8 The Special Senses

LEARNING OBJECTIVES

Upon completion of this chapter, the student should be able to answer the following questions:

1. What is the dark current, and how does the absorption of a photon change it?
2. What are the synaptic pathways for the central and surround portions of the receptive field of an on-center bipolar cell? Of an off-center bipolar cell?
3. What are the receptive field properties of simple and complex cells in the visual cortex?
4. What is the frequency theory of sound encoding? Why is the place theory also required?
5. What are the stimuli that are normally transduced by the hair cells in the semicircular canals and otolith organs?
6. What are the functional consequences of the differing numbers of different receptor molecules between olfactory and gustatory receptor cells?

The evolution of vertebrates shows a trend called **cephalization** in which special sensory organs develop in the heads of animals, along with the corresponding development of the brain. These special sensory systems, which include the visual, auditory, vestibular, olfactory, and gustatory systems, detect and analyze light, sound, and chemical signals in the environment, as well as signal the position and movement of the head. The stimuli transduced by these systems are most familiar to humans when they provide conscious awareness of the environment, but they are equally important as the sensory basis for reflexive and subconscious behavior.

The Visual System

Vision is one of the most important special senses in humans and, along with audition, is the basis for most human communication. The visual system detects electromagnetic waves between 400 and 750 nm long as **visible light,** which enters the eye and affects **photoreceptors** in a specialized sensory epithelium, the **retina.**

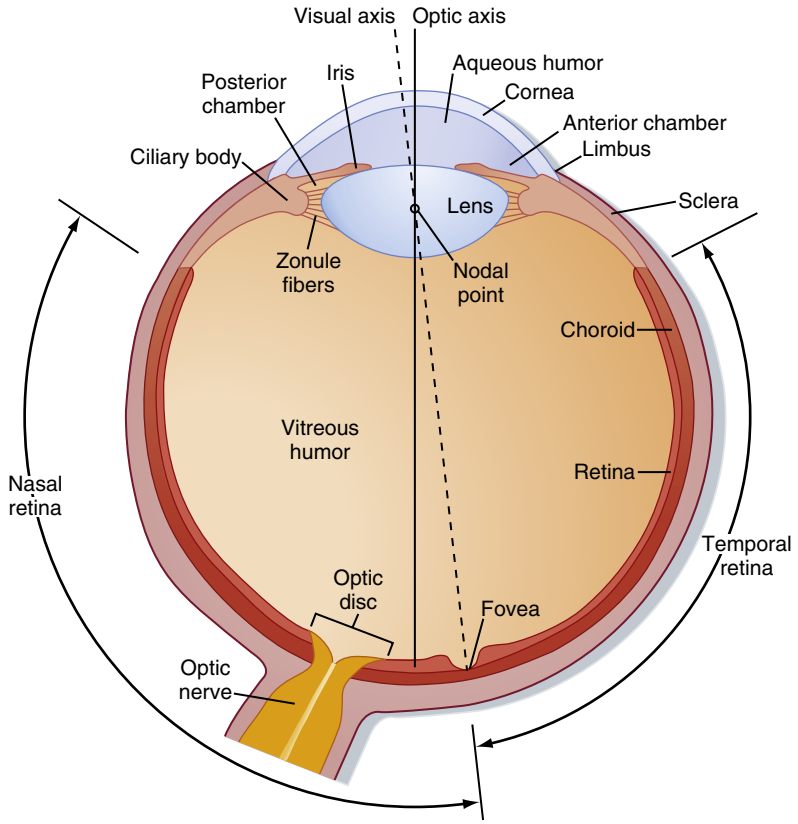
The photoreceptors, rods and cones, can distinguish two aspects of light: its **brightness** (or luminance) and its **wavelength** (or color). **Rods** have high sensitivity for detecting low-light intensities but do not provide well-defined visual images, nor do they contribute to color vision. Rods operate best under conditions of reduced lighting **(scotopic vision).** **Cones,** in contrast, are not as sensitive to light as rods are and thus operate best under daylight conditions **(photopic vision).** Cones are responsible for high visual acuity and color vision.

The retina is an outgrowth of the thalamus. Information processing within the retina is performed by **retinal neurons,** and the output signals are carried to the brain by the axons of **retinal ganglion cells** in the **optic nerves.** There is a partial crossing of these axons in the **optic chiasm** that causes all input from one side of the visual space to pass to the opposite side of the brain. Posterior to the optic chiasm, the axons of retinal ganglion cells form the **optic tracts** and synapse in nuclei of the brain. The main visual pathway in humans targets the **lateral geniculate nucleus (LGN)** of the thalamus and this nucleus, in turn, projects the visual information to the visual cortex. Other visual pathways project to the **superior colliculus, pretectum,** and **hypothalamus,** structures that participate in orientation of the eyes, control of pupil size, and circadian rhythms, respectively.

Structure of the Eye

The wall of the eye is composed of three concentric layers (Fig. 8.1). The outer layer, or the fibrous coat, includes the transparent **cornea,** with its epithelium, and the opaque **sclera.** The middle layer, or vascular coat, includes the iris and the choroid. The **iris** contains both radially and circularly oriented smooth muscle fibers, which make up the pupillary dilator and constrictor muscles, respectively. The **choroid** is rich in blood vessels that support the outer layers of the retina, and it also contains pigment. The innermost layer of the eye, the retina, is embryologically derived from the diencephalon and therefore is part of the central nervous system (CNS). The functional part of the retina covers the entire posterior aspect of the eye except for the optic nerve head, or **optic disc,** which is where the optic nerve axons leave the retina. Because there are no receptors at this location, it is often referred to as the anatomical "blind spot" (see Fig. 8.1).

A number of functions of the eyes are under muscular control. Externally attached extraocular muscles aim the eyes toward an appropriate visual target (see Chapter 9). These muscles are innervated by the **oculomotor nerve (cranial nerve [CN] III),** the **trochlear nerve (CN IV),** and the **abducens nerve (CN VI).** Several muscles are also found within the eye (intraocular muscles). The **muscles in the ciliary body** control lens shape and thereby the focus of



• **Fig. 8.1** Illustration of a view of a horizontal section of the right eye. (Redrawn from Wall GL. *The Vertebrate Eye and Its Adaptive Radiation.* Bloomfield Hills, MI: Cranbrook Institute of Science; 1942.)

images on the retina. The **pupillary dilator** and **sphincter** muscles in the iris control the amount of light entering the eye, in a way similar to that of the diaphragm of a camera. The dilator is activated by the sympathetic nervous system, whereas the sphincter and ciliary muscles are controlled by the parasympathetic nervous system (through the oculomotor nerve; see Chapter 11).

Light enters the eye through the cornea and passes through a series of transparent fluids and structures that are collectively called the **dioptric media.** These fluids and structures consist of the cornea, aqueous humor, lens, and vitreous humor (see Fig. 8.1). The aqueous humor (located in the **anterior** and **posterior chambers**) and the vitreous humor (located in the space behind the lens) help maintain the shape of the eye.

Although the geometrical optic axis of the human eye passes through the nodal point of the lens and reaches the retina at a point between the fovea and the optic disc (see Fig. 8.1), the eyes are oriented by the oculomotor system to a point, called the **fixation point,** on the visual target. Light from the fixation point passes through the nodal point of the lens and is focused on the **fovea.** Light from the remainder of the visual target falls on the retina surrounding the fovea.

Normally, light from a visual target is focused sharply on the retina by the cornea and lens, which bend or refract the light. The cornea is the major refractive element of the eye, with a refractive power of 43 diopters[a] (D). However, unlike the cornea, the lens can change shape and vary its refractive power between 13 and 26 D, thereby giving the lens the ability to adjust optical focus of the eye. **Suspensory ligaments** (or **zonule fibers**) attach to the wall of the eye at the ciliary body (see Fig. 8.1) and hold the lens in place. When the muscles in the ciliary body are relaxed, the tension exerted by the suspensory ligaments flattens the lens. When the ciliary muscles contract, the tension on the suspensory ligaments is reduced; this process allows the somewhat elastic lens to assume a more spherical shape. The ciliary muscles are activated by the parasympathetic nervous system via the oculomotor nerve.

In this way, the lens allows the eye to focus on, or accommodate to, either near or distant objects. For instance, when light from a distant visual target enters a normal eye (one with a relaxed ciliary muscle), the target image is in focus on the retina. However, if the eye is directed at a nearby visual target, the light is initially focused behind the retina (i.e., the image at the retina is blurred) until accommodation occurs; that is, until the ciliary muscle contracts, causing

[a]A diopter is a unit of measurement of optical power that is equal to the reciprocal of the focal length measured in meters. Thus it is a unit of reciprocal length, and a 2-D lens would bring parallel rays of light into focus at a distance of 0.5 m.

the lens to become more spherical, the increased convexity causes the lens to refract the light waves more strongly, bringing the image into focus on the retina.

Proper imaging of light on the retina depends not only on the lens and cornea but also on the iris, which adjusts the amount of light that can enter the eye through the pupil. In this regard, the pupil is analogous to the aperture in a camera, which also controls the depth of field of the image and the amount of spherical aberration produced by the lens. When the pupil is constricted, the depth of field is increased, and the light is directed through the central part of the lens, where spherical aberration is minimal. Pupillary constriction occurs reflexively when the eye accommodates for near vision or adapts to bright light, or both. Thus, when a person reads or does other fine visual work, the quality of the image is improved by adequate light.

IN THE CLINIC

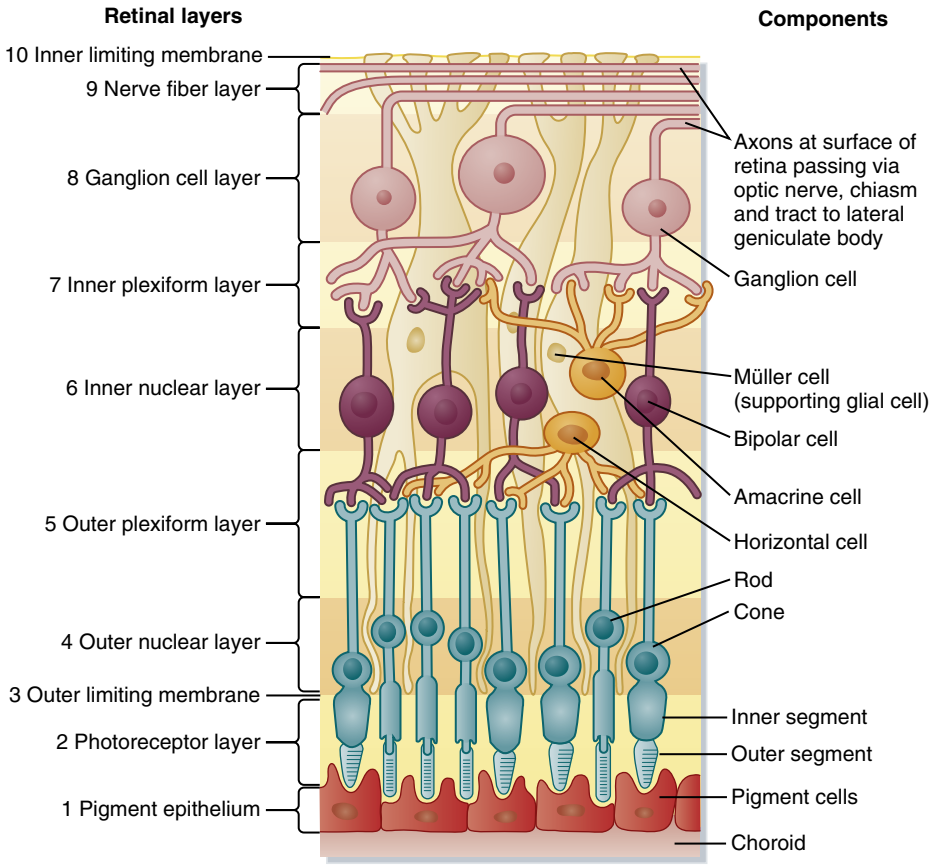
As an individual ages, the elasticity of the lens gradually declines. As a result, accommodation of the lens for near vision becomes progressively less effective, a condition called **presbyopia.** A young person can change the power of the lens by as much as 14 D. However, by the time that a person reaches 40 years of age, the amount of accommodation halves, and after 50 years it can decrease to 2 D or less. Presbyopia can be corrected by convex lenses.

Defects in focus can also be caused by a discrepancy between the size of the eye and the refractive power of the dioptric media. For example, in **myopia** (near-sightedness), the images of distant objects are focused in front of the retina. Concave lenses correct this problem. Conversely, in **hypermetropia** (far-sightedness), the images of distant objects are focused behind the retina; this problem can be corrected with convex lenses. In **astigmatism,** an asymmetry exists in the radii of curvature of different meridians of the cornea or lens (or sometimes of the retina). Astigmatism can often be corrected with lenses that possess complementary radii of curvature.

Retina

Layers of the Retina

The 10 layers of the retina are shown in Fig. 8.2. The outermost portion is the **pigmented epithelium** (layer 1), which is just inside the choroid. The pigment cells have tentacle-like processes that extend into the **photoreceptor layer** (layer 2) and surround the outer segments of the rods and cones. These processes prevent transverse scatter of light between photoreceptors. In addition, they serve a mechanical function in maintaining contact between layers 1 and 2



• **Fig. 8.2** Layers of the retina. Light hitting the retina is coming from the top of the figure and passes through all the superficial layers to reach the photoreceptor rods and cones.

so that the pigmented epithelium can (1) provide nutrients and remove waste from the photoreceptors; (2) phagocytose the ends of the outer segments of the rods, which are continuously shed; and (3) reconvert metabolized visual pigment into a form that can be reused after it is transported back to the photoreceptors.

Retinal glial cells, known as **Müller cells,** play an important role in maintaining the internal geometry of the retina. Müller cells are oriented radially, parallel to the light path through the retina. The outer ends of Müller cells form tight junctions with the inner segments of the photoreceptors, and these numerous connections have the appearance of a continuous layer, the **outer limiting membrane** (layer 3 of the retina).

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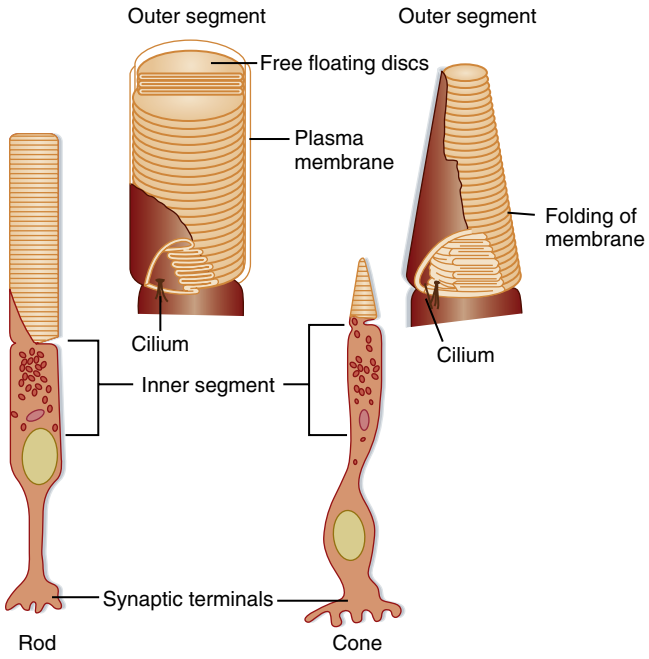
The junction between layers 1 and 2 of the retina in adults represents the surface of contact between the anterior and posterior walls of the embryonic optic cup during development and is structurally weak. Retinal detachment is the separation at this surface and can cause loss of vision, because there is displacement of the retina from the focal plane of the eye. It can also lead to the death of photoreceptor cells, which are maintained by the blood supply of the choroid (the photoreceptor layer itself is avascular). Deterioration of the pigmented epithelium can also result in macular degeneration, a critical loss of high-acuity central and color vision that does not affect peripheral vision.

Inside the external limiting membrane is the **outer nuclear layer** (layer 4) that contains the cell bodies and nuclei of the rods and cones. The **outer plexiform layer** (layer 5) contains synapses between the photoreceptors and retinal interneurons, including bipolar cells and horizontal cells, whose cell bodies are found in the **inner nuclear layer** (layer 6). This layer also contains the cell bodies of other retinal interneurons (the amacrine and interplexiform cells) and the Müller cells.

The **inner plexiform layer** (layer 7) contains synapses between the retinal neurons of the inner nuclear layer, including the bipolar and amacrine cells, and the ganglion cells, whose cell bodies lie in the **ganglion cell layer** (layer 8). As previously mentioned, the ganglion cells are the output cells of the retina; it is their axons that transmit visual information to the brain. These axons form the **optic fiber layer** (layer 9), pass along the inner surface of the retina while avoiding the fovea, and enter the optic disc, where they leave the eye as the optic nerve. The portions of the ganglion cell axons that are in the optic fiber layer remain unmyelinated, but they become myelinated after they reach the optic disc. The lack of myelin where the axons cross the retina helps permit light to pass through the inner retina with minimal distortion.

The innermost layer of the retina is the **inner limiting membrane** (layer 10) formed by the end-feet of Müller cells.

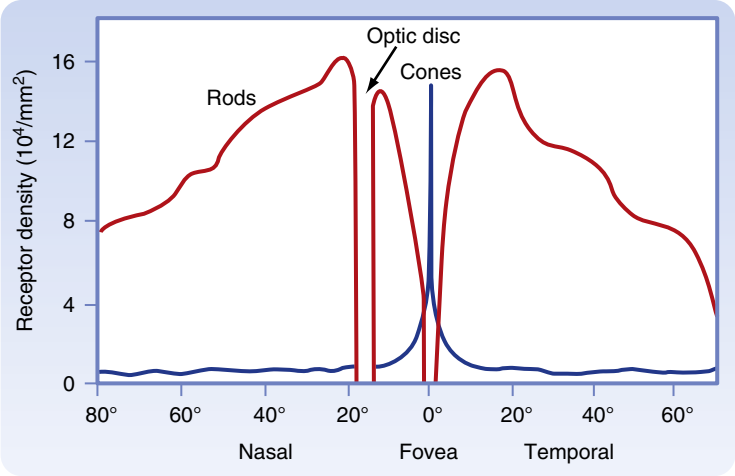


• **Fig. 8.3** Rods and cones. The drawings at the *bottom* show the general features of a rod and a cone. The *insets* show the outer segments.

Structure of Photoreceptors: Rods and Cones

Each rod or cone photoreceptor cell is composed of a cell body (in layer 4), an inner and an outer segment that extend into layer 2, and a set of synaptic terminals that synapse in layer 5 onto other retinal cells (Fig. 8.3). The outer segments of cones are not as long as those of rods, and they contain stacks of disc membranes formed by infoldings of the plasma membrane. The outer segments of rods are longer, and they contain stacks of membrane discs that float freely in the outer segment, having completely disconnected from the plasma membrane when formed at the base. Both sets of discs are rich in visual pigment molecules, but rods have a greater visual pigment density, which partly accounts for their greater sensitivity to light. A single photon can elicit a rod response, whereas several hundred photons may be required for a cone response.

The outer segments of the photoreceptors are connected by a modified cilium to the inner segments, which contain a number of organelles, including numerous mitochondria. The inner segments are the sites where the visual pigment is synthesized before it is incorporated into the membranes of the outer segment. In rods, the pigment is inserted into new membranous discs, which are then displaced distally until they are eventually shed at the apex of the outer segment, where they undergo phagocytosis by cells of the pigmented epithelium. This process determines the rod-like shape of the outer segments of rods. In cones, the visual pigment is inserted randomly into the membranous folds of the outer segment, and shedding, comparable to that seen in rods, does not take place.



• **Fig. 8.4** Graph of a plot of the density of cones and rods as a function of retinal eccentricity from the fovea. Note that cone density peaks at the fovea, rod density peaks at about 20 degrees eccentricity, and no photoreceptors are found at the optic disc, where the ganglion cell axons leave to form the optic nerve. (Data from Cornsweet TN. *Visual Perception.* New York: Academic Press; 1970.)

Regional Variations in the Retina

The **macula lutea** is the area of central vision and is characterized by a slight thickening and a pale color. The thickness is due to the high concentration of photoreceptors and interneurons, which are needed for high-resolution vision. It is pale because both optic nerve fibers and blood vessels are routed around it.

The fovea, which is a depression in the macula lutea, is the region of the retina with the very highest visual resolution and, as noted previously, the light from the fixation point is focused on the fovea. (A major function of eye movements is to bring objects of interest into view on the fovea.) The retinal layers in the foveal region are unusual because several of them appear to be pushed aside into the surrounding macula. Consequently, light can reach the foveal photoreceptors without having to pass through the inner layers of the retina, and both image distortion and light loss are minimized. The fovea has cones with unusually long and thin outer segments, which allows for high packing density. In fact, cone density is maximal in the fovea, providing for high visual resolution, as well as high quality of the image (Fig. 8.4).

The optic disc, where ganglion cell axons leave the retina, lacks photoreceptors and therefore lacks photosensitivity. This area is a so-called blind spot in the visual surface of the retina (see Figs. 8.4 and 8.9). A person is normally unaware of the blind spot because the corresponding part of the visual field can be seen by the contralateral eye and because of the psychological process in which incomplete visual images tend to be completed perceptually.

