16 The Special Senses

Section through the human retina showing bipolar neurons (red), rod cells(white), and yellow cone cells (colored SEM).

People are responsive creatures. The aroma of baking bread makes our mouths water; lightning makes us blink; thunder makes us recoil. Many such sensory stimuli reach us each day and are processed by the nervous system.

We are usually told that we have five senses: touch, taste, smell, sight, and hearing. Actually, touch is a large group of general senses considered in Chapter 14. The other four traditional senses—*smell, taste, sight,* and *hearing*—are called special senses. Receptors for a fifth special sense—*equilibrium,* or the sense of balance—are located in the ear.

In contrast to the widely distributed receptors for the general senses, the **special sensory receptors** are localized and confined to the head region. The receptors for the special senses are not free endings of sensory neurons but distinct **receptor cells.** These are neuronlike epithelial cells or small peripheral neurons that transfer sensory information to other neurons in afferent pathways to the brain. The special sensory receptors are housed either in complex sensory organs (eye or ear) or in distinctive epithelial structures (taste buds or olfactory epithelium). Their sensory information travels to the brain via cranial nerves.

This chapter explores the functional anatomy of the five special senses: the chemical senses of taste and smell, which are special visceral senses (p. 350), and the special somatic senses of vision in the eye, and hearing and equilibrium in the ear.

THE CHEMICAL SENSES: TASTE AND SMELL

➤ Describe the receptors for taste and smell. Describe the paths by which sensory information from these receptors travels to the brain.

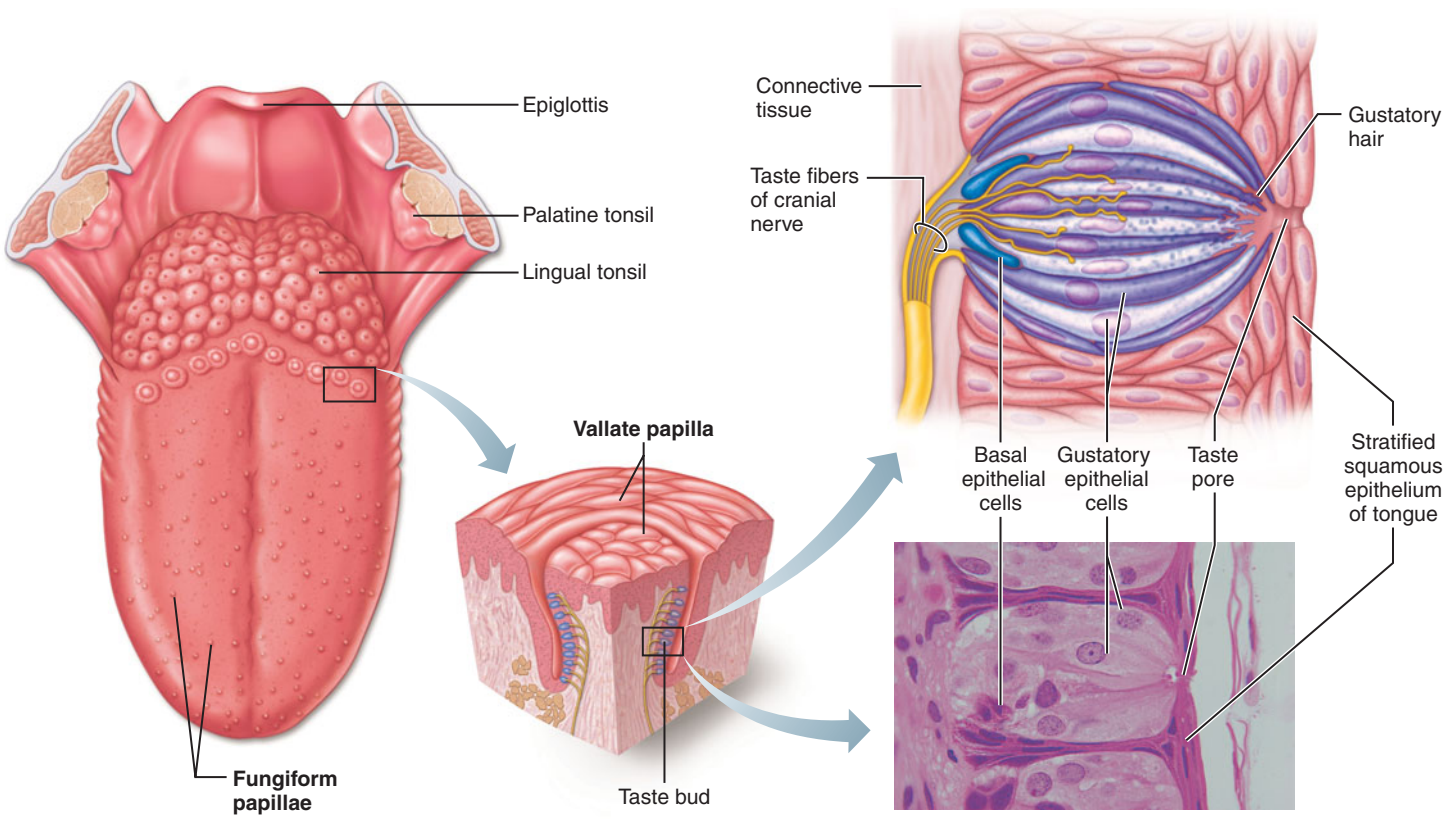
The receptors for taste (gustation) and smell (olfaction) are classified as **chemoreceptors** because they respond to chemical substances—to food chemicals dissolved in saliva and to airborne chemicals that dissolve in fluids on the nasal membranes, respectively.

Taste (Gustation)

Taste Buds

The taste receptors occur in **taste buds** in the mucosa of the mouth and pharynx. The majority of the 10,000 or so taste buds are on the surface of the tongue; a few others occur on the posterior region of the palate (roof of the mouth), on the inner surface of the cheeks, on the posterior wall of the pharynx, and on the epiglottis (a leaf-shaped flap behind the tongue).

Most taste buds occur in peglike projections of the tongue mucosa called **papillae** (pah-pil′e) **(Figure 16.1a)**. Taste buds occur within the epithelium that covers the papillae. In the small **fungiform papillae** scattered over the entire



(a) Taste buds associated with fungiform and vallate papillae

(b) Enlarged section of a vallate papilla

(c) Enlarged view of a taste bud (micrograph, 160×)

FIGURE 16.1 Taste buds on the tongue.

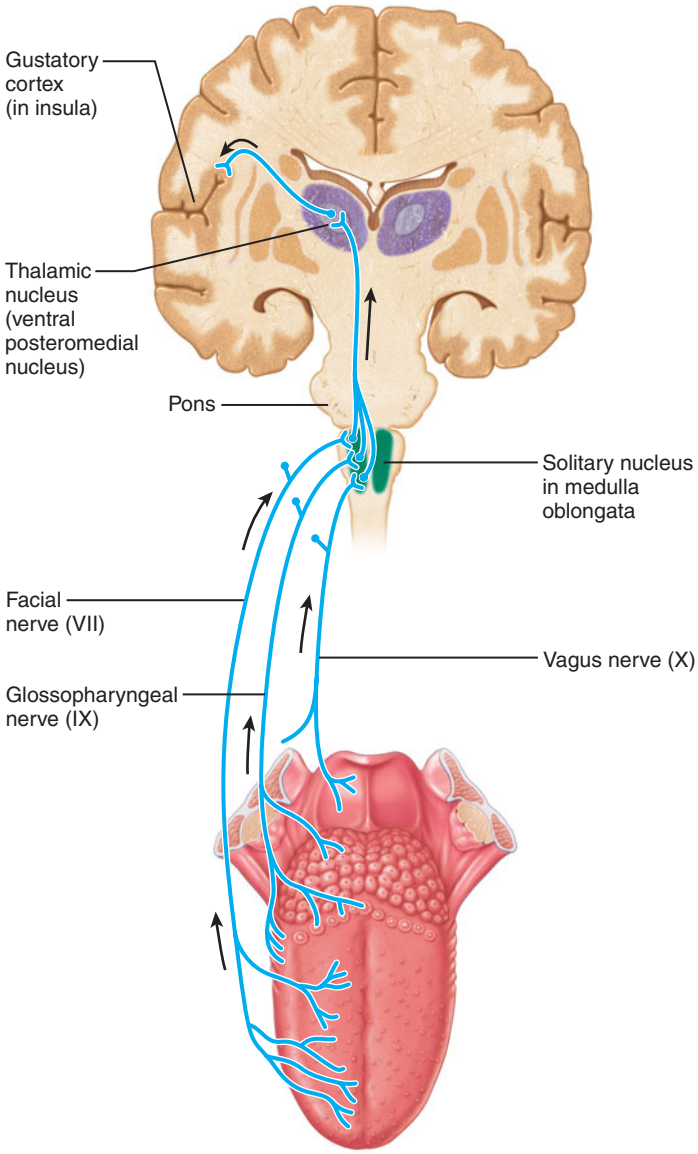


FIGURE 16.2 The gustatory pathway. Taste sensations are carried from the taste buds to the gustatory area of the cerebral cortex.

surface of the tongue, the taste buds are on the apical surface. In the large **vallate papillae** arranged in an inverted V near the back of the tongue, the taste buds are in the side walls (Figure 16.1b).

Each taste bud is a globular collection of 50–100 epithelial cells that resemble a bud on a tree or a closed tulip (Figure 16.1c). Each contains two major cell types: **gustatory epithelial cells** and **basal epithelial cells.** Long microvilli called **gustatory hairs** project from the gustatory epithelial cells and extend through a **taste pore** to the surface of the epithelium. There, these microvilli are bathed in saliva containing the dissolved molecules that stimulate taste. Such molecules bind to the plasma membrane of the microvilli, inducing the gustatory epithelial cells to generate impulses in the sensory nerve fibers that innervate them.

The cells in taste buds are replenished every 7–10 days by the division of the basal epithelial cells, replacing the gustatory epithelial cells that are scraped and burned off during eating. If an entire taste bud is destroyed, a new one will form after its nerve ending grows back into the regenerating epithelium.

Taste Sensations and the Gustatory Pathway

Taste sensations can be described in terms of five basic qualities: sweet, sour, salty, bitter, and umami. Umami (Japanese for "deliciousness") was recognized as a distinct fifth basic taste in the 1980s. It is elicited by a substance called glutamate that is found naturally in meat, aged cheeses, and tomatoes. Although maps that assign specific taste sensations to specific areas of the tongue are common, researchers have long known that these mapped areas are dubious and that all modalities of taste can be elicited from all areas that contain taste buds.

Taste information reaches the brain stem and cerebral cortex through the **gustatory pathway** (Figure 16.2). Sensory fibers carrying taste information from the tongue occur primarily in two cranial nerves: The *facial nerve* (VII) transmits impulses from taste receptors in the anterior two-thirds of the tongue, whereas the *glossopharyngeal nerve* (IX) carries sensations from the tongue's posterior third, as well as from the few buds in the pharynx. Additional taste impulses from the few taste buds on the epiglottis and lower pharynx are conducted by the *vagus nerve* (X). All the sensory neurons that carry taste information synapse in a nucleus in the medulla called the *solitary nucleus.* From there, impulses are transmitted to the thalamus and ultimately to the gustatory area of the cerebral cortex in the insula lobe.

Smell (Olfaction)

The receptors for smell lie in a patch of epithelium on the roof of the nasal cavity (Figure 16.3a). Specifically, the receptors are a part of the **olfactory epithelium** that covers the superior nasal concha and the superior part of the nasal septum and is bathed by swirling air that has been inhaled into the nasal cavity. Sniffing draws more air across the olfactory epithelium and thus intensifies the sense of smell.

The olfactory epithelium is a pseudostratified columnar epithelium (Figure 16.3b) that contains millions of bipolar neurons called **olfactory sensory neurons.** These are surrounded by columnar **supporting epithelial cells.** At the base of the epithelium lie short **basal epithelial cells,** undifferentiated neuroepithelial cells that continually form new olfactory sensory neurons. Thus, olfactory sensory neurons are among the few neurons in the body that undergo replacement throughout adult life.

The cell bodies of olfactory sensory neurons are located in the olfactory epithelium. Each receptor cell has an apical dendrite that projects to the epithelial surface and ends in a knob from which long **olfactory cilia** or "hairs" radiate. At the surface these cilia act as the receptive structures for smell by binding odor molecules to receptor proteins located in the plasma membrane of the cilia. The surface of the epithelium is also coated with a layer of mucus secreted by the nearby supporting cells and by olfactory glands in the underlying

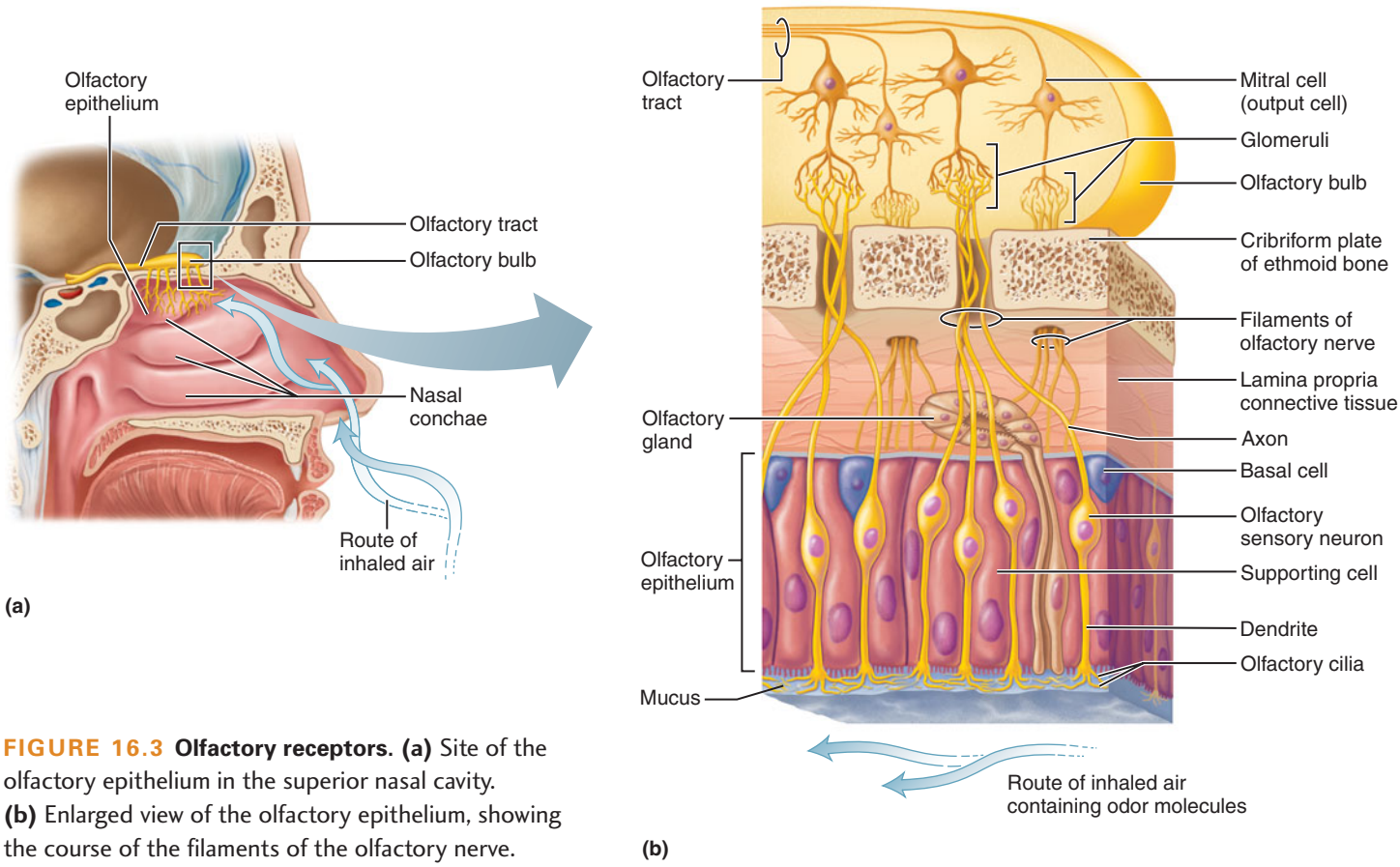


FIGURE 16.3 Olfactory receptors. (a) Site of the olfactory epithelium in the superior nasal cavity. **(b)** Enlarged view of the olfactory epithelium, showing the course of the filaments of the olfactory nerve.

connective tissue (lamina propria). This mucus, which captures and dissolves odor molecules from the air, is renewed continuously, flushing away old odor molecules so that new odors always have access to the olfactory cilia. Unlike other cilia in the body, olfactory cilia are largely immotile.

Each olfactory sensory neuron has an axon that enters the connective tissue of the lamina propria (Figure 16.3b). There, axons gather into nerve bundles called **filaments of the olfactory nerve** (cranial nerve I), which penetrate the cribriform plate of the ethmoid bone and enter the overlying **olfactory bulb** of the forebrain. In this bulb, the olfactory nerve axons branch profusely and synapse with neurons called **mitral cells** (mi′tral; "cap-shaped") in complex synaptic clusters called **glomeruli** (glo-mer′u-li; "balls of yarn"). The mitral cells then relay the olfactory information to other parts of the brain.

Upon receiving stimuli at synapses with olfactory sensory neurons, mitral cells transmit the impulses along the olfactory tract to (1) the limbic region, where smells elicit emotions, and (2) the piriform lobe of the cerebral cortex, represented by the lavender area around the uncus in Figure 13.18b (p. 397). The piriform lobe is thought to process olfactory information into a conscious perception of odor; it also sends this information through a thalamic relay to the orbitofrontal cortex (also seen in Figure 13.18b), where the smells are analyzed and compared to other smells.

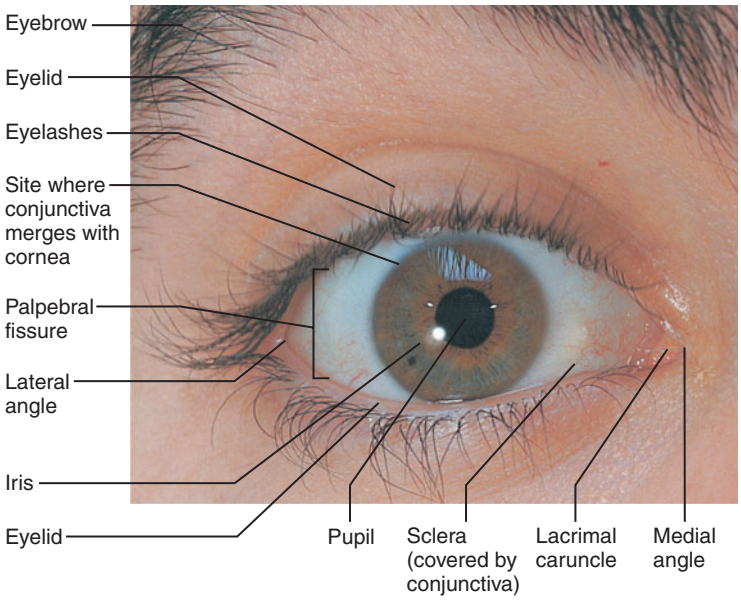
Disorders of the Chemical Senses

Disorders of both taste and olfaction exist, but olfactory disorders are more common. Absence of the sense of smell, called **anosmia** (an-oz′me-ah; "without smell"), typically results from blows to the head that tear the olfactory nerves, from colds or allergies that produce excessive mucus in the nasal cavity, or from physical blockage of the nasal cavity (by polyps, for example). Surprisingly, the cause in one-third of all cases of loss of chemical senses is zinc deficiency, and a prescribed dietary zinc supplement effects a rapid cure.

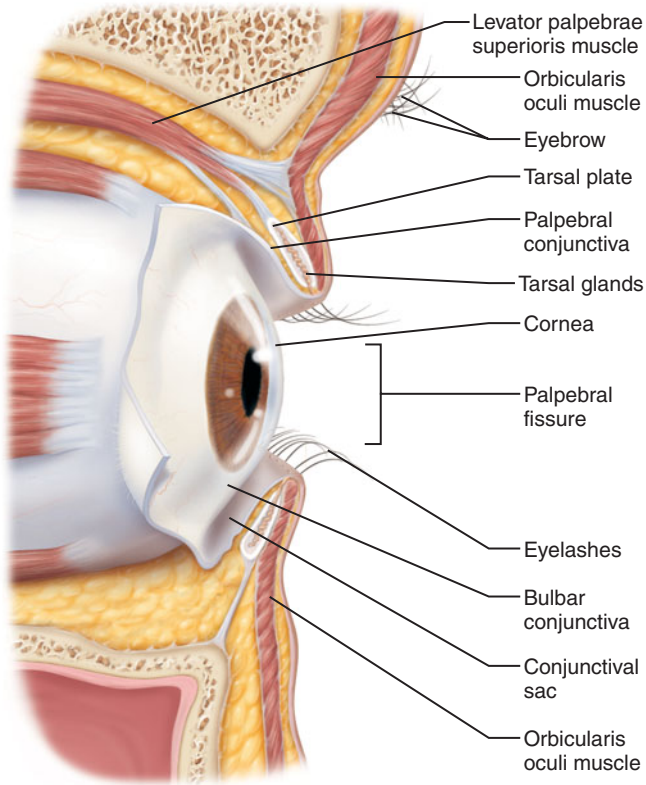
Brain disorders can distort the sense of smell. Some people have **uncinate** (un′sĭ-nāt) **fits,** olfactory hallucinations in which they perceive some imaginary odor, such as that of gasoline or rotting meat. Uncinate fits are so called because the primary olfactory cortex is in the uncinate region, or uncus, of the cerebrum (see p. 399). These fits may result from irritation of the olfactory pathway by brain surgery or head trauma. Olfactory auras—smells imagined by some epileptics just before they go into a seizure—are brief uncinate fits.

