In Chapter 10, we explored the anatomy of skeletal muscle organs and how they work together to accomplish specific body movements. In this chapter, we continue our study of the muscular system by examining the basic characteristics of skeletal muscle tissue. We uncover the mechanisms that permit skeletal muscle tissue to move the body’s framework, as well as perform other functions vital to maintaining a constant internal environment. We also briefly examine smooth and cardiac muscle tissues, contrasting them with skeletal muscle tissue.

GENERAL FUNCTIONS

If you have any doubts about the importance of muscle function to normal life, think about what it would be like without it. It is hard to imagine life with this matchless power lost. However, as cardinal as it is, movement is not the only contribution muscles make to healthy survival. They also perform two other essential functions: production of a large portion of body heat and maintenance of posture.

1. Movement. Skeletal muscle contractions produce movements of the body as a whole (locomotion) or of its parts.
2. Heat production. Muscle cells, like all cells, produce heat by the process known as catabolism (discussed in Chapters 4 and 27). But because skeletal muscle cells are both highly active and numerous, they produce a major share of total body heat. Skeletal muscle contractions therefore constitute one of the most important parts of the mechanism for maintaining homeostasis of temperature.
3. Posture. The continued partial contraction of many skeletal muscles makes possible standing, sitting, and maintaining a relatively stable position of the body while walking, running, or performing other movements.

FUNCTION OF SKELETAL MUSCLE TISSUE

Skeletal muscle cells have several characteristics that permit them to function as they do. One such characteristic is the ability to be stimulated, often called excitability or irritability. Because skeletal muscle cells are excitable, they can re-
spond to regulatory mechanisms such as nerve signals. **Contractility** of muscle cells, the ability to contract or shorten, allows muscle tissue to pull on bones and thus produce body movement. **Extensibility**, the ability to extend or stretch, allows muscles to return to their resting length after having contracted. These characteristics are related to the microscopic structure of skeletal muscle cells. In the following passages, we first discuss the basic structure of a muscle cell. We then explain how a muscle cell’s structural components allow it to perform its specialized functions.

**OVERVIEW OF THE MUSCLE CELL**

Look at Figure 11-1. As you can see, a skeletal muscle is composed of bundles of skeletal muscle fibers that generally extend the entire length of the muscle. They are called fibers, instead of cells, because of their threadlike shape (1 to 40 mm long but with a diameter of only 10 to 100 μm). Skeletal muscle fibers have many of the same structural parts as other cells. Several of them, however, bear different names in muscle fibers. For example, sarclemma is the plasma membrane of a muscle fiber. Sarcoplasm is its cytoplasm. Muscle cells contain a network of tubules and sacs known as the sarcoplasmic reticulum (SR)—a structure analogous, but not identical, to the endoplasmic reticulum of other cells. Muscle fibers contain many mitochondria, and, unlike most other cells, they have several nuclei.

Certain structures not found in other cells are present in skeletal muscle fibers. For instance, bundles of very fine fibers—myofibrils—extend lengthwise along skeletal muscle fibers and almost fill their sarcoplasm. Myofibrils, in turn, are made up of still finer fibers, called thick and thin myofilaments (Figure 11-1, D). Find the label sarcomere in this drawing. Note that a sarcomere is a segment of the myofibril between two successive Z lines (Box 11-1). Each myofibril consists of

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**Box 11–1  A More Detailed Look at the Sarcomere**

The sarcomere is the basic contractile unit of the muscle cell. As you read the explanation of the sarcomere’s structure and function, you might wonder what the Z line, M line, and other components really are—and what they do for the muscle cell.

First of all, it is important that you appreciate the three-dimensional nature of the sarcomere. You can then realize that the Z line is actually a dense plate or disk to which the thin filaments directly anchor. As a matter of fact, the Z line is often called the Z disk. Besides being an anchor for myofibrils, the Z line is useful as a landmark separating one sarcomere from the next.

Detailed analysis of the sarcomere also shows that the thick (myosin) filaments are held together and stabilized by protein molecules that form the M line. Note that the regions of the sarcomere are identified by specific zones or bands:

- **A band**—the segment that runs the entire length of the thick filaments
- **I band**—the segment that includes the Z line and the ends of the thin filaments where they do not overlap the thick filaments
- **H zone**—the middle region of the thick filaments where they do not overlap the thin filaments
- **Later**, as you review the process of contraction, note how these regions change during each step of the process.

In addition to thin and thick filaments, each sarcomere has numerous elastic filaments. Elastic filaments, composed of a protein called titin (connectin), anchor the ends of the thick filaments to the Z line, as the figure shows. The elastic filaments are believed to give myofibrils, and thus muscle fibers, their characteristic elasticity. **Dystrophin**, not shown here, is a protein that holds the actin filaments to the sarcolemma. Dystrophin and a complex of connected molecules anchors the muscle fiber to surrounding matrix so that the muscle doesn’t break during a contraction. Dystrophin and its role in muscular dystrophy is discussed further on p. 332.
a lineup of many sarcomeres, each of which functions as a contractile unit. The A bands of the sarcomeres appear as relatively wide, dark stripes (cross striae) under the microscope, and they alternate with narrower, lighter colored stripes formed by the I bands (see Figure 11-1, D). Because of its cross striae, skeletal muscle is also called *striated muscle*. Electron microscopy of skeletal muscle (Figure 11-2) has revealed details that have revolutionized our concept of its structure and its function.

Another structure unique to muscle cells is a system of transverse tubules, or *T* tubules. This name derives from the fact that these tubules extend transversely across the sarcoplasm, at a right angle to the long axis of the cell. As Figures 11-1, B, and 11-3 show, the *T* tubules are formed by inward extensions of the sarcolemma. The chief function of *T* tubules is to allow electrical signals, or *impulses*, traveling along the sarcolemma to move deeper into the cell.

The SR is also a system of membranous tubules in a muscle fiber. It is separate from the *T* tube system, forming extensive networks of connected canals and sacs. The membrane of the SR continually pumps calcium ions (Ca**++) from the sarcoplasm and stores them within its sacs. Notice in Figures 11-1, B, and 11-3 that a tubular sac of the SR butts up against each side of every *T* tubule in a muscle fiber. This triplet of tubules (a *T* tubule sandwiched between sacs of the SR) is called a *triad*. The triad is an important feature of the muscle cell because it allows an electrical impulse traveling along a *T* tubule to stimulate the membranes of adjacent sacs of the SR.

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**Figure 11-1** Structure of skeletal muscle. A, Skeletal muscle organ, composed of bundles of contractile muscle fibers held together by connective tissue. B, Greater magnification of single fiber showing smaller fibers—myofibrils—in the sarcoplasm. Note sarcoplasmic reticulum and *T* tubules forming a three-part structure called a *triad*. C, Myofibril magnified further to show sarcomere between successive *Z* lines. Cross striae are visible. D, Molecular structure of myofibril showing thick myofilaments and thin myofilaments.

**Figure 11-2** Electron micrographs of striated muscle. B shows detail of A at greater magnification. Note that the myofilaments of each myofibril form a pattern that, when viewed together, produces the striated pattern typical of skeletal muscle.

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**1.** What are the three major functions of the skeletal muscles?

**2.** Name some features of the muscle cell that are not found in other types of cells.

**3.** What causes the striations observed in skeletal muscle fibers?

**4.** Why is the triad relationship between *T* tubules and SR important?
MYOFILAMENTS

Each muscle fiber contains a thousand or more parallel sub-units, called myofibrils, that are only about 1 μm thick. Lying side by side in each myofibril are thousands of thick and thin myofilaments. Over the years, a clear picture of the molecular structure of myofilaments has emerged. This picture reveals the mechanism of how muscle fibers contract and do so powerfully. It is wise, therefore, to take a moment to study the molecular structure of myofilaments before discussing the detailed mechanism of muscle contraction.

First of all, four different kinds of protein molecules make up myofilaments: myosin, actin, tropomyosin, and troponin. The thin filaments are made of a combination of three proteins: actin, tropomyosin, and troponin. Figure 11-4, A, shows that globular actin molecules are strung together like beads to form two fibrous strands that twist around each other to form the bulk of each thin filament. Actin and myosin molecules have a chemical attraction for one another, but, at rest, the active sites on the actin molecules are covered by long tropomyosin molecules. The tropomyosin molecules seem to be held in this blocking position by troponin molecules spaced at intervals along the thin filament (see Figure 11-4, A).

As Figure 11-4, B, shows, the thick filaments are made almost entirely of myosin molecules. Notice that the myosin molecules are shaped like golf clubs, with their long shafts bundled together to form a thick filament and their “heads” sticking out from the bundle. The myosin heads are chemically attracted to the actin molecules of the nearby thin fila-

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**Figure 11-3** Unique features of the skeletal muscle cell. Notice especially the T tubules, which are extensions of the plasma membrane, or sarcolemma, and the sarcoplasmic reticulum (SR), which forms networks of tubular canals and sacs. A triad is a triplet of adjacent tubules: a terminal (end) sac of the SR, a T tubule, and another terminal sac of the SR.

