

Chapter 2

Classification of Injuries and Illnesses

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CHAPTER OBJECTIVES

After you have read this chapter, you will be able to understand:

- Various mechanisms of tissue injury
- The healing process, including the three phases of healing
- The signs and symptoms of injury and illness
- The differences between acute and chronic injuries
- The classifications of injuries and illnesses
- The microorganisms that cause illnesses

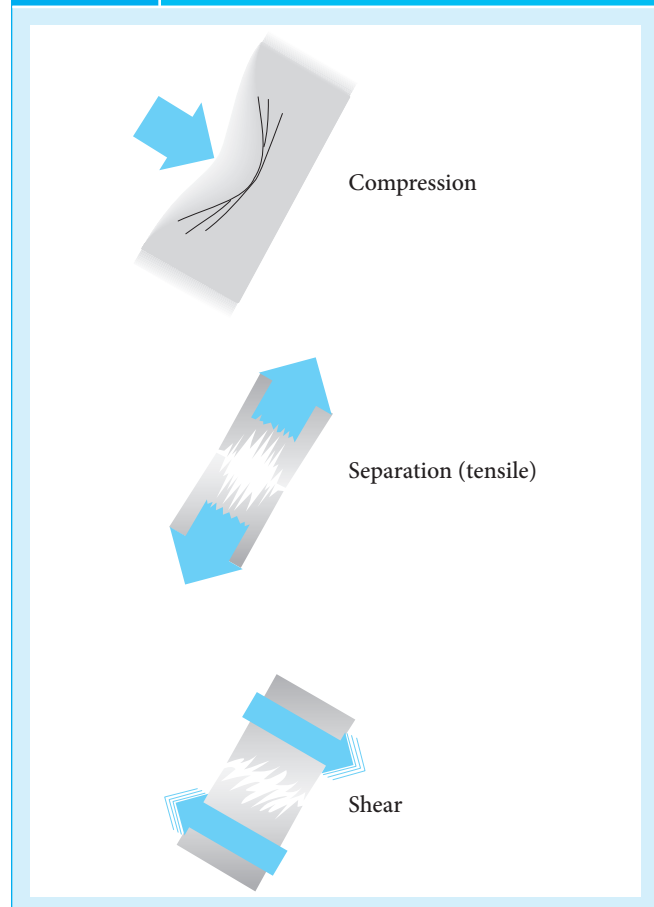
Numerous types of injuries and illnesses exist. An athlete can experience an injury or be overcome by an illness that can be minor or severely disabling. Whether an injured athlete has a broken bone, a muscular injury, or a skin infection, the body must heal in order to return to its original state. The healing process is a systematic means for tissue to respond to the injury and allow the body to resume normal function. This chapter explains the tissue response to injury and introduces you to the general classifications of injuries and illnesses. By understanding these general classifications, you will be able to identify various types of injuries and illnesses.

MECHANISM OF INJURY

Various mechanisms of injury exist. A mechanism of injury is the cause of the injury or the manner in which the injury occurs. Compression, separation (tensile), and shear forces are three very common mechanisms of injury for both soft tissue and bony injuries (**Figure 2-1**).

Figure 2-1

Compression, separation (tensile), and shear forces are mechanisms of injury.



Compression forces occur when the tissue is crushed between two or more objects. During a workout, a weightlifter drops a weight on her foot. Her foot is compressed between the weight and the floor. Consequently, she sustains a metatarsal fracture due to the compressive mechanism of injury.

Tissue separation, or tension, occurs when a structure is pulled apart from either one or both ends. The tissue is stretched beyond the anatomical limit, which is the maximal length the tissue can withstand, and as a result the tissue tears (Eaves, 2010). For example, an athlete twists his ankle in a manner that stretches a ligament. The ligament cannot withstand the force and the athlete sustains an ankle ligament tear, or sprain. The mechanism of injury in this example is a separation force placed on the tissue.

A shear force is applied to the body when one or more forces move across the tissues. If more than one shear force crosses the tissues, the forces typically move in opposite directions. If a soccer player's thigh is kicked by an opponent wearing cleats, the cleats move across and shear the skin. Consequently, a skin wound, such as an abrasion or laceration, can occur.

Regardless of the mechanism of injury, the extent or degree of injury is determined by the severity and intensity with which the force is applied. The size of the area affected also determines the severity of injury. The injury most likely will be more severe if a great amount of force or stress is applied over a small area versus a larger area. In a previous example, a weightlifter dropped a weight on her foot. If the weightlifter dropped the same amount of weight on her thigh, the force applied to the tissue would be less than the force applied to the foot, because the thigh is a larger area than the foot (Anderson, Hall, & Martin, 2000). Also, the speed of the force can cause varying severity of injury. If a baseball player is hit by a 95 mile per hour (mph) pitch versus a 40 mph pitch at the same bodily area thrown from the same distance, the severity of injury caused by the 95 mph pitch will be greater. As you can see, many factors come into play to determine the severity of injury.

Tips from the Field

Tip 1 Remember that each athlete has a different tolerance to and perception of pain. When you ask an athlete how much pain they are experiencing, ask them to rate their pain on a scale of 1 to 10, with 1 being the least pain they have ever experienced and 10 being the most pain they have ever experienced. After they have given you their rating, ask them to describe the circumstance or scenario in which they felt that level of pain before. This information can give you an idea of the severity of injury.

THE HEALING PROCESS

After an injury occurs to an athlete, the healing process begins. The healing process is imperative for an injured or ill athlete. This process is a means to repair the tissues, return the body back to its uninjured state, and restore the body's equilibrium. The three phases of the healing process are the inflammatory phase, the fibroblastic repair phase, and the maturation-remodeling phase (Bahr & Maehlum, 2004; Booher & Thibodeau, 2000; Pfeiffer & Mangus, 2012). These phases occur in a continuum, which means they overlap each other (Figure 2-2). The three phases do not have a rigid time frame when one phase ends and the subsequent phase begins. However, completion of each phase is imperative for the tissue to heal properly. When the healing process is complete, an athlete should be able to obtain normal, pre-injury function.

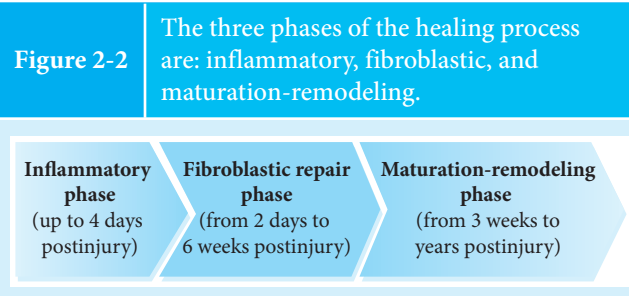
The Inflammatory Phase

The inflammatory phase is the first phase in the healing process for **acute injuries**. This phase can arguably be considered the most important phase. If the inflammatory phase does not properly follow an intended course of events, then the damaged tissue will not move on to the fibroblastic phase. This lack of progression can cause further injury, in particular **chronic injury**, which is discussed later in this chapter.

Acute injury Occurs when the body is suddenly afflicted by trauma or damage to its tissues, which consequently initiates inflammation.

Chronic injury Occurs when an injury is no longer acute, yet remains in the later inflammatory phase or early fibroblastic repair phase of the healing process.

The inflammatory phase begins at the time of injury. Immediately when tissue is damaged vasoconstriction occurs. Vasoconstriction is defined by the blood vessels' lumen, or opening, becoming narrowed and smaller in diameter. Vasoconstriction often takes place for an extremely short amount of time, typically 5 to 10 minutes (Prentice, 2011). During this time, blood flow is slowed to the injured area. After vasoconstriction occurs, vasodilation begins.



