

An ordinary-looking, 30-year-old mother of three is walking down the street in a seedy neighborhood around 10:00 p.m. when she encounters a drugged-out man who presses a knife to her throat and threatens to kill her. Remarkably, the woman shows no signs of fear. Her heart rate doesn't quicken, her breathing doesn't change, she doesn't get nervous. Her completely calm, unruffled response to her attacker so unnerves *him* that he lets her go! Meet the woman who knows no fear.

The woman in this story, known by her initials, S.M., has attracted interest from scientists who are intrigued by her apparent inability to experience fear. In a recent study designed to investigate S.M.'s fearlessness, researchers exposed her to a variety of situations that would trigger fear in most people (Feinstein et al., 2011). For ethical reasons they chose fear-inducing stimuli that posed relatively little risk of actual harm. For example, they took her to an exotic pet store, where they exposed her to snakes and spiders. What was her reaction? Instead of being scared, S.M. was fascinated and repeatedly asked to touch large snakes and a tarantula that were not safe to handle. Next, the scientists took S.M. to a famous haunted house. While other patrons screamed in fright, S.M. giggled and poked one of the monsters in the head—again scaring *him*. Taking yet another tack, the researchers showed S.M. clips from well-known scary movies, such as *The Ring*. She found the clips entertaining, but she experienced no fear. Finally, the researchers used a variety of methods to determine whether S.M. experiences other emotions. Their results revealed that she exhibits all the other basic emotions—anger, sadness, disgust,

happiness, surprise—much like anyone else. Other tests demonstrated that S.M. scores normally on measures of intelligence, language, and memory.

Why is this woman absolutely fearless, yet she experiences other emotions? What could cause such a bizarre, exquisitely specific emotional deficit? It turns out that in childhood S.M. suffered an extremely rare disease that destroyed a small structure called the *amygdala*, which is located in both the right and left halves of her brain. Many studies, mostly with animals, have suggested that the amygdala is a crucial control center for the experience of fear. S.M.'s case provides compelling, new *human* evidence that the amygdala has a unique role in the regulation of fear. By the way, if you are thinking that being fearless sounds appealing, think again. The research on S.M. has shown that she is fundamentally lacking when it comes to detecting when she's in danger, which is why she has a long history of getting herself into perilous situations.

S.M.'s unusual case provides a dramatic demonstration that behavioral functioning is ultimately controlled by the brain. The fact that S.M. feels all the other emotions except fear shows just how precise the biological bases of behavior can be. The human brain is so complex, no computer has ever come remotely close to duplicating it. Your nervous system contains as many cells busily integrating and relaying information as there are stars in our galaxy. Whether you are scratching your nose or composing an essay, the activity of those cells underlies what you do. It is little wonder, then, that many psychologists have dedicated themselves to exploring the biological bases of behavior.

3.1 COMMUNICATION IN THE NERVOUS SYSTEM

Imagine that you are watching a scary movie. As the tension mounts, your palms sweat and your heart beats faster. You begin shoveling popcorn into your mouth, carelessly spilling some in your lap. If someone were to ask you what you are doing at this moment, you would probably say, "Nothing—just watching the movie." Yet some highly complex processes are occurring without your thinking about them. A stimulus (the light from the screen) is striking your eye. Almost instantaneously, your brain is interpreting the light stimulus, and signals are flashing to other parts of your body, leading to a flurry of activity. Your sweat glands are releasing perspiration; your heartbeat is quickening; and muscular movements are enabling your hand to find the popcorn, and more or less successfully, lift it to your mouth.

Even in this simple example, you can see that behavior depends on rapid information processing. Information travels immediately from your eye to your brain, from your brain to the muscles of your arm and hand, and from your palms back to your brain. In essence, your nervous system is a complex communication network in which signals

3.1

Key Learning Goals

- Identify the various parts of the neuron and the main functions of glial cells.
- Describe the neural impulse, and explain how neurons communicate at chemical synapses.
- Discuss some of the functions of acetylcholine, the monoamine neurotransmitters, GABA, and endorphins.

are constantly being received, integrated, and transmitted. The nervous system handles information, just as the circulatory system handles blood. In this section, we take a close look at communication in the nervous system.