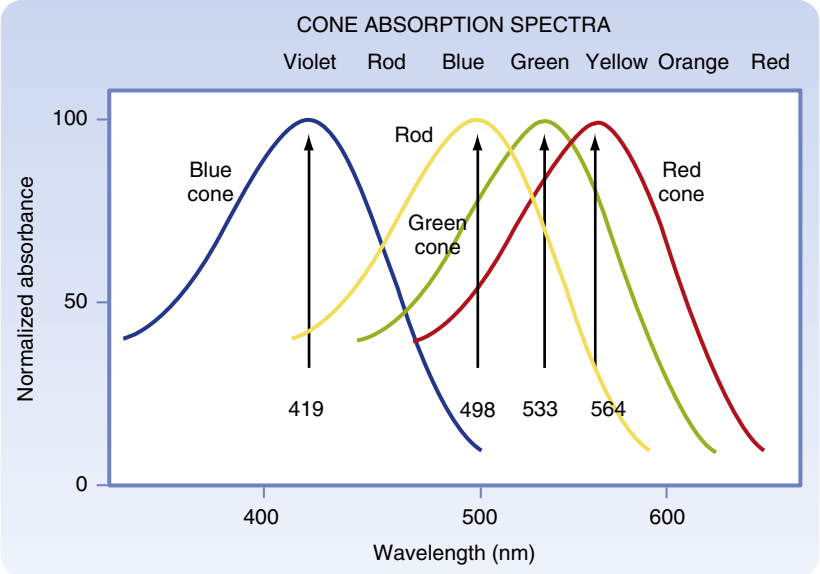
Visual Transduction

To be detected by the retina, light energy must be absorbed. This is primarily the responsibility of the rods and cones (a small class of ganglion cells are also photosensitive), and is primarily accomplished by visual pigment molecules located in their outer segments. For both rods and cones, the pigment molecule consists of a chromophore, 11-*cis* retinal, bound to an opsin protein. The visual pigment found in the outer segments of rods is **rhodopsin,** or visual purple (so named because it has a purple appearance when light has been absorbed). It absorbs light best at a wavelength of 500 nm. Three variants of visual pigment, resulting from the binding of different opsins to retinal, are found in cones (in most species, each cone expresses one of the three cone pigments). The cone pigments absorb best at 419 nm (blue), at 533 nm (green), and at 564 nm (red). However, the absorption spectrum of these visual pigments is broad, so that they overlap considerably (Fig. 8.5).

Despite the differences in spectral sensitivity, the transduction process is similar in rods and cones. The absorption of a photon by a visual pigment molecule leads to the isomerization of 11-*cis* retinal to all-*trans* retinal, release of

IN THE CLINIC

As mentioned, the axons of retinal ganglion cells cross the retina in the optic fiber layer (layer 9) to enter the optic nerve at the optic disc. These axons in the optic fiber layer pass around the macula and fovea, as do the blood vessels that supply the inner layers of the retina. The optic disc can be visualized on physical examination with an **ophthalmoscope.** The normal optic disc has a slight depression in its center. Changes in the appearance of the optic disc are important clinically. For example, the depression may be exaggerated by loss of ganglion cell axons **(optic atrophy),** or the optic disc may protrude into the vitreous space because of edema **(papilledema)** that results from increased intracranial pressure.



• **Fig. 8.5** The spectral sensitivity of the three types of cone pigments and of the rod pigment (Rhodopsin) in the human retina. Note that the curves overlap and that the so-called *blue and red cones* actually absorb maximally in the *violet and yellow ranges*, respectively. (Data from Squire LR, Berg D, Bloom F, du Lac S, Ghosh A, Spitzer N. *Fundamental Neuroscience.* San Diego, CA: Academic Press; 2002.)

the bond with the opsin, and conversion of retinal to retinol. These changes trigger a second-messenger cascade that leads to a change in the electrical activity of the rod or cone (discussed later in this section).

The separation of all-*trans* retinal from opsin also causes both the loss of its ability to absorb light and bleaching (i.e., the visual pigment loses its color). In both rods and cones, regeneration of the visual pigment molecule is a multistep process: the all-*trans* retinal is transported to the retinal pigmented cell layer, where it is reduced to retinol, isomerized, and esterified back to 11-*cis* retinal. It is then transported back to the photoreceptor layer, taken up by outer segments, and recombined with opsin to regenerate the visual pigment molecule, which can again absorb light. There is evidence that cones also use a second pathway to regenerate visual pigment. This pathway is much more rapid and involves transport of the retinal molecule to and from the Müller cells (see Fig. 8.2), rather than the pigmented epithelial cells. The potential importance of this more rapid pathway is discussed later in this chapter in the section "Visual Adaptation."

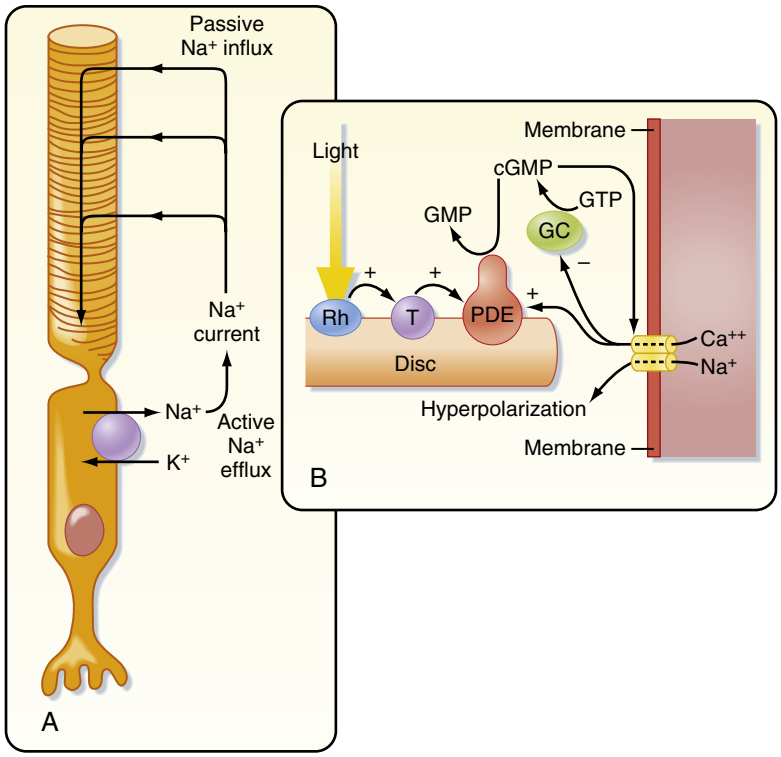
Ultimately, the transduction process triggered by absorption of photons causes the photoreceptor to hyperpolarize. To understand this action and its consequences fully, it is necessary to know the baseline state of the photoreceptor in the dark (i.e., before it absorbs a photon). In darkness, photoreceptors are slightly depolarized (≈–40 mV) in relation to most neurons because cyclic guanosine monophosphate (cGMP)-gated cation channels in their outer segments are open (Fig. 8.6*A*). These channels allow a steady influx of Na^+ and Ca^{++}. The resulting current is known as the **dark current,** and the depolarization it causes leads to the tonic release of the neurotransmitter glutamate at the photoreceptor's synapses.

When light is absorbed in a rod (an equivalent sequence happens in cones), photoisomerization of rhodopsin activates a G protein called **transducin** (see Fig. 8.6*B*). This G protein, in turn, activates **cyclic guanosine monophosphate (cGMP) phosphodiesterase,** which is associated with the rhodopsin-containing discs, hydrolyzes cGMP to 5′-GMP, and lowers the cGMP concentration in the rod cytoplasm. The reduction in cGMP leads to closing of the cGMP-gated cation channels, hyperpolarization of the rod cell membrane, and a reduction in the release of neurotransmitters. Thus, cGMP acts as a "second messenger" to translate the absorption of a photon by rhodopsin into a change in membrane potential.

In sum, in all photoreceptors (cones undergo a process analogous to that described for rod transduction), capture of light energy leads to (1) hyperpolarization of the photoreceptor and (2) a reduction in the release of neurotransmitters. Because of the very short distance between the site of transduction and the synapse, the modulation of neurotransmitter release is accomplished without the generation of an action potential.

Visual Adaptation

Adaptation refers to the ability of the retina to adjust its sensitivity according to ambient light. This ability allows the retina to operate efficiently over a wide range of lighting conditions, and it reflects a switching between the use of the cone and rod systems for bright- and low-light conditions, respectively.



• **Fig. 8.6** **A,** Drawing of a rod with the flow of current in the dark. With the assistance of the Na^+,K^+ pump, the rod is kept depolarized. **B,** Sequence of the second messenger events that follow the absorption of light through the reduction of cGMP. Because cGMP maintains open Na^+ channels in the dark, the results of light absorption are the closing of the Na^+ channels and hyperpolarization of the rod. *cGMP*, Cyclic guanosine monophosphate; *GC*, guanylate cyclase; *GTP*, guanosine triphosphate; *PDE*, phosphodiesterase; *Rh*, rhodopsin; *T*, transducin.

AT THE CELLULAR LEVEL

Rhodopsin contains a chromophore called *retinal,* which is the aldehyde of **retinol,** or vitamin A. Retinol is derived from carotenoids, such as β-carotene, the orange pigment found in carrots. Like other vitamins, retinol cannot be synthesized by humans; instead, it is derived from food sources. Individuals with a severe vitamin A deficiency suffer from "night blindness," a condition in which vision is defective in low-light situations.

The extraordinary sensitivity of rods, which can signal the capture of a single photon, is enhanced by an amplification mechanism in which photoactivation of only one rhodopsin molecule can activate hundreds of transducin molecules. In addition, each phosphodiesterase molecule hydrolyzes thousands of cGMP molecules per second. Similar events occur in cones, but the membrane hyperpolarization occurs much more quickly than in rods and requires thousands of photons.

Light Adaptation

As described previously, absorption of a photon causes 11-*cis* retinal to be converted to all-*trans* retinal, which then splits from the opsin (bleaching). The visual pigments in rods and cones are bleached at a similar rate; however, regeneration of the visual pigment occurs much more rapidly in cones than in rods. This difference is, at least in part, due to the cones' ability to utilize a second pathway for regeneration (see previous section). This more rapid regeneration of visual pigment prevents cones from becoming unresponsive in bright-light conditions. In contrast, the slowness of the regeneration of rhodopsin molecules means that at light levels not much above those found in evening hours, essentially all of the rhodopsin molecules are bleached. Thus, in bright-light conditions, only the cone system is functioning, and the retina is said to be **light-adapted.**

When entering a darkened movie theater, a person can observe evidence of the existing light adaptation (decreased light sensitivity in association with the reduced amount of rhodopsin) in the inability to see the empty seats (or much else). The gradual return of the ability to see the seats while the person remains in the theater reflects the slow regeneration of rhodopsin and recovery of function of the rod system, a process known as **dark adaptation.**

Dark Adaptation

This process refers to the gradual increase in light sensitivity of the retina when in low-light conditions. Rods adapt to darkness slowly as their rhodopsin levels are restored, and indeed, it may take more than 30 minutes for the retina to become fully dark-adapted. In contrast, cones adapt rapidly to darkness, but their adapted threshold is relatively high,

so they do not function when the ambient light level is low. Within 10 minutes in a dark room, rod vision is more sensitive than cone vision and becomes the main system for seeing.

In sum, in the dark-adapted state, primarily rod vision is operative, and thus visual acuity is low and colors are not distinguished (this is called *scotopic vision*). However, when light levels are higher (e.g., when the movie is projected) and cone function resumes (this is called *photopic vision*), visual acuity and color vision are restored. There is an intermediate range of light levels at which rod and cones are both functional *(mesopic vision).*

Color Vision

The visual pigments in the cone outer segments contain different opsins. As a result of these differences, the three types of cones absorb light best at different wavelengths. Although the cone pigments have maximum efficiency closer to violet, green, and yellow wavelengths, they are referred to as blue, green, and red pigments, respectively (see Fig. 8.5). The differences in the cone absorption spectra underlie humans' ability to see colors, as opposed to only shades of gray.

According to the **trichromacy theory,** the differences in absorption efficiency of the cone visual pigments are presumed to account for color vision because a suitable mixture of three colors can produce any other color. However, a neural mechanism must also exist for the analysis of color brightness because the amount of light absorbed by a visual pigment, as well as the subsequent response of the cell, depends on both the wavelength and the intensity of the light (see Fig. 8.5). Two or three of the cone pigments may absorb a particular wavelength of light, but the amount absorbed by each differs according to its efficiency at that wavelength. If the intensity of the light is increased (or decreased), all will absorb more (or less), but the ratio of absorption among them will remain constant. Consequently, there must be a neural mechanism to compare the absorption of light of different wavelengths by the different types of cones for the visual system to distinguish different colors. At least two different kinds of cones are required for color vision. The presence of three kinds decreases the ambiguity in distinguishing colors when all three absorb light, and it ensures that at least two types of cones will absorb most wavelengths of visible light.

The **opponent process theory** is based on observations that certain pairs of colors seem to activate opposing neural processes. Green and red are opposed, as are yellow and blue, as well as black and white. For example, if a gray area is surrounded by a green ring, the gray area appears to acquire a reddish color. Furthermore, a greenish red or a bluish yellow color does not exist. These observations are supported by findings that neurons activated by green wavelengths are inhibited by red wavelengths. Similarly, neurons excited by blue wavelengths may be inhibited by yellow wavelengths. Neurons with these characteristics are present both in the retina and at higher levels of the visual pathway and seem to serve to increase the ability to see the contrast between opposing colors.

Retinal Circuitry

A diagram of the basic circuitry of the retina is shown in Fig. 8.7. Several features of this circuitry are noteworthy: (1) Input to the retina is provided by light striking the photoreceptors. (2) The output is carried by axons of the retinal ganglion cells to the brain. (3) Information is processed within the retina by the interneurons. (4) The most direct pathway through the retina is from a photoreceptor to a bipolar cell and then to a ganglion cell (see Fig. 8.7). (5) More indirect pathways that provide for intraretinal signal processing involve photoreceptors, bipolar cells, amacrine cells, and ganglion cells, as well as horizontal cells to provide lateral interactions between adjacent pathways.

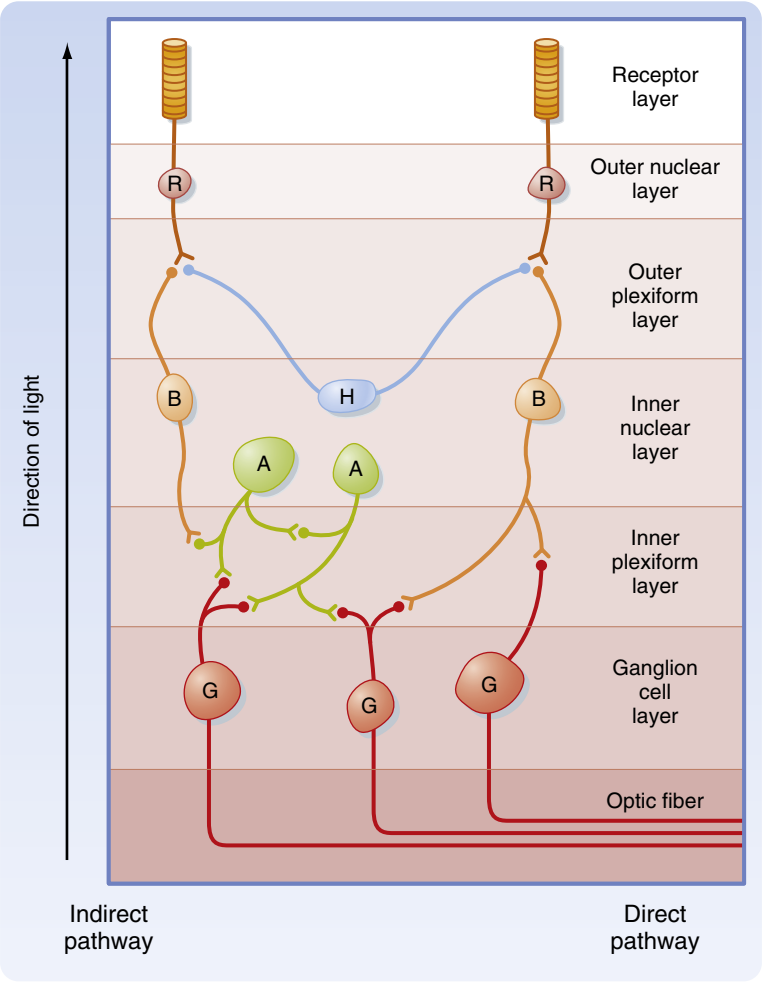
IN THE CLINIC

Observations of color blindness are consistent with the trichromacy theory. In color blindness, a genetic defect (sex-linked recessive), one or more cone mechanisms are lost. People with normal color vision are trichromats because they have three cone mechanisms. Individuals who lack one of the cone mechanisms are called dichromats. When the long-wavelength cone mechanism is absent, the resulting condition is called protanopia; absence of the medium-wavelength system causes deuteranopia; and absence of the short-wavelength system causes tritanopia. Monochromats lack two or more cone mechanisms.

Contrasts in Rod and Cone Pathway Functions

Rod and cone pathways have several important functional differences in their phototransduction mechanisms and their retinal circuitry. As described previously, rods have more visual pigment and a better signal amplification system than cones do, and there are many more rods than cones. As a consequence, rods function better in dim light (scotopic vision), and loss of rod function results in night blindness. In addition, all rods contain the same visual pigment, so they cannot signal color differences. Furthermore, many rods converge onto individual bipolar cells and the results are very large receptive fields and low spatial resolution. Finally, in bright light, most rhodopsin is bleached, so that rods no longer function under photopic conditions.

Cones have a higher threshold to light and thus are not activated in dim light after dark adaptation. However, they operate very well in daylight. They provide high-resolution vision because only a few cones converge onto individual bipolar cells in cone pathways. Moreover, no convergence occurs in the fovea, where the cones make one-to-one connections to bipolar cells. As a result of the reduced convergence, cone pathways have very small receptive fields and can resolve stimuli that originate from sources very close to each other. Cones also respond to sequential stimuli with good temporal resolution. Finally, cones have three different visual pigments and therefore provide for color vision. Loss



• **Fig. 8.7** Basic retinal circuitry. The *arrow* at the *left* indicates the direction of light through the retina. Photoreceptors *(R)* synapse on the dendrites of bipolar cells *(B)* and horizontal cells *(H)* in the outer plexiform layer. The horizontal cells make reciprocal synaptic connections with photoreceptor cells and are electrically coupled to other horizontal cells. Bipolar cells reach synapse on the dendrites of ganglion cells *(G)* and on the processes of amacrine cells *(A)* in the inner plexiform layer. Amacrine cells connect with ganglion cells and other amacrine cells.

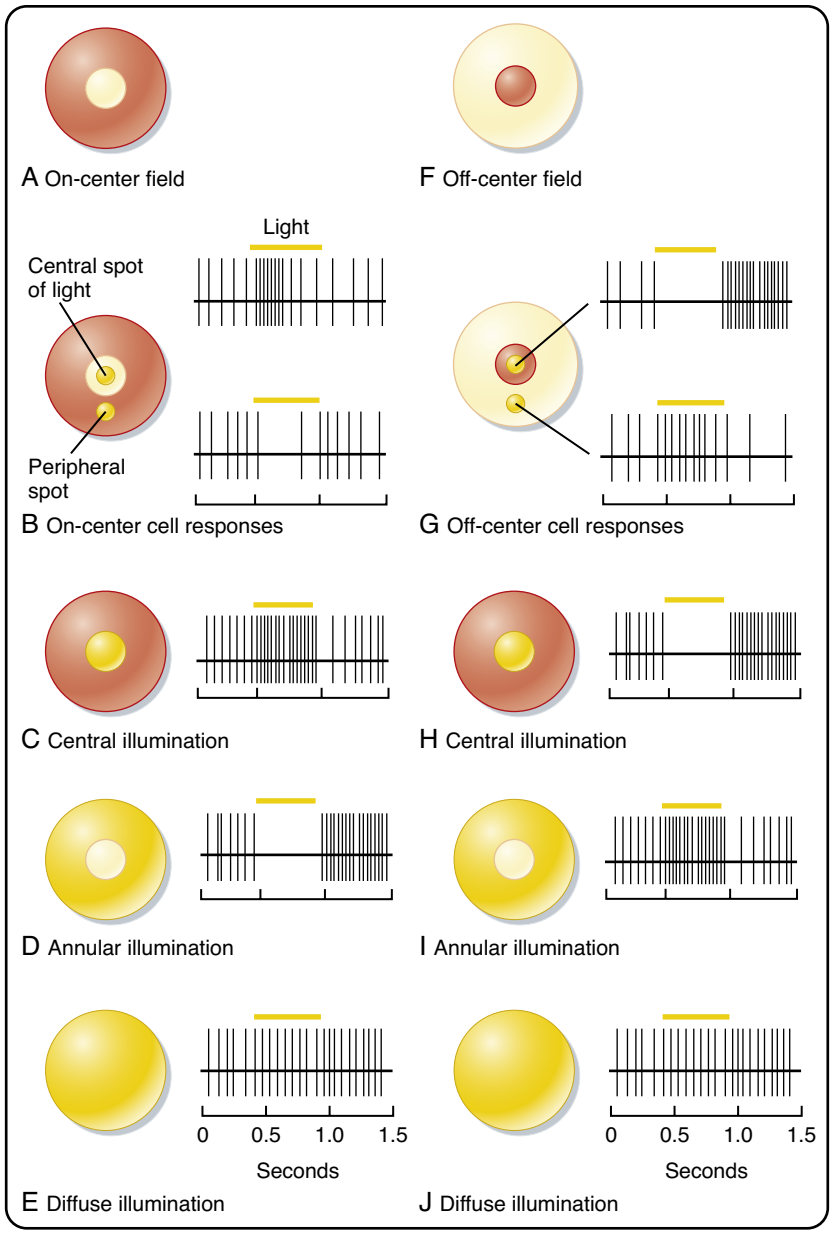
of cone function results in functional blindness; rod vision is not sufficient for normal visual requirements.

