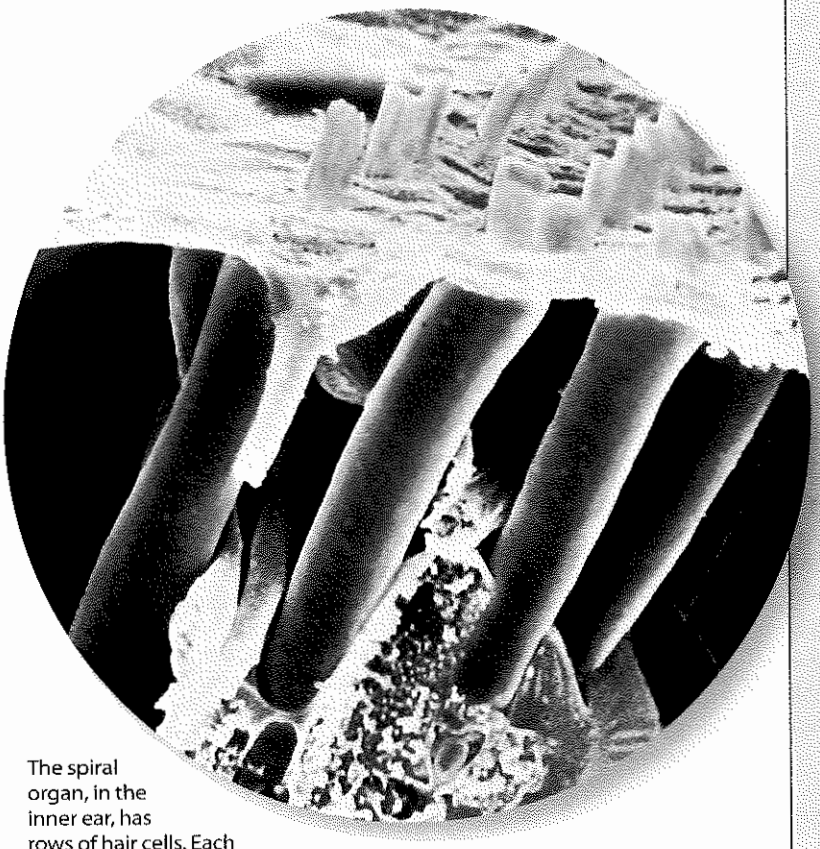
12 Nervous System III
Senses



The spiral organ, in the inner ear, has rows of hair cells. Each row bears up to 100 hairs, which translate sound into neural messages that travel to the brain. In this falsely colored micrograph, the cells appear red and the hairs yellow (2,700×).

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Module 7: Nervous System

Learning Outcomes

After you have studied this chapter, you should be able to:

12.1 Introduction

1 Differentiate between general senses and special senses. (p. 444)

12.2 Receptors, Sensation, and Perception

2 Name the five types of receptors and state the function of each. (p. 445)
3 Explain how receptors trigger sensory impulses. (p. 445)
4 Explain sensation and adaptation. (p. 445)

12.3 General Senses

5 Describe the differences among receptors associated with the senses of touch, pressure, temperature, and pain. (p. 446)
6 Describe how the sensation of pain is produced. (p. 447)
7 Explain the importance of stretch receptors in muscles and tendons. (p. 450)

12.4 Special Senses

8 Explain the relationship between the senses of smell and taste. (p. 452)
9 Describe how the sensations of smell and taste are produced and interpreted. (p. 453)
10 Name the parts of the ear and explain the function of each part. (p. 456)
11 Distinguish between static and dynamic equilibrium. (p. 464)
12 Describe the roles of the accessory organs to the eye. (p. 468)
13 Name the parts of the eye and explain the function of each part. (p. 471)
14 Explain how the eye refracts light. (p. 476)
15 Explain how the brain perceives depth and distance. (p. 480)
16 Describe the visual nerve pathways. (p. 481)

12.5 Life-Span Changes

17 Describe aging-associated changes that diminish the senses. (p. 481)

LEARN PRACTICE ASSESS

Understanding Words

aud-, to hear: *aud*itory—pertaining to hearing.
choroid, skinlike: *choroid* coat—middle, vascular layer of the eye.
cochlea, snail: *cochlea*—coiled tube in the inner ear.
corn-, horn: *corn*ea—transparent outer layer in the anterior portion of the eye.
iris, rainbow: *iris*—colored, muscular part of the eye.
labyrinth, maze: *labyrinth*—complex system of connecting chambers and tubes of the inner ear.
lacri-, tears: *lacri*mal gland—tear gland.
lut-, yellow: macula *lut*ea—yellowish spot on the retina.
macula, spot: *macula* lutea—yellowish spot on the retina.
malle-, hammer: *malle*us—one of the three bones in the middle ear.
ocul-, eye: orbicularis *oculi*—muscle associated with the eyelid.
olfact-, to smell: *olfact*ory—pertaining to the sense of smell.
palpebra, eyelid: levator *palpebrae* superioris—muscle associated with the eyelid.
photo-, light: *photo*receptors—specialized structures in the eye responsive to light.
scler-, hard: *sclera*—tough, outer protective layer of the eye.
therm-, heat: *therm*oreceptor—receptor sensitive to changes in temperature.
tympan-, drum: *tympan*ic membrane—eardrum.
vitre-, glass: *vitre*ous humor—clear, jellylike substance in the eye.

The World Without Color

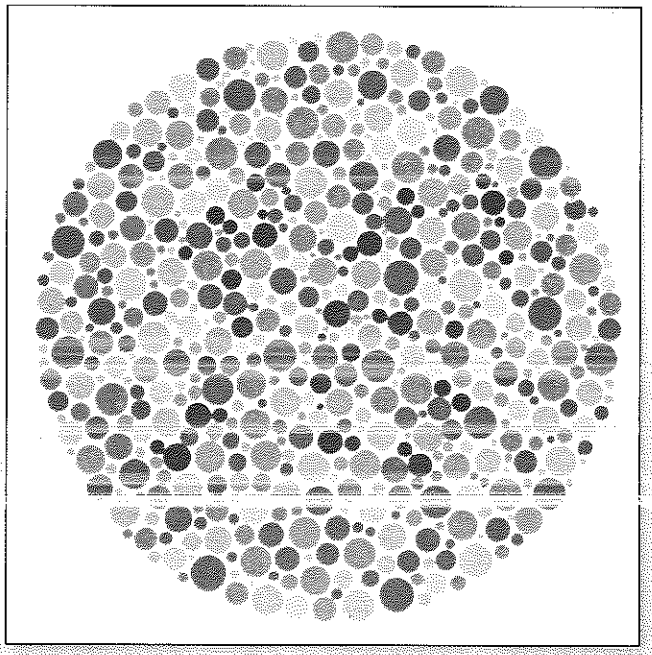
John Dalton, a famous English chemist, saw things differently than most people. In a 1794 lecture, he described his visual world. Sealing wax that appeared red to other people was as green as a leaf to Dalton and his brother. Pink wildflowers were blue, and Dalton perceived the cranesbill plant as "sky blue" in daylight, but "very near yellow, but with a tincture of red" in candlelight. He concluded: "that part of the image which others call red, appears to me little more than a shade, or defect of light." The Dalton brothers, like 7% of males and 0.4% of females today, had the inherited trait of colorblindness.

Dalton was very curious about the cause of his colorblindness, so he made arrangements with his personal physician, Joseph Ransome, to dissect his eyes after he died. During that dissection, Ransome snipped off the back of one eye and removed the retina, where the cone cells that provide color vision are nestled among the more abundant rod cells that impart black-and-white vision. Because Ransome could see red and green normally when he peered through the back of Dalton's eyeball, he concluded that it was not an abnormal filter in front of the eye that altered color vision.

Fortunately, Ransome stored the eyes in dry air, where they remained relatively undamaged. In 1994, Dalton's eyes underwent DNA analysis at London's Institute of Ophthalmology. The research showed that Dalton's remaining retina lacked one of three types of pigments, called photopigments, that enable cone cells to capture certain incoming wavelengths of light.

Although people have studied colorblindness for centuries, we are still learning more about it. Recently, researchers investigated why colorblind men lacking cones that capture green light are affected to different degrees. They discovered that colorblind men who can discern a few shades of green have red cone cells that can detect some wavelengths of light that fall within the green region of the spectrum. Color vision may be more complex than we had thought.

People who are colorblind must function in a multicolored world. To help them overcome the disadvantage of not seeing important color differences, researchers have developed computer algorithms that convert colored video pictures into shades those with colorblindness can see. ■



This circle of dots is a test to determine whether someone is colorblind. Affected individuals cannot see a different color in certain of the dots in such a drawing. As a result, their brains cannot perceive the embedded pattern that forms the number 16 that others can see. The above has been reproduced from *Ishihara's Tests for Colour Blindness* published by Kanehara & Co. Ltd. Tokyo, Japan, but tests for colorblindness cannot be conducted with this reproduction. For accurate testing, the original plates should be used.

12.1 INTRODUCTION

Our senses not only make our lives meaningful, connecting us to the sights, sounds, smells, tastes, and textures of the outside world, but also help our bodies maintain homeostasis by providing information about what is happening on the inside. Sensory receptors are the portals that link our nervous systems to all of these events. The **general senses** are those with receptors widely distributed throughout the body, including the skin, various organs, and joints. The **special senses** have more specialized receptors and are confined to structures in the head, such as the eyes and ears.

All senses work in basically the same way. Sensory receptors are specialized cells (receptor cells) or multicellular structures that collect information from the environment. In some cases, such as smell and taste, the receptor cells have specific molecules on the cell membrane (membrane receptors). Stimulated receptor cells in turn stimulate neurons to conduct impulses along sensory fibers to the brain. There the cerebral cortex forms a perception, which is a person's particular view of the stimulus. Table 12.1 outlines the pathways from sensation to perception that describe an apple using the special senses: smell, taste, sight, and hearing.

Chapter 11 (p. 393) uses the terms *axon* and *nerve fiber* synonymously. Also recall from chapter 11 (p. 425) that unipolar neurons, which include most sensory neurons, are unusual in that the portion of the neuron associated with the dendrites, called a peripheral process, functions like an axon (see fig. 10.7, p. 368). Because of this, and for simplicity, we call the neuron processes that bring sensory information into the CNS "sensory fibers" or "afferent fibers," no matter what type of neuron is involved.

12.2 RECEPTORS, SENSATION, AND PERCEPTION

Recall that all action potentials are the same (all-or-none). The body's ability to respond to different sensory events therefore depends on receptors that respond to specific stimuli. Sensory receptors are diverse but share certain features. Each type of receptor is particularly sensitive to a distinct type

TABLE **12.1** | Information Flow from the Environment Through the Nervous System

Information Flow	Smell	Taste	Sight	Hearing
Sensory receptors	Olfactory receptor cells	Taste bud receptor cells	Rods and cones in retina	Hair cells in cochlea
↓	↓	↓	↓	↓
Impulse in sensory fibers	Olfactory nerve fibers	Sensory fibers in various cranial nerves	Optic nerve fibers	Auditory nerve fibers
↓	↓	↓	↓	↓
Impulse reaches CNS	Cerebral cortex	Cerebral cortex	Midbrain and cerebral cortex	Midbrain and cerebral cortex
↓	↓	↓	↓	↓
Sensation (new experience, recalled memory)	A pleasant smell	A sweet taste	A small, round, red object	A crunching sound
↓	↓	↓	↓	↓
Perception	The smell of an apple	The taste of an apple	The sight of an apple	The sound of biting into an apple

of environmental change and is much less sensitive to other forms of stimulation. The raw form in which these receptors send information to the brain is called **sensation.** The way our brains interpret this information is called **perception.**

Receptor Types

Five types of sensory receptors are recognized, based on their sensitivities to specific stimuli:

1. **Chemoreceptors** (ke″mo-re-sep′torz) respond to changes in the concentration of chemicals. Receptors associated with the senses of smell and taste are of this type. Chemoreceptors in internal organs detect changes in the blood concentrations of oxygen, hydrogen ions, glucose, and other chemicals.
2. **Pain receptors,** also called nociceptors (no″se-sep′torz), respond to tissue damage. Triggering stimuli include exposure to excess mechanical, electrical, thermal, or chemical energy.
3. **Thermoreceptors** (ther″mo-re-sep′torz) sense temperature change.
4. **Mechanoreceptors** (mek″ah-no re-sep′torz) are of several types and sense mechanical forces by detecting changes that deform the receptors. They include a number of receptors in the skin that respond to physical contact, and several receptors in the ear that provide information about balance and vibrations from sound. **Proprioceptors** (pro″pre-o-sep′torz) sense changes in the tensions of muscles and tendons; **baroreceptors** (bar″o-re-sep′torz), also called pressoreceptors, in certain blood vessels detect changes in blood pressure; and **stretch receptors** in the lungs sense degree of inflation.
5. **Photoreceptors** (fo″to-re-sep′torz) in the eyes respond to light energy of sufficient intensity.

Sensory Impulses

Sensory receptors can be ends of neurons or other types of cells that are near neuron extensions. In either case, stimulation locally changes their membrane potentials (receptor potentials), generating a graded electric current that reflects the intensity of stimulation (see chapter 10, p. 375).

If a receptor is a neuron and the change in membrane potential reaches threshold, an action potential is generated and is propagated along the afferent fiber. However, if the receptor is another type of cell, its receptor potential must be transferred to a neuron to trigger an action potential. Peripheral nerves conduct sensory impulses to the central nervous system (CNS), where they are analyzed and interpreted in the brain.

Sensation and Perception

A sensation occurs when sensory neurons reach threshold and the resulting action potentials cause the brain to become aware of that sensory event. A perception occurs when the brain interprets those sensory impulses. Thus, pain is a sensation, but realizing that you have just stepped on a tack is a perception. At the same time that a sensation forms, the cerebral cortex interprets it to seem to come from the receptors being stimulated. This process, which is closely related to perception, is called **projection,** because the brain projects the sensation back to its apparent source. Projection allows a person to pinpoint the region of stimulation. In this way, we perceive that the eyes see an apple, the nose smells it, and the ears hear the teeth crunch into it.

Because all of the impulses conducted on sensory fibers into the CNS are alike, the resulting sensation depends on which region of the cerebral cortex receives the impulse. For example, impulses reaching one region are always interpreted as sounds, and those reaching another are always sensed as touch. (Some receptors, such as those that measure oxygen levels in the blood, do not trigger sensations.)

RECONNECT
To Chapter 11, Functions of the Cerebrum, pages 407–408.

Sensory receptors are specialized to respond to specific stimuli, but they may respond to other stimuli that

are strong enough. In either case the sensations are the same. Pain receptors, for example, can be stimulated by heat, cold, or pressure, but the sensation is always the same because, in each case, the same part of the brain interprets the resulting impulses as pain. Similarly, stimuli other than light, such as a sharp blow to the head, may trigger impulses in visual receptors. When this happens, the person may "see stars," even though no light is entering the eye, because any impulses reaching the visual cortex are interpreted as light. Receptors respond only to specific stimuli, and the brain typically creates the correct sensation for that particular stimulus.

Sensory Adaptation

The brain must prioritize the sensory input it receives, or incoming unimportant information would be overwhelming. For example, until this sentence prompts you to think about it, you are probably unaware of the pressure of your clothing against your skin, or the background noise in the room. This ability to ignore unimportant stimuli is called **sensory adaptation** (sen'so-re ad"ap-ta'shun). It may reflect a decreased response to a particular stimulus from the receptors (peripheral adaptation) or along the CNS pathways leading to the sensory regions of the cerebral cortex (central adaptation). Once adaptation to a particular stimulus occurs, a sensation will happen only if the strength of the stimulus changes.

PRACTICE

1. Distinguish between general and special senses.
2. List the five general types of sensory receptors.
3. What do all types of receptors have in common?
4. Explain how a sensation is different from a perception.
5. What is sensory adaptation?

12.3 GENERAL SENSES

General senses are those whose sensory receptors are widespread, associated with the skin, muscles, joints, and viscera. These senses can be divided into three groups:

1. **Exteroreceptive senses** are associated with changes at the body surface. They include the senses of touch, pressure, temperature, and pain.
2. **Visceroreceptive** (interoceptive) **senses** are associated with changes in viscera (blood pressure stretching blood vessels, an ingested meal stimulating pH receptors in the small intestine, and so on).
3. **Proprioceptive senses** are associated with changes in muscles and tendons and in body position.

Touch and Pressure Senses

The senses of touch and pressure derive from three types of receptors (fig. 12.1). As a group, these receptors sense mechanical forces that deform or displace tissues. The touch and pressure receptors include the following:

1. **Free nerve endings,** the simplest receptors, are common in epithelial tissues, where they lie between epithelial cells. They are responsible for the sensation of itching (fig. 12.1*a*).
2. **Tactile (Meissner's) corpuscles** are small, oval masses of flattened connective tissue cells in connective tissue sheaths. Two or more sensory fibers branch into each corpuscle and end within it as tiny knobs.

   Tactile corpuscles are abundant in hairless portions of skin, such as the lips, fingertips, palms, soles, nipples, and external genital organs. They provide fine touch, such as distinguishing two points on the skin where an object touches, to judge its texture (fig. 12.1*b*).
3. **Lamellated (Pacinian) corpuscles** are relatively large, ellipsoidal structures composed of connective tissue fibers and cells. They are common in the deeper dermal tissues of the hands, feet, penis, clitoris, urethra, and breasts and also in tendons of muscles and ligaments of joints (fig. 12.1*b*). Heavier pressure and stretch stimulate lamellated corpuscles. They also detect vibrations in tissues.

Temperature Senses

Temperature receptors (thermoreceptors) include two groups of free nerve endings in the skin. Those that respond to warmer temperatures are *warm receptors,* and those that respond to colder temperatures are *cold receptors.*

The warm receptors are most sensitive to temperatures above 25°C (77°F) and become unresponsive at temperatures above 45°C (113°F). Approaching 45°C also triggers pain receptors, producing a burning sensation.

Cold receptors are most sensitive to temperatures between 10°C (50°F) and 20°C (68°F). Temperature dropping below 10°C stimulates pain receptors, producing a freezing sensation.

At intermediate temperatures, the brain interprets sensory input from different combinations of warm and cold receptors as an intermediate temperature sensation. Both types of receptors rapidly adapt, so within about a minute of continuous stimulation, the sensation of warm or cold begins to fade. This is why we quickly become comfortable after jumping into a cold swimming pool or submerging into a steaming hot tub.

Sense of Pain

Pain receptors (nociceptors) consist of free nerve endings. These receptors are widely distributed throughout the skin and internal tissues, except in the nervous tissue of the brain, which lacks pain receptors. Pain receptors protect in that they are stimulated when tissues are damaged and may prevent further damage. Most pain sensations are perceived as unpleasant, signaling that action be taken to remove the source of the stimulation.

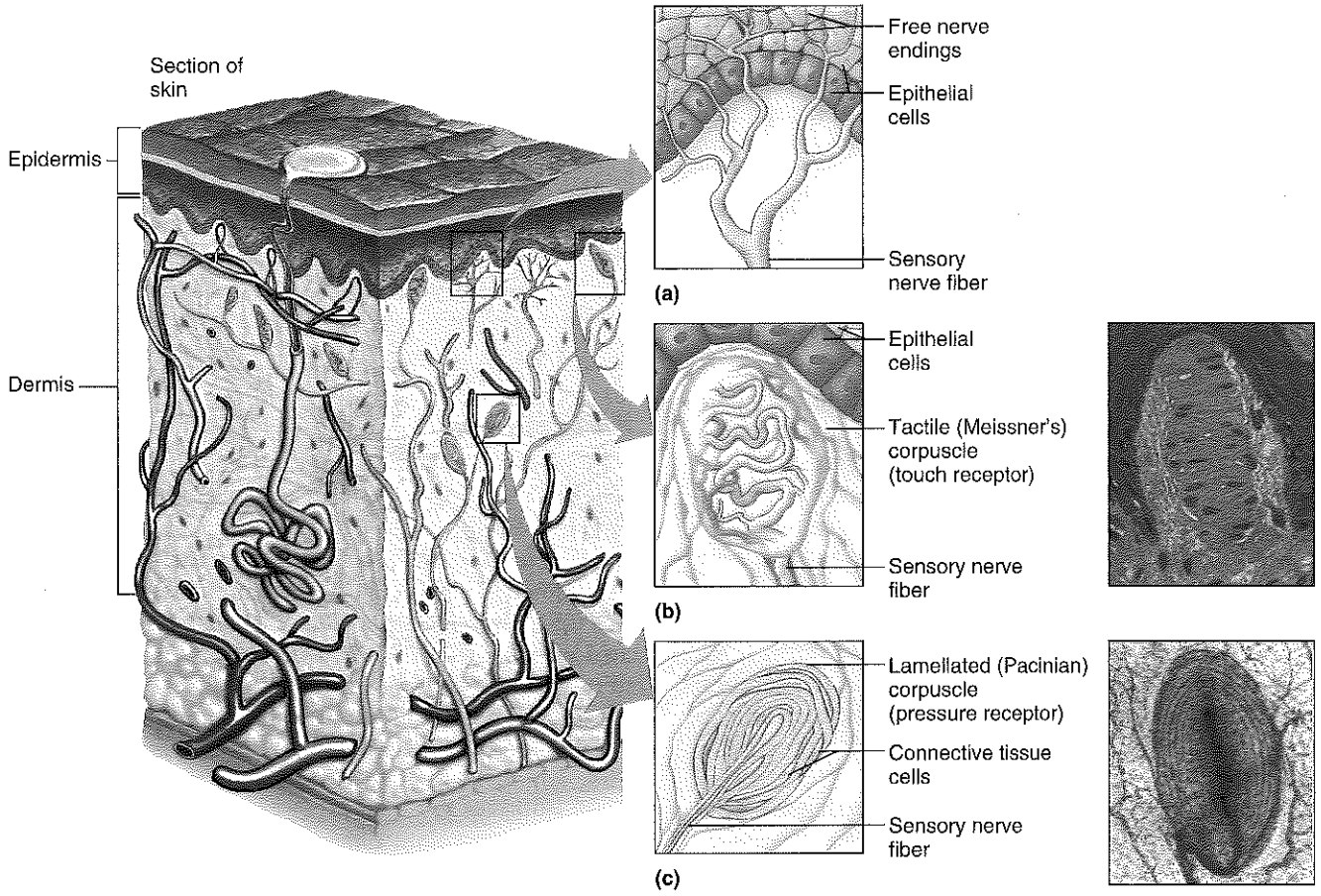


FIGURE 12.1 Touch and pressure receptors include (*a*) free ends of sensory nerve fibers, (*b*) a tactile corpuscle (with 225× micrograph), and (*c*) a lamellated corpuscle (with 50× micrograph).

More than one type of change stimulates most pain receptors. However, some pain receptors are most sensitive to mechanical damage, while others are particularly sensitive to temperature extremes. Some pain receptors are most responsive to chemicals, such as hydrogen ions, potassium ions, or specific breakdown products of proteins, histamine, and acetylcholine. A deficiency of blood flow (ischemia) and the resulting deficiency of oxygen (hypoxia) in a tissue also triggers pain sensation. For example, the sharp pain of a muscle cramp results from interruption of blood flow as the sustained contraction squeezes capillaries, as well as from the stimulation of mechanoreceptors. Also, when blood flow is interrupted, pain-stimulating chemicals accumulate. Increasing blood flow through the sore tissue may relieve the resulting pain, and this is why heat is sometimes applied to reduce muscle soreness. The heat dilates blood vessels and thus promotes blood flow, which helps reduce the concentration of the pain-stimulating substances. In some conditions, accumulating chemicals lower the thresholds of pain receptors, making inflamed tissues more sensitive to heat or pressure.

Pain receptors adapt very little, if at all. Once a pain receptor is activated, even by a single stimulus, it may continue to send impulses into the CNS for some time.

Based on genetic differences, people vary in their sensitivity to pain. One mutation in a gene that encodes a sodium channel protein makes a person feel more severe pain because the channel stays open longer. These people are overrepresented among those with conditions such as osteoarthritis, sciatica, and back pain. They may be more likely to seek care than people with the same conditions who experience less pain. A different and very rare mutation in the same gene has the opposite effect: hereditary sensory and autonomic neuropathy, in which people cannot feel pain, and inadvertently injure themselves. Researchers who are developing new painkilling drugs are studying the ion channels that play a role in sending pain signals.

Visceral Pain

As a rule, pain receptors are the only receptors in viscera whose stimulation produces sensations. Pain receptors in these organs respond differently to stimulation than those associated with surface tissues. For example, localized damage to intestinal tissue during surgical procedures may not elicit any pain sensations, even in a conscious person. However, when visceral tissues are subjected to more widespread stimulation, such as when intestinal tissues are

stretched or when the smooth muscles in the intestinal walls undergo spasms, a strong pain sensation may follow. Once again, the resulting pain results from stimulation of mechanoreceptors and from decreased blood flow accompanied by lower tissue oxygen levels and accumulation of pain-causing chemicals stimulating chemoreceptors.

Visceral pain may feel as if it is coming from some part of the body other than the part being stimulated, in a phenomenon called **referred pain.** For example, pain originating in the heart may be referred to the left shoulder or the medial surface of the left upper limb. Pain from the lower esophagus, stomach, or small intestine may seem to be coming from the upper central (epigastric) region of the abdomen. Pain from the urogenital tract may be referred to the lower central (hypogastric) region of the abdomen or to the sides between the ribs and the hip (fig. 12.2).

Referred pain may derive from *common nerve pathways* that sensory impulses coming both from skin areas and from internal organs use. Pain impulses from the heart seem to be conducted over the same nerve pathways as those from the skin of the left shoulder and the inside of the left upper limb, as shown in figure 12.3. During a heart attack, the cerebral cortex may incorrectly interpret the source of the impulses as the shoulder and the medial surface of the left upper limb, rather than the heart.

Pain originating in the parietal layers of thoracic and abdominal membranes—parietal pleura, parietal pericardium, or parietal peritoneum—is usually not referred. Instead, such pain is felt directly over the area being stimulated.

> Neuropathic pain is an overreaction to a stimulus that would ordinarily cause pain, or a pain response to a normally innocuous stimulus. Reflex sympathetic dystrophy is a form of neuropathic pain that causes an intense burning sensation in a hand or foot, even if the extremity is paralyzed or has been amputated. During the Civil War, it was called "causalgia." Union Army Surgeon S. Weir Mitchell described causalgia as "the most terrible of all tortures."

Pain Pathways

The axons (fibers) that conduct impulses away from pain receptors are of two main types: acute pain fibers and chronic pain fibers.

The *acute pain fibers* (also known as A-delta fibers) are myelinated. They conduct nerve impulses rapidly, at velocities up to 30 meters per second. These impulses are associated with the sensation of sharp pain, which typically seems to originate in a local area of skin. This type of pain seldom continues after the pain-producing stimulus stops.

