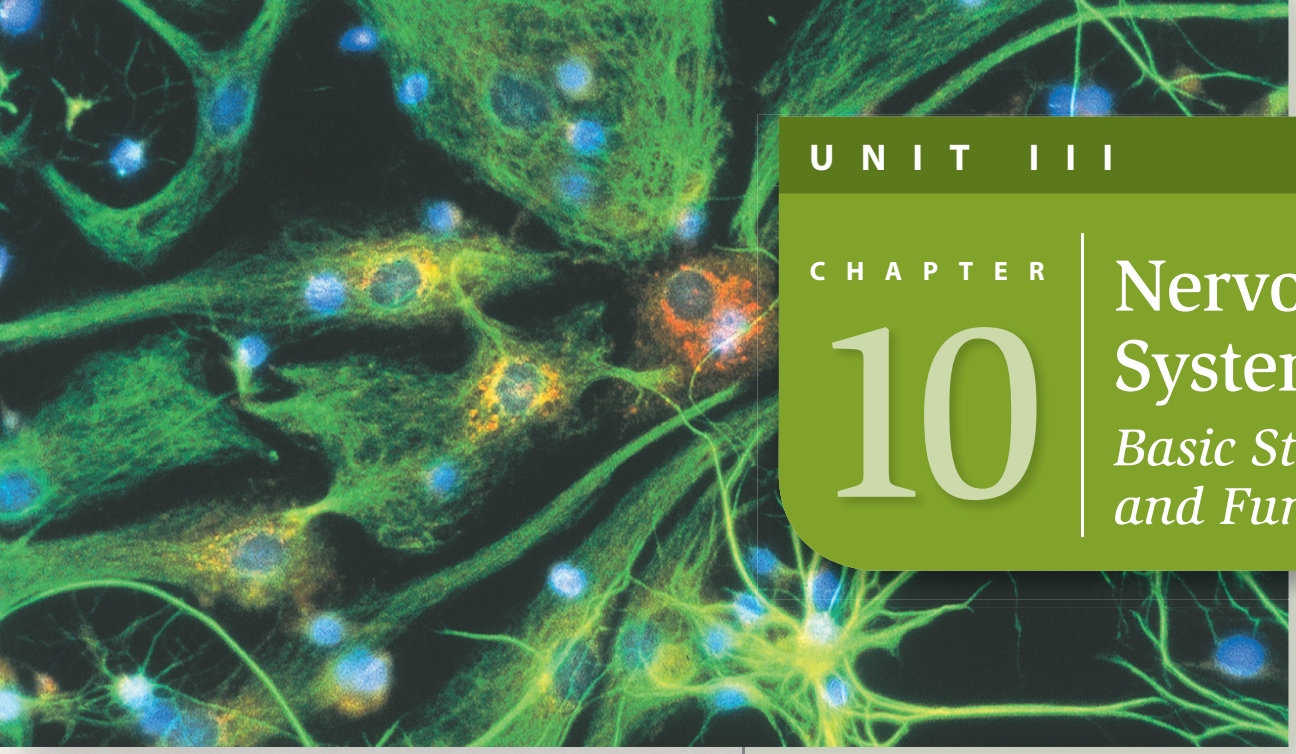


UNIT III

CHAPTER 10

Nervous System I

Basic Structure and Function

These progenitor cells (green) will give rise to astrocytes that supply neurons with nutrients. Cell nuclei are stained blue. Immunofluorescent light micrograph (1,150×).

UNDERSTANDING WORDS

astr-, starlike: *astr*ocyte—star-shaped neuroglial cell.

ax-, axle: *ax*on—cylindrical nerve process that carries impulses away from a neuron cell body.

bi-, two: *bi*polar neuron—neuron with two processes extending from the cell body.

dendr-, tree: *dendr*ite—branched nerve process that serves as the receptor surface of a neuron.

ependym-, tunic: *ependym*a—neuroglial cells that line spaces in the brain and spinal cord.

-lemm, rind or peel: neuri*lemm*a—sheath that surrounds the myelin of a nerve cell process.

moto-, moving: *motor* neuron—neuron that stimulates a muscle to contract or a gland to release a secretion.

multi-, many: *multi*polar neuron—neuron with many processes extending from the cell body.

oligo-, few: *oligo*dendrocyte—small neuroglial cell with few cellular processes.

peri-, all around: *peri*pheral nervous system—portion of the nervous system that consists of the nerves branching from the brain and spinal cord.

saltator-, a dancer: *saltator*y conduction—nerve impulse conduction in which the impulse seems to jump from node to node along the nerve fiber.

sens-, feeling: *sens*ory neuron—neuron that can be stimulated by a sensory receptor and conducts impulses into the brain or spinal cord.

syn-, together: *syn*apse—junction between two neurons.

uni-, one: *uni*polar—neuron with only one process extending from the cell body.

LEARN PRACTICE ASSESS

LEARNING OUTCOMES

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

10.1 Introduction
- **1** Describe the general functions of the nervous system. (p. 354)
- **2** Identify the two types of cells that comprise nervous tissue. (p. 354)
- **3** Identify the two major groups of nervous system organs. (p. 354)

10.2 General Functions of the Nervous System
- **4** List the functions of sensory receptors. (p. 355)
- **5** Describe how the nervous system responds to stimuli. (p. 355)

10.3 Description of Cells of the Nervous System
- **6** Describe the parts of a neuron. (p. 356)
- **7** Describe the relationships among myelin, the neurilemma, and nodes of Ranvier. (p. 358)
- **8** Distinguish between the sources of white matter and gray matter. (p. 358)

10.4 Classification of Cells of the Nervous System
- **9** Identify structural and functional differences among neurons. (p. 359)
- **10** Identify the types of neuroglia in the central nervous system and their functions. (p. 361)
- **11** Describe the role of Schwann cells in the peripheral nervous system. (p. 363)

10.5 The Synapse
- **12** Explain how information passes from a presynaptic neuron to a postsynaptic cell. (p. 365)

10.6 Cell Membrane Potential
- **13** Explain how a cell membrane becomes polarized. (p. 365)
- **14** Describe the events leading to and the conduction of a nerve impulse. (p. 368)
- **15** Compare nerve impulse conduction in myelinated and unmyelinated neurons. (p. 371)

10.7 Synaptic Transmission
- **16** Identify the changes in membrane potential associated with excitatory and inhibitory neurotransmitters. (p. 371)
- **17** Explain what prevents a postsynaptic cell from being continuously stimulated. (p. 374)

10.8 Impulse Processing
- **18** Describe the basic ways in which the nervous system processes information. (p. 374)

BRAIN BANKS

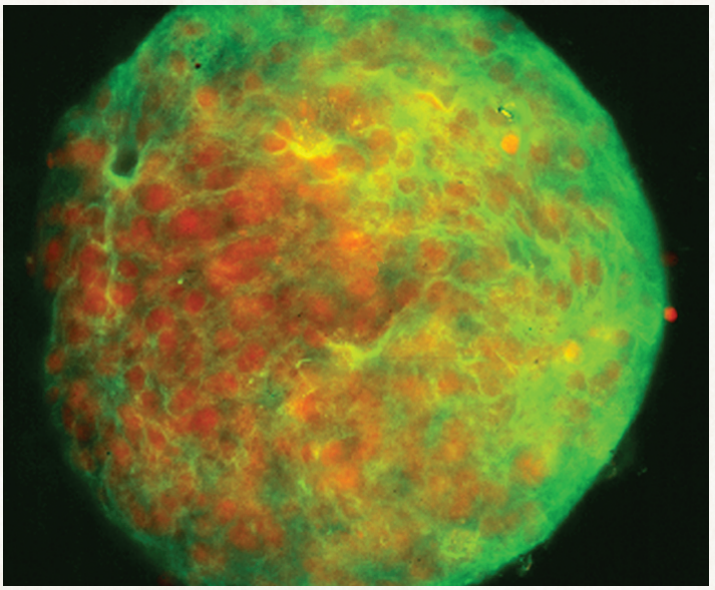
In a large room at the Croatian Institute for Brain Research, rows of shelves hold a variety of fluid-filled jars, a human brain suspended in each. Their sizes differ, reflecting their origins from embryos up to the elderly. Researchers can use the more than 1,000 brains and more than 130,000 histological slides at the bank to investigate brain-based diseases and injuries that affect many millions of people worldwide and also to better understand the functioning of the normal human brain.

In the United States, several brain banks offer tissue sections from thousands of people who willed their brains to science. Unlike donated hearts, lungs, or corneas, which directly help other people, donated brains go to research labs.

Many brain banks are specialized. The bank at Harvard University is devoted to neurodegenerative diseases, such as Alzheimer and Parkinson diseases, while the resource at the University of Maryland in Baltimore focuses on developmental disorders, including Down syndrome and autism. The brain bank at the University of Miami has brains from people who had schizophrenia, depression, amyotrophic lateral sclerosis, and several other disorders, as well as undiseased brains for comparison.

Brains must be removed from the skull within twelve hours of death. Then they are halved and cut into one-centimeter thick sections and frozen in plastic bags. The specimens are provided free to researchers.

Study of brain function and malfunction is also possible at the cellular level. The National Human Neural Stem Cell Resource provides neural stem cells, which function after death longer than neurons because their energetic and oxygen requirements are not as high as those of the more specialized cells. Hospitals collect brain material upon autopsy and send it to the facility, where a special protocol is used to obtain and preserve the cells from several brain areas. These techniques were perfected on the brains of pigs, cats, and sheep. Investigators use the human neural stem cells to study neurodegenerative disorders, stroke, traumatic brain injury, rare inborn errors of metabolism, as well as the development of the incredibly complex human brain from initial stem and progenitor cells. The material in brain and stem cell banks is also being used in drug discovery and in developing new treatments based on cell implants. The chapter opening image shows neural progenitor cells and the photo accompanying this essay shows neural stem cells. ■



Neurospheres cultured in the laboratory consist of neural stem cells. These cells can divide and differentiate to give rise to neural progenitor cells, which in turn divide and differentiate, yielding neurons and neuroglia. In the brain, neural stem cells occupy certain areas but are exceedingly rare. Researchers are attempting to harness the natural ability of neural stem and progenitor cells to divide and replace damaged or diseased neural tissue.

10.1 INTRODUCTION

The nervous system oversees all that we do and largely determines who we are. Through a vast communicating network of cells and the biochemicals that they send and receive, the nervous system can detect changes in the body, make decisions on the basis of the information received, and stimulate muscles or glands to respond. Typically, these responses counteract the effects of the changes, and in this way, the nervous system helps maintain homeostasis. Clinical Application 10.1 discusses how environmental changes may trigger migraine headaches, a common medical problem attributed to the nervous system that may involve its blood supply as well as neurons.

The nervous system is composed predominantly of neural tissue, but also includes blood vessels and connective tissue. Neural tissue consists of two cell types: nerve cells, or **neurons** (nu′ronz), and **neuroglia** (nu-ro′gle-ah) (or neuroglial cells). Neurons are specialized to react to physical and chemical changes in their surroundings. Small cellular processes called **dendrites** (den′drīts) receive the input, and a longer process called an **axon** (ak′son), or nerve fiber, carries the information away from the cell in the form of bioelectric signals called **nerve impulses** (fig. 10.1). **Nerves** are bundles of axons. Neuroglia were once thought only to fill spaces and surround or support neurons. Today, we know that they have many other functions, including nourishing neurons and perhaps even sending and receiving messages.

An important part of the nervous system at the cellular level is not a cell at all, but the small space between a neuron and the cell(s) with which it communicates called a **synapse** (sin′aps). Much of the work of the nervous system is to send and receive electrochemical messages between neurons and other cells at synapses. Biological messenger molecules called **neurotransmitters** (nu″ro-trans-mit′erz) are the actual conveyors of this neural information.

The organs of the nervous system can be divided into two groups. One group, consisting of the brain and spinal cord, forms the **central nervous system (CNS)**, and the other, composed of the nerves (cranial and spinal nerves) that connect the central nervous system to other body parts, is called the **peripheral nervous system (PNS)** (fig. 10.2).

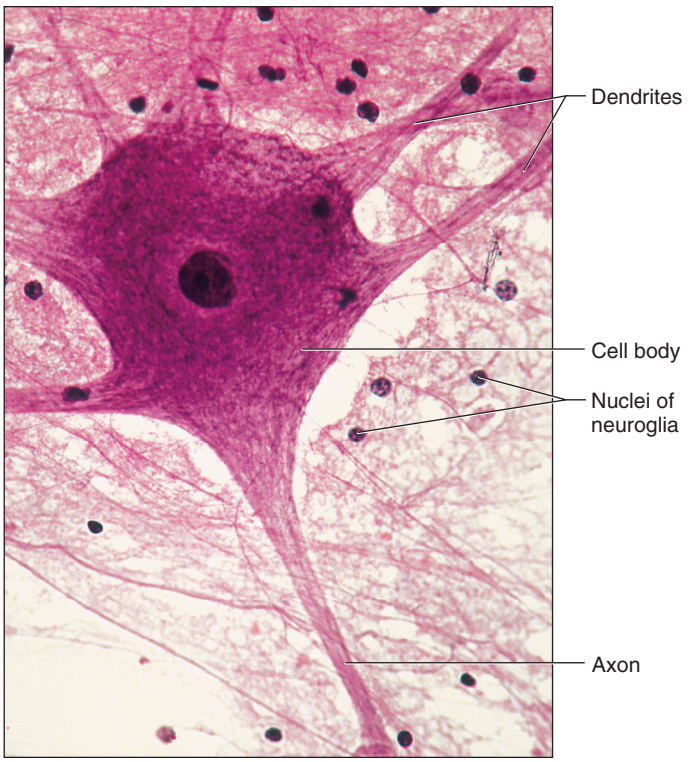


FIGURE 10.1 Neurons are the structural and functional units of the nervous system (600×). Neuroglia are cells that surround and support a neuron, appearing as dark dots. Note the locations of the neuron processes (dendrites and a single axon).

10.2 GENERAL FUNCTIONS OF THE NERVOUS SYSTEM

The three general functions of the nervous system—receiving, deciding, and reacting to stimuli—are termed sensory, integrative, and motor. Structures called **sensory receptors** at the ends of peripheral neurons provide the sensory function of the nervous system (see chapter 11, p. 389). These receptors gather information by detecting changes inside and outside the body. They monitor external environmental factors such as light and sound intensities as well as the temperature, oxygen concentration, and other conditions of the body's internal environment.

Sensory receptors convert (or transduce) their information into nerve impulses, which are then transmitted over peripheral nerves to the CNS. There the signals are integrated—that is, they are brought together, creating sensations, adding to memory, or helping produce thoughts. Following integration, conscious or subconscious decisions are made and then acted upon by means of motor functions.

The motor functions of the nervous system are carried out by neurons that carry impulses from the CNS to responsive structures called *effectors*. These effectors are outside the nervous system and include muscles that contract in response to nerve impulse stimulation and glands that secrete when stimulated. The motor portion of the PNS

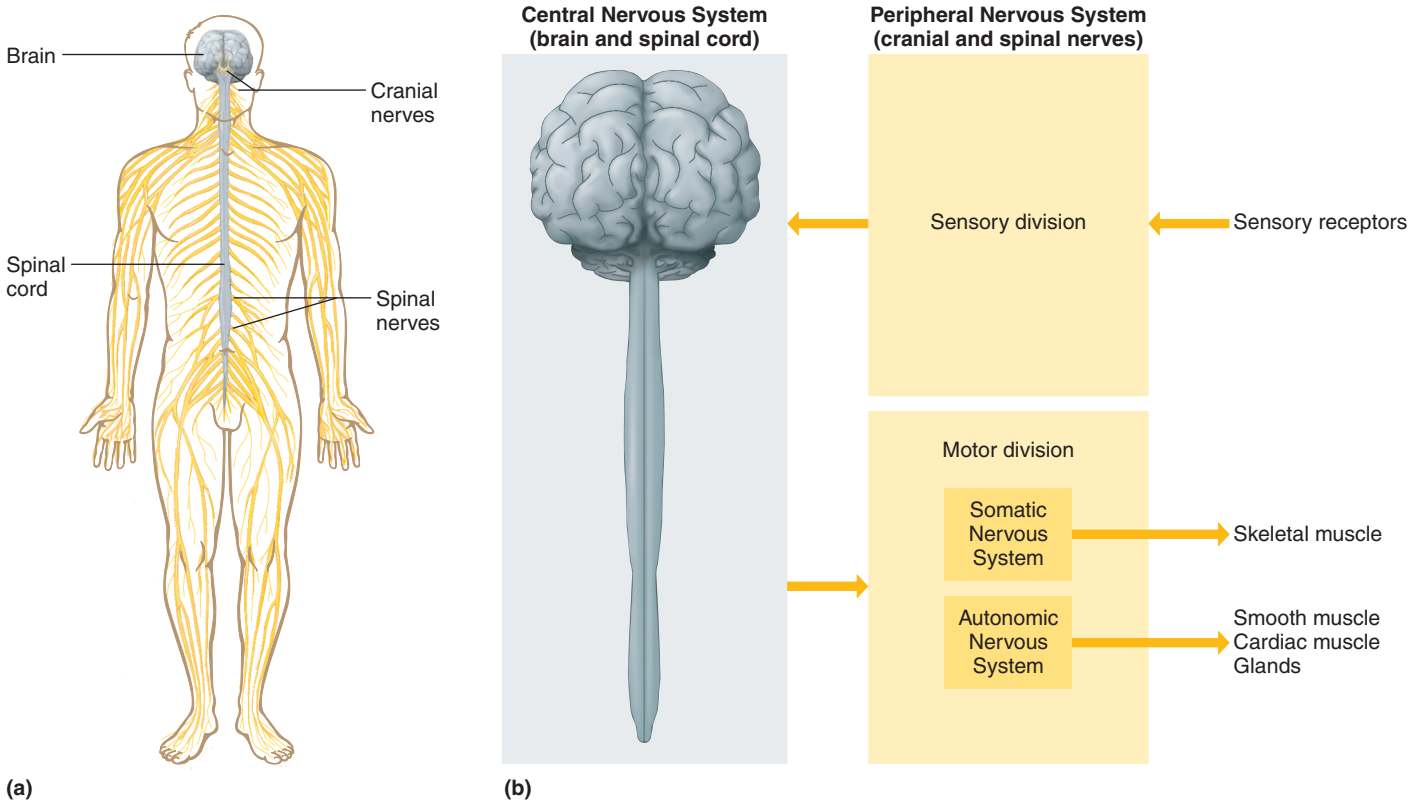


FIGURE 10.2 A diagrammatic representation of the nervous system. (*a*) The nervous system includes the central nervous system (brain and spinal cord) and the peripheral nervous system (cranial nerves and spinal nerves). (*b*) The nervous system receives information from sensory receptors and initiates responses through effector organs (muscles and glands).

