AN INTRODUCTION TO THE MUSCULAR SYSTEM AND ITS DISORDERS

The muscular system includes more than 700 skeletal muscles that are directly or indirectly attached to the skeleton by tendons or aponeuroses. The muscular system produces movement across joints as the contractions of skeletal muscles pull on the attached bones. Muscular activity does not always result in movement, however; it can also help stabilize skeletal elements and prevent movement. Skeletal muscles are also important in guarding entrances and exits of internal passageways, such as those of the digestive, respiratory, urinary, or reproductive systems, and in generating heat to maintain a stable body temperature.

THE MUSCULAR SYSTEM

THE DIAGNOSIS OF MUSCULAR DISORDERS

SIGNS OF MUSCULAR DISORDERS

Skeletal muscles normally contract only under the command of the nervous system. For this reason, clinical observation of muscular activity may provide indirect information about the nervous system, as well as direct information about the muscular system. The assessment of a patient's facial expressions, posture, speech, and gait can be an important part of a physical examination. Classical signs of muscle disorders include the following:

- **Gower’s sign** is a distinctive method of standing up from a sitting or lying position on the floor. This sign is characteristic of young children with muscular dystrophy (p. 67). They move from a sitting position to a standing position by pushing the trunk off the floor with the hands and then moving the hands to the knees. The hands are then used as braces to force the body into the standing position. The extra support is necessary, because the pelvic muscles are too weak to swing the weight of the trunk over the legs.

- **Ptosis** is a drooping of the upper eyelid. It may be seen in myasthenia gravis (p. 68), botulism (p. 68), and myotonic dystrophy (p. 67), or it may follow damage to cranial nerve III, which innervates the levator palpebrae superioris muscle of the eyelid.

- **A muscle mass**—an abnormal dense region within a muscle—is sometimes seen or felt in a skeletal muscle. A muscle mass results from torn muscle or tendon tissue, a hematoma, a parasitic infection such as trichinosis (p. 66), or bone deposition, as in myositis ossificans (p. 54).

- Abnormal contractions may indicate problems with the muscle tissue or its innervation. Muscle spasticity exists when a muscle has excessive tone. A muscle spasm is a sudden, strong, and painful involuntary contraction.

- **Muscle flaccidity** exists when the relaxed skeletal muscle appears soft and loose and its contractions are very weak or absent.

- **Muscle atrophy** is skeletal muscle deterioration, or wasting, due to disuse, immobility, or interference with the normal innervation.

- Abnormal patterns of muscle movement, such as tics, choreiform movements, or tremors, and muscular paralysis are generally caused
by nervous system disorders. We will describe these movements further in sections dealing with abnormal nervous system function.

**SYMPTOMS OF MUSCULAR DISORDERS**

Two common symptoms of muscular disorders are pain and weakness in the affected skeletal muscles. Possible causes of muscle weakness are diagrammed in Figure 33.

Possible causes of muscle pain include the following:

- **Muscle trauma.** Examples of traumatic injuries to a skeletal muscle include a laceration, a deep bruise or crushing injury, a muscle tear, and a damaged tendon.
- **Muscle infection.** Skeletal muscles can be infected by viruses, as in some forms of myositis (muscle inflammation), or colonized by parasitic worms, such as those responsible for trichi-
nosity (p. 66). These types of infections generally produce pain that is restricted to the affected muscles. Diffuse muscle pain can develop in the course of other infectious diseases, such as influenza.

- **Related problems with the skeletal system.** Muscle pain can result from skeletal problems such as arthritis (p. 60–61) or a sprained ligament near the point of muscle origin or insertion.
- **Problems with the nervous system.** Muscle pain can be related to the inflammation of sensory neurons or the stimulation of pain pathways in the central nervous system (CNS).

Muscle strength can be evaluated by applying an opposite force against a specific action. For example, an examiner might exert a gentle extending force on a patient's forearm while asking the patient to flex the elbow. Because the muscular and nervous systems are so closely interrelated, a single symptom, such as muscle weakness, can have a variety of causes (Figure 33). Muscle weakness can also develop as a consequence of a condition that affects the entire body, such as anemia or starvation.

