In every joint, there is a tradeoff between mobility and stability.

**Stable (yet limited mobility)**
The more stable a joint, the less mobile it is.

**Fibrous Joints**
Their primary function is to hold two bones together. They are immobile or slightly mobile. Examples: **Sutures** and **interosseous membrane**

**Cartilaginous Joints**
Their primary function is to resist compression and tension stress and act as resilient shock absorbers. They are immobile or slightly mobile. Example: **Intervertebral joints**

**Synovial Joints**
Their primary function is movement, so they are all freely mobile. Examples: **Glenohumeral joint** (shoulder) and **knee joint**

**Mobile (yet less stable)**
The more mobile a joint, the less stable it is.

**In every joint, there is a tradeoff between mobility and stability.**
9.2 Fibrous Joints

Articulating bones in fibrous joints are connected by dense regular connective tissue. Fibrous joints have no joint cavity; thus, they lack a space between the articulating bones. Most fibrous joints are immobile or at most only slightly mobile; their primary function is to hold together two bones. Examples include the articulations of the teeth in their sockets, sutures between skull bones, and articulations between either the radius and ulna or the tibia and fibula. In this section, we look at the three types of fibrous joints: gomphoses, sutures, and syndesmoses (figure 9.2).

9.2a Gomphoses

LEARNING OBJECTIVE

4. Explain the location and characteristics of gomphoses.

A gomphosis (gom-fo’sis; pl., gomphoses, -sēz; gomphos = bolt) resembles a “peg in a socket.” The only gomphoses in the human body are the articulations of the roots of individual teeth with the alveolar processes (sockets) of the mandible and the maxillae. A tooth is held firmly in place by fibrous periodontal (per’ē-ō-don’tāl; peri = around, odous = tooth) membranes. This joint is immobile and thus is functionally classified as a synarthrosis.

![Gomphosis Diagram](image)

Figure 9.2 Fibrous Joints. Dense regular connective tissue binds the articulating bones in fibrous joints to prevent or restrict movement. (a) A gomphosis is a synarthrosis (immobile joint) between a tooth and the jaw. (b) A suture is a synarthrosis (immobile joint) between bones of the skull. (c) A syndesmosis is classified functionally as an amphiarthrosis, as it permits slight mobility between the radius and the ulna (shown) and between the tibia and fibula (not shown).

The reasons orthodontic braces can be painful and take so long to correctly position the teeth are directly related to the architecture of the gomphosis. The orthodontist’s job is to reposition these normally immobile joints through the use of clamps, bands, rings, and braces. In response to these mechanical stressors, osteoblasts and osteoclasts (see section 7.2e) work together to modify the alveolar process, resulting in the remodeling of the joints and the slow repositioning of the teeth.

WHAT DID YOU LEARN?

3. Where are gomphoses located, and what type of movement do they allow?

9.2b Sutures

LEARNING OBJECTIVE

5. Describe the location and functions of sutures.

Sutures (st’chur; sutura = a seam) are fibrous joints found only between certain bones of the skull. Sutures are functionally classified as synarthroses, since they are immobile joints. Sutures have distinct, interlocking, usually irregular edges that both increase their strength and decrease the number of fractures at these articulations. In addition to joining bones, sutures permit the skull to grow as the brain increases in size during childhood. In an older adult, the dense regular connective tissue in

![Sutures Diagram](image)
the suture becomes ossified, fusing the skull bones together. When the bones have completely fused across the suture line, these obliterated sutures are now called synostoses (sin-os-tō’sēz; sing., -sis; osteon = bone). (See Clinical View 8.2: “Craniosynostosis and Plagiocephaly.”)
The sphen-occipital synchondrosis is found between the body of the sphenoid and the basilar part of the occipital bone. This synchondrosis typically fuses between 18 and 25 years of age, making it a useful tool for assessing the age of the skull (see section 8.4b).

Other examples of synchondroses are formed from costal cartilage. The costochondral (kos-tō-konˈ drāl; costa = rib) joint, the joint between each bony rib and its respective costal cartilage, is a synchondrosis. Finally, the attachment of the first rib to the sternum by costal cartilage (called the first sternocostal joint) is another synchondrosis. Here, the first rib and its costal cartilage are united firmly to the manubrium of the sternum to provide stability to the rib cage. (Note that the sternocostal joints between the sternum and the costal cartilage of ribs 2–7 are synovial joints and not synchondroses.)

### 9.3b Symphyses

#### LEARNING OBJECTIVE

8. Name the locations of symphyses and their functions in these locations.

A symphysis (sim′si-sis; pl., symphyses, -sēz; growing together) has a pad of fibrocartilage between the articulating bones (figure 9.3b). The fibrocartilage resists both compression and tension stresses and acts as a resilient shock absorber. All symphyses are amphiarthroses—thus, they allow slight mobility.

One example of a symphysis is the pubic symphysis, which is located between the right and left pubic bones. In pregnant females, the pubic symphysis becomes more mobile to allow the pelvis to change shape slightly as the fetus passes through the birth canal.

Other examples of symphyses are the intervertebral joints, where the bodies of adjacent vertebrae are both separated and united by intervertebral discs. Individual intervertebral discs allow only slight movements between the adjacent vertebrae; however, the collective movements of all the intervertebral discs afford the spine considerable flexibility.

### 9.4 Synovial Joints

Synovial joints are freely mobile articulations (i.e., diarthroses). Most of the commonly known joints in the body are synovial joints, including the glenohumeral (shoulder) joint, the temporomandibular joint, the elbow joint, and the knee joint.

#### 9.4a Distinguishing Features and Anatomy of Synovial Joints

**LEARNING OBJECTIVES**

9. Identify the characteristics common to all synovial joints.
10. List the basic features of a synovial joint.
11. Describe the composition and function of synovial fluid in a typical synovial joint.

Unlike the joints previously discussed, the bones in a synovial joint are separated by a space called a joint cavity. Functionally, all synovial joints are classified as diarthroses, because all are freely mobile. Often, the terms synovial joint and diarthrosis are equated. All synovial joints include several basic features: an articular capsule, a joint cavity, synovial fluid, articular cartilage, ligaments, nerves, and blood vessels (figure 9.4).

Each synovial joint is composed of a double-layered capsule called the articular (ar-tikˈyu-lər) capsule, or joint capsule. Its outer layer is called the fibrous layer, and the inner layer is a synovial membrane (or synovium) (see section 5.5b). The fibrous layer is formed from dense connective tissue. It strengthens the joint to prevent the bones from being pulled apart. The synovial membrane is a specialized type of connective tissue, the cells of which help produce and secrete synovial fluid (described shortly). This membrane covers all the internal joint surfaces not covered by cartilage and lines the articular capsule.

All articulating bone surfaces in a synovial joint are covered by a thin layer of hyaline cartilage called articular cartilage. This cartilage has numerous functions: It reduces friction in the joint during movement, acts as a spongy cushion to absorb compression placed on the joint, and prevents damage to the articulating ends of the bones. This special hyaline cartilage lacks a perichondrium (see section 7.2c). Mature cartilage is avascular, so it does not have blood vessels to bring nutrients to and remove waste products from the cartilage. The repetitious compression and expansion that occurs during exercise is vital to maintaining healthy articular cartilage because this action enhances its obtaining nutrition and its waste removal.

Only synovial joints house a joint cavity (or articular cavity), a space that permits separation of the articulating bones. The articular cartilage and synovial fluid (described next) within the joint cavity together reduce friction as bones move at a synovial joint.

**Synovial fluid** is a viscous, oily substance located within a synovial joint. It is a product of both the synovial membrane cells...
and the filtrate formed from blood plasma. Synovial fluid has three functions:

1. Synovial fluid lubricates the articular cartilage on the surface of articulating bones (in the same way that oil in a car engine lubricates the moving engine parts).

2. Synovial fluid nourishes the articular cartilage’s chondrocytes. The relatively small volume of synovial fluid must be circulated continually to provide nutrients to and remove wastes from these cells. Whenever movement occurs at a synovial joint, the combined compression and re-expansion of the articular cartilage circulates the synovial fluid into and out of the cartilage matrix.

3. Synovial fluid acts as a shock absorber, distributing stresses and force evenly across the articular surfaces when the pressure in the joint suddenly increases.

Ligaments (lig’-ment; ligamentum = a band) are composed of dense regular connective tissue, and they connect one bone to another bone. Ligaments function to stabilize, strengthen, and reinforce most synovial joints. Intrinsic ligaments represent thickenings of the articular capsule itself. Intrinsic ligaments include extracapsular ligaments outside the joint capsule and intracapsular ligaments within the joint capsule. Extrinsic ligaments are outside of, and physically separate from, the joint capsule. The specific intrinsic and extrinsic ligaments are specific to each type of joint (e.g., knee, shoulder).

All synovial joints have numerous sensory nerves that innervate the articular capsule and associated ligaments. The sensory nerves detect painful stimuli in the joint and report on the amount of movement and stretch within the joint. By monitoring stretching at a joint, the nervous system can detect changes in our posture and adjust body movements. Synovial joints also contain blood vessels that provide oxygen and nutrients and remove wastes.

Tendons (ten’dôn; tendo = extend) are like ligaments and are composed of dense regular connective tissue, but they are not part of the synovial joint itself. Whereas a ligament binds bone to bone, a tendon attaches a muscle to a bone. When a muscle contracts, the tendon from that muscle moves the bone to which it is attached, thus causing movement at the joint. Tendons help stabilize joints because they pass across or around a joint to provide mechanical support, and sometimes they limit the range or amount of movement permitted at a joint.

Synovial joints usually have bursae and fat pads as accessory structures in addition to the main components just described. A bursa (ber’s; pl., bursae, ber’së; a purse) is a fibrous, saclike structure that contains synovial fluid and is lined internally by a synovial membrane (figure 9.5a). Bursae are associated with most

**Figure 9.5 Bursae and Tendon Sheaths.** Synovial fluid–filled structures called bursae and tendon sheaths reduce friction where ligaments, muscles, tendons, and bones rub together. (a) The knee joint contains a number of bursae (blue and purple). (b) The wrist and hand contain numerous tendon sheaths (blue).
synovial joints and are where bones, ligaments, muscles, skin, or tendons overlie each other and rub together. Bursae may be either connected to the joint cavity or completely separate from it. They alleviate the friction resulting from the various body movements, such as where a tendon or ligament rubs against bone. An elongated bursa called a tendon sheath wraps around a tendon where there may be excessive friction. Tendon sheaths are especially common in the confined spaces of the wrist and ankle (figure 9.5b).

Fat pads are often distributed along the periphery of a synovial joint. They act as packing material and provide some protection for the joint. Often, fat pads fill the spaces that form when bones move and the joint cavity changes shape (figure 9.5a).

**WHAT DID YOU LEARN?**

8. What are the basic characteristics of all types of synovial joints?

9. What is the purpose of synovial fluid in the joint?

---

### 9.4b Classification of Synovial Joints

**LEARNING OBJECTIVES**

12. Explain the movement of a joint with respect to the three perpendicular axes of space.

13. Compare and contrast the six types of synovial joints.

Synovial joints are classified by the shapes of their articulating surfaces and the types of movement they allow. Movement of a bone at a synovial joint is best described with respect to three intersecting perpendicular planes or axes:

- A joint is said to be uniaxial (yū-nē-ak’sē-āl; unus = one) if the bone moves in just one plane or axis.
- A joint is biaxial (br-ak’sē-āl; bi = double) if the bone moves in two planes or axes.
- A joint is multiaxial or triaxial (trā-ak’sē-āl; tri = three) if the bone moves in multiple planes or axes.

All synovial joints are diarthroses, as mentioned, but some are more mobile than others. From least mobile to most freely mobile, the six specific types of synovial joints are plane joints, hinge joints, pivot joints, condylar joints, saddle joints, and ball-and-socket joints. These joints and examples of where they are found in the body are shown in figure 9.6.

---

### WHAT DO YOU THINK?

2. If a ball-and-socket joint is more mobile than a gliding joint, which of these two joints is the more stable?

A plane (planus = flat) joint, also called a planar or gliding joint, is the simplest synovial articulation and the least mobile type of diarthrosis. Anatomists have debated how to describe the movement of this joint with respect to perpendicular planes. We describe this type of synovial joint as a uniaxial joint because it usually allows only limited side-to-side movements in a single plane, and because there is no rotational or angular movement with this joint. The articular surfaces of the bones are flat, or planar. Examples of plane joints include the intercarpal and intertarsal joints (the joints between the carpal and tarsal bones, respectively).

A hinge joint is formed by the convex surface of one articulating bone fitting into a concave depression on the other bone in the joint. Movement is confined to a single axis, like the movement seen at the hinge of a door, so a hinge joint is considered a uniaxial joint. An example is the elbow joint. The trochlear notch of the ulna fits directly into the trochlea of the humerus, so the forearm can be moved only anteriorly toward the arm or posteriorly away from the arm. Other hinge joints occur in the knee and the finger (interphalangeal [IP]) joints.

A pivot joint is a uniaxial joint in which one articulating bone with a rounded surface fits into a ring formed by a ligament and another bone. The first bone rotates on its longitudinal axis relative to the second bone. An example is the proximal radio-ulnar joint, where the rounded head of the radius pivots along the ulna and permits the radius to rotate. Another example is the atlantoaxial joint between the first two cervical vertebrae. The rounded dens of the axis fits snugly against an articular facet on the anterior arch of the atlas. This joint pivots when you shake your head “no.”

Condylar (kon′di-lar) joints, also called condyloid or ellipsoid joints, are biaxial joints with an oval, convex surface on one bone that articulates with a concave articular surface on the second bone of the joint. Biaxial joints can move in two axes, such as back-and-forth and side-to-side. Examples of condylar joints are the metacarpophalangeal (met-ā-kar′pō-fā-lan′jē-āl) (MP) joints of fingers 2 through 5. The MP joints are commonly referred to as knuckles. Examine your hand and look at the movements along the MP joints; you can flex and extend the fingers at this joint, which is one axis of movement. You also can move your fingers apart from one another and move them closer together, which is the second axis of movement.

A saddle joint is so named because the articular surfaces of the bones have convex and concave regions that resemble the shape of a saddle. This biaxial joint allows a greater range of movement than either a condylar or hinge joint. The carpometacarpal joint of the thumb (between the trapezium, which is a carpal bone, and the first metacarpal) is an example of a saddle joint. This joint permits the thumb to move toward the other fingers so that we can grasp objects.

Ball-and-socket joints are multiaxial joints in which the spherical articular head of one bone fits into the rounded, cuplike socket of a second bone. Examples of these joints are the coxal (hip) and glenohumeral (shoulder) joints. The multiaxial nature of these joints permits movement in three planes. Move your arm at your shoulder, and note the wide range of movements that can be produced. The ball-and-socket joint is considered the most freely mobile type of synovial joint.

---

### WHAT DID YOU LEARN?

10. What types of movements do each of the six kinds of joints allow?

### 9.5 The Movements of Synovial Joints

Four types of motion occur at synovial joints: gliding motion, angular motion, rotational motion, and special movements (motions that occur only at specific joints) (table 9.2).

#### 9.5a Gliding Motion

**LEARNING OBJECTIVE**

14. Describe gliding motion, and name joints in which it occurs.

Gliding is a simple movement in which two opposing surfaces slide slightly back-and-forth or side-to-side with respect to one another. In a
### Synovial Joints

**Structural Categories**

| Uniaxial | Biaxial | Multiaxial (Triaxial) |

| **Plane joint:** | **Condylar joint:** | **Ball-and-socket joint:** |

- **Plane joint:** Flattened or slightly curved faces slide across one another.
- **Condylar joint:** Oval articular surface on one bone closely interfaces with a depressed oval surface on another bone.
- **Ball-and-socket joint:** Round head of one bone rests within cup-shaped depression in another bone.

| **Example(s)** | **Functional Classification** |

- **Plane joint:** Intercarpal joints, intertarsal joints
- **Condylar joint:** MP (metacarpophalangeal or metatarsophalangeal) joints
- **Ball-and-socket joint:** Glenohumeral (shoulder) joint, hip joint

- **Diarthrosis (freely mobile)**

---

**Figure 9.6 Synovial Joints.** Synovial joints contain a joint cavity within an articular capsule lined by a synovial membrane. All synovial joints are diarthroses. The six types of synovial joints and examples of their locations in the body are shown.
gliding motion, the angle between the bones does not change, and only limited movement is possible in any direction. Gliding motion typically occurs along plane joints, such as between the carpals or the tarsals.

### WHAT DID YOU LEARN?

11. What joints typically use gliding motion?

### 9.5b Angular Motion

#### LEARNING OBJECTIVES

- **15.** Describe angular motion.
- **16.** Name the specific types of angular motion.
- **17.** Give examples of joints that exhibit angular motion.

Angular motion either decreases or increases the angle between two bones. These movements may occur at many of the synovial joints. They include the following specific types:

---

<table>
<thead>
<tr>
<th>Movement</th>
<th>Description</th>
<th>Opposing Movement</th>
</tr>
</thead>
<tbody>
<tr>
<td>GLIDING MOTION</td>
<td>Two opposing articular surfaces slide past each other in almost any direction; the amount of movement is slight</td>
<td></td>
</tr>
<tr>
<td>ANGULAR MOTION</td>
<td>The angle between articulating bones increases or decreases</td>
<td></td>
</tr>
<tr>
<td>Flexion</td>
<td>The angle between articulating bones decreases in an anterior-posterior (AP) plane</td>
<td>Extension</td>
</tr>
<tr>
<td>Extension</td>
<td>The angle between articulating bones increases in an anterior-posterior (AP) plane</td>
<td>Flexion</td>
</tr>
<tr>
<td>Hyperextension</td>
<td>Extension movement continues past 180 degrees</td>
<td>Flexion</td>
</tr>
<tr>
<td>Lateral flexion</td>
<td>The vertebral column moves (bends) in a lateral direction along a coronal plane</td>
<td>None</td>
</tr>
<tr>
<td>Abduction</td>
<td>Lateral movement of a body part away from the midline</td>
<td>Adduction</td>
</tr>
<tr>
<td>Adduction</td>
<td>Lateral movement of a body part toward the midline</td>
<td>Abduction</td>
</tr>
<tr>
<td>Circumduction</td>
<td>A continuous movement that combines flexion, abduction, extension, and adduction in succession; the distal end of the limb or digit moves in a circle</td>
<td>None</td>
</tr>
<tr>
<td>ROTATIONAL MOTION</td>
<td>A bone pivots around its own longitudinal axis</td>
<td></td>
</tr>
<tr>
<td>Pronation</td>
<td>Rotation of the forearm where the palm is turned posteriorly or inferiorly</td>
<td>Supination</td>
</tr>
<tr>
<td>Supination</td>
<td>Rotation of the forearm in which the palm is turned anteriorly or superiorly</td>
<td>Pronation</td>
</tr>
<tr>
<td>SPECIAL MOVEMENTS</td>
<td>Types of movement that do not fit into the previous categories</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>Movement of a body part inferiorly</td>
<td>Elevation</td>
</tr>
<tr>
<td>Elevation</td>
<td>Movement of a body part superiorly</td>
<td>Depression</td>
</tr>
<tr>
<td>Dorsiflexion</td>
<td>Ankle joint movement where the dorsum (superior surface) of the foot is brought toward the anterior surface of the leg</td>
<td>Plantar flexion</td>
</tr>
<tr>
<td>Plantar flexion</td>
<td>Ankle joint movement where the sole of the foot is brought toward the posterior surface of the leg</td>
<td>Dorsiflexion</td>
</tr>
<tr>
<td>Eversion</td>
<td>Twisting motion of the foot that turns the sole laterally or outward</td>
<td>Inversion</td>
</tr>
<tr>
<td>Inversion</td>
<td>Twisting motion of the foot that turns the sole medially or inward</td>
<td>Eversion</td>
</tr>
<tr>
<td>Protraction</td>
<td>Anterior movement of a body part from anatomic position</td>
<td>Retraction</td>
</tr>
<tr>
<td>Retraction</td>
<td>Posterior movement of a body part from anatomic position</td>
<td>Protraction</td>
</tr>
<tr>
<td>Opposition</td>
<td>Special movement of the thumb across the palm toward the fingers to permit grasping and holding of an object</td>
<td>Reposition</td>
</tr>
</tbody>
</table>

1. Some movements (e.g., circumduction) do not have an opposing movement.
flexion and extension, hyperextension, lateral flexion, abduction and adduction, and circumduction.

**Flexion** (flek′shŭn; flecto = to bend) is movement in an anterior-posterior (AP) plane of the body that decreases the angle between the bones. Bones are brought closer together as the angle between them decreases. Examples are the bending of the fingers toward the palm to make a fist, the bending of the forearm toward the arm at the elbow, flexion at the shoulder when the arm is raised anteriorly, and flexion of the neck when the head is bent anteriorly and you look down at your feet.

The opposite of flexion is **extension** (eks-ten′shŭn; extensio = a stretching out), which is movement in an anterior-posterior (AP) plane that increases the angle between the articulating bones. Extension is a straightening action that occurs in an AP plane. Straightening the arm and forearm until the upper limb projects directly away from the anterior side of your body, and straightening the fingers after making a clenched fist, are examples of extension.

When a joint is extended more than 180 degrees, the movement is termed **hyperextension** (hī′per-eks-ten′shŭn; hyper = above normal). For example, if you extend your arm and hand with the palm facing inferiorly, and then raise the back of your hand as if admiring a new ring on your finger, the wrist is hyperextended. If you glance up at the ceiling while standing, your neck is hyperextended. Flexion, extension, and hyperextension of various body parts are illustrated in figure 9.7a–d.

**Lateral flexion** occurs when the trunk of the body moves in a coronal plane laterally away from the body. This type of movement occurs primarily between the vertebrae in the cervical and lumbar regions of the vertebral column (figure 9.7e).

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**Figure 9.7 Flexion, Extension, Hyperextension, and Lateral Flexion.** Flexion decreases the joint angle in an anterior-posterior (AP) plane, whereas extension increases the joint angle in the AP plane. Hyperextension is extension of a joint beyond 180 degrees. Lateral flexion decreases a joint angle, but in a coronal plane. Examples of joints that allow some of these movements are (a) the atlanto-occipital joint, (b) the elbow joint, (c) the radiocarpal joint, (d) the knee joint, and (e) the intervertebral joints.

---

**Abduction** (ab-dŭk′shŭn; duco = to draw) means to move away, and it is a lateral movement of a body part away from the body midline. Abduction occurs when either the arm or the thigh is moved laterally away from the body midline. Abduction of either the fingers or the toes means that you spread them apart, away from the longest digit that acts as the midline. Abducting the wrist (also known as radial deviation) involves pointing the hand and fingers laterally, away from the body. The opposite of abduction is **adduction** (ad-dŭk′shŭn), meaning to move toward. This is the medial movement of a body part toward the body midline. Adduction occurs when the raised arm or thigh is brought back toward the body midline, or in the case of the digits, toward the midline of the hand. Adducting the wrist (also known as ulnar deviation) involves pointing the hand and fingers medially, toward the body. Abduction and adduction of various body parts are shown in figure 9.8.
Circumduction (ser-kūm-dūk′shūn; circum = around) is a sequence of movements in which the proximal end of an appendage remains relatively stationary while the distal end makes a circular motion (figure 9.9). The resulting movement makes an imaginary cone shape. This is demonstrated when you draw a circle on the blackboard. The shoulder remains stationary while your hand moves. The tip of the imaginary cone is the stationary shoulder, while the rounded “base” of the cone is the circle made by the hand. Circumduction is a complex movement that occurs as a result of a continuous sequence of flexion, abduction, extension, and adduction.

**WHAT DID YOU LEARN?**

1. How do flexion and extension differ? What movements are involved in circumduction?

**9.5c Rotational Motion**

LEARNING OBJECTIVE

18. Explain rotational motion, and name joints in which it occurs.

Rotation is a pivoting motion in which a bone turns on its own longitudinal axis (figure 9.10). Rotational movement occurs at the atlantoaxial joint, which pivots when you rotate your head to gesture “no.” Some limb rotations are described as either away from the median plane or toward it. For example, lateral rotation (or external rotation) turns the anterior surface of the femur or humerus laterally, whereas medial rotation (or internal rotation) turns the anterior surface of the femur or humerus medially.

Pronation (prō-nā′shūn) is the medial rotation of the forearm so that the palm of the hand is directed posteriorly or inferiorly. The radius and ulna are crossed to form an X (see section 8.9b).

**Figure 9.9 Circumduction.** Circumduction is a complex movement that involves flexion, abduction, extension, and adduction in succession. Examples of joints that allow this movement are (a) the glenohumeral joint and (b) the hip joint.

(a, b) ©McGraw-Hill Education/Jw Ramsey

**Figure 9.8 Abduction and Adduction.** Abduction moves a body part away from the trunk in a lateral direction, whereas adduction moves the body part toward the trunk in a medial direction. Some examples occur at (a) the glenohumeral joint, (b) the radiocarpal joint, (c) the hip joint, and (d) the metacarpophalangeal (MP) joints.

(a, b, c) ©McGraw-Hill Education/Jw Ramsey; (d, left, right) ©McGraw-Hill Education/Tamara Klein
Supination (sū′pi-nă′shŭn) occurs when the forearm rotates laterally so that the palm faces anteriorly or superiorly. In the anatomic position, the forearm is supinated. Figure 9.10 illustrates pronation and supination.

WHAT DID YOU LEARN?

13 What is pronation, and where in the body may this type of movement be performed?

9.5d Special Movements

LEARNING OBJECTIVE

19. Explain what is meant by special movements, and give examples of joints at which they occur.

Some movements occur only at specific joints and do not readily fit into any of the functional categories previously discussed. These special movements include depression and elevation, dorsiflexion and plantar flexion, eversion and inversion, protraction and retraction, and opposition.

Depression (dĕ-presh′ŭn, de = away, down, presso = to press) is the inferior movement of a part of the body. Examples of depression include opening your mouth (by depressing your mandible) to chew food and the movement of your shoulders in an inferior direction. Elevation (el-ĕ-vă′shŭn) is the superior movement of a body part. Examples of elevation include the superior movement of the mandible while closing the mouth and the movement of the shoulders in a superior direction (shrugging your shoulders). Figure 9.11 illustrates depression and elevation at the glenohumeral joints.

Dorsiflexion and plantar flexion are limited to the ankle joint (figure 9.11b). Dorsiflexion (dŏr-si-flek′shŭn) occurs when the talocrural (ankle) joint is bent such that the dorsum (superior surface) of the foot and the toes moves toward the leg. This movement occurs when you dig in your heels, and it prevents your toes from scraping the ground when you take a step. Plantar flexion (plan′tăr; planta = sole of foot) is a movement of the foot at the talocrural joint so that the toes point inferiorly. When a ballerina is standing on her tiptoes, her ankle joint is in full plantar flexion.

Eversion and inversion are movements that occur at the intertarsal joints of the foot only (figure 9.11c). During eversion (ĕ-ver′zhŭn), the sole of the foot turns to face laterally or outward, whereas the sole of the foot turns medially or inward during inversion (in-ver′zhŭn). (Note: Some orthopedists and runners use the terms pronation and supination when describing foot movements as well, instead of using eversion and inversion. Simply put, eversion is foot pronation, whereas inversion is foot supination.)
9.6 Synovial Joints and Levers

When analyzing synovial joint movement and muscle contraction, anatomists often compare the movement to the mechanics of a lever; this practice of applying mechanical principles to biology is known as biomechanics.

9.6a Terminology of Levers

A lever (lev’er, lē’ver; to lift) is an elongated, rigid object that rotates around a fixed point called the fulcrum (ful’krum). A seesaw is a familiar example of a lever. Levers have the ability to alter the speed and distance of movement produced by a force, the direction of an applied force, and the force strength.

Movement occurs when an effort applied to one point on the lever exceeds a resistance located at some other point. The part of a lever from the fulcrum to the point of effort is called the effort arm, and the lever part from the fulcrum to the point of resistance is the resistance arm. In the body, a long bone acts as a lever, a joint serves as the fulcrum, and the effort is generated by a muscle attached to the bone.

Protraction (prō-trak’šūn) is the anterior movement of a body part from anatomic position, as when jutting your jaw anteriorly at the temporomandibular joint or hunching the shoulders anteriorly by crossing the arms. In the latter case, the clavicles move anteriorly due to movement at both the acromioclavicular and sternoclavicular joints. Retraction (ret-trak’šūn) is the posteriorly directed movement of a body part from the anatomic position. Figure 9.11d illustrates protraction and retraction at the temporomandibular joint.

At the carpometacarpal joint, the thumb moves toward the palmar tips of the fingers as it crosses the palm of the hand. This movement is called opposition (op’ō-shūn) (figure 9.11e). It enables the hand to grasp objects and is the most distinctive digital movement in humans. The opposite movement is called reposition.

WHAT DID YOU LEARN?

14. What is the difference between inversion and eversion, and which joints allow these movements?

15. What is the difference between the effort arm and the resistance arm in a lever?

9.6b Types of Levers

LEARNING OBJECTIVE

22. Compare and contrast the three types of levers in the human body.

Three classes of levers are found in the human body: first-class, second-class, and third-class (figure 9.12).

First-Class Levers

A first-class lever has a fulcrum in the middle, between the effort (force) and the resistance. An example of a first-class lever is a pair of scissors. The effort is applied to the handle of the scissors while the resistance is at the cutting end of the scissors. The fulcrum (pivot for movement) is along the middle of the scissors, between the handle and the cutting ends. In the body, an example of a first-class lever is the atlanto-occipital joint of the neck, where the muscles on the posterior side of the neck (effort) pull inferiorly on the nuchal lines of the skull and oppose the tendency of the head (resistance) to tip anteriorly.

Second-Class Levers

The resistance in a second-class lever is between the fulcrum and the applied effort. A common example of this type of lever is lifting the handles of a wheelbarrow, allowing it to pivot on its wheel at the opposite end and lift a load in the middle. The load weight is the resistance, and the upward lift on the handle is the effort. A small force
can balance a larger weight in this type of lever, because the effort is always farther from the fulcrum than the resistance. Second-class levers are rare in the body, but one example occurs when the foot is depressed (plantar flexed) so that a person can stand on tiptoe. The contraction of the calf muscle causes a pull superiorly by the calcaneal tendon attached to the heel (calcaneus).

**Third-Class Levers**

A **third-class lever** is noted when the effort is applied between the resistance and the fulcrum, as when picking up a small object with a pair of forceps. Third-class levers are the most common levers in the body. A third-class lever is found at the elbow where the fulcrum is the joint between the humerus and ulna, the effort is applied by the biceps brachii muscle, and the resistance is provided by any weight in the hand or by the weight of the forearm itself. The mandible acts as a third-class lever when you bite with your incisors on a piece of food. The temporomandibular joint is the fulcrum, and the temporalis muscle exerts the effort, whereas the resistance is the item of food being bitten.

**WHAT DID YOU LEARN?**

16. How does the position of the fulcrum, resistance, and effort vary in first-class, second-class, and third-class levers?

### 9.7 Features and Anatomy of Selected Joints

Both the axial skeleton and appendicular skeleton exhibit many more joints than are individually discussed here. Table 9.3 summarizes the main features of major joints of the axial skeleton.

The structure and function of the more commonly known articulations of the axial and appendicular skeletons are examined in this section. These are the temporomandibular joint of the skull; the shoulder joint and elbow joint; and the hip joint, knee joint, and talocrural (ankle) joint.

---

**Figure 9.12 Classes of Levers.** (a) In a first-class lever, the fulcrum is located between the resistance and effort, such as with a pair of scissors or the trapezius muscle (in the neck). (b) In a second-class lever, the resistance is between the fulcrum and effort, such as with a wheelbarrow or the calf muscles. (c) The most common type of lever is the third-class lever, where effort is applied between the resistance and the fulcrum, such as with forceps (tweezers) or the arm muscles.

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The temporomandibular joint (TMJ) is subject to various disorders. TMJ disorders are often seen in people who habitually chew gum or grind or clench their teeth. The most common TMJ disorder occurs as a result of alterations in the ligaments that secure the joint, causing progressive internal displacement of the articular disc. As the articular disc is forced out of its normal position, a clicking or popping noise may be heard as the person opens or closes the mouth. Pain from the TMJ disorder may be felt not only within the joint but also in such areas as the paranasal sinuses, tympanic membrane (eardrum), oral cavity, eyes, and teeth. The widespread distribution of pain occurs because all of these structures, including the chewing muscles, mandible, and maxilla, are innervated by numerous sensory branches of the trigeminal nerve (see section 13.9).

### 9.7a Temporomandibular Joint

#### LEARNING OBJECTIVES

23. Describe the features of the temporomandibular joint (TMJ).

24. List the movements of the TMJ.

The temporomandibular (tem′pō-rō-man-dib’yō-lār) joint (TMJ) is the articulation formed at the point where the head of the mandible articulates with the temporal bone—specifically, the articular tubercle of the temporal bone anteriorly and the mandibular fossa posteriorly. This small, complex articulation is the only mobile joint between bones in the skull (figure 9.13 and table 9.3).

The temporomandibular joint has several unique anatomic features. A loose articular capsule surrounds the joint and promotes an extensive range of motion. It contains an articular disc, which is a thick pad of fibrocartilage separating the articulating bones and extending horizontally to divide the synovial cavity into two separate chambers. As a result, the TMJ is really two synovial joints—one between the temporal bone and the articular disc, and a second between the articular disc and the mandible.

Several ligaments support the TMJ. The sphenomandibular ligament (an extracapsular ligament) is a thin band that extends anteriorly and inferiorly from the sphenoid to the medial surface of the mandibular ramus. The temporomandibular ligament (or lateral ligament) is composed of two short bands that extend inferiorly and posteriorly from the articular tubercle of the temporal bone to the mandible.

The temporomandibular joint functions as a hinge during jaw depression and elevation while chewing. It also glides slightly forward during protraction of the jaw for biting, and glides slightly from side to side to grind food between the teeth during chewing.
### Table 9.3

<table>
<thead>
<tr>
<th>Joint Type</th>
<th>Description of Movement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synarthrosis</td>
<td>None allowed</td>
</tr>
<tr>
<td>Diarthrosis</td>
<td>Depression, elevation, lateral displacement, protraction, retraction, slight rotation</td>
</tr>
<tr>
<td>Diarthrosis</td>
<td>Extension and flexion of the head; slight lateral flexion of head to sides</td>
</tr>
<tr>
<td>Diarthrosis</td>
<td>Head rotation</td>
</tr>
<tr>
<td>Amphiarthrosis between vertebral bodies; diarthrosis between articular processes</td>
<td>Extension, flexion, lateral flexion of vertebral column</td>
</tr>
<tr>
<td>Diarthrosis</td>
<td>Some slight gliding</td>
</tr>
<tr>
<td>Amphiarthrosis between lumbar body and base of sacrum; diarthrosis between articular processes</td>
<td>Extension, flexion, lateral flexion of vertebral column</td>
</tr>
<tr>
<td>Synarthrosis between sternum and first ribs; diarthrosis between sternum and ribs 2–7</td>
<td>No movement between sternum and first ribs; some gliding movement permitted between sternum and ribs 2–7</td>
</tr>
</tbody>
</table>

### 9.7b Shoulder Joint

#### LEARNING OBJECTIVES

25. Describe the three individual joints that make up the shoulder articulation.

26. Explain why the glenohumeral joint is relatively unstable.

The joints associated with movement at the shoulder include the sternoclavicular joint, the acromioclavicular joint, and the glenohumeral joint. (Table 9.4 lists the features of the major joints of the pectoral girdle and upper limbs.)

**Sternoclavicular Joint**

The sternoclavicular (ster′nō-kla-vik′yō-lār) joint is a saddle joint formed by the articulation between the manubrium of the sternum and the sternal end of the clavicle (figure 9.14). An articular disc partitions the sternoclavicular joint into two parts and forms two separate synovial cavities. As a result, a wide range of movement is possible, including depression, elevation, and circumduction.

Support and stability are provided to this articulation by the fibers of the articular capsule and by multiple extracapsular ligaments, such as the sternoclavicular and costoclavicular ligaments. This anatomic arrangement makes the sternoclavicular joint very stable. If you fall on an outstretched hand so that force is applied to the joint, the clavicle will fracture before this joint dislocates.

**Acromioclavicular Joint**

The acromioclavicular (ā-kro′mē-ō-kla-vik′yō-lār) joint is a plane joint between the acromion of the scapula and the lateral end of the clavicle (figure 9.15). A fibrocartilaginous articular disc lies within the joint cavity between these two bones. This joint works with both the sternoclavicular joint and the glenohumeral joint to give the upper limb a full range of movement.

**Figure 9.13 Temporomandibular Joint.** The articulation between the head of the mandible and the mandibular fossa of the temporal bone exhibits a wide range of movements.
Several ligaments provide great stability to this joint. The fibrous joint capsule is strengthened superiorly by an acromioclavicular ligament. In addition, a very strong coracoclavicular (kōr′ā-kō-klā-vik′yū-lār) ligament binds the clavicle to the coracoid process of the scapula. If this ligament is torn (as occurs in severe shoulder separations; see Clinical View 9.4: “Shoulder Joint Dislocations”), the acromion and clavicle no longer align properly.

### Table 9.4 Pectoral Girdle and Upper Limb Joints

<table>
<thead>
<tr>
<th>Joint</th>
<th>Articulation Components</th>
<th>Structural Classification</th>
<th>Functional Classification</th>
<th>Description of Movement</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sternoclavicular</strong></td>
<td>Manubrium of sternum and sternal end of clavicle</td>
<td>Synovial (saddle)</td>
<td>Diarthrosis</td>
<td>Depression, elevation, and circumduction</td>
</tr>
<tr>
<td><strong>Acromioclavicular</strong></td>
<td>Acromion of scapula and acromial end of clavicle</td>
<td>Synovial (plane)</td>
<td>Diarthrosis</td>
<td>Gliding of scapula on clavicle</td>
</tr>
<tr>
<td><strong>Glenohumeral</strong></td>
<td>Glenoid cavity of scapula and head of humerus</td>
<td>Synovial (ball-and-socket)</td>
<td>Diarthrosis</td>
<td>Abduction, adduction, circumduction, flexion, extension, lateral rotation, and medial rotation of arm</td>
</tr>
<tr>
<td><strong>Elbow</strong></td>
<td><strong>Humeroulnar joint:</strong> Trochlea of humerus and trochlear notch of ulna</td>
<td>Synovial (hinge)</td>
<td>Diarthrosis</td>
<td>Flexion and extension of forearm</td>
</tr>
<tr>
<td></td>
<td><strong>Humeroradial joint:</strong> Capitulum of humerus and head of radius</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Radioulnar</strong></td>
<td><strong>Proximal joint:</strong> Head of radius and radial notch of ulna</td>
<td>Synovial (pivot)</td>
<td>Diarthrosis</td>
<td>Rotation of radius with respect to ulna</td>
</tr>
<tr>
<td></td>
<td><strong>Distal joint:</strong> Distal end of ulna and ulnar notch of radius</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Radiocarpal</strong></td>
<td>Distal end of radius; scaphoid, lunate, and triquetrum</td>
<td>Synovial (condylar)</td>
<td>Diarthrosis</td>
<td>Abduction, adduction, circumduction, flexion and extension of wrist</td>
</tr>
<tr>
<td><strong>Intercarpal</strong></td>
<td>Adjacent bones in proximal row of carpal bones</td>
<td>Synovial (plane)</td>
<td>Diarthrosis</td>
<td>Gliding</td>
</tr>
<tr>
<td></td>
<td>Adjacent bones in distal row of carpal bones</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Adjacent bones between proximal and distal rows (midcarpal joints)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Carpometacarpal</strong></td>
<td><strong>Thumb:</strong> Trapezium and first metacarpal</td>
<td>Synovial (saddle)</td>
<td>Diarthrosis</td>
<td>Flexion, extension, abduction, adduction, circumduction, and opposition at thumb; gliding at other digits</td>
</tr>
<tr>
<td></td>
<td><strong>Other digits:</strong> Carpals and metacarpals II–V</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Metacarpophalangeal (MP joints, “knuckles”)</strong></td>
<td>Heads of metacarpals and bases of proximal phalanges</td>
<td>Synovial (condylar)</td>
<td>Diarthrosis</td>
<td>Flexion, extension, abduction, adduction, and circumduction of phalanges</td>
</tr>
<tr>
<td><strong>Interphalangeal (IP joints)</strong></td>
<td>Heads of proximal and middle phalanges with bases of middle and distal phalanges, respectively</td>
<td>Synovial (hinge)</td>
<td>Diarthrosis</td>
<td>Flexion and extension of phalanges</td>
</tr>
</tbody>
</table>
**Figure 9.14 Sternoclavicular Joint.** The sternoclavicular joint helps stabilize movements of the entire shoulder.

**INTEGRATE**

**CLINICAL VIEW 9.4**

**Shoulder Joint Dislocations**

Dislocation (dislō-kāˈshən; dis = apart, locatio = placing), a joint injury in which the articulating bones have separated, is common in the shoulder. Although this injury can occur at any of the three shoulder joints, it is more common at either the acromioclavicular joint or the glenohumeral joint than at the sternoclavicular joint.

The term shoulder separation refers to a dislocation of the acromioclavicular joint. This injury often results from a hard blow to the joint, as when a hockey player is “slammed into the boards” or one falls onto the shoulder. Symptoms include tenderness and edema (swelling) in the area of the joint and pain when the arm is abducted more than 90 degrees, because in this position significant movement occurs between the separated bone surfaces. Additionally, the acromion will appear very prominent and pointed. Treatment can range from rest to surgery, depending upon the severity of the dislocation.

A glenohumeral joint dislocation is a very common injury because this joint is very mobile and unstable. This dislocation usually occurs when a fully abducted humerus is struck hard—for example, when a quarterback is hit as he is about to release a football, or when a person falls on an outstretched hand. The initial blow pushes the humerus into the inferior part of the articular capsule and tears the capsule as the humerus dislocates. (The inferior part of the capsule is relatively weak and not protected by muscle tendons as are the other surfaces of the capsule.) Once the humeral head is no longer held in place by the capsule, the anterior thorax (chest) muscles pull superiorly and medially on the humeral head, causing it to lie just inferior to the coracoid process. The result is that the shoulder appears flattened and “squared-off,” because the humeral head is dislocated anteriorly and inferiorly to the glenohumeral joint capsule. Some glenohumeral dislocations can be repaired by “popping” the humerus back into the glenoid cavity. More severe dislocations may need surgical repair.
Figure 9.15 Acromioclavicular and Glenohumeral Joints. (a) Anterior diagrammatic view and cadaver photo of both joints on the right side of the body. (b) Right lateral view and (c) right coronal section show the articulating bones and supporting structures at the shoulder.

(a) ©McGraw-Hill Education/Christine Eckel
Glenohumeral (Shoulder) Joint

The glenohumeral (gle´nō-hyú′mer-āl) joint is commonly referred to as the shoulder joint. It is a ball-and-socket joint formed by the articulation of the head of the humerus and the glenoid cavity of the scapula (figure 9.15). It permits the greatest range of motion of any joint in the body, and so it is both the most unstable joint in the body and the one most frequently dislocated.

The fibrocartilaginous glenoid labrum encircles and covers the surface of the glenoid cavity. A relatively loose articular capsule attaches to the surgical neck of the humerus. The glenohumeral joint has several major ligaments. The coracohumeral (kôr′ā-kō-hú′mer-āl) ligament extends across the space between the coracoid process and the acromion. The large coracohumeral (kôr′ā-kō-hyú′mer-āl) ligament is a thickening of the superior part of the joint capsule. It extends from the coracoid process to the humeral head. The glenohumeral ligaments are three thickenings of the anterior portion of the articular capsule. These ligaments are often indistinct or absent and provide only minimal support. In addition, the tendon of the long head of biceps brachii is within the articular capsule and helps stabilize the humeral head in the joint.

Ligaments of the glenohumeral joint strengthen the joint only minimally. Most of the joint’s strength is due to the rotator cuff muscles surrounding it (see section 11.8b). The rotator cuff muscles (i.e., subscapularis, supraspinatus, infraspinatus, and teres minor) work as a group to hold the head of the humerus in the glenoid cavity. The tendons of these muscles encircle the joint (except for its inferior portion) and fuse with the articular capsule. Because the inferior portion of the joint lacks support from rotator cuff muscles, this area is weak and is the most likely site of injury.

Bursae help decrease friction at the specific places on the shoulder where both tendons and large muscles extend across the joint capsule. The shoulder has a relatively large number of bursae.

WHAT DID YOU LEARN?

18. Why is the shoulder joint considered the most mobile and at the same time the most unstable joint in the human body?

9.7c Elbow Joint

LEARNING OBJECTIVES

27. Describe the elbow joint and its motion.

28. Explain why the elbow joint is relatively stable.

The elbow joint is a hinge joint composed of two articulations: (1) the humeroulnar joint, where the trochlea of the humerus articulates with the trochlear notch of the ulna, and (2) the humeroradial joint, where the capitulum of the humerus articulates with the head of the radius. Both joints are enclosed within a single articular capsule (figure 9.16; table 9.4).

The elbow is an extremely stable joint for several reasons. First, the articular capsule is fairly thick, and thus effectively protects the articulations. Second, the bony surfaces of the humerus and ulna interlock very well, and thus provide a solid bony support. Finally, multiple strong supporting ligaments help reinforce the articular capsule. Because of the tradeoff between stability and mobility, the elbow joint is very stable but is not as mobile as some other joints, such as the glenohumeral joint.

The elbow joint has two main supporting ligaments. The radial collateral ligament (or lateral collateral ligament) is responsible for stabilizing the joint at its lateral surface; it extends around the head of the radius between the anular ligament and the lateral epicondyle of the humerus. The ulnar collateral ligament (or medial collateral ligament) stabilizes the medial side of the joint and extends from the medial epicondyle of the humerus to both the coronoid process and the olecranon of the ulna. In addition, an anular (an′u-lār; anulus = ring) ligament surrounds the neck of the radius and binds the proximal head of the radius to the ulna. The anular ligament helps hold the head of the radius in place.
Despite the support from the capsule and ligaments, the elbow joint is subject to damage from severe impacts or unusual stresses. For example, if you fall on an outstretched hand and the elbow joint is partially flexed, the posterior stress on the ulna combined with contractions of muscles that extend the elbow may break the ulna at the center of the trochlear notch. Sometimes dislocations result from stresses to the elbow. This is particularly true when growth is still occurring at the epiphyseal plate, so children and teenagers may be prone to humeral epicondyle dislocations or fractures.

**WHAT DID YOU LEARN?**

What is the function of the anular ligament in the elbow joint, and what injury may occur to this ligament and joint in young children?
Figure 9.17 Hip Joint. The hip joint is formed by the head of the femur and the acetabulum of the os coxae. The right hip joint is shown in (a) anterior view, (b) posterior view, and (c) coronal section. (d) Cadaver photo of the coxal joint, with the articular capsule cut to show internal structures.

9.7d Hip Joint

LEARNING OBJECTIVES

29. Describe the hip joint and its motions.
30. Explain why the hip joint is more stable than the glenohumeral joint.

The hip joint, also known as the coxal joint, is the articulation between the head of the femur and the relatively deep, concave acetabulum of the os coxae (figure 9.17; table 9.5). A fibrocartilaginous acetabular labrum further deepens this socket. The hip joint’s more extensive bony architecture is therefore much stronger and more...
### Table 9.5 Pelvic Girdle and Lower Limb Joints

<table>
<thead>
<tr>
<th>Joint</th>
<th>Articulation Components</th>
<th>Structural Classification</th>
<th>Functional Classification</th>
<th>Description of Movement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sacroiliac</td>
<td>Auricular surfaces of sacrum and ilium</td>
<td>Synovial (plane)</td>
<td>Diarthrosis</td>
<td>Slight gliding; more movement during pregnancy and childbirth</td>
</tr>
<tr>
<td>Hip (coxal)</td>
<td>Head of femur and acetabulum of os coxae</td>
<td>Synovial (ball-and-socket)</td>
<td>Diarthrosis</td>
<td>Flexion, extension, abduction, adduction, circumduction, medial and lateral rotation of thigh</td>
</tr>
<tr>
<td>Pubic symphysis</td>
<td>Symphyseal surface of both pubic bones</td>
<td>Cartilaginous (symphysis)</td>
<td>Amphiarthrosis</td>
<td>Very slight gliding; more movement during childbirth</td>
</tr>
</tbody>
</table>
| Knee           | **Tibiofemoral joint:** Medial condyle of femur, medial meniscus, and medial condyle of tibia  
                | **Patellofemoral joint:** Patella and patellar surface of femur                           | Synovial (hinge) at tibiofemoral joint,¹ both synovial (hinge) and synovial (plane) at patellofemoral joint | Diarthrosis               | Flexion, extension, lateral rotation of leg in flexed position, slight medial rotation |
| Tibiofibular   | **Superior joint:** Head of fibula and lateral condyle of tibia                           | Synovial (plane)                | Amphiarthrosis             | Slight rotation of fibula during dorsiflexion of foot                                    |
|                | **Inferior joint:** Distal end of fibula and fibular notch of tibia                       | Synovial (plane)                |                           |                                                                                           |
| Talocrural (ankle) | Distal end of tibia and medial malleolus of tibia with talus                             | Synovial (hinge)                | Diarthrosis               | Dorsiflexion and plantar flexion                                                        |
| Intertarsal    | Between the tarsal bones                                                                  | Synovial (plane)                | Diarthrosis               | Eversion and inversion of foot                                                          |
| Tarsometatarsal| Three cuneiforms and cuboid (tarsals) and bases of five metatarsals                      | Synovial (plane)                | Diarthrosis               | Slight gliding                                                                          |
| Metatarsophalangeal (MP) joints | Heads of metatarsals and bases of proximal phalanges                                           | Synovial (condylar)             | Diarthrosis               | Flexion, extension, abduction, adduction, and circumduction of phalanges                |
| Interphalangeal (IP) joints | Heads of proximal and middle phalanges with bases of middle and distal phalanges, respectively | Synovial (hinge)                | Diarthrosis               | Flexion and extension of phalanges                                                      |

¹ Although anatomists classify the tibiofemoral joint as a hinge joint, some kinesiologists and exercise scientists prefer to classify the tibiofemoral joint as a modified condylar joint.
stable than that of the glenohumeral joint. Conversely, the hip joint’s increased stability means that it is less mobile than the glenohumeral joint. The hip joint must be more stable (and thus less mobile) because it supports the body weight.

The hip joint is secured by a strong articular capsule, several ligaments, and a number of powerful muscles. The articular capsule extends from the acetabulum to the troCHANTERS of the femur, enclosing both the femoral head and neck. This arrangement prevents the head from moving away from the acetabulum. The ligamentous fibers of the articular capsule reflect around the neck of the femur. These reflected fibers, called retinacular (ret-i-nak′y-ū-lăr) fibers, provide additional stability to the capsule. Located within the retinacular fibers are retinacular arteries (branches of the deep femoral artery), which supply almost all of the blood to the head and neck of the femur.

The articular capsule is reinforced by three spiraling intracapsular ligaments: The iliofemoral (i′lē-ō-fem′ō-răł) ligament is a Y-shaped ligament that provides strong reinforcement for the anterior region of the articular capsule. The ischiofemoral (i′sē-kē-ō-fem′ō-răł) ligament is a spiral-shaped, and posteriorly located. The pubofemoral (pyū′bō-fem′ō-răł) ligament is a triangular thickening of the capsule’s inferior region. All of these spiraling ligaments become taut when the hip joint is extended, so the hip joint is most stable in the extended position. Try this experiment: Flex your hip joint, and try to move the femur; you may notice a great deal of mobility. Now extend your hip joint (stand up), and try to move the femur. Because those ligaments are taut, you don’t have as much mobility in the joint as you did when the hip joint was flexed.

Another tiny ligament, the ligament of head of femur, also called the ligamentum teres, originates along the acetabulum. Its attachment point is the fovea of the head of the femur. This ligament does not provide strength to the joint; rather, it typically contains a small artery that supplies the head of the femur.

The combination of a deep bony socket, a strong articular capsule, supporting ligaments, and muscular padding gives the hip joint its stability. Movements possible at the hip joint include flexion, extension, abduction, adduction, circumduction, and medial and lateral rotation.

**WHAT DID YOU LEARN?**

- How do the glenohumeral and hip joints compare with respect to their mobility and stability?

**9.7e Knee Joint**

**LEARNING OBJECTIVES**

31. Describe the knee joint and its motion.

32. Name the ligaments that support the knee joint.

The knee joint is the largest and most complex diarthrosis of the body (figure 9.18; table 9.5; see also figure 9.5a). It is primarily a hinge joint, but when the knee is flexed, it is
Figure 9.18 Knee Joint. This joint is the most complex diarthrosis of the body. (a) Anterior superficial, (b) posterior superficial, (c) anterior deep, and (d) posterior deep views reveal the complex interrelationships of the parts of the right knee. Also capable of slight rotation and lateral gliding. Structurally, the knee is composed of two separate articulations: (1) The tibiofemoral (tib-ē-ō-fem′ō-rāl) joint is between the condyles of the femur and the condyles of the tibia, and (2) the patellofemoral joint is between the patella and the patellar surface of the femur.

The knee joint has an articular capsule that encloses only the medial, lateral, and posterior regions of the knee joint. The articular capsule does not cover the anterior surface of the knee joint; rather, the quadriceps femoris muscle tendon passes over the knee joint’s anterior surface. The patella is embedded within this tendon, and the patellar ligament extends beyond the patella and continues to where it attaches on the tibial tuberosity of the tibia. Thus, there is no single unified capsule in the knee, nor is there a common joint cavity. Posteriorly, the capsule is strengthened by several popliteal ligaments.
Knee Ligament and Cartilage Injuries

Although the knee is capable of bearing much weight and has numerous strong supporting ligaments, it is highly vulnerable to injury, especially among athletes. Because the knee is reinforced by tendons and ligaments only, ligamentous injuries to the knee are very common.

The tibial collateral ligament is frequently injured when the leg is forcibly abducted at the knee, such as when a person’s knee is hit on the lateral side. Because the tibial collateral ligament is attached to the medial meniscus, the medial meniscus may be injured as well.

Injury to the fibular collateral ligament can occur if the medial side of the knee is struck, resulting in hyperadduction of the leg at the knee. This type of injury is fairly rare, in part because the fibular collateral ligament is very strong and because medial blows to the knee are not common.

The anterior cruciate ligament (ACL) can be injured when the leg is hyperextended—for example, if a runner’s foot hits a hole. Because the ACL is rather weak compared to the other knee ligaments, it is especially prone to injury. To test for ACL injury, a physician gently tugs anteriorly on the tibia. In this so-called anterior drawer test, too much forward movement indicates an ACL tear.

Posterior cruciate ligament (PCL) injury may occur if the leg is hyperflexed or if the tibia is driven posteriorly on the femur. PCL injury occurs rarely, because this ligament is rather strong. To test for PCL injury, a physician gently pushes posteriorly on the tibia. In this posterior drawer test, too much posterior movement indicates a PCL tear.

The menisci also may be prone to injury. Tears in the menisci may occur due to blows to the knee or due to general overuse of the joint. Because the menisci are composed of fibrocartilage, they cannot regenerate and often must be surgically treated.

The unhappy triad of injuries refers to a triple injury of the tibial collateral ligament, medial meniscus, and anterior cruciate ligament. This is the most common type of football injury. It occurs when a player is illegally “clipped” by a lateral blow to the knee, and the leg is forcibly abducted and laterally rotated. If the blow is severe enough, the tibial collateral ligament tears, followed by tearing of the medial meniscus, as these two structures are connected. The force that tears the tibial collateral ligament and the medial meniscus is thus transferred to the ACL. Because the ACL is relatively weak, it tears as well.

The treatment of ligamentous knee injuries depends upon the severity and type of injury. Conservative treatment involves immobilizing the knee for a period of time to rest the joint. Surgical treatment can include repairing the torn ligaments or replacing the ligaments with a graft from another tendon or ligament (such as the quadriceps tendon). Many knee surgeries may be performed with arthroscopy. Arthroscopy is a type of conservative surgical treatment where a small incision is made in the knee and then an arthroscope (an instrument with a camera and light source) is inserted into the knee, allowing the surgeon to clearly see the surgical area without having to make large incisions.

“Unhappy triad” of injuries to the right knee.
On either side of the knee joint are two collateral ligaments that become taut on extension and provide additional stability to the joint. The fibular collateral ligament (lateral collateral ligament) reinforces the lateral surface of the joint. This ligament extends from the femur to the fibula and prevents hyperadduction of the leg at the knee. (In other words, it prevents the leg from moving too far medially relative to the thigh.) The tibial collateral ligament (medial collateral ligament) reinforces the medial surface of the knee joint. This ligament runs from the femur to the tibia and prevents hyperabduction of the leg at the knee. (In other words, it prevents the leg from moving too far laterally relative to the thigh.) This ligament is attached to the medial meniscus of the knee joint as well, so an injury to the tibial collateral ligament usually affects the medial meniscus.

Deep to the articular capsule and within the knee joint itself are a pair of C-shaped fibrocartilage pads positioned on the condyles of the tibia. These pads are called the medial meniscus and the lateral meniscus. They partially stabilize the joint medially and laterally, act as cushions between articular surfaces, and continuously change shape to conform to the articulating surfaces as the femur moves.

Two cruciate (krūˈshē-ət) ligaments are deep to the articular capsule of the knee joint. They limit the anterior and posterior movement of the femur on the tibia. These ligaments cross each other in the form of an X, hence the name cruciate (which means cross). The anterior cruciate ligament (ACL) extends from the posterior femur to the anterior side of the tibia. When the knee is extended, the ACL is pulled tight and prevents hyper-extension. The ACL prevents the tibia from moving too far anteriorly relative to the femur. The posterior cruciate ligament (PCL) attaches from the anteroinferior femur to the posterior side of the tibia. The PCL becomes taut on flexion, and so it prevents hyperflexion of the knee joint. The PCL also prevents posterior displacement of the tibia relative to the femur.

Humans are bipedal, meaning that we walk on two feet. An important aspect of bipedal locomotion is the ability to “lock” the knees in the extended position and stand erect without tiring the leg muscles. At full extension, the tibia rotates laterally so as to tighten the anterior cruciate ligament and squeeze the menisci between the tibia and femur. Muscular contraction by the popliteus muscle (see section 11.9c) unlocks and flexes the knee joint. (This ability should be distinguished from being unable to bend or straighten the knee because of injury or disease, which is sometimes also called locking.)

**WHAT DID YOU LEARN?**

21. What are the functions of each of the intracapsular ligaments of the knee joint?

---

### 9.7f Talocrural (Ankle) Joint

**LEARNING OBJECTIVE**

33. Describe the talocrural joint and its motion.

The talocrural (ankle) joint is a highly modified hinge joint that permits both dorsiflexion and plantar flexion. It includes two articulations within one joint capsule. One articulation is between the distal end of the tibia and the talus; the other is between the distal end of the fibula and the lateral aspect of the talus (figure 9.19; table 9.5). The medial and lateral malleoli of the tibia and fibula, respectively, form extensive medial and lateral margins and prevent the talus from sliding side-to-side.

The talocrural joint includes several distinctive anatomic features. Its articular capsule covers the distal surfaces of the tibia, the medial malleolus, the lateral malleolus, and the talus. A multipart deltoid ligament (or medial ligament) binds the tibia to the foot on the medial side. This ligament prevents overeversion of the foot. It is incredibly strong and rarely tears; in fact, it typically will pull off the medial malleolus before it ever tears (see Clinical View 9.8: “Ankle Sprains and Pott Fractures”). A much thinner, multipart lateral ligament binds the fibula to the foot on the lateral side. This ligament prevents overinversion of the foot. It is not as strong as the deltoid ligament and is prone to sprains and tears. Two tibiofibular (tibˈē-ə-fibˈyə-lār) ligaments (anterior and posterior) bind the tibia to the fibula.

**WHAT DID YOU LEARN?**

22. What bones articulate at the talocrural joint, and what movements are permitted at this joint?
Ankle Sprains and Pott Fractures

A sprain is a stretching or tearing of ligaments, without fracture or dislocation of the joint. An ankle sprain results from twisting of the foot, almost always due to overinversion. Fibers of the lateral ligament are either stretched (in mild sprains) or torn (in more severe sprains), producing localized swelling and tenderness anteroinferior to the lateral malleolus. Overeversion sprains rarely occur due to the strength of the deltoid (medial) ligament. Recall that ligaments are composed of dense regular connective tissue, which is poorly vascularized (see section 5.2d). Poorly vascularized tissue takes a long time to heal, and that is the case with ankle sprains. These structures are also prone to reinjury.

If overeversion does occur, the injury that usually results is called a Pott fracture (see section 7.8). If the foot is overeverted, it pulls on the deltoid ligament, which is very strong and doesn’t tear. Instead, the pull can avulse (pull off) the medial malleolus of the tibia. The force from the injury then continues to move the talus laterally, as the medial malleolus can no longer restrict side-to-side movements of the ankle. As the talus moves laterally and puts force on the fibula, the fibula fractures as well (usually at its distal end or by the lateral malleolus). Thus, both the tibia and the fibula fracture in this injury, yet the deltoid ligament remains intact.

Figure 9.19 Talocrural Joint. (a) Lateral and (b) medial views of the right foot show that the talocrural joint contains articulations among the tibia, fibula, and talus. This joint permits dorsiflexion and plantar flexion only.
Joints start to form by the sixth week of development and progressively differentiate during the fetal period. In the area of future fibrous joints, the mesenchyme around the developing bone differentiates into dense regular connective tissue, whereas in cartilaginous joints it differentiates either into fibrocartilage or hyaline cartilage.

The development of the synovial joints is more complex. The most laterally placed mesenchyme forms the articular capsule and supporting ligaments of the joint. Just medial to this region, the mesenchyme forms the synovial membrane, which then starts secreting synovial fluid into the joint cavity. The centrally located mesenchyme may be reabsorbed or can form menisci or articular discs, depending upon the type of synovial joint.

Prior to the closure of the epiphyseal plates, some injuries to a young person may result in subluxation or fracture of an epiphysis, with potential adverse effects on the future development and health of the joint; the bone may not reach its potential full length, or the individual may develop arthritic-like changes in the joint.

Arthritis is a rheumatic (i.e., referring to the joints or muscles) disease that involves damage to articular cartilage (see Clinical View 9.9: “Arthritis”). The primary problem that develops in
Articulations are the joints where bones are in contact. Joints differ in structure, function, and the amount of movement they allow.

9.1 Classification of Joints
- The three structural categories of joints are fibrous, cartilaginous, and synovial.
- The three functional categories of joints are synarthroses (immobile joints), amphiarthroses (slightly mobile joints), and diarthroses (freely mobile joints).

9.2 Fibrous Joints
- Fibrous joints lack a joint cavity, and they interconnect articulating bones by dense regular connective tissue.
  9.2a Gomphoses
  - A gomphosis is a synarthrosis between the tooth and either the mandible or the maxillae.
  9.2b Sutures
  - A suture is a synarthrosis that tightly binds the bones of the skull. Fused sutures are called synostoses.
  9.2c Syndesmoses
  - A syndesmosis is an amphiarthrosis, and the bones are connected by interosseous membranes.

9.3 Cartilaginous Joints
- Cartilaginous joints lack a joint cavity; the cartilage between the articulating bones may be either hyaline cartilage or fibrocartilage.
  9.3a Synchondroses
  - A synchondrosis is a synarthrosis where hyaline cartilage is wedged between the articulating bones.
  9.3b Symphyses
  - A symphysis is an amphiarthrosis, where a disc of fibrocartilage is wedged between the articulating bones.

9.4 Synovial Joints
- All synovial joints are diarthroses.
  9.4a Distinguishing Features and Anatomy of Synovial Joints
  - Synovial joints contain an articular capsule, a joint cavity, synovial fluid, articular cartilage, ligaments, nerves, and blood vessels.
  9.4b Classification of Synovial Joints
  - The six types of synovial joints are plane, hinge, pivot, condylar, saddle, and ball-and-socket.

9.5 The Movements of Synovial Joints
- Motions that occur at synovial joints include gliding, angular, rotational, and special.
  9.5a Gliding Motion
  - Gliding is a simple movement where two opposing surfaces slide back-and-forth or side-to-side against one another.
  9.5b Angular Motion
  - Angular movements involve decreasing or increasing the angle of a joint. Angular movements include flexion, extension, hyperextension, lateral flexion, abduction, adduction, and circumduction.
  9.5c Rotational Motion
  - Rotational movements involve a pivoting motion. Examples of rotational movements are lateral rotation, medial rotation, pronation, and supination.
  9.5d Special Movements
  - Special movements include depression and elevation, dorsiflexion and plantar flexion, eversion and inversion, protraction and retraction, and opposition.

WHAT DID YOU LEARN?

What are some ways that joints change as a person ages?

(continued on next page)
### 9.6 Synovial Joints and Levers

- Biomechanics is the practice of applying mechanical principles to biology.

#### 9.6a Terminology of Levers
- Synovial joints may be compared to levers, which have a fixed point (fulcrum) around which movement occurs when effort applied to one point exceeds resistance at another point.

#### 9.6b Types of Levers
- A first-class lever has a fulcrum between the effort and the resistance.
- A second-class lever has the resistance placed between the fulcrum and the effort.
- A third-class lever, the most common type of lever in the human body, has the effort applied between the resistance and the fulcrum.

### 9.7 Features and Anatomy of Selected Joints

- In each articulation, unique features of the articulating bones support the intended movements.

#### 9.7a Temporomandibular Joint
- The temporomandibular joint is an articulation between the head of the mandible and the mandibular fossa of the temporal bone.

#### 9.7b Shoulder Joint
- The sternoclavicular joint and acromioclavicular joint support movement of the shoulder.
- The glenohumeral joint is a ball-and-socket joint between the glenoid cavity of the scapula and the head of the humerus.

#### 9.7c Elbow Joint
- The elbow is a hinge joint among the humerus, radius, and ulna.

#### 9.7d Hip Joint
- The hip joint is a ball-and-socket joint between the head of the femur and the acetabulum of the os coxae.

#### 9.7e Knee Joint
- The knee joint is primarily a hinge joint but is capable of slight rotation and gliding.

#### 9.7f Talocrural (Ankle) Joint
- The talocrural joint is a hinge joint that permits dorsiflexion and plantar flexion of the ankle.

### 9.8 Development and Aging of the Joints

- Joints begin to form during week 6 of development.
- Osteoarthritis is a common joint problem that occurs with aging.

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### CHALLENGE YOURSELF

**Do You Know the Basics?**

1. The greatest range of mobility of any joint in the body is found in the
   a. knee joint.
   b. hip joint.
   c. glenohumeral joint.
   d. elbow joint.

2. A movement of the foot that turns the sole outward or laterally is called
   a. dorsiflexion.
   b. inversion.
   c. eversion.
   d. plantar flexion.

3. A _____ is formed when two bones previously connected by a suture fuse.
   a. gomphosis
   b. synostosis
   c. symphysis
   d. syndesmosis

4. The ligament that helps to maintain the alignment of the condyles between the femur and tibia and to limit the anterior movement of the tibia on the femur is the
   a. tibial collateral ligament.
   b. posterior cruciate ligament.
   c. anterior cruciate ligament.
   d. fibular collateral ligament.

5. Which joint is a diarthrosis?
   a. symphysis
   b. synchondrosis
   c. syndesmosis
   d. saddle

6. In this type of lever, the effort is located between the resistance and the fulcrum. An example would be your knee joint.
   a. first-class
   b. second-class
   c. third-class
   d. Two of the above are correct.
7. A metacarpophalangeal (MP) joint, which has oval articulating surfaces and permits movement in two planes, is what type of synovial joint?
   a. condylar
   b. plane
   c. hinge
   d. saddle
8. All of the following ligaments provide stability to the hip joint except the
   a. ischiofemoral ligament.
   b. pubofemoral ligament.
   c. iliofemoral ligament.
   d. ligament of the head of the femur.
9. Which of the following is a function of synovial fluid?
   a. lubricates the joint
   b. provides nutrients for the articular cartilage
   c. absorbs shock within the joint
   d. All of these are correct.
10. Plantar flexion and dorsiflexion are movements permitted at the __________ joint.
    a. hip
    b. knee
    c. sternoclavicular
    d. talocrural
11. Discuss the factors that influence both the stability and mobility of a joint. What is the relationship between a joint’s mobility and its stability?
12. Both fibrous joints and synovial joints have dense regular connective tissue holding the bones together. So how are these two joints different, both structurally and functionally?
13. List and describe all joints that are functionally classified as synarthroses.
14. How do a hinge joint and a pivot joint compare with respect to structure, function, and location within the body?
15. Compare and contrast first-, second-, and third-class levers.
16. Describe and compare the movements of abduction, adduction, pronation, and supination.
17. Most ankle sprains are overinversion injuries. What are the anatomic reasons that overeversion ankle sprains are relatively uncommon? Are there any overeversion injuries that occur to the ankle?
18. What are the main supporting ligaments of the elbow joint?
19. Compare the functions of the tibial and the fibular collateral ligaments in the knee joint. Which of the two is injured more frequently and why?
20. What are the similarities and differences between osteoarthritis and rheumatoid arthritis?

⚠️ Can You Synthesize What You’ve Learned?

1. During soccer practice, Erin tripped over the outstretched leg of a teammate and fell directly onto her shoulder. She was taken to the hospital in excruciating pain. Examination revealed that the head of the humerus had moved inferiorly and anteriorly into the axilla. What happened to Erin in this injury?
   a. anular ligament
   b. ulnar collateral ligament
   c. radial collateral ligament
   d. coronoid ligament
2. The doctor mentions that this type of injury is common in children younger than 5 years of age. What is one reason for this?
   a. The olecranon of the ulna does not fit in properly with the olecranon fossa of the radius.
   b. The head of the radius is not fully formed.
   c. The medial and lateral epicondyle epiphyseal plates have not yet fused to the rest of the humerus.
   d. The articular capsule of the elbow joint is weak in its anterior surface.
3. What bony feature caused the prominent bump on the lateral side of the elbow?
   a. lateral epicondyle of the humerus
   b. coronoid process of the ulna
   c. head of the radius
   d. radial collateral ligament
4. While Robert was running, he stepped into a pothole and twisted his right ankle. A swelling appeared along the lateral side of this ankle. Which ligament was injured, and what movement resulted in the injury?
   a. deltoid ligament, caused by overeversion of the foot
   b. lateral ligament, caused by overeversion of the foot
   c. deltoid ligament, caused by overinversion of the foot
   d. lateral ligament, caused by overinversion of the foot
5. Most knee ligaments become taut upon extension of the joint, except for one. Which knee ligament becomes taut upon flexion of the joint and prevents hyperflexion of the joint?
   a. anterior cruciate ligament
   b. posterior cruciate ligament
   c. patellar ligament
   d. tibial collateral ligament

⚠️ Can You Apply What You’ve Learned?

Use the following paragraph to answer questions 1–3.

A mother and her 4-year-old son were visiting a toy store, and the child did not want to leave. As the child threw a temper tantrum and resisted his mother, the mother pulled on the boy’s arm to lead him out of the store. Immediately after the pull, the boy cried out in pain and displayed a prominent bump on the lateral side of the elbow. The mother drove the boy to the doctor in a panic. The doctor examined the boy’s elbow and determined the boy had a subluxated head of the radius.

1. Which ligament failed to keep the head of the radius in place when the mother pulled on the boy’s elbow?
   a. anular ligament
   b. ulnar collateral ligament
   c. radial collateral ligament
   d. coronoid ligament
2. The mother drove the boy to the doctor in a panic. The doctor determined the boy had______.
   a. a flexed elbow
   b. a dislocated elbow
   c. a fractured elbow
   d. a sprained elbow
3. After the doctor examined the boy’s elbow and determined the boy had______, the boy was taken immediately to the hospital. The doctor determined the boy had a subluxated head of the radius.
   a. a flexed elbow
   b. a dislocated elbow
   c. a fractured elbow
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and what kinds of injuries can occur if a player gets clipped?

3. Jackie visits her physician because she is experiencing pain by her right ear. The doctor checks her ears and sees no sign of infection. She asks Jackie to open and close her mouth while she palpates the portions of her face adjacent to her ears. Why is the doctor having Jackie move her mouth, when she is experiencing ear pain? How may the two be related? What do you think the doctor will discover when Jackie opens and closes her mouth?

**INTEGRATE**

**ONLINE STUDY TOOLS**

The following study aids may be accessed through Connect.

- **Clinical Case Study:** A Historical Case of Spinal Involvement with Tuberculosis
- **Interactive Questions:** This chapter’s content is served up in a number of multimedia question formats for student study
- **SmartBook:** Topics and terminology include classification of joints; fibrous joints; cartilaginous joints; synovial joints; synovial joints and levers; movements of synovial joints; features and anatomy of selected joints; development and aging of joints
- **Anatomy & Physiology Revealed:** Topics include skull, synovial joint; temporomandibular joint; glenohumeral joint; elbow joint; hand; hip joint; knee joint; tibiofibular joint
- **Animation:** Synovial joints

When we hear the word muscle, most of us think of the muscles that move the skeleton. Over 700 skeletal muscles have been named, and together they form the muscular system. Skeletal muscles, however, are not the only places where muscle tissue is found. Muscle tissue is distributed almost everywhere in the body and is responsible for the movement of materials within and throughout the body. This vital tissue propels the food we eat through the gastrointestinal tract, expels the waste products we produce, adjusts the diameter of blood vessels to regulate blood pressure, and pumps blood to body tissues.

The three types of muscle tissue—skeletal muscle, cardiac muscle, and smooth muscle—were first introduced and compared in section 5.3. Here we describe the details of skeletal muscle anatomy and physiology. The chapter finishes with a brief description of cardiac muscle (which is described in detail in section 19.3f) and a general discussion about smooth muscle. The structure and actions of individual skeletal muscles of the muscular system are explained in chapter 11.
10.1 Introduction to Skeletal Muscle

Skeletal muscle typically composes 40–50% of the weight of a healthy adult. It is primarily attached to the skeleton but is also found, for example, at the openings of the gastrointestinal and urinary tracts. We begin our discussion on skeletal muscle by describing both its general functions and the characteristics of the skeletal muscle cells that primarily compose it.

10.1a Functions of Skeletal Muscle

**LEARNING OBJECTIVE**

1. Explain the five general functions of skeletal muscle.

The hundreds of skeletal muscles within your body perform a wide range of functions. These include:

- **Body movement.** Contraction of your skeletal muscles generates large body movements, such as those of walking, and the smaller, more precise body movements such as picking up an object. It is also responsible for the highly developed movements involved in communicating that occur when speaking, writing, and changing facial expressions; the movements associated with breathing (see section 23.5b); and those involved in the voluntary phase of swallowing (see section 26.2c).

- **Maintenance of posture.** Contraction of specific skeletal muscles stabilizes your trunk, pelvis, legs, neck, and head to keep you erect. These postural muscles contract continuously when you are awake to keep you from collapsing.

- **Protection and support.** Skeletal muscle is arranged in layers within the walls of the abdominal cavity (see figure 11.16) and the floor of the pelvic cavity (see figure 11.17). These layers of muscle protect the internal organs and support their normal position within the abdominopelvic cavity.

- **Regulating elimination of materials.** Circular muscle bands, called sphincters (sfing'kter; sphincter = a band) contract and relax to regulate passage of material. These skeletal muscle sphincters at the orifices (or′i-fis; orificium = opening) of the gastrointestinal and urinary tracts allow you to voluntarily control the expulsion of feces and urine, respectively (see figures 26.23b and 24.28).

- **Heat production.** Energy is required for muscle tissue contraction, and heat is always produced by this energy use (the second law of thermodynamics; see section 3.1c). Thus, muscles are like small furnaces that continuously generate heat and function to help maintain your normal body temperature. You shiver when you are cold because involuntary skeletal muscle contraction gives off heat. Likewise, you sweat during exercise to release the additional heat produced by your working muscles (see sections 1.6b and 6.1d).

**WHAT DID YOU LEARN?**

1. What are the five major functions of skeletal muscle?

10.1b Characteristics of Skeletal Muscle

**LEARNING OBJECTIVE**

2. Describe the five characteristics of skeletal muscle.

Skeletal muscle is composed primarily of muscle cells that exhibit these characteristics—excitability, conductivity, contractility, extensibility, and elasticity:

- **Excitability** is the ability of a cell to respond to a stimulus (e.g., chemical, stretch). The stimulus causes a local change in the resting membrane potential (see section 4.4) by triggering the movement of ions across the plasma membrane of the excitable cell. A skeletal muscle cell responds when its receptors bind neurotransmitter (acetylcholine), which is released from a motor neuron (see section 10.3a).

- **Conductivity** involves an electrical signal that is propagated along the plasma membrane as voltage-gated channels open sequentially during an action potential. These electrical signals functionally connect the plasma membrane of the muscle cell (where stimulation occurs) to the interior of the muscle cell (where contraction occurs; see section 10.3b).

- **Contractility** is exhibited when contractile proteins within skeletal muscle cells slide past one another. Contractility is what enables muscle cells to cause body movement and to perform the other functions of muscles (see section 10.3c). The excitability, conductivity, and contractility of muscle cells may be viewed collectively in figure 10.9 at step 1, step 2, and step 3, respectively.

- **Extensibility** is the lengthening of a muscle cell (see figure 10.25). This lengthening is possible because the contractile proteins slide past one another to decrease their degree of overlap. Muscle’s extensibility is exhibited when we stretch our muscles, such as before exercising.

- **Elasticity** is the ability of a muscle cell to return to its original length following either shortening or lengthening of the muscle. Elasticity of muscle cells is dependent upon the release of tension in the springlike connectin protein associated with contractile proteins (see figure 10.5b).

**WHAT DID YOU LEARN?**

2. Explain the skeletal muscle characteristics of contractility, extensibility, and elasticity. How do these differ?

**CONCEPT CONNECTION**

The characteristic of excitability is exhibited in other body cells—such as nerve cells, called neurons, which may respond to a neurotransmitter (see section 12.8a) and sensory receptors that respond to a specific type of sensory stimulus (see section 16.1a).
10.2 Anatomy of Skeletal Muscle

A single muscle, such as the gracilis muscle on the medial thigh (see figure 11.1), may be composed of thousands of muscle cells that are typically as long as the entire muscle. Because of their potentially extraordinary length, skeletal muscle cells are often referred to as muscle fibers (or myofibers). Here we describe the gross anatomy of skeletal muscle, the microscopic anatomy of individual skeletal muscle fibers, and innervation of skeletal muscle fibers.

10.2a Gross Anatomy of Skeletal Muscle

LEARNING OBJECTIVES

3. Identify and describe the three connective tissue layers associated with a skeletal muscle.
4. Describe the structure and function of a tendon and an aponeurosis.
5. Explain the function of blood vessels and nerves serving a muscle.

A skeletal muscle is an organ, which, recall from section 1.4b, is two or more types of tissue that work together to perform a specific function. Each skeletal muscle is composed of skeletal muscle fibers, connective tissue layers, blood vessels, and nerves. The organization of a muscle is shown in figure 10.1. Observe the specific anatomic arrangement of muscle fibers within a muscle. Notice that many muscle fibers are bundled within a fascicle (fas’i-kl; fascis = bundle); and many fascicles are bundled within the whole skeletal muscle.

WHAT DO YOU THINK?

List the structures of skeletal muscle from largest to smallest: muscle fiber, muscle, and fascicle.

Connective Tissue Components

Three layers of connective tissue are within muscles: the epimysium, the perimysium, and the endomysium. These layers provide protection and support, a means of attachment of the muscle to the skeleton or other structures within the body, and sites for distribution of blood vessels and nerves:

- The epimysium (ep-i-mis’e-ūm; epi = upon, mys = muscle) is a layer of dense irregular connective tissue (see section 5.2d) that surrounds the whole skeletal muscle. This fibrous tissue enshews the entire skeletal muscle to protect and support it like a tough leather sleeve.
- The perimysium (per-i-mis’e-ūm; peri = around) is a layer of dense irregular connective tissue around each fascicle. These tough, fibrous connective tissue sleeves also provide protection and support, but to each bundle of muscle fibers.
- The endomysium (en’dō-mis’e-ūm; endon = within) is composed of areolar connective tissue that surrounds each muscle fiber. These more delicate coverings function to electrically insulate the muscle fibers.

The epimysium, perimysium, and endomysium collectively extend past the muscle fibers to form either a tendon or an aponeurosis. A tendon is a thick, cordlike structure composed of dense regular connective tissue, whereas an aponeurosis (ap’ō-nū-rō’sis; apo = from, neuron = sinew) is a thin, flattened sheet of dense regular connective tissue (see figures 11.5 and 11.16). Both tendons and aponeuroses attach a muscle either to a skeletal component (bone or ligament) or to fascia (described next). Imagine the typical scenario...
as skeletal muscle fibers contract, pulling on the connective tissue sheaths, with the force transferred to a tendon that moves a bone.

**Deep fascia** (fash’ér-ē; band or filler), also called visceral or muscular fascia, is an additional, expansive sheet of dense irregular connective tissue that is external to the epimysium. Deep fascia separates individual muscles; binds together muscles with similar functions; contains nerves, blood vessels, and lymph vessels; and fills spaces between muscles. The deep fascia is internal or deep to a layer called the superficial fascia (or subcutaneous layer; see section 6.1c). The superficial fascia is composed of areolar connective tissue and adipose connective tissue that separates muscle from skin.

**Blood Vessels and Nerves**

Skeletal muscle is *vascularized* (vas′kyū-lər; vas = vessel) by an extensive network of blood vessels. The blood vessels extend through both the epimysium and the perimysium to reach the endomysium, which ensheathes each muscle fiber. The smallest blood vessels called, *capillaries* (see section 20.1c), are associated with the endomysium and function as the site of exchange of substances (e.g., oxygen, glucose, waste products) between the blood and the skeletal muscle fibers (see capillary exchange in section 20.3).

Skeletal muscle is *innervated* (in′ə-vā′tēd; nervus = nerve) by motor neurons (of the somatic nervous system; see section 12.1b), which control skeletal muscle contraction. **Somatic motor neurons** extend from the brain and spinal cord to skeletal muscle fibers. Each motor neuron has a long extension called an *axon* (nerve fiber) that branches extensively at its terminal end (see section 12.2b). The axon extends through all three connective tissue layers to almost make contact with an individual muscle fiber (i.e., there is a very small gap of about 30 nanometers between the motor neuron and muscle fiber). The junction between the axon and the muscle fiber itself is called a *neuromuscular junction*, which is discussed in section 10.2c. Skeletal muscle is classified as *voluntary muscle* because the skeletal muscle fibers can be consciously controlled by the nervous system.

**WHAT DID YOU LEARN?**

3. Identify the location and function of these connective tissue structures associated with muscle: endomysium, perimysium, epimysium, deep fascia, and superficial fascia.

**10.2b Microscopic Anatomy of Skeletal Muscle**

**LEARNING OBJECTIVES**

6. Explain how a skeletal muscle fiber becomes multinucleated.

7. Describe the sarcolemma, T-tubules, sarcoplasmic reticulum, and triad of a skeletal muscle fiber.

8. Distinguish between thick and thin filaments.

9. Explain the organization of myofilaments, myofilaments, and sarcomeres.

10. List and describe the structures associated with energy production within skeletal muscle fibers.

Skeletal muscle fibers (as shown in figure 10.1) are the primary cells forming a skeletal muscle. Skeletal muscle fibers, like other cells, contain cytoplasm with the typical cellular structures, such as the Golgi apparatus, ribosomes, and vesicles (see section 4.6). Note that the cytoplasm in skeletal muscle fibers is more specifically called sarcoplasm (sar′kō-plazm; sark = flesh). In addition, skeletal muscle fibers have several specialized features that we describe here, including the details of its contractile proteins.

**A Multinucleated Cell**

A skeletal muscle fiber is typically between 10 and 500 micrometers (µm) in diameter and may extend the length of the entire muscle, ranging from about 100 µm to 30 centimeters. To reach this length, groups of embryonic muscle cells termed *myoblasts* (mi′ō-blast; blastos = germ) fuse to form single skeletal muscle fibers during development (figure 10.2). During this fusion process, each myoblast nucleus contributes to the eventual total number of nuclei in the fiber. Consequently, skeletal muscle fibers are *multinucleated* (i.e., they have numerous nuclei) (figure 10.3a).

Some myoblasts do not fuse with muscle fibers during development and instead remain in adult skeletal muscle tissue as satellite cells (figure 10.2), which are adult stem cells (see Clinical View 5.4: “Stem Cells”). If a skeletal muscle is injured, some satellite cells may be
Figure 10.3 Structure and Organization of a Skeletal Muscle Fiber. (a) A skeletal muscle fiber is composed predominantly of myofibrils, which extend the length of the muscle fiber. (b) A myofibril is composed of bundles of myofilaments (protein filaments) and is enclosed within segments of the sarcoplasmic reticulum. The sarcoplasmic reticulum is a reservoir for calcium ions (Ca\(^{2+}\)). (c) The sarcolemma is physically connected to the sarcoplasmic reticulum by invaginations of the sarcolemma called T-tubules; both contain voltage-gated Na\(^+\) channels and voltage-gated K\(^+\) channels. (d) The triad is a T-tubule flanked by two terminal cisternae of the sarcoplasmic reticulum. In this region, the T-tubule membrane contains voltage-sensitive Ca\(^{2+}\) channels and the terminal cisternae membrane contains both Ca\(^{2+}\) release channels and Ca\(^{2+}\) pumps.
stimulated to differentiate and then fuse with a damaged skeletal muscle fiber to assist to a limited extent in its repair and regeneration.

**Sarcolemma and T-tubules**

The plasma membrane of a skeletal muscle fiber is called the sarcolemma (sar’kō-ləm’ə; lemma = husk) (figure 10.3a). Deep invaginations of the sarcolemma, called T-tubules or transverse (trans-vers’; trans = across, versus = to turn) tubules, extend into the skeletal muscle fiber as a network of narrow, membranous tubules to the sarcoplasmic reticulum, which is the endoplasmic reticulum (ER) of the muscle (described shortly). Located within the membrane of both the sarcolemma (along its length) and the T-tubules are voltage-gated channels (figure 10.3c). These channels include both voltage-gated Na⁺ channels and voltage-gated K⁺ channels, which participate in conducting an electrical signal (an action potential) as described in section 10.3b. (Note: Channels within the plasma membrane are first introduced in section 4.3a, and voltage-gated and chemically gated channels are discussed in detail in section 12.6a.)

**Myofibrils**

Approximately 80% of the volume of a skeletal muscle fiber is composed of long, cylindrical structures termed myofibrils (mī’ō-fr’il) (figure 10.3a). A skeletal muscle fiber contains hundreds to thousands of myofibrils. Each myofibril extends the entire length of the skeletal muscle fiber (and is about 1 to 2 micrometers in diameter). Note that each myofibril is composed of bundles of contractile proteins called myofilaments and is enclosed in portions of the sarcoplasmic reticulum (figure 10.3b).

**Sarcoplasmic Reticulum**

The sarcoplasmic reticulum (sar-kō’plaz’mik re-tik’ə-lūm; rete = a net) is an internal membrane complex that is similar to the smooth endoplasmic reticulum of other cells (see section 4.6a). Segments of the sarcoplasmic reticulum (SR) fit around the myofibril like a sleeve of membrane netting. At either end of individual sections of the sarcoplasmic reticulum are blind sacs called terminal cisternae (sis-ter’nē; sing., sis-ter’nā; cista = a box), which are much like the hem of a sleeve. Terminal cisternae serve as the reservoirs for calcium ions (Ca²⁺) and are immediately adjacent to each T-tubule (figure 10.3d). Together, two terminal cisternae and a centrally located T-tubule form a structure called a triad. Within the triad, the T-tubule membrane contains voltage-sensitive Ca²⁺ channels (dihydropyridine receptors), which are responsive to electrical signals (i.e., action potentials). The terminal cisterna membrane of the sarcoplasmic reticulum contain Ca²⁺ release channels (ryanodine receptors). It is here in the triad that the connection occurs between the electrical signals (action potentials) propagated along the sarcolemma and T-tubule and the release of calcium from the sarcoplasmic reticulum (see section 10.3b). This release of Ca²⁺ initiates muscle contraction, as described in section 10.3c.

Also embedded within the membrane of the sarcoplasmic reticulum are Ca²⁺ pumps, which move Ca²⁺ from the cytosol into the sarcoplasmic reticulum, where it is stored bound to specialized proteins called calmodulin (kal-mod’ə-lin) and calsequestrin (kal’se-kwes’trin). Calcium pumps function through primary active transport (see section 4.3c) to maintain low cytosol levels of calcium. These pumps return Ca²⁺ to the terminal cisternae of the sarcoplasmic reticulum following its release to initiate muscle contraction.

**Myofilaments**

Myofilaments (mī’ō-fil’ə-mənt; filum = thread) are contractile proteins that are bundled within myofibrils (figure 10.3b). A myofibril is not as long as a myofibril; rather, it takes many successive units of myofilaments to extend the entire length of the myofibril. Myofibril bundles contain two types of myofilaments: thick filaments and thin filaments (figure 10.4).

**Thick Filaments** Thick filaments (or thick myofilaments) are assembled from bundles of 200 to 500 myosin protein molecules (figure 10.4a). Each myosin protein consists of two strands; each strand has a globular head and an elongated tail. The myosin head contains a binding site for actin of the thin filaments. The head also has a catalytic ATPase site where adenosine triphosphate (ATP) attaches and is split into

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**Figure 10.4 Molecular Structure of Thick and Thin Filaments.**

Myofilaments, which include thick filaments and thin filaments, are the contractile proteins bundled within myofibrils. (a) A thick filament consists of 200 to 500 myosin protein molecules. (b) A thin filament is composed of actin, tropomyosin, and troponin proteins.
adenosine diphosphate (ADP) and phosphate (P$_i$). (It is because the head of myosin functions as an ATPase enzyme that myosin is often referred to more specifically as myosin ATPase.) The tails of two strands of a myosin molecule are intertwined. Each myosin molecule composing a thick filament is oriented so that its tails point toward the center of the thick filaments and its heads point toward the ends of the thick filaments. You may find it helpful to think of the myosin protein molecules as two intertwined golf clubs, where many are grouped together with the golf club shafts in the center and the club heads on each end.

**Thin Filaments** Thin filaments (or thin myofilaments) are approximately half of the diameter of thick filaments (about 5 to 6 nanometers). Thin filaments are primarily composed of two strands of actin protein twisted around each other to form a helical shape (figure 10.4b). In each strand of actin, many (about 300 to 400) small, spherical molecules (G, or globular, actin) are connected to form a fibrous strand (F, or filamentous, actin). F-actin resembles two beaded necklaces that are twisted and intertwined together, with G-actin as the individual beads. Each G-actin molecule has a significant feature called a myosin binding site. The myosin head attaches to the myosin binding site of actin during muscle contraction.

Tropomyosin and troponin are regulatory proteins associated with thin filaments. Together they form the tropomyosin-troponin complex. Tropomyosin (trō-pō-mī′-sin) is a short, thin, twisted filament that is a “stringlike” protein. Consecutive tropomyosin molecules cover small regions of the actin strands, including the myosin binding sites in a noncontracting muscle. Troponin (trō′pō-nin) is a globular, or “ball-like,” protein attached to tropomyosin. Troponin contains the binding site for Ca$^{2+}$.

**Organization of a Sarcomere**

Myofilaments within myofibrils are arranged in repeating, microscopic, cylindrical units (2 micrometers in length) called sarcomeres (sar′kō-mer′; meros = part). Figure 10.5a shows several repeating sarcomeres in a section of a myofibril within a skeletal muscle fiber. The number of sarcomeres varies with the length of the myofibril within the skeletal muscle fiber. Each sarcomere is composed of overlapping thick filaments and thin filaments.

A two-dimensional, longitudinal view of a cylindrical sarcomere is shown in figure 10.5b. Here we see that each sarcomere is delineated at both ends by Z discs. Z discs (also called Z lines) are composed of specialized proteins that are positioned perpendicular to the myofilaments and serve as anchors for the thin filaments. Although the Z disc appears as a flat disc when the myofibril is viewed from its end, only the edge of the disc is visible in a side view, and it sometimes looks like a zigzagged line.

The thick filaments and thin filaments overlap within a sarcomere, forming the following regions:

- **I bands** extend from both directions of a Z disc and are bisected by the Z disc. These end regions contain only thin filaments; this region appears light when viewed with a microscope. At

![Figure 10.5 Structure of a Sarcomere.](image)
maximal muscle shortening, the thin filaments are pulled parallel along the thick filaments, causing the I bands to disappear.

- The A band is the central region of a sarcomere that contains the entire thick filament. Thin filaments partially overlap the thick filament on each end of an A band. The A band appears dark when viewed with a microscope. The A band does not change in length during muscle contraction.

- The H zone (also called the H band) is the most central portion of the A band in a resting sarcomere. This region does not have thin filament overlap; only thick filaments are present. During maximal muscle shortening, this zone disappears when the thin filaments are pulled past thick filaments.

- The M line is a thin transverse protein meshwork structure in the center of the H zone. It serves as an attachment site for the thick filaments and keeps the thick filaments aligned during contraction and relaxation events.

The repeating light and dark bands of the overlapping myofilaments form unique striped patterns within a skeletal muscle fiber called striations. Striations are visible when viewing a longitudinal section of skeletal muscle tissue in an image produced by either a light microscope (see figure 10.6b) or an electron microscope (see figure 10.15). This striated appearance is due to both the size and density differences between thin filaments and thick filaments.

Figure 10.5c shows cross sections through various regions in a sarcomere. It presents the relative sizes, arrangements, and organization of thick and thin filaments at different locations within the sarcomere. Notice that in a cross section of an A band the arrangement of thick filaments relative to thin filaments is the following: Each thin filament has three thick filaments around it that form a triangle at its periphery, and each thick filament is sandwiched by six thin filaments.

**WHAT DO YOU THINK?**

If a muscle is contracted and shortening, what happens to the following: (a) width of the A band, (b) width of the H zone, (c) relationship of the Z discs, and (d) width of the I band?

**Other Structural and Functional Proteins** Other proteins have structural and functional roles within muscle fibers. These include connectin and dystrophin (only connectin is shown in figure 10.5).

Connectin (kon-nek’tin), also called titin, is a “cablelike” protein that extends from the Z discs to the M line through the core of each thick filament (figure 10.5b). It stabilizes the position of the thick filament and maintains thick filament alignment within a sarcomere. Additionally, portions of the connectin molecules are coiled and “springlike” so that during sarcomere shortening they are compressed to produce passive tension. This passive tension is then released to return the sarcomere to its normal resting length. Thus, connectin contributes to skeletal muscle fiber elasticity (see section 10.1b).

Dystrophin (dis-tró’fin) is part of a protein complex that anchors myofibrils that are adjacent to the sarcolemma to proteins within the sarcolemma. These proteins of the sarcolemma also extend to the connective tissue of the endomysium that encloses the muscle fiber. Thus, dystrophin links internal myofilament proteins of a muscle fiber to external proteins. The genetic disorder of muscular dystrophy is caused by abnormal structure, or amounts, of dystrophin protein (see Clinical View 10.1: “Muscular Dystrophy”).

**Mitochondria and Other Structures Associated with Energy Production**

Skeletal muscle fibers have a great demand for energy and contain several components that facilitate the production of ATP (see section 10.4a). Skeletal muscle fibers have abundant mitochondria for aerobic cellular respiration (see section 3.4); a typical skeletal muscle fiber contains approximately 300 mitochondria. The fibers also contain glycogen stores (granules called glycosomes) for use as an immediate fuel molecule. Myoglobin (mi-gó’bin) is a molecule unique to muscle tissue. Myoglobin is a reddish, globular protein that is somewhat similar to hemoglobin. It binds oxygen when the muscle is at rest and releases it for use during muscular contraction. This additional source of oxygen provides the means to enhance aerobic cellular respiration and the production of ATP. Skeletal muscle fibers also contain another type of molecule called creatine phosphate (see figure 3.4c). Creatine phosphate provides muscle fibers with a very rapid means of supplying ATP.
INTEGRATE

CLINICAL DYSTROPHY 10.1
Muscular Dystrophy

Muscular dystrophy (disˈtrō-fē) is a collective term for several hereditary diseases in which the skeletal muscles degenerate, lose strength, and are gradually replaced by adipose and fibrous connective tissue. In a viscous cycle, the new connective tissues impede blood circulation, which further accelerates muscle degeneration.

Duchenne muscular dystrophy (DMD) is the most common form of the illness. It is almost exclusively a disease of males and occurs in about 1 in 3500 live births. DMD results from the expression of a sex-linked recessive allele (see section 29.9c), which is the gene that directs the synthesis of dystrophin. In DMD, dystrophin either has an abnormal structure or is produced in insufficient amounts. The defective, reduced, or absent dystrophin results in an unstable sarcolemma, which is susceptible to damage by forces generated during muscle contraction. Excess calcium ions then enter the muscle fibers, damaging the contractile proteins with an accompanying loss of muscle fibers.

For these individuals, muscular difficulties become apparent in early childhood. Walking is a problem; the child falls frequently and has difficulty standing up again. The hips are affected first, followed by the lower limbs, and eventually the abdominal and vertebral muscles. Muscular atrophy causes shortening of the muscles, which results in postural abnormalities such as scoliosis (lateral curvature of the spine; see Clinical View 8.3: “Spinal Curvature Abnormalities”). DMD is an incurable disease, with patients confined to a wheelchair by adolescence. An individual with DMD rarely lives beyond the age of 30, and death typically results from respiratory or heart complications.

The details of how ATP is provided to meet the high energy needs of skeletal muscle fibers are described in section 10.4a.

WHAT DID YOU LEARN?

4. Draw and label a diagram of a sarcomere.
5. Place the following gross anatomic and microscopic anatomic structures in order from largest to smallest: fascicle, myofibril, myofilament, muscle, muscle fiber, and sarcomere. Describe their anatomic relationship.