Synaptic Interactions and Receptive Field Organization

The receptive field of an individual photoreceptor is circular. Light in the receptive field hyperpolarizes the photoreceptor cell and causes it to release less neurotransmitter. The receptive fields of photoreceptors and retinal interneurons determine the receptive fields of the retinal ganglion cells onto which their activity converges. The characteristics of the receptive fields of retinal ganglion cells constitute an important step in visual information processing because all the information about visual events that is conveyed to the brain is contained in ganglion cell activity.

The bipolar cell, which receives input from a photoreceptor, can have either of two types of receptive fields, as shown in Fig. 8.8. Both are described as having a center-surround organization in which the light that strikes the central region of the receptive field either excites or inhibits the cell, whereas the light that strikes a region that surrounds the central portion has the converse effect. The receptive field with a centrally located excitatory region surrounded by an inhibitory annulus is called an **on-center, off-surround receptive field** (see Fig. 8.8*A*). Bipolar cells with such a receptive field are described as "on" bipolar cells. The other type of receptive field has an **off-center, on-surround** arrangement, which characterizes "off" bipolar cells (see Fig. 8.8*F*).

The center response of a bipolar cell receptive field is due to only the photoreceptors that directly synapse with the bipolar cell. Photoreceptor cells respond to light with hyperpolarization and a decrease in glutamate release and respond to the removal of light with depolarization and increased glutamate release. This implies that the difference in the center responses of "on" and "off" bipolar cells lies in their response to glutamate. In fact, off-center bipolar



• **Fig. 8.8** The receptive fields of on-center **(A)** and off-center **(F)** bipolar cells and, below them, the receptive fields of ganglion cells **B** through **E** and **G** through **J** to which they are connected. Ganglion cell responses to central spots (*upper recording*) and peripheral spots (*lower recording*) are shown in **B** and **G.** Also shown are responses to central (**C** and **H**), surround (**D** and **G**), and diffuse whole-field (**E** and **J**) illumination in their receptive fields. The ganglion cells and the on-center and off-center bipolar cells providing input to these ganglion cells have similar receptive fields, whereas ganglion cells increase or decrease their spike frequency, bipolar cells depolarize or hyperpolarize, without generating action potentials. (Redrawn from Squire LR, Berg D, Bloom F, du Lac S, Ghosh A, Spitzer N. *Fundamental Neuroscience.* San Diego, CA: Academic Press; 2002.)

cells have ionotropic glutamate receptor channels that open in response to glutamate, and thus they are excited by the removal of light stimuli from the center of their receptive field. In contrast, on-center bipolar cells have metabotropic glutamate receptors that close their channels in response to glutamate. They are depolarized by light on the center of their receptive field, because the reduced release of glutamate by the photoreceptors results in more open metabotropic channels. Thus, on-center bipolar cells are excited by light stimulation of the center of their receptive fields.

The antagonistic surround response of bipolar cells is due to photoreceptors that surround those that synapse directly on them. These photoreceptors (which also connect directly with their own bipolar cells) synapse with horizontal cells

that participate in complex triadic synapses with many photoreceptors and bipolar cells. The pathway through the horizontal cells results in a response that is opposite in sign to that produced directly by the photoreceptors that mediate the center response. The reason for this is that horizontal cells are depolarized by glutamate released from photoreceptors and thus, like "off" bipolar cells, are hyperpolarized in the light. Moreover, because they are electrically coupled to each other by gap junctions, they have very large receptive fields. Darkness in the periphery of a bipolar cell's receptive field (such as an annulus that does not affect the photoreceptors to which it is directly connected) causes neighboring photoreceptors and horizontal cells to depolarize. The depolarized horizontal cells release GABA onto central (and peripheral) photoreceptor terminals, reducing their release of glutamate. When darkness surrounds central illumination, there is increased excitation of **on-center** bipolar cells. There is a complementary effect on **off-center** bipolar cells when a bright annulus surrounds a central dark spot (see Fig. 8.8).

Bipolar cells may not respond to large or diffuse areas of illumination, covering both the receptors that are responsible for the center response and those that cause the surround response because of their opposing actions. Thus, bipolar cells may not signal changes in the intensity of light that strikes a large area of the retina. On the other hand, a small spot of light moving across the receptive field may sequentially alter the activity of the bipolar cell as the light crosses the receptive field from the surround portion to the center and then back again to the surround portion. This demonstrates that bipolar cells respond best to the local contrast of stimuli and function as contrast detectors.

Amacrine cells receive input from different combinations of on-center and off-center bipolar cells, so that Thus their receptive fields are mixtures of on-center and off-center regions. There are many different types of amacrine cells, and they may use at least eight different neurotransmitters. Accordingly, the contributions of amacrine cells to visual processing are complex.

Ganglion cells may receive dominant input from bipolar cells, dominant input from amacrine cells, or mixed input from amacrine and bipolar cells. When amacrine cell input dominates, the receptive fields of ganglion cells tend to be diffuse, and they are either excitatory or inhibitory. Most ganglion cells, however, are dominated by bipolar cell input and have a center-surround organization, similar to that of the bipolar cells that connect to them (see Fig. 8.8).

The distances between retinal components are short. Hence, modulation of transmitter release by changes in transmembrane potential and the resulting postsynaptic potentials are sufficient for most of the activity in retinal circuits, and action potentials are not required except for ganglion cells and some amacrine cells, which generate action potentials. It is unclear why amacrine cells have action potentials, but ganglion cells must generate them to transmit information over the relatively long distance from the retina to the brain.

P, M, and W Cells

Experiments have shown that in primates, retinal ganglion cells can be subdivided into three general types called **P cells, M cells,** and **W cells.** P and M cells are fairly homogeneous groups, whereas W cells are heterogeneous. P cells are so named because they project to the parvocellular layers of the LGN of the thalamus, whereas M cells project to the magnocellular layers of the LGN. P and M cells have center-surround receptive fields, consistent with being controlled by bipolar cells. W cells have large, diffuse receptive fields and slowly conducting axons. They are probably influenced chiefly through amacrine cell pathways, but less is known about them than about M and P cells.

Several of the physiological differences among these cell types correspond to morphological differences (Table 8.1). For example, P cells have small receptive fields (which corresponds to smaller dendritic trees) and more slowly conducting axons than M cells. In addition, P cells show a linear response in their receptive field; that is, they respond with a sustained, tonic discharge of action potentials in response to maintained light, but do not signal shifts in the pattern

TABLE 8.1 Properties of Retinal Ganglion Cells

Properties	P Cells	M Cells	W Cells
Cell body and axon	Medium sized	Large	Small
Dendritic tree	Restricted	Extensive	Extensive
Receptive field			
Size	Small	Medium	Large
Organization	Center-surround	Center-surround	Diffuse Poorly responsive
Adaptation	Tonic	Phasic	
Linearity	Linear	Nonlinear	
Wavelength	Sensitive	Insensitive	Insensitive
Luminance	Insensitive	Sensitive	Sensitive

of illumination as long as the overall level of illumination is constant. Thus, a small object entering a P cell's central receptive field will change the cell's firing, but continued object movement within the field will not be signaled. P cells respond differently to different wavelengths of light. Because there are blue, green, and red cones, many combinations of color properties are possible, but in fact, P cells have been shown to have opposing responses only to red and green or only to blue and yellow (a combination of red and green). These mechanisms can greatly reduce the ambiguity of color detection caused by the overlap in cone color sensitivity and may provide a substrate for the opponency process observations.

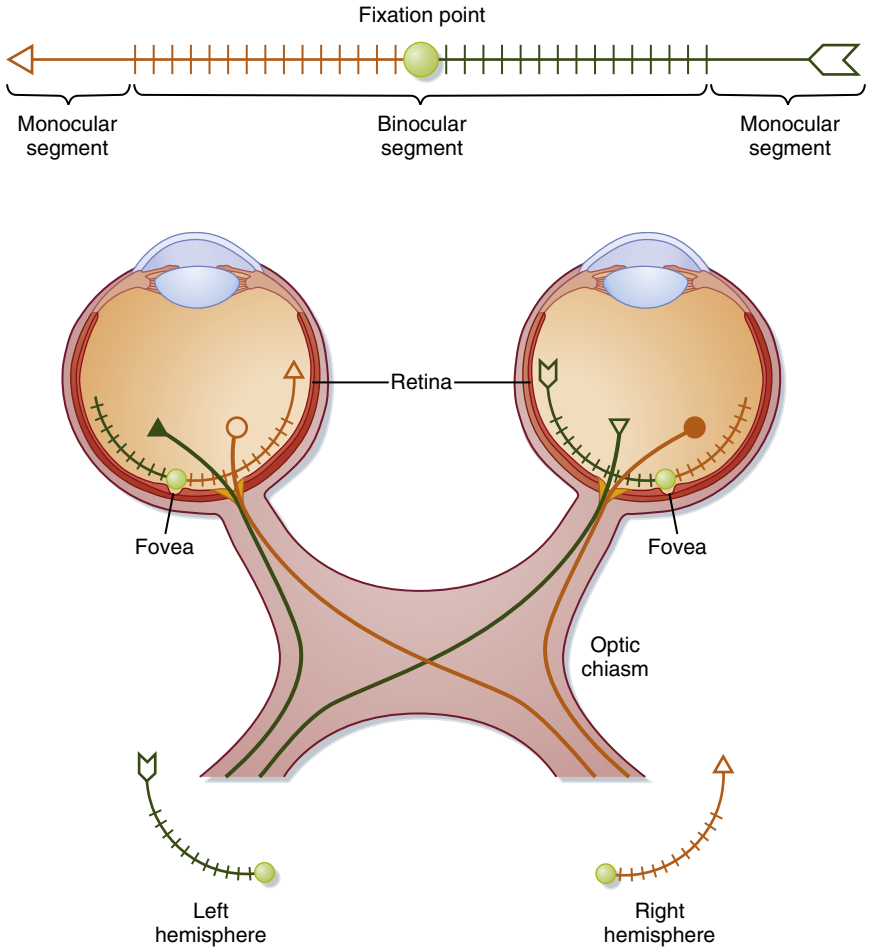
M cells, on the other hand, respond with phasic bursts of action potentials to the redistribution of light, such as would be caused by the movement of an object within their large receptive fields. M cells are not sensitive to differences in wavelength, but are more sensitive to luminance than P cells are.

In summary, the output of the retina consists primarily of ganglion cell axons from (1) sustained, linear P cells with small receptive fields that convey information about color, form, and fine details and (2) phasic, nonlinear M cells with larger receptive fields that convey information about illumination and movement. Both exist in on-center and off-center varieties (Fig. 8.8).

The Visual Pathway

Retinal ganglion cells transmit information to the brain by way of the optic nerve, optic chiasm, and optic tract. Fig. 8.9 shows the relationships among a visual target, the retinal images of the target in the two eyes, and the projections of retinal ganglion cells to the two hemispheres of the brain. The eyes and the optic nerves, chiasm, and tract are viewed from above.

The visual target, an arrow, is in the visual fields of both eyes (see Fig. 8.9) and, in this case, is so long that it extends into the monocular segments of each retina (i.e., one end of the target can be seen by only one eye and the other end by only the other eye). The shaded circle at the center of the target represents the fixation point. The image of the target on



• **Fig. 8.9** Relationships among a visual target (*long arrow,* **top**), images on the retinas of the two eyes **(middle),** and projections of the ganglion cells carrying visual information about these images **(bottom).** The target image is so large that it extends into the monocular segments of the eyes, where one side of it is seen by only the ipsilateral eye. Note how the axons are sorted in the chiasm so that all information about the left visual field of both eyes is conveyed to the right side of the brain and all information about the right visual field is conveyed to the left side.

the retinas is reversed by the lens system. The left half of the visual target is imaged on the nasal retina of the left eye and the temporal retina of the right eye; the left visual field is seen by the left nasal retina and the right temporal retina. Similarly, the right half of the visual target is imaged on and seen by the left temporal retina and the right nasal retina. The lens system also causes an inversion in the vertical axis, with the upper visual field imaged on the lower retina and vice versa.

The axons of retinal ganglion cells may or may not cross in the optic chiasm, depending on the location of the ganglion cell in the retina (see Fig. 8.9). Axons from the temporal portion of each retina pass through the optic nerve, the lateral side of the optic chiasm, and the ipsilateral optic tract and terminate ipsilaterally in the brain. Axons from the nasal portion of each retina pass through the optic nerve, cross to the opposite side in the optic chiasm, and then pass through the contralateral optic tract to end in the contralateral side of the brain. As a result of this arrangement, objects in the left field of vision are represented in the right side of the brain, and those in the right field of vision are represented in the left side of the brain.

Lateral Geniculate Nucleus

Retinal ganglion cell axons can synapse in several parts of the brain, but the main target for vision is the **lateral geniculate nucleus (LGN)** of the thalamus. There is a point-to-point projection from the retina to the LGN, resulting in a retinotopic map. Cells that represent a particular retinal location are aligned along projection lines that can be drawn across the layers of the LGN.

The projection from each eye is distributed to three of the layers of the LGN, one of the magnocellular layers (layers 1 and 2 receive M cell input) and two of the parvocellular layers (layers 3–6 receive P cell input). Color-coded ganglion cells project to groups of cells between the major layers, the intralaminar zones. The properties of LGN neurons are very similar to those of retinal ganglion cells; for example, LGN neurons can be classified as P or M cells, and they have on-center or off-center receptive fields.

The LGN also receives input from the visual areas of the cerebral cortex, the thalamic reticular nucleus, and several nuclei of the brainstem reticular formation. The activity of LGN projection neurons is inhibited by interneurons both in the LGN and in the thalamic reticular nucleus. These cells use GABA as their inhibitory neurotransmitter. In addition, the activity of LGN neurons is influenced by corticofugal pathways and by brainstem neurons that transmit signals via monoamine neurotransmitters. These control systems filter visual information and may be important for selective attention.

Striate Cortex

The LGN projects to the **primary visual cortex** or **striate cortex** by way of the **visual radiations.** The visual radiation fibers carrying information derived from the lower half of the appropriate hemiretinas (and therefore the contralateral upper visual field) project to the **lingual gyrus,** which lies on the medial surface of the occipital lobe, just below the calcarine sulcus. Axons in the visual radiation that represent the contralateral lower visual field project to the adjacent **cuneus gyrus,** which lies just above the calcarine sulcus. Together, the portions of these two gyri that line and border the calcarine sulcus constitute the primary visual cortex (or Brodmann area 17; Fig. 8.10).

Like the LGN, the striate cortex contains a retinotopic map. The representation of the macula occupies the most posterior and largest part of both gyri, and progressively more peripheral areas of the retina are projected to more anterior parts of these gyri. Overall, there is an orderly mapping of retinal loci across the surface of the striate cortex (see Fig. 8.10).

The geniculostriate pathway ends chiefly in layer 4 of the striate cortex (Fig. 8.11), whereas the projection from the intralaminar LGN terminates in so-called blobs in layers 2 and 3. Similarly, axons that represent one eye or the other terminate within layer 4C in alternate adjacent patches that define **ocular dominance columns.** Cortical neurons in such a column respond preferentially to input from one eye. Near the border between two ocular dominance columns, neurons respond about equally to input from the two eyes.

The receptive fields of neurons in the striate cortex, aside from the monocular cells in layer 4C, are more complex than those of LGN neurons. Neurons in other layers may be binocular and respond to stimulation of both eyes, although the input from one eye often dominates (see Chapter 10). In addition, cortical neurons outside layer 4C often show **orientation selectivity** (i.e., they respond best when the stimulus, such as a bar or an edge, is oriented and positioned in a particular way; Fig. 8.12). These "simple cells" appear to be responding as though they received input from cells whose concentric center-surround receptive fields were arranged in such a way that their "on" centers were aligned in a row flanked by antagonistic regions. "Complex" cortical neurons are similar to "simple" cells in that they are orientation specific, but instead of having

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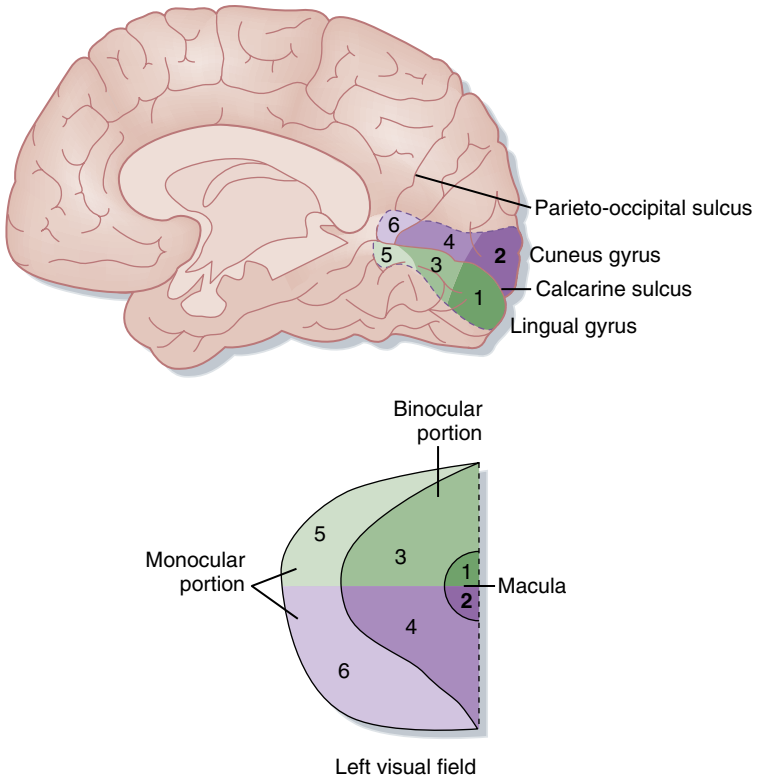
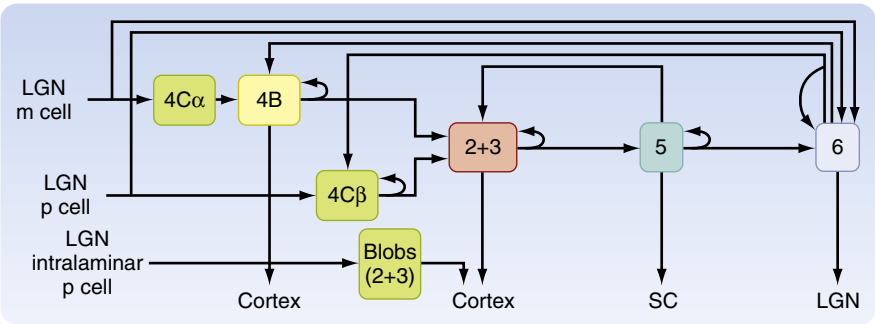
Interruption of the visual pathway at any level causes a defect in the appropriate part of the visual field (see Fig. 8.9). For example, a tiny lesion in the retina would result in a blind spot **(scotoma)** in that eye, whereas a similar lesion in the striate cortex would produce corresponding scotomas in both eyes. Interruption of the optic nerve on one side produces blindness in that eye. Damage to the optic nerve fibers as they cross in the optic chiasm results in loss of vision in both temporal fields of vision; this condition is known as **bitemporal hemianopsia** and occurs because the crossing fibers originate from ganglion cells in the nasal halves of each retina. A lesion of the entire optic tract, LGN, visual radiation, or visual cortex on one side causes **homonymous hemianopsia,** which is loss of vision in the entire contralateral visual field. Partial lesions result in partial visual field defects. For example, a lesion in the lingual gyrus causes an upper **homonymous quadrantanopsia,** which in this case is loss of vision in the contralateral, upper visual field.