The *chronic pain fibers* (C fibers) are unmyelinated. They conduct impulses more slowly than acute pain fibers, at velocities up to 2 meters per second. These impulses cause the dull, aching pain sensation that may be widespread and

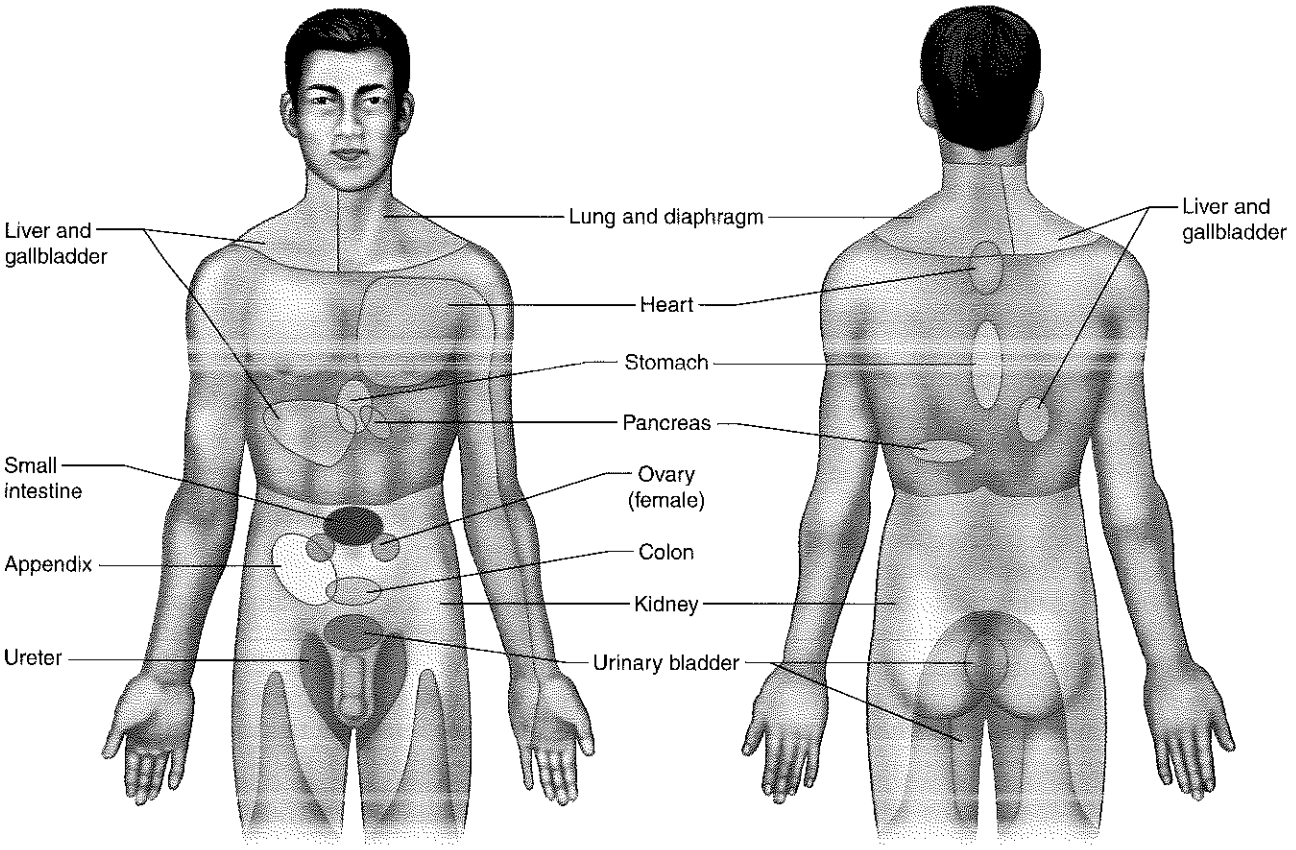


FIGURE 12.2 Surface regions to which visceral pain may be referred.

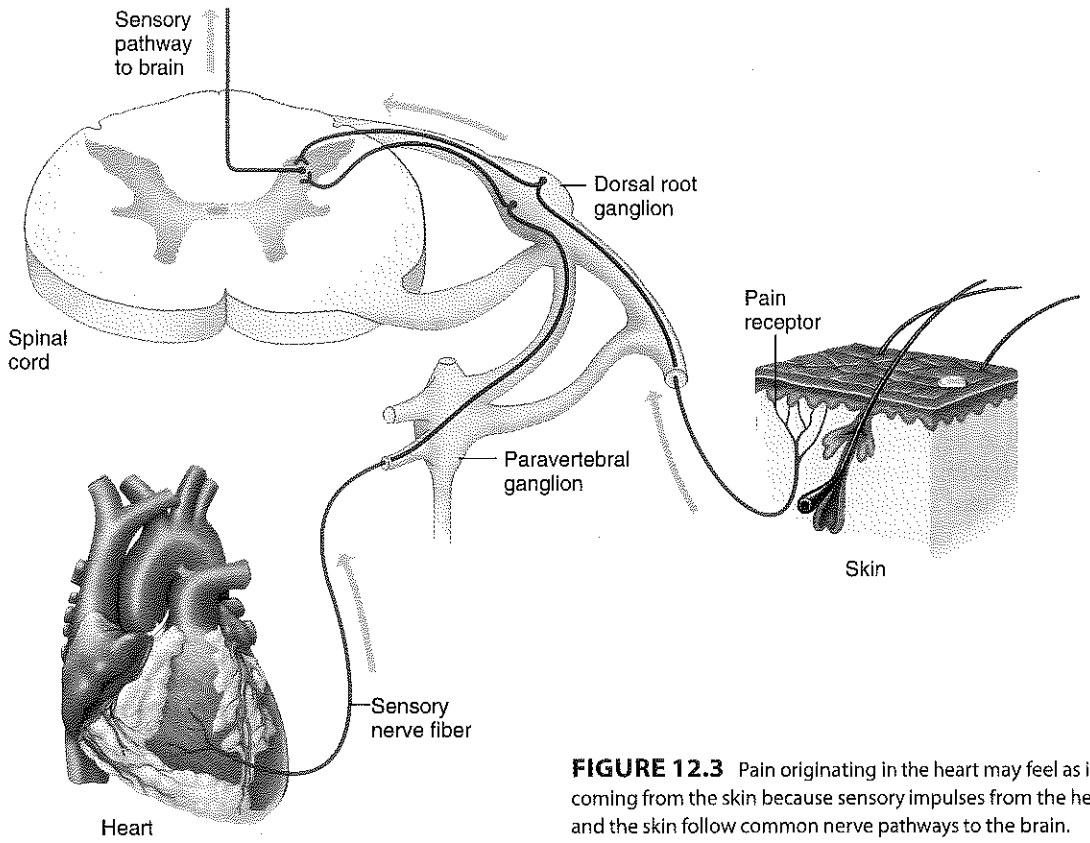


FIGURE 12.3 Pain originating in the heart may feel as if it is coming from the skin because sensory impulses from the heart and the skin follow common nerve pathways to the brain.

difficult to pinpoint. Such pain may continue for some time after the original stimulus ceases. Although acute pain is usually sensed as coming from the surface, chronic pain is felt in deeper tissues as well as in the skin. Visceral pain impulses are usually carried on C fibers.

Usually, an event that stimulates pain receptors triggers impulses on both types of pain fibers. This causes a dual sensation—a sharp, pricking pain, then a dull, aching one. The aching pain is usually more intense and may worsen over time. Chronic pain that resists relief and control can be debilitating.

Pain impulses that originate from tissues of the head reach the brain on sensory fibers of the fifth, seventh, ninth, and tenth cranial nerves (see chapter 11, pp. 420–423). All other pain impulses travel on sensory fibers of spinal nerves, and they pass into the spinal cord by way of the dorsal roots of these spinal nerves.

Upon reaching the spinal cord, pain impulses enter the gray matter of the posterior horn, where they are processed. The fast-conducting fibers synapse with long nerve fibers that cross over to the opposite side of the spinal cord in the anterior and lateral spinothalamic tracts (anterolateral system). The pathway of the impulses carried on the slow-conducting fibers involves one or more interneurons before reaching the long fibers that cross over and ascend to the brain.

In the brain, most of the pain fibers terminate in the reticular formation (see chapter 11, p. 415), and from there impulses are conducted on fibers of still other neurons to the thalamus, hypothalamus, and cerebral cortex. Fibers of the spinothalamic tracts bring pain and temperature information directly to the thalamus.

Regulation of Pain Impulses

Awareness of pain occurs when pain impulses reach the level of the thalamus—that is, even before they reach the cerebral cortex. However, the cerebral cortex must judge the intensity of pain and locate its source, and it is also responsible for emotional and motor responses to pain.

Still other parts of the brain, including areas of gray matter in the midbrain, pons, and medulla oblongata, regulate the flow of pain impulses from the spinal cord (see chapter 11, pp. 413–415). Impulses from special neurons in these areas descend in the lateral funiculus to various levels within the spinal cord. The impulses stimulate the ends of certain nerve fibers to release biochemicals that can block pain signals by inhibiting presynaptic nerve fibers in the posterior horn of the spinal cord.

Among the inhibiting substances released in the posterior horn are neuropeptides called *enkephalins* and the amine *serotonin* (see chapter 10, pp. 380–381). Enkephalins can suppress both acute and chronic pain impulses. Therefore, they can relieve strong pain sensations, much as morphine and other opiate drugs do. In fact, enkephalins were discovered because they bind to the same receptors on neuron membranes as does morphine. Serotonin stimulates other neurons to release enkephalins.

Cannabinoids are chemicals in the plant *Cannibus sativa*, the source of marijuana, that may relieve pain. Neurons in areas of the brain, brainstem, and peripheral nervous system have receptors for cannabinoids that bind a type of neurotransmitter made in the body called anandamide. A synthetic version of the compound in marijuana responsible for most of marijuana's effects (delta-9-tetrahydrocannabinol) is used to treat nausea and vomiting in people receiving cancer chemotherapy and to boost appetite in people who have AIDS.

The *endorphins* are another group of neuropeptides with pain-suppressing, morphinelike actions. They are found in the pituitary gland and in regions of the nervous system, such as the hypothalamus, that relay pain information. Enkephalins and endorphins are released in response to extreme pain, providing natural pain control. Clinical Application 12.1 discusses treatments for severe pain.

PRACTICE

6 Describe three types of touch and pressure receptors.

7 Describe thermoreceptors.

8 What types of stimuli excite pain receptors?

9 What is referred pain?

10 Explain how neuropeptides control pain.

Proprioception

Proprioceptors are mechanoreceptors that send information to the CNS about body position and the length and tension of skeletal muscles. Recall that lamellated corpuscles function as pressure receptors in joints. The other main proprioceptors are stretch receptors: muscle spindles and Golgi tendon organs. Their stimulation does not produce sensations.

Muscle spindles are in skeletal muscles near their junctions with tendons. Each spindle consists of several small, modified skeletal muscle fibers (intrafusal fibers) enclosed in a connective tissue sheath. Near its center, each intrafusal fiber has a specialized nonstriated region with the end of a sensory nerve fiber wrapped around it (fig. 12.4*a*).

The striated portions of the intrafusal fiber contract to keep the spindle taut at different muscle lengths. Thus, if the whole muscle is stretched, the muscle spindle is also stretched, triggering sensory impulses on its nerve fiber. These sensory fibers synapse in the spinal cord with lower motor neurons leading back to the same muscle. In this way, stretch of the muscle spindle triggers impulses that contract the skeletal muscle of which it is a part. This action, called a **stretch reflex,** opposes the lengthening of the muscle and helps maintain the desired position of a limb despite gravitational or other forces tending to move it (see chapter 11, pp. 416–417).

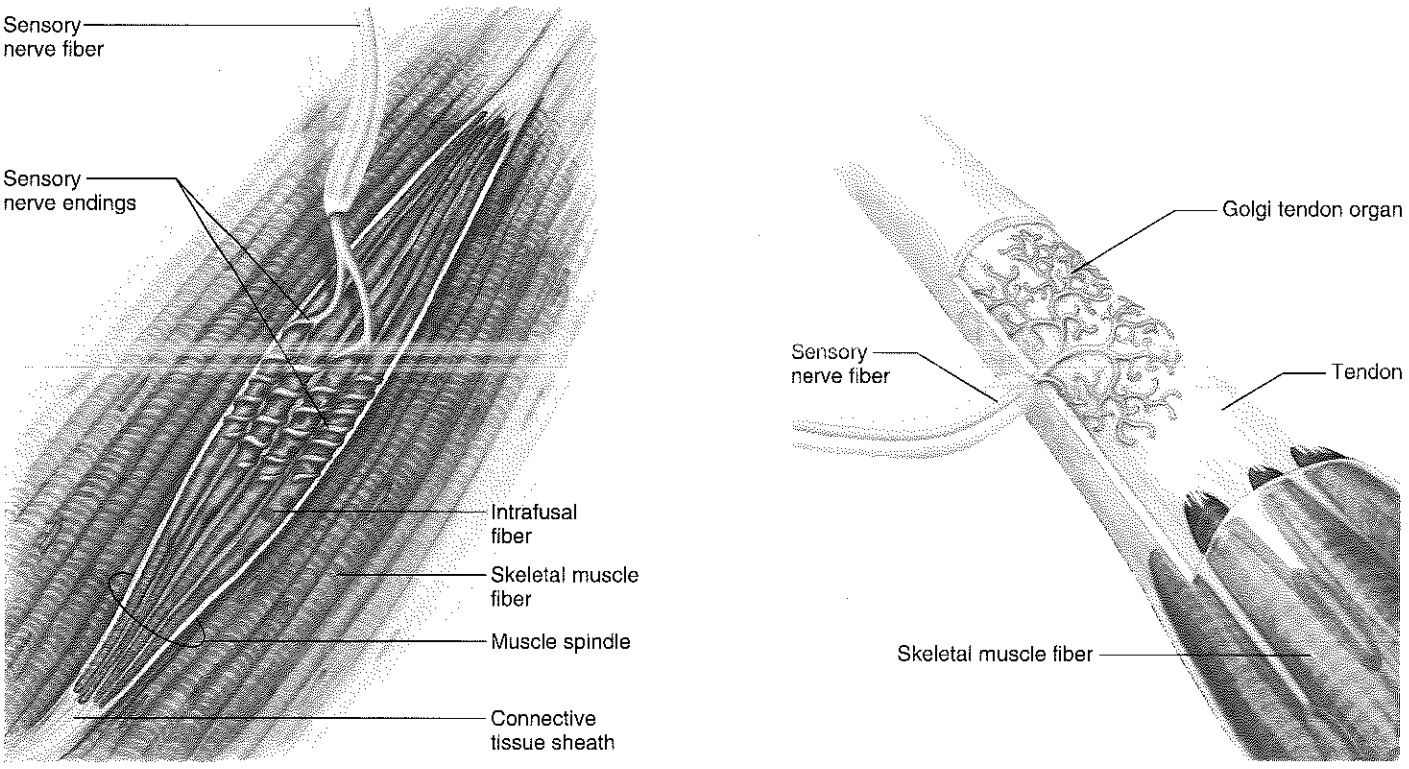
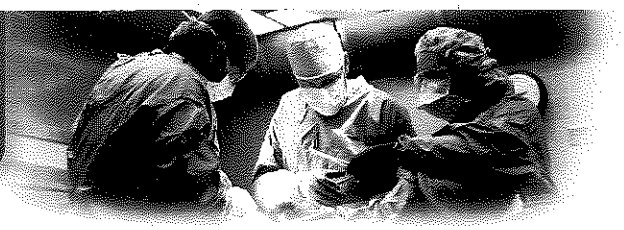


FIGURE 12.4 Stretch receptors maintain posture. (*a*) Increased muscle length stimulates muscle spindles, which stimulate muscle contraction. (*b*) Golgi tendon organs occupy tendons, where they inhibit muscle contraction.

12.1 CLINICAL APPLICATION



Treating Pain

Too many people are in pain. Several studies estimate that at any given time, one in four individuals worldwide is in moderate to severe chronic pain. A quarter of them may be undertreated.

Most pain remedies are nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin, ibuprofen, and COX-2 inhibitors (fig. 12A), or opiates (fig. 12B). The top-selling painkiller is acetaminophen, which is sold under the brand name Tylenol. It has an interesting history.

Acetaminophen was first described in the chemical literature in 1878. In 1886, its analgesic and fever-lowering effects were noted, by accident, but not recognized. Two doctors in France were treating a patient with intestinal parasites with the chemical naphthalene. One day a pharmacist gave them the compound acetanilide in error. Surprisingly, the patient's fever dropped and pain faded. In 1899 a chemist discovered that acetanilide is metabolized to acetaminophen. By 1909 the compound was being produced as a drug, used first in England. It became available by prescription in the United States in 1953 and over the counter in 1960. Acetaminophen does not have the side effects that NSAIDs do, such as irritating the gastrointestinal lining and causing bleeding.

In 2004 a new painkiller became available. Ziconotide is a synthetic version of a peptide that the marine cone snail *Conus magus* releases to paralyze its fish prey (fig. 12C). When researchers noticed that the natural peptide binds to a type of calcium channel protein on spinal cord neurons that receive pain impulses, the effort began to turn the snail's weapon into a pain reliever. Ziconotide is delivered by catheter into the cerebrospinal fluid and is prescribed to relieve intractable chronic pain.

The types of patients in greatest need of pain relief are people with cancer or chronic pain syndromes. Cancer patients take NSAIDs, weak narcotics such as hydrocodone, strong narcotics such as morphine, and opiates delivered directly to the spinal cord via an implanted reservoir. Narcotics are much more likely to be addicting when they are abused to induce euphoria than when they are taken to relieve severe pain. Patients may use devices to control the delivery of pain medications. Anti-anxiety medications can ease the perception of pain.

FIGURE 12A Painkillers come from nature. Aspirin derives from bark of the willow tree.

FIGURE 12B Poppies are the source of opiate drugs.

Chronic pain is of three types: lower back pain, migraine, and myofascial syndrome (inflammation of muscles and their fascia). Treatment approaches include NSAIDs, stretching exercises, injection of local anesthetic drugs into cramping muscles, and antidepressants to raise serotonin levels in the CNS. Chronic pain may also be treated with electrodes implanted near the spinal cord; transcutaneous electrical nerve stimulation (TENS), which also places electrodes on pain-conducting nerves; and an invasive nerve block, which interrupts a pain signal by freezing or by introducing an anesthetic drug. ■

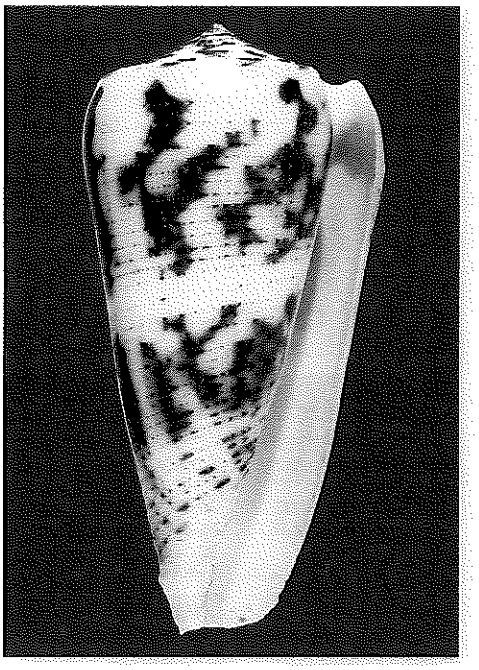


FIGURE 12C A newer analgesic for extreme chronic pain is based on a peptide from the marine cone snail *Conus magus*.

Golgi tendon organs are in tendons close to their attachments to muscles. Each connects to a set of skeletal muscle fibers and is innervated by a sensory neuron (fig. 12.4*b*). Golgi tendon organs have high thresholds, and increased tension stimulates them. Sensory impulses from them produce a reflex that inhibits contraction of the muscle whose tendon they occupy. Therefore, the Golgi tendon organs stimulate a reflex with an effect that is the opposite of a stretch reflex. The Golgi tendon reflex also helps maintain posture, and it protects muscle attachments from being pulled away from their insertions by excessive tension. Table 12.2 summarizes some of the receptors of the general senses and their functions.

Visceral Senses

Receptors in internal organs include lamellated corpuscles and free nerve endings. The information these receptors convey includes the sense of fullness after eating a meal, the discomfort of intestinal gas, and the pain that signals a heart attack.

PRACTICE

11 Describe a muscle spindle.

12 Explain how muscle spindles help maintain posture.

13 Where are Golgi tendon organs located?

14 What is the function of Golgi tendon organs?

12.2 CLINICAL APPLICATION

Mixed-Up Senses—Synesthesia

"The song was full of glittering orange diamonds."

"The paint smelled blue."

"The sunset was salty."

"The pickle tasted like a rectangle."

One in 1,000 people has a condition called synesthesia ("joined sensation"), in which the brain interprets a stimulus to one sense as coming from another. Most common is *grapheme-color type synesthesia,* in which letters, numbers, or time evoke specific colors. In *lexical-gustatory synesthesia,* a name evokes perception of a strong taste or odor. One woman thought of the name *Suzanne* as a strawberry and *Anthony* as a pork chop. These associations are involuntary and specific, and persist over a lifetime.

Most synesthesia is present for as long as a person can remember, but it may develop following brain damage. One woman began to feel touch sensations in response to certain sounds after suffering a stroke. Brain imaging traced the formation of unusual connections between her midbrain and the tactile part of her cerebral cortex. Positron-emission tomography scanning, which highlights blood flow in the cerebral cortex, shows dual areas lit up in synesthetes, corresponding to their shared sensations, and involvement of an area where the temporal, parietal, and occipital lobes meet.

Mutations in at least four genes cause synesthesia. The condition is eight times more prevalent among highly creative individuals than in the general population. Musicians John Mayer, Duke Ellington, Tori Amos, and Syd Barrett are synesthetes, as were architect Frank Lloyd Wright and novelist Vladimir Nabokov.

The physical basis of this curious condition is not well understood. It has been attributed to an immature nervous system that cannot sort out sensory stimuli, or to altered brain circuitry that routes stimuli to the wrong part of the cerebral cortex. Synesthesia may be common in infants, vanishing as synapses are cut back as part of normal development. Perhaps in synesthesia, too many synapses persist, and the person has the burden, or gift, of synesthesia.

Experimental devices that help people with deficient senses use the concept of synesthesia. For example, the "Brainport vision system" enables a person who cannot see to perceive a moving object with touch sensations on the tongue. The device has three parts. Special sunglasses housing a digital video camera link to a base unit worn on a belt, which sends the signals from incoming light patterns to an electrode array the size of a postage stamp, which is implanted on the surface of the tongue tip. Recipients report sensing "pictures painted on the tongue" that feel like bubbles of differing intensities. With practice, the visual cortex learns to interpret the "tactile image" sent to the tongue, and the person becomes able to tell when an object is in the visual field—which can be lifesaving. ■

TABLE **12.2** | Receptors Associated with General Senses

Type	Function	Sensation
Free nerve endings (mechanoreceptors)	Detect changes in pressure	Touch, pressure
Tactile corpuscles (mechanoreceptors)	Detect objects moving over the skin	Touch, texture
Lamellated corpuscles (mechanoreceptors)	Detect changes in pressure	Deep pressure, vibrations, fullness in viscera
Free nerve endings (thermoreceptors)	Detect changes in temperature	Heat, cold
Free nerve endings (pain receptors)	Detect tissue damage	Pain
Free nerve endings (mechanoreceptors)	Detect stretching of tissues, tissue spasms	Visceral pain
Muscle spindles (mechanoreceptors)	Detect changes in muscle length	None
Golgi tendon organs (mechanoreceptors)	Detect changes in muscle tension	None

12.4 SPECIAL SENSES

Special senses are those whose sensory receptors are part of large, complex sensory organs in the head. These senses and their respective organs include the following:

- smell → olfactory organs
- taste → taste buds
- hearing, equilibrium → ears
- sight → eyes

Clinical Application 12.2 discusses an unusual type of sensory abnormality.

Sense of Smell

The ability to detect the strong scent of a fish market, the antiseptic odor of a hospital, the aroma of a ripe melon—and thousands of other smells—is possible thanks to a yellowish patch of tissue the size of a quarter high up in the nasal cavity. This fabric of sensation is a layer of 12 million specialized cells.

Olfactory Receptors

Olfactory receptor cells, and their membrane receptor molecules, sense odors. They are similar to those for taste in that they are chemoreceptors sensitive to chemicals dissolved in liquids. The two chemical senses function closely together and aid in food selection, because we smell food at the same time we taste it. It is often difficult to tell what part of a food sensation is due to smell and what part is due to taste. For this reason, an onion tastes different when sampled with the nostrils closed, because much of the usual onion sensation is due to odor. Similarly, if copious mucous secretions from an upper respiratory infection cover the olfactory receptors, food may seem tasteless. About 75% to 80% of flavor derives from the sense of smell.

Olfactory Organs

The olfactory organs, which contain the olfactory receptor cells, also include epithelial supporting cells. These organs appear as yellowish brown masses within pinkish mucous membrane. They cover the upper parts of the nasal cavity, the superior nasal conchae, and a portion of the nasal septum (fig. 12.5).

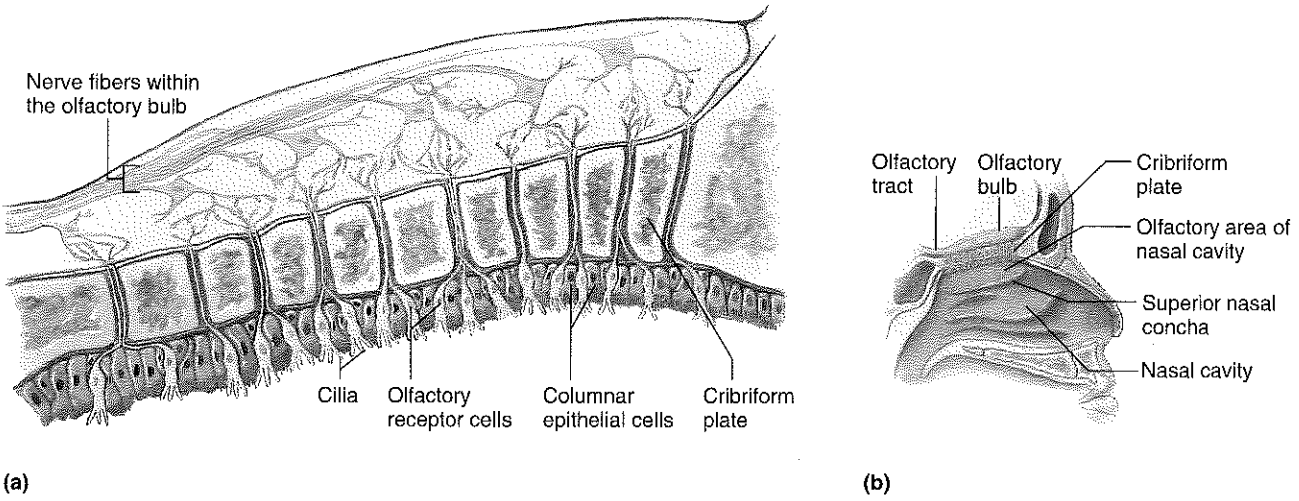


FIGURE 12.5 AP|R Olfactory receptors. (*a*) Columnar epithelial cells support olfactory receptor cells, which have cilia at their distal ends. (*b*) The olfactory area is associated with the superior nasal concha.

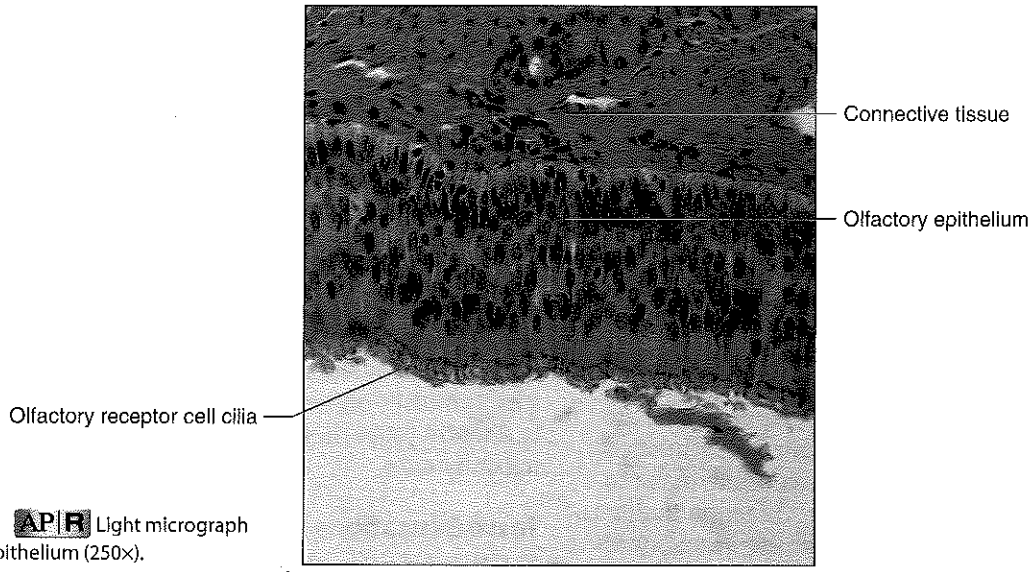


FIGURE 12.6 AP|R Light micrograph of the olfactory epithelium (250×).

The olfactory receptor cells are bipolar neurons surrounded by columnar epithelial cells. These neurons have knobs at the distal ends of their dendrites covered with hairlike cilia. The cilia project into the nasal cavity and are the sensitive portions of the receptor cells (fig. 12.6). Each of a person's 12 million olfactory receptor cells has ten to twenty cilia.

