



# CHAPTER 6

# Vision

## LEARNING OBJECTIVES

- L01** Discuss the major features of visible light as a stimulus.
- L02** Explain the major features and functions of the eye, retina, and photoreceptors.
- L03** Identify the pathways of information from the photoreceptors to the secondary visual cortex.
- L04** Summarize the processes responsible for visual object perception, depth perception, and color vision.
- L05** Describe the changes in the visual system that accompany normal aging.
- L06** Differentiate between the major disorders that affect human vision.

## CHAPTER OUTLINE

- From Sensation to Perception**
  - The Visual Stimulus: Light
  - The Advantages of Light as a Stimulus
  - The Electromagnetic Spectrum
  - Light Interacts with Objects
- Interim Summary 6.1**
- The Structure and Functions of the Visual System**
  - Protecting the Eye
  - The Functional Anatomy of the Eye
  - The Layered Organization of the Retina
  - The Photoreceptors
  - Processing by Retinal Interneurons
  - Optic Nerve Connections
- The Striate Cortex
- Visual Analysis beyond the Striate Cortex
- Interim Summary 6.2**
- Visual Perception**
  - Hierarchies
  - Spatial Frequencies
  - The Perception of Depth
  - Coding Color
- The Life-Span Development of the Visual System**
- Disorders of the Visual System**
  - Amblyopia
  - Cataracts
  - Visual Acuity Problems
  - Blindness
  - Visual Agnosias
- Interim Summary 6.3**
- Chapter Review**

**CONNECTING TO RESEARCH:** Hubel and Wiesel Map the Visual Cortex

**BEHAVIORAL NEUROSCIENCE GOES TO WORK:**  
3-D Animation

**THINKING ETHICALLY:** Are There Sex Differences in Color Preferences?

**BUILDING BETTER HEALTH:** Does Eating Carrots Really Help Your Vision?

## From Sensation to Perception

What is reality? This might sound like a more fitting question for a philosophy course than a neuroscience course, but this question is central to the study of sensation and perception. We believe that an objective physical reality does exist “out there,” but we can only make educated guesses about its features using the human sensory systems we discuss in this chapter and the next.

The physical world provides many sources of information, from light waves to pressure to molecules suspended in the air. Over the course of evolution, organisms have developed sensory systems that **transduce** (translate) different types of information into the action potentials that the nervous system can process. Each organism has sensory capacities that enhance its survival within a particular niche. Ours is a uniquely human version of reality, providing us with just the right set of information we need to survive. We might not be able to sense odor as well as a dog, but we can see colors that dogs cannot see (see ● Figure 6.1). A single physical reality produces very different reactions in different organisms.

**Sensation** begins the process of building a model of reality by bringing relevant information to the central nervous system (CNS). Once the CNS has begun to interpret the information, the process of **perception** is underway. An important gateway to perception is **attention**, which is defined as a narrow focus of consciousness. Although we can consciously command ourselves to “pay attention” to a boring but necessary lecture, many aspects of attention are much more automatic. We naturally attend to unfamiliar, changing, and high-intensity stimuli, as these are likely to have significance for our safety and survival. We adapt to unchanging information like the hum of a computer by paying less attention to it over time.

The process of perception is often a two-way street, where incoming messages inform the brain at the same time the brain is imposing structure on those messages. Consider the following passage:

All you hvae to do to mkae a snetnece raedalbe is to mkae srue taht the fisrt and lsat letrtes of ecah word saty the smae. Wtih prcatcie, tihis porcses becoems mcuh fsater and esaeir.

Clearly, the brain is receiving sensations resulting from the reflection of light from these letters on the page, which are then used to construct words and meanings. This pathway is known as **bottom-up processing**. At the same time, we use our knowledge and expectations to recognize words, even if they are misspelled. This pathway is

**transduction** The transformation of sensory information into neural signals.

**sensation** The process of obtaining information about the environment and transmitting it to the brain for processing.

**perception** The process of interpreting sensory signals sent to the brain.

**attention** A narrow focus of consciousness.

**bottom-up processing** The combining of simpler meanings to construct more complex meanings.



Courtesy of Laura Freberg

● **Figure 6.1 Sensory Systems Differ across Species** Sensory systems have evolved to enhance the survival of members of each species within their own niches. Human vision, seen in the photograph of the author’s puppy on the left, is different than dog vision, simulated on the right. Dogs do a good job of distinguishing blue and yellow but do not see reds and greens the way human beings do.

known as **top-down processing**. In many instances, the world we perceive is the world we expect to perceive (Kinchla & Wolfe, 1979).

## The Visual Stimulus: Light

Vision is one of the most important sensory systems in humans, with about 50 percent of our cerebral cortex responding to visual information but only 3 percent for hearing and 11 percent for touch and pain (Kandel & Wurtz, 2000; Sereno & Tootell, 2005). Whether we're searching for food or scanning the millions of colors displayed by a computer monitor, the process of vision begins with light energy reflected from objects.

Visible light, or the energy we can see, is one form of **electromagnetic radiation** produced by the sun. Electromagnetic radiation can be described as moving waves of energy (see ● Figure 6.2). **Wavelength**, or the distance between successive peaks of waves, is encoded by the visual system either as color or as shades of gray. The **amplitude** of light waves refers to the height of each wave, which is translated by the visual system as brightness. Large-amplitude waves are perceived as bright, and low-amplitude waves are perceived as dim.

Electromagnetic radiation can also be described as the movement of tiny, indivisible particles known as photons. **Photons** always travel at the same speed (the so-called speed of light), but they can vary in the amount of energy they possess. It is this variation in energy levels among photons that gives us waves with different wavelengths and amplitudes. You can think of a light wave as describing the movement of large numbers of photons, much as a wave in the ocean describes the movement of large numbers of water molecules.

## The Advantages of Light as a Stimulus

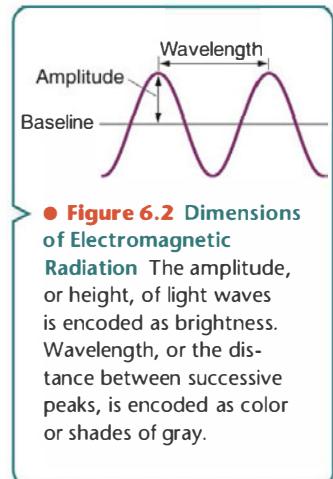
Why is electromagnetic energy, rather than some other feature of the environment, an effective stimulus for a sensory system? Electromagnetic energy, and visible light in particular, has features that make it a valuable source of information. First, electromagnetic energy is abundant in our universe. Second, because electromagnetic energy travels very quickly, there is no substantial delay between an event and an organism's ability to see the event. Finally, electromagnetic energy travels in fairly straight lines, minimizing the distortion of objects. What we see is what we get, literally.

## The Electromagnetic Spectrum

The light from the sun contains a mixture of wavelengths and appears white to the human eye. Placing a prism in sunlight will separate individual wavelengths, which we see as different colors. Light shining through water droplets is affected the same way, producing the rainbows we enjoy seeing after a rainstorm.

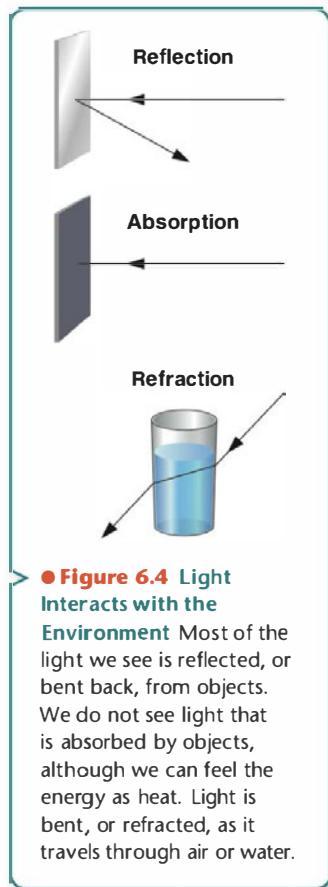
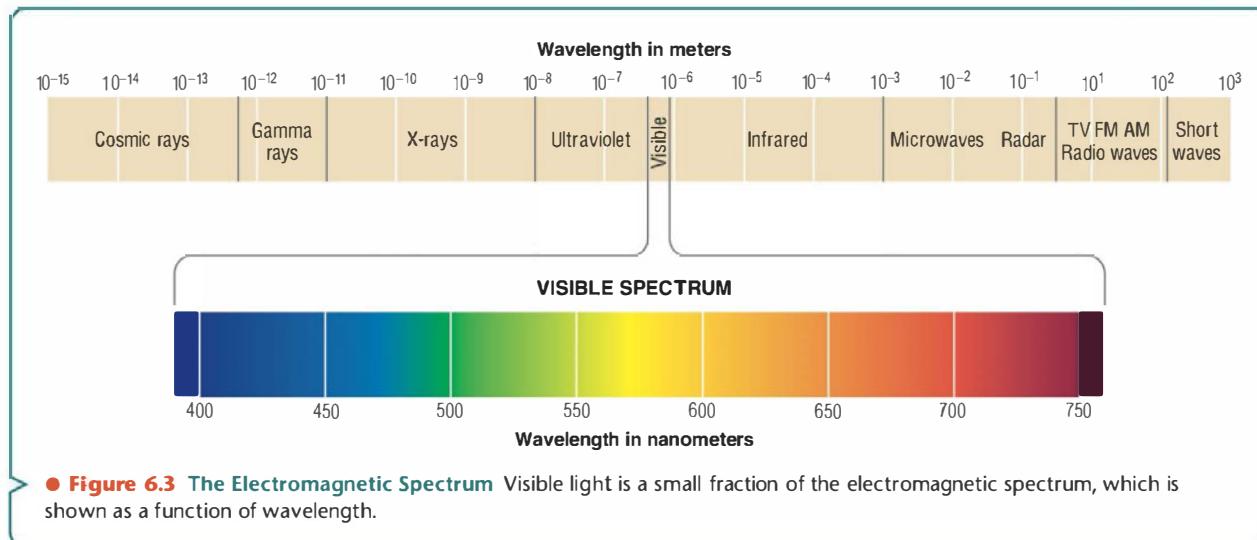
As shown in ● Figure 6.3, light that is visible to humans occupies a small part of the electromagnetic spectrum. The range of electromagnetic energy visible to humans falls between 400 and 700 **nanometers** (nm). A nanometer is  $10^{-9}$  meters, or one billionth of a meter. When we say a light has a wavelength of 400 nanometers, this means that the peaks of the wave are 400 nanometers apart. Shorter wavelengths, approaching 400 nm, are perceived by humans as violet and blue, whereas longer wavelengths, approaching 700 nm, are perceived as red.

Gamma rays, X-rays, ultraviolet rays, infrared rays, microwaves, and radio waves lie outside the range of wavelengths the human eye can detect. These forms of energy have features that make them less desirable stimuli for a sensory system. Shorter wavelengths, such as ultraviolet rays, are typically absorbed by the ozone layer of Earth's atmosphere, leaving little energy left over for most organisms to sense. Nonetheless, some creatures, including insects and birds, are able to see parts of the ultraviolet spectrum. Longer wavelengths, such as microwaves, tend to penetrate objects rather than



**● Figure 6.2 Dimensions of Electromagnetic Radiation** The amplitude, or height, of light waves is encoded as brightness. Wavelength, or the distance between successive peaks, is encoded as color or shades of gray.

**top-down processing** The use of knowledge and expectation to interpret meanings.  
**electromagnetic radiation** Radiation emitted in the form of energy waves.  
**wavelength** The distance between successive peaks of a wave; determines color in visible light.  
**amplitude** The height of a wave; in vision, the source of the subjective experience of brightness.  
**photons** Individual, indivisible, very small particles that form waves of electromagnetic energy.  
**nanometers** A unit of measurement equaling  $10^{-9}$  m used to measure light wave frequency.



**absorption** The ability to retain something rather than reflect or transmit it to another location.

**reflection** The bending back of light toward its source.

**refract** The deflection, or changing of direction, of light at a boundary such as that between air and water.

reflect back from them, a feature that is valuable in cooking but not in vision. However, some snakes, such as pit vipers, boas, and pythons, have developed the ability to “see” the body heat of prey and other predators by sensing infrared radiation.

## Light Interacts with Objects

As shown in **Figure 6.4**, objects can absorb, reflect, or refract electromagnetic radiation. In some cases, an object's physical characteristics will absorb or retain certain wavelengths. In other cases, light is reflected from the surface of objects, or bent back toward the source. Most of the light energy entering the eye has been reflected from objects in the environment.

**Absorption** and **reflection** of light by objects determine the colors we see. The color of an object is not some intrinsic characteristic of the object but, rather, the result of the wavelengths of light that are selectively absorbed and reflected by the object. Instead of saying that my sweater is red, it is more accurate to say that my sweater has physical characteristics that reflect long wavelengths of visible light (perceived as red) and absorb shorter wavelengths. “Light-colored” clothing keeps us cooler because materials perceived as white or light-colored reflect more electromagnetic energy. “Dark” clothing keeps us warmer because these materials absorb more electromagnetic energy. You can easily demonstrate this concept by timing the melting of ice cubes in sunlight when one ice cube is covered by a white piece of cloth and the other by a black piece of cloth.

Air and water **refract**, or change the direction of, traveling waves of light in different ways. Because human eyes developed for use in air, they don't work as well underwater. To see clearly underwater, we need goggles or a face mask to maintain a bubble of air next to the eye. Consequently, even though our bodies are underwater, our eyes remain exposed to light that has been refracted by air, and they function normally. Fish eyes are perfectly adapted to a life underwater. To focus light properly as it is refracted by water, the outer surface of the fish eye is rippled, which reduces distortion or blurriness. In addition, the lens of the eye, which acts like a magnifying glass, is configured differently for viewing light through air or water. The human lens is shaped like an aspirin tablet, whereas the fish lens is shaped like a sphere. Some organisms, such as diving birds, move in and out of the water, which poses problems for either the human or fish eye. The cormorant solves the problem of blurry underwater vision with a special eyelid that closes when the bird dives after a fish.



Zbigniew Leszczynski/AGE Fotostock

● **Figure 6.5** *Anableps* and Challenges with Refraction

To catch the waterborne insects that make up its diet and to avoid predators lurking below the surface, *Anableps anableps* must see both above and below the water. Normally, eyes that are designed to see in air will provide blurry images underwater. *Anableps* solves the dilemma with its unique eyes, each of which has two pupils—one above and one below the water. Locals refer to *Anableps* as *cuatros ojos*, or “four eyes.”

The nearly transparent eyelids act like built-in goggles and maintain the clarity of the bird’s vision while underwater. The *Anableps anableps* of Central and South America, shown in ● Figure 6.5, swims with its eyes half in and half out of the water to watch for its prey above the surface and predators below. Each eye has two pupils, one above and one below the water. The upper half of *Anableps*’s eye is designed to see light coming through air, whereas the lower half is adapted to light moving through the water. Local people refer to the fish as *cuatro ojos*, or “four eyes.”

### INTERIM SUMMARY 6.1

#### || Summary Table: Features of Light as a Stimulus

Feature	Significance
Wavelength	Distance between peaks of the waves; determines the perceived color of objects.
Amplitude	Height of the wave; determines perception of brightness.
Absorption	Objects that absorb more visible light energy appear dark colored.
Reflection	Objects that reflect more visible light energy appear light colored. We perceive the reflected wavelengths as the color of an object.
Refraction	Refraction, as by air and water molecules, changes the direction of light.

#### || Summary Points

- Human vision responds to the visible light portion of the electromagnetic radiation spectrum. Electromagnetic radiation can be described in terms of waves or as the movement of large numbers of particles known as photons. (L01)
- Visible light interacts with objects in the environment in ways that make light a useful source of information. Light is plentiful, travels in a fairly straight line, and reflects off many objects. (L01)

### Review Questions

1. What are the advantages of being able to see in the visible light spectrum as compared with other portions of the electromagnetic spectrum?
  2. What do we mean by absorption, reflection, and refraction of light waves?
- 

## The Structure and Functions of the Visual System

We begin our discussion of visual processing at the eye. Animals have different solutions for the placement of the eyes in the head. Some have eyes in front like humans and cats, whereas others have eyes on the sides of the head, as do rabbits and horses. As we will see later in this chapter, having eyes in the front of the head provides superior depth perception that is advantageous for hunting, so this placement is characteristic of predators. The eyes-on-the-side placement is usually found in prey species and allows these animals to scan large areas of the environment for predators while feeding.

### Protecting the Eye

A number of mechanisms are designed to support and protect the eye. Eyes are located in the bony **orbit** of the skull, which can deflect many blows. In addition, the eye is cushioned by fat. When people are starving, they show a characteristic hollow-eyed look due to the loss of this important fat cushion.

A second line of defense is provided by the eyelids. The eyelids can be opened and closed either voluntarily or involuntarily. Involuntary closure of the eyelids, or a **blink**, both protects the eye from incoming objects and moistens and cleans the front of the eye. Under most circumstances, we blink about once every four to six seconds (Burr, 2005). Spontaneous eyeblink rate is correlated with dopamine function and is altered in conditions involving abnormalities in this neurochemical system. Eyeblink rate is lower in both Parkinson's disease and attention deficit hyperactivity disorder (ADHD; see Chapter 16) (Müller et al., 2007).

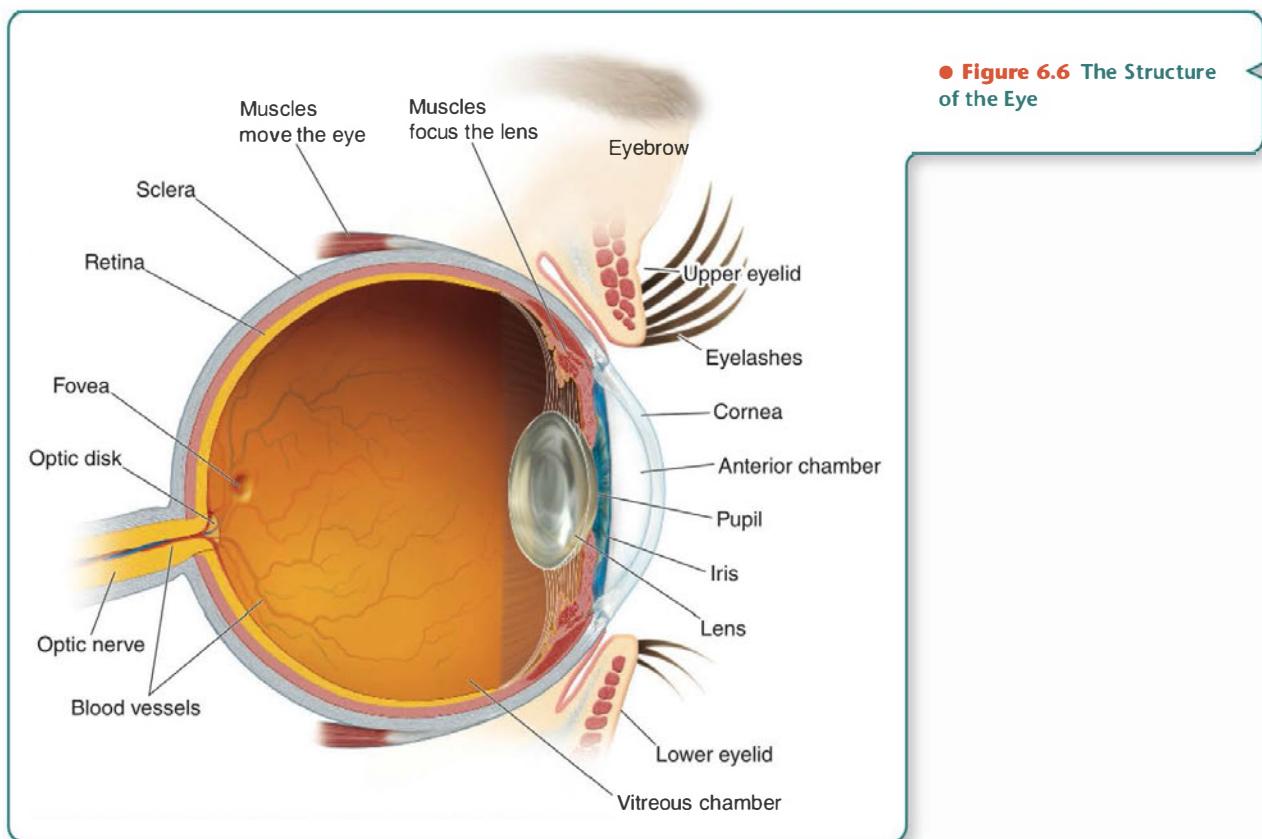
Tears, another feature of the eyes' protective system, are produced in the lacrimal gland at the outer corner of each eye. The fluid not only is composed primarily of water and salt but also contains proteins, glucose, and substances that kill bacteria. Basal tears flush away dust and debris and moisten the eye so that the eyelids don't scratch the surface during blinks. Reflexive tears share a similar composition to basal tears but are released in large quantities in response to chemicals, bright lights, or debris in the eye. Emotional tears contain more hormones than basal or reflex tears, including leu-enkephalin, which acts as a natural painkiller.

### The Functional Anatomy of the Eye

The human eye is roughly a sphere with a diameter of about 24 mm, just under one inch, and individual variations are very small, no more than 1 or 2 mm. Newborns' eyes are about 16–17 mm in diameter (about 6/10 of an inch) and attain nearly their adult size by the age of three years. The "white" of the eye, or **sclera**, provides a tough outer covering that helps the fluid-filled eyeball maintain its shape. The major anatomical features of the eye are illustrated in **Figure 6.6**.

Light entering the eye first passes through the outer layer, or **cornea**. Because the cornea is curved, it begins the process of bending or refracting light to form an image in the back of the eye. The cornea is actually a clear, blood vessel-free extension of the sclera. Special proteins on the surface of the cornea discourage the growth of blood vessels (Cursiefen et al., 2006). The lack of a blood supply and the orderly alignment of

**orbit** The bony opening in the skull that houses the eyeball.  
**blink** A rapid closing of the eyelids.  
**sclera** The white outer covering of the eye.  
**cornea** The transparent outer layer of the eye.



the cornea's fiber structure make it transparent. As living tissue, the cornea still requires nutrients, but it obtains them from the fluid in the adjacent **anterior chamber** rather than from blood. This fluid is known as the **aqueous humor**. The cornea has the dubious distinction of having a greater density of pain receptors than nearly any other part of the body (see Chapter 7).

After light travels through the cornea and the aqueous humor of the anterior chamber, it next enters the **pupil**. The pupil is actually an opening formed by the circular muscle of the **iris**, which comes from the Greek word for "rainbow." The iris adjusts the opening of the pupil in response to the amount of light present in the environment. Pupil diameter is also affected by your emotional state through the activity of the autonomic nervous system (see Chapter 2). Under the influence of the sympathetic division of the autonomic nervous system, the pupil dilates. In times of less arousal during which the parasympathetic nervous system is active, the pupil becomes more constricted.

The color of the iris is influenced primarily by its amount of melanin pigment, which varies from brown to black, in combination with the reflection and absorption of light by other elements in the iris such as its blood supply and connective tissue (see • Figure 6.7). The irises of people with blue or gray eyes contain relatively less melanin than the irises of people with brown eyes. Consequently, some wavelengths are reflected and scattered from the blue or gray iris in ways that are similar to light in the atmosphere, which is also perceived as blue. Green eyes contain a moderate amount of melanin, and brown or black eyes contain the greatest amounts. "Amber" eyes, brown eyes with a golden look, contain an additional yellowish pigment.

Directly behind the iris is the **lens**. The lens helps focus light on the retina in the back of the eye and functions very much like the lens of a camera. Like the cornea, the lens is transparent due to its fiber organization and lack of blood supply. It, too, depends on the aqueous humor for nutrients. Muscles attached to the lens allow us

**anterior chamber** The area of the eye located directly behind the cornea, containing aqueous humor.

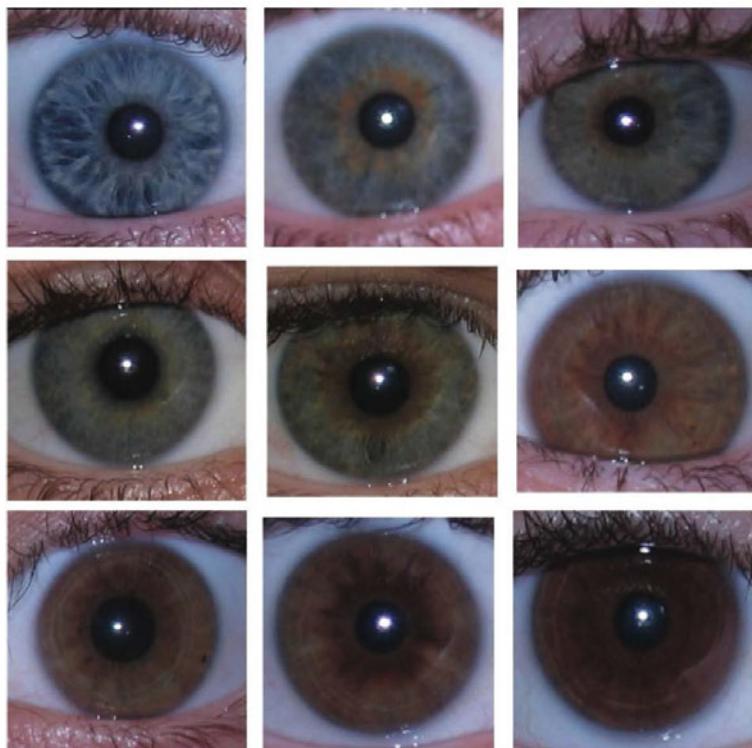
**aqueous humor** The fluid located in the anterior chamber that nourishes the cornea and lens.

**pupil** The opening in the front of the eye controlled by the iris.

**iris** The circular muscle in the front of the eye that controls the opening of the pupil.

**lens** The clear structure behind the pupil and iris that focuses light on the retina.

**Figure 6.7 Iris Colors** The color of the iris is influenced by the amount of melanin pigment, blood supply, and connective tissue. Each person's iris is as individual as a fingerprint.



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to adjust our focus as we look at objects near to us or far away. This process is called **accommodation**.

The major interior chamber of the eye, known as the **vitreous chamber**, is filled with a jellylike substance called **vitreous humor**. Unlike the aqueous humor, which circulates and is constantly renewed, the vitreous humor you have today is the same vitreous humor with which you were born. Under certain circumstances, you can see floaters, or debris, in the vitreous humor, especially as you get older.

Finally, light will reach the **retina** at the back of the eye. The image that is projected on the retina is upside down and reversed relative to the actual orientation of the object being viewed. You can duplicate this process by looking at your image in both sides of a shiny spoon. In the convex or outwardly curving side, you will see your image normally. If you look at the concave or inwardly curving side, you will see your image as your retina sees it. The visual system has no difficulty encoding this image to give us a realistic perception of the actual orientation of objects.

The retina is actually a part of the diencephalon that migrates outward during embryonic development (see Chapter 5). The word *retina* comes from the Latin word for “fisherman’s net.” As the name implies, the retina is a thin but complex network containing special light-sensing cells known as **photoreceptors**. The photoreceptors are located in the deepest layer of the retina. Before light passing through the lens can reach the photoreceptors, it must travel through the vitreous humor, numerous blood vessels, and a number of neural layers. We don’t normally see the blood vessels and neural layers in our eyes due to an interesting feature of our visual system. Our visual system responds to change and tunes out stimuli that remain constant. Because the blood vessels and neural layers are always present, we don’t “see” them.

The blood vessels serving the eye and the axons from the retina forming the optic nerve exit the back of the eye in a place known as the **optic disk**. This area does not contain any photoreceptors at all, which gives each eye a blind spot. Under normal

**accommodation** The ability of the lens to change shape to adjust to the distance of the visual stimulus.

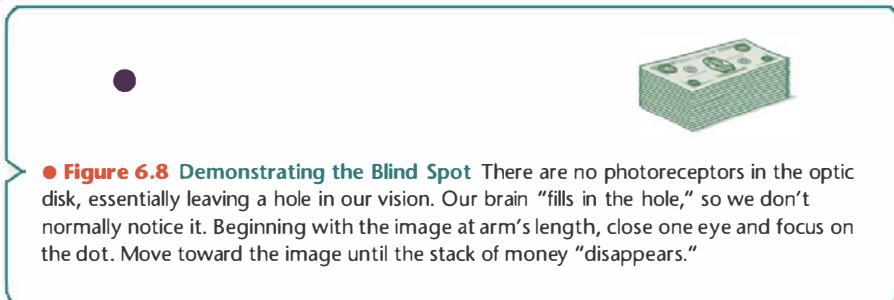
**vitreous chamber** The large inner cavity of the eyeball.

**vitreous humor** The jellylike substance in the vitreous chamber.

**retina** The elaborate network of photoreceptors and interneurons at the back of the eye that is responsible for sensing light.

**photoreceptor** Specialized sensory cell in the retina that responds to light.

**optic disk** The area in the retina where blood vessels and the optic nerve exit the eye.



● **Figure 6.8 Demonstrating the Blind Spot** There are no photoreceptors in the optic disk, essentially leaving a hole in our vision. Our brain “fills in the hole,” so we don’t normally notice it. Beginning with the image at arm’s length, close one eye and focus on the dot. Move toward the image until the stack of money “disappears.”

conditions, we don’t notice these blind spots, but you can find your own by following the directions in ● Figure 6.8.

Toward the middle of the retina, we can see a yellowish area about 6 mm in diameter that is lacking large blood vessels (see ● Figure 6.9). This area is known as the **macula**, from the Latin word for “spot.” When we stare directly at an object, the image of that object is projected by the cornea and lens to the center of the macula. As a result, we say that the macula is responsible for **central vision** as opposed to **peripheral vision**. Peripheral vision is our ability to see objects that are off to the side while looking straight ahead.

In the center of the macula, the retina becomes thin and forms a pit. The pit is known as the **fovea**, which is about 1.8 mm in diameter. In humans, the fovea is particularly specialized for detailed vision and contains only one type of photoreceptor, the cones, which permit vision in bright light. Primates, including humans, are the only mammals whose foveas contain only cones. Other mammals, such as cats, have retinal areas that are similar to a fovea, but these contain both cones and the photoreceptors known as rods, which allow vision in dim light.

The retina is embedded in a pigmented layer of cells called the **epithelium**. These cells support the photoreceptors and absorb random light. Because of this absorption of random light, the interior of the eye looks black when seen through the pupil. When a bright light source, such as a camera flash, is pointed directly at the eye, we see the reflection of the true red color of the retina that results from its rich blood supply. The shine we see reflected from the eyes of some animals at night has a different origin.

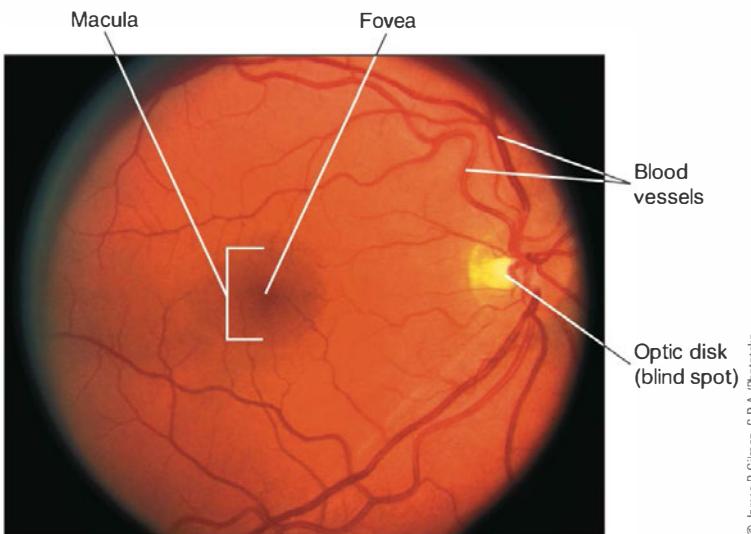
**macula** A 6 mm round area in the retina that is not covered by blood vessels and that is specialized for detailed vision.

**central vision** The ability to perceive visual stimuli focused on the macula of the retina.

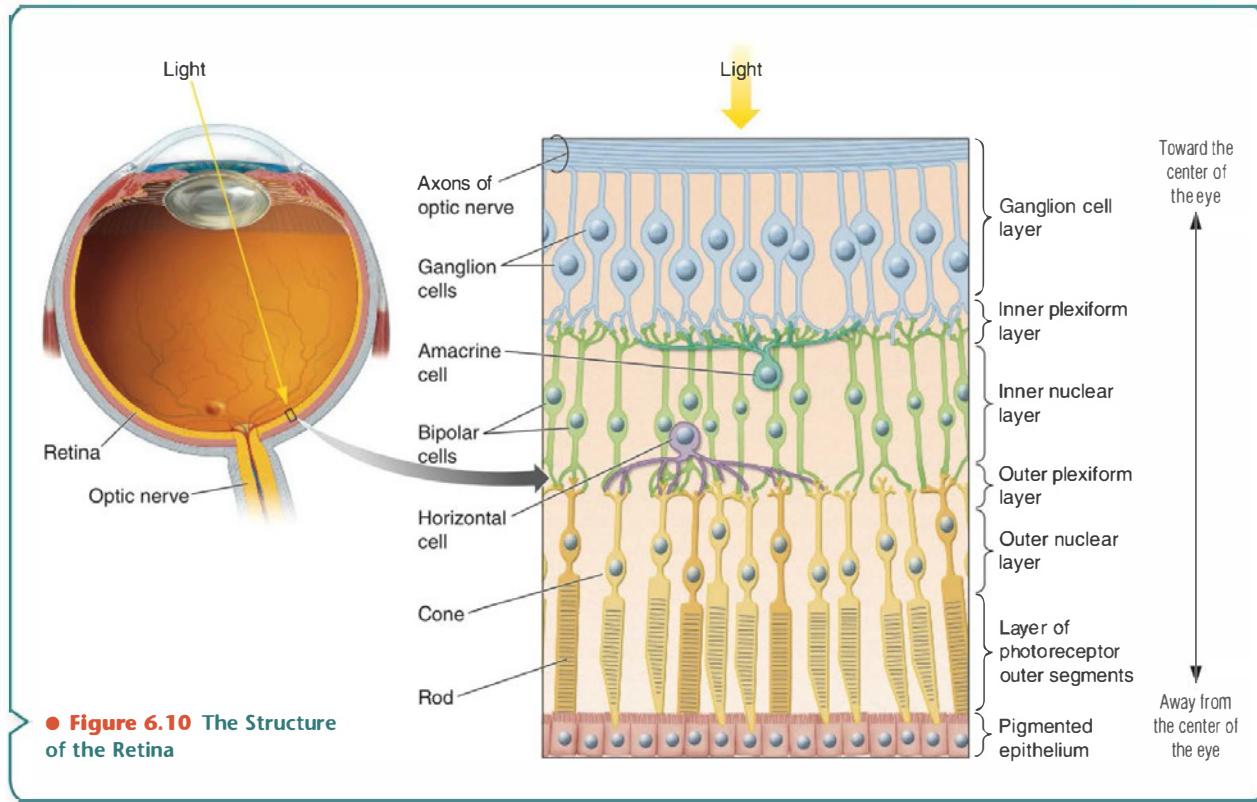
**peripheral vision** The ability to perceive visual stimuli that are off to the side while looking straight ahead.

**fovea** A small pit in the macula specialized for detailed vision.

**epithelium** The pigmented layer of cells supporting the photoreceptors of the retina.



● **Figure 6.9 Landmarks of the Retina** Blood vessels serving the eye and the axons of the optic nerve exit the eye in the optic disk. The macula is an area of the retina not covered by blood vessels. Within the macula is a small pit, known as the fovea. Detailed vision is best for images projected onto the macula and fovea.



**ganglion cell layer** The layer of retinal interneurons farthest from the photoreceptors, which contains ganglion cells and gives rise to the optic nerve.

**ganglion cell** Retinal cell in the ganglion cell layer whose axon leaves the eye as part of the optic nerve.

**inner plexiform layer** The location in the retina containing axons and dendrites that connect the ganglion, bipolar, and amacrine cells.

**amacrine cell** A retinal interneuron in the inner nuclear layer that integrates signals across adjacent segments of the retina.

**bipolar cell** A cell in the inner nuclear layer of the retina that forms part of the straight pathway between the photoreceptors and the ganglion cells.

**inner nuclear layer** The layer of retinal interneurons containing amacrine, bipolar, and horizontal cells.

**outer plexiform layer** The retinal layer containing axons and dendrites forming connections between bipolar cells, horizontal cells, and the photoreceptors.

**horizontal cell** A retinal interneuron located in the inner nuclear layer that integrates signals from across the surface of the retina.

Although it is normally advantageous to reduce reflection in the eye, the epithelium of some nocturnal animals, such as the cat, contains a white compound that acts more like a mirror. By reflecting light through the eye a second time, the odds of perceiving very dim lights at night are improved.

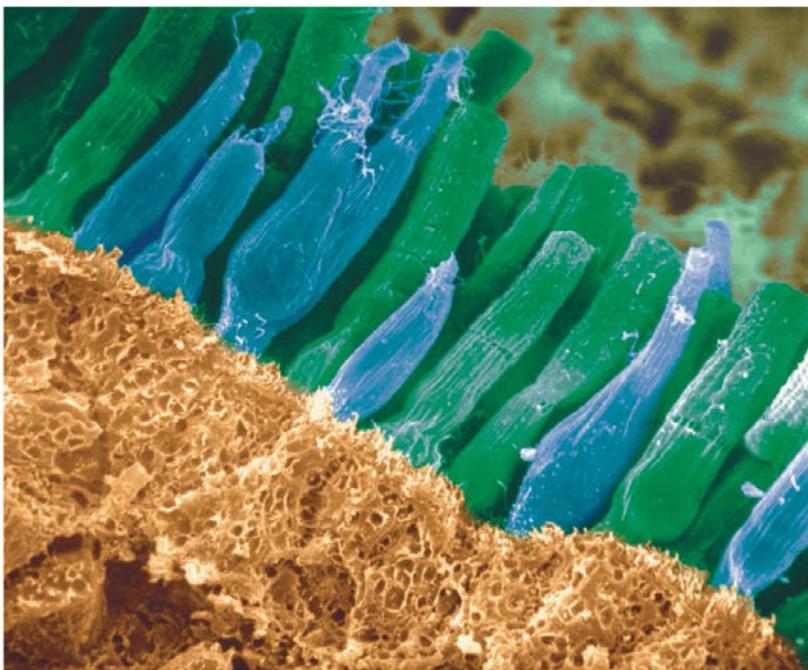
## The Layered Organization of the Retina

Although it is only 0.3 mm thick, the retina contains several layers of neurons and their connections, illustrated in **Figure 6.10**. Three layers of cell bodies are separated by two layers of axons and dendrites.

Beginning toward the center of the eye, the retina's first layer is the **ganglion cell layer**, which contains the **ganglion cells**. Each ganglion cell has a single axon, and these axons form the optic nerve as it leaves the retina. In the **inner plexiform layer**, the dendrites of ganglion cells form connections with the **amacrine** and **bipolar cells**. The cell bodies of the bipolar, amacrine, and horizontal cells are located in the **inner nuclear layer**. In the **outer plexiform layer**, the bipolar cells form connections with **horizontal cells** and the photoreceptors. The **outer nuclear area** contains the cell bodies of the photoreceptors. If you remember that "inner" in this case refers to layers toward the center of the eye, whereas "outer" refers to layers away from the center of the eye, these terms become more reasonable.

## The Photoreceptors

The two types of photoreceptors, **rods** and **cones**, are named according to the shape of their **outer segments**, shown in **Figure 6.11**. The outer segment is the part of the photoreceptor that absorbs light. The outer segment contains **photopigments**, chemicals that interact with incoming light.

**● Figure 6.11 Rods and Cones**

The human eye contains approximately 100 million rods and 3 million cones, named after the shapes of their outer segments.

Each human eye contains about 100 million rods. Rods have a long, cylinder-shaped outer segment containing large numbers of disks, like a large stack of pancakes. These disks contain a photopigment known as **rhodopsin**. Rods are responsible for **scotopic vision**, or the ability to see in dim light. Under ideal conditions, the human eye can see a single photon, or the equivalent of the light from a candle flame 30 miles away on a moonless night (Hecht, Shlaer, & Pirenne, 1942). The cost for this extraordinary sensitivity to light is in the clarity and color of the image provided by the rods. Rods do not provide any information about color, and they do not produce sharp images. At night under starlight, our vision is no better than 20/200. An object seen at night from a distance of only 20 feet would have the same clarity as the object viewed from a distance of 200 feet at high noon. It's a good idea to keep that fact in mind while driving at night.

There are only about 3 million cones in each human eye. Cones are responsible for **photopic vision**, or vision in bright light. Photopic vision is sensitive to color and provides images with excellent clarity. The outer segment of cones is shorter and more pointed than that of the rods. Cones store one of three different photopigments in a folded membrane rather than in disks, as the rods do. Because cones work best in bright light, we do not really see color at night. We might know that we're wearing a green sweater, and, in a sense, we may think it looks green as a result of that memory, but we require fairly bright light and the action of our cones to truly see the color.

Rods and cones respond to a wide range of wavelengths, but their photopigments each have different peak sensitivities (see ● Figure 6.12). There are three classes of cones. The so-called blue or short-wavelength cones, which contain the photopigment cyanolabe, respond maximally to wavelengths of 419 nm (violet). The green, or middle-wavelength cones, containing chlorolabe, have peak responses to 531 nm (green), and the red or long-wavelength cones, containing erythrolabe, peak at 558 nm (yellow) (Dartnall, Bowmaker, & Mollon, 1983; Wald & Brown, 1958). The rhodopsin in rods absorbs photons most effectively at wavelengths of 502 nm (a bluish-green).

**outer nuclear area** The location in the retina containing the cell bodies of the photoreceptors.

**rod** A photoreceptor that responds to low levels of light but not to color.

**cone** A photoreceptor that operates in bright conditions and responds differentially to color.

**outer segment** The portion of a photoreceptor containing photopigments.

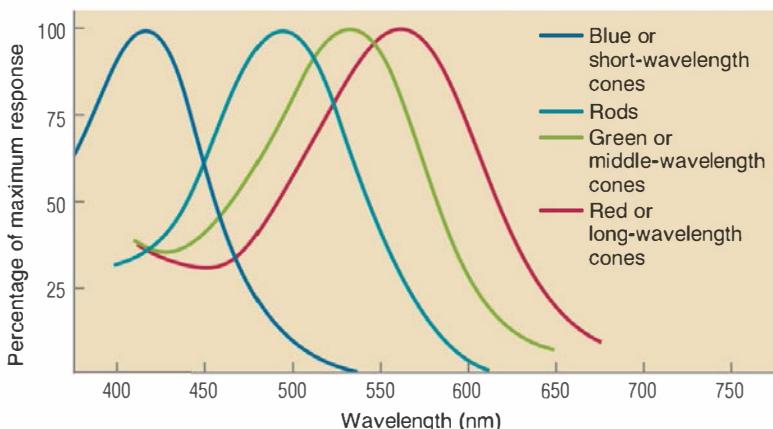
**photopigment** A pigment contained in the photoreceptors of the eye that absorbs light.

**rhodopsin** The photopigment found in rods.

**scotopic vision** The ability to perceive visual stimuli in near darkness due to the activity of rods.

**photopic vision** The ability to perceive visual stimuli under bright light conditions due to the activity of cones.

**Figure 6.12 The Responses of Rods and Cones to Different Wavelengths.** The photopigments contained in rods and cones show peak sensitivities to lights of different wavelengths.



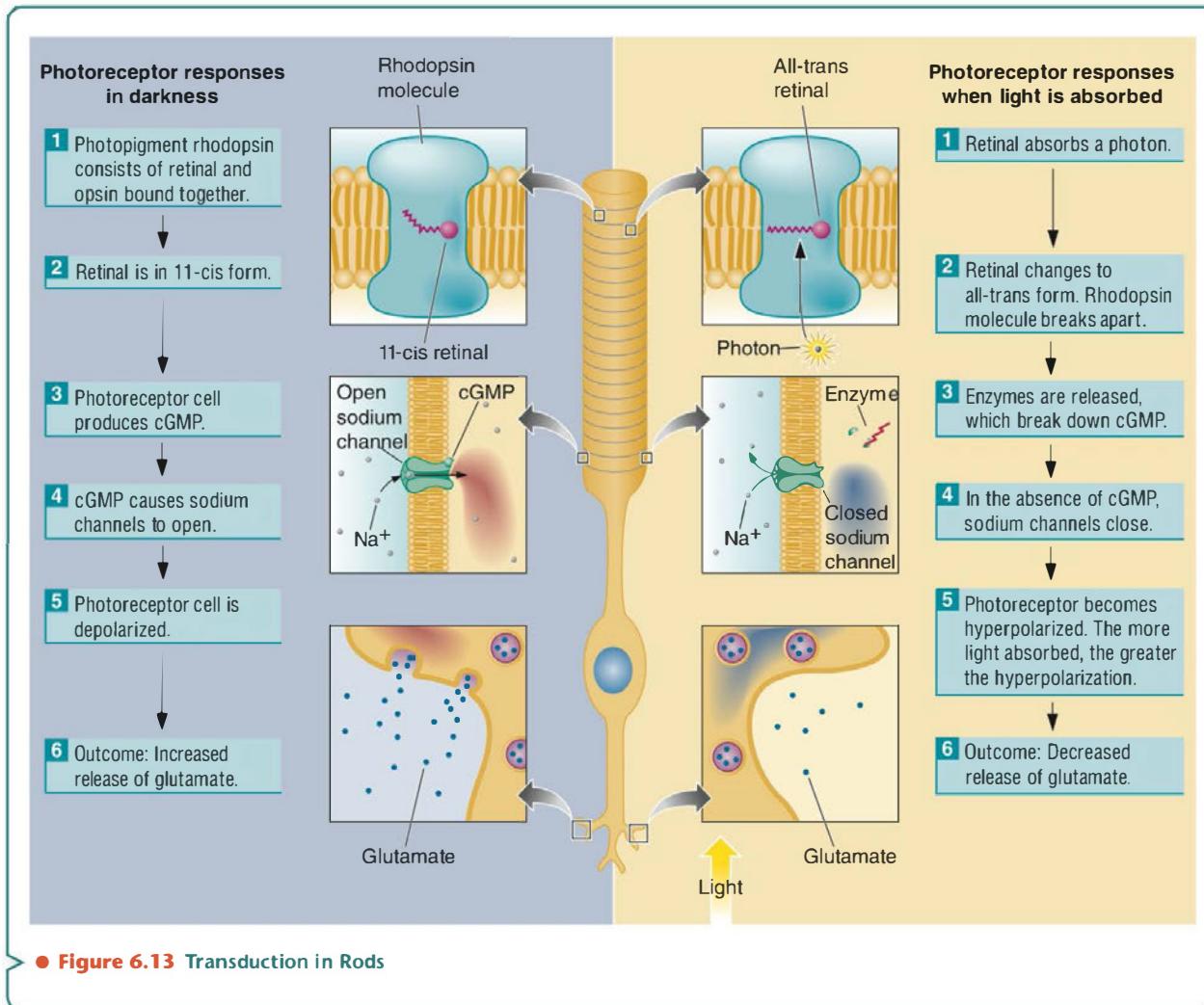
Rods and cones need different amounts of light to respond. Rhodopsin breaks apart when relatively little light has been absorbed, which explains in part the rods' great sensitivity to low levels of light. The cone photopigments are much more resistant to breaking apart and will do so only in the presence of bright light. This is one of the reasons that cones are active in daylight rather than during the night.

As we move from the fovea to the outer margins of the primate retina, the concentration of rods increases and the number of cones decreases. As a result, the center of the retina is superior for seeing fine detail and color in the presence of bright light, whereas the periphery is superior for detecting very dim light. Because of this uneven distribution of rods and cones across the retina, we see better in dim light when we do not look directly at an object. Prior to the invention of night goggles, soldiers traveling at night were trained to look slightly to the side of a location where they suspected enemy movement, rather than straight at the location. Stargazers know that if you look directly at a dim star, it tends to disappear, but if you shift your vision a little to the side, the star becomes visible once again. Table 6.1 summarizes the differences between scotopic and photopic vision.

**TRANSDUCTION BY PHOTORECEPTORS** Photoreceptors transduce light energy into electrical signals that can be sent to the brain for further processing. Because rods and cones transduce light energy in similar ways, we will focus on the process as carried out by rods.

**TABLE 6.1** | Scotopic and Photopic Vision

	Scotopic Vision (Dim Light)	Photopic Vision (Bright Light)
<b>Photoreceptor used</b>	<b>Rods</b>	<b>Cones</b>
Peak wavelength sensitivity	502 nm	420 nm (blue or short-wavelength cones); 530 nm (green or medium-wavelength cones); 560 nm (red or long-wavelength cones)
Ability to distinguish color	None	Color sensitive
Sensitivity to dim light	Excellent	Poor
Acuity	Poor	Excellent
Location of photoreceptors in the retina	Primarily in the periphery	Primarily in the fovea



● Figure 6.13 Transduction in Rods

Rhodopsin, the photopigment found in rods, has two parts, **opsin** and **retinal**. Opsin is a protein chain, and retinal is a chemical made from vitamin A. Vitamin A deficiencies can negatively affect your supply of rhodopsin, so eating carrots, which are rich sources of vitamin A, can truly improve your night vision. When retinal is bound with opsin, the resulting molecule of rhodopsin has a tail that bends at carbon atom number 11. Consequently, this is known as the **11-cis** form of the photopigment. When light enters the eye, photons are absorbed by rhodopsin molecules. As shown in ● Figure 6.13, the absorption of light energy changes the retinal from the 11-cis form to the **all-trans** form. This change in structure causes the rhodopsin molecule to break apart rapidly. To understand what happens next, we need to understand the normal resting state of the photoreceptors.

**THE DARK CURRENT** Photoreceptors operate differently from most neurons. In the generic neuron we discussed in Chapter 3, the membrane potential at rest was approximately  $-70\text{ mV}$ . In contrast, the resting potential of a rod outer segment in complete darkness is about  $-30\text{ mV}$ . In other words, photoreceptors are relatively depolarized (more positive) than typical neurons, even when they are resting. Fesenko, Kolesnikov, and Lyubarsky (1985) discovered that rods are constantly depolarized by

**opsin** A protein found in photopigments.

**retinal** A chemical contained in rhodopsin that interacts with absorbed light.

**11-cis** The form taken by retinal while it is bound to opsin in the absence of light.

**all-trans** The form taken by retinal after light is absorbed by the rod outer segment.

the inward movement of sodium ions through the outer-segment membrane. This movement of positive ions into the resting photoreceptor is known as the **dark current** because it occurs in the dark.

Sodium channels in typical neurons are kept closed when the cells are at rest. However, Fesenko and his colleagues (1985) showed that sodium channels in rods are kept open by a second messenger, **cyclic guanosine monophosphate (cGMP)**, which is constantly produced by the photoreceptor. When rhodopsin molecules break apart after absorbing light energy, enzymes that break down cGMP are released. With less cGMP available, fewer sodium channels remain open, and fewer positive sodium ions enter the cell. The photoreceptor becomes more negative, or hyperpolarized (Baylor, 1987).

When the rod returns to darkness, enzymes stimulate the molecules of retinal and opsin to rejoin as rhodopsin. Rhodopsin takes about 30 minutes to regenerate (Rushton, 1961). This relatively slow regeneration process explains the gradual improvement in vision that takes place when we move from bright sunlight into a darkened theater.

The end result is that photoreceptors are depolarized in the dark and hyperpolarized in the presence of light. Photoreceptors produce graded potentials (signals that vary in size) rather than action potentials. Bright light leads to greater hyperpolarization, whereas dim light leads to less hyperpolarization. Like ordinary neurons, photoreceptors release neurochemicals (the excitatory neurotransmitter glutamate in this case) when depolarized. Photoreceptors release the largest amounts of glutamate while in the dark. When exposure to light produces hyperpolarization, the photoreceptor responds by releasing less glutamate. This might appear counterintuitive to you because stimulation by light is actually reducing the activity of the receptors. Rest assured that the cells downstream of the photoreceptors (bipolar and horizontal cells) are fully capable of sorting out this strange input.

## Processing by Retinal Interneurons

The photoreceptors (rods and cones) are the only true receptor cells in the entire visual system. In Figure 6.10, we saw the four other types of cells in the retina that help process information from the photoreceptors. The bipolar and ganglion cells provide a direct, straight pathway for information from the photoreceptors to the brain that is modified by input from the horizontal and amacrine cells. The horizontal and amacrine cells integrate information across the surface of the retina. You can think of the photoreceptor-bipolar-ganglion connections as running perpendicular to the back of the eye, whereas the horizontal and amacrine connections run parallel to the back of the eye.

**HORIZONTAL CELLS** The horizontal cells are located in the inner nuclear layer. They receive input from the photoreceptors and provide output to another type of cell in the inner nuclear layer, the bipolar cell. The major task of horizontal cells is to integrate information from photoreceptors that are close to one another. The spreading structure of the horizontal cell is well suited to this task. Like the photoreceptors, horizontal cells communicate through the formation of graded potentials rather than of action potentials.

**BIPOLAR CELLS** Bipolar cells, also located in the inner nuclear layer, receive input from photoreceptors and from horizontal cells. In turn, bipolar cells communicate with the amacrine cells in the inner nuclear layer and with ganglion cells. Like the photoreceptors and horizontal cells, bipolar cells produce graded potentials rather than action potentials.

There are two major types of bipolar cells: diffuse and midget. Diffuse bipolar cells are more common in the periphery of the retina, where a single bipolar cell might receive input from as many as 50 rods. In contrast, a midget bipolar cell located in the fovea might receive input from a single cone. This organization contributes to the

**dark current** The steady depolarization maintained by photoreceptors when no light is present.

**cyclic guanosine monophosphate (cGMP)** A second messenger within photoreceptors that is responsible for maintaining the dark current by opening sodium channels.

trade-offs between scotopic and photopic vision. Scotopic vision provides remarkable sensitivity to the presence of dim light at the expense of detail or acuity. Photopic vision does just the opposite. Photopic vision sacrifices sensitivity to dim light while providing highly detailed information. A pinpoint of light falling on any of the 50 photoreceptors connected to a diffuse bipolar cell will influence that cell, making this system very sensitive to dim light. On the other hand, the diffuse bipolar cell is unable to tell the difference between one or several dots of light impacting the rods from which it receives information, reducing the bipolar cell's ability to process fine detail. Light falling anywhere on one or more of its photoreceptors will be treated the same way, providing little if any information about the pattern of light that is present.

In contrast, a midget bipolar cell, influenced by a single cone, can provide information about light falling in a tiny part of the retina. This in turn contributes to the fine detail provided by photopic vision. Any ganglion cell listening to this bipolar cell will be able to localize the light source on the retina with precision. Without the ability to pool information from a number of photoreceptors, however, the midget cell could easily miss the presence of light altogether.

Rather than responding to the total amount of light present, bipolar cells begin the process of identifying contrast, or the relative amount of light falling on one area of the retina as opposed to that falling on another area. Bipolar cells accomplish this task by responding to light falling on photoreceptors located in the bipolar cells' **receptive fields**, a type of organization we will see repeated throughout the visual system as well as in the processing of touch, which is discussed in Chapter 7. Any single visual interneuron, such as a bipolar cell, receives input from one or several photoreceptors located in a specific area on the retina. That area is referred to as the interneuron's receptive field (Hartline, 1938). You can think about the retina as an overlapping mosaic of receptive fields (see Figure 6.14). If a pinpoint light is directed to the retina, it is possible to identify which interneurons are responding to the light by recording their activity. A light stimulus must fit within a cell's receptive field to influence its activity. The cell is "blind" to any light falling outside its receptive field on the retina.