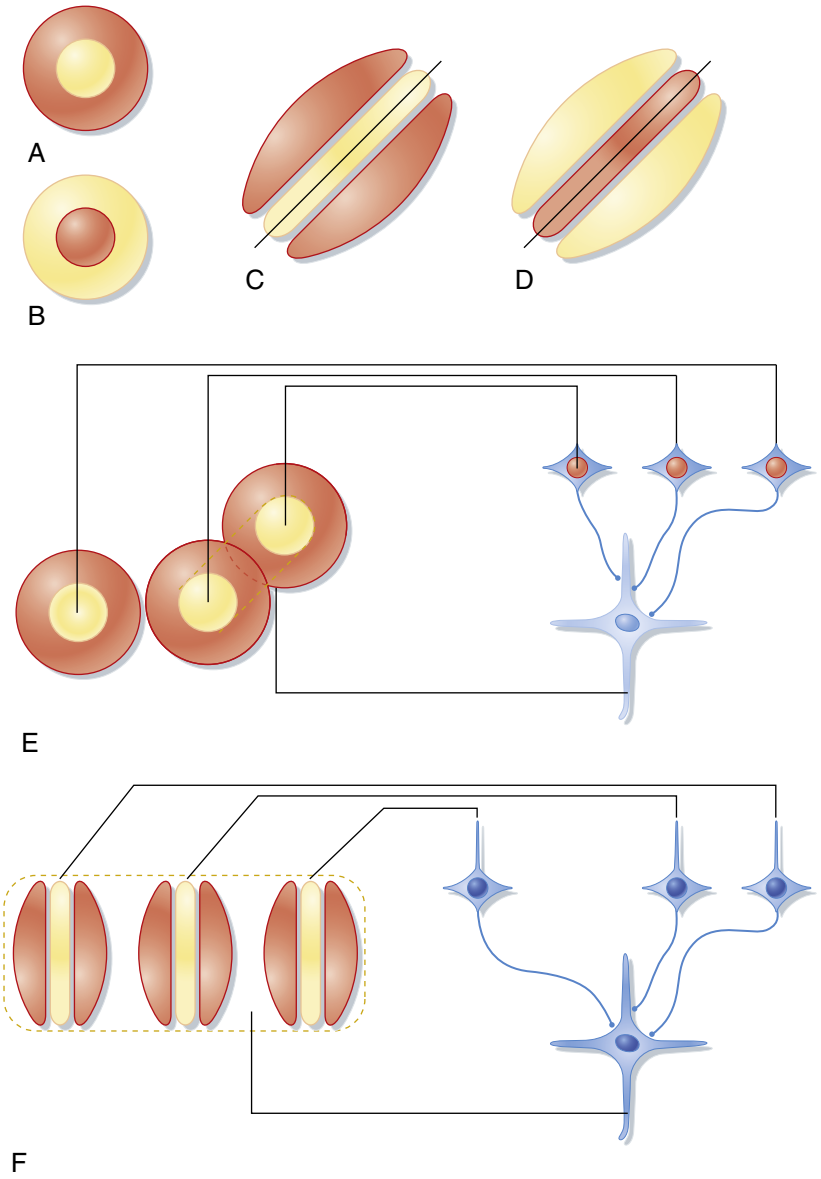
• **Fig. 8.10** The left visual field is relayed (via the lateral geniculate nucleus [LGN] and visual radiation) to the primary visual cortex of the right hemisphere, as a point-to-point retinotopic map. The representation of each part of visual space is proportional to the number of afferent axons with receptive fields in that part of space. As a result, the area of macular representation (near the occipital pole) is larger than that for the rest of the binocular and monocular fields. Note that the lower half of the field is represented in the cuneus gyrus above the calcarine sulcus and the upper half of the field in the lingual gyrus below the sulcus. (Redrawn from Purves D, Augustine G, Fitzpatrick D, et al. *Neuroscience.* 3rd ed. Sunderland, MA: Sinauer; 2004.)



• **Fig. 8.11** Diagram of visual information flow into the visual cortex from the lateral geniculate nucleus *(LGN)* and its projection to the extrastriate cortex, to the superior colliculus *(SC)*, and back to the LGN. *M*, Magnocellular path; *P*, parvocellular path. (Redrawn from Squire LR, Berg D, Bloom F, du Lac S, Ghosh A, Spitzer N. *Fundamental Neuroscience.* San Diego, CA: Academic Press; 2002.)

flanking excitatory and inhibitory zones, they respond best to a particular stimulus orientation anywhere in their receptive field. They may also display **direction selectivity;** that is, they may respond when the stimulus is moved in one direction but not when it is moved in the opposite direction (see Fig. 8.12). The receptive field of a "complex" cell may be thought of as a composite of adjacent "simple" cells with similar orientation selectivity. Because such neurons in a particular zone of the cortex all tend to have the same orientation selectivity, they are considered to form an **orientation column** (Fig. 8.13).

As already discussed, color vision may depend on the presence in the retina of three different types of cones, as well as neurons in the visual pathway that show spectral opposition. Retinal ganglion cells, LGN neurons, and some P cells display spectral opponent properties. The spectral opponent neurons in the striate cortex are found in cortical blobs, and these show double-opponency, in which both the center and

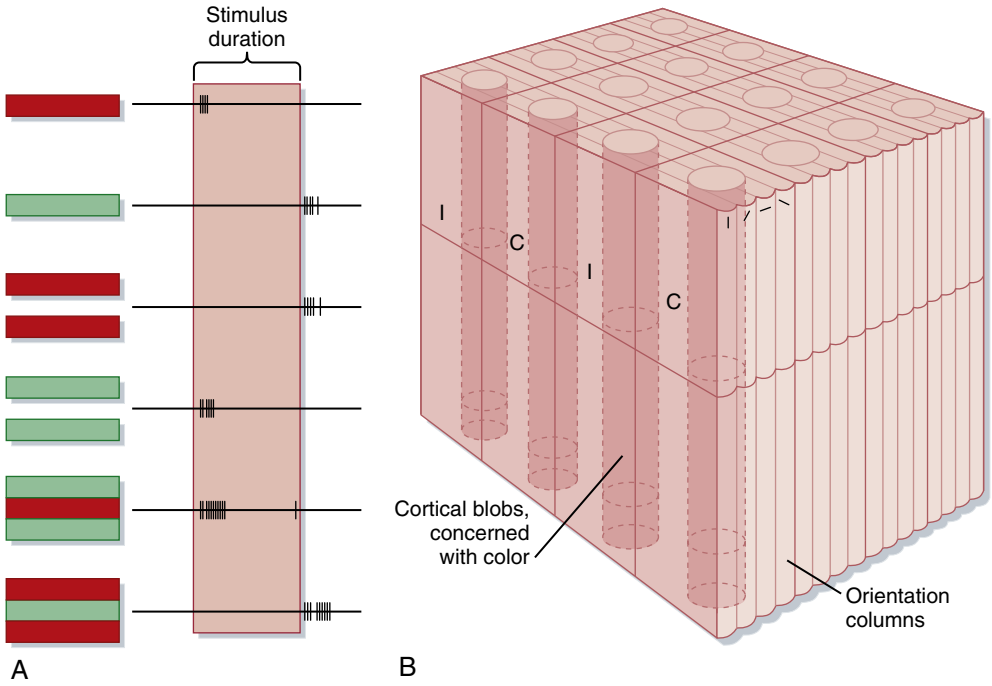


• **Fig. 8.12** Simple and complex receptive fields in the visual cortex can be generated from multiple inputs with concentric fields. **A** and **B** represent on-center and off-center input, respectively, from the retina. If three on-center cells **(A)** with adjacent receptive fields converge onto one cortical neuron **(E),** that neuron, a simple cell, responds best to a long bar stimulus at a specific location and orientation **(C).** For three off-center inputs **(B),** the resulting receptive field is shown in **D.** The convergence of multiple simple cells onto another cortical neuron **(F)** result in a complex cell that responds best to a bar stimulus with a vertical orientation that can be placed anywhere within its receptive field. (Redrawn from Squire LR, Berg D, Bloom F, du Lac S, Ghosh A, Spitzer N. *Fundamental Neuroscience.* San Diego, CA: Academic Press; 2002.)

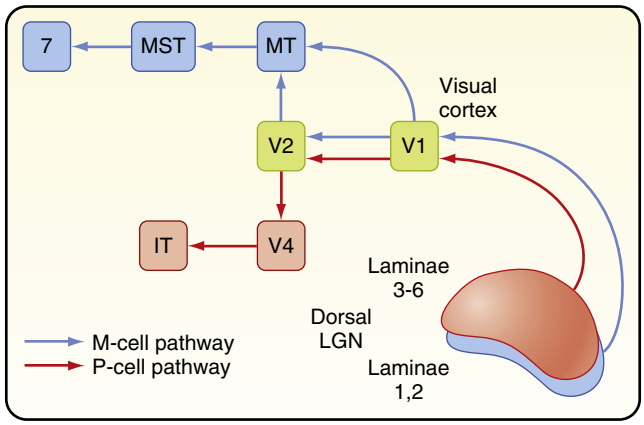
the surround portions respond antagonistically to two colors. Such a cell, whose center responds to red but not green (R^+G^-) and whose surround portion responds to green but not red (R^-G^+), is shown in Fig. 8.13*A*. The relationships between the ocular dominance and orientation columns and the cortical color blobs are shown in Fig. 8.13*B*.

Extrastriate Visual Cortex

In animal studies, at least 25 different visual areas have been identified in the cerebral cortex, in addition to the striate cortex (Brodmann area 17, or V1). The extrastriate areas include several parallel pathways of visual processing. The P pathway originates with P cells and functions in the recognition of form and color. Structures in the P pathway include LGN layers 3 to 6, layer 4Cβ of the striate cortex, V4 (Brodmann area 19), and several areas in the inferotemporal region (Fig. 8.14). Processing of form includes recognition of complex visual patterns, such as faces. Color information is processed separately from form. The M pathway originates with M cells and functions in motion detection and control of eye movement. Cortical structures in the



• **Fig. 8.13** **A,** The receptive field and responses of a double-opponent (R^+G^-/R^-G^+) neuron in a blob of the striate cortex as it responds to various combinations of red and green bars. The best "on" response is to a *red bar* flanked by *two green bars*. **B,** Diagram of the columnar arrangement of the visual cortex. Ocular dominance columns are indicated by *I* (for ipsilateral) and *C* (for contralateral). Orientation columns are indicated by the smaller columns marked with short bars at varying angles. The cortical blobs contain neurons like that in **A** and have spectral opponent-receptive fields.



• **Fig. 8.14** Distribution of P and M cell influences on different areas of the visual cortex. *IT*, Inferotemporal area; *MST*, medial superior temporal area; *MT*, medial temporal area; *V1*, striate cortex; *V2 and V4*, higher order visual areas.

M pathway include layers 4B and 4Cα of the striate cortex and areas MT (medial temporal) and MST (medial superior temporal) on the lateral aspect of the temporal lobe, as well as Brodmann area 7a of the parietal lobe (see Fig. 8.14).

Both P and M pathways contribute to depth perception or stereopsis, which is dependent on slight differences in the retinal images formed in the two eyes. Stereopsis is useful only for relatively nearby objects. However, in such cases, these disparities provide visual cues about depth. Interestingly, the anatomy of the visual pathways indicates that depth perception must be a cortical function because it depends on convergent input from the two eyes and the left/right eye inputs are parallel but segregated in the LGN and in Layer 4 of striate cortex.

The separation of M and P pathways from the retina through the thalamus and all the cortical regions raises the issue of how all the parts are combined to account for the clear, coherent images of events, objects, and persons that humans perceive. It seems unlikely that all the components that represent a percept, such as parts of a face and whether that face belongs to a familiar person, are somehow converged onto a single neuron that recognizes it. Rather, complex percepts probably arise from the coordinated activity of large sets of neurons across multiple regions of the CNS. The process by which a "binding" of such disparate neuronal elements into a percept is unclear, but one working hypothesis is that it may be accomplished by the temporal synchronization of many anatomically distributed neural events.

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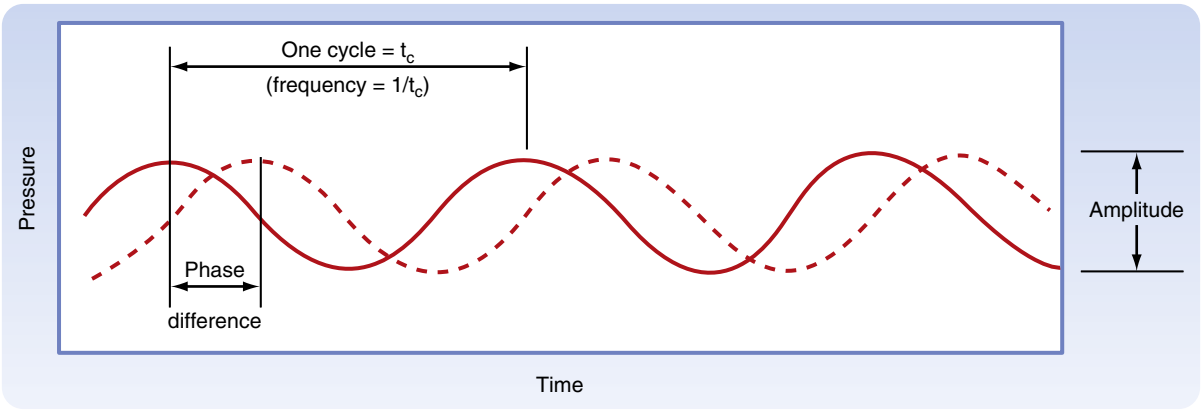
Lesions of the extrastriate visual cortex can produce various deficits. Bilateral lesions of the inferotemporal cortex can result in cortical color blindness **(achromatopsia)** or in an inability to recognize faces, even of close members of the family **(prosopagnosia).** A lesion in area MT or MST can interfere with motion detection and eye movements.



• **Fig. 8.15** Two pure tones are shown by the *solid* and *dashed lines.* Frequency is determined from the wavelength as indicated. Amplitude is the peak-to-peak change in sound pressure. The two tones have the same frequency and amplitude but differ in phase.

Other Visual Pathways

The **superior colliculus** of the midbrain is a layered structure that is important for certain types of eye movements (see Chapter 9). The three most superficial layers are involved exclusively in visual processing, whereas the deeper layers receive multimodal input from the somatosensory and auditory systems, as well as the visual system, particularly from cortical areas involved in eye movement.

Another retinal projection is to the **pretectum,** which bilaterally activates parasympathetic preganglionic neurons in the **Edinger-Westphal nucleus** that cause pupillary constriction in the pupillary light reflex. The pretectal areas are also interconnected through the posterior commissure, and thus the reflex causes both ipsilateral (direct) and contralateral (consensual) pupillary constriction when a light is shown in one eye.

The visual pathways also include connections to nuclei that serve functions other than vision. For example, a retinal projection to the **suprachiasmatic nucleus** of the hypothalamus contributes to circadian rhythmicity (see Chapter 38).

The Auditory and Vestibular Systems

The peripheral parts of the auditory and vestibular systems share components of the bony and membranous labyrinths, use hair cells as mechanical transducers, and transmit information to the CNS through the vestibulocochlear nerve (CN VIII). However, the CNS processing and sensory functions of the auditory and vestibular systems are distinct. The function of the auditory system is to transduce sound. This allows us to recognize environmental cues and to communicate with other organisms. The most complex auditory functions are those involved in language. The function of the vestibular system is to provide the CNS with information related to the position and movements of the head in space. Control of eye movement by the vestibular system is discussed in Chapter 9.

Audition

Sound

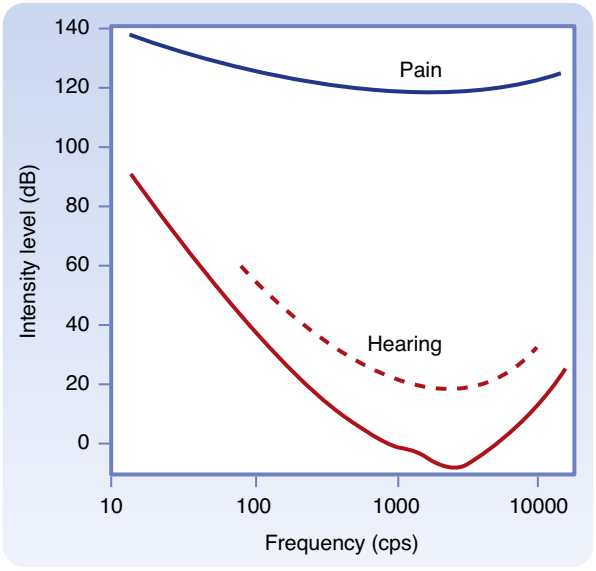
Sound is produced by compression and decompression waves in air or in other elastic media, such as water. Sound frequency is measured in cycles per second, or **hertz (Hz).** For example, the frequency of the middle A musical tone is 440 Hz. Each pure tone results from a sinusoidal wave at a particular frequency and is characterized not only by its frequency but also, instantaneously, by its amplitude and phase (Fig. 8.15). Most naturally occurring sound, however, is a mixture of pure tones. **Noise** is unwanted sound and may have any composition of pure tones. Sound propagates at about 335 m/second in air. The waves are associated with certain pressure changes, called *sound pressure.* The unit of sound pressure is Newtons per meter square (N/m^2), but sound pressure is more commonly expressed as the **sound pressure level (SPL).** The unit of SPL is the **decibel (dB):**

Equation 8.1

$$SPL = 20\log P/P_R$$

where P is sound pressure and P_R is a reference pressure (0.0002 dyne/cm^2, the absolute threshold for human hearing at 1000 Hz). A sound with intensity 10 times greater would be 20 dB; one 100 times greater would be 40 dB.

The normal young human ear is sensitive to pure tones with frequencies that range between about 20 and 20,000 Hz. The threshold for detection of a pure tone varies with its frequency (Fig. 8.16). The lowest thresholds for human hearing are, for pure tones, approximately 3000 Hz. The threshold at these frequencies is approximately −3 to −5 dB, in comparison with the reference 0 dB at 1000 Hz. In reference to this scale, normal speech has an intensity of about 65 dB, and its main frequencies fall in the range of 300 to 3500 Hz. Sounds that exceed 100 dB can damage the peripheral auditory apparatus, and those higher than 120 dB can cause pain and permanent damage. As people age, their thresholds at high frequencies rise, thereby



• **Fig. 8.16** Sound threshold intensities at different frequencies. The *bottom curve* indicates the absolute intensity needed to detect a sound. The *dashed curve* represents the threshold for functional hearing. The *top curve* indicates levels at which sound is painful and damaging.

reducing their ability to hear such tones, a condition called **presbycusis.**

The Ear

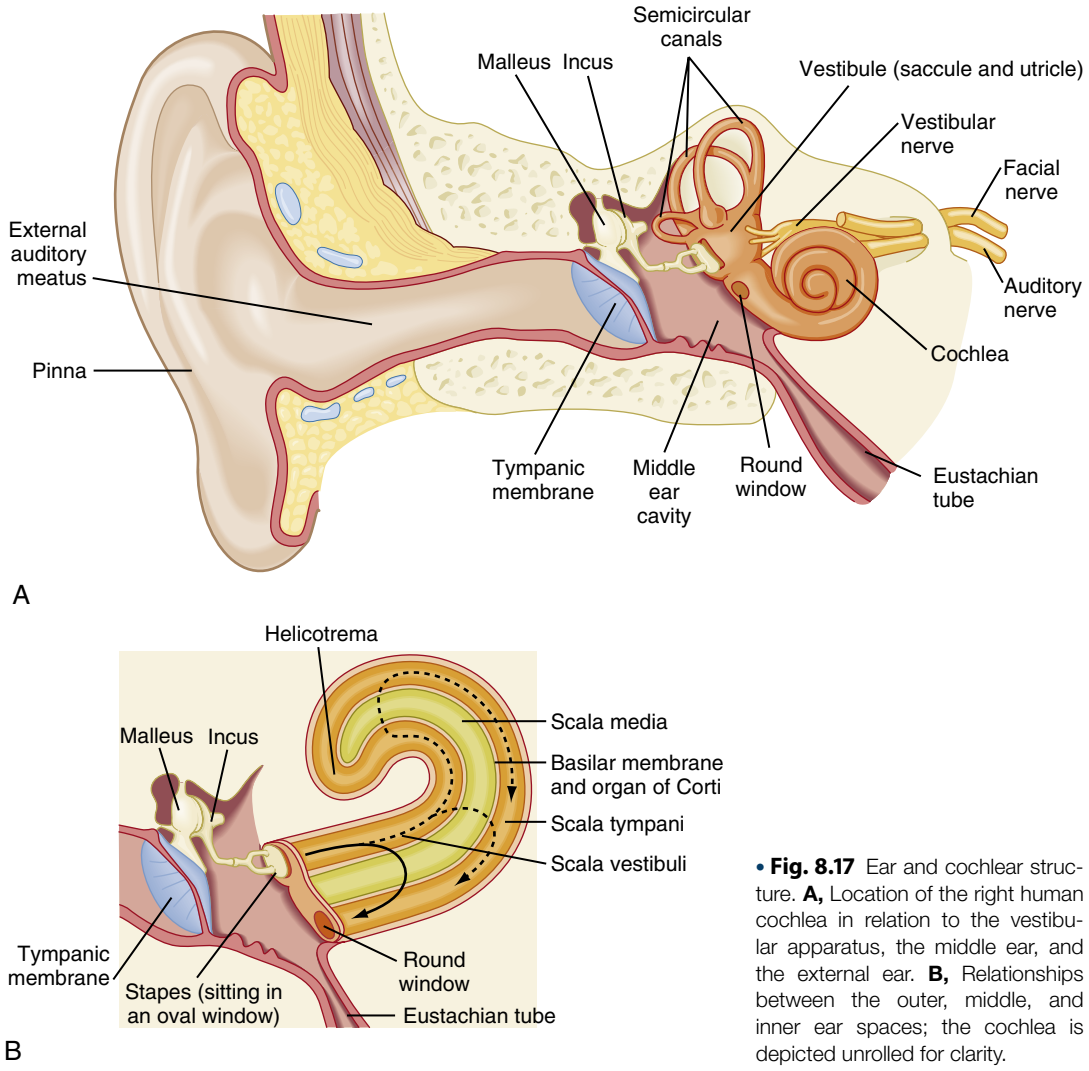
The peripheral auditory apparatus is the ear, which can be subdivided into the external ear, the middle ear, and the inner ear (Fig. 8.17).