Vasodilation is when the blood vessels' openings become larger in diameter. The vasodilation occurs during the beginning stages of the inflammation phase. The damaged tissue; cells, such as white blood cells or mast cells; and enzymes release chemical mediators that cause the vasodilation (Prentice, 2011). Two chemical mediators that are seemingly detrimental to the athlete, yet important to the healing process, are histamine and bradykinin. Histamine causes vasodilation, which increases blood flow to the injured area. It also causes redness, an increased temperature at the injured site, and blood vessel permeability. Like histamine, bradykinin creates blood vessel permeability, and also causes pain at the injured site and surrounding areas (Pfeiffer & Mangus, 2012).

When the blood vessels are vasodilated and their walls are permeable, the damaged red and white blood cells and the plasma fluid in the blood exit the blood vessels. This plasma

Exudate Formed by plasma fluid and cells; it is located in the space outside of the blood vessels.

fluid and cells are called **exudate**. How much exudate is formed depends on factors such as the extent of injury, chemical mediators released, and blood vessel permeability (Prentice, 2010). Exudate accumulates in the space outside the blood vessels. The exudate becomes viscous and remains stagnant along the soft and bony tissues. Due to its viscosity, the exudate

Ecchymosis A black and blue, bruise-like color of the skin that can occur with internal bleeding.

creates the appearance of swelling and **ecchymosis** (Figure 2-3). As the exudate increases, the pressure increases at the injury site. The pressure causes cellular death due to lack of oxygenated blood flow to the area (Eaves, 2010), and pressure on nerve endings causes pain at the injury site. The exudate also prevents the veins from functioning properly. Usually the venous system brings waste, including carbon dioxide, damaged cells, and extracellular fluid, through the circulatory system to clean up the injured area after the trauma. However, the restoration cannot occur until other chemical mediators are released.

Chemical mediators that assist in returning the area to its pre-injury state are heparin, serotonin, leukotrienes, and prostaglandins (Prentice, 2011). Heparin is an anticoagulant, which helps make the exudate less viscous, making venous return possible. Serotonin helps the blood vessels to vasoconstrict and become less permeable. Consequently, the amount of fluid and cells exiting the blood vessels is minimized. Leukotrienes and prostaglandins both impede inflammation and decrease swelling.

At the end of the inflammatory response phase and in response to other chemical mediators, phagocytes go to the

Figure 2-3

The black and blue color seen at the inner thigh in this picture is called ecchymosis.



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injury site to debride the area. Phagocytes include cells such as leukocytes, neutrophils, and macrophages. They consume the waste materials, damaged cells, and foreign matter around the tissues (Pfeiffer & Mangus, 2012). Assisting the phagocytes are platelets, which are sticky substances that attach to the exposed collagen fibers on the blood vessels' walls. These platelets create a plug that helps limit the size of the inflamed area.

At this time, thromboplastin is released from the damaged cells. Thromboplastin initiates a series of events that causes the clot to be formed. The existence of thromboplastin makes prothrombin change to thrombin, which is the sticky substance comprising the clot (Prentice, 2011). A clot begins to form, which, like the platelet plug, limits the size of the inflamed area. The clot also prevents further blood flow to the injured area. As the clot forms, phagocytes continue to debride the area.

This phase of healing is characterized by specific signs and symptoms of inflammation. Signs of inflammation are characteristics that are physically observable. Symptoms of inflammation are characteristics that are not physically observable, but are subjective and reported by the athlete. Signs of inflammation include redness, swelling, increase in

tissue temperature, and loss of function (Figure 2-4). The predominant and most common symptom of inflammation that the athlete experiences is pain. As the inflammatory phase subsides and the second phase of healing begins, these signs and symptoms of inflammation notably subside. The inflammatory phase can take up to 4 days to complete before the fibroblastic repair phase commences (Bahr & Maehlum, 2004).

The Fibroblastic Repair Phase

Usually around the second day postinjury marks the end of the inflammatory phase. At this time, the inflammatory phase overlaps with the beginning of the fibroblastic repair phase. The fibroblastic repair phase has a longer duration than the inflammatory phase, and can take up to 6 weeks to complete. This phase of healing is characterized by a decrease in signs and symptoms of inflammation, continuation of phagocytic activity, fibroblastic activity, and formation of collagen tissue and a fragile scar.

By the time the fibroblastic repair phase begins, the signs and symptoms of inflammation have decreased. New capillaries form at the injury site, which brings oxygenated blood to the area, while phagocytes continue to rid the injured site of debris (Bahr & Maehlum, 2004). Consequently, pressure decreases, which in turn decreases pain and swelling at the site. With

decreased pain and swelling, the athlete is able and willing to move the area. This voluntary movement increases range of motion, or amount of available motion, at the injured area.

Fibroblasts are cells that have the ability to form collagen fibers. They are located in connective tissue and migrate to the injured area. The fibroblasts begin

Collagen A strong protein that comprises bone, soft tissue including skin, cartilage, and other connective tissues.

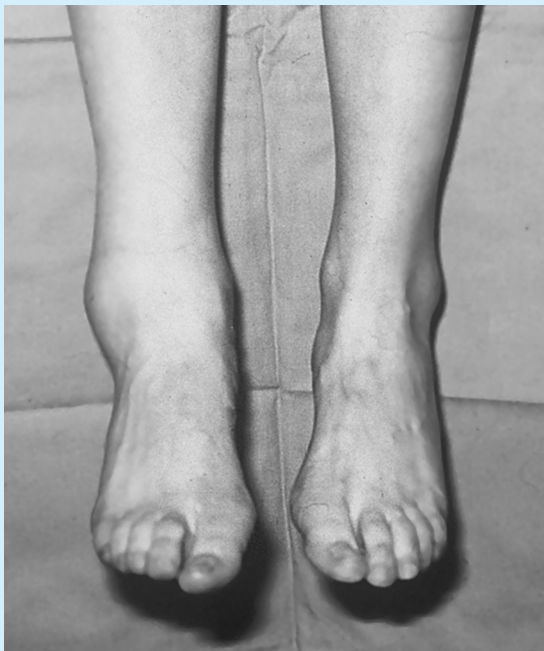
to create a loose meshwork of unorganized collagen at the injury site. The collagen creates a fragile scar at the site of the previously formed clot. This scar formation process is called fibroplasia (Booher & Thibodeau, 2000). Over weeks, the collagen begins to re-form and strengthen as the fibroblasts decrease in number, and thus ends the fibroblastic repair phase.

Tips from the Field

Tip 2 Athletes who have injuries in the fibroblastic repair phase of healing are able to participate in limited activities (with a physician's clearance note, of course). However, if they have any signs or symptoms of inflammation, or even soreness, before, during, or after activity, then their activity must be modified or reduced further. In some cases you may need to stop activity that entails that area (i.e., if it is a lower body injury, you can have your athlete perform upper body activities until the signs and symptoms subside).

Figure 2-4

Swelling is a common sign of inflammation.



Courtesy of AAOS.

The Maturation-Remodeling Phase

During the fibroblastic repair phase, collagen is laid down in a disorganized fashion, which creates a fragile scar. However, as stress is applied to the area, the collagen lines up parallel to how the stress is applied (Booher & Thibodeau, 2000). This organization of the collagen scar tissue fibers occurs in the beginning of the maturation-remodeling phase, which is the final phase of healing. It usually begins around 3 weeks postinjury, and overlaps the end of the fibroblastic repair phase. This final phase of healing is significantly longer than the fibroblastic repair phase and can take years to complete.

As the phase progresses, scar formation continues. Stress on the scar aligns the scar tissue fibers parallel to each other and the lines of stress. This stress creates more organized scar tissue. However, this stress must be applied in a manner that does not disrupt the scar. If too much stress exists, then scar disruption occurs and the healing process takes much longer than originally planned. Over time, the collagen fibers become more organized, and the scar continues to strengthen and decrease in size (Booher & Thibodeau, 2000).