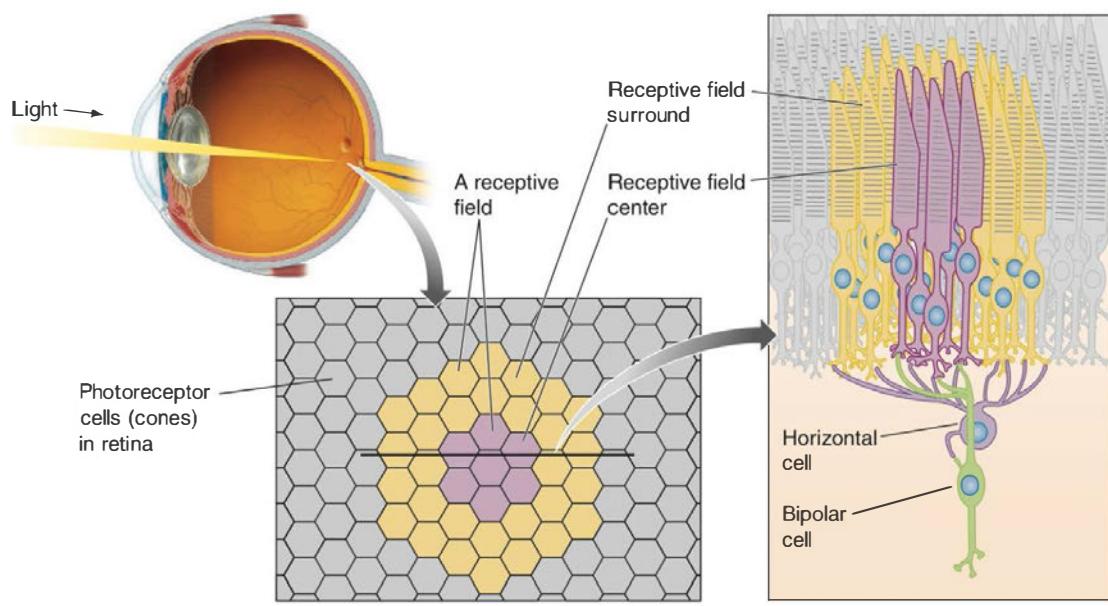
Let's imagine that we are doing a single-cell recording from one bipolar cell to map its receptive field. When we shine a pinpoint of light into our participant's eye, we find that our bipolar cell depolarizes. When we turn the light off, the cell returns to its normal resting status. If we move our light a little bit to the side, then the cell



NS Photography/Shutterstock.com

**Figure 6.14 Visual Receptive Fields** Visual interneurons, including bipolar and ganglion cells (modeled by the spheres), will only respond to light falling on photoreceptors located in the interneuron's receptive field (modeled by the overlapping circles on the image). Light falling outside an interneuron's receptive field has no effect on its activity. The cell is essentially blind to that light.

**receptive field** A location on the retina at which light affects the activity of a particular visual interneuron.



**On-Center/Off-Surround Receptive Field of a Bipolar Cell**

● **Figure 6.15 Retinal Bipolar Cells Have Receptive Fields** An on-center bipolar cell receives direct input from photoreceptors (purple) in the center of its receptive field and indirect input from photoreceptors (yellow) in its surround that communicate first with a horizontal cell that then communicates with the bipolar cell. Light falling on the center and surround of a receptive field always has opposite effects on a cell's activity. In this example, light falling on the center but not the surround depolarizes the cell, while light falling on the surround and not the center hyperpolarizes it. Light falling on both center and surround does not change the cell's activity.

hyperpolarizes. We have discovered that our bipolar cell has three settings: a neutral resting potential in the absence of stimulation, an on response when it depolarizes, and an off response when it hyperpolarizes. The on- and off-response areas of the receptive field make up a doughnut shape on the retina, illustrated in ● Figure 6.15. If we shine light in the center of the doughnut, the bipolar cell depolarizes. Shining a light on the doughnut surrounding the center hyperpolarizes the bipolar cell. A light that covers both center and surround creates an excitatory (“on”) response in the center and an inhibitory (“off”) response in the surround. These effects cancel each other out, and the cell remains neutral.

About half the bipolar cells in the human retina are on-center, and the other half are off-center. On-center cells depolarize when light hits the center of their receptive field. Cells that hyperpolarize when light hits the center of their receptive field are called off-center. Each cone in the fovea makes contact with two bipolar cells, an on-center bipolar cell and an off-center bipolar cell. The on-center cell responds to increases in light falling on the cone, while the off-center cell responds to a decrease in light falling on the cone.

Activity in individual photoreceptors contributes simultaneously to both centers and surrounds of the receptive fields of bipolar cells. Photoreceptors forming synapses with a bipolar cell respond to light in the center of that bipolar cell’s receptive field. These same photoreceptors, however, can also influence nearby bipolar cells through connections with horizontal cells. In these cases, the photoreceptors respond to light falling in the bipolar cells’ surrounds.

The arrangement of receptive fields is referred to as an **antagonistic center-surround organization**. The response of a bipolar cell depends on the amount of light

**antagonistic center-surround organization** A characteristic of visual interneuron receptive fields, in which light illuminating the center has the opposite effect on the cell's activity as light in the surround.

falling on its center relative to the amount of light falling on its surround. It is called antagonistic because light falling on the center of the receptive field always has the opposite effect on the cell's activity from light falling on the surround.

Photoreceptors and horizontal cells serving the center (doughnut hole) and surround (the doughnut) compete with each other to activate the bipolar cell in a process known as **lateral inhibition**. *Lateral* means that the process occurs across or parallel to the surface of the retina. In lateral inhibition, active photoreceptors and horizontal cells limit the activity of neighboring, less active cells. This produces a sharpening, or exaggeration, of the bipolar cells' responses to differences in light falling on adjacent areas. Through lateral inhibition, bipolar cells begin to identify the boundaries of a visual stimulus by making comparisons between light levels falling in adjacent areas of the retina. The message sent by the bipolar cells is "I see an edge or boundary."

**AMACRINE CELLS** Amacrine cells, also located in the inner nuclear layer, form connections with bipolar cells, ganglion cells, and other amacrine cells. Amacrine cells take as many as 40 different shapes and release a variety of neurochemicals, though most release the inhibitory neurotransmitters GABA and glycine (see Chapter 4). Like the horizontal cells, amacrine cells integrate visual messages across the retina, and they also process changes in light as a function of time. This latter ability might contribute to our understanding of visual movement. The amacrine cells have the ability to distinguish between the retinal images produced by movement of observed objects and movement of the eye itself (Ölveczky, Baccus, & Meister, 2003). It is likely that amacrine cells participate in additional functions that are not yet fully understood.

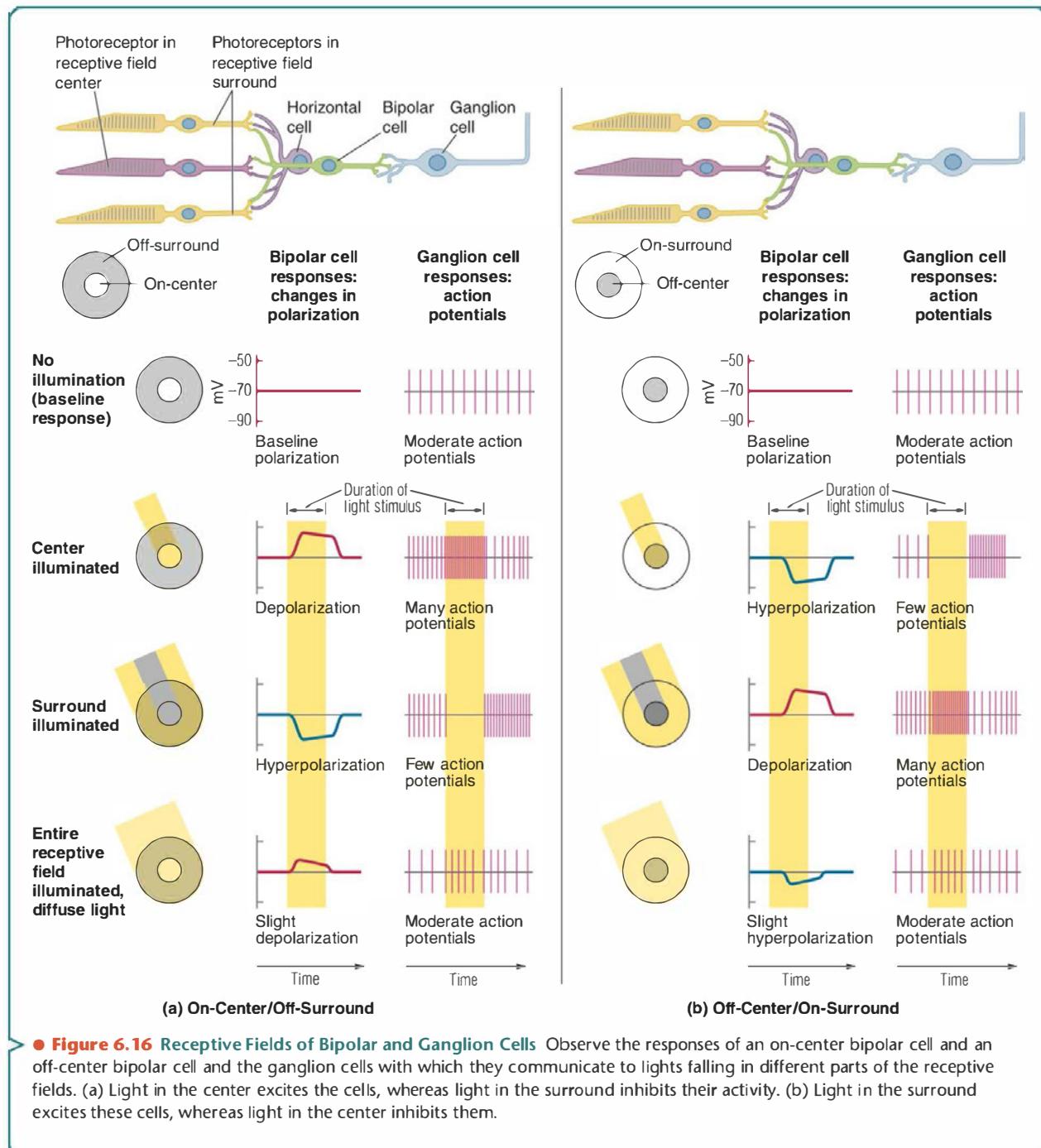
**GANGLION CELLS** Ganglion cells receive input from bipolar and amacrine cells. The axons of ganglion cells leave the eye to form the optic nerve, which travels to higher levels of the brain. Unlike the interneurons and photoreceptors of the retina discussed so far, ganglion cells form conventional action potentials rather than graded potentials. However, ganglion cells are never completely silent. They fire approximately once per second in the absence of stimulation. The presence of light simply changes the ganglion cells' spontaneous rate of signaling. Special types of ganglion cells contain the photopigment melanopsin, and act as photoreceptors themselves. Information from these ganglion cells travels to the hypothalamus and helps maintain our sleep-waking cycles, discussed further in Chapter 11.

The human eye has approximately 1.25 million ganglion cells, yet they must accurately integrate and communicate input from about 103 million photoreceptors. The ganglion cells accomplish this editing task through the organization of their own receptive fields.

**GANGLION RECEPTIVE FIELDS** Ganglion receptive fields show the same antagonistic center-surround organization that we observed in the receptive fields of bipolar cells. As shown in Figure 6.16, ganglion cells replicate the information passed to them by the bipolar cells. On-center bipolar cells connect to on-center ganglion cells, whereas off-center bipolar cells connect to off-center ganglion cells. Instead of depolarizing or hyperpolarizing like the bipolar cells, ganglion cells change their rates of producing action potentials.

Ganglion receptive fields have two important implications for visual processing. First, each ganglion cell responds most to a dot of light of a specific size and less to dots that are larger or smaller than this "ideal." This feature of ganglion cells suggests that they act as a filter for visual information sent to the brain rather than a passive transmitting system. Second, the ganglion cells report on the differences between light falling in the center and surround rather than on the average amount of light falling on the retina. This means that they are sensitive to the contrast between the brightness of adjacent parts of the visual field. Looking at the same page of a book indoors under

**lateral inhibition** The ability of an active neuron to inhibit the activity of adjacent neurons.



artificial light and then outside on a park bench will provide the retina with different average amounts of light. However, you will still see the black letters against the white page pretty much the same way under both conditions due to the ganglion cells' abilities to report on contrast.

Ganglion cell receptive fields vary in size. Receptive fields vary from 0.01 mm in diameter in the macula to 0.5 mm (50 times larger) in the periphery of the retina. Cells with small receptive fields respond best to fine detail.

**TABLE 6.2 |** The Three Types of Ganglion Cells

	<b>M Cells</b>	<b>P Cells</b>	<b>K Cells</b>
Ganglion cells (%)	8–10 percent	70 percent	~20 percent
Apparent purpose	To detect large, low-contrast objects and movement	To provide detailed information about motionless objects, including color	To provide information about color
Response to color	None	Red-green	Blue-yellow
Destination in lateral geniculate nucleus (LGN) of thalamus	Magnocellular layers	Parvocellular layers	Koniocellular layers
Size	Large	Small	Small
Speed	Fast	Slower	Slower
Receptive field size	Large	Small	Small
Sensitivity to contrast	Sensitive to low contrast	Sensitive to high contrast	Sensitive to high contrast
Response to movement	Excellent	Poor	Poor

**THE THREE TYPES OF GANGLION CELLS** About 70 percent of human ganglion cells are **P cells** (P stands for parvocellular, or small cells), 8–10 percent are **M cells** (M stands for magnocellular, or big cells), and the remaining 20 percent or so are **K cells** (K stands for koniocellular).

Differences among M, P, and K cells are summarized in Table 6.2. M cells are larger than P cells and have thicker, faster axons. M cells have larger receptive fields than P cells. M cells receive input from diffuse bipolar cells, while P cells receive input from midget bipolar cells. M cells respond to smaller differences in light between the center and surround, whereas P cells require a greater difference. This implies that M cells respond to subtle differences of contrast such as when viewing gray letters on a black background. P cells respond to larger differences in contrast such as when viewing black letters on a white background. M cells, but not P cells, respond to stimuli that are turned on and off rapidly, such as the flicker of an old television. A final difference is that P cells respond only to lights of a particular color, whereas M cells respond to light regardless of its color. K cells share most of the characteristics of P cells. Many K cells receive input from blue cones, and thus participate in a blue-yellow pathway.

The end result of these differences is that M cells are responsible for providing information about large, low-contrast, moving objects, whereas P cells are responsible for information about smaller, high-contrast, colorful objects. This distinction between magnocellular and parvocellular systems is preserved up through some of the highest levels of cortical visual processing.

## Optic Nerve Connections

The ganglion cell axons exit the eye through the optic disk, forming an optic nerve leaving each eye. The optic nerves preserve the organization of the retina. In other words, axons from adjacent ganglion cells remain next to one another in the optic nerves.

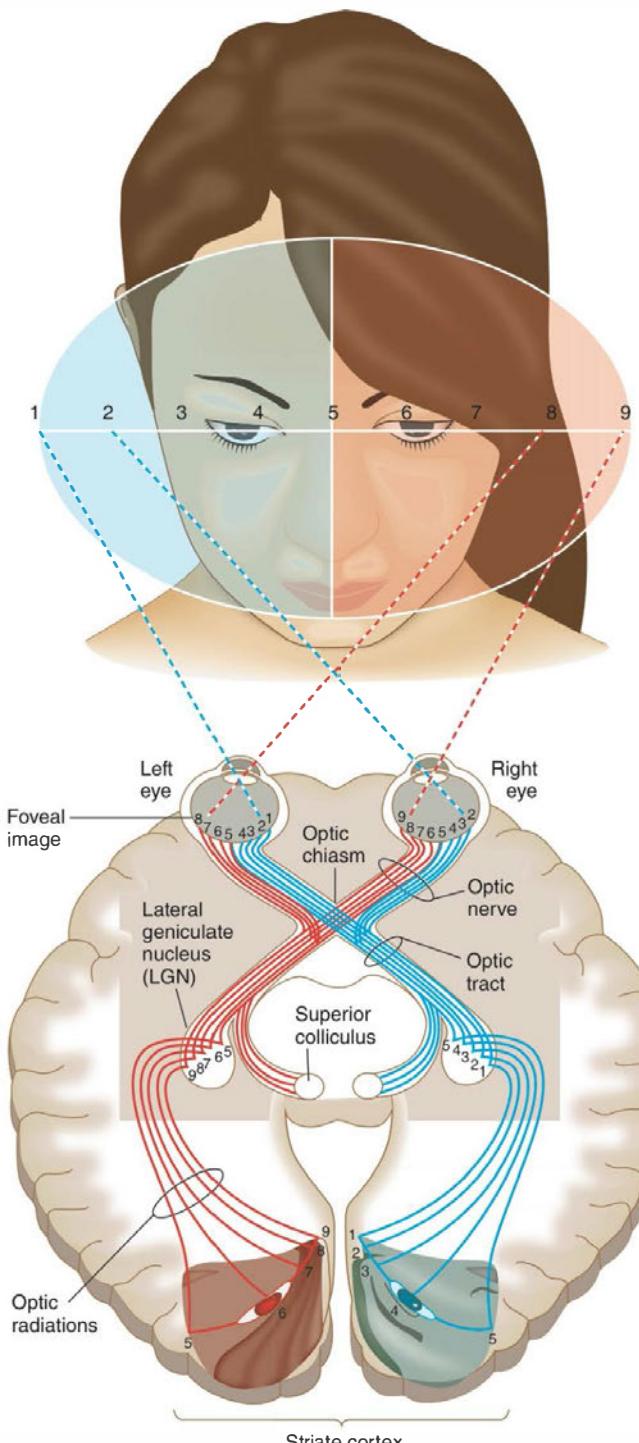
As shown in Figure 6.17, each human optic nerve divides in half, with the outer half (away from the nose) continuing to travel to the same side of the brain (ipsilaterally) while the inner half (closest to the nose) crosses to the other side of the brain (contralaterally). This partial crossing ensures that information from both eyes regarding

**P cell** Small ganglion cell that responds to high contrast and color.

**M cell** Large ganglion cell that responds to all wavelengths regardless of color, subtle differences in contrast, and stimuli that come and go rapidly.

**K cell** A ganglion cell that does not fit the criteria for P or M cells exactly and responds to blue and yellow light.

**• Figure 6.17 The Pathways from Eye to Cortex** The optic nerves leaving the human eye partially cross at the optic chiasm. As a result, images in the left visual field seen by both eyes go to the right hemisphere, whereas images from the right visual field go to the left hemisphere. From the optic chiasm, most axons in the optic tracts synapse in the lateral geniculate nucleus (LGN) of the thalamus. The LGN sends visual information to the primary visual cortex of the occipital lobe.



the same part of the visual field will be processed in the same places in the brain. If you hold your eyes steady by looking at a focal point straight ahead, information from the visual field to the left of the focal point will be transmitted to the right hemisphere. Information from the visual field to the right of the focal point will be transmitted to

the left hemisphere. In humans, about 50 percent of the fibers from each eye cross to the opposite hemisphere. In rabbits and other animals with eyes placed on the side of the head, 100 percent of the fibers cross the midline to the opposite side. Because each of a rabbit's eyes sees a completely different part of the rabbit's visual field, there is no need for the rabbit's brain to reorganize the input.

The optic nerves cross at the **optic chiasm** (named after its X shape, or *chi* in Greek). The nerves continue past the optic chiasm as the **optic tracts**. Almost 90 percent of the axons in the optic tract proceed to the thalamus, which in turn projects to the primary visual cortex located in the occipital lobe of the brain. However, axons from the special light-sensitive ganglion cells that release melanopsin leave the optic tract and synapse in the suprachiasmatic nucleus of the hypothalamus, providing the light information used to regulate daily rhythms (see Chapter 11). Another 10 percent of the axons in the optic tract project to the **superior colliculus** in the midbrain.

**THE SUPERIOR COLICULUS** In many species, including frogs and fish, the superior colliculus is the primary brain structure for processing visual information. Because humans have a cerebral cortex for this purpose, we use the superior colliculus to guide movements of the eyes and head toward newly detected objects in the visual field. The superior colliculus also receives input from the visual cortex, which moderates its activity.

**THE LATERAL GENICULATE NUCLEUS OF THE THALAMUS** Nearly 90 percent of optic tract axons form synapses in the **lateral geniculate nucleus (LGN)**, located in the dorsal thalamus. The LGN is a layered structure that is bent in the middle. The bend is the source of the name *geniculate*, which comes from the Latin for “bent knee” (as does the term *genuflect*, which describes the bending of the knee that Catholics perform prior to entering a pew to worship).

In primates, including humans, the LGN has about the same area as a credit card, but is about three times thicker. The LGN features six distinct stacked layers, numbered from ventral to dorsal. Each layer of the LGN processes a map of one half of the visual field. This mapping allows us to make connections between neural activity and the real world in front of our eyes. Layers 1 and 2 (the most ventral layers) contain larger neurons than the other four layers. These **magnocellular layers** receive input from the M cells in the retina. The other four are referred to as **parvocellular layers**, which receive input from the P cells. Between each of the six layers are very small neurons making up the **koniocellular layers**, which receive input from the K cells. The LGN keeps information from the two eyes completely separate. Figure 6.18 shows how alternating layers of the LGN receive input from the ipsilateral and contralateral eyes.

Neurons in the LGN show the same antagonistic center-surround organization of receptive fields that we observed in the retinal bipolar and ganglion cells. In LGN neurons, however, the lateral inhibition between center and surround is much stronger than we observed among retinal cells. This greater inhibition causes cells in the LGN to amplify or boost the contrast between areas of light and dark.

The exact role of the LGN in visual processing is not well understood, but this area is far from just a passive relay station in the flow of information to the cortex. The LGN receives much more information from the rest of the brain than it sends to the cortex. LGN activity changes in response to different levels of awareness and selective attention, suggesting that the LGN filters information before sending it along to the cortex (Saalmann & Kastner, 2011). The LGN also receives input from the brainstem reticular formation. This input from the cortex and the reticular formation modifies the flow of information to the cortex from the LGN based on levels of arousal and alertness (Saalmann & Kastner, 2011). For example, when you're asleep, someone could lift your eyelid, but you would not “see” (assuming you remained asleep).

**optic chiasm** The area at the base of the brain where the optic nerves cross to form the optic tracts; the location of a partial decussation of the optic nerves in humans.

**optic tracts** The fiber pathways between the optic chiasm and destinations in the forebrain and brainstem.

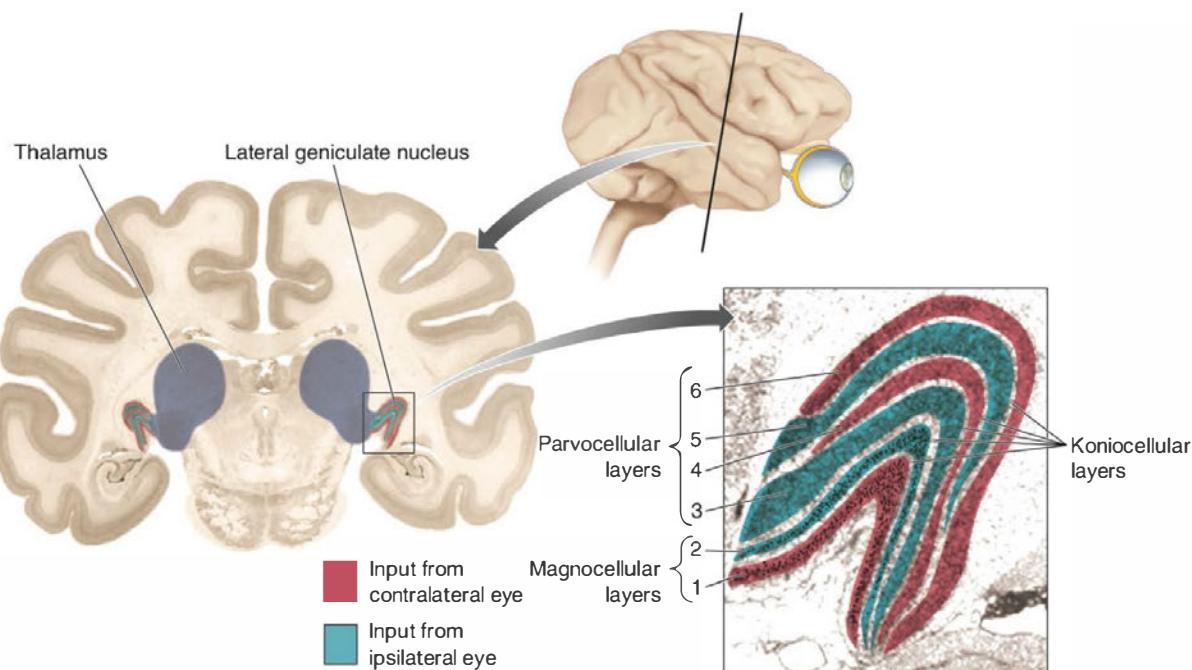
**superior colliculus** A structure in the tectum of the midbrain that guides movements of the eyes and head toward newly detected objects in the visual field.

**lateral geniculate nucleus (LGN)** The nucleus within the thalamus that receives input from the optic tracts.

**magnocellular layers** The two ventral layers of the LGN that receive input from M cells in the ganglion layer of the retina.

**parvocellular layers** The four dorsal layers of the LGN that receive input from P cells in the ganglion layer of the retina.

**koniocellular layers** Layers of very small neurons between the larger six layers of the lateral geniculate nucleus that receive input from K cells in the ganglion layer of the retina.



**► Figure 6.18 The Lateral Geniculate Nucleus** Information from each eye is processed separately in the lateral geniculate nucleus (shown here in the brain of a monkey). Layers 1 and 2 receive input from M ganglion cells in the retina, Layers 3 through 6 receive input from the P ganglion cells, and the koniocellular layers receive input from the K ganglion cells.

## The Striate Cortex

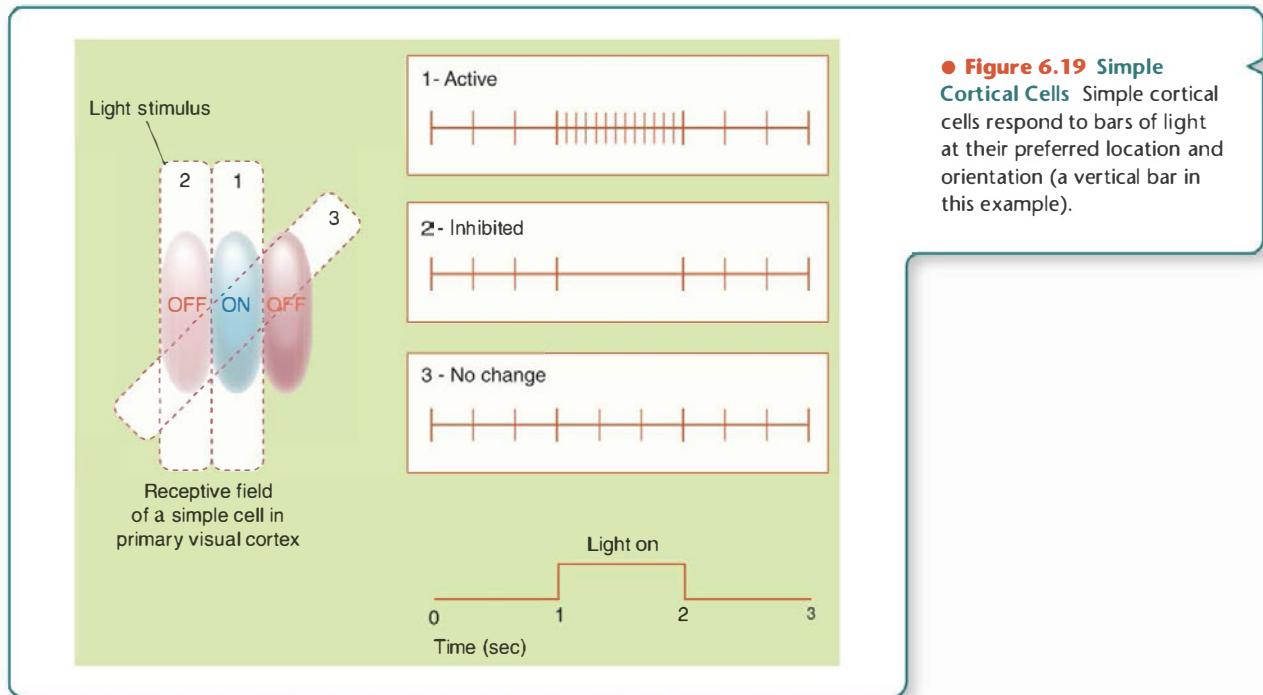
Primary visual cortex is often referred to as **striate cortex**, due to its striped appearance, or as simply V1 (visual area 1). Striate cortex, located in the occipital lobe, contains approximately 200 million neurons as opposed to the 2 million neurons found in the LGN.

The cortex in this area ranges from 1.5 to 2 mm in thickness, or about the height of the letter m on this page. Like other areas of cortex, the striate cortex has six distinct layers (see Chapter 2). Compared with other areas of cortex, striate cortex is relatively thicker in layers II and IV, which receive most of the input from other parts of the brain. Layer IV receives input from the LGN. Striate cortex is thinner than other areas of the cortex in layers III, V, and VI, which contain output neurons that communicate with other parts of the brain.

**CORTICAL MAPPING OF THE VISUAL WORLD** Earlier, we described how precisely the visual field was mapped by the LGN. The same thing is true of the striate cortex. Returning to Figure 6.17, you can trace the pathways from eye to striate cortex that are responding to reflected light from the woman's right eyebrow (numbers 3 and 4). This mapping allows us to use the location of neural activity to understand the position of an object in the visual field.

Figure 6.17 demonstrates another feature of cortical mapping. The areas of the cortex that respond to input from the fovea of the retina (4, 5, and 6) are much larger than the areas responding to images seen in the periphery (1, 2, 3, 7, 8, and 9). Although the fovea contains 0.01 percent of the retina's total area, signals from the fovea are processed by 8 to 10 percent of the striate cortex (Van Essen & Anderson, 1995). This cortical magnification is yet another reason why focusing an image onto the fovea provides the greatest amount of fine detail.

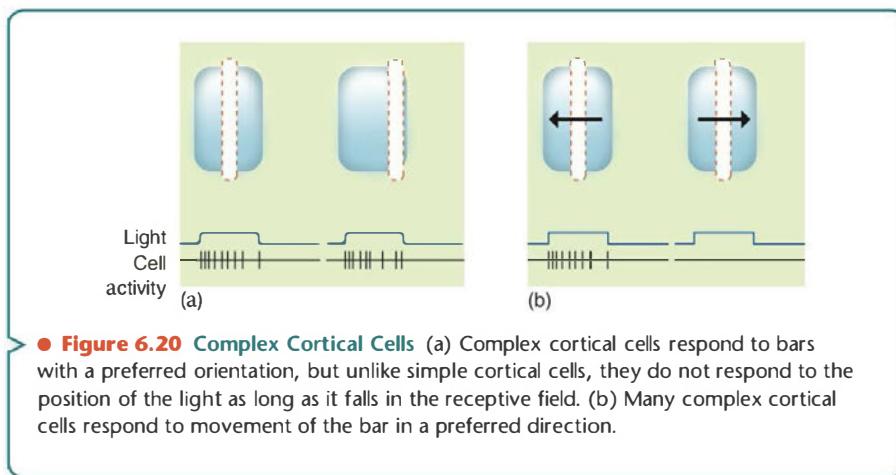
**primary visual cortex/striate cortex** The location in the occipital lobe for the initial cortical analysis of visual input. Also known as V1 (visual area 1).



**● Figure 6.19 Simple Cortical Cells** Simple cortical cells respond to bars of light at their preferred location and orientation (a vertical bar in this example).

**CORTICAL RECEPTIVE FIELDS** Although cortical neurons have receptive fields, these fields do not respond to the simple dots of light that activate bipolar and ganglion cells in the retina and cells within the LGN.

Based on a series of meticulous single-cell recording experiments, Hubel and Wiesel (1959) defined **simple cortical cells** as those cells that respond to stimuli shaped like bars or edges that have a particular slant or orientation in a particular location on the retina (see ●Figure 6.19). These cells probably help us respond to object shape. Simple cortical cell receptive fields maintain an antagonistic center-surround organization, but the shape of the receptive field is more elongated or racetrack-shaped than doughnut-like. Hubel and Wiesel defined **complex cortical cells** as cortical cells that share the simple cells' preference for stimulus size and orientation but without reference to the stimulus's location, as long as it appears in the receptive field (see ●Figure 6.20).



**● Figure 6.20 Complex Cortical Cells** (a) Complex cortical cells respond to bars with a preferred orientation, but unlike simple cortical cells, they do not respond to the position of the light as long as it falls in the receptive field. (b) Many complex cortical cells respond to movement of the bar in a preferred direction.

**simple cortical cell** A cortical interneuron that responds to stimuli in the shape of a bar or edge with a particular slant or orientation in a particular location on the retina.

**complex cortical cell** A cortical interneuron that shows a preferred stimulus size and orientation, and in some cases direction of movement, but not location within the receptive field.

Some complex cortical cells respond to lines moving in a particular direction. A cell might respond to a vertical line moving from right to left across the receptive field but not to a line moving left to right. Consequently, complex cortical cells probably participate in the perception of movement (Regan, Beverley, Cynader, & Lennie, 1979). Fatigue in these directional cells might be responsible for the waterfall illusion (Adams, 1834). If you stare at a waterfall for a minute or two, then look away, the scene being viewed will appear to be moving in an upward direction. This phenomenon occurs due to the temporary fatigue of downward motion detectors.

## Connecting to Research

### HUBEL AND WIESEL MAP THE VISUAL CORTEX

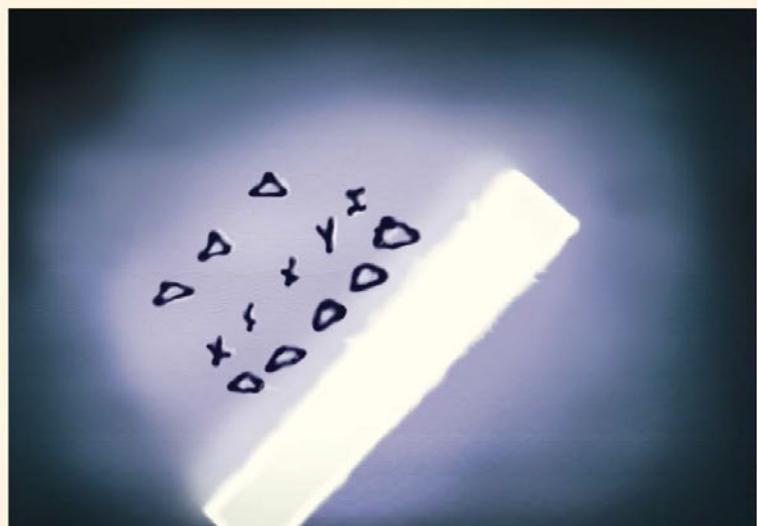
Much of what we know about the organization of the visual cortex was discovered by David Hubel (1926–2013) and Torsten Wiesel (1924–). In a partnership that spanned decades, these researchers painstakingly explored the activity of single cells as they responded to visual stimuli. Their work culminated in their being awarded the 1981 Nobel Prize in Physiology or Medicine.

In one of their early experiments, Hubel and Wiesel observed the activity of single cells in 40 anesthetized cats that were maintained in a stage of “light sleep” according to their EEGs (Hubel & Wiesel, 1962). The cats faced a screen about 4 feet away, on which Hubel and Wiesel projected various patterns of white light (see **Figure 6.21**). Microelectrodes were surgically inserted into the cats’ striate cortex to a depth of about 3–4 mm. As the electrodes were advanced slowly, both moving and stationary stimuli were rapidly presented to see which type of stimulus produced the maximum response by cells near the recording electrode’s tip.

Hubel and Wiesel knew from previous research that retinal cells and cells in the LGN had on- and off-center receptive fields, yet no such organization had ever been demonstrated in the cerebral cortex. They set out to identify whatever organization might characterize this part of the visual system. In the course of this exploration, they found that “the great majority of fields seem to fall naturally into two groups, which we have termed ‘simple’ and ‘complex’” (Hubel & Wiesel, 1962, p. 109). They classified 233 out of 303

cortical cells they evaluated as simple, and the remaining 70 as complex.

In this same report, Hubel and Wiesel discussed orientation columns and ocular dominance columns. Their article featured precise, handmade drawings of the paths of their electrodes and the resulting activity of the cells. They noted that much larger areas of cortex responded to light in the fovea compared to light in the periphery of the retina. This wide-ranging set of results continues to be relevant to our understanding of the visual system 50 years after it was published.



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**Figure 6.21 Stimuli Used by Hubel and Wiesel** While conducting single cell recordings from the visual cortex of an anesthetized cat, Hubel and Wiesel listened for the cell’s response as they moved bars of light across a screen in front of the cat. With marking pens, they indicated areas where a bar produced the biggest response from a cell with x’s and adjacent areas where the cell became silent with o’s. Through this methodical procedure, they were able to map the receptive fields of simple and complex cortical cells in V1.

Some simple and complex cortical cells are also known as **end-stopped cells** (Hubel & Wiesel, 1962). These cells respond most vigorously to a stimulus that falls within their receptive field but not beyond. In other words, a bar of light that is too long for the receptive field would produce a smaller response than a bar that fit the receptive field perfectly. These cells participate in our detection of boundaries like the corners of the page of your textbook. Viewing corners and similar boundaries is essential to the recognition of familiar objects (Biederman, 1987).

**CORTICAL COLUMNS** If we moved a recording electrode perpendicular to the surface of the cortex, we would find that the neurons are organized in a cortical column. Neurons in cortical columns communicate with one another but do not form many connections with neighboring columns more than half a millimeter away (Mountcastle, 1978).

One type of column found in the striate cortex is known as an **ocular dominance column**. These are columns of cortex perpendicular to the cortical surface that respond to input from either the right eye or the left eye but not both. These columns take advantage of the strict segregation of input from either eye that we observed in the LGN. The columns are about 1 mm wide and alternate (right eye–left eye–right eye, etc.) across the surface of the visual cortex.

**Orientation columns** are much thinner than the ocular dominance columns. Each orientation column responds to lines of the same angle. Adjacent columns respond to angles shifted about 10 degrees. A set of these columns that responds to a complete rotation of 180 degrees is referred to as a **hypercolumn**.

**CYTOCHROME OXIDASE BLOBS** **Cytochrome oxidase blobs** are named after an enzyme, cytochrome oxidase (Hubel & Livingstone, 1987; Livingstone & Hubel, 1984; Wong-Riley, 1989). Neurons in areas with high concentrations of cytochrome oxidase appear to process information regarding color.

**CORTICAL MODULES** We have seen how cortical neurons respond to line orientation, movement, and color. At some point, our visual system puts these separate characteristics back together to form coherent images. Hubel and Wiesel (1962) suggested that the unit responsible for this integration is the **cortical module**. As shown in Figure 6.22, cortical modules include two sets of ocular dominance columns, 16 blobs, and two hypercolumns, each responding to the entire 180 degrees of line orientation. As shown in Figure 6.23, newer mapping techniques suggest that modules are more accurately viewed as approximations because the boundaries between modules are much less precise than suggested by Hubel and Wiesel (Blasdel, 1992).

We have about 1,000 modules, and each one makes up a  $2\text{ mm} \times 2\text{ mm}$  area of primary visual cortex. Each module contains the neurons it needs to process the shape, color, and movement of an image falling on a specific part of the retina. You can think of the visual field as a mosaic with 1,000 tiles, each served by a different cortical module.

## Visual Analysis beyond the Striate Cortex

The striate cortex begins, but by no means finishes, the task of processing visual input. At least a dozen additional areas of the human cerebral cortex participate in visual processing. Because these areas are not included in the striate cortex, they are often referred to as extrastriate (*extra* means outside) areas. These areas are also referred to as secondary visual cortex.

Next to the striate cortex is an area known as V2 (visual area 2). If you stain V2 for cytochrome oxidase, a pattern of stripes emerges. Alternating thick and thin stripes are separated by interstripe regions. The thick stripes form part of the magnocellular

**end-stopped cells** A cortical interneuron that responds most vigorously to a stimulus that does not extend beyond the boundaries of its receptive field.

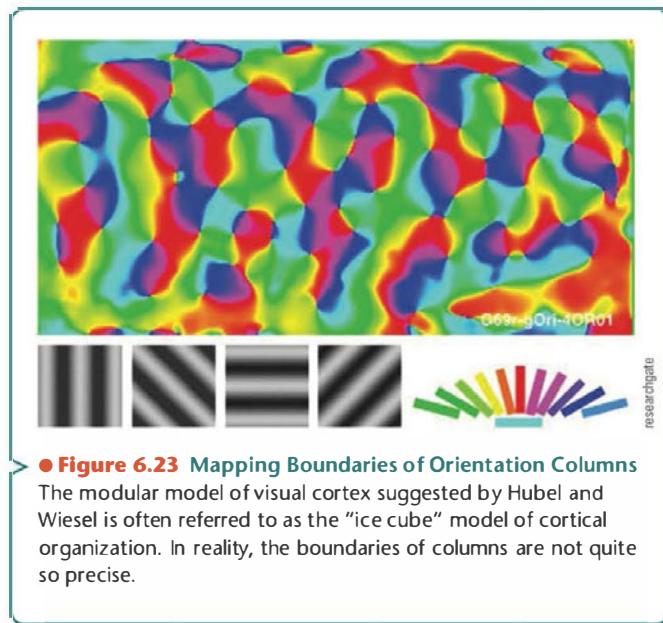
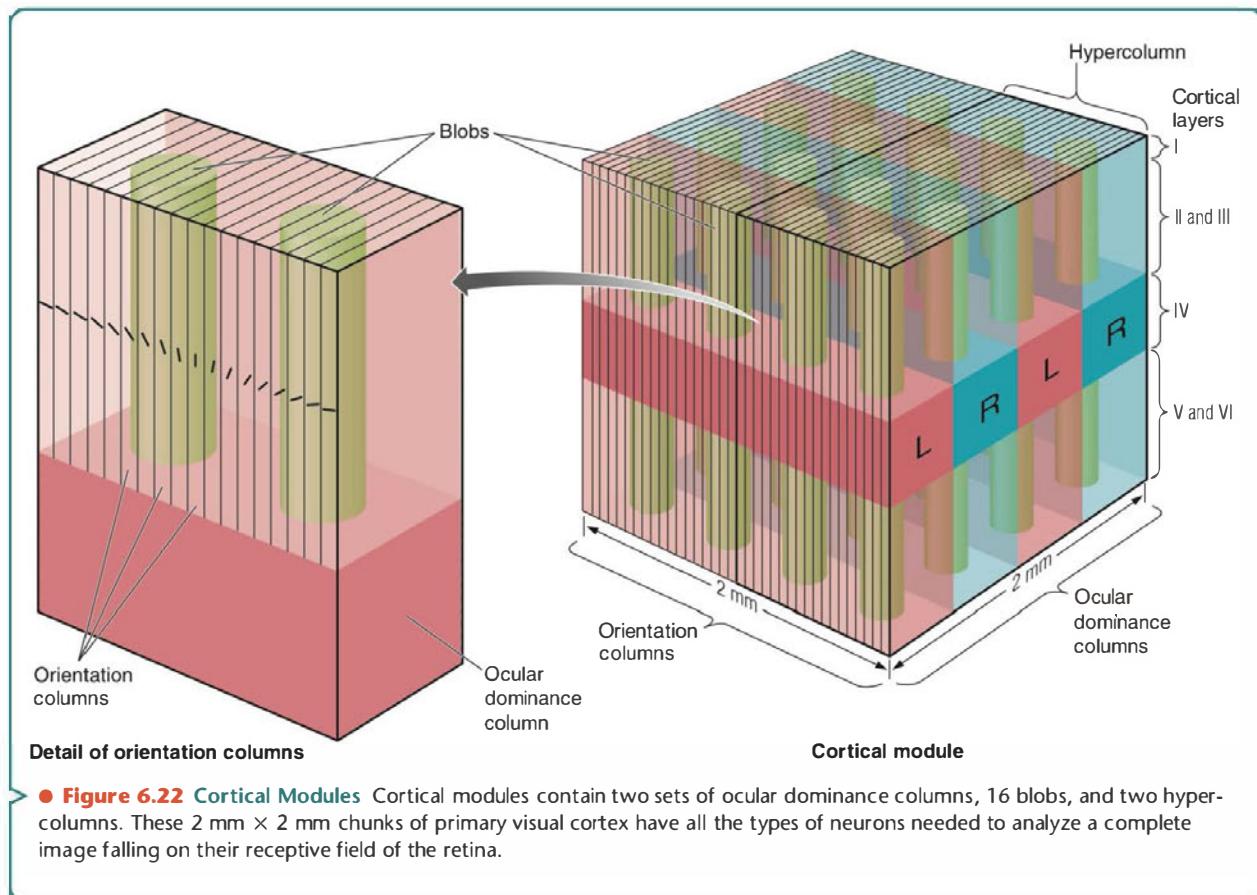
**ocular dominance column** A column of cortex perpendicular to the cortical surface that responds to input from either the right or left eye, but not to both.

**orientation columns** A column of primary visual cortex that responds to lines of a single angle.

**hypercolumn** A complete set of orientation columns that span 180 degrees.

**cytochrome oxidase blobs** An area of primary visual cortex rich in the enzyme cytochrome oxidase that responds to color.

**cortical module** A unit of primary visual cortex containing two sets of ocular dominance columns, 16 blobs, and two hypercolumns.

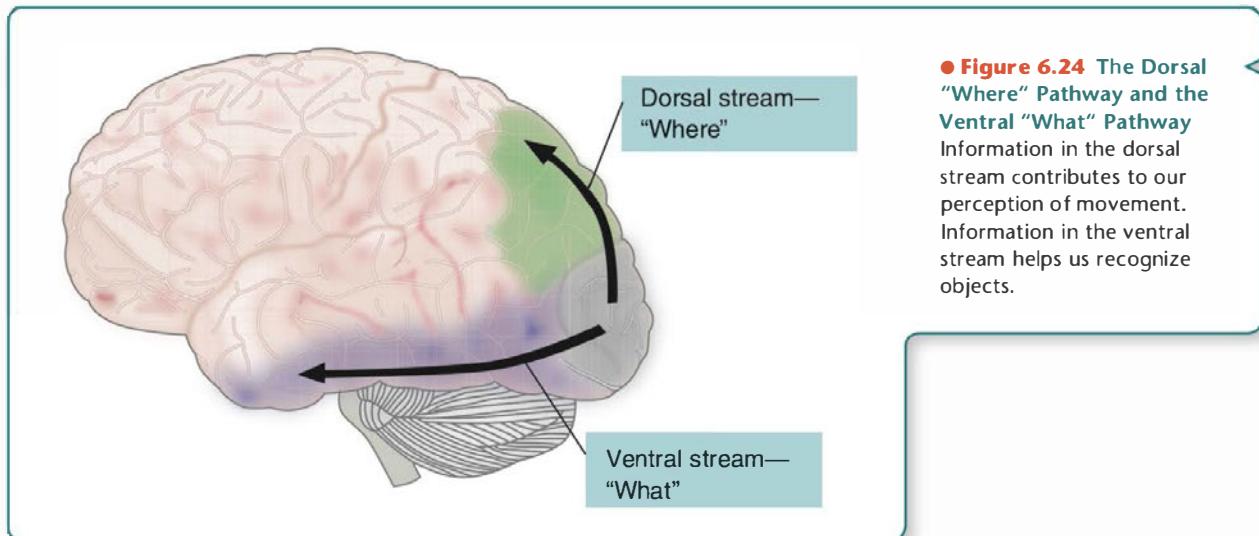


pathway and project to a visual pathway known as the **dorsal stream**. The dorsal stream, shown in **• Figure 6.24**, travels from the primary visual cortex toward the parietal lobe and then proceeds to the medial temporal lobe. This pathway is commonly referred to as the “where” pathway and specializes in the analysis of movement, object locations, and the coordination of eyes and arms in grasping or reaching (Ungerleider & Mishkin, 1982).

The thin stripes and interstripe regions of V2 project to another visual region known as V4, continuing the parvocellular pathway. Area V4 participates in a second major pathway, the **ventral stream**, which proceeds from the primary visual cortex to the inferior temporal lobe. This second pathway, commonly referred to as the “what” pathway, specializes in object recognition (Ungerleider & Mishkin, 1982).

**THE DORSAL STREAM** Area MT, which stands for the medial temporal lobe, receives input from neurons along the dorsal stream and appears to play an important role in the processing of motion. Input to Area MT is primarily from the magnocellular pathways. Recall that the cells in this pathway have large

play an important role in the processing of motion. Input to Area MT is primarily from the magnocellular pathways. Recall that the cells in this pathway have large



**Figure 6.24** The Dorsal “Where” Pathway and the Ventral “What” Pathway  
Information in the dorsal stream contributes to our perception of movement. Information in the ventral stream helps us recognize objects.

receptive fields and often show responses to rapidly changing light conditions and direction of movement. Most of the cells in Area MT respond to movement in a particular direction. Unlike previous motion detectors, however, Area MT cells respond to movement across large regions of the visual field. Patients with damage to Area MT have a condition called *akinetopsia*, which results in their seeing the visual scene as a series of still photographs instead of an ongoing flow of information. These patients experience great difficulty with important tasks such as recognizing that a car is moving toward you (Rizzo, Nawrot, & Zihl, 1995).

Further processing of motion occurs adjacent to Area MT in **Area MST**, which stands for the medial superior temporal lobe. Tanaka and Saito (1989) found that Area MST neurons respond to stimulus rotation, stimulus expansion, and stimulus contraction. These are large, global types of movement that do not produce consistent responses in other areas. Area MST helps us use vision to guide our movements.

Melvyn Goodale and his colleagues (Goodale & Milner, 1992; Milner & Goodale, 1993) suggested that the dorsal stream would be more accurately characterized as a “how” stream than as a “where” stream. According to this view, not only does the dorsal stream tell us an object’s location, but it also provides information about how to interact with an object. Patients with damage to the dorsal stream can judge the orientation of an object, such as lining up a credit card with a slot at a gasoline station, but are unable to combine orientation and action to push the card into the slot.

**THE VENTRAL STREAM** As the information from the primary cortex and Area V2 travels ventrally toward the temporal lobe, we come to Area V4. The cells in this area respond to both shape and color. Cells in Area V4 project to the inferior temporal lobe, or **Area IT**. Cells in Area IT respond to many shapes and colors. In humans and monkeys, a small section of Area IT known as the **fusiform face area (FFA)** appears to respond most vigorously to faces and to members of learned categories, such as species of birds or models of cars (Gauthier, Skudlarski, Gore, & Anderson, 2000) or patterns of chess pieces on a board (Bilalić, Langner, Ulrich, & Grodd, 2011). Monkeys viewing blurred photographs are more likely to report “seeing” faces when their FFAs are stimulated (Afraz, Kiani, & Esteky, 2006).

**dorsal stream** A pathway leading from the primary visual cortex in a dorsal direction thought to participate in the perception of movement.

**ventral stream** A pathway of information from the primary visual cortex to the inferior temporal lobe that is believed to process object recognition.

**Area MT** An area in the medial temporal lobe believed to participate in motion analysis.

**Area MST** An area in the medial superior temporal lobe believed to participate in large-scale motion analysis.

**Area IT** An area in the inferior temporal lobe believed to participate in object recognition.

**fusiform face area (FFA)** An area in the inferior temporal lobe believed to participate in the recognition of familiar faces, especially in the right hemisphere.

## INTERIM SUMMARY 6.2

### || Summary Table: Anatomical Features of the Visual System

Feature	Significance
Cornea	Bends light toward the retina
Anterior chamber	Contains fluid for nourishing the cornea and lens
Iris	Muscle that controls the amount of light entering the eye
Pupil	Opening in the iris
Lens	Focuses light onto the retina
Vitreous chamber	Fluid-filled chamber behind the lens
Retina	Contains photoreceptors and initial processing neurons
Macula	Responsible for central, as opposed to peripheral, vision
Fovea	Pit in the macula specialized for detailed vision
Optic nerves	Axons from retinal ganglion cells that exit the eye
Lateral geniculate nucleus (LGN)	Area of the thalamus that receives input from the optic nerves
Primary visual cortex	Receives input from the lateral geniculate nucleus; responsible for initial processing of an image
Dorsal stream	Analysis of movement
Ventral stream	Object recognition

### || Summary Points

- Before reaching the retina, light travels through the cornea, the anterior chamber, the opening of the pupil controlled by the iris, the lens, and the vitreous chamber. (LO2)
- The retina is a thin layer of visual interneurons and photoreceptors. (LO2)
- The 100 million rods in the human eye are responsible for scotopic (dim light) vision, whereas the 3 million cones are responsible for photopic (bright light) vision. (LO2)
- The human eye's 1.25 million ganglion cells integrate the input from about 103 million photoreceptors and send the information to the brain via action potentials in the optic nerves. The optic tracts proceed to the lateral geniculate nucleus (LGN) of the thalamus, with smaller branches connecting with the hypothalamus and superior colliculi. (LO3)
- The primary visual cortex (striate cortex) is located in the occipital lobe. It contains simple cortical cells and complex cortical cells that participate in the encoding of shape and movement. Information about movement is processed further by the dorsal stream, whereas information about object recognition is processed further by the ventral stream. (LO3)

### || Review Questions

- How would the size of a ganglion cell receptive field predict its ability to respond to fine detail?
- Why is the input from each eye processed separately in the LGN and visual cortex?

## Visual Perception

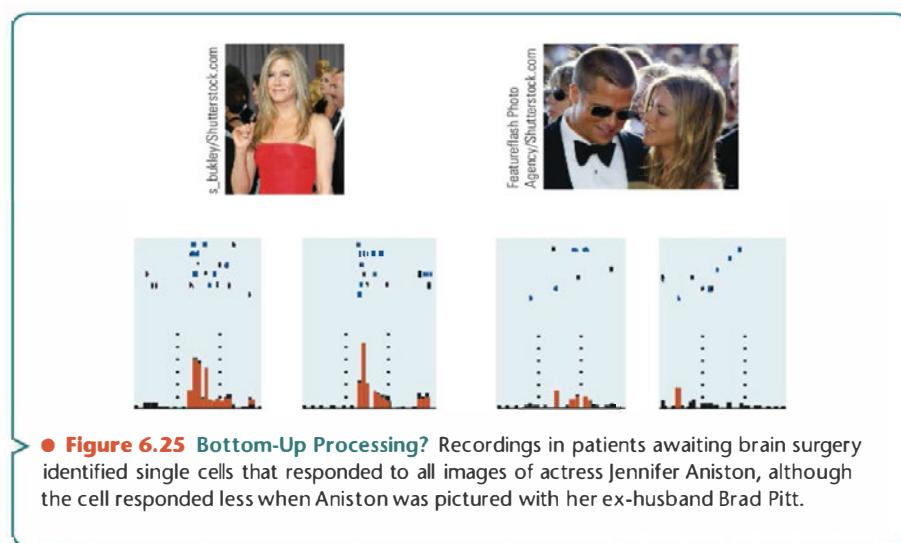
How does the brain construct a model of visual reality out of the input it receives? Before objects can be recognized, information must be processed by the sensory receptors to be analyzed further, a process referred to previously as bottom-up processing. At the same time, the interpretation of this incoming data involves the memories, experiences, and expectations an observer has about an object, leading to top-down processing.

### Hierarchies

The model of cortical visual processing proposed by Hubel and Wiesel implies a bottom-up, hierarchical organization in which simple cells contribute input to increasingly complex cells. At each level of processing, more complex responses are generated from simpler responses. The result of such a system would be a “grandmother cell,” or a single cell that could combine all previous input to tell you that your grandmother was at the door to pay a visit.

The hierarchical model received considerable support from recordings of single cells in the temporal lobes of eight patients undergoing surgery for epilepsy (Quiroga, Reddy, Kreiman, Koch, & Fried, 2005; see ● Figure 6.25). One patient had a cell that fired in response to all images that included actress Jennifer Aniston, but not at all to images of other faces, landmarks, or objects. Another patient had a cell that responded to photos of actress Halle Berry, images of Berry in her Catwoman costume, caricatures of Berry, and even a letter sequence spelling her name.

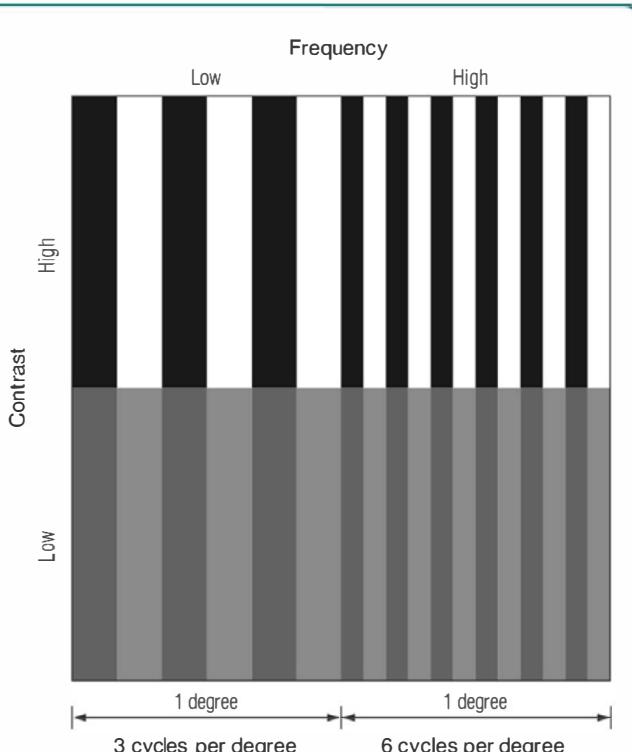
Although the idea of a strict hierarchical structure is attractive in many ways, it does not fit perfectly with what we know about brain organization. First, we would need an immense number of “grandmother cells” to respond to the large numbers of objects and events that we can recognize. This extravagance in the use of cells is out of character for the highly efficient nervous system. Second, the ability of cortical visual neurons to respond equally to changes in more than one stimulus dimension (orientation and movement, for example) is not consistent with the hierarchical model. A true “grandmother cell” should respond only in the presence of its ideal stimulus and never in its absence. Finally, the hierarchical model would struggle to explain our response to the apparently random pattern of dots shown in ● Figure 6.26. Once you know that the image represents a Dalmatian dog, you can instantly pick out the shape of the dog. This requires top-down processing that includes knowledge and memory of the appearance of Dalmatian dogs. It is unlikely that a single cortical cell acting as a Dalmatian dog detector could incorporate such complex inputs from memory.



