Table 10-13 Muscles That Move the Thigh

Muscle	Origin	Insertion	Function	Nerve Supply
Iliopsoas (iliacus, psoas major, and psoas minor)	Ilium (iliac fossa) Vertebrae (bodies of twelfth thoracic to fifth lumbar)	Femur (lesser trochanter)	Flexes thigh Flexes trunk (when femur acts as origin)	Femoral and second to fourth lumbar nerves
Rectus femoris	Ilium (anterior, inferior spine)	Tibia (by way of patellar tendon)	Flexes thigh Extends lower leg	Femoral nerve
Gluteal group				
Maximus	Ilium (crest and posterior surface) Sacrum and coccyx (posterior surface) Sacrotuberous ligament	Femur (gluteal tuberosity) Iliotibial tract	Extends thigh—rotates outward	Inferior gluteal nerve
Medius	Ilium (lateral surface)	Femur (greater trochanter)	Abducts thigh—rotates outward; stabilizes pelvis on femur	Superior gluteal nerve
Minimus	Ilium (lateral surface)	Femur (greater trochanter)	Abducts thigh; stabilizes pelvis on femur Rotates thigh medially	Superior gluteal nerve
Tensor fasciae latae	Ilium (anterior part of crest)	Tibia (by way of iliotibial tract)	Abducts thigh Tightens iliotibial tract	Superior gluteal nerve
Adductor group				
Brevis	Pubic bone	Femur (linea aspera)	Adducts thigh	Obturator nerve
Longus	Pubic bone	Femur (linea aspera)	Adducts thigh	Obturator nerve
Magnus	Pubic bone	Femur (linea aspera)	Adducts thigh	Obturator nerve
Gracilis	Pubic bone (just below symphysis)	Tibia (medial surface behind sartorius)	Adducts thigh and flexes and adducts leg	Obturator nerve

