chapter

Using Therapeutic Modalities to Affect the Healing Process

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OBJECTIVES

Following completion of this chapter, the student will be able to:

- Define inflammation and its associated signs and symptoms.
- Clarify how therapeutic modalities should be used in rehabilitation of various conditions.
- Compare the physiological events associated with the different phases of the healing process.
- Formulate a plan for how specific modalities can be used effectively during each phase of healing and provide a rationale for their use.
- Identify those factors that can interfere with the healing process.

HOW SHOULD THE CLINICIAN USE THERAPEUTIC MODALITIES IN REHABILITATION?

Therapeutic modalities, when used appropriately, can be useful tools in the rehabilitation of the injured patient.[1,2] Like any other tool, their effectiveness is limited by the knowledge, skill, and experience of the clinician using them. For the competent clinician, decisions regarding how and when a modality may best be incorporated should be based on a combination of theoretical knowledge and practical experience. As a clinician, you should not use therapeutic modalities at random, nor should you base their use on what has always been done before. Instead, you must always give consideration to what should work best in a specific injury situation.

There are many different approaches and ideas regarding the use of modalities in injury rehabilitation. Therefore, no "cookbook" exists for modality use. In a given clinical situation, you as a clinician should make your own decision about which modality will be most effective.

In any program of rehabilitation, modalities should be used primarily as adjuncts to therapeutic exercise and certainly not at the exclusion of therapeutic exercises. Rehabilitation protocols and progressions must be based primarily on the physiological responses of the tissues to injury and on an understanding of how various tissues heal (Figure 2–1).[3] Thus, the clinician must understand the healing process to be effective in incorporating therapeutic modalities into the rehabilitative process.