**Figure 6.26 Problems for the Hierarchical Model of Vision** Can you figure out what is in the picture? If I tell you that this is a picture of a Dalmatian dog, can you see the picture clearly? This visual experience would be difficult to explain in terms of bottom-up processing alone. The stimulus doesn't change when you learn the identity of the object, but the interpretation does.



From Richard L. Gregory, "The Medawar Lecture 2001 Knowledge for vision: vision for knowledge," Phil. Trans. R. Soc. B 2005 360, 1231–1251,  
© The Royal Society



**Figure 6.27 Spatial Frequencies** It has been suggested that the visual system works by performing a mathematical analysis of spatial frequencies in the visual field. Gratings can be described in terms of frequency (number of bars in a given distance) or contrast (the difference in intensity between adjacent bars).

## Spatial Frequencies

Striate cortex may not respond to isolated lines and bars but, rather, to patterns of lines. The simplest patterns of lines are known as **gratings**, as shown in Figure 6.27. A high-frequency grating has many bars in a given distance, whereas a low-frequency grating has relatively few bars. A high-contrast grating has a large amount of difference in intensity between bars, such as very bright white next to black. A low-contrast grating has a more subtle difference in intensity between bars, such as a dark gray next to black. The human visual system could perform a rough mathematical analysis, or **spatial frequency analysis**, of the gratings found in the visual field (De Valois & De Valois, 1980). This is different from the hierarchical approach, which implies a reality built out of bars and edges.

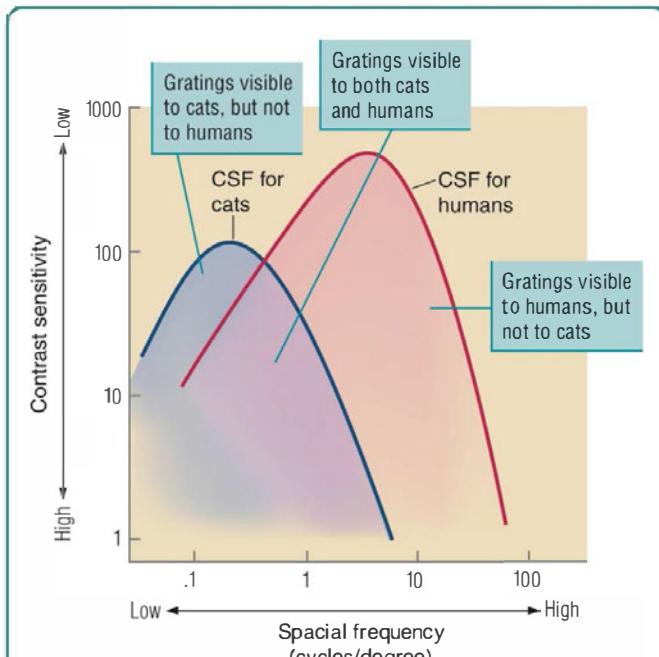
Observing responses to gratings can give us a window into the visual world of other species. Thresholds for contrast can be identified over a range of frequencies. In other words, how much contrast is needed in adjacent bars of a grating before they can be distinguished from a uniform gray stimulus? The resulting graph is known as a **contrast sensitivity function**, or CSF. We can obtain CSFs from nonhuman species by training them to choose a grating rather than a uniformly colored stimulus to obtain food. When the animal's responses become no more accurate than chance, we can assume that the animal can no longer tell the difference between a grating and a uniformly colored stimulus. This determines the CSF for that animal.

● Figure 6.28 compares the CSFs of cats and humans. At higher spatial frequencies (lines in the grating get closer together), your vision is better than your cat's. This means that human beings see more fine detail than cats do. On the other hand, the kitty has an advantage over you at low spatial frequencies. This means that a low-frequency (large), low-contrast (dark gray vs. black) shadow on the wall will get your cat's attention but not yours. You will think the kitty is after ghosts again.

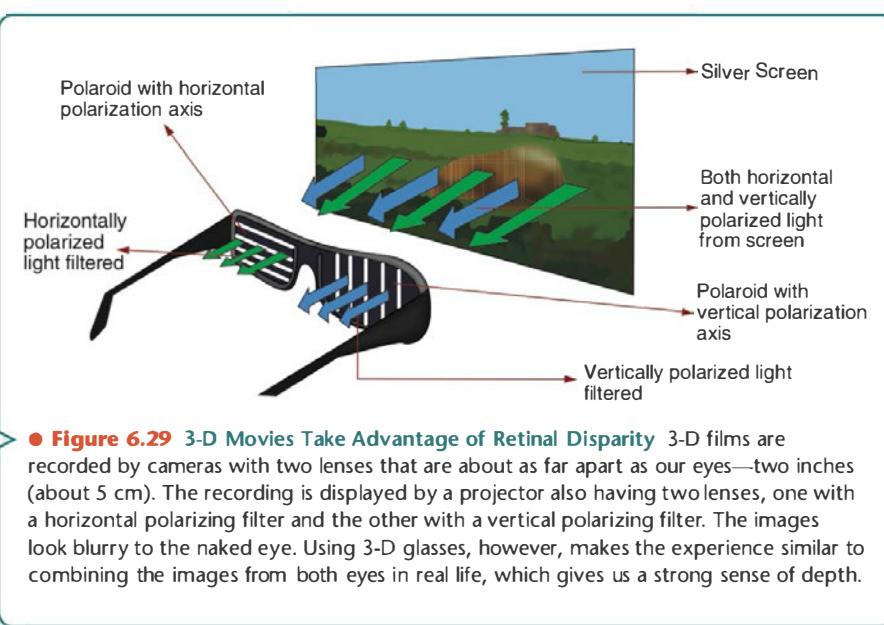
## The Perception of Depth

The image projected on the retina is two-dimensional, so the visual system uses a number of cues to provide a sense of depth. Several of these cues are monocular, requiring the use of only one eye. Perspective, in which lines we expect to be parallel, such as the edges of a road, are made to converge or come together at the horizon, is a centuries-old artistic device to give the illusion of depth on a flat surface such as a painting. Texture, shading, and a comparison of the size of familiar objects can also provide a realistic impression of depth in two dimensions.

The depth cues mentioned so far do not require the use of two eyes. We also have binocular (two-eye) depth cues that are even more effective. When eyes face front, as in humans and other predatory species, the two eyes produce overlapping, but slightly different, images of the visual field (as we saw earlier in Figure 6.17). The differences between the images projected onto the retinas of both eyes result in **retinal disparity**. We can use the degree of disparity as a strong cue for depth. As shown in ● Figure 6.29, movies filmed with 3-D technology take advantage of retinal disparity to give us the illusion of depth.



● **Figure 6.28 A Cat's View of the World** A contrast sensitivity function (CSF) measures how much contrast is needed for a grating to look different from a uniformly colored disk as a function of spatial frequency. The resulting functions for cats and humans show significant overlap. However, you can see more fine detail (higher spatial frequencies) than your cat can. The cat can see better at lower spatial frequencies than you can.



**grating** A striped stimulus used to study responses to spatial frequency.

**spatial frequency analysis** A way of describing visual processing as a basic mathematical analysis of the visual field.

**contrast sensitivity function (CSF)** The mapping of an individual's thresholds for contrast over a range of frequencies.

**retinal disparity** The slightly different views of the visual field provided by the two eyes.

## Behavioral Neuroscience GOES TO WORK

### 3-D ANIMATION

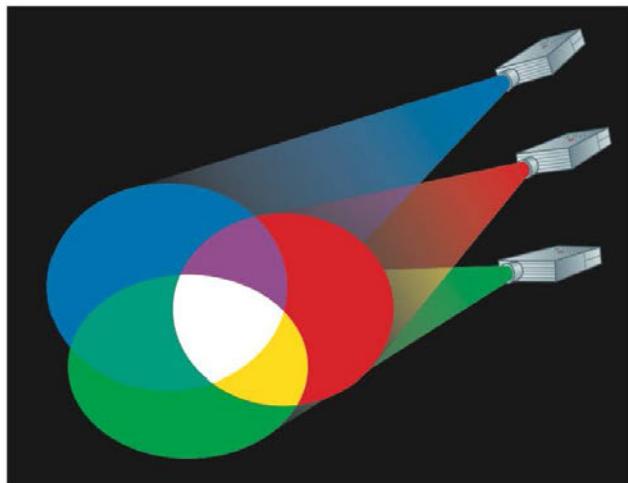


You might be among the millions of moviegoers who are willing to spend extra money to see a film in 3-D. Our understanding of retinal disparity has made the development of this technology possible.

The 3-D movie experience begins with a special camera with two lenses, spaced about two inches (about 5 cm) apart, which is about the same distance between human eyes. Scenes are recorded simultaneously by both lenses, mimicking the viewing of the scenes by your two eyes. The projector, like the camera, also has two lenses spaced closely together. The light from one of the lenses is vertically polarized, which means that particles of light oscillate in one plane only. Normal light oscillates in all directions. The other lens emits light that is horizontally polarized. Newer 3-D projectors use a single lens that switches between the images recorded by each camera hundreds of times per second.

Viewing the projection on a screen would produce a blurry image. Special glasses are needed to sort the images out. Each lens of the glasses is coated with carbon filters that rotate light to be horizontally or vertically polarized, like the filters from the projector. More modern glasses have circular filters that also help reconstruct the images. The brain treats these separate images from each lens of the glasses similarly to the combining of images from our two eyes, resulting in a strong perception of depth.

If you are watching a rather boring 3-D film and would like to explore the boundaries of the technology further, try tilting your head to see what happens to the image. If you are wearing linear polarizing glasses rather than circular ones, this will produce a distorted view of the scene. The brain is unable to interpret the resulting images properly. We usually are not aware of our staring straight ahead at a movie screen, but this is what most of us do.



**Figure 6.30 Mixing Lights** We might be accustomed to thinking of primary colors as red, yellow, and blue, but that works only for paint. In the world of light, red, green, and blue can be mixed to form all other colors. A mixture of all three colors of light looks white.

**Binocular cells** in the cortex respond most vigorously when both eyes are looking at the same features of a visual stimulus (Hubel & Wiesel, 1962). Binocular cells also respond to degrees of retinal disparity. Some cells fire more when their preferred features appear to be at an identical distance. Others fire more when the preferred features are seen by different parts of the two eyes. These cells are known as **disparity-selective cells**. We coordinate the activity of these cells to judge retinal disparity. In areas of the anterior parietal lobe, information about retinal disparity is combined with judgments of how the shape of an object changes due to movement to construct the final impression of a three-dimensional object (Durand et al., 2007; Georgieva, Todd, Peeters, & Orban, 2008).

### Coding Color

Red, green, and blue lights can be mixed to generate all colors, and mixing them together will give you a white light (which can be separated with a prism or by water droplets that produce a rainbow). Consequently, red, green, and blue are considered

**binocular cells** A cell in the cerebral cortex that responds to input from both eyes.

**disparity-selective cell** A binocular cortical cell that responds when its preferred features are seen by different parts of the two eyes.

to be the primary colors of light (see **Figure 6.30**). The human visual system can differentiate approximately 10 million colors.

**THE TRICHROMATIC THEORY** The **trichromatic theory** suggests that human color vision is based on our having three (tri) different color photopigments. As we discussed

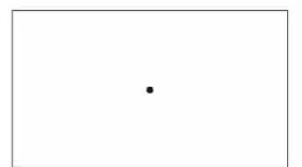
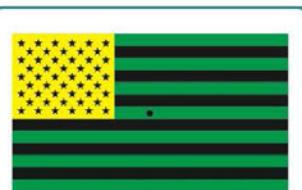
previously, the three photopigments are maximally responsive to lights of different wavelengths.

Based on a series of color-matching tasks with human participants, Thomas Young proposed his trichromatic theory of color vision in 1807, long before any knowledge was available regarding the types of human photoreceptors. Hermann von Helmholtz (1856–1866) expanded Young's theory by proposing three different color receptors in the retina. Both men are credited with the trichromatic theory, which is typically referred to as the Young-Helmholtz theory of color vision.

Although the trichromatic theory accounts for many of the phenomena related to color vision, it leaves other features unexplained. For instance, if you stare at the yellow, green, and white flag in Figure 6.31 and then focus on the dot in the white space below, you will get an afterimage of the flag in its more traditional red, white, and blue. Clearly, there is more to color vision than the trichromatic theory can explain.

**OPPONENT PROCESSES** Ewald Hering observed that mixing blue and yellow lights yielded the sensation of gray and that individuals with color deficiency seemed to lose the ability to discriminate between green and red rather than having difficulties seeing just one of the colors. These observations could not be explained in terms of the trichromatic theory. In his 1878 work, *On the Theory of Sensibility to Light*, Hering suggested an alternate theory of opponent processes based on three types of receptors: a red-green receptor, a blue-yellow receptor, and a black-white receptor.

**Opponent process theory** gains support from the organization of color receptive fields in the visual system (see Figure 6.32). P and K ganglion cells have center-surround receptive fields that respond differentially to color. P cells show red-green center-surround organization. In other words, we can locate receptive fields for P cells that have a center that responds maximally to red and a surround that responds to green. Other P cell receptive fields will have centers that respond maximally to green and surrounds that respond to red. The K cells show antagonistic center-surround organizations responding to blue and yellow. However, unlike the P cells, these blue-yellow ganglion cells come in only one variety. They always have blue centers and yellow surrounds but not the reverse.

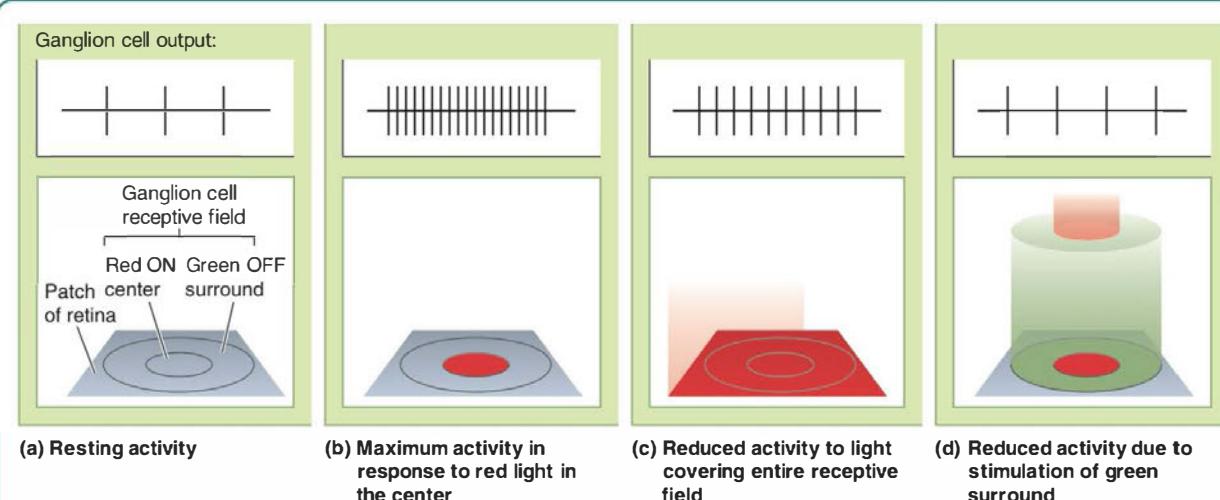


● **Figure 6.31** Color Afterimages Illustrate Opponency

If you stare at the dot in the center of the yellow, green, and white flag for a minute, then shift your gaze to the dot in the white space below, you should see the flag in its traditional red, white, and blue.

**trichromatic theory** The theory that suggests human color vision is based on our possessing three different color photopigments.

**opponent process theory** A theory of human color vision based on three antagonistic color channels: red-green, blue-yellow, and black-white.



● **Figure 6.32** Opponent Process Theory Opponent process theory is consistent with the finding that receptive fields of ganglion cells and LGN cells have antagonistic center-surround organizations for color. Cones maximally sensitive to red and green contribute to red-green receptive fields, which can take red-center green-surround or green-center red-surround forms. Cones sensitive to red and green (the combination in light produces yellow) contribute to the surround of the blue-yellow receptive field, while blue cones contribute to the center. We do not find evidence of yellow-center blue-surround receptive fields.

## THINKING Ethically

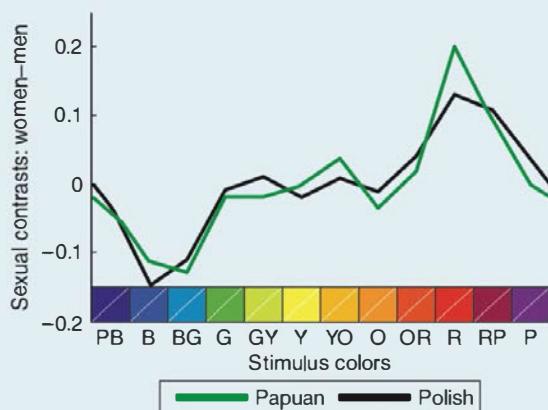
### ARE THERE SEX DIFFERENCES IN COLOR PREFERENCES?

The idea that “pink is for girls and blue is for boys” seems hopelessly sexist to modern thinkers. Why shouldn’t boys and girls enjoy whatever colors please them? Is there any real biological basis for color preferences?

One method for examining any biological contributions to color preferences is to look for cultural differences. If underlying biology plays a substantial role in color preferences, we would not expect to see many differences across cultures. However, this is exactly what we do see. Urban Polish residents showed a preference for shades of blue, while Papuan participants who have had very little contact with outsiders show a preference for red and yellow (Sorokowski, Sorokowska, & Witzel, 2014). Both groups, however, chose yellow-orange as their least favorite color.

In an interesting twist, however, Sorokowski et al. (2014) reported that the degree of difference between men and women in both cultures was very similar in spite of their different overall preferences. As shown in Figure 6.33, the closer a color was to blue, the more men liked it and women disliked it. The closer a color was to red, the more women liked it and men disliked it. This does not necessarily imply

a biological basis for the different preferences, but more research is necessary to identify the sources of these preferences.



**Figure 6.33 Sex Differences in Color Preference** In spite of the overall preference for blue in Poland and red and yellow in Papua, the degree of difference between the sexes in color preference was very similar in both cultures. The closer a color was to blue, the more men preferred it. The closer a color was to red, the more women preferred it.

You may be wondering how the input from three cone types can result in the opposition of four colors: green versus red and blue versus yellow. The green and red case is straightforward. These ganglion cells are receiving input from cones that respond maximally to red or green. The blue and yellow case is slightly different. These ganglion cells receive input from blue cones, of course, which they compare to a mixture of input from red and green cones, which in the world of light add up to yellow.

Which of the two theories of color vision is correct? It appears that both are correct but that they operate at different stages in the analysis of color. The Young-Helmholtz trichromatic theory provides an accurate framework for the functioning of the three types of cones in the retina. At levels of color analysis beyond the retina, Hering’s opponent process theory seems to fit observed phenomena neatly.

**COLOR DEFICIENCY** Occasional errors occur in the genes that encode the cone photopigments. As a result, individuals with these genes show several kinds of atypical responses to color, known as color deficiency. The historical term *colorblindness* is misleading, as most of these individuals do see color, just somewhat differently than the majority of the population.

**Dichromacy** (having two cone photopigments) is the most common type of color deficiency and results from a missing or abnormal cone pigment. Because genes for the red and green photopigments appear on the X chromosome, this type of dichromacy is sex-linked (see Chapter 5). Men are about 10 times more likely to experience this type of color deficiency than women (Hurwicz, 1982). • Figure 6.34

**dichromacy** Having eyes that contain two different cone photopigments.



**• Figure 6.34 Looking Through the Eyes of a Dichromat** This image shows your author, her husband, and Ronnie the Superdog as they appear to typical trichromats (upper left) and to dichromats who are missing the medium (green) photopigment (upper right), the long (red) photopigment (lower left), and the short (blue) photopigment (lower right).

shows us what your author, her husband, and their Australian shepherd (Ronnie the Superdog) might look like to individuals having one of three different types of dichromacy.

There are rare cases in which the blue photopigment is missing. The gene for the blue photopigment is located on chromosome 7 (Nathans, Thomas, & Hogness, 1986), so these cases are not sex-linked and appear equally in males and females. Rarer still are cases of **monochromacy**. This condition occurs when a person has only one type of cone or a complete absence of cones. In either case, the person can't see color at all. Monty Roberts, who served as the inspiration for the film *The Horse Whisperer*, credits his unique ability to study animal motion and expression to his monochromacy (Roberts & Scanlan, 1999). Some individuals have three cone pigments, but their peak response occurs at slightly different wavelengths than is typical. This leads to a condition known as **anomalous trichromacy**.

**monochromacy** The ability to see in black and white only.

**anomalous trichromacy** A condition characterized by having three cone photopigments that respond to slightly different wavelengths than normal.



**• Figure 6.35 Color Contrast** Color contrast, or the differences in appearance when colors are viewed against different backgrounds, is a likely result of color opponency in the central nervous system (CNS). All letters in this image are really the same color.



**• Figure 6.36 “The Dress” and Color Constancy** “The Dress” became an Internet phenomenon as people debated its true colors. Do you see it as black and blue? White and gold? Something else? Although vision scientists are still debating this phenomenon, one explanation is that different assumptions regarding the illumination of the scene would influence judgments of the color of the dress due to color constancy.

These individuals match colors in a slightly different way than most people, but they might not even know that they are unusual. Because of the process of x-inactivation (see Chapter 5), some mothers of anomalous trichromats might actually be tetrachromats (Jordan, Deeb, Bosten, & Mollon, 2010). These individuals match colors in a manner that would be predicted by their having four color photo pigments rather than three.

**COLOR CONTRAST AND COLOR CONSTANCY** Perception does not occur in a vacuum. In the case of **color contrast**, illustrated in •Figure 6.35, colors can look different depending on their context. Because of color contrast, Edwin Land (1959), the inventor of the Polaroid camera, could use red and green filters to give his photographs the appearance of a wide set of colors. Color contrast is primarily an effect of the opponency of color processing in the visual system.

**Color constancy** describes the fact that an object’s colors do not appear to change much even as the light falling on that object changes. Your red sweater looks red outdoors at high noon, under indoor lights in the classroom, and on the way to the parking lot in twilight. Here again, Edwin Land proposed a reasonable explanation for this phenomenon. Land suggested that constancy would occur if the responses of all cones exposed to a scene were “normalized” prior to their final local comparison, which would lead to a perception of color. In other words, the brain compensates for the overall illumination of a scene and adjusts its perception of color accordingly. Your red sweater looks red under different types of light because it is viewed as being “redder” compared to the rest of the scene. These comparisons between local areas and neighboring areas appear to be managed by double opponent cells (Michael, 1985). Double opponent cells compare the amount of color, say red-green, in a local part of the visual scene with the amount of red-green in a neighboring part of the scene (see •Figure 6.36).

## The Life-Span Development of the Visual System

Although we can’t ask young infants about what they can see, we can still establish the same types of contrast sensitivity functions (CSF) that we found earlier for adults and cats. Infants prefer to look at patterns rather than at uniform screens. We assume that if an infant looks at a grating longer than at a uniform circle, the infant must see a difference between the two. If we compare CSF curves for adults and for infants between the ages of one and three months, it becomes apparent that the infant cannot see fine detail at a distance (see •Figure 6.37).



In addition, the CSF curves show that the infant needs more contrast than an adult does to see. This probably relates to the preference most babies show for large, high-contrast, colorful objects.

As we age, predictable changes occur in our vision. In middle age, the lens accommodates more slowly to changes in focal distance. This condition is known as **presbyopia**, or “old sight.” Older adults also have trouble responding quickly to changes in lighting, as when exiting a dark theater into the sunlight. The lens, which provides most of the focus of light onto the retina, continues to grow throughout the life span. As fibers are continually added to the structure of the lens, it takes on a yellow hue. Although this change in color provides more protection from ultraviolet rays that might otherwise harm the aging retina, the yellow lens will also distort the person’s perception of blue and green. Aging is also associated with smaller pupils, probably due to the loss of elasticity in the muscles of the iris. Smaller pupils allow less light into the eye, negatively affecting the quality of vision.

Aging might have a negative effect on the cortical processing of visual information. Compared to younger adults, the visual cortex of older adults does not show an increase in activity as the rate of stimulus presentation increases (Cliff et al., 2013). This change at an early stage of processing might affect the quality of information available to higher cognitive processes. Schmolesky, Wang, Pu, and Leventhal (2000) investigated the effects of aging on neurons responding to line orientation or motion in the primary visual cortex. In young monkeys, these researchers found that 90 percent of the measured neurons showed an orientation preference, whereas 70 percent showed a preference for a direction of movement. In aged monkeys, only 42 percent of the cells showed an orientation preference, and 25 percent showed a direction of movement preference. The aged neurons seemed to become less selective, firing at just about anything going on in the visual field.

## Disorders of the Visual System

A variety of conditions can interfere with vision, ranging from the very mild and correctable to a complete loss of vision. Visual deficits occur due to problems in the eye and retina as well as to central problems in the brain.

### Amblyopia

**Amblyopia**, sometimes referred to as “lazy eye,” occurs when one eye cannot focus on objects. If left untreated, the brain will learn to ignore the input from the less functional eye. Binocular depth perception will be permanently lost.

**color contrast** The fact that colors can look different depending on the surrounding colors.

**color constancy** The concept that an object’s color looks the same regardless of the type of light falling on the object.

**presbyopia** The reduced rate and extent of accommodation by the lens that results from aging.

**amblyopia** A condition also known as lazy eye, in which one eye does not track visual stimuli.

## Cataracts

**Cataracts** result from clouding of the lens of the eye. Cataracts become more frequent with age. However, some ethnic populations, including Arabs and Sephardic Jews, have a high rate of cataracts at birth. Severe cataracts are usually treated by surgically removing the clouded lens. Following surgery, the person requires extremely strong glasses or the implant of an artificial lens. Removal of a lens can also negatively influence color vision. Following removal of a lens for cataracts, French impressionist painter Claude Monet saw nearly everything as blue.

## Visual Acuity Problems

Some common visual acuity problems are illustrated in Figure 6.38. If the eyeball is slightly elongated, the image focused by the lens will fall short of the retina. This condition is known as **myopia**, or nearsightedness. The person can see well when looking at close objects, but the ability to see objects in the distance is impaired. If the eyeball is too short, the best image would be focused somewhere behind the retina. This condition is **hyperopia**, or farsightedness. Distance vision will be quite good, but close objects, including letters on the pages of books or newspapers, will be blurry. Unlike myopia and hyperopia, **astigmatism** does not result from eyeball length. Instead, this condition results from unevenness in the shape of the cornea. These vision problems are typically addressed by the use of corrective lenses (eyeglasses or contact lenses) or laser surgery, which reshapes the cornea.

**cataract** Clouding of the lens.

**myopia** An acuity problem resulting from an elongated eyeball; also known as nearsightedness.

**hyperopia** An acuity problem resulting from a short eyeball; also known as farsightedness.

**astigmatism** A distortion of vision caused by the shape of the cornea.

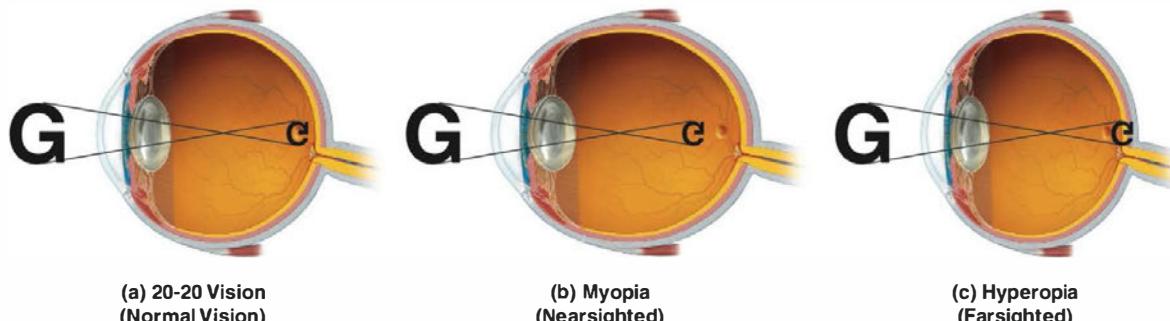
**scotoma** An area in the visual field that can't be seen, usually due to central damage by stroke or other brain injury.

**blindsight** An abnormal condition in which parts of the visual field are not consciously perceived but can be subconsciously perceived by extrastriate cortex.

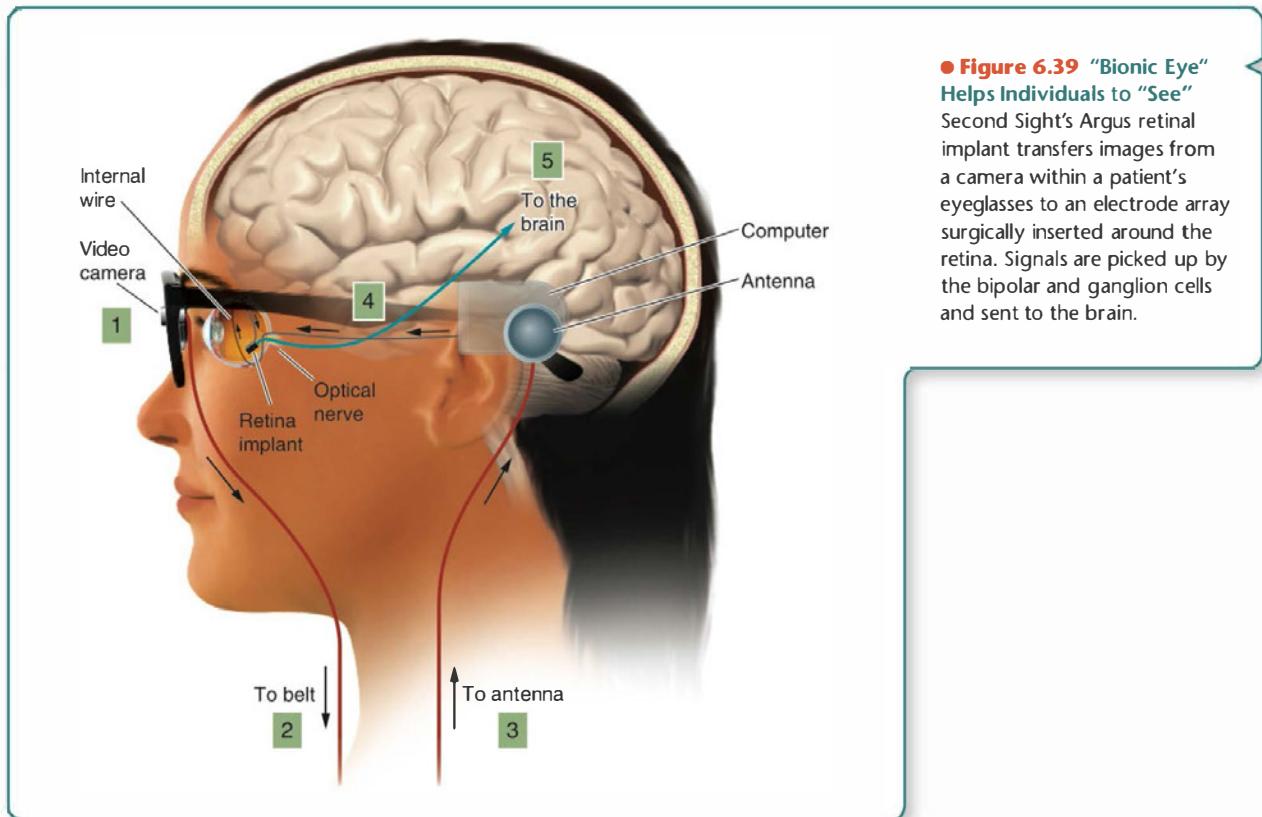
## Blindness

Blindness, or the total loss of vision, can occur as a result of damage at many levels. Damage to the eye or optic nerves could prevent input to normally functioning visual cortical areas. Macular degeneration, in which the cells of the macula begin to die, is one of the leading causes of blindness among older adults. Macular degeneration results in the loss of central vision and a reduction in contrast sensitivity. Although the exact causes of macular degeneration are not known, smoking, high blood pressure, obesity, and exposure to short-wave light (occurring in many artificial lights) are believed to interact with genetic vulnerabilities.

Other individuals are blind due to cortical damage. When the striate cortex is damaged, the patient has a **scotoma**, or region of blindness, that will depend on the exact location and amount of cortical damage. These patients often demonstrate the odd phenomenon of **blindsight** (Cowey & Stoerig, 1991). Although they claim they



● **Figure 6.38** **Eyeball Shape Influences the Quality of Vision** Eyeballs that are either too long or too short cause vision problems, because the focused image falls either short of the retina or beyond the retina.



cannot see lights that are flashed in the area of their scotoma, these patients can point on command to the source of the light. It is likely that visual input to the extrastriate cortex, which does not result in conscious awareness of light, is responsible for these unusual effects (Silvanto, 2008). These findings suggest that processing by striate cortex is essential for conscious awareness of visual stimuli.

The field of vision restoration seeks methods that would lead to recovered vision for those who are blind. Retinitis pigmentosa, a visual degenerative condition resulting from genetic mutations affecting photoreceptors and the underlying epithelium, has shown some benefits from dietary supplements and gene replacement therapies (Geruschat & Dagnelie, 2016). Retinal prosthetic devices can be implanted on, in, or behind the retina (see Figure 6.39). Electrical stimulation from the device interacts with bipolar and ganglion cells to restore some visual perception (Geruschat et al., 2016). Although this restoration is far from perfect, patients reported improved abilities in orientation, mobility, daily life, and interactions with others. As this prosthetic technology improves, it is likely that more normal restored vision will become possible.

## Visual Agnosias

**Visual agnosias** are disorders in which a person can see a stimulus but has difficulty recognizing what is seen. The word *agnosia* comes from the Greek for “without knowledge.” Responding to the image of a carrot, one patient with a visual agnosia said, “I have not the glimmerings of an idea. The bottom point seems solid and the other bits are feathery. It does not seem logical unless it is some sort of brush” (Humphreys & Riddoch, 1987, p. 59). The patient is attending to the major features of the stimulus (pointy end, leafy green part) but can't recognize the object.

**visual agnosia** A disorder in which a person can see a stimulus but cannot identify what is seen.

## Building Better HEALTH

### DOES EATING CARROTS REALLY HELP YOUR VISION?



Your mother may have encouraged you to eat carrots "for your eyes," and as usual, your mother is correct in suggesting that diet can be important to eye health. The American Optometric Association (American Optometric Association, 2017) has listed a number of nutrients that reduce the risks of certain eye diseases, including the formation of cataracts and macular degeneration.

Lutein and zeaxanthin are found in green leafy vegetables and eggs. Consumption of these substances is negatively correlated with risk for cataracts and macular degeneration. Vitamin C from fruits and vegetables

lowers cataract risk and can slow the progression of other types of vision loss. Vitamin E, found in nuts, fortified cereals, and sweet potatoes, protects the eye from damage caused by free radicals. Omega-3 fatty acids, found in some fish, ensure normal development of the eye and help maintain retinal function. Finally, zinc participates in the movement of vitamin A from the liver to the retina. Zinc is usually found in very high concentrations in the eye, and deficits of zinc can cause visual problems.

Diet is not the only contributing factor to maintaining vision into older adulthood, but it can definitely be a good starting place.

In **prosopagnosia**, vision is retained, but the person cannot recognize the faces of people he or she knows (Barton, 2003). Patients with prosopagnosia can tell one face apart from another and can tell the gender of the people from pictures of their faces. In spite of these skills, these patients can't recognize faces as people they know, not even their own image in the mirror. As we mentioned earlier, the fusiform face area (FFA) is probably responsible for facial recognition. Prosopagnosia is usually associated with damage to this area due to stroke or other accidents, although other cases can run in families, indicating a genetic basis (Kennerknecht et al., 2006). In her autobiography, famous ethologist Jane Goodall discloses that she has a type of prosopagnosia (Goodall & Berman, 1999). Apparently, this difficulty did not interfere with her abilities to make remarkable observations of her chimpanzees.

### INTERIM SUMMARY 6.3

#### Summary Table: Examples of Major Visual Disorders

Disorder	Symptoms	Causal Factors	Treatment
Cataracts	Mild blockage of light to complete blindness; distortion of color vision	Clouding of the lens	Surgical removal of the lens
Myopia (nearsightedness)	Difficulty seeing distant objects	Elongation of the eyeball	Corrective lenses, laser surgery
Hyperopia (farsightedness)	Difficulty seeing close objects, reading	Shortening of the eyeball	Corrective lenses, laser surgery
Astigmatism	Difficulty seeing distant objects	Uneven cornea shape	Corrective lenses, laser surgery
Scotoma	Regions of blindness in visual field	Stroke, physical injury to visual cortex	None
Prosopagnosia	Inability to recognize familiar faces	Damage to the fusiform gyrus	None

**prosopagnosia** The inability to recognize known faces.

### || Summary Points

1. The cortex constructs a visual reality through either hierarchical processing or a basic mathematical analysis of contrast and frequencies. (LO4)
2. Depth perception results from monocular and binocular cues. (LO4)
3. The trichromatic theory of color perception is based on the fact that we have three types of cone photopigments that respond differentially to lights of different wavelengths. Our visual system also shows a pattern of red-green and blue-yellow opponency. (LO4)
4. Infants see less fine detail at a distance than adults do. Older adults experience less visual quality due to presbyopia, slow adaptation to changes in light, yellowing of the lens, smaller pupils, and less selectivity in cortical responses to visual input. (LO5)
5. Many conditions can interfere with vision, either at the level of the eye or the level of the brain. (LO6)

### || Review Questions

1. How can contrast sensitivity functions (CSF) tell us what an organism can see?
  2. What do cases of color deficiency teach us about the nature of color vision?
- 

## Chapter Review

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### THOUGHT QUESTIONS

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1. If you had to lose either your scotopic or photopic vision, which would you choose to give up and why? What would be the consequences of your choice?
2. If increasing numbers of ultraviolet rays reaching the earth favor the evolution of a more yellow lens, what effect might this have on the colors we perceive?
3. Currently, few states regularly test the vision of senior drivers. Based on your knowledge of the changes in vision typical of aging, what types of tests would you recommend?

### KEY TERMS

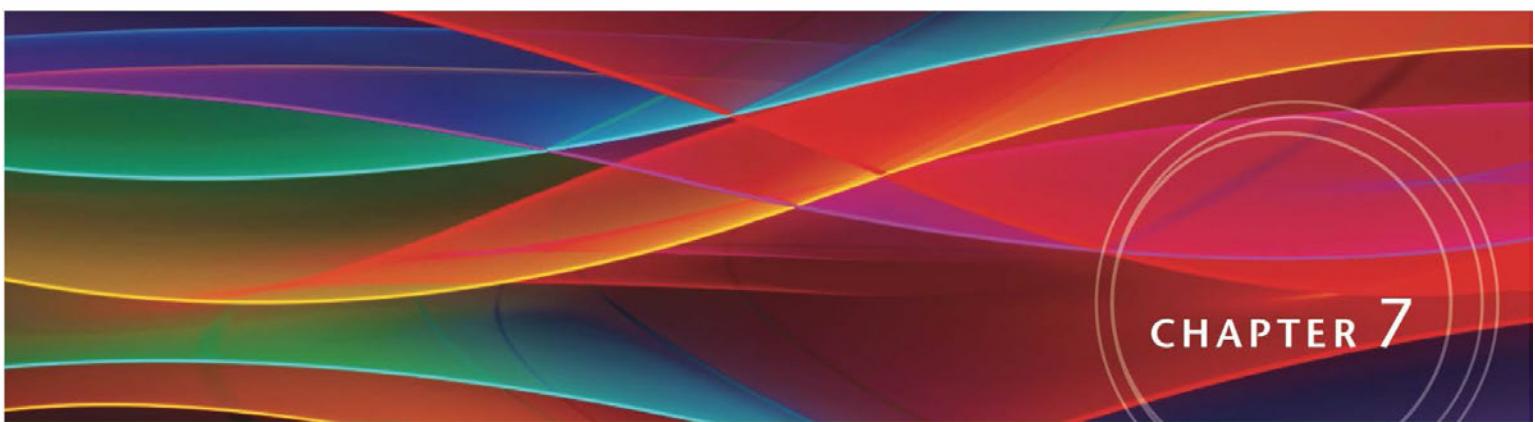
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absorption (p. 186)	cornea (p. 188)	macula (p. 191)
amacrine cell (p. 192)	cortical module (p. 207)	ocular dominance column (p. 207)
antagonistic center-surround organization (p. 198)	cytochrome oxidase blob (p. 207)	opponent process theory (p. 215)
attention (p. 184)	dark current (p. 196)	optic chiasm (p. 203)
bipolar cell (p. 192)	dichromacy (p. 216)	optic disk (p. 190)
bottom-up processing (p. 184)	dorsal stream (p. 208)	optic tracts (p. 203)
central vision (p. 191)	fovea (p. 191)	orientation column (p. 207)
color constancy (p. 218)	fusiform face area (FFA) (p. 209)	perception (p. 184)
color contrast (p. 218)	horizontal cell (p. 192)	peripheral vision (p. 191)
complex cortical cell (p. 205)	hypercolumn (p. 207)	photons (p. 185)
cone (p. 192)	lateral geniculate nucleus (LGN) (p. 203)	photopic vision (p. 193)
contrast sensitivity function (CSF) (p. 212)	lateral inhibition (p. 199)	photopigment (p. 192)
	lens (p. 189)	photoreceptor (p. 190)
		receptive field (p. 197)

retina (p. 190)  
retinal disparity (p. 213)  
rhodopsin (p. 193)  
rod (p. 192)  
scotopic vision (p. 193)

sensation (p. 184)  
simple cortical cell (p. 205)  
striate cortex (p. 204)  
top-down processing (p. 185)  
transduction (p. 184)

trichromatic theory (p. 214)  
ventral stream (p. 208)  
visual agnosias (p. 221)  
vitreous chamber (p. 190)



## CHAPTER 7

# Nonvisual Sensation and Perception

### LEARNING OBJECTIVES

- L01** Identify the major features of sound as a stimulus.
- L02** Trace the process of hearing from the outer ear to the cerebral cortex.
- L03** Explain the perception of pitch, loudness, and the location of sounds.
- L04** Describe the structures and functions of systems responsible for the perception of body position and movement, touch, and pain.
- L05** Describe the structures and pathways responsible for olfaction and gustation.
- L06** Identify the key features of synesthesia.

### CHAPTER OUTLINE

#### Audition

- Sound as a Stimulus
- The Structure and Function of the Auditory System
- Auditory Perception
- Hearing Disorders

#### Interim Summary 7.1

#### The Body Senses

- The Vestibular System
- Touch
- Pain

#### Interim Summary 7.2

#### The Chemical Senses

- Olfaction
- Gustation

#### Synesthesia

#### Interim Summary 7.3

#### Chapter Review

**BUILDING BETTER HEALTH:** Earbuds and Hearing Loss

**THINKING ETHICALLY:** Cochlear Prosthetics and Deaf Culture

**CONNECTING TO RESEARCH:** Phantom Limbs, Mirrors, and Longer Noses

**BEHAVIORAL NEUROSCIENCE GOES TO WORK:** What Is a Perfumer?

## Audition

When Helen Keller, who was both blind and deaf, was asked which disability affected her the most, she replied that blindness separates a person from things, whereas deafness separates a person from people. In addition to processing the speech of others, we use the sense of **audition**, or hearing, to identify objects in the environment and to determine where objects are in relation to our bodies.

Ours is a uniquely human auditory world. Just as we can see a broad but limited range of the electromagnetic radiation spectrum, we can also hear a wide but limited range of sound. Your dog begins howling seconds before you hear the ambulance siren, because the dog's hearing is better than yours for these high-pitched sounds. Neither you nor your dog is likely to hear the even higher pitched vocalizations that bats use to locate food and navigate (see ● Figure 7.1) (Griffin, 1959).

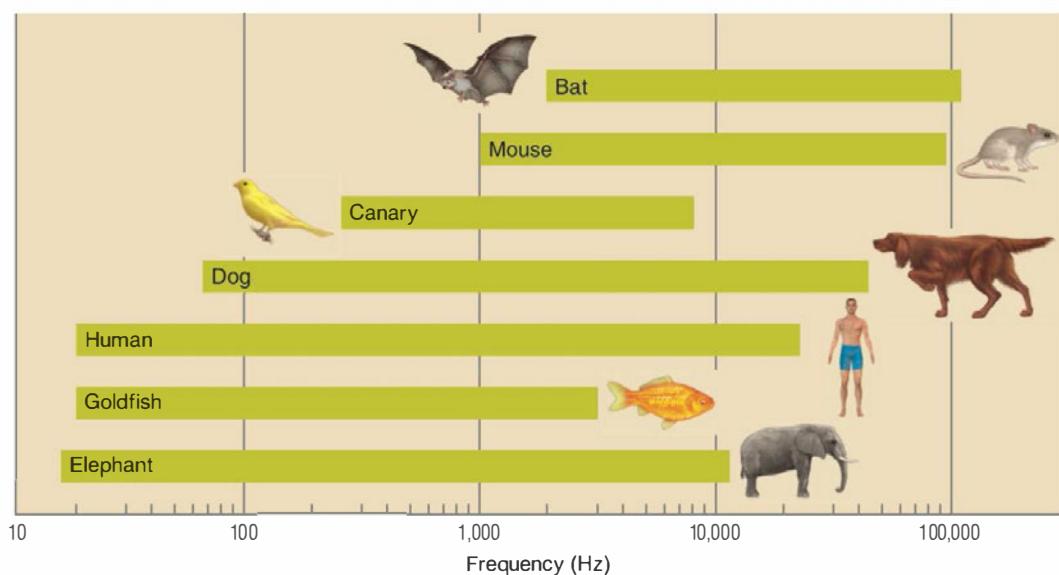
### Sound as a Stimulus

You may have heard the famous riddle by the philosopher George Berkeley, "If a tree falls in the forest and nobody is around to hear it, does it make a sound?" The neuroscientist's answer to this question is both yes and no. The answer is yes if we are asking whether the falling tree produces a physical sound stimulus, but the answer is no if we're talking about sound as the result of the perceptual experience of hearing.

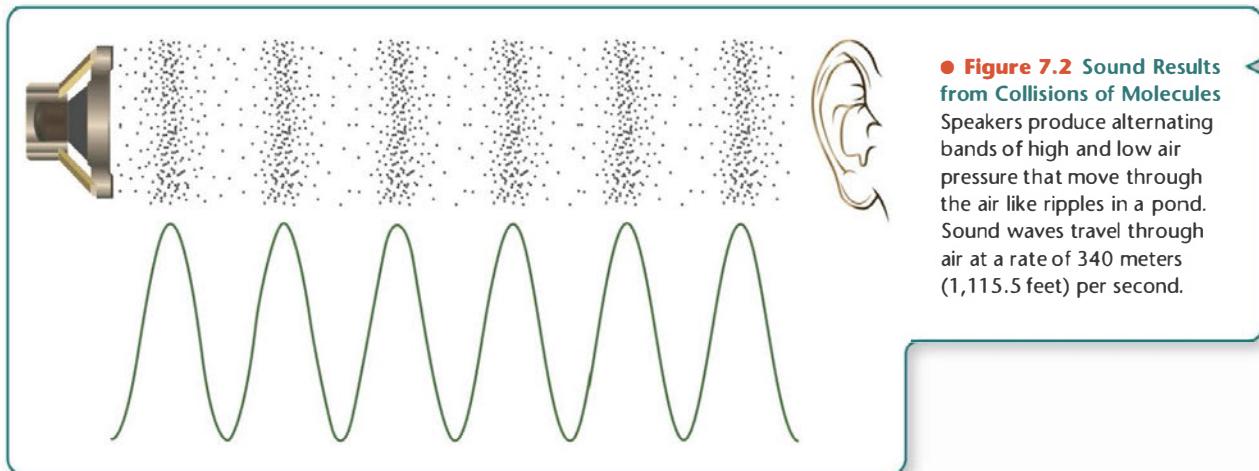
Sound as a physical stimulus begins with the movement of an object. Movement sets off waves of vibration in the form of miniature collisions between adjacent molecules that produce outwardly moving bands of high and low pressure, much like ripples in a pond (see ● Figure 7.2). Because sound waves require this jostling between molecules, sound cannot occur in a vacuum such as outer space. Those explosions we enjoy in Star Wars films may be good entertainment, but they are not great science.

For humans, the medium that carries sound is usually air, but we can also sense sounds that travel through liquids and solids. Like light, sound interacts with the environment as it travels from its source to the perceiver. Fabrics absorb sound waves and

**audition** The sense of hearing.



● **Figure 7.1 The Auditory World Differs across Species** This figure illustrates the range of frequencies that fall within the sensory capacities of several species, including humans.



can be helpful in reducing noise. Sounds reflected from surfaces are used by a number of species, such as bats, for echolocation. Although humans are not the best at using echolocation, we can improve with practice.

As shown in Table 7.1, sound energy, like electromagnetic energy, can be described in the form of waves. The height, or **amplitude**, of a wave indicates the amount of vibration produced by the sound, which in turn is perceived as loudness by the listener. Low-amplitude waves are characteristic of soft sounds, and high-amplitude waves are perceived as loud. The **frequency** of the wave, or the number of cycles per unit of time, indicates wavelength. Wavelength corresponds to the perceived pitch of a sound.

**amplitude** The height of a wave; in audition, amplitude is perceived as loudness.

**frequency** The number of cycles of a periodic wave per unit of time; in audition, frequency is perceived as pitch.

**TABLE 7.1** Sounds Vary Along the Dimensions of Amplitude, Frequency, and Complexity

Wave Characteristic	Perception of Characteristic	Examples
Amplitude (intensity)—Measures the height of the wave. 	Loudness	High-amplitude waves are perceived as loud sounds. 
Frequency (wavelength)—Measures the number of wave cycles per unit of time. 	Pitch	Low-frequency waves are perceived as low-pitched sounds. 
Timbre (complexity)—The distinct quality or uniqueness of a sound.	The "color" or "quality" of tones having equal pitch and loudness.	Pure tones have a single frequency. 
		Complex tones are made up of several frequencies. 

Long wavelengths are perceived as having low pitch, whereas short wavelengths produce sounds with higher pitch. The simplest type of sound wave is a **pure tone**, which has a single frequency like a tone produced by a tuning fork. Pure tones rarely exist in nature, and most sounds consist of combinations of waves. Complex tones combining multiple waves have a characteristic quality known as **timbre**. The same note played by a piano or violin will sound different due to the instruments' impact on timbre. Waves that do not regularly repeat themselves are perceived as **noise**, as opposed to identifiable tones.

**INTENSITY** Human beings can perceive sounds that vary in intensity by a factor of over 10 billion, from the quietest sounds detectable to a jet engine at takeoff. To manage such a wide range of intensities, a logarithmic scale of sound intensity based on the **decibel (dB)** is used. The threshold for hearing, or the least intense sound that a human can hear more than 50 percent of the time, is set at 0 dB. This is roughly equivalent to the sound made by a mosquito flying three meters (about 10 feet) away from you. As shown in Table 7.2, a whisper produces a sound intensity of 20 dB, whereas an iPhone turned up to maximum loudness can reach 120 dB. At 130 dB, we experience pain. This is a useful warning because exposure to sounds at this level of intensity might permanently damage our hearing.

**FREQUENCY** Frequency refers to the number of cycles per unit of time, or the wavelength, of a sound stimulus. The unit used to measure the frequency of sound is the **hertz (Hz)**. A 500 Hz sound completes 500 cycles in one second.

Human hearing ranges from approximately 20 Hz to 20,000 Hz (see Figure 7.1). **Infrasound** refers to frequencies below the range of human hearing. Many animals, including elephants and marine mammals, use infrasound for communication. **Ultrasound** refers to stimuli with frequencies beyond the upper range of human hearing. Ultrasound waves are used to clean objects or to produce noninvasive images for medical purposes.

**TABLE 7.2** | Intensity Levels of Common Sounds

Source of Sound	Intensity Level
Threshold of hearing (TOH)	0 dB
Rustling leaves	10 dB
Whisper	20 dB
Normal conversation	60 dB
Busy street traffic	70 dB
Vacuum cleaner	80 dB
Water at the foot of Niagara Falls	90 dB
Power lawn mower	100 dB
Front rows of rock concert	110 dB
Propeller plane at takeoff	120 dB
Threshold of pain (e.g., machine-gun fire)	130 dB
Military jet at takeoff	140 dB
Instant perforation of eardrum	160 dB

**pure tone** Sound characterized by a single frequency.

**timbre** Distinct quality of a sound due to combinations of frequencies.

**noise** Unsystematic combinations of sound waves.

**decibel (db)** A unit used to express a difference in intensity between two sounds, equal to 20 times the common logarithm of the ratio of the two levels.

**hertz (Hz)** A unit of sound frequency equal to one cycle per second.

**infrasound** Sound at frequencies below the range of human hearing, or lower than about 20 Hz.

**ultrasound** Sound at frequencies above the range of human hearing, or higher than about 20,000 Hz.

## The Structure and Function of the Auditory System

The components that make up the ear are generally divided into three parts: the outer, middle, and inner ear.

**THE OUTER EAR** The major structures of the ear are illustrated in Figure 7.3. The outer ear consists of the structures visible outside the body: the pinna and the auditory canal. The **pinna** serves to collect and focus sounds, just like a funnel. The pinna also plays an important role in locating the source of sound. Movement of the pinna allows some species to further localize sound or to indicate emotional states, as when a dog puts its ears back while snarling. Sound collected by the pinna is channeled through the **auditory canal**, a tube-shaped structure about 3 cm (~1.18 inches) long and about 7 mm (~0.28 inches) wide.

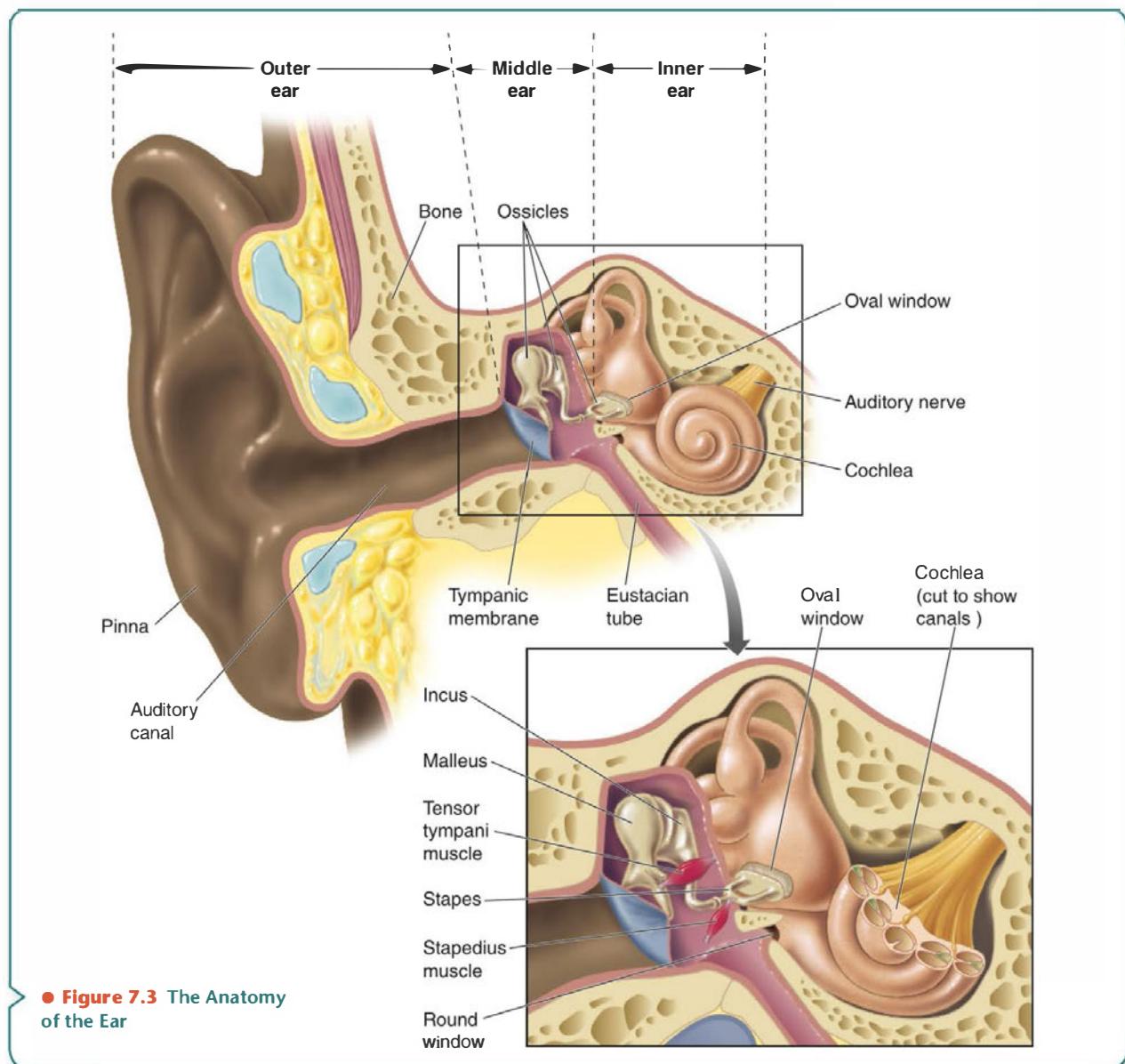
**THE MIDDLE EAR** The **tympanic membrane**, or eardrum, forms the boundary between the outer ear and middle ear. The boundary between the middle ear and inner ear is formed by another membrane, the **oval window**.