Nervous Tissue: The Basic Hardware

Your nervous system is living tissue composed of cells. The cells in the nervous system fall into two major categories: *glia* and *neurons*. Let's look at neurons first.

Neurons

Neurons are individual cells in the nervous system that receive, integrate, and transmit information. They are the basic links that permit communication within the nervous system. The vast majority of them communicate only with other neurons. However, a small minority receive signals from outside the nervous system (from sensory organs) or carry messages from the nervous system to the muscles that move the body.

A highly simplified drawing of two "typical" neurons is shown in **Figure 3.1**. The **soma, or cell body, contains the cell nucleus and much of the chemical machinery common to most cells** (*soma* is Greek for "body"). The rest of the neuron is devoted exclusively to handling information. The neurons in **Figure 3.1** have a number of branched, feeler-like structures called *dendritic trees* (*dendrite* is a Greek word for "tree"). Each individual branch is a *dendrite*. **Dendrites are the parts of a neuron that are specialized to receive information.** Most neurons receive information from many other cells—sometimes thousands of others—and so have extensive dendritic trees.

From the many dendrites, information flows into the cell body, then travels away from the soma along the *axon* (from the Greek for "axle"). **The axon is a long, thin fiber that transmits signals away from the soma to other neurons or to muscles or glands.** Axons may be quite long (sometimes several feet), and they may branch off to communicate with a number of other cells.

In humans, many axons are wrapped in cells with a high concentration of a white, fatty substance called *myelin*. **The myelin sheath is insulating material that encases some axons.** Myelin wrapping functions to speed up the transmission of signals that move along axons (Zorumski, Isenberg, & Mennerick, 2009). By preventing axons from