### DISRUPTION OF NORMAL MUSCLE ORGANIZATION

A variety of disorders are characterized by a disruption in the structural organization of skeletal muscles. We will consider only a few representative examples: necrotizing fasciitis, characterized by a breakdown in the connective tissues of skeletal muscles; trichinosis, characterized by the colonization of muscles by parasites; and fibromyalgia and chronic fatigue syndrome, two muscle disorders of uncertain origin.

### NECROTIZING FASCITIS

Several bacteria produce enzymes such as hyaluronidase or cysteine protease. Hyaluronidase breaks down hyaluronic acid and disassembles the associated proteoglycans. Cysteine protease breaks down connective-tissue proteins. The bacteria that produce these enzymes are dangerous, because they can spread rapidly by liquefying the matrix and dissolving the intercellular cement that holds epithelial cells together. The *streptococci* are one group of bacteria that secrete both of these enzymes. *Streptococcus A* bacteria are involved in many human diseases, most notably "strep throat," an infection of the pharynx. In most cases, the immune response is sufficient to contain and ultimately defeat these bacteria before extensive tissue damage has occurred.

However, tabloid newspapers have a field day with stories of "killer bugs" and "flesh-eating bacteria." The details are horrific: Minor cuts become major open wounds, and interior connective tissues dissolve. This condition is called necrotizing fasciitis. Untreated, it is fatal. Even with rapid diagnosis, aggressive surgical
TABLE 17  Examples of Tests Used in the Diagnosis of Muscle Disorders

<table>
<thead>
<tr>
<th>Diagnostic Procedure</th>
<th>Method and Result</th>
<th>Representative Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle biopsy</td>
<td>Removal of a small amount of affected muscle tissue</td>
<td>Identifies histologic muscle disease; also used to detect cyst formation or larvae to diagnose trichinosis.</td>
</tr>
<tr>
<td>Electromyography (EMG)</td>
<td>Insertion of a probe that transmits measurements of electrical activity in contracting muscles</td>
<td>Abnormal EMG readings occur in disorders such as myasthenia gravis, amyotrophic lateral sclerosis (ALS), and muscular dystrophy.</td>
</tr>
<tr>
<td>MRI</td>
<td>Standard MRI</td>
<td>Detects muscle damage and associated soft-tissue abnormalities.</td>
</tr>
<tr>
<td>Laboratory Test</td>
<td>Normal Values in Blood Plasma or Serum</td>
<td></td>
</tr>
<tr>
<td>Aldolase</td>
<td>Adults: less than 8 U/L 22–59 mU/L (51 units)</td>
<td>Elevated levels occur in muscular dystrophy but not in myasthenia gravis or multiple sclerosis.</td>
</tr>
<tr>
<td>Aspartate aminotransferase (AST or SGOT)</td>
<td>Adults: 7–40 U/L  Children: 15–55 U/L</td>
<td>Elevated levels occur in some muscle diseases and can occur after exercise.</td>
</tr>
<tr>
<td>Creatine phosphokinase (CPK or CK)</td>
<td>Adults: 30–180 IU/L</td>
<td>Elevated levels occur in muscular dystrophy and myositis or after extreme exercise.</td>
</tr>
<tr>
<td>Lactate dehydrogenase (LDH)</td>
<td>Adults: 100–190 U/L</td>
<td>Elevated levels occur in some muscle diseases and in lactic acidosis.</td>
</tr>
<tr>
<td>Electrolytes</td>
<td></td>
<td>Deoxygenation of potassium can cause muscle weakness.</td>
</tr>
<tr>
<td>Potassium</td>
<td>Adults: 3.5–5.0 mEq/L</td>
<td>Decreased calcium levels can cause muscle tremors and tetany; increased levels can cause muscle flaccidity.</td>
</tr>
<tr>
<td>Calcium</td>
<td>Adults: 8.5–10.5 mg/dl</td>
<td></td>
</tr>
</tbody>
</table>

The rapid development of extreme pain in an otherwise minor wound is one warning symptom.