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**Figure 11-4** Structure of myofilaments. A, Thin myofilament. B, Thick myofilament. C, Cross section of several thick and thin myofilaments, showing the relative positions of myofilaments and the myosin heads that will form cross bridges between them.
Box 11-2

Major Events of Muscle Contraction and Relaxation

Excitation and Contraction

1. A nerve impulse reaches the end of a motor neuron, triggering the release of the neurotransmitter acetylcholine.
2. Acetylcholine diffuses rapidly across the gap of the neuromuscular junction and binds to acetylcholine receptors on the motor endplate of the muscle fiber.
3. Stimulation of acetylcholine receptors initiates an impulse that travels along the sarcolemma, through the T tubules, to the sacs of the SR.
4. Ca\(^{2+}\) is released from the SR into the sarcoplasm, where it binds to troponin molecules in the thin myofilaments.
5. Tropomyosin molecules in the thin myofilaments shift, exposing actin's active sites.
6. Energized myosin cross bridges of the thick myofilaments bind to actin and use their energy to pull the thin myofilaments toward the center of each sarcomere. This cycle repeats itself many times per second, as long as adenosine triphosphate (ATP) is available.
7. As the filaments slide past the thick myofilaments, the entire muscle fiber shortens.

Relaxation

1. After the impulse is over, the SR begins actively pumping Ca\(^{2+}\) back into its sacs.
2. As Ca\(^{2+}\) is stripped from troponin molecules in the thin myofilaments, tropomyosin returns to its position, blocking actin's active sites.
3. Myosin cross bridges are prevented from binding to actin and thus can no longer sustain the contraction.
4. Because the thick and thin myofilaments are no longer connected, the muscle fiber may return to its longer, resting length.

Box 11-3 FYI

Rigor Mortis

The term rigor mortis is a Latin phrase that means “stiffness of death.” In a medical context the term rigor mortis refers to the stiffness of skeletal muscles sometimes observed shortly after death. What causes rigor mortis? At the time of death, stimulation of muscle cells ceases. However, muscle fibers of postural muscles may have been in mid-contraction at the time of death—when the myosin-actin cross bridges are still intact. Also, the SR releases much of the Ca\(^{2+}\) it had been storing, causing even more cross bridges to form. ATP is required to release the cross bridges and “energize” the myosin heads for their next attachment. Because the last of a cell’s ATP supply is used up at the time it dies, many cross bridges may be left “stuck” in the contracted position. Thus muscles in a dead body may be stiff because individual muscle fibers ran out of the ATP required to “turn off” a muscle contraction.

THE MECHANISM OF CONTRACTION

To accomplish the powerful shortening, or contraction, of a muscle fiber, several processes must be coordinated in a step-wise fashion. These steps are summarized in the following and in Box 11-2.

Excitation of the Sarcolemma

Under normal circumstances, a skeletal muscle fiber remains “at rest” until it is stimulated by a signal from a special type of nerve cell called a motor neuron. As Figure 11-5 shows, motor neurons connect to the sarcolemma of a muscle fiber at a folded motor endplate to form a junction called a neuromuscular junction. A neuromuscular junction is a type of connection called a synapse, characterized by a narrow gap, or synaptic cleft, across which neurotransmitter molecules transmit signals. When nerve impulses reach the end of a motor neuron fiber, small vesicles release a neurotransmitter, acetylcholine, into the synaptic cleft. Diffusing swiftly across this microscopic gap, acetylcholine molecules contact the sarcolemma of the adjacent muscle fiber. There they stimulate acetylcholine receptors and thereby initiate an
electrical impulse in the sarcolemma. The process of synaptic transmission and induction of an impulse—a process often called *excitation*—is discussed in detail in Chapter 12.

**Contraction**

The impulse, a temporary electrical imbalance, is conducted over the muscle fiber's sarcolemma and inward along the T tubules (Figure 11-6). The impulse in the T tubules triggers the release of a flood of calcium ions from the adjacent sacs of the SR. In the sarcoplasm, the calcium ions combine with troponin molecules in the thin filaments of the myofibrils. Recall that troponin normally holds tropomyosin strands in a position that blocks the chemically active sites of actin. When calcium binds to troponin, however, the tropomyosin shifts to expose active sites on the actin molecules (Figure 11-7). Once the active sites are exposed, energized myosin heads of the

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**Figure 11-6** Effects of excitation on a muscle fiber. Excitation of the sarcolemma by a nerve impulse initiates an impulse in the sarcolemma. The impulse travels across the sarcolemma and through the T tubules, where it triggers adjacent sacs of the SR to release a flood of calcium ions (Ca\(^{++}\)) into the sarcoplasm. The Ca\(^{++}\) are then free to bind to troponin molecules in the thin filaments. This binding, in turn, initiates the chemical reactions that produce a contraction.

**Figure 11-7** The molecular basis of muscle contraction. 
A, Each myosin head in the thick filament moves into a resting position after an ATP binds and transfers its energy. B, Calcium ions released from the SR bind to troponin in the thin filament, allowing tropomyosin to shift from its position blocking the active sites of actin molecules. C, Each myosin head then binds to an active site on a thin filament, displacing the remnants of ATP hydrolysis—adenosine diphosphate (ADP) and inorganic phosphate (Pi). D, The release of stored energy from step A provides the force needed for each head to move back to its original position, pulling actin along with it. Each head will remain bound to actin until another ATP binds to it and pulls it back into its resting position (A). E, This scanning electron micrograph of a thin filament shows the myosin-binding active sites on actin covered by tropomyosin (TM) when calcium is absent. In the presence of calcium, bottom, the tropomyosin has changed position, revealing the active sites on actin.
thick filaments bind to actin molecules in the nearby thin filaments. The myosin heads bend with great force, literally pulling the thin filaments past them. Each head then releases itself, binds to the next active site, and pulls again. Figure 11-8 shows how the sliding of the thin filaments toward the center of each sarcomere quickly shortens the entire myofibril—and thus the entire muscle fiber. This model of muscle contraction has been called the *sliding filament theory*.

**Relaxation**

Almost immediately after the SR releases its flood of calcium ions into the sarcoplasm, it begins actively pumping them back into its sacs once again. Within a few milliseconds, much of the calcium is recovered. Because the active transport carriers of the SR have a greater affinity to calcium than troponin molecules, the calcium ions are stripped off the troponin molecules and returned to the sacs of the SR. As you might suspect, this shuts down the entire process of contraction. Troponin without its bound calcium allows the tropomyosin to once again block actin’s active sites. Myosin heads reaching for the next active site on actin are blocked, and thus the thin filaments are no longer being held—or pulled—by the thick filaments. The muscle fiber may remain at its contracted length, but forces outside the muscle fiber are likely to pull it back to its longer resting length. In short, the contraction process in a skeletal muscle fiber automatically shuts itself off within a small fraction of a second after the initial stimulation.

**ENERGY SOURCES FOR MUSCLE CONTRACTION**

**ATP**

The energy required for muscular contraction is obtained by hydrolysis of a nucleotide called adenosine triphosphate, or ATP. Recall from Chapter 2 (Figure 2-26, p. 62) that this molecule has an adenine and ribose group (together called *adenosine*) attached to three phosphate groups. Two of the three phosphate groups in ATP are attached to the molecule by *high-energy bonds*. Breaking of these high-energy bonds provides the energy necessary to pull the thin myofilaments during muscle contraction. As Figure 11-7, A, shows, before contraction occurs, each myosin cross head moves into a resting position when an ATP molecule binds to it. The ATP molecule breaks its outermost high-energy bond, releasing the inorganic phosphate (Pi) and transferring the energy to the myosin head. In a way, this is like pulling back the elastic band of a sling-shot—the apparatus is “at rest” but ready to spring. When myosin binds to actin, the stored energy is released, and the myosin head does indeed spring back to its original position. Thus the energy transferred from ATP is used to do the work of pulling the thin filaments during contraction. Another ATP molecule then binds to the myosin head which then releases actin and moves into its resting position again—all set for the next “pull.” This cycle repeats, as long as ATP is available and actin’s active sites are unblocked.

Muscle fibers must continually resynthesize ATP because they can store only small amounts of it. Immediately after ATP breaks down, energy for its resynthesis can be supplied by the breakdown of another high-energy compound, creatine phosphate (CP), which is also present in small amounts in muscle fibers (Figure 11-9). Ultimately, energy for both ATP and CP synthesis comes from the catabolism of foods.
Aerobic Respiration

Aerobic respiration may allow the body to avoid the use of oxygen in the short term, but not in the long term. Anaerobic respiration results in the formation of an incompletely catabolized molecule called lactic acid. Lactic acid may accumulate in muscle tissue during exercise and cause a burning sensation. Some of the lactic acid eventually diffuses into the blood and is delivered to the liver, where an oxygen-consuming process later converts it back into glucose. This is one of the reasons that after heavy exercise, when the lack of oxygen in some tissues caused the production of lactic acid, a person may still continue to breathe heavily. The body is repaying the so-called oxygen debt by using the extra oxygen gained by heavy breathing to process the lactic acid that was produced during exercise.