**pinna** The visible part of the outer ear.

**auditory canal** A tube-shaped structure in the outer ear that leads to the tympanic membrane.

**tympanic membrane** The membrane separating the outer and middle ears; also known as the eardrum.

**oval window** The membrane separating the middle and inner ears.



The three **ossicles** bridging the middle ear are the malleus (hammer), incus (anvil), and stapes (stirrup). Each of these tiny bones is about the size of a single letter of print in this text. The purpose of these bones is to transfer sound energy from the outside air to the fluid in the inner ear without losing too much of it. As you may have noticed when swimming, sounds waves originating in air lose much of their energy when they enter the water. If a friend calls out to you while you're under water, the sound is not very clear. The ear faces a similar problem, as sound energy must travel through the air of the outer and middle ears to the fluid of the inner ear.

The middle ear solves this transfer problem in two ways. First, the connections between the ossicles are hinged, which creates a lever action that increases the force of the vibration that the stapes bone delivers to the oval window. Second, force applied to the much smaller oval window produces much more pressure than the same force applied to the larger tympanic membrane. With both force and pressure increased at the oval window, the ear can recover about 23 dB of the 30 dB that would otherwise be lost when sound is transferred from the air in the middle ear to the fluid in the inner ear (Evans, 1982).

**ossicles** The bones that span the middle ear, including the malleus, incus, and stapes.

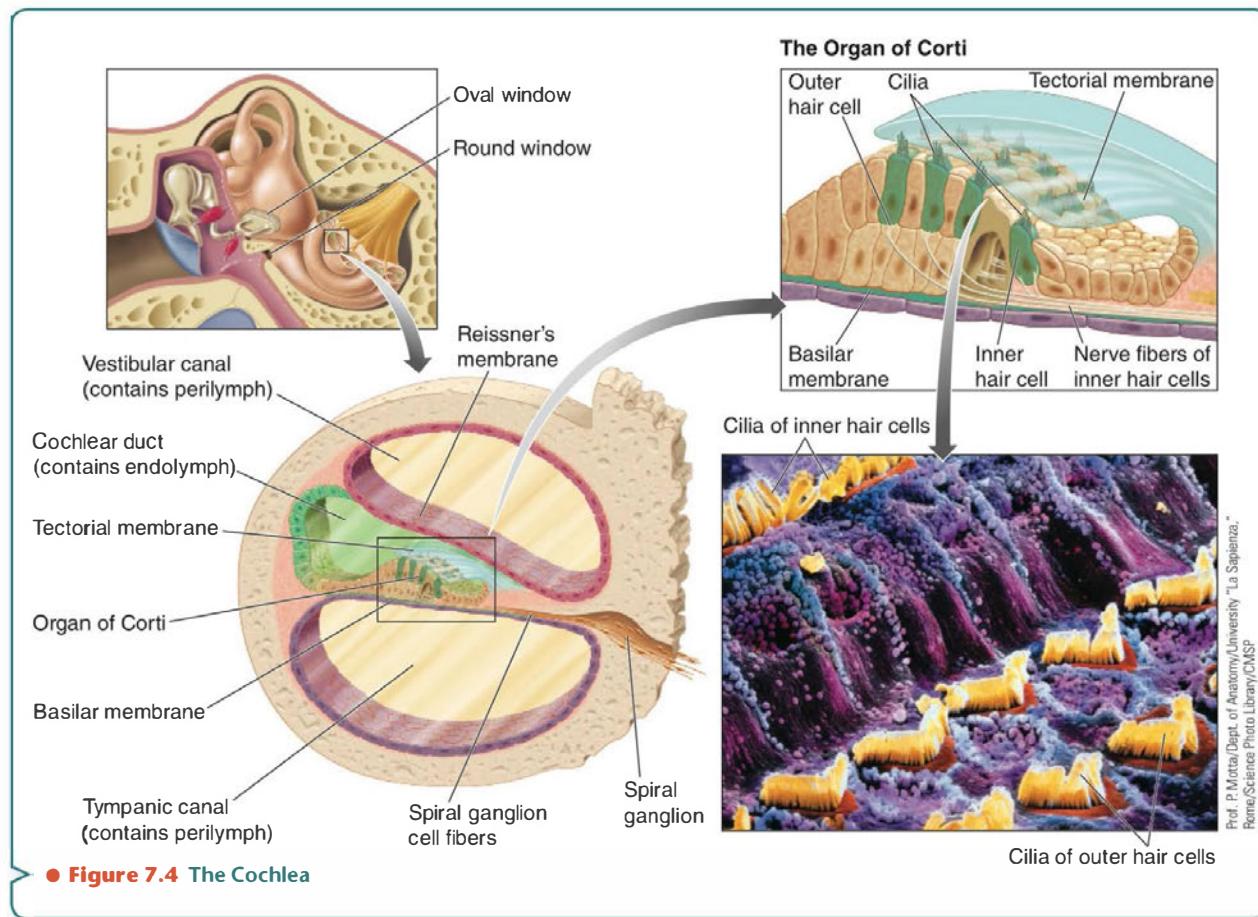
**cochlea** The fluid-filled structure of the inner ear containing auditory receptors.

**vestibular canal** The upper chamber of the cochlea.

**tympanic canal** The lower chamber of the cochlea.

**THE INNER EAR** The inner ear contains two sets of fluid-filled cavities embedded in the temporal bone of the skull. One set, known as the semicircular canals, is part of the vestibular system, which will be discussed later in this chapter. The other set is known as the **cochlea** ("snail" in Greek). The fluid-filled cochlea contains specialized receptor cells that respond to the vibrations transmitted to the inner ear. The cochlea is about 32 mm (0.25 inches) long and 2 mm (.08 inches) in diameter. When rolled up like a snail shell, the human cochlea is about the size of a pea.

The cochlea is divided into three parallel chambers, illustrated in Figure 7.4. Two of the chambers, the **vestibular canal** and the **tympanic canal**, are connected to



• Figure 7.4 The Cochlea

each other near the **apex**, which is the part of the cochlea most distant from the oval window. These two chambers contain a fluid known as **perilymph**, which is similar to cerebrospinal fluid (see Chapter 2). The third chamber, the **cochlear duct**, contains a very different type of fluid, known as **endolymph**. The endolymph is rich in potassium and low in sodium. The fluids (and chambers) are separated by two membranes. **Reissner's membrane** separates the vestibular canal and the cochlear duct. The **basilar membrane** separates the tympanic canal and the cochlear duct.

At the base of the cochlea, at the boundary between the middle and inner ears, the oval window covers the vestibular canal. The tympanic canal is covered by another membrane, known as the **round window**. Because the vestibular and tympanic canals are connected, pressure applied to the oval window by the stapes travels around the apex through the perilymph and pushes the round window out into the middle ear.

Within the cochlear duct is a specialized structure known as the **organ of Corti**, which is responsible for translating vibrations in the inner ear into neural messages. The organ of Corti, consisting of rows of hair cells, rests on the basilar membrane. Over the top of the hair cells, and actually attached to some of them, is the **tectorial (roof) membrane**. The tectorial membrane is attached to the cochlear duct at only one side and can move independently from the basilar membrane.

Several structural features of the basilar membrane are relevant to its response to sound. The membrane is about five times wider at its apex (farthest from the oval window) than at its base (next to the oval window). In addition, the basilar membrane is about 100 times stiffer at its base than at its apex. These structural differences are similar to the range of size and flexibility found in the different strings of a guitar. When vibration produces pressure changes within the cochlea, the basilar membrane responds with a wave-like motion, similar to the motion of a rope or whip that is snapped. It will move less at the stiff, smaller end near the base than at the wide, floppy end at the apex. As shown in ●Figure 7.5, high-frequency sounds will cause a peak vibration of the basilar membrane near its base, whereas low-frequency sounds will cause a peak vibration closer to its apex.

The movement of the basilar membrane is sensed by the hair cells attached to the organ of Corti. Out of the approximately 15,500 hair cells in each human inner ear, about 3,500 of them are known as **inner hair cells**, which are the actual auditory receptors. The inner hair cells are located near the connection between the tectorial membrane and cochlear duct. The remaining 12,000 hair cells are known as **outer hair cells**, which appear to amplify sound. Both types of cells have hairlike **cilia** extending from their tops. Although there are many more outer hair cells in the ear, only 5 percent of the **auditory nerve (cranial nerve VIII)** fibers connect with outer hair cells. The remaining 95 percent of auditory nerve fibers connect with the inner hair cells.

●Figure 7.6 shows how movement of the cilia back and forth within the endolymph alternately hyperpolarizes and depolarizes the hair cells away from their resting potential of approximately -70 mV. The amount of movement needed to produce a response in the hair cells is quite small. If cilia were the size of the Eiffel Tower in Paris, the movement required to produce a response would equate to only 1 cm (about 0.4 inches; Hudspeth, 1983). The depolarization and hyperpolarization of the hair cells result from the opening and closing of mechanically gated potassium channels located in the tips of the cilia. When all of the cilia are straight up, as in a completely quiet environment, the channels are partially open. Small amounts of potassium will enter the cell. When the cilia bend one way, tension on the filaments connecting adjacent cilia opens the channels further, and greater amounts of potassium will enter the cell. Bending the cilia in the opposite direction releases the tension on the filaments, closing the channels.

Normally, when potassium channels are opened, potassium leaves a neuron, causing hyperpolarization (see Chapter 3). However, unlike most extracellular fluid, the endolymph surrounding the hair cells contains a higher concentration of potassium than is found in the intracellular fluid of the hair cells. Consequently, when potassium channels in hair cells open, potassium will move into the relatively negative internal environment of the cell due to both diffusion and electrostatic pressure. Potassium's positive charge depolarizes the hair cell, leading to the opening of voltage-dependent

**apex** The part of the cochlea most distant from the oval window.

**perilymph** Fluid found in the vestibular and tympanic canals of the inner ear.

**cochlear duct** The middle of three chambers of the cochlea.

**endolymph** The fluid found in the cochlear duct.

**Reissner's membrane** A membrane that separates the vestibular canal and cochlear duct.

**basilar membrane** A structure in the cochlea that separates the tympanic canal and the cochlear duct.

**round window** A membrane covering the end of the tympanic canal.

**organ of Corti** A structure within the cochlear duct responsible for transducing vibrations in the inner ear into action potentials.

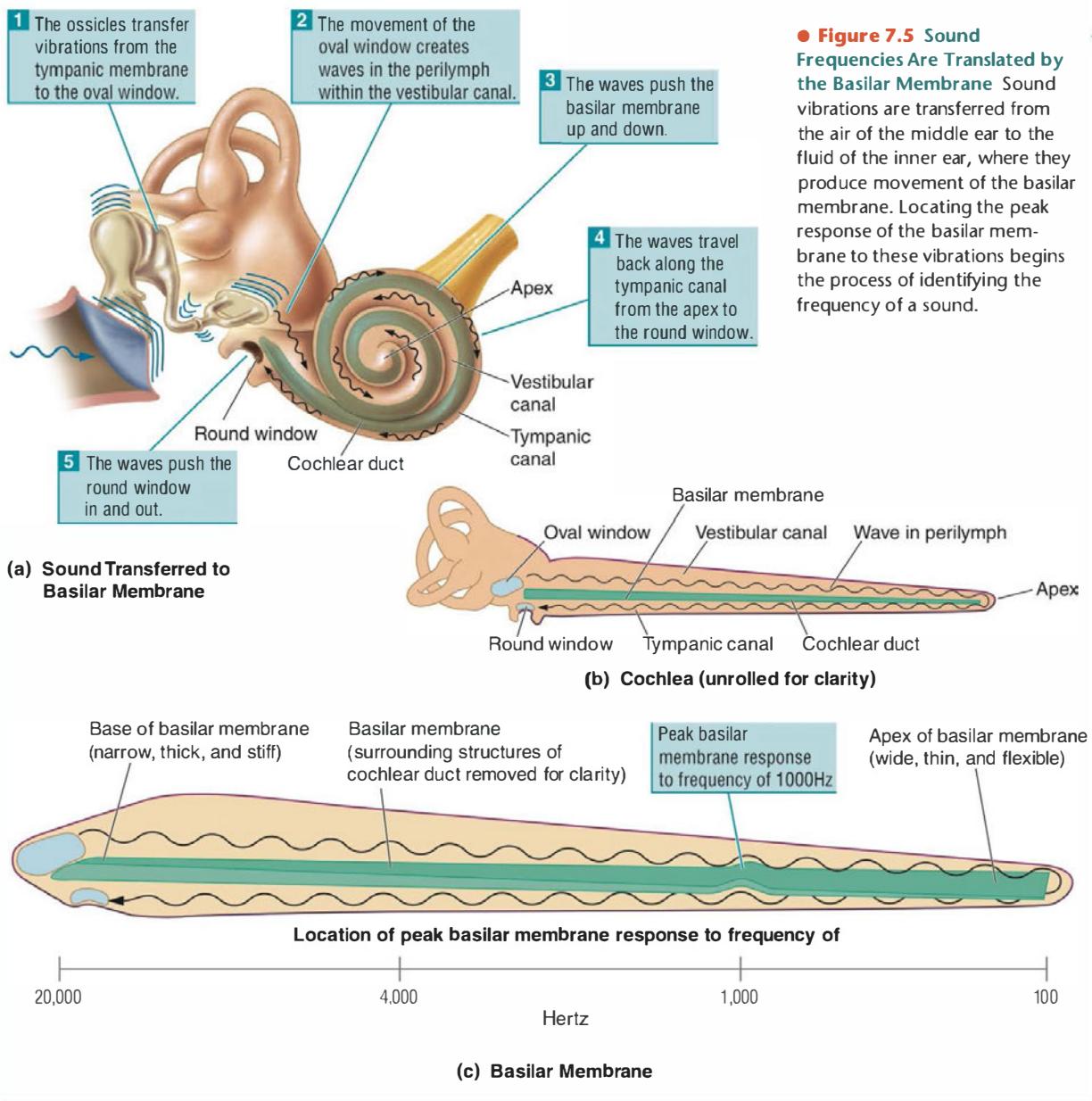
**tectorial (roof) membrane** A membrane that covers the organ of Corti.

**inner hair cells** Auditory receptor cells located near the junction of the tectorial membrane and cochlear duct.

**outer hair cells** Auditory receptor cells located on the Organ of Corti that amplify sound.

**cilia** Microscopic hair-like projections from a cell.

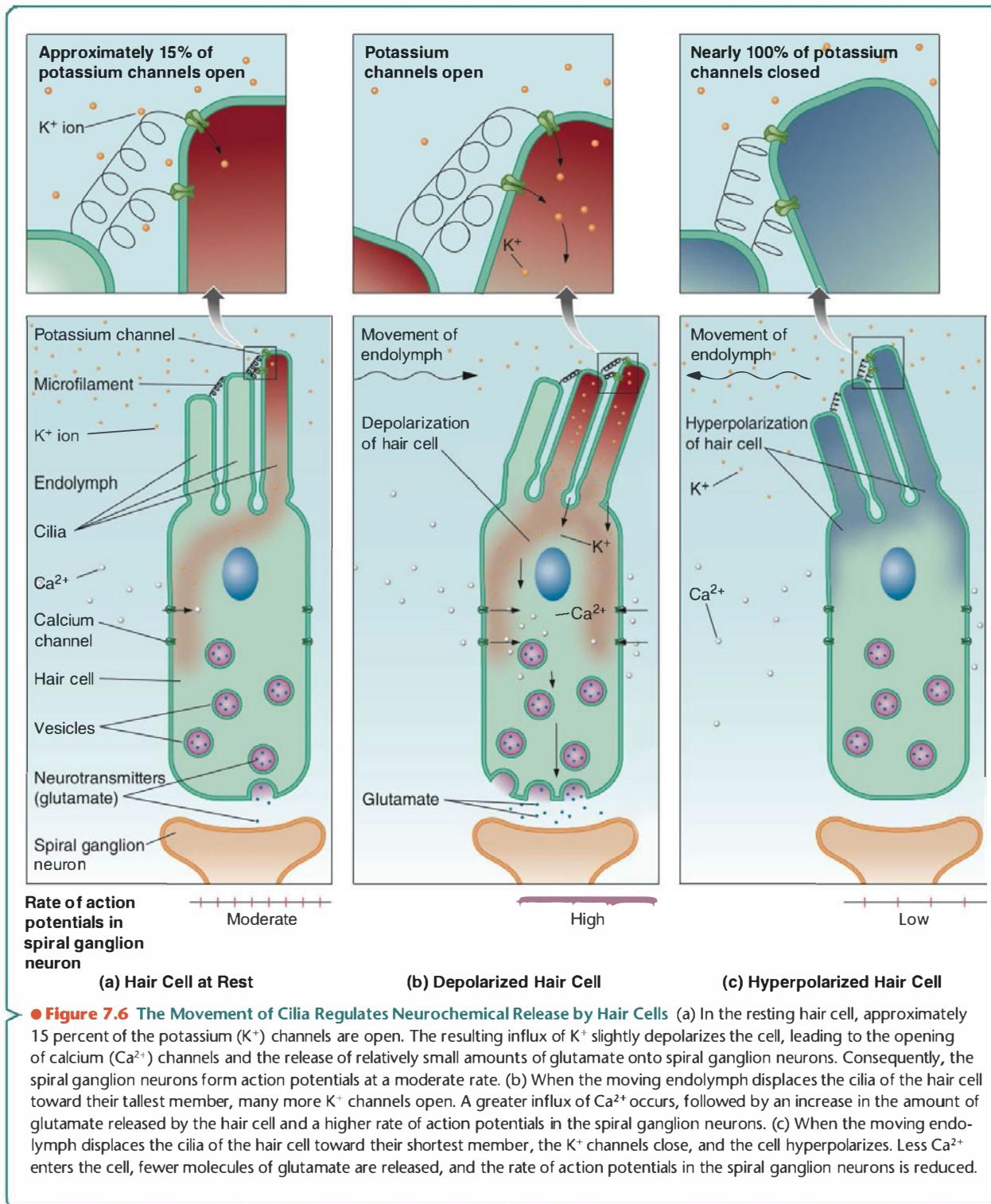
**auditory nerve (cranial nerve VIII)** The nerve that makes contact with the hair cells of the cochlea.



**spiral ganglion neuron** Bipolar neuron found in the inner ear whose axons form the auditory nerve.

calcium channels and neurochemical release. Most hair cells release the excitatory neurochemical glutamate.

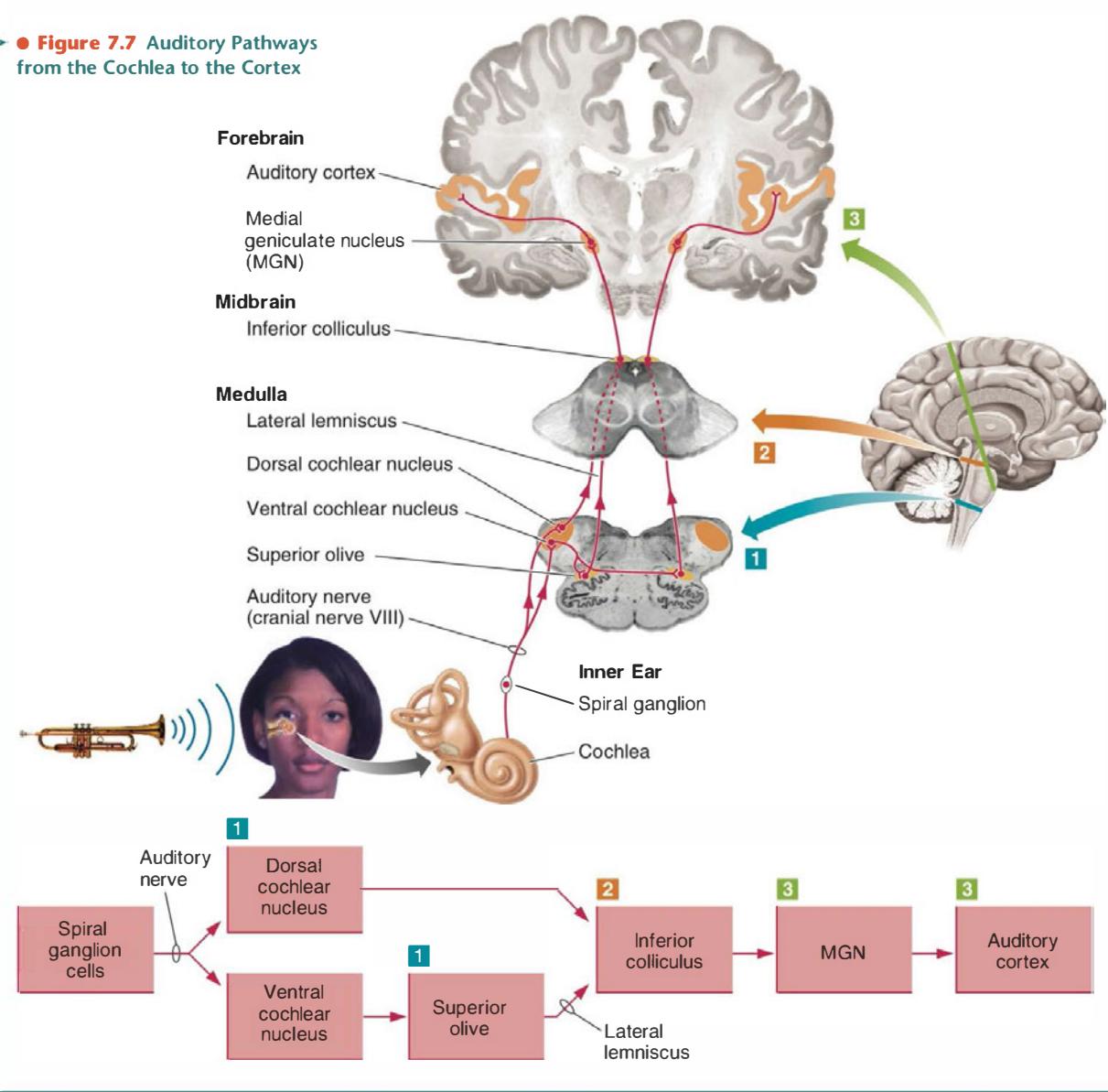
**CENTRAL AUDITORY PATHWAYS** As shown in Figure 7.7, **spiral ganglion neurons**, which are bipolar in structure, connect the hair cells of the cochlea with the brain. Cell bodies of the spiral ganglion neurons are located in the cochlea. One set of their fibers makes contact with the hair cells. The other set projects to the dorsal and ventral cochlear nuclei of the medulla as part of the auditory nerve (cranial nerve VIII). Input from both cochlear nuclei eventually reaches the inferior colliculus of the midbrain. However, axons from the ventral cochlear nuclei first synapse in the superior olive in the pons, which in turn forms connections via a pathway known as the lateral lemniscus with the inferior colliculus. Neurons from the inferior colliculus project



to the **medial geniculate nucleus (MGN)** of the thalamus. In addition to auditory information, the MGN also receives input from the reticular formation of the brainstem. This input adjusts hearing sensitivity based on the organism's state of arousal. The MGN in turn projects to the primary auditory cortex located in the temporal lobe.

**medial geniculate nucleus**  
(MGN) Nucleus of the thalamus  
that receives auditory input.

● **Figure 7.7** Auditory Pathways from the Cochlea to the Cortex



**THE AUDITORY CORTEX** Primary auditory cortex, also known as A1, is located in the temporal lobe, just below the lateral sulcus (see ● Figure 7.8).

Primary auditory cortex is organized in columns that respond to single frequencies. Lower frequencies produce a response in columns located rostrally in A1, whereas higher frequencies produce a response in columns located in the more caudal portions of the area. In some columns, input received by both ears produces a stronger response than input received by a single ear. In other columns, the opposite holds true: input received from a single ear produces a stronger response than input received by both ears. Other neurons within A1 respond to differences in intensity.

Surrounding A1 are areas known collectively as **secondary auditory cortex**. These areas appear to be activated by more complex types of stimuli such as clicks, general bursts of noise, and sounds with particular frequency patterns. Similar to observations in our discussion of vision, separate pathways originating in these areas process the quality of a sound (“what”) and its location (“where”) (Rauschecker, 2011; Rauschecker & Tian, 2000).

#### primary auditory cortex (A1)

Cortex located just below the lateral fissure in the temporal lobe that provides the initial cortical processing of auditory information.

**secondary auditory cortex** Areas surrounding A1 in the temporal lobe that process complex sound stimuli.

## Auditory Perception

Now that we have an understanding of the structures involved with audition, we can turn our attention to the perception of pitch, loudness, and the localization of sounds.

**PITCH PERCEPTION** We associate pitch (the high or low quality of a sound) with frequency, although that is an overly simplistic view. Pitch can vary due to factors other than frequency, such as the intensity or context of a stimulus. For example, listeners perceive a bigger increase in pitch when comparing tones of 500 Hz and 1,000 Hz than they do when comparing tones of 3,000 Hz and 3,500 Hz, although the difference in frequency is the same in both cases.

Most frequencies are systematically encoded by the auditory system through **tonotopic organization**, which describes the fact that neurons responding to one frequency are located next to neurons responding to similar frequencies. Tonotopic organization is found throughout the auditory system, from the basilar membrane up through primary auditory cortex.

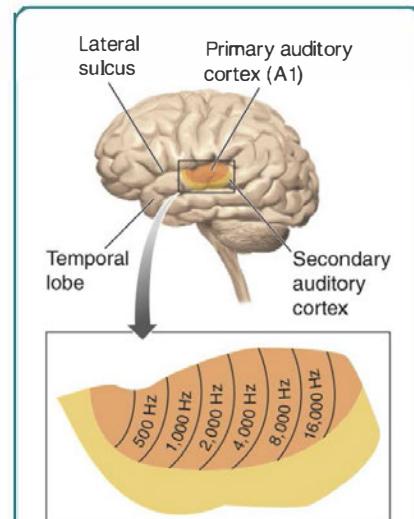
As a result of this tonotopic organization, one cue for assessing the frequency of a sound is the location of active neurons. Georg von Békésy's **place theory** explains the tonotopic organization of the basilar membrane. According to this theory, the peak of the wave traveling along the length of the basilar membrane is correlated with a sound's frequency. Place theory works well for sounds above 4,000 Hz. Below frequencies of 4,000 Hz, the response of the basilar membrane does not allow for precise localization. In these cases, **temporal theory**, in which patterns of neural firing match the actual frequency of a sound, provides a better model than place theory for the processing of sound frequency.

**LOUDNESS PERCEPTION** Although the decibel level of a sound wave and its perceived loudness are related, they are not the same thing. Decibels describe the physical qualities of the sound stimulus, whereas loudness is the human perception of that stimulus. The perception of loudness does not change at the same rate as the decibels do. Loudness doubles with each 10 dB increase in stimulus intensity (Stevens, 1960). In other words, a stimulus that is 10 dB (or 10 times) greater than another is perceived as only twice as loud.

Our ability to detect loudness varies with the frequency of a sound. By allowing participants to adjust the intensity of different tones until they sound equally loud, we can plot functions known as equal loudness contours (see Figure 7.9). To construct these curves, a 1,000 Hz tone (just above B5 or key 63 on a piano) is presented as a model. The 40 dB curve indicates how loud tones of other frequencies must be to be perceived as being as loud as the 40 dB 1,000 Hz comparison tone. Low frequencies are usually perceived as quieter than high frequencies at the same level of intensity. At very high intensities of 80 to 100 dB, you can see that all frequencies are perceived as being nearly equally loud.

Auditory neurons can respond to higher sound amplitudes by increasing their rate of response. However, the range of sound amplitudes we can hear is too broad to be completely encoded in this manner. Normally, a single neuron can respond to a range of about 40 dB, whereas at some frequencies, humans can perceive a range of 130 dB. Although a single neuron might have a limited range of 40 dB, a population of neurons with different ranges can provide the coverage we require. In addition, although auditory neurons have a preferred frequency to which they respond, they will in fact respond to similar frequencies if amplitude is high enough. The recruitment of these additional neurons contributes to our perception of loudness.

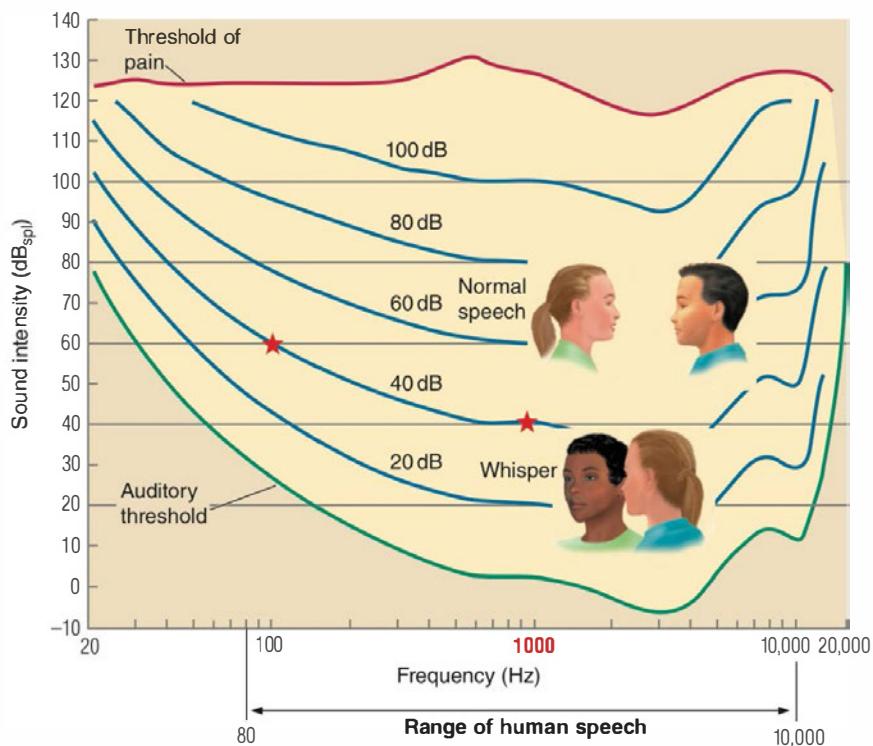
Sounds that last longer are usually perceived as louder. This is due to temporal summation, or an addition of neural responses over time (see Chapter 3). In audition, temporal summation occurs for 100 to 200 milliseconds (msec). A tone lasting 50 msec will be perceived as softer than a tone with the same frequency and amplitude that lasts 300 msec. Tones lasting more than 300 msec, however, will not sound louder than the 300 msec tone.



● **Figure 7.8** **Tonotopic Organization Is Maintained by the Auditory Cortex** Neurons responding to lower frequencies are located in the rostral portions of A1, and those responding to higher frequencies are located in the more caudal portions.

**tonotopic organization** Neurons responding to one frequency are located next to neurons responding to similar frequencies.  
**place theory** The peak response of the basilar membrane is correlated with a sound's frequency.  
**temporal theory** For frequencies below 4000 Hz, the pattern of neural firing matches the frequency of a sound.

**Figure 7.9 Equal Loudness Contours** Perceived loudness is not the same thing as sound intensity, measured in dB and represented by the horizontal lines in this graph. Each curve in this graph represents the sound intensity (dB) at which tones of each frequency match the perceived loudness of a model 1,000 Hz tone at 40 dB. The red stars indicate that a 100 Hz tone at 60 dB sounds about as loud as a 1,000 Hz at 40 dB, because they fall along the same line. Sensitivity is best for sounds having the frequencies typical of speech and rises on either side. This means that sounds with frequencies outside the range of speech must have more intensity to be perceived as well as speech sounds. At high levels of sound intensity, the curves flatten because all frequencies are perceived as being nearly equally loud.

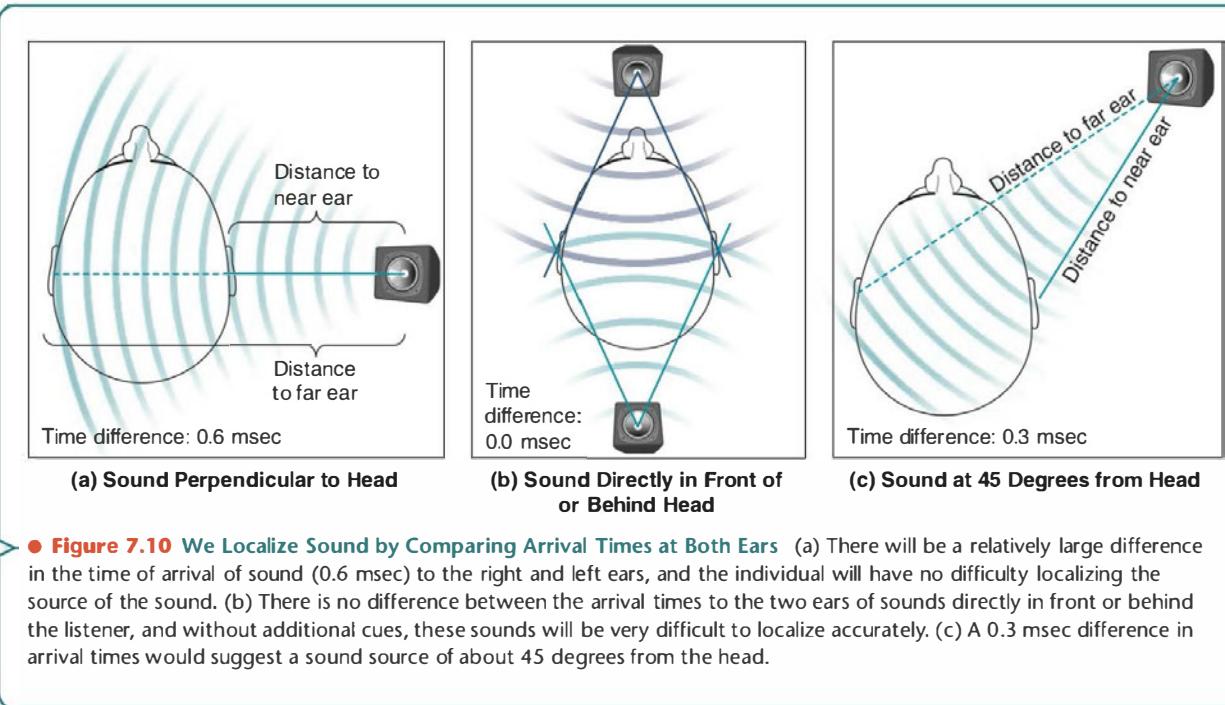


**LOCALIZATION OF SOUND** One of our primary means of localizing sound in the horizontal plane (in front, behind, and to the side) is a comparison of the arrival times of sounds at each ear. The differences in arrival time are quite small, between 0 msec for sounds that are either straight ahead or behind you to 0.6 msec for sounds coming from a point perpendicular to your head on either side (see **Figure 7.10**). Because arrival times from sounds immediately in front or behind you are identical, these sounds are very difficult to localize accurately.

Distinctions between arrival times of sound at each ear are made by neurons in the superior olive. These neurons are known as binaural neurons because they receive input from both ears. Binaural neurons respond most vigorously when input from both ears reaches them simultaneously. If input from the two ears arrives at slightly different times, the cells will respond less vigorously.

In addition to using differences in arrival times to the two ears to localize sounds, we can also localize sound by assessing the differences in the intensities of sound reaching each ear. Because the head blocks some sound waves, a sound “shadow” is cast on the ear farthest away from the source of sound, producing a quieter signal to that ear. However, this system works only for high frequency sounds. Because of their larger wavelengths, lower-frequency sounds move around the head without producing a noticeable shadow.

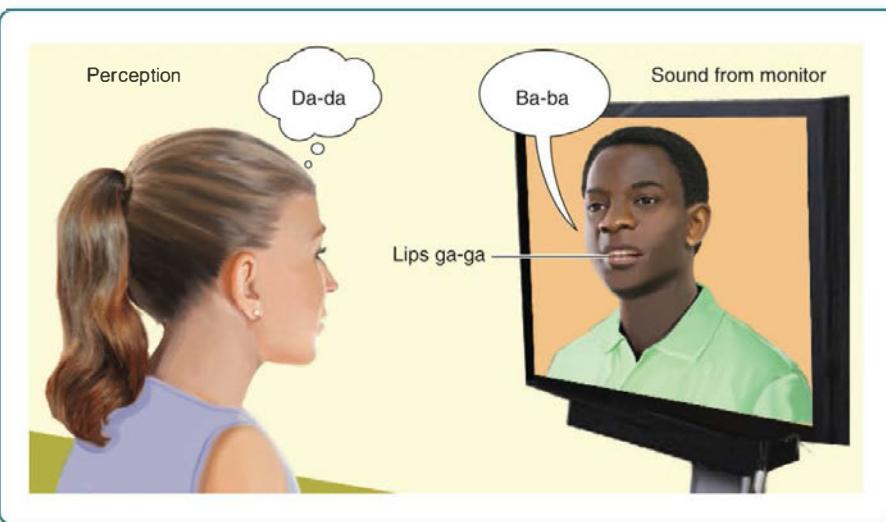
The pinna of the ear is essential for localizing the elevation of sounds in the vertical plane (above or below). When different-shaped false pinnae were attached to human participants, sound localization was impaired. However, with practice wearing their new pinnae, the participants learned to localize sounds correctly (Hofman, Van Riswick, & Van Opstal, 1998). Sound localization also involves vision. While watching a movie, we perceive sound as originating from the actors’ lips, in spite of the fact that the speakers producing the sound are typically above and to the sides of the screen (Alais & Burr, 2004). The McGurk effect, illustrated in **Figure 7.11**, is an illusion that results from our combined perceptions of vision and hearing.



## Hearing Disorders

Hearing loss arises from a wide variety of causes and affects nearly 30 million Americans (Agrawal, Platz, & Niparko, 2008; NIDCD, 2016). A person is considered legally deaf when speech sounds of 82 dB or less cannot be heard. Typical speech occurs at about 60 dB.

Age-related hearing loss results from a variety of factors, including poor circulation to the inner ear or the cumulative effects of a lifetime of exposure to loud noise. After the age of 30, most people cannot hear frequencies above 15,000 Hz. After the age of 50, most people cannot hear sounds above 12,000 Hz, and people over 70 have difficulty with sounds over 6,000 Hz. Because speech normally ranges up to 8,000 Hz, many elderly people begin to have difficulty understanding the speech of others. Teens who



frequent concerts and clubs are much more likely to report hearing problems (Chung, Des Roches, Meunier, & Eavey, 2005; Holgers & Pettersson, 2005). Earbud devices, such as those used by the popular iPhone, boost signals 6 dB to 9 dB and tend to be used for long periods due to their convenience. Experts recommend the 60–60 rule, or using such devices for no more than an hour per day (60 minutes) at no more than 60 percent of their maximum volume, to avoid hearing loss (Garstecki, 2005). Everyday noises from vehicles, machinery, and appliances can also contribute to hearing loss.

Hearing loss resulting from problems in the outer or middle ear is referred to as **conduction loss**. Conduction loss can result from a buildup of wax in the ear canal, infections of the middle ear, and a disease known as **otosclerosis**. Most cases of otosclerosis occur when the stapes (stirrup) becomes immobilized by a buildup of abnormal bone at its base. People with conduction loss can be helped by use of a hearing aid, which acts by amplifying sound signals.

Hearing loss also occurs due to damage to the inner ear, the auditory pathways, or the auditory cortex. Medications, such as quinine and some antibiotics, damage hair cells in sensitive individuals. Nicotine produces hearing loss by reducing blood supply to the ear (Zelman, 1973). Secondhand smoke exposure was sufficient to reduce hearing in adolescents (Lalwani, Liu, & Weitzman, 2011). Damage to the inner ear hair cells is often treated with **cochlear prosthetics**, or “cochlear implants.” As shown in Figure 7.12, electrode arrays are threaded through the round window of the cochlea toward the apex of the basilar membrane. The electrode arrays receive radio signals from a small microphone positioned on the outside of the head behind the ear. In turn, the electrode arrays stimulate auditory nerve fibers. While not the equivalent of the hair cells, the cochlear prosthetics have improved the hearing of large numbers of people.

**conduction loss** Hearing loss due to problems in the outer or middle ears; treated with the use of hearing aids.

**otosclerosis** Hearing loss due to immobilization of the ossicles of the middle ear.

**cochlear prosthetics** Electrode arrays inserted in the cochlea to treat hearing loss due to damaged inner ear hair cells.

## Building Better HEALTH

### EARBUDS AND HEARING LOSS

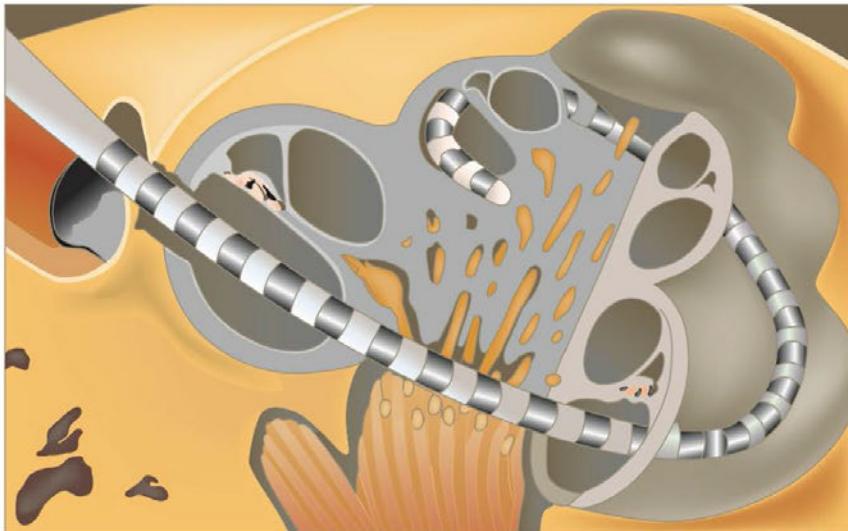
Certain types of hearing loss are associated with cumulative noise exposure over a person's lifetime. Among the risk factors for hearing loss today is the use of headphones to listen to music. As mentioned previously in this chapter, the volume of an iPhone or similar device can easily exceed 120 dB, about the same level of sound intensity produced by a propeller plane at takeoff. This level is only 10 dB below the point at which a sound would be considered painful and can produce hearing loss in as little as 75 minutes. Experts recommend that consumers set the volume of their devices to about 60 percent of maximum and limit their use to about an hour per day (the 60/60 rule), but such recommendations are rarely heeded.

In one study of adolescents and young adults between the ages of 16 to 25 years, approximately 15 percent showed hearing threshold changes at high frequencies, indicative of the type of permanent hearing damage we usually associate with older adults (Dobrucki, Kin, & Kruk, 2013). These researchers also investigated the impact of the type of headphones used on changes in hearing. Headphones were described as closed, semi-open, open, and in-ear. “Openness”

refers to the presence of openings in the back of the earphone, and these varieties are designed for their acoustic qualities rather than safety. Of the four types, use of the in-ear headphones (earbuds) appeared to produce the greatest changes in hearing thresholds.

Compounding the problem is the fact that we adapt rapidly to loudness. To experience the same subjective level of loudness over time, people tend to gradually increase the loudness of their electronic devices. How do you know when enough is enough? In general, if the listener cannot hear anything other than the sound from the device, the sound is far too loud. Symptoms such as ringing or buzzing in the ears, difficulty understanding speech in noisy places, sounds perceived as muffled or coming through a plugged ear, and awareness that television and other electronic device settings must be consistently higher than in the past could indicate early hearing loss and should be evaluated by a professional.

Using headphones other than earbuds, limiting listening time, and using device settings to cap maximum volume (in case we're tempted to adjust due to adaptation) can help preserve hearing.



**Figure 7.12 Cochlear Prosthetics** The electrode array of the cochlear prosthetic is threaded through the cochlea. Sounds picked up by an external microphone are encoded into electrical signals and sent to the electrode array. The signals from the array are picked up by the spiral ganglion fibers and transmitted to the brain.

## THINKING *Ethically*

### COCHLEAR PROSTHETICS AND DEAF CULTURE

It might come as a bit of a surprise to people who can hear that those who cannot hear did not welcome the discovery of cochlear prosthetics in the 1980s and 1990s with open arms (Sparrow, 2005). The choice by some hearing parents to have cochlear prosthetics inserted in their young children was viewed as particularly threatening. Debate over the use of cochlear prosthetics raises interesting questions about what it means to be "normal" and about the rights of minority cultures.

What is the basis for concerns about cochlear prosthetics? Critics of the cochlear prosthetic technology view this as an attempt to "cure" what they consider to be a culture, not a disability, and have even likened use of the technology to "genocide" (Sparrow, 2005). By ensuring that children grow up to use a spoken language instead of a visual (sign) language, the numbers of people with whom members of the deaf community could communicate in their first language would dwindle.

On the other side, supporters of the cochlear prosthetics argue that the technology provides the opportunity to communicate with hearing peers and does not necessarily mean that the child cannot also learn visual language and participate within the deaf community. Several decades of use of cochlear prosthetics

has provided some perspective on the outcomes of the procedure. Evaluations of adolescents who received cochlear prosthetics at a very early age show that 30 percent identified with the deaf community, 32 percent identified with the hearing community, and 38 percent identified with both the hearing and deaf communities (Moog, Geers, Gustus, & Brenner, 2011). Youth identifying with the hearing community did not experience higher levels of adjustment problems. Nearly all youth with cochlear prosthetics reported having hearing friends, and a majority reported having deaf friends as well.

Some people advocate for waiting until a child can make a decision about using cochlear prosthetics. As we will see in a later chapter on sexuality (Chapter 10), people who are viewed by others as "different" often report wishing that they could have input into the choices made on their behalf as opposed to having these choices made for them during childhood by well-intentioned adults. The fact that language acquisition is subject to an apparent critical period (see Chapter 5) complicates the timing of these choices. It is likely that the best outcomes do not emerge from a one-size-fits-all approach, but instead from approaches that are tailored to the individuals in question.

### INTERIM SUMMARY 7.1

#### || Summary Table: Important Structures Related to Audition

Structure	Location	Function
Pinna	Outer ear	Sound collection
Auditory canal	Outer ear	Resonating tube that conducts sound from the outer to middle ear
Tympanic membrane	Middle ear	Movement begins the process of transduction of sound waves to action potentials
Ossicles	Middle ear	Deliver vibration to the oval window of the cochlea
Cochlea	Inner ear	Fluid-filled structure containing auditory receptors
Inner hair cells	Inner ear	Frequency discrimination
Outer hair cells	Inner ear	Amplify responses to sound energy
Spiral ganglion	Inner ear	Source of fibers synapsing with hair cells; forms auditory nerve (cranial nerve VIII) that connects to the medulla
Dorsal cochlear nucleus; ventral cochlear nucleus	Medulla	Receives input from the spiral ganglion cells
Superior olive	Medulla	Transmits information from the ventral cochlear nucleus to the inferior colliculi; important for sound localization
Inferior colliculi	Midbrain	Sound localization; auditory reflexes
Medial geniculate nucleus of the thalamus	Diencephalon	Transmits sound information from the inferior colliculi to auditory cortex; may modulate output based on organism's level of arousal
Primary auditory cortex	A1 in the temporal lobe	Initial level of processing for auditory input
Secondary auditory cortex	Areas surrounding A1	Higher-level processing of auditory input

#### || Summary Points

1. Sound begins with the movement of an object in a medium, producing vibrations in surrounding media that can be described in terms of wavelength and amplitude. **(LO1)**
2. The outer ear contains the pinna and the auditory canal; the middle ear contains the tympanic membrane and ossicles. **(LO2)**
3. The cochlea of the inner ear is responsible for transducing sound waves into neural signals. These signals are carried centrally by the auditory nerve (cranial nerve VIII). Subsequent processing of auditory information occurs in the cochlear nuclei, the

superior olive, the inferior colliculi, the medial geniculate nucleus of the thalamus, and auditory cortex. (L02)

4. Auditory perception involves the analysis of pitch, sound intensity, and localization. (L03)

### Review Questions

1. What are the functions of the outer ear and the middle ear?
2. How does the auditory system process pitch and intensity?

## The Body Senses

The **somatosensory system** provides us with information about the position and movement of our bodies and about touch, skin temperature, and pain. Although these senses might not seem as essential as vision or hearing, we are severely disabled by their loss (see ●Figure 7.13). You might think it would be a blessing to be born without a sense of pain. On the contrary, people who have impaired pain reception typically die prematurely due to their inability to respond to injury.

### The Vestibular System

The **vestibular system** provides information about the position and movements of our heads, which contribute to our sense of balance. When the vestibular system is impaired, perhaps by a bad head cold or by motion sickness, the result is usually an unpleasant period of nausea and dizziness.

**MOVEMENT RECEPTORS** The sensory organs of the vestibular system are found in the inner ear, adjacent to the structures responsible for audition (see ●Figure 7.14). The vestibular structures may be divided into two types, the **otolith organs** and the **semicircular canals**. The otolith organs consist of two separate structures, the **saccule** and the **utricle**.

The otolith organs provide information about the angle of the head relative to the ground, as well as information about **linear acceleration**. We sense linear acceleration when our rate of movement changes, such as when our car pulls away from a stop sign. Both the saccule and utricle contain hair cells similar to those we encountered earlier in our discussion of audition. The hair cells in the saccule are arranged along a vertical membrane, whereas the hair cells in the utricle are arranged along a horizontal membrane. Cilia extend from each hair cell into a gelatinous layer. Covering the gelatinous layer are **otoliths**, which are stones made of calcium carbonate. When the otoliths move due to the acceleration of the head, force is exerted on the hair cells. The hair cells either depolarize or hyperpolarize in response to this force, which in turn affects the firing rates of fibers in the auditory nerve (cranial nerve VIII). Individual hair cells have a preferred direction of head movement to which they respond. As a result of this organization, all possible movements and directions of the head will be encoded by a unique pattern of hair cell responses.

**somatosensory system** The system that provides information about the body senses, including touch, movement, pain, and skin temperature.

**vestibular system** The sensory system that provides information about the position and movement of the head.

**otolith organ** A structure in the inner ear vestibular system that provides information about the angle of the head relative to the ground and about linear acceleration.

**semicircular canal** One of three looping chambers found in the inner ear that provide information regarding the rotation of the head.

**saccule** One of the structures making up the otolith organs.

**utricle** One of the structures making up the otolith organs.

**linear acceleration** The force we perceive when our rate of movement increases.

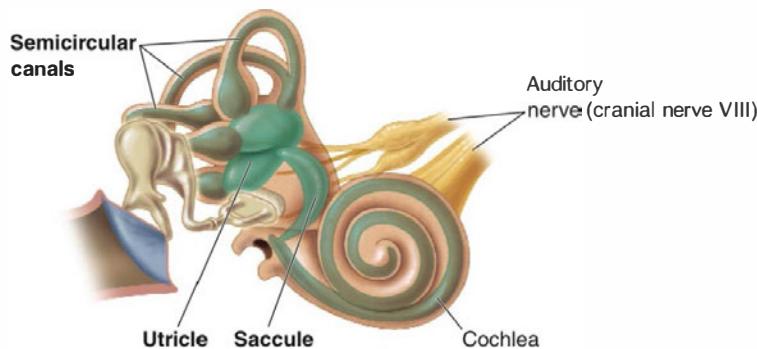
**otolith** A stone made from calcium carbonate that is attached to hair cells in the otolith organs.



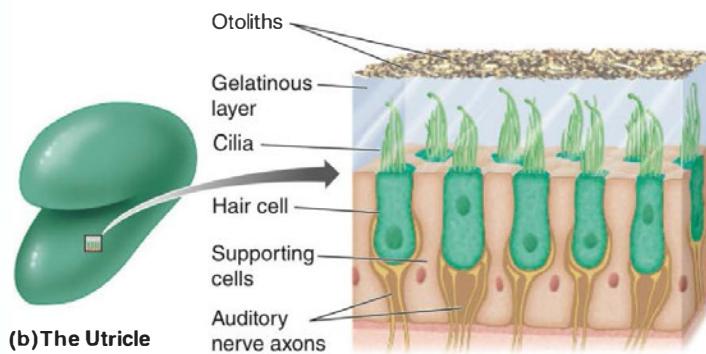
● **Figure 7.13 The Consequences of Feeling No Pain** Ashlyn Blocker was born with a rare condition preventing her from feeling any pain. She went several days with a broken ankle without complaint after falling off her bicycle.

Jeff Riedel/Getty Images

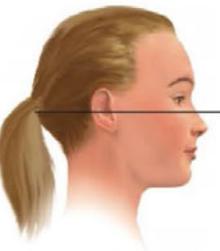
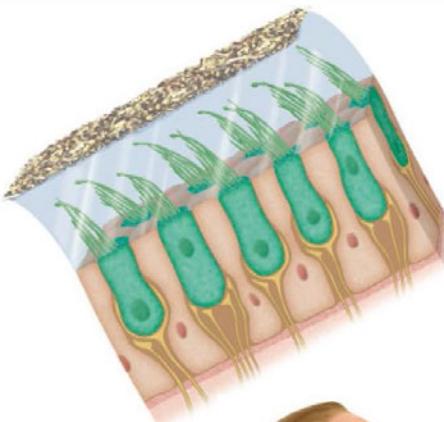
**Figure 7.14 The Vestibular Structures of the Inner Ear** (a) Together, the utricle, saccule, and semicircular canals provide information about the position and movement of the head. (b) The utricle and saccule contain hair cells, whose cilia extend into a gelatinous layer covered by otoliths. (c) Tilting the head exerts force on the cilia of the hair cells, which in turn modify signaling in the auditory nerve (cranial nerve VIII).



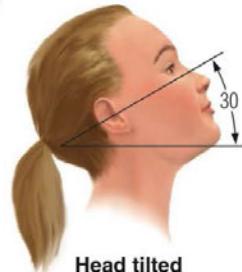
(a) The Vestibular System



(b) The Utricle



Head straight



(c) Activation of Hair Cells

The semicircular canals consist of three looping chambers at approximately right angles to one another. These structures respond to rotational movements of the head and contribute to our ability to walk upright (Fitzpatrick, Butler, & Day, 2006). Rotating the head causes the endolymph within the canals to bend hair cells. When extensive movement stops (perhaps at the end of an amusement park ride), the endolymph reverses its course, and you may have the odd fleeting sensation that your head is moving now in the opposite direction.

