Chapter 2

Understanding and Managing the Healing Process Through Rehabilitation

Linda Stark Bobo, PhD, LAT, ATC
William E. Prentice, PhD, AT Ret, BSPT, FNATA

After reading this chapter, the athletic trainer should be able to:

- Describe the pathophysiology of the healing process.
- Identify the factors that can impede the healing process.
- Discuss the etiology and pathology of various musculoskeletal injuries associated with various types of tissues.
- Compare healing processes relative to specific musculoskeletal structures.
- Explain the importance of initial first aid and injury management of these injuries and their impact on the rehabilitation process.
- Discuss the use of various analgesics, anti-inflammatories, and antipyretics in facilitating the healing process during a rehabilitation program.

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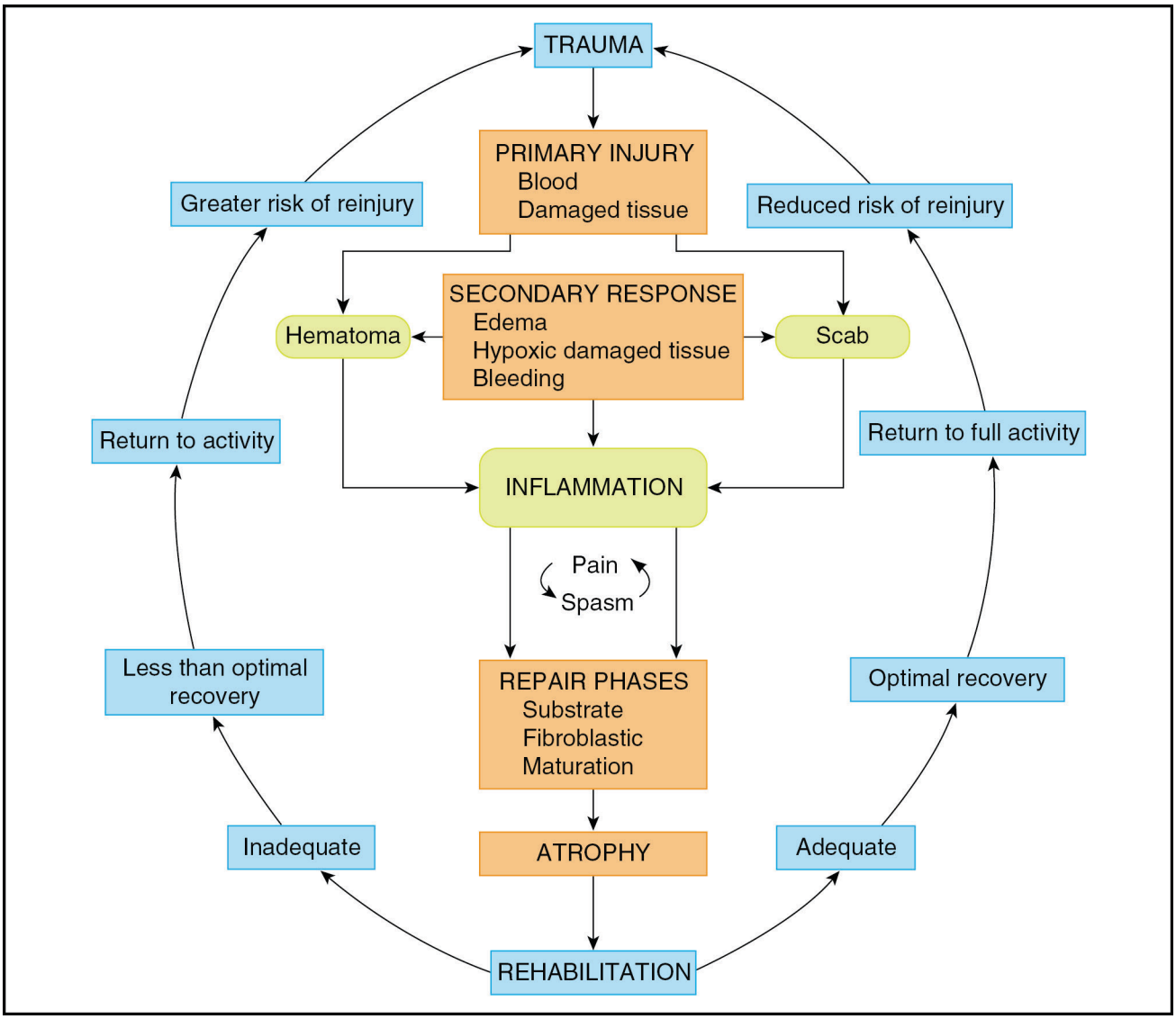


Figure 2-1. A cycle of sport-related injury. (Adapted from Booher JM, Thibadeau GA. *Athletic Injury Assessment*. St. Louis, MO: Mosby; 2000.)[20]

Injury rehabilitation requires sound knowledge and understanding of the etiology and pathology involved in various musculoskeletal injuries that may occur.[68,99] When injury occurs, the athletic trainer is charged with designing, implementing, and supervising the rehabilitation program. Rehabilitation protocols and progressions must be based primarily on the physiologic responses of the tissues to injury and on an understanding of how various tissues heal.[74] Thus, the athletic trainer must understand the healing process to effectively supervise the rehabilitative process. This chapter discusses the healing process relative to the various musculoskeletal injuries that may be encountered by an athletic trainer.

UNDERSTANDING THE HEALING PROCESS

Rehabilitation programs must be based on the cycle of the healing process (Figure 2-1). The athletic trainer must have a sound understanding of the sequence of the various phases of the healing process. The physiologic responses of the tissue to trauma follow a predictable sequence and time frame when the healing environment is healthy and regulated.[87] Decisions on how and when to alter and progress a rehabilitation program should be primarily based on recognition of signs and symptoms, as well as an awareness of the time frames associated with the various phases of healing.[56] The healing process consists of the inflammatory response phase, fibroblastic repair phase, and maturation remodeling phase. It must be stressed that although the phases of healing are presented as 3 separate entities, the healing process is a continuum. Phases of the healing process overlap one another and have no definitive beginning or end points.[63]

Primary Injury

Primary injuries have historically been described as being either acute or chronic. But primary injuries are

