to express our emotions with the silent language of smiles and frowns.

These are distinct from the smooth muscle of blood vessel walls and cardiac muscle of the heart, which work together to circulate blood and maintain blood pressure, and the smooth muscle of other hollow organs, which forces fluids (urine, bile) and other substances (food, a baby) through internal body channels.

Maintaining Posture

We are rarely aware of the workings of the skeletal muscles that maintain body posture. Yet, they function almost continuously, making one tiny adjustment after another so that we can maintain an erect or seated posture despite the never-ending downward pull of gravity.

Stabilizing Joints

As the skeletal muscles pull on bones to cause movements, they also stabilize the joints of the skeleton. Indeed, muscle tendons are extremely important in reinforcing and stabilizing joints that have poorly fitting articulating surfaces (the shoulder joint, for example).

Generating Heat

The fourth function of muscle, generation of body heat, is a by-product of muscle activity. As ATP is used to power muscle contraction, nearly three-quarters of its energy escapes as heat. This heat is vital in maintaining normal body temperature. Since skeletal muscle accounts for at least 40 percent of body mass, it is the muscle type most responsible for heat generation.

As you can see, each of the three muscle types has a structure and function well suited for its job in the body. But since the term ***muscular system*** applies specifically to skeletal muscle, we will be concentrating on this muscle type in this chapter. The most important structural and functional aspects of the three muscle types are outlined in Table 6.1 (p. 179).

Microscopic Anatomy of Skeletal Muscle

As mentioned above and illustrated in Figure 6.3a, skeletal muscle cells are multinucleate. Many oval nuclei can be seen just beneath the plasma membrane, which is called the **sarcolemma** (sar″ko-lem′ah; "muscle husk") in muscle cells. The nuclei are pushed aside by long ribbonlike organelles, the **myofibrils** (mi″o-fi′brilz), which nearly fill the cytoplasm. Alternating **light (I)** and **dark (A) bands** along the length of the perfectly aligned myofibrils give the muscle cell as a whole its striped appearance. (Think of the second letter of *light,* I, and the second letter of *dark,* A, to help you remember which band is which.) A closer look at the banding pattern reveals that the light I band has a midline interruption, a darker area called the *Z disc,* and the dark A band has a lighter central area called the *H zone* (Figure 6.3b). The *M line* in the center of the H zone contains tiny protein rods that hold adjacent thick filaments together.

So why are we bothering with all these terms—dark this and light that? Because the banding pattern reveals the working structure of the myofibrils. First, we find that the myofibrils are actually chains of tiny contractile units called **sarcomeres** (sar′ko-merz), which are aligned end-to-end like boxcars in a train along the length of the myofibrils. Second, it is the arrangement of even smaller structures (myofilaments) *within* sarcomeres that actually produces the banding pattern.

Let's examine how the arrangement of the myofilaments leads to the banding pattern. There are two types of threadlike protein **myofilaments** within each of our "boxcar" sarcomeres (Figure 6.3c). The larger **thick filaments,** also called *myosin filaments,* are made mostly of bundled molecules of the protein **myosin,** but they also contain ATPase enzymes, which split ATP to generate the power for muscle contraction. Notice that the thick filaments extend the entire length of the dark A band. Also, notice that the midparts of the thick filaments are smooth, but their ends are studded with small projections (Figure 6.3d). These projections, or myosin *heads,* are called **cross bridges** when they link the thick and thin filaments together during contraction. The **thin filaments** are composed of the contractile protein called **actin,** plus some regulatory proteins that play a role in allowing (or preventing) myosin head–binding to actin. The thin filaments, also called *actin filaments,* are anchored to the Z disc (a disclike membrane). Notice that the light I band includes parts of two adjacent sarcomeres and contains *only* the thin filaments. Although the thin filaments overlap the ends of the thick filaments, they do not extend into the middle of a relaxed

