10

Tissue Response to Injury

Objectives

When you finish this chapter you should be able to

- Contrast the three phases of the healing process.
- Classify the physiological events that must take place during each phase of healing.
- Identify those factors that may impede the healing process.
- Discuss treatment techniques for modifying soft tissue healing, including using anti-inflammatory medications, therapeutic modalities, exercise rehabilitation, and platelet-rich plasma injections.
- Discuss the healing process relative to various soft-tissue structures, including cartilage, ligament, muscle, tendon, and nerve.
- Describe the healing process as it occurs in bone.
- Formulate a management plan for treating acute fractures.
- Define pain and discuss the various types of pain.
- Understand the neurophysiology of pain.
- Differentiate among the three mechanisms of pain control.
- Examine the various techniques for assessing pain.

Outline

Key Terms

margination
leukocytes
diapedesis
exudate
neutrophils
phagocytes
vasoconstriction
macrophages
lymphocytes
fibroblasts
fibroplasia
collagen
proteoglycans
glycosaminoglycans
microtears
macrotears
NSAIDs
prolotherapy
platelet-rich plasma (PRP)
avascular necrosis
trigger points
nociceptors

Connect Highlights

Visit connect.mcgraw-hill.com for further exercises to apply your knowledge:

- Clinical application scenarios covering physiological events that occur during healing, healing process in bone, and assessment of pain
- Click-and-drag questions covering tissue response to injury, inflammatory response, and soft-tissue healing
- Multiple-choice questions covering healing process, management of acute injuries, techniques and mechanisms for assessing pain, and factors that impede healing
- Selection questions covering factors that impede healing and treatment of inflammation

THE HEALING PROCESS

It is essential for the athletic trainer to possess an in-depth understanding of the healing process. The healing process consists of three phases: the inflammatory response phase, the fibroblastic repair phase, and the maturation-remodeling phase. The athletic trainer should recognize both the sequence and the time frames for these phases of healing and realize that certain physiological events must occur during each of the phases. Anything that an athletic trainer does that interferes with this healing process will likely slow the return to full activity. The healing process must have an opportunity to accomplish what it is supposed to. At best, the goal of the athletic trainer should be to try to create an environment that is conducive to the healing process. There is little that can be done to speed up the process physiologically, but there are many things that may be done during rehabilitation to impede healing. Although the phases of healing are often discussed as three separate entities, the healing process is a continuum. Phases of the healing process overlap one another and have no definitive beginning or end points (Figure 10–1).

10–1 Clinical Application Exercise

A volleyball player has sprained her ankle just 2 days prior to the beginning of the conference tournament. The athlete, her parents, and her coach are extremely concerned that she is going to miss the tournament and want to know if anything can be done to help her get well more quickly.

? What can the athletic trainer tell this patient about the healing process?

Inflammatory Response Phase

Once a tissue is injured, the process of healing begins immediately (Figure 10–2A).[17,44] The destruction of tissue produces direct injury to the cells of the various soft tissues.[45] Cellular injury results in altered metabolism and the liberation of chemical mediators that initiate the inflammatory response (Figure 10–3). It is characterized symptomatically by redness (*rubor*), swelling (*tumor*), tenderness and pain (*dolor*), increased temperature (*calor*), and loss of function (*functio laesa*).[38] *This initial inflammatory response is critical to the entire healing process. If this response does not accomplish what it is supposed to, or if it does not subside, normal healing cannot take place.*[15]

Signs of inflammation:

- Redness (rubor)
- Swelling (tumor)
- Tenderness (dolor)
- Increased temperature (calor)
- Loss of function (functio laesa)

Chemical Mediators The events in the inflammatory response are initiated by a series of interactions involving several chemical mediators.[16] 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. Three chemical mediators, *histamine, leukotrienes,* and *cytokines,* are important in limiting the amount of exudate, and thus swelling, after injury.[3] 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. Leukotrienes and prostaglandins are responsible for **margination,** in which **leukocytes** (neutrophils and macrophages) adhere along the cell walls (Figure 10–2B). They also increase cell permeability locally, thus affecting the passage of fluid, proteins, and neutrophils through cellwalls via **diapedesis** to form **exudate** in the extravascular spaces. Therefore, vasodilation and active hyperemia are important in exudate (plasma) formation and in supplying **neutrophils** to the injured area. As swelling continues and the extravascular pressure increases, the vascular flow to and the lymphatic flow from the area are decreased. The amount of swelling that occurs is directly related to the extent of vessel damage. Cytokines—in particular,

Chemical mediators:

- Histamine
- Leukotrienes
- Cytokines

margination Neutrophils and macrophages line up along the cell wall.

leukocytes Phagocytic cells.

diapedesis Movement of white blood cells out of small arterial vessels.

exudate Accumulation of fluid that penetrates through vessel walls into and joining extravascular space.

neutrophils A type of leukocyte.

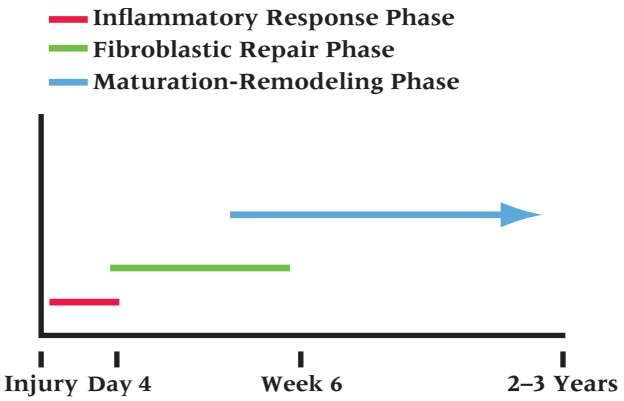


FIGURE 10–1 The three phases of the healing process fall along a continuum.

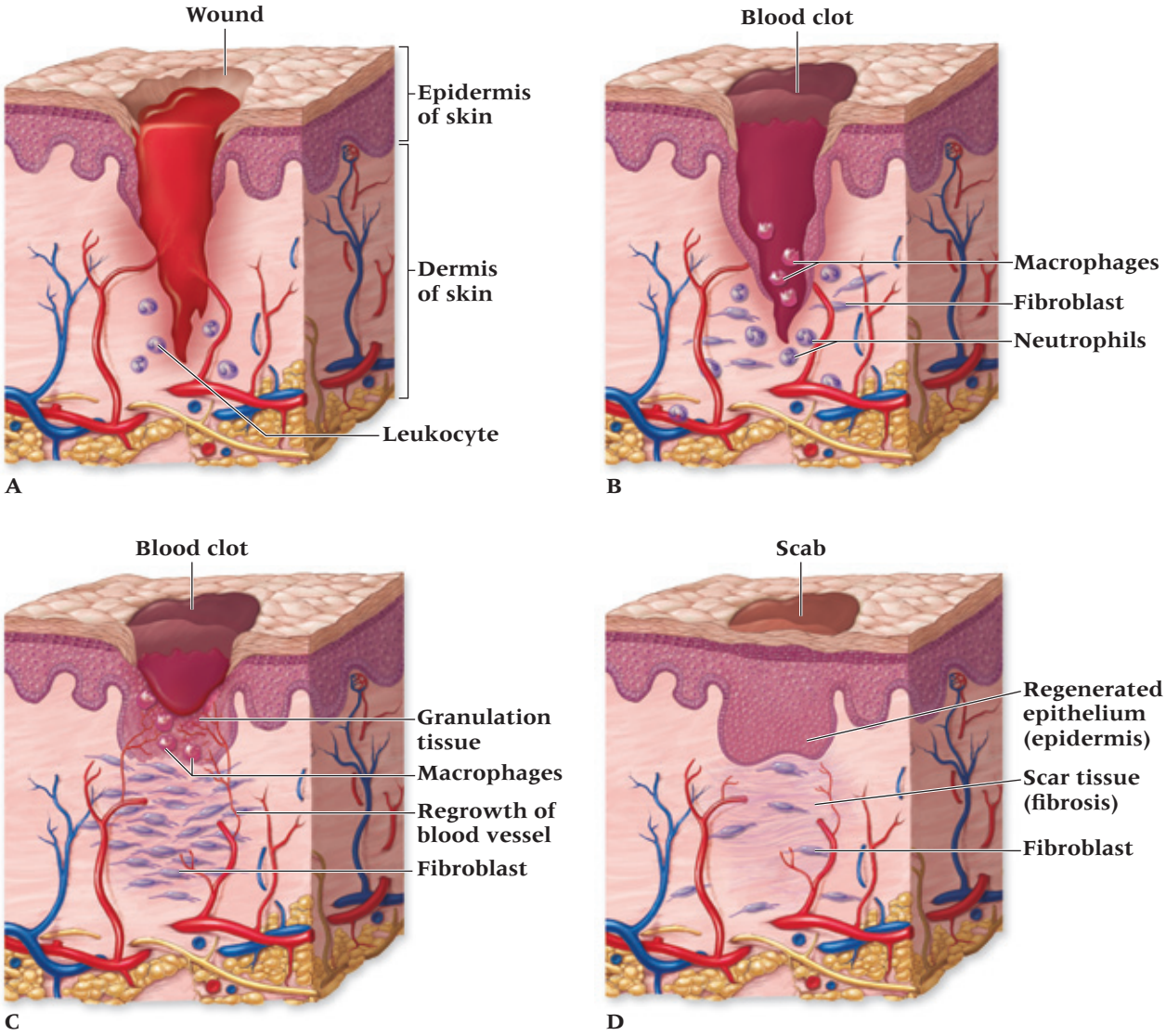


FIGURE 10–2 Initial injury and inflammatory response phase of the healing process. **(A)** Cut blood vessels bleed into the wound. **(B)** Blood clot forms, and leukocytes clean the wound. **(C)** Blood vessels regrow, and granulation tissue forms in the fibroblastic repair phase of the healing process. **(D)** Epithelium regenerates, and connective tissue fibrosis occurs in the maturation-remodeling phase of the healing process.

