Wound Debridement: Therapeutic Options and Care Considerations

Necrotic tissue and other similar substances serve to impede or totally halt wound healing. A wound bed in need of debridement will not improve until this impediment is removed. The importance of debridement has been known for centuries. Early descriptions of debridement date back to Hippocrates who described the deleterious effects of leaving necrotic tissue in wounds.

Benefits of wound debridement

What is wound bed preparation and why is it integrally related to wound debridement? Falanga defines wound bed preparation as the "global management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures." Wound debridement is only one component of this facilitative process. Wound bed preparation is really composed of four considerations that have been organized into the mnemonic device "**TIME**": ***T***issue that is nonviable or deficient must be debrided. ***I***nfection (or inflammation) must be reduced and managed. ***M***oisture imbalance or exudate control must be addressed to avoid desiccation or maceration. ***E***pidermal margins (or edges) of the wound must be examined for nonadvancement. Nonmigration of epidermal cells may signify the need for other adjunctive therapies.

What purposes do wound debridement serve? The word itself provides a clue because debridement derives from the French (*débrider*) meaning "to unbridle" or remove a restraint. The process of debridement is important for several crucial reasons: to enhance wound assessment, to decrease the potential for infection, to activate important cellular activity, and to remove physical barriers to healing (necrotic tissue).

Necrotic tissue prevents recognition of true wound depth, the presence of tunneling and undermining, and deep infected material. Provided adequate blood supply is present, necrotic tissue must be removed from any wound for optimal assessment.

Debridement helps to remove bacteria. Evidence suggests that significant numbers of bacteria in a wound will slow healing. When bacteria exceed 100,000 ($\mathbf{10^5}$) bacteria/g of tissue, wound healing processes do not proceed normally. It is unclear whether bacterial burden is a cause or consequence of impaired healing; however, it is clear that necrotic matter in a wound encourages growth of anaerobic bacteria that are deleterious.

Debridement also helps to remove biofilms, which are theorized to slow wound healing. Biofilms are certain bacteria and other organisms that are covered with an extrapolysaccharide matrix. Biofilms are resistant to antibiotics and the normal immune systems of the host.

Debriding processes may also ameliorate senescent cells. These aged cells have significantly less protein production and proliferation abilities. Debridement acts to reduce the presence of these senescent cells so that younger, healthier cells are available for wound healing. In addition, necrotic tissue leads to the release of endotoxins that inhibit keratinocytes and fibroblast activity.

Another role of debridement is to remove the excess tissue that surrounds chronic wounds. Neuropathic ulcers are often associated with callus formation. Excision of the callus allows tissue-healing cells to proliferate, migrate, and ultimately heal. Another benefit of debridement is its effect on growth factor activity. It is hypothesized that chronic wounds are lacking in these proteins or that they may be unavailable for wound healing processes because of binding to proteins present in the chronic wound. Debridement releases activated platelets that promulgate various growth factors and cytokines.

Necrotic tissue may also act to "splint" a wound; the presence of necrotic tissue can prevent closure by inhibiting wound contraction processes. Conversely, if a wound closes prematurely over necrotic material, it can lead to dead space and potential abscess formation.

The multiple beneficial components of debridement are critically important to optimal outcomes. If initial wound assessment is incorrect, for example, subsequent treatment will likely be problematic. A necrotic wound that is diagnosed as a pressure ulcer but is really pyoderma gangrenosum will not respond to pressure reduction but will respond to medication therapy (eg, steroids and debridement

to follow as necessary). For some persons with pyoderma gangrenosum, debridement may actually worsen the inflammatory process.

The process of wound healing: barriers and facilitators

Normal wound healing (the kind associated with acute wounds) is ordinarily structured in phases. Although the phases are often discussed separately, in reality they overlap. The four phases include hemostasis, inflammation, proliferation, and remodeling. Hemostasis follows injury immediately, and the primary purpose is clot formation. A major cell present in this phase is the platelet. Inflammation targets removal of bacteria and debris and, secondarily, stimulation of cells critical for subsequent phases. The major cell of this phase is the macrophage. Proliferation is the phase in which new blood vessels grow so that granulation tissue will form. The major cells are fibroblasts and endothelial cells. In the final phase, collagen deposited in the scar strengthens tissue to improve tensile strength.