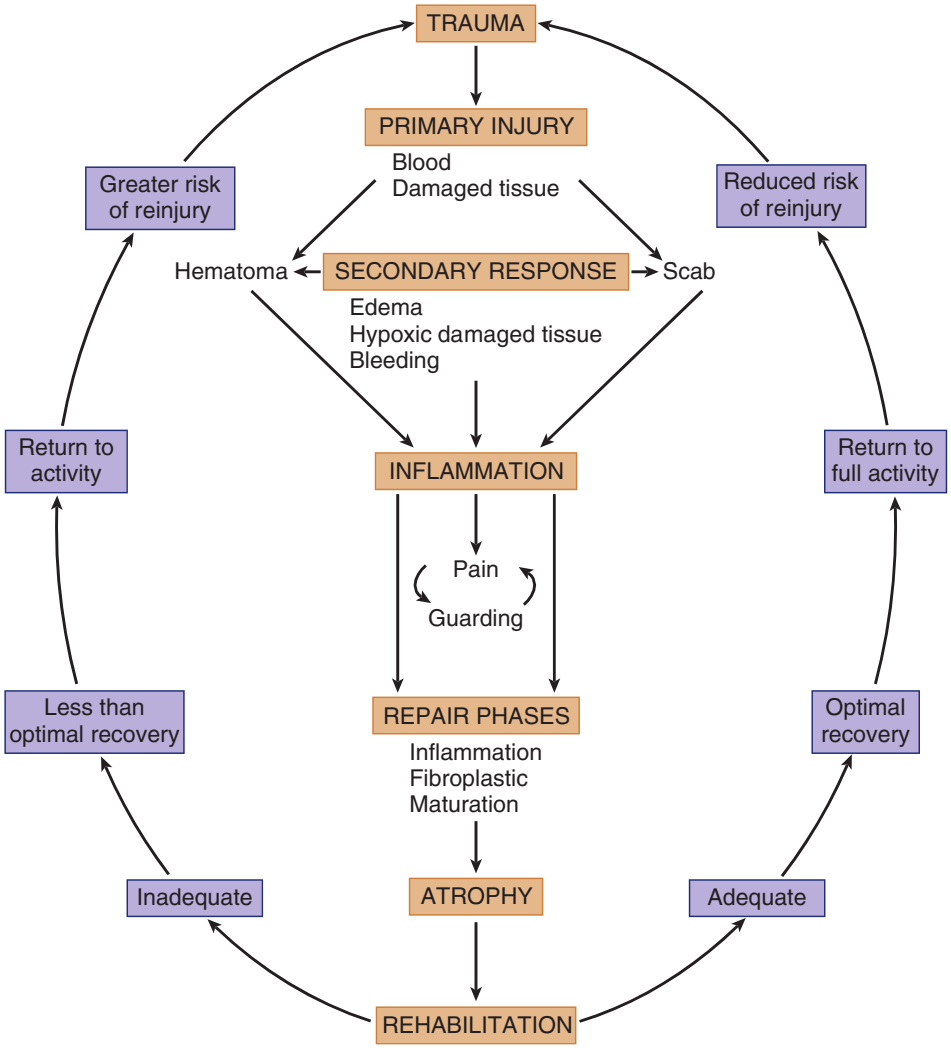


Figure 2–1. A cycle of sport-related injury. Reproduced with permission from Booher J. Thibedeau G. *Athletic Injury Assessment.* St. Louis, MO: McGraw Hill; 1994.

In the physically active population, injuries most often involve the musculoskeletal system and in some instances the nervous system.[4,5] Some health care professionals have debated whether the terms *acute* and *chronic* are appropriate in defining injury.[6] At some point all injuries can be considered acute; in other words, there is always some beginning point for every injury. At what point does an acute injury become a chronic injury? Generally injuries occur either from trauma or from overuse. Acute injuries are caused by trauma; chronic injuries can result from overuse as occurs with the repetitive dynamics of running, throwing, or jumping.[7,8] Thus, the terms *traumatic* and *overuse injuries* are more appropriate.

Primary injuries are almost always described as being either traumatic or overuse resulting from *macrotraumatic* or *microtraumatic* forces. Injuries classified as macrotraumatic occur as a result of trauma and produce immediate pain and disability. Macrotraumatic injuries include fractures, dislocations, subluxations, sprains, strains, and contusions.[9] Microtraumatic injuries are most often overuse injuries and result from repetitive overloading or incorrect mechanics associated with continuous training or competition. They include tendinitis, tenosynovitis, bursitis, and so on. A *secondary injury* is essentially the inflammatory or hypoxia response that occurs with the primary injury.[10]

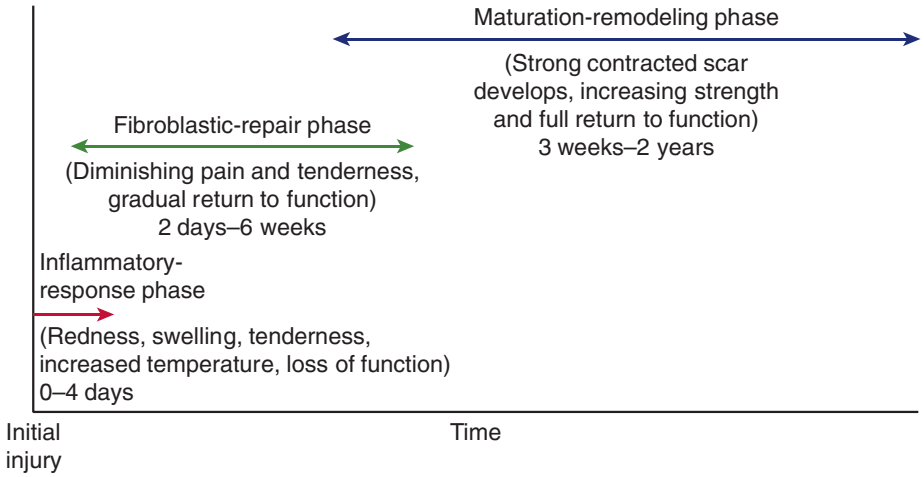


Figure 2–2. The three phases of the healing process fall along an overlapping time continuum.