BONE HEALING VERSUS SOFT TISSUE HEALING

Bone healing is similar to soft tissue healing in regard to the events that occur in the inflammatory phase; however, once the fibroblastic repair phase begins, bone healing is different than soft tissue healing. During the fibroblastic repair phase, osteoblasts from the surrounding bone move to the break, or fracture, in the bone. These osteoblasts, which are a type of fibroblast, form a callus at the injured site (**Figure 2-5**). The callus is a combination of collagen and cartilage, which fills in the break in the bone. Over time, as with soft tissue healing in the maturation-remodeling phase, this bony callus becomes hard and strong (Pfeiffer & Mangus, 2012). Stress is applied to assist the callus to properly form into bone. However, usually some immobilization of the fracture is necessary to promote proper healing. A physician determines whether the fractured bone should be immobilized for days, weeks, months, or not at all. The physician makes this determination depending on various factors including the severity of the fracture, secondary injuries and complications, bone(s) injured, and the athlete's age and health. With proper treatment of the fracture, the injured site will heal and become continuous with the surrounding bone.

SIGNS VERSUS SYMPTOMS

Every injury has its typical signs and symptoms. Signs of injury are physically observable characteristics of injury. Signs are also objective, which means that you are able to see a sign as another person would. The injured athlete's perception cannot change the signs of the injury that you see. Unlike signs

Figure 2-5 A bony callus forming at the site of a metatarsal fracture.



Reproduced with permission from Johnson TR, Steinbach LS (eds). Essentials of Musculoskeletal Imaging. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2004:623.

Figure 2-6 Deformity is a sign of a dislocation.



Courtesy of AAOS.

of injury, symptoms are characteristics of injury that are not physically observable. Instead, symptoms are subjective to the injured person. You cannot see the symptoms that an injured athlete experiences. Because you cannot see symptoms, you can only take the injured athlete's word that the symptom exists. Both signs and symptoms are equally important to determine what type of injury an athlete has sustained, and to consequently render the proper treatment.

Numerous common signs and symptoms of injury exist. Some typical signs include differences in tissue temperature, asymmetry, and malalignment as compared to the opposite, uninjured, or **contralateral** side; ecchymosis; swelling; deformity (**Figure 2-6**); redness; hives; **crepitus**; and loss of function. Some common symptoms include pain, soreness, fever, nausea, **tinnitus**, headache, and numbness. Signs and symptoms of injury are often the same for a multitude of injuries, which makes determining the athlete's injury or illness difficult.

Contralateral The opposite side of the body symmetrical to the injured side. If the injury is at the right knee, then the contralateral side is the left knee.

Crepitus A crackling or grating sensation that you and an athlete may feel during movement or palpation when a structure is injured.

Tinnitus Ringing in the ears.

ACUTE VERSUS CHRONIC INJURIES

An acute injury occurs when the body is suddenly afflicted by trauma or damage to its tissues. Consequently, acute injury initiates inflammation and promotes all inflammatory process events. Acute injuries have a specific, sudden mechanism of injury. An athlete can typically describe and pinpoint the exact time the injury occurred. For example, an athlete at We R Fit Gym who drops a weight on his foot can describe the

exact moment that he fractured his foot. His injury is acute because he had a specific, sudden mechanism or cause of injury. Likewise, immediately upon dropping the weight on his foot, the healing process, in particular the inflammatory phase, begins.

Chronic injuries are unlike acute injuries because with many chronic injuries the athlete cannot identify a specific, sudden mechanism of injury. Also, the signs and symptoms of chronic injuries develop more slowly than with acute injuries. For example, an athlete at We R Fit Gym complains of diffuse pain at both shoulders that has been increasing for 3 weeks. She cannot recall any specific time of injury when she immediately felt symptoms or noticed signs of inflammation. This scenario is very typical when an athlete has a chronic injury.

Another common reason why an injury is deemed chronic is due to the athlete having an acute injury and not receiving proper treatment for the injury. If acute injuries are not treated appropriately, they do not heal properly. The injury does not move through the healing process continuum as it should. Consequently, the injury becomes chronic. This process often occurs with mild to moderate injuries. For example, athletes with acute mild to moderate sprains and strains commonly learn to adapt to or ignore the associated discomfort and other inflammatory signs and symptoms. The athlete continues to participate in activities instead of obtaining proper treatment for the acute injury. In most cases this continued participation hinders the healing process and prolongs the signs and symptoms.

Consider the following scenario: During a Friday night championship game, a basketball player goes up for a lay-up and on her way down she lands with her foot on another player's foot. She inadvertently twists her ankle into inversion, which creates a mild sprain of her anterior talofibular ligament on the lateral side of her ankle. She experiences immediate pain and disability. Almost as quickly as the pain appears, it subsides and she is able to run down the court to play defense. Her team wins the game. She is so excited that she does not pay attention to her acutely injured ankle. The weekend arrives and she has mild ankle pain, swelling, and no loss of motion. She continues to perform her activities of daily living, which include Saturday morning basketball practice. The athlete decides not to see a physician for evaluation, nor does she provide any first aid treatment to her ankle. This scenario is typical for how an athlete's acute injury, due to no or improper treatment, can easily predispose an acute injury to become a chronic injury.

Another reason why an injury becomes chronic is due to an athlete's inability to recognize his or her injury when

experiencing a subacute or overuse injury. A subacute injury exhibits very minimal signs and symptoms as compared to an acute injury, and is usually caused by microtraumas to a body structure. Overuse injuries occur from repetitive motions, and over time the structure is damaged. Overuse injuries can begin as subacute injuries and become chronic injuries. Because chronic injuries do not have a specific, identifiable, sudden mechanism of injury, it is often difficult for an athlete to recognize his or her injury. Chronic injuries are noted as having a mechanism of injury that begins as subacute and occurs over time from repetitive use of the structure involved.

Consider another scenario: A baseball pitcher constantly throws overhead. This constant throwing puts a significant amount of stress on his shoulder musculature and tendons. After every practice and game he believes his shoulder soreness is due to high intensity throwing. He does not consider that his shoulder is injured. Therefore, he does not mention the soreness to his coach. Three weeks later, he goes to his coach and explains that on some days, during and after throwing, his shoulder "feels fine" and on other days his shoulder is "extremely painful." The athlete now complains of loss of motion and strength. The fibroblastic repair phase of healing occurs after the inflammation phase and 3 weeks into the healing process. However, the athlete is still experiencing symptoms of injury that are consistent with the inflammatory phase. He may not have experienced a sudden acute mechanism of injury, but instead he sustained small, subacute injuries that occurred over time. Unfortunately, now the baseball player has sustained a chronic shoulder injury.

Most chronic injuries fluctuate between the inflammatory and fibroblastic repair phases. These injuries have a difficult time progressing through the fibroblastic repair phase and reaching the maturation-remodeling phase. Often, for the chronic injury to heal properly a physician or qualified allied healthcare professional elicits minor trauma, such as friction massage, to the chronic injury site. This microtrauma precipitates an inflammatory response, and reverts the injury to the beginning of the inflammatory response phase. A chronic injury may take many months or even years to heal.

In many instances, chronic injuries can be avoided if athletes closely listen to their bodies and heed acute injury signs and symptoms. Educate your athletes to report any acute inflammatory signs and symptoms of injury to their physician immediately upon recognizing them. Even if the athlete experiences mild signs and symptoms, he or she must receive care to promote the healing process and attempt to avoid chronic injury.

Tips from the Field

Tip 3 When speaking with an athlete regarding their injury, in order to determine the mechanism of injury always ask the question, “What happened?,” even if you saw the injury occur. Obtaining the athlete’s subjective opinion and feelings regarding the injury is important. Do not assume you know exactly what happened and how the injury occurred because you witnessed it happening. On the same lines, to determine whether the injury is acute or chronic, ask the athlete, “When did it happen?” The answer to this question gives you a good idea as to the acute or chronic classification of the injury.