10.2c Innervation of Skeletal Muscle Fibers

LEARNING OBJECTIVES

11. Define a motor unit, and describe its distribution in a muscle and why it varies in size.
12. Describe the three components of a neuromuscular junction.

The anatomic relationship of skeletal muscle fibers and somatic motor neurons that control them is described in this section.

Motor Unit

Somatic motor neurons are nerve cells that transmit electrical signals (nerve signals) from the brain or spinal cord to control skeletal muscle activity (see section 12.1b). The axon of each motor neuron divides into many individual branches to innervate numerous skeletal muscle fibers. A single motor neuron and the skeletal muscle fibers it controls is called a motor unit (figure 10.6).

The number of skeletal muscle fibers a single motor neuron innervates—and thus the size of the motor unit—varies and can range from small motor units that have less than five muscle fibers to large motor units that have several thousand muscle fibers. The size of the motor unit determines the degree of control. There is an inverse relationship between the size of a motor unit and the degree of control. For example, motor neurons innervating extrinsic eye muscles (see section 11.3b) are small because greater control is essential in the muscles that move the eye. In contrast, a single motor neuron controls several thousand individual skeletal muscle fibers in the power-generating muscles in our lower limbs, where less precise control is required.

The skeletal muscle fibers of a motor unit are not clustered within one area of a muscle, but rather are dispersed throughout most of a muscle. Normally, the stimulation of a motor unit does not produce a strong contraction in a localized area within the muscle, but a weak contraction over a wide area.

Neuromuscular Junctions

Each skeletal muscle fiber is typically described as having one neuromuscular junction. A neuromuscular (nūr-ō-mūs′kə-lər) junction is the specific location, usually in the mid-region of the skeletal muscle fiber where it is innervated by a motor neuron (figure 10.7a).
The neuromuscular junction has the following parts: synaptic knob, motor end plate, and synaptic cleft.

**Synaptic Knob** The synaptic (si-nap’tik) knob of a motor neuron is an expanded tip of an axon. Where the axon nears the sarcolemma of a muscle fiber, the synaptic knob enlarges and flattens to cover a relatively large surface area of the sarcolemma. The synaptic knob cytosol houses numerous synaptic vesicles (small membrane sacs) filled with molecules of the neurotransmitter acetylcholine (a-sē’til-kō’lēn) (ACh).

Several points can be made about synaptic knobs (figure 10.7b). First, Ca\(^{2+}\) pumps are embedded within the plasma membrane of the synaptic knob. Prior to the arrival of the electrical signal (nerve signal) at the synaptic knob, Ca\(^{2+}\) pumps within its plasma membrane have established a Ca\(^{2+}\) concentration gradient, with more Ca\(^{2+}\) outside the synaptic knob than inside it. Second, voltage-gated Ca\(^{2+}\) channels are also embedded in the membrane of the synaptic knob. Opening of these channels allows Ca\(^{2+}\) to flow down its concentration gradient from the interstitial fluid into the synaptic knob, which will trigger exocytosis of acetylcholine from the vesicles. Third, vesicles are normally repelled from the synaptic knob plasma membrane.

**Motor End Plate** The motor end plate is a specialized region of the sarcolemma of a skeletal muscle fiber. (It is so named because “motor end” reflects that it is located at the end of a motor neuron and “plate” describes its large, saucerlike appearance.) It has numerous folds and indentations (junction folds) to increase the membrane surface area covered by the synaptic knob. The motor end plate has vast numbers of ACh receptors. These plasma membrane protein channels are chemically gated ion channels (see section 4.3a). Binding of ACh opens these channels, allowing Na\(^{+}\) entry into the muscle fiber and K\(^{+}\) to exit. ACh receptors are like doors; ACh is the only “key” to open these receptor doors.

**Synaptic Cleft** The synaptic cleft is an extremely narrow (30 nanometers), fluid-filled space separating the synaptic knob and the motor end plate. The enzyme acetylcholinesterase (a-sē’til-kō’lēn-es’ter-ās) (AChE) resides within the synaptic cleft (not shown in figure 10.7) and quickly breaks down ACh molecules following their release into the synaptic cleft. (See the electron micrograph of neuromuscular junction in figure 12.22. A detailed view of the breakdown of acetylcholine by acetylcholinesterase is shown in figure 12.25.)

**WHAT DID YOU LEARN?**

6. What is a motor unit, and why does it vary in size?

7. Diagram and label the anatomic structures of a neuromuscular junction.

### 10.2d Skeletal Muscle Fibers at Rest

**LEARNING OBJECTIVE**

13. Describe a skeletal muscle fiber at rest.

Skeletal muscle fibers exhibit several significant features when the muscle is at rest. See figure 10.8 as you read through this section.

One essential feature of skeletal muscle fibers is the electrical charge difference across the sarcolemma; the cytosol right inside the...
plasma membrane is relatively negative in comparison to the interstitial fluid outside of the cell. This electrical charge difference when the cell is at rest is called the resting membrane potential (RMP) (see section 4.4). Skeletal muscle fibers have an RMP of about −90 millivolts (mV). An RMP is established and maintained by both leak channels and Na+/K+ pumps (not shown in figure 10.8). The primary function of the Na+/K+ pumps is to maintain the concentration gradients for Na+ (with more Na+ outside the cell) and K+ (with more K+ inside the cell).

The acetylcholine receptors (chemically gated ion channels) within the motor end plate and the voltage-gated Na+ channels and voltage-gated K+ channels in the sarcolemma and T-tubules are closed, Ca2+ ions are stored within the terminal cisternae of the sarcoplasmic reticulum, and the contractile proteins (myofilaments) within the sarcomeres are in their relaxed position.

**WHAT DID YOU LEARN?**

Describe the distribution of Na+ and K+ at the sarcolemma.

**CONCEPT CONNECTION**

Either an impaired ability or an inability of the nervous system to stimulate skeletal muscle fibers can result in decreased or absent muscle fiber contraction. Causes include damage to any of the following: (1) components of the brain, which initiate nerve impulses for muscle contraction (see Clinical View 13.9: “Cerebrovascular Accident”); (2) the spinal cord, which relays nerve impulses from the brain to many of the skeletal muscles (see Clinical View 14.3: “Treating Spinal Cord Injuries”); and (3) somatic motor neurons, which stimulate the skeletal muscle fibers (see Clinical View 12.5: “Neurotoxicity” and Clinical View 14.2: “Polioymyelitis”). Toxins can also interfere with skeletal muscle contraction (see Clinical View 10.3: “Muscular Paralysis and Neurotoxicity”).

**10.3 Physiology of Skeletal Muscle Contraction**

A motor neuron stimulates skeletal muscle fibers. This stimulation ultimately results in the interaction between myofilaments within the skeletal muscle fibers to produce tension. The resulting tension is exerted on the portions of the skeleton (or other body structures) where the muscle is attached to cause movement in the body.

The anatomic structures and associated physiologic processes of skeletal muscle contraction include the events that occur at the (1) neuromuscular junction; (2) sarcolemma, T-tubules, and sarcoplasmic reticulum; and (3) sarcomeres. An overview of these processes is included in figure 10.9.

**CLINICAL VIEW 10.2**

**Myasthenia Gravis (MG)**

Myasthenia (mi`əs-θê`n-e-a; asthenia = weakness) gravis (MG) is an autoimmune disease that occurs in about 1 in 10,000 people, primarily women between 20 and 40 years of age. A person’s own antibodies attack the neuromuscular junctions, binding ACh receptors into clusters. The abnormally clustered ACh receptors are removed from the muscle fiber sarcolemma by endocytosis, thus significantly diminishing the number of receptors within the sarcolemma. The resulting decreased muscle stimulation causes rapid fatigue and muscle weakness. Eye and facial muscles are often attacked first, producing double vision and drooping eyelids. These symptoms are usually followed by swallowing problems, limb weakness, and overall low physical stamina. Some patients with MG have a normal life span, whereas others die within a short time from paralysis of the respiratory muscles.
Chapter Ten  Muscle Tissue

10.3a Neuromuscular Junction: Excitation of a Skeletal Muscle Fiber

LEARNING OBJECTIVE

14. Explain the events that lead to release of the neurotransmitter ACh from a motor neuron.

The first physiologic event of skeletal muscle contraction is muscle fiber excitation by a somatic motor neuron—an event that occurs at the neuromuscular junction and results in release of ACh and its subsequent binding to ACh receptors. These events are summarized in Figure 10.10.

Calcium Entry at Synaptic Knob

A nerve signal (or nerve impulse), which is the electrical signal that is propagated down an axon, is sent along a motor neuron of the somatic nervous system. (Nerve signals are discussed in detail in Section 12.8c.) The nerve signal triggers the opening of voltage-gated Ca\(^{2+}\) channels within the synaptic knob plasma membrane, and calcium moves down its concentration gradient from the interstitial fluid through the open channels into the synaptic knob. Calcium binds with membrane proteins (synaptotagmin [not shown]) exposed on the external surface of synaptic vesicles (step 1a).

Release of ACh from Synaptic Knob

The binding of Ca\(^{2+}\) to synaptic vesicles triggers the merging of synaptic vesicles with the synaptic knob plasma membrane, resulting in exocytosis of ACh into the synaptic cleft. Acetylcholine is released from approximately 300 vesicles per nerve signal with each vesicle releasing thousands of molecules of ACh (step 1b).

Binding of ACh at Motor End Plate

ACh diffuses across the fluid-filled synaptic cleft to bind with ACh receptors within the motor end plate. This causes excitation of a skeletal muscle fiber (step 1c).

Note that nerve signals are repeatedly propagated along the motor axon (at about 10 to 40 times per second). Thus, these events (steps 1a–1c) will continue until stimulation of the skeletal muscle fiber by the neuron ceases (stops) and acetylcholinesterase catalyzes breakdown of ACh that is within the synaptic cleft (see Clinical View 12.6: “Altered Acetylcholine Function and Changes in Breathing”).

WHAT DID YOU LEARN?

9. What triggers the binding of synaptic vesicles to the synaptic knob membrane to cause exocytosis of ACh?

10.3b Sarcolemma, T-tubules, and Sarcoplasmic Reticulum: Excitation-Contraction Coupling

LEARNING OBJECTIVE

15. Describe the steps in excitation-contraction coupling.

The second physiologic event of muscle contraction is excitation-contraction coupling—an event that involves the sarcolemma, T-tubules,
and sarcoplasmic reticulum. This event “couples,” or links, the events of skeletal muscle stimulation at the neuromuscular junction (first step) to the events of contraction caused by sliding myofilaments within the sarcomeres of skeletal muscle fiber (third step). Three events occur during excitation-contraction coupling: development of an end-plate potential at the motor end plate, initiation and propagation of an action potential along the sarcolemma and T-tubules, and release of Ca\(^{2+}\) from the sarcoplasmic reticulum. These events are summarized in figure 10.11.

**Development of an End-Plate Potential at the Motor End Plate**

The ACh receptors, which are chemically gated ion channels, are stimulated to open temporarily when ACh binds to them (step 2a). The opening of these channels allows relatively small amounts of both Na\(^{+}\) to rapidly diffuse into the skeletal muscle fiber and K\(^{+}\) to slowly diffuse out of the skeletal muscle fiber. More Na\(^{+}\) diffuses in than K\(^{+}\) diffuses out, and there is a net gain of positive charge on the inside of the skeletal muscle fiber. The flow of both Na\(^{+}\) and K\(^{+}\) ions quickly slows and then ceases as the ions meet with resistance. Thus, these changes in membrane potential in the motor end plate are both transient (short-lived) and local. However, if there is sufficient gain of positive charge to change the RMP of about –90 mV to –65 mV, an end-plate potential is produced. An end-plate potential (EPP) is the minimum voltage change (or threshold) in the motor end plate that can trigger opening of voltage-gated channels in the sarcolemma to initiate an action potential.

**Initiation and Propagation of Action Potential Along the Sarcolemma and T-tubules**

The EPP triggers an action potential that is propagated along the sarcolemma and T-tubules of the skeletal muscle fiber (step 2b). An action potential involves two events: depolarization, which causes the inside of the sarcolemma of the skeletal muscle fiber to become positive due to the influx of Na\(^{+}\), and repolarization, which is the returning of the inside of the sarcolemma to its relatively negative resting membrane potential due to the outward flow of K\(^{+}\).

The electrical change of the EPP in the motor end plate stimulates the opening of voltage-gated Na\(^{+}\) channels in the adjacent area of the sarcolemma. The opening of voltage-gated Na\(^{+}\) channels allows Na\(^{+}\) to move rapidly across the sarcolemma down its concentration gradient into the skeletal muscle fiber. Sufficient Na\(^{+}\) enters to cause a reversal of the membrane potential of the sarcolemma. The inside, which was relatively negative, becomes relatively positive with a change in the membrane potential from the threshold value of –65 mV to +30 mV. This reversal in polarity at the sarcolemma is referred to as depolarization.

The propagation of depolarization along the length of the sarcolemma and T-tubules involves the sequential opening of voltage-gated Na\(^{+}\) channels. The inflow of Na\(^{+}\) at the initial portion of the sarcolemma causes adjacent regions of the sarcolemma to experience electrical changes that initiate voltage-gated Na\(^{+}\) channels in these areas to open. Sodium flows in to cause depolarization in this region of the sarcolemma. Adjacent depolarization is repeated rapidly down the sarcolemma and T-tubules. The propagation of an action potential along the sarcolemma and T-tubule is similar to the falling of a series of stacked dominos—once started, it does not stop until it reaches the end.

Voltage-gated K\(^{+}\) channels located along the sarcolemma and T-tubules open immediately following the opening of the voltage-gated Na\(^{+}\) channels. The opening of voltage-gated K\(^{+}\) channels allows K\(^{+}\) to move across the sarcolemma down its concentration gradient and out of the skeletal muscle fiber. Sufficient K\(^{+}\) exits so that the membrane potential at the sarcolemma and T-tubules reverses and the negative resting membrane potential (–90 mV) is reestablished. This process, which changes the membrane potential from +30 mV to reestablish the RMP of –90 mV, is referred to as repolarization. The
opening of voltage-gated $K^+$ channels also occurs sequentially, and repolarization is propagated along the sarcolemma and T-tubules. Repolarization allows the skeletal muscle fiber to propagate a new action potential when stimulated again by a motor neuron. Note that an action potential is a self-sustaining electrical change in the membrane potential that is propagated along the sarcolemma and is caused by the sequential opening of voltage-gated channels. Action potential propagation at the sarcolemma is similar to action potential propagation that occurs in neurons (see section 12.8c).

Figure 10.12 is a graph of the electrical changes at the sarcolemma. These electrical changes include reaching the threshold, depolarization, and repolarization. The period of time that includes depolarization and repolarization is called the refractory period. The refractory period is significant because during this brief period of time the muscle cannot be restimulated. A new action potential can occur only when the resting membrane potential at the sarcolemma has been reestablished.

Release of Calcium from the Sarcoplasmic Reticulum
When the action potential reaches the sarcoplasmic reticulum, it (1) stimulates a conformational change to voltage-sensitive $Ca^{2+}$ channels (dihydropyridine receptors) within the T-tubule membrane, which (2) causes a conformational change in $Ca^{2+}$ release channels (ryanodine receptors) located in the terminal cisternae of the sarcoplasmic reticulum, causing them to open (figure 10.11, step 2c). This allows $Ca^{2+}$ to diffuse out of the cisternae of the sarcoplasmic reticulum into the cytosol. Calcium now “mingles” with the thick filaments and thin filaments within myofibrils.

WHAT DID YOU LEARN?
10. What two events are linked in the physiologic process called excitation-contraction coupling?
11. Provide a description of the events of excitation-contraction coupling.

10.3c Sarcomere: Crossbridge Cycling

LEARNING OBJECTIVE
16. Summarize the changes that occur within a sarcomere during contraction.

The third physiologic event in skeletal muscle contraction involves binding of $Ca^{2+}$ and crossbridge cycling. These events are summarized in figure 10.13.

Calcium Binding
Calcium released from the sarcoplasmic reticulum binds to a subunit of globular troponin, a component of thin filaments. This induces a conformational change in troponin. Recall that troponin is attached to tropomyosin, forming the troponin-tropomyosin complex. When troponin changes shape, the entire troponin-tropomyosin complex is moved and the myosin binding sites of actin are exposed. Crossbridge cycling is initiated (step 3a).
An end-plate potential (EPP) is produced when sufficient Na\(^+\) diffuses out of the muscle fiber to change the RMP from –90 mV to –65 mV.

The threshold (end-plate potential) is reached when ACh receptors, which are chemically gated ion channels, open and sufficient Na\(^+\) enters the motor end plate to change the RMP from –90 mV to –65 mV (the threshold value).

Depolarization occurs as voltage-gated Na\(^+\) channels on the sarcolemma open and Na\(^+\) enters rapidly, reversing the polarity from negative to positive (–65 mV to +30 mV).

Repolarization occurs due to closure of voltage-gated Na\(^+\) channels and opening of voltage-gated K\(^+\) channels on the sarcolemma. K\(^+\) moves out of the cell, and the polarity is reversed from positive to negative (+30 mV to –90 mV).

**Figure 10.12** Events of an Action Potential at the Sarcolemma. A tracing of the membrane voltage (mV) changes associated with an action potential initiated at the neuromuscular junction. Changes occur in just a few milliseconds and result from the opening and closing of voltage-gated Na\(^+\) channels and voltage-gated K\(^+\) channels in the sarcolemma.
Crossbridge Cycling

Crossbridge cycling refers to a four-step process that is repeated (step 3b–e): (1) crossbridge formation (attaching of myosin head to actin), (2) power stroke (pulling thin filament by movement of myosin head), (3) release of myosin head from actin, and (4) resetting of myosin head:

**Crossbridge formation** Myosin heads, which are in the “cocked,” or ready, position attach to exposed myosin binding sites on actin. Binding of each myosin head results in formation of a crossbridge between the thick and thin filament (step 3b).
**Power stroke**  After forming a crossbridge, the myosin head swivels (or ratchets) in what is called a power stroke. The swiveling of a myosin head pulls the thin filament a small distance past the thick filament toward the center of the sarcomere. ADP and P_i are released during this process, and the ATP binding site becomes available again (step 3c).

**Release of myosin head**  ATP then binds to the ATP binding site of a myosin head, which causes the release of the myosin head from the binding site on actin (step 3d).

**Resetting of myosin head**  Myosin ATPase splits ATP into ADP and P_i, providing the energy to reset the myosin head in the cocked position (step 3e).

If Ca^{2+} is still present, and the myosin binding sites are still exposed, then these four steps involving the myosin heads continue: attach, pull, release, and reset. It is the repetitive action of these steps that results in sarcomere shortening, and a sarcomere moves from its relaxed state into a contracted state. Note that calcium levels remain elevated because the skeletal muscle fiber is repeatedly stimulated by the motor neuron at a very rapid rate. Figure 10.14 is an electron micrograph of crossbridges between myosin heads and myosin binding sites in actin.

A sarcomere in both a relaxed and a contracted skeletal muscle fiber that has shortened is shown in figure 10.15. The following changes to the sarcomere occur in the contracted muscle: The H zone disappears, the I band narrows in width and may disappear, and the Z discs in each sarcomere move closer together. However, the thin and thick filaments do not shorten. A description of the repetitive movement of thin filaments sliding past thick filaments is called the sliding filament theory. The three major events of skeletal muscle contraction are integrated in figure 10.16.
Figure 10.16 Skeletal Muscle Contraction. A summary of skeletal muscle contraction, which includes the events at (1) the neuromuscular junction, (2) the sarcolemma, T-tubules, and sarcoplasmic reticulum, and (3) sarcomeres.
2 Sarcolemma, T-tubules, and Sarcoplasmic Reticulum

Excitation-contraction coupling

Neuromuscular Junction

Excitation of a skeletal muscle fiber

1 Sarcolemma, T-tubules, and Sarcoplasmic Reticulum

2 Action potential propagation

3 Sarcomere

Crossbridge cycling (cycle repeats and sarcomere shortens)

2a Ca\(^{2+}\) binds to troponin exposing myosin binding sites on actin.

3a Attach: Crossbridge formation between myosin and actin.

3b Pull: Power stroke motion of myosin head pulls thin filament past it.

3c Re-set: ATP split and myosin head is reset.

ACh is released by exocytosis into synaptic cleft.

A nerve signal triggers voltage-gated Ca\(^{2+}\) channels to open—Ca\(^{2+}\) enters synaptic knob and binds to synaptic vesicles.

ACh binding causes Na\(^{+}\) to rapidly enter the skeletal muscle fiber and K\(^{+}\) to slowly exit the skeletal muscle fiber, which may result in an end-plate potential (EPP).

The EPP initiates an action potential along sarcolemma and T-tubules.

Action potential triggers Ca\(^{2+}\) release from sarcoplasmic reticulum (SR) terminal cisternae.

Motor end plate

Synaptic vesicle

Sarcolemma

T-tubule

Sarcoplasmic reticulum

Thick filament

Crossbridge formation

Myosin binding site

ADP

ATP

Power stroke

Motor end plate

Synaptic vesicle

Sarcolemma

T-tubule

Sarcoplasmic reticulum

Thick filament

Crossbridge formation

Myosin binding site

ADP

ATP

Power stroke
Muscular Paralysis and Neurotoxins

**Muscular paralysis (inability of skeletal muscles to contract)** may occur if either nervous system function at the neuromuscular junction or excitation-contraction coupling is impaired. This damage may be the result of **neurotoxins**, which are toxins that damage nervous system components. Two paralysis conditions caused by toxins are tetanus and botulism.

**Tetanus** is a form of spastic paralysis caused by a toxin produced by the bacterium *Clostridium tetani*. The toxin blocks the release of glycine (an inhibitory neurotransmitter in the spinal cord), resulting in overstimulation by motor neurons of the muscles and excessive muscle contractions. Penetrating wounds contaminated with soil and vegetable matter are especially prone to developing *C. tetani* infection. This condition is potentially life-threatening, and so we routinely are vaccinated against it.

**Botulism** (botˈə-lizm), a potentially fatal muscular paralysis, is caused by a toxin produced by the bacterium, *Clostridium botulinum*. The toxin prevents the release of acetylcholine (ACh) at synaptic knobs and leads to muscular paralysis. Like *C. tetani*, *C. botulinum* is common in the environment and produces its toxin under anaerobic conditions. Most cases of botulism poisoning result from ingesting the toxin in canned foods that were not processed at temperatures high enough to kill the botulism spores. Similarly, ingestion of unpasteurized honey by infants in the first year of life can introduce *C. botulinum* spores into their immature gastrointestinal tracts.

The Food and Drug Administration (FDA) approved the use of botulinum toxin type A (Botox) for temporary diminishing of wrinkles (see Clinical View 6.7: “Botox and Wrinkles”). Botox is also used clinically to help reduce overcontraction of muscle (or spasticity) associated with certain disorders or conditions (e.g., cerebral palsy, multiple sclerosis, torticollis, changes following a stroke or spinal cord injury). Botox injections have become one of the most important treatments for spasticity and are most effective 1 to 2 weeks after the injections, with spasticity reduced for up to 3 to 6 months. Treatments may be repeated as often as every 3 months.

**CLINICAL VIEW 10.4**

**Rigor Mortis**

Within a few hours after the heart stops beating, ATP levels in skeletal muscle fibers have been completely exhausted. The sarcoplasmic reticulum loses its ability to return Ca²⁺ from the sarcoplasm and move it back into the sarcoplasmic reticulum by the Ca²⁺ pumps, which require ATP to function. Remember that ATP is also needed to detach the myosin head of the thick filament from the myosin binding site of actin on the thin filaments. Because ATP is no longer available, the crossbridges between thick and thin filaments cannot detach. As a result, the Ca²⁺ already present in the sarcoplasm, as well as the Ca²⁺ that continues to leak out of the sarcoplasmic reticulum, triggers a sustained contraction in the skeletal muscle fibers. All skeletal muscles lock into a contracted position and the body of the deceased individual becomes rigid. This physiologic state, termed *rigor mortis* (rigˈər mɔrˈtis), continues for about 15 to 24 hours. Rigor mortis gradually disappears because lysosomal enzymes are released within the muscle fibers, causing autolysis (self-destruction and breakdown) of the myofibrils.

Forensic pathologists often use the development and resolution of rigor mortis to establish an approximate time of death. Because a number of factors affect the rate of development and resolution of rigor mortis, environmental conditions need to be taken into consideration. For example, a warmer body will develop and resolve rigor mortis much more quickly than a body of normal temperature. The following chart provides rough guidelines for estimating the death interval, assuming that body temperature and ambient (surrounding environment) temperature are within normal range.

<table>
<thead>
<tr>
<th>Death Interval</th>
<th>Body Temperature</th>
<th>Stiffness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dead less than 3 hours</td>
<td>Warm</td>
<td>No stiffness</td>
</tr>
<tr>
<td>Dead 3–8 hours</td>
<td>Warm, but cooling</td>
<td>Developing stiffness</td>
</tr>
<tr>
<td>Dead 8–24 hours</td>
<td>Ambient temperature</td>
<td>Stiff, but resolving</td>
</tr>
<tr>
<td>Dead 24–36 hours</td>
<td>Ambient temperature</td>
<td>No stiffness</td>
</tr>
</tbody>
</table>
release of passive tension that developed in connectin proteins that were compressed during shortening.

It is interesting to note that a significant amount of ATP is used by the Ca\(^{2+}\) pumps of the sarcoplasmic reticulum. Calcium levels within the cytosol of muscle fibers must be kept low to prevent Ca\(^{2+}\) from binding with phosphate ions (which are released from ATP) to form hydroxyapatite (see section 7.2e), which would calcify and harden muscles in a process similar to that in bone tissue. Thus, ATP is required for both contraction (by myosin ATPase) and relaxation. In fact, if sufficient ATP is not available (as occurs following death), muscle relaxation cannot occur and the muscle remains in a contracted state (see Clinical View 10.4: “Rigor Mortis”).

**WHAT DID YOU LEARN?**

15. How do acetylcholinesterase and Ca\(^{2+}\) pumps function in the relaxation of a muscle?

### 10.4 Skeletal Muscle Metabolism

We discussed how cells form ATP through the process of cellular respiration in section 3.4. Here those concepts are integrated to describe specifically how a muscle fiber meets its very-high-energy needs—and how its various means of supplying ATP are used to classify skeletal muscle fibers into three primary types.

#### 10.4a Supplying Energy for Skeletal Muscle Metabolism

**LEARNING OBJECTIVES**

19. Describe how ATP is made available within skeletal muscle through myosin kinase, creatine kinase, glycolysis, and aerobic cellular respiration.

20. Explain how the means of supplying ATP is related to intensity and duration of exercise.

Most of the ATP required by skeletal muscle fibers is used to reset the myosin heads of the thick filaments during muscle contraction (see section 10.3c), which demands very large amounts of ATP (approximately 2500 ATP molecules per thick filament per second). ATP is also required by the calcium pump within the sarcoplasmic reticulum membrane to return Ca\(^{2+}\) to the terminal cisternae for storage, as described in section 10.3d.

A very limited amount of ATP is already present within skeletal muscle fibers, and additional small amounts can be rapidly produced as phosphate (P\(_i\)) is transferred from one ADP to another ADP, yielding ATP and adenosine monophosphate (AMP), an enzymatic reaction catalyzed by myokinase (figure 10.17a). This usually provides only enough energy for about 5 to 6 seconds of maximal exertion. Thus, meeting these high energy demands requires forming ATP from other sources. These include ATP formed from creatine phosphate, by glycolysis, and through aerobic cellular respiration.

**Creatine Phosphate**

Creatine phosphate (see figure 3.4c) is a molecule with a high-energy chemical bond between creatine and P\(_i\), and is present in tissues with both large and fluctuating energy needs (e.g., muscle, brain). When skeletal muscle is actively contracting, the P\(_i\) in creatine phosphate is readily transferred to ADP to form additional ATP (and creatine), an enzymatic reaction catalyzed by creatine kinase (figure 10.17a). This provides an additional 10 to 15 seconds of energy during maximum exertion.

Later during times of rest, the limited stores of ATP and CP within skeletal muscle are replenished. ATP is formed through cellular respiration, and some of those ATP molecules are used to regenerate creatine phosphate. The process that happens at rest is the reverse of the process that happens during exercise. The P\(_i\) in ATP is transferred to creatine to form additional creatine phosphate and ADP, an enzymatic reaction catalyzed by creatine kinase. These enzymatic reactions involving P\(_i\) transfer to ADP to form ATP are not dependent upon the presence of oxygen.

**CLINICAL VIEW 10.5**

**Creatine Kinase Blood Levels as a Diagnostic Tool**

Creatine kinase, also called creatine phosphokinase, is the enzyme that helps transfer a phosphate between creatine and ATP. Different forms of creatine kinase are present within cardiac muscle and skeletal muscle. The heart muscle form of creatine kinase is found in elevated blood levels in patients suffering from a myocardial infarction (heart attack; see Clinical View 19.5: “Coronary Heart Disease, Angina Pectoris, and Myocardial Infarction”). This provides a specific diagnostic tool for identifying damage to the heart. In contrast, elevated levels of the skeletal muscle form of creatine kinase are used to diagnose degenerative skeletal muscle disease, such as muscular dystrophy (see Clinical View 10.1: “Muscular Dystrophy”). However, note that elevated levels of the skeletal muscle form of creatine kinase may also occur after intense exercise and therefore is not always a sign of disease.

**WHAT DO YOU THINK?**

4. When skeletal muscle tissue is damaged, creatine kinase is released. What general conclusions can be drawn with increasing blood levels of creatine kinase?

**Glycolysis**

Recall from section 3.4b that glycolysis is a metabolic pathway, which involves the breakdown of glucose into two pyruvate molecules, producing a net of 2 ATP molecules (figure 10.17b). It occurs within the cytosol, and although it can function in the presence of oxygen, oxygen is not required. Glucose is made available either directly from glycogen stores within the muscle fiber (through glycogenolysis; see figure 2.19) or delivered by the blood.

One of the main advantages of producing ATP through glycolysis is that it does not require oxygen (i.e., it is nonoxidative). The other is its rapid rate of ATP production (i.e., the amount of ATP produced per time—at almost twice the rate of aerobic cellular respiration). Although lower total amounts of ATP are produced (compared to aerobic cellular respiration), ATP is produced more quickly, a necessary requirement for short bursts of maximum exercise (e.g., running a 100-meter dash).

What happens to the pyruvate molecules that are produced during glycolysis? Recall from section 3.4g that the fate of pyruvate molecules is dependent upon oxygen availability. Pyruvate molecules either (1) enter a mitochondrion to be broken down through aerobic cellular respiration (if sufficient oxygen is available) or (2) are converted to lactate molecules (under conditions of low oxygen availability).
Chapter Ten
Muscle Tissue

Aerobic Cellular Respiration

Aerobic cellular respiration occurs within mitochondria and requires oxygen, which is made available from the blood or released from myoglobin (figure 10.17c). It involves three stages, including the intermediate step, the citric acid cycle, and the electron transport system (see sections 3.4c–f). One of the primary advantages of producing ATP through aerobic cellular respiration is the variety of nutrients that can be oxidized, which include pyruvate (made available through glycolysis), fatty acids, and amino acids (which are deaminated; see section 3.4h). The other advantage is that, although the rate of ATP formation is slower (than in glycolysis), greater amounts of ATP are produced. The specific amounts are dependent upon the nutrient that is oxidized (e.g., pyruvate generates 17 ATP molecules; the fatty acid palmitate generates 129 ATP molecules). These higher amounts are a necessary requirement for longer, more moderate levels of activity (e.g., jogging several miles).

Lactate Formation and Its Fate

Lactate formation from pyruvate occurs under conditions of low oxygen availability. This occurs, for example, during intense exercise when skeletal muscle’s oxygen demands for aerobic cellular respiration cannot be met. The pyruvate molecules are instead converted to lactate molecules, an enzymatic reaction catalyzed by lactate dehydrogenase (see section 3.4g).

What happens to lactate following its formation? Lactate can either enter a mitochondrion within a skeletal muscle fiber, where it is converted back to pyruvate and oxidized to carbon dioxide through aerobic cellular respiration (see section 19.3f) or (2) taken up by the liver to be converted to glucose through gluconeogenesis (see section 27.6c). Glucose molecules are then released by the liver back into the blood, where they may be taken up into a skeletal muscle fiber. This cycling of lactate to the liver, where it is converted to glucose, and the subsequent transport of glucose from the liver to the muscle is called the lactic acid cycle (or Cori cycle). Observe that lactate does not accumulate in skeletal muscles and thus is not the cause of muscle soreness (see Clinical View 10.7: “Muscle Pain Associated with Exercise”).

Energy Supply and Varying Intensity of Exercise

The use of creatine phosphate, glycolysis, and aerobic cellular respiration as the primary means for supplying ATP during physical
activity is dependent upon both the intensity and the duration of the activity. At rest, skeletal muscle obtains the needed ATP almost exclusively through aerobic cellular respiration involving oxidation of fatty acids. To illustrate the use of energy during exercise, we describe the primary means of supplying ATP for runners at a track meet in which individuals run different distances (figure 10.18).

When an individual participates in a 50-meter sprint, an event that may take 5 to 6 seconds, ATP is supplied primarily by available ATP and P_i transfer between two ADP molecules and between creatine phosphate and ADP. In a longer sprint of 400 meters, an event that may take 50 to 60 seconds, ATP is supplied initially by ATP and P_i transfer and then primarily by glycolysis. Finally, in a 1500-meter run, an event that may take 5 to 6 minutes, ATP is supplied by all three means, but primarily by aerobic processes after about the first minute. Keep in mind, however, that there is overlap between the three different energy sources.

Intense exercise that is sustained longer than approximately 1 minute is dependent upon the body’s ability to deliver sufficient oxygen through the cardiovascular and respiratory systems. One consequence of participating in regular aerobic exercise (defined as a sustained exercise of moderate intensity that involves raising the heart rate above the baseline) is that it produces changes within both the respiratory system (see section 23.8b) and the heart and blood vessels of the cardiovascular system (see section 20.7) that enhance oxygen delivery. This allows an individual to more effectively provide ATP through aerobic cellular respiration and, thus, to be able to exercise both at greater levels of intensity and for longer periods of time (see section 10.8a).

**WHAT DID YOU LEARN?**

16. Additional ATP is made immediately available in skeletal muscle through which phosphate-containing molecules?

17. What are the various means for making ATP available in a 1500-meter race?

### 10.5a Criteria for Classification of Muscle Fiber Types

Skeletal muscle fibers that compose a muscle are differentiated into three categories based on two criteria: (1) the type of contraction generated and (2) the primary means used for supplying ATP.

#### Type of Contraction Generated

Skeletal muscle fibers differ in the power, speed, and duration of the muscle contraction generated. **Power** is related to the diameter of a muscle fiber; large muscle fibers have a larger number of myofibrils in parallel, allowing them to produce a more powerful contraction. **Speed** has traditionally been described based on whether the skeletal muscle fiber expresses the relatively slow or fast genetic variant of myosin ATPase, the enzyme that splits ATP (see section 10.3c). Those with a fast variant are called **fast-twitch fibers**, and those with the slow variant are called **slow-twitch fibers**. However, recent
evidence shows that fast-twitch fibers also have both a fast rate of action potential propagation along the sarcolemma and are quick in their Ca\(^{2+}\) release and reuptake by the sarcoplasmic reticulum in comparison to slow-twitch fibers. Thus, fast-twitch fibers initiate a contraction more quickly following stimulation than a slow-twitch fiber (0.01 milliseconds [msec] versus at least 0.02 msec) and produce a contraction of shorter duration (7.5 msec versus 100 msec).

Fast-twitch fibers typically have all three characteristics: They produce a strong contraction, initiate a contraction more quickly following stimulation, and produce a contraction of shorter duration. These characteristics account for why fast-twitch fibers exhibit both power and speed in comparison to slow-twitch fibers.

Means for Supplying ATP
The second criterion to differentiate skeletal muscle fibers is whether the primary means the fiber uses to supply ATP is either aerobic cellular respiration or glycolysis. **Oxidative fibers** specialize in providing ATP through aerobic cellular respiration and have several features that support these processes, including an extensive capillary network, large numbers of mitochondria, and a large supply of the red pigment myoglobin. (The presence of both myoglobin and mitochondria gives these skeletal muscle fibers a red appearance, and they are sometimes called red fibers.) The higher levels of ATP generated provide energy for oxidative fibers to continue contracting for extended periods of time without tiring, or fatiguing—thus, these fibers are also called fatigue-resistant.

In contrast, **glycolytic fibers** specialize in providing ATP more rapidly through glycolysis. Generally, they have fewer structures needed for aerobic cellular respiration—thus, they have less extensive capillary networks, fewer mitochondria, and smaller amounts of myoglobin. (The relatively small amount of myoglobin and mitochondria is why these skeletal muscle fibers have a white appearance and are sometimes called white fibers.) However, they do have large glycogen reserves for supplying glucose for glycolysis, which is useful when oxygen stores are low. Glycolytic fibers generally tire easily after a short time of sustained muscular activity—thus, these fibers are also called fatigable.

### Table 10.1 Structural and Functional Characteristics of Different Types of Skeletal Muscle Fibers

<table>
<thead>
<tr>
<th>Skeletal Muscle Fiber Characteristics</th>
<th>Slow Oxidative (SO) Fibers (Type I Fibers)</th>
<th>Fast Oxidative (FO) Fibers (Type IIa Fibers)</th>
<th>Fast Glycolytic (FG) Fibers (Type IIb Fibers)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ATP Use</strong></td>
<td>Slow</td>
<td>Fast</td>
<td>Fast</td>
</tr>
<tr>
<td><strong>Capacity to Make ATP</strong></td>
<td>High</td>
<td>Moderate</td>
<td>Limited</td>
</tr>
<tr>
<td><strong>Concentration of Capillaries</strong></td>
<td>Extensive</td>
<td>Moderately extensive</td>
<td>Sparse</td>
</tr>
<tr>
<td><strong>Color of Fibers</strong></td>
<td>Red</td>
<td>Lighter red</td>
<td>White (pale)</td>
</tr>
<tr>
<td><strong>Contraction Velocity</strong></td>
<td>Slow</td>
<td>Fast</td>
<td>Fast</td>
</tr>
<tr>
<td><strong>Resistance to Fatigue</strong></td>
<td>Highest</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td><strong>Fiber Diameter</strong></td>
<td>Smallest</td>
<td>Intermediate</td>
<td>Largest</td>
</tr>
<tr>
<td><strong>Number of Mitochondria</strong></td>
<td>Many</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td><strong>Amount of Myoglobin</strong></td>
<td>Large</td>
<td>Medium</td>
<td>Small</td>
</tr>
<tr>
<td><strong>Primary Fiber Function</strong></td>
<td>Endurance (e.g., maintaining posture, marathon running)</td>
<td>Medium duration, moderate movement (e.g., walking, biking)</td>
<td>Short duration, intense movement (e.g., sprinting, lifting weights)</td>
</tr>
<tr>
<td><strong>Muscles with a Large Abundance of Fiber Type</strong></td>
<td>Trunk and lower limb muscles</td>
<td>Lower limb muscles</td>
<td>Upper limb muscles</td>
</tr>
</tbody>
</table>

### WHAT DID YOU LEARN?

19. Explain how a fast-twitch fiber differs from a slow-twitch fiber and how an oxidative fiber differs from a glycolytic fiber.

### 10.5b Classification of Muscle Fiber Types

**LEARNING OBJECTIVE**

23. Compare and contrast the three skeletal muscle fiber types.

Physiologists use both the type of contraction generated and the primary means to supply ATP to differentiate skeletal muscle fibers into three subtypes (table 10.1):

- **Slow oxidative (SO) fibers**, also called type I fibers, typically have half the diameter of other skeletal muscle fibers and contain slow myosin ATPase. These fibers produce contractions that are slower and less powerful. However, they can contract over long periods of time without fatigue because ATP is supplied primarily through aerobic cellular respiration. These fibers appear dark red because of the presence of large amounts of both myoglobin molecules and mitochondria.

- **Fast oxidative (FO) fibers**, also called intermediate fibers or type IIa, are the least numerous of the skeletal muscle fiber types. They are intermediate in size and contain fast myosin ATPase. They produce a fast, powerful contraction with ATP provided primarily through aerobic cellular respiration. However, the vascular supply to fast oxidative fibers is less extensive than the network of capillaries serving SO fibers—thus, the delivery rate of nutrients and oxygen is lower. These fibers also contain myoglobin, but less than the amount found in SO fibers. Consequently, these fibers can be distinguished from SO fibers on a microscopic image because they appear a lighter red than SO fibers.

- **Fast glycolytic (FG) fibers**, also called fast anaerobic fibers or type IIb, are the most prevalent skeletal muscle fiber type. They are largest in diameter, contain fast myosin ATPase, and
provide both power and speed. However, they can contract for only short bursts because ATP is provided primarily through glycolysis. These fibers appear white because of the relative lack of myoglobin and mitochondria.

**WHAT DID YOU LEARN?**

Which skeletal muscle fiber type is slow and fatigue-resistant? What is the advantage of this skeletal muscle fiber type?

### 10.5c Distribution of Muscle Fiber Types

**LEARNING OBJECTIVE**

24. Describe the distribution of skeletal muscle fiber types in a muscle and how this distribution relates to the muscle’s function.

A mixture of muscle fiber types in a typical skeletal muscle is shown in figure 10.19. Although most muscles contain a mixture of all three fiber types, the relative percentage of the muscle fiber types varies among different skeletal muscles of the body and reflects the function of the muscle. For example, the extrinsic muscles of the eye and hand require swift but brief contractions and so they contain a high percentage of FG fibers. In contrast, SO fibers dominate many postural back and calf muscles, which contract almost continually to help us maintain an upright posture.

Variations are also present between individuals, and this is seen most dramatically in high-caliber athletes. Elite distance runners have higher proportions of SO fibers in their lower limb muscles, and top athletes who participate in brief periods of intense activity, such as sprinting or weight lifting, have a higher percentage of FG fibers. These variations in the proportion of the skeletal muscle fiber types are determined primarily by a person’s genes and less so by the type of training. A proportion of FG fibers may develop the appearance and functional capabilities of FO fibers with physical conditioning if the muscle is used repeatedly for endurance events. Whether this shift actually represents a change of skeletal muscle fiber type—or is simply a temporary alteration to the muscle, which reverts back when the training ceases—remains controversial.

**Figure 10.19 Comparison of Fiber Types in Skeletal Muscle.** A cross section of a skeletal muscle using a specific staining technique demonstrates the types of fibers in the muscle. The fibers are distinguished by their shade of color. Slow oxidative (SO) fibers are the darkest; fast oxidative (FO) fibers are less dark than the SO fibers, and fast glycolytic (FG) fibers are the lightest.

**WHAT DID YOU LEARN?**

Muscles that maintain posture are composed primarily of what type of skeletal muscle fibers?

### 10.6 Muscle Tension in Skeletal Muscle

**Muscle tension** is the force generated when a skeletal muscle is stimulated to contract. The term *tension* is used to describe the force that a muscle exerts because a muscle can only pull on a structure. The tension generated by the contractile proteins within a muscle is transferred to its connective tissue coverings, which move a body part (see section 10.2a).

Muscle tension produced in a contracting muscle is measured in several classic laboratory experiments. One variation of these experiments uses the specimen of a gastrocnemius (calf) muscle (see section 11.9c) with an attached sciatic nerve that is excised from a frog. The gastrocnemius muscle is then anchored to an apparatus that produces a *myogram*, a graphic recording of changes in muscle tension when it is stimulated. Here we describe the generation and graphic recording of (1) a muscle twitch; (2) motor unit recruitment; and (3) wave summation, incomplete tetany, and tetany.

**LEARNING OBJECTIVE**

25. Describe what occurs in a skeletal muscle during a single twitch, and relate each event to a graph of a twitch.

Electrodes that are in direct contact with either the skeletal muscle or the sciatic nerve are used to apply single, brief episodes of stimulation to the muscle, and the resulting muscle contraction is recorded using a myograph (figure 10.20). The voltage is increased in increments until the muscle responds, producing a twitch. A *twitch* is defined as a single, brief contraction period and then relaxation period of a skeletal muscle in response to a single stimulation. The minimum voltage needed to stimulate the skeletal muscle to generate a twitch is the *threshold*. The voltage below the threshold is called a *subthreshold stimulus*.

There is a delay called a *latent period* (*lag period*) that occurs after the stimulus is applied and before the contraction of the skeletal muscle fiber begins. There is no change in fiber length during the latent period. This delay can be accounted for by the time necessary for all of the events in excitation-contraction coupling, Ca$^{2+}$ release from the sarcoplasmic reticulum into the cytosol, and the beginning of tension generation within the skeletal muscle fiber. The *contraction period* begins as repetitive power strokes pull the thin filaments past the thick filaments, shortening the sarcomeres; muscle tension increases during muscle contraction. The *relaxation period* begins with release of crossbridges as Ca$^{2+}$ is returned to its storage within the sarcoplasmic reticulum; muscle tension decreases during muscle relaxation. Relaxation depends upon the elasticity of connectin (see figure 10.5b) within muscle tissue to return to its original length following shortening of the muscle.

**WHAT DO YOU THINK?**

Based on skeletal muscle fiber type distribution, would you predict that the duration of a muscle twitch in an extrinsic eye muscle is shorter or longer than it is in the gastrocnemius muscle? Explain.
The time required for a twitch varies based on the predominant type of skeletal muscle fibers composing the muscle (see section 10.5b). The extrinsic eye muscles are predominantly fast-twitch fibers producing a twitch that is as rapid as 7.5 milliseconds, whereas the soleus (deep calf muscle; see figure 11.35) is predominantly slow-twitch fibers producing a twitch that lasts about 100 milliseconds.

**WHAT DID YOU LEARN?**

22. What events are occurring in a muscle that produce the different components of a muscle twitch (latent period, contraction, and relaxation)?

### 10.6b Changes in Stimulus Intensity: Motor Unit Recruitment

**LEARNING OBJECTIVE**

26. Explain the events that occur in motor unit recruitment as the intensity of stimulation is increased.

The gastrocnemius muscle is stimulated repeatedly in a set of experiments to demonstrate motor unit recruitment, and each stimulation event is at a greater voltage. The frequency of stimulation remains the same, and the time between stimulation events is sufficient for the muscle to contract and relax before it is stimulated again. Because motor units vary in their sensitivity to stimulation, each increase in voltage causes a greater number of motor units to contract (figure 10.21). Consequently, the tension generated with each muscle contraction increases until the point of maximum contraction is reached when all motor units have been stimulated. This increase in muscle tension that occurs with an increase in stimulus intensity is called **recruitment**, or **multiple motor unit summation**.

Recruitment helps to account for how our muscles can both exhibit the all-or-none law and exert varying degrees of force. The **all-or-none law** states that if a skeletal muscle fiber contracts in response to stimulation, it will contract completely (all)—and if the stimulus is not sufficient, it will not contract (none). This means that the skeletal muscle fiber contracts maximally or not at all.

The difference in the force and precision of skeletal muscle movement is varied primarily by changing the number of motor units that are activated. If a reduced number of motor units are activated, then fewer skeletal muscle fibers contract and less force is exerted. In contrast, if a greater number of motor units are activated, more skeletal muscle fibers contract and a greater force is exerted. The recruitment of motor units is not random, however. Rather, it is based on size of motor units within a muscle (figure 10.21c). Smallest motor units (which are the most sensitive) are recruited first, with subsequent stimulation of progressively less sensitive larger motor units. This allows for fine motor control when less force is required.
required (e.g., picking up a pencil) and the most power when more force is required (e.g., lifting a suitcase).

**WHAT DID YOU LEARN?**

23. What is recruitment? Explain its importance in the body.

### 10.6c Changes in Stimulus Frequency: Wave Summation, Incomplete Tetany, and Tetany

**LEARNING OBJECTIVE**

27. Distinguish between wave summation, incomplete tetany, and tetany that occur with an increase in frequency of stimulation.

A different set of experiments subjects the skeletal muscle to increasing frequency of stimulation while the voltage remains the same. Note that each of the two graphs in figure 10.22 has a different frequency of stimulation.

The first graph in the figure illustrates the stimulation frequency of a skeletal muscle that occurs at a relatively slow rate (less than 10 stimuli per second) (figure 10.22a). At this rate, each muscle twitch shows that the muscle is contracting and completely relaxing before the next stimulation event. The muscle tension produced in each muscle twitch is the same.

Stimulation can occur so rapidly (e.g., 20 to 50 stimuli per second) that complete relaxation of the skeletal muscle does not occur before the next stimulation event (figure 10.22b). The rapidly restimulated muscle displays a summation of contractile forces as the effect of each new wave is added to the previous wave. This effect is often called either wave summation because contraction waves are added together, or “summed,” or temporal summation because it depends upon increasing frequency (tempo, or timing) of stimulation.

Further increases in stimulation frequency allow less time for relaxation between contraction cycles, and now incomplete tetany (the tension tracing continues to increase and the distance between waves decreases) is noted. Stimulation frequency is further increased (e.g., 40 to 50 stimuli per second) until ultimately the contractions of the skeletal muscle fiber “fuse” and form a continuous contraction that lacks any relaxation. This continuous contraction is called tetany (the tension tracing is a smooth line). If stimulation continues, the muscle reaches fatigue—a decrease in muscle tension occurs from repetitive stimulation (muscle fatigue is described in section 10.7d). Changes in muscle stimulation frequency by the nervous system primarily allow skeletal muscle contraction to exert a coordinated action that gradually increases in force.

Nervous stimulation of skeletal muscles in the human body usually does not exceed 25 stimuli per second. Therefore, muscle tetany is seen only in laboratory experiments. Sustained contractions in the body occur when you are holding something you do not want to drop because the nervous system stimulates different motor units within the same muscle in an overlapping pattern, so the muscle tension can be maintained for a longer period.

**WHAT DID YOU LEARN?**

24. What happens to skeletal muscle during wave summation? Explain its importance in the body.

### 10.7 Factors Affecting Skeletal Muscle Tension Within the Body

The discussion of skeletal muscle tension continues with a description of several factors that influence the action of muscles within the human body, including muscle tone, the length-tension relationship, and whether the muscle tension is generated during an isometric or an isotonic contraction. How muscle fatigue influences our ability to generate muscle tension is described at the end of this section.

#### 10.7a Muscle Tone

**LEARNING OBJECTIVE**


Skeletal muscles do not completely relax, even when at rest. Muscle tone is the resting tension in a skeletal muscle generated by involuntary somatic nervous stimulation of the muscle. Limited numbers of motor units within a muscle are usually stimulated randomly at any given time to maintain a constant tension; the specific motor units being stimulated during rest change continuously so motor units do not become fatigued.

This random contraction of small numbers of motor units causes the skeletal muscle to develop tension, called resting muscle tone. These random contractions do not generate enough tension to cause

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Figure 10.22 Skeletal Muscle Response to Change in Stimulus Frequency. (a) A twitch always produces the same amount of muscle tension. (b) Wave summation, incomplete tetany, and tetany are seen when the muscle is stimulated at varying frequencies that allow different degrees of relaxation.
The resting muscle tone establishes constant tension on the muscle's tendon, thus stabilizing the position of the bones and joints. Another function of muscle tone is to “prime” a muscle for contraction, so that it can respond more readily to stimulation requiring muscle movement. Note that muscle tone decreases during deep sleep (sleep associated with rapid eye movement, or REM, sleep [see section 13.8c]). Consider the difference in muscle tone when carrying an awake child (who has muscle tone) compared to carrying a sleeping child (who temporarily lacks muscle tone). You may have noticed that it is more difficult to carry a sleeping child because the child’s body is less rigid.

**WHAT DID YOU LEARN?**

25. What is the function of skeletal muscle tone?

### 10.7b Isometric Contractions and Isotonic Contractions

#### LEARNING OBJECTIVE

29. Distinguish between isometric and isotonic contractions, and give examples of both.

Two primary factors must be considered when describing the consequences of consciously initiating skeletal muscle contraction: (1) force generated by the muscle and (2) resistance (load) that must be overcome. When skeletal muscle tension is insufficient to overcome the resistance (i.e., force generated is less than the load), there is no movement of the muscle. This type of muscle contraction is called an **isometric** (i-sō-mētr'ık; *iso* = same, *metron* = measure) contraction. Thus, the skeletal muscle contracts and muscle tension increases, but muscle length stays the same. Some examples of isometric contractions include pushing on a wall (a posture for stretching one’s leg muscles), holding a very heavy weight in the gym while your arm does not move, attempting to move a shovel load of snow that is too heavy, and holding a baby in one position (figure 10.23a).

When skeletal muscle tension results in movement of the muscle, this type of muscle contraction is called an **isotonic** (i-sō-ton'ık; *tonos* = tension) contraction. The tone in the skeletal muscle remains the same as the length of muscle changes. Examples of an isotonic contraction include walking, lifting a baby, and swinging a tennis racket. Isotonic contractions are differentiated into two subclasses based on whether the muscle is shortening or lengthening as it contracts (figure 10.23b). The shortening of muscle length is a **concentric contraction**. It occurs because the muscle tension is greater than the resistance. It may occur in the biceps brachii (muscle of the anterior arm) when lifting a baby. In contrast, lengthening of muscle is an **eccentric contraction**. During an eccentric contraction, the muscle exerts less force than that needed to move the load, and the muscle lengthens. If you are holding a 10-pound weight in your hand, and the muscles of your anterior upper arm (e.g., biceps brachii) exert 5 pounds of force, then the biceps muscle lengthens in an eccentric contraction (as your arm extends). This also occurs, for example, in the biceps brachii when placing a baby into a crib. See figure 10.24, showing three graphs that visually represent the relationship of muscle tension and muscle length in the three types of muscle contractions.

**WHAT DID YOU LEARN?**

26. When you flex your biceps brachii while doing “biceps curls,” what is the type of movement?
LEARNING OBJECTIVE

30. Explain the length-tension relationship in skeletal muscle contraction.

The amount of tension a skeletal muscle can generate when stimulated is influenced significantly by the amount of overlap of thick and thin filaments within its muscle fibers when the muscle begins its contraction. This principle is termed the length-tension relationship. A skeletal muscle generates different amounts of tension, depending upon its length at the time of stimulation. The graphical presentation of the length-tension relationship is called the length-tension curve (figure 10.25).

A skeletal muscle fiber stimulated when it is at a normal resting length generates a maximum contractile force because there is optimal overlap of thick and thin filaments, allowing for the largest number of crossbridges to form. In contrast, a muscle that is either already contracted or overly stretched produces a weaker contraction when stimulated. Weaker contractions in muscles that are already contracted occur because the thick filaments are close to the Z discs, and sliding filaments are limited in their movement. Weaker contractions occur in muscles that are overly stretched because there is minimal thick and thin filament overlap for crossbridge formation. So, the tension generated by a skeletal muscle fiber within a muscle is graphically related to its precontraction resting length. (a) If the skeletal muscle fiber is already contracted at the time of stimulation, it does not have the ability to shorten much more, and it exhibits a weak contraction. (b) A skeletal muscle fiber at its normal resting length is generally capable of exhibiting the strongest contraction because of optimal overlap of myofilaments. (c) If the skeletal muscle fiber is very stretched when stimulated, relatively little contraction may occur because myofilaments have minimal overlap.
for example, you may be able to lift a heavier dumbbell when your elbow is partially flexed than when your elbow is fully extended, because there is minimal overlap of thick and thin filaments during the full extension.

Numerous factors have been discussed that influence the amount of muscle tension that is generated by skeletal muscle fibers within the body. The primary four factors are visually summarized in Figure 10.26.

**WHAT DID YOU LEARN?**

**27.** Describe the relative force of contraction that can be developed in your back muscles when you bend at the knees to lift an object and when you bend at the waist to lift an object, based on the length-tension relationship. Explain the significance.

### 10.7d Muscle Fatigue

**LEARNING OBJECTIVE**

**31.** Define muscle fatigue, and explain some of its causes.

**Muscle fatigue** is the reduced ability or the inability of the skeletal muscle to produce muscle tension. The primary cause of muscle fatigue during excessive or sustained exercise (e.g., running a marathon) is a decrease in glycogen stores. However, there are many other causes of muscle fatigue, which are still being debated. Here they are organized by the specific physiologic event of muscle contraction that is affected.

- **Excitation at the neuromuscular junction.** Muscle fatigue may be caused either by insufficient free Ca$^{2+}$ at the neuromuscular junction to enter the synaptic knob or by a decreased number of synaptic vesicles to release neurotransmitter (see section 10.3a). Both limit the ability of somatic motor neurons to stimulate a skeletal muscle.

- **Excitation-contraction coupling.** Muscle fatigue may be due to a change in ion concentration (e.g., Na$^+$, K$^+$) that interferes with the ability of the muscle fiber to conduct an action potential along the sarcolemma (see section 10.3b). This interferes with stimulating release of Ca$^{2+}$ from the sarcoplasmic reticulum.

- **Crossbridge cycling.** Muscle fatigue may result from increased phosphate ion (P$_i$) concentration. Elevated P$_i$ concentration in the muscle sarcoplasm interferes with P$_i$ release from the myosin head during crossbridge cycling, and this slows the rate of cycling. Muscle fatigue also may occur when lower amounts of Ca$^{2+}$ are available for release from the sarcoplasmic reticulum (part of which is due to its binding with the excess P$_i$). Lower Ca$^{2+}$ levels result in less Ca$^{2+}$ binding to troponin, reducing crossbridge formation, which results in a weaker muscle contraction (see section 10.3c). Thus, both an increase in P$_i$ concentration and lower Ca$^{2+}$ levels result in a weaker force generated during muscle contraction.

Lack of ATP is not currently thought to be a primary cause of muscle fatigue. This is because ATP levels are generally maintained through aerobic cellular respiration in mitochondria during sustained exercise. It remains to be determined if ATP may still be a factor because of its location in the cell—that is, within the mitochondria and not in proximity to myofilaments.

**WHAT DID YOU LEARN?**

**28.** How can muscle fatigue result from changes in each of the three primary events of skeletal muscle contraction?
10.8 Effects of Exercise and Aging on Skeletal Muscle

Skeletal muscle is affected by the process of exercise and aging. Here we describe the effects on skeletal muscle of both a sustained exercise program and a lack of exercise, along with the changes that occur as we age.

10.8a Effects of Exercise

LEARNING OBJECTIVE

32. Compare and contrast the changes in skeletal muscle that occur as a result of the two primary types of exercise programs or from the lack of exercise.

Changes in Muscle from a Sustained Exercise Program

The outcome of exercise that involves repetitive stimulation of skeletal muscle fibers depends upon the type of exercise—either endurance exercise or resistance exercise. Endurance (or aerobic) exercise involves sustained, moderate activity that increases heart rate (e.g., running several miles). This type of exercise causes changes that primarily alter how skeletal muscle fibers are supplied with energy (see section 10.4a). The changes that specifically occur to skeletal muscle fibers include (1) an increase in the number of mitochondria and the enzymes within mitochondria, which enhances ATP production through aerobic cellular respiration; (2) an increase in enzymes for using fatty acids in aerobic cellular respiration; and (3) an increase in the amounts of lactate dehydrogenase enzyme (for converting lactate back to pyruvate). The greater availability of fatty acids and pyruvate delays glycogen depletion within skeletal muscle fiber and, thus, fatigue (see section 10.7d).

Endurance exercise also induces changes to the cardiovascular system. The heart wall thickens, which increases the amount of blood that can be pumped by the heart (see section 19.9a), and additional blood vessels form within skeletal muscle through angiogenesis (see section 20.4a). Both of these changes provide more efficient delivery of blood, and additional oxygen is supplied to skeletal muscle. These changes also enhance ATP production through aerobic cellular respiration.

In comparison, resistance exercise, which involves producing forceful muscle contractions (e.g., weight lifting or power lifting), primarily results in stronger skeletal muscles. Resistance exercise stimulates skeletal muscle fibers to increase contractile proteins (myosin, actin), especially in fast glycolytic muscle fibers. These changes primarily result in an increase in muscle size, which is called hypertrophy (hi-për-trō-fē; hyper = above or over, trophe = nourishment). Hypertrophy also results from an increase in the number of mitochondria, greater amounts of myoglobin, and larger glycogen reserves. A slight increase in both ATP and creatine phosphate stores also occurs. Recent evidence suggests that some (limited) increase in the number of muscle fibers also may occur, a process called hyperplasia. An athlete who competes as a bodybuilder or weight lifter might consider using anabolic steroids to stimulate greater muscle mass than would normally occur. However, numerous and significant side effects are associated with their use (see Clinical View 10.9: “Anabolic Steroids as Performance-Enhancing Compounds”).

Changes in Muscle from Lack of Exercise

Lack of exercise (and, thus, lack of muscle use) results primarily in decreasing the skeletal muscle fiber size, a process called atrophy (at’rō-fē; a = without). This causes a decrease in muscle fiber size, tone, and power, and the muscle becomes flaccid. Even a temporary reduction in muscle use can lead to muscular atrophy. Comparing limb muscles before and after a cast that has been worn for a fracture reveals the loss of muscle tone and size for the casted limb. Individuals who suffer damage to the nervous system or are paralyzed by spinal injuries gradually lose skeletal muscle tone and size in the areas affected. Although skeletal muscle atrophy is initially reversible, dead or dying skeletal muscle fibers are not replaced. When extreme atrophy occurs, the loss of gross skeletal muscle function is permanent because muscle is replaced with connective tissue, including adipose connective tissue. For these reasons, physical therapy is required for patients who suffer temporary loss of mobility.

CLINICAL VIEW 10.8

Unbalanced Skeletal Muscle Development

Young athletes today are more likely than in the past to participate in one sport, and to play that sport for more sport seasons; sometimes they play the same sport all year long. Consequently, physical therapists are treating more sports-related injuries. They note that the increase in injuries is often due to unbalanced skeletal muscle development. For example, soccer players are more likely to have overdeveloped hamstring muscles (see figure 11.1b) and underdeveloped quadriceps muscles (see figure 11.1e), putting them at higher risk of injury.

WHAT DID YOU LEARN?

32. What anatomic changes occur in a skeletal muscle fiber when it undergoes hypertrophy?

10.8b Effects of Aging

LEARNING OBJECTIVE

33. Summarize the effects of aging on skeletal muscle.

A slow, progressive loss of skeletal muscle mass typically begins in a person’s mid-30s and becomes more noticeable after the age of 50. There is some loss in skeletal muscle fiber diameter, which is due to a reduction in both the size and the number of myofibrils and the number of myofilaments. However, most loss of muscle mass associated with aging is caused by a decrease in skeletal muscle fiber number. This loss is partly due to decreased physical activity. There is also a progressive loss of motor neurons that stimulate skeletal muscle fibers, which results in skeletal muscle fiber atrophy or loss. Other motor neurons can innervate the skeletal muscle fibers; however, this results in a decrease in fine motor control (because of the increase in motor unit size). Thus, elderly individuals progressively have more difficulty with physical tasks and balance. Strength training can slow the progress by increasing muscle size.

The ability to produce ATP also decreases with aging due to a variety of factors, including a reduction in myoglobin (less oxygen storage capacity) and decreased glycogen storage. Overall, muscle strength and endurance are impaired, and the individual has a tendency to fatigue more quickly. Decreased cardiovascular performance often accompanies aging; thus, the blood supplied to active skeletal muscles is much less in elderly people when they exercise.

Skeletal muscle tissue has a reduced capacity to recover from disease or injury as a person grows older. The number of satellite cells
Anabolic (an-ā-bô’lik) steroids are synthetic substances that mimic the actions of natural testosterone. Recall from section 3.2b that the term anabolism refers to the synthesis of complex molecules (e.g., protein) from simple molecules (e.g., amino acids). To date, over 100 compounds have been developed with anabolic properties, but they all require a prescription for legal use in the United States. Anabolic steroids have only a few accepted medical uses—among them, the treatment of delayed puberty, certain types of impotence, and the wasting condition associated with HIV infection and other diseases. Because anabolic steroids stimulate the manufacture of muscle proteins, these compounds have been used by some athletes as performance enhancers.

Relatively large doses of anabolic steroids are needed to stimulate excessive muscle development that results in extra strength and speed. But this enhanced muscle power and speed comes at a price. Medical professionals have reported many devastating side effects associated with extended anabolic steroid use, including increased risk of heart disease and stroke, kidney damage, liver tumors, testicular atrophy and a reduced sperm count, gynecomastia (enlargement of breast glandular tissue in males), acne, high blood pressure, aggressive behavior, and personality aberrations. Because anabolic steroids mimic the effects of testosterone, female athletes who use them often experience menstrual irregularities, growth of facial hair, and in extreme circumstances atrophy of the uterus and mammary glands; sterility has even been reported. Adding to these problems is the route of administration. Because many of these steroid preparations must be injected, the improper use or sharing of needles raises the possibility of transferring pathogens that cause disease (e.g., AIDS or hepatitis). For all of these reasons, the use of anabolic steroids as performance-enhancing compounds has been widely banned.

Anabolic Steroids as Performance-Enhancing Compounds

In addition to skeletal muscle, two other types of muscle occur in the body: cardiac muscle and smooth muscle (see section 5.3). Here we briefly review the characteristics of cardiac muscle (for a detailed description of cardiac muscle, see section 19.3f).

Cardiac muscle cells are individual muscle cells arranged in thick bundles within the heart wall (figure 10.27). Cardiac muscle cells branch and are both shorter and thicker than skeletal muscle fibers. (They typically have a diameter of about 15 µm and range in length from 50 to 100 µm.) Individual cells are joined to adjacent muscle cells at junctions termed intercalated discs. Intercalated (in-ter’kâ-lât’d) discs are unique to cardiac muscle; they are composed of desmosomes and gap junctions (see section 4.6d). These cells have only one or two nuclei. Cardiac muscle cells are striated because, like skeletal muscle fibers, cardiac muscle cells also contain sarcomeres. Cardiac muscle cells contain a large number of mitochondria, and they use aerobic cellular respiration almost exclusively to generate the ATP required for their unceasing work. In addition, the sarcoplasmic reticulum is not as well developed as in skeletal muscle, so most Ca$^{2+}$ enters the cardiac muscle fibers from interstitial fluid instead.

Cardiac muscle cells are stimulated by a specialized autorhythmic pacemaker. This feature is responsible for the repetitious, rhythmic heartbeat. The autonomic nervous system (a division of the nervous system that controls cardiac muscle contraction, smooth muscle contraction, and gland secretion, which is discussed in chapter 15) controls both the rate and the force of contraction of cardiac muscle.

Figure 10.27 Cardiac Muscle. Cardiac muscle is found only in the heart walls. Cardiac muscle cells branch and are connected by intercalated discs. AP R

WHAT DID YOU LEARN?

What changes in skeletal muscle occur as a result of aging?

10.9 Cardiac Muscle Tissue

LEARNING OBJECTIVE

34. List and describe the similarities and differences between skeletal muscle and cardiac muscle.

In addition to skeletal muscle, two other types of muscle occur in the body: cardiac muscle and smooth muscle (see section 5.3). Here we briefly review the characteristics of cardiac muscle (for a detailed description of cardiac muscle, see section 19.3f).

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Cardiac muscle cells are stimulated by a specialized autorhythmic pacemaker. This feature is responsible for the repetitious, rhythmic heartbeat. The autonomic nervous system (a division of the nervous system that controls cardiac muscle contraction, smooth muscle contraction, and gland secretion, which is discussed in chapter 15) controls both the rate and the force of contraction of cardiac muscle.
10.10 Smooth Muscle Tissue

Smooth muscle tissue is located throughout the body, typically composing approximately 2% of the weight of an adult. Here we describe the general features of smooth muscle, including locations, microscopic anatomy, mechanism of contraction, how it is controlled, and its functional categories.

10.10a Location of Smooth Muscle

**LEARNING OBJECTIVE**

35. Identify organs of various body systems where smooth muscle is located.

Smooth muscle tissue is found in the walls of organs of many different body systems. Smooth muscle function is determined by its location. The following are examples (figure 10.28):

- **Cardiovascular system**: Blood vessels alter blood pressure and the distribution of blood flow.
- **Respiratory system**: Bronchioles (air passageways) control the resistance to airflow that enters and exits the air sacs (alveoli) of the lungs.
- **Digestive system**: The stomach, small intestine, and large intestine mix and propel ingested material as it is moved along the gastrointestinal tract.
- **Urinary system**: Ureters propel urine from the kidney to the urinary bladder, which eliminates urine from the body.
- **Female reproductive system**: The uterus helps expel the baby during delivery.

These are but a few examples of smooth muscle distribution in our bodies. Smooth muscle also composes other specialized structures, including the iris of the eye to control the amount of light entering the eye, the ciliary body of the eye for focusing on an object (see section 16.4b), and the arrector pili muscles that produce *goose bumps* (see section 6.2b).

Smooth muscle is similar to both skeletal muscle and cardiac muscle because it has cells that can increase in size (hypertrophy). Both skeletal muscle and cardiac muscle, however, have limited ability to increase in cell number through mitosis (hyperplasia), whereas smooth muscle retains this ability. The smooth muscle in the wall of the uterus exemplifies this characteristic as it increases in thickness during pregnancy from a combination of hypertrophy and hyperplasia (see section 29.5c). There is a significant advantage for any tissue that retains mitotic ability because following an injury the damaged tissue is replaced with the original tissue (not scar tissue) (see section 4.9). Thus, as you consider the various locations of smooth muscle in the body, keep in mind that if smooth muscle is damaged, it is typically replaced with new smooth muscle tissue and has the potential to continue functioning as it did before.

**WHAT DID YOU LEARN?**

32. Where is smooth muscle located in the human body?

10.10b Microscopic Anatomy of Smooth Muscle

**LEARNING OBJECTIVE**

36. Compare the microscopic anatomy of smooth muscle to skeletal muscle.

Smooth muscle cells are small and fusiform shaped (widest in the middle and tapered on the ends) with a centrally located nucleus (figure 10.29). They typically have a diameter of 5 to 10 micrometers and a length of 50 to 200 micrometers. Thus, their diameter is up to 10 times smaller and their length thousands of times shorter than a skeletal muscle fiber. An endomysium wraps around each smooth muscle cell. The small, tapered ends of the cells overlap the larger, middle area of adjacent cells to provide close packing of cells.

The sarcolemma contains various types of Ca\(^2+\) channels (e.g., voltage-gated, chemically gated, modality gated) that allow these cells to respond to different types of stimuli. Transverse tubules are absent in smooth muscle cells. Instead, the sarcolemmal surface area is increased by invaginations called *caveolae* (kav′ē-ō-lē; pl. *caveola*; small pocket). The sarcoplasmic reticulum is sparse and located close to the sarcolemma, with some caveolae in contact with it. The source of Ca\(^2+\) comes both from the interstitial fluid outside the cell and the sarcoplasmic reticulum.

**Arrangement of Anchoring Proteins and Contractile Proteins of Smooth Muscle**

Smooth muscle contains a unique arrangement of anchoring protein structures that includes the cytoskeleton, dense bodies, and dense...
plasmas. The cytoskeletal network is composed of an extensive array of intermediate filaments (see section 4.6b). The intermediate filaments are linked with dense bodies at points where they interact within the sarcoplasm of the smooth muscle cell, whereas intermediate filaments are linked by dense plaques at points where they attach on the inner surface of the sarcolemma. Thus, intermediate filaments extend across the cell, with dense bodies as “spot welds” that anchor intermediate filaments to each other and dense plaques that anchor the intermediate filaments to the plasma membrane.

The contractile proteins in smooth muscle are arranged between dense bodies and dense plaques, rather than in sarcomeres as they are in both skeletal and cardiac muscle. The Z discs that anchor the sarcomere on either end in skeletal muscle fibers are also absent. Lack of sarcomeres and Z discs contributes to the absence of striations, giving these muscle cells their “smooth” appearance.

The contractile proteins are oriented at oblique angles to the longitudinal axis of the smooth muscle cell and appear to spiral. Consequently, contraction (as described in section 10.10c) results in a twisting of the smooth muscle, which is similar to a corkscrew (figure 10.29c).

Comparison of Myofilaments of Smooth Muscle and Skeletal Muscle
Thick filaments in smooth muscle have myosin heads along their entire length, rather than only at the ends as in skeletal muscle. The more numerous heads can form additional crossbridges with actin to produce a powerful muscle contraction. Additionally, these myosin heads have modifications that allow them to “latch on” to the actin of thin filaments and remain attached without using additional ATP. This mechanism is called the latchbridge mechanism.

WHAT DO YOU THINK?
How does the latchbridge mechanism within smooth muscle cells influence (a) the amount of ATP required for muscle contraction and (b) whether or not smooth muscle is fatigue-resistant?

Smooth muscle thin filaments are composed of actin and tropomyosin, but they do not contain troponin molecules as in skeletal muscle fibers and cardiac muscle cells. Instead, two other proteins are required for initiation of smooth muscle contraction: (1) calmodulin, a protein that binds Ca\(^{2+}\) to form a Ca\(^{2+}\)-calmodulin complex, and (2) myosin light-chain kinase (MLCK), an enzyme that is activated by the Ca\(^{2+}\)-calmodulin complex to phosphorylate the smooth muscle myosin head. The phosphorylation of the smooth muscle myosin head causes activation of its ATPase activity.

A third protein called myosin light-chain phosphatase is an enzyme that dephosphorylates the myosin head, resulting in the inactivation of the ATPase activity. This inactivation is required for relaxation of smooth muscle.

10.10c Mechanism of Smooth Muscle Contraction

LEARNING OBJECTIVE
37. Explain the sequence of steps in smooth muscle contraction.

Smooth muscle contraction resembles skeletal muscle contraction in that it (1) is initiated by Ca\(^{2+}\), (2) involves the sliding of thin filaments past thick filaments, and (3) requires ATP. There are significant differences, however, as shown and described in figure 10.30.

In response to stimulation, Ca\(^{2+}\) enters the smooth muscle cell cytosol from both the interstitial fluid and the sarcoplasmic reticulum. It binds to calmodulin to form a Ca\(^{2+}\)-calmodulin complex, which then binds to MLCK, resulting in its activation. The activated kinase (MLCK) phosphorylates the myosin head to both activate the myosin ATPase activity of the myosin head and allow the myosin head to bind to actin forming a crossbridge. Crossbridge cycling occurs repetitively, as it does in skeletal muscle (see figure 10.13), although more slowly. This results in the thin filament sliding past the thick filament and pulling on the attached dense bodies anchored to the intermediate filaments of the cytoskeleton and the dense plaques.
attached to the sarcolemma. The anchoring filaments move inward, and the entire smooth muscle cell shortens.

Relaxation of smooth muscle is more complex than relaxation of skeletal muscle. In addition to requiring both cessation of stimulation and the removal of Ca\(^{2+}\) from the sarcoplasm, smooth muscle relaxation also requires the dephosphorylation of myosin by myosin light-chain phosphatase. Note that smooth muscle may remain in the contracted state following the removal of Ca\(^{2+}\) and dephosphorylation of the myosin head due to the special latchbridge mechanism that keeps the myosin attached to the thin filament.

**Characteristics of Smooth Muscle Contraction**

Smooth muscle contraction exhibits three characteristics that allow it to effectively fulfill its functions.

**Prolonged Duration of Contraction** Smooth muscle contraction is usually slow to develop with maximum tension at about 500 milliseconds after stimulation. The relatively long latent period is due primarily to both the requirement for phosphorylating the myosin head by MLCK enzymes and variations in the speed of myosin ATPase activity. The duration of contraction typically extends over a 1- to 2-second period due to the slowness of Ca\(^{2+}\) pumps in removing Ca\(^{2+}\) from the cytosol, the requirement for dephosphorylation of the myosin head by phosphatase, and the possibility of myosin locking to actin (latchbridge mechanism). Contraction of smooth muscle does not generally require a rapid onset, but it does require the ability to remain in the contracted state for extended periods of time. This characteristic is important because smooth muscle must maintain continuous tone (tonic contraction) in visceral walls, such as the gastrointestinal tract and blood vessels.

**Fatigue-Resistant** The energy requirements for smooth muscle contraction are relatively low in comparison to skeletal muscle, and ATP is generally supplied through aerobic cellular respiration. The latchbridge mechanism provides the means of maintaining muscle contraction without use of additional ATP. Consequently, smooth muscle is fatigue-resistant (i.e., it may contract for extended periods of time without becoming fatigued). This characteristic is obviously a requirement for maintaining the tonic contractions just described.

**Broader Length-Tension Curve** Smooth muscle exhibits a broader length-tension curve than skeletal muscle. Recall that the force of muscle contraction generated by skeletal muscle is dependent upon its muscle length at the time of stimulation. It shows maximum force at its optimal resting length, but a decreased force of contraction if it is either shortened or lengthened (see figure 10.25). These limitations are due to the Z discs that prevent additional shortening and lack of myosin heads in the center of thick filaments, respectively. Smooth muscle has neither of these limitations—but, it can contract forcefully when compressed to approximately half its resting length or stretched to twice its resting length. Consider, for example, that the storage of urine in the urinary bladder wall results in stretching of the urinary bladder wall (see section 24.8c). The greater the amount of urine, the greater the amount of stretch of the smooth muscle in the urinary bladder wall. The ability of smooth muscle to contract forcefully at varying degrees of stretch allows us to easily empty our bladder regardless of the amount of urine it is holding.

**WHAT DID YOU LEARN?**

- What are the steps of smooth muscle contraction?
- What unique characteristics of smooth muscle allow it to fulfill its functions? Explain.
10.10d Controlling Smooth Muscle

**LEARNING OBJECTIVE**

38. Briefly explain the different means of controlling smooth muscle.

We cannot voluntarily control the contraction of the smooth muscle in the wall of the digestive tract, as we find when our stomach “growls” at an inappropriate time. Smooth muscle, like cardiac muscle, is controlled by the autonomic nervous system. The response of smooth muscle to stimulation by the nervous system—that is, whether it contracts or relaxes—is dependent upon the specific neurotransmitter that is released and the receptors to which the neurotransmitter binds. Smooth muscle within the walls of bronchioles, for example, contracts in response to the release of ACh, and relaxes in response to norepinephrine.

Smooth muscle also contracts in response to being stretched. This physiologic response is called the myogenic (mi’o-jen’ik; genesis = origin) response. The myogenic response occurs, for example, in smooth muscle in the walls of blood vessels, the stomach, and the urinary bladder. Its response, however, is not continuous if the stretch is prolonged. Instead, the smooth muscle exhibits what is called the stress-relaxation response. This occurs when smooth muscle is “stressed” by being stretched. It responds by contracting, but after a given period of time, it relaxes. For example, swallowed materials entering the stomach cause its wall to stretch, and the smooth muscle in the wall initially contracts. After a period of time it relaxes, allowing additional food to more easily enter the stomach.

Smooth muscle is also stimulated to contract by various hormones, a decrease in pH, low oxygen concentration, increased carbon dioxide levels, certain drugs, and pacemaker cells. For example, the hormone oxytocin causes contraction of smooth muscle in the uterus to expel the baby at childbirth (see section 29.6c). A pacemaker (similar to the pacemaker in the heart) stimulates smooth muscle in the walls of the stomach, and relaxes in response to norepinephrine.

Most smooth muscle belongs to the category of single-unit smooth muscle. The smooth muscle cells of single-unit smooth muscle typically form two or three sheets. These sheets of smooth muscle are within the walls of the digestive, urinary, and reproductive tracts, as well as smaller portions of the respiratory tract and most blood vessels. These large sheets of smooth muscle are functionally linked by gap junctions between cells.

Nerve stimulation of single-unit smooth muscle occurs through numerous swellings of the autonomic motor neurons that pass in close proximity to several smooth muscle cells; these swellings are called varicosities (figure 10.31b). Synaptic vesicles within the varicosities contain one type of neurotransmitter (e.g., ACh, norepinephrine). Receptors in smooth muscle cells are scattered diffusely across the sarcolemma of these cells. This contrasts to their distribution in skeletal muscle cells, where receptors are clustered in a motor end plate (see figure 10.7). This scattered and loose arrangement of receptors in single-unit smooth muscle is called a diffuse junction. Neurotransmitter released from varicosities stimulates numerous smooth muscle cells simultaneously. This occurs much like the extensions of a sprayer releasing water onto a lawn or garden. The stimulation may subsequently be spread from cell to cell via gap junctions, and smooth muscle cells contract synchronously as one unit.

Significant features of skeletal muscle, cardiac muscle, and smooth muscle are compared in table 10.2.

**CONCEPT CONNECTION**

Blood flow into a body structure may remain relatively constant (despite changes in systemic blood pressure) due to the myogenic response of the smooth muscle in blood vessels (see section 20.4b), which is especially important in regulating blood flow into the glomeruli (capillaries) of the kidney through renal autoregulation (see section 24.5e).

**WHAT DID YOU LEARN?**

37. What are the various forms of stimulation for controlling smooth muscle?

38. Explain the stress-relaxation response of smooth muscle.

**10.10e Functional Categories of Smooth Muscle**

**LEARNING OBJECTIVES**

39. Explain the primary functional difference between multiunit and single-unit smooth muscle.

40. Compare the location and regulation of both multiunit and single-unit smooth muscle.

Smooth muscle is classified into two broad groups based upon whether the smooth muscle fibers are stimulated to contract either independently or as one unit. Multiunit smooth muscle cells receive stimulation to contract individually, whereas single-unit smooth muscle cells are stimulated to contract in unison (figure 10.31).

**Multiunit smooth muscle** is found within the eye in both the iris and ciliary muscles (see figure 16.15), composing the arrector pili muscles in the skin (see figure 6.10), the wall of larger air passageways within the respiratory system (see figure 23.1), and the walls of larger arteries (see figure 20.3). Smooth muscle cells in these body structures are arranged into motor units, and they have a neuromuscular junction (figure 10.31a). These two features are similar to skeletal muscle, except that the motor neuron here is a component of the autonomic nervous system. The degree of contraction of this smooth muscle is dependent upon the number of motor units activated, thus facilitating increasing degrees of tension as more motor units are stimulated.
<table>
<thead>
<tr>
<th>Muscle Tissue</th>
<th>Skeletal Muscle</th>
<th>Cardiac Muscle</th>
<th>Smooth Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Location</strong></td>
<td>Generally attached to the skeleton (or to subcutaneous tissue); voluntary movement of skeleton, skin or other select body components</td>
<td>Heart only; pumps blood through blood vessels</td>
<td>Walls of hollow organs (e.g., intestines, blood vessels); also located in iris and ciliary body of eye, and arrector pili of the integument; involuntary movement of body components</td>
</tr>
<tr>
<td><strong>Associated Connective Tissue</strong></td>
<td>Epimysium, perimysium, endomysium</td>
<td>Endomysium only</td>
<td>Endomysium only</td>
</tr>
<tr>
<td><strong>Cell Appearance and Shape</strong></td>
<td>Long, cylindrical fibers with multiple peripheral nuclei; striated; T-tubules Diameter: Large (10–500 μm) Length: Long (100 μm–30 cm)</td>
<td>Medium-sized, branching cells with one or two centrally located nuclei; striated; T-tubules; intercalated discs Diameter: Medium (about 15 μm) Length: Short (50–100 μm)</td>
<td>Small, overlapping, fusiform cells with a single centrally located nucleus; nonstriated; caveolae Diameter: Small (5–10 μm) Length: Short (50–200 μm)</td>
</tr>
<tr>
<td><strong>Calcium Source</strong></td>
<td>Well-developed sarcoplasmic reticulum</td>
<td>Sarcoplasmic reticulum not as well developed as in skeletal muscle; most Ca(^{2+}) from interstitial fluid</td>
<td>Sarcoplasmic reticulum not well developed; most Ca(^{2+}) from interstitial fluid</td>
</tr>
<tr>
<td><strong>Contractile Unit and Ca(^{2+}) Binding</strong></td>
<td>Sarcomere; Ca(^{2+}) binds to troponin</td>
<td>Sarcomere; Ca(^{2+}) binds to troponin</td>
<td>No sarcomeres; calcium binds calmodulin, not troponin</td>
</tr>
<tr>
<td><strong>Stimulation</strong></td>
<td>Nervous control is voluntary (regulated by somatic nervous system at neuromuscular junction); excitatory</td>
<td>Autorhythmic due to pacemaker within heart; spread by gap junctions; nervous control is involuntary (by the autonomic nervous system); excitatory or inhibitory</td>
<td>Multiunit smooth muscle: Regulated by autonomic nervous system at neuromuscular junction; excitatory or inhibitory; no gap junctions Single-unit smooth muscle: Autonomic nervous stimulation through varicosities spread by gap junctions; other stimuli (e.g., stretch, pH)</td>
</tr>
<tr>
<td><strong>Rate of Response and Primary Energy Source</strong></td>
<td>Slow oxidative (SO): Slow; aerobic production of ATP Fast oxidative (FO): Rapid and powerful; aerobic production of ATP Fast glycolytic (FG): Rapid and powerful; glycolytic production of ATP</td>
<td>Slow; aerobic production of ATP</td>
<td>Very slow and long duration; aerobic production of ATP</td>
</tr>
<tr>
<td><strong>Tissue Repair Ability</strong></td>
<td>Limited</td>
<td>Limited</td>
<td>Significant ability for repair</td>
</tr>
</tbody>
</table>

(photos): (skeletal muscle) ©Ed Reschke/Getty Images; (cardiac muscle) ©Victor P. Eroschenko; (smooth muscle) ©Victor P. Eroschenko
## CHAPTER SUMMARY

- Muscle tissue moves the skeleton and materials within and throughout the body.

### 10.1 Introduction to Skeletal Muscle
- Skeletal muscles have a number of functions and exhibit certain properties.

#### 10.1a Functions of Skeletal Muscle
- Skeletal muscles produce body movement, maintain posture, protect and support body structures, move and eliminate materials, and produce heat to help maintain body temperature.

#### 10.1b Characteristics of Skeletal Muscle
- Skeletal muscle tissue exhibits excitability, conductivity, contractility, extensibility, and elasticity.

### 10.2 Anatomy of Skeletal Muscle
- Individual skeletal muscles may extend the entire length of a muscle and are called muscle fibers.

#### 10.2a Gross Anatomy of Skeletal Muscle
- Skeletal muscle is ensheathed by three connective tissue layers: an epimysium, a perimysium, and endomysium.
- Tendons or aponeuroses are extensions of these three layers of connective tissue that attach muscle ends to other structures.
- Skeletal muscle is highly vascularized, and it is innervated by motor neurons that exert voluntary control of the muscle.

#### 10.2b Microscopic Anatomy of Skeletal Muscle
- A skeletal muscle fiber is a multinucleated cell.
- The sarcolema, T-tubules, and sarcoplasmic reticulum have specialized membrane pumps and channels that participate in muscle excitability, conductivity, and initiation of muscle contraction.
- Skeletal muscle fibers are filled with myofilaments that house thick and thin protein myofilaments, which are composed of myosin and actin protein, respectively.
- Myofilaments are arranged in repeating, functional units called sarcomeres.
- Other specialized structural and functional proteins of muscle tissue fibers include connectins (titin) and dystrophin.
- Numerous mitochondria, glycogen stores, myoglobin, and creatine phosphate all function to help meet the high energy demands of skeletal muscle tissue.

#### 10.2c Innervation of Skeletal Muscle Fibers
- A motor unit consists of a motor neuron and all the muscle fibers it innervates and controls.
- The neuromuscular junction is the location where a motor neuron innervates a muscle fiber.

#### 10.2d Skeletal Muscle Fibers at Rest
- At rest, skeletal muscle fibers have an RMP of –90 mV with more Na\(^+\) outside the cell and more K\(^+\) inside the cell.

### 10.3 Physiology of Skeletal Muscle Contraction
- Skeletal muscle physiology involves three major events: excitation, excitation-contraction coupling, and crossbridge cycling.

#### 10.3a Neuromuscular Junction: Excitation of a Skeletal Muscle Fiber
- Excitation involves the arrival of a nerve signal to stimulate release of the neurotransmitter acetylcholine (ACh) contained within synaptic vesicles.

#### 10.3b Sarcolemma, T-tubules, and Sarcoplasmic Reticulum: Excitation-Contraction Coupling
- Excitation-contraction coupling links excitation of the muscle by the motor neuron to muscle contraction through the sarcolemma, T-tubules, and sarcoplasmic reticulum.

#### 10.3c Sarcomere: Crossbridge Cycling
- Crossbridge cycling is initiated by the release of Ca\(^{2+}\) from the sarcoplasmic reticulum. This allows myosin heads to bind to actin to pull thin filaments past thick filaments. This process is the sliding filament theory.

#### 10.3d Skeletal Muscle Relaxation
- Muscle returns to the resting state through the natural elasticity of the muscle fiber.

### 10.4 Skeletal Muscle Metabolism
- Skeletal muscle tissue exhibits a high metabolic demand for energy.

#### 10.4a Supplying Energy for Skeletal Muscle Metabolism
- The major means to supply energy to skeletal muscle are limited amounts of ATP, phosphate transfer, glycolysis, and aerobic cellular respiration.
- The duration and intensity of activity determine the primary means of supplying ATP.

#### 10.4b Oxygen Debt
- Oxygen debt is the additional oxygen that must be taken in after exercise to restore pre-exercise conditions.

### 10.5 Skeletal Muscle Fiber Types
- Two criteria are used to classify skeletal muscle fiber types into three categories.

#### 10.5a Criteria for Classification of Muscle Fiber Types
- The criteria for classifying muscle fiber types include the type of contraction generated (power, speed, duration) and the primary means of supplying ATP (glycolysis and aerobic cellular respiration).
### 10.5b Classification of Muscle Fiber Types
- The three subtypes of skeletal muscle fibers are slow oxidative (SO) fibers, fast oxidative (FO) fibers, and fast glycolytic (FG) fibers.

### 10.5c Distribution of Muscle Fiber Types
- Skeletal muscles typically contain all muscle fiber types; however, the relative percentage will vary between different skeletal muscles of the body and between individuals for certain muscles, such as the muscles of the leg.

### 10.6 Muscle Tension in Skeletal Muscle
- Muscle tension is the force generated when a muscle is stimulated to contract.

#### 10.6a Muscle Twitch
- A muscle twitch is a single, brief contraction and then relaxation of a skeletal muscle fiber in response to stimulation.

#### 10.6b Changes in Stimulus Intensity: Motor Unit Recruitment
- Increasing stimulus intensity results in recruitment of additional motor units to produce progressively more muscle tension.

#### 10.6c Changes in Stimulus Frequency: Wave Summation, Incomplete Tetany, and Tetany
- Increasing the stimulus frequency produces three observable changes in the graphic records of muscle tension: wave summation, incomplete tetany, and complete tetany.

### 10.7 Factors Affecting Skeletal Muscle Tension Within the Body
- Four factors are associated with muscle tension in the human body: muscle tone, isometric and isotonic contractions, length-tension relationship, and muscle fatigue.

#### 10.7a Muscle Tone
- Muscle tone is the resting tension in a skeletal muscle to stabilize joints.

#### 10.7b Isometric Contractions and Isotonic Contractions
- Isometric contractions result in the production of tension that does not exceed resistance; there is no muscle shortening.
- Isotonic contractions produce tension that exceeds the resistance; skeletal muscle fibers change length. In concentric isotonic contractions, muscle fibers shorten, and in eccentric isotonic contractions, muscle fibers lengthen.

#### 10.7c Length-Tension Relationship
- Muscle tension is influenced by the degree of overlap of myofilaments at stimulation; this is described as the length-tension relationship.

#### 10.7d Muscle Fatigue
- Fatigue is the reduced ability, or the inability, of the muscle to produce a contractile force.

### 10.8 Effects of Exercise and Aging on Skeletal Muscle
- Skeletal muscle is affected by exercise and aging.

#### 10.8a Effects of Exercise
- The effects of exercise are dependent upon whether the exercise program is primarily an endurance exercise program or a resistance exercise program.

#### 10.8b Effects of Aging
- The general response of skeletal muscle tissue to aging is progressive atrophy and fibrosis that is accelerated by lack of exercise.

### 10.9 Cardiac Muscle Tissue
- Cardiac muscle is composed of striated, branching cells located in the wall of the heart. The rhythm of contraction is controlled by a pacemaker and altered involuntarily by the autonomic nervous system.

### 10.10 Smooth Muscle Tissue
- Smooth muscle is present throughout the body, typically composing approximately 2% of the weight of an adult.

#### 10.10a Location of Smooth Muscle
- Smooth muscle is located in the walls of most organs and in other specialized structures.

#### 10.10b Microscopic Anatomy of Smooth Muscle
- The contractile, anchoring, and regulatory proteins of smooth muscle have several significant differences from skeletal and cardiac muscle, including the lack of striations.

#### 10.10c Mechanism of Smooth Muscle Contraction
- Smooth muscle contraction occurs by sliding of thin filaments past thick filaments, but the mechanism is different from skeletal and cardiac muscle contraction.

#### 10.10d Controlling Smooth Muscle
- Smooth muscle is controlled by the autonomic nervous system, stretch, and various other types of stimuli (e.g., pH).

#### 10.10e Functional Categories of Smooth Muscle
- Smooth muscle is categorized into multiunit smooth muscle and single-unit smooth muscle based on whether the fibers contract independently or as one unit.
1. The unit of skeletal muscle structure that is composed of bundles of myofibrils, enclosed within a sarcolemma, and surrounded by a connective tissue covering called endomysium is a
   a. myofibril.
   b. fascicle.
   c. myofilament.
   d. skeletal muscle fiber.

2. The physiologic event that takes place at the plasma membrane of a skeletal muscle fiber is
   a. release of calcium.
   b. propagation of an action potential.
   c. binding of calcium by troponin.
   d. crossbridge cycling.

3. In a skeletal muscle fiber, Ca\(^{2+}\) is released from
   a. ACh receptors.
   b. the motor end plate.
   c. the sarcoplasmic reticulum.
   d. the sarcolemma and T-tubules.

4. The bundle of dense regular connective tissue that attaches a skeletal muscle to bone is called a(n)
   a. tendon.
   b. ligament.
   c. endomysium.
   d. fascicle.

5. In excitation-contraction coupling, the T-tubules function to
   a. conduct an action potential into the sarcoplasmic reticulum to cause release of calcium.
   b. uptake and store excess Na\(^+\) and K\(^+\) from the sarcoplasm.
   c. keep the thin and thick myofilaments separated.
   d. provide structural support for sarcomeres.

6. During muscle contraction, the I band
   a. hides the H zone.
   b. shortens or narrows.
   c. overlaps the Z line.
   d. always remains the same length.

7. During a concentric contraction of a skeletal muscle fiber, myofibrils
   a. lengthen.
   b. remain the same length.
   c. increase in diameter.
   d. shorten.

8. What event causes a troponin-tropomyosin complex to regain its original shape in muscle relaxation?
   a. stimulation of ACh receptors
   b. diffusion of Na\(^+\) back into transverse tubules
   c. return of Ca\(^{2+}\) into the sarcoplasmic reticulum
   d. breaking of the bond with tropomyosin

9. In sustained, moderate exercise, skeletal muscle is predominantly using ____________ as its energy source.
   a. amino acids
   b. glucagon
   c. fatty acids
   d. creatine phosphate

10. Skeletal muscle and cardiac muscle are similar in that both types of muscle
    a. have cells that branch.
    b. contain intercalated discs.
    c. are under involuntary control.
    d. are striated.

11. Explain the structural relationship between a sarcomere, a myofibril, a myofilament, and a skeletal muscle fiber.

12. Diagram and label a sarcomere, including a thick filament, thin filament, A band, H zone, I band, Z disc, and M line.

13. Explain why the ratio of motor neurons to skeletal muscle fibers is greater in muscles that control eye movement than in postural muscles of the leg.

14. Put the following skeletal muscle contraction events in the order that they occur:
    a. The myosin head swivels toward the center of the sarcomere.
    b. Calcium ions are released from the sarcoplasmic reticulum and bind to troponin.
    c. An action potential is propagated along the sarcolemma and transverse tubules.
    d. Myosin binds to actin, forming crossbridges.
    e. Myosin heads bind ATP molecules and release from actin.
    f. Tropomyosin molecules are moved off active sites on actin.
    g. ATPase splits ATP, providing the energy to reset the myosin head.

15. Explain the various means of providing ATP for skeletal muscle contraction.

16. Explain why athletes who excel at short sprints probably have fewer slow-twitch fibers in their lower limb muscles.

17. Explain why skeletal muscle generates the most force when it is at its resting length at the time of stimulation based on the length-tension relationship.

18. Describe the characteristics of smooth muscle that allow it to contract for extended periods of time and not fatigue.

19. Describe the response of smooth muscle to sustained stretch.

20. Identify the location of both multiunit smooth muscle and single-unit smooth muscle, and explain the difference in how each is regulated.
Can You Apply What You’ve Learned?

1. A bacterial toxin is known to block the release of ACh at the motor end plate of skeletal muscle. Consequently,
   a. the skeletal muscle contracts with increasing force.
   b. the skeletal muscle contracts with increasing frequency.
   c. the ability to stimulate the muscle is impaired.
   d. other neurotransmitters would stimulate the muscle.

2. One of the primary reasons that one individual is faster in a 50-meter sprint than another is
   a. a greater number of muscle fibers of smaller diameter.
   b. more oxidative fibers in the lower limb muscles.
   c. an enhanced ability to deliver oxygen to the muscles.
   d. a greater percentage of fast-twitch fibers in the lower limb muscles.

3. Which electrolyte imbalance is least likely to impair muscle contraction because it is not required in muscle contraction?
   a. F–
   b. Na+
   c. K+
   d. Ca2+

4. Rigor mortis occurs following death because
   a. tropomyosin remains over the myosin binding sites of actin.
   b. myosin heads attach to actin and are not released due to lack of ATP.
   c. the myosin becomes misshapen.
   d. all of the Ca2+ remains within the sarcoplasmic reticulum.