Heat Production

Because the catabolic processes of cells are never 100% efficient, some of the energy released is lost as heat. Because skeletal muscle tissues produce such a massive amount of heat—even when they are doing hardly any work—they have a great effect on body temperature. Recall from Chapter 6 that various heat-loss mechanisms of the skin can be employed to cool the body when it becomes overheated (see Figure 6-7, p. 174). Skeletal muscle tissues can likewise be employed when the body’s temperature falls below the set point value determined by the “thermostat” in the hypothalamus of the brain. As Figure 11-10 shows, a low external temperature can reduce body temperature below the set point. Temperature sensors in the skin and other parts of the body feed this information back to the hypothalamus, which compares the actual value to the set point value (usually about 37° C). The hypothalamus responds to a decrease in body temperature by signaling skeletal muscles to contract. The shivering contractions that result produce enough waste heat to warm the body back to the set point temperature—and homeostatic balance is maintained.

The subject of energy metabolism is discussed more thoroughly in Chapter 27.

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**Figure 11-9**  Energy sources for muscle contraction. A, The basic structure of two high-energy molecules in the sarcoplasm: adenosine triphosphate (ATP) and creatine phosphate (CP). B, This diagram shows how energy released during the catabolism of nutrients can be transferred to the high-energy bonds of ATP directly, or instead stored temporarily in the high-energy bond of the CP. During contraction, ATP is hydrolyzed and the energy of the broken bond transferred to a myosin head.

**Glucose and Oxygen**

Note that continued, efficient nutrient catabolism by muscle fibers requires two essential ingredients: glucose and oxygen. Glucose is a nutrient molecule that contains many chemical bonds. The potential energy stored in these chemical bonds is released during catabolic reactions in the sarcoplasm and mitochondria and transferred to ATP or CP molecules. Some muscle fibers ensure an uninterrupted supply of glucose by storing it in the form of glycogen. Oxygen, which is needed for a catabolic process known as aerobic respiration, can also be stored by cells. During rest, excess oxygen molecules in the sarcoplasm are bound to a large protein molecule called myoglobin. Myoglobin is a reddish pigment similar to the pigment hemoglobin that gives blood its red color. Like hemoglobin, myoglobin contains iron (Fe) groups that attract oxygen molecules and hold them temporarily. When the oxygen concentration inside a muscle fiber decreases rapidly—as it does during exercise—it can be quickly resupplied from the myoglobin. Muscle fibers that contain large amounts of myoglobin take on a deep red appearance and are often called red fibers. Muscle fibers with little myoglobin in them are light pink and are often called white fibers. Most muscle tissues contain a mixture of red and white fibers (Box 11-4).

**Aerobic Respiration**

Aerobic (oxygen-requiring) respiration is a catabolic process that produces the maximum amount of energy available from each glucose molecule. When oxygen concentration is low, however, muscle fibers can shift toward an increased use of another catabolic process: anaerobic respiration. As its name implies, anaerobic respiration does not require the immediate use of oxygen. Besides its ability to produce ATP without oxygen, anaerobic respiration has the added advantage of being very rapid. Muscle fibers having difficulty getting oxygen—or fibers that generate a great deal of force very quickly—may rely on anaerobic respiration to resynthesize their ATP molecules.

**Anaerobic Respiration**

Anaerobic respiration may allow the body to avoid the use of oxygen in the short term, but not in the long term. Anaerobic respiration results in the formation of an incompletely catabolized molecule called lactic acid. Lactic acid may accumulate in muscle tissue during exercise and cause a burning sensation. Some of the lactic acid eventually diffuses into the blood and is delivered to the liver, where an oxygen-consuming process later converts it back into glucose. This is one of the reasons that after heavy exercise, when the lack of oxygen in some tissues caused the production of lactic acid, a person may still continue to breathe heavily. The body is repaying the so-called oxygen debt by using the extra oxygen gained by heavy breathing to process the lactic acid that was produced during exercise.
Types of Muscle Fibers

Skeletal muscle fibers can be classified into three types according to their structural and functional characteristics: (1) slow (red) fibers, (2) fast (white) fibers, and (3) intermediate fibers. Each type is best suited to a particular type or style of muscular contraction. Although each muscle organ contains a mix of all three fiber types, different organs have these fibers in different proportions, depending on the types of contraction that they most often perform.

**Slow fibers** are also called red fibers because they contain a high concentration of myoglobin, the reddish pigment used by muscle cells to store oxygen. They are called slow fibers because their thick myofilaments are made of a type of myosin (Type I) that reacts at a slow rate. Because they contract so slowly, slow fibers are usually able to produce ATP quickly enough to keep pace with the energy needs of the myosin and thus avoid fatigue. This effect is enhanced by a larger number of mitochondria than other fiber types and the rich oxygen store provided by the myoglobin. The slow, nonfatiguing characteristics of slow fibers make them especially well suited to the sustained contractions exhibited by postural muscles. Postural muscles containing a high proportion of slow fibers can hold the skeleton upright for long periods without fatigue.

**Fast fibers** are also called white fibers because they contain very little myoglobin. Fast fibers can contract much more rapidly than slow fibers because they have a faster type of myosin (Type IIx) and because their system of T tubules and SR is more efficient at quickly delivering Ca\(^{++}\) to the sarcoplasm. The price of a rapid contraction mechanism is the rapid depletion of ATP. Despite the fact that fast fibers typically contain a high concentration of glycogen, they have few mitochondria and so must rely primarily on anaerobic respiration to regenerate ATP. Because anaerobic respiration produces relatively small amounts of ATP, fast fibers cannot produce enough ATP to sustain a contraction for very long. Because they can generate great force very quickly but not for a long duration, fast fibers are best suited for muscles that move the fingers and eyes in darting motions.

**Intermediate fibers** have characteristics somewhere in between the two extremes of fast and slow fibers. They are more fatigue resistant than fast fibers and can generate more force more quickly than slow fibers. This type of muscle fiber predominates in muscles that both provide postural support and are occasionally required to generate rapid, powerful contractions. One example is the soleus, or calf muscle, that helps to support the leg but is also used in walking, running, and jumping.

The graph shows that the relative proportions of muscle fiber types, body wide, varies with the type of work a person does with his or her muscles. A person with a spinal cord injury eventually loses nearly all of the postural slow fibers while retaining mostly intermediate and fast fibers. An extreme endurance athlete, on the other hand, develops the slow fibers so much as to greatly dominate the faster fiber types.
Figure 11-10 The role of skeletal muscle tissues in maintaining a constant body temperature. This diagram shows that a drop in body temperature caused by cold weather can be corrected by a negative feedback mechanism that triggers shivering (muscle contraction), which in turn produces enough heat to warm the body.
FUNCTION OF SKELETAL MUSCLE ORGANS

Although each skeletal muscle fiber is distinct from all other fibers, it operates as part of the large group of fibers that form a skeletal muscle organ. Skeletal muscle organs, often simply called muscles, are composed of bundle upon bundle of muscle fibers held together by fibrous connective tissues (see Figure 11-1, A). The details of muscle organ anatomy are discussed in Chapter 10. For now, we turn attention to the matter of how skeletal muscle organs function as a single unit.

MOTOR UNIT

Recall that each muscle fiber receives its stimulus from a motor neuron. This neuron, often called a somatic motor neuron, is one of several nerve cells that enter a muscle organ together in a bundle called a motor nerve. One of these motor neurons, plus the muscle fibers to which it attaches, constitutes a functional unit called a motor unit (Figure 11-11). The single fiber of a somatic motor neuron divides into a variable number of branches on entering the skeletal muscle. The neuron branches of some motor units terminate in only a few muscle fibers, whereas others terminate in numerous fibers. Consequently, impulse conduction by one motor unit may stimulate only a half dozen or so muscle fibers to contract at one time, whereas conduction by another motor unit may activate a hundred or more fibers simultaneously. This fact bears a relationship to the function of the muscle as a whole. As a rule, the fewer the number of fibers supplied by a skeletal muscle's individual motor units, the more precise the movements that muscle can produce. For example, in certain small muscles of the hand, each motor unit includes only a few muscle fibers, and these muscles produce precise finger movements. In contrast, motor units in large abdominal muscles that do not produce precise movements are reported to include more than a hundred muscle fibers each.

MYOGRAPHY

Many experimental methods have been used to study the contractions of skeletal muscle organs. They vary from relatively simple procedures, such as observing or palpating muscles in action, to the more complicated method of electromyography (recording electrical impulses from muscles as they contract). One method of studying muscle contraction particularly useful for the purposes of our discussion is called, simply, myography. Myography, a term that means “muscle graphing,” is a procedure in which the force or tension from the contraction of an isolated muscle is recorded as a line that rises and falls as the muscle contracts and relaxes. To get the muscle to contract, an electrical stimulus of sufficient intensity (the threshold stimulus) is applied to the muscle. A single, brief threshold stimulus produces a quick jerk of the muscle, called a twitch contraction.