CLASSIFICATION OF INJURIES

The body is composed of numerous commonly injured anatomical structures. Likewise, various forces are placed on and created by these structures. Consequently, your athletes are predisposed to injury at some point in their lives. After reading this chapter, you will be able to classify what type of injury your athlete has experienced. Along with acute and chronic injuries, experts have classified injuries into two additional categories based on the tissue type involved. These categories are soft tissue injuries and bone injuries. Soft tissue and bone injuries are acute and chronic and further broken down into numerous classifications. Common soft tissue injuries include sprains, strains, tendinopathies, muscle spasms, contusions, bursitis, and skin wounds. A fracture is a common bone injury, while common joint injuries include dislocations and subluxations.

Soft Tissue Injuries

■ Sprain

A sprain is an injury to a ligament in which the ligamentous fibers are stretched or torn. A sprain typically is caused by a sudden, acute mechanism of injury such as a twisting (shear) or pulling apart (tension) of a joint. A joint is the articulation between two or more bones, which are held in close proximity to each other by ligaments. A ligament can be stretched or completely torn (ruptured); however, not all fibers in a single ligament are always ruptured. A single injured ligament may have intact, stretched, and torn fibers at the same time. As with other acute injuries, when a sprain occurs the healing process begins. The signs and symptoms of inflammation develop immediately after injury. During the healing process, scar tissue fills the space where the ligament tear occurred. The ligament and the scar tissue have plastic qualities. Plasticity means that the tissue does not have elasticity. Elasticity means that

the tissue can be deformed or stretched and return to its original length. Because a ligament has plastic qualities, when it is stretched the ligament remains at that length. This fact is the same for scar tissue laid down when a ligament heals. After injury, the damaged ligament and resultant scar tissue are not strong enough to withstand significant stress. Consequently, it is very common for an athlete to have subsequent injuries occur to the same ligament.

A sprain is classified by using terminology describing its degree of severity. In 1964, the American Medical Association (AMA) charged a Subcommittee on Classification of Sports Injuries with creating standardized athletic injury nomenclature. The Subcommittee finalized the content in 1966, and *Standard Nomenclature of Athletic Injuries* was published by the AMA soon thereafter (AMA, 1966). Since that time, the medical field has utilized this nomenclature as a standard for explaining the severity of injuries. Three degrees of severity for sprains exist—first-, second-, and third-degree. The severity of injury increases from first-degree being the least severe to third-degree being the most severe. With first-degree sprains, the ligaments are stretched, but ligamentous fibers are not necessarily torn. First-degree sprains are often referred to as Grade 1 sprains. These sprains exhibit mild signs and symptoms of inflammation, which include a slight increase in tissue temperature, minimal skin redness at the injured site, mild loss of motion, mild swelling, discomfort to mild pain, and mild stiffness due to edema. Second-degree sprains are often referred to as Grade 2 sprains. These sprains occur when many of the ligament’s fibers are completely torn, yet some remain intact. Grade 2 sprains exhibit moderate signs and symptoms of inflammation. An athlete with a second-degree sprain experiences a definitive increase in tissue temperature, skin redness at the injured site, moderate loss of motion, moderate swelling, significant pain, and joint instability. Third-degree, or Grade 3, sprains are the most severe sprains. The ligament is completely ruptured and the tissue is entirely torn. Consequently, the signs and symptoms of third-degree sprains are severe. Third-degree sprains exhibit a greater localized increase in temperature than do second-degree sprains, notable redness, and significant loss of motion, swelling, pain, and joint instability.

Regardless of the degree of injury, when an athlete suffers a sprain she or he will be adversely affected to some extent. Your role is to provide immediate, proper care and medical referral for an athlete with a sprain.

■ Strain

A strain is an injury to a muscle, tendon, or most commonly the musculotendinous junction (MTJ) where the muscle and

tendon meet (Pfeiffer & Mangus, 2012). The muscle and tendon fibers may be stretched or torn. Not all fibers in one muscle or tendon are necessarily affected. Unlike ligaments, which are plastic, muscles are elastic. Of course, if muscle or tendon fibers cannot withstand the stress applied and are stretched beyond their anatomic limit they rupture. The common mechanisms of injury for a strain include a sudden muscle stretch while at rest, or a quick, forceful muscle contraction. Strains are similar to sprains in that strains are classified into the three degrees previously described. First-, second-, and third-degree strains all have similar signs and symptoms to sprains in regard to pain, redness, swelling, and loss of function. However, with all strains, muscle spasm occurs to some extent and pain is most pronounced during muscle function.

To preliminarily determine whether the injury is a sprain or a strain before you refer your athlete to a physician or summon emergency services, you can keep one rule of thumb regarding ligaments in mind. Ligaments are not contractile tissues as muscles are. Therefore, if an athlete has sprained a ligament, the pain most likely is more pronounced when the joint is passively moved versus actively moved. With passive motion, no muscle contraction occurs. The joint is separated and the bony joint surfaces are moved apart. This passive motion and joint surface distraction consequently stretch the ligament, causing pain. When a muscle or tendon is injured, pain is more pronounced with active movement. Active movement occurs when muscles contract to move the joints through a range of motion. The concentric (shortening) or eccentric (lengthening) contraction of the muscle causes pain. This rule of thumb is not always foolproof, because at times, sprains and strains can occur simultaneously.

Tendinopathy

Tendinopathy is a term describing injury to the tendons. The injuries include tendinitis, tenosynovitis, and tendinosis. Tendinitis is an inflammatory condition of the tendon, whereas tenosynovitis is an inflammatory condition of the synovial sheath that surrounds the tendon. Tendinosis is tendon degeneration. Signs and symptoms of tendinopathies include pain during activity that decreases after activity, crepitus, loss of motion, swelling, and **point tenderness**. The mechanism of injury for tendinitis and tenosynovitis can be acute or chronic. An acute mechanism of injury includes a sudden contraction or stretch of the tendon, which is similar to a strain's mechanism of injury. More commonly, the mechanism of injury for tendinitis and tenosynovitis is microtrauma that occurs to the structure due to repetitive

Point tenderness

Discomfort or pain when an injured area is touched.

overuse. Microtraumas are small injuries to the fibers that collectively, over time, create a chronic injury. The repetitive overuse does not necessarily originate from improper mechanics or function. On the contrary, tendinitis and tenosynovitis often occur with proper function. However, the intensity, quantity, duration, and repetition of tendon function all play a part in the tendinopathy. Tendinosis occurs if tendinitis continues to be a chronic condition and proper treatment is not given. As a result, the tendon does not heal and degenerates.

During activity, athletes create continuous, repetitive joint movements by muscular contractions. Some athletes have more repetition during activities than others. For example, by nature of their sport, swimmers and baseball players have repetitive shoulder motion. The athletes perform at a high level of intensity in order to gain optimal performance. If the athletes do not rest adequately between activities and modify their activity in regard to intensity, duration, and function, then the shoulder can become inflamed due to microtraumas. Soreness is a common symptom of microtrauma. Unfortunately, athletes do not often heed this symptom, and additional microtraumas occur during each subsequent activity. Without rest or treatment for this soreness, the microtraumas eventually summate and cause a major inflammatory process. This inflammation can be extremely disabling to an athlete and will hinder their function to some extent (Figure 2-7).

Figure 2-7 An inflamed Achilles' tendon.