chemokines and interleukin, are the primary regulators of leukocyte traffic and help attract **phagocytes** to the site of inflammation.[16] Responding to the presence of chemokines, macrophages and leukocytes migrate to the site of inflammation within a few hours.

phagocytes Neutrophils, macrophages, and leukocytes that ingest microorganisms, other cells, and foreign particles.

Vascular Reaction The vascular reaction is controlled by chemical mediators and involves vascular spasm, the formation of a platelet plug, blood coagulation, and the growth of fibrous tissue.[22] The immediate vascular response to tissue damage is **vasoconstriction** of the vascular walls in the vessels leading away from the site of injury that lasts for approximately 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 due to vasodilation. 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. Eventually, there is stagnation and stasis.[23] The initial effusion of blood and plasma lasts for 24 to 36 hours.

vasoconstriction Decrease in diameter of a blood vessel.

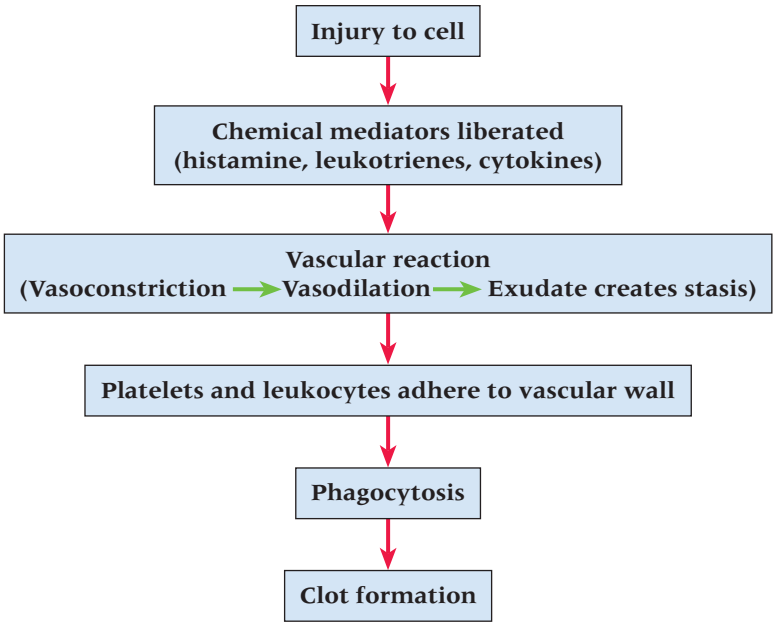


FIGURE 10–3 Inflammatory response sequence.

Function of Platelets 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, eventually forming a plug. These plugs obstruct local lymphatic fluid drainage and thus localize the injury response.[15]

Formation of a Clot 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.[52] Clot formation begins around 12 hours after injury and is completed within 48 hours.

Blood coagulation:

Thromboplastin
↓
Prothrombin
↓
Thrombin
↓
Fibrinogen
↓
Insoluble fibrin clot

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 approximately 2 to 4 days after initial injury.

Chronic Inflammation A distinction must be made between the acute inflammatory response as previously described and chronic inflammation.[47] Chronic inflammation occurs when the acute inflammatory response does not respond sufficiently to eliminate the injuring agent and restore tissue to its normal physiological state. Thus, only low concentrations of the chemical mediators are present. The neutrophils that are normally present during acute inflammation are replaced by **macrophages, lymphocytes, fibroblasts,** and plasma cells.[47] As this low-grade inflammation persists, damage occurs to connective tissue, resulting in tissue necrosis and fibrosis, prolonging the healing and repair 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.[47] The specific mechanisms that cause an insufficient acute inflammatory response are unknown, but they appear to be related to situations that involve overuse or overload with cumulative microtrauma to a particular structure.[15,23] There is

Chronic inflammation occurs from repeated acute microtraumas and overuse.

macrophages Phagocytic cells of the immune system.

lymphocytes Cells that are the primary means of providing the body with immune capabilities.

fibroblasts Cells that produce collagen and elastin.

no specific time frame in which the acute inflammation transitions to chronic inflammation. It does appear that chronic inflammation is resistant to both physical and pharmacological treatments.[18]

Fibroblastic Repair Phase

During the fibroblastic repair 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 (see Figure 10–2C).[22] The period of scar formation, referred to as **fibroplasia,** begins within the first few days after injury and may last for as long as 4 to 6 weeks. During this period, many of the signs and symptoms associated with the inflammatory response subside. The patient may 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.[44]

fibroplasia Period of scar formation.

During this phase, the growth of endothelial capillary buds into the wound is stimulated by a lack of oxygen, after which the wound is capable of healing aerobically. Along with increased oxygen delivery comes an increase in blood flow, which delivers nutrients essential for tissue regeneration in the area.[3]

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.

collagen A strong, fibrous protein found in connective tissue.

Granulation tissue:

- Fibroblasts
- Collagen
- Capillaries

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 the sixth or seventh day, fibroblasts also begin producing collagen fibers that are deposited in a random fashion throughout the forming scar. There are at least 16 types of collagen, but 80 to 90 percent of the collagen in the body consists of Types I, II, and III. Type I collagen is found in skin, fasciae, tendon, bone, ligaments, cartilage, and interstitial tissues; Type II can be found in hyaline cartilage and vertebral disks; 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.[37] 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 to signal the beginning of the maturation phase.

Extracellular matrix:

- Collagen
- Elastin
- Ground substance
- Proteoglycans
- Glycosaminoglycans

proteoglycans Molecules made of protein and carbohydrate.

glycosaminoglycans Carbohydrates that partially compose proteoglycans.

This normal sequence of events in the repair phase leads to the formation of minimal scar tissue. Occasionally, a persistent inflammatory response and continued release of inflammatory products promotes extended fibroplasia and excessive fibrogenesis that can lead to irreversible tissue damage.[12] Fibrosis can occur in synovial structures, as with adhesive capsulitis in the shoulder; in extraarticular tissues, such as tendons and ligaments; in bursae; or in muscle.

Maturation-Remodeling Phase

The maturation-remodeling phase of healing is a long-term process (Figure 10–2D). 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. It involves a decrease in Type III collagen fibers and an increase in Type I fibers.[12] Ongoing breakdown and synthesis of collagen occur with a steady increase in the tensile strength of the scar matrix as well as a decrease in capillaries in that scar. With increased stress and strain, the collagen fibers realign in a position of maximum efficiency parallel to the lines of tension. The tissue gradually assumes normal appearance and function, although a scar is rarely as strong as the normal uninjured tissue. Usually, by the end of approximately 3 weeks, a firm, strong, contracted, nonvascular scar exists. The maturation phase of healing may require several years to be complete.

10–2 Clinical Application Exercise

A football player sustains a grade 2 medial collateral ligament sprain in his left knee. The athlete expresses concern with prolonged immobilization because he does not want to lose strength.

? What methods can be used to prevent atrophy from occurring but still allow healing to take place?

The 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.[37] Therefore, it is critical that injured structures be exposed to progressively increasing loads throughout the rehabilitative process.[25]

Controlled mobilization is superior to immobilization for scar formation, revascularization, muscle regeneration, and reorientation of muscle fibers and tensile properties in animal models.[29] 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.[17] Generally, clinical signs and symptoms disappear at the end of this phase.

As the remodeling phase begins, aggressive active range of motion and strengthening exercises should be incorporated to facilitate tissue remodeling and realignment.[48] To a great extent, pain dictates the rate of progression. With initial injury, pain is intense and 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 indicates that the load is too great for the level of tissue repair or remodeling. The athletic trainer must be aware of the time required for the healing process and realize that being overly aggressive can interfere with that process.

10–3 Clinical Application Exercise

A wrestler receives a sudden twist to his right shoulder, causing a grade 2 strain to the teres minor muscle.