10.1 CLINICAL APPLICATION

Migraine

The signs of a migraine are unmistakable—a pounding head, waves of nausea, sometimes shimmering images in the peripheral visual field, and extreme sensitivity to light or sound. Inherited susceptibilities and environmental factors probably cause migraines. Environmental triggers include sudden exposure to bright light, eating a particular food (chocolate, red wine, nuts, and processed meats top the list), lack of sleep, stress, high altitude, stormy weather, and excessive caffeine or alcohol intake. Hormonal influences may also be involved, because two-thirds of the 300 million people who suffer from migraines worldwide are women between the ages of 15 and 55.

A migraine attack may last only a few hours, or days. It is due to a phenomenon called "cortical spreading depression," in which an intense wave of excitation followed by a brief period of unresponsiveness in certain neurons stimulates the trigeminal nucleus at the base of the brain to produce pain sensations. The excitation and dampening of the activity level of these neurons also triggers changes in blood flow in the brain that were once thought to be the direct cause of migraine.

Drugs called triptans can very effectively halt a migraine attack, but must be taken as soon as symptoms begin. Triptans block the release of neurotransmitter from the trigeminal nerves. Because triptans constrict blood vessels throughout the body, making them dangerous for some people, newer migraine drugs have been developed that block the specific neurotransmitter that the trigeminal nerves release (calcitonin gene-related peptide), better targeting the therapeutic effect.

Several drugs developed to treat other conditions are used on a long-term, daily basis to lessen the frequency of migraines. These drugs include certain antidepressants, anticonvulsants, and drugs used to treat high blood pressure (calcium channel blockers and beta blockers). A physician must consider an individual's family and health history before prescribing these drugs to prevent migraine. ■

can be subdivided into the somatic and the autonomic nervous systems. Generally the **somatic nervous system** oversees conscious (voluntary) activities, such as skeletal muscle contraction. The **autonomic nervous system** controls viscera, such as the heart and various glands, and thus controls subconscious (involuntary) actions.

10.3 DESCRIPTION OF CELLS OF THE NERVOUS SYSTEM

Neurons vary in size and shape. They may differ in the lengths and sizes of their axons and dendrites and in the number of processes. Despite this variability, neurons share certain features. Every neuron has a **cell body,** dendrites, and an axon. Figure 10.3 shows some of the other structures common to neurons.

A neuron's cell body (soma or perikaryon) contains granular cytoplasm, mitochondria, lysosomes, a Golgi apparatus, and many microtubules. A network of fine threads called **neurofibrils** extends into the axon and supports it. Scattered throughout the cytoplasm are many membranous packets of **chromatophilic substance** (Nissl bodies), which consist mainly of rough endoplasmic reticulum. Cytoplasmic inclusions in neurons contain glycogen, lipids, or pigments such as melanin. Near the center of the neuron cell body is a large, spherical nucleus with a conspicuous nucleolus.

Dendrites are typically highly branched, providing receptive surfaces with which processes from other neurons communicate. (In some types of neurons, the cell body provides such a receptive surface.) Some dendrites have tiny, thornlike spines (dendritic spines) on their surfaces, which are contact points for other neurons.

A neuron may have many dendrites, but only one axon. The axon, which often arises from a slight elevation of the cell body (axonal hillock), is a slender, cylindrical process with a nearly smooth surface and uniform diameter. It is specialized to conduct nerve impulses away from the cell body. The cytoplasm of the axon includes many mitochondria, microtubules, and neurofibrils (ribosomes are found only in the cell body). The axon may give off branches, called *collaterals.* Near its end, an axon may have many fine extensions, each with a specialized ending called an *axon terminal.* This ends as a *synaptic knob* close to the receptive surface of another cell, separated only by a space called the **synaptic cleft.**

In addition to conducting nerve impulses, an axon conveys biochemicals produced in the neuron cell body, which can be quite a task in these long cells. In this activity, called *axonal transport,* vesicles, mitochondria, ions, nutrients, and neurotransmitters move from the cell body to the ends of the axon.

In the PNS, neuroglia called **Schwann cells** encase the large axons of peripheral neurons in lipid-rich sheaths. These tight coverings form as layers of cell membrane and wind around the axons somewhat like a bandage wrapped around a finger. The layers are composed of **myelin** (mi′ĕ-lin), which has a higher proportion of lipid than other cell membranes. This coating is called a *myelin sheath.* The parts of the Schwann cells that contain most of the cytoplasm and the nuclei remain outside the myelin sheath and comprise a **neurilemma** (nur″īlem′ah), or *neurilemmal sheath,* which surrounds the myelin sheath. Narrow gaps in the myelin sheath between Schwann cells are called **nodes of Ranvier** (fig. 10.4).

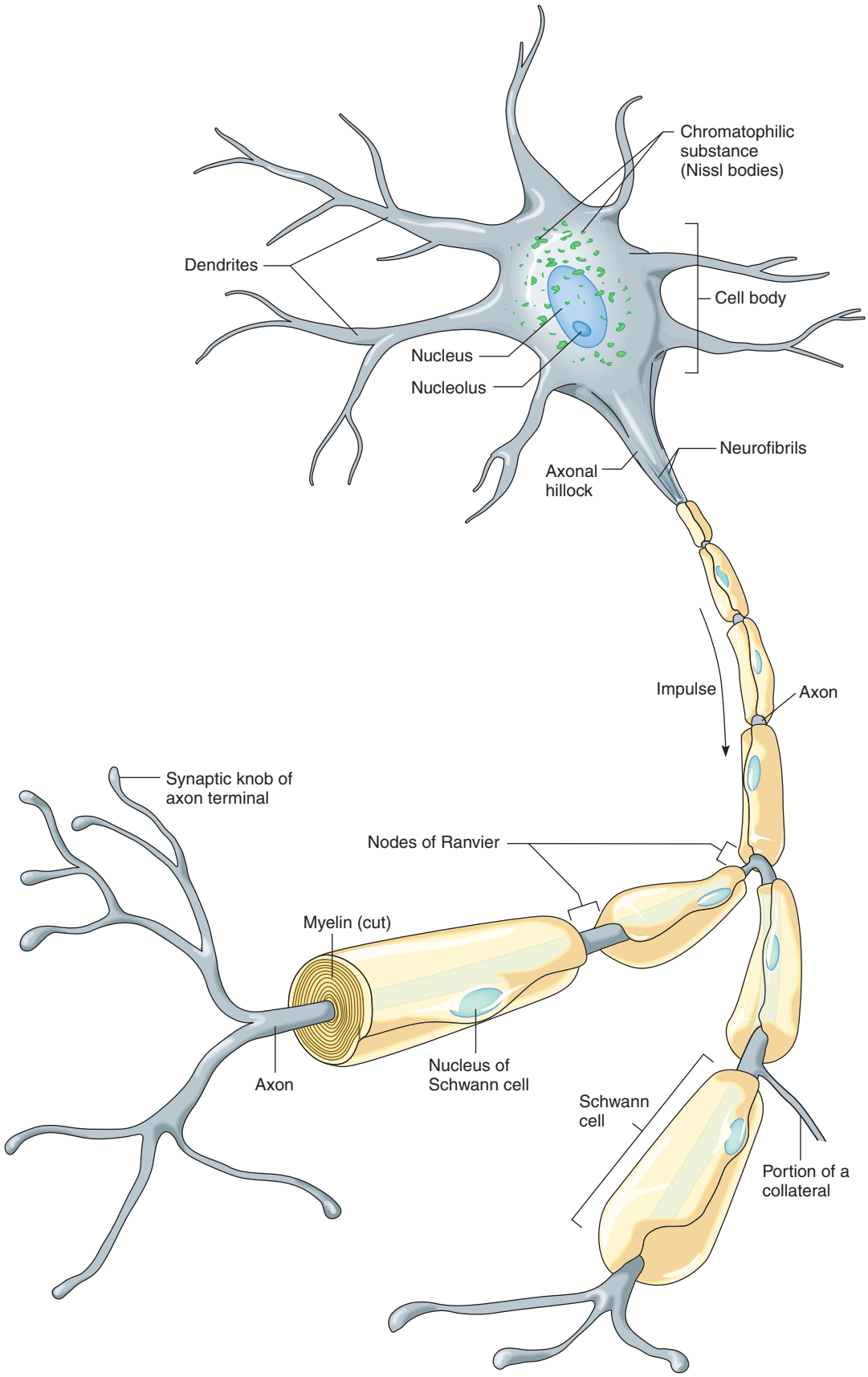


FIGURE 10.3 A common neuron.

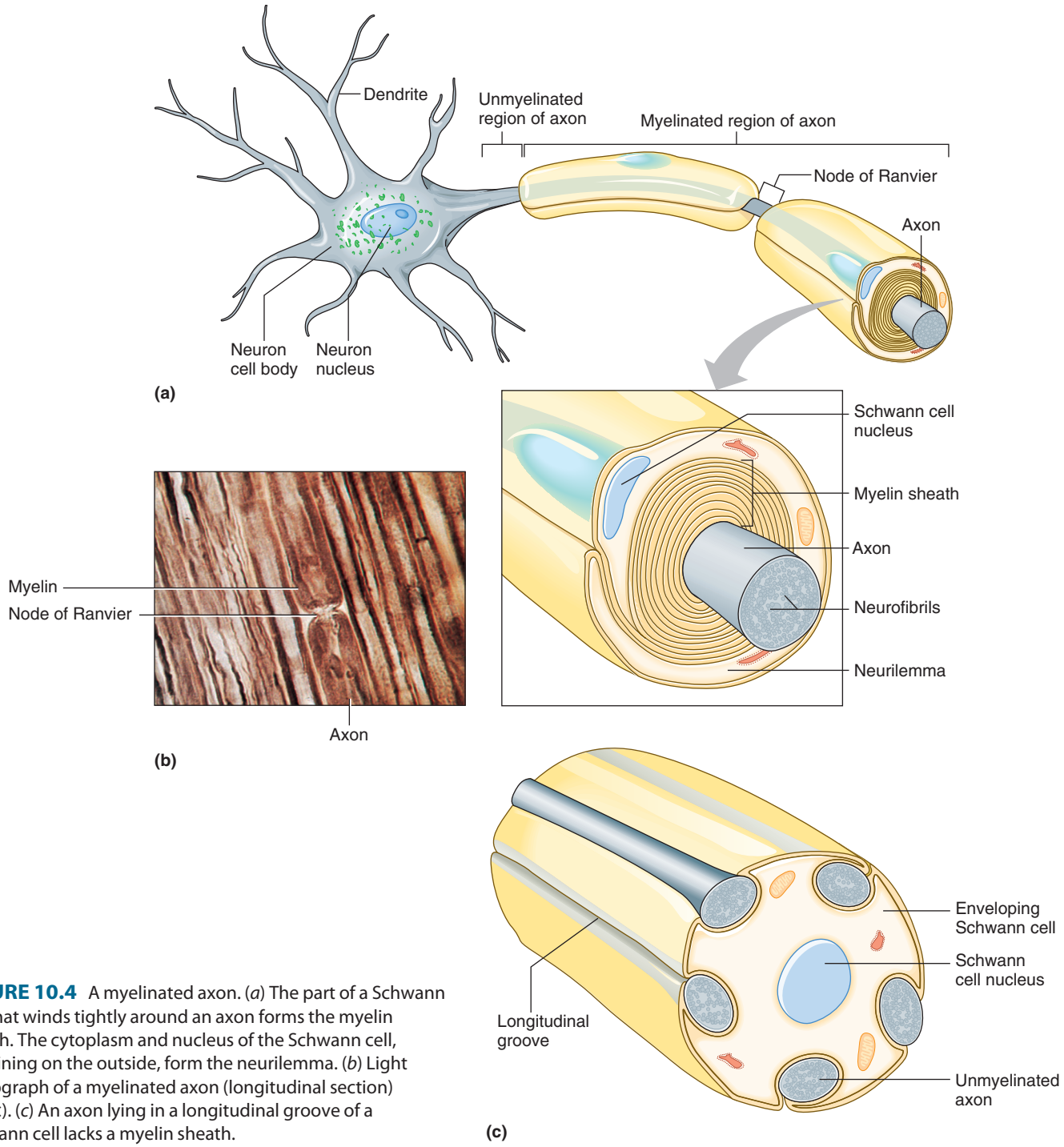


FIGURE 10.4 A myelinated axon. (*a*) The part of a Schwann cell that winds tightly around an axon forms the myelin sheath. The cytoplasm and nucleus of the Schwann cell, remaining on the outside, form the neurilemma. (*b*) Light micrograph of a myelinated axon (longitudinal section) (650×). (*c*) An axon lying in a longitudinal groove of a Schwann cell lacks a myelin sheath.

Schwann cells also enclose, but do not wind around, the smallest axons of peripheral neurons. Consequently, these axons lack myelin sheaths. Instead, the axon or a group of axons may lie partially or completely in a longitudinal groove of Schwann cells.

Axons that have myelin sheaths are called *myelinated* (medullated) axons, and those that lack these sheaths are *unmyelinated axons* (fig. 10.5). Groups of myelinated axons appear white. Masses of such axons impart color to the *white matter* in the brain and spinal cord, but in the CNS another type of neuroglial cell called an **oligodendrocyte** produces myelin. In the brain and spinal cord, myelinated axons lack neurilemmae.

Unmyelinated nerve tissue appears gray. Thus, the *gray matter* in the CNS contains many unmyelinated axons and neuron cell bodies. Clinical Application 10.2 discusses multiple sclerosis, in which neurons in the brain and spinal cord lose their myelin.

PRACTICE

1. List the general functions of the nervous system.
2. Describe a neuron.
3. Explain how an axon in the peripheral nervous system becomes myelinated.

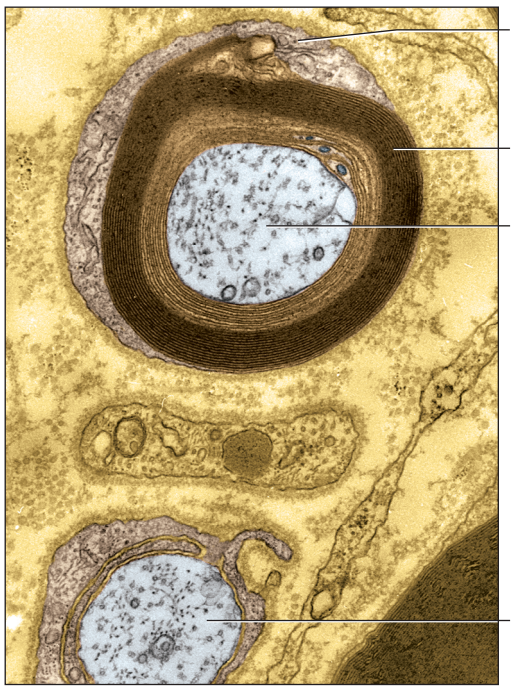


FIGURE 10.5 A falsely colored transmission electron micrograph of myelinated and unmyelinated axons in cross section (30,000×).

Myelin begins to form on axons during the fourteenth week of prenatal development. By the time of birth, many axons are not completely myelinated. All myelinated axons have begun to develop sheaths by the time a child starts to walk, and myelination continues into adolescence.

Excess myelin seriously impairs nervous system functioning. In Tay-Sachs disease, an inherited defect in a lysosomal enzyme causes myelin to accumulate, burying neurons in fat. The affected child begins to show symptoms by six months of age, gradually losing sight, hearing, and muscle function until death occurs by age four. Thanks to genetic screening among people of eastern European descent who are most likely to carry this gene, Tay-Sachs disease is extremely rare.

10.4 CLASSIFICATION OF CELLS OF THE NERVOUS SYSTEM

Neurons and neuroglia are intimately related. They descend from the same neural stem cells and remain associated throughout their existence.