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■ Muscle Spasm

Muscle spasms, or muscle cramps, are involuntary contractions of the muscles (Binkley et al., 2002). The muscles become stiff and hard, which results in pain and loss of motion. Various mechanisms of injury for muscle spasms exist. When a joint, muscle, or bone is injured, the surrounding muscle involuntarily contracts in order to protect the injured area from further harm. A direct blow to a muscle; a sudden, forceful movement, in particular an unexpected stretch; or overuse of a muscle, especially when the muscle is fatigued, can also cause the muscle spasm. If an athlete stretches her hamstrings to the extent that the muscle group cannot tolerate the stretch, the muscles spasm to protect against further injury. If a boxer is punched by another boxer in the abdomen, which injures an internal organ, the abdominal muscles contract and become hardened (guarding) in order to protect the internal organs from further injury. Muscle spasms also occur if a body part remains in a still position over time. Muscle spasms often occur in the neck due to sleeping in the same, and potentially awkward, position for hours. Muscle spasms can also occur from dehydration and lack of electrolytes in the body (Binkley et al., 2002). If an athlete does not hydrate enough or ingest adequate electrolytes before exercise, the athlete may succumb to muscle cramping, which is a heat-related illness. Muscle cramping particularly occurs when exercising in a hot and humid environment due to the amount of water and electrolytes lost when an athlete sweats. If you educate your athletes on proper hydration before, during, and after activity, you can prevent this heat-related muscle cramping from occurring.

■ Contusions

A bruise is another name for a contusion. The mechanism of injury for a contusion is a compression force. Typically this force originates from an object compressing a muscle against the underlying bone (Prentice, 2011). The direct blow from the object crushes the muscle fibers, causing injury to the muscle fibers, capillaries, and underlying structures. The object causing injury can be stationary, such as a weight rack that your athlete walks into, or it can be moving, such as a batted baseball. The contusion's severity depends on a multitude of factors, including the size and speed of the object striking the muscle as well as the size, thickness, and strength of the muscle. In regard to object speed, an object traveling at high speed and striking the body would cause a more severe contusion than the same object traveling at a lower speed. For example, consider the speed of a baseball when a batter hits a line drive and strikes the pitcher in the quadriceps. Then consider the catcher tossing the ball slowly with a high arc at the

pitcher, who fails to catch it and gets hit on his quadriceps. The athlete undoubtedly will have a more significant contusion as a result of the high-speed batted ball. Also, if an athlete has strong, large, hypertrophied muscles, then these muscles can withstand greater compression forces as compared to muscles that are weak, small, and atrophied.

Once a muscle is contused, inflammation begins. Signs and symptoms of a contusion are the same as for sprains and strains—pain, redness, increase in tissue temperature, swelling, and loss of function are typical immediately after injury. Hematomas—blood from damaged, bleeding tissues and capillaries—can accumulate around the muscle and under the skin. The hematomas can sometimes be visible if they form at the skin surface. These hematomas cause ecchymosis. Whenever a severe contusion with significant signs and symptoms occurs to one of your athletes, suspect that the underlying bone, internal organs, or structures other than the muscle are also injured. Consequently, you should refer your athlete to a physician for evaluation.

■ Bursitis

Bursae are fluid-filled sacs located throughout the body. They assist in shock absorption and decrease friction between various anatomical structures in the body, such as ligaments, tendons, bones, and skin. Commonly injured bursae include the prepatellar bursa, which is located at the knee between the skin and the patella; the olecranon bursa, which is located between the skin and the olecranon of the elbow; and the trochanteric bursa, which is located at the hip between the greater trochanter and the gluteal muscles. Bursae can be injured in either an acute or a chronic manner. Acute bursitis is caused by a direct, forceful blow to the body. A common mechanism of injury for acute olecranon bursitis, for example, is a direct blow to the elbow while in maximal flexion against a nonyielding object such as a wall. The injured bursa immediately fills with fluid and appears very swollen. The athlete experiences pain and loss of function at the area. Interestingly enough, the athlete may not have point tenderness when touching the swollen bursa, but most likely has pain when touching the bone under and around the bursa. The bursa may feel like a ball of encapsulated jelly, which can be moved back and forth without eliciting pain. The athlete may not be willing to move the joint due to pain, or may even be unable to move the joint. This inability to move the area is due to the significantly larger bursa taking up space and stretching the skin, thereby preventing movement.

Consider the following scenario: An ice hockey player is competing in a game. He goes after the puck at full speed with his stick in hand. His opponent checks him into the boards.

At that moment, his elbow is flexed and his elbow pad slides just enough for his olecranon to strike the board. The athlete feels immediate pain and disability. A few minutes later, when he returns to the bench, his pain has decreased; however, he is unable to flex his elbow. The olecranon bursa is approximately the size of a golf ball. The athlete touches the bursa and moves it around without much pain. When the athlete touches the underlying olecranon, however, he has significant point tenderness. This point tenderness suggests that the direct blow against the boards may have caused a fracture to the underlying bone. Consequently, the athlete was referred to see a physician for x-rays (**Figure 2-8**).

Bursae can also be injured by a chronic mechanism of injury. This mechanism of injury includes overusing a muscle or tendon, and repetitive friction over a bursa. In these cases, the signs and symptoms of injury are similar to those of acute bursitis; however, they appear slowly, perhaps over hours or days. Also, typically the immediate swelling after an acute bursa injury is more pronounced and appears localized. With chronic bursa injuries, the swelling is more generalized. With knee bursitis, an athlete could have chronic inflammation due to repetitive activities such as deep knee

flexion activities or continual kneeling. For example, an athlete at We R Fit Gym begins an intense squat program with heavy resistance. Her body, in particular her knees, are not accustomed to this exercise. Over a period of days, the athlete begins to feel discomfort and point tenderness. She notices generalized puffiness around the front of the knee. These signs and symptoms continue to increase as the days and activities progress. The athlete's knee function deteriorates. Finally, she decides to see a physician who diagnoses her with chronic knee bursitis. The athlete is allowed to continue training, but with limited knee flexion activities until the signs and symptoms of injury subside.

The injuries discussed in this section are the most common soft tissue injuries that your athletes will experience. The injury severity varies immensely from athlete to athlete and injury to injury. The injury may be so mild that the athlete is able to continue activity participation. On the other hand, the injury may be so severe that the athlete is not capable of or allowed to participate for a period of time. No two soft tissue injuries are the same, and your athletes may experience more than one soft tissue injury simultaneously. Regardless of how many soft tissue injuries your athletes may experience, your role in educating and providing immediate care and referral is imperative to the athletes' well-being and return to full pre-injury function.

Figure 2-8 Acute elbow bursitis.



© SPL/Science Source.

Bone Injuries

Fractures

Most likely at some point during your career, one of your athletes will sustain a fracture. Fractures are disabling to an athlete for a variety of reasons. When an athlete breaks a bone, the bone is immobilized with a splint or cast for a period of weeks to allow proper healing. Sometimes it is necessary to perform surgery to assist in realigning the broken bones into their proper position. Fractures typically do not occur as a single injury. Often another injury exists along with the fracture, such as a ligament rupture, muscle contusion or strain, and nerve and blood vessel injury. Mechanisms of injury for fractures include compression, tension, and shear forces. The signs and symptoms of fractures include the typical signs and symptoms of inflammation, which are pain, swelling, loss of function, redness, and increase in temperature. Other signs and symptoms include crepitus, deformity, joint locking due to bony fragments lodged in the joint space, and loss of sensation due to nerve injury.