Clinical Decision-Making *Exercise 2–1*

A female soccer player sprains her ankle, and the team physician diagnoses it as a grade 1 sprain. The coach wants to know how long the athlete will be out. On what information should the clinician base his or her response?

THE IMPORTANCE OF UNDERSTANDING THE HEALING PROCESS

The healing process is a continuum consisting of three phases:
- inflammatory-response phase;
- fibroblastic-repair phase;
- maturation-remodeling phase.

The decisions made by the clinician on how and when therapeutic modalities may best be used should be based on recognition of signs and symptoms as well as some awareness of the time frames associated with the different phases of the healing process.[11,12] The clinician must have a sound understanding of that process in terms of the predictable sequence of the phases of healing that take place.[13]

The healing process consists of the inflammatory-response phase, the fibroblastic-repair phase, and the maturation-remodeling phase.[50] It must be stressed that although the phases of healing are presented 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[14] (Figure 2–2). The clinician should rely primarily on observation of the signs and symptoms to determine how the healing process is progressing.

Inflammatory-Response Phase

When you hear the term *inflammation*, you automatically think of something negative. The fact is that inflammation is a very important part of the healing process.[15] Without the physiological changes that take place during the inflammatory process, the later stages of healing cannot occur.[16] Once a tissue is injured, the process of healing begins immediately. The destruction of tissue produces direct injury to the cells of the various tissues. Cellular injury disrupts the blood vessels which immediately initiates coagulation and subsequent development of a fibrin clot. This leads to homeostasis of the injured tissue, thus altering metabolism and liberating chemical messengers that initiate the inflammatory response[17] (Figure 2–3).

