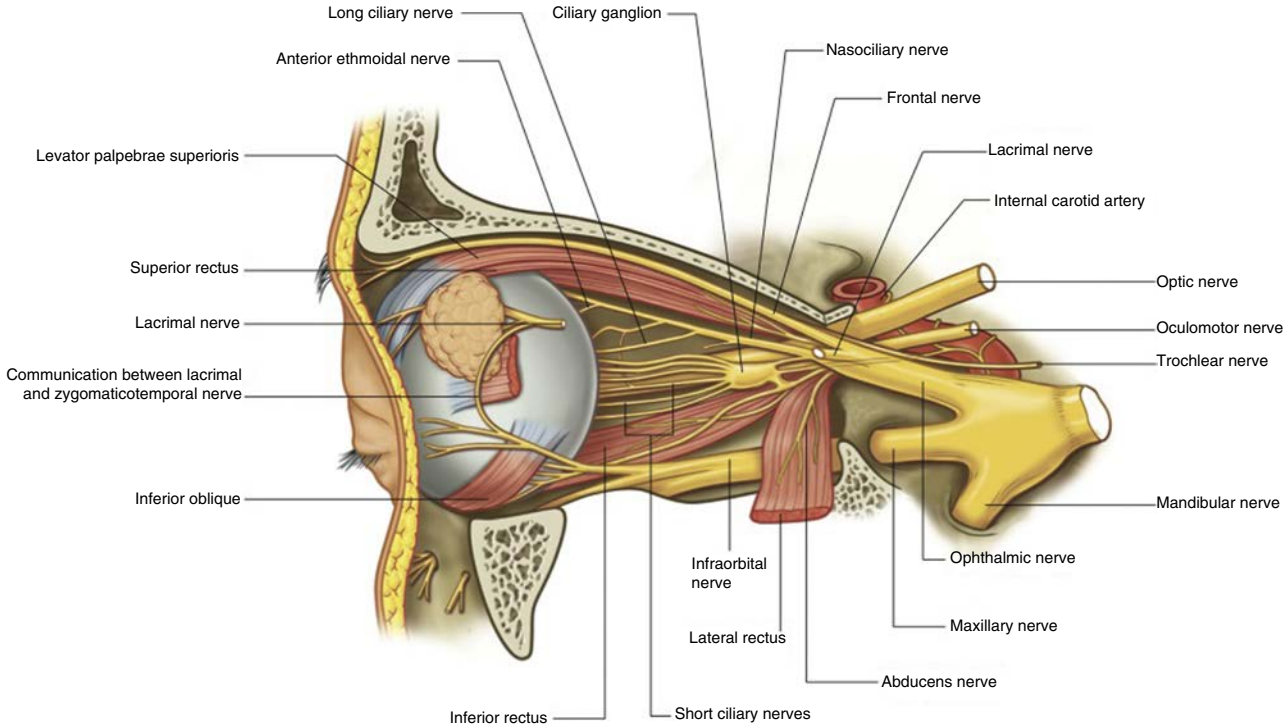


Fig. 6-3 The gasserian ganglion and branches of the trigeminal nerve. (Reproduced with permission. Image published in Standring S, Gray's Anatomy, ed 40. The orbit and accessory visual apparatus. London, Churchill Livingstone, 2008, p 668.)

Table 6-1 Disorders of the Trigeminal Nerve

Painful Conditions
Trigeminal neuralgia Postherpetic neuralgia Trigeminal autonomic cephalgias
Sensory Disturbance
Anesthesia Hypoesthesia Paresthesia Dysesthesia Anesthesia dolorosa Wallenberg syndrome involving the trigeminal nucleus Abnormal touch-position Abnormal two-point discrimination Abnormal conscious proprioception
Hearing Impairment
Impaired hearing from paralysis of the tensor tympani muscle
Motor Impairment
Weakness and atrophy of the muscles of mastication Deviation of the mandible to the affected side Trismus
Impairment of Trigeminal Reflexes
Corneal reflex Jaw jerk reflex Sneeze reflex

