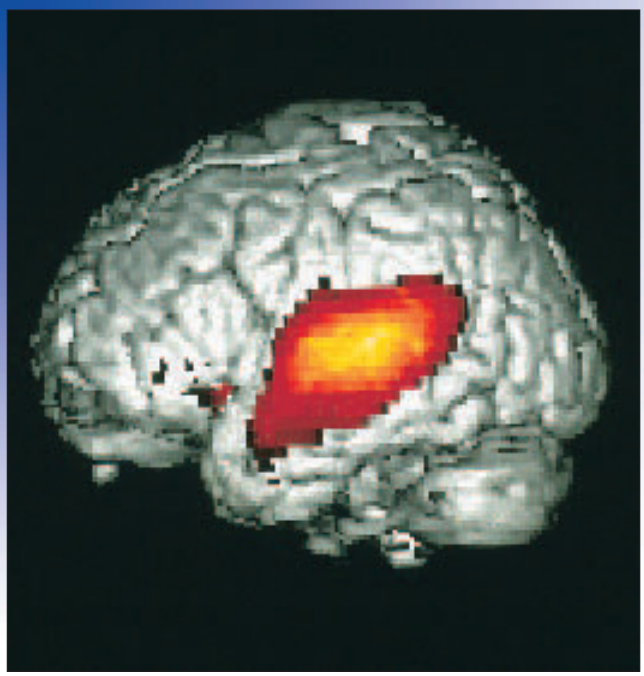


PET scan of the brain hearing sound, showing activity in the temporal lobe

CHAPTER

16

Sense Organs

CHAPTER OUTLINE

INSIGHTS

Brushing Up

To understand this chapter, it is important that you understand or brush up on the following concepts:

- Excitatory and inhibitory postsynaptic potentials (EPSPs and IPSPs) (pp. 468-469)
- Spatial summation (p. 470)
- Neural coding (p. 470)
- Converging circuits of neurons (p. 472)
- Spinal cord tracts (p. 486)
- Decussation (p. 486)

Anyone who enjoys music, art, fine food, or a good conversation appreciates the human senses. Yet their importance extends beyond deriving pleasure from the environment. In the 1950s, behavioral scientists at Princeton University studied the methods used by Soviet Communists to extract confessions from political prisoners, including solitary confinement and sensory deprivation. Student volunteers were immobilized in dark soundproof rooms or suspended in dark chambers of water. In a short time, they experienced visual, auditory, and tactile hallucinations, incoherent thought patterns, deterioration of intellectual performance, and sometimes morbid fear or panic. Similar effects have been seen in burn patients who are immobilized and extensively bandaged (including the eyes) and thus suffer prolonged lack of sensory input. Patients connected to life-support equipment and confined under oxygen tents sometimes become delirious. Sensory input is vital to the integrity of personality and intellectual function.

Furthermore, much of the information communicated by the sense organs never comes to our conscious attention—blood pressure, body temperature, and muscle tension, for example. By monitoring such conditions, however, the sense organs initiate somatic and visceral reflexes that are indispensable to homeostasis and to our very survival in a ceaselessly changing and challenging environment.

Properties and Types of Sensory Receptors

Objectives

When you have completed this section, you should be able to

- define *receptor* and *sense organ;*
- list the four kinds of information obtained from sensory receptors, and describe how the nervous system encodes each type; and
- outline three ways of classifying receptors.

A **receptor** is any structure specialized to detect a stimulus. Some receptors are simple nerve endings, whereas others are **sense organs**—nerve endings combined with connective, epithelial, or muscular tissues that enhance or moderate the response to a stimulus. Our eyes and ears are obvious examples of sense organs, but there are also innumerable microscopic sense organs in our skin, muscles, joints, and viscera.

General Properties of Receptors

All sensory receptors are transducers. A *transducer* is any device that converts one form of energy to another—a microphone, light bulb, or gasoline engine, for example. Sensory transducers convert stimulus energy into electrochemical energy—a meaningful pattern of action potentials. This process of conversion is called **sensory transduction.**

The effect of a stimulus on a receptor is to produce a type of local potential called a **receptor potential**—a graded voltage change across the plasma membrane of the receptor cell. The receptor potential may cause a receptor cell (which is not always a neuron) to release a neurotransmitter that stimulates an adjacent neuron. If the receptor itself is a neuron and the voltage reaches threshold, the neuron fires impulses to the central nervous system (CNS). This may result in a **sensation**—a conscious awareness of the stimulus—but much of the sensory information reaching the CNS produces no sensation. We are seldom aware of information concerning muscle tension and blood pH, for example, but it is vitally important to our homeostasis for the CNS to monitor these conditions.

Sensory receptors transmit four kinds of information—*modality, location, intensity,* and *duration:*

1. **Modality** refers to the type of stimulus or the sensation it produces. Vision, hearing, and taste are examples of sensory modalities. The nervous system distinguishes modalities from each other partly by means of a *labeled line code.* We can think of the brain as having numerous "lines" (nerve fibers) feeding information into it, and each line as being "labeled" to represent a certain modality. All the nerve impulses that arrive at the brain are essentially identical, but impulses arriving on one line have a different meaning than impulses arriving on another. Any impulses from the optic nerve, for example, are interpreted as light. Thus, a blow to the eye may be perceived as a flash of light.
2. **Location** is also encoded by which nerve fibers are firing. A sensory neuron receives input from an area called its **receptive field.** The brain's ability to determine the location of a stimulus depends on the size of this field. In tactile (touch) neurons, for example, a receptive field on one's back may be as big as 7 cm in diameter. Any touch within that area stimulates one neuron, so it is difficult to tell precisely where the touch occurs. Being touched at two points 5 cm apart within the same field would feel like a single touch. On the fingertips, by contrast, receptive fields may be less than 1 mm in diameter. Two points of contact just 2 mm apart would thus be felt separately (fig. 16.1). Thus, we say the fingertips have finer *two-point discrimination* than the skin on the back. This is crucial to such functions as feeling textures and reading Braille. **Sensory projection** is the ability of the brain to identify the site of stimulation, including very small and specific areas within a receptor such as the retina. The pathways followed by sensory signals to their ultimate destinations in the CNS are called **projection pathways.**
3. **Intensity** can be encoded in three ways: (a) as stimulus intensity rises, the firing frequencies of sensory nerve fibers rise (see fig. 12.25, p. 471); (b) intense stimuli recruit greater numbers of nerve

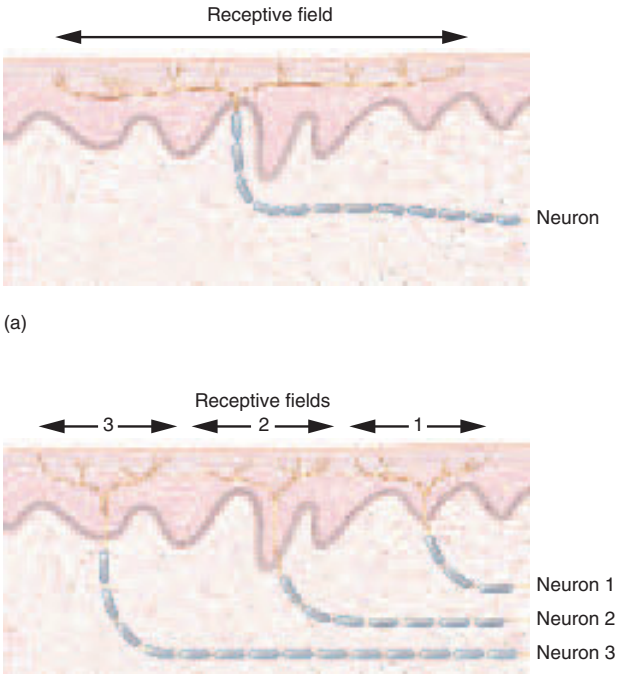


Figure 16.1 Receptive Fields. (*a*) A neuron with a large receptive field, as found in the skin of the back. If the skin is touched in two close-together places within this receptive field, the brain will sense it as only one point of contact. (*b*) Neurons with small receptive fields, as found in the fingertips. Two close-together points of contact here are likely to stimulate two different neurons and to be felt as separate touches. **If the receptive field in figure *a* is 7 cm in diameter, is it possible for two touches 1 cm apart to be felt separately?**

fibers to fire; and (c) weak stimuli activate only the most sensitive nerve fibers, whereas strong stimuli can activate a less sensitive group of fibers with higher thresholds. Thus, the brain can distinguish intensities based on the number and kind of fibers that are firing and the time intervals between action potentials. These concepts were discussed under *neural coding* in chapter 12.

4. **Duration** is encoded in the way nerve fibers change their firing frequencies over time. **Phasic receptors** generate a burst of action potentials when first stimulated, then quickly adapt and sharply reduce or halt signal transmission even if the stimulus continues. Some of them fire again when the stimulus ceases. Lamellated corpuscles, tactile receptors, hair receptors, and smell receptors are rapidly adapting phasic receptors. **Tonic receptors** adapt slowly and generate nerve impulses more steadily. Proprioceptors are among the most slowly adapting tonic receptors because the brain must always be aware of body position, muscle tension, and joint motions. All receptors, however, exhibit sensory **adaptation**—if the stimulus is prolonged, firing frequency and conscious sensation decline. Adapting to hot bath water is an example.

Think About It

Although you may find it difficult to immerse yourself in a tub of hot water or a cold lake, you soon adapt and become more comfortable. In light of this, do you think cold and warm receptors are phasic or tonic? Explain.

Classification of Receptors

Receptors can be classified by several overlapping systems:

1. By stimulus modality:
   - **Chemoreceptors** respond to chemicals, including odors, tastes, and body fluid composition.
   - **Thermoreceptors** respond to heat and cold.
   - **Nociceptors**[1] (NO-sih-SEP-turs) are pain receptors; they respond to tissue damage resulting from trauma (blows, cuts), ischemia (poor blood flow), or excessive stimulation by agents such as heat and chemicals.
   - **Mechanoreceptors** respond to physical deformation caused by vibration, touch, pressure, stretch, or tension. They include the organs of hearing and balance and many receptors of the skin, viscera, and joints.
   - **Photoreceptors,** the eyes, respond to light.
2. By the origins of the stimuli:
   - **Interoceptors** detect stimuli in the internal organs and produce feelings of visceral pain, nausea, stretch, and pressure.
   - **Proprioceptors** sense the position and movements of the body or its parts. They occur in muscles, tendons, and joint capsules.
   - **Exteroceptors** sense stimuli external to the body; they include the receptors for vision, hearing, taste, smell, touch, and cutaneous pain.
3. By the distribution of receptors in the body. There are two broad classes of senses:
   - **General (somesthetic) senses,** with receptors that are widely distributed in the skin, muscles, tendons, joint capsules, and viscera. These include the sense of touch, pressure, stretch,

[1] *noci* = pain

heat, cold, and pain, as well as many stimuli that we do not perceive consciously, such as blood pressure and composition.

- **Special senses,** which are limited to the head and innervated by the cranial nerves. The special senses are vision, hearing, equilibrium, taste, and smell.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

1. What is the difference between a receptor and a nerve ending?
2. Three schemes of receptor classification were presented in this section. In each scheme, how would you classify the receptors for a full bladder? How would you classify taste receptors?
3. What does it mean to say sense organs are transducers? What form of energy do all receptors have as their output?
4. Nociceptors are tonic rather than phasic receptors. Speculate on why this is beneficial to homeostasis.

The General Senses

Objectives

When you have completed this section, you should be able to

- list several types of somesthetic receptors;
- describe the projection pathways for the general senses; and
- explain the mechanisms of pain and the spinal blocking of pain signals.

Receptors for the general senses are relatively simple in structure and physiology. They consist of one or a few sensory nerve fibers and, usually, a sparse amount of connective tissue. These receptors are shown in table 16.1.

Unencapsulated Nerve Endings

Unencapsulated nerve endings are sensory dendrites that are not wrapped in connective tissue. They include free nerve endings, tactile discs, and hair receptors:

- **Free nerve endings** include *warm receptors,* which respond to rising temperature; *cold receptors,* which respond to falling temperature; and *nociceptors* (pain receptors). They are bare dendrites that have no special association with specific accessory cells or tissues. They are most abundant in epithelia and connective tissue.
- **Tactile (Merkel[2]) discs** are tonic receptors for light touch, thought to sense textures, edges, and shapes. They are flattened nerve endings associated with specialized *tactile (Merkel) cells* at the base of the epidermis (see fig. 6.2, p. 193).
- **Hair receptors (peritrichial[3] endings)** monitor the movements of hairs. They consist of a few dendrites entwined around the base of a hair follicle. They respond to any light touch that bends a hair. Because they adapt quickly, we are not constantly annoyed by our clothing bending the body hairs. However, when an ant crawls across our skin, bending one hair after another, we are very aware of it.

Encapsulated Nerve Endings

Encapsulated nerve endings are nerve fibers wrapped in glial cells or connective tissue. Most of them are mechanoreceptors for touch, pressure, and stretch. The connective tissues around a sensory dendrite enhance the sensitivity or specificity of the receptor. We have already considered some encapsulated nerve endings in chapter 15—muscle spindles and Golgi tendon organs. Others are as follows:

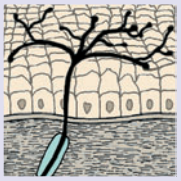
- **Tactile (Meissner[4]) corpuscles** are phasic receptors for light touch and texture. They occur in the dermal papillae of the skin, especially in sensitive hairless areas such as the fingertips, palms, eyelids, lips, nipples, and genitals. They are tall, ovoid- to pear-shaped, and consist of two or three nerve fibers meandering upward through a mass of connective tissue. Tactile corpuscles enable you to tell the difference between silk and sandpaper, for example, by light strokes of your fingertips.
- **Krause[5] end bulbs** are similar to tactile corpuscles but occur in mucous membranes rather than in the skin.
- **Lamellated (pacinian[6]) corpuscles** are phasic receptors for deep pressure, stretch, tickle, and vibration. They consist of numerous concentric layers of Schwann cells surrounding a core of one to several sensory nerve fibers. These receptors occur in the pancreas, some other viscera, and deep in the dermis—especially on the hands, feet, breasts, and genitals.
- **Ruffini[7] corpuscles** are tonic receptors for heavy touch, pressure, stretching of the skin, and joint movements. They are flattened, elongated capsules containing a few nerve fibers and are located in the dermis, subcutaneous tissue, ligaments, tendons, and joint capsules.

[2]Friedrich S. Merkel (1845–1911), German anatomist and physiologist

[3]*peri* = around + *trich* = hair
[4]George Meissner (1829–1905), German histologist
[5]William J. F. Krause (1833–1910), German anatomist
[6]Filippo Pacini (1812–83), Italian anatomist
[7]Angelo Ruffini (1864–1929), Italian anatomist

Table 16.1 Receptors of the General Senses

Unencapsulated Nerve Endings

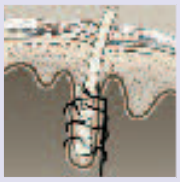


Free Nerve Endings
Location: Widespread, especially in epithelia and connective tissues

Modality: Pain, heat, cold

Tactile Discs
Location: Stratum basale of epidermis

Modality: Light touch, texture, edges, shapes



Hair Receptors
Location: Around hair follicle

Modality: Movement of hairs

Encapsulated Nerve Endings

Tactile Corpuscles
Location: Dermal papillae of fingertips, palms, eyelids, lips, tongue, nipples, and genitals
Modality: Light touch, texture

Krause End Bulbs
Location: Mucous membranes

Modality: Similar to tactile corpuscles

Lamellated Corpuscles
Location: Dermis, joint capsules, breasts, genitals, and some viscera
Modality: Deep pressure, stretch, tickle, vibration

Ruffini Corpuscles
Location: Dermis, subcutaneous tissue, and joint capsules

Modality: Heavy touch, pressure, stretching of skin, joint movements

Muscle Spindles
Location: Skeletal muscles near tendon

Modality: Muscle stretch (proprioception)

Golgi Tendon Organs
Location: Tendons

Modality: Tension on tendons (proprioception)

Somesthetic Projection Pathways

From the receptor to the final destination in the brain, most somesthetic signals travel by way of three neurons called the **first-, second-,** and **third-order neurons.** Their axons are called first- through third-order nerve fibers. The first-order fibers for touch, pressure, and proprioception are large, myelinated, and fast; those for heat and cold are small, unmyelinated, and slower.

Somesthetic signals from the head, such as facial sensations, travel by way of several cranial nerves (especially V, the trigeminal nerve) to the pons and medulla oblongata. In the brainstem, the first-order fibers of these neurons synapse with second-order neurons that decussate and end in the contralateral thalamus. Third-order neurons then complete the route to the cerebrum. Proprioceptive signals are an exception, as the second-order fibers carry these signals to the cerebellum.

Below the head, the first-order fibers enter the dorsal horn of the spinal cord. Signals ascend the spinal cord in the spinothalamic and other pathways as detailed in chapter 13 (see table 13.1 and figure 13.11). These pathways decussate either at or near the point of entry into the spinal cord, or in the brainstem, so the primary somesthetic cortex in each cerebral hemisphere receives signals from the contralateral side of the body.

Signals for proprioception below the head travel up the spinocerebellar tracts to the cerebellum. Signals from the thoracic and abdominal viscera travel to the medulla oblongata by way of sensory fibers in the vagus nerve (X).

Pain

Pain is a discomfort caused by tissue injury or noxious stimulation, and typically leading to evasive action. As undesirable as pain may seem, we would be far worse off without it. We see evidence of its value in such diseases as leprosy and diabetes mellitus, where the sense of pain is lost because of nerve damage *(neuropathy).* The absence of pain makes people unaware of minor injuries. They do not take care of them, so the injuries can become infected and grow worse, to the point that the victim may lose fingers, toes, or entire limbs. In short, pain is an adaptive and necessary sensation. It is mediated by its own specialized nerve fibers, the nociceptors. These are especially dense in the skin and mucous membranes, and occur in virtually all organs, although not in the brain. In some brain surgery, the patient must remain conscious and able to talk with the surgeon; such patients need only a local scalp anesthetic.

There are two types of nociceptors corresponding to different pain sensations. Myelinated pain fibers conduct at speeds of 12 to 30 m/sec and produce the sensation of **fast (first) pain**—a feeling of sharp, localized, stabbing pain perceived at the time of injury. Unmyelinated pain fibers conduct at speeds of 0.5 to 2.0 m/sec and produce the **slow (second) pain** that follows—a longer-lasting, dull, diffuse feeling. Pain from the skin, muscles, and joints is called **somatic pain,** while pain from the viscera is called **visceral pain.** The latter often results from stretch, chemical irritants, or *ischemia* (poor blood flow), and it is often accompanied by nausea.

Injured tissues release several chemicals that stimulate the nociceptors and trigger pain. **Bradykinin** is the most potent pain stimulus known; it is intensely painful when injected under the skin. It not only makes us aware of injuries but also activates a cascade of reactions that promote healing. Serotonin, prostaglandins, and histamine also stimulate nociceptors, as do potassium ions and ATP released from ruptured cells.

Projection Pathways for Pain

Pain signals from the face travel mainly by way of the trigeminal nerve to the pons, while signals from the neck down travel by way of spinal nerves to the dorsal horn of the spinal cord. They synapse in the dorsal horn with second-order neurons that decussate and ascend the contralateral spinothalamic tract. The gracile fasciculus carries signals for visceral pain. By any of these pathways, pain signals arrive at the thalamus, where they are relayed to neurons that carry them to their final destination in the primary somesthetic cortex (postcentral gyrus) of the cerebrum (fig. 16.2). Pain signals also travel up the spinoreticular tract to the reticular formation and ultimately to the hypothalamus and limbic system. Pain signals arriving here activate visceral, emotional, and behavioral reactions to pain.

Pain in the viscera is often mistakenly thought to come from the skin or other superficial sites—for example when the pain of a heart attack is felt "radiating" along the left shoulder and medial side of the arm. This phenomenon is called **referred pain.** It results from the convergence of neuronal pathways in the CNS. In the case of cardiac pain, for example, spinal cord segments T1 to T5 receive input from the heart as well as the chest and arm. Pain fibers from the heart and skin in this region converge on the same spinal interneurons, then follow the same pathway from there to the thalamus and cerebral cortex. The brain cannot distinguish which source the arriving signals are coming from. It acts as if it assumes that signals arriving by this path are most likely coming from the skin, since skin has more pain receptors than the heart and suffers injury more often. Knowledge of the origins of referred pain is essential to the skillful diagnosis of organ dysfunctions (fig. 16.3).

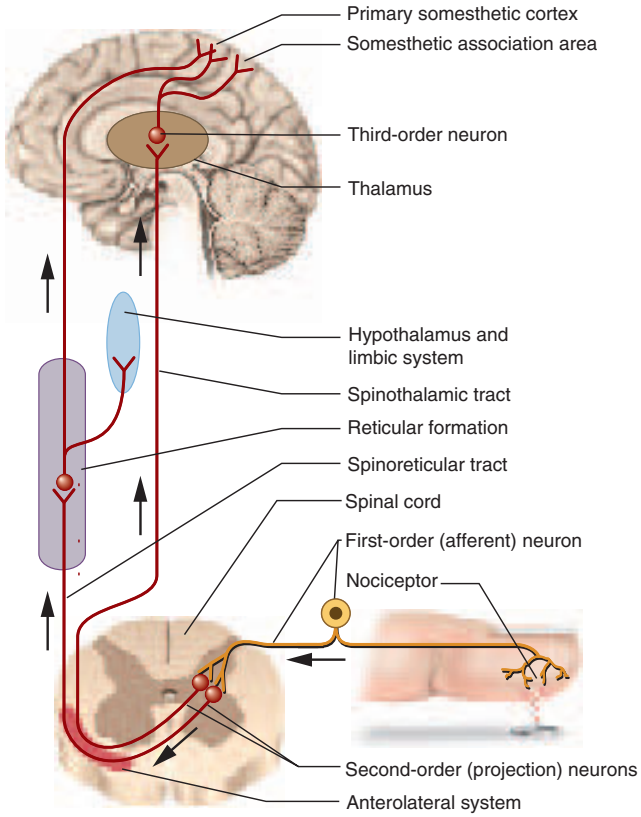


Figure 16.2 Projection Pathways for Pain. A first-order neuron conducts a pain signal to the dorsal horn of the spinal cord, a second-order neuron conducts it to the thalamus, and a third-order neuron conducts it to the cerebral cortex. Signals from the spinothalamic tract pass through the thalamus. Signals from the spinoreticular tract bypass the thalamus on the way to the sensory cortex.

CNS Modulation of Pain

A person's physical and mental state can greatly affect his or her perception of pain. Many mortally wounded soldiers, for example, report little or no pain. The central nervous system (CNS) has **analgesic**[8] (pain-relieving) mechanisms that are just beginning to be understood. The discovery of these mechanisms is tied to the long-known analgesic effects of opium, morphine, and heroin. In 1974, neurophysiologists discovered receptor sites in the brain for these drugs. Since these opiates do not occur naturally in the body, physiologists wondered what normally binds to these receptors. They soon found two analgesic oligopeptides with 200 times the potency of morphine, and named them **enkephalins.**[9] Larger analgesic neuropeptides, the

[8] *an* = without + *alges* = pain
[9] *en* = within + *kephal* = head

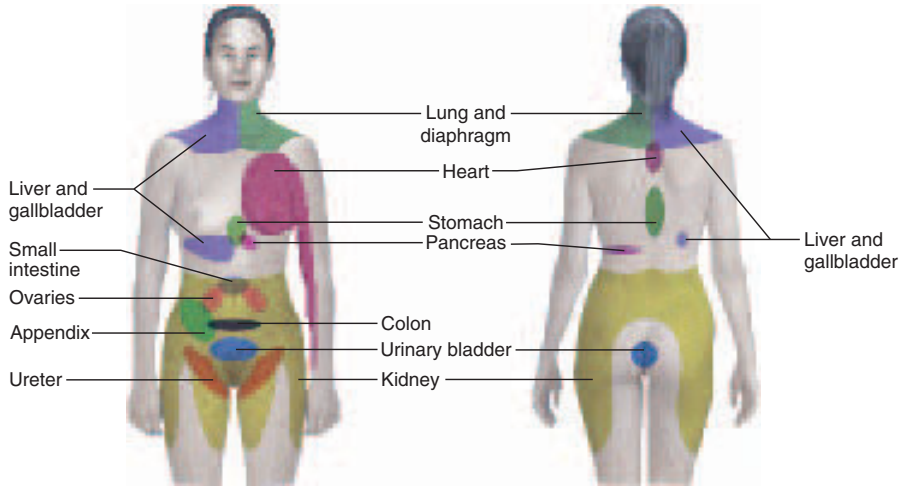


Figure 16.3 **Referred Pain.** Pain from the viscera is often felt in specific areas of the skin.

endorphins[10] and **dynorphins,**[11] were discovered later. All three of these are known as **endogenous opioids** (which means "internally produced opium-like substances").

These opioids are secreted by the CNS, pituitary gland, digestive tract, and other organs in states of stress or exercise. In the CNS, they are found especially in the central gray (periaqueductal) matter of the midbrain (see p. 527) and the dorsal horn of the spinal cord. They are *neuromodulators* (see p. 468) that can block the transmission of pain signals and produce feelings of pleasure and euphoria. They may be responsible for the "second wind" ("runner's high") experienced by athletes and for the aforementioned battlefield reports. Their secretion rises sharply in women giving birth. Efforts to employ them in pain therapy have been disappointing, but exercise is an effective part of therapy for chronic pain and may help because it stimulates opioid secretion.

How do these opioids block pain? For pain to be perceived, signals from the nociceptors must get beyond the dorsal horn of the spinal cord and travel to the brain. Through mechanisms called **spinal gating,** pain signals can be stopped at the dorsal horn. Two of these mechanisms are described here (fig. 16.4).

In one mechanism, neurons of the reticular formation issue **descending analgesic fibers** that travel down the reticulospinal tract and end on interneurons of the dorsal horn. The spinal interneurons form axoaxonic synapses with the first-order pain fibers entering the spinal cord. They secrete enkephalins and dynorphins, which inhibit the pain fibers from secreting their neurotransmitter, **substance P** (think *P* for "pain"[12]). Without substance P, the pain signal goes no farther than this; it never ascends the spinal cord to the brain, and we feel no pain.

Pain signals are also modulated by certain dorsal horn interneurons that not only inhibit the second-order neurons of the pain pathway but also receive input from mechanoreceptors. Mechanoreceptors stimulate the inhibitory interneurons, which then block the transmission of signals by the second-order pain fibers. This may explain why rubbing a sore area makes it feel less painful.

Pain control has had a particularly interesting history, some of which is retold in insight 16.5 at the end of this chapter.

