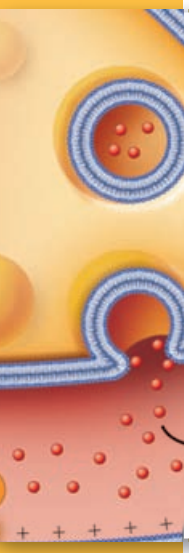
7

Muscles



Major Themes

- Muscle cells shorten on command; no other cells do.
- There are three types of muscle cells: skeletal, cardiac, and smooth.
- Skeletal muscle contracts voluntarily to produce body movements.
- Adenosine triphosphate (ATP), most of which is derived from glucose and fat metabolism, is the energy currency for muscle action.
- Smooth muscle contracts involuntarily to power many internal functions.

Chapter Objectives

Overview of Muscle 228

1. List five functions of muscle tissue.
2. Compare and contrast skeletal, smooth, and cardiac muscle.

Structure of Skeletal Muscle Tissue 231

3. Using a drawing, identify and describe the special features of a skeletal muscle cell, and explain how many such cells, along with connective tissue membranes, are built into a skeletal muscle.

Skeletal Muscle Contraction 233

4. Define *sarcomere* and explain how muscle contraction results from sarcomere shortening.
5. Describe the composition of the thin and thick filaments, and label the parts of the sarcomere.
6. List all the steps involved in muscle contraction, beginning with an action potential in a neuron and ending with the events of cross-bridge cycling.
7. List the steps involved in muscle relaxation.

Muscle Energy 243

8. Identify three uses for ATP in muscle contraction.
9. Explain the benefits and disadvantages of different energy sources (creatine phosphate, glycolysis, and mitochondrial respiration); compare anaerobic and aerobic metabolism.

10. Compare the structure and function of fast glycolytic fibers and slow oxidative fibers.

Case Study: Muscle Energy Metabolism: The Case of Hammid S. 246

11. List different causes of muscle fatigue, referring to the case study.

The Mechanics of Muscle Contraction 248

12. Explain how a stronger contraction results from modifying fiber length and/or recruiting additional motor units.
13. Provide examples of isometric, concentric isotonic, and eccentric isotonic contractions.
14. Discuss the effects of resistance training and endurance exercise on muscles.

Smooth Muscle 252

15. Describe the structural and functional differences between skeletal and smooth muscle.
16. List the steps involved in smooth muscle contraction, including the different types of stimuli that can induce contraction.

Skeletal Muscle Actions 255

17. Identify the prime mover, synergist, and/or antagonist for different body movements at each joint.

The Major Skeletal Muscles 256

18. For each body region (head and neck, upper limb, torso, and lower limb), label the major skeletal muscles on a diagram and indicate their insertion and origin.

"He's had these pains all of his life."

As you read through the following case study, assemble a list of the terms and concepts you must learn in order to understand Hammid's condition.

Clinical History: His mother brought Hammid S., a 10-year-old boy, to a pediatrician's office. The family had emigrated to the United States from Afghanistan 10 months earlier. With the aid of an interpreter she explained that Hammid was becoming increasingly upset by his inability to keep up with the other boys on his soccer team because of the painful muscle cramps that occurred in his legs with strenuous exercise. He had also been complaining that everyday activities requiring significant muscle effort, such as climbing stairs, caused him pain.

She explained further, "He's had these pains all of his life, but not so bad as now. The doctor in Herat told me he probably had liver disease because his urine is red or brown sometimes. But the dark urine always appears after the muscle cramps come. The cramps go away if he rests for a while."

Further questioning revealed that Hammid's older brother and younger sister were not affected by similar symptoms.

Physical Examination and Other Data: Hammid was of normal height and weight for his age, and his vital signs were unremarkable. Muscle size and tone were unremarkable. Mild proximal muscle weakness was present in all extremities and he had difficulty walking on his heels or toes more than 8 or 10 steps because cramps developed in his legs.

Laboratory evaluation revealed abnormally high levels of creatine kinase (a muscle enzyme) in the blood. A presumptive diagnosis of McArdle syndrome (type V glycogen storage disease, due to a genetic deficiency of muscle glycogen phosphorylase, another muscle enzyme) was made and an appointment with a specialist in muscular diseases was arranged.

Need to Know

It is important to understand the terms and concepts listed below before tackling the new information in this chapter.

- Nutrients and ATP ⬅ (Chapter 2)
- Neuron structure, neurotransmitters, and chemical synapses ⬅ (Chapter 4)
- Movements at synovial joints ⬅ (Chapter 6)

Clinical Course: At the specialty clinic, a forearm ischemia test was performed, in which a blood pressure cuff was inflated to cut off blood flow and Hammid was asked to squeeze a rubber ball for a minute or until cramps appeared. Study of blood lactic acid in a forearm vein was abnormal: the normal increase of lactic acid did not occur during the test. A muscle biopsy was performed; it showed increased amounts of glycogen in the muscle fibers and a severe decrease in muscle content of glycogen phosphorylase.

Specialists at the clinic explained to Hammid's parents that his genetic defect was inherited and that no treatment was currently available. The parents were also reassured that although Hammid would have difficulty with strenuous exercise all of his life, he would be unlikely to suffer other problems.

Just as the word *bone* can refer either to an organ or to a tissue, so can the word *muscle;* that is, the biceps muscle (a muscle in the arm) is an organ primarily composed of muscle tissue. We derive the word *muscle* from the Latin *mus* (for "mouse"), a reference to the rippling motion of muscles, which was thought to resemble the movement of mice beneath the skin. In turn, *mus* was derived from earlier Greek, where *mys* (meaning both "mouse" and "muscle") gives us the prefixes *myo-* and *mys-*, which refer to muscle. A **myofilament,** for example, is a specialized cytoskeletal filament of muscle cells. Words referring to muscle tissue may also have the prefix *sarco-*, which is derived from Greek *sarx* (for "flesh"). For example, the cytoplasm of a muscle cell is called the **sarcoplasm.**

Courage is like a muscle strengthened by its use.

Ruth Gordon, American writer and actress (1896–1985)

Overview of Muscle

Muscle comprises about 40% to 50% of body weight. No other cell can do what muscle cells do: they contract (shorten) on conscious command. This ability makes muscle cells responsible for our movements, both visible and invisible: walking, talking, bowel movements, urination, breathing, heartbeats, the dilation and constriction of the pupils of our eyes, and many others. And when we are still—sitting or standing—muscle cells keep us erect.

Functions of Muscle

The core function of muscle is to convert chemical energy into mechanical force. Muscle acts to:

- *Move body parts.* Every movement of our body requires skeletal muscle action, from large movements like walking, to smaller movements like breathing or following a tennis match with our eyes.
- *Maintain body posture.* Although it is not immediately obvious, an uninterrupted sequence of tiny, silent contractions of postural skeletal muscle keeps us erect when we are standing or sitting and keeps our heads from slumping on our shoulders. A related activity is the *stabilization of joints*: In every activity, joints must be stabilized so that they do not swing out of control but operate in a smooth, steady fashion.
- *Adjust the volume of hollow structures.* By their response to unconscious autonomic commands, muscles in the

walls of hollow structures relax to increase volume and contract to decrease it. For example:

- Muscle in the bladder wall relaxes to allow the bladder to expand to accommodate more urine or contracts to expel it.
- Muscle in blood vessel walls relaxes to dilate blood vessels and allow more blood flow or contracts to reduce blood flow.

- *Move substances within the body*. The self-stimulated, automatic contractions of cardiac muscle pump blood through blood vessels; waves of smooth muscle contractions propel intestinal contents down the intestinal tract; and similar waves of smooth muscle contraction power male ejaculation and female orgasm.
- *Produce heat.* Whether it is the conversion of gasoline into vehicular motion or the conversion of glucose into muscle contraction, conversion of energy from one form to another always produces heat as a waste product. The body generates ATP to power muscle contraction. When it does so, about three-fourths of the nutrient energy consumed escapes as heat. Because nearly half of body mass is skeletal muscle, most body heat comes from skeletal muscle contractions. And just as waste heat from an automobile engine is used to warm the car's interior on a frosty day, the heat from muscle contraction is the major source of heat to maintain body temperature. For example, when we shiver with cold, the shivers are involuntary skeletal muscle contractions that generate extra heat to raise body temperature.

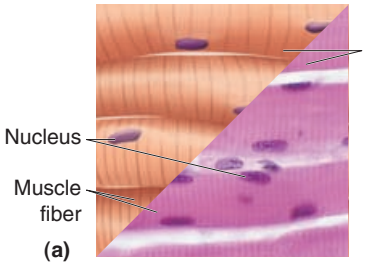
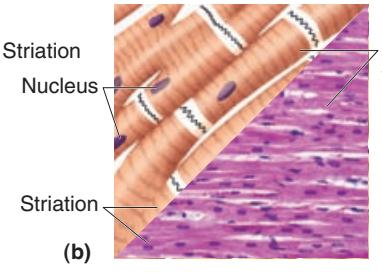
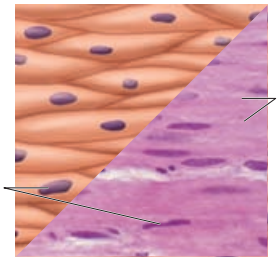
Case Note

7.1. Based on his symptoms, which of the muscle functions just discussed is impaired in Hammid, our patient?

There Are Three Types of Muscle

There are three types of muscle: *skeletal*, *cardiac*, and *smooth* (Table 7.1). Their most important common characteristic

Table 7.1 Muscle Tissue

Characteristic	Skeletal	Cardiac	Smooth
Location	Often attached to bones	Heart	Walls of blood vessels, visceral organs
Appearance	Long, cylindrical fibers	Branching cylindrical fibers	Small cells; sometimes branched
	Thin	Striated	Not striated
	Striated	Single nucleus	Single nucleus
	Multiple nuclei		
	 (a)	 (b)	 (c)
Control	Voluntary	Involuntary	Involuntary
Contraction	Rapid contraction and relaxation	Moderate contraction and relaxation	Slow contraction and relaxation; can maintain for extended periods
Fatigue?	Yes	No	No

is their ability to contract. Their most important differences relate to four qualities:

- *Location*
- *Microscopic appearance*
- Whether or not they are subject to *conscious control*
- The *type of contraction* they generate

Skeletal Muscle Moves the Skeleton

As the name suggests, most **skeletal muscle** is attached to bone and moves the skeleton (Fig. 7.1).

Skeletal muscles form the bulk of our muscle mass and add shape to the body. They make up the body wall, hence the alternate term *somatic muscle* (*soma* = "wall").

Microscopically, skeletal muscle is **striated muscle;** that is, it has cross-stripes (striations) on microscopic examination, an appearance that is intimately related to its function. Mature muscle cells are especially long and thin—up to a foot long—and are typically called **muscle fibers.** Keep in mind throughout this chapter that a muscle fiber is a single mature skeletal muscle cell.

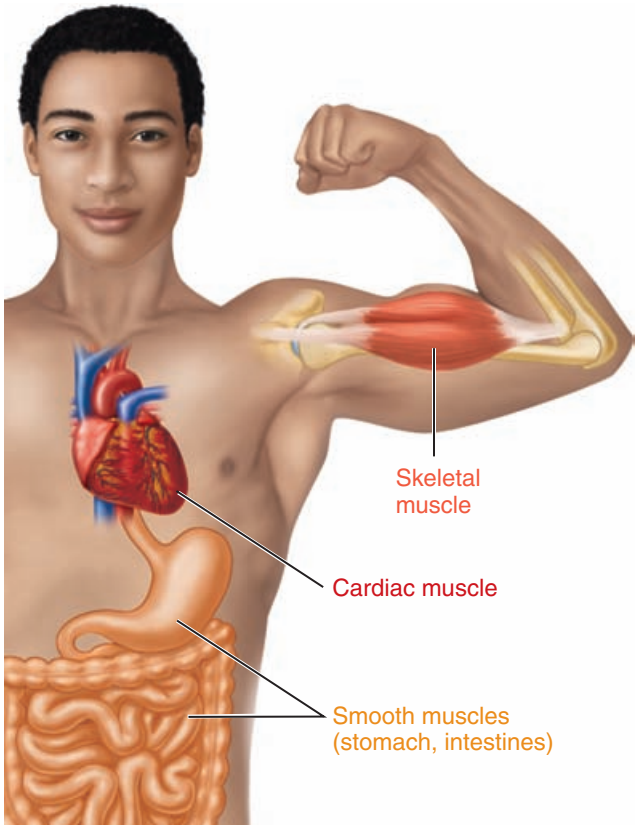


Figure 7.1. Muscles in action. Cardiac muscle (shown in dark red) keeps blood moving, smooth muscle (orange) enables food digestion and urine retention, and skeletal muscle (bright red) moves the body. *Which type of muscle makes up the stomach wall?*

What's more, skeletal muscle is **voluntary muscle;** that is, we can contract and relax it at will. Skeletal muscles can also function outside of our conscious control; for example, the diaphragm contracts and relaxes to keep us breathing while we sleep, and our neck and back muscles maintain our seated posture while our attention is devoted to our work.

Skeletal muscle fibers contract quickly and forcefully and then relax to become ready to contract again. They do not maintain contraction for an extended period of time, and they fatigue after repeated contraction. Further on, we discuss the precise nature of muscle fatigue, which is unique to skeletal muscle; cardiac and smooth muscle do not tire.

Cardiac Muscle Propels Blood through the Body

As its name suggests, **cardiac muscle** tissue is found only in the heart, and accounts for most of its mass (see Fig. 7.1). We perceive cardiac muscle contractions as our heartbeat, which propels blood through the blood vessels of the body.

Microscopically, cardiac muscle is striated, like skeletal muscle. Cardiac muscle cells are much shorter than skeletal muscle fibers, but they are branched and interconnected. The end of one branch is connected intimately to another, producing long cardiac muscle fibers. The result is that cardiac muscle is a network that in many ways behaves like a single huge muscle cell.

Of course, cardiac muscle is **involuntary muscle:** we cannot control its contractions by force of will. That said, some things we deliberately do—such as engaging in meditation—can slow our heartbeat, whereas other things—such as vigorous exercise—can increase it.

Like skeletal muscle fibers, cardiac muscle cells contract quickly and then relax. However, unlike skeletal fibers, they do not fatigue. We will return to cardiac muscle in ➦ Chapter 11 in our discussion of the heart.

Smooth Muscle Powers the Actions of Viscera

Smooth muscle tissue is found in thick layers in the walls of hollow organs such as blood vessels, the urinary bladder, the uterus, and the intestines (Fig. 7.1). The intestines and other abdominal organs are frequently described as the *viscera;* therefore, smooth muscle is also called *visceral muscle.* As noted earlier, it adjusts the volume of hollow structures and helps move substances—from food to blood—throughout the body.

Smooth muscle is named for its microscopic appearance: It is a **nonstriated muscle;** that is, it has a uniform,

smooth appearance without cross-striations. As discussed further on in this chapter, its lack of striations is intimately related to its function.

Smooth muscle is involuntary: we do not command the wall of our stomach to relax to accommodate a large meal. It just happens as the stomach responds automatically to the mechanical stimulation of food bulk, which is but one of many stimuli that govern smooth muscle function. Usually, smooth muscle contracts slowly and can maintain the contraction over a long period. Generally, smooth muscle fibers do not fatigue.

Case Note

7.2. Which type of muscle is affected by Hammid's disease?

All Muscle Tissue Is Extensible

A final important characteristic of all three types of muscle tissue is its *extensibility*—its ability to stretch without tearing. Consider what happens when you open your mouth wide to bite an apple. The muscle that normally brings the jaws together must relax and lengthen in order to permit you to do it. If this muscle were not extensible, your simple action would cause the muscle to tear. The stretchable internal organs of the body—the heart, the bladder, the intestines, the uterus, and so on— are made of cardiac or smooth muscle, which also has this property. By contrast, if a surgeon were to inadvertently stretch the tissue of the brain, the liver, the spleen, or the kidney, it would tear.

7.1 What is the name of a mature skeletal muscle cell?

7.2 Name the two types of striated muscle.

7.3 Name two types of involuntary muscle.

7.4 Which type of muscle tissue experiences fatigue?

Structure of Skeletal Muscle Tissue

Since muscle cells are unique in their ability to contract and lengthen without tearing, it's not surprising that they have an unusual path of development and unique structural features. Again, the structure of cardiac muscle is discussed in ➦ Chapter 11, and that of smooth muscle is discussed later in this chapter.

Myoblasts Fuse to Form Muscle Fibers

During embryonic development, stem cells produce immature muscle cells called **myoblasts** (*blast* = "precursor"). Several myoblasts fuse together to produce each skeletal muscle fiber, so each muscle fiber contains multiple nuclei. Some muscle stem cells persist into adulthood, hidden between the muscle cell membrane and the surrounding connective tissue. These adult muscle stem cells are called **satellite cells,** because of their location at the muscle fiber's periphery. Although mature skeletal muscle cells are fully differentiated and cannot divide, satellite cells can be activated by exercise, injury, or disease to produce new myoblasts that fuse to form new muscle fibers. However, satellite cell activity is not sufficient to repair major skeletal muscle injuries.

Remember This! **Adult muscle stem cells are called satellite cells; they produce myoblasts, which fuse to form skeletal muscle fibers.**

The Structure of a Muscle Cell Reflects Its Function

The following elements of a skeletal muscle cell (fiber) are essential to its function (Fig. 7.2A):

- The cell membrane is called the **sarcolemma.** Like the membrane of any body cell, it acts to contain the cell's contents and shield it from the extracellular environment. As discussed below, this function is especially important in muscle contraction.
- The sarcolemma in muscle cells not only surrounds the cytoplasm but also tunnels deep into the interior of the muscle fiber as a network of **T-tubules.** Action potentials travel down these T-tubules, which enable them to reach every part of the fiber virtually simultaneously to trigger a coordinated muscle contraction.
- Multiple cigar-shaped nuclei reside along the periphery of the cell, immediately beneath the sarcolemma. This location keeps them out of the way of muscle fiber contractions.
- The cytoplasm of the muscle cell, the **sarcoplasm,** is densely packed with the following structures, which are described in more detail further on:
  - **Myofibrils.** These slender, threadlike organelles accomplish the work of muscle contraction. Each

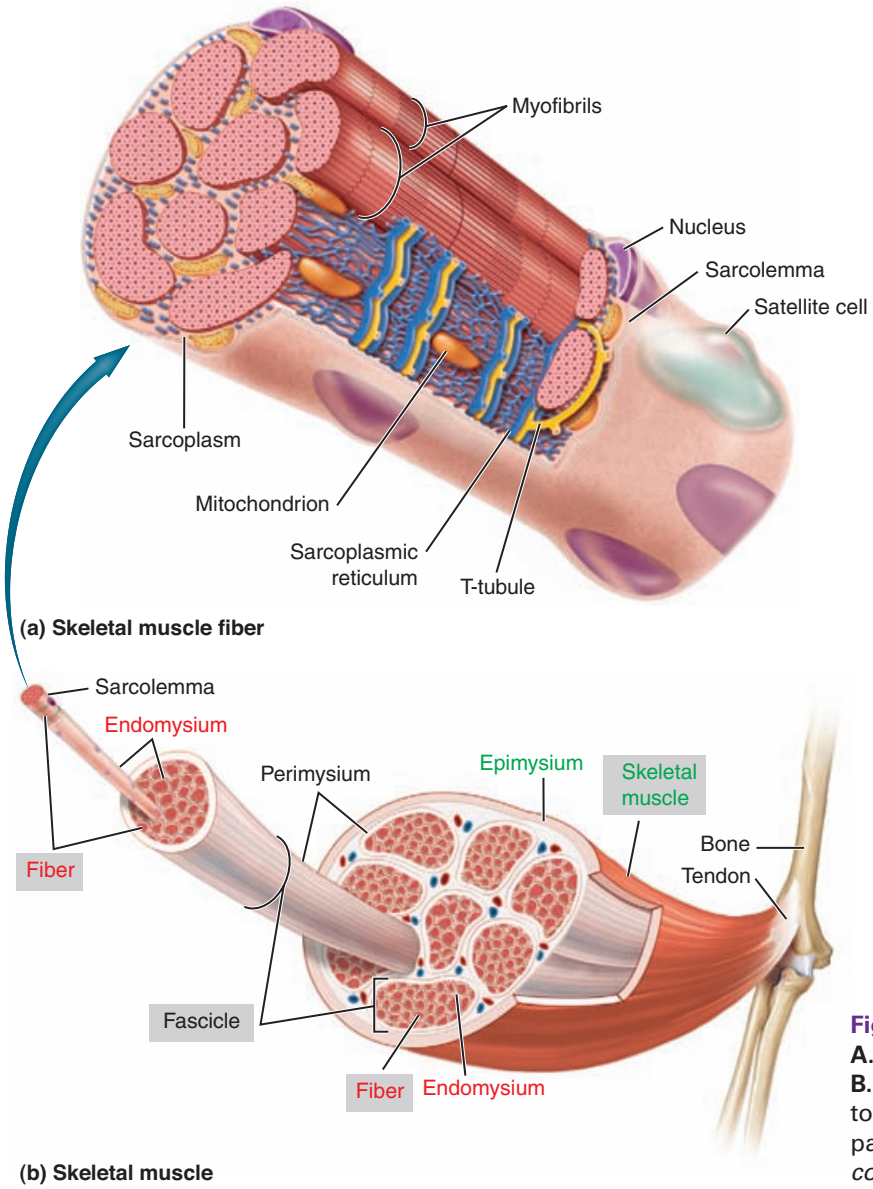


Figure 7.2. Skeletal muscle cells and muscles. **A.** Muscle fibers are packed with myofibrils. **B.** Numerous muscle fibers are packed together to form a fascicle, and many fascicles are packed together to form a muscle. *Name the connective tissue layer surrounding a fascicle.*

myofibril is a bundle of different proteins that runs the entire length of the muscle fiber. Each muscle fiber contains hundreds or thousands of myofibrils.

- **Sarcoplasmic reticulum** (SR). This organelle is a lacy network of fluid-filled tubules similar to the smooth endoplasmic reticulum in other body cells. It stores calcium ions necessary for muscle contraction. T-tubules are in close contact with the SR, separated by a small region of intracellular fluid.
- **Mitochondria.** These organelles generate the ATP that fuels muscle contraction.
- **Myoglobin** (not shown on Fig. 7.2), an iron-containing compound, stores oxygen used to generate energy for muscle contraction.

Case Notes

7.3. Hammid's blood chemistry showed elevated levels of creatine kinase, which is usually confined to the interior of the muscle cell. The cell membrane of his muscle cells must have ruptured, releasing the cell contents into his blood. What are the specific terms used to describe the muscle cell membrane and cytoplasm?

7.4. Hammid's red urine reflects the presence of the compound that stores oxygen inside skeletal muscle cells. Name this compound.

Connective Tissue Wraps Muscle Fibers, Fascicles, and Whole Muscles

Individual skeletal muscle fibers are delicate, and every one of them is wrapped by a sheath of connective tissue called the **endomysium,** which covers, insulates, supports, and protects them (Fig. 7.2B). Satellite cells reside between the endomysium and the sarcolemma.

Groups of about 100 muscle fibers are formed into structural and functional bundles called **fascicles.** These are wrapped with a thicker, tougher sheath of connective tissue called the **perimysium.**

In turn, groups of fascicles form muscles, which are wrapped by a tough and very substantial outer layer of connective tissue, the **epimysium.** As a muscle terminates near its attachment to bone, its epimysium binds together to form a tough and exceptionally strong collagenous tissue that attaches muscle to bone. When formed into a thick, tough cord for attachment at a single point, it is called a **tendon** (see Fig. 7.2B); you learned about tendons attaching to bone in ⬅ Chapter 6. When formed into a sheet for broader, linear attachment, the epimysium is called an **aponeurosis.**

7.5 Are tendons examples of epithelial tissue or connective tissue?

7.6 What is the difference between a muscle fiber, a fascicle, and a myofibril?

7.7 What is the name of the membrane extensions that dip deep into the sarcoplasm?

7.8 What is the difference between the perimysium and the endomysium?

Skeletal Muscle Contraction

You're reading this chapter, and it's time to turn the page. As you lift your hand, you don't consciously direct your muscles to contract to produce your movements. It just happens. But how?

A Motor Unit Is a Motor Neuron and the Muscle Fibers It Controls

Contraction of a skeletal muscle requires communication. A **somatic motor neuron** carries a signal that stimulates contraction in skeletal muscle (a *visceral motor neuron* carries a similar signal to smooth muscle or glands). The cell bodies of motor neurons are located in the brain or spinal cord and send long cytoplasmic extensions called axons out to communicate with muscle fibers. As shown in Figure 7.3, the axon of a motor neuron branches toward its end to make contact with several muscle fibers. These branches are called *axon terminals*. A **motor unit** comprises a somatic motor neuron and the skeletal muscle fibers it controls.

Muscles that require small, highly precise movements (as in the muscles that control eye movement) may have as few as three muscle fibers per motor unit. Muscles responsible for large, powerful movements (in the thigh, for example) may have several thousand muscle fibers per motor unit.

Case Note

7.5. What type of neuron carries the signal to Hammid's muscles?

Motor Neurons Connect to Muscle Fibers at the Neuromuscular Junction

Near its tip, each axon terminal enlarges into a pancake-like swelling called a *synaptic bulb,* which lies flat on the surface of the muscle fiber. A single synaptic bulb meets a skeletal muscle fiber at a chemical synapse called the **neuromuscular junction** (Fig. 7.3B). The components of the neuromuscular junction are (Fig. 7.3C):

- The *synaptic bulb* of the neuron
- The *motor end plate* of the muscle fiber, which is that part of the fiber's sarcolemma across from the synaptic bulb
- The *synaptic cleft*, an exceedingly narrow space that separates the synaptic bulb from the motor end plate—the nerve and muscle fiber do not actually touch.