Disorders of the trigeminal nerve generally take the form of trigeminal neuralgia, but loss of sensation, impairment of hearing secondary to paralysis of the tensor tympani muscle, weakness of muscles of mastication, and loss of trigeminal reflexes can occur (Table 6-1). Trigeminal neuralgia occurs in many patients because of tortuous blood vessels that compress the trigeminal root as it exits the brainstem. Acoustic neuromas, cholesteatomas, aneurysms, angiomas, and bony abnormalities of the skull may also lead to the compression of the trigeminal nerve. The severity of pain produced by trigeminal neuralgia can only be rivaled by that of cluster headache. Uncontrolled pain has been associated with suicide and therefore should be treated as an emergency. Attacks can be triggered by daily activities involving contact with the face such as brushing the teeth, shaving, or washing. Pain can be controlled with medication in most patients. About 2% to 3% of those patients experiencing trigeminal neuralgia also have multiple sclerosis. Trigeminal neuralgia is also called *tic douloureux*. Rarely, isolated paresis of the motor branch of the mandibular division of the trigeminal nerve can occur (Fig. 6-4).

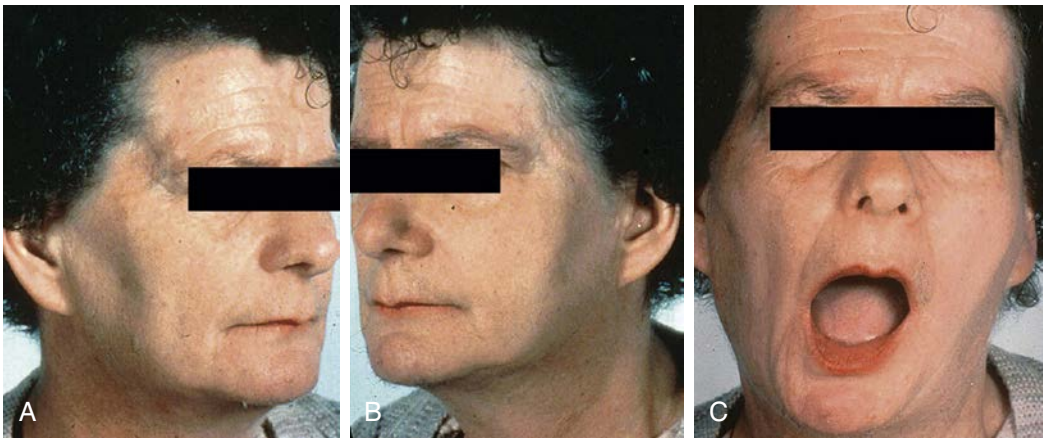


Fig. 6-4 Clinical examination: left V_3 paresis. Photographs are seen in closed-mouth right oblique (A), closed-mouth left oblique (B), and open-mouth (C) positions. Note the hollow concavity of the right cheek (masseter muscle atrophy) and temporal fossa (temporalis muscle atrophy) in (A) compared with the normal left side in (B). (C) On opening the mouth, the jaw deviates toward the right, the side of pathologic findings, because of unopposed action of the right pterygoid muscles. (From Perkin D, Rose FC, Blackwood W, et al: Atlas of Clinical Neurology. London, Gower Medical Publishing; 1986; with permission.)

Suggested Readings

Smoker WRK, Reede DL: Denervation atrophy of motor cranial nerves, Neuroimaging Clinics of North America 18(2):387–411, 2008 May.

Tubbs RS, Rizk E, Shoja MM, et al (eds): Anatomy of the trigeminal nerve. In: Hogan E (ed): Nerves and Nerve Injuries, San Diego, Academic Press, 2015, pp 319–350.

Waldman SD: Trigeminal neuralgia. In: Waldman SD (ed): Atlas of Common Pain Syndromes, ed 3. Philadelphia, Saunders, 2015.