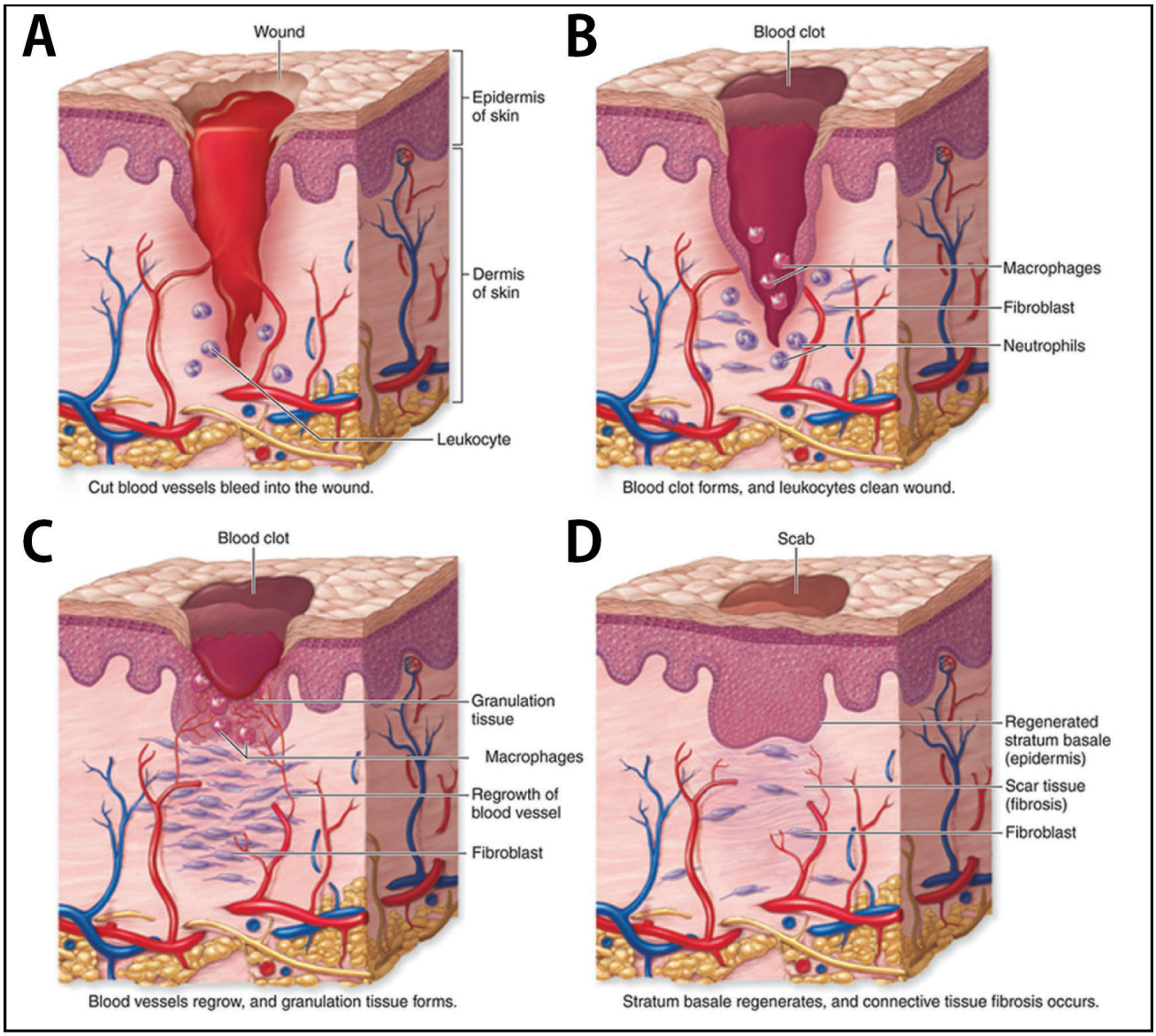


Figure 2-2. Initial injury and inflammatory response phase of the healing process. (A) Cut blood vessels bleed into the wound. (B) Blood clot forms, and leukocytes clean the wound. (C) Blood vessels regrow, and granulation tissue forms in the fibroblastic repair phase of the healing process. (D) Epithelium regenerates, and connective tissue fibrosis occurs in the maturation-remodeling phase of the healing process. (Adapted from McKinley M, O'Loughlin V. *Human Anatomy: An Integrative Approach*. Chicago, IL: McGraw-Hill; 2015.)

perhaps better classified as *traumatic* or *overuse* injuries due to the a lack of clarity with the terms acute and chronic.[39,112,115] Injuries result from either *macrotraumatic* or *microtraumatic* forces.[26] Traumatic injuries result from acute macrotraumatic forces which produce immediate pain and disability. Macrotraumatic injuries include fractures, dislocations, subluxations, sprains, strains, and contusions. Microtraumatic injuries are most often called *overuse injuries* and result from repetitive overloading or incorrect mechanics associated with repeated motion.[9] Microtraumatic injuries include tendinitis, tendinosis, tenosynovitis, bursitis, etc.[108] A secondary injury is essentially the inflammatory or hypoxia response that occurs with the primary injury.

Inflammatory Response Phase

Once a tissue is injured, the process of healing begins immediately[119] (Figure 2-2A). The destruction of tissue produces direct injury to the cells of the various soft tissues. Cellular injury results in altered metabolism and the liberation of materials that initiate the inflammatory response. This phase is characterized symptomatically by redness, swelling, tenderness, and increased temperature.[23] This initial inflammatory response is critical to the entire healing process.[89] If this response does not accomplish what it is supposed to or if it does not subside, normal healing cannot take place.

Inflammation is a process through which leukocytes and other phagocytic cells and exudates are delivered to the

injured tissue. This cellular reaction is generally protective, tending to localize or dispose of injury byproducts (eg, blood and damaged cells) through phagocytosis, thus setting the stage for repair. Local vascular effects, disturbances of fluid exchange, and migration of leukocytes from the blood to the tissues occur.[105]

Clinical Decision-Making Exercise 2-1

A physical education student fell on his wrist playing flag football. It is very swollen, and he has decreased strength and range of motion. The athletic trainer does not suspect a fracture. A decision is made to provide an initial treatment instead of sending the student to the emergency room. What should the athletic trainer's goals be at this time?

Hemostasis

Hemostasis is a vascular reaction that involves vascular spasm, formation of a platelet plug, blood coagulation, and growth of fibrous tissue.[76] The immediate response to tissue damage is a vasoconstriction of the vascular walls in the vessels leading away from the site of injury that lasts for about 5 to 10 minutes. This vasoconstriction presses the opposing endothelial wall linings together to produce a local anemia that is rapidly replaced by hyperemia of the area as a result of vasodilation.[21] This increase in blood flow is transitory and gives way to slowing of the flow in the dilated vessels, thus enabling the leukocytes to slow down and adhere to the vascular endothelium. The completion of hemostasis allows a migration of inflammatory cells leading to stagnation and stasis creating another transition in the inflammatory phase.[34] The initial effusion of blood and plasma lasts for 24 to 36 hours.

Chemical Mediators

The events in the inflammatory response are initiated by a series of interactions involving several chemical mediators, which cause the vascular and cellular changes resulting from inflammation. Some of these chemical mediators are derived from the invading organism, some are released by the damaged tissue, others are generated by several plasma enzyme systems, and still others are products of various white blood cells participating in the inflammatory response. Four chemical mediators—cytokines, leukotrienes, prostaglandins, and histamine—are important in limiting the amount of exudate, and thus swelling, after injury. Cytokines, in particular chemokines and interleukin, are the major regulators of leukocyte traffic and help to attract leukocytes to the actual site of inflammation.[102] Responding to the presence of chemokines, phagocytes enter the site of inflammation within a few hours. Leukotrienes and prostaglandins are responsible for margination, in which leukocytes (neutrophils and macrophages) adhere along the cell walls. They also increase cell permeability locally, thus affecting the passage of the fluid and white blood cells through cell walls via diapedesis to form exudate. Consequently, vasodilation and active hyperemia are important in exudate (plasma) formation and in supplying leukocytes to the injured area. Histamine, released from the injured mast cells, causes vasodilation and increased cell permeability, owing to a swelling of endothelial cells and then separation between the cells. The amount of swelling that occurs is directly related to the extent of vessel damage.

Formation of a Clot

Platelets do not normally adhere to the vascular wall. However, injury to a vessel disrupts the endothelium and exposes the collagen fibers. Platelets adhere to the collagen fibers to create a sticky matrix on the vascular wall, to which additional platelets and leukocytes adhere and eventually form a plug. These plugs obstruct local lymphatic fluid drainage and thus localize the injury response (Figure 2-2B).

The initial event that precipitates clot formation is the conversion of fibrinogen to fibrin. This transformation occurs because of a cascading effect, beginning with the release of a protein molecule called *thromboplastin* from the damaged cell. Thromboplastin causes prothrombin to be changed into thrombin, which, in turn, causes the conversion of fibrinogen into a very sticky fibrin clot that shuts off blood supply to the injured area. Clot formation begins around 12 hours after injury and is completed within 48 hours.