> Dogs can sense subtle odors that people emit when becoming ill with certain conditions. Service dogs are used to sense imminent seizures, drops in blood glucose, and accelerated heart rate. Dogs can detect a chemical in urine that prostate cancer cells release. In one study a dog identified a man whose prostate biopsy was normal. The biopsy was in error—not the dog. Cats have excellent olfaction too.

Chemicals that stimulate olfactory receptor cells, called odorant molecules, enter the nasal cavity and dissolve at least partially in the watery fluids that surround the cilia before they can bond to receptor proteins on the cilia and be detected. An odorant molecule may bind to several of the almost 400 types of olfactory membrane receptors that are part of the olfactory receptor cells, depolarizing these cells and thereby generating action potentials. Note the distinction between sensory receptors and membrane receptors. Sensory receptors may be as small as individual cells or as large as complex organs such as the eye or ear. They respond to sensory stimuli. Membrane receptors are molecules such as proteins and glycoproteins on the cell membranes. They allow cells, such as neurons and olfactory receptor cells, to respond to specific molecules.

Olfactory Pathways

Once olfactory receptor cells are stimulated, impulses are conducted along their axons through tiny openings in the cribriform plates of the ethmoid bone. These fibers (which collectively form the first cranial nerves) synapse with neurons located in the enlargements of the **olfactory bulbs,** which are structures that lie on either side of the crista galli of the ethmoid bone (see figs. 7.23, p. 221 and 12.5).

In the olfactory bulbs, the sensory impulses are analyzed, and as a result, additional impulses travel along the **olfactory tracts** to portions of the limbic system (see chapter 11, pp. 420–421), a brain center for memory and emotions. This is why we may become nostalgic over a scent from the past. A whiff of the perfume that grandma used to wear may bring back a flood of memories. The input to the limbic system also explains why odors can alter mood so easily. For example, the scent of new-mown hay or rain on a summer's morning generally makes us feel good. The main interpreting areas for the olfactory impulses (olfactory cortex) are deep within the temporal lobes and at the bases of the frontal lobes, anterior to the hypothalamus.

Olfactory Stimulation

Biologists are not certain how stimulated receptors encode specific smells, but a leading hypothesis is that each olfactory receptor cell has only one type of olfactory receptor, but that a receptor can bind several different types of odorant molecules. In addition, any one odorant molecule can bind several types of olfactory receptors. The brain interprets this binding information as an olfactory code. In a simplified example, banana might stimulate receptors 2, 4, and 7; garlic, receptors 1, 5, and 9. The olfactory organs are high in the nasal cavity above the usual pathway of inhaled air, so in order to smell a faint odor, a person may have to sniff and force air up to the receptor areas. Olfaction undergoes sensory adaptation rather rapidly, so the intensity of a smell drops about 50% within a second following the stimulation. Within a minute, the receptors may become almost insensitive to a given odor. This is why the odor of a fish market becomes tolerable quickly. However, olfactory receptors that have adapted to one scent remain sensitive to others.

The olfactory receptor neurons are the only nerve cells in direct contact with the outside environment, and as such they may be damaged. Fortunately, basal cells along the basement membrane of the olfactory epithelium regularly divide and yield differentiated cells that replace lost olfactory receptor neurons. These neurons are unusual in that they are regularly replaced when damaged.

PRACTICE

15 Where are the olfactory receptors located?

16 Trace the pathway of an olfactory impulse from a receptor to the cerebrum.

Sense of Taste

Taste buds are the special organs of taste. They resemble orange sections and associate on the surface of the tongue with tiny elevations called **papillae** (figs. 12.7 and 12.8). Taste buds are also scattered in the roof of the mouth, the linings of the cheeks, and the walls of the pharynx.

Taste Receptors

Each taste bud includes a group of modified epithelial cells, the **taste cells** (gustatory cells), that function as sensory receptors. Each of our 10,000 taste buds houses 50 to 150 taste cells. The taste bud also includes epithelial supporting cells. The entire structure of a taste bud is somewhat spherical, with an opening, the **taste pore,** on its free surface. Tiny projections (microvilli), called **taste hairs,** protrude from the outer ends of the taste cells and jut out through the taste pore. These taste hairs are the sensitive parts of the receptor cells.

Interwoven among and wrapped around the taste cells is a network of nerve fibers whose ends closely contact the receptor cell membranes. A stimulated receptor cell triggers an impulse on a nearby nerve fiber, which travels into the brain.

A chemical to be tasted must dissolve in saliva, which is the watery fluid surrounding the taste buds. The salivary glands supply this fluid. To demonstrate the importance of saliva, blot your tongue and try to taste some dry food; then repeat the test after moistening your tongue with saliva.

The sense of taste, like the taste of smell, derives from combinations of chemicals binding specific receptor proteins. This takes place on taste hair surfaces. The binding of the chemical alters membrane polarization, generating sensory impulses on nearby nerve fibers. The degree of change is directly proportional to the concentration of the stimulating substance.

Taste Sensations

The five primary taste sensations are sweet, sour, salty, bitter, and umami (oo-mom'ee). Each of the many flavors we experience results from one of the primary sensations or from a combination of them. The way we experience flavors may also reflect the concentration of chemicals as well as the sensations of smell, texture (touch), and temperature. Furthermore, chemicals in some foods—such as capsaicin in chili peppers—may stimulate pain receptors that cause a burning sensation.

Experiments indicate that each taste cell responds to one taste sensation only, with distinct receptors. Taste cells for each of the five taste sensations are in all areas of the tongue, but are distributed such that each sensation seems to arise most strongly from a particular region. Due to the distribution of taste cells, responsiveness to particular sensations varies from one region of the tongue to another. Sensitivity to a sweet stimulus peaks at the tip of the tongue, whereas responsiveness to sour is greatest at the margins of the tongue, and to bitter at the back. Receptors particularly responsive to salt are widely distributed.

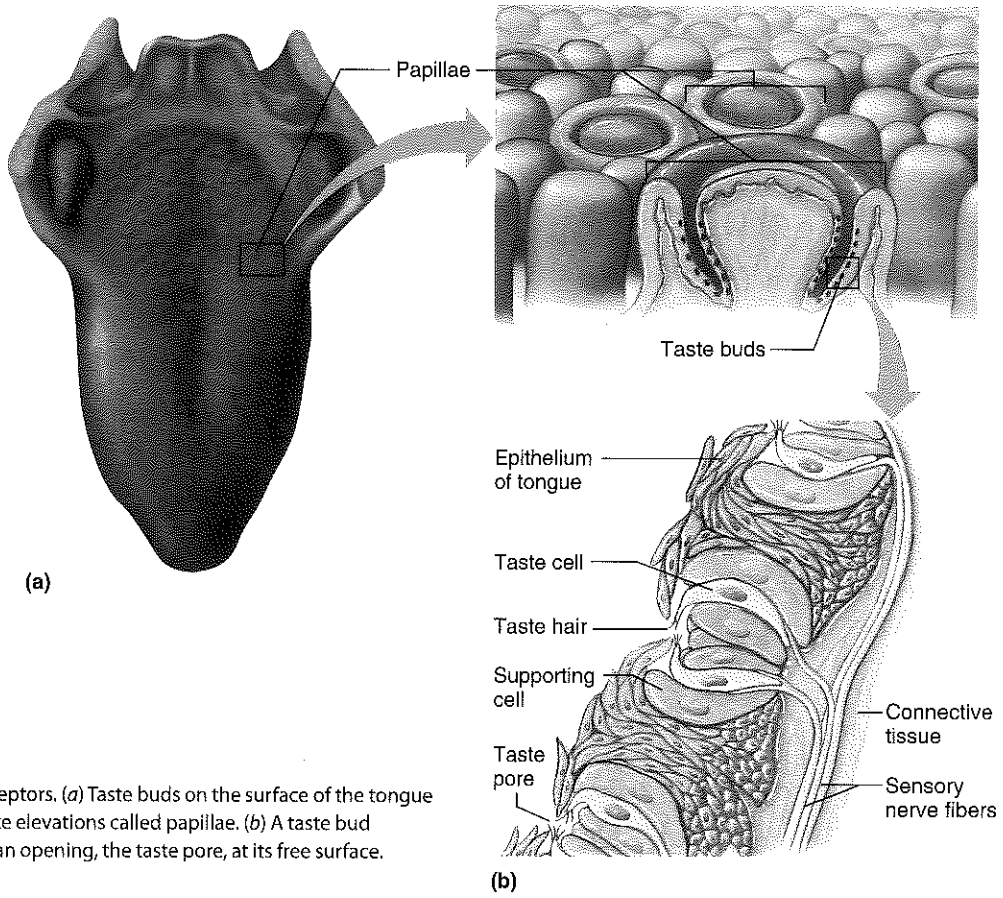


FIGURE 12.7 Taste receptors. (*a*) Taste buds on the surface of the tongue are associated with nipplelike elevations called papillae. (*b*) A taste bud contains taste cells and has an opening, the taste pore, at its free surface.

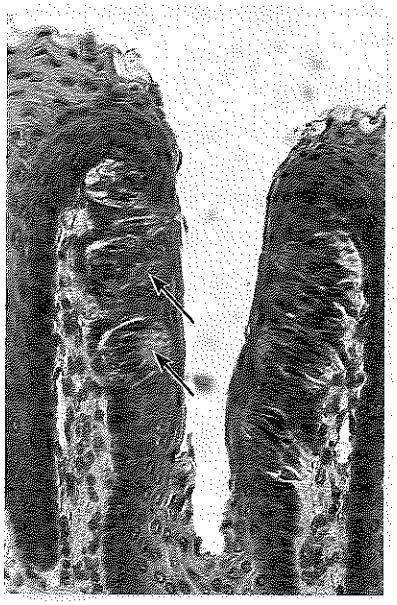


FIGURE 12.8 AP|R A light micrograph of some taste buds (arrows) (225×).

Sweet receptors are usually stimulated by carbohydrates, but a few inorganic substances, including some salts of lead and beryllium, also elicit sweet sensations. Acids stimulate *sour receptors*. The intensity of a sour sensation is roughly proportional to the concentration of the hydrogen ions in the substance being tasted. Ionized inorganic salts mainly stimulate *salt receptors*. The quality of the sensation that each salt produces depends upon the type of positively charged ion that it releases into solution, such as Na^+ from table salt. A variety of chemicals stimulates *bitter receptors*, including many organic compounds. Inorganic salts of magnesium and calcium produce bitter sensations, too. Extreme sensitivity to bitter tastes is inherited, which is why diet colas taste sweet to some people but are bitter to others. Twenty-five types of bitter receptors have been identified. Quite a few of them detect flavors unique to fermented foods.

One group of bitter compounds of particular interest are the *alkaloids*, which include a number of poisons such as strychnine, nicotine, and morphine. Spitting out bitter substances may be a protective mechanism to avoid ingesting poisonous alkaloids in foods.

The taste sensation called *umami* has long been recognized in Japan (the word means "delicious" in Japanese) but has only recently come to the attention of Western taste researchers. Umami arises from the binding of certain amino acids, including glutamic acid and aspartic acid, to specific receptors. The flavor enhancer monosodium glutamate (MSG), used in many prepared foods, is formed from glutamic acid and also stimulates umami receptors.

Taste receptors, like olfactory receptors, rapidly undergo sensory adaptation. The resulting loss of taste can be avoided by moving bits of food over the surface of the tongue to stimulate different receptors at different moments.

12.3 CLINICAL APPLICATION

Smell and Taste Disorders

Imagine a spicy slice of pizza or freshly brewed coffee, and your mouth waters in anticipation. But for millions of people, the senses of smell and taste are dulled, distorted, or gone. Many more of us get some idea of their plight when a cold temporarily stifles these senses.

Compared to the loss of hearing or sight, being unable to taste or smell may seem more an oddity than an illness. People with such ailments would probably disagree. In some situations, a poor or absent sense of smell can be dangerous, such as in a house on fire.

The direct connection between the outside environment and the brain makes the sense of smell vulnerable to damage. Smell and taste disorders can be triggered by colds and flu, allergies, nasal polyps, swollen mucous membranes inside the nose, a head injury, chemical exposure, a nutritional or metabolic problem, or a disease. In many cases, a cause cannot be identified.

Drugs can alter taste and smell in many ways, affecting cell turnover, the neural conduction system, the status of receptors, and changes in nutritional status. Drugs containing sulfur atoms, for example, squelch taste. They include the anti-inflammatory drug penicillamine, the antihypertensive drug captopril (Capoten), and transdermal (patch) nitroglycerin to treat chest pain. The antibiotic tetracycline and the antiprotozoan metronidazole (Flagyl) impart a metallic taste. Cancer chemotherapy and radiation treatment often alter taste and smell.

Exposure to toxic chemicals can affect taste and smell, too. For example, excess exposure to organic solvents in paint thinner can cause *cacosmia*, the association of an odor of decay with normally inoffensive stimuli. ■

We experience a series of flavors as we savor some foods because of a sequence of chemical reactions that take place in foods as we chew them. Imagine biting into a juicy ripe peach. The fruit's tissues tear, first releasing aromatic hydrocarbons. Thirty seconds later, products of fatty acid breakdown appear and, finally, several alcohols are released. This gradual flood of stimulating molecules is one reason why we enjoy eating so much.

Although taste cells are close to the surface of the tongue and are therefore exposed to environmental wear and tear, the sense of taste is not as likely to diminish with age as is the sense of smell. This is because taste cells are modified epithelial cells and divide continually. A taste cell functions for only about three days before it is replaced.

Taste Pathways

Sensory impulses from taste receptor cells in the anterior two-thirds of the tongue travel on fibers of the facial nerve (VII); impulses from receptors in the posterior one-third of the tongue and the back of the mouth pass along the glossopharyngeal nerve (IX); and impulses from receptors at the base of the tongue and the pharynx travel on the vagus nerve (X). These cranial nerves conduct the impulses into the medulla oblongata. From there, impulses ascend to the thalamus and are directed to the gustatory cortex of the cerebrum, located in the parietal lobe along a deep part of the lateral sulcus. Clinical Application 12.3 and table 12.3 discuss disorders of smell and taste.

PRACTICE

17 Why is saliva necessary to taste?

18 Name the five primary taste sensations.

19 What characteristic of taste receptors helps maintain a sense of taste with age?

20 Trace a sensory impulse from a taste receptor to the cerebral cortex.

TABLE **12.3** | Types of Smell and Taste Disorders

	Smell	Taste
Loss of sensation	Anosmia	Ageusia
Diminished sensation	Hyposmia	Hypogeusia
Heightened sensation	Hyperosmia	Hypergeusia
Distorted sensation	Dysosmia	Dysgeusia

RECONNECT

To Chapter 11, Cranial Nerves, pages 420–422 and Table 11.9, page 423.

Sense of Hearing

The organ of hearing, the *ear*, has outer (external), middle, and inner (internal) sections. In addition to making hearing possible, the ear provides the sense of equilibrium.

Outer (External) Ear

The outer ear consists of all of the structures that face the outside. These include an outer, funnel-like structure called the **auricle** (pinna) and an S-shaped tube, the *external acoustic* (ah-kōōs'tik) *meatus* (external auditory canal) that leads inward for about 2.5 centimeters (fig. 12.9). The meatus terminates with the **tympanic membrane** (eardrum). AP|R

The external acoustic meatus passes into the temporal bone. Near this opening, hairs guard the tube. The opening and tube are lined with skin that has many modified sweat glands called *ceruminous glands*, which secrete wax (cerumen). The hairs and wax help keep large foreign objects, such as insects, out of the ear.

Ear wax may be "wet" or "dry." The wet type has a higher lipid content and is brown. It is found among people of European or African ancestry. The dry form is grey and is found among Asians and Native Americans. A single DNA base difference determines whether ear wax is wet or dry.

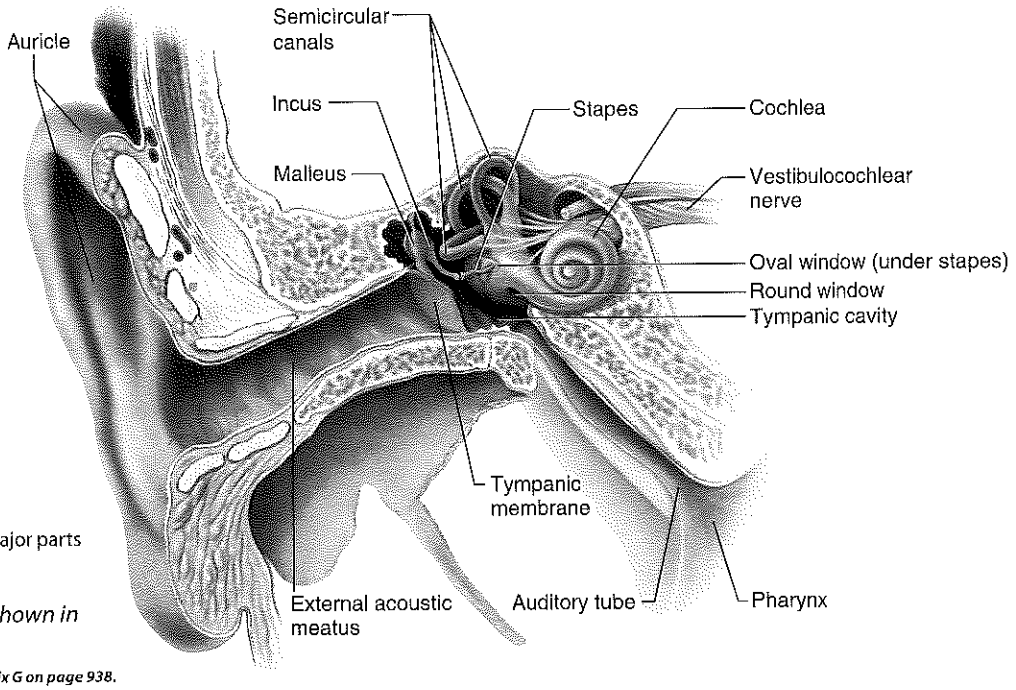


FIGURE 12.9 AP|R Major parts of the ear.

Q: *Which cranial bone is shown in this figure?*

Answer can be found in Appendix G on page 938.

The transfer of vibrations through matter produces sound. Just as vibrating strings or reeds provide the sounds of some musical instruments, vibrating vocal folds in the larynx provide the sounds of the human voice. The auricle of the ear helps collect sound waves traveling through air and directs them into the external acoustic meatus.

After entering the meatus, the sound waves pass to the end of the tube and alter the pressure on the tympanic membrane. The tympanic membrane is a semitransparent membrane covered by a thin layer of skin on its outer surface and by mucous membrane on the inside. It has an oval margin and is cone-shaped, with the apex of the cone directed inward. The tympanic membrane moves back and forth in response to sound waves, reproducing the vibrations of the sound-wave source.

Middle Ear

The **middle ear,** or the **tympanic cavity,** is an air-filled space in the temporal bone that separates the outer and inner ears. It is bounded by the tympanic membrane laterally and the inner ear medially and houses three small bones called **auditory ossicles** (aw′di-to″re os′i-klz).

The three auditory ossicles, called the *malleus,* the *incus,* and the *stapes,* are attached to the wall of the tympanic cavity by tiny ligaments and are covered by mucous membrane. These bones bridge the tympanic membrane and the inner ear, transferring vibrations between these parts. Specifically, the malleus is attached to the tympanic membrane, helping to maintain its conical shape. When the tympanic membrane vibrates, the malleus vibrates in unison with it. The malleus vibrates the incus, and the incus passes the movement on to the stapes. Ligaments hold the stapes to an opening in the wall of the tympanic cavity called the **oval window** (fig. 12.9). Vibration of the stapes, which acts like a piston at the oval window, transfers the vibrations to a fluid within the inner ear. These vibrations of the fluid stimulate the hearing receptors.

In addition to transferring vibrations, the auditory ossicles form a lever system that helps increase (amplify) the force of the vibrations as they pass from the tympanic membrane to the oval window. Also, because the ossicles transfer vibrations from the large surface of the tympanic membrane to a much smaller area at the oval window, the vibrational force strengthens as it travels from the outer to the inner ear. As a result, the pressure (per square millimeter) that the stapes applies at the oval window is about twenty-two times greater than that which sound waves exert on the tympanic membrane.

The middle ear also has two small skeletal muscles attached to the auditory ossicles and controlled by a reflex. One of them, the *tensor tympani,* is inserted on the medial surface of the malleus and is anchored to the cartilaginous wall of the auditory tube. When it contracts, it pulls the malleus inward. The other muscle, the *stapedius,* is attached to the posterior side of the stapes and the inner wall of the tympanic cavity. It pulls the stapes outward when it contracts (fig. 12.10). These muscles are the effectors in the **tympanic reflex,** which is elicited in about one-tenth of a second after a loud, external sound. The reflex contracts the muscles, and the malleus and stapes move. As a result, the bridge of ossicles in the middle ear becomes more rigid, reducing its effectiveness in transferring vibrations to the inner ear.

The tympanic reflex reduces pressure from loud sounds that might otherwise damage the hearing receptors. Ordinary vocal sounds also elicit the tympanic reflex, such as when a person speaks or sings. This action muffles the lower

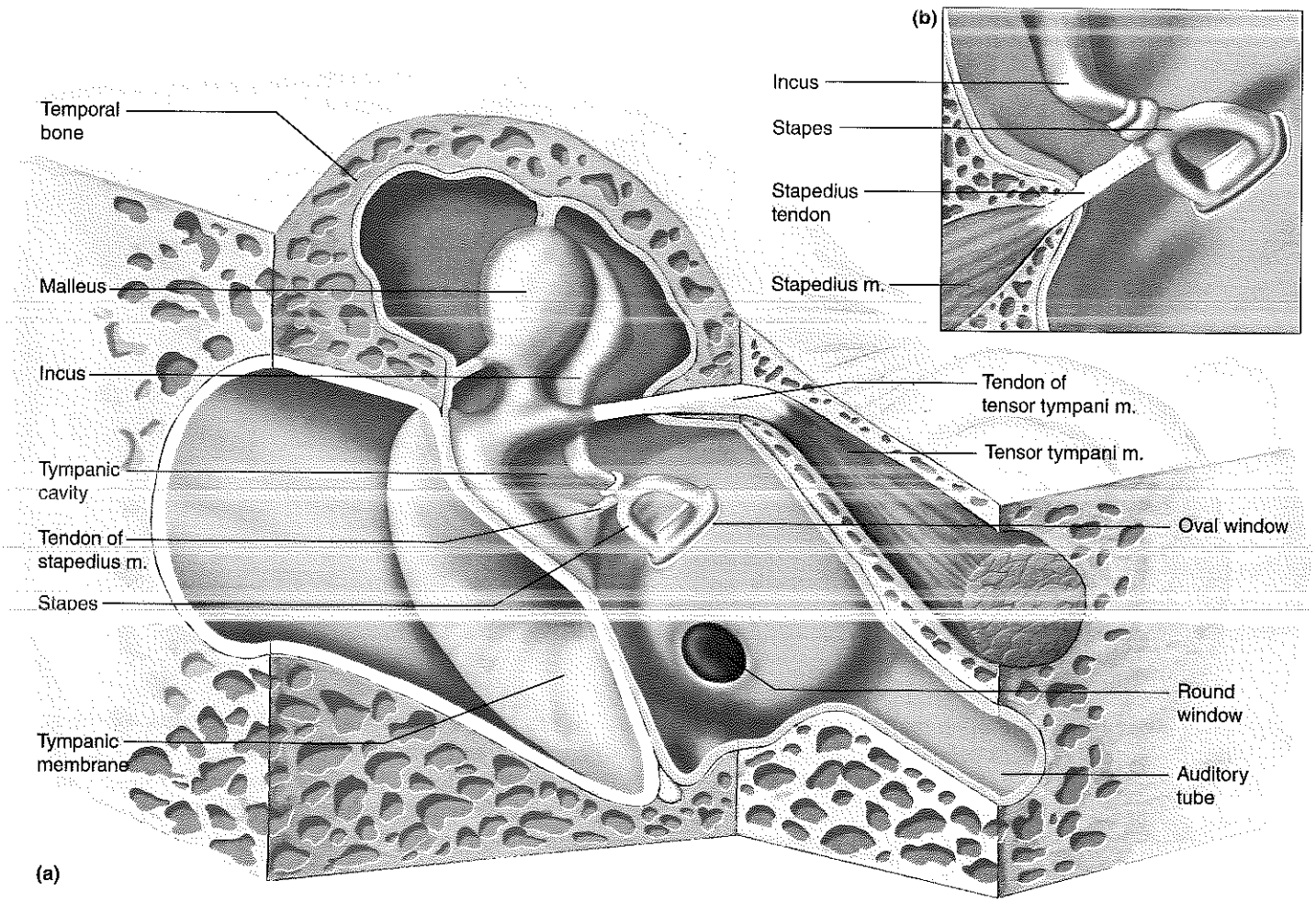


FIGURE 12.10 Two small muscles attached to the (*a*) malleus and (*b*) stapes, the tensor tympani and the stapedius, are effectors in the tympanic reflex. Figure 12.9 does not show these muscles. (*m.* stands for muscle.)

frequencies of such sounds, improving the hearing of higher frequencies, which are common in human vocal sounds. In addition, the tensor tympani muscle maintains tension on the tympanic membrane. This is important because a loose tympanic membrane would not be able to effectively transmit vibrations to the auditory ossicles.

The muscles of the middle ear take 100 to 200 milliseconds to contract. For this reason, the tympanic reflex cannot protect the hearing receptors from the effects of very sudden loud sounds, such as from an explosion or a gunshot. On the other hand, this protective mechanism can reduce the effects of intense sounds that arise slowly, such as the roar of thunder.

Auditory Tube

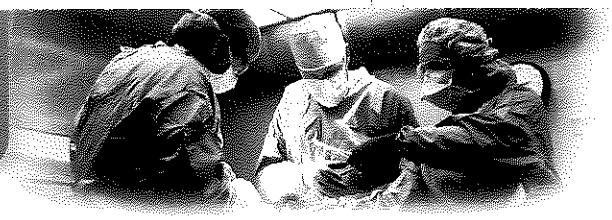
An **auditory tube** (aw′di-to″re tub) (eustachian tube) connects each middle ear to the throat. This tube allows air to pass between the tympanic cavity and the outside of the body by way of the throat (nasopharynx) and mouth. It helps maintain equal air pressure on both sides of the tympanic membrane. This is necessary for normal hearing (see fig. 12.10).

The function of the auditory tube becomes noticeable during rapid change in altitude. As a person descends from a high altitude, the air pressure on the outside of the tympanic membrane steadily increases. This may push the tympanic membrane inward, out of its normal position, impairing hearing.

When the air pressure difference on the sides of the tympanic membrane is great enough, some air may force its way up through the auditory tube into the middle ear. This equalizes the pressure on both sides of the tympanic membrane, which moves back into its regular position, causing a popping sound as normal hearing returns. A reverse movement of air ordinarily occurs when a person ascends from a low altitude.

The auditory tube is usually closed by valvelike flaps in the throat, which may inhibit air movements into the middle ear. Swallowing, yawning, or chewing aid in opening the flaps and can hasten equalization of air pressure.

12.4 CLINICAL APPLICATION



Getting a Cochlear Implant

Yolanda probably lost her hearing when she suffered a high fever at eight weeks of age. When Yolanda was nine months old and still didn't babble like her age-mates, her parents suspected she might be deaf. With hearing aids she did well at a preschool for the deaf. Then Yolanda's parents read about the cochlear implant, a device that does not magically restore hearing, but enables a person to hear certain sounds.

Yolanda received her cochlear implant when she was three years old. Before three is the best time because the brain is rapidly processing speech and hearing as the person masters language. However, even people who lose their hearing as adults can benefit from cochlear implants, because they link the sounds they hear through the device to memories of what sounds were like, perhaps using clues from other senses.

The cochlear implant consists of a part inserted under the skin above the ear that leads to two dozen electrodes placed near the auditory nerve in the cochlea, the snail-shaped part of the inner ear. Yolanda wears a headset that includes a microphone lodged at the back of her ear to pick up incoming sounds and a fanny pack containing a speech processor that digitizes the sounds into coded signals. A transmitter on the headset sends the coded signals, as FM radio waves, to the implant, which changes them to electrical signals and delivers them to the cochlea. Here, the auditory nerve is stimulated and sends neural messages to the brain's cerebral cortex, which interprets the input as sound.