Recall from ⬅ Chapter 4 that chemical synapses use neurotransmitters to transmit the signal between two adjacent cells—in this case, the motor neuron and the muscle fiber. In all synapses the basic process is the same: in response to an action potential in the presynaptic cell, neurotransmitter is released into the synaptic cleft; it then binds to specific receptors on the postsynaptic cell, altering its electrical activity. The neuromuscular junction is more specific—an action potential in the presynaptic cell *always* results in an action potential

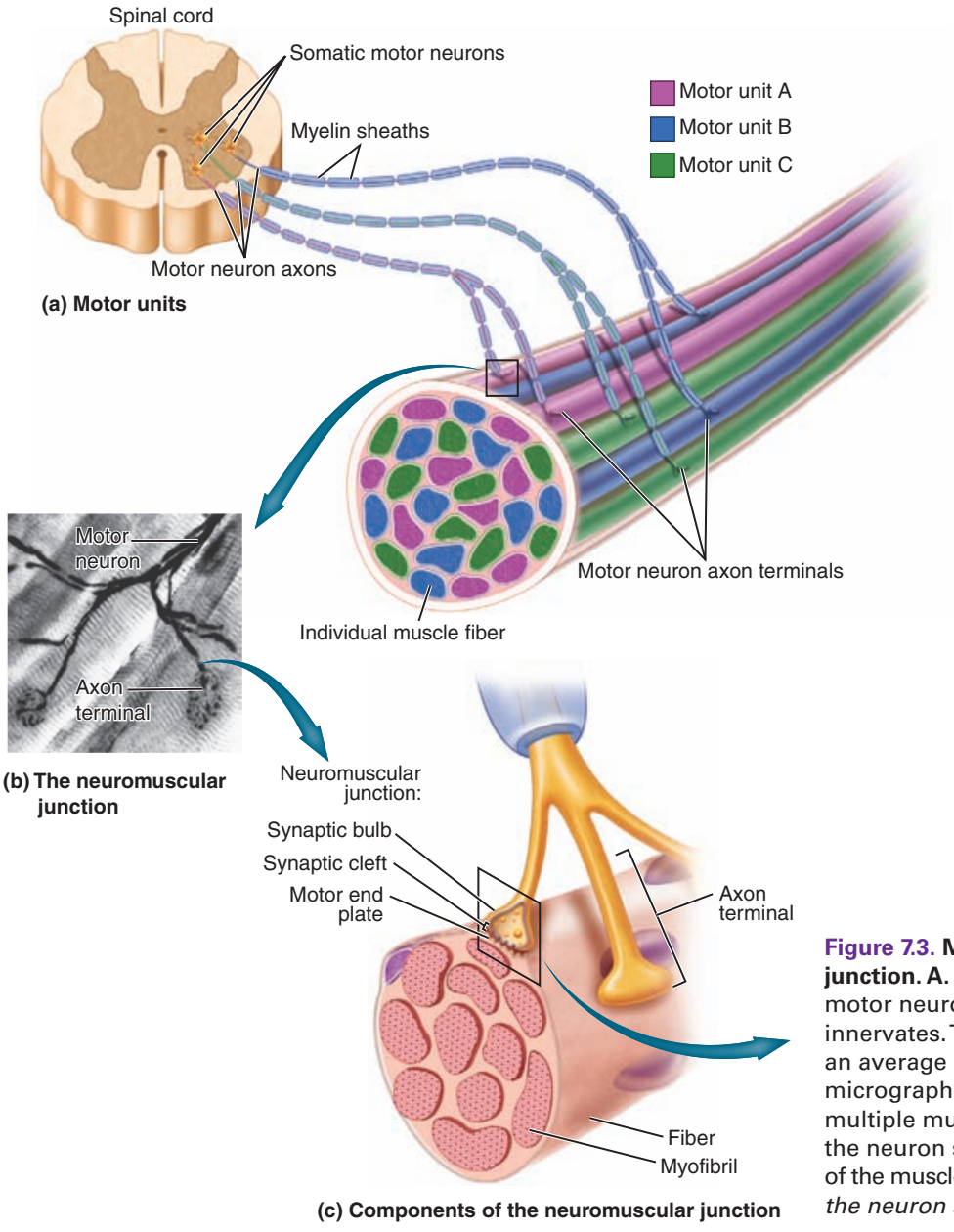


Figure 7.3. Motor units and the neuromuscular junction. A. A motor unit consists of a somatic motor neuron and the skeletal muscle fibers it innervates. This figure shows three motor units; an average muscle will have many more. **B.** This micrograph shows an axon branching to supply multiple muscle fibers. **C.** The synaptic bulb of the neuron synapses with the motor end plate of the muscle fiber. *Name the space that separates the neuron and the muscle cell.*

in the postsynaptic cell. Moreover, every skeletal neuromuscular junction uses the same neurotransmitter (*acetylcholine*) and the same neurotransmitter receptor—the *nicotinic cholinergic receptor* (Fig. 7.4)

This receptor is a ligand-gated ion channel ← (Chapter 4), which opens to allow sodium (Na^+) ions to enter the cell when acetylcholine (the ligand) is bound to it.

The events are as follows:

1. The action potential arrives at the synaptic bulb of the somatic motor neuron (the presynaptic cell). The resulting depolarization triggers acetylcholine release into the synaptic cleft.
2. Acetylcholine (ACh) encounters one of two proteins. Some molecules meet with and are inactivated by *acetylcholinesterase*, an enzyme present in the synaptic cleft and embedded in the sarcolemma. This enzyme is always active, but it cannot keep up with ACh release from firing neurons, so ACh accumulates in the synaptic cleft. The neurotransmitter molecules that escape acetylcholinesterase's grasp meet and bind with a second protein, cholinergic nicotinic receptor in the motor end-plate membrane (the postsynaptic cell).
3. ACh binding opens the channel in the nicotinic receptor. Na^+ entry depolarizes the membrane enough to

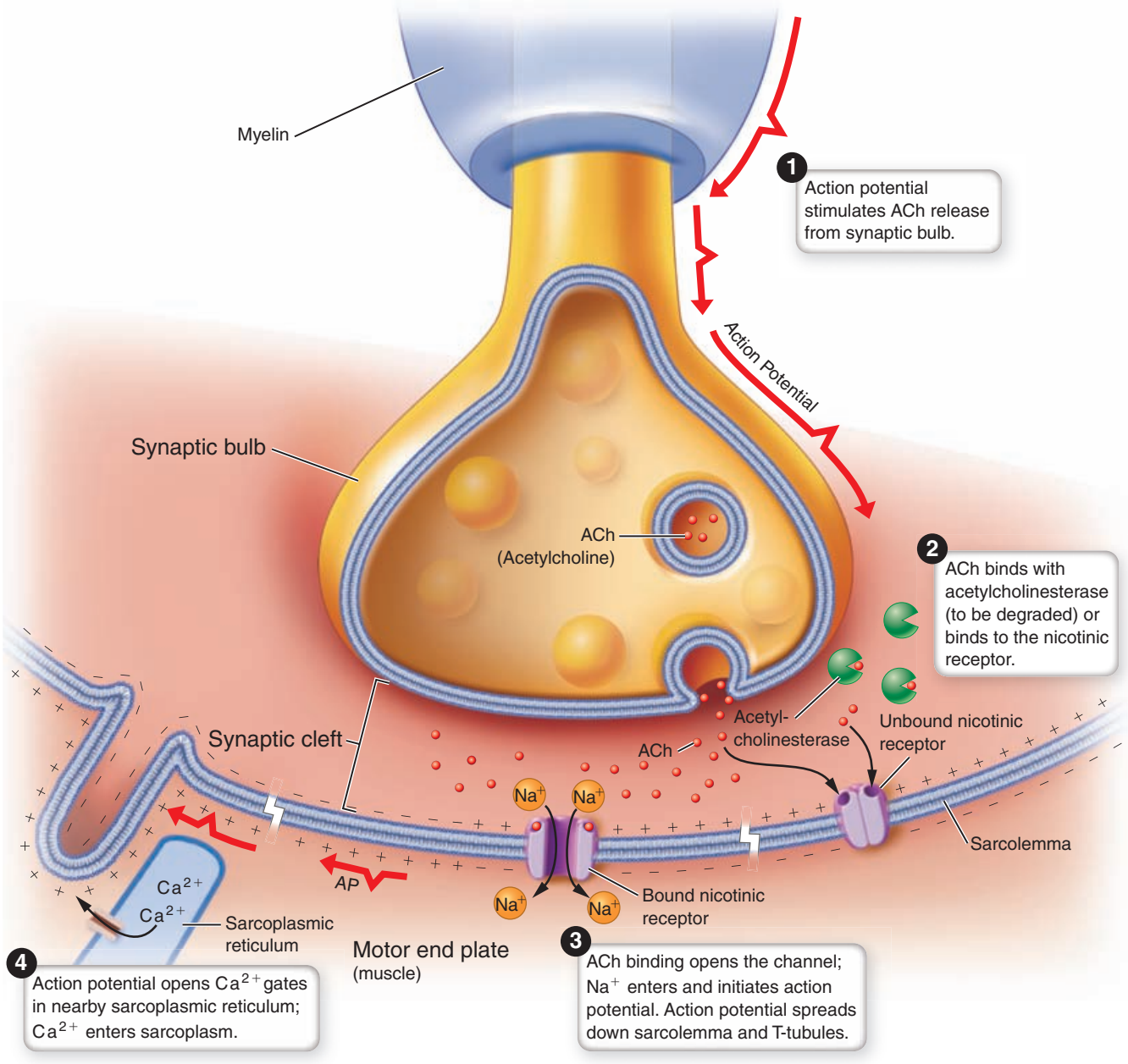


Figure 7.4. Events at the neuromuscular junction. An electrical signal (an action potential) travels down the motor neuron. A chemical signal (ACh) carries the signal across the synaptic cleft and initiates an electrical signal (an action potential) in the muscle cell. *Name the enzyme that terminates ACh's action.*

cause an action potential. The action potential sweeps rapidly over the sarcolemma and races through the network of T-tubules deep within the cell.

4. The action potential triggers the opening of calcium gates in the membrane of the sarcoplasmic reticulum (SR). This releases calcium ions from the sarcoplasmic reticulum (SR) into the sarcoplasm. A specialized calcium (Ca^{2+}) transporter called the *calcium pump* actively transports Ca^{2+} back into the SR. However, in a contracting fiber, the pump cannot keep up with Ca^{2+} release, so Ca^{2+} accumulates in the sarcoplasm. As shown later, it is these calcium ions that stimulate muscle contraction.

The function of chemical synapses can be affected by disease or manipulated or inactivated by drugs or poisons—see the nearby Clinical Snapshot, titled *Beauty and the Beasts,* for more information.

CLINICAL SNAPSHOT

Beauty and the Beasts: Attacking the Neuromuscular Junction

In 2006, untold numbers of women and men voluntarily poisoned the neuromuscular junction of certain facial muscles in order to rid themselves (temporarily) of frown lines. The poison? A toxin, marketed under the name Botox©, which is derived from the anaerobic bacterium *Clostridium botulinum*. Botox©, a protein, is one of the most potent toxins known, one that paralyzes muscles by preventing them from receiving nerve action potentials.

In clinical medicine *C. botulinum* poisoning, or botulism, is a serious, sometimes fatal paralytic condition that is most commonly encountered after ingestion of insufficiently sterilized (undercooked) home-canned meats, fish, vegetables, and fruits contaminated with *C. botulinum*. Botulism may also occur as a consequence of wound infection. The term *botulism* derives from Latin *botulus,* meaning "sausage": the name reflects the fact that the illness was initially recognized as resulting from consumption of contaminated sausage.

C. botulinum toxin acts at the *nerve* side of the neuromuscular junction to prevent synaptic vesicles in the axon from releasing their ACh into the synaptic cleft. If an action potential arrives at the synapse and no ACh is released into the synaptic cleft, the action potential is extinguished without being transferred to muscle. Botulism is characterized by muscle paralysis, which first affects the eyes (double vision, inability to focus) and speech (slurred words) and may cause fatal respiratory paralysis.

However, in small, local doses, the toxin causes limited muscle paralysis, which achieves a pleasing cosmetic effect by relaxing the facial muscles associated with facial wrinkles. For example, following an injection of Botox into the frontalis muscle of the forehead, frown lines disappear; they do not reappear until the effect of the toxin wears off in 4 to 6 months. Botox is also used therapeutically to prevent the muscle spasms that accompany migraine headaches, facial tics (involuntary or habitual contraction of facial muscles), and cervical dystonia (abnormal contractions of neck muscles that move the head).

Other animals exploit the fragility of the neuromuscular junction to paralyze their prey. For example, Taiwanese cobra venom contains a toxin that binds tightly to the ACh receptor on the *muscle* side of the synapse and prevents ACh from binding, which interrupts propagation of the signal. Conversely, venom of the black widow spider causes motor axons to release all of their stored ACh, which overwhelms muscle receptors and interferes with controlled signal transfer across the synapse.

Neuromuscular junction toxins. Botulinum toxin is used to treat frown lines and other facial wrinkles.

Recall from Chapter 4 that signals can be electrical or chemical. The sequence involved in stimulating muscle contraction is as follows:

1. An electrical signal in the somatic motor neuron
2. A chemical signal (ACh) in the synapse
3. An electrical signal in the sarcolemma
4. A chemical signal (calcium) in the sarcoplasm.

But how does a chemical signal—calcium—initiate force generation in the muscle fiber? In order to answer this question, we must delve deeper into the microscopic structure of the muscle fiber, paying particular attention to the myofilaments.

Case Note

7.6. What chemical is released by Hammid's somatic motor neurons to convey nerve signals to muscle cells?

Sarcomeres Are the Functional Units of Myofibrils

Recall that myofibrils are the organelles within the muscle fiber that accomplish the work of muscle contraction. To understand how they contract, we must examine their unusual structure. Each myofibril is, in essence, a bundle of two types of long **myofilaments:** *thick filaments* and *thin filaments*. You can visualize their precise arrangement, which is essential to their function, by imagining myofilaments as thick and thin pencils. Here's how:

- Imagine that the thick filaments (thick pencils) are sharpened on both ends and that the thin filaments (thin pencils) are sharpened on one end and with an eraser at the other (Fig. 7.5A).
- Next, imagine holding a bundle of thin pencils in each hand, with the erasers pointing outward and the sharpened tips pointed at one another.

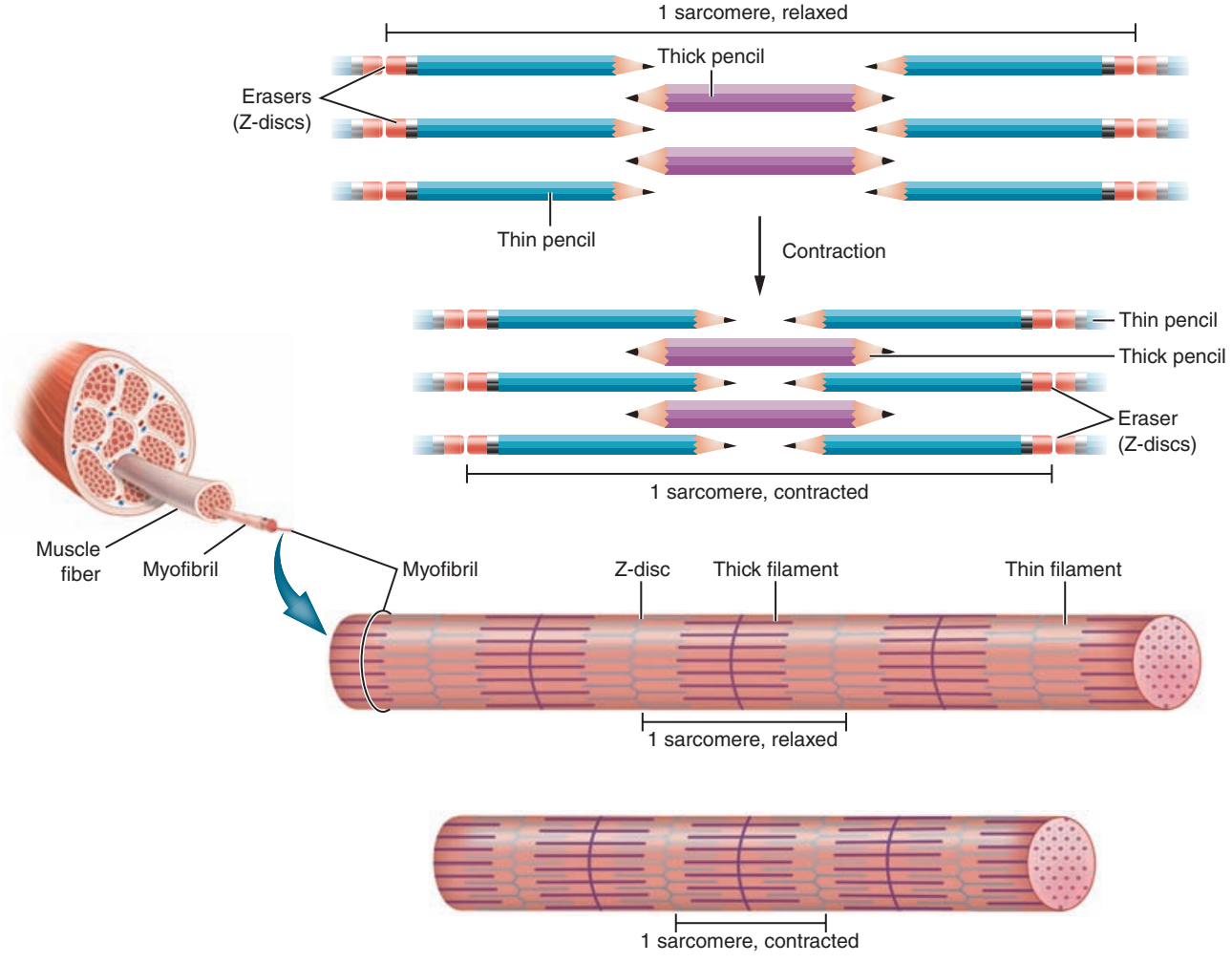


Figure 7.5. Myofibrils are composed of myofilaments. Pencils can be used to model a sarcomere. When the overlap between the pencils increases, the sarcomere shortens. A myofibril consists of many sarcomeres lined up end to end. When individual sarcomeres shorten, the entire muscle fiber (and thus the muscle) shortens. *Which structure is the same length as the muscle—the sarcomere or the myofibril?*

- Now, imagine placing a bundle of thick pencils (sharpened at both ends) between the two bundles of thin pencils. Notice that pointed ends of the thick pencils in this middle bundle face pointed ends of the thin pencils on either side.
- Finally, imagine pushing the thin pencil bundles into the thick pencil bundle in such a way that the thick and thin sharpened tips overlap slightly like interlocked fingertips.

And there you have it: a pencil replica of one **sarcomere,** which is the basic unit of skeletal muscle (Fig. 7.5B). A muscle fiber contains thousands of end-to-end sarcomeres, each a set of interdigitated bundles of thick and thin "pencils" joined at the "erasers" on each end. The joined eraser ends of the thin pencils are analogous to the **Z-discs** of a myofibril, which are found on either side of the sarcomere.

To imagine muscle contraction, imagine sliding the two sets of thin pencils toward each other over the center bundle of thick pencils. As the *overlap* of thick and thin pencils *increases*, the length of the whole sarcomere shortens. This is the essence of muscle contraction— the degree of *overlap* of thick and thin bundles increases as the sarcomere contracts, but the length of each thick and thin myofilament remains unchanged. This model of muscle contraction is called the *sliding filament mechanism,* since filaments are sliding over each other.

A single sarcomere is very small, only a few micrometers in length, but sarcomeres lined up end to end produce a myofibril that runs the entire length of a muscle fiber (Fig. 7.5B). As each sarcomere shortens, the entire myofibril shortens, the muscle fiber shortens, and thus the muscle shortens. Voila! Muscle contraction. Later we will see how the contraction of every sarcomere, myofibril, and muscle fiber of a motor unit occurs at the same time—a property that ensures smooth contraction.

The striated appearance of skeletal muscle examined under a light microscope is an orderly series of light and dark bands produced by the overlap of thick and thin filaments and the end-to-end junctions of sarcomeres. Details are presented in the accompanying Basic Form, Basic Function box, titled *How the Muscle Got Its Stripes.*

Remember This! **During muscle contraction, sarcomeres and myofibrils shorten, but myofilaments do not change in length.**

Myofilaments Are Composed of Contractile Proteins

The molecular structure (form) of thick and thin filaments is essential to their contractile nature (function) (Fig. 7.6).

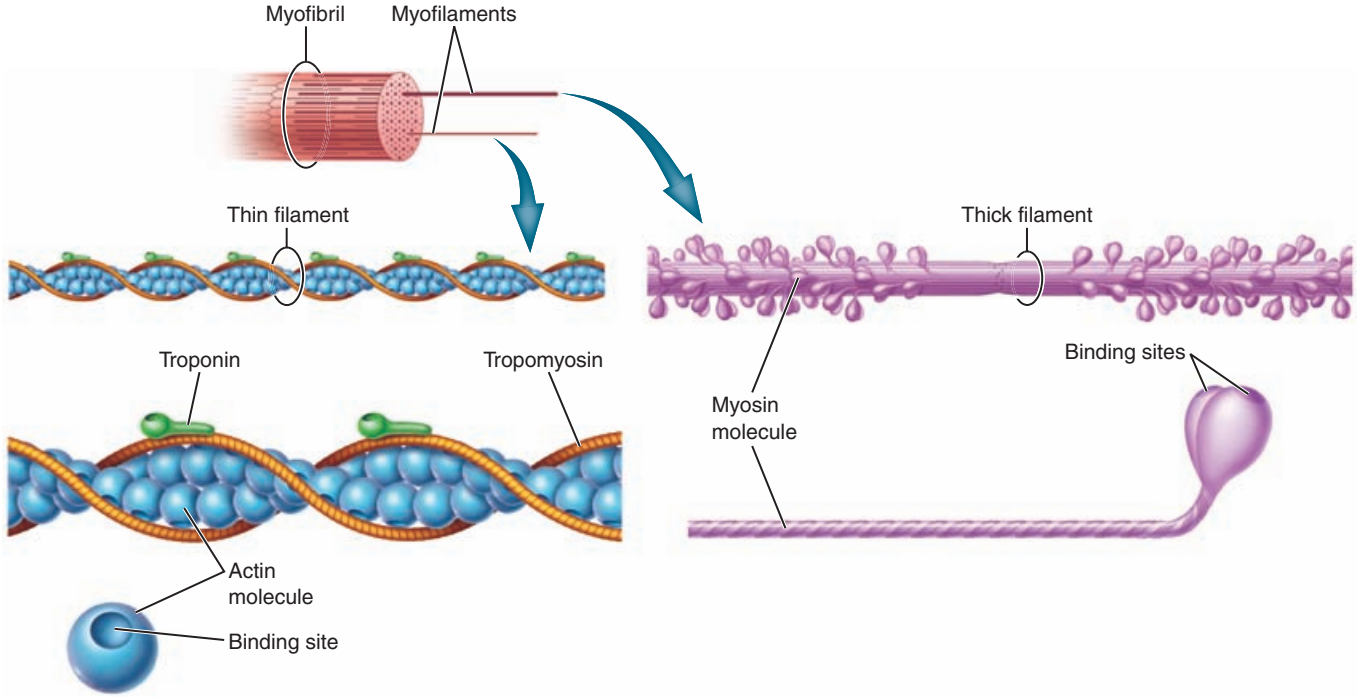


Figure 7.6. Thick and thin filaments. Thick filaments are composed of myosin molecules; thin filaments of actin, troponin, and tropomyosin. *Which protein covers the myosin binding site on the actin molecules?*

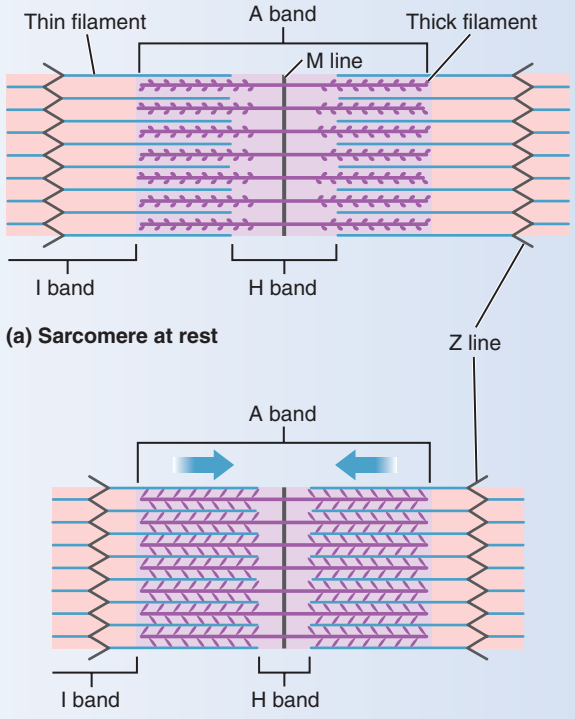
BASIC FORM, BASIC FUNCTION

How the Muscle Got Its Stripes

Recall that striated muscle can be identified by its stripes; it is crossed by alternating light and dark microscopic bands (stria) when examined under a light microscope. The stripes are also useful for another reason—they can help us visualize the minute movements of thick and thin filaments during muscle contraction.