Classification of Neurons

Neurons vary in size and shape and may differ in the lengths and sizes of their axons and dendrites and in the number of dendrites. Based on *structural differences*, neurons can be classified into three major groups, as figure 10.6 shows. Each

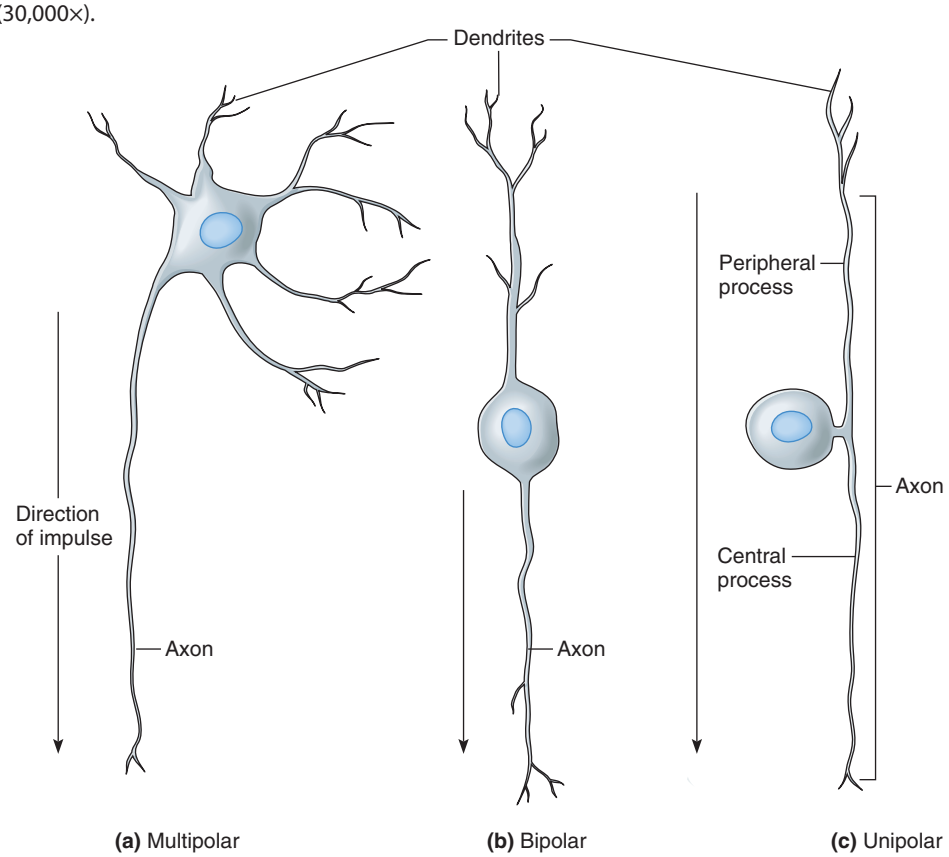


FIGURE 10.6 Structural types of neurons include (*a*) the multipolar neuron, (*b*) the bipolar neuron, and (*c*) the unipolar neuron.

10.2 CLINICAL APPLICATION

Multiple Sclerosis

Multiple sclerosis (MS) is a disorder of the CNS that affects 2.5 million people worldwide, and 400,000 in North America. In addition to overt nervous system symptoms, affected individuals experience disability, mood problems such as depression, and great fatigue. Four subtypes of MS are recognized, based on the pattern of symptomatic periods over time.

In MS, the myelin coating in various sites through the brain and spinal cord becomes inflamed due to an immune response and is eventually destroyed, leaving hard scars, called scleroses, that block the underlying neurons from transmitting messages. Muscles that no longer receive input from motor neurons stop contracting, and eventually, they atrophy. Symptoms reflect the specific neurons affected. Shortcircuiting in one part of the brain may affect fine coordination in one hand; if another brain part is affected, vision may be altered.

The first symptoms of MS are often blurred vision and numb legs or arms, but because in many cases these are intermittent, diagnosis may take awhile. Diagnosis is based on symptoms and repeated magnetic resonance (MR) scans, which can track development of lesions. A diagnostic work-up for MS might also include a lumbar puncture to rule out infection and an evoked potential test to measure electrical signals sent from the brain. About 70% of affected individuals first notice symptoms between the ages of twenty and forty; the earliest known age of onset is three years, and the latest, sixty-seven years. Some affected individuals eventually become permanently paralyzed. Women are twice as likely to develop MS as men, and Caucasians are more often affected than people of other races.

MS may develop when certain infections in certain individuals stimulate T cells (a type of white blood cell that takes part in immune responses) in the periphery, which then cross the blood-brain barrier. Here, the T cells attack myelin-producing cells through a flood of inflammatory molecules and by stimulating other cells to produce antibodies against myelin.

A virus may lie behind the misplaced immune attack that is MS. Evidence includes the observations that viral infection can cause repeated bouts of symptoms, as can MS, and that MS is much more common in some geographical regions (the temperate zones of Europe, South America, and North America) than others, suggesting a pattern of infection.

Various drugs are used to manage MS. Drugs to decrease bladder spasms can temper problems of urinary urgency and incontinence. Antidepressants are sometimes prescribed, and short-term steroid drugs are used to shorten the length of acute disabling relapses. Muscle relaxants ease stiffness and spasms.

Several drugs are used for long-term treatment of MS. Beta interferons are immune system biochemicals that are widely prescribed, even after only one attack if MS seems a likely diagnosis. This treatment diminishes the intensity of flare-ups, but effects on the course of illness over time are not yet known. Beta interferons may cause flulike adverse effects. They are self-injected once to several times a week.

Glatiramer is an alternative to beta interferons. It is prescribed if the course of the disease is "relapsing remitting," with periodic flare-ups. Glatiramer is self-injected daily and dampens the immune system's attack on myelin. It consists of part of myelin basic protein, the most abundant protein of myelin. In response, T cells decrease inflammation. Glatiramer also stimulates increased production of brain-derived neurotrophic factor, which protects axons.

Mitoxantrone is another drug that halts the immune response against CNS myelin, but because it can damage the heart, it is typically used in severe cases of MS and only up to two years. Another drug, natalizumab, prevents T cells from binding blood vessels in the brain, also quelling the abnormal immune response against myelin. It too may have rare but serious adverse effects. ■

type of neuron is specialized to send a nerve impulse in one direction.

1. **Multipolar neurons.** Multipolar neurons have many processes arising from their cell bodies. Only one is an axon; the rest are dendrites. Most neurons whose cell bodies lie within the brain or spinal cord are of this type. The neuron illustrated in figure 10.3 is multipolar.
2. **Bipolar neurons.** The cell body of a bipolar neuron has only two processes, one arising from either end. Although these processes are similar in structure, one is an axon and the other is a dendrite. Bipolar neurons are found in specialized parts of the eyes, nose, and ears.
3. **Unipolar neurons.** Each unipolar neuron has a single process extending from its cell body. A short distance from the cell body, this process divides into two branches, which really function as a single axon: One branch (peripheral process) is associated with dendrites near a peripheral body part. The other branch (central process) enters the brain or spinal cord. The cell bodies of some unipolar neurons aggregate in specialized masses of nerve tissue called **ganglia,** located outside the brain and spinal cord.

Neurons can also be classified by *functional differences* into the following groups, depending on whether they carry information into the CNS, completely within the CNS, or out of the CNS (fig. 10.7).

1. **Sensory neurons** (afferent neurons) carry nerve impulses from peripheral body parts into the brain or spinal cord. At their distal ends, the dendrites of these neurons or specialized structures associated with them act as sensory receptors, detecting changes in the outside world (for example, eyes, ears, or touch receptors in the skin) or in the body (for example, temperature or blood pressure receptors). When sufficiently stimulated, sensory receptors trigger impulses that travel on sensory neuron axons into the brain or spinal cord. Most sensory neurons are

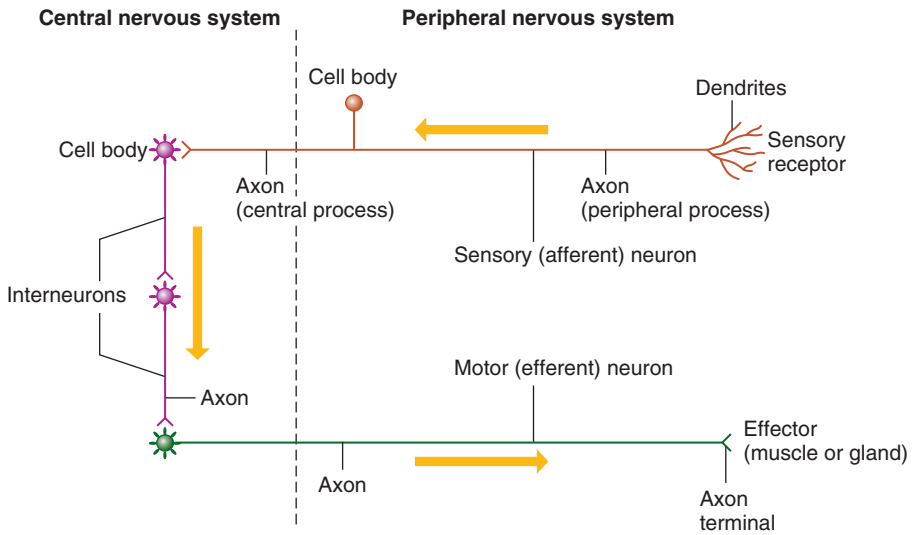


FIGURE 10.7 Neurons are classified by function as well as structure. Sensory (afferent) neurons carry information into the central nervous system (CNS), interneurons are completely within the CNS, and motor (efferent) neurons carry instructions to effectors.

unipolar, as shown in figure 10.7, although some are bipolar.

2. **Interneurons** (also called association or internuncial neurons) lie within the brain or spinal cord. They are multipolar and form links between other neurons. Interneurons transmit impulses from one part of the brain or spinal cord to another. That is, they may direct incoming sensory impulses to appropriate regions for processing and interpreting. Other incoming impulses are transferred to motor neurons.
3. **Motor neurons** (efferent neurons) are multipolar and carry nerve impulses out of the brain or spinal cord to effectors—structures that respond, such as muscles or glands. For example, when motor impulses reach muscles, they contract; when motor impulses reach glands, they release secretions.

   Motor neurons of the somatic nervous system (see fig. 10.2) that control skeletal muscle contraction are under voluntary (conscious) control. Those that control cardiac and smooth muscle contraction and the secretions of glands are part of the autonomic nervous system and are largely under involuntary control.

Table 10.1 summarizes the classification of neurons.

Classification of Neuroglia

Neuroglia were once thought to be mere bystanders to neural function, providing scaffolding and controlling the sites at which neurons contact one another (figs. 10.8 and 10.9). These important cells have additional functions. In the embryo, neuroglia guide neurons to their positions and may stimulate them to specialize. Neuroglia also produce the growth factors that nourish neurons and remove ions and neurotransmitters that accumulate between neurons, enabling them to continue transmitting information. In cell culture experiments, certain types of neuroglia (astrocytes) signal neurons to form and maintain synapses.

Neuroglia of the CNS

The four types of CNS neuroglia are astrocytes, oligodendrocytes, microglia, and ependyma:

1. **Astrocytes.** As their name implies, astrocytes are star-shaped cells. They are commonly found between neurons and blood vessels, where they provide support and hold structures together with abundant cellular processes. Astrocytes aid metabolism of certain substances, such as glucose, and they may help regulate the concentrations of important ions, such as potassium ions, in the interstitial space of nervous tissue. Astrocytes also respond to injury of brain tissue and form a special type of scar tissue, which fills spaces and closes gaps in the CNS. These multifunctional cells also have a nutritive function, regulating movement of substances from blood vessels to neurons and bathing nearby neurons in growth factors. Astrocytes play an important role in the blood-brain barrier, which restricts movement of substances between the blood and the CNS (see Clinical Application 5.1, p. 145). Gap junctions link astrocytes to one another, forming protein-lined channels through which calcium ions travel, possibly stimulating neurons.
2. **Oligodendrocytes.** Oligodendrocytes resemble astrocytes but are smaller and have fewer processes. They form in rows along myelinated axons, and produce myelin in the brain and spinal cord.

   Unlike the Schwann cells of the PNS, oligodendrocytes can send out a number of processes, each of which forms a myelin sheath around a nearby axon. In this way,

TABLE 10.1 | Types of Neurons

A. Classified by Structure		
Type	**Structural Characteristics**	**Location**
1. Multipolar neuron	Cell body with many processes, one of which is an axon, the rest dendrites	Most common type of neuron in the brain and spinal cord
2. Bipolar neuron	Cell body with a process, arising from each end, one axon and one dendrite	In specialized parts of the eyes, nose, and ears
3. Unipolar neuron	Cell body with a single process that divides into two branches and functions as an axon	Found in ganglia outside the brain or spinal cord
B. Classified by Function		
Type	**Functional Characteristics**	**Structural Characteristics**
1. Sensory neuron	Conducts nerve impulses from receptors in peripheral body parts into the brain or spinal cord	Most unipolar; some bipolar
2. Interneuron	Transmits nerve impulses between neurons in the brain and spinal cord	Multipolar
3. Motor neuron	Conducts nerve impulses from the brain or spinal cord out to effectors—muscles or glands	Multipolar

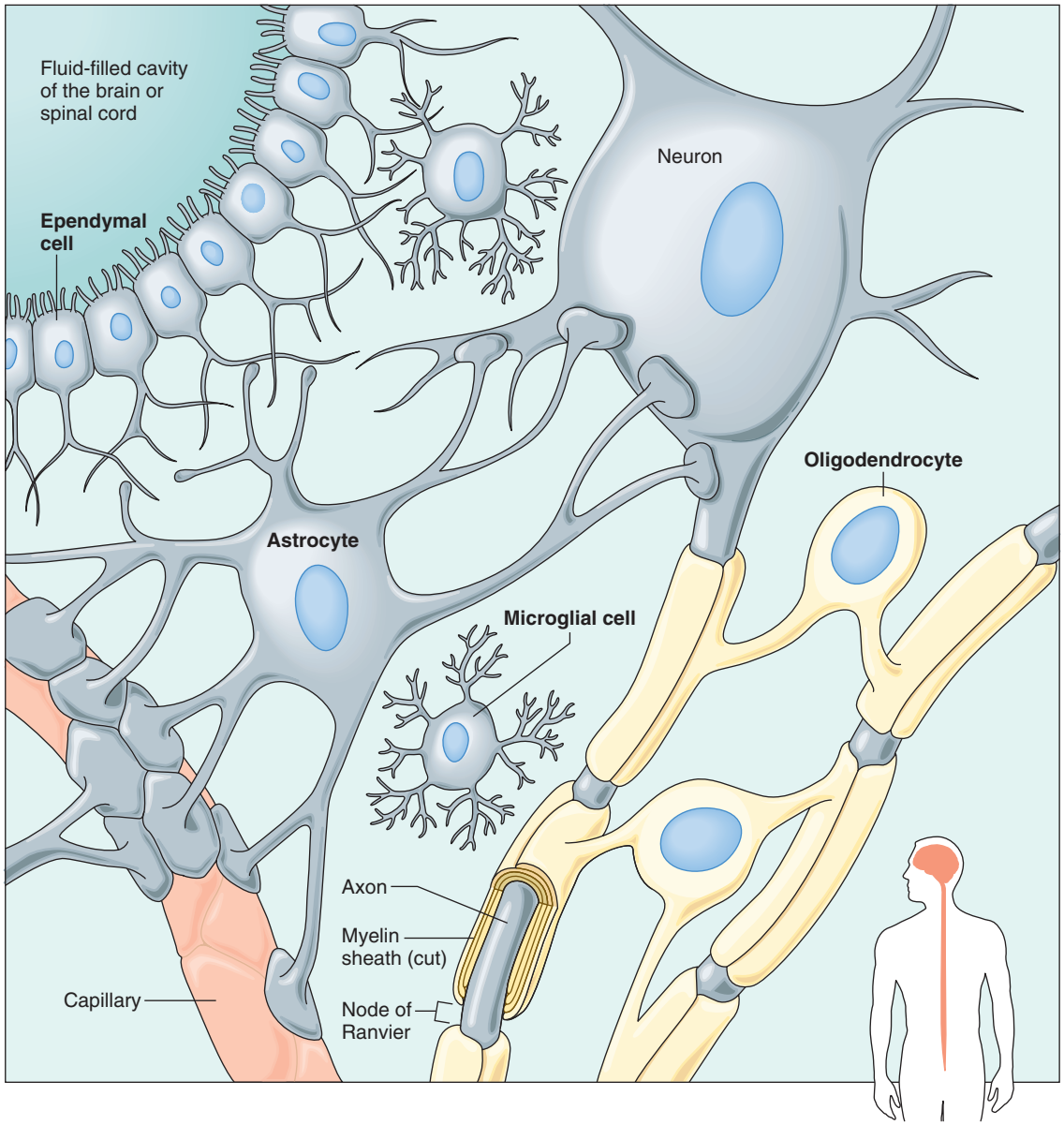


FIGURE 10.8 Types of neuroglia in the central nervous system include the astrocyte, oligodendrocyte, microglial cell, and ependymal cell. Cilia are on most ependymal cells during development and early childhood, but in the adult are mostly on ependymal cells in the ventricles of the brain.