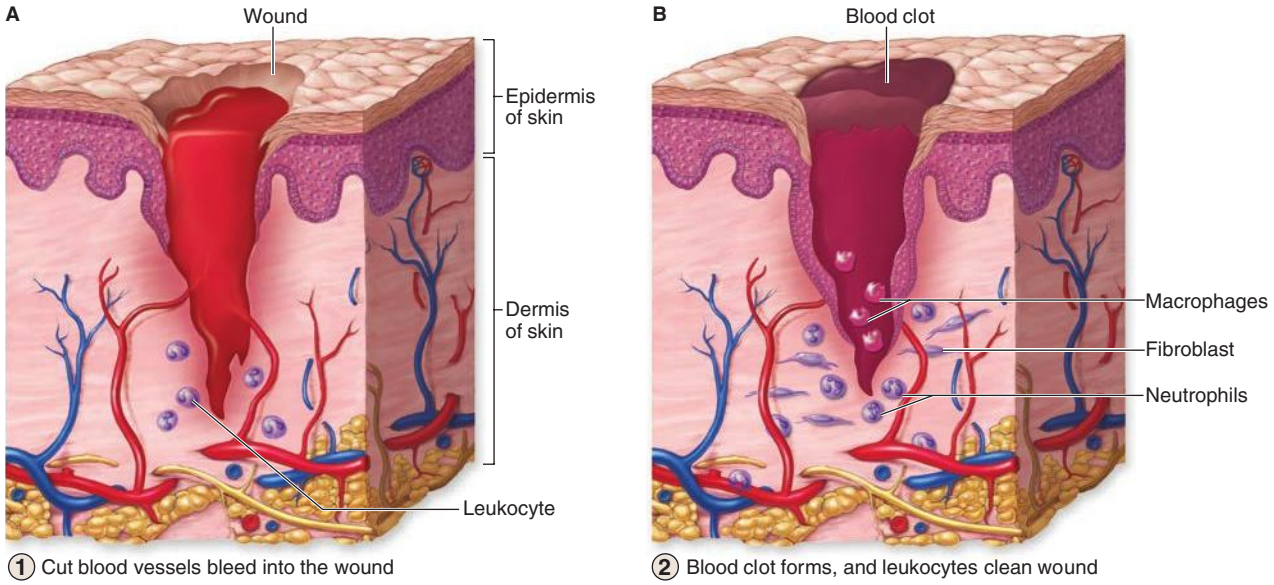


Figure 2–3. Initial injury and inflammatory-response phase of the healing process. (A) Cut blood vessels bleed into the wound. (B) Blood clot forms, and leukocytes clean wound. Reproduced with permission from McKinley M, O'Loughlin VD, Pennefather-O'Brien EE: *Human Anatomy*, 6th ed. New York, NY: McGraw Hill; 2021.

Vascular Reaction

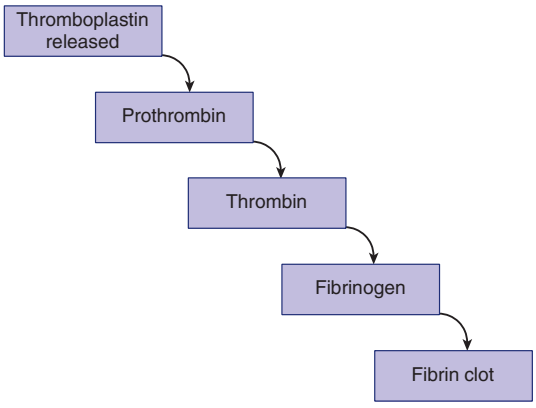
Hemostasis is a vascular reaction that involves vascular spasm, the formation of a platelet plug, blood coagulation, and the growth of fibrous tissue.[18] The immediate response to tissue damage is a vasoconstriction of the vascular walls in the vessels leading away from the site of injury that lasts for approximately 5–10 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 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. The initial effusion of blood and plasma lasts for 24–36 hours.

Signs of inflammation are as follows:
- redness;
- swelling;
- tenderness to touch;
- increased temperature;
- loss of function.

The 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 and eventually form a plug. These plugs obstruct local lymphatic fluid drainage and thus localize the injury response.

The clotting process. The initial event that precipitates clot formation is the conversion of fibrinogen to fibrin.[19] This transformation results from 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 following injury and is completed by 48 hours[20] (Figure 2–4).

Figure 2–4. The clotting process involves a series of physiological events that require as long as 48 hours to complete.



Signs and Symptoms

The inflammatory-response phase is characterized symptomatically by redness, swelling, tenderness, warmth to touch, loss of function, and possibly crepitus.[18]

Cellular Response

Inflammation is a process during which **leukocytes** and other **phagocytic cells** and exudate are delivered to the injured tissue.[21] This cellular reaction is generally protective, tending to localize or dispose of injury by-products (e.g., blood or damaged cells) through phagocytosis, thus setting the stage for repair.[22] Locally, vascular effects, disturbances of fluid exchange, and migration of leukocytes from the blood to the tissues occur.[23]

Chemical Mediators

Chemical mediators include:

- cytokines;
- leukotrines;
- prostaglandins; and
- histamine.

The events in the inflammatory response are initiated by a series of interactions involving several chemical mediators.[24] Resolution of inflammation is driven by a complex set of these chemical mediators which regulate cellular events required to clear inflammatory cells from sites of injury or infection and restore homeostasis. 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, leukotrines, prostaglandins,* and *histamine*—are important in limiting the amount of exudate, and thus swelling, after injury.[25] Cytokines, in particular chemokines and interleukin, are the major regulators of leukocyte traffic and help attract leukocytes to the actual site of inflammation.[26] 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.[22] 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.

As a result of the collective effects of the vascular response, the cellular response and the chemical mediators, 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 2–4 days following initial injury (Figure 2–5).