THE TWITCH CONTRACTION

The quick, jerky twitch contraction seen in a myogram serves as the fundamental model for how muscles operate. The myogram of a twitch contraction shown in Figure 11-12 shows that the muscle does not begin to contract at the instant of stimulation but rather a fraction of a second later. The muscle then increases its tension (or shortens) until a peak is reached, after which it gradually returns to its resting state. These three phases of the twitch contraction are called, respectively, the latent period, the contraction phase, and the relaxation phase. The entire twitch usually lasts less than one tenth of a second.

Figure 11-11  Motor unit. A motor unit consists of one somatic motor neuron and the muscle fibers supplied by its branches.
During the latent period, the impulse initiated by the stimulation travels through the sarcolemma and T tubules to the SR, where it triggers the release of calcium ions into the sarcoplasm. It is not until the calcium binds to troponin and the sliding of the myofilaments begins that contraction is observed. After a few milliseconds, the forceful sliding of the myofilaments ceases and relaxation begins. By the end of the relaxation phase, all of the myosin-actin reactions in all the fibers have ceased.

Twitch contractions of muscle organs rarely happen in the body. Even if we tried to make our muscles twitch voluntarily, they won’t. Instead, our nervous system subconsciously “smoothes out” the movements to prevent injury and to make our movements more useful to us. In other words, motor units are each controlled by separate somatic motor neurons that normally do not all “fire” at the same time. Only when an electrical stimulus is applied, or when overactivity of the nervous system stimulates most of the motor neurons in a muscle, do such contractions occur. However, knowledge of the twitch contraction gives us important insights about the mechanisms of more typical types of muscle organ contractions.

**TREPPE: THE STAIRCASE PHENOMENON**

One interesting effect that can be seen in myographic studies of the twitch contraction is called *treppe*, or the *staircase phenomenon*. Treppe is a gradual, steplike increase in the strength of contraction that can be observed in a series of twitch contractions that occur about 1 second apart (Figure 11-13, B).

In other words, a muscle contracts more forcefully after it has contracted a few times than when it first contracts—a principle used by athletes when they warm up. There are several factors that contribute to this phenomenon. For example, in warm muscle fibers calcium ions diffuse through the sarcoplasm more efficiently and more actin-myosin reactions occur. Also, calcium ions accumulate in the sarcoplasm of muscles that have not had time to relax and pump much of the calcium back into their SR. Thus up to a point, a warm fiber contracts more strongly than a cool fiber. Thus, after the first few stimuli muscle responds to successive stimuli with maximal contractions. Eventually, it will respond with less and less strong contractions. The relaxation phase becomes shorter and finally disappears entirely. In other words, the muscle stays partially contracted—an abnormal state of prolonged contraction called *contracture*.

Repeated stimulation of muscle in time lessens its excitability and contractility and may result in *muscle fatigue*, a condition in which the muscle does not respond to the strongest stimuli. Complete muscle fatigue can be readily induced in an isolated muscle but very seldom occurs in the body. (See Box 11-5.)

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**Figure 11-12**  The twitch contraction. Three distinct phases are apparent: (1) the latent period, (2) the contraction phase, and (3) the relaxation phase.

**Figure 11-13**  Myograms of various types of muscle contractions. A, A single twitch contraction. B, The treppe phenomenon, or “staircase effect,” is a steplike increase in the force of contraction over the first few in a series of twitches. C, Incomplete tetanus occurs when a rapid succession of stimuli produces “twitches” that seem to add together (wave summation) to produce a rather sustained contraction. D, Complete tetanus is a smoother sustained contraction, produced by the summation of “twitches” that occur so close together that the muscle cannot relax at all.
MUSCLE TONE

A tonic contraction (tonus, “tone”) is a continual, partial contraction in a muscle organ. At any one moment a small number of the total fibers in a muscle contract, producing a tautness of the muscle rather than a recognizable contraction and movement. Different groups of fibers scattered throughout the muscle contract in relays. Tonic contraction, or muscle tone, is the low level of continuous contraction characteristic of the muscles of normal individuals when they are awake. It is particularly important for maintaining posture. A striking illustration of this fact is the following: when a person loses consciousness, muscles lose their tone, and the person collapses in a heap, unable to maintain a sitting or standing posture. Muscles with less tone than normal are described as flaccid, and those with more than normal tone are called spastic.

Muscle tone is maintained by negative feedback mechanisms centered in the nervous system, specifically in the spinal cord. Stretch sensors in the muscles and tendons detect the degree of stretch in a muscle organ and feed this information back to an integrator mechanism in the spinal cord. When the actual stretch (detected by the stretch receptors) deviates from the set point stretch, signals sent via the somatic motor neurons adjust the strength of tonic contraction. This type of subconscious mechanism is often called a spinal reflex (discussed further in Chapters 12 to 15).

THE GRADED STRENGTH PRINCIPLE

Skeletal muscles contract with varying degrees of strength at different times—a fact called the graded strength principle. Because muscle organs can generate different grades of strength, we can match the force of a movement to the demands of a specific task (Box 11-6).

Various factors contribute to the phenomenon of graded strength. We have already discussed some of these factors. For example, we stated that the metabolic condition of individual fibers influences their capacity to generate force. Thus if many fibers of a muscle organ are unable to maintain a high level of ATP and become fatigued, the entire muscle organ suffers some loss in its ability to generate maximum force of contraction. On the other hand, the improved metabolic conditions that produce the Treppe effect allow a muscle organ to increase its contraction strength.

Another factor that influences the grade of strength exhibited by a muscle organ is the number of fibers contracting simultaneously. Obviously, the more muscle fibers contracting at the same time, the stronger the contraction of the entire muscle organ. How large this number is depends on how many motor units are activated or recruited. Recruitment of motor units, in turn, depends on the intensity and frequency of stimulation. In general, the more intense and the more frequent a stimulus, the more motor units are recruited and the stronger the contraction. Figure 11-14 shows that increasing the strength of the stimulus beyond the threshold level of the most sensitive motor units causes an increase in strength of contraction. As the threshold level of...
Effects of Exercise on Skeletal Muscles

Most of us believe that exercise is good for us, even if we have no idea what or how many specific benefits can come from it. Some of the good consequences of regular, properly practiced exercise are greatly improved muscle tone, better posture, more efficient heart and lung function, less fatigue, and looking and feeling better.

Skeletal muscles undergo changes that correspond to the amount of work that they normally do. During prolonged inactivity, muscles usually shrink in mass, a condition called disuse atrophy. Exercise, on the other hand, may cause an increase in muscle size called hypertrophy.

Muscle hypertrophy can be enhanced by strength training, which involves contracting muscles against heavy resistance. Isometric exercises and weight lifting are common strength-training activities. This type of training results in increased numbers of myofilaments in each muscle fiber. Although the number of muscle fibers stays the same, the increased number of myofilaments greatly increases the mass of the muscle.

Endurance training, often called aerobic training, does not usually result in muscle hypertrophy. Instead, this type of exercise program increases a muscle’s ability to sustain moderate exercise over a long period. Aerobic activities such as running, bicycling, or other primarily isotonic movements increase the number of blood vessels in a muscle without significantly increasing its size. The increased blood flow allows a more efficient delivery of oxygen and glucose to muscle fibers during exercise. Aerobic training also causes an increase in the number of mitochondria in muscle fibers. This allows production of more ATP as a rapid energy source.

The maximal strength that a muscle can develop is directly related to the initial length of its fibers—this is the length-tension relationship (Figure 11-15). A muscle that begins a contraction from a short initial length cannot develop much tension because its sarcomeres are already compressed. Conversely, a muscle that begins a contraction from an overstretched initial length cannot develop much tension because the thick myofilaments are too far away from the thin myofilaments to effectively pull them and thus compress the sarcomeres. The strongest maximal contraction is possible only when the muscle organ has been stretched to an optimal initial length. To illustrate this point, extend your elbow fully and try to contract the biceps brachii muscle on the ventral side of your upper arm. Now flex the elbow just a little and contract the biceps again. Try it a third time with the elbow completely flexed. The greatest tension—seen as the largest “bulge” of the biceps—occurs when the elbow is partly flexed and the biceps only moderately stretched.

Another factor that influences the strength of a skeletal muscle contraction is the amount of load imposed on the muscle. Within certain limits, the heavier the load, the stronger the contraction. Lift your hand with palm up in front of you and then put this book in your palm. You can feel your arm muscles contract more strongly as the book is placed in your hand. This occurs because of a stretch reflex, a response in which the body tries to maintain a constancy of muscle length (Figure 11-16). An increased load threatens to stretch the muscle beyond the set point length that you are trying to maintain. Your body exhibits a negative feedback response when it detects the increased stretch caused by an increased load, feeds the information back to an integrator in the nervous system, and increases its stimulation of the muscle to counteract the stretch. This reflex maintains a relatively constant muscle length as load is increased up to a maximum sustainable level. When the load becomes too heavy and thus threatens to cause injury to the muscle or skeleton, the body abandons this reflex and forces you to relax and drop the load.

The major factors involved in the graded strength principle are summarized in Figure 11-17.