As a result of a combination of these factors, the injured area becomes walled off during the inflammatory stage of healing. The leukocytes phagocytize most of the foreign debris toward the end of the inflammatory phase, setting the stage for the fibroblastic phase. This initial inflammatory response lasts for about 2 to 4 days after initial injury.

Clinical Decision-Making Exercise 2-2

A swimmer who does the back stroke suffered a second-degree latissimus dorsi strain. The coach wants to know why he cannot be ready to compete the next day. What should the athletic trainer tell the coach about the healing process and how long it may take the strain to heal?

Chronic Inflammation

A distinction must be made between the acute inflammatory response as previously described and chronic inflammation. Chronic inflammation occurs when the acute inflammatory response does not respond sufficiently to eliminate the injuring agent and restore tissue to its normal physiologic state. Thus, the regulation of chemical mediators is compromised.[77, 94] The neutrophils that are normally present during acute inflammation are replaced by macrophages, lymphocytes, fibroblasts, and plasma cells. As this low-grade inflammation persists, damage occurs to

connective tissue, resulting in tissue necrosis and fibrosis prolonging the healing process. Chronic inflammation involves the production of granulation tissue and fibrous connective tissue. These cells accumulate in a highly vascularized and innervated loose connective tissue matrix in the area of injury.[97] The specific mechanisms that cause an insufficient acute inflammatory response are unknown, but can be related to situations that involve overuse or overload with cumulative microtrauma to a particular structure.[3,15] Physical and pharmacological treatments have minimal effects on chronic inflammation.[89, 93]

Use of Anti-Inflammatory Medications

A physician will routinely prescribe nonsteroidal anti-inflammatory drugs (NSAIDs) for a patient who has sustained an injury.[103] These medications are certainly effective in minimizing pain and swelling associated with inflammation and can enhance return to normal activity. There is some disagreement as to whether the use of NSAIDs acutely following injury might actually interfere with inflammation, thus delaying the healing process.[88]

Fibroblastic Repair Phase

During the fibroblastic phase of healing, proliferative and regenerative activity leading to scar formation and repair of the injured tissue follows the vascular and exudative phenomena of inflammation[42] (Figure 2-2C). The period of scar formation referred to as *fibroplasia* begins within the first few hours after injury and can last as long as 4 to 6 weeks. During this period, many of the signs and symptoms associated with the inflammatory response subside. The patient might still indicate some tenderness to touch and will usually complain of pain when particular movements stress the injured structure. As scar formation progresses, complaints of tenderness or pain gradually disappear.[30]

During this phase, growth of endothelial capillary buds into the wound is stimulated by a lack of oxygen, after which the wound is capable of healing aerobically.[23] Along with increased oxygen delivery comes an increase in blood flow, which delivers nutrients essential for tissue regeneration in the area.[23]

The formation of a delicate connective tissue called *granulation tissue* occurs with the breakdown of the fibrin clot. Granulation tissue consists of fibroblasts, collagen, and capillaries. It appears as a reddish granular mass of connective tissue that fills in the gaps during the healing process.

As the capillaries continue to grow into the area, fibroblasts accumulate at the wound site, arranging themselves parallel to the capillaries. Fibroblastic cells begin to synthesize an extracellular matrix that contains protein fibers of collagen and elastin, a ground substance that consists of nonfibrous proteins called *proteoglycans*, glycosaminoglycans, and fluid. On about day 6 or 7, fibroblasts also begin producing collagen fibers that are deposited in a random fashion throughout the forming scar. As the collagen continues to proliferate, the tensile strength of the wound rapidly increases in proportion to the rate of collagen synthesis. As the tensile strength increases, the number of fibroblasts diminishes, signaling the beginning of the maturation phase.

This normal sequence of events in this repair phase leads to the formation of minimal scar tissue. Occasionally, a persistent inflammatory response and continued release of inflammatory products can promote extended fibroplasia and excessive fibrogenesis, which can lead to irreversible tissue damage.[13] Fibrosis can occur in synovial structures, as with adhesive capsulitis in the shoulder, in extra-articular tissues including tendons and ligaments, in bursa, or in muscle.

Clinical Decision-Making Exercise 2-3

A cross-country runner strained her quadriceps. How will the healing process for this injury differ from the process for a ligamentous injury?

The Importance of Collagen

Collagen is a major structural protein that forms strong, flexible, inelastic structures that hold connective tissue together. There are at least 17 types of collagen, but 80% to 90% of the collagen in the body consists of types I, II, and III. Type I collagen is found in skin, fascia, tendon, bone, ligaments, cartilage, and interstitial tissues; type II can be found in hyaline cartilage and vertebral discs; and type III is found in skin, smooth muscle, nerves, and blood vessels. Type III collagen has less tensile strength than does type I and tends to be found more in the fibroblastic repair phase. Collagen enables a tissue to resist mechanical forces and deformation. Elastin, however, produces highly elastic tissues that assist in recovery from deformation. Collagen fibrils are the load-bearing elements of connective tissue. They are arranged to accommodate tensile stress but are not as capable of resisting shear or compressive stress. Consequently, the direction of orientation of collagen fibers is along lines of tensile stress.[62]

Collagen has several mechanical and physical properties that allow it to respond to loading and deformation, permitting it to withstand high tensile stress. The mechanical properties of collagen include elasticity, which is the capability to recover normal length after elongation; viscoelasticity, which allows for a slow return to normal length and shape after deformation; and plasticity, which allows for permanent change or deformation. The physical properties include force relaxation, which indicates the decrease in the amount of force needed to maintain a tissue at a set amount of displacement or deformation over time; creep response, which is the ability of a tissue to deform over time while a constant load is imposed; and hysteresis, which is the amount of relaxation a tissue has undergone during deformation and displacement. Injury results when the mechanical and physical limitations of connective tissue are exceeded.[74]

Maturation Remodeling Phase

The maturation remodeling phase of healing is a long-term process (Figure 2-2D). This phase features a realignment or remodeling of the collagen fibers that make up scar tissue according to the tensile forces to which that scar is subjected. Ongoing breakdown and synthesis of collagen occur with a steady increase in the tensile strength of the scar matrix. With increased stress and strain, the collagen fibers realign in a position of maximum efficiency parallel to the lines of tension.[11] The tissue gradually assumes normal appearance and function, although a scar is rarely as strong as the normal injured tissue. Usually by the end of about 3 weeks, a firm, strong, contracted, nonvascular scar exists. The maturation phase of healing might require several years to be totally complete.

Role of Progressive Controlled Mobility During the Healing Process

Wolff's law states that bone and soft tissue will respond to the physical demands placed on them, causing them to remodel or realign along lines of tensile force.[116] Consequently, it is critical that injured structures be exposed to progressively increasing loads throughout the rehabilitative process.[89]

In animal models, controlled mobilization is superior to immobilization for scar formation, revascularization, muscle regeneration, and reorientation of muscle fibers and tensile properties.[120] However, a brief period of immobilization of the injured tissue during the inflammatory response phase is recommended and will likely facilitate the process of healing by controlling inflammation, thus reducing clinical symptoms. As healing progresses to the repair phase, controlled activity directed toward return to normal flexibility and strength should be combined with protective support or bracing.[54] Generally, clinical signs and symptoms disappear at the end of this phase.

As the remodeling phase begins, aggressive active range of motion (ROM) and strengthening exercises should be incorporated to facilitate tissue remodeling and realignment. To a great extent, pain will dictate the rate of progression. With initial injury, pain is intense; it tends to decrease and eventually subside altogether as healing progresses. Any exacerbation of pain, swelling, or other clinical symptoms during or after a particular exercise or activity indicate that the load is too great for the level of tissue repair or remodeling. The therapist must be aware of the time required for the healing process and realize that being overly aggressive can interfere with that process.