Think About It

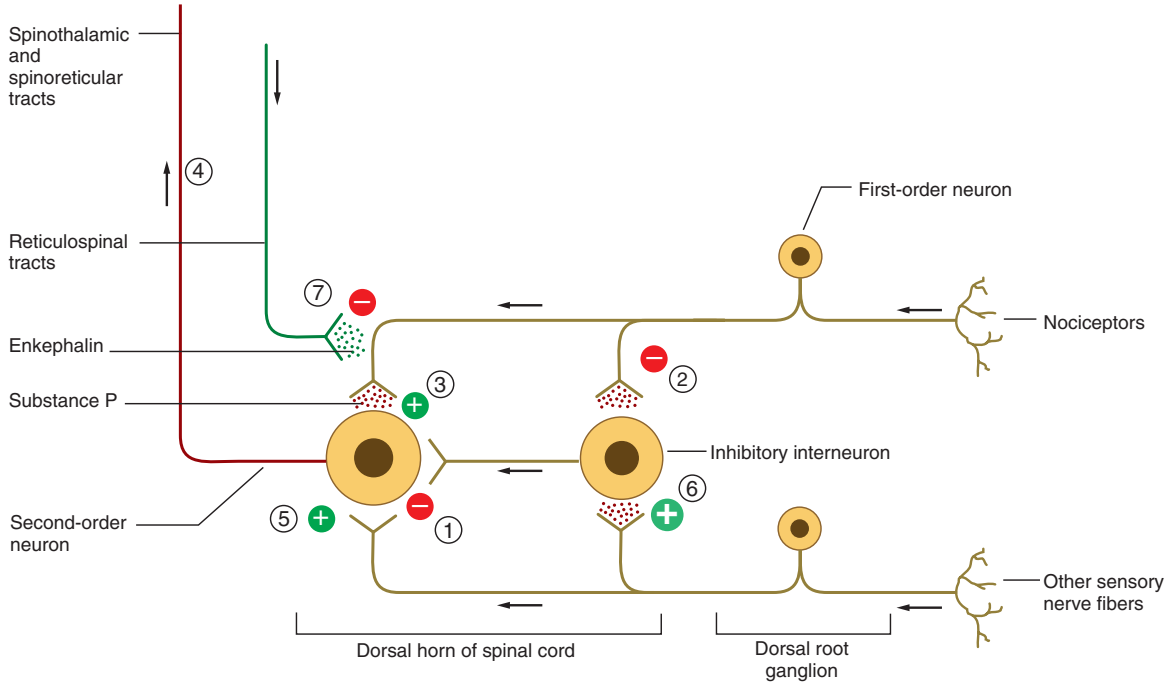
How is the phenomenon of presynaptic inhibition (see p. 470) relevant to the spinal gating of pain?

Before You Go On

Answer the following questions to test your understanding of the preceding section:

5. What stimulus modalities are detected by free nerve endings?
6. Name any four encapsulated nerve endings and identify their stimulus modalities.
7. Where do most second-order somesthetic neurons synapse with third-order neurons?
8. Explain the phenomenon of referred pain in terms of the neural pathways involved.
9. Explain the roles of bradykinin, substance P, and endorphins in the perception of pain.

[10]acronym, from *end*ogenous m*orphine*like substance
[11]*dyn* = pain
[12]Named *substance P* because it was first discovered in a *powdered* extract of brain and intestine



1. In the absence of a pain stimulus, an inhibitory interneuron of the spinal cord prevents transmission of pain signals.
2. When tissue damage stimulates a nociceptor, the nociceptor inhibits the inhibitory interneuron.
3. The nociceptor also releases substance P, which stimulates the second-order neuron.
4. The second-order neuron sends a pain signal to the brain.
5. Some sensory neurons other than nociceptors also stimulate the second-order neuron.
6. These sensory neurons, however, have an even stronger effect on the inhibitory interneuron, thus blocking the transmission of pain signals.
7. Neurons of the reticular formation release enkephalin, which, by presynaptic inhibition, blocks the release of substance P. Thus the brain can reduce the transmission of the pain signal to itself.

Figure 16.4 **Spinal Gating of Pain Signals.**

The Chemical Senses

Objectives

When you have completed this section, you should be able to

- explain how taste and smell receptors are stimulated; and
- describe the receptors and projection pathways for these two senses.

Taste and smell are the chemical senses. In both cases, receptor potentials are created by the action of environmental chemicals on sensory cells.

Taste

Taste **(gustation)** is a sensation that results from the action of chemicals on the **taste buds.** There are about 4,000 of these, located on the tongue of course, but also inside the cheeks and on the soft palate, pharynx, and epiglottis.

Anatomy

The tongue, where the sense of taste is best developed, is marked by four types of bumps called **lingual papillae** (fig. 16.5*a*):

1. **Filiform**[13] **papillae** are tiny spikes without taste buds. They are responsible for the rough feel of a cat's tongue and are important to many mammals for grooming the fur. They are the most abundant papillae on the human tongue, but they are small and play no gustatory role. They are, however, important to appreciation of the texture of food.
2. **Foliate**[14] **papillae** are also weakly developed in humans. They form parallel ridges on the sides of the tongue about two-thirds of the way back from

[13] *fili* = thread + *form* = shaped
[14] *foli* = leaf + *ate* = like

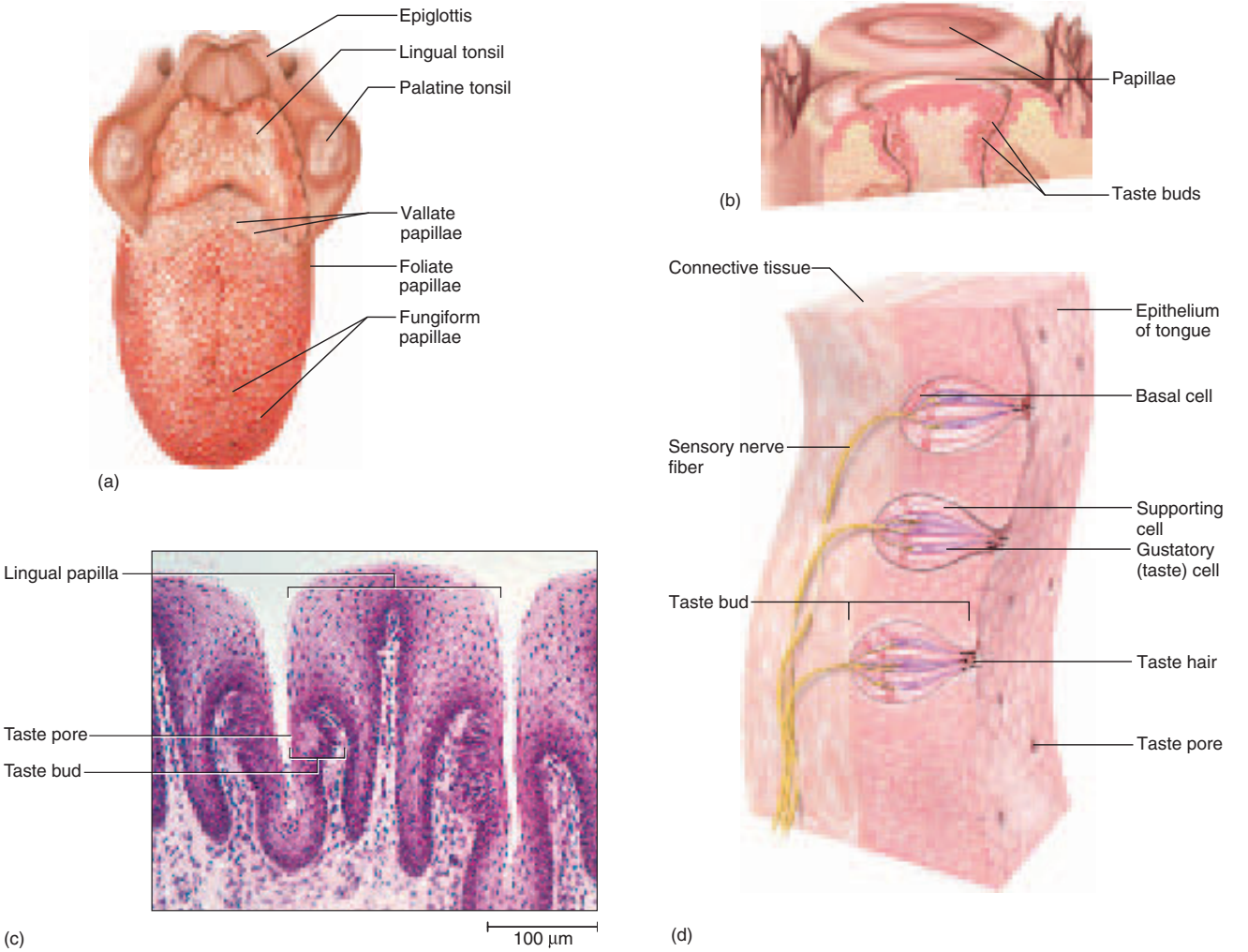


Figure 16.5 **Taste (gustatory) Receptors.** (*a*) Dorsal view of the tongue and locations of its papillae. (*b*) Detail of the vallate papillae. (*c*) Taste buds on the walls of two adjacent foliate papillae. (*d*) Structure of the taste buds.

the tip. Most of their taste buds degenerate by the age of 2 or 3 years.

3. **Fungiform**[15] (FUN-jih-form) **papillae** are shaped somewhat like mushrooms. Each has about three taste buds, located mainly on the apex. These papillae are widely distributed but especially concentrated at the tip and sides of the tongue.
4. **Vallate**[16] **(circumvallate) papillae** are large papillae arranged in a V at the rear of the tongue. Each is surrounded by a deep circular trench. There are only 7 to 12 of them, but they contain about half of all our taste buds—around 250 each, located on the wall of the papilla facing the trench (fig. 16.5*b*).

Regardless of location and sensory specialization, all taste buds look alike (fig. 16.5*c, d*). They are lemon-shaped groups of 40 to 60 cells of three kinds—*taste cells, supporting cells,* and *basal cells.* **Taste (gustatory) cells** are more or less banana-shaped and have a tuft of apical microvilli called **taste hairs** that serve as receptor surfaces for taste molecules. The hairs project into a pit called a **taste pore** on the epithelial surface of the tongue. Taste cells are epithelial cells, not neurons, but they synapse with sensory nerve fibers at their base. A taste cell lives 7 to 10 days and is then replaced by mitosis and

[15] *fungi* = mushroom + *form* = shaped
[16] *vall* = wall + *ate* = like, possessing

differentiation of basal cells. Supporting cells have a similar shape but no taste hairs. They lie between the taste cells.

Physiology

To be tasted, molecules must dissolve in the saliva and flood the taste pore. On a dry tongue, sugar or salt has as little taste as a sprinkle of sand. Physiologists currently recognize five primary taste sensations:

1. **Salty,** produced by metal ions such as sodium and potassium. Since these are vital electrolytes, there is obvious value in our ability to taste them and in having an appetite for salt. Electrolyte deficiencies can cause a craving for salt; many animals such as deer, elephants, and parrots thus seek salt deposits when necessary. Pregnancy can lower a woman's electrolyte concentrations and create a craving for salty food.
2. **Sweet,** produced by many organic compounds, especially sugars. Sweetness is associated with carbohydrates and foods of high caloric value. Many flowering plants have evolved sweet nectar and fruits that entice animals to eat them and disperse their pollen and seeds. Thus, our fondness for fruit has coevolved with plant reproductive strategies.
3. **Sour,** usually associated with acids in such foods as citrus fruits.
4. **Bitter,** associated with spoiled foods and with alkaloids such as nicotine, caffeine, quinine, and morphine. Bitter alkaloids are often poisonous, and this sensation usually induces a human or animal to reject a food. While flowering plants make their fruits temptingly sweet, they often load their leaves with bitter, toxic alkaloids to deter animals from eating them.
5. **Umami,** is a "meaty" taste produced by amino acids such as aspartic and glutamic acids. The taste is best known from the salt of glutamic acid, monosodium glutamate (MSG). Pronounced "ooh-mommy," the word is Japanese slang for "delicious" or "yummy."

The many flavors we perceive are not simply a mixture of these five primary tastes but are also influenced by food texture, aroma, temperature, appearance, and one's state of mind, among other things. Many flavors depend on smell; without its aroma, cinnamon merely has a faintly sweet taste, and coffee and peppermint are bitter. Some flavors such as pepper are due to stimulation of free endings of the trigeminal nerve. Food scientists refer to the texture of food as *mouthfeel.* Filiform and fungiform papillae of the tongue are innervated by the *lingual nerve* (a branch of the trigeminal) and are sensitive to texture.

All of the primary tastes can be detected throughout the tongue, but certain regions are more sensitive to one category than to others. The tip of the tongue is most sensitive to sweet tastes, which trigger such responses as licking, salivation, and swallowing. The lateral margins of the tongue are the most sensitive areas for salty and sour tastes. Taste buds in the vallate papillae at the rear of the tongue are especially sensitive to bitter compounds, which tend to trigger rejection responses such as gagging to protect against the ingestion of toxins. The threshold for the bitter taste is the lowest of all—that is, we can taste lower concentrations of alkaloids than of acids, salts, and sugars. The senses of sweet and salty are the least sensitive. It is not yet known whether umami stimulates any particular region of the tongue more than other regions.

Sugars, alkaloids, and glutamate stimulate taste cells by binding to receptors on the membrane surface, which then activate G proteins and second-messenger systems within the cell. Sodium and acids penetrate into the cell and depolarize it directly. By either mechanism, stimulated taste cells then release neurotransmitters that stimulate the sensory dendrites at their base.

Projection Pathways

Taste buds stimulate the facial nerve (VII) in the anterior two-thirds of the tongue, the glossopharyngeal nerve (IX) in the posterior one-third, and the vagus nerve (X) in the palate, pharynx, and epiglottis. All taste fibers project to the *solitary nucleus* in the medulla oblongata. Second-order neurons from this nucleus relay the signals to two destinations: (1) nuclei in the hypothalamus and amygdala that activate autonomic reflexes such as salivation, gagging, and vomiting, and (2) the thalamus, which relays signals to the insula and postcentral gyrus of the cerebrum, where we become conscious of the taste.

Smell

The receptor cells for smell **(olfaction)** form a patch of epithelium, the **olfactory mucosa,** in the roof of the nasal cavity (fig. 16.6). This location places the olfactory cells close to the brain, but it is poorly ventilated; forcible sniffing is often needed to identify an odor or locate its source. Nevertheless, the sense of smell is highly sensitive. We can detect odor concentrations as low as a few parts per trillion. Most people can distinguish 2,000 to 4,000 odors, and some can distinguish up to 10,000. On average, women are more sensitive to odors than men are, and they are measurably more sensitive to some odors near the time of ovulation than during other phases of the menstrual cycle. Olfaction is highly important in the social interactions of other animals and, in more subtle ways, to humans (see insight 16.1).

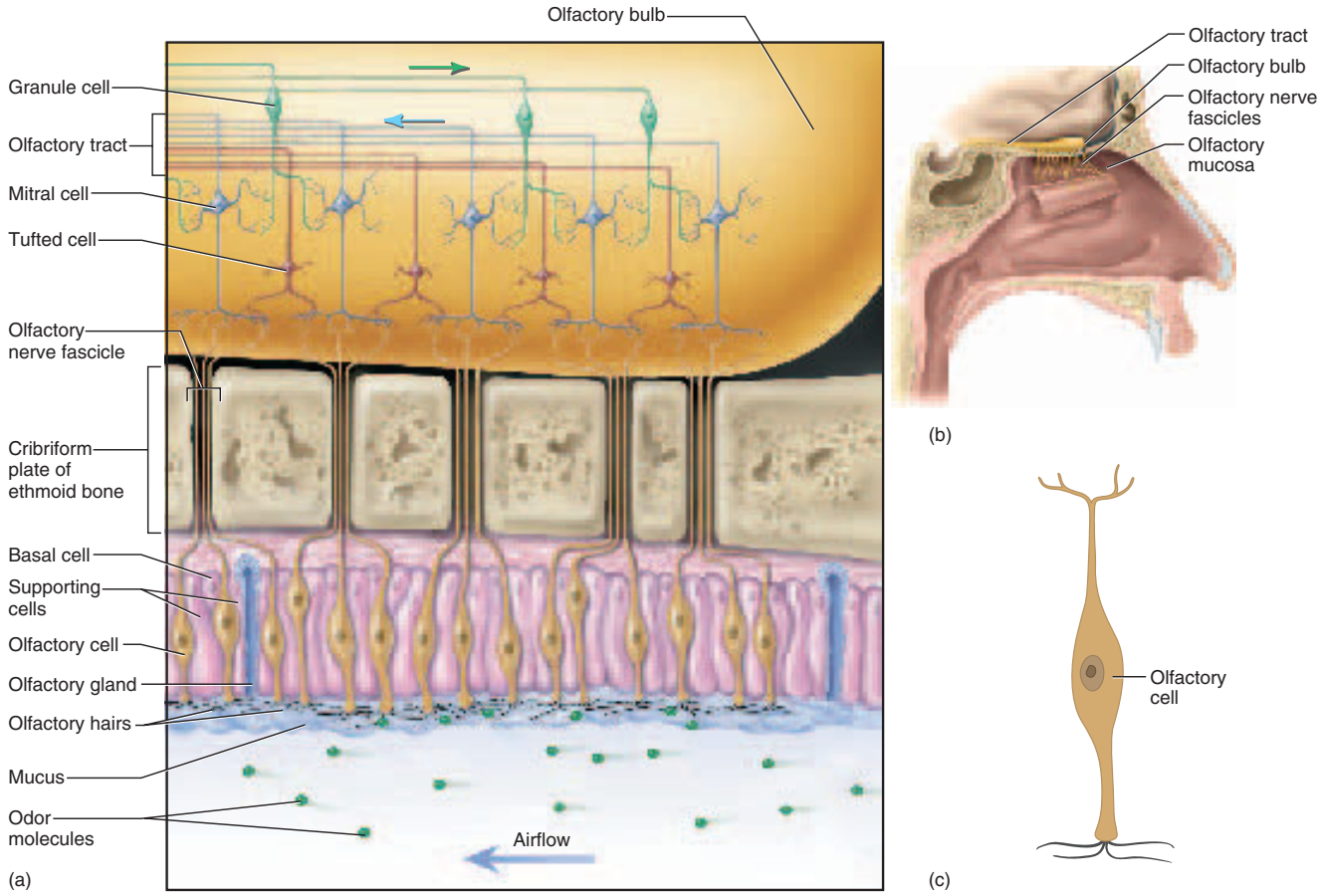


Figure 16.6 Smell (olfactory) Receptors. (*a*) Neural pathways from the olfactory mucosa of the nasal cavity to the olfactory tract of the brain. (*b*) Location of the major structures in relation to the nasal and cranial cavities. (*c*) Detail of an olfactory cell.

Insight 16.1 Evolutionary Medicine

Human Pheromones

There is an abundance of anecdote, but no clear experimental evidence, that human body odors affect sexual behavior. There is more adequate evidence, however, that a person's sweat and vaginal secretions affect other people's sexual physiology, even when the odors cannot be consciously smelled. Experimental evidence shows that a woman's apocrine sweat can influence the timing of other women's menstrual cycles. This can produce a so-called *dormitory effect* in which women who live together tend to have synchronous menstrual cycles. The presence of women stimulates men's beards to grow faster, and the presence of men seems to influence female ovulation. When a woman is ovulating or close to that time, and therefore fertile, her vaginal secretions contain pheromones called *copulines.* These have been shown to raise men's testosterone levels.

Anatomy

The olfactory mucosa covers about 5 cm^2 of the superior concha and nasal septum. It consists of 10 to 20 million **olfactory cells** as well as epithelial supporting cells and basal cells. The rest of the nasal cavity is lined by a nonsensory *respiratory mucosa.*

Unlike taste cells, which are epithelial, olfactory cells are neurons. They are shaped a little like bowling pins. The widest part, the soma, contains the nucleus. The neck and head of the cell are a modified dendrite with a swollen tip bearing 10 to 20 cilia called **olfactory hairs.** Unlike most cilia, these are immobile, but they have binding sites for odor molecules. They lie in a tangled mass embedded in a thin layer of mucus. The basal end of each cell tapers to become an axon. These axons collect into small fascicles that leave the nasal cavity through pores *(cribriform foramina)* in the ethmoid bone.

Collectively, the fascicles are regarded as cranial nerve I (the olfactory nerve).

Olfactory cells are the only neurons in the body directly exposed to the external environment. Apparently this is hard on them, because they have a life span of only 60 days. Unlike most neurons, however, they are replaceable. The basal cells continually divide and differentiate into new olfactory cells.

Physiology

Efforts to identify a few primary odors comparable to the five primary tastes have been indecisive and controversial. It is difficult even to specify what properties are needed to give a molecule an odor. At a minimum, it must be volatile—able to evaporate and be carried by the inhaled airstream—but the intensity of an odor is not simply proportional to volatility. Water is highly volatile but has no odor, while musk has a pronounced odor but is poorly volatile.

Sensory transduction begins when a molecule binds to a receptor on an olfactory hair. The receptor triggers the production of a second messenger which, in turn, opens ion channels in the membrane. Na^+ enters the cell and creates a receptor potential. In many cases, the second messenger is cAMP, but some odors activate other second-messenger systems.

Some odors stimulate nociceptors of the trigeminal nerve rather than olfactory cells; these include ammonia, menthol, chlorine, and hot peppers. "Smelling salts" are used to revive unconscious persons by strongly stimulating the trigeminal nerve with ammonia fumes.

The olfactory sense adapts quickly—we may therefore be unaware of our own body odors or have difficulty locating a gas leak in a room. Adaptation does not occur in the receptor cells but is due to synaptic inhibition in the olfactory bulbs of the brain.

Projection Pathways

When olfactory fibers pass through the cribriform plate, they enter a pair of **olfactory bulbs** beneath the frontal lobes of the brain. In the bulbs, they synapse with neurons called *mitral cells* and *tufted cells* (fig. 16.6*a*), whose axons form bundles called the **olfactory tracts.** The tracts follow a complex pathway leading to the medial side of the temporal lobes (fig. 16.7). Olfactory input to the amygdala and hypothalamus can trigger emotional and visceral reactions. For example, the odor of certain foods, perfume, a hospital, or decaying flesh can evoke strong emotional responses, and some odors can cause us to sneeze, cough, salivate, secrete stomach acid, or vomit. Olfactory signals differ from other sensory inputs in that they reach the cerebral cortex without passing through the thalamus.

The cerebral cortex sends feedback to *granule cells* in the olfactory bulbs. The granule cells, in turn, inhibit the mitral cells. An effect of this is that odors can change in quality and significance under different conditions. Food may smell more appetizing when you are hungry, for example, than it does after you have just eaten.

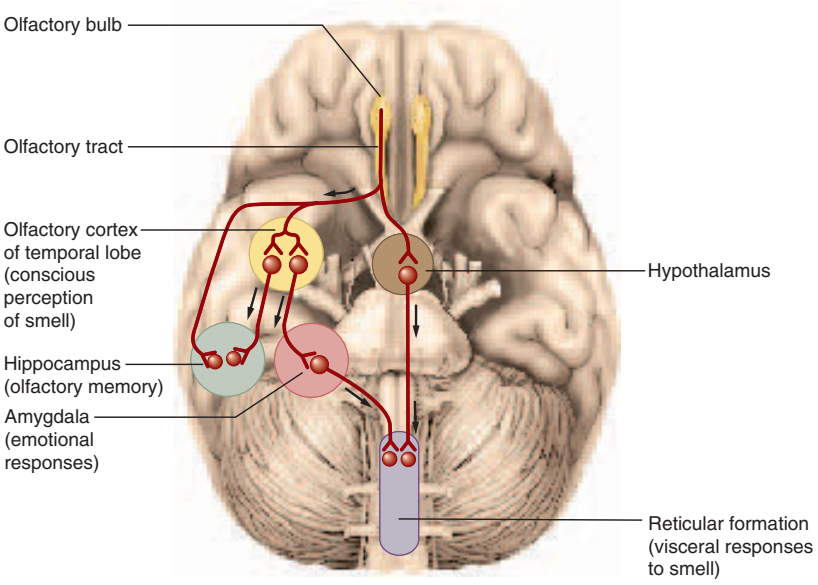


Figure 16.7 Olfactory Projection Pathways in the Brain.

Think About It

Which taste sensations could be lost after damage to (1) the facial nerve or (2) the glossopharyngeal nerve? A fracture of which cranial bone would most likely eliminate the sense of smell?

Before You Go On

Answer the following questions to test your understanding of the preceding section:

10. What is the difference between a lingual papilla and a taste bud? Which is visible to the naked eye?
11. List the primary taste sensations and discuss their adaptive significance (survival value).
12. Which cranial nerves carry gustatory impulses to the brain?
13. What part of an olfactory cell binds odor molecules?

Hearing and Equilibrium

Objectives

When you have completed this section, you should be able to

- identify the properties of sound waves that account for pitch and loudness;
- describe the gross and microscopic anatomy of the ear;
- explain how the ear converts vibrations to nerve signals and discriminates between sounds of different intensity and pitch;
- explain how the vestibular apparatus enables the brain to interpret the body's position and movements; and
- describe the pathways taken by auditory and vestibular signals to the brain.

Hearing is a response to vibrating air molecules and *equilibrium* is the sense of motion and balance. These senses reside in the inner ear, a maze of fluid-filled passages and sensory cells. This section explains how the fluid is set in motion and how the sensory cells convert this motion into an informative pattern of action potentials.

The Nature of Sound

To understand the physiology of hearing, it is necessary to appreciate some basic properties of sound. **Sound** is any audible vibration of molecules. It can be transmitted through water, solids, or air, but not through a vacuum. Our discussion is limited to airborne sound.

Sound is produced by a vibrating object such as a tuning fork, a loudspeaker, or the vocal cords. Consider a loudspeaker producing a pure tone. When the speaker cone moves forward, it pushes air molecules ahead of it. They collide with other molecules just ahead of them, and energy is thus transferred from molecule to molecule until it reaches the eardrum. No one molecule moves very far; they simply collide with each other like a series of billiard balls until finally, some molecules collide with the eardrum and make it vibrate. The sensations we perceive as the pitch and loudness of the sound are related to the physical properties of these vibrations.

Pitch

Pitch is our sense of whether a sound is "high" (treble) or "low" (bass). It is determined by the frequency at which the sound source, eardrum, and other parts of the ear vibrate. One movement of a vibrating object back and forth is called a *cycle,* and the number of cycles per second (cps or hertz, Hz) is called **frequency.** The lowest note on a piano, for example, is 27.5 Hz, middle C is 261 Hz, and the highest note is 4,176 Hz. The most sensitive human ears can hear frequencies from 20 to 20,000 Hz. The *infrasonic* frequencies below 20 Hz are not detected by the ear, but we sense them through vibrations of the skull and skin, and they play a significant role in our appreciation of music. The inaudible vibrations above 20,000 Hz are *ultrasonic.* Human ears are most sensitive to frequencies ranging from 1,500 to 4,000 Hz. In this range, we can hear sounds of relatively low energy (volume), whereas sounds above or below this range must be louder to be audible (fig. 16.8). Normal speech falls within this frequency range. Most of the hearing loss suffered with age is in the range of 250 to 2,050 Hz.

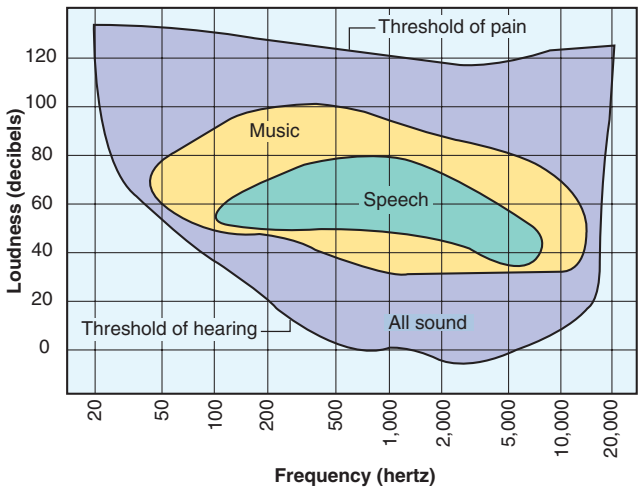


Figure 16.8 The Range of Human Hearing. People with very sensitive ears can hear sounds from 20 to 20,000 hertz, but sounds must be louder at these extremes than they are in the midrange to be heard. Our ears are most sensitive to frequencies of 1,500 to 5,000 hertz, where we can hear relatively soft sounds. Thus, the threshold of hearing varies with the frequency of the sound. Most sounds above 120 decibels are painful to the ear.

How would the shape of this graph change in a case of moderate hearing loss between 200 and 5,000 Hz?