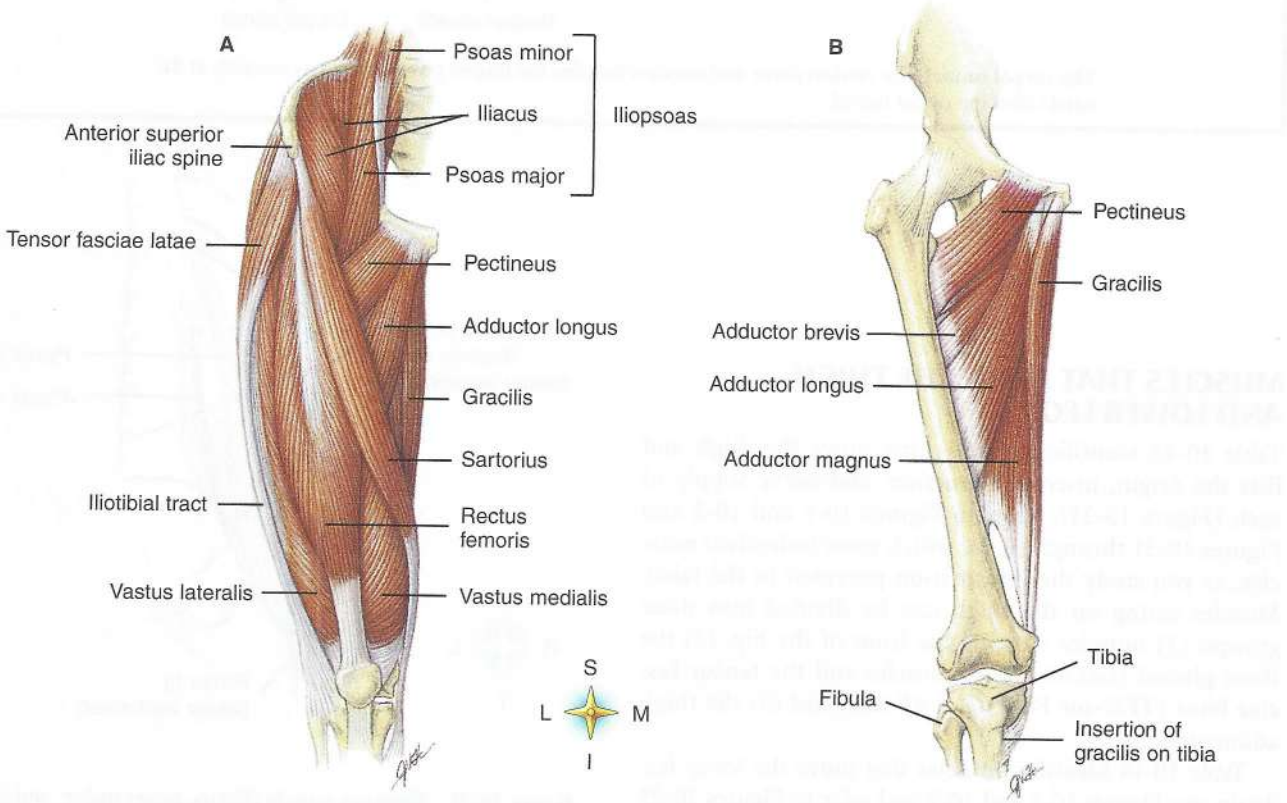


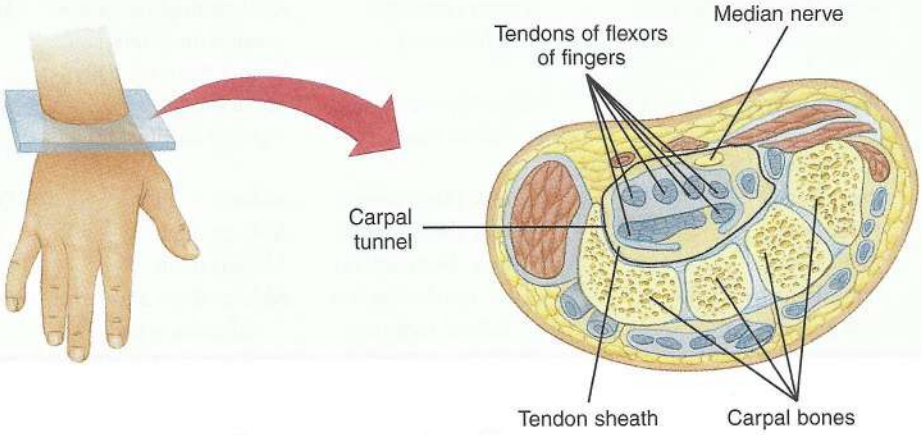
Figure 10-22 Muscles of the anterior thigh. A, Anterior view of the right thigh. B, Adductor region of the right thigh. Tensor fasciae latae, sartorius, and quadriceps muscles have been removed.

Box 10-3 HEALTH MATTERS

Carpal Tunnel Syndrome

Some epidemiologists specialize in the field of occupational health, the study of health matters related to work or the workplace. Many problems seen by occupational health experts are caused by repetitive motions of the wrists or other joints. Word processors (typists) and meat cutters, for example, are at risk of developing conditions caused by repetitive motion injuries.

One common problem often caused by such repetitive motion is **tenosynovitis** (ten-o-sin-o-VYE-tis)– inflammation of a tendon sheath. Tenosynovitis can be painful, and the swelling characteristic of this condition can limit movement in affected parts of the body. For example, swelling of the tendon sheath around tendons in an area of the wrist known as the *carpal tunnel* can limit movement of the wrist, hand, and fingers. The figure shows the relative positions of the tendon sheath and median nerve within the carpal tunnel. If this swelling, or any other lesion in the carpal tunnel, presses on the *median nerve,* a condition called **carpal tunnel syndrome** may result. Because the median nerve connects to the palm and radial side (thumb side) of the hand, carpal tunnel syndrome is characterized by weakness, pain, and tingling in this part of the hand. The pain and tingling may also radiate to the forearm and shoulder. Prolonged or severe cases of carpal tunnel syndrome may be relieved by injection of antiinflammatory agents. A permanent cure is sometimes accomplished by surgical cutting or removal of the swollen tissue pressing on the median nerve.



The carpal tunnel. The median nerve and muscles that flex the fingers pass through a concavity in the wrist called the carpal tunnel.

MUSCLES THAT MOVE THE THIGH AND LOWER LEG

Table 10-13 identifies muscles that move the thigh and lists the origin, insertion, function, and nerve supply of each (Figure 10-21). Refer to Figures 10-1 and 10-2 and Figures 10-21 through 10-24, which show individual muscles, as you study the information provided in the table. Muscles acting on the thigh can be divided into three groups: (1) muscles crossing the front of the hip, (2) the three **gluteal** (GLOO-tee-al) muscles and the **tensor fasciae latae** (TEN-sor FASH-ee LAT-tee), and (3) the thigh adductors.