? What hemodynamic changes occur in the first hour of this acute injury?

Factors That Impede Healing

Extent of Injury The nature or amount of the inflammatory response is determined by the extent of the tissue injury. **Microtears** of 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.[16]

microtears Overuse.

macrotears Acute trauma.

Edema The increased pressure caused by swelling retards the healing process, causes separation of tissues, inhibits neuromuscular control, produces reflexive neurological changes, and impedes nutrition in the injured part. Edema is best controlled and managed during the initial first-aid management period, as described previously.[32]

Hemorrhage Bleeding occurs with even the smallest amount of damage to the capillaries. Bleeding produces the same negative effects on healing as does the accumulation of edema, and its presence produces additional tissue damage and thus exacerbation of the injury.[52]

Poor Vascular Supply Injuries to tissues with a poor vascular supply heal poorly and slowly. This response is likely related to a failure in the initial delivery of phagocytic cells and fibroblasts necessary for scar formation.[52]

Separation of Tissue Mechanical separation of tissue can significantly affect the course of healing. A wound that has smooth edges that are 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.[9,52]

10–4 Clinical Application Exercise

A patient sustained a grade 2 lateral ankle sprain 3 weeks ago. It was given proper immediate and follow-up care.

? What repair has taken place during this time?

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

Atrophy Wasting away of muscle tissue begins immediately with injury. Strengthening and early mobilization of the injured structure retard atrophy.[9]

Corticosteroids The 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.[16]

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 and cause excessive granulation tissue, and frequently causes large, deformed scars.[16]

Humidity, Climate, and Oxygen Tension Humidity significantly influences the process of epithelization. Occlusive dressings stimulate 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 allows the necrotic debris to more easily 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 aging. Degenerative diseases, such as diabetes and arteriosclerosis, also become a concern for older patients and may affect wound healing.[43] Nutrition is important for wound healing. In particular, vitamins C (collagen synthesis and immune system), K (clotting), and A (immune system); zinc for the enzyme systems; and amino acids play critical roles in the healing process.[28] Meeting the Dietary Recommended Intake (DRI) for vitamins is sufficient for wound healing.

10–5 Clinical Application Exercise

A patient complains of a swollen ankle that never became completely resolved since he sustained a sprain 9 months ago.

? What is the reason for this chronic swelling?

SOFT-TISSUE HEALING

Cell Structure and Function

All organisms, from the simplest to the most complex, are composed of cells (Figure 10–4). The properties of a specific soft tissue of the body are derived from the structure and function of the cells. Individual cells contain a *nucleus* surrounded by *cytoplasm* and are enclosed by a *cell membrane* that selectively allows substances to enter and leave the cell. The nucleus contains *chromosomes*, which consist of DNA and protein. The functional and structural elements within the cell are called *organelles* and include *mitochondria, ribosomes, endoplasmic reticulum, centrioles, Golgi apparatusus,* and *microtubules*.[37]

All the tissues of the body can be defined as soft tissue except for bone. The human body has four types of soft tissue: epithelial tissue, which consists of the skin and the lining of vessels and many organs; connective tissue, which consists of tendons, ligaments, cartilage, fat, and blood vessels; muscle, which can be skeletal (striated), cardiac, or smooth; and nervous tissue, which consists of the brain, spinal cord, and nerves.[37]

Soft tissue can undergo adaptations as a result of healing and of the rehabilitative process following injury.[13] Soft-tissue adaptations include the following:

- Metaplasia—coversion of one kind of tissue into a form that is not normal for that tissue.
- Dysplasia—abnormal development of tissue
- Hyperplasia—excessive proliferation of normal cells in the normal tissue arrangement.
- Atrophy—a decrease in the size of tissue due to cell death and resorption or decreased cell proliferation.
- Hypertrophy—an increase in the size of a tissue without necessarily increasing the number of cells.

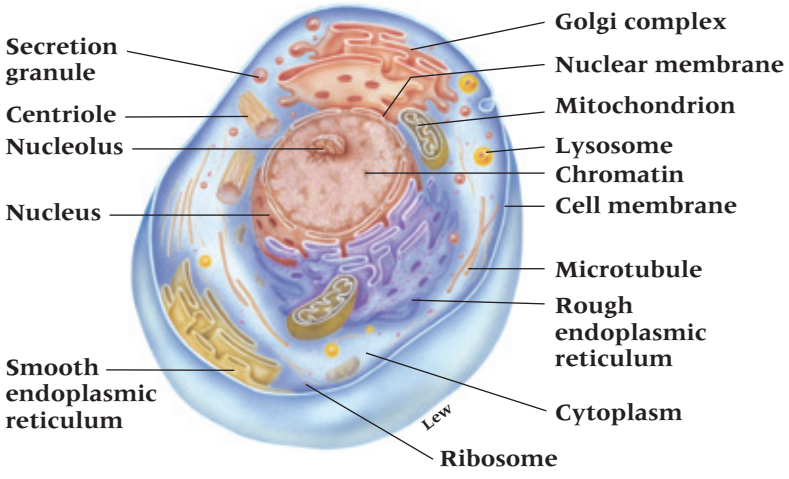


FIGURE 10–4 Structure of a cell.

Cartilage Healing

Cartilage has a relatively limited healing capacity.[30] 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.[46] Injuries to articular cartilage alone fail to elicit clot formation or a cellular response. For the most part, the chondrocytes adjacent to the injury are the only cells that show any signs of proliferation and synthesis of matrix. Thus, the defect fails to heal, although the extent of the damage tends to remain the same.[19]

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.[19] By approximately 2 months, normal collagen has been formed.[8]

Ligament Healing

The healing process in the sprained ligament follows a course of repair similar to that of other vascular tissues.[41] Immediately after injury and for approximately 72 hours, there is a loss of blood from damaged vessels and an attraction of inflammatory cells into the injured area. If a ligament is sprained outside of a joint capsule (extraarticular ligament), bleeding occurs in a subcutaneous space. If an intraarticular ligament is injured, bleeding occurs inside the joint capsule until either clotting occurs or the pressure becomes so great that bleeding ceases.[10]

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,[27] It is essential that the torn ends of the ligament be reconnected by bridging of this clot. A synthesis of collagen and a ground substance of proteoglycan in an intracellular matrix contributes to the proliferation of the scar that bridges the torn ends of the ligament. Initially, this scar is soft and viscous, but eventually it 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 a maximum increase in the collagen density of the scar.[41] Failure to produce enough scar and failure to reconnect the ligament to the appropriate location on a bone are the two reasons ligaments are likely to fail.

Over the next several months, the scar continues to mature, with the realignment of collagen occurring in response to progressive stresses and strains.[27] The maturation of the scar may require as long as 12 months to complete.[3] The exact length of time required for maturation depends on mechanical factors, such as apposition of torn ends and length of immobilization.[51]

Factors Affecting Ligament Healing Surgically repaired extraarticular ligaments have healed with decreased scar formation and are generally stronger than unrepaired ligaments initially, although this strength advantage may not be maintained as time progresses.[10] Nonrepaired ligaments heal by fibrous scarring, effectively lengthening the ligament and producing some degree of joint instability. With intraarticular ligament tears, the presence of synovial fluid dilutes the hematoma, thus preventing the formation of a fibrin clot and spontaneous healing.[51]

Several studies have shown 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 exhibit weakening of the insertion of the ligament to bone.[41] 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.[21]

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 structures that surround that joint, primarily muscles and their tendons, must be strengthened. The increased muscle tension provided by strength training can improve the stability of the injured joint.[25]

Muscle Healing

Injuries to muscle tissue involve processes of healing and repair similar to those of other tissues. Initially, there will be hemorrhage and edema followed almost immediately by phagocytosis to clear debris. Within a few days, there is a proliferation of ground substance, and fibroblasts begin producing a gel-type matrix that surrounds the connective tissue, leading to fibrosis and scarring. At the same time, myoblastic cells (satellite cells) form in the area of injury, which eventually leads to the regeneration of new myofibrils. Thus, the regeneration of both connective tissue and muscle tissue has begun.[25]

Collagen fibers undergo maturation and orient themselves along lines of tensile force according to Wolff's law. Active contraction of the muscle is critical in regaining normal tensile strength.[25,50]

Regardless of the severity of the strain, the time required for rehabilitation is fairly lengthy. In many instances, rehabilitation time for a muscle strain is longer than for a ligament sprain. These incapacitating muscle strains occur most often in the large, force-producing hamstring and quadriceps muscles of the lower extremity. The treatment of hamstring strains requires a healing period of at least 6 to 8 weeks and

a considerable amount of patience. Trying to return to activity too soon often causes reinjury to the area of the musculotendinous unit that has been strained, and the healing process must begin again.