Loudness

Loudness is the perception of sound energy, intensity, or **amplitude** of vibration. In the speaker example, amplitude is a measure of how far forward and back the cone vibrates on each cycle and how much it compresses the air molecules in front of it. Loudness is expressed in decibels (dB), with 0 dB being the threshold of human hearing. Every 10 dB step up the scale represents a sound with 10 times greater intensity. Thus, 10 dB is 10 times threshold, 20 dB is 100 times threshold, 30 dB is 1,000 times threshold, and so forth. Normal conversation has a loudness of about 60 dB. At most frequencies, the threshold of pain is 120 to 140 dB, approximately the intensity of a loud thunderclap. Prolonged exposure to sounds greater than 90 dB can cause permanent loss of hearing.

Anatomy of the Ear

The ear has three regions called the *outer, middle,* and *inner ear.* The first two are concerned only with transmitting sound to the inner ear, which houses the transducer that converts fluid motion to action potentials.

Outer Ear

The **outer (external) ear** is essentially a funnel for conducting air vibrations to the eardrum. It begins with the fleshy **auricle,** or **pinna,** on the side of the head, shaped and supported by elastic cartilage except for the earlobe. It has a predictable arrangement of named whorls and recesses that direct sound into the auditory canal (fig. 16.9).

The **auditory canal** is slightly S-shaped and about 3 cm long in adults (fig. 16.10). It is lined with skin and supported by fibrocartilage at its opening and by the temporal bone for the rest of its length. Ceruminous and sebaceous glands in the canal produce secretions that mix with dead skin cells and form *cerumen* (earwax). Cerumen normally dries and falls from the canal, but sometimes it becomes impacted and interferes with hearing.

Middle Ear

The **middle ear** is located in the **tympanic cavity** of the temporal bone. It begins with the eardrum, or **tympanic**[17] **membrane,** which closes the inner end of the auditory canal and separates it from the middle ear. The membrane is about 1 cm in diameter and slightly concave on its outer surface. It is suspended in a ring-shaped groove in the temporal bone and vibrates freely in response to sound. It is innervated by sensory branches of the vagus and trigeminal nerves and is highly sensitive to pain.

[17] *tympan* = drum

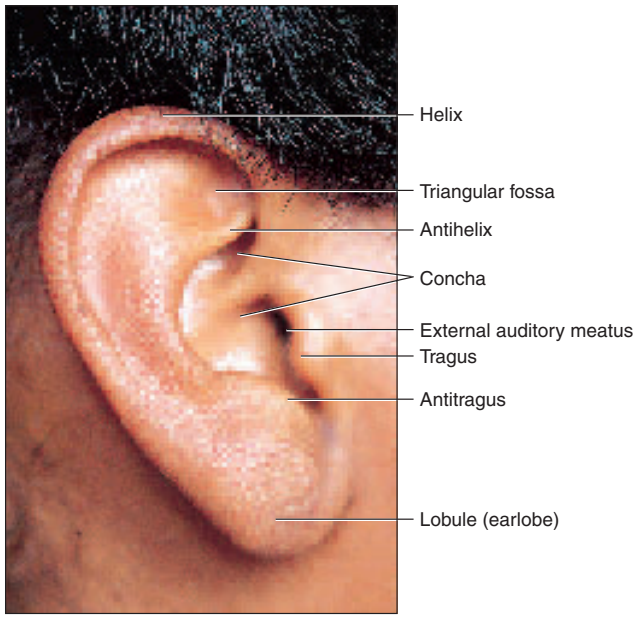


Figure 16.9 **Anatomy of the Auricle (pinna) of the Ear.**

Posteriorly, the tympanic cavity is continuous with the mastoidal air cells in the mastoid process. It is filled with air that enters by way of the **auditory (eustachian**[18]**) tube,** a passageway to the nasopharynx. (Be careful not to confuse *auditory tube* with *auditory canal.*) The auditory tube is normally flattened and closed, but swallowing or yawning opens it and allows air to enter or leave the tympanic cavity. This equalizes air pressure on both sides of the eardrum and allows it to vibrate freely. Excessive pressure on one side or the other dampens the sense of hearing. The auditory tube also allows throat infections to spread to the middle ear.

The tympanic cavity, a space only 2 to 3 mm wide between the outer and inner ear, contains the three smallest bones and two smallest skeletal muscles of the body. The bones, called the **auditory ossicles,**[19] connect the eardrum to the inner ear. Progressing inward, the first is the **malleus,**[20] which has an elongated *handle* attached to the inner surface of the eardrum; a *head,* which is suspended from the wall of the tympanic cavity; and a *short process,* which articulates with the next ossicle. The second bone, the **incus,**[21] articulates in turn with the **stapes**[22] (STAY-peez). The stapes has an arch and *footplate* that give it a

[18] Bartholomeo Eustachio (1520–74), Italian anatomist
[19] *oss* = bone + *icle* = little
[20] *malleus* = hammer
[21] *incus* = anvil
[22] *stapes* = stirrup

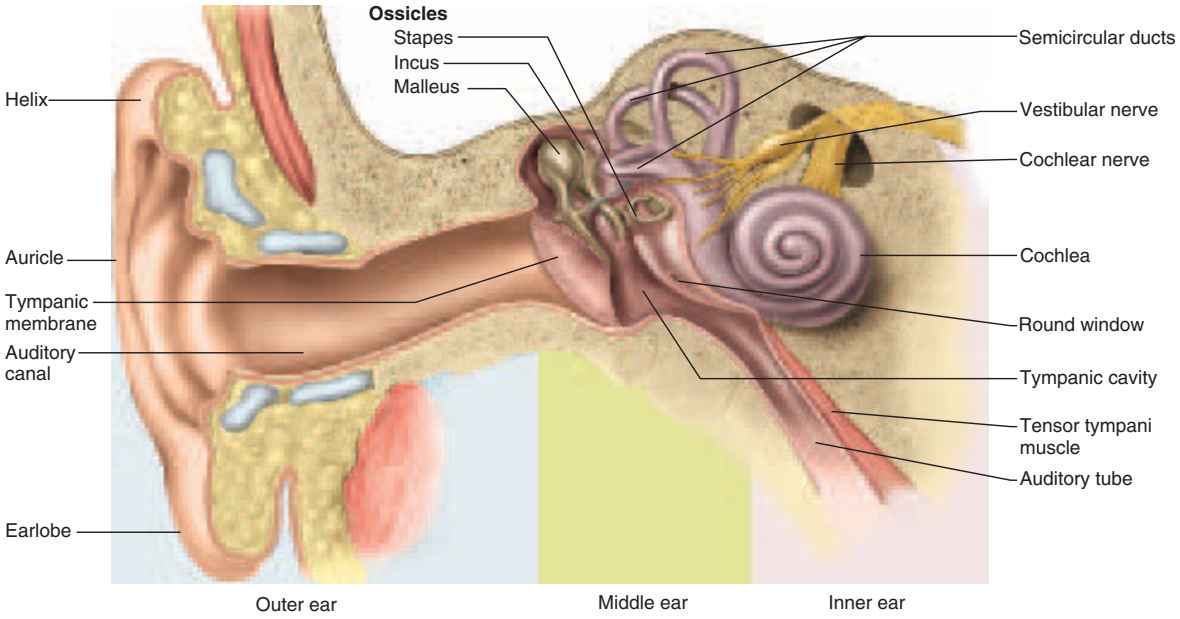


Figure 16.10 **Internal Anatomy of the Ear.**

shape like a stirrup. The footplate, shaped like the sole of a steam iron, is held by a ringlike ligament in an opening called the **oval window,** where the inner ear begins.

The muscles of the middle ear are the stapedius and tensor tympani. The **stapedius** (stay-PEE-dee-us) arises from the posterior wall of the cavity and inserts on the stapes. The **tensor tympani** (TEN-sor TIM-pan-eye) arises from the wall of the auditory tube, travels alongside it, and inserts on the malleus. The function of these muscles is discussed under the physiology of hearing.

Insight 16.2 Clinical Application

Middle-Ear Infection

Otitis[23] **media** (middle-ear infection) is common in children because their auditory tubes are relatively short and horizontal. Upper respiratory infections can easily spread from the throat to the tympanic cavity and mastoidal air cells. Fluid accumulates in the cavity and produces pressure, pain, and impaired hearing. If otitis media goes untreated, it may spread from the mastoidal air cells and cause meningitis, a potentially deadly infection (see insight 14.1). Otitis media can also cause fusion of the middle-ear bones and result in hearing loss. It is sometimes necessary to drain fluid from the tympanic cavity by lancing the eardrum and inserting a tiny drainage tube—a procedure called *myringotomy.*[24] The tube, which is eventually sloughed out of the ear, relieves the pressure and permits the infection to heal.

[23] *ot* = ear + *itis* = inflammation
[24] *myringo* = eardrum + *tomy* = cutting

Inner Ear

The **inner ear** is housed in a maze of temporal bone passageways called the **bony labyrinth,** which is lined by a system of fleshy tubes called the **membranous labyrinth** (fig. 16.11). Between the bony and membranous labyrinths is a cushion of fluid called **perilymph** (PER-ih-limf), similar to cerebrospinal fluid. Within the membranous labyrinth is a fluid called **endolymph,** similar to intracellular fluid.

The labyrinths begin with a chamber called the **vestibule,** which contains organs of equilibrium to be discussed later. The organ of hearing is the **cochlea**[25] (COC-lee-uh), a coiled tube that arises from the anterior side of the vestibule. In other vertebrates, the cochlea is straight or slightly curved. In most mammals, however, it assumes the form of a snail-like spiral, which allows a longer cochlea to fit in a compact space. In humans, the spiral is about 9 mm wide at the base and 5 mm high. Its apex points anterolaterally. The cochlea winds for about 2.5 coils around an axis of spongy bone called the **modiolus**[26] (mo-DY-oh-lus). The modiolus is shaped like a screw; its threads form a spiral platform that supports the fleshy tube of the cochlea.

A vertical section cuts through the cochlea about five times (fig. 16.12*a*). A single cross section looks like

[25] *cochlea* = snail
[26] *modiolus* = hub

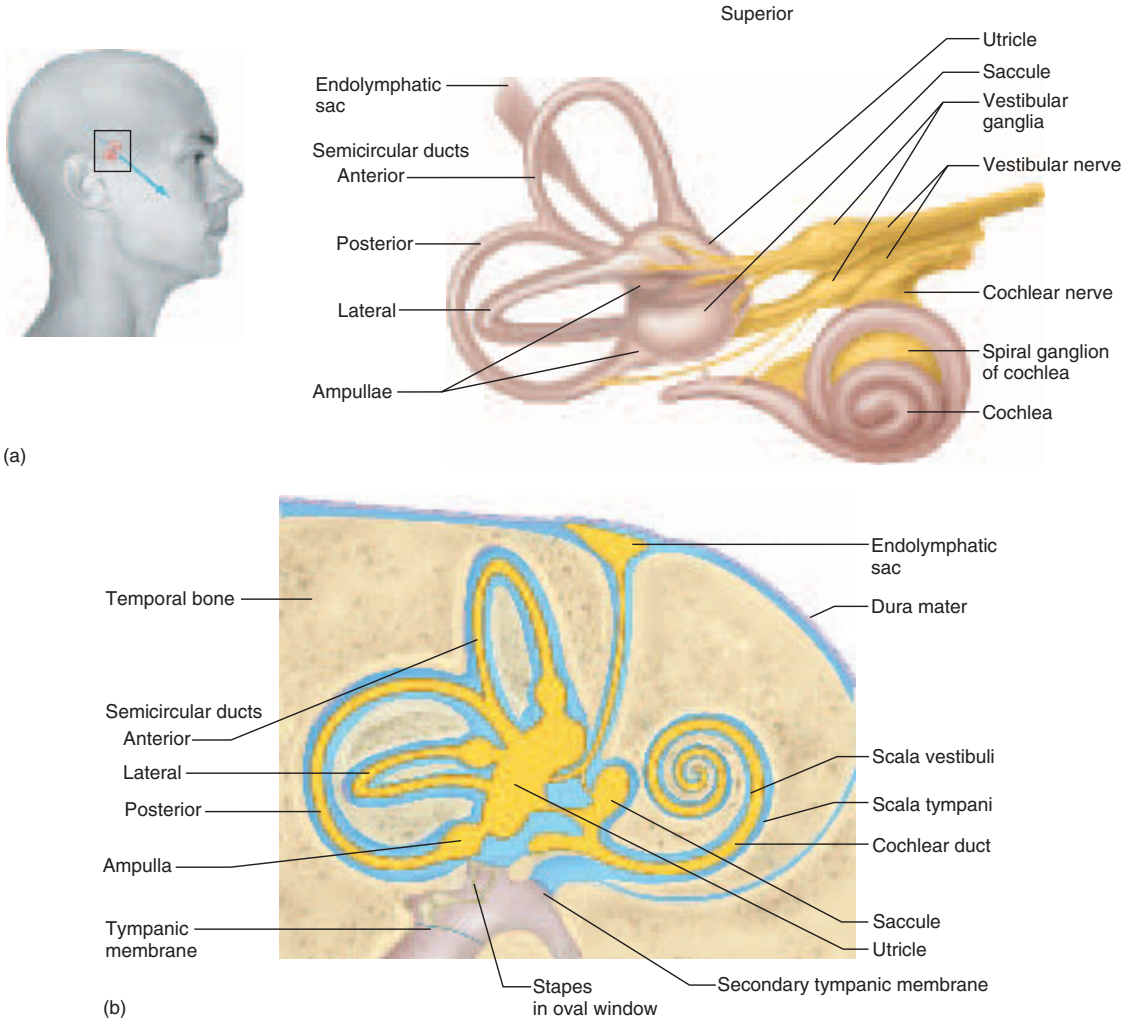


Figure 16.11 Anatomy of the Inner Ear. (*a*) The membranous labyrinth and its nerves. (*b*) Relationship of the perilymph (*blue*) and endolymph (*yellow*) to the labyrinth.

figure 16.12*b*. It is important to realize that the structures seen in cross section actually have the form of spiral strips winding around the modiolus from base to apex.

The cochlea has three fluid-filled chambers separated by membranes. The superior one is called the **scala**[27] **vestibuli** (SCAY-la vess-TIB-you-lye) and the inferior one is the **scala tympani.** These are filled with perilymph and communicate with each other through a narrow channel at the apex of the cochlea. The scala vestibuli begins near the oval window and spirals to the apex; from there, the scala tympani spirals back down to the base and ends at the **round window** (see fig. 16.10). The round window is covered by a membrane called the *secondary tympanic membrane.*

The middle chamber is a triangular space, the **cochlear duct (scala media).** It is separated from the scala vestibuli above by a thin **vestibular membrane** and from the scala tympani below by a much thicker **basilar membrane.** Unlike those chambers, it is filled with endolymph rather than perilymph. Within the cochlear duct, supported on the basilar membrane, is the **organ of Corti**[28] (COR-tee), a thick epithelium with associated structures (fig. 16.12*c*). It is the transducer that converts vibrations into nerve impulses, so we must pay particular attention to its structural details.

The organ of Corti has an epithelium composed of **hair cells** and **supporting cells.** Hair cells are named for

[27] *scala* = staircase

[28] Alfonso Corti (1822–88), Italian anatomist

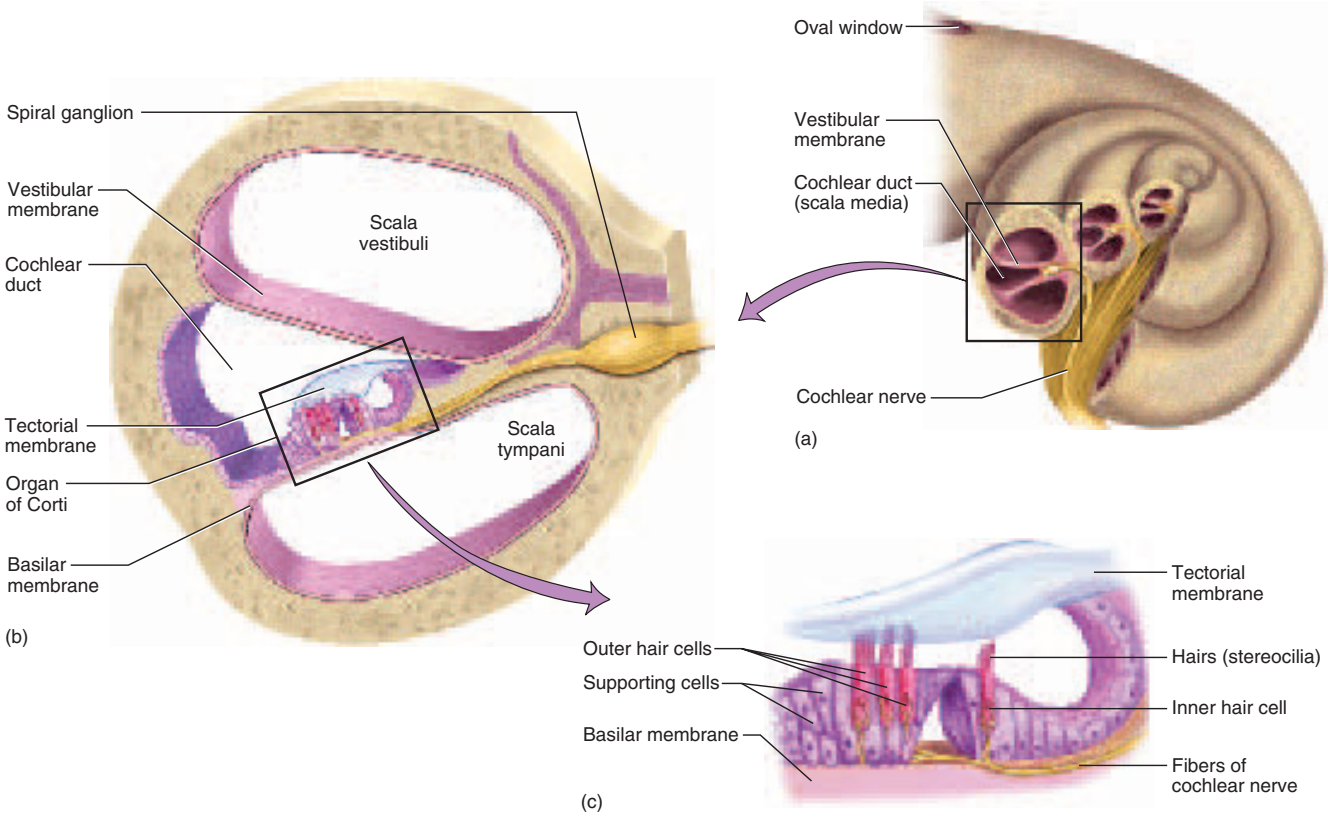


Figure 16.12 **Anatomy of the Cochlea.** (*a*) Vertical section. The apex of the cochlea faces downward and anterolaterally in anatomical position. (*b*) Detail of one section through the cochlea. (*c*) Detail of the organ of Corti.

the long, stiff microvilli called **stereocilia**[29] on their apical surfaces. (Stereocilia should not be confused with true cilia. They do not have an axoneme of microtubules as seen in cilia, and they do not move by themselves.) Resting on top of the stereocilia is a gelatinous **tectorial**[30] **membrane.**

The organ of Corti has four rows of hair cells spiraling along its length (fig. 16.13). About 3,500 of these, called **inner hair cells (IHCs),** are arranged in a row on the medial side of the basilar membrane (facing the modiolus). Each of these has a cluster of 50 to 60 stereocilia, graded from short to tall. Another 20,000 **outer hair cells (OHCs)** are neatly arranged in three rows across from the inner hair cells. Each outer hair cell has about 100 stereocilia arranged in the form of a V, with their tips embedded in the tectorial membrane. All that we hear comes from the IHCs, which supply 90% to 95% of the sensory fibers of the cochlear nerve. The function of the OHCs is to adjust

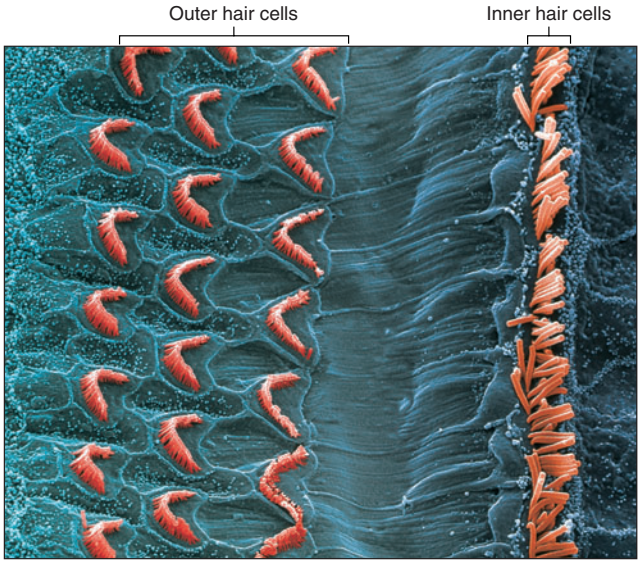


Figure 16.13 **Apical Surfaces of the Cochlear Hair Cells (SEM).**

[29] *stereo* = solid
[30] *tect* = roof

the response of the cochlea to different frequencies and enable the IHCs to work with greater precision. We will see shortly how this is done. Hair cells are not neurons, but synapse with nerve fibers at their base—the OHCs with both sensory and motor neurons and the IHCs with sensory neurons only.

The Physiology of Hearing

We can now examine the way in which sound affects the ear and produces action potentials.

The Middle Ear

The auditory ossicles provide no amplification; vibrations of the stapes against the inner ear normally have the same amplitude as vibrations of the eardrum against the malleus. Why have auditory ossicles, then? There are two answers to this. One is that the eardrum, which moves in air, vibrates easily, whereas the stapes footplate must vibrate against the fluid of the inner ear. This fluid puts up a much greater resistance to motion than air does. If airborne sound waves struck the footplate directly, they would not have enough energy to overcome this resistance and move the stapes. The eardrum, however, has 18 times the area of the oval window. By concentrating the energy of the vibrating eardrum on an area 1/18 that size, the ossicles create a greater force per unit area at the oval window and overcome the resistance of the endolymph.

The ossicles and their muscles also have a protective function. In response to a loud noise, the tensor tympani pulls the eardrum inward and tenses it, while the stapedius reduces mobility of the stapes. This **tympanic reflex** muffles the transfer of vibrations from the eardrum to the oval window. The reflex probably evolved in part for protection from loud but slowly building noises such as thunder. It has a latency of about 40 msec, which is not quick enough to protect the inner ear from sudden noises such as gunshots. The tympanic reflex also does not adequately protect the ears from sustained loud noises such as factory noise or loud music. Such noises can irreversibly damage the hair cells of the inner ear by fracturing their stereocilia. It is therefore imperative to wear ear protection when using firearms or working in noisy environments.

The middle-ear muscles also help to coordinate speech with hearing. Without them, the sound of your own speech would be so loud it could damage your inner ear, and it would drown out soft or high-pitched sounds from other sources. Just as you are about to speak, however, the brain signals these muscles to contract. This dampens the sense of hearing in phase with the inflections of your own voice and makes it possible to hear other people while you are speaking.

Think About It

What type of muscle fibers—slow oxidative or fast glycolytic (see p. 429)—do you think constitute the stapedius and tensor tympani? That is, which type would best suit the purpose of these muscles?

Stimulation of Cochlear Hair Cells

To produce a sensation of sound, vibration of the auditory ossicles leads to vibration of the basilar membrane on which the hair cells rest. A simple mechanical model of the ear (fig. 16.14) makes it easy to see how this happens. The stapes pushes on the perilymph of the scala vestibuli; the perilymph pushes the vestibular membrane down; the vestibular membrane pushes on the endolymph of the cochlear duct; and the endolymph pushes the basilar membrane down. (The vestibular membrane is omitted from the diagram for simplicity; it has no significant effect on the mechanics of the cochlea.) The basilar membrane puts pressure on the perilymph of the scala tympani below it, and the secondary tympanic membrane bulges outward to relieve this pressure. In short, as the stapes goes in-out-in, the secondary tympanic membrane goes out-in-out, and the basilar membrane goes down-up-down. It is not difficult to see how this happens—the only thing hard to imagine is that it can happen as often as 20,000 times per second!

The vestibular membrane separates the perilymph of the scala vestibuli from the endolymph of the cochlear duct. In order for the hair cells to function properly, the tips of their stereocilia must be bathed in endolymph. Endolymph has an exceptionally high K^+ concentration, which creates a strong electrochemical gradient from the

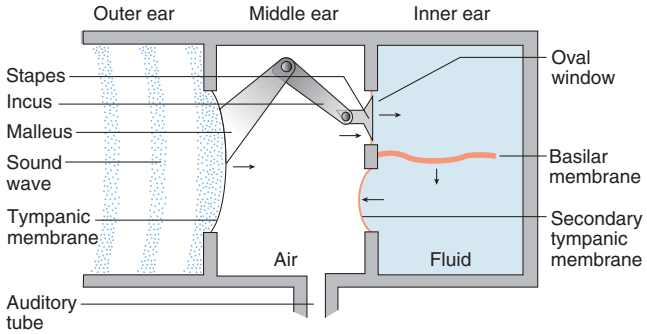


Figure 16.14 Mechanical Model of the Ear. Each inward movement of the tympanic membrane pushes inward on the auditory ossicles of the middle ear and fluid of the inner ear. This pushes down on the basilar membrane, and pressure is relieved by an outward bulge of the secondary tympanic membrane. Thus the basilar membrane vibrates up and down in synchrony with the vibrations of the tympanic membrane.

Why would high air pressure in the middle ear reduce the movements of the basilar membrane of the inner ear?

tip to the base of a hair cell. This gradient provides the potential energy that ultimately enables a hair cell to work.

The tectorial membrane is especially important in cochlear mechanics. Remember that the stereocilia of the outer hair cells have their tips embedded in it, and those of the inner hair cells come very close to it. The tectorial membrane is anchored to the modiolus, which holds it relatively still as the basilar membrane and hair cells vibrate up and down. Movement of the basilar membrane thus bends the hair cell stereocilia back and forth.

At the tip of each stereocilium of the inner hair cells is a single transmembrane protein that functions as a mechanically gated ion channel. A fine, stretchy protein filament called a **tip link** extends like a spring from the ion channel of one stereocilium to the side of the stereocilium next to it (fig. 16.15). The stereocilia increase in height progressively, so that all but the tallest ones have tip links leading to taller stereocilia beside them. When a taller stereocilium bends away from a shorter one, it pulls on the tip link and opens the ion channel of the shorter stereocilium. The channel is nonselective, but since the predominant ion of the endolymph is K^+, the primary effect of this gating is to allow a quick burst of K^+ to flow into each hair cell. This depolarizes the hair cell while the channel is open, and when the stereocilium bends the other way its channel closes and the cell becomes briefly hyperpolarized. During the moments of depolarization, a hair cell releases a neurotransmitter that stimulates the sensory dendrites synapsing with its base. Each depolarization thus generates action potentials in the cochlear nerve.

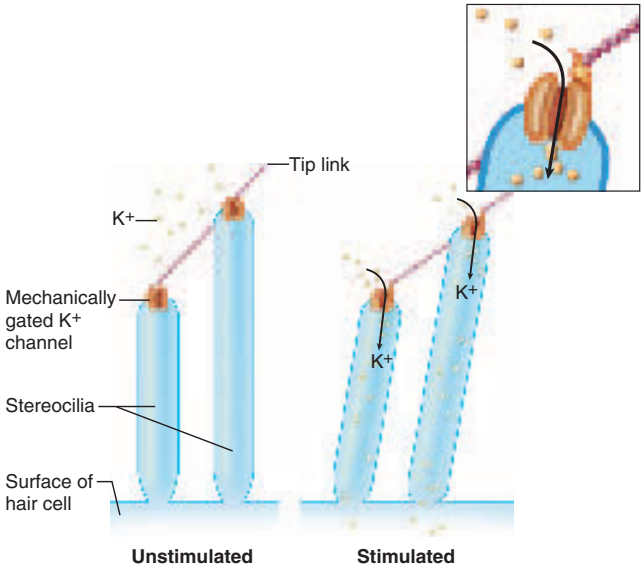


Figure 16.15 Potassium Gates of the Cochlear Hair Cells. Each stereocilium has a gated K^+ channel at its tip. Vibrations of the cochlea cause each stereocilium to bend and, with its tip link, pull open the K^+ channel of the adjacent stereocilium. The inflow of K^+ depolarizes the hair cell.