Table 10-14 identifies muscles that move the lower leg. Again, see Figures 10-1 and 10-2 and refer to Figures 10-25 and 10-26 as you study the table.

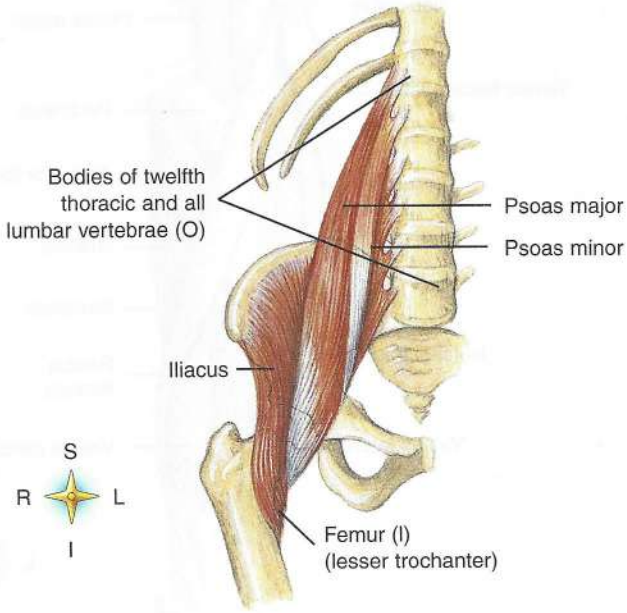


Figure 10-21 Iliopsoas muscle (iliacus, psoas major, and psoas minor muscles). *O,* Origin; *I,* insertion.

MYOFILAMENTS

Each muscle fiber contains a thousand or more parallel subunits, called **myofibrils**, that are only about 1 m 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-

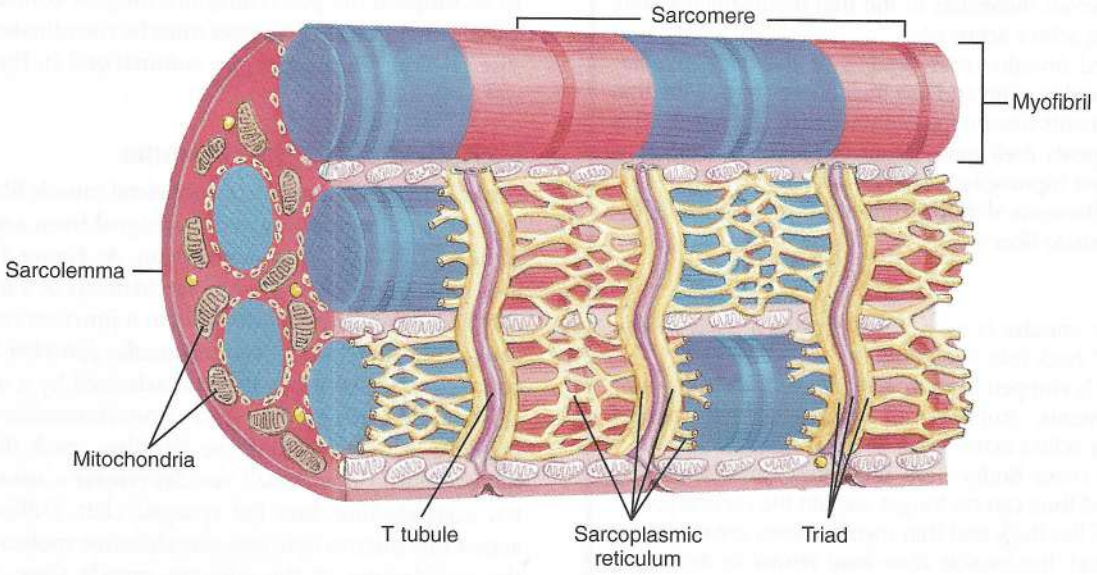


Figure 11-3 Unique features of the skeletal muscle cell. Notice especially the T tubules, which are extensions of the plasma membrane, or sarcolemma, and the sarcoplasmic reticulum (SR), which forms networks of tubular canals and sacs. A triad is a triplet of adjacent tubules: a terminal (end) sac of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