5. An athlete participates in aerobic exercise three times a week. One of the changes is an increased ability to deliver oxygen to her skeletal muscles. Over time she notices that she can continue the exercise with greater intensity and duration. The reason for this change is that there is a(n)
   a. greater response from phosphate transfer.
   b. greater production of ATP from glycolysis and less from aerobic cellular respiration.
   c. greater production of ATP from aerobic cellular respiration.
   d. increased production of lactate.

Can You Synthesize What You’ve Learned?

1. Your anatomy and physiology course is required for a career in forensics, and one of the short essays is an explanation of why the body becomes stiff after death. Provide an answer for an individual who has some understanding of skeletal muscle physiology.

2. Describe the effect of the botulinum toxin, which inhibits the release of acetylcholine at the neuromuscular junction. Would the poison curare, which competes for acetylcholine receptors (by attaching to the acetylcholine receptors and preventing acetylcholine from binding) have a similar effect? Explain.

3. Smooth muscle is within the urinary bladder wall. Explain why, if you initially have the sensation of having to urinate, the sensation sometimes passes. Base your answer on the stress-relaxation response.
The partitioning of the skeletal system into axial and appendicular divisions provides a useful guideline for subdividing the muscular system. **Axial muscles** have both their attachments on parts of the axial skeleton. Axial muscles support and move the head and vertebral column, function in nonverbal communication by affecting facial features, move the mandible during chewing, assist in food processing and swallowing, aid in breathing, and both support and protect the abdominal and pelvic organs. The **appendicular muscles** control the movements of the upper and lower limbs, and stabilize and control the movements of the pectoral and pelvic girdles. These muscles are organized into groups based upon their location in the body or the part of the skeleton they move. Some muscles of both divisions are shown in figure 11.1.

The muscles in this chapter have been organized into groups according to their location in the body. For each group, tables provide descriptions of the muscles as well as information about their action, attachments, and innervation. (Note: The word *innervation* refers to the nerve(s) that control(s) a muscle and stimulate(s) it to contract. For further information about the nerves listed in the tables, see sections 13.9 and 14.5.)
Figure 11.1 Body Musculature. (a) Anterior view shows superficial muscles on the right side of the body and some deeper muscles on the left side. (b) Posterior view shows superficial muscles on the left side of the body and some deeper muscles on the right side. Labels for the axial muscles are in bold; not all muscles shown in the figure are identified.
11.1 Skeletal Muscle Composition and Actions

We examined the macroscopic and microscopic anatomy of skeletal muscle in section 10.2. Here we discuss the attachments of a skeletal muscle, the organizational patterns exhibited by skeletal muscle fibers, and the general actions of skeletal muscles.

11.1a Skeletal Muscle Attachments

**LEARNING OBJECTIVE**

1. Compare and contrast the superior (or proximal) and inferior (or distal) attachments of a skeletal muscle.

At the ends of a muscle, the three connective tissue layers (epimysium, perimysium, and endomysium) merge to form a fibrous **tendon**, which attaches the muscle to bone, skin, or another muscle. Tendons usually have a thick, cordlike structure. Sometimes, the tendon forms a thin, flattened sheet, termed an **aponeurosis** (ap″-nō′-ro″-sis; apo = from, neuron = sinew).

Most skeletal muscles extend between bones and cross at least one mobile joint. Upon contraction, one of the bones moves while the other bone usually remains fixed. Previously, the less movable attachment was called the **origin** and the more movable attachment was called the **insertion**. However, the origin and insertion of muscles are not always easily determined by either movement or position, and some anatomists and clinicians are no longer using this terminology. Thus, we typically will use the terms **superior attachment** and **inferior attachment** when discussing most axial muscles and **proximal attachment** and **distal attachment** when discussing the muscles moving the appendicular skeleton. For muscles moving the axial skeleton, the superior attachment often (but not always) is more movable than the inferior attachment; thus, the superior portion of the body is pulled toward the inferior portion when the muscles contract, as in flexing your neck or doing an abdominal crunch. In muscles moving the appendicular skeleton, the distal attachment typically is more movable than the proximal attachment, and when muscles contract, the distal attachment moves toward the proximal attachment, as shown in **figure 11.2**. The biceps brachii muscle’s proximal attachment is the scapula, and the distal attachment is the radius. As the biceps brachi contracts, the radius is pulled toward the scapula, causing the arm to flex at the elbow.

There are some exceptions where there is no clearly defined superior or inferior attachment of an axial muscle. With those exceptions, slightly alternative terminology will be used and noted in the appropriate tables.

**WHAT DID YOU LEARN?**

1. What is the difference between the proximal and distal attachment of a skeletal muscle?

11.1b Organizational Patterns of Skeletal Muscle Fibers

**LEARNING OBJECTIVE**

2. Describe and differentiate between the organizational patterns in muscle fascicles.

As mentioned in section 10.2a, bundles of muscle fibers, termed **fascicles**, lie parallel to each other within each muscle. However, the organization of fascicles in different muscles often varies. There are four different patterns of fascicle arrangement: circular, parallel, convergent, and pennate (figure 11.3).

**Circular Muscles**

A **circular muscle** has concentrically arranged muscle fascicles around an opening or recess. A circular muscle is also called a **sphincter**, and

**Figure 11.2 Muscle Attachments.** For appendicular muscles, the proximal attachment is less movable than the distal attachment, as shown in this view of the biceps brachii muscle.

**Figure 11.3 Organization of Muscle Fibers.** Muscle fascicles may be organized into one of four basic patterns: circular, parallel, convergent, or pennate.
its contraction decreases the passageway diameter. An example is the orbicularis oris muscle that encircles the opening of the mouth.

Parallel Muscles

The fascicles in a parallel muscle run parallel to its long axis. Sometimes these muscles are cylindrical with an expanded central region. In this case, parallel muscles have a central body, called the belly, or gaster. When this muscle contracts and shortens, the muscle increases in diameter. Parallel muscles have high endurance but are not strong. Examples of parallel muscle include the rectus abdominis (an anterior abdominal muscle) and the biceps brachii of the arm.

Convergent Muscles

A convergent (kon-ver’jent) muscle has widespread muscle fascicles over a broad area that converge on a common attachment site. This attachment site may be a single tendon, a tendinous sheet, or a slender band of collagen fibers known as a raphe (rā’fē; rhaphe = seam). These muscle fibers are often triangular in shape, resembling a broad fan with a tendon at the tip. A convergent muscle is versatile—that is, the direction of its pull can be modified merely by activating a specific, single group of muscle fibers at any one time. However, when the fibers in a convergent muscle all contract at once, they do not pull as hard on the tendon as a parallel muscle of the same size because the muscle fibers on opposite sides of the tendon are not working together; rather, they are pulling in different directions. An example of a convergent muscle is the pectoralis major of the chest.

Pennate Muscles

Pennate (pen’āt; penna = feather) muscles are so named because the fascicles exhibit the same angle with respect to their tendon—that is, they resemble a large feather. Pennate muscles have one or more tendons extending through their body, and the fascicles are arranged at an oblique angle to the tendon. Because pennate muscle fibers pull at an angle to the tendon, this type of muscle does not move its tendons as far as parallel muscles move their tendons. However, the contraction of a pennate muscle generates more tension than does a parallel muscle of the same size, and thus it exerts a greater force.

There are three types of pennate muscles:

- In a unipennate muscle, all of the muscle fascicles are on the same side of the tendon. The extensor digitorum, a long muscle that extends the fingers, is a unipennate muscle.
- A bipennate muscle, the most common type, has muscle fascicles on both sides of the tendon. The interosseous muscles on both the palmar and dorsal sides of the metacarpals are composed of bipennate muscles that help adduct and abduct the digits.
- A multipennate muscle has branches of the tendon within the muscle and fascicles arranged on both sides of each tendon branch. The triangular deltoid that covers the superior surface of the shoulder joint is a multipennate muscle.

**WHAT DID YOU LEARN?**

2. Which muscle is stronger—a pennate muscle or a parallel muscle?

### 11.1c Actions of Skeletal Muscles

**LEARNING OBJECTIVE**

3. Differentiate between agonists, antagonists, and synergists.

Skeletal muscles generally do not work in isolation; rather, they work together to produce movements. Muscles are grouped according to their primary actions into three types: agonists, antagonists, and synergists.

An agonist (ag’on-ist; agon = a contest), also called a prime mover, is a muscle that contracts to produce a particular movement, such as extending the forearm. The triceps brachii on the posterior side of the humerus is an agonist when it causes extension of the forearm.

An antagonist (an-tag’ō-nist; anti = against) is a muscle whose actions oppose those of the agonist. For example, if the agonist produces extension, the antagonist produces flexion. The contraction of the agonist lengthens the antagonist, and vice versa. As this movement occurs, the lengthened muscle usually does not relax completely. Instead, the tension within the muscle being lengthened is adjusted to control the speed of the movement and ensure that it is smooth. When the triceps brachii acts as an agonist to extend the forearm, the biceps brachii muscle on the anterior side of the humerus acts as an antagonist to stabilize the movement and produce the opposing action, which is flexion of the forearm.

A synergist (sin’er-jist; syn = with, ergo = work) is a muscle that assists the agonist in performing its action. Usually, synergists are most useful at the start of a movement when the agonist is lengthened and cannot exert much power. The biceps brachii of the arm is a synergist to the brachialis muscle of the arm (the agonist) when both work together to flex the elbow joint. Synergists may also assist an agonist by preventing movement at a joint and thereby stabilizing the agonist. In this case, these synergistic muscles are called fixators.

**WHAT DID YOU LEARN?**

3. What is the difference between an agonist and a synergist?
11.2 Skeletal Muscle Naming

**LEARNING OBJECTIVES**

4. List the seven characteristics of muscles that may contribute to their names.
5. Give examples of muscles whose names contain an indication of action, specific body region, attachments, orientation of muscle fibers, shape, size, and muscle heads.

In section 1.5, you learned some of the anatomic terminology used to describe the body; in chapter 8, you saw how anatomic terms are applied to the bones of the skeleton. Naming of skeletal muscles follows similar conventions, and usually muscle names provide clues to their identification. As figure 11.4 shows, skeletal muscles are named according to the following criteria:

- **Muscle action.** Names that indicate the primary function or movement of the muscle include *flexor, extensor*, and *pronator*. These are such common actions that the names almost always contain other clues to the appearance or location of the muscle. For example, the flexor digitorum longus muscle is a long muscle responsible for flexing the digits.

- **Specific body regions.** The rectus femoris is on the thigh (*femur*), and the tibialis anterior is on the *anterior* surface of the *tibia*. Muscles that are close to the body surface often are termed *superficialis* (si′per-fish-ē-ā′lis) or *externus* (eks-ter′nus). In contrast, deeper placed muscles may have names such as *profundus* (prō-fun′dus; deep) or *internus*.

- **Muscle attachments.** Many muscle names identify their prominent attachments. For example, the sternocleidomastoid attaches to the sternum, clavicule, and mastoid process of the temporal bone.

- **Orientation of muscle fibers.** The rectus abdominis muscle is named for its lengthwise-running muscle fibers; *rectus* means “straight.” Similarly, names such as *oblique* or *obliquus* (ob-It′kwōs) indicate muscles with fibers extending at an angle to the longitudinal axis of the body. The internal and external oblique muscles are abdominal muscles that have angled muscle fibers.

- **Muscle shape.** Examples of shape in muscle names include *deltoid* (shaped like a triangle), *orbicularis* (circular muscle fibers), *rhomboid* (shaped like a rhombus), and *trapezius* (shaped like a trapezoid). Short muscles may be called *brevis* (brev′is); long muscles are called *longus* (lon′gūs) or *longissimus* (lon-jis′tús-mūs; longest). *Teres* (ter′ēz) muscles are both long and round.

- **Muscle size.** Large muscles may be called *magnus* (big), *major* (bigger), or *maximus* (biggest). Small muscles may be called *minor* (smaller) or *minimus* (smallest).

Examples of muscles named by size include the buttocks muscles: *gluteus maximus, gluteus medius*, and *gluteus minimus*.

- **Number of muscle heads at an attachment site.** Some muscles are named after how many muscle bellies or heads each contains at the superior or proximal attachment sites. A *biceps* muscle has two heads at its proximal attachments, a *triceps* muscle has three heads, and a *quadriceps* muscle has four heads.

**WHAT DID YOU LEARN?**

4. What are some words used in muscle names that refer to muscle shape?
5. The gluteus maximus muscle gets its name from which categories for naming muscles?
11.3 Muscles of the Head and Neck

The muscles of the head and neck are separated into several specific groups. Almost all of these muscles (except for a few muscles of the anterior neck) attach to either the bones of the skull or the hyoid bone.

11.3a Muscles of Facial Expression

**LEARNING OBJECTIVES**

6. Name the muscles that move the forehead, the skin around the eyes, and the nose, and describe their actions.

7. List the muscles that move the mouth and cheeks and their actions.

The muscles of facial expression attach from the superficial fascia or skull bones to the superficial fascia of the skin (figure 11.5). When these muscles contract, they pull on the skin, causing it to move. Most of these muscles are innervated by the seventh cranial nerve (CN VII), termed the facial nerve (see section 13.9).

The epicranius is composed of the occipitofrontalis muscle and a broad epicranial aponeurosis, also called the galea aponeurotica. The frontal belly of the occipitofrontalis is superficial to the frontal bone on the forehead. When this muscle contracts, it raises the eyebrows and wrinkles the skin of the forehead. The occipital belly of the occipitofrontalis covers the posterior aspect of the skull. When this muscle contracts, it retracts the scalp slightly.

Deep to the frontal belly of the occipitofrontalis is the corrugator supercilii. This muscle draws the eyebrows together and forms vertical wrinkle lines around the nose. The orbicularis oculi consists of circular muscle fibers that surround the eye’s orbit. When this muscle contracts, the eyelid closes, as when you wink, blink, or squint. The levator palpebrae superioris elevates the upper eyelid when you open your eyes.

Several muscles of facial expression are associated with the nose. The nasalis elevates the corners of the nostrils. When you “flare” your nostrils, you are using the nasalis muscles. If you wrinkle your nose in distaste after smelling a foul odor, you have used your procerus muscle. This muscle is continuous with the frontal belly of the occipitofrontalis muscle and runs over the bridge of the nose, where it produces transverse wrinkles when it contracts.

The mouth is the most expressive part of the face. The orbicularis oris consists of muscle fibers that encircle the opening of the mouth. When this muscle contracts, you close your mouth. In addition, when you “pucker up” for a kiss, you are using this muscle. The depressor labii inferioris does what its name suggests—it pulls the lower lip inferiorly. The depressor anguli oris is considered the “frown” muscle, because it pulls the corners of the mouth inferiorly. (Note, however, that it takes more muscles than just this one to produce a frown.)

In contrast, some muscles of the mouth elevate part or all of the upper lips. The levator labii superioris pulls the upper lip superiorly, as if a person were sneering or snarling. The levator anguli oris pulls the corners of the mouth superiorly and laterally. The zygomaticus major and zygomaticus minor work with the levator anguli oris.
Figure 11.5 Muscles of Facial Expression.
muscles. You use all of these muscles when you smile. The **risorius** pulls the corner of the lips laterally; you use this muscle if you make a closed-mouth smile.

The **mentalis** attaches to the lower lip, and when it contracts, it protrudes the lower lip (as when a person “pouts”). The **platysma** tenses the skin of the neck and pulls the lower lip inferiorly. If you stand in front of a mirror and tense the skin of your neck, you can see these thin muscles bulging out.

The **buccinator** compresses the cheek against the teeth when we chew (and is the reason our cheeks don’t bulge out like a squirrel’s when we eat). Infants use the buccinator when they suckle at the breast. Some trumpet players (such as Dizzy Gillespie) have stretched out their buccinator muscles, allowing their cheeks to be “puffy” with air when they play the trumpet.

**Table 11.1** summarizes the attachments and movements of the muscles of facial expression. **Figure 11.6** illustrates how these muscles produce some of the more characteristic expressions.

### Table 11.1

**Muscles of Facial Expression**

<table>
<thead>
<tr>
<th>Region/Muscle</th>
<th>Action(s)</th>
<th>Bony Attachment(s) (B)/Soft Tissue Attachment(s) (S)¹</th>
<th>Innervation (see section 13.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SCALP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epicranus (ep‘t-kra’-nē-us; epi = over, cran = skull): Composed of an epicranial aponeurosis and the two bellies of the occipitofrontalis muscle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal belly of occipitofrontalis (ok-sip‘i-tō-fron-tā’lis)</td>
<td>Moves scalp, eyebrows; wrinkles skin of forehead</td>
<td>B: Epicranial aponeurosis&lt;br&gt;S: Skin and subcutaneous layer of forehead and eyebrows</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Occipital belly of occipitofrontalis (occipito = base of skull)</td>
<td>Retracts scalp</td>
<td>B: Superior nuchal line&lt;br&gt;S: Epicranial aponeurosis</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td><strong>Nose</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nasalis (nā’zā-lis)</td>
<td>Compresses bridge and depresses tip of nose; elevates corners of nostrils</td>
<td>B: Maxillae and alar cartilage of nose&lt;br&gt;S: Dorsum of nose</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Procerus (prō-sē’rūs) (procerus = long)</td>
<td>Moves and wrinkles nose</td>
<td>B: Nasal bone and lateral nasal cartilage&lt;br&gt;S: Aponeurosis at bridge of nose and skin of forehead</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td><strong>Mouth</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buccinator (buk’sī-nā’tōr) (bucco = cheek)</td>
<td>Compresses cheek, holds food between teeth during chewing</td>
<td>B: Alveolar processes of mandible and maxillae&lt;br&gt;S: Orbicularis oris muscle</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Depressor anguli oris (dī-pres’ōr ăng’gū-lē-or-is) (depressor = depresses; angul = angle; oris = mouth)</td>
<td>Draws corners of mouth inferiorly and laterally (“frown” muscle)</td>
<td>B: Body of mandible&lt;br&gt;S: Skin at inferior corner (angle) of mouth</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Depressor labii inferioris (lā’bē-lī-in-frē’ō-or-is) (labi = lip; infer = below)</td>
<td>Draws lower lip inferiorly</td>
<td>B: Body of mandible lateral to midline&lt;br&gt;S: Skin at inferior lip</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Levator anguli oris (le-vā’tor, le-vā’er) (levi = raise)</td>
<td>Draws corners of mouth superiorly and laterally (“smile” muscle)</td>
<td>B: Lateral maxilla&lt;br&gt;S: Skin at superior corner (angle) of mouth</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Levator labii superioris (śū-pē’ō-or-is) (śū = open, pēr = edge)</td>
<td>Opens lips; raises and furrows the upper lip (“Elvis” lip snarl)</td>
<td>B: Zygomatic bone; maxilla&lt;br&gt;S: Skin and muscle of superior lip</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Mentalis (men-tā’lis) (ment = chin)</td>
<td>Protrudes lower lip (“pout”); wrinkles chin</td>
<td>B: Central mandible&lt;br&gt;S: Skin of chin</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td>Orbicularis oris (ōr-bik’yō-lā’tris) (orb = circular)</td>
<td>Compresses and purses lips (“kiss” muscle)</td>
<td>B: Maxilla and mandible; blend with fibers from other facial muscles&lt;br&gt;S: Encircling mouth; skin and muscles at angles to mouth</td>
<td>CN VII (facial nerve)</td>
</tr>
</tbody>
</table>

1. The muscles of facial expression arise on bone, and their more movable attachment typically is on muscle, tendon, subcutaneous tissue, or integument. Thus, it is more appropriate to refer to bony attachment and soft tissue attachment for these muscles.
### Table 11.1

<table>
<thead>
<tr>
<th>Region/Muscle</th>
<th>Action(s)</th>
<th>Bony Attachment(s) (B)/Soft Tissue Attachment(s) (S)</th>
<th>Innervation (see section 13.9)</th>
</tr>
</thead>
</table>
| **Risorius**  
(ri-sör′ē-ōs)  
*risor* = laughter | Draws corner of lip laterally; tenses lips | B: Deep fascia associated with masseter muscle  
S: Skin at angle of mouth | CN VII (facial nerve) |
| **Zygomaticus major**  
(zy̞-gō-mat′i-kus)  
*zygomatic* = cheekbone  
*major* = greater | Elevates corner of mouth  
(“smile” muscle) | B: Zygomatic bone  
S: Skin at superolateral edge of mouth | CN VII (facial nerve) |
| **Zygomaticus minor**  
*minor* = lesser | Elevates corner of mouth  
(“smile” muscle) | B: Zygomatic bone  
S: Skin of superior lip | CN VII (facial nerve) |
| **Corrugator supercilli**  
(kər′ū-gā′-ter su̞′per′silˈō-)  
*corrugo* = to wrinkle  
*cilium* = eyelid | Pulls eyebrows inferiorly and medially; forms vertical wrinkles above nose | B: Medial end of superciliary arch  
S: Skin superior to supraorbital margin and superciliary arch | CN VII (facial nerve) |
| **Levator palpebrae superioris**  
(pal-pē′brā)  
*palpebra* = eyelid | Elevates superior eyelid | B: Lesser wing of sphenoid bone  
S: Superior tarsal plate and skin of superior eyelid | CN III (oculomotor nerve) |
| **Orbicularis oculi**  
(ok′yū-lī)  
*ocul* = eye | Closes eye; produces winking, blinking, squinting (“blink” muscle) | B: Medial wall or margin of the orbit  
S: Skin surrounding eyelids | CN VII (facial nerve) |
| **Platysma**  
(plă-tiz′mă)  
*platy* = flat | Pulls lower lip inferiorly; tenses skin of neck | B: Fascia of deltoid and pectoralis major muscles and acromion of scapula  
S: Skin of cheek and mandible | CN VII (facial nerve) |

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2. *Platysma* is an exception to the “bony attachment” rule. Its traditional “origin” arises primarily from muscle.
11.3b Extrinsic Eye Muscles

**LEARNING OBJECTIVES**

8. Become familiar with the six extrinsic muscles of the eye, and describe how each affects eye movement.

9. Name the three cranial nerves that innervate the extrinsic eye muscles, and identify which muscles they act upon.

The extrinsic eye muscles, often called extraocular muscles, move the eyes. They are termed extrinsic because they attach to the outer white surface of the eye, called the sclera (see section 16.4b). There are six extrinsic eye muscles: the four rectus muscles (medial, lateral, inferior, and superior) and the two oblique muscles (inferior and superior) (figure 11.7).

The four rectus eye muscles arise from a common tendinous ring in the orbit. These muscles attach to the anterior part of the eye and are named according to which part of the eye they are located (medial, lateral, inferior, or superior).

The medial rectus attaches to the anteromedial surface of the eye and pulls the eye medially (adducts the eye). It is innervated by CN III (oculomotor nerve). The lateral rectus attaches to the
The eye inferiorly (but attaches to the inferior sclera of the eye. The axis of the eye; that is why both muscles also move the eye slightly in the medial direction.

11.7

Look at your nose). The inferior and superior rectus muscles are innervated by CN III. Figure illustrates that the superior and inferior rectus muscles do not pull directly parallel to the long axis of the eye; that is why both muscles also move the eye slightly in the medial direction.

The oblique eye muscles arise from within the orbit and attach to the posterolateral part of the sclera of the eye. The inferior oblique elevates the eye and turns the eye laterally. This muscle passes through a pulleylike loop, called the trochlea, in the anteromedial orbit. This muscle attaches to the superior posterior part of the eye, so contracting this muscle pulls the posterior part of the eye superiorly (but depresses the anterior surface of the eye). This muscle is innervated by CN IV (trochlear). (Notice that this nerve’s name is derived from the trochlea that holds the superior oblique in place.)

Table 11.2 compares the extrinsic muscles of the eye. Refer to section 13.9 to review the cranial nerves mentioned in this section.

### WHAT DID YOU LEARN? Which extrinsic eye muscles abduct the eye (move the eye laterally)?

### CONCEPT CONNECTION

Vision problems may be due to musculoskeletal issues, nervous system issues, or both. For example, if a person cannot abduct his right eye, that indicates that CN VI (abducens nerve) may be injured (see section 13.9). In addition, some vision problems may be due to a single weak extrinsic muscle, and correcting this muscle imbalance (with either exercises or wearing a patch over the eye with the stronger extrinsic muscles) helps alleviate the vision problem. Thus, a physician must integrate both the innervation and muscle information to properly diagnose a patient’s visual disturbance.

### CLINICAL VIEW 11.3

**Strabismus and Diplopia**

The extrinsic eye muscles move the left and right eyes in unison, so both eyes focus on the same image. *Strabismus* (stră-biz’mus; strabismos = a squinting) is a condition where both eyes cannot focus on the same image, and the gaze of one eye is displaced. Strabismus may be caused by cranial nerve injury (see section 13.9) or weak eye muscles of one eye, or it may occur when the brain favors vision in the stronger eye (which is the cause of lazy eye, where the weaker eye doesn’t track properly and may favor one position). Individuals with strabismus may experience *diplopia* (dip’lō’pe’a; diplos = double, ops = eye), or *double vision*.

### Table 11.2 Extrinsic Eye Muscles

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Orbit Attachment (O)/Eyeball Attachment (E)</th>
<th>Innervation (see section 13.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RECTUS MUSCLES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medial rectus (mē’dē-āl rek’tus) rectus = straight</td>
<td>Moves eye medially (adducts eye)</td>
<td>O: Common tendinous ring E: Anteromedial surface of eye</td>
<td>CN III (oculomotor nerve)</td>
</tr>
<tr>
<td>Lateral rectus (lat’er-āl)</td>
<td>Moves eye laterally (abducts eye)</td>
<td>O: Common tendinous ring E: Anterolateral surface of eye</td>
<td>CN VI (abducens nerve)</td>
</tr>
<tr>
<td>Inferior rectus (in tér-ō)</td>
<td>Moves eye inferiorly (depresses eye) and medially (adducts eye)</td>
<td>O: Common tendinous ring E: Anteroinferior surface of eye</td>
<td>CN III (oculomotor nerve)</td>
</tr>
<tr>
<td>Superior rectus (sē-prō)</td>
<td>Moves eye superiorly (elevates eye) and medially (adducts eye)</td>
<td>O: Common tendinous ring E: Anterosuperior surface of eye</td>
<td>CN III (oculomotor nerve)</td>
</tr>
<tr>
<td><strong>OBLIQUE MUSCLES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior oblique (in tér-ō) obliquus = slanting</td>
<td>Moves eye superiorly (elevates eye) and laterally (abducts eye)</td>
<td>O: Anterior orbital surface of maxilla E: Posterosuperior, lateral surface of eye</td>
<td>CN III (oculomotor nerve)</td>
</tr>
<tr>
<td>Superior oblique (sē-prō) obliquus = slanting</td>
<td>Moves eye inferiorly (depresses eye) and laterally (abducts eye)</td>
<td>O: Sphenoid bone E: Posterosuperior, lateral surface of eye</td>
<td>CN IV (trochlear nerve)</td>
</tr>
</tbody>
</table>

1. The less movable attachment for an eye muscle is on the bony orbit or tendinous ring, whereas the more movable attachment is on the eyeball.
The **temporalis** (or temporal muscle) is a broad, fan-shaped muscle that extends from the temporal lines of the skull and attaches to the coronoid process of the mandible. It elevates and retracts (pulls posteriorly) the mandible. You can palpate the temporalis by placing your fingers along your temple (lateral skull at same level of orbits) as you open and close your jaw. The muscle you feel contracting is the temporalis.

The **masseter** elevates and protracts (pulls anteriorly) the mandible. It is the most powerful and important of the masticatory muscles. This short, thick muscle is superficial to the temporalis. You can feel the movement of the masseter by palpating near the angle of the mandible as you open and close your mouth.

The **lateral and medial pterygoid** muscles arise from the pterygoid processes of the sphenoid bone and attach to the mandible. Both pterygoids protract the mandible and move it from side to side during chewing. These movements maximize the effectiveness of the teeth while chewing or grinding foods of various consistencies. The medial pterygoid also elevates the mandible.

**Table 11.3** summarizes the characteristics of the muscles of mastication.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action(s)</th>
<th>Superior Attachment(s) (S)/Inferior Attachment(s) (I)¹</th>
<th>Innervation (see section 13.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Temporalis</strong></td>
<td>Elevates and retracts mandible</td>
<td>S: Superior and inferior temporal lines I: Coronoid process of mandible</td>
<td>CN V₃ (trigeminal nerve, mandibular division)</td>
</tr>
<tr>
<td>(temp-po-r′-lish)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tempora = pertaining to</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>temporal bone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Masseter</strong></td>
<td>Elevates and protracts mandible; agonist (prime mover) of mandible elevation</td>
<td>S: Zygomatic arch I: Ramus (lateral surface) and angle of mandible</td>
<td>CN V₃ (trigeminal nerve, mandibular division)</td>
</tr>
<tr>
<td>(mas′-tēr)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maseter = chewer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Medial pterygoid</strong></td>
<td>Elevates and protracts mandible; produces side-to-side movement of mandible</td>
<td>S: Maxilla, palatine, and medial surface of lateral pterygoid plate I: Medial surface of mandibular ramus</td>
<td>CN V₃ (trigeminal nerve, mandibular division)</td>
</tr>
<tr>
<td>(ter′-i-goyd)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pterygoid = winglike</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Lateral pterygoid</strong></td>
<td>Protracts mandible; produces side-to-side movement of mandible</td>
<td>S: Greater wing of sphenoid and lateral surface of lateral pterygoid plate I: Condylar process of mandible</td>
<td>CN V₃ (trigeminal nerve, mandibular division)</td>
</tr>
</tbody>
</table>

¹. The superior attachments of the mastication muscles are less movable than the inferior attachments.
Muscles That Move the Tongue

The tongue is an agile, highly mobile organ. It is composed of intrinsic muscles that compose the tongue, which curl, squeeze, and fold the tongue during chewing and speaking.

The extrinsic muscles of the tongue arise from other head and neck structures and attach to the tongue. The extrinsic muscles end in the suffix -glossus, meaning tongue (figure 11.9). These extrinsic tongue muscles are used in various combinations to accomplish the precise, complex, and delicate tongue movements required for proper speech and food manipulation within the mouth. Most of these muscles are innervated by CN XII, the hypoglossal nerve (see section 13.9).

Table 11.4 summarizes the characteristics of the muscles that move the tongue.

Pharynx Muscles

The pharynx (far‘ingks), commonly known as the throat, is a funnel-shaped tube that lies posterior to both the oral and nasal cavities (see figure 26.4). Several muscles help form or attach to this tube and aid in swallowing (figure 11.10). Most pharyngeal muscles are innervated by CN X (vagus nerve).

The primary pharynx muscles are the pharyngeal constrictors (superior, middle, and inferior). When food is swallowed and enters the pharynx, these muscles contract sequentially to initiate swallowing and force the bolus inferiorly into the esophagus (see section 26.2c). Other pharyngeal muscles help elevate or tense the palate when swallowing. These muscles are summarized in table 11.5.

WHAT DID YOU LEARN?

9. What movements do the medial and lateral pterygoid muscles perform?
10. What is the general action of the extrinsic muscles of the tongue?

11.3d Muscles of the Anterior Neck:
The Hyoid Muscles

LEARNING OBJECTIVE

13. Contrast the actions of the four suprahyoid muscles and the four infrahyoid muscles.

Table 11.4  Muscles That Move the Tongue

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action(s)</th>
<th>Head or Neck Attachment(s) (H)/Tongue Attachment(s) (T)¹</th>
<th>Innervation (see section 13.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genioglossus</td>
<td>Protracts tongue</td>
<td>H: Mental spines of mandible</td>
<td>CN XII (hypoglossal nerve)</td>
</tr>
<tr>
<td>(jē′nē-o-glos′ūs)</td>
<td>geni = chin</td>
<td>T: Inferior region of tongue; hyoid bone</td>
<td></td>
</tr>
<tr>
<td>glossus = tongue</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stylloglossus</td>
<td>Elevates and retracts tongue</td>
<td>H: Styloid process of temporal bone</td>
<td>CN XII (hypoglossal nerve)</td>
</tr>
<tr>
<td>(strī′lo-glos′ūs)</td>
<td>stylo = pertaining to styloid process of temporal bone</td>
<td>T: Side and inferior aspect of tongue</td>
<td></td>
</tr>
<tr>
<td>Hyoglossus</td>
<td>Depresses and retracts tongue</td>
<td>H: Hyoid bone</td>
<td>CN XII (hypoglossal nerve)</td>
</tr>
<tr>
<td>(hī′o-glos′ūs)</td>
<td>hyo = pertaining to hyoid bone</td>
<td>T: Inferolateral side of tongue</td>
<td></td>
</tr>
<tr>
<td>Palatoglossus</td>
<td>Elevates posterior part of tongue</td>
<td>H: Anterior surface of soft palate</td>
<td>CN X (vagus nerve) via pharyngeal plexus of nerves</td>
</tr>
<tr>
<td>(pal-ā-tō-glos′ūs)</td>
<td>palato = palate</td>
<td>T: Side and posterior aspect of tongue</td>
<td></td>
</tr>
</tbody>
</table>

¹ The tongue attachments are the more movable parts of these muscles.
**Figure 11.10 Pharyngeal Constrictors, Palate Muscles, and Laryngeal Elevators.** A right lateral view reveals some of the muscles that constrict the pharynx when swallowing, move the palate, and elevate the larynx (palatopharyngeus and salpingopharyngeus not shown).

**Table 11.5**  
Muscules of the Pharynx

<table>
<thead>
<tr>
<th>Region/Muscle</th>
<th>Action(s)</th>
<th>Origin/Insertion</th>
<th>Innervation (see section 13.9)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PALATE MUSCLES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Levator veli palatini      | Elevates soft palate when swallowing                                       | O: Petrous part of temporal bone  
I: Soft palate                                         | CN X (vagus nerve)                                     |
| (vel’t pal’ā-te’nī) velum = veil |                                                                            |                                                       |                                                     |
| Tensor veli palatini       | Tenses soft palate and opens auditory tube when swallowing or yawning      | O: Sphenoid bone; region around auditory tube  
I: Soft palate                                           | CN V3 (trigeminal nerve, mandibular division)          |
| (ten’sor) tensus = to stretch |                                                                            |                                                       |                                                     |
| **PHARYNGEAL CONSTRICTORS**|                                                                            |                                                       |                                                     |
| Superior constrictor       | Constricts pharynx in sequence to force bolus into esophagus; superior is innermost | O: Pterygoid process of sphenoid bone; medial surface of mandible  
I: Posterior median raphe (muscle fiber union from both sides) | CN X (vagus nerve) via branches of pharyngeal plexus |
| (kon-strīk’ter, -tôr) constringo = to draw together |                                                                            |                                                       |                                                     |
| Middle constrictor         | Constricts pharynx in sequence                                             | O: Hyoid bone  
I: Posterior median raphe                                | CN X (vagus nerve) via branches of pharyngeal plexus |
| Inferior constrictor       | Constricts pharynx in sequence; inferior is outermost                      | O: Thyroid and cricoid cartilage  
I: Posterior median raphe                                  | CN X (vagus nerve) via branches of pharyngeal plexus |
| **LARYNGEAL (VOICE BOX) ELEVATORS** |                                                                            |                                                       |                                                     |
| Palatopharyngeus           | Elevates pharynx and larynx                                               | O: Soft palate  
I: Side of pharynx and thyroid cartilage of larynx      | CN X (vagus nerve) via branches of pharyngeal plexus |
| (pal’a-tō-fā-rin’je-ūs) pharynx = pharynx |                                                                            |                                                       |                                                     |
| Salpingopharyngeus         | Elevates pharynx and larynx                                               | O: Auditory tube  
I: Blends with palatopharyngeus on lateral wall of pharynx | CN X (vagus nerve) via branches of pharyngeal plexus |
| (sal-ping’gō-fā-rin’je-ūs) salpinx = trumpet |                                                                            |                                                       |                                                     |
| Stylopharyngeus            | Elevates pharynx and larynx                                               | O: Styloid process of temporal bone  
I: Side of pharynx and thyroid cartilage of larynx       | CN IX (glossopharyngeal nerve) via branches of pharyngeal plexus |
| (stī’lō-fā-rin’je-ūs) |                                                                            |                                                       |                                                     |

1. Only the pharyngeal constrictors are discussed in the text.
2. Here, we use the terms origin and insertion because the terms superior attachment and inferior attachment are not applicable for all of the muscles in this table.
The muscles of the anterior neck are divided into the **suprahyoid muscles**, which are superior to the hyoid bone, and the **infrahyoid muscles**, which are inferior to the hyoid bone (figure 11.11).

The suprahyoid muscles are associated with the floor of the mouth. In general, these muscles act as a group to elevate the hyoid bone during swallowing or speaking. Some of these muscles perform additional functions: The **digastric** has two bellies, anterior and posterior. The anterior belly extends from the mental protuberance of the mandible to the hyoid bone, and the posterior belly continues from the hyoid bone to the mastoid part of the temporal bone. The two bellies are united by an intermediate tendon that is held in position by a fibrous loop. In addition to elevating the hyoid bone, the digastric muscle can also depress the mandible. The **geniohyoid** attaches to the mental spines of the mandible and the hyoid bone. This muscle elevates the hyoid bone. The broad, flat **mylohyoid** attaches to the mylohyoid line of the mandible and the hyoid bone, and it forms the muscular floor to the mouth. When this muscle contracts, it both elevates the hyoid bone and raises the floor of the mouth. The muscle fibers of the left and right mylohyoid are aligned in a V shape. The **stylohyoid** connects the styloid process of the skull and the hyoid bone. Upon contraction, it elevates the hyoid bone, causing the floor of the oral cavity to elongate during swallowing.

As swallowing is completed, the infrahyoid muscles contract to influence the position of the hyoid bone and the larynx (see section 23.3a). In general, these muscles either depress the hyoid bone or depress the thyroid cartilage of the larynx. The **omohyoid** contains two thin muscle bellies anchored in place by a fascia “sling.” This muscle is lateral to the sternohyoid and extends from the superior border of the scapula to the hyoid, where it depresses the hyoid bone. The **sternohyoid** extends from the sternum to the hyoid bone, where it depresses the hyoid bone. The **sternothyroid** is deep to the sternohyoid. It extends from the sternum to the thyroid cartilage of the larynx. It depresses the thyroid cartilage to return it to its original position after swallowing. The **thyrohyoid** extends from...
### Table 11.6 Muscles of the Anterior Neck

<table>
<thead>
<tr>
<th>Region/Muscle</th>
<th>Action(s)</th>
<th>Superior Attachment(s) (S)/Inferior Attachment(s) (I)</th>
<th>Innervation (see sections 13.9 and 14.5d)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SUPRAHYOID MUSCLES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digastric (dī-gas′trik)</td>
<td>Depresses mandible; elevates hyoid bone</td>
<td>S: Anterior belly: Mandible near mental protuberance; posterior belly, mastoid process</td>
<td>Anterior belly: CN V3 (trigeminal nerve, mandibular division)</td>
</tr>
<tr>
<td>di = two</td>
<td>I: Hyoid bone via fascia sling</td>
<td>Posterior belly: CN VII (facial nerve)</td>
<td></td>
</tr>
<tr>
<td>gaster = belly</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Geniohyoid (jē-nē-0-hī′oyd)</td>
<td>Elevates hyoid bone</td>
<td>S: Mental spines of mandible</td>
<td>First cervical spinal nerve (C1) via CN XII (hypoglossal nerve)</td>
</tr>
<tr>
<td></td>
<td>I: Hyoid bone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mylohyoid (mī-lō-hī′oyd)</td>
<td>Elevates hyoid bone; elevates floor of mouth</td>
<td>S: Mylohyoid line of mandible</td>
<td>CN V3 (trigeminal nerve, mandibular division)</td>
</tr>
<tr>
<td>myle = molar</td>
<td>I: Hyoid bone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stylohyoid (stī-lō-hī′oyd)</td>
<td>Elevates hyoid bone</td>
<td>S: Styloid process of temporal bone</td>
<td>CN VII (facial nerve)</td>
</tr>
<tr>
<td></td>
<td>I: Hyoid bone</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>INFRAHYOID MUSCLES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Omohyoid (ō-mō-hī′oyd)</td>
<td>Depresses hyoid bone; fixes hyoid during opening of mouth (depression of mandible)</td>
<td>S: Hyoid bone</td>
<td>Cervical spinal nerves C1–C3 through ansa cervicalis (from cervical plexus)</td>
</tr>
<tr>
<td>omo = shoulder</td>
<td>I: Superior border of scapula</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sternohyoid (ster′nō-hī′oyd)</td>
<td>Depresses hyoid bone</td>
<td>S: Hyoid bone</td>
<td>Cervical spinal nerves C1–C3 through ansa cervicalis (from cervical plexus)</td>
</tr>
<tr>
<td>sterno = sternum</td>
<td>I: Manubrium of sternum and medial end of clavicle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sternothyroid (ster′nō-thī′royd)</td>
<td>Depresses thyroid cartilage of larynx; fixes hyoid</td>
<td>S: Thyroid cartilage of larynx</td>
<td>Cervical spinal nerves C1–C3 through ansa cervicalis (from cervical plexus)</td>
</tr>
<tr>
<td>thyro = thyroid cartilage</td>
<td>I: Posterior surface of manubrium of sternum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thyrohyoid (thī-rō-hī′oyd)</td>
<td>Depresses hyoid bone and elevates thyroid cartilage of larynx; fixes hyoid</td>
<td>S: Hyoid bone</td>
<td>First cervical spinal nerve C1 via CN XII (hypoglossal nerve)</td>
</tr>
<tr>
<td></td>
<td>I: Thyroid cartilage of larynx</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Note that with most of the infrahyoid muscles, the superior attachment is the more movable part of each muscle, whereas the inferior attachment tends to be more movable for the suprahyoid muscles.

---

The thyroid cartilage of the larynx to the hyoid bone. It depresses the hyoid bone and elevates the thyroid cartilage to close off the larynx during swallowing. In addition, the omohyoid, sternohyoid, and thyrohyoid help anchor the hyoid so the digastric can depress the mandible.

**Table 11.6** summarizes the characteristics of these muscles.

---

**WHAT DO YOU THINK?**

Since muscles frequently are named for their attachment sites, what do you think the prefix *-omo* in omohyoid means?

**WHAT DID YOU LEARN?**

List the four suprahyoid muscles, and describe a common action for them.

---

**11.3e Muscles That Move the Head and Neck**

**LEARNING OBJECTIVE**

14. Compare and contrast the actions of the anterolateral neck muscles and the posterior neck muscles.
Muscles that move the head and neck arise from the vertebral column, the thoracic cage, and the pectoral girdle, and attach to bones of the cranium (figures 11.11 and 11.12).

**Anterolateral Neck Muscles**

The anterolateral neck muscles all flex the neck. The main muscles in this group are the sternocleidomastoid and the three scalenes.

The **sternocleidomastoid** is a thick, cordlike muscle that extends from the sternum and clavicle to the mastoid process posterior to the ear. Contraction of both sternocleidomastoid muscles (called **bilateral contraction**) flexes the neck. Contraction of just one sternocleidomastoid muscle (termed **unilateral contraction**) results in lateral flexion of the neck to its own side (as occurs when you tilt your head as if to touch your ear to your shoulder), as well as rotation of the head to the opposite side. Thus, if the left sternocleidomastoid muscle contracts, it rotates the head to the right side of the body. The three **scalene muscles** (anterior, middle, and posterior) work with the sternocleidomastoid to flex the neck. In addition, the scalene muscles elevate the first and second ribs during forced inspiration (see section 23.5b).

**Posterior Neck Muscles**

Many of the posterior neck muscles work together to extend the neck (figure 11.13). The trapezius attaches to the skull and helps extend the neck, but its primary function is to help move the pectoral girdle.

When the left and right **sphenius capitis**, **sphenius cervicis**, **semispinalis capitis**, and **longissimus capitis** muscles bilaterally contract, they extend the neck. Unilateral contraction rotates (turns) the head and neck to the same side.

A group of muscles called the **suboccipital muscles** includes the obliquus capitis superior, obliquus capitis inferior, rectus capitis posterior major, and rectus capitis posterior minor.
Figure 11.13 Posterior Neck Muscles. An illustration shows the deep and deeper muscles that extend and rotate the head and neck.

The oblique muscles rotate the head to the same side, whereas the rectus muscles extend the head and neck. **Table 11.7** summarizes the characteristics of the muscles of the head and neck.

### WHAT DID YOU LEARN?

12. Which neck muscles extend the neck? Which neck muscles flex the neck?

### 11.4 Muscles of the Vertebral Column

#### LEARNING OBJECTIVES

15. Name and describe the three groups of erector spinae muscles.

16. Describe the actions of the transversospinalis and quadratus lumborum muscles.

---

**Table 11.7** Muscles That Move the Head and Neck

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action(s)</th>
<th>Superior Attachment(s) (S)/Inferior Attachment(s) (I)</th>
<th>Innervation (see sections 13.9 and 14.5c, d)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sternocleidomastoid</strong></td>
<td>Bilateral action^2^: Flexes neck</td>
<td>S: Mastoid process</td>
<td>CN XI (accessory nerve)</td>
</tr>
<tr>
<td>(ster’nə-kli’dē-mās-toyd)</td>
<td>Unilateral action^3^: Lateral flexion of neck, rotation of head to opposite side</td>
<td>I: Manubrium of the sternum and sternal end of clavicle</td>
<td></td>
</tr>
<tr>
<td><em>sterno</em> = sternum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>cleido</em> = clavicle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>masto</em> = mastoid process</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Scalene muscles (anterior, middle, posterior)</strong> (see also table 11.9)</td>
<td>Flex neck (when 1st rib is fixed); elevate 1st and 2nd ribs during forced inspiration when neck is fixed</td>
<td>S: Transverse processes of cervical vertebrae</td>
<td>Cervical spinal nerves</td>
</tr>
<tr>
<td>(skā’lēnz)</td>
<td>I: Superior surface of 1st and 2nd ribs</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>scalene</em> = uneven</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Splenius capitis and cervicis</strong></td>
<td>Bilateral action: Extends neck</td>
<td>S: Occipital bone and mastoid process</td>
<td>Cervical spinal nerves</td>
</tr>
<tr>
<td>(splē’nē-ŭs ka-pi-tis) (ser’vi’sis)</td>
<td>Unilateral action: Rotates head to same side</td>
<td>I: Ligamentum nuchae</td>
<td></td>
</tr>
<tr>
<td><em>splenion</em> = bandage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Longissimus capitis</strong></td>
<td>Bilateral action: Extends neck</td>
<td>S: Mastoid process</td>
<td>Cervical and thoracic spinal nerves</td>
</tr>
<tr>
<td>(lon-gis’i-mās)</td>
<td>Unilateral action: Rotates head to same side</td>
<td>I: Transverse process of T₃–T₄ and articular processes of C₅–C₇ vertebrae</td>
<td></td>
</tr>
<tr>
<td><em>longissimus</em> = longest</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>caput</em> = head</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Obliquus capitis superior</strong></td>
<td>Rotates head to same side</td>
<td>S: Inferior nuchal line of occipital bone</td>
<td>Suboccipital nerve (posterior ramus of C₁ spinal nerve)</td>
</tr>
<tr>
<td>(ob-lĭ’kwās)</td>
<td></td>
<td>I: Transverse process of atlas</td>
<td></td>
</tr>
<tr>
<td><em>obliquus</em> = slanting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Obliquus capitis inferior</strong></td>
<td>Rotates head to same side</td>
<td>S: Transverse process of atlas</td>
<td>Suboccipital nerve (posterior ramus of C₁ spinal nerve)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Spinous process of axis</td>
<td></td>
</tr>
<tr>
<td><strong>Rectus capitis posterior major</strong></td>
<td>Extends neck</td>
<td>S: Inferior nuchal line of occipital bone</td>
<td>Suboccipital nerve (posterior ramus of C₁ spinal nerve)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Spinous process of atlas</td>
<td></td>
</tr>
<tr>
<td><strong>Rectus capitis posterior minor</strong></td>
<td>Extends neck</td>
<td>S: Inferior nuchal line of occipital bone</td>
<td>Suboccipital nerve (posterior ramus of C₁ spinal nerve)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Posterior tubercle of atlas</td>
<td></td>
</tr>
</tbody>
</table>

1. Here, the superior attachments are more movable than the inferior attachments.
2. **Bilateral action** means both the left and right muscles are contracting together.
3. **Unilateral action** means only one muscle (either the left or right muscle) is contracting.
CLINICAL VIEW 11.4
Congenital Muscular Torticollis

*Congenital muscular torticollis* (CMT), often known as *wryneck*, is a condition where a newborn presents with a shortened and tightened sternocleidomastoid muscle that may persist into childhood. It is thought to be a result of trauma resulting from either a difficult birth or prenatal position of the fetus. The trauma causes a hematoma and fibrosing of the muscle tissue. Pediatricians also have seen an increase in acquired muscular torticollis among newborns who are kept in infant seats for extended periods of time outside of the car. Children with CMT often tilt their heads to the affected side and their chins to the unaffected side. Because the child typically favors a particular head position when sleeping, *plagiocephaly* (flattening of the head; see Clinical View 8.2: “Craniosynostosis and Plagiocephaly”) often accompanies CMT.

CMT treatment typically involves stretching the affected muscle several times a day, changing sleeping positions, and making the child use the affected side more frequently. A newer approach to treatment of CMT is the use of botulinum toxin type A (Botox, which impairs contraction of the affected muscle), combined with stretching. In cases that do not respond to the treatments mentioned, sternocleidomastoid release surgery is recommended. The sternocleidomastoid is cut from at least one attachment point, repositioned, and reattached, and Botox may be injected into the muscle.

The muscles of the vertebral column are very complex; they have multiple attachments, and they exhibit extensive overlap (figure 11.14 and table 11.8). All of these muscles are covered by the most superficial back muscles, which move the upper limb.

Note that the neck is the cervical portion of the vertebral column. Thus, the posterior muscles discussed previously in connection with neck extension (splenius cervicis, splenius capitis, longissimus capitis, semispinalis capitis) extend the *cervical region* of the vertebral column.

The *erector spinae* are used to maintain posture and to help an individual stand erect. When the left and right erector spinae muscles contract together, they extend the vertebral column. If the erector spinae muscles on only one side contract, the vertebral column flexes laterally toward that side.
The erector spinae muscles are organized into three groups. The muscles are named based upon the body region with which they are associated.

- **The iliocostalis group** is the most laterally placed of the three erector spinae components. It is composed of three parts: cervical, thoracic, and lumbar, which attach either to the angles of the ribs or to the transverse processes of cervical vertebrae.

- **The longissimus group** is medial to the iliocostalis group. The fibers of the longissimus muscle group attach to the transverse processes of the vertebrae. The longissimus group is composed of three parts: capitis, cervical, and thoracic.

- **The spinalis group** is the most medially placed of the erector spinae muscles. The spinalis muscle fibers attach to the spinous processes of the vertebrae. The spinalis group is composed of cervical and thoracic parts.

Deep to the erector spinae, a group of muscles collectively called the **transversospinalis muscles** connect and stabilize the vertebrae. This group includes several specific muscles, which are listed in Table 11.8. A final pair of muscles help move the vertebral column. The **quadratus lumborum muscles** are located primarily in the lumbar region. When the left and right quadratus lumborum muscles bilaterally contract, they extend the vertebral column. When either the left or right quadratus lumborum muscle unilaterally contracts, it laterally flexes the vertebral column.

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Superior Attachment(s) (S)/Inferior Attachment(s) (I) $^1$</th>
<th>Innervation (see sections 14.5c, d, f)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iliocostalis group</td>
<td>Bilateral action: Extends vertebral column; maintains posture</td>
<td>S: Angles of ribs; transverse processes of cervical vertebrae</td>
<td>Cervical, thoracic, and lumbar spinal nerves</td>
</tr>
<tr>
<td><em>(ili-o-kos-ta’lis)</em></td>
<td>Unilateral action: Laterally flexes vertebral column</td>
<td>I: Tendon from posterior part of iliac crest, posterior sacrum, and lumbar spinous processes</td>
<td></td>
</tr>
<tr>
<td><em>ilio = ilium</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>cost = rib</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Longissimus group</td>
<td>Bilateral action: Extends vertebral column; maintains posture</td>
<td>S: Mastoid process of temporal bone and transverse processes of cervical and thoracic vertebrae</td>
<td>Cervical and thoracic spinal nerves</td>
</tr>
<tr>
<td><em>(lon-gis’i-mus)</em></td>
<td>Unilateral action: Rotates head and laterally flexes vertebral column</td>
<td>I: Tendon from posterior part of iliac crest, posterior sacrum, and lumbar spinous processes</td>
<td></td>
</tr>
<tr>
<td><em>Spinalis group</em></td>
<td>Bilateral action: Extends vertebral column; maintains posture</td>
<td>S: Spinal process of axis and thoracic vertebrae</td>
<td>Cervical and thoracic spinal nerves</td>
</tr>
<tr>
<td><em>(spt-nal’is)</em></td>
<td>Unilateral action: Laterally flexes vertebral column</td>
<td>I: Lumbar spinous processes (thoracic part) and C7 spinous process (cervical part)</td>
<td></td>
</tr>
<tr>
<td><em>spin = spine</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transversospinalis group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multifidus</td>
<td>Bilateral action: Extends vertebral column toward opposite side</td>
<td>S: Spinal process of vertebrae located 2–4 segments superior to inferior attachment</td>
<td>Cervical, thoracic, and lumbar spinal nerves</td>
</tr>
<tr>
<td><em>(mul-tif’i-dus)</em></td>
<td>Unilateral action: Rotates vertebral column toward opposite side</td>
<td>I: Sacrum and transverse processes of each vertebra</td>
<td></td>
</tr>
<tr>
<td><em>multus = much</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>findo = to cleave</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotatores</td>
<td>Bilateral action: Extends vertebral column toward opposite side</td>
<td>S: Spinal process of immediately superior vertebra</td>
<td>Cervical, thoracic, and lumbar spinal nerves</td>
</tr>
<tr>
<td><em>(ro-ta’torz)</em></td>
<td>Unilateral action: Rotates vertebral column toward opposite side</td>
<td>I: Transverse processes of immediately inferior vertebra</td>
<td></td>
</tr>
<tr>
<td>Semispinalis group</td>
<td>Bilateral action: Extends vertebral column and neck</td>
<td>S: Occipital bone and spinal processes of cervical and thoracic vertebra</td>
<td>Cervical and thoracic spinal nerves</td>
</tr>
<tr>
<td><em>(sem’è-spnal’is)</em></td>
<td>Unilateral action: Laterally flexes vertebral column and neck</td>
<td>I: Transverse processes of C4–T12 vertebra</td>
<td></td>
</tr>
<tr>
<td><em>Spinous extensors and lateral flexors</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quadratus lumborum</td>
<td>Bilateral action: Laterally flexes vertebral column</td>
<td>S: 12th rib; transverse processes of lumbar vertebra</td>
<td>Thoracic and lumbar spinal nerves</td>
</tr>
<tr>
<td><em>(kwah-drä’tus lum-bor’um)</em></td>
<td>Unilateral action: Laterally flexes vertebral column</td>
<td>I: Iliac crest</td>
<td></td>
</tr>
<tr>
<td><em>quad = four-sided</em></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. The superior attachments for vertebral column muscles are more movable typically than the inferior attachments.

2. **Bilateral action** means both the left and right muscles are contracting together.

3. **Unilateral action** means only one muscle (either the left or right muscle) is contracting.

**WHAT DID YOU LEARN?**

Which muscles form the erector spinae, and what are the general actions of the erector spinae?
11.5 Muscles of Respiration

**LEARNING OBJECTIVES**

17. List the posterior and anterior thoracic muscle groups involved in respiration, and describe their actions.

18. Describe the role of the diaphragm in breathing and in raising intra-abdominal pressure.

The process of respiration involves inspiration and expiration. During **inspiration**, several muscles contract to increase the dimensions of the thoracic cavity to allow the lungs to fill with air. During **expiration**, some respiratory muscles contract and others relax, collectively decreasing the dimensions of the thoracic cavity and forcing air out of the lungs (see section 23.5b).

The muscles of respiration are on the posterior and anterior surfaces of the thorax. These muscles are covered by more superficial muscles (such as the pectoral muscles, trapezius, and latissimus dorsi) that move the upper limb.

Two posterior thorax muscles assist with respiration. The **serratus posterior superior** attaches to ribs 2–5 (figure 11.14) and elevates these ribs during forced inspiration, thereby increasing the lateral dimensions of the thoracic cavity. The **serratus posterior inferior** attaches to ribs 8–12 and depresses those ribs during forced expiration. (Normal, quiet expiration takes no active muscular effort.)

Several groups of anterior thorax muscles change the dimensions of the thorax during respiration (figure 11.15). The scalene muscles (discussed previously with other neck muscles) help elevate the first and second ribs during forced inspiration, thereby increasing the dimensions of the thoracic cavity.

**Figure 11.15 Muscles of Respiration.** These skeletal muscles contract rhythmically to alter the size of the thoracic cavity and facilitate respiration. (a) Anterior view. (b) A cadaver photo provides an anterolateral view, with the inferior ribs cut to expose the thoracic cavity and the superior surface of the diaphragm. (c) Lateral views demonstrate fiber directions of the external and internal intercostals. (d) Inferior view of the diaphragm. © McGraw-Hill Education/Christine Eckel
The external intercostals extend inferomedially from the superior rib to the adjacent inferior rib. The external intercostals assist in expanding the thoracic cavity by elevating the ribs during inspiration. This movement is like lifting a bucket handle—that is, as the bucket handle (rib) is elevated, its distance from the center of the bucket (thorax) increases. Thus, contraction of the external intercostals increases the transverse dimensions of the thoracic cavity. The internal intercostals lie deep to the external intercostals, and their muscle fibers are at right angles to the external intercostals. The internal intercostals depress the ribs only during forced expiration. A small transversus thoracis extends across the inner surface of the thoracic cage and attaches to ribs 2–6. It helps depress the ribs during forced expiration.

Finally, the diaphragm is an internally placed, dome-shaped muscle that forms a partition between the thoracic and abdominal cavities. The term diaphragm refers to a muscle or group of muscles that covers or partitions an opening. The diaphragm is the most important muscle associated with breathing. The muscle fibers of the diaphragm converge from its margins toward a fibrous central tendon. During inspiration, the diaphragm contracts and the central tendon is pulled inferiorty toward the abdominal cavity, thereby increasing the vertical dimensions of the thoracic cavity.

Table 11.9 summarizes the characteristics of the muscles of respiration. Further details about muscles of respiration are found in section 23.5b.

### Table 11.9 Muscles of Respiration

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action(s)</th>
<th>Superior Attachment(s) (S)/Inferior Attachment(s) (I)¹</th>
<th>Innervation (see sections 14.5c, d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serratus posterior superior</td>
<td>Elevates ribs during forced inspiration</td>
<td>S: Spinous processes of C₇–T₃ vertebrae</td>
<td>Thoracic spinal nerves</td>
</tr>
<tr>
<td>(sèr-a’tūs) serratus = a saw</td>
<td></td>
<td>I: Lateral borders of ribs 2–5</td>
<td></td>
</tr>
<tr>
<td>Serratus posterior inferior</td>
<td>Depresses ribs during forced expiration</td>
<td>S: Inferior borders of ribs 8–12 or 9–12</td>
<td>Thoracic spinal nerves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Spinous processes of T₁₁–L₃ vertebrae</td>
<td></td>
</tr>
<tr>
<td>Scalene muscles</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(anterior, middle posterior) (described in table 11.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>External intercostals</td>
<td>Elevate ribs during quiet and forced inspiration</td>
<td>S: Inferior border of superior rib</td>
<td>Thoracic spinal nerves</td>
</tr>
<tr>
<td>(in’ter-kos’talz) inter = between cost = rib</td>
<td></td>
<td>I: Superior border of inferior rib</td>
<td></td>
</tr>
<tr>
<td>Internal intercostals</td>
<td>Depresses ribs during forced expiration</td>
<td>S: Superior border of inferior rib</td>
<td>Thoracic spinal nerves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Inferior border to superior rib</td>
<td></td>
</tr>
<tr>
<td>Transversus thoracis</td>
<td>Depresses ribs during forced expiration</td>
<td>S: Costal cartilages 2–6</td>
<td>Thoracic spinal nerves</td>
</tr>
<tr>
<td>(trans-ver’sūs thō-ra’sis)</td>
<td></td>
<td>I: Posterior surface of xiphoid process and inferior region of sternum</td>
<td></td>
</tr>
<tr>
<td>Diaphragm</td>
<td>Contraction causes flattening of diaphragm (moves inferiorly) during inspiration and thus expands thoracic cavity; increases pressure in abdominopelvic cavity</td>
<td>S: Central tendon</td>
<td>Phrenic nerves (C₇–C₃)</td>
</tr>
<tr>
<td>(dī’frām) dia = across phragm = partition</td>
<td></td>
<td>I: Inferior internal surface of ribs 7–12; xiphoid process of sternum and costal cartilages of inferior 6 ribs; lumbar vertebrae</td>
<td></td>
</tr>
</tbody>
</table>

1. The superior attachment of these muscles is more movable, except for serratus posterior superior and internal intercostals.

### LEARNING OBJECTIVES

19. List the four pairs of abdominal muscles.
20. Compare the actions of the rectus abdominis muscle with the oblique muscles and transversus abdominis.

The anterolateral wall of the abdomen is reinforced by four pairs of muscles that collectively compress and hold the abdominal organs in place: the external oblique, internal oblique, transversus abdominis, and rectus abdominis (figure 11.16). These muscles also work
Figure 11.16 Muscles of the Abdominal Wall. The abdominal muscles compress abdominal contents and flex the vertebral column. (a) An illustration depicts the anterior view of some superficial and deep muscles. (b) A cadaver photo provides an anterolateral view of the muscles of the abdominal wall. (c) Diagrams show some individual abdominal muscles, ranging from superficial to deep. (b) ©McGraw-Hill Education/Christine Eckel
The fibers of the external intercostals and external oblique muscles run in the same direction—inferomedially (downward and toward the body’s midline). This is the same direction that you put your hands in your pockets.

The fibers of the internal intercostals and internal oblique muscles run perpendicular (in the opposite direction) to the external muscles—superomedially (upward and toward the body’s midline).

together to flex and stabilize the vertebral column. When the oblique and transversus abdominis muscles unilaterally contract, they laterally flex the vertebral column.

The muscle fibers of the superficial external oblique are directed inferomedially. The external oblique is composed of muscle along the lateral abdominal wall and forms an aponeurosis as it projects anteriorly. Inferiorly, the aponeurosis of the external oblique forms a strong, cordlike inguinal ligament that extends from the anterior superior iliac spine to the pubic tubercle. Immediately deep to the external oblique is the internal oblique. Its muscle fibers project superomedially, which is at right angles to the external oblique. Like the external oblique, this muscle forms an aponeurosis as it projects anteriorly. Unilaterally, the external oblique on one side of the body and the internal oblique on the opposite side of the body work together to rotate the vertebral column.

The deepest muscle is the transversus abdominis, whose fibers project transversely across the abdomen and which has an aponeurosis as it projects anteriorly. When the transversus abdominis unilaterally contracts, it laterally flexes the vertebral column.

The rectus abdominis is a long, straplike muscle that extends vertically the entire length of the anteromedial abdominal wall between the sternum and the pubic symphysis. It is partitioned into four segments by three fibrous tendinous intersections, which form the traditional “six-pack” of a muscular, toned abdominal wall. The rectus abdominis is enclosed within a fibrous sleeve called the rectus sheath, which is formed from the aponeuroses of the external oblique, internal oblique, and transversus abdominis muscles. The left and right rectus sheaths are connected by a vertical fibrous strip termed the linea alba.

Table 11.10 summarizes the characteristics of the muscles of the abdominal wall.

You have probably noticed that multiple muscles may work together to perform a common function. For example, several neck muscles and back muscles work together to extend the vertebral column. Learning muscles in groups according to common function helps most students assimilate the anatomy information. Table 11.11 summarizes the actions of various axial muscles and groups them according to common function. Note that a muscle that has multiple functions is listed in more than one group.

What are the main actions of the abdominal muscles?

How Do Inguinal Hernias Form, and Why Are Males More Prone to Get Them?

An inguinal hernia is the most common type of hernia. The inguinal region is one of the weakest areas of the abdominal wall. Within this region is a canal (inguinal canal) that allows the passage of the spermatic cord in males (see figure 28.15 in section 28.4a), and a smaller structure called the round ligament in females (see figure 28.9 in section 28.3c). The inguinal canal, or the superficial inguinal ring associated with it, is often the site of a rupture or separation of the abdominal wall. Males are more likely to develop inguinal hernias than females because their inguinal canals and superficial inguinal rings are larger to allow room for the spermatic cord. Rising pressure in the abdominal cavity, as might develop while straining to lift a heavy object, provides the force to push a segment of the small intestine into the canal.

How Do Physicians Test for an Inguinal Hernia?

The physician inserts a finger in the depression formed by the superficial inguinal ring and asks the patient to turn his or her head and cough, because the act of coughing increases intra-abdominal pressure and would potentially induce a portion of intestine to poke through the ring if there was a problem. While the patient coughs, the physician palpates the superficial inguinal ring to make sure no intestine is protruding through the ring.

11.7 Muscles of the Pelvic Floor

LEARNING OBJECTIVES

21. Describe the functions of the pelvic floor muscles.

22. Identify the boundaries of the perineum.

The floor of the pelvic cavity is formed by three layers of muscles and associated fasciae, collectively known as the pelvic diaphragm. The pelvic diaphragm extends from the ischium and pubis of the ossa coxae across the pelvic outlet to the sacrum and coccyx. These muscles collectively form the pelvic floor and support the pelvic viscera (figure 11.17).

The diamond-shaped region between the lower appendages is called the perineum (per′i-nē′um). The perineum has four significant bony landmarks: the pubic symphysis anteriorly, the coccyx posteriorly, and both ischial tuberosities laterally. A transverse line drawn between the ischial tuberosities partitions the perineum into an anterior urogenital triangle, which contains the external genitalia and urethra, and a posterior anal triangle, which contains the anus (figure 11.17b, c).
### Table 11.10 Muscles of the Abdominal Wall

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action(s)</th>
<th>Superior Attachment(s) (S)/Inferior Attachment(s) (I)</th>
<th>Innervation (see sections 14.5c, f)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>External oblique</strong></td>
<td>Bilateral action: Flexes vertebral column and compresses abdominal wall Unilateral action: Laterally flexes vertebral column; rotates vertebral column to same side</td>
<td>S: External and inferior borders of the inferior 8 ribs (ribs 5–12) I: Linea alba by a broad aponeurosis; some to iliac crest</td>
<td>Spinal nerves T8–T12, L1</td>
</tr>
<tr>
<td><strong>Internal oblique</strong></td>
<td>Bilateral action: Flexes vertebral column and compresses abdominal wall Unilateral action: Laterally flexes vertebral column; rotates vertebral column to opposite side</td>
<td>S: Lumbar fascia, inguinal ligament, and iliac crest I: Linea alba, pubic crest, inferior rib surfaces of the inferior 4 ribs (9–12); costal cartilages of ribs 8–10</td>
<td>Spinal nerves T8–T12, L1</td>
</tr>
<tr>
<td><strong>Transversus abdominis</strong></td>
<td>Bilateral action: Flexes vertebral column and compresses abdominal wall Unilateral action: Laterally flexes vertebral column</td>
<td>S: Iliac crest, cartilages of inferior 6 ribs (ribs 7–12); lumbar fascia; inguinal ligament I: Linea alba and pubic crest</td>
<td>Spinal nerves T8–T12, L1</td>
</tr>
<tr>
<td><strong>Rectus abdominis</strong></td>
<td>Flexes vertebral column and compresses abdominal wall</td>
<td>S: Xiphoid process of sternum; inferior surfaces of ribs 5–7 I: Superior surface of pubis near pubic symphysis</td>
<td>Spinal nerves T7–T12</td>
</tr>
</tbody>
</table>

1. *Bilateral action* means both the left and right muscles are contracting together.
2. *Unilateral action* means only one muscle (either the left or right muscle) is contracting.

---

### Table 11.11 Muscle Actions on the Axial Skeleton

<table>
<thead>
<tr>
<th>Extend the Neck and/or Vertebral Column</th>
<th>Flex the Neck and/or Vertebral Column</th>
<th>Laterally Flex the Vertebral Column</th>
<th>Rotate the Head to One Side</th>
<th>Elevate the Ribs</th>
<th>Depress the Ribs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Splenius muscles¹</td>
<td>Sternocleidomastoid²</td>
<td>Quadratus lumborum²</td>
<td>Serratus posterior superior</td>
<td>Serratus posterior inferior</td>
<td></td>
</tr>
<tr>
<td>Erector spinae (iliocostalis, longissimus, spinalis)</td>
<td>Scalene muscles¹</td>
<td>External oblique²</td>
<td>Splenius muscles²</td>
<td>External intercostals</td>
<td>Internal intercostals</td>
</tr>
<tr>
<td>Quadratus lumborum¹</td>
<td>External oblique¹</td>
<td>Internal oblique²</td>
<td>Longissimus capitis³</td>
<td>Scalenus muscles (1st and 2nd ribs only)</td>
<td>Transversus thoracis</td>
</tr>
<tr>
<td>Transversospinalis group¹</td>
<td>Internal oblique¹</td>
<td>Transversus abdominis²</td>
<td>Obliquus capitis inferior²</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rectus capitis posterior major and minor¹</td>
<td>Transversus abdominis¹</td>
<td>Rectus abdominis¹</td>
<td>Obliquus capitis superior²</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. *Bilateral action*
2. *Unilateral action of muscles*
Figure 11.17 Muscles of the Pelvic Floor. The pelvic cavity floor is composed of muscle layers that form the urogenital and anal triangles, extend across the pelvic outlet, and support the organs in the pelvic cavity. (The puborectalis muscle is not shown.) (a) Superior view of the female pelvic floor. Inferior views show (b) both the superficial and deep muscles of the male and (c) female perineal regions. Muscles of the pelvic floor are in bold.
<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Origin/Insertion¹</th>
<th>Innervation (see section 14.5g)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ANAL TRIANGLE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coccygeus (kok-sij′-ē-ús)</td>
<td>Forms pelvic floor and supports pelvic viscera</td>
<td>O: Ischial spine</td>
<td>Spinal nerves S4–S5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Lateral and inferior borders of sacrum and coccyx</td>
<td></td>
</tr>
<tr>
<td><strong>External anal sphincter</strong></td>
<td>Constricts anal opening; must voluntarily relax to defecate</td>
<td>O: Perineal body</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td>(ʼa-nāl sfin′g-k′tēr) anal = referring to anus</td>
<td></td>
<td>I: Encircles anal opening</td>
<td></td>
</tr>
<tr>
<td>sphin = squeeze</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Levator ani</strong> (lē-vā′-tor, lē-vā-ter ʼā-nī; levator = raises; ani = anus): Group of muscles that form the anterior and lateral parts of the pelvic diaphragm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iliococcygeus (il′-ē-ō-kok-si′jē-ūs)</td>
<td>Forms pelvic floor and supports pelvic viscera</td>
<td>O: Pubis and ischial spine</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Coccyx and median raphe</td>
<td></td>
</tr>
<tr>
<td>Pubococcygeus (py′bō-kok-si′jē-ūs)</td>
<td>Forms pelvic floor and supports pelvic viscera</td>
<td>O: Pubis and ischial spine</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Coccyx and median raphe</td>
<td></td>
</tr>
<tr>
<td>Puborectalis (py′bōrek′tāl-is) rectal = rectum</td>
<td>Supports anorectal junction; must relax to defecate</td>
<td>O: Pubis and ischial spine</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Coccyx and median raphe</td>
<td></td>
</tr>
<tr>
<td><strong>UROGENITAL TRIANGLE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Superficial layer</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulbospongiosus (female)</td>
<td>Narrows vaginal opening; compresses and stiffens clitoris</td>
<td>O: Sheath of collagen fibers at base of clitoris</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td>(bul′bō-spūn′jē-ō′-sūs) bulbon = bulb spongio = sponge</td>
<td></td>
<td>I: Perineal body</td>
<td></td>
</tr>
<tr>
<td>Bulbospongiosus (male)</td>
<td>Ejects urine or semen; compresses base of penis; stiffens penis</td>
<td>O: Sheath of collagen fibers at base of penis</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td>(ish′ē-ō-kav′er-nō′-sūs) caverna = hollow chamber</td>
<td></td>
<td>I: Median raphe and perineal body</td>
<td></td>
</tr>
<tr>
<td>Ischiocavernosus</td>
<td>Assists with erection of penis or clitoris</td>
<td>O: Ischial tuberosities and ischial ramus</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td>(ish′ē-ō-kav′er-nō′-sūs) caverna = hollow chamber</td>
<td></td>
<td>I: Pubic symphysis</td>
<td></td>
</tr>
<tr>
<td><strong>Superficial transverse perineal muscle</strong> (per′i-nē′āl)</td>
<td>Supports pelvic organs</td>
<td>O: Ramus of ischium</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td><strong>Deep layer (urogenital diaphragm)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep transverse perineal muscle</td>
<td>Supports pelvic organs</td>
<td>O: Ischial ramus</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I: Median raphe of urogenital diaphragm</td>
<td></td>
</tr>
<tr>
<td>External urethral sphincter</td>
<td>Constricts urethra; must voluntarily relax in order to urinate</td>
<td>O: Rami of ischium and pubis</td>
<td>Pudendal nerve (S2–S4)</td>
</tr>
<tr>
<td>(yū-rē-thrāl)</td>
<td></td>
<td>I: Median raphe of urogenital diaphragm</td>
<td></td>
</tr>
</tbody>
</table>

¹ Here, we use the terms origin and insertion because the terms superior attachment and inferior attachment are not applicable for all of the muscles in this table.

Table 11.12 describes the specific muscles of the pelvic floor and perineum, and summarizes their characteristics.

**WHAT DID YOU LEARN?**

What are the functions of the pelvic floor muscles?

**INTEGRATE**

**CONCEPT CONNECTION**

The muscles of the pelvic floor may become stretched or torn after childbirth (see section 29.6), so women may not have adequate support for the pelvic organs. These issues may become more problematic as a woman ages. As a result, incontinence (leakage of urine) is a common complaint among women who have given birth. Strengthening the pelvic floor muscles (with exercises such as Pilates or Kegel exercises) often alleviates these problems.
Figure 11.18 Anterior Muscles Associated with the Proximal Upper Limb. This anterior view compares some components of both the axial and appendicular musculature. Only those muscles that move the upper limb are labeled. Superficial muscles are shown on the right side of the body, and deep muscles are shown on the left side.

Figure 11.19 Posterior Muscles Associated with the Proximal Upper Limb. This posterior view compares some components of both the axial and appendicular musculature. Only those muscles that move the upper limb are labeled. Superficial muscles are shown on the left side of the body, and deep muscles are shown on the right.
11.8 Muscles of the Pectoral Girdle and Upper Limb

Muscles that move the pectoral girdle and upper limbs are organized into specific groups:

- Muscles that move the pectoral girdle
- Muscles that move the glenohumeral joint/arm
- Arm and forearm muscles that move the elbow joint/forearm
- Forearm muscles that move the wrist joint, hand, and fingers
- Intrinsic muscles of the hand

Some of these muscles are superficial, and others are deep (figures 11.18 and 11.19).

11.8a Muscles That Move the Pectoral Girdle

**LEARNING OBJECTIVE**

23. Compare and contrast how the anterior and posterior thoracic muscles move the pectoral girdle.

The muscles of the pectoral girdle arise from the axial skeleton and attach to either the scapula or the clavicle. These muscles both stabilize the scapula and move it to increase the arm’s angle of movements. Some of the superficial muscles of the thorax are grouped together according to the scapular movement they direct: elevation, depression, protraction, or retraction (figure 11.20).

**Figure 11.20 Actions of Some Thoracic Muscles on the Scapula.** Individual muscles may contribute to different, multiple actions. (a) The scapula can be retracted or protracted. When you are standing upright and have good posture, your scapulae are retracted. Conversely, poor posture demonstrates scapular protraction. (b) Muscles that elevate and depress the scapula. (c) Muscles that rotate the scapula.
The muscles that move the pectoral girdle are classified according to their location in the thorax as either anterior or posterior thoracic muscles. The anterior thoracic muscles are the pectoralis minor, serratus anterior, and subclavius (figure 11.21a).

The pectoralis minor is deep to the pectoralis major. This muscle helps depress and protract (pull anteriorly) the scapula. When your shoulders are hunched forward, the pectoralis minor muscle is contracting. The serratus anterior is a large, flat, fan-shaped muscle positioned...
Muscles that move the pectoral girdle do not adhere to any one set of attachment terms. We use terminology that best describes the muscle attachments.

The posterior thoracic muscles are the levator scapulae, rhomboid major, rhomboid minor, and trapezius (figure 11.21b). The levator scapulae arises from multiple heads on the transverse processes of the cervical vertebrae and attaches to the superior angle of the scapula. As its name implies, its primary action is to elevate the scapula. It can also inferiorly rotate the scapula so that the glenoid cavity moves inferiorly.

Both the rhomboid major and rhomboid minor are located deep to the trapezius. These rhomboid muscles are parallel bands that are directed inferolaterally from the vertebral column to the scapula. They help elevate and retract (adduct) the scapula, as when you are standing up straight. The rhomboid muscles also inferiorly rotate the scapula.

The trapezius is a large, diamond-shaped muscle that arises from the skull and vertebral column to both the scapula and the clavicle of the pectoral girdle laterally. The trapezius can elevate, depress, retract, or rotate the scapula, depending upon which fibers of the muscle are contracting.

Table 11.13 summarizes the characteristics of the thoracic muscles that move the pectoral girdle.

### WHAT DID YOU LEARN?

List the posterior thoracic muscles that move the pectoral girdle, and describe their common action(s).

---

**Table 11.13** Thoracic Muscles That Move the Pectoral Girdle

<table>
<thead>
<tr>
<th>Group</th>
<th>Action(s)</th>
<th>Attachments: Superior (S)/Inferior (I) or Medial (M)/Lateral (L)</th>
<th>Innervation (see sections 13.9 and 14.5d, e)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ANTERIOR MUSCLES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pectoralis minor</td>
<td>Protracts (abducts) and depresses scapula</td>
<td>S: Coracoid process of scapula I: Ribs 3–5</td>
<td>Medial pectoral nerve (C8–T1)</td>
</tr>
<tr>
<td>(pectus = chest)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serratus anterior</td>
<td>Agonist in scapula protraction; rotates scapula superiorily</td>
<td>S: Medial border of scapula; anterior surface I: Ribs 1–8, anterior and superior margins</td>
<td>Long thoracic nerve (C5–C7)</td>
</tr>
<tr>
<td>(serratus = saw)</td>
<td>(so glenoid cavity moves superiorily); stabilizes scapula</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subclavius</td>
<td>Depresses and stabilizes clavicle</td>
<td>S: Inferior surface of clavicle I: Rib 1</td>
<td>Nerve to subclavius (C5–C6)</td>
</tr>
<tr>
<td>(sub = under clav = clavicle)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>POSTERIOR MUSCLES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Levator scapulae</td>
<td>Elevates scapula; rotates scapula inferiorly (moves glenoid cavity inferiorly)</td>
<td>S: Transverse processes of C7–C4 I: Superior part of medial border of scapula</td>
<td>Cervical nerves (C3–C4) and dorsal scapular nerve (C5)</td>
</tr>
<tr>
<td>(levator = raises)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhomboid major</td>
<td>Elevates and retracts (adducts) scapula; rotates scapula inferiorly</td>
<td>M: Spinous processes of T2–T3 L: Medial border of scapula from spine to inferior angle</td>
<td>Dorsal scapular nerve (C5)</td>
</tr>
<tr>
<td>(rhomboid = diamond-shaped)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhomboid minor</td>
<td>Elevates and retracts (adducts) scapula; rotates scapula inferiorly</td>
<td>M: Spinous processes of C3–T1 L: Medial border of scapula superior to spine</td>
<td>Dorsal scapular nerve (C5)</td>
</tr>
<tr>
<td>Trapezius</td>
<td>Superior fibers: Elevate scapula; rotate scapula superiorly</td>
<td>M: Occipital bone (superior nuchal line); ligamentum nuchae; spino-cranial nerves of C7–T12 L: Clavicle; acromion process and spine of scapula</td>
<td>Accessory nerve (CN XI)</td>
</tr>
<tr>
<td>(trapezius = irregular four-sided figure)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Muscles that move the pectoral girdle do not adhere to any one set of attachment terms. We use terminology that best describes the muscle attachments.
11.8b Muscles That Move the Glenohumeral Joint/Arm

LEARNING OBJECTIVES

24. List the muscles that extend, flex, adduct, and abduct the glenohumeral joint.
25. Compare the actions of the four scapular muscles of the rotator cuff.

The phrases “moving the glenohumeral joint” and “moving the arm or humerus” mean the same thing. A movement such as flexion of the arm requires movement at the glenohumeral joint. Throughout this text, we refer to both the joint where the movement is occurring and the body region that is being moved to minimize any confusion.

The glenohumeral joint is crossed by 11 muscles that attach distally to the arm (humerus) or forearm (radius and/or ulna) (figure 11.21). The latissimus dorsi is a broad, triangular muscle located on the inferior part of the back. Often, it is referred to as the “swimmer’s muscle,” because its actions are required for many swimming strokes. It is the prime arm extensor and adductor, and it medially rotates the arm. The pectoralis major is a large, thick, fan-shaped muscle that covers the superior part of the thorax. It is the principal flexor and adductor of the arm, and it medially rotates the arm.

The latissimus dorsi and pectoralis major muscles are the primary attachments of the arm to the trunk, and they are the prime movers of the glenohumeral joint. These muscles are antagonists with respect to arm flexion and arm extension. However, these two muscles work together (synergistically) when performing other movements, such as adducting and medially rotating the humerus.

The triceps brachii and biceps brachii, discussed in detail with the muscles that move the elbow joint (see section 11.8c), also span the glenohumeral joint to move the humerus. Specifically, the long head of the triceps brachii arises from the infraglenoid tubercle of the scapula, and helps extend and adduct the arm. The long head of the biceps brachii arises from the supraglenoid tubercle of the scapula and assists in flexing the arm.

The seven remaining muscles that move the humerus at the glenohumeral joint are termed the scapular muscles, because they arise from the scapula. They include the deltoid, the coracobrachialis, the teres major, and the four muscles of the rotator cuff.

The deltoid is a thick, powerful muscle that forms the rounded contour of the shoulder. Note that the fibers of the deltoid arise from three different points, and these different fiber groups all perform different functions: (1) The anterior fibers are the primary flexors of the arm and medially rotate the arm. (2) The middle (lateral) fibers abduct the arm; in fact, the deltoid is the prime abductor of the arm. (3) The posterior fibers are the principal extensors of the arm and laterally rotate the arm. The coracobrachialis is a synergist to the pectoralis major in flexing and adducting the arm. The teres major works synergistically with the latissimus dorsi by extending, adducting, and medially rotating the arm.

Among the scapular muscles, four rotator cuff muscles (subscapularis, supraspinatus, infraspinatus, and teres minor) provide the strength and stability of the glenohumeral joint (figure 11.22). These muscles attach the scapula to the humerus (see also figure 9.15). The specific movements of each muscle are best learned when relating their actions to pitching a ball:

- The subscapularis is used when you wind up for a pitch. It medially rotates the arm.
- The supraspinatus is used when you start to execute the pitch, by fully abducting the arm.
- The infraspinatus and teres minor help slow down the pitching arm upon completion of the pitch. These two muscles adduct and laterally rotate the arm.

Table 11.14 summarizes the characteristics of the muscles that move the glenohumeral joint and arm.

CLINICAL VIEW 11.6

Rotator Cuff Injuries

A rotator cuff injury is the result of trauma or disease that affects any portion of the rotator cuff musculature or tendons. Extensive and repetitive use of the rotator cuff muscles can cause tearing of muscle fibers or rupture of tendon attachments. The rotator cuff muscles also may be injured upon a fall on the shoulder or by trying to lift an object that is too heavy. The supraspinatus muscle is most commonly involved, likely because the tendon may become impinged (pinched) inferior to the acromion during use of the muscle. The risk of rotator cuff injuries increases with age due to years of use, reduced blood flow to the muscles and tendons as we age, and the increased likelihood of developing bone spurs in the shoulder that impinge on the tendons.

Common symptoms of a rotator cuff injury include swelling and tenderness in the area of the shoulder, as well as pain with specific shoulder movements, especially abduction. The pain may range from moderate to severe. This syndrome is especially common in baseball players because the repetitive shoulder movements while pitching and throwing the ball can impinge the supraspinatus tendon against the acromion. Painters also may experience rotator cuff injuries due to the repetitive overhead upper limb movements involved with their work. Diagnosis may be confirmed with both a physical (that asks about a patient history and performs range of movement activities to the affected upper limb) and imaging studies, such as MRI or ultrasound.

Treatment is dependent upon on the severity of the injury. Initially, the pain may be controlled with icing, NSAIDs (e.g., aspirin), or corticosteroid shots to the affected region. Physical therapy is used to restore the shoulder’s range of motion and strengthen affected muscles. Severe rotator cuff injuries that have not been helped with nonsurgical therapies typically require surgical repair, which may include removal of bone spurs as well as repair of the torn tendon. This surgery may be done arthroscopically (a small camera called an arthroscope is inserted through a small incision into the joint to guide the physician’s use of small surgical instruments), or a more invasive open repair surgery may be required. Physical therapy is required after surgery to restore range of motion.
Figure 11.22 Rotator Cuff Muscles. The rotator cuff muscles reinforce the glenohumeral joint and secure the head of the humerus in the glenoid cavity. (a) The subscapularis is located on the anterior aspect of the scapula and medially rotates the humerus (as in winding up for a pitch). (b) The supraspinatus abducts the humerus (as in executing the pitch), whereas the infraspinatus and teres minor laterally rotate the humerus (as in completing the pitch and slowing down the pitching arm). These three muscles are located along the posterior aspect of the scapula.

Table 11.14 Muscles That Move the Glenohumeral Joint/Arm

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5e)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUSCLES ARISING FROM THE AXIAL SKELETON</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Latissimus dorsi</td>
<td>Extends arm (agonist); adducts arm (agonist); medially rotates arm (&quot;swimmer’s muscle&quot;)</td>
<td>P: Spineous processes of T₇–T₁₂; ribs 8–12; iliac crest; thoracolumbar fascia</td>
<td>Thoracodorsal nerve (C₆–C₈)</td>
</tr>
<tr>
<td>(lā-tis’i-mūs dōr’st)</td>
<td></td>
<td>D: Intertubercular sulcus of humerus</td>
<td></td>
</tr>
<tr>
<td>latissimus = widest</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dorsi = back</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pectoralis major</td>
<td>Flexes arm (agonist); adducts and medially rotates arm</td>
<td>P: Medial clavicle; costal cartilages of ribs 2–6; body of sternum</td>
<td>Lateral pectoral (C₅–C₇) and medial pectoral (C₈–T₁) nerves</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Lateral part of intertubercular sulcus of humerus</td>
<td></td>
</tr>
<tr>
<td>MUSCLES ARISING FROM THE SCAPULA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deltoid</td>
<td>Anterior fibers: Flex and medially rotate arm</td>
<td>P: Acromial end of clavicle; acromion and spine of scapula</td>
<td>Axillary nerve (C₅–C₆)</td>
</tr>
<tr>
<td>(del’toyd)</td>
<td>Middle fibers: Adduct arm (agonist)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>delta = triangular</td>
<td>Posterior fibers: Extend and laterally rotate arm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coracobrachialis</td>
<td>Adducts and flexes arm</td>
<td>P: Coracoid process of scapula</td>
<td>Musculocutaneous nerve (C₅–C₆ nerve fibers)</td>
</tr>
<tr>
<td>(kōr’a-kō-brā-kē-a’lis)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>coraco = coracoid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>brachi = arm</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(continued on next page)
### Table 11.15

#### Summary of Muscle Actions at the Glenohumeral Joint/Arm

<table>
<thead>
<tr>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5e)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Abduction</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deltoid (middle fibers)</td>
<td>Inferior lateral border and inferior angle of scapula</td>
<td>Lower subscapular nerve (C5–C6)</td>
</tr>
<tr>
<td><strong>Adduction</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Latissimus dorsi</td>
<td>Inferior lateral border and inferior angle of scapula</td>
<td>Lower subscapular nerve (C5–C6)</td>
</tr>
<tr>
<td><strong>Extension</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pectoralis major</td>
<td>Greater tubercle of humerus</td>
<td>Suprascapular nerve (C5–C6)</td>
</tr>
<tr>
<td><strong>Flexion</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infraspinatus</td>
<td>Supraglenoid tubercle of scapula</td>
<td>Musculocutaneous nerve (C5–C6 nerve fibers)</td>
</tr>
<tr>
<td><strong>Lateral Rotation</strong></td>
<td>Infraglenoid tubercle of scapula</td>
<td>Radial nerve (C5–C7 nerve fibers)</td>
</tr>
<tr>
<td>Teres minor</td>
<td>Infraspinous fossa or scapula</td>
<td></td>
</tr>
<tr>
<td><strong>Medial Rotation</strong></td>
<td>Greater tubercle of humerus</td>
<td></td>
</tr>
<tr>
<td>Supraspinatus</td>
<td>Supraglenoid tubercle of scapula</td>
<td>Musculocutaneous nerve (C5–C6 nerve fibers)</td>
</tr>
</tbody>
</table>

#### Rotator Cuff Muscles

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5e)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Teres major</strong> (ter′ez) teres = round</td>
<td>Extends, adducts, and medially rotates arm</td>
<td>Inferior lateral border and inferior angle of scapula</td>
<td>Lower subscapular nerve (C5–C6)</td>
</tr>
<tr>
<td><strong>Triceps brachii (long head)</strong> (tr′i-seps brak′kē-t) triceps = three heads</td>
<td>Extends and adducts arm</td>
<td>Supraglenoid tubercle of scapula</td>
<td>Musculocutaneous nerve (C5–C6 nerve fibers)</td>
</tr>
<tr>
<td><strong>Biceps brachii (long head)</strong> (bit′seps) biceps = two heads</td>
<td>Flexes arm</td>
<td>Supraglenoid tubercle of scapula</td>
<td>Musculocutaneous nerve (C5–C6 nerve fibers)</td>
</tr>
</tbody>
</table>

The muscles that move the arm at the glenohumeral joint are grouped in **Table 11.15** according to different types of actions. Note that a muscle that has multiple functions is listed in more than one group. For example, the deltoid may abduct, extend, or flex the humerus, depending upon whether its middle, posterior, or anterior fibers are contracting. Pectoralis major and coracobrachialis both adduct and flex the humerus, so they are listed in both the adduction and flexion columns. We recommend that you copy the columns multiple times and then test your knowledge by trying to write out all of the muscles in a group without looking at your notes.

#### WHAT DID YOU LEARN?

19. Which muscles extend the arm at the glenohumeral (shoulder) joint?
20. Identify the rotator cuff muscles, and describe their actions.
When you move the elbow joint, you move the bones of the forearm. Thus, the phrase “flexing the elbow joint” is synonymous with “flexing the forearm.” Keep this point in mind as we discuss the muscles that move the elbow joint and forearm.

The muscles in the limbs are organized into compartments, which are surrounded by deep fascia. Each compartment houses functionally related skeletal muscles, as well as their associated nerves and blood vessels. In general, muscles in the same compartment tend to perform similar functions. Figure 11.23 provides a visual overview of how the muscles are organized into compartments. Note how muscles in opposite compartments tend to be antagonists. For example, the anterior forearm muscles are primarily flexors and pronators, whereas the posterior forearm muscles are primarily extensors and supinators. Likewise, in the lower limb, knee extensors are in the anterior compartment of the thigh, whereas knee flexors are in the posterior compartment of the thigh. Hip adductors are in the medial compartment of the thigh, whereas a hip abductor is in the lateral compartment of the thigh. These compartments may help you to learn the muscles in common functional groups. If you know what compartment a muscle is in, you likely can figure out what action that muscle performs, and vice versa.

What do you think?

The brachialis is on the anterior surface of the arm. Without looking at the muscle tables, determine whether this muscle flexes or extends the elbow joint. How did you reach your conclusion?

The muscles of the arm may be subdivided into an anterior compartment and a posterior compartment. The anterior compartment primarily contains elbow flexors, so it is also known as the flexor compartment. Muscles in this compartment are supplied by the deep brachial artery and are innervated by the musculocutaneous nerve. Muscles in this compartment include the coracobrachialis (note that this muscle is an arm flexor and not an elbow flexor), biceps brachii, brachialis, and brachioradialis muscles. Put your hand on your anterior arm, and then flex your elbow. Observe how these muscles bulge as they contract, affirming that these anterior compartment arm muscles flex the elbow.

The posterior compartment contains elbow extensors, so this compartment is also called the extensor compartment. These muscles receive their blood supply from the deep brachial artery and are innervated by the radial nerve. The primary muscle in this compartment is the triceps brachii. Put your hand on your posterior arm, and then extend your elbow. You can feel your muscles contracting as you perform this action, affirming that posterior arm muscles extend the elbow. Perform these activities each time we discuss muscles in a particular limb compartment so you can see and feel for yourself how these muscles move.

Muscles of the Arm’s Anterior Compartment

On the anterior side of the humerus are the principal flexors of the forearm: the biceps brachii and brachialis (figure 11.24). The biceps brachii is a large, two-headed muscle on the anterior surface of the humerus. This muscle flexes the elbow joint, and it is a powerful supinator of the forearm when the elbow is flexed. (An example of this supination movement occurs when you tighten a screw with your right hand.) The tendon of the long head of the biceps brachii crosses the shoulder joint, and so this muscle helps flex the humerus as well (albeit weakly).

The brachialis is deep to the biceps brachii on the anterior surface of the humerus. It is the most powerful flexor of the forearm at the elbow. The brachioradialis is another prominent muscle on the anterolateral surface of the forearm. It is a synergist in elbow flexion, effective primarily when the prime movers of forearm flexion have already partially flexed the elbow.

Muscles of the Arm’s Posterior Compartment

The posterior compartment of the arm contains two muscles that extend the forearm at the elbow: the triceps brachii and the anconeus (figure 11.25). The triceps brachii is the large, three-headed muscle on the posterior surface of the arm. The long head of the triceps brachii also crosses the glenohumeral joint, where it helps extend the humerus. All three parts of this muscle merge to form a common distal attachment on the olecranon of the ulna. A weak elbow extensor is the small anconeus that crosses the posterolateral region of the elbow.
**Figure 11.23 Muscle Compartmentalization.** In the upper limb, the (a) arm and (b) forearm both may be divided into anterior (flexor) compartments and posterior (extensor) compartments. (c) The thigh may be split into four compartments, whereas the (d) leg is split into three compartments. Each compartment contains muscles that tend to perform similar movements.
Figure 11.24 Anterior Muscles That Move the Elbow Joint/Forearm. (a) The right arm and shoulder show the anterior muscles that produce movements at the elbow joint, labeled in bold. (b) Superficial and deep anterior arm muscles.

Figure 11.25 Posterior Muscles That Move the Elbow Joint/Forearm. (a) The right arm and shoulder show the posterior muscles that produce movements at the elbow joint, labeled in bold. (b) Superficial and deep posterior arm muscles.
Muscles of the Forearm That Act on the Elbow Joint

Some forearm muscles pronate or supinate the forearm (figure 11.26). As their names imply, both the pronator teres and pronator quadratus rotate the radius across the surface of the ulna to pronate the forearm. These muscles are located in the anterior compartment of the forearm. They are antagonistic to the supinator in the posterior compartment of the forearm. The supinator works synergistically with the biceps brachii to supinate the forearm.

Table 11.16 summarizes the characteristics of the muscles that move the forearm, and table 11.17 groups them according to common function. By learning these muscles as groups, you can gain a better understanding of how they work together to perform specific functions.

Table 11.16 Muscles That Move the Forearm

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5e)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FLEXORS (ANTERIOR ARM)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Biceps brachii          | Flexes forearm; supinates forearm when elbow flexed (agonist) | P: Long head: Supraglenoid tubercle of scapula  
                      |                                                   | Short head: Coracoid process of scapula  
                      |                                                   | D: Radial tuberosity and bicipital aponeurosis          | Musculocutaneous nerve (C5–C6 nerve fibers) |
| Long head               | Flexes arm (long head only)                    |                                                      |                                                             |
| Short head              |                                                |                                                      |                                                             |
| Brachialis (brʌk'əl-əl'is) | Flexes arm (agonist)                           | P: Distal anterior surface of humerus  
                      |                                                   | D: Tuberosity and coronoid process of ulna               | Musculocutaneous nerve (C5–C6 nerve fibers) |
| Brachioradialis (brʌk'ə-rəd'əl-əl'is) | Flexes forearm                               | P: Lateral supracondylar ridge of humerus  
                      |                                                   | D: Styloid process of radius                             | Radial nerve (C6–C7 nerve fibers) |
| **EXTENSORS (POSTERIOR ARM)** |                                                |                                                      |                                                             |
| Triceps brachii         | Extends forearm (agonist)                      | P: Long head: Infraglenoid tubercle of scapula  
                      |                                                   | Lateral head: Posterior humerus superior to radial groove  
                      |                                                   | Short head: Posterior humerus inferior to radial groove  
                      |                                                   | D: Olecranon of ulna                                     | Radial nerve (C5–C7 nerve fibers) |
| Long head               | Extends and adducts arm (long head only)       |                                                      |                                                             |
| Lateral head            |                                                |                                                      |                                                             |
| Medial head             |                                                |                                                      |                                                             |
| Anconeus (ang-kō'né-əs) | Extends forearm                                | P: Lateral epicondyle of humerus  
                      | ankon = elbow                                        | D: Olecranon of ulna                                       | Radial nerve (C6–C8 nerve fibers) |
| **PRONATORS (ANTERIOR FOREARM MUSCLES)** |                                                |                                                      |                                                             |
| Pronator teres          | Pronates forearm                               | P: Medial epicondyle of humerus and coronoid process of ulna  
                      |                                                   | D: Lateral surface of radius                             | Median nerve (C6–C7 nerve fibers) |
| Pronator quadratus (prō-nä-tōr kwah-drä'tōs) | Pronates forearm                             | P: Distal one-fourth of ulna  
                      |                                                   | D: Distal one-fourth of radius                            | Median nerve (C8–T1 nerve fibers) |
| **SUPINATOR (POSTERIOR FOREARM MUSCLE)** |                                                |                                                      |                                                             |
| Supinator (sū'pi-nä-tōr) | Supinates forearm                             | P: Lateral epicondyle of humerus and ulna distal to radial notch  
                      |                                                   | D: Anterolateral surface of radius distal to radial tuberosity | Radial nerve (C6–C8 nerve fibers) |
Muscle name indicate only a slight effect.