Chronic wounds do not heal in this orderly and efficient way. Rather, systemic and local factors impede normal phase progression. These chronic wounds have been called “stuck” or “stunned” wounds. Barriers include systemic issues such as older (or very young) age, stress, malnutrition, poor tissue oxygenation, immune suppression, concomitant diseases like diabetes or cancer, medication therapy (steroids or chemotherapy), or irradiation. Local factors are also critically important, including poor perfusion, tissue edema, high bacterial burden, lack of wound moisture, use of cytotoxic agents, mechanical stressors, inappropriate wound care, and, pertinent to the current discussion, the presence of necrotic tissue. The last factor is of major importance. It is not accidental that the first factor in the TIME mnemonic is tissue debridement. Nonviable or deficient tissue will impede further improvement because it will be impossible to halt infection, to keep the wound bed moisture balanced, and to help epidermal edges come together. Stated simply, gangrenous, necrotic, devitalized, and ischemic tissue need to be debrided.

Debridement is a salient component of facilitators to wound healing. These facilitators include good nutrition, wound protection, a moist wound environment, adequate oxygen supply, appropriate bioburden, and amelioration of the cause of the wound if possible. However, even in the presence of multiple facilitators, overcoming necrotic tissue in a wound bed is difficult.

Special role of wound bed debridement

The positive clinical outcome of wound debridement is a viable wound base. This viability allows for the correct functioning of growth factors and decreased inflammatory cytokines, proteases, and deleterious substances. Debridement should be distinguished from wound cleansing. Wound cleansing is used to remove foreign materials, reduce bioburden, and ameliorate odor and exudates. Topical cleansing products include antiseptics, antibiotics, detergents, surfactants, saline, and water. Wound cleansing will not effectively debride a wound that has substantial necrotic tissue.

Chronic nonhealing wounds can endanger patients' well being. Bone infection (osteomyelitis), septicemia, and generalized sepsis seriously threaten patients' lives. Even without progressing to this level of severity, large chronically nonhealing wounds can lose large amounts of protein.

Optimal wound debridement is based on comprehensive patient and wound assessment. For example, a necrotic pressure ulcer will not improve despite quality debridement processes if the true causative factor (pressure) is not reduced or eliminated. Experienced clinicians can attest to the fact that previously treated pressure ulcers may develop new necrotic tissue if further pressure damage ensues. Similarly, no degree of debridement will control the venous hypertension associated with venous stasis ulcers. Once basic causes are addressed effectively, debridement of the wound bed can progress. Mounting evidence supports good wound cleansing, and debridement enhances wound healing. If gentle nontoxic cleansing does not remove superficial necrotic, nonviable tissue then other debridement methods should be enacted.

One caveat is noteworthy. Successful wound debridement will make a wound look bigger (and possibly worse) to nonprofessionals. The enlargement of the wound is actually promoting healing.

Documentation by the wound care professional should alert clinicians and appropriate significant others that debridement will likely make a wound look as if it is deteriorating before it will eventually improve.

What does nonviable tissue look like? Necrotic tissue generally takes two forms: slough and eschar. Slough is dead tissue that is moist and stringy and yellow, tan, gray, or greenish-gray in color. Eschar is desiccated dead tissue that looks leathery and may vary from thick to thin. Eschar is most often black but can also be red or tannish brown. Both slough and eschar are attached to the wound bed.

A critically important concept grounds the optimal use of debridement. Some wounds should not be debrided. An extremity ulcer with stable eschar is an example. For a limb without good blood supply, the eschar acts as a physiologic barrier to infection. The eschar should not be removed but rather protected. Likewise, a person who is at an end of life stage and has poor peripheral perfusion should likely not be subjected to invasive surgery. Not all patients with necrotic wounds need surgery before they die. Conversely, it is also central to optimal care to recognize when debridement is needed urgently. A person who has diabetes mellitus and presents with a necrotic foot ulcer that has clinical signs of infection (induration, fever, erythema, and exudate) needs surgical debridement in the immediate future.

Evidence-based practice and wound debridement

Traditionally, wound care and wound debridement specifically have been grounded in best practices approaches. Best practice approaches have been based on expert opinion, tradition, and anecdotal experience. In contemporary health care, best practice approaches are acceptable in areas where there is insufficient evidence to generate evidence-based guidelines.

More recently, wound debridement approaches have been scrutinized, and a more rigorous evidence base is emerging. The need to remove necrotic tissue is widely accepted. Indeed, the National Guideline Clearinghouse, in its recommendations for pressure ulcer treatment, stated that necrotic tissues should be debrided based on patient condition, treatment goals, and the amount of necrotic tissue in the wound bed. This recommendation is based on existing high quality evidence-based guidelines. However, no randomized controlled trials have been conducted that examine the effect of healing of debridement versus no debridement of chronic wounds. Indeed, to generate such a trial would create substantial ethical dilemmas for its researchers.