Yolanda's audiologist turned on the speech processor a month after the surgery. At first, the youngster heard low sounds and sometimes responded with a low hum. She grabbed at the processor, realizing it was the source of the sound. Gradually, the little girl learned from context what certain sounds meant. One day when Carlos signed "father" and said "poppy," Yolanda signed back and tried to say the word! Able to connect mouth movements to sounds to concepts, Yolanda was well on her way to hearing. About 200,000 people worldwide have received cochlear implants since the devices became available in 1984. ■

FIGURE 12D Yolanda received a cochlear implant when she was three years old. The device enables her to detect enough sounds to effectively communicate.

Signs of a middle ear infection (otitis media) in a toddler include irritability, fever, and tugging on the painful ear. Using an instrument called an otoscope reveals a red and bulging tympanic membrane.

Ear infections occur because the mucous membranes that line the auditory tubes are continuous with the linings of the middle ears, enabling bacteria infecting the throat or nasal passages to reach the ear. The bacteria that most commonly cause middle ear infection are *Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis*. This route to infection is greater in young children because their auditory tubes are shorter than those in adults.

Acute otitis media is treated with antibiotics, but these might not be prescribed at first for a child older than two years who is not in severe pain, because such infections tend to clear up on their own in a few days. A child with recurrent otitis media might have brief surgery to fit the affected ear with a tympanostomy tube, to lower the risk of hearing loss. The tube drains the ear through a small hole in the tympanic membrane. APR

Inner (Internal) Ear

The inner ear is a complex system of intercommunicating chambers and tubes called a **labyrinth** (lab'i-rinth). Each ear has two such regions—the osseous labyrinth and the membranous labyrinth.

The *osseous labyrinth* is a bony canal in the temporal bone; the *membranous labyrinth* is a tube that lies within the osseous labyrinth and has a similar shape (fig. 12.11*a*). Between the osseous and membranous labyrinths is a fluid called *perilymph,* secreted by cells in the wall of the bony canal. In the membranous labyrinth is a slightly different fluid called *endolymph.*

The parts of the labyrinths include a **cochlea** (kok'le-ah) that functions in hearing and three **semicircular canals** that provide a sense of equilibrium. A bony chamber called the **vestibule,** between the cochlea and the semicircular canals, houses membranous structures that serve both hearing and equilibrium.

The cochlea is shaped like a snail shell, coiled around a bony core (modiolus) with a thin, bony shelf (spiral lamina) that wraps around the core (fig. 12.11*b*). The shelf divides the bony labyrinth of the cochlea into upper and lower compartments. The upper compartment, called the *scala vestibuli,* leads from the oval window to the apex of the spiral. The lower compartment, the *scala tympani,* extends from the apex of the cochlea to a membrane-covered opening in the wall of the inner ear called the **round window.** These compartments constitute the bony labyrinth of the cochlea, and they are filled with perilymph. At the apex of the cochlea, a small opening (helicotrema) connects the fluids in the chambers (figs. 12.11*b* and 12.12).

A portion of the membranous labyrinth in the cochlea, called the *cochlear duct* (scala media), lies between the two bony compartments and is filled with endolymph. The cochlear duct ends as a closed sac at the apex of the cochlea. The duct is separated from the scala vestibuli by a *vestibular membrane* (Reissner's membrane) and from the scala tympani by a *basilar membrane* (see fig. 12.12). Clinical Application 12.4 describes an effective treatment for hearing loss called a cochlear implant.

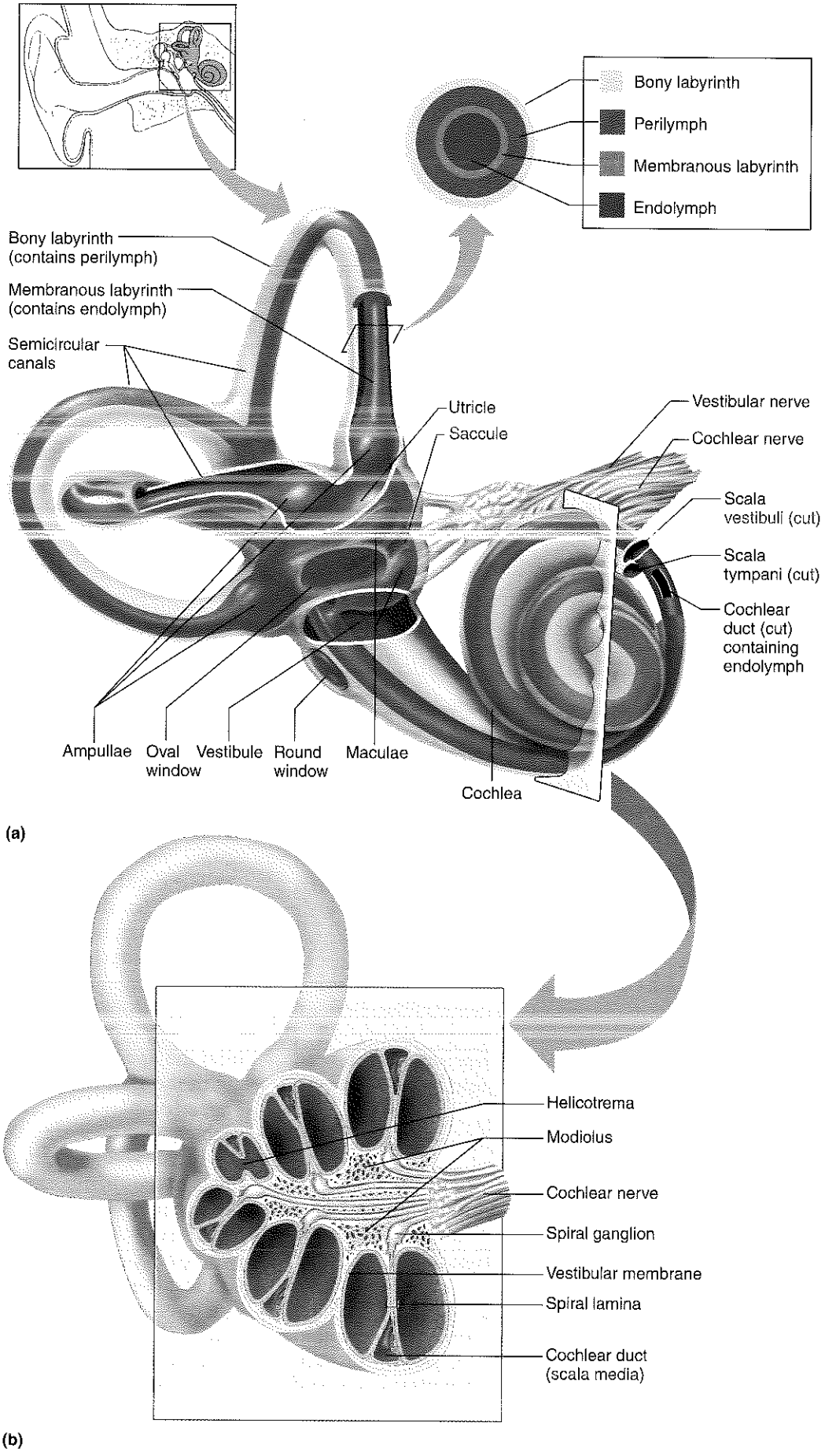


FIGURE 12.11 AP|R In the inner ear (*a*) perilymph separates the osseous labyrinth from the membranous labyrinth, which contains endolymph. (*b*) The spiral lamina coils around a bony core, the modiolus.

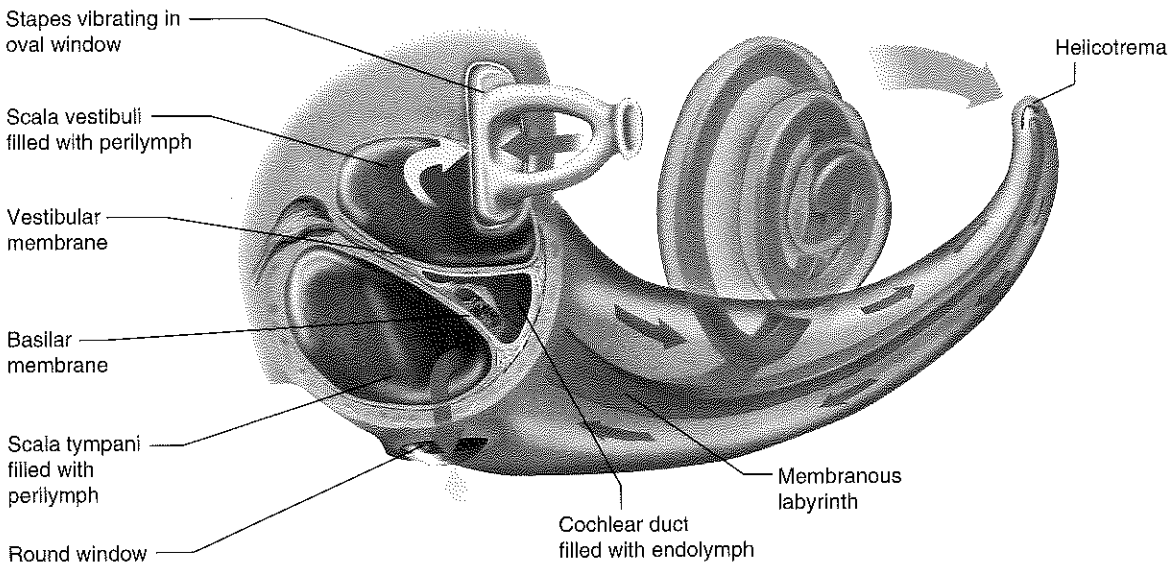


FIGURE 12.12 AP|R The cochlea is a coiled, bony canal with a membranous tube (labyrinth) inside. If the cochlea could be unwound, the membranous labyrinth would be seen ending as a closed sac at the apex where the bony canal makes a U-turn.

The basilar membrane extends from the bony shelf of the cochlea and forms the floor of the cochlear duct. It has many thousands of stiff, elastic fibers that lengthen from the base of the cochlea to its apex. Vibrations entering the perilymph at the oval window travel along the scala vestibuli and pass through the vestibular membrane to enter the endolymph of the cochlear duct, where they move the basilar membrane. After passing through the basilar membrane, the vibrations enter the perilymph of the scala tympani, and movement of the membrane covering the round window dissipates their force into the air in the tympanic cavity.

The **spiral organ** (organ of Corti), which contains about 16,000 hearing receptor cells, is on the superior surface of the basilar membrane and stretches from the apex to the base of the cochlea. The receptor cells, called **hair cells**, are in four parallel rows, with many hairlike processes known as stereovilli (also called stereocilia) that extend into the endolymph of the cochlear duct. Above these hair cells is a *tectorial membrane,* attached to the bony shelf of the cochlea. It passes like a roof over the receptor cells, contacting the tips of their hairs (figs. 12.13 and 12.14).

Different frequencies of vibration move different regions along the length of the basilar membrane. A particular sound frequency bends the hairs of a specific group of receptor cells against the tectorial membrane. Other frequencies deflect hair cells elsewhere along the cochlea.

The greater the deflection of the basilar membrane pushing the hair cells upward against the tectorial membrane, the louder the sound. However, recall from chapter 10 (p. 377) that action potentials are all-or-none. More intense stimulation of the hair cells causes more action potentials per second to reach the brain, and we sense a louder sound.

Hearing receptor cells are epithelial cells, but they respond to stimuli somewhat like neurons (see chapter 10, pp. 372–377). For example, when a receptor cell is at rest, its membrane is polarized. When its hairs bend, selective ion channels open and its cell membrane depolarizes. The membrane then becomes more permeable, specifically to calcium ions. The receptor cell has no axon or dendrites, but it does have neurotransmitter-containing vesicles in the cytoplasm near its base. As calcium ions diffuse into the cell, some of these vesicles fuse with the cell membrane and release neurotransmitter to the outside. The neurotransmitter stimulates the dendritic ends of nearby sensory neurons, and in response they transmit impulses along the cochlear branch of the vestibulocochlear nerve (cranial nerve VIII) to the brain.

The ear of a young person with normal hearing can detect sound waves with frequencies varying from about 20 to 20,000 or more vibrations per second. The range of greatest sensitivity is between 2,000 and 3,000 vibrations per second (fig. 12.15).

Auditory Pathways

The cochlear branches of the vestibulocochlear nerves enter the auditory nerve pathways that extend into the medulla oblongata and proceed through the midbrain to the thalamus. From there they pass into the auditory cortices of the temporal lobes of the cerebrum, where they are interpreted. On the way, some of these fibers cross over, so that impulses arising from each ear are interpreted on both sides of the brain. Consequently, damage to a temporal lobe on one side of the brain is not necessarily accompanied by complete hearing loss in the ear on that side (fig. 12.16).

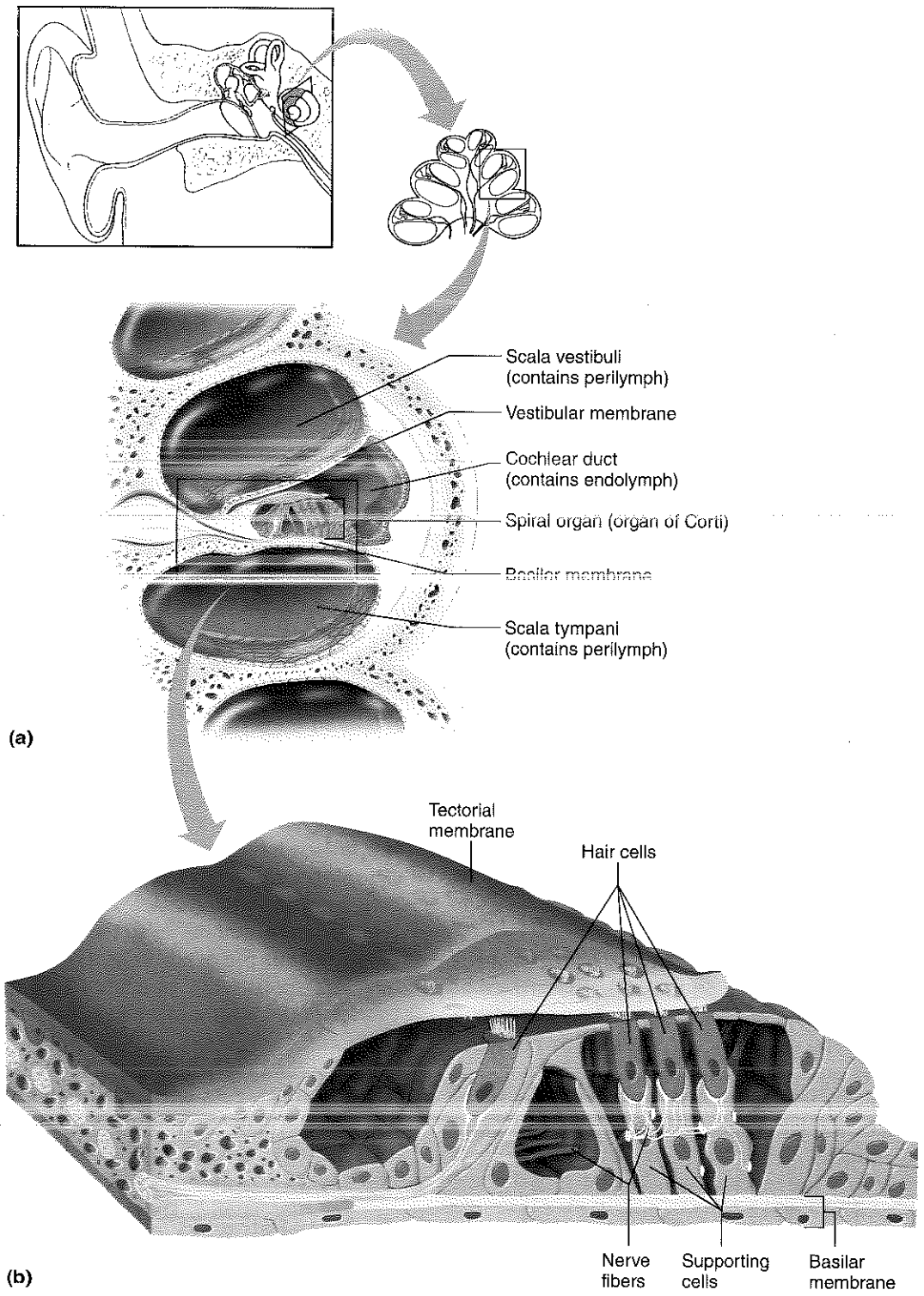
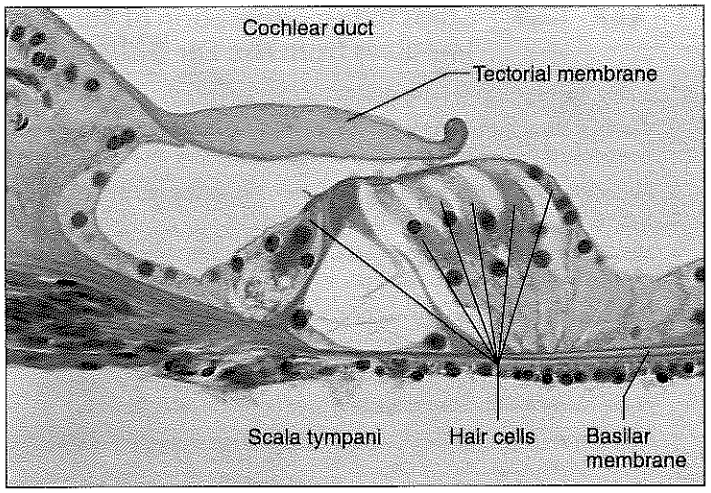
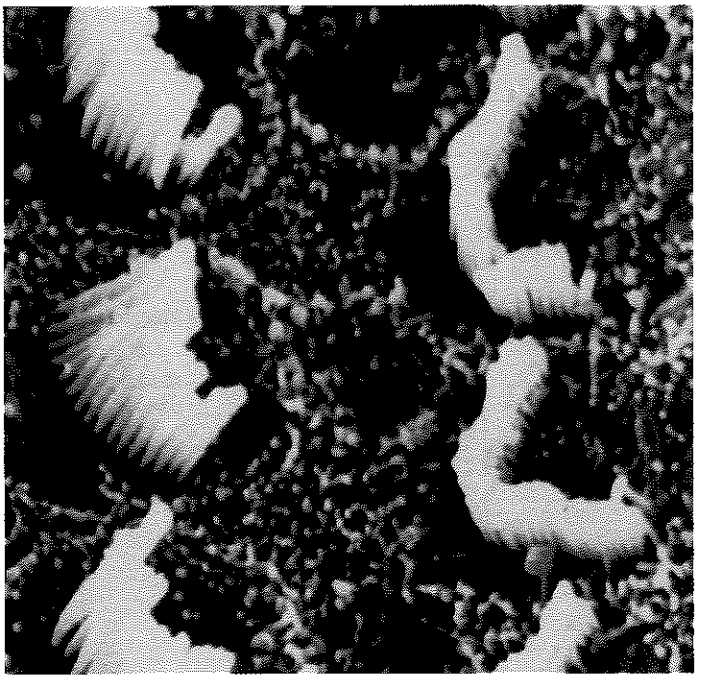


FIGURE 12.13 Cochlea. (*a*) Cross section of the cochlea. (*b*) Spiral organ and the tectorial membrane.



(a)



(b)

FIGURE 12.14 AP|R Spiral organ. (*a*) A micrograph of the spiral organ and the tectorial membrane (300×). (*b*) A scanning electron micrograph of hair cells in the spiral organ, looking down on the "hairs" (bright yellow) (6,700×).

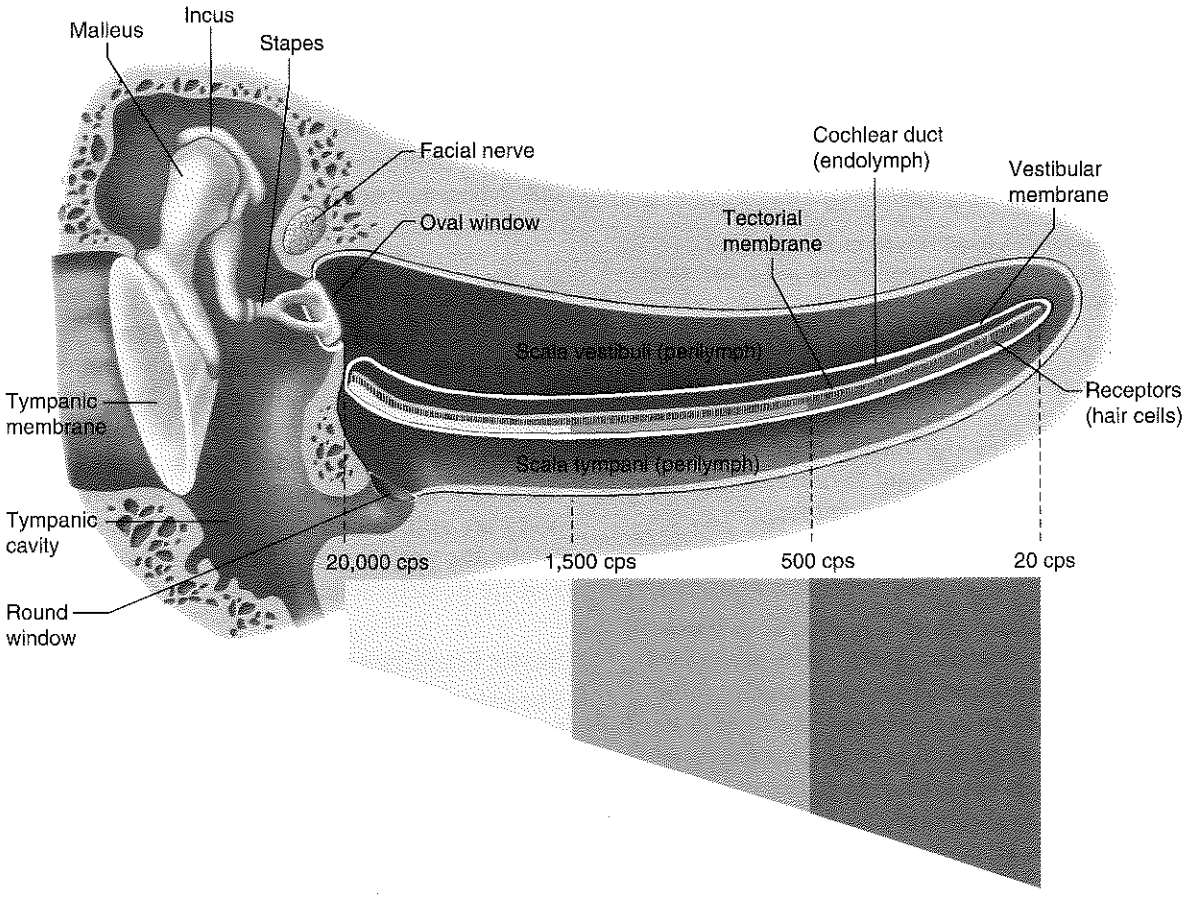


FIGURE 12.15 How the cochlea might look if it could be straightened out. Receptors in regions of the cochlear duct sense different frequencies of vibration, expressed in cycles per second (cps).

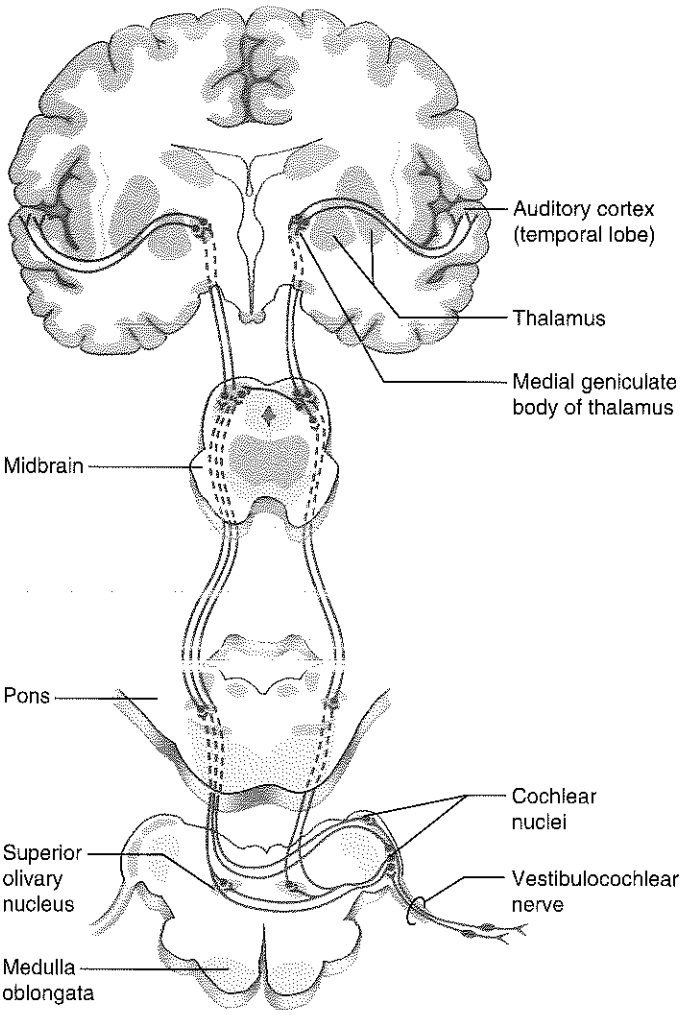


FIGURE 12.16 The auditory nerve pathway extends into the medulla oblongata, proceeds through the midbrain to the thalamus, and passes into the auditory cortex of the cerebrum.

Table 12.4 summarizes the pathway of vibrations through the parts of the middle and inner ears. Clinical Application 12.5 examines types of hearing loss.

Units called *decibels* (dB) measure sound intensity as a logarithmic scale. The decibel scale begins at 0 dB, which is the intensity of the sound least perceptible by a normal human ear. A sound of 10 dB is 10 times as intense as the least perceptible sound; a sound of 20 dB is 100 times as intense; and a sound of 30 dB is 1,000 times as intense. A whisper has an intensity of about 40 dB, normal conversation measures 60–70 dB, and heavy traffic produces about 80 dB. A sound of 120 dB, such as a rock concert, produces discomfort; and a sound of 140 dB, such as a jet plane at takeoff, causes pain. Frequent or prolonged exposure to sounds with intensities above 90 dB can cause permanent hearing loss.

TABLE 12.4 | Steps in the Generation of Sensory Impulses from the Ear AP R

1. Sound waves enter the external acoustic meatus.
2. Waves of changing pressures cause the tympanic membrane to reproduce the vibrations coming from the sound-wave source.
3. Auditory ossicles amplify and transmit vibrations to the end of the stapes.
4. Movement of the stapes at the oval window transmits vibrations to the perilymph in the scala vestibuli.
5. Vibrations pass through the vestibular membrane and enter the endolymph of the cochlear duct.
6. Different frequencies of vibration in endolymph move specific regions of the basilar membrane, stimulating specific sets of receptor cells.
7. A receptor cell depolarizes; its membrane becomes more permeable to calcium ions.
8. In the presence of calcium ions, vesicles at the base of the receptor cell release neurotransmitter.
9. Neurotransmitter stimulates nearby sensory neurons.
10. Sensory impulses are conducted along fibers of the cochlear branch of the vestibulocochlear nerve.
11. The auditory cortex of the temporal lobe interprets the sensory impulses.

PRACTICE

21 Describe the outer, middle, and inner ears.

22 Explain how sound waves are transmitted through the parts of the ear.

23 Describe the tympanic reflex.

24 Distinguish between the osseous and membranous labyrinths.

25 Explain the function of the spiral organ.

Sense of Equilibrium

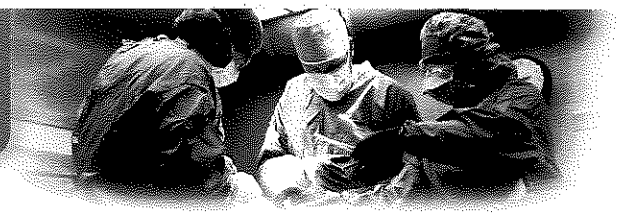
The feeling of equilibrium derives from two senses—**static equilibrium** (stat'ik e'kwĭ-lib're-um) and **dynamic equilibrium** (di-nam'ik e'kwĭ-lib're-um). Different sensory organs provide these two components of equilibrium. The organs associated with static equilibrium sense the position of the head, maintaining stability and posture when the head and body are still. When the head and body suddenly move or rotate, the organs of dynamic equilibrium detect the motion and aid in maintaining balance.

