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Overview: Acute and Chronic Wounds

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Tissue injury is an inevitability of life. From the time we are young and sustain frequent cuts and scrapes to later years when surgery, trauma, or illness may result in more extensive tissue damage, we repeatedly experience the cycle of tissue damage–tissue repair. In most cases, wounds heal after an unimpeded and largely uneventful course. However, the healing process is not perfect, and healing impairments do occur. For individuals with wounds that persist and do not follow a normal healing trajectory, there are serious health concerns and quality of life issues. The magnitude and incidence of nonhealing or slow healing wounds are increasing as the population ages and chronic health conditions associated with wounds such as venous hypertension and diabetes become more prevalent. The most common types of chronic wounds are classified as venous, pressure, or neuropathic ulcers, which together account for 70% of all chronic wounds [1]. Statistics on chronic wound prevalence vary, however reports indicate that 0.2% to 1% of the population in developed countries suffer from venous ulcers, 0.5% from pressure ulcers, and 5% to 10% of people with diabetes experience neuropathic ulcers [2–4]. Presently, wounds are categorized broadly as acute or chronic, with specific wound types in each major category. At the advent of injury, all wounds can be considered acute regardless of cause. Accidents, trauma, burns, and surgery are events that precipitate acute wounds. Wound closure and the establishment of a functional result within an acceptable amount of time characterize acute wounds [5]. The healing process or trajectory can be depicted graphically as the percentage of the wound healed over time [6]. According to the present definition, wounds are considered chronic when healing time does not follow the expected course or fall within the range of what is considered a normal healing trajectory. The entire process is prolonged, and wounds diagnosed or categorized as chronic

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often resist attempts at treatment. In addition, the quality of tissue may be poor, and a functional closure is not achieved so the wounds may reoccur. In terms of the healing trajectory, the curve is shifted away from normal, indicating the delay in healing that typifies chronic wounds [1].

The basic physiologic responses to acute injury have been understood for some time. More detailed knowledge of cellular and intracellular responses to injury has recently developed. Understanding of the biologic intricacies of repair particularly at the molecular level continues to advance and inform the development of wound therapies and management strategies. It is through this growing description and knowledge of normal tissue repair that differences in the biology of nonhealing wounds has become clearer. These differences help explain variations in the healing trajectory of nonhealing wounds and offer promise for future treatments.

The process of tissue repair

The process of wound healing after full thickness injury is characterized by cell and tissue responses that can be grouped into major phases that include (1) hemostasis, inflammatory and proliferative cellular responses, and (2) synthesis of extracellular matrix consisting of new blood vessels, collagen, connective and epithelial tissue, and subsequent remodeling of this newly formed tissue. There are differences in the extent of repair depending on the type of tissue that is damaged and the depth of injury. Superficial or partial thickness injuries primarily require regeneration of new epithelium and minimal connective tissue synthesis, whereas full thickness, open wounds require the synthesis of new vessels, collagen, epithelium, and contractile forces to achieve closure.

Hemostasis, inflammatory and proliferative responses

Reestablishment of hemostasis is the physiologic goal of the body and the first major response to tissue injury. Hemostasis is achieved through the activation of the clotting cascade that is initiated when platelets are exposed to collagen from damaged blood vessels. Vasoconstriction and platelet aggregation follow, and the deposition of the fibrin clot establishes a provisional extracellular matrix [7]. The provisional matrix provides a scaffold for cell migration and entry into the injured area.

The inflammatory and proliferative cellular responses begin 24 to 48 hours after injury with the infiltration of neutrophils and then macrophages (peaking at approximately 5 days), fibroblasts (peaking at 7–9 days), and lymphocytes (peaking at approximately day 7) into the area of injury. Growth factors combined with adhesion molecules play critical roles in orchestrating and enabling the cellular responses required for successful healing. A large number of growth factors have been identified and linked to healing. Throughout the healing process, growth factors are released by cells