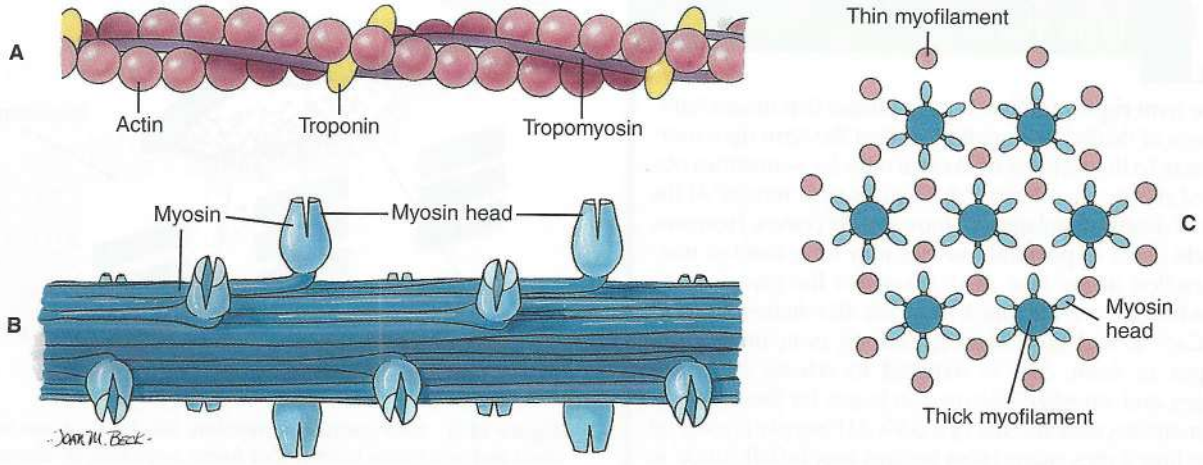


Figure 11-4 Structure of myofilaments. A, Thin myofilament. B, Thick myofilament. C, Cross section of several thick and thin myofilaments, showing the relative positions of myofilaments and the myosin heads that will form cross bridges between them.

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 impulses that travels along the sarcolemma, through the T tubules, to the sacs of the SR.
4. Ca^{++} 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^{++} back into its sacs.
2. As Ca^{++} is stripped from troponin molecules in the think 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.

ments, so they angle toward the thin filaments. When they bridge the gap between adjacent myofilaments, the myosin heads are usually called **cross bridges.**

Within a myofibril the thick and thin filaments alternate, as shown in Figure 11-1, *D*. This arrangement is crucial for contraction. Another fact important for contraction is that the thin filaments attach to both Z lines of a sarcomere and that they extend in from the Z lines partway toward the center of the sarcomere. When the muscle fiber is relaxed, the thin filaments terminate at the outer edges of the H zones. In contrast, the thick myosin filaments do not attach directly to the Z lines, and they extend only the length of the A bands of the sarcomeres.

THE MECHANISM OF CONTRACTION

To accomplish the powerful shortening, or contraction, of a muscle fiber, several processes must be coordinated in a stepwise 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

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^{++} 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.

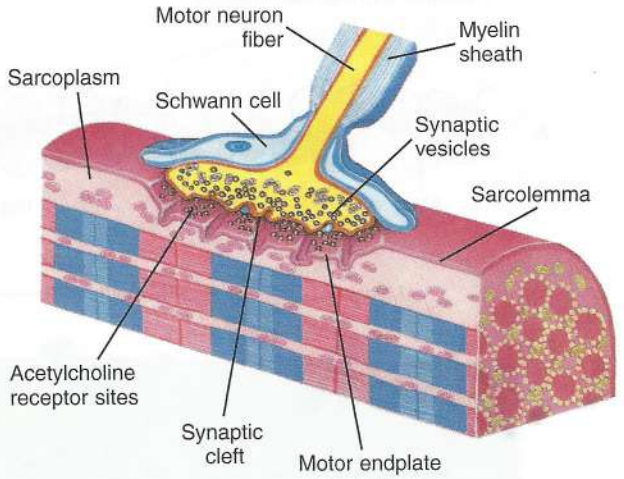


Figure 11-5 Neuromuscular junction. This figure shows how the distal end of a motor neuron fiber forms a synapse, or "chemical junction," with an adjacent muscle fiber. Neurotransmitters (specifically, acetylcholine) are released from the neuron's synaptic vesicles and diffuse across the synaptic cleft. There they stimulate receptors in the motor endplate region of the sarcolemma.