Static Equilibrium

The organs of static equilibrium are in the vestibule, which is a bony chamber between the semicircular canals and the cochlea. More specifically, the membranous labyrinth inside the vestibule consists of two expanded chambers—a **utricle** (u'trĭ-kl) and a **saccule** (sak'ūl). The larger utricle communicates with the saccule and the membranous portions of the semicircular canals; the saccule, in turn, communicates with the cochlear duct (fig. 12.17).

The utricle and saccule each has a small patch of hair cells and supporting cells called a **macula** (mak'u-lah) on its wall.

12.5 CLINICAL APPLICATION



Hearing Loss

About 8% of people have some degree of hearing loss. Several factors can impair hearing, including interference with transmission of vibrations to the inner ear *(conductive deafness)* or damage to the cochlea or the auditory nerve and its pathways *(sensorineural deafness)*. Disease, injury, and heredity all can impair hearing. There are more than 100 forms of inherited deafness, and many are part of syndromes. About 95% of cases of hearing loss are conductive. One cause is accumulated dry wax or a foreign object in the ear, which plugs the acoustic meatus. Changes in the tympanic membrane or auditory ossicles can also block hearing. The tympanic membrane may harden as a result of disease, becoming less responsive to sound waves, or an injury may tear or perforate it.

A common disorder of the auditory ossicles is *otosclerosis*, in which new bone is deposited abnormally around the base of the stapes. This interferes with the movement of the ossicles that is necessary to transmit vibrations to the inner ear. Surgery often can restore some hearing by chipping away the bone that holds the stapes in position or replacing the stapes with a wire or plastic substitute.

Two tests used to diagnose conductive deafness are the Weber test and the Rinne test. In the Weber test, the handle of a vibrating tuning fork is pressed against the forehead. A person with normal hearing perceives the sound coming from directly in front, whereas a person with sound conduction blockage in one middle ear hears the sound coming from the impaired side.

In the Rinne test, a vibrating tuning fork is held against the bone behind the ear. After the sound is no longer heard by conduction through the bones of the skull, the fork is moved to just in front of the external acoustic meatus. In middle ear conductive deafness, the vibrating fork can no longer be heard, but a normal ear will continue to hear its tone.

Very loud sounds can cause sensorineural deafness. If exposure is brief, hearing loss may be temporary, but when exposure is repeated and prolonged, such as occurs in foundries, near jackhammers, or on a firing range, impairment may be permanent. Such hearing loss begins as the hair cells develop blister-like bulges that eventually pop. The tissue beneath the hair cells swells and softens until the hair cells, and sometimes the neurons, leaving the cochlea become blanketed with scar tissue and degenerate. Other causes of sensorineural deafness include tumors in the CNS, brain damage as a result of vascular accidents, and the use of certain drugs.

Hearing loss and other ear problems can begin gradually. Signs include:

- difficulty hearing people talking softly
- inability to understand speech when there is background noise
- ringing in the ears
- dizziness
- loss of balance

New parents should notice whether their infant responds to sounds in a way that indicates normal hearing. Hearing exams are part of a well-baby visit to a doctor. If the baby's responses indicate a possible problem, the next step is to see an audiologist, who identifies and measures hearing loss.

Often a hearing aid can help people with conductive hearing loss. A hearing aid has a tiny microphone that picks up sound waves and converts them to electrical signals, which are then amplified. An ear mold holds the device in place, either behind the outer ear, in the outer ear, or in the ear canal. ■

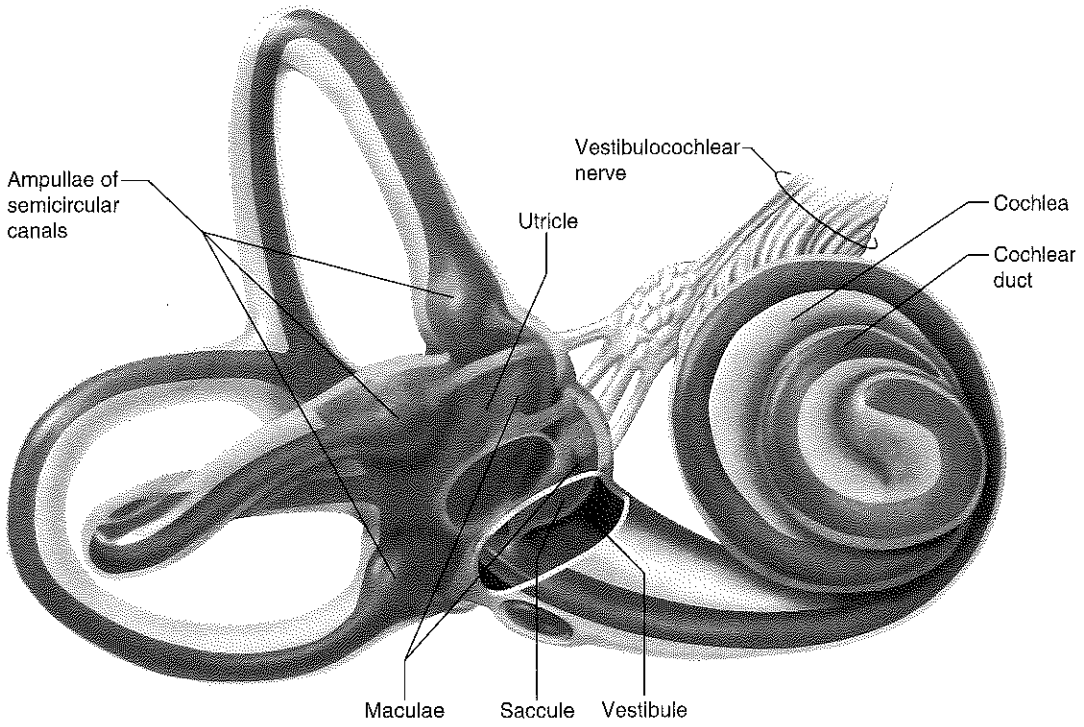


FIGURE 12.17 AP R The saccule and utricle, expanded portions of the membranous labyrinth, are in the bony chamber of the vestibule. (Compare with figure 12.11.)

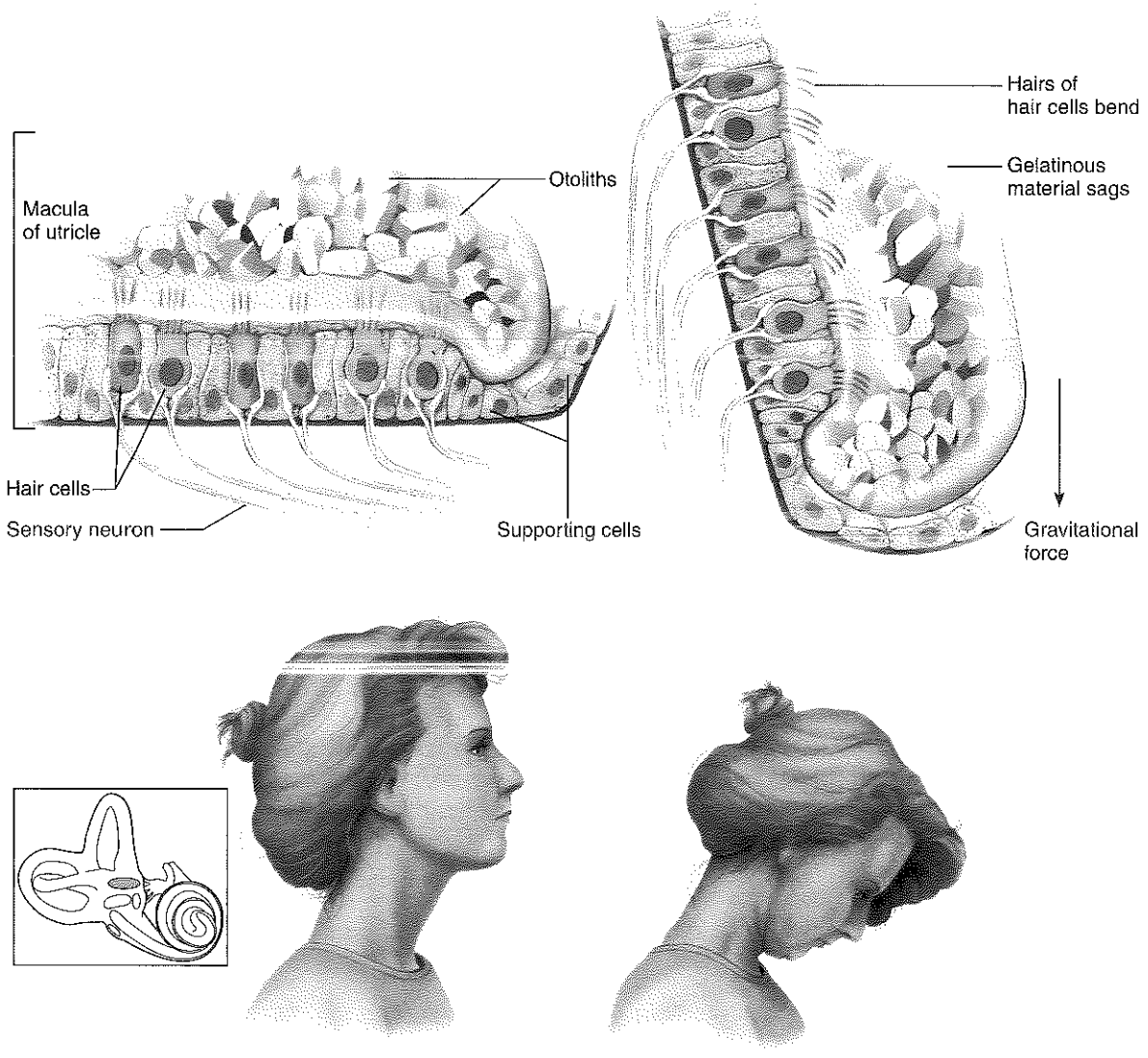


FIGURE 12.18 The maculae respond to changes in head position. (*a*) Macula of the utricle with the head in an upright position. (*b*) Macula of the utricle with the head bent forward.

When the head is upright, the hairs of the macula in the utricle project vertically, while those in the saccule project horizontally. In both the utricle and saccule, the hairs contact a sheet of gelatinous material (otolithic membrane) that has crystals of calcium carbonate (otoliths) embedded on its surface. These particles add weight to the gelatinous sheet, making it more responsive to changes in position. The hair cells, which are the sensory receptors, have dendrites of sensory neurons wrapped around their bases. These neurons are associated with the vestibular portion of the vestibulocochlear nerve.

Gravity stimulates hair cells to respond. This usually happens when the head bends forward, backward, or to one side. Such movements tilt the gelatinous mass of one or more maculae, and as the material sags in response to gravity, the hairs projecting into it bend. This action stimulates the hair cells, and they signal their associated neurons (figs. 12.18 and 12.19). The resulting impulses are conducted into the CNS by means of the vestibular branch of the vestibulocochlear nerve, informing the brain of the head's position. The brain responds

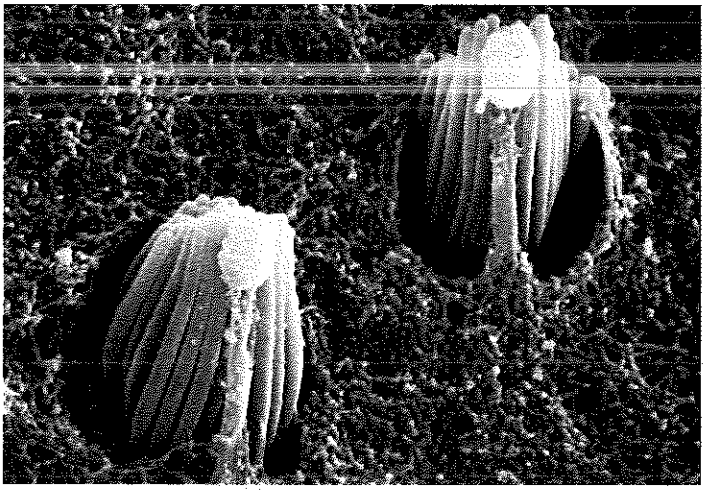


FIGURE 12.19 Scanning electron micrograph of hairs of hair cells, such as those in the utricle and saccule (8,000×).

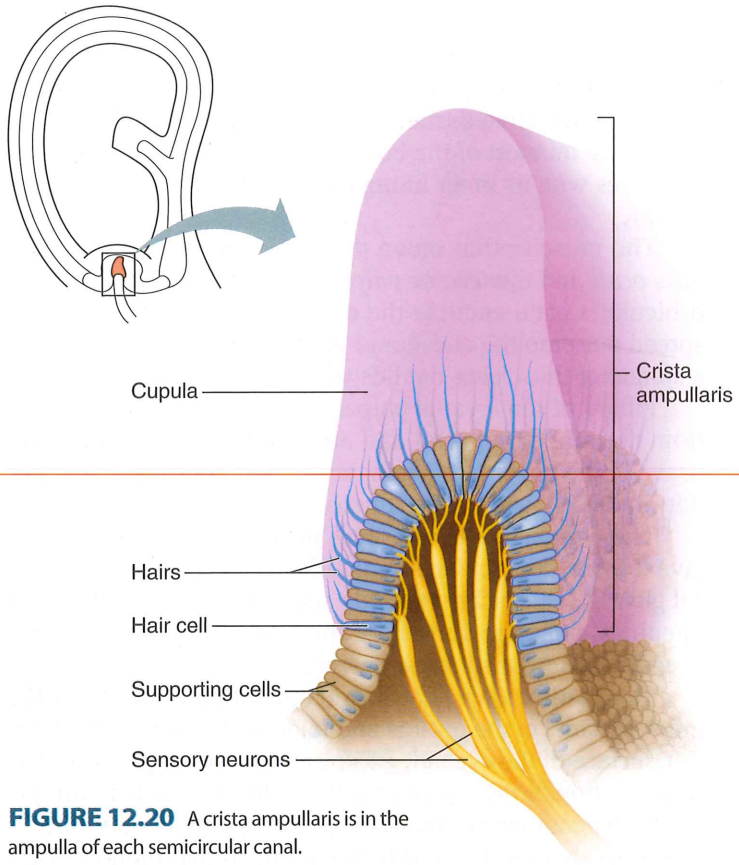


FIGURE 12.20 A crista ampullaris is in the ampulla of each semicircular canal.

by sending motor impulses to skeletal muscles, which may contract or relax appropriately to maintain balance.

The maculae also participate in the sense of dynamic equilibrium. For example, if the head or body is thrust forward or backward abruptly, the gelatinous mass of the maculae lags slightly behind, and the hair cells are stimulated. In this way, the maculae aid the brain in detecting movements such as falling and in maintaining posture while walking.

Dynamic Equilibrium

Each semicircular canal follows a circular path about 6 millimeters in diameter. The three bony semicircular canals lie at right angles to each other and occupy three different planes in space. Two of them, the *anterior canal* and the *posterior canal,* stand vertically, whereas the third, the *lateral canal,* is horizontal. Their orientations closely approximate the three body planes (see chapter 1, p. 30).

Suspended in the perilymph of each bony canal is a membranous semicircular canal that ends in a swelling called an **ampulla** (am-pul'ah). The ampullae communicate with the utricle of the vestibule.

An ampulla contains a septum that crosses the tube and houses a sensory organ. Each of these organs, called a **crista ampullaris,** has a number of sensory hair cells and supporting cells. As in the maculae, the hairs of the hair cells extend upward into a dome-shaped gelatinous mass called the *cupula.* Also, the hair cells are connected at their bases to dendrites of neurons that make up part of the vestibular branch of the vestibulocochlear nerve (fig. 12.20).

When the head or torso moves, the semicircular canals move as well, but initially the fluid inside the membranous

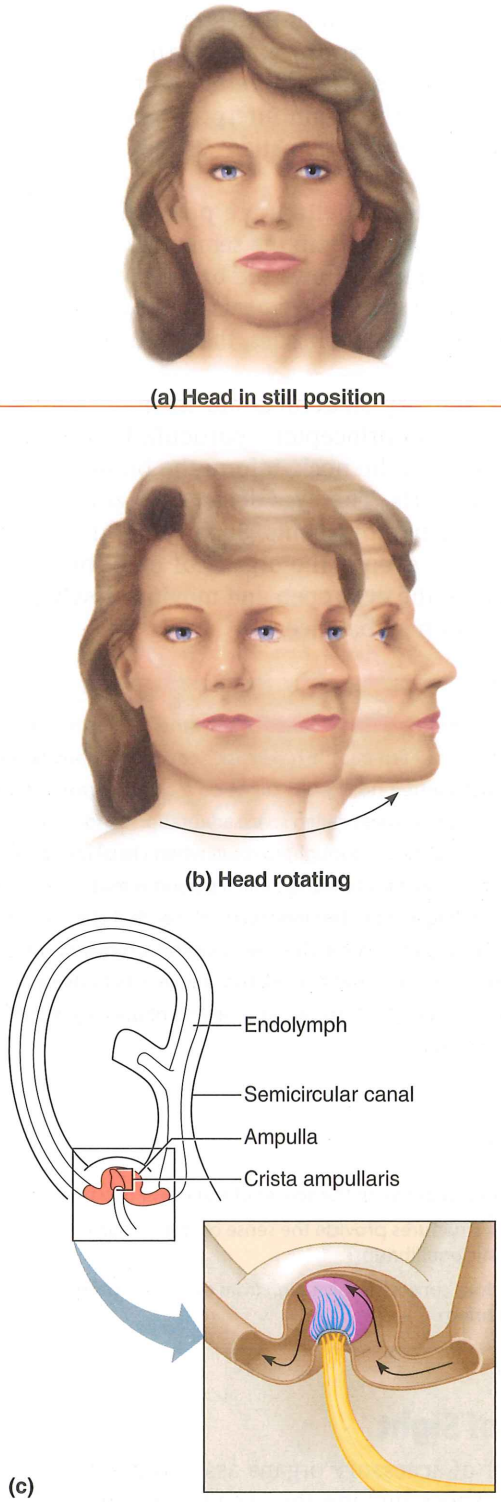


FIGURE 12.21 Equilibrium. (*a*) When the head is stationary, the cupula of the crista ampullaris remains upright. (*b*) When the head is moving rapidly, (*c*) the cupula bends opposite the motion of the head, stimulating sensory receptors.

canals tends to remain stationary because of inertia. This bends the cupula in one or more of the canals in a direction opposite that of the head and torso movement, and the hairs embedded in it also bend. The moving of the hairs stimulates the hair cells to signal their associated neurons, and as a result, impulses are conducted to the brain (fig. 12.21).

Because the orientation of the semicircular canals approximates the three anatomical planes, movements in different directions affect different combinations of semicircular canals. The brain interprets impulses originating from these different combinations as movements in different directions.

Parts of the cerebellum are particularly important in interpreting impulses from the semicircular canals. Analysis of such information allows the brain to predict the consequences of rapid body movements, and by modifying signals to appropriate skeletal muscles, the cerebellum can maintain balance.

Other sensory structures aid in maintaining equilibrium. Various proprioceptors, particularly those associated with the joints of the neck, inform the brain about the position of body parts. The eyes detect changes in posture that result from body movements. Such visual information is so important that even if the organs of equilibrium are damaged, keeping the eyes open and moving slowly is sufficient to maintain normal balance.

Motion sickness is a disturbance of the inner ear's sensation of balance. Nine out of ten people have experienced this nausea and vomiting, usually when riding in a car or on a boat. Astronauts suffer a form of motion sickness called space adaptation syndrome.

Motion sickness is thought to result when visual information contradicts the inner ear's sensation that a person is motionless. Consider a woman riding in a car. Her inner ears tell her that she is not moving, but the passing scenery tells her eyes that she is moving. The problem is compounded if she tries to read. The brain reacts to these seemingly contradictory sensations by causing a feeling of queasiness, which may lead to vomiting.

PRACTICE

26 Distinguish between the senses of static and dynamic equilibrium.

27 Which structures provide the sense of static equilibrium? of dynamic equilibrium?

28 How does sensory information from other receptors help maintain equilibrium?

Sense of Sight

A number of accessory organs assist the visual receptors in the eyes. These include the eyelids and lacrimal apparatus that help protect the eyes and a set of extrinsic muscles that move them.

Visual Accessory Organs

Each eye, lacrimal gland, and associated extrinsic muscles are housed in the orbital cavity of the skull. The orbit, lined with the periosteum of various bones, also contains fat, blood vessels, nerves, and connective tissues.

Each **eyelid** (palpebra) is composed of four layers—skin, muscle, connective tissue, and conjunctiva. The skin of the eyelid, the thinnest of the body, covers the lid's outer surface and fuses with its inner lining near the margin of the lid (fig. 12.22).

The muscles that move the eyelids include the *orbicularis oculi* and the *levator palpebrae superioris*. Fibers of the orbicularis oculi encircle the opening between the lids and spread out onto the cheek and forehead. This muscle acts as a sphincter that closes the lids when it contracts.

Fibers of the levator palpebrae superioris muscle arise from the roof of the orbit and are inserted in the connective tissue of the upper lid. When these fibers contract, the upper lids are raised, and the eye opens.

The connective tissue layer of the eyelid, which helps give it form, contains many modified sebaceous glands (tarsal glands). Ducts carry the oily secretions of these glands to openings along the borders of the lids. This secretion helps keep the lids from sticking together.

The **conjunctiva** is a mucous membrane that lines the inner surfaces of the eyelids and folds back to cover the anterior surface of the eyeball, except for its central portion (cornea). Although the tissue that lines the eyelids is relatively thick, the conjunctiva that covers the eyeball is very thin. It is also freely movable and transparent, so that blood vessels are clearly visible beneath it.

Most children who arrive at school with "pinkeye" are sent home. Pinkeye is a form of *conjunctivitis*, or inflammation of the conjunctiva. When caused by bacteria, it is highly contagious. It is not usually contagious when caused by a virus.

The **lacrimal apparatus** consists of the *lacrimal gland*, which secretes tears, and a series of *ducts*, which carry the tears into the nasal cavity (fig. 12.23). The gland is in the orbit, superior and lateral to the eye. It secretes tears continuously, which pass through tiny tubules and flow downward and medially across the eye.

Two small ducts (superior and inferior canaliculi) collect tears, and their openings (puncta) can be seen on the medial borders of the eyelids. From these ducts, the fluid moves into the *lacrimal sac*, which lies in a deep groove of the lacrimal bone, and then into the *nasolacrimal duct*, which empties into the nasal cavity.

Glandular cells of the conjunctiva also secrete a tearlike liquid that, together with the secretion of the lacrimal gland, moistens and lubricates the surface of the eye and the lining of the lids. Tears contain an enzyme, *lysozyme*, that has antibacterial properties, reducing the risk of eye infections.

Tear glands secrete excessively when a person is upset or when the conjunctiva is irritated. Tears spill over the edges of the eyelids, and the nose fills with fluid. When a person cries, parasympathetic nerve fibers carry motor impulses to the lacrimal glands.

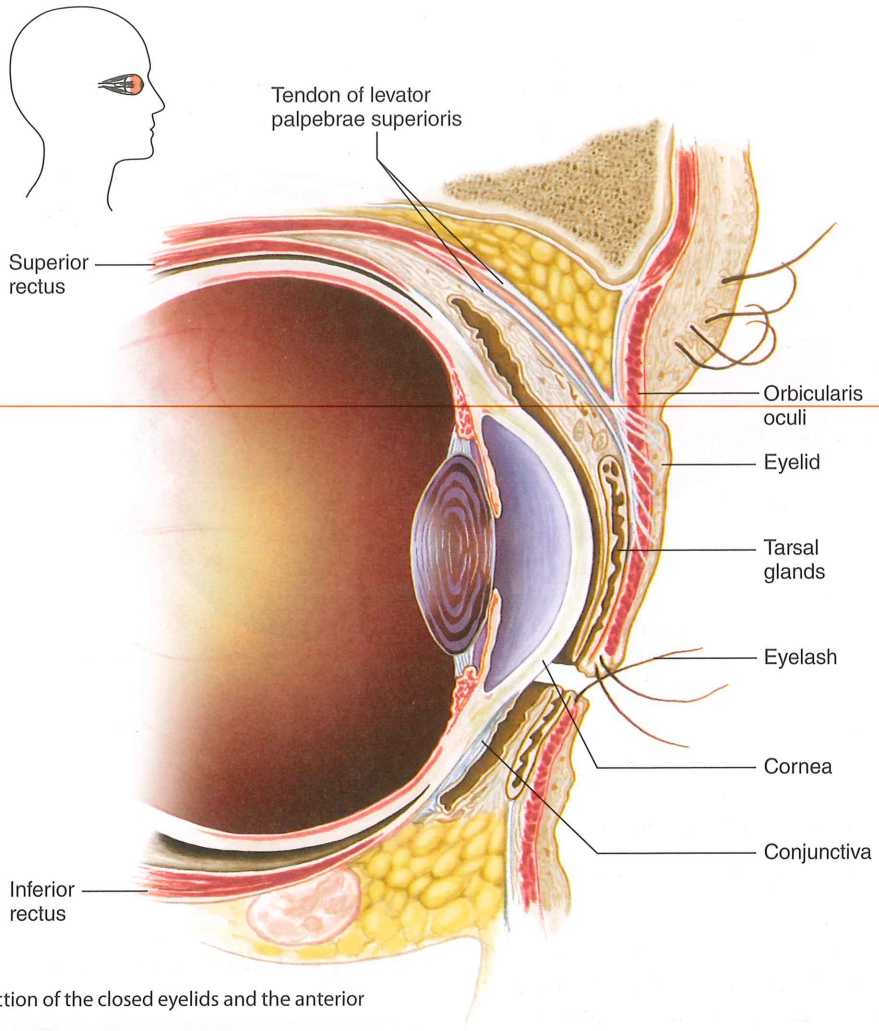


FIGURE 12.22 Sagittal section of the closed eyelids and the anterior portion of the eye.

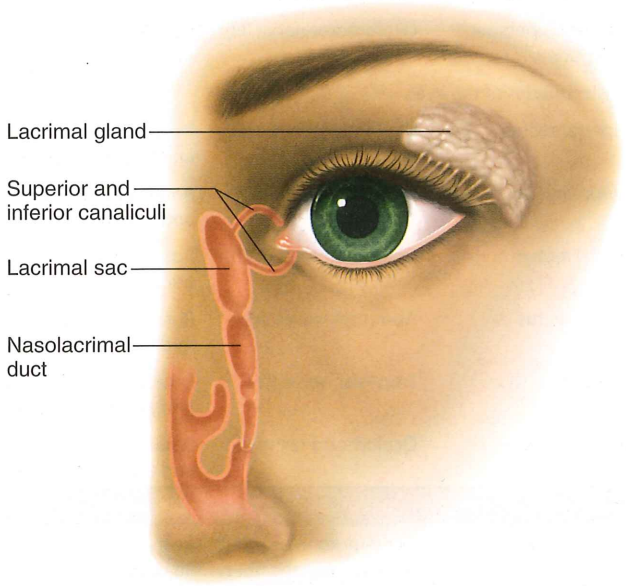


FIGURE 12.23 The lacrimal apparatus consists of a tear secreting gland and a series of ducts.

The **extrinsic muscles** of the eye arise from the bones of the orbit and are inserted by broad tendons on the eye's tough outer surface. Six such muscles move the eye in various directions (fig. 12.24). Although any given eye movement may use more than one muscle, each one is associated with one primary action, as follows:

1. **Superior rectus**—rotates the eye upward and toward the midline.
2. **Inferior rectus**—rotates the eye downward and toward the midline.
3. **Medial rectus**—rotates the eye toward the midline.
4. **Lateral rectus**—rotates the eye away from the midline.
5. **Superior oblique**—rotates the eye downward and away from the midline.
6. **Inferior oblique**—rotates the eye upward and away from the midline.