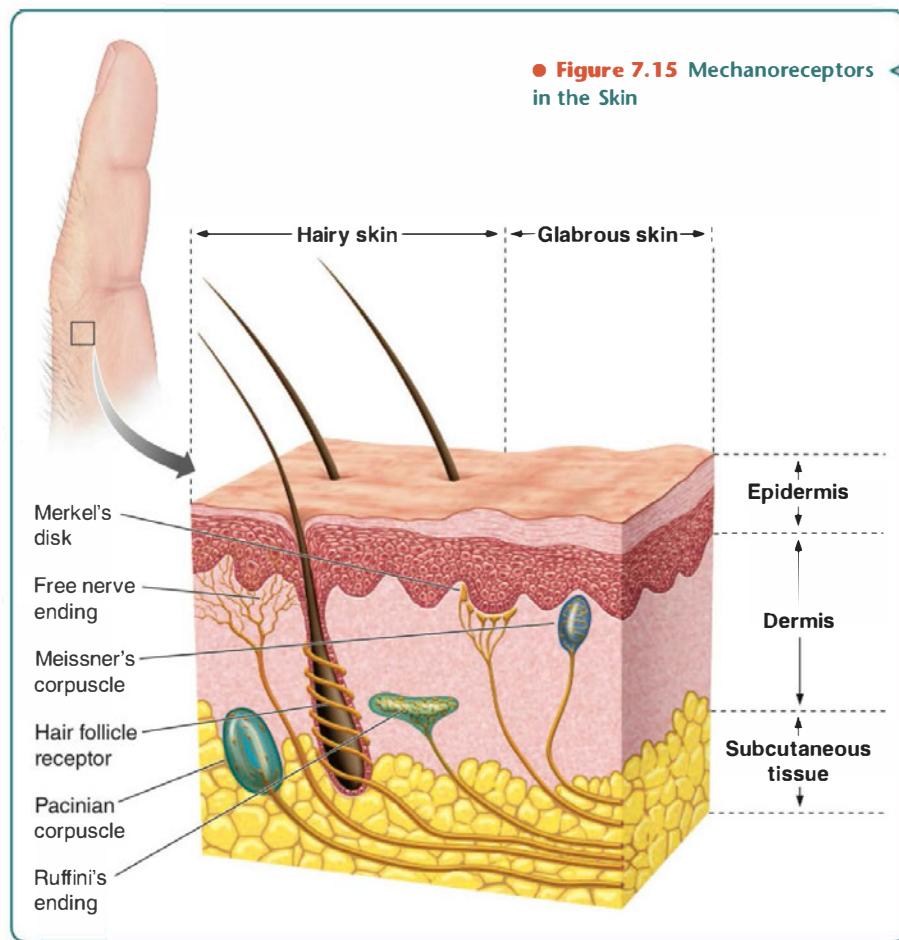
**CENTRAL PATHWAYS** Axons originating in the otolith organs and semicircular canals form part of the auditory nerve (cranial nerve VIII). These axons synapse in the vestibular nuclei of the pons and medulla and in the cerebellum. The cerebellum also participates in maintaining balance and motor coordination (see Chapter 2). In turn, information from the cerebellum, the visual system, and the somatosensory systems converge on the vestibular nuclei. This allows us to coordinate information from the vestibular system with other relevant sensory input.

Axons from the vestibular nuclei make connections both in the spinal cord and in higher levels of the brain. Input to the spinal cord motor neurons provides a means to adjust our posture to keep our balance. Vestibular nuclei axons also form connections with the **ventral posterior (VP) nucleus of the thalamus**, which receives information regarding touch and pain as well. From the VP nucleus, information is sent to the **primary somatosensory cortex** in the parietal lobe and primary motor cortex in the frontal lobe.

Input from the vestibular system is highly integrated with visual processing. It is essential for accurate vision that we maintain a stable view of our surroundings regardless of what our body is doing. Rotations of the head result in reflexive movements of the eyes in the opposite direction. As a result of these reflexes, you maintain a steady view of the world while riding on the most extreme roller coaster. On the other hand, if your vestibular senses and your visual senses give conflicting information to your brain, you may feel nauseated or dizzy.

## Touch

Our sense of touch begins with our skin, the largest and heaviest organ of the human body. Our skin provides a boundary separating what is inside from what is outside. It prevents dehydration and protects the body from dirt and bacteria. Human skin comes in two basic varieties, hairy skin and **glabrous**, or hairless, skin. Human glabrous skin is found on the lips, palms of the hands, and soles of the feet. When viewed in cross-section, as in ● Figure 7.15, the skin can be divided into the



### ventral posterior (VP) nucleus

The nucleus of the thalamus that receives information regarding pain, touch, and the position and movement of the head.

### primary somatosensory cortex

(S1) Cortex located in the postcentral gyrus of the parietal lobe that is responsible for the initial cortical processing of somatosensory input.

### glabrous

Hairless skin.

outer layer of **epidermis** and the inner layer of the **dermis**. The outermost layer of the epidermis is actually constructed of dead cells. Below the dermis, we find **subcutaneous tissue**, which contains connective tissues and fat. Human skin varies dramatically in thickness across different areas of the body, from about half a millimeter (0.02 inch) on your face to 20 times that thickness (10 mm or 0.4 inch) on the bottom of your foot.

**TOUCH RECEPTORS** The majority of the receptor cells for touch are referred to as **mechanoreceptors**. This term reflects the response of these receptor cells to physical displacement such as bending or stretching. In addition to their locations in the skin, mechanoreceptors are also found in our blood vessels, joints, and internal organs. Those unpleasant sensations of pressure from a too-full stomach or bladder are provided courtesy of mechanoreceptors in the walls of these organs.

Although mechanoreceptors come in a wide variety of shapes and sizes, they share a number of common features. Within each mechanoreceptor are unmyelinated axon fibers. The membranes of these axons contain sodium ion channels that respond to physical stretching or changes in membrane tension. When the membrane of the axon is stretched, the ion channels open, and sodium enters the cell. If sufficient amounts of sodium enter the cell, an action potential is generated.

<b>epidermis</b>	Outer layer of the skin.
<b>dermis</b>	The layer of skin below the epidermis.
<b>subcutaneous tissue</b>	The layer of tissue below the dermis that contains fat and connective tissue.
<b>mechanoreceptor</b>	A touch receptor that responds to physical displacement such as bending or stretching.
<b>encapsulated receptors</b>	A receptor in which the axon terminal is surrounded by a fluid-filled capsule formed of connective tissue.
<b>Meissner's corpuscles</b>	An encapsulated mechanoreceptor located near the surface of the skin that senses pressure.
<b>Pacinian corpuscles</b>	An encapsulated mechanoreceptor located deeper in the skin, in joints, and in the digestive tract that senses pressure and vibration.
<b>Merkel's disks</b>	A nonencapsulated mechanoreceptor located near the surface of the skin that senses pressure.
<b>Ruffini's endings</b>	A nonencapsulated mechanoreceptor located deep in the skin that senses stretch.

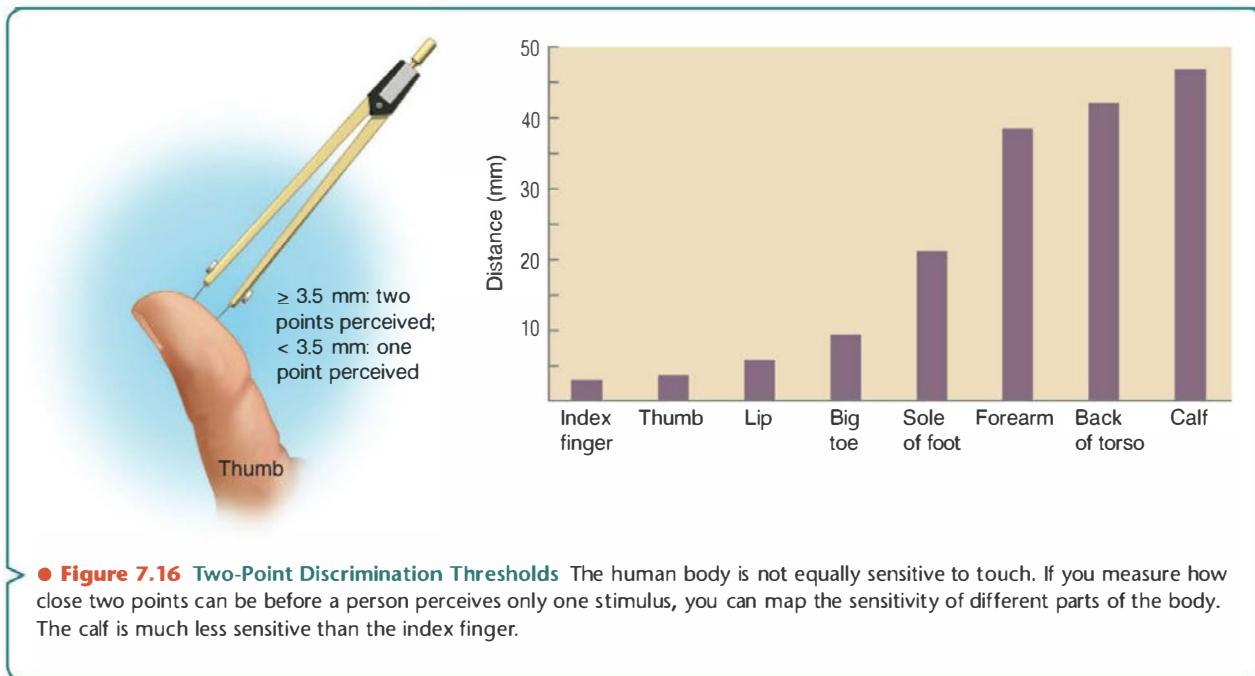
Mechanoreceptors are categorized according to their structure, size of receptive field, rate of adaptation, and type of information that is processed. Structurally, mechanoreceptors are either **encapsulated** or not. In **encapsulated receptors**, the axon fibers are surrounded by a fluid-filled capsule formed of connective tissue. The two major types of encapsulated mechanoreceptors are the **Meissner's corpuscles** and the **Pacinian corpuscles**. The Meissner's corpuscles are found at the junction of the epidermis and dermis, whereas the Pacinian corpuscles are located deep in the skin, in the joints, and in the digestive tract. Nonencapsulated receptors include the **Merkel's disks** and **Ruffini's endings**. The Merkel's disks, like the Meissner's corpuscles, are located in the upper areas of the skin, whereas the Ruffini's endings are located at deeper levels. We also find free nerve endings distributed within the skin. As their name implies, these receptors do not have any specialized structure but are simply the unmyelinated nerve endings of sensory neurons. In addition, some receptors wrap themselves around hair follicles and respond to the bending of a hair (see Table 7.3).

In vision, we spoke of a neuron's receptive field as the area of the retina in which light affects the activity of that neuron. In the case of touch, a receptive field describes the area of skin or other tissue that provides information to a particular receptor. Meissner's corpuscles and Merkel's disks both have very small receptive fields, which means that they can identify the borders of very small stimuli. In contrast, Pacinian corpuscles and Ruffini's endings have very large receptive fields and provide only general information about the borders of stimuli.

Variations in sensitivity from one part of the body to the next result from the density and receptive field size of the mechanoreceptors serving that area. Sensitivity

**TABLE 7.3** Major Features of the Mechanoreceptors

Mechanoreceptor	Encapsulated?	Rate of Adaptation	Size of Receptive Field	Quality of Stimulus Sensed
Meissner's corpuscles	Yes	Rapid	Small	Pressure
Pacinian corpuscles	Yes	Rapid	Large	Vibration
Merkel's disks	No	Slow	Small	Pressure
Ruffini's endings	No	Slow	Large	Stretch



of various parts of the body can be assessed using a two-point discrimination test (see ● Figure 7.16). This test measures how close together two stimuli have to be before the person can perceive only a single stimulus. Our fingers and lips are far more sensitive than our backs and the calves of our legs. Not only do fingers and lips have a greater density of mechanoreceptors overall than other areas of the body, but they also contain high concentrations of Merkel's disks and Meissner's corpuscles, with their small receptive fields.

A receptor's rate of adaptation refers to the length of time it will continue to respond to unchanging stimuli. A receptor that adapts rapidly will respond vigorously when stimulation begins or ends but will remain rather inactive while the stimulus is applied continuously. In contrast, a slow-adapting receptor will continue to respond steadily as long as the stimulus is present. We can classify our four major types of mechanoreceptors according to their rate of adaptation. Meissner's corpuscles and Pacinian corpuscles both demonstrate rapid adaptation, whereas Merkel's disks and Ruffini's endings are slow adapting.

A final feature of mechanoreceptors is the type of information processed. The relationship of mechanoreceptor type to the quality of the sensation is neither precise nor perfectly understood. In general, however, mechanoreceptors appear to be somewhat specialized in the type of information they provide. Free nerve endings typically supply information regarding pain, which we will discuss later in this chapter. Meissner's corpuscles, Merkel's disks, and Pacinian corpuscles all provide information about pressure. However, due to having larger receptive fields, the Pacinian corpuscles do not provide the fine spatial resolution that characterizes the Meissner's corpuscles and Merkel's disks. Pacinian corpuscles are superior to the others in detecting vibrating stimuli. The Ruffini's endings provide input regarding stretch.

**TOUCH PATHWAYS** We begin the journey from the mechanoreceptor in the skin back toward the brain by looking at the nerves that serve the receptors. The cell bodies of the mechanoreceptors are located in the dorsal root ganglia and their cranial nerve equivalents. As we observed in Chapter 3, these unipolar cells feature a single process

Class of Axon	Diameter of Axon	Speed of Transmission	Receptor Types
A $\alpha$ (Alpha-alpha) fibers	13–20 $\mu\text{m}$ Axon Myelin	80–120 m/sec	Feedback from muscle fibers
A $\beta$ (Alpha-beta) fibers	6–12 $\mu\text{m}$	35–75 m/sec	Mechanoreceptors of skin: Meissner's corpuscles Merkel's disks Pacinian corpuscles Ruffini's endings
A $\delta$ (Alpha-delta) fibers	1–5 $\mu\text{m}$	5–30 m/sec	Pain, temperature receptors of skin: Free nerve endings
C fibers	0.2–1.5 $\mu\text{m}$	0.5–2 m/sec	Pain, temperature receptors of skin: Free nerve endings

► **Figure 7.17 The Four Classes of Sensory Axons Differ in Size and Speed** The largest, fastest afferent axons (A $\alpha$ ) serve the muscles and will be discussed in Chapter 8. The second-largest and fastest axons (A $\beta$ ) serve the mechanoreceptors. Fast, sharp pain and skin temperature are carried by the myelinated A $\delta$  fibers, whereas the small, unmyelinated C fibers carry dull, aching pain and skin temperature.

leaving the cell body that branches. One branch extends to the periphery, ending in a mechanoreceptor. The other branch travels centrally by joining the dorsal roots of the spinal cord.

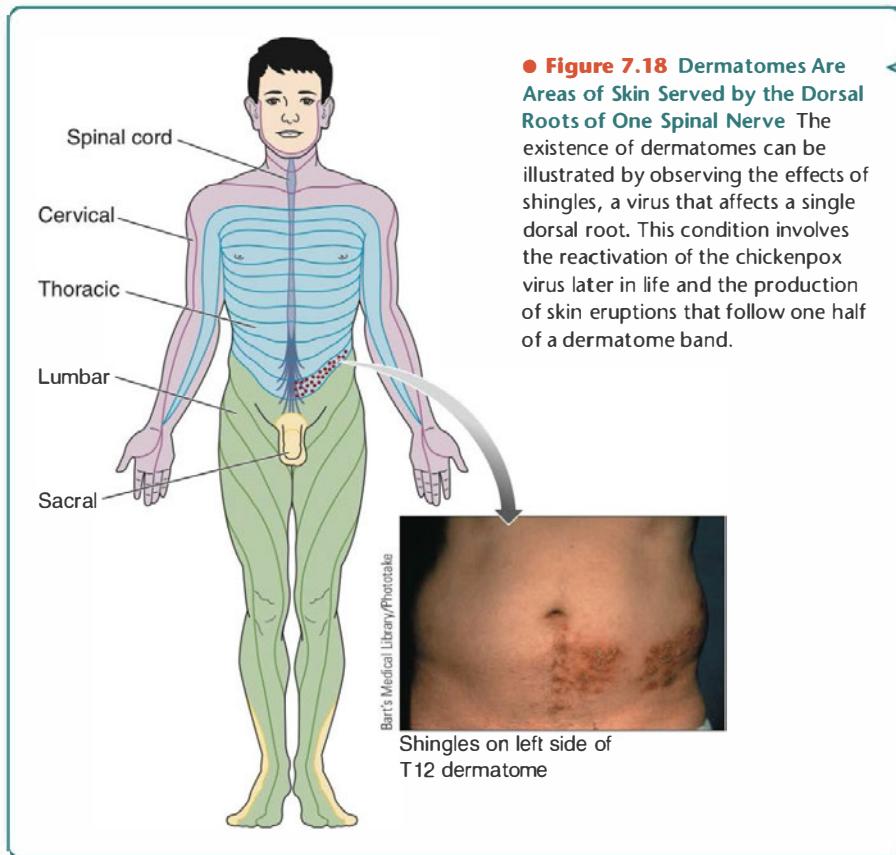
As shown in ► Figure 7.17, the sensory fibers of the peripheral nervous system are classified into four categories based on diameter and speed. The largest fibers, called A $\alpha$  (alpha-alpha), carry information from the muscles and will be discussed in Chapter 8. The smaller three sets of fibers serve the mechanoreceptors. The second-largest set, the A $\beta$  (alpha-beta) class, carries information from the Meissner's corpuscles, Merkel's disks, Pacinian corpuscles, and Ruffini's endings toward the central nervous system (CNS). The smallest two groups, the myelinated A $\delta$  (alpha-delta) fibers and the unmyelinated C fibers, carry information from the free nerve endings regarding pain and skin temperature.

The area of the skin surface served by the dorsal roots of one spinal nerve is known as a **dermatome**. As shown in ► Figure 7.18, dermatomes are easily identified in the disease known as shingles, which is caused by the same virus responsible for chickenpox (*varicella zoster virus* or VZV). In some people, the virus awakens decades after the original infection and produces skin eruptions. In addition, the virus increases the excitability of the sensory neurons, producing a burning, painful sensation along the dermatome. The reactivated virus appears to confine its mischief to a single dorsal root, leading to symptoms in one half of an individual dermatome. Fortunately, an effective vaccine for VZV is now available.

The pathways leading from the mechanoreceptors to the brain are illustrated in ► Figure 7.19. When the axons from the mechanoreceptors enter the spinal cord, they follow a route called the **dorsal column-medial lemniscal pathway**. As suggested by the pathway's name, axons join the white matter of the ipsilateral dorsal column and

**dermatome** The area of skin surface served by the dorsal root of a spinal nerve.

**dorsal column-medial lemniscal pathway** The pathway taken by touch information from the mechanoreceptors to the brain.

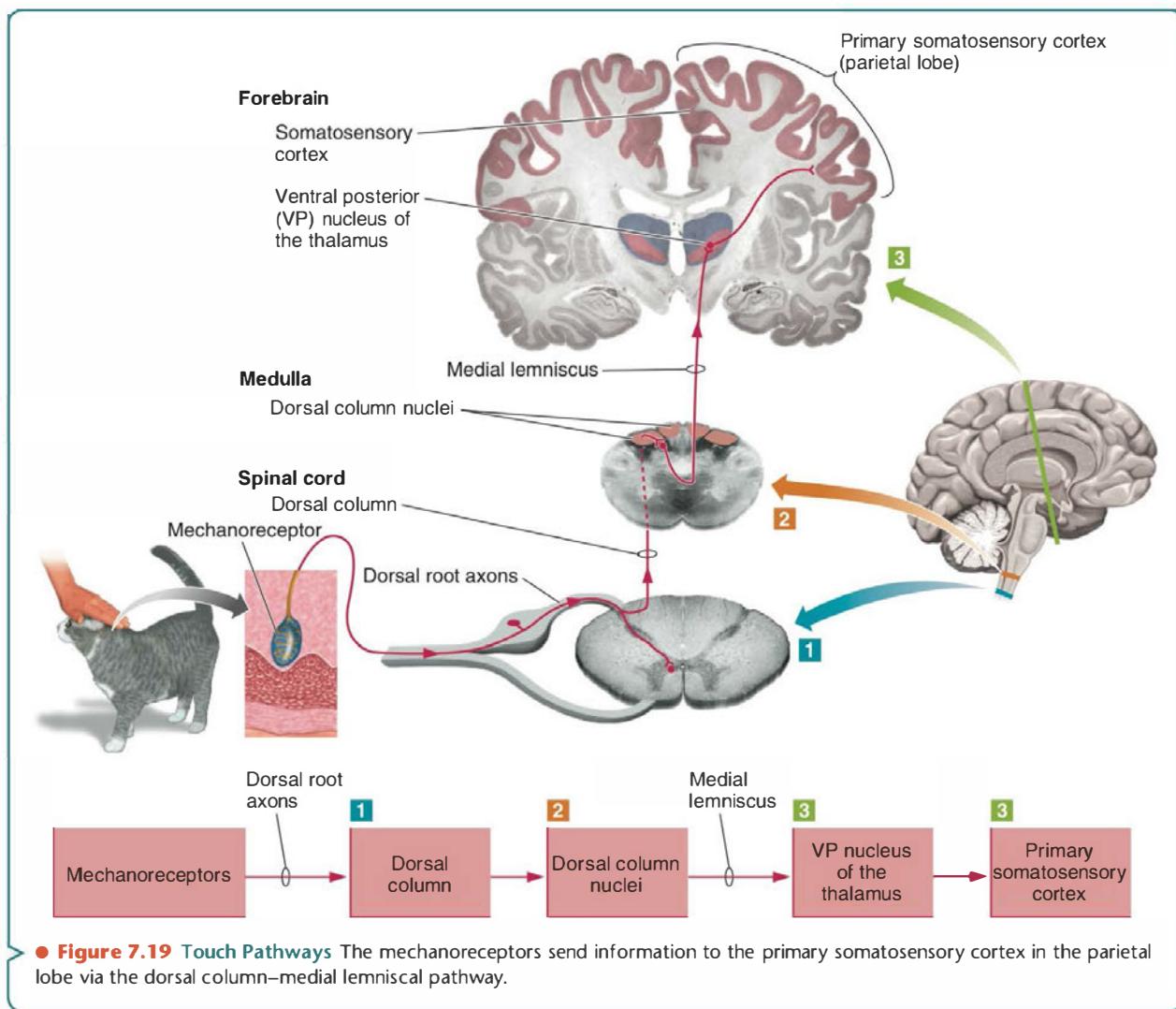


make their first synapse in the dorsal column nuclei of the medulla. The lack of synapses until this point greatly contributes to the speed of this system.

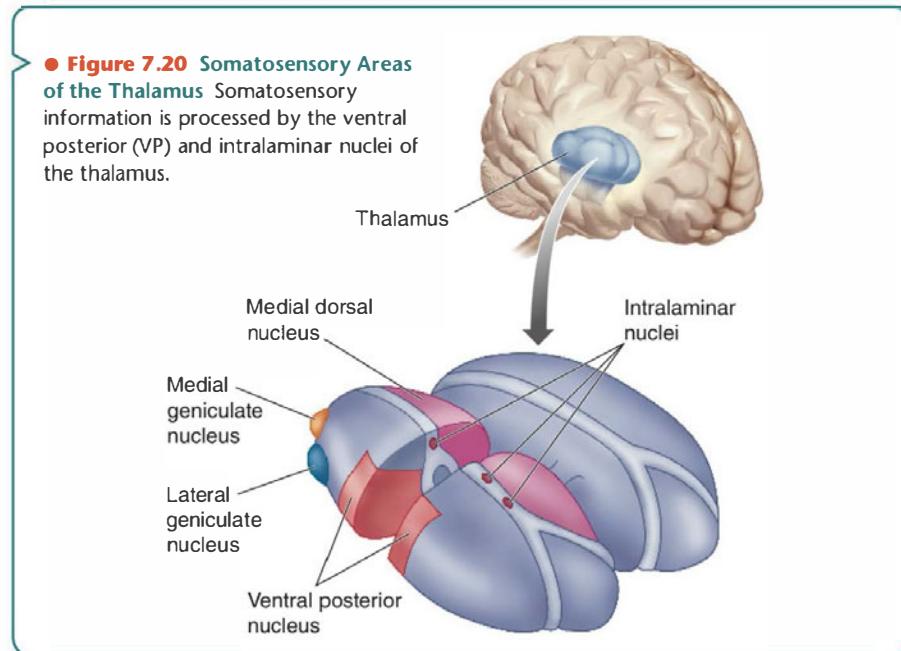
Axons from the dorsal column nuclei form a large band of white matter known as the medial lemniscus, which crosses the midline of the medulla. From this point forward, sensory information is processed contralaterally. In other words, the left side of the brain processes touch information from the right side of the body. The medial lemniscus continues to travel rostrally through the medulla, pons, and midbrain before synapsing on the ventral posterior (VP) nucleus of the thalamus, shown in ●Figure 7.20. Axons from the VP nucleus travel to the primary somatosensory cortex of the parietal lobe.

Touch information from the head reaches the VP nucleus by alternate routes involving a number of cranial nerves. Sensation from mechanoreceptors in the skin of the face, mouth, tongue, and the dura mater of the brain travels along branches of the trigeminal nerve (cranial nerve V). Additional input from other parts of the head is carried by the facial nerve (VII), the glossopharyngeal nerve (IX), and the vagus nerve (X). Axons forming these cranial nerves synapse on their respective ipsilateral nuclei in the brainstem, which serve the same purpose as the dorsal column nuclei. From these cranial nerve nuclei, axons cross the midline and travel to the VP nucleus, which in turn passes the information to the primary somatosensory cortex in the parietal lobe.

You may be wondering what the dorsal column nuclei, cranial nerve nuclei, and the VP nucleus might be doing to the incoming sensory information. These synapses provide opportunities for the cortex to influence the input it receives through descending pathways. In addition, activity in one neuron inhibits its neighbor, leading to a sharpening or enhancement of its signal.



● **Figure 7.19 Touch Pathways** The mechanoreceptors send information to the primary somatosensory cortex in the parietal lobe via the dorsal column–medial lemniscal pathway.



**SOMATOSENSORY CORTEX** Primary somatosensory cortex, also known as S1, is found in the postcentral gyrus of the parietal lobe, just caudal to the central sulcus that divides the parietal and frontal lobes. Secondary somatosensory cortex (S2) is located within the lateral sulcus.

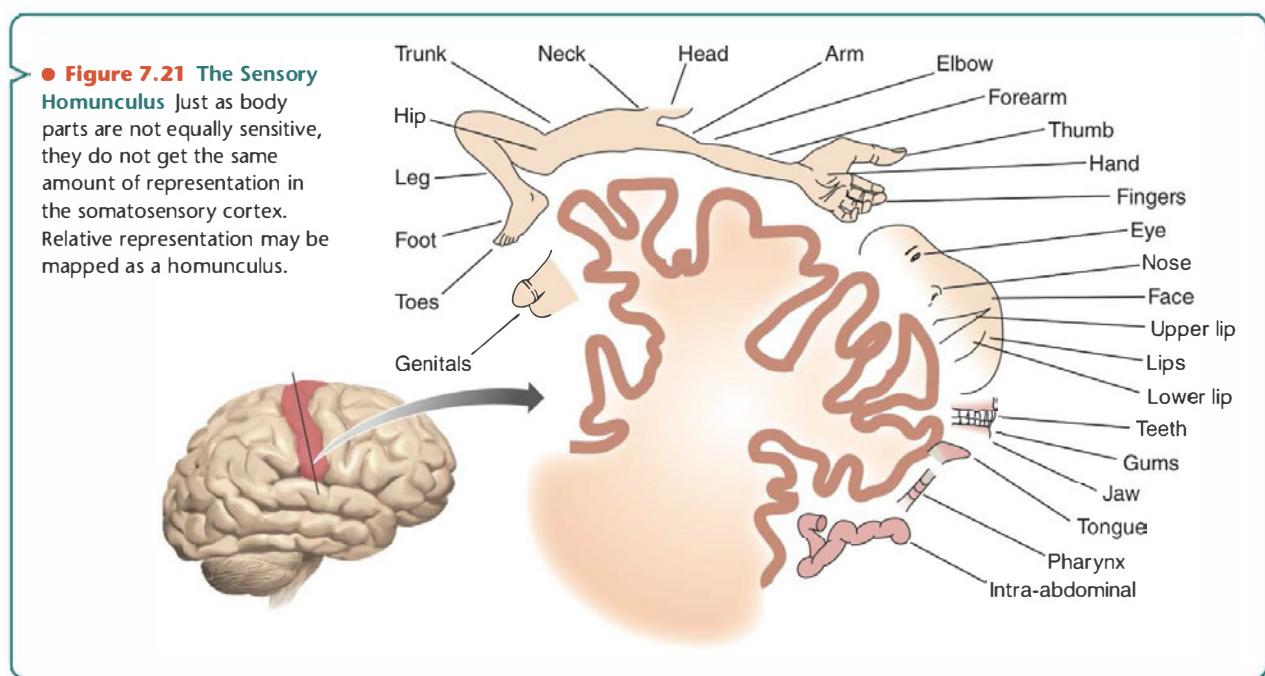
Using single-cell recording, we can demonstrate that areas of the cortex serving the head and neck are located at the lower, ventral part of the postcentral gyrus, whereas areas serving the legs and feet extend over the top of the gyrus onto the medial surface of the parietal lobe. A map of the body's representation in the cortex is known as a *homunculus*, or "little man." An example of a homunculus is shown in Figure 7.21. Areas of the body receive cortical representation according to their need for precise sensory feedback. In humans, the hands and face are given a much larger portion of the cortex than their size would suggest. Rats devote a great deal of space to whiskers, whereas lips seem to have a high priority in squirrels and rabbits.

**PLASTICITY OF TOUCH** The somatosensory cortex is capable of plasticity, which means it can rearrange itself in response to changes in the amount of input it receives. In adult monkeys who have had a finger surgically removed, the areas of the somatosensory cortex previously responsive to the missing finger now respond to stimulation of adjacent fingers (Kaas, Nelson, Sur, & Merzenich, 1981). In these cases, the brain is adapting to a reduction in input from a specific part of the body. Increased stimulation from a body part will also result in changes in the mapping of the somatosensory cortex. When monkeys were trained to use specific fingers to discriminate between tactile surfaces to earn a food reward, the area of the somatosensory cortex receiving input from the trained fingertips actually expanded (Wang, Merzenich, Sameshima, & Jenkins, 1995). This process represents a reorganization of synapses.

Plasticity occurs in the human somatosensory cortex as a result of both loss and enhancement of input. Amputation of a limb often causes **phantom pain**, in which pain is perceived as arising in the missing body part, or **referred sensations**, in which touching a body part such as the cheek is perceived as touch of the missing limb (Ramachandran, 2005). Brain imaging confirms that the part of somatosensory cortex previously responsive to an amputated arm subsequently responded to touching the face (Ramachandran & Rogers-Ramachandran, 2000). Other types of referred

**phantom pain** Pain that is perceived as arising from a missing body part.

**referred sensations** The perception of touch of a body part as arising from a missing body part.



## Connecting to Research

### PHANTOM LIMBS, MIRRORS, AND LONGER NOSES

Vilayanur Ramachandran and Diane Rogers-Ramachandran (2000) explored the reactions of both amputees and nonamputees to touch. As a result of their investigations, they propose a "remapping hypothesis" to explain the changes they observe resulting from amputation.

Among a group of 18 individuals with arm amputations, 8 perfectly referred sensation from the face to the now-missing limb. In other words, touching specific parts of the face produced simultaneous feelings of the face and the missing arm. The sensations were also modality specific, such as feeling hot or cold or massage.

Some patients experience movement as well as sensation in their phantom limbs. In some instances, this can result in pain. One patient examined by Ramachandran and Rogers-Ramachandran experienced the sensation of a tightly clenched hand that could not be relaxed. The phantom nails dug into the palm of the phantom hand, resulting in excruciating sensations of pain. Use of a mirror box to superimpose the reflection of the real hand on the felt location of the phantom left hand allowed the patient to gain relief by "unclenching" the phantom hand (see **Figure 7.22**).

Ramachandran and Rogers-Ramachandran (2000) argue that body image is remarkably flexible even in people who have not experienced amputation. You can try the "phantom nose" experiment yourself (Ramachandran & Hirstein, 1997). One person is blindfolded while seated. The second person sits to the side of the first person. The experimenter uses his or her left hand to take the blindfolded participant's left index finger and use it to tap or stroke the other person's nose in a random, unpredictable manner. Simultaneously, the experimenter



Media for Medical SAR/Alamy Stock Photo

**Figure 7.22 Mirror Boxes and Phantom Pain** Ramachandran's mirror box has two compartments. The patients place their affected limb in one side (covered) and their unaffected limb in the side with the mirror. The reflection gives the illusion of once again having two limbs, which relieves many symptoms of phantom limb.

uses his or her right index finger to perform the same movements on the blindfolded person's nose. After only a few seconds, the blindfolded person begins to sense that his or her nose has been stretched or dislocated.

How can we account for these odd sensations? Ramachandran and Hirstein (1997) propose that the participant assumes that the matched tapping of nose and finger cannot be occurring by chance, resulting in the brain's use of the "simpler" explanation that the nose is displaced. The authors further argue that sensory systems have evolved to extract statistical regularities from the natural world around us. Most of the time, that works, but illusions can illustrate how tenuous the relationship between "reality" and our beliefs about reality can really be.

sensation occur as well. One patient was embarrassed to report that he experienced a sensation of orgasm in his missing foot. Such observations suggest that the brain rapidly reorganizes its representation of the body following amputation.

The increased representation due to training that was observed in monkeys has a parallel in the cortical organization of some musicians. Highly trained string musicians have a larger than normal area of somatosensory cortex representing touch in the fingers (Münte, Altenmüller, & Jäncke, 2002). A similar reorganization of somatosensory cortex occurs when blind individuals learn to read Braille (Pascual-Leone & Torres, 1993). Neural plasticity in the somatosensory system is quite flexible. Sighted

participants who wore blindfolds initially activated only S1 while performing a Braille task (Pascual-Leone & Hamilton, 2001). After a few days, however, they began to recruit V1, or primary visual cortex, during the task. After no longer wearing blindfolds while reading Braille, they reverted to their initial pattern of S1 without V1 activity.

**SOMATOSENSORY DISORDERS** Damage to primary somatosensory cortex produces deficits in both sensation and movement of body parts served by the damaged area (Corkin, Milner, & Rasmussen, 1970). Damage to secondary somatosensory cortex, particularly on the right side of the brain, results in an odd phenomenon known as **neglect syndrome**. Patients with this syndrome have difficulty perceiving either a part of the body or a part of the visual field. Drawings by a patient with neglect syndrome may be seen in Figure 7.23. Oliver Sacks (1985) described a patient with neglect syndrome who believed that the hospital staff was playing a horrible joke on him by putting an amputated leg, which was actually his own very firmly attached leg, into his bed. While trying to remove the leg from his bed, the man frequently fell on the floor. Fortunately for Sacks's patient and others with neglect syndrome, the condition generally improves over time.

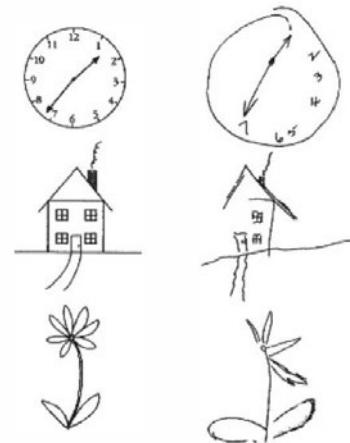
## Pain

**Pain** combines sensation with emotion and cognition. No other sensory modality is as dramatically affected by culture, emotion, context, and experience as our sense of pain. The connection between culture and the experience of pain is vividly illustrated by the hook-swinging ritual practiced in India (Melzack & Wall, 1983). This ritual, designed to promote the health of children and crops, involves hanging a male volunteer from steel hooks embedded into the skin and muscles of his back. Instead of suffering excruciating pain, the volunteers appear to be in a state of exaltation.

**A PURPOSE FOR PAIN** Given the anguish experienced by chronic pain patients, it would seem initially miraculous to be able to do away with this sensory modality altogether. Research on those born without effective pain reception suggests otherwise. Patients who perceive no pain frequently die prematurely, due to injuries and degeneration of joints and the spine (Cox et al., 2006). We need pain to remind us to stop when we are injured, to assess a situation before proceeding, and to allow the body time to heal. Pain is not a perfect warning system. In many potentially fatal conditions, such as in some types of cancer, pain does not surface until the condition is quite advanced. In other cases, the pain people experience far exceeds the threat to their safety. The pain associated with many headaches, stomachaches, and backaches occurs in the absence of any tissue damage.

**RECEPTORS FOR PAIN** Free nerve endings that respond to pain are called **nociceptors**. Nociceptors respond to a variety of stimuli associated with tissue damage. Some nociceptors respond most vigorously to mechanical injury such as the damage caused by a sharp object. The pressure of the mechanical stimulus on the nociceptor membrane opens mechanically gated ion channels, leading to the generation of action potentials. Other nociceptors respond to extreme temperature. Some nociceptors appear to respond to both mechanical stimulation and temperature.

A variety of chemicals can also activate nociceptors. Chemicals released when a cell is damaged, such as potassium ions, enzymes, histamine, and adenosine triphosphate (ATP), stimulate nociceptor activity. The soreness we experience after exercising vigorously is produced by a buildup of lactic acid. Lactic acid produces an increase in hydrogen ions in the extracellular fluid. These ions activate nociceptors, which in turn send unpleasant messages of soreness to the brain. An interesting class of nociceptors



**Figure 7.23 Drawings by a Patient with Neglect Syndrome** Damage to the secondary somatosensory cortex, particularly on the right hemisphere, results in neglect syndrome. Patients with this syndrome have difficulty perceiving a part of the body or a part of the visual field. Because the patient attempting to copy the model cannot perceive the entire visual field, he or she draws only part of each object. Patients seem to be blissfully unaware they have this problem until others point it out to them.

Source: From Springer, S. P., Deutsch, G., *Left Brain, Right Brain*. New York: W. H. Freeman, 1989, p. 193. Reprinted by permission.

**neglect syndrome** A condition resulting from secondary somatosensory cortex that produces difficulty perceiving a body part or part of the visual field.

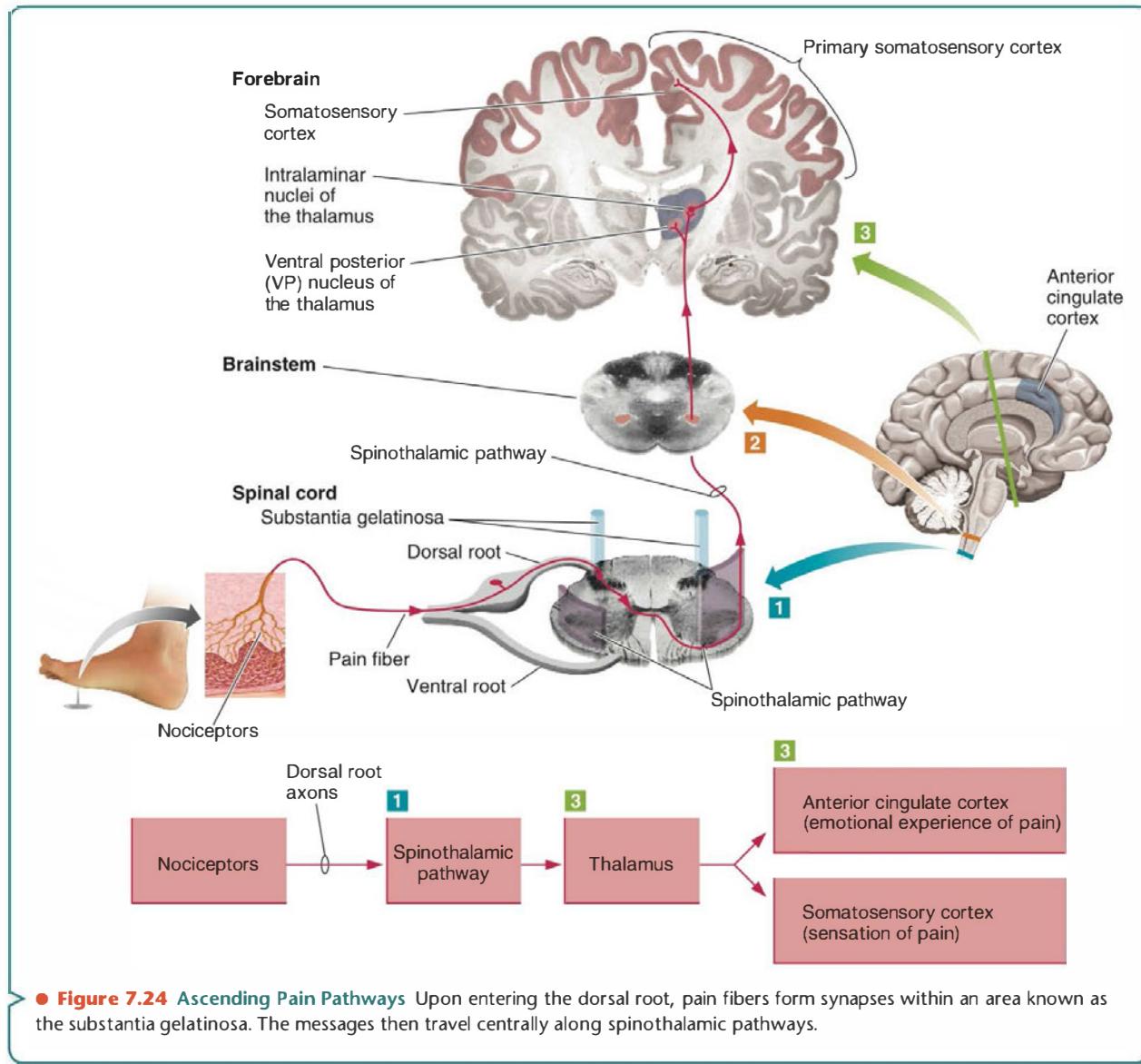
**pain** The sense that provides information about tissue damage.  
**nociceptor** A free nerve ending that responds to painful stimuli.

responds to chemicals known as vanilloids, a group that includes capsaicin (Caterina et al., 1997). Capsaicin is best known as the ingredient found in hot peppers, and it is responsible for the heat sensations we enjoy while eating spicy foods.

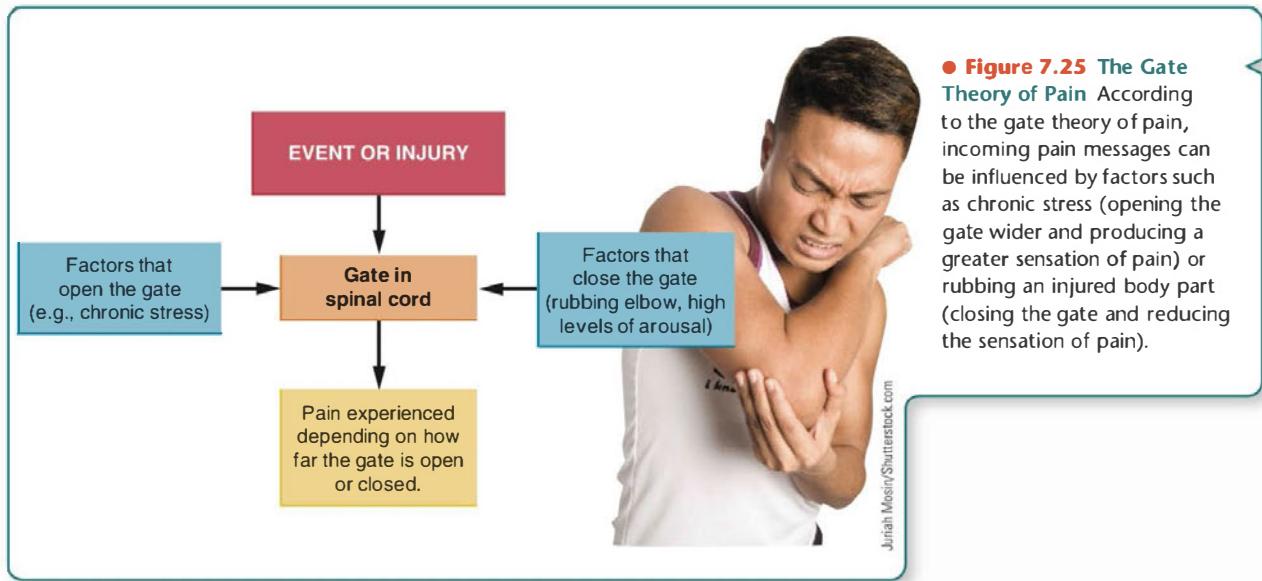
**PAIN PATHWAYS TO THE BRAIN** Information from the nociceptors is carried toward the CNS by two types of nerve fiber. The faster, myelinated A $\delta$  fibers are responsible for that quick, sharp “ouch.” The slower, unmyelinated C fibers are responsible for dull, aching types of pain sensation. Both types of ascending pain fibers use glutamate as their primary neurotransmitter (Jin, Nishioka, Wakabayashi, Fujita, & Yonehara, 2006).

As shown in Figure 7.24, pain fibers enter the spinal cord via the dorsal root. Once inside the cord, they synapse in the **substantia gelatinosa**, a group of cells in the outer gray matter of the dorsal horn. These cells also receive descending input from the brain. In addition to releasing glutamate, pain fibers are capable of releasing the neuropeptide **Substance P** in the dorsal horn of the spinal cord. Substance P stimulates changes in the dendrites of the cells in the substantia gelatinosa. These structural changes provide for adaptations to pain based on personal experience (Mantyh et al., 1997).

**substantia gelatinosa** A group of cells in the outer gray matter of the dorsal horn that receives input from pain fibers.  
**Substance P** A neurochemical associated with the sense of pain.



● **Figure 7.24 Ascending Pain Pathways** Upon entering the dorsal root, pain fibers form synapses within an area known as the substantia gelatinosa. The messages then travel centrally along spinothalamic pathways.



**Figure 7.25 The Gate Theory of Pain** According to the gate theory of pain, incoming pain messages can be influenced by factors such as chronic stress (opening the gate wider and producing a greater sensation of pain) or rubbing an injured body part (closing the gate and reducing the sensation of pain).

According to Melzack and Wall's (1967) influential **gate theory of pain**, a feedback loop in the dorsal horn determines which signals ultimately reach the brain (see Figure 7.25). Excitatory signals from gate cells open the gate, allowing the message to continue. Chronic stress can make the opening of the gate more likely, and many people are more acutely aware of pain when stressed. Inhibitory signals from gate cells can close the gate, stopping the pain message from proceeding to the brain. High levels of arousal during emergencies can close the gate, making people unaware of how badly they have actually been injured.

Fibers originating in the substantia gelatinosa immediately cross the midline of the spinal cord to join the **spinothalamic pathway** that runs the length of the ventral surface of the spinal cord. These fibers travel up through the brainstem and finally synapse within the thalamus. Pain information from the head and neck is transmitted to the thalamus in a similar manner. This information travels first along the trigeminal nerve, which synapses in the spinal trigeminal nucleus of the brainstem. Fibers from the spinal trigeminal nucleus form the **trigeminal lemniscus**, which terminates in the thalamus.

The spinothalamic and trigeminal lemniscus fibers synapse in one of two locations in the thalamus. Some fibers synapse in the VP nucleus, which also receives information from touch receptors. However, input regarding pain remains separate in the VP nucleus from input regarding touch. Other fibers connect with the **intralaminar nuclei** of the thalamus, shown in Figures 7.20 and 7.24. Both areas of the thalamus then form connections with the anterior cingulate cortex (ACC) and, to a lesser degree, the somatosensory cortex of the parietal lobe. The ACC participates in our anticipation and emotional responses to pain. If participants are told to expect a mild amount of pain from placing their hands in hot water, the ACC is less active than when they are told to expect more pain (Rainville, Duncan, Price, Carrier, & Bushnell, 1997). People who chose an immediate but large electric shock over a delayed but smaller shock, demonstrating that they "dreaded" the upcoming shock, showed increased activity in the ACC (Berns et al., 2006). Remembering pain and anticipating further pain, perhaps when facing a second similar surgery for the same condition, activates the prefrontal cortex. Activity in the prefrontal cortex is correlated with cognition and executive control related to pain.

**MANAGING PAIN** Although pain certainly has its purpose, we are also motivated to help those who face the challenges of chronic pain.

**gate theory of pain** An explanation of the effects of context on the perception of pain.

**spinothalamic pathway** A pathway carrying pain information from the substantia gelatinosa to the thalamus.

**trigeminal lemniscus** A pathway for pain information from the head and neck that connects the spinal trigeminal nucleus and the thalamus.

**intralaminar nuclei** Nuclei within the thalamus that receive pain information.

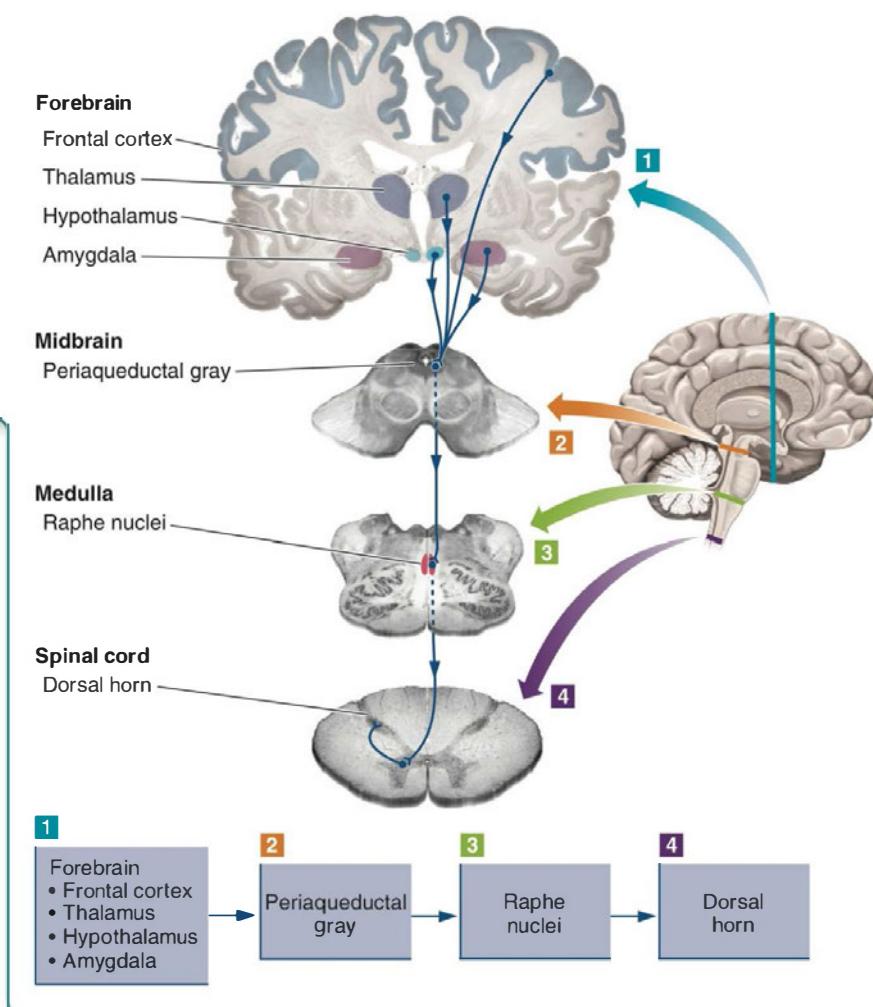
Although some individual differences in response to pain are due to culture and experience, a person's number of endogenous opioid receptors (see Chapter 4) also influences pain sensitivity (Zubieta et al., 2001). Researchers administered painful injections of saltwater into participants' facial muscles. Participants reported on their pain levels while undergoing PET scanning. Those with the highest amount of opioid activity as shown by the PET scans reported less pain.

In some cases, the pain signal can be modified by additional sensory input. Most of us spontaneously respond to bumping our elbow by rubbing it vigorously. Rubbing your elbow might actually lessen the ability of pain receptors to communicate with the brain. According to the gate theory discussed previously, input from touch fibers might compete with input from nociceptors for activation of cells in the substantia gelatinosa. Activation of the touch fibers effectively reduces the amount of pain information that can reach the brain.

In other cases, descending control from the brain has a dramatic influence on our perception of pain. As noted in Chapter 2, many higher-level brain structures form connections with the periaqueductal gray (PAG) of the midbrain, which also receives ascending pain signals (see Figure 7.26). The convergence of descending control and ascending pain information in the PAG provides an opportunity for the modification of incoming pain messages by higher-level cognitive processes. Electrical stimulation of the PAG generally produces a significant reduction in pain (Barbaro, 1988). In addition, the

**Figure 7.26 Descending Messages Influence Pain Perception**

**Perception** Many higher-level structures of the brain form connections with the periaqueductal gray (PAG) of the midbrain, a major locus for endogenous opioid activity. The PAG in turn forms connections with the raphe nuclei in the medulla and neurons in the spinal cord that can modulate incoming pain information.



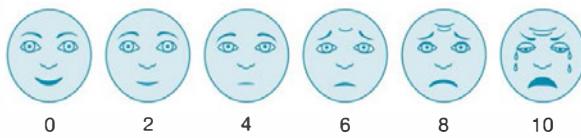
PAG contains large numbers of opioid receptors, which interact with opioids such as morphine. The pain relief achieved through the use of opioids probably occurs in large part due to the drugs' actions in the PAG.

Many people use profanity in response to pain, and this appears to lessen the subjective intensity of pain, at least for people who use profanity sparingly (Stephens & Umland, 2011). The exact mechanism for this effect is not known, but the ability of swearing to initiate sympathetic nervous system arousal might contribute to the reduced experience of pain. As mentioned previously, high levels of arousal associated with sympathetic activity are capable of reducing the perceived intensity of ascending pain signals.

One of the most troubling types of pain is chronic pain, which continues long after injuries have healed and affects nearly 10 percent of the U.S. population. Typical medications, such as aspirin and opioids, are relatively ineffective for managing this type of pain. Chronic pain is associated with increased activity in prefrontal areas, whereas physical pain (such as a burn) produces more activity in the thalamus (Millecamp et al., 2007). Researchers have suggested that chronic pain is more of a memory problem than a sensory problem, as if the brain has difficulty forgetting about the pain (Millecamp et al., 2007).

Being disabled by pain during an emergency is not in the best interests of survival, so it should come as no surprise that extreme stress often reduces the perception of pain. Stress impacts the pain system at several levels. As discussed previously, the high arousal that occurs in stressful situations might act to close pain gates in the substantia gelatinosa. In addition, stress might produce analgesia, or pain relief, by promoting the release of endorphins in the brain. In cases of more chronic stress, pain gates might open more than under normal circumstances, making people even more aware of existing problems.

Attitudes toward pain and experience with pain also play significant roles in our perceptions of the experience (see Figure 7.27). Athletes and nonathletes share similar pain thresholds, or levels of stimulation identified as painful. However, these groups are quite different in their tolerance of pain (Sternberg, Bailin, Grant, & Gracely, 1998). In particular, athletes in contact sports such as boxing, rugby, and football appear to tolerate higher levels of pain before identifying a stimulus as painful. A sense of control can reduce the need for pain medication. Patients who are allowed to self-administer morphine for pain actually require less medication than patients who receive injections from hospital staff (Viscusi & Schechter, 2006).



● **Figure 7.27 Individuals Experience Different Pain**

**Intensities** Health care providers often need to assess a person's subjective experience of pain, which can be very different from person to person. With adult patients, simply asking individuals to rate their pain on a scale of one to ten is often sufficient. With children, the Faces Pain Scale can be useful.

Source: Hicks, C. L., von Baeyer, C. L., Spafford, P. A., van Korlaar, I., & Goodenough, B. (2001). The Faces Pain Scale-Revised: toward a common metric in pediatric pain measurement. *Pain*, 93(2), 173-183.

## INTERIM SUMMARY 7.2

### Summary Table: Somatosensory Pathways

Sensory Modality	Receptor Types	Axon Types	Route to Somatosensory Cortex
Vestibular system	Hair cells within the saccule, utricle, and semicircular canals	Varied	<ul style="list-style-type: none"> <li>Fibers join the auditory nerve (cranial nerve VIII).</li> <li>Axons synapse in cerebellum and vestibular nuclei.</li> <li>Vestibular nuclei axons project to the spinal cord motor neurons and to the VP nucleus of the thalamus.</li> </ul>

(continued)

Sensory Modality	Receptor Types	Axon Types	Route to Somatosensory Cortex
			<ul style="list-style-type: none"> <li>Information travels from the VP nucleus to primary somatosensory and motor cortex.</li> </ul>
Touch	Mechanoreceptors	A $\beta$ fibers	<ul style="list-style-type: none"> <li>Fibers enter spinal cord via dorsal root.</li> <li>Axons join the dorsal column and synapse in the dorsal column nuclei of the medulla.</li> <li>Axons from the dorsal column nuclei form the medial lemniscus and synapse in the VP nucleus of the thalamus. Cranial neurons V, VII, IX, and X carry touch information from the head to brainstem nuclei, which form connections with the VP nucleus of the thalamus.</li> <li>The VP nucleus projects to primary somatosensory cortex in the parietal lobe.</li> </ul>
Pain	Nociceptors (free nerve endings)	A $\delta$ fibers and C fibers	<ul style="list-style-type: none"> <li>Fibers enter spinal cord via dorsal root.</li> <li>Fibers synapse in substantia gelatinosa.</li> <li>Pain information travels via the spinothalamic pathway and trigeminal lemniscus to the VP nucleus and intralaminar nuclei of the thalamus.</li> <li>Information is transmitted to the anterior cingulate cortex, primary somatosensory cortex, and prefrontal cortex.</li> </ul>

### Summary Points

1. The vestibular system provides information about the position and movement of the head. (LO4)
2. Major mechanoreceptors located within the skin include Meissner's corpuscles, Pacinian corpuscles, Merkel's disks, Ruffini's endings, and free nerve endings. (LO4)
3. Touch information travels along the dorsal column-medial lemniscal pathway to the dorsal column nuclei of the medulla, to the contralateral ventral posterior nucleus of the thalamus, and to the primary somatosensory cortex of the parietal lobe. (LO4)
4. Pain is sensed by free nerve endings called nociceptors. Ascending pain fibers synapse in the substantia gelatinosa, the thalamus, and the anterior cingulate and somatosensory cortices. Descending information regarding pain is transmitted to the periaqueductal gray (PAG). (LO4)

### Review Questions

1. How is the primary somatosensory cortex organized? How does experience change the organization of primary somatosensory cortex?
2. What factors modify an individual's perception of a painful stimulus?