Tendon Healing

Unlike most soft-tissue healing, tendon injuries pose a problem.[4,17] The injured tendon requires dense fibrous union of the separated ends and both extensibility and flexibility at the site of attachment.[33] Thus, an abundance of collagen is required to achieve good tensile strength. Unfortunately, collagen synthesis can become excessive, resulting in fibrosis, in which adhesions form in surrounding tissues and interfere with the gliding that is essential for smooth motion.[36] Fortunately, over a period of time the scar tissue of the surrounding tissues becomes elongated in its structure because of a breakdown in the cross-links between fibrin units and thus allows the necessary gliding motion. A tendon injury that occurs where the tendon is surrounded by a synovial sheath can be potentially devastating.[49]

A typical time frame for tendon healing would be that during the second week the healing tendon adheres to the surrounding tissue to form a single mass.[29] During the third week, the tendon separates to varying degrees from the surrounding tissues. However, the tensile strength is not sufficient to permit a strong pull on the tendon for at least 4 to 5 weeks, the danger being that a strong contraction can pull the tendon ends apart.[24,35,42]

Nerve Healing

Specialized tissue, such as nerve cells, cannot regenerate once the nerve cell dies. In an injured peripheral nerve, however, the nerve fiber can regenerate significantly if the injury does not affect the cell body.[37] The proximity of the axonal injury to the cell body can significantly affect the time required for healing. The closer an injury is to the cell body, the more difficult the regenerative process. In the case of a severed nerve, surgical intervention can markedly enhance regeneration.

For regeneration to occur, an optimal environment for healing must exist.[5] When a nerve is cut, several degenerative changes occur that interfere with the neural pathways (Figure 10–5). Within the first 3 to 5 days, the portion of the axon distal to the cut begins to degenerate and breaks into irregular segments. There is also a concomitant increase in metabolism and protein production by the nerve cell body to facilitate the regenerative process. The neuron in the cell body contains the genetic material and produces the chemicals necessary to maintain the axon. These substances cannot be transmitted to the distal part of the axon, and eventually there will be complete degeneration.[37]

In addition, the myelin portion of the Schwann cells around the degenerating axon also degenerates,

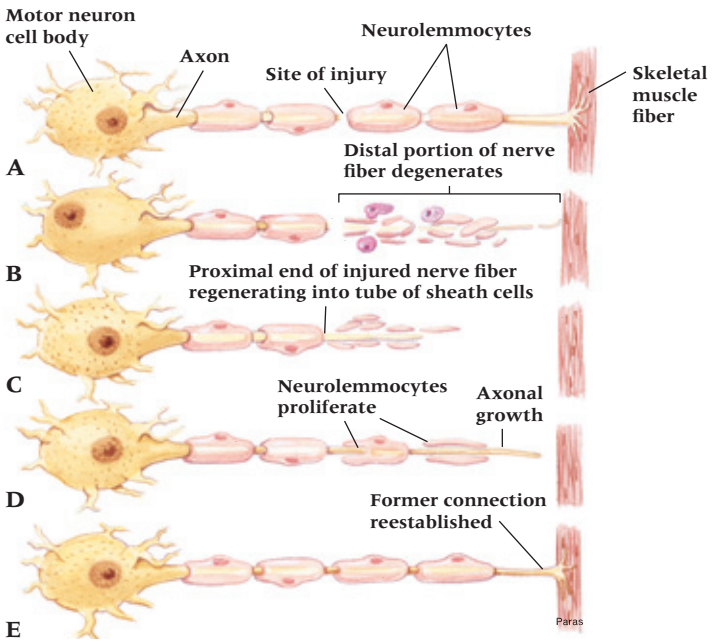


FIGURE 10–5 Neuron regeneration.
(A) If a neuron is severed through a myelinated axon, the proximal portion may survive, but **(B)** the distal portion will degenerate through phagocytosis. **(C & D)** The myelin layer provides a pathway for regeneration of the axon. **(E)** Innervation is restored.

and the myelin is phagocytized. The Schwann cells divide, forming a column of cells in place of the axon. If the cut ends of the axon contact this column of Schwann cells, the chances are good that an axon will eventually reinnervate distal structures. If the proximal end of the axon does not make contact with the column of Schwann cells, reinnervation will not occur.[37]

The axon proximal to the cut has minimal degeneration initially and then begins the regenerative process with growth from the proximal axon. Bulbous enlargements and several axon sprouts form at the end of the proximal axon. Within about two weeks, these sprouts grow across the scar that has developed in the area of the cut and enter the column of Schwann cells. Only one of these sprouts will form the new axon, while the others will degenerate. Once the axon grows through the Schwann cell columns, remaining Schwann cells proliferate along the length of the degenerating fiber and the neurolemmocytes form new myelin around the growing axon, which will eventually reinnervate distal structures.[20]

Regeneration is slow, at a rate of only 3 to 4 millimeters per day. Axon regeneration can be obstructed by scar formation due to excessive fibroplasia. Damaged nerves within the central nervous system regenerate very poorly compared with nerves in the peripheral nervous system. Central nervous system axons lack connective tissue sheaths, and the myelin-producing Schwann cells fail to proliferate.[7]

Modifying Soft-Tissue Healing

The healing process is unique in each patient. In addition, different tissues vary in their ability to regenerate. For example, cartilage regenerates to some degree from the perichondrium, striated muscle is limited in its regeneration, and peripheral nerve fibers can regenerate only if their damaged ends are opposed. Usually, connective tissue will readily regenerate, but, as is true of all tissue, this possibility is dependent on the availability of nutrients.

Age and general nutrition can play a role in healing. Older patients may be more delayed in healing than are younger patients. In a patient with a poor nutritional status, injuries may heal more slowly than normal. Patients with certain organic disorders may heal slowly. For example, blood conditions such as anemia and diabetes often inhibit the healing process because of markedly impaired collagen deposition. Many of the current treatment approaches are designed to enhance the healing process. Current treatments are anti-inflammatory medications, therapeutic modalities, exercise rehabilitation, and prolotherapy.

Methods to modify soft-tissue healing:

- Anti-inflammatory medications
- Therapeutic modalities
- Exercise rehabilitation

Anti-inflammatory Medications It is a common practice for a physician to routinely prescribe nonsteroidal anti-inflammatory drugs **(NSAIDs)** for patients who have sustained an injury.[18] These medications are certainly effective in minimizing the pain and swelling associated with inflammation and may enhance a return to full activity. However, there are some concerns that the use of NSAIDs acutely following injury may actually interfere with inflammation, thus delaying the healing process. The use of NSAIDs is further discussed in Chapter 17.

NSAIDs Nonsteroidal anti-inflammatory drugs.

Therapeutic Modalities Both cold and heat are used for different conditions. In general, heat facilitates an acute inflammatory response and cold slows the inflammatory response.[32]

A number of electrical modalities are used for the treatment of inflammation stemming from sports injuries. These procedures involve penetrating heat devices, such as shortwave and ultrasound therapy, and electrical stimulation, including transcutaneous electrical nerve stimulation (TENS) and electrical muscle stimulation (EMS)[32] (see Chapter 15).

Exercise Rehabilitation A major aim of soft-tissue rehabilitation through exercise is pain-free movement, full-strength power, and full extensibility of associated muscles. The ligamentous tissue, if related to the injury, should become pain free and have full tensile strength and full range of motion. The dynamic joint stabilizers should regain full strength and power.[18]

Immobilization of a part after injury or surgery is not always good for all injuries. When a part is immobilized over an extended period of time, adverse biochemical changes occur in collagenous tissue. Early mobilization used in exercise rehabilitation that is highly controlled may enhance the healing process[19] (see Chapter 16).

Prolotherapy Prolotherapy is a technique that involves injection of an irritant, nonpharmacological solution (e.g., dextrose, phenol, glycerine, lidocaine) into soft tissue for the purpose of increasing the inflammatory response, thus enhancing the healing process.[39] It is also referred to in the literature as proliferation therapy, proliferative injection therapy, and regenerative injection

Prolotherapy Injecting an irritant solution into a tendon or ligament to facilitate healing.

therapy. Prolotherapy has been used primarily to facilitate strengthening of weakened connective tissue (i.e., tendons and ligaments) and to reduce pain although it has also been used in treating a variety of other musculoskeletal conditions.[39] Injections are typically repeated every 3 to 6 weeks until no longer necessary. There is currently limited evidence in the literature to support the use of prolotherapy, although a number of randomized clinical trials are looking at this technique. Consequently, third-party payers are reluctant to reimburse for this therapeutic treatment.