TRICHINOSIS

Trichinosis (trik-i-NO-sis; trichos, hair + nosos, disease) results from infection by the parasitic nematode Trichinella spiralis. Symptoms include diarrhea, weakness, and muscle pain and are caused by the invasion of skeletal muscle tissue by larval worms, which create small pockets within the perimysium and endomysium (Figure 35a). Muscles of the tongue, eyes, diaphragm, chest, and legs are most often affected.

Larvae are common in the flesh of pigs, horses, dogs, and other mammals. The larvae are killed when the meat is cooked; people are most often exposed by eating undercooked infected pork. Once eaten, the larvae mature within the human intestinal tract, where they mate and produce eggs. The new generation of larvae then migrates through the body tissues to reach the muscles, where they complete their early development. The migration and subsequent settling produce a generalized achingness, muscle and joint pain, and swelling in infected tissues. An estimated 1.5 million Americans carry Trichinella in their muscles, and up to 300,000 new infections occur each year. The mortality rate for people who have symptoms severe enough to require treatment is approximately 1 percent.

FIBROMYALGIA AND CHRONIC FATIGUE SYNDROME

Fibromyalgia (-algia, pain) is a disorder that has formally been recognized only since the mid-1980s. Although first described in the early 1800s, the condition is still somewhat controversial, because the symptoms cannot be definitively linked to any anatomical or physiological abnormalities. However, physicians now recognize a distinctive pattern of symptoms with the diagnostic criteria of widespread musculoskeletal pain for 3 months or more, and tenderness in 11 or more of 18 specific tender points. Sleep disorders, depression, and irritable bowel syndrome also occur alongside fibromyalgia.

Fibromyalgia may be the most common musculoskeletal disorder affecting women under 40 years of age; from 3 to 6 million individuals in the United States may have this condition. The four most common tender points are (1) the medial surface of the knee, (2) the area distal to the lateral epicondyle of the humerus, (3) the area near the external occipital crest of the skull, and (4) the junction between the second rib and its costal cartilage. An additional clinical criterion is that the pains and stiffness cannot be explained by other mechanisms.

Most of the symptoms mentioned could be attributed to other problems. For example, chronic depression can, by itself, lead to
fatigue and poor-quality sleep. As a result, the pattern of tender points is the diagnostic key to fibromyalgia. This symptom distinguishes fibromyalgia from chronic fatigue syndrome (CFS). The current symptoms accepted as a definition of CFS include (1) a sudden onset, generally following a viral infection, (2) disabling fatigue, (3) muscle weakness and pain, (4) sleep disturbance, (5) fever, and (6) enlargement of cervical lymph nodes. Roughly twice as many women as men are diagnosed with CFS.

Attempts to link either fibromyalgia or CFS to a viral infection, adrenal gland dysfunction, or to some physical or psychological trauma have not been successful, and the causes remain unknown. For both conditions, treatment is at present limited to relieving symptoms when possible. For example, anti-inflammatory medications may help relieve pain, antidepressant medications may improve sleep patterns and reduce depression, and exercise programs may help maintain a normal range of motion. Reassurance that fibromyalgia is not progressive, crippling, or life-threatening may help sufferers.

THE MUSCULAR DYSTROPHIES

The muscular dystrophies (DIS-trö-fiz) are inherited diseases caused by a mutation in one of the many genes that affect muscle function. Progressive muscle weakness and deterioration occurs with variable severity depending on the type of mutation. One of the most common and best understood conditions is Duchenne's muscular dystrophy (DMD), which may be inherited from the mother or, in 30 percent of cases, from spontaneous mutation. Symptoms of this form of muscular dystrophy start in childhood, commonly between the ages of 3 and 7. The condition is caused by mutation of the DMD gene, which codes for the protein dystrophin. This gene is located on the X chromosome and generally affects males (it can occur in females only if a very rare form of chromosome duplication can occur). DMD occurs at an incidence of roughly 30 per 100,000 male births. Having only 0–5 percent of the normal amount of dystrophin in muscle fibers, these children develop progressive muscular weakness, and require wheelchairs by age 8 to 12. Most individuals die before age 20, due to respiratory paralysis or cardiac problems. Skeletal muscles are primarily affected, although for some reason the facial muscles continue to function normally. In later stages of the disease, the facial muscles and cardiac muscle tissue may also become involved.