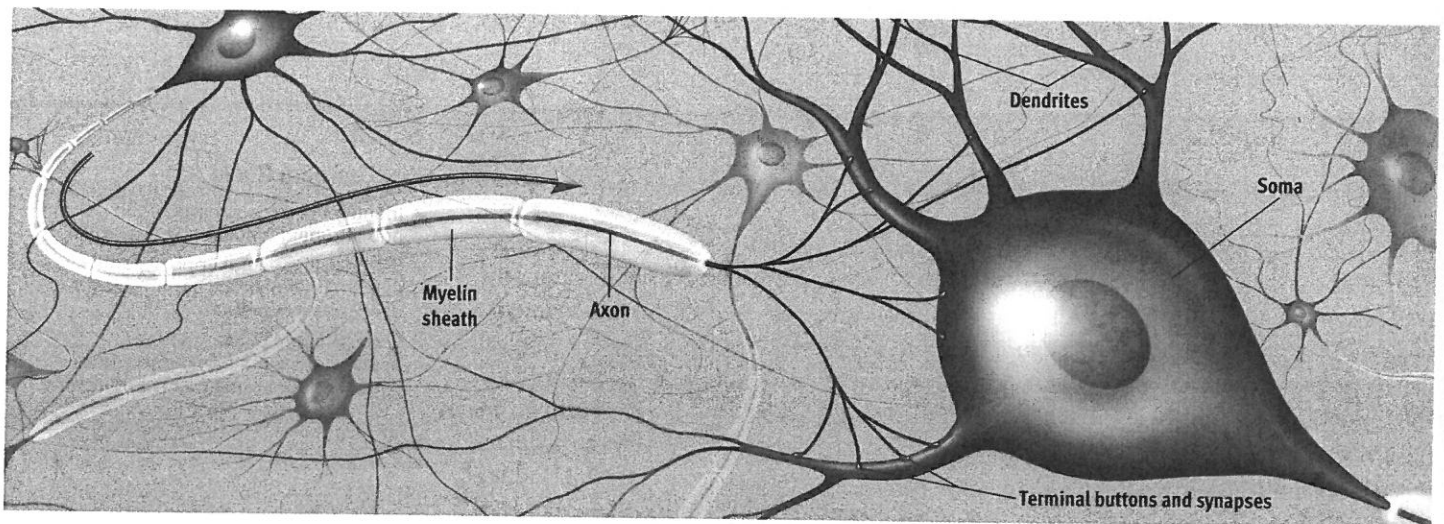


FIGURE 3.1

Structure of the neuron. Neurons are the communication links of the nervous system. This diagram highlights the key parts of a neuron, including specialized receptor areas (dendrites); the cell body (soma); the axon fiber along which impulses are transmitted; and the terminal buttons, which release chemical messengers that carry signals to other neurons. Neurons vary considerably in size and shape and are usually densely interconnected.

sprouting in new directions, myelin also stabilizes axon structure and patterns of connectivity in neural networks (Fields, 2014). If certain axons' myelin sheaths deteriorate, signals may not be transmitted effectively. The loss of muscle control seen with the disease *multiple sclerosis* is due to a degeneration of myelin sheaths (Joffe, 2009). That said, not all axons are myelinated, and among those that are, the myelin sheaths can be distributed in a variety of ways. In some axons, segments that have myelin sheaths may be interspersed with long segments that are unmyelinated (Tomassy et al., 2014).

The axon ends in a cluster of **terminal buttons**, which are small knobs that secrete chemicals called **neurotransmitters**. These chemicals serve as messengers that may activate neighboring neurons. The points at which neurons interconnect are called **synapses**. A **synapse** is a junction where information is transmitted from one neuron to another (*synapse* is from the Greek for "junction"). To summarize, information is received at the dendrites, is passed through the soma and along the axon, and is transmitted to the dendrites of other cells at meeting points called synapses.

Glia

Glia are cells found throughout the nervous system that provide various types of support for neurons. Glia (literally "glue") tend to be much smaller than neurons. It was once thought that they outnumbered neurons by as much as 10 to 1, but recent research suggests that the human brain consists of roughly equal numbers of neurons and glial cells (Azevedo et al., 2009). Glial cells serve many functions. For example, they supply nourishment to neurons, help remove neurons' waste products, and provide insulation around many axons. The myelin sheaths that encase some axons are derived from special types of glial cells. Glia also play a complicated role in the development of the nervous system in the human embryo.

These functions, which have been known for many years, made glial cells the unsung heroes of the nervous system. Until recently, it was thought that the "glamorous" work in the nervous system—the transmission and integration of informational signals—was the exclusive province of the neurons. New research, however, suggests that glia may also send and receive chemical signals (Deitmer & Rose, 2010; Fields, 2011). Some types of glia can detect neural impulses and send signals to other glial cells, some of which can feed signals back to neurons. Surprised by this discovery, neuroscientists are now trying to figure out how this signaling system interfaces with the neural communication system. One view is that glia *modulate* the signaling of neurons, dampening or amplifying synaptic activity (Halassa & Haydon, 2010). Another view is that glial cells' primary role is to shield synapses from the "chatter" of surrounding neuronal activity, thus enhancing the signal-to-noise ratio in the nervous system (Nedergaard & Verkhratsky, 2012). These alternate viewpoints are not necessarily incompatible, and much remains to be learned.

Recent research suggesting that glial cells may play a role in a variety of major disorders underscores their importance. For example, some studies indicate that dysfunction in glial cells may contribute to the cognitive impairment seen in schizophrenic disorders (Mitterauer, 2011) and to some forms of depressive disorders (Jellinger, 2013). Another line of research has raised the possibility that a gradual deterioration of glial tissue might contribute to Alzheimer's disease (Olabarria et al., 2010). Glial cells have also been implicated as a key factor in the experience of chronic pain (Ji, Berta, & Nedergaard, 2013).