Clinical Decision-Making Exercise 2-4

A track athlete is recovering from a grade 1 ankle sprain. Beginning exercises as soon as possible will increase the injured runner's chances of recovering quickly and strongly. Why is this so?

Factors That Impede Healing

Extent of Injury

The nature of the inflammatory response is determined by the extent of the tissue injury. Microtears or soft tissue involve only minor damage and are most often associated with overuse. Macrotears involve significantly greater destruction of soft tissue and result in clinical symptoms and functional alterations. Macrotears are generally caused by acute trauma.[101]

Edema

The increased pressure caused by swelling retards the healing process, causes separation of tissues, inhibits neuromuscular control, produces reflexive neurologic changes, and impedes nutrition in the injured part. Edema is best controlled and managed during the initial first-aid management period, as described previously.[7]

Bleeding

Bleeding occurs with even the smallest amount of damage to the capillaries. It can also produce the same negative effects on healing as does the accumulation of edema. The presence of blood produces additional tissue damage, thus exacerbation of the injury.[45]

Poor Vascular Supply

Injuries to tissues with a poor vascular supply heal poorly and at a slow rate. This response is likely related to a failure in the initial delivery of phagocytic cells and fibroblasts necessary for scar formation.[45]

Separation of Tissue

Mechanical separation of tissue can significantly impact the course of healing. A wound that has smooth edges in good apposition will tend to heal by primary intention with minimal scarring. Conversely, a wound that has jagged, separated edges must heal by secondary intention, with granulation tissue filling the defect, and excessive scarring.

Muscle Spasm

Muscle spasm causes traction on the torn tissue, separates the two ends, and prevents approximation. Local and generalized ischemia can result from spasm.

Atrophy

Wasting away of muscle tissue begins immediately with injury. Strengthening and early mobilization of the injured structure retard atrophy.

Corticosteroids

Use of corticosteroids in the treatment of inflammation is controversial. Steroid use in the early stages of healing has been demonstrated to inhibit fibroplasia, capillary proliferation, collagen synthesis, and increases in tensile strength of the healing scar. Their use in the later stages of healing and with chronic inflammation is debatable.[7]

Keloids and Hypertrophic Scars

Keloids occur when the rate of collagen production exceeds the rate of collagen breakdown during the maturation phase of healing. This process leads to hypertrophy of scar tissue, particularly around the periphery of the wound.

Infection

The presence of bacteria in the wound can delay healing, causes excessive granulation tissue, and frequently causes large, deformed scars.[20]

Humidity, Climate, and Oxygen Tension

Humidity significantly influences the process of epithelialization. Occlusive dressing stimulates the epithelium to migrate twice as fast without crust or scab formation. The formation of a scab occurs with dehydration of the wound and traps wound drainage, which promotes infection. Keeping the wound moist provides an advantage for the necrotic debris to go to the surface and be shed.

Oxygen tension relates to the neovascularization of the wound, which translates into optimal saturation and maximal tensile strength development. Circulation to the wound can be affected by ischemia, venous stasis, hematomas, and vessel trauma.

Health, Age, and Nutrition

The elastic qualities of the skin decrease with age. Degenerative diseases, such as diabetes and arteriosclerosis, also become a concern of the older patient and can affect wound healing. Malnutrition, smoking, and alcohol have been shown to negatively affect the healing process.[5,97] A patient who is undernourished or malnourished or is even post-operative,[5] prolongs the inflammatory response phase and thus delays the fibroblastic phase by decreasing the proliferation of fibroblasts and subsequently the formation of collagen, thus reducing tensile strength of the healing wound. It can also increase the risk for infection by decreasing T-cell function and phagocytic activity.[97]

THE IMPORTANCE OF NUTRITION TO THE HEALING PROCESS

It appears that nutrition plays a critical role in the healing process regardless of the type of tissue that is injured.[97] Healing of injuries to any of the body's tissues requires optimal intake of various nutrients, which collectively provide the energy necessary to fuel the healing process. Nutrients have three major functions: production of energy; growth, repair, and maintenance of all body tissues; and regulation of body processes.[40] Nutrients are categorized into 6 major classes: carbohydrates, fats (also called *lipids*), proteins, water, vitamins, and minerals. Carbohydrates, proteins, and fats are referred to as the *macronutrients*, the absorbable components of food from which energy is derived.[74] Multiple factors, both individually and collectively, dictate energy demands, including age, gender, activity level, basal metabolic rate, nutritional status, body mass index, stress of illness, severity, size and number of wounds, and stage in the healing process.[112]

Carbohydrates are the body's most efficient immediate source of energy. During digestion, carbohydrates are easily broken down and converted to glucose. The glucose that is not needed to provide immediate energy is stored as glycogen in the liver and muscle cells. A supply of glucose must be kept available to prevent the use of protein for energy. This is called the *protein-sparing action* of glucose.

Like carbohydrates, fats are a primary source of energy. Fats are the most concentrated source of energy, providing more than twice the calories per gram when compared with carbohydrates or proteins. Omega-3 fatty acids aid in lowering the collection of inflammatory mediators.[91] However, the role of fat in wound healing is not clear.[75] Moreover, diets high in fat and alcohol can contribute to increased inflammation and dysregulation of wound healing.[82]

Proteins play the most critical role throughout the whole healing process.[112] Proteins make up the major structural components of the body. They are needed for the growth, maintenance, and repair of all body tissues. The cells that make up the immune system, including leukocytes, macrophages, lymphocytes, monocytes, and phagocytic cells, are composed primarily of proteins. These cells each play an essential role during the inflammatory response phase, which initiates the healing process. Protein is also necessary for the synthesis of enzymes involved in wound healing, many hormones, antibodies that help fight infection, and proliferation of connective tissue cells and collagen.[75] Protein, along with sufficient caloric intake, is recommended.[91]

Vitamins, minerals, and water are considered to be *micronutrients*, which are necessary for regulating normal body functions.[74] They do not provide energy, but without sufficient quantities of micronutrients, the energy from macronutrients cannot be utilized. Vitamin A stimulates the immune system by increasing the number of macrophages and monocytes in a wound during inflammation and has been shown to reduce effects of corticosteriod use.[91] Both Vitamins A and C enhance wound healing by stimulating epithelialization and increasing collagen deposition by fibroblasts. Vitamin C also enhances capillary formation and neutrophil activity.[97,112]

Magnesium, copper, and zinc are minerals, each of which contributes to tissue healing. Magnesium is a cofactor for enzymes necessary for protein and collagen formation and tissue growth during wound healing. Copper is important in the formation of hemoglobin. Zinc is essential in numerous aspects of cellular metabolism including immune function, DNA synthesis, and protein

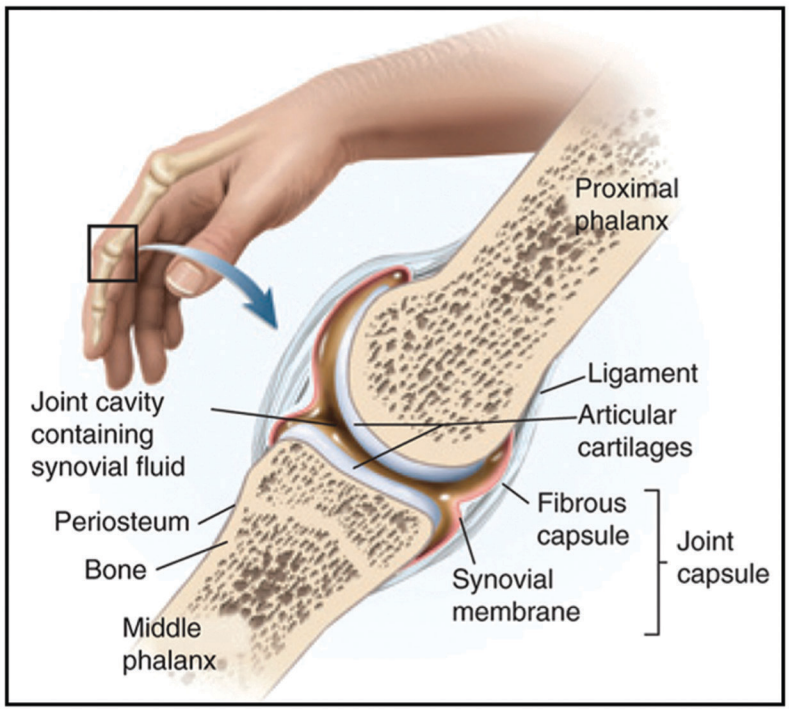


Figure 2-3. General anatomy of a synovial joint. (Reprinted with permission from Saladin K. *Anatomy & Physiology*. 6th ed. New York, NY: McGraw-Hill; 2014.)[83]

and collagen synthesis.[75] Zinc supplementation is only recommended if a deficiency exists.[91]