Dystrophin is a large protein that attaches thin filaments of the sarcomeres to an anchoring protein on the sarcolemma, providing mechanical strength to the muscle fiber and connecting the myofibrils to the sarcolemma. In children with DMD, calcium channels remain open for an extended period, and calcium levels in the sarcoplasm rise to the point at which key proteins denature. Inflammation occurs and the muscle fiber then degenerates resulting in elevated plasma levels of the muscle enzyme creatinine phosphokinase. Steroid treatment slows progression for up to three years at the price of significant side effects. Rats with a form of DMD treated by various forms of gene and stem cell therapy have sometimes improved, giving hope that an effective treatment may be possible.

Women carrying the defective gene are asymptomatic, but each of their sons will have a 50 percent chance of developing DMD. Prenatal genetic testing allows for carrier detection and fetal diagnosis of DMD by 8 weeks gestation.

MYOTONIC DYSTROPHY

Myotonic dystrophy is a form of muscular dystrophy that occurs in the United States with an incidence of 13.5 per 100,000 population. Symptoms may develop in infancy, but more commonly they develop after puberty. As with other forms of muscular dystrophy, adults developing myotonic dystrophy experience a gradual reduction in muscle strength and control. Problems with other systems, especially the cardiovascular and digestive systems, typically develop. There is no effective treatment.
The inheritance of myotonic dystrophy is unusual, because children of an individual with this condition commonly develop symptoms that are more severe than those of the parent. The increased severity of the condition appears to be related to the presence of multiple copies of a specific gene on chromosome 19. For some reason, the nucleotide sequence of that gene gets repeated several times, and the number can increase from generation to generation. This phenomenon has been called a "genetic stutter." The greater the number of copies, the more severe are the symptoms. It is not known why the stutter develops or how the genetic duplication affects the severity of the condition. Evidence indicates that the extra nucleotides interfere in some way with the transcription of an adjacent gene involved with the control of muscle tone.

**PROBLEMS WITH THE CONTROL OF MUSCLE ACTIVITY**

Another group of disorders interferes with normal neuromuscular communication by affecting either the nerve's ability to issue commands or the muscle's ability to respond. Anything that interferes with neural function or with excitation-contraction coupling will cause muscular paralysis. Three examples are worth noting:

1. **Botulism** results from the consumption of canned or smoked foods contaminated with a bacterial toxin. The toxin prevents the release of ACh at the synaptic terminals, leading to a potentially fatal muscular paralysis.

2. The progressive muscular paralysis of **myasthenia gravis** results from the loss of ACh receptors at the junctional folds. The primary cause is a misguided attack on the ACh receptors by the immune system. Genetic factors play a role in predisposing individuals to this condition.

3. The motor paralysis caused by **polio** is the result of viral damage to motor neurons.

**BOTULISM**

*Botulinos* (bot-ú-LI-nus) toxin prevents the release of acetylcholine (ACh) at synaptic terminals, thereby producing a severe and potentially fatal paralysis of the skeletal muscles. A case of botulinal poisoning is called **botulism**. The toxin is produced by the bacterium *Clostridium botulinum*, which feeds on oxygen to grow and reproduce. Because this bacterium can live quite well in a sealed can or jar, most cases of botulism are linked to improper canning or storing procedures, followed by failure to cook the food adequately before it is eaten. Home-canned tuna or beets, smoked fish, and cold soups are foods most commonly linked to botulism. Boiling for a half-hour destroys both the toxin and the bacteria.

Symptoms generally begin 12–36 hours after a contaminated meal is eaten. The initial symptoms are typically disturbances in vision, such as double vision or a painful sensitivity to bright light. These symptoms are followed by other sensory and motor problems, including incoordinated speech and an inability to stand or walk. Roughly half of botulism patients experience intense nausea and vomiting. These symptoms persist for days to weeks, followed by a gradual recovery; some patients are still in recovery after a year.