External Ear

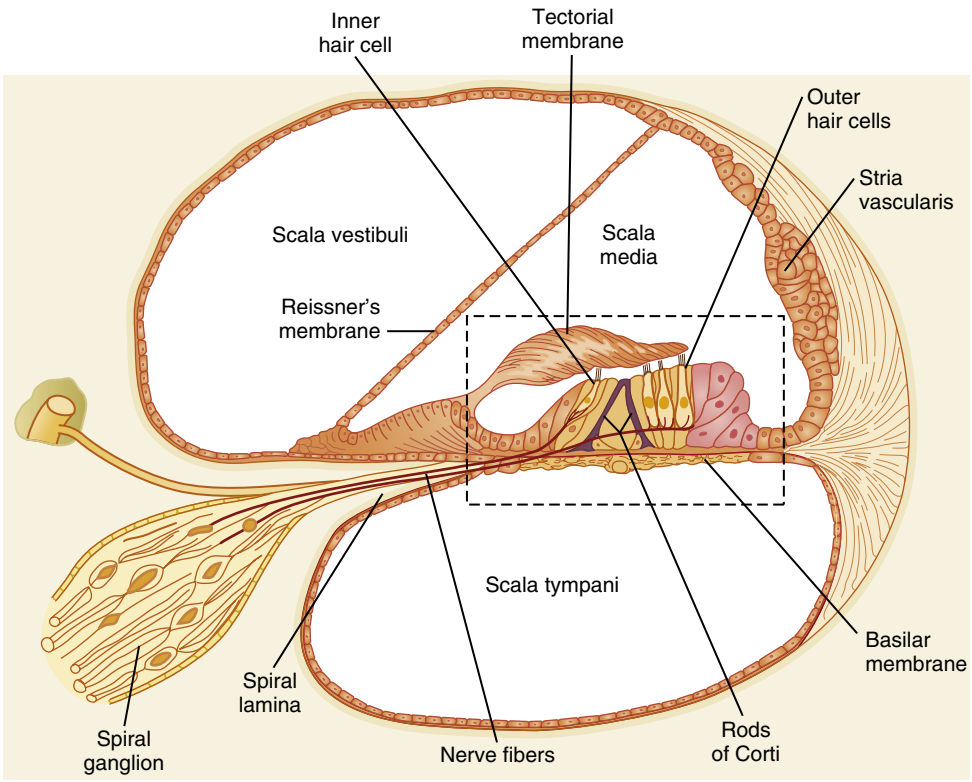
The external ear includes the pinna and the external auditory meatus (auditory canal). The auditory canal contains glands that secrete **cerumen,** a waxy protective substance. The pinna helps direct sounds into the auditory canal and plays a role in sound localization. The auditory canal transmits the sound pressure waves to the tympanic membrane. In humans, the auditory canal has a resonant frequency of about 3500 Hz, and this resonance contributes to the low perceptual threshold for sounds in that range.

Middle Ear

The external ear is separated from the middle ear by the **tympanic membrane** (see Fig. 8.17*A*). The middle ear



• **Fig. 8.17** Ear and cochlear structure. **A,** Location of the right human cochlea in relation to the vestibular apparatus, the middle ear, and the external ear. **B,** Relationships between the outer, middle, and inner ear spaces; the cochlea is depicted unrolled for clarity.



C

• **Fig. 8.17, cont'd C,** Drawing of a cross-section through the cochlea. The organ of Corti (see Fig. 8.18*A*,*B*) is outlined.

contains air. Three ossicles are present and serve to link the tympanic membrane to the oval window of the inner ear. Adjacent to the oval window is the round window, another membrane-covered opening between the middle ear and inner ear (see Fig. 8.17*A* and *B*).

The ossicles include the **malleus,** the **incus,** and the **stapes.** The stapes has a footplate that inserts into the oval window. Behind the oval window is a fluid-filled component of the inner ear, the **vestibule.** It is continuous with a tubular structure known as the **scala vestibuli.** Inward movement of the tympanic membrane by a sound pressure wave causes the chain of ossicles to push the footplate of the stapes into the oval window (see Fig. 8.17*B*). This movement of the stapes footplate in turn displaces the fluid within the scala vestibuli. The pressure wave that ensues within the fluid is transmitted through the **basilar membrane** of the **cochlea** to the **scala tympani** (described later), and it causes the round window to bulge into the middle ear.

The tympanic membrane and the chain of ossicles serve as an impedance-matching device. The ear must detect sound waves traveling in air, but the neural transduction mechanism depends on movement in the fluid-filled cochlea, where acoustic impedance is much higher than that of air. Therefore, without a special device for impedance matching, most sound reaching the ear would simply be reflected, as are voices from shore when a person is swimming under water. Impedance matching in the ear depends on (1) the ratio of the surface area of the large tympanic membrane to that of the smaller oval window and (2) the mechanical advantage of the lever system formed by the ossicles. This impedance matching is sufficient to increase the efficiency of energy transfer by nearly 30 dB in the range of hearing from 300 to 3500 Hz.

IN THE CLINIC

The middle ear also serves other functions. Two muscles are found in the middle ear: the tensor tympani attached to the malleus and the stapedius attached to the stapes. When these muscles contract, they damp movements of the ossicles and decrease the sensitivity of the acoustic apparatus. This action can protect the acoustic apparatus against damaging sounds that can be anticipated. However, a sudden explosion can still damage the acoustic apparatus because reflex contraction of the middle ear muscles does not occur quickly enough. The chamber of the middle ear connects to the pharynx through the eustachian tube. Pressure differences between the external ear and middle ear can be equalized through this passage. If fluid collects in the middle ear, as during an infection, the eustachian tube may become blocked. The resulting pressure difference between the external ear and middle ear can produce painful displacement of the tympanic membrane, and in extreme cases, cause rupture of the tympanic membrane. Unequalized pressure changes as a result of flying or diving can also cause discomfort.

Inner Ear

The inner ear includes the bony and membranous labyrinths. The bony labyrinth is a complex but continuous series of spaces in the temporal bone of the skull, whereas the membranous labyrinth consists of a series of soft tissue spaces and channels lying inside the bony labyrinth. The cochlea and the vestibular apparatus are formed from these structures.

The cochlea is a spiral-shaped organ (see Fig. 8.17*A* and *B*). In humans, the spiral consists of $2\frac{3}{4}$ turns from a broad base to a narrow apex, although its internal lumen is small at the base and wide at the top. The apex of the cochlea faces laterally (see Fig. 8.17*A*). The bony labyrinth component of the cochlea is subdivided into several chambers. The vestibule is the space facing the oval window (see Fig. 8.17*A*). Continuous with the vestibule is the scala vestibuli, the spiral-shaped chamber that extends to the apex of the cochlea, where it meets and merges with the **scala tympani** at the **helicotrema.** The scala tympani is another spiral-shaped space that winds back down the cochlea and ends at the round window (see Fig. 8.17*B*). Separating the two, except at the helicotrema, is the scala media enclosed in the membranous labyrinth.

The **scala media,** or **cochlear duct** (see Fig. 8.17*B* and *C*), is a membrane-bound spiral tube that extends along the cochlea, between the scala vestibuli and scala tympani. One wall of the scala media is formed by the **basilar membrane,** another by **Reissner's membrane,** and the third by the **stria vascularis** (see Fig. 8.17*C*).

The spaces within the cochlea are filled with fluid. The fluid in the bony labyrinth, including the scala vestibuli and scala tympani, is **perilymph,** which closely resembles cerebrospinal fluid. The fluid in the membranous labyrinth, including the scala media, is endolymph, which is very different from perilymph. **Endolymph,** generated by the **stria vascularis,** contains high [K^+] (about 145 mM) and low [Na^+] (about 2 mm) and has a high positive potential (about +80 mV) with regard to the perilymph. As a result, a very large potential gradient (about 140 mV) exists across the membranes of the hair cell cilia that extend into the endolymph. (These hair cells, which are the sensory receptors for sound, are discussed in more detail later.)

The neural apparatus responsible for transduction of sound is the **organ of Corti** (see Fig. 8.17*C*), which is located within the cochlear duct. It lies on the basilar membrane and consists of several components, including three rows of **outer hair cells,** a single row of **inner hair cells,** a gelatinous **tectorial membrane,** and a number of types of supporting cells. The organ of Corti in humans contains 15,000 outer and 3500 inner hair cells. The **rods of Corti** help provide a rigid scaffold. Located on the apical surface of the hair cells are stereocilia, which can be described as nonmotile cilia that contact the tectorial membrane.

The organ of Corti is innervated by nerve fibers of the cochlear division of the vestibulocochlear nerve (CN VIII). The 32,000 auditory afferent fibers in humans originate in sensory ganglion cells in the **spiral ganglion.** These nerve fibers penetrate the organ of Corti and terminate at the bases of the hair cells (Fig. 8.18; see also Fig. 8.17*C*). Approximately 90% of the fibers end on inner hair cells, and the remainder end on outer hair cells. Thus, approximately 10 afferent fibers supply each inner hair cell, whereas other afferent fibers diverge to supply about five outer hair cells each. The inner hair cells clearly provide most of the neural information about acoustic signals that the CNS processes for hearing. The sensory function of the outer hair cells is less clear.

In addition to afferent fibers, the organ of Corti is supplied by efferent fibers, most of which terminate on the outer hair cells. These cochlear efferent fibers originate in the superior olivary nucleus of the brainstem and are often called **olivocochlear fibers.** The length of the outer hair cells varies; this characteristic suggests that changes in outer hair cell length may affect the sensitivity, or "tuning," of the inner hair cells. The cochlear efferent fibers may control outer hair cell length. Such a mechanism could conceivably influence the sensitivity of the cochlea and the way that the brain recognizes sound. Other efferent fibers that end on cochlear afferent fibers may be inhibitory, and they may help improve frequency discrimination.

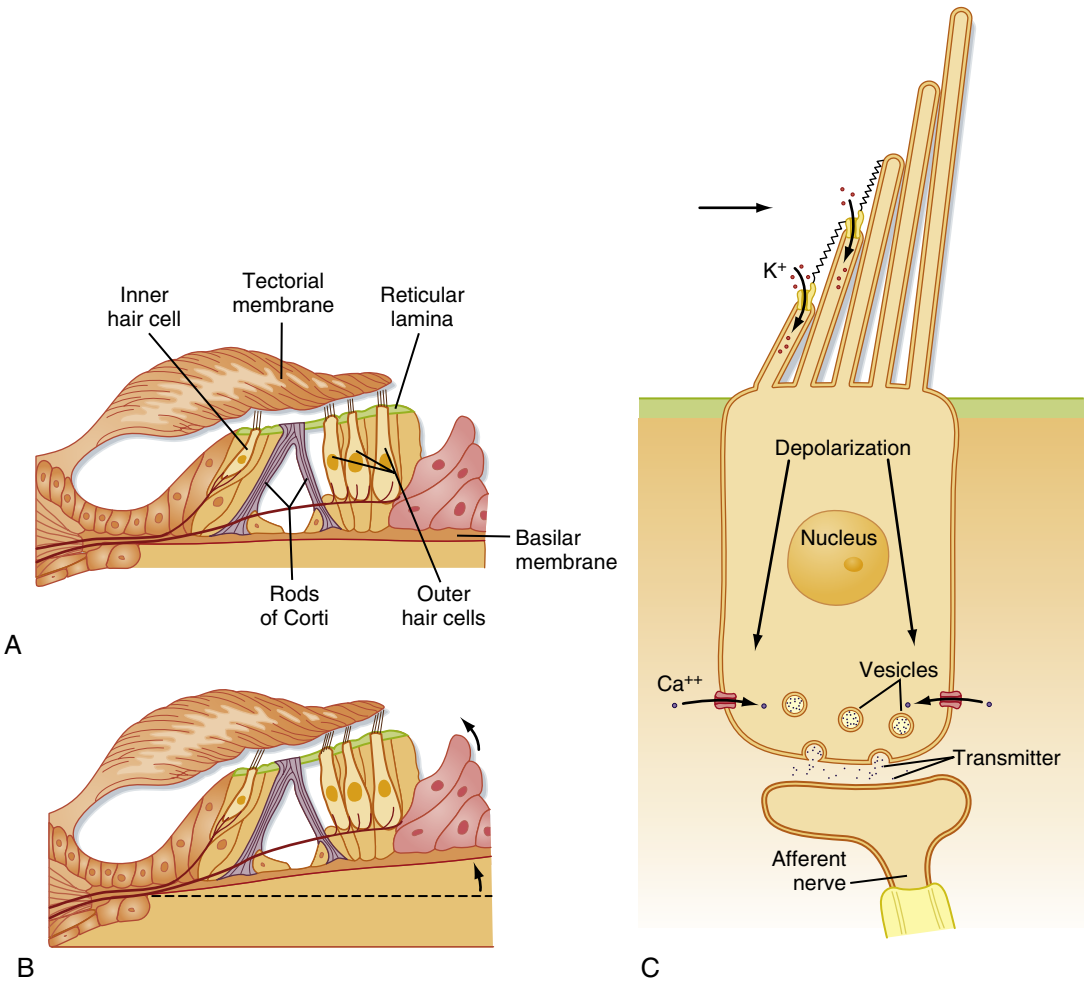
Sound is transduced by the organ of Corti. Sound waves that reach the ear cause the tympanic membrane to oscillate, and these oscillations are transmitted to the scala vestibuli by the ossicles. This creates a pressure difference between the scala vestibuli and the scala tympani (see Fig. 8.17*B*) that serves to displace the basilar membrane and, with it, the organ of Corti (see Fig. 8.18*A* and *B*). Because of the shear forces set up by the relative displacement of the basilar and tectorial membranes, the stereocilia of the hair cells bend. Upward displacement bends the stereocilia toward the tallest cilium, which leads to K^+ influx through K^+ channels and depolarization of the hair cells; downward deflection bends the stereocilia in the opposite direction, which closes K^+ channels and hyperpolarization of the hair cells (see the following section).

IN THE CLINIC

A common cause of deafness is the destruction of hair cells by loud sounds. Hair cells can be destroyed, for example, by exposure to industrial noise or by listening to loud music. Typically, hair cells in certain parts of the cochlea are selectively damaged by exposure to high levels of sound at particular frequencies (as predicted by the place theory (see explanation later in chapter)), and thus hearing may be lost over a discrete frequency range. Presbycusis, or the loss of high-frequency hearing with age, is probably increased by the loss of hair cells as a result of long-term noise exposure in urban environments. Diminished hearing can also be caused by high levels of antibiotics, which are known to damage K^+ channels on hair cells.

Sound Transduction

In view of the wide range of frequencies and amplitudes of sound stimuli, it is no surprise that hair cell transduction must provide for a fast response. The fast response to



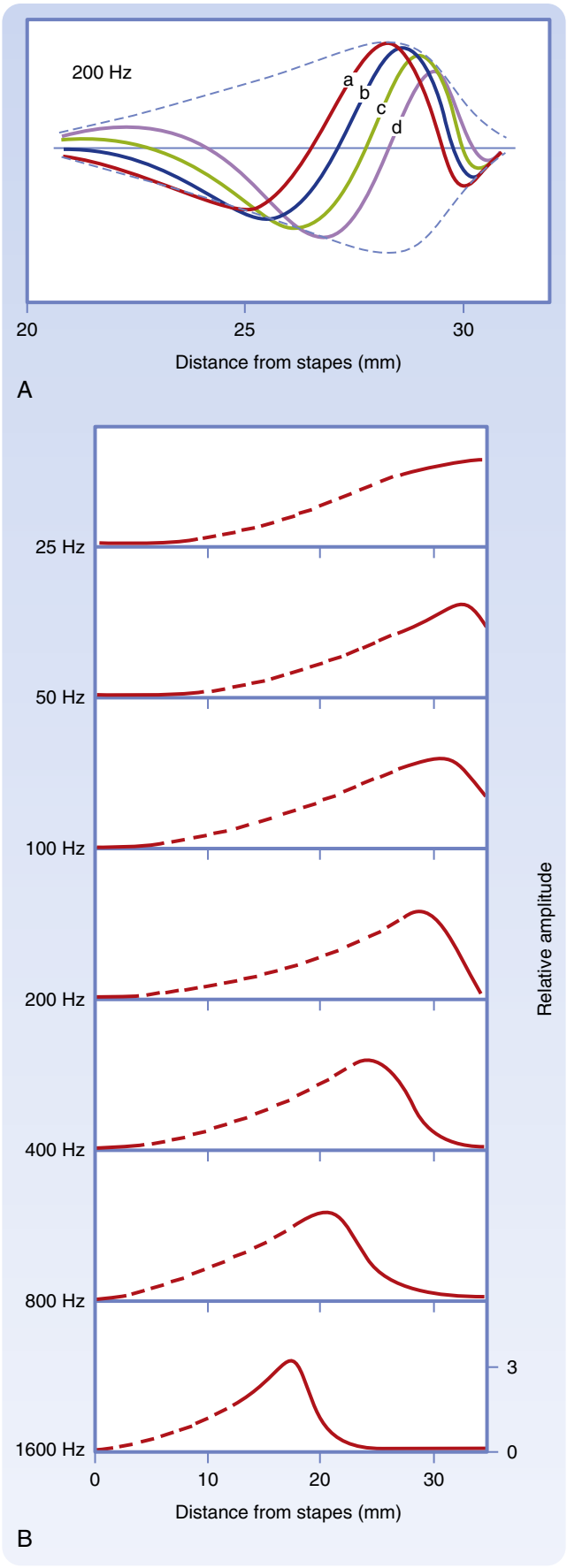
• **Fig. 8.18** Detail of the organ of Corti at rest **(A)** and with upward movement of the basilar membrane **(B).** The upward movement causes the stereocilia to bend because of shear forces produced by relative displacement of the hair cells and the tectorial membrane. **C,** Diagram of a hair cell with tip link connections between the hair cell cilia to show how shear forces open mechanoreceptor K^+ channels and depolarize the hair cell.

deflection of the cilia is based on direct opening of ion channels by so-called tip links that connect the tip of each stereocilium with the shaft of the next taller one (see Fig. 8.18*C*). With deflection, the tip links are subjected to a lever action that transiently opens the channels, allows the entry of K^+ (because of the high $[K^+]$ and high potential in endolymph), and depolarizes the hair cell. Several mechanisms have been proposed to account for the equally important rapid adaptation necessary for a high-frequency response. A "spring" response by the tip links would allow the attachment point of the tip link to be moved along the stereocilium's shaft to reset the mechanical leverage of the tip link. In addition, it has been observed that Ca^{++} can enter and bind to the open channel, change it to require greater opening force, and thereby reduce the statistical probability of opening.

The potential gradient that induces movement of ions into hair cells includes both the resting potential of the hair cells and the positive potential of the endolymph. As noted previously, the total gradient across the apical membrane of hair cells is about 140 mV. Therefore, a change in K^+ conductance in the apical membranes of hair cells results in a rapid current flow that produces the **receptor potential** in these cells. This current flow can be recorded extracellularly as a **cochlear microphonic potential,** an oscillatory event that has the same frequency as the acoustic stimulus. The cochlear microphonic potential represents the sum of the receptor potentials of a number of hair cells.

Hair cells, like retinal photoreceptors, release glutamate when depolarized. The neurotransmitter produces an excitatory postsynaptic potential (EPSP) in the cochlear afferent nerve fibers with which the hair cell synapses. In summary, sound is transduced when oscillatory movements of the basilar membrane cause transient changes in the transmembrane voltage of the hair cells and, finally, the generation of action potentials in cochlear afferent nerve fibers. The activity of a large number of cochlear afferent fibers in the auditory nerve can be recorded extracellularly as a compound action potential.

On the basis of differences in width and tension, investigators originally concluded that different parts of the basilar



• **Fig. 8.19** Different frequencies of sound result in different amplitudes of displacement at different sites along the organ of corti. **A,** Traveling wave produced in the basilar membrane by a sound of 200 Hz. The curves at *a, b, c, and d* represent displacement of the basilar membrane at different times, and the *dashed line* is the envelope formed by the peaks of the wave at different times. Maximum deflection occurs at about 29 mm from the oval window. **B,** Envelopes of traveling waves produced by several frequencies of sound. Note that the maximum displacement varies with frequency and is closest to the stapes when the frequency is highest. (Redrawn from von Bekesy G. *Experiments in Hearing.* New York: McGraw-Hill; 1960.)

membrane have different resonant frequencies. For example, the basilar membrane is about 100 μm wide at the base and 500 μm wide at the apex. It also has higher tension at the base. Thus, the investigators predicted that the base would vibrate at higher frequencies than would the apex, as do the shorter strings of musical instruments. Experiments have shown that the basilar membrane moves as a whole in traveling waves (Fig. 8.19), but displacement of the basilar membrane is maximal nearer the base of the cochlea during high-frequency tones and maximal nearer the apex during low-frequency tones.