1. Boldface indicates an agonist; others are synergists. Parentheses around an entire muscle name indicate only a slight effect.

<table>
<thead>
<tr>
<th>Table 11.17</th>
<th>Summary of Muscle Actions at the Elbow Joint/Forearm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extension</td>
<td>Flexion</td>
</tr>
<tr>
<td>Triceps brachii</td>
<td>Brachialis</td>
</tr>
<tr>
<td>(Anconeus)</td>
<td>Biceps brachii</td>
</tr>
<tr>
<td>Brachioradialis</td>
<td></td>
</tr>
</tbody>
</table>

1. Boldface indicates an agonist; others are synergists. Parentheses around an entire muscle name indicate only a slight effect.

11.8d Forearm Muscles That Move the Wrist Joint, Hand, and Fingers

**LEARNING OBJECTIVES**

28. Describe the muscles of the anterior compartment and their actions, and identify the layer in which each resides.

29. Explain the actions of the muscles of the posterior compartment, and identify the layer in which each resides.

Most muscles in the forearm move the hand at the wrist, the fingers, or both. These muscles are called extrinsic muscles of the wrist and hand, because the muscles arise from the forearm, not the wrist or hand. Palpate your own forearm; it is bigger near the elbow because the bellies of these forearm muscles form the bulk of this region, whereas distally at the wrist, only long tendons of these muscles are present.

Deep fascia partitions the forearm muscles into an anterior (flexor) compartment and a posterior (extensor) compartment (figure 11.23). Most of the anterior compartment muscles arise from the medial epicondyle of the humerus via a common flexor tendon (figure 11.27). Muscles in the anterior compartment of the forearm generally tend to flex the wrist, the metacarpophalangeal (MP) joints. Some of these also flex the interphalangeal (IP) joints of the fingers. Most of the posterior compartment muscles arise from the lateral epicondyle of the humerus via a common extensor tendon. Muscles in the posterior compartment of the forearm tend to extend the wrist. Some also extend the MP joints and the IP joints.

**Retinacula of the Forearm**

At the wrist, the deep fascia of the forearm forms thickened, fibrous bands termed retinacula (ret-i-nak’u-la; retinao = to hold back). The retinacula help hold the tendons close to the bone and prevent the tendons from “bowstringing” outward. The palmar (anterior) surface of the carpal bones is covered by the flexor retinaculum. Flexor tendons of the digits and the median nerve pass through the tight space between the bones and the flexor retinaculum, which is called the carpal tunnel. The extensor retinaculum (see figure 11.29) is superficial to the dorsal surface of the carpal bones. Extensor tendons of the wrist and digits pass between the bones and the extensor retinaculum.

**Muscles of the Forearm’s Anterior Compartment**

The muscles of the anterior compartment of the forearm may be subdivided into a superficial layer, an intermediate layer, and a deep layer. The muscles of the superficial and intermediate layers arise from the common flexor tendon that attaches to the medial epicondyle of the humerus. The deep layer of muscles arises directly on the bones of the forearm.

Note that not all anterior forearm muscles cause flexion. Both the pronator teres and the pronator quadratus, discussed previously, are located in the anterior compartment of the forearm but their primary function is pronation. Likewise, the supinator muscle is in the posterior compartment of the forearm, yet its primary function is supination.

The superficial layer of anterior forearm muscles is arranged from the lateral to the medial surface of the forearm in the following order: pronator teres (described previously), flexor carpi radialis, palmaris longus, and flexor carpi ulnaris. The flexor carpi radialis tendon is prominent on the lateral side of the forearm. This muscle flexes the wrist and abducts the hand at the wrist. The palmaris longus is absent in some individuals. This narrow, superficial muscle on the anterior surface of the forearm weakly assists in wrist flexion. The flexor carpi ulnaris flexes the wrist and adds the hand at the wrist.

You can determine the positioning of the three superficial muscles of the anterior forearm and the pronator teres muscle on your own body by performing the exercise shown in figure 11.28. Wrap your thumb around the medial epicondyle of the other arm so your thumb is positioned behind the elbow. Align your little finger along the medial border of your forearm. The natural placement of your four fingers overlies the placement of superficial layer muscles.

The intermediate layer in the anterior compartment of the forearm contains a single muscle (figure 11.27b). The flexor digitorum superficialis splits into four tendons, each of which attaches distally to the middle phalanges of fingers 2–5. This muscle crosses over the wrist, MP joints, and PIP (proximal interphalangeal) joints of fingers 2–5; thus, it flexes all of these joints. Since the flexor digitorum superficialis does not cross over the DIP (distal interphalangeal) joints of these fingers, it cannot move the DIP joints.

**CONCEPT CONNECTION**

Because the median nerve (see section 14.5e) passes deep to the flexor retinaculum, this nerve may be pinched within the carpal tunnel. Thus, the musculoskeletal anatomy is related to the proper functioning of selected components of the nervous system.
Figure 11.27 Anterior Forearm Muscles. The anterior forearm muscles pronate the forearm or flex the wrist and fingers. They may be subdivided into superficial, intermediate, and deep layers. (a) Illustration and cadaver photo show the superficial muscles of the right anterior forearm. Illustrations of the (b) intermediate and (c) deep muscles of the right anterior forearm. (c) © McGraw-Hill Education/Christine Eckel
The deep layer of the forearm anterior compartment muscles includes the flexor pollicis longus (lateral side), the flexor digitorum profundus (medial side), and the pronator quadratus (deep) (figure 11.27c). The flexor pollicis longus attaches to the distal phalanx of the thumb and flexes the MP and IP joints of the thumb. In addition, because this muscle crosses the wrist joint, it can weakly flex the wrist. The flexor digitorum profundus lies deep to the flexor digitorum superficialis. This muscle splits into four tendons, which attach distally to the distal phalanges of fingers 2–5. The flexor digitorum profundus flexes the wrist, MP joints, PIP joints, and DIP joints of fingers 2–5.

Muscles of the Forearm’s Posterior Compartment

Muscles of the posterior compartment of the forearm are primarily wrist and finger extensors. An exception is the supinator, which helps supinate the forearm. The posterior compartment muscles may be subdivided into a superficial layer and a deep layer.

The superficial layer of posterior forearm muscles arises from a common extensor tendon on the lateral epicondyle of the humerus (figure 11.29a). These muscles are positioned laterally to medially as follows: The extensor carpi radialis longus is medial to the brachioradialis. It extends the wrist and abducts the hand at the wrist. The extensor carpi radialis brevis works synergistically with the extensor carpi radialis longus. The extensor digitorum splits into four tendons that attach distally to the distal phalanges of fingers 2–5. It extends the wrist, MP joints, PIP joints, and DIP joints of fingers 2–5. The extensor digiti minimi attaches to the distal phalanx of the pinky (finger 5). It works with the extensor digitorum to extend the little finger. On the medial surface of the posterior forearm, the extensor carpi ulnaris attaches distally to the fifth metacarpal bone, where it acts to extend the wrist and adduct the hand.

The deep layer muscles arise directly from the bones of the posterior forearm and attach distally to the wrist or hand (figure 11.29b). These muscles weakly extend the wrist and do the following other functions: (1) The abductor pollicis longus abducts the thumb. (2) The extensor pollicis brevis attaches to the proximal phalanx of the thumb and helps extend the MP joint of the thumb. (3) The extensor pollicis longus attaches distally to the distal phalanx of the thumb, so it extends the MP and IP joints of the thumb. (4) The extensor indicis extends the MP, PIP, and DIP joints of the index finger (finger 2).

Table 11.18 summarizes the characteristics of the muscles that move the wrist joint, hand, and fingers.

### CLINICAL VIEW 11.8

**Carpal Tunnel Syndrome**

The space between the carpal bones and the flexor retinaculum is the carpal tunnel. Numerous finger flexor tendons extend through this tunnel as well as the median nerve, which innervates the skin on the lateral palmar region of the hand and the muscles that move the thumb. Any compression of either the median nerve or the tendons in the tunnel results in carpal tunnel syndrome. The syndrome is characterized by pain and paresthesia (par-eh-thē′zē-ŏ; aisthesis = sensation), which is the feeling of “pins and needles.” Sometimes, more extensive sensory loss, as well as motor loss, occurs in the muscles of the hand supplied by the median nerve.
Figure 11.29 Posterior Forearm Muscles. The posterior forearm muscles supinate the forearm or extend the wrist or fingers. They may be subdivided into (a) superficial and (b) deep layers, as shown in these views of the right forearm.

(a) ©McGraw-Hill Education/Christine Eckel

(b) ©McGraw-Hill Education/Christine Eckel
<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)</th>
<th>Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5e)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexor carpi radialis</td>
<td>Flexes wrist; abducts hand</td>
<td>P: Medial epicondyle of humerus</td>
<td>D: Base of metacarpals II and III</td>
<td>Median nerve (C6–C7 nerve fibers)</td>
</tr>
<tr>
<td>Palmaris longus</td>
<td>Flexes wrist (weakly)</td>
<td>P: Medial epicondyle of humerus</td>
<td>D: Flexor retinaculum and palmar aponeurosis</td>
<td>Median nerve (C6–C7 nerve fibers)</td>
</tr>
<tr>
<td>Flexor carpi ulnaris</td>
<td>Flexes wrist; adducts hand</td>
<td>P: Medial epicondyle of humerus; olecranon and posterior surface of ulna</td>
<td>D: Pisiform and hamate bones; base of metacarpal V</td>
<td>Ulnar nerve (C8–T1)</td>
</tr>
<tr>
<td>Flexor digitorum superficialis</td>
<td>Flexes wrist; flexes 2nd–5th MP joints and PIP joints</td>
<td>P: Medial epicondyle of humerus; coronoid process of ulna</td>
<td>D: Middle phalanges of fingers 2–5</td>
<td>Median nerve (C6–C7 nerve fibers)</td>
</tr>
<tr>
<td>Flexor pollicis longus</td>
<td>Flexes MP joint of thumb, IP joint of thumb; flexes wrist (weakly)</td>
<td>P: Anterior shaft of radius; interosseous membrane</td>
<td>D: Distal phalanx of thumb</td>
<td>Median nerve (C6–C7 nerve fibers)</td>
</tr>
<tr>
<td>Flexor digitorum profundus</td>
<td>Flexes wrist; flexes 2nd–5th MP joints, PIP joints, and DIP joints</td>
<td>P: Anteromedial surface of ulna; interosseous membrane</td>
<td>D: Distal phalanges of fingers 2–5</td>
<td>Lateral one-half of muscle innervated by median nerve (C6–C8 nerve fibers), medial one-half innervated by ulnar nerve (C8 nerve fibers)</td>
</tr>
<tr>
<td>Extensor carpi radialis longus</td>
<td>Extends wrist; abducts hand</td>
<td>P: Lateral supracondylar ridge of humerus</td>
<td>D: Base of metacarpal II</td>
<td>Radial nerve (C6–C7 nerve fibers)</td>
</tr>
<tr>
<td>Extensor carpi radialis brevis</td>
<td>Extends wrist; abducts hand</td>
<td>P: Lateral epicondyle of humerus</td>
<td>D: Base of metacarpal III</td>
<td>Radial nerve (C6–C7 nerve fibers)</td>
</tr>
<tr>
<td>Extensor digitorum</td>
<td>Extends wrist; extends 2nd–5th MP joints, PIP joints and DIP joints</td>
<td>P: Lateral epicondyle of humerus</td>
<td>D: Distal and middle phalanges of fingers 2–5</td>
<td>Radial nerve (C6–C8 nerve fibers)</td>
</tr>
<tr>
<td>Extensor digiti minimi</td>
<td>Extends MP and PIP joints of finger 5; extends wrist (weakly)</td>
<td>P: Lateral epicondyle of humerus</td>
<td>D: Proximal phalanx of finger 5</td>
<td>Radial nerve (C6–C8 nerve fibers)</td>
</tr>
<tr>
<td>Extensor carpi ulnaris</td>
<td>Extends wrist; adducts hand</td>
<td>P: Lateral epicondyle of humerus; posterior border of ulna</td>
<td>D: Base of metacarpal V</td>
<td>Radial nerve (C6–C8 nerve fibers)</td>
</tr>
<tr>
<td>Abductor pollicis longus</td>
<td>Abducts thumb; extends wrist (weakly)</td>
<td>P: Proximal dorsal surfaces of radius and ulna; interosseous membrane</td>
<td>D: Lateral edge of metacarpal I</td>
<td>Radial nerve (C6–C8 nerve fibers)</td>
</tr>
<tr>
<td>Extensor pollicis brevis</td>
<td>Extends MP joint of thumb; extends wrist (weakly)</td>
<td>P: Posterior surface of radius; interosseous membrane</td>
<td>D: Proximal phalanx of thumb</td>
<td>Radial nerve (C6–C8 nerve fibers)</td>
</tr>
<tr>
<td>Extensor pollicis longus</td>
<td>Extends MP and IP joints of thumb; extends wrist (weakly)</td>
<td>P: Posterior surface of ulna; interosseous membrane</td>
<td>D: Distal phalanx of thumb</td>
<td>Radial nerve (C6–C8 nerve fibers)</td>
</tr>
<tr>
<td>Extensor indicis</td>
<td>Extends MP PIP and DIP joints of finger 2; extends wrist (weakly)</td>
<td>P: Posterior surface of ulna; interosseous membrane</td>
<td>D: Tendon of extensor digitorum</td>
<td>Radial nerve (C6–C8 nerve fibers)</td>
</tr>
</tbody>
</table>
INTEGRATE
LEARNING STRATEGY
To remember the functions of the palmar and dorsal interosseous muscles, use this mnemonic phrase: **PAD-DAB**
(Palmar interossei **AD**uct the fingers, whereas **D**orsal interossei **AB**duct the fingers.)
### Table 11.19: Intrinsic Muscles of the Hand

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5e)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>THENAR GROUP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Flexor pollicis brevis | Flexes thumb | P: Flexor retinaculum, trapezium  
D: Proximal phalanx of thumb | Median nerve (C8–T1 nerve fibers) |
| Abductor pollicis brevis | Abducts thumb | P: Flexor retinaculum, scaphoid, trapezium  
D: Lateral side of proximal phalanx of thumb | Median nerve (C8–T1 nerve fibers) |
| Opponens pollicis (0-pōnens) | Opposition of thumb | P: Flexor retinaculum and trapezium  
D: Lateral side of metacarpal I | Median nerve (C8–T1 nerve fibers) |
| **HYPOTHENAR GROUP** | | | |
| Flexor digiti minimi brevis | Flexes finger 5 | P: Hamate bone, flexor retinaculum  
D: Proximal phalanx of finger 5 | Ulnar nerve (C8–T1) |
| Abductor digiti minimi | Abducts finger 5 | P: Pisiform bone, tendon of flexor carpi ulnaris  
D: Proximal phalanx of finger 5 | Ulnar nerve (C8–T1) |
| Opponens digiti minimi | Opposition of finger 5 | P: Hamate bone, flexor retinaculum  
D: Metacarpal bone V | Ulnar nerve (C8–T1) |
| **MIDPALMAR GROUP** | | | |
| Lumbricals (lūm′bri-kālz) | Flexes 2nd–5th MP joints and extends the 2nd–5th PIP and DIP joints | P: Tendons of flexor digitorum profundus  
D: Dorsal tendons on fingers 2–5 | Median nerve (lateral two lumbricals 1, 2) and ulnar nerve (medial two lumbricals 3, 4) |
| Dorsal interossei (dōr′sāl′ in′ter-os′ē-t) | Abducts fingers 2–5; flexes MP joints 2–5 and extends the PIP and DIP joints | P: Adjacent, opposing faces of metacarpals  
D: Dorsal tendons on fingers 2–5 | Ulnar nerve (C8–T1) |
| Palmar interossei | Adducts fingers 2–5; flexes MP joints 2–5 and extends the PIP and DIP joints | P: Metacarpal bones II, IV, V  
D: Sides of proximal phalanges bases for fingers 2, 4, and 5 | Ulnar nerve (C8–T1) |
| Adductor pollicis | Adducts thumb | P: Oblique head: Capitate bone, bases of metacarpals II, III  
Transverse head: Metacarpal III  
D: Medial side of proximal phalanx of thumb | Ulnar nerve (C8–T1) |

### Table 11.20: Summary of Muscle Actions at the Wrist and Hand

<table>
<thead>
<tr>
<th>Hand Abduction</th>
<th>Hand Adduction</th>
<th>Wrist Extension</th>
<th>Wrist Flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexor carpi radialis</td>
<td>Extensor carpi ulnaris</td>
<td>Extensor digitorum</td>
<td>Flexor carpi radialis</td>
</tr>
<tr>
<td>Extensor carpi radialis brevis</td>
<td>Flexor carpi ulnaris</td>
<td>Extensor carpi radialis brevis</td>
<td>Flexor carpi ulnaris</td>
</tr>
<tr>
<td>Extensor carpi radialis longus</td>
<td>Extensor carpi radialis longus</td>
<td>Flexor digitorum superficialis</td>
<td></td>
</tr>
<tr>
<td>(Extensor indicis)1</td>
<td>Extensor carpi ulnaris</td>
<td>Flexor digitorum profundus</td>
<td>(Palmaris longus)</td>
</tr>
<tr>
<td>(Extensor pollicis longus)</td>
<td>(Extensor pollicis brevis)</td>
<td>(Flexor pollicis longus)</td>
<td></td>
</tr>
<tr>
<td>(Abductor pollicis longus)</td>
<td>(Extensor digiti minimi)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Finger Abduction</th>
<th>Finger Adduction</th>
<th>MP/IP Joint Extension</th>
<th>MP/IP Joint Flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dorsal interossei</td>
<td>Palmar interossei</td>
<td>Extensor digitorum (MP and IP)</td>
<td>Flexor digitorum profundus (MP, PIP and DIP)</td>
</tr>
<tr>
<td>Abductor pollicis longus</td>
<td>Adductor pollicis</td>
<td>Extensor indicis (MP and IP)</td>
<td>Flexor digitorum superficialis (MP and PIP only)</td>
</tr>
<tr>
<td>Abductor pollicis brevis</td>
<td>Extensor pollicis brevis (MP)</td>
<td>Flexor pollicis longus (MP and IP)</td>
<td></td>
</tr>
<tr>
<td>Abductor digiti minimi</td>
<td>Extensor pollicis longus (MP and IP)</td>
<td>Flexor pollicis brevis (MP)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Extensor digiti minimi (MP and IP)</td>
<td>Flexor digiti minimi (MP)</td>
<td></td>
</tr>
<tr>
<td>Lumbricals (IP)</td>
<td>Dorsal interossei (IP)</td>
<td>Lumbricals (MP)</td>
<td></td>
</tr>
<tr>
<td>Dorsal interossei (IP)</td>
<td>Palmar interossei (IP)</td>
<td>Dorsal interossei (MP)</td>
<td></td>
</tr>
</tbody>
</table>

1. Parentheses around a muscle name indicate only a slight effect. MP = metacarpophalangeal joint; IP = interphalangeal joint
The specific thenar, hypothenar, and midpalmar muscles that control movements of the fingers are described in detail in table 11.19 and grouped according to common muscle actions in table 11.20.

**WHAT DID YOU LEARN?**

Which group of intrinsic hand muscles is primarily responsible for moving the thumb?

### 11.9 Muscles of the Pelvic Girdle and Lower Limb

The most powerful and largest muscles in the body are those of the lower limb. These muscles support the weight of the body and move the lower limbs during locomotion. The lower limb muscles also are organized in compartments like the upper limb (see figure 11.23).

As with the muscles of the pectoral girdle and upper limb, the pelvic girdle and lower limb muscles can be organized into groups:

- Muscles that move the hip joint/thigh
- Thigh muscles that move the knee joint/leg
- Muscles of the leg that move the ankle, foot, and toes
- Intrinsic muscles of the foot

#### 11.9a Muscles That Move the Hip Joint/Thigh

**LEARNING OBJECTIVES**

31. Compare and contrast the functions of the muscles in the anterior, medial, lateral, and posterior compartments of the thigh.

32. Describe the actions of the three gluteal muscles.

Note that in the subsequent discussion the phrases “moving the hip joint” and “moving the thigh” mean the same thing. The *fascia lata*, the deep fascia of the thigh, encircles the thigh muscles like a supportive stocking and tightly binds them. The fascia lata partitions the thigh muscles into compartments, each with its own blood and nerve supply. The anterior compartment muscles flex the thigh or extend the knee. The muscles of the medial compartment adduct the thigh. The single muscle in the lateral compartment abducts the thigh. Most muscles of the posterior compartment act as both extensors of the thigh and flexors of the knee. Some of these muscles also abduct the thigh. We discuss the muscles that move the thigh first; those that move the knee are discussed in section 11.9b.

Multiple muscles attach to the anterior thigh and flex the hip joint (figure 11.31a): The *psoas major* and the *iliacus* originate on the lumbar vertebrae and ilium, respectively, but they share the distal attachment at the lesser trochanter of the femur. Because the two muscles merge and attach to the femur, they are collectively referred to as the *iliopsoas*. Together, these muscles work synergistically to flex the thigh. The *rectus femoris* and a long, thin muscle called the *sartorius* flex the thigh. Both are examined in section 11.9b with the thigh muscles that move the leg.

Six muscles are located in the **medial compartment** of the thigh. Most of these muscles adduct the thigh, and some of them perform additional functions. The *adductor longus*, *adductor brevis*, *adductor magnus*, *gracilis*, and *pectineus* also flex the thigh. The adductor magnus also extends and laterally rotates the thigh. The *obturator externus* does not adduct the thigh, but it laterally rotates the thigh.

On the lateral thigh is a single muscle called the *tensor fasciae latae* (figure 11.31b). It attaches to a lateral thickening of the fascia lata, called the *iliotibial tract* (or *iliotibial band*), which extends from the iliac crest to the lateral condyle of the tibia. The tensor fasciae latae abducts and medially rotates the thigh.

The posterior muscles that move the thigh include gluteal muscles and the “hamstring” muscle group. The *gluteus maximus* is the largest of the three gluteal muscles; it is the chief extensor of the thigh and it laterally rotates the thigh. Deep to the gluteus maximus are the *gluteus medius* and *gluteus minimus*, which abduct and medially rotate the thigh (figure 11.31c).

Deep to the gluteal muscles are a group of muscles that collectively laterally rotate the thigh, as when the legs are crossed with one ankle resting on the knee. These muscles are organized from superior to inferior within the posterior thigh as the *psoas major*, *iliacus*, *obturator internus*, *inferior gemellus*, and *quadratus femoris*.

Finally, the posterior thigh contains a group of muscles that are collectively referred to as the *hamstrings* because a ham is strung up by these muscles while being smoked. The hamstring muscles are the *biceps femoris*, *semimembranosus*, and *semitendinosus*. These muscles share a common proximal attachment on the ischial tuberosity of the os coxae, and they attach distally on the leg. Thus, these muscles move both the thigh and the knee. Their primary thigh movement is extension. These muscles are discussed again in section 11.9b.

Table 11.21 summarizes the characteristics of the muscles that move the hip joint and thigh, and table 11.22 groups these muscles according to their common actions on the thigh.
Figure 11.31 Muscles That Act on the Hip and Thigh. (a) Anterior, (b) lateral, and (c) deep posterior views of the right thigh. Most muscles that act on the thigh (femur) originate from the os coxae. (Obturator externus not shown.)

- Psoas minor
- Psoas major
- Iliacus
- Iliopsoas
- Pectineus
- Adductor longus
- Adductor brevis
- Adductor magnus
- Iliac crest
- Tensor fasciae latae
- Sartorius
- Rectus femoris
- Gluteus medius
- Gluteus maximus
- Vastus lateralis
- Iliotibial tract
- Biceps femoris, long head
- Semimembranosus
- Biceps femoris, short head
- Patella
- Gastrocnemius
- Gracilis
- Adductor magnus
- Semitendinosus
- Biceps femoris, long head
- Iliotibial tract
- Gluteus medius (cut)
- Gluteus minimus
- Gluteus medius (cut)
- Gluteus maximus (cut)
- Quadratus femoris
- Iliac crest
- Sacrum
- Gluteus maximus (cut)
- Obturator internus
- Superior gemellus
- Inferior gemellus
- Ischial tuberosity
- Gracilis

(c) Right thigh, deep posterior view

422 Chapter Eleven Muscular System: Axial and Appendicular Muscles
### Muscles That Move the Hip Joint/Thigh

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)</th>
<th>Distal Attachment(s) (D)</th>
<th>Innervation (see sections 14.5f, g)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ANTERIOR THIGH COMPARTMENT (THIGH FLEXORS)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psoas major</td>
<td>Flexes thigh</td>
<td>P: Transverse processes and bodies of T12–L5 vertebrae</td>
<td>Branches of lumbar plexus (L2–L3)</td>
<td></td>
</tr>
<tr>
<td>(sō’as) psoa = loin muscle</td>
<td></td>
<td>D: Lesser trochanter of femur with iliacus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iliacus</td>
<td>Flexes thigh</td>
<td>P: Iliac fossa</td>
<td>Femoral nerve (L2–L3 nerve fibers)</td>
<td></td>
</tr>
<tr>
<td>(ī-lī’ā-kūs) iliac = ilium</td>
<td></td>
<td>D: Lesser trochanter of femur with psoas major</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sartorius</td>
<td>Flexes thigh and laterally rotates thigh; flexes leg and medially rotates leg</td>
<td>P: Anterior superior iliac spine</td>
<td>Femoral nerve (L2–L3 nerve fibers)</td>
<td></td>
</tr>
<tr>
<td>(sar-tō-r′ē-ū-s) sartor = tailor</td>
<td></td>
<td>D: Tibial tuberosity, medial side</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>Flexes thigh; extends leg</td>
<td>P: Anterior inferior iliac spine</td>
<td>Femoral nerve (L2–L4)</td>
<td></td>
</tr>
<tr>
<td>(fem′ō-ris)</td>
<td></td>
<td>D: Quadriceps tendon to patella and then patellar ligament to tibial tuberosity</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MEDIAL THIGH COMPARTMENT (THIGH ADDUCTORS)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adductor longus</td>
<td>Adducts thigh; flexes thigh</td>
<td>P: Pubis near pubic symphysis</td>
<td>Obturator nerve (L2–L4)</td>
<td></td>
</tr>
<tr>
<td>Adductor brevis</td>
<td>Adducts thigh; flexes thigh</td>
<td>P: Inferior ramus and body of pubis</td>
<td>Obturator nerve (L2–L3 nerve fibers)</td>
<td></td>
</tr>
<tr>
<td>Adductor magnus</td>
<td>Adducts thigh; flexes thigh (adductor part); extends thigh (hamstring part); laterally rotates thigh</td>
<td>P: Inferior ramus of pubis and ischial tuberosity</td>
<td>Adductor part: Obturator nerve (L2–L4) hamstring part: Tibial division of sciatic nerve (L2–L4 nerve fibers)</td>
<td></td>
</tr>
<tr>
<td>(mag’nūs) magnus = large</td>
<td></td>
<td>D: Upper third of linea aspera of femur</td>
<td>Adductor part: Obturator nerve (L2–L4) hamstring part: Tibial division of sciatic nerve (L2–L4 nerve fibers)</td>
<td></td>
</tr>
<tr>
<td>Gracilis</td>
<td>Adducts thigh; flexes thigh; flexes leg</td>
<td>P: Inferior ramus and body of pubis</td>
<td>Obturator nerve (L2–L4)</td>
<td></td>
</tr>
<tr>
<td>(gras′i-lis) gracilis = slender</td>
<td></td>
<td>D: Upper medial surface of tibia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pectineus</td>
<td>Adducts thigh; flexes thigh</td>
<td>P: Pectineal line of pubis</td>
<td>Femoral nerve (L2–L4) or obturator nerve (L2–L4)</td>
<td></td>
</tr>
<tr>
<td>(pek′ti-nē-ūs) pectin = comb</td>
<td></td>
<td>D: Pectineal line of femur</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obturator externus</td>
<td>Laterally rotates thigh</td>
<td>P: Margins of obturator foramen and obturator membrane</td>
<td>Obturator nerve (L3–L4 nerve fibers)</td>
<td></td>
</tr>
<tr>
<td>(ob′tū-rā-tōr eks-ter′nūs) obturator = any structure that occludes an opening</td>
<td></td>
<td>D: Trochanteric fossa of posterior femur</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>LATERAL THIGH COMPARTMENT (THIGH ABDUCTOR)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tensor fasciae latae</td>
<td>Abducts thigh; medially rotates thigh</td>
<td>P: Iliac crest and lateral surface of anterior superior iliac spine</td>
<td>Superior gluteal nerve (L4–S1)</td>
<td></td>
</tr>
<tr>
<td>(ten′sōr fas′hā lā′tē) tensor = to make tense fascia = band lata = wide</td>
<td></td>
<td>D: Iliotibial band</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>GLUTEAL GROUP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gluteus maximus</td>
<td>Extends thigh; laterally rotates thigh</td>
<td>P: Iliac crest, sacrum, coccyx</td>
<td>Inferior gluteal nerve (L5–S2)</td>
<td></td>
</tr>
<tr>
<td>(glū-tē′ūs mak′si-mūs) glutos = buttock maximus = largest</td>
<td></td>
<td>D: Iliotibial tract of fascia lata; linea aspera and gluteal tuberosity of femur</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gluteus medius</td>
<td>Abducts thigh; medially rotates thigh</td>
<td>P: Posterior iliac crest; lateral surface between posterior and anterior gluteal lines</td>
<td>Superior gluteal nerve (L4–S1)</td>
<td></td>
</tr>
<tr>
<td>(me′dē-ūs) medius = middle</td>
<td></td>
<td>D: Greater trochanter of femur</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gluteus minimus</td>
<td>Abducts thigh; medially rotates thigh</td>
<td>P: Lateral surface of ilium between inferior and anterior gluteal lines</td>
<td>Superior gluteal nerve (L4–S1)</td>
<td></td>
</tr>
<tr>
<td>(min-i-mūs) minimus = smallest</td>
<td></td>
<td>D: Greater trochanter of femur</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(continued on next page)
### Table 11.21

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see sections 14.5f, g)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DEEP MUSCLES OF THE GLUTEAL REGION (LATERAL THIGH ROTATORS)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Piriformis (pir′i-fŏr′mis)</td>
<td>Laterally rotates thigh</td>
<td>P: Anterolateral surface of sacrum  D: Greater trochanter</td>
<td>Nerve to piriformis (S1–S2)</td>
</tr>
<tr>
<td><em>pirum</em> = pear  <em>forma</em> = form</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior gemellus (jē-mel′ŭs)</td>
<td>Laterally rotates thigh</td>
<td>P: Ischial spine and tuberosity  D: Greater trochanter</td>
<td>Nerve to obturator internus (L5–S1)</td>
</tr>
<tr>
<td><em>gemin</em> = twin, double</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obturator internus (in-ter′nŭs)</td>
<td>Laterally rotates thigh</td>
<td>P: Posterior surface of obturator membrane; margins of obturator foramen  D: Greater trochanter</td>
<td>Nerve to obturator internus (L5–S1)</td>
</tr>
<tr>
<td>Inferior gemellus</td>
<td>Laterally rotates thigh</td>
<td>P: Ischial tuberosity  D: Obturator internus tendon</td>
<td>Nerve to quadratus femoris (L5–S1)</td>
</tr>
<tr>
<td>Quadratus femoris</td>
<td>Laterally rotates thigh</td>
<td>P: Lateral border of ischial tuberosity  D: Intertrochanteric crest of femur</td>
<td>Nerve to quadratus femoris (L5–S1)</td>
</tr>
<tr>
<td><strong>HAMSTRING GROUP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>Extends thigh (long head only); flexes leg (both long head and short head); laterally rotates leg</td>
<td>P: Long head: Ischial tuberosity  Short head: Linea aspera of femur  D: Head of fibula</td>
<td>Long head: Tibial division of sciatic nerve (L4–S1 nerve fibers)  Short head: Common fibular division of sciatic nerve (L5–S1 nerve fibers)</td>
</tr>
<tr>
<td><em>Long head</em>  <em>Short head</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Semimembranosus (sem′ĭ-mem-brā-no-sŭs)</td>
<td>Extends thigh; flexes leg; medially rotates leg</td>
<td>P: Ischial tuberosity  D: Posterior surface of medial condyle of tibia</td>
<td>Tibial division of sciatic nerve (L4–S1 nerve fibers)</td>
</tr>
<tr>
<td><em>semi</em> = half  <em>membranosus</em> = membrane</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Semitendinosus (sem′ĭ-ten-di-no-sŭs)</td>
<td>Extends thigh; flexes leg; medially rotates leg</td>
<td>P: Ischial tuberosity  D: Proximal medial surface of tibia</td>
<td>Tibial division of sciatic nerve (L4–S1 nerve fibers)</td>
</tr>
<tr>
<td><em>tendinosus</em> = tendon</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 11.22

**Summary of Muscle Actions at the Hip Joint/Thigh**

<table>
<thead>
<tr>
<th>Abduction</th>
<th>Adduction</th>
<th>Extension</th>
<th>Flexion</th>
<th>Lateral Rotation</th>
<th>Medial Rotation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gluteus medius</td>
<td>Adductor brevis, longus, magnus</td>
<td>Gluteus maximus&lt;sup&gt;1&lt;/sup&gt;</td>
<td>Iliopsoas</td>
<td>Adductor magnus (hamstring part)</td>
<td>Gluteus medius</td>
</tr>
<tr>
<td>Gluteus minimus</td>
<td>Gracilis</td>
<td>Adductor magnus (hamstring part)</td>
<td>Adductor brevis, longus, magnus (adductor part)</td>
<td>Gluteus maximus</td>
<td>Gluteus minimus</td>
</tr>
<tr>
<td>Tensor fasciae latae</td>
<td>Pectineus</td>
<td>Biceps femoris (long head)</td>
<td>Pectineus</td>
<td>Sartorius</td>
<td>Tensor fasciae latae</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Semimembranosus</td>
<td>Sartorius</td>
<td>Obturator externus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Semitendinosus</td>
<td>Rectus femoris</td>
<td>Obturator internus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Gracilis</td>
<td>Piriformis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Superior gemellus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Inferior gemellus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Quadratus femoris</td>
<td></td>
</tr>
</tbody>
</table>

<sup>1</sup> Boldface indicates an agonist; others are synergists.
What Did You Learn?

List the compartments of the thigh, and describe the common action of the muscles in each.

11.9b Thigh Muscles That Move the Knee Joint/Leg

Learning Objectives

33. List the muscles of the thigh's anterior compartment that move the knee joint.
34. Describe the muscles of the thigh that flex the knee joint.

The muscles that act on the knee form most of the mass of the thigh. They include muscles of the anterior compartment and posterior compartment of the thigh, as well as certain muscles already described in section 11.9a.

Muscles of the Thigh’s Anterior Compartment

The anterior (extensor) compartment of the thigh is composed of the large quadriceps femoris, the prime mover of knee extension (figure 11.32). The quadriceps femoris is a composite muscle with four heads: the rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius. All four muscles converge on a single quadriceps tendon, which extends to the patella and then continues inferiorly as the patellar ligament and attaches distally to the tibial tuberosity.

Also within the anterior compartment is the sartorius, which acts on both the hip and knee joints, flexing and laterally rotating the thigh while flexing and medially rotating the leg. This muscle is the longest in the body and is termed the tailor’s muscle because it helps us sit cross-legged, as tailors used to do.

Muscles of the Thigh’s Medial Compartment

The gracilis muscle (in the medial compartment of the thigh) not only adducts and flexes the thigh, as described in section 11.9a, but also flexes the leg, since it spans the knee joint.

Muscles of the Thigh’s Posterior Compartment

The posterior (flexor) compartment of the thigh contains the three hamstring muscles discussed previously (figure 11.33). These muscles also flex the leg. The biceps femoris is a two-headed muscle that attaches distally to the lateral side of the leg. This muscle also can laterally rotate the leg when the leg is flexed. The semimembranosus and semitendinosus attach distally to the medial side of the leg. The semimembranosus and semitendinosus also medially rotate the leg when the leg is flexed.

Finally, several leg muscles span the knee joint and flex the leg. These muscles (gastrocnemius, plantaris, and popliteus) are discussed in section 11.9c, as we examine muscles of the leg.

Table 11.23 summarizes the characteristics of the thigh muscles that move the knee joint and leg.

What Did You Learn?

List the thigh muscles that flex the knee joint.
Figure 11.32  Muscles of the Anterior Thigh.  Muscles of the anterior thigh flex the thigh and extend the leg.  (a) Illustration and cadaver photo show an anterior view of the right thigh.  (b) Individual muscles of the right anterior thigh.  © McGraw-Hill Education/Christine Eckel
Figure 11.33 Muscles of the Gluteal Region and Posterior Thigh. Muscles of the posterior thigh extend the thigh and flex the leg. (a) Illustration and cadaver photo show the gluteal and posterior muscles of the right thigh. (b) Individual muscles that extend the thigh are shown in bold (note the short head of biceps femoris does not participate in thigh extension).
### Table 11.23 Thigh Muscles That Move the Knee Joint/Leg

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5f)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LEG EXTENSORS (ANTERIOR THIGH MUSCLES)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Rectus femoris</strong></td>
<td>Extends leg; flexes thigh</td>
<td>P: Anterior inferior iliac spine</td>
<td>Femoral nerve (L2–L4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Quadriceps tendon to patella and then patellar ligament to tibial tuberosity</td>
<td></td>
</tr>
<tr>
<td><strong>Vastus intermedius</strong> <em>(vas’tūs in-ter-mē’dē-ūs)</em></td>
<td>Extends leg</td>
<td>P: Anterolateral surface of femur</td>
<td>Femoral nerve (L2–L4)</td>
</tr>
<tr>
<td><em>(vastus = great)</em> <em>(intermedius = intermediate)</em></td>
<td></td>
<td>D: Quadriceps tendon to patella and then patellar ligament to tibial tuberosity</td>
<td></td>
</tr>
<tr>
<td><strong>Vastus lateralis</strong> <em>(lat-er-ā’līs)</em></td>
<td>Extends leg</td>
<td>P: Greater trochanter and linea aspera</td>
<td>Femoral nerve (L2–L4)</td>
</tr>
<tr>
<td><em>(vastus = great)</em> <em>(lateralis = lateral)</em></td>
<td></td>
<td>D: Quadriceps tendon to patella and then patellar ligament to tibial tuberosity</td>
<td></td>
</tr>
<tr>
<td><strong>Vastus medialis</strong> <em>(mē-dē-ā’līs)</em></td>
<td>Extends leg</td>
<td>P: Intertrochanteric line and linea aspera of femur</td>
<td>Femoral nerve (L2–L4)</td>
</tr>
<tr>
<td><em>(medialis = medial)</em></td>
<td></td>
<td>D: Quadriceps tendon to patella and then patellar ligament to tibial tuberosity</td>
<td></td>
</tr>
<tr>
<td><strong>LEG FLEXORS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sartorius</strong></td>
<td>Flexes thigh and rotates thigh laterally; flexes leg and rotates leg medially</td>
<td>See table 11.21</td>
<td>See table 11.21</td>
</tr>
<tr>
<td><strong>Gracilis</strong></td>
<td>Adducts and flexes thigh; flexes leg</td>
<td>See table 11.21</td>
<td>See table 11.21</td>
</tr>
<tr>
<td><strong>Hamstrings (biceps femoris, semimembranosus, semitendinosus)</strong></td>
<td>Extend thigh; flex leg; laterally rotates leg (biceps femoris); medially rotate leg (semimembranosus and semitendinosus)</td>
<td>See table 11.21</td>
<td>See table 11.21</td>
</tr>
</tbody>
</table>

### 11.9c Leg Muscles That Move the Ankle, Foot, and Toes

**LEARNING OBJECTIVES**

35. Compare and contrast the muscles of the three compartments of the leg and their actions.

36. Distinguish between the muscles of the superficial layer and deep layer of the leg's posterior compartment.

The muscles that move the ankle, foot, and toes are housed within the leg and are called the **crural muscles**. Some of these muscles also help flex the leg. The deep fascia partitions the leg musculature into three compartments (anterior, lateral, and posterior), each with its own blood supply and innervation, and muscles in the same compartment tend to share common functions (see figure 11.23).
Muscles of the Leg’s Anterior Compartment

Anterior compartment leg muscles dorsiflex the foot, extend the toes, or both (figure 11.34). The extensor digitorum longus dorsiflexes the foot and extends toes 2–5. The extensor hallucis longus sends a tendon to the dorsum of the great toe (hallux), and so it dorsiflexes the foot and extends the great toe. The fibularis tertius (peroneus tertius) dorsiflexes and weakly everts the foot. The tibialis anterior is the primary dorsiflexor of the foot. This muscle attaches to the medial plantar side of the foot, so it also inverts the foot. Analogous to tendons of the wrist, tendons of the muscles within the anterior compartment are held tightly against the ankle by multiple deep fascia thickenings, collectively referred to as extensor retinaculum.

Muscles of the Leg’s Lateral Compartment

The lateral compartment leg muscles contain two synergistic muscles that are very powerful plantar flexors (figure 11.35). The long, flat fibularis longus (peroneus brevis) attaches distally to the plantar side of the foot. The fibularis brevis (peroneus brevis) lies deep to the fibularis longus, and its tendon attaches distally to the base of the fifth metatarsal.

Muscles of the Leg’s Posterior Compartment

The posterior compartment of the leg is composed of seven muscles separated into superficial and deep groups (figure 11.36). The superficial muscles and most of the deep muscles plantar flex the foot at the ankle.

The superficial layer of the posterior compartment contains the gastrocnemius, soleus, and plantaris. The gastrocnemius (gas’tro-kne’mi-əs; gastr = belly, kneme = leg) has two thick muscle bellies that form the prominence on the posterior part of the leg often referred to as the “calf.” This muscle spans both the knee and the ankle joints; it flexes the leg and plantar flexes the foot. The soleus is a broad, flat muscle deep to the gastrocnemius (solea = sandal). This muscle plantar flexes the foot. The gastrocnemius and soleus are collectively known as the triceps surae, and together they are the most powerful plantar flexors of all the leg muscles. These two muscles share a common tendon of distal attachment, the calcaneal tendon (Achilles tendon). The plantaris is a small muscle that is absent in some individuals. It is a weak leg flexor and plantar flexor of the foot.

The deep layer of the posterior compartment contains four muscles. The flexor digitorum longus attaches to the distal phalanges of toes 2–5, plantar flexes the foot, and flexes the MP, PIP, and DIP joints of these toes. The flexor hallucis longus plantar flexes the foot and flexes the MP and IP joints of the great toe. The tibialis posterior is the deepest of the posterior compartment muscles. It plantar flexes and inverts the foot. The popliteus flexes the leg and medially rotates the tibia slightly to “unlock” the fully extended knee joint. This muscle’s attachments are within the popliteal region, so it only moves the knee, not the foot.

Table 11.24 summarizes the characteristics of the muscles that move the leg. Table 11.25 groups muscles according to their action on the leg. Note that many thigh and leg muscles are involved with leg flexion.

Table 11.24

<table>
<thead>
<tr>
<th>Muscle Name</th>
<th>Action on Leg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibularis longus</td>
<td>Dorsiflexing</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>Inverting</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>Plantar flexing</td>
</tr>
<tr>
<td>Tibialis brevis</td>
<td>Plantar flexing</td>
</tr>
<tr>
<td>Extensor digitorum longus</td>
<td>Plantar flexing</td>
</tr>
<tr>
<td>Extensor hallucis longus</td>
<td>Plantar flexing</td>
</tr>
<tr>
<td>Extensor retinaculum</td>
<td></td>
</tr>
<tr>
<td>Extensor digitorum brevis</td>
<td></td>
</tr>
</tbody>
</table>

Figure 11.34 Muscles of the Anterior Leg. The anterior muscles of the leg dorsiflex the foot and extend the toes. (a) The illustration shows an anterior view of the right leg. (b) Individual muscles of the right anterior leg.
Figure 11.35 Muscles of the Lateral Leg. (a) Illustration and cadaver photo show a lateral view of the right leg. (b) The fibularis longus and the fibularis brevis evert and plantar flex the foot. (c) ©McGraw-Hill Education/Christine Eckel
Figure 11.36 Muscles of the Posterior Leg. The posterior muscles of the leg plantar flex the foot and flex the toes. (a) Superficial and (b) deep views of the posterior right leg. (c) Selected individual muscles of the deep posterior compartment.
### Table 11.24

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5g)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ANTERIOR COMPARTMENT (DORSIFLEXORS AND TOE EXTENDERS)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extensor digitorum longus</td>
<td>Dorsiflexes foot; extends toes 2–5</td>
<td>P: Lateral condyle of tibia; anterior surface of fibula; interosseous membrane D: Distal phalanges of toes 2–5</td>
<td>Deep fibular nerve (L4–S1)</td>
</tr>
<tr>
<td>Extensor hallucis longus</td>
<td>Dorsiflexes foot; extends great toe (1)</td>
<td>P: Anterior surface of fibula; interosseous membrane D: Distal phalanx of great toe (1)</td>
<td>Deep fibular nerve (L4–S1)</td>
</tr>
<tr>
<td>Fibularis tertius</td>
<td>Dorsiflexes foot; everts foot (weakly)</td>
<td>P: Anterior distal surface of fibula; interosseous membrane D: Base of metatarsal V</td>
<td>Deep fibular nerve (L5–S1 fibers)</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>Dorsiflexes foot; inverts foot (agonist)</td>
<td>P: Lateral condyle and proximal shaft of tibia; interosseous membrane D: Metatarsal I and 1st (medial) cuneiform</td>
<td>Deep fibular nerve (L4–S1)</td>
</tr>
<tr>
<td><strong>LATERAL COMPARTMENT (EVERTORS AND PLANTAR FLEXORS)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibularis longus</td>
<td>Everts foot (agonist); plantar flexes (weakly)</td>
<td>P: Head and upper two-thirds of shaft fibula; lateral condyle of tibia D: Base of metatarsal I; medial cuneiform</td>
<td>Superficial fibular nerve (L5–S2)</td>
</tr>
<tr>
<td>Fibularis brevis</td>
<td>Everts foot (agonist); plantar flexes (weakly)</td>
<td>P: Midlateral shaft of fibula D: Base of metatarsal V</td>
<td>Superficial fibular nerve (L5–S2)</td>
</tr>
<tr>
<td><strong>POSTERIOR COMPARTMENT (PLANTAR FLEXORS, FLEXORS OF THE LEG AND TOES)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Superficial layer</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>Flexes leg; plantar flexes foot (agonist)</td>
<td>P: Superior and posterior surfaces of lateral and medial condyles of femur D: Calcaneus (via calcaneal tendon)</td>
<td>Tibial nerve (L4–S1 nerve fibers)</td>
</tr>
<tr>
<td>Soleus</td>
<td>Plantar flexes foot (agonist)</td>
<td>P: Head and proximal shaft of fibula; medial border of tibia D: Calcaneus (via calcaneal tendon)</td>
<td>Tibial nerve (L4–S1 nerve fibers)</td>
</tr>
<tr>
<td>Plantaris (plantar′is)</td>
<td>Plantar flexes foot</td>
<td>P: Lateral supracondylar ridge of femur D: Posterior region of calcaneus</td>
<td>Tibial nerve (L4–S1 nerve fibers)</td>
</tr>
<tr>
<td><strong>Deep layer</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor digitorum longus</td>
<td>Plantar flexes foot; flexes MP, PIP, and DIP joints of toes 2–5</td>
<td>P: Posteromedial surface of tibia D: Distal phalanges of toes 2–5</td>
<td>Tibial nerve (L5–S1 nerve fibers)</td>
</tr>
<tr>
<td>Flexor hallucis longus</td>
<td>Plantar flexes foot; flexes MP and IP joints of great toe (1)</td>
<td>P: Posterior lower two-thirds of fibula D: Distal phalanges of great toe (1)</td>
<td>Tibial nerve (L5–S1 nerve fibers)</td>
</tr>
<tr>
<td>Tibialis posterior</td>
<td>Plantar flexes foot; inverts foot (agonist)</td>
<td>P: Fibula, tibia, and interosseous membrane D: Metatarsals II–IV; navicular bone; cuboid bone; all cuneiforms</td>
<td>Tibial nerve (L5–S1 nerve fibers)</td>
</tr>
<tr>
<td>Popliteus (pop-li-tē′ús)</td>
<td>Flexes leg; mediually rotates tibia to unlock the knee</td>
<td>P: Lateral condyle of femur D: Posterior, proximal surface of tibia</td>
<td>Tibial nerve (L4–L5 nerve fibers)</td>
</tr>
</tbody>
</table>

### Table 11.25

**Summary of Muscle Actions at the Knee Joint/Leg**

<table>
<thead>
<tr>
<th>Extension</th>
<th>Flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quadriceps femoris</td>
<td>Sartorius</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>Gracilis</td>
</tr>
<tr>
<td>Vastus lateralis</td>
<td>Biceps femoris</td>
</tr>
<tr>
<td>Vastus intermedius</td>
<td>Semimembranosus</td>
</tr>
<tr>
<td>Vastus medialis</td>
<td>Gastrocnemius</td>
</tr>
<tr>
<td>Popliteus (Plantaris)</td>
<td></td>
</tr>
</tbody>
</table>

1. Parentheses around an entire muscle name indicate only a slight effect.
11.9d Intrinsic Muscles of the Foot

**LEARNING OBJECTIVE**

37. Identify the muscles of each group and their actions.

The intrinsic muscles of the foot have both proximal and distal attachments within the foot. They support the arches and move the toes to aid locomotion. Most of these muscles are comparable to the intrinsic muscles of the hand, meaning that they have similar names and locations. However, the intrinsic muscles of the foot rarely perform all the precise movements their names suggest.

The intrinsic foot muscles form a dorsal group and a plantar group. The dorsal group contains only two muscles: the extensor hallucis brevis and the extensor digitorum brevis (see figure 11.34). The extensor hallucis brevis extends the MP joint of the great toe, whereas the extensor digitorum brevis extends the MP and PIP joints of toes 2–4.

**WHAT DO YOU THINK?**

4 The extensor digitorum brevis only goes to toes 2–4, so how is it possible to extend your little toe (toe 5)?

The plantar surface of the foot is supported by the plantar aponeurosis formed from the deep fascia of the foot. This aponeurosis extends between the phalanges of the toes and the calcaneus. It also encloses the plantar muscles of the foot. The plantar muscles are grouped into four layers (figure 11.37) and are described in detail in table 11.26.

Table 11.27 groups the leg and intrinsic foot muscles according to their common actions on the foot.

**WHAT DID YOU LEARN?**

29 Identify the intrinsic muscles of the foot that extend the toes.
### Table 11.26  Intrinsic Muscles of the Foot

<table>
<thead>
<tr>
<th>Group/Muscle</th>
<th>Action(s)</th>
<th>Proximal Attachment(s) (P)/Distal Attachment(s) (D)</th>
<th>Innervation (see section 14.5g)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DORSAL SURFACE (TOE EXTENSORS)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extensor hallucis brevis</td>
<td>Extends MP joint of great toe (1)</td>
<td>P: Calcaneus and inferior extensor retinaculum</td>
<td>Deep fibular nerve (S1–S2 nerve fibers)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Proximal phalanx of great toe (1)</td>
<td></td>
</tr>
<tr>
<td>Extensor digitorum brevis</td>
<td>Extends MP and PIP joints of toes 2–4</td>
<td>P: Calcaneus and inferior extensor retinaculum</td>
<td>Deep fibular nerve (S1–S2 nerve fibers)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Middle phalanges of toes 2–4</td>
<td></td>
</tr>
<tr>
<td><strong>PLANTAR SURFACE (TOE FLEXORS, ABDUCTORS, ADDUCTORS)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Layer 1 (superficial)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor digitorum brevis</td>
<td>Flexes MP and PIP joints of toes 2–5</td>
<td>P: Calcaneus</td>
<td>Medial plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Middle phalanges of toes 2–5</td>
<td></td>
</tr>
<tr>
<td>Abductor hallucis</td>
<td>Abducts great toe (1)</td>
<td>P: Calcaneus</td>
<td>Medial plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Medial side of proximal phalanx of great toe (1)</td>
<td></td>
</tr>
<tr>
<td>Abductor digitii minimi</td>
<td>Abducts toe 5</td>
<td>P: Calcaneus (inferior surface tuberosity)</td>
<td>Lateral plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Lateral side of proximal phalanx of toe 5</td>
<td></td>
</tr>
<tr>
<td>Layer 2 (deep)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quadratus plantae</td>
<td>Pulls on flexor digitorum longus tendons to flex toes 2–5</td>
<td>P: Calcaneus, long plantar ligament</td>
<td>Lateral plantar nerve (S2–S3)</td>
</tr>
<tr>
<td><em>planta</em> = sole of foot</td>
<td></td>
<td>D: Tendons of flexor digitorum longus</td>
<td></td>
</tr>
<tr>
<td>Lumbricals</td>
<td>Flexes MP joints and extends PIP and DIP joints of toes 2–5</td>
<td>P: Tendons of flexor digitorum longus</td>
<td>Medial plantar nerve (1st lumbrical); lateral plantar nerve (2nd–4th lumbricals)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Tendons of extensor digitorum longus</td>
<td></td>
</tr>
<tr>
<td>Layer 3 (deeper)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adductor hallucis</td>
<td>Adducts great toe (1)</td>
<td>P: Transverse head: Capsules of MP joints III–V</td>
<td>Lateral plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oblique head: Bases of metatarsals II–IV</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Lateral side of proximal phalanx of great toe (1)</td>
<td></td>
</tr>
<tr>
<td>Flexor hallucis brevis</td>
<td>Flexes MP joint of great toe (1)</td>
<td>P: Cuboid and lateral cuneiform bones</td>
<td>Medial plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Proximal phalanx of great toe (1)</td>
<td></td>
</tr>
<tr>
<td>Flexor digitii minimi brevis</td>
<td>Flexes MP joint of toe 5</td>
<td>P: Metatarsal V</td>
<td>Lateral plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Proximal phalanx of toe 5</td>
<td></td>
</tr>
<tr>
<td>Layer 4 (deepest)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsal interossei</td>
<td>Abducts toes</td>
<td>P: Adjacent sides of metatarsals</td>
<td>Lateral plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Sides of proximal phalanges of toes 2–4</td>
<td></td>
</tr>
<tr>
<td>Plantar interossei</td>
<td>Adducts toes</td>
<td>P: Sides of metatarsals III–V</td>
<td>Lateral plantar nerve (S2–S3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D: Medial side of proximal phalanges of toes 3–5</td>
<td></td>
</tr>
</tbody>
</table>

### Table 11.27  Summary of Leg and Foot Muscle Actions at the Foot and Toes

<table>
<thead>
<tr>
<th></th>
<th>FOOT</th>
<th>TOES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dorsiflexion</strong></td>
<td><strong>Plantar Flexion</strong></td>
<td><strong>Eversion</strong></td>
</tr>
<tr>
<td>Tibialis anterior¹</td>
<td>Gastrocnemius</td>
<td>Fibularis longus</td>
</tr>
<tr>
<td>Extensor digitorum longus</td>
<td>Soleus</td>
<td>Fibularis brevis</td>
</tr>
<tr>
<td>(Extenor hallucis longus)</td>
<td>Flexor digitorum longus</td>
<td>(Fibularis tertius)</td>
</tr>
<tr>
<td>(Fibularis tertius)</td>
<td>Flexor hallucis longus</td>
<td>Tibialis posterior</td>
</tr>
<tr>
<td>(Fibularis brevis)</td>
<td>(Fibularis longus)</td>
<td></td>
</tr>
<tr>
<td>(Plantaris)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Boldface indicates an agonist; others are synergists. Parentheses around an entire muscle name indicate only a slight effect. MP = metacarpophalangeal joint; IP = interphalangeal joint.
### CHAPTER SUMMARY

**11.1 Skeletal Muscle Composition and Actions**
- Axial muscles attach to components of the axial skeleton, whereas appendicular muscles stabilize or move components of the appendicular skeleton.
- Skeletal muscle typically has various attachments (previously called *origin* and *insertion*), and the fascicles are organized in one of four basic patterns.

**11.1a Skeletal Muscle Attachments**
- The superior (or proximal) attachment typically is the less movable attachment of a muscle, whereas the inferior (or distal) attachment is the more movable attachment.

**11.1b Organizational Patterns of Skeletal Muscle Fibers**
- Muscle fibers may be arranged in circular, parallel, convergent, or pennate patterns.

**11.1c Actions of Skeletal Muscles**
- An agonist is a prime mover, whereas an antagonist opposes the agonist.
- A synergist assists an agonist.

**11.2 Skeletal Muscle Naming**
- Muscles are named according to muscle action, body region, muscle attachment, orientation of the muscle fibers, shape, size, and muscle heads.

**11.3 Muscles of the Head and Neck**
- Muscles of the head and neck are separated into groups based upon their specific activities.

**11.3a Muscles of Facial Expression**
- The muscles of facial expression arise from the skull and often attach to the skin.

**11.3b Extrinsic Eye Muscles**
- The six extrinsic eye muscles attach to the external surface of the eye and control the eye’s movement.

**11.3c Muscles of the Oral Cavity and Pharynx**
- The muscles of mastication elevate and move the mandible during chewing.
- Intrinsic tongue muscles form the tongue itself, whereas extrinsic tongue muscles move the tongue during food manipulation, swallowing, and speech.
- Pharynx muscles function during swallowing.

**11.3d Muscles of the Anterior Neck: The Hyoid Muscles**
- The suprahyoid muscles elevate the hyoid bone, whereas the infrahyoid muscles depress the hyoid bone and move the thyroid cartilage of the larynx during swallowing or speaking.

**11.3e Muscles That Move the Head and Neck**
- Anterolateral neck muscles flex the head and neck, whereas posterior neck muscles extend the head and neck.

**11.4 Muscles of the Vertebral Column**
- The erector spinae muscles and other deep back muscles extend the vertebral column.

**11.5 Muscles of Respiration**
- The intercostal muscles, transversus thoracis, and the thoracic diaphragm change the shape of the thoracic cavity when we breathe.

**11.6 Muscles of the Abdominal Wall**
- The abdominal wall muscles compress the abdomen, hold the abdominal organs in place, and flex the vertebral column.

**11.7 Muscles of the Pelvic Floor**
- The muscles of the pelvic floor support the pelvic organs and form a muscular wall that covers the inferior pelvic opening.

**11.8 Muscles of the Pectoral Girdle and Upper Limb**
- Five groups of muscles are associated with pectoral girdle and upper limb movement: muscles that move (1) the pectoral girdle; (2) the glenohumeral joint/arm; (3) the elbow joint/forearm; (4) the wrist joint, hand, and fingers; and (5) the intrinsic muscles of the hand.

**11.8a Muscles That Move the Pectoral Girdle**
- Anterior thoracic muscles tend to depress the scapula or clavicle, protract the scapula or clavicle, or both. The posterior thoracic muscles, in comparison, elevate the scapula, retract the scapula, or both.

**11.8b Muscles That Move the Glenohumeral Joint/Arm**
- The pectoralis major flexes the arm, and the latissimus dorsi and teres major extend it, whereas all adduct and medially rotate the arm.
- The deltoid flexes, extends, and abducts the arm.
- The rotator cuff muscles provide strength and stability to the glenohumeral joint.

**11.8c Arm and Forearm Muscles That Move the Elbow Joint/Forearm**
- The principal flexors are on the anterior side of the arm, and the principal extensors are on the posterior side of the arm.
- The pronator teres and pronator quadratus pronate the forearm, whereas the supinator and biceps brachii supinate the forearm.

**11.8d Forearm Muscles That Move the Wrist Joint, Hand, and Fingers**
- Anterior forearm muscles flex the wrist and finger joints, whereas posterior forearm muscles extend the wrist and joints of the fingers.
- The tendons of the anterior and posterior forearm muscles are held in place by bands of dense regular connective tissue called retinacula.
11.8e Intrinsic Muscles of the Hand
- The intrinsic muscles may be divided into three groups: (1) the thenar group (moves the thumb), (2) hypothenar group (moves the little finger), and (3) the midpalmar group (moves fingers 2–5).

11.9 Muscles of the Pelvic Girdle and Lower Limb
- Four groups of muscles are associated with the pelvis and lower limb: (1) muscles that move the hip joint/thigh, (2) thigh muscles that move the knee joint/leg, (3) leg muscles, and (4) intrinsic muscles of the foot.

11.9a Muscles That Move the Hip Joint/Thigh
- Anterior thigh muscles flex the thigh.
- Gluteus maximus and the posterior thigh muscles (hamstrings) extend the thigh.
- Gluteus medius, gluteus minimus, and tensor fasciae latae abduct the thigh.
- Most medial thigh muscles adduct and flex the thigh.

11.9b Thigh Muscles That Move the Knee Joint/Leg
- The quadriceps femoris extends the leg.
- The gracilis, sartorius, and posterior thigh muscles (hamstrings) flex the leg.

11.9c Leg Muscles That Move the Ankle, Foot, and Toes
- Anterior leg muscles dorsiflex the foot, extend the toes, or both. One muscle (tibialis anterior) also inverts the foot.
- Lateral leg muscles evert the foot and plantar flex the foot.
- Posterior leg muscles plantar flex the foot, flex the toes, or both. One muscle (tibialis posterior) also inverts the foot.

11.9d Intrinsic Muscles of the Foot
- Dorsal muscles extend the toes.
- The four layers of plantar muscles can potentially flex, extend, abduct, or adduct the toes.
16. Identify the compartments of the arm (brachium), the muscles in each compartment, and their function.
17. Compare and contrast the flexor digitorum superficialis and the flexor digitorum profundus; where does each attach distally, how are their tendons interrelated, and what muscle actions do they perform?
18. What muscles are responsible for thigh extension? Which of these is the prime mover of thigh extension?
19. What leg muscles allow a ballet dancer to rise up and balance on her toes?
20. Which muscles are responsible for foot inversion?

**Can You Apply What You’ve Learned?**

1. A 50-year-old woman was concerned about the appearance of “crow’s-feet” by her eyes. She was told by her physician that these wrinkles were caused by years of squinting and blinking using which muscle?
   a. frontal belly of occipitofrontalis
   b. orbicularis oculi

2. Eliza complained of double vision and went to see her optometrist. The optometrist tested the function of various eye muscles and discovered that Eliza could not move her right eye medially. Which muscle may be injured?
   a. superior oblique
   b. lateral rectus
   c. medial rectus
   d. inferior oblique

3. After an intensive workout, George felt especially sore in his posterior arm regions. What repetitive workout activity most likely caused the soreness?
   a. flexing the humerus
   b. flexing the forearm
   c. pronating the forearm
   d. extending the forearm

4. While Carly was playing soccer, she was kicked in the anterior thigh by an opposing teammate. Due to this injury, what muscle function may have been difficult to perform?
   a. extending the knee
   b. flexing the knee
   c. extending the thigh
   d. dorsiflexing the foot

5. Joshua broke his fibula and had to wear a cast for 6 weeks. The muscles attaching to this bone atrophied during this time. What muscle function was Joshua unable to perform as a result?
   a. plantar flexing the foot
   b. flexing the knee
   c. evertmg the foot
   d. dorsiflexing the foot

**Can You Synthesize What You’ve Learned?**

1. Albon is a 45-year-old male who characterizes himself as a “couch potato.” He exercises infrequently and has a rounded abdomen (“beer belly”). While helping a friend move some heavy furniture, he felt a sharp pain deep within his abdomino-pelvic cavity. An emergency room resident told Albon that he had suffered an inguinal hernia. What is this injury, how did it occur, and how might Albon’s poorly developed abdominal musculature have contributed to it?

2. While training on the balance beam, Pat slipped during her landing from a back flip and fell, straddling the beam. Although only slightly sore from the fall, she became concerned when she suddenly lost the ability to completely control her urination. What might have happened to Pat’s pelvic floor structures during the fall?

3. After falling while skateboarding, Karen had surgery on her elbow. During her recovery, she must visit the physical therapist to improve muscle function around the elbow. Develop a series of exercises that may improve all of Karen’s elbow movements, and determine which muscles are being helped by each exercise.

4. Why is it more difficult for Eric to lift a heavy weight when his forearm ispronated than when his forearm is in the supine position?
Throughout the day, your body perceives and responds to multiple sensations. You smell spring flowers, feel the touch of a hand on your shoulder, and are visually aware of your environment. You control multiple muscle movements to walk, talk to the person sitting next to you, and hold this textbook. Other muscle movements occur without your voluntary input: Your heart beats, your stomach churns to digest your breakfast, and you jump at the sound of a honking horn. All of these sensations and muscle movements are interpreted or controlled by your nervous system.

Here we introduce the study of the nervous system by first describing its general functions and overall organization and then discussing the components of nervous tissue, which is the primary tissue of the nervous system. The next several chapters investigate different aspects of the nervous system, including the structure and function of the brain and cranial nerves (see chapter 13), spinal cord and spinal nerves (see chapter 14), organization and function of the autonomic nervous system (see chapter 15), and senses (see chapter 16).
12.1 Introduction to the Nervous System

The nervous system is composed of the brain, spinal cord, nerves, and ganglia and its primary tissue is nervous tissue (see section 5.4). Here we examine the general functions of the nervous system, how it is organized both structurally and functionally, and the general anatomy of nerves and ganglia.

12.1a General Functions of the Nervous System

**LEARNING OBJECTIVE**
1. Describe the three general functions of the nervous system.

The nervous system serves as the body’s primary communication and control system. It provides a rapid means of integrating and regulating body functions through electrical signals transmitted along specialized nervous tissue cells called *neurons* to accomplish the following:

- **Collect information.** *Receptors* are specialized nervous system structures that monitor changes in both the internal and external environment called **stimuli** *(sing., *stimulus)*. For example, receptors in the skin (see section 16.2a) detect stimuli associated with touch—this sensory information then is relayed along neurons to the spinal cord and brain.

- **Process and evaluate information.** After processing sensory information, the brain and spinal cord determine what response, if any, is required.

- **Initiate response to information.** The brain and spinal cord initiate a response as motor information is relayed along neurons to structures called **effectors**. Effectors include all three types of muscle tissue (see section 5.3) and glands (see section 5.1d). The effect may be either muscle contraction (or relaxation) or a change in gland secretion activity.

12.1b Organization of the Nervous System

**LEARNING OBJECTIVES**
2. Identify the structural components included in the CNS and those in the PNS.
3. Explain the functional organization of the nervous system.

Anatomists and physiologists have devised various ways to organize the structural and functional components of the nervous system. However, always keep in mind that such artificial divisions are merely intended to simplify discussion—there is only one nervous system.

**Structural Organization: Central Versus Peripheral Nervous System**

The nervous system consists of two anatomic divisions: the central nervous system and the peripheral nervous system *(figure 12.1a)*. The **central nervous system (CNS)** includes the brain and spinal cord. The brain is protected and enclosed within the skull, whereas the spinal cord is housed and protected within the vertebral canal.

The **peripheral (pê-rif’ér-äl) nervous system (PNS)** includes **nerves**, which are bundles of axons of neurons and **ganglia** *(gang’gĕ-lē; sing., *ganglion*; swelling), which are clusters of neuron cell bodies located along nerves. Nerves and ganglia are described in more detail in section 12.1c.

**Functional Organization: Sensory Versus Motor Nervous System**

The nervous system has two functional divisions: the sensory nervous system and the motor nervous system *(figure 12.1b)*. Both the sensory nervous system and the motor nervous system have CNS and PNS components.

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**Figure 12.1 Organization of the Nervous System.** The nervous system is organized into both structural and functional categories.

(a) Structural organization

- Central nervous system (CNS)
  - Brain
  - Spinal cord
- Peripheral nervous system (PNS)
  - Nerves
  - Ganglia

(b) Functional organization

- **Sensory nervous system** detects stimuli and transmits information from receptors to the CNS.
- **Motor nervous system** initiates and transmits information from the CNS to effectors.
  - **Somatic sensory**
    - Sensory input that is consciously perceived from receptors (e.g., eyes, ears, and skin)
  - **Visceral sensory**
    - Sensory input that is not consciously perceived from receptors of blood vessels and internal organs (e.g., heart)
  - **Somatic motor**
    - Motor output that is consciously controlled; effector is skeletal muscle
  - **Autonomic motor**
    - Motor output that is not consciously or involuntarily controlled; effectors are cardiac muscle, smooth muscle, and glands
Sensory Nervous System  The sensory nervous system, or afferent (af’er-ent; afferens = to bring to) nervous system, is responsible for receiving sensory information from receptors that detect stimuli and transmitting this information to the CNS. This information from the receptors to the CNS is called sensory input.

The sensory nervous system may be further subdivided based upon whether we are consciously aware of the stimulus that is detected. The two components are the somatic sensory and the visceral sensory. The somatic (sō-mat’ik; soma = body) sensory components detect stimuli that we can consciously perceive. Receptors of the somatic sensory nervous system include the receptors associated with the five senses (i.e., sight, smell, taste, hearing, and touch) and proprioceptors (receptors in joints and muscles that detect body position).

In comparison, the visceral (vis′ĕ-ră; viscus = internal organ) sensory components detect stimuli that we typically do not consciously perceive. Receptors of the visceral sensory nervous system include structures located within blood vessels and internal organs (e.g., heart, stomach, kidneys). Visceral receptors detect chemical composition of the blood or stretch of an organ wall, for example. The various types of receptors are described in detail in chapter 16.

Motor Nervous System  The motor nervous system, or efferent (ef′er-ent; efferens = to bring out) nervous system controls effectors (muscles and glands). This system is responsible for initiating and transmitting motor information from the CNS to the effectors. This information from the CNS to effectors is called motor output.

The motor nervous system, like the sensory nervous system, is subdivided into somatic and visceral parts. Here the distinction is based upon whether the stimulated effector can be controlled consciously (voluntarily). The somatic motor component initiates and transmits motor output from the CNS to skeletal muscles. For example, you exert voluntary control over your leg muscles as you press on the accelerator of your car.

The autonomic (aw-tŏ-nom′ik; auto = self, nomos = law) motor (or visceral motor) component innervates and regulates cardiac muscle, smooth muscle, and glands without our conscious control. We can neither voluntarily make our heart stop beating nor prevent our stomachs from growing. The autonomic motor component has two further subdivisions—sympathetic and parasympathetic—which are described in chapter 15.

Certain diseases are associated with specific components of the nervous system. For example, shingles (a painful skin rash caused by varicella zoster virus; see Clinical View 14.4: “Shingles”) infects somatic sensory neurons extending from receptors in the skin, whereas polio (caused by the poliovirus; see Clinical View 14.2: “Poliomyelitis”) preferentially infects somatic motor neurons to skeletal muscle, which in some cases may result in muscle weakness and paralysis.

WHAT DID YOU LEARN?

What are the two primary functional divisions of the nervous system? How do they differ?

12.1c Nerves and Ganglia

LEARNING OBJECTIVES

4. Describe the structure of a nerve, including the three layers of connective tissue wrappings.

5. Explain how nerves are classified structurally and functionally.

Nerves

A nerve is an organ composed of a cablelike bundle of axons (see section 5.4), connective tissue layers, and blood vessels, and it is a component of the peripheral nervous system. The organization of a nerve is shown in figure 12.2a. This figure is specifically of a spinal nerve that is extending from a section of the spinal cord, but all nerves have similar features. Observe the specific anatomic arrangement of axons within a nerve, and notice that within the nerve many axons are bundled into a structure called a fascicle (fas’i-kl; fascis = bundle) and numerous fascicles are present within the whole nerve. This is an arrangement similar to that of a skeletal muscle organ (see section 10.2a).

A nerve is similar to skeletal muscle in that it has three successive connective tissue wrappings:

- The epineurium (ep-i-nū’rē-ŭm; epi = upon) is a thick layer of dense irregular connective tissue that encloses the nerve. This fibrous tissue sheathes the entire nerve to protect and support it like a tough leather sleeve.
INTEGRATE

A similar bundling arrangement by connective tissue characterizes muscle (see section 10.2a). However, the entire muscle is enclosed by the epimysium, the fascicle is wrapped by the perimysium, and the skeletal muscle fibers are ensheathed by the endomysium.

CONCEPT CONNECTION

Nerves are vascularized (vas'kyū-lār-īzd; vas = vessel) by an extensive network of blood vessels, which branch and extend through both the epineurium and the perineurium to become capillaries (microscopic blood vessels; see section 20.1c). Capillaries are associated with the endoneurium and function as the site of exchange of substances (e.g., oxygen, glucose, waste products) between axons of neurons and the blood (see capillary exchange in section 20.3).

Structural and Functional Classification of Nerves

Nerves can be classified both structurally and functionally. Structural classification is based upon the CNS component from which the nerve extends: Cranial nerves extend from the brain (discussed in detail in section 13.9), and spinal nerves extend from the spinal cord (discussed in detail in section 14.5). Functional
classification of nerves is based upon the functional type of neuron (sensory neuron or motor neuron) a nerve contains. Sensory nerves contain only sensory neurons that relay information to the CNS, and motor nerves contain primarily motor neurons that relay information from the CNS. In contrast, mixed nerves contain both sensory and motor neurons. Most named nerves (including all spinal nerves and most cranial nerves) are mixed nerves. However, in mixed nerves, individual sensory or motor neurons still transmit only one type of information.

Ganglia

Whereas a nerve is a bundle of axons within the peripheral nervous system, a ganglion is a cluster of neuron cell bodies within the peripheral nervous system (figure 12.2c). The cluster of cell bodies results in a swelling, or enlarged portion, along the length of a nerve, which is often large enough to be observed with the naked eye. Specific types of ganglia include the dorsal root ganglia associated with sensory neurons that extend into the spinal cord and are associated with spinal nerves (figure 12.2; see also figure 14.3) and the ganglia associated with motor neurons that extend to autonomic effectors for the parasympathetic division (see figure 15.5) and the sympathetic division (see figure 15.6).

WHAT DID YOU LEARN?

3. What are the three connective tissue wrappings in a nerve, and what specific structure does each ensheathe?

12.2 Nervous Tissue: Neurons

Nervous tissue is the primary tissue of the nervous system and is composed of two distinct cell types: neurons and glial cells (first introduced in section 5.4). Neurons are excitable cells that initiate and transmit electrical signals, and glial cells are nonexcitable cells that primarily support and protect the neurons. We describe neurons here and glial cells in detail in section 12.4.

12.2a General Characteristics of Neurons

LEARNING OBJECTIVE

6. Describe five distinguishing features common to all neurons.

The basic structural unit of the nervous system is the neuron (nū’ron). These cells have several special characteristics:

- **Excitability.** Excitability is responsiveness to a stimulus (e.g., chemical, stretch, pressure change). The stimulus causes a local electrical change in the resting membrane potential (see section 4.4a) in the excitable cell by initiating the movement of ions across the plasma membrane of the excitable cell. These local electrical changes are called graded potentials (described in section 12.8a).

- **Conductivity.** Conductivity involves an electrical change that is quickly propagated along the plasma membrane as voltage-gated channels open sequentially during an action potential (described in section 12.8c). Thus, keep in mind that whereas excitability refers to the ability to initiate a local electrical change (a graded potential), conductivity refers to the ability to propagate (or move) an electrical change along the plasma membrane (an action potential).

- **Secretion.** Neurons release neurotransmitters in response to conductive activity (described in section 12.8d). Neurotransmitters (nū’trō-trans’mít’ér) are molecules stored in vesicles and when released bind to an excitable cell to cause either an excitatory or an inhibitory effect on these target cells (other neurons or effectors). These three characteristics of neurons (excitability, conductivity, and secretion) can be viewed collectively in the Concept Overview figure 12.23 at step 1, step 3, and step 4, respectively.

- **Extreme longevity.** Most neurons formed during fetal development are still functional in very elderly individuals.

- **Amitotic.** During fetal development, most neurons lose the ability to form new cells through cell division (i.e., mitotic; see section 4.9). Specific exceptions include the neurons in certain areas of the brain and in the olfactory epithelium of the nose (see section 16.3a).

Prevailing medical wisdom for years was that the number of neurons in your brain shortly after birth is set for your lifetime. Recent studies, however, have shown that this is not always the case.

INTEGRATE

CONCEPT CONNECTION

The characteristic of excitability (ability to respond to a stimulus) is exhibited in other body cells, including skeletal muscle, which may respond to a neurotransmitter (see section 10.3a), and sensory receptors, which respond to a variety of stimuli, depending on the specific type of receptor (see section 16.1d). For example, mechanoreceptors of the skin are stimulated by pressure (see section 16.2a), whereas photoreceptors of retinal cells of the eye are stimulated by light (see section 16.4d). All excitable cells maintain a resting membrane potential (see section 4.4b).
Researchers investigating the brain’s *hippocampus* (a region involved in memory processing; see section 13.7a) have found that this region of the brain contains a population of neural stem cells (see Clinical View 5.4: “Stem Cells”). These stem cells were once thought to give rise only to new glial cells (see section 12.4) in adults, but it is now clear that under special circumstances they can mature into neurons. Researchers have learned that the surrounding glial cells provide the chemical signals that direct a stem cell down the path of neuron maturation. Olfactory neurons associated with smell also retain mitotic ability and are replaced every 40 to 60 days (see section 16.3a).

### WHAT DID YOU LEARN?

4. Explain the neuron characteristics of excitability, conductivity, and secretion.

#### 12.2b Neuron Structure

**LEARNING OBJECTIVES**

7. Describe the three basic anatomic features common to most neurons.

8. Identify and describe the structures unique to neurons.

Neurons come in many shapes and sizes, but they typically share certain basic structural features that include a cell body, dendrites, and an axon (figure 12.3). The dendrites and axon are cellular extensions that project from the spherically shaped cell body—with the axon emanating from the triangular, cone-shaped region of the cell body called the **axon hillock** (hil’lok; *hillock* = cone).

The neuron **cell body** (or *soma*) houses both the nucleus and the cytoplasm. The nucleus contains chromatin (see section 4.7b) and a prominent nucleolus, which synthesizes the cell body’s large number of ribosomes (see section 4.6b). The cytoplasm within the cell body, which is more specifically called the **perikaryon** (*per’i-kar’e-on; peri = around, karyon = kernel), is composed of the typical cellular organelles such as the endoplasmic reticulum, Golgi apparatus, ribosomes, and mitochondria (see section 4.6). (Note that some anatomists use the term *perikaryon* to describe the entire cell body.)

A distinctive features of a neuron cell body is that it contains a large number of ribosomes, as mentioned. The ribosomes are either attached to the endoplasmic reticulum (ER) as part of an extensive rough ER or are free ribosomes within the cytosol (see figure 4.27b). Collectively, they readily absorb basic dyes when a nervous tissue sample is stained for viewing with a microscope (see section 4.1a); thus, they appear as dark-staining bodies and are referred to as **chromatophilic** (krō-mā-tō-fil’ık; *chromo* = color, *phileo* = to love) **substance**. (They are also referred to as *Nissl bodies*, after the microscopist Franz Nissl, who first described them.) It is the chromatophilic substance (along with the absence of myelin, a glistening coat formed by insulating cells; described in section 12.4c) that accounts for the gray color of *gray matter* seen in gross dissections of the brain and spinal cord (see figure 13.4). Note that the axon hillock is the only portion of the cell body that lacks chromatophilic substance.

The cell body serves as the neuron’s control center because (1) it contains both the nucleus and the cytoplasm and (2) it functions in many of the cell’s metabolic activities (see section 4.6). It also transmits **graded potentials** along its plasma membrane to the axon (see section 12.8a). Graded potentials are received from the dendrites and initiated at the cell body.

**Dendrites** (den’drīt; *dendrites* = relating to a tree) tend to be relatively short, small, tapering processes that branch off the cell body. Some neurons have many dendrites; others have only one. Dendrites, like the cell body, are not insulated with myelin. Dendrites transmit **graded potentials** along their plasma membrane toward the cell body (see section 12.8a). The greater the number of dendrites, the more input a neuron may receive.

The single **axon** (ak’son) (or *nerve fiber*) is typically a longer process emanating from the cell body to make contact with other neurons, muscle cells, or gland cells. Specialized terminology is used for both the axon’s cytoplasm and its plasma membrane. The cytoplasm within an axon is called **axoplasm**, and the plasma membrane of an axon is called an **axolemma**. Unlike the cell body, the axon is devoid of chromatophilic substance. This distinctive difference allows the cell body to be distinguished from the axon when nervous tissue is viewed with a microscope.

An axon gives rise to a few side branches called **axon collaterals**. Most axons and their collaterals branch extensively at their distal end into an array of fine terminal extensions. The extreme tips of these fine extensions are slightly expanded regions called **synaptic** (*si-nap’tik*) **knobs**, also called **synaptic bulbs**, *end bulbs*, or **terminal boutons**. Within the synaptic knobs are numerous **synaptic vesicles** containing neurotransmitter. A synaptic knob ends at a functional junction...
Figure 12.3 Structures in a Typical Neuron. Anatomic structures of a neuron. (a) Terminal ends of an axon, illustrating the synaptic knobs of a presynaptic neuron, and (b) a postsynaptic neuron, including dendrites, cell body, and axon. The flow of electrical signals is from dendrites, to the cell body to the axon, until it reaches the synaptic knob, which releases neurotransmitter. (c) A synapse between the two neurons. (d) Photomicrograph of a motor neuron.

(c) Synapse
(d) Photomicrograph of a neuron

Chromatophilic substance
Cytoskeleton
Dendrite
Nucleus
Nucleolus
Cell body
Axon hillock
Neuron or effector (muscle or gland)

(d) ©Ed Reschke/Getty Images
called a synapse (described in section 12.3). Axons function in the initiation and propagation of action potentials along their axolemma (or plasma membrane; see sections 12.8b, c), which trigger synaptic vesicles to release neurotransmitter from the synaptic knobs (see section 12.8d).

Axons (and axon collaterals) may be insulated with a myelin sheath, which is formed by a certain type of glial cells (either neurolemmocytes or oligodendrocytes). Neurofibril nodes are the uninsulated regions of the axon between the myelin sheaths. The formation of the myelin sheath is described in section 12.4c.

Cytoskeleton

The entire neuron has an extensive cytoskeleton (see section 4.6b), which is composed of a type of intermediate filament called a neurofilament (nūr′ō-fil′ə-ment; filamentum = thread), microtubules, and microfilaments (actin). These proteins are within the cell body and extend into the dendrites and axon. They function in maintaining neuron shape and providing structural support. The neurofilaments aggregate to form parallel bundles called neurofibrils (nūr′ō-fibril; fibrillum = fiber). Microtubules, which are embedded in parallel clusters alongside the neurofilaments of an axon, participate in cellular transport within axons (i.e., axonal transport; described in section 12.2).

A protein (called tau) that stabilizes microtubules of the neuron cytoskeleton is associated with Alzheimer disease. (See Clinical View 13.13: “Alzheimer Disease: The ‘Long Goodbye’”).

WHAT DID YOU LEARN?

What are the functions of these neuron structures: dendrites, axon, synaptic vesicles, and neurofibrils?

12.2c Neuron Transport

LEARNING OBJECTIVE

9. Distinguish between fast axonal transport and slow axonal transport, and give examples of the different substances moved by each.

Axons are generally dependent upon the nucleus and organelles within the cell body to provide them with newly synthesized materials (e.g., organelles, vesicles, molecules) and to break down or recycle their used materials. To accomplish this, substances are moved in both directions through an axon. Anterograde (an′ter-ō-grād; ante = before, gradior = to step) transport is the movement of materials from the cell body toward synaptic knobs, and retrograde (ret′rō-grād; retro = backward) transport is the movement of materials from synaptic knobs toward the cell body. The transport processes are classified as either fast axonal transport or slow axonal transport, depending upon the relative speed of movement.

Fast Axonal Transport

Fast axonal transport occurs at approximately 400 millimeters per day. The mechanism involves movement along microtubules. You may find it helpful to think of this process as similar to a substance being pulled along a train track. The power for this movement comes from specialized motor proteins (e.g., kinesin, dynein) that split ATP to supply the energy needed.

Substances can be moved via fast axonal transport in either direction (anterograde or retrograde). Cellular structures formed in the cell body are moved by anterograde transport toward the synaptic knobs and include vesicles, organelles, and glycoproteins required at the synapse. Used vesicles to be broken down and recycled, and potentially harmful agents, are moved via retrograde transport from the synaptic knob toward the cell body (see Clinical View 12.1: “Pathogenic Agents and Fast Axonal Transport”). Interestingly, new research results support the idea that some of these vesicles are transporting hormonelike molecules for the purposes of communication between neurons. This represents a means of neurons communicating information in a retrograde direction from synaptic knobs to the cell body. Research continues in this area.

Slow Axonal Transport

Slow axonal transport occurs at approximately 0.1 to 3 millimeters per day. This type of movement results from the flow of the axoplasm (an axon’s cytoplasm), and is also called axoplasmic flow. The materials are only moved from the cell body toward the synaptic knob (anterograde). These substances include enzymes, cytoskeletal components, and new axoplasm for regenerating axons.

WHAT DID YOU LEARN?

Which type of axonal transport is both anterograde and retrograde? Give examples of substances transported by this method.
12.2d Classification of Neurons

**LEARNING OBJECTIVES**

10. Name and describe the four structural categories of neurons.
11. Identify the three functional categories of neurons and where each is primarily located.

Neurons vary in morphology and location. Similar to the components of the entire nervous system, they are classified according to both their structure and their function.

**Structural Classification**

Neurons are classified structurally based upon the number of neuron processes (i.e., axon or dendrites) emanating directly from the cell body. Thus, they may be classified as multipolar, bipolar, unipolar, or anaxonic neurons (table 12.1).

**Multipolar neurons** have many dendrites and a single axon that extends from the cell body. These are the most common type of neurons in the human body.

<table>
<thead>
<tr>
<th>Neuron Type</th>
<th>Structure</th>
<th>Description</th>
<th>Examples of Functional Types</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multipolar neuron</td>
<td>Dendrites</td>
<td>Multiple processes extend directly from the cell body; typically many dendrites and one axon; most common type of neuron</td>
<td>All motor neurons; most interneurons</td>
</tr>
<tr>
<td>Bipolar neuron</td>
<td>Dendrite</td>
<td>Two processes extend directly from the cell body; one dendrite and one axon; relatively limited in where they are located</td>
<td>Some special sense neurons (e.g., retina of eye, olfactory epithelium in nose)</td>
</tr>
<tr>
<td>Unipolar neuron</td>
<td>Axon</td>
<td>Single short process extends directly from the cell and looks like a T as a result of the fusion of two processes into one long axon</td>
<td>Most sensory neurons</td>
</tr>
<tr>
<td>Anaxonic neuron</td>
<td>Dendrites</td>
<td>Processes are only dendrites; no axon present</td>
<td>Interneurons</td>
</tr>
</tbody>
</table>
Bipolar neurons have two processes that extend from the cell body—one dendrite and one axon. The location of these neurons is relatively limited in humans.

Unipolar neurons have a single, short neuron process that emerges from the cell body and branches like a T. These neurons are also called pseudounipolar (puh-soo-doo-nil-poh-lahr; pseudo = false, uni = one) because they start out as bipolar neurons during development, but their two processes fuse into a single process. The naming of the branched processes in unipolar neurons has been a source of confusion with regard to the common definitions of dendrites and axons. It seems most appropriate to call the short, multiple-branched receptive endings dendrites (which exhibit graded potentials; see section 12.8a). The other portion is called the axon because these processes, like other axons, generate and conduct action potentials (see sections 12.8c, d). These axons are composed of the combined peripheral process (from dendrites to the cell body) and central process (from the cell body into the CNS).

Anaxonic (an-aks’on-ik; an = without) neurons have only dendrites and no axons. They are different from other types of neurons because they produce graded potentials, but they do not produce action potentials.

Functional Classification

Neurons are classified functionally according to the direction in which action potentials are propagated relative to the CNS. The three categories are sensory neurons, motor neurons, and interneurons (figure 12.4).

Sensory neurons (also known as afferent neurons) are the neurons of the sensory nervous system. They are responsible for conducting sensory input from both somatic sensory (e.g., touch receptors) and visceral sensory receptors (e.g., stretch receptors within the urinary bladder) to the CNS. Most sensory neurons are unipolar. However, a few somatic sensory neurons are bipolar, such as those in the retina of the eye (see figure 16.12) and olfactory epithelium of the nose (see figure 16.5).

Motor neurons (also known as efferent neurons) are the neurons of the motor nervous system, conducting motor output from the CNS to both somatic effectors (i.e., skeletal muscle) and autonomic effectors (i.e., cardiac muscle, smooth muscle, and glands). All motor neurons are multipolar.

Interneurons (also known as association neurons) lie entirely within the CNS. They receive stimulation from many other neurons and carry out the integrative function of the nervous system—that is, they receive, process, and store information and “decide” how the body responds to stimuli. Interneurons facilitate communication between sensory and motor neurons. Interneurons outnumber all other neurons; it is estimated that 99% of our neurons are interneurons. Interneurons are either multipolar neurons or anaxonic neurons. Neuron classification based upon structure and function is integrated into table 12.1.

WHAT DID YOU LEARN?

1. How are the different processes that extend from a cell body used to structurally classify neurons?

2. Where are interneurons located, and what is their function?

12.3 Synapses

LEARNING OBJECTIVES

12. Define a synapse.

13. Describe the essential structural and functional differences between a chemical synapse and an electrical synapse.

A synapse (sin’aps; syn = together, hapto = to clasp) is the specific location where a neuron is functionally connected to either another neuron or an effector (muscle or gland). There are two types of synapses in the human body: chemical synapses and electrical synapses. Most synapses within the nervous system are chemical synapses.

A chemical synapse between two neurons is composed of a presynaptic (pre-si-nap’tik; pre = before) neuron (see figure 12.3a), which is the signal producer (releases neurotransmitter), and a postsynaptic (post-si-nap’tik; post = after) neuron (see figure 12.3b),
which is the signal receiver, or target (binds neurotransmitter). The synapse may be between the axon of the presynaptic neuron and any portion of the surface of a postsynaptic neuron (dendrite, cell body, or axon), except those regions that are covered by a myelin sheath (described in section 12.4c). Most commonly, a synapse is with a dendrite of the postsynaptic neuron. The synaptic knob of the presynaptic neuron does not quite make contact with the postsynaptic neuron (figure 12.3c). The two neurons are separated by an extremely narrow, fluid-filled gap (of about 30 nanometers) called the synaptic cleft.

Transmission between a presynaptic and postsynaptic neuron occurs when neurotransmitter molecules stored in synaptic vesicles are released from the synaptic knob of a presynaptic neuron into the synaptic cleft. Some of the neurotransmitter diffuses across the synaptic cleft to bind to receptors within the plasma membrane of the postsynaptic neuron to initiate a graded potential (see section 12.8a). There is a synaptic delay associated with neurotransmitter release at chemical synapses. The delay is the time between the neurotransmitter release from the presynaptic cell, its diffusion across the synaptic cleft, and neurotransmitter binding to receptors in the postsynaptic neuron plasma membrane. This delay is usually between 0.3 and 0.5 millisecond. Note that one postsynaptic neuron may, and often does, receive signals from more than one presynaptic neuron simultaneously.

A second type of synapse (which is much less common in humans) is an electrical synapse. An electrical synapse is composed of a presynaptic neuron and a postsynaptic neuron physically bound together. Gap junctions (see section 4.6d) are present in the plasma membranes of both neurons and facilitate the flow of ions between the cells. The cells act as though they shared a plasma membrane. Thus, the electrical signal passes between the cells with essentially no synaptic delay. Electrical synapses are located within limited regions of the brain and the eyes.

**WHAT DID YOU LEARN?**

9. What is a chemical synapse within the nervous system, and how does it function?

### 12.4 Nervous Tissue: Glial Cells

Glial cells are the other distinct cell type within nervous tissue. These nonexcitable cells primarily support and protect the neurons.

#### 12.4a General Characteristics of Glial Cells

**LEARNING OBJECTIVE**

14. List the distinguishing features of glial cells.

Glial (gli’āl; gliā = glue) cells are sometimes referred to as neuroglia (nū-rog’ē-a). They are found within both the CNS and the PNS. Glial cells are both smaller than neurons and capable of producing new glial cells through cell division (i.e., glial cells have mitotic ability; see section 4.9b). Glial cells do not transmit electrical signals, but they do assist neurons with their functions. The glial cells cooperate to physically protect and help nourish neurons as well as provide an organized, supporting scaffolding for all the nervous tissue. During development, glial cells form the framework that guides young, migrating neurons to their final destinations. Recent evidence has

![Figure 12.5 Glial Cells of the Central Nervous System (CNS).](image)

Four types of glial cells are located within the CNS: astrocytes, ependymal cells, microglia, and oligodendrocytes. These cells differ in both structure and function.

<table>
<thead>
<tr>
<th>Astrocyte Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Helps form the blood-brain barrier</td>
</tr>
<tr>
<td>2. Regulates interstitial fluid composition</td>
</tr>
<tr>
<td>3. Provides structural support and organization to the central nervous system (CNS)</td>
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<td>4. Assists with neuronal development</td>
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<td>5. Replicates to occupy space of dying neurons</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Ependymal Cell Functions</th>
</tr>
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<tbody>
<tr>
<td>1. Lines ventricles of brain and central canal of spinal cord</td>
</tr>
<tr>
<td>2. Assists in production and circulation of cerebrospinal fluid (CSF)</td>
</tr>
</tbody>
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<tr>
<th>Microglial Cell Functions</th>
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<tbody>
<tr>
<td>1. Phagocytic cell that moves through the CNS</td>
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<tr>
<td>2. Protects the CNS by engulfing infectious agents and other potentially harmful substances</td>
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</table>
shown that glial cells are critical for the normal function at neural
synapses, both maintaining the anatomic structure of synapses and
modifying transmission that occurs there.

Glia: far outnumber neurons. The nervous tissue of a young
adult may contain 35 to 100 billion neurons and 100 billion to 1 tril-
ion glial cells. Collectively, glial cells account for roughly half the
volume of the nervous system.

WHAT DID YOU LEARN?
If a person has a brain tumor, is it more likely to have developed from
neurons or from glial cells? Why?

12.4b Types of Glial Cells

LEARNING OBJECTIVE
15. Describe the structure and function of the four types of glial cells within
the CNS and the two types of glial cells within the PNS.

Glial Cells of the CNS

Four types of glial cells are found in the central nervous system
(CNS). These different cells are astrocytes, ependymal cells, micro-
glia, and oligodendrocytes (figure 12.5). They can be distinguished
based upon size, intracellular organization, and the presence of spe-
cific cytoplasmic processes.

Astrocytes (as′trō-sīt; astron = star) exhibit a starlike shape due
to projections from their surface. These numerous cell processes have
contact with both neurons and blood capillaries (smallest blood ves-
sels) (see section 20.1c). Astrocytes are the most abundant glial cells
in the CNS and constitute over 90% of the nervous tissue in some areas
of the brain. Astrocytes nurture, protect, support, and guide
neurons, as follows:

- **Help form the blood-brain barrier.** The ends of astrocyte
  processes are called perivascular feet: They both cover and
  wrap around capillaries in the brain. The perivascular feet
  and the brain capillaries together contribute to a blood-brain
  barrier (BBB). The BBB strictly controls movement of
  substances from exiting the blood and entering the nervous
tissue in the brain. The BBB protects the delicate neurons of
  the brain from toxins, but at the same time allows needed
  nutrients to pass through (see section 13.2d).

- **Regulate interstitial fluid composition.** Astrocytes help
  maintain an optimal chemical composition of the interstitial
  fluid (fluid around cells) within the brain. For example,
  astrocytes regulate potassium ion concentration by absorbing
  these ions to maintain a constant potassium ion concentration
  that is critical to electrical activity of neurons.

- **Form structural support.** The cytoskeleton in astrocytes
  strengthens these cells to provide a structural framework to
  support and organize neurons within the CNS.

- **Assist neuronal development.** Astrocytes help direct the
  development of neurons in the fetal brain by secreting chemicals
  that regulate the formation of connections between neurons.

- **Occupy the space of dying neurons.** When neurons are
damaged and die, the space they formerly occupied is often
filled by astrocytes that replicate through cell division.

Ependymal (e-pen′di-măl) cells are ciliated simple cuboidal or
simple columnar epithelial cells (see table 5.2) that line the internal
cavities (ventricles) of the brain (see figure 13.7) and the central
canal of the spinal cord (see figure 14.3). These cells have slender
processes that branch extensively to make contact with other glial
cells in the surrounding nervous tissue.

Ependymal cells and nearby blood capillaries together form a net-
work called the choroid (ko′royd) plexus (see figure 13.8). The cho-
roid plexus helps produce cerebrospinal fluid (CSF), a clear liquid that
bathes the external surfaces of the CNS and fills its internal cavities.
The cilia of ependymal cells help circulate the CSF (see section 13.2c).

