PSYCHOLOGY: THE SCIENCE OF BEHAVIOR, 6/e

Neil R. Carlson, *University of Massachusetts, Amherst*
C. Donald Heth, *The University of Alberta*
Harold Miller, *Brigham Young University*
John W. Donahoe, *University of Massachusetts, Amherst*
William Buskist, *Auburn University*
G. Neil Martin, *Middlesex University*

0-205-47289-3

SAMPLE CHAPTER 4

Allyn & Bacon
75 Arlington St., Suite 300
Boston, MA 02116
www.ablongman.com

4

Biology of Behavior

CHAPTER OUTLINE

The Brain and Its Components

Structure of the Nervous System • Cells of the Nervous System • The Excitable Axon: The Action Potential • Communication with Other Cells: Synapses • A Simple Neural Circuit

The central nervous system consists of the brain and the spinal cord. The peripheral nervous system consists of nerves that connect the central nervous system to sense organs, muscles, and glands. The primary functions of the brain are to control behavior, to process information about the environment, and to regulate the physiological processes of the body. The brain floats in cerebrospinal fluid, enclosed by the meninges, and is protected from many chemicals by the blood–brain barrier. The functions of the brain are accomplished by circuits of neurons (nerve cells), supported by glial cells. Neurons communicate with one another by releasing chemicals called neurotransmitters. The message transmitted from place to place—the action potential—is a change in the electrochemical properties of the neuron. Synapses, the junctions between neurons, are either excitatory or inhibitory: Excitatory synapses increase a neuron's activity, and inhibitory synapses decrease it.

Drugs and Behavior

Effects of Drugs on Synaptic Transmission • Neurotransmitters, Their Actions, and Drugs That Affect Them • *Evaluating Scientific Issues: "Physiological" versus "Psychological" Drug Addiction*

Drugs that affect behavior do so by facilitating or interfering with synaptic transmission. The most important neurotransmitters are glutamate, which has excitatory effects, and GABA, which has inhibitory effects. Other important categories of neurotransmitters include acetylcholine; the monoamines (dopamine, norepinephrine, and serotonin); the peptides; and the cannabinoids. Circuits of neurons that secrete particular neurotransmitters have different effects on behavior; this fact accounts for the effects of drugs that facilitate or interfere with them. Although the side effects of addictive drugs (tolerance and withdrawal symptoms) are important, they are not responsible for addiction. Addiction is caused by the effects of drugs on neurons in the brain that are involved in reinforcement (reward).

Study of the Brain

Experimental Ablation • Visualizing the Structure of the Brain • Measuring the Brain's Activity • Stimulating the Brain's Activity • Altering Genetics • *Biology and Culture: Environmental Effects on Brain Development*

Physiological psychologists study the biological processes of the brain using experimental ablation, electrical and chemical recording or stimulation, and genetic manipulation. The development of scanning devices has revolutionized the study of the living human brain. Research shows that the development of the nervous system is influenced by both genetic and environmental variables. Recent studies have found that new neurons can be produced even in adult brains.

Control of Behavior and the Body's Physiological Functions

Organization of the Cerebral Cortex • Lateralization of Function • Vision • Audition • Somatosensation and Spatial Perception • Planning and Moving • Episodic and Spatial Memory: Role of the Hippocampus • Emotions: Role of the Amygdala • Control of Internal Functions and Automatic Behavior

The cerebral cortex, the outer layer of the cerebral hemispheres, receives sensory information, controls perceptual and learning processes, and formulates plans and actions. The thalamus relays sensory information to the cerebral cortex. Some brain functions are lateralized—controlled primarily by one side of the brain. Each of the four lobes of the brain is involved with specific activities: The occipital lobe and the temporal lobe control seeing; the temporal lobe controls hearing; the parietal lobe controls perception of the body and the space around it; and the frontal lobe controls motor activities, planning, attention to emotionally related stimuli, spontaneous behavior, and speech. The cerebellum helps control rapid, skilled movements, and the basal ganglia help control automatic movements, especially slower ones. The hippocampus plays a critical role in episodic memory and spatial navigation. The amygdala is involved in emotions—especially negative ones. The brain stem and the hypothalamus control species-typical behaviors, such as those involved in eating, drinking, fighting, courting, mating, and caring for offspring. The hypothalamus also regulates internal functions through its control of the autonomic nervous system and the endocrine system. Endocrine glands secrete hormones, which affect physiological functions and behavior.

Miss S. was a 60-year-old woman who had a history of high blood pressure, which was not responding well to the medication she was taking. One evening she was sitting in her reclining chair reading the newspaper when the phone rang. She got out of her chair and walked to the phone. As she did, she began to feel giddy and stopped to hold on to the kitchen table. She had no memory of what happened after that.

The next morning a neighbor, who usually stopped by to have coffee with Miss S., found her lying on the floor, mumbling incoherently. The neighbor called an ambulance, which took Miss S. to a hospital.

Two days after her admission, the neurological resident in charge of her case told a group of us that she had had a stroke in the back part of the right side of the brain. He attached a CT scan to an illuminated viewer mounted on the wall and showed us a white spot caused by the accumulation of blood in a particular region of her brain. (You can look at the scan yourself; it is shown in Figure 4.21.)

We then went to see Miss S. in her hospital room. Miss S. was awake but seemed a little confused. The resident greeted her and asked how she was feeling. "Fine, I guess," she said. "I still don't know why I'm here."

"Can you see the other people in the room?"

"Why, sure."

"How many are there?"

She turned her head to the right and began counting. She stopped when she had counted the people at the foot of her bed. "Seven," she reported. "What about us?" asked a voice from the left of her bed. "What?" she said, looking at the people she had already counted. "Here, to your left. No, toward your left!" the voice repeated. Slowly, rather reluctantly, she began turning her head to the left. The voice kept insisting, and finally, she saw who was talking. "Oh," she said, "I guess there are more of you."

The resident approached the left side of her bed and touched her left arm. "What is this?" he asked. "Where?" she said. "Here," he answered, holding up her arm and moving it gently in front of her face.

"Oh, that's an arm."

"An arm? Whose arm?"

"I don't know." She paused. "I guess it must be yours."

"No, it's yours. Look, it's a part of you." He traced with his fingers from her arm to her shoulder.

"Well, if you say so," she said, sounding unconvinced.

When we returned to the residents' lounge, the chief of neurology said that we had seen a classic example of unilateral (one-sided) neglect, caused by damage to a particular part of the brain. "I've seen many cases like this," he explained. "People can still perceive sensations from the left side of their bodies, but they just don't pay attention to them. A woman will put makeup on only the right side of her face, and a man will shave only half of his beard. When these patients put on a shirt or a coat, they will use their left hand to slip it over their right arm and shoulder, but then they'll just forget about their left arm and let the garment hang from one shoulder. They also don't look at things located toward the left—or even at the left halves of things. Once I saw a man who had just finished eating breakfast. He was sitting in his bed, with a tray in front of him. There was half a pancake on his plate. 'Are you all done?' I asked. 'Sure,' he said. I turned the plate around so that the uneaten part was on his right. He gave a startled look and said, 'Where the hell did that come from?'"

The human brain is the most complex object that we know. As far as our species is concerned, it is the most important piece of living tissue in the world. It is also the only object capable of studying itself. (If it could not do so, this chapter could not exist.) Our perceptions, our thoughts, our memories, and our emotions are all products of our brains. If a surgeon transplants a heart, a liver, or a kidney—or even all three organs—we do not ask ourselves whether the identity of the recipient has been changed. But if a brain transplant were feasible (it isn't), we would undoubtedly say that the owner of the brain was getting a new body rather than the reverse.

The Brain and Its Components

The brain is the largest part of the nervous system. It contains anywhere between 10 billion and 100 billion nerve cells—no one has counted them all—and about as many helper cells, which take care of important support and housekeeping functions. For many decades neuroscientists have known that the brain contains many different types of nerve cells. These cells differ in shape, size, and the kinds of chemicals they produce, and they perform different functions.

To understand how the brain works, we need to understand how individual nerve cells work and how they communicate with one another. Let's look first at the basic structure of the nervous system and at the nature and functions of the cells that compose it.

Structure of the Nervous System

The brain has three major functions: Controlling behavior, processing and retaining the information we receive from the environment, and regulating the body's physiological processes. How does it accomplish these tasks?

The brain cannot act alone. It needs to receive information from the body's sense organs, and it must be connected with the muscles and glands of the body if it is to affect behavior and physiological processes. The nervous system consists of two divisions. The brain and the spinal cord make up the **central nervous system**. The **spinal cord** is a long, thin structure attached to the base of the brain and running the length of the spinal column. The central nervous system communicates with the rest of the body through the **peripheral nervous system,** which consists of **nerves**—bundles of fibers that transmit information to and from the central nervous system. Sensory information (information about what is happening in the environment or within the body) is conveyed from sensory organs to the brain and spinal cord. Information from the head and neck region (for example, from the eyes, ears, nose, and tongue) reaches the brain through the **cranial nerves**. Sensory information from the rest of the body reaches the spinal cord (and ultimately the brain) through the **spinal nerves.** The cranial nerves and spinal nerves also carry information away from the central nervous system. The brain controls muscles, glands, and internal organs by sending messages to these structures through these nerves. (See **Table 4•1**.)

TABLE 4•1 The Major Divisions of the Nervous System

Central Nervous System (CNS)	Peripheral Nervous System (PNS)
Brain	Nerves
Spinal cord	

Figure 4•1 shows an overview of the nervous system. The man's back has been opened and the back part of the vertebral column has been removed so that we can see the spinal cord and the nerves attached to it. The skull has been opened, and a large opening has been cut in the *meninges,* the membranes that cover the central nervous system, so that we can see the surface of the brain.

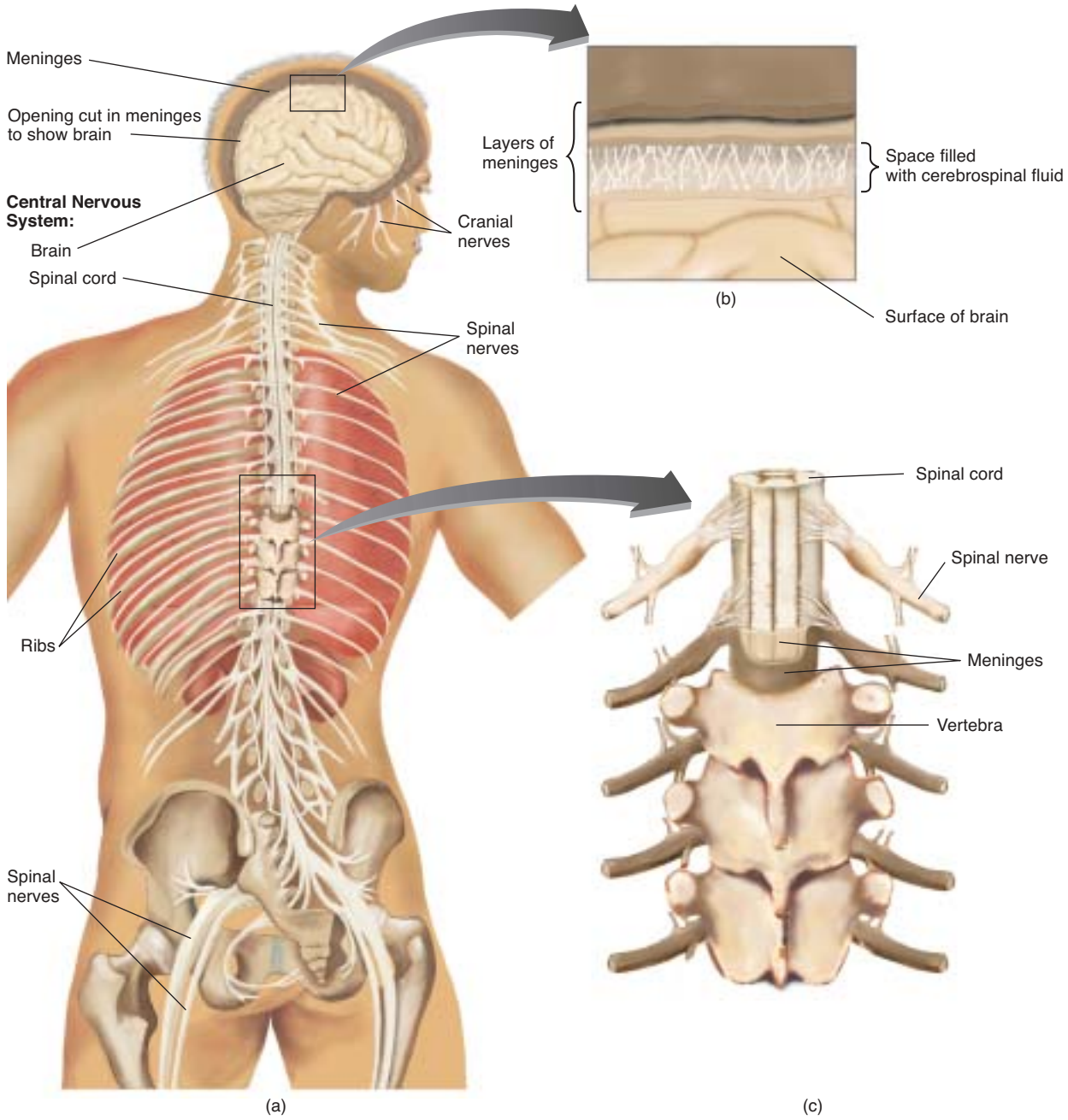
The human brain consists of three major parts: the *brain stem,* the *cerebellum*, and the *cerebral hemispheres*. **Figure 4•2** shows a view of the left side of the brain. The lower portions of the cerebellum and brain stem extend beneath the left cerebral hemisphere; the upper portions are normally hidden. We also see the *thalamus,* a part of the brain described later in this chapter.

If the human brain is cut away from the spinal cord and removed from the skull, it looks as if it has a handle or stem. The **brain stem** is one of the most primitive regions of the brain, and its functions are correspondingly basic—primarily control of physiological functions and automatic behaviors. In fact, the brains of some animals, such as amphibians, consist primarily of a brain stem and a simple cerebellum.

The **cerebellum,** attached to the back of the brain stem, looks like a miniature version of the cerebral hemispheres. The primary function of the cerebellum is to control and coordinate movements; especially rapid, skilled movements. The pair of **cerebral hemispheres** (the two halves of the *cerebrum*) constitutes the largest part of the human brain. The cerebral hemispheres contain the parts of the brain that evolved most recently—and thus are involved in perceptions, memories, and behaviors of particular interest to psychologists. (Refer to Figure 4.2.)

Because the central nervous system is vital to an organism's survival, it is exceptionally well protected. The brain is encased in the skull, and the spinal cord runs through the middle of the spinal column—through a stack of hollow bones known as **vertebrae**. (See Inset C, Figure 4.1.) Both the brain and the spinal cord are enclosed by a three-layered set of membranes called the **meninges**. (*Meninges* is the plural of *meninx,* the Greek word for "membrane." You have probably heard of a disease called *meningitis,* which is an inflammation of the meninges.) The brain and spinal cord do not come into direct contact with the bones of the skull and

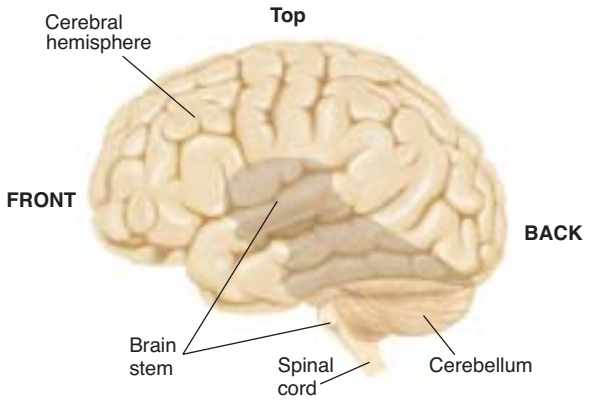
FIGURE 4•1 The central nervous system (brain and spinal cord) and the peripheral nervous system (cranial nerves and spinal nerves).



vertebrae. Instead, they float in a clear liquid called **cerebrospinal fluid (CSF)**. This fluid fills the space between two of the meninges and provides a cushion surrounding the brain and spinal cord, protecting them from being bruised by the bones that encase them. CSF is produced in the **cerebral ventricles,** hollow, fluid-filled chambers located within the brain. (See Inset B, Figure 4.1.)

The brain is protected from chemical assault as well as physical shock. The cells of the body receive water and nutrients from the capillaries, the smallest of the blood vessels. In most of the body, the walls of the capillaries have small openings that let chemicals freely pass from the blood into the surrounding tissue. The brain is an exception: Its capillaries do not have these openings, so fewer substances can pass from the blood to the brain. This impediment to the exchange of chemicals is called the **blood–brain barrier.** Its major function is to diminish the likelihood that toxic chemicals found in what we eat or drink can find their way into the brain, where they might do damage to neurons. Of course, there are many poisons that can affect the brain, so this barrier is not foolproof.

FIGURE 4•2 A view of the left side of the brain, showing its three major parts: brain stem, cerebellum, and cerebral hemisphere. The thalamus is attached to the upper end of the brain stem.



The surface of the cerebral hemispheres is covered by the **cerebral cortex.** (The word *cortex* means "bark" or "rind.") The cerebral cortex consists of a thin layer of tissue approximately 3 millimeters thick. It is often referred to as **gray matter** because of its appearance. It contains billions of nerve cells. (The structure and functions of nerve cells are described in the next section.) It is in the cerebral cortex that perceptions take place, memories are stored, and plans are formulated and executed. The nerve cells in the cerebral cortex are connected to other parts of the brain by bundles of nerve fibers called **white matter,** so named because of the shiny white appearance of the substance that coats and insulates these fibers. **Figure 4•3** shows a slice of the brain. As you can see, the gray matter and white matter are distinctly different.

The human cerebral cortex is very wrinkled; it is full of bulges separated by grooves. The bulges are called *gyri* (singular: *gyrus*), and the large grooves are called *fissures*. Fissures and gyri expand the amount of surface area of the cortex and greatly increase the number of nerve cells it can contain. Animals with the largest and most complex brains, including humans and the higher primates, have the most wrinkled brains and thus the largest cerebral cortexes.

As we saw, the peripheral nervous system consists of the cranial and spinal nerves that connect the central nervous system with sense organs, muscles, internal organs, and glands. Nerves are bundles of many thousands of individual fibers, all wrapped in a tough, protective membrane. Under a microscope, nerves look something like telephone cables, with their bundles of wires. Like the individual wires in a telephone cable, nerve fibers transmit messages through the nerve, from a sense organ to the brain or from the brain to a muscle or gland. (See **Figure 4•4**.)

Cells of the Nervous System

Neurons, or nerve cells, are the elements of the nervous system that bring sensory information to the brain, store memories, reach decisions, and control the activity of the muscles. They are assisted in their task by another kind of cell: the **glia.** Glia (or *glial cells*) get their name from the Greek word for glue. At one time scientists thought that glia simply held neurons—the important elements of the nervous system—in place. They do that, but they also do much more. During development of the brain, some types of glial cells form long fibers that guide developing neurons from their place of birth to their final resting place. Other types of glia manufacture chemicals that neurons need to perform their tasks and absorb chemicals that might impair neurons' functioning. Others form protective insulating sheaths around nerve fibers. Still others serve as the brain's immune system, protecting it from invading microorganisms.

But let's get back to neurons. Neurons are cells that can receive information from other neurons (or from cells in sense organs), process this information, and communicate

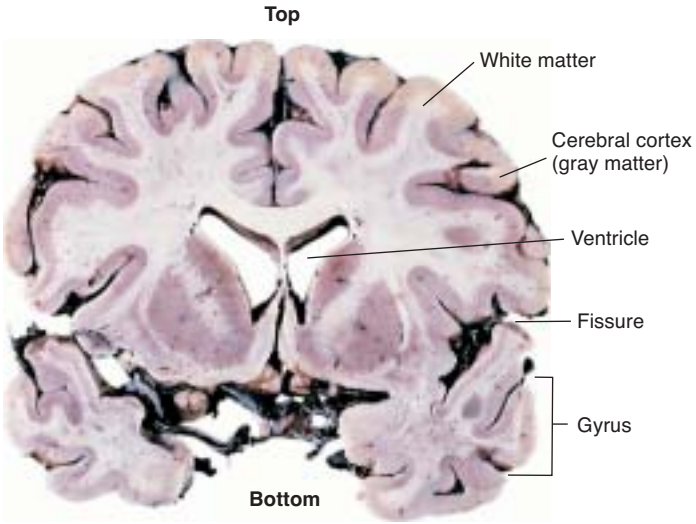
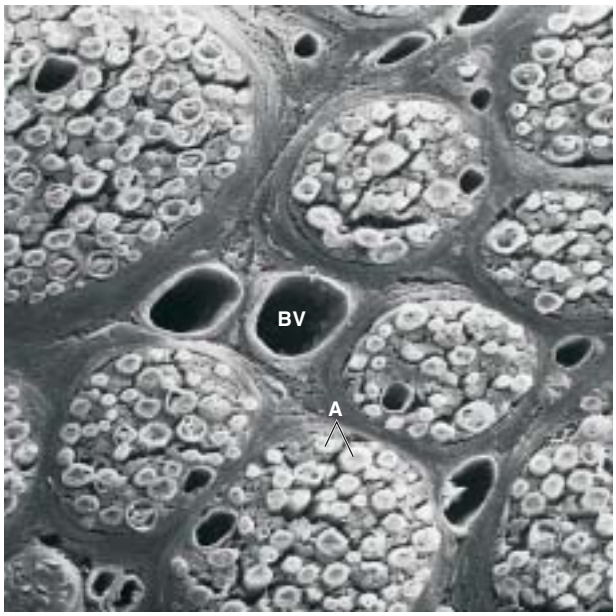


FIGURE 4•3 A photograph of a slice of a human brain showing fissures and gyri and the layer of cerebral cortex that follows these convolutions.
(Harvard Medical School/Betty G. Martindale)

FIGURE 4•4 A scanning electron micrograph of the cut end of a nerve, showing bundles of nerve fibers (also known as axons) and sheaths of connective tissue that encase them. BV = blood vessel; A = individual axons.

(From *Tissues and Organs: A Text-Atlas of Scanning Electron Microscopy*, by Richard G. Kessel and Randy H. Kardon. Copyright © 1979 by W. H. Freeman and Co. Reprinted by permission from the authors.)



the processed information to other neurons (or to cells in muscles, glands, or internal organs). Thus, neurons contain structures specialized for receiving, processing, and transmitting information. These structures are shown in **Figure 4•5**.