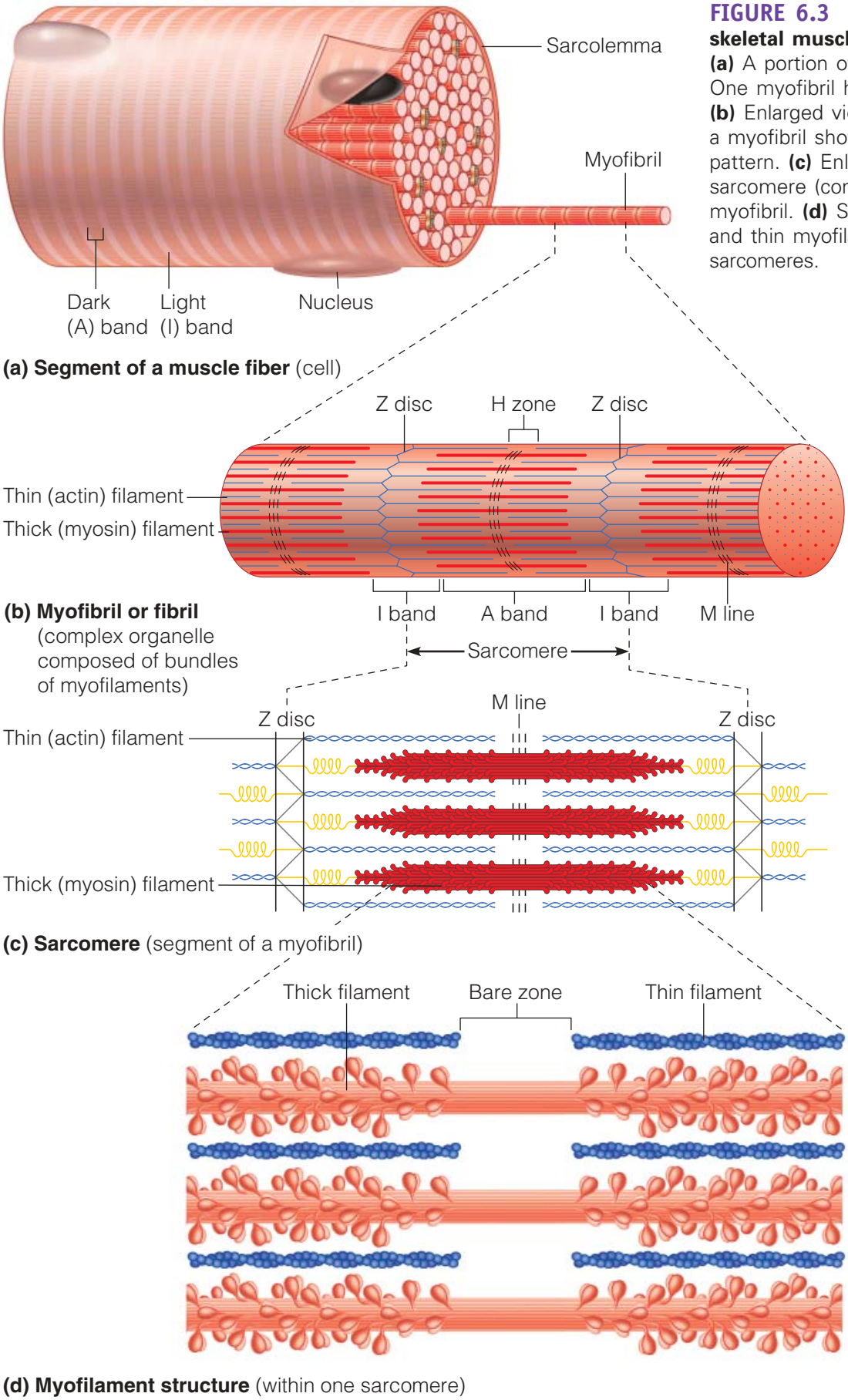

FIGURE 6.3 Anatomy of a skeletal muscle fiber (cell). **(a)** A portion of a muscle fiber. One myofibril has been extended. **(b)** Enlarged view of a section of a myofibril showing its banding pattern. **(c)** Enlarged view of one sarcomere (contractile unit) of a myofibril. **(d)** Structure of the thick and thin myofilaments found in the sarcomeres.