Box 11-5 SPORTS AND FITNESS
Muscle Fatigue

Broadly defined, **muscle fatigue** is simply a state of exhaustion (a loss of strength or endurance) produced by strenuous muscular activity. *Physiological muscle fatigue* is caused by a relative lack of ATP, rendering the myosin heads incapable of producing the force required for further muscle contractions. The low levels of ATP that produce fatigue may result from a depletion of oxygen or glucose in muscle fibers or from the inability to regenerate ATP quickly enough. The most frequent cause of physiological fatigue is depletion of glycogen in the muscle. High levels of lactic acid or other metabolic waste products also contribute to physiological fatigue. Under ordinary circumstances, however, complete physiological fatigue seldom occurs. It is usually *psychological fatigue* that produces the exhausted feeling that stops us from continuing a muscular activity. Thus in physiological muscle fatigue, we *cannot* contract our muscles, but in psychological muscle fatigue, we simply *will not* contract our muscles because we feel tired.

TETANUS

The concept of the simple twitch can help us understand the smooth, sustained types of contraction that are commonly observed in the body. Such smooth, sustained contractions are called *tetanic contractions*, or, simply, ***tetanus.*** Figure 11-13, *C*, shows that if a series of stimuli come in a rapid enough succession, the muscle does not have time to relax completely before the next contraction phase begins. Muscle physiologists describe this effect as *multiple wave summation*—so named because it seems as if multiple twitch waves have been added together to sustain muscle tension for a longer time. The type of tetanus produced when very short periods of relaxation occur between peaks of tension is called *incomplete tetanus*. It is "incomplete" because the tension is not sustained at a completely constant level. Figure 11-13, *D*, shows that when the frequency of stimuli increases, the distance between peaks of tension decrease to a point at which they seem to fuse into a single, sustained peak. This produces a very smooth type of tetanic contraction, called *complete tetanus*.

In a normal body, tetanus results from the coordinated contractions of different motor units within the muscle organ. These motor units fire in an overlapping time sequence to produce a "relay team" effect that results in a sustained contraction. Tetanus is the kind of contraction exhibited by normal skeletal muscle organs most of the time.

Quick Review

1. What are the three phases of a twitch contraction? What molecular events occur during each of these phases?
2. What is the difference between a twitch contraction and a tetanic contraction?
3. How does the treppe effect relate to the warm-up exercises of athletes?

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

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.)

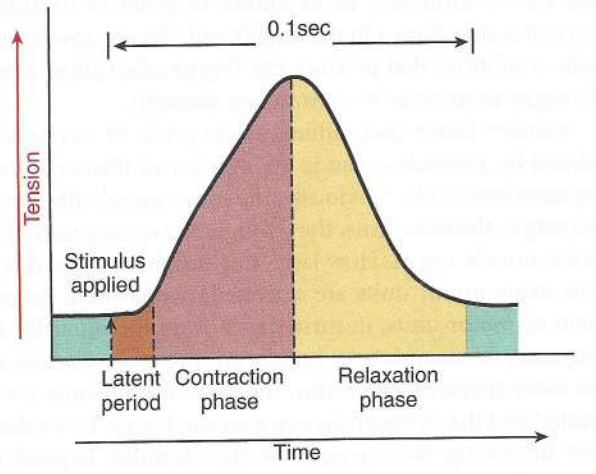


Figure 11-12 The twitch contraction. Three distinct phases are apparent: (1) the latent period, (2) the contraction phase, and (3) the relaxation phase.