Dendrites, treelike growths attached to the body of a nerve cell, function principally to receive messages from other neurons. (*Dendron* means "tree.") They transmit the information they receive down their "trunks" to the cell body. The dendrites of some neurons receive information from other neurons through **dendritic spines,** small protuberances on their surfaces. The **soma,** or cell body, is the largest part of the neuron and contains the mechanisms that control the metabolism and maintenance of the cell. In most neurons, the soma also receives messages from other neurons. The nerve fiber, or **axon,** carries messages away from the soma toward the cells with which the neuron communicates. These messages, called *action potentials,* consist of brief changes in the electrical charge of the axon.

Axons end in **terminal buttons,** which are located at the ends of the "twigs" that branch off their ends. Terminal buttons secrete a chemical called a **neurotransmitter** whenever an action potential is sent down the axon (that is, whenever the axon fires). The neurotransmitter affects the activity of the other cells with which the neuron communicates. Thus, the message is conveyed *chemically* from one neuron to another. Most drugs that affect the nervous system and hence alter a person's behavior do so by affecting the chemical transmission of messages between cells.

Many axons, especially long ones, are insulated with a substance called *myelin*. The white matter located beneath the cerebral cortex gets its color from the **myelin sheaths** around the axons that travel through these areas. Myelin, part protein and part fat, is produced by glial cells that wrap parts of themselves around segments of the axon, leaving small bare patches of the axon between them. (Refer to Figure 4.5.) The principal function of myelin is to insulate axons from one another and thus to prevent the scrambling of messages. Myelin also increases the speed of the action potential.

To appreciate how important the myelin sheath is, consider the symptoms of one neurological disease. In some disorders, people's immune systems begin to attack parts of their own bodies. One such disorder is *multiple sclerosis,* so named because an autopsy of the brain and spinal cord will show numerous patches of hardened, damaged tissue. (*Skleros* is Greek for "hard.") The immune systems of people who have multiple sclerosis attack a protein in the myelin sheath of axons in the central nervous system, stripping it away. Although most of the axons survive this assault, they can no longer function normally, and so—depending on where the damage occurs—people who have multiple sclerosis suffer from a variety of neurological symptoms.

Figure 4.5 is very schematic; it's designed to illustrate the important parts of neurons and the synaptic connections between them. **Figure 4•6** is a photograph made with a scanning electron microscope. It shows the actual appearance of a neuron and some terminal buttons that form synapses with it. The terminal buttons were broken off from their axons when the tissue was being prepared, but by comparing this photograph with Figure 4.5 you can begin to imagine some of the complexity of the nervous system.

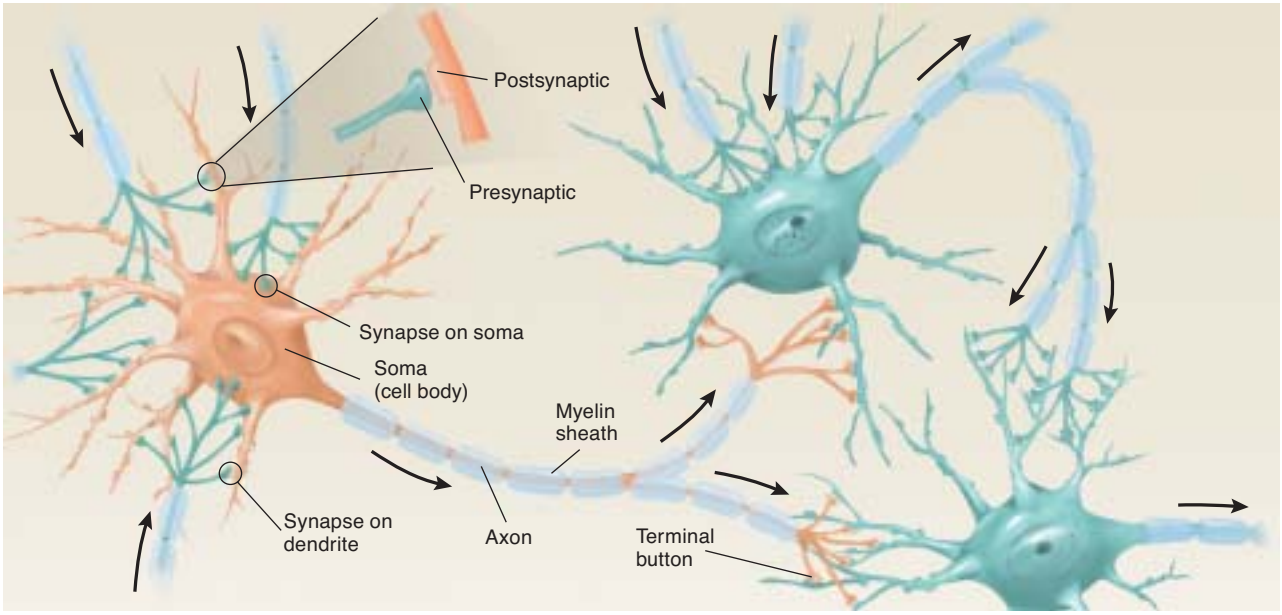
The Excitable Axon: The Action Potential

The message carried by the axon—the action potential—involves an electrical current, but it does not travel down the axon the way electricity travels through a wire. Electricity travels through a wire at hundreds of millions of feet per second. But, as you learned in Chapter 1, Hermann von Helmholtz discovered that the axon transmits information at a much slower rate—about 90 feet per second.

The membrane of an axon is electrically charged. When the axon is resting (that is, when no action potential is occurring), the inside is charged at –70 millivolts (thousandths of a volt) with respect to the outside. An **action potential** is an abrupt, short-lived reversal in the electrical charge of an axon. This temporary reversal begins at the end of the axon that attaches to the soma and is transmitted to the end that divides into small branches capped with terminal buttons. For convenience, an action potential is usually referred to as the *firing* of an axon.

The electrical charge of an axon at rest—the **resting potential**—occurs because of an unequal distribution of positively and negatively charged particles inside the axon and in the fluid that surrounds it. These particles, called **ions,** are

FIGURE 4•5 The basic parts of a neuron and its connections with other neurons (synapses). The detail depicts the structure of a synapse.



produced when various substances—including ordinary table salt—are dissolved in water. Molecules of table salt (sodium chloride) break down into positively charged sodium ions (Na^+) and negatively charged chloride ions (Cl^-). (In case you were wondering, sodium is abbreviated as *Na* because its original Latin name was *natrium*.) Normally, ions cannot penetrate the membrane that surrounds all cells. However, the membrane of axons contains special submicroscopic proteins that serve as ion channels or ion transporters. **Ion channels** can open or close; when they are open, a particular ion can enter or leave the axon. As we will see, the membrane of the axon contains two types of ion channels: sodium channels and potassium channels. **Ion transporters** work like pumps. They use the energy resources of the cell to transport particular ions into or out of the axon. (See **Figure 4•7**.)

When the axon is resting, the outside of the membrane is positively charged (and the inside is negatively charged) because the fluid inside the axon contains more negatively charged ions and fewer positively charged ions. When the membrane of the axon is resting, its ion channels are closed, so ions cannot move in or out of the axon. An action potential is caused when the end of the axon attached to the soma becomes excited, which opens sodium ion channels located there. (I'll describe this excitation later.) The opening of these ion channels permits positively charged sodium ions (Na^+) to enter; this reverses the membrane potential at that location. This reversal causes nearby ion channels to open, which produces another reversal at *that* point. The process continues all the way to the terminal buttons at the ends of the branches at the other end of the axon.

Note that an action potential is a *brief* reversal of the membrane's electrical charge. As soon as the charge reverses, the sodium ion channels close and potassium ion channels open for a short time, letting positively charged potassium ions (K^+) flow out of the axon. This outflow of positive ions restores the normal electrical charge. Thus, an action potential resembles the "wave" that sports fans often make in a stadium during a game. People in one part of the stadium stand up, raise their arms over their heads, and sit down again. People seated next to them see that a wave is starting, so they do the same—and the wave travels around the stadium. Everyone remains at the same place, but the effect is that of something

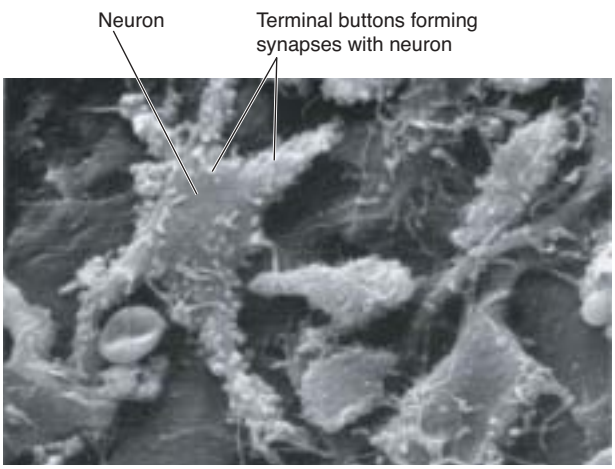
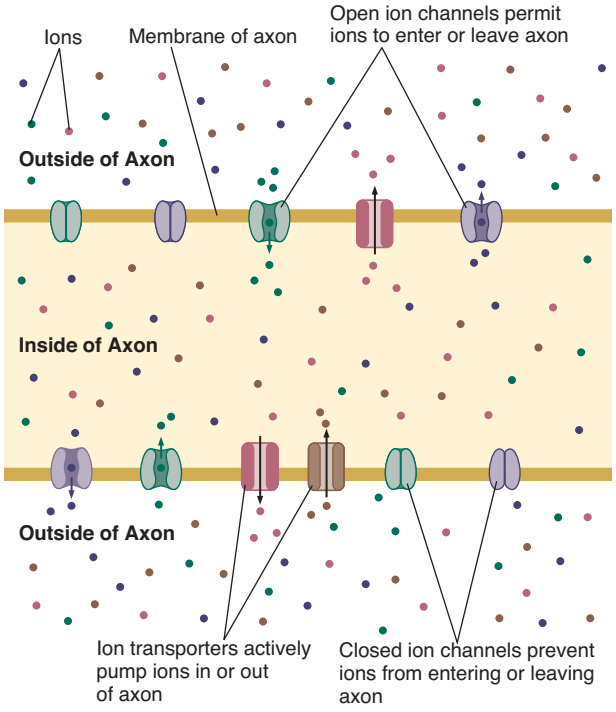


FIGURE 4•6 A scanning electron micrograph of a neuron.

(From *Tissues and Organs: A Text-Atlas of Scanning Electron Microscopy*, by Richard G. Kessel and Randy H. Kardon. Copyright © 1979 by W. H. Freeman and Co. Reprinted by permission from the authors.)

FIGURE 4•7 Ion channels and ion transporters. These structures regulate the numbers of ions found inside and outside the axon. An unequal distribution of positively and negatively charged ions is responsible for the axon's electrical charge.



circling in the stands around the playing field. Similarly, electricity does not really travel down the length of an axon. Instead, the entry of positive ions in one location reverses the charge at that point and causes ion channels in the adjacent region to open, and so on. (See **Figure 4•8**.)

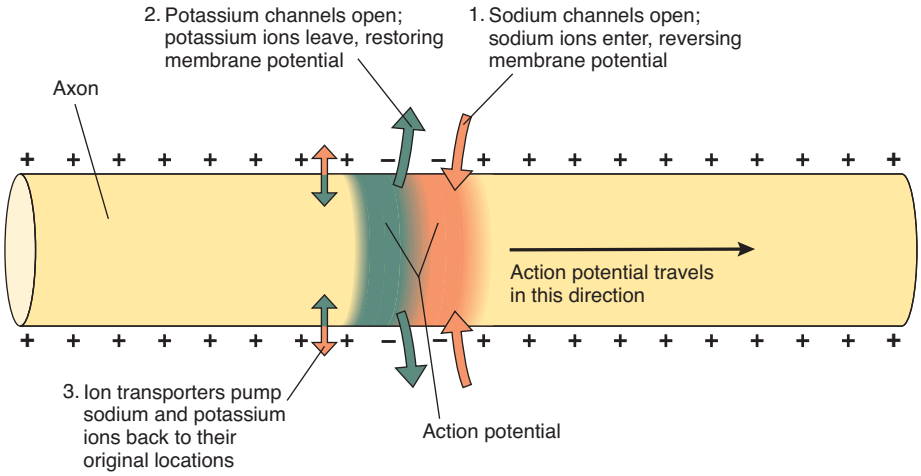
You may be wondering what happens to the sodium ions that enter the axon and the potassium ions that leave it. This is where the ion transporters come in. As diagrammed in Figure 4.8, after an action potential has moved along the axon, the ion transporters pump sodium ions out of the axon and pump potassium ions back in, restoring the normal balance.

An action potential is an all-or-none event—either it happens or it does not. Action potentials in a given axon are all the same size; there are no large or small action potentials. This fact has been stated as the **all-or-none law**. But if action potentials cannot vary in size, how can axons convey quantitative information? For example, how can **sensory neurons**—neurons that receive information from sense receptors—tell the brain about the strength of a stimulus? And how can **motor neurons**—neurons whose axons form synapses with a muscle—tell the muscle how forcefully to contract? The answer is simple: A single action potential is not the basic element of information; rather, quantitative information is represented by an axon's *rate of firing*. Strong stimuli (such as bright lights) trigger a high rate of firing in axons of sensory neurons that receive visual information. Similarly, a high rate of firing in the axons of motor neurons causes strong muscular contractions.

Communication with Other Cells: Synapses

Neurons communicate with other cells through synapses, by means of a process known as *synaptic transmission*. A **synapse** is the junction of a terminal button of one neuron and the membrane of another cell—another neuron or a cell in a muscle, a gland, or an internal organ. Let us first consider synapses between one neuron and another. The terminal button belongs to the **presynaptic neuron**—the neuron "before the synapse" that sends the message. As we saw, when terminal buttons become active, they release a chemical called a neurotransmitter. The neuron that receives the message (that detects the neurotransmitter) is called the **postsynaptic neuron**—the neuron "after the synapse." (Refer to detail, Figure 4.5.) A neuron receives messages from many terminal buttons, and in turn its terminal buttons form synapses with

FIGURE 4•8 Movement of sodium and potassium ions during the action potential. Sodium ions (Na^+) are represented by red arrows, potassium ions (K^+) by green arrows.



many other neurons. The drawing in Figure 4.5 is much simplified; thousands of terminal buttons can form synapses with a single neuron.

There are two basic types of synapses: *excitatory synapses* and *inhibitory synapses*. Excitatory synapses do just what their name implies—when the axon fires, the terminal buttons release a neurotransmitter that excites the postsynaptic neurons with which they form synapses. The effect of this excitation is to increase the rate of firing of the axons of the postsynaptic neurons. Inhibitory synapses do just the opposite—when they are activated, they *lower* the rate at which these axons fire.

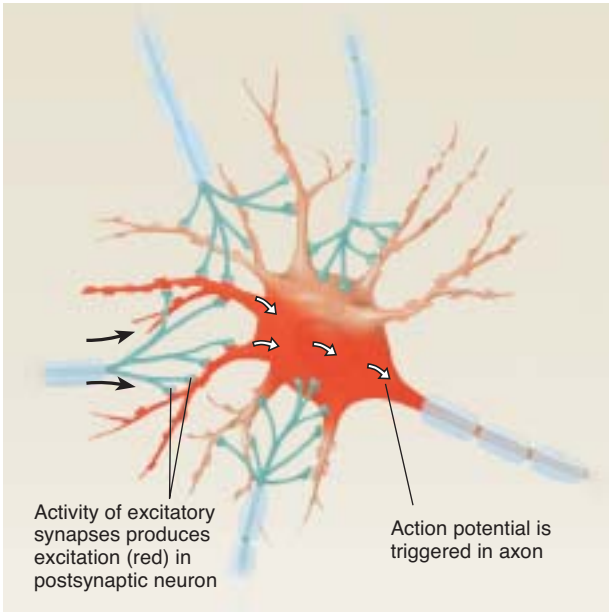
The rate at which a particular axon fires is determined by the activity of all the synapses on the dendrites and soma of the cell. If the excitatory synapses are more active, the axon will fire at a high rate. If the inhibitory synapses are more active, it will fire at a low rate or perhaps not at all. (See **Figure 4•9**.)

How do molecules of a neurotransmitter exert their excitatory or inhibitory effect on the postsynaptic neuron? When an action potential reaches a terminal button, it causes the terminal button to release a small amount of a neurotransmitter into the **synaptic cleft,** the fluid-filled space between the terminal button and the membrane of the postsynaptic neuron. (Note that the terminal button and the presynaptic membrane do not touch each other.) The neurotransmitter causes reactions in the postsynaptic neuron that either excite or inhibit it. These reactions are triggered by special submicroscopic protein molecules embedded in the postsynaptic membrane called **neurotransmitter receptors**. (See **Figure 4•10**.)

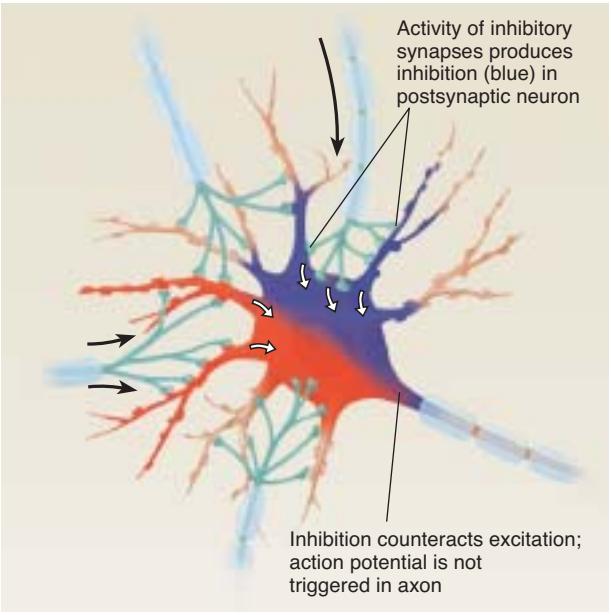
A molecule of a neurotransmitter binds with its receptor the way a particular key fits in a particular lock. After their release from a terminal button, molecules of a neurotransmitter diffuse across the synaptic cleft, bind with the receptors, and activate them. Once they are activated, the receptors produce excitatory or inhibitory effects on the postsynaptic neuron. They do so by opening ion channels. Most ion channels found at excitatory synapses permit sodium ions to enter the postsynaptic membrane; most of those found at inhibitory synapses permit potassium ions to leave. (See **Figure 4•11**.)

As I mentioned earlier, multiple sclerosis is caused by an autoimmune disorder that attacks a protein in the myelin sheaths of axons in the central nervous system. Another autoimmune disorder attacks a different protein—the neurotransmitter receptor that is found in the membrane of muscle fibers. Almost as fast as new receptors are produced, the immune system destroys them. The result of this attack is progressive *myasthenia gravis,* or "grave muscle weakness." Myasthenia gravis is not a very common disorder, but most experts believe that many mild cases go undiagnosed. I will have more to say about this disorder later in this chapter, in a section on drugs that affect synaptic transmission.

The excitation or inhibition produced by a synapse is short-lived; the effects soon pass away, usually in a fraction of a second. At most synapses the effects are terminated by a process called **reuptake**. Molecules of the neurotransmitter are released and are quickly taken up again by the terminal button, so the neurotransmitter has only a short time to stimulate the postsynaptic receptors. (See **Figure 4•12**.) The rate at which the terminal button takes back the neurotransmitter determines how prolonged the effects of the chemical on the postsynaptic neuron will be. The faster the neurotransmitter is taken back, the shorter its effects will be on the postsynaptic neuron. As we will see, some drugs affect the nervous system by slowing down the rate of reuptake, thus prolonging the effects of the neurotransmitter.



(a)



(b)

FIGURE 4•9 Interaction between the effects of excitatory and inhibitory synapses. Excitatory and inhibitory effects combine to determine the rate of firing of the neuron.

FIGURE 4•10 The release of a neurotransmitter from a terminal button. The drawing depicts the inset portion of Figure 4.5. *Top:* Before the arrival of an action potential. *Middle:* Just after the arrival of an action potential. Molecules of neurotransmitter have been released. *Bottom:* Activation of receptors. The molecules of neurotransmitter diffuse across the synaptic cleft and some of them activate receptors in the postsynaptic membrane.