The dark bands, called A bands, are dark because they contain the more opaque thick filaments. The light bands, called I bands, are light because they are composed exclusively of thin filaments. Recall, however, that thin and thick filaments overlap. The dark (A) bands are darkest on each end, where they overlap with thin filaments: this packing of thick and thin filaments blocks the most light. The H zone is the relatively paler region within the A band where only the thick filaments are present. In the center of the light (I) band is a zigzag line; which is the Z-disc, where the bundles of thin fibers meet and which marks the place where sarcomere units join together.

Note that a sarcomere is the space between Z-discs and is formed by half of a light (I) zone at each end and dark (A) band in the center; and that the light (I) zone is formed of the butting ends of two sarcomeres. Note further that when a sarcomere shortens, the Z-discs move closer together, I bands shorten, but the A band remains the same length. Why? Because the A band is a thick filament, which always stays the same length. The I bands, conversely, are thin filaments that do not overlap with thick filaments. As we increase the overlap between thick and thin filaments, more of the thin filament slides into the A band, where it is obscured by the thick filaments. Finally, what happens to H zone, representing thick filaments not overlapping with thin filaments? As with the I band, it shrinks as the sarcomere shortens.



Muscle fiber zones and lines.

Thick filaments are bundles of **myosin** protein. Each molecule of myosin is composed of a long shaft (the tail), one end of which terminates in two globular *heads*, somewhat like a two-headed golf club, one head up the shaft a bit from the other. Each myosin head has two important binding sites, one for ATP and one for thin filaments. When a myosin head is bound to the thin filaments it forms a *cross-bridge*. Many myosin molecules, with their heads pointing in opposite directions, are bundled together to form a thick filament. The molecules overlap like golf clubs taped together to form a chain, with the heads protruding over much of the length—at one end the shafts of the myosin molecules are joined end to end to form the headless central segment of the thick filament.

Thin filaments are composed of three proteins—*actin*, *tropomyosin*, and *troponin*. The main constituent is **actin,** a small globular protein. Each thin filament contains two long strands of actin molecules that are twisted together, much like a necklace composed of two intertwined strings of pearls. Each actin molecule (that is, each "pearl") contains a binding site for a myosin head on a thick filament. In the resting state, however, this binding site is covered by **tropomyosin,** which prevents myosin binding until a signal for contraction arrives from the nerve that innervates the muscle. **Troponin,** the third constituent, controls

the tropomyosin molecules, keeping them in place over the binding sites in relaxed muscle but moving them out of the way for contraction to occur.

Remember This! **Levels of skeletal muscle organization, from largest to smallest, are: muscle → fascicle (bundle of muscle fibers) → muscle fiber (muscle cell) → myofibril (bundle of myofilaments) → myofilament (strands of contractile proteins) → contractile protein.**

Sarcomeres Shorten via the Cross-Bridge Cycle

Recall from above that the thick and thin myofilaments themselves do not shorten; they merely slide by one another in a way that shortens the total length of the sarcomere (and, of course, the myofibril itself). In the pencil analogy, this process is accomplished by sliding the bundles of thin pencils toward each other over the bundle of thick pencils. In the muscle cell, the task of sliding the bundles toward each other is the job of the myosin heads. They succeed in producing this movement via a series of three events collectively called the *cross-bridge cycle:*

- cross-bridge formation
- the power stroke
- cross-bridge detachment

The *power stroke* is the part of the cycle in which the thin filament actually moves. Of the body's many molecular movements, this is among the strangest and most effective. So let's take a close look at how the power stroke occurs before considering the cross-bridge cycle as a whole.

The key operators in the power stroke are the myosin heads. Each head serves as a claw that grabs a "pearl" of actin on a thin filament, anchors itself to it, and snaps backward, pulling the thin filament along the myosin tail a short distance. After this short pull, the myosin heads release, recock, and reattach to another actin pearl further along the thin filament, ready to snap backward again. In this way, the thick and thin filaments ratchet along one another, like someone (the thick filament) pulling up a rope (the thin filament) arm over arm.

Now let's review the full sequence of events that produce muscle contraction (Fig. 7.7). In a muscle fiber at rest, myosin-binding sites on actin molecules are covered by tropomyosin. In response to an action potential in the sarcolemma and T-tubules, Ca^{2+} is released from the sarcoplasmic reticulum (SR). Ca^{2+} binds to and activates troponin, which moves tropomyosin out of the way, exposing the myosin-binding site on each actin molecule (steps 1 to 3 on Fig. 7.7). Once these binding sites are exposed, the cross-bridge cycle (steps 4 to 7) can begin.

Cross-bridge formation occurs when "energized" myosin heads bind actin (step 4). Why do we characterize the myosin heads as energized? Recall from ← Chapter 2 that energy is released when ATP is cleaved into adenosine diphosphate (ADP) and phosphate. In a resting muscle fiber, the ATP has already been cleaved and the products, ADP and phosphate, are bound to the myosin heads. The energy released by ATP cleavage is stored in the "cocked" position of the myosin heads; that is, the myosin head is energized (step 4).

This stored energy is used in step 5, the power stroke, to pivot the myosin heads and move the thin filament. The ADP and phosphate molecules diffuse away immediately after the power stroke, but the cross-bridge remains in place.

The final step in the cross-bridge cycle, detachment, can occur only with the help of additional ATP. Only when a fresh ATP molecule binds to the myosin head (step 6) does the myosin head release from the actin, ready to begin another cross-bridge cycle (step 7).

Cross-bridge cycling occurs in waves, somewhat like a centipede's gait, so that the sliding motion is smooth, not jerky, as it would otherwise be if every myosin head pulled simultaneously like a rowing team. Such smooth waves of molecular increments, repeated quickly thousands of times, cause muscle fibers to shorten. Also, at any point in the contraction, some of the myosin heads are attached to the actin, so that the thin filaments cannot slide back to their original positions.

Case Note

7.7. Hammid's parents were told that his muscles could not get enough energy (i.e., ATP) for prolonged effort. Where does ATP bind in the myofilament?

It might help you to remember the events of the cross-bridge cycle if you understand that **rigor mortis,** the muscle stiffening that begins a few hours after death, is due to the lack of ATP. In death, the body can no longer generate ATP. Therefore the cross-bridge cycle can proceed up to step 5, where the myosin heads are firmly bound to the actin binding sites. And there things stop: relaxation cannot occur because, without a fresh ATP molecule, the myosin heads cannot detach from the

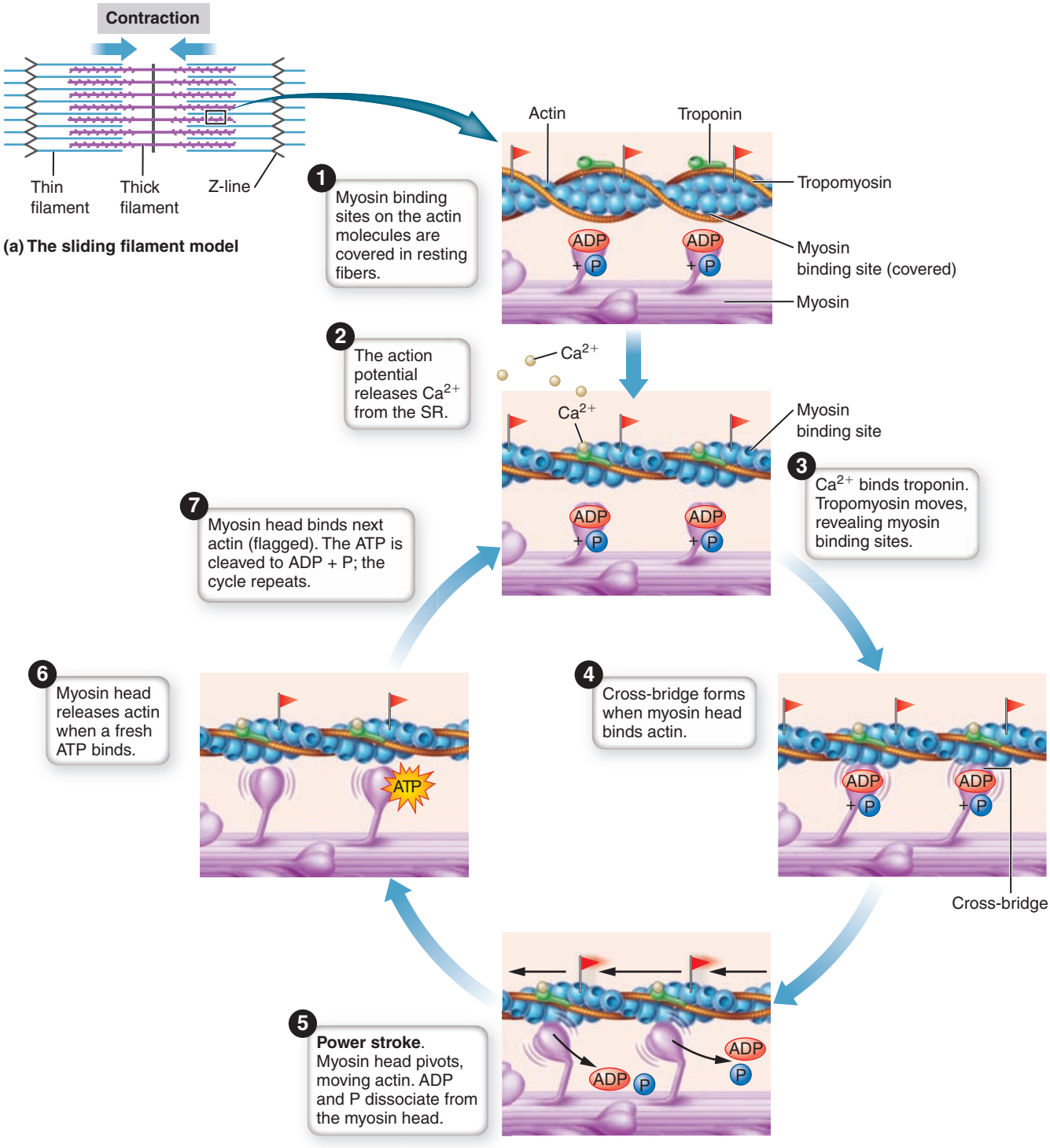


Figure 7.7. Muscle contraction. A. Thick filaments pull thin filaments toward each other during muscle contraction. **B.** The steps in muscle contraction. Notice that the flagged actin molecule has moved (from step 4 to step 6) relative to the thick filament head. *Does ATP bind to actin or myosin?*

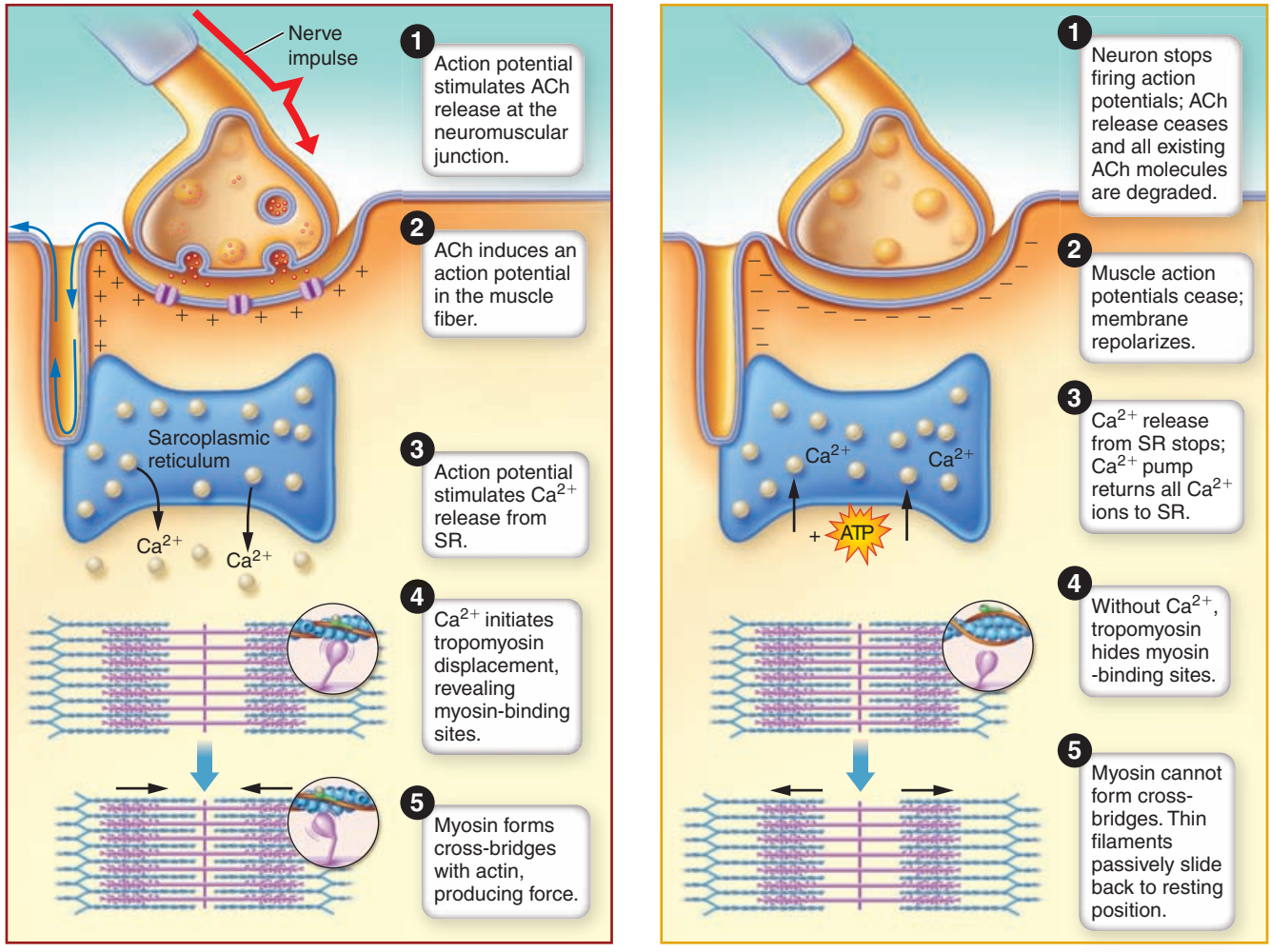
actin. Rigor mortis loosens its death grip on the skeleton after about 24 hours, as enzymes escape from lysosomes and digest myofibrils, allowing muscle to relax.

Remember This! **ATP binding causes the cross-bridge to release. The energy from ATP cleavage is necessary for the power stroke.**

Muscle Relaxes When Cross-Bridge Cycling Ceases

We have now covered all of the elements of a successful muscle contraction, from the arrival of an action potential at the neuromuscular junction to the cross-bridge cycle. You can review these events in Figure 7.8A. Muscle relaxation, an equally important component of any muscle contraction, is essentially the reverse of these steps (Fig. 7.8B).

1. Without continued action potentials in the motor neuron, ACh release ceases. The constant efforts of acetylcholinesterase finally degrade all ACh molecules in the synaptic cleft.
2. Without ACh, the nicotinic receptor channels close, and action potentials in the sarcolemma cease.
3. The SR calcium channels close when the action potentials cease. The Ca^{2+} pump takes up remaining Ca^{2+} ions into the SR.
4. As the sarcoplasmic Ca^{2+} concentration drops, Ca^{2+} dissociates from the troponin. Tropomyosin resumes its previous position over the myosin binding sites.



(a) Muscle contraction **(b) Muscle relaxation**

Figure 7.8. Muscle contraction and relaxation. A. Muscles contract by sarcomere shortening when calcium is present in the sarcoplasm. **B.** Muscles relax when calcium is pumped out of the sarcoplasm. *Which organelle stores calcium in muscle cells?*

5. Myosin can no longer bind actin—the thick filaments "lose their grip" on the thin filaments. Remember that muscle tissue is elastic, so the sarcomere rapidly returns to its resting length.

7.9 What is a motor unit?

7.10 How does the electrical signal in the neuron create an electrical signal in the muscle fiber?

7.11 Name three proteins found in thin filaments.

7.12 Does calcium bind thick filaments or thin filaments?

Muscle Energy

A steady supply of ATP is required to maintain every one of our cells, but muscle cells have particularly high energy needs. ATP fuels three important aspects of muscle activity:

- *Sarcolemma membrane potential:* Recall from ← Chapter 4 that Na^+/K^+-ATPase is responsible for maintaining the Na^+ and K^+ gradients across the cell membrane, which are required for action potentials.
- *Cross-bridge cycling:* The myosin heads use the energy from ATP cleavage for the myosin head power stroke, and the cross-bridge breaks when a fresh ATP molecule binds.
- *Muscle relaxation:* The calcium pump uses ATP to actively transport calcium into the sarcoplasmic reticulum.

Recall that ATP stores energy in a chemical bond. The energy in this bond is released when a phosphate is removed from ATP, generating ADP, as shown in this reaction:

$$ATP + H_2O \rightarrow ADP + H_2O + PO4 + \text{energy}$$

Study of this reaction reveals that energy is required to force it in the opposite direction; that is, to convert energy-depleted ADP back into ATP. As shown next, we get most of this energy from the chemical bonds in nutrients.

Different Processes Can Generate ATP

Muscle cells are constantly generating ATP by a variety of processes. In general, processes that produce larger amounts of ATP involve more chemical reactions and thus require more time to complete. A contracting muscle fiber may use all of the processes to varying degrees, depending on the muscle type, the intensity of contraction, and the duration of the muscular activity.

ATP Stores and Creatine Phosphate Provide Immediate Energy

Muscles store a small amount of ATP (generated earlier by nutrient metabolism) to fuel the first few seconds of activity. However, since muscle fibers rupture if muscle ATP stores fall too low, various protective mechanisms usually prevent excessive depletion of ATP stocks. One of these mechanisms involves **creatine phosphate,** a molecule unique to muscle (Fig. 7.9A). It works by converting some of the energy-depleted ADP molecules back into ATP molecules by transferring its phosphate to ADP, a reaction that generates creatine plus ATP. Muscle cells contain only enough creatine phosphate to fuel about 10 seconds of activity. However, when the muscle fibers are at rest, they can regenerate their creatine phosphate stores by using ATP obtained from nutrients. A high-energy phosphate molecule is transferred to a creatine molecule, producing ADP plus a new molecule of creatine phosphate.

Glycolysis Produces Pyruvate and ATP

Glycolysis (*glyco-* = "sugar"; *-lysis* = "to break"), the breakdown of glucose into *pyruvate*, is the fastest method of generating ATP from nutrients (Fig. 7.9B). The initial source of glucose is *glycogen,* a glucose polymer stored within muscle fiber. Glycogen must be broken into individual glucose molecules (actually, glucose-6-phosphate), which are then used to generate ATP. This reaction is called **glycogenolysis,** and is catalyzed by an enzyme, *glycogen phosphorylase*. Blood glucose can also be used, but glycogen is more abundant and supplies glucose-6-phosphate at a faster rate.

Glycolysis occurs in the cytosol of muscle cells and is an *anaerobic* process; that is, it does not *require* oxygen, although it can also occur in the presence of oxygen. It generates three ATP molecules per glucose molecule derived from glycogen. When blood glucose is used, only two ATP molecules are generated per glucose molecule, because it costs one ATP molecule to convert blood glucose into glucose-6-phosphate.

Pyruvate, the end-product of glycolysis, can be a source of additional ATP. However, for reasons discussed in → Chapter 15, pyruvate is frequently converted first into **lactic acid.** About half of this lactic acid will be converted back into pyruvate within the same muscle cell, during the

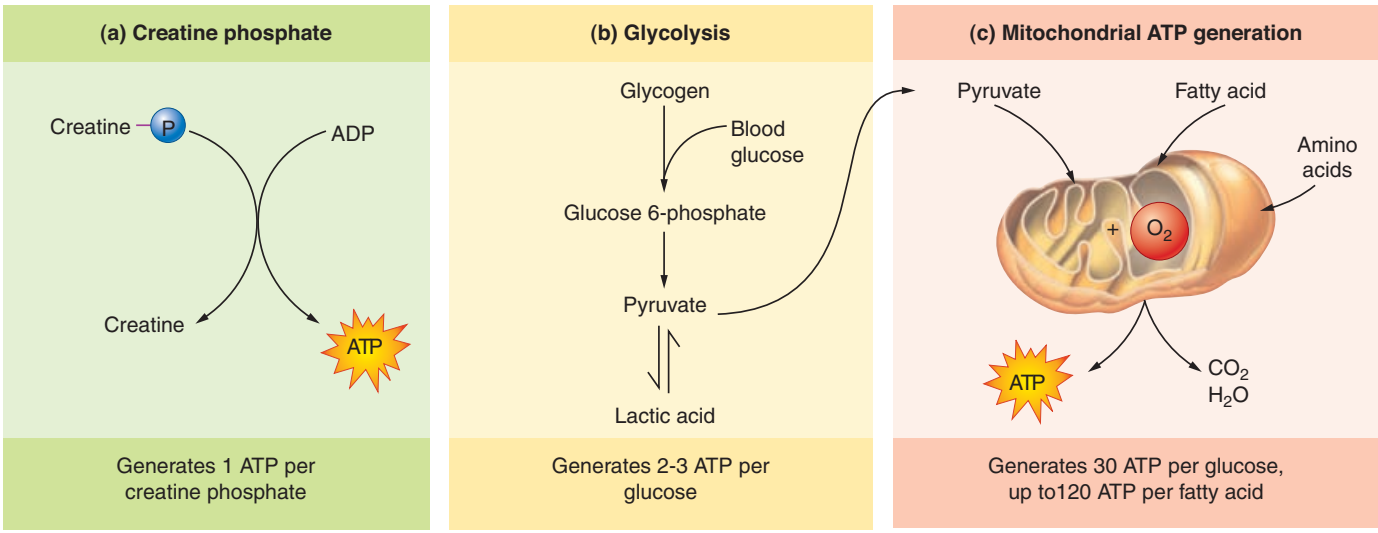


Figure 7.9. Muscle energy. A. Creatine phosphate transfers its phosphate group to ADP to generate ATP. When ATP is abundant, this reaction runs in reverse to regenerate creatine phosphate molecules at the expense of ATP. **B.** Glycolysis converts glucose produced by glycogen breakdown (or arriving in blood) into pyruvate. Pyruvate can be converted into lactic acid, and lactic acid can be converted back into pyruvate. **C.** Mitochondria generate large amounts of ATP from pyruvate, fatty acids, or amino acids. *Which substance can be used directly to generate ATP—lactic acid or pyruvate?*

infinitesimally brief rest between individual contractions (muscle fibers in a contracting muscle take turns producing force). Most of the remaining lactic acid will travel to nearby muscle cells where it, too, will be converted back into pyruvate. However, a very small amount of lactic acid travels to the liver and is converted into glucose.

Case Note

7.8. Is Hammid suffering from a shortage of ATP, creatine phosphate, or calcium?

Mitochondrial ATP Production Meets Long-Term Energy Needs

Mitochondria contain a host of enzymes that completely break down various nutrients and generate large amounts of ATP (Fig. 7.9C). The complex series of chemical reactions performed by these enzymes can be divided into two stages—the *citric acid cycle* and *mitochondrial respiration*—and is discussed in detail in ➡ Chapter 15.

Mitochondrial ATP generation is described as *aerobic* because, unlike glycolysis, it *requires* oxygen. Most of the required oxygen comes from oxygen bound to hemoglobin in blood, but some of it is obtained from oxygen bound to myoglobin in muscle. Although their oxygen need is absolute, mitochondria are not picky about their nutrient source—they effectively metabolize pyruvate (generated by glycolysis) and fatty acids. The fatty acids can come from blood or from lipid droplets within the muscle fiber.

The reaction is as follows:

$$\text{Pyruvate or fatty acids} + O_2 \rightarrow CO_2 + H_2O + \text{ATP}$$

Mitochondria provide a slow and steady supply of ATP—they generate 30 ATPs per glucose molecule (recall that glycolysis also generates 2 to 3 ATPs per glucose molecule), or a staggering 120 ATPs per fatty acid molecule.

Remember This! **Mitochondria do not directly break down glucose to generate ATP. Instead, they use pyruvate generated by glycolysis.**

Especially in individuals consuming more protein than their body requires, blood amino acids are taken up by muscle fibers and used by mitochondria to generate ATP. However, body protein is not usually broken down to generate amino acids for energy. Most body organs are built on a framework of protein; hence, proteins are used for fuel only as a last resort—using amino acids to generate ATP is akin to burning the house down to keep warm. This is why, for example, people who are starving lose muscle mass—they are burning muscle protein to stay alive.

Case Notes

7.9. Our patient Hammid cannot convert glycogen into glucose. Name the enzyme that accomplishes this reaction.

7.10. Which process is defective in Hammid's muscle cells—glycogenolysis or glycolysis?

Muscle Cells Contract Aerobically or Anaerobically

Jogging and other endurance activities are often described as "aerobic exercise" because oxygen-dependent mitochondria generate most of the required ATP, from glycolysis-derived pyruvate, fatty acids, and perhaps amino acids. Muscle cells function aerobically if three conditions are met:

1. The muscle cell contains abundant mitochondria.
2. The muscle cell is supplied with adequate oxygen.
3. The ATP needs of the muscle cell are low or moderate.

Conversely, athletic activities requiring short-lived, powerful contractions are often described as "anaerobic exercises," because they meet their ATP needs using processes that do not require oxygen (stored ATP, creatine phosphate, and glycolysis). Anaerobic metabolism depends on muscle glycogen stores, since blood glucose delivery is too slow to keep up with demand. Most of the lactic acid produced as a glycolytic end product travels to nearby muscle cells for further metabolism. However, since lactic acid is generated faster than the noncontracting cells can convert it back into pyruvate, lactic acid often accumulates in blood. Most investigators do not believe that this lactic acid has any deleterious effects on muscle function. Nevertheless, for reasons to be discussed, muscle cells cannot generate ATP by anaerobic metabolism for long without tiring.