The major risk of botulism poisoning is respiratory paralysis and death by suffocation. Treatment is supportive: bed rest, observation, and, if necessary, the use of a mechanical respirator. In severe cases, an antitoxin and drugs that promote the release of ACh, such as guanidine hydrochloride, may be administered. The overall mortality rate in the United States is about 10 percent.

A commercial form of botulinum toxin, "Botox," is injected into selected muscles to treat problems such as spasms of the eyelid and neck muscles or unequal extra-ocular muscle strength in strabismus (crossed eyes). The effect is temporary and may need to be repeated. In recent years, Botox treatments have also been increasingly used by aging baby boomers to suppress facial wrinkles.

**MYASTHENIA GRAVIS**

Myasthenia gravis (mi-as-THE-né-uh GRA-vis) is characterized by a general muscular weakness that tends to be most pronounced in the muscles of the arms, head, and chest. The first symptom is generally a weakness of the eye muscles and drooping eyelids. Facial muscles are commonly weak as well, and the individual develops a peculiar smile known as the "myasthenic snarl." As the disease progresses, weakness of the pharynx leads to problems with chewing and swallowing, and holding the head upright becomes difficult.

The muscles of the upper chest and upper limbs are next to be affected. All the voluntary muscles of the body may ultimately be involved. Severe myasthenia gravis produces respiratory paralysis, with a mortality rate of 5–10 percent. However, the disease does not always progress to such a life-threatening stage. For example, roughly 20 percent of people with the disease experience no symptoms other than eye problems.

The condition results from a decrease in the number of ACh receptors on the motor end plate. Before the remaining receptors can be stimulated enough to trigger a strong contraction, the ACh molecules are destroyed by cholinesterase. As a result, muscular weakness develops.

The primary cause of myasthenia gravis appears to be a malfunction of the immune system. Roughly 70 percent of individuals with myasthenia gravis have an abnormal thymus, an organ involved with the maintenance of normal immune function. In myasthenia gravis, the immune response attacks the ACh receptors of the motor end plate as if they were foreign proteins. For unknown reasons, 1.5 times as many women as men are affected. The typical age at onset is 20–30 for women, versus over 60 for men. Estimates of the incidence of this disease in the United States range from 2 to 10 cases per 100,000 population.

One approach to therapy involves the administration of drugs, such as *neostigmine*, that are termed **cholinesterase inhibitors**. As their name implies, these compounds are enzyme inhibitors; they tie up the active sites on the enzyme where cholinesterase...
normally binds ACh. With cholinesterase activity reduced, the concentration of ACh at the synapse can rise enough to stimulate the surviving receptors and produce muscle contraction. Corticosteroid therapy is typically beneficial, as is surgical removal of the thymus.

**POLIO**

Because skeletal muscles depend on their motor neurons for stimulation, disorders that affect the nervous system can have an indirect effect on the muscular system. Polio is caused by the poliovirus, a virus that does not produce clinical symptoms in roughly 95 percent of infected individuals. The virus produces variable symptoms in the remaining 5 percent. Some individuals develop a nonspecific illness resembling the flu. Other individuals develop a brief meningitis (p. 83), an inflammation of the protective membranes that surround the CNS. In still another group of people, the virus attacks somatic motor neurons in the CNS. When those neurons are destroyed, the lack of motor innervation causes paralysis and progressive atrophy of the dependent muscles.

In this third form of the disease, the individual develops a fever 7–14 days after infection. The fever subsides, but recurs roughly a week later, accompanied by muscle pain, cramping, and paralysis of some or all muscles in one or more limbs. Respiratory paralysis may also occur, and the mortality rate of this form of polio is 2–5 percent in children and 15–30 percent in adults. If the individual survives, some degree of recovery generally occurs over a period of up to six months.

For unknown reasons, the survivors of paralytic polio may develop progressive muscular weakness in muscles not previously affected 20–30 years after the initial infection. This postpolio syndrome is characterized by fatigue, muscle pain and weakness, and, in some cases, muscle atrophy. There is no treatment for the condition, although rest seems to help.