Sensory Coding

Our ability to distinguish loudness and pitch depends on the ability of the cochlea to respond differently to vibrations of different amplitude and frequency. Loud sounds produce more vigorous vibrations of the organ of Corti. This excites a greater number of hair cells over a broader area of basilar membrane and triggers a higher frequency of action potentials in the cochlear nerve fibers. If the brain detects intense activity in nerve fibers from a broad region of the organ of Corti, it interprets this as a loud sound.

Frequency discrimination requires a more sophisticated mechanism. The basilar membrane is spanned by short, stiff collagen fibers of various lengths. At its proximal (basal) end, the basilar membrane is attached, narrow, and stiff. At its distal (apical) end, it is unattached, five times wider than at the base, and more flexible. Think of the basilar membrane as analogous to a rope stretched tightly between two posts. If you pluck the rope at one end, a wave of vibration travels down its length and back. This produces a standing wave, with some regions of the rope vertically displaced more than others. Similarly, a sound causes a standing wave in the basilar membrane. The peak amplitude of this wave is near the distal end in the case of low-frequency sounds and nearer the proximal (attached) end with sounds of higher frequencies. When the brain receives signals mainly from inner hair cells at the distal end, it interprets the sound as low-pitched; when signals come mainly from the proximal end, it interprets the sound as high-pitched (fig. 16.16). Speech, music, and other everyday sounds, of course, are not pure tones—they create complex patterns of vibration in the basilar membrane that must be decoded by the brain.

Cochlear Tuning

Just as we tune a radio to receive a certain frequency, we also tune our cochlea to receive some frequencies better than others. The outer hair cells (OHCs) are supplied with a few sensory fibers (5%–10% of those in the cochlear nerve), but more importantly, they receive motor fibers from the brain.

In response to sound, the OHCs trigger nerve signals to the medulla by way of the sensory neurons, and the pons sends signals immediately to the OHCs by way of the motor neurons. In response, the hair cells contract by about 10% to 15%. Because an OHC is anchored to the basilar membrane below and its stereocilia are embedded in the tectorial membrane above, contraction of an OHC reduces the basilar membrane's freedom to vibrate. This results in some regions of the organ of Corti sending fewer signals to the brain than neighboring regions, so the brain can better distinguish between the more active and less

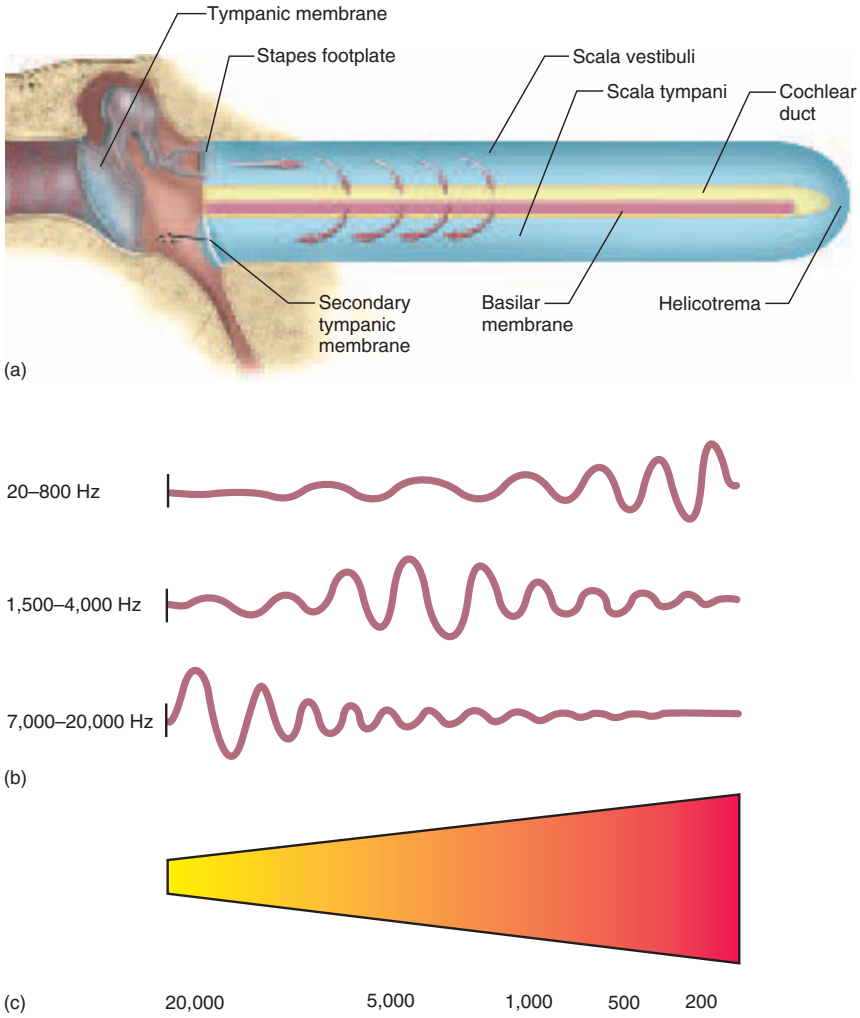


Figure 16.16 Frequency Response of the Basilar Membrane of the Cochlea. (*a*) The cochlea, uncoiled and laid out straight. (*b*) Sounds produce a standing wave of vibration along the basilar membrane. The peak amplitude of the wave varies with the frequency of the sound, as shown here. The amount of vibration is greatly exaggerated in this diagram to clarify the standing wave. (*c*) The taper of the basilar membrane and its correlation with sound frequencies. High frequencies (7,000–20,000 Hz) are best detected by hair cells near the narrow proximal end at the *left* and low frequencies (20–800 Hz) by hair cells near the wider distal end at the *right*.

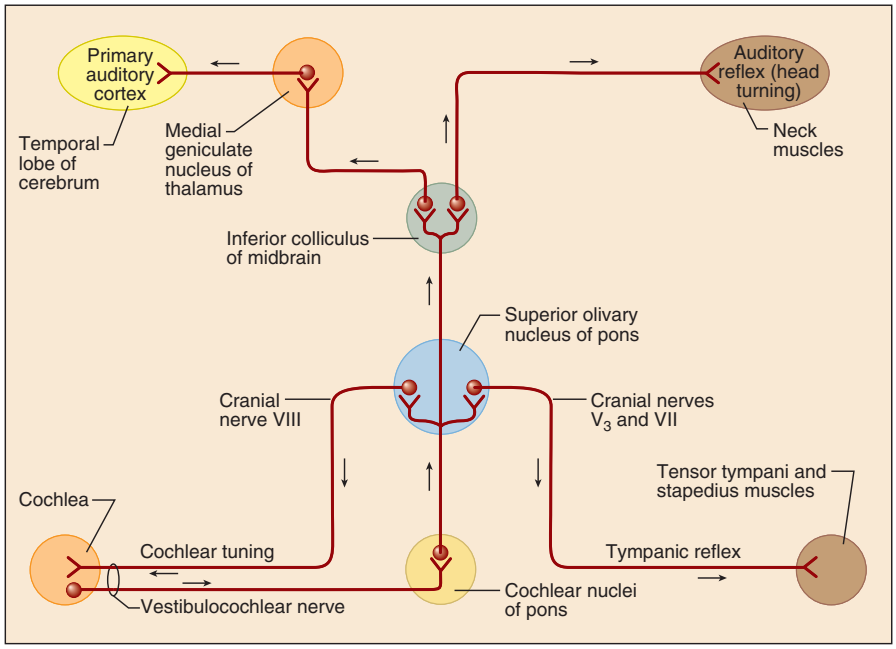
active hair cells and sound frequencies. When OHCs are experimentally incapacitated, the inner hair cells (IHCs) respond much less precisely to differences in pitch.

There is another mechanism of cochlear tuning involving the inner hair cells. The pons sends efferent fibers to the cochlea that synapse with the sensory nerve fibers near the base of the IHCs. The efferent fibers can inhibit the sensory fibers from firing in some areas of the cochlea, and thus enhance the contrast between signals from the more responsive and less responsive regions. Combined with the previously described role of the OHCs, this sharpens the tuning of the cochlea and our ability to discriminate sounds of different pitch.

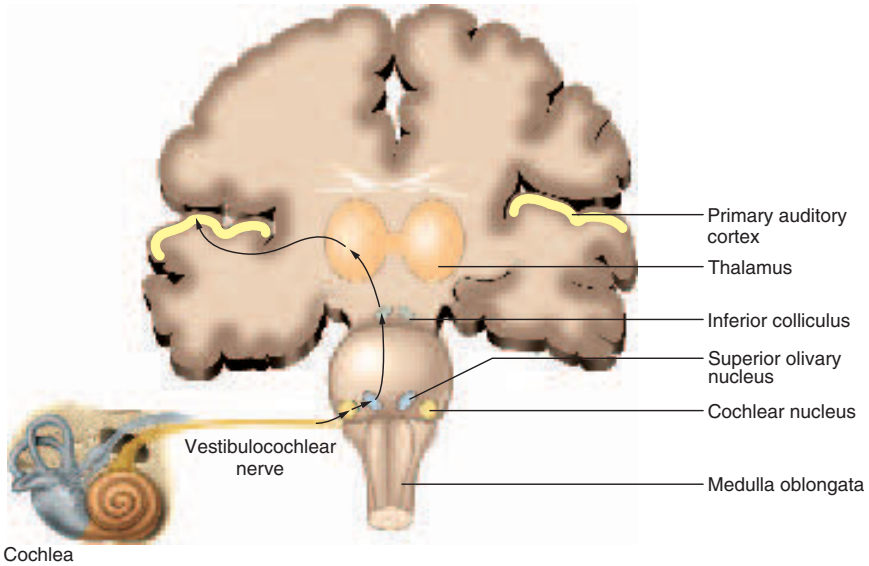
The Auditory Projection Pathway

A **spiral ganglion,** wound around the modiolus, is composed of bipolar sensory neurons. Their dendrites originate at the hair cells and their axons form the **cochlear nerve.** The cochlear nerve joins the *vestibular nerve,* discussed later, and the two together become the *vestibulocochlear nerve* (cranial nerve VIII).

The cochlear nerve fibers from each ear lead to *cochlear nuclei* on both sides of the pons. There, they synapse with second-order neurons that ascend to the nearby *superior olivary nucleus* of the pons (fig. 16.17). By way of cranial nerve VIII, the superior olivary nucleus



(a)



(b)

Figure 16.17 Auditory Pathways in the Brain. (*a*) Schematic. (*b*) Brainstem and frontal section of the cerebrum, showing the locations of auditory processing centers.

issues the efferent fibers back to the cochlea that are involved in cochlear tuning. By way of cranial nerves V_3 and VII, it issues motor fibers to the tensor tympani and stapedius muscles, respectively. The superior olivary nucleus also functions in **binaural**[31] **hearing**—comparing signals from the right and left ears to identify the direction from which a sound is coming.

Other fibers from the cochlear nuclei ascend to the inferior colliculi of the midbrain. The inferior colliculi help to locate the origin of a sound in space, process fluctuations in pitch that are important for such purposes as understanding another person's speech, and mediate the startle response and rapid head turning that occur in reaction to loud or sudden noises.

Third-order neurons begin in the inferior colliculi and lead to the thalamus. Fourth-order neurons begin here and complete the pathway to the primary auditory cortex, which is in the superior margin of the temporal lobe deep within the lateral sulcus (see photo on p. 585). The temporal lobe is the site of conscious perception of sound, and it completes the information processing essential to binaural hearing. Because of extensive decussation in the auditory pathway, damage to the right or left auditory cortex does not cause a unilateral loss of hearing.

[31] *bin* = two + *aur* = ears

Insight 16.3 Clinical Application

Deafness

Deafness means any hearing loss, from mild and temporary to complete and irreversible. *Conductive deafness* results from any condition that interferes with the transmission of vibrations to the inner ear. Such conditions include a damaged eardrum, otitis media, blockage of the auditory canal, and *otosclerosis*.[32] Otosclerosis is the fusion of auditory ossicles to each other or fusion of the stapes to the oval window, which prevents the bones from vibrating freely. *Sensorineural (nerve) deafness* results from the death of hair cells or any of the nervous elements concerned with hearing. It is a common occupational disease of factory and construction workers, musicians, and other people. Deafness leads some people to develop delusions of being talked about, disparaged or cheated. Beethoven said his deafness drove him nearly to suicide.

[32] *oto* = ear + *scler* = hardening + *osis* = process, condition

Equilibrium

The original function of the ear in vertebrate evolution was not hearing, it was **equilibrium**—coordination and balance. Only later did vertebrates evolve the cochlea, middle-ear structures, and auditory function of the ear. In humans, the receptors for equilibrium constitute the **vestibular apparatus,** which consists of three **semicircular ducts** and two chambers—an anterior **saccule** (SAC-yule) and a posterior **utricle**[33] (YOU-trih-cul) (see fig. 16.11).

The sense of equilibrium is divided into **static equilibrium,** the perception of the orientation of the head when the body is stationary, and **dynamic equilibrium,** the perception of motion or acceleration. Acceleration is divided into *linear acceleration,* a change in velocity in a straight line, as when riding in a car or elevator, and *angular acceleration,* a change in the rate of rotation. The saccule and utricle are responsible for static equilibrium and the sense of linear acceleration; the semicircular ducts detect only angular acceleration.

The Saccule and Utricle

Each of these chambers has a 2 by 3 mm patch of hair cells and supporting cells called a **macula.**[34] The **macula sacculi** lies nearly vertically on the wall of the saccule, and the **macula utriculi** lies nearly horizontally on the floor of the utricle (fig. 16.18*a*).

Each hair cell of a macula has 40 to 70 stereocilia and one true cilium called a **kinocilium.**[35] The tips of the stereocilia and kinocilium are embedded in a gelatinous **otolithic membrane.** This membrane is weighted with calcium carbonate–protein granules called **otoliths**[36] (fig. 16.18*b*), which add to the density and inertia of the membrane and enhance the sense of gravity and motion.

Figure 16.18*c* shows how the macula utriculi detects tilt of the head. With the head erect, the otolithic membrane bears directly down on the hair cells and stimulation is minimal. When the head is tilted, however, the weight of the membrane bends the stereocilia and stimulates the hair cells. Any orientation of the head causes a combination of stimulation to the utricules and saccules of the two ears. The brain interprets head orientation by comparing these inputs to each other and to other input from the eyes and stretch receptors in the neck.

The inertia of the otolithic membranes is especially important in detecting linear acceleration. Suppose you are sitting in a car at a stoplight and then begin to move. The heavy otolithic membrane of the macula utriculi briefly lags behind the rest of the tissues, bends the stereocilia backward, and stimulates the cells. When you stop at the next light, the macula stops but the otolithic membrane keeps on going for a moment, bending the stereocilia forward. The hair cells convert this pattern of stimulation to nerve signals, and the brain is thus advised of changes in your linear velocity.

If you are standing in an elevator and it begins to move up, the otolithic membrane of the vertical macula

[33] *saccule* = little sac; *utricle* = little bag
[34] *macula* = spot
[35] *kino* = moving
[36] *oto* = ear + *lith* = stone

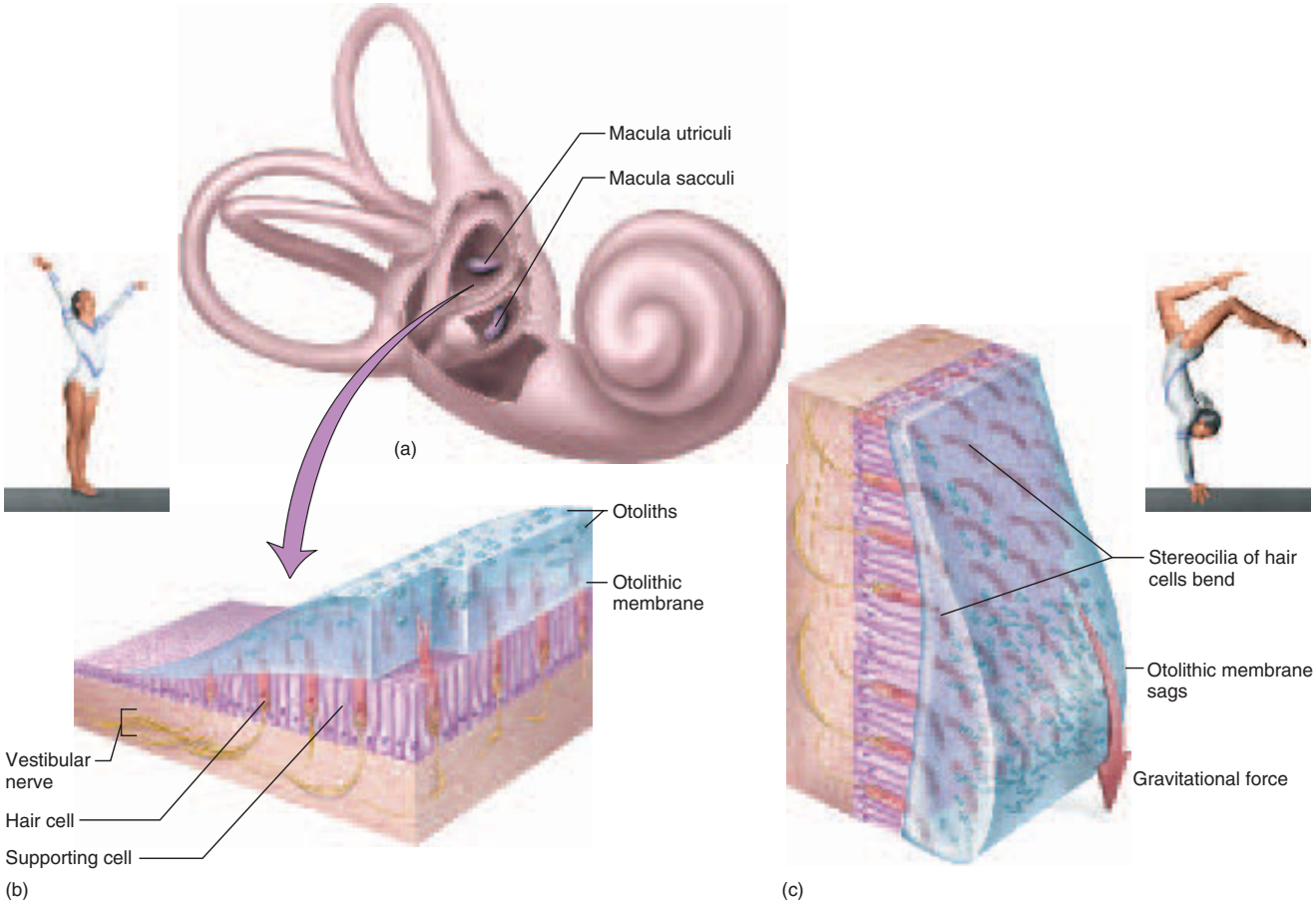


Figure 16.18 The Saccule and Utricle. (*a*) Locations of the macula sacculi and macula utriculi. (*b*) Structure of a macula. (*c*) Action of the otolithic membrane on the hair cells when the head is tilted.

sacculi lags behind briefly and pulls down on the hairs. When the elevator stops, the otolithic membrane keeps going for a moment and bends the hairs upward. The macula sacculi thus detects vertical acceleration.

The Semicircular Ducts

Rotational acceleration is detected by the three *semicircular ducts* (fig. 16.19), each housed in an osseous *semicircular canal* of the temporal bone. The **anterior** and **posterior semicircular ducts** are positioned vertically, at right angles to each other. The **lateral semicircular duct** is about 30° from the horizontal plane. The orientation of the ducts causes a different duct to be stimulated by rotation of the head in different planes—turning it from side to side as in gesturing "no," nodding up and down as in gesturing "yes," or tilting it from side to side as in touching your ears to your shoulders.

The semicircular ducts are filled with endolymph. Each duct opens into the utricle and has a dilated sac at one end called an **ampulla.**[37] Within the ampulla is a mound of hair cells and supporting cells called the **crista**[38] **ampullaris.** The hair cells have stereocilia and a kinocilium embedded in the **cupula,**[39] a gelatinous membrane that extends from the crista to the roof of the ampulla. When the head turns the duct rotates, but the endolymph lags behind. It pushes the cupula, bends the stereocilia, and stimulates the hair cells. After 25 to 30 seconds of continual rotation, however, the endolymph catches up with the movement of the duct and stimulation of the hair cells ceases.

Think About It

The semicircular ducts do not detect motion itself, but only acceleration—a *change in the rate* of motion. Explain.

[37] *ampulla* = little jar
[38] *crista* = crest, ridge
[39] *cupula* = little tub

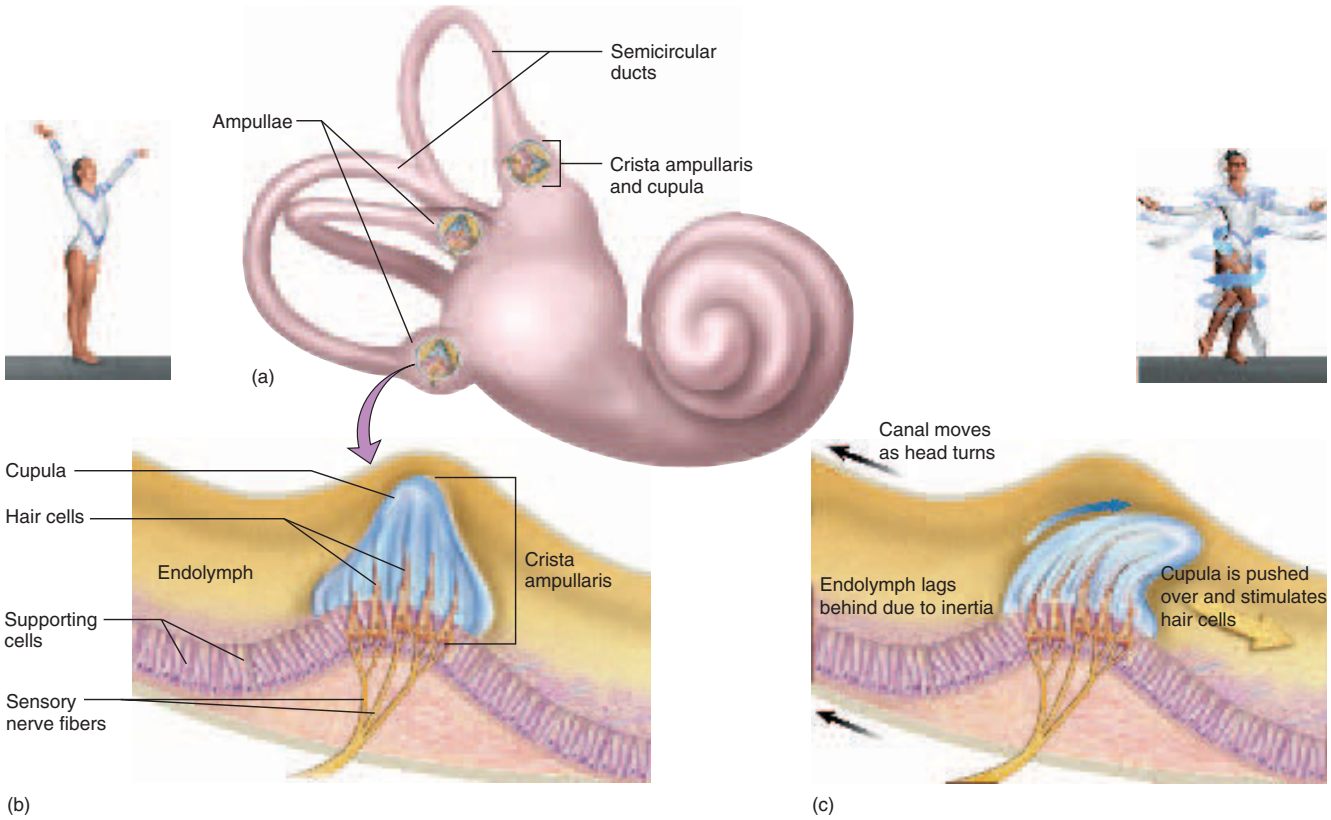


Figure 16.19 The Semicircular Ducts. (*a*) Structure of the semicircular ducts, with each ampulla opened to show the crista ampullaris and cupula. (*b*) Detail of the crista ampullaris. (*c*) Action of the endolymph on the cupula and hair cells when the head is rotated.

Equilibrium Projection Pathways

Hair cells of the macula sacculi, macula utriculi, and semicircular ducts synapse at their bases with sensory fibers of the **vestibular nerve.** This nerve joins the cochlear nerve to form the vestibulocochlear nerve (VIII). Most fibers from the vestibular apparatus terminate in the **vestibular nucleus** of the pons, while others project to the cerebellum. Fibers from the vestibular nucleus project caudally to the cervical spinal cord, and from there, fibers lead to the brainstem nuclei for the cranial nerves that control eye movements—the oculomotor (III), trochlear (IV), and abducens (VI) nerves. Other fibers from the cervical spinal cord lead by way of the accessory nerve (XI) to muscles that move the head and neck.

Reflex pathways to the extrinsic eye muscles enable us to fixate visually on a point in space while the head is moving. To observe this effect, hold this book in front of you at a comfortable reading distance and fix your gaze on the middle of the page. Move the book left and right about once per second, and you will be unable to read it. Now hold the book still and shake your head from side to side at the same rate. This time you will be able to read it because the reflex pathway compensates for your head movements and keeps your eyes fixed on the target.

Before we move on to the sense of vision, you may wish to review the anatomy of the ear (table 16.2) and think back on the function of each component.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

14. What physical properties of sound waves correspond to the sensations of loudness and pitch?
15. What is the benefit of having auditory ossicles and muscles in the middle ear?
16. Explain how vibration of the tympanic membrane ultimately produces fluctuations of membrane voltage in a cochlear hair cell.
17. How does the brain recognize the difference between high C and middle C musical notes? Between a loud sound and a soft one?
18. How does the function of the semicircular ducts differ from the function of the saccule and utricle?
19. How is sensory transduction in the semicircular ducts similar to that in the saccule and utricle?

Table 16.2 Anatomical Review of the Ear

Outer Ear (fig. 16.9)

- Auricle (pinna)
- Auditory canal (external auditory meatus)
- Ceruminous glands
- Helix
- Antihelix
- Triangular fossa
- Concha
- Tragus
- Antitragus
- Lobe

Middle Ear (fig. 16.10)

- *Tympanic membrane (eardrum)*
- *Tympanic cavity*
- *Auditory ossicles*
  - Malleus
  - Incus
  - Stapes
    - Footplate
- *Muscles*
  - Stapedius
  - Tensor tympani
- *Auditory (eustachian) tube*

Inner Ear (figs. 16.10 and 16.11)

- *Oval window*
- *Labyrinths and fluids*
  - Bony labyrinth
  - Membranous labyrinth
  - Perilymph
  - Endolymph
- *Cochlea*
- *Vestibular apparatus*
- *Round window*
- *Secondary tympanic membrane*

Cochlea (fig. 16.12)

- Modiolus
- Scala vestibuli
- Scala tympani
- Helicotrema
- Cochlear duct
- Vestibular membrane
- Organ of Corti
  - Supporting cells
  - Inner hair cells
  - Outer hair cells
  - Stereocilia
  - Tectorial membrane
  - Basilar membrane
- Neural components
  - Spiral ganglion
  - Cochlear nerve

Vestibular Apparatus (figs. 16.18 and 16.19)

- Vestibule
  - Saccule
    - Macula sacculi
  - Utricle
    - Macula utriculi
  - Otolithic membranes
- Semicircular ducts
  - Ampulla
  - Crista ampullaris
  - Cupula
- Vestibular nerve

Projection Pathways (fig. 16.17)

- Vestibulocochlear nerve
- Cochlear nucleus
- Vestibular nucleus
- Superior olivary nucleus
- Inferior and superior colliculi
- Thalamus
- Primary auditory cortex

Vision

Objectives

When you have completed this section, you should be able to

- describe the anatomy of the eye and its accessory structures;
- describe the structure of the retina and its receptor cells;
- explain how the optical system of the eye creates an image on the retina;
- explain how the retina converts this image to nerve impulses;
- explain why different types of receptor cells and neuronal circuits are required for day and night vision; and
- trace the visual projection pathways in the brain.