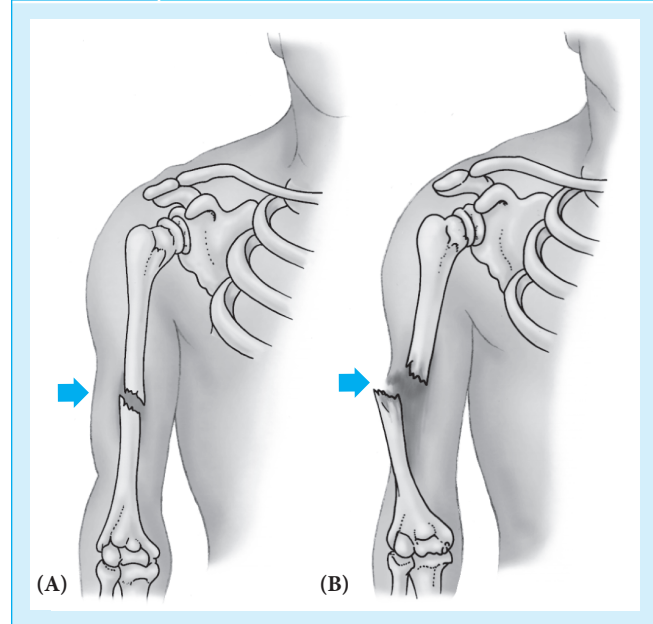
Fractures are classified as closed or open, displaced or nondisplaced, and complete or incomplete. Most fractures are closed fractures, which occur when the broken bones do not protrude through the skin. When the broken bones protrude through the skin, the bones create an open wound, and the fracture therefore is classified as an open or compound

fracture. These fractures are more troublesome than closed fractures because the open wound is susceptible to infection, which could become **systemic** and potentially fatal. Likewise, significant external bleeding may occur, which can also be fatal (**Figure 2-9**). Fractures also are classified as displaced or nondisplaced. If the ends of the fractured bones are not in proper alignment with each other, then the fracture is displaced. If the ends of the bones remain in alignment, then the fracture is nondisplaced. Displaced fractures are often more severe and have the potential to cause additional trauma due to bone movement. When the fractured bones have been pushed out of alignment, more opportunity exists for injury to other structures, including nerves and blood vessels. When fractures are open they are also displaced. Closed fractures can be displaced or nondisplaced. Fractures can occur through the entire bone, creating two or more separate pieces, which is called a complete fracture. An incomplete fracture is when the fracture line does not pass through the entire bone, which leaves part of the bone intact.

Specific terminology is used to describe the type of fracture line. Some of the more common fractures are classified as transverse, oblique, greenstick, comminuted, compression (impacted), spiral, avulsion, stress, or epiphyseal fractures (**Figure 2-10**).

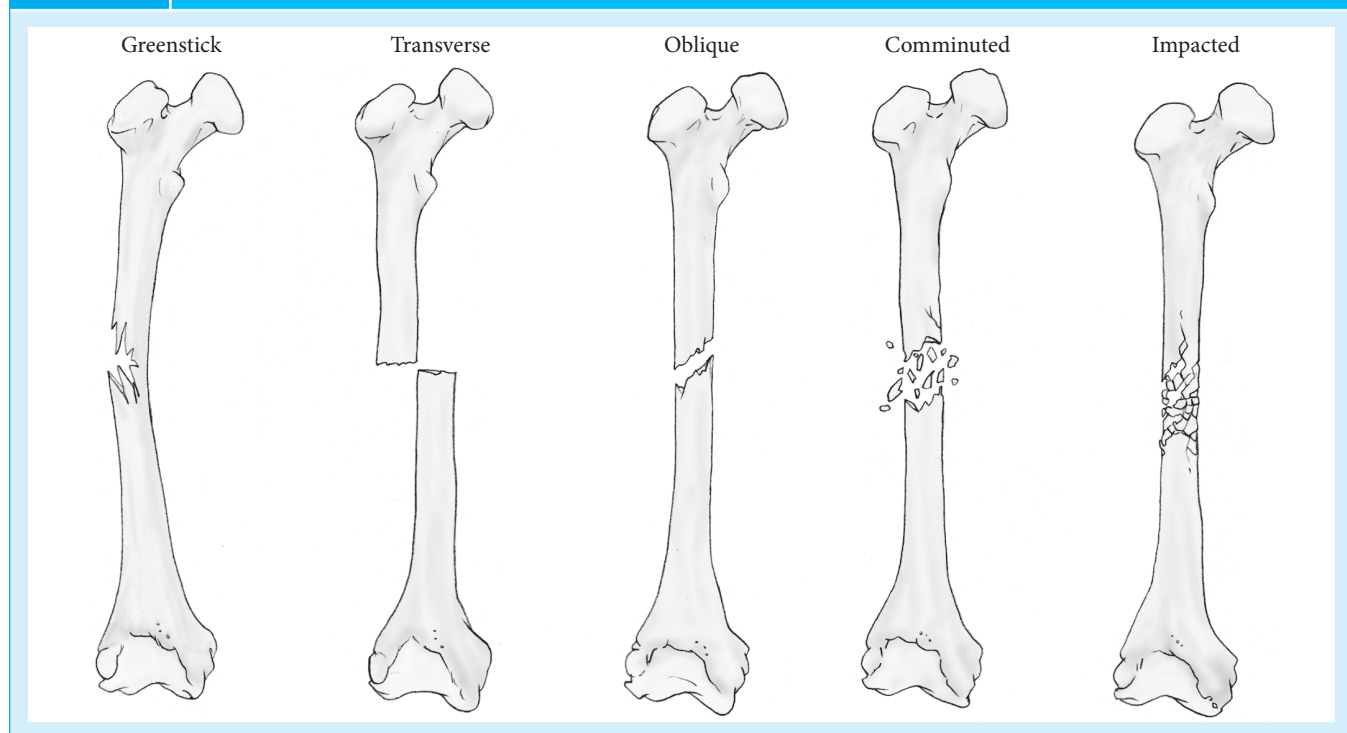
Systemic Throughout the entire body.

Figure 2-9 (A) Closed fracture, (B) open fracture.



A transverse fracture is a complete fracture with a fracture line that is perpendicular to the long axis of the bone (Starkey, Brown, & Ryan, 2010). An oblique fracture line is similar to the transverse fracture line, except it runs on a diagonal to the

Figure 2-10 Types of fractures.



bone's long axis. A greenstick fracture typically occurs in young children whose bones are not completely finished growing and have not entirely hardened. These fractures are incomplete fractures. The side opposite the force is broken, but the side where the stress was applied is intact. A comminuted fracture is a complete fracture with numerous tiny pieces. These bone fragments are floating around in the space between the ends of the bones. They also can dislodge from the space and move around among the tissues, causing further damage.

Compression or impacted fractures occur when stress is applied to the ends of the long bones. This type of fracture can occur if an athlete jumps or falls from a great height, such as a tall ladder or the roof of a house. The compressive forces from the ground and the athlete's body on the femur or tibia can cause the compressive fracture. Likewise, this same mechanism of injury can cause a compression fracture of the spinal vertebrae.

Spiral fractures occur as a result of torsion and compression forces. These forces are directed along the long axis of the bone, and create a spiral fracture line appearance. Consider how a spiral fracture can occur to a football player during a game. The athlete is running with the ball and is tackled. The force causes his hand to be planted on the ground. The hand creates a compressive force up the radius and ulna to the humerus. During the tackle, his body, including his shoulder joint, rotates, which creates torsion. Consequently, a spiral fracture occurs to the humerus.

Avulsion fractures occur when a piece of bone is pulled off the main bone by a ligament or tendon. When a torsion stress occurs to a joint, the ligament or tendon sometimes can withstand the force. Consequently, the ligament or tendon pulls the bone from its attachment, creating a fracture. Often avulsion fractures are difficult to distinguish from a sprain or a strain at the ligament's or tendon's insertion, respectively. Ultimately when an avulsion fracture is suspected, refer the athlete for x-rays to obtain a correct diagnosis.

A fracture that occurs from repetitive or atypical stress over a period of time is referred to as a stress fracture. Stress fractures typically occur in weight-bearing bones due to the force continually placed on them by the athlete contacting the ground or another surface in a repetitive manner. Therefore, these fractures are not considered to have an acute, traumatic mechanism of injury. Most stress fractures occur to the lower extremities due to the constant pounding during running or jumping activities. The muscles and bone cannot readily dissipate the constant, repetitive force. Over time, which could be many weeks, the bone begins to break down. A particular stress fracture symptom is pain that occurs before and after weight-bearing activity. Sometimes the athlete has a dull aching pain at night that wakes them. They often are able to

function, but pain causes their function to decline. The athlete will have point tenderness at the site of the stress fracture. If you suspect your athlete has a stress fracture, stop the athlete's training activities and refer them to a physician for evaluation. The physician will most likely order x-rays for the athlete. When viewing an x-ray, you cannot see a stress fracture unless it has already healed. On an x-ray, the healed stress fracture appears as a stark white line on the bone.