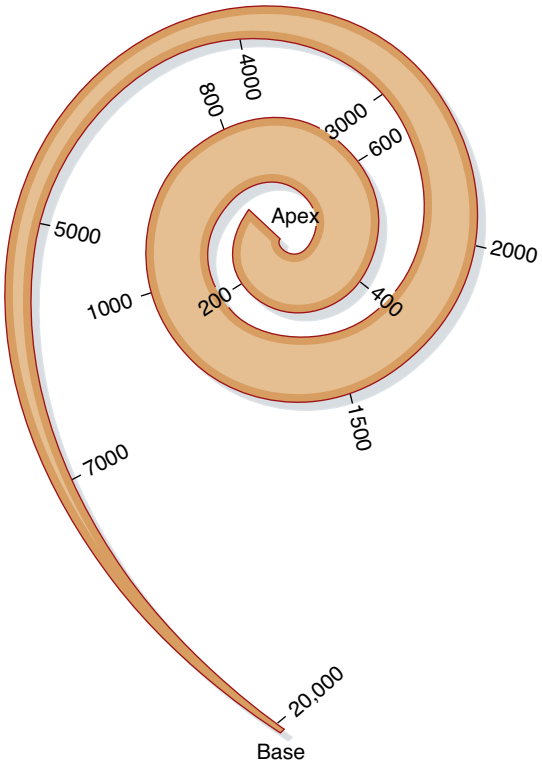
In effect, the basilar membrane serves as a frequency analyzer; it distributes the stimulus along the organ of Corti, and different hair cells respond differentially to particular frequencies of sound. This is the basis of the **place theory of hearing.** In addition, hair cells located at different places along the organ of Corti may be tuned to different frequencies because of variations in their stereocilia and biophysical properties. As a result of these factors, the basilar membrane and organ of Corti have a so-called tonotopic map (Fig. 8.20).

Cochlear Nerve Fibers

Neurotransmitter release by hair cells in the organ of Corti can evoke action potentials in the primary afferent fibers of the cochlear nerve. Afferent fibers in the vestibulocochlear nerve (CN VIII) are bipolar cells with a myelin sheath around the cell bodies, as well as around the axons. The cell bodies are in the spiral ganglion, their peripheral processes synapse at the base of hair cells, and their central processes synapse in the cochlear nuclei of the brainstem.

Characteristic Frequencies

A cochlear afferent fiber discharges maximally when stimulated by a particular sound frequency called its **characteristic frequency.** The characteristic frequency can be determined from a tuning curve for the fiber (Fig. 8.21). A **tuning curve** is a plot of the threshold for activation of the nerve fiber by different sound frequencies. The major factor that influences the activity of individual afferent fibers is the location along the basilar membrane of the hair cells that they innervate. The location of those hair cells is important because for any given sound frequency, there is a site of maximum displacement of the basilar membrane as the pressure wave travels along its length (see Fig. 8.19). Typically, tuning curves are sharp near the characteristic



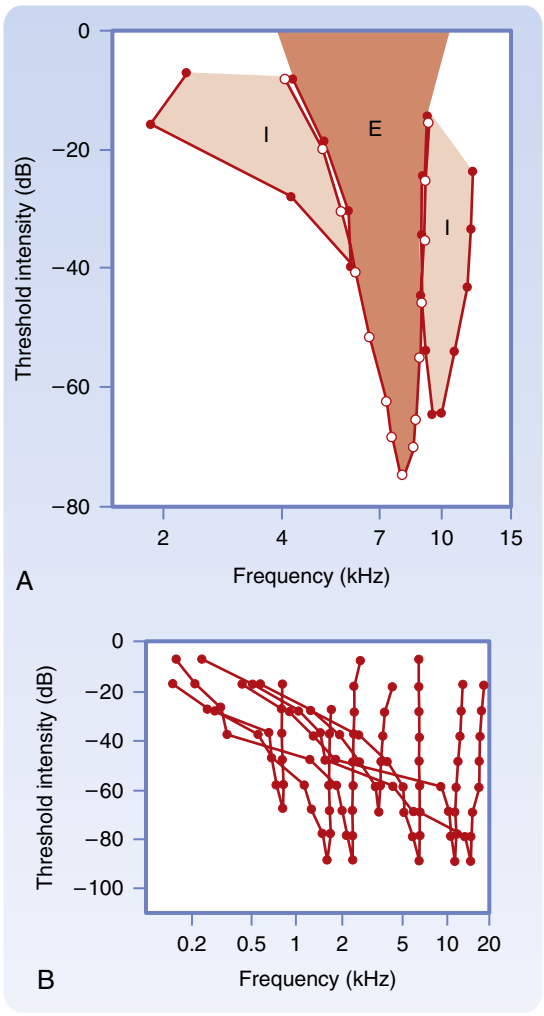
• **Fig. 8.20** The tonotopic map of the cochlea. (Redrawn from Stuhlman O. *An Introduction to Biophysics.* New York: John Wiley & Sons; 1943.)

frequency, but they broaden at high sound pressure levels. Tuning curves can have excitatory and inhibitory areas (see Fig. 8.21*A*). The sharpness of the excitatory regions may reflect inhibitory processes.

Encoding

The different features of an acoustic stimulus are encoded in the discharges of cochlear nerve fibers. Duration is signaled by the duration of activity; intensity is signaled both by the amount of neural activity and by the number of fibers that discharge. For low-frequency sounds, the frequency is signaled by the tendency of an afferent fiber to discharge in phase with the stimulus (**phase locking;** see Fig. 8.22*A*). If the tone is much more than 1 kHz, a single fiber cannot discharge with every cycle, but phase locking can also occur for sounds with periods shorter than the absolute refractory period of the afferent fiber. This allows the CNS to detect higher frequency information from the activity of a population of afferent fibers, each of which discharges in phase with the stimulus and which, as a group, signal the frequency of the stimulus (see Fig. 8.22*B*). This observation is the basis of the **frequency theory of hearing.**

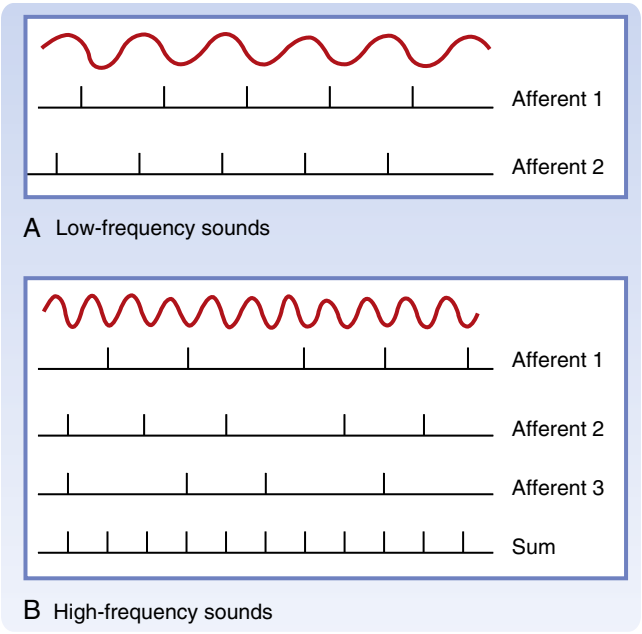
For still higher frequencies (>5000 Hz), the place theory dominates: the CNS interprets sounds that activate afferent fibers supplying hair cells near the base of the cochlea as being of high frequency. Both place theory and frequency theory are necessary to explain the frequency coding of sound **(duplex theory)** across the entire range from 20 to 20,000 Hz.



• **Fig. 8.21** Tuning curves of neurons in the auditory system. Tuning curves can be considered as receptive field plots. **A,** Tuning curve with central excitatory frequencies *(E)* and flanking inhibitory frequencies *(I)*. **B,** Tuning curves for cochlear nerve fibers. (**A,** Redrawn from Arthur RM, Pfeiffer RR, Suga, N. *J Physiol [Lond]* 1971;212:593. **B,** Redrawn from Katsui Y. In: Rosenblith WA, ed. *Sensory Communication.* Cambridge, MA: MIT Press; 1961.)

IN THE CLINIC

An important, although relatively uncommon, condition that can interrupt the function of cochlear nerve fibers is a vestibular schwannoma (also known as an **acoustic neuroma).** It is a tumor of the Schwann cells insulating the vestibulocochlear nerve (CN VIII). As the tumor grows, irritation of cochlear nerve fibers may cause a ringing sound in the affected ear **(tinnitus)** and diminished hearing. Eventually, conduction in cochlear nerve fibers is blocked, and the ear becomes deaf. The tumor may be operable while still small; therefore, early diagnosis is important. If the tumor is allowed to enlarge substantially, it can interrupt the entire vestibulocochlear nerve, thereby disrupting both auditory and vestibular function. These schwannomas can also impinge on, or distort, neighboring cranial nerves (e.g., V, VII, IX, and X), leading to compression of cerebellar peduncles and consequent disruption of cerebellar function.



• **Fig. 8.22 A,** At low frequencies, individual auditory afferent fibers can respond at each cycle to the signal frequency. **B,** At higher frequencies, each afferent fiber generates an action potential only at certain cycles, limited by its maximum firing frequency. However, the overall population of afferent fibers can still signal stimulus frequency by their aggregate firing frequency.

Central Auditory Pathway

Cochlear afferent fibers synapse on neurons of the dorsal and ventral cochlear nuclei. The neurons in these nuclei have axons that contribute to the central auditory pathways. Some of the axons from the cochlear nuclei cross to the contralateral side and ascend in the **lateral lemniscus,** the main ascending auditory tract. Others connect with various ipsilateral or contralateral nuclei, such as the **superior olivary nuclei,** which project through the ipsilateral and contralateral lateral lemnisci. Each lateral lemniscus ends in an **inferior colliculus** of the midbrain. Neurons of the inferior colliculus project to the **medial geniculate nucleus** (**MGN**) of the thalamus, which gives rise to the auditory radiation. The auditory radiation ends in the **primary auditory cortex** (Brodmann areas 41 and 42), located on the superior surface of the temporal lobe.

The input from each ear is bilaterally represented in the ascending auditory system pathway at the level of the lateral lemniscus and above. As a consequence, unilateral deafness may occur with isolated lesions of the cochlear nuclei or more peripheral structures. Central lesions do not cause unilateral deafness, although they may interfere with overall sensitivity to speech or with sound localization.

Functional Organization of the Central Auditory System

Receptive Fields and Tonotopic Maps

The responses of neurons in several structures that belong to the auditory system can be described by **tuning curves** (see Fig. 8.21*B*). By plotting the distribution of the characteristic frequencies of neurons within a nucleus or in the auditory cortex, a **tonotopic map** may be revealed in which neurons are ordered according to their "best" frequencies. Tonotopic maps have been found in the cochlear nuclei, superior olivary complex, inferior colliculus, medial geniculate nucleus, and auditory cortex. A given auditory structure may, in fact, contain several tonotopic maps.

Binaural Interactions

Most auditory neurons at levels above the cochlear nuclei respond to stimulation of either ear (i.e., they have **binaural receptive fields**). Binaural receptive fields contribute to sound localization. A human can distinguish sounds originating from sources separated by as little as 1 degree. The auditory system uses several clues to judge the origin of sounds, including differences in the time (or phase) of arrival of the sound at the two ears, and differences in sound intensity on the two sides of the head.

For example, neurons in the medial superior olivary nucleus have medial and lateral dendrites. The synapses on the medial dendrites are largely excitatory, and they originate from the contralateral ventral cochlear nucleus. Those on the lateral dendrites are mostly inhibitory and come from the ipsilateral ventral cochlear nucleus. Differences in the phase of the sound reaching the two ears affect the strength and timing of the excitation and inhibition reaching a particular medial superior olivary neuron. The lateral superior olivary nucleus processes differences in the sound intensity that reaches the two ears to provide information about the source of the sound. The activity of superior olivary neurons can provide information about sound localization.

Cortical Organization

Several features of the primary auditory cortex resemble those of other primary sensory areas. Not only are sensory maps—in this case, tonotopic maps—present in the auditory cortex but also this cortical region performs feature extraction. Neurons in the primary auditory cortex form **isofrequency columns** (in which the neurons in the column have the same characteristic frequency), and they also form alternating columns, known as summation and suppression columns. Neurons in **summation columns** are more responsive to binaural than to monaural input. Neurons in **suppression columns** are less responsive to binaural than to monaural stimulation, and, accordingly, the response to one ear is dominant. Some neurons are selective for the direction of frequency change.

Bilateral lesions of the auditory cortex have some effect on the ability to distinguish the frequency or intensity of different sounds, and they reduce the abilities to localize sound and to understand speech. Unilateral lesions, however, have little effect, especially if the nondominant (for language) hemisphere is involved (see Chapter 10). Evidently, frequency discrimination depends on activity at lower levels of the auditory pathway, possibly the inferior colliculus.

IN THE CLINIC

Two simple tests are often used clinically to distinguish the most important types of deafness: **conduction loss** and **sensorineural loss.** Conduction hearing loss occurs in disorders of the external ear (e.g., ear canal blocked by cerumen) or middle ear (e.g., rupture of the eardrum). Sensorineural hearing loss reflects disorders of the inner ear, the cochlear nerve, or central connections.

The **Weber test** is used to evaluate the magnitude of conduction hearing loss. In this test, the base of a vibrating tuning fork is placed against the middle of the person's forehead, and the person is asked to localize the sound. Normally, the sound is not localized to a particular ear. However, if the person has conductive hearing loss (e.g., due to a punctured tympanic membrane, fluid in the middle ear, otosclerosis, or loss of continuity of the ossicular chain), the sound is localized to the deaf ear because it is conducted to the cochlea through bone. The sound is also conducted to the cochlea of the undamaged ear, but bone-conducted sound does not activate the organ of Corti as well as does sound conducted normally through the tympanic membrane and ossicle chain. One reason why the sound in the Weber test is not localized to the normal ear may be that hearing in the normal ear is inhibited by the ambient sound level **(auditory masking).** Conversely, in people with sensorineural hearing loss (e.g., due to damage to the organ of Corti, the cochlear nerve, or the cochlear nuclei), the sound is localized to the normal side.

In the **Rinne test,** a vibrating tuning fork is placed against the bone behind the person's ear, and the person is asked to indicate when the sound dies out. The tuning fork is then held near the external auditory meatus of that ear. In people with normal hearing, the sound is again heard because the sound is more effectively transmitted to the cochlea in air (i.e., air conduction is better than bone conduction). If the conduction mechanism is damaged, the sound is not heard when the tuning fork is held near the external auditory meatus. Bone conduction in this case is better than air conduction. If the hearing loss is sensorineural, the sound is heard again when the tuning fork is placed by the external auditory meatus, because with sensorineural hearing loss, the inner ear and cochlear nerve are less able to transmit impulses regardless of whether the sound vibrations reach the cochlea via air or bone. Because air conduction is more effective than bone conduction, the bone conduction pattern seen with sensorineural hearing loss is the same as in a normal ear.

The Vestibular System

The vestibular system detects angular and linear accelerations of the head. Signals from the vestibular system allow the body to make adjustments in posture that maintain balance and trigger head and eye movements to stabilize the visual image on the retina. The following description of the vestibular system emphasizes the sensory aspects of vestibular function, and it introduces the central vestibular pathways. The role of the vestibular apparatus in motor control is discussed in Chapter 9.

The Vestibular Apparatus

Structure of the Vestibular Labyrinth

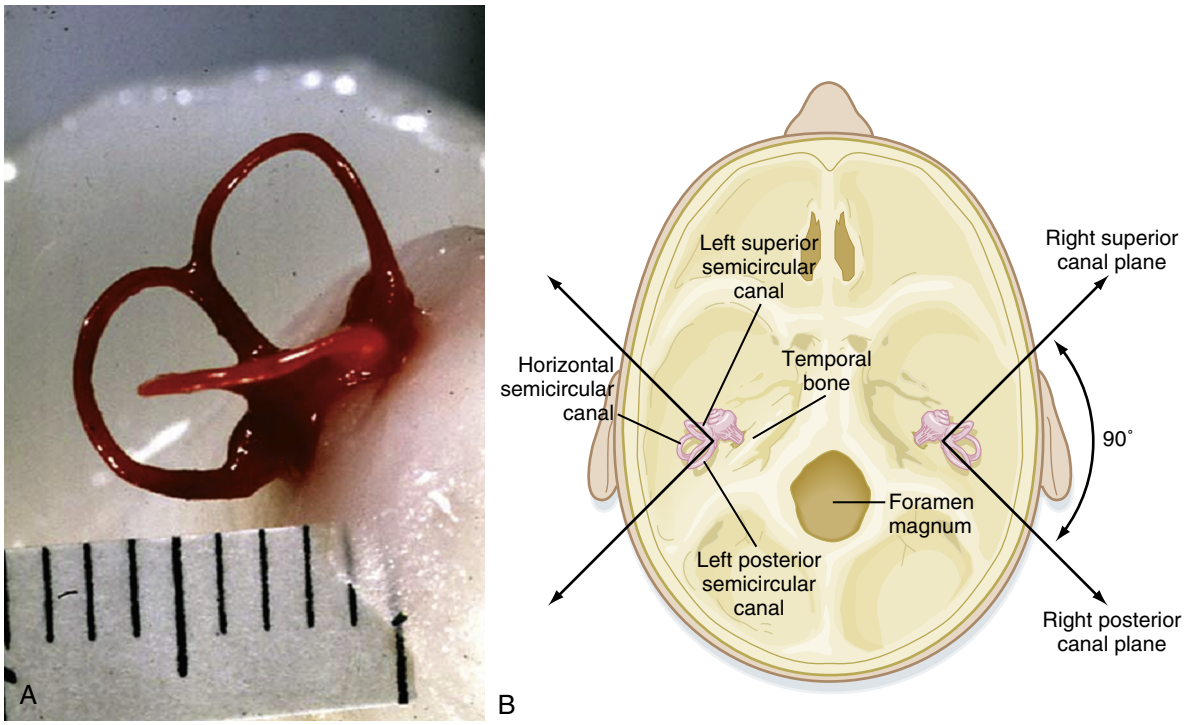
The vestibular apparatus, like the cochlea, consists of a component of the membranous labyrinth located within the bony labyrinth. The vestibular apparatus on each side is composed of three **semicircular canals** and two **otolith organs** (Fig. 8.23; see also Fig. 8.17*A*). These structures contain endolymph and are surrounded by perilymph. The semicircular canals are named the **horizontal, anterior,** and **posterior** canals. The otolith organs are the **utricle** and the **saccule** (together indicated as "Vestibule" in Fig. 8.17*A*). Each semicircular canal has a swelling called an **ampulla** at the point where it joins the utricle. The saccule connects with the cochlea, through which endolymph (produced by the stria vascularis of the cochlea) can reach the vestibular apparatus.

The three semicircular canals on one side are matched with corresponding coplanar semicircular canals on the other side. The horizontal canals on each side of the head correspond, as do the anterior canal on one side and the posterior canal on the other side (see Fig. 8.23*B*). This arrangement allows the sensory epithelia, in corresponding pairs of canals on the two sides, to cooperate in sensing acceleration of the head about three nearly orthogonal axes in space. The horizontal canals are not truly horizontal; rather, they lie in the horizontal plane if the head is tilted down 30 degrees in relation to the horizon.

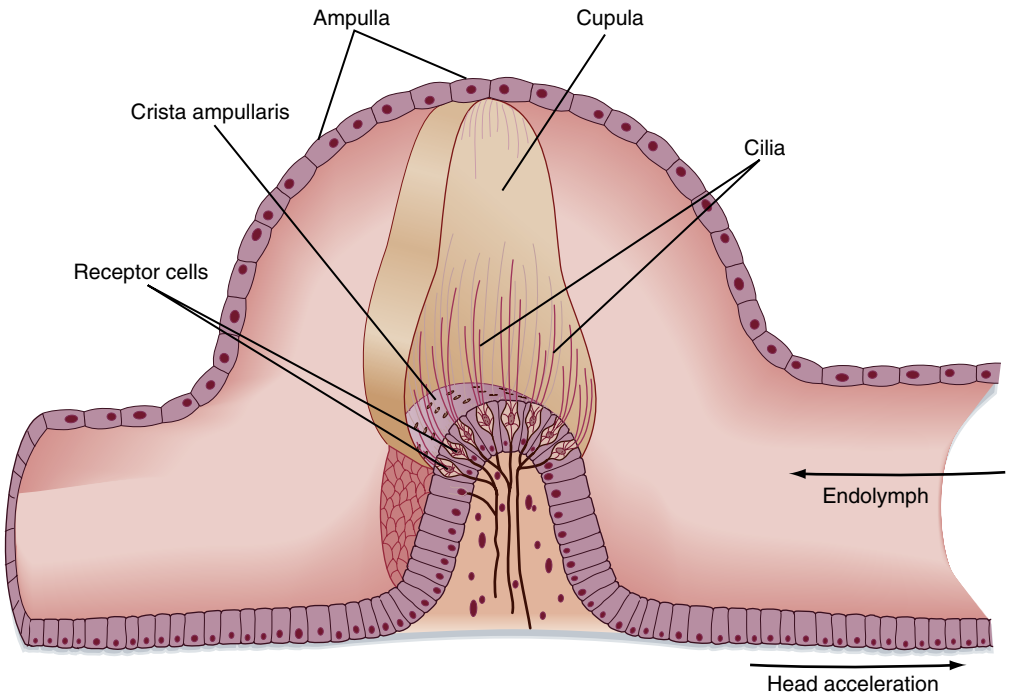
The ampulla of each of the semicircular canals contains a sensory epithelium called a **crista ampullaris,** or **ampullary crest** (Fig. 8.24). An ampullary crest consists of a ridge, transverse to the long axis of the canal, that is covered by epithelium containing vestibular hair cells. These hair cells are innervated by primary afferent fibers of the vestibular nerve, which is a subdivision of the vestibulocochlear nerve (CN VIII).

Like cochlear hair cells, each vestibular hair cell contains a set of stereocilia on its apical surface. However, unlike cochlear hair cells, vestibular hair cells also contain a large single kinocilium. The cilia on ampullary hair cells are embedded in a gelatinous structure called the *cupula.* The cupula and the crista occlude the lumen of the ampulla completely. Movement of endolymph, produced by angular acceleration of the head about an axis perpendicular to the plane of the canal, deflects the cupula and consequently bends the cilia on the hair cells, thereby opening or closing K^+ channels. The cupula has the same specific gravity as endolymph, and thus it is unaffected by linear acceleratory forces, such as gravity.