The motor units of the extrinsic eye muscles have the fewest muscle fibers (five to ten) of any muscles in the body, enabling them to move the eyes with great precision. Also,

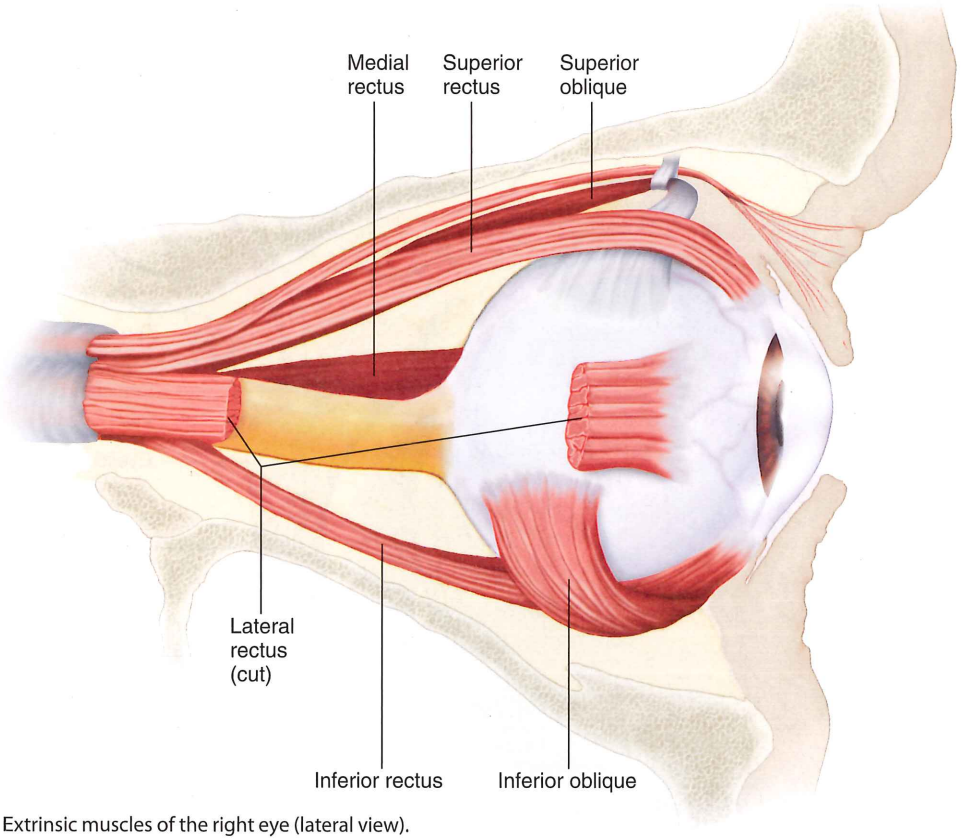


FIGURE 12.24 AP|R Extrinsic muscles of the right eye (lateral view).

Q: *Are the extrinsic muscles of the eye under voluntary control?*

Answer can be found in Appendix G on page 938.

the eyes move together so that they align when looking at something. Such alignment is the result of complex motor adjustments that contract certain eye muscles while relaxing their antagonists. For example, when the eyes move to the right, the lateral rectus of the right eye and the medial rectus of the left eye must contract. At the same time, the medial rectus of the right eye and the lateral rectus of the left eye must relax. A person whose eyes are not coordinated well enough to align has *strabismus*. Table 12.5 summarizes the muscles associated with the eyelids and eye.

One eye deviating from the line of vision produces double vision (diplopia). If it persists, the brain may eventually suppress the image from the deviated eye, and the turning eye may experience partial vision loss (suppression amblyopia). Treating the eye deviation early in life with exercises, eyeglasses, and surgery can prevent such monocular vision loss. Vision screening programs for children can detect this problem.

PRACTICE

29 Explain how the eyelid is moved.

30 Describe the conjunctiva.

31 What is the function of the lacrimal apparatus?

32 Describe the function of each extrinsic eye muscle.

TABLE 12.5 | Muscles Associated with the Eyelids and Eyes

Skeletal Muscles	Innervation	Function
Muscles of the eyelids		
Orbicularis oculi	Facial nerve (VII)	Closes eye
Levator palpebrae superioris	Oculomotor nerve (III)	Opens eye
Extrinsic muscles of the eyes		
Superior rectus	Oculomotor nerve (III)	Rotates eye upward and toward midline
Inferior rectus	Oculomotor nerve (III)	Rotates eye downward and toward midline
Medial rectus	Oculomotor nerve (III)	Rotates eye toward midline
Lateral rectus	Abducens nerve (VI)	Rotates eye away from midline
Superior oblique	Trochlear nerve (IV)	Rotates eye downward and away from midline
Inferior oblique	Oculomotor nerve (III)	Rotates eye upward and away from midline
Smooth Muscles	**Innervation**	**Function**
Ciliary muscles	Oculomotor nerve (III) parasympathetic fibers	Relax suspensory ligaments
Iris, circular muscles	Oculomotor nerve (III) parasympathetic fibers	Constrict pupil
Iris, radial muscles	Sympathetic fibers	Dilate pupil

Structure of the Eye

The eye is a hollow, spherical structure about 2.5 centimeters in diameter. Its wall has three distinct layers—an outer *fibrous tunic,* a middle *vascular tunic,* and an inner *nervous tunic.* The spaces in the eye are filled with fluids that support its wall and internal structures that help maintain its shape. Figure 12.25 shows the major parts of the eye.

The Outer Tunic

The anterior sixth of the outer tunic bulges forward as the transparent **cornea** (kor′ne-ah), which is the window of the eye that helps focus entering light rays. It is largely composed of connective tissue with a thin surface layer of epithelium. The cornea is transparent because it contains no blood vessels and the collagenous fibers form unusually regular patterns.

The cornea is well supplied with sensory nerve fibers that enter its margin and radiate toward its center. These fibers are associated with many pain receptors that have very low thresholds. Cold receptors are also abundant in the cornea, but heat and touch receptors are not.

> The most common cause of blindness worldwide is loss of transparency of the cornea. Each year, 40,000 corneal transplants are performed in the United States. Corneal transplants do not evoke an immune response, but they only work if the transplanted tissue includes stem cells that are normally found in a cell layer, called the limbus, which separates the cornea from the conjunctiva. (The cornea itself does not contain stem cells—their nuclei are so large that they would block light rays, impairing vision.)

The cornea is continuous with the **sclera** (skle′rah), the white portion of the eye, along its circumference. The sclera makes up the posterior five-sixths of the outer tunic and is opaque due to many large, seemingly disorganized collagenous and elastic fibers. The sclera protects the eye and is an attachment for the extrinsic muscles.

In the back of the eye, the **optic** (op′tik) **nerve** and blood vessels pierce the sclera. The dura mater that encloses these structures is continuous with the sclera.

The Middle Tunic

The middle, or vascular, tunic of the eyeball (uveal layer) includes the **choroid coat,** the ciliary body, and the iris. The choroid coat, in the posterior five-sixths of the globe of the eye, loosely joins the sclera. Blood vessels pervade the choroid coat and nourish surrounding tissues. The choroid coat also contains abundant pigment-producing melanocytes that give it a brownish-black appearance. The melanin of these cells absorbs excess light and helps keep the inside of the eye dark.

The **ciliary body,** which is the thickest part of the middle tunic, extends forward from the choroid coat and forms an internal ring around the front of the eye. In the ciliary body are many radiating folds called *ciliary processes* and two distinct groups of muscle fibers that constitute the *ciliary muscles.* Figure 12.26 shows these structures.

Many strong but delicate fibers, called *suspensory ligaments* (zonular fibers), extend inward from the ciliary processes and hold the transparent **lens** in position. The distal ends of these fibers are attached along the margin of a thin capsule that surrounds the lens. The body of the lens, which

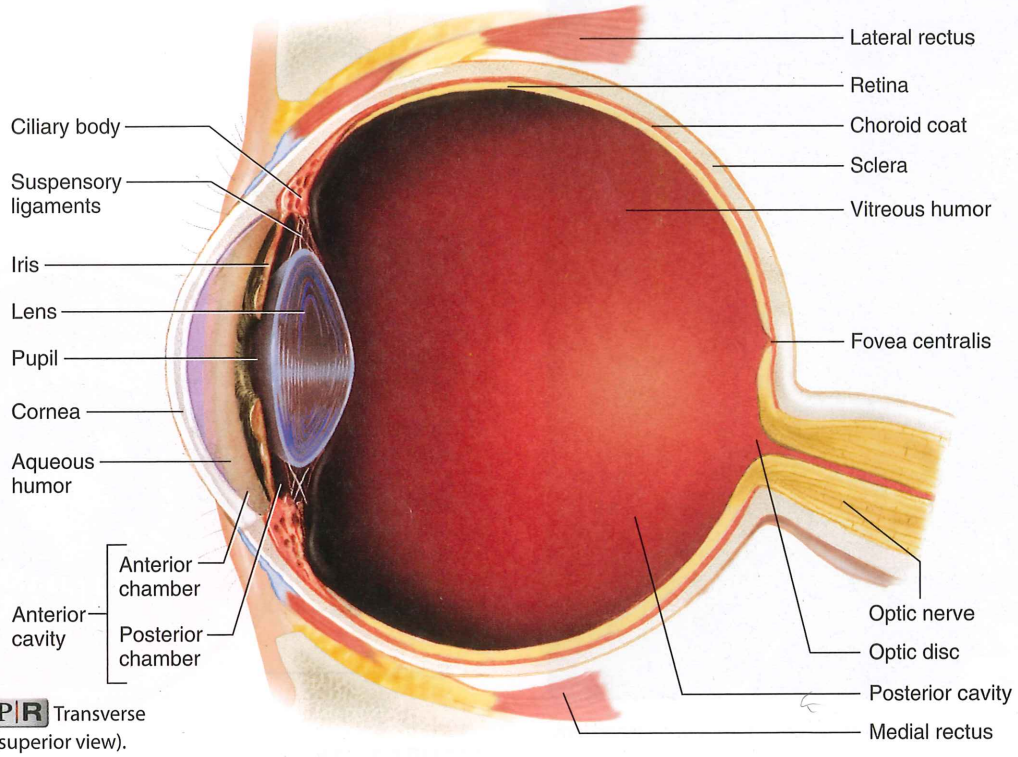


FIGURE 12.25 AP|R Transverse section of the right eye (superior view).

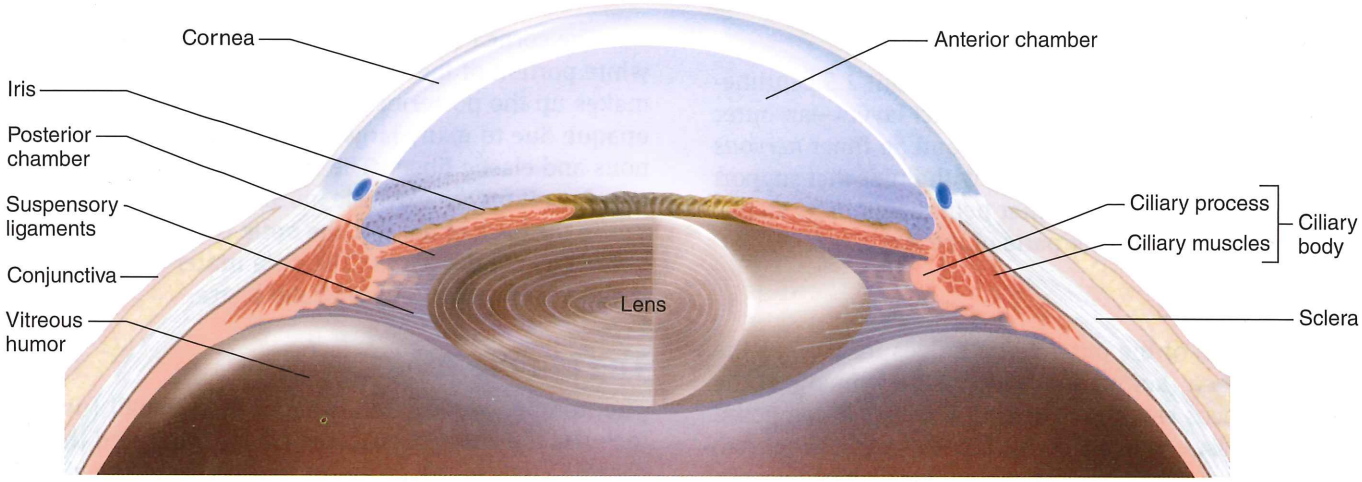


FIGURE 12.26 AP|R Anterior portion of the eye.

lacks blood vessels, lies directly behind the iris and pupil and is composed of specialized epithelial cells.

The cells of the lens originate from a single layer of epithelium beneath the anterior portion of the lens capsule. The cells divide, and the new cells on the surface of the lens capsule differentiate into specialized columnar epithelial cells called *lens fibers,* which constitute the substance of the lens. Lens fiber production continues slowly throughout life, thickening the lens from front to back. Simultaneously, the deeper lens fibers are compressed toward the center of the structure (fig. 12.27). More than 90% of the proteins in a lens cell are lens crystallins, which aggregate into the fibers. These proteins, along with the absence of organelles that scatter light (mitochondria, endoplasmic reticula, and nuclei), provide the transparency of the lens.

The lens capsule is a clear, membranelike structure largely composed of intercellular material (fig. 12.28). It is quite elastic, a quality that keeps it under constant tension. As a result, the lens can assume a globular shape. However, the suspensory ligaments attached to the margin of the capsule are also under tension, and they pull outward, flattening the capsule and the lens.

If the tension on the suspensory ligaments relaxes, the elastic capsule rebounds, and the lens surface becomes more convex. This change, called **accommodation** (ah-kom″o-da′shun), occurs in the lens when the eye focuses to view a close object.

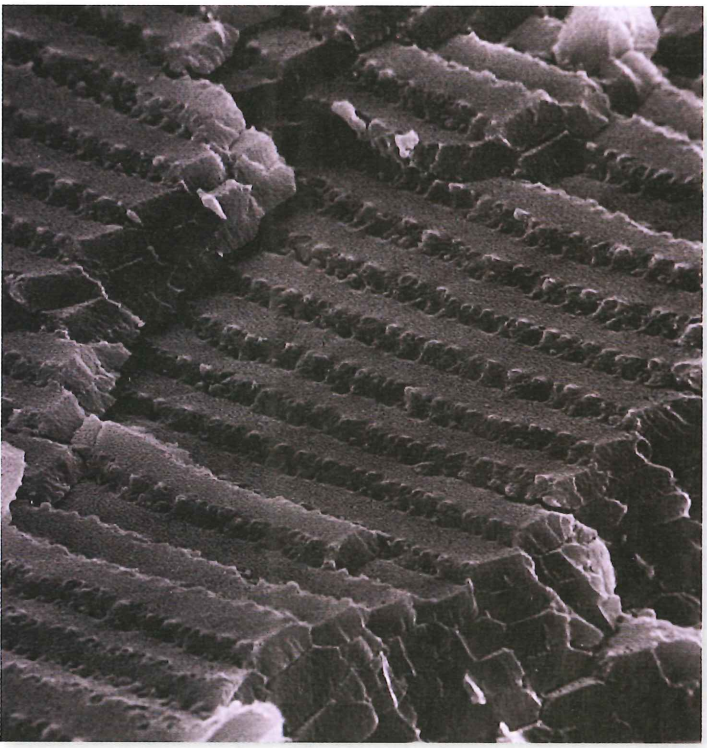


FIGURE 12.27 A scanning electron micrograph of the long, flattened lens fibers (2,650×). Note the fingerlike junctions where one fiber joins another.

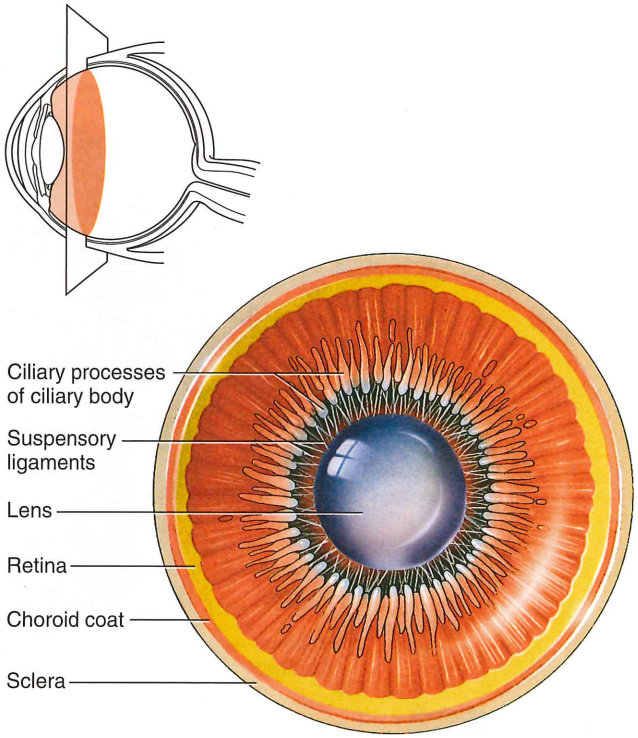


FIGURE 12.28 Lens and ciliary body viewed from behind.

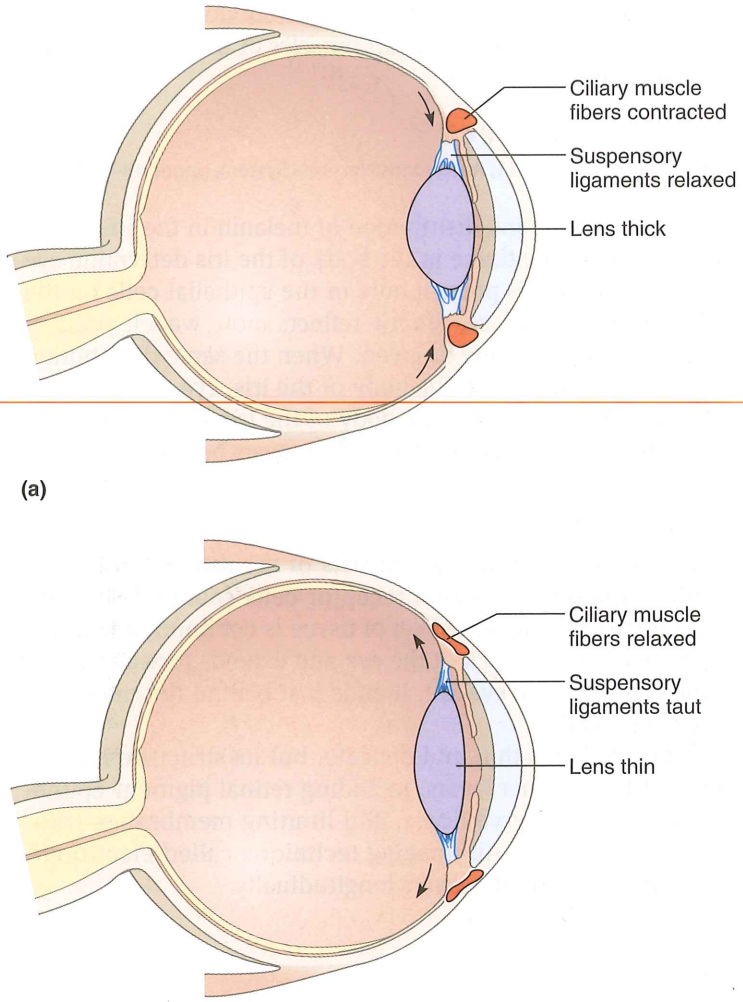


FIGURE 12.29 In accommodation, (*a*) the lens thickens as the ciliary muscle fibers contract. (*b*) The lens thins as ciliary muscle fibers relax.

The ciliary muscles relax the suspensory ligaments during accommodation. One set of these muscle fibers forms a circular sphincterlike structure around the ciliary processes. The fibers of the other set extend back from fixed points in the sclera to the choroid coat. When the circular muscle fibers contract, the diameter of the ring formed by the ciliary processes decreases; when the other fibers contract, the choroid coat is pulled forward, and the ciliary body shortens. Both of these actions relax the suspensory ligaments, thickening the lens. In this thickened state, the lens is focused for viewing objects closer than before (fig. 12.29*a*).

To focus on a distant object, the ciliary muscles relax, increasing tension on the suspensory ligaments. The lens thins again (fig. 12.29*b*).

PRACTICE

33 Describe the outer and middle tunics of the eye.

34 What factors contribute to the transparency of the cornea?

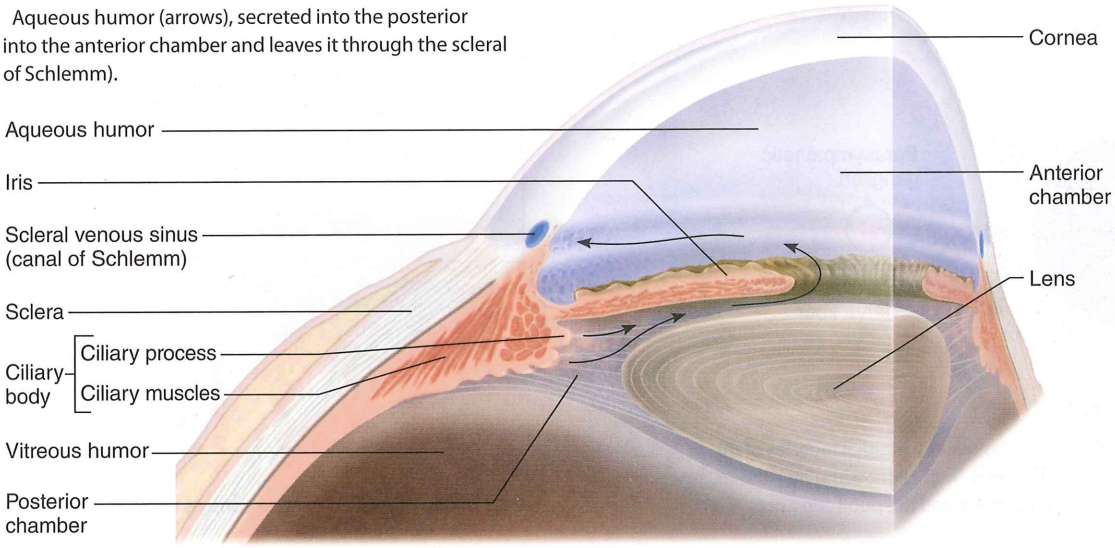
35 How does the shape of the lens change during accommodation?

36 Why would reading for a long time lead to "eye fatigue," whereas looking at something distant is restful?

The **iris** is a thin diaphragm mostly composed of connective tissue and smooth muscle fibers. Seen from the outside, it is the colored portion of the eye. The iris extends forward from the periphery of the ciliary body and lies between the cornea and the lens. It divides the space separating these parts, called the *anterior cavity,* into an *anterior chamber* (between the cornea and the iris) and a *posterior chamber* (between the iris and the vitreous humor, occupied by the lens).

The epithelium on the inner surface of the ciliary body continuously secretes a watery fluid called **aqueous humor** into the posterior chamber. The fluid circulates from this chamber through the **pupil,** which is a circular opening in the center of the iris, and into the anterior chamber (fig. 12.30). Aqueous humor fills the space between the cornea and the lens, providing nutrients and maintaining the shape of the front of the eye. It leaves the anterior chamber through veins

FIGURE 12.30 Aqueous humor (arrows), secreted into the posterior chamber, circulates into the anterior chamber and leaves it through the scleral venous sinus (canal of Schlemm).



and a special drainage canal, called the scleral venous sinus (canal of Schlemm), in the wall of the anterior chamber at the junction of the cornea and the sclera (fig. 12.30).

The smooth muscle fibers of the iris form two groups, a *circular set* and a *radial set*. These muscles control the size of the pupil, through which light passes. The circular set of muscle fibers acts as a sphincter, and when it contracts, the pupil gets smaller (constricts) and the intensity of the light entering decreases. When the radial muscle fibers contract, the diameter of the pupil increases (dilates) and the intensity of the light entering increases.

The sizes of the pupils change constantly in response to pupillary reflexes triggered by such factors as light intensity, gaze, accommodation, and variations in emotional state. For example, bright light elicits a reflex, and impulses are conducted along parasympathetic nerve fibers to the *circular muscles* of the irises. The pupils constrict in response. Conversely, in dim light, impulses are conducted on sympathetic nerve fibers to the *radial muscles* of the irises, and the pupils dilate (fig. 12.31).

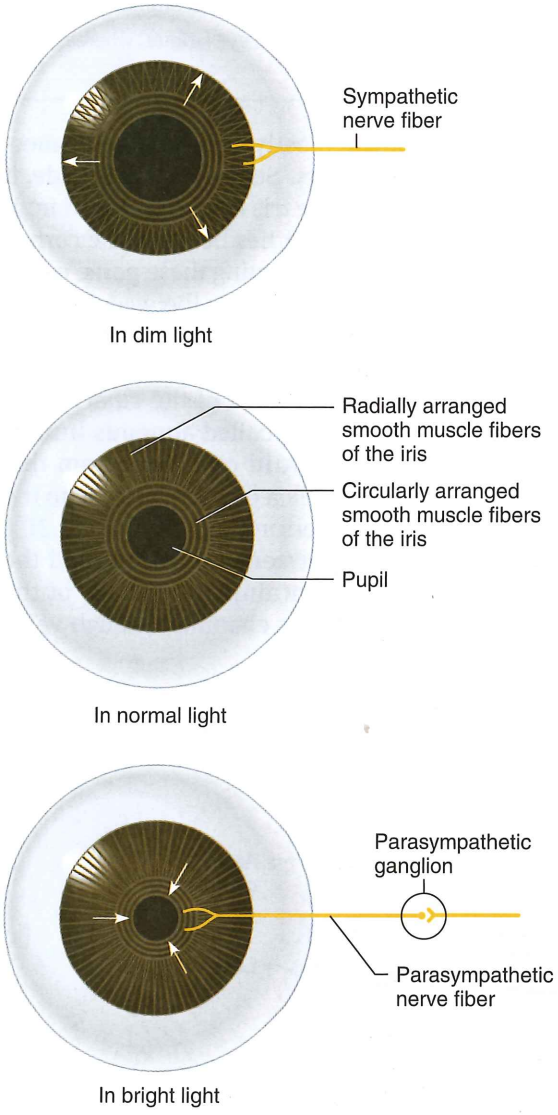


FIGURE 12.31 Dim light stimulates the radial muscles of the iris to contract, and the pupil dilates. Bright light stimulates the circular muscles of the iris to contract, and the pupil constricts.

RECONNECT

To Chapter 11, Autonomic Nervous System, pages 434–435.

The amount and distribution of melanin in the irises and the density of the tissue in the body of the iris determine eye color. If melanin is present only in the epithelial cells on the iris's posterior surface, the iris reflects more wavelengths of light, and appears blue or green. When the same distribution of melanin is denser in the body of the iris, eye color is gray. When melanin is within the body of the iris as well as in the posterior epithelial covering, the iris appears brown.

The Inner Tunic

The inner tunic of the eye consists of the **retina** (ret'ĭ-nah), which contains the visual receptor cells (photoreceptors). This nearly transparent sheet of tissue is continuous with the optic nerve in the back of the eye and extends forward as the inner lining of the eyeball. It ends just behind the margin of the ciliary body.

The retina is thin and delicate, but its structure is complex. It has distinct layers, including retinal pigment epithelium, neurons, nerve fibers, and limiting membranes (figs. 12.32 and 12.33). An imaging technique called electroretinography displays the layers longitudinally.

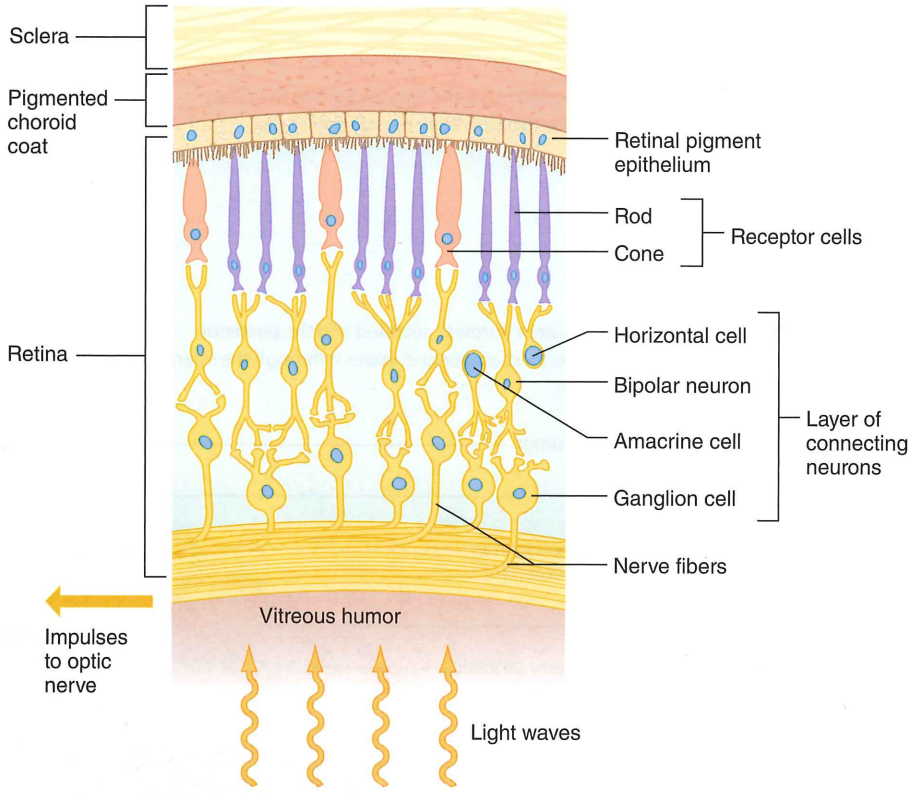


FIGURE 12.32 The retina consists of several cell layers.

