Current Controversies:

Are Chronic Wounds a Factor of Time or Aberrant Wound Biochemistry?

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HISTORICALLY, wounds have been categorized based upon duration; for example, ulcers that have been open for two to four weeks are viewed as chronic. However, as clinicians make decisions relating to treatment protocols, "factors of time" may only represent one component in the evaluation process.

By definition, acute wounds are those that heal in an orderly and well-orchestrated time frame, have no significant underlying pathologies to preclude healing, and rarely recur. Conversely, chronic wounds heal slowly, are frequently observed in patients with underlying comorbidities, and often have high rates of recidivism. How do these underlying conditions affect healing trajectories?

The answer may lie in understanding the science behind wound healing. The acute wound represents a nurturing environment filled with active growth factors and highly functioning fibroblasts and receptor sites, while retaining low levels of proteases and bacteria; this dynamic fosters robust cell proliferation and predictable wound healing as the cells move from hemostasis, through inflammation, proliferation, and remodeling with seamless efficiency.

However, the chronic wound milieu contains a toxic and aberrant biochemistry that supports ongoing inflammation, high levels of proteases (i.e. MMP 2,8,9 and serine elastase), and exorbitant bacterial loads leading to replicative senescence/ poor cell proliferation. This abnormal cascade stagnates receptor sites and essential cell activity (i.e. fibroblasts); the result is an ulcer that remains "stuck" in the inflammatory phase of wound healing in a cycle that remains unbroken without appropriate intervention.

In a consensus document lead by Snyder[1] and colleagues, it was hypothesized that when an individual with an underlying disease (i.e. diabetes) develops a seemingly "acute" ulceration, there may be an immediate paradigm shift in the lesion's biochemical profile leading to a "stalled" wound from the moment the epithelium is breached; patient factors and medications may also play a role. Therefore, a chronic and/or stalled ulcer may be viewed in similar fashion irrespective of duration; treatment in either scenario must include management of elevated bioburden and excessive protease activity while maintaining a moist wound healing environment.

As part of the wound bed preparation model (i.e. debridement, control of infection and moisture, and wound edge preparation) dressings that reverse this toxic environment (i.e. silver, ORC-collagen, instillation with negative pressure wound therapy) remain essential pieces of the treatment algorithm.

References:
1. Snyder RJ, Driver V, Fife C, et al. (2011) Using a diagnostic tool to identify elevated protease activity levels in chronic and stalled wounds: a consensus panel discussion. Ostomy Wound Management. 57(12): 36–46