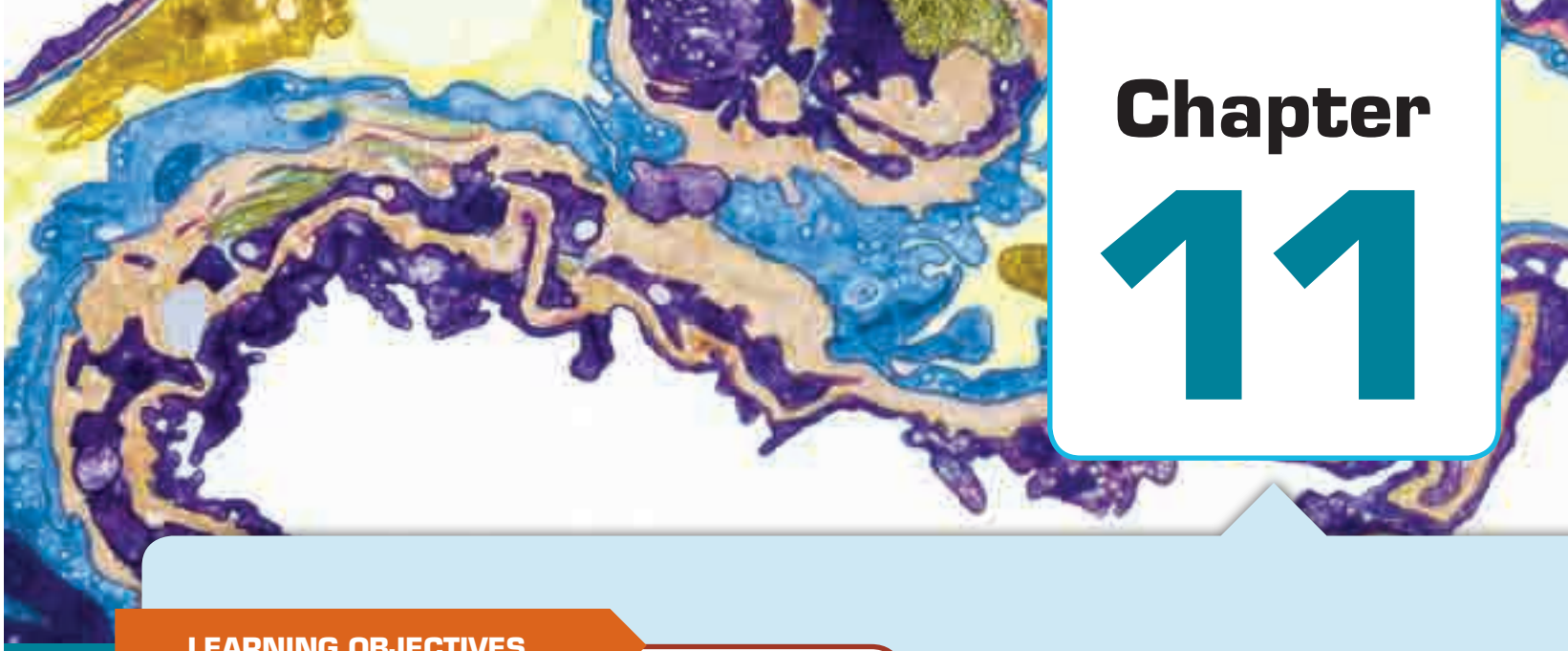


Chapter

11

Neural Function

LEARNING OBJECTIVES

- Discuss normal neural anatomy and physiology.
- Describe and compare congenital neurologic disorders.
- Compare and contrast traumatic neurologic disorders.
- Compare and contrast infectious neurologic disorders.
- Describe and compare vascular neurologic disorders.
- Compare and contrast types of seizure disorders.
- Compare and contrast chronic degenerative neurologic disorders.
- Compare and contrast types of dementia.
- Describe and compare cancers of the nervous system.

KEY TERMS

action potential
afferent nerve
afferent tracts
AIDS dementia complex
Alzheimer's disease (AD)
amyotrophic lateral sclerosis (ALS)
arachnoid layer
ascending fibers
aura
automatism
autonomic hyperreflexia
autonomic nervous system
autoregulation
axon
basal ganglia
basilar skull fracture
brain
brain stem
cauda equina
cauda equina syndrome
central nervous system (CNS)
cerebellum
cerebral aneurysm
cerebral contusion
cerebral palsy (CP)
cerebral vascular accident (CVA)
cerebrospinal fluid (CSF)
cerebrum
chorea
comminuted skull fracture
communicating cerebrospinal fluid flow
compound skull fracture
concussion
countercoup
coup
cranial nerves
Creutzfeldt-Jakob disease (CJD)
Cushing's reflex
Cushing's triad
dementia
dendrite
depolarization
depressed skull fracture
dermatome
descending fibers
diencephalon
dorsal root
dura mater
efferent nerve
efferent tracts
encephalitis
epidural hematoma
epilepsy
epithalamus
flexor reflex
focal seizure
foramen magnum
frontal lobe
generalized seizure
gyrus
hematoma
hemorrhagic stroke
herniation
Huntington's disease
hydrocephalus
hypothalamus
increased intracranial pressure
interneuron
intracerebral hematoma
ischemic stroke
linear skull fracture
lobe
longitudinal fissure
medulla
meninges
meningitis
meningocele
midbrain
Monro-Kellie hypothesis
motor nerve
multiple sclerosis (MS)
myasthenia gravis
myasthenic crisis
myelin sheath
myelomeningocele
nerve
neuroglia
neuromelanin
neuron
neurotransmitter
node of Ranvier
noncommunicating cerebrospinal fluid flow
obstructive cerebrospinal fluid flow
occipital lobe
parasympathetic nervous system
parietal lobe
Parkinson's disease
peripheral nerve
peripheral nervous system (PNS)
pia mater
plexus
pons
postictal period
postsynaptic cell membrane
presynaptic terminal
prion
resting potential
reticular activation system
reticular formation
rootlet
Schwann cell
seizure
sensory nerve
spina bifida
spina bifida occulta
spinal cord
spinal cord injury (SCI)
spinal reflex arc
spinal shock
status epilepticus
subarachnoid hemorrhage
subdural hematoma
substantia nigra
subthalamus
sulcus
sympathetic nervous system (SNS)
synapse
synaptic cleft
temporal lobe
terminal bouton
thalamus
transient ischemic attack (TIA)
traumatic brain injury (TBI)
ventral root
ventricle
vertebral canal
white matter

The nervous system consists of complex structures that control many body functions and cognition. The functions this system manages include (1) structures such as muscles, glands, and organs; (2) heartbeat; (3) blood flow; (4) breathing; (5) digestion; (6) urination; and (7) defecation. The nervous system works with other systems to maintain homeostasis by receiving and responding to input from the environment. Disorders of the nervous system may be acute or chronic, but regardless, these conditions often have grave or life-altering effects on the body. Causes of these disorders include congenital defects, trauma, infections, tumors, chemical imbalances, and vascular changes.

Anatomy and Physiology

The nervous system is an intricate network of specialized cells and tissue that receive and react to environmental stimuli on a physiologic and cognitive level. To communicate this input, these structures conduct electric impulses between the brain and the rest of the body. The nervous system consists of three main components—brain, spinal cord, and nerves. The brain and spinal cord make up the **central nervous system (CNS)**, and the nerves make up the **peripheral nervous system (PNS)**.

Central Nervous System

The skull and vertebral column house and protect the brain and spinal cord. Additionally, a set of three tough membranes, called **meninges**, encase the CNS (**Figure 11-1**). The **dura mater** is the outer and toughest layer. The **arachnoid layer** is the middle layer, named for its spider web–like vascular system. The **pia mater** is the innermost layer that rests directly on the brain and spinal cord. **Cerebrospinal fluid (CSF)** is a plasmalike liquid that fills the space between the arachnoid and the pia mater layers to provide additional cushion and support to the CNS. The choroid plexus cells in the brain's ventricles continuously produce the CSF. The **ventricles** are interconnected, hollow areas of the brain where CSF fills and flows freely between them. Excess CSF drains into the bloodstream.

The **brain** is located within the skull and contains billions of neurons. Neural tissue contains two basic types of cells—neuroglia and neurons. **Neuroglia** cells

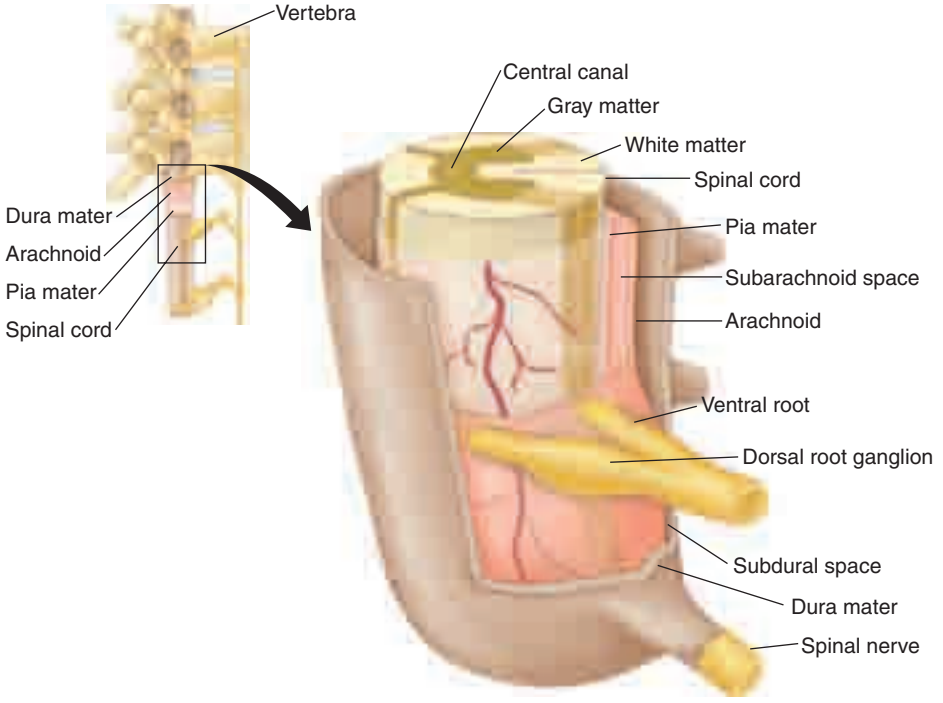


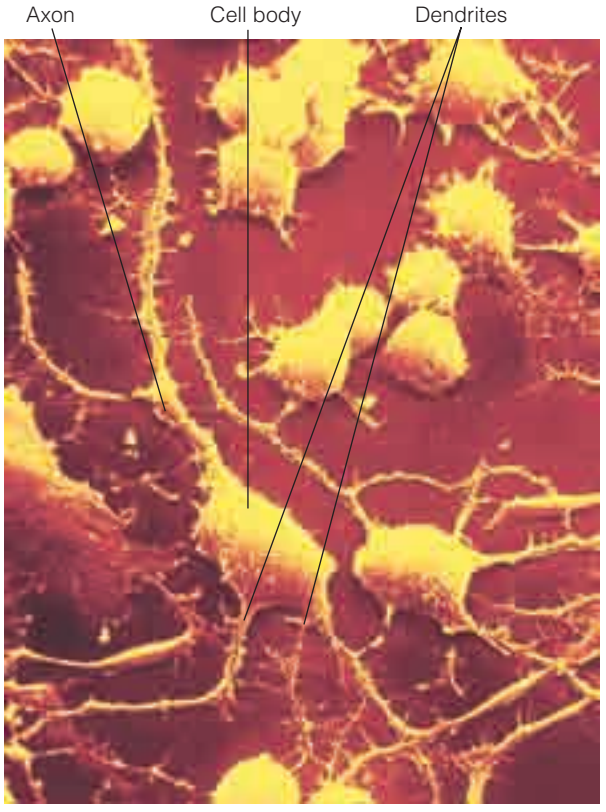
Figure 11-1

The meninges enclose the brain and the spinal cord.

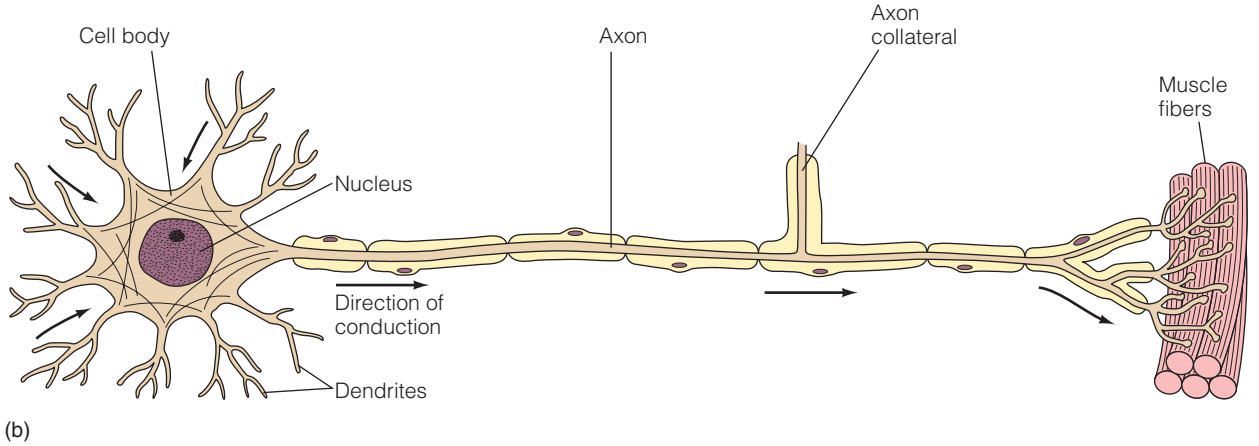
provide several important supportive roles in the nervous system. Neuroglia cells scaffold neural tissue as well as isolate and protect neuron cell membranes. Additionally, neuroglia cells regulate interstitial fluid, defend the neuron against pathogens, and assist with neural repair.

Neurons are the fundamental unit of the nervous system that generate bioelectrical impulses and transmit them from one area of the body to another. Neurons occur in several sizes and shapes, but all neurons share similar characteristics. Neurons do not have the ability to divide; therefore, when neurons are lost due to aging or injury, they cannot be replaced. Not all cell death results in loss of functioning. For example, if neurons are damaged in one area of the brain, neurons in other areas can eventually assume those functions. In the PNS, severed nerves can regenerate to a point to reestablish connections with the tissue it once supplied. In the brain or spinal cord, severed axons cannot be repaired. Severed spinal cord nerves result in paralysis and loss of sensation below the area of damage. In addition to being unable to divide, nerve cells require a constant supply of oxygen and glucose. This characteristic makes neurons vulnerable to the effects of hypoxia and hypoglycemia. Neurons can begin dying within minutes of these events.

Most neurons have a spherical cell body that houses the nucleus, most of the cytoplasm, and organelles. Neurons contain projections called **axons** and **dendrites** that make connections with nearby cells (**Figure 11-2**). Axons transmit impulses away from the cell body, and dendrites transmit impulses toward the



(a)



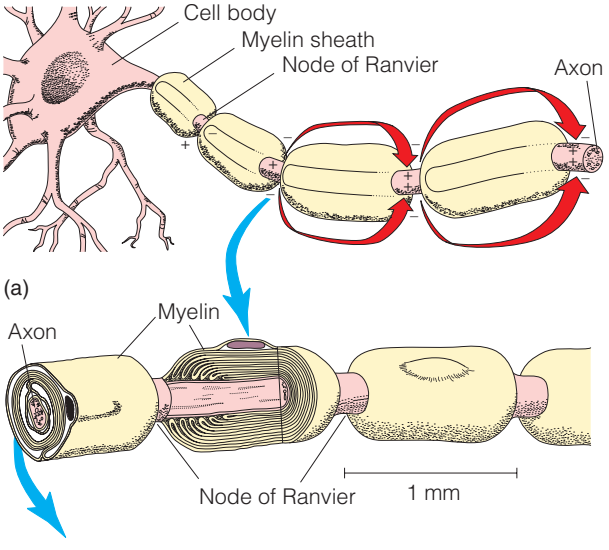
(b)

Figure 11-2

A neuron. (a) A scanning electron micrograph of the cell body and dendrites. (b) Collateral branches may occur along the length of the axon. In motor neurons, when the axon terminates, it branches many times, ending in individual muscle fibers.

cell body. When the axon reaches its destination, it often branches into several small fibers that terminate into miniscule bulges, called **terminal boutons**. These terminal boutons communicate with neurons, muscle fibers, or glands. Axons may be surrounded by a **myelin sheath** that increases the rate of impulse transmission approximately 400 times faster than unmyelinated nerves (**Figure 11-3**). **Schwann cells** produce the myelin sheath, and these Schwann cells are separated by **nodes of Ranvier**. Because of the myelin, the impulses move at greater speeds down the axon, jumping from one node to the next (like stones skipping across water). Bundles of these myelinated nerves are referred to as **white matter**. Impulses move in a slow, wavelike pattern in unmyelinated nerves. A **synapse** refers to the gap between the neurons. This gap includes the **presynaptic terminal** (e.g., terminal bouton or some similar structure), the **synaptic cleft** (space between neurons), and a **postsynaptic cell membrane** (**Figure 11-4**). The presynaptic and postsynaptic terminals are opposite ends of the nerve.

Electrical impulses of the nervous system are not like the electrical current that powers appliances, which is formed by the flow of electrons. Small ionic changes (e.g., potassium and sodium moving across cell membranes) generate neural impulses. Creating this charge is referred to as **action potential** (**Figure 11-5**). The plasma side of the neuron membrane has a slight charge at rest, or **resting potential**, because of the sodium ions concentrated on the outside of the cell. When the neuron is stimulated, protein gates open and sodium flows into the cell. The rapid inflow of positively charged sodium ions increases the charge (this is called **depolarization**). Immediately following depolarization, the cell membrane returns to its resting state by the rapid outflow of the positively charged potassium ions. When generated, these impulses travel down the nerve to trigger the release of **neurotransmitters** from the presynaptic terminal. The neurotransmitters cross the synaptic cleft, only in one direction, to stimulate an electrical reaction in nearby neurons. Synaptic transmission of the impulse takes a mere millisecond. This electrical reaction passes through those neurons to the next synapse, where the process repeats. At each synaptic transmission, a small burst of neurotransmitters is released and then removed. Neurotransmitters are either destroyed by enzymes or reabsorbed by the postsynaptic membrane to be recycled for the next transmission. In addition to stimulating the action potential of neurons, some neurotransmitters inhibit action potential.



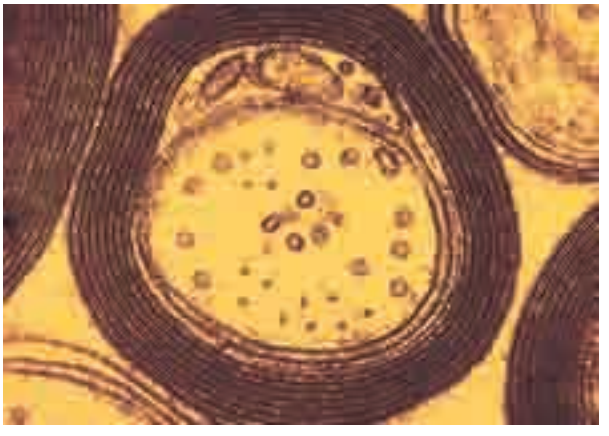
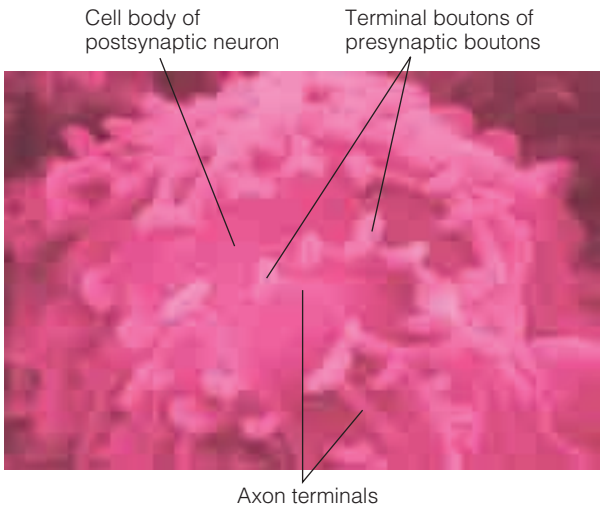

(c)

Figure 11-3

A myelinated nerve. (a) The myelin sheath allows impulses to "jump" from node to node, greatly accelerating the rate of transmission. (b) The node of Ranvier. (c) A transmission electron micrograph of an axon in the cross section, showing a myelin sheath.



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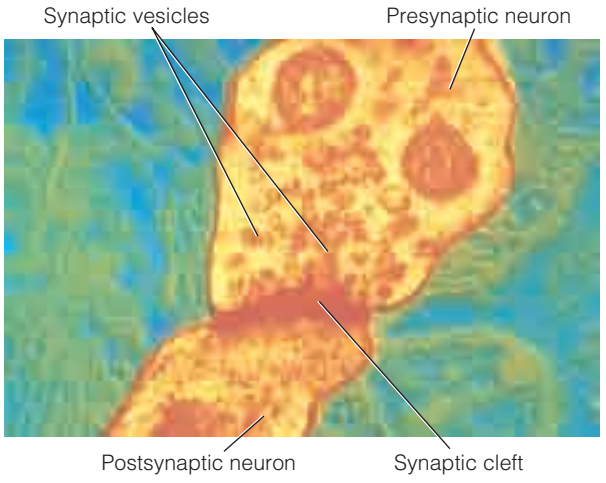

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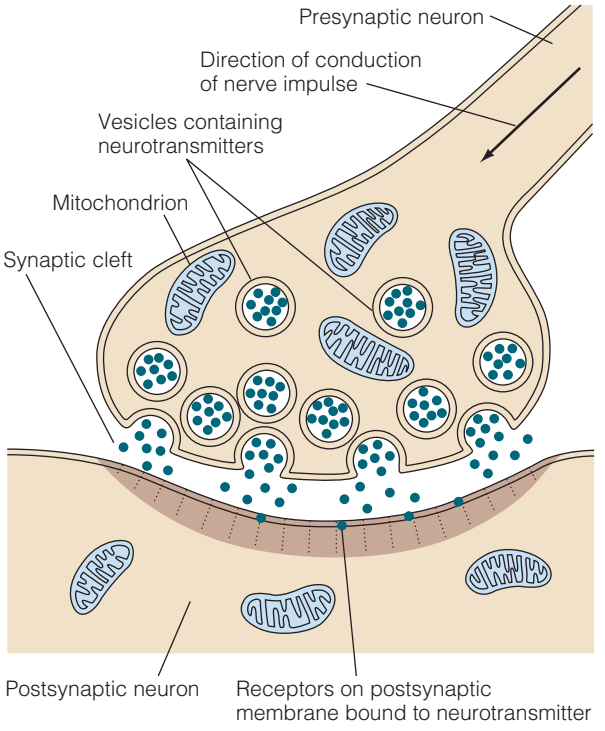


(c)

Figure 11-4

The function of neurotransmitters in the synaptic cleft. (a) A scanning electron micrograph showing the terminal boutons of an axon ending on the cell body of another neuron. (b) A transmission electron micrograph showing the details of the synapse. (c) The arrival of the impulse stimulates the release of neurotransmitters held in synaptic vesicles in the axon terminals. Neurotransmitter diffuses across the synaptic cleft and binds to the postsynaptic membrane, where it elicits another action potential that travels down the dendrite to the cell body.

The brain is responsible for a variety of physiologically vital functions and cognitive activities. The brain accomplishes these functions in part through a set of cranial nerves. Twelve pairs of **cranial nerves** branch directly from the base of the brain (**Figure 11-6**). Some of the cranial nerves only carry sensory fibers (I, II, and VIII), others only carry motor fibers (III, IV, VI, XI, and XII), and few carry both (V, VII, IX, and X). Each nerve travels from the brain through the foramen to its destination.