Light and Vision

Vision (sight) is the perception of objects in the environment by means of the light that they emit or reflect. *Light* is visible electromagnetic radiation. Human vision is limited to wavelengths ranging from about 400 to 750 nm. The *ultraviolet (UV)* radiation just below 400 nm and the *infrared (IR)* radiation just above 700 nm are invisible to us, although some animals can see a little farther into those ranges than we can. Most solar radiation that reaches the surface of the earth falls within this range; radiation of shorter and longer wavelengths is generally filtered out by ozone, carbon dioxide, and water vapor in the atmosphere. Vision is thus adapted to take advantage of the radiation that is most available to us.

Yet there is further reason for vision to be limited to this range of wavelengths. To produce a physiological response, light must cause a *photochemical reaction*—a change in chemical structure caused by light energy. When an electron absorbs a photon of light, it is boosted to a higher energy level (orbit) around its nucleus and may transfer to another atom. The transfer of an electron from one atom to another is the essence of a chemical reaction. Ultraviolet radiation has so much energy that it ionizes organic molecules and kills cells. It is useful for sterilizing food and instruments, but it has too much energy for the biochemical processes of vision. Infrared radiation has too little energy to activate the visual process. It warms the tissues (heat lamps are based on this principle) but does not usually cause chemical reactions.

Accessory Structures of the Orbit

Before considering the eye itself, let's survey the accessory structures located in and around the orbit (figs. 16.20 and 16.21). These include the *eyebrows, eyelids, conjunctiva, lacrimal apparatus,* and *extrinsic eye muscles:*

- The **eyebrows** probably serve mainly to enhance facial expressions and nonverbal communication (see p. 204), but they may also protect the eyes from glare and help to keep perspiration from running into the eye.

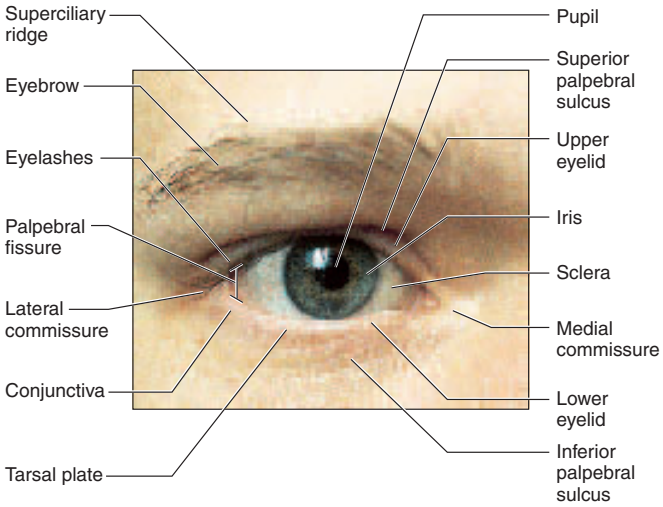


Figure 16.20 **External Anatomy of the Orbital Region.**

- The **eyelids,** or **palpebrae** (pal-PEE-bree), block foreign objects from the eye, prevent visual stimuli from disturbing our sleep, and blink periodically to moisten the eye with tears and sweep debris from the surface. The eyelids are separated from each other by the **palpebral fissure** and meet each other at the corners called the **medial** and **lateral commissures (canthi).** The eyelid consists largely of the orbicularis oculi muscle covered with skin (fig. 16.21*a*). It also contains a supportive fibrous **tarsal plate,** which is thickened along the margin of the eyelid. Within the plate are 20 to 25 **tarsal glands** that open along the margin. They secrete an oil that coats the eye and reduces tear evaporation. The **eyelashes** are guard hairs that help to keep debris from the eye. Touching the eyelashes stimulates hair receptors and triggers the blink reflex.
- The **conjunctiva** (CON-junk-TY-vuh) is a transparent mucous membrane that covers the inner surface of the eyelid and anterior surface of the eyeball, except for the cornea. Its primary purpose is to secrete a thin mucous film that prevents the eyeball from drying. It is richly innervated and highly sensitive to pain. It is also very vascular, which is especially evident when the vessels are dilated and the eyes are "bloodshot." Because it is vascular and the cornea is not, the conjunctiva heals more readily than the cornea when injured.
- The **lacrimal**[40] **apparatus** (fig. 16.21*b*) consists of the lacrimal (tear) gland and a series of ducts that drain the

[40] *lacrim* = tear

Chapter 16

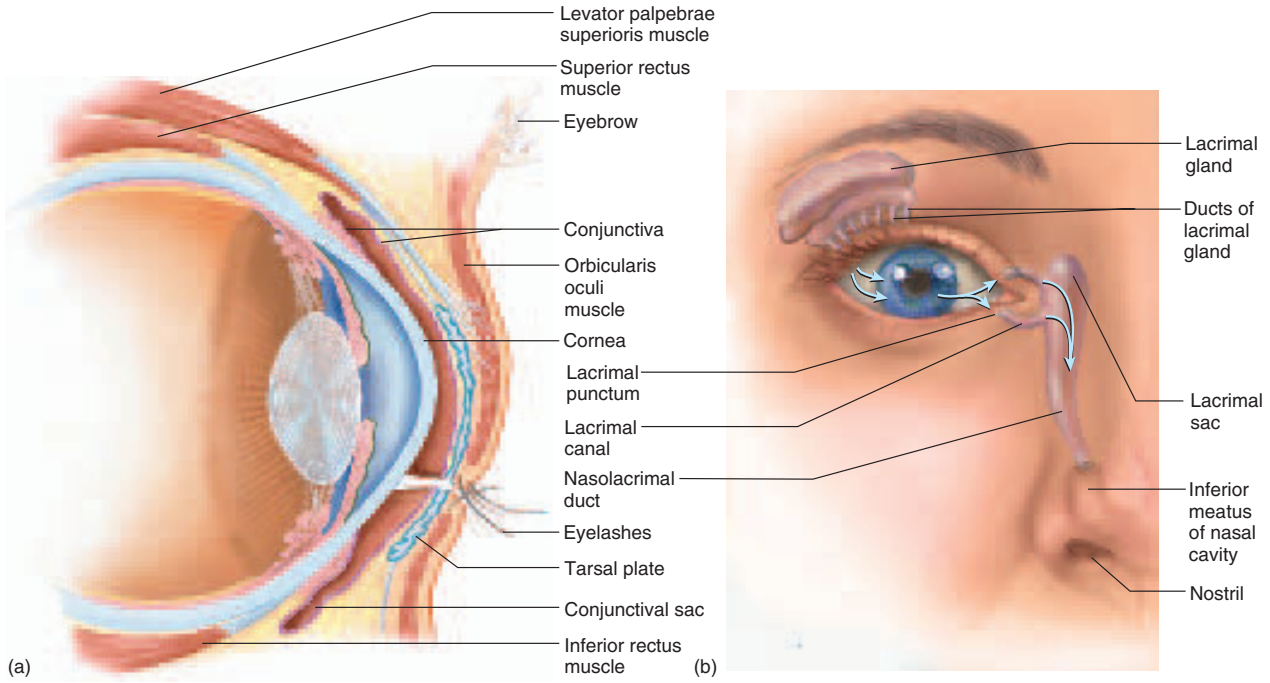


Figure 16.21 **Accessory Structures of the Orbit.** (*a*) Sagittal section of the eye and orbit. (*b*) The lacrimal apparatus.

tears into the nasal cavity. The **lacrimal gland,** about the size and shape of an almond, is nestled in a shallow fossa of the frontal bone in the superolateral corner of the orbit. About 12 short ducts lead from the lacrimal gland to the surface of the conjunctiva. Tears function to cleanse and lubricate the eye surface, deliver oxygen and nutrients to the conjunctiva, and prevent infection by means of a bactericidal enzyme, *lysozyme.* Periodic blinking spreads the tears across the eye surface. On the margin of each eyelid near the medial commissure is a tiny pore, the **lacrimal punctum.**[41] This is the opening to a short **lacrimal canal,** which leads to the **lacrimal sac** in the medial wall of the orbit. From this sac, a **nasolacrimal duct** carries the tears to the inferior meatus of the nasal cavity—thus an abundance of tears from crying or watery eyes can result in a runny nose. Once the tears enter the nasal cavity, they normally flow back to the throat and we swallow them. When we have a cold, the nasolacrimal ducts become swollen and obstructed, the tears cannot drain, and they may overflow from the brim of the eye.

- The **extrinsic eye muscles** are the six muscles attached to the walls of the orbit and to the external surface of the eyeball. *Extrinsic* means arising externally; it distinguishes these from the *intrinsic* muscles inside the eyeball, to be considered later. The extrinsic muscles move the eye (fig. 16.22). They include four *rectus* ("straight") muscles and two *oblique* muscles. The **superior, inferior, medial,** and **lateral rectus** originate on the posterior wall of the orbit and insert on the anterior region of the eyeball, just beyond the visible "white of the eye." They move the eye up, down, medially, and laterally. The **superior oblique** travels along the medial wall of the orbit. Its tendon passes through a fibrocartilage ring, the **trochlea**[42] (TROCK-lee-uh), and inserts on the superolateral aspect of the eyeball. The **inferior oblique** extends from the medial wall of the orbit to the inferolateral aspect of the eye. To visualize the function of the oblique muscles, suppose you turn your eyes to the right. The superior oblique muscle will slightly depress your right eye, while the inferior oblique slightly elevates the left eye. The opposite occurs when you look to the left. This is the primary function of the oblique muscles, but they also slightly rotate the eyes, turning the "twelve o'clock pole" of each eye slightly toward or away from the nose. Most of the extrinsic muscles are supplied by the oculomotor nerve (cranial nerve III), but the superior oblique is innervated by the trochlear nerve (IV) and the lateral rectus by the abducens (VI).

[41] *punct* = point

[42] *trochlea* = pulley

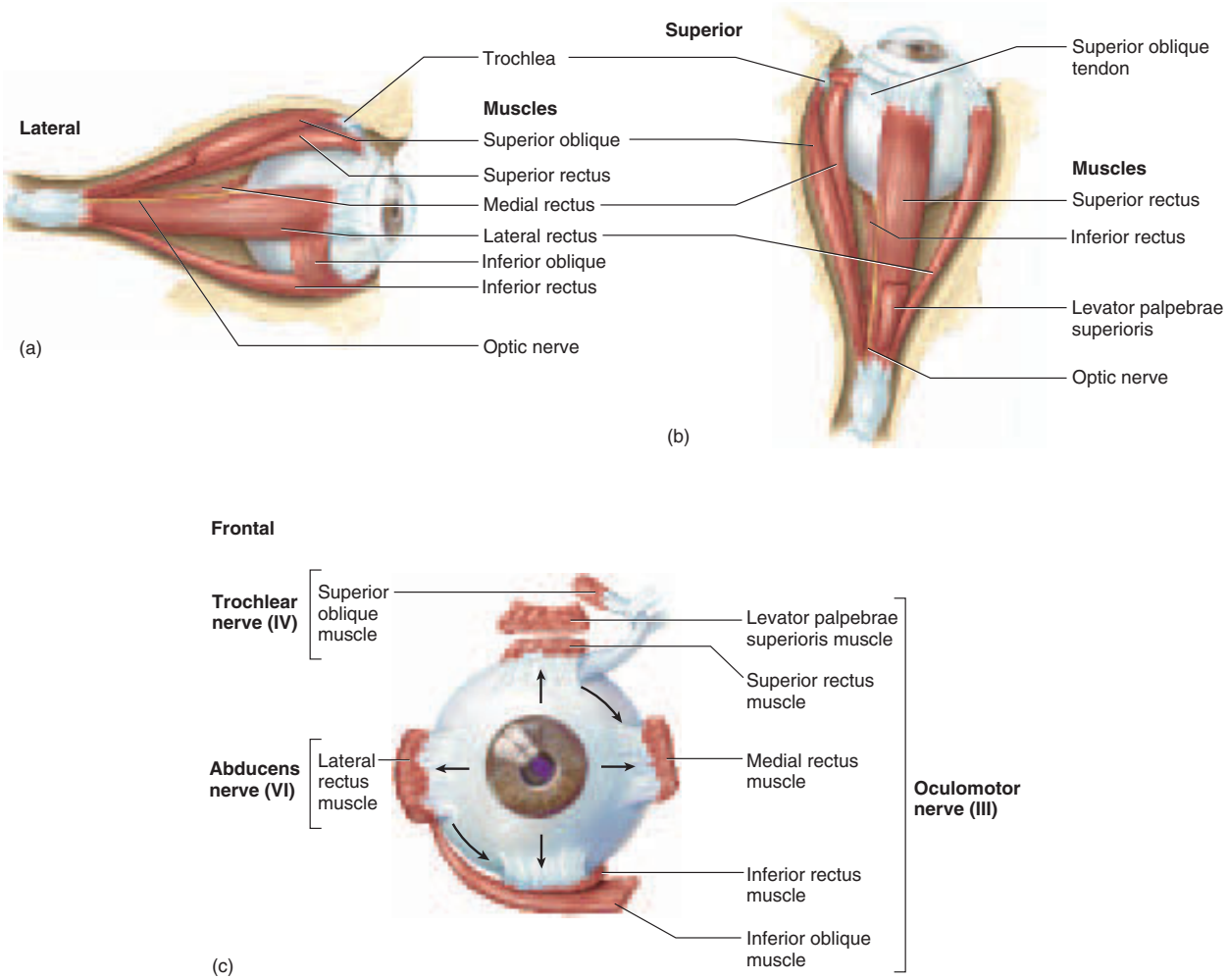


Figure 16.22 Extrinsic Muscles of the Eye. (*a*) Lateral view of the right eye. (*b*) Superior view of the right eye. (*c*) Innervation of the extrinsic muscles; arrows indicate the eye movement produced by each muscle.

The eye is surrounded on the sides and back by **orbital fat.** It cushions the eye, gives it freedom of motion, and protects blood vessels and nerves as they pass through the rear of the orbit.

Anatomy of the Eye

The eyeball itself is a sphere about 24 mm in diameter (fig. 16.23) with three principal components: (1) three layers (tunics) that form the wall of the eyeball; (2) optical components that admit and focus light; and (3) neural components, the retina and optic nerve. The retina is not only a neural component but also part of the inner tunic. The cornea is part of the outer tunic as well as one of the optical components.

The Tunics

The three tunics of the eyeball are as follows:

- The outer **fibrous layer** (tunica fibrosa). This is divided into two regions: the sclera and cornea. The **sclera**[43] (white of the eye) covers most of the eye surface and consists of dense collagenous connective tissue perforated by blood vessels and nerves. The **cornea** is the transparent region of modified sclera that admits light into the eye.
- The middle **vascular layer** (tunica vasculosa), also called the **uvea**[44] (YOU-vee-uh) because it resembles a

[43] *scler* = hard, tough
[44] *uvea* = grape

peeled grape in fresh dissection. It consists of three regions—the choroid, ciliary body, and iris. The **choroid** (CO-royd) is a highly vascular, deeply pigmented layer of tissue behind the retina. It gets its name from a histological resemblance to the chorion of the pregnant uterus. The **ciliary body,** a thickened extension of the choroid, forms a muscular ring around the lens. It supports the iris and lens and secretes a fluid called the aqueous humor. The **iris** is an adjustable diaphragm that controls the diameter of the **pupil,** its central opening. The iris has two pigmented layers. One is a posterior *pigment epithelium* that blocks stray light from reaching the retina. The other is the *anterior border layer,* which contains pigmented cells called **chromatophores.**[45] High concentrations of melanin in the chromatophores give the iris a black, brown, or hazel color. If the melanin is scanty, light reflects from the posterior pigment epithelium and gives the iris a blue, green, or gray color.

- The **inner layer** (tunica interna), which consists of the retina and optic nerve.

The Optical Components

The optical components of the eye are transparent elements that admit light rays, bend (refract) them, and focus images on the retina. They include the *cornea, aqueous humor, lens,* and *vitreous body.* The cornea has been described already.

- The **aqueous humor** is a serous fluid secreted by the ciliary body into the **posterior chamber,** a space between the iris and lens (fig. 16.24). It flows through the pupil into the **anterior chamber** between the cornea and iris. From here, it is reabsorbed by a ringlike blood vessel called the **scleral venous sinus** (*canal of Schlemm*[46]). Normally the rate of reabsorption balances the rate of secretion (see insight 16.4 for an important exception).
- The **lens** is suspended behind the pupil by a ring of fibers called the **suspensory ligament** (figs. 16.23 and 16.25), which attaches it to the ciliary body. Tension on the ligament somewhat flattens the lens so it is about 9.0 mm in diameter and 3.6 mm thick at the middle. When the lens is removed from the eye and

[45] *chromato* = color + *phore* = bearer

[46] Friedrich S. Schlemm (1795–1858), German anatomist

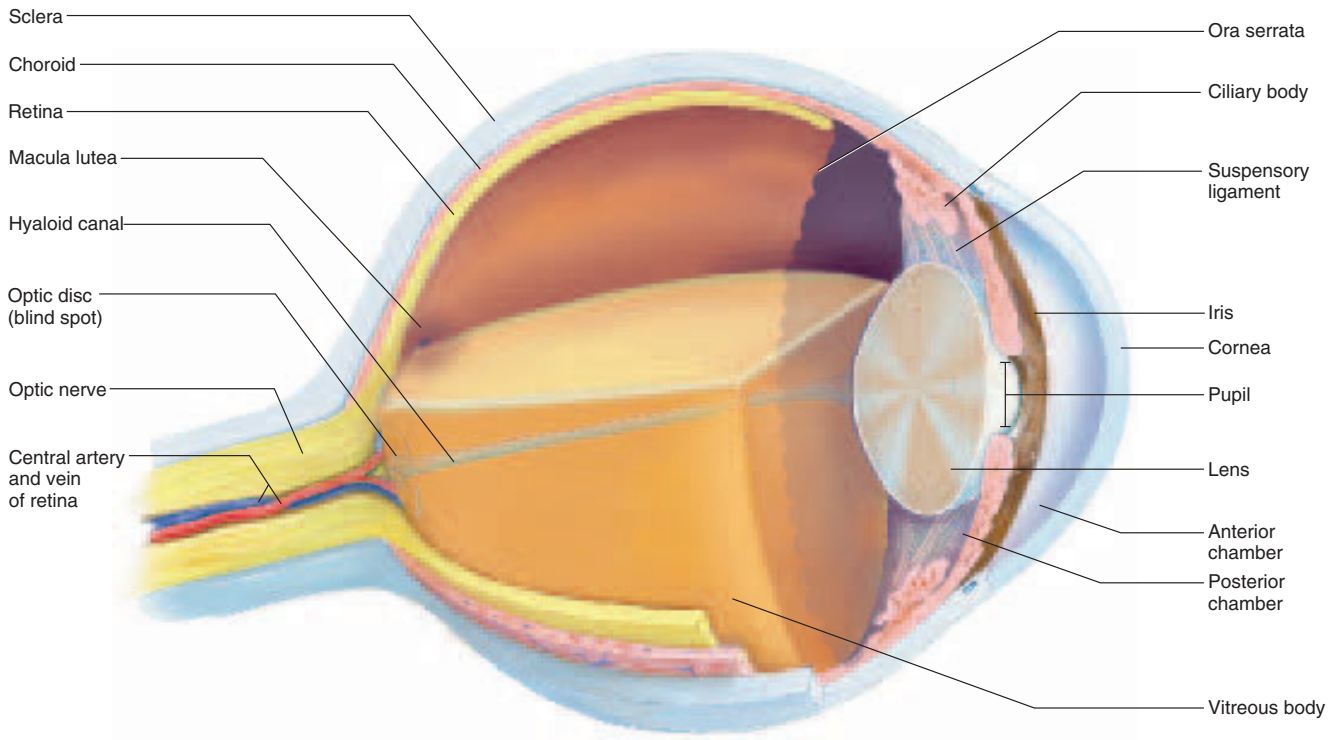


Figure 16.23 **The Eye, Sagittal Section.** The vitreous body has been omitted from the upper half to reveal structures behind it.

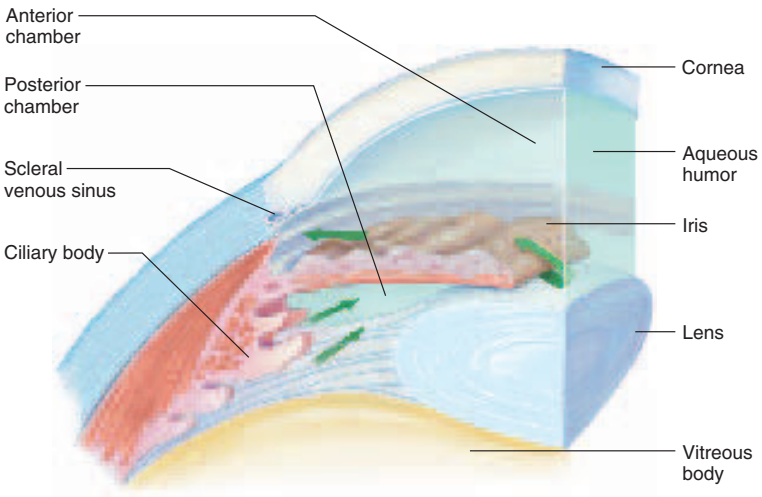


Figure 16.24 **Production and Reabsorption of Aqueous Humor.**

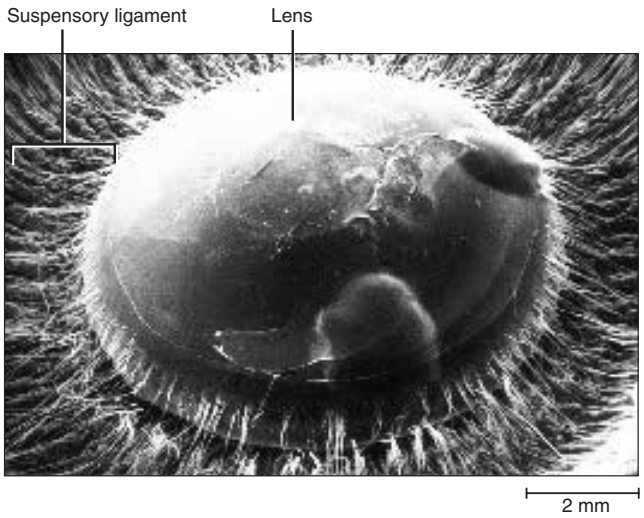


Figure 16.25 **The Lens of the Eye, Posterior View (SEM).**

not under tension, it relaxes into a more spheroid shape and resembles a plastic bead.

- The **vitreous**[47] **body** *(vitreous humor)* is a transparent jelly that fills the large space behind the lens. An oblique channel through this body, called the *hyaloid canal,* is the remnant of a *hyaloid artery* present in the embryo (see fig. 16.23).

[47] *vitre* = glassy

Insight 16.4 Clinical Application

Cataracts and Glaucoma

The two most common causes of blindness are cataracts and glaucoma. A *cataract* is a clouding of the lens. It occurs as the lens thickens with age, and it is a common complication of diabetes mellitus. It causes the vision to appear milky or as if one were looking from behind a waterfall.[48] Cataracts may also stem from heavy smoking and exposure to the UV radiation of the sun. They can be treated by replacing the natural lens with a plastic one. The implanted lens improves vision almost immediately, but glasses still may be needed for near vision.

Glaucoma is a state of elevated pressure within the eye that occurs when the scleral venous sinus is obstructed and aqueous humor is not reabsorbed as fast as it is secreted. Pressure in the anterior and posterior chambers drives the lens back and puts pressure on the vitreous body. The vitreous body presses the retina against the choroid and compresses the blood vessels that nourish the retina. Without a good blood supply, retinal cells die and the optic nerve may atrophy, producing blindness. Symptoms often go unnoticed until the damage is irreversible. In late stages, they include dimness of vision,[49] reduced visual field, and colored halos around artificial lights. Glaucoma can be halted with drugs or surgery, but lost vision cannot be restored. This disease can be detected at an early stage in the course of regular eye examinations. The field of vision is checked, the optic nerve is examined, and the intraocular pressure is measured with an instrument called a *tonometer.*

[48] *cataract* = waterfall
[49] *glauco* = grayness

The Neural Components

The neural components are the retina and optic nerve. The **retina** forms from a cup-shaped outgrowth of the diencephalon (see chapter 14); it is actually a part of the

brain—the only part that can be viewed without dissection. It is a thin transparent membrane attached at only two points—the **optic disc,** where the optic nerve leaves the rear *(fundus)* of the eye, and its scalloped anterior margin, the **ora serrata.** The retina is pressed smoothly against the rear of the eyeball by the pressure of the vitreous body. It can become detached (buckle away from the wall of the eyeball) by blows to the head or because of insufficient pressure from the vitreous body. A *detached retina* may cause blurry areas in the field of vision. It leads to blindness if the retina remains separated for too long from the choroid, on which it depends for oxygen, nutrition, and waste removal.

The retina is examined with an illuminating and magnifying instrument called an *ophthalmoscope.* Directly posterior to the center of the lens, on the visual axis of the eye, is a patch of cells called the **macula lutea**[50] about 3 mm in diameter (fig. 16.26). In the center of the macula is a tiny pit, the **fovea**[51] **centralis,** which produces the most finely detailed images for reasons that will be apparent later. About 3 mm medial to the macula lutea is the optic disc. Nerve fibers from all regions of the retina converge on this point and exit the eye to form the optic nerve. Blood vessels enter and leave the eye by way of the optic disc. Eye examinations serve for more than evaluating the visual system; they allow for a direct, noninvasive examination of blood vessels for signs of hypertension, diabetes mellitus, atherosclerosis, and other vascular diseases.

The optic disc contains no receptor cells, so it produces a **blind spot** in the visual field of each eye. You can detect your blind spot and observe an interesting visual phenomenon with the help of figure 16.27. Close or cover your right eye and hold the page about 30 cm (1 ft) from your face. Fixate on the X with your left eye. Without taking your gaze off the X, move the page slightly forward and back, or right and left, until the red dot disappears. This occurs because the image of the dot is falling on the blind spot of your left eye.

You should notice something else happen at the same time as the dot disappears—a phenomenon called **visual filling.** The green bar seems to fill in the space where the dot used to be. This occurs because the brain uses the image surrounding the blind spot to fill in the area with similar, but imaginary, information. The brain acts as if it is better to assume that the unseen area probably looks like its surroundings than to allow a dark blotch to disturb your vision.

Before we begin to study visual physiology, it would be advisable to review and be sure you thoroughly understand the anatomy of the eye (table 16.3).