Embryonic Development of the Chemical Senses

The development of the olfactory epithelium and taste buds is straightforward. Olfactory epithelium derives from paired olfactory placodes, which are platelike thickenings of the



(a) Surface anatomy of the right eye



(b) Lateral view; some structures shown in sagittal section

FIGURE 16.4 Accessory structures of the eye.

surface ectoderm on the embryonic snout region (see Figure 22.20, p. 660). Taste buds develop, upon stimulation by gustatory nerves, from the epithelium lining the embryonic mouth and pharynx, an epithelium that is derived from ectoderm and endoderm.

check your understanding

1. What type of tissue forms the receptor cells for taste?
2. What type of cell are the olfactory receptors? When these cells are damaged, are they replaced?

For answers, see Appendix B.

THE EYE AND VISION

Vision is the dominant sense in humans: Approximately 70% of the sensory receptors in the body are in the eyes, and 40% of the cerebral cortex is involved in processing visual information (see p. 398). The visual receptor cells (photoreceptors) sense and encode the patterns of light that enter the eye; subsequently, the brain invests these signals with meaning, fashioning visual images of the world.

The visual organ is the **eye,** a spherical structure with a diameter of about 2.5 cm (1 inch). Only the anterior one-sixth of the eye's surface is visible; the rest of the eye lies in the cone-shaped bony *orbit,* where it is surrounded by a protective cushion of fat. Behind the eye, the posterior half of the orbit contains the optic nerve, the arteries and veins to the eye, and the extrinsic eye muscles.

Accessory Structures of the Eye

➤ Describe the anatomy and function of the accessory structures of the eye.

Eyebrows

The **eyebrows** consist of coarse hairs in the skin on the superciliary arches (brow ridges of the skull). They shade the eyes from sunlight and prevent perspiration running down the forehead from reaching the eyes.

Eyelids

Anteriorly, the eyes are protected by the mobile **eyelids,** or **palpebrae** (pal′pĕ-bre). The upper and lower lids are separated by the **palpebral fissure** (eye slit) and meet each other at the medial and lateral angles, or eye corners **(Figure 16.4a)**. The medial angle contains a reddish elevation called the **lacrimal caruncle** (kar′ung-k'l; "a bit of flesh"). Glands here produce the gritty "eye sand" reputedly left by the legendary Sandman at night. In most Asian people, a vertical fold of skin called the *epicanthic fold* occurs on both sides of the nose and sometimes covers the medial angle.

The eyelids are thin, skin-covered folds supported internally by connective tissue structures called **tarsal plates** (Figure 16.4b). These stiff plates give the eyelids their curved shape and serve as attachment sites for the eye-closing muscle, the *orbicularis oculi* (see p. 278).

The **levator palpebrae superioris** ("lifter of the upper eyelid") is the skeletal muscle that voluntarily opens the eye. It runs anteriorly from the posterior roof of the orbit, enters

the upper eyelid, and inserts on the tarsal plate. The inferior part of the aponeurosis of this muscle contains fibers of smooth muscle, called the *superior tarsal muscle,* an involuntary muscle that prevents the upper eyelid from drooping (discussed previously on p. 471).

Projecting from the free margin of each eyelid are the **eyelashes.** Because the follicles of these hairs are richly innervated by nerve endings, even slight pressure on the eyelashes will trigger reflexive blinking.

Several types of glands occur in the eyelids. **Tarsal glands** are modified sebaceous glands embedded in the tarsal plates (Figure 16.4b). About 25 of these vertical glands line up side by side in the upper eyelid; fewer than this line up in the lower lid. The tarsal gland ducts open along the edge of the eyelids. They release an oil that lubricates the surface of the eye. Other glands are associated with the hair follicles of the eyelashes: These *ciliary glands* (*cilium* = eyelash) include typical sebaceous glands whose ducts open into the hair follicles, and modified sweat glands that lie between the follicles. Infection of a tarsal gland results in an unsightly cyst called a **chalazion** (kah-la′ze-on; "swelling"), whereas infection of the ciliary glands is called a **sty.**

Conjunctiva

The **conjunctiva** (con″junk-ti′vah; "joined together") is a transparent mucous membrane that covers the inner surfaces of the eyelids as the **palpebral conjunctiva** and folds back over the anterior surface of the eye as the **bulbar conjunctiva** (Figure 16.4b). The bulbar conjunctiva, which covers the white of the eye but not the cornea (the transparent tissue over the iris and pupil; Figure 16.4a), is a very thin membrane, and blood vessels are clearly visible beneath it. (These vessels are responsible for "bloodshot" eyes.) When an eye is closed, the slitlike space that forms between the eye surface and the eyelids is the **conjunctival sac,** which is where a contact lens would lie.

Microscopically, the conjunctiva consists of a stratified columnar epithelium underlain by a thin lamina propria of loose connective tissue. Its epithelium contains scattered goblet cells that secrete a lubricating mucus that prevents the eyes from drying. A deficiency of vitamin A, a vitamin required for maintaining epithelia throughout the body, prevents the conjunctiva from secreting mucus. As a result, the conjunctiva dries up and becomes scaly, impairing vision.

Inflammation of the conjunctiva, called **conjunctivitis,** is a relatively common condition. Conjunctivitis irritates the eyes and makes them red. A highly contagious form of conjunctivitis caused by bacteria or viruses is called **pinkeye.**

Lacrimal Apparatus

The **lacrimal apparatus** (lak′rĭ-mal; "tear"), which keeps the surface of the eye moist with lacrimal fluid (tears), consists of a gland and ducts that drain the lacrimal fluid into the nasal cavity **(Figure 16.5)**. The **lacrimal gland,** lying in the orbit superolateral to the eye, produces lacrimal fluid, which enters the superior part of the conjunctival sac through several small excretory ducts. Blinking the eye spreads this fluid inferiorly across the eyeball to the medial angle. At the medial angle,

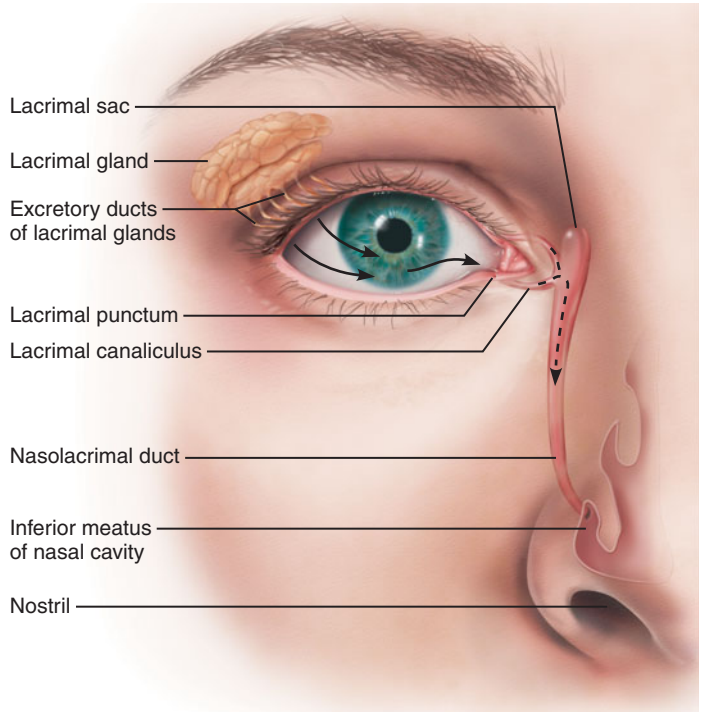


FIGURE 16.5 The lacrimal apparatus. Arrows indicate the direction of flow of lacrimal fluid (tears) from the lacrimal gland to the nasal cavity.

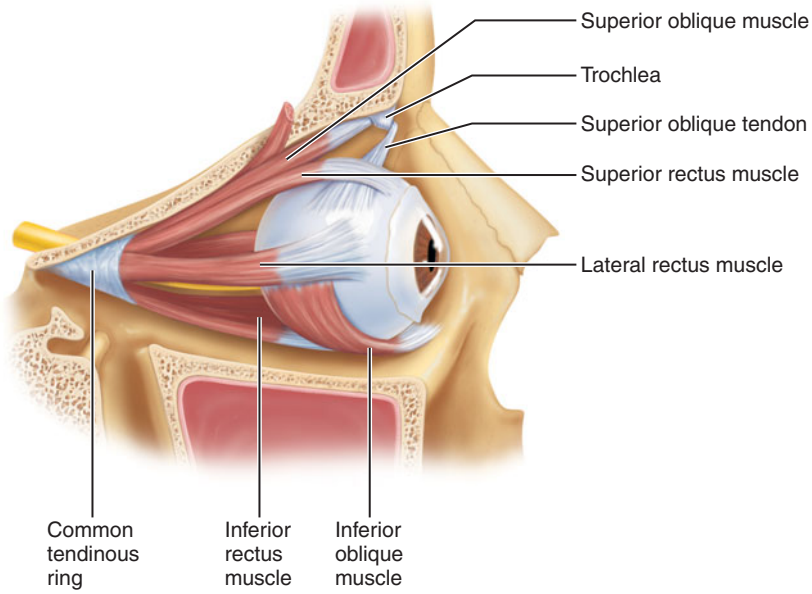
each lid contains a tiny opening called the **lacrimal punctum** ("puncture") which empties into a small tube, the **lacrimal canaliculus** ("small canal"). From these canals, the fluid drains into the **lacrimal sac** in the medial orbital wall. Finally, the fluid enters the **nasolacrimal duct,** which empties into the nasal cavity at the inferior nasal meatus. Because lacrimal fluid ultimately empties into the nasal cavity, people sniffle when they cry. In people with colds, the viral infection in the nasal passages can spread into the nasolacrimal duct and lacrimal sac, producing inflammation that causes these passages to swell shut. The resulting blockage prevents the drainage of lacrimal fluid from the eye surface; the fluid accumulates, and the eyes water.

Lacrimal fluid contains mucus, antibodies, and **lysozyme,** an enzyme that destroys bacteria. When the eye surface is irritated by dust or fumes (from an onion, for example), lacrimal secretion increases to wash away the irritant.

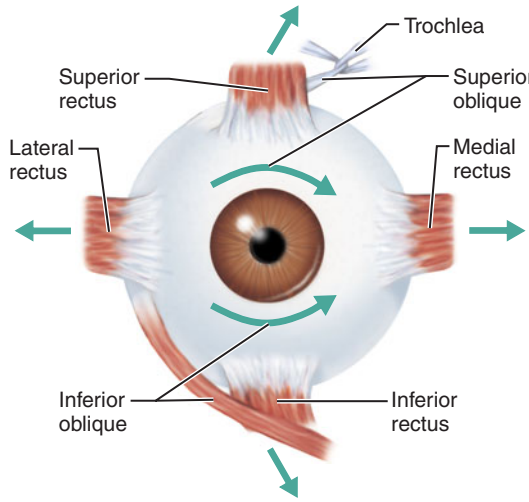
Extrinsic Eye Muscles

Six straplike **extrinsic** (outer) **eye muscles (Figure 16.6)**, which originate from the walls of the orbit and insert onto the outer surface of the eyeball, control the movement of each eye and hold the eyes in the orbits.

Four of the extrinsic eye muscles are *rectus* muscles (*rectus* = straight). These originate from the **common tendinous ring,** or **anular ring** (Figure 16.6a), at the posterior point of the orbit. From there, they run straight to their insertions on the anterior half of the eyeball. The **lateral rectus muscle** turns the eye laterally (outward), whereas the **medial**



(a) Lateral view of the right eye



(b) Anterior view of the right eye

Muscle	Action	Controlling cranial nerve
Lateral rectus	Moves eye laterally	VI (abducens)
Medial rectus	Moves eye medially	III (oculomotor)
Superior rectus	Elevates eye and turns it medially	III (oculomotor)
Inferior rectus	Depresses eye and turns it medially	III (oculomotor)
Inferior oblique	Elevates eye and turns it laterally	III (oculomotor)
Superior oblique	Depresses eye and turns it laterally	IV (trochlear)

(c) Summary of muscle actions and innervating cranial nerves

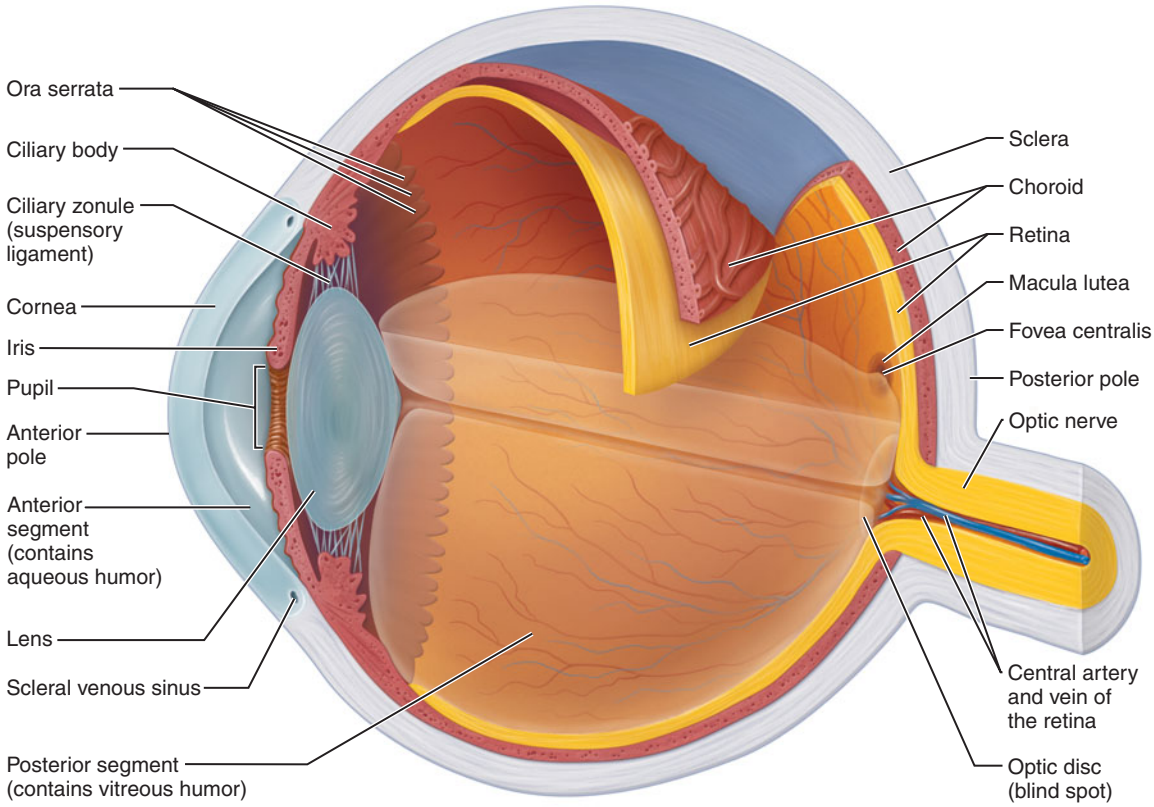
FIGURE 16.6 Extrinsic eye muscles. In (b), arrows indicate eye movement resulting from contraction of each muscle.

rectus muscle turns it medially (inward). The **superior** and **inferior rectus muscles** turn the eye superiorly and medially, and inferiorly and medially, respectively. It is easy to deduce the actions of the rectus muscles from their names and locations (Figure 16.6b and c).

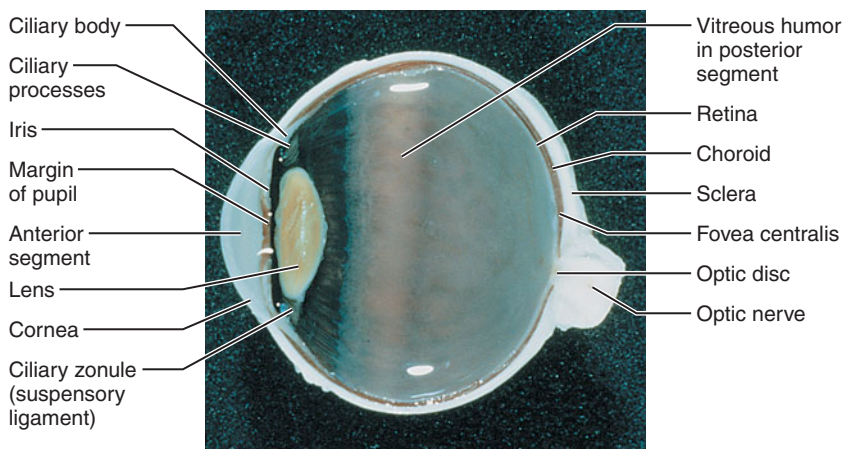
The actions of the two *oblique* muscles are not so easily deduced, however, because they take indirect paths through the orbit. The **superior oblique muscle** (Figure 16.6a) originates posteriorly near the common tendinous ring, runs anteriorly along the medial orbit wall, and then loops through a ligamentous sling, the **trochlea** ("pulley"), which is suspended from the frontal bone in the anteromedial part of the orbit roof. From there, its tendon runs posteriorly and inserts on the eye's posterolateral surface. Because its tendon approaches from an anterior and medial direction, the superior oblique depresses the eye and turns it laterally (down and out) (Figure 16.6b and c). Because the **inferior oblique muscle** originates on the anteromedial part of the orbit floor and angles back to insert on the posterolateral part of the eye, the inferior oblique elevates the eye and turns it somewhat laterally (up and out).

The lateral pull of the oblique muscles counteracts the medial pull of the superior and inferior recti to produce strict elevation and depression of the eye. Also, the superior and inferior recti can neither elevate nor depress an eye that has been turned far medially to look inward, so the two oblique muscles must do that.

STRABISMUS The extrinsic muscles of both eyes are closely controlled by centers in the midbrain so that the eyes move together in unison when we look at objects. If this coordination is disrupted, double vision results, because the two eyes do not look at the same point in the visual field. This misalignment of the eyes is called **strabismus** (strah-biz'mus), meaning "cross-eyed" or "squint-eyed." In strabismus, the affected eye is turned either medially or laterally with respect to the normal eye. Strabismus results from weakness or paralysis of extrinsic eye muscles caused by damage to the oculomotor nerve or other problems. Immediate surgical correction is recommended because over time the brain comes to disregard the image from the affected eye; as a result, the entire visual pathway from that eye degenerates, making the eye functionally blind.



(a) Diagrammatic view. The vitreous humor is illustrated only in the bottom part of the eyeball.



(b) Photograph of the human eye.

FIGURE 16.7 Internal structure of the eye (sagittal section).

check your understanding

3. What is the conjunctiva, and where is it located?
4. What muscle is not functioning in a person whose eye turns medially? What nerve innervates this muscle?

For answers, see Appendix B.

Anatomy of the Eyeball

- Describe the structure and function of the layers of the eye, the lens, and the humors of the eye.
- Explain the structure of the retina and the photoreceptors.

The eye is a complex organ whose many components not only protect and support the delicate photoreceptor cells but also gather, focus, and process light into precise images. Figure 16.7 summarizes the anatomy of the eye. Because the eyeball is shaped roughly like a globe, it is said to have poles. Its most anterior point is the **anterior pole,** and its most posterior point is the **posterior pole.** Its external wall consists of three *layers,* and its internal cavity contains fluids called *humors.* The *lens,* a structure that helps to focus light, is supported vertically within the internal cavity, dividing it into anterior and posterior segments. The anterior segment is filled with the liquid *aqueous humor,* whereas the posterior segment is filled with the jellylike *vitreous humor.*

Three layers form the external wall of the eye: the fibrous layer, the vascular layer, and the sensory layer (retina).

The Fibrous Layer

The **fibrous layer** is the most external layer. It consists of dense connective tissue arranged into two different regions: sclera and cornea (Figure 16.7). The opaque white, tough **sclera** (skle′rah; "hard") forms the posterior five-sixths of the fibrous layer. Seen anteriorly as the "white of the eye," the sclera protects the eyeball and provides shape and a sturdy anchoring site for the extrinsic eye muscles. The sclera corresponds to the dura mater that covers the brain.

The anterior sixth of the fibrous layer is the transparent **cornea,** through which light enters the eye. This round window bulges anteriorly from its junction with the sclera. The cornea consists of a thick layer of dense connective tissue sandwiched between a superficial corneal epithelium and a deep corneal endothelium. At the junction of the cornea and sclera, between the corneal epithelium and the conjunctiva, are epithelial stem cells that continually renew the corneal epithelium. The cornea's connective tissue layer contains hundreds of sheets of collagen fibers stacked like the pages in a book; the transparency of the cornea is due to this regular alignment of collagen fibers. The cornea not only lets light into the eye but also forms part of the light-bending apparatus of the eye (see p. 495).

The cornea is avascular—it receives oxygen from the air in front of it, and oxygen and nutrients from the aqueous humor that lies posterior to it.

The cornea is richly supplied with nerve endings, most of which are pain receptors. (This is why some people can never adjust to wearing contact lenses.) Touching the cornea causes reflexive blinking and an increased secretion of tears. Even with these protective responses, the cornea is vulnerable to damage by dust, slivers, and other objects. Fortunately, its capacity for regeneration and healing is extraordinary.

CORNEAL TRANSPLANTS "Eye banks" are institutions that receive and store corneas for use in the surgical replacement of severely damaged corneas, a procedure called a **corneal transplant.** The cornea is one of the few structures in the body that can be transplanted from one person to another with only minimal risk of rejection. Because it has no blood vessels, it is beyond the reach of the body's immune system. In the 10% of transplanted corneas that are rejected, the damage to the recipient's original cornea was so extensive that it destroyed the corneoscleral junction. Without that region, the corneal epithelium cannot regenerate, so *conjunctival* epithelium overgrows the new cornea, in the process attracting new blood vessels carrying the immune cells that cause rejection. This problem can be alleviated by grafting corneal epithelium from the patient's healthy eye onto the newly transplanted cornea.