Although glia may contribute to information processing in the nervous system, the bulk of this crucial work is handled by the neurons. Thus, we need to examine the process of neural activity in more detail.

The Neural Impulse: Using Energy to Send Information

What happens when a neuron is stimulated? What is the nature of the signal—the *neural impulse*—that moves through the neuron? These were the questions that Alan Hodgkin and Andrew Huxley set out to answer in their groundbreaking experiments with axons removed

▶ REALITY CHECK

Misconception

Neurons are responsible for all the information processing in the nervous system.

Reality

Until recently, it was thought that the transmission and integration of informational signals was the exclusive role of the neurons. However, newer research has demonstrated that glial cells also play an important role in information processing.

from squid. Why did they choose to work with squid axons? Because squid have a pair of “giant” axons that are about a hundred times larger than those in humans (which still makes them only about as thick as a human hair). This large size permitted Hodgkin and Huxley to insert fine wires called *microelectrodes* into the axons. By using the microelectrodes to record the electrical activity in individual neurons, Hodgkin and Huxley were able to unravel the mystery of the neural impulse.

The Neuron at Rest: A Tiny Battery

Hodgkin and Huxley (1952) learned that the neural impulse is a complex electrochemical reaction. Both inside and outside the neuron are fluids containing electrically charged atoms and molecules called *ions*. Positively charged sodium and potassium ions and negatively charged chloride ions flow back and forth across the cell membrane, but they do not cross at the same rate. The difference in flow rates leads to a slightly higher concentration of negatively charged ions inside the cell. The resulting voltage means that the neuron at rest is a tiny battery, a store of potential energy. **The resting potential of a neuron is its stable, negative charge when the cell is inactive.** As shown in Figure 3.2(a), this charge is about -70 millivolts, roughly one-twentieth of the voltage of a flashlight battery.

The Action Potential

As long as the voltage of a neuron remains constant, the cell is quiet, and no messages are being sent. When the neuron is stimulated, channels in its cell membrane open, briefly allowing

positively charged sodium ions to rush in. For an instant, the neuron's charge is less negative, but then it gradually becomes positive, creating an action potential (McCormick, 2008). **An action potential is a very brief shift in a neuron's electrical charge that travels along an axon.** The firing of an action potential is reflected in the voltage spike shown in Figure 3.2(b). Like a spark traveling along a trail of gunpowder, the voltage change races down the axon.

After the firing of an action potential, the channels in the cell membrane that opened to let in sodium close up. Some time is needed before they are ready to open again, and until that time, the neuron cannot fire. **The absolute refractory period is the minimum length of time after an action potential, during which another action potential cannot begin.** This “down time” isn't very long, only 1 or 2 milliseconds.

The All-or-None Law

The neural impulse is an all-or-none proposition, like firing a gun. You can't half-fire a gun. The same is true of the neuron's firing of action potentials. Either the neuron fires or it doesn't, and its action potentials are all the same size (Kandel, Barres, & Hudspeth, 2013). That is, weaker stimuli do not produce smaller action potentials and stronger stimuli do not evoke larger action potentials.

Even though the action potential is an all-or-nothing event, neurons *can* convey information about the strength of a stimulus. They do so by varying the *rate* at which they fire action potentials. In general, a stronger stimulus will cause a