The major regions of the brain include the cerebrum (including cerebral cortex), diencephalon (thalamus and hypothalamus), brain stem (pons, midbrain,

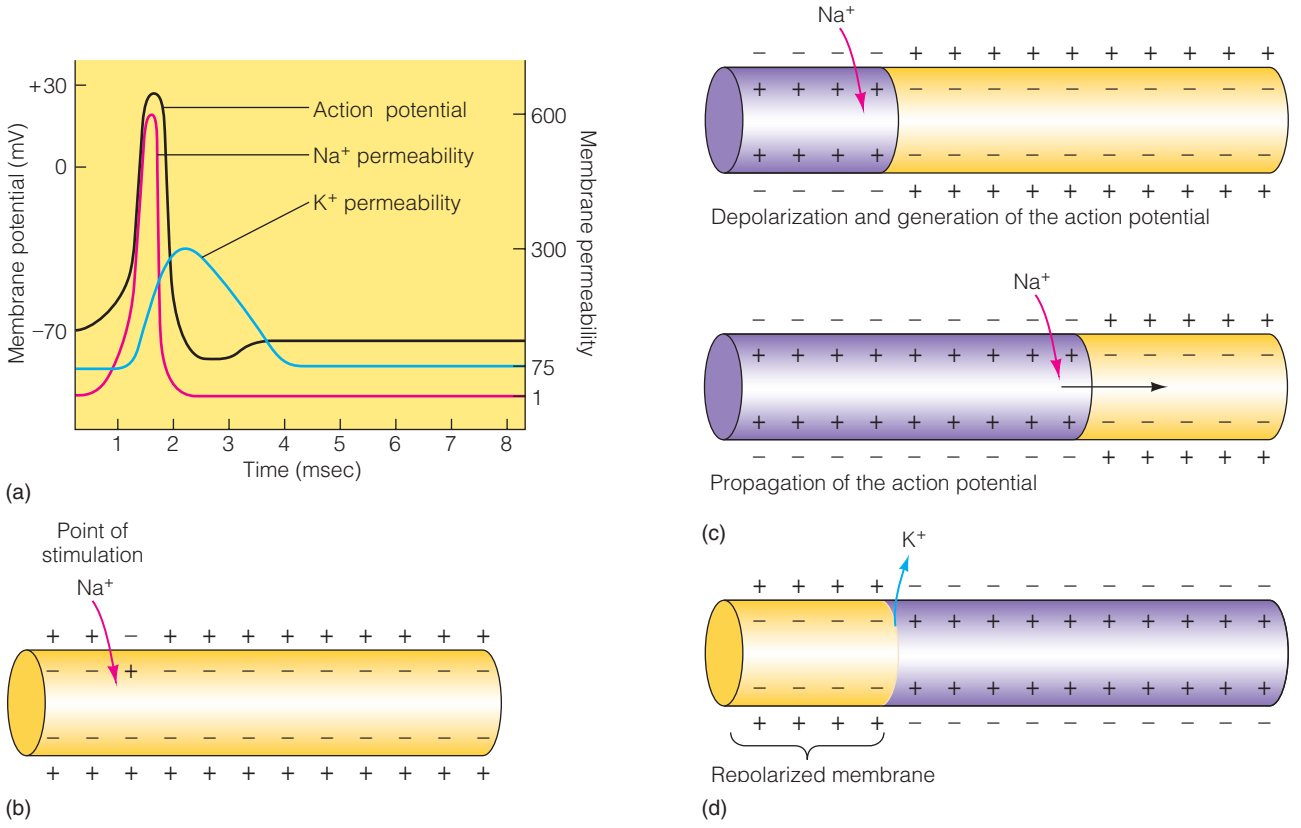


Figure 11-5

Action potential. (a) Stimulating the neuron creates a bioelectric impulse, which is recorded as an action potential. The resulting potential shifts from 270 millivolts to 130 millivolts. The membrane is said to be depolarized. This graph shows the shift in potential and the change in the permeability of sodium (Na^+) and potassium (K^+) ions, which is largely responsible for the action potential. (b) The influx of sodium ions and the depolarization that occur at the point of stimulation. (c) The impulse travels along the membrane as a wave of depolarization. (d) The efflux of potassium ions restores the resting potential, allowing the neuron to transmit additional impulses almost immediately.

and medulla), and cerebellum (**Figure 11-7**). The **cerebrum** is the largest of the regions (80% of total mass) and controls the higher thought processes. A thin layer of gray matter, referred to as cerebral cortex, surrounds the cerebrum (**Figure 11-8**). A thick central core of white matter lies beneath the gray matter. This white matter contains bundles of axons that transmit impulses from the cerebral cortex to the spinal cord, enhancing communication and coordination of activities. The cerebrum is divided into right and left hemispheres by a **longitudinal fissure**. Although minor shifts of one hemisphere into the other may occur, impinging of one hemisphere on the other can have significant and life-threatening effects. Numerous folds, or **gyri**, increase the surface area of the cerebrum. **Sulci** refer to the grooves in between the gyri. At birth, these folds are minimal, but they increase as the brain develops into adulthood. Within each hemisphere are subdivisions called **lobes** named for the bone of the skull that covers it (**Figure 11-9**). The **frontal lobe** facilitates voluntary motor activity and plays a role in personality traits. The **parietal lobe** receives and interprets sensory input with the exception of smell, hearing, and vision. The **occipital lobe** processes visual information. The **temporal lobe** plays an essential role in hearing and memory. Areas within and across these lobes can be classified as three types—motor (which stimulates muscle activity), sensory (which receives sensory information), and association (which integrates information and initiates coordinated responses).

The **diencephalon** includes the thalamus and hypothalamus (**Figure 11-10**). The **thalamus** receives and relays most of the sensory input, affects mood, and initiates body movements (especially those associated with fear or anger). The **subthalamus** participates in

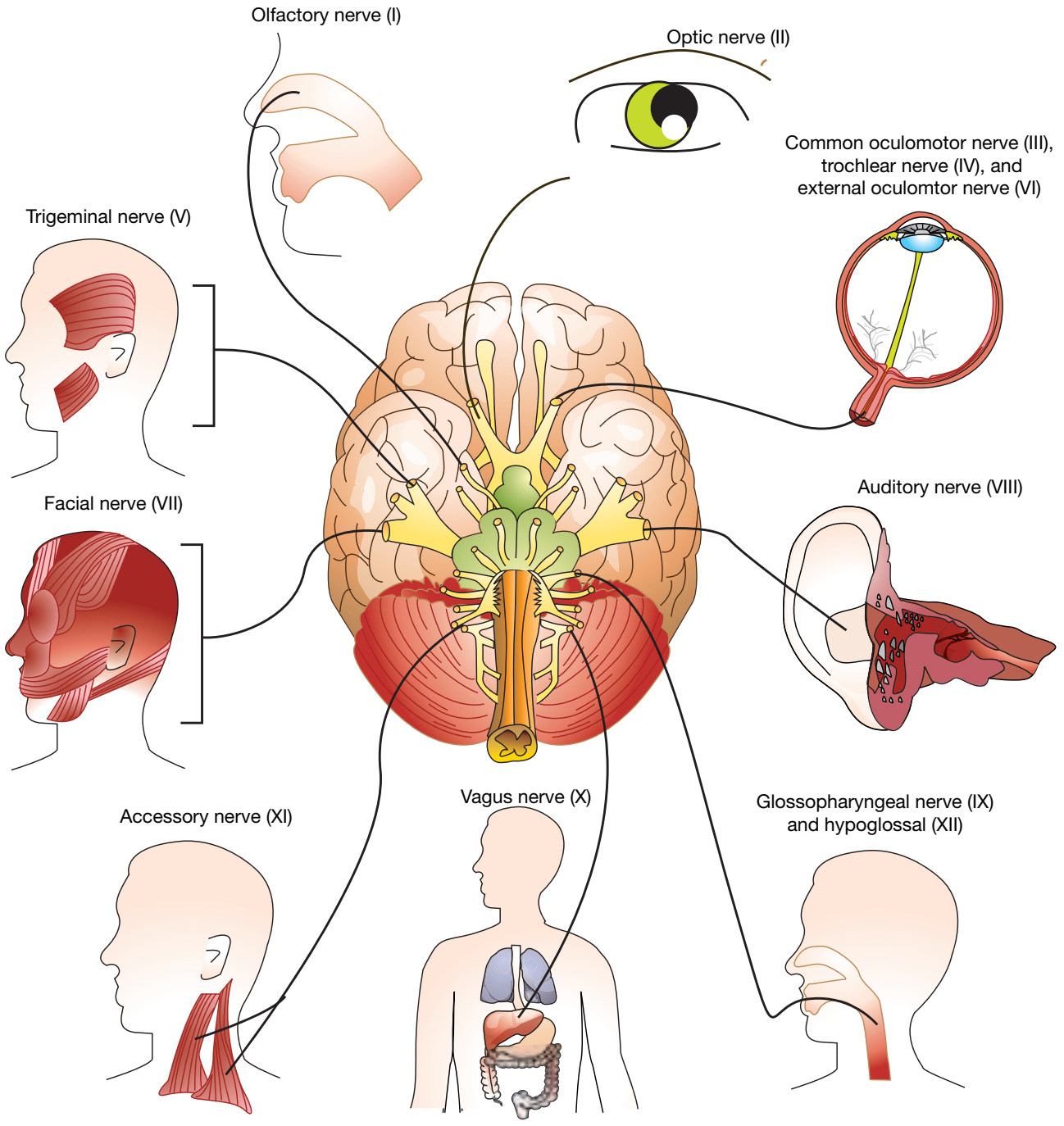


Figure 11-6

The cranial nerves.

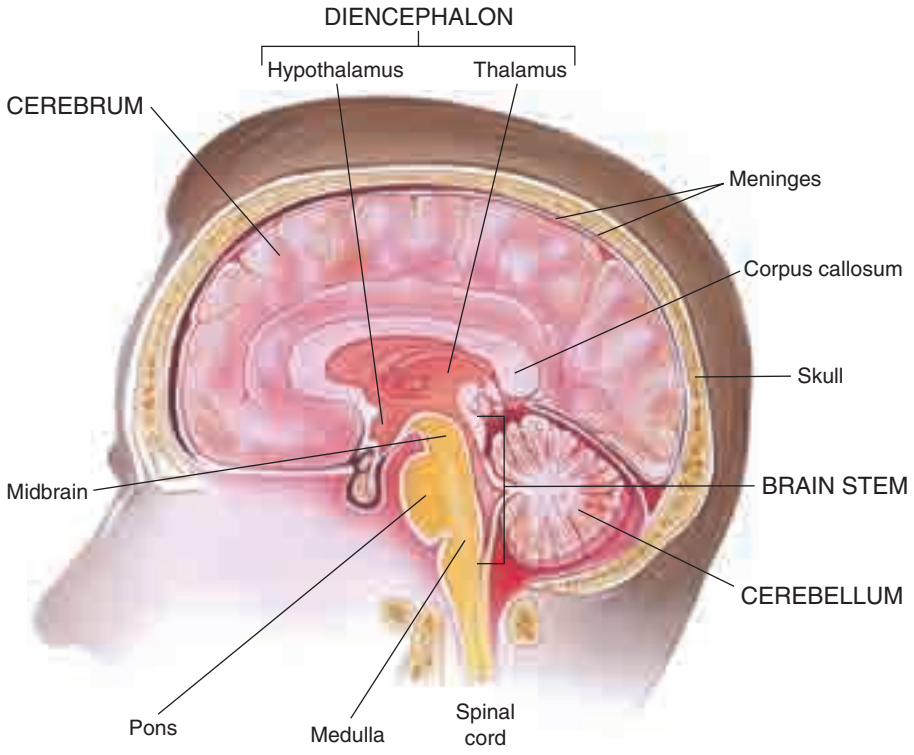


Figure 11-7

The major regions of the brain.

motor activities, but the functions of the **epithalamus**, especially the pineal body, are unclear. The **hypothalamus** is the most inferior portion of the diencephalon; it regulates many bodily functions (see Chapter 10).

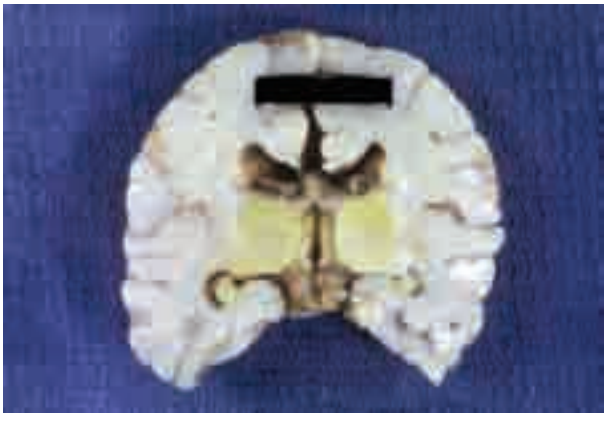


Figure 11-8

The cerebrum.

The **brain stem** (including the pons, cerebellum, and medulla) connects the brain to the spinal cord. The brain stem is crucial for many basic body functions (e.g., maintaining heart rate, blood pressure, and respiration), and injury to the brain stem can easily result in death. The brain stem collaborates with the hypothalamus to regulate these vital activities. In addition to containing control regions, the brain stem is a main thoroughfare for information traveling to and from the brain. Of the 12 cranial nerves, 10 exit from the brain stem. The **pons** contains nerves that regulate sleep and breathing. The **midbrain** is the smallest region of the brain, and it acts as a sort of relay station for auditory and visual information. The midbrain controls the visual and auditory systems as well as eye movement. The **medulla** is a conduction pathway for ascending and descending nerve tracts. The medulla coordinates heart rate, peripheral vascular resistance, breathing, swallowing, vomiting, coughing, and sneezing. Most of the many nerve fibers passing through the brain stem have branches that terminate in a region of the brain stem called the **reticular formation**. The reticular formation acts like a gatekeeper, receiving all incoming and outgoing information. The reticu-

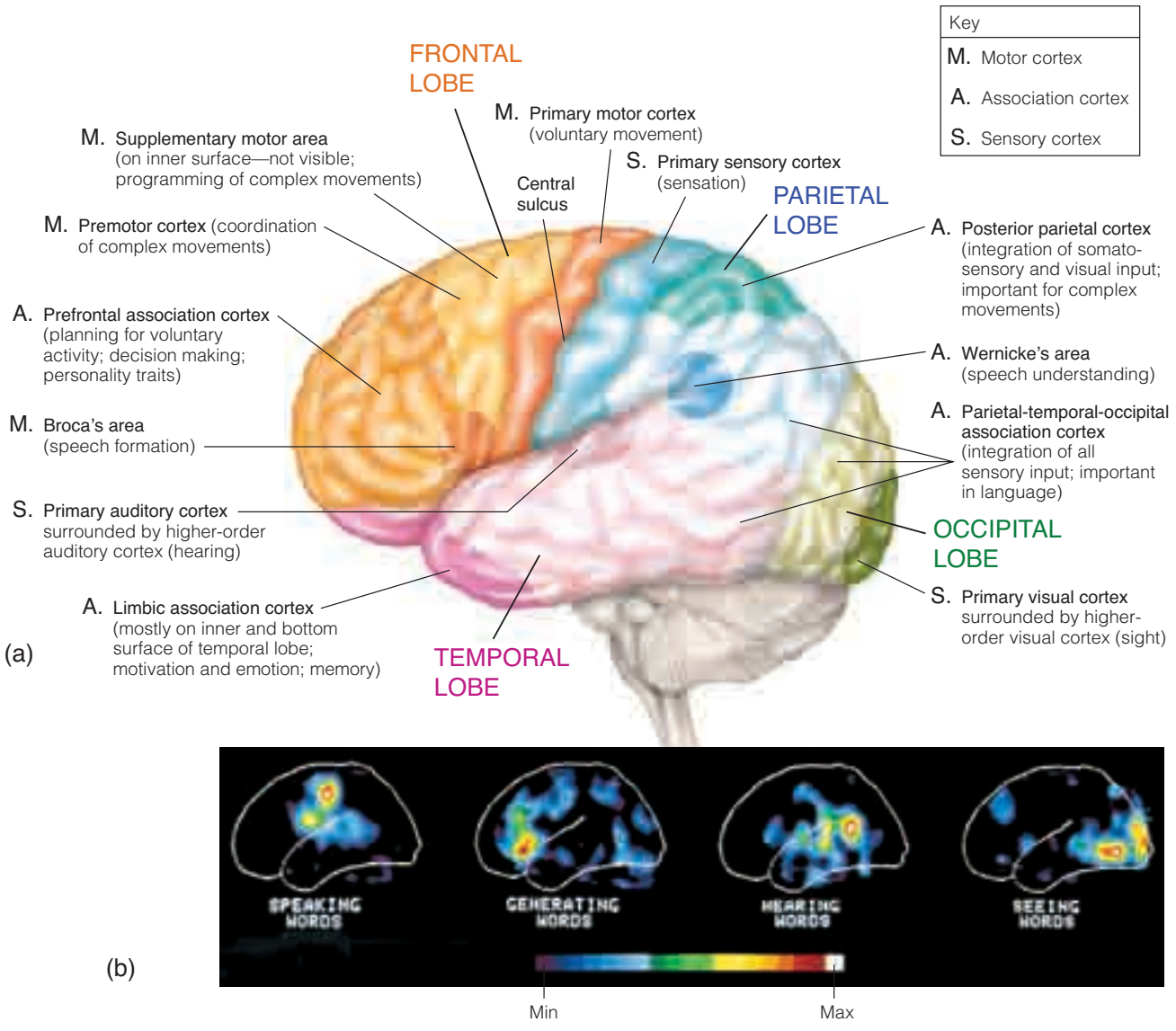


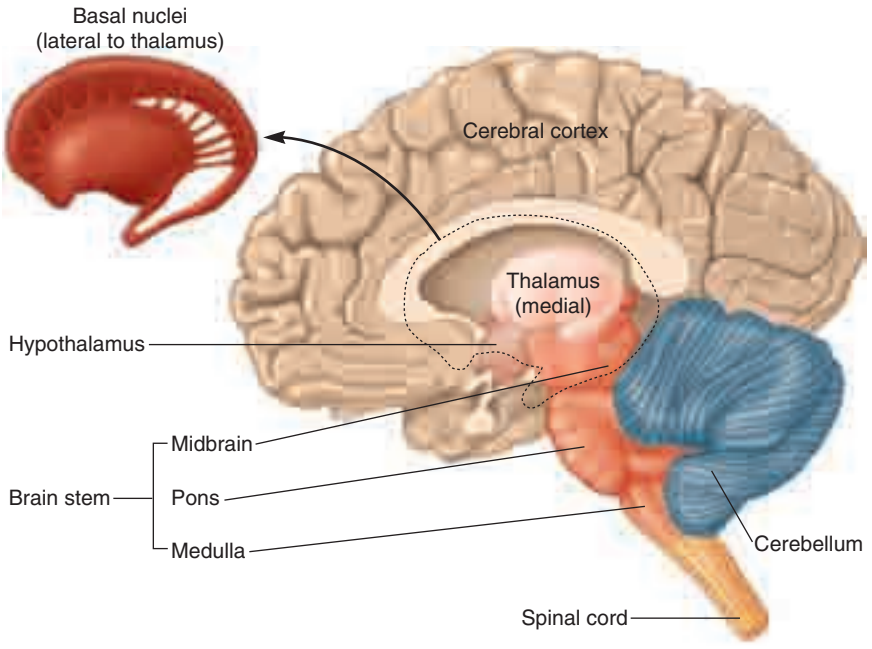
Figure 11-9

The lobes of the cerebrum. (a) The cerebral cortex has three principal functions: receiving sensory input, integrating sensory information, and generating motor responses. Special sensory areas handle vision, smell, taste, and hearing. (b) A PET scan reveals the locations of increased blood flow in the brain during performance of certain tasks.

lar formation sends impulses to the cerebral cortex through specialized nerve fibers. These fibers make up the **reticular activation system** (**Figure 11-11**). The reticular formation and reticular activation system are responsible for alertness during the day and can prevent sleeping at night.

The **cerebellum** communicates with other regions of the brain to coordinate the synergistic motion of muscle movement and balance as well as cognition. Deep within the cerebrum, diencephalon, and midbrain is a set of key structures called the **basal ganglia**. The basal ganglia play a pivotal role in coordination, motor movement, and posture. Portions of the cerebrum and diencephalon comprise the limbic system (**Figure 11-12**). The limbic system works in conjunction with the hypothalamus to influence instinctive behavior, emotions, motivation, mood, pain, and pleasure.

The **spinal cord** exits the skull through the large and only opening in the skull, called the **foramen magnum**. The spinal cord extends through the **vertebral**



Cerebral cortex

- Receives sensory information from skin, muscles, glands, and organs
- Sends messages to move skeletal muscles
- Integrates incoming and outgoing nerve impulses
- Performs associative activities such as thinking, learning, and remembering

Basal nuclei

- Play a role in the coordination of slow, sustained movements
- Suppress useless patterns of movement

Thalamus

- Relays most sensory information from the spinal cord and certain parts of the brain to the cerebral cortex
- Interprets certain sensory messages such as those of pain, temperature, and pressure

Hypothalamus

- Controls various homeostatic functions such as body temperature, respiration, and heartbeat
- Directs hormone secretions of the pituitary

Cerebellum

- Coordinates subconscious movements of skeletal muscles
- Contributes to muscle tone, posture, balance, and equilibrium

Brain stem

- Origin of many cranial nerves
- Reflex center for movements of eyeballs, head, and trunk
- Regulates heartbeat and breathing
- Plays a role in consciousness
- Transmits impulses between brain and spinal cord

Figure 11-10

The regions of the brain and their functions.

canal to the second lumbar vertebra. At this point, the spinal cord transitions into individual nerve roots referred to as the **cauda equina**. The spinal cord consists of 31 pairs of spinal nerves that branch off at regular intervals (**Figure 11-13**).

The central portion of the spinal cord is an H-shaped area of gray matter, which contains nerve cell bodies. White matter comprised of nerve fiber tracts, or pathways, surround the gray matter (**Figure 11-14**). **Ascending fibers**, or **afferent tracts**, carry sensory

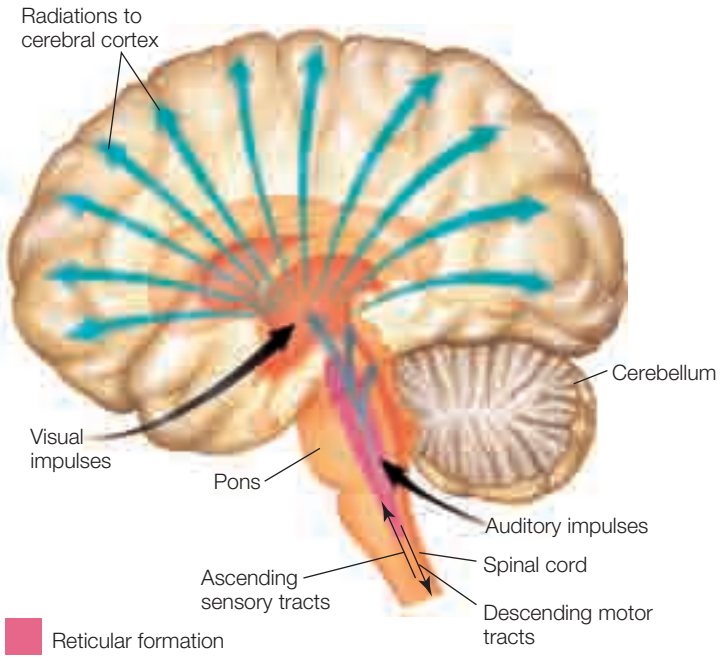


Figure 11-11

The reticular activation system.

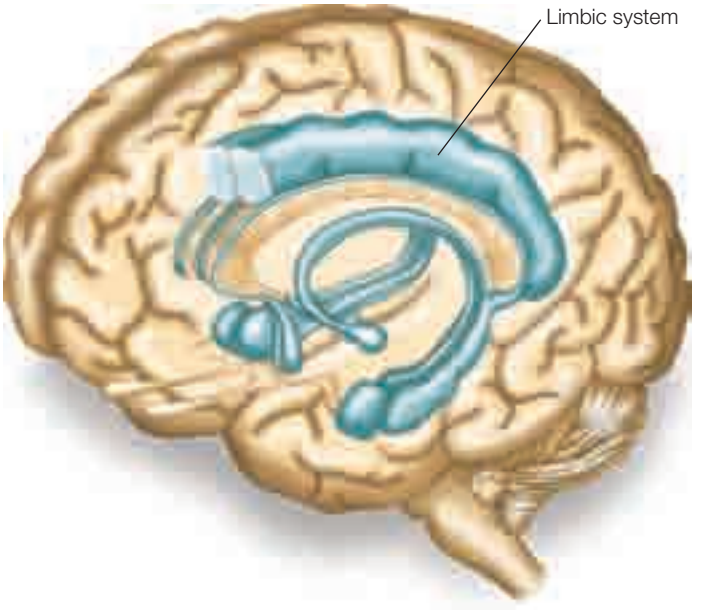


Figure 11-12

The limbic system. The odd assortment of structures shown in green is the limbic system. The limbic system is the seat of emotions such as joy and instincts and is home to other functions as well.