The Vascular Layer

The **vascular layer,** the middle coat of the eyeball, has three parts: the *choroid,* the *ciliary body,* and the *iris* (Figure 16.7).

The **choroid** (ko′roid; "membrane") is a highly vascular, darkly pigmented membrane that forms the posterior five-sixths of the vascular layer. Its many blood vessels nourish the other layers of the eye. The brown color of the choroid is produced by melanocytes, whose pigment, melanin, helps absorb light, thereby preventing light from scattering within the eye and creating visual confusion. The choroid layer of the eye corresponds to the arachnoid and pia mater around the brain.

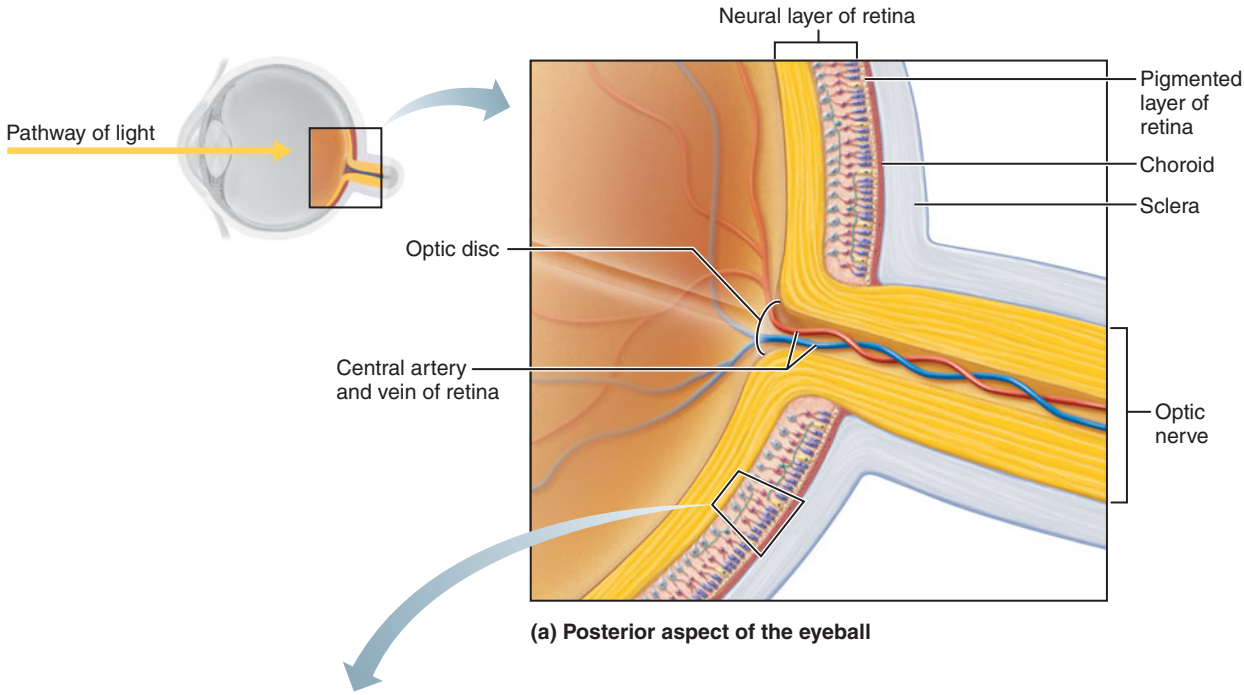
Anteriorly, the choroid is continuous with the **ciliary body,** a thickened ring of tissue that encircles the lens (Figure 16.7). The ciliary body consists chiefly of smooth muscle called the **ciliary muscle,** which acts to focus the lens. Nearest the lens, the posterior surface of the ciliary body is thrown into radiating folds called **ciliary processes.** The halo of fine fibrils that extends from these processes to attach around the entire circumference of the lens is called the **ciliary zonule** (Figure 16.7).

The **iris** ("rainbow") is the visible, colored part of the eye. It lies between the cornea and lens, and its base attaches to the ciliary body (Figure 16.7). Its round central opening, the **pupil,** allows light to enter the eye. The iris contains both circularly arranged and radiating smooth muscle fibers, the *sphincter* and *dilator pupillae* muscles, that act to vary the size of the pupil. In bright light and for close vision, the sphincter pupillae contracts to constrict the pupil. In dim light and for distant vision, the dilator pupillae contracts to widen the pupil, allowing more light to enter the eye. Constriction and dilation of the pupil are controlled by parasympathetic and sympathetic fibers, respectively, as described in Chapter 15. The constriction of the pupils that occurs when a bright light is flashed in the eye is a protective response called the **pupillary light reflex.**

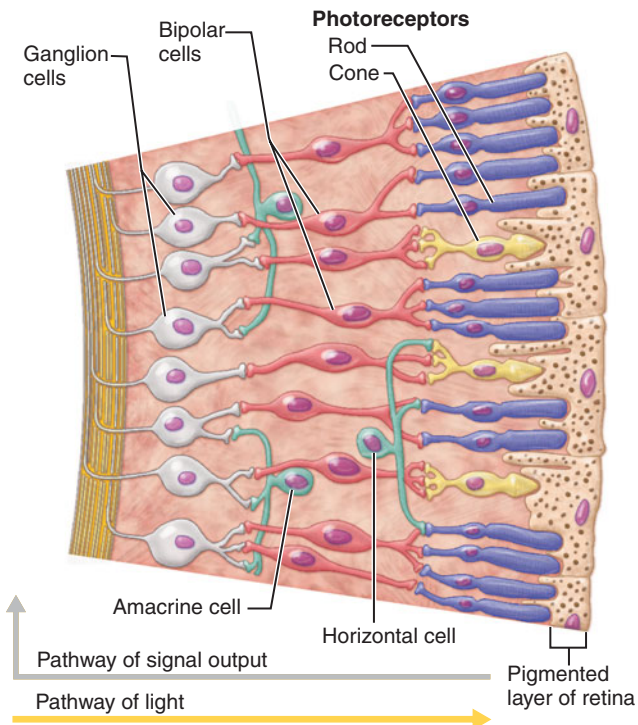
Although irises come in many colors, they contain only brown pigment. Variation in eye color reflects the amount of pigmentation in the iris. All people except albinos have a layer of pigmented cells on the posterior surface of the iris. Brown-eyed people have many pigment cells on the anterior surface of the iris as well. Blue-eyed people, by contrast, do not have pigment on the anterior surface. Blue eyes result from the reflection of light off the pigmented posterior surface. Hazel-eyed people have some pigment in the anterior portion of the iris.

The Inner Layer

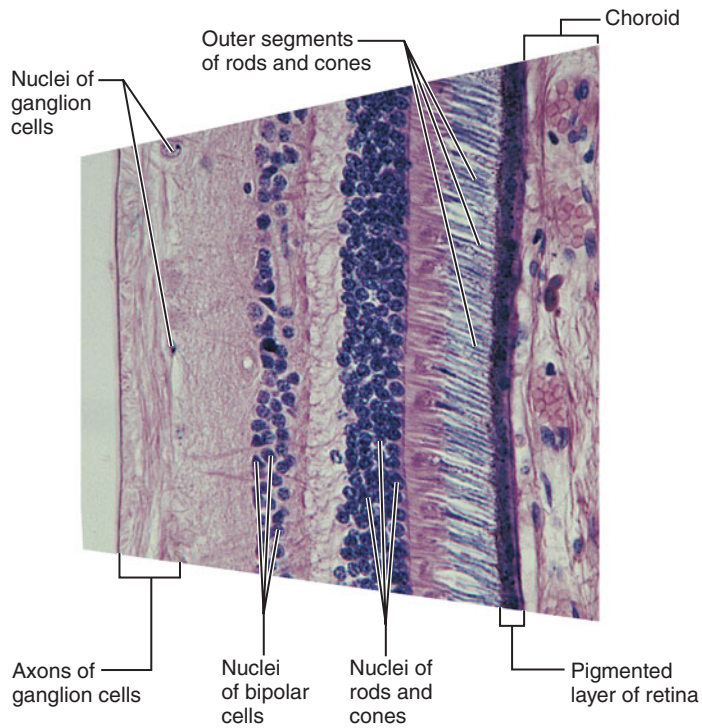
The **inner layer** contains the *retina* and the *optic nerve.* The **retina** consists of two layers: a thin pigmented layer and a far thicker neural layer **(Figure 16.8a)**. The outer **pigmented layer,** which lies against the choroid, is a single layer of flat-to-columnar melanocytes. Like the choroid, it functions to absorb light and prevent it from scattering within the eye. The much thicker inner **neural layer** is a sheet of nervous tissue that contains the light-sensitive photoreceptor cells. The neural and pigmented layers of the retina are held together by



(a) Posterior aspect of the eyeball



(b) Cells of the neural layer of the retina



(c) Photomicrograph of retina

FIGURE 16.8 Microscopic anatomy of the retina. (a) The axons of the ganglion cells form the optic nerve, which leaves the back of the eye at the optic disc. **(b)** Light (indicated by the yellow arrow) passes through the retina to the photoreceptor cells (rods and cones). Information (output signals) flows in the opposite direction via bipolar and ganglion cells. **(c)** Photomicrograph (145×).

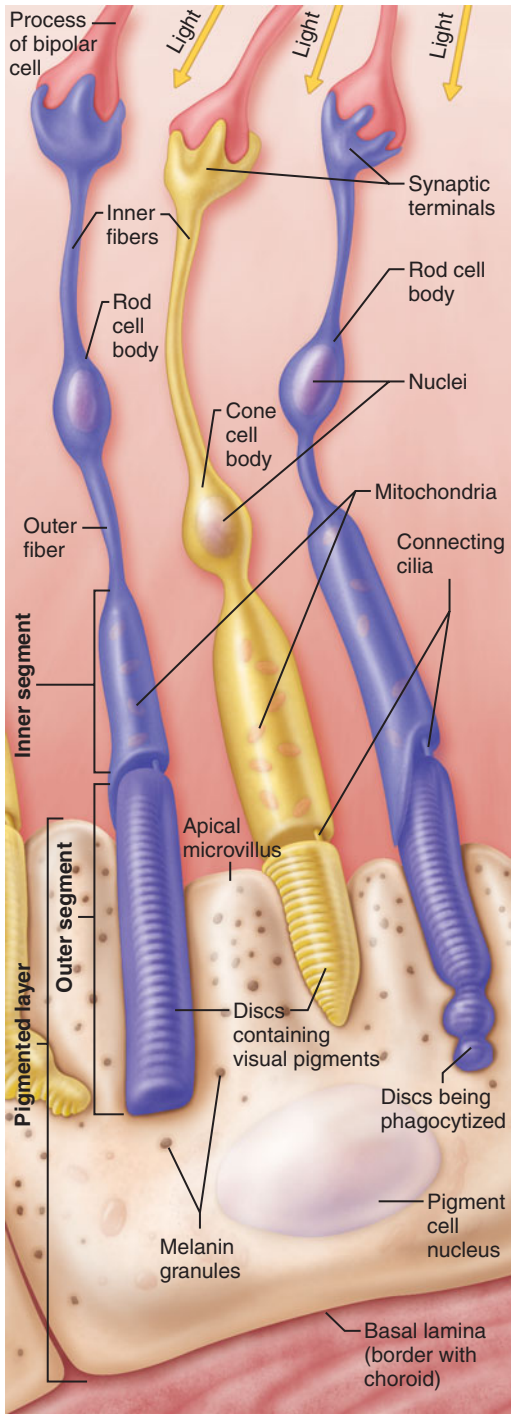


FIGURE 16.9 Photoreceptors of the retina. The outer segments of the rods and cones are embedded in the pigmented layer of the retina.

a thin film of extracellular matrix, but they are not tightly fused. Only the neural layer plays a direct role in vision. The pigmented layer supports the photoreceptive cells by removing damaged portions of those cells, maintaining the proper ionic concentration in the fluid surrounding them, recycling the vitamin A derivative used for light detection, and transporting nutrients from the choroid vessels to the photoreceptor cells.

The neural layer contains three main types of neurons. From external to internal, these are the **photoreceptor cells, bipolar cells,** and **ganglion cells** (Figure 16.8b and c). When stimulated by light, the photoreceptor neurons signal the bipolar cells, which then signal the ganglion cells to generate nerve impulses potentials. Axons from the ganglion cells run along the internal surface of the retina and converge posteriorly to form the **optic nerve,** which runs from the eye to the brain (Figure 16.8a).

The retina also contains interneurons—including amacrine cells and horizontal cells (Figure 16.8b)—that process and modify visual information before it is sent to higher brain centers for further processing.

Photoreceptors The photoreceptor cells are of two types: *rod cells* and *cone cells.* The more numerous **rod cells** are more sensitive to light and permit vision in dim light. Because rod cells provide neither sharp images nor color vision, things look gray and fuzzy when viewed in dim light. **Cone cells,** by contrast, operate best in bright light and enable high-acuity color vision. Three subtypes of cone cells are sensitive to blue, red, and green light, respectively.

Photoreceptors are considered neurons, but they also resemble tall epithelial cells turned upside down, with their "tips" immersed in the pigmented layer **(Figure 16.9)**. Both rod cells and cone cells have an *outer segment* joined to an *inner segment* by a connecting cilium. In each rod cell, the inner and outer segments together form a rod-shaped structure, which connects to the nucleus-containing *cell body* by an *outer fiber.* In each cone cell, the inner and outer segments form a cone-shaped structure, which joins to the cell body directly. In both cell types, the cell body is continuous with an *inner fiber* that synapses with the bipolar cell.

The outer segments are the receptor regions of the rod and cone cells. Each outer segment is a modified cilium whose plasma membrane has folded inward to form hundreds of membrane-covered discs. Light-absorbing *visual pigments* are present within the vast membrane of these discs. The extensive folding of the plasma membrane into discs greatly magnifies the surface area available for trapping light. When light particles hit the visual pigment, the pigment is modified, and a nerve impulse is generated. The stimulated photoreceptor cells signal the bipolar neurons with which they synapse. This reaction initiates the flow of visual information to the brain.

The photoreceptors are highly vulnerable to damage by intense light or heat. These cells cannot regenerate if destroyed, but they continually renew and replace their outer segments through the addition of new discs. In this normal recycling process, as new discs are added to one end of the stack, old discs are removed at the other end by retinal pigment cells phagocytizing the tips of the rods and cones (Figure 16.9).

Regional Specializations of the Retina In certain regions of the eye, the retina differs from its "typical" structure just described.

In the anterior part of the eye, the neural layer ends at the posterior margin of the ciliary body. This junction is called

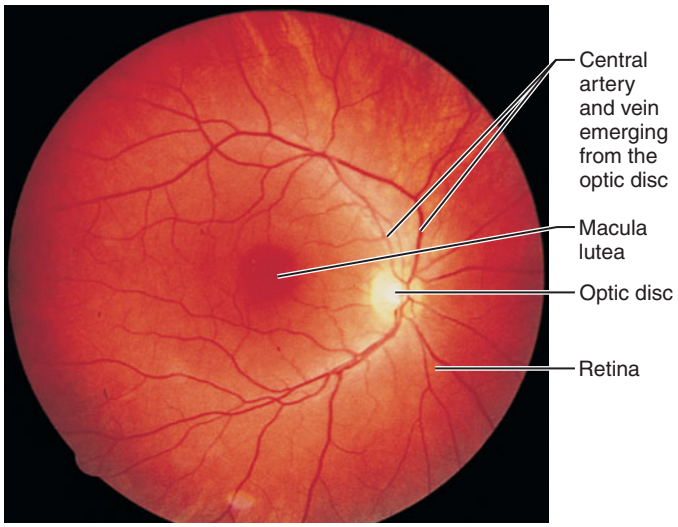


FIGURE 16.10 The posterior wall of the right eye as seen with an ophthalmoscope.

the **ora serrata** (o′rah se-rah′tah; "sawtoothed mouth") (see Figure 16.7a). The pigmented layer extends anteriorly beyond the ora serrata to cover the ciliary body and to form the pigmented layer of the posterior iris.

The posterior part of the eye contains several special areas of the retina (Figure 16.7). Lying precisely at the eye's posterior pole is the **macula lutea** (mak′u-lah lu′te-ah; "yellow spot"). At the center of the macula lutea is a tiny pit called the **fovea centralis** (fo′ve-ah sen-trah′lis; "central pit"). The fovea contains only cones and provides maximal visual acuity. Because the fovea lies directly in the anterior-posterior axis of the eye, things are most clearly seen when we look straight at them. The macula contains mostly cones and the density of cones declines with increasing distance from the macula. For this reason, peripheral vision is not as sharp as central vision. A few millimeters medial to the fovea is the **optic disc** (Figure 16.7), a circular elevation where the axons of ganglion cells converge to exit the eye as the optic nerve. The optic disc is called the *blind spot* because it lacks photoreceptors, and light focused on it cannot be seen.

Blood Supply of the Retina The retina receives its blood from two different sources. The outer third of the retina, containing the photoreceptors, is supplied by capillaries in the choroid, whereas its inner two-thirds is supplied by the **central artery** and **vein of the retina,** which enter and leave the eye by running through the center of the optic nerve (see Figure 16.7 and Figure 16.8a). These vessels radiate from the optic disc, giving rise to a rich network of tiny vessels that weave among the axons on the retina's inner face. This vascular network is clearly visible through an ophthalmoscope, a handheld instrument that shines light through the pupil and illuminates the retina **(Figure 16.10)**. Physicians observe the tiny retinal vessels for signs of hypertension, diabetes, and other diseases that damage the smallest blood vessels.

DETACHED RETINA The retina's pattern of vascularization contributes to a potentially blinding condition called **retinal detachment,** in which the loosely joined neural and pigmented layers of the retina separate ("detach") from one another. The detachment begins with a tear in the retina, which may result from a small hemorrhage, a blow to the eye, or age-related degeneration. The tear allows the jellylike vitreous humor from the eye's interior (see the next section) to seep between the two retinal layers. With the neural layer detached, the photoreceptors, now separated from their blood supply in the choroid, are kept alive temporarily by nutrients obtained from the inner retinal capillaries. However, because these capillaries are too distant to supply them permanently, the photoreceptors soon die. Early symptoms of retinal detachment include seeing flashing lights or spots that float across the field of vision, and having objects appear as if seen through a veil. If the detachment is diagnosed early, blindness may be prevented by reattaching the retina with laser surgery before permanent damage to the photoreceptors occurs.

Internal Chambers and Fluids

The lens and its halolike ciliary zonule divide the eye into posterior and anterior segments (Figure 16.7). The **posterior segment** is filled with the clear **vitreous humor** (*vitreus* = glassy), a jellylike substance that contains fine fibrils of collagen and a ground substance that binds tremendous amounts of water. Indeed, water constitutes over 98% of its volume. The functions of vitreous humor are to (1) transmit light, (2) support the posterior surface of the lens and hold the neural retina firmly against the pigmented layer, and (3) help maintain *intraocular pressure* (the normal pressure within the eye), thereby counteracting the pulling forces of the extrinsic eye muscles.

The **anterior segment** of the eye **(Figure 16.11)** is divided into an **anterior chamber** between the cornea and iris, and a **posterior chamber** between the iris and lens. The entire anterior segment is filled with **aqueous humor,** a clear fluid similar to blood plasma (*aqueous* = watery). Unlike the vitreous humor, which forms in the embryo and lasts a lifetime, aqueous humor is renewed continuously and is in constant motion. This process is illustrated in Figure 16.11. After being formed as a filtrate of the blood from capillaries in the ciliary processes (1), the aqueous humor enters the posterior chamber, flows through the pupil into the anterior chamber (2), and drains into a large vessel at the corneoscleral junction, the **scleral venous sinus,** which returns it to the blood (3). An equilibrium in the rates at which the aqueous humor forms and drains results in a constant intraocular pressure, which supports the eyeball internally. Furthermore, the aqueous humor supplies nutrients and oxygen to the avascular lens and cornea.

