

# The Path to Wound Healing

A Clinical Reference Guide to the  
Primary Phases of Wound Healing



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## **A Clinical Reference Guide to the Primary Phases of Wound Healing**

A wound is defined as injury to the skin that may or may not involve an opening of the skin and can include damage to underlying tissues.<sup>1</sup> Wounds can be classified in many ways, and based on knowledge and understanding of skin structures, wounds are classified as partial-thickness or full-thickness. Partial-thickness wounds involve only partial-thickness of the skin layers, that is, they are confined to the epidermal and dermal layers. Full-thickness wounds involve total loss of the skin layers and loss of the deeper tissue layers, which may include subcutaneous tissue, muscle and bone. The time frame for repair and the repair process differ significantly.<sup>2</sup>

# Acute vs. Chronic Wounds

Another way to classify wounds is to consider them acute versus chronic. Acute wounds heal within an expected, predictable time frame and result in durable closure. In a healthy host an acute wound heals because of a physiologically complicated process or healing cascade of growth factors, cytokines and matrix proteins that keep the acute wound on a healing trajectory. However, this is not the case for all wounds, and complications may develop quickly, especially in those manifesting as deep penetrating wounds such as gunshot or stab wounds. Unhealthy individuals or those with multiple comorbidities may take longer to heal due to a combination of health/disease-related issues.<sup>3</sup>

Clinically, chronic wounds such as pressure injuries (ulcers), vascular ulcers, and neuropathic wounds behave differently and may be extremely slow to heal. A chronic wound by definition is a wound that has failed to progress through the “normal” healing process. Most wounds with a duration greater than 30 days are considered chronic wounds, and examples include pressure injuries, traumatic wounds, and lower extremity wounds.<sup>2</sup> Contributing factors include increased bacterial load, excessive amounts of matrix metalloproteinases or MMPs, elastase, and inflammatory by-products such as cytokines that may prolong the inflammatory processes and contribute to senescent, aberrant, or quiescent cells. In recent years wound care research analyzing the cellular, biochemical, and molecular components of acute and chronic wounds has significantly expanded the detailed complexities of normal wound healing and the pathophysiologic mechanisms of chronic wounds and has afforded us with treatment options to correct or control the underlying pathology. This has enabled us to change or update best practice based on current and ongoing research and evidence-based literature.



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# Overview: The Phases of Wound Healing

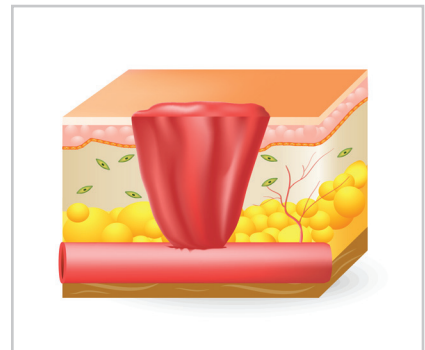
Historically, wound healing was regarded as a mysterious process, with wound care and management based on physician or practitioner preference as opposed to scientific principles. Hippocrates was the first to describe healing by first and second intention.<sup>4</sup> We now know that repair is a complex process based on scientific principles and evidence-based practice. Wound healing is a dynamic sequence of events that a wound follows as it progresses toward closure. It is critical to remember that wound healing is not linear, but is rather a cascade of events with many processes and cellular functions dependent on those that come before them. If this does not happen, healing slows or stalls, and next steps cannot occur properly. Then the wound is classified as a chronic wound. Wounds can progress both forward and backward through the phases depending on intrinsic and extrinsic forces at work within the patient. Now let's discuss the individual phases of wound healing in more detail.



Wound healing is a dynamic sequence of events that a wound follows as it progresses toward closure.

## Hemostasis

With injury that extends beyond the epidermis and into the dermis, bleeding occurs and triggers a series of overlapping events including coagulation pathways activation, fibrin clot formation, fibrinolysis supporting cell migration, release of growth factors, and vasoconstriction. This is known as hemostasis and occurs within 15 minutes of the initial injury. Blood supply to the skin is provided by the capillaries in the dermis, and when these vessels become damaged the coagulation pathways are activated as a result of platelet activation and aggregation, resulting in fibrin clot formation. This process is followed by fibrinolysis or clot breakdown, which releases growth factors. Vasoconstriction also furthers the process. Hemostasis is the body's normal response to tissue injury and actually initiates the wound healing cascade.<sup>2,5</sup>



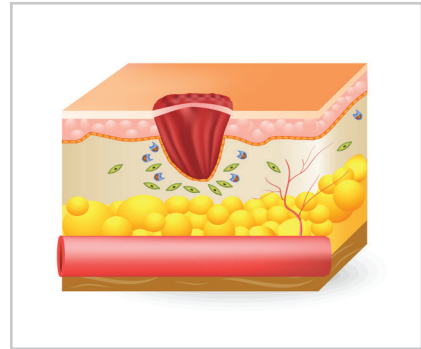
## Inflammatory Phase

The inflammatory phase usually last from one to five days. During the inflammatory phase fibrinolysis progresses and capillaries dilate, becoming permeable and allowing fluid and proteins to enter the area. Cell recruitment via chemotaxis, phagocytosis, and debridement occurs. There is also a release of cytokines and other bioactive mediators that trigger cell growth and activation, as well as reepithelialization. As a result of these processes clinical observations at this time may include erythema, localized edema, warmth, and pain. These are normal physiological responses to the inflammatory phase and not indicators of infection. Classic signs and symptoms of local infection may mimic the normal inflammatory responses. However, erythema, localized edema, heat, and pain beyond the expected time frame of the inflammatory phase may be signs and symptoms of a local infection.<sup>2</sup>