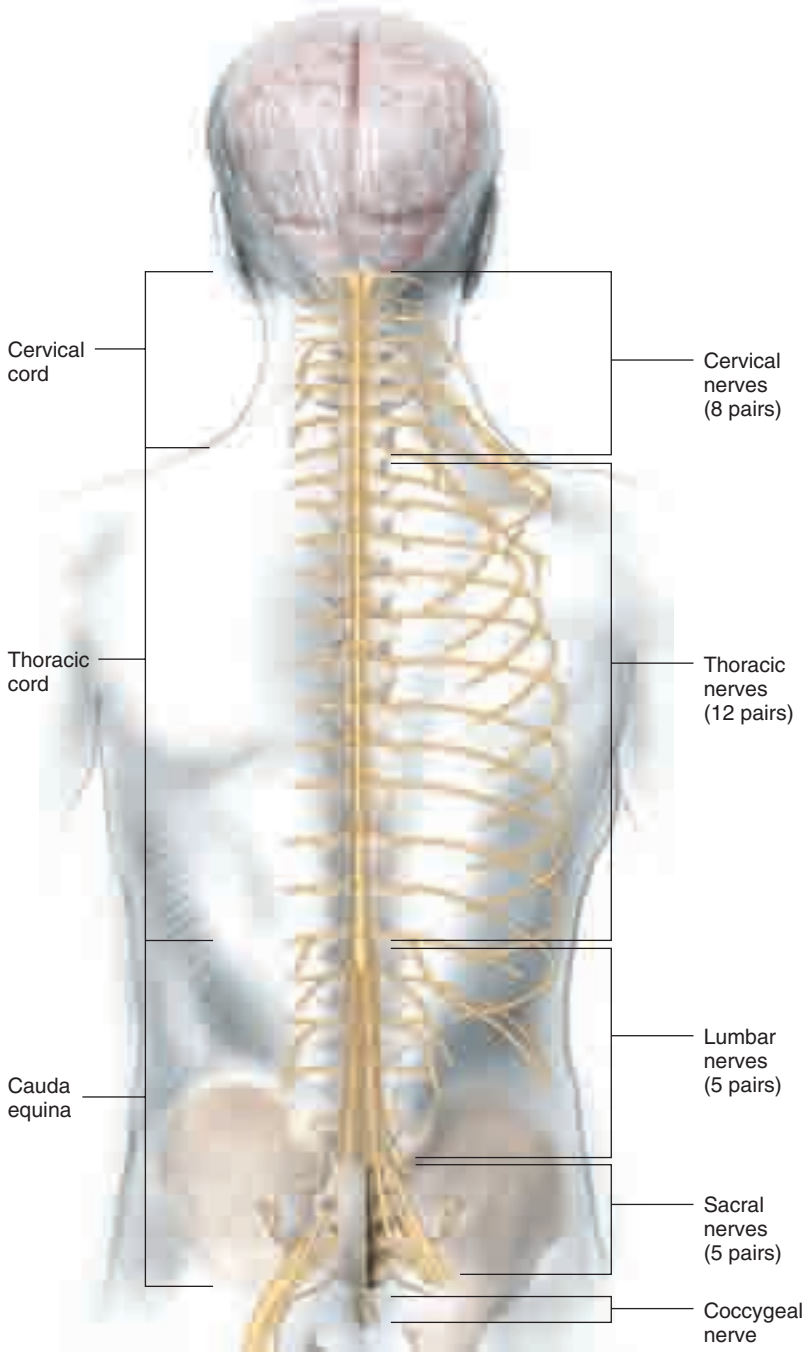


Figure 11-13

The spinal cord.

information in the form of action potentials from the periphery back to the brain. **Descending fibers**, or **efferent tracts**, carry motor impulses in the form of action potentials from the brain to the fibers of the PNS. The ascending fibers have a variety of tracts that communicate specific sensory input. These pathways include anterior spinothalamic tracts (which permit sensations of light touch, pressure, tickling, and itching), lateral spinothalamic tracts (which allow the sensations of pain and temperature), spinocerebellar tracts (which establish the body's position in relation to the cerebellum), corticospinal tracts (which coordinate

movements, especially in the hands), vestibulospinal tracts (which are responsible for involuntary movements), and reticulospinal tracts (also responsible for involuntary movements).

The **spinal reflex arcs** refer to the process that creates an unconscious response to stimuli (**Figure 11-15**). An example of this arc can be seen when the patella is gently tapped with a reflex hammer. The tendon stretch reflex is elicited when the patella is tapped, causing the lower leg to sharply move forward (called extension) and then backward (called flexion). **Flexor reflex** refers to a withdrawal reflex in response to touching an unpleasant stimulus (e.g., extreme heat). The flexor reflex causes the muscles of a limb to withdraw the limb from the source of the stimulus without any conscious action. The tracts of the spinal cord and brain regulate these impulses.

Peripheral Nervous System

The **nerves** of the PNS consist of bundles of nerve fibers, and each fiber is part of the neuron. These nerves transport messages to and from the CNS. These nerves end on receptors that respond to a variety of internal and external stimuli. The 31 spinal nerve pairs (8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal) branch directly off the spinal cord to make up the PNS. Each spinal nerve pair is named for the vertebral level at which it exits the spinal cord (e.g., C3 is the 3rd cervical nerve and T12 is the 12th thoracic nerve) and innervates specific areas of the body (**Figure 11-16**). Ganglia refer to collections of nerve cell bodies outside the CNS. Spinal nerves arise from several small nerves called **rootlets** along the dorsal and ventral surfaces of the spinal cord (**Figure 11-17**). Approximately 6–8 rootlets combine to form each **dorsal root** and **ventral root**. These roots come together to form the spinal nerve.

Each spinal nerve of the PNS is comprised of two types of nerves—sensory and motor. The **sensory nerves**, or **afferent nerves**, carry impulses (regarding information) from the body to the brain. A **dermatome** is the area of the skin innervated by a given pair of spinal sensory nerves. Each spinal nerve, with the exception of C1, has a specific body surface area

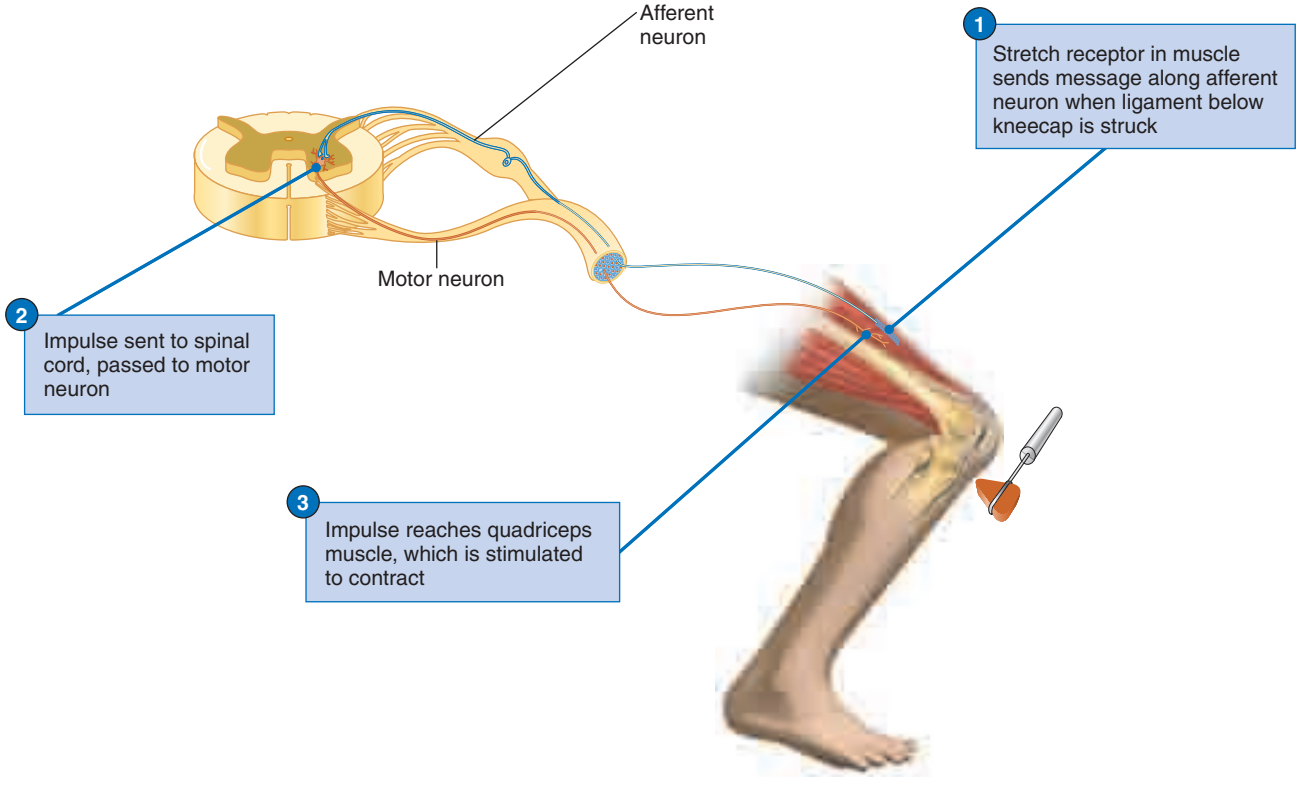


Figure 11-14

Spinal cord nerve tracts.

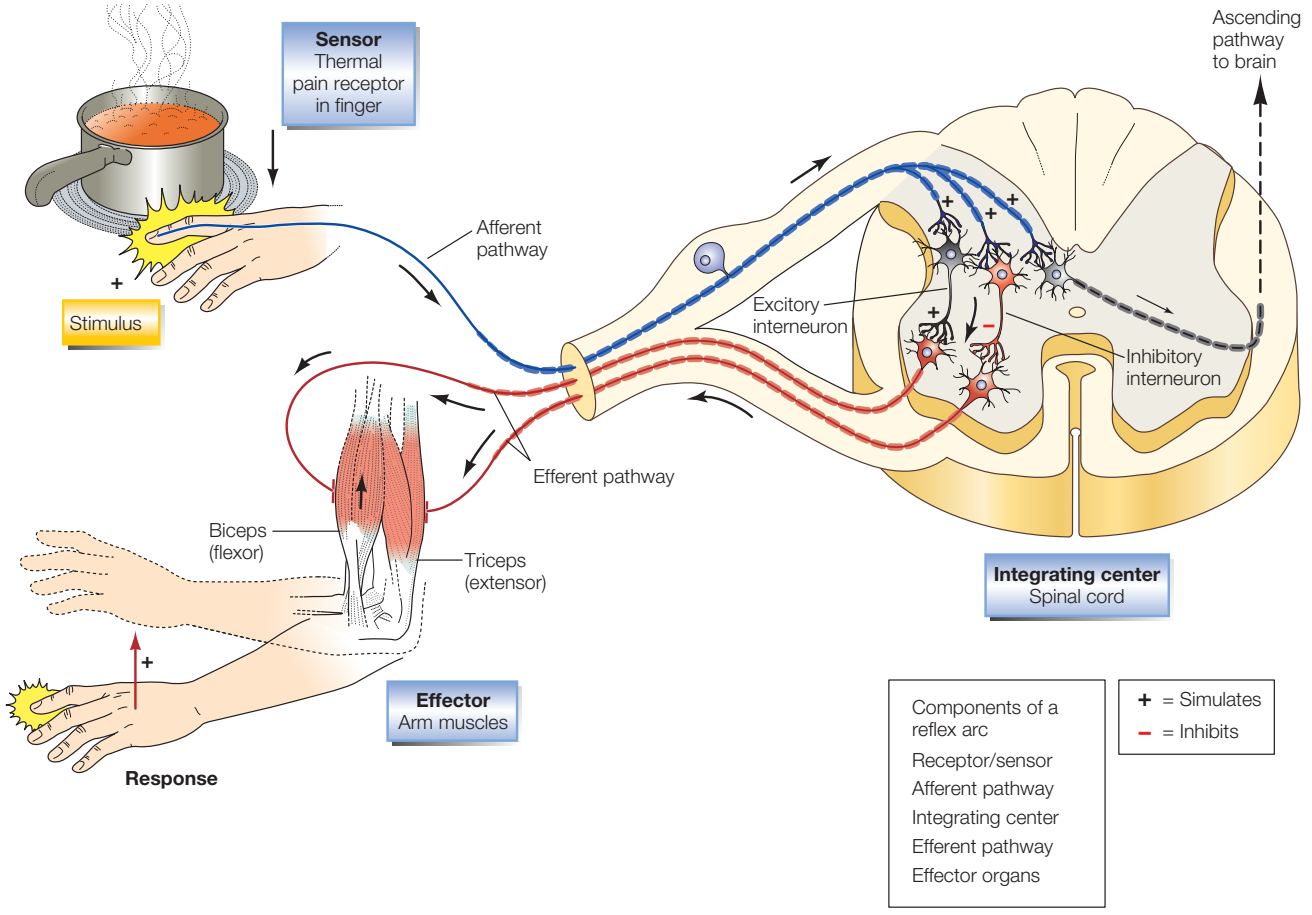


Figure 11-15

The spinal reflex arc. When you accidentally touch a hot pan on the stove, you withdraw your hand before your brain even knows what is happening. This occurs because of a reflex arc. Sensory fibers send impulses to the spinal cord. The sensory impulses stimulate motor neurons in the spinal cord. This causes muscle contraction in the flexor muscles (1) and inhibits muscle contraction in the extensor muscles (2), allowing you to withdraw your hand. Nerve impulses also ascend to the brain to let it know what is happening.

to which it obtains sensory information. The **motor nerves**, or **efferent nerves**, carry impulses (regarding action) from the brain to the corresponding muscle receptor, resulting in muscle contraction and movement. **Interneurons** connect the sensory and motor neurons in the spinal cord.

Several nerves intersect to form an organized collaboration, or **plexus**. Four plexuses occur in the body—the cervical (located at C1 to C4), brachial (located at C5 to T1), lumbar (located at L1 to L4), and sacral (located at L4 to S4). These plexuses branch into the **peripheral nerves** that supply sensory and motor functions to many areas of the body.

Autonomic Nervous System

The **autonomic nervous system** controls smooth muscles and is responsible for the fight-or-flight response (see Chapter 2). The autonomic nervous system is not under conscious control and affects such activities as heart rate, blood pressure, and intestinal motility. The autonomic nervous system has two subdivisions—sympathetic and parasympathetic. These two divisions have an antagonistic effect with each other to aid in maintaining homeostasis (**Figure 11-18**). The **sympathetic nervous system (SNS)** is responsible for the fight-or-flight response. This response is initiated when a person

MYTH BUSTERS

Some myths surrounding the brain warrant discussion.

MYTH 1: The brain is gray.

The living, pulsing brain currently residing in your skull is not just a dull, bland gray organ like often depicted in movies; the brain is also white, black, and red. Like many myths, this one has a grain of truth, because much of the brain is gray. Sometimes the entire brain is referred to as gray matter. However, the brain also contains white matter, which comprises nerve fibers that connect the gray matter. The black component is called **substantia nigra**, which is Latin for "black substance." The substantia nigra is black because of **neuromelanin**, a specialized type of pigment. Finally, we have red because of the many blood vessels in the brain.

MYTH 2: Listening to classical music, especially by Mozart, increases intelligence.

How did this myth get started? In the 1950s, a physician named Albert Tomatis began the trend, claiming success using Mozart's music to help people with speech and auditory disorders. In the 1990s, 36 students in a study at the University of California at Irvine listened to 10 minutes of a Mozart sonata before taking an IQ test. The study reported that the students' IQ scores went up by about 8 points, and the Mozart effect was born. Multiple products have been sold based on this assumption. However, the original University of California at Irvine study has been controversial in the scientific community. Other scientists have been unable to replicate the original results, and current scientific evidence does not support that listening to Mozart, or any other classical music, increases intelligence. However, some evidence indicates that learning an instrument improves concentration, self-confidence, and coordination. Mozart certainly cannot hurt you, and you might even enjoy it if you try it, but you will not get any smarter.

MYTH 3: You only use 10% of your brain.

This myth is probably one of the most well-known myths about the brain. This assumption seems puzzling at first glance. We have the biggest brain in proportion to our bodies of any animal, so why would we not use all of it? Many people have jumped on the idea, writing books and selling products that claim to tap into the other 90%. Believers in psychic abilities point to this ability as proof, saying that people with these abilities have tapped into the rest of their brains. This myth is false. In addition to 100 billion neurons, the brain is also full of other types of cells that are continually in use. Significant neurologic deficits can occur from even minor damage depending on the location, so it is highly unlikely that we could function with only 10% of our brain in use. Brain scans have shown that no matter what we are doing, our brains are always active. Some areas are more active at any one time than others, but unless we have brain damage, no one part of the brain is completely turned off. So, there is no hidden, extra potential you can tap into, in terms of actual brain space.

MYTH 4: Games like sudoku and Brain Age keep your brain young.

There is some truth to this myth! Continued mental engagement has benefits, and puzzles can help you get good at a specific skill, like memorizing grocery lists or hand–eye coordination. But most evidence suggests that practicing a task only helps you get better at that particular task. Far better for mental function is physical exercise. Regular fitness exercise is especially effective in the elderly, who may suffer from gradual problems with cognitive function such as planning ahead and abstract thinking.

LEARNING POINTS

How do we learn and store memories? Learning is the acquisition and retention of new information, and memory is the storage and recall of information. Both are dependent on proper nutrition and adequate sleep. Newly acquired memory is first stored in the short-term memory, where it is held for seconds to hours. Cramming for tests puts most of the information into short-term memory. Unfortunately, soon after the test, the information fades—a good reason not to cram! Long-term memory holds information for days to years. Transferring information from short- to long-term requires special efforts such as repetition, mnemonics, and rhymes. Recalling information in short term is often faster than recalling information in long-term memory. When information is lost from short-term memory, it is usually lost forever. Information you cannot recall from long-term memory, on the other hand, is often still there; it just requires time or stimuli to extract it. However, not all information in long-term memory is stored forever. Memories are stored in neurons throughout the cerebral cortex (especially the temporal lobe), cerebellum, and the limbic system. The hippocampus seems to be crucial in transferring information from short- to long-term memory. So, use this knowledge to help you study by not cramming and moving information from short- to long-term memory by paying attention, making the information memorable, and relating new information with facts you already know. But do not forget that you still have to get plenty of rest and eat a balanced diet to optimize your learning potential!

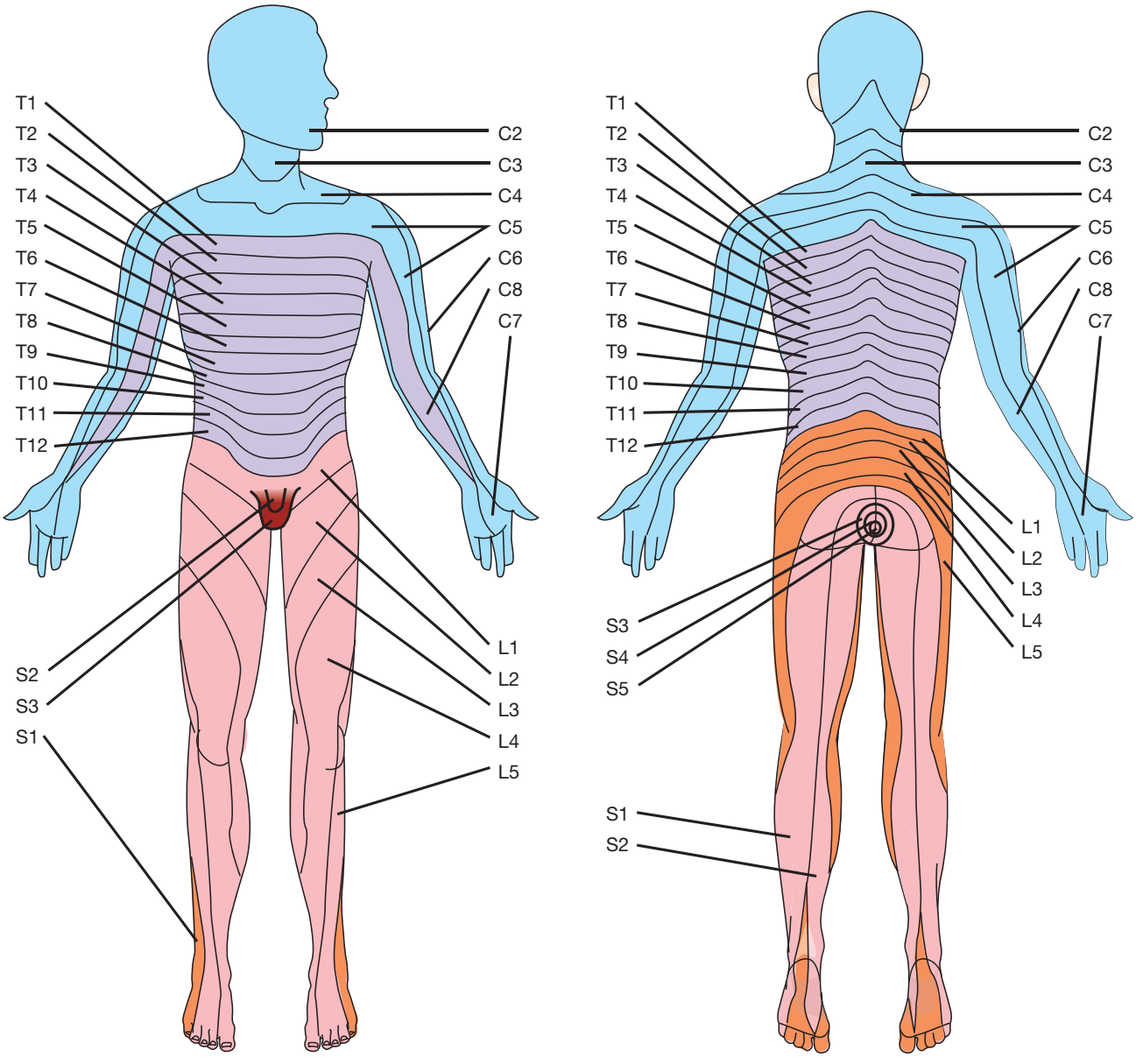


Figure 11-16

Spinal nerve innervation.

is startled or faced with danger and is augmented by secretions of the adrenal medulla. The **parasympathetic nervous system** is responsible for the rest-and-digest response. Neurotransmitters and receptors are important in the autonomic nervous system because the SNS and the parasympathetic nervous system will stimulate or inhibit these sites, leading to the physiologic response (**Table 11-1**). The SNS stimulates the adrenergic receptors while the parasympathetic nervous system stimulates the cholinergic receptors. Medications can be given that can stimulate or inhibit these receptors as well.

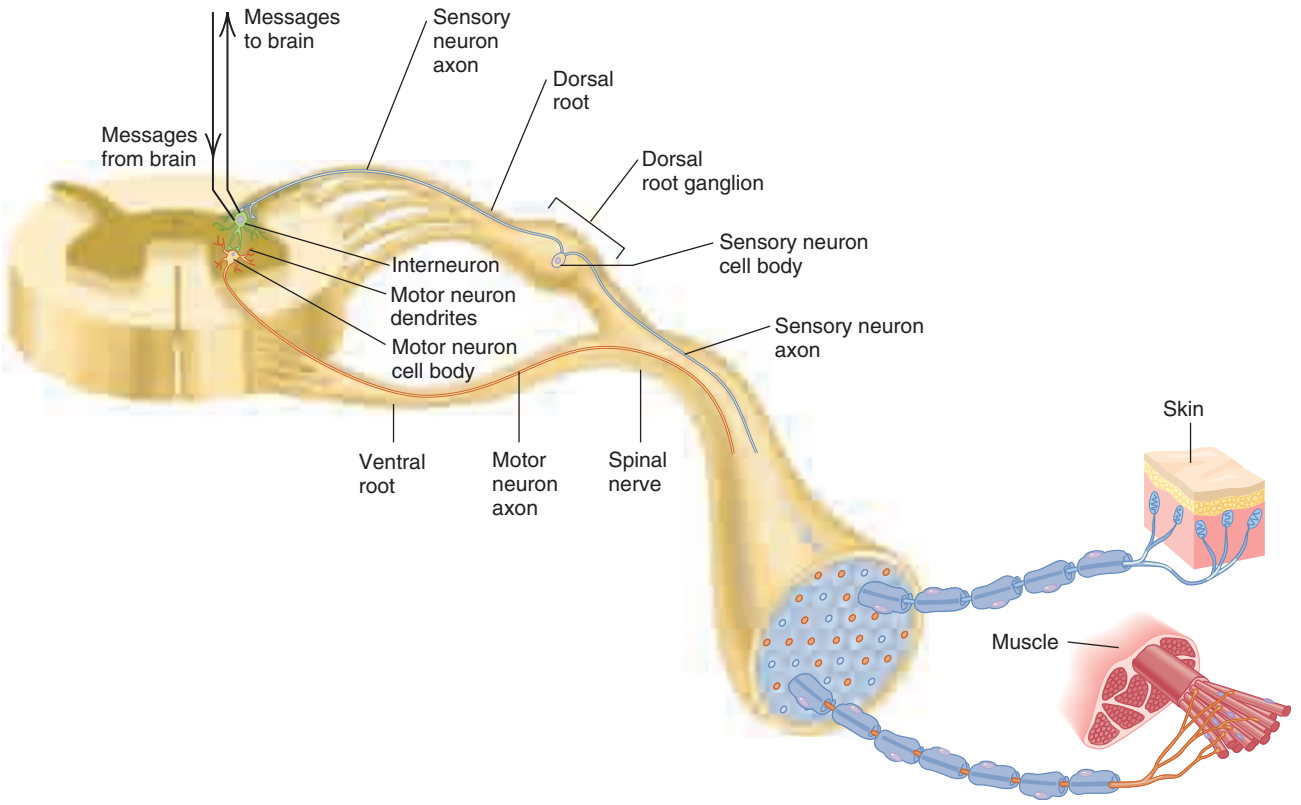


Figure 11-17

The dorsal root ganglion.