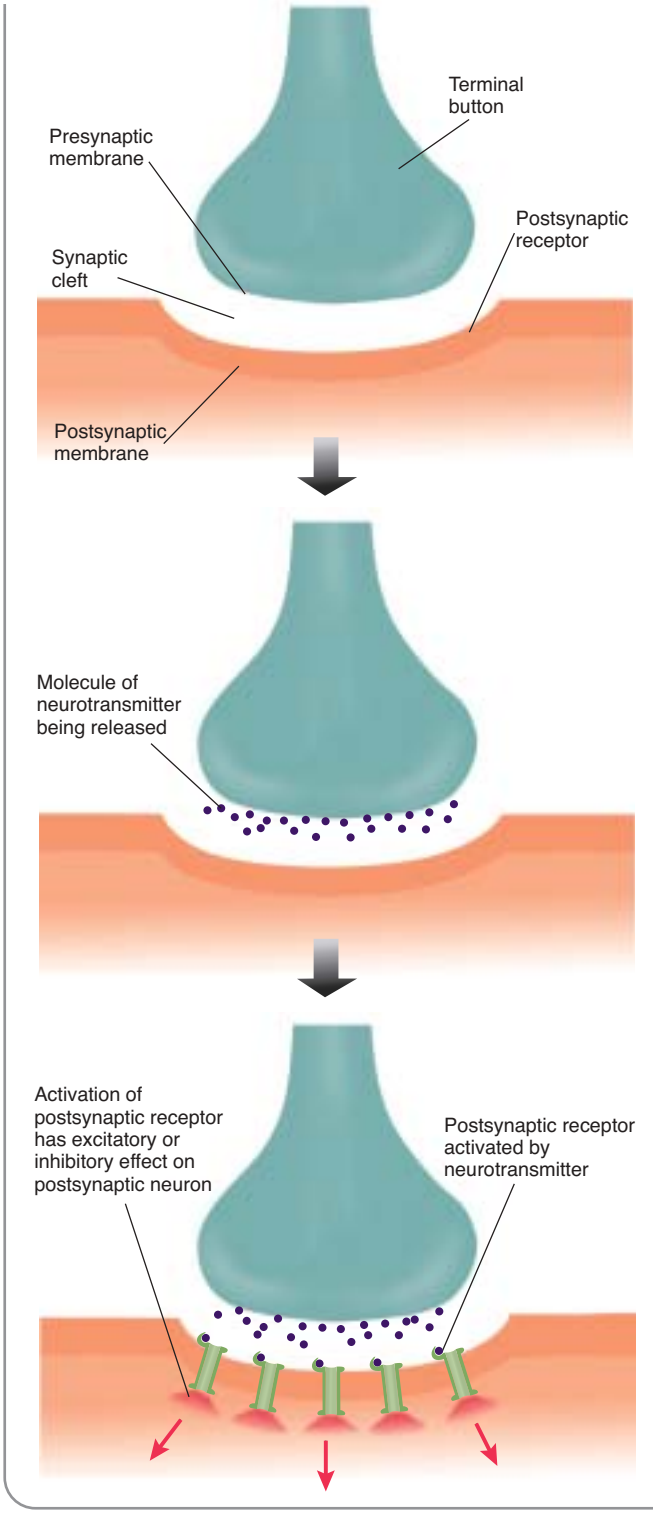
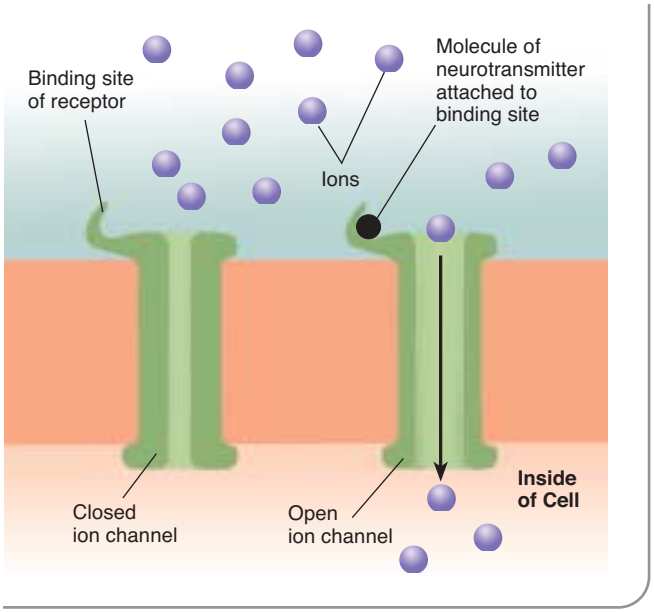


FIGURE 4•11 Postsynaptic receptors. An ion channel opens when a molecule of the neurotransmitter binds with the receptor. For purposes of clarity the drawing is schematic; molecules of neurotransmitter are actually much larger than individual ions.

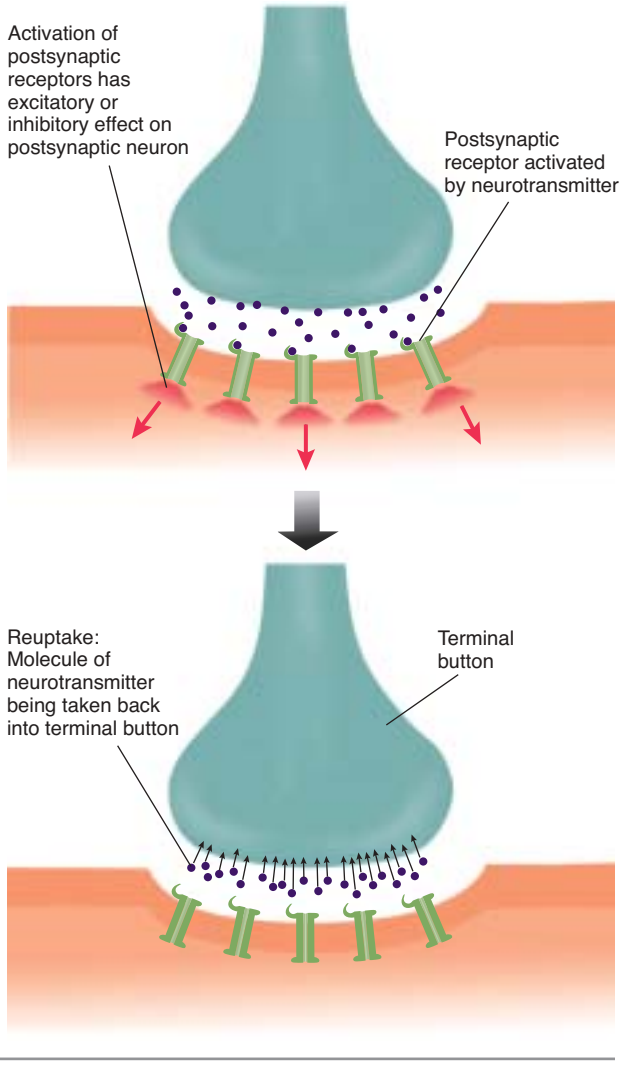


A Simple Neural Circuit

Let's try to put together what we know about neurons, action potentials, and synapses by seeing how a simple neural circuit works. The trillions of interconnections of the billions of neurons in our central nervous system provide us with the capacities for perception, thinking, memory, and action. Although we do not yet know enough to draw a neural wiring diagram for such complex functions, we can in fact diagram some of the simpler reflexes that are triggered by certain kinds of sensory stimuli. For example, when your finger touches a painfully hot object, your hand withdraws. When your eye is touched, your eyes close and your head draws back. When a baby's cheek is touched, it turns its mouth toward the object, and if the object is of the appropriate size and texture, the baby begins to suck. All these activities occur quickly, without thought.

We begin by examining a simple assembly of three neurons and a muscle that control a withdrawal reflex. Suppose you think your iron is cold, but you touch it to be sure. In fact, it is hot, and the heat stimulates the dendrites of sensory neurons in your finger. As a result, messages are sent down the axon to terminal buttons located in the spinal cord. These terminal buttons release a neurotransmitter that excites an **interneuron,** a neuron located entirely within the central nervous system. The terminal buttons of the interneuron release a neurotransmitter that excites a motor neuron. The axon of the motor neuron joins a nerve and travels to a muscle. When the axon fires, the terminal buttons of the motor neuron

FIGURE 4•12 Reuptake of molecules of neurotransmitter.



release their neurotransmitter, causing the muscle cells to contract. As a result, your hand moves away from the hot iron. (See **Figure 4•13**.)

The next example adds a little complexity to the circuit. Suppose you have removed a hot casserole from the oven. As you walk to the table to put it down, the heat begins to penetrate the rather thin potholders you are using. The pain caused by the hot casserole triggers a withdrawal reflex that would tend to make you drop it. And yet you manage to keep hold of the casserole long enough to get to the table and put it down. What prevented your withdrawal reflex from making you drop the dish on the floor?

As we saw earlier, the rate at which a neuron fires depends on the relative activity of the excitatory and inhibitory synapses on it. The pain from the hot casserole increases the activity of excitatory synapses on the motor neurons, which tend to cause the hand to open. However, this excitation is counteracted by inhibition from another source—the brain. The brain contains neural circuits that recognize what a disaster it would be if you dropped the casserole on the floor. These neural circuits send information to the spinal cord that prevents the withdrawal reflex from making you drop the dish.

Figure 4•14 shows how this information reaches the spinal cord. As you can see, an axon from a neuron in the brain reaches the spinal cord, where it forms a synapse with an inhibitory interneuron. When the neuron in the brain becomes active, it excites this inhibitory interneuron. The interneuron releases an inhibitory neurotransmitter, which decreases the rate of firing of the motor neuron, preventing your hand from opening. This circuit provides an example of a contest between two competing tendencies: to drop the casserole and to hold on to it. Complex decisions about behavior are made within the brain by much more complicated circuits of neurons, but the basic principles remain the same.

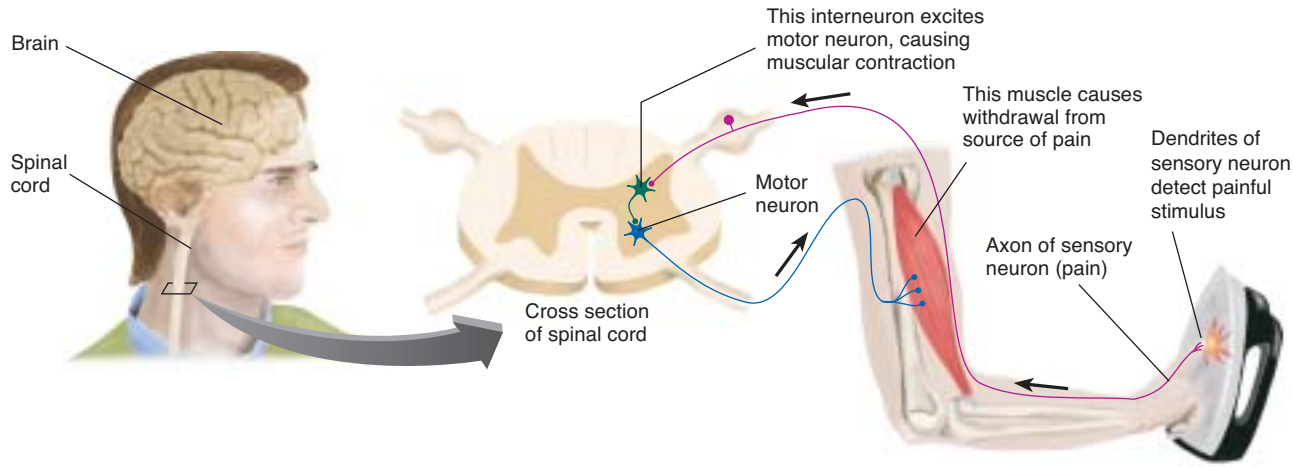


FIGURE 4•13 A schematic representation of the elements of a withdrawal reflex. Although this figure shows just one sensory neuron, one interneuron, and one motor neuron, in reality many thousands of each type of neuron would be involved.

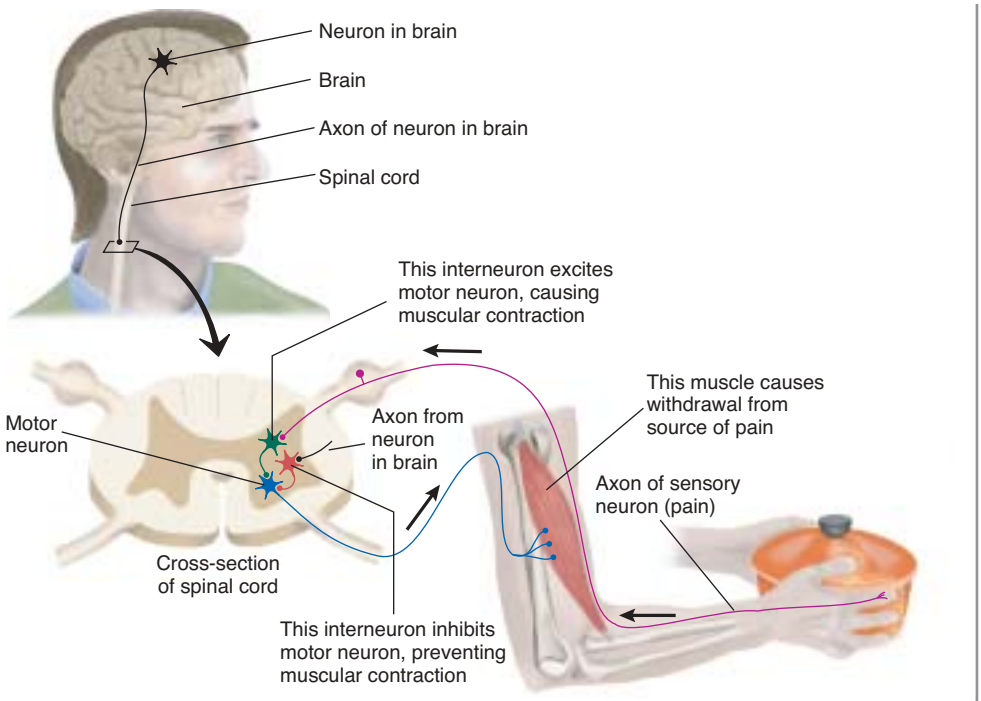


FIGURE 4•14 A schematic representation of a withdrawal reflex being inhibited by the brain.

Of course, reflexes are more complicated than this description, and the mechanisms that inhibit them are even more so. And thousands of neurons are involved in this process. The five neurons shown in Figure 4.14 represent many others: Dozens of sensory neurons detect the hot object, hundreds of interneurons are stimulated by their activity, hundreds of motor neurons produce the contraction—and thousands of neurons in the brain must become active if the reflex is to be inhibited. Yet this simple model provides an overview of the process by which decisions are made in the nervous system.

Interim Summary

The Brain and Its Components

The brain has three major functions: controlling behavior, processing and storing information about the environment, and regulating the body's physiological processes.

The central nervous system consists of the spinal cord and the three major divisions of the brain: the brain stem, the cerebellum, and the cerebral hemispheres. The central nervous system floats in a pool of cerebrospinal fluid, contained by the meninges, which protects it from physical shock. The blood–brain barrier protects the brain from toxic substances in the blood. The cerebral cortex, which covers the cerebral hemispheres, is wrinkled by fissures and gyri. The brain communicates with the rest of the body through the peripheral nervous system, which includes the spinal nerves and cranial nerves.

The basic element of the nervous system is the neuron, with its dendrites, soma, axon, and terminal buttons. Neurons are assisted in their tasks by glia, which provide physical support, aid in the development of the nervous system, provide neurons with chemicals they need, remove unwanted chemicals, provide myelin sheaths for axons, and protect neurons from infections.

One neuron communicates with another (or with cells of muscles, glands, or internal organs) through synapses. A synapse is the junction of the terminal button of the presynaptic neuron with the membrane of the postsynaptic cell. Synaptic communication is chemical; when an action potential travels down an axon (when the axon "fires"), it causes a neurotransmitter to be released by the terminal buttons. An action potential consists of a brief change in the electrical charge of the axon, produced by the brief entry of positively charged sodium ions into the axon followed by a brief exit of positively charged potassium ions. Ions enter the axon through ion channels, and ion transporters eventually restore the proper concentrations of ions inside and outside the cell.

Molecules of the neurotransmitter released by terminal buttons bind with neurotransmitter receptors in the postsynaptic membrane and either excite or inhibit the firing of the postsynaptic cell. The combined effects of excitatory and inhibitory synapses acting on a particular neuron determine the rate of firing of that neuron. The reflex is the simplest element of behavior, and it illustrates the contest between excitation and inhibition.

QUESTIONS TO CONSIDER

1. The brain is the seat of our perceptions, thoughts, memories, and feelings. Why, then, do we so often refer to our hearts as the location of our feelings and emotions? For example, why do you think we say, "He acted with his heart, not with his head"?
2. The blood–brain barrier keeps many chemicals in the blood out of the brain. There are a few places in the brain where this barrier does not exist, including the part of the brain stem that contains the neural circuits that trigger vomiting. Can you think of an explanation for the lack of a blood–brain barrier in this region?

Drugs and Behavior

Long ago, people discovered that the sap, fruit, leaves, bark, or roots of various plants could alter their perceptions and behavior, could be used to relieve pain or treat diseases, or could be used as poisons to kill animals for food. They also discovered that some substances affected people's moods in ways that they wanted to experience again and again.

Why do plants produce chemicals that have specific effects on the cells of our nervous system? They do so because the chemicals are toxic to animals—primarily insects—that eat them. Our neurons are also sensitive to these chemicals, so they affect us as well. Of course, some chemicals produced by plants have beneficial effects in humans, and have consequently been extracted or synthesized in the laboratory for use as therapeutic drugs. The therapeutic use of drugs are of obvious benefit to society, and the abuse of addictive drugs is responsible for much misery and unhappiness. But drugs are also important tools to help scientists discover how the brain works. For example, we know that certain drugs relieve anxiety and others reduce the symptoms of schizophrenia. Discovering how these drugs affect the brain can help our understanding of the causes of these disorders and can provide information we need to develop even better forms of treatments.

Effects of Drugs on Synaptic Transmission

Drugs that affect our thoughts, perceptions, emotions, and behavior do so by affecting the activity of neurons in the brain. As we saw, communication between neurons involves the release of neurotransmitters, which bind with receptors and either excite or inhibit the activity of the postsynaptic cell. Drugs can affect this process in may ways. They can stimulate or inhibit the release of neurotransmitters, mimic the effects of neurotransmitters on postsynaptic receptors, block these effects, or interfere with the reuptake of a neurotransmitter once it is released. Through these mechanisms (and others too complicated to describe here), a drug can alter the perceptions, thoughts, and behaviors controlled by particular neurotransmitters. Let's briefly examine some of these mechanisms. In the next section I will provide specific examples of drugs and the neurotransmitters they affect.

Stimulating or Inhibiting the Release of Neurotransmitters Some drugs stimulate certain terminal buttons to release their neurotransmitter continuously, even when the axon is not firing. Other drugs prevent certain terminal buttons from releasing their neurotransmitter when the axon fires. The effects of a particular drug are usually specific to one neurotransmitter. (See **Figure 4•15**.)

Stimulating or Blocking Postsynaptic Receptors Neurotransmitters produce their effects by stimulating postsynaptic receptors; this excites or inhibits postsynaptic neurons by open-

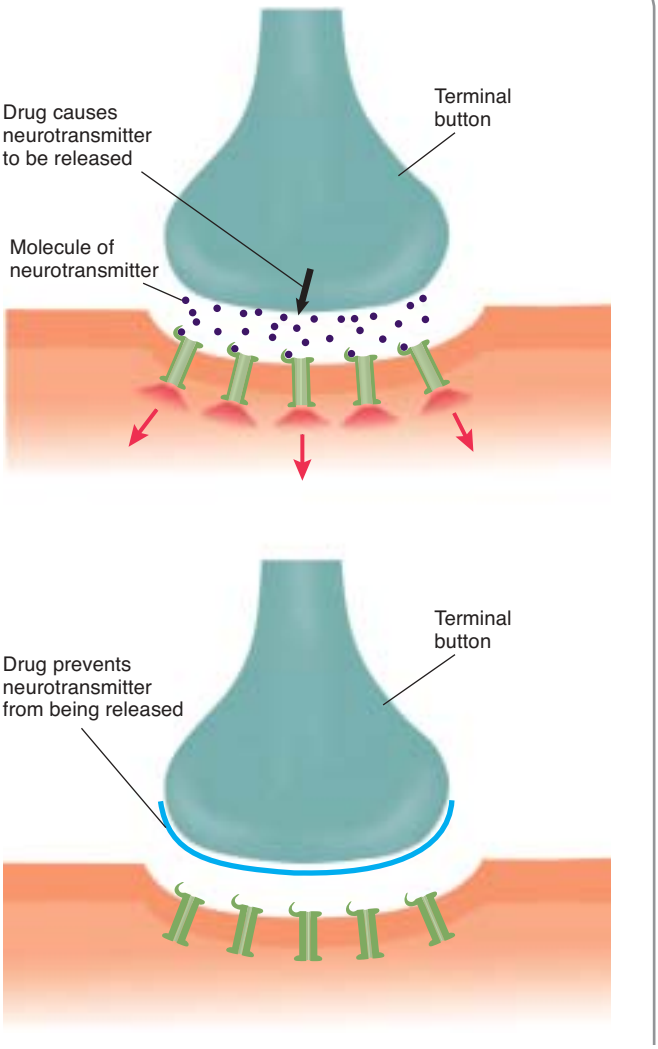


FIGURE 4•15 Effects of drugs on the release of a neurotransmitter. *Top:* Stimulation of release. *Bottom:* Inhibition of release.

ing ion channels and permitting ions to enter or leave the neurons. Some drugs mimic the effects of particular neurotransmitters by directly stimulating particular kinds of receptors. If we use the lock-and-key analogy to describe the effects of a neurotransmitter on a receptor, then a drug that stimulates receptors works like a master key, turning the receptors on even when the neurotransmitter is not present. (See **Figure 4•16**.)

Some drugs bind with receptors and do *not* stimulate them, This action blocks receptors, making them inaccessible to the neurotransmitter and thus inhibiting synaptic transmission. To continue the lock-and-key analogy, a drug that blocks receptors plugs up the lock so that the key will no longer fit into it.

Inhibiting Reuptake The effects of most neurotransmitters are kept brief by the process of reuptake. Molecules of the neurotransmitter are released by a terminal button, stimulate the receptors in the postsynaptic membrane for a fraction of a second, and are then taken back into the terminal button. Some drugs inhibit the process of reuptake so that molecules of the neurotransmitter continue to stimulate the postsynaptic receptors for a long time. Therefore, inhibition of reuptake increases the effect of the neurotransmitter. (See **Figure 4•17**.)

Neurotransmitters, Their Actions, and Drugs That Affect Them

Now that we've seen the most important ways that drugs can affect synaptic transmission, let's look at the most important neurotransmitters and consider some examples of drugs that interact with them. Because neurotransmitters have two general effects on postsynaptic membranes—excitatory or inhibitory—you might expect that there would be two kinds of neurotransmitters. But in reality there are many different kinds—several dozen, at least.

In the brain most synaptic communication is accomplished by two neurotransmitters: **glutamate,** which has excitatory effects, and **GABA,** which has inhibitory effects. (If you really want to know, *GABA* stands for *gamma-amino butyric acid.*) Almost every neuron in the brain receives excitatory input from terminal buttons that secrete glutamate and inhibitory input from terminal buttons that secrete GABA. (Another inhibitory neurotransmitter, *glycine,* is found in the lower brain stem and the spinal cord, but I won't discuss this chemical here.)

What do all the other neurotransmitters do? In general, they have modulating effects rather than information-transmitting effects. That is, the release of neurotransmitters other than glutamate and GABA tends to activate or inhibit entire circuits of neurons that are involved in particular brain functions. These effects include facilitation of learning, control of wakefulness and vigilance, suppression of impulsive behaviors, and suppression or enhancement of anxiety. Thus, because particular drugs can selectively affect neurons that secrete particular neurotransmitters, these drugs can have specific effects on behavior.

Given the importance of glutamate and GABA, let's look at these two neurotransmitters first.

Glutamate As I just mentioned, glutamate is the most important excitatory neurotransmitter in the brain. It is also the major excitatory neurotransmitter in the spinal cord. With the exception of neurons that detect painful stimuli, all sensory organs transmit information to the brain through axons whose terminals release glutamate.