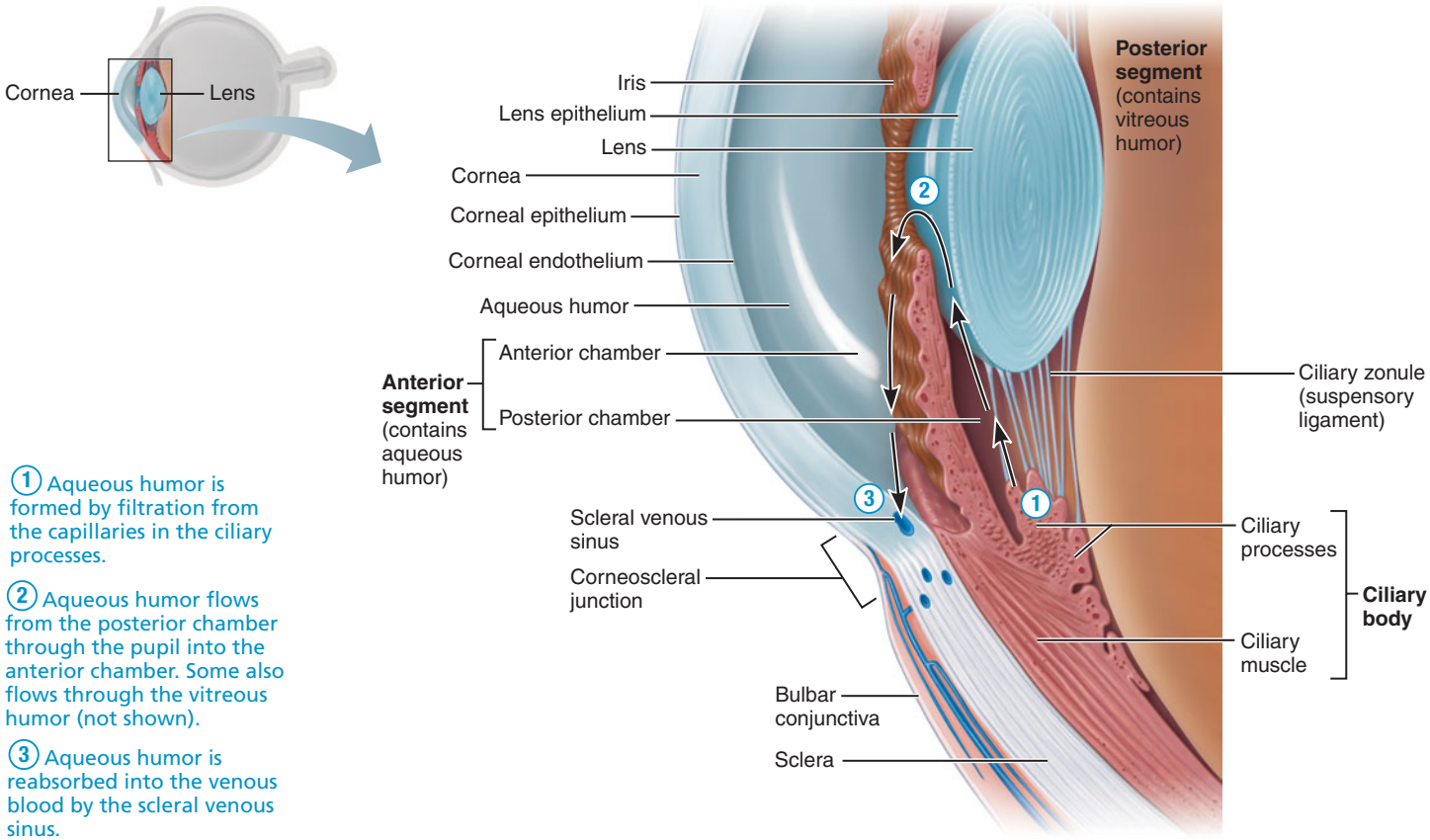


FIGURE 16.11 The anterior segment of the eye and circulation of aqueous humor.
The arrows indicate the circulation pathway.

GLAUCOMA When the aqueous humor drains more slowly than it forms, the result is **glaucoma** (glaw-ko'-mah), a disease in which intraocular pressure increases to dangerous levels, causing compression of the retina and the optic nerve. Glaucoma results from an obstruction of outflow, usually from a clogging of the permeable net through which aqueous humor drains into the scleral venous sinus. The resulting destruction of the optic nerve eventually causes blindness. Even though vision can be saved if the condition is detected early, glaucoma often steals sight so slowly and painlessly that most people do not realize they have a problem until the damage is done. Late signs include blurred vision, seeing halos around lights, and headaches. The examination for glaucoma involves testing for high intraocular pressure. A puff of air is directed at the cornea and the amount of deformation of the sclera is measured. A glaucoma exam should be done yearly after age 40, because glaucoma affects fully 2% of people over that age. Early glaucoma is treated with eye drops that increase drainage or decrease production of aqueous humor.

The Lens

The **lens** is a thick, transparent, biconvex disc that changes shape to allow precise focusing of light on the retina (Figure 16.11). It is enclosed in a thin elastic capsule and is held in place posterior to the iris by its ciliary zonule. Like the cornea, it lacks blood vessels, which would interfere with transparency.

The lens has two components: the lens epithelium and the lens fibers. The **lens epithelium,** confined to the anterior surface, consists of cuboidal cells. The subset of epithelial cells around the edge of the lens disc transforms continuously into the elongated **lens fibers** that form the bulk of the lens. These fibers, which are packed together like the layers in an onion, contain no nuclei and few organelles. They do, however, contain precisely folded proteins that make them transparent. New lens fibers are added continuously, so the lens enlarges throughout life. It becomes denser, more convex, and less elastic with age. As a result, its ability to focus light is gradually impaired.

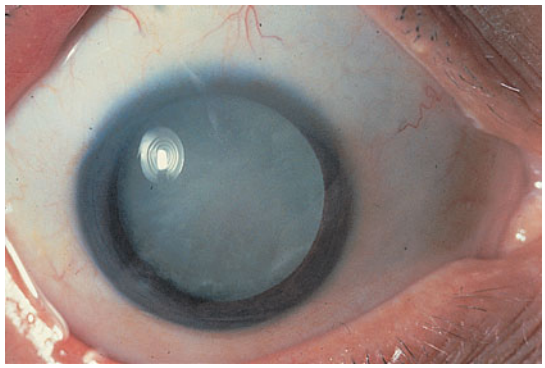


FIGURE 16.12 Photograph of a cataract. The *lens* is milky and opaque, not the cornea.

CATARACT A **cataract** (kat'ah-rakt; "waterfall") is a clouding of the lens (Figure 16.12) that causes the world to appear distorted, as if seen through frosted glass. Some cataracts are congenital, but most result from age-related changes in the lens. Recent evidence indicates that excessive exposure to sunlight, heavy smoking, and certain medications including oral steroids, long-term aspirin use, and tamoxifen (used to treat breast cancer), are linked to cataract formation. No matter the cause, cataracts seem to result from an inadequate delivery of nutrients to the deeper lens fibers. Fortunately, surgical removal of the damaged lens and its replacement by an artificial lens can save a cataract patient's sight.

check your understanding

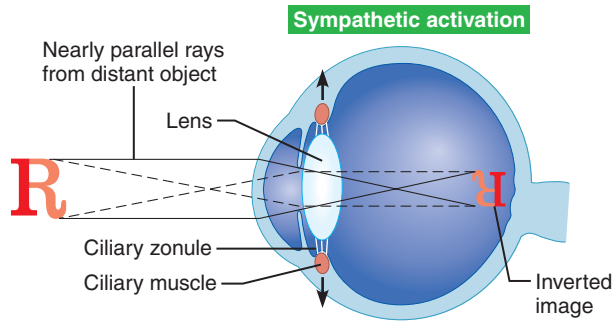
5. Differentiate the cornea from the choroid with reference to the location, structure, and function of each.
6. What portion of the visual field is lost in a person with degeneration of the macula lutea (macular degeneration)?
7. What is the aqueous humor, where is it located, and what are its functions?

For answers, see Appendix B

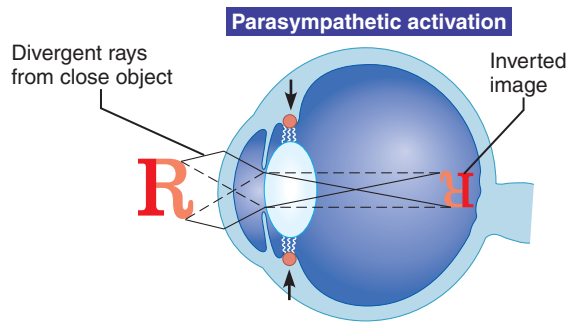
The Eye as an Optical Device

➤ Explain how light is focused for close vision.

From each point on an object, light rays radiate in every direction. Some of these light rays enter the eye of the viewer (Figure 16.13). Rays from a distant point are parallel to one another as they reach the eye, whereas rays from a nearby point diverge markedly as they enter the eye. If one is to see clearly, the eye must be able to bend all these light rays so that they converge on the retina at a single *focal point.* The light-bending parts of the eye, called **refractory media,** are



(a) Lens is flattened for distant vision. Sympathetic input relaxes the ciliary muscle, tightening the ciliary zonule, and flattening the lens.



(b) Lens bulges for close vision. Parasympathetic input contracts the ciliary muscle, loosening the ciliary zonule, allowing the lens to bulge.

FIGURE 16.13 Focusing for distant and close vision.

the cornea, the lens, and the humors. The cornea does most of the light bending, the lens does some of it, and the humors do a minimal amount.

Although the lens is not as powerful as the cornea in bending light, its curvature is adjustable. This adjustability allows the eye to focus on nearby objects—a process called **accommodation.** A resting eye, with its lens stretched along its long axis by tension in the ciliary zonule, is "set" to focus the almost-parallel rays from distant points. Therefore, distance vision is the natural state (Figure 16.13a). The diverging rays from *nearby* points must be bent more sharply if they are to focus on the retina. To accomplish this, the lens is made rounder: The ciliary muscle contracts in a complex way that releases most of the tension on the ciliary zonule. No longer stretched, the lens becomes rounder as a result of its own elastic recoil (Figure 16.13b). Accommodation is controlled by the parasympathetic fibers that signal the ciliary muscle to contract.

Focusing on nearby objects is accompanied by pupillary constriction, which prevents the most divergent light rays from entering the eye and passing through the extreme edges of the lens. Such rays would not be focused properly and would cause blurred vision.

For simplicity, this discussion of eye focusing is confined to single-point images; how the eye focuses the "multiple-point" images of large objects is beyond the scope of this text.

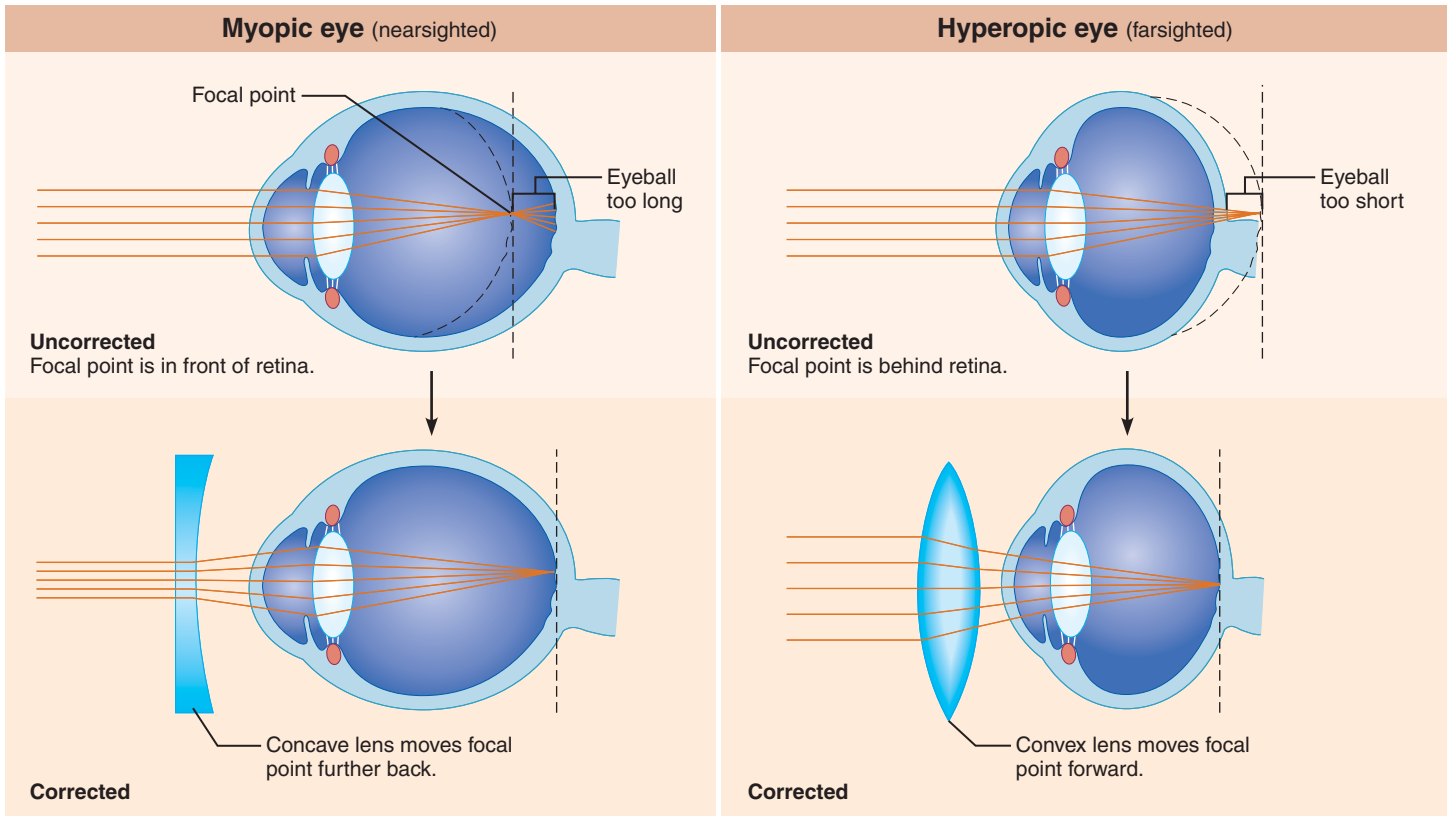


FIGURE 16.14 Eye-focusing disorders.

However, it must be noted that the convex lens of the eye, just like the convex lens of a camera, produces images that are upside down and reversed from right to left. Therefore, an inverted and reversed image of the visual field is projected onto each retina (Figure 16.13). The cerebral cortex then "flips" the image back, so that we see things as they are actually oriented.

FOCUSING DISORDERS Focusing disorders of the eye are common (Figure 16.14). **Myopia,** commonly referred to as nearsightedness, occurs when the shape of the eye or the bending of the lens results in a focal point for distant objects that is in front of the retina, creating a blurry image on the retina. Concave lenses, which diverge the incoming rays of light and thus move the focal point posteriorly, correct for this disorder. **Hyperopia,** or farsightedness, results when the eye is short, causing the focal point to occur behind the retina. For viewing distant objects, the lens of the eye can adequately correct for this, enabling clear vision; for viewing close objects, convex corrective lenses, which converge the light rays as they approach the eye and move the focal point anteriorly, are needed. **Presbyopia** affects people as they reach middle age. The lens becomes thicker and less elastic and thus less able to accommodate for near vision. As with hyperopia, convex corrective lenses, commonly called reading glasses, correct for this disorder.

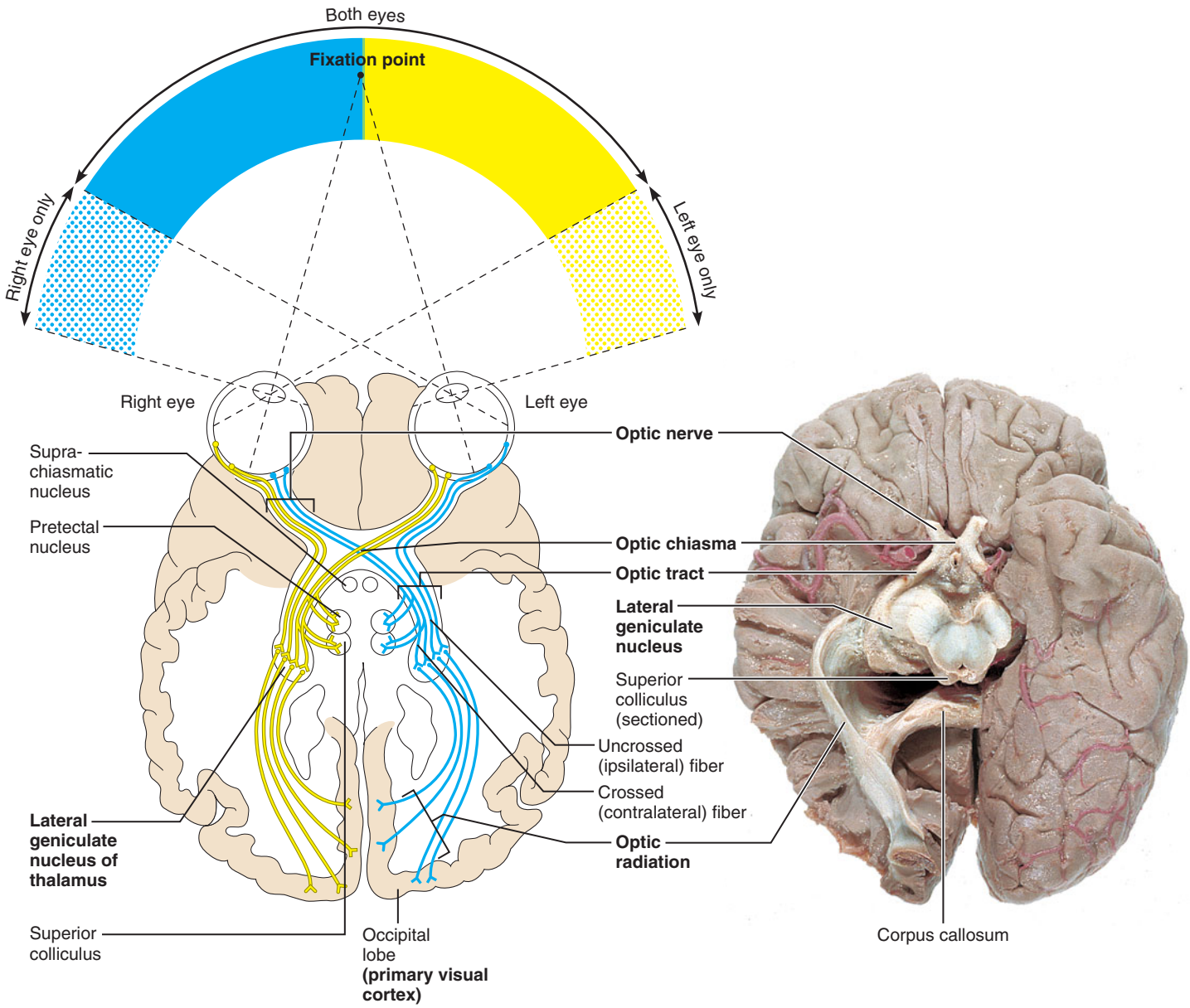
Visual Pathways

➤ Trace the pathway of nerve impulses from the retina to the cerebral cortex.

Visual information leaves the eye and travels to the brain for complex processing. Most of this visual information goes to the cerebral cortex, which is responsible for conscious "seeing" (recall Chapter 13), but some goes to nuclei in the midbrain and diencephalon, which control reflexes and subconscious behaviors that require visual input.

Visual Pathway to the Cerebral Cortex

Visual information travels to the cerebral cortex through the main **visual pathway** (Figure 16.15). Axons of the ganglion cells exit the eye in the **optic nerve.** At the X-shaped **optic chiasma** (ki-as′mah; "cross"), which lies anterior to the hypothalamus, the axons from the medial half of each eye decussate and then continue in an **optic tract.** However, axons from the area of the retina lateral to the fovea do not cross at the optic chiasma; they continue to the ipsilateral optic tract. The paired optic tracts sweep posteriorly around the hypothalamus and send most of their axons to the **lateral geniculate nucleus of the thalamus,** where they synapse with thalamic neurons. Axons of those neurons then project through the internal capsule to form the **optic radiation** of fibers in the cerebral white matter. These fibers reach the **primary visual cortex** in the occipital lobe, where conscious perception of visual images occurs.



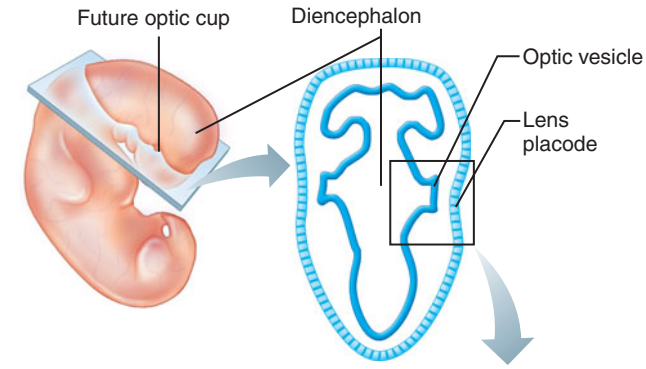
(a) The visual fields of the two eyes overlap considerably. Note that fibers from the lateral portion of each retinal field do not cross at the optic chiasma.

(b) Photograph of human brain, with the right side dissected to reveal internal structures.

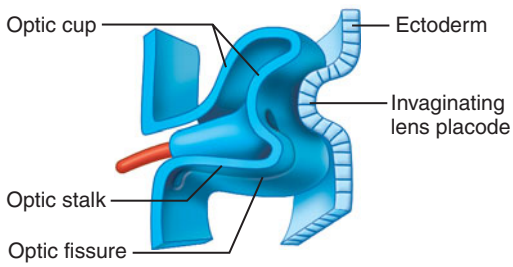
FIGURE 16.15 Visual pathway to the brain and visual fields, inferior view.

The partial decussation of axons in the optic chiasma relates to depth perception, which is also called stereoscopic, or three-dimensional, vision. To understand this, you can visualize the retina of each eye as divided into a medial and a lateral half (see the dashed lines extending from the fixation point that bisect the eyes in Figure 16.15a). Recall that the lens system of each eye reverses all images. Because of this reversal, the medial half of each retina receives light rays from the lateral (peripheral) part of the visual field, that is, from objects that lie either to the left or to the right rather than straight ahead. Correspondingly, the lateral half of each retina receives an image of the central part of the visual field. Only those axons from the *medial* halves of the two retinas cross over at the optic chiasma. The result is that all information from the left half of the visual field (shown in yellow in Figure 16.15a) is directed through the right optic tract to be perceived by the right cerebral cortex. Likewise, the right half of visual space (shown in blue) is perceived by the left visual cortex. Each cerebral cortex receives an image of half the visual field, as viewed by the two different eyes from slightly different angles. The cortex then compares these two similar but different images and, in doing so, creates the perception of depth.