Platelet-Rich Plasma (PRP) Injections **Platelet-rich plasma (PRP)** injections are a type of prolotherapy that uses the patient's own platelets to promote the natural healing of a variety of musculoskeletal conditions such as tendinosis, tendinitis, ligament sprains, muscle strains, injuries to fibrocartilage, osteoarthritis, and wound healing.[6,31] To prepare a PRP injection, a small amount of the patient's own blood is drawn into a vial. The blood is then spun in a centrifuge to separate the blood into its various components: plasma, platelets and white blood cells, and red blood cells. The red blood cells are drained away, and the concentrated platelets, white cells, and some plasma are centrifuged again to separate the platelet-rich plasma from the platelet-poor plasma; then an anticoagulant is added to prevent early platelet clotting. The PRP is then injected into and around the injured tissues.[34] The concentrated platelets release bioactive proteins that include growth factors and signaling proteins that stimulate wound healing and tissue repair. Growth factors are peptides secreted by many different tissues (including platelets) that activate intracellular pathways responsible for growth, differentiation, and development of cells. Specific growth factors are responsible for healing in musculoskeletal tissues.[6] The concentrated platelets can increase the growth factors by as much as eightfold.[31] The signaling proteins attract stem cells that multiply and function to repair and rebuild damaged tissue. Following an injection, there is usually an increase in pain for 5 to 10 days. The number of injections that are necessary depends upon the type of condition, severity, and the age of the patient.[34] Because this is a relatively new technique for treating musculoskeletal injuries, there are comparatively few randomized controlled trial studies that offer strong evidence supporting its effectiveness. Also, it is currently an expensive treatment, costing about $1,000 per injection.

Platelet-rich plasma (PRP) Using blood plasma that has been enriched with platelets to stimulate healing of bone and soft tissue.

BONE HEALING

Healing of injured bone tissue is similar to soft-tissue healing in that all phases of the healing process may be identified, although bone regeneration capabilities are somewhat limited. However, the functional elements of healing differ significantly from those of soft tissue. The 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.[26] Trauma to bone may 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 10–6). Hemorrhaging from the marrow is contained by the periosteum and the surrounding soft tissue in the region of the fracture. In about

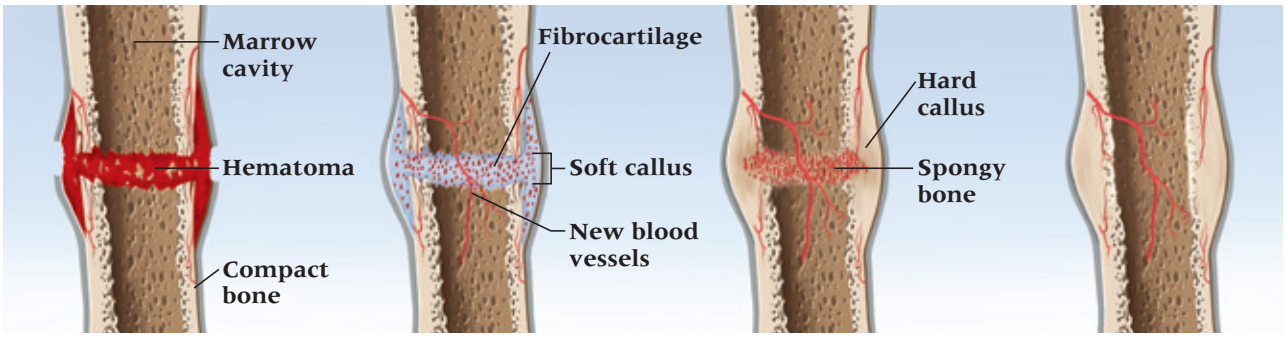


FIGURE 10–6 The healing of a fracture.
(A) Blood vessels are broken at the fracture line; the blood clots and forms a fracture hematoma. **(B)** Blood vessels grow into the fracture and a fibrocartilage soft callus forms. **(C)** The fibrocartilage becomes ossified and forms a bony callus made of spongy bone. **(D)** Osteoclasts remove excess tissue from the bony callus and the bone eventually resembles its original appearance.

one week, fibroblasts have begun 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 primarily of 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, which eventually replace the cartilage. Finally, the callus crystallizes into bone, at which point remodeling of the bone begins. The callus can be divided into two 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.[26,37]

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 on the healing bone.[26] Wolff's law maintains that a bone will adapt to mechanical stresses and strains by changing size, shape, and structure. Therefore, once the cast is removed, the bone must be subjected to normal stresses and strains, so that tensile strength may be regained before the healing process is complete.[2]

The time required for bone healing is variable and based on a number of factors, such as the severity of the fracture, site of the fracture, extensiveness of the trauma, and age of the patient.[2] Normal periods of immobilization range from as short as three weeks for the small bones in the hands and feet to as long as eight weeks for the long bones of the upper and lower extremities. In some instances—for example, the four small toes—immobilization may not be required for healing. The healing process is certainly not complete when the splint or cast is removed. Osteoblastic and osteoclastic activity may continue for 2 to 3 years after severe fractures.

Management of Acute Fractures In the treatment of acute fractures, the bones commonly must be immobilized completely until X-ray studies reveal that the hard callus has been formed. It is up to the physician to know the various types of fractures and the best form of immobilization for each fracture.[26] Fractures can keep a patient from activity for several weeks or months, depending on the nature, extent, and site of the fracture. During this period, certain conditions can seriously interfere with the healing process:[2,26,37]

10–6 Clinical Application Exercise

A field hockey player falls and sustains an acute fracture of the left humerus.

? What are the healing events typical of this acute bone fracture?

Conditions that interfere with fracture healing:

- Poor blood supply
- Poor immobilization
- Infection
- Soft tissues between severed ends of bone.

- If there is a *poor blood supply to the fractured area* and one of the parts of the fractured bone is not properly supplied by the blood, that part will die and union or healing of the fracture will not take place. This condition is known as **avascular necrosis** and often occurs in the head of the femur, the navicular bone in the wrist, the talus in the ankle, and isolated bone fragments. The condition is relatively rare among vital, healthy, young patients except in the navicular bone of the wrist.

avascular necrosis
A portion of the bone degenerates due to a poor blood supply.

- *Poor immobilization of the fracture site,* resulting from poor casting by the physician and permitting motion between the bone parts, may not only prevent proper union but also, in the event that union does transpire, cause deformity to develop.
- *Infection* can materially interfere with the normal healing process, particularly in the case of a compound fracture, which offers an ideal situation for the development of a severe streptococcal or staphylococcal infection. The increased use of antibiotics has considerably reduced the prevalence of these infections coincidental with or immediately after a fracture. The closed fracture is not immune to contamination because infections within the body or poor blood supply can render it susceptible. If the fracture site becomes and remains infected, the infection can interfere with the proper union of the bone.
- Soft tissues that become positioned between the severed ends of the bone—such as muscle, connective tissue, or other soft tissue immediately adjacent to the fracture—can prevent proper bone union, often necessitating surgical intervention.

Healing of Stress Fractures

As discussed in Chapter 9, stress fractures may be created by cyclic forces that adversely load a bone at a susceptible site. Fractures may be the result of axial compression or tension created by the pull of muscles. Stress on ligamentous and bony tissue can be either positive and increase relative strength or negative and lead to tissue weakness. Bone produces an electrical potential in response to the stress of tension and compression. As a bone bends, tension is created on its convex side along with a positive electrical charge; conversely, on the concave or compressional side, a negative electrical charge is created. Torsional forces produce tension circumferentially. Constant tension caused by axial compression or stress by muscular activity can result in an increase in bone resorption and, subsequently, a microfracture. In other words, if the osteoclastic activity is greater than the osteoblastic activity, the bone becomes increasingly susceptible to stress fractures.[14]

10–7 Clinical Application Exercise

A cross-country runner sustains a stress fracture of her left tibia. Her left leg is ¾ inch shorter than her right leg.

? ?What is a possible cause of this injury?

Like the healing of acute fractures, the healing of stress fractures involves restoring a balance of osteoclastic and osteoblastic activity. Achieving this balance requires recognition of the situation as early as possible. Stress fractures that go unhealed will eventually develop into complete cortical fractures that may, over a period of time, become displaced. A decrease in activity and the elimination of other factors in training that cause stress will allow the bone to remodel and to develop the ability to withstand stress.[14]

PAIN

Pain can be defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[1] Pain is a subjective sensation with more than one dimension and an abundance of descriptors of its qualities and characteristics. Pain is composed of a variety of human discomforts, rather than being a single entity. The perception of pain can be subjectively modified by past experiences and expectations. Much of what is done to treat patients' pain is to change their perceptions of pain. Certainly, reducing pain is an essential part of treatment. The athletic trainer's goal is to control acute pain by encouraging the body to heal through exercise designed to progressively increase functional capacity and to return the patient to full activity as swiftly and safely as possible.

Types of Pain

Pain can be described according to a number of categories, such as pain sources, acute versus chronic pain, and referred pain.[12]

Pain Sources Pain sources are cutaneous, deep somatic, visceral, and psychogenic.[11] Cutaneous pain is usually sharp, bright, and burning and can have a fast or slow onset. Deep somatic pain stems from structures such as tendons, muscles, joints, periosteum, and blood vessels. Visceral pain originates from internal organs. Visceral pain is diffused at first and later may be localized, as in appendicitis. In psychogenic pain, the individual feels pain but the cause is emotional rather than physical.[12]

10–8 Clinical Application Exercise

A butterfly swimmer has been experiencing low back pain for more than 6 months. The pain is described as aching and throbbing.