[50] *macula* = spot + *lutea* = yellow
[51] *fovea* = pit, depression

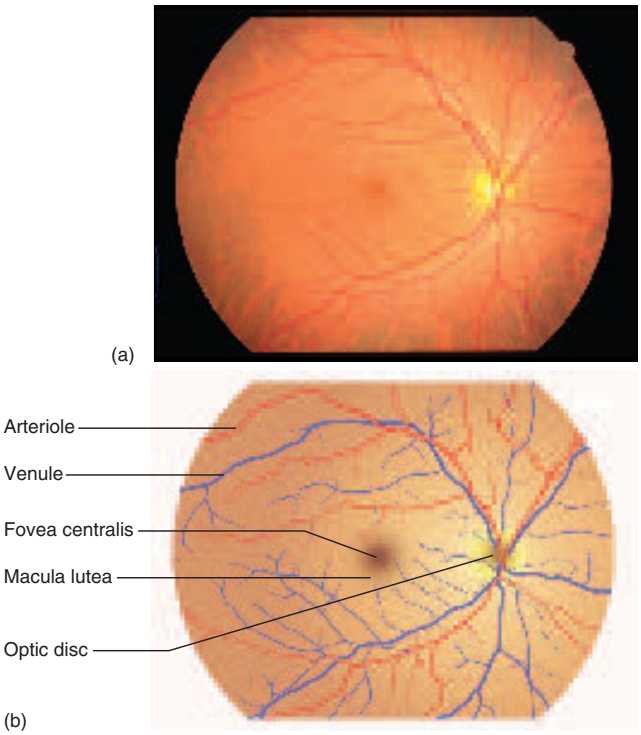


Figure 16.26 The Fundus (rear) of the Eye. (*a*) As seen with an ophthalmoscope; (*b*) anatomical features of the fundus. Note the blood vessels diverging from the optic disc, where they enter the eye with the optic nerve. An eye examination also serves as a partial check on cardiovascular health.

Formation of an Image

The visual process begins when light rays enter the eye, focus on the retina, and produce a tiny inverted image. When fully dilated, the pupil admits five times as much light as it does when fully constricted. Its diameter is controlled by two sets of contractile elements in the iris: (1) The **pupillary constrictor** consists of smooth muscle cells that encircle the pupil. When stimulated by the parasympathetic nervous system, it narrows the pupil and admits less light to the eye. (2) The **pupillary dilator** consists of a spokelike arrangement of modified contractile epithelial cells called *myoepithelial cells.* When stimulated by the sympathetic nervous system, these cells contract, widen the pupil, and admit more light to the eye (see fig. 15.9, p. 577). Pupillary constriction and dilation occur

Figure 16.27 Demonstration of the Blind Spot and Visual Filling. See text for explanation of how to conduct this demonstration.

Table 16.3 Anatomical Review of the Eye and Accessory Structures

Accessory Organs (figs. 16.20 and 16.21)		
Eyebrows	*Conjunctiva*	*Extrinsic muscles* (fig. 16.22)
Eyelids (palpebrae)	*Lacrimal apparatus (fig. 16.21b)*	Superior oblique
Orbicularis oculi	Lacrimal gland	Trochlea
Tarsal plate	Lacrimal puncta	Inferior oblique
Tarsal glands	Lacrimal canal	Superior rectus
Medial and lateral commissures	Lacrimal sac	Inferior rectus
Eyelashes	Nasolacrimal duct	Lateral rectus
Palpebral fissure		Medial rectus
		Orbital fat
Tunics of the Eye (fig. 16.23)		
Fibrous layer	*Vascular layer (uvea)*	*Inner layer*
Sclera	Choroid	Retina
Cornea	Ciliary body	Optic nerve
	Iris	
Optical Components (figs. 16.23–16.25)		
Cornea	Aqueous humor	Suspensory ligament
Pupil	Scleral venous sinus	Vitreous body
Anterior chamber	Lens	Hyaloid canal
Posterior chamber		
Neural Components (fig. 16.26)		
Retina	Optic disc	Macula lutea
Optic nerve	Ora serrata	Fovea centralis

in two situations: when light intensity changes and when we shift our gaze between distant and nearby objects. Pupillary constriction in response to light is called the **photopupillary reflex.** It is also described as a *consensual light reflex* because both pupils constrict even if only one eye is illuminated.

The photopupillary reflex is mediated by a parasympathetic reflex arc. When light intensity rises, signals are transmitted from the eye to the *pretectal region* just rostral to the tectum of the midbrain. Preganglionic parasympathetic fibers originate in the midbrain and travel by way of the oculomotor nerve to the ciliary ganglion in the orbit. From here, postganglionic parasympathetic fibers continue into the eye, where they stimulate the pupillary constrictor. Sympathetic innervation to the pupil originates, like all other sympathetic efferents, in the spinal cord. Preganglionic fibers lead from the thoracic cord to the superior cervical ganglion. From there, postganglionic fibers follow the carotid arteries into the head and lead ultimately to the pupillary dilator.

Refraction

Image formation depends on **refraction,** the bending of light rays. Light travels at a speed of 300,000 km/sec (186,000 mi/sec) in a vacuum, but it slows down slightly in air, water, glass, and other media. The *refractive index* of a medium *(n)* is a measure of how much it retards light rays relative to air. The refractive index of air is arbitrarily set at $n = 1.00$. If light traveling through air strikes a medium of higher refractive index at a 90° *angle of incidence,* it slows down but does not change course—the light rays are not bent. If it strikes at any other angle, however, the light is refracted (fig. 16.28*a*). The greater the difference in refractive index between the two media, and the greater the angle of incidence, the stronger the refraction is.

As light enters the eye, it passes from a medium with $n = 1.00$ (air) to one with $n = 1.38$ (the cornea). Light rays striking the very center of the cornea pass straight through, but because of the curvature of the cornea, rays striking off-center are bent toward the center (fig. 16.28*b*). The

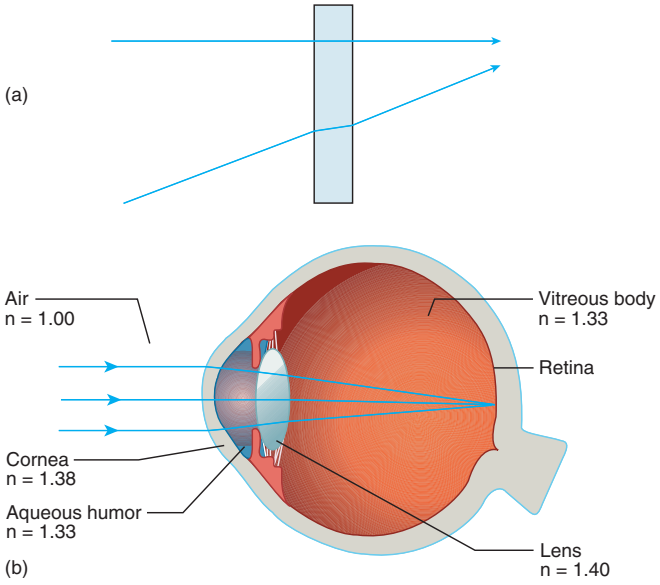


Figure 16.28 Principles of Refraction. (*a*) A refractive medium does not bend light rays that strike it at a 90° angle but does bend rays that enter or leave it at any other angle. (*b*) Refractive indices of the media from air to retina. The greater the difference between the refractive indices of two media, the more strongly light rays are refracted when passing from one to the other. In vision, most refraction (focusing) occurs as light passes from air to cornea.

aqueous humor has a refractive index of 1.33 and does not greatly alter the path of the light. The lens has a refractive index of 1.40. As light passes from air to cornea, the refractive index changes by 0.38; but as it passes from aqueous humor to lens, the refractive index changes by only 0.07. Thus, the cornea refracts light more than the lens does. The lens merely fine-tunes the image, especially as you shift your focus between near and distant objects.

The Near Response

Emmetropia[52] (EM-eh-TRO-pee-uh) is a state in which the eye is relaxed and focused on an object more than 6 m (20 ft) away, the light rays coming from that object are essentially parallel, and the rays are focused on the retina without effort. (An *emmetropic eye* does not need a corrective lens to focus the image.) If the gaze shifts to something closer, light rays from the source are too divergent to be focused without effort. In other words, the eye is automatically focused on things in the distance unless you make an effort to focus elsewhere. For a wild animal or our prehistoric ancestors, this arrangement would be adaptive because it allows for alertness to predators at a distance.

The **near response** (fig. 16.29), or adjustment to close-range vision, involves three processes to focus an image on the retina:

1. **Convergence of the eyes.** Move your finger gradually closer to a baby's nose and the baby will

[52] *em* = in + *metr* = measure + *opia* = vision

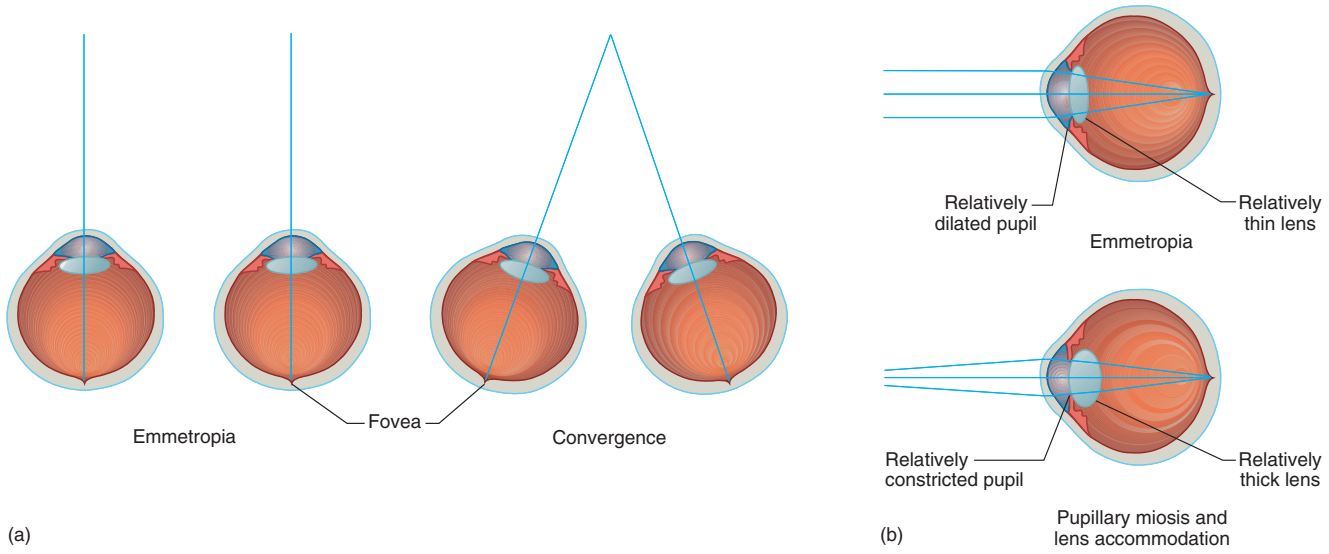


Figure 16.29 Emmetropia and the Near Response. (*a*) Superior view of both eyes fixated on an object more than 6 m away (*left*), and both eyes fixated on an object closer than 6 m (*right*). (*b*) Lateral view of the eye fixated on a distant object (*top*) and nearby object (*bottom*).

go cross-eyed. This **convergence** of the eyes orients the visual axis of each eye toward the object in order to focus its image on each fovea. If the eyes cannot converge accurately—for example, when the extrinsic muscles are weaker in one eye than in the other—double vision or *diplopia*[53] results. The images fall on different parts of the two retinas and the brain sees two images. You can simulate this effect by pressing gently on one eyelid as you look at this page; the image of the print will fall on noncorresponding regions of the two eyes and cause you to see double.

2. **Constriction of the pupil.** Lenses cannot refract light rays at their edges as well as they can closer to the center. The image produced by any lens is therefore somewhat blurry around the edges; this *spherical aberration* is quite evident in an inexpensive microscope. It can be minimized by screening out these peripheral light rays, and for this purpose, the pupil constricts as you focus on nearby objects. Like the diaphragm of a camera, the pupil thus has a dual purpose—to adjust the eye to variations in brightness and to reduce spherical aberration.
3. **Accommodation of the lens. Accommodation** is a change in the curvature of the lens that enables you to focus on a nearby object. When you look at something nearby, the ciliary muscle surrounding the lens contracts. This narrows the diameter of the ciliary body, relaxes the fibers of the suspensory ligament, and allows the lens to relax into a more convex shape (fig. 16.30). In emmetropia, the lens is about 3.6 mm thick at the center; in accommodation, it thickens to about 4.5 mm. A more convex lens refracts light more strongly and focuses the divergent light rays onto the retina. The closest an object can be and still come into focus is called the **near point of vision.** It depends on the flexibility of the lens. The lens stiffens with age, so the near point averages about 9 cm at the age of 10 and 83 cm by the age of 60.

Some common defects in image formation are listed in table 16.4.

Think About It

Which extrinsic muscles of the eyes are the prime movers in convergence?

Sensory Transduction in the Retina

The conversion of light energy into action potentials occurs in the retina. We begin our exploration of this process with the cellular layout of the retina (fig. 16.32). From there we go to the pigments that absorb light and then to what happens when light is absorbed.

The most posterior layer of the retina is the **pigment epithelium,** a layer of darkly pigmented cuboidal cells whose basal processes interdigitate with receptor cells of the retina. The pigment here is not involved in nerve signaling;

[53] *dipl* = double + *opia* = vision

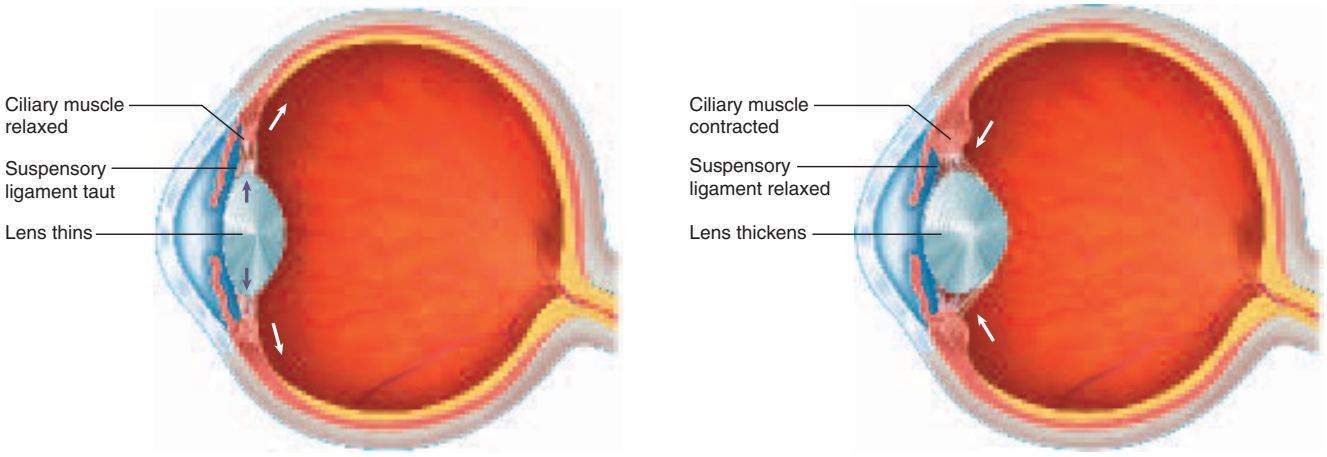


Figure 16.30 Accommodation of the Lens. (*a*) In the relaxed (emmetropic) eye, the ciliary muscle is relaxed and dilated. It puts tension on the suspensory ligament and flattens the lens. (*b*) In accommodation, the ciliary muscle contracts and narrows in diameter. This reduces tension on the suspensory ligament and allows the lens to relax into a more convex shape.

Table 16.4 Common Defects of Image Formation

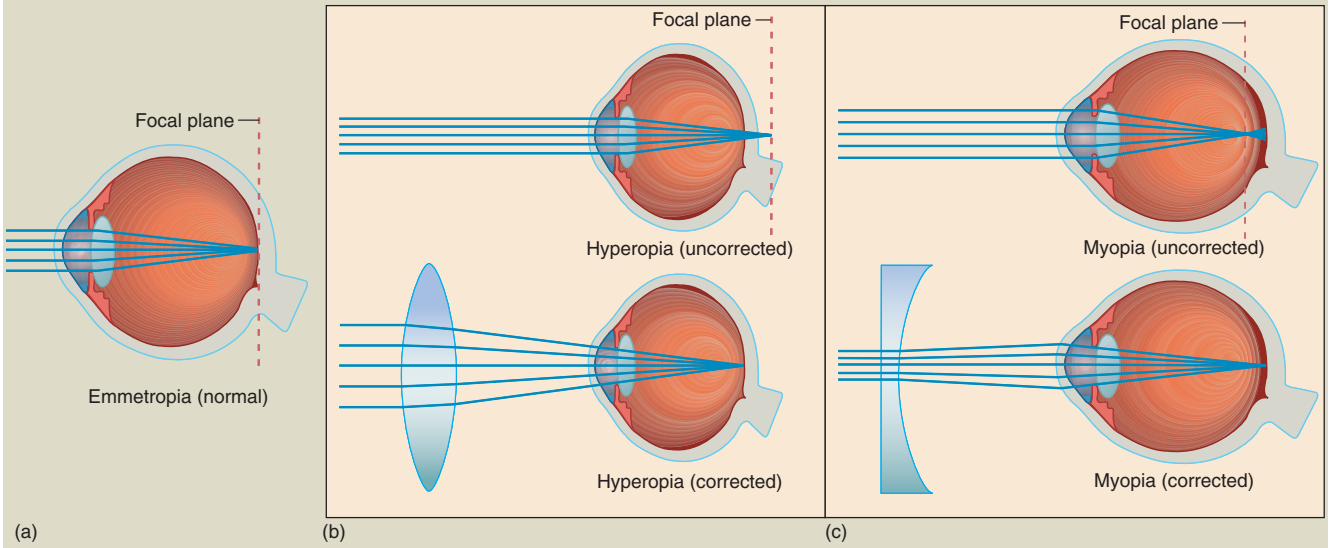


Figure 16.31 Two Common Visual Defects and the Effects of Corrective Lenses. (*a*) The normal emmetropic eye, with light rays converging on the retina. (*b*) Hyperopia (far-sightedness) and the corrective effect of a convex lens. (c) Myopia (near-sightedness) and the corrective effect of a concave lens.

Presbyopia	Reduced ability to accommodate for near vision with age because of declining flexibility of the lens. Results in difficulty in reading and doing close handwork. Corrected with bifocal lenses.
Hyperopia	Farsightedness—a condition in which the eyeball is too short. The retina lies in front of the focal point of the lens, and the light rays have not yet come into focus when they reach the retina (see top of fig. 16.31*b*). Causes the greatest difficulty when viewing nearby objects. Corrected with convex lenses, which cause light rays to converge slightly before entering the eye.
Myopia	Nearsightedness—a condition in which the eyeball is too long. Light rays come into focus before they reach the retina and begin to diverge again by the time they fall on it (see top of fig. 16.31*c*). Corrected with concave lenses, which cause light rays to diverge slightly before entering the eye.
Astigmatism	Inability to simultaneously focus light rays that enter the eye on different planes. Focusing on vertical lines, such as the edge of a door, may cause horizontal lines, such as a tabletop, to go out of focus. Caused by a deviation in the shape of the cornea so that it is shaped like the back of a spoon rather than like part of a sphere. Corrected with cylindrical lenses, which refract light more in one plane than another.

rather, its purpose is to absorb light that is not absorbed first by the receptor cells and to prevent it from degrading the visual image by reflecting back into the eye. It acts like the blackened inside of a camera to reduce stray light.

The neural components of the retina consist of three principal cell layers. Progressing from the rear of the eye forward, these are composed of *photoreceptor cells, bipolar cells,* and *ganglion cells:*

1. **Photoreceptor cells.** The photoreceptors are all cells that absorb light and generate a chemical or electrical signal. There are three kinds of photoreceptors in the retina: rods, cones, and some of the ganglion cells. Only the rods and cones produce visual images; the ganglion cells are discussed shortly. **Rods** and **cones** are derived from the same stem cells that produce ependymal cells of the brain. Each rod or cone has an **outer segment** that points toward the wall of the eye and an **inner segment** facing the interior (fig. 16.33). The two segments are separated by a narrow constriction containing nine pairs of microtubules; the outer segment is actually a highly modified cilium specialized to absorb light. The inner segment contains mitochondria and other organelles. At its base, it gives rise to a cell body, which contains the nucleus, and to processes that synapse with retinal neurons in the next layer.

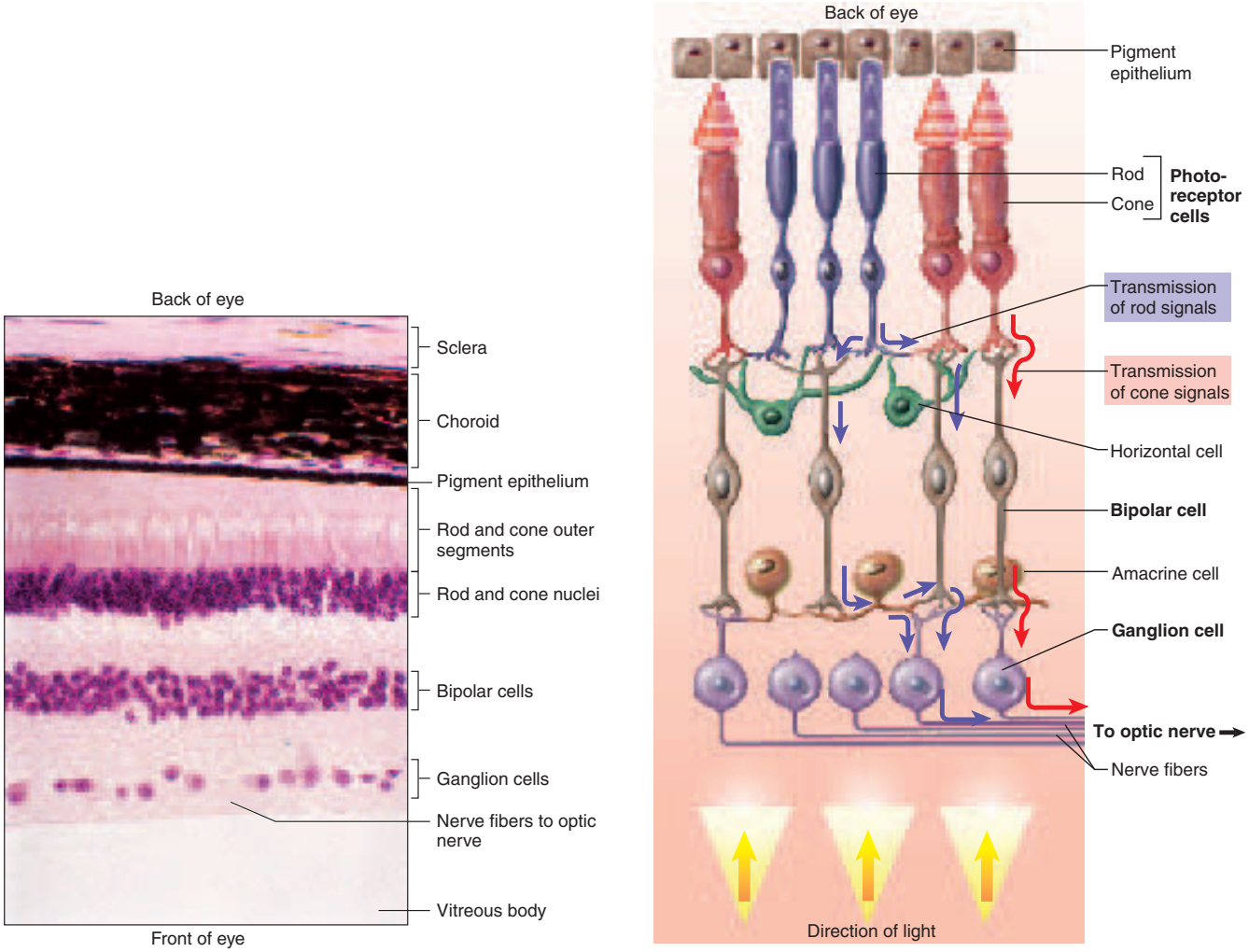


Figure 16.32 Histology of the Retina. (*a*) Photomicrograph. (*b*) Schematic of the layers and synaptic relationships of the retinal cells.

In a rod, the outer segment is cylindrical and resembles a stack of coins in a paper roll—there is a plasma membrane around the outside and a neatly arrayed stack of about 1,000 membranous discs inside. Each disc is densely studded with globular proteins—the visual pigment *rhodopsin,* to be discussed later. The membranes hold these pigment molecules in a position that results in the most efficient light absorption. Rod cells are responsible for **night (scotopic[54]) vision;** they cannot distinguish colors from each other.

A cone cell is similar except that the outer segment tapers to a point and the discs are not detached from the plasma membrane but are parallel infoldings of it. Cones function in bright light; they are responsible for **day (photopic[55]) vision** as well as color vision.

2. **Bipolar cells.** Rods and cones synapse with the dendrites of **bipolar cells,** the first-order neurons of the visual pathway. They in turn synapse with the ganglion cells described next (see fig. 16.32*b*). There are approximately 130 million rods and 6.5 million cones in one retina, but only 1.2 million nerve fibers in the optic nerve. With a ratio of 114 receptor cells to 1 optic nerve fiber, it is obvious that there must be substantial *neuronal convergence*

[54] *scot* = dark + *op* = vision

[55] *phot* = light + *op* = vision

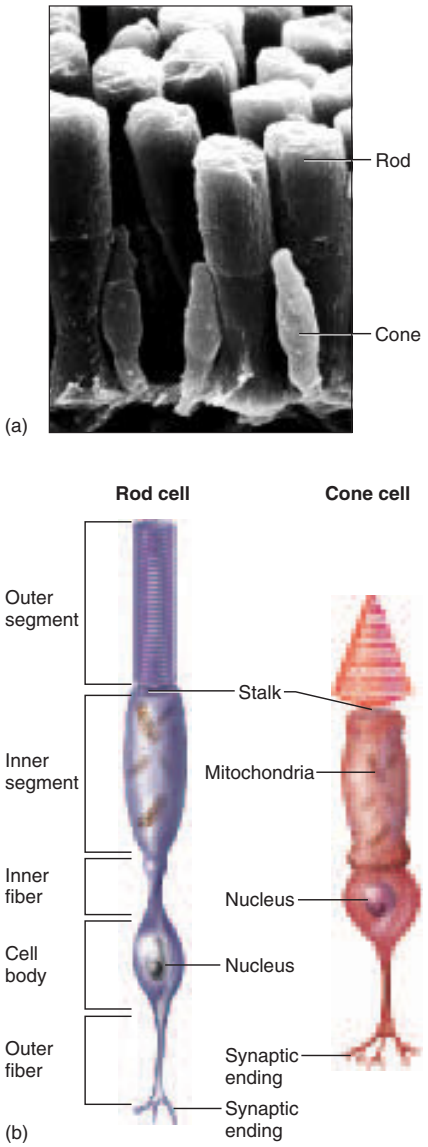


Figure 16.33 Rod and Cone Cells. (*a*) Rods and cones of a salamander retina (SEM). The tall cylindrical cells are rods and the short tapered cells (*foreground*) are cones. (*b*) Structure of rods and cones.

and information processing in the retina itself before signals are transmitted to the brain proper. Convergence begins with the bipolar cells.

3. **Ganglion cells. Ganglion cells** are the largest neurons of the retina, arranged in a single layer close to the vitreous body. They are the second-order neurons of the visual pathway. Most ganglion cells receive input from multiple bipolar cells. The ganglion cell axons form the optic nerve. Some of the ganglion cells absorb light directly and transmit signals to brainstem nuclei that control pupillary diameter and the body's circadian rhythms. They do not contribute to visual images but detect only light intensity.