Table 11-1 Types of Autonomic Receptors

Neurotransmitter	Receptor	Primary Locations	Responses
Acetylcholine (cholinergic)	Nicotinic	Postganglionic neurons	Stimulation of smooth muscle and gland secretions
	Muscarinic	Parasympathetic target: organs other than the heart	Stimulation of smooth muscle and gland secretions
		Heart	Decreased heart rate and force of contraction
Norepinephrine (adrenergic)	$Alpha_1$	All sympathetic target organs except the heart	Constriction of blood vessels, dilation of pupils
	$Alpha_2$	Presynaptic adrenergic nerve terminals	Inhibition of release of norepinephrine
	$Beta_1$	Heart and kidneys	Increased heart rate and force of contraction; release of renin
	$Beta_2$	All sympathetic target organs except the heart	

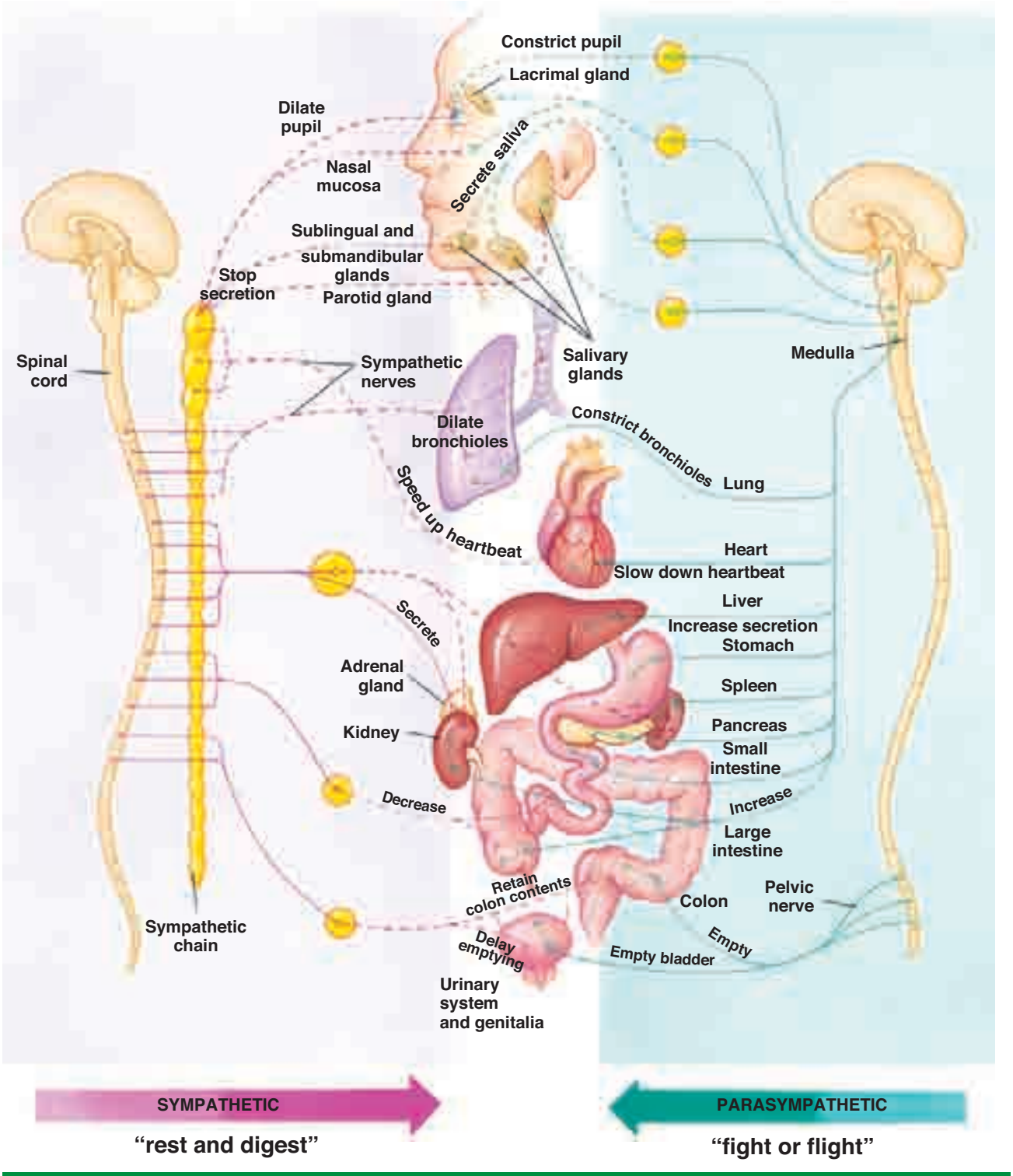


Figure 11-18

Comparison of the two divisions of the autonomic nervous system.

Congenital Neurologic Disorders

Congenital defects of the nervous system are often serious with lifelong consequences. These disorders often have limited treatment options and require long-term management of complications.

Hydrocephalus

Hydrocephalus is a condition in which excess CSF accumulates within the skull, which dilates the ventricles and compresses the brain and blood vessels (**Figure 11-19**). The pressure from the excess CSF thins the cortex, causing severe brain damage. The CSF accumulates when the CSF flow is disrupted (referred to as **noncommunicating** or an **obstructive hydrocephalus**) or when it is not properly absorbed by the bloodstream (referred to as **communicating hydrocephalus**). Hydrocephalus is a common condition (affecting 1 out of 500 children), which may be present at birth or develop later in life. Risk factors for hydrocephalus at any age include prematurity, pregnancy complications, other congenital defects (especially nervous system defects), nervous system tumors, CNS infections, cerebral hemorrhage, and severe head injuries. If left untreated, hydrocephalus is often fatal (50–60% mortality rate). Prognosis depends on early treatment and comorbidity.

Clinical manifestations of hydrocephalus reflect the increased intracranial pressure (ICP). These mani-

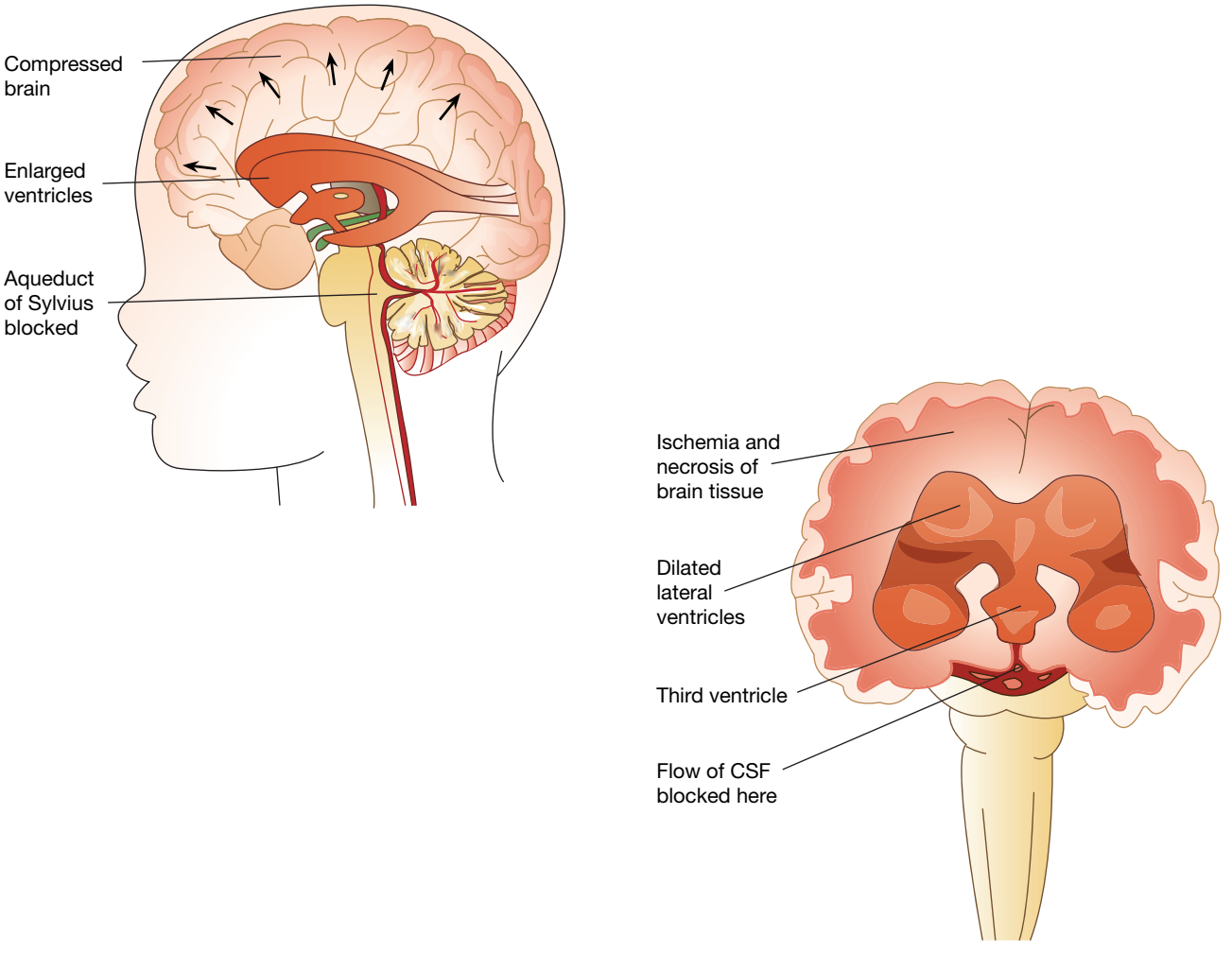


Figure 11-19

Hydrocephalus development.

festations vary by age group, underlying etiology, and disease progression. In infants, clinical manifestations often include:

- An unusually large head (**Figure 11-20**)
- A rapid increase in the head size
- A bulging fontanelle, or soft spot, on the top of the head
- Vomiting (often projectile)
- Lethargy
- Irritability
- High-pitched cry
- Feeding difficulties
- Seizures
- Eyes that gaze downward (setting-sun appearance)
- Development delay

In older children and adults, the head cannot enlarge because the sutures have closed. Clinical manifestations in these groups may include:

- Headache followed by vomiting
- Nausea
- Blurred vision or diplopia (double vision)
- Sluggish pupil response to light
- Eyes that gaze downward (setting-sun appearance)
- Problems with balance, coordination, or gait
- Extreme fatigue
- Slowing or regression of development
- Memory loss
- Confusion
- Urinary incontinence
- Irritability
- Personality changes
- Impaired performance in school or work

Figure 11-20

Hydrocephalus.

Diagnostic procedures for hydrocephalus may be conducted during pregnancy or after birth. These procedures consist of a history, physical examination (including head circumference measurement and a neurologic assessment), head computed tomography (CT), head magnetic resonance imaging (MRI), head X-ray, and prenatal ultrasound. The goal of treatment is to minimize brain damage by reducing CSF. Blockages are surgically removed, if possible. If the blockage cannot be removed, a shunt (flexible tube) may be placed within the brain to allow CSF to flow around the blocked area. The shunt tubing travels to another part of the body, such as the peritoneal cavity or right atrium, where the extra CSF can be drained and absorbed. Shunt replacement may be needed periodically as a child grows or if it becomes blocked or infected. Antibiotic therapy will treat hydrocephalus caused by an infection or if a shunt infection develops. An endoscopic third ventriculostomy can also be performed to relieve pressure without replacing the shunt. Follow-up examinations generally continue throughout a child's life to monitor developmental progress and to manage any intellectual, neurologic, or physical problems. A multidisciplinary team (e.g., nurses, occupational therapists, educational specialists, social services personnel, and support groups) can provide emotional support and assistance with the care of those patients who have significant brain damage.

Spina Bifida

Spina bifida is the most common birth defect in the United States, affecting approximately 1 child of every 1,500 births each year (Centers for Disease Control and Prevention, 2010a). Spina bifida is a neural tube defect that can vary in severity from mild to debilitating. Neural tube development begins early in pregnancy, starting at the cervical area and progressing toward the lumbar area, and the neural tube usually closes by the 4th week of gestation. In spina bifida, the posterior spinous processes on the vertebrae fail to fuse. This

opening permits the meninges and spinal cord to herniate, resulting in neurologic impairment. The lumbar area of the vertebrae is most commonly the site of the defect. The exact cause of spina bifida is unknown, but it is thought to be a result of genetic and environmental influences. Spina bifida is most common in Caucasian and Hispanic populations. Additional maternal risk factors for developing this defect include family history of neural defects, folate deficiency, certain medications (e.g., antiseizure agents), diabetes mellitus, prepregnancy obesity, and increased body temperature (e.g., from hot tubs, saunas, and tanning beds). Complications of spina bifida include physical and neurologic impairments as well as hydrocephalus and meningitis. Children with spina bifida are usually of normal intelligence, but they may have learning problems because of the chronic nature of the condition.

Spina bifida occurs in three forms, each varying in severity (**Figure 11-21**). These types include:

- **Spina bifida occulta** is the mildest form. It results in a small gap in one or more of the vertebrae. The spinal nerves and meninges do not usually protrude through the opening, so most children with this form have no clinical manifestations and experience no neurologic deficits. The defect may not be evident other than a dimple, birthmark, or tuft of hair over the site.
- **Meningocele** is a rare form that involves the same bony defect as in spina bifida occulta, but the meninges protrude through the vertebral opening. The meninges and CSF form a sac on the surface of the infant's back. Transillumination (shining light through the tissue) can confirm the absence of nerve tissue in the sac. Because the spinal cord develops normally, neurologic impairment is usually not present, and these membranes can be removed by surgery with little or no damage to nerve pathways. However, infection or rupture of the sac can lead to neurologic impairment.
- **Myelomeningocele**, also known as open spina bifida, is the most severe form. In this form, the spinal canal remains open along several vertebrae in the lower or middle back. The meninges, spinal cord, spinal nerves, and CSF protrude through this large opening at birth and form a sac on the infant's back (**Figure 11-22**). Skin covers the sac in some cases. However, tissues and nerves are exposed in most cases, making the infant vulnerable to life-threatening infections. Neurologic impairment (often including paralysis), bowel and bladder problems (e.g., incontinence, urinary tract infections, and constipation), seizures, and other medical complications (e.g., skin conditions and latex allergies) are common.

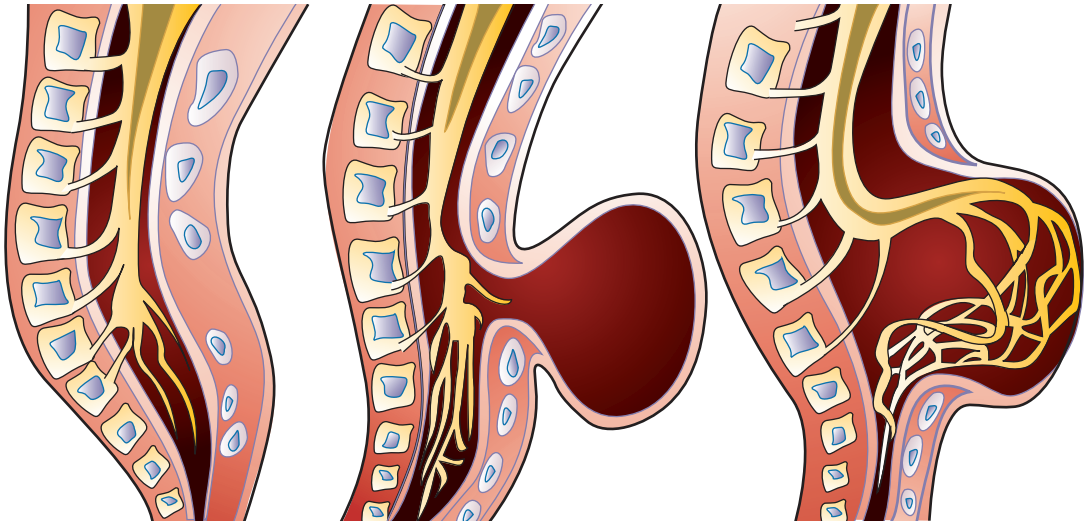


Figure 11-21

Most common types of spina bifida.

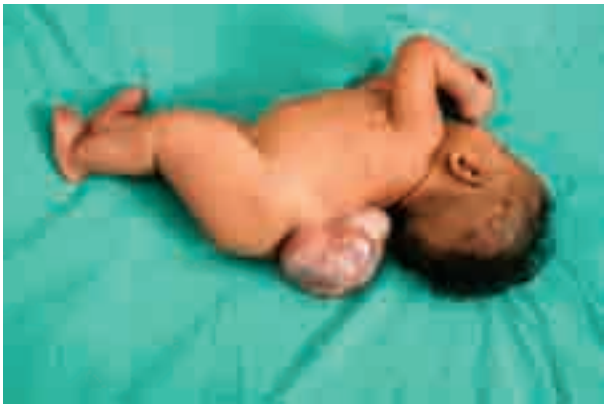


Figure 11-22

Meningomyocele.

Clinical manifestations depend on the type and severity of spina bifida. Diagnostic procedures may be performed during pregnancy or after birth. These procedures may include a history, physical examination, check of maternal serum and amniotic fluid alpha-fetoprotein levels, prenatal ultrasound, spinal X-ray, spinal CT, and spinal MRI. Treatment strategies vary depending on the type and severity. For instance, spina bifida occulta often requires no treatment. Surgery is the mainstay of treatment for the other two types; however, suggested timing of the surgery (in utero, immediately after birth, or delayed) remains debated. Surgery usually includes replacing meninges and closing the vertebral opening. A shunt may be placed during surgery to control hydrocephalus. Performing the surgical repair in utero may enhance outcomes but will not restore lost neurologic functioning. Additional risk may be incurred with this procedure including premature delivery and death. If spina bifida is diagnosed before birth, cesarean delivery is preferred to prevent rupture of the sac or damage to any exposed nerves. Long-term support of a multidisciplinary team (e.g., a nurse, physical therapist, social worker, and an education specialist) will be necessary to limit complications and promote positive outcomes.

Cerebral Palsy

Cerebral palsy (CP) refers to a group of nonprogressive disorders that appear in infancy or early childhood and permanently affect motor movement and muscle coordination. In addition to motor dysfunction, other cerebral functioning may be affected (e.g., cognition and communication). CP usually results from damage to the cerebellum during the prenatal period (often during childbirth), but it can occur any time during the first 3 years of life, when the brain is developing. CP can also occur because of brain abnormalities. In the United States, CP occurs in approximately 3–4 out of 1,000 births. CP is not curable, but the right treatment can make a significant impact on the child's prognosis. These therapies are costly. According to the CDC (2004), the average lifetime cost (direct and indirect) for one person with CP is estimated to be $921,000 (in 2003 dollars). The estimated lifetime costs (direct and indirect) for all people with CP who were born in 2000 will total $11.5 billion (in 2003 dollars). Contributing factors to developing CP include:

- Prematurity
- Breech births (feet first rather than head first)
- Multiple fetuses
- Hypoxia
- Hypoglycemia (in either the mother or the child)
- Cerebral hemorrhage
- Neurologic infections (e.g., meningitis and encephalitis)
- Head injury

CASE STUDY

M.S. is a 26-year-old woman pregnant with her first child. Her husband accompanied her to all her prenatal visits. An ultrasound during a routine visit at 34 weeks' gestation revealed that the baby had hydrocephalus and a myelomeningocele. The parents were initially devastated but remained very excited about the birth of their first child. M.S. was scheduled for a cesarean section at 38 weeks' gestation, and the couple was anxious about their child's condition and care following birth.

M.S. delivered a baby boy by caesarean section; he was transferred to the pediatric intensive care unit. On admission to the nursery, the baby's vital signs and weight were within normal limits, but the head circumference was large. He had bulging fontanelles and a high-pitched cry. The nurse noted a saclike projection in the lumbar region of his spine.

1. Discuss the rationale for delivering the infant by cesarean section.
2. Discuss the significance of the infant's clinical manifestations.
3. Discuss the complications associated with myelomeningocele.

- Maternal infections during pregnancy (e.g., rubella and varicella)
- Maternal exposure of toxins during pregnancy (e.g., mercury)
- Severe jaundice

Clinical manifestations of CP may or may not be evident at birth. These manifestations vary in severity from mild to severe. CP may affect the entire body (resulting in quadriplegia) or one area (resulting in diplegia); CP may affect one side or both sides of the body. Manifestations may include:

- Persistence of early reflexes (e.g., Moro reflex)
- Development delays
- Ataxia (lack of muscle coordination when performing voluntary movements)
- Spasticity (stiff muscles)
- Hyperreflexia (exaggerated reflexes)
- Asymmetrical walking gait, with one foot or leg dragging
- Unusual positioning of limbs when resting or when held up (e.g., scissor position of the legs)
- Excessive drooling
- Difficulties swallowing, sucking, or speaking
- Facial grimaces
- Tremors
- Difficulty with precise motions (e.g., writing and buttoning a shirt)

Complications of CP result because of these clinical manifestations and may include:

- Balance and coordination issues
- Contractures (shortening of a muscle causing severe limitation in movement)
- Malnutrition
- Communication issues and speech delays
- Learning or cognition difficulties
- Seizures
- Visual issues
- Urinary incontinence
- Chronic pain

Diagnostic procedures for CP include a history, physical examination, head CT, head MRI, and electroencephalogram (EEG) as well as hearing and vision screening. Treatment strategies focus on maximizing functioning and minimizing complications. Management is long term and requires a multidisciplinary team (e.g., the primary care provider, nurses, a social worker, a physical therapist, an occupational therapist, a speech therapist, a dietician, and education specialists). Strategies often include:

- Muscle relaxants
- Botulinum toxin type A (Botox) injections directly into spastic muscles
- Antiseizure medications
- Pain management (e.g., massage therapy and analgesics)
- Physical therapy
- Occupational therapy
- Speech therapy
- Braces and orthopedic devices (e.g., splints)
- Ambulation devices (e.g., walker and wheelchair)
- Surgical procedures to relieve contractures or to sever nerves of spastic muscles
- Support groups
- Individualized education program

Infectious Neurologic Disorders

Nervous system infections can have serious effects because of initiating the infectious and inflammatory response (see Chapter 2). These infections can be caused by a number of bacterial, viral, and fungal pathogens. Regardless of the causative agent, neurologic compromise (either temporary or permanent) can result. Early diagnosis and treatment is imperative for positive outcomes.

Meningitis

Meningitis refers to an inflammation of the meninges, usually resulting from an infection. The CSF may also become affected. Any number of bacteria (e.g., *Neisseria meningitidis*, *Streptococcus pneumoniae*, and *Haemophilus influenzae*) and viruses (e.g., enterovirus, measles, influenza, and herpes) can cause this infection. The infectious agents invade the meninges through the blood or nearby structures or by direct access (e.g., wounds). Additional causes of meningitis include tumors and allergens. The infection or irritant triggers the inflammatory process, leading to swelling

of the meninges and increased ICP. Risk factors for developing meningitis include being less than 25 years of age, living in a community setting (e.g., a college dormitory), pregnancy, working with animals, and immunodeficiency. Depending on the cause of the infection, meningitis can be self-limiting (as with viral) or life-threatening (as with acute bacterial). Complications of meningitis include permanent neurologic damage, seizures, hearing loss, blindness, speech difficulties, learning disabilities, behavior problems, paralysis, renal failure, adrenal gland failure, shock, and death.