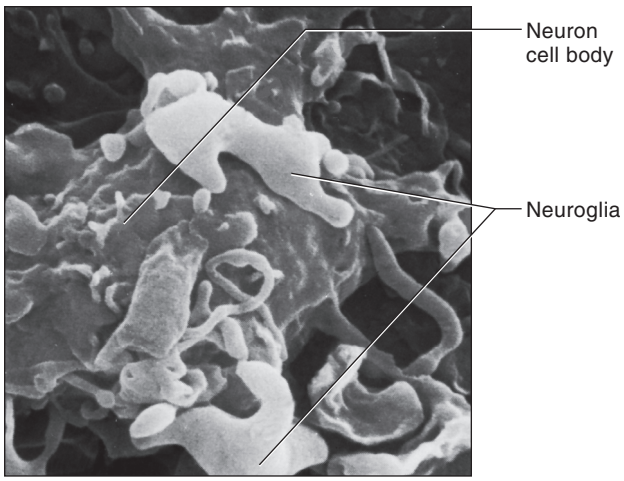


FIGURE 10.9 A scanning electron micrograph of a neuron cell body and some of the neuroglia associated with it (10,000×). (Tissues and Organs: *A Text-Atlas of Scanning Electron Microscopy,* by R. G. Kessel and R. H. Kardon, (c) 1979 W. H. Freeman and Company.)

a single oligodendrocyte may provide myelin for many axons. However, these cells do not form neurilemmae.

3. **Microglia.** Microglial cells are small and have fewer processes than other types of neuroglia. These cells are scattered throughout the CNS, where they help support neurons and phagocytize bacterial cells and cellular debris. They usually proliferate whenever the brain or spinal cord is inflamed because of injury or disease.
4. **Ependyma.** Ependymal cells are cuboidal or columnar in shape and may have cilia. They form the inner lining of the *central canal* that extends downward through the spinal cord. Ependymal cells also form a one-cell-thick epithelial-like membrane that covers the inside of spaces in the brain called *ventricles* (see chapter 11, pp. 385–386). Here, gap junctions join ependymal cells, forming a porous layer through which substances diffuse freely between the interstitial fluid of the brain tissues and the fluid (cerebrospinal fluid) in the ventricles.

   Ependymal cells also cover the specialized capillaries called *choroid plexuses* associated with the ventricles of the brain. Here they help regulate the composition of the cerebrospinal fluid.

Neuroglia, which comprise more than half of the volume of the brain and outnumber neurons 10 to 1, are critical to neuron function.

Abnormal neuroglia are associated with certain disorders. Most brain tumors, for example, consist of neuroglia that divide too often. Neuroglia that produce toxins may lie behind some neurodegenerative disorders. In one familial form of amyotrophic lateral sclerosis (ALS, or Lou Gehrig's disease), astrocytes release a toxin that destroys motor neurons, causing progressive weakness. In Huntington disease (HD), which causes uncontrollable movements and cognitive impairment, microglia in the brain release a toxin that damages neurons. In both ALS and HD, only specific sets of neurons are affected. Identifying the unexpected roles of neuroglia in nervous system disorders suggests new targets for treatments.

Neuroglia of the PNS

The two types of neuroglia in the peripheral nervous system are Schwann cells and **satellite cells:**

1. Schwann cells produce the myelin on peripheral myelinated neurons, as described earlier.
2. Satellite cells support clusters of neuron cell bodies called *ganglia*, in the PNS.

Table 10.2 summarizes the characteristics and functions of neuroglia.

TABLE 10.2 | Types of Neuroglia

Type	Characteristics	Functions
CNS		
Astrocytes	Star-shaped cells between neurons and blood vessels	Structural support, formation of scar tissue, transport of substances between blood vessels and neurons, communicate with one another and with neurons, mop up excess ions and neurotransmitters, induce synapse formation
Oligodendrocytes	Shaped like astrocytes, but with fewer cellular processes, occur in rows along axons	Form myelin sheaths in the brain and spinal cord, produce nerve growth factors
Microglia	Small cells with few cellular processes and found throughout the CNS	Structural support and phagocytosis (immune protection)
Ependyma	Cuboidal and columnar cells in the inner lining of the ventricles of the brain and the central canal of the spinal cord	Form a porous layer through which substances diffuse between the interstitial fluid of the brain and spinal cord and the cerebrospinal fluid
PNS		
Schwann cells	Cells with abundant, lipid-rich membranes that wrap tightly around the axons of peripheral neurons	Speed neurotransmission
Satellite cells	Small, cuboidal cells that surround cell bodies of neurons in ganglia	Support ganglia in the PNS

Regeneration of Neuroglia and Axonal

Injury to the cell body usually kills the neuron, and because mature neurons do not divide, the destroyed cell is not replaced unless neural stem cells become stimulated to proliferate. However, a damaged peripheral axon may regenerate. For example, if injury or disease separates an axon in a peripheral nerve from its cell body, the distal portion of the axon and its myelin sheath deteriorate within a few weeks. Macrophages remove the fragments of myelin and other cellular debris. The proximal end of the injured axon develops sprouts shortly after the injury. Influenced by nerve growth factors that nearby neuroglia secrete, one of these sprouts may grow into a tube formed by remaining basement membrane and connective tissue. At the same time, any remaining Schwann cells proliferate along the length of the degenerating portion and form new myelin around the growing axon.

Growth of a regenerating axon is slow (3 to 4 millimeters per day), but eventually the new axon may reestablish the former connection (fig. 10.10). Nerve growth factors, secreted by neuroglia, may help direct the growing axon. However, the regenerating axon may still end up in the wrong place, so full function often does not return.

If an axon of a neuron within the CNS is separated from its cell body, the distal portion of the axon will degenerate, but more slowly than a separated axon in the PNS. However, axons in the CNS lack neurilemmae, and the myelin-producing oligodendrocytes do not proliferate following injury. Consequently, the proximal end of a damaged axon that begins to grow has no tube of sheath cells to guide it. Therefore, regeneration is unlikely.

If a peripheral nerve is severed, it is important that the two cut ends be connected as soon as possible so that the regenerating sprouts of the axons can more easily reach the tubes formed by the basement membranes and connective tissues on the distal side of the gap. When the gap exceeds 3 millimeters, the regenerating axons may form a tangled mass called a neuroma. It is composed of sensory axons and is painfully sensitive to pressure. Neuromas sometimes complicate a patient's recovery following limb amputation.

PRACTICE

4. What are neuroglia?
5. Name and describe four types of neuroglia.
6. What are some functions of neuroglia?
7. Explain how an injured peripheral axon might regenerate.
8. Explain why functionally significant regeneration is unlikely in the CNS.

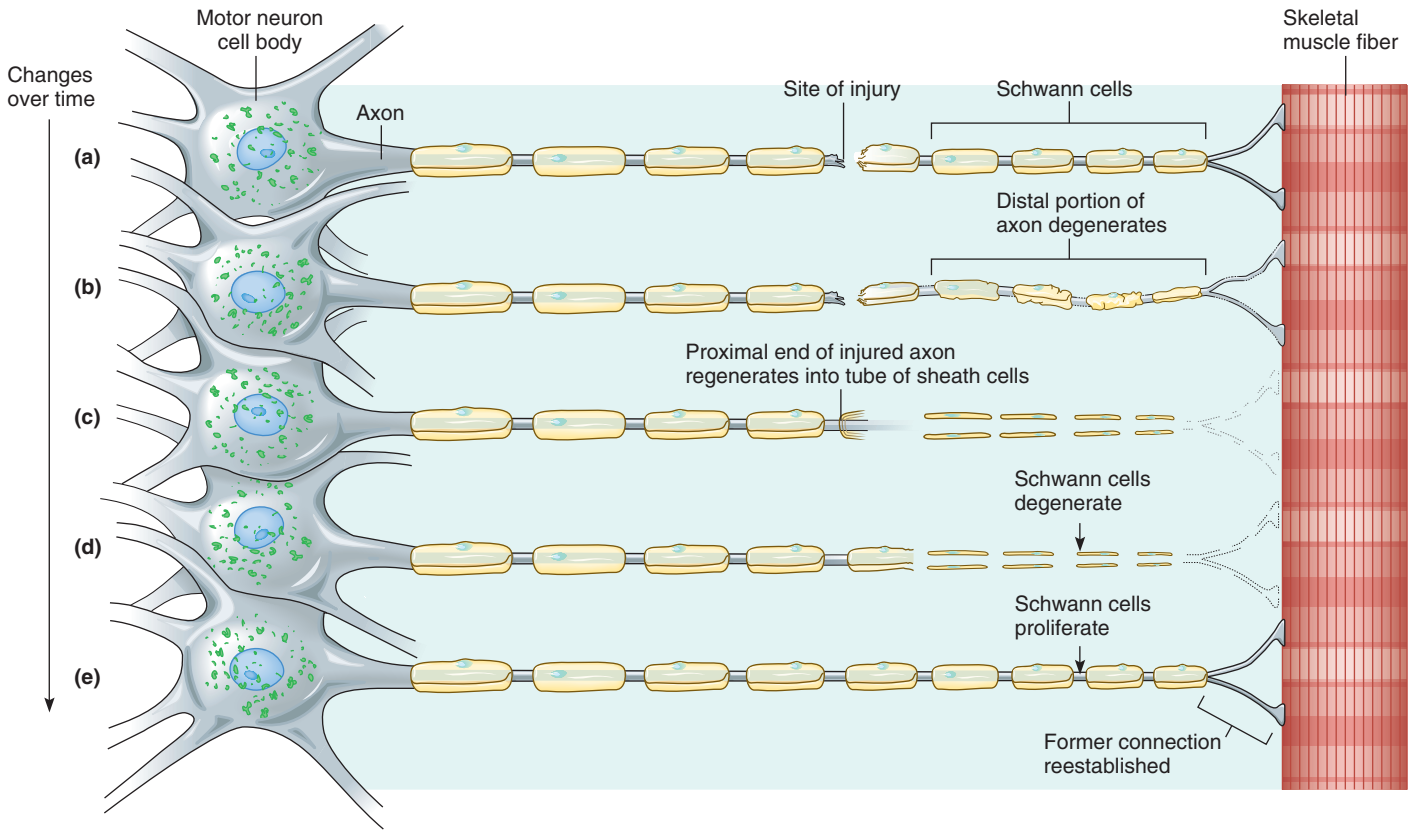


FIGURE 10.10 If a myelinated axon is injured, the following events may occur over several weeks to months: (*a*) The proximal portion of the axon may survive, but (*b*) the portion distal to the injury degenerates. (*c* and *d*) In time, the proximal portion may develop extensions that grow into the tube of basement membrane and connective tissue cells that the axon previously occupied and (*e*) possibly reestablish the former connection. Nerve growth factors that neuroglia secrete assist in the regeneration process.

Neurons do not divide. New neural tissue arises from neural stem cells, which give rise to neural progenitor cells that can give rise to neurons or neuroglia. In the adult brain, the rare neural stem cells are in a region called the dentate gyrus and near fluid-filled cavities called ventricles.

Neural stem cells were discovered in the 1980s, in songbirds—the cells were inferred to exist because the numbers of neurons waxed and waned with the seasons, peaking when the birds learned songs. Moving songbirds far from food, forcing them to sing longer, resulted in more brain neurons, thanks to the stem cells. In the 1990s, researchers identified the cells in brain slices from marmosets and tree shrews given a drug that marks dividing cells. Then they were discovered in humans when a researcher learned that patients with tongue and larynx cancer were taking the drug to mark their cancer cells. Five patients donated their brains after their deaths, and researchers identified the cells. Today, human neural stem and progenitor cells are being used to screen drugs and are being delivered as implants to experimentally treat a variety of brain disorders. One day, a person's neural stem cells may be coaxed to help heal from within.

10.5 THE SYNAPSE

Nerve impulses pass from neuron to neuron (or to other cells) at synapses (fig. 10.11). A **presynaptic neuron** brings the impulse to the synapse and, as a result, stimulates or inhibits a **postsynaptic neuron** (or a muscle or gland). A *synaptic cleft,* or gap, separates the two cells (fig. 10.12), which are connected functionally, not physically. The process by which the impulse in the presynaptic neuron signals the postsynaptic cell is called **synaptic transmission.**

A nerve impulse travels along the axon to the axon terminal. Axons usually have several rounded synaptic knobs at their terminals, which dendrites lack. These knobs have arrays of membranous sacs, called synaptic vesicles, that contain neurotransmitter molecules. When a nerve impulse reaches a synaptic knob, voltage-sensitive calcium channels open and calcium diffuses inward from the extracellular fluid. The increased calcium concentration inside the cell initiates a series of events that fuses the synaptic vesicles with the cell membrane, where they release their neurotransmitter by exocytosis.

Once the neurotransmitter binds to receptors on a postsynaptic cell, the action of neurotransmitter on the postsynaptic cell is either excitatory (turning a process on) or inhibitory (turning a process off). The net effect on the postsynaptic cell depends on the combined effect of the excitatory and inhibitory inputs from as few as 1 to 100,000 or more presynaptic neurons.

10.6 CELL MEMBRANE POTENTIAL

A cell membrane is usually electrically charged, or *polarized,* so that the inside is negatively charged with respect to the outside. This polarization is due to an unequal distribution of positive and negative ions on either side of the membrane. It is important in the conduction of muscle and nerve impulses.

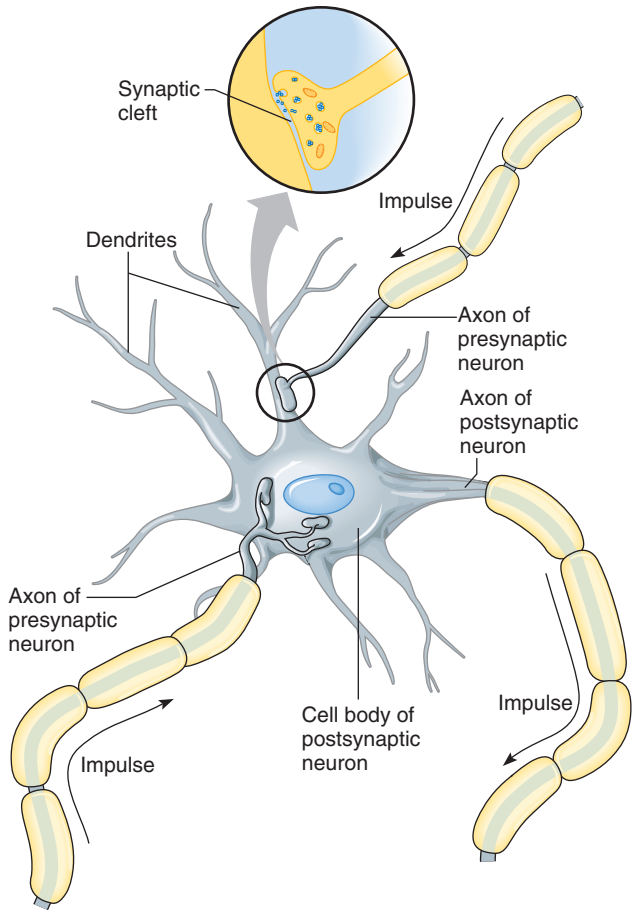


FIGURE 10.11 For an impulse to continue from one neuron to another, it must cross the synaptic cleft. A synapse usually separates an axon and a dendrite or an axon and a cell body.

Distribution of Ions

Potassium ions (K^+) are the major intracellular positive ion (cation), and sodium ions (Na^+) are the major extracellular cation. The distribution is created largely by the sodium–potassium pump (Na^+/K^+pump), which actively transports sodium ions out of the cell and potassium ions into the cell. It is also in part due to channels in the cell membrane that determine membrane permeability to these ions. These channels, formed by membrane proteins, can be selective; that is, a particular channel may allow only one type of ion to pass through and exclude all other ions of different size and charge. Thus, even though concentration gradients are present for sodium and potassium, the ability of these ions to diffuse across the cell membrane depends on the presence of channels.

RECONNECT
To Chapter 3, Cell Membrane, page 80.

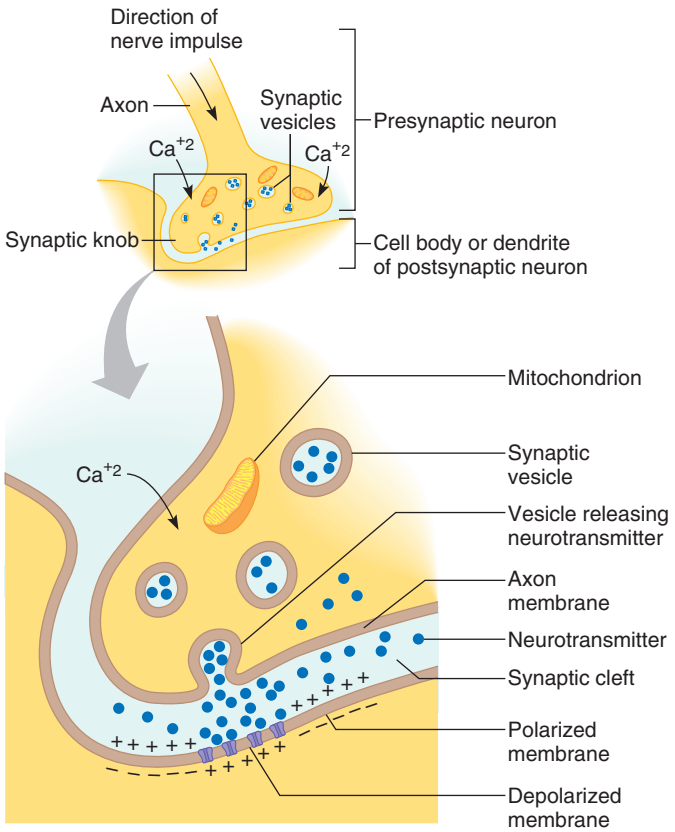


FIGURE 10.12 The synapse. (*a*) When a nerve impulse reaches the synaptic knob at the end of an axon, synaptic vesicles release a neurotransmitter that diffuses across the synaptic cleft. In this case the neurotransmitter is excitatory. (*b*) A transmission electron micrograph of a synaptic knob filled with synaptic vesicles (37,500×).