The sensory epithelia of the otolith organs are called the **macula utriculi** and the **macula sacculi** (Fig. 8.25). The utricle is oriented nearly horizontally; the saccule is oriented vertically. Their hair cells are embedded in the epithelium that overlies each macula. As in the ampullary crests, the stereocilia and kinocilia of the macula project into a gelatinous mass. However, the gelatinous mass in the macula contains numerous **otoliths** ("ear stones") composed of calcium carbonate crystals. Together, the gelatinous mass and its otoliths are known as an **otolithic membrane.** The otoliths increase the specific gravity of the otolithic membrane to about twice that of the endolymph. Hence, the otolithic membrane tends to move when subjected to acceleration, whether linear (such as that produced by gravity) or angular, particularly when the center of rotation is outside the head.



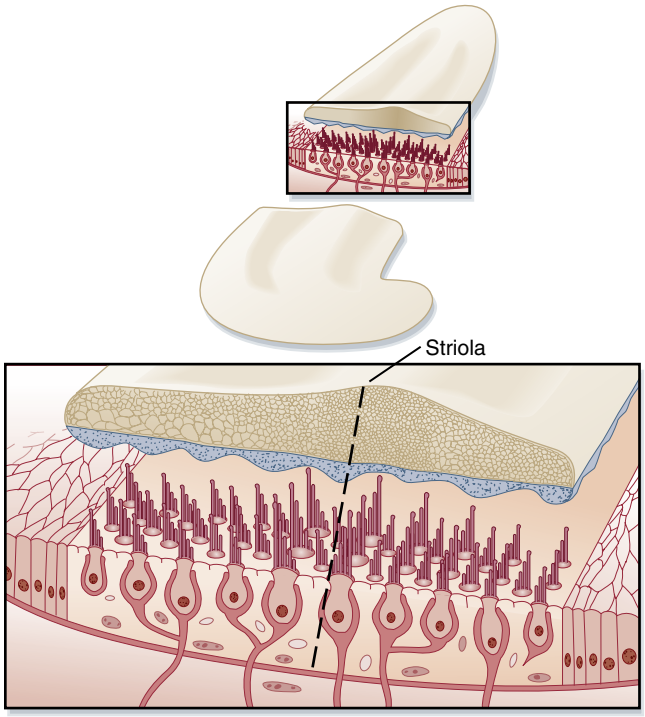
• **Fig. 8.23** **A,** Lateral view of the right semicircular canals of a rhesus monkey that were dissected after being filled with plastic. Note the ampullae associated with each canal. Scale is in millimeters. **B,** Overhead view of the base of the skull showing the orientation of structures of the inner ear. Coplanar pairs of semicircular canals include the horizontal canals, as well as the anterior and contralateral posterior canals. (**A,** Courtesy of Dr. John Simpson, New York University School of Medicine. **B,** Redrawn from Haines DE. *Fundamental Neuroscience for Basic and Clinical Applications.* 3rd ed. Philadelphia: Churchill Livingstone; 2006.)



• **Fig. 8.24** Drawing of a crista ampullaris inside an ampulla. The stereocilia and the kinocilium of each hair cell extend into the cupula, which extends across the entire cross-section of the ampulla. Head movement (acceleration) to the right would result in endolymph pressure to the left and deflection of the cupula to the left.

Innervation of Sensory Epithelia of the Vestibular Apparatus

The cell bodies of the primary afferent fibers of the vestibular nerve are located in the Scarpa ganglion. The neurons are bipolar, and their cell bodies, as well as axons, are myelinated. Peripherally, the vestibular nerve gives off separate branches to each of the vestibular epithelia; centrally, it accompanies the cochlear and facial nerves as they enter the internal auditory meatus of the skull.



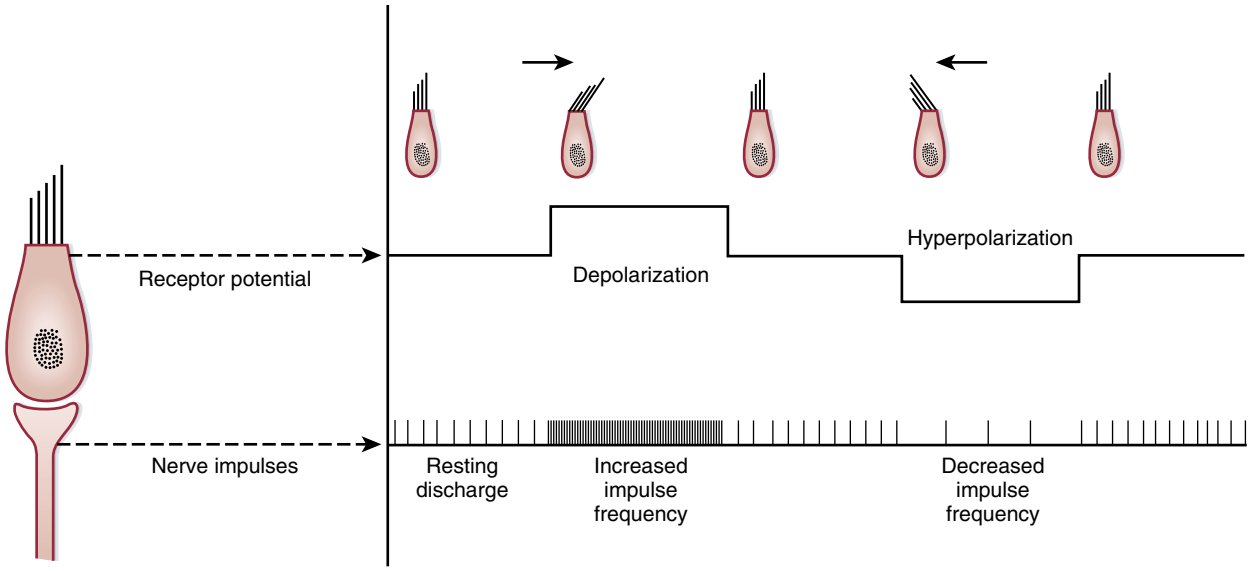
• **Fig. 8.25** Structure of one of the otolith organs, the saccule. Note the orderly variation in kinocilium orientation, as well as their mirror symmetry with regard to the striola. (Redrawn from Lindeman HH. *Adv Otorhinolaryngol* 1973;20:405.)

Vestibular Transduction

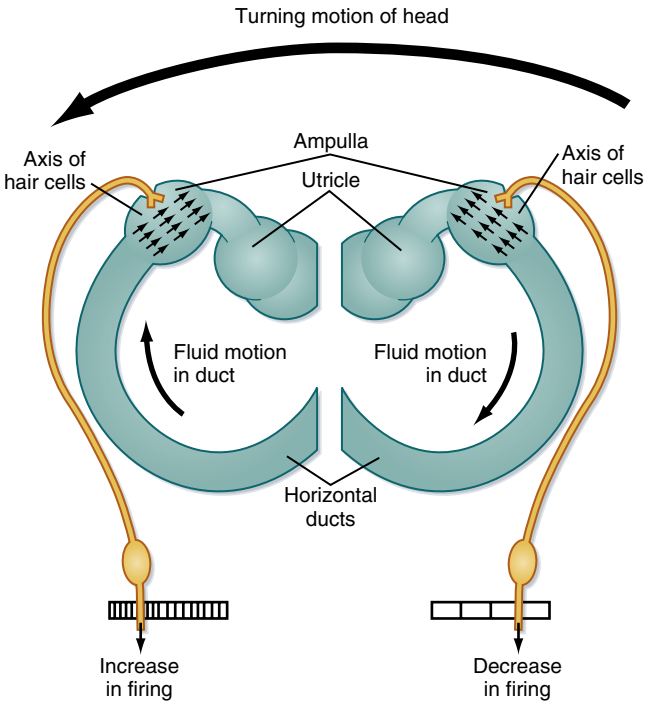
Like cochlear hair cells, vestibular hair cells are functionally polarized, and the transduction mechanism is presumed to be similar. When the stereocilia are bent toward the longest cilium (in this case, the **kinocilium**), conductance of the apical membrane increases for cations and, because of the high K^+ concentration of the endolymph, K^+ enters, and the vestibular hair cell is depolarized (Fig. 8.26). Conversely, when the cilia are bent away from the kinocilium, the hair cell is hyperpolarized. The hair cell releases glutamate tonically, so that the afferent fiber on which it synapses has a resting discharge. When the hair cell is depolarized, more neurotransmitter is released, and the discharge rate of the afferent fiber increases. Conversely, when the hair cell is hyperpolarized, less neurotransmitter is released, and the firing rate of the afferent fiber slows.

Semicircular Canals

Angular accelerations of the head produce small movement of the endolymph in relation to the head (Fig. 8.27). This happens because the inertia of the endolymph causes it to resist the initial acceleration of the membranous labyrinth. This lag pushes on the cupula, causes the cilia to bend, and consequently changes the discharge rates of the vestibular afferent fibers. All the cilia in a given ampullary crest are oriented in the same direction. In the horizontal canal, the cilia are oriented toward the utricle, and in the other ampullae, they are oriented away from the utricle.



• **Fig. 8.26** Functional polarization of vestibular hair cells. When the stereocilia are bent toward the kinocilium, the hair cell is depolarized, and the afferent fiber is excited. When the stereocilia are bent away from the kinocilium, the hair cell is hyperpolarized, and the afferent discharge slows or stops. (Redrawn from Kandel ER, Schwartz JH. *Principles of Neural Science.* New York: Elsevier; 1981.)

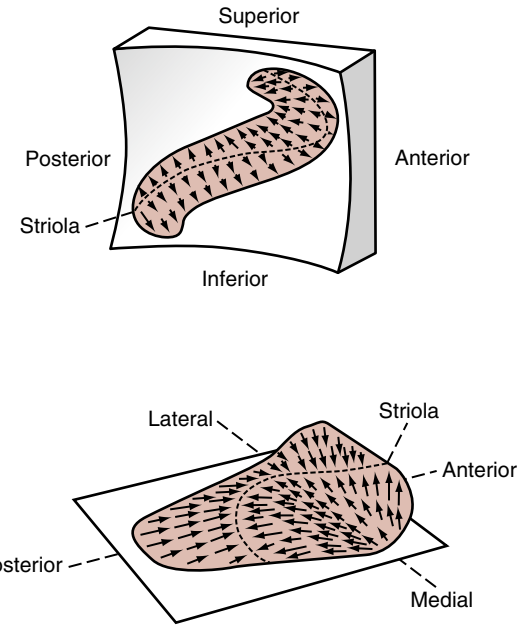


• **Fig. 8.27** Effect of leftward head movement on the activity of vestibular afferent fibers supplying hair cells in the horizontal semicircular canals. The perspective in the figure is from above the head looking down. In this case, head rotation (acceleration) to the left causes endolymph pressure to the right and the result is increased output from the left canal and decreased output from the right canal. *Small arrows* in the ampulla indicate the functional polarity of the hair cells. The *large curved arrow* at the top indicates movement of the head; *smaller curved arrows* indicate relative movement of the endolymph.

The way in which angular acceleration of the head affects the discharge of vestibular afferent fibers is exemplified by the activity that originates from the horizontal canals. Fig. 8.27 shows the horizontal canals and utricle, as seen from above as the head is rotated (accelerated) to the left. As acceleration to the left begins, the inertia of the endolymph in the horizontal canals increases pressure toward the right side. This causes the cilia to bend on hair cells of the ampulla of the left horizontal canal toward the utricle and bends the cilia of the right canal away from the utricle. These actions increase the firing rate in the afferent fibers on the left and decrease the firing rate of the afferent fibers on the right. Once the head is moving at a constant velocity of rotation (i.e., no acceleration), there would be no force on either cupula, and therefore the hair cells of both canals would be firing as they do at rest. However, when the indicated rotation is stopped, the inertia of the endolymph creates a force on both cupulas, but in the direction opposite to that caused by the original acceleration. This results in an increase in the discharge rate of afferent fibers on the right side and a decrease in the discharge rate on the left. This post-rotatory effect is of functional and clinical significance.

Otolith Organs

Unlike the hair cells in the ampullary crests, not all the hair cells in the otolith organs are oriented in the same direction.



• **Fig. 8.28** Functional polarization of hair cells in the otolith organs. **A,** The saccule. **B,** The utricle. The striola in each case is indicated by the *dotted line.* (Redrawn from Spoendlin HH. In: Wolfson RJ, ed. *The Vestibular System and Its Diseases.* Philadelphia: University of Pennsylvania Press; 1966.)

IN THE CLINIC

Disruption of the vestibular labyrinth, as in **Meniere's disease,** can result in transient rhythmic conjugate deviations of the eyes, followed by quick return saccades. This condition is known as **nystagmus** (see Chapter 9). These eye movements are accompanied by a sense of **vertigo** and often **nausea.** The brain interprets a difference in the input from the two sides of the vestibular system as head motion. It is thought that the disruption is due to changes in endolymph concentration and consequent activation of hair cells. Disruption of one labyrinth produces an asymmetry of input that results in abnormal eye movement and associated psychological effects.

Instead, they are oriented in relation to a ridge, called the **striola,** along the otolith organ (see Fig. 8.25). In the utricle, the hair cells on either side of the striola are polarized toward the striola, whereas in the saccule they are polarized away from the striola. Because the striola in each otolith organ is curved, there are hair cells with all orientations in the plane of the organ (Fig. 8.28). In any particular orientation of the head, the cilia of the hair cells are bent to varying extents according to their orientation in relation to the gravitational vector. This results in a particular pattern of input from the otolith organs to the CNS. When the head is tilted to a new position, the orientation of the otolithic membranes in relation to the gravitational vector changes, and so the cilia of the hair cells are bent in a new way. This change in the bending of the cilia of the hair cells changes

the pattern of input from the otolith organs to the CNS and creates the sensation of movement, as well as possibly triggering various reflexes. Similarly, a linear acceleration caused by other forces, such as might occur in a fall or the angular acceleration when a car turns around a curve (angular accelerations have linear centripetal and instantaneous tangential components), also affects output from the otolith organs.

Central Vestibular Pathways

The vestibular afferent fibers project to the brainstem through the vestibular nerve. As previously mentioned, the cell bodies of these afferent fibers are located in the Scarpa ganglion. These primary afferent fibers terminate in the **vestibular nuclei** (see Fig. 4.6*D–E*), which are located in the rostral medulla and caudal pons, and in specific regions of the **cerebellum**, most prominently in the **nodulus.**

The vestibular nuclei give rise to various projections, including projections through the **medial longitudinal fasciculus** (**MLF**; see Fig. 4.6*C–E*) to the oculomotor nuclei. Therefore, it is not surprising that the vestibular nuclei exert powerful control over eye movements (the **vestibule-ocular reflex; VOR**). Other projections give rise to the **lateral** and **medial vestibulospinal tracts,** which, respectively, provide for the activation of trunk and neck muscles and thereby contribute to equilibrium and to head movements **(vestibulocolic reflex).** There are vestibular pathways to the cerebellum, the reticular formation, and the contralateral vestibular complex, as well as to the thalamus. The pathway to the thalamus, via a projection to the cerebral cortex, mediates conscious sensation of vestibular activity. Vestibular reflexes and clinical tests of vestibular function are described in Chapter 9.

The Chemical Senses

The senses of **gustation** (taste) and **olfaction** (smell) help detect chemical stimuli that are present in food and drink, or in the air. While these senses might not be considered as important as some of the other senses, they contribute considerably to quality of life and food selection, and they are important stimulants of digestion. In other animals, the chemical senses have greater survival value, and their activation evokes a number of social behaviors, including mating, territoriality, and feeding.

Taste

The stimuli that we commonly know as tastes are actually mixtures of five elementary taste qualities: salty, sweet, sour, bitter, and umami.[b] Taste stimuli that are particularly effective in eliciting these sensations are, respectively, sodium chloride, sucrose, hydrochloric acid, quinine, and monosodium glutamate. Umami has been described as having a proteinaceous, meaty character.

[b]The existence of a sixth, taste, fat (free fatty acids), is currently being debated.

Taste Receptors

The sensation of taste depends on the activation of chemoreceptors located in taste buds. A taste bud consists of a group of 50–150 receptor cells, as well as supporting cells and basal cells (Fig. 8.29*A*). The chemoreceptor cells synapse at their bases with primary afferent nerve fibers, and their peaks have microvilli that extend toward a taste pore. Chemoreceptor cells live only about 10 days. They are continuously replaced by new chemoreceptor cells that differentiate from basal cells located near the base of the taste bud.

Chemoreceptor molecules, each specialized for one type of taste stimulus, sit on the microvilli of chemoreceptor cells and detect molecules that diffuse into the taste pore from the overlying mucus of the tongue, part of which originates from glands adjacent to the taste buds. Some stimuli can pass directly into the cell to depolarize it (Na^+ for salty and H^+ for sour) or open cation channels to generate a receptor potential (also salty and sour), whereas others (sucrose, quinine, and glutamate for sweet, bitter, and umami) activate a second messenger that can either open cation channels or directly activate intracellular Ca^{++} stores (see Fig. 8.29*B*). In each case, depolarization of the receptor results in the release of glutamate, and consequently, action potentials in the primary afferent nerve fiber that are transmitted to the CNS.

Coding of taste, however, is not based entirely on the selectivity of the chemoreceptors for the different primary qualities because each cell responds to a range of stimuli, although most intensely to one. Because most natural tastes have chemicals that effect responses from a number of chemoreceptors, recognition of taste quality appears to depend on the patterned input from a population of chemoreceptors, each responding differentially to the components of the stimulus. The intensity of the stimulus is reflected in the total amount of activity evoked.

Distribution and Innervation of Taste Buds

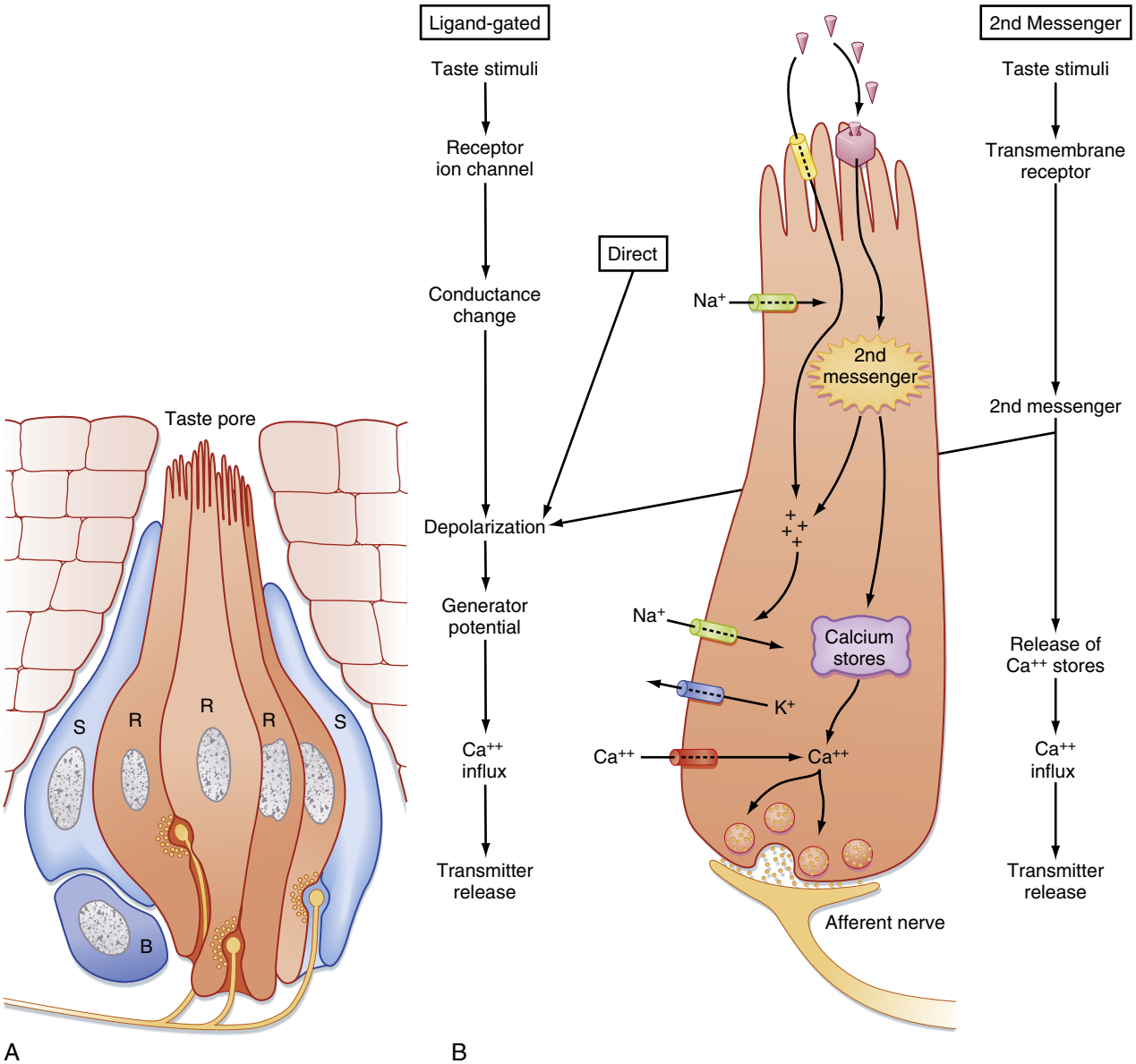
Taste buds are located on different types of taste papillae found on the tongue, palate, pharynx, and larynx. Types of taste papillae include **fungiform** and **foliate papillae** on the anterior and lateral aspects, respectively, of the tongue and **circumvallate papillae** on the base of the tongue (see Fig. 8.29*C*). The circumvallate papillae may contain several hundred taste buds. The tongue in humans may have several thousand taste buds. The sensitivity of different regions of the tongue for different taste qualities varies slightly because taste buds responding to each type of taste are widely distributed. The taste buds are innervated by three cranial nerves. The chorda tympani branch of the **facial nerve** (CN VII) innervates taste buds on the anterior two thirds of the tongue, and the **glossopharyngeal nerve** (CN IX) innervates taste buds on the posterior third of the tongue (see Fig. 8.29*C*). The **vagus nerve** (CN X) innervates a few taste buds in the larynx and upper esophagus.