Microglia (mi-kro′g-lē-ā; micro = small) are typically small cells
that have slender branches extending from the main portion of the cell.
They represent the smallest percentage of CNS glial cells, with some
estimates of their prevalence as low as 5%. They are classified as phago-
cytic cells (macrophages) of the immune system (see section 22.2a).
Microglial cells wander through the CNS and replicate in response to
an infection. They protect the CNS against microorganisms (e.g., bacte-
ria) and other potentially harmful substances by engulfing and destroy-
ing them through phagocytosis (see figure 22.3a). Microglia also
function in removing debris from dead or damaged nervous tissue that
results from infections, inflammation, trauma, and brain tumors.

Oligodendrocytes (ol′i-gō-den′drō-sīt; oligos = few) are large
cells with a bulbous (round) body and slender cytoplasmic extensions or
processes. The extensions of oligodendrocytes wrap around and
insulate axons within the CNS to form a myelin sheath through a
process called myelination (see section 12.4c). This insulation allows
for faster propagation of action potentials along the axon.

Glia: Cells of the PNS

Two types of glial cells are found in the peripheral nervous system
(PNS). These specialized glial cells function in insulating neurons and
include satellite cells and neurolemmocytes (figure 12.6).
Satellite cells are flattened cells arranged around neuronal cell bodies in a ganglion (see section 12.1c). The satellite cells physically separate cell bodies from their surrounding interstitial fluid. They function to electrically insulate the cell body and regulate the continuous exchange of nutrients and waste products between neuron cell bodies and their environment.

Neurolemmocytes (nūr-ō-lem′ō-sīt) are also called Schwann cells. These elongated and flattened cells wrap around and insulate axons within the PNS to form a myelin sheath through myelination (see section 12.4c). As with myelin sheaths formed by oligodendrocytes in the CNS, this allows for faster propagation of action potentials along the axon. The process of forming the myelin sheath by both neurolemmocytes and oligodendrocytes is described in section 12.4c.

**WHAT DID YOU LEARN?**

11. If a person suffers from meningitis (an inflammation of the meningeal coverings around the brain), which type of glial cell usually replicates in response to the infection?

12. Which specific type of glial cell forms a myelin sheath associated with axons in the PNS?

**12.4c Myelination**

**LEARNING OBJECTIVES**

16. Define myelination, and describe the composition and function of a myelin sheath.

17. Distinguish between the myelination process carried out by neurolemmocytes in the PNS and by oligodendrocytes in the CNS.

**Myelination** (mī′e-lā-nā′shən) is the process by which part of an axon is wrapped with **myelin** (mī′e-lin). Myelin is the insulating covering around the axon that consists of repeating concentric layers of plasma membrane of glial cells. Myelination is completed by neurolemmocytes in the PNS and by oligodendrocytes in the CNS. Myelin mainly consists of the plasma membrane of these glial cells and contains a large proportion of lipids and a smaller amount of proteins. The high lipid content of the myelin gives an axon a distinct, glossy-white appearance and effectively insulates an axon.
Figure 12.7 illustrates the process of myelinating a PNS axon. The neurolemmocyte starts to encircle a 1-millimeter portion of an axon. As the neurolemmocyte continues to wrap around the axon, the cytoplasm and nucleus of the neurolemmocyte are squeezed to the periphery of the neurolemmocyte. The overlapping inner layers of the plasma membrane form the myelin sheath. The periphery of the neurolemmocyte contains the cytoplasm and nucleus and is called the neurilemma (nūr-i-lē′mə; lemma = husk). This process is similar to what would happen if you were to take a balloon with a small amount of water in it and wrap it numerous times around a pencil. The balloon is wrapped over and over around a section of your pencil and the part of the balloon containing water is pushed to the outside. The wrapped layers of balloon represent the myelin sheath, and the external portion of the balloon with the water represents the neurilemma.

A neurolemmocyte in the PNS can myelinate only a 1-millimeter portion of a single axon. Thus, if an axon is longer than 1 millimeter (and most PNS axons are), it takes many neurolemmocytes to myelinate the entire axon. Figure 12.8a shows an axon that has seven neurolemmocytes wrapped around it. The axons in many of the nerves in the body have hundreds or thousands of neurolemmocytes along their entire length. The gaps between the neurolemmocytes are called neurofibril (nū′rō-fi′bril) nodes, or nodes of Ranvier.

An oligodendrocyte in the CNS, in comparison, can myelinate a 1-millimeter portion of multiple axons and not just one. Figure 12.8b shows oligodendrocytes myelinating portions of three different axons. The cytoplasmic extensions of the oligodendrocyte wrap repeatedly around a portion of each axon where plasma membrane layers of the oligodendrocyte form the myelin sheath. Note that no

INTEGRATE

CLINICAL VIEW 12.3
Nervous System Disorders Affecting Myelin

Multiple sclerosis (MS) is progressive demyelination of neurons in the central nervous system accompanied by the destruction of oligodendrocytes. MS is an autoimmune disorder (see Clinical View 22.4: “Autoimmune Disorders”), because the body’s immune cells mistake the oligodendrocytes as foreign and attack them. As a result, the propagation of action potentials is disrupted, leading to impaired sensory perception and motor coordination. Repeated inflammatory events at myelinated sites cause scarring (sclerosis), and in some cases function is permanently lost. The disease usually affects young adults between the ages of 18 and 40. It is five times more prevalent in individuals of European descent than in African-Americans. Among the typical symptoms are vision problems, muscle weakness and spasms, urinary tract infections and bladder incontinence, and drastic mood changes.

Guillain-Barré syndrome (GBS) is a disorder in which inflammation causes loss of myelin from the peripheral nerves and spinal nerve roots (see figure 14.3). It is characterized by muscle weakness that begins in the distal limbs but rapidly advances to involve proximal muscles as well (ascending paralysis). Most cases of GBS are preceded by an acute, flu-like illness, although no specific infectious agent has ever been identified. The condition in rare instances may follow an immunization. Even though GBS appears to be an immune-mediated condition, the use of steroids provides little, if any, measurable improvement. In fact, most people recover almost all neurologic function on their own with little medical intervention. A potential concern reported by the Centers for Disease Control and Prevention is the correlation that has been observed between countries with Zika virus outbreaks and an increased incidence of GBS. However, for an individual who is infected with the Zika virus, the risk of developing GBS is very low.

Figure 12.7 Myelination of PNS Axons. A myelin sheath surrounds most axons. In the PNS, neurolemmocytes form both a myelin sheath and neurilemma in a series of sequential stages. AP R

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Figure 12.8 Myelin Sheaths in the PNS and CNS. (a) In the PNS, the neurolemmocyte ensheathes only one small part of a single axon to form both a myelin sheath and a neurilemma. (b) In the CNS, an oligodendrocyte wraps around a small part of multiple axons, forming a myelin sheath (but no neurilemma is formed).

Neurilemma is formed as the axons of CNS neurons are myelinated. Neurofibril nodes are also located between adjacent oligodendrocyte “wraps.” You may find it helpful to imagine that an oligodendrocyte is like a latex glove with fluid added to it. Each finger of the glove is wrapped numerous times around the axon of different neurons, and the fluid is pushed into the hand of the glove. Remember, myelination in both the PNS and CNS allows for faster propagation of action potentials (see sections 12.8b, c).

Not all axons are myelinated. Unmyelinated axons in the PNS (figure 12.9) are also associated with neurolemmocytes, which help to protect and support the axon. However, no myelin sheath covers them. Thus, the axon merely rests in a depressed portion of the neurolemmocyte, but its plasma membrane does not form repeated layers around the axon. In the CNS, unmyelinated axons are not associated with oligodendrocytes.

**WHAT DID YOU LEARN?**

13 What is the function of the myelin sheath? How does myelination of axons occur in the PNS?

Figure 12.9 Unmyelinated Axons. (a) Unmyelinated axons are partially surrounded by a neurolemmocyte but are not wrapped in a myelin sheath. (b) An electron micrograph shows a myelinated axon and several unmyelinated axons.

(b) © Don W. Fawcett/Science Source
12.5 Axon Regeneration

**LEARNING OBJECTIVES**

18. Identify factors that influence regeneration of PNS axons, and explain why axon regeneration in the CNS is limited.

19. Describe the events of Wallerian degeneration and axon regrowth.

PNS axons are vulnerable to cuts, crushing injuries, and other types of trauma. However, a damaged axon can regenerate (i.e., grow back) if the cell body remains intact and a critical amount of neurilemma remains. The degree of success of PNS axon regeneration depends upon two primary factors: (1) the amount of damage and (2) the distance between the site of the damaged axon and the structure it innervates. The possibility of repair is decreased with an increase in either of these two factors.

Neurolemmocytes play an active role in regeneration. This process is illustrated in figure 12.10 and follows these stages:

1. The axon is severed by some type of trauma.
2. The portion proximal to the trauma seals off by membrane fusion and swells. The swelling is a result of axoplasmic flow (slow axonal transport; see section 12.2c) from the neuron cell body through the axon. At the same time, the axon severed from the cell body and the myelin sheath surrounding the axon break down—a process called Wallerian (waw-lē′rē-ān) degeneration. Macrophages remove the debris through phagocytosis (see section 22.3b). However, the neurilemma in the distal region survives.
3. The neurilemma and the remaining endoneurium form a regeneration tube.
4. The axon begins to regenerate and remyelination occurs. The regeneration tube guides the axon sprout as it begins to grow rapidly through the regeneration tube at a rate of about 2 to 5 millimeters per day. This occurs under the influence of nerve growth factors released by the neurolemmocytes.
5. Innervation is restored as the axon reestablishes functional contact with its original structure. The structure is either a receptor for sensory neurons to regain sensory perception or an effector for motor neurons to regain control of a muscle or gland.

Potential regeneration of damaged neurons within the CNS (i.e., the brain or spinal cord) is very limited for several reasons. First, oligodendrocytes do not release a nerve growth factor, and in fact they actively inhibit axon growth by producing and secreting several growth-inhibitory molecules. Second, the large number of axons crowded within the CNS tends to complicate regrowth activities. Finally, both astrocytes and connective tissue coverings may form some scar tissue that obstructs axon regrowth. Medical researchers are attempting to overcome these limitations to treat patients with spinal cord injuries. (See Clinical View 14.3: “Treating Spinal Cord Injuries.”)

**Figure 12.10 Regeneration of PNS Axons.** Following injury to a peripheral nerve, the severed axon may be repaired and grow out to reinnervate the receptor or effector (in this case, a skeletal muscle fiber).

**WHAT DID YOU LEARN?**

14. What two primary factors determine the effectiveness of PNS axon regeneration?

15. How does the process of nerve regeneration occur in the PNS?
12.6 Plasma Membrane of Neurons

The ability of the nervous system to integrate and regulate body functions is ultimately dependent upon the transmission of electrical signals (i.e., graded potentials and action potentials) by neurons. Here we discuss the details of the plasma membrane of neurons that make transmitting electrical signals possible. Included are descriptions of the various types of pumps and channels in a neuron’s plasma membrane and their typical distribution within a neuron.

12.6a Types of Pumps and Channels

**LEARNING OBJECTIVE**

20. Distinguish between a pump and a channel, and describe the three specific states of a voltage-gated Na\(^+\) channel.

Neurons contain transport proteins for moving substances across the plasma membrane. These include both pumps and various types of channels. Transport proteins were discussed in section 4.3 and are reviewed here.

**Pumps** maintain specific concentration gradients by moving substances up (against) a concentration gradient, a process that requires cellular energy. The plasma membrane of neurons contains both sodium-potassium (Na\(^+\)/K\(^+\)) pumps (see figure 4.15) and calcium (Ca\(^{2+}\)) pumps (see figure 4.14). A great deal of energy is required to power the vast number of Na\(^+\)/K\(^+\) pumps in a neuron’s plasma membrane—approximately two-thirds of a neuron’s energy expenditure!

**Channels** provide the means for a substance to move down (with) its concentration gradient. Neurons contain the following major types of channels:

- **Leak (passive) channels.** These channels are always open, allowing continuous diffusion of a specific type of ion from a region of high concentration to a region of low concentration. Examples of leak channels are sodium ion (Na\(^+\)) leak channels and potassium ion (K\(^+\)) leak channels.

- **Chemically gated channels.** These channels are normally closed. They temporarily open in response to binding of a neurotransmitter. When open, they allow a specific type of ion (or ions) to diffuse across the plasma membrane. Examples of chemically gated channels include chemically gated K\(^+\) channels and chemically gated chloride ion (Cl\(^-\)) channels.

- **Voltage-gated channels.** These channels are also normally closed, but they temporarily open in response to changes in electrical charge (potential) across the plasma membrane. When open, they allow a specific type of ion to diffuse across the membrane. Examples of voltage-gated channels include voltage-gated Na\(^+\) channels, voltage-gated K\(^+\) channels, and voltage-gated Ca\(^{2+}\) channels. Most gated channels have one gate that is in either one of two states: closed or open. Voltage-gated Na\(^+\) channels are unique in that they have two gates (an **activation gate** and an **inactivation gate**) and thus exhibit three states (figure 12.11).

![Figure 12.11 Voltage-Gated Na\(^+\) Channels.](image-url)
Three States of Voltage-Gated Na\(^+\) Channels

1. **Resting state.** Although the inactivation gate is open, the activation gate is closed, and entry of Na\(^+\) is prevented.

2. **Activation state.** Both the inactivation gate (which remains open) and the activation gate are open (activation gate opens in response to a voltage change); Na\(^+\) moves into the cell through the open channel.

3. **Inactivation state.** Although the activation gate is open, the inactivation gate is temporarily closed (for several milliseconds) following activation of the Na\(^+\) channel—during this time, it cannot be stimulated to reopen, and entry of Na\(^+\) is prevented.

The resting state of voltage-gated Na\(^+\) channels is reestablished as the inactivation gate opens and the activation gate closes. Note that repolarization (described in section 12.8c) triggers the voltage-gated Na\(^+\) channels to make this change (i.e., inactivate state to resting state).

**Modality gated channels** are an additional type of channel that is normally closed. These channels open (or close) in response to a stimulus other than a chemical or a voltage change. Modality gated channels are components of sensory neurons, which detect changes in the external or internal environment. For example, receptor cells of the skin contain modality gated channels that are stimulated by mechanical pressure to open (see section 16.2a) and receptor cells of the eye (photoreceptors) contain modality gated channels that are stimulated by light to close (see section 16.4d). Modality gated channels and their specific function in sensory perception (e.g., touch, vision) are described in more detail throughout chapter 16.

**WHAT DID YOU LEARN?**

16. Describe the three states of voltage-gated Na\(^+\) channels in neurons.

12.6b Distribution of Pumps and Channels

**LEARNING OBJECTIVES**

21. List the channels and pumps that are located along the entire neuron, and identify the general function of each.

22. Identify and describe the four functional neuron segments, including the distribution of channels and pumps in each.

Some pumps and channels are located throughout the entire neuron plasma membrane, whereas others are primarily located only in specific segments of a neuron’s plasma membrane. Their distribution is related to function. Here we discuss the distribution of the types of pumps and channels within the neuron plasma membrane (Figure 12.12). Note that the distribution of channels and pumps shown has been simplified for the purposes of this discussion and reflects their primary location in a typical multipolar neuron.

**Entire Plasma Membrane of a Neuron**

Na\(^+\) leak channels, K\(^+\) leak channels, and Na\(^+\)/K\(^+\) pumps are located throughout the entire neuron plasma membrane. These specific leak channels and pumps are important in establishing and maintaining the resting membrane potential (RMP) (see section 4.4b) of neurons (described in section 12.7b).

**Plasma Membrane of Functional Segments in a Neuron**

A typical neuron is functionally organized into four segments: receptive segment, initial segment, conductive segment, and transmissive segment. Each region differs in the primary types of channels and pumps located within its plasma membrane:

- **The receptive segment** includes both dendrites and the cell body, which are the regions of the neuron that receive stimuli to excite the neuron. Chemically gated channels (cation channels, K\(^+\) channels, and Cl\(^-\) channels) are located in this segment; no significant numbers of voltage-gated channels are present. (Note that cation channels allow the passage of both Na\(^+\) into the neuron and K\(^+\) out of the neuron. However, more Na\(^+\) moves into the neuron than K\(^+\) moves out.)

- **The initial segment** is commonly considered to be the region of the axon hillock. This segment contains both voltage-gated Na\(^+\) channels and voltage-gated K\(^+\) channels. (Note: Recent studies involving neuron function suggest that the specific location of the initial segment may vary and in some cases may be located within the beginning regions of the axon. We will use the convention of equating the initial segment with the axon hillock.)

**INTEGRATE LEARNING STRATEGY**

Entry of Na\(^+\) is prevented by either one of the two gates being closed, but the two gates are not both closed at the same time.

- **Resting state:** Activation gate is closed, which prevents Na\(^+\) entry.
- **Inactivation state:** Inactivation gate is closed, which prevents Na\(^+\) entry.
Figure 12.12 Distribution of Pumps and Channels in the Plasma Membrane of a Neuron. (a) Na\(^{+}/K^{+}\) pumps, Na\(^{+}\) leak channels, and K\(^{+}\) leak channels are found throughout the entire plasma membrane of a neuron. Additional types of channels and pumps are present only in specific functional segments of the neuron, as shown (b–e). Note that this represents a simplified depiction of the distribution of channels and pumps in a multipolar neuron plasma membrane.
The conductive segment is equivalent to the length of the axon. Like the initial segment, it contains both voltage-gated Na\(^+\) channels and voltage-gated K\(^+\) channels.

The transmissive segment includes the synaptic knobs and contains both voltage-gated Ca\(^{2+}\) channels and Ca\(^{2+}\) pumps.

**WHAT DID YOU LEARN?**

Which functional segment of a neuron contains chemically gated channels? Which functional segments contain voltage-gated channels?

### 12.7 Introduction to Neuron Physiology

Initiating and transmitting electrical currents is central to the function of neuron physiology. Necessary to this process is establishing and maintaining a resting membrane potential that can be changed from its resting value. Here we first describe both the basic principles of electric currents (and Ohm’s law) and the physiologic conditions of a neuron at rest.

#### 12.7a Neurons and Ohm’s Law

**LEARNING OBJECTIVE**

23. Integrate the concepts of voltage, current, and resistance with neuron structure and function.

Recall from section 3.1 that electrical energy is the movement of charged particles, and that all usable forms of energy are available to do work. Neuron activity is dependent upon electrical energy, specifically electric currents. Three important and relevant characteristics are associated with electric currents: voltage, current, and resistance.

- **Voltage** is the measure of the amount of difference in electrical charge between two areas and represents potential energy. The unit of measurement is volts (V) or millivolts (mV); 1 V = 1000 mV. The larger the difference in charge, the higher the voltage. You may have noticed that batteries are available in different sizes (e.g., a small battery [1.5 V] or a large battery [12 V]). The voltage is an indication of the relative potential energy stored in the battery.

- **Current** is the movement of charged particles across the barrier that separates this charge difference. The greater the movement of charged particles, the greater the current. Movement of charged particles (or current) is electrical energy that can be harnessed to do work (e.g., a current of electrons occurs between the positive and negative terminals in a battery when placed in a flashlight and the flashlight is turned on).

- **Resistance** is the opposition to the movement of charged particles. This is the barrier between the charged areas. The larger the resistance, the lower the current.

The relationship of voltage, current, and resistance is expressed by **Ohm’s law**:

\[
\text{Current} = \frac{\text{Voltage}}{\text{Resistance}}
\]

This expression shows that current is directly related to voltage and inversely related to resistance. Thus, a greater current is possible with a larger voltage difference and a lower resistance.

Let us relate the general concepts of electric currents to neurons (figure 12.13). In neurons,

- Charged particles are ions such as Na\(^+\) and K\(^+\) (not as in a battery or with electricity, which involves the flow of electrons).

- There is a difference in charge on either side of the plasma membrane (voltage) due to an unequal distribution of ions (figure 12.13a).

- The plasma membrane phospholipid bilayer offers resistance because it generally does not allow the passage of ions (figure 12.13b).

- Resistance is altered across the plasma membrane through ion channels that open and close. Resistance is decreased when ion channels are open and increased when ion channels are closed.

- Current is generated when either positively charged ions or negatively charged ions diffuse across the plasma membrane through open channels (figure 12.13c).

**INTEGRATE**

**CONCEPT CONNECTION**

Potential energy is the energy due to position (see section 3.1a). Recall that ion gradients represent potential energy because of their position relative to the plasma membrane. Kinetic energy is the energy of motion. Ions can flow across the plasma membrane through open channels, establishing ion currents (see figure 3.1a). It is this movement of ions (which is a type of kinetic energy) that is harnessed when electrical signals (i.e., graded potentials and action potentials) are transmitted in a neuron.
Neurons and Ohm’s Law. A neuron exhibits (a) voltage at its plasma membrane because of the charge difference, (b) resistance at the phospholipid bilayer of its plasma membrane because these molecules prevent the movement of ions across the plasma membrane, and (c) current when ion channels open to allow ions to flow across the plasma membrane.

WHAT DID YOU LEARN?
What is the role of ions, the phospholipid bilayer, and plasma membrane channels in neurons relative to the concepts of current, voltage, and resistance?

12.7b Neurons at Rest

**LEARNING OBJECTIVES**

24. Describe the conditions of a neuron at rest.

25. Define resting membrane potential, and state its typical value for neurons.

26. Explain how the resting membrane potential is established and maintained in neurons.

Neurons at rest have several important characteristics (figure 12.14a):

- Ion concentration gradients exist for K⁺, Na⁺, and Cl⁻ across the plasma membrane along the entire neuron. At the plasma membrane, there is relatively more K⁺ in the cytosol than in the interstitial fluid (IF) surrounding the neuron, whereas there is more Na⁺ and Cl⁻ in the IF than in the cytosol. These gradients are established by Na⁺/K⁺ pumps that move three Na⁺ out of the neuron for every two K⁺ moved in. (Chloride ion follows the movement of Na⁺.)

- A Ca²⁺ concentration gradient exists at the synaptic knob. Calcium pumps within this segment continuously pump Ca²⁺ from within the synaptic knob to the surrounding IF. Thus, there is more Ca²⁺ in the IF outside the synaptic knob than within the cytosol in the synaptic knob.

- Gated channels are closed. These channels include the chemically gated channels in the receptive segment, the voltage-gated Na⁺ channels and voltage-gated K⁺ channels in both the initial segment and the conductive segment, and voltage-gated Ca²⁺ channels in the transmissive segment.

- There is an electrical charge difference (an electrical gradient) across the plasma membrane; the cytosol adjacent to the plasma membrane is relatively negative in comparison to the IF outside of the cell. This electrical charge difference is called a **membrane potential**. When the neuron is at rest, the membrane potential is more specifically called the **resting membrane potential (RMP)** (see section 4.4). The RMP of a neuron is typically ~70 millivolts (mV), but can range between ~40 mV and ~90 mV.

A voltmeter is used to measure the voltage difference across the neuron plasma membrane. This is done by placing one microelectrode into the neuron and the other microelectrode outside the neuron in the interstitial fluid (figure 12.14b). The value of the voltage difference is negative (typically ~70 mV in neurons) because the voltage of the cytosol at the plasma membrane is relatively negative compared to the voltage measured in the IF outside the plasma membrane—that is, more positive ions reside outside a neuron than just inside a
neuron when it is at rest. (Consider the simplified example of having 100+ on the outside of a cell and 30+ on the inside of the cell; the inside has a relatively negative value of –70.)

Membrane potentials were described in detail in section 4.4. Here we review how a resting membrane potential is both established and maintained and apply these concepts to neurons. See figure 12.14b as you read through this section.

Establishing and Maintaining the Resting Membrane Potential

Establishing and maintaining the RMP is dependent upon the distribution of ions (as just described) as well as additional substances. These include negatively charged phosphate ions (P_i) as components of organic molecules (e.g., ATP) and negatively charged protein molecules. Both are more prevalent within a neuron’s cytosol than in the surrounding IF.

The RMP is chiefly a consequence of the movement of ions across the plasma membrane through leak channels (both K^+ leak channels and Na^+ leak channels).

The Role of K^+

Potassium diffusion is the most important factor in establishing the specific value of the resting membrane potential. K^+ movement is dependent upon its electrochemical gradient, which is the combination of the electrical gradient at the plasma membrane and the K^+ chemical concentration gradient. Potassium ions exit the neuron via K^+ leak channels moving down their relatively steep chemical concentration gradient moving into the IF. The loss of K^+ leaves relatively more negatively charged structures (e.g., P_i, proteins) inside the cell. These structures remain within the cell because they are too large to cross the plasma membrane.
The movement of K\(^+\) to the outside of a cell is, however, opposed by the electrical gradient. The positive charge on the outside of the cell repels the movement of K\(^+\), and the negative charge on the inside of the cell attracts K\(^+\). Thus, K\(^+\) movement is facilitated by its chemical concentration gradient but opposed by the electrical gradient. As additional K\(^+\) diffuses out of the neuron, the inside becomes more negative. Consequently, the pull to keep K\(^+\) in the cell is greater. At some point, the force of the electrical gradient that opposes K\(^+\) movement becomes equal to the force of the chemical gradient allowing K\(^+\) out of a cell. Thus, K\(^+\) movement has reached equilibrium. If only K\(^+\) leak channels were present in neurons, the loss of the K\(^+\) would result in an RMP with a value of −90 mV. This charge difference of −90 mV across the plasma membrane would represent the membrane potential.

**The Role of Na\(^+\)** The typical neuron RMP is −70 mV. The difference between −90 mV established by K\(^+\) movement only and the RMP of −70 mV is primarily the result of Na\(^+\) movement into a neuron. The Na\(^+\) enters the cell through Na\(^+\) leak channels. It moves down its chemical concentration gradient, and it is “pulled” into the cell by the electrical gradient. Both forces facilitate the movement of Na\(^+\) into a neuron. However, limited numbers of Na\(^+\) leak channels (as compared to K\(^+\) leak channels) prevent as much Na\(^+\) moving into the neuron as K\(^+\) moves out.

**The Role of Na\(^+\)/K\(^+\) Pumps** The Na\(^+\)/K\(^+\) pumps play a relatively small role in establishing a resting membrane potential. These pumps contribute approximately −3 mV of the total −70 mV difference by moving more Na\(^+\) out of the neuron than K\(^+\) is pumped into the neuron. The Na\(^+\)/K\(^+\) pumps have a more significant role in maintaining the concentration gradients of both K\(^+\) and Na\(^+\). These concentration gradients allow for the diffusion of Na\(^+\) and K\(^+\) as part of the neuron’s generation of an electric current, as described in section 12.8.

**WHAT DID YOU LEARN?**

19. Describe the conditions of a neuron at rest in terms of the RMP, concentration gradients for Na\(^+\), K\(^+\), Cl\(^−\) along the entire neuron and Ca\(^2+\) at the synaptic knob; and the state of the gated channels.
20. Explain how an RMP is established and maintained in neurons.

### 12.8 Physiologic Events in the Neuron Segments

Here we discuss the physiologic events that occur in the functional neuron segments from the time of initial stimulation at the dendrites and cell body until release of a neurotransmitter from a synaptic knob. An overview of the events that occur in the receptive, initial, conductive, and transmissive segments is provided in figure 12.15.

#### 12.8a Receptive Segment

**LEARNING OBJECTIVES**

27. Describe a postsynaptic potential.
28. Compare and contrast the action of neurotransmitters in developing both excitatory and inhibitory postsynaptic potentials (graded potentials) in the receptive segment.
29. Graph and explain an excitatory postsynaptic potential (EPSP) and an inhibitory postsynaptic potential (IPSP).

We begin our discussion with figure 12.16, which shows the arrangement of several presynaptic neurons in close proximity to one postsynaptic neuron. Between each presynaptic and postsynaptic neuron is a synaptic cleft that is the fluid-filled space between them. Each presynaptic neuron releases neurotransmitter that binds with receptors (chemically gated channels) in the receptive segment—dendrites and cell body—of a postsynaptic neuron.

The establishment of graded potentials is the significant event that occurs in the receptive segment of a neuron. **Graded potentials** are relatively small (less than 1 mV), short-lived changes in the resting membrane potential that are caused by the movement of small amounts of ion across the plasma membrane. Graded potentials have the following characteristics:

**Graded potentials are established in the receptive segment by the opening of chemically gated channels.** Recall that there are three types of chemically gated channels in the receptive segment of a neuron: chemically gated cation channels, chemically gated K\(^+\) channels, and chemically gated Cl\(^−\) channels. Neurotransmitter released from a presynaptic neuron binds with a specific type of chemically gated channel, triggering it to open, which temporarily allows passage of a small amount of a specific type of ion (or ions) across the plasma membrane. The ions then move along the plasma membrane in a local current.
The local currents associated with graded potentials are short-lived (1 millisecond to a few milliseconds) because the flow or current of ions along the plasma membrane experiences resistance. Consider that when cation channels open, and Na⁺ enters a cell and moves along the inside of the plasma membrane, Na⁺ experiences resistance from the contents of the cytosol. Consequently, the local current of Na⁺ becomes weaker and eventually ceases. Thus, a graded potential lasts only as long as the channels are open and until the local ion current ceases.

Graded potentials vary in both the degree of change and the direction of change of the RMP. The degree of change is dependent upon the magnitude of the stimulus. A larger stimulus opens more chemically gated channels, and more ions flow across the plasma membrane than occurs during a weaker stimulus. Thus, the larger the stimulus, the more channels that open, the greater the flow of ions, and the larger the current. (The term graded in graded potentials reflects this difference in magnitude.) The direction of change (i.e., whether the membrane potential becomes more positive or more negative) is dependent upon the type of chemically gated channel that opens. For example, the opening of chemically gated cation channels allows more Na⁺ (positively charged ion) to enter the neuron (than K⁺ to exit), causing the inside of the neuron to become more positive (e.g., −70 mV to −69 mV). This change in the membrane potential in the positive direction is called depolarization (dē-pōˈlər-ə-tĭshən; de = away). In contrast, the opening of either chemically gated K⁺ channels (which allows positively charged potassium ions [K⁺] to exit the neuron) or chemically gated Cl⁻ channels (which allows negatively charged chloride ions [Cl⁻] to enter the neuron) cause the inside of the neuron to become more negative (e.g., −70 mV to −71 mV). This change in the membrane potential in the negative direction is called hyperpolarization (hīˈpər-pōˈlər-ə-tĭshən; hyper = above).

Graded potentials that occur in postsynaptic neurons are specifically called postsynaptic potentials. Postsynaptic potentials that result in the neuron becoming more positive (i.e., depolarized) are more specifically called excitatory postsynaptic potentials (EPSPs), whereas those that result in the neuron becoming more negative (i.e., hyperpolarized) are called inhibitory postsynaptic potentials (IPSPs). Note that numerous postsynaptic potentials—both excitatory and inhibitory—are typically generated because a postsynaptic neuron can bind many neurotransmitter molecules simultaneously.
Figure 12.16 Postsynaptic Potentials in the Receptive Segment. Neurotransmitter released from presynaptic neurons crosses the synaptic cleft and initiates a graded (postsynaptic) potential (which is a local electrical change from the resting membrane potential [RMP]). Binding of a neurotransmitter causes either (a) an excitatory postsynaptic potential (EPSP) or (b) an inhibitory postsynaptic potential (IPSP), depending upon the neurotransmitter released and the specific type of receptor to which it binds.

**Generation of EPSP**

1. Neurotransmitter released from presynaptic neurons binds to postsynaptic neuron receptors, which are chemically gated cation channels, causing them to open.

   ![Diagram](image)

   - **Presynaptic neurons**
   - **Neurotransmitter**
   - **Postsynaptic neuron**
   - **Chemically gated cation channel**
   - **RMP = -70 mV**
   - **Na⁺ flows into neuron (faster than K⁺ flows out).**
   - **Inside of neuron becomes more positive (less negative); called EPSP (e.g., -68 mV).**
   - **EPSP moves toward the initial segment.**

**Generation of IPSP**

1. Neurotransmitter released from presynaptic neurons binds to postsynaptic neuron receptors, which are either chemically gated K⁺ channels or chemically gated Cl⁻ channels, causing them to open.

   ![Diagram](image)

   - **Presynaptic neurons**
   - **Neurotransmitter**
   - **Postsynaptic neuron**
   - **Chemically gated K⁺ channel**
   - **Chemically gated Cl⁻ channel**
   - **RMP = -70 mV**
   - **Either K⁺ flows out of or Cl⁻ flows into the neuron, depending on the type of channel stimulated.**
   - **Inside of neuron becomes more negative; called IPSP (e.g., -72 mV).**
   - **IPSP moves toward the initial segment.**
Generation of an EPSP

The release of neurotransmitter from a presynaptic neuron may result in an EPSP (figure 12.16a) as described here:

1. The neurotransmitter crosses the synaptic cleft and binds specifically to a postsynaptic neuron receptor that is a chemically gated cation channel, causing it to open.

2. These channels allow both Na⁺ and K⁺ to move down their respective concentration gradients. However, more Na⁺ moves into the neuron than K⁺ moves out. The amount of neurotransmitter determines the number of chemically gated cation channels that open.

3. Consequently, the inside of the neuron becomes slightly more positive, or less negative, by the net gain of positively charged ions. This temporary, less negative state is called an excitatory postsynaptic potential (EPSP). (See graph in figure 12.16a.)

4. The local current of Na⁺ becomes weaker as it moves along the neuron plasma membrane toward the initial segment and decreases in intensity with the distance traveled from the site of neurotransmitter binding.

Generation of an IPSP

The release of neurotransmitter from a presynaptic neuron may instead result in an IPSP (figure 12.16b), as described here:

1. The neurotransmitter crosses the synaptic cleft and binds to a postsynaptic neuron chemically gated K⁺ channel or a chemically gated Cl⁻ channel, depending upon the neurotransmitter and channels present.

2. If the neurotransmitter binds to a receptor that is a chemically gated K⁺ channel, this channel opens and K⁺ moves out of the neuron down its concentration gradient, causing a loss of positively charged ions. In contrast, if the neurotransmitter binds to a receptor that is a chemically gated Cl⁻ channel, opening of this channel allows Cl⁻ to flow down its concentration gradient to move into the neuron, causing the gain of negatively charged ions. The amount of neurotransmitter determines the number of either chemically gated K⁺ or chemically gated Cl⁻ channels that open.

3. Consequently, the inside of the cell becomes slightly more negative if either chemically gated K⁺ channels open and K⁺ exits or chemically gated Cl⁻ channels open and Cl⁻ enters. This temporary, more negative state is called an inhibitory postsynaptic potential (IPSP). (See graph in figure 12.16b.)

4. The local current of ions becomes weaker as it moves along the neuron plasma membrane toward the initial segment and decreases in intensity with the distance traveled from the site of neurotransmitter binding.

The degree of change in the RMP is dependent upon the amount of neurotransmitter bound per unit of time. As more neurotransmitter is released by presynaptic neurons, more channels open in the receptive segment of the postsynaptic neuron, and there is a greater change in the membrane potential.

Figure 12.17 shows both an illustration (figure 12.17a) and a photo (figure 12.17b) of numerous presynaptic neurons with a postsynaptic neuron. Neurotransmitter is typically being released from multiple presynaptic neurons and quickly released from the same presynaptic neuron over a very short period of time. The result is many EPSPs, many IPSPs, or both being generated simultaneously (or within a narrow period of time) within the receptive segment. The outcome (or effect) of these EPSPs and IPSPs is determined in the initial segment.

What do you think?

1. Does the generation of IPSPs make it more likely or less likely that an action potential (a nerve signal) will be sent?

What did you learn?

21. How are EPSP and IPSP graded potentials established in the receptive segment of a neuron?

12.8b Initial Segment

Learning Objective

30. Define summation, and describe the two types of summation that can occur in the initial segment.

The local currents of ions associated with the graded potentials (Na⁺ ion currents with EPSPs and K⁺ and Cl⁻ ion currents with IPSPs) that are established in the receptive segment move along the plasma membrane toward the initial segment. The outcome of these multiple local currents is determined when they arrive in the initial segment (axon hillock). The changes in the membrane potential associated with these graded postsynaptic potentials are “added” in the initial segment to determine if an action potential is initiated. The process is called summation. The sensitivity of voltage-gated channels to open in response to a minimum voltage change in membrane potential is the determining factor if an action potential is initiated. The minimum voltage change is called the threshold membrane potential. On average, the value for the threshold membrane potential is about −55 mV (although the specific value does vary somewhat between different types of neurons). This is a change of +15 mV from the RMP. When this threshold is reached, the voltage-gated channels are stimulated to open, which will initiate the generation of an action potential that will be propagated along the axon.

A single EPSP is incapable of causing the postsynaptic neuron to reach threshold. Additionally, IPSPs negate the effect of EPSPs. The occurrence of both EPSPs and IPSPs together results in a “tug-of-war” as to whether the threshold is reached. Thus, numerous EPSPs must be generated in the receptive segment and arrive at the initial segment simultaneously, or nearly at the same time, if the threshold is to be reached.

Threshold can be reached through two types of summation, called spatial summation and temporal summation, which may act in concert to produce an effect:

- Spatial summation occurs when multiple presynaptic neurons release neurotransmitter at various locations onto the receptive segment, thus generating EPSPs, IPSPs, or both in the postsynaptic neuron. Figure 12.17c shows the possible outcomes if just two postsynaptic potentials established by different presynaptic neurons reach the initial segment simultaneously. Two EPSPs (established by P1 and P2) are added together, and both move the membrane potential toward the threshold; two IPSPs (established by P6 and P7) are added together to move the membrane potential away from the threshold; and if an EPSP (established by P1) and IPSP (established by P6) arrive simultaneously, the EPSP (established by P1), which moves the membrane potential toward the threshold, is canceled out by the IPSP (established by P6), which moves the membrane potential away from the threshold.
Spatial summation: Different presynaptic neurons initiate postsynaptic potentials within a narrow period of time.

**Two simultaneous EPSPs sum to produce a greater EPSP**

<table>
<thead>
<tr>
<th>P1 Stimulated</th>
<th>P2 Stimulated</th>
<th>P1 + P2 Stimulated</th>
</tr>
</thead>
<tbody>
<tr>
<td>-65</td>
<td>-70</td>
<td>-65</td>
</tr>
<tr>
<td>-75</td>
<td>-70</td>
<td>-65</td>
</tr>
</tbody>
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**Two simultaneous IPSPs sum to produce a greater IPSP**

<table>
<thead>
<tr>
<th>P6 Stimulated</th>
<th>P7 Stimulated</th>
<th>P6 + P7 Stimulated</th>
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</thead>
<tbody>
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<td>-70</td>
<td>-65</td>
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<tr>
<td>-75</td>
<td>-70</td>
<td>-65</td>
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**A simultaneous IPSP and EPSP cancel each other out**

<table>
<thead>
<tr>
<th>P1 Stimulated</th>
<th>P6 Stimulated</th>
<th>P1 + P6 Stimulated</th>
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<tbody>
<tr>
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<td>-70</td>
<td>-65</td>
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<tr>
<td>-75</td>
<td>-70</td>
<td>-65</td>
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Temporal summation: The same presynaptic neuron initiates postsynaptic potentials rapidly within a narrow period of time.

**Two EPSPs elicited in rapid succession sum to produce a larger EPSP**

<table>
<thead>
<tr>
<th>P1</th>
<th>P1 + P1</th>
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<tbody>
<tr>
<td>-65</td>
<td>-65</td>
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<td>-70</td>
<td>-70</td>
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**Two IPSPs elicited in rapid succession sum to produce a larger IPSP**

<table>
<thead>
<tr>
<th>P6</th>
<th>P6 + P6</th>
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<td>-65</td>
<td>-65</td>
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<tr>
<td>-70</td>
<td>-70</td>
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**Two simultaneous EPSPs sum to produce a greater EPSP**

<table>
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<tr>
<th>P1 Stimulated</th>
<th>P2 Stimulated</th>
<th>P1 + P2 Stimulated</th>
</tr>
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<tbody>
<tr>
<td>-65</td>
<td>-70</td>
<td>-65</td>
</tr>
<tr>
<td>-75</td>
<td>-70</td>
<td>-65</td>
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</tbody>
</table>

**Two simultaneous IPSPs sum to produce a greater IPSP**

<table>
<thead>
<tr>
<th>P6 Stimulated</th>
<th>P7 Stimulated</th>
<th>P6 + P7 Stimulated</th>
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<tr>
<td>-65</td>
<td>-70</td>
<td>-65</td>
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<tr>
<td>-75</td>
<td>-70</td>
<td>-65</td>
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</table>

**A simultaneous IPSP and EPSP cancel each other out**

<table>
<thead>
<tr>
<th>P1 Stimulated</th>
<th>P6 Stimulated</th>
<th>P1 + P6 Stimulated</th>
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<tr>
<td>-65</td>
<td>-70</td>
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<td>-75</td>
<td>-70</td>
<td>-65</td>
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**Temporal summation** occurs when a single presynaptic neuron repeatedly releases neurotransmitter to produce either multiple EPSPs or IPSPs in the postsynaptic neuron at the same location within a very short period of time. Figure 12.17d shows the possible outcomes if one presynaptic neuron rapidly establishes two postsynaptic potentials (which, because they are established by the same presynaptic neuron, must be either two EPSPs or two IPSPs). Two EPSPs (both established by P1) are added together, and both move the membrane potential toward the threshold; two IPSPs (both established by P6) are added together to move the membrane potential away from the threshold.
Typically, both spatial summation and temporal summation are occurring simultaneously. When the graded potentials arrive at the initial segment within a small period of time, they can either contribute to (if EPSPs) or interfere with (if IPSPs) the threshold value being reached (as shown in figure 12.17c, d). If the threshold value is reached, an action potential is initiated. (Graded potentials typically last for approximately 15 milliseconds.) Any change in voltage below the threshold value is not sufficient to open voltage-gated channels and is called a subthreshold value; these channels remain closed and no action potential is initiated. (See the Learning Strategy on summation to help you with this concept.)

The all-or-none law (see section 10.6b) applies to action potentials propagated along the plasma membrane of neurons. If the threshold is reached, an action potential is initiated and propagated along the axon without decreasing in intensity (all). If only a subthreshold value is reached, it is not initiated (none). Additionally, voltage changes with a value greater than the threshold (e.g., voltage change of +20 mV) result in the same intensity in the action potential that is initiated as occurs when the threshold is reached. The initial segment is sometimes called the trigger zone because what occurs there is similar to what happens when firing a gun. Sufficient pressure is applied to the trigger of a gun and the bullet is fired from the chamber, or insufficient pressure is placed on the trigger and the gun is not fired. Likewise, the bullet travels at the same velocity regardless of whether the pressure placed on the trigger is greater than needed.

**WHAT DID YOU LEARN?**

What is the significance of the threshold membrane potential in the initial segment of a neuron?

12.8c Conductive Segment

**LEARNING OBJECTIVES**

31. Describe and graph an action potential.

32. Explain propagation of an action potential in both unmyelinated and myelinated axons.

33. Define refractory period, and explain the difference between the absolute refractory period and relative refractory period associated with transmitting an action potential.

The conductive segment is equivalent to the total length of the axon. The main activity of the conductive segment is propagation of an action potential along the axolemma (the axon plasma membrane). An action potential involves two processes: depolarization and repolarization. We first introduced the term depolarization in section 12.8a as the gain of positive ions that results in the membrane potential becoming more positive (e.g., −70 mV to −69 mV). Here, the term depolarization more specifically refers to the gain of positive charge within a neuron that occurs to such an extent to change the plasma membrane potential from negative to positive. This reversal of polarity is due to the opening of voltage-gated Na⁺ channels and the subsequent movement of Na⁺ into the cell. Repolarization (rē-pōl-ē-rā-zhūn) is the return of polarity from positive back to negative (the RMP). Repolarization is due to the opening of voltage-gated K⁺ channels and the subsequent movement of K⁺ out of the cell.

Once initiated, action potentials are propagated along an axon to the synaptic knob as both voltage-gated Na⁺ channels and voltage-gated K⁺ channels open sequentially along the length of the axolemma. The propagation of an action potential is called a nerve signal or nerve impulse. (Note that voltage-gated K⁺ channels are opening directly behind the opening of voltage-gated Na⁺ channels. Thus, repolarization directly follows depolarization within any segment of an axon.) The details of both depolarization and its propagation and repolarization and its propagation are described here in detail. Refer to figure 12.18 as you read through this section.

Depolarization and Its Propagation

The following events are associated with depolarization and its propagation (figure 12.18a):

1. Initially, the voltage-gated Na⁺ channels are closed and the membrane potential is −70 mV (the resting membrane potential).
2. Sodium ions flow within the cytosol into the region from adjacent areas. The membrane potential becomes more positive moving away from −70 mV. Voltage-gated Na⁺ channels are triggered to open when sufficient Na⁺ flows into the region to change the membrane potential from −70 mV to −55 mV (the threshold value).
Figure 12.18 Generation of an Action Potential. An action potential involves (a) depolarization, which occurs as $\text{Na}^+$ moves into the axon through open voltage-gated $\text{Na}^+$ channels to change the membrane potential from $-70 \text{ mV}$ to $+30 \text{ mV}$, and (b) repolarization, which occurs as $\text{K}^+$ moves out of the axon through open voltage-gated $\text{K}^+$ channels to reverse the polarity from $+30 \text{ mV}$ to $-70 \text{ mV}$. (Refer to figure 12.11 to review states of voltage-gated $\text{Na}^+$ channels.)
Voltage-gated Na⁺ channels remain open to allow rapid Na⁺ entry into the axon to cause depolarization. Sufficient Na⁺ enters the axon to reverse the membrane potential from negative (−55 mV) to positive (+30 mV). (Note that the exact value can vary from 0 mV to +50 mV.) This movement of Na⁺ is extremely small, representing a change of approximately 0.01% in the Na⁺ concentration, but is sufficient to cause depolarization at the plasma membrane.

The voltage-gated Na⁺ channels are opened for only a very short duration and then close, changing from the activation state to the temporary inactivation state. This temporarily prevents their reopening.

Steps 1–4 are repeated in adjacent regions downstream from the cell body (farther away from the cell body) as Na⁺ flows within the cytosol to adjacent regions of the axon’s plasma membrane, causing these regions downstream to also become more positive and to reach threshold value (−55 mV). (Note that only the voltage-gated Na⁺ channels downstream are triggered to open. The voltage-gated Na⁺ channels upstream are temporarily in the inactivation state and are prevented from reopening: thus, the action potential is propagated in only one direction—away from the cell body.)

Adjacent depolarization is repeated rapidly down the plasma membrane of the axon toward the synaptic knob, and propagation of depolarization occurs. The propagation of depolarization is analogous to the tipping of a row of standing dominoes. Once the first domino is initiated to fall, each domino falls in sequence until the last domino is reached.

Re polarization and Its Propagation

Simultaneously, the following events that are associated with repolarization and its propagation are occurring in each segment of the axon directly behind the events of depolarization (figure 12.18b):

The reaching of the threshold value (−55 mV) also triggers voltage-gated K⁺ channels to open. These channels are relatively slow to open and are not completely open until about the point that depolarization has ended. Voltage-gated K⁺ channels remain open to allow rapid K⁺ exit from the axon to cause repolarization. Sufficient K⁺ exits the axon to change the membrane potential from positive (+30 mV) to its negative RMP (−70 mV). (Note that repolarization triggers the voltage-gated Na⁺ channels to change from the inactivation state to the resting state. This allows voltage-gated Na⁺ channels to be stimulated to open again to send a new nerve signal; see section 12.6a.)

The voltage-gated K⁺ channels typically remain open longer than the time needed to reestablish the resting membrane potential (−70 mV). The inside of the neuron during this brief time is more negative than the RMP, or hyperpolarized (decreasing to approximately −80 mV).

Steps 5–7 are repeated in adjacent regions downstream from the cell body (as adjacent voltage-gated K⁺ channels open and then close). Consider that propagation of repolarization is similar to the resetting up of a row of dominoes so that the process can happen again. Observe figure 12.21a, which visually presents the events of an action potential in both depolarization and repolarization, including how repolarization directly follows the events of depolarization in each region of the axon as the action potential is propagated along the axon. The electrical changes of an action potential can be measured by electrodes placed on the inside and outside of the plasma membrane (figure 12.18). These electrical changes, which are sometimes called a spike potential, are shown and described in figure 12.19.

**Figure 12.19 Events of an Action Potential.** A tracing of the membrane voltage changes (measured in millivolts [mV]) associated with an action potential initiated in the initial segment. Changes occur in just a few milliseconds, result from the opening and closing of voltage-gated Na⁺ channels and voltage-gated K⁺ channels in the plasma membrane of the axon, and are sometimes referred to as a spike potential, reflecting the shape of the recorded potential.
A refractory period is the brief time period after an action potential has been initiated during which an axon is either incapable of generating another action potential or a greater than normal amount of stimulation is required to generate another action potential. The excitable neuron plasma membrane recovers at this time and becomes ready to respond to another stimulus. The refractory period has two phases: the absolute refractory period and the relative refractory period (figure 12.20).

The absolute refractory period is the time (about 1 millisecond) after an action potential onset when no amount of stimulus, no matter how strong, can initiate a second action potential. During this time, the voltage-gated Na\(^+\) channels are first opened to cause depolarization, and then voltage-gated K\(^+\) channels are open to cause repolarization. Repolarization triggers the voltage-gated Na\(^+\) channels to change from the inactivated state to the resting state (see section 12.6a). Consequently, no voltage difference across the plasma membrane of an axon during this time can open the voltage-gated Na\(^+\) channels for the next action potential until repolarization has occurred. The absolute refractory period ensures that the action potential moves along the axon in only one direction toward the synaptic knobs. (See steps 3 and 4 in figure 12.20.)

The relative refractory period occurs immediately after the absolute refractory period. Another action potential can now be initiated in an axon only if the stimulation of the plasma membrane is greater than the stimulus normally needed to generate an action potential. At this time, voltage-gated Na\(^+\) channels have returned to their resting state, but the neuron is hyperpolarized due to the slightly extended time that voltage-gated K\(^+\) channels remain open during repolarization. (See step 5 in figure 12.20.)

Continuous Conduction and Saltatory Conduction

Specifically how an action potential is propagated along the axon is dependent upon whether the axon is unmyelinated or myelinated. Continuous conduction occurs in unmyelinated axons and involves the sequential opening of voltage-gated Na\(^+\) channels and voltage-gated K\(^+\) channels located within the axon plasma membrane along the entire length of the axon. When previously discussing the process of action potential conduction, we described it as it would occur in an unmyelinated axon (figure 12.21a). Saltatory (sal’ta-tôr’ë; saltare = to jump) conduction occurs in myelinated axons (figure 12.21b). Here, action potentials do not occur in regions of the axon that are myelinated—rather, they are propagated only at neurofibril nodes. This is due to anatomic differences in the two types of regions of a myelinated axon. Myelinated regions of an axon contain limited numbers of voltage-gated Na\(^+\) channels and voltage-gated K\(^+\) channels, and myelin is a great insulator that prevents ion movement across the plasma membrane even if additional channels are

Figure 12.20 Refractory Periods.
The absolute refractory period is from the point of depolarization until repolarization is almost complete. During this time, it is not possible to initiate another action potential, no matter how strong the stimulus. The relative refractory period is the time immediately following the absolute refractory period, during which a stronger than normal stimulus is required to initiate another action potential because the neuron is hyperpolarized (below the RMP of –70 mV).
A new action potential occurs at the next neurofibril node.

In unmyelinated axons, voltage-gated channels open sequentially along the entire length of an axon, and the process is called continuous conduction. In myelinated axons, voltage-gated channels open only at the neurofibril nodes, and the process is referred to as saltatory conduction.

**Figure 12.21 Propagation of an Action Potential.** Propagation of an action potential occurs from the initial segment to the synaptic knob. (a) In unmyelinated axons, voltage-gated channels open sequentially along the entire length of an axon, and the process is called continuous conduction. (b) In myelinated axons, voltage-gated channels open only at the neurofibril nodes, and the process is referred to as saltatory conduction.

Present. In contrast, neurofibril nodes have a relatively large number of both voltage-gated Na⁺ channels and voltage-gated K⁺ channels and lack myelin insulation. Ions are relatively free to flow into and out of the axon in these regions when the channels are open. A nerve signal is transmitted along a myelinated axon, as follows:

- **Action potential occurs at the neurofibril node.** Depolarization is due to opening of voltage-gated Na⁺ channels at the neurofibril node; Na⁺ diffuses into the axon. (This is followed by repolarization as voltage-gated K⁺ channels open, and K⁺ diffuses out.)

- **Na⁺ diffusion (but no action potential) occurs at the myelinated region of an axon.** The Na⁺ diffuses through axoplasm of the axon internal to the axolemma (which is insulated by myelin). Two critical aspects should be noted regarding this Na⁺ diffusion: (1) It is relatively fast, faster than the events at the neurofibril nodes, and (2) as Na⁺ diffuses through the axoplasm, it experiences resistance and the local current decreases in intensity (becomes weaker) with distance.

- **A new action potential occurs at the next neurofibril node.** The arrival of the relatively weak Na⁺ current at the next neurofibril node is sufficient to cause the opening of voltage-gated Na⁺ channels located there. This results in the establishing of a new action potential as Na⁺ enters the axon and a new local current is established. This process repeats as the nerve signal continues down the length of an axon until it reaches the synaptic knobs. The transmission of a nerve signal along an axon is called saltatory conduction because the action potential occurs only at neurofibril nodes; thus, it seems to “jump” from node to node (a distance of approximately 1 mm). Transmission of a nerve signal in a myelinated axon is much faster (120 meters per second) than in an unmyelinated axon (2 meters per second), because an action potential is generated only at the neurofibril nodes of myelinated axon rather than along the entire length of unmyelinated axon. Saltatory conduction also is more efficient because less energy is required by Na⁺/K⁺ pumps to maintain the RMP.
12.8d Transmissive Segment

LEARNING OBJECTIVES

34. Describe events that occur when the propagated action potential reaches the transmissive segment.

35. Explain the general role of Ca^{2+} in neurotransmitter release.

Recall that the transmissive segment is the synaptic knob of the neuron. The main activity that occurs at the transmissive segment is the release of neurotransmitter from synaptic vesicles (figure 12.22). Prior to the arrival of the nerve signal, Ca^{2+} pumps embedded in the plasma membrane of a synaptic knob (not shown in figure) establish a calcium concentration gradient by pumping Ca^{2+} out to the IF. Consequently, there is more calcium outside the synaptic knob than inside it.

When the nerve signal reaches the synaptic knob at the end of the axon, the voltage change associated with depolarization (+30 mV) triggers the opening of voltage-gated Ca^{2+} channels. Calcium ions move down their concentration gradient from the interstitial fluid into the synaptic knob. Calcium ions bind to proteins associated with synaptic vesicles, which triggers a series of events, resulting in fusion of synaptic vesicles with the plasma membrane of the synaptic knob. Neurotransmitter is subsequently released into the synaptic cleft by exocytosis (see section 4.3c). Approximately 300 vesicles are released per nerve signal. The neurotransmitter then diffuses across the cleft between the synaptic knob and the cell to be stimulated. There it binds to specific cellular protein receptors of another neuron or an effector organ (muscle or gland). Note that exocytosis of neurotransmitter is facilitated by numerous proteins (e.g., synaptotagmin, SNARE proteins), and research continues in this area to determine the exact functional role of each of these proteins.

CLINICAL VIEW 12.5

Neurotoxicity

Neurotoxicity is the damage caused to nervous tissue (neurons and glial cells) when exposed to neurotoxins. Neurotoxins can be substances produced within the body (e.g., beta amyloid, oxygen free radicals), substances from microbes (e.g., botulinum toxin, tetanus toxin), synthetic substances (e.g., pesticides, industrial solvents), ethanol, or chemicals used in medical treatment for chemotherapy, radiation treatment, and organ transplants.

Neurotoxins cause harm to nervous tissue by one of several mechanisms, including the following:

- Interfering with propagation of action potentials (e.g., tetrodotoxin, found in some fish and amphibians, blocks Na^{+} channels; agitoxin from scorpions interferes with K^{+} channels; lead causes loss of myelination primarily in somatic motor neurons that control skeletal muscle)
- Altering events that occur at a synapse (e.g., botulinum toxin, which causes botulism, blocks the release of acetylcholine from synaptic vesicles at the neuromuscular junction of skeletal muscle fibers; toxin produced by Clostridium tetani, which causes tetanus, blocks the release of inhibitory transmitters within the spinal cord, which results in overstimulation of the muscles and excessive muscle contractions)
- Inducing detrimental structural changes to a neuron that can result in the death of the neuron cell (e.g., mercury decreases protein synthesis; Alzheimer disease involves formation of neurofibrillary tangles)

Symptoms of neurotoxicity may include muscle weakness or spasticity, numbness (decreased ability to feel), loss of memory, impaired vision, decreased mental ability, headache, and behavioral problems.
It has been widely accepted that each neuron releases only one type of neurotransmitter. Today there is ample evidence to support that most neurons actually synthesize and release more than one type of neurotransmitter. However, each vesicle within a neuron typically contains only one type of neurotransmitter, and generally only one type of neurotransmitter is released at a time. The type of neurotransmitter released is dependent upon the frequency of action potentials that reach the synaptic knob. The physiologic processes of a neuron in the four major functional segments—the receptive segment, initial segment, conductive segment, and transmissive segment—are summarized in figure 12.23.

**WHAT DID YOU LEARN?**

25 What is the sequence of events from the arrival of the propagated action potential (a nerve signal) at the synaptic knob until the release of neurotransmitter into the synaptic cleft?
Figure 12.23 Events of Neuron Physiology. Neuron physiology involves specific events that occur in the four functional segments of a neuron: (1) receptive segment, (2) initial segment, (3) conductive segment, and (4) transmissive segment.

**INTEGRATE CONCEPT OVERVIEW**

**INITIAL SEGMENT: “Trigger Zone”**
Summation of EPSPs and IPSPs includes both spatial summation of two or more presynaptic neurons and temporal summation of one presynaptic neuron; rapidly releasing neurotransmitter determines if threshold (~55 mV) is reached.

**RECEPTIVE SEGMENT: Establishing Graded Potentials: EPSPs and IPSPs**

- Neurotransmitter is released from presynaptic neuron; it binds with chemically gated cation channels; more Na⁺ enters neuron than K⁺ exits and inside becomes more positive, which is an excitatory postsynaptic potential (EPSP).

- Neurotransmitter is released from presynaptic neuron, which binds with either chemically gated K⁺ channels, and K⁺ exits neuron or to chemically gated Cl⁻ channels, and Cl⁻ enters neuron. In either case, the inside becomes more negative, which is an inhibitory postsynaptic potential (IPSP).
3 CONDUCTIVE SEGMENT

Action potential

Depolarization: Opening of voltage-gated Na⁺ channels in response to reaching threshold. Na⁺ moves into axon.

Repolarization: Opening of voltage-gated K⁺ channels that immediately follows depolarization to reestablish RMP. K⁺ moves out of axon.

Nerve signal: Propagation of Action Potential

Action potentials are propagated at neurofibril nodes (in myelinated axons) and are propagated from the initial segment to the synaptic knob.

Diffusion of Na⁺ through axoplasm

Repolarization

Depolarization

Action potential

4 TRANSMISSIVE SEGMENT: Release of Neurotransmitter

Arrival of a nerve signal at the synaptic knob triggers the opening of voltage-gated Ca²⁺ channels. Ca²⁺ enters the synaptic knob, causing the subsequent release of neurotransmitter from synaptic vesicles by exocytosis.

Voltage-gated Ca²⁺ channel

Neurotransmitter

Synaptic vesicle

Neurotransmitter binds with receptors on either another neuron or an effector (muscle or gland).
12.9 Characteristics of Action Potentials

Here we compare graded potentials and action potentials (the two types of electrical signals in neurons) and describe several aspects of action potential propagation, including velocity of action potentials and frequency of action potentials.

12.9a Graded Potentials Versus Action Potentials

**LEARNING OBJECTIVE**

36. Compare graded potentials and action potentials.

Recall that two types of electrical signals are associated with neurons—graded potentials and action potentials. Graded potentials (described in section 12.8a) occur in the receptive segment of a neuron (dendrites and cell bodies) and are due to the opening of chemically gated channels. The chemically gated channels open temporarily to allow passage of a relatively small amount of a specific type of ion across the plasma membrane. This results in the membrane potential becoming either more positive (depolarization) or more negative (hyperpolarization) than the resting membrane potential. The degree of change is dependent upon the magnitude of the stimulus; thus, it is graded. A larger stimulus opens more chemically gated channels, and more ions flow across the plasma membrane than occurs during a weaker stimulus. The established local current of ions exhibits decreased intensity (decreased flow of ions) as the ions move along the plasma membrane; thus, graded potentials are short-lived (1 millisecond to a few milliseconds) and travel relatively short distances.

An action potential, in comparison, is generated within the initial segment (see section 12.8b) and propagated along the conductive segment of the neuron (see section 12.8c). An action potential is initiated when voltage-gated channels open in response to a minimum voltage change (threshold value). First, voltage-gated Na\(^+\) channels open, allowing Na\(^+\) into a neuron to cause depolarization (reversal of membrane potential from negative to positive). These voltage-gated channels then close and voltage-gated K\(^+\) channels open, allowing K\(^+\) out to cause repolarization (return of membrane potential from positive to negative). An action potential is self-propagating and maintains its intensity (charge difference) as it moves along the axon to the synaptic knob because of the successive opening of voltage-gated Na\(^+\) channels followed immediately by the successive opening of voltage-gated K\(^+\) channels. Propagation of an action potential is called a nerve signal (or nerve impulse). Action potentials obey the all-or-none law because any voltage sufficient to open the voltage-gated channels (threshold value) initiates an action potential (all), whereas any voltage below the threshold (subthreshold) is not sufficient to open these channels and an action potential is not sent (none). The characteristics of these two very distinctive electrical events that occur at the plasma membrane are summarized in table 12.2.

### Table 12.2 Graded Potential Versus Action Potential

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Graded Potential</th>
<th>Action Potential</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neuron segment</strong></td>
<td>Dendrites and cell body</td>
<td>Axon</td>
</tr>
<tr>
<td><strong>Channels</strong></td>
<td>Chemically gated channels</td>
<td>Voltage-gated channels</td>
</tr>
<tr>
<td><strong>Direction of voltage change</strong></td>
<td>Positive or negative</td>
<td>Positive then negative</td>
</tr>
<tr>
<td><strong>Amount of voltage change</strong></td>
<td>Relatively small change</td>
<td>Relatively large change that causes temporary reversal of polarity</td>
</tr>
<tr>
<td><strong>Degree of voltage change</strong></td>
<td>Dependent upon magnitude of the stimulus</td>
<td>Generally does not vary</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>1 msec to a few msec</td>
<td>Self-propagating along axon (time varies by length of axon)</td>
</tr>
<tr>
<td><strong>Distance traveled</strong></td>
<td>Relatively short distance</td>
<td>Length of axon</td>
</tr>
<tr>
<td><strong>Change in intensity</strong></td>
<td>Decreases with distance</td>
<td>Same intensity (because voltage-gated channels continue to open in sequence)</td>
</tr>
</tbody>
</table>

1. The regions listed reflect the most common location for that type of potential.

**INTEGRATE CONCEPT CONNECTION**

Both graded potentials and action potentials also occur in skeletal muscle fibers. Graded potentials occur in the motor end plate of skeletal muscle fibers, and when the threshold is reached, they are called an end-plate potential. An end-plate potential initiates an action potential along the sarcolemma of a skeletal muscle fiber to trigger the release of calcium from the sarcoplasmic reticulum (see section 10.3b). Calcium ion binds to troponin to initiate the sliding of contractile proteins within the skeletal muscle fiber (see section 10.3c).

**WHAT DID YOU LEARN?**

26. Explain how action potentials differ from graded potentials.
12.9b Velocity of Action Potential Propagation

LEARNING OBJECTIVES

37. Describe the two primary factors that influence the velocity of action potential propagation.
38. Identify the criteria used to distinguish the groups of nerve fibers.

Propagation of an action potential (the nerve signal) along an axolemma (axon plasma membrane) varies in its velocity and is influenced primarily by two factors—the diameter of an axon and the myelination of an axon:

- **Diameter of the axon.** Nerve signal velocity is generally faster in axons with a larger diameter. This is because there is less resistance to the movement of ions within the larger axon, allowing these axons to reach threshold more rapidly than smaller axons.

- **Myelination of the axon.** Myelination of the axon was described in section 12.4c and is the more important factor influencing nerve signal velocity. Nerve signal velocity occurs more rapidly in myelinated axons than in unmyelinated axons (see figure 12.21).

A **nerve fiber** is an axon and its myelin sheath. Nerve fibers are classified into three major groups called A, B, and C, based upon their nerve signal velocity. **Group A** nerve fibers each have a nerve signal velocity that may be as fast as 150 meters per second; these fibers have a large diameter and are myelinated. Most somatic sensory neurons that extend from sensory receptors to the CNS (e.g., those relaying visual input), and all somatic motor neurons that extend from the CNS to skeletal muscles, are included in this group. **Group B** nerve fibers each propagate nerve signals at approximately 15 meters per second, and **group C** nerve fibers each propagate nerve signals at 1 meter per second; group B and group C nerve fibers are generally small in diameter, unmyelinated, or both. Visceral sensory and autonomic motor neurons, as well as small somatic sensory neurons that extend from the receptors of the skin to the CNS, are included in groups B and C.

WHAT DID YOU LEARN?
27. What are the general characteristics of group A nerve fibers, and what functions do they normally serve?

12.9c Frequency of Action Potentials

LEARNING OBJECTIVE

39. Describe how action potentials vary in frequency.

Action potentials are always propagated along an axon (as nerve signals) at the same **amplitude** (change in voltage). However, action potential **frequency** can vary and is dependent upon the stimulus strength. As the stimulus strength increases, the frequency of action potentials increases (up to the point of maximum frequency). Consider the following: A brighter light initiates more nerve signals to be relayed from the eye along the optic nerve to the brain, and a loud sound initiates more nerve signals to be relayed along the vestibulocochlear nerve from the inner ear to the brain. The brain then interprets the increased frequency of nerve signals as a more intense stimulus (see section 16.1c). In addition, frequency of action potentials relayed along somatic motor neurons to skeletal muscle increases muscle tension, as described in section 10.6c and shown in figure 10.22.

Recall from section 12.8d that varying frequency of action potentials can also influence the type of neurotransmitter released from the synaptic knobs for those neurons that store and release more than one type of neurotransmitter.

WHAT DID YOU LEARN?
28. Explain how frequency of action potentials differs from the velocity of an action potential propagation.
12.10 Neurotransmitters and Neuromodulation

Neurotransmitters are released into the synaptic cleft and their action is modified by neuromodulation. Here we describe the different means of classifying neurotransmitters based upon chemical structure and function, prior to reviewing critical features of acetylcholine and other specific types of neurotransmitters. We then discuss how the action of neurotransmitters can be altered through the process of neuromodulation.

12.10a Classification of Neurotransmitters

**LEARNING OBJECTIVES**

40. Identify the four classes of neurotransmitters based upon chemical structure.

41. Describe how neurotransmitters are classified based upon function.

Conventionally, neurotransmitters have been defined as small, organic molecules that (1) are synthesized by neurons and stored within vesicles in synaptic knobs; (2) are released from the vesicles when an action potential triggers calcium entry into the synaptic knob; (3) bind to a specific receptor in a target cell (neuron, muscle, or gland); and (4) trigger a physiologic response in the target cell. There are estimated to be approximately 100 known neurotransmitters. However, it should be noted that some molecules that are called neurotransmitters (e.g., nitric oxide) do not meet all of these criteria.

Neurotransmitters are classified based upon their chemical structure and function (figure 12.24). There are four categories of neurotransmitters based upon the chemical structure (figure 12.24a):

- **Acetylcholine (ACh).** The structure of ACh is significantly different from the other neurotransmitters and for this reason is placed in its own category.
- **Biogenic amines** (also called monoamines). They are derived from certain amino acids (see figure 2.25) by the removal of a carboxyl group (—COOH) and the addition of another functional group (e.g., an hydroxyl group) by enzymatic pathways within the cytosol. The functional group added determines whether the molecule belongs to either catecholamines (dopamine, norepinephrine, and epinephrine) that are synthesized from the amino acid tyrosine or indolamines, which include histamine (synthesized from histidine) and serotonin (synthesized from tryptophan).
- **Amino acids.** These include glutamate, aspartate, serine, glycine (see figure 2.25), and gamma aminobutyric acid (GABA, a modified amino acid). Some controversy remains about how chemical structures that are so plentiful in the cell for protein synthesis also can serve the neurotransmitter communication function.
- **Neuropeptides** (or peptides). These are chains of amino acids that range in length from 2 to 40 amino acids. Examples of neuropeptides include the natural opiates (e.g., enkephalins, beta-endorphins), and substance P.

Neurotransmitter classification based upon function (figure 12.24b) reflects the specific effect that a neurotransmitter has on the membrane potential of a target cell. Neurotransmitters are considered excitatory if they induce an EPSP, whereas they are inhibitory if they induce an IPSP (see section 12.8a). (Note that some neurotransmitters may be either excitatory or inhibitory depending upon the specific response they cause in their target organs.)

Another neurotransmitter classification based upon function reflects whether the target cell response is either direct (i.e., the neurotransmitter directly binds to the receptor of the target cell to cause opening of an ion channel) or indirect (i.e., the neurotransmitter binds to a receptor that activates the second messenger pathway involving G protein; see G proteins in section 4.5b). The second messenger ultimately can trigger much more diverse effects, including the opening of ion channels, the activation of an existing enzymatic pathway, or transcription of genes for the synthesis of new proteins.

**WHAT DID YOU LEARN?**

29. Describe how neurotransmitters are classified based upon structure and function.

12.10b Features of Neurotransmitters

**LEARNING OBJECTIVE**

42. Describe how acetylcholine functions as a neurotransmitter.

43. Discuss the different mechanisms for removing neurotransmitter from the synaptic cleft.

Here we discuss the synthesis and function of acetylcholine (ACh) in detail because it exhibits all of the classical features of neurotransmitters (as described in section 12.10a) and is the most understood. Several attributes about acetylcholine are considered, including its (1) synthesis, (2) removal from the synaptic cleft, and (3) interaction...
with target cells. Please view figure 12.25 as you read through this section. Specific diseases, drugs, and poisons that alter the normal function of ACh are also included in this figure (see Clinical View 12.6: “Altered Acetylcholine Function and Changes in Breathing”).

The neurotransmitter acetylcholine is released from neurons located throughout the body. These include the somatic motor neurons at the neuromuscular junction (as described in section 10.3a) and many neurons of the autonomic nervous system (see chapter 15). Acetylcholine also acts as a neuromodulator (see section 12.10c) within the central nervous system, where it acts to increase attention and arousal. The process of acetylcholine synthesis, release and removal is discussed below and shown in figure 12.25.

**Synthesis and release (step a).** Acetylcholine is synthesized from acetate and choline and then stored inside synaptic vesicles within the synaptic knobs of a neuron. Thousands of molecules of ACh are released by exocytosis into the synaptic cleft in response to the arrival of a nerve signal in the presynaptic neuron. The more frequent the nerve signals, the greater the amount of acetylcholine released.

**Removal from synaptic cleft (step b).** Some ACh molecules will be immediately digested by acetylcholinesterase, an enzyme that resides in the synaptic cleft. Acetylcholine is digested into acetate and choline, and then the choline is taken up into the neuron that released the ACh. Some of the ACh molecules cross the synaptic cleft and bind to target cell receptors. These ACh molecules will quickly dissociate from the receptors usually within 1 millisecond to then be digested by acetylcholinesterase.

**Interaction with Target Cells (step c).** The effect ACh has on a target cell depends upon the specific type of receptor embedded within the plasma membrane of the target cell. The receptors that bind acetylcholine are either nicotinic receptors or one of several subtypes of muscarinic receptors (see section 15.5b). ACh interacts directly with nicotinic receptors, causing the opening of ion channels and the production of an EPSP (see section 12.8a). In comparison, the interaction of ACh with muscarinic receptors causes ion channels to open indirectly through the second messenger pathway that involves G protein (see section 4.5b). Interestingly, the result may be the formation of an EPSP or an IPSP depending upon the specific subtype of muscarinic receptor to which ACh binds.

**WHAT DO YOU THINK?**

Predict the general effect of a drug that crosses the blood-brain barrier and inhibits the action of acetylcholinesterase.

**WHAT DID YOU LEARN?**

How is it possible for acetylcholine to generate either an EPSP or an IPSP?

---

**Figure 12.25 Acetylcholine Release, Removal from Synaptic Cleft, and Action.** Acetylcholine (a) is synthesized and released from synaptic knobs into a synaptic cleft, (b) is removed from the synaptic cleft through enzymatic breakdown by acetylcholinesterase, and (c) interacts with target cells either directly or indirectly. Each of these processes is altered by specific types of toxins, chemical poisons, or drugs.
Some substances or conditions may alter the response of acetylcholine at the neuromuscular junction of skeletal muscles. The response is increased (or excited) in some cases, whereas in others it may be decreased (or inhibited). This altered response differentially affects the skeletal muscle of breathing at the neuromuscular junction (NMJ).

**Substances or Conditions that Decrease Response at the NMJ:**

Some substances that decrease the response at the NMJ include (a) botulinum toxin (figure 12.25, step a), which decreases the release of ACh (see Clinical View 10.3: “Muscular Paralysis and Neurotoxins”), (b) cobratoxin and curare (figure 12.25, step c), which act as competitive inhibitors of ACh receptors (preventing binding of ACh), and (c) loss of acetylcholine receptors in myasthenia gravis (see Clinical View 10.2: “Myasthenia Gravis”). The outcome of this decreased response at the NMJ is ultimately expressed as a weakness in skeletal muscles, including muscles of breathing (e.g., diaphragm; see figure 23.19). Impaired breathing muscle contractions may be fatal if sufficiently severe.

**Substances or Conditions that Increase Response at the NMJ:**

Substances or conditions that increase the response at the NMJ include acetylcholinesterase inhibitors, which interfere with the breakdown of acetylcholine within the neuromuscular junction. Organophosphates (e.g., chemicals found in some insecticides) are acetylcholinesterase inhibitors. The outcome of this increased response at the NMJ is ultimately expressed as overstimulation of skeletal muscles, including muscles of breathing. This prevents breathing muscles from relaxing and may be fatal. Unfortunately, overexposure to common insecticides often is the cause of this poisoning.

**WHAT DID YOU LEARN?**

31. How does nitric oxide act as a neuromodulator?

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**CONCEPT CONNECTION**

Other neurotransmitters include adenosine triphosphate (ATP; a molecule described in section 2.7d) and adenosine (which is merely adenosine without the three phosphates). Adenosine, for example, when bound to adenosine receptors in the brain, has an inhibitory effect. Caffeine acts as a stimulant by blocking adenosine receptors and acting as a competitive inhibitor (see section 3.3f).
### Table 12.3 Neurotransmitters

<table>
<thead>
<tr>
<th>Neurotransmitter</th>
<th>Description/Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACETYLCHOLINE (ACh)</strong></td>
<td>The primary neurotransmitter used at the neuromuscular junction (see section 10.3a) and by most of the autonomic nervous system (see section 15.6).</td>
</tr>
<tr>
<td><img src="image" alt="ACh structure" /></td>
<td></td>
</tr>
<tr>
<td><strong>BIOGENIC AMINES</strong></td>
<td>Molecules synthesized from an amino acid by removal of the carboxyl group and retaining the single amine group; also called monoamines</td>
</tr>
<tr>
<td><img src="image" alt="BIOGENIC AMINES structure" /></td>
<td></td>
</tr>
<tr>
<td>Catecholamines</td>
<td>A distinct group of biogenic amines that contain a catechol chemical group; originally described as hormones (chemicals produced by a gland in one part of the body that affect cells in other parts of the body)</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Produces inhibitory activity in the brain; important roles in cognition (learning, memory), motivation, behavior, and mood; decreased levels in Parkinson disease; amphetamines increase release; cocaine decreases removal from synaptic cleft; ecstasy increases release</td>
</tr>
<tr>
<td>Norepinephrine (noradrenaline)</td>
<td>Neurotransmitter of peripheral autonomic nervous system (sympathetic division) and various regions of the CNS; amphetamines increase release; cocaine decreases removal from synaptic cleft; ecstasy increases release</td>
</tr>
<tr>
<td>Epinephrine (adrenaline)</td>
<td>Has various effects in the thalamus, hypothalamus, and spinal cord</td>
</tr>
<tr>
<td>Indolamines</td>
<td>A distinct group of biogenic amines that contain an indole chemical group.</td>
</tr>
<tr>
<td>Histamine</td>
<td>Neurotransmitter of the CNS; plays a role in sleep and memory</td>
</tr>
<tr>
<td>Serotonin</td>
<td>Has various functions in the brain related to sleep, appetite, cognition (learning, memory), and mood; fluoxetine (Prozac) decreases reuptake; ecstasy increases the release; LSD binds to most serotonin receptors</td>
</tr>
<tr>
<td><strong>AMINO ACIDS</strong></td>
<td>Molecules with both carboxyl (—COOH) and amine (—NH₂) groups and various R groups; building blocks of proteins; act as signaling molecules in the nervous system</td>
</tr>
<tr>
<td><img src="image" alt="AMINO ACIDS structure" /></td>
<td></td>
</tr>
<tr>
<td>Glutamate</td>
<td>Excites activity in nervous system to promote cognitive function in the brain (learning and memory); most common neurotransmitter in the brain; stroke causes excessive release, resulting in neuron death</td>
</tr>
<tr>
<td>Aspartate</td>
<td>Excites activity primarily in descending motor pathways through the spinal cord to skeletal muscle</td>
</tr>
<tr>
<td>Serine</td>
<td>Activates diverse areas in the brain</td>
</tr>
<tr>
<td>Gamma-aminobutyric acid (GABA)</td>
<td>Modified amino acid that is synthesized from glutamate; primary inhibitory neurotransmitter in the brain; also influences muscle tone; alcohol, diazepam (Valium), and barbiturates increase inhibitory effects of GABA</td>
</tr>
<tr>
<td>Glycine</td>
<td>Inhibits activity between neurons in the brain, spinal cord, and eye; strychnine blocks receptors that bind glycine</td>
</tr>
<tr>
<td><strong>NEUROPEPTIDES</strong></td>
<td>Small molecules made of chains of amino acids; generally act through G proteins to cause more diverse effects; opioids include enkephalins, endorphins, endomorphins, dynorphins, and nociceptin; methadone binds at opiate receptors</td>
</tr>
<tr>
<td><img src="image" alt="NEUROPEPTIDES structure" /></td>
<td></td>
</tr>
<tr>
<td>Enkephalin (an opioid)</td>
<td>Helps regulate response to something that is perceived to be noxious or potentially painful</td>
</tr>
<tr>
<td>Beta-endorphin (an opioid)</td>
<td>Prevents release of pain signals from neurons and fosters a feeling of well-being; morphine mimics endorphins; heroin is converted to morphine in the body</td>
</tr>
<tr>
<td>Neuropeptide Y</td>
<td>Involved in memory regulation and energy balance (increased food intake and decreased physical activity)</td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Inhibits activities of neurons in specific brain areas</td>
</tr>
<tr>
<td>Substance P</td>
<td>Assists with pain information transmission into the brain</td>
</tr>
<tr>
<td>Cholecystokinin</td>
<td>Stimulates neurons in the brain to help mediate satiation (fullness) and repress hunger</td>
</tr>
<tr>
<td>Neurotensin</td>
<td>Helps control and moderate the effects of dopamine</td>
</tr>
<tr>
<td><strong>OTHERS</strong></td>
<td>Small molecules made of chains of amino acids; generally act through G proteins to cause more diverse effects; opioids include enkephalins, endorphins, endomorphins, dynorphins, and nociceptin; methadone binds at opiate receptors</td>
</tr>
<tr>
<td>Adenosine</td>
<td>Part of a nucleotide (a building block of nucleic acid); has an inhibitory effect on neurons in the brain and spinal cord</td>
</tr>
<tr>
<td>Nitric oxide</td>
<td>Involved in learning and memory; relaxation of muscle in the digestive tract; relaxation of smooth muscle in blood vessels</td>
</tr>
<tr>
<td>Endocannabinoids</td>
<td>Most prevalent receptors in the brain</td>
</tr>
</tbody>
</table>
Chapter Twelve
Nervous System: Nervous Tissue

12.11 Neural Integration and Neuronal Pools of the CNS

LEARNING OBJECTIVE

46. Identify the four different types of neuronal pools, and explain how they function.

The nervous system coordinates and integrates neuronal activity in part because billions of interneurons within the nervous system are grouped in complex patterns called neuronal pools, or neuronal circuits or pathways. Neuronal pools are identified based upon function into four types of circuits: converging, diverging, reverberating, and parallel-after-discharge (figure 12.26). A pool may be localized, with its neurons confined to one specific location, or its neurons may be distributed in several different regions of the CNS. However, all neuronal pools are restricted in their number of input sources and output destinations.

The converging circuit involves inputs that come together (converge) at a single postsynaptic neuron (figure 12.26a). This neuron receives input from several presynaptic neurons. For example, multiple sensory neurons synapse on the neurons in the salivary nucleus in the brainstem, causing the salivary nucleus to alter activity of salivary glands to produce saliva at mealtime. The various inputs originate from more than one stimulus: smelling food, seeing dinnertime on the clock, hearing food preparation activities, or seeing pictures of food in a magazine. These multiple inputs lead to a single output: the production of saliva.

A diverging circuit spreads information from one presynaptic neuron to several postsynaptic neurons, or from one pool to multiple pools (figure 12.26b). The neurons in the brain control the movements of skeletal muscles in the legs during walking and stimulate the muscles in the back to maintain posture and balance while walking. In this case, a single or a few inputs lead to multiple outputs.

Reverberating circuits utilize feedback to produce a repeated, cyclical stimulation of the circuit: This cyclical feedback is termed reverberation (figure 12.26c). Once activated, a reverberating circuit may continue to function until the cycle is broken by either inhibitory stimuli or synaptic fatigue. The repetitious nature of a reverberating circuit ensures that we continue breathing while we are asleep.

In a parallel-after-discharge circuit, input is transmitted simultaneously along several neuron pathways to a common postsynaptic cell (figure 12.26d). Note that neuron pathways in a parallel-after-discharge circuit vary in the number of neurons within the pathway and thus the number of synapses within the pathway. Recall from section 12.3 that neuron-to-neuron communication at a synapse involves a synaptic delay (the time delay for the events at a synapse). Consequently, the greater the number of neurons in the pathway, the greater the number of synapses and the greater the amount of time required to transmit the information. This results in the information arriving from the point of stimulus input to the common postsynaptic cell at varying times. You might find it helpful to think of the arrival of information from each group of neurons to the common postsynaptic cell as an “echo” of the original stimulus input. This type of circuit is believed to be involved in higher-order thinking; for example, it reinforces the repetitive neural activity needed for performing precise mathematical calculations.

WHAT DID YOU LEARN?

12. How are neurons arranged in a converging circuit?
13. What are the differences between a reverberating circuit and a parallel-after-discharge circuit?

Figure 12.26 Neuronal Pools. Neuronal pools are groups of neurons arranged in specific patterns (circuits) through which input is conducted and distributed. Four types of neuronal pools are recognized.
## CHAPTER SUMMARY

<table>
<thead>
<tr>
<th>12.1 Introduction to the Nervous System</th>
</tr>
</thead>
<tbody>
<tr>
<td>• The nervous system interprets and controls all sensory input from receptors and motor output to effectors.</td>
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<table>
<thead>
<tr>
<th>12.1a General Functions of the Nervous System</th>
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<tbody>
<tr>
<td>• The nervous system is composed of the brain, spinal cord, nerves, and ganglia.</td>
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<table>
<thead>
<tr>
<th>12.1b Organization of the Nervous System</th>
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<tbody>
<tr>
<td>• The nervous system is organized structurally into the central nervous system (CNS) and the peripheral nervous system (PNS).</td>
</tr>
<tr>
<td>• The nervous system is organized functionally into a sensory component and a motor component.</td>
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<table>
<thead>
<tr>
<th>12.1c Nerves and Ganglia</th>
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<tbody>
<tr>
<td>• A nerve is a collection of axons that are wrapped in connective tissue.</td>
</tr>
<tr>
<td>• The entire nerve is enclosed with an epineurium. Fascicles of axons are ensheathed with a perineurium, and each axon (and its surrounding neurolemmocyte) is wrapped with an endoneurium.</td>
</tr>
<tr>
<td>• A ganglion is a cluster of neuron cell bodies located along a nerve.</td>
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<thead>
<tr>
<th>12.2 Nervous Tissue: Neurons</th>
</tr>
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<tbody>
<tr>
<td>• Nervous tissue is composed of excitable neurons that initiate and transmit graded potentials and action potentials, and glial cells that support and protect them.</td>
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<table>
<thead>
<tr>
<th>12.2a General Characteristics of Neurons</th>
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<tbody>
<tr>
<td>• General characteristics of neurons include excitability, conductivity, secretion, and longevity; in addition, they are typically amitotic.</td>
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<thead>
<tr>
<th>12.2b Neuron Structure</th>
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<tbody>
<tr>
<td>• A generalized neuron has a cell body. Processes typically extending from the cell body are numerous short, tapering dendrites and a single and often long axon.</td>
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<thead>
<tr>
<th>12.2c Neuron Transport</th>
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<tbody>
<tr>
<td>• Neurons transport substances between the cell body and synaptic knobs by fast axonal transport and slow axonal transport.</td>
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<thead>
<tr>
<th>12.2d Classification of Neurons</th>
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<tbody>
<tr>
<td>• Neurons are classified structurally as multipolar, bipolar, unipolar, and anaxonic by the number of processes attached to the cell body.</td>
</tr>
<tr>
<td>• The three functional categories of neurons are sensory neurons, motor neurons, and interneurons.</td>
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</table>

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<thead>
<tr>
<th>12.3 Synapses</th>
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<tbody>
<tr>
<td>• A synapse is the functional junction of a neuron with either another neuron or an effector.</td>
</tr>
<tr>
<td>• Synapses are either chemical synapses or electrical synapses.</td>
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</table>

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<thead>
<tr>
<th>12.4 Nervous Tissue: Glial Cells</th>
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<tr>
<td>• Glial cells are the other distinct cell type of nervous tissue.</td>
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<table>
<thead>
<tr>
<th>12.4a General Characteristics of Glial Cells</th>
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<tbody>
<tr>
<td>• Glial cells are nonexcitable cells that primarily support and protect the neurons.</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>12.4b Types of Glial Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Four types of glial cells within the central nervous system are astrocytes, ependymal cells, microglia, and oligodendrocytes.</td>
</tr>
<tr>
<td>• Two types of glial cells within the peripheral nervous system are satellite cells and neurolemmocytes.</td>
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</tbody>
</table>

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<thead>
<tr>
<th>12.4c Myelination</th>
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<tbody>
<tr>
<td>• Myelination is the process by which part of an axon is wrapped and insulated with myelin.</td>
</tr>
<tr>
<td>• Neurolemmocytes myelinate axons in the PNS, and oligodendrocytes myelinate axons in the CNS.</td>
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<tr>
<th>12.5 Axon Regeneration</th>
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<tbody>
<tr>
<td>• Regeneration of damaged neurons is limited to PNS axons.</td>
</tr>
<tr>
<td>• A PNS axon can regrow to reestablish innervation if the cell body is intact and a critical amount of neurilemma remains.</td>
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<tr>
<th>12.6 Plasma Membrane of Neurons</th>
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<tbody>
<tr>
<td>• Establishing and changing a resting membrane potential is dependent upon various types of pumps and channels within a neuron’s plasma membrane.</td>
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</table>

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<thead>
<tr>
<th>12.6a Types of Pumps and Channels</th>
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</thead>
<tbody>
<tr>
<td>• Pumps and channels are membrane proteins that facilitate movement of ions across the neuron plasma membrane.</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>12.6b Distribution of Pumps and Channels</th>
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<tbody>
<tr>
<td>• Some membrane transport proteins are located along the entire neuron, and some are primarily in specific functional neuron segments.</td>
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<thead>
<tr>
<th>12.7 Introduction to Neuron Physiology</th>
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<tbody>
<tr>
<td>• Neuron physiology involves the initiation and transmission of electric currents.</td>
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<thead>
<tr>
<th>12.7a Neurons and Ohm’s Law</th>
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</thead>
<tbody>
<tr>
<td>• Ohm’s law (current = voltage/resistance) has application in the principles of neuron physiology.</td>
</tr>
</tbody>
</table>
### 12.7b Neurons at Rest
- Neurons at rest have a negative resting membrane potential (RMP), which on average is ~70 mV.
- Other characteristics of neurons at rest include closed gated channels; Na<sup>+</sup>, K<sup>+</sup>, and Cl<sup>−</sup> concentration gradients along the length of the axon; and a Ca<sup>2+</sup> concentration gradient at the synaptic knob.

### 12.8 Physiologic Events in the Neuron Segments

#### 12.8a Receptive Segment
- The receptive segment includes the dendrites and cell body. This segment involves the formation and propagation of graded potentials: both excitatory postsynaptic potentials (EPSPs) and inhibitory postsynaptic potentials (IPSPs).

#### 12.8b Initial Segment
- The summation (or adding together) of EPSPs and IPSPs that reach the initial segment determines whether the threshold value (~55 mV) is reached and an action potential is initiated.

#### 12.8c Conductive Segment
- The conductive segment is involved in the propagation of an action potential (nerve signal), a process that involves depolarization and repolarization.
- The brief period of time that an axon is either incapable of generating an action potential or a greater than normal amount of stimulation is required to generate another action potential is called the refractory period.
- Saltatory conduction occurs if the axon is myelinated.

#### 12.8d Transmissive Segment
- The transmissive segment involves exocytosis of neurotransmitter from synaptic vesicles, which are located within synaptic knobs.

### 12.9 Characteristics of Action Potentials

#### 12.9a Graded Potentials Versus Action Potentials
- Graded potentials are short-lived electrical signals that occur in the dendrites and cell body due to opening of chemically gated channels, whereas action potentials are self-propagating electrical signals that are initiated in the initial segment, propagated along an axon, and result from the sequential opening of voltage-gated channels.

#### 12.9b Velocity of Action Potential Propagation
- The velocity of action potentials (or nerve signals) is greater in larger and myelinated axons.
- Nerve fibers, which are axons and their myelin sheath, are classified into three groups based upon the velocity of the nerve signal propagation.

#### 12.9c Frequency of Action Potentials
- Frequency of action potentials occurs with increased stimulation of the neuron.

### 12.10 Neurotransmitters and Neuro-modulation

#### 12.10a Classification of Neurotransmitters
- Neurotransmitters are conventionally described as molecules synthesized by neurons, which are then stored within vesicles in synaptic knobs and, when released, bind to specific receptors in a target cell to trigger a physiologic response.
- Major classes of neurotransmitters include acetylcholine, biogenic amines, amino acids, and neuropeptides.

#### 12.10b Features of Neurotransmitters
- Acetylcholine (ACh) is discussed in detail because it exhibits all of the classical features of neurotransmitters and is the most understood.

#### 12.10c Neuromodulation
- Neuromodulation is the release of chemicals other than neurotransmitters that either increase the responsiveness to a neurotransmitter (facilitation) or decrease the responsiveness to a neurotransmitter (inhibition).

### 12.11 Neural Integration and Neuronal Pools of the CNS
- Interneurons are organized into neuronal pools, which are groups of interconnected neurons with specific functions and are classified as converging, diverging, reverberating, and parallel-after-discharge.
1. The cell body of a neuron does all of the following except
   a. release neurotransmitter into the synaptic cleft.
   b. produce synaptic vesicles containing neurotransmitter that are subsequently transported to the synaptic knob.
   c. conduct graded potentials to the initial segment.
   d. receive graded potentials from dendrites.

2. Neurons that have only two processes attached to the cell body are called
   a. unipolar.
   b. bipolar.
   c. multipolar.
   d. efferent.

3. Which neurons are located only within the CNS?
   a. afferent neurons
   b. glial cells
   c. sensory neurons
   d. interneurons

4. EPSPs are caused by the movement of
   a. Na⁺ out of the cell.
   b. Na⁺ into the cell.
   c. K⁺ into the cell.
   d. both Na⁺ and K⁺ into the cell.

5. The glial cells that help produce and circulate cerebrospinal fluid in the CNS are
   a. satellite cells.
   b. microglia.
   c. ependymal cells.
   d. astrocytes.

6. Which of the following is a part of the PNS?
   a. microglia
   b. spinal cord
   c. brain
   d. neurolemmocyte

7. An action potential is generated when threshold is reached, at which time
   a. voltage-gated K⁺ channels close.
   b. voltage-gated Na⁺ channels open.
   c. chemically gated Na⁺ channels open.
   d. Ca²⁺ enters the cell.

8. Which type of neuronal pool utilizes feedback to repeatedly stimulate the circuit?
   a. converging circuit
   b. diverging circuit
   c. reverberating circuit
   d. parallel-after-discharge circuit

9. At an electrical synapse, presynaptic and postsynaptic membranes interface through
   a. neurofibril nodes.
   b. gap junctions.
   c. telodendria.
   d. neurotransmitters.

10. The two primary factors that influence the speed of an action potential propagation are axon diameter and
    a. myelination.
    b. the type of associated glial cell(s).
    c. concentration of K⁺ in the cell.
    d. the length of the axon.

11. What are the four structural types of neurons? How do they compare to the three functional types of neurons?

12. Identify the principal glial cell types, and briefly discuss the function of each type.

13. How does myelination differ between the CNS and the PNS?

14. Describe the procedure by which a PNS axon may repair itself (axon regeneration).

15. Describe how the resting membrane potential is established and maintained in a neuron.

16. Compare and contrast graded potentials and action potentials.

17. Explain summation of EPSPs and IPSPs and the relationship to the initiation of an action potential.

18. Graph and explain the events associated with an action potential.

19. Explain the mechanism for the release of neurotransmitter from the synaptic knob.

20. List and briefly describe the major types of neurotransmitters.

---

**Can You Apply What You’ve Learned?**

1. Andrew was taken to the doctor’s office after he was bitten by a stray dog. The concern was that the dog might be infected with the rabies virus. The rabies virus infects neurons by using which method that normally transports materials from the synaptic knob to the cell body?
   a. anterograde transport
   b. fast axonal transport
   c. slow axonal transport
   d. All of these are correct.

2. An elderly neighbor was diagnosed with an astrocytoma tumor in the brain. This cancer affects what types of cell?
   a. ependymal cells
   b. microglia
   c. astrocytes
   d. satellite cells
3. Cynthia has received her lab results and is told that her blood calcium levels are abnormal. The event in neuron transmission most likely to be affected is
   a. summation of graded potentials in the initial segment.
   b. production of graded potentials in the dendrites and cell body.
   c. release of neurotransmitter from the synaptic knob.
   d. propagation of an action potential in the axon.

4. Heidi’s physician prescribed a medication that is known to block the reuptake of serotonin neurotransmitter from the synaptic cleft. This medication affects what segment in neuron transmission that is responsible for releasing the neurotransmitter?
   a. initial segment
   b. conductive segment
   c. transmissive segment
   d. receptive segment

5. Sarah wants to call her new friend, Julie, and needs to write down her phone number but cannot find a pen. She continues to repeat the number over and over. This is most likely occurring in what type of neuronal pool?
   a. reverberating circuit
   b. divergent circuit

Can You Synthesize What You’ve Learned?

1. Over a period of 6 to 9 months, Marianne began to experience vision problems as well as weakness and loss of fine control of the skeletal muscles in her leg. Blood tests revealed the presence of antibodies (immune system proteins) that attack myelin. Beyond the presence of the antibodies, what was the cause of Marianne’s vision and muscular difficulties?

2. Surgeons were able to reattach Irving’s amputated limb, sewing both the nerves and the blood vessels back together. After the surgery, which proceeded very well, the limb regained its blood supply almost immediately, but the limb remained motionless and Irving had no feeling in it for several months. Why did it take longer to reestablish innervation than circulation?

3. Certain types of neurotoxins prevent depolarization of the axon. What specific type of channel is impaired?
The human brain, while weighing on average about 3 pounds, is able to simultaneously process billions of pieces of information. It is continuously receiving sensory input from sensory receptors and initiating motor output to control effectors. This organ allows us to understand complex information, write poetry, and compute mathematical problems. The brain, which is part of the central nervous system (CNS), is associated with the 12 pairs of cranial nerves, which are considered part of the peripheral nervous system (PNS). The brain and cranial nerves are described in this chapter.
13.1 Brain Organization and Development

We begin our study of the brain by introducing its four major regions and surface landmark structures. Next we provide the essentials of embryonic brain development that help clarify how the structures of the adult brain are named and related. Then we examine the distribution of gray and white matter in the brain as a whole.

13.1a Overview of Brain Anatomy

LEARNING OBJECTIVE

1. Describe the general regions of the brain.

The brain is composed of four major regions: the cerebrum, diencephalon, brainstem, and cerebellum. Figure 13.1 shows the major parts of the adult brain from several views, including the lateral view (figure 13.1a), inferior view (figure 13.1b), and midsagittal view (figure 13.1c). The cerebrum is divided into two halves, called the left and right cerebral hemispheres. Each hemisphere may be further subdivided into five functional areas called lobes. Our skull volume limits the size of the brain, so the outer brain tissue of the cerebrum is folded on itself so that more neurons can fit within the cranium. These folds of brain tissue are called gyri (jir′i; sing., gyrus; gyros = circle). The shallow depressions between these folds are called sulci (sul′i; sing., sul′ki; furrow, ditch), and the deeper grooves are named fissures (fish′ur).

Note also as you observe the images in figure 13.1 that

- The cerebellum is inferior to the cerebrum (which is best viewed in figure 13.1a).
- The brainstem has three regions: the midbrain, pons, and medulla oblongata (which are best viewed in figures 13.1b, c).
- The diencephalon is organized into the epithalamus, thalamus, and hypothalamus (which are essentially internal structures and are best viewed in figure 13.1c).
- Twelve pairs of cranial nerves extend from the brain (which are best viewed in figure 13.1b).