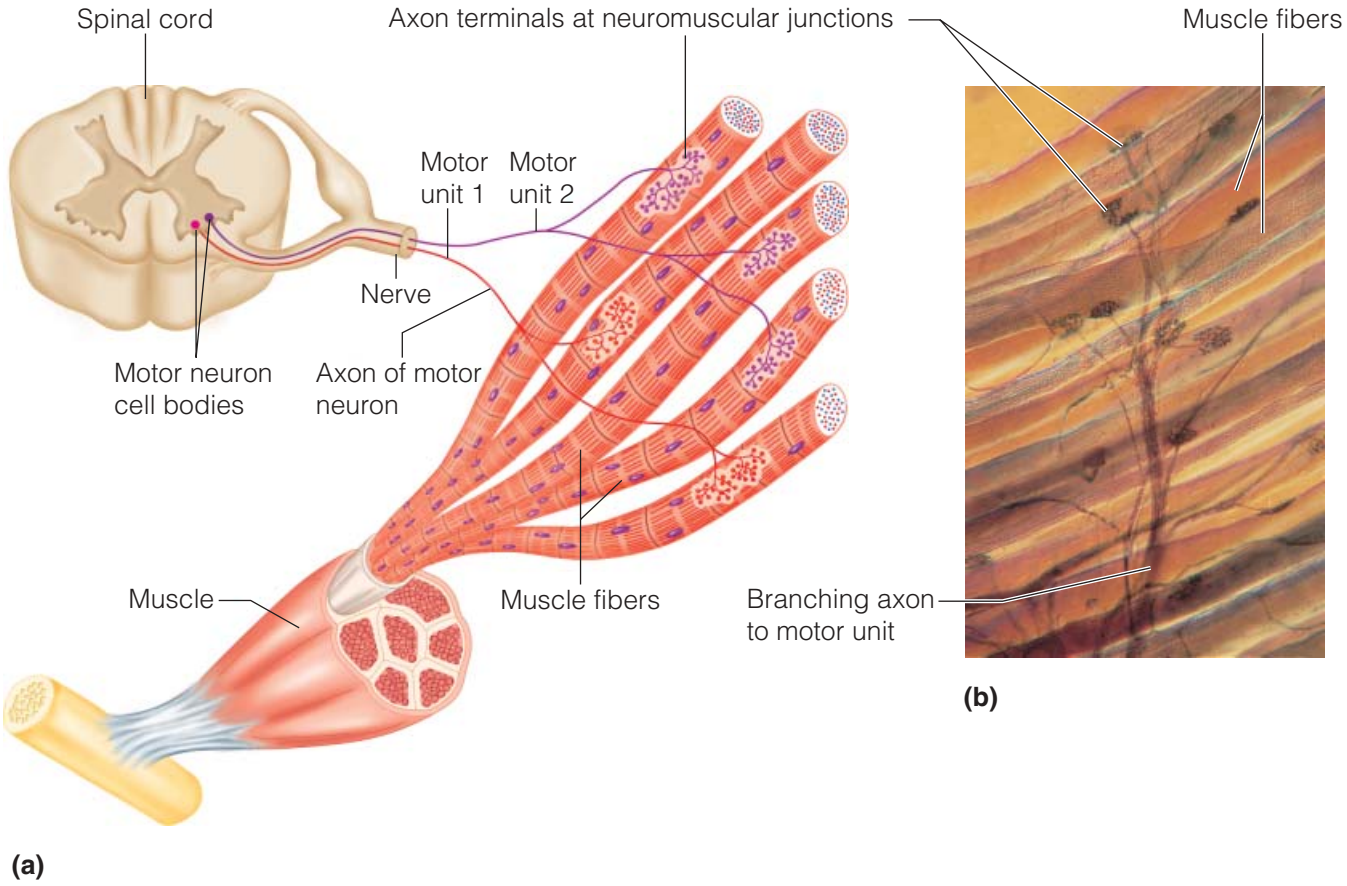

FIGURE 6.4 **Motor units.** Each motor unit consists of a motor neuron and all the muscle fibers it activates. **(a)** Portions of two motor units are shown. The motor neurons reside in the spinal cord, and their axons extend to the muscle. Within the muscle, each axon divides into a number of axon terminals, distributed to muscle fibers scattered throughout the muscle. **(b)** Photomicrograph of a portion of a motor unit (110×). Notice the diverging axon terminals and the neuromuscular junctions with the muscle fibers.