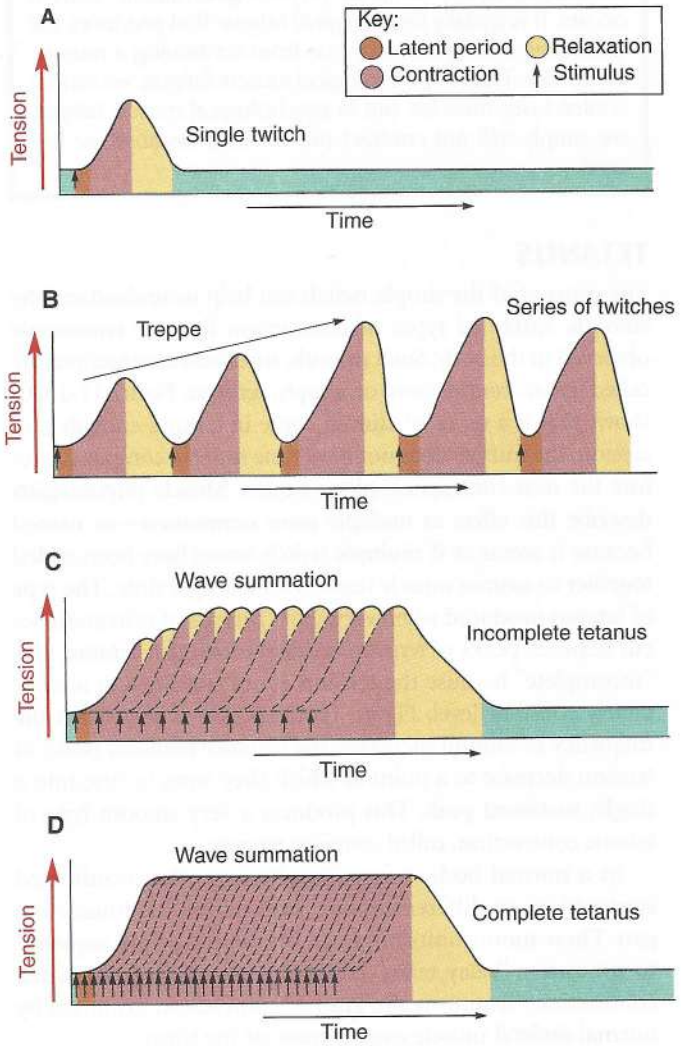


Figure 11-13 Myograms of various types of muscle contractions. A, A single twitch contraction. B, The treppe phenomenon, or "staircase effect," is a steplike increase in the force of contraction over the first few in a series of twitches. C, Incomplete tetanus occurs when a rapid succession of stimuli 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.

Box 11-6

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.

each additional motor unit is crossed, the strength of contraction increases. This continues as the strength of stimulation increases until the maximal level of contraction is reached. At this point, the limits of the muscle organ to recruit new motor units have been reached. Even if stimulation increases above the maximal level, the muscle cannot contract any more strongly. As long as the supply of ATP holds out, the muscle organ can sustain a tetanic contraction at the maximal level when motor units contract and relax in overlapping "relays" (see Figure 11-13, *D*).

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

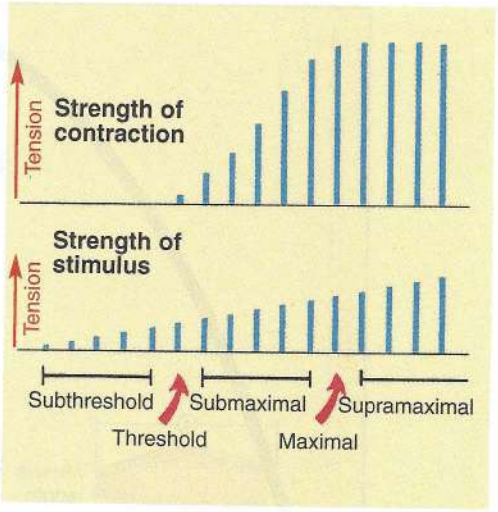


Figure 11-14 The strength of muscle contraction compared with the strength of the stimulus. After the threshold stimulus is reached, a continued increase in stimulus strength produces a proportional increase in muscle strength until the maximal level of contraction strength is reached.