Polio has been almost completely eliminated from the U.S. population due to a successful immunization program. In 1954, 18,000 new cases occurred in the United States; there were 8 in 1976, and none since 1994. The World Health Organization reports that polio has been eradicated from the entire Western Hemisphere. Unfortunately, many parents today refuse to immunize their children against the poliovirus, because they assume that the disease has been “conquered.” Failure to immunize is a mistake, because (1) there is still no cure for polio, (2) the virus remains in the environment in many areas of the world, and (3) up to 38 percent of children aged 1–4 years have not been immunized. A major epidemic could therefore develop very quickly if the virus were brought into the United States from another part of the world.

For years, the vaccine that has been used is the oral Sabin vaccine, preferred for its ease of administration and better immune stimulation than the injectable vaccine. However, unlike the injected vaccine, the oral vaccine carries a 1 in 1 million risk that the immunized person will develop polio. In 1996, the Centers for Disease Control and Prevention (CDC) recommended the use of either a combination of injected and oral vaccines or the injected vaccine alone. By 2000, polio had become so rare worldwide that the CDC recommended only the injected vaccine for infants in the U.S.

**DELAYED ONSET MUSCLE SORENESS**

You have probably experienced muscle soreness the day after a period of physical exertion (Figure 36a). Considerable controversy exists over the source and significance of this pain, which is known as delayed onset muscle soreness (DOMS) and has several interesting characteristics:

- DOMS is distinct from the soreness you experience immediately after you stop exercising. The initial short-term soreness is probably related to the biochemical events associated with muscle fatigue.
- DOMS generally begins several hours after the exercise period ends and may last 3 or 4 days.
- The amount of DOMS is highest when the activity involves eccentric contractions (where a muscle elongates despite producing tension). Activities dominated by concentric or isometric contractions produce less soreness.
- Levels of CPK and myoglobin are elevated in the blood, indicating damage to muscle cell membranes. The nature of the activity (eccentric, concentric, or isometric) has no effect on these levels, nor can the levels be used to predict the degree of soreness experienced.

Three mechanisms have been proposed to explain DOMS:

1. Small tears may exist in the muscle tissue, leaving muscle fibers with damaged membranes. The sarcolemma of each damaged muscle fiber permits the loss of enzymes, myoglobin, and other chemicals that may stimulate nearby pain receptors.
2. The pain may result from muscle spasms in the affected skeletal muscles. In some studies, stretching the muscle involved after exercise can reduce the degree of soreness.
3. The pain may result from tears in the connective tissue framework and tendons of the skeletal muscle.

Some evidence supports each of these mechanisms, but it is unlikely that any one tells the entire story. For example, muscle fiber damage is certainly supported by biochemical findings, but if that

- **FIGURE 36**

Delayed Onset Muscle Soreness. A rigorous workout can cause lingering pain whose origins are uncertain.
were the only factor, the type of activity and level of circulating enzymes would be correlated with the level of pain experienced and such is not the case.

**POWER, ENDURANCE, AND ENERGY RESERVES**

Figure 37 compares the power/endurance curves for anaerobic and aerobic activities. The first half minute of peak activity is totally supported by the mobilization of ATP and CP (creatine phosphate) reserves (Figure 37a). Thereafter roughly two-thirds of the energy requirements of skeletal muscles operating at peak activity levels are met via glycolysis, with associated lactic acid generation.

At modest activity levels, a skeletal muscle can rely on aerobic respiration to provide ATP. At peak levels of activity, the muscle relies primarily on anaerobic metabolism. The level of activity at which the muscle must begin relying on anaerobic metabolism to meet its energy demands is called the anaerobic threshold. If energy demands are kept below the anaerobic threshold, muscular activity can be continued until nutrient sources are exhausted. In a trained athlete, muscle fatigue may not occur for several hours (Figure 37b).