sarcomere, and thus the central region (the H zone, which lacks actin filaments and looks a bit lighter) is sometimes called the *bare zone.* When contraction occurs, and the actin-containing filaments slide toward each other into the center of the sarcomeres, these light zones disappear because the actin and myosin filaments are completely overlapped. For now, however, just recognize that it is the precise arrangement of the myofilaments in the myofibrils that produces the banding pattern, or striations, in skeletal muscle cells.

Not shown in Figure 6.3 is another very important muscle fiber organelle—the **sarcoplasmic reticulum (SR),** a specialized smooth endoplasmic reticulum. The interconnecting tubules and sacs of the SR surround each and every myofibril just as the sleeve of a loosely crocheted sweater surrounds your arm. The major role of this elaborate system is to store calcium and to release it on demand when the muscle fiber is stimulated to contract. As you will see, calcium provides the final "go" signal for contraction.

Skeletal Muscle Activity

Stimulation and Contraction of Single Skeletal Muscle Cells

Muscle cells have some special functional properties that enable them to perform their duties. The first of these is *irritability,* the ability to receive and

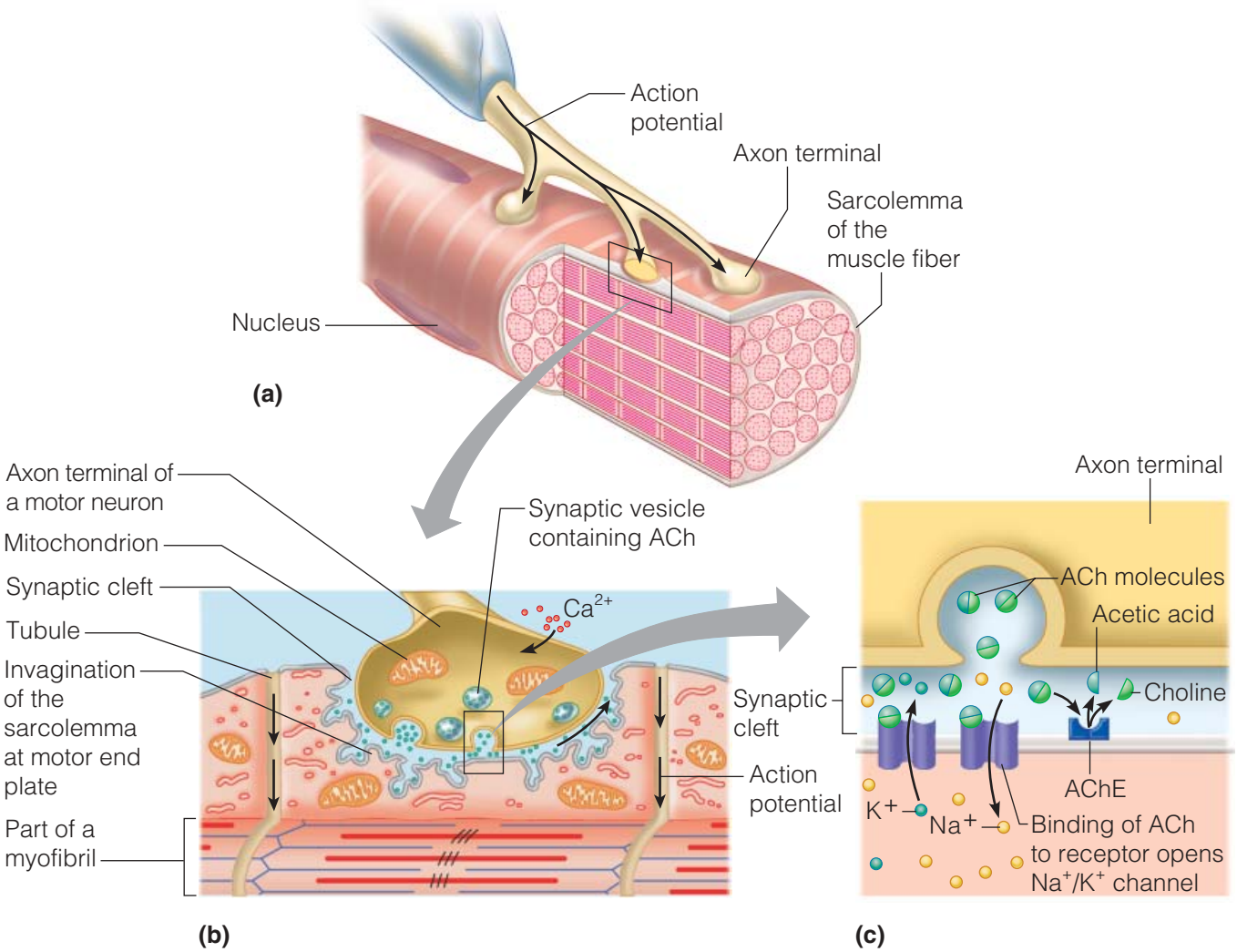