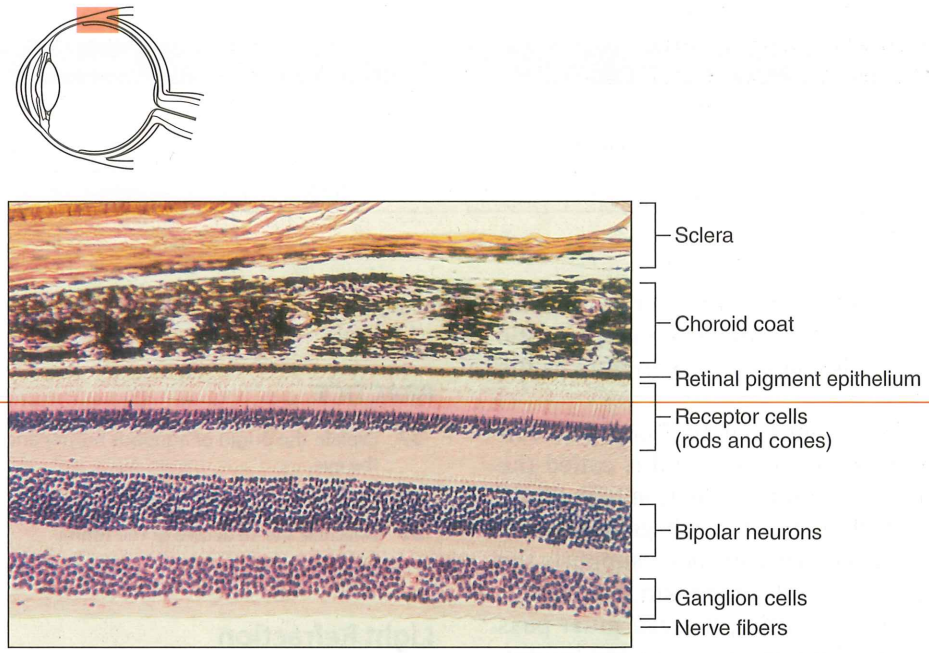
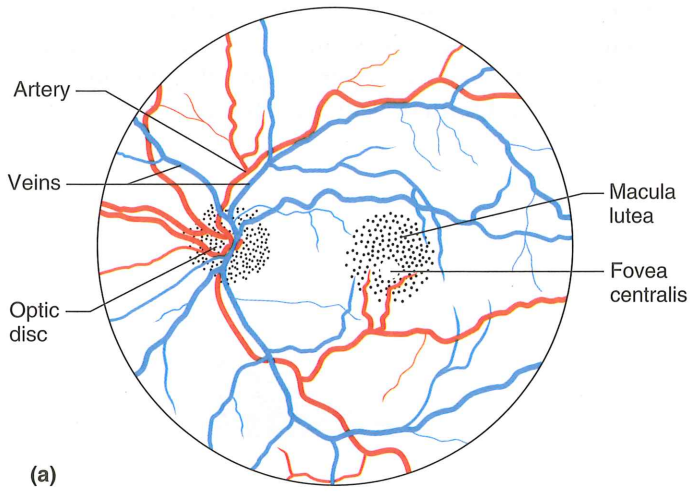


FIGURE 12.33 AP|R Note the layers of cells and nerve fibers in this light micrograph of the retina (75×).

There are five major groups of retinal neurons. The nerve fibers of three of these groups—the *receptor cells, bipolar neurons,* and *ganglion cells*—provide a direct pathway for impulses triggered in the photoreceptors to the optic nerve and brain. The nerve fibers of the other two groups of retinal cells, called *horizontal cells* and *amacrine cells,* pass laterally between retinal cells (see fig. 12.32). The horizontal and amacrine cells modify the impulses conducted on the fibers of the direct pathway.

In the central region of the retina is a yellowish spot called the **macula lutea** that occupies about 1 square millimeter. A depression in its center, called the **fovea centralis,** is in the region of the retina that produces the sharpest vision.

Just medial to the fovea centralis is an area called the **optic disc** (fig. 12.34). Here the nerve fibers from the retina leave the eye and become parts of the optic nerve. A central artery and vein also pass through at the optic disc. These vessels are continuous with capillary networks of the retina,



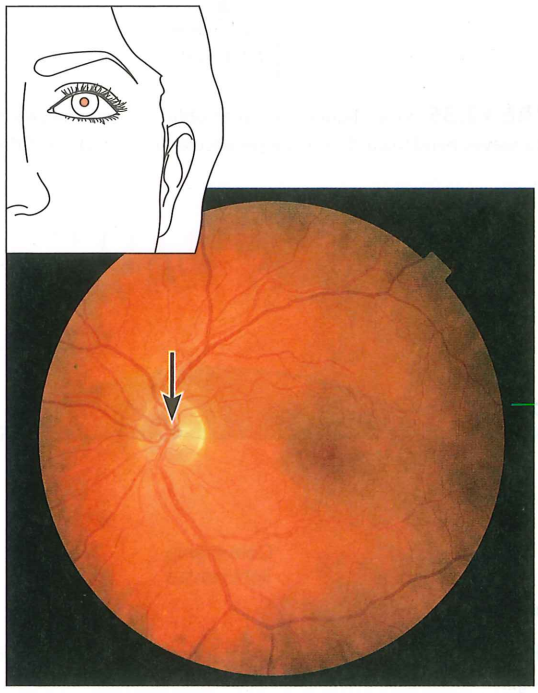


FIGURE 12.34 AP|R The retina. (*a*) Major features of the retina. (*b*) Nerve fibers leave the retina in the area of the optic disc (arrow) to form the optic nerve in this magnified view (53×).

TABLE 12.6 | Layers of the Eye

Layer/Tunic	Posterior Portion	Function	Anterior Portion	Function
Outer layer	Sclera	Protection	Cornea	Light transmission and refraction
Middle layer	Choroid coat	Blood supply, pigment prevents reflection	Ciliary body, iris	Accommodation; controls light intensity
Inner layer	Retina	Photoreception, impulse conduction	None	

and together with vessels in the underlying choroid coat, they supply blood to the cells of the inner tunic. The optic disc lacks receptor cells, so it is commonly referred to as the *blind spot* of the eye.

The space enclosed by the lens, ciliary body, and retina is the largest compartment of the eye and is called the *posterior cavity.* It is filled with a transparent, jellylike fluid called **vitreous humor,** which with some collagenous fibers comprise the **vitreous body.** The vitreous body supports the internal structures of the eye and helps maintain its shape.

In summary, light waves entering the eye must pass through the cornea, aqueous humor, lens, vitreous humor, and several layers of the retina before they reach the photoreceptors (see fig. 12.32). Table 12.6 summarizes the layers of the eye.

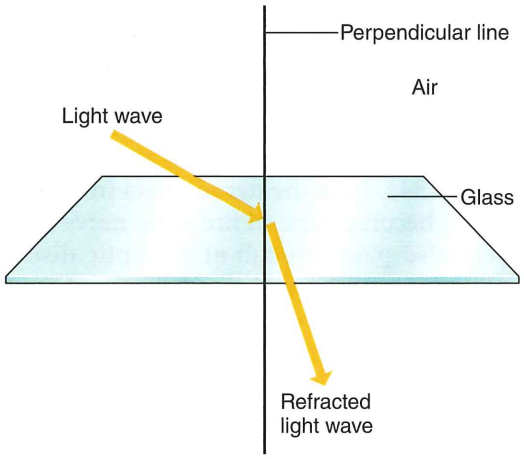


FIGURE 12.35 When light passes at an oblique angle from air into glass, the light waves bend toward a line perpendicular to the surface of the glass.

PRACTICE

37 Explain the origin of aqueous humor and trace its path through the eye.

38 How is the size of the pupil regulated?

39 Describe the structure of the retina.

Light Refraction

When a person sees an object, either the object is giving off light, or it is reflecting light waves from another source. These light waves enter the eye, and an image of what is seen focuses upon the retina. The light rays must bend to be focused, which is a phenomenon called **refraction** (re-frak'shun).

Refraction occurs when light waves pass at an oblique angle from a medium of one optical density into a medium of a different optical density. For example, as figure 12.35 shows, when light passes obliquely from a less-dense medium such as air into a denser medium such as glass, or from air into the cornea of the eye, the light is bent toward a line perpendicular to the surface between these substances. When the surface between such refracting media is curved, a lens is formed. A lens with a *convex* surface causes light waves to converge, and a lens with a *concave* surface causes light waves to diverge (fig. 12.36). Clinical Application 12.6 discusses some familiar visual problems resulting from abnormal refraction.

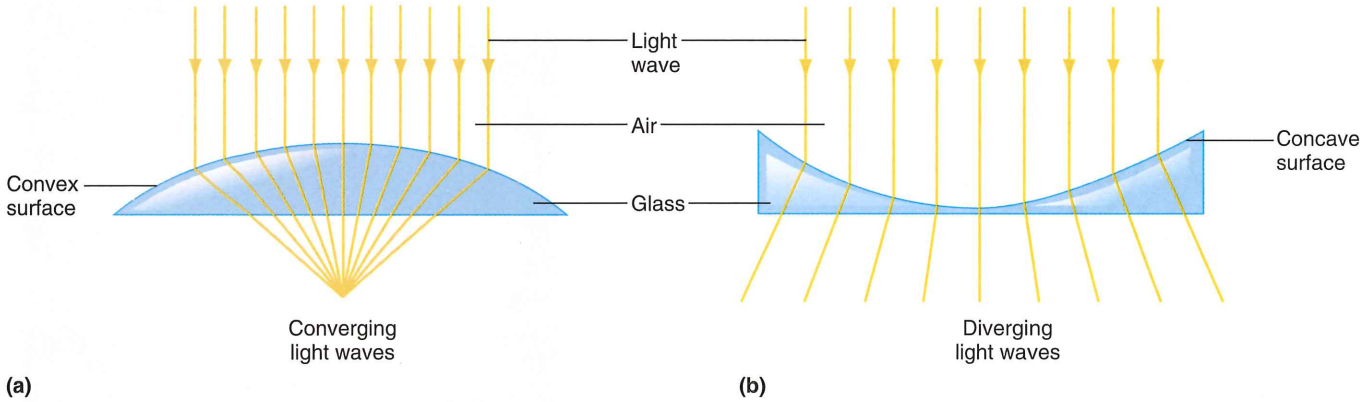


FIGURE 12.36 Light waves passing through a lens. (*a*) A lens with a convex surface causes light waves to converge. (*b*) A lens with a concave surface causes them to diverge.

12.6 CLINICAL APPLICATION

Refraction Disorders

For many people, after the age of forty-five, it seems as if the print in magazines and on medicine bottles suddenly becomes too small to read. The problem is not in the print, but in a lessening of the elastic quality of the lens capsule. In the condition presbyopia, or farsightedness of age, eyes remain focused for distant vision. Eyeglasses or contact lenses can usually make up for the eye's loss of refracting power.

Other visual problems result from eyeballs that are too long or too short for sharp focusing. If an eyeball is too long, light waves focus in front of the retina, blurring the image. In other words, the refracting power of the eye, even when the lens is flattened, is too great. Although a person with this problem may be able to focus on close objects by accommodation, distance vision is invariably poor. For this reason, the person is said to be *nearsighted*. Eyeglasses or contact lenses with concave surfaces that focus images farther from the front of the eye treat nearsightedness (myopia).

If an eye is too short, light waves are not focused sharply on the retina because their point of focus lies behind it. A person with this condition may be able to bring the image of distant objects into focus by accommodation, but this requires contraction of the ciliary muscles at times when these muscles are at rest in a normal eye. Still more accommodation is necessary to view closer objects, and the person may suffer from ciliary muscle fatigue, pain, and headache when doing close work.

Most people with short eyeballs are unable to accommodate enough to focus on the very close objects. They are said to be *farsighted*. Eyeglasses or contact lenses with *convex* surfaces can remedy this condition (hyperopia) by focusing images closer to the front of the eye (figs. 12E and 12F).

Another refraction problem, *astigmatism*, reflects a defect in the curvature of the cornea or the lens. The normal cornea has a spherical curvature, like the inside of a ball; most astigmatic corneas have an elliptical curvature, like the bowl of a spoon. As a result, some portions of an image are in focus on the retina, but other portions are blurred, and vision is distorted.

Without corrective lenses, astigmatic eyes tend to accommodate back and forth reflexly in an attempt to sharpen focus. The consequence of this continual action is often ciliary muscle fatigue and headache. ■

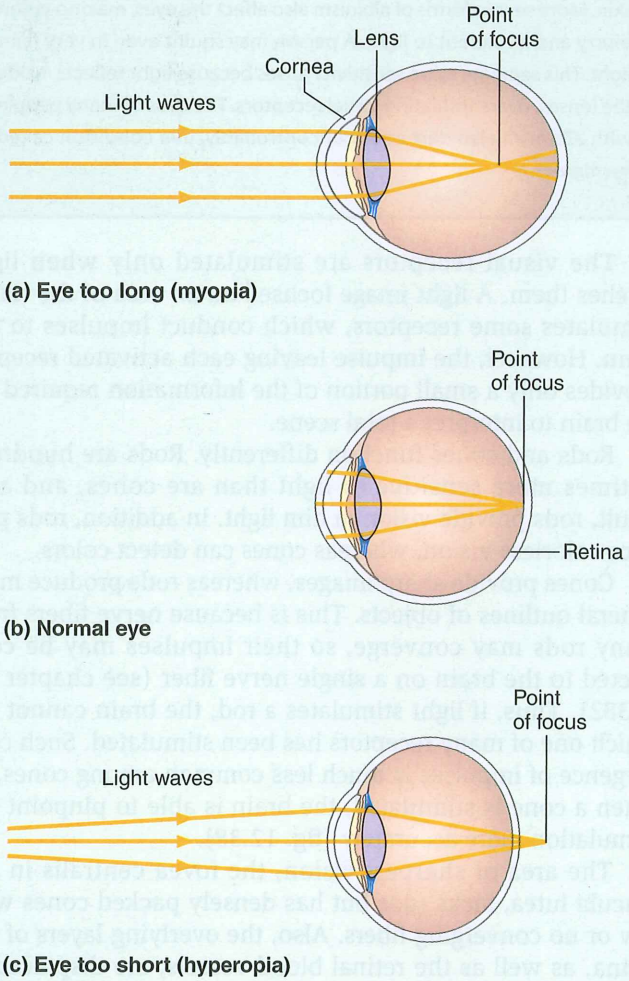


FIGURE 12E Point of focus. (*a*) Myopia (nearsightedness)—If an eye is too long, the focus point of images lies in front of the retina. (*b*) In a normal eye, the focus point is on the retina. (*c*) Hyperopia (farsightedness)—If an eye is too short, the focus point lies behind the retina.

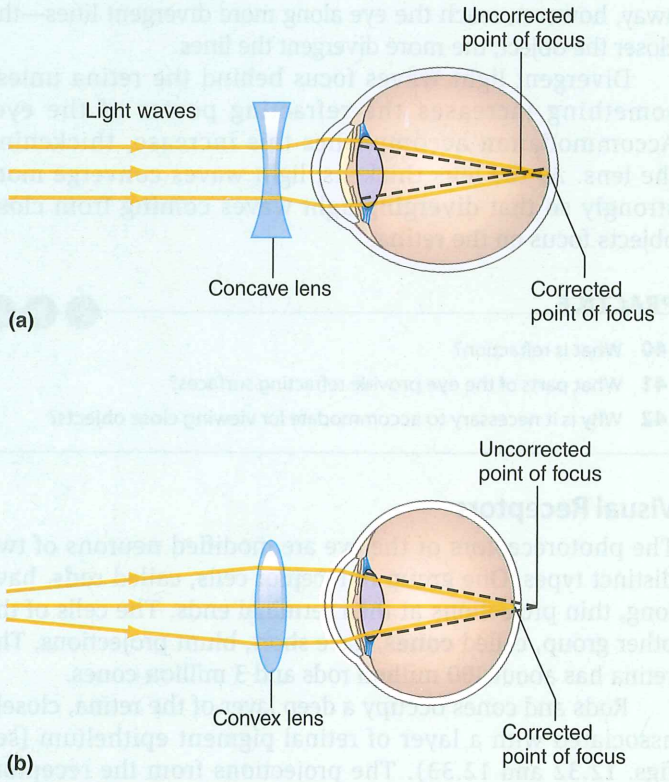


FIGURE 12F Corrective lenses. (*a*) A concave lens corrects nearsightedness. (*b*) A convex lens corrects farsightedness.

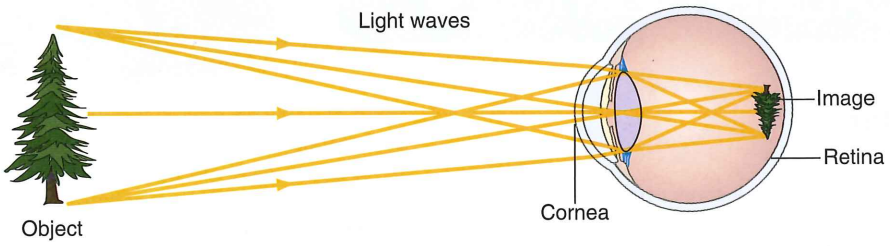


FIGURE 12.37 The image of an object forms upside down on the retina.

The convex surface of the cornea refracts light waves from objects outside the eye, providing about 75% of the total refractive power of the eye. The light is refracted again by the convex surface of the lens and to a lesser extent by the surfaces of the fluids in the eye chambers.

If the shape of the eye is normal, light waves are focused sharply upon the retina, much as a motion-picture image is focused on a screen for viewing. Unlike the motion-picture image, however, the one formed on the retina is upside down and reversed from left to right (fig. 12.37). When the visual cortex of the cerebrum interprets such an image, it corrects the reversals and objects are seen in their real positions.

Light waves coming from objects more than 20 feet away travel in nearly parallel lines, and the cornea and the lens in its more flattened or "at-rest" condition focuses the light waves on the retina. Light waves arriving from objects less than 20 feet away, however, reach the eye along more divergent lines—the closer the object, the more divergent the lines.

Divergent light waves focus behind the retina unless something increases the refracting power of the eye. Accommodation accomplishes this increase, thickening the lens. As the lens thickens, light waves converge more strongly so that diverging light waves coming from close objects focus on the retina.

PRACTICE

40 What is refraction?

41 What parts of the eye provide refracting surfaces?

42 Why is it necessary to accommodate for viewing close objects?

Visual Receptors

The photoreceptors of the eye are modified neurons of two distinct types. One group of receptor cells, called **rods,** have long, thin projections at their terminal ends. The cells of the other group, called **cones,** have short, blunt projections. The retina has about 100 million rods and 3 million cones.

Rods and cones occupy a deep layer of the retina, closely associated with a layer of retinal pigment epithelium (see figs. 12.32 and 12.33). The projections from the receptors extend into the pigmented layer and contain light-sensitive visual pigments.

The retinal pigment epithelium absorbs light waves that the receptor cells do not absorb, and with the pigment of the choroid coat, keeps light from reflecting off the surfaces inside the eye. The retinal pigment epithelium also stores vitamin A, which the receptor cells use to synthesize visual pigments.

Albinism is an inherited condition in which an enzyme required to produce pigment is missing, causing very pale, highly sun-sensitive skin. More severe forms of albinism also affect the eyes, making vision blurry and intolerant to light. A person may squint even in very faint light. This separate extra sensitivity arises because light reflects inside the lenses, overstimulating visual receptors. The eyes of many people with albinism also dart about uncontrollably, in a condition called *nystagmus.*

The visual receptors are stimulated only when light reaches them. A light image focused on an area of the retina stimulates some receptors, which conduct impulses to the brain. However, the impulse leaving each activated receptor provides only a small portion of the information required for the brain to interpret a total scene.

Rods and cones function differently. Rods are hundreds of times more sensitive to light than are cones, and as a result, rods provide vision in dim light. In addition, rods produce colorless vision, whereas cones can detect colors.

Cones provide sharp images, whereas rods produce more general outlines of objects. This is because nerve fibers from many rods may converge, so their impulses may be conducted to the brain on a single nerve fiber (see chapter 10, p. 382). Thus, if light stimulates a rod, the brain cannot tell which one of many receptors has been stimulated. Such convergence of impulses is much less common among cones, so when a cone is stimulated, the brain is able to pinpoint the stimulation more accurately (fig. 12.38).

The area of sharpest vision, the fovea centralis in the macula lutea, lacks rods but has densely packed cones with few or no converging fibers. Also, the overlying layers of the retina, as well as the retinal blood vessels, are displaced to the sides in the fovea, which more fully exposes the receptors to incoming light. Consequently, to view something in detail, a person moves the eyes so that the important part of an image falls upon the fovea centralis.

The concentration of cones decreases in areas farther away from the macula lutea, whereas the concentration of

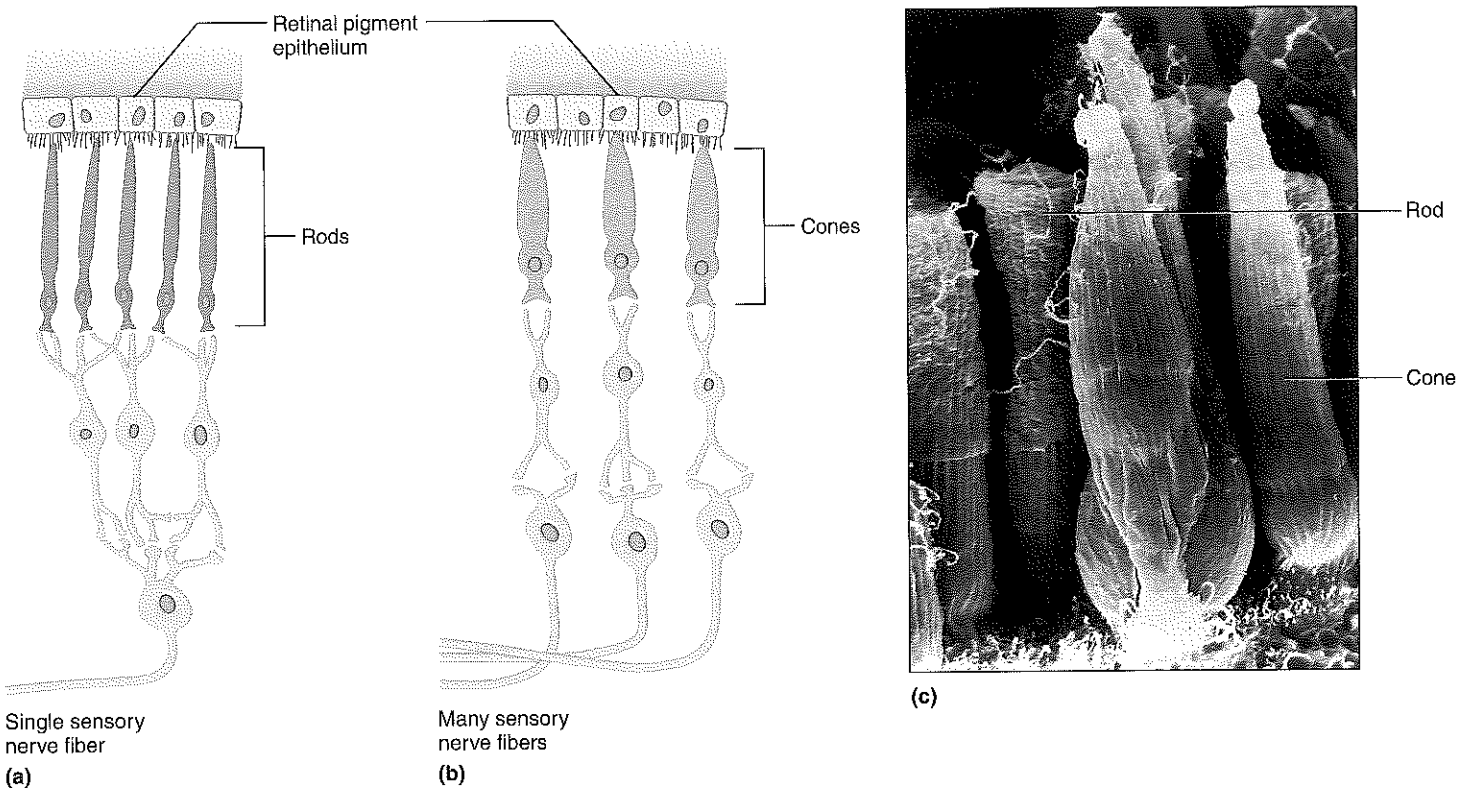


FIGURE 12.38 AP R Rods and cones. (*a*) A single sensory nerve fiber conducts impulses from several rods to the brain. (*b*) Separate sensory nerve fibers conduct impulses from cones to the brain. (*c*) Scanning electron micrograph of rods and cones (1,350×).

rods increases in these areas. Also, the degree of convergence among the rods and cones increases toward the periphery of the retina. As a result, the visual sensations from images focused on the sides of the retina are blurred compared with those focused on the central portion of the retina.

A forceful blow to the bony orbit can displace structures in and around the eye. The suspensory ligaments may tear, and the lens may become dislocated into the posterior cavity, or the retina may pull away from the underlying vascular choroid coat. Once the retina is detached, photoreceptor cells may die from lack of oxygen and nutrients. Unless such a *detached retina* is repaired surgically, visual loss or blindness may result.

Visual Pigments

Rods and cones contain light-sensitive pigments that decompose when they absorb light energy. The light-sensitive pigment in rods is **rhodopsin** (ro-dop'sin), or visual purple, and it is embedded in membranous discs stacked in these receptor cells (fig. 12.39). A single rod cell may have 2,000 interconnected discs, derived from the cell membrane. In the presence of light, rhodopsin molecules break down into molecules of a colorless protein called *opsin* and a yellowish organic molecule called *retinal* (retinene) synthesized from vitamin A.

In darkness, a nucleotide called *cyclic guanosine monophosphate* (cGMP) keeps sodium channels open in portions of the receptor cell membranes. When rhodopsin molecules absorb light, they change shape and release opsin, in trillionths of a second. The released opsin then becomes an active enzyme, which activates a second enzyme (transducin), which, in turn, activates another enzyme (phosphodiesterase). The third enzyme of this series breaks down cGMP, and as the concentration of cGMP decreases, sodium channels close, and the receptor cell membrane hyperpolarizes (see chapter 10, p. 375). The degree of hyperpolarization is directly proportional to the intensity of the light stimulating the receptor cells.

The hyperpolarization reaches the synaptic end of the cell, inhibiting release of neurotransmitter. Through a complex mechanism, decreased release of neurotransmitter by photoreceptor cells either triggers or inhibits action potentials in nearby retinal neurons. Consequently, complex patterns of impulses are conducted away from the retina, through the optic nerve, and into the brain, where they are interpreted as vision.