Two directional terms are often used to describe relative positions of brain anatomy. Anterior is synonymous with rostral (meaning “toward the nose”), and posterior is synonymous with caudal (meaning “toward the tail”).

WHAT DID YOU LEARN?

1. What are the four major regions of the brain?

A contusion is a TBI where there is bruising of the brain due to trauma that causes blood to leak from small vessels into the subarachnoid space (a fluid-filled space surrounding the brain). The bruising may appear on a computed tomography (CT) scan of the head (see Clinical View 1.4: “Medical Imaging”). Usually, the person immediately loses consciousness (normally for no longer than 5 minutes). Respiration abnormalities and decreased blood pressure sometimes occur as well.

Of particular concern is a rare but serious condition called second impact syndrome (SIS), where an individual experiences a second brain injury prior to the resolution of the first injury and develops severe brain swelling and possibly death as a result. For this reason, it is essential that the original TBI completely heals before an individual is allowed to resume a behavior that may put the individual at risk for another TBI. Both severe traumatic brain injury and repetitive TBIs may cause long-term cognitive deficits and motor impairment. Individuals may need physical, occupational, and speech therapy to regain a portion of these functions.

Interestingly, preliminary research has shown that TBI patients who received therapeutic progesterone made a greater and faster recovery than individuals with similar TBIs who did not receive the therapy. Thus, a reproductive hormone (progesterone) also appears to help the nervous system with its healing.
Figure 13.1 The Human Brain. The brain is a complex organ that is formed from several subdivisions. (a) An illustration and a cadaver photo of a left lateral view show the cerebrum, cerebellum, and portions of the brainstem (in bold); the diencephalon is not visible. ©McGraw-Hill Education/Christine Eckel

(continued on next page)
Figure 13.1 The Human Brain (continued). (b) An illustration and a cadaver photo of the brain in inferior view best demonstrate the cranial nerves arising from the base of the brain. The major regions of the brain are in bold. ©McGraw-Hill Education/Christine Eckel
Figure 13.1 The Human Brain (continued). (c) Internal structures such as the thalamus and hypothalamus are best seen in midsagittal view in an illustration and a cadaver photo. The major regions of the brain are in bold. © McGraw-Hill Education/Christine Eckel
13.1b Development of Brain Divisions

LEARNING OBJECTIVES

2. Describe the general process of nervous tissue development and neurulation.

3. Provide the scientific names for the embryonic forebrain, midbrain, and hindbrain.

4. Name the five secondary brain vesicles, describe their embryonic origins, and list the adult brain structures that are formed by each.

Before we can understand how the divisions of the brain form, we first must explore how the nervous system is derived from ectoderm, one of the three primary germ layers (see figure 5.13). The process of neurulation is then described.

Neurulation

The formation of nervous tissue begins in the embryo during the third week of development with a thickening of a portion of the ectoderm (see section 5.6a). This portion of the ectoderm specifically overlies the notochord, which is a tightly packed group of mesoderm cells positioned on the developing embryo midline (see section 29.3b) (figure 13.2). The thickened ectoderm is called the neural plate. The neural plate is induced by the underlying notochord to form a neural tube, which begins the process called neurulation (nūr’ə-lā′shən). Neurulation ultimately forms all nervous tissue structures. The process of neurulation is shown in figure 13.2 and explained here:

1. The neural plate develops a central longitudinal indentation called the neural groove. As this is occurring, cells along the lateral margins of the neural plate proliferate, becoming the thickened neural folds. The tips of the neural folds form neural crest cells (or simply, the neural crest).

2. The neural folds elevate and approach one another as the neural groove continues to deepen. The neural crest cells are now at the very highest point of the neural groove. When viewed from a superior angle, the neural folds resemble the sides of a hot dog roll, with the neural groove represented by the opening in the roll.

3. The neural crest cells begin to pinch off from the neural folds and form other structures.

4. By the end of the third week, the neural folds have met and fused at the midline as the neural groove starts to form a neural tube, which has an internal space called the neural canal. The neural tube initially fuses at its midline, and later the portions of the neural folds slightly superior and inferior to this midline fuse as well. Thus, the neural tube forms as the neural folds “zip” together both superiorly and inferiorly.

For a short time, the neural tube is open at both its ends. These openings, called neuropores (nūr’ō-pôr), close during the end of the fourth week. The opening closest to the future head is the cranial neuropore, whereas the opening closest to the future buttocks region is the caudal neuropore (see figure 29.12 in section 29.3b). If these openings do not close, the developing human will have a neural tube defect (see Clinical View 13.2: “Neural Tube Defects”). The developing neural tube forms the central nervous system. In particular, the cranial part of the neural tube expands to form the brain, while the caudal part of the neural tube expands to form the spinal cord (see section 14.7).

Figure 13.2 Nervous System Development. The process of neurulation begins in the third week, and the neural tube finishes closing by the end of week 4.

1. Neural folds and neural groove form from the neural plate.
2. Neural folds elevate and approach one another.
3. Neural crest cells begin to pinch off from the neural folds and form other structures.
4. Neural folds fuse to form the neural tube.
Development of the Brain

The brain develops from the cranial part of the neural tube in the human embryo. The neural tube undergoes disproportionate growth rates in different regions. This growth has formed three primary brain vesicles by the late fourth week of development, which eventually give rise to all the different regions of the adult brain (table 13.1). The names of these vesicles describe their relative positions in the developing head: The forebrain is called the prosencephalon (pros-en-sef′-å-lon; proso = forward, enkephalos = brain); the midbrain is called the mesencephalon (mez-en-sef′-å-lon; mes = middle); and the hindbrain is called the rhombencephalon (rom-ben-sef′-å-lon; rhombo = rhomboid) for its rhomboidal shape (figure 13.3a).

By the fifth week of development, the three primary vesicles further develop into a total of five secondary brain vesicles (figure 13.3b):

- The telencephalon (tel-en-sef′-å-lon; tel = head end) arises from the prosencephalon and eventually forms the cerebrum.
- The diencephalon (di-en-sef′-å-lon; dia = through) also derives from the prosencephalon, and it eventually forms the thalamus, hypothalamus, and epithalamus.
- The mesencephalon is the only primary vesicle that does not form a new secondary vesicle. It becomes the midbrain.
- The metencephalon (met-′en-sef′-å-lon; meta = after) arises from the rhombencephalon and eventually forms the pons and cerebellum.
### Table 13.1 Major Brain Structures: Embryonic Through Adult

<table>
<thead>
<tr>
<th>Embryonic Development</th>
<th>Secondary Brain Vesicles (Future Adult Brain Regions)¹</th>
<th>Neural Canal Derivative²</th>
<th>Structures Within Brain Regions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anterior</strong></td>
<td>Telencephalon</td>
<td>Lateral ventricles</td>
<td>Cerebrum</td>
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<td></td>
<td>Diencephalon</td>
<td>Third ventricle</td>
<td>Epithalamus, Thalamus, Hypothalamus</td>
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<tr>
<td></td>
<td>Mesencephalon (midbrain)</td>
<td>Cerebral aqueduct</td>
<td>Midbrain</td>
</tr>
<tr>
<td></td>
<td>Rhombencephalon (hindbrain)</td>
<td>Fourth ventricle (superior part)</td>
<td>Pons, cerebellum</td>
</tr>
<tr>
<td></td>
<td>Metencephalon</td>
<td>Fourth ventricle (inferior part); part of central canal</td>
<td>Medulla oblongata</td>
</tr>
</tbody>
</table>

1. The embryonic secondary vesicles form the adult brain regions—thus, they share the same names.
2. The neural canal in each specific brain region will form its own named space.

The anatomic *structure* of *gray matter* within the CNS primarily derives its color from the cell bodies and dendrites of the neurons that compose it. One indication that a general *brain* structure is composed of gray matter is that these areas are often (though not always) designated as

- **Cortex**, which is a superficial layer of gray matter (like the bark of a tree), or
- **Nucleus, or center**, which are clusters of neuron cell bodies within gray matter that are either close to the surface or deep within the brain.

The outer (superficial) gray matter of the cerebrum, for example, is the *cerebral cortex* and the inner (deep) gray matter of the cerebrum is the *cerebral nuclei* (figure 13.4a). Another example is the outer gray matter of the cerebellum, which is the *cerebellar cortex* (figure 13.4b). Note that the spinal cord has the reverse pattern: outer white matter and inner gray matter.

The general *function* of each of the different regions of gray matter, in either the brain or the spinal cord, is to serve as an integrating and processing area. Specifically, the *synapses* within the gray matter allow for integration and processing to occur.

The anatomic *structure* of *white matter*, in comparison, derives its color from the bundles of myelinated axons that compose it. These bundles of myelinated axons within the CNS are called *tracts* and are located on or close to the surface (outer white matter) or deep (inner white matter) (figure 13.4). Tracts within the brain typically have specific names (e.g., corpus callosum, internal capsule, peduncles). Note that the white matter in the spinal cord is subdivided into

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### WHAT DID YOU LEARN?

2. How does the neural plate form a neural tube?
3. Identify the five secondary vesicles, and list the adult brain structures they form.

**13.1c Gray Matter and White Matter Distribution**

**LEARNING OBJECTIVE**

5. Compare and contrast the general composition, function, and distribution of gray and white matter throughout the central nervous system.

Two distinct tissue areas are present within the brain and spinal cord: gray matter and white matter. As you read through this section, view figure 13.4, which shows the distribution of gray matter and white matter in various areas of the central nervous system (CNS) in both the brain and the spinal cord.
**INTEGRATE**

**LEARNING STRATEGY**

In general, gray matter within the brain and spinal cord consists primarily of dendrites and cell bodies that serve as processing, or “decision-making,” areas, whereas white matter is composed of myelinated axons that relay nerve signals to and from the gray matter.

funiculi and white commissures (figure 13.4d), which are described in section 14.3a. (Recall from section 12.1c that, within the peripheral nervous system [PNS], bundles of axons are called nerves and aggregates or clusters of cell bodies are called ganglia.)

The general function of the bundles of axons forming white matter, in either the brain or the spinal cord, is to relay nerve signals (see section 12.8c). White matter provides the means for information to be transmitted between different regions of the brain and spinal cord and between the brain and the body. Table 13.2 may be used as a quick reference for these general terms regarding gray matter and white matter.

**WHAT DID YOU LEARN?**

4 Where is gray matter located within the cerebrum and spinal cord?

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**Table 13.2 Glossary of Nervous System Structures**

<table>
<thead>
<tr>
<th>Structure</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CENTRAL NERVOUS SYSTEM (CNS)</strong></td>
<td></td>
</tr>
<tr>
<td>Gray matter</td>
<td></td>
</tr>
<tr>
<td>Cortex</td>
<td>Superficial layer of gray matter</td>
</tr>
<tr>
<td>Nucleus</td>
<td>Center that displays discrete anatomic boundaries</td>
</tr>
<tr>
<td>Center</td>
<td>Cluster of neuron cell bodies within the central nervous system (CNS)</td>
</tr>
<tr>
<td><strong>WHITE MATTER</strong></td>
<td></td>
</tr>
<tr>
<td>Tract</td>
<td>Bundles of axons within the CNS that share a common origin and destination</td>
</tr>
<tr>
<td>Funiculus</td>
<td>Bundles of axons (tracts) in a specific area of the spinal cord</td>
</tr>
<tr>
<td>Peduncle</td>
<td>Stalklike structure composed of tracts connecting two regions of the brain</td>
</tr>
<tr>
<td><strong>PERIPHERAL NERVOUS SYSTEM (PNS)</strong></td>
<td></td>
</tr>
<tr>
<td>Ganglion</td>
<td>Cluster of neuron cell bodies in the peripheral nervous system (PNS)</td>
</tr>
<tr>
<td>Nerve</td>
<td>Bundle of axons in the PNS</td>
</tr>
<tr>
<td>Nerve plexus</td>
<td>Network of nerves</td>
</tr>
<tr>
<td>Pathway</td>
<td>Composed of two or more neurons that relay nerve signals between the CNS and body structures</td>
</tr>
</tbody>
</table>
13.2 Protection and Support of the Brain

The brain is both protected and isolated by multiple structures. The bony cranium provides rigid support (see section 8.2), while protective connective tissue membranes called meninges surround and partition portions of the brain. Cerebrospinal fluid (CSF) acts as a cushioning fluid between specific layers of meninges. Finally, the brain has a unique blood-brain barrier to prevent entry of harmful substances from the blood into the brain.

13.2a Cranial Meninges

**LEARNING OBJECTIVES**

6. Compare and contrast the structure and locations of the three meninges, and identify the spaces between the meninges.

7. Describe the four cranial dural septa, and give their locations.

The cranial meninges (mē-ninʼjes, mē-nin-jēz; sing., meninx, menʼingks; membrane) are three connective tissue layers that separate and support the soft tissue of the brain from the bones of the cranium, enclose and protect some of the blood vessels that supply the brain, and contain and help circulate cerebrospinal fluid. From deep (closest to the brain) to superficial...
**INTEGRATE**

**CLINICAL VIEW 13.3**

**Meningitis and Encephalitis**

**Meningitis** is the inflammation of the meninges, and typically it is caused by viral or bacterial infection. Early symptoms may include fever, severe headache, vomiting, and a stiff neck (because pain from the meninges may be referred[see section 16.2b] to the posterior neck). Bacterial meningitis typically produces more severe symptoms and may result in brain damage and death if left untreated. Both viral and bacterial meningitis are contagious and may be spread through respiratory droplets or oral secretions, so it is a disease that may spread rapidly through college dormitories or military barracks (where individuals live in close quarters). Thus, most teenagers and military recruits are advised to get the bacterial meningitis vaccine (which protects them against the most common bacterial strains that cause meningitis) prior to attending college or the military, respectively.

**Encephalitis** (en-sef-ă-lī΄ tis; enkephalos = brain, itis = inflammation) is an acute inflammatory disease of the brain, most often due to viral infection. Symptoms include drowsiness, fever, headache, neck pain, coma, and paralysis. Death may occur.

(farthest from the brain), the cranial meninges are the pia mater, the arachnoid mater, and the dura mater (figure 13.5).

**Pia Mater**

The pia mater (pī′ ă mah′ter; pia = tender, mater = mother) is the innermost of the cranial meninges. It is a thin layer of delicate areolar connective tissue that tightly adheres to the brain and follows every contour of the brain surface.

**Arachnoid Mater**

The arachnoid (ā-rak′noid) mater, also called the arachnoid membrane, lies external to the pia mater. The term arachnoid means “resembling a spider web,” and this meninx is so named because it is partially composed of a delicate web of collagen and elastic fibers, termed the arachnoid trabeculae. Immediately deep to the arachnoid mater is the subarachnoid space, which contains cerebrospinal fluid (discussed in section 13.2c). The arachnoid trabeculae extend through this space from the arachnoid to the underlying pia mater. Both the arachnoid trabeculae and cerebrospinal fluid support cerebral arteries and veins within the subarachnoid space.

**Figure 13.5 Cranial Meninges.** A coronal section of the superior portion of the head depicts the organization of the three meningeal layers: the pia mater, the arachnoid mater, and the dura mater. In the midline, folds of the inner meningeal layer of the dura mater form the falx cerebri, which partitions the two cerebral hemispheres. The inner meningeal layer and the outer periosteal layer are separate at various locations to form the dural venous sinuses, such as the superior sagittal sinus (shown here), which drain blood away from the brain.
**INTEGRATE**

**CLINICAL VIEW 13.4**

**Epidural and Subdural Hematomas**

A pooling of blood outside a vessel is referred to as a **hematoma** (hē-mā-tō’mā; hemato = blood, onoma = tumor). An **epidural hematoma** is a pool of blood forming in the epidural space of the brain, usually due to a severe blow to the head. The adjacent brain tissue becomes distorted and compressed as a result of the hematoma continuing to increase in size. Severe neurologic injury and death may occur if the bleeding is not stopped and the accumulated blood removed by surgically drilling a hole in the skull, suctioning out the blood, and ligating (tying off) the bleeding vessel.

A **subdural hematoma** is a hemorrhage that occurs in the subdural space. These hematomas typically result from ruptured veins caused by either fast or violent rotational motion of the head. Blood pools in this space and compresses the brain, although usually these events occur more slowly than with an epidural hematoma. Subdural hematomas are treated similarly to epidural hematomas.

**Dura Mater**

The **dura mater** (dū’rā; dura = tough) is the strongest of the meninges, as its Latin name indicates. This outer, dense irregular connective tissue covering is composed of two layers. The **meningeal** (mē-nin’jē-āl, men’in-jē’āl) layer is immediately superficial to the arachnoid. The **periosteal** (per-i-os’tē-āl) layer, the more superficial layer, forms the periosteum on the internal surface of the cranial bones. The meningeal layer is usually fused to the periosteal layer, except in specific areas where these two layers are separate and form large, blood-filled spaces called **dural venous sinuses**. (A *sinus* is a modified vein; see section 20.1d.) Dural venous sinuses are typically triangular in cross section, and unlike most other veins, they do not have valves to regulate venous blood flow. The dural venous sinuses drain blood from the brain, and most of the specific sinuses are shown in figure 13.6.

Two potential spaces are associated with the dura mater: the epidural space and the subdural space. The dura mater and the bones of the skull may be separated by an **epidural** (ep-i-dū’rāl) space, which contains the arteries and veins that nourish the meninges and bones of the cranium. The **subdural** (sub-dū’rāl) space is positioned between the arachnoid mater and the overlying dura mater. Either the epidural space or the subdural space may become a real space if blood or fluid accumulates within it (see Clinical View 13.4: “Epidural and Subdural Hematomas”).

**Cranial Dural Septa**

The meningeal layer of the dura mater extends as flat partitions into the cranial cavity at four locations. Collectively, these double layers of dura mater are called **cranial dural septa** (sing., *septum* = wall). These membranous partitions separate specific parts of the brain and provide additional stabilization and support to the brain. There are four cranial dural septa: the falx cerebri, tentorium cerebelli, falx cerebelli, and diaphragma sellae (figure 13.6). The falx cerebri (falks sé-rē’bri; falx = sickle) is the largest of the four dural septa. This large, sickle-shaped, vertical fold of dura mater is located in the midsagittal plane and projects into the longitudinal fissure between the left and right cerebral hemispheres. Anteriorly, its inferior portion attaches to the crista galli of the ethmoid bone; posteriorly, its inferior portion attaches to the internal occipital crest (see section 8.2b). Located within the superior and inferior margins of this dural septum are two dural venous sinuses: the **superior sagittal sinus** and the **inferior sagittal sinus**, respectively.

The **tentorium cerebelli** (ten-tō’rē-ūn ser-e-bel’ī) is a horizontally oriented fold of dura mater that separates both the occipital and temporal lobes of the cerebrum from the cerebellum. It is named for the fact that it forms a dural “tent” over the cerebellum. The **transverse sinuses** are within its posterior border, whereas the **straight sinus** is within its midsagittal region. The anterior surface of the tentorium cerebelli has a gap, or opening, called the **tentorial notch** (or **tentorial incisure**), to allow for the passage of the brainstem.

Extending into the midsagittal line inferior to the tentorium cerebelli is the **falx cerebelli**, a sickle-shaped, vertical partition that divides the left and right cerebellar hemispheres. A tiny **occipital** (ok-sip’i-tāl; occiput = back of head) **sinus** (another dural venous sinus) is within its posterior vertical border.

The **diaphragma sellae** (dr-ā-frag’mā sel’ē; sella = saddle) is the smallest of the dural septa. It forms a roof over the sella turcica of the sphenoid bone. A small opening within it allows for the passage of a thin stalk, called the infundibulum, that attaches the pituitary gland to the base of the hypothalamus (described in section 13.4c).

![Figure 13.6 Cranial Dural Septa](image_url)

A midsagittal section and posterior view of the head show the orientation of the falx cerebri, falx cerebelli, tentorium cerebelli, and diaphragma sellae.
### 13.2a Brain Ventricles

**LEARNING OBJECTIVE**

8. Describe the anatomy and location of the ventricles.

Ventricles (ven′tri-kl; little cavity) are cavities or expansions within the brain that are derived from the neural canal (the lumen of the embryonic neural tube). All of the ventricles are lined with ependymal cells (see section 12.4b) and contain cerebrospinal fluid. The ventricles are connected with one another as well as with the central canal of the spinal cord (figure 13.7).

There are four ventricles within the brain: Two lateral ventricles are in the cerebrum, separated by a thin medial partition called the septum pellucidum (pe-lū′si-dum; pellucid = transparent) (see figure 13.1c). Within the diencephalon is a smaller, thinner ventricle called the third ventricle (figure 13.7). Each lateral ventricle is connected with the third ventricle through an opening called the interventricular foramen (formerly called the foramen of Munro). A narrow canal called the cerebral aqueduct (ak′we-dŭkt) (also called the mesencephalic aqueduct and formerly called the aqueduct of Sylvius) passes through the midbrain and connects the third ventricle with the tetrahedron-shaped fourth ventricle. The fourth ventricle is located between the pons, medulla oblongata, and cerebellum. It opens to the subarachnoid space via paired lateral apertures and a single median aperture. The fourth ventricle narrows at its inferior end before it merges with the slender central canal of the spinal cord.

### 13.2c Cerebrospinal Fluid

**LEARNING OBJECTIVES**

9. Explain the three functions of cerebrospinal fluid.

10. Trace the circulation of cerebrospinal fluid, beginning with its origin and ending with its removal.

Cerebrospinal (sē-rō′brō-spal) fluid (CSF) is a clear, colorless liquid that circulates within the ventricles and subarachnoid space. CSF bathes the exposed surfaces of the central nervous system and completely surrounds it. CSF performs several important functions:

- **Buoyancy.** The brain floats within the CSF, thereby reducing its apparent weight by more than 95%; this prevents the brain from being crushed under its own weight. Without CSF to support it, portions of the brain would sink through the foramen magnum.

- **Protection.** CSF provides a liquid cushion to protect delicate neural structures from sudden movements. When you try to walk quickly in a swimming pool, your movements are slowed as the water acts as a “movement buffer.” CSF likewise helps slow movements of the brain if the skull or body moves suddenly and forcefully.

- **Environmental stability.** CSF transports nutrients and chemical messengers to the brain and removes waste products from the brain. Additionally, CSF protects nervous tissue from chemical fluctuations that would disrupt neuron function. The waste products and excess CSF are eventually transported into the venous circulation.

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**Figure 13.7 Ventricles of the Brain.** The ventricles are formed from the embryonic neural canal. They contain cerebrospinal fluid (CSF), which transports nutrients, chemical messengers, and waste products. (a) Lateral and (b) anterior views show the positioning and relationships of the ventricles.
CSF Formation, Circulation, and Removal

The production of CSF by the brain occurs at a rate of about 500 milliliters (mL) (or 1/2 liter) per day, with the volume of CSF in the subarachnoid space at any given time ranging between 100 mL and 160 mL. Cerebrospinal fluid is initially formed by the choroid plexus (ko’royd plek’sūs; chorioeides = membrane, plexus = a braid), a region of specialized tissue in each ventricle. The choroid plexus is composed of a layer of glial cells called ependymal (ep-en’di-mål; ependyma = an upper garment) cells (see section 12.4b) and the blood capillaries that lie within the pia mater (figure 13.8). Thus, in order to enter a ventricle, fluid within the blood must cross these structures: blood capillary wall, the pia mater, and an ependymal cell.

The formation of CSF by the choroid plexus occurs as follows: Blood plasma (a watery fluid containing glucose and ions such as K⁺, Na⁺, Cl⁻, and Ca²⁺; see section 18.2) is filtered from the blood capillaries across both the capillary wall and the pia mater. Its composition is modified by the ependymal cell as it moves through these cells. Fluid is then released into a ventricle and is now referred to as CSF. The chemical composition of CSF within the ventricles is slightly different from the filtered blood plasma with relatively more Na⁺ and Cl⁻ and relatively less glucose, K⁺, and Ca²⁺. (Note that red blood cells, white blood cells, platelets, and plasma proteins are not filtered from the blood capillaries and are thus not normal components of CSF.)

Following its formation, the clear CSF circulates through the ventricles, where additional fluid is added by ependymal cells (see figure 12.5). The CSF then circulates from the ventricles into the subarachnoid space. Once CSF enters the subarachnoid space, its volume increases with the addition of excess interstitial fluid from the brain (which is formed during capillary exchange; see section 20.3). Thus, CSF is collectively formed by the choroid plexus (about 30%), ependymal cells lining the ventricles (about 30%), and fluid added into the subarachnoid space (about 40%).

The CSF must be continuously reabsorbed from the subarachnoid space at the same rate as its formation so that the fluid does not accumulate and compress or damage the nervous tissue. Reabsorption of CSF occurs at arachnoid villi (vil’t; villi = shaggy hair), which are fingerlike extensions of the arachnoid mater that project through the dura mater into the dural venous sinuses. A collection of arachnoid villi form an arachnoid granulation (see figure 13.5). As additional CSF is formed, fluid pressure rises within the subarachnoid space, which forces CSF from the subarachnoid space across the arachnoid villi to return to the blood within the dural venous sinuses. As a result, the arachnoid villi provide a conduit for a one-way flow of excess CSF to be returned to the blood within the dural venous sinuses. Figure 13.9 describes in detail and shows the process of CSF production, circulation, and removal.

CLINICAL VIEW 13.5

Hydrocephalus

Hydrocephalus (hi-dro-sef’a-lus; hydro = water, kephale = head) refers to the pathologic condition of excessive CSF, which often leads to brain distortion. Most cases of hydrocephalus result from either an obstruction in CSF flow or impaired absorption of CSF at the arachnoid villi.

If hydrocephalus develops in a young child, the head becomes enlarged and neurologic damage may result. If hydrocephalus develops after the cranial sutures have closed, the brain may be compressed within the fixed cranium as the ventricles expand, resulting in permanent brain damage. Hydrocephalus may be treated surgically by implanting shunts (tubes) that drain excess CSF to other body regions (usually the drainage site is the peritoneum in the abdominal cavity). The fluid is then absorbed into the blood.

WHAT DID YOU LEARN?

1. Where is CSF first produced, where does it circulate, and how does it get removed?
2. What are the three main functions of cerebrospinal fluid?
1. CSF is produced by the choroid plexus in the ventricles.

2. CSF flows from the lateral ventricles, through the interventricular foramen into the third ventricle, and then through the cerebral aqueduct into the fourth ventricle.

3. CSF in the fourth ventricle passes through the paired lateral apertures or the single median aperture, and into the subarachnoid space as well as the central canal of the spinal cord.

4. As the CSF flows through the subarachnoid space, it provides buoyancy to support the brain.

5. Excess CSF flows into the arachnoid villi, then drains into the dural venous sinuses. The greater pressure on the CSF in the subarachnoid space ensures that CSF moves into the venous sinuses without permitting venous blood to enter the subarachnoid space.

**Figure 13.9 Production and Circulation of Cerebrospinal Fluid.** (a) A midsagittal section identifies the sites where cerebrospinal fluid (CSF) is formed and the pathway of its circulation toward the arachnoid villi. (b) CSF flows from the arachnoid villi into the dural venous sinuses.
**13.2d Blood-Brain Barrier**

**LEARNING OBJECTIVES**

11. Describe the components that form the blood-brain barrier.

12. Explain how the blood-brain barrier protects the brain.

Blood is delivered to the brain as part of the general circulation (see section 20.10a). The brain is protected from the contents within the blood by the **blood-brain barrier (BBB)**. This barrier strictly regulates which substances can and cannot be filtered from the blood to enter the interstitial fluid of the brain. As a result, the BBB helps prevent exposure of neurons in the brain to drugs, waste products in the blood, and variations in levels of normal substances (e.g., ions, hormones) that could adversely affect brain function.

The BBB is formed of specialized capillaries surrounded by astrocytes. Capillaries are typically composed of an endothelial lining resting on a basement membrane (see table 20.2, which shows types of capillaries). Capillaries forming the BBB exhibit three significant structural differences from other capillaries (figure 13.10). (1) The endothelial cells contain tight junctions, which prevent the passage of materials between cells (see section 4.6d). Thus, most substances are forced through the endothelial cells and their movement is controlled by membrane transport processes (see section 4.3). (2) The capillary wall is made more substantial by a thickened basement membrane that further restricts the passage of substances from the blood into the brain. (3) The capillaries forming the BBB are wrapped in the **perivascular feet of astrocytes** (discussed in section 12.4b), which form the outermost portion of the BBB. The BBB acts as a gatekeeper to control which materials pass from the blood into the brain.

However, this barrier is not absolute. Recall from section 4.3a that lipid-soluble molecules readily cross plasma membranes by simple diffusion. Thus, lipid-soluble molecules such as nicotine, alcohol, and some anesthetics, can diffuse across the endothelial plasma membranes (and thus past the blood-brain barrier) and into the interstitial fluid of the CNS to reach the brain neurons. In addition, drugs such as cocaine and methamphetamine can damage this barrier.

There are several important exceptions to the presence of the BBB in the brain. It is markedly reduced or missing in three distinct locations in the CNS: the choroid plexus, hypothalamus, and pineal gland. The capillaries of the choroid plexus must be permeable to produce CSF, and the hypothalamus and pineal gland produce certain hormones that must have ready access to the blood.

**WHAT DID YOU LEARN?**

10. How does the blood-brain barrier protect nervous tissue?

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**13.3 Cerebrum**

The **cerebrum** is the location of conscious thought processes and the origin of all complex intellectual functions. It is readily identified as the two large hemispheres on the superior aspect of the brain (see figure 13.1). The functional activities in your cerebrum enable you to read and comprehend the words in this textbook, turn its pages, form and remember ideas, and talk about what you’ve learned with your peers. It is the center of
your intelligence, reasoning, thought, memory, and judgment, as well as your voluntary control of skeletal muscle movement and conscious perception of your senses (e.g., vision, hearing, touch, smell, and taste). Associated with the cerebrum are distinctive regions of gray matter: a cerebral cortex and a cerebral nucleus. Recall from section 13.1c that the cerebral cortex (se-rē′bral kor′tek-s; cerebro = brain, cortex = bark) is the surface layer of gray matter of the cerebrum and that the cerebral nuclei are the internal, deep gray matter within the cerebrum. The cerebral gray matter, like all gray matter, functions as centers of integration and processing.

13.3a Cerebral Hemispheres

LEARNING OBJECTIVES

13. Describe the anatomic structure of the left and right cerebral hemispheres, and explain their general functions.
14. Identify the role of the corpus callosum.

The cerebrum is composed of two halves, called the left and right cerebral hemispheres (hem′i-sfĕr; hemi = half, sphaira = ball) (figure 13.11). The paired cerebral hemispheres are separated by a narrow, deep cleft called the longitudinal fissure, which extends along the midsagittal plane. The cerebral hemispheres are separate from one another, except at a few locations where bundles of axons called tracts form white matter regions that allow for communication between them (see figure 13.14). The largest of these white matter tracts, the corpus callosum (kŏr′pus kal′-ō′sŭm; corpus = body, callosum = hard) connects the hemispheres (see a midsagittal section of the corpus callosum in figure 13.1c). The corpus callosum provides the main method of communication between these hemispheres.

WHAT DO YOU THINK?

2. One past treatment for severe epilepsy was to cut the corpus callosum, thus confining epileptic seizures to just one cerebral hemisphere. How would cutting the corpus callosum affect communication between the left and right hemispheres?

WHAT DID YOU LEARN?

11. What is the general function of the cerebrum?
12. What is the function of the corpus callosum?

13.3b Lobes of the Cerebrum

LEARNING OBJECTIVE

15. Explain the physical boundaries, important features, and functions of each cerebral lobe.

Each cerebral hemisphere is divided into five anatomically distinct lobes. Four of these lobes are visible on the external surface and are named for the overlying cranial bones: the frontal, parietal, temporal, and occipital lobes (figure 13.12a). The fifth lobe, called the insula, is not visible at the surface of the hemispheres. The cerebral cortex of each lobe exhibits specific cortical regions and association areas.

The frontal lobe lies deep to the frontal bone and forms the anterior part of the cerebral hemisphere figures 13.11 and 13.12a). The frontal lobe ends posteriorly at a deep groove called the central sulcus that marks its boundary with the parietal lobe. The inferior border of the frontal lobe is marked by the lateral sulcus, a deep groove that separates the frontal and parietal lobes from the temporal lobe. An important anatomic feature of the frontal lobe is the precentral gyrus, which is the mass of nervous tissue immediately anterior to the central sulcus. The frontal lobe is primarily concerned with voluntary motor functions (including motor functions involved with speech), concentration, verbal communication, decision making, planning, and personality.

The parietal (pă-rī′tă-l) lobe lies deep to the parietal bone and forms the superoposterior part of each cerebral hemisphere. It terminates anteriorly at the central sulcus, posteriorly at a relatively indistinct parieto-occipital sulcus, and laterally at the lateral sulcus. An important anatomic feature of this lobe is the postcentral gyrus, which is the mass of nervous tissue immediately posterior to the central sulcus. The cerebral cortex of the parietal lobe is involved with general sensory functions, such as evaluating the shape and
(a) Lobes of the Brain and Their Functional Areas

- **Central sulcus**
- **Frontal lobe (retracted)**
  - Primary motor cortex (in precentral gyrus)
  - Premotor cortex
  - Frontal eye field
  - Motor speech area (Broca area)
  - Prefrontal cortex
- **Insula**
  - Primary gustatory cortex
- **Temporal lobe (retracted)**
  - Premotor cortex
  - Somatic motor association area
- **Parietal lobe**
  - Primary somatosensory cortex (in postcentral gyrus)
  - Somatosensory association area
- **Occipital lobe**
  - Primary visual cortex
  - Visual association area

(b) Motor and Association Areas

- **Motor speech area**
  - Regulates skeletal muscle movements involved with speech
- **Primary motor cortex**
  - Initiates voluntary skeletal muscle activity
- **Premotor cortex**
  - Somatic motor association area
  - Plans and coordinates learned, skilled motor activities involving skeletal muscles
- **Frontal eye field**
  - Regulates the skeletal muscles that perform movements for binocular vision
(d) Functional Brain Regions

Prefrontal cortex
- Involved with higher intellectual functions (concentration, decision making, planning), personality

Wernicke area
- This multi-association area helps us understand spoken and written language.

Primary gustatory cortex
- Processes taste information and provides conscious awareness of taste

Primary somatosensory cortex
- Receives and interprets somatic information from receptors for touch, proprioception, and pain

Primary auditory cortex
- Processes and interprets sounds, stores auditory memories

Primary visual cortex
- Processes, stores, and integrates visual information

(c) Sensory Areas

Primary olfactory cortex
- Provides conscious awareness of odors

INTEGRATE CONCEPT OVERVIEW

Figure 13.12 Anatomic and Functional Areas of the Cerebrum. (a) Each cerebral hemisphere is partitioned into five lobes, each typically containing specific cortical regions and association areas. (b) The major motor areas and their association cortices are highlighted. (c) The major sensory areas and their association cortices are shown. (d) A functional brain region acts as a multi-association area to integrate information from several association areas.
texture of objects being touched and sensory input regarding body position from proprioceptors within our joints and muscles.

The **temporal lobe** lies internal to the temporal bone and inferior to the lateral sulcus. The cerebral cortex of this lobe is involved with hearing and smell.

The **occipital lobe** lies internal to the occipital bone and forms the posterior region of each hemisphere. The cerebral cortex of the occipital lobe is responsible for processing incoming visual information and storing visual memories.

The **insula** (in′sū-lā; island) is a small lobe deep to the lateral sulcus. It can be observed by laterally reflecting (pulling aside) the temporal lobe to reach the insula.

Research has shown that specific structural areas of the cerebral cortex have distinct motor and sensory functions (as introduced in section 13.3b). In contrast, some higher mental functions, such as language and memory, are dispersed over large areas. We have organized the functions of the cerebral cortex into motor functions and their association areas and sensory functions and their association areas. Note that the central sulcus serves as an anatomic landmark that separates motor functions that are controlled by the frontal lobe, and sensory input that is relayed to the parietal lobe, occipital lobe, and temporal lobe, as well as the internal insula.

### Motor Areas

The cortical areas that control motor functions are housed within the frontal lobes, as just described. The **primary motor cortex**, also called the somatic motor area, is specifically located within the precentral gyrus of the frontal lobe (figure 13.12). Neurons in this area control voluntary skeletal muscle activity. The axons of these neurons project contralaterally (to the opposite side) within either the brainstem or the spinal cord. Thus, the left primary motor cortex controls the skeletal muscles on the right side of the body and the right primary motor cortex controls the skeletal muscles on the left side of the body.

The distribution of the primary motor cortex innervation to various body parts can be diagrammed as a **motor homunculus** (hō-mūngk′-ū-lūs; little man) on the precentral gyrus (figure 13.13a).

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**13.3c Functional Areas of the Cerebrum**

**LEARNING OBJECTIVES**

16. Locate and list the functions of the motor cortical regions and their association areas.

17. Differentiate among the sensory cortical regions and their association areas.

18. Explain the functions of the prefrontal cortex, and hypothesize why this brain region may function differently in adults and teenagers.

19. Describe the main actions of the Wernicke area.

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**WHAT DID YOU LEARN?**

List the five cerebral lobes and the main functions of each.
The bizarre, distorted proportions of the homunculus body reflect the amount of cortex dedicated to each body part. For example, the hands are represented by a much larger area of cortex than the trunk, because the hand muscles perform much more detailed, precise movements than do the trunk muscles. From a functional perspective, more motor activity is devoted to the human hand than in other animals because our hands are adapted for the precise, fine motor movements needed to manipulate the environment, and many motor units are devoted to muscles that move the hand and fingers.

Certain motor functions have been mapped to specific areas of the frontal lobe, including the motor speech area and the frontal eye field. The motor speech area (also known as the Broca area) is located in most individuals within the inferolateral portion of the left frontal lobe (figure 13.12). This region is responsible for regulating the breathing and controlling the muscular movements necessary for vocalization. The frontal eye field is within the frontal lobe immediately superior to the motor speech area. This cortical area controls and regulates the eye movements needed for reading and coordinating binocular vision. Some investigators include the frontal eye fields within the premotor area.

The primary motor cortical regions are connected to adjacent association areas that coordinate discrete skeletal muscle movement (figure 13.12). The premotor cortex also is called the somatic motor association area, and it is located within the frontal lobe immediately anterior to the precentral gyrus. It is primarily responsible for coordinating learned, skilled motor activities, such as moving the eyes in a coordinated fashion when reading a book or playing the guitar. An individual who has sustained trauma to this area would still be able to understand written letters and words but would have difficulty reading because his or her eyes couldn’t follow the lines on a printed page.

Sensory Areas

The cortical areas within the parietal, temporal, and occipital lobes and the insula are involved with conscious awareness of sensation, as described in section 13.3b. Each of the major senses has a distinct cortical area. Keep in mind as you read through this section that each primary sensory cortical region is the specific area of the cortex that receives sensory input from a specific type of receptor. In addition, each primary cortical region typically has an association area. The general function of association areas is to receive input from the primary region and integrate the current sensory input with previous experiences and memories.

The primary somatosensory cortex is housed within the postcentral gyrus of the parietal lobes. Neurons within this cortex receive general somatic sensory information from receptors of the skin regarding touch, pressure, pain, and temperature, as well as sensory input from proprioceptors from the joints and muscles regarding the conscious interpretation of body position. We typically are conscious of the sensations received by this cortex. A sensory homunculus may be traced on the postcentral gyrus surface, similar to a motor homunculus (figure 13.13b). The surface area of somatosensory cortex devoted to a body region indicates the amount of sensory information collected within that region. Thus, the lips, fingers, and genital region occupy larger portions of the homunculus, whereas the trunk of the body has proportionately fewer receptors, so its associated homunculus region is.
smaller. (However, the sensory homunculus shown is not precise, and there is extensive overlap between nearby body regions in the primary somatosensory cortex.)

The somatosensory association area is located within the parietal lobe and lies immediately posterior to the primary somatosensory cortex. It integrates sensory information and interprets sensations to determine the texture, temperature, pressure, and shape of objects. The somatosensory association area allows us to identify known objects without seeing them. For example, even when our eyes are closed, we can tell the difference between the coarse feel of a handful of dirt; the smooth and round shape of a marble; and the thin, flat, rounded surface of a coin because those interpretations of the textures and shapes have already been stored in the somatosensory association area.

The primary auditory cortex is located within the temporal lobe, where it receives and processes incoming visual information. The primary somatosensory cortex versus the somatosensory association area.

Sensory information for sight, sound, taste, and smell arrives at cortical regions other than the parietal lobe (figure 13.12). The primary visual cortex is located within the occipital lobe, where it receives and processes incoming visual information. The visual association area is located within the occipital lobe and it surrounds the primary visual area. It enables us to process visual information by analyzing color, movement, and form and to use this information to identify the things we see. For example, when we look at a face, the primary visual cortex receives bits of visual information, but the visual association area is responsible for integrating all of this information into a recognizable picture of a face.

The primary auditory cortex is located within the temporal lobe, where it receives and processes auditory information. The auditory association area is located within the temporal lobe, posterior to the primary auditory cortex. Within this association area, the cortical neurons interpret the characteristics of sound and store memories of sounds heard in the past. The next time a song is playing over and over in your head, you will know that this auditory association area is responsible.

The primary olfactory (of-a-k’tô-r; olfactus = smell) cortex is also located within the temporal lobe and provides conscious awareness of smells. Finally, the primary gustatory (gus’tà-rôr; gustatio = taste) cortex is within the insula and is involved in processing taste information.

**Functional Brain Regions**

A functional brain region acts as a multi-association area between lobes for integrating information from individual association areas. One functional brain region is the prefrontal cortex, located in the most anterior (rostral) portions of the frontal lobes (figure 13.12). The prefrontal cortex is associated with many higher intellectual functions such as complex thought, judgment, expression of personality, planning future behaviors, and decision making. By retrieving and coordinating information from multiple areas of the brain, the prefrontal cortex also will evaluate potential consequences of one’s actions, and in so doing will modulate one’s behavior based on societal norms. Interestingly, the prefrontal cortex continues to develop into our teens and 20s, as the axons continue to myelinate in this region and unnecessary synapses are removed. As a result, neuroscientists hypothesize, the reason many teenagers may have difficulty in planning and are impulsive, emotional, and risk takers is because the prefrontal cortex has not fully matured.

Another functional brain region is the Wernicke area, which is typically located only within the left hemisphere. The Wernicke area is involved in recognizing, understanding, and comprehending

### Clinical View 13.7

**Autism Spectrum Disorder**

Autism spectrum disorder (ASD), also known simply as autism, is a widely variable disorder of neural development that affects 1 in 88 children in the United States. It typically is recognized in early childhood, but diagnosis may be difficult until a child is older. Since 2013, the phrase autism spectrum disorder has been used to group and describe a variety of similar disorders, including autistic disorder, childhood disintegrative disorder, and Asperger syndrome. ASD varies in severity among those affected (hence the term spectrum in its name), but all are characterized by some form of social and communication difficulties. Some children may experience delays in language acquisition or may be completely nonverbal. Social interaction is difficult, ranging from inability to reciprocate interest during a conversation to withdrawal into the child’s “own world.” Intelligence also varies widely, from severe cognitive delay to savantlike skills in focused areas like math or music.

Individuals with ASD often are highly sensitive to stimuli such as loud noises or unfamiliar people, and may struggle in adjusting to changes in routine. Discomfort due to overstimulation or frustration in the inability to communicate can lead to tantrums or “meltdowns.” Other behaviors and traits commonly associated with ASD include repetitive motions like hand flapping or rocking, resistance to changes in routine (e.g., insisting on wearing the same shirt or eating the same meal each day), inability to engage in pretend play, inability to gauge the feelings of others, and intense interest in a particular activity or subject.

ASD is believed to stem from an inability of the brain to process information between neurons. However, the specific mechanisms and causes of the condition are not well understood or agreed upon. Genetic factors are thought to be involved, in part because autism affects males four times more often than females, and it often manifests in siblings. Biochemical and environmental factors have also been explored as potential causes, but few definitive answers exist. The disturbing aspect of this condition is that the number of cases has steadily increased since the late 1980s. The ability to detect the condition has improved, which may have increased the incidence of diagnosis.

A fraudulent paper published in 1988 claimed that the measles, mumps, and rubella (MMR) vaccine was linked to an increased risk of developing autism. In the years that followed, the paper was shown to have manipulated data and the study was inherently flawed, resulting in a retraction of the paper and the author (who was an MD) losing his medical license for serious professional misconduct. Numerous studies since then have shown no link between vaccines and developing autism. Unfortunately, the misconception that vaccines cause ASD still persists among some and has led to both a decline in vaccination rates and a resulting increase in disease outbreaks.

Treatment for ASD includes proven methods of speech and behavioral therapy, as well as holistic approaches that involve various diets, supplements, and experimental procedures. Some children with autism will go on to develop skills and live independent lives, whereas others will not. The biggest predictors for independence in adulthood are level of intelligence and ability to communicate.
spoken or written language. As you may expect, the Wernicke area and the motor speech area must work together for fluent communication to occur.

**WHAT DID YOU LEARN?**
14. Where is each of the motor areas located, and what are their functions?
15. In general, what is the purpose of the association areas?
16. Why does the prefrontal cortex perform differently in a teenager versus an adult?

### 13.3d Central White Matter

#### LEARNING OBJECTIVE
20. Identify the three main tracts of the central white matter.

The **central white matter** of the cerebrum lies deep to the gray matter of the cerebral cortex. It is composed primarily of myelinated axons, as described in section 13.1c. Most of these axons are grouped into bundles called **tracts**, which are classified as association tracts, commissural tracts, or projection tracts (figure 13.14).

**Association tracts** connect different regions of the cerebral cortex within the same hemisphere. Short association tracts are composed of arcuate (ar′kū-it; arcuatus = bowed) fibers; they connect neighboring gyri within the same lobe. (Fibers are bundles of axons.) An example of an association tract that is composed of arcuate fibers is the tract that connects the premotor cortex with the primary motor cortex, both of which are in the frontal lobe. The longer association tracts, called the **longitudinal fasciculi** (fā-sık′ū-lī; fascis = bundle), connect gyri in different lobes of the same hemisphere. An example of a longitudinal fasciculus is the tract that connects the Wernicke area to the motor speech area.

**Commissural** (kom-i-sūr′āl) tracts extend between the cerebral hemispheres through axonal bridges called commissures. The prominent commissural tracts that link the left and right cerebral hemispheres include the large, C-shaped corpus callosum and the smaller anterior and posterior commissure (see figure 13.17).

**Projection tracts** link the cerebral cortex to both the inferior brain regions and the spinal cord (figure 13.14). Examples of projection tracts are the corticospinal tracts that carry motor signals from the cerebrum to the brainstem and spinal cord (see section 14.4c). The packed group of axons in these tracts passing in between the cerebral nuclei and the gray matter of the thalamus is called the **internal capsule** (see figure 13.4a).

#### WHAT DID YOU LEARN?
17. What portions of the brain are linked by each type of tract: (a) association, (b) commissural, and (c) projection tracts?

**CONCEPT CONNECTION**
Recall from section 12.8c that nerve signals are relayed along axons. The tracts in the brain contain thousands of axons, which transmit nerve signals to allow for communication among the different regions of the brain.

**INTEGRATE**

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**Figure 13.14 Cerebral White Matter Tracts.** White matter tracts are composed primarily of myelinated axons. Three major groups of axons are recognized based upon their distribution. (a) A sagittal view shows arcuate fibers and longitudinal fasciculi association tracts, which extend between gyri within one cerebral hemisphere. (b) A coronal view shows how commissural tracts extend between cerebral hemispheres, whereas projection tracts extend between the cerebral hemispheres and the brainstem.
13.3e Cerebral Lateralization

LEARNING OBJECTIVES

21. Explain the phenomenon of cerebral lateralization.
22. Identify the functions of left and right hemispheres in most individuals.

Anatomically, the left and right cerebral hemispheres appear identical, but careful examination reveals a number of differences. Humans tend to have shape asymmetries of the frontal and occipital lobes of the brain, called petalias (pe′tal-ē-ă) (figure 13.15a). Right-handed individuals typically have right frontal petalias, meaning that the right frontal lobe projects farther than the left frontal lobe, and left occipital petalias, meaning that the left occipital lobe projects farther than the right occipital lobe. Conversely, left-handed individuals are inclined to have the reverse pattern (left frontal and right occipital petalias). The hemispheres also differ with respect to some of their functions. Each hemisphere tends to be specialized for certain tasks, a phenomenon called cerebral lateralization (lat′er-al′zā-shun) or hemispheric lateralization (figure 13.15b). Higher-order centers in both hemispheres tend to have different but complementary functions.

In most people, the left hemisphere is the categorical hemisphere. It usually contains the Wernicke area and the motor speech area. It is specialized for language abilities and is important in performing sequential and analytical reasoning tasks, such as those required in science and mathematics. This hemisphere appears to direct or partition information into smaller fragments for analysis. The term categorical hemisphere reflects this hemisphere’s function in categorization and identification.

The other hemisphere (the right, in most people) is called the representational hemisphere, because it is concerned with visuospatial relationships and analyses. It is the seat of imagination and insight, musical and artistic skill, perception of patterns and spatial relationships, and comparison of sights, sounds, smells, and tastes.

Both cerebral hemispheres remain in constant communication through commissures, especially through the corpus callosum, which contains hundreds of millions of axons that project between the hemispheres.

Laterization of the cerebral hemispheres develops early in life (prior to 5–6 years of age). In a young child, the functions of a damaged or removed hemisphere are often taken over by the other hemisphere before lateralization is complete. Some aspects of lateralization differ between the sexes. Women have a thicker posterior part of the corpus callosum due to additional commissural axons in this region. Adult males typically exhibit more lateralization than females and suffer more functional loss when one hemisphere is damaged.

Cerebral lateralization is highly correlated with handedness. Right-handed individuals tend to have a slightly different lateralization pattern than those who are left-handed. In about 95% of the population, the left hemisphere is the categorical hemisphere, thus correlating with the 90% incidence of right-handed individuals in the population. However, the correlation is not nearly as strict among left-handed people, who may have either hemisphere as their categorical hemisphere. Interestingly, a thicker corpus callosum in left-handers suggests that more signals may be relayed between their hemispheres. Finally, the left hemisphere is the speech-dominant hemisphere.
The cerebral nuclei (also called the basal nuclei) are paired, irregular masses of gray matter buried deep within the central white matter in the basal (deepest) region of the cerebral hemispheres inferior to the floor of the lateral ventricle (Figure 13.16; see also figure 13.4). These masses of gray matter also are sometimes incorrectly called the basal ganglia. However, the term ganglion [sing.] is best restricted to clusters of neuron cell bodies outside the CNS, whereas a nucleus is a collection of cell bodies within the CNS. In general, the cerebral nuclei primarily help regulate motor output initiated by the cerebral cortex, to help inhibit unwanted movements. Diseases that affect the cerebral nuclei (such as Parkinson disease and Huntington disease—see Clinical View 13.10: “Brain Ailments and Disorders”) often are associated with jerky, involuntary movements.
Cerebral nuclei have multiple components, and each component has its own specific functions related to the overall function of the cerebral nuclei. These components include the caudate nucleus, lentiform nucleus, claustrum, and amygdaloid body.

- The C-shaped caudate (kaw’dāt; cauda = tail) nucleus has an enlarged head and a slender, arching tail that parallels the curve of the lateral ventricle. Each time a person initiates a walking movement, the neurons in this nucleus stimulate the appropriate skeletal muscles to produce the pattern and rhythm of arm and leg movements associated with walking.

- The lentiform (len’ti-form; lenticula = lentil, forma = shape) nucleus is a compact, triangular mass, made up of both the putamen (pū-tā-men; shell) and globus pallidus (glō’būs pal’i-dūs; globus = ball, pallidus = pale), two masses of gray matter positioned between the insula lobe of the cerebrum and the lateral wall of the thalamus. The putamen functions in controlling skeletal muscular movement at the subconscious level. The globus pallidus both excites and inhibits the activities of the thalamus to regulate skeletal muscle tone.

- The claustrum (klaws’trūm; barrier) is a thin sliver of gray matter formed by a layer of neurons located immediately internal to the insula. It processes visual information at a subconscious level.

- The amygdaloid (ā-mig’dā-loyd; amygdala = almond) body (often just called the amygdala) is an expanded region at the tail of the caudate nucleus. It participates in the expression of emotions, control of behavioral activities, and development of moods (see the limbic system in section 13.7a).

The term corpus striatum (strī-ă-tūm; striped) describes the striated appearance of the white matter of the internal capsule positioned between the gray matter of the caudate nucleus and the lentiform nucleus.

**WHAT DID YOU LEARN?**

What is the general function of the cerebral nuclei, and what are the anatomic components of the nuclei?

**INTEGRATE**

### CLINICAL VIEW 13.10

**Brain Ailments and Disorders**

Brain disorders may be expressed by a malfunction in processing of sensory input, transmitting motor output, or some combination of both activities. Some brain disorders include headache, cerebral palsy, Huntington disease, and Parkinson disease.

- **Headache** typically is due either to dilated blood vessels in the skull or muscle contraction (as may occur when an individual develops eyestrain from staring at a computer screen too long). **Migraine headaches** are severe, recurring headaches that often affect only one side of the head. Headaches are not a brain disorder, but they sometimes accompany other diseases or brain disorders.

- **Cerebral palsy** (paw’lā) is a group of neuromuscular disorders that usually result from damage to an infant’s brain before, during, or immediately after birth. Three forms of cerebral palsy involve impairment of skeletal motor activity to some degree: athetoid, characterized by slow, involuntary, writhing hand movements; ataxic, marked by lack of muscular coordination; and spastic, exhibiting increased muscular tone. Intellectual impairment and speech difficulties sometimes accompany this disorder.

- **Huntington disease** is an autosomal dominant hereditary disease that affects the cerebral nuclei. It causes rapid, jerky, involuntary movements that usually start unilaterally in the face but over months and years progress to the arms and legs. Progressive intellectual deterioration also occurs, including personality changes, memory loss, and irritability. The disease has an onset age of mid 30s–40s and is fatal within 10 to 20 years.

**Parkinson disease** is a slow-progressing neurologic condition that affects muscle movement and balance. The cause of the disease is under debate; some researchers believe the disease results from abnormal clumpings of a protein called α-synuclein in the brain, while other research suggests the disease is an autoimmune disorder (because some with the disease have dopamine producing neurons that exhibit antigens, which could trigger an autoimmune response).

Parkinson patients exhibit stiff posture, an expressionless face, slow voluntary movements, a resting tremor (especially in the hands), and a shuffling gait. The disease is caused by a deficiency of the neurotransmitter dopamine, which results from decreased dopamine production of degenerating neurons in the substantia nigra (nuclei in the midbrain; see section 13.5a). Dopamine deficiency prevents brain cells of the substantia nigra from inhibiting the cerebral nuclei. By the time symptoms develop, the person has lost 80–90% of the cells responsible for producing dopamine. Current treatments include medications that enhance the amount of dopamine in the remaining cells of the substantia nigra and medications (e.g., rasagiline) to treat the symptoms.

Boxer Muhammad Ali and actor Michael J. Fox, two famous Parkinson patients, have advocated for increased research funding for the disease.
13.4 Diencephalon

The diencephalon is sandwiched between the inferior regions of the cerebral hemispheres and for this reason is often referred to as the “in-between brain.” The diencephalon components include the epithalamus, the thalamus, and the hypothalamus (figure 13.17). The 3rd ventricle also is associated with the diencephalon (see figure 13.7).

13.4a Epithalamus

**LEARNING OBJECTIVES**

24. List components located in the epithalamus, and describe their functions.

25. Explain how circadian rhythm is regulated.

The epithalamus (ep′i-thal′ă-mūs epi = upon) partially forms the posterior roof of the diencephalon and covers the third ventricle. The posterior portion of the epithalamus houses the pineal gland and the habenular nuclei.

The pineal (pin′ē-āl; pineus = pineconelike) gland, or pineal body, is an endocrine gland (see section 17.11a). It secretes the hormone melatonin, which appears to help regulate day-night cycles known as the body’s circadian rhythm. (Some companies are marketing the sale of melatonin in pill form as a cure for jet lag and insomnia, although this cure has yet to be proven.)

The habenular (hā-ben′ū-lār; habena = strap) nuclei relay signals from the limbic system (see section 13.7a) to the midbrain and are involved in visceral and emotional responses to odors.

**WHAT DID YOU LEARN?**

21. What are the location and function of the pineal gland?

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**Figure 13.17 Diencephalon.** The diencephalon (outlined in purple) encloses the third ventricle and connects the cerebral hemispheres to the brainstem. The right portion of the diencephalon is outlined and shown here in midsagittal section. The diencephalon and its major subdivisions are listed in bold. APF R
13.4b Thalamus

LEARNING OBJECTIVE

26. Discuss the action of the thalamus on sensory information.

The thalamus (thal′-ă-mūs; bed) forms the superolateral walls of the third ventricle (figure 13.17), and its paired oval masses of gray matter that lie on either side of the third ventricle (figure 13.18). When viewed in midsagittal section, the thalamus is located between the anterior commissure and the pineal gland. The interthalamic adhesion (or intermediate mass) is a small, midline mass of gray matter that connects the right and left thalamic bodies.

Each part of the thalamus has about a dozen major thalamic nuclei that are organized into groups; axons from these nuclei project to particular regions of the cerebral cortex. Sensory nerve signals from all the conscious senses except olfaction converge on the thalamus and synapse in at least one of its nuclei. For example, the ventral posterior nuclei relay sensory information to the primary somatosensory cortex of the parietal lobe, whereas auditory information is relayed through the medial geniculate nuclei.

The thalamus is the principal and final relay point for incoming sensory information that is processed and then projected to the appropriate lobe of the cerebral cortex. Only a relatively small portion of the sensory information that arrives at the thalamus is forwarded to the cerebrum because the thalamus acts as an information filter. For example, the thalamus is responsible for filtering out the sounds and sights in a crowded cafeteria when you are trying to study. The thalamus also “clues in” the cerebrum about where this sensory information came from. For example, the thalamus lets the cerebrum know that sensory information it receives came from the eye, indicating that the information is visual.

WHAT DO YOU THINK?

1. If there were no thalamus, how would this affect the cerebrum’s interpretation of sensory stimuli?

WHAT DID YOU LEARN?

22. What is the general function of the thalamus?

13.4c Hypothalamus

LEARNING OBJECTIVE

27. Describe seven functions of the hypothalamus.

The hypothalamus (hi′pŏ-thal′-ă-mūs; hypo = under) is the anteroinferior region of the diencephalon. A thin, stalklike infundibulum (in-fŭn-di-bŭ-lŭm; funnel) extends inferiorly from the hypothalamus to attach to the pituitary gland (figure 13.19).

The hypothalamus has numerous functions controlled by specific nuclei, as listed in table 13.3:

- Master control of the autonomic nervous system. The hypothalamus is a major autonomic integration center. In essence, it is the “president” of the corporation known as the autonomic nervous system (described in detail in chapter 15). It projects descending axons to autonomic nuclei in the brainstem that influence heart rate, blood pressure, digestive activities, and respiration.

- Master control of the endocrine system. The hypothalamus is also “president” of another corporation—the endocrine system—overseeing most but not all of that system’s functions. The hypothalamus secretes hormones that control secretory activities in the anterior pituitary gland, and it produces both antidiuretic hormone and oxytocin, which are stored in the posterior pituitary gland. Its function in the endocrine system is described in detail in section 17.7.

- Regulation of body temperature. The body’s thermostat is located within the hypothalamus. Neurons in the preoptic area detect altered blood temperatures and signal other hypothalamic nuclei, which control the mechanisms that heat or cool the body (see sections 1.6b, 6.1d, and 27.8b).

Figure 13.18 Thalamus. (a) Lateral view of the brain identifies the approximate location of the thalamus. (b) The thalamus is composed of clusters of nuclei organized into groups, as shown in this enlarged view. Not all nuclei may be seen from this angle. AP/RE
**Hypothalamus.** The hypothalamus is located anteroinferior to the thalamus and is organized into multiple nuclei.

<table>
<thead>
<tr>
<th>Nucleus or Hypothalamic Region</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior nucleus</td>
<td>“Thirst center” (stimulates fluid intake); autonomic control center</td>
</tr>
<tr>
<td>Arcuate nucleus</td>
<td>Regulates appetite, release of gonadotropin-releasing hormone, release of growth hormone–releasing hormone, and release of prolactin-inhibiting hormone</td>
</tr>
<tr>
<td>Mammillary body</td>
<td>Directs sensations related to olfaction; controls swallowing</td>
</tr>
<tr>
<td>Paraventricular nucleus</td>
<td>Produces oxytocin primarily</td>
</tr>
<tr>
<td>Preoptic area</td>
<td>“Thermostat” (regulates body temperature)</td>
</tr>
<tr>
<td>Suprachiasmatic nucleus</td>
<td>Regulates sleep-wake (circadian) rhythm</td>
</tr>
<tr>
<td>Supraoptic nucleus</td>
<td>Produces antidiuretic hormone (ADH) primarily</td>
</tr>
<tr>
<td>Ventromedial nucleus</td>
<td>“Satiety center” (produces hunger and satiety sensations)</td>
</tr>
</tbody>
</table>

**LEARNING STRATEGY**

Four hypothalamus functions—regulation of body temperature, control of food intake, control of water intake, and regulation of sleep-wake (circadian) rhythms—are all related to body comfort. Consider that you feel most comfortable when you are not too hot or cold, are not hungry or thirsty, and have adequate sleep.

**WHAT DID YOU LEARN?**

13.5 How does the hypothalamus control our feelings of hunger and thirst?

**Brainstem**

The brainstem connects the cerebrum, diencephalon, and cerebellum to the spinal cord. Three regions form the brainstem. From superior to inferior, these include the midbrain, the pons, and the medulla oblongata (figure 13.20).

The brainstem is a bidirectional passageway for all tracts extending between the major regions of the brain and the spinal cord. It also contains many autonomic centers and reflex centers required for regulating body functions necessary for survival (e.g., breathing, blood pressure) and it houses nuclei of many of the cranial nerves. The reticular formation, which extends through all three regions of the brainstem, is discussed in section 13.7b.
The midbrain, or mesencephalon, is the superior portion of the brainstem. The midbrain has several major components, which are visible externally in Figure 13.20. Figure 13.21 shows cross sections of the midbrain and allows us to view its internal structures. The tracts (bundles of myelinated axons) associated with the midbrain are first discussed and then the midbrain nuclei (gray matter) are described based on their position from anterior to posterior.

**13.5a Midbrain**

**LEARNING OBJECTIVES**

28. List the major features of the midbrain.

29. Identify the locations and functions of structures that are visible in a cross-sectional view of the midbrain.

30. Explain the involuntary actions produced by the superior and inferior colliculi of the tectal plate.
Cerebral peduncles (pē’dūŋ-kl; pedunculus = little foot) are motor tracts located on the anterolateral surfaces of the midbrain. Descending axon bundles of the pyramidal system (corticospinal tracts; see section 14.4c) project through the cerebral peduncles and relay voluntary motor commands from the primary motor cortex of each cerebral hemisphere. Additionally, the superior cerebellar peduncles connect the midbrain to the cerebellum (figure 13.20). Bands of myelinated sensory axons composing a medial lemniscus extend from the medulla oblongata, through the pons and midbrain, to the thalamus (see section 14.4b).

The substantia nigra (sū-bān’s-tā-nē’ə-grā; substantia = substance, niger = black) consists of bilaterally symmetric nuclei within the midbrain (figure 13.21). Its name derives from its almost black appearance due to melanin pigmentation. The substantia nigra houses clusters of neurons that produce the neurotransmitter dopamine, which affects brain processes to control movement, emotional response, and ability to experience pleasure and pain. Degeneration of these cells in the substantia nigra is a pathology that underlies Parkinson disease (see Clinical View 13.10: “Brain Ailments and Disorders”).

The tegmentum (teg-men’tūm; covering structure) is sandwiched between the nuclei of the substantia nigra and the periaqueductal gray matter. The tegmentum contains the pigmented red nuclei and the reticular formation. The reddish color of these nuclei is due to both blood vessel density and iron pigmentation in the neuronal cell bodies. The tegmentum integrates information from the cerebrum and cerebellum and issues involuntary motor commands to the erector spinae muscles of the back (see section 11.4) to help maintain posture while standing, bending at the waist, or walking.

Extending through the midbrain is the cerebral aqueduct connecting the third and fourth ventricles (see section 13.2b); it is surrounded by a region called the periaqueductal gray matter. The nuclei of the oculomotor nerve (CN III) and the trochlear nerve (CN IV) are housed in the midbrain (these nerves are described in detail in section 13.9).

The tectum (tek’tūm; roof) is the most posterior region of the midbrain. It contains two pairs of sensory nuclei, the superior and inferior colliculi, which are collectively called the tectal plate (quadrigeminal plate, or corpora quadrigemina). These nuclei are relay stations in the processing pathway of visual and auditory sensory input. The superior colliculi (šū-pər′ō-lık’’-yû-lê; sing., colliculus = mound) are the superior nuclei. They are called visual reflex centers because they help visually track moving objects and control reflexes such as turning the eyes and head in response to a visual stimulus. For example, the superior colliculi are at work when you think you see a large animal running at you and turn suddenly toward the image. The paired inferior colliculi are the auditory reflex centers, meaning that they control reflexive turning of the head and eyes in the direction of a sound, such as a sudden, loud bang.

**WHAT DID YOU LEARN?**

24. What is the function of the substantia nigra, and what disease may affect its proper working?

25. What parts of the midbrain contain paired visual and auditory sensory nuclei?

### 13.5b Pons

#### LEARNING OBJECTIVES

31. Identify the respiratory center located in the pons.

32. Identify the actions of the superior olivary complex.

The pons (ponz; bridge) is a bulging region on the anterior part of the brainstem (figures 13.20 and 13.22). Sensory and motor tracts are located within the pons and extend through it to connect to the brain and spinal cord. Additionally, the middle cerebellar peduncles are transverse axons that connect the pons to the cerebellum.

The pons houses automatic nuclei in the pontine respiratory center (previously called the pneumotaxic [nū-mō-tā’ksik] center). This vital center, along with the medullary respiratory center within the medulla oblongata, regulates the skeletal muscles of breathing. The primary function of the pontine respiratory center is to regulate a smooth transition between breathing in and breathing out (see section 23.5c).

The superior olivary nuclei are located in the inferior portion of the pons. Each nucleus receives auditory input and is involved in the pathway for sound localization.

The pons also houses sensory and motor cranial nerve nuclei for the trigeminal (CN V), abducens (CN VI), and facial (CN VII) cranial nerves. Some of the nuclei for the vestibulocochlear cranial nerve (CN VIII) also are located there.
The medulla oblongata (me-dū’lā ob-long-gah’tū; medulla = marrow or middle, oblongus = rather long) is often simply called the medulla. It is the most inferior part of the brainstem and is continuous with the spinal cord inferiorly. The most inferior portion of the medulla has a flattened, rounded shape and narrow central canal. As this tubelike opening extends (superiorly and anteriorly) toward the medulla has a flattened, rounded shape and narrow central canal. As this tubelike opening extends (superiorly and anteriorly) toward the pons, the central canal enlarges and becomes the inferior portion of the fourth ventricle. All communication between the brain and spinal cord involves tracts that ascend or descend through the medulla oblongata (figure 13.23; see also figures 13.20 and 13.22).

Several external landmarks are readily visible on the medulla oblongata. The anterior surface exhibits two longitudinal ridges called the pyramids (pir’ā-mid), which house the motor projection tracts called the corticospinal (pyramidal) tracts that extend through the medulla oblongata (see section 14.4c). In the anterior region of the medulla, most of the axons of the pyramidal tracts cross to the opposite side of the brain at a point called the decussation (dé-kō-sā’shūn; decussate = to cross in the form of an X) of the pyramids. As a result of the crossover, each cerebral hemisphere controls the voluntary movements of the opposite side of the body. Immediately lateral to each pyramid is a distinct bulge, called the olive, which contains a large fold of gray matter called the inferior olivary nucleus. The inferior olivary nuclei relay ascending sensory nerve signals, especially proprioceptive information, to the cerebellum. Additionally, paired inferior cerebellar peduncles (see figure 13.20) are tracts that connect the medulla oblongata to the cerebellum.

The medulla oblongata contains several autonomic nuclei, which group together to form centers that regulate functions vital for life. The most important autonomic centers in the medulla oblongata and their functions include:

- **The cardiovascular center**, composed of both the cardiac center, which regulates both the heart’s rate and its force of contraction to alter cardiac output (see section 19.5b), and the vasomotor center, which controls the contraction and relaxation of smooth muscle within the walls of the smallest arteries (the arterioles) to alter these vessels’ diameter. Both cardiac output and blood vessel diameter influence blood pressure (see section 20.6).

- **The medullary respiratory center**, which regulates the respiratory rate. It is composed of a ventral respiratory group and a dorsal respiratory group. These groups are influenced by the pontine respiratory center (see section 23.5c). The primary function of the medullary respiratory center is to rhythmically initiate nerve signals that cause contraction of breathing muscles (see section 23.5c).

- Other nuclei in the medulla, which are involved in coughing, sneezing, salivation, swallowing, gagging, and vomiting reflexes.

**WHAT DO YOU THINK?**

Based on your understanding of the medulla oblongata’s functions, would you expect severe injury to the medulla oblongata to cause death or merely be disabling? Why?
The cerebellum (ser-e-bel’ūm; little brain) is the second largest part of the brain. It coordinates fine control over skeletal muscle actions and stores memories of movement patterns, such as the playing of scales on a piano.

### 13.6a Structural Components of the Cerebellum

**LEARNING OBJECTIVES**

35. Name the parts and landmarks of the cerebellum.

36. Identify the three tracts through which the brainstem is linked to the cerebellum.

The cerebellum is composed of left and right cerebellar hemispheres (figure 13.24). Each hemisphere consists of two lobes, the anterior lobe and the posterior lobe, which are separated by the primary fissure. A narrow band of nervous tissue known as the vermis (ver’mis; worm) lies along the midline between the left and right cerebellar lobes. The cerebellar hemispheres and vermis have surface folds called folia (fō’lē-ā; folium = leaf). (These folds are similar to the gyri of the cerebrum.)

The cerebellum is partitioned internally into three regions: an outer gray matter called the cerebellar cortex, an internal region of white matter, and the deepest gray matter layer that is composed of cerebellar nuclei. The internal region of white matter is called the arbor vitae (ar’bōr vit’e; arbor = tree, vita = life) because its distribution pattern resembles the branches of a tree (see section 13.1c).

Three thick nerve tracts, called peduncles, connect the cerebellum with the brainstem (see figure 13.20b). The superior cerebellar peduncles connect the cerebellum to the midbrain (see section 13.5a). The middle cerebellar peduncles connect the cerebellum to the pons (see section 13.5b). The inferior cerebellar peduncles connect the cerebellum to the medulla oblongata (see section 13.5c).

### WHAT DID YOU LEARN?

27. Where are the pyramids located, and what is their function?

28. What are the main autonomic centers located in the medulla?

### 13.6b Functions of the Cerebellum

**LEARNING OBJECTIVE**

37. Explain the functions of the cerebellum.
The cerebellum lies posterior to the pons and medulla oblongata of the brainstem. (a) A midsagittal section shows the relationship of the cerebellum to the brainstem. (b) A superior view compares the anterior and posterior lobes of the cerebellum. (Note: The cerebrum and diencephalon have been removed.)

**INTEGRATE**

**CONCEPT CONNECTION**

The precise movements of our fingers rely on multiple systems. The skeletal system provides structural support for the muscles and tissues within the fingers, the muscular system is responsible for the movement of the fingers, and the nervous system sends the nerve signals to the muscular system to control the patterned movements.

The cerebellum has several additional functions. Skeletal muscle activity is adjusted to maintain equilibrium and posture. It also receives proprioceptive (sensory) information from the muscles and joints and uses this information to regulate the body’s position. For example, you are able to balance on one foot because the cerebellum takes the proprioceptive information from the body joints and maps out a muscle tone plan to keep the body upright. Finally, the proprioceptive information from the body’s muscles and joints is sent to the cerebellum and then to the cerebrum. Thus, the cerebrum is made aware of the position of each body joint and its muscle tone, even if the person is not looking at the joint. For example, if you close your eyes, you are still aware of which body joints are flexed and which are extended.

**INTEGRATE**

**CLINICAL VIEW 13.11**

**Effects of Alcohol and Drugs on the Cerebellum**

A variety of drugs, and alcohol in particular, can temporarily or permanently impair cerebellar function. Alcohol intoxication leads to the following symptoms of impaired cerebellar function, which are used in the classic sobriety tests performed by police officers:

- **Disturbance of gait.** A person under the influence of alcohol or drugs rarely walks in a straight line but appears to sway and stagger. In addition, falling and bumping into objects are likely, due to the temporary cerebellar disturbance.
- **Loss of balance and posture.** When attempting to stand on one foot, a person who is intoxicated usually tips and falls over.
- **Inability to detect proprioceptive information.** When asked to close the eyes and touch the nose, a person under the influence of drugs or alcohol frequently misses the mark. This impairment is due to reduced ability to sense proprioceptive information, compounded by uncoordination of skeletal muscles.
Voluntary movements
The primary motor cortex and the basal nuclei in the forebrain send impulses through the nuclei of the pons to the cerebellum.

Assessment of voluntary movements
Proprioceptors in skeletal muscles and joints report degree of movement to the cerebellum.

Integration and analysis
The cerebellum compares the planned movements (motor signals) against the results of the actual movements (sensory signals).

Corrective feedback
The cerebellum sends impulses through the thalamus to the primary motor cortex and to motor nuclei in the brainstem.

Figure 13.25 Cerebellar Pathways. Input to the cerebellum from the motor cortex of the cerebrum and the pons (dark red arrows), and the sensory input relayed from proprioceptors to the cerebellum (blue arrow). Within the cerebellum, the integration and analysis of input information occurs (green arrows). Corrective feedback output from the cerebellum (yellow arrows) extends through the cerebellar peduncles (not shown).

The cerebellum continuously receives convergent input from both the various sensory pathways and the motor pathways in the brain (figure 13.25). In this way, the cerebellum unconsciously perceives the position of the body, receives the plan for movement, and then follows the activity to see if it was carried out correctly. When the cerebellum detects a disparity between the intended and actual movement, it may generate error-correcting nerve signals. These nerve signals are transmitted to both the premotor and primary motor cortices via the brainstem and the thalamus. Descending pathways then transmit these error-correcting signals to the motor neurons. Thus, the cerebellum influences and controls movement by indirectly affecting the excitability of motor neurons.

WHAT DID YOU LEARN?
31 What are the functions of the cerebellum?

13.7 Functional Brain Systems
The brain has two important functional systems that work together for a common purpose. These are considered functional brain systems because their structures are not confined to one major region of the brain but are located throughout two or more regions of the brain. These systems are the limbic system and the reticular formation.

13.7a Limbic System

LEARNING OBJECTIVES
38. Describe the main functions of the limbic system.
39. List the seven structures that compose the limbic system, and summarize their actions.

The limbic (lim’bik; limbus = edge) system is composed of multiple cerebral and diencephalic structures that collectively process and experience emotions. Thus, the limbic system is sometimes referred to as the emotional brain. The structures of the limbic system form a ring or border around the diencephalon. Although neuroanatomists continue to debate the components of the limbic system, the brain structures commonly recognized are shown in figure 13.26 and listed here:

1. The cingulate (sin’gū-lāt; cingulum = girdle, to surround) gyrus is an internal mass of cerebral cortex located within the longitudinal fissure and superior to the corpus callosum. This cortical mass may be seen only in sagittal section, and it surrounds the diencephalon. It receives input from the other components of the limbic system.
2. The parahippocampal gyrus is a mass of cerebral cortical tissue in the temporal lobe. Its function is associated with the hippocampus.
3. The hippocampus (hip-ō-kam’pəs; seahorse) is a component of the cerebrum located superior to the parahippocampal gyrus. It connects to the diencephalon via the fornix. As its name implies, this nucleus is shaped like a seahorse. Both the hippocampus and the parahippocampal gyrus are essential in storing memories and forming long-term memory.
4. The amygdaloid body (of the cerebral nuclei) connects to the hippocampus. The amygdaloid body is involved in several aspects of emotion, especially fear. It can also help store and code memories based on how a person emotionally perceives them—for example, as related to fear, extreme happiness, or sadness.
5. The **olfactory bulbs**, **olfactory tracts**, and **olfactory cortex** are part of the limbic system as well. You have probably experienced how particular odors can provoke certain emotions or be associated with certain memories (see section 16.3a).

6. The **fornix** (fōr′niks; arch) is a thin tract of white matter that connects the hippocampus with limbic system structures of the diencephalon.

7. Various nuclei in the diencephalon, such as the **anterior thalamic nuclei**, the **habenular nuclei** of the epithalamus, the **septal nuclei**, and the **mammillary** (mam′i-lār-ē; mammilla = nipple) **bodies** of the hypothalamus, interconnect other parts of the limbic system and contribute to its overall function.

**WHAT DID YOU LEARN?**

- What are the components of the limbic system?
- What are the main functions of the limbic system?

### 13.7b Reticular Formation

#### LEARNING OBJECTIVES

40. Describe the components and function of the reticular formation.

41. Explain the anatomy and function of the reticular activating system (RAS).

Projecting vertically through the core of the midbrain, pons, and medulla is a loosely organized mass of gray matter called the **reticular formation** (figure 13.27). The reticular formation extends slightly into the diencephalon and the spinal cord as well. This functional brain system has both motor and sensory components.

The motor component of the reticular formation communicates with the spinal cord and is responsible for regulating muscle tone (especially when the muscles are at rest). This motor component also assists in autonomic motor functions, such as respiration, blood pressure, and heart rate, by working with the autonomic centers in the medulla and pons.

Figure 13.26 **Limbic System.** The components of the limbic system affect behavior and emotions. The olfactory cortex of the temporal lobe and the habenular nuclei of the epithalamus are not shown.

Figure 13.27 **The Reticular Formation.** The reticular formation receives and processes various types of input from sensory receptors (blue arrows). It participates in cyclic activities such as arousing the cortex to consciousness (purple arrows) and controlling the sleep-wake cycle. Some motor output from the reticular formation influences muscle activity (red arrow).
The sensory component of the reticular formation is responsible for alerting the cerebrum to incoming sensory information. This sensory component is called the reticular activating system (RAS), and it contains sensory axons that project to the cerebral cortex. The RAS processes visual, auditory, and touch stimuli and uses this information to keep us in a state of mental alertness. Additionally, the RAS arouses us from sleep. The sound of an alarm clock can awaken us because the RAS receives this sensory stimulus and sends it to the cerebrum. Conversely, under conditions of little or no stimuli, such as when you are in bed with the lights out and no sounds are disturbing you, the RAS is not stimulated and you find it easier to sleep.

Consciousness includes an awareness of sensation, voluntary control of motor activities, and the activities necessary for higher mental processing. It involves the simultaneous activity of large areas of the cerebral cortex. Levels of consciousness exist on a continuum. The highest state of consciousness and cortical activity is alertness, in which the individual is responsive, aware of self, and well oriented to person, place, and time.

**WHAT DID YOU LEARN?**

44. How is the reticular activating system related to the reticular formation?

## 13.8 Integrative Functions and Higher-Order Brain Functions

Higher-order brain functions include learning, memory, and reasoning. These functions occur within the cerebral cortex and involve multiple brain regions connected by complicated networks and arrays of axons. Both conscious and unconscious processing of information are involved in higher-order brain functions, and this processing may be continually adjusted or modified.

### 13.8a Development of Higher-Order Brain Functions

**LEARNING OBJECTIVE**

42. Describe the relationship between age and higher-order brain functioning.

From infancy on, our motor control and processing capabilities become increasingly complex as we grow and mature. During the first year of life, the number of cortical neurons continues to increase. The myelination of many CNS axons continues throughout the first 2 years. (As a result, pediatricians recommend that infants and toddlers drink whole milk instead of skim milk, so their bodies will have adequate fat intake to support the development of myelin and the brain in general.) The brain grows rapidly in size and complexity so that by the age of 5, brain growth is 95% complete. (The rest of the body doesn’t reach its adult size until puberty.)

As the CNS continues to develop, some neurons expand their number of connections, providing the increased number of synaptic junctions required for increasingly complex reflex activities and processing. During this same period, the brain will “prune” various synaptic connections, so only the most commonly used connections will remain. Some CNS axons remain unmyelinated until the teenage years (e.g., some of the axons in the prefrontal cortex). In general, the axons of PNS neurons continue to myelinate past puberty. A person’s ability to carry out higher-order brain functions is a direct result of the level of nervous system maturation.

**WHAT DID YOU LEARN?**

35. What are some implications of the brain’s anatomic development not being complete until the mid-teens?

## 13.8b Electroencephalogram

**LEARNING OBJECTIVE**

43. Describe how an electroencephalogram examines brain activity.

An electroencephalogram (EEG) is a diagnostic test where electrodes are attached to the head to record the electrical activity of the brain (figure 13.28). This procedure is performed to investigate sleep disorders and lesions, and to determine if an individual is in a coma or a persistent vegetative state (see Clinical View 13.12: “Pathologic States of Consciousness”). EEGs also may evaluate a seizure, which is an event of abnormal electrical activity in the brain. There are different types of seizures, some of which may result in a brief blackout and others that may result in shaking and muscle spasms. Epilepsy is the condition where a person experiences repeated seizures over time (see Clinical View 13.8: “Epilepsy and Cerebral Lateralization”).

An EEG measures and plots four types of brain waves (i.e., alpha, beta, theta, and delta). The distribution and frequency of these waves vary, depending upon whether the person is a child or an adult and if the individual is in a deep sleep, having a seizure, or experiencing a pathologic state of consciousness. For example, alpha and beta waves are typically seen in an awake or alert state, whereas theta and delta waves are more common during sleep. The presence of theta and delta waves in an awake adult is suggestive of a brain abnormality. Each electrode attached to a person’s head will register a brain wave over that region of the head, so a patient’s EEG printout will show multiple brain waves over a period of time.

**INTEGRATE**

**CLINICAL VIEW 13.12**

**Pathologic States of Unconsciousness**

When a person is asleep, he or she is technically unconscious, but not pathologically so. However, other unconscious conditions are pathologic. These pathologic states of unconsciousness may occur due to traumatic brain injury (TBI), CVA, low blood sugar, or diseases of the liver or kidney.

A brief loss of consciousness, termed fainting or syncope (sin’ko-pe; cutting short), often signals inadequate cerebral blood flow due to low blood pressure, as might follow hemorrhage or sudden emotional stress. Stupor (stä’per; stupeo = to be stunned) is a moderately deep level of unconsciousness from which the person can be aroused only by extreme repeated or painful stimuli.

A coma is a deep and profound state of unconsciousness from which the person cannot be aroused, even by repeated or painful stimuli. A person in a coma is alive but unable to respond to the environment and is not aware. A coma represents the deepest level of unconsciousness.

A persistent vegetative state is a long-term condition where the person may be unconscious or awake, but has no self-awareness or awareness of one’s surroundings. Some people in this state exhibit reflexive or spontaneous movements, such as moving their eyes, grimacing, crying, and smiling. However, there is no purposeful movement.
Sleep may be subdivided into two main types, **non-REM** (non-rapid eye movement) and **REM** (rapid eye movement) sleep. Both types are distinguished by their EEG patterns and the absence or presence of rapid eye movements, respectively. In addition, it is during REM sleep that we have our most memorable dreams (although we may not remember all of our dreams when we awake). We spend about 75% of our total sleep time in non-REM sleep, and the remaining 25% in REM sleep. Some sleep scientists believe that non-REM sleep is meant for body repair, whereas REM sleep is consolidating and organizing memories, because the brain is very active during this period and it uses as much oxygen as when an individual is awake.

Non-REM sleep may be further subdivided into four stages. The EEG has helped scientists detect these four stages. We cycle through these non-REM stages and REM sleep multiple times throughout a normal-length sleep cycle, as shown in **figure 13.29**. The different stages of non-REM sleep differ in the types of brain waves present (e.g., alpha, beta, theta, and delta) and the ease at which one may be awakened. After about 90 minutes of non-REM sleep, the first incidence of REM sleep occurs and typically lasts about 10 minutes. The body then cycles back into non-REM sleep and then a longer period of REM sleep.

The amount of sleep a person needs varies based on age and health. Infants typically need up to 17 to 18 hours of sleep a day, and this number drops as we get older. Teens typically need between 8.5 and 9.5 hours of sleep a night, whereas the average adult needs about 7 to 8 hours of sleep. Lack of sleep has been associated with depression, impaired memory, and decreased immune function.

The term **insomnia** refers to the difficulty in falling asleep and staying asleep. Insomnia becomes more prevalent as we age, and certain medications may interfere with sleep (including how frequently we experience REM sleep) as well. **Sleep apnea** is where a person experiences repeated breathing interruptions during sleep. These breathing interruptions cause the individual to wake repeatedly throughout the night, so the individual is at risk to a variety of ailments associated with lack of sleep. Sleep apnea may be treated with a CPAP (continuous positive airway pressure) machine, where air is pumped through a mask that the patient wears during sleep so as to keep the airways open and allow the individual to sleep uninterrupted (see Clinical View 23.12: “Apnea”).

**WHAT DID YOU LEARN?**

44. What are the main differences between non-REM and REM sleep? During what percentage of our sleep cycle are we in each type of sleep?

**LEARNING OBJECTIVES**

46. Identify the brain areas in which cognition occurs.

47. Explain how lesions to different regions of the cortex affect cognition.

Mental processes such as awareness, knowledge, memory, perception, and thinking are collectively called **cognition** (kog-ni’shun). The association areas of the cerebrum (see section 13.3c), which form about 70% of the nervous tissue in the brain, are responsible for both cognition and the processing and integration of information between sensory input and motor output areas.
Various studies of individuals suffering from brain lesions (caused by cancer, infection, stroke, and trauma) have provided insight into the functions of these areas of the brain. For example, the prefrontal cortex (frontal association area) integrates information from the sensory, motor, and association areas to enable the individual to think, plan, and execute appropriate behavior. Thus, an individual with a frontal lobe lesion exhibits personality abnormalities.

If an individual loses the ability to detect and identify stimuli (term loss of awareness) on one side of the body, or on the limbs on that side, the primary somatosensory area in the hemisphere opposite the affected side of the body has been damaged.

An individual who has agnosia (ag-nōˈzē-ə; a = without, gnosis = knowledge) displays an inability either to recognize or to understand the meaning of sounds or words. Specific symptoms of agnosia vary, depending upon the location of the lesion within the cerebrum. 

A Model of Information Processing

Information processing begins when a stimulus is perceived by the sensory organs. The stimulus is then processed by the brain, which involves encoding, storage, and retrieval. Encoding requires the proper functioning of memory consolidation. storage, and forgetting (the elimination of trivial or nonuseful information).

Neuroscientists classify memory in various ways. For example, sensory memory occurs when we form important associations based on sensory input from the environment, such as the sounds coming from a crowded cafeteria, the smell from the food line, and the bright lights from the room. Sensory memory typically lasts for milliseconds to 1 second at most.

Short-term memory (STM) is generally characterized by limited capacity (approximately seven small segments of information) and brief duration (typically lasting less than 1 minute unless the information is rehearsed). Suppose that, in a Friday morning anatomy and physiology lecture, your instructor lists the general functions of the cerebral lobes on the board. Unless you study this information over the weekend, you will probably not recall it by Monday’s lecture.

Some short-term memory, if adequately repeated and assessed, may be converted to long-term memory. Once information is placed into long-term memory (LTM), it may exist for limitless periods of time. So, for example, if over the weekend you practice retrieving the information from lecture (by rewriting your notes from memory or quizzing yourself) and/or work with a study partner to explain a lecture concept, you likely will store the information as LTM within the cerebral lobes. Not only will these practices help you to be well prepared for your next examination, but you may even remember this information for years to come. (However, information in LTM needs to be retrieved occasionally or it can be “lost,” and our ability to store and retrieve information declines with aging.)

It appears that our brain must organize complex information in short-term memory prior to storing it in long-term memory (figure 13.30). Conversion from STM to LTM is called encoding, or memory consolidation. Encoding requires the proper functioning of two components of the limbic system: the hippocampus and the amygdaloid body (see section 13.7a). The hippocampus is required for the formation of STM, whereas LTM is stored primarily in the corresponding association areas of the cerebral cortex. For example, voluntary motor activity memory is stored in the premotor cortex, whereas memory of sounds is stored in the auditory association area.

Because STM and LTM involve different anatomic structures, loss of the ability to form STM does not affect the maintenance or accessibility of LTM.

13.8e Memory

LEARNING OBJECTIVES

48. Compare and contrast short-term and long-term memory, and describe the parts of the brain involved with each.

49. Name the two regions of the limbic system involved in conversion of short-term memory to long-term memory.

Memory is a versatile element of human cognition involving different lengths of time and different storage capacities. Storing and retrieving information requires higher-order brain functions and depends upon complex interactions among different brain regions. On a broader scale, in addition to memory, information management depends upon complex interactions among different brain regions. For example, the prefrontal cortex and the limbic system in expression of emotions.

Emotional expression varies widely. For example, an automobile accident may cause those involved and some observers to cry, scream, or totally lose “emotional control,” whereas the responding emergency personnel generally appear stoic, wearing masked expressions as they go about their professional duties.

Expression of our emotions is interpreted by our limbic system but ultimately is controlled by the prefrontal cortex. Irrespective of how we feel, this cortical region decides the appropriate way to show our feelings. Researchers have identified the emotional control centers of the brain by using traditional techniques as well as by examining the behavior of experimental animals and individuals with brain lesions. Although interpreting the results is often difficult because of the complexities of both the brain and our behavior, researchers have learned that many important aspects of emotion also depend upon an intact, functional amygdaloid body and hippocampus (components of the limbic system). If specific regions of either of these structures are damaged or artificially stimulated, we exhibit either deadened or...
Alzheimer disease (AD) has become the leading cause of dementia in the developed world. (Dementia refers to a general loss of cognitive abilities, including memory, language, and decision-making skills.)

What are the classic symptoms of AD?
AD typically becomes clinically apparent in one’s 70s or later; early onset Alzheimer disease is the diagnosis when an individual develops symptoms before the age of 65. An AD diagnosis is often delayed because of confusion with other forms of cognitive impairment. Symptoms include slow, progressive loss of higher intellectual functions and changes in mood and behavior. AD gradually causes language deterioration, impaired visuospatial skills, indifferent attitude, and poor judgment, while leaving motor function intact. Patients become confused and restless, often asking the same question repeatedly. AD progresses relentlessly over months and years, and thus has come to be known as “the long goodbye.” Eventually, it robs its victims of their memory, their former personality, and even the capacity to speak.

What causes AD?
The underlying cause of AD remains a mystery, although both genetics and environment seem to play a role. Postmortem examinations of the brains of AD patients show marked and generalized cerebral atrophy. Microscopic examinations of brain tissue reveal a profound decrease in the number of cerebral cortical neurons, and a proliferation of two abnormal types of structures: amyloid plaques and neurofibrillary tangles. Amyloid plaques are insoluble deposits of a protein termed beta amyloid as well as portions of neurons and microglial cells (see section 12.4b). The neurofibrillary tangles are formed from a protein called tau that is hyperphosphorylated (contains excess amounts of phosphate). Researchers are not sure if the plaques and tangles are the cause of AD, or merely a by-product of the disease’s manifestation. Biochemical alterations also occur, most significantly a decreased level of the neurotransmitter acetylcholine in the cerebrum.

Is there a cure or test for AD?
There is no cure for AD, although some medications help alleviate the symptoms and seem to slow the progress of the disease. In the meantime, researchers are trying to develop diagnostic tests that can better predict who may be at risk for AD. Until recently, the only way to definitively diagnose AD was at autopsy, when the brain could be macroscopically and microscopically examined. Now, positron emission tomography (PET) scans appear to be able to identify the early brain changes seen with AD.

Recent research has suggested that difficulty or loss in identifying common smells (e.g., lemon, cinnamon) is linked with an increased risk in developing AD. In fact, this loss of smell may be one of the first signs of developing the disease, presumably because the brain regions involved with smell are among the first regions to develop the amyloid plaques and neurofibrillary tangles of AD.
exaggerated expressions of aggression, affection, fear, love, pain, pleasure, or sexuality, as well as anomalies in learning and memory.

WHAT DID YOU LEARN?

What portions of the brain and limbic system are involved with modulation of emotion?

INTEGRATE

CLINICAL VIEW 13.15

Dyslexia

Dyslexia (dis-lek’sē-ă; dys = bad; lexēs = word) is an inherited learning disability characterized by problems with single-word decoding. It often runs in families. Affected individuals not only have trouble reading but may also have problems writing and spelling accurately. These individuals may be able to recognize letters normally, but they demonstrate a level of reading competence far below that expected for their level of intelligence. Their writing may be disorganized and uneven, with the letters of words in incorrect order or even completely reversed. Some individuals appear to outgrow this condition, or at least develop improved reading ability over time. This improvement may reflect neural maturation or retraining of parts of the brain to better decode words and symbols. Some researchers have postulated that dyslexia is a form of disconnect syndrome, in which transfer of information between the cerebral hemispheres through the corpus callosum is impaired.

13.8g Language

LEARNING OBJECTIVE

51. List the cerebral centers involved in written and spoken language, and describe how these centers work together.

The higher-order processes involved in language include reading, writing, speaking, and understanding words. Recall that two important cortical areas involved in speech integration are the Wernicke area and the motor speech area (Broca area) (see section 13.3c). The Wernicke area is involved in interpreting what we read or hear, whereas the motor speech area receives nerve signals originating from the Wernicke area and then helps regulate the motor activities needed for us to speak. Thus, the Wernicke area is central to our ability to recognize written and spoken language. Immediately posterior to the Wernicke area is the angular gyrus, a region that processes the words we read into a form that we can speak (figure 13.31). First, the Wernicke area sends a speech plan to the motor speech area, which initiates a specific patterned motor program that is transmitted to the primary motor cortex. Next, the primary motor cortex signals other motor neurons, which then stimulate the muscles of the cheeks, larynx, lips, and tongue to produce speech.

The Wernicke area is in the categorical hemisphere in most people (see section 13.3e). In the representational hemisphere, a cortical region opposite the Wernicke area recognizes the emotional content of speech. A lesion in this area of the cerebrum can make a person unable to understand emotional nuances, such as bitterness or happiness, in spoken words. A lesion in the cortical region of the representational hemisphere opposite the motor speech area results in aprosodia, which causes dull, emotionless speech.

Several speech disorders affect the interpretation, processing, and execution of language (including sign language). For example, apraxia (a-prak’sē-ă; pratto = to do) of speech is a motor function disorder. Individuals are consciously aware of what they want to say but are unable to coordinate and execute the motor commands needed to produce the speech. As a result, these individuals may have difficulty in producing recognizable sounds and sequencing them properly for normal speech. In contrast, an individual with aphasia (a-fā’sē; speechlessness) has difficulty understanding speech or writing, or is unable to produce comprehensible speech. Aphasic individuals may have consistent difficulties

Figure 13.31 Functional Areas Within the Cerebral Cortex. (a) The left cerebral hemisphere in most people houses the Wernicke area, the motor speech area, and the prefrontal cortex. (b) A PET scan shows the areas of the brain that are most active during speech. (b1, 2) ©WDCN/Univ. College London/Science Source; (b3) ©National Cancer Institute/Science Source
There are 12 pairs of cranial nerves, which are designated with both a number and a name. They are numbered with Roman numerals according to their positions originating on the brain, beginning with the most anteriorly placed nerve. Note that the number is sometimes preceded by the prefix **CN** (figure 13.32).

The name of each nerve generally has some relation to its function. The 12 pairs of cranial nerves are the olfactory (CN I), optic (CN II), oculomotor (CN III), trochlear (CN IV), trigeminal (CN V), abducens (CN VI), facial (CN VII), vestibulocochlear (CN VIII), glossopharyngeal (CN IX), vagus (CN X), accessory (CN XI), and hypoglossal (CN XII).