FIGURE 6.5 **The neuromuscular junction. (a)** Axon terminal of a motor neuron forming a neuromuscular junction with a muscle fiber. **(b)** The axon terminal contains vesicles filled with the neurotransmitter acetylcholine (ACh), which is released when the nerve impulse reaches the axon terminal. The sarcolemma is highly invaginated (folded) adjacent to the synaptic cleft and acetylcholine receptors are present in these folds. **(c)** Acetylcholine diffuses across the synaptic cleft and attaches to ACh receptors on the sarcolemma, initiating changes in the electrical condition of the sarcolemma.

respond to a stimulus. The second, *contractility,* is the ability to shorten (forcibly) when an adequate stimulus is received.

The Nerve Stimulus and the Action Potential

Skeletal muscle cells must be stimulated by nerve impulses to contract. One motor neuron (nerve cell) may stimulate a few muscle cells or hundreds of them, depending on the particular muscle and the work it does. One neuron and all the skeletal muscle cells it stimulates are a **motor unit** (Figure 6.4). When a long threadlike extension of the neuron, called the *nerve fiber* or **axon,** reaches the muscle, it branches into a number of **axon terminals,** each of which forms junctions with the sarcolemma of a different muscle cell (Figure 6.5). These junctions are called **neuromuscular** (literally, "nerve-muscle") **junctions.** Although the nerve endings and the muscle cells' membranes are very close, they never touch. The gap between them, the **synaptic cleft,** is filled with tissue (interstitial) fluid.