? What type of pain is this athlete experiencing?

Acute versus Chronic Pain Acute pain lasts less than 6 months. Tissue damage occurs and serves as a warning to the patient. Chronic pain, on the other hand, has a duration longer than 6 months. The International Association for the Study of Pain describes chronic pain as that which continues beyond the usual normal healing time.[8]

Referred Pain Referred pain occurs away from the actual site of irritation. This pain has been called an error in perception. Each referred pain site must also be considered unique to each individual. Symptoms and signs vary according to the nerve fibers affected. Response may be motor, sensory, or both. Four types of referred pain are myofascial, sclerotomic, myotomic, and dermatomic pain.

Myofascial Pain As discussed in Chapter 9, **trigger points** are small, hyperirritable areas within a muscle in which nerve impulses bombard the central nervous system and are expressed as a referred pain. Acute and chronic musculoskeletal pain can be caused by myofascial trigger points.[12] Such pain sites have variously been described as fibrositis, myositis, myalgia, myofasciitis, and muscular strain.

trigger points Small, hyperirritable areas within a muscle.

An active trigger point is hyperirritable and causes an obvious complaint. Pain radiating from an active

trigger point does not follow a usual area of distribution, such as sclerotomes, dermatomes, or peripheral nerves. The trigger point pain area is called the reference zone, which can be close to the point of irritation or a considerable distance from it.[12]

Sclerotomic, Myotomic, and Dermatomic Pain Deep pain may originate from sclerotomic, myotomic, or dermatomic nerve irritation or injury.[11] A sclerotome is an area of bone or fascia that is supplied by a single nerve root. Myotomes are muscles supplied by a single nerve root. Dermatomes also are in an area of skin supplied by a single nerve root.

Sclerotomic pain is deep, aching, and poorly localized pain. Sclerotomic pain impulses can be projected to regions in the brain such as the hypothalamus, limbic system, and reticular formation and can cause depression, anxiety, fear, or anger. Autonomic changes, such as changes in vasomotor tone, blood pressure, and sweating, may also occur.

Dermatomic pain, in contrast to sclerotomic pain, is sharp and well localized. Unlike sclerotomic pain, dermatomic pain projects mainly to the thalamus and is relayed directly to the cortex, skipping autonomic and affective responses.

Nociceptors and Neural Transmission

Pain receptors known as **nociceptors,** or free nerve endings, are sensitive to mechanical, thermal, and chemical energy.[11] They are commonly found in skin, periosteum surrounding bone, teeth, meninges, and some organs.

nociceptors Pain receptors.

Afferent nerve fibers transmit impulses from the nociceptors toward the spinal cord, while *efferent* nerve fibers, such as motor neurons, transmit impulses from the spinal cord toward the periphery. First-order, or primary, afferents transmit impulses from a nociceptor to the dorsal horn of the spinal cord. There are four types of first-order neurons: Aα, Aβ, Aδ, and C. Aα and Aβ fibers are characterized as large-diameter afferents and Aδ and C fibers as small-diameter afferents. Aδ and C fibers transmit sensations of pain and temperature. Aδ neurons originate from nociceptors located in skin and transmit "fast pain," while C neurons originate from both superficial tissue (skin) and deeper tissue (ligaments and muscle) and transmit "slow pain."[12]

Second-order afferent fibers carry sensory messages from the dorsal horn to the brain and are categorized as nociceptive specific. Second-order afferents receive input from Aβ, Aδ, and C fibers. Second-order afferents serve relatively large, overlapping receptor fields. Nociceptive-specific second-order afferents respond exclusively to noxious stimulation and receive input only from Aδ and C fibers. All of these neurons synapse with third-order neurons, which carry information via ascending spinal tracts to various brain centers, where the input is integrated, interpreted, and acted upon.[12]

Facilitators and Inhibitors of Synaptic Transmission

For information to pass between neurons, a transmitter substance must be released from one neuron terminal, enter the synaptic cleft, and attach to a receptor site on the next neuron. This occurs primarily due to chemicals called *neurotransmitters.* However, it has been shown that several compounds that are not true neurotransmitters can facilitate or inhibit synaptic activity. These include *serotonin,* which is active in descending pathways; *norepinephrine,* which inhibits pain transmission between first- and second-order neurons; *substance P,* which is active in small-diameter primary afferent neurons; *enkephalins,* found in descending pathways; and *β-endorphin,* found in the central nervous system.[40]

Neurotransmitters:

- Serotonin
- Norepinephrine
- Substance P
- Enkephalins
- β-endorphin

Mechanisms of Pain Control

The neurophysiological mechanisms of pain control have not been fully explained. To date, three models of pain control have been proposed: the gate control theory, descending pathway pain control, and the release of β-endorphin. It is likely that some as-yet-unexplained combination of these three models is responsible for pain modulation.[1]

Gate Control Theory Sensory information coming from cutaneous receptors in the skin enters the ascending Aβ afferents and is carried to the substantia gelatinosa in the dorsal horn of the spinal cord (Figure 10–7). Likewise, pain messages from the nociceptors are carried along the Aδ and C afferent fibers and enter the dorsal horn. Sensory information coming from Aβ fibers overrides or inhibits the "pain information" carried along Aδ and C fibers, thus inhibiting, or effectively "closing the gate" to, the transmission of pain information to second-order neurons. Consequently, pain information is not transmitted and never reaches sensory centers in the brain. The gate control theory of pain control occurs at the spinal cord level.[12]

Descending Pathway Pain Control Stimulation of descending pathways in the spinal cord may also inhibit pain impulses carried along the Aδ and C afferent fibers (Figure 10–8). It is theorized that previous experiences, emotional influences, sensory

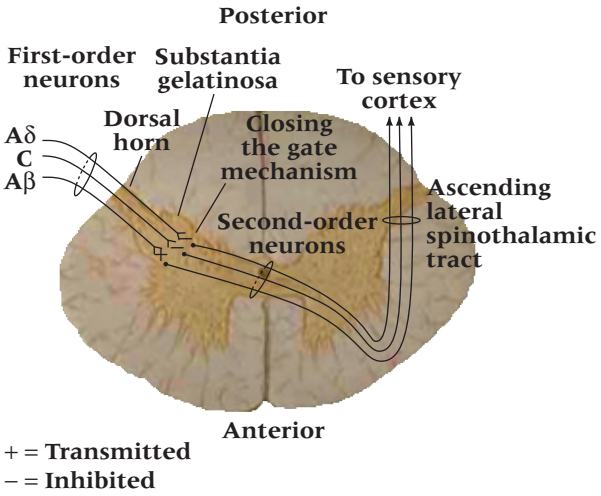


FIGURE 10–7 Gate control theory. Sensory information carried on Aβ fibers "closes the gate" to pain information carried on Aδ and C fibers in the substantia gelatinosa, preventing the transmission of pain to sensory centers in the cortex.

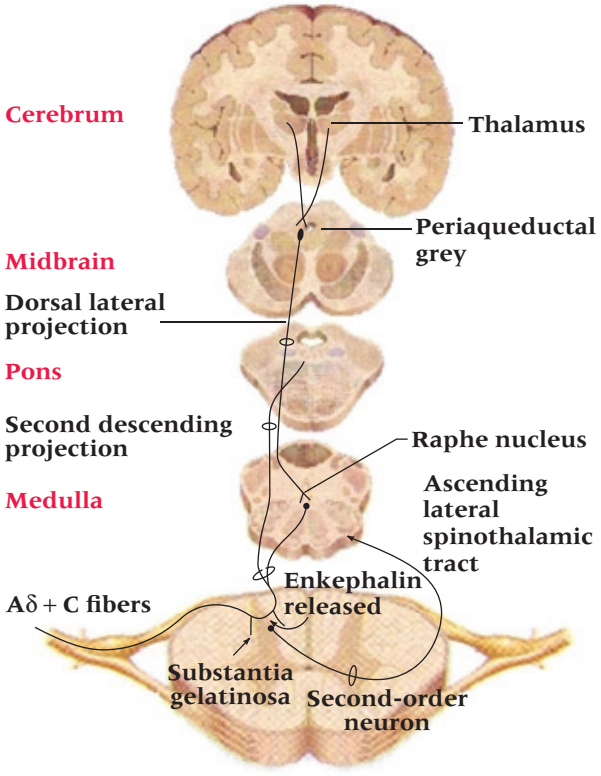


FIGURE 10–8 Descending pathway pain control. Influence from the thalamus stimulates the periaqueductal grey, the raphe nucleus, and the pons to inhibit the transmission of pain impulses through the ascending tracts.