Water is the most essential of all the nutrients. Ensuring adequate water intake is necessary for perfusion and oxygenation of healthy and healing tissues. Prevention and treatment of skin breakdown requires optimal fluid intake.[97]

INJURIES TO ARTICULAR STRUCTURES

Before discussing injuries to the various articular structures, a review of joint structure is in order[58] (Figure 2-3). All synovial joints are composed of two bones that articulate with one another to allow motion in one or more places. The articulating surfaces of the bone are lined with a very thin, smooth, cartilaginous covering called a hyaline cartilage. All joints are entirely surrounded by a thick, ligamentous joint capsule. The inner surface of this joint capsule is lined by a very thin synovial membrane that is highly vascularized and innervated. The synovial membrane produces synovial fluid, the functions of which include lubrication, shock absorption, and nutrition of the joint.[58]

Some joints contain a thick fibrocartilage called a *meniscus*. The knee joint, for example, contains two wedge-shaped menisci that deepen the articulation and provide shock absorption in that joint. Finally, the main structural support and joint stability is provided by the ligaments, which may be either thickened portions of a joint capsule or totally separate bands.

Ligament Sprains

Ligaments are composed of dense connective tissue arranged in parallel bundles of collagen composed of rows of fibroblasts. Although bundles are arranged in parallel, not all collagen fibers are arranged in parallel. Ligaments and tendons are very similar in structure. However, ligaments are usually more flattened than tendons, and collagen fibers in ligaments are more compact. The anatomical positioning of the ligaments determines in part what motions a joint can make.

A sprain involves damage to a ligament that provides support to a joint. A ligament is a tough, relatively inelastic band of tissue that connects one bone to another. A ligament's primary function is three-fold: to provide stability to a joint, to provide control of the position of one articulating bone to another during normal joint motion, and to provide proprioceptive input or a sense of joint position through the function of free nerve endings or mechanoreceptors located within the ligament.[74]

If stress is applied to a joint that forces motion beyond its normal limits or planes of movement, injury to the ligament is likely (Figure 2-4). The severity of damage to the ligament is classified in many different ways; however, the most commonly used system involves three grades (degrees) of ligamentous sprain:

1. Grade 1 sprain: There is some stretching or perhaps tearing of the ligamentous fibers, with little or no joint instability. Mild pain, little swelling, and joint stiffness might be apparent.
2. Grade 2 sprain: There is some tearing and separation of the ligamentous fibers and moderate instability of

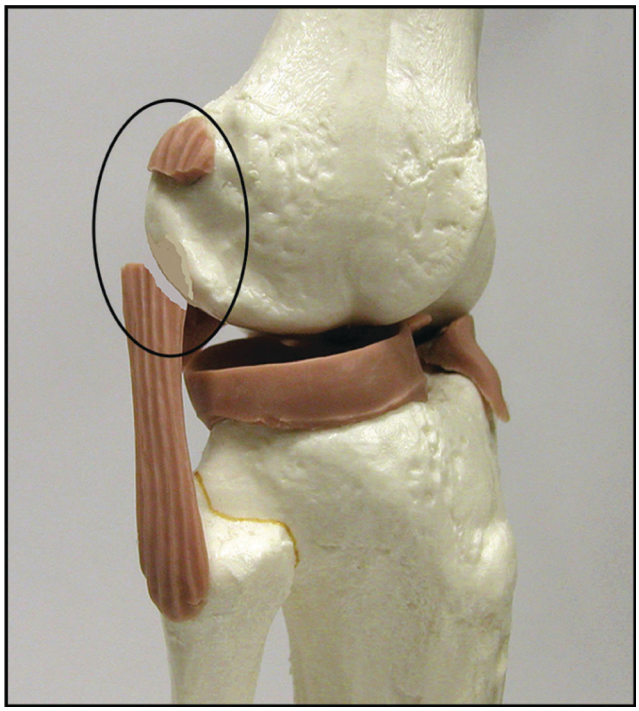


Figure 2-4. Grade 3 ligament sprain in the knee joint.

the joint. Moderate-to-severe pain, swelling, and joint stiffness should be expected.

Clinical Decision-Making Exercise 2-5

A basketball player twisted his ankle today in practice. The mechanism and the location of pain suggest an inversion sprain. There is gross laxity with the anterior drawer test and talar tilt test. The swelling is severe and profuse over the lateral side of the ankle. The athlete is incapable of dorsiflexion and has only a few degrees of plantar flexion. He is experiencing very little pain. How would you grade the severity of this injury?

3. Grade 3 sprain: There is total rupture of the ligament, manifested primarily by gross instability of the joint. Severe pain might be present initially, followed by little or no pain because of total disruption of nerve fibers. Swelling might be profuse, and thus the joint tends to become very stiff some hours after the injury. A third-degree sprain with marked instability usually requires some form of immobilization lasting several weeks. Frequently, the force producing the ligament injury is so great that other ligaments or structures surrounding the joint are also injured. With cases in which there is injury to multiple joint structures, surgical repair reconstruction may be necessary to correct an instability.

Clinical Decision-Making Exercise 2-6

Why is it likely that an athlete with a grade 3 ligament sprain initially will experience little pain, relative to the severity of the injury?

Physiology of Ligament Healing

The healing process in the sprained ligament follows the same course of repair as with other vascular tissues. Immediately after injury and for about 72 hours, there is a loss of blood from damaged vessels and attraction of inflammatory cells into the injured area. If a ligament is sprained outside of a joint capsule (extra-articular ligament), bleeding occurs in a subcutaneous space. If an intra-articular ligament is injured, bleeding occurs inside of the joint capsule until either clotting occurs or the pressure becomes so great that bleeding ceases.

During the next 6 weeks, vascular proliferation with new capillary growth begins to occur along with fibroblastic activity, resulting in the formation of a fibrin clot. It is essential that the torn ends of the ligament be reconnected by bridging this clot. Synthesis of collagen and ground substance of proteoglycan as constituents of an intracellular matrix contributes to the proliferation of the scar that bridges between the torn ends of the ligament. This scar initially is soft and viscous, but it eventually becomes more elastic. Collagen fibers are arranged in a random woven pattern with little organization. Gradually, there is a decrease in fibroblastic activity, a decrease in vascularity, and an increase to a maximum in collagen density of the scar.[44] Failure to produce enough scar and failure to reconnect the ligament to the appropriate location on a bone are the two reasons why ligaments are likely to fail.

During the next several months, the scar continues to mature, with the realignment of collagen occurring in response to progressive stresses and strains. The maturation of the scar may require as long as 12 months to complete.[44] The exact length of time required for maturation depends on mechanical factors such as apposition of torn ends and length of the period of immobilization.