One type of glutamate receptor (the *NMDA receptor*) plays a critical role in the effects of environmental stimulation on the developing brain and is also responsible for many of the changes in synaptic connections that are responsible for learning. This receptor is partially deactivated by alcohol, which accounts for the fact that binge drinkers often have no memory for what happened while they were drunk. In addition, researchers believe that the effect of alcohol on this receptor is responsible for the dangerous convulsions that

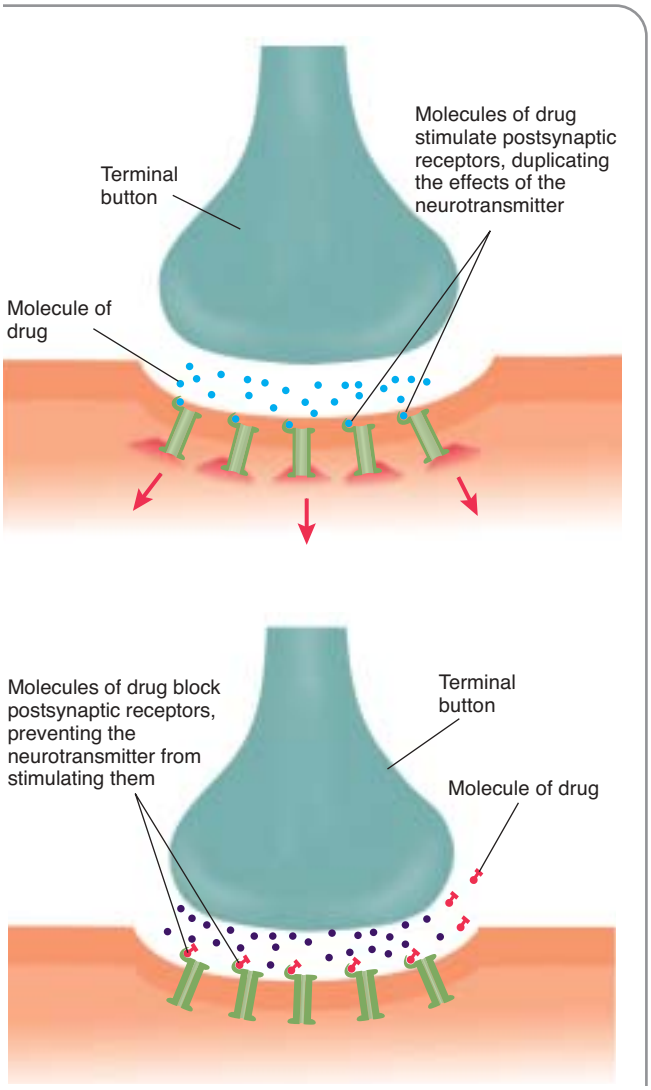
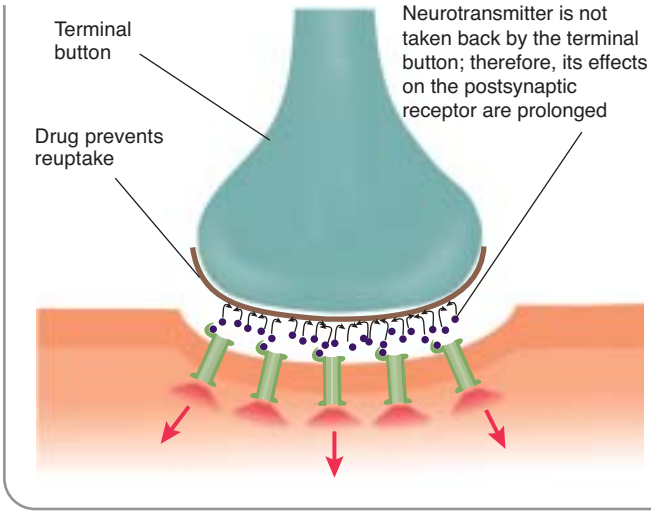


FIGURE 4•16 Effects of drugs on the postsynaptic receptors. *Top:* Stimulation of receptors, mimicking the effects of the neurotransmitter. *Bottom:* Blocking of receptors, preventing the neurotransmitter from binding with the receptors.

FIGURE 4•17 Reuptake inhibition. Drugs that block reuptake of the neurotransmitter strengthen and prolong the effects of the neurotransmitter on postsynaptic receptors.



can be caused by sudden withdrawal from heavy, long-term alcohol abuse. When this receptor is suppressed for a long time, a compensatory mechanism makes it become more sensitive to glutamate. If the person suddenly stops taking alcohol, a rebound effect causes glutamate to have such a strong effect that the normal balance of excitation and inhibition in the brain is disrupted.

GABA Some drugs depress behavior, causing relaxation, sedation, or even loss of consciousness. Most of these drugs act on a particular type of GABA receptor (the $GABA_A$ receptor), increasing its sensitivity to the neurotransmitter. **Barbiturates** act this way. In low doses, barbiturates have a calming effect. In progressively higher doses, they produce difficulty in walking and talking, unconsciousness, coma, and death. Barbiturates are abused by people who want to achieve the relaxing, calming effect of the drugs, especially to counteract the anxiety and irritability that can be produced by stimulants. A dose of a barbiturate sufficient to cause relaxation is not much lower than a fatal dose; thus, these drugs do not have much of a safety factor. Physicians rarely prescribe barbiturates.

By far the most commonly used depressant drug is ethyl alcohol, the active ingredient in alcoholic beverages. This drug also acts on the $GABA_A$ receptor. The effects of alcohol and barbiturates are additive: A moderate dose of alcohol plus a moderate dose of barbiturates can be fatal.

Many **antianxiety drugs** are members of a family known as the **benzodiazepines,** which include the well-known tranquilizer Valium (diazepam). These drugs, too, act on $GABA_A$ receptors on neurons in various parts of the brain, including a region that is involved in fear and anxiety. Benzodiazepines are much safer than barbiturates—a lethal dose is more than a hundred times higher than a therapeutic dose. They are sometimes used to treat people who are afflicted by periodic attacks of severe anxiety. In addition, some benzodiazepines serve as sleep medications. These drugs also are used to treat the convulsions caused by sudden withdrawal from heavy, long-term alcohol abuse.

Acetylcholine **Acetylcholine (ACh)** is the primary neurotransmitter secreted by the axons of motor neurons, and it is also released by several groups of neurons in the brain. Because all muscular movement is accomplished by the release of acetylcholine, you will not be surprised to learn that the immune systems of people with myasthenia gravis (described in the previous section) attack acetylcholine receptors.

The axons and terminal buttons of acetylcholinergic neurons are distributed widely throughout the brain. Three systems have received the most attention from neuroscientists. One system controls most of the characteristics of REM sleep—the phase of sleep during which most dreaming occurs. Another system is involved in activating neurons in the cerebral cortex and facilitating learning, especially perceptual learning. A third system controls the functions of another part of the brain involved in learning: the hippocampus. (I will describe this structure later in this chapter.)

Two drugs, botulinum toxin and the venom of the black widow spider, affect the release of acetylcholine. **Botulinum toxin,** produced by a bacterium that can grow in improperly canned food, prevents the release of ACh. The drug is an extremely potent poison; someone once calculated that a teaspoonful of pure botulinum toxin could kill the world's entire human population. Extremely dilute solutions (they had better be!) of this drug, usually referred to as *botox,* can be injected into people's facial muscles to stop muscular contractions that are causing wrinkles. **Black widow spider venom** has the opposite effect: It stimulates the release of ACh. Although the effects of black widow spider venom can also be fatal to infants or frail, elderly people, the venom is much less toxic than botulinum toxin.

▲ *The venom of the black widow spider causes the release of acetylcholine, which can cause numbness, muscle pain and cramps, sweating, salivation, and difficulty breathing. Fortunately, a single bite is very rarely fatal for a healthy adult.*

Although the effects of most neurotransmitters on the postsynaptic membrane are terminated by reuptake, acetylcholine is an exception. After being released by the terminal button, ACh is deactivated by an enzyme that is present in the postsynaptic membrane. This enzyme, *AChE (acetylcholinesterase),* can be inactivated by various drugs. One of them, **neostigmine,** can help people with myasthenia gravis. The drug lets the patient regain some strength, because the acetylcholine that is released has a more prolonged effect on the few acetylcholine receptors that remain. (Fortunately, neostigmine cannot cross the blood–brain barrier, so it does not affect the AChE found in the central nervous system.)

The best known drug that affects acetylcholine receptors is **nicotine,** found in the leaves of the tobacco plant, *Nicotiniana tabacum.* Nicotine is a highly addictive drug; as evidence, consider the fact that after undergoing surgery for lung cancer, approximately 50 percent of patients continue to smoke. The addictive nature of nicotine indicates that acetylcholine plays a role in the reinforcement (reward) mechanisms of the brain. I'll have more to say about the nature of reinforcement later in this chapter and in Chapters 5 and 13.

Another drug, **curare,** blocks acetylcholine receptors. Because these are the receptors on muscles, curare, like botulinum toxin, causes paralysis. However, the effects of curare are much faster. The drug is extracted from several species of plants found in South America, where it was discovered long ago by people who used it to coat the tips of arrows and darts. Within minutes of being struck by one of these points, an animal collapses, ceases breathing, and dies. Nowadays, curare (or any of various drugs with the same site of action) is used to paralyze patients who are to undergo surgery so that their muscles will relax completely and not contract when they are cut with a scalpel. An anesthetic also must be used, because a person who receives only curare will remain perfectly conscious and sensitive to pain, even though paralyzed. And, of course, a respirator must supply air to the lungs during the procedure.

▲ *Injections of botox (a dilute solution of botulinum toxin) are used to smooth out wrinkles. The drug blocks the release of acetylcholine, which paralyzes muscles whose contraction causes the wrinkles.*

▲ *This native of Peru is inserting a curare-tipped dart into his blowgun. Curare kills animals by blocking acetylcholine receptors, which paralyzes muscles and causes suffocation.*

Monoamines Dopamine, norepinephrine, and serotonin are three chemicals that belong to a family of compounds called **monoamines.** Because the molecular structures of these substances are similar, some drugs affect the activity of all of them to some degree. The monoamines are produced by several systems of neurons in the brain. Most of these systems consist of a relatively small number of cell bodies located in the brain stem, whose axons branch repeatedly and give rise to an enormous number of terminal buttons distributed throughout many regions of the brain. Monoaminergic neurons thus serve to modulate the function of widespread regions of the brain, increasing or decreasing the activities of particular brain functions.

Dopamine (DA) has been implicated in several important functions, including movement, attention, learning, and the reinforcing effects of drugs that people tend to abuse. A progressive degenerative disease that destroys one set of DA neurons causes **Parkinson's disease,** a movement disorder characterized by tremors, rigidity of the limbs, poor balance, and difficulty in initiating movements. People with Parkinson's disease are given a drug called L-DOPA. Once this chemical reaches the brain, it is taken up by the DA neurons that still

survive and is converted to dopamine. As a result, these neurons release more dopamine, which alleviates the patients' symptoms.

Several drugs inhibit the reuptake of dopamine, thus serving to prolong and strengthen its effects. The best known of these drugs are amphetamine and cocaine. The fact that people abuse these drugs indicates that dopamine plays an important role in reinforcement. (In fact, nicotine exerts its reinforcing effect by indirectly increasing the activity of terminal buttons that release dopamine.)

Dopamine has been implicated as a neurotransmitter that might be involved in schizophrenia, a serious mental disorder whose symptoms include hallucinations, delusions, and disruption of normal, logical thought processes. Drugs such as Thorazine (chlorpromazine) and Clozaril (clozapine) relieve the symptoms of this disorder, apparently by blocking particular types of dopamine receptors. The physiology of schizophrenia is discussed in Chapter 17.

Almost every region of the brain receives input from neurons that secrete the second monoamine, **norepinephrine (NE).** Release of NE (also known as *noradrenaline)* appears to cause an increase in vigilance—attentiveness to events in the environment. Norepinephrine also plays a role in the control of REM sleep.

The third monoamine neurotransmitter, **serotonin,** has complex behavioral effects. Serotonin plays a role in the regulation of mood; in the control of eating, sleep, and arousal; and in the regulation of pain. A deficiency in the release of serotonin in the cerebral cortex is associated with alcoholism and antisocial behavior. Like NE neurons, serotonin-secreting neurons are involved in the control of REM sleep. Drugs such as Prozac (fluoxetine), which inhibit the reuptake of serotonin and thus strengthen and prolong its effects, are used to treat depression, anxiety disorder, and obsessive-compulsive disorder. A drug that causes the release of serotonin (fenfluramine) was used as an appetite suppressant in the 1990s, but adverse side effects took this drug off the market.

Several hallucinogenic drugs appear to produce their effects by interacting with serotonergic transmission. For example, **LSD** (lysergic acid diethylamide) produces distortions of visual perceptions that some people find awesome and fascinating but that simply frighten other people. This drug, which is effective in extremely small doses, stimulates one category of serotonin receptor.

Peptides As I have discussed, terminal buttons excite or inhibit postsynaptic neurons by releasing neurotransmitters. These chemicals travel a very short distance and affect receptors located on a small patch of the postsynaptic membrane. But some neurons release chemicals that get into the general circulation of the brain and stimulate receptors on many thousands of neurons, some located a considerable distance away. These chemicals are called **neuromodulators,** because they modulate the activity of the neurons they affect. We can think of neuromodulators as the brain's own drugs. As these chemicals diffuse through the brain, they can activate or inhibit circuits of neurons that control a variety of functions; thus, they can modulate particular categories of behavior.

Most neuromodulators are peptides. (The most important exception to this rule is described in the next subsection.) **Peptides** are molecules that consist of two or more amino acids attached together by special chemical links called peptide bonds. One of the best known families of peptides is the **endogenous opioids.** *Endogenous* means "produced from within"; *opioid* means "like opium." Several years ago it became clear that opiates—drugs such as opium, morphine, and heroin—reduce pain because they have direct effects on the brain. (Please note that the term *opioid* refers to endogenous chemicals, and *opiate* refers to drugs.) The endogenous opioids stimulate special opioid receptors located on neurons in several parts of the brain. Their behavioral effects include decreased sensitivity to pain and a tendency to persist in ongoing behavior. Opioids are released while an animal is engaging in important species-typical behaviors, such as mating or fighting. The behavioral effects of opioids ensure that a mating animal or an animal fighting to defend itself is less likely to be deterred by pain; thus, conception is more likely to occur and a defense is more likely to be successful.

People abuse opiates not because opiates reduce pain, however, but because they cause the release of dopamine in the brain, which has a reinforcing effect on behavior. It is this reinforcing effect that normally encourages an animal performing a useful and important behavior to continue in that behavior. Unfortunately, the reinforcing effect is not specific to useful and important behaviors and can lead to addiction.

To help drug addicts pharmacologists have developed drugs that block opioid receptors. One of them, **naloxone,** is used clinically to reverse opiate intoxication. This drug has saved the lives of many drug abusers brought to the emergency room in heroin-induced comas. An injection of naloxone blocks the effects of the heroin, and the person quickly revives.

Various peptide neuromodulators other than the opioids play important roles in behaviors important to survival, such as control of eating and metabolism, drinking, mineral balance, mating, parental care, and social bonding. Some reduce anxiety; others increase it. Some promote eating; others curb the appetite. Research on the effects of these chemicals is discussed in later chapters.

Cannabinoids You have undoubtedly heard of *Cannabis sativa,* the plant that produces hemp and marijuana. You probably also know that the plant produces a resin that has physiological effects on the brain. The principal active ingredient in this resin is THC (tetrahydrocannibinol), which affects perception and behavior by activating receptors located on neurons in the brain. THC mimics the effects of **endogenous cannabinoids**—chemicals produced and released by neurons in the brain.

THC produces analgesia and sedation, stimulates appetite, reduces nausea caused by drugs used to treat cancer,

▲ *The effects of the endogenous cannabinoids, produced and released in the brain, are mimicked by THC, the active ingredient of* Cannabis sativa, *the marijuana plant.*

relieves asthma attacks, decreases pressure within the eyes in patients with glaucoma, and reduces the symptoms of certain motor disorders. On the other hand, THC interferes with concentration and memory, alters visual and auditory perception, and distorts perception of the passage of time (Iversen, 2003). Devane and colleagues (1992) discovered the first—and most important—endogenous cannabinoid, a lipidlike (fatlike) substance, which they named **anandamide,** from the Sanskrit word *ananda,* or "bliss."

Cannabinoid receptors are found on terminal buttons of neurons that secrete glutamate, GABA, acetylcholine, dopamine, norepinephrine, and serotonin. (That is, almost all of the neurotransmitters I've mentioned in this chapter.) Thus, the secretion of anandamide—or the smoking of marijuana—alters the release of these neurotransmitters, and this has widespread effects in the brain. Recent research indicates that the endogenous cannabinoids modulate the synaptic changes that appear to be responsible for learning, which accounts for the fact that THC disrupts short-term memory (Fegley et al., 2004). Cannabinoids also appear to play an essential role in the reinforcing effects of opiates: A genetically engineered mutation that prevents the production of cannabinoid receptors abolishes the reinforcing effects of morphine, but not of cocaine, amphetamine, or nicotine (Cossu et al., 2001).

Table 4•2 lists the neurotransmitters discussed in this section, summarizes their effects, and lists some drugs that interact with them.

Evaluating Scientific Issues

"Physiological" versus "Psychological" Drug Addiction

As we all know, some drugs have very potent reinforcing effects, which lead some people to abuse them or even to become addicted to them. Some people (psychologists, health professionals, and laypeople) believe that "true" addiction is caused by the unpleasant physiological effects that occur when an addict tries to stop taking the drug. For example, in the 1960s Eddy and colleagues (1965) defined *physical dependence* as "an adaptive state that manifests itself by intense physical disturbances when the administration of a drug is suspended" (p. 723). In contrast, they defined *psychic dependence* as a condition in which a drug produces "a feeling of satisfaction and a psychic drive that requires periodic or continuous administration of the drug to produce pleasure or to avoid discomfort" (p. 723). Many people regard the latter as less important than the former. But, as we shall see, the reverse is true.

Evidence for Physiological Addiction

For many years, heroin addiction has been considered the prototype for all drug addictions. People who habitually take heroin (or other opiates) become physically dependent on the drug—that is, they show *tolerance* and *withdrawal symptoms*. **Tolerance** is the decreased sensitivity to a drug that comes from its continued use; drug users must take larger and larger amounts of the drug in order for it to be effective. Once people have taken an opiate regularly enough to develop tolerance, they will suffer withdrawal symptoms if they stop taking the drug. **Withdrawal symptoms** are primarily the opposite of the effects of the drug itself. For example, heroin produces euphoria; withdrawal from it produces *dysphoria*—a feeling of anxious misery. (*Euphoria* and *dysphoria* mean "easy to bear" and "hard to bear," respectively.) Heroin produces constipation; withdrawal from it produces nausea, cramping, and diarrhea. Heroin produces relaxation; withdrawal from it produces agitation. Most investigators believe that the withdrawal symptoms are produced by the body's attempt to compensate for the unusual condition of heroin intoxication. That is, most systems of the body, including those controlled by the brain, are regulated so that they stay at an optimal value. When a drug artificially changes these systems for a prolonged time, homeostatic mechanisms begin to produce the opposite reaction, which partially compensates for the disturbance from the optimal value. These compensatory mechanisms account for the fact that more and more heroin must be taken to achieve the effects that were produced when the person first started taking the drug. They also account for the symptoms of withdrawal: When the person stops taking the drug, the compensatory mechanisms make themselves felt, unopposed by the action of the drug.

How Important Is Physiological Addiction?

Heroin addiction has provided such a striking example of drug dependence that some authorities have concluded that

TABLE 4•2 The Major Neurotransmitters, Their Primary Effects, and Drugs That Interact with Them

Neurotransmitter	Primary Effects	Drugs That Interact with Neurotransmitter	Effects of Drugs
Glutamate	Primary excitatory neurotransmitter in brain	Alcohol	Desensitization of NMDA receptor
GABA	Primary excitatory neurotransmitter in brain	Barbiturates Benzodiazepines ("tranquilizers") Alcohol	Desensitization of $GABA_A$ receptor
Acetylcholine (ACh)	Excites muscular contraction, activates cerebral cortex, controls REM sleep, controls hippocampus	Botulinum toxin Black widow spider venom Neostigmine Nicotine	Blocks release of ACh Stimulates release of ACh Blocks AChE; enhances effects of ACh Stimulates ACh receptors
Monoamines			
Dopamine (DA)	Facilitates movement, attention, learning, reinforcement	L-DOPA Amphetamine, cocaine Antipsychotic drugs	Increases synthesis of dopamine Inhibit reuptake of dopamine Block dopamine receptors
Norepinephrine (NE)	Increases vigilance, controls REM sleep		
Serotonin	Regulates mood; controls eating, sleep, arousal, regulation of pain; suppresses risky behaviors	Fluoxetine (Prozac) LSD	Inhibits reuptake of serotonin Stimulates certain serotonin receptors
Endogenous opioids	Reduce pain, reinforce ongoing behavior	Opiates (heroin, morphine, etc.) Naloxone	Stimulate opioid receptors Blocks opioid receptors
Anandamide (endogenous cannabinoid)	Analgesia, nausea reduction, decreased pressure in eyes, interference with short-term memory, increased appetite	THC	Stimulates cannabinoid receptors

"real" addiction does not occur unless a drug causes tolerance and withdrawal. Without doubt, withdrawal symptoms make it difficult for a person to stop taking heroin—they help keep the person hooked. But withdrawal symptoms do not explain why a person becomes a heroin addict in the first place; that fact is explained by the drug's reinforcing effect. Certainly, people do not start taking heroin so that they will become physically dependent on it and feel miserable when they go without it. Instead, they begin taking it because it makes them feel good.

Even though the withdrawal effects of heroin make it difficult to stop taking the drug, these effects alone are not sufficient to keep most people hooked. In fact, when the cost of their heroin habit gets too high, some addicts quit cold turkey. Doing so is not as painful as most people believe; withdrawal symptoms have been described as similar to a bad case of the flu—unpleasant, but survivable. After a week or two, when their nervous systems have adapted to the absence of the drug, these addicts may recommence their habit, which now costs less to sustain. If the only reason for taking the drug were to avoid unpleasant withdrawal symptoms, addicts would be incapable of following this strategy. The reason that people take—and continue to take—drugs such as heroin is that the drugs give them a pleasurable "rush"; in other words, the drugs have a reinforcing effect on their behavior.