Clinical manifestations of meningitis result from the inflammation of the meninges. Initially, these manifestations mimic an influenza infection (e.g., fever, chills, and malaise). Clinical manifestations usually develop over a couple of days and include:

- Fever and chills
- Mental status changes (e.g., confusion and lethargy)
- Nausea and vomiting
- Photophobia
- Severe headache
- Stiff neck (meningismus)
- Agitation
- Bulging fontanelle
- Decreased consciousness
- Opisthotonos (abnormal positioning that involves rigidity and severe arching of the back with the head thrown backward)
- Poor feeding or irritability in children
- Tachypnea (increased breathing)
- Rash

Diagnostic procedures of meningitis include a history, physical examination, throat cultures, lumbar puncture with CSF analysis, polymerase chain reaction test, and head CT. Treatment varies depending on the underlying etiology. Strategies may include antibiotics (if bacterial), hydration, and fever management. Vaccinations, including those for *Haemophilus influenzae*, pneumococcal, and meningococcal, are the cornerstone of meningitis prevention.

Encephalitis

Encephalitis refers to an inflammation of the brain and spinal cord, usually resulting from an infection. A virus (e.g., coxsackievirus, echovirus, poliovirus, adenovirus, herpes virus, cytomegalovirus, Eastern equine encephalitis virus, West Nile virus, St. Louis virus, measles, and mumps) most frequently causes this infection. Viral exposure occurs through respiratory inhalation of droplets, ingestion of contaminated food or beverages, insect bites (especially mosquitoes and parasites), and skin contact. Encephalitis can also result from bacterial infections such as Lyme disease, tuberculosis, and syphilis. The infection triggers the inflammatory response that causes vasodilatation, increased capillary permeability, and leukocyte infiltration. The inflammatory process can cause nerve cell degeneration and diffuse brain destruction. Encephalitis may be primary or secondary. Primary encephalitis involves direct viral infection of the brain and spinal cord. In secondary encephalitis, a viral infection first occurs elsewhere in the body and then travels to the brain.

Most cases of encephalitis are mild and self-limiting, but in rare cases, encephalitis can be severe and life threatening. Those particularly vulnerable to more severe progression of encephalitis include immune compromised persons (e.g., those with AIDS), young children, older adults, those living in high-incidence areas, and those frequently outdoors. Complications of encephalitis include cerebral edema, cerebral hemorrhage, and brain damage.

Clinical manifestations of encephalitis result from meningeal irritation and neurologic damage. These manifestations are similar to meningitis but with a more gradual onset. In most cases, clinical manifestations are mild and go undetected. When present, manifestations of encephalitis may include:

- Flulike symptoms (e.g., fever, lethargy, and joint pain)
- Headache
- Neck rigidity
- Confusion and hallucinations
- Personality changes (e.g., flat affect, impaired judgment, and withdrawal from social interactions)
- Diplopia
- Seizures
- Muscle weakness
- Paresthesia or paralysis
- Loss of consciousness
- Tremors
- Abnormal deep tendon reflexes

- Rash
- Bulging fontanelle (in infants)

Diagnostic procedures for encephalitis include a history, physical examination, head CT, head MRI, EEG, lumbar puncture with CSF analysis, polymerase chain reaction test, and serum viral antibodies. Encephalitis is usually self-limiting, so treatment is largely supportive. Treatment strategies often include:

- Rest
- Adequate nutrition, including plenty of liquids
- Reorientation and emotional support
- Analgesics and antipyretics to relieve headaches and fever
- Antiviral agents (if viral)
- Antibiotic therapy (if bacterial)
- Corticosteroids to reduce cerebral edema
- Antiseizure agents
- Sedatives to treat irritability and restlessness
- Physical, speech, and occupational therapy as necessary for any residual neurologic dysfunction

Many of the encephalitis causative organisms can be prevented. Prevention strategies include vaccinations, wearing protective clothing when outside (e.g., long-sleeve shirts), using mosquito repellant, and eliminating water sources around the home (e.g., standing water in containers).

Traumatic Neurologic Disorders

Traumatic neurologic disorders vary significantly in severity and presentation depending on the location and extent of damage. Even minor injuries can have substantial effects on neurologic functioning. Traumatic injuries to the nervous system can result from a number of events that cause physical damage (e.g., motor vehicle accidents, gunshot wounds, and falls). Commonly, a number of these traumatic conditions overlap and occur concurrently (e.g., subdural hematoma and increased intracranial pressure).

Brain Injuries

A **traumatic brain injury (TBI)** is usually caused by a sudden and violent blow or jolt to the head (called a closed injury) or a penetrating (known as an open injury) head wound that disrupts the normal brain function. However, not all blows or jolts to the head result in a TBI. With TBIs, the brain collides with the skull (**Figure 11-23**) and any penetrating objects (**Figure 11-24**). These events can bruise the brain, damage nerve fibers, and cause hemorrhaging. According to the CDC (2010c), the main sources of TBI are falls (25%), motor vehicle accidents (17%), penetration of an object (17%), and assaults (10%). TBIs vary from mild (e.g., a brief change in mental status or consciousness) to severe (e.g., an extended period of unconsciousness or amnesia after the injury). TBIs contribute to a substantial number of deaths and cases of permanent disability annually. According to the CDC (2010d), 1.7 million Americans sustain a TBI each year—50,000 of those die. Persons at highest risk for experiencing a TBI include:

- Males (twice as likely as females)
- Young children 0–4-year-olds and 15–19-year-olds
- Adults 75 years of age or older
- Certain military personnel (e.g., paratroopers)
- African Americans (highest death rates)

Many TBIs will result in a wide range of long-term and potentially life-altering complications such as changes in thinking, sensation, language, or emotions. TBIs can increase the risk for seizures, Alzheimer's disease, and Parkinson's disease. Multiple mild TBIs can have an accumulative effect and result in neurologic dysfunction, cognitive deficits, and death. This damage can be seen in the recent study regarding professional football players (Schwenk et al., 2007) and a growing number of evidence. These players, especially those who encounter routine impacts (e.g., linemen), had higher rates of cognitive deficits (e.g., memory impairment) and neurologic diseases (e.g., Alzheimer's disease, Parkinson's disease, and depression).

Closed TBIs often result in a couple of conditions. **Concussion** describes a momentary interruption of brain function. Concussions usually result from a mild blow to the head that causes sudden movement of the brain, disrupting neurologic functioning. Concussions may or may not lead to a loss of consciousness. Amnesia, confusion, sleep disturbances, and headaches may follow a concussion for weeks or months. **Cerebral contusion** refers to a bruising of the brain with rupture of small blood vessels and edema. Most contusions result from a blunt blow to the head that causes the brain to make sudden impact with the skull. The initial area the brain impacts with the skull is

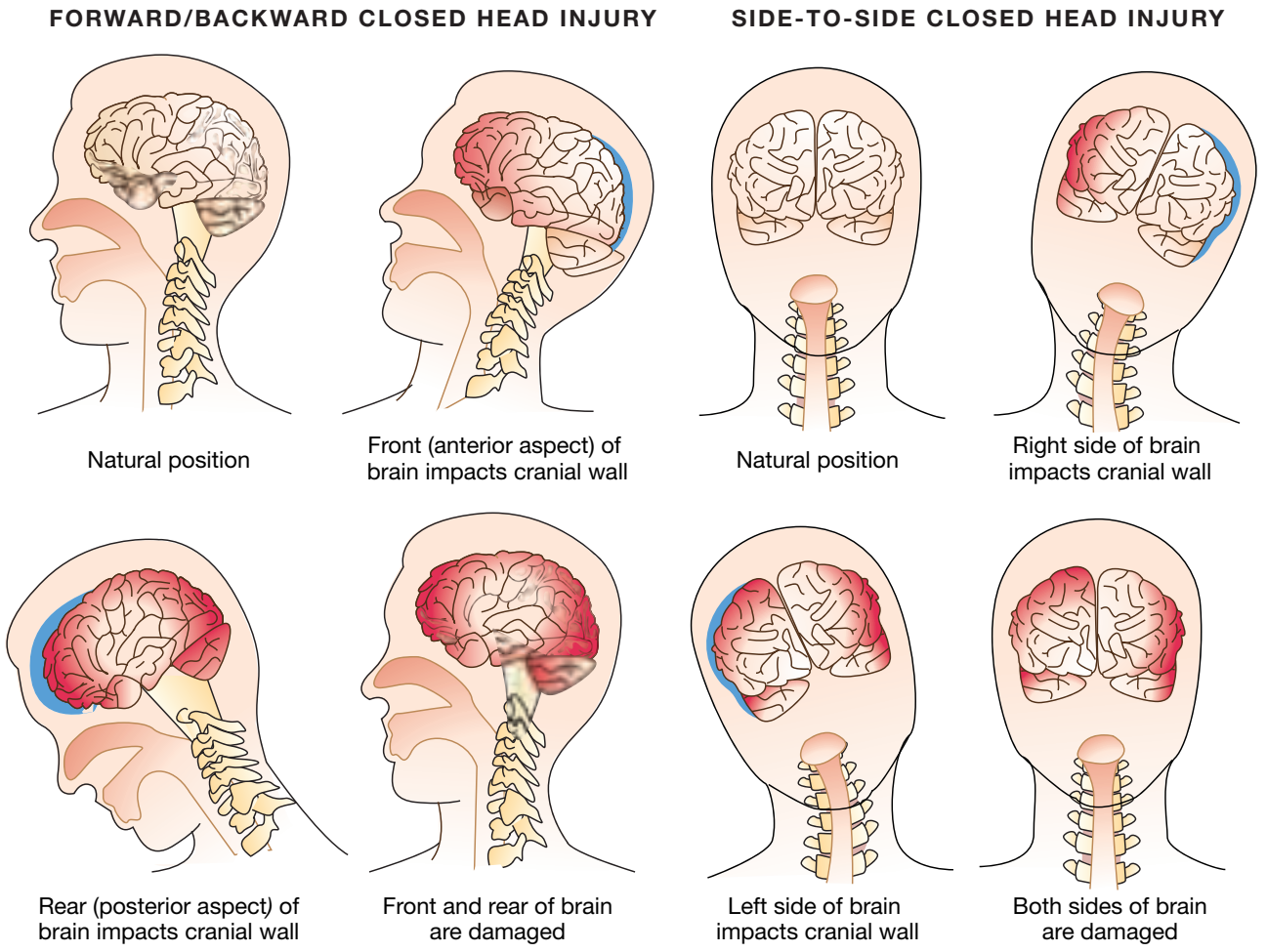


Figure 11-23

Closed traumatic brain injury.

referred to as the **coup**. The brain then rebounds and impacts with the opposite side of the skull, causing another area of damage referred to as the **countercoup** (**Figure 11-25**). Contusions vary in severity depending on the extent of damage and the amount of bleeding. The presence and severity of residual effects depend on that severity.

Open TBIs can result in serious issues. In addition to the tissue damage from the impact of the brain with the skull, open TBIs can cause damage from the penetrating object and skull fragments. The skull fractures as the object breaches it. Much like when an egg is broken, the skull usually ends up in multiple pieces when encountering an external force. A fracture may be a **linear skull fracture** (a simple crack), a **comminuted skull fracture** (several fracture lines), a **compound skull fracture** (a fracture where the brain tissue is exposed), a **depressed skull fracture** (the bone fragments are displaced into the brain), or a **basilar skull fracture** (located at the base of the skull and usually accompanied by CSF leakage). In addition to the brain damage from impact and penetrating objects, open TBIs are at higher risk for developing infections because of a break in the first line of defense (see Chapter 2). As discussed in previous sections, infections of the nervous system can have serious consequences.

Clinical manifestations of TBIs may be vague and develop slowly, or they may be sudden and severe. Symptoms may improve and then suddenly worsen. The outward appearance of the head is not an indication of the injury severity—serious injuries can occur with the skin and the skull intact. When a TBI is suspected, the individual should be asked to give an

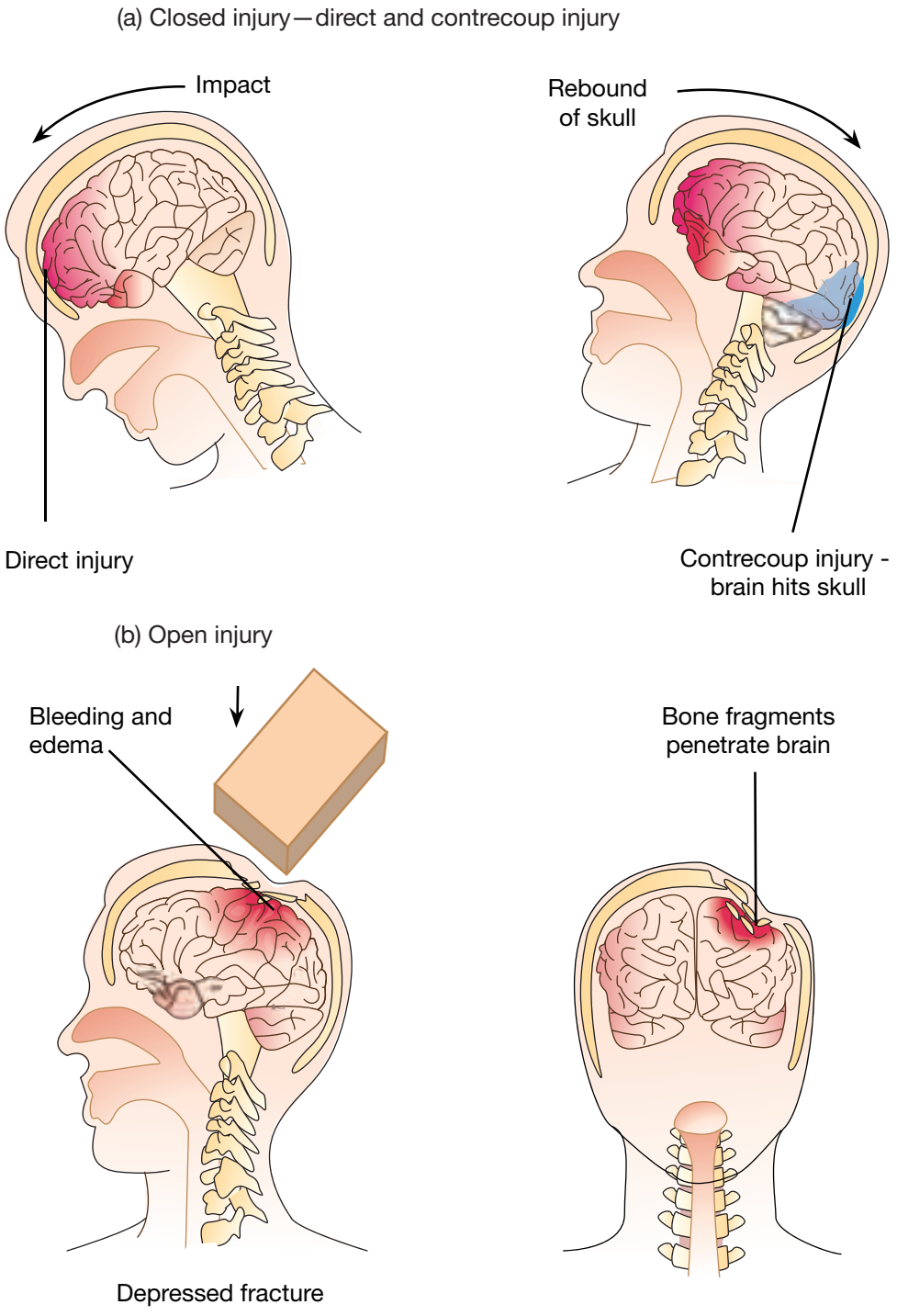


Figure 11-24

Open traumatic brain injury.

account of the accident. Not being able to recall details is an indication of a TBI. Additional clinical manifestations may include:

- Indications of a concussion (e.g., amnesia, confusion, and headache)
- Changes in or unequal size of pupils
- Seizures
- Asymmetrical facial features
- Fluid draining from the nose, mouth, or ears (may be clear or bloody; likely CSF)

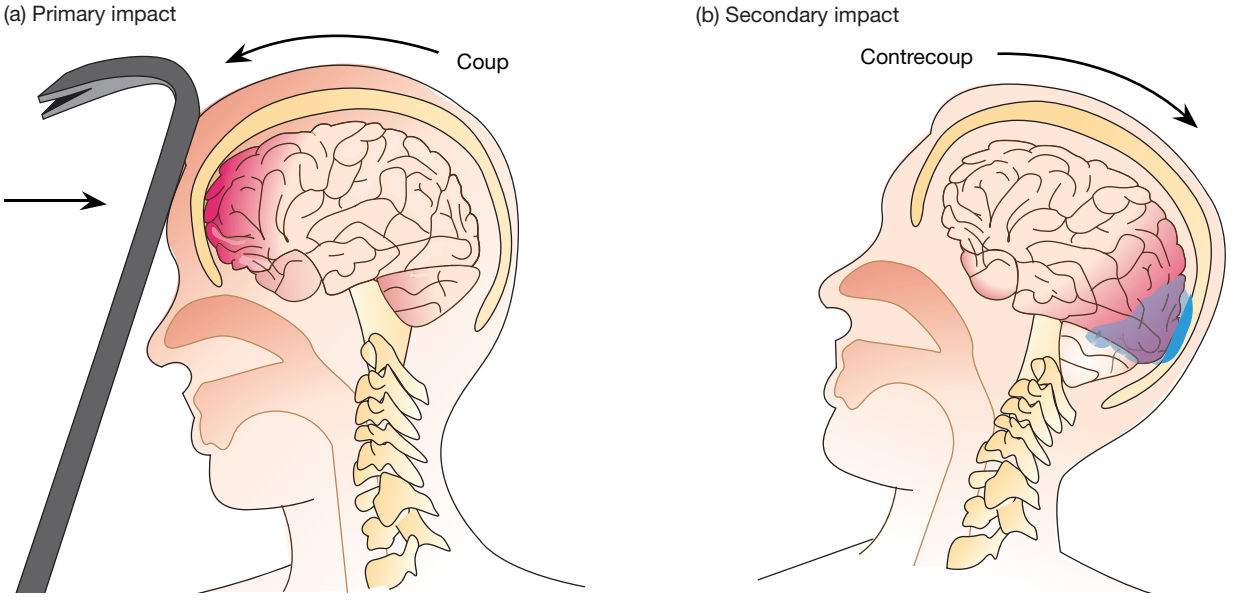


Figure 11-25

Coup and contracoup injuries.

- Fracture in the skull or face, bruising of the face, swelling at the site of the injury, or scalp wound
- Impaired hearing, smell, taste, speech, or vision
- Inability to move one or more limbs
- Irritability (especially in children), personality changes, or unusual behavior
- Loss of consciousness
- Bradypnea (slowed breathing)
- Hypotension
- Restlessness
- Lack of coordination
- Lethargy
- Stiff neck
- Vomiting

Diagnostic procedures for TBI consist of a history, physical examination (including using the Glasgow Coma Scale [**Figure 11-26**]), head CT, head MRI, and ICP monitoring. Treatment strategies vary depending on the severity and time since injury. Immediate emergency care for TBI focuses on limiting brain damage. Mild TBIs usually require no treatment other than rest and analgesics (specifically acetaminophen [Tylenol]) if headache is present. Nonsteroidal anti-inflammatory drugs, such as aspirin and ibuprofen (Motrin), should be avoided because they can increase bleeding risk. Cold compresses can be applied to any outward edema. Severe brain injuries usually require hospitalization and often need intensive care. Osmotic diuretics (e.g., mannitol) may be given to reduce cerebral edema.

LEARNING POINTS

Immediately following a head injury there are some key actions that should be avoided, including:

- Do *not* apply direct pressure to a bleeding site; cover a wound with sterile gauze.
- Do *not* wash a head wound that is deep or bleeding a lot.
- Do *not* remove any object sticking out of a wound.
- Do *not* move the person unless it is absolutely necessary.
- Do *not* shake the person if he or she seems dazed.
- Do *not* remove a helmet if you suspect a serious head injury.
- Do *not* pick up a fallen child with any sign of a head injury.
- Do *not* drink alcohol within 48 hours of a serious head injury

Parameter	Score	Response
Eye opening	Spontaneous	4
	To voice	3
	To pain	2
	No response	1
Best verbal response	Oriented, converses	5
	Disoriented, converses	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response or intubated	1
Best motor response	Follows commands	6
	Localizes response (pushes away stimulus)	5
	Withdraws	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1

Highest score = 15; lowest score = 3

Terms and Descriptive Behaviors for Levels of Consciousness

- Alert—Fully awake; aware of self and environment; appropriate, spontaneous response to stimuli.
- Confusion—Disoriented to person, time, and place (progresses from time to person to place); has difficulty following commands; may be agitated or irritable; may hallucinate.
- Delirium—disoriented to person, place and time; often agitated and uncooperative.
- Lethargy—Orientated to time, person, and place but somnolent; speech and thought processes slowed.
- Obtundation—decreased alertness accompanied by psychomotor retardation; can be aroused only with repeated verbal or tactile stimulation.
- Stupor—Awakens only to vigorous stimulation such as shaking; responds appropriately to painful stimuli; verbal responses are incomprehensible.
- Coma—Cannot be aroused; does not respond to verbal or tactile stimulation; brain stem reflexes may or may not be intact; may exhibit decerebrate or decorticate posturing.
- Light coma—Can be aroused; no spontaneous movement; withdraws appropriately to painful stimuli; brain stem reflexes (pupillary responses, gag, and corneal reflexes) are intact.
- Deep coma—Cannot be aroused; unresponsive to painful stimuli; absent brain stem reflexes; decerebrate posturing.

Figure 11-26

Glasgow Coma Scale.

Additionally, antiseizure agents and sedatives may be needed. Surgery can be performed to remove blood or repair fractures. Physical, speech, and occupational therapy may be required after the acute injury phase to minimize residual neurologic dysfunction. Prevention strategies for TBIs include wearing a seat belt when driving or riding in a motor vehicle, using appropriate child safety seats, wearing a helmet when appropriate (e.g., when playing sports, riding a bicycle, or skating), making the home safe (e.g., removing tripping hazards, having adequate lighting, using safety gates), storing firearms in locked cabinets, never driving impaired, and supervising children when playing.

Increased Intracranial Pressure

Increased intracranial pressure describes increased volume in the limited space of the cranial cavity. Increased ICP may occur because of a TBI as well as other conditions that would increase the volume in the skull (e.g., tumor, hydrocephalus, cerebral edema, and hemorrhage). The delicate pressure–volume relationship between ICP; volume of CSF, blood, and brain tissue; and cerebral perfusion is explained by the **Monro-Kellie hypothesis**. The Monro-Kellie hypothesis states that the cranial cavity cannot be compressed, and the volume inside the cavity is fixed (normal ICP is 60–200 mm H_2O or 4–15 mm Hg). The skull and its components (blood, CSF, and brain tissue) create a state of volume equilibrium, such that any increase in volume of one component must be compensated by a decrease in volume of another. This compensation is primarily accomplished by shifts in the CSF and, to a lesser extent, blood volume. These fluids respond to increases in volume of the remaining components. For example, an area of bleeding into the brain tissue (e.g., epidural hematoma) will be compensated by the downward displacement of CSF and venous blood. Transient increases in ICP routinely occur with position changes, coughing, or sneezing. These compensatory mecha-

nisms are able to maintain a normal ICP for changes in volume up to a point (approximately 100–120 mL of volume increases).

In addition to shifting volumes, the brain has two other compensatory mechanisms to maintain tissue perfusion—autoregulation and the Cushing's reflex. With **autoregulation**, the blood vessels dilate to increase blood flow and constrict if the ICP is increased. The **Cushing's reflex** is a complex cascade of events that results in increased blood pressure. When the mean arterial pressure (average blood pressure) drops below the ICP, the hypothalamus increases sympathetic stimulation. This stimulation causes vasoconstriction, increased cardiac contractility, and increased cardiac output. If unresolved, the increased ICP eventually leads to what is described as the **Cushing's triad**—increased blood pressure, bradycardia, and changes in respiratory pattern (**Figure 11-27**). Baroreceptors in the carotid arteries detect the increase in blood pressure, triggering a parasympathetic response through vagal stimulation that induces bradycardia. Bradycardia may also be stimulated by the increased ICP impinging on the vagal nerve, causing a parasympathetic response. As pressure escalates inside the skull, space becomes limited and the brain tissue shifts downward. An irregular respiratory pattern, called Cheyne-Stokes, and bradypnea typically result from increased pressure on the brain stem due to swelling, or from a brain stem herniation.