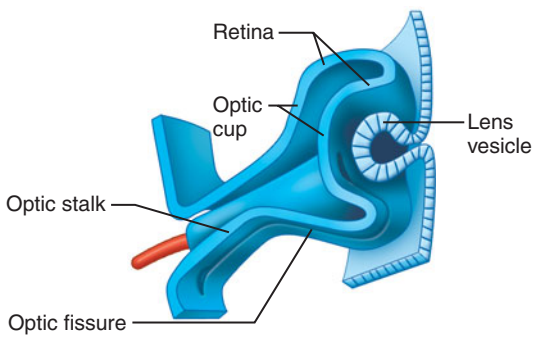
These relationships explain patterns of blindness that follow damage to different visual structures. Destruction of one eye or one optic nerve eliminates true depth perception and causes a loss of peripheral vision on the side of the damaged eye. Thus, if the "Left eye" in Figure 16.15a were lost, nothing could be seen in the visual area stippled yellow in that figure.



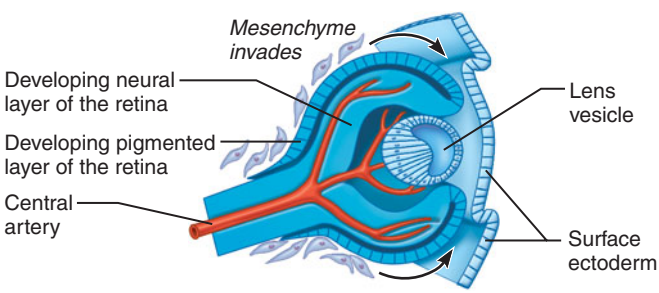
(a) Week 4, early. Outpocketing of the diencephalon forms the optic vesicles.



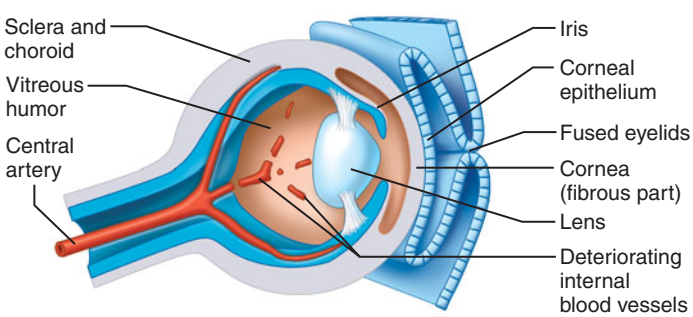
(b) Week 4, late. Optic vesicles invaginate to form the optic cups. The overlying surface ectoderm thickens to form the lens placode.



(c) Week 5. Lens placode invaginates and forms the lens vesicle.



(d) Week 6. The neural and pigmented layers of the retina differentiate from the optic cup. Central artery reaches the interior of the eye. Mesenchyme derived from neural crest invades.



(e) Week 7. Mesenchyme surrounds and invades the optic cup to form the fibrous and vascular layers and the vitreous humor. Lens vesicle forms the lens. Surface ectoderm forms the corneal epithelium and the conjunctiva.

FIGURE 16.16 Embryonic development of the eye.

However, if damage occurs beyond the optic chiasma—in an optic tract, the thalamus, or the visual cortex—then the entire opposite half of the visual field is lost. For example, a stroke affecting the left visual cortex leads to blindness (blackness) throughout the right half of the visual field.

Visual Pathways to Other Parts of the Brain

Some axons from the optic tracts send branches to the midbrain (Figure 16.15a). These branches go to the **superior colliculi,** reflex nuclei controlling the extrinsic eye muscles (discussed on p. 388), and to the **pretectal nuclei,** which mediate the pupillary light reflexes. Other branches from the optic tracts run to the **suprachiasmatic nucleus** of the hypothalamus, which is the "timer" that runs our daily biorhythms and requires visual input to keep it in synchrony with the daylight-darkness cycle.

Disorders of the Eye and Vision

The most common of the visual disorders—glaucoma, cataracts, and disorders of accommodation—have already been discussed. This section covers some other common and important eye disorders, namely *age-related macular degeneration, retinopathy of prematurity,* and *trachoma.*

Age-related macular degeneration (AMD) is a progressive deterioration of the retina that affects the macula lutea and leads to loss of central vision. It is the main cause of vision loss in those over age 65, and 200,000 Americans develop it each year. Early stages or mild forms of AMD involve the buildup of visual pigments in the macula caused by loss of cells in the pigmented layer of the retina that normally remove the damaged visual pigments. Continued accumulation of the pigment is associated with the "dry" form of AMD, in which many of the macular photoreceptors die. Far less common is the "wet" form, in which new blood vessels grow into the retina from the choroid layer, then bleed and cause scarring and detachment of the retina. The ultimate cause is unknown. AMD is largely untreatable, although laser treatments can kill some of the growing vessels in the wet form.

Retinopathy of prematurity is a visual impairment that affects many infants born so prematurely that they need to receive oxygen in an oxygen tent. When the infant is weaned from the high concentrations of oxygen, new blood vessels start to grow extensively within the eyes. These abnormal vessels have weak walls, and they hemorrhage, leading to retinal detachment and then blindness. Two treatments to kill many of the growing vessels at their source, laser and cryosurgery (applying cold needles to a circle of points around the eyeball just external to the ora serrata), have been only slightly successful in preventing loss of vision in this disorder.

Trachoma (trah-ko′mah; "rough growth") is a highly contagious infection of the conjunctiva and cornea, caused by the bacterium *Chlamydia trachomatis.* It is transmitted by hand-to-eye contact, by flies that go from eye to eye, or by placing contaminated objects in or near the eye (towels, eye liner, etc.). Symptoms begin with an inflammation of the conjunctiva of the upper eyelid; then the conjunctiva and cornea become highly vascularized, and finally, scarred. The corneal scarring reduces vision and causes blindness. Common worldwide, trachoma blinds millions of people in third-world countries. It is effectively treated with eye ointments containing antibiotic drugs.

Embryonic Development of the Eye

➤ Describe the embryonic development of the eye.

The eyes develop as outpocketings of the brain. By week 4, paired lateral outgrowths called **optic vesicles** protrude from the diencephalon **(Figure 16.16a)**. Soon, these hollow vesicles indent to form double-layered **optic cups** (Figure 16.16b). The proximal parts of the outgrowths, called the **optic stalks,** form the basis of the optic nerves.

Once a growing optic vesicle reaches the overlying surface ectoderm, it signals the ectoderm to thicken and form a **lens placode.** By week 5, this placode has invaginated to form a **lens vesicle** (Figure 16.16c). Shortly thereafter, the lens vesicle pinches off into the optic cup, where it becomes the lens.

The internal layer of the optic cup differentiates into the neural retina, whereas the external layer becomes the pigmented layer of the retina (Figure 16.16d). The **optic fissure,** a groove on the underside of each optic stalk and cup, serves as a direct pathway for blood vessels to reach and supply the interior of the developing eye. When this fissure closes, the optic stalk becomes a tube through which the optic nerve fibers, originating in the retina, grow centrally to reach the diencephalon. The blood vessels that were originally within the optic fissure now lie in the center of the optic nerve.

The fibrous layer, vascular layer, and vitreous humor form from head mesenchyme that surrounds the early optic cup and invades the cup's interior. The central interior of the eyeball has a rich blood supply during development, but these blood vessels degenerate, leaving only those in the vascular layer and retina (Figure 16.16e).

check your understanding

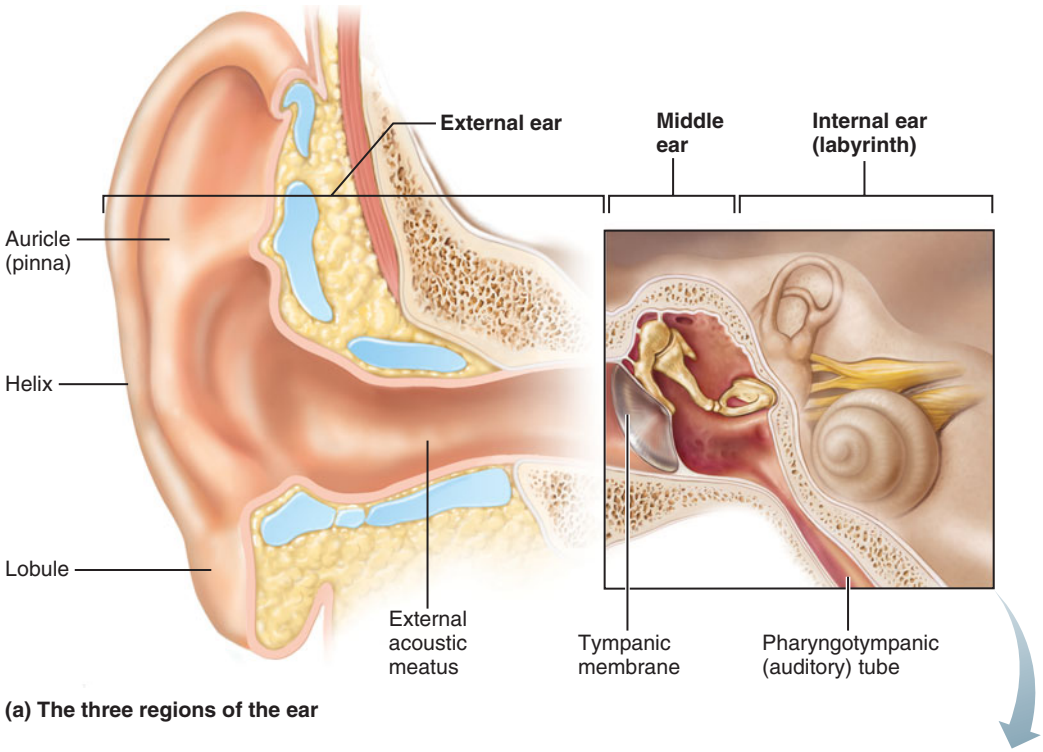
8. In a normal eye, when the lens accommodates for viewing close objects, does it become rounder or more oval in shape?
9. Why do middle-aged to older adults develop difficulty focusing on close objects?
10. Trace the visual pathway from the medial half of the retina of the right eye to the cerebral cortex.
11. Which structure of the eye forms from the outpocketing of the neural tube?

For answers, see Appendix B.

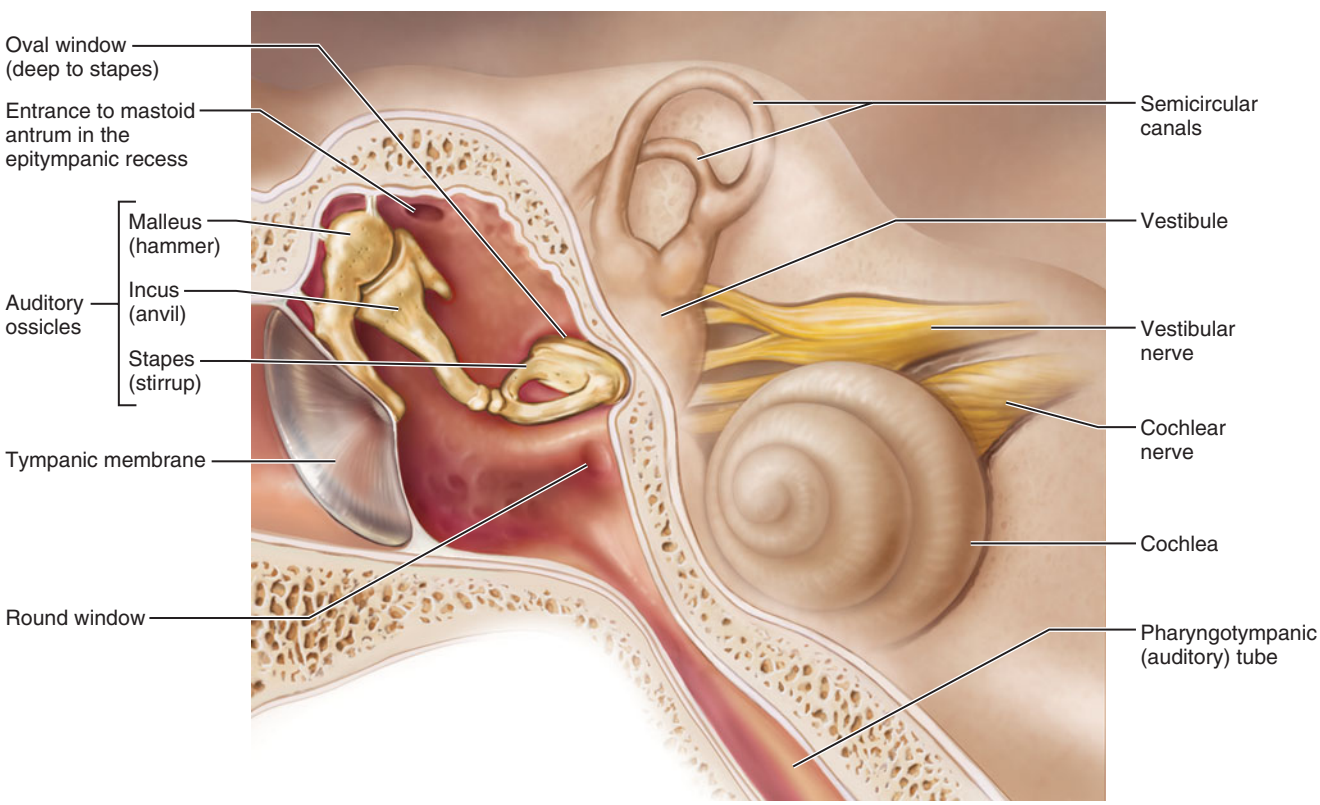
THE EAR: HEARING AND EQUILIBRIUM

➤ List the basic structures of the external and middle ear and their corresponding functions.

The **ear,** the receptor organ for both hearing and equilibrium, has three main regions: the *external ear,* the *middle ear,* and the *internal ear* **(Figure 16.17a)**. The external and middle



(a) The three regions of the ear



(b) Middle and internal ear

FIGURE 16.17 Structure of the ear. In (b) the bony labyrinth of the internal ear is illustrated.

ears participate in hearing only, whereas the internal ear functions in both hearing and equilibrium.

The External Ear

The **external ear** consists of the auricle and the external acoustic meatus. The **auricle,** or **pinna,** is what most people call the ear—the shell-shaped projection that surrounds the opening of the external acoustic meatus. Most of the auricle, including the **helix** (rim), consists of elastic cartilage covered with skin. Its fleshy, dangling **lobule** ("earlobe"), however, lacks supporting cartilage. The function of the auricle is to gather and funnel (and thereby amplify) sound waves coming into the external acoustic meatus. Moreover, the way that sound bounces off the ridges and cavities of the auricle provides the brain with clues about whether sounds come from above or below.

The **external acoustic meatus** is a short tube (about 2.5 cm long) running medially, from the auricle to the eardrum. Near the auricle, its wall consists of elastic cartilage, but its medial two-thirds tunnels through the temporal bone. The entire canal is lined with skin that contains hairs, as well as sebaceous glands and modified apocrine sweat glands called *ceruminous* (sĕ-roo′mĭ-nus) *glands.* The ceruminous and sebaceous glands secrete yellow-brown *cerumen,* or earwax (*cere* = wax). Earwax traps dust and repels insects, keeping them out of the auditory canal.

Sound waves entering the external acoustic meatus hit the thin, translucent **tympanic membrane,** or eardrum (*tympanum* = drum), which forms the boundary between the external and middle ears. It is shaped like a flattened cone, the apex of which points medially into the middle ear cavity. Sound waves that travel through the air set the eardrum vibrating, and the eardrum in turn transfers the vibrations to tiny bones in the middle ear (discussed next).

PERFORATED EARDRUM Undue pressure from a cotton swab or sharp object in the external acoustic meatus can tear the tympanic membrane, a condition called a **perforated eardrum.** A more common cause, however, is a middle ear infection, in which the accumulation of pus medial to the eardrum exerts pressure that bursts the thin membrane. Perforated eardrums heal well, but small amounts of scarring can permanently diminish hearing acuity.

The Middle Ear

The **middle ear,** or *tympanic cavity,* is a small, air-filled space inside the petrous part of the temporal bone. It is lined by a thin mucous membrane and is shaped like a hockey puck standing on its side (Figure 16.17b). Its *lateral boundary* is the tympanic membrane; its *medial boundary* is a wall of bone that separates it from the inner ear. Two small holes penetrate this medial wall: a superior **oval window** and an inferior **round window.** Superiorly, the middle ear arches upward as the **epitympanic recess** (*epi* = over); its *superior boundary* is the roof of the petrous portion of the temporal bone, which is so thin here that middle ear infections can spread to the overlying meninges and brain. The *posterior wall* of the middle ear opens into the **mastoid antrum,** a canal leading to the *mastoid air cells* in the mastoid process. Infections can spread from the middle ear to the mastoid air cells, using the antrum as a passageway. The *anterior wall* of the middle ear lies just behind the internal carotid artery, the main artery to the brain, and it also contains the opening of the pharyngotympanic tube (discussed shortly). The *inferior boundary* of the middle ear is a thin, bony floor, under which lies the important internal jugular vein. Middle ear infections may burst through this floor and clot the blood in this vein.

The **pharyngotympanic tube,** formerly named the *auditory tube* or *eustachian tube,* links the middle ear to the pharynx (Figure 16.17). About 4 cm (1.5 inches) long, it runs medially, anteriorly, and inferiorly. Its lateral third consists of bone and occupies a groove on the inferior surface of the skull; its medial two-thirds is cartilage and opens into the side wall of the superior pharynx behind the nasal cavity. This normally flattened and closed tube can be opened briefly by swallowing or yawning so that the air pressure in the middle ear equalizes with the outside air pressure. This is important because the eardrum does not vibrate freely unless the pressure on both its surfaces is the same. Differences in air pressure build up across the eardrum during rapid changes in altitude (as during takeoff and landing in an airplane). The next time your ears "pop" in such a situation, remember that yawning is the best way to open your pharyngotympanic tubes and equalize the air pressures.

MIDDLE EAR INFECTIONS Infection and inflammation of the middle ear, called **otitis media** (o-ti′tis; "ear inflammation"), usually starts as a throat infection that spreads to the middle ear through the pharyngotympanic tube. Fluid and pus can build up in the middle ear cavity and exert painful pressure within this enclosed space. More children than adults develop otitis media because the child's pharyngotympanic tube is shorter and enters the pharynx at a less acute angle. Extremely common, otitis media accounts for one-third of all visits to pediatricians in the United States and is frequently treated with antibiotics. However, the overuse of antibiotics has led to bacterial resistance, making persistent and recurrent cases increasingly difficult to treat.

Children with persistent otitis media sometimes have their eardrums lanced and have ear tubes inserted through the eardrum. Such a **myringotomy** (mir″ing-got′-o-me; "lancing the eardrum") allows the middle ear to drain and relieves the pressure. The tiny tube that is inserted through the eardrum during myringotomy permits the pus to drain into the external ear. This tube is left in the eardrum and falls out by itself within a year.

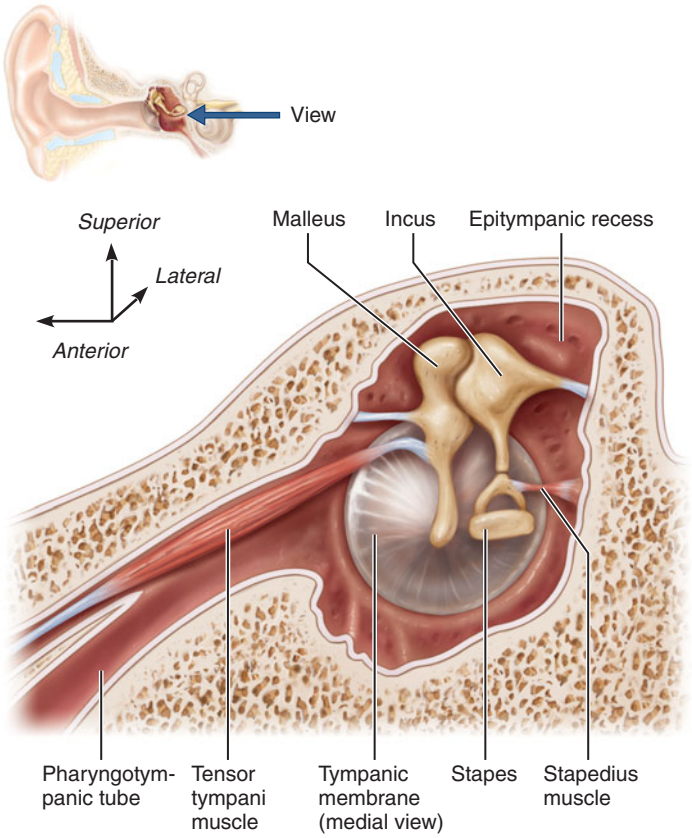


FIGURE 16.18 Skeletal muscles associated with the auditory ossicles (right ear, medial view).

The tympanic cavity is spanned by the three smallest bones in the body, the **auditory ossicles** (Figure 16.17b), which transmit the vibrations of the eardrum across the cavity to a fluid in the inner ear. From lateral to medial, the auditory ossicles are the **malleus** (mal′e-us), or hammer, which looks like a club with a knob on top; the **incus** (ing′kus), or anvil, which resembles a tooth with two roots; and the **stapes** (sta′pēz), which looks like the stirrup of a saddle. The handle of the malleus attaches to the eardrum, and the base of the stapes vibrates against the *oval* window. Most people have trouble remembering whether the stapes fits into the oval window or into the round window inferior to it: To remember, think of the footplate of a saddle stirrup, which is usually oval, not round.