There are two other kinds of evidence that contradict the assertion that drug addiction is caused by physical dependence. First, some very potent drugs, including cocaine, do not produce physical dependency. That is, people who take the drug do not show tolerance; and if they stop, they do not show withdrawal symptoms. As a result, experts believed for many years that cocaine was a relatively innocuous drug, not in the same league as heroin. Obviously, they were wrong: Cocaine is even more addictive than heroin. As a matter of fact, laboratory animals that can press a lever and give themselves injections of cocaine are more likely to die than those who can give themselves injections of heroin. Second, some drugs produce physical dependence (tolerance and withdrawal symptoms) but are not abused (Jaffe, 1985).

The reason for this is that they do not have reinforcing effects on behavior—they are simply not fun to take.

What Should We Conclude?

The most important lesson we can learn from the mistaken distinction between "physiological" and "psychological" addiction is that we should never underestimate the importance of "psychological" factors. After all, given that behavior is controlled by circuits of neurons in the brain, even "psychological" factors involve physiological mechanisms. People often pay more attention to physiological symptoms than psychological ones—they consider them more "real." But behavioral research has now shown that an exclusive preoccupation with physiology can hinder our understanding of the causes of addiction.

Interim Summary

Drugs and Behavior

Many chemicals found in nature have behavioral effects, and many more have been synthesized in the laboratory. Drugs can facilitate or interfere with synaptic activity. Facilitators include drugs that cause the release of a neurotransmitter (such as the venom of the black widow spider); drugs that directly stimulate postsynaptic receptors, thus duplicating the effects of the neurotransmitter itself (such as nicotine); and drugs that inhibit the reuptake of a neurotransmitter (such as amphetamine and cocaine). Drugs that interfere with synaptic activity include those that inhibit the release of a neurotransmitter (such as botulinum toxin) and those that block receptors (such as curare).

In the brain most synaptic communication is accomplished by two neurotransmitters: glutamate, which has excitatory effects, and GABA, which has inhibitory effects. Acetylcholine (ACh) controls muscular movements and is involved in control of REM sleep, activation of the cerebral cortex, and modulation of a brain structure involved in memory. Nicotine stimulates ACh receptors, and curare blocks them (and causes paralysis). Neostigmine, which is used to treat myasthenia gravis, suppresses the destruction of ACh by an enzyme. The monoamines also modulate important brain functions. Dopamine (DA) facilitates movements and plays a role in reinforcing behaviors. L-DOPA, which stimulates production of DA, is used to treat Parkinson's disease; and cocaine produces reinforcing effects on behavior by blocking the reuptake of dopamine. Drugs that block dopamine receptors are used to treat the symptoms of schizophrenia. The release of norepinephrine (NE) increases vigilance, and NE-secreting neurons play a role in the control of REM sleep. The release of serotonin helps suppress aggressive behavior and risk-taking behavior, and drugs that inhibit the reuptake of serotonin are used to treat anxiety disorders, depression, and obsessive-compulsive disorder.

Most peptides serve as neuromodulators, which resemble neurotransmitters but travel farther and are dispersed more widely, where they can modulate the activity of many neurons. The best-known neuromodulators are the endogenous opioids, which are released when an animal is engaged in important behavior. They serve to reduce pain and reinforce the ongoing behavior. Anandamide, the most important of the endogenous cannabinoids, helps regulate the release of many neurotransmitters. THC, the active ingredient in marijuana, acts on cannabinoid receptors and mimics the effects of anandamide. Cannabinoids have some beneficial effects but also impair short-term memory.

Opiates produce tolerance and withdrawal symptoms, which make their habitual use increasingly expensive and make quitting more difficult. But the primary reason for addiction is the reinforcing effect, not the unpleasant symptoms produced when an addict tries to quit. Tolerance appears to be produced by homeostatic mechanisms that counteract the effects of the drug. The distinction between "physiological" addiction (complete with tolerance and withdrawal effects) and "psychological" addiction (lacking these effects) has obscured the true cause of addiction: the reinforcing effect of the drug. Cocaine was once thought to be relatively harmless because it does not produce "real" (that is, physiological) addiction; obviously, we now know better.

QUESTIONS TO CONSIDER

1. As we saw, opioids are useful neuromodulators because they encourage an animal to continue fighting or mating. Can you think of other behaviors that might be influenced by neuromodulators? Can you think of mental or behavioral problems that might be caused if too much or too little of these neuromodulators were secreted?
2. Suppose that someone takes a drug for anxiety. Suppose further that she is planning to go out for drinks with friends. Her husband advises her to enjoy an evening with her friends but not to have any drinks. Why is this a good suggestion?
3. Many useful drugs have been found in nature, and more are yet to be discovered. What are some of the consequences of deforestation, especially of tropical forests with unusually rich diversity of species? Who owns the resources and the drugs that are discovered—the indigenous people who live there? The governments of the countries where the resources are located? The pharmaceutical companies that extract and develop purified forms of the substances?
4. If you were in charge of the research department of a pharmaceutical company, what new behaviorally active drugs would you seek? Analgesics? Antianxiety drugs? Antiaggression drugs? Memory-improving drugs? Should behaviorally active drugs be taken only by people who clearly have afflictions such as schizophrenia, depression,

or obsessive-compulsive disorder? Or should we try to find drugs that help people who want to improve their intellectual performance or social adjustment or simply to feel happier?

Study of the Brain

As Chapter 1 explained, many scientists are interested in the brain, and many psychologists are involved in brain research with humans and laboratory animals. We now have at our disposal a range of research methods that would have been impossible to imagine just a few decades ago. We have ways to identify neurons that contain particular chemicals. We have ways to use special microscopes to observe particular ions entering living neurons when the appropriate ion channels open. We have ways to inactivate individual genes or to insert new genes in laboratory animals to see what happens to the animals' physiology and behavior. We have ways to view details of the structure of a living human brain and to study the activity of various brain regions while the person is performing various perceptual or behavioral tasks. In fact, just listing and briefly describing these methods would take up an entire chapter. In this section I will describe only the most important research methods, which will give you a taste of the research performed by physiological psychologists.

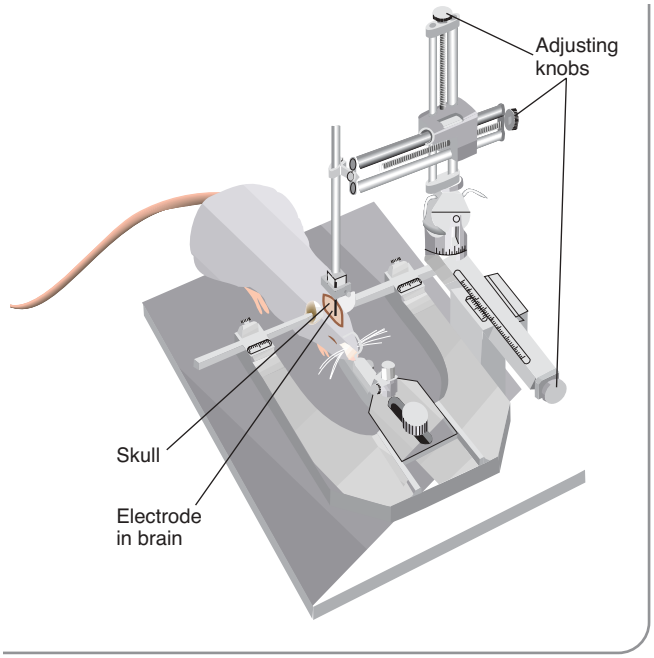
Experimental Ablation

The earliest research method of physiological psychology involved the study of brain damage. As Chapter 1 described, Pierre Flourens developed the method of experimental ablation in studies with laboratory animals, and Paul Broca applied this method when he studied a man whose brain damage had destroyed his language abilities.

To study the effect of experimental brain disruption on animal behavior, the investigator produces a **brain lesion**, an injury to a particular part of the brain, and then studies the effects of the lesion on the animal's behavior. Of course, researchers do not deliberately damage the brains of humans in order to study their functions. Instead, like Paul Broca, we study the behavior of people whose brains have been damaged by a stroke, by disease, or by head injury. If particular behaviors are disrupted, we can conclude that the damaged part of the brain must somehow be involved in those behaviors.

To produce a brain lesion in laboratory animals, the researcher must follow the ethical rules described in Chapter 2. The researcher first anaesthetizes an animal, prepares it for surgery, and drills a hole in its skull. In most cases the region under investigation is located deep within the brain. To reach this region, the investigator uses a special device called a **stereotaxic apparatus** to insert a fine wire (called an electrode) or a thin metal tube (called a cannula) into a particular location in the brain. (The term *stereotaxic* refers to the ability to manipulate an object in three-dimensional space. See **Figure 4•18**.)

FIGURE 4•18 A stereotaxic apparatus, used to insert a wire or a cannula into a specific portion of an animal's brain.



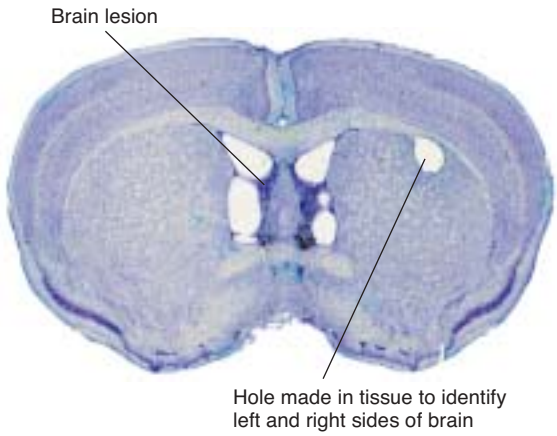
Once the correct region is located, its function can be altered. Experimenters can produce *electrolytic lesions* by passing an electrical current through the electrode, which produces heat that destroys a small portion of the brain around the tip of the electrode. Alternatively, they may establish *excitotoxic lesions* by injecting a chemical through the cannula that overstimulates neurons in the region around the tip of the cannula, which kills the neurons. After a few days the animal recovers from the operation, and the researcher can assess its behavior. Later, the investigator can remove the animal's brain from the skull, slice it, and examine it under a microscope to determine the true extent of the lesion. (See **Figure 4•19**.)

Obviously, researchers studying the behavior of a person with brain damage cannot remove the brain and examine it (unless the person happens to die and the family consents to an autopsy for this purpose). This means that researchers seldom have the opportunity to examine the brains of patients they have studied. Fortunately, the development of brain scanners permits us to determine the location and extent of damage to a living brain.

Visualizing the Structure of the Brain

Brain scanning techniques were originally developed to permit physicians to determine the causes of patients' neurological symptoms by locating regions of brain damage, visualizing brain tumors, or revealing abnormalities in

FIGURE 4•19 A brain lesion made with the aid of a stereotaxic apparatus. The photograph shows a thin slice of a mouse brain, stained with a dye that shows the location of cell bodies.



brain structure caused by faulty development. But once researchers gained the ability to see the three-dimensional structure of the brain, they could correlate brain damage or abnormalities in brain development with the observations they had made of the behavior and abilities of patients they had studied.

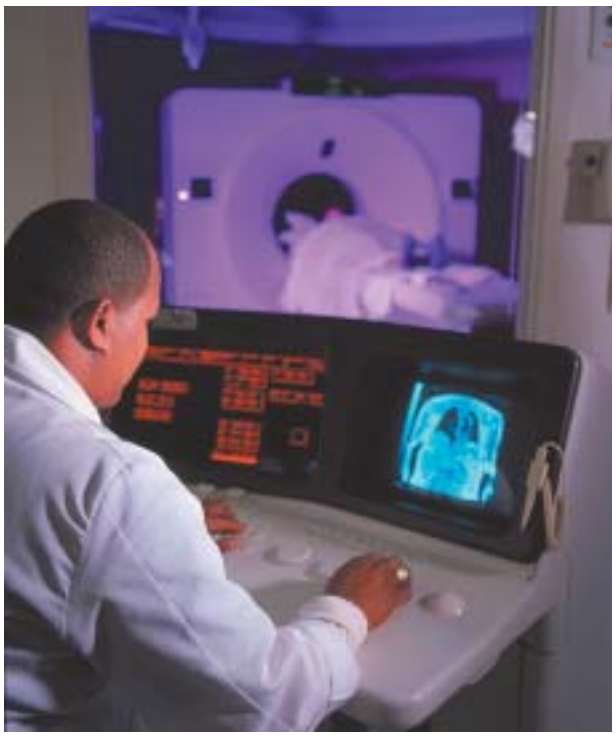
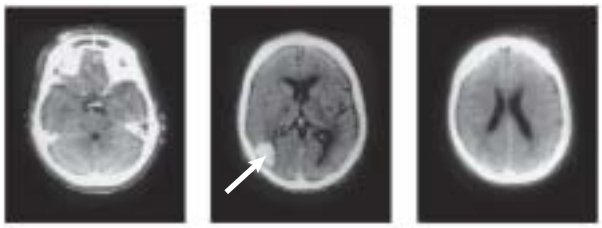


FIGURE 4•20 A patient whose brain is being scanned by a computerized tomography (CT) scanner.
(Photo © Casey McNamara/Index Stock Imagery, Inc.)

FIGURE 4•21 CT scans from a patient with a brain lesion caused by a damaged area (the white spot in the lower left corner of scan 2). Because left and right are traditionally reversed on CT scans, the damaged area is actually in the right hemisphere.
(Courtesy of Dr. J. McA. Jones, Good Samaritan Hospital, Portland, Oregon.)

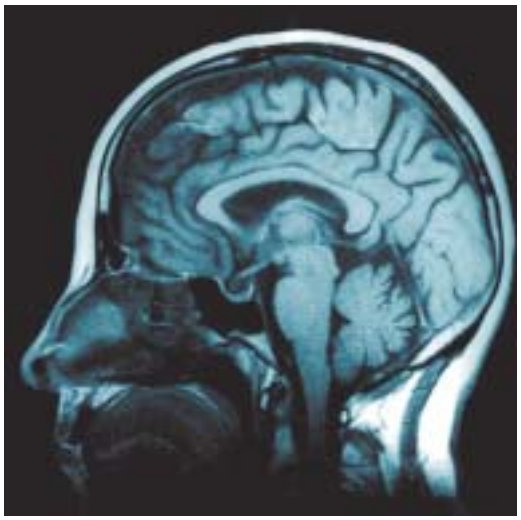


The first machine to reveal the three-dimensional structure of the brain was the **CT scanner** (see **Figure 4•20**). (CT stands for *computerized tomography*. *Tomos,* meaning "cut," describes the CT scanner's ability to produce a picture that looks like a slice of the brain. The device is often called a *CAT scanner*—the *A* is for *axial*—but the neurologists I've talked with use the term *CT*. They probably think that *CAT* sounds a little too cute.) The scanner sends a narrow beam of X-rays through a person's head. The beam is moved around the head, and a computer calculates the amount of radiation that passes through it at various points along each angle. The result is a two-dimensional image of a "slice" of the person's head, parallel to the top of the skull.

Figure 4•21 shows three CT scans of the brain of a patient with an injury—Miss S., whose case is described in this chapter's opening vignette. The scans are arranged from the bottom of the brain (scan 1) to the top (scan 3). You can easily see the damaged area, a white spot, in the lower left corner of scan 2.

A more recent brain-imaging technique is known as **magnetic resonance imaging (MRI)**. MRI scans are produced by placing a person's head within a strong magnetic field. This field causes the molecules within its influence to become aligned with the lines of magnetic force. A radio signal is then generated around the person, which has the effect of tilting these aligned atoms, just as you might nudge a spinning top and cause it to wobble. The scanner measures the time it takes the molecules to stop wobbling and recover to their aligned state. Because different molecules take different times to recover, an image can be constructed that distinguishes between different materials within the head, such as gray matter, white matter, and cerebrospinal fluid. MRI scanners can produce images of the brain with higher resolution than those produced by CT scanners (see **Figure 4•22**). However, CT scanners are still in use because they are cheaper and do not contain magnets; the magnetism exerted by an MRI scanner can interact with objects such as pacemakers or metal clips that have been placed in a patient's body.

FIGURE 4•22 An MRI scan of a human brain.
(Photo copyright © ISM/Phototake. All rights reserved.)



Measuring the Brain's Activity

Because the brain's physiology involves both electrical and chemical processes, measuring techniques have been developed for each. **Microelectrodes** are extremely thin wires that able to detect the electrical currents of individual neurons. With suitable amplification, microelectrodes can be used to measure the minute electrical changes of individual action potentials. Arrays of dozens of ultrathin wires can even enable a researcher to simultaneously record the activity of dozens of neurons. Other electrical recording techniques involve larger electrodes placed outside the skull. These electrodes can measure the electrical activity of large groups of neurons. For example, the **electroencephalogram (EEG)** makes a graph on a long sheet of paper of the brain's activity, recorded through metal disks attached to a person's skull (see **Figure 4•23**). The EEG can be used to diagnose seizure disorders (epilepsy) and to monitor the various stages of sleep (described in Chapter 9).

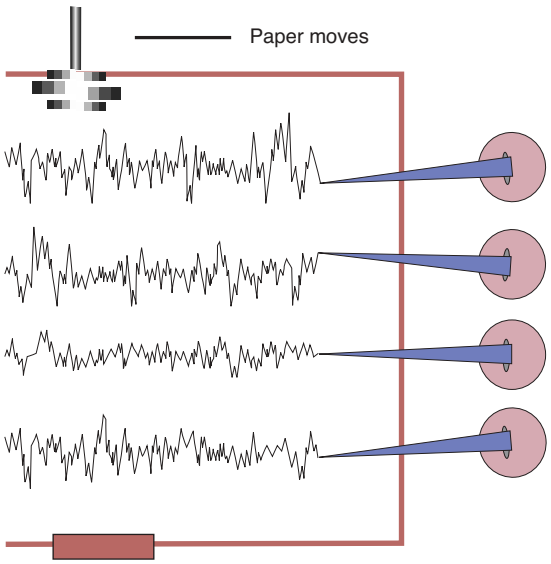
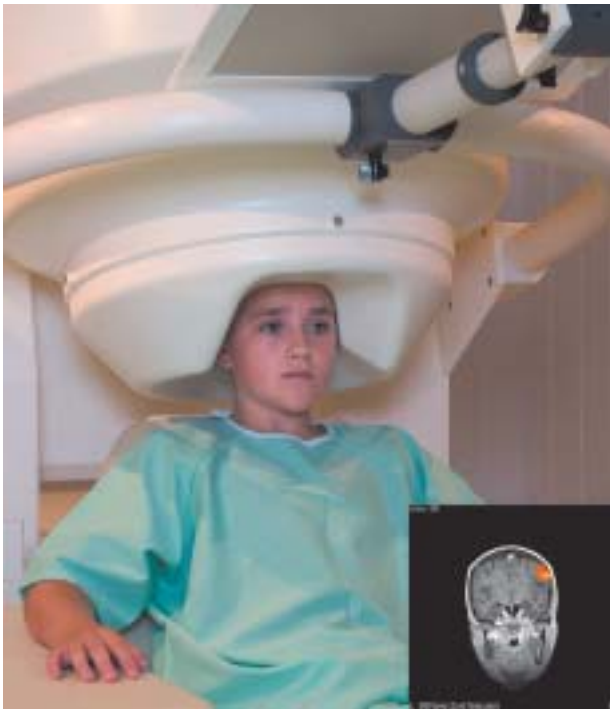


FIGURE 4•23 A record from an EEG machine. The pens trace changes in the electrical activity of the brain, recorded by electrodes placed on a person's scalp.

FIGURE 4•24 Magnetoencephalography. The recording apparatus enclosing a person's head is shown on the monitor to the left. The regions of increased electrical activity are shown in the inset in the lower right, superimposed on an image of the brain derived from an MRI scan.
(Photo courtesy of VSM MedTech Ltd.)



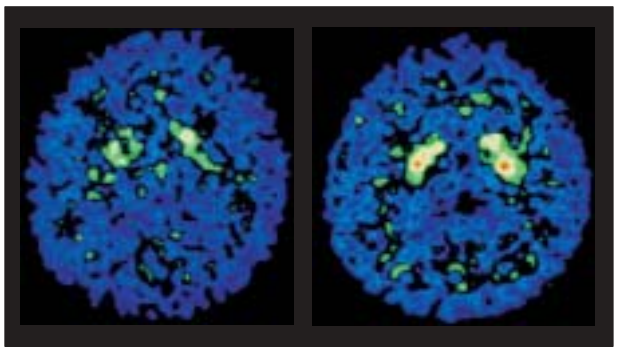
In another method, known as **magnetoencephalography (MEG),** a recording device detects the minute magnetic fields that are produced by the electrical activity of neurons in the cerebral cortex. These devices can be used clinically—for example, to find brain abnormalities that produce seizures so that they can be removed surgically. MEG can also be used in experiments to measure regional brain activity that accompanies the performance of various behaviors or cognitive tasks. (See **Figure 4•24**.)

The metabolic activity of specific brain regions can be measured by two special scanning methods: *PET scanning* and *functional MRI scanning*. **Positron emission tomography (PET)** takes advantage of the fact that when radioactive molecules decay, they emit subatomic particles called positrons. The first step in PET is to give a person an injection of a radioactive chemical that accumulates in the brain. (The chemical eventually breaks down and leaves the cells.

The dose given to humans is harmless.) The person's head is placed in the PET scanner, which detects the positrons. The computer determines which regions of the brain have taken up the radioactive chemical, and it produces a picture of a slice of the brain, showing which regions contain the highest concentrations of the chemical. Researchers can use a wide variety of chemicals. For example, they can use a chemical that accumulates in metabolically active cells, in which case the PET scan reveals the brain regions that are most active. They can also use chemicals that bind with particular receptors (for example, serotonin receptors) and determine which brain regions contain these receptors.

Figure 4•25 shows yet another use of PET. The scans were taken before and after dopamine-secreting neurons were surgically implanted into the brain of a person with Parkinson's disease. The scan shows an increase in the amount of dopamine in a region of the brain that controls movements, revealed by the presence of a radioactive chemical that becomes incorporated into molecules of dopamine.

The most recent development in brain imaging is **functional MRI (fMRI).** Biomedical engineers have devised modifications to existing MRI scanners that measure the rate of metabolism in regions of the brain by detecting levels of oxygen in the brain's blood vessels. Functional MRI scans have a higher resolution than PET scans, they can be acquired much more rapidly, and they do not require the production of radioactive chemicals with very short half-lives, which is expensive. Thus, fMRI has become the preferred method of measuring the activity of the human brain. (See **Figure 4•26**.)