Some channels are always open, whereas others may be either open or closed, somewhat like a gate. Both chemical and electrical factors can affect the opening and closing of these *gated channels* (fig. 10.13).

Resting Potential

A resting nerve cell is not being stimulated to send a nerve impulse. Under resting conditions, nongated (always open) channels determine the membrane permeability to sodium and potassium ions.

Sodium and potassium ions follow the laws of diffusion described in chapter 3 (pp. 90 and 92) and show a net movement from areas of high concentration to areas of low concentration across a membrane as their permeabilities permit. The resting cell membrane is only slightly permeable to these ions, but the membrane is more permeable to potassium ions than to sodium ions (fig. 10.14*a*). Also, the cytoplasm of these cells has many negatively charged ions (anions) which include phosphate (PO_4^{-2}), sulfate (SO_4^{-2}), and proteins, that are synthesized inside the cell and cannot diffuse through cell membranes.

If we consider a hypothetical neuron, before a membrane potential has been established, we would expect potassium to diffuse out of the cell more rapidly than sodium could diffuse in. This means that every millisecond (as the membrane potential is being established in our hypothetical cell), a few more positive ions leave the cell than enter it (fig. 10.14*a*). As a result, the outside of the membrane gains a slight surplus of positive charges, and the inside reflects a surplus of the impermeant negatively charged ions. This creates a separation of positive and negative electrical charges between the inside and outside surfaces of the cell membrane (fig. 10.14*b*). All this time, the cell continues to expend metabolic energy in the form of ATP to actively transport sodium and potassium ions in opposite directions, thus maintaining the concentration gradients for those ions responsible for their diffusion in the first place.

The difference in electrical charge between two points is measured in units called volts. It is called a potential difference because it represents stored electrical energy that can be used to do work at some future time. The potential difference across the cell membrane is called the **membrane potential** (transmembrane potential) and is measured in millivolts.

In the case of a resting neuron, one that is not sending impulses or responding to other neurons, the membrane potential is termed the **resting potential** (resting membrane potential) and has a value of –70 millivolts (fig. 10.14*b*). The negative sign is relative to the inside of the cell and is due to the excess negative charges on the inside of the cell membrane. To understand how the resting potential provides the energy for sending a nerve impulse down the axon, we must first understand how neurons respond to signals called stimuli.

With the resting membrane potential established, a few sodium ions and potassium ions continue to diffuse across the cell membrane. The negative membrane potential helps sodium ions enter the cell despite sodium's low permeability, but it hinders potassium ions from leaving the cell despite potassium's higher permeability. The net effect is that three sodium ions "leak" into the cell for every two potassium ions that "leak" out. The Na^+/K^+ pump exactly balances these leaks by pumping three sodium ions out for every two potassium ions it pumps in (fig. 10.14*c*).

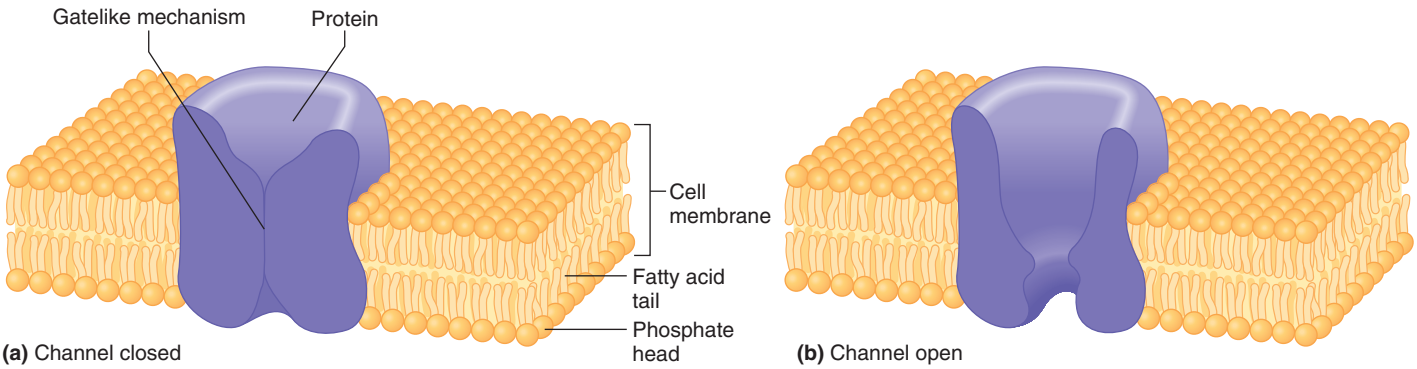


FIGURE 10.13 A gatelike mechanism can (*a*) close or (*b*) open some of the channels in cell membranes through which ions pass.

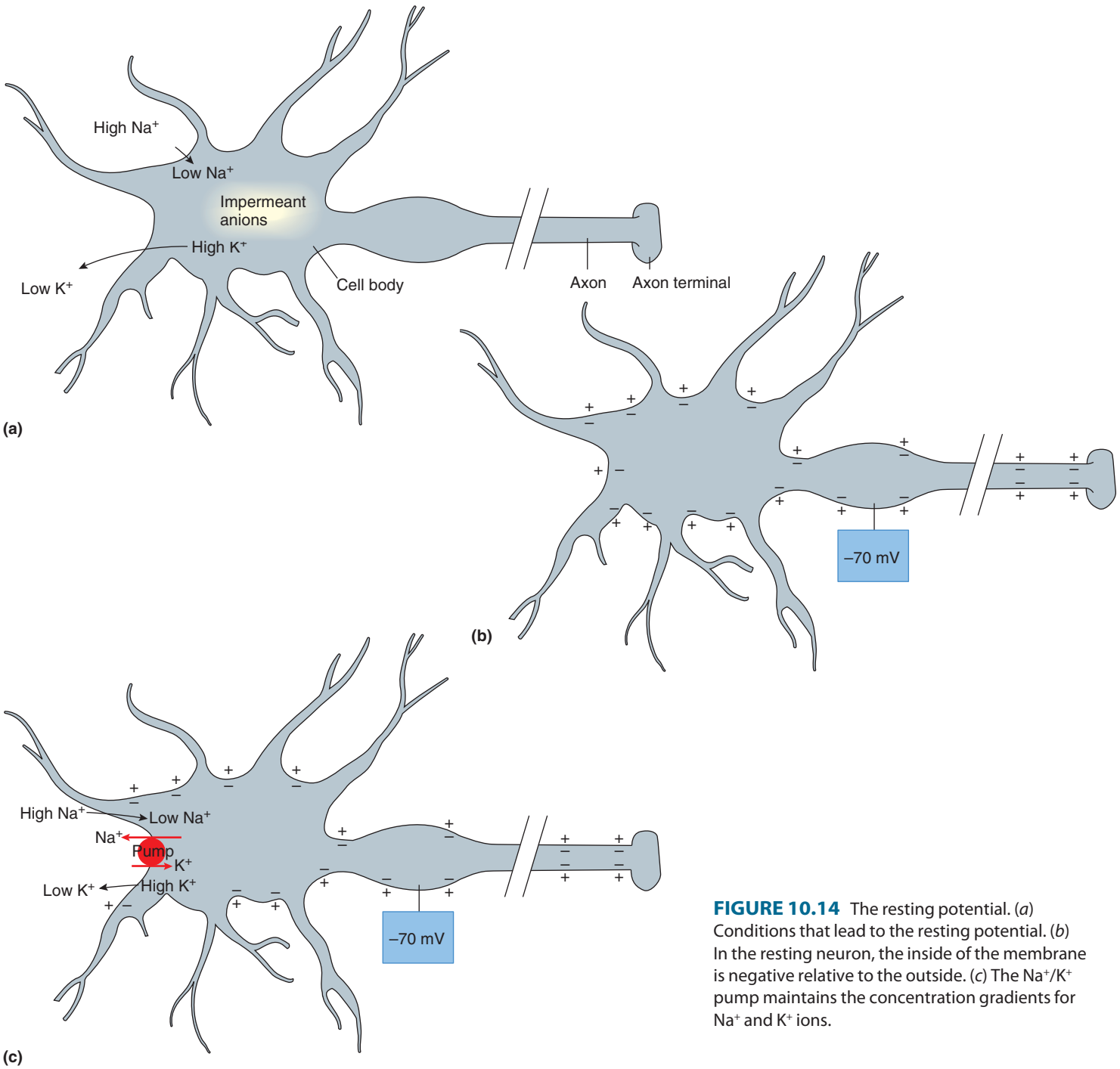


FIGURE 10.14 The resting potential. (*a*) Conditions that lead to the resting potential. (*b*) In the resting neuron, the inside of the membrane is negative relative to the outside. (*c*) The Na^+/K^+ pump maintains the concentration gradients for Na^+ and K^+ ions.

Local Potential Changes

Neurons are excitable; that is, they can respond to changes in their surroundings. Some neurons, for example, detect changes in temperature, light, or pressure outside the body, whereas others respond to signals from inside the body, often from other neurons. In either case, such changes or stimuli usually affect the membrane potential in the region of the membrane exposed to the stimulus, causing a local potential change.

Typically, the environmental change affects the membrane potential by opening a gated ion channel. If, as a result, the membrane potential becomes more negative than the resting potential, the membrane is *hyperpolarized*. If the membrane becomes less negative (more positive) than the resting potential, the membrane is *depolarized*.

Local potential changes are graded. This means that the degree of change in the resting potential is directly proportional to the intensity of the stimulation. For example, if the membrane is being depolarized, the greater the stimulus, the greater the depolarization. If neurons are sufficiently depolarized, the membrane potential reaches a level called the **threshold potential,** approximately –55 millivolts in a neuron. If threshold is reached, an **action potential** results, which is the basis for the nerve impulse.

In many cases, a single depolarizing stimulus is not sufficient to bring the postsynaptic cell to threshold. For example, if presynaptic neurons release enough neurotransmitter to open some chemically-gated sodium channels for a moment, the depolarization that results might be insufficient to reach threshold (fig. 10.15*a*). Such a subthreshold depolarization will not result in an action potential.

If the presynaptic neurons release more neurotransmitter, or if other neurons that synapse with the same cell join in the effort to depolarize, threshold may be reached, and an action potential results. The mechanism uses another type of ion channel, a voltage-gated sodium channel that opens when threshold is reached (fig. 10.15*b*).

Action Potentials

In a multipolar neuron, the first part of the axon, the *initial segment,* is often referred to as the **trigger zone** because it contains many voltage-gated sodium channels. At the resting membrane potential, these sodium channels remain closed, but when threshold is reached, they open for an

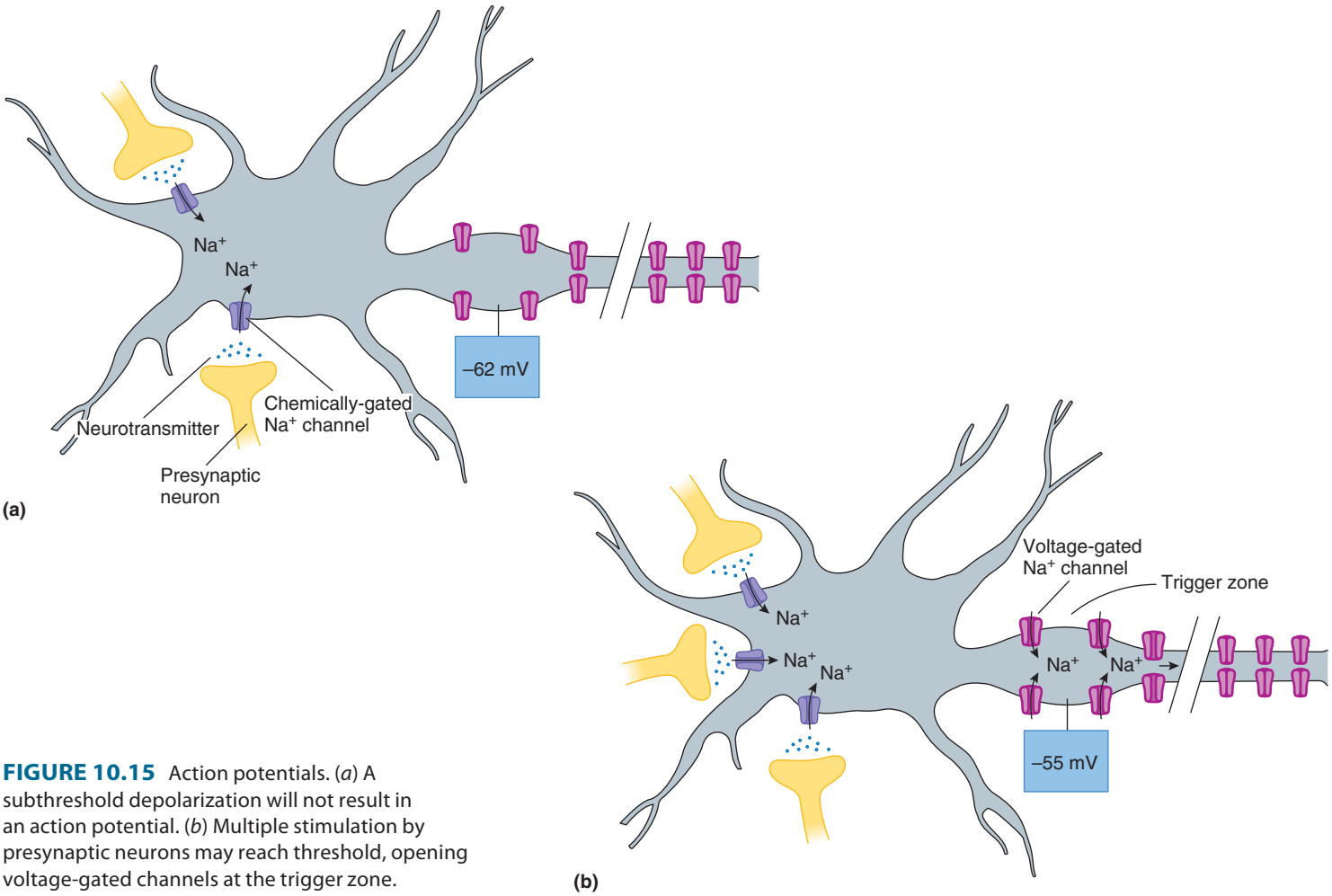


FIGURE 10.15 Action potentials. (*a*) A subthreshold depolarization will not result in an action potential. (*b*) Multiple stimulation by presynaptic neurons may reach threshold, opening voltage-gated channels at the trigger zone.

instant, briefly increasing sodium permeability. Sodium ions diffuse inward across that part of the cell membrane, down their concentration gradient, aided by the attraction of the sodium ions to the negative electrical condition on the inside of the membrane.

As the sodium ions diffuse inward, the membrane potential changes from its resting value (fig. 10.16*a*) and momentarily becomes positive on the inside (still considered depolarization). At the peak of the action potential, the membrane potential may reach +30mV (fig. 10.16*b*).

The voltage-gated sodium channels quickly close, but at almost the same time, slower voltage-gated potassium channels open and briefly increase potassium permeability. As potassium ions diffuse outward across that part of the membrane, the inside of the membrane becomes negatively charged once more. The membrane is thus repolarized (note in fig. 10.16*c* that it hyperpolarizes for an instant). The voltage-gated potassium channels then close as well. In this way, the resting potential is quickly reestablished, and it remains in the resting state until it is stimulated again (fig. 10.17). The active transport mechanism in the membrane works to maintain the original concentrations of sodium and potassium ions.

Axons are capable of action potentials, but the cell body and dendrites are not. An action potential at the trigger zone causes an electric current to flow a short distance down the axon, which stimulates the adjacent membrane to reach its threshold level, triggering another action potential. The second action potential causes another electric current to flow farther down the axon. This sequence of events results in a series of action potentials sequentially occurring all the way to the end of the axon without decreasing in amplitude, even if the axon branches. The propagation of action potentials along an axon is the nerve impulse (fig. 10.18).