## The Chemical Senses

Philosopher Immanuel Kant (1798) considered olfaction, or the sense of smell, to be the “most dispensable” sense. Nonetheless, our chemical senses, olfaction and gustation, do provide warning of danger, such as smelling smoke from a fire or the taste of spoiled food. Contrary to Kant’s view, people who have lost their sense of smell due to head injury often experience profound depression (Doty et al., 1997).

### Olfaction

**Olfaction** begins with the detection of molecules suspended in the air. In addition to having the capacity to be suspended in air, olfactory stimuli must be small and water-repellant. We cannot sense every type of molecule suspended in the air. Natural gas, for example, has no detectable odor, so gas companies add an odorant so that we can sense potentially dangerous gas leaks in our homes.

Air containing olfactory stimuli is taken in through the nostrils and circulated within the nasal cavities connected to the nostrils. The congestion you experience during a bad cold limits this circulation, reducing your ability to smell.

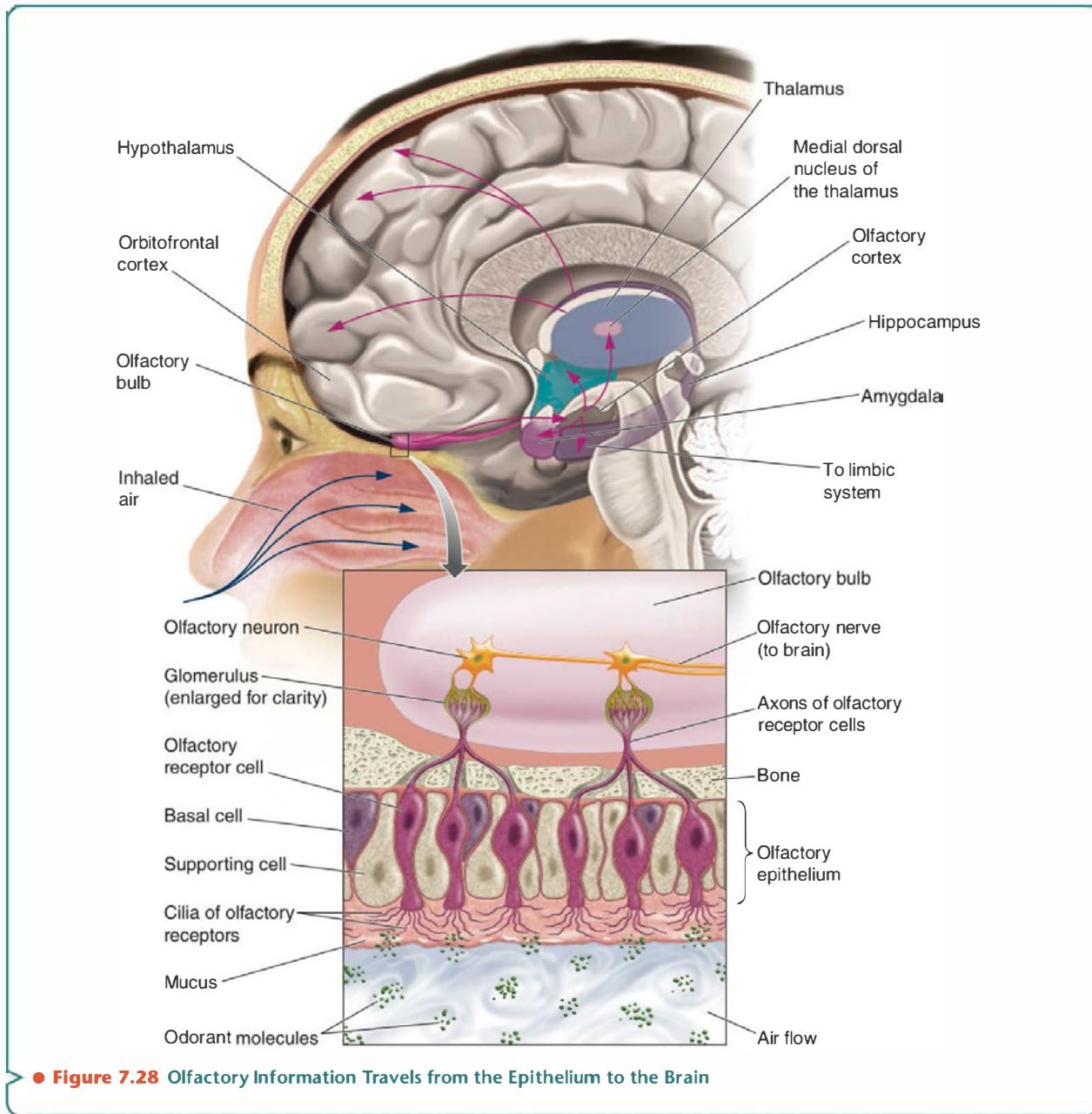
Individuals vary in their sensitivity to smell. As we age, our sensitivity to smell decreases (Rawson, 2006). Females are generally more sensitive to smell than males (Brand & Millot, 2001), and smokers are less sensitive to smell than nonsmokers (Katotomichelakis et al., 2007). Women taking oral contraceptives are less sensitive to odors, particularly those associated with social and sexual behavior such as musk, than naturally cycling women (Renfro & Hoffmann, 2013). Our ability to perceive a particular odor is also affected by how long we are exposed to the odor. Smell adapts rapidly, a fact to keep in mind when applying your favorite perfume.

**OLFACTORY RECEPTORS** The neural receptors for olfaction are contained in a thin sheet of cells within the nasal cavity known as the **olfactory epithelium**, illustrated in Figure 7.28. Unlike most other types of neurons, olfactory receptor cells regularly die and are replaced in a cycle lasting approximately four to six weeks. In addition to the receptor cells, the olfactory epithelium also contains glia-type support cells that produce mucus. Basal cells in the epithelium give rise to new receptors when needed.

The approximately 10,000 olfactory receptor cells in each nostril are bipolar, having two branches extending from the cell body. One branch reaches out to the surface of the epithelium. Cilia, or hairlike structures, extend from the end of this branch into the mucus that covers the epithelium. Molecules dissolved in the mucus bind and interact with these cilia. The binding of an odorant molecule to a receptor site on a cilium begins a process that results in an influx of sodium and calcium into the receptor neuron. If the resulting depolarization is large enough, it will produce action potentials sent along the branch of the receptor neuron that projects toward the olfactory bulb. These fibers collectively form the olfactory nerve (cranial nerve I), which makes its way centrally to the olfactory bulb.

Olfactory receptors must catalog the many thousands of different smells that we are able to discriminate. Buck and Axel (1991) suggested that mammals can have approximately 1,000 types of receptor cells to accomplish this task, although human beings have only about 350 to 400 (Spehr & Munger, 2009). How do such a small number of receptors encode information about a wide array of odorants? A shape-pattern theory suggests that odorant molecules interact with receptors like keys fitting into locks. A modification of this approach suggests that odorants can activate specific combinations of receptors. Overall patterns of receptor activity, as opposed to the response of a single type, could distinguish among many separate odorants. Other researchers believe that the olfactory system responds to the vibration of odorant molecules rather than to their shapes (Brookes, Hartoutsiou, Horsfield, & Stoneham, 2007; Turin, 2002).

**olfaction** The sense of smell.  
**olfactory epithelium** The nasal cavity area containing olfactory receptors.



• **Figure 7.28** Olfactory Information Travels from the Epithelium to the Brain

**OLFACTORY PATHWAYS** The axons from the olfactory receptor cells make their way to one of our two olfactory bulbs via the olfactory nerve (cranial nerve I).

As these fibers proceed toward the brain, they are quite vulnerable to damage, particularly from traffic accidents. As the head is jerked back and forth due to impact, the olfactory fibers leaving the nose can be sheared by the nearby edges of the skull bones (think of the nasal “triangle” of the skeleton). People typically respond to the resulting loss of their sense of smell by developing symptoms of depression (Deems et al., 1991) and often resort to flavoring their food with pepper sauce to give it some noticeable flavor. As we will see in our discussion of taste, the flavor of a food results from a combination of taste and olfaction, with olfaction playing a leading role.

Receptor axons synapse within olfactory bulb structures known as **glomeruli**. In each glomerulus, approximately 25,000 olfactory receptor axons form synapses on about 100 olfactory neurons. Each glomerulus receives information from only one type of receptor cell. In this manner, precise information about the odorant stimulus is transmitted to the olfactory bulbs. It is likely that the bulbs also participate in some initial sorting of odorant categories, but further work is necessary before an odorant can actually be identified. The processing of components making up complex odors remains separate in the olfactory bulb, suggesting that the integration of these components and recognition of odors (the perfume of a rose, for example) occurs at even higher levels of processing in the brain (Lin, Shea, & Katz, 2006). The olfactory bulbs, along with the hippocampus, are a major destination for cells produced in adult neurogenesis (see Chapter 5). Cells originating in the ventricular zone of the lateral ventricles migrate forward to the olfactory bulbs, regularly providing a fresh supply of cells for this structure (Lazarini et al., 2014).

A long tradition, originating with nineteenth-century studies conducted by Paul Broca (see Chapter 13), claims that human olfactory abilities are not very good compared to other animals. This view has been challenged by contemporary comparisons that show that human olfactory bulbs have about an average number of neurons compared to 24 other mammalian species (McGann, 2017). Humans have rather large olfactory bulbs compared to mice and rats, which are often assumed to have superior olfactory abilities. However, differences in olfactory processing in the brain might also affect olfactory abilities. To make room in the brain for processing the amount of visual input available to primates, other systems might have been given a lower priority (Gilad, Wiebe, Przeworski, Lancet, & Pääbo, 2004).

Olfaction is unique among the major senses in that information travels to the cerebral cortex without synapsing in the thalamus first. Axons from the olfactory bulbs form the **olfactory tracts**. Axons from the olfactory tracts synapse in the **olfactory cortex**. The olfactory cortex is located at the base of the frontal lobe extending onto the medial surface of the temporal lobe. The olfactory cortex forms connections with the **medial dorsal nucleus** of the thalamus, which in turn projects to the insula and the orbitofrontal cortex (Shipley & Reyes, 1991). The orbitofrontal cortex might participate in the identification of the pleasant or unpleasant qualities of olfactory stimuli. The olfactory cortex forms connections with many subcortical structures, including the amygdala and hypothalamus.

Olfactory information is widely distributed in the brain and is used by systems involved with odor identification, motivation, emotion, and memory. Olfaction is disturbed by a number of types of psychopathology. People with schizophrenia show deficits in olfaction, possibly due to general problems with frontal lobe functioning (see Chapter 16). Their immediate family members also show deficits in olfaction relative to healthy controls, suggesting a genetic basis for differences in olfactory function (Compton et al., 2006). Patients with major depressive disorder, anorexia nervosa, and alcoholism show specific patterns of olfactory deficits, illustrating the complex relationships between olfaction and emotion (Lombion-Pouthier, Vandel, Nezelof, Haffen, & Millot, 2006).

Olfaction typically becomes less sensitive with age, but more serious dysfunction in olfaction can predict the development of REM sleep behavior disorder (see Chapter 11), Parkinson's disease, and Lewy body disease by up to seven years prior to the emergence of other symptoms (Driver-Dunckley et al., 2014). Different patterns of olfactory deficits characterize Alzheimer's disease, which potentially might allow olfactory function to be useful in diagnosing this condition at an early stage (Golden, et al., 2016).

## Gustation

The most likely original purpose of our sense of **gustation**, or taste, was to protect us from eating poisonous or spoiled food. Many bitter-tasting substances are actually poisonous, and we are attracted to tastes that boost our chances of survival. Gustation

**glomeruli** Structures within the olfactory bulb where olfactory receptor axons form synapses with olfactory neurons.

**olfactory tract** A fiber pathway connecting the olfactory bulbs to the olfactory cortex.

**olfactory cortex** Cortex in the frontal and temporal lobes that responds to the sense of smell.

**medial dorsal nucleus** The area of the thalamus that processes olfactory information.

**gustation** The sense of taste.

is a small part of the eating experience. When we eat, we perceive not only the taste of the food but also qualities such as temperature, texture, and consistency (Gibson, 1966). In addition, gustation interacts with olfaction to give us the flavor of a food. You have probably noticed that food just doesn't taste very good when your sense of smell is decreased by a bad cold. If you close your eyes and hold your nose, you are unable to distinguish between a slice of apple and a slice of raw potato.

Gustation begins with the dissolving of molecules in the saliva of the mouth. Saliva is similar in chemical composition to saltwater. Substances that do not dissolve in saliva cannot be tasted, although you can still obtain information about the size, texture, and temperature of objects in the mouth. Most of us are familiar with four major categories of taste: sweet, sour, salty, and bitter. However, you may not have heard of a fifth proposed type of taste, known by the Japanese term **umami**, which, roughly translated, means savory or meaty. Umami tastes are associated with proteins, especially glutamate. In addition, some taste receptors respond to free fatty acids, enabling us to detect the fats in food (Gilbertson, Fontenot, Liu, Zhang, & Monroe, 1997). Still other receptors alert us to the presence of carbohydrates (Turner, Byblow, Stinear, & Gant, 2014).

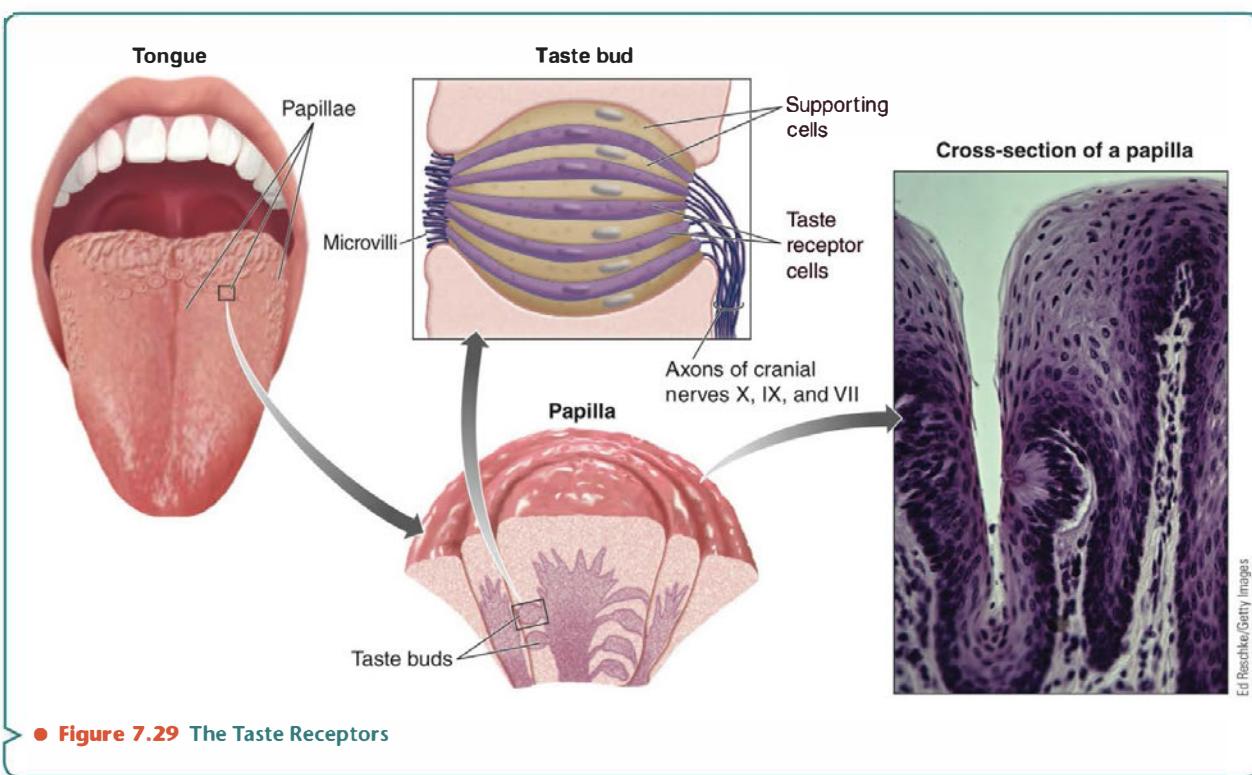
**GUSTATORY RECEPTORS** Receptors for taste are found not only on the tongue but also in other parts of the mouth and even in the gut (Egan & Margolskee, 2008). For our present purposes, we'll confine our discussion to the receptors of the tongue, shown in Figure 7.29.

Each bump on the surface of the tongue, known as a **papilla**, contains somewhere between 1 and 100 **taste buds**, which are too small to be seen with the naked eye (Nelson, 1998). Some papillae, such as those in the very center of the tongue, contain no taste buds at all. Taste buds live for only about 10 days, after which time they are replaced. Burning your tongue on hot liquid reduces your sense of taste initially, but recovery occurs within a few days. In total, the average person has about 6,000 taste buds, although significant variation can occur from one person to the next (Miller & Reedy, 1990). Linda Bartoshuk (2000) has identified people with unusually

**umami** A proposed taste category associated with the presence of proteins.

**papilla** Bumps on the tongue that contain taste buds.

**taste buds** Structures that contain taste receptors.



• Figure 7.29 The Taste Receptors

high numbers of taste buds, whom she has named “supertasters.” Not only do these individuals experience taste with greater intensity, giving them what Bartoshuk refers to as a “neon” sense of taste compared to the “pastels” experienced by others, but they also appear to be more sensitive to oral pain and to the texture of foods. If you’re wondering whether you are a supertaster, here are a few clues. People who dislike bitter foods (dark chocolate, black coffee, broccoli) and citrus are more likely to be supertasters. You can observe your tongue after painting it with blue food coloring to show the density of papillae, which are more numerous in supertasters (see ● Figure 7.30).

Most taste buds contain somewhere between 50 and 150 taste receptor cells. The taste receptor cells are not technically considered to be neurons, although they do have the capability of forming synapses. Each taste receptor has a number of thin fibers known as microvilli that extend into the saliva. Substances interact with the microvilli in different ways. Some molecules, such as sodium, pass through the cell’s ion channels. Others bind to ion channels, either blocking them or opening them. Finally, some taste molecules bind to receptors on the taste cell and activate second messenger systems (see Chapter 4). The tongue also contains pain receptors that are sensitive to capsaicin, the main “hot” ingredient in peppers. Mice lacking capsaicin receptors happily consumed water containing capsaicin at levels that were rejected by normal mice (Caterina et al., 1997).

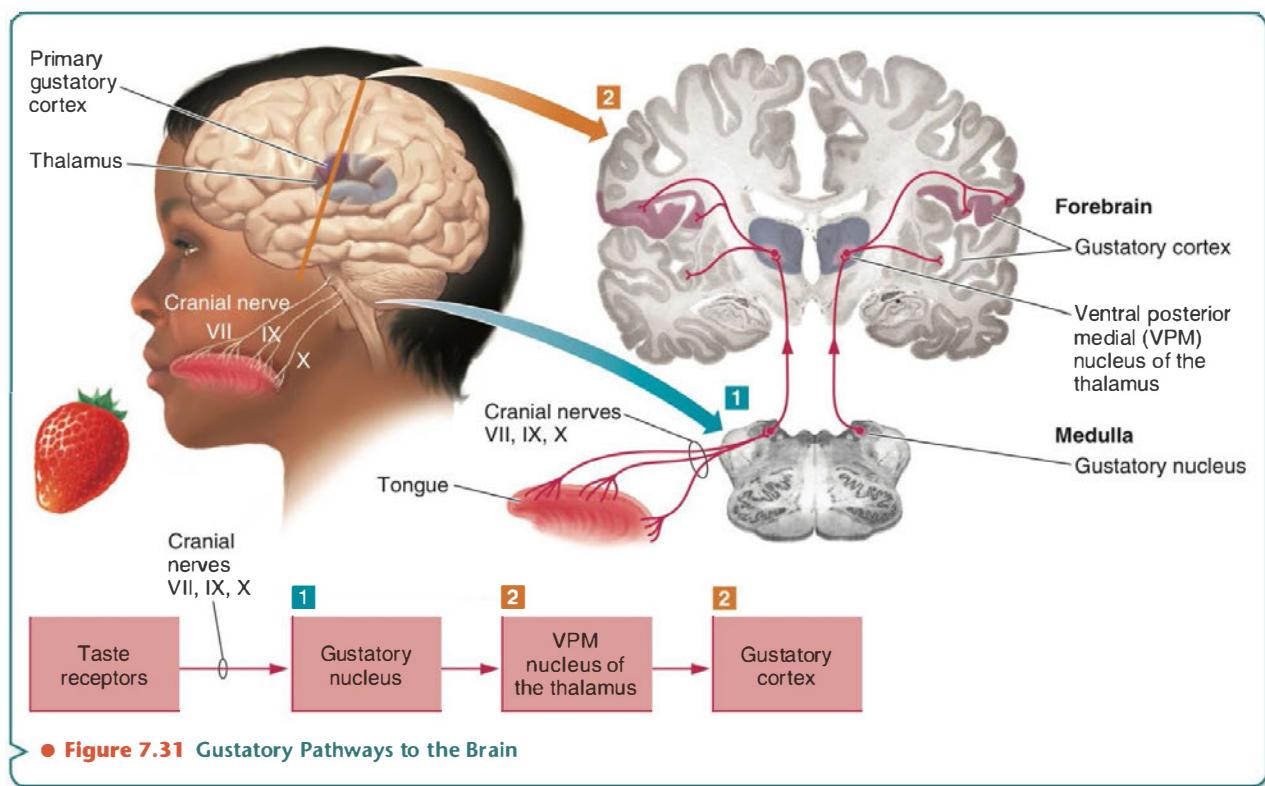
For many years, researchers believed that receptors for different taste qualities were located in different parts of the tongue. You probably have seen little maps showing where each taste was supposedly sensed. More recent research demonstrates that this earlier view is incorrect. Receptors for all taste qualities are found in all areas of the tongue (Huang et al., 2006).

**GUSTATORY PATHWAYS** Pathways linking the taste receptors to the brain are shown in ● Figure 7.31. Although taste receptors do not have a true axon, they are



Roger Fréberg

● **Figure 7.30 Supertasters Have More Papillae** Using blue food coloring, you can color your tongue to make your papillae stand out. The presence of more papillae than normal suggests a person is a supertaster.



**gustatory nucleus** A location within the solitary nucleus that receives gustatory input from cranial nerves VII, IX, and X.

**ventral posterior medial (VPM) nucleus of the thalamus** The nucleus of the thalamus that receives information regarding taste.

**gustatory cortex** Area at the junction of the frontal lobe, parietal lobe, and insula that processes gustatory information.

**synesthesia** Experience in one sensory pathway elicits an automatic activation in another sensory pathway.

able to contact and influence taste fibers serving the tongue. These taste fibers form parts of cranial nerves VII, IX, and X. These nerves in turn form synapses with the **gustatory nucleus**, which is a part of the solitary nucleus of the medulla. Axons from the gustatory nucleus synapse in the **ventral posterior medial (VPM) nucleus of the thalamus**. Finally, axons from the VPM nucleus synapse in the **gustatory cortex** at the junction of the frontal and parietal lobes, extending into the anterior insula (de Araujo & Simon, 2009). The recognition of a type of taste is probably determined at this level. Other fibers make their way to the orbitofrontal cortex. As in the case of olfaction, this input probably encodes the pleasantness, or emotional qualities, of taste.

## Synaesthesia

A little more than 4 percent of the human population regularly experiences **synesthesia**, or cross-modal perceptions such as hearing smells or seeing tastes.

A dramatic case of synesthesia was described by the Russian neuroscientist Alexander Luria (1968). Luria's patient, known as S, not only experienced synesthesia but also possessed a remarkable memory due to the distinctiveness of his sensory experiences. S described a tone as "a brown strip against a dark background that had red, tongue-like edges. The sense of taste he experienced in response to

### Behavioral Neuroscience GOES TO WORK

#### WHAT IS A PERFUMER?

A quick tour of your home will reveal a large number of products containing scent, from your shampoo to your laundry detergent. Who designs these scents, and what is their method?

A perfumer, often called "a nose," is responsible for creating fragrances to be used not just in perfume but in many other household products as well. For many years, perfumers learned their craft as apprentices, but professional training schools have emerged in the past few decades. Entrance to these schools usually requires a background in chemistry. Training to become a perfumer begins with memorization of hundreds of both natural and synthetic materials, followed by learning how to combine "notes." ●Figure 7.32 shows a "nose" at work in front of his array of scents, known as an "organ." After further training, the perfumer will be tasked with creating new fragrances. Because of the complexity involved in the training, perfumers usually specialize in fine fragrances (perfumes), personal care fragrances, and household fragrances.

Do perfumers have a naturally superior sense of smell? According to brain imaging studies of student and expert perfumers, the answer to this question is



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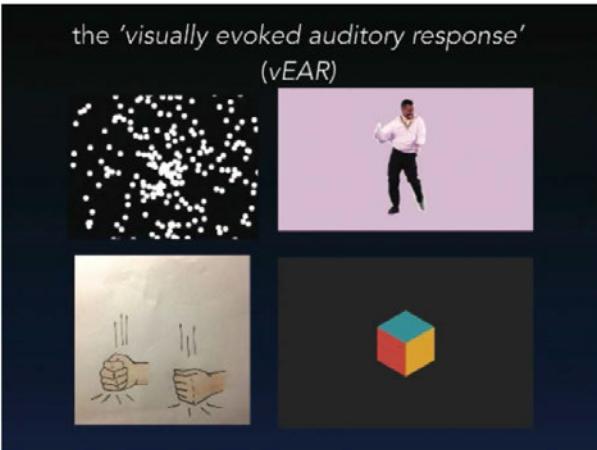
● **Figure 7.32 A Perfumer at Work** A perfumer, or "nose," works at his organ. Perfumers must develop the ability to remember large numbers of scents.

probably "no" (Plailly, Delon-Martin, & Royet, 2012). Instead, the extensive training and experience of the expert perfumers appears to enhance their ability to generate olfactory mental images. Changes in the primary olfactory cortex, orbitofrontal cortex, and hippocampus are associated with improved memory for odors.

this tone was like that of sweet and sour borscht, a sensation that gripped his entire tongue" (Luria, 1968, p. 23).

Some synesthesia experiences appear to be surprisingly common. Twenty-two percent of participants appeared to "hear" motion they observed visually (Fassnidge, Cecconi Marcotti, & Freeman, 2017). In other words, if you watch a fan moving, your brain might conjure up a whirring sound (see Figure 7.33). The participants who "heard" motion performed better on a task that required them to identify sequences in a series of flashing lights, possibly because the sound provided a rhythm they could track.

The causes of synesthesia are not completely understood (Meier & Rothen, 2015). Some cases may be genetic, reflecting different connectivity patterns during brain development. In other cases, synesthesia is acquired, either by training, posthypnotic suggestion, drug exposure, or brain injury. It is likely that multiple causal pathways underlie this phenomenon in different individuals.



**Figure 7.33 Hearing Movement** About one in five participants reported "hearing" movement while viewing stimuli like these. Musicians were particularly likely to show this type of synesthesia.

StudyShare Inc.

### INTERIM SUMMARY 7.3

#### Summary Table: Major Features of the Chemical Sensory Systems

Sensory Modality	Receptor Types	Pathways and Connections
Olfaction	Bipolar cells embedded in the olfactory epithelium	<ul style="list-style-type: none"> <li>Axons from the receptors synapse in the glomeruli of the olfactory bulbs.</li> <li>Axons from the olfactory bulbs form the olfactory tract and synapse in the olfactory cortex.</li> <li>The olfactory cortex sends information to the thalamus, limbic system, insula, and orbitofrontal cortex.</li> </ul>
Gustation	Taste buds on the tongue and elsewhere in the mouth	<ul style="list-style-type: none"> <li>Fibers serving the taste receptors join cranial nerves VII, IX, and X.</li> <li>These axons synapse in the gustatory nucleus of the medulla.</li> <li>Axons from the gustatory nucleus synapse in the ventral posterior medial (VPM) nucleus of the thalamus.</li> <li>VPM axons synapse in the gustatory cortex.</li> </ul>

#### Summary Points

1. Olfactory receptors lining the olfactory epithelium of the nasal cavity respond to airborne molecules by sending messages via the olfactory nerve to the olfactory bulb. The olfactory bulb axons project to the olfactory cortex, which forms widely distributed connections with the cerebral cortex and the limbic system. (LO5)

2. Interactions between taste receptors and dissolved chemicals activate parts of cranial nerves VII, IX, and X, which synapse with the gustatory nucleus of the medulla. Gustatory nucleus axons synapse in the ventral posterior medial nucleus of the thalamus, which in turn projects to the gustatory cortex and to the orbitofrontal cortex. **(LO5)**
3. Some individuals have cross-modal sensory experiences, such as "hearing" movement. **(LO6)**

### Review Questions

1. What are the major features of olfactory receptors?
  2. What are the major types of taste stimuli?
- 

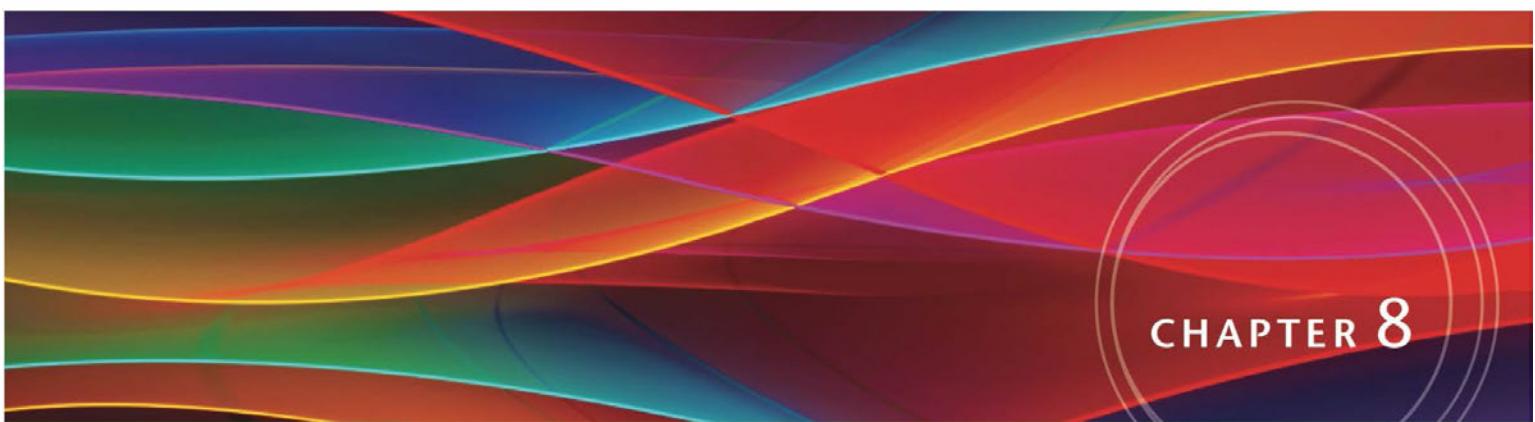
## Chapter Review

### THOUGHT QUESTIONS

1. If you had to give up one of your senses, which one would it be, and why?
2. What steps can you take to reduce environmental causes of hearing loss?
3. Why does the representation of pain information in the CNS make it difficult to treat chronic pain with surgery?
4. How might the gradual loss of olfactory sensitivity affect the eating habits of seniors?

### KEY TERMS

amplitude (p. 227)	Merkel's disks (p. 244)	Ruffini's endings (p. 244)
audition (p. 226)	nociceptor (p. 251)	saccule (p. 241)
auditory nerve (cranial nerve VIII) (p. 231)	olfaction (p. 257)	semicircular canals (p. 241)
basilar membrane (p. 231)	olfactory cortex (p. 259)	somatosensory system (p. 241)
cochlea (p. 230)	olfactory tracts (p. 259)	spiral ganglion neurons (p. 232)
decibel (dB) (p. 228)	organ of Corti (p. 231)	substantia gelatinosa (p. 252)
gate theory of pain (p. 253)	ossicles (p. 230)	synesthesia (p. 262)
glomerulus/glomeruli (p. 259)	otolith organs (p. 241)	taste buds (p. 260)
gustation (p. 259)	otoliths (p. 241)	temporal theory (p. 235)
gustatory cortex (p. 262)	Pacinian corpuscles (p. 244)	tonotopic organization (p. 235)
hertz (Hz) (p. 228)	pain (p. 251)	umami (p. 260)
intralaminar nuclei (p. 253)	papilla (p. 260)	utricle (p. 241)
mechanoreceptors (p. 244)	pinna (p. 229)	ventral posterior medial (VPM) nucleus (p. 262)
medial dorsal nucleus (p. 259)	place theory (p. 235)	ventral posterior (VP) nucleus (p. 243)
medial geniculate nucleus (p. 233)	primary auditory cortex (p. 234)	vestibular system (p. 241)
Meissner's corpuscles (p. 244)	primary somatosensory cortex (p. 243)	



# CHAPTER 8

# Movement

## LEARNING OBJECTIVES

- L01** Describe the physical structure of muscle fibers and explain the process of muscle fiber contraction.
- L02** Explain the processes responsible for the initiation and control of muscle contractions.
- L03** Differentiate among the structures and processes responsible for providing feedback from muscles to the central nervous system.
- L04** Describe the major motor reflexes.
- L05** Explain the initiation and control of movement and the ability to understand the movement of others.
- L06** Identify the causes, symptoms, and treatments of major disorders of movement.

## CHAPTER OUTLINE

- Muscles**
  - Types of Muscles
  - Muscle Anatomy and Contraction
  - The Effects of Exercise on Muscle
  - The Effects of Aging on Muscles
- Neural Control of Muscles**
  - Alpha Motor Neurons
  - The Motor Unit
  - The Control of Muscle Contractions
  - The Control of Spinal Motor Neurons
- Interim Summary 8.1**
- Reflex Control of Movement**
  - Reciprocal Inhibition at Joints
  - The Flexor Reflex
  - Spinal Reflexes Related to Walking
- Interim Summary 8.2**
- Disorders of Movement**
  - Toxins
  - Myasthenia Gravis
  - Muscular Dystrophy
  - Polio
  - Accidental Spinal Cord Injury (SCI)
  - Amyotrophic Lateral Sclerosis (ALS; Lou Gehrig's Disease)
  - Parkinson's Disease
  - Huntington's Disease
- Interim Summary 8.3**
- Chapter Review**

**THINKING ETHICALLY:** Gene Doping for Strength

**CONNECTING TO RESEARCH:** Mirror Neurons

**BUILDING BETTER HEALTH:** When Vaccination Is Not Enough

**BEHAVIORAL NEUROSCIENCE GOES TO WORK:** Physical Therapy

## Muscles

Muscles make up the majority of the human body's tissues and are responsible for the movement of the body and the movement of materials within the body.

**smooth muscle** A type of muscle found in the lining of the digestive tract, within arteries, and in the reproductive system; controlled by the autonomic nervous system.

**striated muscle** A type of muscle named for its striped appearance; including cardiac and skeletal muscles.

**cardiac muscle** A type of striated muscle found in the heart.

**skeletal muscle** A type of striated muscle that is attached to bones and is responsible for the majority of body movements.

### Types of Muscles

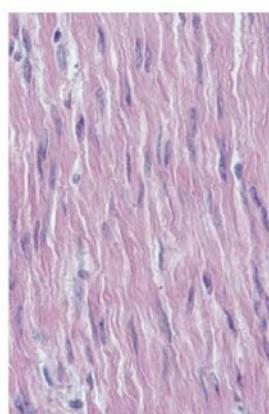
The muscles of the body, shown in Figure 8.1, can be divided into two types based on their appearance: smooth muscle and striated muscle. **Smooth muscle** is found in the lining of the digestive tract, within the arteries, and in the reproductive system. Smooth muscles move nutrients through the digestive tract, control blood pressure, and mix sperm with seminal fluid, among other tasks. The smooth muscles are controlled by the autonomic nervous system (see Chapter 2).

**Striated muscle**, named after its striped appearance, can be further divided into two types, cardiac muscle and skeletal muscle. **Cardiac muscle** produces the pumping action of the heart. The **skeletal muscles** attached to bones produce the majority of body movement. Other skeletal muscles are responsible for moving our eyes and lungs. The human body contains somewhere between 640 and 850 skeletal muscles,

● **Figure 8.1** The Human Body Has Three Types of Muscle

**Muscle types** are named after their appearance or location. (a) Smooth muscle is found in the digestive tract, blood vessels, and reproductive system. (b) Striated (striped) muscle gets its name from its striped appearance. There are two types of striated muscle. Cardiac muscle keeps our hearts beating. Skeletal muscles move our bones, eyes, and lungs.

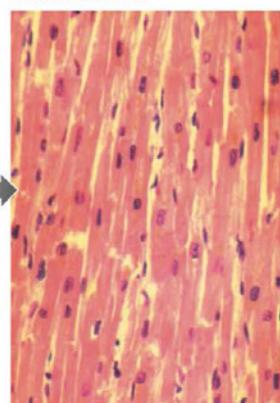
Intestinal smooth muscle



TinyDevil/Shutterstock.com

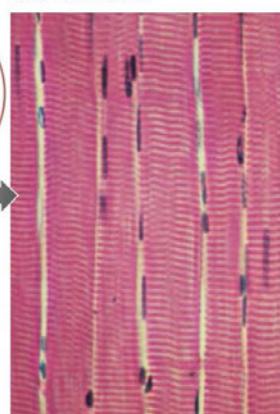
(a) Smooth Muscle

Cardiac muscle



© Lester V. Bergman/Corbis

Skeletal muscle



Science History Images/Alamy Stock Photo

(b) Striated Muscle

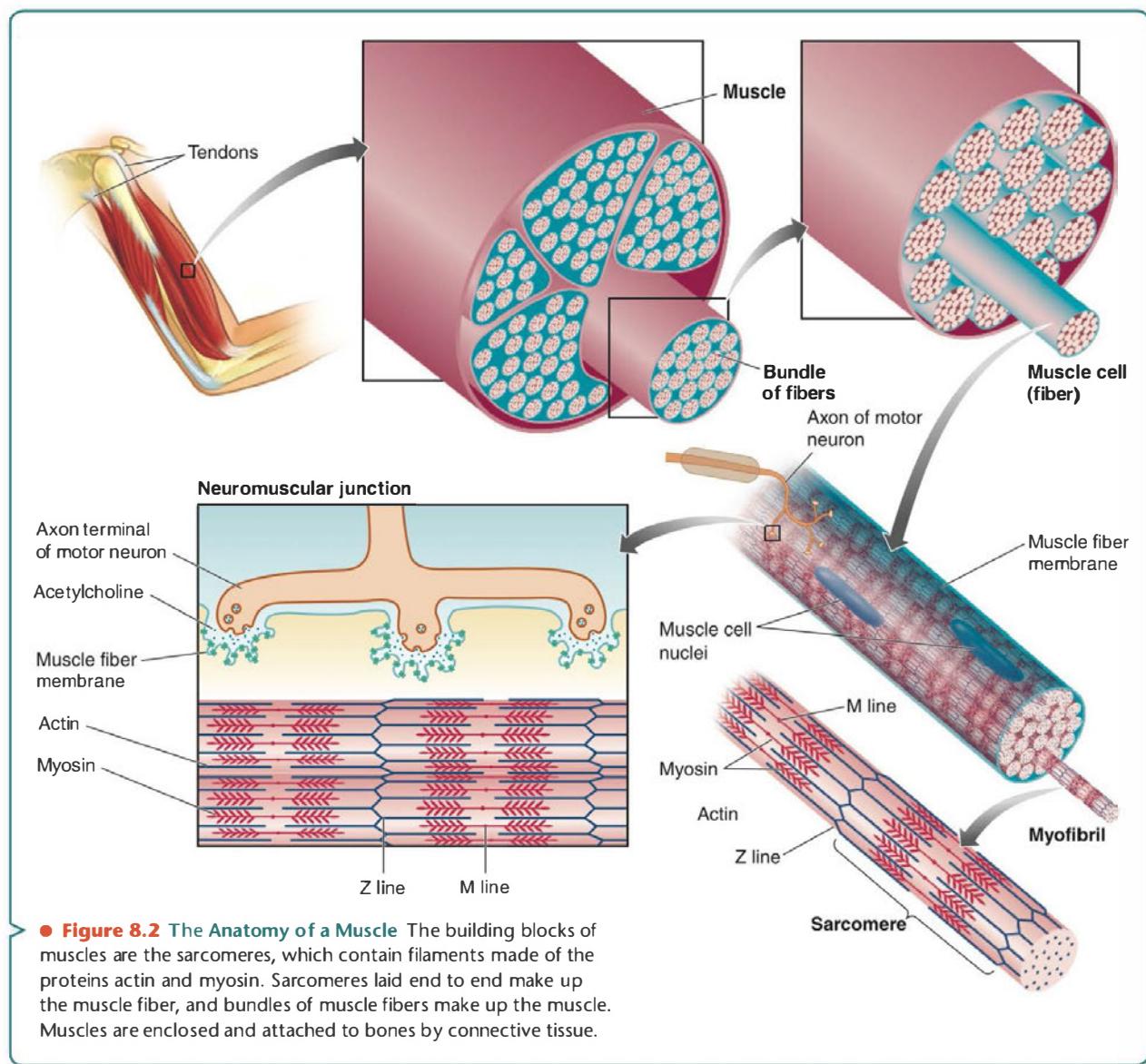
depending on which anatomist you ask. Our discussion of movement will focus on the skeletal muscles because these are responsible for the majority of our behavior.

## Muscle Anatomy and Contraction

Skeletal muscles, illustrated in ● Figure 8.2, are made up of long, very thin cells referred to as **muscle fibers**. Human muscle fiber cells usually extend the length of the muscle and are up to 30 cm (11 in) long and from 0.05 mm (0.002 in) to 0.15 mm (.006 in) wide.

**THE MUSCLE FIBER** The membranes encasing each muscle fiber are similar to the membranes of neurons. Like neural membranes, the muscle fiber membrane contains receptor sites, in this case for the neurotransmitter acetylcholine (ACh). When a molecule of ACh binds to a receptor site in the muscle fiber membrane, sodium channels open. Sodium rushing into the muscle fiber depolarizes the cell and triggers an action potential. In Chapter 3, we observed that action potentials in neurons travel

**muscle fiber** An individual muscle cell.



**twitch** The contraction of a single muscle fiber.

**myofibril** A long fiber strand running the length of a muscle fiber that is responsible for contraction.

**sarcomere** A myofibril segment bound on either side by a Z line and spanned by thin filaments.

**Z line** A boundary line for each sarcomere within a myofibril.

**actin** A protein that makes up the thin filaments of the myofibril.

**myosin** A protein that makes up the thick filaments of the myofibril.

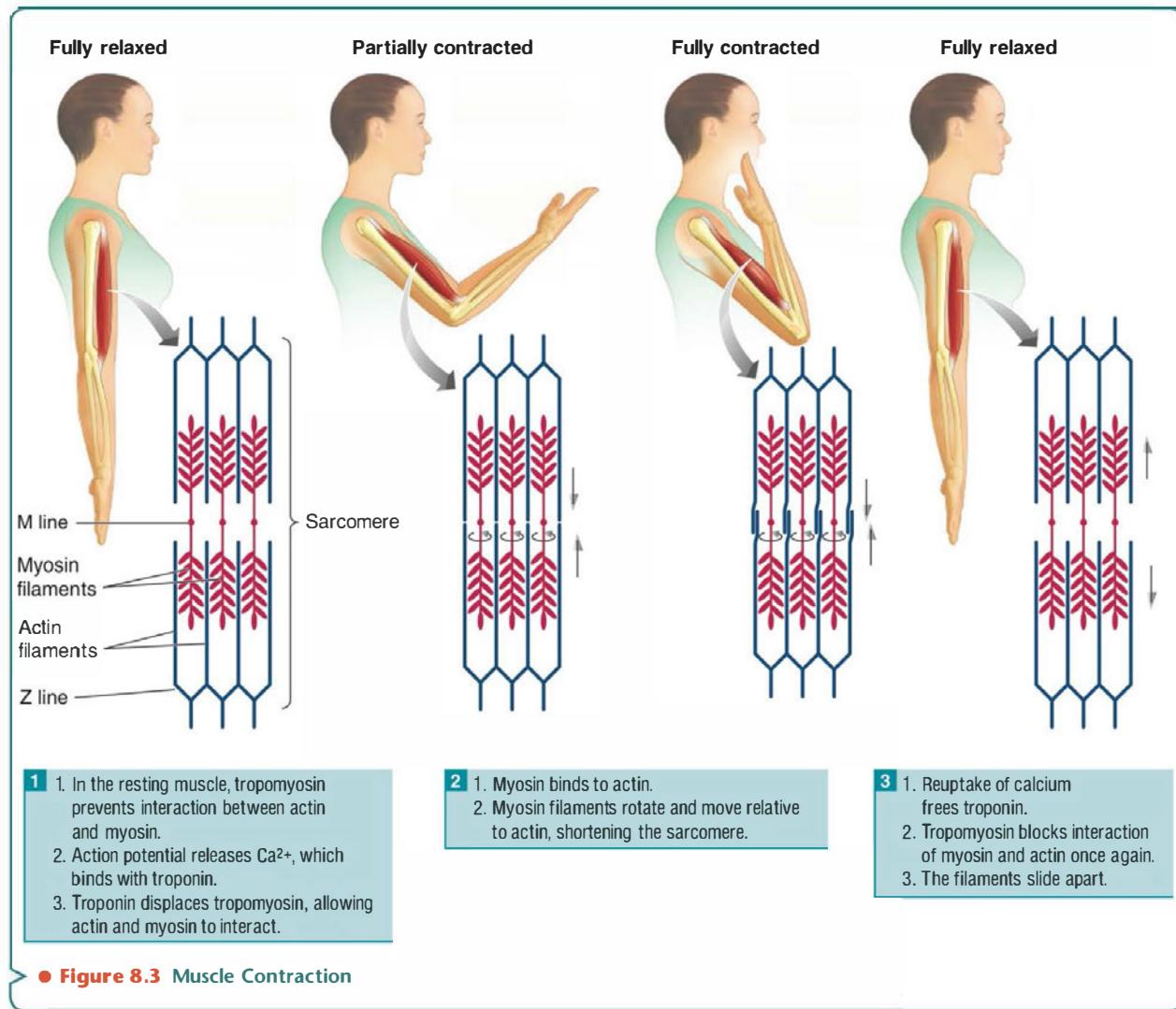
**M line** The middle of the sarcomere where myosin fibers are anchored.

in only one direction—from the axon hillock down the length of the axon. In contrast, the action potential in a muscle fiber spreads out in two directions on either side of the receptor site, which is located in the middle of the fiber. Each action potential produces a single contraction of the muscle fiber, known as a **twitch**.

**THE STRUCTURE OF MYOFIBRILS** The interior of the muscle fiber is made up of long strands of protein called **myofibrils**. The myofibrils run the length of the fiber and are responsible for producing fiber contractions.

Single segments of a myofibril are called **sarcomeres**, which are arranged end to end. The boundary of each sarcomere is known as a **Z line** because the boundary looks like a Z shape under a microscope. Attached to each Z line are a number of thin filaments made up of the protein **actin**. Lying between each pair of thin filaments is a thick filament made up of the protein **myosin**. Myosin filaments are anchored at the middle of the sarcomere, known as the **M line**.

**MUSCLE FIBER CONTRACTION** Muscle contractions are caused by the movement of the thick myosin filaments along the length of the thin actin filaments. As the filaments slide by each other, the Z lines move closer together, and the sarcomere shortens. As the sarcomeres shorten, the muscle contracts. This process is illustrated in Figure 8.3.



In a resting muscle fiber, actin binding sites are covered by the protein **tropomyosin**, which prevents actin from interacting with myosin. The arrival of an action potential at the muscle fiber is the catalyst for a series of events that allows actin and myosin to interact. The action potential triggers the release of calcium from internal organelles within the muscle fiber. Calcium in turn binds with another protein, **troponin**, which then displaces tropomyosin. Now unblocked, actin can bind with myosin.

As a result of the binding of actin and myosin, the myosin molecules rotate, causing the thick myosin filaments to slide past the thin actin filaments. In a process requiring energy, the myosin molecules subsequently separate from the actin molecules. As long as calcium and energy are still present in the muscle fiber, the binding and unbinding process repeats itself, and the myosin filaments move step by step along the length of the actin filaments, pulling the Z lines closer to the M line. Consequently, the muscle fiber gradually contracts.

In the absence of further action potentials, the muscle fiber will relax. Calcium is taken up again by the internal organelles, in a process similar to the reuptake of a neurochemical by a presynaptic neuron. When troponin is no longer bound by calcium, tropomyosin once again blocks the interaction of myosin and actin, and the thin and thick filaments slide apart. The sarcomeres and the muscle fiber return to their resting length. If there is a shortage of energy in the cell, the detachment of myosin molecules from actin molecules can't take place, and the muscle becomes locked in its contracted state. This process accounts for the muscular stiffness, or rigor mortis, that occurs after death.

**FIBER TYPES AND SPEED** In humans, the thick myosin filaments in muscle fibers come in three varieties. Type I fibers are known as **slow-twitch fibers**, and types IIa and IIb are known as **fast-twitch fibers**. Type IIa fibers are also known as fast-twitch, fatigue-resistant fibers, while type IIb are also known as fast-twitch, fatigable fibers. Type IIb fibers can contract up to 10 times faster than type I fibers. The contraction velocity of type IIa fibers falls somewhere between type I and type IIb (Scott, Stevens, & Binder-Macleod, 2001). Most skeletal muscles contain mixtures of all three types of fibers but in different proportions. The postural muscles of the back, neck, and legs are dominated by slow-twitch fibers. The muscles of the arms and shoulders contain higher proportions of fast-twitch fibers.

Fast- and slow-twitch fibers use energy differently. Slow-twitch fibers use **aerobic metabolism**, which requires oxygen, whereas the fast-twitch fibers use **anaerobic metabolism**, which occurs in the absence of oxygen. Endurance activities, such as distance running, rely primarily on aerobic slow-twitch fibers. Explosive, powerful movements, such as sprinting, jumping, and weightlifting, employ anaerobic fast-twitch fibers. Muscles dominated by fast-twitch fibers appear white (like the white meat of a turkey breast), whereas those containing slow-twitch fibers appear dark or red (like the dark meat of a turkey's legs and thighs). The red color reflects the presence of myoglobin, an iron-based muscle protein that stores the oxygen necessary for aerobic metabolism, and larger numbers of capillaries. Diving animals, such as seals and whales, owe part of their ability to remain submerged to the unusually high amounts of myoglobin contained in their muscles.

The average adult human has approximately equal numbers of fast- and slow-twitch fibers in the quadriceps muscle on the front of the thigh. However, people vary widely in the composition of their muscles. Jesper Andersen and his colleagues (Andersen, Schjerling, & Saltin, 2000) have observed some people with as few as 19 percent slow-twitch fibers in the quadriceps and other people with up to 95 percent slow-twitch fibers. This variation is probably the result of a single gene, ACTN3, which normally encodes a protein used by fast-twitch fibers (MacArthur & North, 2007). If people receive two copies of a common mutation of the ACTN3 gene, they will produce none of the fast-twitch protein at all and would likely excel at long-distance running and other endurance sports. The proportion of fast- and slow-twitch fibers in the quadriceps muscle of people engaged in different levels of activity is shown in Figure 8.4.

**tropomyosin** A protein that covers actin binding sites in a resting muscle fiber, preventing actin from interacting with myosin.

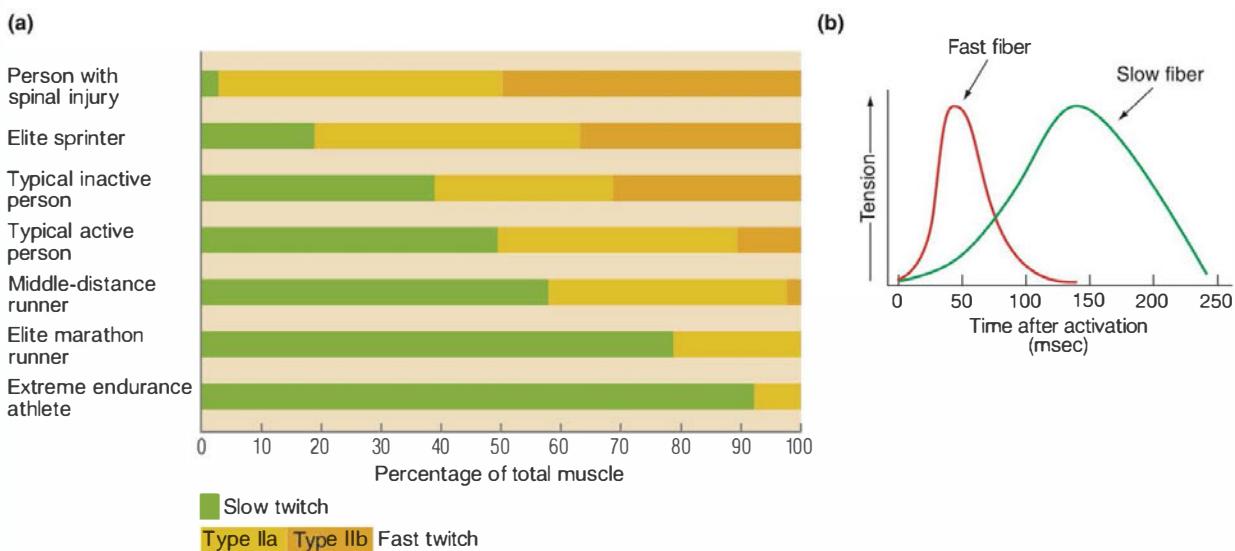
**troponin** A protein that when bound by calcium displaces tropomyosin, allowing actin to interact with myosin.

**slow-twitch fiber** A muscle fiber containing type I myosin filaments and large numbers of mitochondria that contracts slowly using aerobic metabolism; primarily responsible for movement requiring endurance.

**fast-twitch fiber** A muscle fiber containing type IIa or type IIb myosin filaments that contains few mitochondria, uses anaerobic metabolism, and contracts rapidly; primarily responsible for movement requiring explosive strength.

**aerobic metabolism** A chemical process that requires oxygen.

**anaerobic metabolism** A chemical process that does not require oxygen.



► **Figure 8.4 Human Fiber Types** (a) The average adult has approximately equal amounts of fast- and slow-twitch muscles. People who have spinal injury lose most of their slow-twitch fibers. Athletes involved in power events, such as sprinting, usually have a higher proportion of fast-twitch muscles than athletes involved with endurance sports. (b) Because fast fibers produce muscle tension very quickly, they are useful for explosive movements such as sprinting and weightlifting. The slower fibers can produce equal tension, but require a longer time to do so. Slower fibers are used in endurance activities such as long distance running and swimming.

Source: Data from Anderson, Schjerling, & Saltin (2000).

## The Effects of Exercise on Muscle

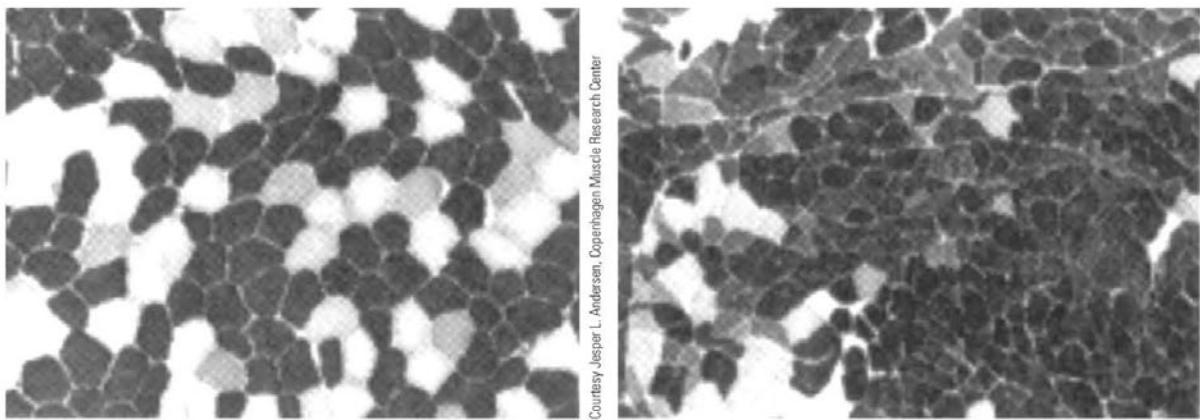
We know that exercise can build muscles. In many cases, muscle enlargement is desirable. However, enlargement of the cardiac muscle often results in life-threatening heart disease. Muscle enlargement occurs in response to muscle fiber damage. When fibers are damaged due to weightlifting or other strenuous activity, more actin and myosin filaments are produced. Lack of activity reverses this process quickly because filament proteins are either broken down faster or synthesized more slowly when muscles are not used (Vandenburgh, Chromiak, Shansky, Del Tutto, & Lemaire, 1999). During space travel, in which lower gravity reduces the activity of major postural muscles, astronauts can lose as much as 20 percent of their muscle mass in as little as two weeks (Andersen et al., 2000).