Anaerobic metabolism occurs in three circumstances. The first is a matter of imposed demand; that is, hard work. Anaerobic metabolism provides an extra energy kick when oxygen delivery to the muscle cell cannot keep up with the needs of mitochondrial respiration. The second is a matter of anatomy: anaerobic metabolism preferentially occurs in some muscle cells, called *glycolytic muscle fibers* (discussed later in the text). The third is a matter of timing: we use anaerobic metabolism when we begin to exercise, because the mitochondria take a few minutes to make enough ATP. It is important to note that the latter two circumstances do not reflect inadequate oxygen supply.

Remember This! **The terms *anaerobic metabolism* and *glycolysis* are often used synonymously, but erroneously, since glycolysis is the necessary first step in both aerobic metabolism and anaerobic metabolism.**

Skeletal Muscle Fibers Are Oxidative or Glycolytic

Muscle fibers can be classified according to their primary method of ATP generation. **Slow-twitch (oxidative, type I) fibers** are optimized for aerobic metabolism (Fig. 7.10).

They contain many mitochondria and an abundant supply of myoglobin, which stores oxygen. Slow-twitch fibers are packed with blood vessels that keep them supplied with glucose, oxygen, and fatty acids. Slow-twitch fibers are generally thin, slow to contract, and slow to fatigue. They are thus well suited to muscles that are continuously at work, such as the muscles that maintain posture. They also come into play during endurance exercise. Myoglobin is reddish, and slow-twitch fibers, reflecting their high myoglobin content, are dark reddish-brown.

On the other hand, **fast-twitch (glycolytic, type II) fibers** are optimized for anaerobic metabolism (Table 7.2). They need large supplies of creatine phosphate, glycolytic enzymes, and glycogen because the muscle fiber will generate only three ATPs per glucose molecule. They have less myoglobin, fewer mitochondria, and

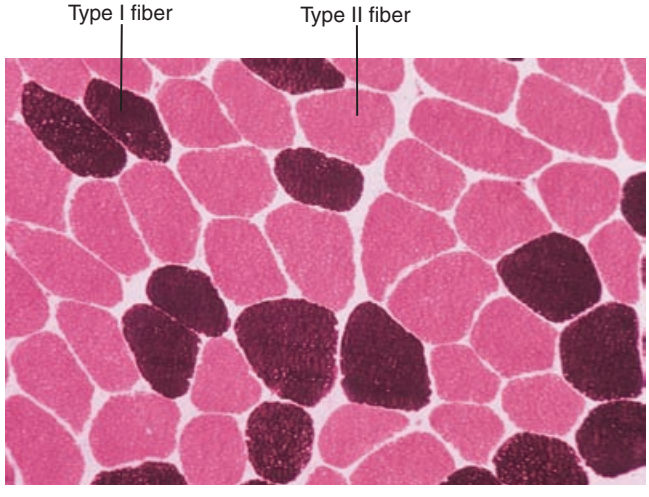


Figure 7.10. Muscle fiber types. The muscle fibers in this micrograph have been stained for the slow type of myosin found in slow-twitch (oxidative) muscle fibers. *Which fibers would contain fewer mitochondria—the darker cells or the lighter cells?*

Table 7.2 Muscle Fiber Types

Characteristic	Fast-Twitch Glycolytic	Slow-Twitch Oxidative
Appearance	White	Red
Primary ATP source	Anaerobic metabolism	Aerobic metabolism
Mitochondria/capillaries	Few	Many
Glycogen reserves	High	Low
Myoglobin content	Low	High
Rate of fatigue	Rapid	Slow
Fiber size	Large	Small
Contraction speed	Fast	Slow

fewer blood vessels than slow-twitch fibers. Thus, fast-twitch fibers are pale or whitish. Although they tire quickly, they are large and strong; thus they are well suited to explosive, large movements (such as lifting a heavy box or sprinting). Want to know more? Refer to the *Type IIa muscle fibers: The Best of Both Worlds* box on **http://thepoint.lww.com/McConnellandHull.com** for information about "superfibers" that combine the advantages of slow- and fast-twitch types.

To memorize these distinctions, it may help you to recall that chicken, turkey, and quail breast is "white meat" because it is composed mainly of fast-twitch fibers to power intense wing motion for short flights. By contrast, ducks and doves are migratory birds and their breast meat is reddish "dark meat" because it is composed of slow-twitch fibers to power sustained flights over hundreds of miles.

Most human skeletal muscles are a mixture of slow- and fast-twitch fibers; however, the fibers of any given motor unit are all of the same type. The percentage of fast and slow fibers in each muscle is genetically determined: some people have more fast-twitch fibers in certain muscles; others have more slow-twitch fibers in the same muscles. What's more, proportions vary according to muscle location and function. For example, the muscles of the upper limb and shoulder are predominantly fast-twitch fibers because they are used intermittently and briefly to produce large amounts of force for activities such as manipulating tools, lifting, or throwing. The muscles that power eye movements are composed entirely of fast-twitch fibers. By contrast, the muscles of the spine and neck are predominantly slow-twitch fibers, because these muscles are in constant use maintaining posture.

Case Note

7.11. Recall that Hammid cannot walk on his toes without cramping. The muscle required for toe-walking, the *gastrocnemius,* has few mitochondria and large muscle fibers. Is it primarily composed of fast-twitch or slow-twitch fibers?

Case Discussion

Muscle Energy Metabolism: The Case of Hammid S.

Hammid suffers from a genetic defect in which he lacks an enzyme—glycogen phosphorylase—that is essential for the breakdown of glycogen. This reaction, called glycogenolysis, is necessary to provide the large amounts of glucose required for strenuous muscle activity.

Understanding how muscle gets its energy supplies is the key to understanding Hammid's signs and symptoms (Fig. 7.11). Recall that muscle obtains energy in three different ways:

1. ATP stores and creatine phosphate fuel the first few seconds of any contraction.
2. Glycogenolysis (glycogen breakdown), followed by glycolysis (pyruvate generation from glucose) can also generate energy relatively quickly at the beginning of the contraction. This process also provides an extra "kick" of energy when large amounts of ATP are needed in a short time period.
3. Aerobic metabolism, which requires oxygen to metabolize pyruvate (generated by glycolysis) or fatty acids, provides a steady supply of ATP over the long term. This process can use muscle stores of glycogen and fat or blood supplies of glucose and fatty acids.

Note that Hammid has no difficulty *initiating* muscle contractions, because his muscles have a normal, small store of ATP and creatine phosphate. This enables him to get under way. Nor is his long-term daily activity impaired—he is okay as long as demand is low. Using mitochondrial respiration, he can burn fatty acids from fat, or amino acids from protein, and he can even burn glucose obtained from his blood. But when demand is

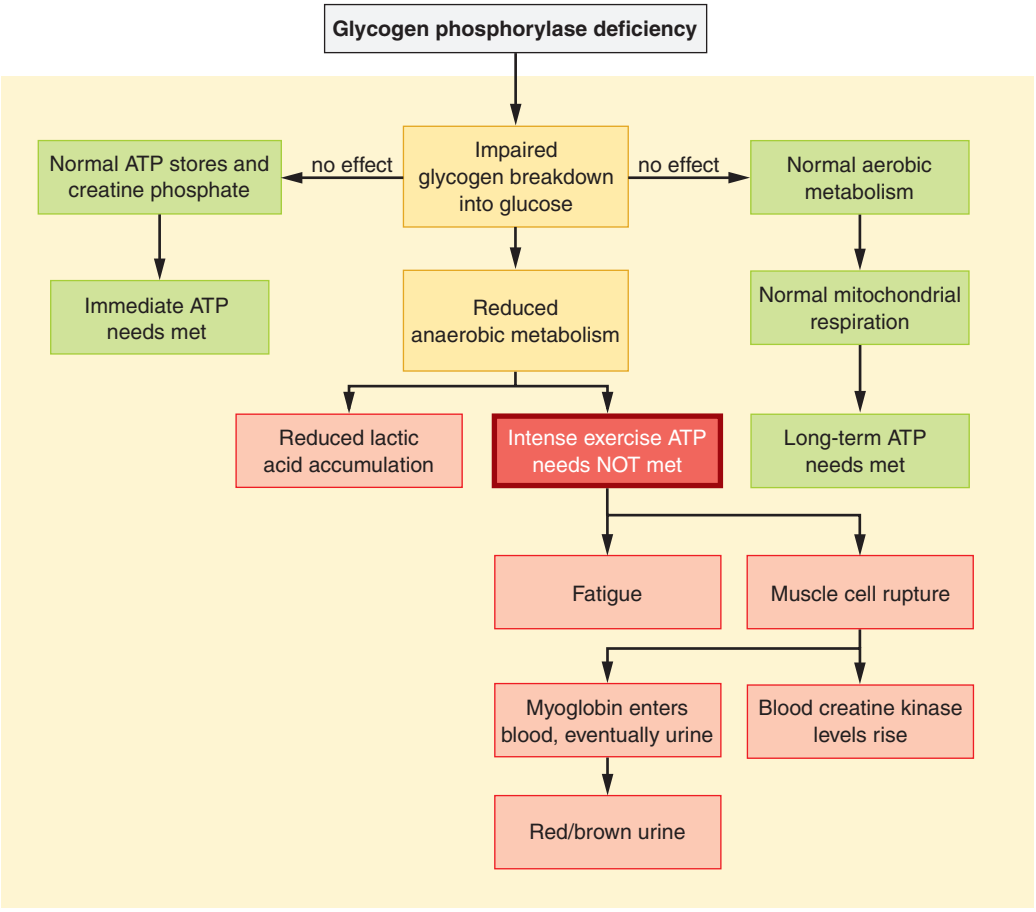


Figure 7.11. Muscle energy metabolism and Hammid S. *How do we know that some of Hammid's muscle cells have ruptured?*

high, his body cannot supply glucose by breaking down his abundant stores of glycogen because his defective gene cannot make the enzyme necessary to do the job. Hammid's problem therefore arises when he engages in sustained, vigorous activities that exhaust the available fuel. After a few minutes of strenuous effort, he consumes his entire supply of blood glucose; mitochondrial respiration is too slow to supply all of the demands for ATP, and his ability to obtain glucose from glycogen is defective.

Confirming the diagnosis is the important observation that Hammid's blood lactic acid did not rise as it normally should with strenuous activity. Why? Because during intense exercise, a normal person can use glycogenolysis to generate the large amount of glucose required for anaerobic metabolism. As glycolysis rapidly breaks down many glucose molecules into pyruvate, the pyruvate is converted into lactic acid. However, Hammid's metabolism is not normal—he can't break down glycogen to supply glucose. His glycolytic system must rely on blood glucose alone and quickly exhausts the supply before excess pyruvate can accumulate and be converted into lactate.

When Hammid's muscles call for large amounts of fuel, the call goes unanswered, and the ATP levels in the muscle cell fall dangerously low. As a result, muscle cells rupture, releasing their contents (which include myoglobin and the creatine kinase enzyme) into the blood and eventually into the urine. Muscle cramps occur, creatine kinase levels are elevated in Hammid's blood, and myoglobin stains his urine brown.

Hammid's parents were advised to steer him away from vigorous activities like sprinting and soccer and to encourage moderate exercise such as jogging or hiking, which would increase the ability of his muscles to perform mitochondrial respiration. They were also instructed to make sure that he consumes a candy bar or a sugar-containing drink such as orange juice about 30 minutes before exercise in order to elevate his blood glucose. Finally, they were advised to insist that Hammid stop exercising if cramping occurred.

Case Note

7.12. On the molecular level, what is Hammid's problem?

Skeletal Muscle Experiences Fatigue

When muscle is vigorously exercised for a long time, it loses the ability to respond to nerve stimulation, a condition known as **muscle fatigue.** Fiber contraction becomes weaker and weaker and finally stops altogether. We used to think that muscle fatigue reflected ATP depletion or lactic acid accumulation, but we now know that neither of these hypotheses can account for most cases of fatigue. So why does fatigue occur? The causes are many and varied, reflecting the nature of the exercise and the training state of the individual.

The major limit in submaximal endurance exercise is the ability to generate ATP. Untrained muscles fatigue because they have a blood delivery problem—they don't have enough capillaries perfusing their oxidative fibers. One of the benefits of endurance training is the growth of more blood vessels supplying oxidative fibers. In these trained individuals, glycogen stores then become the limiting factor.

Fatigue in maximal anaerobic exercise is thought to reflect phosphate accumulation. Recall that the energy is liberated from ATP by cleaving off one phosphate. Maximal exercise uses a lot of ATP in a short time, resulting in the accumulation of many phosphates. Phosphate interferes with contraction directly, by blocking cross-bridge formation, as well as indirectly, by reacting with calcium in the SR and reducing its release into the sarcoplasm.

However, we rarely see true muscle fatigue of the types described above, which are also known as *peripheral fatigue*. As accomplished athletes say, "The mind wears out before the muscle." Essentially, untrained athletes find the sensations created by exercise unpleasant; they, therefore, lessen their effort in order to gain relief. Also, many conditions (such as increased body temperature) lead the brain to send fewer signals to muscles. Thus, the most common cause of fatigue originates in the central nervous system and is thus called *central fatigue*.

Case Notes

7.13. Why do you think Hammid's muscles fatigue so easily?

7.14. Many athletes "carbo-load" in order to build up their glycogen stores and increase their resistance to fatigue. Would carbo-loading help Hammid?

7.13 Name the two sources of ATP that fuel the first seconds of a contraction.

7.14 Some of the ATP used by muscle cells is used to actively transport a specific ion into the SR. Name this ion.

7.15 True or false: A single ATP molecule is generated when creatine is converted into creatine phosphate.

7.16 True or false: The muscle cell uses up all of its stored ATP before it begins to generate more.

7.17 Which nutrient can generate ATP without entering the mitochondria—glucose or fatty acids?

7.18 What is the end product of glycolysis?

7.19 True or false: Lactic acid is a source of ATP—it can be used by muscle fibers to generate pyruvate, which can be used to generate ATP.

7.20 Which nutrient generates more ATP per molecule—glucose or fatty acid?

7.21 Which ATP-generating processes are considered to be anaerobic—that is, which do not require oxygen?

7.22 Which of the following is *not* a requirement for aerobic metabolism: abundant oxygen, large glycogen stores, or abundant mitochondria?

7.23 Which fibers receive a greater blood supply—type I or type II?

7.24 Name three causes of muscle fatigue in endurance exercise.

The Mechanics of Muscle Contraction

The force of a muscle contraction is exquisitely controlled; we can use the same muscles to hold a delicate glass ornament and to wring water from a face towel. The force an individual muscle exerts depends on:

- The force exerted by each contracting fiber
- The number of motor units contracting

Individual Fibers Provide Force

Recall that muscle contraction is accomplished by cross-bridges formed between the myosin heads of the thick filaments and the actin binding sites of thin filaments.

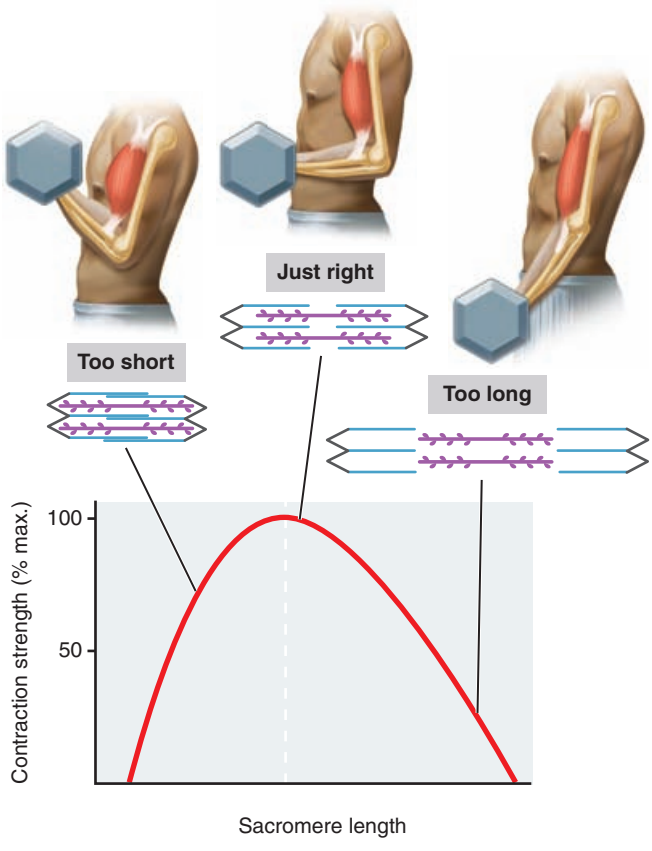
Contraction strength depends, therefore, upon how many cross-bridges form. Cross-bridge number, in turn, depends on how many myosin heads can reach the thin filaments and how many binding sites on the thin filaments are available.

Contractile Power Depends on Muscle Fiber Length

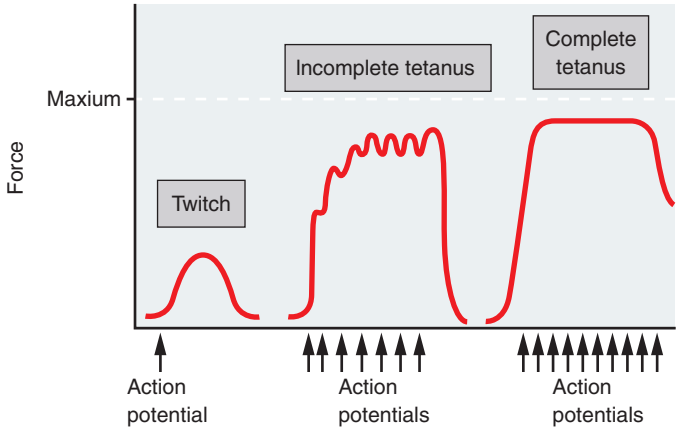
Sarcomere length, and thus muscle length, is one determinant of the force developed by an individual muscle fiber. At the optimal sarcomere length, all of the myosin heads are positioned to be in contact with actin molecules and form cross-bridges, and the contraction will generate the maximum amount of tension possible (Fig. 7.12A, middle). This property of muscle is called the *length–tension relationship*. At very short sarcomere lengths, the thin filaments are pulled so close that they meet in the middle and overlap, which covers their binding sites and interferes with the ability to form cross-bridges with thick filaments (Fig. 7.12A, left).

At very long sarcomere lengths, the exact opposite occurs—the thin filaments are so far apart that they lose most of their contact with the thick filaments (Fig. 7.12A, right). Thus, they contract poorly.

If we remember that sarcomeres are lined up end to end in a muscle, it is possible to extrapolate this length–tension relationship to the behavior of an entire muscle. Try performing a biceps curl. Holding a weight in your hand, start with the arm straight, elbow extended. In this position, the biceps muscle is relaxed and lengthened. Lift the weight, palm up, by flexing your elbow. As you do, the muscle shortens. Notice that the action is most difficult at the very beginning and very end of the curl because the sarcomeres are too long at the outset and too short at the end. Conversely, the middle portion of the curl is relatively easy, because the sarcomeres are at their optimal length and can generate the most force.



(a) The length-tension relationship of skeletal muscle



(b) Twitches and tetanus

Figure 7.12. Determinants of force. A. The force generated by individual fibers varies according to the muscle length, which determines the sarcomere length. At the optimum length, all myosin heads are able to form cross-bridges with actin molecules. **B.** The force generated by individual fibers depends upon the frequency of stimulation. Everyday productive muscle contractions usually involve incomplete tetanus. *Which type of contraction is invoked by a single action potential?*

Physiological Contractions Are Unfused Tetanus

A single action potential in a muscle fiber results in a weak, transient muscle contraction called a *twitch* (Fig. 7.12B, far left). A slightly stronger state of contraction results if a second action potential occurs before the twitch is finished; that is, the force of the two twitches is *summed* together. Subsequent action potentials result in progressively greater force, until a third state called *incomplete tetanus* is reached, in which the muscle fiber only relaxes slightly between subsequent contractions (Fig. 7.12B, right). Only in maximal contractions, such as lifting the heaviest weight possible for a single repetition, do we see the fourth state of contraction, *complete tetanus,* in which action potentials arrive so frequently that the fiber does not relax at all between contractions (Fig. 7.12B, right).

These responses to different action potential frequencies underline the importance of *calcium* in force generation—recall that calcium enables cross-bridge formation, and calcium reuptake into the sarcoplasmic reticulum results in relaxation. A single action potential does not release enough calcium to bind all of the troponin molecules, so not enough cross-bridges can form to generate maximum force. However, with repeated stimulation, the rate of Ca^{2+} release is greater than the rate of Ca^{2+} reuptake, so Ca^{2+} levels rise progressively higher with each successive action potential. The rate of calcium release is so high in complete tetanus that all binding sites are continually occupied, generating continuous, maximal force.

In everyday contractions, each skeletal muscle fiber receives action potentials at a high enough frequency to induce incomplete tetanus. In other words, contraction in an individual muscle fiber is all or none—*individual muscle fibers contract maximally or not at all.* We do not perceive the partial relaxations between subsequent contractions, because muscle fibers in different motor units alternate contracting and relaxing.

Remember This! **In an everyday contraction at a given fiber length, contraction of individual muscle fibers is all or none, as the fiber contracts in incomplete tetanus.**

Contractile Power Depends on Number of Motor Units Involved

Recall that a motor unit is a group of muscle fibers innervated by a single motor neuron (Fig. 7.3). Motor units vary in size and in the force they can generate: slow-twitch muscle fibers are usually grouped into small motor units, whereas motor units containing fast-twitch muscle fibers are usually larger. Motor units, like individual muscle fibers, contract maximally or not at all. Thus, the amount of contractile power generated by an entire muscle depends on the number and type of motor units involved. The process of adding additional motor units to produce a graded increase of force is called *recruitment*.

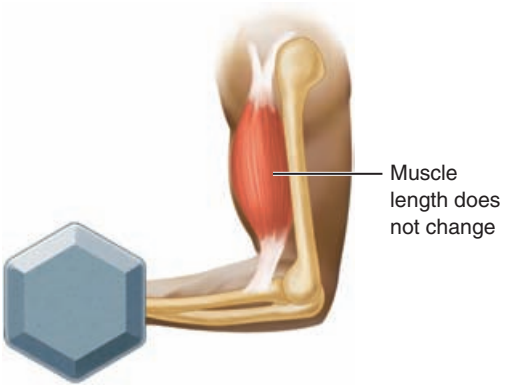
As a skeletal muscle contracts, first only a few motor units are stimulated, and they are recruited in specific order. Slow-twitch fibers are recruited first; fast-twitch fibers are recruited if more force is necessary. Even at peak muscle force, not all motor units are active at the same time: they rotate in and out of service, some relaxing after using up their resources while others fill the need for contractile force until they, too, need a break.

Muscle fibers of various motor units are intermingled, so that two fibers of the same motor unit are not adjacent to each other—some will be on one side of the muscle or deep within, others on the other side or superficial. This means that even a weak contraction (which recruits only a few motor units) will recruit muscle fibers scattered throughout the muscle to ensure symmetrical contraction. Otherwise, a weak contraction would activate only one region of the muscle and the contraction would pull unevenly on the bone.

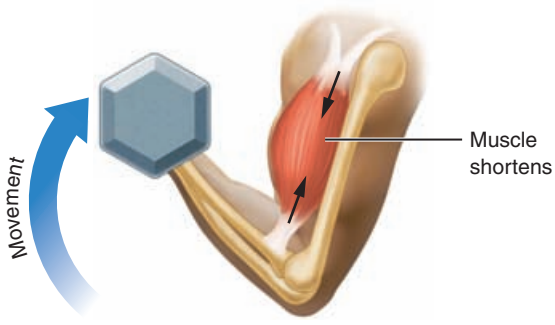
Muscle Fiber Contraction May or May Not Produce Movement

So far our assumption has been that contraction of a muscle fiber causes it to shorten. These dynamic or **isotonic contractions**—literally, "same tone" or "same force" contractions—are the stuff of everyday movement. Constant force is maintained over the course of a contraction, but the length changes. For example, lifting a weight in the gym or chewing your food are motions powered by isotonic contractions. Isotonic contractions can be classified into two subtypes:

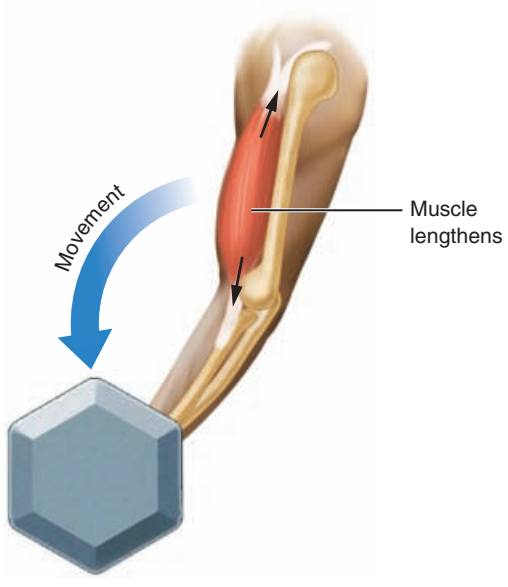
- *Concentric contractions* shorten the muscle, bringing the muscle attachment closer to the origin, as in raising a weight in a biceps curl (Fig. 7.13B). In concentric contractions, myofilaments slide; sarcomeres, fibers, and muscles shorten; and movement occurs.
- *Eccentric contractions*, conversely, generate a restraining force as the muscle lengthens (Fig. 7.13C), enabling the weight to be smoothly and controllably lowered following a biceps curl. In eccentric contractions, the myosin heads grab onto the actin filaments and slow the rate of movement, somewhat like applying a brake. Contrary to intuition, eccentric contractions



(a) Isometric contraction



(b) Concentric isotonic contraction



(c) Eccentric isotonic contraction

Figure 7.13. Isometric and concentric contractions. A. Isometric contractions, such as those keeping a heavy weight stationary, generate force but do not change muscle length. **B.** In a concentric contraction, the muscle shortens as it generates force to (in this example) raise a weight. **C.** In an eccentric contraction, the muscle lengthens as it generates force to (in this example) lower a weight. *During which type(s) of contraction does cross-bridge cycling occur?*

are actually more powerful than concentric contractions; that is, you use greater strength in lowering a heavy object than in lifting it.