Herniation is a feared complication of increased ICP. **Herniation** refers to the displacement of brain tissue. There are several types of herniation (**Figure 11-28**). The cerebral hemispheres, diencephalon, and midbran displace downward in transtentorial (central) herniation. The pressure created by this herniation impairs the cerebral blood flow, CSF, reticular activation system, and respirations. Uncal (uncinate) herniation occurs when the uncus (a hooklike anterior end of the hippocampal gyrus) of the temporal lobe shifts downward past the tentorium cerebelli (the extension of the dura mater that separates the cerebellum from the inferior portion of the occipital lobes). This herniation creates pressure on cranial nerve III, the posterior cerebral artery, and the reticular activation system. Cerebellar, or tonsillar (intrafratentorial), herniation occurs when the cerebellar tonsils (rounded lobules on the undersurface of each cerebellar hemisphere) are pushed downward through the foramen magnum. This herniation compresses the brain stem and vital centers, causing death.

Regardless of the cause, increased ICP past the point of compensation compresses cerebral blood

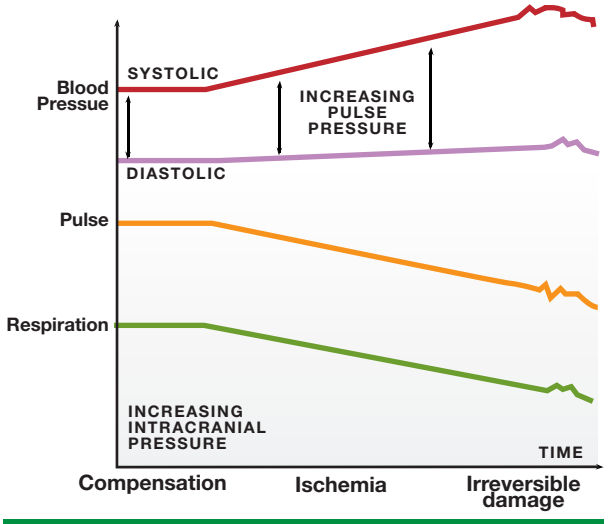


Figure 11-27

Vital sign changes with increased intracranial pressure.

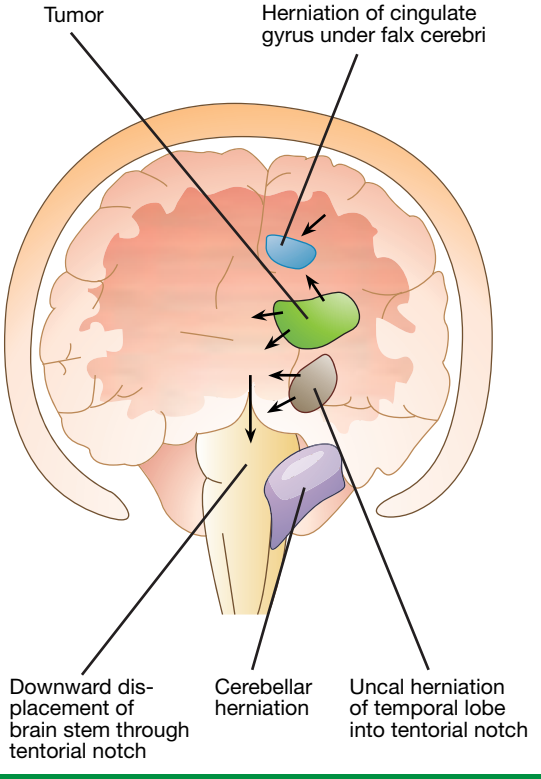


Figure 11-28

Types of herniation.

vessels and other structures as well as shifts content (**Figure 11-29**). Eventually, brain tissue dies. Increased ICP is a life-threatening situation that requires prompt treatment. If left untreated, increased ICP causes declining neurologic function, leading to death.

Clinical manifestations of increased ICP vary depending on age and reflect the effects of the rising pressures. These manifestations generally include:

- Decreasing level of consciousness (this results from pressure on the brain stem and cerebral cortex)
- Vomiting, often projectile (results from pressure on the medulla)
- Increasing blood pressure with increasing pulse pressure (the difference between systolic and diastolic pressure) (results of Cushing's reflex)

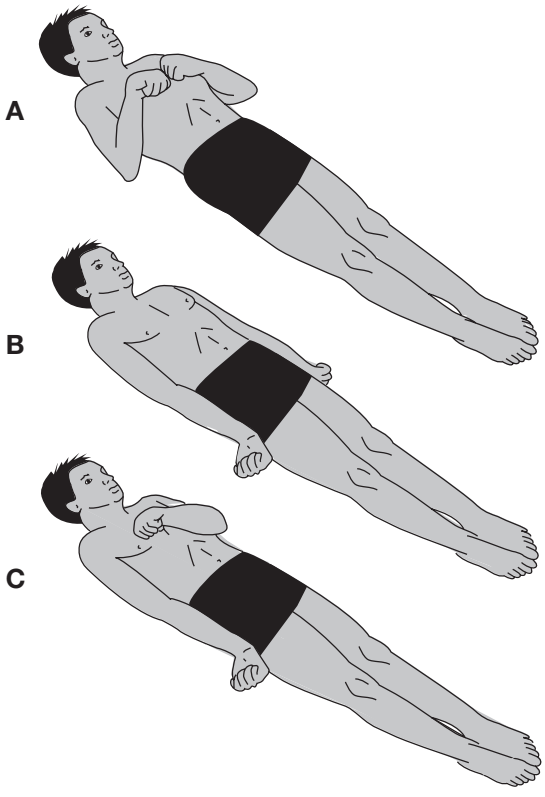


Figure 11-29

Decorticate and decerebrate posturing. (a) Decorticate response. Flexion of arms, wrists, and fingers with adduction in upper extremities. Extension, internal rotation, and plantar flexion in lower extremities. (b) Decerebrate response. All four extremities in rigid extension with hyperpronation of forearms and plantar extension of feet. (c) Decorticate response on the left side of the body and decerebrate response on the right side of the body.

- Bradycardia (response to the increasing blood pressure)
- Papilledema (results from increased pressure of CSF, which causes swelling around the optic disk)
- Fixed and dilated pupils (which results from pressure on cranial nerve III)
- Posturing

Manifestations in infants include:

- Separated sutures
- Bulging fontanelle

Manifestations in older children and adults include:

- Behavior changes
- Severe headache (results from stretching of the dura and walls of the large blood vessels)
- Lethargy
- Neurologic deficits
- Seizures

Diagnostic procedures for increased ICP consist of a history, physical examination (including completing the Glasgow Coma Scale), head CT, head MRI, and ICP monitoring. Increased ICP requires prompt diagnosis and treatment for optimal patient outcomes. Treatment strategies vary depending on the underlying etiology, and attempts should be made to resolve the source of pressure if possible (e.g., remove tumor or blood). Additional strategies are similar to those for TBIs and may include respiratory support (e.g., oxygen therapy or endotracheal intubation with mechanical ventilation), semi-Fowler's positioning, draining excess CSF, osmotic diuretics, corticosteriods, seizure precautions (e.g., low lighting and minimal stimulation), antiseizure agents, sedatives, stool softeners (because straining increases ICP), antiulcer agents (for those at high risk for stress ulcers), thermoregulation, and glucose management. Rarely, surgical removal of a skull segment may be performed.

Hematomas

Secondary brain damage can be caused by additional injurious factors such as hemorrhaging. A **hematoma** is a collection of blood in the tissue that develops from ruptured blood vessels. Hematomas can develop immediately or slowly because of a TBI or surgery. Hematomas are classified by their location (**Figure 11-30**).

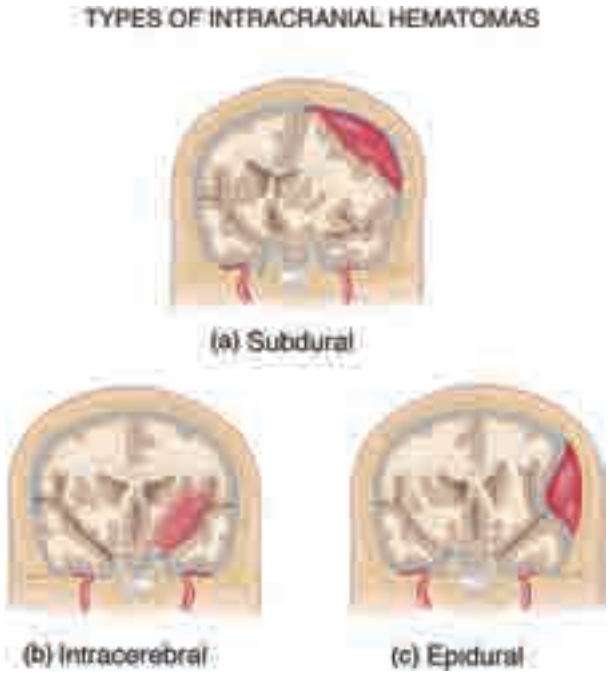


Figure 11-30

Types of hematomas. (a) Beneath the dura but outside the brain (subdural hematoma). (b) Within the substance of the brain tissue (intracerebral hematoma). (c) Outside the dura and under the skull (epidural hematoma).

Epidural hematomas result from bleeding between the dura and skull, usually caused by an arterial tear. Clinical manifestations of epidural hematomas include marked neurologic dysfunction that usually develops within a few hours of injury. The typical symptom pattern of an epidural hematoma is a brief loss of consciousness, followed by a short period of alertness, then loss of consciousness again. This pattern may not appear in all people. **Subdural hematomas** develop between the dura and arachnoid, frequently caused by a small venous tear (**Figure 11-31**). Because it is a result of a venous tear, subdural hematomas generally develop slowly. Subdural hematomas follow several patterns. With acute subdural hematomas, manifestations of neurologic deficits are present within 24 hours of an injury. This type progresses rapidly and has a high mortality. With subacute subdural hematomas, ICP increases over a period of about a week after the injury. With chronic subdural hematomas, manifestations develop several weeks after the injury because of a slow leak. Chronic subdural hematomas are more common in elderly adults because of brain atrophy, giving the hematoma more space to develop. **Intracerebral hematomas** result from bleeding in the brain tissue itself. Intracerebral hematomas are caused by

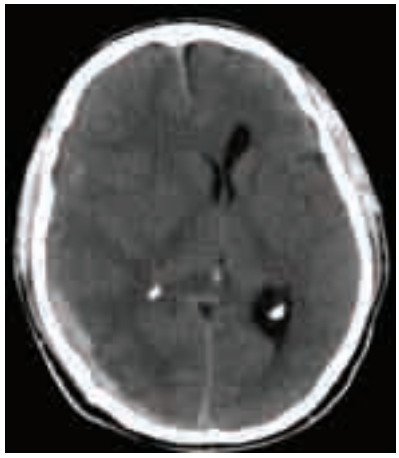


Figure 11-31

Midline shift associated with right-sided subdural hematoma.

contusion or shearing injuries but can also result from hypertension, cerebral vascular accidents (strokes), aneurysms, or vascular abnormalities. In addition to these hematomas, a **subarachnoid hemorrhage** results from bleeding in the space between the arachnoid and pia. The primary clinical presentation is a severe headache that has a sudden onset and that is worse near the back of the head.

In all types of hematomas, the bleeding leads to localized pressure on nearby tissue and increases ICP. Blood may coagulate and form a solid mass. The hematoma becomes encapsulated by fibroblasts, and blood cells within the capsule lyse. The fluid from the hemolysis exerts osmotic pressure, drawing more fluid into the capsule. This edema increases the size of the mass, applying pressure on the surrounding tissue and increasing ICP. Bleeding can trigger vasospasms, worsening ischemia. Additionally, increasing ICP can result in herniation.

Diagnostic procedures for all types of hematomas and hemorrhaging consist of a history, physical examination (including completing the Glasgow Coma Scale), head CT, head MRI, cerebral angiogram, and intracranial pressure monitor. Treatment strategies depend on the location and bleeding severity. No treatment may be required in mild cases in which the volume is small and the bleeding has ceased. Surgical removal of the blood through a burr hole or a craniotomy is often required. In some cases, removal of the blood may not be possible. In these cases, significant residual neurologic deficits that require physical, speech, and occupational therapy may remain. Additionally, strategies similar

to those for TBIs and increased ICP (e.g., respiratory management, seizure precautions, and thermoregulation) may be required.

Spinal Cord Injuries

Spinal cord injuries (SCIs) result from direct injury to the spinal cord or indirectly from damage to surrounding bones, tissues, or blood vessels. SCIs are often caused by motor vehicle accidents, falls, violence, and sports injuries. Minor injuries to the spinal cord can occur because of weakening vertebral structures (e.g., rheumatoid arthritis or osteoporosis). Direct damage can occur if the spinal cord is pulled, pressed sideways, or compressed (**Figure 11-32**). This damage may occur if the head, neck, or back twists abnormally during an accident or injury. Hemorrhage, fluid accumulation, and edema can occur inside or outside the spinal cord (but within the spinal canal). The accumulation of blood or fluid can compress the spinal cord and damage it. **Spinal shock** refers to a temporary suppression of neurologic function because of spinal cord compression. In spinal shock, neurologic function gradually returns. SCIs are most common in Caucasians and males, with 40.2 years being the average age at injury.

SCIs result in a significant loss of neurologic functioning, often requiring extensive, long-term management. SCIs can also result in death, either immediately or because of complications (e.g., pneumonia, embolism, or septicemia). The degree of dysfunction depends on the severity of the injury and the location (Figure 11-16). The injury may result in a partial or complete disruption of the neurons and neural tracts anywhere along the spinal cord. SCIs are classified based on the location of damage (e.g., C4, T12) and degree of function. An injury to one of the eight cervical segments of the spinal cord causes quadriplegia (tetraplegia)—loss of all or most function in all four limbs. Injury to the thoracic, lumbar, or sacral regions causes paraplegia—loss of lower extremity function. The individual may experience complete paralysis (no voluntary use of the affected limbs) or incomplete paralysis (some voluntary use of the affected limbs). Incomplete quadriplegia is the most frequently occurring injury (approximately 30%). The spinal cord does not extend beyond the first lumbar vertebra, so injuries at and below this level do not cause SCIs. However, they may cause **cauda equina syndrome** (injury to the nerve roots in the area of the cauda equina).Complications of SCIs are numerous and can contribute to mortality associated with SCIs. These complications may include:

- **Autonomic hyperreflexia** (a massive sympathetic response that can cause headaches, hypertension, tachycardia, seizures, stroke, and death; most commonly associated with injuries above T6)

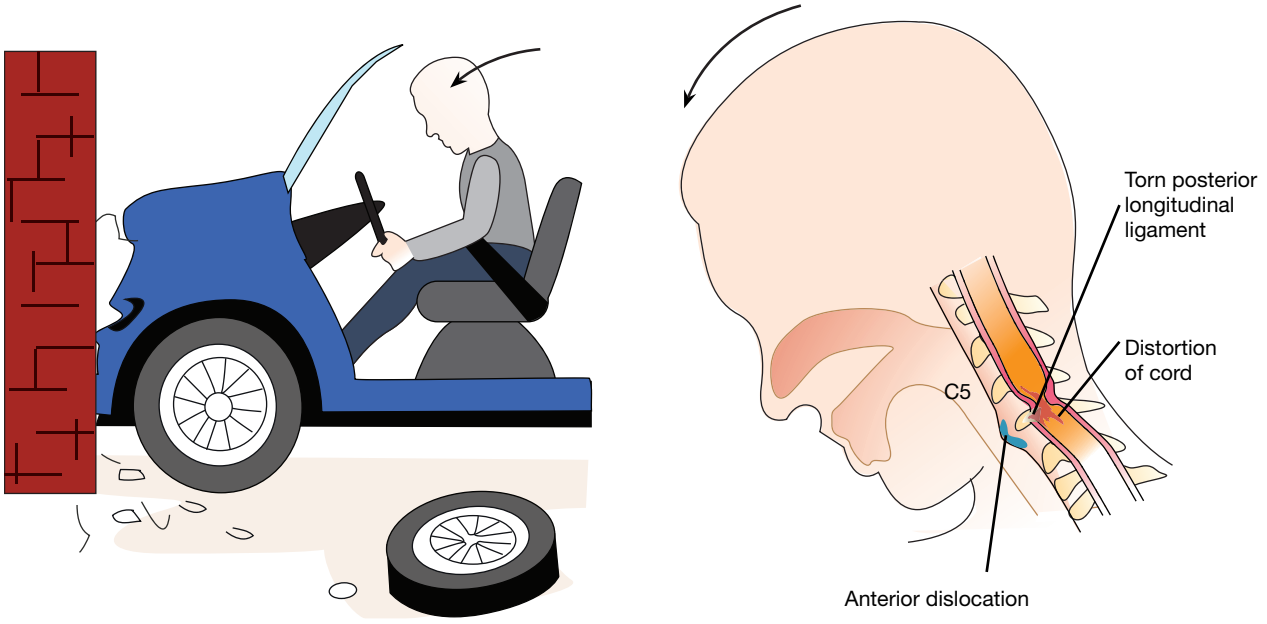


Figure 11-32

Spinal cord injuries.

- Neurogenic shock (an abnormal vasomotor response secondary to disruption of sympathetic impulses)
- Respiratory failure (caused by paralysis of the respiratory muscles)
- Effects of immobility (e.g., constipation, pulmonary infections, urinary infections, thrombus, impaired skin integrity, contractures)
- Changes in bowel and bladder function (e.g., urinary retention, incontinence, and constipation)
- Sexual dysfunction (e.g., erectile dysfunction)
- Chronic pain

Clinical manifestations of SCIs depend on the level of injury. Cervical injuries can affect both the upper and lower extremities. Manifestations of cervical injuries include:

- Breathing difficulties resulting from paralysis of the respiratory muscles
- Loss of normal bowel and bladder control (e.g., constipation, incontinence, and bladder spasms)
- Paresthesia
- Sensory changes
- Spasticity (increased muscle tone)
- Pain
- Weakness or paralysis
- Blood pressure instability
- Temperature fluctuations
- Diaphoresis

Thoracic injuries affect the lower extremities, and the symptoms can be the same as those for cervical injuries. Lumbar sacral injuries can affect the lower extremities in varying degrees. Manifestations of lumbar sacral injuries are similar to those of cervical injuries, with the exception of breathing difficulties.

Diagnostic procedures for SCIs consist of a history, physical examination (including a neurologic assessment), spinal CT, spinal MRI, spinal X-ray, and spinal myelogram (X-ray using contrast dye). SCIs are medical emergencies requiring immediate treatment. Strategies include immediate interventions to minimize residual effects and long-term interventions to limit complications. Immediate strategies may include:

- Immobilization of the spine
- Corticosteroid agents to reduce swelling
- Spinal traction to reduce the fracture and immobilize the spine
- Surgical repair of vertebral fractures or surgical removal of the fluid compressing the spinal cord (this is called decompression laminectomy)
- Respiratory management (e.g., oxygen therapy and endotracheal intubation with mechanical ventilation)
- Bed rest

Long-term strategies may include:

- Physical, occupational, and speech therapy
- Mobility assistive devices (e.g., a wheelchair)
- Long-term respiratory management (e.g., mechanical ventilation)
- Meticulous skin care
- Bowel and bladder training or management (e.g., catheterization and stool softeners)
- Antispasmotic agents and botulinum toxin type A (Botox) injections to treat muscle spasms
- Pain management
- Nutritional support
- Prompt treatment of infections (pneumonia is the leading cause of death)

Vascular Neurologic Disorders

Vascular neurologic disorders generally involve ischemic injuries to the brain resulting from occlusion of blood flow or hemorrhage. These disorders vary significantly in severity and presentation depending on the location and extent of damage. Often these disorders result in some degree of neurologic dysfunction. These conditions may occur due to congenital abnormalities or chronic diseases such as hypertension, hypercholesterolemia, and atherosclerosis.

Transient Ischemic Attack

A **transient ischemic attack (TIA)** refers to a temporary episode of cerebral ischemia that results in symptoms of neurologic deficits. TIAs are often called ministrokes because these neurologic deficits mimic a cerebral vascular accident (CVA) or stroke except that these deficits resolve within 24 hours (1–2 hours in most cases). TIAs may occur singly or in a series. TIAs are warning signs that a CVA may be impending; however, not all CVAs are preceded by a TIA. This ischemia can occur

because of a cerebral artery occlusion (e.g., thrombus, embolus, or plaque), cerebral arteries narrowing (e.g., atherosclerosis or spasms), or cerebral artery injury (e.g., inflammation or hypertension). Additional risk factors of TIAs include migraines, smoking, diabetes mellitus, advancing age, inadequate nutrition, hypercholesterolemia, oral contraceptive usage, excessive alcohol consumption, and illicit drug use. Complications of TIAs include permanent brain damage from the lack of oxygen and glucose, injury from falls, and CVA from the ischemia.

Clinical manifestations of TIAs begin suddenly and last for a short period. Within 24 hours, symptoms disappear completely. TIAs are not strokes, but the manifestations are the same. These manifestations reflect the location of the ischemia and may include:

- Muscle weakness or paralysis of the face, arm, or leg (usually unilateral)
- Paresthesia on one side of the body
- Aphasia (difficulty speaking) or receptive aphasia (difficulty understanding spoken language)
- Dysphagia (difficulty swallowing)
- Dysgraphia (difficulty writing)
- Difficulty reading
- Vision issues (e.g., diplopia, nystagmus, and partial or complete loss of vision)
- Changes in sensation (e.g., touch, pain, temperature, pressure, hearing, and taste)
- Change in levels of consciousness (e.g., lethargy, unconscious, or coma)
- Personality, mood, or emotional changes
- Confusion
- Agnosia (inability to recognize or identify sensory stimuli)
- Ataxia
- Vertigo (abnormal sensation of movement) or dizziness
- Incontinence of bowel or bladder

Because clinical manifestations often resolve prior reaching a healthcare facility, diagnosis may be made based on a history alone. Additional diagnostic procedures consist of a physical examination (including a neurologic assessment and blood pressure), head CT, head MRI, carotid ultrasound, cerebral arteriogram, EEG, serum clotting studies, blood chemistry, complete blood count, erythrocyte sedimentation rate test (can identify inflammatory process), and serum lipids test. Treatment strategies for TIAs focus on preventing the occurrence of a CVA. These strategies typically include managing any underlying conditions (e.g., hypertension, atherosclerosis, and diabetes mellitus). Medications, such as antiplatelet aggregation agents (e.g., aspirin and clopidogrel [Plavix]) or anticoagulants (e.g., warfarin [Coumadin]), may be used to prevent clotting. Angioplasty (balloon dilatation) can open narrowed arteries, or a carotid endarterectomy (surgical removal of plaque) can increase cerebral blood flow. Lifestyle management includes smoking cessation, minimizing dietary cholesterol and fat, increasing dietary fruits and vegetables, exercising regularly, limiting alcohol consumption, and eliminating illicit drug use.

Cerebral Vascular Accident

Much like a TIA, a **cerebral vascular accident (CVA)**, or stroke, refers to an interruption of cerebral blood supply (**Figure 11-33**). The chief difference between a CVA and TIA is that CVA damage is permanent. A CVA is an infarction of the brain, so it is often referred to as a brain attack. This interruption of blood flow may result from a total vessel occlusion (e.g., thrombus, embolus, or plaque) or cerebral vessel rupture (e.g., cerebral aneurysm, arteriovenous malformation, or hypertension); therefore, there are two major types of CVA—ischemic and hemorrhagic. **Ischemic strokes** are the most common (80%), and **hemorrhagic strokes** are the most deadly. Five minutes (sometimes less) of altered tissue perfusion can lead to irreversible cell damage from the lack of oxygen and glucose. CVA can result in significant neurologic dysfunction and death. CVA is the chief cause of long-term disability

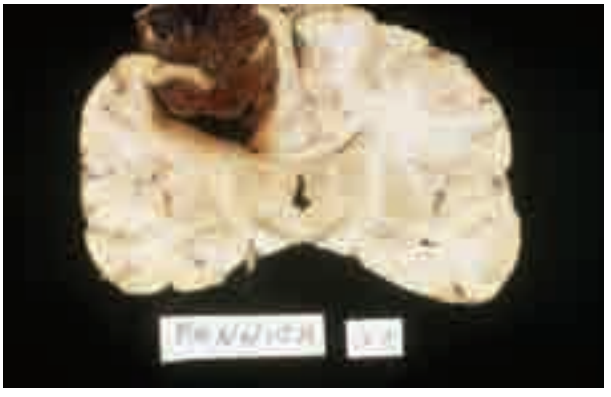


Figure 11-33

Cerebral vascular accident (CVA).

and third leading cause of death in the United States (CDC, 2009b). In the United States, someone experiences a CVA every 40 seconds. In 2009, the cost associated with this widespread problem with extensive consequences in the United States reached nearly $69 billion. CVA prevalence and mortality in the United States is highest among African Americans and those living in the Southeast. Additional risk factors include physical inactivity, obesity, hypertension, smoking, hypercholesterolemia, diabetes mellitus, atherosclerosis, oral contraceptive usage, excessive alcohol consumption, and illicit drug use.