CHAPTER 7

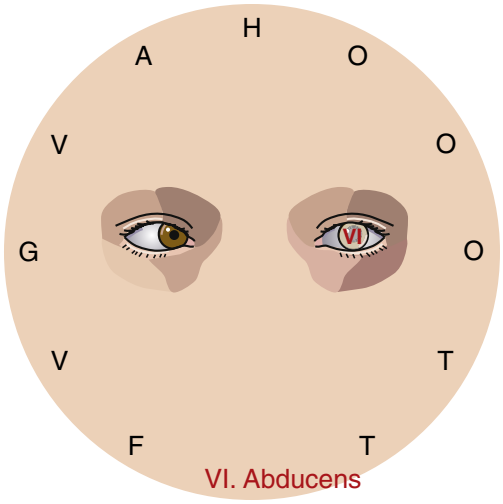
The Abducens Nerve—Cranial Nerve VI

The abducens nerve is the sixth cranial nerve and is denoted by the Roman numeral VI. The abducens nerve is composed of somatic general efferent motor fibers. It innervates the lateral rectus extraocular muscle of the ipsilateral orbit (Fig. 7-1, Fig. 7-2). Contraction of the lateral rectus extraocular muscle abducts the globe. As outlined in Chapter 4, the lateral rectus extraocular muscle works in concert with the five other extraocular muscles to allow the eye to perform its essential functions of tracking and fixation of objects.

The fibers of the abducens nerve originate from the abducens nucleus, which is located just ventral to the fourth ventricle in the caudal pons at the level of the facial colliculus. As the abducens nerve leaves the abducens nucleus, it travels ventrally, exiting the brainstem at the border of the pons and medullary pyramids. The abducens nerve then courses superiorly adjacent to the ventral surface of the pons where, upon reaching the apex of the petrous portion of the temporal bone, the nerve abruptly turns anteriorly to enter the cavernous sinus (Fig. 7-3). After entering the cavernous sinus, the abducens nerve runs anteriorly along the lateral wall of the sinus with the oculomotor (cranial nerve III), trochlear (cranial nerve IV), and trigeminal (cranial nerve V) nerves. Exiting the cavernous sinus, the abducens nerve enters the orbit via the superior orbital fissure and passes through the tendinous ring of the extraocular muscles to innervate the lateral rectus muscle (see Fig. 7-3).

Disorders of the abducens nerve can be caused by central lesions that affect the abducens nucleus such as stroke (especially of the pons) or space-occupying lesions such as tumor, abscess, or aneurysm. Increased intracranial pressure due to subdural hematoma, sagittal sinus thrombosis, or abscess can compromise the nucleus and/or the efferent fibers of the abducens nerve as they exit the brainstem and travel toward the orbit, with resultant abnormal nerve function. Traction on the abducens nerve due to loss of cerebrospinal fluid has also been implicated in cranial nerve VI palsy. Small vessel disease due to diabetes or vasculitis associated with temporal arteritis may cause ischemia and even infarction of the abducens nerve, with resultant pathologic symptoms. Statistically, microvascular disease associated with diabetes is far and away the most common cause of isolated abducens (cranial nerve VI) palsy.

In almost all disorders of the abducens nerve, symptoms will take the form of a palsy of the lateral rectus muscle. This most commonly presents clinically as horizontal diplopia, with the patient unable to fixate on an



VI. Abducens nerve
- Motor
  - Eye movement
  - Lateral rectus (L.R.) muscle

Fig. 7-1 The abducens nerve is the sixth cranial nerve and is denoted by the Roman numeral VI. The abducens nerve is composed of somatic general efferent motor fibers. It innervates the lateral rectus extraocular muscle of the ipsilateral orbit. Contraction of the lateral rectus extraocular muscle abducts the globe.