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

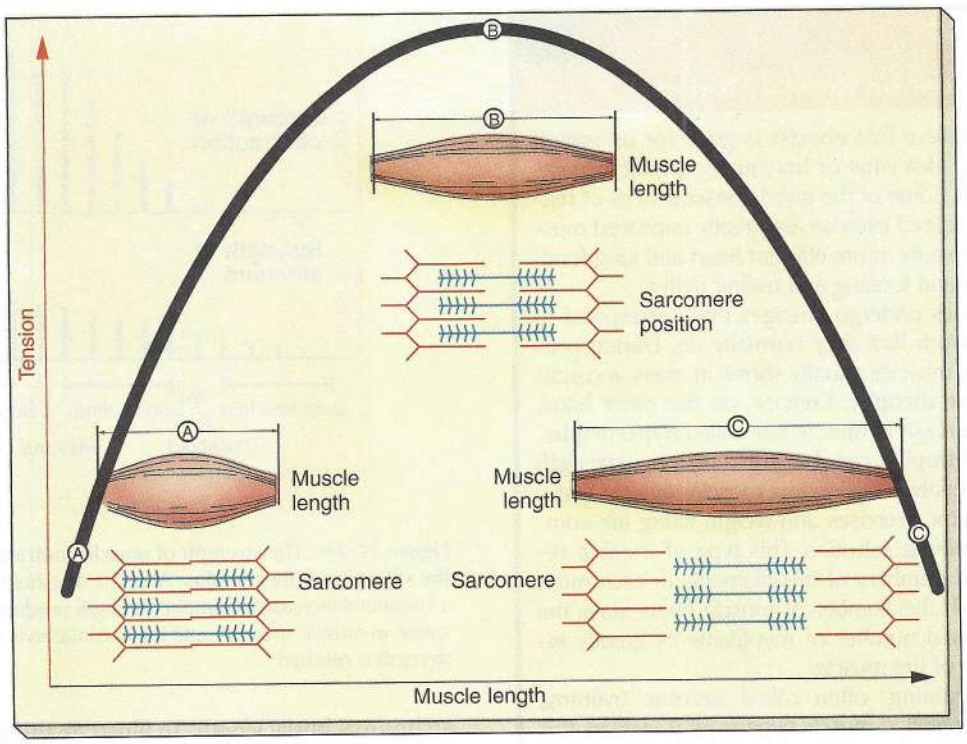


Figure 11-15 The length-tension relationship. As this graph of muscle tension shows, the maximum strength that a muscle can develop is directly related to the initial length of its fibers. At a short initial length the sarcomeres are already compressed and thus the muscle cannot develop much tension (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).

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.