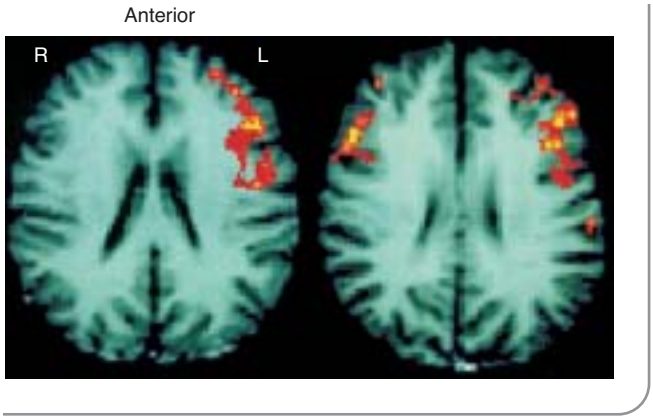
(a) (b)

FIGURE 4•25 PET scans of a patient with Parkinson's disease showing accumulation of radioactive L-DOPA in a brain region involved in movement that receives input from terminal buttons that secrete dopamine. (a) Preoperative scan. (b) Scan taken 13 months after a transplant of dopamine-secreting cells. The increased uptake of L-DOPA indicates that the transplant was secreting dopamine.

(Adapted from Widner, H., Tetrud, J., Rehncrona, S., Snow, B., Brundin, P., Gustavii, B., Björklund, A., Lindvall, O., and Langston, J. W. *New England Journal of Medicine,* 1992, *327,* 1556–1563. Copyright © 1992 Massachusetts Medical Society. All rights reserved.)

FIGURE 4•26 Functional MRI scans of human brains, showing localized increases in neural activity of males (left) and females (right) while they were judging whether pairs of written words rhymed.

(From Shaywitz, B. A., et al., *Nature,* 1995, *373,* 607–609. By permission.)



Stimulating the Brain's Activity

So far, this section has discussed studying the brain by destroying parts of it (or observing the effects of such destruction when it occurs naturally), visualizing the brain's structure, and measuring the brain's activity. Another research method artificially activates neurons in particular parts of the brain to see what effects this stimulation has on behavior. For example, weak electrical stimulation of one part of a laboratory animal's brain, just sufficient to trigger action potentials in axons in that region, has a reinforcing (rewarding) effect on the animal's behavior. If the animal has the opportunity to press a lever that delivers a brief pulse of electricity through electrodes that have been surgically implanted in its brain, it will do so—up to thousands of times an hour. (See **Figure 4•27**.) The implication is that the brain has a system of neurons involved in reinforcement; and this hypothesis is confirmed by recording studies in both humans and laboratory animals, which indicate that the neurons activated by this stimulation also are activated by events that reinforce behavior, such as the administration of food, water, or addictive drugs. One functional MRI study of heterosexual male college students even found that the sight of a photograph of a beautiful woman activates this region (Aharon et al., 2001).

As we saw in the previous subsection, neural activity induces magnetic fields that can be detected by means of magnetoencephalography. Similarly, magnetic fields can be used to stimulate neurons by inducing electrical currents in brain tissue. **Transcranial magnetic stimulation (TMS)** uses a coil of wires, usually arranged in the shape of the numeral 8, to

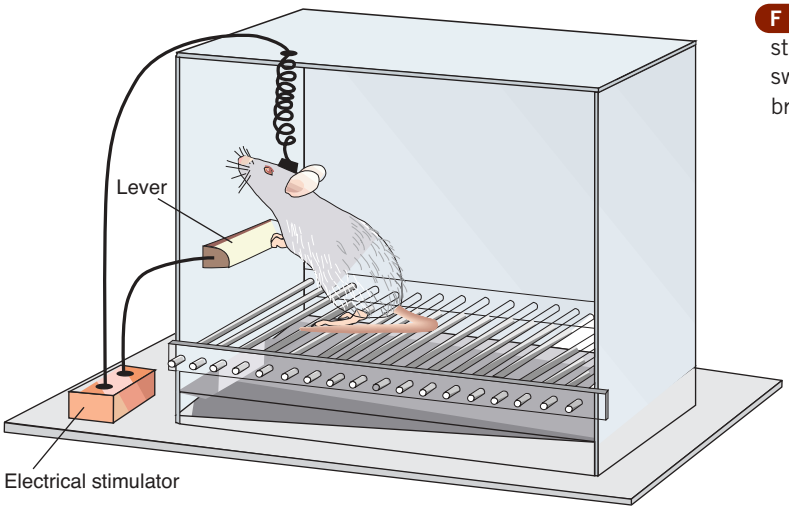


FIGURE 4•27 An example of an electrical stimulation experiment. When the rat presses the switch, it receives a brief pulse of electricity to its brain through electrodes.

stimulate neurons in the human cerebral cortex. The stimulating coil is placed on top of the skull so that the crossing point in the middle of the 8 is located immediately above the region to be stimulated. Pulses of electricity send magnetic fields that activate neurons in the cortex. Because the processing of information in the cerebral cortex involves intricate patterns of activity in particular circuits of neurons, the stimulation disrupts normal activity in that region of the brain. For example, stimulation of a particular region of the cerebral cortex will disrupt a person's ability to detect movements in visual stimuli. These findings confirm the results of recording and lesion studies with laboratory animals and studies of people with brain damage, which indicate that this region in involved in perception of visual movement. In addition, TMS has been used to treat the symptoms of mental disorders such as depression.

Figure 4•28 shows an electromagnetic coil used in transcranial magnetic stimulation and its placement on a person's head.

Altering Genetics

Thanks to the advances in genetics discussed in Chapter 3, neuroscientists can now manipulate genetic mechanisms that control the development of the nervous system. For example, a **targeted mutation** (a genetic "knockout") can be produced in mice. This procedure inactivates a gene—for example, the gene responsible for producing a particular neurotransmitter or a particular receptor. The effects of the knockout on the animals' behavior suggest what the normal role of the neurotransmitter might be. For example, a targeted mutation that prevents production of a particular peptide causes a hereditary sleep disorder known as narcolepsy, which, we now know, is caused by degeneration of the neurons that secrete this peptide (Chemelli et al., 1999).

Researchers also can insert genes into animals' DNA, which can alter the development of the brain or the functioning of particular types of neurons after the animals are born. For example, Tang and colleagues (1999) found that a genetic modification that increased the production of a particular type of receptor increased the animals' learning ability in a particular task. Along with the findings of other experiments, these results suggest that these receptors are involved in producing changes in synapses that are responsible for memory formation.

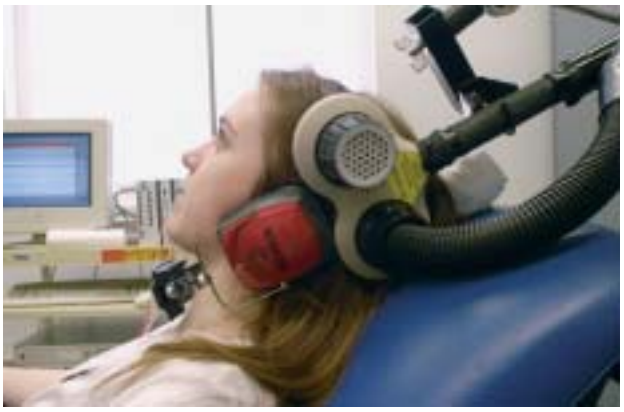


FIGURE 4•28 Transcranial magnetic stimulation. The coil applies electromagnetic stimulation of the brain, which interferes with the region of the cerebral cortex below the crossing point of the figure 8 of the coil.

Biology and Culture

Environmental Effects on Brain Development

One of the oldest controversies in psychology concerns the respective roles of nature and nurture in human development. Normally, when people ask, "Is it caused by biological or social factors?" or "Is it innate or learned?" they are referring to the origins of a particular behavior, talent, or personality trait. Almost always, biology and innateness are placed on the "nature" side of the dichotomy; social factors and learning are placed on the "nurture" side. Historically, this was considered to be an appropriate division.

As you will see throughout this book, however, most modern psychologists consider the nature–nurture issue to be a relic of outdated thinking about behavior. That is, as we learn more about the ways in which behaviors, talents, and personality traits develop, we discover that both types of factors enter in: biological and social, hereditary and cultural. The task of the modern psychologist is not to find out which one of these factors is more important but to discover the particular roles played by each of them and to determine the ways in which they interact.

As you know, the body develops according to a program established by the genes. The only way the genes can influence our personalities and behavior is through their effect on physical development. Brain development obviously plays a critical role in this regard, but the endocrine system and the structure of other parts of our bodies have important effects, too. For example, having a male or female body certainly influences our behavior and affects the way we are treated by other people. Because these factors also may are so important, many people believe that heredity is the sole influence on normal development of the brain. Few consider the possibility that environmental factors also may have important influences on the normal development of the brain, and that this development may extend not just to the early months of infancy, but through to adulthood. Modern methods have begun to show us how the brain responds to experience.

Evidence for the Effects of Experience on Brain Development

Can the environment affect brain development? In the 1960s Mark Rosenzweig and his colleagues began a research program designed to examine this question (Rosenzweig and Bennett, 1996). The researchers divided litters of rats and placed the animals into two kinds of environments: enriched and impoverished. The enriched environment contained such things as running wheels, ladders, slides, and toys that the rats could explore and manipulate. The researchers changed these objects every day to maximize the animals' experiences and to ensure that they would learn as much as possible. In contrast, the impoverished environments were plain cages in a dimly illuminated, quiet room.

Rosenzweig and his colleagues found many differences in the brains of the animals raised in the two environments. The brains of rats raised in the enriched environment had a thicker cerebral cortex, a better blood supply, more protein content, and more acetylcholine (a neurotransmitter that appears to play an important role in learning). Subsequent studies have found changes on a microscopic level as well. Greenough and Volkmar (1973) found that the neurons of rats raised in the enriched environment had larger and more complex dendritic trees. Turner and Greenough (1985) found that synapses in their cerebral cortexes were larger and that more synapses were found on each of their neurons. And changes occur even in the adult brain: Sirevaag, Black, Shafron, and Greenough (1988) found that when rats were placed in an enriched environment between the ages of 30 and 60 days (young adulthood), the capillaries in their visual cortexes grew more branches and their surface areas increased, presumably to accommodate the growth that was stimulated by the experience.

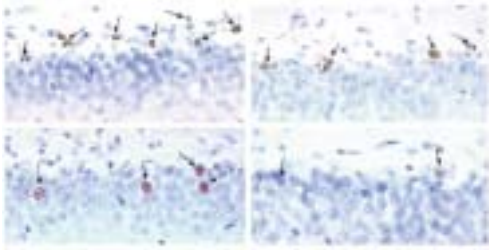
Even the brains of adult humans can be modified by experience. If you've ever visited London, England, you probably found it to be a confusing city. Built around archaic footpaths and courtyards, its modern streets are a baffling maze, made all the worse by modern one-way traffic laws. To be a taxi driver in this environment and navigate this maze requires exceptional spatial skill. London taxi drivers spend years learning the street layout, and only after passing a demanding exam are they licensed to operate on the street. Learning takes place in the brain; the drivers' navigational ability must be a result of changes in the neural circuitry of their brain that occurred during their years of training. Using MRI technology, Maguire and colleagues (2000) found that these taxi drivers' brains were physically different from those of other Londoners—a portion of the hippocampus, a part of the brain known to be involved in learning, was enlarged. In fact, the size of this region of a cabby's hippocampus was positively correlated with his or her ability to navigate the London streets.

Although a reasonable explanation for the enlarged hippocampus of London cabbies is that their training induced growth in this structure, it is possible that some people are born with larger hippocampuses and that these are the people most likely to be able to successfully complete the training and pass the exam. (As you will recall from Chapter 2, correlation does not prove causation.) Let's examine some evidence to assess the plausibility of the hypothesis that learning can change the size of parts of the brain.

One possibility is that learning stimulates the growth of new synaptic connections, which would entail the growth of new dendrites and branches of axons. These alterations might then cause an expansion of the brain regions where the growth occurs. There is some evidence for this phenomenon. For example, the region of the brain that is devoted to the analysis of sensory information from the fingers of the left hand is larger in musicians who play stringed instruments. (These fingers are used to press the strings.) A similar phenomenon is seen in the brains of blind people who learn to read Braille (Elbert et al., 1995; Sadato et al., 1996).

FIGURE 4•29 Effects of learning on neurogenesis, seen in sections through a part of the hippocampus of rats that received training on a learning task or were exposed to a control condition that did not lead to learning. Arrows indicate newly formed cells.

(From Leuner, B., Mendolia-Loffredo, S., Kozorovitskiy, Y., Samburg, D., Gould, E., and Shors, T. J. *Journal of Neuroscience,* 2004, *24,* 7477–7481. Copyright © 2004 by the Society of Neuroscience. Reprinted by permission.)



What about the possibility that learning can encourage the growth of new neurons? For many years researchers believed that *neurogenesis* (production of new neurons) cannot take place in the fully developed brain. However research has now shown this belief to be incorrect—the adult brain contains some **stem cells** that can divide and produce new neurons. (Stem cells are undifferentiated cells that can divide and produce any of a variety of differentiated cells.) Researchers can detect the presence of newly produced cells by administering a small amount of a radioactive form of one of the chemicals that stem cells use to produce the DNA needed for neurogenesis. The next day, the investigators remove the animals' brains, stain slices of the brain with a special dye, and examine them under a microscope. Such studies have found evidence for neurogenesis in just two parts of the adult brain: the hippocampus and the *olfactory bulb,* which is involved in the sense of smell (Doetsch and Hen, 2005). Evidence indicates that exposure to new odors can increase the survival rate of new neurons in the olfactory bulbs, and training on a learning task can enhance neurogenesis in the hippocampus. (See **Figure 4•29**.) So perhaps learning to navigate in London really does increase the size of a cabby's hippocampus. In addition, depression or exposure to stress can suppress neurogenesis in the hippocampus, and drugs that reduce stress and depression can reinstate neurogenesis.

Environmental stimulation does not begin at the time of birth. While in the uterus, fetuses feel the movements of their mothers' bodies and hear the sounds of their mothers' voices and sounds from the external environment that pass through the abdominal wall. After they are born, infants receive much environmental stimulation when they are nursed, when they are bathed, when their diapers are changed, and when they are simply held and cuddled. This stimulation clearly contributes to normal development. When infants are born prematurely and must be placed in isolators, they are deprived of the stimulation that occurs in the uterus and receive less handling than do normal full-term infants. Several studies have found that gentle stroking of premature infants can reduce the effects of this environmental deprivation; it increases their growth rates and rates of motor development (Solkoff, Yaffe, Weintraub, & Blase, 1969; Solkoff and Matuszak, 1975). According to evidence from experiments using infant rats reviewed by Schanberg and Field (1987), stroking and handling an infant may stimulate the release of hormones necessary for normal growth and development (including development of the brain).

Conclusions

We've looked at only a few of the many effects of environment on physiological development that researchers have discovered so far. It is clear that the brain does not develop in a vacuum. Instead, its development is shaped and guided by interactions with the environment. One of the most exciting recent discoveries about the adult brain is that it is still capable of growing new neurons, and that growth and survival of these neurons can be affected by interactions with the environment. Consequently, development is not a process confined to the immature. Not only has the nature–nurture issue become a relic of the past; so has the assumption that physiology is solely a product of heredity. Interactions between genes and environment begin early in development and continue throughout life.

Interim Summary

Study of the Brain

The study of the brain, with all its complexity, requires a variety of research methods. Some methods alter the brains of laboratory animals. These methods may include selective destruction of parts of the brain, recording of the brain's electrical or chemical activity, electrical or chemical stimulation of specific brain regions, or modification of the parts of the genetic code that affect neural processes. Electroencephalography and magnetoencephalography reveal the electrical events in the human brain. Other methods, including CT scans, PET imaging, and structural and functional MRI scans, provide images of the structure and activity of the human brain.

The nature–nurture controversy was important in the past, when psychologists asked whether particular behaviors, talents, or personality traits were caused by hereditary factors ("nature") or by experience ("nurture"). This controversy is now over, because psychologists realize that almost all characteristics are affected by both factors. What is less generally recognized is that the normal development of the brain—often assumed to be programmed solely by hereditary factors—is also affected by the environment.

QUESTIONS TO CONSIDER

1. Would you like to have an electrode placed in your brain so that you could see what reinforcing (rewarding) brain stimulation feels like? Why or why not?
2. Suppose it were necessary to make an MRI scan of your brain. Would you want to see the scans afterwards?
3. Suppose you had an fMRI scanner and many volunteers. You could present various types of stimuli while scans were being taken, and you could have the volunteers perform various types of mental tasks and behaviors that did not involve their moving around. What kinds of experiments would you perform?
4. Although the basic program that controls brain development is contained in our chromosomes, environmental factors also can influence this process. Why do you think the process of development is not completely automatic and programmed? What is the evolutionary benefit of letting the environment influence it? Would humans be better off if development were simply automatic, or does such flexibility have some potential benefits?

Control of Behavior and the Body's Physiological Functions

As mentioned earlier, the brain has three major functions: controlling behavior, processing and retaining information about the environment, and regulating the physiological functions of the body. The first two roles look outward toward the environment, and the third looks inward. The outward-looking roles include several functions: perceiving events in the environment, learning about them, making plans, and acting. The inward-looking role requires the brain to measure and regulate internal characteristics such as body temperature, blood pressure, and nutrient levels. The outward-looking roles are, of course, of particular interest to psychology. This section will examine how the brain performs all three kinds of functions, beginning with the portions of the brain that control behavior and process information.

The cells of the brain are organized in *modules*—clusters of neurons that communicate with one another. Modules are connected to other modules, receiving information from some of them, processing this information, and sending the results on to others. Particular modules have particular functions, just as the transistors, resistors, and capacitors in a computer chip do. The task of psychologists interested in understanding the brain is to identify the modules, discover their functions, trace their interconnections, and understand the ways in which the activities of these complex assemblies give rise to our perceptions, memories, feelings, and actions. Despite the progress we have made so far, the end of this task is not even remotely in sight.

Organization of the Cerebral Cortex

If we want to understand the brain functions most important to the study of behavior—perceiving, learning, planning, and moving—we should start with the cerebral cortex. Because we will be discussing the various regions of the cerebral cortex, it will be good to start with the names used for them. The cerebral cortex contains a large groove, or fissure, called the **central fissure.** The central fissure provides an important dividing line between the anterior (front) part of the cerebral cortex and the posterior (back) regions. (See **Figure 4•30**.)

As Figure 4.30 shows, the cerebral cortex is divided into four areas, or *lobes*, named for the bones of the skull that cover them: the frontal lobe, parietal lobe, temporal lobe, and occipital lobe. Of course, the brain contains two of each lobe, one in each hemisphere, on each side of the brain. The **frontal lobe** (the "front") includes everything in front of the central fissure. The **parietal lobe** (the "wall") is located on the side of the cerebral hemisphere, just behind the central fissure, in back of the frontal lobe. The **temporal lobe** (the "temple") juts forward from the base of the brain, beneath the frontal and parietal lobes. The **occipital lobe** (*ob*, "against"; *caput*, "head") lies at the very back of the brain, behind the parietal and temporal lobes. The discussions that follow will look in detail at the functions of each of these lobes.

Regions of Primary Sensory and Motor Cortex We become aware of events in our environment by means of the five major senses: vision, audition, olfaction (smell), gustation (taste), and the somatosenses (the "body" senses: touch, pain, and temperature). Three areas of the cerebral cortex receive information from the sensory organs. The **primary visual cortex,** which receives visual information, is located at the back of the brain, on the inner surfaces of the occipital lobe. The **primary auditory cortex,** which receives auditory information, is located within the temporal lobe on the inner surface of a deep fissure in the side of the brain. The **primary somatosensory cortex,** a vertical strip near the middle of the cerebral hemispheres on the parietal lobe, receives information from the body senses. As Figure 4.30 shows, different regions of the primary somatosensory cortex receive information from different regions of the body. In addition, the base of the somatosensory cortex receives gustatory information, and a portion of the frontal lobe, not visible from the side, receives olfactory information.

The three regions of primary sensory cortex in each hemisphere receive information from the opposite side of the body. Thus, the primary somatosensory cortex of the left hemisphere learns what the right hand is holding, the left primary visual cortex learns what is happening to the person's right, and so on. The connections between the sensory organs and the cerebral cortex are said to be **contralateral** (*contra,* "opposite"; *lateral,* "side"). However, the two most primitive forms of sensory information, smell and taste, are transmitted to the **ipsilateral** hemisphere. That is, the right side of the tongue and the right nostril send information to the right side of the brain.

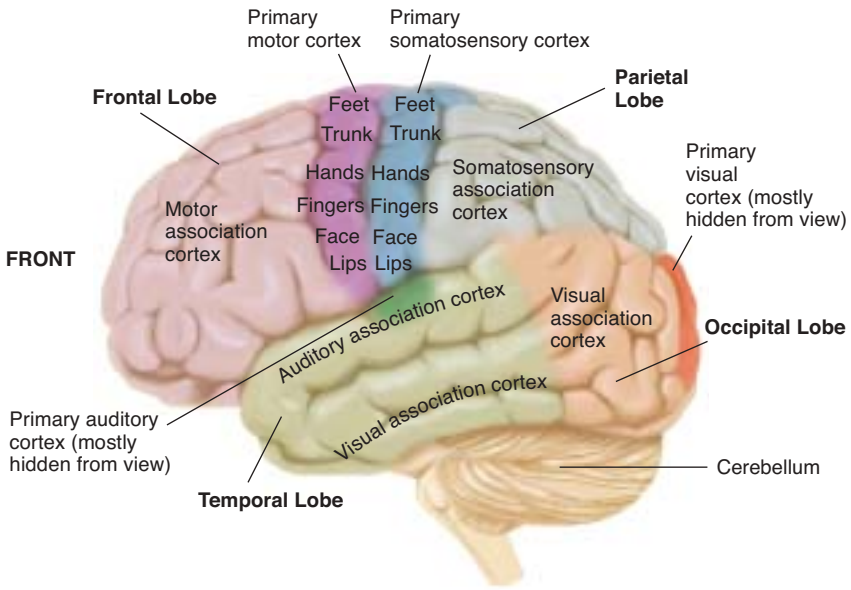


FIGURE 4•30 A side view of the human brain, showing the location of the four lobes of the cerebral cortex, the primary sensory and motor areas, and the regions of association cortex. The central fissure is the dividing line between the primary motor cortex and the primary somatosensory cortex.