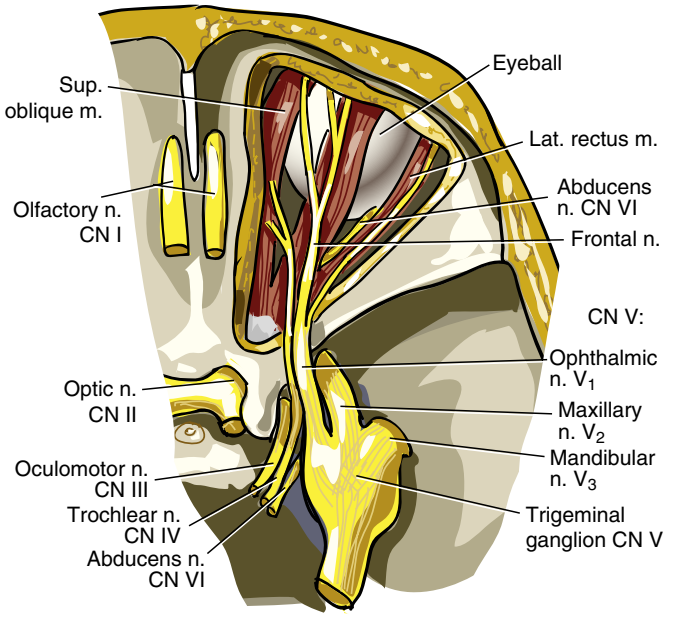


Fig. 7-2 The relationship of the abducens nerve and the lateral rectus muscle. (From Waldman SD: Pain Management, 2nd ed. Color drawings by Joseph I. Bloch. Philadelphia, Saunders, 2011, Fig. 42-2.)

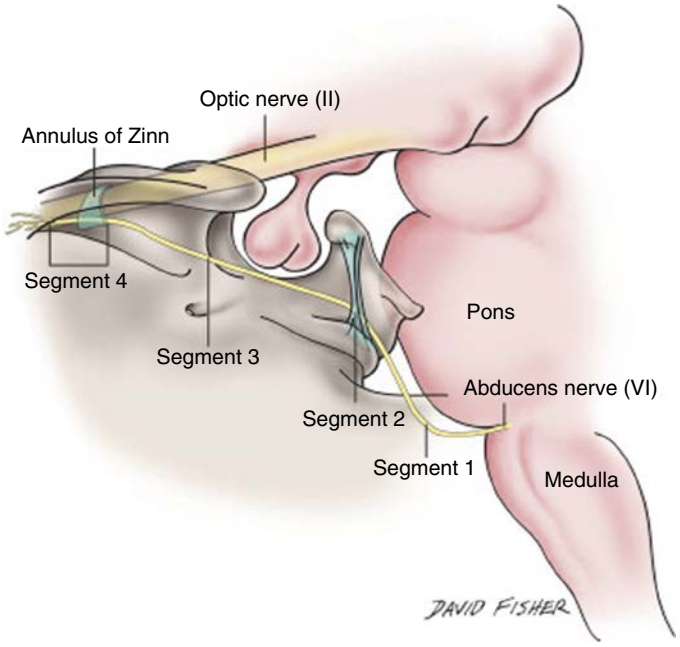


Fig. 7-3 The course of the abducens nerve. Occulomotor nerve (III), Muscle (M). (From Smoker WRK, Reede DL: Denervation atrophy of motor cranial nerves. Neuroimaging Clinics of North America, 18(2):387-411, May 2008.)