ISOTONIC AND ISOMETRIC CONTRACTIONS

The term isotonic literally means “same tension” (iso-, “equal”; tonic, “tension”). An isotonic contraction is a contraction in which the tone or tension within a muscle remains the same as
the length of the muscle changes (Figure 11-18, A). Because the muscle is moving against its resistance (load) in an isotonic contraction, the energy of contraction is used to pull on the thin myofilaments and thus change the length of a fiber’s sarcomeres. Put another way, in isotonic contractions the myosin cross bridges “win” the tug-of-war against a light load and are thus able to pull the thin myofilaments. Because the muscle is moving in an isotonic contraction, it is also called dynamic tension.

There are two basic varieties of isotonic contractions (Figure 11-18, A). Concentric contractions are those in which the movement results in shortening of the muscle, as when you pick up this book. Eccentric contractions are those in which the movement results in lengthening of the muscle being contracted. For example, when you slowly lower the book you have just picked up, you are contracting the same muscle you just used to lift it—but this time you are lengthening the muscle, not shortening it.

An isometric contraction, in contrast to the isotonic contraction, is a contraction in which muscle length remains the same while the muscle tension increases (Figure 11-18, B). The term isometric literally means “same length.” You can observe isometric contraction by lifting up on a stationary handrail and feeling the tension increase in your arm muscles. Isometric contractions can do work by “tightening” to resist a force, but they do not produce movements. In isometric contractions, the tension produced by the “power stroke” of the myosin cross bridges cannot overcome the load placed on the muscle. Using the tug-of-war analogy, we can say that in isometric contractions the myosin cross

**Figure 11-15** The length-tension relationship. As this graph of muscle tension shows, the maximum strength that a muscle can develop is directly related to the initial length of its fibers. At a short initial length the sarcomeres are already compressed and thus the muscle cannot develop much tension (position A). Conversely, the thick and thin myofilaments are too far apart in an overstretched muscle to generate much tension (position B). Maximum tension can be generated only when the muscle has been stretched to a moderate, optimal length (position C).

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**Box 11-7 HEALTH MATTERS**

**Abnormal Muscle Contractions**

**Cramps** are painful muscle spasms (involuntary twitches). Cramps often occur when a muscle organ is mildly inflamed, but they can be a symptom of any irritation or ion and water imbalance.

**Convulsions** are abnormal, uncoordinated tetanic contractions of varying groups of muscles. Convulsions may result from a disturbance in the brain or seizure in which the output along motor nerves increases and becomes disorganized.

**Fibrillation** is an abnormal type of contraction in which individual fibers contract asynchronously rather than at the same time. This produces a flutter of the muscle but no effective movement. Fibrillation can also occur in cardiac muscle, where it reduces the heart’s ability to pump blood.
Figure 11-16  The stretch reflex. The strength of a muscle organ can be matched to the load imposed on it by a negative feedback response centered in the spinal cord. Increased stretch (caused by increased load) is detected by a sensory nerve fiber attached to a muscle cell (called a muscle spindle) specialized for this purpose. The information is integrated in the spinal cord and a correction signal is relayed through motor neurons back to the same muscle, which increased tension to return to the set point muscle length.

Figure 11-17  Factors that influence the strength of muscle contraction.
bridges reach a “draw”—they hold their own against the load placed on the muscle but do not make any progress in sliding the thin myofilaments. Because muscles remain stable during isotonic contraction, it is also called static tension.

**FUNCTION OF CARDIAC AND SMOOTH MUSCLE TISSUE**

Cardiac and smooth muscle tissues operate by mechanisms similar to those in skeletal muscle tissues. The detailed study of cardiac and smooth muscle function will be set aside until we discuss specific smooth and cardiac muscle organs in later chapters. However, it may be helpful to preview some of the basic principles of cardiac and smooth muscle physiology so that we can compare them with those that operate in skeletal muscle tissue. Table 11-1 summarizes the characteristics of the three major types of muscle.

**CARDIAC MUSCLE**

Cardiac muscle, also known as striated involuntary muscle, is found in only one organ of the body: the heart. Forming the
bulk of the wall of each heart chamber, cardiac muscle contracts rhythmically and continuously to provide the pumping action necessary to maintain a relative constancy of blood flow through the internal environment. As you shall see, its physiological mechanisms are well adapted to this function.

The functional anatomy of cardiac muscle tissue resembles that of skeletal muscle to a degree but exhibits specialized features related to its role in continuously pumping blood. As Figure 11-19 shows, each cardiac muscle fiber contains parallel myofibrils. Each myofibril comprises sarcomere, and the fibers themselves are interconnected by T tubules, sarcoplasmic reticulum, and intercalated disks.

### Table 11-1 Characteristics of Muscle Tissues

<table>
<thead>
<tr>
<th>Skeletal</th>
<th>Cardiac</th>
<th>Smooth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Principal location</td>
<td>Skeletal muscle organs</td>
<td>Walls of many hollow organs</td>
</tr>
<tr>
<td>Principal functions</td>
<td>Movement of bones, heat production, posture</td>
<td>Movement in walls of hollow organs (peristalsis, mixing)</td>
</tr>
<tr>
<td>Type of control</td>
<td>Voluntary</td>
<td>Involuntary</td>
</tr>
<tr>
<td>Structural features</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Striations</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Nucleus</td>
<td>Many near sarcolemma</td>
<td>Single</td>
</tr>
<tr>
<td>T tubules</td>
<td>Narrow; form triads with SR</td>
<td>Large diameter; form diads with SR, regulate Ca$^{2+}$ entry into sarcoplasm</td>
</tr>
<tr>
<td>Sarcomplasmic reticulum</td>
<td>Extensive; stores and releases Ca$^{2+}$</td>
<td>Less extensive than in skeletal muscle</td>
</tr>
<tr>
<td>Cell junctions</td>
<td>No gap junctions</td>
<td>Intercalated disks</td>
</tr>
<tr>
<td>Contraction style</td>
<td>Rapid twitch contractions of motor units usually summate to produce sustained titanic contractions; must be stimulated by a neuron</td>
<td>Syncytium of fibers compress heart chambers in slow, separate contractions (does not exhibit tetanus or fatigue); exhibits autorhythmicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Very poorly developed</td>
</tr>
</tbody>
</table>

The functional anatomy of cardiac muscle tissue resembles that of skeletal muscle to a degree but exhibits specialized features related to its role in continuously pumping blood. As Figure 11-19 shows, each cardiac muscle fiber contains parallel myofibrils. Each myofibril comprises sarcomeres, and the fibers themselves are interconnected by T tubules, sarcoplasmic reticulum, and intercalated disks.
eres that give the whole fiber a striated appearance. However, the cardiac muscle fiber does not taper like a skeletal muscle fiber but, instead, forms strong, electrically coupled junctions (intercalated disks) with other fibers. This feature, along with the branching exhibited by individual cells, allows cardiac fibers to form a continuous, electrically coupled mass, called a syncytium (meaning “unit of combined cells”). Cardiac muscles thus form a continuous, contractile band around the heart chambers that conducts a single impulse across a virtually continuous sarcolemma—features necessary for an efficient, coordinated pumping action.

Unlike skeletal muscle, in which a nervous impulse excites the sarcolemma to produce its own impulse, cardiac muscle is self-exciting. Cardiac muscle cells thus exhibit a continuing rhythm of excitation and contraction on their own, although the rate of self-induced impulses can be altered by nervous or hormonal input. Figure 11-20 shows that impulses triggering cardiac muscle contractions are much more prolonged than the nerve impulses that trigger skeletal. Because the sarcolemma of the cardiac muscle sustains each impulse longer than in skeletal muscle, Ca\(^{2+}\) remains in the sarcoplasm longer. This means that even though many adjacent cardiac muscle cells contract simultaneously, they exhibit a prolonged contraction rather than a rapid twitch. It also means that impulses cannot come rapidly enough to produce tetanus. Because it cannot sustain long tetanic contractions, cardiac muscle does not normally run low on ATP and thus does not experience fatigue. Obviously, this characteristic of cardiac muscle is vital to keeping the heart continuously pumping.

Although the cardiac muscle fiber has T tubules and SR, they are arranged a little differently than in skeletal muscle fibers. The T tubules are larger, and they form diads (double structures) rather than triads (triple structures), with a rather sparse SR. Much of the calcium (Ca\(^{2+}\)) that enters the sarcoplasm during contraction enters from the outside of the cells through the T tubules, rather than from storage in the SR.

The structure and function of the heart are discussed further in Chapters 18 and 19.

**SMOOTH MUSCLE**

As we mentioned in Chapter 5, smooth muscle is composed of small, tapered cells with single nuclei. Smooth muscle cells do not have T tubules and have only loosely organized sarcoplasmic reticula. The calcium required for contraction comes from outside the cell and binds to a protein called calmodulin, rather than to troponin, to trigger a contraction event.