Tips from the Field

Tip 4 If you are unsure whether your athlete has sustained a stress fracture, immediately have your athlete stop any sign- and symptom-provoking activities, and refer them to a physician for evaluation. Do not forget to get a clearance note from the athlete's physician prior to allowing your athlete to return to activity.

Epiphyseal fractures can be dangerous if they occur to young children. The epiphysis is at the end of the bone where new bone is formed. This bone formation occurs during the growing years. Once an adult has stopped growing, the epiphyseal plates, or growth plates, close and solidify. Epiphyseal fractures occur at these growth plates. When a growing athlete sustains a fracture at this site, the fracture can be extremely detrimental to their limb growth. The fracture may disrupt the osteoblast (bone forming) and osteoclast (bone breakdown) activity. Consequently, the bone may stop growing and close prematurely, creating a shortened limb as compared to the other side. Any mechanisms of injury previously discussed can cause an epiphyseal fracture, and the signs and symptoms are similar to other fractures. Epiphyseal fractures may occur as stress fractures or as acute, traumatic fractures. All acute fractures are medical emergencies. Therefore, to ensure proper athlete care, your primary role in treatment of the athlete's acute fracture is summoning emergency personnel immediately.

Joint Injuries

Injuries to the joints are common. Not only can the ligaments and tendons that cross over joints become injured, but the bony articulation at the joint also can be damaged. Stresses placed on the joint from bodily movements and outside forces can cause dislocations and subluxations, which are two very common joint injuries.

Dislocations

Dislocations, or luxations, are acute joint injuries that occur when one end of a bone is forced out of its normal position relative to the other bone (Figure 2-11). The mechanisms of

Figure 2-11 A dislocated finger.

Courtesy of AAOS.

injury for a dislocation are a direct blow to the joint by an outside force or a force created by the athlete's body along with the bones moving in a manner in which they are unaccustomed. Some joints are more easily dislocated than others. The glenohumeral (shoulder), patellofemoral (kneecap), and phalangeal joints (fingers) are typically the most frequently dislocated joints. These joints are most commonly dislocated due to their shallow bony articulations. Signs and symptoms of dislocations are readily apparent. Obvious deformity is a very prevalent sign exhibited when a dislocation occurs. The bone protrudes from its normal position, creating disfigurement. Swelling may or may not be present. The athlete cannot move the joint because the bones are out of place. The athlete has significant pain, and possibly some sensation loss. Picture a quarterback preparing to throw the football with his arm in a cocked position. Suddenly the opposing lineman goes for the ball and tackles the quarterback. The opponent's force pushes the quarterback's arm in a posterior, or backwards, position, which results in a shoulder dislocation. In another scenario, a basketball player attempts to catch a ball that forcefully hits the tip of her finger. The ball forces her finger to move out of alignment. The distal phalange dislocates and moves on top of the middle phalange.

Unfortunately, once an athlete dislocates a joint, the chance of the same joint dislocating again increases. Additional dislocations are possible due to the joint structures, including the capsule, ligaments, tendons, and muscle, becoming significantly stretched and injured with the first dislocation. Consequently, the bones that comprise the joint are not held as tightly in place as they were prior to injury. The force necessary to dislocate the same joint a second time does not have to be as great as with the initial injury. Keep in mind that the force needed to cause a dislocation can also cause a concurrent fracture, along with nerve and blood vessel damage at the

injury site (Anderson et al., 2000). Because secondary injuries are possible with dislocations, they must be treated as medical emergencies. Only physicians are qualified to relocate the bones. Never put the bones back into normal alignment, because you can do further harm to your athlete. Instead, call your local emergency number immediately.

Subluxations

Subluxations occur when a bone within a joint moves out of normal alignment and then spontaneously moves back into normal position. The movement out of normal joint alignment occurs due to an outside force, such as a direct blow from an object, or an internal force, such as a forceful muscle contraction. Unlike dislocations, subluxations can be acute or chronic (Starkey et al., 2010). The mechanism of injury for an acute subluxation is the same as for a dislocation. An example of an acute subluxation is when a humeral head rapidly moves out of normal joint position and then immediately back into normal joint position on its own. This injury is classified as acute due to the traumatic mechanism of injury. Similar to a dislocation, the possibility for future subluxations exists. After an athlete sustains his or her first acute subluxation, he or she may have repetitive subluxations of the same joint. These recurrent subluxations are classified as chronic. Along with the glenohumeral joint, chronic subluxations often occur at the patellofemoral joint.

The signs and symptoms of subluxations include pain, point tenderness, loss of function, swelling, and possible loss of sensation. Unlike dislocations, because a subluxed joint is in normal alignment, you do not see deformity. However, because the joint was out of normal alignment temporarily, consider that secondary injury may have occurred. Therefore, in order for your athlete to obtain proper care, refer her or him for immediate physician evaluation.

THE BODY'S RESPONSE TO ILLNESS

Athletes are prone to illnesses like any other person. The healthier the athlete is psychologically and physically, the less chance she or he will acquire an illness; however, athletes cannot entirely prevent illnesses. A variety of common causes of illness exist, including nutritional deficiencies, lack of exercise, impaired immunity, mental stress, heredity, toxins, and microorganism infection (Neighbors & Tannehill-Jones, 2000). Over time, lack of exercise, or even a lack of minimal physical activity, decreases bodily function, causing weakened muscle and bones, and immune system deterioration. Consequently, the athlete's immune system cannot fight infection properly and illness results. Mental stress can increase to the point where the body can no longer tolerate the stress.

Again, the immune system weakens and illness can invade the body. Hereditary illness is due to abnormalities in an athlete's chromosomes in utero. These chromosomal abnormalities may not cause illness until later in life, or perhaps not at all (Neighbors & Tannehille-Jones, 2000).

Toxins are poisonous substances produced by a living cell that enters an athlete's body and causes illness. A bee creates a toxin as a defense mechanism, which is released when the bee stings a person or an animal. For some athletes, this sting may be deadly, because the venom released causes an athlete to go into anaphylactic shock. Anaphylactic shock occurs when a person is severely allergic to a toxin, such as bee venom. The toxin causes swelling in and constriction of the air passages, which makes the person unable to breathe. Consequently, anaphylactic shock is a serious medical emergency.

Microorganisms entering the body can cause illness and infection. The three microorganisms that commonly cause illness are bacteria, viruses, and fungi. These microorganisms are the predominant causes for athletes' everyday illnesses.

Bacteria are cells that reproduce in the body and produce systemic illnesses and localized infections. When an illness affects the entire body it is a systemic illness; a localized illness or infection is when the bacteria affect only a particular area. Many types of bacteria exist. Some bacteria are very strong and difficult to eliminate from the body, such as methicillin-resistant *Staphylococcus aureus* (MRSA), whereas others are more easily destroyed, such as *Campylobacter*, which causes food poisoning (Crowley, 2010). Fortunately for athletes, antibiotics can be used to break down the microorganisms and return the athlete's body to a normal state. Bacteria also cause common illnesses and infections such as acne, sinus infections, strep throat, meningitis, gastroenteritis, and Lyme disease (Pommerville, 2012).