OTOSCLEROSIS Excessive growth of bone tissue in the walls of the middle ear cavity can cause the fusion of the footplate of the stapes to the oval window. As a result, the stapes cannot move, and deafness results. This condition, called **otosclerosis** (o″to-sklĕ-ro′sis; "hardening of the ear"), is a common age-related problem that affects 1 in every 200 people. It can be treated by a delicate surgery that removes the stapes and replaces it with a prosthetic (artificial) one.

Tiny ligaments suspend the ossicles in the middle ear, and tiny synovial joints link the ossicles into a chain. By concentrating the vibrations of the eardrum onto the much smaller oval window, the ossicles amplify the pressure of the sound vibrations about 20-fold. Without the ossicles, people could hear only loud sounds.

Two tiny skeletal muscles occur in the middle ear cavity (Figure 16.18). The **tensor tympani** (ten′sor tim′pah-ni) originates on the cartilage part of the pharyngotympanic tube and inserts on the malleus. The **stapedius** (stah-pe′de-us) runs from the posterior wall of the middle ear to the stapes. When the ears are assaulted by very loud sounds, these muscles contract reflexively to limit the vibration of the ossicles and thus prevent damage to the hearing receptors (discussed shortly).

check your understanding

12. What structure separates the external acoustic meatus from the middle ear cavity?
13. Name the four openings or holes into the middle ear cavity.
14. Which auditory ossicle abuts the tympanic membrane?

For answers, see Appendix B.

The Internal Ear

- Name the parts of the bony and membranous labyrinths in the internal ear.
- Describe the receptors for hearing and equilibrium.

The **internal ear,** also called the **labyrinth** ("maze") because of its mazelike, complex shape (Figure 16.17), lies within the thick, protective walls of the petrous part of the temporal bone. The internal ear consists of two main divisions: the bony labyrinth and the membranous labyrinth (Figure 16.19).

The **bony labyrinth** is a *cavity* in the petrous bone consisting of a system of twisting channels that has three parts. From posterolateral to anteromedial, these parts are the *semicircular canals,* the *vestibule,* and the *cochlea* (Figure 16.17). Textbooks often picture the bony labyrinth as though it were a solid object, but it is actually a cavity.

The **membranous labyrinth** is a continuous series of membrane-walled sacs and ducts that fit loosely within the bony labyrinth and more or less follow its contours (Figure 16.19). The main parts of the membranous labyrinth are (1) the *semicircular ducts,* one inside each semicircular canal; (2) the *utricle* and *saccule,* both in the vestibule; and (3) the *cochlear duct* in the cochlea. The wall of the membranous labyrinth—its "membrane"—is a thin layer of connective tissue lined by a simple squamous epithelium. Parts of this epithelium are thickened and contain the receptors for equilibrium and hearing. The parts of the bony and membranous labyrinths are summarized in Table 16.1.

The membranous labyrinth is filled with a clear fluid called **endolymph** (en′do-limf; "internal water"). External to the membranous labyrinth, the bony labyrinth is filled with

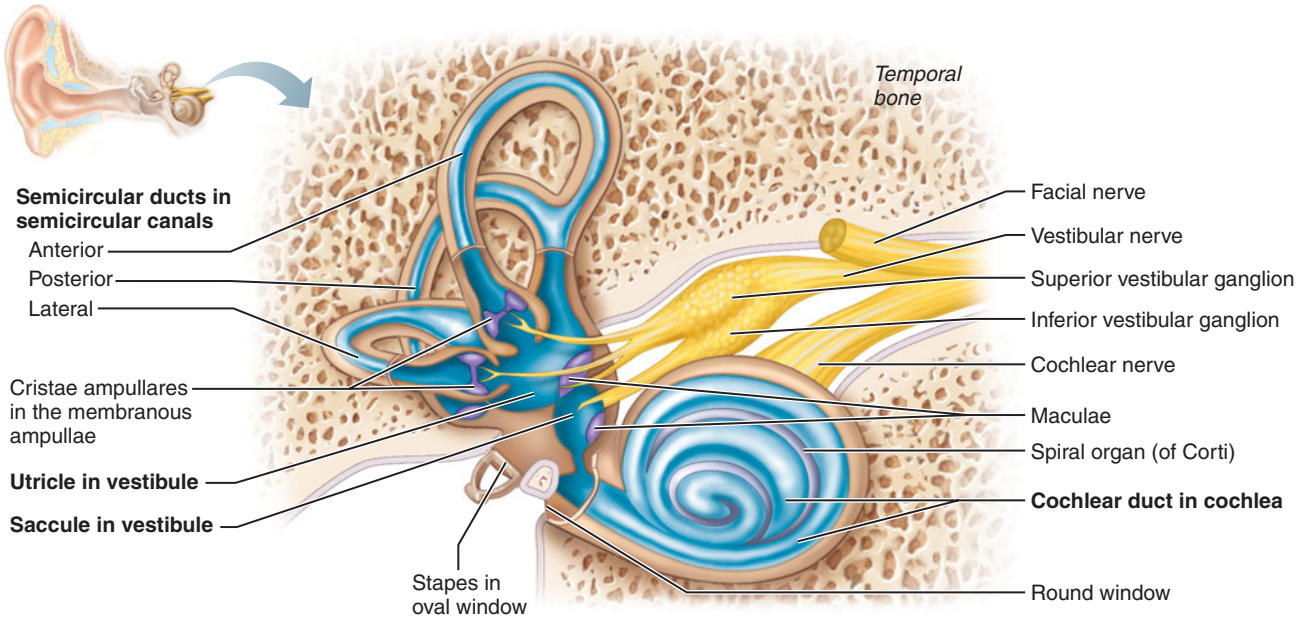


FIGURE 16.19 The internal ear. The membranous labyrinth (blue) lies within the chambers of the bony labyrinth (tan). The locations of the sensory organs for hearing (spiral organ) and equilibrium (maculae and cristae ampullares) are shown in purple.

another clear fluid called **perilymph** (per′ĭ-limf; "surrounding water"). The perilymph is continuous with the cerebrospinal fluid that fills the subarachnoid space. Nowhere are the perilymph and endolymph continuous with one another.

You will explore the basic parts of the bony and membranous labryinth next, beginning with the cochlea. It is located most inferiorly in the labyrinth, and it contains receptors for hearing.

The Cochlea

The **cochlea** (kok′le-ah; "snail shell") is a spiraling chamber in the bony labyrinth (Figures 16.17 and 16.19). It is about the size of a split pea. From its attachment to the vestibule at its base, it coils for about two and a half turns around a pillar of bone called the **modiolus** (mo-di′o-lus) **(Figure 16.20a)**. The modiolus is shaped like a screw whose tip lies at the *apex of the cochlea,* pointing anterolaterally. Just as screws have threads, the modiolus has a spiraling projection of bone called the **osseous spiral lamina.** Running through the bony core of the modiolus is the **cochlear nerve,** which is the cochlear division of the vestibulocochlear nerve.

The coiled part of the membranous labyrinth within the cochlea is called the **cochlear duct** (Figure 16.20a and b).This is the location of the sensory receptors for hearing. This duct winds through the cochlea and ends blindly in the cochlear apex. Within the cochlea, the endolymph-filled cochlear duct (or **scala media**) lies between two perilymph-filled chambers of the bony labyrinth, the **scala vestibuli** and **scala tympani** (*scala* = ladder) (Figure 16.20b). As shown in Figure 16.21, the scala vestibuli is continuous with the vestibule near the base of the cochlea where it abuts the oval window. The scala tympani ends at the round window at the base of the cochlea. The scala vestibuli and scala tympani are continuous with each other at the apex of the cochlea, in a region called the **helicotrema** (hel″ĭ-ko-tre′mah; "the hole in the spiral").

TABLE 16.1 The Inner Ear: Basic Structures of the Bony and Membranous Labyrinths

Bony Labyrinth	Membranous Labyrinth (Within Bony Labyrinth)	Functions of the Membranous Labyrinth
Semicircular canals	Semicircular ducts	Equilibrium: rotational (angular) acceleration of the head
Vestibule	Utricle and saccule	Equilibrium: static equilibrium and linear acceleration of the head.
Cochlea	Cochlear duct	Hearing

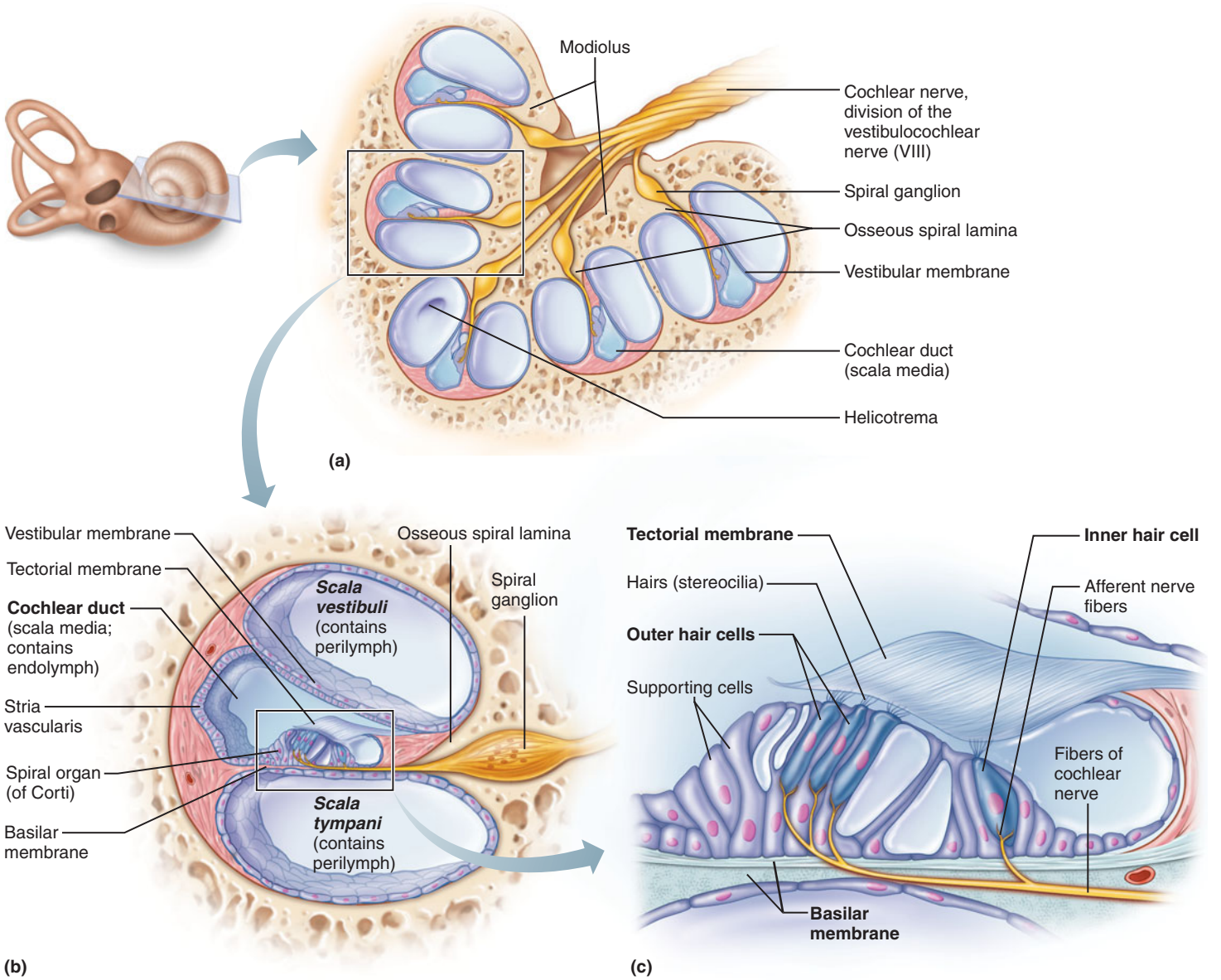
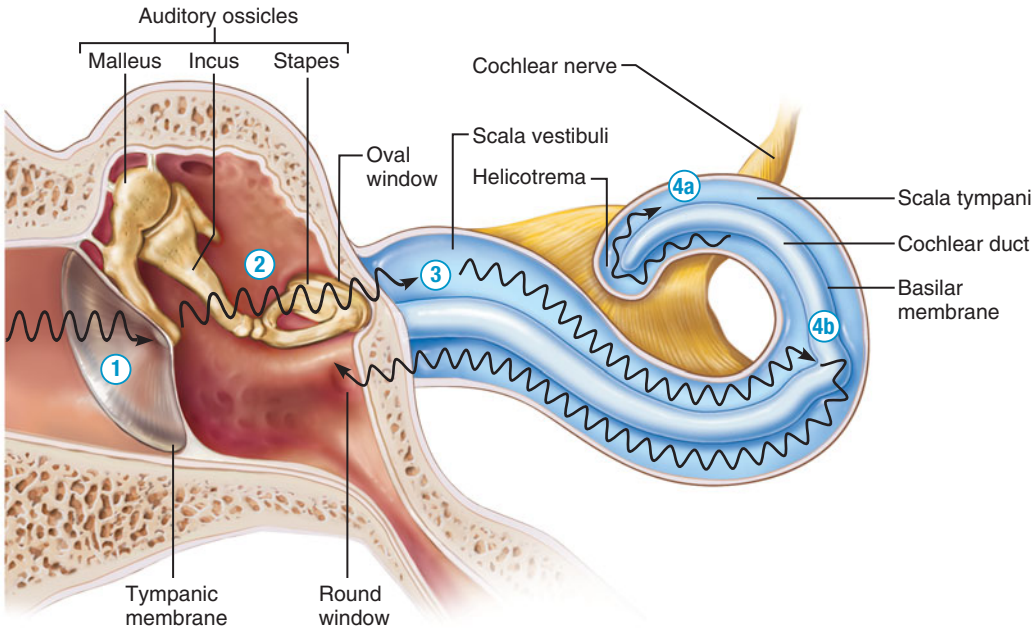


FIGURE 16.20 Anatomy of the cochlea. (a) A section through the right cochlea, superior half removed. The area containing the helicotrema is at the apex of the cochlea. **(b)** Magnified cross section of one turn of the cochlea, showing the three scalae: scala vestibuli, scala tympani, and cochlear duct (scala media). **(c)** Detailed structure of the spiral organ (of Corti). **(d)** Color-enhanced scanning electron micrograph of the spiral organ showing the cochlear hair cells (3000×).



1 Sound waves vibrate the tympanic membrane.

2 Auditory ossicles vibrate. Pressure is amplified.

3 Pressure waves created by the stapes pushing on the oval window move through fluid in the scala vestibuli.

4a Sounds with frequencies below hearing travel through the helicotrema and do not excite hair cells.

4b Sounds in the hearing range go through the cochlear duct, vibrating the basilar membrane and deflecting hairs on inner hair cells.

FIGURE 16.21 Role of the cochlea in hearing. The cochlea is drawn as if uncoiled. The structure of the basilar membrane is such that it segregates sound according to frequency—its basal part vibrates in response to high-pitched sounds, whereas its apical part vibrates in response to low-pitched sounds.

The cochlear duct contains the receptors for hearing (Figure 16.20b). The "roof" of the cochlear duct, separating it from the scala vestibuli, is the **vestibular membrane.** The external wall of this duct is the *stria vascularis* ("vascularized streak"), an unusual epithelium that contains capillaries and secretes the endolymph of the inner ear. The floor of the cochlear duct consists of the osseous spiral lamina plus an attached sheet of fibers called the **basilar membrane.** The basilar membrane supports the **spiral organ (of Corti),** the receptor epithelium for hearing. This tall epithelium (Figure 16.20c) consists of columnar *supporting cells* and one row of **inner** and three rows of **outer hair cells,** which are the receptor cells. At the cell's apex, the tips of the hairs, the *stereocilia*, are embedded in a gel-like **tectorial membrane** ("roofing membrane"); at their base the hair cells synapse with sensory fibers of the cochlear nerve. These nerve fibers belong to bipolar neurons, whose cell bodies occupy a **spiral ganglion** in the osseous spiral lamina and modiolus (see Figure 16.20b) and whose central fibers project to the brain.

Both the inner and outer hair cells have three stereocilia of increasing length extending from the apical surface of each cell. The stereocilia in the inner hair cells are arranged linearly; those in the outer hair cells form a W pattern (Figure 16.20d).

How do sound waves stimulate the hair cells in the spiral organ? First, sound vibrations travel from the eardrum through the ossicles, causing the stapes to oscillate back and forth against the oval window (**Figure 16.21** 1 and 2). This oscillation sets up pressure waves in the perilymph of the scala vestibuli (Figure 16.21 3), which are transferred to the endolymph of the cochlear duct (Figure 16.21 4b). These waves cause the basilar membrane to vibrate up and down. The hair cells in the spiral organ move along with the basilar membrane (Figure 16.20c), but the overlying tectorial membrane (in which the hairs are anchored) does not move. Therefore, the movements of the hair cells cause their hairs to bend. Each time such bending occurs in a specific direction, the hair cells release neurotransmitters that excite the cochlear nerve fibers, which carry the vibratory (sound) information to the brain. The vibrations of the basilar membrane set the perilymph vibrating in the underlying scala tympani. These vibrations then travel to the round window, where they push on the membrane that covers that window, thereby dissipating their remaining energy into the air of the middle ear cavity. Without this release mechanism, echoes would reverberate within the rigid cochlear box, disrupting sound reception.

The inner and outer hair cells in the spiral organ have different functions. The *inner* hair cells are the true receptors that transmit the vibrations of the basilar membrane to the cochlear nerve. The *outer* hair cells are involved with actively tuning the cochlea and amplifying the signal. The outer hair cells receive efferent fibers from the brain that cause these cells to stretch and contract, enhancing the responsiveness of the inner hair cell receptors. Overall, this active mechanism amplifies sounds some 100 times, so that we can hear the faintest sounds.The mobility of the outer hair cells is also responsible for producing ear sounds *(otoacoustic emissions)*. Detection of spontaneous otoacoustic emissions is used to test hearing in newborns.

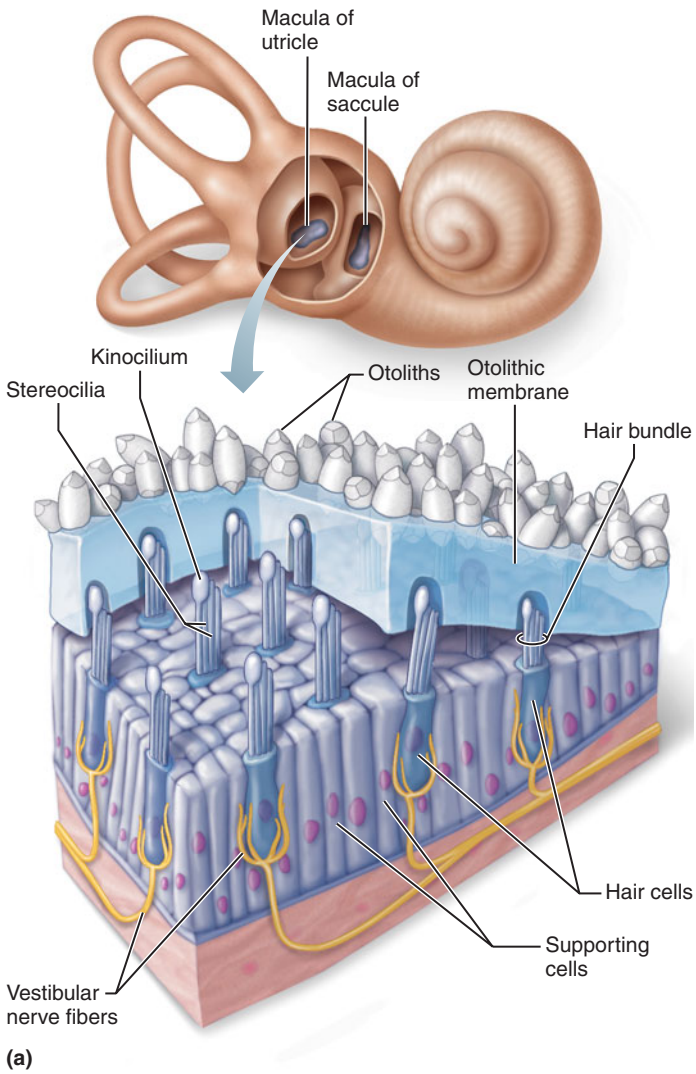


FIGURE 16.22 The maculae in the internal ear.
(a) Structure of a macula. **(b)** Function of the macula of the utricle in signaling the position of the head.

The Vestibule

The **vestibule** is the central cavity of the bony labyrinth (Figure 16.19). It lies just medial to the middle ear, and the oval window is in its lateral bony wall. Suspended within its perilymph are the two egg-shaped parts of the membranous labyrinth, the **utricle** (u′tri-k'l; "leather bag") and the **saccule** ("little sac"). The utricle is continuous with the semicircular ducts; the saccule, with the cochlear duct.

The utricle and saccule each house a spot of sensory epithelium called a **macula** (mak′u-lah; "spot") (Figure 16.19 and Figure 16.22a). Both the macula of the utricle and the macula of the saccule contain receptor cells that monitor the position of the head when the head is held still. This aspect of the sense of balance is called *static equilibrium.* These receptor cells also monitor straight-line changes in the speed and the direction of head movements—that is, *linear acceleration*—but not rotational movements of the head.