Factors Affecting Ligament Healing

Surgically repaired extra-articular ligaments have healed with decreased scar formation and are generally stronger than unrepaired ligaments initially, although this strength advantage might not be maintained as time progresses. Unrepaired ligaments heal by fibrous scarring effectively lengthening the ligament and producing some degree of joint instability. With intra-articular ligament tears, the presence of synovial fluid dilutes the hematoma, thus preventing formation of a fibrin clot and spontaneous healing.[46]

Data from several studies indicate that actively exercised ligaments are stronger than those that are immobilized. Ligaments that are immobilized for several weeks after injury tend to decrease in tensile strength and also exhibit weakening of the insertion of the ligament to bone.[74] Thus, it is important to minimize periods of immobilization and progressively stress the injured ligaments while exercising caution relative to biomechanical considerations for specific ligaments.[44]

It is not likely that the inherent stability of the joint provided by the ligament before injury will be regained. Thus, to restore stability to the joint, the other structures that surround that joint (primarily muscles and their tendons) must be strengthened. The increased muscle tension provided by resistance training can improve stability, neuromuscular control, and function of the injured joint.[10, 44]

Cartilage Damage

Cartilage is a type of rigid connective tissue that provides support and acts as a framework in many structures. It is composed of chondrocyte cells contained in small chambers called *lacunae*, surrounded completely by an intracellular matrix. The matrix consists of varying ratios of collagen and elastin and a ground substance made of proteoglycans and glycosaminoglycans, which are nonfibrous protein molecules. These proteoglycans act as sponges and trap large quantities of water, which allow cartilage to spring back after being compressed.[36] Cartilage has a poor blood supply, thus healing after injury is very slow. There are three types of cartilage. Hyaline cartilage is found on the articulating surfaces of bone and in the soft part of the nose. It contains large quantities of collagen and proteoglycan. Fibrocartilage forms the intervertebral disc and menisci located in several joint spaces. It has greater amounts of collagen than proteoglycan and is capable of withstanding a great deal of pressure. Elastic cartilage is found in the auricle of the ear and the larynx. It is more flexible than the other types of cartilage and consists of collagen, proteoglycan, and elastin.[83]

Osteoarthrosis is a degenerative condition of bone and cartilage in and about the joint (Figure 2-5). Arthritis is defined as primarily an inflammatory condition with possible destruction and associated pain.[84] Arthrosis is primarily a degenerative process with destruction of cartilage, remodeling of bone, and possible secondary inflammatory components.

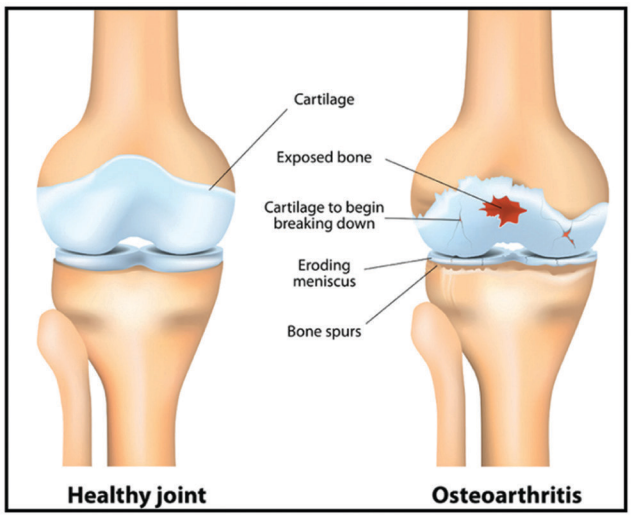


Figure 2-5. Osteoarthritis is a degeneration and erosion of the hyaline cartilage.

Cartilage fibrillation is the releasing of fibers or groups of fibers and ground substance into the joint.[38] Peripheral cartilage that is not exposed to weightbearing or compression–decompression mechanisms is particularly likely to fibrillate. Fibrillation is typically found in the degenerative process associated with poor nutrition or disuse. This process can then extend even to weightbearing areas, with progressive destruction of cartilage proportional to stresses applied on it. When forces are increased, thus increasing stress, osteochondral or subchondral fractures can occur. Concentration of stress on small areas can produce pressures that overwhelm the tissue's capabilities.[37] Typically, lower limb joints have to handle greater stresses, but their surface area is usually larger than the surface area of upper limbs. The articular cartilage is protected to some extent by the synovial fluid, which acts as a lubricant. It is also protected by the subchondral bone, which responds to stresses in an elastic fashion. It is more compliant than compact bone, and microfractures can be a means of force absorption. Trabeculae might fracture or might be displaced due to pressures applied on the subchondral bone. In compact bone, fracture can be a means of defense to dissipate force. In the joint, forces might be absorbed by joint movement and eccentric contraction of muscles.[28, 121]

In the majority of joints where the surfaces are not congruent, the applied forces tend to concentrate in certain areas, which increases joint degeneration. Osteophytosis occurs as a bone attempts to increase its surface area to decrease contact forces. People typically describe this growth as "bone spurs." Chondromalacia is the nonprogressive transformation of cartilage with irregular surfaces and areas of softening. It typically occurs first in nonweightbearing areas and may progress to areas of excessive stress.[31]

In physically active individuals, certain joints may be more susceptible to a response resembling osteoarthrosis.[35] The proportion of body weight resting on the joint, the pull of the musculotendinous unit, any significant external force applied to the joint, and comorbidities like obesity or diabetes[98] are predisposing factors. Altered joint mechanics caused by laxity or previous trauma are also factors that come into play.[60] The intensity of forces can be great, as in the hip, where the previously mentioned factors can produce pressures or forces 4 times that of body weight and up to 10 times that of body weight on the knee.

Typically, muscle forces generate more stress than body weight itself. Particular injuries are conducive to osteoarthritic changes such as subluxation and dislocation of the patella, osteochondritis dissecans, recurrent synovial effusion, and hemarthrosis. Also, ligamentous injuries can bring about a disruption of proprioceptive mechanisms, loss of adequate joint alignment, and meniscal damage in the knees with removal of the injured meniscus.[64] Other

factors that have an impact are loss of full ROM, poor muscular power and strength, and altered biomechanics of the joint. Spurring and spiking of bone are not synonymous with osteoarthrosis if the joint space is maintained and the cartilage lining is intact. It may simply be an adaptation to the increased stress of physical activity.[38]

Physiology of Cartilage Healing

Cartilage has a relatively limited healing capacity. When chondrocytes are destroyed and the matrix is disrupted, the course of healing is variable, depending on whether damage is to cartilage alone or also to subchondral bone. Injuries to the articular cartilage alone fail to elicit clot formation or a cellular response. The conversion of some hypertrophic chondrocytes into osteoblasts has been discovered, but the process of how is still being researched. Hypertrophic chondrocyte apoptosis is not the sole contributing factor to failed healing.[41,47,50]

If subchondral bone is also affected, inflammatory cells enter the damaged area and formulate granulation tissue. In this case, the healing process proceeds normally, with differentiation of granulation tissue cells into chondrocytes occurring in about 2 weeks. At about 2 months, normal collagen has been formed.