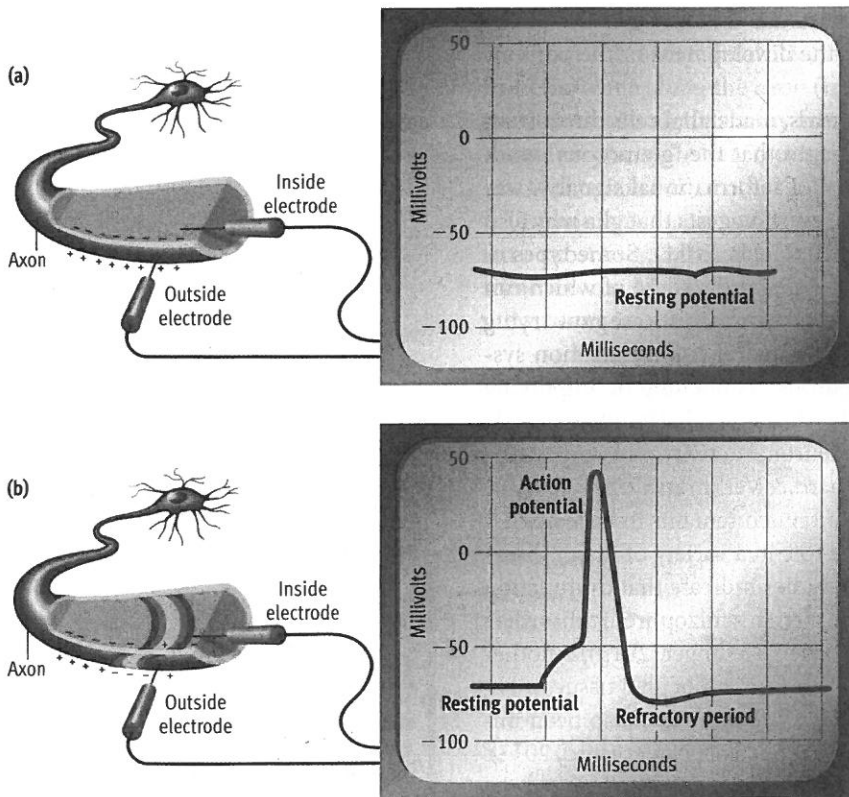


FIGURE 3.2

The neural impulse. The electrochemical properties of the neuron allow it to transmit signals. The electric charge of a neuron can be measured with a pair of electrodes connected to an oscilloscope, as Hodgkin and Huxley showed with a squid axon. Because of its exceptionally thick axons, the squid has frequently been used by scientists studying the neural impulse. **(a)** At rest, the neuron's voltage hovers around -70 millivolts. **(b)** When the axon is stimulated, there is a brief jump in a neuron's voltage, resulting in a spike on the oscilloscope recording of the neuron's electrical activity. This change in voltage, called an action potential, travels along the axon like a spark traveling along a trail of gunpowder.

cell to fire a more rapid volley of neural impulses than a weaker stimulus will. For example, a dim light might trigger 5 action potentials per second in a visual cell, whereas brighter lights might trigger 100 or 200 impulses per second (Burkhardt, 2010).

The Synapse: Where Neurons Meet

In the nervous system, the neural impulse functions as a signal. For that signal to have any meaning for the system as a whole, it must be transmitted from the neuron to other cells. As noted earlier, this transmission takes place at special junctions called *synapses*, which depend on *chemical* messengers.

Sending Signals: Chemicals as Couriers

A “typical” synapse is shown in **Figure 3.3**. The first thing that you should notice is that the two neurons don’t actually touch. They are separated by the *synaptic cleft*, a microscopic gap between the terminal button of one neuron and the cell membrane