However, the thing common to all muscle contraction is *force*, not movement. For example, if you try to lift a weight far beyond your strength, your muscles will contract but the weight won't budge: the fibers are generating force but not shortening because you are attempting to move an object that is—at least for you—immovable. Contractions that do not alter muscle length are called **isometric contractions**—literally, "same length" contractions (Fig. 7.13A). Force is generated and the muscle tenses; however, the myofibrils do not slide and the muscle does not change length. We perform isometric contractions all the time in order to oppose the downward force of gravity. For instance, the weight lifter in Figure 7.13C is exerting just enough upward force to offset the force of gravity pulling the weight downward. Similarly, isometric contractions maintain our upright *body posture*. Think about it: you do not have to concentrate on contracting your neck muscles in order to keep your head erect during the day, nor do you have to think about keeping your spine erect while sitting or standing. Subconscious, imperceptible isometric contractions do the job so you can focus on other matters.

Muscle tone is a state of subconscious isometric contraction that occurs even in voluntarily relaxed muscle. It maintains muscle in a healthy state, much the way that normal physical stress maintains healthy bone. If the nerve supply to a muscle is interrupted, perhaps because of an accident, the muscle loses its tone and becomes flaccid (soft, flabby). If the nerve connection is not reestablished, muscle fibers begin to shrink (*atrophy*). Complete lack of muscle tone is called *flaccid paralysis* and occurs when somatic motor nerves are unable to deliver action potentials to the muscle. For example, flaccid paralysis occurs with administration of Botox, which blocks the release of ACh from the somatic motor neuron at the neuromuscular synapse. The loss of facial wrinkles is due to induced flaccid paralysis of facial muscles that bunch skin into wrinkles. Flaccid paralysis also occurs with the severing of a peripheral nerve, or with severe spinal cord injury. In each of these examples the brain is not involved. By contrast, *spastic paralysis* is due to damage to the brain, which impairs the control of muscles. With brain lesions, voluntary control is lost, leaving the spinal cord to send uncontrolled action potentials to muscle, which causes uncontrolled muscle contraction. For example, the awkward, stiff gait of some patients with brain damage from stroke, cerebral palsy, or head injury is a manifestation of spastic paralysis.

Case Note

7.15. Will Hammid have trouble with forceful isometric contractions?

Remember This! **The thing common to all muscle contraction is force, not movement.**

Exercise Has a Positive Effect on Muscles

The saying "use it or lose it" applies to muscles just as it does to the practice of a skill. A worked muscle is a healthy muscle, and muscle improves its health according to the type of work it performs. Exercise improves the power and endurance of skeletal muscle. But the greatest benefit of exercise lies elsewhere: every system in the body is improved by physical exercise (see Chapter 18). Among nonsmokers, regular exercise is arguably the most important single activity for improving general health. Smokers benefit from exercise too, but the gain is small compared with the positive effect of quitting smoking.

Muscle *power* is improved by strength training regimes (also called *resistance training*), such as weight lifting, that increase muscle size. These exercises require repeated short bursts of powerful muscle action that overload and stress the muscle. We used to think that adult muscles grew only by enlarging existing muscle fibers with new myofibrils. Although this process does occur, it now seems certain that significant muscle growth reflects the participation of muscle stem cells, the satellite cells. Recall that the stem cells of adult muscle are located at the periphery of the muscle fiber. Exercise stimulates these stem cells to proliferate, producing new myoblasts that fuse with existing muscle fibers to make them larger. Myoblasts may also fuse with each other to produce entirely new muscle fibers.

Muscle power is critical in athletic endeavors requiring a large amount of force output, including the 100-yard dash, the pole vault, the high jump, and weight lifting. Note that these activities are often called *anaerobic* because they rely on anaerobic metabolism. Anaerobic exercises also enhance the ability of the larger, stronger muscle cells to produce ATP, using creatine phosphate and glycolysis.

Muscle *endurance* (resistance to fatigue) is improved by *aerobic* exercise that relies on mitochondrial ATP generation. These exercises require sustained low-level muscle action to improve muscle blood supply and increase the number of mitochondria. Endurance exercise also activates satellite cells, but muscles do not grow significantly bigger. Athletic performances that rely on aerobic conditioning include long-distance running, cross-country skiing, cycling, and long-distance swimming events. As discussed in later chapters, aerobic exercise also exerts beneficial effects on many other body systems, particularly the cardiovascular and respiratory systems.

Case Note

7.16. Hammid wants to build up his muscles by lifting weights, but his physicians advise against it. Why?

7.25 What is the difference between incomplete and complete tetanus, and which occurs more frequently?

7.26 True or false: Muscle contraction is always the strongest when the muscle is as long as possible.

7.27 Which type of motor unit is recruited first—that containing slow-twitch (type I) or fast-twitch (type II) fibers?

7.28 To generate a stronger contraction in skeletal muscle independent of muscle length, do we vary the force produced by each muscle fiber, alter the force produced by each motor unit, or vary the number of motor units recruited?

7.29 Give an example of an isometric and an isotonic muscle contraction.

7.30 Name an aerobic and an anaerobic exercise.

Smooth Muscle

Despite its functional importance, it is difficult for smooth muscle to get the respect it deserves. In the gym or on the athletic field, cardiac and skeletal muscles get all of the attention, as sweaty athletes admire their muscles and count their heart rates. Meanwhile, smooth muscle labors along, slow and reliable, tirelessly and quietly doing various jobs, such as massaging food through the gut to provide energy for the show, regulating blood flow by adjusting the diameter of blood vessels, and tightening sphincter muscles to hold urine and feces for release at another time.

Sheets of smooth muscle occur in the walls of all but the smallest blood vessels and in the walls of hollow organs: the intestines, the bronchial airway, the urinary and reproductive tracts, and others.

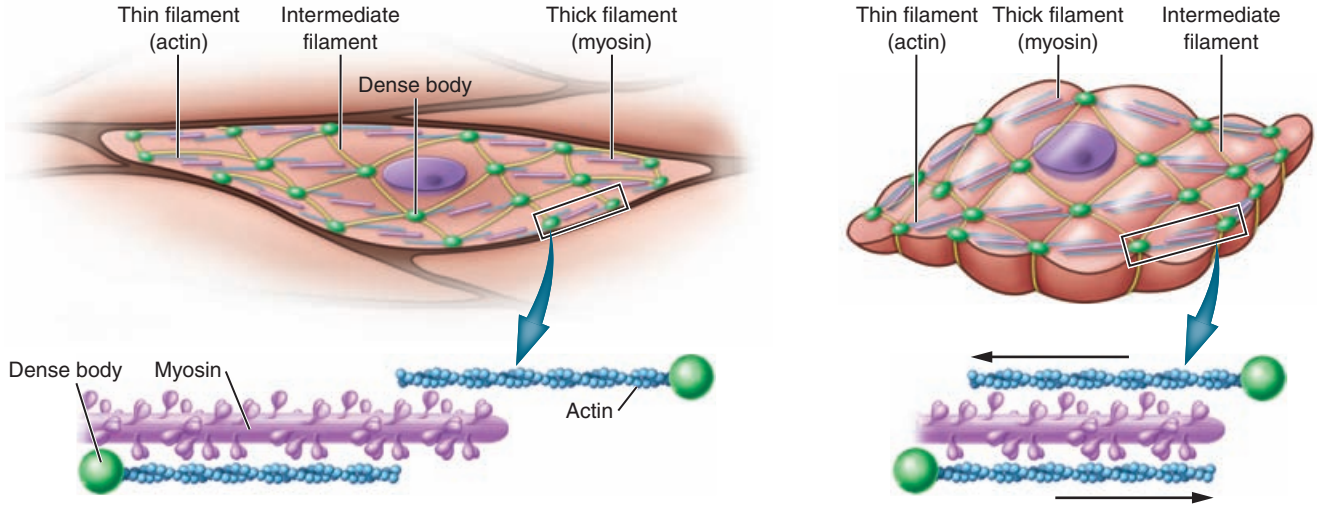
Smooth muscle takes about 25 times as long to contract as skeletal muscle and consumes only about 1% as much energy. Since smooth muscle contractions are relatively slow and do not generate the explosive force characteristic of skeletal muscle, aerobic metabolism, using nutrients from the blood, can easily meet smooth muscle's low energy needs—no need for anaerobic metabolism or stored glycogen here. Actin–myosin cross-bridges may latch semipermanently in a **latch state,** not unlike the rigor mortis that occurs after death, in which the cross-bridge cycle ceases while actin and myosin remain bound together. This latch state enables smooth muscle to maintain muscle tension without expending any energy at all, a state called *smooth muscle tone*. This low-level contraction is necessary for the proper function of blood vessels and other hollow structures that must maintain their size or shape against constant pressure.

The structure of smooth muscle cells and tissue is fundamentally different from that of skeletal and cardiac muscle (Fig. 7.14; Table 7.1). Not surprisingly, these structural differences account for the different contraction characteristics of smooth muscle: its slow, sustainable contraction; its tirelessness; its stretchiness; and its ability to propagate automatic waves of contraction.

Smooth Muscle Differs Structurally from Skeletal Muscle

Recall that in skeletal muscle the muscle cells are called fibers because they are very long and thin. In contrast, smooth muscle cells are short and plump. They have pointed ends and a bulge in the middle to accommodate a single nucleus, which lies squarely in the center of the cell, not to one side as in cardiac and skeletal muscle fibers. They are small for two reasons: their contractions are relatively weak, requiring fewer myofibrils, and they rely primarily on aerobic metabolism, which means they don't require large stores of glycogen.

Smooth muscle cells are formed upon a three-dimensional criss-cross structure of noncontractile **intermediate filaments** ← (Chapter 3), which are interconnected somewhat like a schoolyard jungle gym (intermediate filaments also strengthen skeletal muscle fibers but are organized differently). The filaments are interconnected by *dense bodies*, small dense protein discs scattered over the sarcolemma (muscle cell membrane). Dense bodies are the functional equivalent of the Z-disc in skeletal muscle; that is, they are anchor points for the filaments. Smooth muscle contraction, like that of skeletal muscle, is enabled by myofilaments—thick myosin filaments and thin actin filaments. These myofilaments are not arranged in perfectly ordered ranks, so that, unlike skeletal muscle, no dark-and-light pattern of striae (stripes) is created. Because of the



(a) Relaxed smooth muscle (b) Contracted smooth muscle

Figure 7.14. Smooth muscle. A. A relaxed smooth muscle cell. Myosin molecules are interspersed between the actin molecules. **B.** A contracted cell. Myosin heads pull on thin filaments, increasing the overlap between the two filament types and shortening the cell. *How are actin molecules anchored—by Z-lines or dense bodies?*

arrangement of myofilaments and their association with the dense bodies, smooth muscle cells bulge out as they shorten (Fig. 7.14B). Even though smooth muscle cells are much shorter than skeletal muscle cells, the myofilaments inside of smooth muscle cells are longer. In addition, the thick (myosin) filaments in smooth muscle have protruding heads along their entire length, so there is no headless zone like the "golf club shaft" in skeletal myosin. As a result, the length–tension relationship illustrated in Figure 7.12 does not apply to smooth muscle. Even when smooth muscle cells are stretched greatly, at least some of the myosin heads can still contact actin, so the filaments can continue to claw out contractile force regardless of cell length.

The arrangement of smooth muscle cells into tissues also contributes to the muscle's stretchiness. Most smooth muscle cells are layered upon one another to form sheets of cells similar to multiple layers of shingles on a roof. This arrangement enables smooth muscle to be stretched in many directions without tearing as the cells slide across one another to accommodate the stretch.

Remember This! **Intermediate filaments form the scaffolding of a smooth muscle cell, and myofilaments contract the cell.**

In Smooth Muscle, Calcium Acts on Myosin, Not Actin

To understand smooth muscle contraction and how it differs from skeletal muscle contraction, recall some of the details of the latter. Cross-bridge cycling requires that myosin heads in thick filaments bind to actin in thin filaments in order to claw out a contraction, but access to the thin-filament binding sites is controlled by troponin. A surge of Ca^{2+} ions stimulates troponin to expose the binding site. The myosin head then engages the actin binding site for the power stroke of contraction. In smooth muscle cells, the steps of the cross-bridge cycle detailed in Figure 7.7 are still relevant. However, smooth muscle differs in both the *source* and the *role* of the Ca^{2+} ions:

- *Source of Ca^{2+} ions*. Smooth muscle cells have very little SR. Instead, in smooth muscle, Ca^{2+} influx comes mainly through the cell membrane from extracellular fluid.
- *Role of Ca^{2+} ions*. Smooth muscle cells contain no troponin, so myosin binding sites on the thin filaments are always exposed. Instead of controlling the access to thin-filament binding sites, calcium in smooth muscle regulates the activity of the myosin heads on thick filaments. That is, only if calcium is present does the myosin cleave ATP and move through the cross-bridge cycle.

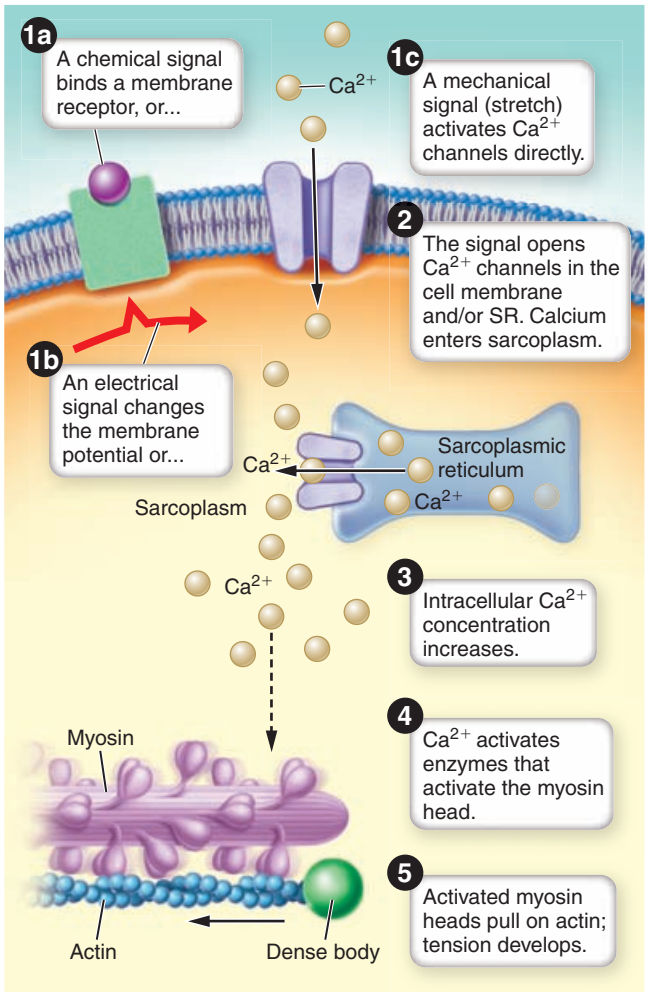


Figure 7.15. Smooth muscle regulation. Calcium enters the cytoplasm in response to a chemical, mechanical, or electrical signal and indirectly stimulates activity of the myosin head. The activated myosin molecules form cross-bridges with actin and contract the muscle. *True or false: Most of the calcium comes from the extracellular fluid.*

Because of these two important differences, the events of smooth muscle contraction differ from those in skeletal muscle (Fig. 7.15). Considerable variation exists in the mechanism of smooth muscle contraction, but a typical sequence is as follows:

1. An event—a chemical signal (e.g., neurotransmitter), an electrical signal (e.g., graded or action potential), or a mechanical signal (e.g., stretch)—activates calcium channels in the cell membrane and, in some cases, in the SR. Chemical signals must use a second-messenger system. These signals are discussed further below.
2. Calcium enters the cytoplasm from the extracellular fluid and possibly the limited amount of SR.
3. The intracellular Ca^{2+} concentration increases.
4. Through a number of intervening enzymatic steps, calcium activates myosin heads.

5. Activated myosin heads form cross-bridges with actin molecules, and the filaments slide upon one another, causing muscle contraction.

Remember This! **The myosin heads are regulated in smooth muscle; the binding sites on actin molecules are regulated in skeletal muscle.**

As with skeletal muscle, relaxation of smooth muscle begins when calcium is actively removed from the cytoplasm. In the case of smooth muscle, it is accomplished primarily by membrane transport proteins. However, recall that the myosin heads were enzymatically activated to initiate muscle contraction. They must therefore be enzymatically deactivated in order to stop cross-bridge cycling and induce smooth muscle relaxation. The enzyme *myosin phosphatase* does the job.

Case Note

7.17. Based on the information provided here, will Hammid have trouble with smooth muscle function? Why or why not?

Smooth Muscle Contraction Is Involuntary

Smooth muscle movement is involuntary; that is, it is not subject to conscious control, like skeletal muscle. Some smooth muscle is innervated by the autonomic nervous system, an important division of the nervous system that itself is not subject to voluntary control (see ➡ Chapter 8).

However, autonomic nerves do not innervate all smooth muscles. Some smooth muscles are stimulated to contract by hormones or by local chemical signals such as prostaglandins, hydrogen ions, and gases (carbon dioxide, oxygen, and nitric oxide). Consider, for example, the smooth muscle lining blood vessels ➡ (Chapter 11). Smooth muscle cells in the walls of blood vessels contract or relax in response to locally produced paracrine factors secreted by neighboring cells that signal their need for more or less blood flow. Contraction of these muscle cells constricts the blood vessel, reducing blood flow, whereas relaxation expands the vessel, increasing blood flow.

Smooth muscle is also stimulated by mechanical signals. This homeostatic mechanism prevents overstretching of blood vessels and other tissues and thereby prevents injury. Consider, for instance, a stomach overstretched by a very large meal. The stomach muscle begins to contract as the stomach is filled to capacity, preventing tearing of the stomach muscle (and, incidentally, inducing discomfort that prevents further food consumption).

Finally, the cells in some smooth muscles have unstable membrane potentials, which generate self-stimulating action potentials called *pacemaker activity*. In the gastrointestinal tract, for example, pacemaker activity generates waves of smooth muscle contraction (*peristalsis*) that massage food from one end of the tract to the other ➡ (Chapter 14). As we will see in ➡ Chapter 11, cardiac muscle is also self-stimulating.

Smooth Muscle Contracts as a Single Unit

Groups of smooth muscle cells contract in unison because the cells are connected to one another by gap junctions (⬅ Chapter 4), tiny liquid tunnels from one cell to the next, which allow rapid spread of the signal through all cells. When an electrical or chemical signal stimulates one cell, the change sweeps through the entire network of muscle cells and they contract as a single unit. Thus, contraction strength in smooth muscle cannot be varied by changing the number of contracting cells, as in skeletal muscle, which contains muscle fibers that are electrically insulated from one another. Instead, the amount of tension generated by individual smooth muscle cells varies according to the amount of calcium allowed into the cell from the extracellular fluid, which in turn activates greater or fewer numbers of myosin heads.

7.31 True or false: The calcium causing smooth muscle contraction usually comes from the extracellular fluid, but the calcium causing skeletal muscle contraction usually comes from the SR.

7.32 Would you find troponin in smooth muscle?

7.33 To generate a stronger contraction in smooth muscle, do we vary the force produced by each muscle fiber or vary the number of muscle cells contracting?

Skeletal Muscle Actions

Skeletal muscles move bones or stabilize them in certain positions, and (in the case of facial muscles) move skin and associated fascia. Most muscles cross a joint and act to move one bone in relation to the other. The end of the muscle that serves as an anchor for the movement is called the *origin;* the end that moves a body part is the *insertion*.

The contraction of a muscle pulls (never pushes!) the insertion toward the origin. Consider, for instance, the *masseter* muscle, with its origin on the zygomatic process

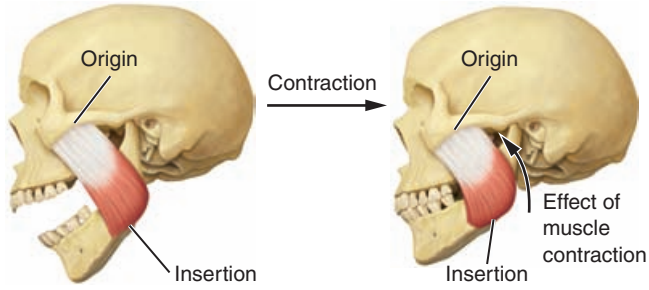


Figure 7.16. Origin and insertion. Most muscles span a joint and attach to two bones. The origin of the muscle attaches to the less movable bone; the insertion to the more movable bone. *In this illustration, does the muscle insert into the mandible or the temporal bone?*

of the temporal bone, and its insertion on the mandible (Fig. 7.16).

Contraction of this muscle closes the jaw, pulling the mandible (insertion) closer to the zygomatic process (origin). The words *origin* and *insertion* may not have literal meaning for the ends of certain muscles of the torso because the body part at both ends move. For example, some muscles attach to the spine at both ends and bend the spine, so it is arguable which end is the origin and which the insertion.

The action exerted by a particular muscle varies according to where it is attached and how the fibers are oriented. For example, a circular muscle surrounds the mouth. When it contracts, it purses the mouth, as in a kiss.

In producing movement, the actions of different muscles often complement or oppose each other. The role of a muscle in a particular movement can be described as follows:

- *Prime mover* (or *agonist*): the main muscle responsible for a given movement. The large quadriceps muscle on the anterior thigh is the prime mover that extends (straightens) the leg at the knee.
- *Antagonist:* a muscle that opposes the action of the prime mover. Antagonists must relax and lengthen to permit the movement, and they often exert the opposite action when they contract. The hamstrings muscles on the posterior leg must relax and lengthen when the quadriceps femoris straightens the leg.
- *Synergist:* a muscle that assists the action of the prime mover. Some synergists, called *fixators*, prevent the movement of a nearby joint. Remember that muscles shorten when they contract, bringing the insertion closer to the origin. Without fixators, the origin would also move toward the insertion. For instance, when we inhale deeply, several neck muscles stop the neck from flexing so that other muscles can elevate the rib cage.

To put these interactions together, let's consider how we raise the arm laterally at the shoulder (abduction). The deltoid muscle is the prime mover; the supraspinatus (a rotator cuff muscle deep to the deltoid) is a synergist important for the initiation of the movement. Gravity is the major antagonist, but muscular antagonists to the deltoid include the pectoralis major and the latissimus dorsi muscles (both muscles adduct the arm). Many muscles act as fixators by stabilizing the scapula, including the trapezius and pectoralis minor. All of these muscles can be visualized in Plate 7.5 at the end of the chapter.

Case Note

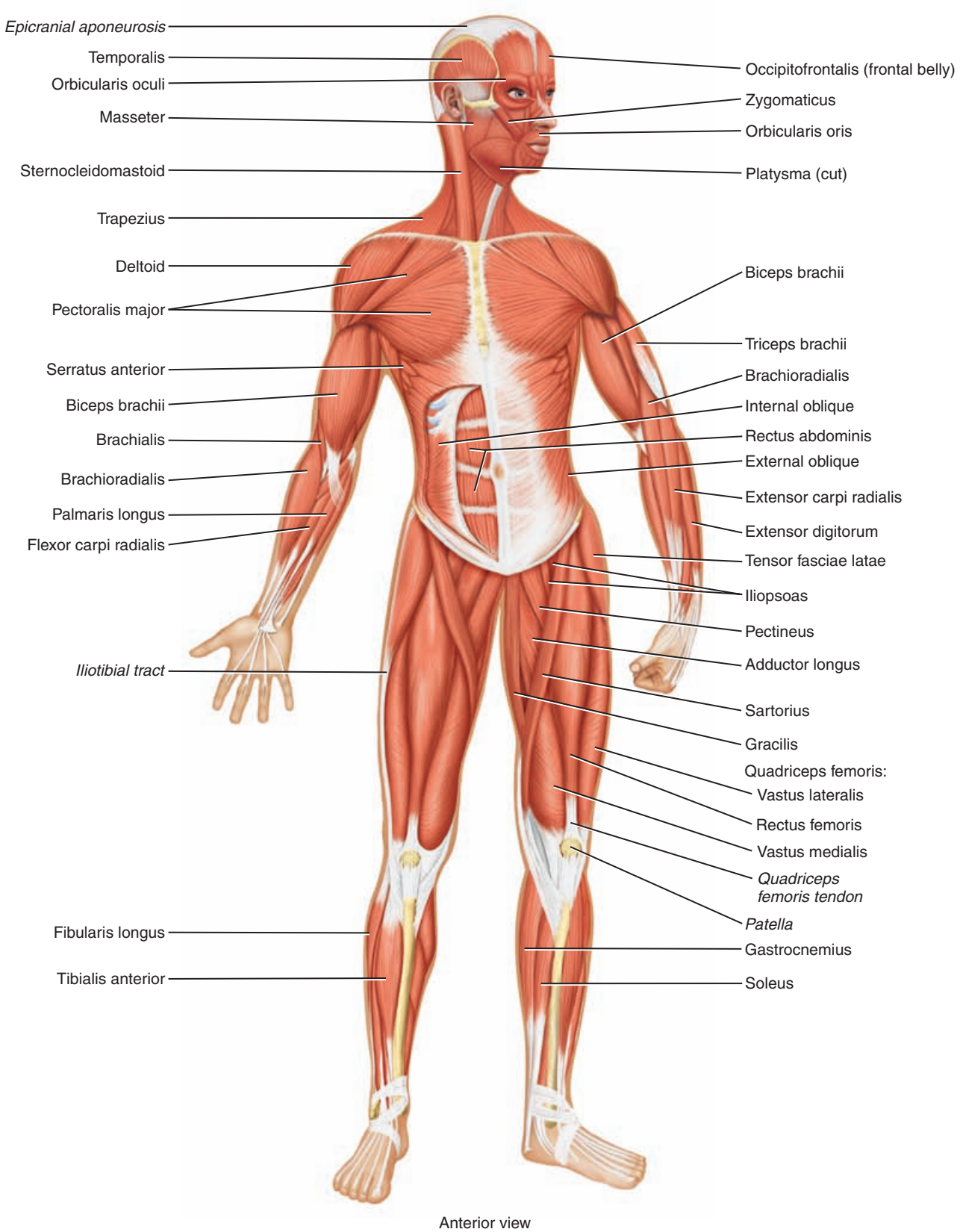
7.18. When Hammid walks on his heels, the gastrocnemius muscle contracts and the peroneus longus muscle relaxes. Which muscle is the prime mover and which is the antagonist?