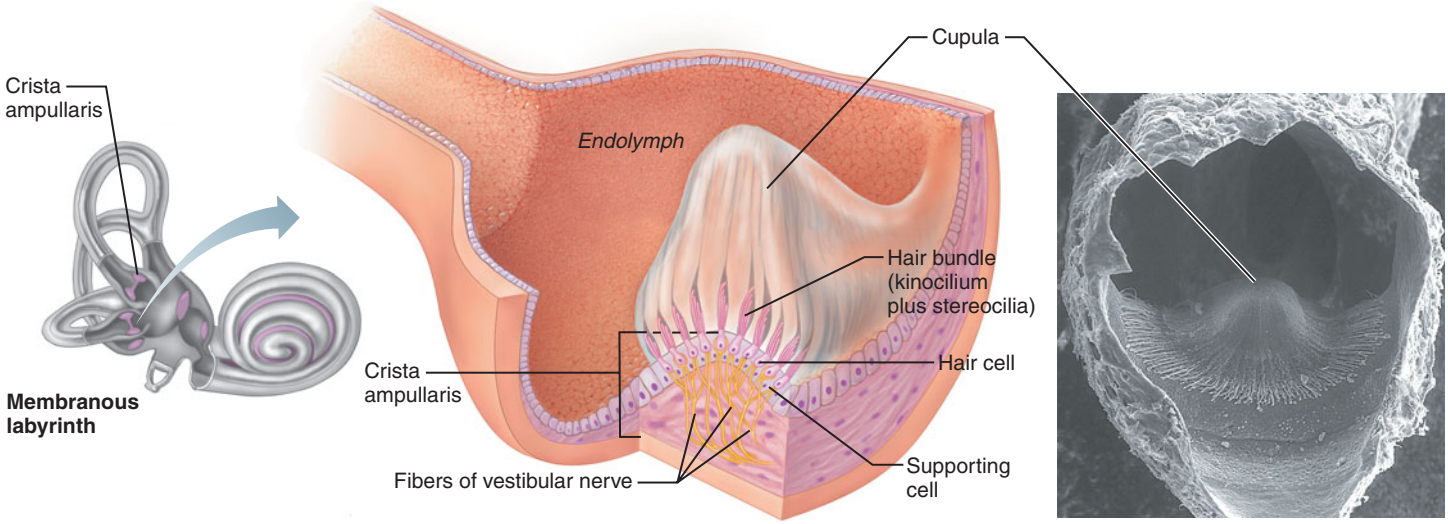
Each macula is a patch of epithelium containing columnar **supporting cells** and scattered receptors called **hair cells** (see Figure 16.22a). The hair cells synapse with sensory fibers of the **vestibular nerve,** which is the vestibular division of the vestibulocochlear nerve. Named for the hairy look of its free surface, each hair cell has many stereocilia (long microvilli) and a single kinocilium (a true cilium) protruding from its apex. The tips of these stiff hairs are embedded in an overlying **otolithic** (o″to-lith′ik) **membrane,** which is actually a jellylike disc that contains heavy crystals of calcium carbonate called **otoliths** ("ear stones").

It is easy to understand how the maculae and otoliths contribute to the sense of static equilibrium. The macula of the *utricle* has a *horizontal* orientation within the ear (Figure 16.22a). When one holds the head in a tilted position (Figure 16.22b), the heavy otolithic membrane pulls downward, bending the receptor hairs and signaling the vestibular nerve to tell the brain that the head is tilted. The macula of the *saccule,* in contrast, has a *vertical* orientation within the ear (Figure 16.22a), so its heavy otoliths pull downward on the hairs whenever the head is upright, signaling the brain that the head is in this untilted position. It is also easy to understand how both maculae monitor the movements of linear acceleration: Whenever the body jolts forward, upward, or sideways in a straight line, the heavy otolithic membrane lags behind, again bending the hairs and signaling the brain.

The maculae are innervated by two branches of the vestibular nerve (Figure 16.19). The sensory neurons in this nerve are bipolar neurons, with cell bodies located in the *superior* and *inferior vestibular ganglia.* These ganglia lie in the internal acoustic meatus of the petrous temporal bone. The pathway of the vestibular nerve fibers within the brain is discussed shortly (p. 508).

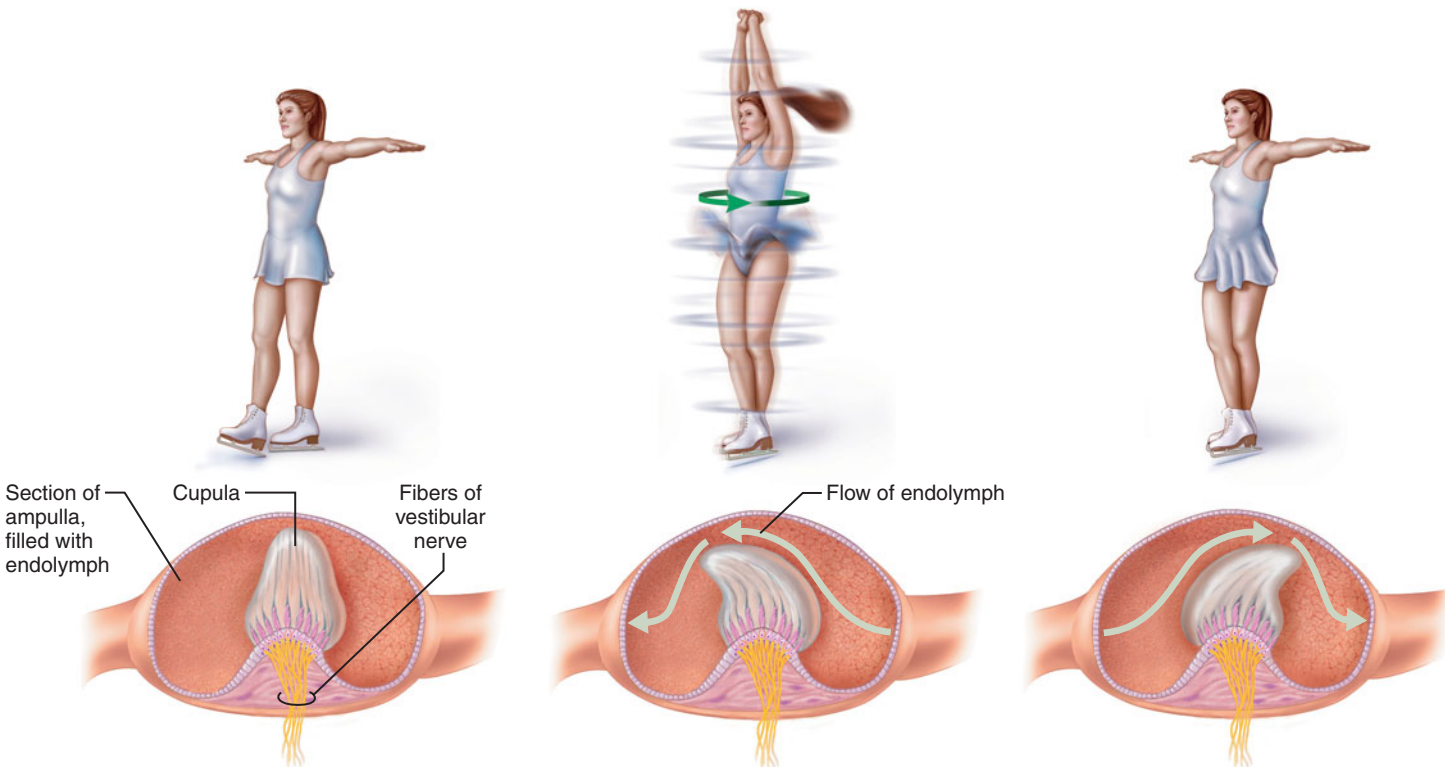
The Semicircular Canals

Whereas the vestibule houses the receptors for static equilibrium and linear acceleration, the semicircular canals house receptors for *rotational* acceleration of the head. The three **semicircular canals** of the bony labyrinth lie posterior and lateral to the vestibule (Figure 16.19). Each of these canals actually describes about two-thirds of a circle and has an



(a) Anatomy of a crista ampullaris in a semicircular canal

(b) Scanning electron micrograph of a crista ampullaris (45×)



(c) Movement of the cupula during rotational acceleration and deceleration

FIGURE 16.23 Location, structure, and function of the crista ampullaris in the internal ear.

expansion at one end called an **ampulla** ("flask"). Each canal lies in one of the three planes of space: The **anterior** and **posterior semicircular canals** lie in vertical planes at right angles to each other, whereas the **lateral semicircular canal** *(horizontal canal)* lies almost horizontally. Snaking through each semicircular canal is part of the membranous labyrinth, the **semicircular duct.** Each semicircular duct has a swelling called a **membranous ampulla** within the corresponding bony ampulla.

Each membranous ampulla houses a small crest called a **crista ampullaris,** or "crest of the ampulla" (Figure 16.19 and Figure 16.23). The three cristae ampullares contain the

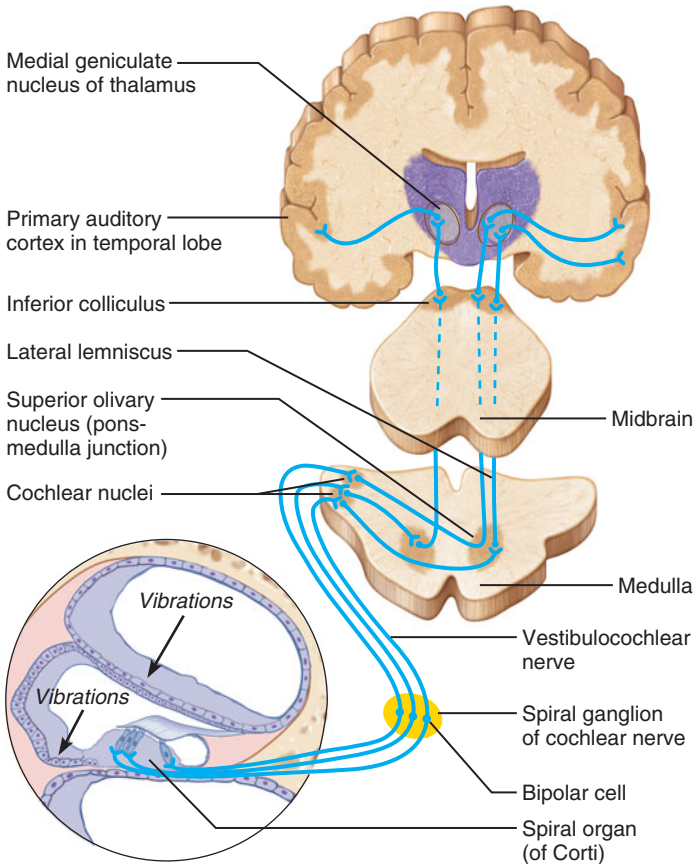


FIGURE 16.24 The auditory pathway. This simplified diagram shows only the pathway from the right ear.

receptor cells that measure *rotational (angular) acceleration* of the head, as occurs when a figure skater spins or a gymnast does a flip. Each crista has an epithelium on its top that, like the maculae, contains *supporting cells* and receptor *hair cells.* The "hairs" of these hair cells project into a tall, jellylike mass that resembles a pointed cap, the **cupula** (ku′pu-lah; "little barrel"); the basal parts of the hair cells synapse with fibers of the vestibular nerve.

Because the three semicircular ducts lie in three different planes, each crista responds to head rotation in a different plane of space. When the head starts to rotate, the endolymph in the semicircular duct lags behind at first, pushing on the cupula and bending the hairs (Figure 16.23c). As their hairs bend, the hair cells depolarize and change the pattern of impulses carried by vestibular nerve fibers to the brain.

check your understanding

15. What is the difference between the membranous labyrinth and the bony labyrinth of the internal ear?
16. Where is perilymph located within the cochlea?
17. Where in the cochlear duct is each of these membranes located: vestibular membrane, the basilar membrane, the tectorial membrane? Vibrations in which membrane stimulate the hair cells of the spiral organ?
18. Which sensory receptors monitor stationary head position and linear movements of the head? Where are these receptors located?

For answers, see Appendix B.

Auditory and Equilibrium Pathways

➤ Describe the pathways taken by auditory and equilibrium information through the brain.

As is the case for all sensory information, information on equilibrium and hearing travels to the brain for processing and integration.

The ascending **auditory pathway** transmits auditory information primarily from the cochlear receptors of the inner hair cells to the cerebral cortex **(Figure 16.24)**. First, impulses pass through the cochlear nerve to the **cochlear nuclei** in the medulla. From there, some neurons project to the **superior olivary nuclei,** which lie at the junction of the medulla and pons. Beyond this, the axons ascend in the **lateral lemniscus** (a fiber tract) to the **inferior colliculus** (the auditory reflex center in the midbrain), which projects to the **medial geniculate nucleus** of the thalamus. Axons of the thalamic neurons then project to the primary auditory cortex, which provides conscious awareness of sound. The auditory pathway is unusual in that not all of its fibers cross over to the other side of the brain. Therefore, each primary auditory cortex receives impulses from both ears. Clinically, this phenomenon makes identifying damage to the primary auditory cortex on one side difficult, because such damage produces only a minimal loss of hearing.

The superior olivary nuclei and inferior colliculus are not merely relay stations along the auditory pathway, but perform important functions of their own. For example, both these structures participate in the localization of sounds. For more on the function of the inferior colliculus, refer to p. 388.

The **equilibrium pathway** transmits information on the position and movements of the head via the vestibular nerve to the brain stem. Equilibrium is the only special sense for which most information goes to the *lower* brain centers—which are primarily reflex centers—rather than to the "thinking" cerebral cortex. This pathway reflects the fact that the responses to a loss of balance, such as stumbling, must be rapid and reflexive: In the time it takes you to "think about" correcting a fall, you would hit the ground. The vestibular nuclei in the medulla (shown in Figure 13.12c and discussed on p. 384) and the cerebellum (p. 388) are the major brain centers for processing information on equilibrium. A minor pathway to the cerebral cortex provides conscious awareness of the position and movements of the head. In this minor pathway, vestibular nerve fibers project to the vestibular nuclei, then to the thalamus, and then to the posterior insula of the cerebrum.

Disorders of Equilibrium and Hearing

➤ List the causes and symptoms of motion sickness, Ménière's syndrome, and deafness.

Motion Sickness

Motion sickness is a common disorder of equilibrium in which particular motions (such as riding in a car or aboard a ship) lead to nausea and vomiting. The cause of this condition has been difficult to determine. The most popular theory is that it arises from a mismatch of sensory inputs. For example, if you are in a rocking ship, visual inputs indicate that your body is fixed with reference to a stationary environment (your cabin), but your vestibular apparatus detects movement. The brain's resulting confusion somehow leads to motion sickness. Another theory is that motion sickness occurs because the vestibular nuclei lie near, and may project to, the centers in the medulla that control vomiting. Antimotion drugs can relieve the symptoms of motion sickness by blocking signals from the inner ear to the vomiting center.

Ménière's Syndrome

In a disorder called **Ménière's syndrome,** the membranous labyrinth is apparently distorted by excessive amounts of endolymph. Affected individuals experience a variety of symptoms: equilibrium so disturbed that standing is nearly impossible; transient but repeated attacks of vertigo, nausea, and vomiting; and "howling" in the ears, such that hearing is impaired and perhaps ultimately lost. Although less severe cases can be managed by antimotion drugs, more debilitating attacks may require diuretics (drugs that increase urine output) and restriction of dietary intake of salt—both of which decrease the volume of extracellular fluid and consequently that of endolymph. Severe cases may require surgery either to drain excess endolymph from the inner ear or to cut the vestibular nerve to relieve the vertigo. A last resort, usually deferred until all hearing is lost, is removal of the entire labyrinth.

Deafness

Any hearing loss, no matter how slight, is considered deafness. The two types of deafness, *conduction deafness* and *sensorineural deafness,* have different causes.

Conduction deafness occurs when sound vibrations cannot be conducted to the internal ear. It can be caused by earwax blocking the external acoustic meatus, a ruptured eardrum, otitis media, or otosclerosis.

Sensorineural deafness results from damage to the hair cells or to any part of the auditory pathway to the brain. Most often it results from the normal, gradual loss of hearing receptor cells that occurs throughout life. In other cases, hair cells can be destroyed at an earlier age by a single, explosively loud noise or by repeated exposure to loud music, factory noise, or airport noise. Strokes and tumors that damage the auditory cortex can also cause sensorineural deafness. When deafness reflects damage to hair cells, hearing aids can help. Traditional hearing aids simply amplify sounds, an effective strategy if the loss of hair cells is not too great. For complete sensorineural deafness, **cochlear implants** are available. Placed in the temporal bone, these devices convert sound energy into electrical signals and deliver these signals directly to the cochlear nerve fibers. Modern models have up to 24 electrodes, each responding to a different frequency, and are so effective that even children who were born deaf can hear well enough to learn to speak.

Embryonic Development of the Ear

➤ Compare and contrast the embryonic derivations of the external, middle, and internal ears.

Development of the ear begins in the fourth week after conception **(Figure 16.25)**. First the internal ear begins to form from a thickening of the surface ectoderm called the **otic placode,** which lies lateral to the hindbrain on each side of the head (Figure 16.25a). This placode invaginates to form the **otic pit** (Figure 16.25b). Then its edges fuse to form the **otic vesicle,** which detaches from the surface epithelium, as shown on the right side of Figure 16.25c. The otic vesicle takes on a complex shape and becomes the membranous labyrinth. The mesenchyme tissue around the otic vesicle becomes the petrous temporal bone—that is, the walls of the bony labyrinth.

As the internal ear develops, the middle ear starts to form. Lateral outpocketings called *pharyngeal pouches* form from the endoderm-lined pharynx (Figure 16.25b–d). The middle ear cavity and the pharyngotympanic tube develop from the first of the pharyngeal pouches. The ossicles, which will bridge the middle ear cavity, develop from cartilage bars associated with the first and second pharyngeal pouches.

Turning to the external ear, the external acoustic meatus differentiates from the first **branchial groove,** an indentation of the surface ectoderm (Figure 16.25b–d). The auricle of the external ear grows from a series of bulges around this branchial groove.

check your understanding

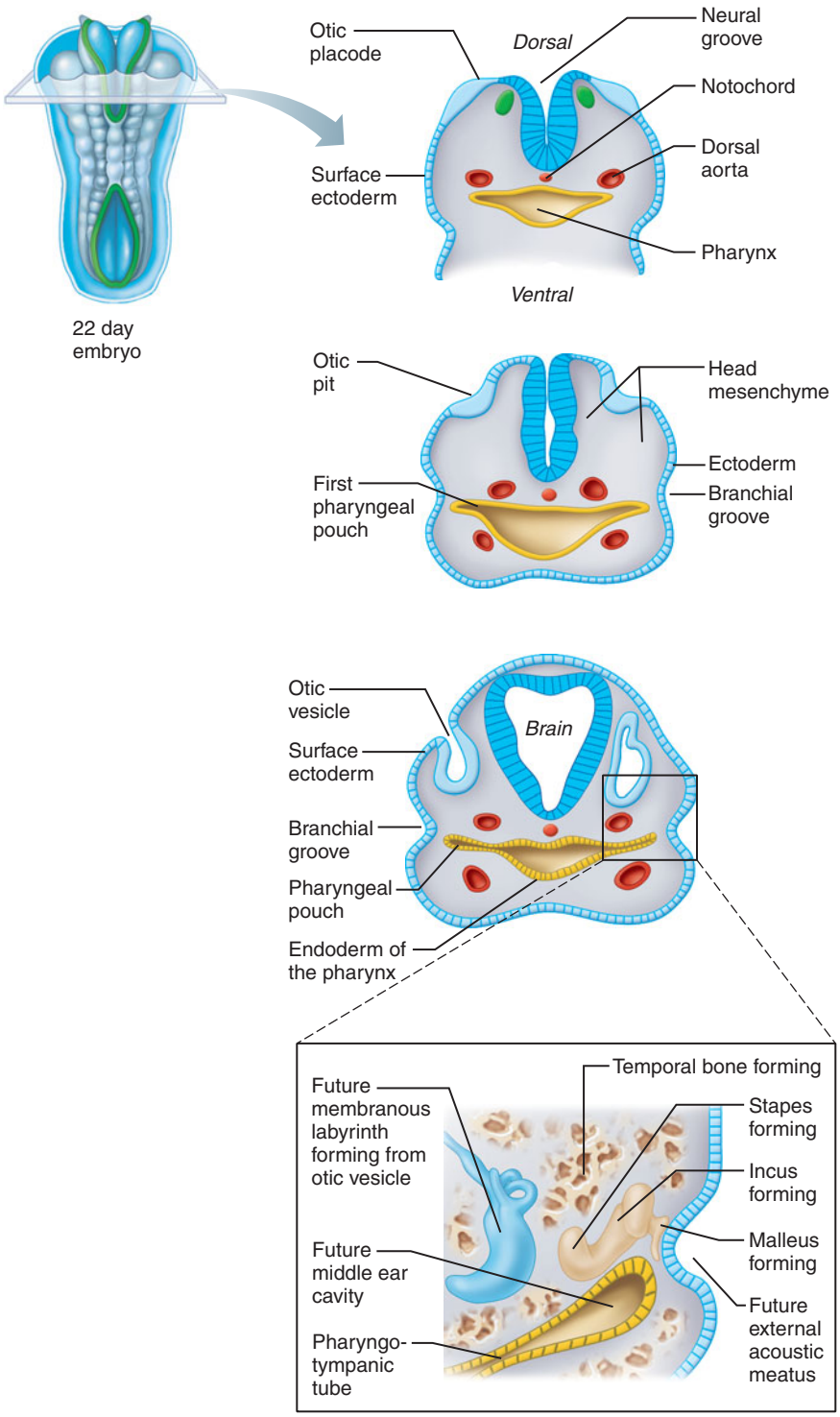
19. What type of deafness results from damage to the cells of the spiral ganglion?
20. Which embryonic germ layer (ectoderm, mesoderm, or endoderm) forms the membranous labyrinth?
21. What brain regions receive input from the vestibular nerves and process information on equilibrium?

For answers, see Appendix B.

THE SPECIAL SENSES THROUGHOUT LIFE

➤ Describe the changes in the special senses that occur with aging.