**WHAT'S NEW IN ANATOMY?**

Like most people, you might assume that every anatomical structure in the human body was described centuries ago, and that nothing is "new" in the field of anatomy. Many people were surprised in 1996, however, when anatomical researchers at the University of Maryland documented the existence of a previously unknown skeletal muscle. This "new" muscle, named the sphenomandibularis muscle, extends from the lateral surface of the sphenoid to the mandible (Figure 38). The sphenomandibularis muscle assists the muscles of mastication.

The research began with a computer analysis of the Virtual Human database, a digitized photographic atlas of cross-sectional anatomy. That initial work was then supported by careful cadaver dissections. Although the discovery of the sphenomandibularis muscle remains controversial (it may have been described previously as a portion of the temporalis muscle), it is a good example of how modern technologies are providing new perspectives on the human body.

**MUSCULAR SYSTEM DISORDERS**

**HERNIAS**

When the abdominal muscles contract forcefully, pressure in the abdominopelvic cavity can increase dramatically. That pressure is applied to internal organs. If the individual exhales at the same time, the pressure is relieved because the diaphragm can move upward as the lungs collapse. But during vigorous isometric exercises or when lifting a weight while holding one's breath, pressure in the abdominopelvic cavity can rise to 106 kg/cm², roughly 100 times the normal pressure. A pressure that high can cause a variety of problems, including hernias. A hernia develops when a viscerum organ or part of an organ protrudes abnormally through an opening in a surrounding muscular wall or partition. There are

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*FIGURE 37*

Muscular Performance and Endurance. (a) At peak levels of activity, skeletal muscles rely primarily on glycolysis for ATP production, with associated lactic acid production. Initial burst activity is supported by ATP and CP reserves. Muscles operating at peak levels fatigue rapidly. (b) Muscular activity can continue for extended periods when ATP demands are kept below the anaerobic threshold.
trapped or twisted, surgery may be required to prevent serious complications. Inguinal hernias are not always caused by unusually high abdominal pressures; injuries to the abdomen or inherited weakness or distensibility of the canal can have the same effect.

The esophagus and major blood vessels pass through openings in the diaphragm, the muscle that separates the thoracic and abdomino-pelvic cavities. In a diaphragmatic hernia abdominal organs slide into the thoracic cavity. If entry is through the esophageal hiatus, the passageway used by the esophagus, a hiatal hernia (hi-
A-tal; hiatus, a gap or opening) exists. The severity of the condition depends on the location and size of the herniated organ or organs. Hiatal hernias are very common, and most go unnoticed, although they may increase the severity of gastric acid entry into the esophagus (gastroesophageal reflux disease, or GERD, commonly known as heartburn). Radiologists see them in about 30 percent of individuals whose upper gastrointestinal tracts are examined with barium-contrast techniques.

When clinical complications other than GERD develop, they generally do so because abdominal organs that have pushed into the thoracic cavity are exerting pressure on structures or organs there. Like inguinal hernias, a diaphragmatic hernia can result from congenital factors or from an injury that weakens or tears the diaphragm. If abdominal organs occupy the thoracic cavity during fetal development, the lungs may be poorly developed at birth.

**SPORTS INJURIES**

Sports injuries affect amateurs and professionals alike. A five-year study of college football players indicated that 73.5 percent experienced mild injuries, 21.5 percent moderate injuries, and 11.6 percent severe injuries during their playing careers. Contact sports are not the only activities that show a significant injury rate: A study of 1650 joggers running at least 27 miles a week reported 1819 injuries in a single year.

Muscles and bones respond to increased use by enlarging and strengthening. Poorly conditioned individuals are therefore more likely than people in good condition to subject their bones and muscles to intolerable stresses. Training is also important in minimizing the use of antagonistic muscle groups and in keeping joint movements within the intended ranges of motion. Planned warm-up exercises before athletic events stimulate circulation, improve muscular performance and control, and help prevent injuries to muscles, joints, and ligaments. Stretching exercises after an initial warm-up will stimulate blood flow to muscles and help keep ligaments and joint capsules supple. Such conditioning extends the range of motion and may prevent sprains and strains when sudden loads are applied.

