Perspective

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# Review Note on Clinical Recommendation

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## Description

Systemic steroids cause wounds to heal with incomplete granulation tissue and reduced wound contraction. Glucocorticoids also inhibit production of hypoxia-inducible factor-1 (HIF-1), a key transcriptional factor in healing wounds. Beyond effects on repair itself, systemic corticosteroids may increase the risk of wound infection.

While systemic corticosteroids inhibit wound repair, topical application produces quite different effects. These phases and their biophysiological functions must occur in the proper sequence, at a specific time, and continue for a specific duration at an optimal intensity. There are many factors that can affect wound healing which interfere with one or more phases in this process, thus causing improper or impaired tissue repair.

## Clinical Recommendations

Clinical recommendations suggest that, to avoid anti-platelet effects, individuals should discontinue NSAIDs for a time period equal to 4 to 5 times the half-life of drugs before surgery. There are few data to suggest that short-term NSAIDs have a negative impact on healing.

However, the question of whether long-term NSAIDs interfere with wound healing remains open. In animal models, systemic use of ibuprofen has demonstrated an anti-proliferative effect on wound healing, resulting in decreased numbers of fibroblasts, weakened breaking strength, reduced wound contraction, delayed epithelialization, and impaired angiogenesis. The effects of low-dose aspirin on healing are not completely clear. The skin healing process, according to Mitchel et al., illustrates the principles of repair for the majority of tissues.

Cell response in the inflammatory stage is characterized by the influx of leukocytes in the wound area. Such a response is very quick and coincides with the key signs of inflammation, which are revealed by the edema and the erythema at the location of the lesion. Parallel to all of the aforementioned events, the epithelial coating cells, through the action of specific cytokines, proliferate and migrate from the borders of the wound in an attempt to close it, which is called re-epithelialization. Attempts to restore the lesion induced by a local aggression begin very early on in the inflammatory stage.

One lesion is created by all of the stimuli that break the physical continuity of functional tissues. The stimuli that cause lesions can be external or internal, as well as physical, chemical, electric, or thermal. Approximately ten hours after the onset of the lesion, there is a development and stretching of the pseudopod projections of the keratinocytes, a loss of the extracellular matrix-cell and cell-cell contacts, a retraction of the tonofilaments, and the formation of actin filaments in the extremities of its cytoplasm's. A quick activation of the immune cells in the tissue may also occur, as happens with astrocytes, gamma-delta cells, and Langerhans cells, which secrete chemokine's and cytokines.

Inflammation is a localized and protective tissue response that is unleashed by the lesion, causing tissue destruction. The inflammatory response continues with the active recruitment of the neutrophils in response to the activation of the complement system, platelet degranulation, and bacterial degradation products.

These are attracted by many inflammatory cytokines produced by activated platelets, endothelial cells, and degradation products of pathogenic agents. Though first studied in a tumor context, the expression of normal promoter and/or inhibitor regulatory genes from cell growth, which are expressed in the cells present in the extracellular matrix, occurs in the healing process. Liu et al. described that the epithelial-mesenchyme transition can be regulated by microRNAs, as seen in miR-221, as well as by other oncogenes. The epithelial cells firmly adhere one to another, forming layers in which the basoapical polarity can be observed.

## Extracellular Matrix

Wounds that exhibit impaired healing, including delayed acute wounds and chronic wounds, generally have failed to progress through the normal stages of healing. Within the wound bed, fibroblasts produce collagen as well as glycosaminoglycans and proteoglycans, which are major components of the Extracellular Matrix (ECM). Once skin is injured, micro-organisms that are normally sequestered at the skin surface obtain access to the underlying tissues. The state of infection and replication status of the micro-organisms determines whether the wound is classified as having contamination, colonization, local infection/critical colonization, and/or spreading invasive infection.

Contamination is the presence of non-replicating organisms on a wound, while colonization is defined as the presence of replicating micro-organisms on the wound without tissue damage. Local infection/critical colonization are an intermediate stage, with micro-organism replication and the beginning of local tissue responses. In the reparative dermis, fibroblasts and endothelial cells are the most prominent cell types present and support capillary growth, collagen formation, and the formation of granulation tissue at the site of injury.

Following robust proliferation and ECM synthesis, wound healing enters the final remodeling phase, which can last for years. In this phase, regression of many of the newly formed capillaries occurs, so that vascular density of the wound returns to normal. One critical feature of the remodeling phase is ECM remodeling to an architecture that approaches that of the normal tissue. Such wounds frequently enter a state of pathologic inflammation due to a postponed, incomplete, or uncoordinated healing process. Most chronic wounds are ulcers that are associated with ischemia, diabetes mellitus, venous stasis disease, or pressure.

Non-healing wounds affect about 3 to 6 million people in the United States, with persons 65 years and older accounting for 85% of these events. In addition to the direct influences of anxiety and depression on endocrine and immune function, stressed individuals are more likely to have unhealthy habits, which include poor sleep patterns, inadequate nutrition, less exercise, and a greater propensity for abuse of alcohol, cigarettes, and other drugs. . Low-dosage aspirin, due to its anti-platelet function, is commonly used as a preventive therapeutic for cardiovascular disease, but not as an anti-inflammatory drug.