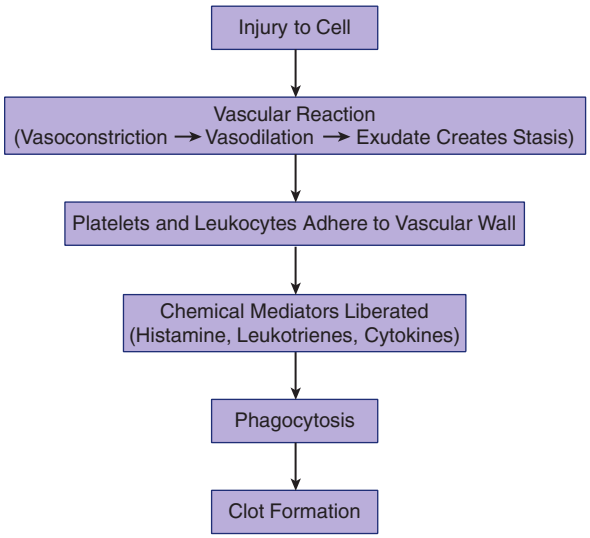


Figure 2–5. The sequence of the inflammatory response.

In chronic inflammation, neutrophils are replaced with:
- macrophages;
- lymphocytes;
- fibroblasts;
- plasma cells.

Granulation tissue consists of:
- capillaries;
- collagen;
- fibroblasts.

The extracellular matrix contains:
- collagen;
- elastin;
- ground substance.

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 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.[27] 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.[27] 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.[27,28] There is 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 pharmacologic treatments.[29]

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.[30] The period of scar formation referred to as **fibroplasia** begins within the first few hours following injury and may last for as long as 4–6 weeks.

Signs and Symptoms

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 will gradually disappear.[31]

Revascularization

During this phase, growth of endothelial capillary buds into the wound (angiogenesis) is stimulated by a lack of oxygen. Thus, the wound is now 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[32] (Figure 2–6).

Formation of Scar

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, which contains protein fibers of collagen and elastin, a ground substance that consists of nonfibrous proteins called proteoglycans, glycosaminoglycans, and fluid.[33] 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.[34] As the tensile strength increases, the number of fibroblasts diminishes to signal the beginning of the maturation phase.

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 can promote extended fibroplasia and excessive fibrogenesis that can lead to irreversible tissue damage.[34] Fibrosis can occur in synovial structures, as is the case with adhesive capsulitis in the shoulder; in extra-articular tissues, including tendons and ligaments; in bursa; or in muscle.

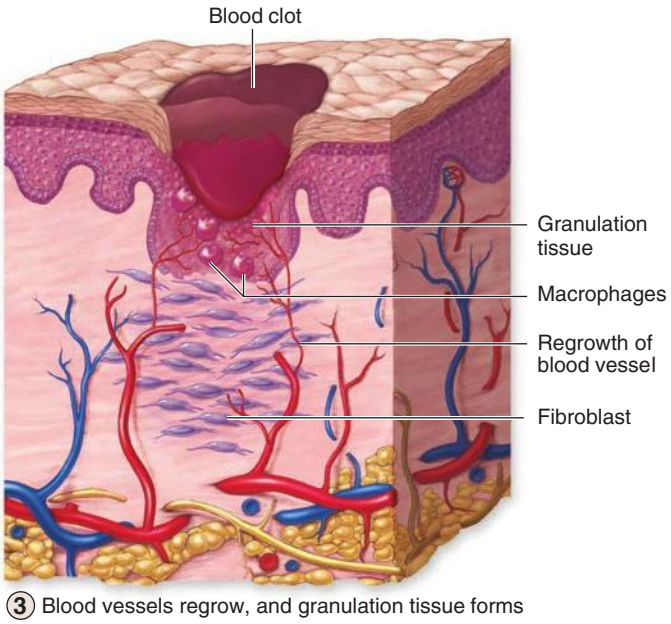


Figure 2–6. Blood vessels regrow, and granulation tissue forms in the fibroblastic-repair phase of the healing process. Reproduced with permission from McKinley M, O'Loughlin VD, Pennefather-O'Brien EE: *Human Anatomy*, 6th ed. New York, NY: McGraw Hill; 2021.