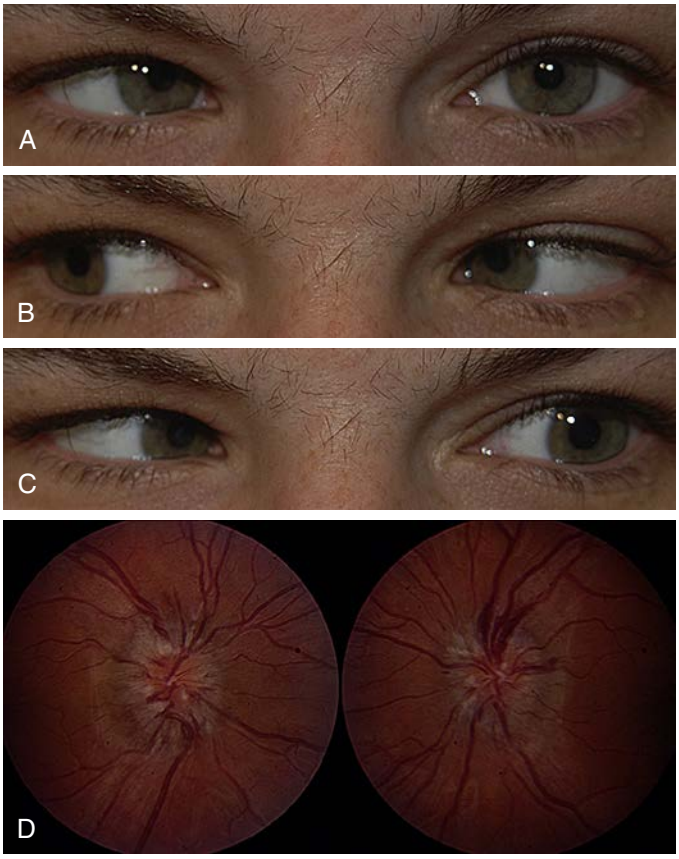


Fig. 7-4 A 29-year-old woman with left sixth nerve palsy due to pseudotumor cerebri following pregnancy. Brain MRI, MR venogram, and CSF constituents were normal. Opening pressure was 35 cm H_2O. (A) Esotropia in primary gaze. (B) Normal right gaze. (C) Left abduction deficit with intact right adduction. (D) bilateral papilledema (From Prasad S, Volpe NJ, Paralytic strabismus: third, fourth, and sixth nerve palsy. Neurologic Clinics, 28(3):803-833, August 2010.)

object placed laterally to the affected side. Clinically, the patient will be unable to abduct the eye on the affected side past the midline gaze combined with the inability to adduct the eye opposite the lesion past midline gaze (Fig. 7-4).

Suggested Readings

Prasad S, Volpe NJ: Paralytic strabismus: third, fourth, and sixth nerve palsy, Neurologic Clinics 28(3):803–833, August 2010.

Waldman SD: Post-dural puncture headache. In: Waldman SD (ed): Atlas of Uncommon Pain Syndromes, ed 3. Philadelphia, Saunders, 2015.

Wojno TH: Orbital disease. In: Palay DA, Krachmer JH (eds): Primary Care Ophthalmology, ed 2. Philadelphia, Mosby, 2005, pp 275–292.

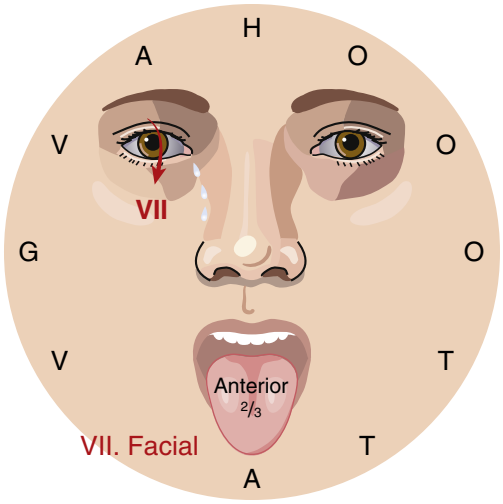
CHAPTER 8
The Facial Nerve—Cranial Nerve VII

The facial nerve is the seventh cranial nerve and is denoted by the Roman numeral VII. The facial nerve is made up of four types of fibers, each with its own unique function (Fig. 8-1). The first and most important type of fiber is the branchial motor special efferent component (Fig. 8-2). Making up the largest portion of facial nerve fibers, the branchial motor component provides voluntary control of the muscles of facial expression, including buccinator, occipitalis, and platysma muscles, as well as the posterior belly of the digastric, stylohyoid, and stapedius muscles.