There are other retinal cells, but they do not form layers of their own. **Horizontal cells** and **amacrine**[56] **cells** form horizontal connections among rod, cone, and bipolar cells. They play diverse roles in enhancing the perception of contrast, the edges of objects, and changes in light intensity. In addition, much of the mass of the retina is composed of astrocytes and other types of glial cells.

Visual Pigments

The visual pigment of the rods is called **rhodopsin** (ro-DOP-sin), or *visual purple.* Each molecule consists of two major parts (moieties)—a protein called **opsin** and a vitamin A derivative called **retinal** (rhymes with "pal"), also known as **retinene** (fig. 16.34). Opsin is embedded in the disc membranes of the rod's outer segment. All rod cells contain a single kind of rhodopsin with an absorption peak at a wavelength of 500 nm. The rods are less sensitive to light of other wavelengths.

In cones, the pigment is called **photopsin (iodopsin).** Its retinal moiety is the same as that of rhodopsin, but the opsin moieties have different amino acid sequences that determine which wavelengths of light the pigment absorbs. There are three kinds of cones, which are identical in appearance but optimally absorb different wavelengths of light. These differences, as you will see shortly, enable us to perceive different colors.

The pigment employed by the photosensitive ganglion cells is thought to be *melanopsin,* but this is still awaiting proof.

The Photochemical Reaction

The events of sensory transduction are probably the same in rods and cones, but rods and rhodopsin have been better studied than cones and photopsin. In the dark, retinal has a bent shape called ***cis*-retinal.** When it absorbs light, it changes to a straight form called ***trans*-retinal,** and the retinal dissociates from the opsin (fig. 16.35). Purified rhodopsin changes from violet to colorless when this happens, so the process is called the **bleaching** of rhodopsin.

For a rod to continue functioning, it must regenerate rhodopsin at a rate that keeps pace with bleaching. When *trans*-retinal dissociates from opsin, it is transported to the pigment epithelium, converted back to *cis*-retinal, returned to the rod outer segment, and reunited with opsin. It takes about 5 minutes to regenerate 50% of the bleached rhodopsin. Cone cells are less dependent on the pigment epithelium and regenerate half of their pigment in about 90 seconds.

[56] *a* = without + *macr* = long + *in* = fiber

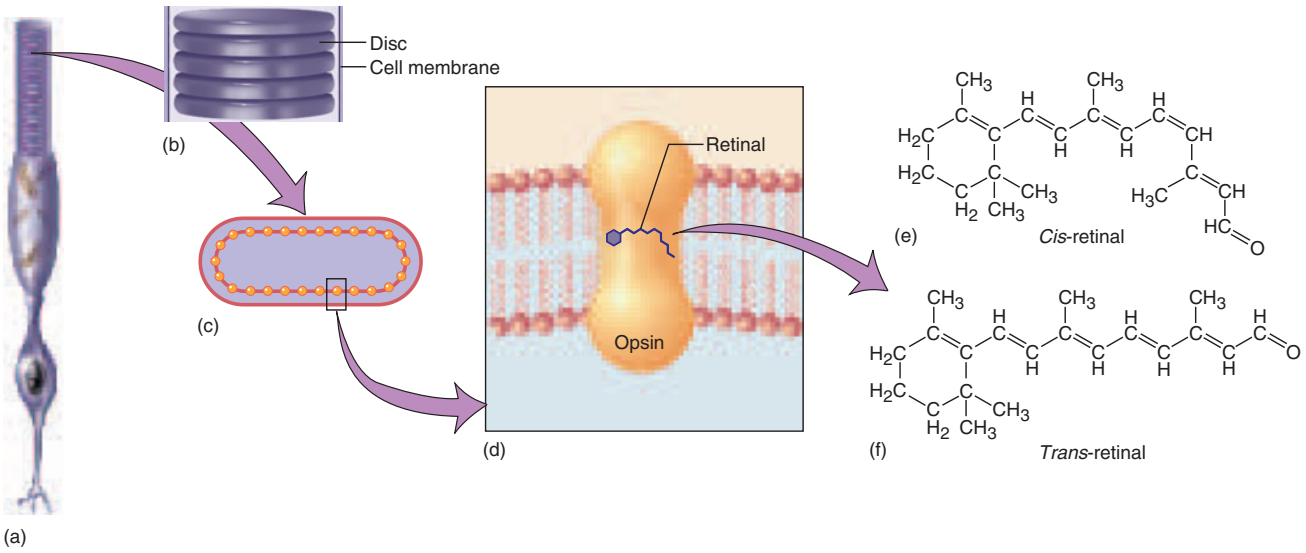


Figure 16.34 Structure and Location of the Visual Pigments. (*a*) A rod cell. (*b*) Detail of the rod outer segment. (*c*) One disc of the outer segment showing the membrane studded with pigment molecules. (*d*) A pigment molecule, embedded in the unit membrane of the disc, showing the protein moiety, opsin, and the vitamin A derivative, retinal. (*e*) *Cis*-retinal, the isomer present in the absence of light. (*f*) *Trans*-retinal, the isomer produced when the pigment absorbs a photon of light.

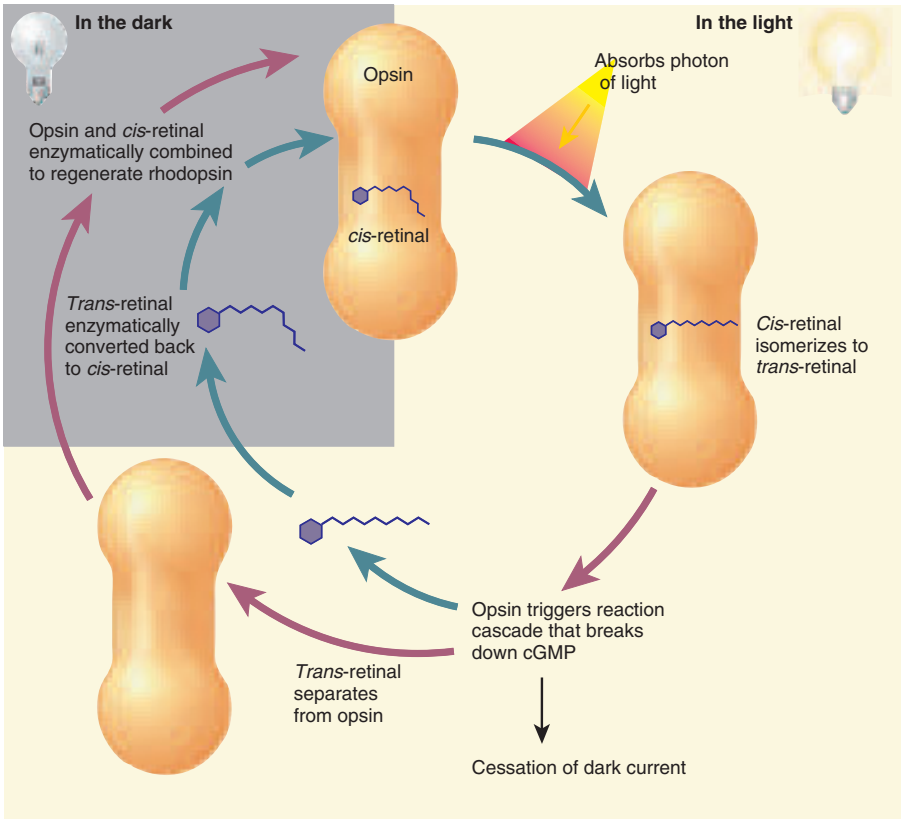


Figure 16.35 The Bleaching and Regeneration of Rhodopsin. The *yellow* background indicates the bleaching events that occur in the light; the *gray* background indicates the regenerative events that are independent of light. The latter events occur in light and dark but are able to outpace bleaching only in the dark.

Generating the Optic Nerve Signal

In the dark, rods do not sit quietly doing nothing. They exhibit a **dark current,** a steady flow of sodium ions into the outer segment, and as long as this is happening, they release a neurotransmitter, glutamate, from the basal end of the cell (fig. 16.36*a*). When a rod absorbs light, the dark current and glutamate secretion cease (fig. 16.36*b*). The on-and-off glutamate secretion influences the bipolar cells in ways we will examine shortly, but first we will explore why the dark current occurs and why it stops in the light.

The outer segment of the rod has ligand-regulated Na^+ gates that bind cyclic guanosine monophosphate (cGMP) on their intracellular side. cGMP opens the gate and permits the inflow of Na^+. This Na^+ current reduces the membrane potential of the rod from the −70 mV typical of neurons to about −40 mV. This depolarization stimulates glutamate secretion. Two mechanisms, however, prevent the membrane from depolarizing more than that: (1) The rod has nongated K^+ channels in the inner segment, which allow K^+ to leave as Na^+ enters. (2) The inner segment has a high density of Na^+-K^+ pumps, which constantly pump Na^+ back out of the cell and bring K^+ back in.

Why does the dark current cease when a rod absorbs light? The intact rhodopsin molecule is essentially a dormant enzyme. When it bleaches, it becomes enzymatically active and triggers a cascade of reactions that ultimately break down several hundred thousand molecules of

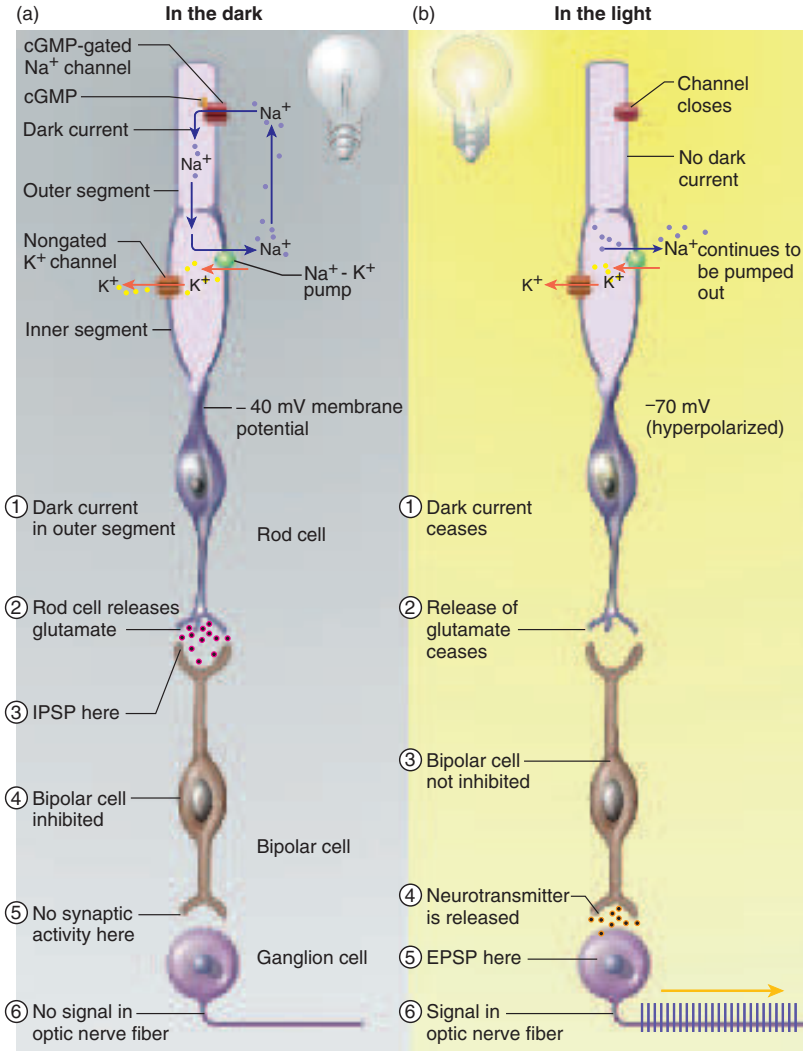


Figure 16.36 Mechanism of Generating Visual Signals. (*a*) In the dark, cGMP opens a sodium gate and a dark current in the rod cell stimulates glutamate release. (*b*) In the light, cGMP breaks down and its absence shuts off the dark current and glutamate secretion. The bipolar cell in this case is inhibited by glutamate and stimulates the ganglion cell when glutamate secretion decreases.

cGMP. As cGMP is degraded, the Na^+ gates in the outer segment close, the dark current ceases, and the Na^+-K^+ pump shifts the membrane voltage toward −70 mV. This shift causes the rod to stop secreting glutamate. The sudden drop in glutamate secretion informs the bipolar cell that the rod has absorbed light.

There are two kinds of bipolar cells. One type is inhibited (hyperpolarized) by glutamate and thus excited (depolarized) when its secretion drops. This type of cell is excited by *rising* light intensity. The other type is excited by glutamate and inhibited when its secretion drops, so it is excited by *falling* light intensity. As your eye scans a scene, it passes areas of greater and lesser brightness. Their images on the retina cause a rapidly changing pattern of bipolar cell responses as the light intensity on a patch of retina rises and falls.

When bipolar cells detect fluctuations in light intensity, they stimulate ganglion cells either directly (by synapsing with them) or indirectly (via pathways that go through amacrine cells). Each ganglion cell receives input from a circular patch of retina called its receptive field. The principal function of most ganglion cells is to code for contrast between the center and edge of its receptive field—that is, between an object and its surroundings. Ganglion cells are the only retinal cells that produce action potentials; all other retinal cells produce only graded local potentials. The ganglion cells respond with rising and falling firing frequencies which, via the optic nerve, provide the brain with a basis for interpreting the image on the retina.

Light and Dark Adaptation

Light adaptation occurs when you go from the dark into bright light. If you wake up in the night and turn on a lamp, at first you see a harsh glare; you may experience discomfort from the overstimulated retinas. Your pupils quickly constrict to reduce the intensity of stimulation, but color vision and visual acuity (the ability to see fine detail) remain below normal for 5 to 10 minutes—the time needed for pigment bleaching to adjust retinal sensitivity to this light intensity. The rods bleach quickly in bright light, and cones take over. Even in typical indoor light, rod vision is nonfunctional.

On the other hand, suppose you are sitting in a bright room at night and there is a power failure. Your eyes must undergo **dark adaptation** before you can see well enough to find your way in the dark. Your rod pigment was bleached by the lights in the room while the power was on, but now in the relative absence of light, rhodopsin regenerates faster than it bleaches. In 20 to 30 minutes, the amount of rhodopsin is sufficient for your eyes to have reached essentially maximum sensitivity. Dilation of the pupils also helps by admitting more light to the eye.

The Duplicity Theory

You may wonder why we have both rods and cones. Why can't we simply have one type of receptor cell that would produce detailed color vision, both day and night? The **duplicity theory** of vision holds that a single type of receptor cell cannot produce both high sensitivity and high resolution. It takes one type of cell and neuronal circuit to provide sensitive night vision and a different type of receptor and circuit to provide high-resolution daytime vision.

The high sensitivity of rods in dim light stems partly from the cascade of reactions leading to cGMP breakdown described earlier; a single photon leads to the breakdown of hundreds of thousands of cGMP molecules. But the sensitivity of scotopic (rod) vision is also due to the extensive neuronal convergence that occurs between the rods and ganglion cells. Up to 600 rods converge on each bipolar cell, and many bipolar cells converge on each ganglion cell. This allows for a high degree of *spatial summation* in the scotopic system (fig. 16.37*a*). Weak stimulation of many rod cells can produce an additive effect on one bipolar cell, and several bipolar cells can collaborate to excite one ganglion cell. Thus, a ganglion cell can respond in dim light that only weakly stimulates any individual rod. Scotopic vision is functional even at a light intensity less than starlight reflected from a sheet of white paper. A shortcoming of this system is that it cannot resolve finely detailed images. One ganglion cell receives input from all the rods in about 1 mm^2 of retina—its receptive field. What the brain perceives is therefore a coarse, grainy image similar to an overenlarged newspaper photograph.

Around the edges of the retina, receptor cells are especially large and widely spaced. If you fixate on the middle of this page, you will notice that you cannot read the words near the margins. Visual acuity decreases rapidly as the image falls away from the fovea centralis. Our peripheral vision is a low-resolution system that serves mainly to alert us to motion in the periphery and to stimulate us to look that way to identify what is there.

When you look directly at something, its image falls on the fovea, which is occupied by about 4,000 tiny cones and no rods. The other neurons of the fovea are displaced to one side so they won't interfere with light falling on the cones. The smallness of these cones is like the smallness of the dots in a high-quality photograph; it is partially responsible for the high-resolution images formed at the fovea. In addition, the cones here show no neuronal convergence. Each cone synapses with only one bipolar cell and each bipolar cell with only one ganglion cell. This gives each foveal cone a "private line to the brain," and each ganglion cell of the fovea reports to the brain on a receptive field of just 2 μm^2 of retinal area (fig. 16.37*b*). Cones distant from the fovea exhibit some neuronal convergence but not nearly as much as rods do. The price of this lack of convergence at the fovea, however, is that cone

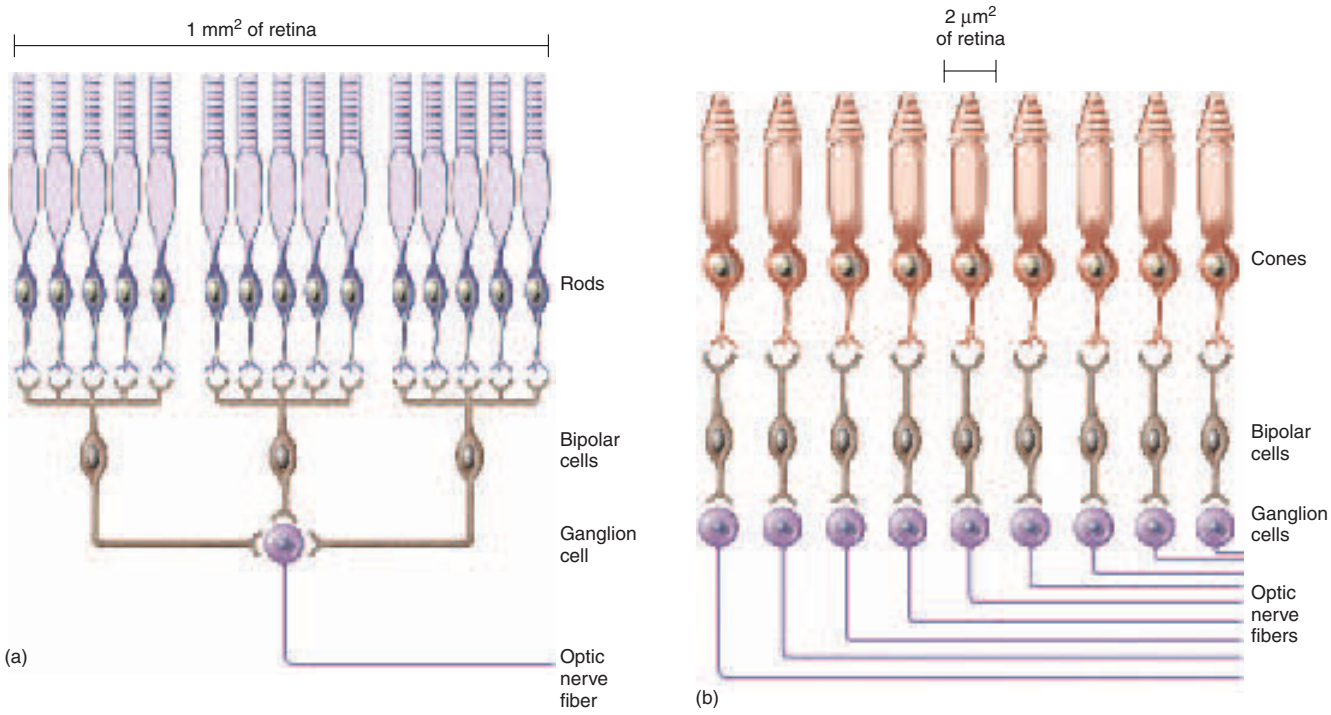


Figure 16.37 The Duplicity Theory of Vision. (*a*) In the scotopic (night vision) system, many rods converge on each bipolar cell and many bipolar cells converge on each ganglion cell (via amacrine cells, not shown). This allows extensive spatial summation—many rods add up their effects to stimulate a ganglion cell even in dim light. However, it means that each ganglion cell (and its optic nerve fiber) represents a relatively large area of retina and produces a grainy image. (*b*) In the photopic (day vision) system, there is little neuronal convergence. In the fovea, represented here, each cone has a "private line" to the brain, so each optic nerve fiber represents a tiny area of retina, and vision is relatively sharp. However, the lack of convergence prevents spatial summation. Photopic vision does not function well in dim light because weakly stimulated cones cannot collaborate to stimulate a ganglion cell.

cells have little spatial summation, and the cone system therefore has less sensitivity to light. The threshold of photopic (cone) vision lies between the intensity of starlight and moonlight reflected from white paper.

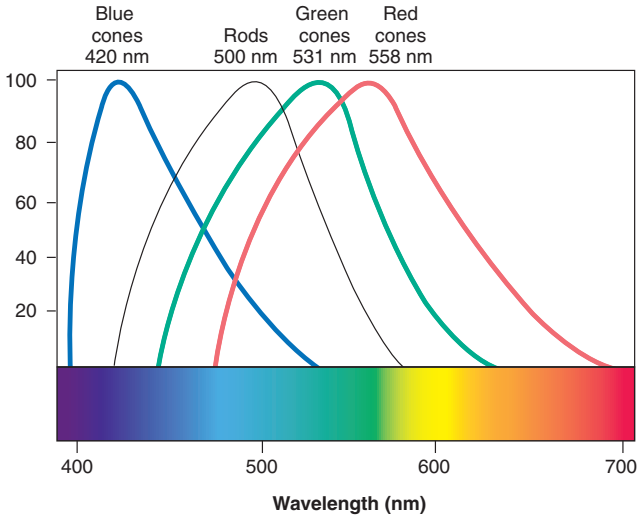
Think About It

If you look directly at a dim star in the night sky, it disappears, and if you look slightly away from it, it reappears. Why?

Color Vision

Most nocturnal vertebrates have only rod cells, but many diurnal animals are endowed with cones and color vision. Color vision is especially well developed in primates for evolutionary reasons discussed in chapter 1. It is based on three kinds of cones named for the absorption peaks of their photopsins: **blue cones,** with peak sensitivity at 420 nm; **green cones,** which peak at 531 nm; and **red cones,** which peak at 558 nm. Red cones do not peak in the red part of the spectrum (558 nm light is perceived as orange-yellow), but they are the only cones that respond at all to red light. Our perception of different colors is based on a mixture of nerve signals representing cones with different absorption peaks. In figure 16.38, note that light at 400 nm excites only the blue cones, but at 500 nm, all three types of cones are stimulated. The red cones respond at 60% of their maximum capacity, green cones at 82% of their maximum, and blue cones at 20%. The brain interprets this mixture of signals as blue-green. The table in figure 16.38 shows how some other color sensations are generated by other response ratios.

Some individuals have a hereditary lack of one photopsin or another and consequently exhibit **color blindness.** The most common form is *red-green color blindness,* which results from a lack of either red or green cones and renders a person incapable of distinguishing these and related shades from each other. For example, a person with normal *trichromatic* color vision sees figure 16.39 as the number 16, whereas a person with red-green color



Wavelength (nm)	Percent of maximum cone response (red:green:blue)	Perceived hue
400	0:0:50	Violet
450	0:30:72	Blue
500	60:82:20	Blue-green
550	97:85:0	Green
625	35:3:0	Orange
675	5:0:0	Red

Figure 16.38 Absorption Spectra of the Retinal Cells. In the *middle column* of the table, each number indicates how strongly the respective cone cells respond as a percentage of their maximum capability. At 550 nm, for example, red cones respond at 97% of their maximum, green cones at 85%, and blue cones not at all. The result is a perception of green light.
If you were to add another row to this table, for 600 nm, what would you enter in the middle and right-hand columns?

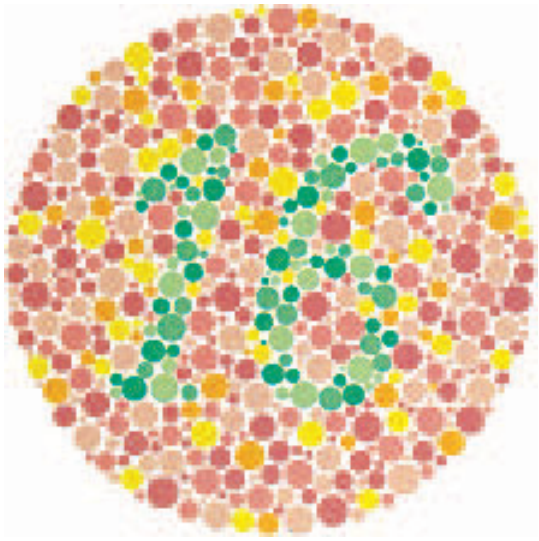


Figure 16.39 A Test for Red-Green Color Blindness. Persons with normal vision see the number 16. Persons with red-green color blindness see no discernible number. Reproduced from *Ishihara's Tests for Colour Blindness,* Kenahara Trading Co., Tokyo, copyright © Isshin-Kai Foundation. Accurate tests of color vision cannot be performed with such reprinted plates, but must use the original plates.

blindness sees no number. Red-green color blindness is a sex-linked recessive trait. It occurs in about 8% of males and 0.5% of females. (See p. 149 to review sex linkage and the reason such traits are more common in males.)

Stereoscopic Vision

Stereoscopic vision (stereopsis) is depth perception—the ability to judge how far away objects are. It depends on having two eyes with overlapping visual fields, which allows each eye to look at the same object from a different angle. Stereoscopic vision contrasts with the *panoramic vision* of mammals such as rodents and horses, where the eyes are on opposite sides of the head. Although stereoscopic vision covers a smaller visual field than panoramic vision and provides less alertness to sneaky predators, it has the advantage of depth perception. The evolutionary basis of depth perception in primates was considered in chapter 1 (p. 11).

When you fixate on something within 30 m (100 ft) away, each eye views it from a slightly different angle and focuses its image on the fovea centralis. The point on which the eyes are focused is called the *fixation point.* Objects farther away than the fixation point cast an image somewhat medial to the foveas, and closer objects cast their images more laterally (fig. 16.40). The distance of an image from the two foveas provides the brain with information used to judge the position of other points relative to the fixation point.

The Visual Projection Pathway

The first-order neurons in the visual pathway are the bipolar cells of the retina. They synapse with the second-order neurons, the retinal ganglion cells, whose axons are the fibers of the optic nerve. The optic nerves leave each orbit through the optic foramen and then converge on each other to form an X, the **optic chiasm**[57] (ky-AZ-um), immediately inferior to the hypothalamus and anterior to the pituitary. Beyond this, the fibers continue as a pair of **optic tracts** (see p. 548). Within the chiasm, half the fibers of each optic nerve cross over to the opposite side of the brain (fig. 16.41). This is called **hemidecussation,**[58] since

[57] *chiasm* = cross, X
[58] *hemi* = half + *decuss* = to cross, form an X

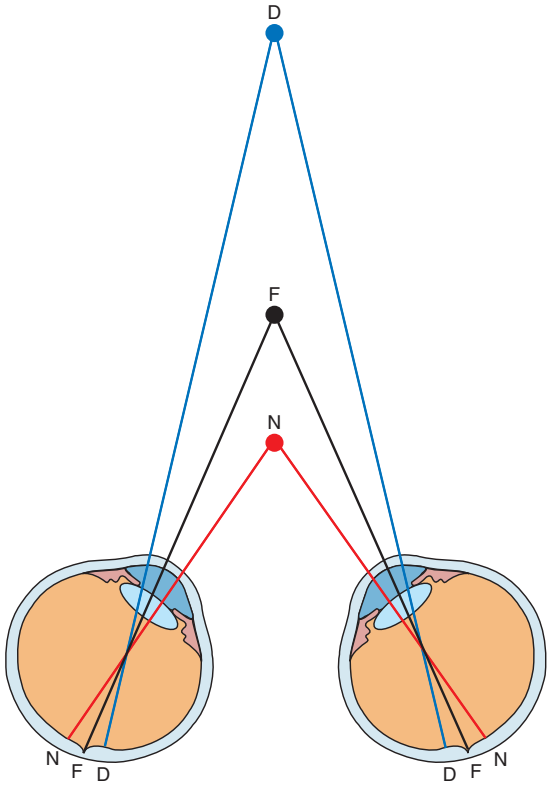


Figure 16.40 The Retinal Basis of Stereoscopic Vision (depth perception). When the eyes are fixated on the fixation point (*F*), more distant objects (*D*) are focused on the retinas medial to the fovea and the brain interprets them as being farther away than the fixation point. Nearby objects (*N*) are focused lateral to the fovea and interpreted as being closer.

only half of the fibers decussate. As a result, objects in the left visual field, whose images fall on the right half of each retina (the medial half of the left eye and lateral half of the right eye), are perceived by the right cerebral hemisphere. Objects in the right visual field are perceived by the left hemisphere. Since the right brain controls motor responses on the left side of the body and vice versa, each side of the brain needs to see what is on the side of the body where it exerts motor control. In animals with panoramic vision, nearly 100% of the optic nerve fibers of the right eye decussate to the left brain and vice versa.