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

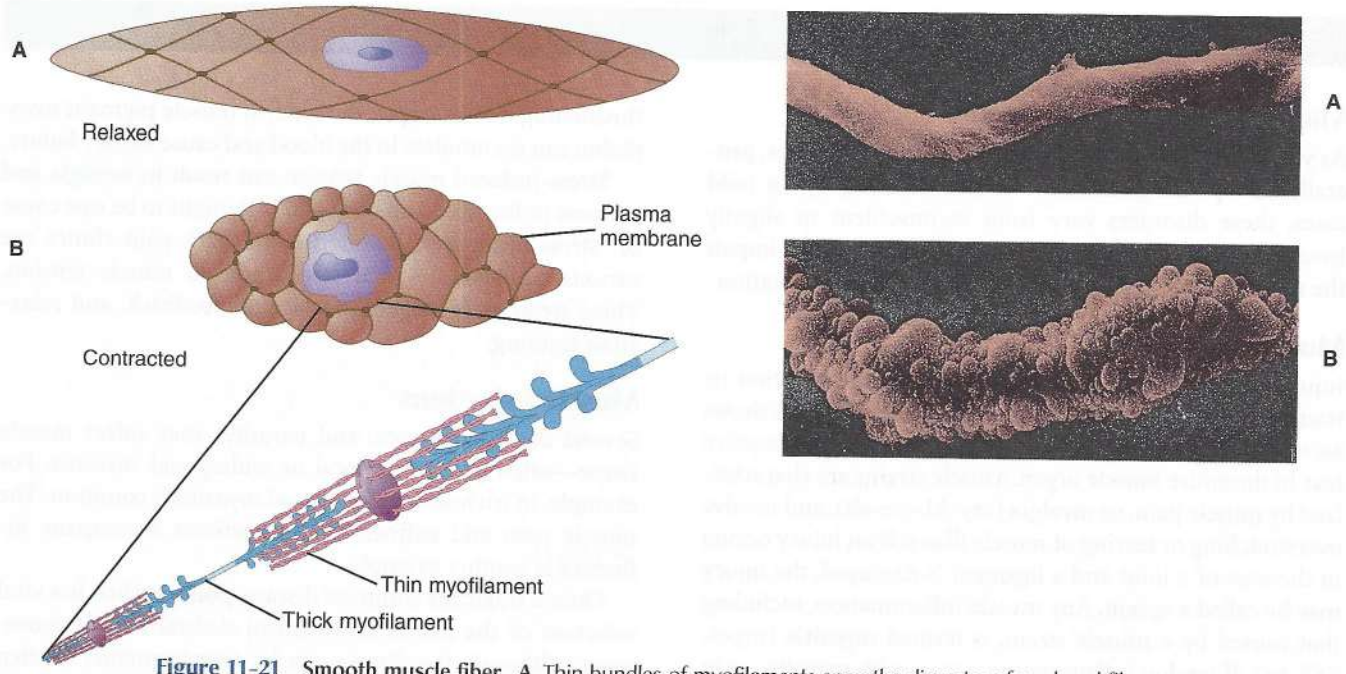


Figure 11-21 Smooth muscle fiber. **A**, Thin bundles of myofilaments span the diameter of a relaxed fiber. The scanning electron micrograph *(right)* shows that the surface of the cell is rather flat when the fiber is relaxed. **B**, During contraction, sliding of the myofilaments causes the fiber to 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.

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.

Quick Check

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.

MECHANISMS OF DISEASE

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-jee-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.

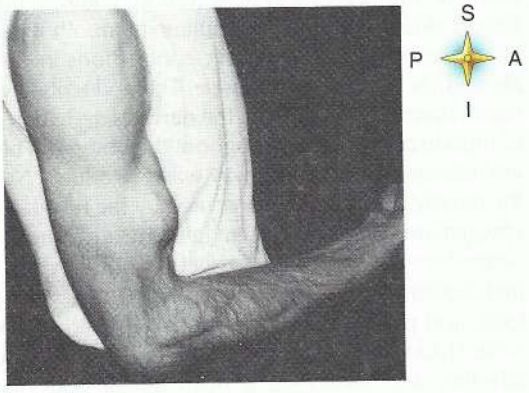


Figure 11-22 Muscle strain. Severe strain of the biceps brachii muscle. In a severe muscle strain, a muscle may break in two pieces, causing a visible gap in muscle tissue under the skin. Notice how the broken ends of the muscle reflexively contract (spasm) to form a knot of tissue.

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 "strangulated" 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.

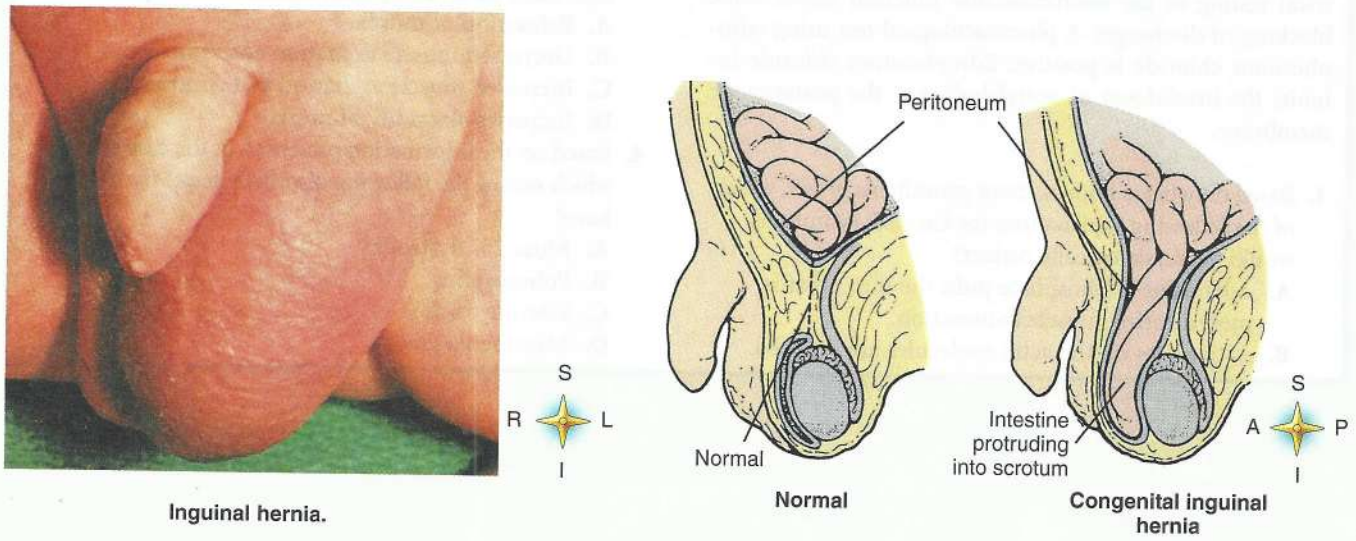


Figure 11-23 Inguinal hernia in infant male.

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