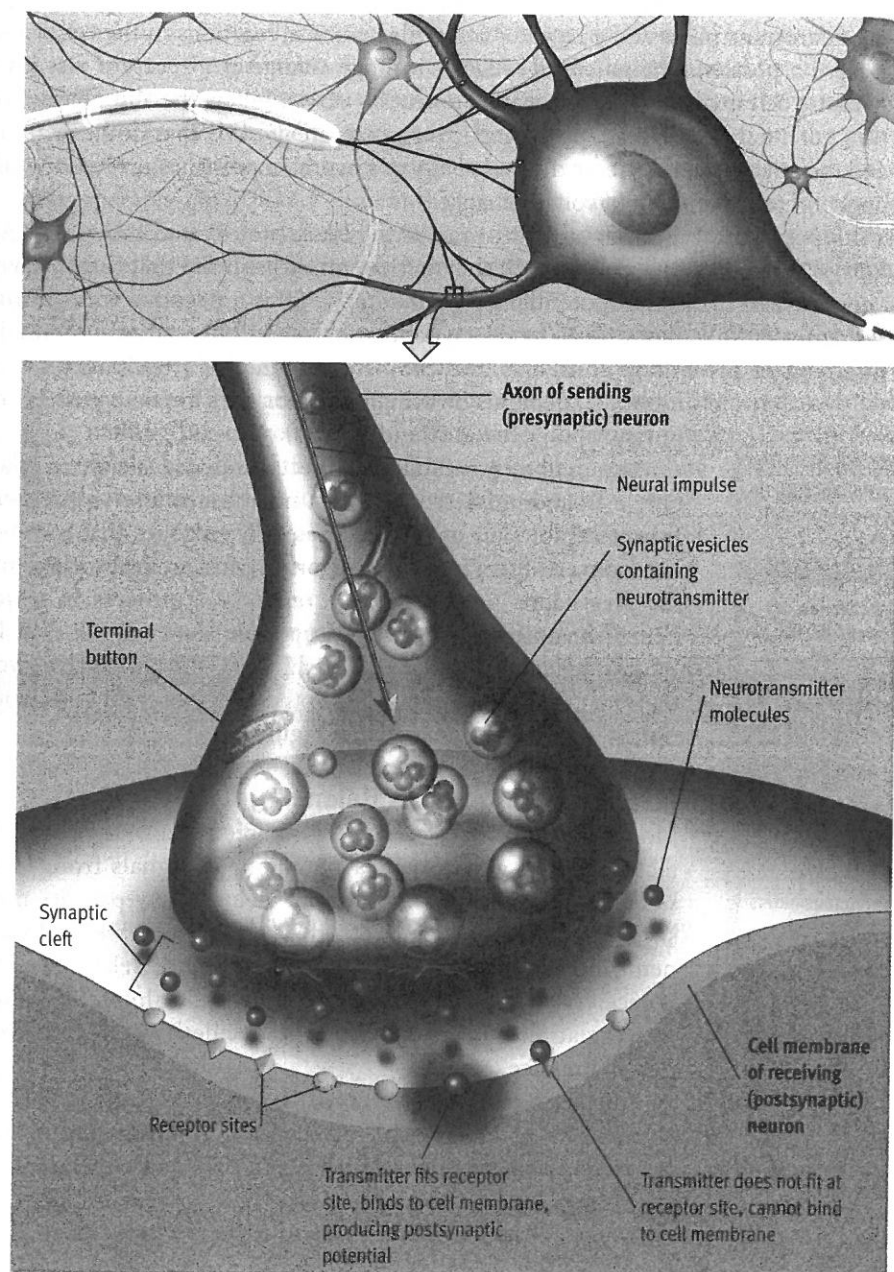


FIGURE 3.3

The synapse. When a neural impulse reaches an axon's terminal buttons, it triggers the release of chemical messengers called neurotransmitters. The neurotransmitter molecules diffuse across the synaptic cleft and bind to receptor sites on the postsynaptic neuron. A specific neurotransmitter can bind only to receptor sites that its molecular structure will fit into, much like a key must fit a lock.

of another neuron. Signals have to cross this gap to permit neurons to communicate. In this situation, the neuron that sends a signal across the gap is called the *presynaptic neuron*, and the neuron that receives the signal is called the *postsynaptic neuron*.

How do messages travel across the gaps between neurons? As mentioned earlier, the arrival of an action potential at an axon's terminal buttons triggers the release of **neurotransmitters—chemicals that transmit information from one neuron to another**. Within the buttons, most of these chemicals are stored in small sacs, called *synaptic vesicles*. The neurotransmitters are released when a vesicle fuses with the membrane of the presynaptic cell and its contents spill into the synaptic cleft (Schwarz, 2008). After their release, neurotransmitters diffuse across the synaptic cleft to the membrane of the receiving cell. There they may bind with special molecules in the postsynaptic cell membrane at various *receptor sites*. These sites are specifically “tuned” to recognize and respond to some neurotransmitters but not to others (Siegelbaum & Kandel, 2013).