Dietary planning can also be important in preventing injuries to muscles during endurance events, such as marathon running. Emphasis has commonly been placed on the importance of carbohydrates, leading to the practice of “carbohydrate loading” before a marathon. But while operating within aerobic limits, muscles also utilize amino acids extensively, so an adequate diet must include both carbohydrates and proteins.

Improved playing conditions, equipment, and regulations also play a role in reducing the incidence of sports injuries. Jogging shoes, ankle or knee braces, helmets, mouth guards, and body padding are examples of equipment that can be effective. The
substantial penalties now earned for personal fouls in contact sports have reduced the numbers of neck and knee injuries.

Several traumatic injuries common to those engaged in active sports can also affect nonathletes, although the primary causes may be very different. A partial listing of activity-related conditions includes the following:

- **Bone bruise**: bleeding within the periosteum of a bone
- **Bursitis**: an inflammation of the bursae at joints
- **Muscle cramps**: prolonged, involuntary, and painful muscular contractions
- **Sprains**: tears or breaks in ligaments or tendons
- **Strains**: tears in muscles
- **Stress fractures**: cracks or breaks in bones subjected to repeated stresses or trauma
- **Tendinitis**: an inflammation of the connective tissue surrounding a tendon

Finally, many sports injuries would be prevented if people who engage in regular exercise would use common sense and recognize their personal limitations. It can be argued that some athletic events, such as the ultramarathon, place such excessive stresses on the cardiovascular, muscular, respiratory, and urinary systems that these events cannot be recommended, even for athletes in peak condition.

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**INTRAMUSCULAR INJECTIONS**

Drugs are commonly injected through hollow needles into tissues rather than directly into the bloodstream (accessing blood vessels may be technically more complicated). An **intramuscular (IM) injection** introduces a fairly large amount of a drug, which will then enter the circulation gradually. The drug is introduced into the mass of a large skeletal muscle. Uptake is generally faster and accompanied by less tissue irritation than when drugs are administered **intradermally** or **subcutaneously** (injected into the dermis or subcutaneous layer, respectively). Depending on the size of the muscle, up to 5 ml of fluid may be injected at one time, and multiple injections are possible. A decision on the injection technique and the injection site is based on the type of drug and its concentration.

For IM injections, the most common complications involve accidental injection into a blood vessel or piercing of a nerve. The sudden entry of massive quantities of a drug into the bloodstream can have fatal consequences, and damage to a nerve can cause motor paralysis or sensory loss. Thus, the site of injection must be selected with care. Bulky muscles that contain few large vessels or nerves are ideal sites. The gluteus medius muscle or the posterior, lateral, superior part of the gluteus maximus muscle is commonly selected. The deltoid muscle of the arm, about 2.5 cm (1 in.) distal to the acromion, is another effective site. Probably most satisfactory from a technical point of view is the vastus lateralis muscle of the thigh; an injection into this thick muscle will not encounter vessels or nerves, but may cause pain later when the muscle is used in walking. This is the preferred injection site in infants before they start walking, as their gluteal and deltoid muscles are relatively small (Figure 40). The site is also used in elderly patients or others with atrophied gluteal and deltoid muscles.
Questions

1. Because of their interconnected nature, disorders of which system can be determined through clinical observation of muscular activity?
2. What are some of the classical signs of muscle disorders? Explain them.
3. What are some tests that might be done to help determine muscular disorders?
4. What are the symptoms of trichinosis? How do the larval worms cause the disease?
5. How is fibromyalgia distinguished from CFS?
6. What does the enzyme creatinine phosphokinase do? (It is not creatine phosphate!)
7. What is the function of Dystrophin? In DMD kids, what happens to the calcium channels.
8. Describe Botulism.
9. What causes polio? What are its symptoms? What are the issues with the vaccine?
10. What is DOMS? Why are the argued causes of DOMS currently?
11. What is anaerobic threshold?
13. What is the advantage of IM injections over intradermal or subcutaneous?
14. What are possible complications associated with IM injection?
15. List all the disorders/diseases mentioned in this reading and list their symptoms.