The second functional component of the facial nerve is the visceral motor component, which is made up of general visceral efferent fibers (see Fig. 8-2). The visceral motor component provides parasympathetic innervation of the mucous membranes of nasopharynx, hard and soft palate, and the lacrimal, submandibular, and sublingual glands (see Fig. 8-2).

The third functional component of the facial nerve is the special sensory component, which is made up of special afferent fibers (see Fig. 8-2). The special sensory component provides taste sensation for the anterior two thirds of the tongue as well as the hard and soft palates (see Fig. 8-2).

The fourth functional component of the facial nerve is the general sensory component, which is made up of general somatic afferent fibers (see Fig. 8-2). The general sensory component of the facial nerve provides sensory innervation for the skin of the concha of the auricle and for a small area behind the ear. The visceral motor, special sensory, and general sensory components are covered in a clearly defined fascial sheath separate from the branchial motor special efferent fibers and collectively are known as the *nervus intermedius*.



VII. Facial nerve
- Motor
  - Facial expression
  - Cry, spit
  - Close eye
- Sensory
  - Taste anterior 2/3 of tongue

Fig. 8-1 The facial nerve is the seventh cranial nerve and is denoted by the Roman numeral VII. The facial nerve is made up of four types of fibers, each with its own unique function.

The facial nerve provides sensory, motor, and pre-ganglion parasympathetic fibers to the head. The motor portion of the nerve arises from the facial nerve nucleus of the pons (Fig. 8-3). The sensory portion of the nerve arises from the nervus intermedius at the inferior margin of the pons. It is at the point where the sensory portion of the nerve leaves the pons that it is susceptible to compression by aberrant blood vessels that can cause a trigeminal neuralgia–like syndrome known as *geniculate neuralgia* and a facial dystonia known as *hemifacial spasm*. After leaving the pons, the motor and sensory fibers of the facial nerve join to travel across the subarachnoid space and enter the internal auditory meatus to pass through the petrous temporal bone. It is at this point that swelling, infection, and inflammation of the facial nerve can cause Bell's palsy. The nerve then exits the base of the skull via the stylomastoid foramen. It passes downward and then turns forward to pass through the parotid gland, where it divides into fibers that provide innervation to the muscles of facial expression. The nerve is frequently injured during surgery of the parotid gland.

The most common disorder of the facial nerve encountered in clinical practice is Bell's palsy. Presenting as sudden paralysis of the muscles of facial expression, this disorder is quite distressing to the patient (Fig. 8-4). The signs and symptoms of Bell's palsy in addition to the facial paralysis are listed in Table 8-1. The intensity of symptoms associated with Bell's palsy can range from mild to severe with an onset to peak of 48 hours. While the exact etiology of Bell's palsy remains elusive, it is believed that the most likely cause of this cranial nerve palsy is nerve inflammation, swelling, and ischemia due to viral infection. The herpes simplex virus has been most commonly implicated in this disorder, and there is anecdotal evidence that the addition of acyclovir to a short course of oral prednisone will shorten the course of the disease and improve the outcome. However, the most