A nerve impulse is similar to the muscle impulse mentioned in chapter 9, page 290. In the muscle fiber, stimulation at the motor end plate triggers an impulse to travel over

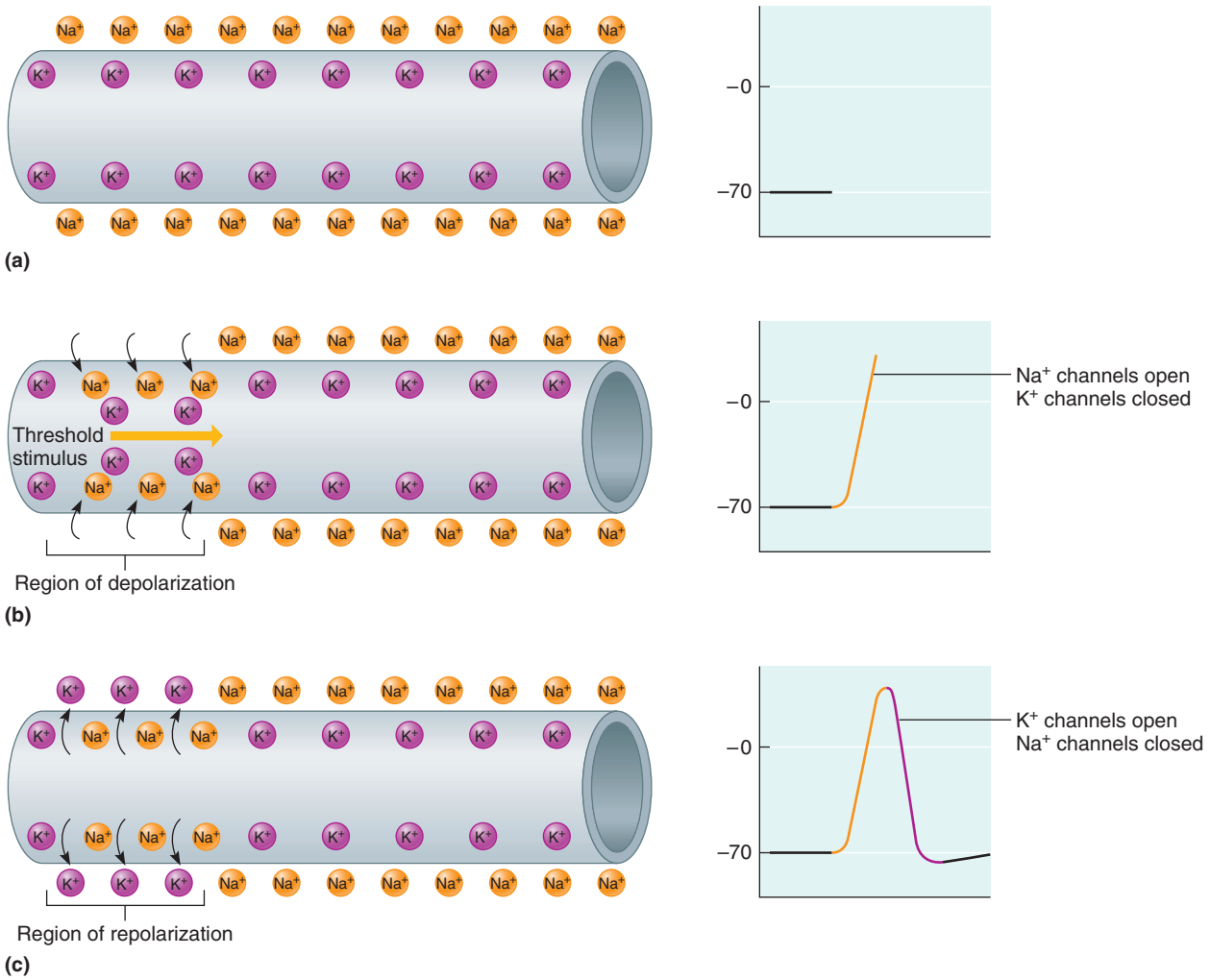


FIGURE 10.16 At rest (*a*), the membrane potential is about –70 millivolts. When the membrane reaches threshold (*b*), voltage-sensitive sodium channels open, some Na^+ diffuses inward, and the membrane is depolarized. Soon afterward (*c*), voltage-sensitive potassium channels open, K^+ diffuses out, and the membrane is repolarized. (Negative ions not shown.)

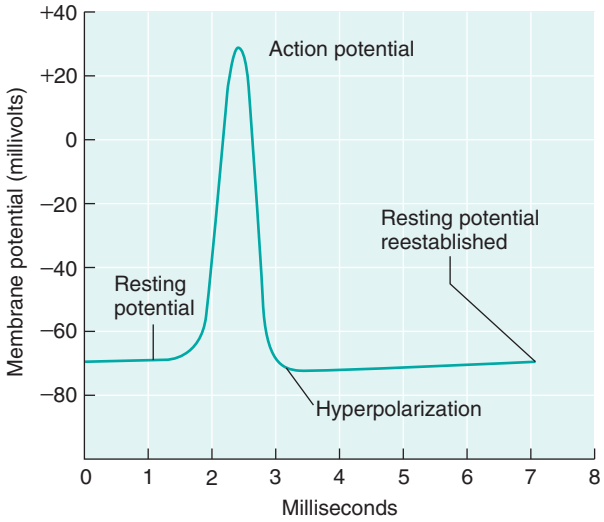


FIGURE 10.17 An oscilloscope records an action potential.

the surface of the fiber and down into its transverse tubules. See table 10.3 for a summary of the events leading to the conduction of a nerve impulse.

All-or-None Response

Nerve impulse conduction is an all-or-none response. In other words, if a neuron responds at all, it responds completely. Thus, a nerve impulse is conducted whenever a stimulus of threshold intensity or above is applied to an axon and all impulses carried on that axon are the same strength. A greater intensity of stimulation produces more impulses per second, not a stronger impulse.

Refractory Period

For a short time following passage of a nerve impulse, a threshold stimulus will not trigger another impulse on an axon. This brief period, called the **refractory period,** has two parts. During the *absolute refractory period,* which lasts about 1/2,500 of a second, the axon's membrane is changing in sodium permeability and cannot be stimulated. This is followed by a *relative refractory period,* when the membrane reestablishes its resting potential. While the membrane is in the relative refractory period, even though repolarization is incomplete, a threshold stimulus of high intensity may trigger an impulse.

As time passes, the intensity of stimulation required to trigger an impulse decreases until the axon's original excitability is restored. This return to the resting state usually takes from 10 to 30 milliseconds.

The refractory period limits how many action potentials may be generated in a neuron in a given period. Remembering that the action potential takes about a millisecond, and adding the time of the absolute refractory period, the maximum theoretical frequency of impulses in a neuron is about 700 per second. In the body, this limit is rarely achieved—frequencies of about 100 impulses per second are common.

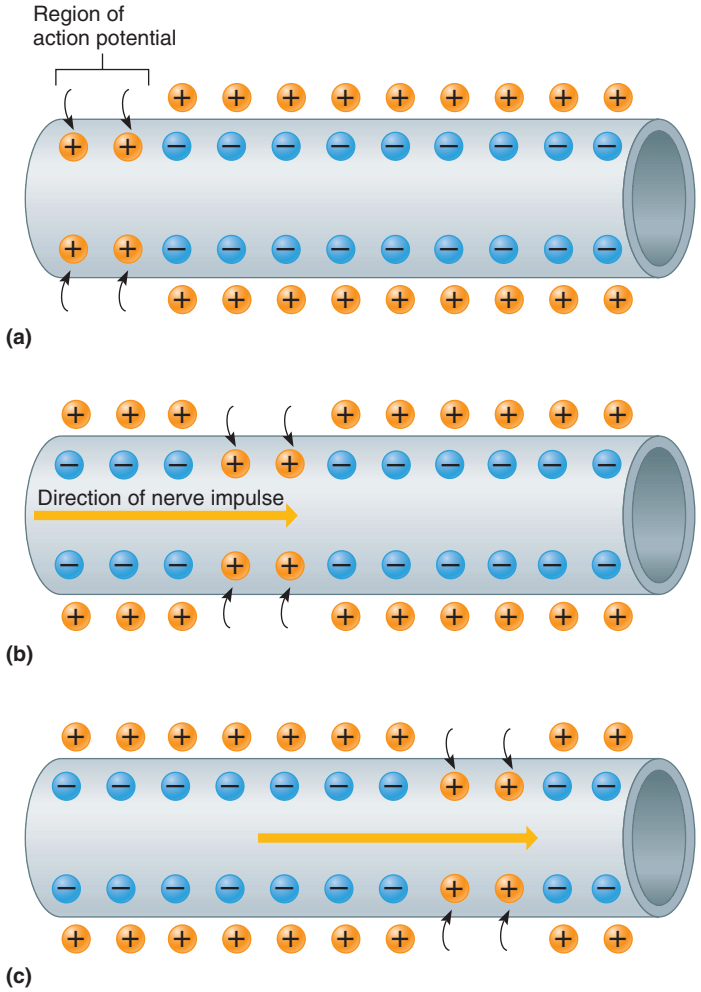


FIGURE 10.18 Nerve impulse. (*a*) An action potential in one region stimulates the adjacent region, and (*b* and *c*) a wave of action potentials (a nerve impulse) moves along the axon.

TABLE 10.3 | Events Leading to Nerve Impulse Conduction

1. Nerve cell membrane maintains resting potential by diffusion of Na^+ and K^+ down their concentration gradients as the cell pumps them up the gradients.
2. Neurons receive stimulation, causing local potentials, which may sum to reach threshold.
3. Sodium channels in the trigger zone of the axon open.
4. Sodium ions diffuse inward, depolarizing the membrane.
5. Potassium channels in the membrane open.
6. Potassium ions diffuse outward, repolarizing the membrane.
7. The resulting action potential causes an electric current that stimulates adjacent portions of the membrane.
8. Action potentials occur sequentially along the length of the axon as a nerve impulse.

Impulse Conduction

An unmyelinated axon conducts an impulse over its entire surface. A myelinated axon functions differently. Myelin contains a high proportion of lipid that excludes water and water-soluble substances. Thus, myelin serves as an electrical insulator and prevents almost all flow of ions through the membrane that it encloses.

It might seem that the myelin sheath would prevent conduction of a nerve impulse, and this would be true if the sheath were continuous. However, nodes of Ranvier between Schwann cells or oligodendrocytes interrupt the sheath (see fig. 10.3). At these nodes, the axon membrane has channels for sodium and potassium ions that open during a threshold depolarization.

When a myelinated axon is stimulated to threshold, an action potential occurs at the trigger zone. This causes an electric current to flow away from the trigger zone through the cytoplasm of the axon. As this local current reaches the first node, it stimulates the membrane to its threshold level, and an action potential occurs there, sending an electric current to the next node. Consequently, in a nerve impulse traveling along a myelinated axon, action potentials occur only at the nodes. The action potentials appear to jump from node to node, so this type of impulse conduction is called **saltatory conduction.** Conduction on myelinated axons is many times faster than conduction on unmyelinated axons (fig. 10.19).

The diameter of the axon also affects the speed of nerve impulse conduction—the greater the diameter, the faster the impulse. An impulse on a thick, myelinated axon, such as that of a motor neuron associated with a skeletal muscle, might travel 120 meters per second, whereas an impulse on a thin, unmyelinated axon, such as that of a sensory neuron associated with the skin, might move only 0.5 meter per second. Clinical Application 10.3 discusses factors that influence nerve impulse conduction.

PRACTICE

9 Summarize how a resting potential is achieved.
10 Explain how a polarized axon responds to stimulation.
11 List the major events of an action potential.
12 Define *refractory* period.
13 Explain how impulse conduction differs in myelinated and unmyelinated axons.

10.7 SYNAPTIC TRANSMISSION

Released neurotransmitter molecules diffuse across the synaptic cleft and react with specific molecules called *receptors* in the postsynaptic neuron membrane. Effects of neurotransmitters vary. Some open ion channels, and others close them. These ion channels respond to neurotransmitter molecules, so they are called *chemically-gated,* in contrast to the voltage-gated ion channels that participate in action potentials. Changes in chemically-gated ion channels create local potentials, called **synaptic potentials,** which enable one neuron to affect another.

Synaptic Potentials

Synaptic potentials can depolarize or hyperpolarize the receiving cell membrane. For example, if a neurotransmitter binds to a postsynaptic receptor and opens sodium ion channels, the ions diffuse inward, depolarizing the membrane, possibly triggering an action potential. This type of membrane change is called an **excitatory postsynaptic potential** (EPSP), and it lasts for about 15 milliseconds.

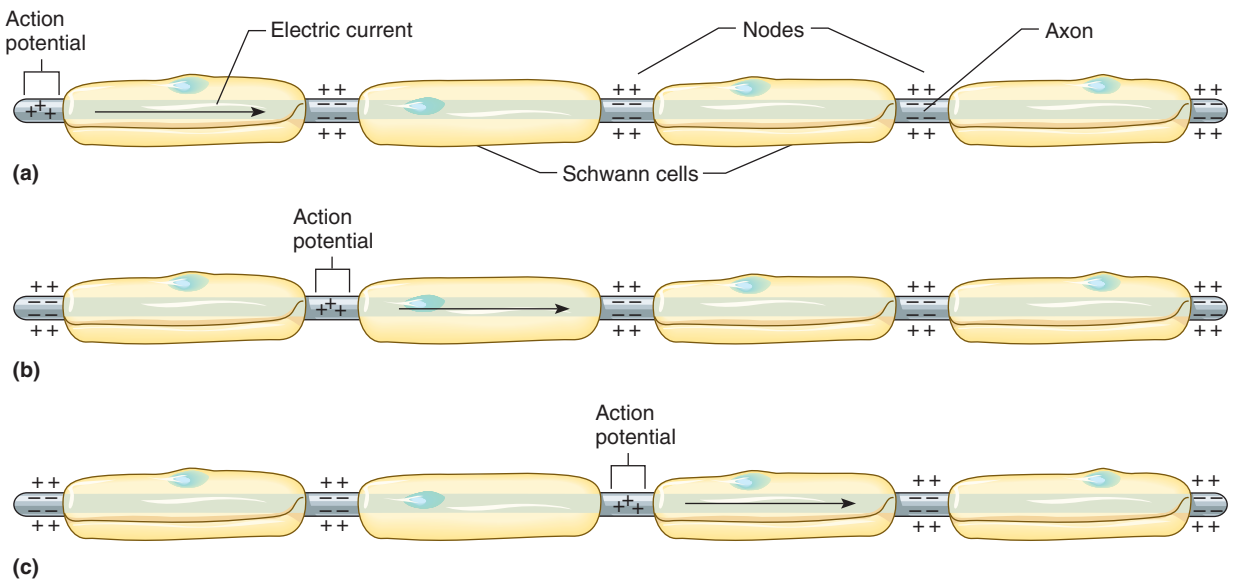


FIGURE 10.19 On a myelinated axon, a nerve impulse appears to jump from node to node.

10.3 CLINICAL APPLICATION

Factors Affecting Impulse Conduction

Painful muscle cramps, convulsions, paralysis, and anesthesia can each result from changes in the permeability of axons to particular ions. A number of substances alter axon membrane permeability to ions.

Calcium ions are required to close sodium channels in axon membranes during an action potential. If calcium is deficient, then sodium channels remain open, and sodium ions diffuse through the membrane continually so that impulses are transmitted repeatedly. If these spontaneous impulses travel along axons to skeletal muscle fibers, the muscles continuously spasm (tetanus or tetany). This can happen to women during pregnancy as the developing fetus uses maternal calcium. Tetanic contraction may also occur when the diet lacks calcium or vitamin D or when prolonged diarrhea depletes the body of calcium.

A small increase in the concentration of extracellular potassium ions causes the resting potential of nerve fibers to be less negative (partially depolarized). As a result, the threshold potential is reached with a less intense stimulus than usual. The affected fibers are excitable, and the person may experience convulsions.

If the extracellular potassium ion concentration is greatly decreased, the resting potentials of the nerve fibers may become so negative that action potentials are not generated. In this case, impulses are not triggered, and muscles become paralyzed.

Certain anesthetic drugs, such as procaine, decrease membrane permeability to sodium ions. In the tissue fluids surrounding an axon, these drugs prevent impulses from passing through the affected region. Consequently, the drugs keep impulses from reaching the brain, preventing perception of touch and pain. ■

If a different neurotransmitter binds other receptors and increases membrane permeability to potassium ions, these ions diffuse outward, hyperpolarizing the membrane. An action potential is now less likely to occur, so this change is called an **inhibitory postsynaptic potential** (IPSP). Some inhibitory neurotransmitters open chloride ion channels. In this case, if sodium ions enter the cell, negative chloride ions are free to follow, opposing the depolarization.

In the brain and spinal cord, each neuron may receive the synaptic knobs of a thousand or more axons on its dendrites and cell body (fig. 10.20). Furthermore, at any moment, some of the postsynaptic potentials are excitatory on a particular neuron, while others are inhibitory.

The integrated sum of the EPSPs and IPSPs determines whether an action potential results. If the net effect is more excitatory than inhibitory, threshold may be reached and an action potential triggered. Conversely, if the net effect is inhibitory, no impulse is transmitted.

Summation of the excitatory and inhibitory effects of the postsynaptic potentials commonly takes place at the trigger zone, usually in a proximal region of the axon, but found also in the distal peripheral process of some sensory neurons. This region has an especially low threshold for triggering an action potential; thus, it serves as a decision-making part of the neuron.

PRACTICE

14 Describe a synapse.
15 Explain the function of a neurotransmitter.
16 Distinguish between an EPSP and an IPSP.
17 Describe the net effects of EPSPs and IPSPs.