The optic tracts pass laterally around the hypothalamus, and most of their axons end in the **lateral geniculate**[59] (jeh-NIC-you-late) **nucleus** of the thalamus. Third-order neurons arise here and form the **optic radiation** of fibers in the white matter of the cerebrum. These project to the primary visual cortex of the occipital lobe, where the conscious visual sensation occurs. A lesion in the occipital lobe can cause blindness even if the eyes are fully functional.

A few optic nerve fibers take a different route in which they project to the midbrain and terminate in the superior colliculi and pretectal nuclei. The superior colliculi control the visual reflexes of the extrinsic eye muscles, and the pretectal nuclei are involved in the photopupillary and accommodation reflexes.

Space does not allow us to consider much about the very complex processes of visual information processing in the brain. Some processing, such as contrast, brightness, motion, and stereopsis, begins in the retina. The primary visual cortex in the occipital lobe is connected by association tracts to nearby visual association areas in the posterior part of the parietal lobe and inferior part of the temporal lobe. These association areas process retinal data in ways beyond our present consideration to extract information about the location, motion, color, shape, boundaries, and other qualities of the objects we look at. They also store visual memories and enable the brain to identify what we are seeing—for example, to recognize printed words or name the objects we see. What is yet to be learned about visual processing promises to have important implications for biology, medicine, psychology, and even philosophy.

Before You Go On

Answer the following questions to test your understanding of the preceding section:

20. Why can't we see wavelengths below 350 nm or above 750 nm?
21. Why are light rays bent (refracted) more by the cornea than by the lens?
22. List as many structural and functional differences between rods and cones as you can.
23. Explain how the absorption of a photon of light leads to depolarization of a bipolar retinal cell.
24. Discuss the duplicity theory of vision, summarizing the advantage of having separate types of retinal photoreceptor cells for photopic and scotopic vision.

Insight 16.5 Medical History

Anesthesia—From Ether Frolics to Modern Surgery

Surgery is as old as civilization. People from the Stone Age to the pre-Columbian civilizations of the Americas practiced *trephination*—cutting a hole in the skull to let out "evil spirits" that were thought to cause headaches. The ancient Hindus were expert surgeons for their time, and the Greeks and Romans pioneered military surgery. But until the nineteenth century, surgery was a miserable and dangerous business, done only as a last resort and with little hope of the patient's survival. Surgeons rarely attempted anything more complex than amputations or kidney stone removal. A surgeon had to be somewhat indifferent to the struggles and screams of his patient. Most operations

[59] *geniculate* = bent like a knee

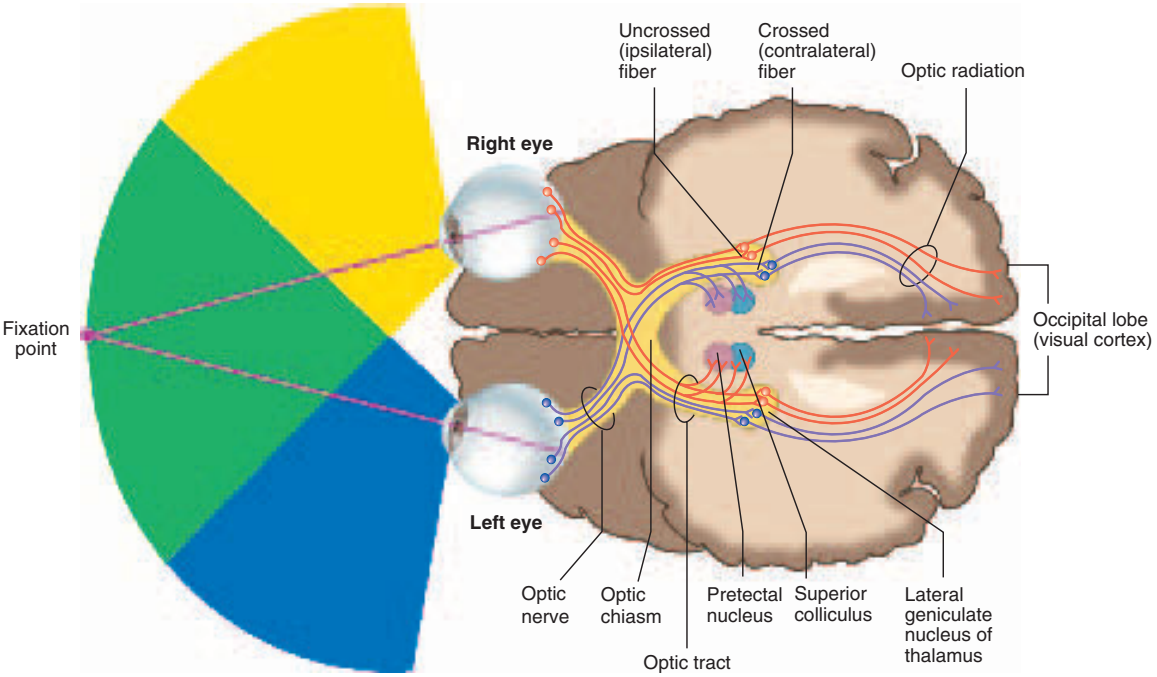


Figure 16.41 The Visual Projection Pathway. Diagram of hemidecussation and projection to the primary visual cortex. *Blue* and *yellow* indicate the receptive fields of the left and right eyes; *green* indicates the area of overlap and stereoscopic vision. Nerve fibers from the medial side of the right eye (*red*) descussate to the left side of the brain, while fibers from the lateral side remain on the right side of the brain. The converse is true of the left eye. The right occipital lobe thus monitors the left side of the visual field and the left occipital lobe monitors the right side.

If a stroke destroyed the optic radiation of the right cerebral hemisphere, how would it affect a person's vision? Would it affect the person's visual reflexes?

had to be completed in 3 minutes or less, and a strong arm and stomach were more important qualifications for a surgeon than extensive anatomical knowledge.

At least three things were needed for surgery to be more effective: better knowledge of anatomy, *asepsis*[60] for the control of infection, and *anesthesia*[61] for the control of pain. Early efforts to control surgical pain were crude and usually ineffective, such as choking a patient into unconsciousness and trying to complete the surgery before he or she awoke. Alcohol and opium were often used as anesthetics, but the dosage was poorly controlled; some patients were underanesthetized and suffered great pain anyway, and others died of overdoses. Often there was no alternative but for a few strong men to hold the struggling patient down as the surgeon worked. Charles Darwin originally intended to become a physician, but left medical school because he was sickened by observing "two very bad operations, one on a child," in the days before anesthesia.

In 1799, Sir Humphrey Davy suggested using nitrous oxide to relieve pain. His student, Michael Faraday, suggested ether. Neither of these ideas caught on for several decades, however. Nitrous oxide ("laughing gas") was a popular amusement in the 1800s, when traveling showmen went from town to town demonstrating its effects on volunteers from the audience. In 1841, at a medicine show in Georgia, some students were impressed with the volunteers' euphoric giggles and antics and asked a young local physician, Crawford W. Long, if he could make some nitrous oxide for them. Long lacked the equipment to synthesize it, but he recommended they try ether. Ether was commonly used in small oral doses for toothaches and "nervous ailments," but its main claim to popularity was its use as a party drug for so-called ether frolics. Long himself was a bit of a *bon vivant* who put on demonstrations for some of the young ladies, with the disclaimer that he could not be held responsible for whatever he might do under the influence of ether (such as stealing a kiss).

At these parties, Long noted that people sometimes suffered considerable injuries without feeling pain. In 1842, he had a patient who was terrified of pain but needed a tumor removed from his neck. Long excised the tumor without difficulty as his patient sniffed ether from a towel. The operation created a sensation in town, but other physicians ridiculed Long and pronounced anesthesia dangerous. His medical practice declined as people grew afraid of him, but over the next 4 years he performed eight more minor surgeries on patients under ether. Struggling to overcome criticisms that the effects he saw were due merely to hypnotic suggestion or individual variation in sensitivity to pain, Long even compared surgeries done on the same person with and without ether.

Long failed to publish his results quickly enough, and in 1844 he was scooped by a Connecticut dentist, Horace Wells, who had tried nitrous oxide as a dental anesthetic. Another dentist, William Morton of Boston, had tried everything from champagne to opium to kill pain in his patients. He too became interested in ether and gave a public demonstration at Massachusetts General Hospital, where he etherized a patient and removed a tumor. Within a month of this successful and sensational demonstration, ether was being used in other cities of the

United States and England. Morton patented a "secret formula" he called Morton's Letheon,[62] which smelled suspiciously of ether, but eventually he went broke trying to monopolize ether anesthesia and he died a pauper. His grave near Boston bears the epitaph:

WILLIAM T. G. MORTON
Inventor and Revealer of Anaesthetic Inhalation
Before Whom, in All Time, Surgery was Agony.
By Whom Pain in Surgery Was Averted and Annulled.
Since Whom Science Has Control of Pain.

Wells, who had engaged in a bitter feud to establish himself as the inventor of ether anesthesia, committed suicide at the age of 33. Crawford Long went on to a successful career as an Atlanta pharmacist, but to his death he remained disappointed that he had not received credit as the first to perform surgery on etherized patients.

Ether and chloroform became obsolete when safer anesthetics such as cyclopropane, ethylene, and nitrous oxide were developed. These are *general anesthetics* that render a patient unconscious by crossing the blood-brain barrier and blocking nervous transmission through the brainstem. Most general anesthetics apparently deaden pain by activating GABA receptors and causing an inflow of Cl^-, which hyperpolarizes neurons and makes them less likely to fire. Diazepam (Valium) also employs this mechanism. *Local anesthetics* such as procaine (Novocain) and tetracaine selectively deaden specific nerves. They decrease the permeability of membranes to Na^+, thereby reducing their ability to produce action potentials.

A sound knowledge of anatomy, control of infection and pain, and development of better tools converged to allow surgeons time to operate more carefully. As a result, surgery became more intellectually challenging and interesting. It attracted a more educated class of practitioner, which put it on the road to becoming the remarkable lifesaving approach that it is today.

[60] *a* = without + *sepsis* = infection
[61] *an* = without + *esthesia* = feeling, sensation
[62] *lethe* = oblivion, forgetfulness

Chapter Review

Review of Key Concepts

Properties and Types of Sensory Receptors (p. 568)

1. Sensory *receptors* range from simple nerve endings to complex sense organs.
2. *Sensory transduction* is the conversion of stimulus energy into a pattern of action potentials.
3. Transduction begins with a *receptor potential* which, if it reaches threshold, triggers the production of action potentials.
4. Receptors transmit four kinds of information about stimuli: *modality, location, intensity,* and *duration.*
5. Receptors can be classified by modality as *chemoreceptors, thermoreceptors, nociceptors, mechanoreceptors,* and *photoreceptors.*
6. Receptors can also be classified by the origins of their stimuli as *interoceptors, proprioceptors,* and *exteroceptors.*
7. *General (somesthetic) senses* have receptors widely distributed over the body and include the senses of touch, pressure, stretch, temperature, and pain. *Special senses* have receptors in the head only and include vision, hearing, equilibrium, taste, and smell.

The General Senses (p. 588)

1. Unencapsulated nerve endings are simple sensory nerve fibers not enclosed in specialized connective tissue; they include *free nerve endings, tactile discs,* and *hair receptors.*
2. Encapsulated nerve endings are nerve fibers enclosed in glial cells or connective tissues that modify their sensitivity. They include *muscle spindles, Golgi tendon organs, tactile corpuscles, Krause end bulbs, lamellated corpuscles,* and *Ruffini corpuscles.*
3. Somesthetic signals from the head travel the trigeminal and other cranial nerves to the brainstem, and those below the head travel up the spinothalamic tract and other pathways. Most signals reach the contralateral primary somesthetic cortex, but proprioceptive signals travel to the cerebellum.
4. Pain is a sensation that occurs when nociceptors detect tissue damage or potentially injurious situations.
5. *Fast pain* is a relatively quick, localized response mediated by myelinated nerve fibers; it may be followed by a less localized *slow pain* mediated by unmyelinated fibers.
6. *Somatic pain* arises from the skin, muscles, and joints, and may be *superficial* or *deep pain. Visceral pain* arises from the viscera; it is less localized and is often associated with nausea.
7. Injured tissues release bradykinin, serotonin, prostaglandins, and other chemicals that stimulate nociceptors.
8. Pain signals travel from the receptor to the cerebral cortex by way of *first-* through *third-order neurons.* Pain from the face travels mainly by way of the trigeminal nerve to the pons, medulla, thalamus, and primary somesthetic cortex in that order. Pain from lower in the body travels by way of spinal nerves to the spinothalamic tract, thalamus, and somesthetic cortex.
9. Pain signals also travel the spinoreticular tract to the reticular formation and from there to the hypothalamus and limbic system,

producing visceral and emotional responses to pain.
10. *Referred pain* is the brain's misidentification of the location of pain resulting from convergence in sensory pathways.
11. *Enkephalins, endorphins,* and *dynorphins* are analgesic neuropeptides *(endogenous opioids)* that reduce the sensation of pain. Pain awareness can also be reduced by the *spinal gating* of pain signals.

The Chemical Senses (p. 592)

1. Taste *(gustation)* results from the action of chemicals on the *taste buds,* which are groups of sensory cells located on some of the *lingual papillae* and in the palate, pharynx, and epiglottis.
2. *Foliate, fungiform,* and *vallate papillae* have taste buds; *filiform papillae* lack taste buds but sense the texture of food.
3. The primary taste sensations are salty, sweet, sour, bitter, and umami. Flavor is a combined effect of these tastes and the texture, aroma, temperature, and appearance of food. Some flavors result from the stimulation of free nerve endings.
4. Some taste chemicals (sugars, alkaloids, and glutamate) bind to surface receptors on the taste cells and activate second messengers in the cell; sodium and acids penetrate into the taste cell and depolarize it.
5. Taste signals travel from the tongue through the facial and glossopharyngeal nerves, and from the palate, pharynx, and epiglottis through the vagus nerve. They travel to the medulla oblongata and then by one route to the hypothalamus and amygdala, and by another route to the thalamus and cerebral cortex.
6. Smell *(olfaction)* results from the action of chemicals on *olfactory cells* in the roof of the nasal cavity.
7. Odor molecules bind to surface receptors on the *olfactory hairs* of the olfactory cells and activate second messengers in the cell.
8. Nerve fibers from the olfactory cells assemble into fascicles that collectively constitute cranial nerve I, pass through foramina of the cribriform plate, and end in the olfactory bulbs beneath the frontal lobes of the cerebrum.
9. Olfactory signals travel the *olfactory tracts* from the bulbs to the temporal lobes, and continue to the hypothalamus and amygdala. The cerebral cortex also sends signals back to the bulbs that moderate one's perception of smell.

Hearing and Equilibrium (p. 597)

1. Sound is generated by vibrating objects. The *amplitude* of the vibration determines the *loudness* of a sound, measured in *decibels (db),* and the *frequency* of vibration determines the *pitch,* measured in *hertz (Hz).*
2. Humans hear best at frequencies of 1,500 to 4,000 Hz, but sensitive ears can hear sounds from 20 Hz to 20,000 Hz. The threshold of hearing is 0 db and the threshold of pain is about 140 db; most conversation is about 60 db.
3. The *outer ear* consists of the *auricle* and *auditory canal.* The *middle ear* consists of the tympanic membrane and an air-filled tympanic cavity containing three bones *(malleus, incus,* and *stapes)* and two muscles *(tensor tympani* and *stapedius).* The inner ear consists of fluid-filled chambers and tubes (the *membranous labyrinth*) including the *vestibule, semicircular ducts,* and *cochlea.*
4. The most important part of the cochlea, the organ of hearing, is the spiral *organ of Corti,* which includes sensory *hair cells.* A row of 3,500 *inner hair cells* generates the signals we hear, and three rows of *outer hair cells* tune the cochlea to enhance its pitch discrimination.
5. Vibrations in the ear move the *basilar membrane* of the cochlea up and down. As the hair cells move up and down, their stereocilia bend against the relatively stationary tectorial membrane above them. This opens K^+ channels at the tip of each stereocilium, and the inflow of K^+ depolarizes the cell. This triggers neurotransmitter release, which initiates a nerve signal.
6. *Loudness* determines the amplitude of basilar membrane vibration and the firing frequency of the associated auditory neurons. *Pitch* determines which regions of the basilar membrane vibrate more than others, and which auditory nerve fibers respond most strongly.
7. The cochlear nerve joins the vestibular nerve to become cranial nerve VIII. Cochlear nerve fibers project to the pons and from there to the inferior colliculi of the midbrain, then the thalamus, and finally the primary auditory cortex of the temporal lobes.
8. *Static equilibrium* is the sense of the orientation of the head; *dynamic equilibrium* is the sense of linear or angular acceleration of the head.
9. The *saccule* and *utricle* are chambers in the vestibule of the inner ear, each with a *macula* containing sensory hair cells. The *macula sacculi* is nearly vertical and the *macula utriculi* is nearly horizontal.
10. The hair cell stereocilia are capped by a weighted gelatinous *otolithic membrane.* When pulled by gravity or linear acceleration of the body, these membranes stimulate the hair cells.
11. Any orientation of the head causes a combination of stimulation to the four maculae, sending signals to the brain that enable it to sense the orientation. Vertical acceleration also stimulates each macula sacculi, and horizontal acceleration stimulates each macula utriculi.
12. Each inner ear also has three *semicircular ducts* with a sensory patch of hair cells, the *crista ampullaris,* in each duct. The stereocilia of these hair cells are embedded in a gelatinous *cupula.*
13. Tilting or rotation of the head moves the ducts relative to the fluid (endolymph) within, causing the fluid to push the cupula and stimulate the hair cells. The brain detects angular acceleration of the head from the combined input from the six ducts.
14. Signals from the utricle, saccule, and semicircular ducts travel the *vestibular nerve,* which joins the cochlear nerve in cranial nerve VIII. Vestibular nerve fibers lead to the pons and cerebellum.

Vision (p. 610)

1. Vision is a response to electromagnetic radiation with wavelengths from about 400 to 750 nm.
2. Accessory structures of the orbit include the eyebrows, eyelids, conjunctiva, lacrimal apparatus, and extrinsic eye muscles.

3. The wall of the eyeball is composed of an outer *fibrous layer* composed of *sclera* and *cornea;* middle *vascular layer* composed of *choroid, ciliary body,* and *iris;* and an *inner layer* composed of the *retina* and beginning of the *optic nerve.*
4. The optical components of the eye admit and bend (refract) light rays and bring images to a focus on the retina. They include the *cornea, aqueous humor, lens,* and *vitreous body.* Most refraction occurs at the air-cornea interface, but the lens adjusts the focus.
5. The neural components of the eye absorb light and encode the stimulus in action potentials transmitted to the brain. They include the *retina* and *optic nerve.* The sharpest vision occurs in a region of retina called the *fovea centralis,* while the *optic disc,* where the optic nerve originates, is a blind spot with no receptor cells.
6. The relaxed *(emmetropic)* eye focuses on objects 6 m or more away. A *near response* is needed to focus on closer objects. This includes convergence of the eyes, constriction of the pupil, and *accommodation* (thickening) of the lens.
7. Light falling on the retina is absorbed by visual pigments in the *outer segments* of the *rod* and *cone* cells. Rods function at low light intensities (producing night, or *scotopic,* vision) but produce monochromatic images with poor resolution. Cones require higher light intensities (producing day, or *photopic,* vision) and produce color images with finer resolution.
8. Light absorption bleaches the *rhodopsin* of rods or the *photopsins* of the cones. In rods (and probably cones), this stops the *dark current* of Na^+ flow into the cell and the release of glutamate from the inner end of the cell.
9. Rods and cones synapse with *bipolar cells,* which respond to changes in glutamate secretion. Bipolar cells, in turn, stimulate *ganglion cells.* Ganglion cells are the first cells in the pathway that generate action potentials; their axons form the optic nerve.
10. The eyes respond to changes in light intensity by *light adaptation* (pupillary constriction and pigment bleaching) and *dark adaptation* (pupillary dilation and pigment regeneration).
11. The *duplicity theory* explains that a single type of receptor cell cannot produce both high light sensitivity (like the rods) and high resolution (like the cones). The neuronal convergence responsible for the sensitivity of rod pathways reduces resolution, while the lack of convergence responsible for the high resolution of cones reduces light sensitivity.
12. Three types of cones—blue, green, and red—have slight differences in their photopsins that result in peak absorption in different regions of the spectrum. This results in the ability to distinguish colors.
13. *Stereoscopic vision (depth perception)* results from each eye viewing an object from a slightly different angle, so its image falls on different areas of the two retinas.
14. Fibers of the optic nerves *hemidecussate* at the *optic chiasm,* so images in the left visual field project from both eyes to the right cerebral hemisphere, and images on the right project to the left hemisphere.
15. Beyond the optic chiasm, most nerve fibers end in the *lateral geniculate nucleus* of the thalamus. Here they synapse with third-order neurons whose fibers form the *optic radiation* leading to the primary visual cortex of the occipital lobe.
16. Some fibers of the optic nerve lead to the superior colliculi and pretectal nuclei of the midbrain. These midbrain nuclei control visual reflexes of the extrinsic eye muscles, pupillary reflexes, and accommodation of the lens in near vision.

Selected Vocabulary

receptor 586
modality 586
projection pathway 586
nociceptor 587
proprioceptor 587
first- to third-order neuron 589
referred pain 590
analgesic 590
gustation 592
taste cell 593
olfaction 594
olfactory cell 595
vestibule 599
cochlea 599
organ of Corti 600
hair cell 601
equilibrium 606
semicircular duct 606
conjunctiva 610
cornea 612
retina 614
optic disc 615
fovea centralis 615
refraction 616
near response 617
rod 619
cone 619
rhodopsin 621
optic chiasm 626

Testing Your Recall

1. Hot and cold stimuli are detected by
   a. free nerve endings.
   b. proprioceptors.
   c. Krause end bulbs.
   d. lamellated corpuscles.
   e. tactile corpuscles.

2. _____ is a neurotransmitter that transmits pain sensations to second-order spinal neurons.
   a. Endorphin
   b. Enkephalin
   c. Substance P
   d. Acetylcholine
   e. Norepinephrine

3. _____ is a neuromodulator that blocks the transmission of pain sensations to second-order spinal neurons.
   a. Endorphin
   b. Enkephalin
   c. Substance P
   d. Acetylcholine
   e. Norepinephrine

4. Taste buds of the vallate papillae are most sensitive to
   a. bitter.
   b. sour.
   c. sweet.
   d. umami.
   e. salty.
5. The higher the frequency of a sound,
   a. the louder it sounds.
   b. the harder it is to hear.
   c. the more it stimulates the distal end of the organ of Corti.
   d. the faster it travels through air.
   e. the higher its pitch.
6. Cochlear hair cells rest on
   a. the tympanic membrane.
   b. the secondary tympanic membrane.
   c. the tectorial membrane.
   d. the vestibular membrane.
   e. the basilar membrane.
7. The acceleration you feel when an elevator begins to rise is sensed by
   a. the anterior semicircular duct.
   b. the organ of Corti.
   c. the crista ampullaris.
   d. the macula sacculi.
   e. the macula utriculi.
8. The color of light is determined by
   a. its velocity.
   b. its amplitude.
   c. its wavelength.
   d. refraction.
   e. how strongly it stimulates the rods.
9. The retina receives its oxygen supply from
   a. the hyaloid artery.
   b. the vitreous body.
   c. the choroid.
   d. the pigment epithelium.
   e. the scleral venous sinus.
10. Which of the following statements about photopic vision is false?
    a. It is mediated by the cones.
    b. It has a low threshold.
    c. It produces fine resolution.
    d. It does not function in starlight.
    e. It does not employ rhodopsin.
11. The most finely detailed vision occurs when an image falls on a pit in the retina called the _____ .
12. The only cells of the retina that generate action potentials are the _____ cells.
13. The retinal dark current results from the flow of _____ into the receptor cells.
14. The gelatinous membranes of the macula sacculi and macula utriculi are weighted by calcium carbonate and protein granules called _____.
15. Three rows of _____ in the cochlea have V-shaped arrays of stereocilia and tune the frequency sensitivity of the cochlea.
16. The _____ is a tiny bone that vibrates in the oval window and thereby transfers sound vibrations to the inner ear.
17. The _____ of the midbrain receive auditory input and trigger the head-turning auditory reflex.
18. The apical stereocilia of a gustatory cell are called _____ .
19. Olfactory neurons synapse with mitral cells and tufted cells in the _____ , which lies inferior to the frontal lobe.
20. In the phenomenon of _____ , pain from the viscera is perceived as coming from an area of the skin.

Answers in Appendix B

True or False

Determine which five of the following statements are false, and briefly explain why.

1. The sensory (afferent) nerve fibers for touch end in the thalamus.
2. Things we touch with the left hand are perceived only in the right cerebral hemisphere.
3. Things we see with the left eye are perceived only in the right cerebral hemisphere.
4. Some chemoreceptors are interoceptors and some are exteroceptors.
5. The vitreous body occupies the posterior chamber of the eye.
6. Descending analgesic fibers prevent pain signals from reaching the spinal cord.
7. Cranial nerve VIII carries signals for both hearing and balance.
8. The tympanic cavity is filled with air, but the membranous labyrinth is filled with liquid.
9. Rods and cones release their neurotransmitter in the dark, not in the light.
10. All of the extrinsic muscles of the eye are controlled by the oculomotor nerve.

Answers in Appendix B

Testing Your Comprehension

1. The principle of neuronal convergence is explained on page 472. Discuss its relevance to referred pain and scotopic vision.
2. What type of cutaneous receptor enables you to feel an insect crawling through your hair? What type enables you to palpate a patient's pulse? What type enables a blind person to read braille?
3. Contraction of a muscle usually puts more tension on a structure, but

contraction of the ciliary muscle puts less tension on the lens. Explain how.

4. Janet has terminal ovarian cancer and is in severe pelvic pain that has not yielded to any other treatment. A neurosurgeon performs an *anterolateral cordotomy,* cutting across the anterolateral region of her lumbar spinal cord. Explain the rationale of this treatment and its possible side effects.
5. What would be the benefit of a drug that blocks the receptors for substance P?

Answers at the Online Learning Center

Answers to Figure Legend Questions

16.1 Two touches are felt separately if they straddle the boundary between two separate receptive fields.
16.8 The lower margin of the violet zone ("all sound") would be higher in that range.
16.14 It would oppose the inward movement of the tympanic membrane, and thus reduce the amount of vibration transferred to the inner ear.
16.38 Approximately 68:20:0
16.41 It would cause blindness in the left half of the visual field. It would not affect the visual reflexes.

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