Table 13.4 summarizes the main motor and sensory functions of each cranial nerve. For easier reference, each main function of a nerve is color-coded. Blue represents a sensory function,
### Table 13.4

<table>
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<tr>
<th>Cranial Nerve</th>
<th>Sensory Function</th>
<th>Somatic Motor Function</th>
<th>Parasympathetic Motor (Autonomic) Function¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (olfactory)</td>
<td>Olfaction (smell)</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>II (optic)</td>
<td>Vision</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>III (oculomotor)</td>
<td>None ²</td>
<td>Four extrinsic eye muscles (medial rectus, superior rectus, inferior rectus, inferior oblique), levator palpebrae superioris muscle (elevates eyelid)</td>
<td>Innervates sphincter pupillae muscle in eye to make pupil constrict; contracts ciliary muscles to make lens of eye more rounded (as needed for near vision)</td>
</tr>
<tr>
<td>IV (trochlear)</td>
<td>None ²</td>
<td>Superior oblique extrinsic eye muscle</td>
<td>None</td>
</tr>
<tr>
<td>V (trigeminal)</td>
<td>General sensory from anterior scalp, nasal cavity, nasopharynx, entire face, most of oral cavity, teeth, anterior two-thirds of tongue; part of auricle of ear; meninges</td>
<td>Muscles of mastication, mylohyoid, digastric (anterior belly), tensor tympani, tensor veli palatini</td>
<td>None</td>
</tr>
<tr>
<td>VI (abducens)</td>
<td>None ²</td>
<td>Lateral rectus extrinsic eye muscle</td>
<td>None</td>
</tr>
<tr>
<td>VII (facial)</td>
<td>Taste from anterior two-thirds of tongue</td>
<td>Muscles of facial expression, digastric (posterior belly), stylohyoid, stapedius</td>
<td>Increases secretion from lacrimal gland of eye, submandibular and sublingual salivary glands</td>
</tr>
<tr>
<td>VIII (vestibulocochlear)</td>
<td>Hearing (cochlear branch); equilibrium (vestibular branch)</td>
<td>None ³</td>
<td>None</td>
</tr>
<tr>
<td>IX (glossopharyngeal)</td>
<td>General sensory and taste from posterior one-third of tongue, general sensory from part of pharynx, visceral sensory from carotid bodies</td>
<td>One pharyngeal muscle (stylopharyngeus)</td>
<td>Increases secretion from parotid salivary gland</td>
</tr>
<tr>
<td>X (vagus)</td>
<td>Visceral sensory information from heart, lungs, most abdominal organs General sensory information from external acoustic meatus, tympanic membrane, part of pharynx, laryngopharynx, and larynx</td>
<td>Most pharyngeal muscles; all laryngeal muscles ³</td>
<td>Innervates smooth muscle and glands of heart, lungs, larynx, trachea, most abdominal organs</td>
</tr>
<tr>
<td>XI (accessory)</td>
<td>None ²</td>
<td>Trapezius muscle, sternocleidomastoid muscle</td>
<td>None</td>
</tr>
<tr>
<td>XII (hypoglossal)</td>
<td>None ²</td>
<td>Intrinsic and extrinsic tongue muscles</td>
<td>None</td>
</tr>
</tbody>
</table>

1. The autonomic nervous system contains a parasympathetic division and sympathetic division. Some cranial nerves contain parasympathetic axons and are listed in this table. Detailed information about these divisions is found in section 15.2.
2. These nerves do contain some tiny proprioceptive sensory axons from the muscles, but in general, these nerves tend to be described as motor only.
3. A few motor axons travel with this nerve to the inner ear, but they are not considered a significant component of the nerve.

---

**INTEGRATE**

**LEARNING STRATEGY**

Developing a code or phrase called a mnemonic (nē-mon′ık) may help you remember the cranial nerves. Here is a sample mnemonic for the cranial nerves:

- Oh (olfactory) final (facial)
- once (optic) very (vestibulocochlear)
- one (oculomotor) good (glossopharyngeal)
- takes (trochlear) vacations (vagus)
- the (trigeminal) are (accessory)
- anatomy (abducens) heavenly! (hypoglossal)

pink stands for a somatic motor function (see section 12.1b), and orange denotes a parasympathetic motor function. Table 13.5 lists the individual cranial nerves and discusses their functions, origins, and pathways. The color-coding in table 13.4 carries over to table 13.5, so you can easily determine whether a cranial nerve has sensory function, motor function, or both.

**WHAT DID YOU LEARN?**

Which cranial nerves have sensory functions only?
### Table 13.5 Cranial Nerves

#### CN I Olfactory Nerve (ol-fak′tô-rē; olfacio = to smell)

<table>
<thead>
<tr>
<th>Description</th>
<th>Special sensory nerve that conducts olfactory (smell) sensation from the nose to the brain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory function</td>
<td>Olfaction (smell)</td>
</tr>
<tr>
<td>Origin</td>
<td>Receptors (bipolar neurons) in olfactory epithelium of nasal cavity</td>
</tr>
<tr>
<td>Pathway</td>
<td>Travels through the cribriform foramina of ethmoid bone and synapses in the olfactory bulbs, which extend to various locations within the brain, including the primary olfactory cortex of the temporal lobe</td>
</tr>
<tr>
<td>Conditions caused by nerve damage</td>
<td>Anosmia (partial or total loss of smell)</td>
</tr>
<tr>
<td>How to test for nerve damage</td>
<td>Test smell (have patient close eyes, close one nostril, and inhale an odor with the other nostril).</td>
</tr>
</tbody>
</table>

#### CN II Optic Nerve (op′tik; ops = eye)

<table>
<thead>
<tr>
<th>Description</th>
<th>Special sensory nerve that conducts visual information from the retina of the eye to the brain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory function</td>
<td>Vision</td>
</tr>
<tr>
<td>Origin</td>
<td>Retina of the eye</td>
</tr>
<tr>
<td>Pathway</td>
<td>Enters cranium via optic canal of sphenoid bone; left and right optic nerves unite at optic chiasm; optic tract travels to lateral geniculate nucleus of thalamus; nerve fibers project to the primary visual cortex of the occipital lobe</td>
</tr>
<tr>
<td>Conditions caused by nerve damage</td>
<td>Anopsia (visual defects)</td>
</tr>
<tr>
<td>How to test for nerve damage</td>
<td>Test vision (cover one eye and have patient view a visual acuity chart with the other eye).</td>
</tr>
</tbody>
</table>
### Table 13.5 Cranial Nerves (continued)

#### CN III OCULOMOTOR NERVE (ok′lō-mō′tŏr; oculus = eye, motorius = moving)

<table>
<thead>
<tr>
<th>Description</th>
<th>Motor nerve that innervates four of the six extrinsic eye muscles, an upper eyelid muscle, and intrinsic eye muscles (smooth muscle within the eye)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatic motor function</td>
<td>Contracts four extrinsic eye muscles (superior rectus, inferior rectus, medial rectus, inferior oblique) to move eye and levator palpebrae superioris muscle to elevate eyelid</td>
</tr>
<tr>
<td>Parasympathetic motor function</td>
<td>Contracts sphincter pupillae muscle of iris to make pupil constrict and contracts smooth muscle of ciliary body to make lens of eye more spherical (as needed for near vision)</td>
</tr>
<tr>
<td>Origin</td>
<td>Oculomotor and Edinger Westphal nuclei within the midbrain</td>
</tr>
<tr>
<td>Pathway</td>
<td>Leaves cranium via superior orbital fissure and travels to eye and eyelid (parasympathetic axons travel to ciliary ganglion, and postganglionic parasympathetic axons then travel to iris and ciliary muscles)</td>
</tr>
<tr>
<td>Conditions caused by nerve damage</td>
<td>Ptosis (upper eyelid droop); paralysis of most eye muscles, leading to strabismus (eyes not in parallel/deviated improperly), diplopia (double vision), focusing difficulty, dilated pupil (mydriasis)</td>
</tr>
<tr>
<td>How to test for nerve damage</td>
<td>Determine if the upper eyelid droops, examine if pupil constricts in response to light, examine eye movement (have patient follow a moving object with eyes).</td>
</tr>
</tbody>
</table>

#### CN IV TROCHLEAR NERVE (trŏk′lē-ar; trochlea = a pulley)

<table>
<thead>
<tr>
<th>Description</th>
<th>Motor nerve that innervates one extrinsic eye muscle (superior oblique) that loops through a pulley-shaped ligament called a trochlea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatic motor function</td>
<td>Contracts one extrinsic eye muscle (superior oblique) to move eye inferiorly and laterally</td>
</tr>
<tr>
<td>Origin</td>
<td>Trochlear nucleus within the midbrain</td>
</tr>
<tr>
<td>Pathway</td>
<td>Leaves cranium via superior orbital fissure and travels to superior oblique muscle</td>
</tr>
<tr>
<td>Conditions caused by nerve damage</td>
<td>Paralysis of superior oblique, leading to strabismus (eyes not in parallel/deviated improperly), diplopia (double vision)</td>
</tr>
<tr>
<td>How to test for nerve damage</td>
<td>Examine eye movement (have patient follow a moving object with eyes).</td>
</tr>
</tbody>
</table>

(continued on next page)
**Table 13.5** Cranial Nerves (continued)

**CN V TRIGEMINAL NERVE** (trɪ-jemˈə-nəl; _trigeminus_ = threefold)

<table>
<thead>
<tr>
<th>Description</th>
<th>Mixed nerve that consists of three divisions: ophthalmic (V₁), maxillary (V₂), and mandibular (V₃); receives sensory nerve signals from face, oral cavity, nasal cavity, meninges, and anterior scalp and innervates muscles of mastication</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sensory function</strong></td>
<td>Sensory stimuli for this nerve are touch, temperature, and pain.</td>
</tr>
<tr>
<td>V₁: Conducts sensory nerve signals from cornea, nose, forehead, anterior scalp, meninges</td>
<td>V₂: Conducts sensory nerve signals from nasal mucosa, palate, gums, cheek, meninges</td>
</tr>
<tr>
<td>V₃: Conducts sensory nerve signals from anterior two-thirds of tongue, meninges, skin of chin, lower jaw, lower teeth; one-third from sensory axons of auricle of ear</td>
<td></td>
</tr>
<tr>
<td><strong>Somatic motor function</strong></td>
<td>Innervates muscles of mastication (temporalis, masseter, lateral and medial pterygoids), mylohyoid, anterior belly of digastric, tensor tympani muscle, and tensor veli palatini</td>
</tr>
<tr>
<td><strong>Origin</strong></td>
<td>Nucli in the pons</td>
</tr>
<tr>
<td><strong>Pathway</strong></td>
<td>V₁: Sensory axons enter cranium via superior orbital fissure and travel to trigeminal ganglion before entering pons.</td>
</tr>
<tr>
<td>V₂: Sensory axons enter cranium via foramen rotundum and travel to trigeminal ganglion before entering pons.</td>
<td>V₃: Sensory axons travel through foramen ovale to trigeminal ganglion before entering pons. Motor axons leave pons and exit cranium via foramen ovale to supply muscles.</td>
</tr>
<tr>
<td><strong>Conditions caused by nerve damage</strong></td>
<td>Trigeminal neuralgia (tic douloureux) is caused by inflammation of the sensory components of the trigeminal nerve and results in intense, pulsating pain lasting from minutes to several hours.</td>
</tr>
<tr>
<td><strong>How to test for nerve damage</strong></td>
<td>Have patient close mouth against resistance; also have patient close eyes and then determine if an object (such as a feather) moved along the face can be felt.</td>
</tr>
</tbody>
</table>
### CN VI ABDUCENS NERVE (ab-du′senz; to move away from)

**Description**
Motor nerve that innervates one extrinsic eye muscle (lateral rectus) to move the eye

**Somatic motor function**
Contracts lateral rectus for eye abduction (moving eye laterally)

**Origin**
Pontine (abducens) nucleus in the pons

**Pathway**
Leaves cranium through superior orbital fissure and travels to lateral rectus muscle

**Conditions caused by nerve damage**
Paralysis of lateral rectus limits lateral movement of eye, diplopia (double vision)

**How to test for nerve damage**
Examine eye movement (have patient follow a moving object with eyes) and determine if the eye is able to be abducted.

### CN VII FACIAL NERVE (fa′shəl)

**Description**
Mixed nerve that conduct taste sensations from anterior two-thirds of tongue; relays motor output to muscles of facial expression, lacrimal (tear) gland, and salivary glands inferior to the tongue (submandibular salivary gland and sublingual salivary gland)

**Sensory function**
Conducts taste sensations from anterior two-thirds of tongue

**Somatic motor function**
The five major motor branches (temporal, zygomatic, buccal, mandibular, and cervical) innervate the muscles of facial expression, the posterior belly of the digastric muscle, and the stylohyoid and stapedius muscles.

**Parasympathetic motor function**
Increases secretions of the lacrimal gland of the eye; increases secretions of the submandibular and sublingual salivary glands

**Origin**
Nuclei in the pons

**Pathway**
Sensory axons travel from the tongue via the chorda tympani branch of the facial nerve through a tiny foramen to enter the skull, and axons synapse at the geniculate ganglion of the facial nerve. Somatic motor axons leave the pons and enter the temporal bone through the internal acoustic meatus, project through temporal bone, and emerge through the stylomastoid foramen to innervate the muscles of facial expression. Parasympathetic motor axons leave the pons, enter the internal acoustic meatus, leave with either the greater petrosal nerve or chorda tympani nerve, and travel to an autonomic ganglion before innervating their respective glands.

**Conditions caused by nerve damage**
Decreased tearing (dry eye) and decreased salivation (dry mouth); loss of taste sensation to anterior two-thirds of tongue; nerve paralysis (also known as Bell palsy; see Clinical View 11.2: “Idiopathic Facial Nerve Paralysis”) characterized by paralyzed facial muscles, lack of orbicularis oculi contraction, sagging at corner of mouth

**How to test for nerve damage**
Have patient smile, blink, and squint—inability to move facial muscles on one side indicates nerve damage.
### CN VIII VESTIBULOCOCHLEAR NERVE

**Description**: Sensory nerve with two branches that conducts equilibrium and auditory (hearing) sensations from inner ear to brain.

**Sensory function**: Vestibular branch conducts nerve signals for equilibrium, while cochlear branch conducts nerve signals for hearing.

**Origin**
- **Vestibular branch**: Hair cells in the vestibule of the inner ear.
- **Cochlear branch**: Cochlea of the inner ear.

**Pathway**: Sensory cell bodies of the vestibular branch are located in the vestibular ganglion, whereas sensory cell bodies of the cochlear branch are located in the spiral ganglion near the cochlea. The vestibular and cochlear branches merge, and together enter cranial cavity through internal acoustic meatus and travel to junction of the pons and the medulla oblongata.

**Conditions caused by nerve damage**: Lesions in vestibular branch produce loss of balance, nausea, vomiting, and dizziness; lesions in cochlear branch result in deafness (loss of hearing).

**How to test for nerve damage**: Test hearing.

### CN IX GLOSSOPHARYNGEAL NERVE

**Description**: Mixed nerve that receives taste and touch sensations from posterior one-third of the tongue; innervates one pharynx muscle and the parotid salivary gland.

**Sensory function**: General sensation and taste from the posterior one-third of tongue; general sensation from most of pharynx; relays sensory information from the carotid arteries—from both chemoreceptors (which monitor blood levels of CO₂, H⁺, and O₂) and baroreceptors (which monitor blood pressure).

**Somatic motor function**: Contracts a pharynx muscle (stylopharyngeus) for swallowing.

**Parasympathetic motor function**: Increases secretion of parotid salivary gland.

**Origin**: Sensory axons originate on taste buds and mucosa of posterior one-third of tongue, as well as the carotid bodies. Motor axons originate in nuclei in the medulla oblongata.

**Pathway**: Sensory axons travel from posterior one-third of tongue and carotid bodies along nerve through the inferior or superior ganglion into the jugular foramen, and travel to pons. Somatic motor axons leave cranium via jugular foramen and travel to stylopharyngeus. Parasympathetic motor axons travel to otic ganglion and then to parotid gland.

**Conditions caused by nerve damage**: Reduced salivary secretion (dry mouth); loss of taste sensations to posterior one-third of tongue.

**How to test for nerve damage**: Have patient open mouth and say “ahhh”—the soft palate should elevate and the uvula should remain in the midline under normal conditions.
### Table 13.5 Cranial Nerves (continued)

**CN X VAGUS NERVE (vā’gūs; wandering)**

<table>
<thead>
<tr>
<th>Description</th>
<th>Mixed nerve that innervates structures in the head and neck and in the thoracic and abdominal cavities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory function</td>
<td>Visceral sensory information from heart, lungs, and most abdominal organs; general sensory information from external acoustic meatus, tympanic membrane (eardrum), laryngopharynx (inferior part of throat), and larynx (voice box)</td>
</tr>
<tr>
<td>Somatic motor function</td>
<td>Controls most pharynx muscles (for swallowing) and all larynx muscles (for production of speech)</td>
</tr>
<tr>
<td>Parasympathetic motor function</td>
<td>Innervates smooth muscle and glands of thoracic and most abdominal organs and cardiac muscle of the heart</td>
</tr>
<tr>
<td>Origin</td>
<td>Motor nuclei in medulla oblongata</td>
</tr>
<tr>
<td>Pathway</td>
<td>Leaves cranium via jugular foramen before traveling and branching extensively in neck, thorax, and abdomen; sensory neuron cell bodies are located in the superior and inferior ganglia associated with the nerve</td>
</tr>
<tr>
<td>Conditions caused by nerve damage</td>
<td>Paralysis leads to a variety of larynx problems, including hoarseness, monotone voice, or complete loss of voice. Other lesions may cause difficulty in swallowing or impaired gastrointestinal tract motility.</td>
</tr>
<tr>
<td>How to test for nerve damage</td>
<td>Ask patient if he has difficulties in swallowing. Determine if voice is hoarse or monotone. Have patient open mouth and say “ahhh”—the soft palate should elevate and the uvula should remain in the midline under normal conditions.</td>
</tr>
</tbody>
</table>

(continued on next page)
Table 13.5 Cranial Nerves (continued)

### CN XI ACCESSORY NERVE (ak-ses′ō-rē)

**Description**
Motor nerve that innervates trapezius and sternocleidomastoid muscles, also assists CN X to innervate pharynx muscles; formerly called the spinal accessory nerve.

**Somatic motor function**
Cranial root: Travels with CN X to assist in innervating the pharynx muscles
Spinal root: Contracts trapezius and sternocleidomastoid muscles.

**Origin**
Cranial root: Nucleus in medulla oblongata
Spinal root: Nucleus in spinal cord.

**Pathway**
Spinal root travels superiorly to enter skull through foramen magnum; there, cranial and spinal roots merge and leave the skull via jugular foramen. Once outside the skull, cranial root splits to travel with CN X (vagus) to innervate pharynx muscles, and spinal root travels to sternocleidomastoid and trapezius.

**Conditions caused by nerve damage**
Paralysis of trapezius and sternocleidomastoid.

**How to test for nerve damage**
Have patient elevate or shrug shoulders (tests trapezius function) or have patient turn head to opposite side (tests sternocleidomastoid function).

### CN XII HYPOGLOSSAL NERVE (hi-pō-glos′āl; hypo = below, glossus = tongue)

**Description**
Motor nerve that innervates both intrinsic and extrinsic tongue muscles.

**Somatic motor function**
Contracts intrinsic and extrinsic tongue muscles to move tongue.

**Origin**
Hypoglossal nucleus in medulla oblongata.

**Pathway**
Leaves cranium via hypoglossal canal; travels inferior to mandible and to inferior surface of tongue.

**Conditions caused by nerve damage**
Swallowing and speech difficulties due to impaired tongue movement.

**How to test for nerve damage**
Have patient protrude (stick out) tongue: If a single hypoglossal nerve (either left or right) is paralyzed, a protruded (stuck-out) tongue deviates to the side of the damaged nerve.
CHAPTER SUMMARY

13.1 Brain Organization and Development

13.1a Overview of Brain Anatomy
- The brain has two cerebral hemispheres that exhibit folds called gyri with shallow sulci and deep fissures in between.

13.1b Development of Brain Divisions
- Three primary vesicles (prosencephalon, mesencephalon, and rhombencephalon) form from the neural tube by the late fourth week of development.
- Five secondary vesicles (telencephalon, diencephalon, mesencephalon, metencephalon, and myelencephalon) form from the primary vesicles by the fifth week of development.

13.1c Gray Matter and White Matter Distribution
- Gray matter is composed primarily of dendrites and cell bodies of neurons and functions in processing and integrating information, whereas white matter is composed primarily of bundles of myelinated axons and functions in relaying nerve signals to and from gray matter.

13.2 Protection and Support of the Brain
- The brain is protected and isolated by the cranium, cranial meninges, cerebrospinal fluid, and a blood-brain barrier.

13.2a Cranial Meninges
- The cranial meninges are the pia mater, arachnoid mater, and dura mater.
- The cranial dural septa are folds of dura mater that project between the major parts of the brain and stabilize the brain’s position.

13.2b Brain Ventricles
- Fluid-filled spaces in the brain are the paired lateral ventricles, the third ventricle, the cerebral aqueduct, and the fourth ventricle.

13.2c Cerebrospinal Fluid
- Cerebrospinal fluid (CSF) is a clear fluid that provides buoyancy, protection, and a stable environment for the brain and spinal cord.
- The choroid plexus (formed from ependymal cells and capillaries) produces CSF in the ventricles.
- CSF leaves the ventricles and enters the subarachnoid space, where it circulates around the brain and spinal cord. Excess CSF returns to the venous circulation through the arachnoid villi.

13.2d Blood-Brain Barrier
- The blood-brain barrier regulates movement of materials between the blood and the interstitial fluid of the brain.

13.3 Cerebrum
- The cerebrum is the center of our sensory perception, thought, memory, judgment, and voluntary motor actions.

13.3a Cerebral Hemispheres
- The left and right cerebral hemispheres are separated by a longitudinal fissure.

13.3b Lobes of the Cerebrum
- Each hemisphere contains five lobes: four superficial lobes (frontal, parietal, temporal, occipital lobes) and the insula, which is not visible from the surface.

13.3c Functional Areas of the Cerebrum
- The primary motor cortex in the frontal lobe directs voluntary movements.
- The primary somatosensory cortex in the parietal lobe collects somatic sensory information from skin and proprioceptors.
- Other primary cortices and association areas are housed in each of the five lobes.

13.3d Central White Matter
- The central white matter contains three major groups of axons: association tracts, commissural tracts, and projection tracts.

13.3e Cerebral Laterization
- The left hemisphere is the categorical hemisphere in most individuals, and the right is the representational hemisphere.

13.3f Cerebral Nuclei
- The cerebral nuclei are masses of gray matter located within the cerebrum.

13.4 Diencephalon
- The diencephalon is composed of the epithalamus, thalamus, and hypothalamus.

13.4a Epithalamus
- The epithalamus forms part of the posterior roof of the diencephalon; it contains the pineal gland (which secretes melatonin) and habenular nuclei (help relay signals from the limbic system to the midbrain).

13.4b Thalamus
- The thalamus is the main relay point for integrating, assimilating, and amplifying sensory signals sent to the cerebrum.

13.4c Hypothalamus
- The hypothalamus oversees the endocrine and autonomic nervous systems and houses many control and integrative centers.

(continued on next page)
### 13.5 Brainstem
- The brainstem is composed of the midbrain, pons, and medulla oblongata.

#### 13.5a Midbrain
- The midbrain contains cerebral peduncles, medial lemniscus, substantia nigra, tegmentum, tectal plate, and nuclei for two cranial nerves.

#### 13.5b Pons
- The pons contains axon tracts, the pontine respiratory center, and nuclei of four cranial nerves.

#### 13.5c Medulla Oblongata
- The medulla oblongata connects the brain to the spinal cord. It contains a cardiovascular center, a medullary respiratory center, sensory processing centers, and nuclei for four cranial nerves.

### 13.6 Cerebellum

#### 13.6a Structural Components of the Cerebellum
- The cerebellum is composed of left and right cerebellar hemispheres, with the vermis in between.
- Cerebellar peduncles are thick axon tracts that connect the cerebellum to different parts of the brainstem.

#### 13.6b Functions of the Cerebellum
- The cerebellum helps maintain posture and balance and fine-tunes skeletal muscle contractions, leading to smooth, coordinated movement.

### 13.7 Functional Brain Systems

#### 13.7a Limbic System
- The limbic system (i.e., the emotional brain) includes a group of structures that surround the corpus callosum and thalamus. The limbic system functions in memory and emotional behavior.

#### 13.7b Reticular Formation
- The reticular formation is gray matter that extends through the brainstem. It participates in cyclic activities such as arousing the cortex to consciousness and controlling the sleep-wake cycle.

### 13.8 Integrative Functions and Higher-Order Brain Functions

#### 13.8a Development of Higher-Order Brain Functions
- Higher-order functions mature and increase in complexity as development proceeds.

#### 13.8b Electroencephalogram
- An electroencephalogram monitors brain activity by measuring brain waves through the use of electrodes.

#### 13.8c Sleep
- Sleep is a period of rest for the brain and involves cycles of REM (rapid eye movement) and non-REM (non-rapid eye movement) activity.

#### 13.8d Cognition
- Mental processes such as awareness, knowledge, memory, perception, and thinking are collectively called cognition.

#### 13.8e Memory
- Memory is a higher-order brain function involving the storage and retrieval of information gathered through previous activities.

#### 13.8f Emotion
- Emotion is controlled by the limbic system and is regulated by the prefrontal cortex.

#### 13.8g Language
- The motor speech area initiates a specific motor program for the movements involved in speech, whereas the Wernicke area is responsible for recognition of spoken and written language.

### 13.9 Cranial Nerves
- Twelve pairs of nerves, called cranial nerves, project from the brain. Each nerve has a specific name and function and is designated by a Roman numeral.

### Challenge Yourself

**Do You Know the Basics?**

1. Which cranial nerve is responsible for innervating the intrinsic and extrinsic tongue muscles?
   - a. accessory (CN XI)
   - b. glossopharyngeal (CN IX)
   - c. trigeminal (CN V)
   - d. hypoglossal (CN XII)

2. The subdivision of the brain that does not initiate somatic motor movements, but rather coordinates and fine-tunes those movements, is the
   - a. medulla oblongata.
   - b. cerebrum.
   - c. cerebellum.
   - d. diencephalon.
3. Which of these is the least likely to affect information transfer from STM (short-term memory) to LTM (long-term memory)?
   a. emotional state
   b. repetition or rehearsal
   c. auditory association cortex
   d. cerebral nuclei

4. All of the following are functions of the hypothalamus except:
   a. controls endocrine system.
   b. regulates sleep-wake cycle.
   c. controls autonomic nervous system.
   d. initiates voluntary skeletal muscle movement.

5. All of the following statements are accurate about the choroid plexus except:
   a. it is located within the ventricles of the brain.
   b. it is composed of ependymal cells and blood capillaries.
   c. it receives and filters all sensory information.
   d. it produces and circulates cerebrospinal fluid.

6. The ______ are descending motor tracts on the anterolateral surface of the midbrain.
   a. cerebral peduncles
   b. inferior colliculi
   c. pyramids
   d. tegmenta

7. Which cerebral lobe is located immediately posterior to the central sulcus and superior to the lateral sulcus?
   a. frontal lobe
   b. parietal lobe
   c. temporal lobe
   d. occipital lobe

8. The primary motor cortex is located in which cerebral structure?
   a. precentral gyrus
   b. postcentral gyrus
   c. motor speech area
   d. prefrontal cortex

9. The ______ are the isolated, innermost gray matter areas near the base of the cerebrum, inferior to the lateral ventricles.
   a. auditory association areas
   b. cerebral nuclei
   c. substantia nigra
   d. corpus callosum axons

10. Which structure contains autonomic nuclei involved in regulating respiration?
    a. pons
    b. superior colliculi
    c. cerebellum
    d. thalamus

11. Describe (a) how and where the cerebrospinal fluid is formed, (b) its subsequent circulation, and (c) how and where it is reabsorbed into the vascular system.

12. Which specific area of the brain may be impaired if you cannot tell the difference between a smooth and a rough surface using your hands only?

13. What activities occur in the visual association area?

14. Describe the relationship between the cerebral nuclei and the cerebellum in motor activities.

15. List the functions of the hypothalamus.

16. Describe the pathway by which the pressure applied to the right hand during a handshake is transmitted and perceived in the left primary somatosensory cortex.

17. Identify the components of the limbic system.

18. During surgery to remove a tumor from the occipital lobe of the left cerebrum, a surgeon must cut into the brain to reach the tumor. List in order (starting with the covering skin) all the layers that must be cut through to reach the tumor.

19. What is the difference between apraxia of speech and aphasia?

20. Which cranial nerves are associated with some aspect of eye movements or vision?

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**Can You Apply What You’ve Learned?**

Use the following paragraph to answer questions 1 and 2.
Alex went to the dentist to get a cavity filled. The dentist used an anesthetic to numb the teeth prior to drilling.

1. Which nerve likely was anesthetized?
   a. CN IV (trochlear)
   b. CN V (trigeminal)
   c. CN VII (facial)
   d. CN IX (glossopharyngeal)

2. After the filling was inserted, Alex’s gums on the same side of the tooth remained numb for a short while. What other problems may Alex temporarily experience until the anesthetic wears off?
   a. inability to close the mouth
   b. inability to protrude the tongue
   c. dry mouth
   d. numbness of the lips

Use the following paragraph to answer questions 3 and 4.
Shannon was the pitcher on her softball team. During one game, a batter hit the ball and it ricocheted off the left side of Shannon’s head, knocking her temporarily unconscious. She eventually regained consciousness but experienced severe pain near her left temple. Within a few hours, Shannon had trouble moving her right upper limb and became lethargic. Her team captain took her to the emergency room, where the physician diagnosed her with an epidural hematoma.

3. An epidural hematoma causes an accumulation of blood to develop between what two structures?
   a. the skull and the periosteal layer of the dura mater
   b. the periosteal and meningeal layers of the dura mater
   c. the meningeal layer of the dura mater and the arachnoid mater
   d. the arachnoid mater and the pia mater

4. Shannon experienced problems with moving her right upper limb because the hematoma likely was impinging on what brain structure?
   a. left precentral gyrus
   b. left postcentral gyrus
   c. left cerebral nuclei
   d. left cerebellum
5. A 25-year-old male named Carlos went to the optometrist with the complaint of double vision (diplopia). The optometrist performed various eye tests on Carlos. Carlos was able to read an eye chart with each eye but experienced the double vision when he tried to use both eyes to focus. The optometrist noticed that when he had Carlos look laterally with each eye, Carlos’s right eye did not move as far laterally as his left could. Based on these tests, the optometrist suspected that the muscle innervated by _________ was not working properly.
   a. CN II (optic)
   b. CN III (oculomotor)
   c. CN IV (trochlear)
   d. CV VI (abducens)

Can You Synthesize What You’ve Learned?

1. Peyton felt strange when she awoke one morning. She could not hold a pen in her right hand when trying to write an entry in her diary, and her muscles were noticeably weaker on the right side of her body. Additionally, her husband noticed that she was slurring her speech, so he took her to the emergency room. What does the ER physician suspect has occurred? Where in the brain might the physician suspect that abnormal activity or perhaps a lesion is located, and why?

2. Parkinson disease is the result of decreased levels of the neurotransmitter dopamine in the brain. However, these patients cannot take dopamine in drug form because the drug cannot reach the brain. What anatomic structure prevents the drug from reaching the brain? How could this anatomic structure be beneficial to an individual under normal circumstances?

3. During a robbery at his convenience store, Dustin was shot in the right cerebral hemisphere. He survived, although some specific functions were impaired. Would Dustin have been more likely or less likely to have survived if he had been shot in the medulla oblongata? Why?
The spinal cord is about 18 inches long and is about as wide as a piece of rope. It is continuously relaying (1) sensory input from the body to the brain as well as (2) motor output from the brain to the body. Thus, it serves as the means of communication between the body and the brain. The spinal cord, which is part of the central nervous system (CNS), is associated with 31 pairs of spinal nerves. We explore the structure and function of the spinal cord and spinal nerves in this chapter.
14.1 Overview of the Spinal Cord and Spinal Nerves

Our study of the spinal cord and spinal nerves begins by providing an overview of their two primary functions. We then describe the general structure of both the spinal cord and the spinal nerves, which will help you to begin to integrate how these nervous system components operate together in performing these functions.

14.1a General Functions

LEARNING OBJECTIVE

1. Describe the two primary functions of the spinal cord and spinal nerves.

The spinal cord and its attached spinal nerves serve two important functions. Their first function is to provide an essential structural and functional link between the brain and the torso and limbs of the body. Sensory input is relayed from the torso and limbs to the brain, and motor output is relayed from the brain to the torso and limbs. These vital inputs and outputs are relayed along neuron pathways that are within the spinal cord and spinal nerves. For example, when you pick up an object, sensory information about that object (shape, weight, temperature) is relayed along one or more spinal nerves and then through the spinal cord to reach the brain, where the information is interpreted. Additionally, the movement of your limbs (e.g., while you are walking) is controlled by nerve signals initiated in the brain; these nerve signals are then relayed through the spinal cord and then the spinal nerves to the skeletal muscles of your arms and legs. Notice that both sensory input and motor output are relayed along the pathways within the spinal cord and spinal nerves.

The second important function of the spinal cord and spinal nerves is their role in spinal reflexes. These involve nervous system responses that do not require the involvement of the brain, but instead have the spinal cord as the integration center. Spinal reflexes initiate our quickest reactions to a stimulus. It is through spinal reflexes that the spinal cord exhibits some functional independence from the brain. Spinal reflexes initiate responses that do not require the involvement of the brain, but instead have the spinal cord as the integration center. They are our quickest reactions to a stimulus. It is through spinal reflexes that the spinal cord exhibits some functional independence from the brain.

WHAT DID YOU LEARN?

2. What are the general shape, diameter, and length of the spinal cord, and where is it housed?

14.1b Spinal Cord Gross Anatomy

LEARNING OBJECTIVE

2. Describe the general structure of the spinal cord and its four anatomic subdivisions.

The spinal cord is a roughly cylindrical nervous system structure that extends through the vertebral column to the inferior border of the L1 vertebra (figure 14.1). The superior end of the spinal cord is continuous with the medulla oblongata of the brain, and its inferior end tapers (narrowed) to form the conus medullaris (ko’nos med’u-lär’is; ko’nos = cone, medulla = middle). A typical adult spinal cord is approximately 20–21 inches (50–53 cm) long in length. (These dimensions are similar to a width of your finger that would have the length from your fingertip to your elbow.) Observe in figure 14.1 that the spinal cord does not extend the entire length of the vertebral column, but ends at approximately the superior border of the small of your back. This is because growth of the individual vertebrae continues longer than the growth of the spinal cord; thus, an adult spinal cord is shorter than the vertebral column.

Two longitudinal depressions extend the full length of the spinal cord. The posterior median sulcus is a narrow groove on the posterior surface, whereas the anterior median fissure is a slightly wider groove on the anterior surface (not shown in figure 14.1).

There are four continuous subdivisions (parts) of the spinal cord (from superior to inferior): (1) the cervical part, which is continuous with the medulla oblongata, (2) the thoracic part, (3) the lumbar part, and (4) the sacral part. (Some references further divide the sacral part into a sacral part and a coccygeal part.) Notice in figure 14.1 that two areas of the spinal cord are wider than other cord areas: The cervical enlargement is the wider area in the cervical part, and the lumbosacral enlargement is the wider area in the lumbar and sacral parts. These regions of the spinal cord are enlarged due to the presence of a greater number of neurons within the spinal nerves extending from these spinal cord parts to innervate the upper and lower limbs, respectively.

14.1c Spinal Nerve Identification and Gross Anatomy

LEARNING OBJECTIVES

3. Discuss the naming of the 31 pairs of spinal nerves, and provide a general description of a spinal nerve and its composition.

General Description of a Nerve

Nerves were described in detail in section 12.1c. Recall that a nerve is an organ composed of a cablelike bundle of axons, which are enclosed within successive connective tissue wrappings. These include the epineurium, which ensheaths the entire nerve; the perineurium, which encloses each fascicle (bundle) of axons; and the endoneurium, which electrically insulates each axon (see figure 12.2a, b).

Naming Spinal Nerves

The spinal cord is associated with 31 pairs of spinal nerves (figure 14.1a). Each spinal nerve is typically identified by the first letter of the spinal cord part to which it attaches, followed by a number. Thus, each side of the spinal cord contains 8 cervical nerves (called C1–C8), 12 thoracic nerves (T1–T12), 5 lumbar nerves (L1–L5), 5 sacral nerves (S1–S5), and 1 coccygeal nerve (C01). Spinal nerve names are readily distinguished from cranial nerve names (discussed in section 13.9) because cranial nerves are designated by either CN followed by a roman numeral (e.g., CN I, CN II) or a specific name (e.g., olfactory nerve, optic nerve). The nerve plexuses (which are labeled in figure 14.1a, such as the brachial plexus) are extensions of spinal nerves and are discussed in section 14.5.

Gross Anatomy of a Nerve

Each spinal nerve anchors to the spinal cord by two roots, a posterior root and an anterior root, and each of these roots is composed of multiple rootlets (figure 14.2). The posterior root houses sensory neurons (see section 12.2d) that extend from sensory receptors. These sensory neurons relay nerve signals from the sensory receptors to the spinal cord. Observe that the sensory neurons that compose the spinal nerves are unipolar neurons (see section 12.2d). The dendrites of...
these sensory neurons form the sensory receptors (see section 16.1b), and their axons extend from the dendrites to the spinal cord. It is critical to note that the cell bodies of these sensory neurons (which are positioned along the length of the axon) are located external to the spinal cord and form the posterior root ganglion (see figure 12.2c).

The anterior root contains motor neurons that extend to effectors (muscle or glands; figure 14.2). These motor neurons relay nerve signals from the spinal cord and control muscles and glands. Observe that the motor neurons that compose the spinal nerves are multipolar neurons (see section 12.2d). Both the dendrites and the cell bodies of motor neurons, unlike those of sensory neurons, are housed within the spinal cord; thus, the anterior root does not contain a ganglion along its length. Motor neuron axons exit from the spinal cord within the anterior root and extend through the spinal nerve to their terminal ends, which innervate an effector. Thus, a significant difference between anterior and posterior roots is that each anterior root lacks a ganglion along its length. This is because, as mentioned, the dendrites and cell bodies of motor neurons are within the spinal cord and the anterior root contains only the axons of these neurons.

Each spinal nerve forms where the posterior root (containing sensory neurons) and the anterior root (containing motor neurons) join. Thus, both sensory and motor neurons compose each spinal nerve, and it is classified as a mixed nerve (see section 12.1c). If you compare a spinal nerve to a cable composed of multiple wires, the “wires” within a spinal nerve are the sensory and motor axons, and each “wire” transmits signals in one direction only.
Posterior root

Cauda Equina

The spinal cord does not extend the entire length of the vertebral column, but typically ends at the inferior border of the L₁ vertebra, as described (figure 14.1). Consequently, roots of the lumbar, sacral and coccygeal spinal nerves do not extend horizontally from the spinal cord (as illustrated in figure 14.2 for the more superior spinal nerves). Instead, these spinal nerve roots extend inferiorly from the conus medullaris until the specific location where they exit the vertebral column (figure 14.1a, c). Collectively, these spinal nerve roots form a structure called the cauda equina (kaw’dă ē-kwi’nā). They are so named because they resemble a horse’s tail (cauda = tail, equus = horse). Note that the conus medullaris (at the L₁ vertebra) represents (1) the inferior end of the spinal cord and (2) the most superior portion of the spinal roots forming the cauda equina.

WHAT DID YOU LEARN?

3. What is the total number of spinal nerves, and how are they specifically identified?
4. How is the cauda equina formed? What composes the cauda equina?

14.2 Protection and Support of the Spinal Cord

LEARNING OBJECTIVES

5. Discuss the relationship of the spinal cord and spinal nerves to the vertebral column.
6. Describe the locations and functions of the spinal cord meninges, and compare and contrast the three spaces associated with the spinal cord meninges.

The spinal cord is collectively protected by a bony structure, meninges, and cerebrospinal fluid, just like the brain. The bony framework that houses the spinal cord is the vertebral column (see figure 8.1 in section 8.5a). It is formed by 26 stacked vertebrae and the intervertebral discs between vertebrae. The vertebral column physically protects the spinal cord and is flexible enough to allow for movement of the torso. All of the stacked vertebral foramina collectively form the vertebral canal, which houses both the spinal cord and the cauda equina. Note that the different parts of the spinal cord described in section 14.1b do not match up exactly with the vertebrae of the same name (figure 14.1a). For example, the lumbar part of the spinal cord is actually closer to the inferior thoracic vertebrae than to the lumbar vertebrae. This apparent discrepancy is due to the continued growth of individual vertebrae after spinal cord growth is complete.

Each spinal nerve exits the vertebral column through an intervertebral foramen (which is a lateral opening between two adjacent stacked vertebrae; figure 14.3a). Note that each of the more superior spinal nerves (i.e., cervical and thoracic) extends horizontally through its associated intervertebral foramen at the same level (as shown in figure 14.3a). The more inferior spinal nerves (i.e., lumbar, sacral and coccygeal) have roots that extend inferiorly as part of the cauda equina, and each of these spinal nerves then extends through its intervertebral foramen, which is inferior to where the roots are anchored to the spinal cord (see figure 14.1a, c).

You might wonder why there are eight cervical nerves and only seven cervical vertebra (see section 8.5c). The first seven cervical spinal nerves (C1–C7) exit the vertebral canal and extend through an intervertebral foramen that is superior to the vertebra of the same number. For example, the C2 spinal nerve exits the vertebral canal...
Figure 14.3 Spinal Meninges and Structure of the Spinal Cord. (a) A cross section of the spinal cord shows the relationship between the meningeal layers and the superficial landmarks of the spinal cord and vertebral column. (b) Anterior view shows the spinal cord and meninges.
through the intervertebral foramen between the C₁ and C₂ vertebrae. The eighth cervical spinal nerve, in comparison, exits the intervertebral foramen inferior to the C₇ vertebra. All remaining spinal nerves inferior to the C₈ nerve exit the vertebral canal and extend through an intervertebral foramen inferior to the vertebra of the same number. Thus, for example, the T₂ spinal nerve exits the vertebral canal through the intervertebral foramen inferior to the T₃ vertebra.

The spinal cord meninges (mē-nin′jes, mē-nin′jēz; sing., meninx, men′inx; membrane) are connective tissue membranes that protect and encapsulate the spinal cord within the vertebral canal. (They are continuous with the cranial meninges, described in section 13.2a.) The spinal cord meninges, layered from innermost to outermost, are: pia mater, arachnoid mater, and dura mater.

The pia mater directly adheres to the external surface of the spinal cord. It is the delicate, innermost meningeal layer, which is a meshlike membrane composed of both elastic and collagen fibers. Pia mater extensions form two structures: denticulate ligaments and the filum terminale. Denticulate (den-tik′ū-lät; dentatus = toothed) ligaments are the numerous paired, triangular extensions present along the spinal cord. These pia mater extensions suspend and anchor the spinal cord laterally to the arachnoid and dura mater (see figures 14.1b and 14.3a). The filum terminale (fī-lûm ter′ mi-nāl; terminus = end) is a thin strand of pia mater that anchors the conus medullaris to the coccyx bone. It extends within the cauda equina and can be viewed in figure 14.1a, c). Both types of pia mater extensions help stabilize the spinal cord within the vertebral canal.

The arachnoid mater lies external to the pia mater. It is partially composed of a delicate web of both collagen and elastic fibers termed the arachnoid trabeculae. Immediately deep to the arachnoid mater is the subarachnoid space. Cerebrospinal fluid (CSF) circulates within this space (both around the spinal cord and around the brain). Cerebrospinal fluid can be analyzed (e.g., for infectious agents) following its removal from the subarachnoid space by the clinical procedure called a lumbar puncture (see Clinical View 14.1: “Lumbar Puncture”).

The outermost layer of meninges is the dura mater. It is composed of dense irregular connective tissue. The dura mater associated with the spinal cord has only one layer (unlike the dura mater covering the brain, which is composed of both a periosteal layer and meningeal layer; see section 13.2a). Extensions of the dura mater ensheathe the spinal nerve roots and merge with the connective tissue layer that surrounds the spinal nerves (i.e., the epineurium; see section 12.1c). Two spaces are associated with the dura mater: the subdural space and the epidural space. The subdural space is a potential space internal to the dura mater (between the arachnoid mater and dura mater). The epidural space is a space external to the dura mater. The epidural space is a clinically significant area that houses adipose and areolar connective tissue, as well as blood vessels. Epidural anesthetics, such as may be used to lessen pain during childbirth, are introduced into this space. (See Clinical View: 29.7: “Anesthetic Procedures to Facilitate True Labor.”)

**WHAT DO YOU THINK?**

1. What are the similarities and differences among the meningeal layers and spaces that a hypodermic needle must pass through (or enter) for a lumbar puncture to remove cerebrospinal fluid from the subarachnoid space and for an epidural to deliver a drug to the epidural space?

**WHAT DID YOU LEARN?**

5. Where are the epidural, subdural, and subarachnoid spaces located? Which space contains CSF?

**INTEGRATE**

**Clinical View 14.1**

**Lumbar Puncture**

It is sometimes necessary to analyze the cerebrospinal fluid (CSF) to determine whether an infection or a disorder of the central nervous system is present. The clinical procedure for obtaining CSF is known as a lumbar puncture (commonly referred to as a spinal tap). The needle must be inserted through the skin, back muscles, and ligamentum flavum (between vertebrae). Then, the needle must pass through the epidural space, dura mater, and arachnoid mater and enter the subarachnoid space to obtain approximately 3 to 9 milliliters of CSF.

Since the adult spinal cord typically ends at the level of the L₁ vertebra, a lumbar puncture must be performed inferior to this level to ensure that the needle does not pierce the spinal cord. A lumbar puncture typically is made at the level of either the L₂ and L₃ vertebrae or the L₃ and L₄ vertebrae. To locate this level, the physician palpates the highest points of the iliac crests, which are at the same horizontal level as the spinous process of the L₁ vertebra. The physician can then insert the lumbar puncture needle either directly above or directly below the spinous process of L₄ when the vertebral column is flexed.

**Site of needle insertion for a lumbar puncture.**
14.3 Sectional Anatomy of the Spinal Cord and Spinal Roots

We now examine the spinal cord and a pair of associated spinal roots in cross section to explore their structural and functional relationship. The spinal cord as shown in figure 14.4 appears in cross section as a roughly cylindrical structure that is slightly flattened both posteriorly and anteriorly. It has a relatively narrow posterior median sulcus and a slightly wider anterior median fissure that are readily visible. Observe that the spinal cord is partitioned into two areas: an inner gray matter region and an outer white matter region. Here we discuss the general composition and function of these regions.

14.3a Distribution of Gray Matter

LEARNING OBJECTIVES

7. Identify the four anatomic locations of gray matter on either side of the spinal cord.
8. Describe the structures that form each gray matter region.
9. Trace sensory input to the spinal cord and motor output from the spinal cord.

Gray matter was first discussed in section 13.1c, where it was described as (1) being primarily composed of the dendrites and cell bodies of neurons and (2) functioning as a processing center. The gray matter within the spinal cord is centrally located, and its shape resembles a letter H or a butterfly. The gray matter is subdivided into the following components on each side of the spinal cord: a posterior horn, a lateral horn, an anterior horn, and a bar of gray matter that connects the left and right sides called the gray commissure. The gray matter of each horn is discussed first. You will find it helpful to refer to figure 14.4b as you read through this section.

Posterior horns are both the left and right posterior masses of gray matter. The gray matter forming the posterior horns is due to the presence of the dendrites and cell bodies of interneurons (the neurons that are located completely within the CNS; see section 12.2d). Sensory neurons within the spinal nerves extend through the posterior root and synapse with the dendrites and cell bodies of the interneurons within the posterior horn. The posterior horn gray matter on each side of the spinal cord is subdivided regionally into both somatic sensory nuclei and visceral sensory nuclei based upon the specific type of sensory neurons that synapse there.

The somatic sensory nuclei (light blue–shaded region) is the site for synapses between somatic sensory neurons (light blue line) that extend from somatic sensory receptors (e.g., tactile receptors within the skin; see section 16.2a) and the interneurons within the posterior horns of the spinal cord. (Recall that somatic refers to “body” in general).

The visceral sensory nuclei (dark blue–shaded region) is the location for synapses between visceral sensory neurons (dark blue line) that extend from visceral sensory receptors (e.g., baroreceptors...
of the urinary wall) to the interneurons within the posterior horns of the spinal cord. (Recall that visceral refers to an internal organ.)

**Anterior horns** are both the left and right anterior masses of gray matter. The gray matter of the anterior horns is due to the presence of the dendrites and cell bodies of somatic motor neurons. Collectively, they form the somatic motor nuclei (red shaded area), which forms the entire anterior horn on each side of the spinal cord. The axons of somatic motor neurons (red line) extend to and innervate a somatic effector. The somatic effector includes only the muscle that can be controlled consciously or voluntarily (i.e., skeletal muscle). Note: A type of virus that specifically targets somatic motor neurons within the spinal cord and potentially result in muscle paralysis is the poliovirus (see Clinical View 14.2: “Poliomyelitis”).

**Lateral horns** are both the left and right lateral masses of gray matter. They are located only within the T1–L2 parts of the spinal cord, not the entire length of the spinal cord. The gray matter of the lateral horns is due to the presence of the dendrites and cell bodies of autonomic motor neurons. Collectively, they form the autonomic motor nuclei (orange shaded region), which makes up the entire lateral horn on each side of the spinal cord. The axons of autonomic motor neurons (orange line) extend to and innervate autonomic (or visceral) effectors. Autonomic effectors include those body structures that are not controlled consciously or voluntarily (i.e., cardiac muscle, smooth muscle, and glands). (Examples of autonomic effectors include cardiac muscle of the heart, smooth muscle of the stomach wall, and exocrine glands of the pancreas. Autonomic effectors are components of the autonomic nervous system and are discussed in detail in chapter 15.)

**WHAT DO YOU THINK?**

2 How do the posterior horns differ in their composition and function from both the anterior horns and lateral horns?

**Posterior Root and Anterior Root Composition**

The composition of both the posterior root and the anterior root was first described in section 14.1c. Recall that the posterior root contains sensory neurons that extend from sensory receptors. We know that these sensory neurons include somatic sensory neurons, which relay nerve signals from somatic sensory receptors, and visceral sensory neurons, which relay nerve signals from visceral sensory receptors. Both types of sensory neurons synapse with interneurons within the gray matter of the posterior horns. In comparison, the anterior root contains motor neurons that extend to effectors. These motor neurons include somatic motor neurons, which extend to skeletal muscle, and autonomic (or visceral) motor neurons, which extend to autonomic effectors. As noted in section 14.1c, a posterior root has an associated ganglion (posterior root ganglion) and an anterior root does not.

**Gray Commissure**

The gray commissure (kom′i-shür; commissura = a seam) within the spinal cord forms a bar of gray matter connecting the left and right sides of the posterior, lateral, and anterior horns. The gray commissure is an unusual gray matter region because it primarily houses unmyelinated axons (which lack the whitish-colored myelin and, thus, appear gray in color). This bar of gray matter serves as a communication route between the right and left sides of the spinal cord. The central canal is a small, internal channel that extends through the center of the gray commissure along the entire length of the spinal cord. As with the ventricles of the brain, the central canal is formed during embryonic development from the neural canal within the neural tube (see section 14.7). The central canal contains cerebrospinal fluid (CSF), which enters this space from the fourth ventricle of the brain (see figure 13.7).

**CLINICAL VIEW 14.2**

**Poliomyelitis**

Poliomyelitis (pö′lë-me-ël′i-tis; polio = gray, myelos = marrow, ilits = infection) is an infection caused by one of the three strains of poliovirus. Infection is by oral-fecal or oral-oral route, and common routes of transmission are through contaminated food or water supplies. Most cases of polio are mild and may result in digestive or flulike symptoms. However, in about 1% of the cases, the virus spreads to the nervous system and attacks somatic motor neurons in the anterior horn of the spinal cord. (The disease got its name by describing the inflammation of the gray matter of the spinal cord.) These cases are referred to as paralytic polio. Here, the motor neurons are damaged or destroyed, resulting in paralysis of the muscles innervated by those segments of the spinal cord. Paralysis may be temporary or permanent, depending upon the extent of somatic motor neuron damage. Polio is rare in the Western world due to an active vaccination program, but it is still endemic in Pakistan, Afghanistan, Nigeria, Syria, and Chad (where vaccination programs have been disrupted or incomplete).

**WHAT DID YOU LEARN?**

6 Place the following structures in order for relaying sensory input to the spinal cord: posterior horn, sensory receptor (e.g., tactile receptors of the skin), spinal nerve, and posterior root.

7 Place the following structures in order for relaying motor output from the spinal cord to skeletal muscle: anterior horn, effector (skeletal muscle), spinal nerve, and anterior root.

**14.3b Distribution of White Matter**

**LEARNING OBJECTIVES**

10. Identify the locations of white matter within the spinal cord.

11. List the three anatomic divisions of the white matter, and explain their general composition.

White matter was first discussed in section 13.1c, where it was described as being (1) primarily composed of myelinated axons and (2) functioning to relay nerve signals. The white matter of the spinal cord is external to the gray matter and on each side of the cord is partitioned into three distinct anatomic structural regions based upon their location within the spinal cord. Each of these regions is called a funiculus (fú-nik′ū-lús; pl. funiculi,1 fú-ník’ū-lī; funis = cord) (figure 14.4). Each posterior funiculus is white matter that lies between the posterior gray horns on the posterior side of the cord and the posterior median sulcus. The lateral funiculus is the white matter on each lateral side of the spinal cord. The anterior funiculus is composed of white matter that occupies the space on each anterior side of the cord between anterior gray horns and the anterior median fissure; the anterior funiculi are interconnected by the white commissure.

1 Note: Anterior and lateral funiculi were formerly called columns. The Federative Committee on Anatomical Terminology (FCAT) now states that the term column refers to structures within the gray matter of the spinal cord, whereas funiculus refers to the white matter regions.
The axons within each funiculus are organized into smaller structural units (or bundles of myelinated axons) called fasciculi (fás-ik′ū-lē; fascis = bundle) (figure 14.5). White matter on each side of the cord can also be referred to as tracts, which have common functions. Individual tracts are either (1) sensory (or ascending) tracts, which conduct nerve signals from the spinal cord to the brain, or (2) motor (or descending) tracts, which conduct nerve signals from the brain to the spinal cord. Figure 14.5 shows sensory tracts in blue shading on one side of the spinal cord and motor tracts in red shading on the other side of the spinal cord. However, keep in mind that the spinal cord is symmetric; both sensory tracts and motor tracts are on both sides of the cord.

Observe in figure 14.5 that each funiculus region (posterior, lateral, and anterior funiculus) contains sensory tracts (blue shading), and the lateral and anterior funiculi contain motor tracts (orange and red shading). Thus, sensory input is relayed to the brain within each fasciculus, whereas motor output from the brain is relayed only within the lateral and anterior funiculi (not within the posterior funiculus).

Tracts are myelinated axons that have a common origin, a common destination, and a similar function. The name of each tract reflects its origin and destination. For example, sensory tracts usually begin with the prefix spino-, indicating that they originate in the spinal cord. The second part of the name provides its destination. An example is the sensory spinothalametic tract, which extends from the spinal cord to the thalamus. Another example is the spinocerebellar tract, which extends from the spinal cord to the cerebellum. (The fasciculus gracilis and fasciculus cuneatus are exceptions to how sensory tracts are named.) Motor pathways begin either with cortico-, indicating an origin in the cerebral cortex, or with the name of a brainstem nucleus (such as rubro-, indicating an origin within the red nucleus of the midbrain; see section 13.5a). Thus, the corticospinal tract extends from the cerebral cortex to the spinal cord. How tracts within the spinal cord function in sensory and motor pathways is discussed in section 14.4.

Figure 14.5 Tracts and Fasciculi Within the Spinal Cord. The major sensory (ascending) tracts and fasciculi are bilaterally symmetric tracts and shown here in shades of blue. The major motor (descending) tracts are bilaterally symmetric tracts and shown here in shades of red and orange.

Figure 14.6 presents representative cross sections through each spinal cord part. This illustration helps us to realize that both the size and shape of the spinal cord in each section along its length vary in each spinal cord part. The difference in the relative amounts of gray matter and white matter reflects the function of that part of the spinal cord. For example, the lumbar part of the spinal cord has a greater amount of gray matter because more neuron cell bodies are located there that have axons extending from there to innervate the lower limbs.

WHAT DID YOU LEARN?

What are the three types of funiculi? List the specific tracts found in each.

INTEGRATE

LEARNING STRATEGY

The white matter within the spinal cord is identified by the use of four terms based upon either structure or function.

Terms to identify structure:

- Funiculus: The specific location of the white matter (e.g., posterior funiculus is the white matter forming the posterior aspect of the spinal cord)
- Fasciculus: A structural subdivision of a funiculus that shares common features (e.g., fasciculus gracilis, which is within a posterior funiculus)

Terms to identify function:

- Tract: Myelinated axons that relay nerve signals from a common origin to a common destination (e.g., spinothalametic tract, which relays nerve signals from the spinal cord to the thalamus)
- Pathway: A more general term that includes all of the neurons (and associated structures) that relay nerve signals between the brain and the body. Spinal pathways include components within the brain (e.g., cerebrum), components of the spinal cord (identified as either fasciculi or tracts), and spinal nerves.
14.4 Sensory and Motor Pathways

Conduction pathway is an inclusive term that refers to all of the series of neurons (and their associated structures) that relay signals between the brain and the body. Pathways that extend between the brain and the torso and limbs include components that are (1) within the brain (e.g., cerebrum), (2) within the spinal cord (either a fasciculus or tract that extends through the spinal cord), and (3) the individual neurons within spinal nerves. Pathways also include integration and processing centers (gray matter) at different locations along each type of pathway where the neurons synapse.

14.4a Overview of Conduction Pathways

LEARNING OBJECTIVES

13. List the features common to all pathways.

Conduction pathways are identified as either sensory or motor pathways, depending upon the direction nerve signals are relayed relative to the brain. Sensory pathways include the sensory neurons that relay sensory input to the brain. Sensory pathways are also called ascending pathways because the nerve signals are relayed from the sensory receptors superiorly to the brain. Motor pathways include the series of motor neurons that relay motor output from the brain. Motor pathways are also called descending pathways because the nerve signals are relayed from the brain inferiorly to the body’s muscles and glands. Most conduction pathways—whether sensory or motor—share several general characteristics:

- **Paired tracts.** All pathways are composed of paired tracts. Thus, a pathway on one side of the CNS has a matching tract on the other side of the CNS.
- **Composed of two or more neurons.** Most pathways are composed of a series of two or three neurons that form the pathway.
- **Common location of neuron cell bodies.** Neuron cell bodies are located in one of three general places: the posterior root ganglion, the gray horns within the spinal cord, or nuclei within the brain along the pathway.
- **Common location of axons.** The axons of the different neurons extend through spinal nerves, the spinal cord (as named tracts or fasciculi), and the brain.
- **Decussation.** Most pathways include neurons that cross over, or decussate (de-kū-sāt′; decusseo = to make in the form of an X), from one side of the body to the other side at some point along the pathway—within either the spinal cord or the brain. This means that the left side of the brain receives sensory input from or initiates motor output to the right side of the body, whereas the right side of the brain receives sensory input from or initiates motor output to the left side of the body. The term contralateral (kon-trā-lat’er-āl; contra = opposite, latus = side) is used to indicate the relationship to the opposite side. Over 90% of all neurons within pathways decussate.
- **Limited ipsilateral pathway.** Pathways may have some neurons (about 10%) that remain on the same side of the body. The term ipsilateral (ip-si-lat’er-āl; ipse = same) is used to indicate the relationship to the same side.

WHAT DID YOU LEARN?

9. What characteristics are common to most conduction pathways?
Sensory pathways have been described as ascending pathways that relay sensory information from sensory receptors to the brain. Sensory input transmitted through the spinal cord (discussed in this section) is detected by general sense receptors. Understanding these pathways requires some preliminary discussion regarding general sense receptors (which are described in detail in sections 16.1 and 16.2).

Overview of Sensory Receptors

General sense receptors are sensory receptors located throughout the body (and are distinguished from the special sense receptors, which are limited to the head—the eyes, ears, nose, and tongue). General sense receptors are subdivided into two categories: somatic sensory (or somatosensory) receptors and visceral sensory receptors. Somatic sensory (or somatosensory) receptors are tactile receptors or proprioceptors. Tactile receptors are housed within both the skin and mucous membranes that line body cavities. These sensory receptors monitor characteristics of an object (e.g., texture). Proprioceptors are located within joints, muscles, and tendons to detect stretch and pressure relative to position and movement of the skeleton and skeletal muscles. Visceral sensory receptors are located in the walls of the viscera (internal organs) and blood vessels. They detect changes to an organ or a blood vessel (e.g., stretch).

Categorization of Sensory Pathways

Consequently, sensory pathways are organized into two categories depending upon the type of general sensory receptor involved. Somatosensory pathways process stimuli received from somatosensory receptors (e.g., tactile receptors within the skin, proprioceptors), whereas visceral sensory pathways process stimuli received from visceral sensory receptors (i.e., from the receptors of internal organs). We limit our discussion to somatosensory pathways.

Sensory pathways use a series of two or three neurons to transmit nerve signals from the sensory receptors to the brain, which are the primary neuron, secondary neuron, and tertiary neuron.

- The primary neuron (or first-order neuron) is the first neuron in the chain of neurons. The primary neuron extends from the sensory receptor to the CNS (brain or spinal cord), where it synapses with a secondary neuron.
- The secondary neuron (or second-order neuron) is an interneuron that extends from the primary neuron to either the tertiary neuron or the cerebellum.
- The tertiary neuron (or third-order neuron) is also an interneuron. It extends from the secondary neuron to the cerebrum (specifically, the primary somatosensory cortex of the parietal lobe; see section 13.3c). Pathways that lead to the cerebrum do not have a tertiary neuron.

There are three major types of somatosensory pathways: the posterior funiculus–medial lemniscal pathway, the anterolateral pathway, and the spinocerebellar pathway. As you read the discussion of these sensory pathways, consider the following for each: (1) what type of sensory receptors are involved and what type of sensory information they are providing to the brain, (2) the location of each sensory neuron within the chain of two or three neurons that compose the pathway, and (3) what region of the brain receives and processes this sensory information.

Posterior Funiculus–Medial Lemniscal Pathway

The posterior funiculus–medial lemniscal pathway uses a chain of three sensory neurons to communicate with the brain about a specific stimulus (figure 14.7). This pathway originates at either of the two types of somatosensory receptors: (1) tactile receptors housed within both the skin and mucous membranes or (2) proprioceptors within joints, muscles, and tendons. This sensory input is providing information to the brain (specifically, the cerebral cortex) about discriminative touch, precise pressure, and vibration and limb position (proprioception). This pathway is bilaterally symmetric—but to avoid confusion, only sensory input from the right side of the body is shown here. Decussation of axons occurs just prior to the medial-lemniscus in the medulla oblongata. The primary neuron is purple, the secondary neuron is blue, and the tertiary neuron is green.

Figure 14.7 Posterior Funiculus–Medial Lemniscal Pathway. This pathway transmits sensory information about discriminative touch, precise pressure, and vibration and limb position (proprioception). This pathway is bilaterally symmetric—but to avoid confusion, only sensory input from the right side of the body is shown here. Decussation of axons occurs just prior to the medial-lemniscus in the medulla oblongata. The primary neuron is purple, the secondary neuron is blue, and the tertiary neuron is green.
• The axon of the **secondary neuron** (blue line) extends from the somatosensory receptor to the spinal cord (via the posterior root). The secondary neuron synapses with the tertiary neuron within the posterior horn of the spinal cord, as described in section 14.3a. (Note: The synapsing of the primary neuron at the level of the spinal cord is the most significant structural difference of the anterolateral pathway when compared to the posterior funiculus–medial lemniscal pathway.)

• The axon of the **primary neuron** (purple line) extends from the somatosensory receptor into the spinal cord (via the posterior root). The primary neuron synapses with the secondary neuron within the posterior horn of the spinal cord, as described in section 14.3a. (Note: The synapsing of the primary neuron at the level of the spinal cord is the most significant structural difference of the anterolateral pathway when compared to the posterior funiculus–medial lemniscal pathway.)

• The axon of the **tertiary neuron** (green line) extends from the thalamus to the cerebrum (specifically to a location within the primary somatosensory cortex housed within the postcentral gyrus of the parietal lobe; see figure 13.13 in section 13.3c). Conscious perception of the tactile or proprioceptor sensory input occurs within the parietal lobe.

The name of this pathway (posterior funiculus–medial lemniscal pathway) is derived from the two components of white matter that it extends through: the posterior funiculus within the spinal cord and medial lemniscus within the brain.

### WHAT DO YOU THINK?

3 With respect to the posterior funiculus–medial lemniscal pathway, (1) what type of sensory receptors are involved, and what type of sensory information are they providing to the brain, (2) what is the location of each of the sensory neurons within the chain of three neurons that compose this pathway, and (3) what region of the brain receives the sensory information?

### Anterolateral Pathway

The **anterolateral pathway** (or spinothalamic pathway) uses a chain of three neurons to communicate with the brain about a specific stimulus (figure 14.8). This pathway originates at tactile somatosensory receptors within both the skin and mucous membranes. This sensory input is providing information to the brain (specifically, the cerebral cortex) about crude touch and pressure as well as pain and temperature. Typically, sensations that require us to act in response to the stimulus (such as either an itch that makes us want to scratch or tickling that makes us jerk away) are relayed through the anterolateral pathway.

Three sensory neurons compose the chain of neurons within this pathway:

- The axon of the **primary neuron** (purple line) extends from the somatosensory receptor into the spinal cord (via the posterior root). The primary neuron synapses with the secondary neuron within the posterior horn of the spinal cord, as described in section 14.3a. (Note: The synapsing of the primary neuron at the level of the spinal cord is the most significant structural difference of the anterolateral pathway when compared to the posterior funiculus–medial lemniscal pathway.)

- The axon of the **secondary neuron** (blue line) extends from the spinal cord to the thalamus. The axons project within the spinothalamic tract—either within the anterior funiculus (via the anterior spinohalamic tract) or the lateral funiculus (via the lateral spinohalamic tract) to the thalamus. The thalamus “filters” the incoming sensory input as described in section 14.4b. (Decussation occurs to the opposite side within the spinal cord as the axons extend into the spinohalamic tract.)

- The axon of the **tertiary neuron** (green line) extends from the thalamus to the cerebrum (specifically, to a location within the primary somatosensory cortex housed within the postcentral gyrus of the parietal lobe (see figure 13.13 in section 13.3c). Conscious perception of the tactile or proprioceptor sensory input occurs within the parietal lobe.

The principal name of this pathway (anterolateral pathway) is derived from the location of the two funiculi through which it ascends (anterior funiculus and lateral funiculus). Its secondary name (spinotbhalamic pathway) is derived from the tracts that relay the nerve signals within the spinal cord to the thalamus.

### WHAT DO YOU THINK?

4 Regarding the anterolateral pathway, (1) what type of sensory receptor is involved, and what type of sensory information is being provided to the brain, (2) what is the location of each of the sensory neurons within the chain of three neurons that compose this pathway, and (3) what specific region of the brain receives the sensory information?

### Spinocerebellar Pathway

The **spinocerebellar pathway** uses a chain of only two neurons to communicate with the brain about a specific stimulus (figure 14.9). This pathway originates at proprioceptors within joints, muscles, and tendons at different locations in the body. This sensory input is providing information to the brain (specifically, the cerebellum) related to subconscious postural input, which helps in maintaining balance and posture (see section 13.6b).
Spinocerebellar pathway

Posterior spinocerebellar tract
Anterior spinocerebellar tract
Spinocerebellar pathway

The name of this pathway (spinocerebellar pathway) is derived from the origin of its tracts that ascend from the spinal cord to the cerebellum.

**Table 14.1 Functions and Neuron Locations of Principal Sensory Spinal Cord Pathways**

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Posterior Funiculus–Medial Lemniscal</th>
<th>Anterolateral</th>
<th>Spinoceullar</th>
<th>Spinoceullar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Components of Pathway</td>
<td>Fasciculus Cuneatus</td>
<td>Fasciculus Gracilis</td>
<td>Anterior Spinothalamic Tract</td>
<td>Lateral Spinothalamic Tract</td>
</tr>
<tr>
<td>Function</td>
<td>Sensory input for limb position and discriminative touch, precise pressure, and vibration sensation</td>
<td>Sensory input for crude touch, pressure, pain, and temperature</td>
<td>Sensory input sent from proprioceptors to cerebellum for subconscious interpretation</td>
<td></td>
</tr>
<tr>
<td>Relays input from</td>
<td>Relays input from upper limb, superior trunk, neck, posterior head</td>
<td>Relays input for crude touch and pressure</td>
<td>Relays input for pain and temperature</td>
<td></td>
</tr>
<tr>
<td>Primary neuron</td>
<td>Cell bodies within posterior root ganglion</td>
<td>Cell bodies within spinal cord</td>
<td>Cell bodies within posterior root ganglion</td>
<td></td>
</tr>
<tr>
<td>Secondary neuron</td>
<td>Extends from medulla oblongata to thalamus</td>
<td>Extends from spinal cord to thalamus</td>
<td>Extends from spinal cord to cerebellum</td>
<td></td>
</tr>
<tr>
<td>Tertiary neuron</td>
<td>Extends from thalamus to cerebral cortex</td>
<td>Extends from thalamus to cerebral cortex</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Structures involved in</td>
<td>Axons of secondary neurons decussate just prior to medial lemniscus</td>
<td>Axons of secondary neurons decussate within spinal cord at level of entry</td>
<td>Some axons decussate in spinal cord and pons, whereas other axons do not decussate</td>
<td></td>
</tr>
</tbody>
</table>

Two sensory neurons compose the chain of neurons within this pathway. (There is no tertiary neuron.)

- The axon of the primary neuron (purple line) extends from a proprioceptor into the spinal cord (via the posterior root). The primary neuron synapses with the secondary neuron within the posterior horn of the spinal cord. (This is similar to the synapse between primary and secondary neurons within the anterolateral pathway.)

- The axon of the secondary neuron (blue line) extends from the spinal cord within the spinocerebellar tract—either within the anterior or within the posterior portion of the lateral funiculus to the cerebellum.

The name of this pathway (spinocerebellar pathway) is derived from the origin of its tracts that ascend from the spinal cord to the cerebellum.

**WHAT DO YOU THINK?**

5 Regarding the spinocerebellar pathway, (1) what type of sensory receptor is involved, and what type of sensory information are they providing to the brain, (2) what is the location of each of the sensory neurons within the chain of two neurons that compose this pathway, and (3) which region of the brain receives the sensory information?

**WHAT DID YOU LEARN?**

10 What are the general locations and functions of primary, secondary, and tertiary neurons in sensory pathways?

11 What types of information does the posterior funiculus–medial lemniscal pathway transmit?
14.4c Motor Pathways

LEARNING OBJECTIVES

17. Define a motor pathway, and describe its actions.
18. Distinguish between an upper motor neuron and a lower motor neuron, based upon function and cell body location.
19. Compare and contrast the direct and indirect motor pathways.

Motor pathways are the descending pathways that originate within the brain and act to control effectors. Here we discuss the motor pathways that specifically control skeletal muscle of the torso and limbs. These motor pathways originate from the cerebral cortex, the cerebral nuclei, or the brainstem (figure 14.10).

At least two motor neurons are present within the motor pathway to transmit signals from the brain to the body: an upper motor neuron and a lower motor neuron.

- An upper motor neuron is the first neuron in a chain of neurons. The cell body of the upper motor neuron is housed within the cerebral cortex, cerebral nuclei, or a specific nucleus within the brainstem. Axons of the upper motor neuron synapse either directly upon lower motor neurons (in direct pathways) or upon interneurons that ultimately synapse upon lower motor neurons (in indirect pathways). The upper motor neurons either excite or inhibit the activity of lower motor neurons.

- The lower motor neuron is the last neuron in the chain of neurons. The cell body of a lower motor neuron is housed within the anterior horn of the spinal cord (as described in section 14.3a). Axons of the lower motor neurons exit the spinal cord through the anterior root and project to and innervate a specific skeletal muscle. The lower motor neuron always excites the skeletal muscle fibers to contract.

![Figure 14.10 Corticospinal Tracts](image)

Motor neuron axons form two types of motor pathways: the direct pathway and indirect pathway. The direct pathway is responsible for conscious control of skeletal muscle activity; the indirect pathway is responsible for subconscious (or reflexive) control of skeletal muscle.

**Direct Pathway**

The direct (or pyramidal) pathway uses a chain of only two motor neurons to communicate between the brain and the skeletal muscles (figure 14.10). This pathway originates in the primary motor cortex of the cerebral frontal lobe. The direct pathway name is derived from the presence of only one upper motor neuron and one lower motor neuron. The name pyramidal is derived from the pyramid-like shape of the cell bodies of the upper motor neurons within gray matter of the cerebral cortex.

- The axon of the upper motor neuron extends from the frontal lobe of the cerebral cortex through the internal capsule and cerebral peduncles of the brain (see section 13.3d) and through a corticospinal tract within the spinal cord. This axon synapses on the lower motor neuron within the anterior horn of the spinal cord. The dendrites and cell bodies of the lower motor neurons form the gray matter of the anterior horn.

- The axon of the lower motor neuron extends from the spinal cord through the anterior root into the spinal nerve to innervate the target skeletal muscle.

The direct pathways are housed within one of two pathways within the spinal cord: the lateral corticospinal tract and anterior corticospinal tract. These two pathways differ in several significant ways, including the specific muscles they innervate and control:

- The lateral corticospinal tracts (which composes 85% of the direct pathway) innervates skeletal muscles that control skilled movements in the limbs, such as playing a guitar, dribbling a soccer ball, or typing on your computer keyboard.

- The anterior corticospinal tracts (which composes the other 15% of the direct pathway) innervate axial skeletal muscle.

Decussation of the lateral corticospinal tracts occurs to the opposite side within the brain at the medulla oblongata (specifically, at pyramids of the medulla oblongata; see section 13.5c), whereas the anterior corticospinal tracts decussate through the anterior gray commissure at the level of a spinal cord segment.

INTEGRATE

CONCEPT CONNECTION

The corticobulbar (kör’ti-kō-bŭl’bar) pathways are another type of direct pathway, but they originate from the facial region of the motor homunculus within the primary motor cortex to help regulate activity of muscles of the face and neck. Note that these tracts differ from the others because (1) they do not pass through the spinal cord and (2) they involve cranial nerves (instead of spinal nerves). Axons of these upper motor neurons extend to the brainstem, where they synapse with lower motor neuron cell bodies that are housed within brainstem cranial nerve nuclei. Axons of these lower motor neurons help form some of the cranial nerves (see section 13.9).

Indirect Pathway

Several nuclei within the brainstem initiate motor commands for skeletal muscle activities that occur at a subconscious, or reflexive, level. The indirect pathway is so named because upper motor neurons originate within brainstem nuclei and take a complex, circuitous route through the
The different tracts of the indirect pathway are grouped according to their primary functions as either a lateral pathway or a medial pathway. The lateral pathway regulates and controls precise, discrete movements and tone in flexor muscles of the limbs—for example, the type of movement required to gently lay a baby in a crib. This pathway consists of the rubrospinal (rubro = red) tracts that originate in the red nucleus of the midbrain (see section 13.5a).

The medial pathway regulates reflexive muscle tone and gross movements of the muscles of the head, neck, proximal parts of the limbs, and trunk. Within the medial pathway, three groups of tracts originate in the midbrain, pons, or medulla oblongata.

- The reticulospinal (re-tik-ə-lō-spīn′āl) tracts originate from the reticular formation in the midbrain (see section 13.5a). They help control reflexive movements related to posture and maintaining balance.
- The tectospinal (tek-tō-spīn′āl) tracts extend from the superior and inferior colliculi in the tectum of the midbrain to help regulate reflexive positional changes of the upper limbs, eyes, head, and neck as a consequence of visual and auditory stimuli.
- The vestibulospinal (ves-tīb′ə-lō-spīn′āl) tracts originate within vestibular nuclei of the brainstem. Nerve signals conducted within these tracts regulate reflexive muscular activity that helps maintain balance during sitting, standing, and walking.

Table 14.2 summarizes the characteristics of the principal types of motor pathways. Figure 14.11 summarizes the main differences between the sensory and motor pathways.

**WHAT DID YOU LEARN?**

12. What are the locations and functions of upper and lower motor neurons in the motor pathways?
13. What are the differences between direct and indirect motor pathways?

**CLINICAL VIEW 14.3**

**Treating Spinal Cord Injuries**

Spinal cord injuries frequently leave individuals paralyzed and unable to perceive sensations to varying degrees, depending upon the location and extent of the injury. In recent years, advances have been made in the treatment of spinal cord injuries (although some of the findings are still preliminary). Prompt use of steroids immediately after the injury appears to preserve some muscular function that might otherwise be lost. Early use of antibiotics has substantially reduced the number of deaths caused by pulmonary and urinary tract infections that accompany spinal cord injuries. Recent research with rats has achieved reconnection and partial restoration of function of severed spinal cords. In addition, other research indicates that neural stem cells may be able to regenerate spinal cord axons.

**INTEGRATE**

**Table 14.2** Principal Motor Spinal Cord Pathways

<table>
<thead>
<tr>
<th>Tract</th>
<th>Manner of Decussation</th>
<th>Destination of Upper Motor Neurons</th>
<th>Termination Site</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIRECT PATHWAY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corticobulbar tracts</td>
<td>All cranial nerve motor nuclei receive bilateral (both ipsilateral and contralateral) input except CN VI, VII to the lower face, and XII (these nerves receive only contralateral input)</td>
<td>Brainstem only</td>
<td>Cranial nerve nuclei; reticular formation</td>
<td>Voluntary movement of cranial and facial muscles</td>
</tr>
<tr>
<td>Lateral corticospinal tracts</td>
<td>All decussate at the pyramids</td>
<td>Lateral funiculus</td>
<td>Gray matter region between posterior and anterior horns; anterior horn; all levels of spinal cord</td>
<td>Voluntary movement of appendicular muscles</td>
</tr>
<tr>
<td>Anterior corticospinal tracts</td>
<td>Decussation occurs in spinal cord at level of lower motor neuron cell body</td>
<td>Anterior funiculus</td>
<td>Gray matter region between posterior and anterior horns; anterior horn; cervical part of spinal cord</td>
<td>Voluntary movement of axial muscles</td>
</tr>
<tr>
<td><strong>INDIRECT PATHWAY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Lateral Pathway</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rubrospinal tract</td>
<td>Decussate at ventral tegmentum of midbrain</td>
<td>Lateral funiculus</td>
<td>Lateral region between posterior and anterior horns; anterior horn; cervical part of spinal cord</td>
<td>Regulates and controls precise discrete movements and tone in flexor muscles of the limbs</td>
</tr>
<tr>
<td><strong>Medial Pathway</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reticulospinal tract</td>
<td>No decussation (ipsilateral)</td>
<td>Anterior funiculus</td>
<td>Medial region between posterior and anterior horns; anterior horn; all parts of spinal cord</td>
<td>Controls reflexive movements related to posture and maintaining balance</td>
</tr>
<tr>
<td>Tectospinal tract</td>
<td>Decussate at dorsal tegmentum of midbrain</td>
<td>Anterior funiculus</td>
<td>Medial region between posterior and anterior horns; anterior horn; cervical part of spinal cord</td>
<td>Regulates reflexive positional changes of the upper limbs, eyes, head, and neck due to visual and auditory stimuli</td>
</tr>
<tr>
<td>Vestibulospinal tract</td>
<td>Some decussate (contralateral) and some do not (ipsilateral)</td>
<td>Anterior funiculus</td>
<td>Medial region between posterior and anterior horns; anterior horn; medial tracts to cervical and superior thoracic parts of spinal cord; lateral tracts to all parts of spinal cord</td>
<td>Regulates reflexive muscular activity that helps maintain balance during sitting, standing, and walking</td>
</tr>
</tbody>
</table>
Figure 14.11 Differences Between Sensory and Motor Pathways.

(a) Sensory pathways transmit nerve signals from sensory receptors that ascend from the anterior, posterior, and lateral funiculi of the spinal cord to the brain. These pathways use up to three neurons to transmit this information (primary, secondary, and tertiary neurons). (b) Motor pathways transmit nerve signals from the brain and descend to effectors. These pathways typically travel through the anterior and lateral funiculi of the spinal cord, and they use two neurons (upper and lower motor neurons).

(a) Sensory Pathways
- Nerve signals ascend to the brain in sensory pathways.

(b) Motor Pathways
- Nerve signals descend from the brain in motor pathways.

Most sensory pathways travel in the posterior and lateral funiculi of the spinal cord.

Sensory pathways to the cerebrum use up to three neurons: a primary, secondary, and a tertiary neuron.

Motor pathways use at least two motor neurons: one or more upper motor neurons and a lower motor neuron.

| Upper motor neuron (cell body located in cerebral cortex or a brainstem nucleus) |
| Lower motor neuron (cell body located in anterior horn or a brainstem nucleus) |

INTEGRATE CONCEPT OVERVIEW
14.5 Spinal Nerves

We have discussed how the 31 pairs of spinal nerves (C1–C8, T1–T12, L1–L5, S1–S5, and C01) have axons of sensory neurons that extend from sensory receptors through the posterior root to the spinal cord (with their cell bodies forming the posterior root ganglia) and axons of motor neurons that extend from the spinal cord to effectors (muscles and glands). Here we describe the peripheral distribution of the spinal nerves.

14.5a General Distribution of Spinal Nerves

LEARNING OBJECTIVES

20. For each spinal nerve (except C1 and C01), identify the opening where that nerve exits from the vertebral column.
21. Compare and contrast the anterior and posterior rami of a spinal nerve.
22. Define a dermatome, and explain its clinical significance.

Each spinal nerve (except C1 and C01) extends through an intervertebral foramen to exit the vertebral column (as described in section 14.2). Each spinal nerve splits almost immediately into two primary branches, termed rami (figure 14.12). The posterior (dorsal) ramus (ra’müs; pl., rami, ra’mi; branch) is the smaller of the two main branches. It innervates the deep muscles of the back (e.g., erector spinae and transversospinals; see section 11.4) and the skin of the back.

The anterior (ventral) ramus is the larger of the two main branches. The anterior ramus splits into multiple other branches, which innervate skin and skeletal muscles of the anterior and lateral portions of the trunk, the upper limbs, and the lower limbs. Many of the anterior rami go on to form nerve plexuses, which are described in the following sections.

Additional rami, called the rami communicantes, are also associated with spinal nerves. These rami contain axons associated with the autonomic nervous system (ANS). Each set of rami communicantes extends between the spinal nerve and a spherical structure called the sympathetic trunk ganglion. These ganglia are interconnected and form a beaded necklace–like structure called the sympathetic trunk that extends parallel and lateral to the vertebral column. The structures associated with the ANS are described in detail in section 15.4a.

WHAT DO YOU THINK?

10. Why is an anterior ramus so much larger than a posterior ramus?

Dermatomes

A dermatome (der’mä-tōm; derma = skin, tome = a cutting) is a specific segment of skin innervated by a single spinal nerve. All spinal nerves except for C1 innervate a segment of skin, and each area of the skin that is innervated by a specific spinal nerve has been mapped. Collectively, this map is called a dermatome map (figure 14.13). The dermatome map follows a segmental pattern along the body (although there is slight overlap between adjacent spinal nerves). For example, the horizontal segment of skin around the umbilicus (navel) region is supplied by the anterior ramus of the T10 spinal nerve.

Dermatomes are clinically important because they can indicate potential damage to one or more spinal nerves. For example, if a patient experiences anesthesia (an’es-thē’zē-ā; loss of sensation or numbness) along the medial side of the arm and forearm, the C8 spinal nerve may be damaged.

Dermatomes are also involved in referred visceral pain, a phenomenon in which pain or discomfort from one organ is mistakenly referred to a dermatome. For example, the appendix is innervated by axons from the T10 regions of the spinal cord, so appendicitis typically causes referred visceral pain to the T10 dermatome in the umbilicus region rather than in the abdominopelvic region of the appendix itself. Thus, pain in a dermatome typically arises from an organ nowhere near the dermatome. Referred visceral pain is explored further in section 16.2b.

WHAT DID YOU LEARN?

14. What are the differences between an anterior ramus and a posterior ramus of a typical spinal nerve?
15. What is a dermatome, and why may a dermatome be clinically significant?
A dermatome is an area of skin supplied by a single spinal nerve. These diagrams only approximate the dermatomal distribution.

Some individuals (usually adults over 50) experience a reactivation of their childhood chicken pox infection, a condition termed shingles (shing'gls). Psychological stress, other infections (such as a cold or the flu), and even a sunburn can trigger the development of shingles.

During the initial infection, the chicken pox virus (varicella-zoster) sometimes leaves the skin and invades the posterior root ganglia. There, the virus remains latent until adulthood, when it becomes reactivated and proliferates, traveling through the sensory axons to the dermatome. (The word shingles is derived from the Latin word cingulum, meaning “girdle,” reflecting the dermatomal pattern of its spread.) The virus gives rise to a rash and blisters along the dermatome, which are often accompanied by intense burning or tingling pain.

Antiviral medication (e.g., acyclovir) may reduce the severity and duration of symptoms of shingles. Additionally, older adults may receive a vaccine for shingles, which may help prevent or reduce the severity of the disease.
14.5b Nerve Plexuses

LEARNING OBJECTIVE

23. Define a nerve plexus.

A nerve plexus (plek′sūs; a braid) is a network of interweaving anterior rami of spinal nerves. The anterior rami of most spinal nerves form nerve plexuses on both the right and left sides of the body. These nerve plexuses then split into multiple "named" nerves that innervate various body structures. The main plexuses are the cervical plexuses, brachial plexuses, lumbar plexuses, and sacral plexuses (see figure 14.1).

WHAT DO YOU THINK?

7. What is the benefit of having an intricate nerve plexus, rather than a single spinal nerve that innervates a structure?

Nerve plexuses are organized such that axons from each anterior ramus extend to body structures through several different branches. In addition, each terminal branch of the plexus houses axons from several different spinal nerves. Thus, damage to a single segment of the spinal cord or damage to a single spinal nerve generally does not result in complete loss of innervation to a particular muscle or region of skin.

Most of the thoracic spinal nerves, as well as nerves S5–Co1, do not form plexuses. We discuss the anterior rami of thoracic spinal nerves (called intercostal nerves) first, followed by the individual nerve plexuses.

WHAT DID YOU LEARN?

16. What is the composition of a typical nerve plexus?

14.5c Intercostal Nerves

LEARNING OBJECTIVE

24. Identify the distribution of the intercostal nerves.

The anterior rami of spinal nerves T1–T11 are called intercostal nerves because they are located within the intercostal space sandwiched between two adjacent ribs (figure 14.14). T12 is

Figure 14.14 Intercostal Nerves. Intercostal nerves are the anterior rami of thoracic spinal nerves. They are typically distributed as shown here.
The intercostal nerves innervate much of the torso wall and portions of the upper limb (see the dermatomal map in figure 14.13). The specific innervation pattern of the T1–T12 nerves is as follows:

- A portion of the anterior ramus of T1 helps form the brachial plexus, but a branch of it is housed within the first intercostal space.
- The anterior ramus of nerve T2 emerges from its intervertebral foramen and innervates the intercostal muscles of the second intercostal space. Additionally, a branch of T2 transmits sensory information from the skin covering the axilla and the medial surface of the arm.
- Anterior rami of nerves T3–T6 follow the costal grooves of the ribs to innervate the intercostal muscles and receive sensations from the anterior and lateral chest wall.
- Anterior rami of nerves T7–T12 innervate not only the inferior intercostal spaces but also the abdominal muscles and their overlying skin.

**WHAT DID YOU LEARN?**

17. In general, what do the intercostal nerves innervate?

### 14.5d Cervical Plexuses

**LEARNING OBJECTIVES**

25. List the nerves of the cervical plexuses.
26. Explain the action of the phrenic nerve.

The left and right cervical plexuses are located deep on each side of the neck, immediately lateral to cervical vertebrae C1–C4 (figure 14.15). They are formed primarily by the anterior rami of spinal nerves C1–C4. The fifth cervical spinal nerve is not considered part of the cervical plexus, although it contributes some axons to one of the plexus branches. Branches of the cervical plexus called a subcostal nerve, because it arises inferior to the ribs, not between two ribs. With the exception of T1, the intercostal nerves do not form plexuses. The intercostal nerves innervate much of the torso wall and portions of the upper limb (see the dermatomal map in figure 14.13).
hypoglossal and phrenic are not considered part of this plexus. Note: rami of spinal nerves C5–T1 ply the upper limb. Each brachial plexus is formed by the anterior branches of the cervical plexuses are described in detail in table 14.3. The left and right brachial plexuses are networks of nerves that supply the upper limb. Each brachial plexus is formed by the anterior rami of spinal nerves C5–T1 (figure 14.16). The components of the brachial plexus extend laterally from the neck, pass superior to the first rib, and then continue into the axilla. Each brachial plexus innervates the pectoral girdle and the entire upper limb of one side.

**Structure of the Brachial Plexus**

Structurally, each brachial plexus is more complex than a cervical plexus and is composed of anterior rami, trunks, divisions, and cords when examined from a medial to lateral perspective. The anterior rami (sometimes called roots) of the brachial plexus are simply the continuations of the anterior rami of spinal nerves C5–T1. These rami emerge through the intervertebral foramina and extend to the neck. The five rami unite in the posterior triangle of the neck to form the superior, middle, and inferior trunks. Nerves C5 and C6 unite to form the superior trunk; nerve C7 remains as the middle trunk; and nerves C8 and T1 unite to form the inferior trunk.

Portions of each trunk divide deep to the clavicle into an anterior division and a posterior division (shown in green and purple, respectively, in figure 14.16). These contain axons that primarily innervate the anterior and posterior parts of the upper limb, respectively. At the axilla, these anterior and posterior divisions converge to form three cords. They are named with respect to their position near the axillary artery:

- The **posterior cord** is posterior to the axillary artery and is formed by the posterior divisions of the superior, middle, and inferior trunks; therefore, it contains portions of C5–T1 nerves.
- The **medial cord** is medial to the axillary artery and is formed by the anterior division of the inferior trunk; it contains portions of nerves C8–T1.

### Table 14.3 Branches of the Cervical Plexuses

<table>
<thead>
<tr>
<th>Nerves</th>
<th>Anterior Rami</th>
<th>Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MOTOR BRANCHES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ansa cervicalis</td>
<td>C1, C2, C3</td>
<td>Geniohyoid; infrahyoid muscles (omohyoid, sternohyoid, sternothyroid, and thyrohyoid)</td>
</tr>
<tr>
<td>Superior root</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior root</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segmental branches</td>
<td>C1–C4</td>
<td>Anterior and middle scalenes</td>
</tr>
<tr>
<td><strong>CUTANEOUS BRANCHES</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Greater auricular</td>
<td>C2, C3</td>
<td>Skin on ear; connective tissue capsule covering parotid gland</td>
</tr>
<tr>
<td>Lesser occipital</td>
<td>C2</td>
<td>Skin of scalp superior and posterior to ear</td>
</tr>
<tr>
<td>Supraclavicular</td>
<td>C3, C4</td>
<td>Skin on superior part of chest and shoulder</td>
</tr>
<tr>
<td>Transverse cervical</td>
<td>C2, C3</td>
<td>Skin on anterior part of neck</td>
</tr>
</tbody>
</table>

Note: Although CN XII (hypoglossal) and the phrenic nerve (C3, C4, C5) travel with the nerves of the cervical plexus, hypoglossal and phrenic are not considered part of this plexus.
Anterior rami: C5, C6, C7, C8, T1
Trunks: superior, middle, inferior
Anterior divisions
Posterior divisions
Cords: posterior, lateral, medial
Terminal branches

Nerve to subclavius
Superior trunk
Middle trunk
Lateral pectoral nerve
Subscapular nerves
Medial pectoral nerve
Musculocutaneous nerve
Median nerve
Axillary nerve
Radial nerve
Ulnar nerve

C5 vertebral
C6 vertebral
T1 vertebral

(a) Anterior view
(b) Right axilla, anterior view
(c) Right upper limb, anterior view

Figure 14.16 Brachial Plexus. Anterior rami of nerves C5–T1 form the brachial plexus, which innervates the upper limb. (a) Rami, trunks, divisions, and cords form the subdivisions of this plexus. (b) A cadaver photo shows major nerves from the brachial plexus. (c) Complete pathways of main brachial plexus branches are shown in an anterior view of the right upper limb. © McGraw-Hill Education/Christine Eckel
The lateral cord is lateral to the axillary artery and is formed from the anterior divisions of the superior and middle trunks; thus, it contains portions of nerves C5–C7.

Terminal Branches of the Brachial Plexus

Finally, five major terminal branches emerge from the three cords: the axillary nerve (from the posterior cord), median nerve (from the medial and lateral cords), musculocutaneous nerve (from the lateral cord), radial nerve (from the posterior cord), and ulnar nerve (from the medial cord). The nerves are compared in table 14.4.

The axillary nerve traverses through the axilla and posterior to the surgical neck of the humerus. The axillary nerve innervates both the deltoid and teres minor muscles (see section 11.8b). It receives sensory nerve signals from the superolateral part of the arm.

The median nerve extends along the midline of the arm and forearm, and deep to the carpal tunnel in the wrist. It innervates most of the anterior forearm muscles, the thenar muscles, and the lateral two lumbricals (see section 11.8e). It receives sensory nerve signals from the palmar side of the lateral 3½ fingers (thumb, index finger, middle finger, and the lateral half of the ring finger) and from the dorsal tips of these fingers.

The musculocutaneous (müs’kū-lō-kū-tā’nē-ūs) nerve innervates the anterior arm muscles (coracobrachialis, biceps brachii, and brachialis), which flex the humerus, flex the forearm, or both (see sections 11.8b and c). It also receives sensory information from the lateral surface of the forearm.

The radial nerve extends along the posterior side of the arm and then along the radial side of the forearm. The radial nerve innervates the posterior arm muscles (forearm extensors) and the posterior forearm muscles (extensors of the wrist and digits, and the supinator of the forearm, see sections 11.8c and d). It receives sensory nerve signals from the posterior arm and forearm surface and the dorsolateral side of the hand.

The ulnar nerve descends along the medial side of the arm. It extends posterior to the medial epicondyle of the humerus and then extends along the ulnar side of the forearm. It innervates some of the anterior forearm muscles (the medial region of the flexor digitorum profundus and all of the flexor carpi ulnaris). It also innervates most of the intrinsic hand muscles, including the hypothenar muscles, the palmar and dorsal interossi, and the medial two lumbricals (see section 11.8e). It receives sensory input from the skin of the dorsal and palmar aspects of the medial 1½ fingers (the pinky finger and the medial half of the ring finger).

The brachial plexus also gives off numerous other nerves that innervate portions of the upper limb and pectoral girdle. These branches are not as large as the terminal branches (table 14.4).

### Table 14.4 Branches of the Brachial Plexus

<table>
<thead>
<tr>
<th>Terminal Branch</th>
<th>Anterior Rami</th>
<th>Motor Innervation</th>
<th>Cutaneous Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axillary Nerve</td>
<td>C5, C6</td>
<td>Deltoïd (arm abductor)</td>
<td>Superolateral arm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Teres minor (lateral rotator of arm)</td>
<td></td>
</tr>
</tbody>
</table>

WHAT DID YOU LEARN?

19. What spinal nerves typically compose the brachial plexus?
20. Which nerve might you have damaged if you have difficulty abducting your arm and have anesthesia (lack of sensation) along the superolateral arm?
21. How do the ulnar nerve and radial nerve compare with respect to motor and cutaneous innervation?
<table>
<thead>
<tr>
<th>Terminal Branch</th>
<th>Anterior Rami</th>
<th>Motor Innervation</th>
<th>Cutaneous Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Median Nerve</strong></td>
<td>C6–T1</td>
<td>Most anterior forearm muscles (pronators, flexors of wrist, digits)</td>
<td>Palmar aspects and dorsal tips of lateral 3½ digits (thumb, index finger, middle finger, and ½ of ring finger)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flexor carpi radialis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flexor digitorum superficialis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pronator teres</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pronator quadratus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lateral ½ of flexor digitorum profundus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flexor pollicis longus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Thenar (thumb) muscles</strong> (move thumb)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flexor pollicis brevis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abductor pollicis brevis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Opponens pollicis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Lateral 2 lumbricals</strong> (flex MP joints and extend PIP and DIP joints)</td>
<td></td>
</tr>
<tr>
<td><strong>Musculocutaneous Nerve</strong></td>
<td>C5–C7</td>
<td>Anterior arm muscles (flex humerus, flex elbow joint, supinate forearm)</td>
<td>Lateral region of forearm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coracobrachialis</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Biceps brachii</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Brachialis</td>
<td></td>
</tr>
</tbody>
</table>
### Table 14.4  Branches of the Brachial Plexus (continued)

<table>
<thead>
<tr>
<th>Terminal Branch</th>
<th>Anterior Rami</th>
<th>Motor Innervation</th>
<th>Cutaneous Innervation</th>
</tr>
</thead>
</table>
| **Radial Nerve** | **C5–T1**     | **Posterior arm muscles** *(extend forearm)*  
Long head of triceps brachii | Lateral cord  
Posterior cord  
Medial cord  
Long head of triceps brachii  
Medial head of triceps brachii  
Anconeus  
Supinator  
Extensor carpi ulnaris  
Extensor digiti minimi  
Extensor digitorum  
Extensor pollicis longus  
Extensor pollicis brevis  
Abductor pollicis longus  
Extensor indicis  
**Brachioradialis** *(flexes forearm)* |
| **Posterior region of arm**  
**Posterior region of forearm**  
Dorsal aspect of lateral 3 digits (except their distal tips) |
| **Ulnar Nerve** | **C8–T1**     | **Anterior forearm muscles** *(flexors of wrist and digits)*  
Medial ½ of flexor digitorum profundus  
Medial 2 lumbricals | Lateral cord  
Posterior cord  
Medial cord  
Long head of triceps brachii  
Medial head of triceps brachii  
Anconeus  
Supinator  
Extensor carpi ulnaris  
Intrinsic hand muscles |
| **Dorsal and palmar aspects of medial 1½ digits (little finger; medial aspect of ring finger)** |

(continued on next page)
Brachial Plexus Injuries

Injuries to parts of the brachial plexus are fairly common, especially in individuals aged 15–25. Minor plexus injuries may be treated by simply resting the limb. More severe brachial plexus injuries may require nerve grafts or nerve transfers, and for very severe injuries, no effective treatment exists.

Axillary Nerve Injury

The axillary nerve can be compressed within the axilla, or it can be damaged if the surgical neck of the humerus is broken (recall that the axillary nerve extends posterior to the surgical neck of the humerus). A patient whose axillary nerve is damaged has great difficulty abducting the arm due to paralysis of the deltoid muscle, as well as anesthesia (loss of sensation) along the superolateral skin of the arm.