7.34 When a muscle contracts, which part moves more—the origin or the insertion?

7.35 What is the name of a muscle that assists the action of a prime mover?

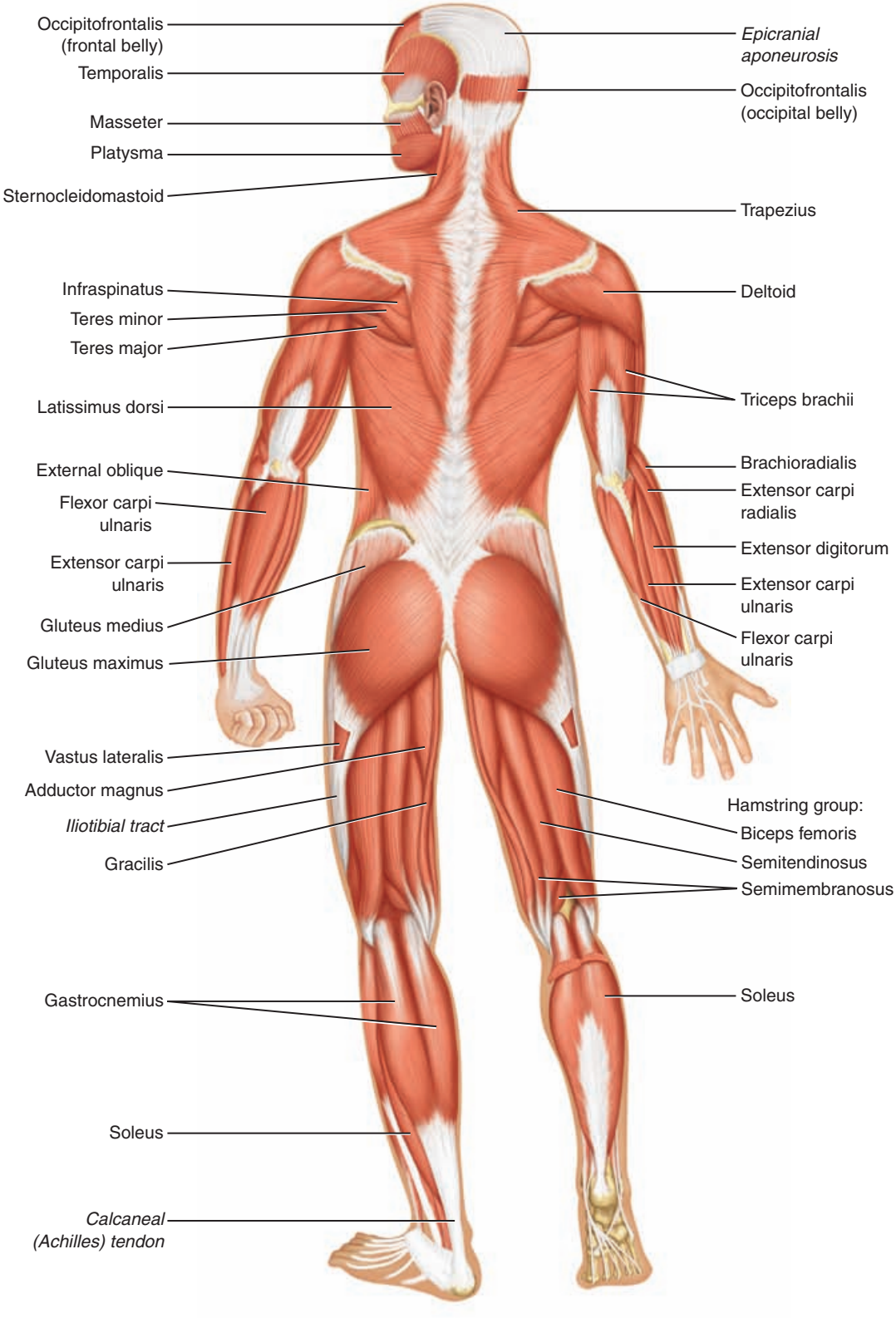
The Major Skeletal Muscles

The human body contains hundreds of muscles, ranging in size from the large, powerful thigh muscles to the tiny muscles that move our eyes. We cover a subset of these muscles, which we've chosen because they are important in body posture or movement or because they are important landmarks. As discussed in the History of Science box, titled *Medical Art and the History of Human Dissection,* artists have represented the human body for various purposes for millennia, but only began rendering its internal structures for scientific study a few hundred years ago. Use our illustrations and the accompanying tables to learn the location and shape of the major skeletal muscles. You can make the task of learning muscle anatomy easier by (a) learning the word parts used to name muscles and (b) performing the actions of each muscle as you read about it. Figures 7.17 and 7.18 provide an overview of the major superficial muscles. Plates 7.1 to 7. 9 provide more detailed views of the muscles in each region and summarize their important actions.



Anterior view

Figure 7.17. Superficial muscles, anterior view. *Two structures are labeled that are not muscles. Name them.*



Posterior view

Figure 7.18. Superficial muscles, posterior view. *Based on its name and your knowledge of movements at synovial joints, find a muscle that brings the lower limb closer to the midline.*

THE HISTORY OF SCIENCE

Medical Art and the History of Human Dissection

This book is filled with wonderful medical art, without which we would have an impoverished understanding of human form and function. These illustrations of muscles and other organs depict a reality documented by repeated dissections over many centuries. Can you imagine going through your daily life without knowing what your muscles look like? Or your heart? Or your brain? Until about 500 years ago, very few people knew such things.

The oldest depictions of the human form were not much more than stick figures rendered many thousands of years ago on the walls of caves (part A). They served an artistic purpose, perhaps for religious rites, and had no scientific intent. No early civilization attempted to depict the body's internal structure because every culture held that the sanctity of the human body forbade human dissection. However, there was deep interest in the human form as an object of art. In the last few centuries before the Common Era (BCE) the Greeks sculpted unparalleled masterpieces of the human form—strong, youthful figures predominated, their muscles clearly depicted beneath the surface, but the interest was artistic, not scientific (part B).

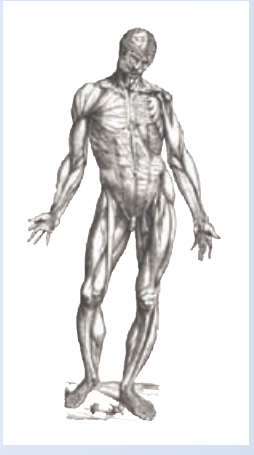
Then in the fourth century BCE, Herophilus of Chalcedon (350–280 BCE), a Greek, dissected human corpses. Herophilus described the brain, spinal cord, and nerves, speculating that they were of central importance to human function. The Egyptians soon followed when Alexander the Great (356–323 BCE) authorized dissections in Alexandria. But the descriptions produced by these ancient anatomists were largely narrative, not pictorial, and they were not informative by modern standards. The illustrations that were included were flat; they lacked perspective and mainly served to decorate the manuscript.

Although bodies continued to be dissected for the next thousand years, the knowledge that dissection could have provided was largely ignored as irrelevant by the physicians of the day. That's because they were steeped in the theories of Hippocrates (460–370 BCE), who defined good health as a proper balance among four supposed humors: phlegm (mucus), blood, black bile, and yellow bile. An excess of one or more of these humors, Hippocrates believed, caused illness. Thus, an understanding of anatomy was of no great use in this medical system.

With the coming of the Renaissance in western Europe in the 14th century, the modern scientific method was born and the facts revealed by human dissection began to be understood correctly for the first time. In the 16th century, Andreas Vesalius (1514–1564), a Dutchman, performed dissections, retained artists to depict the findings, and in 1543 published his momentous *De Humani Corporis Fabrica* (*On the Workings of the Human Body*), which for the first time depicted muscles, bones, and other body parts with remarkable clarity and artistic ingenuity (part C).

(a)

(b)



(c)

Muscle portrayals. A. Cave drawings. **B.** Greek sculpture. **C.** Vesalius's drawings.

Plate 7.1 Muscles of Facial Expression

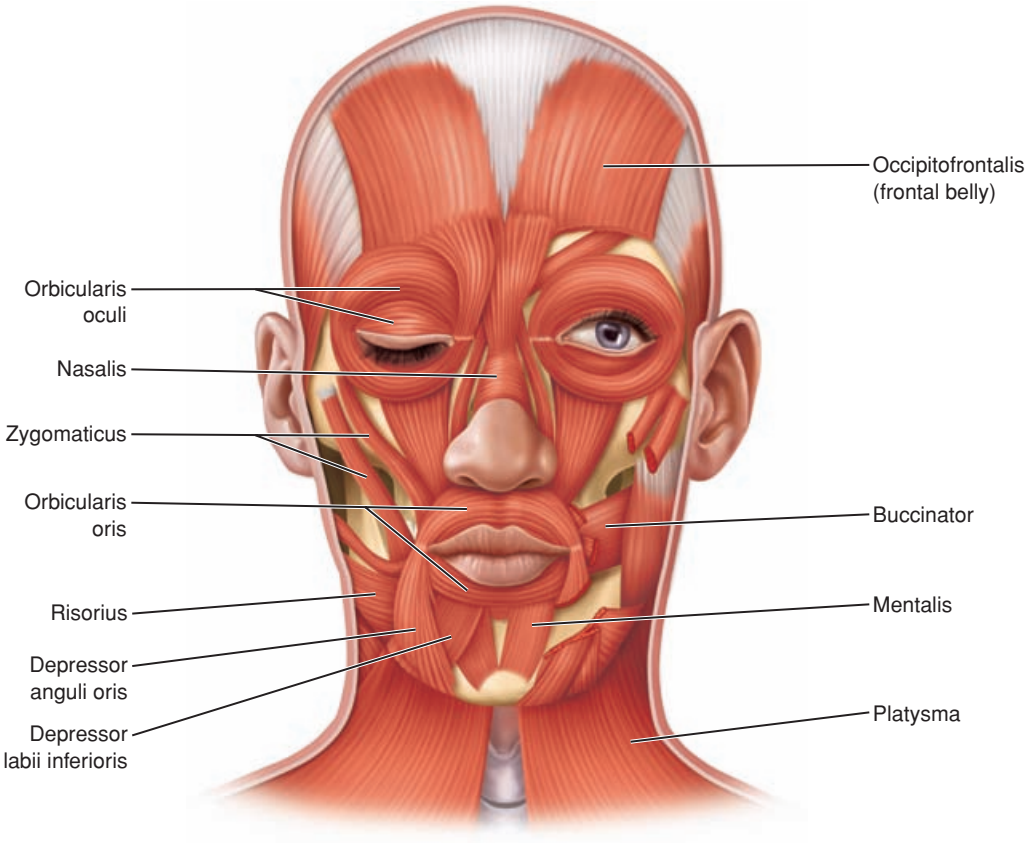


Plate 7.1 Muscles of Facial Expression

Name	Origin	Insertion	Action
Facial muscles attach to skin or other muscles rather than bones. Minute contractions of these muscles produce the subtle movements of skin and muscle we interpret as facial expressions.			
Occipitofrontalis, frontal belly *(occipit = base of skull; frontal = forward part)*	Epicranial aponeurosis (tendon)	Eyebrow, forehead skin	Raises eyebrows, wrinkles forehead
Occipitofrontalis, occipital belly *(occipit = base of skull; frontal = forward part)*	Occipital and temporal bones	Epicranial aponeurosis	Pulls scalp backward
Orbicularis oculi *(orb = circular; ocul = eye)*	Frontal bone, maxilla (eye orbit, medial wall)	Skin encircling eye	Closes eyelid
Nasalis *(nasal = nose)*	Maxilla	Bridge of nose (cartilage)	Brings sides of nose towards nasal septum
Zygomaticus *(zygoma = cheekbone)*	Zygomatic bone	Skin, muscle at lip corners	Raises corner of mouth, as in smiling
Orbicularis oris *(orb = circular; oris = mouth)*	Maxilla, deep surface of skin	Skin at mouth corners	Closes and protrudes lips (kissing, sucking), shapes lips (speech)
Depressor labii inferioris *(depressor = downward; labi = lip; infer = below)*	Mandible	Orbicularis oris	Depresses lower lip (when showing impatience)
Mentalis *(mentum = chin)*	Mandible	Chin skin	Elevates, protrudes lower lip (pouting)
Depressor anguli oris *(depressor = downward; anguli = corner; oris = mouth)*	Mandible	Mouth (angle)	Brings down mouth corners (frowning)
Buccinator *(bucia = cheek)*	Maxilla, mandible (alveolar processes)	Orbicularis oris	Flattens cheek (smiling, pushes food against molars, whistling, wind instruments)
Risorius *(risor = laugher)*	Platysma, masseter	Mouth angle	Draws mouth corner laterally (grinning)
Platysma *(platys = flat)*	Fascia covering deltoid, pectoralis major	Mandible	Tenses skin when teeth are clenched (resulting in skin ridges), depresses mandible, helps depressor anguli oris

Plate 7.2 Muscles Controlling the Jaw and Moving the Head

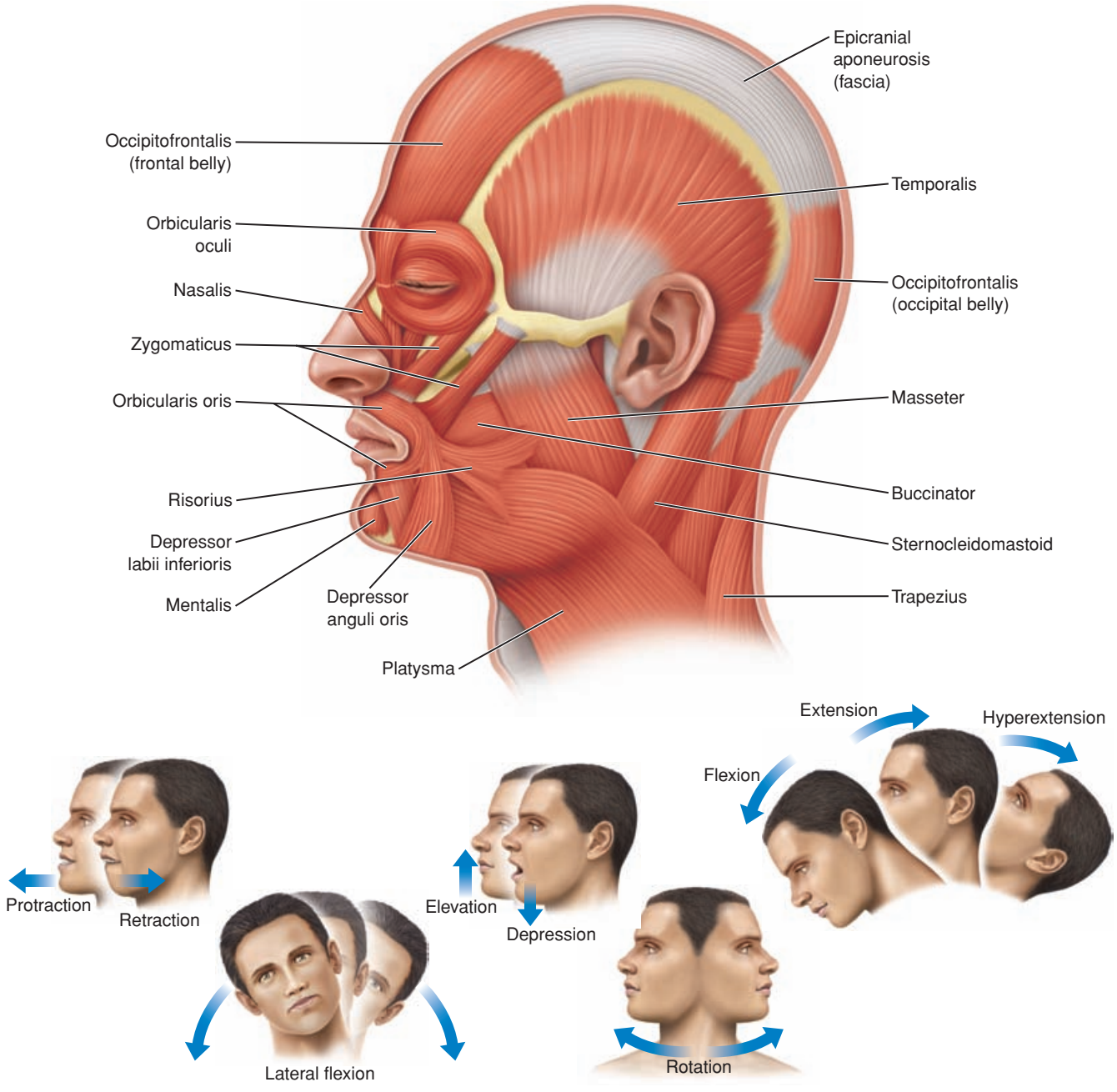
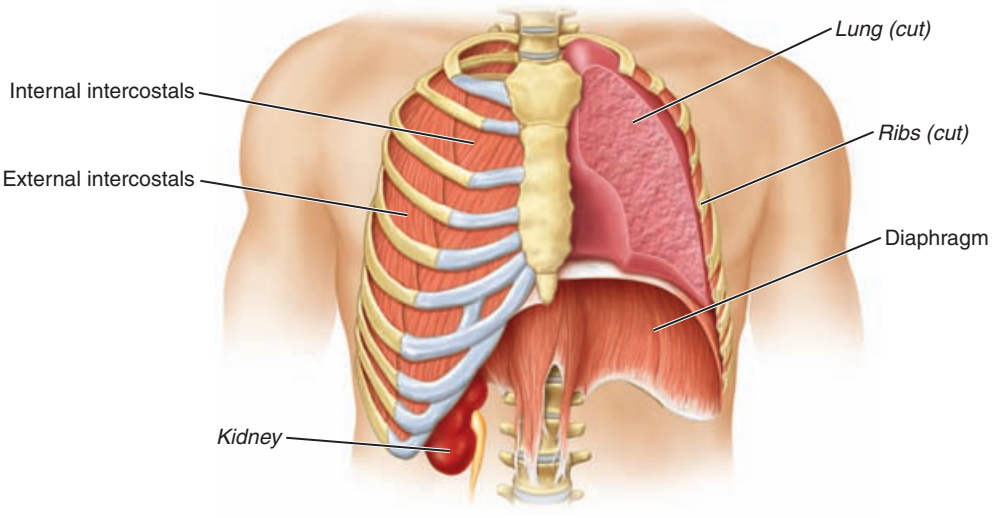


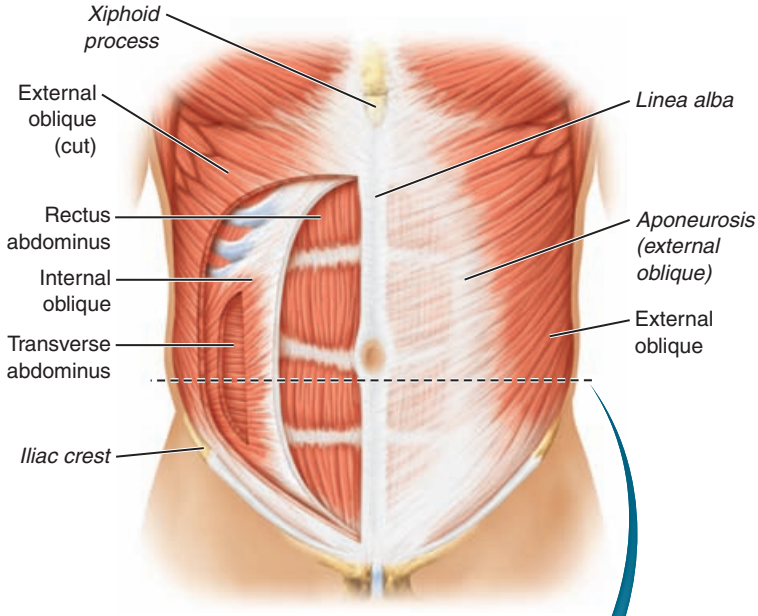
Plate 7.2 Muscles Controlling the Jaw and Moving the Head

Name	Origin	Insertion	Action
The powerful muscles of the jaw move it upwards and sideways for speech and chewing (gravity is the prime mover for depressing the jaw). Yet other muscles move the entire head – flexing, extending, and rotating it on the cervical axis.			
Masseter *(maseter = chewer)*	Temporal bone (zygomatic process)	Mandible	Elevates jaw (biting, chewing)
Temporalis *(temporal = of the side of the head)*	Temporal bone	Mandible	Elevates jaw, retracts chin
Pterygoids *(deep muscles; not shown)*	Sphenoid bone	Mandible	Elevates jaw, protrudes chin
Sternocleidomastoid *(sternon = breastbone; cleido = clavicle; mastoid = mastoid process of temporal bone)*	Sternum, clavicle	Temporal bone (mastoid process), occipital bone	Together: flexes neck (brings chin to chest) Separately: laterally flexes, rotates neck (ear approaches shoulder on same side)
Trapezius (also see Plate 7.5) *(trapezoid = flat with four sides)*	Occipital bone, vertebrae (C7, thoracic)	Clavicle, scapula (acromion, spine)	Extends neck; also moves shoulder
Erector spinae (see Plate 7.3) *(erector = raise; spinae = of the spine)*	Ribs and vertebrae	Occipital bone, temporal bone, ribs, vertebrae	Extends neck (also moves vertebral column)

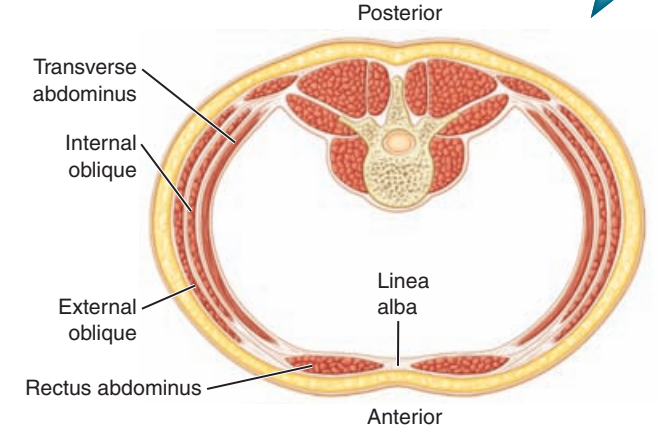
Plate 7.3 Muscles of the Thorax: Muscles that Move the Vertebral Column, Abdominal Muscles, and Respiratory Muscles