Clinical manifestations of CVA are similar to those of a TIA except that CVA symptoms do not resolve. These manifestations may improve with time and therapy, but these manifestations can remain, creating complications. In addition to the manifestations of neurologic impairment associated with TIAs, headaches may be present with hemorrhagic strokes because of increasing ICP.

Diagnostic procedures for CVA consist of a history, physical examination (including a neurologic assessment), head CT, head MRI, carotid ultrasound, cerebral arteriogram, serum clotting studies, blood chemistry, and complete blood count. A CVA is a medical emergency that requires prompt treatment to minimize brain damage. Determining whether the CVA is ischemic or hemorrhagic in origin prior to treatment is crucial because the interventions vary depending on the type. Additionally, some interventions for ischemic strokes can worsen hemorrhagic strokes (e.g., thrombolytic agents). The differential diagnosis should be made as soon as possible because early treatment will improve outcomes. Optimally, treatment should be delivered within 3 hours of symptom onset; therefore, persons or family members of persons who seem to be experiencing a CVA should make note of when the symptoms began. Ischemic strokes are treated with thrombolytic agents (to dissolve any clots) and aspirin (to limit platelet activity). This treatment is contraindicated in persons with a recent history of bleeding issues. Additionally, procedures such as angioplasty or carotid endarterectomy may be necessary for ischemic strokes. Surgical repair of aneurysms or arteriovenous malformations as well as blood removal may be required for hemorrhagic strokes. Corticosteroids may also be administered with either type to reduce cerebral edema, and antihypertensive agents may be used to reduce blood pressure. A multidisciplinary approach (using a team made up of a nurse, physical therapist, speech therapist, occupational therapist, dietician, and social worker) should be initiated as soon as the patient is stable and may be required long term to minimize or prevent complications. Depending on the degree of dysfunction, strategies may be necessary to prevent complications of immobility (e.g., constipation, impaired skin integrity, contractures, and infections).

Cerebral Aneurysm

A **cerebral aneurysm** is a localized outpouching of a cerebral artery (see Chapter 4). This weakening of the artery may occur as a congenital defect or develop later in life because of conditions such as hypertension, connective tissue diseases (e.g., Marfan syndrome), TBIs, and arterial wall infections (**Figure 11-34; Figure 11-35**). This bulging artery segment can put pressure on surrounding tissue. Additionally, the aneurysm may leak or rupture, causing a CVA or death. There are several types of aneurysm, but most cerebral aneurysms are berry or saccular. Cerebral aneurysms most frequently occur in multiples on the circle of Willis.

Many cerebral aneurysms are asymptomatic until they grow large enough to compress surrounding structures or ruptures. Clinical manifestations that may appear as the aneurysm compresses nearby structures include vision issues (e.g., diplopia and loss of vision), headache, eye pain, or neck pain. A sudden, severe headache is an indication that the aneurysm has ruptured. Additional manifestations resemble those of increased ICP and CVA.

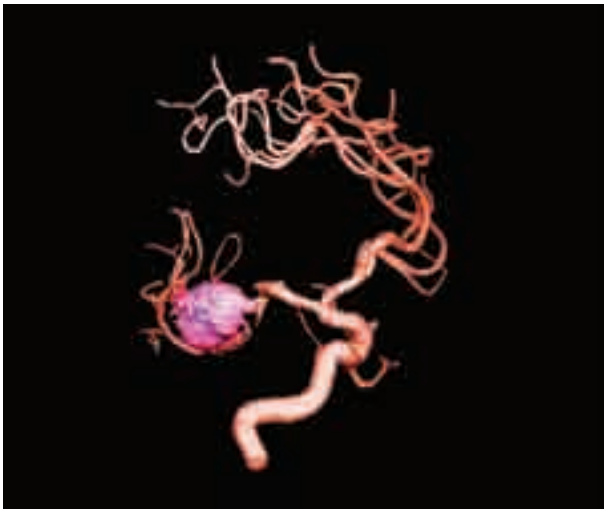


Figure 11-34

Cerebral aneurysm.

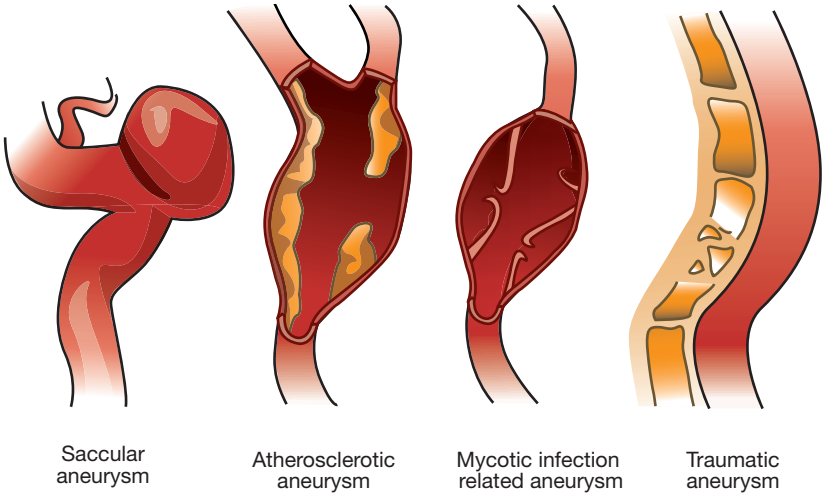


Figure 11-35

Types of cerebral aneurysms.

Often diagnosis occurs inadvertently with a head CT or MRI. Additional diagnostic procedures include a history, physical examination, cerebral arteriography, and EEG. If discovered prior to rupture, treatment strategies include surgical repair (if possible) and managing contributing factors (e.g., hypertension). Rupture is a medical emergency that requires immediate surgical repair. Additional strategies are similar to those for a CVA and subarachnoid hemorrhage.

Seizure Disorders

A **seizure** is a transient physical or behavior alteration that results from an abnormal electrical activity in the brain. Mechanisms that may be responsible for this abnormal electrical activity include altered membrane ion channels, altered extracellular electrolytes, and imbalanced excitatory and inhibitory neurotransmitters. Some neurons are hypersensitive or remain in a partial state of depolarization, increasing excitability. Seizures can occur secondary to trauma, hypoglycemia, electrolyte disorders, acidosis, infection, tumors, or chemical ingestion (e.g., medications, illicit drugs, and alcohol). Additionally, seizures can occur as a disorder referred to as **epilepsy**. Epilepsy results from spontaneous firing of abnormal neurons and is characterized by recurrent seizures for which there is no underlying or correctable cause. According to the CDC (2010b), epilepsy affects about 2 million Americans. About 10% of people will experience a seizure sometime during their lifetime, and about 3% will have had a diagnosis of epilepsy by age 80. Complications of seizures include brain damage, TBIs, aspiration, mood disorders, and **status epilepticus** (seizures that last longer than 20 minutes or subsequent seizures occur before the individual has fully regained consciousness).

Seizures can be classified into two broad categories—focal and generalized. **Focal seizures**, also called partial seizures, occur in just one part of the brain. About 60% of people with epilepsy have focal seizures. These seizures vary depending on the area of the brain affected, and they are frequently described by the area of the brain in which they originate (**Figure 11-36**). In a simple focal seizure, the individual having the seizure remains conscious but experiences unusual feelings or sensations that can take many forms. The individual may experience sudden and unexplainable feelings of joy, anger, sadness, or nausea. Additionally, the individual may hear, smell, taste, see, or feel things that are not real. In a complex focal seizure, the individual has changes in or loss of consciousness, producing a dreamlike experience. People having a complex focal seizure may display strange, repetitious behaviors (e.g., blinking, twitching, moving one's mouth, walking in a circle) called **automatisms**. These seizures usually last just a few seconds. Some people with focal seizures, especially complex focal seizures, may experience **auras** (unusual sensations just prior to an impending seizure). These auras are actually simple focal seizures in which the person maintains consciousness. The symptoms an individual has and the progression of those symptoms tend to be similar with every seizure. The symptoms of focal seizures can easily be confused

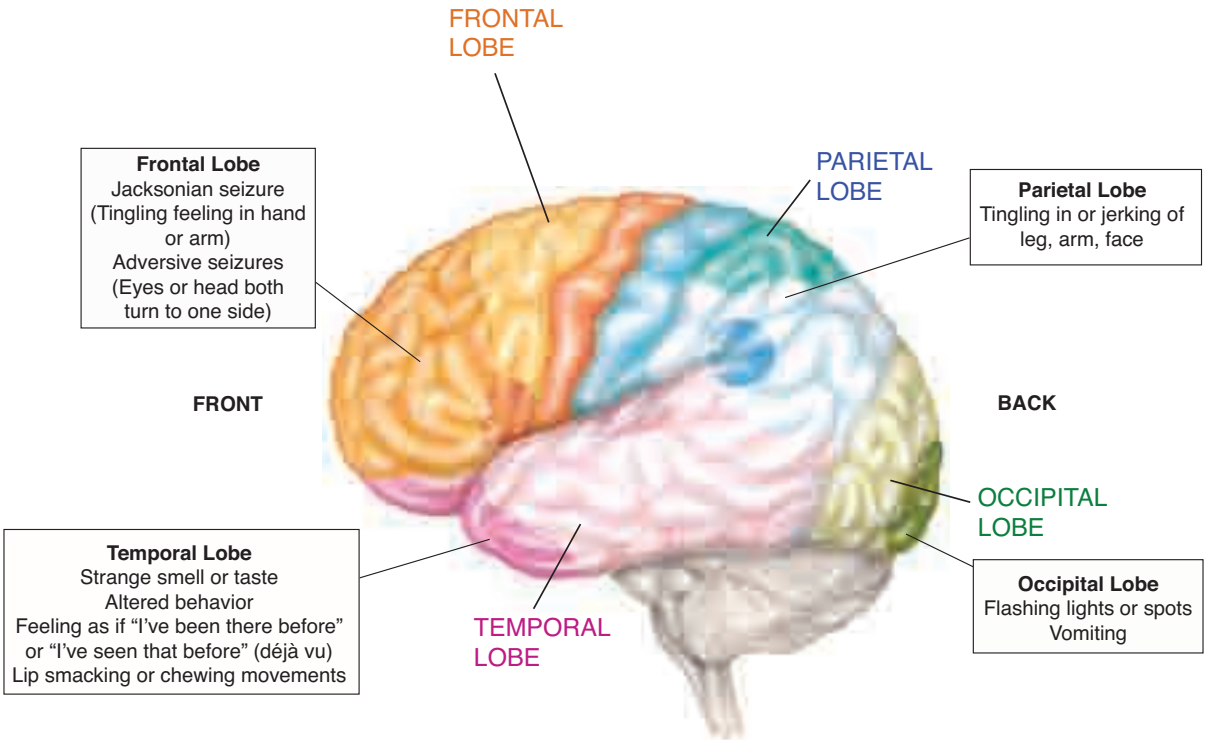


Figure 11-36

Manifestations of focal seizures depending on the regions of the brain.

with other disorders (e.g., migraine headaches, narcolepsy, syncope, and psychiatric disorders), so those disorders should be ruled out.

Generalized seizures are a result of abnormal neuronal activity on both sides of the brain. These seizures may cause loss of consciousness, falls, or massive muscle spasms. There are many kinds of generalized seizures. A person having an absence seizure (previously called a petit mal seizure) may appear to be staring into space and/or have jerking or twitching muscles (**Figure 11-37**). Tonic seizures cause stiffening of muscles of the body, generally those in the back and extremities. Clonic seizures cause repeated jerking movements of muscles on both sides of the body. Myoclonic seizures cause jerks or twitches of the upper body, arms, or legs (**Figure 11-38**). Atonic seizures cause a loss of normal muscle tone. The affected person will fall down or may drop his or her head involuntarily. Tonic-clonic seizures (previously called grand mal seizures) cause a mixture of symptoms, including stiffening of the body and repeated jerks of the arms and/or legs as well as loss of consciousness (**Figure 11-39**). The individual having a generalized seizure may be confused, fatigued, and fall into a deep sleep following the seizure; the time during which these manifestations occur is referred to as the **postictal period**. Not all seizures can be easily defined as either focal or generalized. Some people have seizures that begin as focal seizures but then spread to the entire brain. Other people may have both types of seizures but with no clear pattern.

Diagnostic procedures for seizure disorders consist of a history (including a description of the seizure activity if possible), physical examination, head CT, head MRI, head positron emission tomography (PET), and EEG. Treatment focuses on preventing the occurrence and limiting the duration of the seizure activity. Treatment strategies can be grouped into two categories—those to manage acute seizures and those to prevent seizures. Most seizures resolve spontaneously within a few minutes, but employing safety precautions can prevent injury. During a seizure, positioning the individual on his or her side can prevent aspiration (vomiting is common). Additionally, the head should be protected. Items should not be forced in between the individual's teeth; it is more likely to cause harm than to help. Attempts should not be made to restrain the individual; this also is more likely to cause injury. Airway management and oxygen therapy may also be

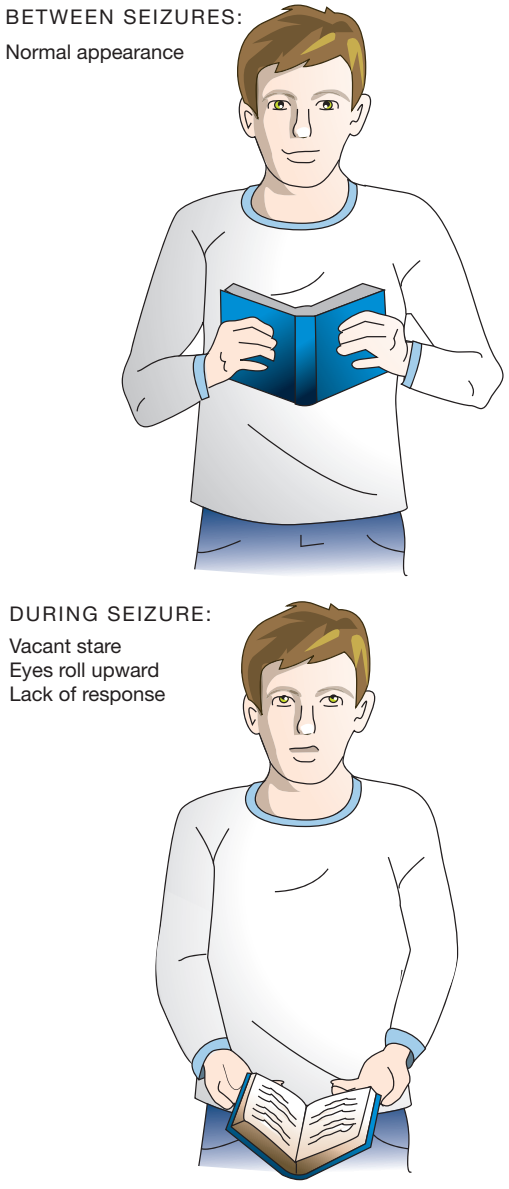


Figure 11-37

Absence seizures.

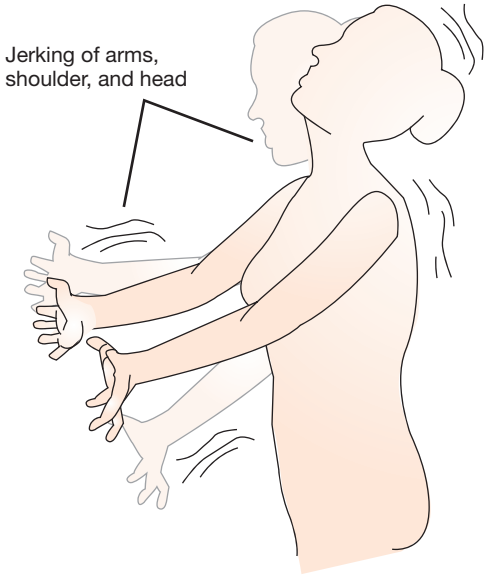


Figure 11-38

Myoclonic seizures.

necessary to minimize hypoxia. If status epilepticus develops, medication (e.g., muscle relaxants, antiseizure agents) will often be administered intravenously to stop the seizure. Following a seizure, the individual should be allowed to sleep as desired. For epilepsy, antiseizure agents will be administered daily to minimize the frequency and duration of seizure activity. These medications require close monitoring and accurate administration to ensure therapeutic dosing and limit side effects. If medications are not successful in controlling seizure activity, surgical resection or transaction of the region in which the abnormal electrical activity originates might be necessary. Additionally,

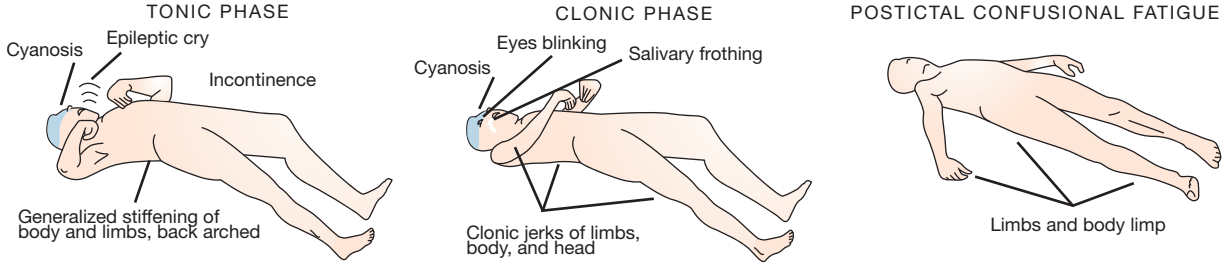


Figure 11-39

Tonic-clonic seizures.

persons with seizure disorders should wear a medical-alert bracelet and avoid precipitating factors (e.g., sleep deprivation, alcohol, illicit drugs, and excessive stimuli).

Chronic Degenerative Disorders

Chronic degenerative disorders of the nervous system include those conditions in which neurologic function deteriorates over time. These conditions usually result in significant neurologic dysfunction that requires lifelong management. These disorders are not usually preventable, and often treatment options are limited.

Multiple Sclerosis

Multiple sclerosis (MS) is a debilitating autoimmune condition that involves a progressive and irreversible demyelination of brain, spinal cord, and cranial nerves neurons. This damage occurs in diffuse patches throughout the nervous system and slows or stops nerve impulses. The progression of this damage varies from person to person. Like most autoimmune disorders, the underlying cause is unknown. According to the National Institutes of Health (2010b), approximately 300,000 Americans have MS. The prevalence rates are the highest among women, Caucasians, and those living in temperate climates. The onset of symptoms usually occurs between 20 and 40 years of age. Complications of MS include epilepsy, paralysis (most often the legs), and depression.

Clinical manifestations of MS vary depending on the degree of damage and the specific nerves affected; however, MS is characterized by remissions and exacerbations. Exacerbations may last for days to months. Fever, hot baths, sun exposure, and stress can trigger or worsen these episodes. Although remissions and exacerbations are common, the disease may continue to progress without remissions. Clinical manifestations include:

- Fatigue
- Loss of balance
- Muscle spasms
- Paresthesia or abnormal sensation in any area
- Problems moving arms or legs
- Weakness in one or more arms or legs
- Unsteady gait
- Lack of coordination
- Tremor in one or more arms or legs
- Constipation and stool leakage
- Urinary frequency, urgency, hesitancy, or incontinence
- Vision issues (e.g., diplopia and vision loss)
- Decreased attention span, poor judgment, and memory loss
- Difficulty reasoning and solving problems
- Dizziness
- Hearing loss
- Sexual dysfunction
- Slurred speech
- Dysphagia

There is no definitive test for MS, which can delay diagnosis. Diagnostic procedures for MS may consist of a history, physical examination (including a neurologic assessment), MRI studies (brain and spinal cord), lumbar puncture with CSF analysis (this often shows high levels of protein, gamma globulin, and lymphocytes), and nerve conduction studies. No cure for MS exists; however, treatment can slow the progression. Treatment strategies focus on minimizing symptoms and maximizing quality of life. These strategies include medications such as corticosteroids (treats exacerbations), interferons (slows damage), and immunemodulators (suppresses immune response). Additionally, physical and occupational therapy along with assistive devices (e.g., wheelchair, walkers, and handrails) can maximize functioning. Coping strategies, support, proper nutrition, and adequate rest can promote and maintain overall health.

Parkinson's Disease

Parkinson's disease is a progressive condition involving the destruction of the substantia nigra in the brain. This destruction results in a lack of dopamine, a chemical messenger that allows smooth, coordinated muscle movement. When approximately 80% of the dopamine-producing cells are destroyed, movement issues that typically include tremors (involuntary shaking) of the hands and head develop. The tremors may disappear or decrease when the body part is moved intentionally. The cause of Parkinson's disease is unknown. According to the NIH (2010c), approxi-

mately 500,000 Americans have been diagnosed with Parkinson's disease, and prevalence rates are evenly distributed across gender, social, ethnic, geographic, and economic groups.

Clinical manifestations of Parkinson's disease vary depending on the degree of dopamine deficit. These manifestations often include (**Figure 11-40**):

- Slowing or stopping of automatic movements (e.g., blinking)
- Constipation
- Dysphagia
- Drooling
- Unsteady gait
- Masklike appearance to face
- Myalgia
- Problems with movement, including the following:
  - Difficulty initiating or continuing movement (e.g., walking or getting out of a chair)
  - Loss of fine hand movements (writing may become small and difficult to read; eating can become more difficult)
  - Shuffling gait
  - Slowed movements
- Rigid or stiff muscles (often beginning in the legs)
- Tremors
  - Tremors usually occur in the limbs at rest or when the arm or leg is held out
  - Tremors go away during purposeful movement
  - Eventually, tremors can be seen in the head, lips, tongue, and feet
  - Tremors may be worse when the affected individual is tired, excited, or stressed
  - Finger-thumb rubbing (called "pill-rolling" tremor) may be present
- Slowed, quieter speech with monotone voice
- Stooped position
- Anxiety, stress, and tension
- Confusion
- Dementia
- Depression
- Syncope
- Hallucinations
- Memory loss
- Seborrhea (oily skin)

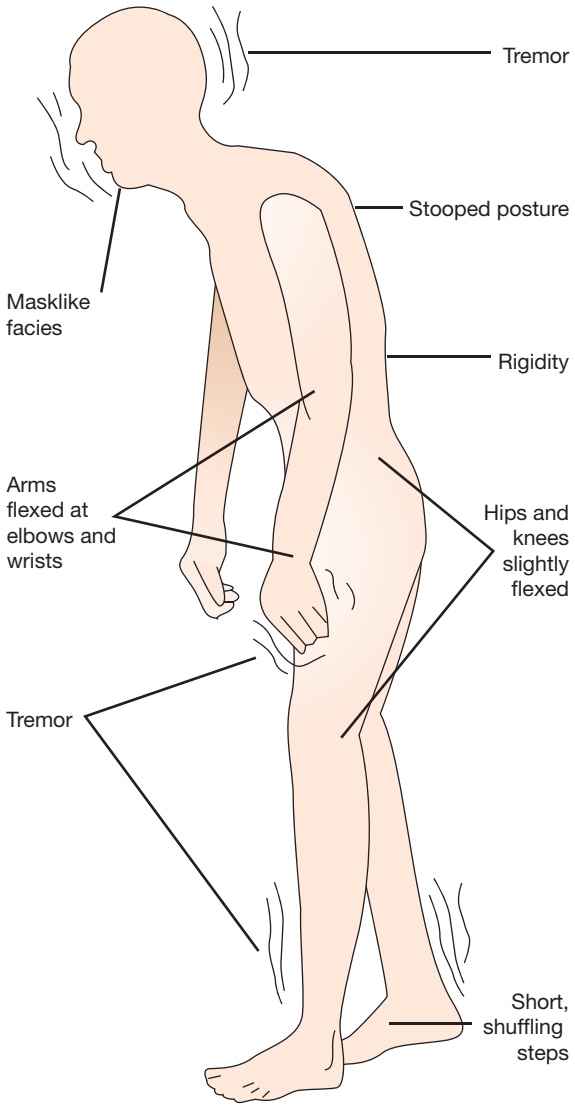


Figure 11-40

Clinical presentation of Parkinson's disease.

Much like MS, Parkinson's disease does not have a definitive test. Diagnostic procedures consist of a history, physical examination (including neurologic assessment), and other tests to rule out other conditions. There is no cure for Parkinson's disease. The goal of treatment is to control symptoms. Medications (e.g., levadopa and dopamine agonists) can increase the levels of dopamine, but the effects of the medications often diminish over time, requiring dose increases.