The region of the cerebral cortex most directly involved in the control of movement is the **primary motor cortex** within the frontal lobe, located just in front of the primary somatosensory cortex. Neurons in different parts of the primary motor cortex are connected to muscles in different parts of the body. The connections, like those of the sensory regions of the cerebral cortex, are contralateral; the left primary motor cortex controls the right side of the body and vice versa. Thus, for example, if a neurosurgeon electrically stimulates the "hand" region of the left primary motor cortex, the patient's right hand will move. (Refer to Figure 4.30.) I like to think of the strip of primary motor cortex as the keyboard of a piano, with each key controlling a different movement. We will see shortly who the "player" of this piano is.

Association Cortex The regions of primary sensory and motor cortex occupy only a small part of the cerebral cortex. The rest of the cerebral cortex accomplishes what is done between sensation and action: perceiving, learning and remembering, planning, and moving. These processes take place in the *association areas* of the cerebral cortex. The anterior region is involved in movement-related activities, such as planning and executing behaviors. The posterior part is involved in perceiving and learning.

Each primary sensory area of the cerebral cortex sends information to adjacent regions, called the **sensory association cortex**. Circuits of neurons in the sensory association cortex analyze the information received from the primary sensory cortex; perception takes place there, and memories are stored there. The regions of the sensory association cortex located closest to the primary sensory areas receive information from only one sensory system. For example, the region closest to the primary visual cortex analyzes visual information and stores visual memories. Regions of the sensory association cortex located far from the primary sensory areas receive information from more than one sensory system; thus, they are involved in several kinds of perceptions and memories. These regions make it possible to integrate information from more than one sensory system. For example, we can learn the connection between the sight of a particular face and the sound of a particular voice. (Refer again to Figure 4.30.)

Just as regions of the sensory association cortex of the posterior part of the brain are involved in perceiving and remembering, so the frontal association cortex is involved in the planning and execution of movements. The anterior part of the frontal lobe—known as the **prefrontal cortex**—contains the **motor association cortex**. The motor association cortex controls the primary motor cortex; thus, it directly controls behavior. If the primary motor cortex is the keyboard of the piano, then the motor association cortex is the piano player.

Obviously, we behave in response to events happening in the world around us. Therefore, the sensory association cortex of the posterior part of the brain sends information about the environment—and information about what we have learned from past experience—to the motor association cortex (prefrontal cortex), which translates the information into plans and actions. (See **Figure 4•31**.)

The Thalamus If you stripped away the cerebral cortex and the white matter that lies under it, you would find the **thalamus,** located in the heart of the cerebral hemispheres. (Thalamos is Greek for "inner chamber.") The thalamus is divided into two parts, one in each cerebral hemisphere. Each part looks rather like a football, with the long axis oriented from front to back. **Figure 4•32** shows the two halves of the thalamus, along with several other brain structures that will be described later in this chapter.

The thalamus performs two basic functions. The first—and most primitive—is similar to that of the cerebral cortex. Parts of the thalamus receive sensory information, other parts

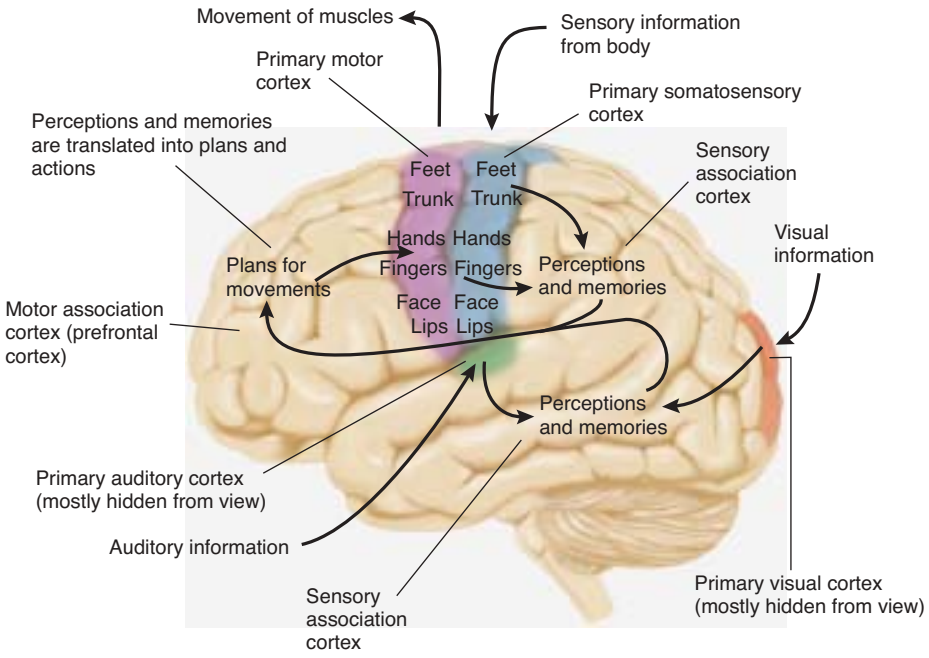


FIGURE 4•31 The relation between the association cortex and the regions of primary sensory and motor cortex. Arrows refer to the flow of information.

integrate the information, and still other parts assist in the control of movements through their influence on circuits of neurons in the brain stem. However, the second role of the thalamus—that of a relay station for the cortex—is even more important. As the cerebral hemispheres evolved, the cerebral cortex grew in size and its significance for behavioral functions increased. The thalamus took on the function of receiving sensory information from the sensory organs, performing some simple analyses, and passing the results on to the primary sensory cortex. Thus, all sensory information (except for olfaction, which is the most primitive of all sensory systems) is sent to the thalamus before it reaches the cerebral cortex.

Lateralization of Function

Although the two cerebral hemispheres cooperate with each other, they do not perform identical functions. Some functions are *lateralized*—performed by neural circuits located primarily on one side of the brain. In general, the left hemisphere participates in the *analysis* of information—the extraction of the elements that make up the whole of an experience. This ability makes the left hemisphere particularly good at recognizing *serial events*—events whose elements occur one after another. The left hemisphere also is involved in controlling serial behaviors. The serial functions performed by the left hemisphere include verbal activities, such as talking, understanding the speech of other people, reading, and writing. In general, damage to the various regions of the left hemisphere disrupts these abilities. (In a few people the functions of the left and right hemispheres are reversed.) We'll look at language and the brain in more detail in Chapters 9 and 10.

In contrast, the right hemisphere is specialized for *synthesis*; it is particularly good at putting isolated elements together to perceive things as a whole. For example, our ability to draw sketches (especially of three-dimensional objects), read maps, and construct complex objects out of smaller elements depends heavily on circuits of neurons located in the

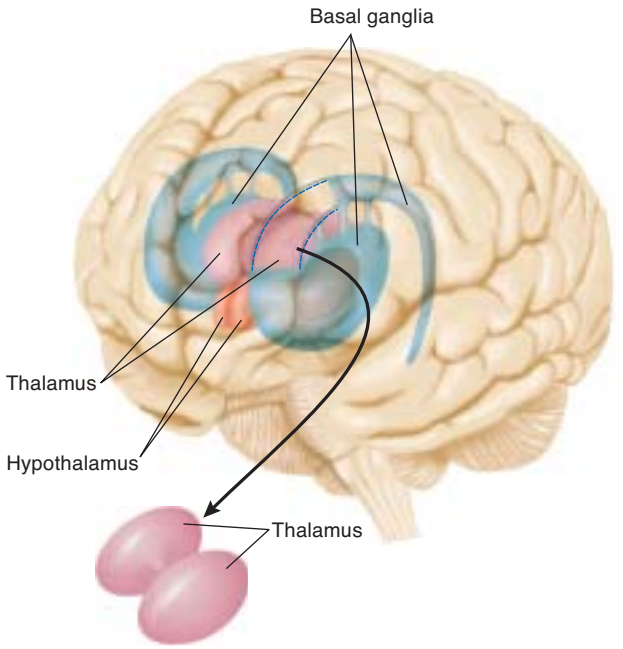


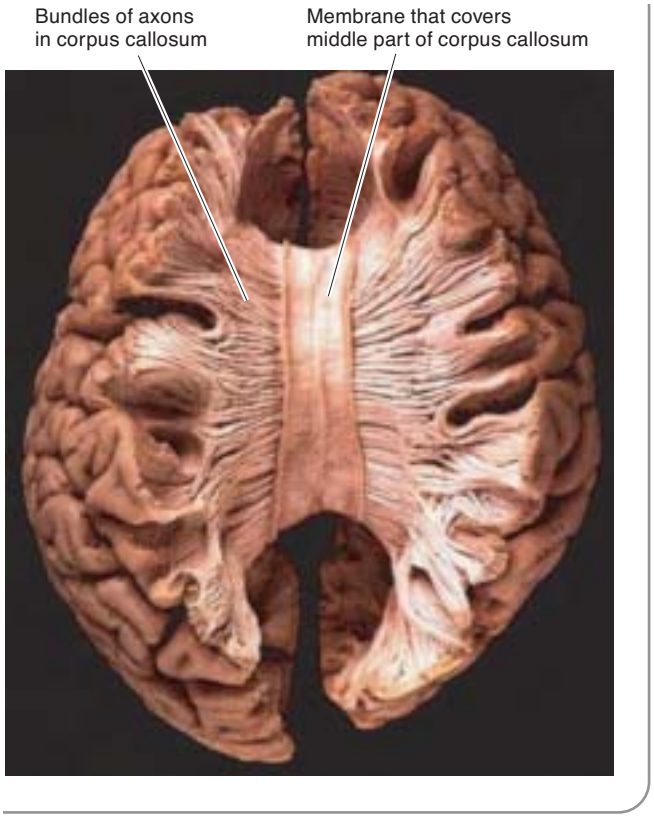
FIGURE 4•32 The location of the basal ganglia, thalamus, and hypothalamus, ghosted into a semitransparent brain.

right hemisphere. The right hemisphere is also especially involved in understanding the meaning of metaphorical statements such as "People who live in glass houses shouldn't throw stones" or the moral of stories such as the one about the race between the tortoise and the hare. Damage to the right hemisphere disrupts these abilities.

We are not aware of the fact that each hemisphere perceives the world differently. Although the two cerebral hemispheres perform somewhat different functions, our perceptions and our memories are unified. This unity is accomplished by the **corpus callosum,** a large band of axons that connects the two cerebral hemispheres. The corpus callosum connects corresponding parts of the left and right hemispheres: the left and right temporal lobes, the left and right parietal lobes, and so on. Because of the corpus callosum, each region of the association cortex knows what is happening in the corresponding region of the opposite side of the brain. **Figure 4•33** shows a photograph of a brain, viewed from above, that has been partially dissected. We see bundles of axons that pass through the corpus callosum, connecting groups of neurons in corresponding regions of the left and right hemispheres.

If the corpus callosum connects the two hemispheres and permits them to interchange information, what happens if the corpus callosum is cut? In fact, neurosurgeons sometimes deliberately cut the corpus callosum (in a procedure called the *split-brain operation*) to treat a certain type of epilepsy. As a result, the two hemispheres process information independently and sometimes even attempt to engage in competing behaviors. I'll describe the interesting effects of this operation on perceptions and consciousness in Chapter 9.

FIGURE 4•33 A photograph of a brain, viewed from above, that has been partially dissected, showing bundles of axons that pass through the corpus callosum.
(Courtesy of Terence H. Williams, Nedzad Gluhbegovic, Jean Y. Jew, and The University of Iowa Virtual Hospital.)



Vision

The primary business of the occipital lobe—and of the lower part of the temporal lobe—is seeing. Total damage to the primary visual cortex, located in the inner surface of the posterior occipital lobe, produces blindness. Because the visual field is "mapped" onto the surface of the primary visual cortex, a small lesion in the primary visual cortex produces a "hole" in a specific part of the field of vision.

The visual association cortex is located in the rest of the occipital lobe and in the lower portion of the temporal lobe. (Refer to Figure 4.30.) Damage to the visual association cortex will not cause blindness. In fact, visual acuity may be very good; people with such damage may be able to see small objects and may even be able to read. However, they will not be able to *recognize* objects by sight. For example, when looking at a drawing of a clock, these individuals may say that they see a circle, two short lines forming an angle in the center of the circle, and some dots spaced along the inside of the circle; but they will not be able to recognize what the picture shows. On the other hand, if handed a real clock, they will immediately recognize it by touch. This fact tells us that these people have not simply forgotten what clocks are. Similarly, people may fail to recognize their spouses by sight but will be able to do so from the sound of the spouses' voice. This deficit in visual perception is called **visual agnosia** (*a-*, "without"; *gnosis,* "knowledge"). We'll deal with this phenomenon further in Chapter 7.

Audition

The temporal lobe contains both the primary auditory cortex and the auditory association cortex. The primary auditory cortex is hidden from view on the inner surface of the upper temporal lobe. The auditory association cortex is located on the lateral surface of the upper temporal lobe. (Refer to Figure 4.30.) Damage to the primary auditory cortex leads to hearing losses, whereas damage to the auditory association cortex produces more complex deficits. Damage to the left auditory association cortex causes language deficits. People with such damage are no longer able to comprehend speech, presumably because they have lost the circuits of neurons that decode speech sounds. However, the deficit is more severe than that. They also lose the ability to produce meaningful speech; their speech becomes a jumble of words. We'll look again at language deficits produced by brain damage in Chapter 10.

Damage to the right auditory association cortex does not seriously affect speech perception or production, but it does

affect people's ability to recognize nonspeech sounds, including patterns of tones and rhythms. The damage also can impair the ability to perceive the location of sounds in the environment. The right hemisphere is very important in the perception of space, and the contribution of the right temporal lobe to this function is to participate in perceiving the placement of sounds.

Somatosensation and Spatial Perception

The primary functions of the parietal lobe are perception of our own body and the location of objects in the world around us. (Refer to Figure 4.30.) Damage to parts of the parietal lobe that receive information from the visual system disrupts people's ability to perceive and remember the location of items in their environment. Damage to parts of the left parietal lobe can disrupt the ability to read or write without causing serious impairment in the ability to talk and understand the speech of other people. Damage to part of the right parietal lobe can interfere with people's ability to perceive designs and three-dimensional shapes. A person with such damage can analyze a picture into its parts but has trouble integrating these parts into a consistent whole. Thus, he or she has difficulty drawing a coherent picture. (See **Figure 4•34**.)

The right parietal lobe also plays a role in people's ability to pay attention to stimuli located toward the opposite (left) side of the body. As we saw in the opening vignette, Miss S. displayed a symptom called unilateral neglect. A CT scan of her brain (shown in Figure 4.21) reveals that her stroke damaged part of the association cortex of the right parietal lobe.

Most neuropsychologists believe that the left parietal lobe plays an important role in our ability to keep track of the location of the moving parts of our own body, whereas the right parietal lobe helps us keep track of the space around us. People with right parietal lobe damage usually have difficulty with spatial tasks such as reading maps. People with left parietal lobe damage usually have difficulty identifying parts of their own bodies by name. For example, when asked to point to their elbows, they may actually point to their shoulders.

People with damage to the left parietal lobe often have difficulty performing arithmetic calculations. This deficit is probably related to other spatial functions of the parietal lobe. For example, try to multiply 55 by 12 without using pencil and paper. Close your eyes and work on the problem for a while. Then try to analyze how you did it. Most people report that they try to imagine the numbers arranged one above the other as they would be if they were using paper and pencil. In other words, they "write" the problem out mentally. Apparently, damage to the parietal lobes makes it impossible for people to keep the imaginary numbers in place and remember what they are.

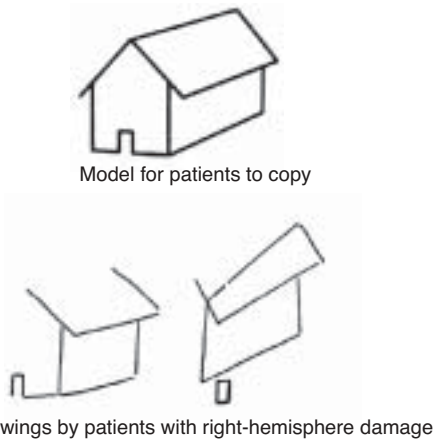


FIGURE 4•34 Attempts to copy a drawing of a house by patients with damage to the right parietal lobes.

(Adapted from Gainotti, G., and Tiacci, C. (1970). *Neuropsychologia, 8,* 1970, 289–303, with permission from Elsevier.)

Planning and Moving

As we have seen, a considerable amount of the brain is devoted to gathering and storing sensory information. Similarly, much of the brain is involved in the control of movement.

The Frontal Lobes The frontal lobes occupy the largest portion of the cerebral cortex. Although the principal function of the frontal lobes is motor activity, they also are involved in planning strategies for action, evaluating them, and changing them if necessary. They also contain a region involved in the control of speech. (Refer to Figure 4.30.)

Damage to the primary motor cortex produces a very specific effect: paralysis of the side of the body opposite to the brain damage. If a portion of the region is damaged, then only the corresponding parts of the body will be paralyzed. However, damage to the prefrontal cortex (refer to Figure 4.31) produces more complex behavioral deficits.

People with damage to the frontal lobes show *perseveration*—they have difficulty adopting new strategies. One of the reasons for this tendency appears to be that these people have difficulty in evaluating the success of what they are doing. If given a task to solve, they may solve it readily; but if the problem is changed, they will fail to abandon the strategy and learn a new one. They have little insight into their own problems and are uncritical of their performance on various tasks.

In terms of daily living, the most important consequences of damage to the frontal lobes are probably lack of foresight and difficulty making plans. A person with frontal lobe damage might perform fairly well on a test of intelligence but be unable to hold a job. Presumably, planning is related to the general motor functions of the frontal lobes. Just as we can use the posterior regions of the brain to imagine something we have perceived, so we can use the frontal region to imagine something we might do. Perhaps we test various possible actions by imagining ourselves doing them and guessing what the consequences of these actions might be. When people's

frontal lobes are damaged, they often do or say things that have unfavorable consequences because they have lost the ability to plan their actions.

As discussed in Chapter 1, Paul Broca discovered that damage to a particular region of the left frontal lobe disrupts speech. This region, which we now call Broca's area, lies at the base of the frontal lobe, just in front of the "face" region of the primary motor cortex. Thus, Broca's area controls the muscles used for talking. Circuits of neurons located in Broca's area appear to contain memories of the sequences of muscle movements that are needed to pronounce words. We'll discuss more on the effects of lesions in Broca's area in Chapter 10.

The Cerebellum The cerebellum ("little cerebrum") plays an important role in the control of movement. (Refer to Figure 4.30.) The cerebellum receives sensory information, especially about the position of body parts, so it knows what the parts of the body are doing. It also receives information from the cortex of the frontal lobes, so it knows what movements the frontal lobes intend to accomplish. The cerebellum is basically a computer that compares the location of body parts with the intended movements and assists the frontal lobes in executing these movements—especially rapid, skilled ones. Without the cerebellum, the frontal lobes would produce jerky, uncoordinated, inaccurate movements—which is exactly what happens when a person's cerebellum is damaged. Besides helping the frontal lobes accomplish their tasks, the cerebellum monitors information regarding posture and balance; it keeps us from falling down when we stand or walk, and it produces eye movements that compensate for changes in the position of the head.

Recently researchers have discovered that the cerebellum may also play a role in people's cognitive abilities. For a long time neurologists have known that cerebellar damage can interfere with people's ability to speak, but the deficit seemed to involve control of the speech muscles rather than the cognitive abilities involved in language. In the 1990s, however, researchers making PET scans of the brains of people working on various types of cognitive tasks discovered that parts of their cerebellums became active—even when the people were not moving. Many neuroscientists now believe that as we learn more about the cerebellum we will discover that its functions are not limited to motor tasks. By the way, the cerebellum contains about as many neurons as the cerebrum does.

The Basal Ganglia The **basal ganglia** are a collection of groups of neurons located in the depths of the cerebral hemispheres, adjacent to the thalamus. (Refer to Figure 4.32.) The basal ganglia are involved in the control of movements—particularly slow movements, and those that involve the large muscles of the body. For example, Parkinson's disease is caused by degeneration of dopamine-secreting neurons in the midbrain whose axons travel to parts of the basal ganglia. The release of dopamine in the basal ganglia helps facilitate movements. The symptoms of Parkinson's disease are weakness, tremors, rigidity of the limbs, poor balance, and difficulty in initiating movements.

Episodic and Spatial Memory: Role of the Hippocampus

The **limbic system,** a set of structures located in the cerebral hemispheres, plays an important role in learning and memory, and in the expression of emotion. The limbic system consists of several regions of the **limbic cortex**—the cerebral cortex located around the edge of the cerebral hemispheres where they join with the brain stem. (*Limbus* means "border"; hence the term *limbic* system.) Besides the limbic cortex, the most important components of the limbic system are the *hippocampus* and the *amygdala*. The hippocampus and the amygdala get their names from their shapes; *hippocampus* means "sea horse" and *amygdala* means "almond."

Figure 4•35 shows a view of the right hemisphere of the brain, rotated slightly and seen from the left. We can see the limbic cortex, located on the inner surface of the right cerebral hemisphere. The left hippocampus and amygdala, located in the middle of the temporal lobe, are shown projecting out into the place where the missing left hemisphere would be. We can also see the right hippocampus and amygdala, "ghosted in." We also see a structure that does not belong to the limbic system—the corpus callosum. As I mentioned earlier, the corpus callosum consists of a band of nerve fibers that enables the left and right cerebral hemispheres to communicate with each other.

We already encountered the **hippocampus** earlier in this chapter, when I discussed evidence that when London taxi drivers successfully learn to navigate around the city, part of

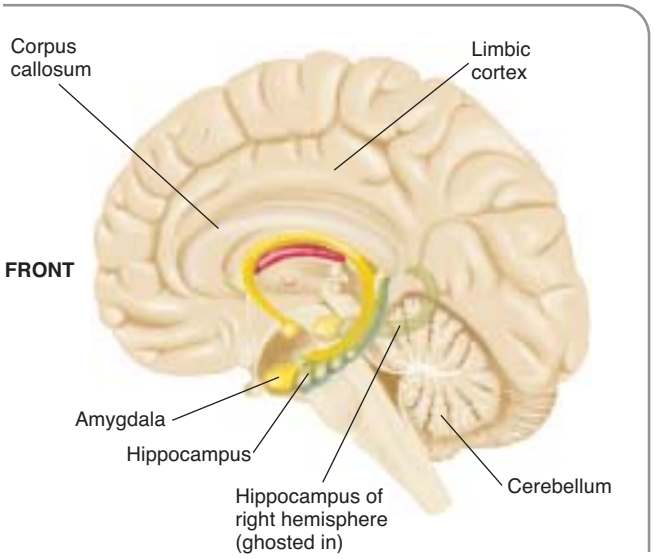


FIGURE 4•35 The principal structures of the limbic system.

their hippocampus increases in size. The hippocampus also is involved in *episodic memory*—that is, in our ability to learn and remember experience from our daily lives. As we will see in Chapter 8, when the hippocampus is destroyed, people can still remember events that occurred before their brains were damaged, but they lose the ability to learn anything new. For them, "yesterday" is always the time before their brain damage occurred. Everything after that slips away, just as the memory of a dream often slips away soon after a person awakens. In addition, although these people can find their way around places that were familiar to them before the damage occurred, they are unable to learn to navigate new neighborhoods—or even the interiors of buildings that are new to them.