The lack of striations in smooth muscle fibers results from the fact that the thick and thin myofilaments are arranged quite differently than in skeletal or cardiac muscle fibers. As Figure 11-21 shows, thin arrangements of myofilaments crisscross the cell and attach at their ends to the cell’s plasma membrane. When cross bridges pull the thin filaments together, the muscle “balls up” and thus contracts the cell. Because the myofilaments are not organized into sarcomeres, they have more freedom of movement and thus can contract a smooth muscle fiber to shorter lengths than in skeletal and cardiac muscle.

There are two types of smooth muscle tissue: visceral and multiunit. In visceral, or single-unit, muscle, gap junctions join individual smooth muscle fibers into large, continuous sheets—much like the syncytium of fibers observed in cardiac muscle. This is the most common type of smooth muscle, forming a muscular layer in the walls of many hollow structures such as the digestive, urinary, and reproductive tracts. Like cardiac muscle, this type of smooth muscle commonly exhibits a rhythmic self-excitation or autorhythmicity (meaning “self-rhythm”) that spreads across the entire tissue. When these rhythmic, spreading waves of contraction become strong enough, they can push the contents of a hollow organ progressively along its lumen. This phenomenon, called peristalsis, moves food along the digestive tract, assists the flow of urine to the bladder, and pushes a baby out of the womb during labor. Such contractions can also be coordinated to produce mixing movements in the stomach and other organs.

Multiunit smooth muscle tissue does not act as a single unit (as in visceral muscle) but instead is composed of many independent single-cell units. Each independent fiber does not usually generate its own impulse but rather responds only to nervous input. Although this type of smooth muscle can form thin sheets, as in the walls of large blood vessels, it
is more often found in bundles (for example, the arrector pili muscles of the skin) or as single fibers (such as those surrounding small blood vessels).

The structure and function of smooth muscle organs are discussed in later chapters.

Figure 11-21  Smooth muscle fiber. A, Thin bundles of myofilaments span the diameter of a relaxed fiber. The scanning electron micrograph (right) shows that the surface of the cell is rather flat when the fiber is relaxed. B, During contraction, sliding of the myofilaments causes the fiber to shorten by 'balling up.' The micrograph shows that the fiber becomes shorter and thicker, exhibiting 'dimples' where the myofilament bundles are pulling on the plasma membrane.

1. How do slow, separate, autorhythmic contractions of cardiac muscle make it well suited to its role in pumping blood?
2. What produces the striations in cardiac muscle?
3. How are myofilaments arranged in a smooth muscle fiber?

THE BIG PICTURE

Muscle Tissue and the Whole Body

The function of all three major types of muscle (skeletal, smooth, and cardiac) is integral to the function of the entire body. What does the function of muscle tissue contribute to the homeostasis of the whole body? First, all three types of muscle tissue provide the movement necessary for survival. Skeletal muscle moves the skeleton so that we can seek shelter, gather food, and defend ourselves. All three muscle types produce movements that power vital homeostatic mechanisms such as breathing, blood flow, digestion, and urine flow.

The relative constancy of the body’s internal temperature could not be maintained in a cool external environment if not for the “waste” heat generated by muscle tissue—especially the large mass of skeletal muscle found throughout the body. Maintenance of a relatively stable body position—posture—is also a primary function of the skeletal muscular system. Posture, specific body movements, and other contributions of the skeletal muscular system to the homeostasis of the whole body was discussed in Chapter 10. The homeostatic roles of smooth muscle organs and the cardiac muscle organ (the heart) are examined in later chapters.

Like all tissues of the body, muscle tissue gives and takes. A number of systems support the function of muscle tissues. Without these systems, muscle would cease to operate. For example, the nervous system directly controls the contraction of skeletal muscle and multiunit smooth muscle. It also influences the rate of rhythmic contractions in cardiac muscle and visceral smooth muscle. The endocrine system produces hormones that assist the nervous system in regulation of muscle contraction throughout the body. The blood delivers nutrients and carries away waste products. Nutrients for the muscle are ultimately procured by the respiratory system (oxygen) and digestive system (glucose and other foods). The respiratory system also helps get rid of the waste of muscle metabolism, as does the urinary system. The liver processes lactic acid produced by muscles and converts it back to glucose. The immune system helps defend muscle tissue against infection and cancer—as it does for all body tissues. The fibers that comprise muscle tissues, then, are truly members of the large, interactive ‘society of cells’ that forms the human body.
Major Muscular Disorders

As you might expect, muscle disorders, or myopathies, generally disrupt the normal movement of the body. In mild cases, these disorders vary from inconvenient to slightly troublesome. Severe muscle disorders, however, can impair the muscles used in breathing—a life-threatening situation.

Muscle Injury

Injuries to skeletal muscles resulting from overexertion or trauma usually result in a muscle strain. Figure 11-22 shows an unusually severe muscle strain that resulted in a massive tear to the entire muscle organ. Muscle strains are characterized by muscle pain, or myalgia (my-AL-je-ah), and involve overstretching or tearing of muscle fibers. If an injury occurs in the area of a joint and a ligament is damaged, the injury may be called a sprain. Any muscle inflammation, including that caused by a muscle strain, is termed myositis (my-O-SYE-tis). If tendon inflammation occurs with myositis, as in a charley horse, the condition is termed fibromyositis (fi-bro-my-O-SYE-tis). Although inflammation may subside in a few hours or days, it usually takes weeks for damaged muscle fibers to repair. Some damaged muscle cells may be replaced by fibrous tissue, forming scars. Occasionally, hard calcium is deposited in the scar tissue.

Cramps are painful muscle spasms (involuntary twitches). Cramps often result from mild myositis or fibromyositis, but they can be a symptom of any irritation or of an ion and water imbalance.

Minor trauma to the body, especially a limb, may cause a muscle bruise, or contusion. Muscle contusions involve local internal bleeding and inflammation. Severe trauma to a skeletal muscle may cause a crush injury. Crush injuries greatly damage the affected muscle tissue, and the release of muscle fiber contents into the bloodstream can be life threatening. For example, the reddish muscle pigment myoglobin can accumulate in the blood and cause kidney failure.

Stress-induced muscle tension can result in myalgia and stiffness in the neck and back and is thought to be one cause of “stress headaches.” Headache and back-pain clinics use various strategies to treat stress-induced muscle tension. These treatments include massage, biofeedback, and relaxation training.

Muscle Infections

Several bacteria, viruses, and parasites may infect muscle tissue—often producing local or widespread myositis. For example, in trichinosis, widespread myositis is common. The muscle pain and stiffness that sometimes accompany influenza is another example.

Once a tragically common disease, poliomyelitis is a viral infection of the nerves that control skeletal muscle movement. Although the disease can be asymptomatic, it often causes paralysis that may progress to death. Virtually eliminated in the United States as a result of a comprehensive vaccination program, it still affects millions in other parts of the world.

Muscular Dystrophy

Muscular dystrophy (DIS-tro-fee) is not a single disorder but a group of genetic diseases characterized by atrophy (wasting) of skeletal muscle tissues. Some, but not all, forms of muscular dystrophy can be fatal.

The common form of muscular dystrophy is Duchenne (doo-SHEN) muscular dystrophy (DMD). This form of the disease is also called pseudohypertrophy (meaning “false muscle growth”) because the atrophy of muscle is masked by excessive replacement of muscle by fat and fibrous tissue. DMD is characterized by mild leg muscle weakness that progresses rapidly to include the shoulder muscles. The first signs of DMD are apparent at about 3 years of age, and the stricken child is usually severely affected within 5 to 10 years. Death from respiratory or cardiac muscle weakness often occurs by the time the individual is 21 years old.

We now know that DMD is caused by a mutation in X chromosome, although other factors may be involved. DMD occurs primarily in boys. Because girls have two X chromosomes and boys only one, genetic diseases involving X chromosome abnormalities are more likely to occur in boys. This is true because girls with one damaged X chromosome may not exhibit an “X-linked” disease if their other X chromosome is normal (see Chapter 34). The gene involved in DMD normally codes for the protein dystrophin (DIS-trof-in), which forms strands in each skeletal muscle fiber and helps to hold the cytoskeleton to the sarcolemma. Dystrophin thus helps to keep the muscle fiber from breaking during contractions. Normal dystrophin is missing in DMD because a deletion or mutation of part of the dystrophin gene (the largest human gene ever discovered) causes the resulting
protein to be nonfunctional (it has the wrong shape to do the job). Thus in DMD muscle fibers break apart more easily—causing the symptoms of progressive muscle weakness.

**Myasthenia Gravis**

*Myasthenia gravis* (my-es-THEE-nee-ah GRA-vis) is a chronic disease characterized by muscle weakness, especially in the face and throat. Most forms of this disease begin with mild weakness and chronic muscle fatigue in the face, then progress to wider muscle involvement. When severe muscle weakness causes immobility in all four limbs, a *myasthenic crisis* is said to have occurred. A person in myasthenic crisis is in danger of dying from respiratory failure because of weakness in the respiratory muscles.

Myasthenia gravis is an autoimmune disease in which the immune system attacks muscle cells at the neuromuscular junction. Nerve impulses from motor neurons are then unable to fully stimulate the affected muscle.