A mature scar will be devoid of physiological function, it will have less tensile strength than the original tissue, and it is not as well vascularized.

The Importance of Collagen

Collagen is a major structural protein that forms strong, flexible, and inelastic structures that hold connective tissue together. There are at least 16 types of collagen, but 80%–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 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. 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 loadbearing 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.[51]

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.[51]

Maturation-Remodeling Phase

The maturation-remodeling phase of healing is a long-term process. This phase features a realignment or remodeling of the collagen fibers that make up the scar tissue according to the tensile forces to which that scar is subjected (Figure 2–7).[17] Ongoing breakdown and

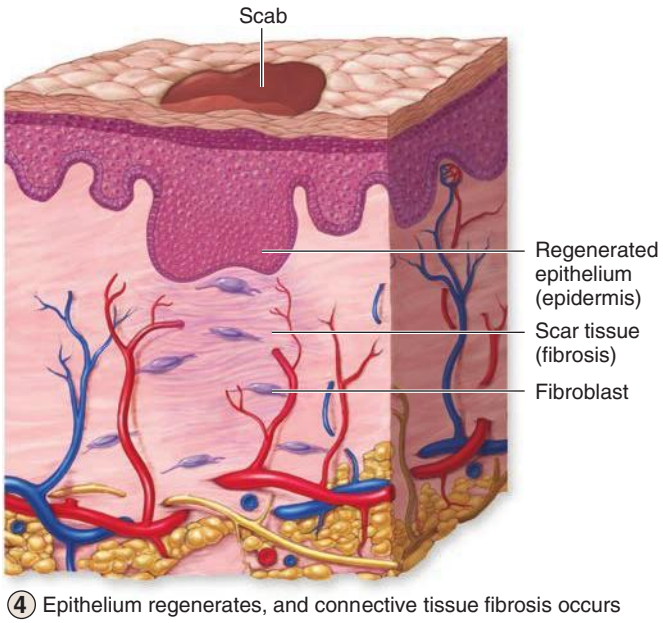


Figure 2–7. Epithelium regenerates, and connective tissue fibrosis occurs in the maturation-remodeling phase of the healing process. Reproduced with permission from McKinley M, O'Loughlin VD, Pennefather-O'Brien EE: *Human Anatomy*, 6th ed. New York, NY: McGraw Hill; 2021.

synthesis of collagen occur with a steady increase in the tensile strength of the scar matrix. With increased stress and strain, the collagen fibers will realign in a position of maximum efficiency parallel to the lines of tension.[35] 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 approximately 3 weeks, a firm, strong, contracted, nonvascular scar exists. The maturation phase of healing may require several years to be totally complete.

FACTORS THAT IMPEDE HEALING

The healing process may be influenced by a variety of factors that may influence the course and outcome of that process.[26] See Table 2–1 for a list of 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. They are generally caused by 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.[26]

Hemorrhage. Bleeding occurs with even the smallest amount of damage to the capillaries. It 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.[26]

Poor vascular supply. Injuries to tissues with a poor vascular supply heal poorly and at a slow rate. This is likely related to a failure in the delivery of phagocytic cells initially and also of fibroblasts necessary for formation of scar.

Separation of tissue. Mechanical separation of tissue can significantly impact 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

Table 2–1 Factors That Impede Healing

Extent of injury
Edema
Hemorrhage
Poor vascular supply
Separation of tissue
Muscle spasm
Atrophy
Corticosteroids
Keloids and hypertrophic scars
Infection
Humidity, climate, and oxygen tension
Health, age, and nutrition

must heal by second intention, with granulation tissue filling the defect and causing excessive scarring.[36]

Muscle spasm. Muscle spasm causes traction on the torn tissue, separates the two ends, and prevents approximation. Both 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 retards atrophy.