Receiving Signals: Postsynaptic Potentials

When a neurotransmitter and a receptor molecule combine, reactions in the cell membrane cause a **postsynaptic potential (PSP)**, a **voltage change at a receptor site on a postsynaptic cell membrane**. Postsynaptic potentials do *not* follow the all-or-none law as action potentials do. Instead, postsynaptic potentials are *graded*. That is, they vary in size, and they increase or decrease the *probability* of a neural impulse in the receiving cell in proportion to the amount of voltage change.

Two types of messages can be sent from cell to cell: excitatory and inhibitory. An *excitatory PSP* is a positive voltage shift that *increases* the likelihood that the postsynaptic neuron will fire action potentials. An *inhibitory PSP* is a negative voltage shift that *decreases* the likelihood that the postsynaptic neuron will fire action potentials. The direction of the voltage shift, and thus the nature of the PSP (excitatory or inhibitory), depends on which receptor sites are activated in the postsynaptic neuron (Kandel, 2000).

The excitatory or inhibitory effects produced at a synapse last only a fraction of a second. Then neurotransmitters drift away from receptor sites or are inactivated by enzymes that metabolize (convert) them into inactive forms. Most are reabsorbed into the presynaptic neuron through **reuptake**, a process in which **neurotransmitters are sponged up from the synaptic cleft by the presynaptic membrane**. Reuptake allows synapses to recycle their materials. Reuptake and the other key processes in synaptic transmission are summarized in Figure 3.4.

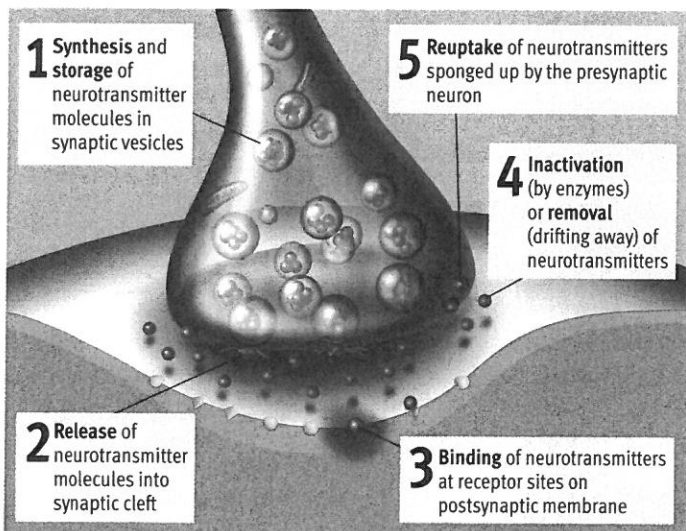


FIGURE 3.4

Overview of synaptic transmission. The main processes in synaptic transmission are summarized here. The five key processes involved in communication at synapses are (1) synthesis and storage, (2) release, (3) binding, (4) inactivation or removal, and (5) reuptake of neurotransmitters. As you'll see in this chapter and the remainder of the book, the effects of many phenomena—such as pain, drug use, and some diseases—can be explained in terms of how they alter one or more of these processes (usually at synapses releasing a specific neurotransmitter).

Integrating Signals: A Balancing Act

Most neurons are interlinked in complex, dense networks. In fact, a neuron may receive a symphony of signals from *thousands* of other neurons. That same neuron may pass its messages along to thousands of other neurons, as well. Thus, a neuron must do a great deal more than simply relay messages it receives. It must *integrate* excitatory and inhibitory signals arriving at many synapses before it “decides” whether to fire a neural impulse.

Most neurons are interlinked in complex chains, pathways, circuits, and networks. Our perceptions, thoughts, and actions depend on *patterns* of neural activity in elaborate, widely distributed neural networks (van den Heuvel & Sporns, 2013). These networks consist of interconnected neurons that frequently fire