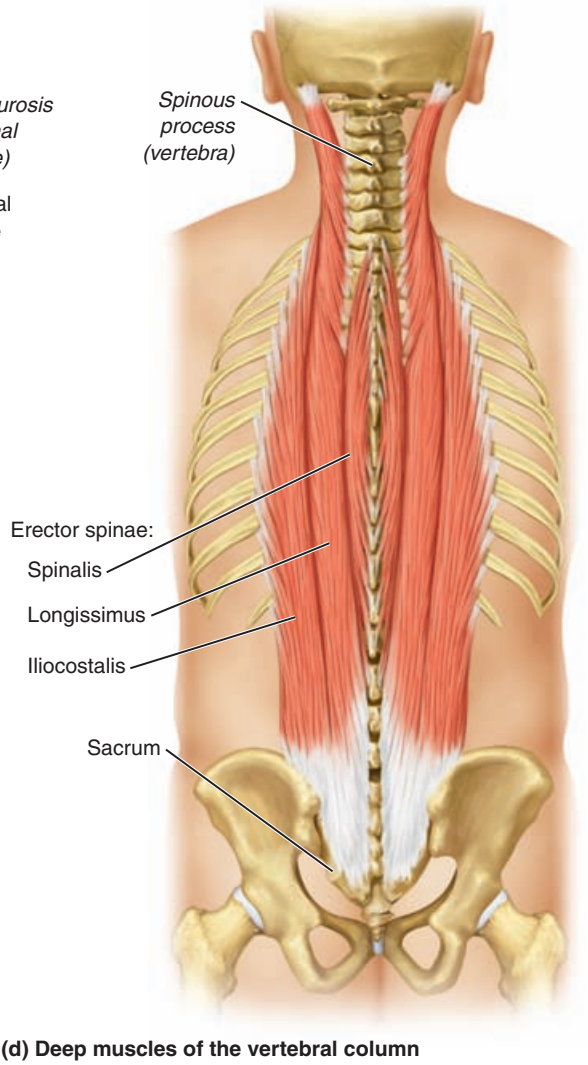
(a) Muscles of respiration



(b) Abdominal muscles, frontal view



(c) Abdominal muscles, transverse section



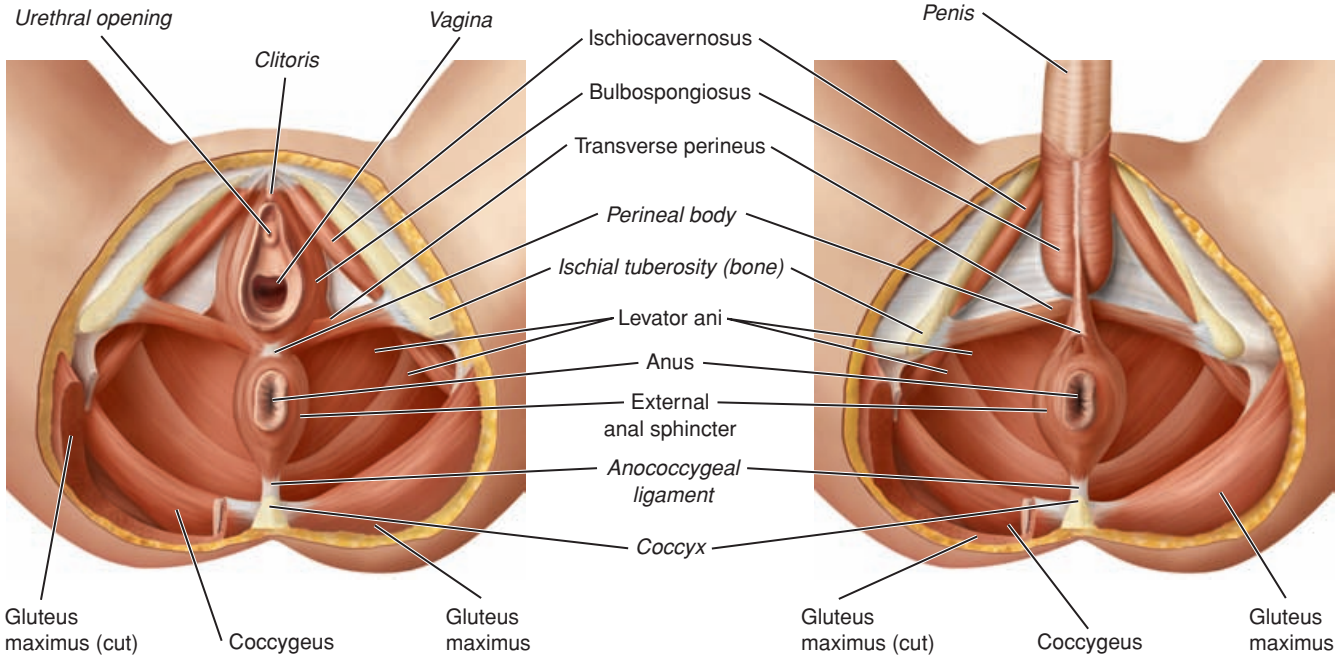
(d) Deep muscles of the vertebral column

Plate 7.3 Muscles of the Thorax: Muscles that Move the Vertebral Column, Abdominal Muscles, and Respiratory Muscles

Name	Origin	Insertion	Action
Many muscles illustrated on this plate do not move bones. Instead, they are involved in the minute movements of respiration and in compressing the abdominal contents.			
Rectus abdominus *(rectus = straight; abdominus = abdomen)*	Pubis	Xiphoid process (sternum), ribs	Flexes spinal column, compresses abdomen
External oblique *(external = closer to the surface; oblique = slanting)*	5th–12th ribs	Ilium, pubis, linea alba	Both: flex spinal column, compress abdomen One: rotate, laterally flex vertebral column
Internal oblique *(internal = farther from the surface; oblique = slanting)*	Iliac crest	10th–12th ribs, linea alba	Same as external obliques
Transverse abdominis *(transverse = across; abdominis = abdomen)*	Iliac crest, intercostal cartilage of 7th–12th ribs	Xiphoid process, linea alba, pubis	Compresses abdomen
Erector spinae: spinalis, longissimus, and iliocostalis groups *(erector = raise; spinae = of the spine; longissimus = longest; iliocostal = related to the ribs)*	Tendon arising at ilium, sacrum, and lumbar vertebrae	Occipital bone, temporal bone, ribs, vertebrae	Both sides: extends vertebral column (also extends head) One side: laterally flexes vertebral column
Sternocleidomastoid (see Plate 7.2)	Sternum, clavicle	Temporal bone (mastoid process), occipital bone	Together: flexes cervical section of vertebral column (also moves head)
Diaphragm *(diaphragma = barrier or partition)*		Xiphoid process, costal cartilage of inferior ribs, lumbar vertebrae	Contracts to expand thorax, resulting in inhalation; relaxes to shrink thorax, resulting in exhalation
Internal intercostals *(internal = farther from the surface; intercostal = between the ribs)*	Superior border of ribs	Inferior border of rib above	Depress ribs; active exhalation
External intercostals *(external = closer to the surface; intercostal = between the ribs)*	Inferior border of ribs	Superior border of rib below	Elevate ribs during inhalation

Plate 7.4 Muscles of the Perineum

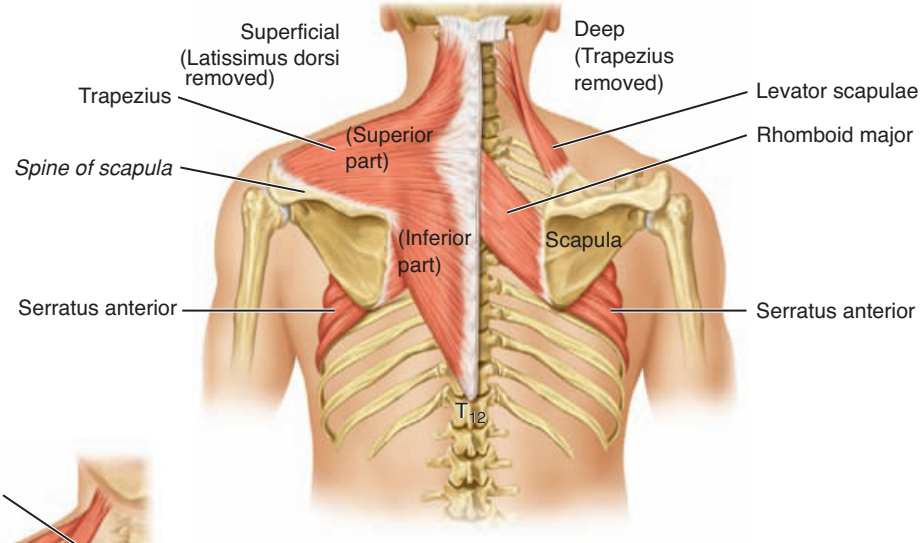
Name	Origin	Insertion	Action
The perineum lies below the pelvic outlet, and is the pelvic floor. Although they receive little attention, the muscles of the perineum support the weight of the abdominal organs, ensure urinary and rectal continence (that is, bladder and bowel control), and participate in reproductive behaviors. The perineal body is a small, but complex, structure composed of connective tissue and muscle; it is the origin for many perineal muscles; this structure is sometimes damaged during childbirth.			
Transverse perineus *(transverse = across; perineum = region between anus and genitals)*	Ischial tuberosity	Perineal body	Stabilizes perineum
Levator ani *(levator = raiser; ani = anus)*	Pubis, ischial spine	Coccyx, urethra, rectum, perineum	Aids defecation; stabilizes perineum
External anal sphincter *(external = closer to the surface; anal = anus; sphincter = tightener)*	Anococcygeal ligament, coccyx	Perineal body	Closes anus
Ischiocavernosus *(ischio = pelvis; cavernosus = hollow tissue of penis or clitoris)*	Ischial tuberosity, pubis	Clitoris (females), penis (males)	Maintains clitoral or penile erection by compressing veins
Bulbospongiosus *(bulbo = swollen; spongiosus = like a sponge)*	Penis (males) or Perineal fascia (females)	Perineal body, clitoris (females), penis (males)	Maintains clitoral or penile erection by compressing veins; aids in expelling last drops of urine or semen (males); constricts vagina (females)
Coccygeus *(coccyx = lower tip of spine)*	Ischium	Coccyx, lower sacrum	Stabilizes perineum; pulls coccyx forward during defecation, childbirth



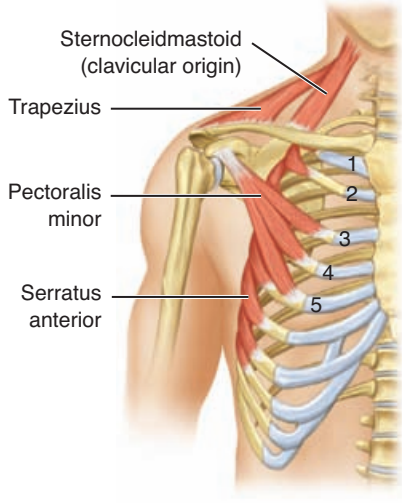
(a) Female (b) Male

Plate 7.5 Muscles that Move and Stabilize the Pectoral Girdle

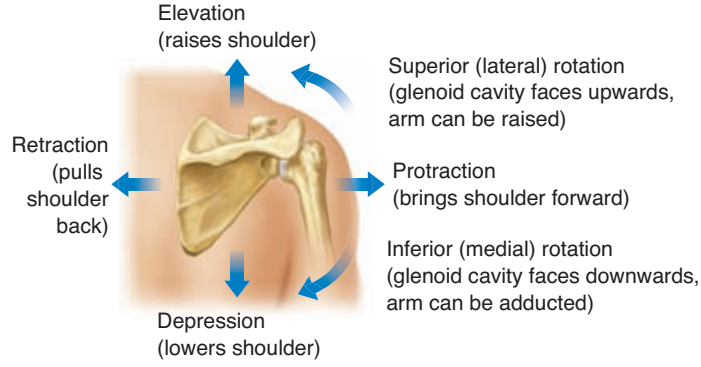
Name	Origin	Insertion	Action
The muscles shown here anchor the upper limbs to the body by fixing the scapula in place. They also move the scapula, thereby moving the shoulder up or down, forward or backward. They also rotate the glenoid cavity, containing the head of the humerus, to enable lateral movements of the humerus.			
Levator scapulae *(levator = raiser; scapulae = scapula)*	Vertebrae C1–C4 (transverse processes)	Scapula (coracoid process)	Elevates and rotates the scapula inferiorly; fixes scapula (also flexes neck laterally)
Trapezius *(trapezi = shaped like a trapezoid)*	Occipital bone, vertebrae (C7, thoracic)	Clavicle, scapula (acromion, spine)	Superior part elevates scapula, inferior part depresses scapula; both parts together retract scapula
Pectoralis minor *(pector = chest; minor = lesser)*	2nd–5th ribs	Scapula (coracoid process)	Protracts scapula
Rhomboid major *(rhomboid = shaped like a rhombus; major = greater)*	Vertebrae T1–T4	Scapula	Retracts and rotates scapula inferiorly; used for forcible downward movements (like hammering)
Serratus anterior *(serratus = saw-toothed; anterior = before)*	Superior ribs	Scapula	Called the boxers muscle; important in punching and pushing because it protracts and stabilizes the scapula so that the shoulder moves down and forward; rotates scapula superiorly



Posterior view



Anterior view



Movements of the scapula

Plate 7.6 Muscles that Move the Arm (Humerus) at the Shoulder Joint

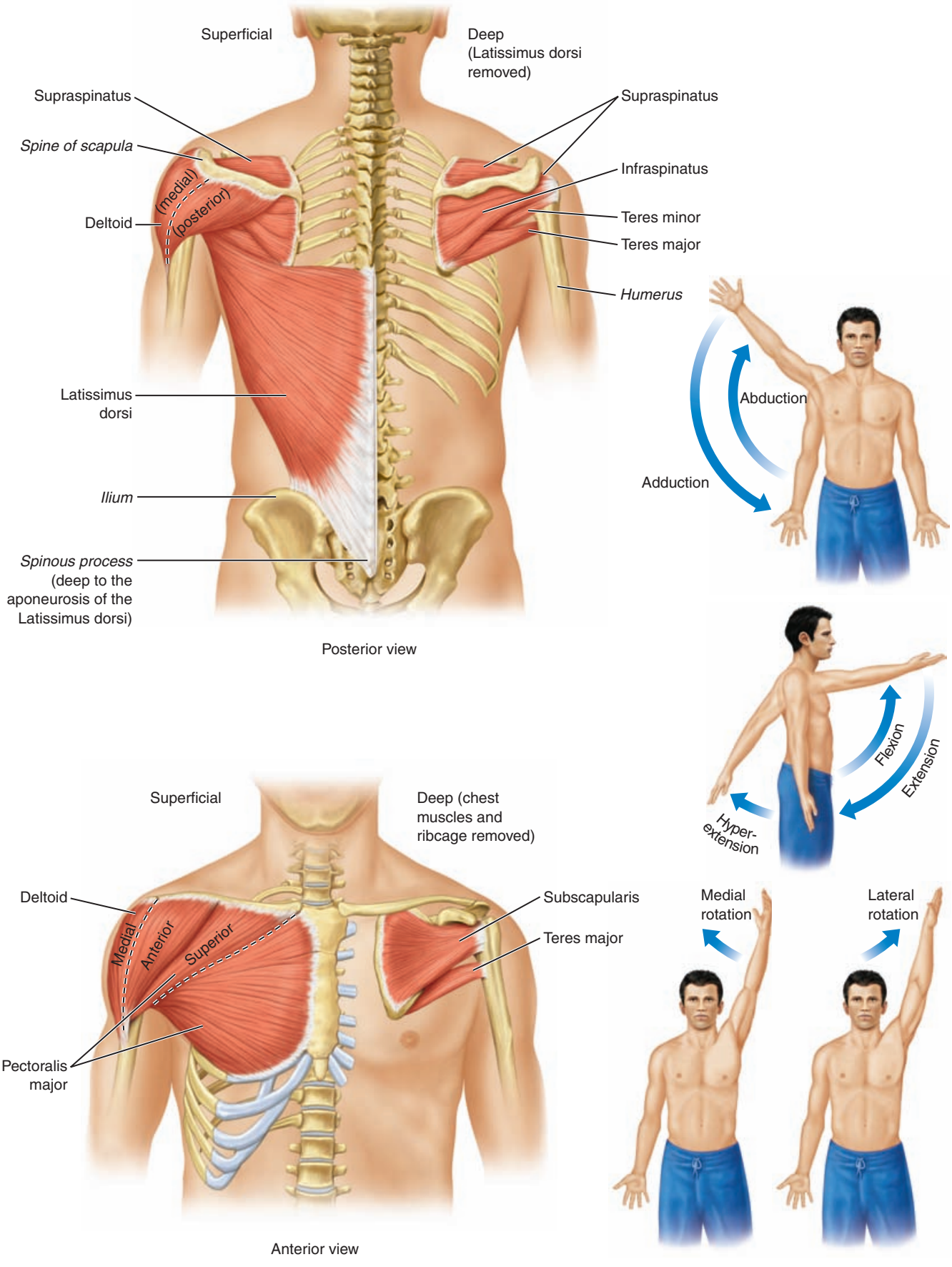
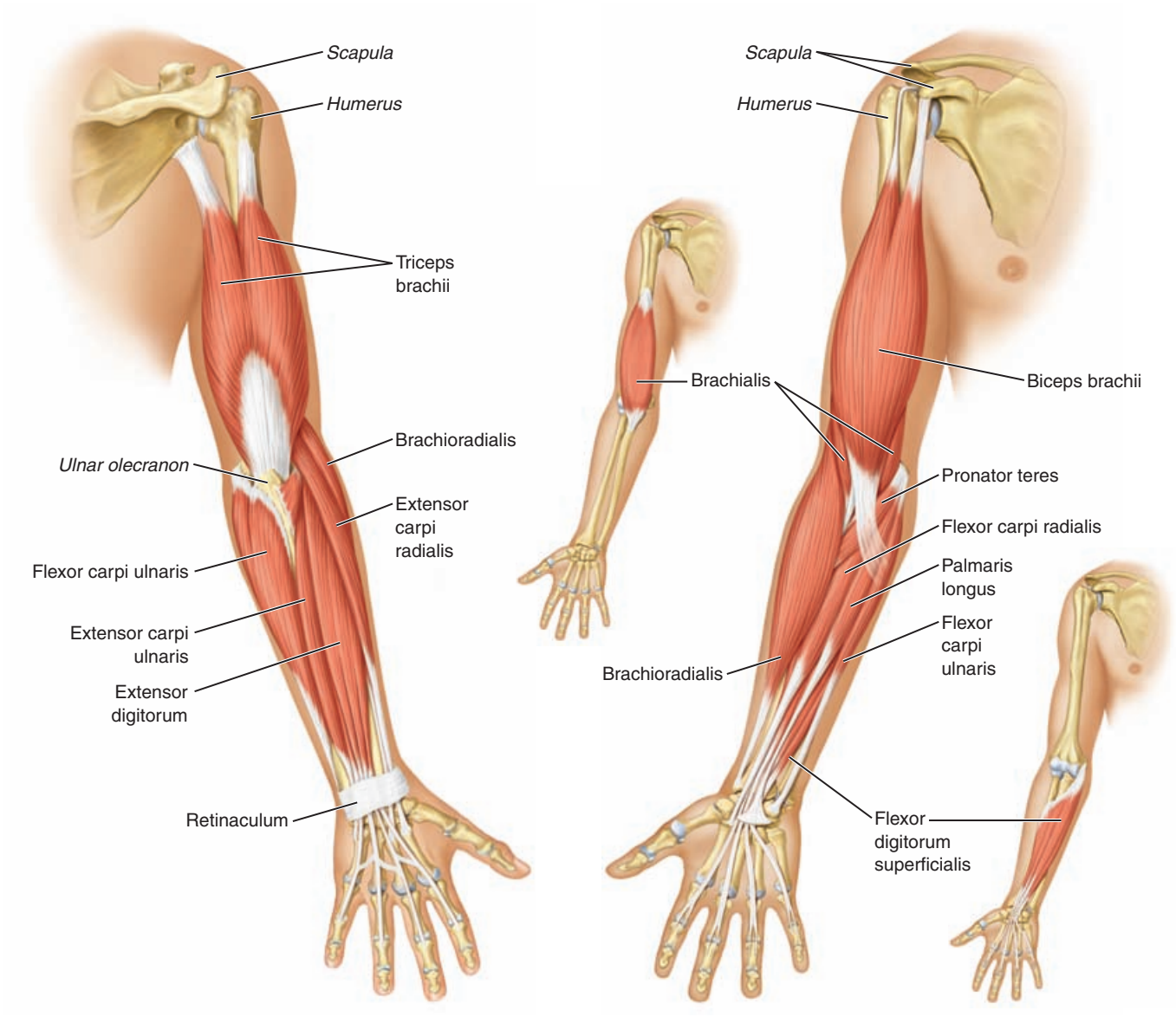


Plate 7.6 Muscles that Move the Arm (Humerus) at the Shoulder Joint

Name	Origin	Insertion	Action
The shoulder joint is the most mobile joint in the body, moving the humerus in all possible dimensions. The shoulder joint is stabilized in part by the "rotator cuff" muscles, also known as the SITS muscles (supraspinatus, infraspinatus, teres minor, subscapsularis). Their tendons form the rotator cuff by blending with the articular capsule to strengthen and stabilize the shoulder joint. Also, the muscles are tonically contracted to hold the humeral head in the shallow glenoid cavity.			
Latissimus dorsi *(latissimus = widest; dorsi = of the back)*	Vertebrae, sacrum, ilium	Humerus	"Climbing muscle"; extends and hyperextends humerus; adducts humerus behind back (i.e., to scratch an itch over the scapula); medially rotates humerus
Pectoralis major *(pector = chest; major = greater)*	Clavicle, sternum, cartilage of ribs	Humerus	Adducts and medially rotates humerus; superior portion flexes humerus
Teres major *(teres = long and round; major = greater)*	Scapula	Humerus	Adducts, medially rotates humerus; helps in extension from flexed position; helps stabilize shoulder joint when deltoid is active
Supraspinatus* *(supra = above; spina = spine of scapula)*	Scapula	Humerus	Assists deltoid to complete abduction
Infraspinatus* *(infra = below; spina = spine of scapula)*	Scapula	Humerus	Laterally rotates humerus
Teres minor* *(teres = long and round; minor = lesser)*	Scapula	Humerus	Laterally rotates humerus
Subscapularis* *(sub = beneath; scapularis = scapula)*	Subscapular fossa	Humerus	Medially rotates humerus
Deltoid *(deltoid = shaped like a triangle)*	Clavicle, scapula (spine and acromion)	Humerus	Forms rounded contour of shoulder; entire muscle abducts humerus; swings arms during walking (anterior part helps pectoralis major flex humerus; posterior part helps latissimus dorsi extend humerus)

**Part of the rotator cuff*

Plate 7.7 Muscles that Move the Forearm, Hand, and Fingers



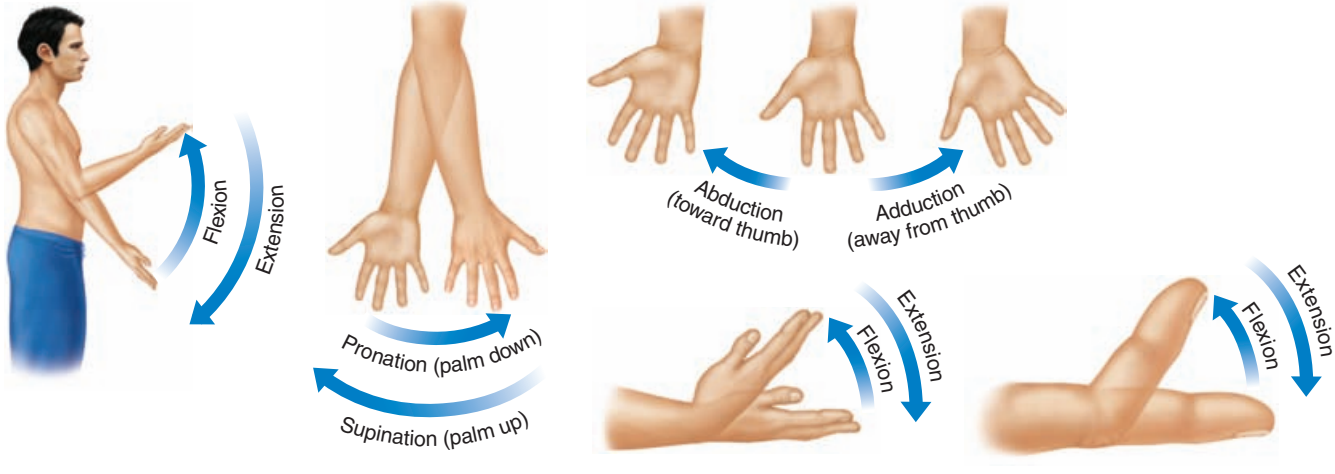
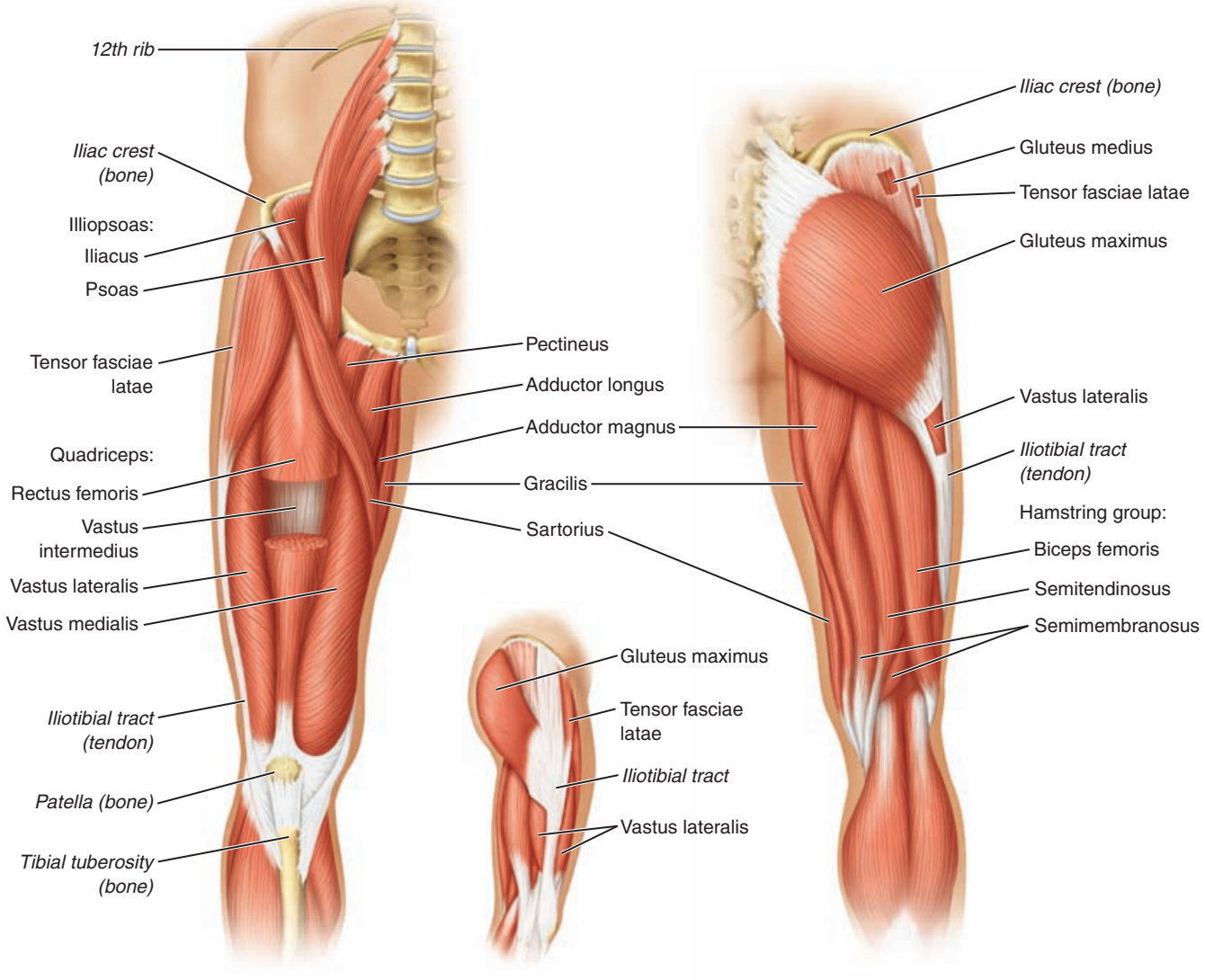