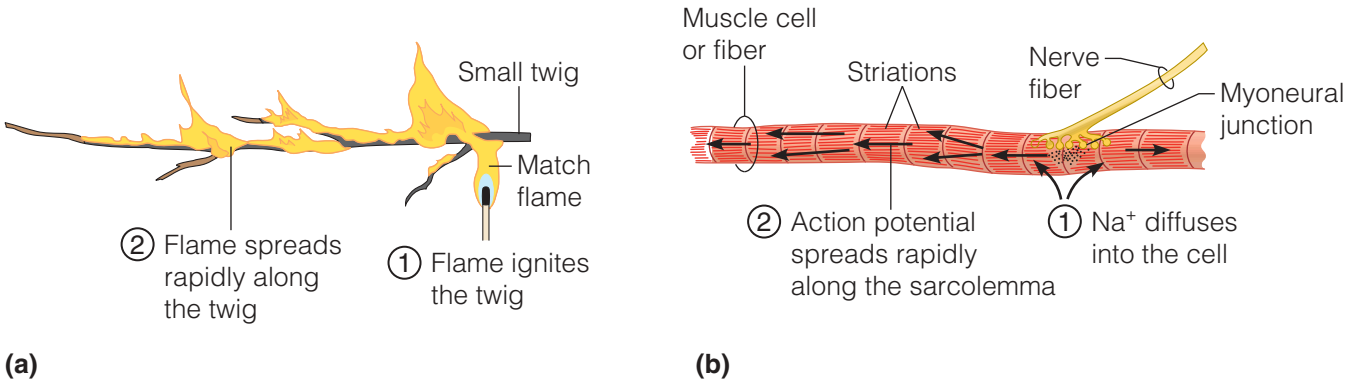

FIGURE 6.6 Comparison of the action potential to a flame consuming a dry twig. (a) The first event in igniting a dry twig is holding the match flame under one area of the twig. The second event is the twig's bursting into flame when it has been heated enough and the flame's spreading to burn the entire twig. **(b)** The first event in exciting a muscle cell is the rapid diffusion of sodium ions (Na^+) into the cell when the permeability of the sarcolemma changes. The second event is the spreading of the action potential along the sarcolemma when enough sodium ions have entered to upset the electrical conditions in the cell.

Now that we have described the structure of the neuromuscular junction, we are ready to examine what happens there. When the nerve impulse reaches the axon terminals, a chemical referred to as a **neurotransmitter** is released. The specific neurotransmitter that stimulates skeletal muscle cells is **acetylcholine** (as″e-til-ko′len), or **ACh.** Acetylcholine diffuses across the synaptic cleft and attaches to receptors (membrane proteins) that are part of the sarcolemma. If enough acetylcholine is released, the sarcolemma at that point becomes *temporarily* more permeable to sodium ions (Na^+), which rush into the muscle cell and to potassium ions (K^+) which diffuse out of the cell. However, more Na^+ enters than K^+ leaves. This gives the cell interior an excess of positive ions, which reverses the electrical conditions of the sarcolemma and opens more channels that allow Na^+ entry only. This "upset" generates an electrical current called an **action potential.** Once begun, the action potential is unstoppable; it travels over the entire surface of the sarcolemma, conducting the electrical impulse from one end of the cell to the other. The result is contraction of the muscle cell.

It should be mentioned that while the action potential is occurring, acetylcholine, which began the process, is broken down to acetic acid and choline by enzymes (acetylcholinesterase, or AchE) present on the sarcolemma (see Figure 6.5c). For this reason, a single nerve impulse produces only one contraction. This prevents continued contraction of the muscle cell in the absence of additional nerve impulses. The muscle cell relaxes until stimulated by the next round of acetylcholine release.

This series of events is explained more fully on pp. 231–233 in the discussion of nerve physiology, but perhaps it would be helpful to compare this to some common event, such as lighting a match under a small dry twig (Figure 6.6). The charring of the twig by the flame can be compared to the change in membrane permeability that allows sodium ions into the cell. When that part of the twig becomes hot enough (when enough sodium ions have entered the cell), the twig will suddenly burst into flame, and the flame will consume the twig (the action potential will be conducted along the entire length of the sarcolemma). The events that return the cell to its resting state include (1) diffusion of potassium ions (K^+) out of the cell, and (2) operation of the sodium-potassium pump, the active transport mechanism that moves the sodium and potassium ions back to their initial positions.

Mechanism of Muscle Contraction: The Sliding Filament Theory

What causes the filaments to slide? This question brings us back to the myosin heads that protrude all around the ends of the thick filaments. When muscle fibers are activated by the nervous system as just described, the myosin heads attach to binding sites on the thin filaments, and the sliding begins. Energized by ATP, each cross bridge attaches and detaches several times during a contraction, acting much like a tiny oar to generate tension and pull the thin filaments toward the center of the sarcomere. As this event occurs simultaneously in sarcomeres throughout the cell, the muscle cell shortens (Figure 6.7). The attachment of the myosin cross bridges to actin requires calcium ions (Ca^{2+}). So where does the calcium come from? As indicated in Figure 6.5b, action potentials (black arrows) pass deep into the muscle cell along membranous tubules that fold inward from the sarcolemma. Inside the cell, the action potentials stimulate the sarcoplasmic reticulum to release calcium ions into the cytoplasm. The calcium ions trigger the binding of myosin to actin initiating filament sliding. This sliding process and the precise role of calcium are depicted in Figure 6.8. When the action potential ends, calcium ions are immediately reabsorbed into the SR storage areas, and the muscle cell relaxes and settles back to its original length. This whole series of events takes just a few thousandths of a second.