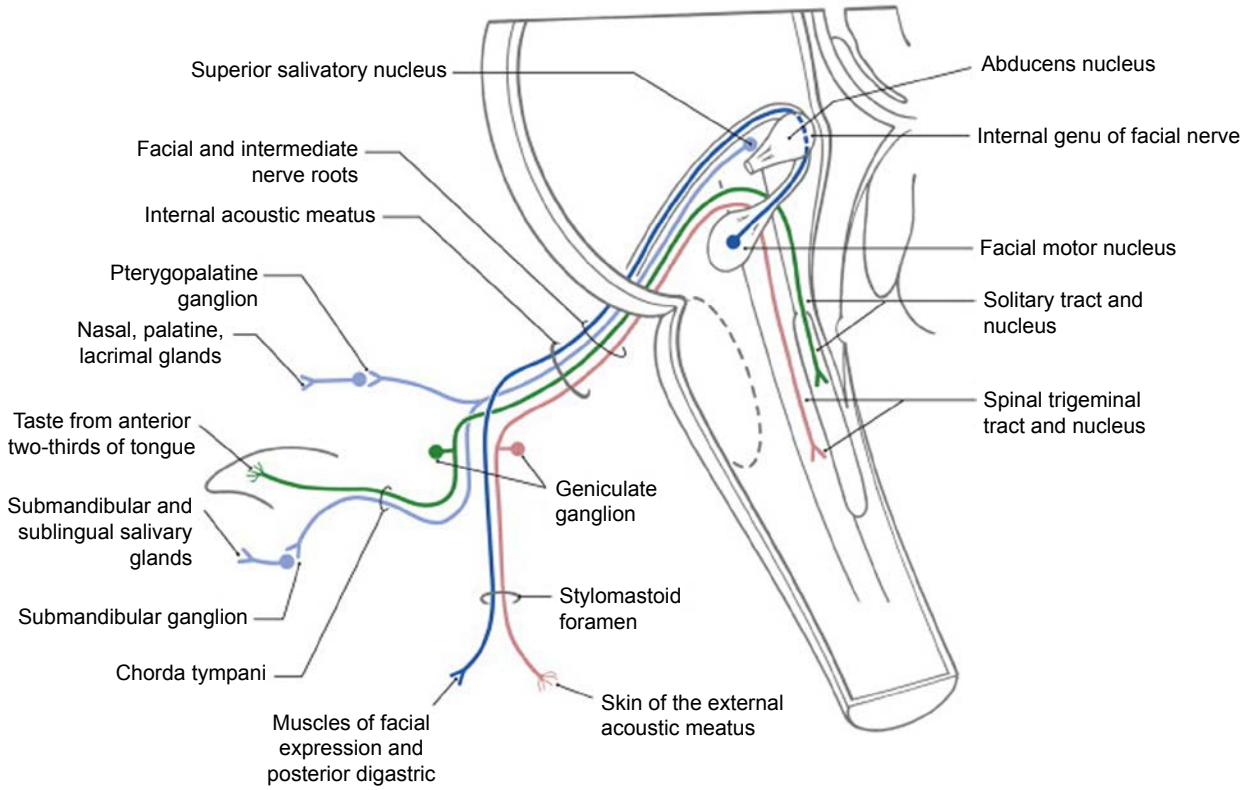


Fig. 8-2 The four functional components of the facial nerve. Facial Motor Efferent = Dark Blue Special Visceral Efferent = Light Blue General Sensory Afferent = Red Special Visceral Afferent = Green. (From Lysek MC Jr: Chapter 24 - Anatomy of the Facial Nerve. In: Tubbs RS, et al [eds]: Nerves and Nerve Injuries. San Diego, Academic Press, 2015, pp 357-369.