Injuries to the knee articular cartilage are extremely common, and until recently, methods for treatment did not produce good long-term outcomes.[16] A better understanding of how articular cartilage responds to injury has produced various techniques that result in long-term success.[33,100] Autologous chondrocyte implantation uses a patient's own harvested cartilage cells that are grown ex vivo, and reimplanted in a full-thickness articular surface defect. Results are available with up to 10 years of follow up, and more than 80% of patients have shown improvement with relatively few complications. Mesenchymal stem cells (MSCs) and hydrogels have also proven successful in repairing chondral bone.[96,109] The use of MSCs, found in connective tissue, does not require lab growth and can be implanted with a one-step process.[109] Specific hydrogel materials are still being researched, but this approach has many advantages.[96]

INJURIES TO BONE

Bone is a type of connective tissue consisting of both living cells and minerals deposited in a matrix (Figure 2-6). Each bone consists of 3 major components. The epiphysis is an expanded portion at each end of the bone that articulates with another bone. Each articulating surface is covered by an articular, or hyaline, cartilage. The diaphysis is the shaft of the bone. The epiphyseal, or growth plate is the major site of bone growth and elongation. Once bone growth ceases, the plate ossifies and forms the epiphyseal line. With the exception of the articulating surfaces, the bone is completely enclosed by the periosteum, a tough, highly vascularized and innervated fibrous tissue.[24]

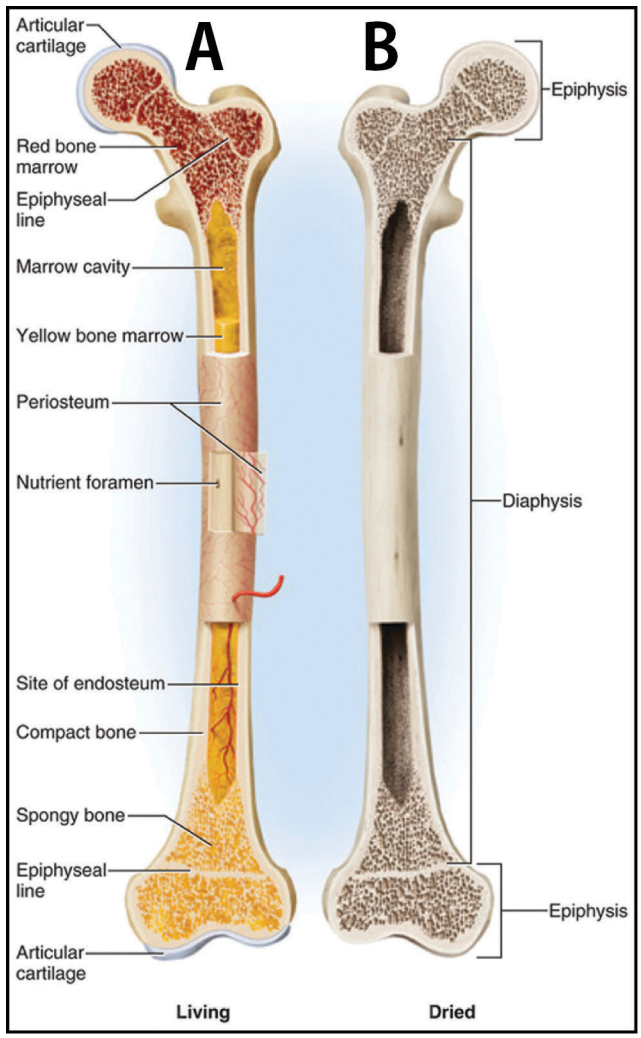


Figure 2-6. The gross structure of the long bones includes the diaphysis, epiphysis, articular cartilage, and periosteum. (Reprinted with permission from Saladin K. *Anatomy & Physiology.* 6th ed. New York, NY: McGraw-Hill; 2014.)[83]

The 2 types of bone material are cancellous (spongy) and compact (cortical). Cancellous bone contains a series of air spaces referred to as *trabeculae*, whereas cortical bone is relatively solid. Cortical bone in the diaphysis forms a hollow medullary canal in long bone, which is lined with endosteum and filled with bone marrow. Bone has a rich blood supply that facilitates the healing process after injury. Bone has the functions of support, movement, and protection. Furthermore, bone stores and releases calcium into the bloodstream and manufactures red blood cells.[105]

Bone is constantly undergoing a remodeling process in which osteoblast cells produce new bone while osteoclasts function to remodel or realign bone relative to the normal tensile forces (stresses or strains) applied to the bone.[57]

Fractures

Fractures are extremely common injuries among the athletic population. They are generally classified as being either open or closed. A closed fracture involves little or

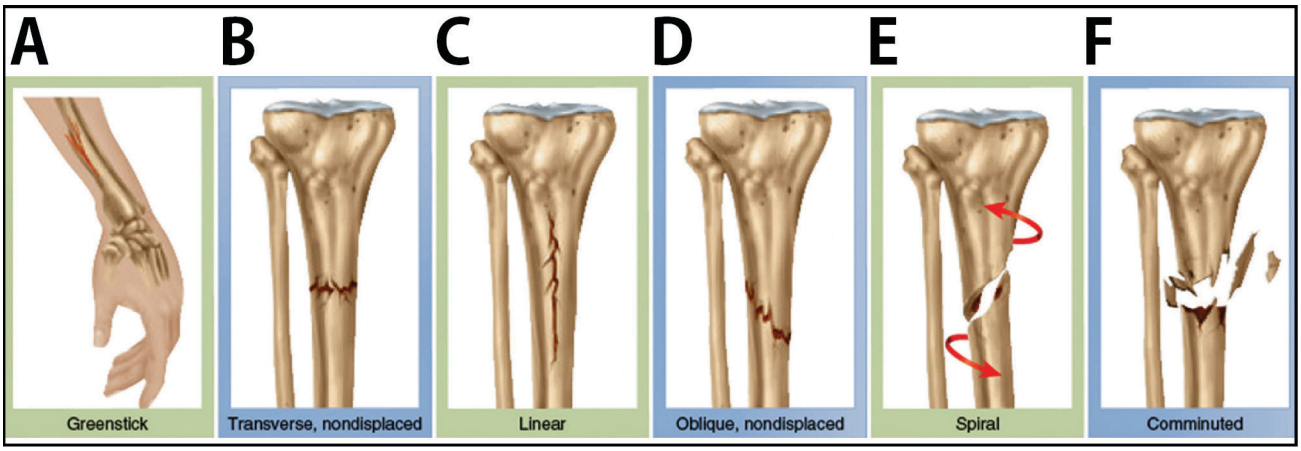


Figure 2-7. Fractures of bone. (A) Greenstick. (B) Transverse. (C) Linear. (D) Oblique. (E) Spiral. (F) Comminuted. (Reprinted with permission from Prentice WE. *Essentials of Athletic Injury Management*. 11th ed. New York, NY: McGraw-Hill; 2019.)

no displacement of bones and, thus, little or no soft tissue disruption. An open fracture involves enough displacement of the fractured ends that the bone actually disrupts the cutaneous layers and breaks through the skin. Both fractures can be relatively serious if not managed properly, but an increased possibility of infection exists in an open fracture. Fractures may also be considered complete, in which the bone is broken into at least 2 fragments, or incomplete, where the fracture does not extend completely across the bone.

The varieties of fractures that can occur include greenstick, transverse, oblique, spiral, comminuted, avulsion, and stress. A *greenstick fracture* (Figure 2-7A) occurs most often in children whose bones are still growing and have not yet had a chance to calcify and harden. It is called a *greenstick fracture* because of the resemblance to the splintering that occurs to a tree twig that is bent to the point of breaking. Because the twig is green, it splinters but can be bent without causing an actual break.

A *transverse fracture* (Figure 2-7B) involves a crack perpendicular to the longitudinal axis of the bone that goes all the way through the bone. Displacement might occur; however, because of the shape of the fractured ends, the surrounding soft tissue (*eg,* muscles, tendons, and fat) sustains relatively little damage.

A *linear fracture* runs parallel to the long axis of a bone and is similar in severity to a transverse fracture (Figure 2-7C).

An *oblique fracture* (Figure 2-7D) results in a diagonal crack across the bone and 2 very jagged, pointed ends that, if displaced, can potentially cause significant soft tissue damage. Oblique and spiral fractures are the 2 types most likely to result in compound fractures.