Under certain circumstances, changes in muscle fiber type do occur. People who are paralyzed as a result of spinal cord injury experience a dramatic increase in fast-twitch fibers and a loss of slow-twitch fibers (see Figure 8.4). Apparently, neural input to the muscle, which is lacking in cases of spinal cord injury, is necessary for the maintenance of slow-twitch fibers.

## The Effects of Aging on Muscles

The loss of muscle mass as a result of aging begins as early as age 25 and accelerates through the remainder of the life span. By the age of 50, most people have lost at least 10 percent of the muscle mass they had at age 25, and that figure rises to 50 percent by the age of 80. Muscle strength is related to muscle mass, but the decline in strength in older adulthood is much more rapid than the loss of muscle mass, suggesting that muscle quality declines as well as overall size (Goodpaster et al., 2006).

As shown in ► Figure 8.5, the muscle fibers of young people seen in cross-section are angular, whereas in older adults the fibers are rounder. In addition, youthful



● **Figure 8.5 Aging Affects the Quantity, Shape, and Distribution of Muscle Fibers** As we age, we lose muscle fibers, which cannot be regrown. A comparison of the young muscle in the left photo with the aged muscle in the right photo shows other changes. The young muscle has angular-shaped fibers and a checkerboard distribution of fast- (light) and slow- (dark) twitch muscles. The aged muscle shows rounded fibers and clustering of fiber types.

## THINKING Ethically

### GENE DOPING FOR STRENGTH

Athletics represents the nature of competition, and athletes appear to be willing to go to great lengths to achieve a "competitive edge." The current use of performance-enhancing drugs, such as anabolic steroids, is likely to be eclipsed in the near future by new, genetically based methods. Gene doping refers to gene therapy for people with no known medical justification. Among the targets for gene doping are myostatin, erythropoietin (EPO), insulin-like growth factor-1 (IGF-1), vascular endothelial growth factor, fibroblast growth factor, peroxisome proliferator-activated receptor-delta (PPAR $\delta$ ), and cytosolic phosphoenolpyruvate carboxykinase (PEPCK-C) (van der Gronde, de Hon, Haisma, & Pieters, 2013). Genetic treatments targeting diseases involving these substances, such as muscular dystrophy (described later in this chapter), could potentially be hijacked by athletes (Fischetto & Bermon, 2013). The strong likelihood that athletes would manipulate combinations of genes, not single genes, would further complicate detection (van der Gronde et al., 2013).

Myostatin is a protein that normally inhibits muscular growth. Inhibition of myostatin due to genetic mutation is associated with unusual musculature and strength. The remarkable musculature of Belgian blue cattle, like the one shown in ● Figure 8.6, results from a mutation in the myostatin gene. Increasing musculature on a frame that was not designed to support it, however, is risky. It is likely that athletes manipulating myostatin would suffer

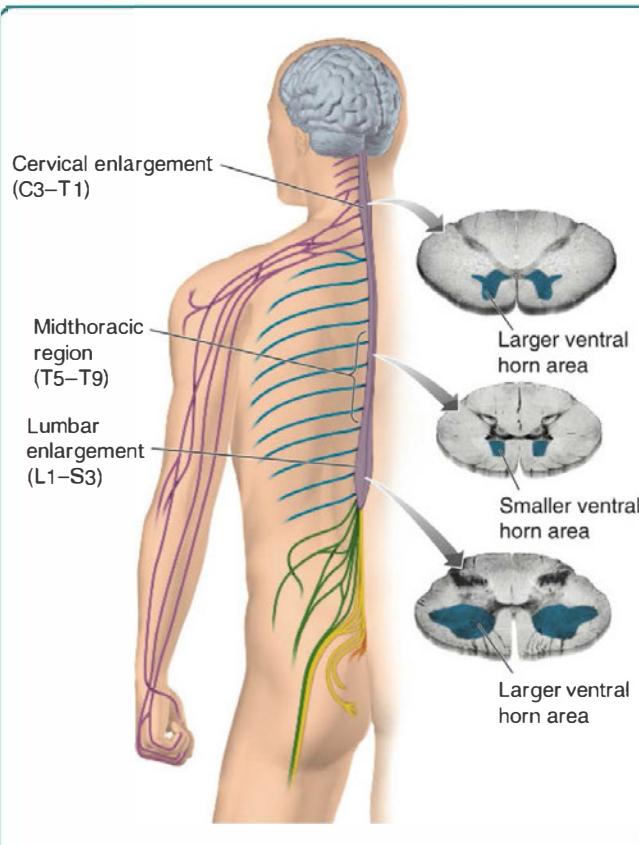


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● **Figure 8.6 Myostatin and Muscle Development** The replacement of typical genes with genes that enhance performance, or gene doping, is likely to replace the use of steroids and other drugs to improve athletic performance. One candidate for genetic modification is the gene for myostatin, a protein that limits muscular growth. Belgian Blue cattle have a mutation in the myostatin gene that produces unusual muscle development.

the same or even worse problems with damage to bones and connective tissue already experienced by those who use steroids to boost muscle growth.

The development of gene editing technologies such as CRISPR is likely to move the possibility of gene doping for athletes ahead (Wells, 2016). In preparation for the inevitable use of these technologies by athletes, proactive methods for detecting changes in a person's genome are under development (Baoutina et al., 2016).



**► Figure 8.7 Distribution of Spinal Motor Neurons** The spinal cord bulges in the segments that serve the arms and legs, due to the large number of alpha motor neurons located in the ventral parts of those segments. In comparison, relatively few alpha motor neurons serve the torso.

muscles have a more even distribution of slow-and fast-twitch fibers. In the muscle of the older adult, the types appear clustered together. Older adults appear to have a much higher proportion of hybrid muscles, neither slow nor fast, when compared with younger people, largely due to a selective atrophy of type II fibers.

Age-related changes also occur in the neurons that control muscles. When older adults move a muscle, the firing rates of the associated neurons are lower than the rates seen in younger adults, resulting in slower and weaker muscle responses (Knight & Kamen, 2007). Although one cannot stop age-related changes in muscle fibers and neural firing rates, remaining physically active can help offset this decline.

## Neural Control of Muscles

The contraction of skeletal muscles is directly controlled by motor neurons originating in either the spinal cord or in the nuclei of the cranial nerves in the brainstem. Just as skeletal muscles are not evenly distributed throughout the body, motor neurons are not evenly distributed throughout the spinal cord. As shown in ► Figure 8.7, the ventral horns of the spinal cord, which contain the motor neurons, appear larger in segments C (Cervical) 3 through T (Thoracic) 1 and again in L (Lumbar) 1 through S (Sacral) 3. The enlargement of the ventral horns in these areas is due to the greater number of motor neurons required to innervate the muscles of the arms and legs, respectively.

## Alpha Motor Neurons

The spinal motor neurons directly responsible for contracting muscles are known as **alpha motor neurons**. These are large myelinated neurons capable of rapid signaling. The alpha motor neurons form highly efficient connections with muscle fibers at a location called the **neuromuscular junction**, shown in ► Figure 8.8. Because of the efficiency of this connection, the arrival of a single action potential in the motor neuron terminal is capable of producing an action potential in the muscle fiber, leading to a single contraction, or twitch.

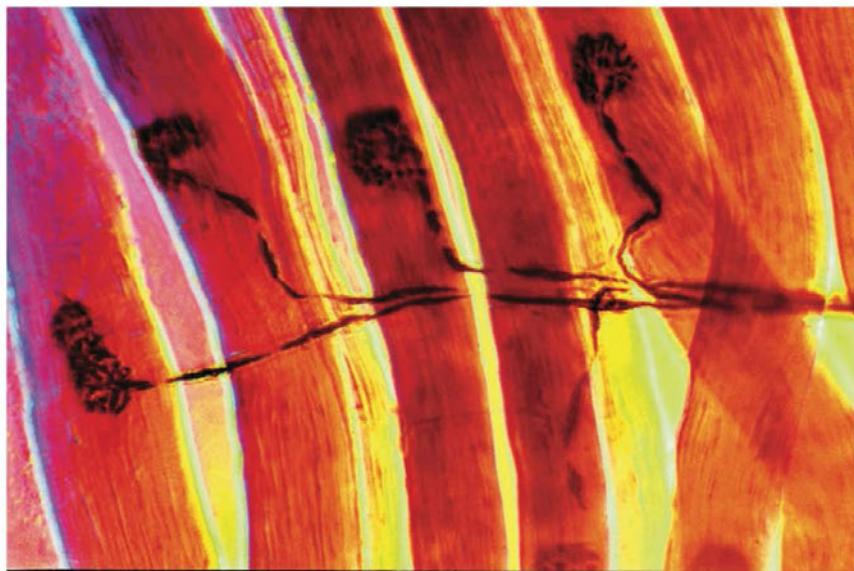
**alpha motor neuron** A spinal motor neuron directly responsible for signaling a muscle fiber to contract.

**neuromuscular junction** A synapse formed between an alpha motor neuron axon terminal and a muscle fiber.

**motor unit** The combination of a single alpha motor neuron and all the muscle fibers that it innervates.

## The Motor Unit

A **motor unit** is made up of a single alpha motor neuron and all the muscle fibers it innervates. Although a single muscle fiber receives input from only one alpha motor neuron, alpha motor neurons can innervate many muscle fibers via their collaterals. Muscles used in fine movement (such as those in the hands and fingers) are controlled by motor neurons that innervate smaller numbers (10–100) of muscle fibers. This allows for the coordinated but individualized movements of muscle fibers needed for precise movement. Muscles that are not involved with fine movement, like your back or thigh muscles, are controlled by motor neurons serving 1,000 or more muscle fibers.



**Figure 8.8 The Neuromuscular Junction** This image shows alpha motor neurons making contact with individual muscle fibers. The little dots at the end of each motor neuron fiber are individual axon terminals.

Wood/Custom Medical Stock Photo/Getty Images

A single motor unit includes either fast- or slow-twitch fibers but not a mixture of both (Burke, 1978). Motor neurons serving slow-twitch muscles have smaller cell bodies and innervate fewer muscle fibers. Consequently, these units generate relatively little force. In contrast, motor neurons serving the very fast IIb fibers have relatively large cell bodies and large-diameter axons, which are capable of rapid signaling. These motor units generate 100 times the force produced by units made up of slow-twitch fibers (Loeb & Ghez, 2000). Motor units containing type IIA fibers fall between these extremes.

The motor units innervating the fibers making up a single muscle, such as the quadriceps muscle of your thigh, are collectively known as a **motor neuron pool**. The cell bodies of motor neuron pools form column-shaped nuclei in the spinal cord.

## The Control of Muscle Contractions

As mentioned previously, a single action potential in the alpha motor neuron usually results in a single contraction in the associated muscle fiber. How, then, do we manage to produce muscular movements of different forces and durations?

There are two methods for controlling the force of our movements (Guyton, 1991). The first method, the **rate code**, is to vary the firing rate of motor neurons. Rapid firing by the motor neuron produces a sustained contraction in the muscle fiber. Contractions last longer than action potentials, allowing temporal summation to occur at the neuromuscular junction (see Chapter 3). The muscle fiber responds with increasing contraction. When the muscle cannot contract any farther, it has reached a state known as **tetanus**. This term is also used to describe the extreme muscle contraction resulting from infection by the bacterium *Clostridium tetani* and its neurotoxin, tetanospasmin, discussed in Chapter 3.

The second method for varying muscle responses is known as **recruitment**, or the “size principle.” As an increased load is placed on a muscle, as when you pick up a heavy object, more motor units are recruited to provide extra tension in the muscle. Recruitment proceeds according to the nature of the motor units (Henneman, 1991). Smaller, slow-twitch units are recruited first, followed by the intermediate type IIA units, and finally the largest, fastest type IIb units. The smaller neurons that innervate slow-twitch fibers are more easily excited by synaptic input, which explains why they

**motor neuron pool** The collection of motor neurons that innervates a single muscle.

**rate code** Variations in the firing rate of motor neurons to meet the need for a certain amount of contraction.

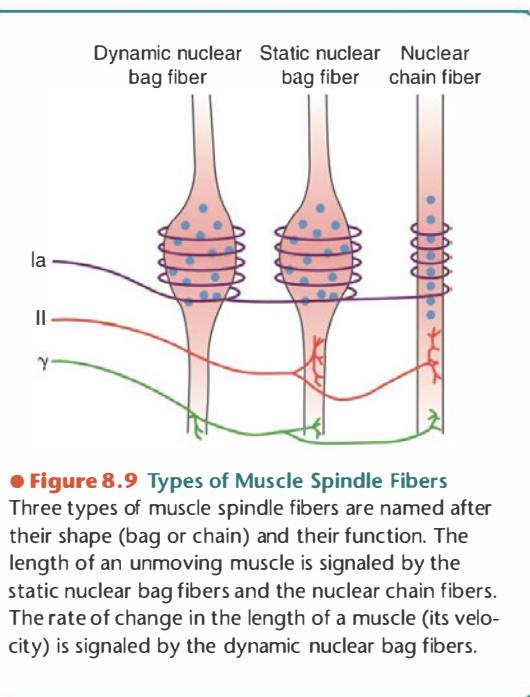
**tetanus** The point at which a muscle cannot contract farther.

**recruitment** The process of gradually activating more motor units as an increasing load is placed on a muscle.

are recruited first. Recruiting the smaller motor units first ensures that the body uses the smallest amount of force and energy to get a job done.

## The Control of Spinal Motor Neurons

The alpha motor neurons do not initiate movement on their own. Instead, these neurons react to information about the external environment and the current state of the body. To respond appropriately, alpha motor neurons require input from three types of neurons: neurons from muscle spindles and Golgi tendon organs, neurons of the brainstem and motor cortex, and spinal interneurons.



**Figure 8.9 Types of Muscle Spindle Fibers**

Three types of muscle spindle fibers are named after their shape (bag or chain) and their function. The length of an unmoving muscle is signaled by the static nuclear bag fibers and the nuclear chain fibers. The rate of change in the length of a muscle (its velocity) is signaled by the dynamic nuclear bag fibers.

**muscle spindle** A sensory structure that provides feedback regarding muscle stretch.

**intrafusal muscle fiber** One of the fibers that makes up a muscle spindle.

**extrafusal muscle fiber** One of the fibers outside the muscle spindle that is responsible for contracting the muscle.

**nuclear chain fibers** A type of muscle spindle fiber that provides information about muscle length.

**static nuclear bag fibers** A type of muscle spindle fiber that provides information about muscle length.

**dynamic nuclear bag fibers** A type of muscle spindle fiber that provides information about the velocity of change in the length of a muscle.

**Ia sensory fiber** A large and fast sensory axon that connects a muscle spindle to neurons in the spinal cord.

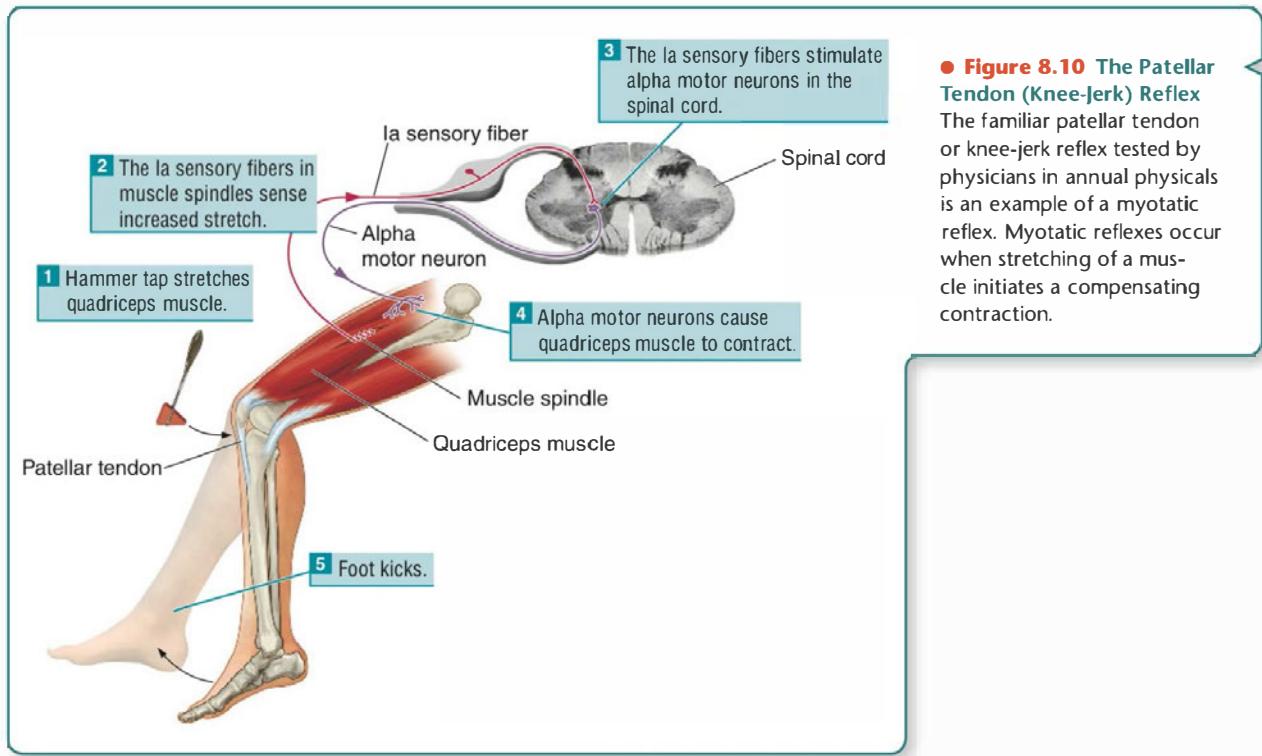
**group II sensory fiber** Sensory fiber that provides information about muscle length.

ily for force, such as those in the torso and legs. Larger numbers of muscle spindles provide the more precise feedback required for fine movements.

Figure 8.9 illustrates three types of muscle spindle fibers: nuclear chain fibers, static nuclear bag fibers, and dynamic nuclear bag fibers. The distinction between the types is based on their structure and the type of information each communicates. **Nuclear chain fibers** and **static nuclear bag fibers** provide information about the length of a muscle. **Dynamic nuclear bag fibers** provide information about the rate of change in the length of a muscle, or its velocity of change.

Wrapped around the middle section of each type of spindle fiber are very large, very fast axons known as **group Ia sensory fibers**. Ia fibers are a type of the Aα fibers we mentioned in Chapter 7 (see Figure 7.17). These fibers generate action potentials every time the muscle spindle stretches. Because the Ia fibers interact with all three types of muscle spindle fibers, they provide information about both length and velocity. Contacting only the static nuclear bag fibers and the nuclear chain fibers are **group II sensory fibers**. Because these fibers do not interact with the dynamic nuclear bag fibers, they provide information about length, but not velocity. Group Ia fibers fire rapidly during the stretch of a muscle (length plus velocity) but begin to fire more slowly at the end of the stretch (length only). In contrast, the group II fibers fire rather steadily as a muscle is stretched. Within the spinal cord, the group Ia and group II fibers synapse on interneurons and on the alpha motor neurons.

Here's how the system works. As the cup in our example is placed in your hand, your arm muscle will stretch due to the added weight. The stretching of your arm



muscle will also stretch the muscle spindle. The Ia and II fibers surrounding the muscle spindle fibers will sense the increased stretch and will excite the alpha motor neurons in the spinal cord. Excitation of the alpha motor neurons will cause the muscle to balance the stretch with further contraction. The cup is now safe.

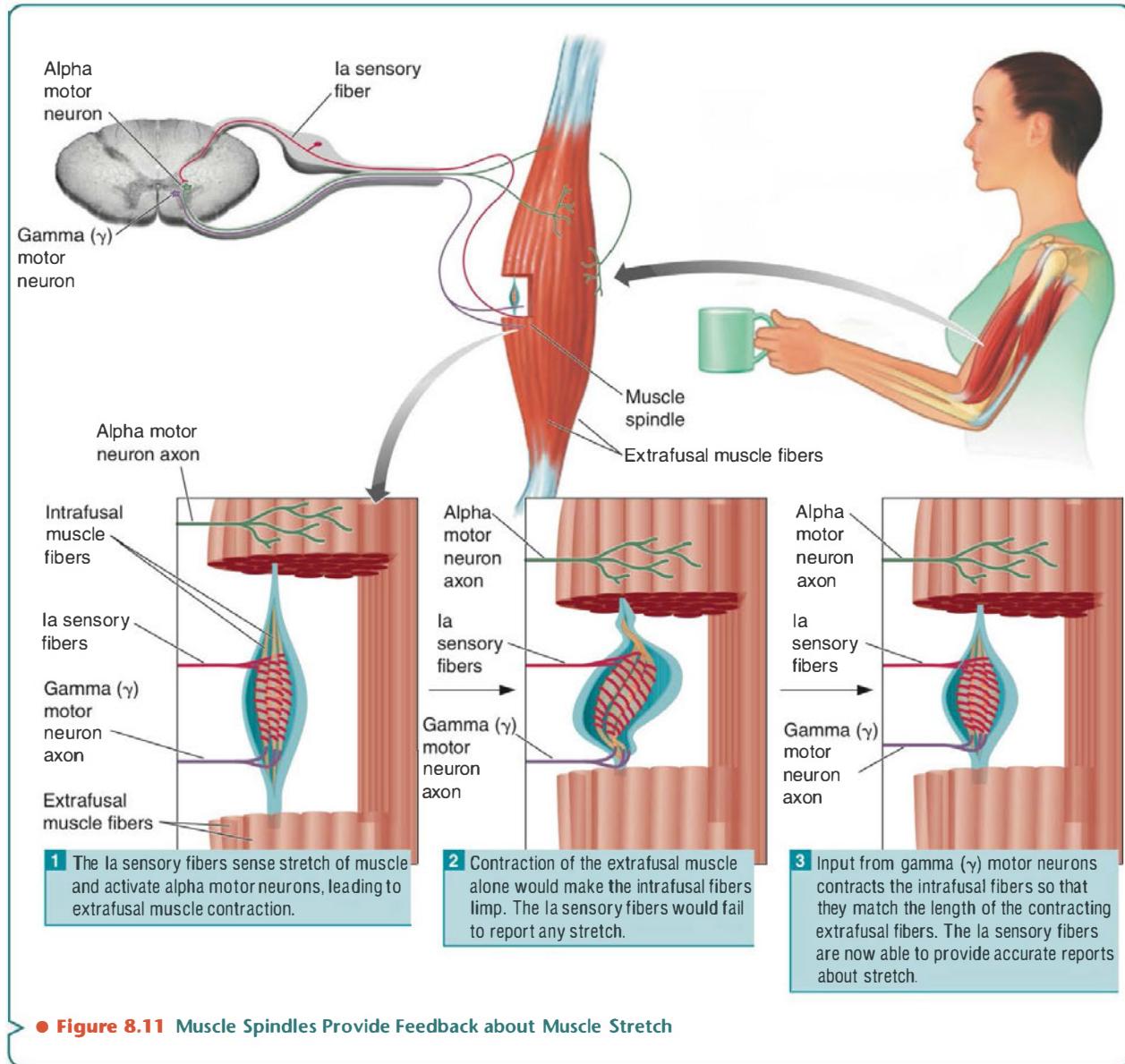
Contraction in response to sensing stretch is known as a **myotatic reflex**. Myotatic reflexes are examples of **monosynaptic reflexes**, in which only a single synapse between a sensory neuron and a motor neuron is involved. In addition to helping us hold a cup of coffee steadily, myotatic reflexes allow us to maintain a steady posture. Tilting to the side stretches muscles, and the resulting contractions from the myotatic reflex pulls you back to center. Another familiar type of myotatic reflex is the patellar tendon or knee-jerk reflex often performed as part of an annual physical (see ●Figure 8.10). The physician taps the tendon located just below the knee, stretching the quadriceps muscle of the thigh. To compensate for the stretch, the quadriceps contracts and the foot kicks out. This test helps to determine whether a person's myotatic reflexes are in good working order. Some medical conditions, such as diabetes and Parkinson's disease, reduce or eliminate the reflex. Other medical conditions, such as meningitis (see Chapters 2 and 15), often produce exaggerated responses.

We have seen that the alpha motor neurons provide input to the extrafusal fibers. The intrafusal fibers have their own set of motor neurons, known as **gamma motor neurons**, shown in Figures 8.9 and 8.11. Why would the intrafusal fibers need input when they do not contribute to the overall contraction of a muscle? Without the gamma motor neurons, the intrafusal fibers could not provide accurate information about how far the muscle was stretched. When the extrafusal fibers contract, the intrafusal fibers would become limp if they did not also contract. This would cause the Ia and II sensory fibers to stop signaling, and the brain and spinal cord would not know how long the muscle was. To solve this problem, gamma motor neurons and alpha motor neurons are activated simultaneously by input from the brain. The gamma motor neurons cause a small contraction of the spindle at nearly the same time that the alpha motor neurons

**myotatic reflex** The contraction of a muscle in response to sensory information about its having been stretched.

**monosynaptic reflex** A spinal reflex, such as the patellar reflex, that requires the action of only one synapse between sensory and motor neurons.

**gamma ( $\gamma$ ) motor neuron** A small spinal neuron that innervates the muscle spindles.



• **Figure 8.11** Muscle Spindles Provide Feedback about Muscle Stretch

contract the extrafusel fibers. In this way, the spindle matches the length of the muscle, and the Ia and II fibers can provide continuous feedback.

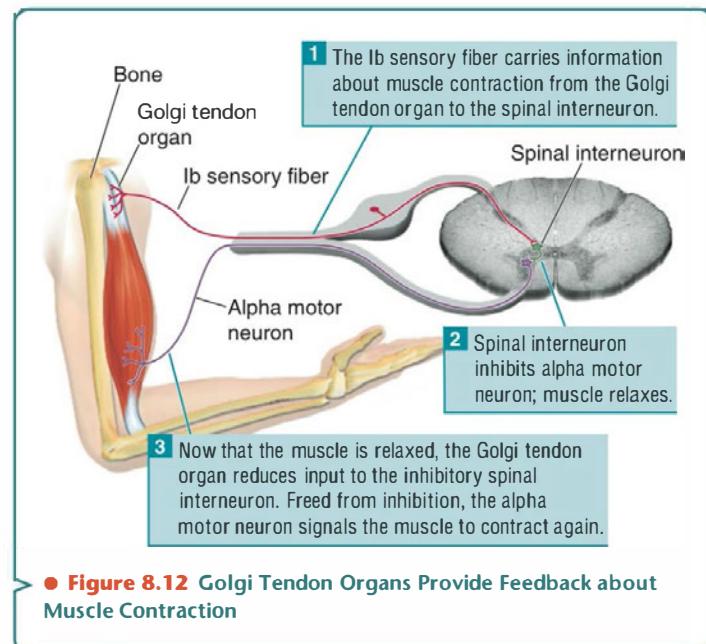
**FEEDBACK FROM GOLGI TENDON ORGANS** The muscle spindles do a good job of providing the brain and spinal cord with information about muscle length, or the degree of stretch. However, we also need feedback regarding the degree of muscle contraction, or force. This information is provided by the **Golgi tendon organs**, which are located at the junction between a muscle and its tendon.

Like the muscle spindles, the Golgi tendon organs are innervated by sensory axons, the **Ib sensory fibers**. Ib fibers are a second type of Alpha-alpha (A<sub>a</sub>) fiber but are smaller and slower than the Ia fibers that innervate the spindles. The Ib fibers from the Golgi tendon organs enter the spinal cord and form synapses with spinal interneurons. In turn, these interneurons form inhibitory synapses on alpha motor neurons.

**Golgi tendon organ** A structure located in the tendons of muscles that provides information about muscle contraction.

**Ib sensory fiber** A small, slower Alpha-alpha (A<sub>a</sub>) sensory axon that connects the Golgi tendon organs to neurons in the spinal cord.

To understand how the Golgi tendon organ feedback loop works, we return to the example of holding a coffee cup steady. The Ia and II fibers from muscle spindles in your fingers and arm sense the stretch resulting from holding the cup and activate the alpha motor neurons. The Golgi tendon organs respond to the resulting increase in muscle tension by sending signals to the spinal interneurons via the Ib sensory fibers. In response to this input, the interneurons inhibit the alpha motor neurons, and muscle contraction is reduced. However, the reduced muscle contraction results in less Golgi tendon organ activity, less input to the spinal interneurons, and less inhibition of the alpha motor neurons. The muscle contracts again. Not only does this system help prevent damage to the muscle fibers from too much contraction, but it also maintains the steady control over muscle tension that we need, particularly for fine motor movements. This process is illustrated in Figure 8.12.



● **Figure 8.12** Golgi Tendon Organs Provide Feedback about Muscle Contraction

**FEEDBACK FROM JOINTS** In addition to receiving feedback about muscle length and tension, we also receive information about position and movement from mechanoreceptors in the tissues surrounding each joint. These mechanoreceptors, which we discussed in Chapter 7, respond primarily to movement of the joint, and they are relatively quiet when the joint is at rest. Receptors located in the skin near joints also supply information about movement and position (Edin, 2001). Free nerve endings can signal pain resulting from extreme joint positions. It appears that joint receptors are not entirely necessary for judging the location of a joint. People who have undergone full hip or knee replacement surgery lose all of their joint receptors in the procedure, yet they can still describe the position of their joint without looking.

### INTERIM SUMMARY 8.1

#### Summary Table: The Control of Spinal Motor Neurons

Cell Type	Location	Source of Input to These Cells	Fiber Type	Action Produced
Alpha motor neurons	Ventral horns of the spinal cord, some cranial nerve nuclei	Motor cortex, brainstem, muscle spindles, spinal interneurons		Alpha motor neurons produce action potentials in muscle fibers, leading to contraction.
Muscle spindles	Lie parallel to muscle fibers within a muscle	Respond to stretch of associated muscle fibers	Ia and II sensory fibers	Muscle spindles synapse with alpha motor neurons and spinal interneurons, allowing muscle contraction to adjust to muscle stretching.

(continued)

Cell Type	Location	Source of Input to These Cells	Fiber Type	Action Produced
Gamma motor neurons	Ventral horns of the spinal cord, some cranial nerve nuclei	Motor cortex, brainstem		Gamma motor neurons contract the muscle spindle to match spindle length to surrounding muscle fibers.
Golgi tendon organs	Junctions between muscles and tendons	Respond to tension in muscle	Ib sensory fibers	Golgi tendon organs synapse with spinal interneurons, inhibiting activity of alpha motor neurons, reducing contraction.

### Summary Points

1. Muscles can be divided into three types: smooth muscles, cardiac muscles, and skeletal muscles. Together, the cardiac and skeletal muscles are known as striated muscles. (LO1)
2. Skeletal muscles are made up of individual muscle fibers surrounded by a membrane similar to that of a neuron. Muscle fiber contractions result from the movement of myosin filaments relative to actin filaments. (LO1)
3. Alpha motor neurons in the spinal cord or cranial nerve nuclei produce muscle contraction through their activity at the neuromuscular junction. (LO2)
4. Muscle spindles provide information about muscle stretch. Gamma motor neurons help the muscle spindle match the length of the muscle, providing continuous feedback. (LO3)
5. Golgi tendon organs located in the connective tissue between muscle and bone provide information regarding muscle contraction. Additional joint receptors provide information regarding the movement of joints. (LO3)

### Review Questions

1. What is the sequence of events within the myofibril that leads to contraction?
2. What is a motor unit, and how do the units differ in terms of size, precision of movement, and muscle fiber type?

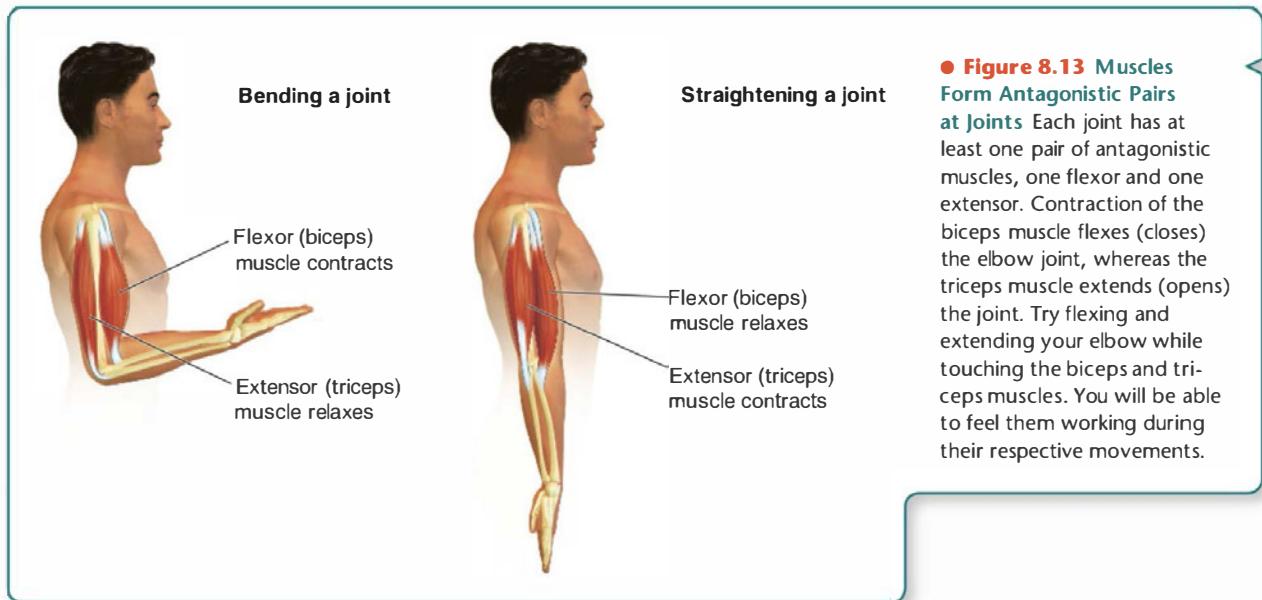
## Reflex Control of Movement

The spinal cord is responsible for a number of reflex movements designed to protect us from injury, to maintain posture, and to coordinate the movement of our limbs. Most of these reflexes are examples of **polysynaptic reflexes**, or reflexes requiring more than one synapse. In contrast, the myotatic reflexes discussed previously are monosynaptic, or requiring only a single synapse between a sensory neuron and a motor neuron.

### Reciprocal Inhibition at Joints

Muscles can do only one thing: contract. The contraction of a single muscle can either straighten or bend a joint, but not both. As a result, a muscle is able to pull a bone in a

**polysynaptic reflex** A spinal reflex that requires interaction at more than one synapse.



**● Figure 8.13 Muscles**

**Form Antagonistic Pairs at Joints** Each joint has at least one pair of antagonistic muscles, one flexor and one extensor. Contraction of the biceps muscle flexes (closes) the elbow joint, whereas the triceps muscle extends (opens) the joint. Try flexing and extending your elbow while touching the biceps and triceps muscles. You will be able to feel them working during their respective movements.

single direction but is unable to push it back. Relaxing the muscle will not necessarily cause a limb to move back to its original position.

To move a joint in two directions requires two muscles. Muscles are arranged at a joint in **antagonistic pairs**, as shown in ● Figure 8.13. Muscles that straighten joints are referred to as **extensors**, and muscles that bend joints are known as **flexors**. To bend the knee, the flexor muscles of the thigh must contract while the extensor muscles relax. To straighten the leg, the extensor muscles must contract while the flexors relax. Under most circumstances, flexors and extensors at the same joint are prevented from simultaneously contracting by a polysynaptic reflex known as **reciprocal inhibition**.

Coordinating this reciprocal inhibition requires inhibitory spinal interneurons. The Ia fibers branch in the spinal cord. As discussed previously, one branch communicates with the alpha motor neuron serving the muscle that is contracting to keep muscle tension steady. The other branch communicates with an inhibitory interneuron, which in turn synapses onto the alpha motor neuron serving the opposing muscle that needs to relax. Thus, information about contraction of the first muscle is transmitted by the Ia fiber to the inhibitory interneuron, which in turn prevents the alpha motor neuron from contracting the opposing muscle.

In some circumstances, such as when we stiffen our elbows while catching a ball, the stability provided by simultaneous contraction of antagonistic muscle pairs is actually desirable. Achieving simultaneous contraction rather than reciprocal inhibition requires descending control from the brain, which acts by changing the firing patterns of the inhibitory spinal interneurons.

## The Flexor Reflex

Another familiar example of a polysynaptic reflex is the **flexor reflex**. We rely on flexor reflexes to protect us from further injury, such as when we jerk our hand away after touching a hot surface on the stove. It's a good thing that the spinal cord, rather than the brain, manages this function. By the time the brain perceived the problem, generated solutions, evaluated solutions, and implemented solutions, your hand would be in bad shape. The flexor reflex begins as sensory neurons transmit information about the painful stimulus to interneurons in the spinal cord.

**antagonistic pair** Two opposing muscles, one a flexor and one an extensor, arranged at a joint.

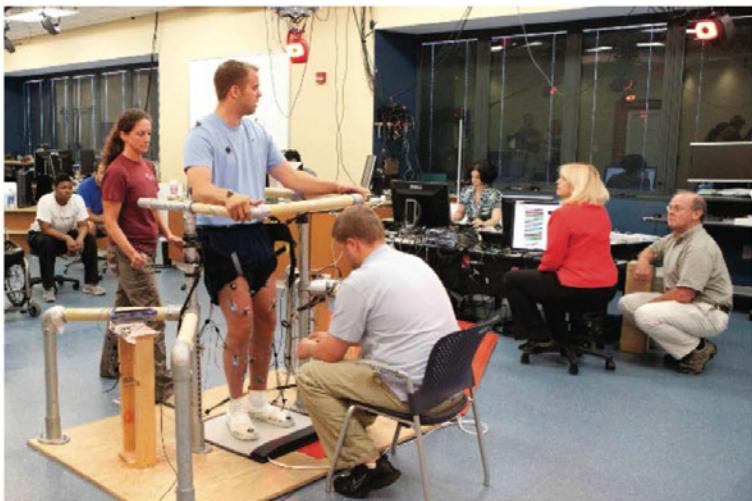
**extensor** A muscle that acts to straighten a joint.

**flexor** A muscle that acts to bend a joint.

**reciprocal inhibition** A polysynaptic reflex that prevents the simultaneous contraction of flexors and extensors serving the same joint.

**flexor reflex** A polysynaptic spinal reflex that produces withdrawal of a limb from a painful stimulus.

**● Figure 8.14** The use of epidural stimulation to activate central pattern generators in the spinal cord has been used to restore some voluntary movement to patients with spinal cord injury.



University of Louisville

The interneurons excite the alpha motor neurons serving the flexor muscles of the affected limb. At the same time, alpha motor neurons serving the opposing muscle, the extensor, are inhibited. As a result, your hand is successfully pulled back from the heat source.

### Spinal Reflexes Related to Walking

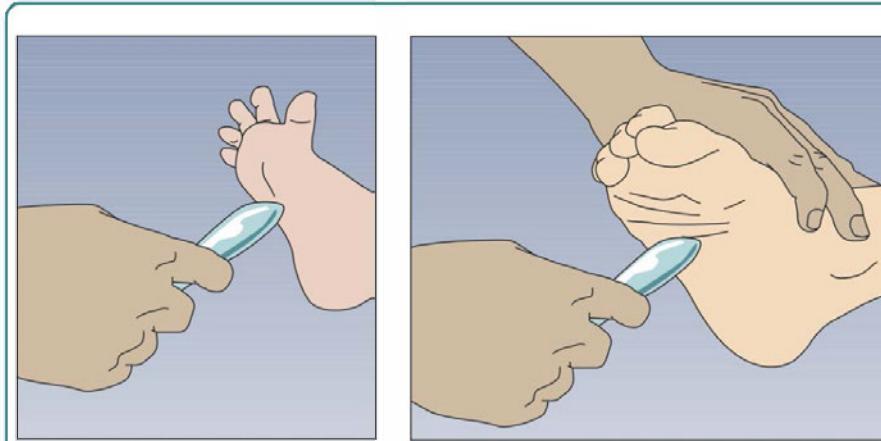
More complicated polysynaptic reflexes help coordinate the movement of several limbs at once. When we walk, we naturally balance our weight from side to side and swing our arms. These complex movements require many more synapses than the previous simple reflexes. In addition, there is growing evidence that the rhythm of movements such as walking is governed by spontaneously active central pattern generators within the spinal cord (Gordon & Whelan, 2006; Grillner et al., 1998; Marder & Bucher, 2001). In some patients, like the one shown in ●Figure 8.14, epidural stimulation of the central pattern generators of the spinal cord restored standing, voluntary movement, and assisted stepping in patients with spinal cord injury (Harkema et al., 2011; Minassian, McKay, Binder, & Hofstoetter, 2016).

### Reflexes over the Lifespan

The reflexes we have discussed so far are present throughout the human life span. Other types of reflexes are characteristic of young children but tend to diminish as the nervous system matures. These childhood reflexes are not lost by the nervous system, but they become inhibited or overwritten by other processes. When this normal inhibition of childhood reflexes fails due to brain damage or the use of alcohol and other drugs, the reflexes will reappear.

Among the normal childhood reflexes is the **Babinski sign**, a type of polysynaptic flexion reflex. When you stroke the bottom surface of the foot, infants will spread their toes with the big toe pointing up. The Babinski sign does not appear to confer any particular benefit to the infant. Instead, the reflex probably reflects the immaturity of the infant's motor system. In typical adults, stroking the bottom of the foot causes the toes to curl down, not up. Adults with damage to either the motor cortex or spinal motor pathways will show the infant's version of the reflex (Gardner, 1968). An illustration comparing the typical adult reaction with the infant's Babinski sign may be seen in ●Figure 8.15.

**Babinski sign** A polysynaptic reflex present in infants and adults with neural damage, in which stroking the sole of the foot causes the toes to spread with the big toe pointing upward.



(a) Typical infant Version of Babinski Sign

(b) Typical Reaction to the Stroking of the Foot in Subjects over Two Years Old

**● Figure 8.15 The Babinski Sign** (a) Stroking the bottom of an infant's foot causes the toes to spread and the big toe to point upward, a movement known as the Babinski sign. (b) Stroking the bottom of an adult's foot results in the downward curling of the toes. In adults, the Babinski sign often indicates brain damage.

## Motor Systems of the Brain

The alpha motor neurons that contract muscle fibers are at the lowest end of a chain of command for initiating movement. In addition to input from interneurons and stretch receptors, the alpha motor neurons receive direction from neurons located in the cerebellum, basal ganglia, red nucleus, brainstem, and cerebral cortex. As we will see, some of these motor pathways initiate voluntary movement, whereas others are responsible for subconscious, automatic movements.

The central motor system is not strictly hierarchical, however. Multiple parallel pathways communicate from one level of the motor system to the next. As a result, we usually observe complete paralysis only as a result of damage to the lowest levels of the hierarchy in the spinal cord. Damage to the brain itself, for example, can definitely impact movement in a negative way, but moving is still typically possible due to the ability of one pathway to compensate for damage to another.

### Spinal Motor Pathways

Motor neurons in the spinal cord can be located according to their roles as serving flexors and extensors and whether they serve proximal or distal parts of the body (see Chapter 2). Motor neurons associated with flexors are located dorsally to those associated with extensors. Motor neurons associated with distal structures, such as your hands, are located laterally to those than serve more proximal structures, like your torso.

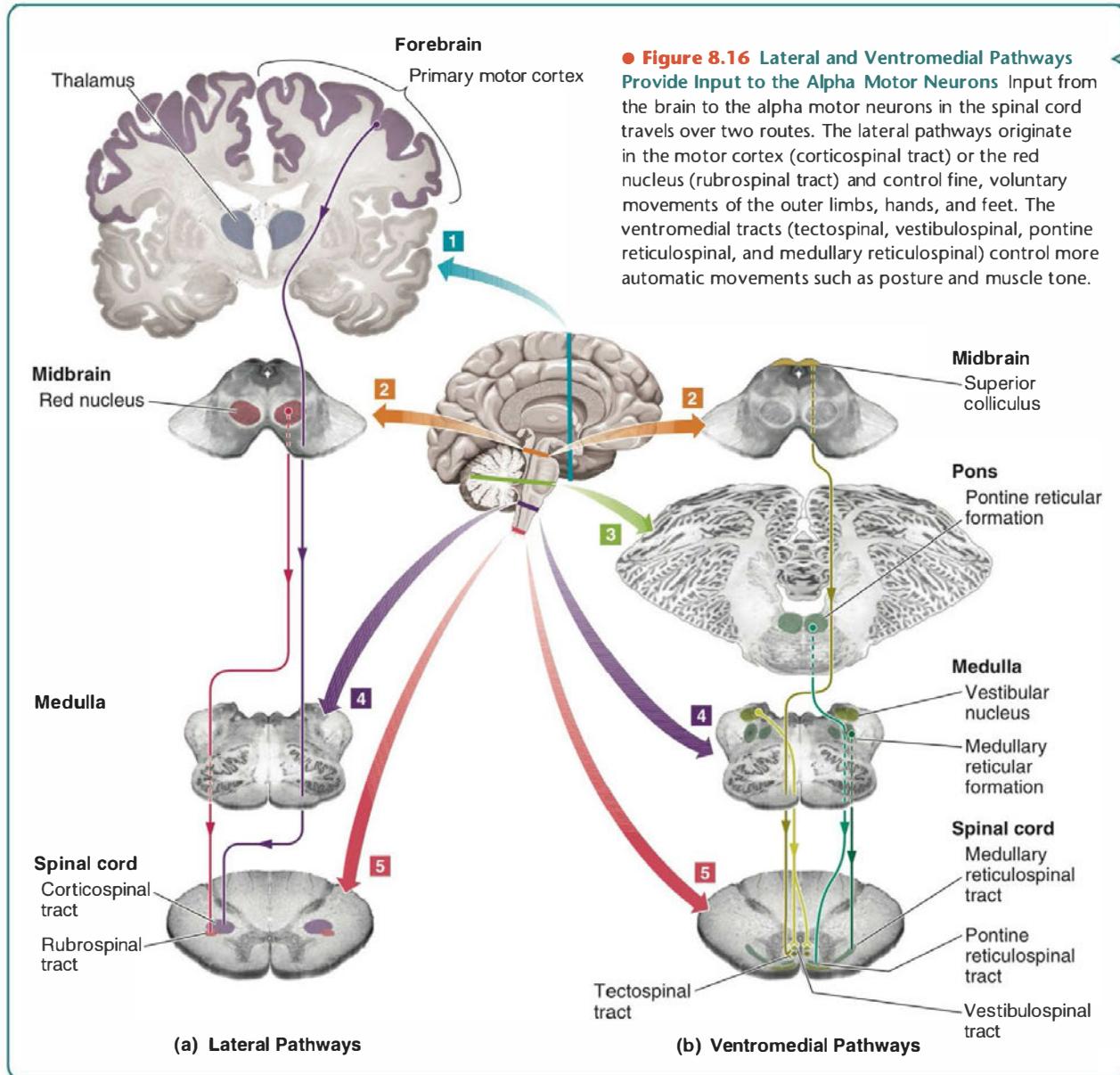
The brain manages movement of the head and neck through the **corticobulbar tract**, which connects primary motor cortex with the brainstem nuclei serving the cranial nerves, and of the rest of the body through connections with spinal alpha motor neurons. As shown in ● Figure 8.16, signals from the brain to the spinal alpha motor neurons travel along two routes. The first route, known as the **lateral pathway**, is located in the lateral part of the spinal column. This pathway originates in the cerebral cortex and in the red nucleus of the midbrain and is the pathway through which the brain controls voluntary fine movements of the hands, feet, and outer limbs. You use this pathway to write notes, drive your car, and type on your keyboard.

The second route, known as the **ventromedial pathway**, travels along the ventromedial part of the spinal column. Most of the neurons that supply axons to this

**corticobulbar tract** A pathway connecting primary motor cortex with brainstem cranial nerve nuclei to manage movement of the head and neck.

**lateral pathway** A large collection of axons that originates in the cerebral cortex, synapses on either the red nucleus or alpha motor neurons, and controls voluntary movements.

**ventromedial pathway** A spinal motor pathway originating in the brainstem and carrying commands for subconscious, automatic movements of the neck and torso.



pathway are located in the brainstem rather than in the cerebral cortex. As a result, the ventromedial pathway carries commands from the brain for automatic movements in the neck, torso, and portions of the limbs close to the body. You use the ventromedial pathway for behaviors such as maintaining posture and muscle tone and moving the head in response to visual stimuli. The functions of these two pathways are easier to remember if you think of the lateral pathway as a long-distance system serving more distal structures (hands, feet, limbs) and the ventromedial pathway as a relatively local system serving more proximal structures (neck and torso).

Cell bodies giving rise to the axons of the lateral pathway are located either in the primary motor cortex of the frontal lobe (the corticospinal tract) or in the red nucleus of the midbrain (the rubrospinal tract). As is true of most anatomical terms, the first part of each term refers to the origin of the pathway (*rubro* refers to red) and the last part of the term refers to the pathway's endpoint.

The fibers of the **corticospinal tract** are some of the fastest and longest in the central nervous system (CNS). This tract is also one of the largest in the nervous system, featuring approximately 1 million axons in humans. This is the only descending system that makes direct synapses with alpha motor neurons. Humans and other primates have more of these directly synapsing corticospinal neurons than most other animals, and this feature probably accounts for our remarkable fine motor coordination of the hands and fingers.

The **rubrospinal tract** connects the red nucleus of the midbrain to the alpha motor neurons of the spinal cord. As you may recall from Chapter 2, the red nucleus receives substantial input from the motor cortex. Consequently, the motor cortex exerts both direct control (via the corticospinal tract) and indirect control (via the rubrospinal tract) on the alpha motor neurons of the spinal cord. However, the main source of input to the red nucleus is the cerebellum, so this pathway is probably one way that learned patterns of movement are communicated to the muscles.

As these pathways travel from the brain to the spinal cord, they decussate or cross the midline. The rubrospinal tract decussates immediately in the midbrain, and the corticospinal tract decussates at the junction of the medulla and the spinal cord. Crossing the midline means that the right hemisphere's motor cortex and red nucleus controls the left side of the body, whereas the left hemisphere's motor cortex and red nucleus controls the right side of the body. Although the advantages of this organization remain a mystery, the results are most obvious when a person has damaged either the motor cortex or the descending lateral pathways due to a stroke or other accident. If the damage occurs in the right hemisphere, movement on the left side of the body will be affected. If the damage occurs in the left hemisphere, movement on the right side of the body will be affected.

The four ventromedial pathways stimulate the alpha motor neurons to help maintain posture and carry out reflexive responses to sensory input such as moving the head and torso in coordination with our eye movements. The ventromedial pathways also assist in behaviors such as walking, in spite of the fact that these behaviors are largely under cortical control. These pathways originate in various parts of the brainstem, including the vestibular nuclei of the medulla, the superior colliculi of the midbrain, and the reticular formation in the pons and medulla. Once again, you can use the names of these tracts to remember their points of origin. The **tectospinal tract** originates in the tectum of the midbrain, primarily in the superior and inferior colliculi (see Chapter 2). The **vestibulospinal tract** originates in the vestibular nuclei of the medulla, and the **pontine and medullary reticulospinal tracts** originate in the reticular formation at the levels of the pons and medulla, respectively. The reticulospinal tract ensures that the flexor reflex described earlier will result from painful stimuli, but not other types of input.

## The Cerebellum

The cerebellum participates in the maintenance of balance and coordination, as well as in the learning of motor skills (see Chapter 2). The cerebellum's role in movement is probably its best understood function, and most of its output connects with other motor systems, but it would be inaccurate to conclude that its functions are restricted to movement. The cerebellum also participates in language and other cognitive processes, discussed further in Chapter 12.

Although the cerebellum does not appear to initiate movement, it plays a very important role in the sequencing of complex movements. As you ride a bicycle or shoot a basket, your cerebellum is coordinating the contraction and relaxation of muscles at just the right time.

To understand the value of the cerebellum, it's helpful to see what happens when the cerebellum is not working. One common example of poor cerebellar function occurs when a person drinks alcohol. The cerebellum is one of the first structures in the brain to show the effects of alcohol, leading to a lack of balance and coordination. As a result, law enforcement personnel check the function of the cerebellum to assess

**corticospinal tract** A pathway connecting the motor cortex to alpha motor neurons in the spinal cord.

**rubrospinal tract** A pathway connecting the red nucleus of the midbrain to the alpha motor neurons of the spinal cord.

**tectospinal tract** A ventromedial pathway connecting the tectum of the midbrain to the alpha motor neurons in the spinal cord.

**vestibulospinal tract** A ventromedial pathway that connects the vestibular nuclei of the medulla to the alpha motor neurons of the spinal cord.

**pontine reticulospinal tract** A ventromedial pathway connecting the reticular formation in the pons to the alpha motor neurons of the spinal cord.

**medullary reticulospinal tract** A ventromedial pathway connecting the reticular formation in the medulla to the alpha motor neurons of the spinal cord.

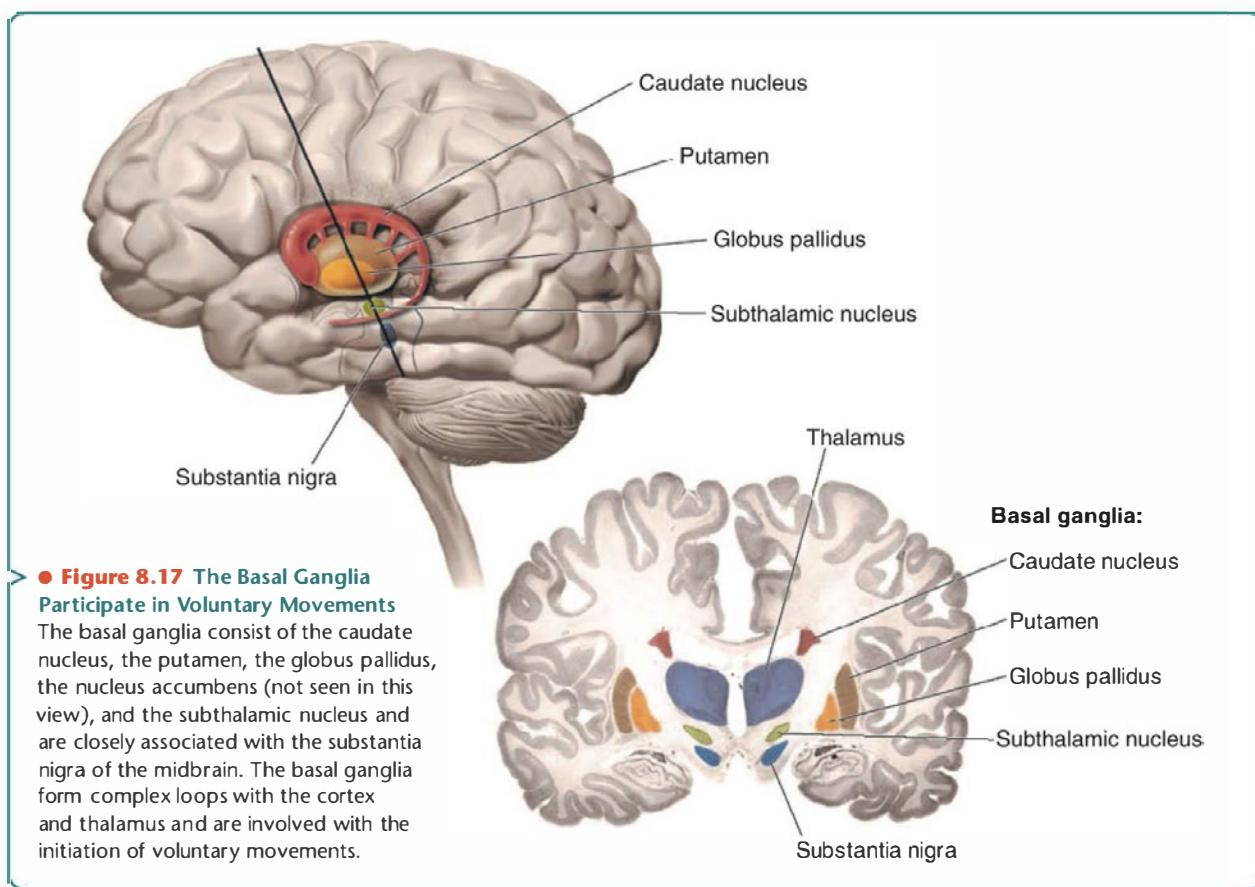
drunkenness. Most sobriety tests, such as walking a straight line, are essentially the same as the tests a neurologist would use to diagnose lesions in the cerebellum.