In bright light, nearly all of the rhodopsin in the rods decomposes, sharply reducing the sensitivity of these receptors (the rhodopsin loses its purplish color as a result, and is said to have "bleached"). The cones continue to function, however, and in bright light, we therefore see in color. In dim

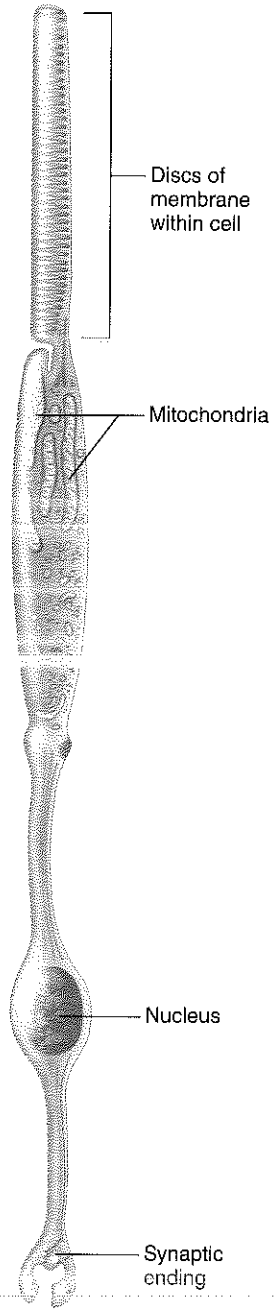


FIGURE 12.39 Rhodopsin is embedded in discs of membrane stacked in the rod cells.

light, rhodopsin can be regenerated from opsin and retinal faster than it is broken down. This regeneration requires cellular energy, which ATP provides (see chapter 4, p. 127). Under these conditions, the rods continue to function and the cones remain unstimulated. Hence, we see only shades of gray in dim light.

The light sensitivity of an eye whose rods have converted the available opsin and retinal to rhodopsin increases about 100,000 times, and the eye is said to be *dark adapted.* A person needs dark-adapted eyes to see in dim light. For example, when going from daylight into a darkened theater, it may be difficult to see well enough to locate a seat, but soon the eyes adapt to the dim light, and vision improves. Later, leaving the theater and entering the sunlight may cause discomfort or even pain. This occurs at the moment that most of the rhodopsin decomposes in response to the bright light. At the same time, the light sensitivity of the eyes decreases greatly, and they become *light adapted.*

Too little vitamin A in the diet reduces the amount of retinal, impairing rhodopsin production and sensitivity of the rods. The result is poor vision in dim light, called nightblindness.

The light-sensitive pigments of cones, called *iodopsins,* are similar to rhodopsin in that they are composed of retinal combined with a protein; the protein, however, differs from the protein in the rods. The three sets of cones in the retina all contain an abundance of one of three different visual pigments.

The wavelength of a particular type of light determines the color perceived from it. For example, the shortest wavelengths of visible light are perceived as violet, whereas the longest wavelengths of visible light are seen as red. One type of cone pigment (erythrolabe) is most sensitive to red light waves, another (chlorolabe) to green light waves, and a third (cyanolabe) to blue light waves. The sensitivities of these pigments overlap somewhat. For example, both red and green light pigments are sensitive to orange light waves. On the other hand, red pigment absorbs orange light waves more effectively.

The color perceived depends upon which sets of cones the light in a given image stimulates. If all three types of sets of cones are stimulated, the light is perceived as white, and if none are stimulated, it is seen as black.

Examination of the retinas of different people reveals that individuals have unique patterns of these three cone types, all apparently able to provide color vision. Some parts of the retina are even normally devoid of one particular type, yet the brain integrates information from all over to "fill in the gaps," creating a continuous overall image. People who lack a cone type due to a mutation are colorblind.

Stereoscopic Vision

Stereoscopic vision (stereopsis) simultaneously perceives distance, depth, height, and width of objects. Such vision is possible because the pupils are 6–7 centimeters apart. Consequently, close objects (less than 20 feet away) produce slightly different retinal images. That is, the right eye sees a little more of one side of an object, while the left eye sees a little more of the other side. The visual cortex superimposes and interprets the two images. The result is the perception of a single object in three dimensions (fig. 12.40).

Stereoscopic vision requires vision with two eyes (binocular vision). Therefore, a person with only one functional eye is less able to accurately judge distance and depth. To compensate, a person with one eye can use the relative sizes and positions of familiar objects as visual clues.

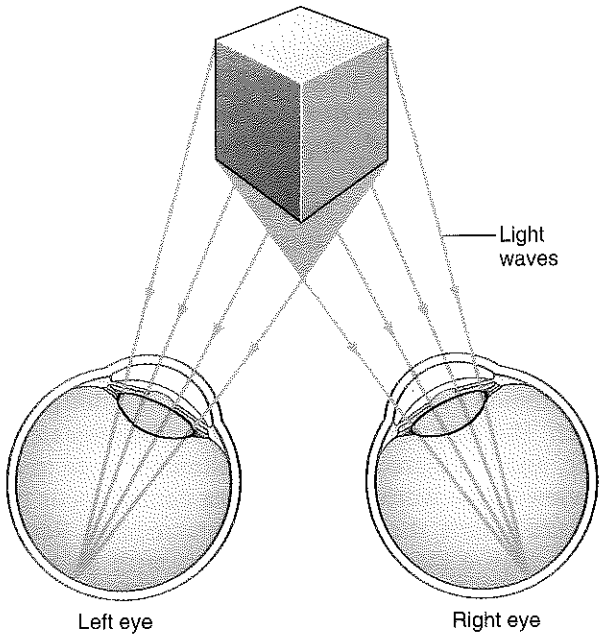


FIGURE 12.40 Stereoscopic vision results from formation of two slightly different retinal images.

Visual Pathways

The axons of the ganglion cells in the retina leave the eyes, forming the *optic nerves* (see chapter 11, p. 421). Just anterior to the pituitary gland, these nerves give rise to the X-shaped *optic chiasma,* and in the chiasma, some of the fibers cross over. Specifically, the fibers from the nasal (medial) half of each retina cross over, whereas those from the temporal (lateral) halves do not. In this way, fibers from the nasal half of the left eye and the temporal half of the right eye form the right *optic tract;* fibers from the nasal half of the right eye and the temporal half of the left eye form the left optic tract.

The nerve fibers continue in the optic tracts, and before they reach the thalamus, a few of them enter nuclei that function in various visual reflexes. Most of the fibers, however, enter the thalamus and synapse in part of its posterior portion (lateral geniculate body). From this region, the visual sensory fibers enter pathways called *optic radiations,* and these pathways lead to the visual cortex of the occipital lobes (fig. 12.41).

Each visual cortex receives impulses from each eye, so a person may develop partial blindness in both eyes if either visual cortex is injured. For example, if the right visual cortex (or the right optic tract) is injured, sight may be lost in the temporal side of the right eye and the nasal side of the left eye. Similarly, damage to the central portion of the optic chiasma, where fibers from the nasal sides of the eyes cross over, blinds the nasal sides of both eyes.

Sensory fibers not leading to the thalamus conduct visual impulses downward into the brainstem. These impulses are important for controlling head and eye movements associated with visually tracking an object; for controlling the simultaneous movements of both eyes; and for controlling certain visual reflexes, such as those that move the muscles of the iris.

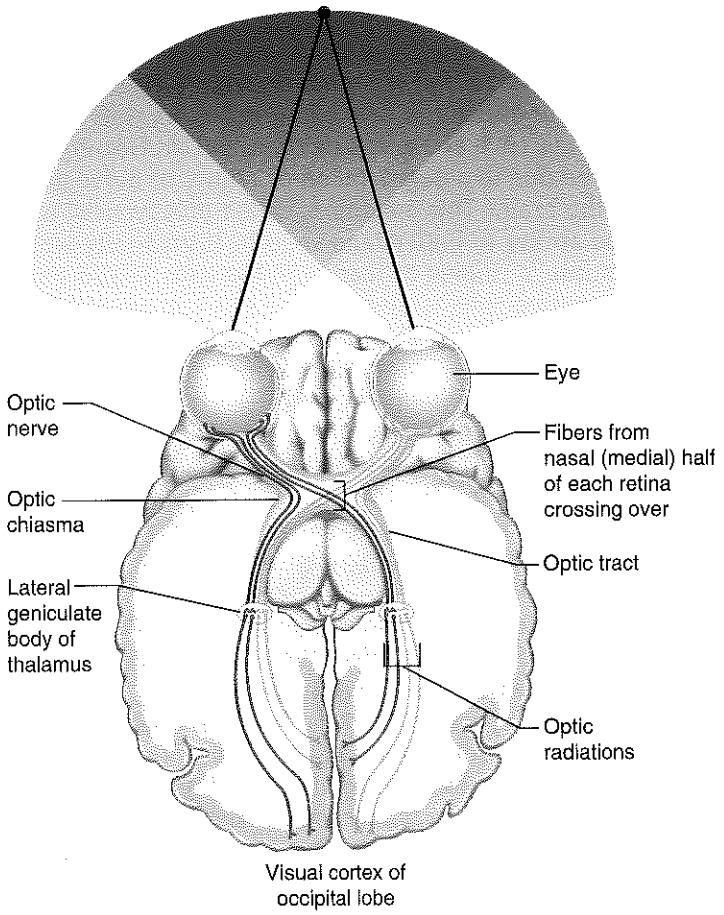


FIGURE 12.41 AP|R The visual pathway includes the optic nerve, optic chiasma, optic tract, and optic radiations.

PRACTICE

43 Distinguish between the rods and the cones of the retina.

44 Explain the roles of visual pigments.

45 What factors make stereoscopic vision possible?

46 Trace the pathway of visual impulses from the retina to the occipital cortex.

12.5 LIFE-SPAN CHANGES

We often first become aware of aging-associated changes through diminished senses. By age forty, a book may need to be held farther away from the eyes. By the fifties, the senses of smell and taste may begin to diminish, which usually reflects anosmia, a loss of olfactory receptors.

By age sixty, a quarter of the population experiences noticeable hearing loss, and from ages sixty-five to seventy-four, the percentage reaches a third. Half of all people over

age eighty-five cannot hear adequately. Age-related hearing loss may be the result of decades of cumulative damage to the sensitive hair cells of the spiral organ in the inner ear. It becomes more difficult to hear high pitches, as well as particular sounds, such as *f, g, s, t, sh, th, z,* and *ch.* Hearing loss may also be due to a degeneration or failure of nerve pathways to the brain. This condition, called presbycusis, may affect the ability to understand speech. It gradually worsens. Tinnitus, a ringing or roaring in the ears, is also more common among older adults. Hearing aids can often restore some hearing. A person to whom the ordinary sounds of life are hopelessly garbled may show symptoms of paranoia, depression, or social withdrawal.

Vision may decline with age for several reasons. "Dry eyes" are common. Too few tears, or poor quality tears, lead to itching and burning eyes, and diminished vision. In some cases, too many tears result from oversensitivity to environmental effects, such as wind, intense light, or a change in temperature.

With age, tiny dense clumps of gel or crystal-like deposits form in the vitreous humor. When these clumps cast shadows on the retina, the person sees small moving specks in the field of vision. These specks, or *floaters,* are most apparent when looking at a plain background, such as the sky or a blank wall. Also with age, the vitreous humor may shrink and pull away from the retina. This may mechanically stimulate receptor cells of the retina, and the person may see flashes of light.

The inability to read small print up close as one gets older, called presbyopia, results from a loss of elasticity in the lens, preventing it from changing shape easily. After age seventy, the iris cannot dilate as well as it once did, halving the amount of light that can enter the eye. Brighter lights can counter this effect.

Glaucoma develops in the eyes as a person ages when the rate of aqueous humor formation exceeds the rate of its removal. Fluid accumulates in the anterior chamber of the eye, raising the fluid pressure. As this pressure is transmitted to all parts of the eye, in time, the blood vessels that supply the receptor cells of the retina may squeeze shut, cutting off the nutrient and oxygen supply. The result may eventually be permanent blindness.

Drugs, or traditional or laser surgery to promote the outflow of aqueous humor, can treat glaucoma if it is diagnosed early. However, because glaucoma in its early stages typically produces no symptoms, discovery of the condition usually depends on measuring the intraocular pressure using an instrument called a *tonometer.*

A common eye disorder particularly in older people is *cataract.* The lens or its capsule slowly becomes cloudy, opaque, and discolored, adding a yellowish tinge to a person's view of the world. Clear images cannot focus on the retina, and in time, the person may become blind. Removing the lens with a laser and replacing it with an artificial implant can treat cataract. Afterward, patients report that their surroundings are no longer yellow.

Several conditions affect the retinas of an older person. In a form of age-related macular degeneration, and also in people with diabetes who develop diabetic retinopathy, tiny blood vessels extend into the macula, making images in the center of the visual field appear wavy. Retinal detachment becomes more common.

Despite these various problems, many older individuals continue to enjoy sharp, functional senses well into the upper decades of life.

PRACTICE

47 Why do smell and taste diminish with age?

48 What are some causes of age-related hearing loss?

49 Describe visual problems likely to arise with age.

CHAPTER SUMMARY

12.1 INTRODUCTION (PAGE 444)

Sensory receptors are sensitive to internal and external environmental changes and initiate impulses to the brain and spinal cord.

12.2 RECEPTORS, SENSATION, AND PERCEPTION (PAGE 444)

1. Receptor types
   a. Each type of receptor is sensitive to a distinct type of stimulus.
   b. The major types of receptors include:
      (1) Chemoreceptors, sensitive to changes in chemical concentration.
      (2) Pain receptors (nociceptors), sensitive to tissue damage.
      (3) Thermoreceptors, sensitive to temperature changes.
      (4) Mechanoreceptors, sensitive to mechanical forces.
      (5) Photoreceptors, sensitive to light.
2. Sensory impulses
   a. When receptors are stimulated, membrane potentials change.
   b. Receptor potentials are transferred to nerve fibers, triggering action potentials.
3. Sensation and perception
   a. Sensation is an awareness resulting from sensory stimulation.
   b. Perception is when a particular part of the sensory cortex interprets the sensory stimulation.
   c. The cerebral cortex projects a sensation back to the region of stimulation.
4. Sensory adaptations are adjustments of sensory receptors to continuous stimulation. Impulses are triggered at slower rates.

12.3 GENERAL SENSES (PAGE 446)

General senses receive information from receptors in skin, muscles, joints, and viscera. They can be grouped as exteroceptive, visceroceptive, and proprioceptive senses.

1. Touch and pressure senses
   a. Free ends of sensory nerve fibers are the receptors for the sensations of touch and pressure.
   b. Tactile corpuscles are the receptors for the sensations of light touch.
   c. Lamellated corpuscles are the receptors for the sensations of heavy pressure and vibrations.
2. Temperature senses
   a. Thermoreceptors include two sets of free nerve endings that are heat and cold receptors.
   b. Combinations of input from both receptor types are interpreted as intermediate temperatures.
3. Sense of pain
   a. Pain receptors
      (1) Pain receptors are free nerve endings that tissue damage stimulates.
      (2) Pain receptors provide protection; do not adapt rapidly; and can be stimulated by changes in temperature, mechanical force, and chemical concentration.
   b. The only receptors in viscera that provide sensations are pain receptors.
      (1) These receptors are most sensitive to certain chemicals and lack of blood flow.
      (2) The sensations they produce may feel as if they come from some other part of the body (referred pain).
   c. Pain pathways
      (1) The two main types of pain fibers are acute pain fibers and chronic pain fibers.
      (2) Acute pain fibers are fast conducting; chronic pain fibers are slower conducting.
      (3) Pain impulses are processed in the dorsal horn of the spinal cord, and they ascend in the spinothalamic tracts.
      (4) Within the brain, pain information passes through the thalamus or the reticular formation before being relayed to the cerebral cortex.
   d. Regulation of pain impulses
      (1) Awareness of pain occurs when impulses reach the thalamus.
      (2) The cerebral cortex judges the intensity of pain and locates its source.
      (3) Impulses descending from the brain cause neurons to release pain-relieving substances, such as enkephalins and serotonin.
      (4) Endorphin is a pain-relieving biochemical produced in the brain.
   e. Certain neuropeptides synthesized in the CNS inhibit pain impulses.
4. Proprioception
   a. Stretch receptors provide information about the condition of muscles and tendons.
   b. Muscle spindles are stimulated when a muscle is relaxed, and they initiate a reflex that contracts the muscle.
   c. Golgi tendon organs are stimulated when muscle tension increases, and they initiate a reflex that relaxes the muscle.
5. Visceral receptors include lamellated corpuscles and free nerve endings.

12.4 SPECIAL SENSES (PAGE 452)

Special senses have receptors in complex sensory organs of the head.

1. Sense of smell
   a. Olfactory receptors
      (1) Olfactory receptors are chemoreceptors that chemicals dissolved in nasal secretions stimulate.
      (2) Olfactory receptors function with taste receptors and aid in food selection.
   b. Olfactory organs
      (1) The olfactory organs consist of receptor cells and supporting cells in the nasal cavity.
      (2) Olfactory receptor cells are neurons with cilia containing membrane receptor proteins.
   c. Olfactory pathways.
      (1) Smell information travels from the olfactory receptor cells through the olfactory bulbs and olfactory tracts.
      (2) This information goes to interpreting centers in the limbic system.
   d. Olfactory stimulation
      (1) Olfactory impulses may result when an odorant molecule stimulates a distinct set of receptor cells.
      (2) Olfaction undergoes rapid sensory adaptation.
      (3) Olfactory receptors are often damaged by environmental factors and are replaced from a pool of stem cells.
2. Sense of taste
   a. Taste receptors
      (1) Taste buds consist of receptor cells and supporting cells.
      (2) Taste cells have taste hairs that sense particular chemicals dissolved in water.
      (3) Taste hair surfaces have receptor sites to which chemicals combine and trigger impulses to the brain.
   b. Taste sensations
      (1) The five primary taste sensations are sweet, sour, salty, bitter, and umami.
      (2) Various taste sensations result from the stimulation of one or more sets of taste receptors.
      (3) Each of the five primary types of taste cells is particularly sensitive to a certain group of chemicals.
   c. Taste pathways
      (1) Sensory information from taste receptors travels on fibers of the facial, glossopharyngeal, and vagus nerves.
      (2) This information is carried to the medulla oblongata and ascends to the thalamus and then to the gustatory cortex in the parietal lobes.
3. Sense of hearing
   a. The outer ear includes the auricle, the external acoustic meatus, and the tympanic membrane. It collects sound waves created by vibrating objects and transfers the vibrations to the middle ear.
   b. Middle ear
      (1) Auditory ossicles of the middle ear transfer sound waves from the tympanic membrane

to the oval window of the inner ear. They also increase the force of these waves.
(2) Skeletal muscles attached to the auditory ossicles provide the tympanic reflex, which protects the inner ear from the effects of loud sounds.

c. Auditory tubes connect the middle ears to the throat and help maintain equal air pressure on both sides of the tympanic membranes.
d. Inner ear
(1) The inner ear consists of a complex system of connected tubes and chambers—the osseous and membranous labyrinths. It includes the cochlea, which houses the spiral organ.
(2) The spiral organ includes the hearing receptors that are stimulated by vibrations in the fluids of the inner ear.
(3) Different frequencies of vibrations stimulate different sets of receptor cells; the human ear can detect sound frequencies from about 20 to 20,000 vibrations per second.
e. Auditory pathways
(1) The axons from hearing receptors are found in the cochlear branch of the vestibulocochlear nerves.
(2) Auditory information travels into the medulla oblongata, midbrain, and thalamus and is interpreted in the temporal lobes of the cerebrum.

4. Sense of equilibrium
a. Static equilibrium maintains the stability of the head and body when they are motionless. The organs of static equilibrium are in the vestibule.
b. Dynamic equilibrium balances the head and body when they are suddenly moved or rotated. The organs of this sense are in the ampullae of the semicircular canals.
c. Other structures that help maintain equilibrium include the eyes and the proprioceptors associated with certain joints.

5. Sense of sight
a. Visual accessory organs include the eyelids and lacrimal apparatus that protect the eye and the extrinsic muscles that move the eye.
b. Structure of the eye
(1) The wall of the eye has an outer, a middle, and an inner tunic that function as follows:
(a) The outer layer (sclera) is protective, and its transparent anterior portion (cornea) refracts light entering the eye.
(b) The middle layer (choroid coat) is vascular and has pigments that help keep the inside of the eye dark.
(c) The inner layer (retina) includes visual receptor cells.
(2) The lens is a transparent, elastic structure. The ciliary muscles control its shape.
(3) The iris is a muscular diaphragm that controls the amount of light entering the eye; the pupil is an opening in the iris.
(4) Spaces in the eye are filled with fluids (aqueous and vitreous humors) that help maintain its shape.
c. Light refraction
(1) Light waves are primarily refracted by the cornea and lens to focus an image on the retina.
(2) The lens must thicken to focus on close objects.
d. Visual receptors
(1) The visual receptors are rods and cones.
(2) Rods are responsible for colorless vision in dim light, and cones provide color vision.
e. Visual pigments
(1) A light-sensitive pigment in rods (rhodopsin) decomposes in the presence of light and triggers a complex series of reactions that initiate action potentials on the optic nerve.
(2) Three sets of cones provide color vision. Each set has a different light-sensitive pigment, and each set is sensitive to a different wavelength of light; the color perceived depends on which set or sets of cones are stimulated.
f. Stereoscopic vision
(1) Stereoscopic vision provides perception of distance and depth.
(2) Stereoscopic vision occurs because of the formation of two slightly different retinal images that the brain superimposes and interprets as one image in three dimensions.
(3) A one-eyed person uses relative sizes and positions of familiar objects to judge distance and depth.
g. Visual pathways
(1) Nerve fibers from the retina form the optic nerves.
(2) Some fibers cross over in the optic chiasma.
(3) Most of the fibers enter the thalamus and synapse with others that continue to the visual cortex of the occipital lobes.
(4) Other impulses pass into the brainstem and function in various visual reflexes.

12.5 LIFE-SPAN CHANGES (PAGE 481)

Diminished senses are often one of the first noticeable signs of aging.

1. Age-related hearing loss may reflect damage to hair cells of the spiral organ, degeneration of nerve pathways to the brain, or tinnitus.
2. Age-related visual problems include dry eyes, floaters and light flashes, presbyopia, glaucoma, cataracts, macular degeneration, and retinal detachment.

CHAPTER ASSESSMENTS

12.1 Introduction

1 Explain the difference between a general sense and a special sense. (p. 444)

12.2 Receptors, Sensation, and Perception

2 Match each sensory receptor to the type of stimulus to which it is likely to respond: (p. 445)

(1) chemoreceptor	A. approaching headlights
(2) pain receptor	B. a change in blood pressure
(3) thermoreceptor	C. the smell of roses
(4) mechanoreceptor	D. an infected tooth
(5) photoreceptor	E. a cool breeze

3 Explain how sensory receptors stimulate sensory impulses. (p. 445)

4 Explain the difference between a sensation and a perception. (p. 445)

5 Explain the projection of a sensation. (p. 445)

6 Define *sensory adaptation.* (p. 446)

7 You fill up the tub to take a hot bath, but the water is too hot. You test it a second and third time within a few seconds, and it feels OK. Which of the following is the most likely explanation? (p. 446)
   a. The water has cooled down unusually quickly.
   b. Your ability to sense heat has adapted.
   c. Your nervous system is suddenly not functioning properly.
   d. Someone added ice cubes to your bath.
   e. none of the above

12.3 General Senses

8 Explain how general senses can be grouped. (p. 446)

9 Describe the functions of free nerve endings, tactile corpuscles, and lamellated corpuscles. (p. 446)

10 Describe the functions of the two classes of thermoreceptors. (p. 446)

11 Compare pain receptors with the other types of somatic receptors. (p. 446)

12 List the conditions likely to stimulate visceral pain receptors. (p. 447)

13 Define *referred pain,* and provide an example. (p. 448)

14 Contrast the pathways involved in the production of acute and chronic pain. (p. 448)

15 Explain how neuropeptides relieve pain. (p. 449)

16 Distinguish between muscle spindles and Golgi tendon organs. (p. 450)

12.4 Special Senses

17 Explain how the senses of smell and taste function together to create the perception of the flavors of foods. (p. 452)

18 Which two of the following are part of the olfactory organs? (p. 453)
   a. olfactory receptors
   b. columnar epithelial cells in the nasal mucosa
   c. the nose
   d. the brain
   e. the mouth

19 Trace each step in the pathway from an olfactory receptor to the interpreting center of the cerebrum. (p. 454)

20 Salivary glands are important in taste because _____. (p. 454)
   a. they provide the fluid in which food molecules dissolve
   b. the taste receptors are located in salivary glands
   c. salivary glands are part of the brain
   d. lamellar corpuscles are activated
   e. salivary glands conduct action potentials

21 Name the five primary taste sensations and indicate a specific stimulus for each. (p. 454)

22 Explain why taste sensation is less likely to diminish with age than olfactory sensation. (p. 455)

23 Trace each step in the pathway from a taste receptor to the interpreting center of the cerebrum. (p. 456)

24 Match the ear area with the associated structure: (p. 456)

(1) outer ear	A. cochlea
(2) middle ear	B. eardrum
(3) inner ear	C. auditory ossicles

25 Trace each step in the pathway from the external acoustic meatus to hearing receptors. (p. 457)

26 Describe the functions of the auditory ossicles. (p. 457)

27 Identify the parts of the tympanic reflex, explain how they work, and explain the importance of this reflex. (p. 457)

28 The function of the auditory tube is to _____. (p. 458)
   a. equalize air pressure on both sides of the eardrum
   b. transmit sound vibrations to the eardrum
   c. contain the hearing receptors
   d. contain the auditory ossicles
   e. secrete cerumen

29 Distinguish between the osseous and membranous labyrinths. (p. 459)

30 Describe the cochlea and its function. (p. 459)

31 Which of the following best describes hearing receptor "hair cells"? (p. 461)
   a. They are neurons.
   b. They lack ion channels.
   c. They are epithelial, but function like neurons.
   d. They are made of keratin.
   e. They are attached to the tympanic membrane.

32 Explain how a hearing receptor stimulates a sensory neuron. (p. 461)

33 Trace each step in the pathway from the spiral organ to the interpreting centers of the cerebrum. (p. 461)

34 Describe the organs of static and dynamic equilibrium and their functions. (p. 464)

35 Explain how the sense of vision helps maintain equilibrium. (p. 468)

36 Match the visual accessory organ with its function: (p. 468)

(1) eyelid	A. moves the eye
(2) conjunctiva	B. covers the eye
(3) lacrimal gland	C. lines the eyelids
(4) extrinsic muscle	D. produces tears

37 Name the three layers of the eye wall and describe the functions of each layer. (p. 471)

38 Explain why looking at a close object causes fatigue in terms of how accommodation is accomplished. (p. 473)

39 Explain the mechanisms of pupil constriction and pupil dilation. (p. 474)

40 Distinguish between the fovea centralis and the optic disc. (p. 475)

41 The following are compartments in the eye. In which one is vitreous humor found? (p. 476)
   a. anterior chamber
   b. posterior chamber
   c. anterior cavity
   d. posterior cavity
   e. lens
42 Explain how light is focused on the retina. (p. 478)
43 Distinguish between rods and cones. (p. 478)
44 Explain why cone vision is generally more acute than rod vision. (p. 478)
45 Describe the function of rhodopsin. (p. 479)
46 Explain why rod vision may be more important under dim light conditions. (p. 480)
47 Describe the relationship between light wavelength and color vision. (p. 480)
48 Define *stereoscopic vision*. (p. 480)
49 Explain why a person with normal binocular vision is able to judge distance and depth of close objects more accurately than a person who has lost one eye. (p. 480)
50 Trace each step in the pathway from the retina to the visual cortex. (p. 481)

12.5 Life-Span Changes

51 Explain the basis of fading senses of smell and taste with aging. (p. 481)
52 List three causes of hearing loss associated with aging. (p. 482)
53 Explain five problems that can interfere with vision as a person ages. (p. 482)

INTEGRATED ASSESSMENTS/CRITICAL THINKING

OUTCOMES 2.2, 11.5, 12.2, 12.3, 12.4

1. Positron emission tomography (PET) scans of the brains of people who have been blind since birth reveal high neural activity in the visual centers of the cerebral cortex when these people read Braille. When sighted individuals run their fingers over the raised letters of Braille, their visual centers do not show increased activity. Explain these findings.

OUTCOMES 6.5, 11.6, 12.2

2. Why are some serious injuries, like a bullet entering the abdomen, relatively painless, but others, such as a burn, considerably more painful?

OUTCOMES 11.5, 12.2, 12.4

3. Loss of the sense of smell often precedes the major symptoms of Alzheimer disease and Parkinson disease. What additional information is needed to use this association to prevent or treat these diseases?

OUTCOMES 12.2, 12.3

4. A patient with heart disease experiences pain at the base of the neck and in the left shoulder and upper limb during exercise. How would you explain the likely origin of this pain to the patient?

OUTCOMES 12.2, 12.4

5. People who are deaf due to cochlear damage do not suffer motion sickness. Why not?
6. Labyrinthitis is an inflammation of the inner ear. What symptoms would you expect in a patient with this disorder?