perception, and other factors influence the transmission of pain messages and thus the perception of pain. The information coming from higher centers in the brain along efferent descending pathways in the spinal cord causes a release of two neurotransmitter-like substances, enkephalin and norepinephrine, into the dorsal horn, which together block or

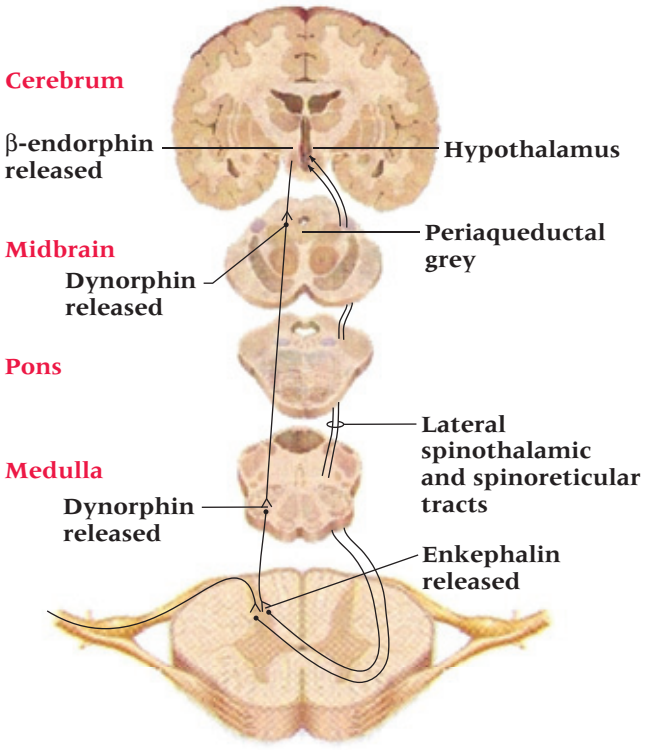


FIGURE 10–9 β-endorphin released from the hypothalamus and dynorphin released from the periaqueductal grey and the medulla.

inhibit the synaptic transmission of impulses from the Aδ and C afferent fibers to second-order afferent neurons.[12]

Release of β-Endorphin It has been shown that noxious (painful) stimulation of nociceptors resulting in the transmission of pain information along Aδ and C afferents can stimulate the release of an opiate-like chemical called β-endorphin from the hypothalamus and anterior pituitary (Figure 10–9). β-endorphin is endogenous to the central nervous system and is known to have strong analgesic effects. The exact mechanisms by which β-endorphin produces these potent analgesic effects are unclear. Acupuncture, acupressure, and point stimulation using electrical currents are all techniques that may stimulate the release of β-endorphin.[12]

Pain Assessment

Pain is a complex phenomenon that is difficult to evaluate and quantify because it is subjective. Thus, obtaining an accurate and standardized assessment of pain is problematic. A number of validated assessment tools are available that allow the athletic trainer to develop a pain profile by identifying the type of pain a patient is experiencing, quantifying the intensity of pain, evaluating the effect of the pain experience on a patient's level of functioning, and assessing the psychosocial impact of pain.

Pain measurement tools include simple *unidimensional scales* or *multidimensional questionnaires.* Pain measurement should include both the time frame and the clinical context of the pain. With unidimensional scales, individuals with acute pain are usually asked to describe their pain "right now" and may be asked about the average intensity over a fixed period to provide information on the course of the pain. Examples of commonly used unidimensional scales are verbal rating scales, the numeric rating scale, and the visual analog scale. Multidimensional pain assessment tools are more comprehensive pain assessments that require the determination of the quality of the pain and its effect on mood and function. They are used mainly to quantify these aspects of pain, and they take longer to administer than the unidimensional scales. The McGill Pain Questionnaire is an example of a multidimensional pain assessment tool.

Visual Analog Scales Visual analog scales are quick and simple tests that consist of a line, usually 2½ inches (10 cm) in length, the extremes of which are taken to represent the limits of the pain experience. The patient simply places a mark on that line based on the perceived level of pain. Scales can be completed daily or more often (Figure 10–10).

Pain Charts Pain charts are used to establish spatial properties of pain. They involve a two-dimensional graphic chart on which the patient assesses the location of pain and a number of subjective components. The patient colors the chart in areas that correspond to pain (Figure 10–11).

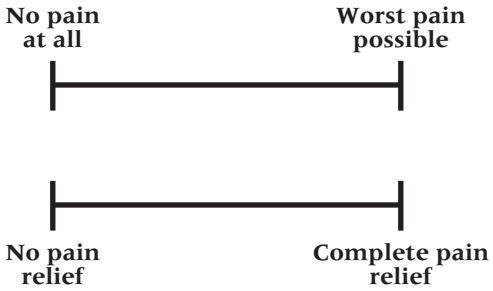


FIGURE 10–10 Visual analog scales.

McGill Pain Questionnaire The McGill Pain Questionnaire lists 78 words that describe pain, which are grouped into 20 sets and divided into four categories representing dimensions of the pain experience. It may take up to 20 minutes to complete, and the questionnaire is administered every 2 to 4 weeks (Figure 10–12).

Activity Pain Indicators Profile The Activity Pain Indicators Profile is a 64-question self-report tool used to assess functional impairment associated with pain. It measures the frequency of certain behaviors, such as housework, recreation, and social activities, that produce pain.

Numeric Rating Scale The numeric rating scale is the most common acute pain profile used in sports medicine. The patient is asked to verbally rate pain on a scale from 1 to 10, with 10 representing the worst pain he or she has experienced or can imagine. Usually, the scale is administered verbally before

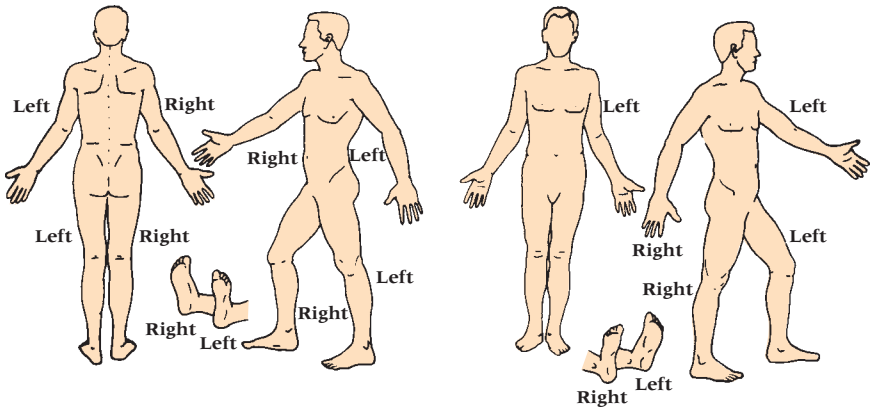


FIGURE 10–11 Pain chart. Use the following instructions: "Please use all of the figures to show me exactly where all your pains are, and where they radiate to. Shade or draw with *blue marker*. Only the athlete is to fill out this sheet. Please be as precise and detailed as possible. Use *yellow marker* for numbness and tingling. Use *red marker* for burning or hot areas, and *green marker* for cramping. Please remember: blue = pain, yellow = numbness and tingling, red = burning or hot areas, green = cramping." Used with permission from Melzack R: *Pain measurement and assessment*, New York, 1983, Raven Press.

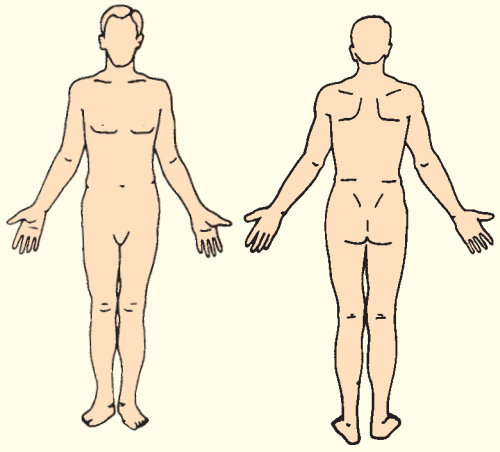
McGill Pain Questionnaire

Patient's Name _____ Date _____ Time _____ am/pm

PRI S _____ A _____ E _____ M _____ PRI (T) _____ PPI _____

(1–10) (11–15) (16) (17–20) (1–20)