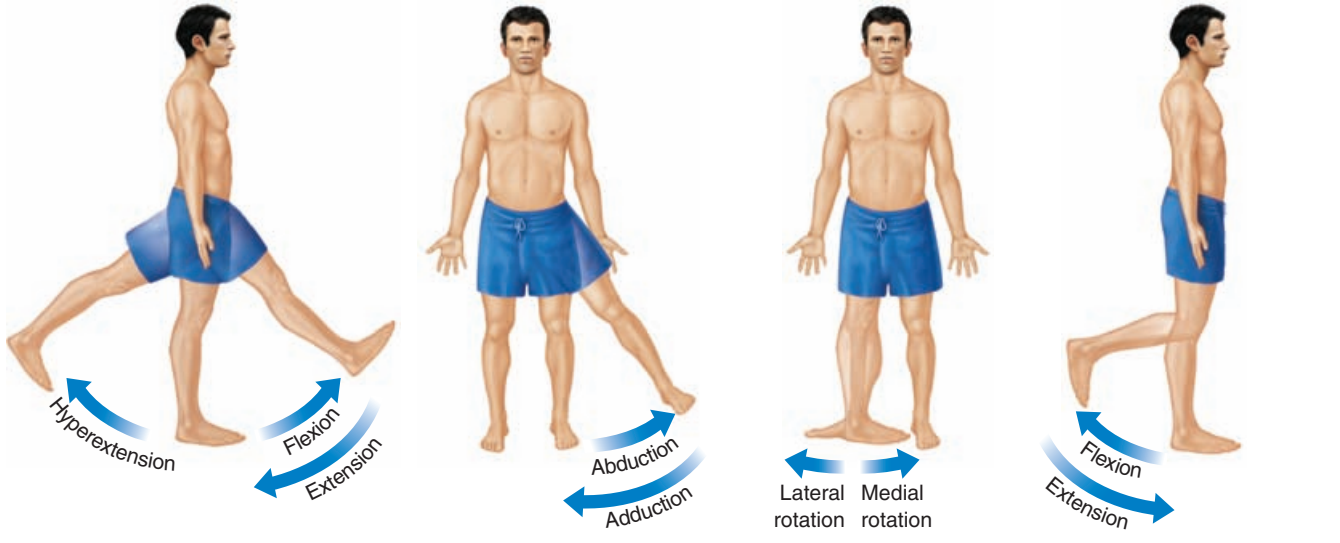
Plate 7.7 Muscles that Move the Forearm, Hand, and Fingers

Name	Origin	Insertion	Action
The muscles of the forearm can be separated into four groups: 1) those that flex and extend at the elbow joint; 2) those that flex or extend at the wrist joint; 3) those that flex or extend the fingers and thumb; and 4), those at that pronate or supinate the forearm.			
Brachialis *(brachi = arm)*	Humerus	Ulna	Flexes elbow (primary mover, all positions)
Brachioradialis *(brachi = arm; radi = radius)*	Humerus	Radius	Flexes elbow; assists brachialis when quick movements are required
Biceps brachii *(biceps = two heads; brachi = arm)*	Scapula (coracoid process and tubercle)	Radius, forearm fascia	Supinates elbow (primary mover), flexes elbow when forearm is supine (not when pronated)
Triceps brachii *(triceps = three heads; brachi = arm)*	Scapula, humerus	Ulnar olecranon	Extends elbow (primary mover)
Extensor carpi radialis *(extensor = increases joint angle; carpus = wrist; radi = radius)*	Humerus	2nd metacarpal	Extends, abducts wrist; necessary to clench fist
Pronator teres *(pronate = turn palm down; teres = long and round)*	Humerus, ulnar coronoid process	Radius	Pronates, flexes elbow
Flexor carpi radialis *(flex = decreases joint angle; carpus = wrist; radi = radius)*	Humerus	2nd and 3rd metacarpals	Flexes, abducts wrist (hand moves anterolaterally)
Palmaris longus *(palma = palm; longus = long)*	Humerus	Fascia	Weak wrist flexor
Flexor carpi ulnaris *(flex = decreases joint angle; carpus = wrist; ulnaris = ulna)*	Humerus, ulna	5th metacarpal	Flexes, adducts wrist
Extensor carpi ulnaris *(extensor = increases joint angle; carpus = wrist; ulnaris = ulna)*	Humerus, posterior ulna	5th metacarpal	Extends, adducts wrist; necessary to clench fist
Flexor digitorum superficialis *(flex = decreases joint angle; digit = finger or toe; superficial = near the surface)*	Humerus, ulna, radius	Middle phalanx, each finger	Flexes four fingers at proximal IP joint
Extensor digitorum *(extensor = increases joint angle; digit = finger or toe)*	Humerus	Distal and middle phalanges, each finger	Extends four fingers at all IP joints

Plate 7.8 Muscles that Move the Thigh and Leg



Anterior view | Lateral view | Posterior view



Movements at the hip (of the thigh) | Movements at the knee (of the leg)

Plate 7.8 Muscles that Move the Thigh and Leg

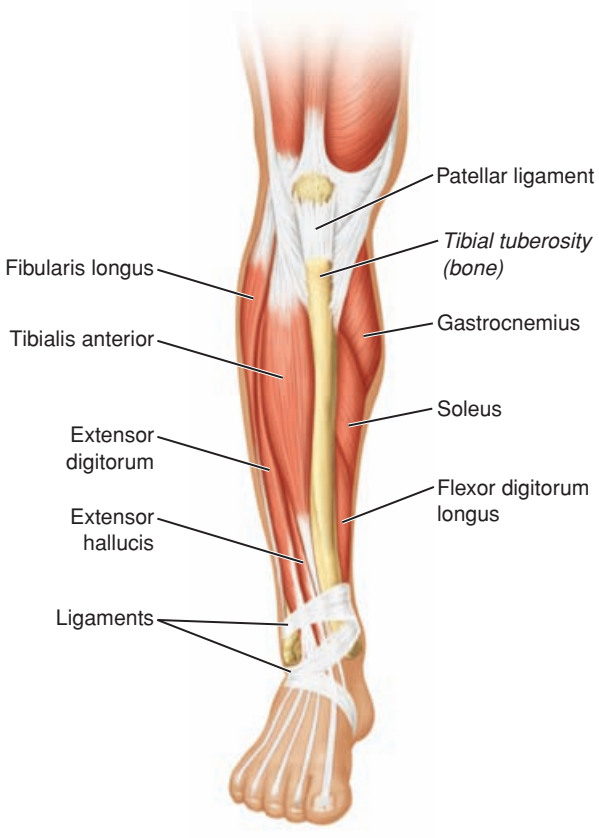
Name	Origin	Insertion	Action
Muscles of the thigh are large and powerful, befitting their role in maintaining an erect stance, in walking or running, or in lifting heavy loads. Some of these muscles act only to move the hip joint; others, the knee; and some move both. The anterior muscles generally act to flex the hip and extend the knee, as in the foreswing of walking. The posterior muscles generally act to extend the hip and flex the knee, as in the backswing of walking. A third large and powerful group, located medially, acts to adduct the thigh and has no effect on the leg. Abduction and rotation of the thigh is relatively weak and attained by small muscles or is a minor function of large muscles.			
Iliacus *(iliac = ilium)*	Ilium	Femur (lesser trochanter)	Flexes, laterally rotates hip; flexes vertebral column
Psoas *(psoas = muscle of the loin)*	Lumbar vertebrae	Joins iliacus to insert into femur (lesser trochanter)	Flexes, laterally rotates hip; flexes vertebral column
Sartorius *(sartor = tailor, referencing their traditional cross-legged position)*	Iliac spine	Tibia	Crosses the leg: flexes, abducts, and laterally rotates hip; flexes knee
Quadriceps Femoris: *(quadriceps = four heads; femoris = femur)*			
Rectus femoris *(rectus = straight; femoris = femur)*	Iliac spine	Four muscles join and insert into the patella, then the tibial tuberosity	Extends knee; flexes hip
Vastus lateralis *(vastus = large; lateralis = lateral)*	Femur (greater trochanter, linea aspera)		Extends knee
Vastus medialis *(vastus = large; medialis = medial)*	Femur (greater trochanter, linea aspera)		Extends knee
Vastus intermedius *(vastus = large; intermedius = middle)*	Femur		Extends knee
Gracilis *(gracile = slender)*	Pubis	Tibia	Adducts and medially rotates hip; flexes knee
Adductor longus *(adduct = move toward the centerline; longus = long)*	Pubic crest and symphysis	Femur (linea aspera)	Adducts, medially rotates, and flexes hip
Adductor magnus *(adduct = move toward the centerline; magnus = large)*	Pubis, ischium	Femur (linea aspera)	Adducts, medially rotates, and extends hip
Pectineus *(pectin = comb)*	Pubis	Femur	Adducts, flexes hip

Plate 7.8 Muscles that Move the Thigh and Leg (continued)

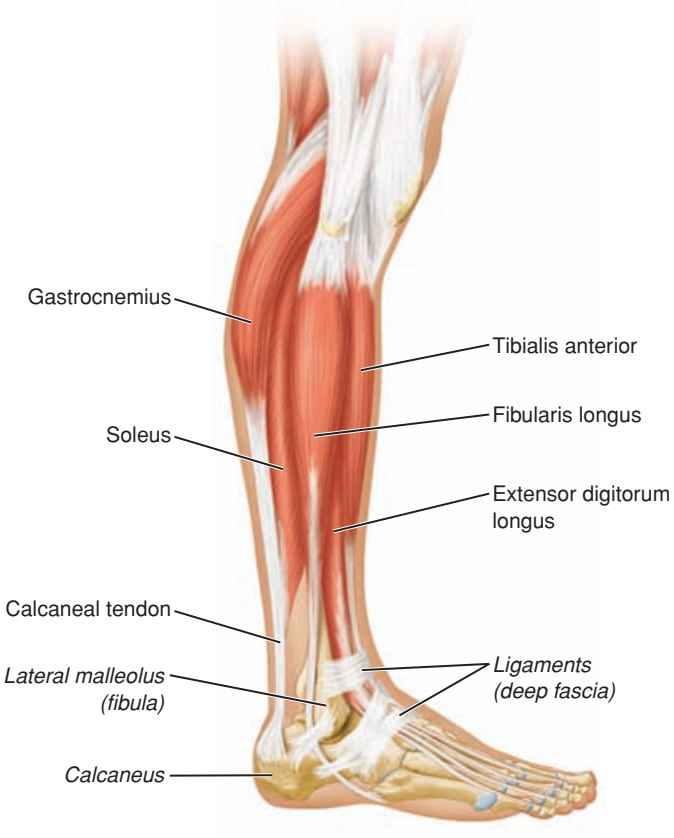
Name	Origin	Insertion	Action
Tensor fasciae latae *(tensor = tightener; fasciae = fascia; lat = wide)*	Ilium	Iliotibial tract, eventually tibia	Abducts, flexes hip
Gluteus medius *(glute = buttock; medius = middle)*	Ilium	Femur (greater trochanter)	Abducts, laterally rotates hip
Gluteus maximus *(glute = buttock; maximus = largest)*	Iliac crest, sacrum, coccyx	Iliotibial tract, femur (linea aspera)	Extends, laterally rotates hip
Hamstring group: *(referring to the tendons behind the knee)*			
Biceps femoris *(biceps = two heads; femoris = femur)*	Ischial tuberosity, linea aspera of femur	Fibula (head) and tibia (lateral condyle)	Flexes knee; extends hip
Semitendinosus *(semi = half; tendo = tendon)*	Ischial tuberosity	Proximal tibia	Flexes knee; extends hip
Semimembranosus *(semi = half; membran = membrane)*	Ischial tuberosity	Tibia (medial condyle)	Flexes knee; extends hip

Plate 7.9 Muscles that Move the Foot and Toes

Name	Origin	Insertion	Action
Muscles of the leg move the foot and toes and are divided into anterior, posterior and lateral groups according to their position in relation to the interosseous membrane that joins the tibia and fibula. Muscles in the anterior compartment extend the toes and dorsiflex the ankle, a weak movement but one critical to avoid dragging the toes while walking. Muscles in the posterior compartment flex the toes and plantarflex the foot, powerful movements when walking. Muscles in the lateral compartment evert the foot. The small muscles of the foot aid in all movements of the toes and help fine tune and stabilize body movements involving the foot.			
Tibialis anterior *(tibialis = tibia; anterior = front)*	Tibia: lateral condyle/body	1st cuneiform and metatarsal	Dorsiflexes, inverts ankle
Extensor digitorum longus *(extensor = increase joint angle; digitorum = finger or toe; longus = long)*	Tibia	Distal phalanges, 2nd to 5th toes	Extends 4 toes, dorsiflexes ankle
Extensor hallucis *(extensor = increase joint angle; hallux = great toe)*	Fibula	Phalanx of great toe	Extends great toe, dorsiflexes ankle
Fibularis longus *(fibularis = fibula; longus = long)*	Fibula, tibia (lateral condyle)	Medial cuneiform and first metatarsal of foot	Everts ankle; keeps leg steady when balancing on one foot
Gastrocnemius *(gastro = belly; cnem = leg)*	Femur: lateral, medial condyles	Calcaneus (via Achilles tendon)	Plantarflexes ankle; raises heel when walking; flexes knee; important in rapid movements (running, jumping)



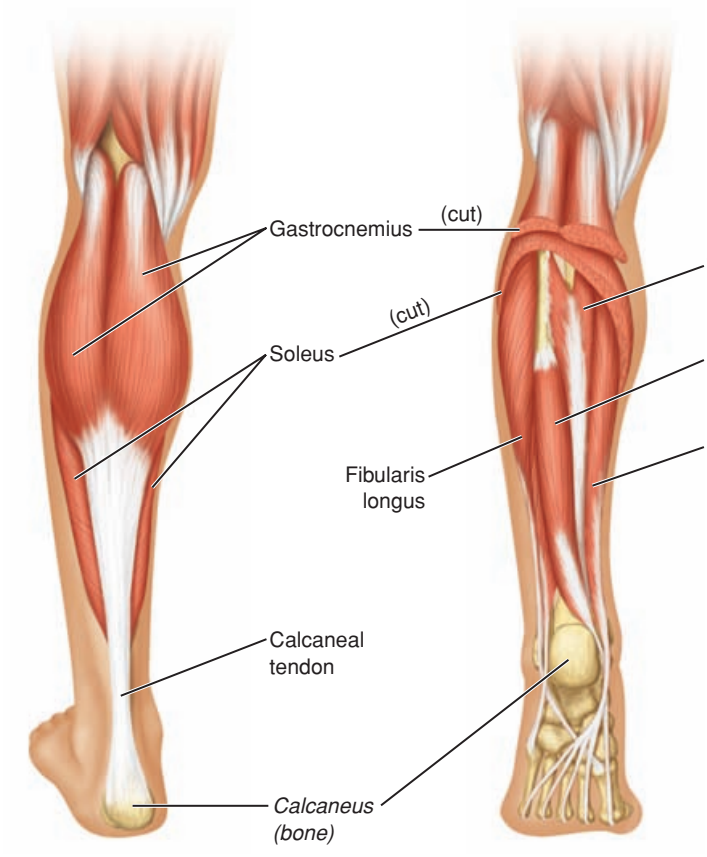
Anterior view, right leg



Lateral view, right leg

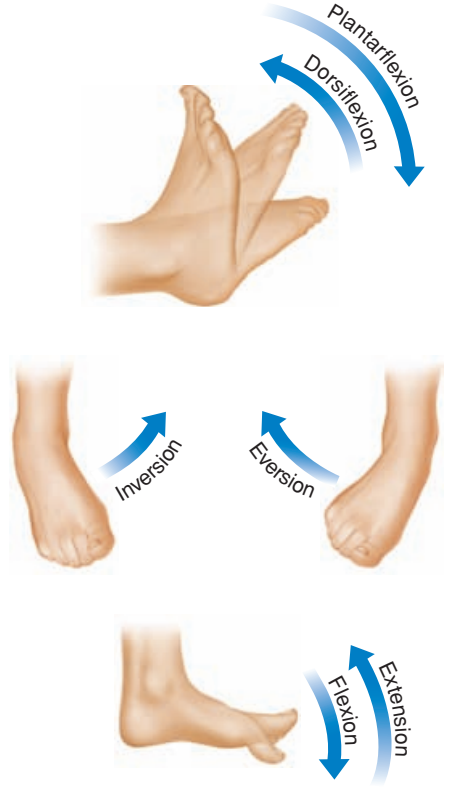
Plate 7.9 Muscles that Move the Foot and Toes (continued)

Name	Origin	Insertion	Action
Soleus *(soleus = a flat fish)*	Fibula (head) and proximal tibia	Calcaneus (via Achilles tendon)	Plantarflexes ankle (slow); contracts alternately with leg extensor muscles to maintain balance when walking
Tibialis posterior *(tibialis = tibia; posterior = rear)*	Tibia, fibula	Multiple tarsals and metatarsals	Plantarflexes and inverts ankle
Flexor digitorum longus *(flexor = decreases joint angle; digitorum = finger or toe; longus = long)*	Posterior tibia	Distal phalanges, 2nd to 5th toes	Flexes lateral 4 toes, plantarflexes ankle, supports longitudinal foot arches
Flexor hallucis *(flexor = decreases joint angle; hallux = great toe)*	Posterior fibula	Base of great toe	Flexes great toe; supports longitudinal foot arches; push-off muscle during running and jumping



Posterior superficial view, left leg

Posterior deep view, left leg (foot plantar flexed)



Word Parts

Latin/Greek Word Parts	English Equivalents	Examples
my/o	Muscle	myoglobin; a globular protein found in muscle
sarco-	Flesh; muscle	sarcolemma: membrane (-lemma) of a muscle cell
tropo-	To turn	troponin: molecule (-in) that turns (moves tropomyosin out of the way) in order to produce muscle contraction
-metric	Length	isometric: contraction with no change (iso-) in length
-ton/o	Tension	isotonic: contraction with no change (iso-) in tension
con-	Together	concentric: muscle contraction that brings two bones together (a shortening contraction)
ec-	Away	eccentric: muscle contraction that moves two bones away from each other (a lengthening contraction)
syn-	Together	synergist: muscle working together with the prime mover
ant-	Against	antagonist: muscle working against the prime mover

Chapter Challenge

CHAPTER RECALL

1. **Which of the following characteristics apply to skeletal muscle (SK) and which to smooth muscle (SM)? Write all that apply.**
   a. Muscle is striped (striated) in appearance.
   b. Muscle contractions cannot be consciously controlled.
   c. Muscle tissue found in the stomach and intestinal wall.
   d. Muscle fibers that fatigue after repeated contractions.

2. **The outer membrane of a muscle cell is called the**
   a. sarcoplasm.
   b. sarcolemma.
   c. sarcoplasmic reticulum.
   d. endosomal membrane.

3. **Which of the following statements applies to the neuromuscular junction of skeletal muscles?**
   a. Uses norepinephrine as the neurotransmitter.
   b. Consists of multiple varicosities scattered over numerous muscle fibers.
   c. Its activation results in calcium entering the cell from the extracellular fluid.
   d. The neurotransmitter receptors are also sodium channels.

4. **Thin filaments**
   a. are anchored by dense bodies in smooth muscle.
   b. are composed only of actin.
   c. are found in skeletal muscle but not smooth muscle.
   d. are composed of myosin.

5. **A sarcomere**
   a. is a neuron and the muscle fiber it innervates.
   b. runs the entire length of a muscle.
   c. is joined to adjacent sarcomeres by the Z disc.
   d. is the functional unit of both skeletal and smooth muscle.

6. **During muscle contraction,**
   a. thick filaments shorten.
   b. thin filaments shorten.
   c. both thick and thin filaments shorten.
   d. neither thick nor thin filaments shorten.

7. **The role of calcium in smooth muscle contraction involves**
   a. activating enzymes that activate the myosin heads.
   b. providing energy for cross-bridge cycling.
   c. revealing myosin binding sites on actin molecules.
   d. initiating action potentials in the muscle cell.

8. **In skeletal muscle, fresh molecules of ATP are required for**
   a. detaching the myosin heads from the actin.
   b. maintaining the sodium concentration gradient.
   c. providing the energy for movement of the myosin heads.
   d. all of the above.

9. **Type I fibers**
   a. are slow to fatigue.
   b. contain large stores of glycogen.
   c. are the strongest type of fiber.
   d. contain few mitochondria.

10. **An example of an isometric contraction during a pushup would be**
    a. holding yourself immobile in the pushup position.
    b. contracting the triceps brachii as you lower your body to the floor.
    c. contracting the biceps brachii as you raise your body from the floor.
    d. b and c.

11. **The eyelid is closed by the actions of the**
    a. orbicularis oris.
    b. orbicularis oculi.
    c. mentalis.
    d. occipitofrontalis.

12. **Contraction of the sternocleidomastoid muscle on one side of the body only will**
    a. raise one shoulder.
    b. lower one shoulder.
    c. bring one ear closer to the shoulder on the same side.
    d. move one shoulder anteriorly.

13. **The most superficial abdominal muscle is the**
    a. transverse abdominis.
    b. internal oblique.
    c. external oblique.
    d. rectus abdominis.

14. **The muscle that retracts the scapula is the**
    a. serratus anterior.
    b. pectoralis minor.
    c. pectoralis major.
    d. rhomboid major.

15. **Which of the following muscles is *not* part of the rotator cuff?**
    a. Deltoid
    b. Supraspinatus
    c. Teres minor
    d. Subscapularis

16. **The primary mover for forearm flexion is the**
    a. biceps brachii.
    b. brachialis.
    c. brachioradialis.
    d. triceps brachii.

17. **The thigh muscle that originates on the ilium and inserts into the greater trochanter of the femur is the**
    a. sartorius.
    b. psoas.
    c. gluteus medius.
    d. tensor fascia lata.

18. **The muscle that abducts the thigh is the:**
    a. gacilis.
    b. pectineus.
    c. adductor longus.
    d. gluteus medius.

19. **The levator ani originates on the**
    a. ischium.
    b. pubis.
    c. perineal fascia.
    d. ilium.

20. **Contraction of the extensor hallucis would result in**
    a. inversion.
    b. eversion.
    c. plantarflexion.
    d. dorsiflexion.

21. **Which of the following muscles is part of the hamstring group?**
    a. Gracilis
    b. Vastus lateralis
    c. Semimembranosus
    d. Rectus femoris

22. **Which of the following muscles extends the hand?**
    a. Palmaris longus
    b. Extensor digitorum
    c. Extensor carpi ulnaris
    d. Pronator teres

23. **Hiking to the top of a mountain, you encounter a view so beautiful that your jaw drops, opening your mouth. The prime mover for this action is**
    a. the temporalis muscle.
    b. the masseter muscle.
    c. the depressor labii inferioris muscle.
    d. gravity.

24. **You have your eye on a delicious chocolate chip cookie at a bake sale. But as your hand reaches out to get the cookie, someone else snatches it. You walk away, pouting. The prime mover in this action of the lips is the**
    a. mentalis.
    b. buccinators.
    c. zygomaticus.
    d. risorius.

25. **Which of the following muscles strongly flexes the spinal column?**
    a. rectus abdominis
    b. erector spinae
    c. transverse abdominis
    d. internal intercostals

CONCEPTUAL UNDERSTANDING

26. **List five functions of muscle tissue. Specify the type of muscle tissue involved—skeletal, cardiac, and/or smooth.**

27. **Compare and contrast anaerobic metabolism and aerobic metabolism under the following categories:**
    a. nutrient types used
    b. approximate number of ATP molecules produced from one glucose molecule (and, if appropriate, fatty acid molecule)
    c. requirement for oxygen

28. **Returning for your second year of college, you notice that your friend has significantly "bulked up" and his muscles are visibly larger.**
    a. What sort of exercise results in significant muscle growth?
    b. Discuss the role of satellite cells in muscle growth.

APPLICATION

29. **A new drug has been developed that blocks acetylcholinesterase. You are looking for something to relax your muscles. Would this drug be appropriate to use? Explain why or why not.**

30. **While attending the ballet, you notice a dancer raising her heels to stand on her tiptoes.**
    a. Name this action using the movement terminology you learned in Chapter 6.
    b. What is the prime mover for this action?
    c. Name a synergistic muscle involved in this action.
    d. Name an antagonistic muscle involved.

You can find the answers to these questions on the student Web site at
http://thepoint.lww.com/McConnellandHull