Emotions: Role of the Amygdala

Damage to the **amygdala,** located in the middle of the temporal lobe, just in front of the hippocampus, affects emotional behavior—especially negative emotions, such as those caused by painful, threatening, or stressful events. In addition, the amygdala controls physiological reactions that help provide energy for short-term activities such as fighting or fleeing. If an animal's amygdala is destroyed, it no longer reacts to prevent events that normally produce stress and anxiety. We might think that an animal would be better off if it did not become "stressed out" by unpleasant or threatening situations, but research has shown that animals with damaged amygdalas do not survive in the wild. These animals fail to compete successfully for food and other resources, and they often act in ways that provoke attacks by other animals. Similarly, people with damage to the amygdala must live in institutions where they can be cared for so that they will not harm themselves or others. We'll look at the role of the amygdala in emotion and stress in Chapters 13 and 16.

Control of Internal Functions and Automatic Behavior

The brain stem and the hypothalamus are involved in homeostasis and control of species-typical behaviors. **Homeostasis** (from the root words *homoios*, "similar," and *stasis,* "standstill") refers to maintenance of a proper balance of physiological variables such as temperature, concentration of fluids, and the amount of nutrients stored within the body. **Species-typical behaviors** are the more or less automatic behaviors exhibited by most members of a species that are important to survival, such as eating, drinking, fighting, courting, mating, and caring for offspring.

The Brain Stem

The brain stem contains three structures: the *medulla,* the *pons,* and the *midbrain*. **Figure 4•36** shows a view of the left side of the brain. The cerebral hemispheres are semitransparent so that the details of the brain stem can be seen. We also see the *hypothalamus* and the *pituitary gland,* which are discussed below.

The brain stem contains circuits of neurons that control functions vital to the survival of the organism in particular and the species in general. For example, circuits of neurons in the **medulla,** the part of the brain stem adjacent to the spinal cord, control heart rate, blood pressure, rate of respiration, and—especially in simpler animals—crawling or swimming motions. Circuits of neurons in the **pons,** the part of the brain stem just above the medulla, are involved in control of sleep and wakefulness. Circuits of neurons in the **midbrain,** the part of the brain stem just above the pons, control movements used in fighting and sexual behavior and decrease sensitivity to pain while a person is engaged in these activities.

The Hypothalamus

Hypo- means "less than" or "beneath"; and, as its name suggests, the **hypothalamus** is located below the thalamus, at the base of the brain. (Refer to Figure 4.36.) The hypothalamus is a small region, consisting of less than 1 cubic centimeter of tissue (smaller than a grape). But relative importance far exceeds its size.

The hypothalamus, like the brain stem, participates in homeostasis and species-typical behaviors. It receives sensory information, including information from receptors inside the organs of the body; thus, it is informed about changes in the organism's physiological status. It also contains specialized sensors that monitor various characteristics of the blood that flows through the brain, such as temperature, nutrient content, and amount of dissolved salts. In turn, the hypothalamus controls the **pituitary gland**, an endocrine gland attached by a stalk to the base of the hypothalamus. (Refer to Figure 4.36.)

Hormones are chemicals produced by *endocrine glands* (from the Greek *endo-,* "within," and *krinein,* "to secrete"). (As

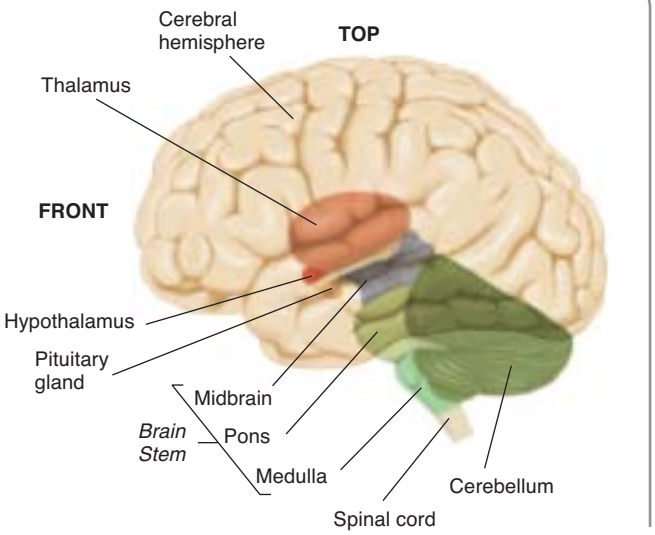


FIGURE 4•36 The divisions of the brain stem: the medulla, the pons, and the midbrain. The thalamus, hypothalamus, and pituitary gland are attached to the anterior end of the brain stem.

we will see in Chapter 13, hormones also are secreted by fat tissue and by special cells in the walls of the stomach and intestines.) **Endocrine glands** secrete hormones into the blood, which carries them to all parts of the body. **Hormones** are chemicals similar to neurotransmitters or neuromodulators, except that they act over much longer distances. Like neurotransmitters and neuromodulators, hormones produce their effects by stimulating receptors. These receptors are located on (or in) particular cells, which are known as **target cells**. When hormones bind with their receptors, they produce physiological reactions in these cells. Almost every cell of the body contains hormone receptors of one kind or another. This includes neurons, which means that hormones can affect behavior by altering the activity of particular groups of neurons in the brain. For example, sex hormones have important effects on behavior, which will be discussed in later chapters.

The pituitary gland has been called the "master gland," because the hormones it secretes act on target cells located in other endocrine glands; thus, the pituitary gland controls the activity of other endocrine glands. And because the hypothalamus controls the pituitary gland, the hypothalamus controls the endocrine system. The more important endocrine glands and the functions they regulate are shown in **Figure 4•37**.

The hypothalamus also controls much of the activity of the **autonomic nervous system (ANS),** a division of the peripheral nervous system that consists of nerves that control the functions of the glands and internal organs. The other division of the peripheral nervous system—the one that transmits information from sense organs to the central nervous system and from the central nervous system to the muscles, is called the **somatic nervous system.** Through the nerves of the autonomic ("self governing") nervous system, the hypothalamus controls activities such as sweating, shedding tears, salivating, secreting digestive juices, changing the size of blood vessels (which alters blood pressure), and the secretions of some endocrine glands. The autonomic nervous system has two branches. The **sympathetic branch** directs activities that involve the expenditure of energy. For example, activity of the sympathetic branch can increase the flow of blood to the muscles when we are about to fight someone or run away from a dangerous situation. In contrast, the **parasympathetic branch** controls quiet activities such as digestion of food. For example, activity of the parasympathetic branch stimulates the secretion of digestive enzymes and increases the flow of blood to the digestive system. (See **Table 4•3** and **Figure 4•38**.)

Psychophysiologists can monitor the activity of the autonomic nervous system and its relation to psychological phenomena such as emotion. For example, when people become angry, their heart rate and blood pressure rise. The lie detector, described in Chapter 13, works (or, more accurately, is

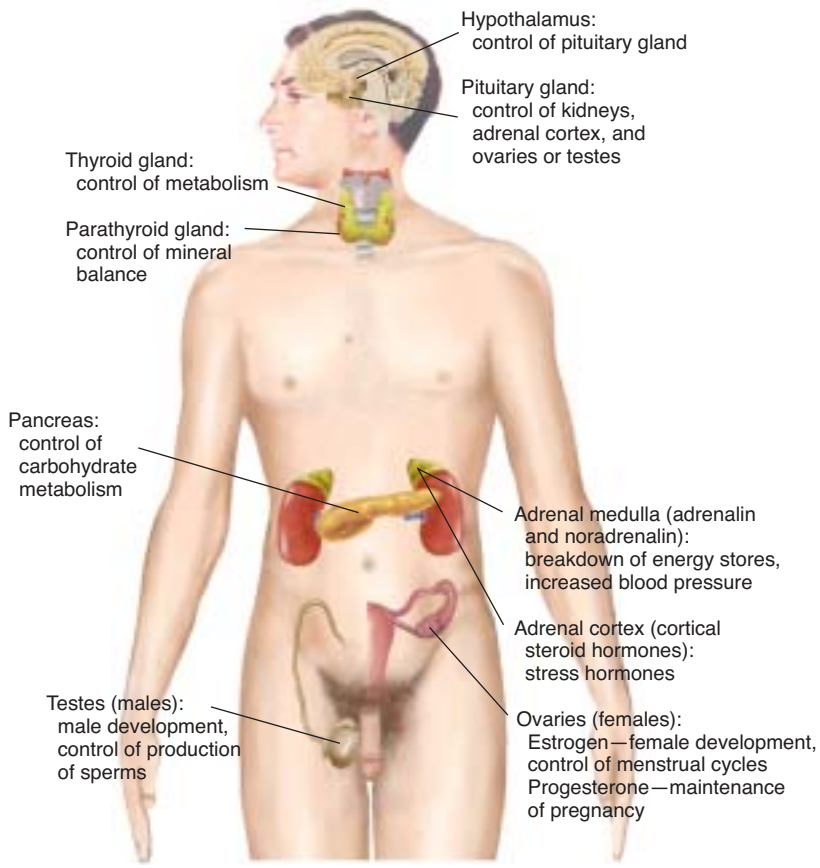


FIGURE 4•37 The location and primary functions of the principal endocrine glands.

TABLE 4•3 The Major Divisions of the Peripheral Nervous System

Division	Function
Somatic Nervous System	
Sensory nerves	Transmission of information from sense organs to central nervous system
Motor nerves	Control of skeletal muscles
Autonomic Nervous System	
Sympathetic branch	Support of activities that require the expenditure of energy (through increased blood flow to muscles, increased supply of nutrients of muscles)
Parasympathetic branch	Support of quiet activities that help restore energy supplies (through increased blood flow to digestive system, secretion of digestive enzymes)

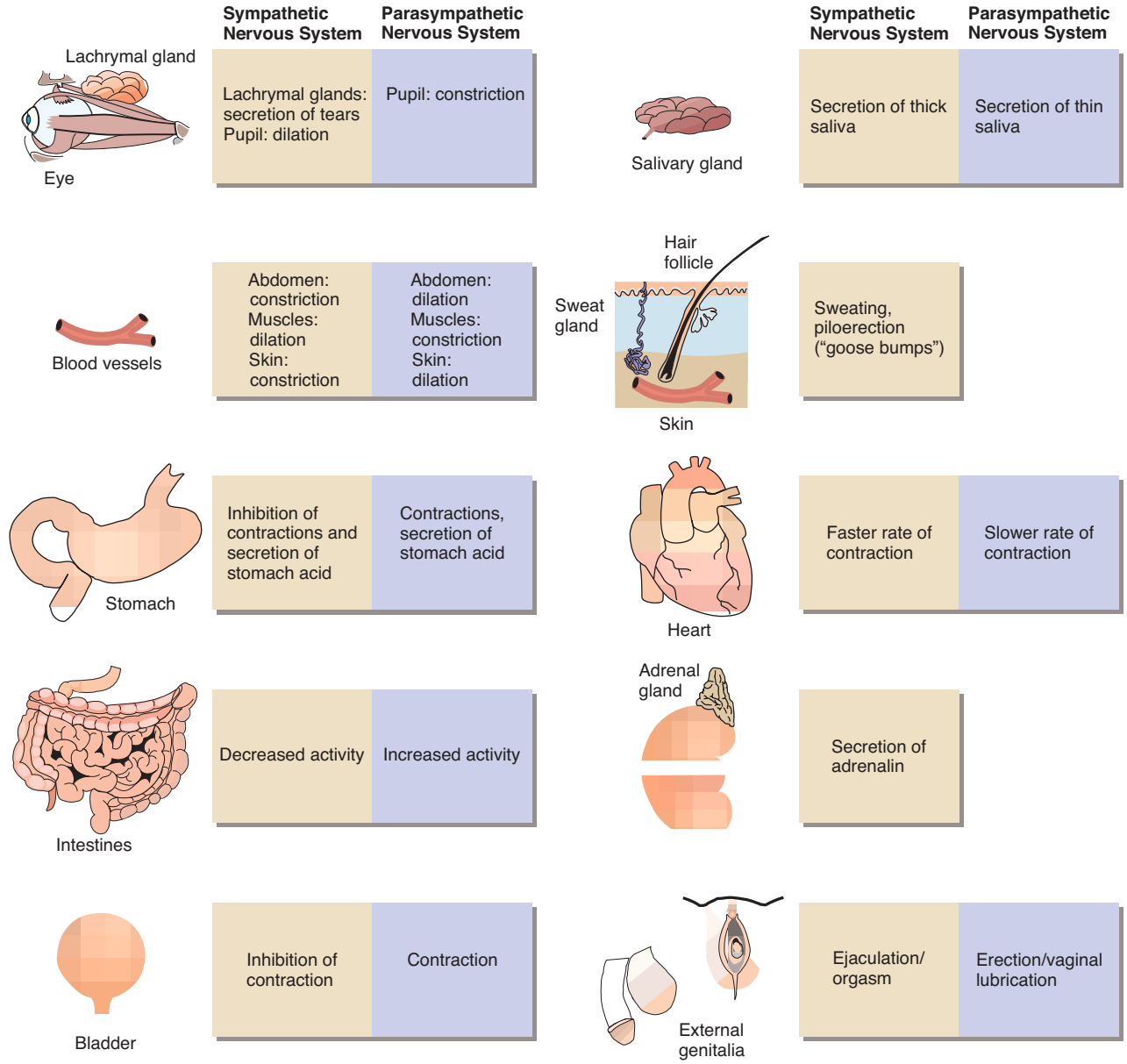


FIGURE 4•38 The organs controlled by the autonomic nervous system. The reciprocal actions of the sympathetic and parasympathetic branches are noted next to each organ.

said to work) by recording emotional responses controlled by the autonomic nervous system.

The homeostatic functions of the hypothalamus can involve either internal physiological changes or behavior. For example, the hypothalamus is involved in the control of body temperature. It can directly lower a person's body temperature by causing sweating to occur, or it can raise it by causing shivering to occur. If these measures are inadequate, the hypothalamus can send messages to the cerebral cortex that will cause the person to engage in a learned behavior, such as turning on an air conditioner or turning up the thermostat. Damage to the hypothalamus can cause impaired regulation of body temperature, changes in food intake, sterility, and stunting of growth.

Interim Summary

Control of Behavior and the Body's Physiological Functions

Anatomically, the cerebral cortex is divided into four lobes: frontal, parietal, occipital, and temporal. Functionally, the cerebral cortex is organized into the primary sensory cortex (with its visual, auditory, and somatosensory regions); the primary motor cortex; and the association cortex. The association cortex consists of sensory regions that are responsible for perceiving and learning and the motor regions that are responsible for planning and acting. Within the cerebral hemispheres, the thalamus relays sensory information to the cerebral cortex.

Some brain functions are lateralized; that is, the right and left hemispheres are involved with somewhat different functions. The left hemisphere is mostly concerned with analysis—with the extraction of information about details of perception, such as the series of sounds that constitute speech or the symbols that constitute writing. The right hemisphere is mostly concerned with synthesis—with putting together a perception of the general form and shape of things from smaller elements that are present at the same time. The two hemispheres share information through the corpus callosum, a large bundle of axons.

The three lobes behind the central fissure are generally concerned with perceiving, learning, and remembering: visual information in the occipital and lower temporal lobes, auditory information in the upper temporal lobe, and somatosensory information in the parietal lobe. The other functions of these lobes are related to these perceptual processes; for example, the parietal lobes are concerned with perception of space as well as knowledge about the body. The frontal lobes are concerned with motor functions, including the planning of strategies for action. A region of the left frontal cortex (Broca's area) is specialized for control of speech. The cerebellum and basal ganglia assist the frontal lobes with the details of executing movements.

The limbic system includes the limbic cortex as well as the hippocampus and the amygdala, both located within the temporal lobe. The hippocampus is involved in learning and memory; people with damage to this structure can recall old memories but are unable to learn anything new. The amygdala is involved in emotions and emotional behaviors, such as defense and aggression, and it plays an important role in physiological reactions that have beneficial effects in the short run.

The brain stem, which consists of the medulla, the pons, and the midbrain, contains neural circuits that control vital physiological functions and produce species-typical automatic movements such as those used in locomotion, fighting, and sexual behavior. The hypothalamus receives sensory information from sense receptors elsewhere in the body and also contains its own specialized receptors, such as those used to monitor body temperature. It controls the pituitary gland, which in turn controls most of the endocrine glands of the body; it also controls the internal organs through the autonomic nervous system. Hormones, secreted by endocrine glands, are chemicals that act on hormone receptors in target cells and produce physiological reactions in these cells. The hypothalamus can control homeostatic processes directly and automatically through its control of the pituitary gland and the autonomic nervous system, or it can cause neural circuits in the cerebral cortex to execute more complex, learned behavior.

QUESTIONS TO CONSIDER

1. If you were to have a stroke (and let's hope you don't), in which region of the cerebral cortex and in which hemisphere would you prefer the brain damage to be located? Why?
2. Damage to the corpus callosum produces different behavioral deficits depending on whether the anterior or the posterior corpus callosum is affected. Why do you think this is so?
3. Explain why a brain lesion that impairs a person's ability to speak often also affects movements of the right side of the body.
4. The cerebellum is one of the largest parts of the brain and contains billions of neurons. What does this fact suggest about the complexity of the task of coordinating movements of the body?
5. Suppose that you wanted to build a lie detector. You would monitor reactions that might indicate emotional responses produced by the act of lying. What behavioral and physiological functions would you want to record?
6. Tranquilizers reduce negative emotional reactions. In what part (or parts) of the brain do you think these drugs might act? Why?

Suggestions for Further Reading

Grilly, D. M. (2002). *Drugs and human behavior* (4th ed.). Boston: Allyn and Bacon.

Meyer, J. S., & Quenzer, L. F. (2005) *Psychopharmacology: Drugs, the brain, and behavior.* Sunderland, MA: Sinauer Associates.

If you are interested in learning more about the effects of drugs that are often abused, you may want to read these books, both of which contain much helpful information about the effects of popular drugs and their use and abuse in society.

Carlson, N. R. (2005). *Foundations of physiological psychology* (6th ed.). Boston: Allyn and Bacon.

My introductory textbook of physiological psychology discusses the topics presented in this chapter in more detail.

Key Terms

acetylcholine (ACh) (p. 97)

action potential (p. 88)

all-or-none law (p. 90)

amygdala (p. 116)

anandamide (p. 100)

antianxiety drug (p. 97)

autonomic nervous system (ANS) (p. 117)

axon (p. 88)

barbiturate (p. 97)

basal ganglia (p. 115)

benzodiazepine (p. 97)

black widow spider venom (p. 97)

blood–brain barrier (p. 86)

botulinum toxin (p. 97)

brain lesion (p. 103)

brain stem (p. 85)

central fissure (p. 110)

central nervous system (p. 85)

cerebellum (p. 85)

cerebral cortex (p. 87)

cerebral hemispheres (p. 85)

cerebral ventricle (p. 86)

cerebrospinal fluid (CSF) (p. 86)

contralateral (p. 110)

corpus callosum (p. 113)

cranial nerve (p. 85)

CT scanner (p. 104)

curare (p. 98)

dendrite (p. 88)

dendritic spine (p. 88)

dopamine (DA) (p. 98)

electroencephalogram (EEG) (p. 105)

endocrine gland (p. 117)

endogenous cannabinoid (p. 99)

endogenous opioid (p. 99)

frontal lobe (p. 110)

functional MRI (fMRI) (p. 106)

GABA (p. 96)

glia (p. 87)

glutamate (p. 96)

gray matter (p. 87)

hippocampus (p. 115)

homeostasis (p. 116)

hormone (p. 117)

hypothalamus (p.116)

interneuron (p. 92)

ion (p. 88)

ion channel (p. 89)

ion transporter (p. 89)

ipsilateral (p. 110)

limbic cortex (p. 115)

limbic system (p. 115)

LSD (p. 99)

magnetic resonance imaging (MRI) (p. 104)

magnetoencephalography (MEG) (p. 105)

medulla (p. 116)

meninges (p. 85)

microelectrode (p. 105)

midbrain (p. 116)

monoamine (p. 98)

motor association cortex (p. 111)

motor neuron (p. 90)

myelin sheath (p. 88)

naloxone (p. 99)

neostigmine (p. 98)

nerve (p. 85)

neuron (p. 87)

neuromodulator (p. 99)

neurotransmitter (p. 88)

neurotransmitter receptor (p. 91)

nicotine (p. 98)

norepinephrine (NE) (p. 99)

occipital lobe (p. 110)

parasympathetic branch (p. 117)

parietal lobe (p. 110)

Parkinson's disease (p. 98)

peptide (p. 99)

peripheral nervous system (p. 85)

pituitary gland (p. 116)

pons (p. 116)

positron emission tomography (PET) (p. 105)

postsynaptic neuron (p. 90)

prefrontal cortex (p. 111)

presynaptic neuron (p. 90)

primary auditory cortex (p. 110)

primary motor cortex (p. 111)

primary somatosensory cortex (p. 110)

primary visual cortex (p. 110)

resting potential (p. 88)

reuptake (p. 91)

sensory association cortex (p. 111)

sensory neuron (p. 90)

serotonin (p. 99)

soma (p. 88)

somatic nervous system (p. 117)

species-typical behavior (p. 116)

spinal cord (p. 85)

spinal nerve (p. 85)

stem cells (p. 109)

stereotaxic apparatus (p. 103)

sympathetic branch (p. 117)

synapse (p. 90)

synaptic cleft (p. 91)

target cell (p. 117)

targeted mutation (p. 107)

temporal lobe (p. 110)

terminal button (p. 88)

thalamus (p. 111)

transcranial magnetic stimulation (TMS) (p. 106)

tolerance (p. 100)

vertebra (p. 85)

visual agnosia (p. 113)

white matter (p. 87)

withdrawal symptom (p. 100)