Neurotransmitters

The nervous system produces at least thirty different types of neurotransmitters. Some neurons release only one type of neurotransmitter; others produce two or three types. Neurotransmitters include *acetylcholine*, which stimulates skeletal muscle contractions (see chapter 9, p. 290); a group

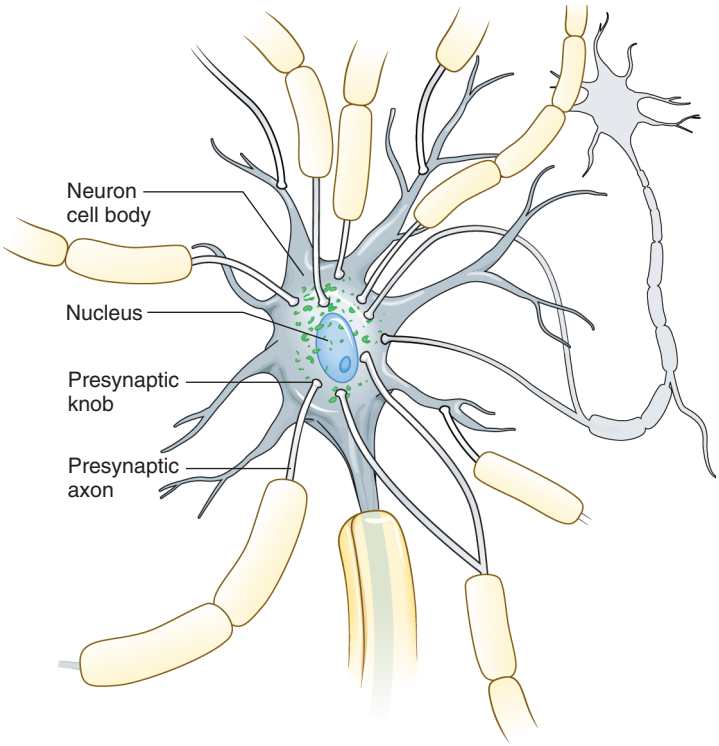


FIGURE 10.20 The synaptic knobs of many axons may communicate with the cell body of a neuron.

of compounds called *monoamines* (such as epinephrine, norepinephrine, dopamine, and serotonin), which are modifications of amino acids; a group of unmodified *amino acids* (such as glycine, glutamic acid, aspartic acid, and gamma-aminobutyric acid—GABA); and a large group of *peptides* (such as enkephalins and substance P), which are short chains of amino acids.

The peptide neurotransmitters are synthesized in the rough endoplasmic reticulum of the neuron cell bodies and transported in vesicles down the axon to the nerve terminal. Other neurotransmitters are synthesized in the cytoplasm of the nerve terminals and stored in vesicles. When an action potential passes along the membrane of a synaptic knob, it increases the membrane's permeability to calcium ions by opening its calcium ion channels. Calcium ions diffuse inward, and in response, some of the synaptic vesicles fuse with the presynaptic membrane and release their contents by exocytosis into the synaptic cleft. The more calcium that enters the synaptic knob, the more vesicles release neurotransmitter. Table 10.4 lists the major neurotransmitters and their actions. Tables 10.5 and 10.6 list disorders and drugs that alter neurotransmitter levels, respectively.

TABLE 10.4 | Some Neurotransmitters and Representative Actions

Neurotransmitter	Location	Major Actions
Acetylcholine	CNS	Controls skeletal muscle actions
	PNS	Stimulates skeletal muscle contraction at neuromuscular junctions. May excite or inhibit at autonomic nervous system synapses
Biogenic amines		
Norepinephrine	CNS	Creates a sense of well-being; low levels may lead to depression
	PNS	May excite or inhibit autonomic nervous system actions, depending on receptors
Dopamine	CNS	Creates a sense of well-being; deficiency in some brain areas associated with Parkinson disease
	PNS	Limited actions in autonomic nervous system; may excite or inhibit, depending on receptors
Serotonin	CNS	Primarily inhibitory; leads to sleepiness; action is blocked by LSD, enhanced by selective serotonin reuptake inhibitor antidepressant drugs
Histamine	CNS	Release in hypothalamus promotes alertness
Amino acids		
GABA	CNS	Generally inhibitory
Glutamate	CNS	Generally excitatory
Neuropeptides		
Enkephalins, endorphins	CNS	Generally inhibitory; reduce pain by inhibiting substance P release
Substance P	PNS	Excitatory; pain perception
Gases		
Nitric oxide	CNS	May play a role in memory
	PNS	Vasodilation

TABLE 10.5 | Disorders Associated with Neurotransmitter Imbalances

Condition	Symptoms	Imbalance of Neurotransmitter in Brain
Alzheimer disease	Memory loss, depression, disorientation, dementia, hallucinations, death	Deficient acetylcholine
Clinical depression	Debilitating, inexplicable sadness	Deficient norepinephrine and/or serotonin
Epilepsy	Seizures, loss of consciousness	Excess GABA leads to excess norepinephrine and dopamine
Huntington disease	Cognitive and behavioral changes, loss of coordination, uncontrollable dancelike movements, death	Deficient GABA
Hypersomnia	Excessive sleeping	Excess serotonin
Insomnia	Inability to sleep	Deficient serotonin
Mania	Elation, irritability, overtalkativeness, increased movements	Excess norepinephrine
Parkinson disease	Tremors of hands, slowed movements, muscle rigidity	Deficient dopamine
Schizophrenia	Inappropriate emotional responses, hallucinations	Deficient GABA leads to excess dopamine
Tardive dyskinesia	Uncontrollable movements of facial muscles	Deficient dopamine

TABLE 10.6 | Drugs That Alter Neurotransmitter Levels

Drug	Neurotransmitter Affected*	Mechanism of Action	Effect
Tryptophan	Serotonin	Stimulates neurotransmitter synthesis	Sleepiness
Reserpine	Norepinephrine	Decreases packaging of neurotransmitter into vesicles	Decreases blood pressure
Curare	Acetylcholine	Blocks receptor binding	Muscle paralysis
Valium	GABA	Enhances receptor binding	Decreases anxiety
Nicotine	Acetylcholine	Activates receptors	Increases alertness
	Dopamine	Elevates levels	Sense of pleasure
Cocaine	Dopamine	Blocks reuptake	Euphoria
Tricyclic antidepressants	Norepinephrine	Blocks reuptake	Antidepressant
	Serotonin	Blocks reuptake	Antidepressant
Monoamine oxidase inhibitors	Norepinephrine	Blocks enzymatic degradation of neurotransmitter in presynaptic cell	Antidepressant
Selective serotonin reuptake inhibitors	Serotonin	Blocks reuptake	Antidepressant, Anti-anxiety agent
Dual reuptake inhibitors	Serotonin, norepinephrine	Blocks reuptake	Mood elevation

*Others may be affected as well.

RECONNECT
To Chapter 3, Exocytosis, page 97.

After a vesicle releases its neurotransmitter, it becomes part of the cell membrane. Endocytosis eventually returns it to the cytoplasm, where it can provide material to form new secretory vesicles. Table 10.7 summarizes this process, called vesicle trafficking.

To keep signal duration short, enzymes in synaptic clefts and on postsynaptic membranes rapidly decompose some neurotransmitters. The enzyme **acetylcholinesterase,** for example, decomposes acetylcholine on postsynaptic membranes. Other neurotransmitters are transported back into the synaptic knob of the presynaptic neuron or into nearby neurons or neuroglia, a process called *reuptake.* The enzyme **monoamine oxidase** inactivates the monoamine neurotransmitters epinephrine and norepinephrine after reuptake. This enzyme is found in mitochondria in the synaptic knob. Destruction or removal of neurotransmitter prevents continuous stimulation of the postsynaptic neuron.

Neuropeptides

Neurons in the brain or spinal cord synthesize **neuropeptides.** These peptides act as neurotransmitters or as *neuromodulators*—substances that alter a neuron's response to a neurotransmitter or block the release of a neurotransmitter.

Among the neuropeptides are the *enkephalins,* present throughout the brain and spinal cord. Each enkephalin molecule is a chain of five amino acids. Synthesis of enkephalins increases during periods of painful stress, and they bind to the same receptors in the brain (opiate receptors) as the narcotic morphine. Enkephalins relieve pain sensations and probably have other functions. Another morphinelike peptide, *beta endorphin,* is found in the brain and cerebrospinal fluid. It acts longer than enkephalins and is a much more potent pain reliever (Clinical Application 10.4).

Substance P is a neuropeptide that consists of eleven amino acids and is widely distributed. It functions as a neurotransmitter (or perhaps as a neuromodulator) in the neurons that transmit pain impulses into the spinal cord and on to the brain. Enkephalins and endorphins may relieve pain by inhibiting the release of substance P from pain-transmitting neurons.

TABLE 10.7 | Events Leading to Neurotransmitter Release

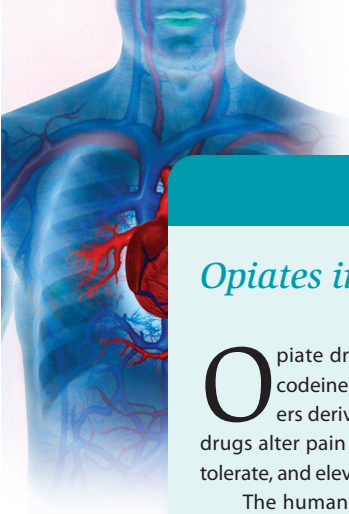
1. Action potential passes along an axon and over the surface of its synaptic knob.
2. Synaptic knob membrane becomes more permeable to calcium ions, and they diffuse inward.
3. In the presence of calcium ions, synaptic vesicles fuse to synaptic knob membrane.
4. Synaptic vesicles release their neurotransmitter by exocytosis into the synaptic cleft.
5. Synaptic vesicles become part of the membrane.
6. The added membrane provides material for endocytotic vesicles.

10.8 IMPULSE PROCESSING

The way the nervous system processes and affects nerve impulses reflects, in part, the organization of neurons and axons in the brain and spinal cord.

Neuronal Pools

Interneurons, the neurons completely in the CNS, are organized into **neuronal pools.** These are groups of neurons that



10.4 CLINICAL APPLICATION

Opiates in the Human Body

Opiate drugs, such as morphine, heroin, codeine, and opium, are potent painkillers derived from the poppy plant. These drugs alter pain perception, making it easier to tolerate, and elevate mood.

The human body produces opiates, called endorphins (for "endogenous morphine"), that are peptides. Like the poppy-derived opiates that they structurally resemble, endorphins influence mood and perception of pain.

The discovery of endorphins began in 1971 in research laboratories at Stanford University and the Johns Hopkins School of Medicine, where researchers exposed pieces of brain tissue from experimental mammals to morphine. The morphine was radioactively labeled (some of the atoms were radioactive isotopes) so researchers could follow its destination in the brain.

The morphine bound receptors on neurons that transmit pain. Why, the investigators wondered, would an animal's brain cells have receptors for a plant chemical? One explanation was that a mammal's body could manufacture opiates. The opiate receptors, then, would normally bind the body's opiates (the endorphins) but would also bind the chemically similar compounds from poppies. Researchers have since identified several types of endorphins in the human brain and associated their release with situations involving pain relief, such as acupuncture and analgesia to mother and child during childbirth. Endorphin release is also associated with "runner's high." PET scans reveal endorphins binding opiate receptors after conditioned athletes run for two hours.

Endorphins explain why some people addicted to opiate drugs such as heroin experience withdrawal pain when they stop taking the drug. Initially, the body interprets the frequent binding of heroin to its endorphin receptors as an excess of endorphins. To bring the level down, the body slows its own production of endorphins. Then, when the person stops taking the heroin, the body becomes short of opiates (heroin and endorphins). The result is pain.

Opiate drugs can be powerfully addicting when abused—that is, taken repeatedly by a person who is not in pain. These same drugs, however, are extremely useful in dulling severe pain, particularly in terminal illnesses. ■

synapse with each other and perform a common function, even though their cell bodies may be in different parts of the CNS. Each neuronal pool receives input from neurons (which may be part of other pools), and each pool generates output. Neuronal pools may have excitatory or inhibitory effects on other pools or on peripheral effectors.

As a result of incoming impulses and neurotransmitter release, a particular neuron of a neuronal pool may be excited by some presynaptic neurons and inhibited by others. If the net effect is excitatory, threshold may be reached, and an outgoing impulse triggered. If the net effect is excitatory but subthreshold, an impulse will not be triggered, but because the neuron is close to threshold, it will be much more responsive to any further excitatory stimulation. This condition is called **facilitation** (fah-sil″ĭ-tă′shun).

Convergence

Any single neuron in a neuronal pool may receive impulses from two or more other neurons. Axons originating from different parts of the nervous system leading to the same neuron exhibit **convergence** (kon-ver′jens).

Incoming impulses often represent information from various sensory receptors that detect changes. Convergence allows the nervous system to collect, process, and respond to information.

Convergence makes it possible for a neuron to sum impulses from different sources. For example, if a neuron receives subthreshold stimulation from one input neuron, it may reach threshold if it receives additional stimulation from a second input neuron. Thus, an output impulse triggered from this neuron reflects summation of impulses from two sources (fig. 10.21*a*). Such an output impulse may travel to a particular effector and evoke a response.

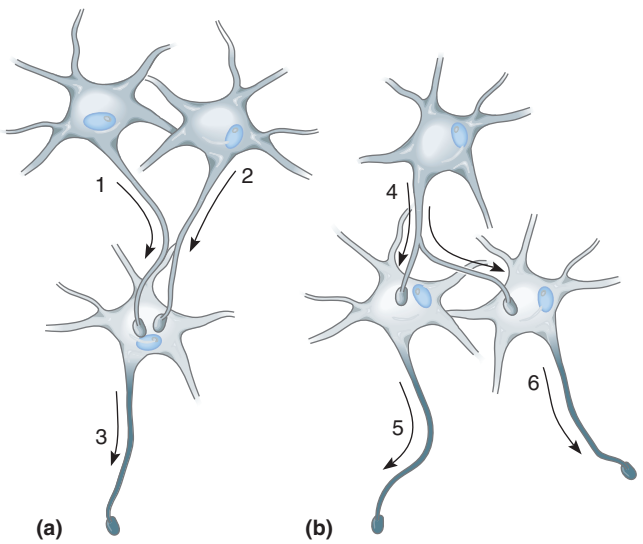


FIGURE 10.21 Impulse processing in neuronal pools. (*a*) Axons of neurons 1 and 2 converge to the cell body of neuron 3. (*b*) The axon of neuron 4 diverges to the cell bodies of neurons 5 and 6.

Divergence

A neuron has a single axon, but axons may branch at several points. Thus, impulses leaving a neuron of a neuronal pool may exhibit **divergence** (di-ver′jens) by reaching several other neurons. For example, one neuron may stimulate two others; each of these, in turn, may stimulate several others, and so forth. Such a pattern of diverging axons can amplify an impulse—that is, spread it to increasing numbers of neurons within the pool (fig. 10.21*b*).

As a result of divergence, an impulse originating from a single neuron in the CNS may be amplified so that sufficient impulses reach the motor units in a skeletal muscle to cause forceful contraction. Similarly, an impulse originating from a sensory receptor may diverge and reach several different regions of the CNS, where the resulting impulses can be processed and acted upon.

The nervous system enables us to experience the world and to think and feel emotion. This organ system is also sensitive to outside influences. Clinical Application 10.5 discusses one way that an outside influence can affect the nervous system—drug addiction.

PRACTICE

18 Define *neuropeptide*.

19 What is a neuronal pool?

20 Define *facilitation*.

21 What is convergence?

22 What is the relationship between divergence and amplification?

CHAPTER SUMMARY

10.1 INTRODUCTION (PAGE 354)

1. The nervous system is a network of cells that sense and respond to stimuli in ways that maintain homeostasis.
2. The nervous system is composed of neural tissue, including neurons and neuroglia, blood vessels and connective tissue.
3. Neurons have processes that receive (dendrites) and send (axons) bioelectric signals (neurotransmitters) that cross spaces (synapses) between them.
4. Organs of the nervous system are divided into the central and peripheral nervous systems.

10.2 GENERAL FUNCTIONS OF THE NERVOUS SYSTEM (PAGE 355)

1. Sensory receptors detect changes in internal and external body conditions.
2. Integrative functions gather sensory information and make decisions that affect motor functions.
3. Motor impulses stimulate effectors to respond.
   a. The motor portion of the PNS that carries out voluntary activities is the somatic nervous system.
   b. The motor portion of the PNS that carries out involuntary activities is the autonomic nervous system.

10.3 DESCRIPTION OF CELLS OF THE NERVOUS SYSTEM (PAGE 356)

1. Neurons vary in size, shape, sizes and lengths of axons and dendrites, and number of dendrites.
2. A neuron includes a cell body, cell processes, and the organelles usually found in cells.
3. Neurofibrils support axons.
4. Chromatophilic substance is mostly rough ER and is scattered throughout the cytoplasm of neurons.
5. Dendrites and the cell body provide receptive surfaces.
6. A single axon arises from the cell body and may be enclosed in a myelin sheath and a neurilemma.
7. White matter consists of myelinated axons, and gray matter consists of unmyelinated axons and cell bodies.

10.4 CLASSIFICATION OF CELLS OF THE NERVOUS SYSTEM (PAGE 359)

1. Classification of neurons
   a. Neurons are structurally classified as multipolar, bipolar, or unipolar.
   b. Neurons are functionally classified as sensory neurons, interneurons, or motor neurons.
2. Classification of neuroglia
   a. Neuroglia are abundant and have several functions.
   b. They fill spaces, support neurons, hold nervous tissue together, help metabolize glucose, help regulate potassium ion concentration, produce myelin, carry on phagocytosis, rid synapses of excess ions and neurotransmitters, nourish neurons, and stimulate synapse formation.
   c. They include astrocytes, oligodendrocytes, microglia, and ependymal cells in the CNS and Schwann cells and satellite cells in the PNS.
   d. Malfunctioning neuroglia can cause disease.
   e. Neuroglia are involved in axonal regeneration.
      (1) If a neuron cell body is injured, the neuron is likely to die; neural stem cells may proliferate and produce replacements.

10.5 CLINICAL APPLICATION

Drug Addiction

Drug abuse and addiction are long-standing problems. A 3,500-year-old Egyptian document decries reliance on opium. In the 1600s, a smokable form of opium enslaved many Chinese, and the Japanese and Europeans discovered the addictive nature of nicotine. During the American Civil War, morphine was a widely used painkiller; cocaine was introduced a short time later to relieve veterans addicted to morphine. Today, abuse of drugs intended for medical use continues. LSD was originally used in psychotherapy but was abused in the 1960s as a hallucinogen. PCP was an anesthetic before being abused in the 1980s.