**Hernias**

Weakness of abdominal muscles can lead to a *hernia*, or protrusion, of an abdominal organ (commonly the small intestine) through an opening in the abdominal wall. There are several types of hernias. The most common one, *inguinal hernia* (Figure 11-23), occurs when the hernia extends down the inguinal canal, often into the scrotum or labia. Males experience this most often, and it can occur at any age. Women may experience a *femoral hernia* below the groin because of changes during pregnancy.

Hernia is referred to as “reducible” when the protruding organ is manipulated back into the abdominal cavity, either naturally by lying down or by manual reduction through a surgical opening in the abdomen. A “strangled” hernia occurs when the mass is not reducible and blood flow to the affected organ (i.e., intestine) is stopped. Obstruction and gangrene can occur. Pain and vomiting are usually experienced and emergency surgical intervention is required.

![Inguinal hernia.](image)

*Figure 11-23* Inguinal hernia in infant male.
CASE STUDY

Cecelia Pulaski, age 27, noticed changes in her energy level accompanied with muscle weakness. Particularly when she swallowed, she would sometimes feel that food was stuck in her throat. She had difficulty combing her hair, and she noticed that her voice was very weak. The weakness would usually improve when she rested. She was admitted to the hospital for myalgia, paresthesia, and immobility of all extremities. At the time of admission, she was having difficulty breathing. She recently experienced an extremely stressful divorce.

On physical examination, Ms. Pulaski is unable to close her eyes completely. Her pupils respond normally to light and show normal accommodation. She has lost 15 pounds in the last month. Her tongue has several fissures. Her laboratory data are essentially normal except for a positive antibody test, which is indicative of an autoimmune disorder attacking muscle cells at the neuromuscular junction. Electrical testing of the neuromuscular junction shows some blocking of discharges. A pharmacological test using edrophonium chloride is positive. Edrophonium chloride inhibits the breakdown of acetylcholine at the postsynaptic membrane.

1. Based on what is known about myasthenia gravis, which of the following explanations for Cecelia’s symptoms would be physiologically correct?
   A. Adenosine triphosphate pulls the thin myofilaments during muscle contraction.
   B. Active sites on the actin molecules are exposed.
   C. A flood of calcium ions combines with troponin molecules in the thin filament myofibrils.
   D. Nerve impulses from motor neuromuscular junctions are unable to fully stimulate the affected muscle.

2. Based on the action of edrophonium chloride, as stated above, how will this drug work in Ms. Pulaski’s case?
   Edrophonium chloride:
   A. Increases the availability of acetylcholine at the postsynaptic receptor sites
   B. Decreases the availability of acetylcholine at the postsynaptic receptor sites
   C. Increases the attachment of thick myosin filaments to the sarcomere
   D. Decreases electrical impulses in the sarcolemma

3. Based on the action of edrophonium chloride, as stated above, which one of the following physical effects will most likely be noted by Ms. Pulaski?
   A. Relaxation of muscle
   B. Decreased muscle excitation and contraction
   C. Increased muscle excitation and contraction
   D. Increased flaccidity of muscle

4. Based on the information presented in the case study, which one of the following disorders does Ms. Pulaski have?
   A. Muscular dystrophy
   B. Poliomyelitis
   C. Fibromyositis
   D. Myasthenia gravis
INTRODUCTION
A. Muscular system is responsible for moving the framework of the body
B. In addition to movement, muscle tissue performs various other functions

GENERAL FUNCTIONS
A. Movement of the body as a whole or of its parts
B. Heat production
C. Posture

FUNCTION OF SKELETAL MUSCLE TISSUE
A. Characteristics of skeletal muscle cells
   1. Excitability (irritability)—ability to be stimulated
   2. Contractility—ability to contract, or shorten, and produce body movement
   3. Extensibility—ability to extend, or stretch, allowing muscles to return to their resting length
B. Overview of the muscle cell (Figures 11-1 through 11-3)
   1. Muscle cells are called fibers because of their thread-like shape
   2. Sarcolemma—plasma membrane of muscle fibers
   3. Sarcomplasmic reticulum
      a. Network of tubules and sacs found within muscle fibers
      b. Membrane of the sarcoplasmic reticulum continually pumps calcium ions from the sarcoplasm and stores the ions within its sacs
   4. Muscle fibers contain many mitochondria and several nuclei
   5. Myofibrils—numerous fine fibers packed close together in sarcoplasm
   6. Sarcomere
      a. Segment of myofibril between two successive Z lines
      b. Each myofibril consists of many sarcomeres
      c. Contractile unit of muscle fibers
   7. Striated muscle
      a. Dark stripes called A bands; light H zone runs across midsection of each dark A band
      b. Light stripes called I bands; dark Z line extends across center of each light I band
   8. T tubules
      a. Transverse tubules extend across the sarcoplasm at right angles to the long axis of the muscle fiber
      b. Formed by inward extensions of the sarcolemma
      c. Membrane has ion pumps that continually transport Ca$^{2+}$ ions inward from the sarcoplasm
      d. Allow electrical impulses traveling along the sarcolemma to move deeper into the cell
   9. Triad
      a. Triplet of tubules; a T tubule sandwiched between two sacs of sarcoplasmic reticulum. Allows an electrical impulse traveling along a T tubule to stimulate the membranes of adjacent sacs of the sarcoplasmic reticulum
C. Myofilaments (Figure 11-4)
   1. Each myofibril contains thousands of thick and thin myofilaments
   2. Four different kinds of protein molecules make up myofilaments
      a. Myosin
         (1) Makes up almost all the thick filament
         (2) Myosin “heads” are chemically attracted to actin molecules
         (3) Myosin “heads” are known as cross bridges when attached to actin
      b. Actin—globular protein that forms two fibrous strands twisted around each other to form the bulk of the thin filament
      c. Tropomyosin—protein that blocks the active sites on the actin molecules
      d. Troponin—protein that holds tropomyosin molecules in place
   3. Thin filaments attach to both Z lines of a sarcomere and extend part way toward the center
   4. Thick myosin filaments do not attach to the Z lines
D. The mechanism of contraction
   1. Excitation and contraction (Figures 11-5 through 11-7; Table 11-1)
      a. A skeletal muscle fiber remains at rest until stimulated by a motor neuron
      b. Neuromuscular junction—motor neurons connect to the sarcolemma at the motor endplate (Figure 11-5)
      c. Neuromuscular junction is a synapse where neurotransmitter molecules transmit signals
      d. Acetylcholine—the neurotransmitter released into the synaptic cleft that diffuses across the gap, stimulates the receptors, and initiates an impulse in the sarcolemma
      e. Nerve impulse travels over the sarcolemma and inward along the T tubules, which triggers the release of calcium ions
      f. Calcium binds to troponin, causing the tropomyosin to shift and expose active sites on the actin
      g. Sliding filament theory (Figure 11-8)
         (1) When active sites on the actin are exposed, myosin heads bind to them
         (2) Myosin heads bend, pulling the thin filaments past them
         (3) Each head releases, binds to the next active site, and pulls again
         (4) The entire myofibril shortens
2. Relaxation
   a. Immediately after the Ca\(^{++}\) ions are released, the sarcoplasmic reticulum begins actively pumping them back into the sacs
   b. Ca\(^{++}\) ions are removed from the troponin molecules, shutting down the contraction
3. Energy sources for muscle contraction (Figure 11-9)
   a. Hydrolysis of ATP yields the energy required for muscular contraction
   b. Adenosine triphosphate (ATP) binds to the myosin head and then transfers its energy to the myosin head to perform the work of pulling the thin filament during contraction
   c. Muscle fibers continually resynthesize ATP from the breakdown of creatine phosphate
   d. Catabolism by muscle fibers requires glucose and oxygen
   e. At rest, excess O\(_2\) in the sarcoplasm is bound to myoglobin
      (1) Red fibers—muscle fibers with high levels of myoglobin
      (2) White fibers—muscle fibers with little myoglobin
   f. Aerobic respiration occurs when adequate O\(_2\) is available
   g. Anaerobic respiration occurs when low levels of O\(_2\) are available and results in the formation of lactic acid
   h. Skeletal muscle contraction produces waste heat that can be used to help maintain the set point body temperature (Figure 11-10)