All special senses are functional, to a greater or lesser degree, at birth. Smell and taste are sharp in newborns, and the likely reason that infants relish food that adults consider bland is that children have more taste buds than adults do. Most



(a) 22 days. Surface ectoderm adjacent to the neural groove thickens to form otic placodes.

(b) 26 days. Invagination of the otic placode forms the otic pit. First branchial groove forms as surface ectoderm invaginates. The endoderm-lined pharynx extends outward, forming the first pharyngeal pouch.

(c) 28 days. Otic pits invaginate further until they pinch off from surface, forming the otic vesicles.

(d) Weeks 5–8.

Internal ear: Membranous labyrinth forms from the otic vesicle; bony labyrinth develops from head mesenchyme.

Middle ear: Middle ear cavity and pharyngotympanic tube form from the first pharyngeal pouch.

External ear: External acoustic meatus develops from first branchial groove.

FIGURE 16.25 Embryonic development of the ear. At left, a surface view of the embryo shows the plane of the sections.

people experience no difficulties with their chemical senses throughout childhood and young adulthood, but starting in the fourth decade of life, the ability to taste and smell declines. This decline reflects a gradual loss of the chemoreceptors, which are replaced more slowly than in younger years.

Even though the fetus cannot see in the darkness of the uterus, the photoreceptors are fully formed in the posterior retina by 25 weeks after conception, and the neuronal connections of the visual pathway have developed even earlier. Visual experiences during the first 8 months after birth fine-tune these synaptic connections.

Congenital problems of the eyes are relatively rare, although maternal rubella (German measles) during the first trimester of pregnancy may cause congenital blindness or cataracts. In newborns, the eyeballs are foreshortened, so all infants are hyperopic (farsighted); only gray tones are perceived,

eye movements are uncoordinated, and often only one eye is used at a time. By 3 months, however, infants can focus an image on the retina's fovea centralis for sharp vision, and babies can follow moving objects with their eyes. By the age of 6 months, depth perception is present, color vision is well developed, and the earlier hyperopia is almost gone.

As a person ages, the lens loses its clarity and becomes discolored, and it begins to scatter light (the resulting glare is why older people should look slightly away from oncoming headlights while driving at night). The dilator muscles of the iris become less efficient, so the pupils stay partly constricted. All these changes decrease the amount of light reaching the retina, such that visual acuity is dramatically lower in people over 70. In addition, elderly individuals are susceptible to conditions that cause blindness, such as glaucoma, retinal detachment, diabetes mellitus, and age-related macular degeneration.

Newborn infants can hear, but their early responses to sounds are mostly reflexive—for example, crying and clenching the eyelids in response to a startling noise. Infants can hear low-pitched and middle-pitched sounds at birth, but the ability to hear high-pitched sounds is a postnatal development. By the third or fourth month, infants can localize sounds and will turn to the voices of family members. By 12 months, babies know all the sounds of their native language, and critical listening begins in toddlers as they learn to speak.

Congenital structural abnormalities of the external ear, including partly or fully missing pinnae and closed or absent auditory canals, are fairly common; less common is sensorineural deafness due to maternal rubella infections during pregnancy. Except for the common ear infections of childhood, few problems usually affect the ears until old age. By about age 60, however, deterioration of the spiral organ becomes noticeable. Humans are born with about 20,000 hair cells in each ear, but they are gradually lost. The ability to hear high-pitched sounds fades first. This gradual loss of hearing with age is called **presbycusis** (pres″bĭ-ku′sis), literally, "old hearing." It is the most common type of sensorineural deafness. Although it is considered a disability of old age, it is becoming more common in younger people as the modern world grows noisier.

In mammals, the hair cells of the internal ear of both hearing and equilibrium do not regenerate naturally. However, gene therapy techniques have induced new hair cell formation. The gene *Math1,* introduced into the inner ear fluid of adult guinea pigs, resulted in the growth of new hair cells. *Math1* encodes for a protein that induces an immature ear cell to become a hair cell. This research could lead to the treatment of many types of hearing loss.

RELATED CLINICAL TERMS

CONGENITAL DEAFNESS One of every 500–1000 children is born deaf because of such physical factors as an immobile stapes, overproduction of perilymph, and various malformations of the middle and internal ear. The ultimate causes of congenital deafness are less well understood, though it can result from maternal mumps, syphilis, or rubella during pregnancy. Many congenital cases are genetically based, and at least four different genes have been identified that, when mutated, cause deafness.

OPHTHALMOLOGY (of″thal-mol′o-je; "eye study") The study of the eye and eye diseases. An *ophthalmologist* is a medical doctor whose specialty is treating eye disorders. By contrast, an *optometrist* is a licensed nonphysician who measures vision and prescribes corrective lenses.

OTITIS EXTERNA (SWIMMER'S EAR) Inflammation and infection of the external acoustic meatus, caused by bacteria or fungi that enter the canal from outside, especially when the canal is moist.

OTORHINOLARYNGOLOGY (o″to-ri″no-lar″ing-gol′o-je) The study of the ear, nose, and larynx and the diseases of these body regions.

SCOTOMA (sko-to′mah; *scoto* = darkness) A blind spot in the visual field other than the normal blind spot caused by the optic disc; often reflects the presence of a brain tumor pressing on nerve fibers along the visual pathway.

TINNITUS (tĭ-ni′-tus) Persistent noise—a ringing, whistling, humming, buzzing, or screeching—that seems to come from the ears in 10% to 20% of all elderly people, causing great distress and annoyance. It often first appears after a loud noise or an injury to the head or cochlea. Recent evidence suggests that tinnitus is analagous to phantom limb pain (see p. 421)—it is a "phantom cochlear noise" caused by destruction of some neurons along the auditory pathway. With the other neurons of this pathway now deprived of their normal input, nearby axons grow in and reinnervate these neurons, and the CNS interprets background signals from the new axons as noise. Treatments include masking the noise with soothing sounds, counseling, and biofeedback; drugs are largely unsuccessful.

CHAPTER SUMMARY

You can use the following media study tool for additional help when you review specific key topics of Chapter 16.

PAL = ***Practice Anatomy Lab***™

The Chemical Senses: Taste and Smell (pp. 483–486)

Taste (Gustation) (p. 483–484)

1. Most taste buds are on the tongue, in the epithelium of fungiform and circumvallate papillae.
2. Taste buds contain gustatory epithelial cells and basal epithelial cells that replace damaged gustatory cells. The gustatory epithelial cells are excited when taste-stimulating chemicals bind to their microvilli.
3. The five basic qualities of taste are sweet, sour, salty, bitter, and umami.

4. The sense of taste is served by cranial nerves VII, IX, and X, which send impulses to the medulla. From there, impulses travel to the thalamus and the taste area of the cerebral cortex.

Smell (Olfaction) (pp. 484–485)

5. The olfactory epithelium is located in the roof of the nasal cavity. This epithelium contains olfactory sensory neurons, supporting epithelial cells, and basal epithelial cells.
6. The olfactory sensory neurons are ciliated bipolar neurons. Odor molecules bind to the cilia, exciting the neurons. Axons of these neurons form the filaments of the olfactory nerve (cranial nerve I).
7. Olfactory nerve axons transmit impulses to the olfactory bulb. Here, these axons synapse with mitral cells in structures called glomeruli.
8. After receiving input from the olfactory sensory neurons, the mitral cells send this olfactory information through the olfactory tract to the olfactory cortex and limbic system.

Disorders of the Chemical Senses (p. 485)

9. Disorders of smell include anosmia (inability to smell) and uncinate fits (smell hallucinations).

Embryonic Development of the Chemical Senses (p. 485–486)

10. The olfactory epithelium and taste buds develop from the epithelia on the face and mouth/pharynx, respectively.

The Eye and Vision (pp. 486–499)

11. The eye is located in the bony orbit and is cushioned by fat. The cone-shaped orbit also contains nerves, vessels, and extrinsic muscles of the eye.

Accessory Structures of the Eye (pp. 486–489)

12. Eyebrows shade and protect the eyes.
13. Eyelids protect and lubricate the eyes by reflexive blinking. Each eyelid contains a supporting tarsal plate, the roots of the eyelashes, and tarsal and ciliary glands. Muscles in the eyelids include the levator palpebrae superioris, which opens the eye, and the orbicularis oculi, which closes the eye.
14. The conjunctiva is a mucosa that covers the inner surface of the eyelids (palpebral conjunctiva) and the white of the eye (bulbar conjunctiva). Its mucus lubricates the eye surface.
15. The lacrimal gland secretes lacrimal fluid (tears), which spreads medially across the eye surface and drains into the nasal cavity through the lacrimal canaliculi, lacrimal sac, and nasolacrimal duct.
16. The six extrinsic eye muscles are the lateral and medial rectus (which turn the eye laterally and medially, respectively); the superior and inferior rectus (which elevate and depress the eye, respectively, but also turn it medially); and the superior and inferior obliques (which depress and elevate the eye, respectively, but also turn it laterally).

Anatomy of the Eyeball (pp. 489–495)

17. The wall of the eye has three layers. The most external, fibrous layer consists of the posterior sclera and the anterior cornea. The tough sclera protects the eye and gives it shape. The cornea is the clear window through which light enters the eye.
18. The middle, pigmented vascular layer consists of the choroid, the ciliary body, and the iris. The choroid provides nutrients to the retina's photoreceptors and prevents the scattering of light within the eye. The ciliary body contains smooth ciliary muscles that control the shape of the lens and ciliary processes that secrete aqueous humor. The iris contains smooth muscle that changes the size of the pupil.
19. The sensory layer contains the retina and the optic nerve. The retina consists of an outer pigmented layer and an inner neural layer. The neural layer contains photoreceptors (rod and cone cells) and other types of neurons. Light influences the photoreceptors, which signal bipolar cells, which signal ganglion cells. The axons of ganglion neurons run along the inner retinal surface toward the optic disc, forming the optic nerve.
20. The outer segments of the rods and cones contain light-absorbing pigment in membrane-covered discs. Light modifies this pigment to initiate the flow of signals through the visual pathway.
21. Two important spots on the posterior retinal wall are (1) the macula lutea with its fovea centralis (area of highest visual acuity) and (2) the optic disc (blind spot), where axons of ganglion cells form the optic nerve.
22. The outer third of the retina (photoreceptors) is nourished by capillaries in the choroid, whereas the inner two-thirds is supplied by the central vessels of the retina.
23. The posterior segment of the eye, posterior to the lens, contains the gel-like vitreous humor. The anterior segment, anterior to the lens, is divided into anterior and posterior chambers by the iris. The anterior segment is filled with aqueous humor, which continually forms at the ciliary processes in the posterior chamber, flows into the anterior chamber, and drains into the scleral venous sinus.
24. The biconvex lens helps to focus light. It is suspended in the eye by the ciliary zonule attached to the ciliary body. Tension in the zonule resists the lens's natural tendency to round up.

The Eye as an Optical Device (pp. 495–496)

25. As it enters the eye, light is bent by the cornea and the lens and focused on the retina. The cornea accounts for most of this refraction, but the lens allows focusing on objects at different distances.
26. The resting eye is set for distance vision. Focusing on near objects requires accommodation (allowing the lens to round as ciliary muscles release tension on the ciliary zonule). The pupils also constrict. Both these actions are controlled by parasympathetic fibers in the oculomotor nerve.
27. Eye-focusing disorders include myopia (nearsightedness), hyperopia (farsightedness), presbyopia (loss of lens elasticity with age).

Visual Pathways (pp. 496–499)

28. The visual pathway to the brain begins with some processing of visual information in the retina. From there, ganglion cell axons carry impulses via the optic nerve, optic chiasma, and optic tract to the lateral geniculate nucleus of the thalamus. Thalamic neurons project to the primary visual cortex.
29. At the optic chiasma, axons from the medial halves of the retinas decussate. This phenomenon provides each visual cortex with information on the opposite half of the visual field as seen by both eyes. The visual cortex compares the views from the two eyes and generates depth perception.

Disorders of the Eye and Vision (p. 499)

30. Three blinding disorders were considered, two that damage the retina (age-related macular degeneration and retinopathy of prematurity) and one that damages the cornea (trachoma).

Embryonic Development of the Eye (p. 499)

31. Each eye starts as an optic vesicle, a lateral outpocketing of the embryonic diencephalon. This vesicle then invaginates to form the optic cup, which becomes the retina. The overlying ectoderm folds to form the lens. The fibrous and vascular layers derive almost entirely from mesenchyme around the optic cups.

The Ear: Hearing and Equilibrium (pp. 499–509)

The External Ear (p. 501)

32. The auricle and external acoustic meatus constitute the external ear, which acts to gather sound waves. The tympanic membrane (eardrum) transmits sound vibrations to the middle ear.

The Middle Ear (pp. 501–502)

33. The middle ear is a small cavity within the temporal bone. Its boundaries are the eardrum laterally, the bony wall of the inner ear medially, a bony roof, a thin bony floor, a posterior wall that opens into the mastoid antrum, and an anterior wall that opens into the pharyngotympanic tube.

34. The pharyngotympanic tube, which consists of bone and cartilage, runs to the pharynx and equalizes air pressure across the eardrum.

35. The auditory ossicles (malleus, incus, and stapes), which help to amplify sound, span the middle ear cavity and transmit sound vibrations from the eardrum to the oval window. The tiny tensor tympani and stapedius muscles dampen the vibrations of very loud sounds.

The Internal Ear (pp. 502–508)

36. The internal ear consists of the bony labyrinth (semicircular canals, vestibule, and cochlea), which is a chamber that contains the membranous labyrinth (semicircular ducts, utricle and saccule, and cochlear duct). The bony labyrinth contains perilymph, whereas the membranous labyrinth contains endolymph.

37. The coiled cochlea is divided into three parts (scalae). Running through its center is the cochlear duct (scala media), which contains the spiral organ. The latter is an epithelium that lies on the basilar membrane and contains the hair cells (receptors for hearing). The other two parts of the cochlea are the scala vestibuli and the scala tympani parts of the bony labyrinth.

38. In the mechanism of hearing, sound vibrations transmitted to the stapes vibrate the fluids in the cochlea. This vibrates the basilar membrane and spiral organ, and in turn bends the hairs of the receptor cells, whose tips are anchored in a nonmoving tectorial membrane. Bending of the hairs produces impulses in the cochlear nerve.

39. The saccule and utricle each contain a macula, a spot of receptor epithelium that monitors static equilibrium and linear acceleration. A macula contains hair cells whose "hairs" are anchored in an overlying otolithic membrane. Forces on the otolithic membrane, caused by gravity and linear acceleration of the head, bend the hairs and initiate impulses in the vestibular nerve.

40. The semicircular ducts lie in three planes of space (anterior vertical, posterior vertical, and lateral horizontal). Their cristae ampullares contain hair cells that monitor rotational acceleration. The "hairs" of these cells are anchored in an overlying cupula. Forces on the cupula, caused by rotational acceleration of the head, bend the hairs and initiate impulses in the vestibular nerve.

Auditory and Equilibrium Pathways (p. 508)

41. Impulses generated by the hearing receptors travel along the cochlear nerve to the cochlear nuclei in the medulla. From there, auditory information passes through several nuclei in the brain stem (superior olivary, inferior colliculus) to the medial geniculate nucleus of the thalamus and cerebral auditory cortex.

42. Impulses generated by the equilibrium receptors travel along the vestibular nerve to the vestibular nuclei and the cerebellum. These brain centers initiate responses that maintain balance. There is also a minor equilibrium pathway to the posterior insula of the cerebral cortex.

Disorders of Equilibrium and Hearing (p. 509)

43. Motion sickness, brought on by particular movements, causes nausea and vomiting. Ménière's syndrome is an overstimulation of the hearing and equilibrium receptors caused by an excess of endolymph in the membranous labyrinth.

44. Conduction deafness results from interference with the conduction of sound vibrations to the internal ear. Sensorineural deafness reflects damage to auditory receptor cells or neural pathways.

Embryonic Development of the Ear (p. 509)

45. The membranous labyrinth develops from the otic placode, a thickening of ectoderm superficial to the hindbrain.

46. A pouch from the pharynx becomes the middle ear cavity and the pharyngotympanic tube. The outer ear is formed by an external branchial groove (external acoustic meatus) and by swellings around this groove (auricle).

The Special Senses Throughout Life (pp. 509–511)

47. The chemical senses are sharpest at birth and decline with age as the replacement of receptor cells slows.

48. The eye is foreshortened (farsighted) at birth. Depth perception, eye coordination, and color vision develop during early childhood. With age, the lens loses elasticity and clarity, and visual acuity declines. Eye problems that may develop with age are presbyopia, cataracts, glaucoma, retinal detachment, and age-related macular degeneration.

49. Initially, infants respond to sound only in a reflexive manner. By 5 months, infants can locate sound. Critical listening develops in toddlers. Obvious age-related loss of hearing (presbycusis) occurs in a person's 60s and 70s.

PAL **Human Cadaver/Nervous System/Special Senses**

REVIEW QUESTIONS

Multiple Choice/Matching Questions

For answers, see Appendix B.

1. Sensory impulses transmitted over the facial, glossopharyngeal, and vagus nerves are involved in the special sense of (a) taste, (b) vision, (c) equilibrium, (d) smell.

2. The part of the fibrous layer of the eye that is white, tough, and opaque is the (a) choroid, (b) cornea, (c) retina, (d) sclera.

3. The transmission of sound vibrations through the internal ear occurs chiefly through (a) nerve fibers, (b) air, (c) fluid, (d) bone.

4. Of the neurons in the retina, which form the optic nerve? (a) bipolar neurons, (b) ganglion neurons, (c) cone cells, (d) horizontal neurons.
5. Blocking the scleral venous sinus might result in (a) a sty, (b) glaucoma, (c) conjunctivitis, (d) Ménière's syndrome, (e) a chalazion.
6. Conduction of sound from the middle ear to the internal ear occurs via vibration of the (a) malleus against the tympanic membrane, (b) stapes in the oval window, (c) incus in the round window, (d) tympanic membrane against the stapes.
7. The structure that allows the air pressure in the middle ear to be equalized with that of the outside air is the (a) cochlear duct, (b) mastoid air cells, (c) endolymph, (d) tympanic membrane, (e) pharyngotympanic tube.
8. The receptors for static equilibrium that report the position of the head in space relative to the pull of gravity are in the (a) spiral organ (of Corti), (b) maculae, (c) crista ampullaris, (d) cupulae, (e) joint kinesthetic receptors.
9. Paralysis of a medial rectus muscle would affect (a) accommodation, (b) refraction, (c) depth perception, (d) pupil constriction.
10. A light ray passes through the refractory structures of the eye in this order: (a) vitreous humor, lens, aqueous humor, cornea; (b) cornea, aqueous humor, lens, vitreous humor; (c) cornea, vitreous humor, lens, aqueous humor; (d) lens, aqueous humor, cornea, vitreous humor.
11. The optic disc is the site where (a) more rods than cones occur, (b) the macula lutea is located, (c) only cones occur, (d) the optic nerve exits the eye.
12. Which of these is not a basic taste sensation? (a) bitter, (b) tsunami, (c) sour, (d) sweet.

Short Answer Essay Questions

13. An anatomy student was arguing with his grandfather. Granddad, who believed in folk wisdom, insisted that there are only five senses. The student, however, said that there are at least ten senses. Decide who was right, and then list all the senses you know. (See Table 12.1, p. 350.)
14. (a) What is the precise location of the olfactory epithelium? (b) Trace the pathway of olfactory stimuli from the olfactory epithelium to the cerebral cortex.
15. What and where is the fovea centralis, and why is it important?
16. Name two special senses whose receptor cells are replaced throughout life, and two special senses whose receptor cells are replaced so slowly that there can be no functional regeneration.
17. (a) Describe the embryonic derivation of the retina. (b) Explain how the middle ear cavity forms.
18. Describe some effects of aging on the eye and the ear.
19. Trace the auditory pathway to the cerebral cortex.
20. Compare and contrast the functions of the inferior oblique and superior rectus muscles.
21. (a) What is the difference, if any, between a semicircular canal and semicircular duct? Between the cochlea and cochlear duct? (b) Name the three parts of the membranous labyrinth of the internal ear. Which of these parts is for hearing, and which are for balance?
22. Describe the function and the innervation of both the sphincter and dilator muscles of the pupil (in the iris of the eye).

CRITICAL REASONING & CLINICAL APPLICATION QUESTIONS

1. Enrique's uncle tells the physician that 3-year-old Enrique gets many "earaches." Upon questioning, the uncle reveals that Enrique has not had a sore throat for a long time and is learning to swim. Does Enrique have otitis media or otitis externa, and does he need ear tubes? Explain your reasoning.
2. Nine children attending the same day-care center developed inflamed eyes and eyelids. What is the most likely cause and name of this condition?
3. Dr. Nakvarati used an instrument to blow a puff of air on Mr. Jefferson's eye during his annual physical examination on his 60th birthday. The eye deformed very little, indicating that the intraocular pressure was too high. What was Mr. Jefferson's probable condition?
4. Lionel suffered a ruptured artery in his middle cranial fossa, and a pool of blood compressed his left optic tract, destroying its axons. What part of the visual field was blinded?
5. Ming, a student in optometry school, felt very sad when she saw some premature babies at the hospital who were born before 25 weeks after conception. She knew that many of these children would soon be blind for life. Explain why.
6. Jan, a senior citizen, developed a constant howling sound in her ear that would not stop. It was very annoying and stressful, and she had to go to counseling to learn how to live with this awful noise. What was her condition called, and what are some other treatments she might receive for it? (See this chapter's Related Clinical Terms.)
7. Describe the effect on vision of a tumor in the hypothalamus or pituitary gland that compresses the optic chiasma, destroying the axons crossing through this structure.