How does the cerebellum help us coordinate sequenced movements? It appears that the cerebellum is able to inform the motor cortex about such factors as the direction, force, and timing required to carry out a skilled movement. In many cases, this process requires learning. The cerebellum is constantly comparing the cortex's intended movements with what actually happened. Adjustments as needed are made for future activity.

## The Basal Ganglia

Moving rostrally through the motor hierarchy toward the cerebral cortex, we come next to the basal ganglia, a collection of large nuclei embedded within the white matter of the cerebral hemispheres. The location of the basal ganglia is illustrated in **Figure 8.17**. Among their many tasks, only some of which involve motor activity, the basal ganglia participate in the choice and initiation of voluntary movements.

As we saw in Chapter 2, the basal ganglia consist of the caudate nucleus, the putamen, the globus pallidus, the nucleus accumbens, and the subthalamic nucleus. Some anatomists include the substantia nigra of the midbrain in their discussion of the basal ganglia, due to the close linkages between these structures. Complex interactive loops connect the basal ganglia with the thalamus and with the motor cortex in the frontal lobe. The basal ganglia interact with the thalamus via two pathways, a direct pathway that excites the thalamus and an indirect pathway that inhibits the thalamus. As a result, you might think of the basal ganglia as a gate or filter for intentional activity, in which motor programs associated with reward will be carried out while competing motor programs unlikely to produce reward in the present circumstances are inhibited.



In other words, the basal ganglia do not initiate movement, but without the “approval” of the basal ganglia, the cerebral cortex cannot send motor commands to lower levels of the system.

As we will see later in the chapter, a number of disorders result from abnormalities in the basal ganglia, including Parkinson’s disease and Huntington’s disease. These conditions feature motor activity that is either lower (Parkinson’s) or higher (Huntington’s) than normal. In addition to these two primarily motor disorders, the basal ganglia are implicated in a number of psychological disorders, including obsessive-compulsive disorder and attention deficit hyperactivity disorder (see Chapter 16). Like the cerebellum, the basal ganglia participate in a number of important cognitive functions in addition to their motor responsibilities, such as the formation and management of various types of implicit or unconscious memories (see Chapter 12).

## The Motor Cortex

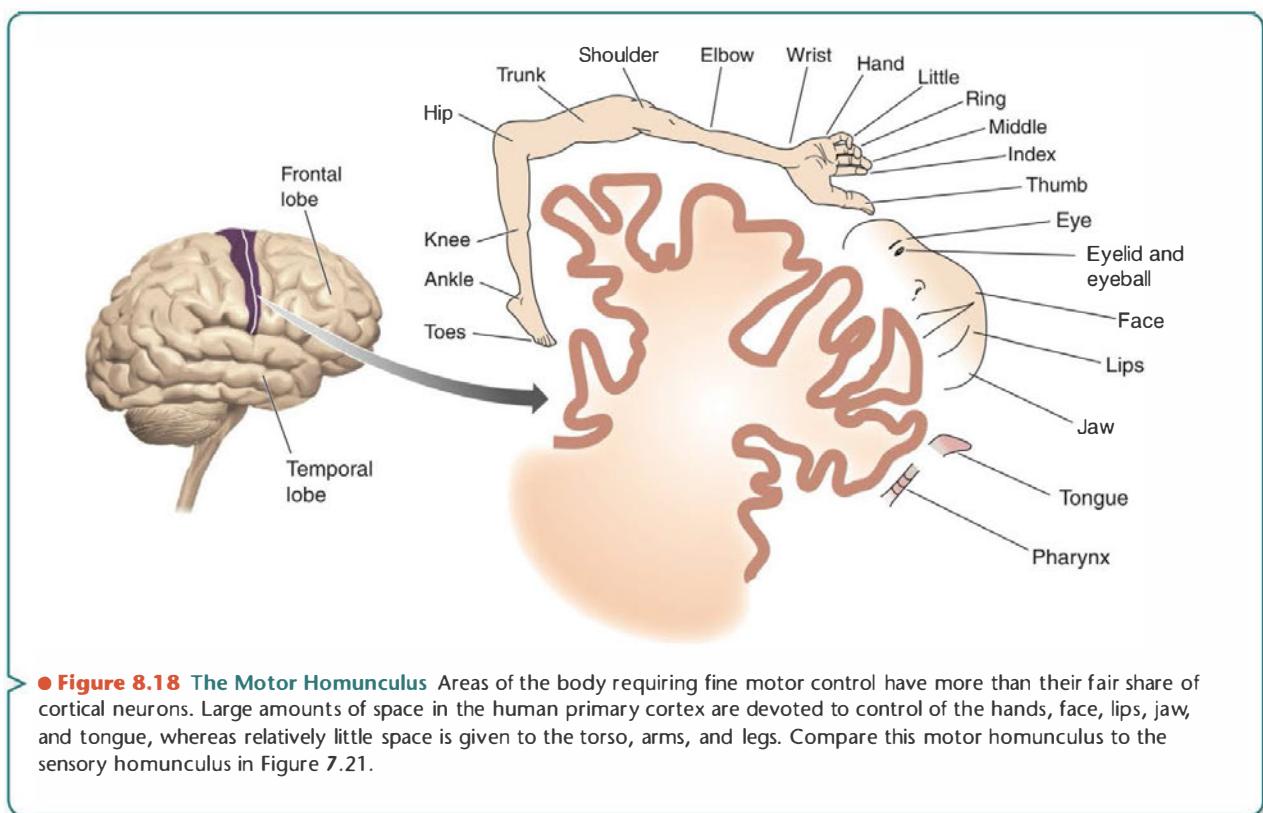
Cortex located in the precentral (before the central sulcus) gyrus has been identified as primary motor cortex (M1), the main source of voluntary motor control. The primary motor cortex not only forms direct connections with the spinal alpha motor neurons, but it also influences the activity of the other motor pathways, including the rubrospinal, tectospinal, and reticulospinal pathways discussed earlier. In the gyrus just rostral to primary motor cortex, additional motor areas were identified by Wilder Penfield (Penfield & Rasmussen, 1950). Penfield named one the **supplementary motor area (SMA)** and the other the premotor area (PMA). The PMA is usually referred to today as **premotor cortex**.

**THE ORGANIZATION OF PRIMARY MOTOR CORTEX** Using stimulation techniques, we can map the primary motor cortex in the precentral gyrus. You can see an example of a motor cortex map, or homunculus, in Figure 8.18. In Chapter 7,

### supplementary motor area

(SMA) Motor area located in the gyrus rostral to the precentral gyrus; involved with managing complex sequences of movement.

**premotor cortex** A motor area located in the gyrus rostral to the precentral gyrus; this area participates in holding a motor plan until it can be implemented; formerly referred to as the premotor area (PMA).



we saw a similar map of the sensory cortex in the parietal lobe. The premotor and supplementary motor areas of the cortex also are organized as homunculi.

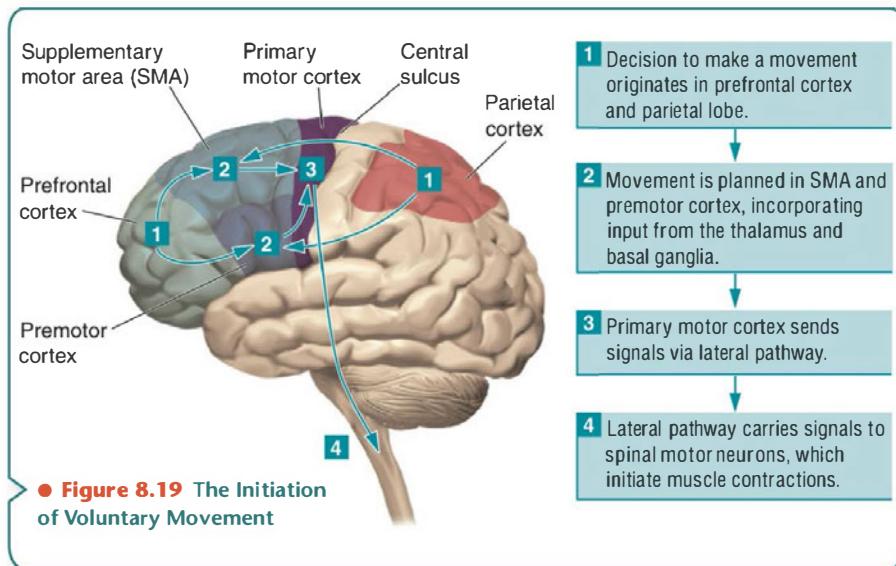
The first thing you might notice is that the homunculus is upside down. Neurons controlling voluntary movement of the head are found in the most ventral portions of the precentral gyrus, whereas neurons controlling the feet are located in the opposite direction. Neurons serving the feet are found where the gyrus has actually curved across the top of the hemisphere into the longitudinal fissure, which separates the two hemispheres.

As we observed in the case of the sensory homunculus (see Figure 7.21), the proportions of the homunculus are quite different from the proportions of our body. Once again, parts of the body that require delicate control are given a larger share of cortical territory. Face, lips, jaw, and tongue are given a great deal of space to manage the fine movements required by speech. Hands also get a disproportionate amount of cortical space, allowing for the many fine movements we need for tool use. In contrast, the torso gets very little space, especially considering the actual size of this body part. There isn't much to do with torsos other than maintain posture, bend, lift, and twist.

**THE INITIATION AND AWARENESS OF MOVEMENT** What happens when we decide to move? You might be thinking that this would be a good time to close your text and take a snack break (which is fine as long as you also decide to come back and reopen the book). We might like to believe that we are consciously aware of our intent to move, but the brain makes a commitment to a choice of movement as many as 10 seconds before we become consciously aware of the decision (Soon, Brass, Heinze, & Haynes, 2008). This delay is needed by higher level systems to prepare for an upcoming movement. It is possible that our conscious awareness of our movement might serve to “interpret,” or make sense out of our own actions, rather than actually guiding or initiating these actions (Gazzaniga, 2011).

Imaging technologies such as PET and fMRI have been used to track the initiation of movement in human volunteers (Deiber, Honda, Ibanez, Sadato, & Hallett, 1999; Roland, 1993). Participants were asked to carry out movements of the fingers from memory. As shown in Figure 8.19, the first areas to show increased activity were the frontal and parietal lobes. These areas might be viewed as the parts of the brain that actually “think” about the movement and its consequences before the movement is initiated.

Within the frontal lobe, several areas appear to be involved with motor control: the supplementary motor area (SMA), premotor cortex, and components of



the anterior cingulate cortex (ACC). Planning of internally generated movements is associated with activity in SMA, while externally initiated and guided movements are associated with activity in the premotor cortex. The ACC appears to coordinate an organism's history of reward with the selection of voluntary movement (Shima & Tanji, 1998).

Activity in the premotor cortex appears to hold a complex plan until it can be implemented. Weinrich and Wise (1982) taught monkeys to move their arms toward a target. The monkeys were given one signal that identified the location of the target, followed by another signal that told the monkey that it was time to respond. Premotor cortical neurons fired in response to the first signal and continued firing until the second signal came on, at which time the monkey initiated the correct arm movement. Premotor cortex activity helps us bridge delays between the planning and initiation of movement.

Premotor cortex and SMA are also the targets of input from the thalamus. We saw previously that the basal ganglia form connections with the thalamus. It is at this point in the sequence that the basal ganglia influence the choice of intentional behaviors. If input from the thalamus to the premotor cortex and SMA is not overly inhibited by the basal ganglia, the sequence will continue. If the input from the thalamus is inhibited, the sequence will be abandoned.

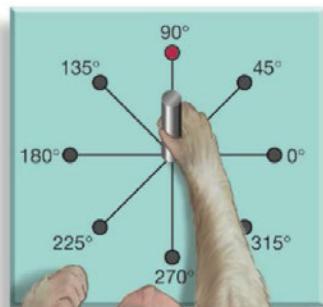
Premotor cortex and SMA activation is followed by activation in the primary motor cortex. Inputs from the premotor cortex, SMA, and the thalamus converge on very large cells located in Layer V of the primary motor cortex. These cells are referred to as **pyramidal cells** because of their pyramid shape, and the diameter of their cell bodies can be nearly 0.1 mm (0.004 in). Pyramidal cell axons are an important source of input to the brainstem and to spinal motor neurons. Once the motor cortex is activated, information flows down the lateral pathways to the spinal cord, either directly or through the red nucleus of the midbrain. The axons from the lateral pathways then synapse on the alpha motor neurons, which initiate the muscle contractions needed to carry out the descending commands.

What exactly is the primary motor cortex telling the alpha motor neurons in the spinal cord to do? Neurons in the primary motor cortex produce action potentials between 5 and 100 msec prior to any observable movement. This suggests that primary motor cortex commands are transmitted and implemented by the alpha motor neurons. Primary motor cortex input also transmits information about the amount of force needed for a movement (picking up a feather or a bowling ball), but it is up to the alpha motor neurons to adjust their rate of firing and extent of their recruitment to fit the requirements of this command.

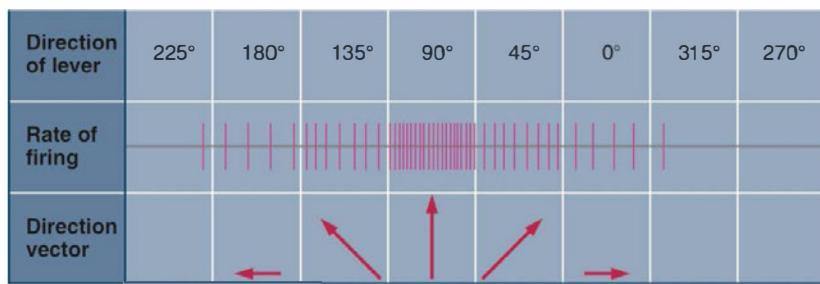
**THE CODING OF MOVEMENT** In the case of vision, single-cell recording techniques can be used to discover the precise type of stimulus to which a particular cell will respond. Similar efforts to identify the responsibilities of single neurons in the primary motor cortex have produced curious results. Even though our movements are generally very precise, individual primary motor cortex neurons seem to be active during a wide range of movements. This finding led Apostolos Georgopoulos and his colleagues (Georgopoulos, Taira, & Lukashin, 1993) to propose the idea that movement is encoded by populations of motor neurons rather than by single cells.

In this research, monkeys were observed as they moved an arm in one of eight possible directions. Single cells in the monkey's primary motor cortex responded to a wide range of movement, but they responded most vigorously to a single preferred direction. The best predictor for whether the monkey's arm would move in a particular direction was a population vector, or the sum of the activity of the entire population of neurons in the area being observed. An example of the population vectors computed by Georgopoulos et al. (1993) is shown in Figure 8.20. This finding predicts that large numbers of motor cortex cells, rather than a small group, should be active during any type of movement. It also suggests that the direction of a movement represents the averaging of all inputs from the entire population of neurons.

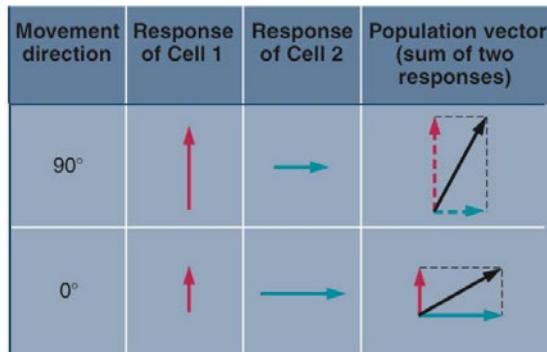
**pyramidal cell** A large, pyramid-shaped neuron found in the output layers (Layers III and V) of the cerebral cortex, including primary motor cortex.



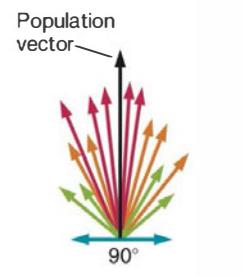
(a) The Monkey and Apparatus



(b) Responses of a Single Cell



(c) Individual Direction Vectors and Population Vectors from Two Neurons



(d) Vector Representing Summed Activity from a Population of Neurons

**► Figure 8.20 The Direction of Movement Is Encoded by Populations of Neurons** (a) Monkeys were trained to move a joystick in response to a small light moving around a circle. (b) Although single-cell recordings indicated that neurons responded most vigorously to a preferred direction, they also indicated that cells would respond to directions that varied as much as 45 degrees from the preferred direction. (c) Responses of single cells can be combined into population vectors. (d) The summed response of each population of neurons accurately predicts the direction of the monkey's movement. In other words, the population of cells essentially tallies up individual cell "votes" on the best direction to move.

**MIRROR NEURONS** Not only do motor neurons code for movement, but they might also help us understand the behavior of others. **Mirror neurons** are special neurons in the premotor cortex and in the inferior parietal lobule that fire whenever an individual carries out an action such as reaching or simply watches another individual carry out the same act (Di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992). When discussing mirror functions in humans, we generally refer to them as mirror systems rather than mirror neurons (Fabbri-Destro & Rizzolatti, 2008). Research in monkeys has been done with single cell recording, while demonstration of mirror systems in humans has used transcranial magnetic stimulation, EEG, MEG, and brain imaging (PET, fMRI; see Chapter 1).

Although the exact functions of mirror neurons remain unknown, mirror neurons might form the basis for imitation, empathy, and theory of mind (TOM), the ability to predict and understand the thoughts of others (Fogassi et al., 2005). Mirror neuron activity might allow us to simulate others' thoughts subconsciously by literally putting ourselves mentally in another person's shoes (Gazzola, Aziz-Zadeh, & Keysers, 2006). Although some researchers suggested that mirror neuron function is disturbed in individuals with autism spectrum disorder (see Chapter 16),

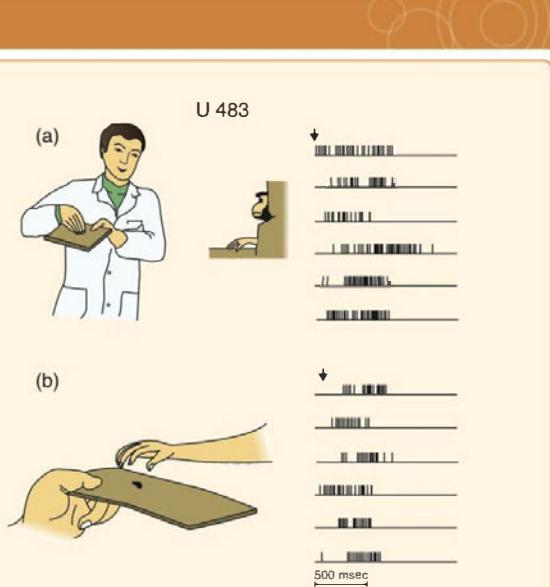
**mirror neuron** A special motor neuron that responds to a particular action, whether that action is performed or simply observed.

## Connecting to Research

### MIRROR NEURONS

**D**uring a routine single-cell recording experiment using a rhesus monkey, a group of Italian scientists led by Giacomo Rizzolatti noticed something rather peculiar. Certain neurons located in the inferior premotor cortex seemed to become especially active when the monkey performed certain actions, such as reaching for a peanut, and also became active when one of the researchers picked up the peanut (see ● Figure 8.21). In other words, the neurons seemed to be encoding “reaching for food” regardless of who or what did the reaching. The researchers dubbed these neurons “mirror neurons” (Di Pellegrino et al., 1992).

While we might take this finding for granted today, at the time of this report in 1992, the existence of mirror neurons was somewhat surprising. Previously, neuroscientists had believed that premotor neurons retrieved movements in response to sensory stimuli. In other words, when the monkey sees a peanut (visual stimulus), one appropriate motor action is to pick up the peanut. However, the mirror neurons apparently encoded the meaning of gestures by others toward the stimulus as well. The authors suggest that for a social species like the monkey, understanding actions performed by other monkeys could provide important guidance for the selection of appropriate movements.



● **Figure 8.21 Recording a Mirror Neuron** Recordings of a single cell (U 483) in response to seeing an experimenter reach for a peanut (a) and in response to the monkey reaching for a peanut (b) are shown on the right. The arrow indicates the onset of reaching behavior.

Data from Di Pellegrino, Fadiga, Fogassi, Gallese, and Rizzolatti (1992).

other researchers argue that a “broken mirror” explanation of social deficits in autism spectrum disorder is overly simplistic (Fan, Decety, Yang, Liu, & Cheng, 2010). Evidence has shown that individuals with autism spectrum disorder show normal basic motor imitation, suggesting that other mechanisms underlie emotion understanding and empathy (Schulte-Rüther et al., 2016).

### INTERIM SUMMARY 8.2

#### Summary Table: Central Motor Systems

Structure or Pathway	Location	Principal Connections	Functions
Lateral pathways	Lateral spinal cord	Motor cortex and red nucleus to spinal motor neurons	Voluntary fine movements of hands, feet, and outer limbs
Ventromedial pathways	Ventromedial spinal cord	Brainstem to spinal motor neurons	Maintain posture; carry out reflexive responses to sensory input

(continued)

Structure or Pathway	Location	Principal Connections	Functions
Cerebellum	Hindbrain	Spinal motor neurons, forebrain motor systems	Sequencing of complex movements, muscle tone, balance, and coordination
Basal ganglia	Forebrain	Motor cortex, thalamus	Choice and initiation of voluntary movements
Primary motor cortex	Precentral gyrus of the frontal lobe	Other cortical areas, basal ganglia, brainstem, spinal motor neurons	Initiation of voluntary movements
Premotor cortex	Rostral to the primary motor cortex	Thalamus, primary motor cortex	Managing movement strategies
Supplementary motor area (SMA)	Rostral to the primary motor cortex	Thalamus, primary motor cortex	Managing movement strategies
Anterior cingulate cortex	Rostral portion of the gyrus dorsal to the corpus callosum	Red nucleus and spinal motor neurons	Selection of voluntary movement based on prior reward

### Summary Points

- Monosynaptic reflexes, such as the myotatic reflex, require the interaction of only one sensory and one motor neuron. Polysynaptic reflexes involve more than one synapse. **(LO4)**
- The lateral pathways connect the primary motor cortex and red nucleus with the spinal motor neurons and are responsible for voluntary movements. The ventromedial pathways originate in the brainstem and are responsible for reflexive movements. **(LO5)**
- The cerebellum is involved with the timing and sequencing of complex movements. The basal ganglia form complex loops with the cortex and the thalamus and serve as a gate or filter for intentional activity. **(LO5)**
- Cortical areas involved in the control of voluntary movement include the primary motor cortex, the supplementary motor area (SMA), the premotor cortex, and the anterior cingulate cortex (ACC). **(LO5)**
- The initiation of voluntary movement is correlated with sequential activity in the prefrontal cortex and parietal cortex, followed by activity in the premotor cortex and SMA, and finally by activity in the pyramidal cells of the primary motor cortex. These primary motor neurons control movement as a function of cell population activity rather than as a function of single-cell activity. **(LO5)**
- Special motor neurons known as mirror neurons are active when an individual either performs a movement or sees another individual perform the same movement, forming a basis for the understanding of the behavior of others. **(LO5)**

### Review Questions

- What motor functions are carried out by reflexive or involuntary processes?
- What steps lead to the initiation of a voluntary movement?

## Disorders of Movement

We can learn a great deal about the neural control of movement by observing what goes wrong when the system is damaged. Because movement disorders obviously cause enormous human suffering, our further understanding might also lead to more effective treatments.

### Toxins

A variety of toxic substances interfere with movement. Many of these toxins affect the neurochemical ACh, which is used by alpha motor neurons to communicate with muscle fibers at the neuromuscular junction. Toxins that are cholinergic agonists boost the activity of ACh at the neuromuscular junction, affecting muscle tone. For example, black widow spider venom overstimulates the release of ACh from the alpha motor neuron, causing the muscles to spasm in painful convulsions (see Chapter 4). In unusually severe cases, the alpha motor neuron runs out of ACh and can no longer signal the muscles to contract, leading to paralysis and possibly death.

Although large doses of cholinergic agonists are capable of producing death, cholinergic antagonists are generally far more dangerous and potent. These substances paralyze muscles, including those required for respiration. We observed the deadly actions of the cholinergic antagonists curare and botulinum toxin in Chapter 4. The venom of the Taiwanese cobra also binds tightly to the nicotinic ACh receptor and is nearly impossible to dislodge, leading rapidly to paralysis and death.

Sarin, a synthetic chemical used as a bioweapon, is a colorless, odorless liquid that can evaporate into a gas (see Figure 8.22). Sarin acts as a neurotoxin by inhibiting acetylcholinesterase (AChE), the enzyme that normally breaks down ACh in the synaptic gap. In the presence of sarin, ACh continues to remain active, and muscles are continually stimulated. This produces rapid fatigue and respiratory failure, as new messages from the motor system to diaphragm muscles are unable to produce movement.



Reuters Photographer/Reuters

**Figure 8.22 Neurotoxins Often Affect the Neuromuscular Junction** Sarin was released on several trains by members of the Aum Shinrikyo cult in a Tokyo subway in 1995, killing 12 people and sickening thousands of others. Rescue workers wore special suits for protection against the deadly neurotoxin.

## Myasthenia Gravis

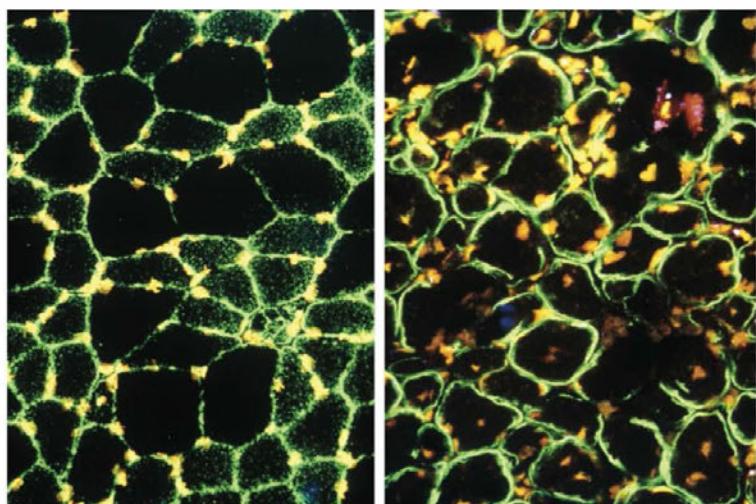
**Myasthenia gravis** results when a person's immune system, for reasons that are not well understood, produces antibodies that bind to the nicotinic ACh receptor (Engel, 1984) or to other proteins associated with the postsynaptic membrane at the neuromuscular junction (Guptill, Soni, & Meriggioli, 2016). Over time, the ACh receptors degenerate and become less efficient, leading to extreme muscle weakness and fatigue. The degeneration usually affects the muscles of the head first, resulting in droopy eyelids and slurred speech. As the degeneration proceeds, the patient might experience difficulty swallowing and breathing.

Myasthenia gravis is typically treated with medications that suppress the immune system. This approach slows the production of the troublesome antibodies. Another approach is to administer medications that inhibit AChE, the enzyme that deactivates ACh at the synapse. Because the ability of AChE to break down ACh is reduced, more ACh remains active in the synaptic gap. Fortunately, most cases of myasthenia gravis have a positive long-term prognosis when these treatment regimens are adhered to carefully (Guptill et al., 2016).

## Muscular Dystrophy

**Muscular dystrophy** is not a single disease but, rather, a group of inherited diseases characterized by progressive muscle degeneration. These diseases are caused by abnormalities involving the protein dystrophin, which makes up part of the muscle fiber membrane. Dystrophin protects the membrane from injuries due to the force that occurs during normal movement. Muscular dystrophy is a sex-linked disorder, typically affecting males, because the gene responsible for encoding dystrophin is located on the X chromosome (see Chapter 5).

In the most severe type of muscular dystrophy, Duchenne muscular dystrophy, dystrophin is not produced at all (see Figure 8.23). Faulty dystrophin is present in cases of Becker muscular dystrophy. Without the protective action of dystrophin, muscles are damaged during normal activity. The muscle turns to scar tissue before



Pandit Landmann/Science Source

**● Figure 8.23 Cellular Changes in Muscular Dystrophy** Healthy muscle cells are shown on the left, while cells unable to produce the protein dystrophin are shown on the right. Recent breakthroughs in gene therapy might eventually provide relief from some forms of muscular dystrophy.

**myasthenia gravis** An autoimmune condition caused by the degeneration of acetylcholine (ACh) receptors at the neuromuscular junction, resulting in muscle weakness and fatigue.

**muscular dystrophy** A group of diseases characterized by extreme muscle development followed by muscle wasting, due to abnormalities in the protein dystrophin.

# Building Better HEALTH

## WHEN VACCINATION IS NOT ENOUGH



In the United States, we take it for granted that children will be immunized against polio and that the risk of contracting this disease is virtually zero. Other parts of the world are not so fortunate. Although vaccines are effective and available, war, poverty, and distrust of vaccination makes total eradication of polio challenging.

A key strategy for targeting vaccination efforts is careful surveillance (Global Polio Eradication Initiative, 2017). India has been successful at reducing polio rates by instructing parents and health care providers to report any evidence of paralysis, especially in children, that

might be polio. In response to such reports, public health officials are able to test for the presence of the virus and plan vaccination programs accordingly. Egypt has been even more proactive. Due to its past history as a problem area for wild poliovirus strains, Egyptian scientists collect sewage samples and test for the virus even in the absence of any reports of paralysis in the population. Individuals carrying the virus are relatively likely to remain free from symptoms, although they can pass the virus to others. Proactive detection of the virus in 2012 led to waves of vaccination that kept the country polio free.

gradually wasting away. This disorder eventually reduces mobility and results in a shortened life span.

Currently, there are no effective treatments for muscular dystrophy. However, gene therapy, in which faulty dystrophin genes are replaced with functioning genes, has produced improvement in animal models of muscular dystrophy (Shin et al., 2013). Researchers are getting closer to conducting human trials using this approach (Nelson et al., 2016).

### Polio

**Polio**, or poliomyelitis, occurs only in human beings and is caused by a contagious virus that specifically targets and destroys spinal alpha motor neurons. *Polio* comes from the ancient Greek word for “gray” and refers to damage to the gray matter of the spinal cord. Symptoms can include muscle weakness and paralysis, usually of the legs. If the infection affects higher levels of the CNS, breathing can be compromised. This situation leads to the patient’s reliance on assisted respiration, such as an “iron lung.”

Following the development of effective vaccines, the last wild variety case of polio in the United States was reported in 1979, and worldwide eradication of the virus seems imminent. However, cases continue to occur in areas affected by war and extreme poverty, such as Afghanistan and Pakistan (Morales, 2016).

### Accidental Spinal Cord Injury (SCI)

The spinal cord can be accidentally damaged when the protective vertebrae surrounding the cord are broken and compress or sever the cord itself. These tragic accidents, such as that suffered by Hollywood actor Christopher Reeve (1952–2004) while horseback riding in 1995 and by approximately 8,000 Americans per year in automobile accidents, generally result in permanent paralysis of the muscles served by neurons below the level of damage. Approximately 250,000 people with accidental spinal cord injury live in the United States alone (Curtis, Gabel, Marsala, & Ciacci, 2016).

If the damage occurs in the cervical, or neck, region of the spinal cord, the person will experience quadriplegia, or the loss of movement in both arms and legs. If the damage occurs in the lumbar region of the lower back, the person will experience paraplegia, the loss of movement in the legs.

Until his death in 2004, Reeve lobbied for additional research funds for spinal cord damage and its treatment. Although a “cure” is still years away, there is

**polio** A contagious viral disease that attacks the spinal motor neurons, producing paralysis.



Simon Bruty/Sports Illustrated/Getty Images

**Figure 8.24 Emergency Treatment of Spinal Injury Is Improving** In 2007, Buffalo Bills tight end Kevin Everett sustained a serious neck injury that left him paralyzed on the field. After his physicians performed a number of controversial treatments, including cooling his body, Everett made a remarkable recovery. Although treatment of existing spinal injury is still in the future, emergency care of spinal injuries is reducing the severity of the damage.

progress. A combination of electrical stimulation, serotonin and dopamine agonists, and locomotor training (active walking as opposed to passive treadmill work, going up stairs, and avoiding obstacles) restored voluntary movement in rats with paralyzed hind legs (Silva, Sousa, Reis, & Salgado, 2014; van den Brand et al., 2012). Treatments designed to enhance axon regeneration and sprouting in animal models appear encouraging (Ruschel et al., 2015). Human trials have indicated that patients tolerate injection of stem cells into the injury site of the spinal cord without adverse effects, although benefits have not yet been reported (Curtis et al., 2016).

Emergency treatment immediately following an injury is becoming more successful in minimizing paralysis. In 2007, NFL football player Kevin Everett sustained a spinal injury during a game that his physician described as “catastrophic.” Everett’s doctors used steroids, surgery, and a controversial cooling technique to prevent swelling in his spinal cord (Cappuccino, 2008). Against all odds, Everett eventually regained nearly normal movement (see Figure 8.24).

### Amyotrophic Lateral Sclerosis (ALS; Lou Gehrig’s Disease)

**Amyotrophic lateral sclerosis (ALS)** is also known as Lou Gehrig’s disease, after the outstanding baseball player of the 1930s whose life was ended by the disease. Physicist Stephen Hawking also is diagnosed with ALS. ALS results from the degeneration of motor neurons in the spinal cord, brainstem, and motor cortex. The muscles served by these motor neurons degenerate when their input ceases. Patients experience muscle weakness, and about 15 percent experience frontotemporal dementia (see Chapter 15). Most patients die within 3 to 5 years after the onset of symptoms, and only 4 percent of

## Behavioral Neuroscience GOES TO WORK

### PHYSICAL THERAPY



Management of the remediation of movement disorders is primarily the domain of the physical therapist (PT). Physical therapists assess movement issues and administer remediation in the form of special exercises, education, manipulation, and other similar techniques.

Although the idea of treating movement problems with exercise dates back to ancient times, events occurring in the twentieth century promoted the rapid acceptance of this practice. Polio outbreaks at the beginning of the twentieth century led to the use of massage and remedial exercise as treatments. Soldiers recovering from injuries in World Wars I and II benefited from physical therapy techniques. Today, physical therapists not

only treat patients with existing movement disorders but also attempt to prevent the development of movement disorders in vulnerable populations.

Physical therapists typically hold either a Doctor of Physical Therapy (DPT) or Master of Physical Therapy (MPT) degree and are licensed on a state-by-state basis in the United States (American Physical Therapy Association [APTA], 2017). The course of study is broad, from biomechanics to neuroscience to psychology and communication. Neuroscience, and in particular its contributions to the understanding of pain (see Chapter 7), has played an increasingly important role in the physical therapy profession.

patients survive longer than 10 years after diagnosis. Hawking's more than 50 years as an ALS patient is highly unusual. Rates of ALS have increased by 50 percent worldwide over the last 50 years (Donati, Demory, & Arnal, 2014).

The causes of ALS are still somewhat mysterious, but scientists are beginning to identify candidate genes that predict risk of the disease. Approximately 5 to 10 percent of the cases run in families. About 15 to 25 percent of patients with familial ALS (FALS) had an abnormal version of a gene known as SOD-1 (Rosen et al., 1993). SOD-1 codes for superoxide dismutase copper/zinc, a key enzyme in oxidative metabolism. More than 100 mutant variations of SOD-1 have been identified in patients with FALS (Selverstone, 2005). The mutant forms of SOD-1 cause damage to motor neurons in multiple ways, including interference with the normal functions of mitochondria (Hervias, Beal, & Manfredi, 2006). As mentioned previously, some individuals are diagnosed with both ALS and frontotemporal dementia (FTD). An analysis of families in which members had either ALS or FTD identified abnormalities in the C9ORF72 gene on chromosome 9 (DeJesus-Hernandez et al., 2011). Intermediate numbers of codon repeats in ATXN2, above normal but below those associated with another motor condition—spinocerebellar ataxia, predicted risk for ALS (Sproviero et al., 2017). Abnormal RNA metabolism (Robberecht & Philips, 2013) and excess glutamate activity (Foran & Trotti, 2009) have also been implicated as causes of ALS.

Currently, there are no “cures” for people with the disease (see Figure 8.25). Two medications have been approved to treat ALS in the United States. Riluzole reduces the activity of glutamate in the brain and extends life an average of two to three months (Miller, Mitchell, & Moore, 2012). Edaravone, initially developed to treat stroke, appears to act as an antioxidant and slows loss of function in some patients with ALS. Research continues in the effort to identify effective treatments. In particular, researchers are investigating the possible use of stem cell therapies (Lunn, Sakowski, & Feldman, 2014). The 2014 social media phenomenon of the ALS “ice bucket” challenge raised more than \$115 million in one 8-week period, accelerating research into potential cures.

## Parkinson’s Disease

**Parkinson’s disease** is characterized by a progressive difficulty in all movements, muscular tremors in the resting hand, and frozen facial expressions. These symptoms were first observed and described by the English physician James Parkinson in the early 1800s.

Patients with Parkinson’s disease experience enormous difficulty initiating voluntary movements, such as standing up from a chair. The disease produces a characteristically stooped posture. Patients’ reflexive movements are also impaired, and they fall easily if they lose their balance. Parkinson’s is often associated with drops in blood pressure, dizziness, and other symptoms of autonomic nervous system disorder



ALS Residence Initiative [ASRI]

**Figure 8.25 Steve Saling and ALS Residences** When Steve Saling, a successful landscape architect, learned that he had ALS at age 38, he went to a conference to learn about his future living options. He connected with Barry Berman of the Leonard Florence Center for Living in Boston. Steve used his design skills to help build a facility that allowed residents the most independence possible. The facility uses PEAC, a comprehensive automation system that opens doors and operate elevators and other equipment using any wireless device.

**amyotrophic lateral sclerosis (ALS)** A disease in which motor neurons of the spinal cord, brainstem, and motor cortex progressively deteriorate, leading to death.

**Parkinson’s disease** A degenerative disease characterized by difficulty in moving, muscular tremors, and frozen facial expressions.

(Goldstein, Holmes, Dendi, Bruce, & Li, 2002). Eventually, the condition leads to premature death. Normally, Parkinson's disease affects people after the age of 50 years. Women enjoy a slight protection from Parkinson's disease compared to men, possibly due to higher striatal dopamine levels supported by estrogen activity (Haaxma et al., 2007; Shulman, 2007).

The direct causes of Parkinson's disease are quite clear. This disease occurs when the dopaminergic neurons of the substantia nigra in the brainstem begin to degenerate. As we discussed previously, the substantia nigra forms close connections with the basal ganglia in the cerebral hemispheres. The end result of degeneration in the substantia nigra is a lack of typical dopaminergic activity in the basal ganglia. Because the basal ganglia are intimately involved with the production of voluntary movements, it should come as no surprise that the patients show great difficulties in voluntary movement.

The factors responsible for the degeneration of the substantia nigra remain unknown. It is likely that genetic vulnerabilities play a role, but only about 15 to 25 percent of people with Parkinson's disease report having a relative with the condition. Genetic mutations in 15 genes directly cause some cases and increase risk factors in others (Bonifati, 2014; Singleton, Farrer, & Bonifati, 2013). In early-onset cases, genes encoding a substance known as alpha synuclein are abnormal (Halliday & McCann, 2008). As a result, alpha synuclein forms filaments within neurons that interfere with axonal transport. This process might account for the degeneration of the dopaminergic axons originating in the substantia nigra.

Other cases appear to result from exposure to environmental toxins. Support for this hypothesis originated from an unfortunate accidental experiment involving young heroin addicts who suddenly developed symptoms of Parkinson's disease (Langston, 1985). The addicts had shared a homemade synthetic heroin, which contained a chemical known as MPTP. When MPTP binds with the enzyme monoamine oxidase, which is found in large quantities in the substantia nigra, it forms a very toxic substance known as MPP<sup>+</sup>. MPP<sup>+</sup> is attracted to the pigment neuromelanin, which is also found in large quantities in the substantia nigra. You may recall that substantia nigra means "black substance" and that the structure is named in part because of its pigmentation. The affinity between MPP<sup>+</sup> and neuromelanin results in the accumulation of MPP<sup>+</sup> in the substantia nigra, leading to degeneration of the neurons.

Obviously, the vast majority of people with Parkinson's disease have never been exposed to heroin, synthetic or otherwise. However, similar toxins that act like MPTP, such as the herbicide paraquat, are present in the environment. People who report having applied insecticides and herbicides on their home gardens or farms are significantly more likely to develop Parkinson's disease than relatives who do not have any direct pesticide exposure (Ritz, Paul, & Bronstein, 2016). Exposure to commonly used solvents such as toluene differentiated between identical twins with and without Parkinson's disease (Goldman et al., 2012). These toxins bind to the neuromelanin of the substantia nigra, which initially has a protective effect. However, the neuromelanin appears to release the toxins under certain circumstances, allowing them to produce damage (Karlsson & Lindquist, 2013).

One of the strangest findings in the quest to understand Parkinson's disease is the fact that drinking caffeinated coffee reduces the odds of developing the disease (Chuang et al., 2016). Men who didn't drink any coffee at all were four to five times more likely to develop Parkinson's disease than were the heaviest coffee drinkers, who drank about five cups of coffee per day. Animal research suggests that caffeine might play a role in preventing neural degeneration resulting from a number of conditions, such as stroke, Parkinson's disease, and Alzheimer's disease (Sonsalla et al., 2012), but this should not inspire you to head immediately for your nearest coffee shop. These correlational data do not allow us to say that reduced risk for Parkinson's disease was *caused* by coffee consumption (Wierzejska, 2016). It is possible that the physiology of people who can tolerate larger amounts of caffeine also makes them resistant to Parkinson's disease.

Not only does caffeine have a potential protective effect against the development of Parkinson's disease, but investigators have also explored the possibility of its use as a

treatment (Ferreira et al., 2016; Rivera-Oliver & Díaz-Ríos, 2014). Caffeine and other substances that block adenosine receptors (see Chapter 4) appear to improve movement and wakefulness in both human patients and animal models of the disease.

The traditional treatment for Parkinson's disease is the medication levodopa, or l-dopa. L-dopa is a precursor in the synthesis of dopamine (see Chapter 4), so additional l-dopa should help the neurons in the substantia nigra manufacture more of the neurochemical. L-dopa crosses the blood-brain barrier, whereas dopamine itself does not. However, l-dopa loses its effectiveness as the numbers of substantia nigra neurons decrease and feedback loops inhibit the further production of dopamine. Dyskinesia, or involuntary movements, eventually are experienced by the vast majority of patients treated with l-dopa (Bastide et al., 2015). Because l-dopa affects all dopaminergic systems, not just those originating in the substantia nigra, it produces multiple undesirable side effects. Increasing overall dopamine activity often results in psychotic behavior, including hallucination and delusional thinking (Carey, Pinheiro-Carrera, Dai, Tomaz, & Huston, 1995). As we noted in Chapter 4, the increased activity of dopaminergic neurons associated with schizophrenia and with the abuse of dopamine agonists such as amphetamine can produce similar types of psychotic behaviors.

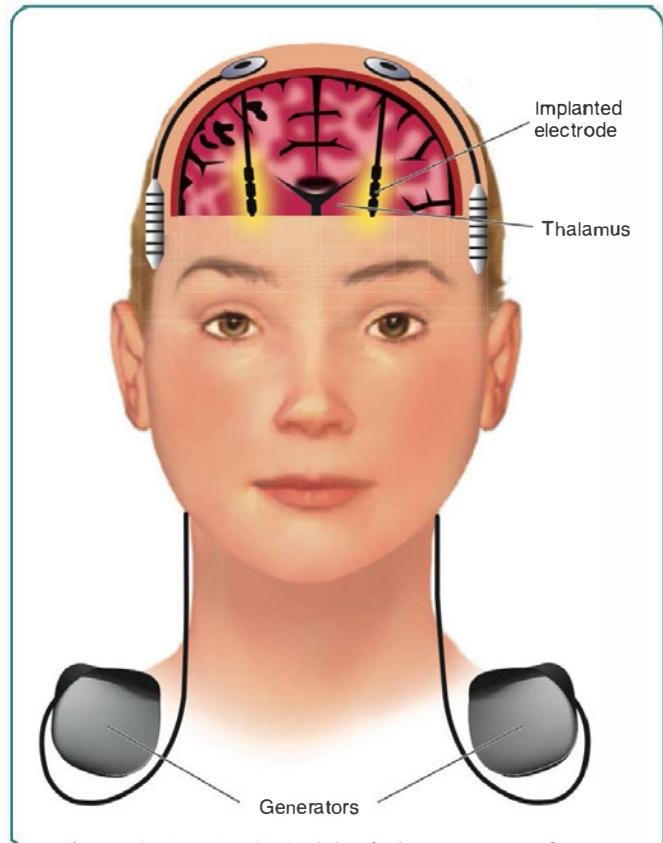
Surgery has also been used to treat advanced cases of Parkinson's disease. In a procedure known as a pallidotomy, a part of the globus pallidus of the basal ganglia is destroyed (Young, Li, Vermeulen, Clayton, & Hesselgesser, 2014). Another surgical approach to Parkinson's is the thalamotomy, in which a small area of the thalamus is destroyed (Elias et al., 2013). Pallidotomy and thalamotomy do not require invasive surgery but instead are conducted with ultrasound and gamma knife technologies. These procedures, when successful, generally reduce unwanted muscle tension and tremor. An alternative to surgery is electrical stimulation, shown in Figure 8.26 (Little et al., 2013; Odekerken et al., 2013). Wires are surgically implanted in the thalamus, globus pallidus, or subthalamic nucleus and are connected to two pulse generators each implanted near the patient's collarbone. The generators maintain a steady electrical signal that interferes with signals that lead to tremor.

Transplantation of stem cells has restored movement in animal models (Rhee et al., 2011). Gene therapy might also lead to effective treatments, as initial experiments in human patients appear promising and relatively safe (Palfi et al., 2014). More work needs to be done before this type of treatment is widely available, but the outlook is optimistic.

## Huntington's Disease

**Huntington's disease** is a genetic disorder that usually strikes in middle age and produces involuntary, jerky movements. As the disease progresses, cognitive symptoms such as depression, hallucination, and delusion occur. Fifteen to 20 years after the onset

**Huntington's disease** A genetic disorder beginning in middle age that results in jerky, involuntary movements and progresses to psychosis and premature death.



**Figure 8.26 Deep Brain Stimulation Treatment for Parkinson's Disease** To date, thousands of patients with Parkinson's disease have been treated with this electricity-based technique that requires the insertion of two generators under the skin, usually near the collarbone. The generators, each about two inches in diameter, emit tiny electrical pulses that pass along wires, also under the skin, through electrodes implanted in select areas of the brain. Some patients experience a tingling sensation, but typically the stimulation pulses go unnoticed.

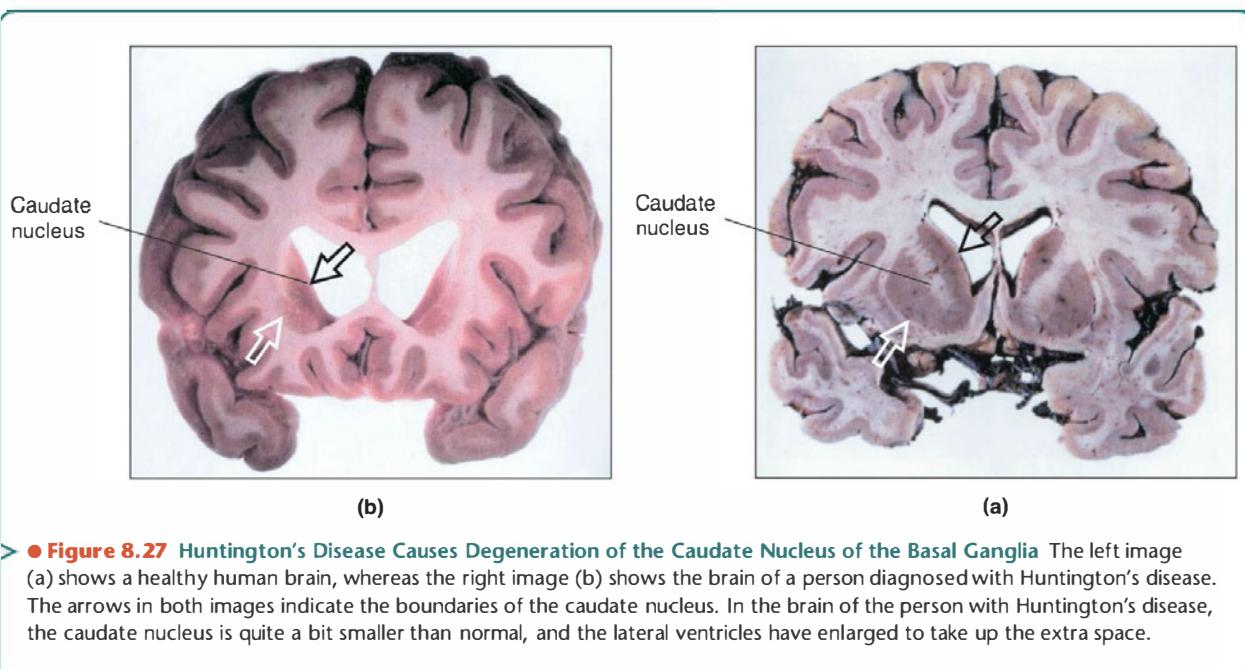
Images provided courtesy of Sanjiv Sam Gambhir, M.D., Ph.D., Stanford University.

of symptoms, the patient dies. There is no known cure for this disease, which affects about one person out of 1,000. The disease was first identified by George Huntington, a doctor on Long Island, in 1872. Eventually, Huntington's original patients were found to be part of one extended family going back to a pair of brothers who came to America from England in 1630. A number of family members were burned as witches in Salem in 1693, probably due to the odd behaviors associated with the disease (Ridley, 1999).

The cause of Huntington's disease is simple and well understood. The Huntington gene on Chromosome 4, named after George Huntington, encodes the brain protein huntingtin. At the end of the Huntington gene is a codon, or sequence that encodes an amino acid, that can repeat between 6 and more than 100 times (see Chapter 5). Most people have between 10 and 15 repeats of this sequence. A person having fewer than 35 repeats will remain healthy, but a person with 39 or more repeats will develop Huntington's disease. Higher numbers of repeats are correlated with an earlier onset of symptoms (Gusella et al., 1996). To make matters worse, this gene is one of the few examples of a dominant gene for a disease. It doesn't help at all to have one normal Huntington gene. If you have one abnormal gene, you will have the disease. If one of your parents has the disease, then you have a 50 percent chance of developing the disease yourself. The identification of the Huntington gene in 1993 made genetic testing possible for those who are at risk for the disease (Gusella & MacDonald, 1993).

How does the mutant huntingtin protein produce the symptoms of Huntington's disease? The version of the huntingtin protein produced by the abnormal gene appears to accumulate, particularly in the cells of the basal ganglia. This accumulation of abnormal proteins forms a sticky lump in the cells and triggers cellular suicide (apoptosis; see Chapter 5). A comparison of the basal ganglia from a healthy person and a patient with Huntington's disease is shown in Figure 8.27.

Currently, there are no effective treatments for Huntington's disease, in spite of the fact that the responsible gene has been recognized for quite a few years. Brain imaging shows that changes in brain volume begin to occur long before the appearance of symptoms leads to diagnosis (Ross et al., 2014). Investigation of these changes might lead to treatments that delay the onset of symptoms or slow the progression of the disease. For example, the administration of high doses of creatine has been explored in patients whose status is premanifest (genetically tested to show they have the disease, but no



**► Figure 8.27 Huntington's Disease Causes Degeneration of the Caudate Nucleus of the Basal Ganglia** The left image (a) shows a healthy human brain, whereas the right image (b) shows the brain of a person diagnosed with Huntington's disease. The arrows in both images indicate the boundaries of the caudate nucleus. In the brain of the person with Huntington's disease, the caudate nucleus is quite a bit smaller than normal, and the lateral ventricles have enlarged to take up the extra space.

symptoms yet) and at risk (not tested but having a parent with the disease) (Rosas et al., 2014). Theoretically, the genetic testing of all offspring of patients with Huntington's disease and their subsequent use of assisted reproduction, selecting out all affected embryos, would end the disease for all time in one generation. But this is a highly complex psychosocial decision that is unlikely to be chosen by all concerned.

### INTERIM SUMMARY 8.3

#### || Summary Table: Major Disorders of the Motor Systems

Disorder	Symptoms	Causes	Treatments
Myasthenia gravis	Muscle weakness, fatigue	Autoimmune damage to the nicotinic ACh receptor	Medications that inhibit the immune system or AChE
Muscular dystrophy	Progressive muscle degeneration	Abnormalities in the gene that encodes for the protein dystrophin	None currently approved; gene replacement and muscle cell replacement under investigation
Polio	Damage to spinal motor neurons leading to mild to severe muscle paralysis	Virus	Prevented by immunization
Accidental spinal cord injury	Paralysis in muscles served by the spinal cord areas below the point of damage	Compression or laceration of the spinal cord	None currently approved; stem cell therapy and methods for promoting axon regrowth are under investigation
Amyotrophic lateral sclerosis (ALS)	General weakness, muscle atrophy, cramps, muscle twitching	Possible link to genetic inheritance	Two approved medications slow the progression of the disease
Parkinson's disease	Progressive difficulty initiating movement	Genetic risk and exposure to toxins	Electrical stimulation, surgical removal of sections of the basal ganglia or thalamus, medication; gene therapy and stem cell implants under investigation
Huntington's disease	Involuntary, jerky movements; depression, hallucinations, delusions	Abnormalities in the gene that encodes for the protein huntingtin	Experimental gene replacement, stem cell implants, and medications under investigation

#### || Summary Points

1. A number of toxins interfere with movement due to their action at the cholinergic neuromuscular junction. (**L06**)
2. Myasthenia gravis is an autoimmune disease that produces extreme muscle weakness and fatigue due to the breakdown of ACh receptor sites at the neuromuscular junction. Muscular dystrophy is a collection of diseases that produces extreme muscular

development followed by muscular degeneration. Polio is a contagious viral disease that attacks spinal motor neurons, causing some degree of paralysis. Accidental spinal injury results in the loss of voluntary movement produced by nerves below the level of injury. (LO6)

3. Amyotrophic lateral sclerosis (ALS) is a degenerative condition resulting from the progressive loss of motor neurons in the spinal cord, brainstem, and motor cortex. (LO6)
4. Parkinson's disease produces difficulty moving, tremor in resting body parts, frozen facial expressions, and reduced heart innervation. (LO6)
5. Huntington's disease is a genetic disorder characterized by involuntary movement and cognitive decline. (LO6)

### Review Questions

1. What effects can toxins have on the neuromuscular junction, and how does this affect movement?
  2. What physical changes underlie the symptoms of Parkinson's disease?
- 

## Chapter Review

### THOUGHT QUESTIONS

1. What advice would you give an older adult who wanted to maintain or improve muscle strength?
2. Why do you think we evolved three types of muscle fibers?
3. Damage or abnormalities in the prefrontal cortex and basal ganglia might be responsible for some impulsive behavior. Using your knowledge of the initiation of movement, explain why abnormalities in these areas might lead to impulsivity.
4. If you had a parent with Huntington's disease, would you undergo the available genetic screening? What factors would influence your decision?
5. At what levels of the motor system do we treat fine motor activities, such as speech and the movement of our fingers and hands, differently from gross motor activities, such as posture?

### KEY TERMS

actin (p. 268)  
alpha motor neuron (p. 272)  
amyotrophic lateral sclerosis (ALS) (p. 294)  
extensor (p. 279)  
extrafusal muscle fiber (p. 274)  
fast-twitch fiber (p. 269)  
flexor (p. 279)  
flexor reflex (p. 279)  
gamma motor neuron (p. 275)  
Golgi tendon organ (p. 276)  
Huntington's disease (p. 297)  
Ia sensory fiber (p. 274)  
Ib sensory fiber (p. 276)  
intrafusal muscle fiber (p. 274)

lateral pathway (p. 281)  
M line (p. 268)  
mirror neuron (p. 288)  
monosynaptic reflex (p. 275)  
motor unit (p. 272)  
muscle fiber (p. 267)  
muscle spindle (p. 274)  
muscular dystrophy (p. 292)  
myasthenia gravis (p. 292)  
myofibril (p. 268)  
myosin (p. 268)  
myotatic reflex (p. 275)  
neuromuscular junction (p. 272)  
Parkinson's disease (p. 295)  
polio (p. 293)

polysynaptic reflex (p. 278)  
premotor cortex (p. 285)  
recruitment (p. 273)  
sarcomeres (p. 268)  
skeletal muscle (p. 266)  
slow-twitch fiber (p. 269)  
smooth muscle (p. 266)  
striated muscle (p. 266)  
supplementary motor area (SMA) (p. 285)  
troponin (p. 269)  
tropomyosin (p. 269)  
twitch (p. 268)  
ventromedial pathway (p. 281)  
Z line (p. 268)