Corticosteroids. Use of corticosteroids such as cortisone 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.

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, that is out of proportion to normal scarring. The result is a raised, firm, thickened, red scar.[34,36]

Infection. The presence of bacteria in the wound can delay healing, cause excessive granulation tissue, and frequently cause large deformed scars.[36]

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 provides an advantage for the necrotic debris to go to the surface and be shed.[37]

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 of the older patient and may affect wound healing. Nutrition is important for wound healing. In particular, vitamins C, K, A, and E, zinc, and amino acids play critical roles in the healing process.[34]

It has been shown that malnutrition negatively affects the healing process.[52] A patient who is undernourished or malnourished 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.[52]

HOW SHOULD THERAPEUTIC MODALITIES BE USED THROUGHOUT THE REHABILITATION PROCESS?

Using Modalities in the Immediate First Aid Management of Injury

Table 2–2 summarizes the various modalities that may be used in the different phases of the healing process. Modality use in the initial treatment of injury should be directed toward protecting the injured tissue from further injury, reducing the secondary hypoxic injury that results from the acute inflammatory response, and controlling pain while limiting swelling. If swelling can be minimized initially, the amount of time required for injury rehabilitation can be significantly reduced.

For many years, the recommendation for managing acute musculoskeletal injuries has included the immediate application of ice, compression, and elevation in combination with some type of protection (elastic wrap, tape, crutches, walking boot, etc.) and/or rest or restricted activity. The acronyms *RICE* and *PRICE* have both been commonly used to refer to this combination of simultaneously applied treatment techniques that have been well accepted as a best practice recommendation by most health care providers. Despite this near-unanimous clinical consensus, there is limited evidence from high-quality randomized clinical trials that support the use of these interventions.[53] Most recently, it has been recommended that a more appropriate acronym would be **POLICE**, which stands for protection, optimal loading, ice, compression, and elevation (Figure 12–22).[54]

Protecting the damaged tissue from further injury is an extremely important component of any treatment program. Once a tissue is injured, it immediately begins the healing process. Subjecting the injured part to additional unnecessary external stresses and strains may cause additional bleeding and further damage that can interfere with the essential biological processes in the acute inflammatory stage of the healing process. Thus, short periods of protection (using crutches, braces, etc.), which include rest and immobilization, are recommended immediately following acute soft tissue injury.

Optimal loading refers to determining and subsequently incorporating the appropriate progression from protecting the tissue to prevent exacerbation of the injury, to mechanically loading the tissue to facilitate healing. Early functional activity encourages early recovery.[54] Longer periods of rest during which injured tissues are unloaded may produce adverse changes to joint biomechanics and tissue morphology. Progressive mechanical loading of injured tissues following the acute inflammatory stage of healing promotes cellular responses that improve the structural characteristics of collagen, thus facilitating healing.[51]

Cryotherapy (*ice*) is said to produce vasoconstriction, at least superficially and perhaps indirectly in the deeper tissues, and thus limits the bleeding that always occurs with injury. Ice bags, cryocuffs, cold packs, and ice massage may all be used. Cold baths should be avoided because the extremities must be placed in a gravity-dependent position. Cold whirlpools also place the extremities in the gravity-dependent position and produce a massaging action that is likely to retard clotting. The importance of applying ice immediately following injury for limiting acute swelling through vasoconstriction has probably been overemphasized. The initial use of ice is more important for decreasing the secondary hypoxic response associated with tissue injury (see Chapter 9). Analgesia, which occurs through stimulation of sensory cutaneous nerves via the gating mechanism, blocks or reduces pain (see Chapter 4).[38]

Immediate *compression* has been demonstrated to be an effective technique for limiting swelling. An intermittent compression device may be used to provide even pressure around an injured extremity. The pressurized sleeve mechanically reduces the amount of space available