1 Flickering	11 Tiring	Brief
Quivering	Exhausting	Momentary
Pulsing		Transient
Throbbing	12 Sickening	
Beating	Suffocating	Rhythmic
Pounding		Periodic
2 Jumping	13 Fearful	Intermittent
Flashing	Frightful	
Shooting	Terrifying	Continuous
3 Pricking	14 Punishing	Steady
Boring	Gruelling	Constant
Drilling	Cruel	
Stabbing	Vicious	
Lancinating	Killing	
4 Sharp	15 Wretched	
Cutting	Blinding	
Lacerating		
5 Pinching	16 Annoying	
Pressing	Troublesome	
Gnawing	Miserable	
Cramping	Intense	
Crushing	Unbearable	
6 Tugging	17 Spreading	
Pulling	Radiating	
Wrenching	Penetrating	
7 Hot	Piercing	
Burning	18 Tight	
Scalding	Numb	
Searing	Drawing	
8 Tingling	Squeezing	
Itchy	Tearing	
Smarting	19 Cool	
Stinging	Cold	
9 Dull	Freezing	
Sore	20 Nagging	
Hurting	Nauseating	
Aching	Agonizing	
Heavy	Dreadful	
10 Tender	Torturing	
Taut	PPI	
Rasping	0 No pain	
Splitting	1 Mild	
	2 Discomforting	
	3 Distressing	
	4 Horrible	
	5 Excruciating	



E = External
I = Internal

COMMENTS

FIGURE 10–12 McGill Pain Questionnaire. The descriptors fall into four groups: sensory, 1 to 10; affective, 11 to 15; evaluative, 16; and miscellaneous, 17 to 20. The rank value for each descriptor is based on its position in the word set. The sum of the rank values is the pain rating index (PRI). The present pain intensity (PPI) is based on a scale of 0 to 5.
Used with permission from Melzack R: *Pain measurement and assessment*, New York, 1983, Raven Press.

and after treatment. When treatments provide pain relief, questions are asked about the extent and duration of the relief.

Treating Pain

An athletic trainer can approach pain management using a variety of treatment options, including therapeutic modalities and medications.

Therapeutic Modalities Many therapeutic modalities can provide pain relief.[32] There is not one best therapeutic agent for pain control. The athletic trainer must select the therapeutic agent that is most appropriate for each athlete, based on the athletic trainer's knowledge of the modalities and professional judgment (see Chapter 15). In no situation should the athletic trainer apply a therapeutic agent

without first developing a clear rationale for the treatment. The therapeutic modalities used to control pain do little to promote tissue healing. They should be used to relieve acute pain following injury or surgery or to control pain and other symptoms, such as swelling, to promote progressive exercise. The athletic trainer should not lose sight of the effects of the modalities or the importance of progressive exercise in restoring the athlete's functional ability.

The athletic trainer can make use of the gate control mechanism of pain control by using superficial heat or cold, electrical stimulating currents, massage, and counterirritants to stimulate the large-diameter Aα and Aβ efferent nerve fibers. Noxious stimulation of acupuncture and trigger points using either electrical stimulating currents or deep acupressure massage techniques can mediate the release of β-endorphin.[12]

Medications A physician may choose to prescribe oral or injectable medications in treating a patient. The most commonly used medications are classified as analgesics, anti-inflammatory agents, or both.[10] The athletic trainer should become familiar with these drugs and note whether the patient is taking any medications (see Chapter 17). It is also important to work with the referring physician or a pharmacist to make sure that the patient takes the medications appropriately.

Psychological Aspect of Pain

Pain, especially chronic pain, is a subjective psychological phenomenon.[40] When painful injuries are treated, the total patient must be considered, not just the pain or condition. Even in the most well-adjusted person, pain creates emotional changes. Constant pain often causes self-centeredness and an increased sense of dependency. Chapter 11 discusses in great detail the psychosocial aspects of dealing with injury and managing pain.

Patients vary in their pain thresholds (Figure 10–13). Some can tolerate enormous pain, whereas others find mild pain almost unbearable. Pain is perceived as being worse at night because persons are alone, more aware of themselves, and devoid of external diversions. Personality differences can also cause differences in pain toleration. For example, patients who are anxious, dependent, and immature have less tolerance for pain than those who are relaxed and emotionally in control.

FIGURE 10–13 Coping with pain in sports is as much psychological as it is physical.

A number of theories about how pain is produced and perceived by the brain have been advanced. Only in the last few decades has science demonstrated that pain is both a psychological and a physiological phenomenon and is therefore unique to each individual.[4,40] Sports activities demonstrate this fact clearly. Through conditioning, an athlete learns to endure the pain of rigorous activity and to block the sensations of a minor injury. It is perhaps most critical for the athletic trainer to recognize that all pain is very real to the patient.

10–9 Clinical Application Exercise

A gymnast is receiving electrical stimulation for chronic low back pain.

? What is the purpose of administering electrical stimulation for the chronic pain?

SUMMARY

- The three phases of the healing process are the inflammatory response phase, the fibroblastic repair phase, and the maturation-remodeling phase, which occur in sequence but overlap one another in a continuum.
- During the inflammatory response phase, debris is phagocytized (cleaned up). The fibroblastic repair phase involves the deposition of collagen fibers to form a firm scar. During the maturation-remodeling phase, collagen is realigned along lines of tensile force and the tissue gradually assumes normal appearance and function.
- Factors that may impede the healing process include the extent of the injury, edema,

hemorrhage, poor vascular supply, separation of tissue, muscle spasm, atrophy, corticosteroids, keloids and hypertrophic scars, infection, climate and oxygen tension, health, age, and nutrition.
- Treatment techniques that can be used to modify soft-tissue healing include anti-inflammatory medications, therapeutic modalities, exercise rehabilitation, and platelet-rich plasma injections.
- The healing of soft tissues, including cartilage, ligament, muscle, and nerve, follows a similar course. Unlike the other tissues, nerve has the capability of regenerating.
- Bone healing following fracture involves increased activity of osteoblastic cells and osteoclastic cells.
- Pain is a response to a noxious stimulus that is subjectively modified by past experiences and expectations.
- Pain is classified as either acute or chronic and can exhibit many different patterns.
- Three models of pain control are the gate control theory, descending pathway pain control, and the release of β-endorphin from higher centers of the brain.
- Pain perception may be influenced by a variety of cognitive processes mediated by the higher brain centers.

WEB SITES

American Academy of Pain Management: www.aapainmanage.org

American Pain Society: www.ampainsoc.org

World Union of Wound Healing Societies: www.wuwhs.org

SOLUTIONS TO CLINICAL APPLICATION EXERCISES

10–1 Little can be done to speed up the healing process physiologically. This athlete must realize that certain physiological events must occur during each phase of the healing process. Any interference with this healing process during a rehabilitation program will likely slow return to full activity. The healing process must have an opportunity to accomplish what it is supposed to.

10–2 Immobilization during the inflammatory process may be beneficial; however, controlled mobilization helps the tissue decrease atrophy and enhance the healing process. Controlled mobilization allows the athlete to perform progressive strengthening exercises in a timely manner.

10–3 Initially, a transitory vasoconstriction with the start of blood coagulation of the broken blood vessels occurs. Dilation of the vessels in the region of injury follows, along with the activation of chemical mediators via key cells.

10–4 A grade 2 lateral ankle sprain implies that the joint capsule and ligaments are partially torn. At 3 weeks, the injury has been cleaned of debris and is undergoing the process of secondary healing. Granulation tissue fills the torn areas, and fibroblasts are beginning to form scar tissue.

10–5 In its acute phase, the injury was not allowed to heal properly. As a result, the injury became chronic, with a proliferation of scar tissue, lymphocytes, plasma cells, and macrophages.

10–6 Uncomplicated acute bone healing goes through five stages: hematoma formation, cellular proliferation, callus formation, ossification, and remodeling.

10–7 Because it is shorter, the left leg has the greater stress during running. This stress creates increased tension on the tibia's concave side, causing an increase in osteoclastic activity.

10–8 The pain is considered to be chronic, deep somatic pain stemming from the low back muscles. It is conducted primarily by the C-type nerve fibers.

10–9 The purpose is to stimulate the large, rapidly conducting nerve fibers to inhibit the smaller and slower nerves that carry pain impulses.

REVIEW QUESTIONS AND CLASS ACTIVITIES

1. What are the three phases of healing, and what are the approximate time frames for each of these three phases?
2. What are the physiological events associated with the inflammatory response phase of the healing process?
3. How can you differentiate between acute and chronic inflammation?
4. How is collagen laid down in the area of injury during the fibroblastic repair phase of healing?
5. Explain Wolff's law and the importance of controlled mobility during the maturation-remodeling phase of healing.
6. What are some of the factors that can have a negative impact on the healing process?
7. Discuss treatment techniques for modifying soft-tissue healing, including anti-inflammatory medications, therapeutic modalities, exercise rehabilitation, and platelet-rich plasma injections.
8. Compare and contrast the course of healing in cartilage, ligaments, muscle, and nerve.
9. What is a basic definition of pain?
10. What are the different types of pain?
11. What are the characteristics of the various sensory receptors?
12. How does the nervous system relay information about painful stimuli?
13. Describe how the gate control mechanism of pain modulation may be used to modulate pain.
14. How does descending pathway pain control modulate pain?
15. What are the opiate-like substances, and how do they modulate pain?
16. What are the assessment scales available to help the athletic trainer determine the extent of pain perception?
17. How can pain perception be modified by cognitive factors?

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