Radial Nerve Injury

The radial nerve is especially subject to injury during humeral shaft fractures or in injuries to the lateral elbow. Nerve damage results in paralysis of the extensor muscles of the forearm, wrist, and fingers. A common clinical sign of radial nerve injury is wrist drop, where the patient is unable to extend his or her wrist. The patient also experiences anesthesia along the posterior arm, the forearm, and the part of the hand normally supplied by this nerve.

Posterior Cord Injury

The posterior cord of the brachial plexus (which includes the axillary and radial nerves) may be injured by improper use of crutches, a condition called crutch palsy. Similarly, the posterior cord can also be compressed if a person drapes the upper limb over the back of a chair for an extended period of time. Because this can happen if someone passes out in a drunken stupor, this condition is also referred to as drunkenard's paralysis.

Median Nerve Injury

The median nerve may be impinged or compressed as a result of carpal tunnel syndrome (see Clinical View: 11.8 “Carpal Tunnel Syndrome”) or by any deep laceration of the wrist. Median nerve injury often results in paralysis of the thenar group of muscles. The classic sign of median nerve injury is the ape hand deformity, which develops over time as the thenar eminence wastes away until the hand eventually resembles that of an ape (apes lack well-developed thumb muscles). The lateral two lumbricals are also paralyzed, and sensation is lost in the part of the hand supplied by the median nerve.

Ulnar Nerve Injury

The ulnar nerve may be injured by fractures or dislocations of the elbow because of this nerve’s close proximity to the medial epicondyle of the humerus. When you “hit your funny bone,” you actually have hit your ulnar nerve. Most of the intrinsic hand muscles are paralyzed, so the person is unable to adduct or abduct the fingers. In addition, the person experiences sensory loss along the medial side of the hand. A clinician can test for ulnar nerve injury by having a patient hold a piece of paper tightly between the fingers as the doctor tries to pull it away. If the person has weak or paralyzed interossei muscles, the paper can be easily extracted.

Superior Trunk Injury

The superior trunk of the brachial plexus can be injured by excessive separation of the neck and shoulder, as when a person riding a motorcycle is flipped from the bike and lands on the side of the head. A superior trunk injury affects the C5 and C6 anterior rami, so any brachial plexus branch that has these nerves is also affected to some degree.

Inferior Trunk Injury

The inferior trunk of the brachial plexus can be injured if the arm is excessively abducted, as when a neonate’s arm is pulled too hard during delivery. In children and adults, inferior trunk injuries happen when grasping something above the head in order to break a fall—for example, grabbing a branch to keep from falling out of a tree. An inferior trunk injury involves the C8 and T1 anterior rami, so any brachial plexus branch that is formed from these nerves (such as the ulnar nerve) also is affected to some degree.

| Table 14.4 | Branches of the Brachial Plexus (continued) |
| --- | --- | --- | --- |
| Smaller Branches of the Brachial Plexus | Anterior Rami | Motor Innervation | Cutaneous Innervation |
| Dorsal scapular | C5 | Rhomboids, levator scapulae | |
| Long thoracic | C5–C7 | Serratus anterior | |
| Lateral pectoral | C5–C7 | Pectoralis major | |
| Medial pectoral | C8–T1 | Pectoralis major | Medial side of arm |
| Medial cutaneous nerve of arm | C8–T1 | Pectoralis minor | |
| Medial cutaneous nerve of forearm | C8–T1 | Medial side of forearm | |
| Nerve to subclavius | C5–C6 | Subclavius | |
| Suprascapular | C5–C6 | Supraspinatus, infraspinatus | |
| Subscapular nerves | C5–C6 | Subscapularis, teres major | |
| Thoracodorsal (nerve to latissimus dorsi) | C6–C8 | Latissimus dorsi | |
The left and right lumbar plexuses are formed from the anterior rami of spinal nerves L1–L4 located lateral to the L1–L4 vertebrae and along the psoas major muscle in the posterior abdominal wall (figure 14.17). This plexus innervates the inferior abdominal wall, anterior thigh, medial thigh, and skin of the medial leg. The lumbar plexus is structurally less complex than the brachial plexus. However, like the brachial plexus, the lumbar plexus is subdivided into an anterior division and a posterior division. The primary nerves of the lumbar plexus are listed in table 14.5.
### Table 14.5 Branches of the Lumbar Plexus

<table>
<thead>
<tr>
<th>Main Branch</th>
<th>Motor Innervation</th>
<th>Cutaneous Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Femoral Nerve</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L2–L4</td>
<td>Anterior thigh muscles</td>
<td>Anterior thigh</td>
</tr>
<tr>
<td></td>
<td>Anterior</td>
<td>Inferomedial thigh</td>
</tr>
<tr>
<td></td>
<td>Rectus femoris (extends knee)</td>
<td>Medial side of leg</td>
</tr>
<tr>
<td></td>
<td>Vastus lateralis</td>
<td>Most medial aspect of foot</td>
</tr>
<tr>
<td></td>
<td>Vastus intermedius</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vastus medialis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Iliopsoas (flexes hip)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sartorius (flexes hip and knee)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pectineus1 (flexes hip)</td>
<td></td>
</tr>
<tr>
<td><strong>Obturator Nerve</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L2–L4</td>
<td>Medial thigh muscles (adduct and flex hip)</td>
<td>Superomedial thigh</td>
</tr>
<tr>
<td></td>
<td>Adductors</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gracilis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pectineus1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Obturator externus (laterally rotates thigh)</td>
<td></td>
</tr>
</tbody>
</table>

1. Pectineus may be innervated by the femoral nerve, obturator nerve, or branches from both nerves.
The main nerve of the posterior division of the lumbar plexus is the femoral nerve. This nerve innervates the anterior thigh muscles, such as the quadriceps femoris (knee extensor) and the sartorius, psoas, and iliacus (hip flexors, see sections 11.9a and b). It also receives sensory input from the skin of the anterior and inferomedial thigh as well as the medial aspect of the leg.

The main nerve of the anterior division is the obturator nerve, which extends through the obturator foramen of the os coxae to the medial thigh. There, the nerve innervates the medial thigh muscles (which adduct the thigh, see section 11.9a) and conducts sensory input from the superomedial skin of the thigh. Smaller branches of each lumbar plexus innervate the abdominal wall, portions of the external genitalia, and the inferior portions of the abdominal muscles (table 14.5; see also section 11.6).

What did you learn?

Which nerve of the lumbar plexus might you have damaged if you have difficulty extending your knee?

14.5g Sacral Plexuses

Learning Objectives

31. List the spinal nerves that form the sacral plexus.

32. Describe the composition of the sciatic nerve, and compare its branches.

The left and right sacral plexuses are formed from the anterior rami of spinal nerves L4–S4 and are located immediately inferior to the lumbar plexuses (figure 14.18). The lumbar and sacral plexuses are sometimes considered together as the lumbosacral plexus. The nerves emerging from a sacral plexus innervate the gluteal region, pelvis, perineum, posterior thigh, and almost all of the leg and foot.

The anterior rami of the sacral plexus are organized into an anterior division and a posterior division. The nerves that are formed from the anterior division tend to innervate muscles that flex (or plantar flex) parts of the lower limb, whereas the posterior division nerves tend to innervate muscles that extend (or dorsiflex) part of the lower limb. Table 14.6 lists the main and smaller nerves of the sacral plexus.

The sciatic (sī-āt′i-k) nerve, also known as the ischiadic (is-ki′-āt′ik; hip joint) nerve, is the largest and longest nerve in the body. It is formed from portions of both the anterior and posterior divisions of the sacral plexus. This nerve projects from the pelvis through the greater sciatic notch of the os coxae and extends into the posterior region of the thigh. The sciatic nerve is actually composed of two divisions—the tibial division and the common fibular division—wrapped in a common sheath.

Just superior to the popliteal fossa, the two divisions of the sciatic nerve split into two nerves. The tibial nerve is formed from the anterior divisions of the sciatic nerve. In the posterior thigh, the tibial division of the sciatic nerve innervates the hamstrings (except for the short head of the biceps femoris) and the hamstring part of the adductor magnus. It extends within the posterior compartment of the leg, where it innervates the plantar flexors of the foot and the toe flexors (see sections 11.9c and d). In the foot, the tibial nerve splits into the lateral and medial plantar nerves, which innervate the plantar muscles of the foot and receive sensory input from the skin covering the sole of the foot.

The common fibular (common peroneal) nerve is formed from the posterior division of the sciatic nerve. As the common fibular division of the sciatic nerve, it innervates the short head of the biceps femoris muscle (see section 11.9b). Along the lateral knee, as it wraps around the neck of the fibula, this nerve nerve splits into two main branches: the deep fibular nerve and the superficial fibular nerve.

### Table 14.6 Brachial Plexuses (continued)

<table>
<thead>
<tr>
<th>Smaller Branches of the Lumbar Plexus</th>
<th>Anterior Rami</th>
<th>Motor Innervation</th>
<th>Cutaneous Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ilioinguinal</td>
<td>L1</td>
<td>Partial innervation to abdominal muscles (flex vertebral column)</td>
<td>Superior lateral gluteal region</td>
</tr>
<tr>
<td>Genitofemoral</td>
<td>L1, L2</td>
<td>Partial innervation to abdominal muscles (flex vertebral column)</td>
<td>Inferior abdominal wall</td>
</tr>
<tr>
<td>Lateral femoral cutaneous</td>
<td>L2, L3</td>
<td></td>
<td>Small area in anterior superior thigh</td>
</tr>
</tbody>
</table>

### CLINICAL VIEW 14.6

Sacral Plexus Injuries

Some branches of the sacral plexus are readily subject to injury. For example, a poorly placed gluteal intramuscular injection can injure the superior or inferior gluteal nerves, and in some cases even the sciatic nerve. Additionally, a herniated intervertebral disc may impinge on the nerve branches that form the sciatic nerve. Injury to the sciatic nerve produces a condition known as sciatica (sī-at′i-kā), which is characterized by pain down the posterior of the thigh and leg.

The common fibular nerve is especially prone to injury due to fracture of the neck of the fibula or compression from a leg cast that is too tight. The anterior and lateral leg muscles may be paralyzed and leave the person unable to dorsiflex and evert the foot. One classic sign of fibular nerve injury is foot drop. Because the person cannot dorsiflex the foot to walk normally, he or she compensates by flexing the hip to lift the affected area and keep from tripping or stubbing the toes.
The **deep fibular (deep peroneal) nerve** extends through the anterior compartment of the leg and terminates between the first and second toes. It innervates the anterior leg muscles (which dorsiflex the foot and extend the toes) and the muscles on the dorsum of the foot (which extend the toes, see sections 11.9c and d). In addition, this nerve receives sensory input from the skin between the first and second toes on the dorsum of the foot.

The **superficial fibular (superficial peroneal) nerve** extends through the lateral compartment of the leg. Just proximal to the ankle, this nerve becomes superficial along the anterior part of the ankle and dorsum of the foot. The superficial fibular nerve innervates the lateral compartment muscles of the leg (foot evertors and weak plantar flexors, see sections 11.9c and d). It also receives sensory input from most of the dorsal surface of the foot and the anteroinferior part of the leg.

---

**WHAT DID YOU LEARN?**

What anterior rami form the sacral nerve plexus, and what general areas of the body does it innervate?
<table>
<thead>
<tr>
<th>Main Branch</th>
<th>Anterior Rami</th>
<th>Motor Innervation</th>
<th>Cutaneous Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sciatic Nerve</strong></td>
<td>L4–S3</td>
<td>(See tibial and common fibular nerves)</td>
<td>(See tibial and common fibular nerves)</td>
</tr>
<tr>
<td>(Composed of tibial and common fibular divisions wrapped in a common sheath)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Tibial Nerve</strong></td>
<td>L4–S3</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Common Fibular Nerve</strong></td>
<td>L4–S2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 14.6 Branches of the Sacral Plexus

<table>
<thead>
<tr>
<th>Branches of the Sacral Plexus</th>
<th>Motor Innervation</th>
<th>Cutaneous Innervation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Posterior thigh muscles</strong></td>
<td><em>extend hip and flex knee</em></td>
<td></td>
</tr>
<tr>
<td><strong>Long head of biceps femoris</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Semitendinosus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Semitendinosus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Part of adductor magnus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Posterior leg muscles</strong></td>
<td><em>flex knee and plantar flex foot</em></td>
<td></td>
</tr>
<tr>
<td><strong>Flexor digitorum longus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Flexor hallucis longus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Gastrocnemius</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Soleus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Popliteus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Tibialis posterior</strong></td>
<td><em>inverts foot</em></td>
<td></td>
</tr>
<tr>
<td><strong>Plantar foot muscles</strong></td>
<td><em>via medial and lateral plantar nerve branches</em></td>
<td></td>
</tr>
</tbody>
</table>

Branches to the heel, and via its medial and lateral plantar nerve branches (which supply the sole of the foot)

See deep fibular and superficial fibular nerves

(continued on next page)
<table>
<thead>
<tr>
<th>Table 14.6 Branches of the Sacral Plexus (continued)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main Branch</strong></td>
</tr>
</tbody>
</table>
| Deep Fibular Nerve                                 | L4–S1             | *Anterior leg muscles* *(dorsiflex foot, extend toes)*  
Tibialis anterior *(inverts foot)*  
Extensor hallucis longus  
Extensor digitorum longus  
Fibularis tertius  
**Dorsum foot muscles** *(extend toes)*  
Extensor hallucis brevis  
Extensor digitorum brevis | Dorsal interspace between first and second toes |
| Superficial Fibular Nerve                          | L5–S2             | *Lateral leg muscles* *(evert foot and weakly plantar flex foot)*  
Fibularis longus  
Fibularis brevis | Anteroinferior part of leg; most of dorsum of foot |
| Smaller Branches of the Sacral Plexus              |                   |                                             |                                             |
| Inferior gluteal nerve                             | L5–S2             | *Gluteus maximus* *(extends thigh)* |                                             |
| Superior gluteal nerve                             | L4–S1             | *Gluteus medius, gluteus minimus, and tensor fasciae latae* *(abduct thigh)* |                                             |
| Posterior femoral cutaneous nerve                  | S1–S3             |                                             | *Skin on posterior thigh*                     |
| Pudendal nerve                                     | S2–S4             | *Muscles of perineum, external anal sphincter, external urethral sphincter* | *Skin on external genitalia*                   |
14.6 Reflexes

Here we consider the characteristics of a reflex, the components of a reflex arc, the classification of reflexes, the different spinal reflexes, and how reflexes are tested in a clinical setting.

14.6a Characteristics of Reflexes

LEARNING OBJECTIVES

33. Describe the properties of a reflex.
34. Explain the general function of a reflex.

Reflexes are rapid, preprogrammed, involuntary responses of muscles or glands to a stimulus. An example of a reflex occurs when you accidentally touch a hot burner on a stove. Instantly and automatically, you remove your hand from the stimulus (the hot burner), even before you are completely aware that your hand was touching something extremely hot.

All reflexes have similar properties:

- A stimulus is required to initiate a reflex.
- A rapid response requires that few neurons are involved and synaptic delay (see section 12.3) is minimal.
- A preprogrammed response occurs the same way every time.
- An involuntary response requires no conscious intent or preawareness of the reflex activity. Thus, reflexes are usually not suppressed.

A reflex is a survival mechanism; it allows us to quickly respond to a stimulus that may be detrimental to our well-being without having to wait for the brain to process the information. Awareness of the stimulus occurs after the reflex action has been completed, in time to correct or avoid a potentially dangerous situation. (This is possible because sensory input has reached the cerebral cortex.)

WHAT DID YOU LEARN?

24. What are the four main properties of a reflex?

Figure 14.19 Simple Reflex Arcs. A reflex arc is a neural pathway composed of neurons that control rapid, subconscious, preprogrammed responses to a stimulus.

14.6b Components of a Reflex Arc

LEARNING OBJECTIVE

35. List the structures involved in a reflex arc and the steps in its action.

A reflex arc includes a sensory receptor, an effector, and the neural wiring between the two. It always begins at a receptor in the PNS, communicates with the CNS, and ends at a peripheral effector, either a muscle or a gland. The number of intermediate steps varies, depending upon the complexity of the reflex. Generally, five steps are involved in a reflex, as illustrated in figure 14.19 and described here:

1. A stimulus activates a sensory receptor. A sensory receptor (dendritic endings of a sensory neuron or specialized receptor cells) responds to external and internal stimuli, such as temperature, pressure, or tactile changes. Proprioceptors are sensory receptors found in muscles and tendons, and a stimulus to a proprioceptor (such as the tapping of tendon) may initiate a reflex as well.

2. The sensory neuron transmits a nerve signal to the CNS. A sensory neuron transmits a nerve signal from the receptor to the spinal cord (or brain).

3. Information from the nerve signal is processed in the integration center by interneurons. More complex reflexes may use a number of interneurons within the CNS to integrate and process incoming sensory nerve signals and transmit information to the motor neuron. The simplest reflexes do not involve interneurons; rather, the sensory neuron synapses directly on a motor neuron in the CNS.

4. The motor neuron transmits a nerve signal from the CNS to an effector. A motor neuron transmits a nerve signal from the CNS to a peripheral effector organ—a gland or a muscle.

5. The effector responds to the nerve signal from the motor neuron. An effector is a muscle or a gland that responds to the nerve signal from the motor neuron. This response is intended to counteract or remove the original stimulus.
WHAT DID YOU LEARN?
25 Identify (in order) the five steps of a reflex arc.

14.6c Classifying Spinal Reflexes

LEARNING OBJECTIVE
36. Explain the five ways a reflex may be classified.

The specific components (or attributes) of a reflex can vary. Some reflexes involve the spinal cord, whereas others involve the brain. Some involve skeletal muscle and some involve other muscle types or glands. Some have only two neurons and some have more. We describe five different ways of classifying a reflex:

- **Spinal reflex or cranial reflex.** A reflex may be identified by the specific area of the central nervous system (integration center) that serves as the processing site. Spinal reflexes involve the spinal cord, whereas cranial reflexes involve the brain.

- **Somatic reflex or visceral reflex.** This classification criterion is determined by the type of effector that is stimulated by the motor neurons involved in the reflex. Somatic reflexes involve skeletal muscle as the effector. Visceral (or autonomic) reflexes involve cardiac muscle, smooth muscle, or a gland as the effector.

- **Monosynaptic reflex or polysynaptic reflex.** A reflex may also be classified by the number of neurons participating in the reflex. A monosynaptic (mon′-ə-si-nap′tik; monos = single) reflex has only a sensory neuron and a motor neuron (figure 14.20). The axon of the sensory neuron synapses directly on the motor neuron, whose axon projects to the effector. Thus, there is only one synapse between neurons. Monosynaptic reflexes are the simplest, and they are the most rapid. With only one synaptic delay, the response is very prompt. A polysynaptic (pol′-ə-si-nap′tik; polys = many) reflex has one or more interneurons positioned between the sensory and the motor neuron. These reflex arcs are more complicated and not as rapid.

- **Ipsilateral reflex or contralateral reflex.** The reflex may also be classified based upon whether it involves only one side of the body. An ipsilateral reflex is a reflex in which both the receptor and effector organs are on the same side of the spinal cord. A contralateral reflex is a reflex that involves an effector on the opposite side of the body from the receptor that detected the stimulus. Note that this terminology is only applicable to reflexes that involve the limbs. For example, an ipsilateral effect occurs when the muscles in your left arm contract to pull your left hand away from a hot object. In comparison, a contralateral effect occurs when you step on a sharp object with your left foot and then contract the muscles in your right leg to maintain balance as you withdraw your left leg from the damaging object.

- **Innate reflex or acquired reflex.** The reflex may be classified based upon whether you are born with it. An innate reflex is a reflex that you are born with, whereas an acquired reflex is one that is developed after birth.

As you read about the reflexes described in this section, see if you can determine how each would be classified based upon these five criteria. Keep in mind that there may be instances where you do not have enough information to classify the reflex in all five ways. All of the reflexes discussed in section 14.6d are spinal reflexes with skeletal muscle as the effector. Thus, these reflexes are classified as both spinal reflexes and somatic reflexes.

**Figure 14.20 Monosynaptic and Polysynaptic Reflexes.** The minimal number of neurons and the pathways of a monosynaptic reflex (left) are compared to those of a polysynaptic reflex (right).
14.6d Spinal Reflexes

**LEARNING OBJECTIVE**

37. Name and describe four common spinal reflexes.

Four common spinal reflexes, which involve skeletal muscle as the effector, include the stretch reflex, the Golgi tendon reflex, the withdrawal (flexor) reflex, and the crossed-extensor reflex. These reflexes can be initiated by proprioceptors or pain receptors (nociceptors).

Those reflexes involving proprioceptors include stretch reflexes and Golgi tendon reflexes. Recall from section 14.4a that a **proprioceptor** resides in a joint, muscle, or tendon. These sensory receptors specifically detect any change to that structure, such as change in stretch or tension associated with muscle contraction and muscle stretching.

A **muscle spindle** is a proprioceptor that detects changes in stretch within a muscle; for this reason, a muscle spindle is also known as a **stretch receptor** (figure 14.21). A muscle spindle is composed of **intrafusal muscle fibers** surrounded by a connective tissue capsule. These intrafusal muscle fibers lack myofilaments in their central regions and are contractile only at their distal regions. (Actin and myosin are found only at the ends of these fibers.) These muscle fibers are innervated by both sensory neurons (which relay nerve signals to the spinal cord) and **gamma (γ) motor neurons**, so named because gamma refers to motor neurons with small-diameter axons. Gamma motor neurons stimulate the contractile fibers at the distal ends of the intrafusal muscle fibers to contract, which elongates the inner portion of the muscle spindle fiber, causing the muscle spindle to be more sensitive to any additional stretch. (Thus, the gamma motor neurons function prior to the reflex to increase the sensitivity of a muscle to stretch.)

Around the muscle spindle are **extrafusal muscle fibers**, which are innervated by **alpha (α) motor neurons**, so named because these motor neurons have the largest diameter axons. (One study tip to remember the difference between the two neuron types is that gamma goes within the muscle, and alpha wraps around the muscle.) A muscle spindle is associated with a type of reflex called a stretch reflex. Alpha motor neurons stimulate the extrafusal muscle fiber of a skeletal muscle to contract.

**Stretch Reflex**

The **stretch reflex** is a reflex that is initiated by a muscle spindle proprioceptor and involves a muscle reflexively contracting in response to stretching of a muscle (figure 14.21). When the muscle spindle is stretched, the sensation is detected by sensory neurons that are wrapped around the intrafusal muscle fibers of the muscle spindle. The sensory neurons transmit nerve signals to the spinal cord (CNS), where they synapse with the alpha motor neurons associated with that muscle. The alpha motor neurons then transmit nerve signals to the extrafusal muscle fibers, which causes the muscle to contract and thus resist the stretch.

The triceps reflex is an example of a stretch reflex. The triceps reflex may be classified as follows: It is a spinal reflex (it involves the spinal cord as the integration center), a somatic reflex (skeletal muscle is the effector), a monosynaptic reflex (involves no interneurons), and

---

**Figure 14.21 Stretch Reflex.** A stretch reflex is a simple monosynaptic reflex. A stretching force detected by a muscle spindle results in the contraction of that muscle. Conversely, antagonistic muscle contraction is dampened, in a process called reciprocal inhibition.
an ipsilateral reflex (the receptor and effector are located on the same side of the spinal cord) and is innate (we are born with this reflex). The stimulus (e.g., the reflex hammer tap on the triceps brachii tendon) stretches the muscle spindle in the triceps brachii muscle. Sensory neurons transmit nerve signals to the spinal cord, where they synapse with the alpha motor neurons. The alpha motor neurons transmit nerve signals to the extrafusal muscle fibers in triceps brachii, thereby initiating contraction of the muscle and extending the elbow joint.

Note in figure 14.21 that a stretch reflex also is indirectly involved in a process called reciproc al inhibition. When the sensory nerve signals reach the spinal cord, some of the sensory axons synapse with interneurons. These interneurons synapse with alpha motor neurons that inhibit antagonistic muscle contraction. In the case of the triceps reflex, the biceps brachii muscle is the inhibited antagonistic muscle. Thus, as the triceps brachii is stimulated, reciprocal inhibition results in the biceps brachii contraction being dampened, so the triceps movement will not be opposed by the biceps brachii.

The stretch reflex is a monosynaptic reflex, but the corresponding reciprocal inhibition is polysynaptic, because it uses an interneuron within the circuit.

**Golgi Tendon Reflex**

A **Golgi tendon reflex** is a reflex that is initiated by a Golgi tendon organ proprioceptor. A **Golgi tendon organ** is composed of sensory nerve endings within a tendon or near a muscle-tendon junction and detects change in tension (stretch) in a muscle tendon when a muscle contracts. Whereas the stretch reflex prevents muscles from stretching excessively, the Golgi tendon reflex prevents muscles from doing the opposite: tensing or contracting excessively. The Golgi tendon reflex is a polysynaptic reflex that results in muscle relaxation in response to increased tension at a Golgi tendon organ (figure 14.22).

As a muscle contracts, its associated tendon stretches, resulting in increased tension in the tendon and activation of the Golgi tendon organ. Sensory neurons in the Golgi tendon organ transmit nerve signals to interneurons in the spinal cord, which in turn inhibit the alpha motor neurons in the same muscle. When the motor neurons are inhibited, the associated muscle is allowed to relax, thus protecting the muscle and tendon from excessive tension damage.

Note that the sensory neurons also communicate with other interneurons in the spinal cord that stimulate alpha motor neurons for the antagonistic muscles. This process is called **reciprocal activation**.

**Figure 14.22 Golgi Tendon Reflex.** A Golgi tendon reflex is a polysynaptic reflex. A contraction force detected by a Golgi tendon organ (within the tendon of the muscle) results in relaxation of that muscle. Conversely, antagonistic muscles are stimulated to contract, a process called reciprocal activation.
So, for example, if a Golgi tendon organ in the quadriceps femoris muscle detects excessive tension, then the Golgi tendon reflex ultimately relaxes the quadriceps femoris muscle, and reciprocal activation results in the hamstrings being stimulated to contract. 

Golgi tendon reflexes help protect a muscle or tendon from injury due to excessive tension and help ensure that the muscle contraction process occurs smoothly and efficiently. In cases where the muscle is under extreme tension (such as when one lifts a very heavy weight), a Golgi tendon reflex may nullify a stretch reflex (and thus the person drops the weight due to the excessive tension on the muscle). Thus, weightlifters are encouraged to have “spotters” who can help prevent the heavy weight from dropping unexpectedly.

**Withdrawal Reflex**

A withdrawal (flexor) reflex involves muscles contracting to withdraw the body part away from a painful stimulus. This reflex involves pain receptors, termed nociceptors (see section 16.1d). It is initiated by a painful stimulus, such as touching something very hot or painful (figure 14.23). This stimulation initiates a nerve signal that is transmitted by a sensory neuron to the spinal cord. Interneurons receive the sensory nerve signal and stimulate motor neurons to the flexor muscles. These flexor muscles contract in response.

For example, when you step on a sharp object, sensory neurons detect the sensation and transmit nerve signals to the spinal cord. They synapse with interneurons, which stimulate motor neurons to contract the flexor muscles (in this case, the hamstrings of the lower limb). You lift the lower limb up and away from the painful stimulus. In addition, reciprocal inhibition occurs with the extensor (quadriceps) muscles of the same leg, so the hamstrings can contract unimpeded. (Reciprocal inhibition is not shown in figure 14.23.)

**Crossed-Extensor Reflex**

The crossed-extensor reflex often occurs in conjunction with the withdrawal reflex, usually in the lower (weight-bearing) limbs (figure 14.23). In essence, when the withdrawal reflex is occurring in...
Table 14.7

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Spinal Nerve Segments Tested</th>
<th>Normal Action of Effector</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps reflex</td>
<td>C5, C6</td>
<td>Flexes elbow when biceps brachii tendon is tapped</td>
</tr>
<tr>
<td>Triceps reflex</td>
<td>C6, C7</td>
<td>Extends elbow when triceps brachii tendon is tapped</td>
</tr>
<tr>
<td>Abdominal reflexes</td>
<td>T8–T12</td>
<td>Contract abdominal muscles when one side of the abdominal wall is briskly stroked</td>
</tr>
<tr>
<td>Cremasteric reflex</td>
<td>L1, L2</td>
<td>Elevates testis (due to contraction of cremaster muscle in scrotum) when medial side of thigh is briskly stroked</td>
</tr>
<tr>
<td>Patellar (knee-jerk) reflex</td>
<td>L2–L4</td>
<td>Extends knee when patellar ligament is tapped</td>
</tr>
<tr>
<td>Ankle (Achilles) reflex</td>
<td>S1</td>
<td>Plantar flexes ankle when calcaneal tendon is tapped</td>
</tr>
<tr>
<td>Plantar reflex</td>
<td>L5, S1</td>
<td>Flexes toes when plantar side of foot is briskly stroked</td>
</tr>
</tbody>
</table>

1. This is the normal reflex response in adults; in adults with spinal cord damage and in normal infants, the Babinski sign occurs, which is extension of the great toe and fanning of the other toes.
14.7 Development of the Spinal Cord

**Learning Objective**

39. Describe how the neural tube forms the gray matter structures in the spinal cord.

Recall from section 13.1b that the caudal (inferior) part of the neural tube forms the spinal cord. As the caudal part of the neural tube differentiates and specialization, the spinal cord begins to develop (figure 14.24). However, this developmental process is much less complex than that of the brain. A hollow neural canal in the neural tube develops into the central canal of the spinal cord. Note that the neural canal doesn’t shrink in size; rather, the neural tube around it grows at a rapid rate. Thus, as the neural tube walls grow and expand, the neural canal in the newborn appears as a tiny channel called the central canal.

During the fourth and fifth weeks of embryonic development, the neural tube starts to grow rapidly and unevenly. Part of the neural tube forms the outer white matter of the spinal cord, whereas other components form the inner gray matter. By the sixth week of development, a horizontal groove called the sulcus limitans (lim‘i-tanz; limes = boundary) forms in the lateral walls of the central canal (figure 14.24). The sulcus limitans also represents a dividing point in the neural tube as two specific regions become evident: the basal plates and alar plates.

The basal plates lie anterior to the sulcus limitans. The basal plates develop into the anterior and lateral horns, motor structures of the gray matter. They also form the anterior part of the gray commissure.

The alar (əˈlär; əla = wing) plates lie posterior to the sulcus limitans. By about the ninth week of development, the alar plates develop into posterior horns, sensory structures of the gray matter. They also form the posterior part of the gray commissure.

**Figure 14.24 Spinal Cord Development.** The spinal cord begins development as a tubular extension of the brain. (a) A cross section shows the structures of the neural tube of an embryo in week 4 of development. Transverse sections show (b) the formation of the basal and alar plates at week 6 and (c) the developing spinal cord at week 9.
During the embryonic period, the spinal cord extends the length of the vertebral canal. However, during the fetal period, growth of the vertebral column (and its vertebral canal) outpaces that of the spinal cord. By the sixth fetal month, the spinal cord is at the level of the S1 vertebra; in contrast, a newborn’s spinal cord ends at about the L2 vertebra. By adulthood, the spinal cord length extends only to the level of the L1 vertebra. This disproportionate growth explains why the lumbar, sacral, and coccygeal parts of the spinal cord and its associated nerve roots do not lie next to their respective vertebrae (as described in section 14.1).

### Chapter Summary

<table>
<thead>
<tr>
<th>Section</th>
<th>Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>14.1 Overview of the Spinal Cord and Spinal Nerves</td>
<td>• The spinal cord is part of the central nervous system (CNS) and has 31 pairs of spinal nerves that extend from it.</td>
</tr>
<tr>
<td>14.1a General Functions</td>
<td>• The spinal cord and spinal nerves serve as a pathway for sensory and motor nerve signals and are responsible for reflexes.</td>
</tr>
<tr>
<td>14.1b Spinal Cord Gross Anatomy</td>
<td>• The adult spinal cord traverses the vertebral canal and typically ends at the level of the L1 vertebra.</td>
</tr>
<tr>
<td>14.1c Spinal Nerve Identification and Gross Anatomy</td>
<td>• There are 31 pairs of spinal nerves: 8 pairs of cervical nerves, 12 pairs of thoracic nerves, 5 pairs of lumbar nerves, 5 pairs of sacral nerves, and 1 pair of coccygeal nerves.</td>
</tr>
<tr>
<td>14.2 Protection and Support of the Spinal Cord</td>
<td>• Protection and support of the spinal cord includes the bony vertebral column, meninges, and cerebrospinal fluid (CSF). • The meninges include the pia mater, arachnoid mater, and dura mater. • A potential subdural space lies between the dura mater and arachnoid mater, the epidural space is external to the dura mater and contains adipose connective tissue and blood vessels, and the subarachnoid space lies between the arachnoid mater and pia mater and contains cerebrospinal fluid (CSF).</td>
</tr>
<tr>
<td>14.3 Sectional Anatomy of the Spinal Cord and Spinal Roots</td>
<td>• Gray matter is centrally located and composed of neuron cell bodies, dendrites, unmyelinated axons, and glial cells. • White matter is peripherally located and composed of myelinated axons.</td>
</tr>
<tr>
<td>14.3a Distribution of Gray Matter</td>
<td>• Gray matter is composed of three horns: posterior (contains sensory axons and interneurons), anterior (contains cell bodies of somatic motor neurons), and lateral (contains cell bodies of autonomic motor neurons).</td>
</tr>
<tr>
<td>14.3b Distribution of White Matter</td>
<td>• White matter is organized into three pairs of funiculi, most of which contain sensory (ascending) tracts and motor (descending) tracts.</td>
</tr>
<tr>
<td>14.4 Sensory and Motor Pathways</td>
<td>• The central nervous system (CNS) communicates with the body through conduction pathways that extend through the spinal cord.</td>
</tr>
<tr>
<td>14.4a Overview of Conduction Pathways</td>
<td>• Sensory pathways transmit ascending information from sensory receptors to the CNS, whereas motor pathways transmit descending information from the brain to muscles and glands.</td>
</tr>
<tr>
<td>14.4b Sensory Pathways</td>
<td>• Sensory pathways use primary, secondary, and tertiary neurons. • The posterior funiculus–medial lemniscal pathway transmits stimuli of fine proprioception, discriminative touch, precise pressure, and proprioception to the cerebrum (parietal lobe). • The anterolateral pathway transmits stimuli related to crude touch, pressure, pain, and temperature to the cerebrum (parietal lobe). • The spinocerebellar pathway transmits stimuli from proprioceptors to the cerebellum.</td>
</tr>
<tr>
<td>14.4c Motor Pathways</td>
<td>• Motor pathways use upper and lower motor neurons. • Somatic motor commands extend through either the direct system (for conscious control) or the indirect system (for unconscious control). • The direct system that extends through the spinal cord consists of the corticospinal tracts. • The indirect system consists of the lateral pathway (rubrospinal tracts) and the medial pathway (reticulospinal, tectospinal, and vestibulospinal tracts).</td>
</tr>
<tr>
<td>14.5 Spinal Nerves</td>
<td>• A spinal nerve is formed from the union of an anterior root and a posterior root.</td>
</tr>
<tr>
<td>14.5a General Distribution of Spinal Nerves</td>
<td>• Spinal nerves have two branches: A posterior ramus innervates the skin and deep muscles of the back, and an anterior ramus innervates the skin and muscle of the anterior and lateral portions of the trunk and the limbs.</td>
</tr>
<tr>
<td>14.5b Nerve Plexuses</td>
<td>• A nerve plexus is a network of interwoven anterior rami. Nerve plexuses occur in pairs.</td>
</tr>
<tr>
<td>14.5c Intercostal Nerves</td>
<td>• The anterior rami of spinal nerves T1–T11 do not form a plexus, but rather form the intercostal nerves. Nerve T12 is called a subcostal nerve.</td>
</tr>
<tr>
<td>14.5d Cervical Plexuses</td>
<td>• Each cervical plexus is formed from the anterior rami of C1–C4 spinal nerves. It innervates the anterior neck muscles and the skin along the neck and shoulders.</td>
</tr>
</tbody>
</table>

**WHAT DID YOU LEARN?**

What structures develop from the alar plates, and what structures develop from the basal plates?
### Chapter Summary (continued)

14.5 Spinal Nerves (continued)

- **14.5e Brachial Plexuses**
  - Each brachial plexus is formed from the anterior rami of spinal nerves C5–T1. It innervates an upper limb.

- **14.5f Lumbar Plexuses**
  - Each lumbar plexus is formed from the anterior rami of spinal nerves L1–L4. It innervates the anterior and medial thigh, the lower abdominal wall, and the skin of the medial leg.

- **14.5g Sacral Plexuses**
  - Each sacral plexus is formed from the anterior rami of spinal nerves L4–S4. It innervates most of the lower limb as well as the perineum.

14.6 Reflexes

- A reflex is a rapid, preprogrammed response of muscles or glands to a stimulus.

- **14.6a Characteristics of Reflexes**
  - A reflex is a rapid, preprogrammed, involuntary response to a stimulus.

- **14.6b Components of a Reflex Arc**
  - The five steps of a reflex are (1) activation of a receptor by a stimulus, (2) nerve signal propagation along a sensory neuron to the CNS, (3) integration and processing of information by interneurons, (4) nerve signal propagation along a motor neuron, and (5) effector response.

- **14.6c Classifying Spinal Reflexes**
  - Reflexes may be classified as to whether they are (a) spinal or cranial, (b) somatic or visceral, (c) monosynaptic or polysynaptic, (d) ipsilateral or contralateral, and (e) innate or acquired.

- **14.6d Spinal Reflexes**
  - A stretch reflex is monosynaptic and contracts the muscle in response to increased stretch in a muscle spindle.
  - A Golgi tendon reflex is polysynaptic and prevents muscles from tensing excessively.
  - A withdrawal reflex is polysynaptic and activates flexor muscles to immediately remove a body part from a painful stimulus.
  - A crossed-extensor reflex stimulates the extensor muscles in the opposite limb, in response to the withdrawal reflex.

- **14.6e Reflex Testing in a Clinical Setting**
  - Reflex testing can help diagnose nervous system or muscular disorders.
  - Hypoactive reflexes, in which the response is diminished, may indicate spinal cord damage or muscle pathology.
  - Hyperactive reflexes, in which the response is abnormally strong, may indicate damage to the brain or spinal cord.

14.7 Development of the Spinal Cord

- The neural tube forms basal plates and alar plates.
- Basal plates form the anterior horns, lateral horns, and anterior half of the gray commissure.
- Alar plates form the posterior horns and the posterior half of the gray commissure.

### Challenge Yourself

**Do You Know the Basics?**

1. Identify the meningeal layer immediately deep to the subdural space.
   - a. arachnoid mater
   - b. pia mater
   - c. dura mater
   - d. epidural space

2. The anterior root of a spinal nerve contains
   - a. axons of both motor and sensory neurons.
   - b. axons of sensory neurons only.
   - c. interneurons.
   - d. axons of motor neurons only.

3. Where are tertiary neurons found?
   - a. extending between the posterior horn and anterior horn
   - b. extending between the posterior horn and the brainstem
   - c. extending between the thalamus and the primary somatosensory cortex
   - d. extending between the primary motor cortex and brainstem

4. Which of the following is an example of a sensory pathway?
   - a. reticulospinal tract
   - b. spinocerebellar tract
   - c. corticobulbar tract
   - d. tectospinal tract

5. The radial nerve originates from the ___________ plexus.
   - a. cervical
   - b. brachial
   - c. lumbar
   - d. sacral

6. Which structure sends motor nerve signals to the deep back muscles and receives sensory nerve signals from the skin of the back?
   - a. posterior root
   - b. posterior ramus
   - c. anterior root
   - d. anterior ramus
7. Which statement is accurate about intercostal nerves?
   a. They are formed from the posterior rami of spinal nerves.
   b. They form a thoracic plexus of nerves.
   c. They originate from the thoracic part of the spinal cord.
   d. They innervate the lower limb.

8. The __________ nerve innervates the anterior thigh muscles and the skin on the anterior thigh.
   a. femoral
   b. obturator
   c. sciatic
   d. tibial

9. A __________ reflex is monosynaptic and responds to stretching in a muscle spindle.
   a. withdrawal
   b. crossed-extensor
   c. Golgi tendon
   d. stretch

10. Which statement is correct about reflexes?
    a. The patellar reflex tests the S1–S3 spinal nerve segments.
    b. A hypoactive reflex may indicate damage to the neuromuscular junction.
    c. The more hyperactive the reflex, the healthier the individual.
    d. A normal biceps reflex response is extension of the elbow when the biceps tendon is tapped.

11. Identify the spinal cord parts, which spinal nerves are associated with them, and their relationship to the corresponding vertebrae.

12. List the three gray matter horns on each side of the spinal cord, and discuss the neuronal composition of each. In addition, list which types of nuclei (motor or sensory) are located in each horn.

13. Compare the main differences between the posterior funiculus–medial lemniscal pathway and the anterolateral pathway.

14. Describe the location and function of upper and lower motor neurons in the motor pathways.

15. What are the main terminal branches of the brachial plexus, and what muscles do these terminal branches innervate?

16. What anterior rami form the lumbar plexus, and what in general does this plexus innervate?

17. What muscles do the tibial and common fibular nerves innervate?

18. What are the five basic steps involved in a reflex arc?

19. What are the differences between a stretch and Golgi tendon reflex?

20. Where are the basal and alar plates of the neural tube located, and what does each form?

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**Can You Apply What You’ve Learned?**

Use the following paragraph to answer questions 1–3.

Madeline is an active 18-year-old who fell off her bike, fracturing the medial epicondyle of her humerus. In addition to experiencing severe pain in her elbow, she had numbness along the medial side of her hand. She went to the emergency room and the physician examined her.

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1. After x-raying her elbow, the physician performed further tests on Madeline to determine what nerve damage she may have experienced. What nerve likely was injured when she fractured her medial epicondyle?
   a. radial
   b. ulnar
   c. musculocutaneous
   d. median

2. What diagnostic test would best help determine the potential damage to this nerve?
   a. Have Madeline extend her elbow against resistance.
   b. Have Madeline flex her wrist and fingers against resistance.
   c. Have Madeline hold a piece of paper between her fingers while the physician tries to pull the paper away.
   d. Have Madeline flex her elbow while the physician applies gentle pressure to the anterior forearm muscles.

3. What other muscle function could be impaired in this type of injury?
   a. adduction of the thumb
   b. extension of the thumb
   c. flexion of the thumb
   d. abduction of the thumb

---

**Can You Synthesize What You’ve Learned?**

1. Arthur dove off a small cliff into water that was shallower than he expected and he hit his head. He is now a quadriplegic, which means that both his upper and lower limbs are paralyzed. Approximately where is the location of his injury? What is the likelihood that Arthur will recover from this injury?

2. Jessica fractured her fibula and had to wear a leg cast for several weeks. When the cast was removed, Jessica had trouble walking normally and experienced “foot drop.” What structure likely was pinched by the leg cast, resulting in the foot drop?

3. Juanita was walking barefoot on the sidewalk when she stepped on a piece of glass with her right foot. Her right leg reflexively lifted up, away from the shard of glass. What is this type of reflex called? In addition, Juanita did not fall down when she lifted her leg. What action in her left lower limb helped stabilize her?
The following study aids may be accessed through Connect.

**Clinical Case Study:** A Young Man with Rapidly Developing Muscle Weakness

**Interactive Questions:** This chapter's content is served up in a number of multimedia question formats for student study

**SmartBook:** Topics and terminology include spinal cord gross anatomy; protection and support of the spinal cord; sectional anatomy of the spinal cord; spinal cord conduction pathways; spinal nerves; reflexes; development of the spinal cord

**Anatomy & Physiology Revealed:** Topics include cervical, thoracic, lumbar, and sacral regions of spinal cord; spinal nerves; brachial plexus; cervical plexus, lumbosacral plexus; reflex arc
15.1 Comparison of the Somatic and Autonomic Nervous Systems
15.1a Functional Organization
15.1b Lower Motor Neurons of the Somatic Versus Autonomic Nervous System
15.1c CNS Control of the Autonomic Nervous System

15.2 Divisions of the Autonomic Nervous System
15.2a Functional Differences
15.2b Anatomic Differences in Lower Motor Neurons
15.2c Degree of Response

15.3 Parasympathetic Division
15.3a Cranial Components
15.3b Pelvic Splanchnic Nerves

15.4 Sympathetic Division
15.4a Organization and Anatomy of the Sympathetic Division
15.4b Sympathetic Pathways

15.5 Autonomic Plexuses and the Enteric Nervous System
15.5a Autonomic Plexuses
15.5b Enteric Nervous System

15.6 Comparison of Neurotransmitters and Receptors of the Two Divisions
15.6a Overview of ANS Neurotransmitters
15.6b Cholinergic Receptors
15.6c Adrenergic Receptors

15.7 Interactions Between the Parasympathetic and Sympathetic Divisions
15.7a Autonomic Tone

INTEGRATE: CONCEPT OVERVIEW
Comparison of the Parasympathetic and Sympathetic Divisions of the ANS
15.7b Dual Innervation
15.7c Systems Controlled Only by the Sympathetic Division

15.8 Autonomic Reflexes

On a twisting downhill slope, an Olympic skier is concentrating on controlling her body to negotiate the course faster than anyone else in the world. Compared to the spectators in the viewing areas, her pupils are dilated, and her heart is beating faster and pumping more blood to her skeletal muscles. At the same time, all organ system functions not needed in the race are practically shut down. Digestion, urination, and defecation can wait until the race is over. The skier exhibits a state of heightened readiness, called the fight-or-flight response, because the sympathetic division of the autonomic nervous system (ANS) is dominant. When the race is finished and she stops to rest and eat, the parasympathetic division of the ANS will dominate to meet the demands of digesting the meal. Thus, both divisions of the ANS—the sympathetic division and the parasympathetic division—function to regulate the body’s response to changing demands—whether they are the demands of exercise or the demands of supplying nutrients to the body.

Acupuncture is one of the mainstays of traditional Chinese medicine (TCM) as well as complementary and alternative medicine (CAM). TCM is based on the premise that disease and pathology are due to a blockage of qi (chē; vital energy) at some location in the body. An acupuncturist inserts long, thin, metallic needles into the skin at specific acupuncture sites to promote qi circulation. Acupuncture is effective at relieving some types of pain, and many studies suggest it can relieve nausea associated with chemotherapy or other medical treatments. Acupuncture has also been used to treat depression, anxiety, infertility and some neurologic disorders. Some researchers hypothesize that acupuncture manipulates our autonomic nervous system (ANS) in ways that are not yet understood. Because the ANS controls our internal environment and innervates our viscera, practices such as acupuncture may help the workings of these structures.
15.1 Comparison of the Somatic and Autonomic Nervous Systems

We introduce this chapter on the autonomic nervous system by first describing how the nervous system is organized into a somatic nervous system and an autonomic nervous system. Here we compare the somatic and autonomic nervous systems’ functional organization, their lower motor neurons, and the CNS regions that control the autonomic nervous system.

15.1a Functional Organization

**LEARNING OBJECTIVE**

1. List the similarities and differences between the SNS and the ANS.

Recall from section 12.1 that anatomists and physiologists have devised various ways to organize the nervous system both structurally and functionally. Here, we introduce a slightly different way of functionally organizing the nervous system into two systems based upon whether we are conscious of the process. The two components are the somatic nervous system and the autonomic nervous system (figure 15.1).

The somatic nervous system (SNS) includes processes that are perceived or controlled consciously (figure 15.1a). The somatic sensory portion includes detection of stimuli and transmission of nerve signals from the special senses (i.e., vision, hearing, equilibrium, smell, and taste), skin, and proprioceptors (receptors in joints and muscles that detect body position) to the CNS. The somatic motor portion involves initiation and transmission of nerve signals from the CNS to control skeletal muscles. This is exemplified by voluntary activities such as getting out of a chair, picking up a book, and throwing a ball for a dog to chase. Both the sensory input we consciously perceive and the motor output we voluntarily initiate to skeletal muscle involve the cerebrum (see section 13.3c). Reflexive skeletal muscle activity is controlled by the brainstem and spinal cord (see section 14.6), whereas coordination of skeletal muscle movements is the function of the cerebellum (see section 13.6b).

The autonomic (aw-to-nom′ik; auto = self, nomos = law) nervous system (ANS), also called the autonomic motor or visceral motor system, includes processes regulated below the conscious level (figure 15.1b). This system is a motor system only (see section 12.1b). These autonomic motor components initiate and transmit nerve signals from the CNS to cardiac muscle, smooth muscle, and glands. The autonomic nervous system often responds to input from visceral sensory components, such as receptors that detect stimuli associated with blood vessels and internal organs (viscera). Some of these sensory neurons, for example, monitor carbon dioxide concentration in the blood, whereas others detect pressure by measuring stretch in smooth muscles of visceral walls. Note that the visceral sensory structures are not part of the ANS per se, but instead simply transmit input from sensory receptors that may result in motor output by the ANS.

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**Figure 15.1 Comparison of Somatic and Autonomic Nervous Systems.** The nervous system is functionally organized into the (a) somatic nervous system, which involves processes that we consciously perceive and control, and the (b) autonomic nervous system, which involves processes that are regulated below the conscious level. (Note that the visceral sensory structures are not part of the ANS per se.)
The function of the ANS is to maintain homeostasis, or a constant internal environment (see section 1.6). Thus, the ANS regulates all physiologic processes that must be maintained by the nervous system to keep the body alive, including the regulation of heart rate, blood pressure, body temperature, sweating, and digestion. The ANS keeps these processes within optimal ranges and adjusts the variables to meet changing body needs.

**WHAT DID YOU LEARN?**

1. What criterion is used to organize the nervous system into the SNS and the ANS? What sensory and motor components are associated with each of the two systems?

15.1b Lower Motor Neurons of the Somatic Versus Autonomic Nervous System

**LEARNING OBJECTIVES**

2. Compare and contrast lower motor neurons in the SNS and ANS.

3. Describe how the two-neuron chain in the ANS facilitates communication and control.

One significant anatomic difference between the somatic nervous system and autonomic nervous system is the number of lower motor neurons that extend from the CNS (see section 14.4c). A single lower motor neuron extends from the CNS to skeletal muscle fibers in the somatic nervous system (figure 15.1a). The cell body of a lower motor neuron lies within the brainstem or the spinal cord, and its axon exits the CNS in either a cranial nerve or a spinal nerve. The preganglionic axon extends from this cell body and exits the CNS in either a cranial nerve or a spinal nerve. This axon projects to an autonomic ganglion in the peripheral nervous system. Preganglionic neurons have myelinated axons that typically are small in diameter, and nerve signals always result in the release of acetylcholine to excite the second neuron.

Imagine that you are flying from Indianapolis to Miami for spring break: Your first flight from Indianapolis to Chicago is the preganglionic axon. Although flying north to Chicago is out of your way, the airline sends you to an airport hub because it is more efficient to send all Indianapolis passengers to this main location before they take different flights throughout the United States.

The airport hub in Chicago is the autonomic ganglion, the point where preganglionic and postganglionic flights meet up. Other preganglionic flights are arriving at the airport hub, and here all these passengers will connect with other flights.

Your connecting flight from Chicago to Miami is the ganglionic neuron. This flight will take you to your final destination, just as a postganglionic axon sends a nerve signal to an effector organ. On the plane with you are people from other preganglionic flights who all want to go to Miami as well.

Is using two different flights the most direct way for you to get from Indianapolis to Florida? Of course not. But it is the most cost-efficient (or energy-efficient) way for the airlines to transport and disperse many passengers with a limited number of planes.

The connecting-flight arrangement also allows for the extensive convergence of passengers to an airport hub, and for the extensive divergence of passengers from the hubs to the final destinations. Thus, the two motor neurons used in the ANS allow for neuronal convergence and divergence.

The autonomic nervous system is similar to connecting airline flights and airport hubs in that both try to group and disperse many different items (nerve signals or passengers) with a limited number of neurons or flights.

In comparison, a chain of two lower motor neurons extends from the CNS to innervate cardiac muscle, smooth muscle, and glands in the ANS (figure 15.1b and figure 15.2). The first of the two ANS motor neurons is the preganglionic (pre′gang-lē-on′ik) neuron. Its cell body lies within the brainstem or the spinal cord. A preganglionic axon extends from this cell body and exits the CNS in either a cranial nerve or a spinal nerve. This axon projects to an autonomic ganglion in the peripheral nervous system. Preganglionic neurons have myelinated axons that typically are small in diameter, and nerve signals always result in the release of acetylcholine to excite the second neuron.

The second neuron in this pathway is called a ganglionic neuron, sometimes referred to as a postganglionic neuron. (Note that the term postganglionic is not accurate, as the cell body of the neuron resides within the ganglion, not after the ganglion.) Its cell body resides within an autonomic ganglion. A postganglionic axon extends from the cell body to an effector (cardiac muscle, smooth muscle, or a gland). (It is appropriate to call the axon postganglionic because it extends from the autonomic ganglion.) Ganglionic neurons have unmyelinated axons that are even smaller in diameter than preganglionic axons. The neurotransmitter released from the ganglionic neuron in response to a nerve signal is either ACh or norepinephrine (NE). Both neurotransmitters can either excite or inhibit an effector depending upon the type of receptors present within the effector (a concept described in section 15.6 in detail). Because motor neurons of the ANS are small and mostly unmyelinated, propagation of nerve signals is relatively slow in comparison to nerve signal propagation along somatic motor axons.

The two-neuron motor pathway in the ANS has a distinctive advantage over the one lower motor neuron of the somatic nervous system: It allows for increasing communication and control. This occurs because of neuronal convergence and neuronal divergence (see section 12.11). Neuronal convergence (kon-ver′jens; convergo = to incline together) occurs because axons from numerous preganglionic neurons synapse with and influence a single ganglionic neuron. Neuronal divergence (di-ver′jens;
Table 15.1 Comparison of Somatic and Autonomic Motor Nervous Systems

<table>
<thead>
<tr>
<th>Feature</th>
<th>Somatic Nervous System</th>
<th>Autonomic Nervous Systems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional Organization</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensory input</td>
<td>Special senses, skin, proprioceptors</td>
<td>Visceral senses (and some somatic senses)</td>
</tr>
<tr>
<td>Effectors</td>
<td>Skeletal muscle fibers</td>
<td>Cardiac muscle cells, smooth muscle cells, glands</td>
</tr>
<tr>
<td>CNS regions of control</td>
<td>Cerebrum, thalamus, cerebellum, brainstem, spinal cord</td>
<td>Hypothalamus, brainstem, spinal cord</td>
</tr>
<tr>
<td></td>
<td>Cerebrum, thalamus, limbic system (regulated by hypothalamus, brainstem, spinal cord)</td>
<td></td>
</tr>
<tr>
<td>Motor Neurons</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of neurons in pathway</td>
<td>One neuron from CNS: Somatic motor neuron axon extends from CNS to effector</td>
<td>Two neurons from CNS: Preganglionic neuron has preganglionic axon that projects to ganglionic neuron; ganglionic neuron has postganglionic axon that projects to effector</td>
</tr>
<tr>
<td>Axon properties</td>
<td>Myelinated and thicker in diameter; fast nerve signal propagation</td>
<td>Preganglionic axons are myelinated and small in diameter Postganglionic axons are unmyelinated and smaller in diameter; both have relatively slow nerve signal propagation</td>
</tr>
<tr>
<td>Neurotransmitter released</td>
<td>Acetylcholine (ACh)</td>
<td>Preganglionic axons release ACh Postganglionic axons release either ACh or norepinephrine (NE)</td>
</tr>
<tr>
<td>Response of effector</td>
<td>Excitation only</td>
<td>Either excitation or inhibition</td>
</tr>
<tr>
<td>Ganglia associated with motor neurons</td>
<td>None</td>
<td>Parasympathetic division: terminal ganglia, intramural ganglia Sympathetic division: sympathetic trunk ganglia, prevertebral ganglia</td>
</tr>
</tbody>
</table>

Figure 15.2 Lower Motor Neurons of the Autonomic Nervous System. The autonomic nervous system employs a chain of two lower motor neurons, a preganglionic neuron and a ganglionic neuron. The dendrites and cell body of a preganglionic neuron are housed within the CNS (brain or spinal cord). The preganglionic axon synapses with a ganglionic neuron within an autonomic ganglion. The postganglionic axon extends to an effector organ, which includes cardiac muscle, smooth muscle, and glands.

di = apart) occurs because axons from one preganglionic cell synapse with and influence numerous ganglionic neurons.

Table 15.1 compares the characteristics of the somatic and autonomic nervous systems.

WHAT DID YOU LEARN?

2. What are the anatomic features that distinguish the motor neurons in the SNS and ANS?

15.1c CNS Control of the Autonomic Nervous System

LEARNING OBJECTIVE

4. Describe the CNS hierarchy that controls the autonomic nervous system.

Several levels of CNS complexity are required to coordinate and regulate ANS function. Thus, despite the name autonomic, the ANS is a regulated nervous system, not an independent one. Autonomic function is regulated by three CNS regions: the hypothalamus, brainstem, and spinal cord (figure 15.3). These CNS regions may be influenced by the cerebrum, thalamus, and limbic system.

The hypothalamus is the integration and command center for autonomic functions (see section 13.4c). It contains nuclei that control visceral functions in both divisions of the ANS, and it communicates with other CNS regions, including the brainstem and spinal cord. The hypothalamus is the central brain structure involved in emotions and physiologic processes, which are regulated through the ANS. For example, the sympathetic division’s fight-or-flight response originates in the sympathetic nucleus in this brain region.

The brainstem nuclei mediate visceral reflexes (see section 13.5). These reflex centers control changes in blood pressure, blood vessel diameter, digestive activities, heart rate, pupil size, and eye lens shape for focusing on close-up objects.
Some autonomic responses, notably the parasympathetic activities associated with defecation and urination (in children), are processed and controlled at the level of the spinal cord without the involvement of the brain. However, the higher centers in the brain may consciously prevent defecation and urination by controlling the external sphincters.

ANS activities are affected by conscious activities in the cerebral cortex and subconscious communications between association areas in the cortex and the centers of parasympathetic and sympathetic control in the hypothalamus. Additionally, sensory processing in the thalamus (see section 13.4b) and emotional states controlled in the limbic system (see section 13.7a) directly affect the hypothalamus.

### 15.2 Divisions of the Autonomic Nervous System

The motor component of the ANS is further subdivided into the parasympathetic division and the sympathetic division. Here we discuss the general functional and anatomic differences between the two divisions, as well as the degree of response (local or mass activation) that is possible when each division is activated.

#### 15.2a Functional Differences

**LEARNING OBJECTIVE**

5. Describe the general functions of the parasympathetic and sympathetic divisions of the autonomic nervous system.

The divisions perform dramatically different functions—but instead of being thought of as antagonistic, they should be considered complementary. The parasympathetic (par-ä-sim-pà-thet’ik; para = alongside, sympathee = to feel with) division functions to maintain homeostasis when we are at rest. This division is primarily concerned with conserving energy and replenishing nutrient stores. Because it is most active when the body is at rest or digesting a meal, the parasympathetic division has been nicknamed the *rest-and-digest* division.

#### 15.2b Anatomic Differences in Lower Motor Neurons

**LEARNING OBJECTIVE**

6. Compare and contrast the anatomic differences in the lower motor neurons and associated ganglia of the parasympathetic and sympathetic divisions.

Anatomically, these two divisions are similar in that they typically both use a preganglionic neuron and a ganglionic neuron to innervate cardiac muscle, smooth muscle, or glands. Additionally, both divisions have autonomic ganglia that house the ganglionic neuron cell bodies. One of the major differences is where the preganglionic neuron cell bodies are housed in the CNS (figure 15.4). Parasympathetic preganglionic cell bodies are located in either the brainstem or the lateral gray matter of the S2–S4 spinal cord segments, and for this reason this division is also termed the *craniosacral* (kra’ne-ô-sâ’krâl) division. In comparison, sympathetic preganglionic neuron cell bodies are located in the lateral horns of the T1–L2 spinal cord segments, and so this division also goes by the phrase the *thoracolumbar* (thôr’ä-kō-lûm’bar) division.

Other anatomic differences between the parasympathetic and sympathetic nervous system include:

- **Length of preganglionic and postganglionic axons.** Parasympathetic preganglionic axons are longer, and postganglionic axons are shorter, when compared to their counterparts in the sympathetic division. In the sympathetic division, preganglionic axons are shorter and postganglionic axons are longer.
- **Number of preganglionic axon branches.** Parasympathetic preganglionic axons tend to have fewer (less than 4) branches,
Autonomic Motor Nervous System

Parasympathetic Division (craniosacral division)

- Origin: Preganglionic neurons located in brainstem nuclei and S2–S4 segments of spinal cord (craniosacral)
- Functions:
  - Brings body to homeostasis in conditions of “rest-and-digest”
  - Conserves energy and replenishes nutrient stores

- CN III (oculomotor)
- CN VII (facial)
- CN IX (glossopharyngeal)
- CN X (vagus)
- Pelvic splanchnic nerves

- S2–S4 segments of spinal cord

Sympathetic Division (thoracolumbar division)

- Origin: Preganglionic neurons located in lateral horns of T1–L2 segments of spinal cord (thoracolumbar)
- Functions:
  - Brings body to homeostasis in conditions of “fight-or-flight”
  - Increases alertness and metabolic activities

- Sympathetic trunk
- CN III (oculomotor)
- CN VII (facial)
- CN IX (glossopharyngeal)
- CN X (vagus)
- Pelvic splanchnic nerves

- T1–L2 segments of spinal cord

Figures 15.4 Comparison of Parasympathetic and Sympathetic Divisions. The neurons of the parasympathetic division extend from the brainstem and sacral region, whereas the neurons of the sympathetic division extend from the thoracic and lumbar regions of the cord. The parasympathetic division axons exhibit very little branching, and autonomic ganglia lie close to or within the effector organ. The sympathetic division axons of both neurons show much branching, and autonomic ganglia lie close to the vertebral column.

WHAT DID YOU LEARN?

Describe the general anatomic differences in the parasympathetic and sympathetic divisions.

15.2c Degree of Response

LEARNING OBJECTIVE

7. Explain why parasympathetic activation is local and discrete, and sympathetic activation can result in mass activation.

It is the combination of long preganglionic axons with limited branches that results in a local response when the parasympathetic division is activated. Parasympathetic activity regulates either one or only a few structures at the same time without having to “turn on” or “turn off” all the other organs.

In comparison, the combination of short preganglionic axons with more extensive branching within the sympathetic division allows for significant neuronal divergence and facilitates the activation of many structures simultaneously, a process called mass activation. This process is facilitated when the adrenal medulla is stimulated by the sympathetic division, which causes this gland to release norepinephrine and epinephrine into the blood (see sections 17.2b and 17.9a). Mass activation is especially important in response to stress, when it is necessary to coordinate rapid changes in activity with numerous structures at once. Think of all the bodily changes that are initiated when you are exercising or scared: changes that include an increase in heart rate and blood pressure, increases in the amount of air that enters the lungs, dilation of the pupils, and mobilization of energy reserves from the liver. Keep in mind, however, that there are times when the sympathetic division may activate a single effector. For example, only a single effector is involved when the sympathetic division stimulates smooth muscle to increase the diameter of the pupil of the eye during low-light conditions (see section 16.4b).
15.3 Parasympathetic Division

The parasympathetic division is primarily concerned with maintaining homeostasis at rest and is functionally considered the rest-and-digest division. The parasympathetic division is also called the craniocervical division because of the anatomic origin of its preganglionic neuron from the brainstem and sacral region of the spinal cord. The two types of ganglia associated with the parasympathetic division are small and generally not individually named but are referred to as either the terminal (ter′mi-năl; terminus = a boundary) ganglia, which are located close to the effector, or the intramural (in′tră-mū′răl; intra = within, murus = wall) ganglia, which are located within the wall of the target organ. The exceptions are the four relatively large parasympathetic ganglia associated with the head and neck, which are individually named (e.g., ciliary ganglion). Here we discuss the details of the structure and function of the cranial and sacral components of the parasympathetic division. Table 15.2 summarizes the different nerves associated with the parasympathetic division.

### 15.3a Cranial Components

#### LEARNING OBJECTIVE

8. Name the four cranial nerves associated with the parasympathetic division, and describe their actions.

The cranial nerves containing neurons from the parasympathetic division are the oculomotor (CN III), facial (CN VII), glossopharyngeal (CN IX), and vagus (CN X) nerves. (Recall that these cranial nerves are paired; they are found on the left and right sides of the body.) Review section 13.9 for summaries and illustrations of the cranial nerve pathways and the locations of their associated parasympathetic ganglia. The first three of these nerves transmit parasympathetic innervation to the head, whereas the vagus nerve is the source of parasympathetic innervation for the thoracic and most abdominal organs (figure 15.5). Here we discuss the function and then the anatomic pathway for the parasympathetic component of each of these four cranial nerves.

**Oculomotor Nerve (CN III)**

The oculomotor nerve (CN III) innervates both (1) the ciliary muscle (within the eye) to adjust the shape of the lens to see close-up objects and (2) the sphincter pupillae muscle of the iris (which constricts the pupil) to allow less light into the eye, such as when we first walk outside on a bright, sunny day (see section 16.4b).

The axons of the preganglionic neurons extend from cell bodies housed in nuclei within the midbrain to the ciliary (sil′e-ar-ē; ciliaris = eyelash) ganglion within the orbit. Postganglionic axons project from this ganglion to the effectors (ciliary muscle and iris).

**Facial Nerve (VII)**

The facial nerve (CN VII) innervates the submandibular and sublingual salivary glands in the floor of the mouth (see section 26.2b), lacrimal glands in the superior portion of each orbit (see section 16.4a), and small glands of the nasal cavity, oral cavity, and palate. Stimulation by the parasympathetic division increases release of secretion by these glands. Your mouth waters when you smell an aromatic meal due in part to these parasympathetic neurons within the facial nerve.

### Table 15.2: Parasympathetic Division Outflow

<table>
<thead>
<tr>
<th>Nerve(s)</th>
<th>Origin of Preganglionic Neurons</th>
<th>Autonomic Ganglia</th>
<th>Effectors Innervated</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN III (oculomotor)</td>
<td>Midbrain</td>
<td>Ciliary ganglion</td>
<td>Eye; ciliary muscles to alter the shape of the lens for close vision; iris (sphincter pupillae muscle) to constrict pupil</td>
</tr>
<tr>
<td>CN VII (facial)</td>
<td>Pons</td>
<td>Pterygopalatine ganglion</td>
<td>Lacrimal glands; glands of nasal cavity, palate, oral cavity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Submandibular ganglion</td>
<td>Submandibular and sublingual salivary glands</td>
</tr>
<tr>
<td>CN IX (glossopharygeal)</td>
<td>Medulla oblongata</td>
<td>Otic ganglion</td>
<td>Parotid salivary glands</td>
</tr>
<tr>
<td>CN X (vagus)</td>
<td>Medulla oblongata</td>
<td>Terminal and intramural ganglia</td>
<td>Thoracic viscera and most abdominal viscera</td>
</tr>
<tr>
<td>Pelvic splanchnic nerves</td>
<td>S2–S4 segments of spinal cord</td>
<td>Terminal and intramural ganglia</td>
<td>Some abdominal viscera and most pelvic viscera</td>
</tr>
</tbody>
</table>
Figure 15.5 Overview of Parasympathetic Pathways. Preganglionic neurons of the parasympathetic division extend from the brain and sacral region of the spinal cord. Ganglionic neurons are located in terminal and intramural ganglia, and extend from these ganglia to innervate the viscera.
The axons of the preganglionic parasympathetic neurons extend from the pons to terminate at one of two ganglia. Some preganglionic axons terminate within the submandibular (sub-man-di-búl′ăr; sub = under) ganglion, which is located near the angle of the mandible. Postganglionic axons projecting from this ganglion innervate the submandibular and sublingual salivary glands. Other preganglionic axons terminate within the pterygopalatine (ter′i-gó-pal′ă-fin; pterygo = wing-shaped, palatine = of the palate) ganglion, which is positioned near the junction of the maxilla and palatine bones. Postganglionic axons projecting from this ganglion innervate the lacrimal glands, as well as small glands of the nasal cavity, oral cavity, and palate.

"WHAT DO YOU THINK?"

1. The pterygopalatine ganglion is sometimes nicknamed the hay fever ganglion. Why is this nickname appropriate?

**Glossopharyngeal Nerve (CN IX)**

The glossopharyngeal nerve (CN IX) innervates the parotid salivary glands and stimulates these glands to increase the release of their secretions. The axons of the preganglionic neurons extend from cell bodies within the brainstem to the otic (ō’tik; ous = ear) ganglion, which is anterior to the ear. Postganglionic axons project from this ganglion to innervate the parotid salivary glands.

Vagus Nerve (CN X)

The vagus nerve (CN X) innervates the thoracic organs and most of the abdominal organs, as well as the gonads (ovaries and testes). The term vagus means “wanderer,” which describes the wandering pathway the vagus nerve makes as it projects inferiorly through the neck and extends throughout the trunk. The influence of the vagus nerve is extensive, given the significant number of autonomic effectors this cranial nerve innervates. The vagus affects the following structures:

- **Heart:** decreases heart rate (see section 19.9b)
- **Bronchi/bronchioles:** constricts to decrease airflow into the air sacs (alveoli) of the lungs (see section 23.5d)
- **Gastrointestinal (GI) tract:** stimulates secretions released from the GI tract wall, increases motility (or movement) of the contents through the GI tract, and relaxes sphincters to allow the passage of contents within the GI tract (see section 26.1c)
- **Liver:** stimulates glycogenesis, which is glycogen formation from glucose (see section 27.6c)

The axons of the preganglionic neurons of the vagus nerve extend through one or more plexuses (see section 15.5a) and continue to either a terminal or an intramural ganglion. Postganglionic axons project from the ganglion to the effector organ.

"WHAT DID YOU LEARN?"

- Which four cranial nerves have a parasympathetic component? What organs are innervated and what physiologic response is caused by each?

**15.3b Pelvic Splanchnic Nerves**

**LEARNING OBJECTIVE**

9. Explain the actions of the pelvic splanchnic nerves.

The remaining parasympathetic preganglionic axons originate from preganglionic neuron cell bodies housed within the lateral gray regions of the S2–S4 spinal cord segments. These preganglionic axons extend through the anterior root and then branch to form the pelvic splanchnic (splan’knik; splanchnic = visceral) nerves, which contribute to a superior and inferior hypogastric plexus on each side of the body. The preganglionic axons that continue through each plexus project to the ganglionic neurons within either the terminal or intramural ganglia. The postganglionic axons extend to the effector.

The target organs innervated include the distal portion of the large intestine, the rectum, the urinary bladder, the distal part of the ureter, and most of the reproductive organs. The parasympathetic regulation of these target organs causes increased smooth muscle motility (muscle contraction) and secretory activity in these portions of the digestive tract (see chapter 26), contraction of smooth muscle in the urinary bladder wall and relaxation of the smooth muscle of the internal urethral sphincter (which facilitates urination; see section 24.8c), and erection of the female clitoris and the male penis (see sections 28.3g and 28.4f).

**15.4 Sympathetic Division**

The sympathetic division is primarily concerned with preparing the body for exercise and emergencies, and is functionally considered the fight-or-flight division. Recall from section 15.2b that the lower motor neurons of the sympathetic division extend only from the thoracic and lumbar regions of the spinal cord (T1–L2), which is why this division is also called the thoracolumbar division. The two types of ganglia associated with the sympathetic division are the sympathetic trunk ganglia and the prevertebral ganglia.

**15.4a Organization and Anatomy of the Sympathetic Division**

**LEARNING OBJECTIVES**

10. Give the location of the sympathetic preganglionic neuron cell bodies.
11. Describe the left and right sympathetic trunks and ganglia.
12. Compare and contrast white and gray rami regarding their location and composition.
13. Explain the differences between the sympathetic trunk ganglia and the prevertebral ganglia.

The sympathetic division is much more anatomically complex than the parasympathetic division, so we first describe its anatomic components and then its pathways (figure 15.6). The sympathetic preganglionic neuron cell bodies are housed in the lateral horn of the T1–L2 regions of the spinal cord. (See figure 14.4b in section 14.3a, which compares the location of autonomic motor and the somatic motor cell bodies in the spinal cord.) From there, the preganglionic sympathetic axons travel with somatic motor axons to exit the spinal cord through first the anterior roots and then the T1–L2 spinal nerves. However, these preganglionic sympathetic axons remain with the spinal nerve for only a short distance before they branch from the spinal nerve.

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1. It is unclear what function, if any, these parasympathetic axons have on the gonads.
Figure 15.6 Overview of Sympathetic Pathways. Preganglionic axons of the sympathetic division extend from the T1–L2 regions of the spinal cord. Ganglionic axons are located in the sympathetic trunk and prevertebral ganglia, and extend to the target organs. (Left) The outflow of preganglionic axons and the distribution of postganglionic axons innervating the skin. (Right) Sympathetic postganglionic axon pathways to internal organs.
Sympathetic Trunks and Sympathetic Trunk Ganglia

Immediately lateral to the vertebral column and anterior to the paired spinal nerves are the left and right sympathetic trunks (figure 15.7). A sympathetic trunk looks much like a pearl necklace. The “string” of the necklace is composed of bundles of axons, whereas the “pearls” are the sympathetic trunk ganglia (also known as paravertebral or sympathetic chain ganglia), which house sympathetic ganglionic neuron cell bodies.

One sympathetic trunk ganglion typically is associated with each spinal nerve. However, the cervical portion of each sympathetic trunk is partitioned into only three sympathetic trunk ganglia—the superior, middle, and inferior cervical ganglia—as opposed to the eight cervical spinal nerves (figure 15.6). The superior cervical ganglion contains postganglionic sympathetic neuron cell bodies whose axons are distributed primarily to structures within the head and neck and to some thoracic viscera. These postganglionic axons innervate the sweat glands and smooth muscle in blood vessels of the head and neck, the dilator pupillae muscle of the eye, and the superior tarsal muscle of the eye (which elevates the eyelid). The middle and inferior cervical ganglia also house neuron cell bodies that extend postganglionic axons to the thoracic viscera (e.g., heart, bronchi and bronchioles of the lungs).

White and Gray Rami

Connecting the spinal nerves to each sympathetic trunk are rami communicantes (rä’mi kō-mū-ni-kan’tēz; sing., ramus communicans; rami = branches, communico = to share with someone) (figure 15.7; see also figure 14.13). White rami communicantes (or simply white rami) are composed of preganglionic sympathetic axons from the T1–L2 spinal nerves to the sympathetic trunk. Thus, white rami are associated only with the T1–L2 spinal nerves. Recall that preganglionic axons are myelinated, giving these rami a whitish appearance. White rami are similar to entrance ramps onto a highway.

Gray rami communicantes (or simply gray rami) are composed of postganglionic sympathetic axons that extend from the sympathetic trunk to the spinal nerve. Postganglionic axons are unmyelinated, so these rami have a grayish appearance. Gray rami connect to all spinal nerves, including the cervical, sacral, and coccygeal spinal nerves. By these routes, the sympathetic information that started out in the thoracolumbar region can be dispersed to all parts of the body. Gray rami are similar to exit ramps from a highway.

Sympathetic Splanchnic Nerves

Sympathetic splanchnic nerves are composed of preganglionic sympathetic axons that did not synapse in a sympathetic trunk ganglion (figure 15.6). They run anteriorly from the
sympathetic trunk to most of the abdominal and pelvic viscera. These splanchnic nerves should not be confused with the pelvic splanchnic nerves associated with the parasympathetic division, described in section 15.3. Some of the larger splanchnic nerves have specific names, such as thoracic splanchnic nerves, lumbar splanchnic nerves, or sacral splanchnic nerves.

**Prevertebral Ganglia**

Splanchnic nerves typically terminate in prevertebral (or collateral) ganglia differ from the sympathetic trunk ganglia in that (1) they are anterior to the vertebral column (prevertebral) on the anterolateral surface of the aorta and (2) they are located only in the abdominopelvic cavity. Prevertebral ganglia include the celiac, superior mesenteric, and inferior mesenteric ganglia.

The **celiac ganglia** are adjacent to the origin of the celiac artery. The greater thoracic splanchnic nerves (composed of axons from T5–T9 segment of the spinal cord) synapse on ganglionic neurons within each celiac ganglion. Postganglionic axons from the celiac ganglia innervate the stomach, spleen, liver, gallbladder, and proximal part of the duodenum (first part of small intestine) and part of the pancreas.

The **superior mesenteric ganglia** are adjacent to the origin of the superior mesenteric artery. They receive sympathetic preganglionic axons from the T10–T12 segments of the spinal cord. Postganglionic axons extending from the superior mesenteric ganglia innervate the distal half of the duodenum, the remainder of the small intestine, the proximal part of the large intestine, part of the pancreas, the kidneys, and the proximal parts of the ureters.

The **inferior mesenteric ganglia** are adjacent to the origin of the inferior mesenteric artery. They receive sympathetic preganglionic axons via the lumbar splanchnic nerves, which originate in the L1–L2 segments of the spinal cord. The postganglionic axons project to and innervate the distal part of the large intestine, the rectum, the urinary bladder, distal parts of the ureters, and most of the reproductive organs.

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**WHAT DID YOU LEARN?**

9. What is the difference between sympathetic trunk ganglia and prevertebral ganglia?

10. What are the structural and functional differences between the white and gray rami communicantes?
15.4b Sympathetic Pathways

**LEARNING OBJECTIVES**

14. Describe the four pathways of sympathetic neurons.
15. Compare and contrast which general effector organs are innervated by each pathway.

All sympathetic preganglionic neurons originate in the lateral gray horns of the T1–L2 regions of the spinal cord. The axons of the preganglionic sympathetic neurons travel with somatic motor axons to exit the spinal cord within the anterior roots and then through the spinal nerves. However, these preganglionic sympathetic axons remain with the spinal nerve for only a short distance before they leave the spinal nerve within the white ramus. It is at this point where the major pathways of the sympathetic division differ. Each type of pathway is dependent upon the location of the effector organ being innervated. Axons exit the sympathetic trunk by one of four pathways.

**Spinal Nerve Pathway**

The spinal nerve pathway extends from the spinal cord to effectors of the skin of the neck, torso, and limbs. Skin effectors include sweat glands, smooth muscle forming arrector pili muscles (which produce "goose bumps"), and smooth muscle cells within the walls of blood vessels (see section 6.2a). In this pathway, a preganglionic neuron synapses with a ganglionic neuron in a sympathetic trunk ganglion at either the same or different level (figure 15.8a). The postganglionic axon extends through a gray ramus that is at the same "level" as the ganglionic neuron. For example, if the preganglionic and ganglionic neurons synapse in the L4 ganglion, the postganglionic axon extends through the gray ramus at the level of the L4 spinal nerve. After the postganglionic axon extends through the gray ramus, it enters the spinal nerve and extends to its target organ.

**Postganglionic Sympathetic Nerve Pathway**

The postganglionic sympathetic nerve pathway extends from the spinal cord to the internal organs of the thoracic cavity (including the esophagus, heart, lungs, and thoracic blood vessels), the effectors of the skin of the head (sweat glands, arrector pili, and blood vessels of the skin), the neck viscera, and the superior tarsal and dilator pupillae muscles in the eye (for increasing the amount of light entering the eye) (see section 16.4b). In this pathway, the preganglionic neuron synapses with a ganglionic neuron in a sympathetic trunk ganglion, but the postganglionic axon does not leave the trunk via a gray ramus (figure 15.8b). Instead, the postganglionic axon extends away from the sympathetic trunk ganglion and projects directly to the effector organ.

**Splanchnic Nerve Pathway**

The splanchnic nerve pathway extends from the spinal cord to the abdominal and pelvic organs (e.g., stomach, small intestines, kidney). In this pathway, a preganglionic neuron does not synapse with a ganglionic neuron in a sympathetic trunk ganglion. Rather, the preganglionic axons pass through the sympathetic trunk ganglia without synapsing and extend to the prevertebral ganglia (figure 15.8c). There, the postganglionic axon synapses with a ganglionic neuron. The postganglionic axon then projects to the effector organs.

**Adrenal Medulla Pathway**

The final pathway is the adrenal medulla pathway (figure 15.8d). In this pathway, the internal region of the adrenal gland, called the adrenal (á-dré’nál; ad = to, ren = kidney) medulla, is directly innervated by preganglionic sympathetic axons. (There is no ganglionic neuron.) The axons of the preganglionic neuron extend through both the sympathetic trunk and the prevertebral ganglia and then synapse on neurosecretory cells within the adrenal medulla. Stimulation of these cells causes the release of epinephrine (ep’i-nef’rin) and norepinephrine (nör-e-pé-nef’rin). 

![Figure 15.8 Types of Sympathetic Pathways](image_url)

Figure 15.8 Types of Sympathetic Pathways. Pathways of (a) a spinal nerve, (b) a postganglionic sympathetic nerve, (c) a splanchnic nerve, and (d) the adrenal medulla.
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**Table 15.3 Sympathetic Division Pathways**

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Destination</th>
<th>Spinal Segment Origin</th>
<th>Postganglionic Axon Pathway from Sympathetic Trunk</th>
<th>Effector Innervated¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinal nerve</td>
<td>Integumentary structures</td>
<td>T1–L2</td>
<td>Via cervical gray rami to all spinal nerves</td>
<td>Sweat glands, arrector pili muscles, blood vessels in skin of neck, torso, and limbs</td>
</tr>
<tr>
<td>Postganglionic sympathetic nerve</td>
<td>Head and neck viscera</td>
<td>T1–T2 (primarily from T1)</td>
<td>Via superior cervical ganglion and travel with blood vessels to the head and neck viscera</td>
<td>Sweat glands, arrector pili muscles, and blood vessels in skin of head; dilator pupillae muscle of eye; superior tarsal muscle of eye; neck viscera</td>
</tr>
<tr>
<td>Thoracic organs</td>
<td></td>
<td>T1–T5</td>
<td>Via cervical and thoracic ganglia to autonomic nerve plexuses near organs</td>
<td>Esophagus, heart, lungs, blood vessels within thoracic cavity</td>
</tr>
<tr>
<td>Splanchnic nerve</td>
<td>Most abdominal organs</td>
<td>T5–T12</td>
<td>Via thoracic splanchnic nerves to prevertebral ganglia (e.g., celiac, superior mesenteric, and inferior mesenteric ganglia)</td>
<td>Abdominal portion of esophagus, stomach, liver, gallbladder, spleen, pancreas, small intestine, most of large intestine, kidneys, ureters, adrenal glands, blood vessels within abdominopelvic cavity</td>
</tr>
<tr>
<td>Pelvic organs</td>
<td></td>
<td>T10–L2</td>
<td>Via lumbar and sacral splanchnic nerves to autonomic nerve plexuses that travel to effectors</td>
<td>Distal part of large intestine, anal canal, and rectum; distal part of ureters; urinary bladder; reproductive organs</td>
</tr>
<tr>
<td>Adrenal medulla</td>
<td>Adrenal gland</td>
<td>T8–T12</td>
<td>No ganglionic neuron; axons of preganglionic neurons extend via thoracic splanchnic nerves directly to adrenal medulla</td>
<td>Neurosecretory cells of adrenal medulla</td>
</tr>
</tbody>
</table>

¹. Sympathetic axons innervate smooth muscle, cardiac muscle, and glands associated with the organs listed.

**norepinephrine** (nɔrˈep-i-nərˈfriːn) into the blood (see section 17.9a). (The relative amounts of these two hormones released are not equal. Typically, epinephrine accounts for approximately 80% and norepinephrine accounts for 20% of the hormone molecules that are released.) Both of these hormones then circulate within the blood and bind to many of the same receptors as norepinephrine, and in so doing help potentiate (prolong) the effects of the sympathetic stimulation. For example, if you narrowly miss getting into a car accident, your heart continues to beat quickly, you breathe rapidly, and you feel tense and alert well after the event because of prolonged effects of the sympathetic stimulation of the adrenal medulla. Details for epinephrine and norepinephrine are listed at the end of chapter 17 in Table R.5: “Regulating the Stress Response with Catecholamines and Glucocorticoids.” Table 15.3 summarizes the sympathetic division pathways.

**WHAT DO YOU THINK?**

2. When a person is very stressed and tense, his or her blood pressure typically rises. What aspect of the sympathetic division causes this rise in blood pressure?

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**Figure 15.4**

(c) Splanchnic nerve pathway (to abdominal and pelvic viscera)

(d) Adrenal medulla pathway

**Chapter Fifteen**

**Nervous System: Autonomic Nervous System**

595
15.5 Autonomic Plexuses and the Enteric Nervous System

Both divisions of the autonomic nervous system innervate organs through specific axon bundles called autonomic plexuses. Communication between neurons and effectors is through specific neurotransmitters (described in detail in section 15.6). The enteric nervous system is an autonomic array of neurons that may act independently or be affected by ANS innervation. We discuss autonomic plexuses first.

15.5a Autonomic Plexuses

**LEARNING OBJECTIVE**

16. Describe the structure and location of the five autonomic plexuses.

Autonomic plexuses are collections of sympathetic postganglionic axons and parasympathetic preganglionic axons, as well as some visceral sensory axons. These sympathetic and parasympathetic axons are close to one another, but they do not interact or synapse with each another. Although these plexuses look like disorganized masses of axons, they provide a complex innervation pattern to their target organs (figure 15.9).

In the mediastinum of the thoracic cavity, the cardiac plexus consists of sympathetic postganglionic axons that originate in the cervical and thoracic sympathetic trunk ganglia, as well as parasympathetic preganglionic axons from the vagus nerve. Increased sympathetic activity increases heart rate and blood pressure, whereas increased parasympathetic activity decreases heart rate (see section 19.5b).

The pulmonary plexus consists of sympathetic postganglionic axons from the cervical and thoracic sympathetic trunk ganglia and parasympathetic preganglionic axons from the vagus nerve. The axons project to the bronchi and bronchioles of the lungs. Sympathetic innervation causes bronchodilation (increase in the diameter of the bronchi and bronchioles of the lung), whereas stimulation of this parasympathetic pathway causes bronchoconstriction (reduction in the diameter of the bronchi and bronchioles; see section 23.3c).

The esophageal plexus consists of sympathetic postganglionic axons from the cervical and sympathetic trunk ganglia and parasympathetic preganglionic axons from the vagus nerve. Sympathetic innervation will inhibit muscle motility. Smooth muscle activity in the inferior esophageal wall is coordinated by parasympathetic axons that control the swallowing reflex in the inferior region of the esophagus by innervating the cardiac sphincter, a valve through which swallowed food and drink must pass (see section 26.2c).

The abdominal aortic plexus consists of the celiac plexus, superior mesenteric plexus, and inferior mesenteric plexus. It

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**Figure 15.9 Autonomic Plexuses.** Autonomic plexuses are located in both the thoracic and abdominopelvic cavities. This anterior view shows the cardiac, pulmonary, and esophageal plexuses in the thoracic cavity and the abdominal aortic plexus (celiac, superior mesenteric, inferior mesenteric plexuses) in the abdominopelvic cavity.
innervates all abdominal and some pelvic organs. The abdominal aortic plexus is composed of sympathetic postganglionic axons projecting from the prevertebral ganglia and parasympathetic preganglionic axons from either the vagus nerve or the pelvic splanchnic nerves. Note that the celiac plexus is also known as the solar plexus, and it is partly responsible for the sensation of “getting the wind knocked out of you” when you are hit hard in the epigastric region.

The hypogastric plexus consists of a complex meshwork of sympathetic postganglionic axons (from the aortic plexus and the lumbar region of the sympathetic trunk) and preganglionic parasympathetic axons from the pelvic splanchnic nerves. Its axons innervate viscera within the pelvic region.

15.5b Enteric Nervous System

**LEARNING OBJECTIVE**
17. Explain the function and location of the enteric nervous system (ENS).

The enteric nervous system (ENS) is an array of neurons (both autonomic motor and visceral sensory) that are arranged throughout the wall of the gastrointestinal (GI) tract, from the esophagus to the anus. The ENS not only innervates the smooth muscle and glands of the GI tract but also mediates the complex coordinated reflexes for peristalsis, or movement of materials through the GI tract (see section 26.1c). ENS neurons are located both within numerous small ganglia throughout the GI tract wall and within two large plexuses: (1) the submucosal plexus (also called the Meissner plexus) and (2) the myenteric plexus (also called the Auerbach plexus). Although the enteric nervous system can function independently of the rest of the ANS, the ANS division may adjust its activity. In general, the parasympathetic division increases ENS activity, and the sympathetic division decreases ENS activity.

**WHAT DID YOU LEARN?**
13. What basic structures form an autonomic plexus?
14. Where is the enteric nervous system located, and what is its function?

15.6 Comparison of Neurotransmitters and Receptors of the Two Divisions

Transmission of a nerve signal to synaptic knobs causes the release of neurotransmitters into the synaptic cleft. The ANS utilizes several types of neurotransmitters, which are discussed here.

15.6a Overview of ANS Neurotransmitters

**LEARNING OBJECTIVE**
18. Identify the targets of the cholinergic and adrenergic neurotransmitters of the ANS.

Acetylcholine (ACh) and norepinephrine (NE) are the main neurotransmitters used in the ANS (figure 15.10). These neurotransmitters will bind to specific receptors on the postsynaptic cell. Depending upon the receptor type, the neurotransmitter may cause either stimulation or inhibition.

![Figure 15.10 Comparison of Neurotransmitters in the Autonomic Nervous System](image)

Figure 15.10 Comparison of Neurotransmitters in the Autonomic Nervous System. In the parasympathetic pathway, both the preganglionic and postganglionic axons release acetylcholine (ACh). In the sympathetic pathways, all preganglionic axons and a few specific postganglionic axons release ACh. Most postganglionic sympathetic axons release norepinephrine (NE).
Neurons that synthesize and release acetylcholine (ACh) are called cholinergic (kol-in-er′jik; ergon = work) neurons. Receptors that bind ACh are called cholinergic receptors. We will see in section 15.6b that there are two types of cholinergic receptors (nicotinic and muscarinic), and each responds differently to binding of ACh. Cholinergic neurons include the following:

- All sympathetic and parasympathetic preganglionic neurons
- All parasympathetic ganglionic neurons
- The specific sympathetic ganglionic neurons that innervate sweat glands of the skin *(Note that there are nicotinic receptors at the neuromuscular junctions of skeletal muscle, but the neurons involved are somatic, not autonomic.)*

Neurons that synthesize and secrete norepinephrine (NE) are called adrenergic (ad-re-ner′jik) neurons. Most other sympathetic ganglionic neurons are adrenergic. Receptors that bind NE (or a related molecule, like epinephrine) are called adrenergic receptors. These receptors are subdivided into alpha (α) and beta (β) types and are discussed in further detail in section 15.6c.

**WHAT DID YOU LEARN?**

15. Which ANS neurons are cholinergic? Which are adrenergic?

15.6b Cholinergic Receptors

**LEARNING OBJECTIVE**

19. Describe the two types of cholinergic receptors and the action of each when the neurotransmitter acetylcholine binds to them.

Two categories of cholinergic receptors, nicotinic and muscarinic, have been identified in the CNS and PNS. They were differentiated and named (nicotinic or muscarinic) because molecules that are similar to ACh bind to them and cause their stimulation:

Nicotinic receptors were so named because nicotine (i.e., the chemical compound found in tobacco) selectively binds to these receptors. These receptors are found on the cell bodies and dendrites of all ganglionic neurons (figure 15.10), as well as on adrenal medulla cells. When nicotinic receptors bind ACh, they open ion channels to allow greater movement of sodium ions (Na+) into the cell than potassium ions (K+) out of the cell. Thus, the membrane depolarizes and an excitatory postsynaptic potential (EPSP) is produced (see section 12.8a). In other words, ACh binding to nicotinic receptors always produces a stimulatory or excitatory response.

Nicotinic receptors have various subtypes. These subtypes account for the difference in response that a given neurotransmitter may have at different locations. For example, the nicotinic receptor at the neuromuscular junction is blocked by the toxin curare, but it is not blocked by the cholinergic drugs hexamethonium and mecamylamine. The reverse conditions exist for nicotinic receptors on postganglionic neurons. Here, hexamethonium and mecamylamine bind, but curare does not.

**WHAT DO YOU THINK?**

3. What effect, if any, would smoking have on the nicotinic receptors of the ANS?

Muscarinic receptors were so named because they respond to muscarine, a mushroom toxin. They are found in all target organs stimulated by the parasympathetic division and in the few selected sympathetic target cells (e.g., sweat glands in the skin and blood vessels within skeletal muscle). There are different subtypes of muscarinic receptors, which have different effects on various body systems. These different subclasses of muscarinic receptors are either stimulated or inhibited by binding ACh. For example, the binding of ACh to smooth muscle in the gastrointestinal (GI) tract will result in contraction and increased motility (movement) of the muscle, whereas binding of ACh to muscarinic receptors on cardiac muscle pacemaker cells results in decreasing heart rate. The drug pilocarpine (used to treat glaucoma) binds to muscarinic receptors to stimulate ciliary muscles to contract, which facilitates drainage of aqueous humor from the anterior chamber of the eye (see figure 16.15). All muscarinic receptors use second messenger systems (see section 17.5b).

Nicotinic and muscarinic receptors are compared in table 15.4.
WHAT DID YOU LEARN?

16. Where are nicotinic and muscarinic receptors each located?

17. When a neurotransmitter binds to a nicotinic effector, is the effect excitatory (stimulatory) or inhibitory?

15.6c Adrenergic Receptors

LEARNING OBJECTIVES

20. List the neurotransmitters categorized as catecholamines.

21. Name the four adrenergic receptors, and give the locations of each.

Recall from section 4.5b that signaling molecules (e.g., neurotransmitters, hormones) are called ligands when they specifically bind to receptors in the plasma membrane. The class of ligands that bind to adrenergic receptors in neurons is called biogenic amines, or monoamines. One category of biogenic amines is called catecholamines because of the presence of a catechol ring structure in the molecule. Catecholamines include dopamine, norepinephrine, and epinephrine (see section 17.3a).

As mentioned in section 15.6a, the two types of adrenergic receptors are alpha (α) and beta (β) receptors, which may be further divided into subclasses such as α₁, α₂, β₁, and β₂. Target cells with α receptors typically are stimulated, whereas those cells with β receptors may be either stimulated or inhibited in response to binding neurotransmitter (or hormone). (Note that there are exceptions to this rule.) These types of receptors may be further subdivided, as follows:

- **α₁ receptors** are the most common adrenergic receptors of the sympathetic division. These receptors are located within plasma membranes of most smooth muscle cells and stimulate smooth muscle contraction. The specific organs with α₁ receptors include most blood vessels (including those going to the skin, GI tract, and kidneys), arrector pili muscles, uterus, ureters, internal urethral sphincter, and dilator pupillae muscle of the eye. These receptors are involved with vasoconstriction of the above blood vessels, contraction of arrector pili muscles, contraction of the uterine wall, contraction of the ureters, closing of the internal urethral sphincter, and dilation of the pupil, respectively.

- **α₂ receptors** are located throughout the CNS (e.g., brainstem) and decrease norepinephrine release, thereby inhibiting sympathetic activity. Stimulation of these CNS α₂ receptors also causes sedation and analgesia (i.e., the inability to sense pain). The receptors also are in the pancreas to inhibit insulin secretion. In addition, these receptors are located in GI sphincters, and when they are stimulated they constrict the sphincter.
Table 15.5  Adrenergic Receptors

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Alpha (α) Receptors</th>
<th>Beta (β) Receptors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>α₁</td>
<td>α₂</td>
</tr>
<tr>
<td>Primary locations and specific actions</td>
<td>Pancreas (inhibits insulin release)</td>
<td>Heart (both sinoatrial node and cardiac muscle; increases heart rate and force of contraction)</td>
</tr>
<tr>
<td></td>
<td>CNS (decreases norepinephrine release and thus inhibits sympathetic activity; causes sedation and analgesia)</td>
<td>Kidney (stimulates release of renin to increase blood pressure)</td>
</tr>
<tr>
<td></td>
<td>GI sphincters (cause contraction)</td>
<td></td>
</tr>
<tr>
<td>General effect</td>
<td>Excitatory</td>
<td>Inhibitory or excitatory</td>
</tr>
<tr>
<td>Examples of drugs that interact with receptor</td>
<td>Phenylephrine causes vasoconstriction of nasal blood vessels, decreasing nasal secretions.</td>
<td>Clonidine is used to treat high blood pressure by stimulating α₂ receptors in the vasomotor center of the brainstem.</td>
</tr>
</tbody>
</table>

**INTEGRATE**

**CLINICAL VIEW 15.2**  
**Epinephrine for Treatment of Asthma**

*Asthma* is a condition in which airflow into the lung is decreased due to the narrowing of the air passages called bronchioles (see Clinical View 23.6: “Asthma”). Bronchioles contain β₂ receptors, and epinephrine (hormone) binds more effectively to β₂ receptors and causes greater relaxation of the smooth muscles of bronchioles than does norepinephrine. The greater the degree of bronchodilation, the greater the rate of airflow into and out of the lungs. Consequently, epinephrine (not norepinephrine) is the active ingredient in medicines for treating asthma (e.g., the medicine in an *EpiPen*).

**WHAT DID YOU LEARN?**

18. What are the different types of catecholamines?

19. How is it possible for the stimulation of adrenergic receptors to result in either vasoconstriction or vasodilation of selected blood vessels?

15.7 Interactions Between the Parasympathetic and Sympathetic Divisions

Most organs are innervated by both divisions of the autonomic nervous system, and these control the targets continuously to varying degrees. The parasympathetic and sympathetic division effects are compared in table 15.6 and figure 15.11.

15.7a Autonomic Tone

**LEARNING OBJECTIVE**

22. Discuss the nature of autonomic tone and its effects.

The parasympathetic and sympathetic divisions both continuously release neurotransmitter to regulate specific target organs for either sustained stimulation or inhibition, a process referred to as the **autonomic tone**. Activity of an organ may be controlled merely by the change in tone within a single ANS division.