Viruses are smaller microorganisms than bacteria; however, unlike bacteria, viruses must attach to a host cell in the athlete's body in order to live and replicate (Neighbors & Tannehille-Jones, 2000). Viruses can replicate and mutate. This mutation makes it extremely difficult to destroy the virus with any medicinal agent. Antibiotics or other drugs are not able to destroy the virus as they effectively destroy bacteria. Consequently, over-the-counter (OTC) and prescription drugs are utilized to alleviate the athlete's signs and symptoms. The viral illness must run its course. The athlete's immune system attempts to fight the virus, break it down, and eliminate it from the body. Common viral illnesses include herpes simplex 1 virus (cold sores), conjunctivitis (pink eye), rhinoviruses (head colds), and verruca virus (warts) (Pommerville, 2012). Fortunately, vaccines exist that can help prevent an athlete from obtaining certain viruses. Common immunizations include those for hepatitis B virus (HBV), influenza (flu), and

measles, mumps, and rubella (MMR). In response to these immunizations, the body creates antibodies that fight the virus, so the athlete does not acquire the illness or infection.

Fungi are plantlike organisms that thrive in dark, moist environments. Many fungal infections occur on the skin and remain localized, such as tinea corporis (ringworm), tinea cruris (jock itch), tinea pedis (athlete's foot), and *Candida albicans* (yeast infection). Although rare, fungal infections can become significant systemic illnesses. Usually, normal bodily bacteria prevent fungi from causing a systemic illness, such as a pulmonary fungal infection. However, if bacteria are not functioning properly, acute systemic fungal illness can occur (Crowley, 2010). Fungal infections can be treated by antifungal oral and topical medications.

Regardless of the microorganism, bacteria, viruses, and fungi can be contagious, meaning they can be spread from athlete to athlete, either by direct contact, such as touching the infected area, or by indirect contact, such as the transference through water molecules in saliva or a sneeze. An ill athlete is considered contagious for a specific period of time, but this duration varies depending on the microorganism. Common contagious illnesses and infections include the flu, pink eye, ringworm, and herpes.

Tips from the Field

Tip 5 If your athlete has any skin lesions that appear as pustules, rashes, hives, or any unusual, unfamiliar markings, immediately have your athlete see a doctor. Your athlete must not be permitted to participate in activities with your organization or in your facility until they have received written clearance from their physician and provided it to you. The skin lesion may be infectious and contagious. Immediately disinfect any object and area that your athlete has contacted, because the microorganism causing the potential infection could be transferred to your other athletes.

SUMMARY

Now that you have learned about the healing process and classification of injuries and illnesses, various classifications, mechanisms, and signs and symptoms of injury are familiar. This information will assist you in providing athlete care.

DISCUSSION QUESTIONS

1. What are the key events that occur in the three phases of healing?
2. What is the difference between a sign and a symptom of injury and illness?

3. What are differences between sprains and strains? What can you do to preliminarily determine if an athlete has a sprain or a strain?
4. How can an injury go from being acute to chronic? What classifies an injury as being chronic?
5. Why are compound fractures medical emergencies? Why could an athlete's life be threatened if she has a compound fracture?
6. Why is it important that exercise scientists do not reduce a dislocated joint? What could happen to the athlete if an untrained person relocates the bones?
7. What is bursitis, and how can it affect an athlete's performance?
8. What microorganisms cause illnesses, and how do they differ? What general treatments can be utilized to rid the organisms from the body and decrease signs and symptoms of illness?

CASE STUDY

Listen to Your Body

Megan is a 15-year-old, slender cross-country runner at Ocean Crest High School. She has just won the county championship meet. As a result, Megan has an automatic entry into the state championship meet, which takes place in 1 month. The state championship meet distance is twice as long as any other meet she has ever participated in. Likewise, it takes place on unfamiliar terrain for Megan, which includes rugged, steep hills. To prepare for this meet, Megan's coach instructs her to train 5 days per week. He says that he will run with her each day, because he wants to motivate and push her to her training limit.

Megan's training intensity is significantly increased as compared to her previous training for the county championship. She runs farther each day on various surfaces including dirt, blacktop, and all-weather track. Megan begins training up and down hills, which she is not accustomed to doing. After a few days of intense training, as a gift her mother purchases Megan new running sneakers, which she wears the next day to train. Megan sees that she is progressing after just a few days of vigorous training. She thinks to herself that if she also runs on weekends, she will improve faster and become better. Consequently, Megan decides to train on her 2 days off, making her training schedule 7 days per week. She does not schedule any rest days.

After about 2 weeks of training, Megan begins to experience some discomfort in the bottom of her right foot at the end of her workouts. She believes that the discomfort will go away on its own. Also, Megan is afraid to tell her mother or coach about the discomfort because she is fearful that she would not be allowed to continue her 7 days per week training. Megan tries to ignore the discomfort and work through it. A few days after her discomfort began, the discomfort turns into dull, aching, constant pain after the workouts. The pain not only is considerably worse after workouts, but Megan also starts to feel the pain during the workout. Then she begins to experience aching on the bottom of her right foot even when her foot is at rest while she is sitting or lying down.

One day during a workout, the pain is so bad that it begins to affect her gait and she starts to limp. She is not able to put her right foot flat on the ground and instead walks on her right foot's toes. At this point, she still tries to complete the workout, but due to the pain the workout suffers considerably to the point where she cannot run at all and complete the workout. Megan cannot tolerate the pain and finally realizes that her foot is not getting any better. She immediately tells her coach and mother about what has occurred over the last 3 weeks. The next day, her mother takes her to the doctor, who says that Megan cannot run for a month or more, which prevents her from competing in the state championship meet.

1. What types of injuries could Megan have?
2. What factors led to Megan's injury?
3. What could Megan have done when she first began to train for the state championship meet in an attempt to prevent this injury?

REFERENCES

- American Medical Association. (1966). Annual reports. *Journal of the American Medical Association*, 198(4), 403. Retrieved May 31, 2011, from <http://jama.ama-assn.org/content/198/4/397.full.pdf+html>
- Anderson, M. K., Hall, S. J., & Martin, M. (2000). *Sports injury management* (2nd ed.). Philadelphia: Lippincott, Williams & Wilkins.
- Bahr, R., & Maehlum, S. (Eds.). (2004). *Clinical guide to sports injuries: An illustrated guide to the management of injuries in physical activity*. Champaign, IL: Human Kinetics.
- Binkley, H. M., Beckett, J., Casa, D. J., Kleiner, D. M., & Plummer, P. E. (2002). National Athletic Trainers' Association position statement: Exertional heat illnesses. *Journal of Athletic Training*, 37(3), 329–343. Retrieved June 3, 2011, from <http://www.nata.org/sites/default/files/ExternalHeatIllnesses.pdf>
- Booher, J. M., & Thibodeau, G. A. (2000). *Athletic injury assessment* (4th ed.). New York: McGraw-Hill.
- Crowley, L. V. (2010). *An introduction to human disease: Pathology and pathophysiology correlations* (8th ed.). Sudbury, MA: Jones and Bartlett.

Eaves, T. (2010). *The practical guide to athletic training*. Sudbury, MA: Jones and Bartlett Learning.

Neighbors, M., & Tannehill-Jones, R. (2000). *Human diseases*. Albany, NY: Delmar Thompson Learning.

Pfeiffer, R. P., & Mangus, B. C. (2012). *Concepts of athletic training* (6th ed.). Sudbury, MA: Jones and Bartlett Learning.

Pommerville, J. C. (2012). *Guide to infectious diseases by body system* (2nd ed.). Sudbury, MA: Jones and Bartlett Learning.

Prentice, W. E. (2011). *Principles of athletic training: A competency-based approach* (14th ed.). New York: McGraw-Hill.

Prentice, W. E. (2010). *Essentials of athletic injury management* (8th ed.). New York: McGraw-Hill.

Starkey, C., Brown, S. D., & Ryan, J. (2010). *Examination of orthopedic and athletic injuries* (3rd ed.). Philadelphia: F.A. Davis.