## Proliferative Phase

The next phase of wound healing is called the proliferative phase, which may take anywhere from 5 to 25 days and occurs only in full-thickness wounds or stage 3 and 4 pressure injuries. During this time macrophages communicate cellular signals to initiate granulation and epithelialization. The activity of fibroblast supports collagen synthesis and endothelial cells, which support the formation of new blood vessels or angiogenesis. The development of granulation tissue, contraction, and epithelialization occur in this phase. Healthy granulation tissue should be beefy red and have a bumpy appearance similar to a raspberry's surface. The newly formed granulation tissue consists of a matrix of fibrin, fibronectin, collagens, proteoglycans, glycosaminoglycans, and other glycoproteins. As the sides or edges of the wound contract or shrink, the wound's size decreases, resulting in a smaller area in need of granulation tissue. It also results in a smaller area of soft tissue defect and scarring. During epithelialization lateral and vertical migration of new epithelium, which manifests as a thin, silvery, white to pink layer at the wound edges, continues until the wound's surface is closed. If abnormalities occur during the proliferative phase there is an increased risk of wound dehiscence or opening of the wound edges of a previously closed wound that healed by primary intention.<sup>2</sup>

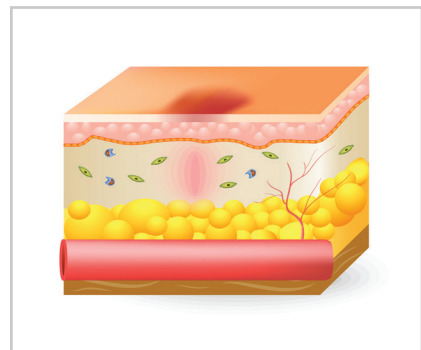


Cellular activities during maturation include capillary regression, new collagen deposition, and remodeling via apoptosis.

## Maturation Phase

The final phase in full-thickness wound healing is the maturation phase, also referred to as the remodeling phase, which begins approximately 21 to 30 days after wounding when the wound surface has closed with new epithelium. Completion of the maturation phase may take up to 24 months. Again, the maturation phase occurs only in full-thickness wounds and in stage 3 and 4 pressure injuries.<sup>2</sup>

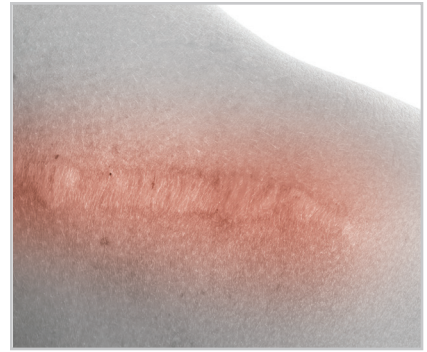
Collagen deposited during the proliferative phase is scattered in a random fashion. Cellular activities during maturation include capillary regression, new collagen deposition, and remodeling via apoptosis. In apoptosis old collagen is destroyed and replaced with collagen bundles, which are thicker and organized to provide tensile strength. This is also known as cross-linking. Clinical observations include continued contraction, shrinking, thinning, and paling of the scar. Is the tensile strength of a resolved full-thickness wound the same as that of the uninjured tissue? Within three weeks the tensile strength of the scar tissue is 20% of normal uninjured skin. As this phase progresses the tensile strength of scar tissue gradually reaches a maximum of 70% to 80% of that of the original tissue. For this reason, the area is always at increased risk of reinjury.<sup>2,5</sup>





## Scar Formation

A scar is the final result of the maturation phase and wound healing. It is essentially avascular and acellular and restores tissue continuity, as well as some degree of function and tensile strength. The scar may be normal, hypertrophic, or keloid. Hypertrophic scar formation and keloid scars are examples of abnormal repair or excessive scarring. Both types appear raised, are red or pink, and are often pruritic. Hypertrophic scars are typically confined to the original incisional or scar area, whereas keloids expand beyond the incision into the surrounding tissue. A wound is considered healed when continuity of the skin is reestablished, resulting in durable closure of tissue strength that is sufficient for normal activity.<sup>2,6</sup>

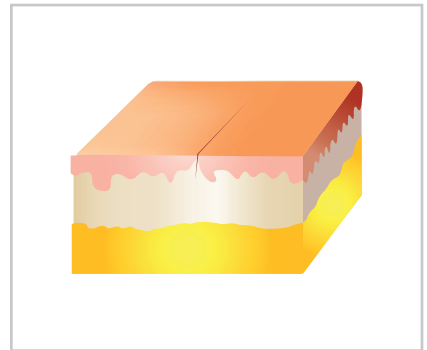


## Wound Closure



“The classification of wound closure... is based on the ideal of primary surgical closure for all wounds.”

The classification of wound closure or repair as primary, delayed primary (also referred to as tertiary intention), or secondary intention closure is based on the ideal of primary surgical closure for all wounds. In primary closure the wound edges are approximated by sutures, staples, or other means as they fuse and heal by epithelialization. An example is a surgical incision. Delayed primary closure means there is a risk of bioburden, and closure is delayed until bioburden is controlled. These wounds also heal by epithelialization. An example might be an abdominal wound that is initially left open for management of drainage and edema and then later closed.



In secondary closure the wound edges cannot be approximated so the wound does not close on its own due to significant tissue damage or loss. Wounds that close by secondary intention heal by granulation tissue formation, contraction, and epithelialization. Instead of a thin scar these scars are larger and, with decreased tensile strength, are at increased risk of reinjury. An example is a chronic wound such as a pressure injury or a laceration or incision that has opened or dehisced.

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