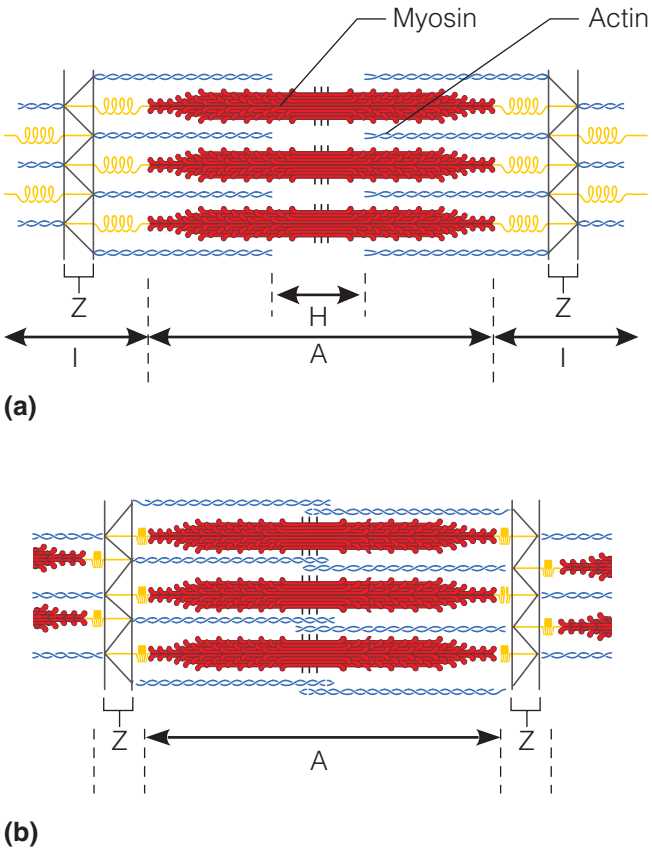

FIGURE 6.7 Diagrammatic views of a sarcomere. (a) Relaxed; **(b)** fully contracted. Notice that in the contracted sarcomere, the light H zone in the center of the A band has disappeared, the Z discs are closer to the thick filaments, and the I bands have nearly disappeared. The A bands move closer together but do not change in length.

Contraction of a Skeletal Muscle as a Whole

Graded Responses

In skeletal muscles, the "all-or-none" law of muscle physiology applies to the *muscle cell,* not to the whole muscle. It states that a muscle cell will contract to its fullest extent when it is stimulated adequately; it never partially contracts. However, skeletal muscles are organs that consist of thousands of muscle cells, and they react to stimuli with **graded responses,** or different degrees of shortening. In general, graded muscle contractions can be produced two ways: (1) by changing the *frequency* of muscle stimulation, and (2) by changing the *number* of muscle cells being stimulated. A muscle's response to each of these is briefly described next.

Muscle Response to Increasingly Rapid Stimulation

Although **muscle twitches** (single, brief, jerky contractions) sometimes occur as a result of certain nervous system problems, this is *not* the way our muscles normally operate. In most types of muscle activity, nerve impulses are delivered to the muscle at a very rapid rate—so rapid that the muscle does not get a chance to relax completely between stimuli. As a result, the effects of the successive contractions are "summed" (added) together, and the contractions of the muscle get stronger and smoother. When the muscle is stimulated so rapidly that no evidence of relaxation is seen and the contractions are completely smooth and sustained, the muscle is said to be in **fused,** or **complete,**