A *spiral fracture* (Figure 2-7E) is similar to an oblique fracture in that the angle of the fracture is diagonal across the bone. In addition, an element of twisting or rotation causes the fracture to spiral along the longitudinal axis of the bone. Spiral fractures, once common in ski injuries, occur just above the top of the boot when the bindings on the ski failed to release when the foot was rotated. These injuries are now less common as a result of improvements in equipment design.

A *comminuted fracture* (Figure 2-7F) is a serious problem that can require an extremely long time for rehabilitation. In the comminuted fracture, multiple fragments of bone must be surgically repaired and fixed with screw, wires, plates, rods, and/or pins. If a fracture of this type occurs to a weightbearing bone in the leg, a permanent discrepancy in leg length can develop.

An *avulsion fracture* occurs when a fragment of bone is pulled away at the bony attachment of a muscle, tendon, or ligament. Avulsion fractures are common in the fingers and some of the smaller bones, but they can also occur in larger bones where tendinous or ligamentous attachments are subjected to a large amount of force.

Perhaps the most common fracture resulting from physical activity is the *stress fracture*. Unlike the other types of fractures that have been discussed, the stress fracture results from overuse or fatigue rather than acute trauma.[53] Overuse creates increased stresses that eventually cause irritation and inflammation of the bone. This suppresses osteoblastic cell activity, thus the osteoblasts cannot match the need for new bone ultimately causing small microfractures of the bone. Common sites for stress fractures include the weight-bearing bones of the leg and foot. In either case, repetitive forces transmitted through the bones produce irritations and microfractures at a specific area in the bone. The pain usually begins as a dull ache that becomes progressively more painful day after day. Initially, pain is most severe during activity. However, when a stress fracture actually develops, pain tends to become worse after the activity is stopped.[70]

The biggest problem with a stress fracture is that it often does not show up on a radiograph until the overuse activity that causes the problem is eliminated and the osteoblasts begin laying down subperiosteal callus or bone, at which point a small white line, or callus, appears. However, a bone scan might reveal a potential stress fracture in as little as 2 days after onset of symptoms. If a stress fracture is suspected, the patient should stop any activity that

produces added stress or fatigue to the area for a minimum of 14 days. Stress fractures do not usually require casting but might become normal fractures that must be immobilized if handled incorrectly.[47,49] If a fracture occurs, it should be managed and rehabilitated by a qualified orthopedist and athletic trainer.

Clinical Decision-Making Exercise 2-7

A Little League player collided with the catcher when sliding home. Radiographs did not show a fracture, but a bone scan shows a hot spot. What type of fracture would you suspect this young athlete has?

Physiology of Bone Healing

Healing of injured bone tissue is similar to soft tissue healing in that all phases of the healing process can be identified, although bone regeneration capabilities are somewhat limited. However, the functional elements of healing differ significantly from those of soft tissue. Tensile strength of the scar is the single most critical factor in soft tissue healing, whereas bone has to contend with a number of additional forces, including torsion, bending, and compression.[105] Trauma to bone can vary from contusions of the periosteum to closed, nondisplaced fractures to severely displaced open fractures that also involve significant soft tissue damage. When a fracture occurs, blood vessels in the bone and the periosteum are damaged, resulting in bleeding and subsequent clot formation (Figure 2-8). Hemorrhaging from the marrow is contained by the periosteum and the surrounding soft tissue in the region of the fracture. In about 1 week, fibroblasts begin laying down a fibrous collagen network. The fibrin strands within the clot serve as the framework for proliferating vessels. Chondroblast cells begin producing fibrocartilage, creating a callus between the broken bones. At first, the callus is soft and firm because it is composed of primarily collagenous fibrin. The callus becomes firm and more rubbery as cartilage begins to predominate. Bone-producing cells called osteoblasts begin to proliferate and enter the callus, forming cancellous bone trabeculae that eventually replace the cartilage. Finally, the callus crystallizes into bone, at which point remodeling of the bone begins. The callus can be divided into 2 portions: the external callus located around the periosteum on the outside of the fracture and the internal callus found between the bone fragments. The size of the callus is proportional both to the damage and to the amount of irritation to the fracture site during the healing process. Also, during this time, osteoclasts begin to appear in the area to resorb bone fragments and clean up debris.[83,105]

The remodeling process is similar to the growth process of bone in that the fibrous cartilage is gradually replaced by fibrous bone and then by more structurally efficient lamellar bone. Remodeling involves an ongoing process during which osteoblasts lay down new bone and osteoclasts remove and break down bone according to the forces placed upon the healing bone.[24] Wolff's law maintains that a bone will adapt to mechanical stresses and strains by changing size, shape, and structure.[116] Therefore, once the cast is removed, the bone must be subjected to normal stresses and strains so that tensile strength can be regained before the healing process is complete.[55,57]

The time required for bone healing is variable and based on a number of factors, such as severity of the fracture, site of the fracture, extensiveness of the trauma, and age of the patient. Normal periods of immobilization range from as short as 3 weeks for the small bones in the hands and feet to as long as 8 weeks for the long bones of the upper and lower extremities. In some instances, such as fractures in the 4 small toes, immobilization might not be required for healing. The healing process is certainly not complete

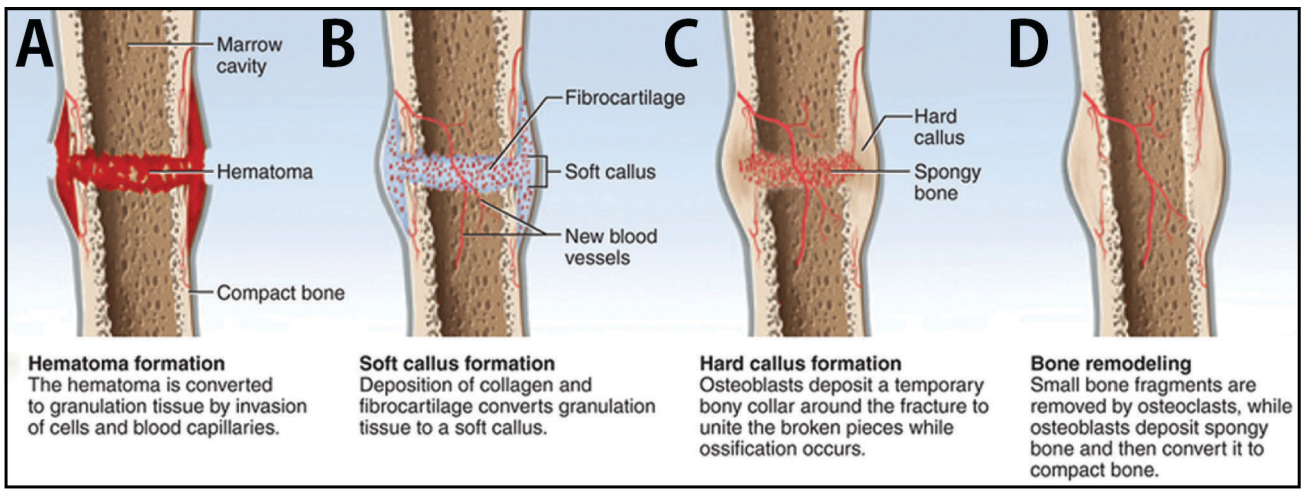


Figure 2-8. The healing of a fracture. (A) Blood vessels are broken at the fracture line; the blood clots and forms a fracture hematoma. (B) Blood vessels grow into the fracture and a fibrocartilage soft callus forms. (C) The fibrocartilage becomes ossified and forms a bony callus made of spongy bone. (D) Osteoclasts remove excess tissue from the bony callus and the bone eventually resembles its original appearance. (Reprinted with permission from Saladin K. *Anatomy & Physiology*. 6th ed. New York, NY: McGraw-Hill; 2014.)[83]