FUNCTION OF SKELETAL MUSCLE ORGANS
A. Muscles are composed of bundles of muscle fibers that are held together by fibrous connective tissue
B. Motor unit (Figure 11-11)
   1. Motor unit—motor neuron plus the muscle fibers to which it attaches
   2. Some motor units consist of only a few muscle fibers, whereas others consist of numerous fibers
   3. Generally, the smaller the number of fibers in a motor unit, the more precise the movements available; the larger the number of fibers in a motor unit, the more powerful a contraction is available
C. Myography
D. Twitch contraction (Figure 11-12)
   1. A quick jerk of a muscle that is produced as a result of a single, brief threshold stimulus (generally occurs only in experimental situations)
   2. The twitch contraction has three phases
      a. Latent phase—nerve impulse travels to the sarcoplasmic reticulum to trigger release of Ca\(^{++}\)
      b. Contraction phase—Ca\(^{++}\) binds to troponin and sliding of filaments occurs
      c. Relaxation phase—sliding of filaments ceases
E. Treppe—the staircase phenomenon (Figure 11-13, B)
   1. Gradual, steplike increase in the strength of contraction that is seen in a series of twitch contractions that occur 1 second apart
   2. Eventually, the muscle responds with less forceful contractions, and relaxation phase becomes shorter
   3. If relaxation phase disappears completely, a contraction occurs
F. Tetanus—smooth, sustained contractions
   1. Multiple wave summation—multiple twitch waves are added together to sustain muscle tension for a longer time
   2. Incomplete tetanus—very short periods of relaxation occur between peaks of tension (Figure 11-13, C)
   3. Complete tetanus—the stimulation is such that twitch waves fuse into a single, sustained peak (Figure 11-13, D)
G. Muscle tone
   1. Tonic contraction—continual, partial contraction of a muscle
   2. At any one time, a small number of muscle fibers within a muscle contract, producing a tightness or muscle tone
   3. Muscles with less tone than normal are flaccid
   4. Muscles with more tone than normal are spastic
   5. Muscle tone is maintained by negative feedback mechanisms
H. Graded strength principle
   1. Graded strength principle—skeletal muscles contract with varying degrees of strength at different times
   2. Factors that contribute to the phenomenon of graded strength (Figure 11-17)
      a. Metabolic condition of individual fibers
      b. Number of muscle fibers contracting simultaneously; the greater the number of fibers contracting, the stronger the contraction
      c. Number of motor units recruited
      d. Intensity and frequency of stimulation (Figure 11-14)
   3. Length-tension relationship (Figure 11-15)
      a. Maximal strength that a muscle can develop bears a direct relationship to the initial length of its fibers
      b. A shortened muscle's sarcomeres are compressed, therefore the muscle cannot develop much tension
      c. An overstretched muscle cannot develop much tension because the thick myofilaments are too far from the thin myofilaments
      d. Strongest maximal contraction is possible only when the skeletal muscle has been stretched to its optimal length
4. Stretch reflex (Figure 11-16)
   a. The load imposed on a muscle influences the strength of a skeletal contraction
   b. Stretch reflex—the body tries to maintain a constancy of muscle length in response to increased load
c. Maintains a relatively constant length as load is increased up to a maximum sustainable level

I. Isotonic and isometric contractions (Figure 11-18)

1. Isotonic contraction
   a. Contraction in which the tone or tension within a muscle remains the same as the length of the muscle changes
      (1) Concentric—muscle shortens as it contracts
      (2) Eccentric—muscle lengthens while contracting
   b. Isotonic—literally means “same tension”
   c. All of the energy of contraction is used to pull on thin myofilaments and thereby change the length of a fiber’s sarcomeres

2. Isometric contraction
   a. Contraction in which muscle length remains the same while the muscle tension increases
   b. Isometric—literally means “same length”

3. Most body movements occur as a result of both types of contractions

FUNCTION OF CARDIAC AND SMOOTH MUSCLE TISSUE

A. Cardiac muscle (Figure 11-19)
   1. Found only in the heart, forming the bulk of the wall of each chamber
   2. Also known as striated involuntary muscle
   3. Contracts rhythmically and continuously to provide the pumping action needed to maintain a constant blood flow
   4. Cardiac muscle resembles skeletal muscle but has specialized features related to its role in continuously pumping blood
      a. Each cardiac muscle contains parallel myofibrils (Figure 11-19)
      b. Cardiac muscle fibers form strong, electrically coupled junctions (intercalated disks) with other fibers; individual cells also exhibit branching
      c. Syncytium—continuous, electrically coupled mass
      d. Cardiac muscle fibers form a continuous, contractile band around the heart chambers that conducts a single impulse across a virtually continuous sarcolemma
      e. T tubules are larger and form diads with a rather sparse sarcoplasmic reticulum
   f. Cardiac muscle sustains each impulse longer than in skeletal muscle, therefore impulses cannot come rapidly enough to produce tetanus (Figure 11-20)
   g. Cardiac muscle does not run low on ATP and does not experience fatigue
   h. Cardiac muscle is self-stimulating

B. Smooth muscle
   1. Smooth muscle is composed of small, tapered cells with single nuclei (Figure 11-21)
   2. No T tubules are present, and only a loosely organized sarcoplasmic reticulum is present
   3. Ca++ comes from outside the cell and binds to calmodulin instead of troponin to trigger a contraction
   4. No striations, because thick and thin myofilaments are arranged differently than in skeletal or cardiac muscle fibers; myofilaments are not organized into sarcomeres
   5. Two types of smooth muscle tissue
      a. Visceral muscle (single unit)
         (1) Gap junctions join smooth muscle fibers into large, continuous sheets
         (2) Most common type; forms a muscular layer in the walls of hollow structures such as the digestive, urinary, and reproductive tracts
         (3) Exhibits autorhythmicity, producing peristalsis
      b. Multiunit
         (1) Does not act as a single unit but is composed of many independent cell units
         (2) Each fiber responds only to nervous input

THE BIG PICTURE: MUSCLE TISSUE AND THE WHOLE BODY

A. Function of all three major types of muscle is integral to the function of the entire body
B. All three types of muscle tissue provide the movement necessary for survival
C. Relative constancy of the body’s internal temperature is maintained by “waste” heat generated by muscle tissue
D. Maintains the body in a relatively stable position
1. Define the terms **sarcolemma**, **sarcoplasm**, and **sarcoplasmic reticulum**.
2. Describe the function of the sarcoplasmic reticulum.
3. How are acetylcholine, $\text{Ca}^{++}$, and adenosine triphosphate (ATP) involved in the excitation and contraction of skeletal muscle?
4. Describe the general structure of ATP and tell how it relates to its function.
5. How does ATP provide energy for a muscle contraction?
6. Describe the anatomical arrangement of a motor unit.
7. List and describe the different types of skeletal muscle contractions.
8. Define the term **recruited**.
10. What are the effects of exercise on skeletal muscles?

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**CRITICAL THINKING QUESTIONS**

1. Explain how skeletal muscles provide movement, heat, and posture. Are all of these functions unique to muscles? Explain your answer.
2. The characteristic of excitability is shared by what other system? Relate contractility and extensibility to the concept of agonist and antagonist discussed in Chapter 10.
3. What structures are unique to skeletal muscle fibers? Which of the structures are involved primarily in contractility and which are involved in excitability?
4. Explain how the structure of myofilaments is related to their function.
5. Explain how the sliding filament theory allows for the shortening of a muscle fiber.
6. Compare and contrast the role of $\text{Ca}^{++}$ in excitation, contraction, and relaxation of skeletal muscle.
7. People who exercise seriously are sometimes told to work a muscle until they “feel the burn.” In terms of how the muscle is able to release energy, explain what is going on in the muscle early in the exercise and when the muscle is “burning.”
8. Using fiber types, design a muscle for a marathon runner and a different muscle for a 100-yard–dash sprinter. Explain your choice.
9. Explain the meaning of a “unit of combined cells” as it relates to cardiac muscle. How does this structural arrangement affect its function?
10. Which of the two smooth muscle types would be most affected by damage to the nerves that stimulate them?
CAREER CHOICES

Massage Therapist

Massage therapy improves circulation and helps to correct imbalances in the soft tissue areas of the body, as in muscles and fascia. As a massage therapist, I am self-employed. My clientele consist of hospital-referred patients for lymph drainage therapy and people who seek traditional massage for various reasons. The majority of my work consists of home visits; however, I also work part-time at Endless Creations Spa and at private massage parties.

I have been massaging people since I was 5 years old. I massaged my other classmates whenever they had problems or hurt themselves. It has always been natural and enjoyable to work on others with my hands.

My certifications are as follows:

- CMT—Certified Massage Therapist (Swedish Massage)
- MLDT—Manual Lymph Drainage Massage Therapist
- CDT—Combined Decongestive Therapist
- OBT—Oriental Bodywork Therapist (Shiatsu)
- NCTMB—Nationally Certified Therapist of Massage and Bodywork

I am also certified in La Stone Therapy, Reiki 2, Reflexology, and Myofacial Release. I have completed additional study in Zero Balancing, NeuroMuscular Therapy, Sports Massage, and CrainoSacral Therapy.

Some of the current trends in the massage profession are La Stone Therapy, treatment of cancer patients (with a doctor’s approval), and stress and pain relief.

My job is extremely rewarding. I rarely encounter a client in a bad mood before a massage, and never after completion! It is very rewarding to see a client or patient’s problem improve, or simply see a look of contentment on their face. There is a personal, respectful bond between the therapist and the client. In addition this profession can be financially rewarding.

Knowledge of anatomy and physiology is definitely needed to effectively treat muscular/skeletal problems because isolation of the involved muscle is necessary. I still review anatomy and physiology of the muscular, skeletal, and lymphatic areas all the time. My advice is to study anatomy and physiology in a quiet room, do not study an entire chapter in one sitting, spread the chapter out, and take notes!