Evidence for the effectiveness of different methods of debridement is generally lacking, and methods of measurement are poorly controlled. Fortunately, some controlled trials are beginning to elucidate the “best” methods in selected situations and in comparison with other methods. For example, Sherman studied a cohort of 103 patients with 145 ulcers. Sixty-one of 70 patients received maggot or conventional treatment of wounds (moisture retentive dressings). He found that maggot debridement therapy was statistically significantly better in achieving greater and faster debridement than conventional therapy.

A recent study examined five randomized controlled trials of debridement of diabetic foot ulcers. Three trials used hydrogel compared with two trials that used sharp debridement and one trial that used larval therapy. The pooled analysis showed that the hydrogels were significantly more effective than gauze or standard care in healing diabetic foot ulcers. Another recent study examined the efficacy of two enzymatic agents (collagenase and papain-urea) on pressure ulcer debridement. The researchers concluded that debridement was more rapid with the papain-urea formulation. In 1999, Bradley et al reviewed 35 randomized controlled trials and summarized the evidence for relative effectiveness of different debridement methods. The studies used dextranomer beads, cadexomer iodine, hydrogels, enzymatic agents, zinc oxide tape, surgery, or sharp debridement, and maggots. The authors concluded that evidence was insufficient to promote one debridement method over another. Steed et al found in a retrospective review of data on diabetics with plantar ulcers with good blood supply that frequent sharp debridement coupled with recombinant growth factor therapy had a higher rate of healing versus those patients who underwent growth factor therapy alone.

Knowledge about the optimal frequency, extent, and type of debridement is limited. For the greatest level of support, systematic reviews should include only true experimental studies.

One way in which these systematic reviews are linked to patient care is clinical practice guidelines. These guidelines include available research evidence such as reviews of controlled clinical trials plus other available evidence pertaining to treatment and evaluation of outcomes. These guidelines are generally broader in scope. Clinical practice guidelines for wound care are available from many sources such as the Wound, Ostomy, Continence Nurses Society (www.wocn.org), the National Guideline Clearinghouse (www.guideline.gov), and the Agency for Healthcare Research and Quality (www.ahrq.gov/clinic/epcix.htm), to name only a few.

But no systematic review can replace critical clinical expertise. Once clinicians determine that wound bed debridement is necessary and safe, they need to select an appropriate method or methods, cognizant of how debridement processes work and the advantages and disadvantages associated with them. In this way, correct methods can be listed to appropriate patients.

Methods of wound debridement

Multiple methods are available for wound debridement, including surgical or sharp, mechanical, chemical, autolytic, enzymatic, biotherapeutic, laser, and “other” methods. Some methods are considered “selective” in that they remove only the necrotic or devitalized tissue. Nonselective methods remove normal as well as necrotic tissue. For obvious reasons, selective methods are usually preferred. Generally, there is no one best approach. Each method is appropriate for certain clinical situations and may be used in combination effectively—and so goes the search for the ultimate debridement tool or method. Rather, the choice of debridement method depends on multiple contextual factors associated with the patient and the wound. However, optimal use of wound bed debridement techniques ultimately depends on education and experience. An interesting phenomenon is occurring related to wound debridement methods. Older more “alternative” methods are reemerging as legitimate methods of topical therapy and debridement. These methods include biotherapy (eg, maggots) and the use of natural substances that can be categorized as “other” types of debridement, including the topical use of honey. In three recent controlled clinical trials, honey was associated with faster healing in superficial burns than transparent dressings or silver sulfadiazine. Honey is also associated with autolytic debridement, deodorizing action, and an antibacterial action.

Patient and family wishes must be considered along with best available evidence. Sometimes a best practices approach is not taken because a patient does not wish aggressive (or conversely, conservative) therapy.

Quality patient education and cultural competence and sensitivity play critical roles in the use of debridement approaches. In today's multicultural society, caregivers must be cognizant of ethnic and religious preferences. In the author's experience, patients or caregivers may be uncooperative with debridement approaches based on erroneous interpretations or perceptions. For example, a patient and his family initially refused an enzymatic debriding process because they thought it contained substances proscribed by their religion. Another patient feared a negative pressure wound device because it would injure (electrically shock) him. Both patients agreed to therapy when full processes and ingredients were explained and documentation was shared.

Another component of patient education regarding debridement is the need for ongoing debridement in chronic wounds. Maintenance debridement is necessary in chronic wounds in which the underlying pathology is associated with continuous recurrence of slough and eschar. Patients need to be counseled that continuing debridement does not constitute treatment failure or poor patient compliance.

Recent research shows that the most traditional, cost ineffective method, saline wet to dry gauze, is still the most commonly used approach even when there is little evidence to support its use (clean open surgical wounds healing by secondary intention). This suggests that tradition, lack of education, and poor understanding of cost efficacy drive many physicians' debridement choices.