Central Taste Pathways

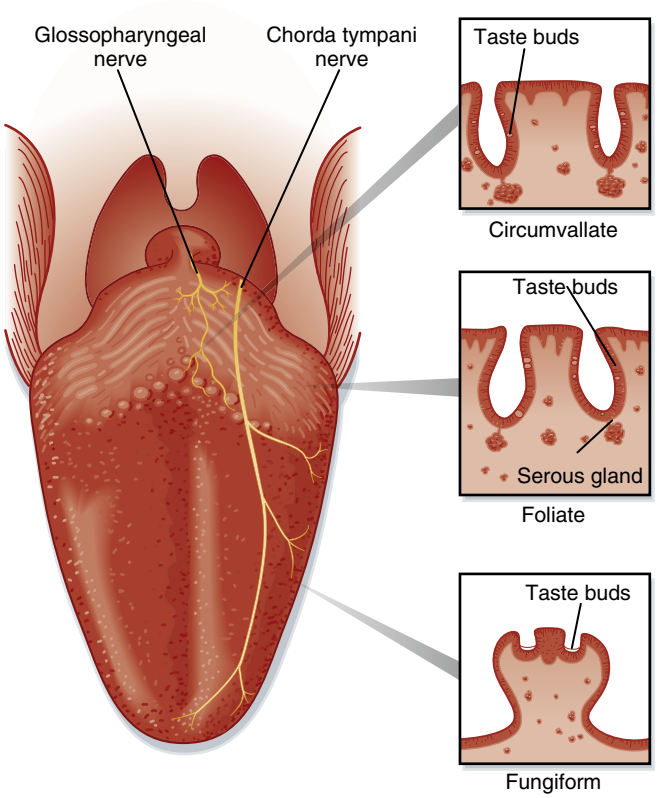
The cell bodies of taste fibers in cranial nerves VII, IX, and X are located in the **geniculate, petrosal,** and **nodose ganglia,** respectively. The central processes of the afferent fibers enter the medulla, join the solitary tract, and synapse in the **nucleus of the solitary tract** (see Fig. 4.6*D–E*). In some animals, including several rodent species, the second-order taste neurons of the solitary nucleus project rostrally to the ipsilateral parabrachial nucleus. The parabrachial nucleus then projects to the small-celled (parvocellular) part of the **ventroposterior medial** (VPMpc) nucleus of the thalamus. In monkeys, the solitary nucleus projects directly to the VPMpc nucleus. The VPMpc nucleus is connected to two different gustatory areas of the cerebral cortex: one in the face area of the S1 cortex and the other in the insula. An unusual feature of the central gustatory pathway is that it is predominantly an uncrossed pathway (unlike the central somatosensory pathways, which are predominantly crossed).

Olfaction

The sense of smell is much better developed in some animals **(macrosmatic animals; e.g., pigs, bears, and dogs)** than in humans. The ability of dogs to track other animals on the basis of odor is legendary, as is the use of **pheromones** by insects to attract mates. However, olfaction contributes to humans' emotional life, and odors can effectively conjure up memories. It also helps people avoid consuming



• **Fig. 8.29 A,** A taste bud is shown with the taste pore at the top and its innervation below. *B,* Basal cell; *R,* ciliated taste receptor cells; *S,* supporting cells. **B,** Taste receptor cell showing second messenger, ligand-gated, and direct depolarization resulting in depolarization of the cell.



C

• **Fig. 8.29, cont'd C,** Distribution of the taste buds on the tongue and their innervation. (Redrawn from Squire LR, et al [eds]. *Fundamental Neuroscience.* San Diego, CA: Academic Press; 2002.)

spoiled food and detect dangerous situations. For example, an unpleasant odorant is added to odorless, colorless natural gas so that people can easily detect a leak.

Odor has more primary qualities than taste does.[c] As many as 1000 different odor receptors are coded in the human genome, and although only approximately 350 types are functional, they represent the largest population of G protein–coupled receptors in the genome. The olfactory mucosa also contains somatosensory receptors of the trigeminal nerve. When performing clinical tests of olfaction, clinicians must avoid activating these somatosensory receptors with thermal or noxious stimuli, such as the ammonia used in "smelling salts."

Olfactory Receptors

The olfactory chemoreceptor cells are located in the **olfactory mucosa,** a specialized part of the nasopharynx. Olfactory chemoreceptors are bipolar nerve cells (Fig. 8.30). The nonmotile cilia on the apical surface of these cells contain chemoreceptors that detect odorant chemicals dissolved in the overlying mucus layer. From its opposite side, the cell gives off an unmyelinated axon that joins other **olfactory nerve filaments** and penetrates the base of the skull through openings in the **cribriform plate** of the ethmoid bone. These olfactory nerves synapse in the **olfactory bulb,** a portion of the cerebral hemisphere of the brain located at the base of the cranial cavity, just below the frontal lobe (Fig. 8.31).

Humans have about 10 million olfactory chemoreceptors. Like taste cells, olfactory chemoreceptors have a short life span (roughly, 60 days), and they are also continuously replaced. However, olfactory receptor cells are true neurons and, as such, are one of two neuron types that are continuously regenerated throughout life (granule cells of the hippocampal dentate gyrus are the other neuron type).

The olfactory mucosa is exposed to odorant molecules by ventilatory air currents or from the oral cavity during feeding. Sniffing increases the influx of odorants. The odorants are temporarily bound in mucus to an olfactory binding protein that is secreted by a gland in the nasal cavity.

Olfactory coding resembles taste coding in that most natural odors are complex and consist of many molecules that excite a wide variety of olfactory chemoreceptors. Coding for a particular perceived odor depends on the responses of many olfactory chemoreceptors, and the strength of the odorant is represented by the overall amount of afferent neural activity.

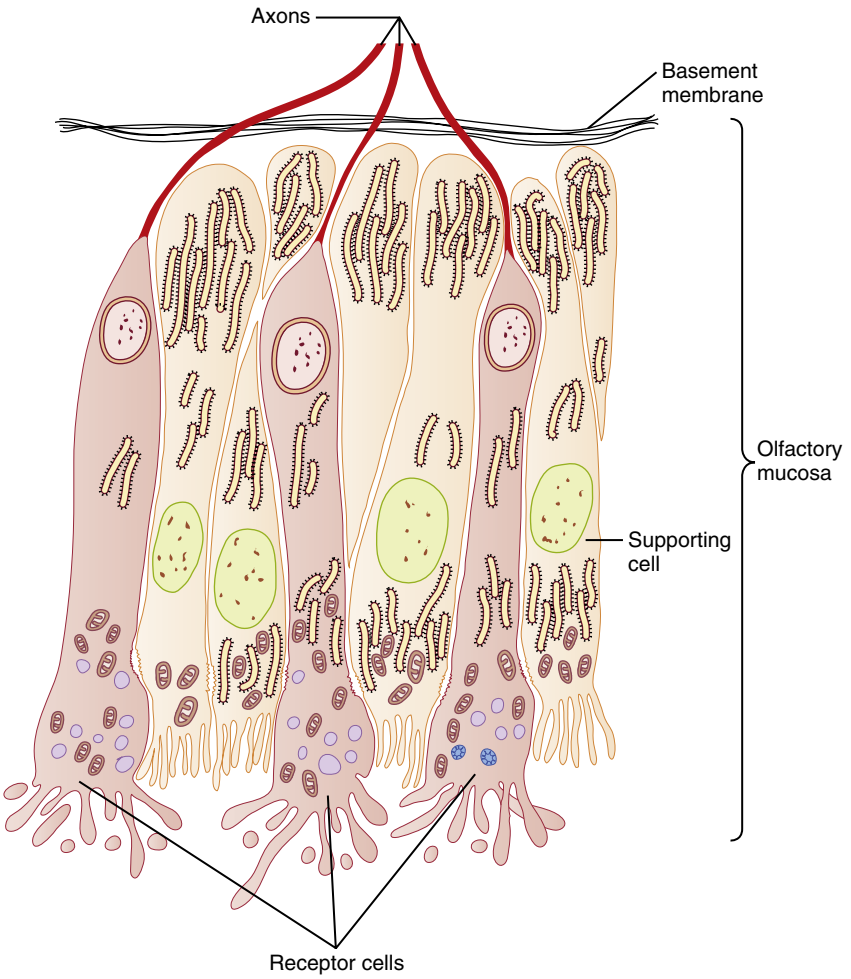
Central Pathways

The initial synapse of the olfactory pathway is located in the olfactory bulb, which is a specialized portion of the cerebral cortex and located on the underside of the frontal lobe. It contains **mitral cells,** interneurons **(granule cells; periglomerular cells),** and distinct synaptic clusters (**glomeruli**; see Fig. 8.31) in which theses interact with olfactory afferent fibers. As the olfactory afferent fibers reach the olfactory bulb from the olfactory mucosa, they branch as they approach an olfactory glomerulus to synapse on the dendrites of mitral cells. Each glomerulus is the target of thousands of olfactory afferent fibers, but all the afferent fibers to a single glomerulus convey input from the one type of olfactory receptor. This is all the more remarkable because olfactory receptor cells are being regenerated continuously and new axons must therefore navigate their way to a correct glomerulus.

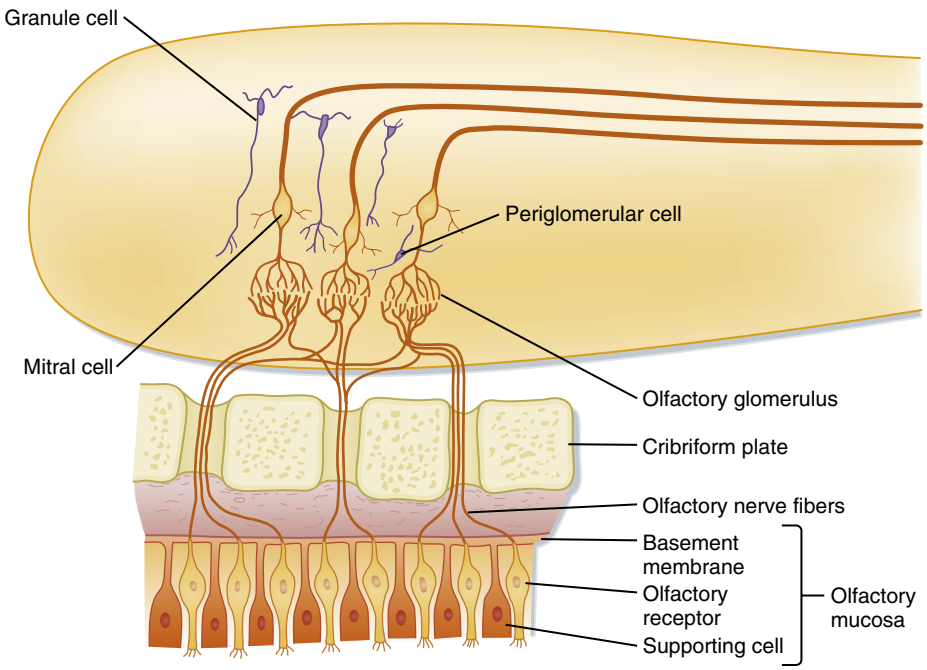
The granule and periglomerular cells are inhibitory interneurons. They form **dendrodendritic reciprocal synapses** with the dendrites of mitral cells. Activity in a mitral cell depolarizes these inhibitory cells, and they in turn inhibit the original and adjacent glomeruli. Because each glomerulus is specialized by being the target of afferent fibers for a unique combination of odor qualities, this appears to be a way of enhancing stimulus contrast, much the way horizontal cells do in the retina. In addition, it provides a mechanism for adaptation to continuous stimulation.

The axons of mitral cells leave the olfactory bulb and enter the olfactory tracts. From here, the olfactory connections

[c]The conscious perception of **flavor,** particularly of foods, is the result of both olfactory and gustatory input based on directly inhaled odor, taste from the food as it is macerated in the mouth, and retronasal odor from the volatile molecules that are released by maceration and pass up into the nasal cavity from the pharynx.



• **Fig. 8.30** Olfactory chemoreceptors and supporting cells. (Redrawn from de Lorenzo AJD. In: Zotterman Y, ed. *Olfaction and Taste.* Elmsford, NY: Pergamon; 1963.)



• **Fig. 8.31** Drawing of a sagittal section through an olfactory bulb, showing terminations of the olfactory chemoreceptor cells in the olfactory glomeruli and the intrinsic neurons of the olfactory bulb. The axons of the mitral cells are shown exiting in the olfactory tract to the right. (Modified from House EL, Pansky B. *A Functional Approach to Neuroanatomy.* 2nd ed. New York: McGraw-Hill; 1967.)

become highly complex. Within the olfactory tracts is a nucleus, called the **anterior olfactory nucleus,** that receives input from the olfactory bulb and projects to the contralateral olfactory bulb through the **anterior commissure.** As each olfactory tract approaches the base of the brain, it splits into the **lateral** and **medial olfactory striae.** Axons of the lateral olfactory stria synapse in the primary olfactory cortex, which includes the **prepiriform cortex** (and, in many animals, the piriform lobe). The medial olfactory stria includes projections to the **amygdala,** as well as to the basal forebrain. These structures are portions of, or directly connected to, the limbic system (see Chapter 10).

Of note is that the olfactory pathway is the only sensory system that does not have an obligatory synaptic relay in the thalamus before signals reach the cortex. However, olfactory information does reach the mediodorsal nucleus of the thalamus, and it is then transmitted to the prefrontal and orbitofrontal cortex. The functional roles of olfaction, in addition to the conscious perception of odor, include providing much of the subtleties of taste by enhancing the narrow range of gustatory receptors with the wide repertoire of olfactory receptors. In addition, via its intimate connections with limbic and, by extension, hypothalamic structures, it provides input to subconscious mechanisms related to emotions, memory, and sexual behavior.

IN THE CLINIC

Olfaction is not generally examined in a routine neurological examination. However, smell can be tested by having the patient inhale and identify an odorant. One nostril should be examined at a time while the other nostril is occluded. Strong odorants, such as ammonia, should be avoided because they also activate trigeminal nerve fibers. Smell sensation can be lost **(anosmia)** after a basal skull fracture, or after damage to one or both olfactory bulbs or tracts by a tumor (such as an **olfactory groove meningioma**). Concussions can cause anosmia because the sudden movement of the brain inside the skull can shear the small unmyelinated olfactory nerve fibers. An aura of a disagreeable odor, often the smell of burning rubber, occurs during **uncinate seizures,** which are epileptic seizures that originate in the medial temporal lobe.

Key Points

1. Light enters the eye through the cornea and lens and is focused on the retina, which lines the back of the eye. The cornea is the most powerful refractive surface, but the lens has a variable power that allows images of near objects to be focused on the retina. The iris regulates depth of field and the amount of illumination that enters the eye.
2. The outer segments of the photoreceptor cells transduce light. Photoreceptors synapse on retinal bipolar cells, which in turn synapse on other interneurons and on ganglion cells. The ganglion cells project to the brain through the optic nerve. The optic disc, where the optic nerve leaves the retina, contains no photoreceptors and is therefore a blind spot. The portion of the retina with the highest degree of spatial resolution is the fovea and the surrounding macula.
3. Rod photoreceptors have high sensitivity, do not discriminate among colors, and function best under low-light levels. Cone photoreceptors have lower sensitivity but higher spatial resolution. Color vision relies on the three types of cones that have different spectral sensitivities.
4. Bipolar cells and many ganglion cells have concentric receptive fields with an on-center/off-surround or off-center/on-surround organization. Horizontal cells mediate this center-surround antagonism. Photoreceptor, bipolar, and horizontal cells respond to stimulation by modulating their membrane potential and their release of neurotransmitters, but ganglion cells respond by generating action potentials.
5. The axons of ganglion cells in the temporal retina project to the brain ipsilaterally; those in the nasal retina cross in the optic chiasm. Because the lens inverts the image that falls on the retina, each side of the visual field is projected to the contralateral side of the brain for both eyes. In the lateral geniculate nucleus (LGN) of the thalamus, the input from each eye terminates in separate layers, and the M ganglion cells (sensitive to movement) and P ganglion cells (sensitive to detail and color) project to separate layers as well.
6. The LGN projects to primary visual (striate) cortex via the visual radiation and terminates largely in layer 4, where there is an orderly retinotopic map. Within the map, information from each eye maps to alternating adjacent points to create ocular dominance columns that extend vertically in the cortex. Striate cortical neurons outside of layer 4 respond best to bar or edge stimuli oriented in a particular way. Cells that "prefer" a particular stimulus orientation are grouped in orientation columns.
7. The extrastriate visual areas have different functions. Some in the inferotemporal cortex are influenced chiefly by P cells, and they function in form detection, color vision, and face discrimination. M cells influence regions of the middle temporal and parietal cortex, which function in motion detection and the control of eye movements.
8. A pure tone is characterized in terms of its amplitude, frequency, and phase. Natural sounds are combinations of pure tones. Sound pressure is measured in decibels (dB), in relation to a reference level.

9. The pinna and auditory canal convey airborne sound waves to the tympanic membrane. The three small bones (ossicles) of the middle ear transmit the vibrations of the tympanic membrane to the oval window of the fluid-filled inner ear. Hearing is most sensitive at about 3000 Hz because of the dimensions of the auditory canal and the mechanics of the ossicles.
10. The cochlea of the inner ear has three main compartments: the scala vestibuli, the scala tympani, and the intervening scala media (cochlear duct). The cochlear duct is bounded on one side by the basilar membrane, on which lies the organ of Corti, the sound transduction mechanism.
11. When the basilar membrane oscillates in response to pressure waves introduced into the scala vestibuli at the oval window, the stereocilia of the hair cells of the organ of Corti are subjected to shear forces, which open mechanoreceptor K^+ channels. This results in a membrane conductance change that modulates the release of neurotransmitters on to cochlear nerve fibers.
12. High-frequency sounds best activate the hair cells near the base of the cochlea, and low-frequency sounds activate cells near the apex. Such a tonotopic organization is also present in central auditory structures, including the cochlear nuclei, superior olivary complex, inferior colliculus, medial geniculate nucleus, and primary auditory cortex.
13. Auditory processing at many sites in the central auditory pathway contributes to sound localization, frequency and intensity analysis, and speech recognition.
14. The vestibular apparatus is part of the inner ear. It includes three semicircular canals (horizontal, anterior, and posterior) and two otolith organs (utricle and saccule) on each side. These transduce, respectively, angular and linear accelerations of the head. The three semicircular canals are mutually orthogonal, so they can resolve angular acceleration of the head about any axis of rotation.
15. In each semicircular canal, there are sensory hair cells whose cilia extend into a cupula, which blocks the cross-section of the endolymph-filled canal. Angular head acceleration displaces the endolymph and the cupula, bending the cilia. If the stereocilia bend toward the kinocilium, the hair cell is depolarized, which causes an increase in the firing rate in the afferent fiber.
16. In the otolith organs, the cilia project into an otolithic membrane. Acceleration of the head, as with linear movement, or change in position in relation to gravity displaces the otolithic membrane (because of the mass of the otoliths) and changes the firing patterns of the hair cells, depending on their orientation.
17. Central vestibular pathways include afferent connections to the vestibular nuclei and the cerebellum. Activation of the vestibular afferent fibers is detected by the brain as head acceleration or position change and is relayed via the vestibular nuclei to pathways that mediate compensatory eye movements, neck movements, and adjustments to posture.
18. Taste buds contain chemoreceptor cells arranged around a taste pore. Taste buds are located on several kinds of papillae on the tongue and in the pharynx and larynx. Five types of taste-receptor cells detect the five elementary qualities of taste: salty, sweet, sour, bitter, and umami. Complex flavors are signaled by the patterned activity of multiple classes of taste receptor and by central correlation with accompanying olfactory input.
19. Afferent taste fibers synapse in the nucleus of the solitary tract. The thalamic relay is via a part of the ventroposterior medial nucleus to the taste-receiving areas located in the S1 cortex and the insula.
20. Odors are detected by olfactory chemoreceptor cells, which are continuously regenerated in the olfactory mucosa. These cells are true neurons that are endowed with a wide array of G protein–coupled receptors that enable the detection of hundreds of odor molecules.
21. Individual olfactory axons project to olfactory glomeruli, specific for each stimulus type, in the olfactory bulb. They synapse on the dendrites of mitral cells, which have reciprocal synapse with inhibitory interneurons. This synaptic organization in the glomerulus underlies stimulus adaptation and contrast enhancement.