Why do certain drugs compel a person to repeatedly use them, even when knowing that doing so is dangerous? Eating hot fudge sundaes is highly enjoyable, but we usually don't feel driven to consume them repeatedly. The biology of neurotransmission helps to explain drug addiction.

When a drug alters the activity of a neurotransmitter on a postsynaptic neuron, it either halts or enhances synaptic transmission. A drug that binds to a receptor, blocking a neurotransmitter from binding, is called an *antagonist*. A drug that activates the receptor, triggering an action potential, or that helps a neurotransmitter to bind, is called an *agonist*. The effect of a drug depends upon whether it is an antagonist or an agonist; on the particular behaviors the affected neurotransmitter normally regulates; and in which parts of the brain drugs affect neurotransmitters and their binding to receptors. Many addictive substances bind to receptors for the neurotransmitter dopamine, in a brain region called the nucleus accumbens.

With repeated use of an addictive substance, the number of receptors it targets can decline. When this happens, the person must use more of the drug to feel the same effect. For example, neural pathways that use the neurotransmitter norepinephrine control arousal, dreaming, and mood. Amphetamine enhances norepinephrine activity, thereby heightening alertness and mood. Amphetamine's structure is so similar to that of norepinephrine that it binds to norepinephrine receptors and triggers the same changes in the postsynaptic membrane.

Cocaine has a complex mechanism of action, both blocking reuptake of norepinephrine and binding to molecules that transport dopamine to postsynaptic cells. The drug valium causes relaxation and inhibits seizures and anxiety by helping GABA, an inhibitory neurotransmitter used in a third of the brain's synapses, bind to receptors on postsynaptic neurons. Valium is therefore a GABA agonist.

Nicotine causes addiction, which supplies enough of the other chemicals in cigarette smoke to destroy health. An activated form of nicotine binds postsynaptic nicotinic receptors that normally receive acetylcholine. When sufficient nicotine binds, a receptor channel opens, allowing positive ions in (fig. 10A). When a certain number of positive ions enter, the neuron releases dopamine from its other end, which provides the pleasurable feelings associated with smoking.

When a smoker increases the number of cigarettes smoked, the number of nicotinic receptors increases. This happens because of the way that the nicotine binding impairs the recycling of receptor proteins, so receptors are produced faster than they are taken apart. After a period of steady nicotine exposure, many of the receptors malfunction and no longer admit the positive ions that trigger the nerve impulse. This may be why as time goes on it takes more nicotine to produce the same effects—a hallmark of addiction. ■

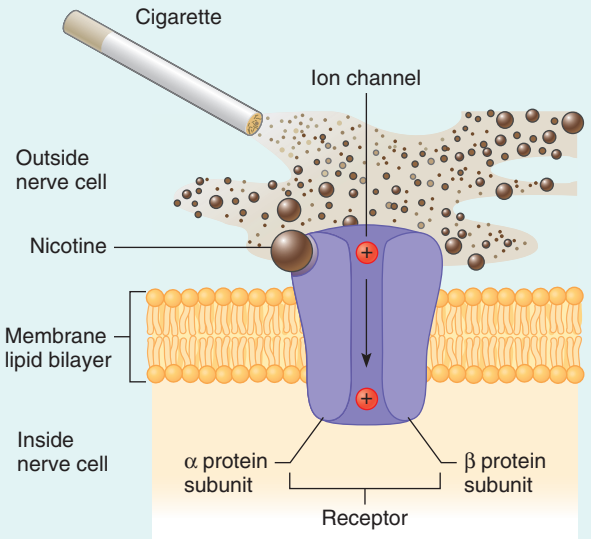
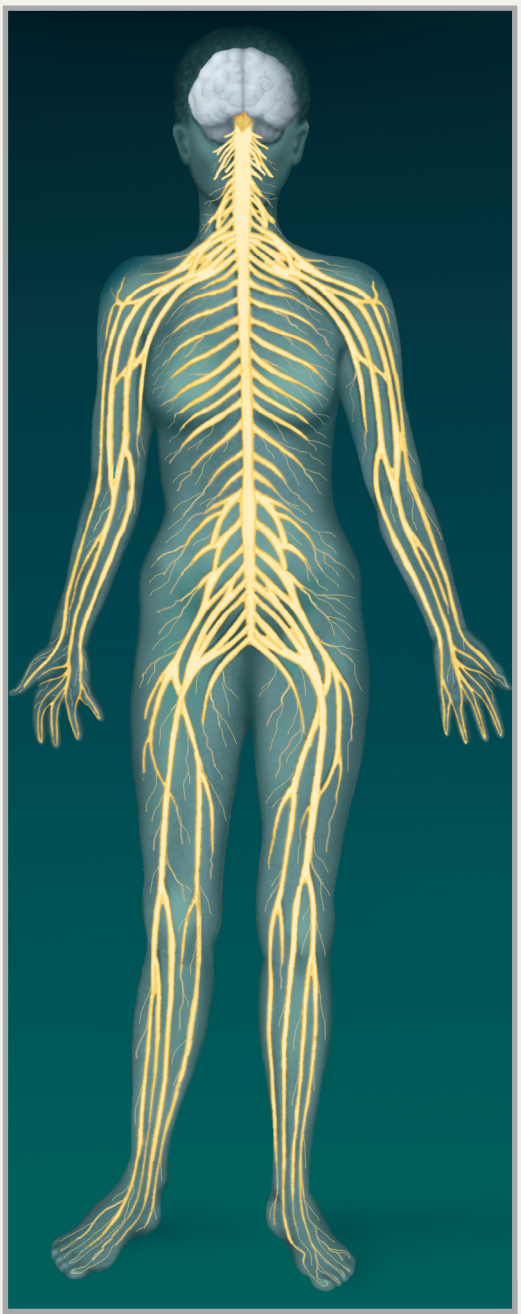


FIGURE 10A Nicotine binds and transiently alters postsynaptic receptors that normally bind the neurotransmitter acetylcholine. As a result, positive ions enter the cell, triggering dopamine release. With frequent smoking, receptors accumulate and soon become nonfunctional. Nicotine's effects on the nervous system are complex.

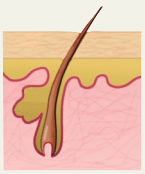
INNERCONNECTIONS | *Nervous System*



Nervous system

Nerves carry impulses that allow body systems to communicate.

Integumentary System

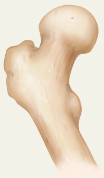


Sensory receptors provide the nervous system with information about the outside world.

Lymphatic System

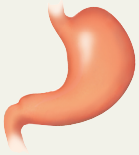
Stress may impair the immune response.

Skeletal System



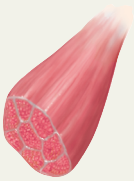
Bones protect the brain and spinal cord and help maintain plasma calcium, important to neuron function.

Digestive System



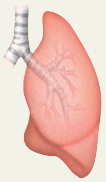
The nervous system can influence digestive function.

Muscular System



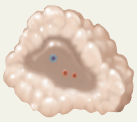
Nerve impulses control movement and carry information about the position of body parts.

Respiratory System



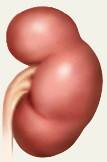
The nervous system alters respiratory activity to control oxygen levels and blood pH.

Endocrine System



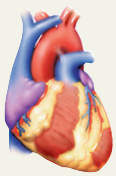
The hypothalamus controls secretion of many hormones.

Urinary System



Nerve impulses affect urine production and elimination.

Cardiovascular System



Nerve impulses help control blood flow and blood pressure.

Reproductive System

The nervous system plays a role in egg and sperm formation, sexual pleasure, childbirth, and nursing.

(2) If a peripheral axon is severed, its distal portion will die, but under the influence of nerve growth factors, the proximal portion may regenerate and reestablish connections, if a tube of connective tissue guides it.
(3) Significant regeneration is not likely in the CNS.

10.5 THE SYNAPSE (PAGE 365)

A synapse is a junction between two cells. A synaptic cleft is the gap between parts of two cells at a synapse. Synaptic transmission is the process by which the impulse in the presynaptic neuron signals the postsynaptic cell.

1. A nerve impulse travels along the axon to a synapse.
2. Axons have synaptic knobs at their distal ends that secrete neurotransmitters.
3. The neurotransmitter is released when a nerve impulse reaches the end of an axon, and the neurotransmitter diffuses across the synaptic cleft.
4. A neurotransmitter reaching a postsynaptic neuron or other cell may be excitatory or inhibitory.

10.6 CELL MEMBRANE POTENTIAL (PAGE 365)

A cell membrane is usually polarized as a result of an unequal distribution of ions on either side. Channels in membranes that allow passage of some ions but not others control ion distribution.

1. Distribution of ions
   a. Membrane ion channels, formed by proteins, may be always open or sometimes open and sometimes closed.
   b. Potassium ions pass more readily through resting neuron cell membranes than do sodium and calcium ions.
   c. A high concentration of sodium ions is on the outside of the membrane, and a high concentration of potassium ions is on the inside.
2. Resting potential
   a. Large numbers of negatively charged ions, which cannot diffuse through the cell membrane, are inside the cell.
   b. In a resting cell, more positive ions leave the cell than enter it, so the inside of the cell membrane develops a negative charge with respect to the outside.
3. Local potential changes
   a. Stimulation of a membrane affects its resting potential in a local region.
   b. The membrane is depolarized if it becomes less negative; it is hyperpolarized if it becomes more negative.
   c. Local potential changes are graded and subject to summation.
   d. Reaching threshold potential triggers an action potential.
4. Action potentials
   a. At threshold, sodium channels open and sodium ions diffuse inward, depolarizing the membrane.
   b. Slightly later, potassium channels open and potassium ions diffuse outward, repolarizing the membrane.
   c. This rapid change in potential is an action potential.
   d. Many action potentials can occur before active transport reestablishes the original resting potential.
   e. The propagation of action potentials along a nerve fiber is an impulse.
5. All-or-none response
   a. A nerve impulse is an all-or-none response. If a stimulus of threshold intensity is not applied to an axon, an action potential is not generated.
   b. All the impulses conducted on an axon are the same.
6. Refractory period
   a. The refractory period is a brief time following passage of a nerve impulse when the membrane is unresponsive to an ordinary stimulus.
   b. During the absolute refractory period, the membrane cannot be stimulated; during the relative refractory period, the membrane can be stimulated with a high-intensity stimulus.
7. Impulse conduction
   a. An unmyelinated axon conducts impulses that travel over its entire surface.
   b. A myelinated axon conducts impulses that travel from node to node.
   c. Impulse conduction is more rapid on myelinated axons with large diameters.

10.7 SYNAPTIC TRANSMISSION (PAGE 371)

Neurotransmitter molecules diffuse across the synaptic cleft and react with receptors in the postsynaptic neuron membrane.

1. Synaptic potentials
   a. Some neurotransmitters can depolarize the postsynaptic membrane, possibly triggering an action potential. This is an excitatory postsynaptic potential (EPSP).
   b. Others hyperpolarize the membrane, inhibiting an action potential. This is an inhibitory postsynaptic potential (IPSP).
   c. EPSPs and IPSPs are summed in a trigger zone of the neuron.
2. Neurotransmitters
   a. The nervous system produces at least thirty types of neurotransmitters.
   b. Calcium ions diffuse into synaptic knobs in response to action potentials, releasing neurotransmitters.
   c. Neurotransmitters are quickly decomposed or removed from synaptic clefts.
3. Neuropeptides
   a. Neuropeptides are chains of amino acids.
   b. Some neuropeptides are neurotransmitters or neuromodulators.
   c. They include enkephalins, endorphins, and substance P.

10.8 IMPULSE PROCESSING (PAGE 374)

The way impulses are processed reflects the organization of neurons in the brain and spinal cord.

1. Neuronal pools
   a. Neurons are organized into pools in the CNS.
   b. Each pool receives, processes, and may conduct impulses away.
   c. Each neuron in a pool may receive excitatory and inhibitory stimuli.
   d. A neuron is facilitated when it receives subthreshold stimuli and becomes more excitable.
2. Convergence
   a. Impulses from two or more axons may converge on a single postsynaptic neuron.
   b. Convergence enables a neuron to sum impulses from different sources.
3. Divergence
   a. Impulses from a presynaptic neuron may reach several postsynaptic neurons.
   b. Divergence amplifies impulses.

CHAPTER ASSESSMENTS

10.1 Introduction

1 Describe how the nervous system detects change associated with the body and reacts to that change to maintain homeostasis. (p. 354)

2 Distinguish between neurons and neuroglia. (p. 354)

3 Which of the following descriptions is accurate? (p. 354)

a. A neuron has a single dendrite, which sends information.
b. A neuron has a single axon, which sends information.
c. A neuron has many axons, which receive information.
d. A neuron has many dendrites, which send information.

4 Explain the difference between the central nervous system (CNS) and the peripheral nervous system (PNS). (p. 354)

10.2 General Functions of the Nervous System

5 List three general functions of the nervous system. (p. 355)

6 Distinguish a sensory receptor from an effector. (p. 355)

7 Distinguish between the types of activities that the somatic and autonomic nervous systems control. (p. 356)

10.3 Description of Cells of the Nervous System

8 Match the part of a neuron on the left with the description on the right (p. 356):

(1) dendrites	A. fine threads in an axon
(2) chromatophilic substance	B. part of neuron from which axon and dendrites extend
(3) axon	C. highly branched, multiple processes that may have spines
(4) cell body	D. sends nerve impulses
(5) neurofibrils	E. rough endoplasmic reticulum

9 Explain how Schwann cells encase large axons including the formation of myelin, the neurilemma, and the nodes of Ranvier. (p. 358)

10 What do Schwann cells and oligodendrocytes have in common, and how do they differ? (p. 358)

11 Distinguish between myelinated and unmyelinated axons. (p. 358)

10.4 Classification of Cells of the Nervous System

12 Describe the three types of neurons classified on the basis of structure. (p. 360)

13 Describe the three types of neurons classified on the basis of function (p. 360)

14 List six functions of neuroglia. (p. 361)

15 Describe the neuroglia of the CNS. (p. 361)

16 Explain how malfunctioning neuroglia can harm health. (p. 363)

17 Describe the neuroglia of the PNS. (p. 363)

18 Explain how an injured neuron may regenerate. (p. 364)

10.5 The Synapse

19 The _____ _____ brings the impulse to the synapse, whereas the _____ _____ on the other side of the synapse is stimulated or inhibited as a result of the synaptic transmission. (p. 365)

20 Explain how information is passed from a presynaptic neuron to a postsynaptic cell. (p. 365)

21 Diffusion of which of the following ions into the synaptic knob triggers the release of neurotransmitter? (p. 365)

a. Na^{+}
b. Ca^{+2}
c. Cl^{-}
d. K^{+}

10.6 Cell Membrane Potential

22 Define *resting potential.* (p. 366)

23 Distinguish among polarized, hyperpolarized, and depolarized. (p. 368)

24 Explain why the "trigger zone" of a neuron is named as such. (p. 368)

25 List in correct order the changes that occur during an action potential. (p. 368)

26 Explain the relationship between an action potential and a nerve impulse. (p. 369)

27 Define *refractory period.* (p. 370)

28 Explain the importance of the nodes of Ranvier and conduction in myelinated fibers as opposed to conduction in unmyelinated fibers. (p. 371)

10.7 Synaptic Transmission

29 Distinguish between excitatory and inhibitory postsynaptic potentials. (p. 371)

30 Explain how enzymes within synaptic clefts and reuptake of neurotransmitter prevents continuous stimulation of the postsynaptic cell. (p. 374):

10.8 Impulse Processing

31 Explain what determines the output of a neuronal pool in terms of input neurons, excitation, and inhibition. (p. 374)

32 Define *facilitation.* (p. 375)

33 Distinguish between convergence and divergence. (p. 375)

INTEGRATIVE ASSESSMENTS/CRITICAL THINKING

OUTCOMES 10.3, 10.4

1. Why are rapidly growing cancers that originate in nervous tissue more likely to be composed of neuroglia than of neurons?

OUTCOMES 10.3, 10.4, 10.6

2. In Tay-Sachs disease, an infant rapidly loses nervous system functions as neurons in the brain become covered in too much myelin. In multiple sclerosis, cells in the CNS have too little myelin. Identify the type of neuroglia implicated in each of these conditions.

OUTCOMES 10.4, 10.5, 10.7

3. How would you explain the following observations?
   a. When motor nerve fibers in the leg are severed, the muscles they innervate become paralyzed; however, in time, control over the muscles often returns.
   b. When motor nerve fibers in the spinal cord are severed, the muscles they control become permanently paralyzed.

OUTCOMES 10.5, 10.6, 10.7

4. Drugs that improve early symptoms of Alzheimer disease do so by slowing the breakdown of acetylcholine in synaptic clefts in certain parts of the brain. From this information, suggest a neurotransmitter imbalance that lies behind Alzheimer disease.

OUTCOME 10.6

5. What might be deficient in the diet of a pregnant woman complaining of leg muscle cramping? How would you explain this to her?

OUTCOME 10.6

6. People who inherit familial periodic paralysis often develop very low blood potassium concentrations. How would you explain that the paralysis may disappear quickly when potassium ions are administered intravenously?

WEB CONNECTIONS

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