Medications may reach maximum dosing, and symptom control will be lost. Deep brain stimulation is a common surgical treatment for Parkinson's disease. Additionally, physical and occupational therapy along with assistive devices (e.g., wheelchair, walkers, and handrails) can maximize functioning. Coping strategies, support, proper nutrition, and adequate rest can promote and maintain overall health.

Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS), also called Lou Gehrig's disease after the famous baseball player who died of it, is a disease that involves damage of the upper motor neurons of the cerebral cortex and lower motor neurons of the brain stem and spinal cord. Sensory neurons, cognitive function, and cranial nerves III, IV, and VI are not affected. The nerves lose their ability to trigger muscle movement, resulting in muscle weakness, disability, paralysis, and eventually death (usually within 5 years of onset of symptoms). ALS may also increase the risk for dementia. In most cases, the cause of ALS is undetermined, but genetics plays a role in 10% of cases. Researchers are exploring several possible causes of ALS. The first possible cause is free radical damage (see Chapter 1). The inherited form of ALS often involves a mutation in a gene responsible for producing a strong antioxidant enzyme that protects cells from damage caused by free radicals. The second possible cause being explored is glutamate's influence. People who have ALS typically have higher than normal levels of glutamate, a chemical messenger in the brain, in their CSF. Too much glutamate is toxic to some nerve cells. Finally, possible autoimmune responses are being studied as a possible trigger for ALS.

The exact number of cases in the United States is unknown, but the NIH (2010a) estimates that 20,000 Americans have ALS. Though this condition is not necessarily common, ALS is a public concern because there is no means to preventing the continuous and rapid decline in motor function. In 2010, a National ALS registry was launched to collect, manage, and analyze information about people with ALS. This registry will provide information that will illuminate the scope and epidemiology of the problem as well as guide practice and research.

Clinical manifestations of ALS become progressively worse as more motor neurons are damaged. The loss of upper motor neurons results in spastic paralysis and hyperreflexia, and the loss of lower motor neurons results in flaccid paralysis. Early manifestations of ALS include:

- Footdrop (difficulty lifting the front of the foot and toes)
- Lower extremities weakness
- Hand weakness or clumsiness
- Slurred speech or dysphagia
- Muscle cramps and twitching in upper extremities and the tongue

The disease frequently begins in the upper or lower extremities and then spreads to other parts of the body. As the disease advances, muscles become progressively weaker until they are paralyzed. ALS eventually affects chewing, swallowing, speaking, and breathing.

Like other degenerative neurologic disorders, there is no definitive test for ALS. Diagnostic procedures are often used to rule out other conditions. These procedures consist of a history, physical examination (including a neurologic assessment), electromyogram (an electrode is inserted into the muscles to measure electrical activity), nerve conduction studies, MRI studies (head and spinal cord), lumbar puncture with CSF analysis, and muscle biopsy. ALS has no cure. Treatment strategies focus on slowing the progression and controlling symptoms. Riluzole (Rilutek), a benzothiazole, is the only medication approved by the Food and Drug Administration for slowing ALS. The drug appears to slow the disease's progression in some people, perhaps by reducing levels of glutamate. Additionally, stem cell therapy is being explored as a possible treatment. Antispasmodic agents may be given to treat muscle spasms. Physical, occupational, and speech therapy, along with assistive devices (e.g., wheelchairs and braces) can maximize muscle function. Because of aspiration and dysphagia risk, nutritional support including high-caloric foods, soft or pureed foods, thickened liquids, and parenteral feedings will become critical to maintaining optimal health as muscles weaken. Respiratory management (e.g., oxygen therapy, pulmonary hygiene, respiratory treatments, and mechanical ventilation) will become necessary as muscle weakness progresses. Coping strategies and support for the patient and caregivers can be helpful as the condition worsens.

Myasthenia Gravis

Myasthenia gravis is an autoimmune condition in which acetylcholine receptors are impaired or

destroyed by IgG autoantibodies. This acetylcholine receptor compromise leads to a disruption of normal communication between the nerve and muscle at the neuromuscular junction. This disruption causes weakness of the voluntary skeletal muscles because of inadequate nerve stimulation. Muscle weakness typically increases during periods of activity and improves after periods of rest. Muscles that control eye and eyelid movement, facial expression, chewing, talking, and swallowing are often, but not always, involved in the disorder. The muscles that control breathing and neck and limb movements may also be affected. Myasthenia gravis is common (2–3 cases per 10,000 people) and affects gender, ethnic, and age groups equally. The exact trigger for the autoimmune response is unclear, but the thymus gland is thought to play a role. Persons with myasthenia gravis often have a thymus gland abnormality (e.g., hyperplasia and tumors). Some factors can worsen myasthenia gravis and cause a **myasthenic crisis**, including fatigue, illness, stress, extreme heat, alcohol consumption, and certain medications (e.g., beta blockers, calcium channel blockers, quinine, and some antibiotics). Myasthenic crisis is a potentially life-threatening complication, which occurs when the muscles become too weak to maintain adequate ventilation.

Clinical manifestations of myasthenia gravis reflect the muscle weakness. These manifestations may include:

- Breathing difficulty
- Dysphagia
- Difficulty climbing stairs, lifting objects, or rising from a seated position
- Dysarthria
- Drooping head
- Facial paralysis or weakness
- Fatigue
- Hoarseness or changing voice
- Eye and vision issues (e.g., diplopia, ptosis, blurred vision, and difficulty maintaining gaze)

Diagnosis of myasthenia gravis is primarily made on clinical presentation. Diagnostic procedures consist of a history, physical examination (including a neurologic assessment), edrophonium test (a short-acting anticholinesterase inhibitor called edrophonium is injected and a sudden, although temporary, improvement in muscle strength indicates possible myasthenia gravis), serum antibody levels, nerve conduction study, electromyogram, and thymus CT or MRI. Myasthenia gravis has no cure, but treatment strategies can manage symptoms. Medications used to treat the disorder include anticholinesterase agents, which help improve neuromuscular transmission and increase muscle strength. Immunosuppressive drugs may also be used; these medications improve muscle strength by suppressing the production of abnormal antibodies. Other therapies include a thymectomy, plasmapheresis (removal of abnormal antibodies from the blood), and high doses of immunoglobulins. Additional self-care strategies to maximize health and functioning include proper nutrition, adequate rest, assistive devices, coping strategies, and support.

Huntington's Disease

Huntington's disease (HD), or Huntington's chorea, is a condition caused by a genetically programmed degeneration of neurons in the brain. HD is an autosomal dominant disorder (see Chapter 1) involving a defect on chromosome 4. The defect causes a segment of DNA, called a CAG repeat, to occur many more times than usual. Normally, this section of DNA is repeated 10–35 times within the DNA coding sequence, but it is repeated 36–120 times in persons with HD. This defect leads to progressive atrophy of the brain, particularly in the basal ganglia and the frontal cortex (**Figure 11-41**). The ventricles dilate, gamma-aminobutyric acid levels diminish, and acetylcholine levels fall. As the gene is transmitted from one generation to the next, the number of repeats (called CAG repeat expansion) tends to increase. With a larger number of repeats, the chance of developing symptoms at an earlier age increases. Therefore, as the disease is transmitted in families, it becomes evident at younger and younger ages. The

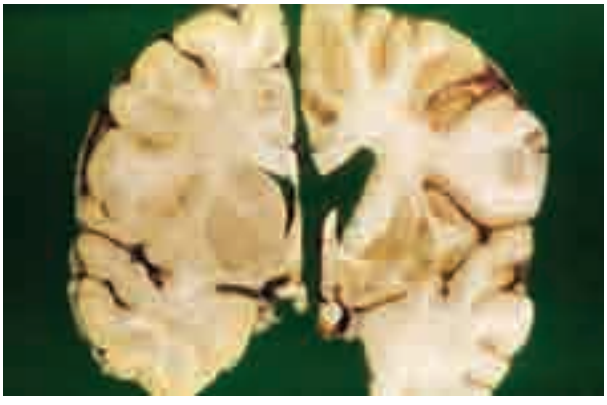


Figure 11-41

Neurologic changes of Huntington's disease.

earlier HD symptoms appear, the faster it progresses. Most cases of HD appear between 30 and 40 years of age, but HD may appear in childhood or adolescence in a small number of cases. In general, the duration of the illness ranges from 10 to 30 years. The most common causes of death are infection (most often pneumonia), injuries related to a fall, or other complications (e.g., suicide). According to the NIH (2009), more than 15,000 Americans have HD. At least 150,000 others have a 50% risk of developing the disease, and thousands more of their relatives live with the possibility of developing HD.

Clinical manifestations of HD reflect the cerebral atrophy caused by the neural degeneration. Initially, manifestations are insidious and vary from person to person. Family members may first notice that the individual experiences mood swings or becomes uncharacteristically irritable, apathetic, passive, depressed, or angry. Other behavioral symptoms may include antisocial behavior, hallucinations, paranoia, and psychosis. These symptoms may lessen as the disease progresses or, in some individuals, may continue and include aggression or severe depression. HD may produce a dementia as the individual's judgment, memory, and other cognitive functions become affected. Early signs often include having trouble driving, learning new things, remembering facts, answering questions, or making decisions. Some people may even display changes in handwriting. As the disease progresses, concentration on intellectual tasks becomes increasingly difficult. In some people, the disease may begin with uncontrolled, rapid, jerky movements (**chorea**) (e.g., tremors, grimaces, and twitching) in the fingers, feet, face, or trunk. These movements often intensify when the person is anxious. HD can also begin with mild clumsiness, unsteady gait, and rigidity. Some people develop chorea manifestations later, after the disease has progressed. Chorea often creates serious problems with ambulation, increasing the likelihood of falls. As the disease progresses, speech becomes slurred and other functions (e.g., swallowing, eating, speaking, and walking) continue to decline. Many people with HD remain aware of their environment and are able to express emotions, but some cannot recognize their family members.

Because of its psychologic manifestations, HD is often mistaken for psychiatric disorders. Diagnostic procedures of HD include a history, physical examination, psychiatric evaluation, genetic testing for the defective gene (either before or after the onset of symptoms), head CT, head MRI, and head PET. There is no cure and no treatment to stop the progression. Treatment strategies focus on slowing the progression and managing symptoms to maximize functioning. Tetrabenazine (Xenazine) is the first medication specifically approved by the Food and Drug Administration for the treatment of HD signs and symptoms. Xenazine reduces the jerky, involuntary movements of HD by increasing the amount of dopamine available in the brain. Tranquilizers and antipsychotic agents can control movements, violent outbursts, and hallucinations. Antidepressant agents can control depression and the obsessive-compulsive rituals that some people with HD develop. Some evidence suggests that coenzyme Q10 may also slow the course of the disease. Physical, occupational, and speech therapy can maximize function. Coping strategies, support, adequate hydration, proper nutrition, and regular exercise for both the patient and caregivers can support optimal health. New therapies are currently under investigation, including stem cell therapy, new medications, and new combinations of existing medications.

Dementia

Dementia refers to a group of conditions in which cortical function is decreased, impairing cognitive skills (e.g., language, logical thinking, judgment, and learning) and motor coordination. Issues with memory are common with dementia and include short-term memory losses as well as confusion of historical events. Behavioral and personality changes interfere with relationships, work, and activities of daily living. Vascular disease (e.g., atherosclerosis), infections, toxins, and genetic conditions may cause dementia. There are several types of dementia, each with limited treatment options. Although great strides have been made in recent years, most types of dementia remain poorly understood.

Alzheimer's Disease

Alzheimer's disease (AD) is the most common form of dementia. In AD, healthy brain tissue degenerates and atrophies (**Figure 11-42**). This atrophy causes a steady decline in memory and mental abilities. The exact etiology of AD is unknown, but three pathologic characteristics are associated with AD. Amyloid plaques, which contain fragments of a protein called beta-amyloid peptide, mix with a collection of additional proteins, neuron remnants, and other nerve cell pieces. Neurofibrillary tangles, found inside neurons, are abnormal collections of a protein called tau. Normal tau is required for healthy neurons; however, in

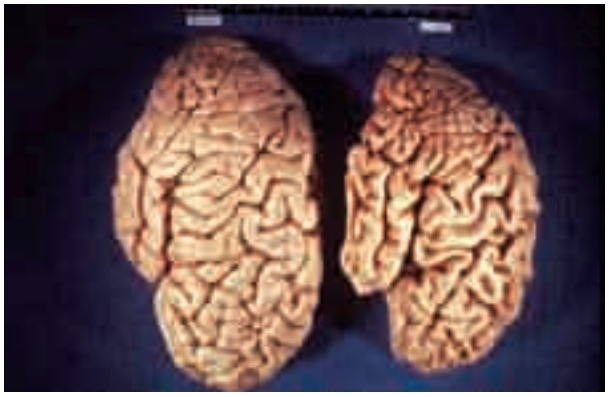


Figure 11-42

Alzheimer's disease.

AD, tau clumps together. As a result, neurons fail to function normally and eventually die. Finally, connections between neurons responsible for memory and learning are lost. Neurons cannot survive when their connections to other neurons are lost. As neurons die throughout the brain, the affected regions begin to atrophy, or shrink. By the final stage of AD, damage is widespread and brain tissue has shrunk significantly.

AD is not a part of normal aging, but risk does increase with age (onset is usually after 60 years of age). Prevalence rates are higher in women, in part because of a longer life expectancy. Some evidence suggests that AD rates are higher in those persons with less education, but the precise reason why this occurs is unknown. Some researchers theorize that the more the brain is used, the more synapses are created, which provides a greater reserve with aging. Additional risk factors include family history, hypertension, hypercholesterolemia, diabetes mellitus, and history of TBI. According to the NIH (2008), as many as 5 million Americans suffer from AD; this number is double what it was in 1980. AD has recently surpassed diabetes mellitus as the sixth leading cause of death among American adults. Notably, mortality rates for AD are on the rise, unlike heart disease and cancer death rates, which are continuing to decline. Complications such as infections (primarily pneumonia and urinary tract infections), injuries related to falls, malnutrition, dehydration, and decubitus ulcers contribute to the mortality associated with AD.

The onset of AD tends to be insidious. Clinical manifestations may start with mild memory loss and confusion, but AD eventually leads to irreversible mental impairment that destroys a person's ability to remember, reason, learn, and imagine. This course may extend 10–20 years. Clinical manifestations may include:

- Memory loss (e.g., one might repeat things, forget conversations or appointments, misplace things, and eventually forget the names of family members and everyday objects)
- Problems with abstract thinking (e.g., trouble balancing a checkbook, a problem that progresses to trouble recognizing and dealing with numbers)
- Difficulty finding the right word to express thoughts or even follow conversations
- Difficulty reading and writing
- Disorientation, even in familiar surroundings
- Loss of judgment (e.g., not knowing what to do if food on the stove is burning)
- Difficulty performing familiar tasks (e.g., driving, cooking, bathing, dressing, and eating)
- Personality changes (e.g., mood swings, paranoia, stubbornness, withdrawal, depression, anxiety, and aggression)
- Hallucinations
- Incontinence of bowel or bladder

Diagnosis of AD is often difficult and often involves ruling out other conditions. Diagnostic procedures consist of a history, physical examination (including a neurologic assessment and mental status evaluation), head CT, head MRI, and head PET. There is no cure for AD, nor are there any therapies that will slow the progression. Medications can manage symptoms and maximize functioning. Cholinesterase inhibitors (e.g., donepezil [Aricept], rivastigmine [Exelon], and galantamine [Razadyne]) can improve neurotransmitter levels in the brain in some cases. Memantine (Namenda) is the newest drug approved specifically to treat AD. Memantine blocks N-methyl-D-aspartic acid receptors, which are glutamate receptors. Memantine may be given in combination with a cholinesterase inhibitor. Other medications may be given to control aggression. Alternative therapies that may improve symptoms include vitamin E, ginkgo, and Huperzine A, but the research evidence is mixed regarding the efficacy of these therapies. Other strategies may include memory aids (e.g., calendars), nutritional support, physical exercise, cognitive activities, safety precautions (e.g., supervision and removing clutter), maintaining a calm environment, and social interactions (e.g., adult day care).

Coping strategies and support for both the patient and the caregiver can decrease stress and anxiety.

Creutzfeldt-Jakob Disease

Creutzfeldt-Jakob disease (CJD) is a rare, but rapidly progressive form of dementia caused by an infectious prion. A **prion** is an abnormal protein particle that causes proteins to fold abnormally, especially in nervous tissue. The prion renders the protein dysfunctional, creating plaques and vacuoles (empty spaces) (**Figure 11-43**). CJD occurs in two types (classic and variant) and three main categories (sporadic, hereditary, and acquired). Although also caused by a prion, classic CJD is *not* related to bovine spongiform encephalopathy, or mad cow disease. However, the new variant *is* related to bovine spongiform encephalopathy. The most common form of classic CJD occurs sporadically, caused by the spontaneous transformation of normal prion proteins into abnormal prions. This sporadic disease occurs worldwide, including the United States, at an annual rate of approximately 1 case per 1 million people (CDC, 2009a). Hereditary CJD is rare and occurs when the abnormal protein is inherited. Finally, acquired CJD is rare (accounting for fewer than 1% of cases worldwide) and occurs when the individual is exposed to infected materials (e.g., via tissue transplants and ingestion). The prion is resistant to common methods of sterilization and disinfection. CJD has a long incubation period (up to 40 years) after being introduced into the brain; however, CJD is rapidly progressing and always fatal (usually within 1 year of onset).

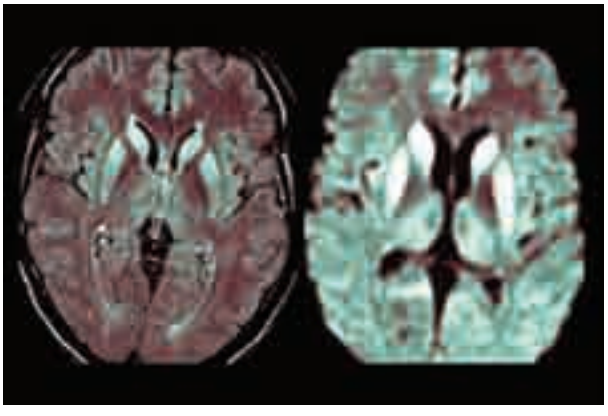


Figure 11-43

Creutzfeldt-Jakob disease.

Clinical manifestations of CJD develop rapidly and include:

- Blurred vision
- Ataxia
- Hallucinations
- Lack of coordination
- Muscle twitching
- Myoclonic jerks or seizures
- Spasticity
- Anxiety
- Personality changes
- Profound confusion or disorientation
- Lethargy
- Speech impairment

Diagnostic procedures for CJD consist of a history, physical examination (including a neurologic assessment and mental status evaluation), EEG, head MRI, and other tests to rule out other forms of dementia (e.g., lumbar puncture, serum tests). There is no known cure for CJD. Interleukins and other immunomodulator agents may slow the progression of the disease. Custodial care (nonmedical care that assists with activities of daily living) may be required early in the course of the disease. Medications may be needed to control aggressive behaviors, spasticity, pain, and seizure activity. Providing a safe environment, controlling aggressive or agitated behavior, and meeting physiologic needs may require monitoring and assistance in the home or in an institutionalized setting. Family counseling may help in coping with the changes required for home care.

AIDS Dementia Complex

Dementia is common in later stages of AIDS (see Chapter 2) and is referred to as **AIDS dementia complex**, or human immunodeficiency virus– (HIV–) associated encephalopathy. The HIV invades the brain tissue and may be exacerbated by other infections and tumors that are frequently associated with AIDS. Clinical manifestations include encephalitis, behavioral changes, and a gradual decline in cognitive function (e.g., trouble with concentration, memory, and attention). Persons with AIDS dementia complex also show progressive slowing of motor function with a loss of dexterity and coordination. In children with congenital HIV, the brain is often affected, causing mental retardation and delayed motor development. A staging system is used

to describe the condition's progression. The staging system ranges from 0 (normal) to 4 (nearly vegetative). Diagnostic procedures of AIDS dementia complex consist of a history, physical examination (including a neurologic assessment and mental status evaluation), head CT, head MRI, and biopsy. When left untreated, AIDS dementia complex can be fatal. Aggressive antiretroviral therapy is the cornerstone of treatment.

Cancers of the Nervous System

Nervous system malignancies can originate in the brain or spinal cord, and they may spread from other sites. Regardless of the etiology, these cancers can result in significant neurologic dysfunction and death. Typical cancer diagnosis, staging, and treatments are usually utilized (see Chapter 1).

Brain Tumors

Brain tumors, whether malignant or benign, can be life threatening because they often increase ICP and are difficult to access (**Figure 11-44**). Brain tumors may be primary, but most are secondary tumors. Any cancer can spread to the brain, but the most common types that do so include breast cancer, colon cancer, kidney cancer, lung cancer, melanoma, and sarcoma. Primary tumors are thought to arise from genetic mutations. The risk for this mutation increases with age and exposure to radiation and occupational chemicals. In the United States, prevalence and mortality rates of brain tumors are highest among Caucasians and males (National Cancer Institute, 2009). Complications of brain tumors include neurologic deficits, seizures, personality changes, and death. The 5-year survival rate for brain tumors is nearly 35%. Clinical manifestations of brain tumors vary depending on size and location. These manifestations reflect the increase in ICP and may include:

- New onset or change in pattern of headaches
- Headaches that gradually become more frequent and more severe

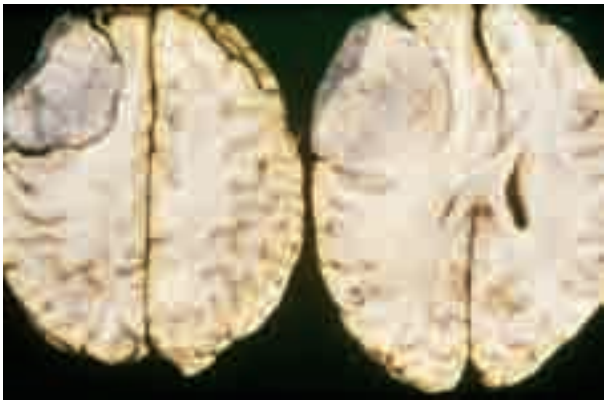


Figure 11-44

Brain tumor.

- Unexplained nausea or vomiting
- Vision problems (e.g., blurred vision, diplopia, or loss of peripheral vision)
- Gradual loss of sensation or movement in an extremity
- Balance difficulties
- Speech difficulties
- Confusion
- Hearing problems
- Hormonal (endocrine) disorders

Diagnostic procedures consist of a history, physical examination (including a neurologic assessment), head MRI, biopsy, and other tests to determine cancer histology. Treatment of brain tumors depends on the size and location of the originating cancer, if any. If possible, surgical removal of the tumor is recommended. Additional treatment options include radiation and chemotherapy. Regardless of the strategy, rehabilitation will be necessary to minimize residual neurologic dysfunction. Rehabilitation will likely require physical, occupational, and speech therapy.

Chapter Summary

The nervous system is a complex network that receives, organizes, and responds to internal and external stimuli, which is vital for homeostasis. The nervous system controls all sensory and motor functions. Damage to this system, even minor, can result in significant neurologic deficits. The nature and severity of those deficits depends on the location and extent of damage. This damage can result from trauma, infections, tumors, chemical imbalances, or genetic conditions. Regardless of the neurologic disorder, the individual may face significant neurologic dysfunction and even death. Supporting neurologic health involves strategies such as observing safety precautions (e.g., wearing safety equipment), avoiding illicit drug use, minimizing alcohol consumption, getting vaccinations, and maintaining adequate nutrition.

Case Study Answers

1. Delivering the child by cesarean section prevents damage to the fragile myelomeningocele, which could worsen neurologic complications.
2. Classic manifestations of hydrocephalus include bulging fontanelles, a high-pitched cry, and a large head circumference. A neonate's fontanelles should be flat; bulging indicates increased pressure within the brain. A high-pitched cry also is a sign of ICP. The saclike projection in his lumbar region is consistent with spina bifida.
3. Myelomeningoceles can cause life-threatening infections in the neonate if the sac loses its integrity prior to surgical closure. In addition, myelomeningocele leads to neurologic deficits similar to a spinal cord injury including neurogenic bladder and bowel, weakness of the lower extremities, and paralysis when located in the lumbar region. Defects located higher than the thoracic level result in more severe neurologic damage; those in the thoracic level and above may cause preterm or neonatal death. Because the central nervous system, including all of its components, develops early, hydrocephalus most commonly occurs in conjunction with neural tube defects. Latex allergies are common in children with neural tube defects because of the need for daily intermittent urinary catheterization.

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Resources

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www.mayoclinic.com

www.medlineplus.gov

www.michaeljfox.com

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