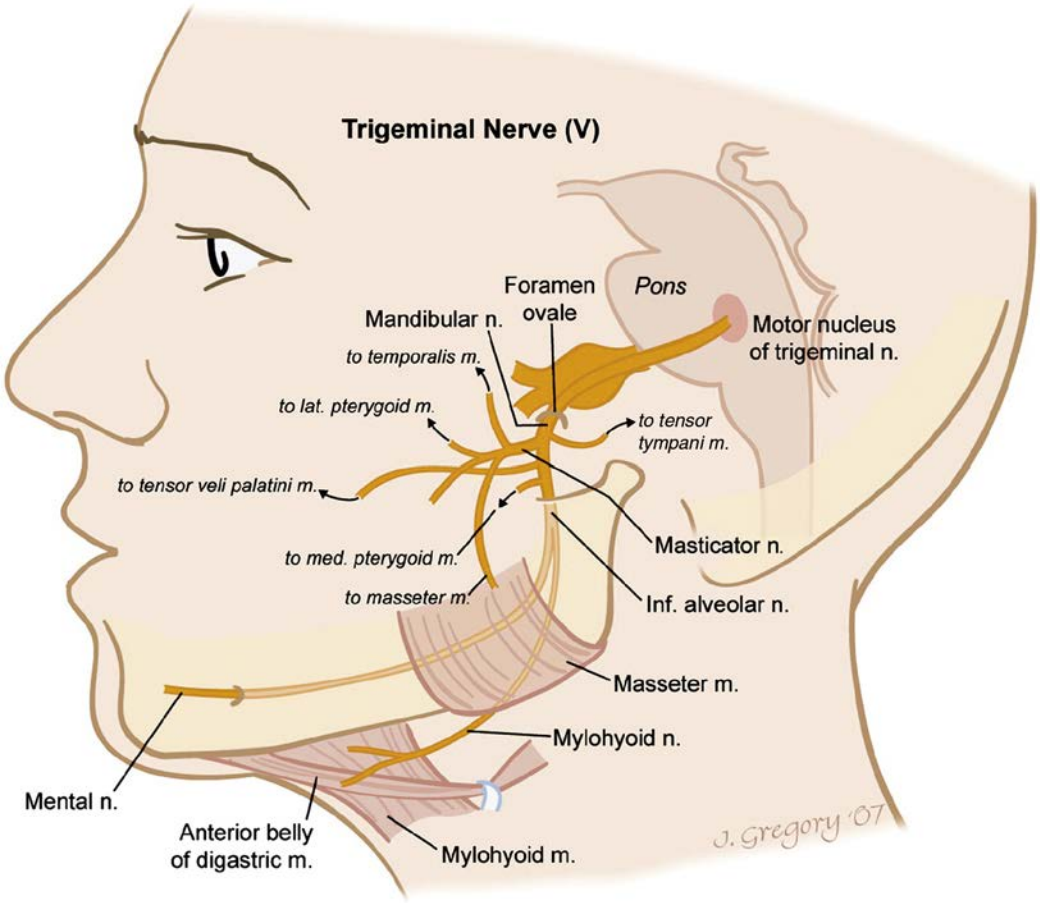


Fig. 8-3 Anatomy of the facial nerve. (From Smoker WRK, Reede DL: Denervation atrophy of motor cranial nerves. Neuroimag Clin N Am 2008; 18[2]:387-411.)

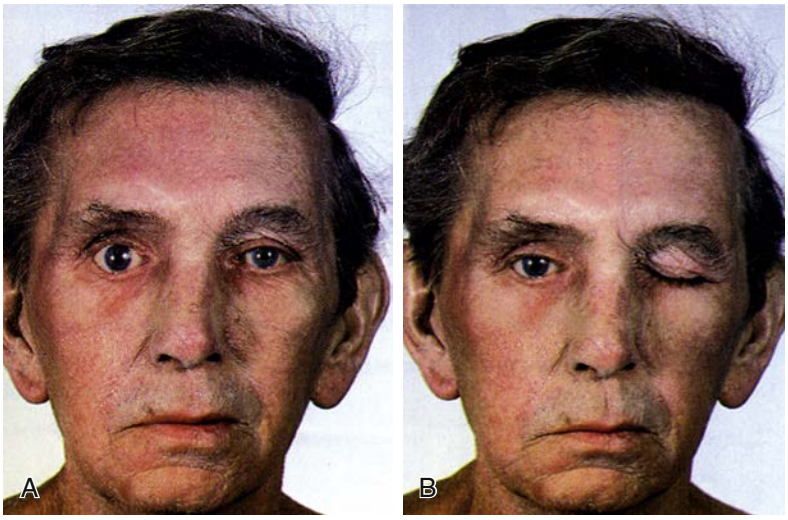


Fig. 8-4 Bell's palsy. Clinical examination: lower motor neuron right facial nerve paralysis. Photographs with eyes open (A) and eyes closed (B). Note involvement of all facial muscles with drooping of the mouth, flattening of the nasolabial fold, and inability to close the eye. (From Perkin D, Rose FC, Blackwood W, et al: Atlas of Clinical Neurology. London, Gower Medical Publishing, 1986; with permission.)