Scope of practice

In all states, surgical and conservative debridement fall under the scope of practice for physicians, nurse practitioners, and physician assistants. Physical therapists, physical therapy assistants, occupational therapists, certified occupational therapy assistants, and nurses (both registered nurses and licensed practical/vocational nurses) are allowed to perform conservative sharp debridement in some, but not all, states.

What if you are licensed in a state that doesn't address if you can perform sharp debridement? In this situation, you should use a decision tree or algorithm to determine whether you can proceed. Some questions to ask are:

- Did my wound care training prepare me for debridement and am I competent to provide this service? Remember that in addition to the skill, you need to know how to manage the patient's pain and bleeding.
- Will I be providing this service under the supervision of a prescribing clinician who has expertise in debridement?
- Does my facility allow me to perform this procedure? (See *Sharp debridement policies and procedures*.)

The answers will help you make the best decision for you and your patient.

For more information or examples of policies, email the authors at cutabove@sharpdebridement.com. You can also **download a sample policy**[A]. ■

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Note: This article is intended to provide general information. For specific legal questions, contact an attorney.

Online Resource

A. woundcareadvisor.com/wp-content/uploads/2016/07/PP-Debridement-tool.pdf

Preparing the wound bed: Basic strategies, novel methods

Proper preparation promotes optimal outcomes.

By Kulbir Dhillon, NP, WCC

The goal of wound-bed preparation is to create a stable, well-vascularized environment that aids healing of chronic wounds. Without proper preparation, even the most expensive wound-care products and devices are unlikely to produce positive outcomes.

To best prepare the wound bed, you need to understand wound healing physiology and wound care basics, as well as how to evaluate the patient's overall health and manage wounds that don't respond to treatment. (See *Normal wound healing*.)

Basic wound care: DIME

To choose the right method of wound-bed preparation for a particular wound, first assess your patient's condition, wound history, physical wound characteristics,

and availability of resources. Local wound-bed preparation factors can be summarized by the acronym DIME—**D**ebridement, **I**nfection or **I**nflammation, **M**oisture balance, and **E**pithelial advancement. These four components address the various pathophysiologic abnormalities underlying chronic wounds.

D: Debridement

Nonviable tissue must be debrided because cell debris impairs healing. Research and clinical evidence show that debriding necrotic or fibrous tissue accelerates wound healing. (See *Types of debridement*.)

I: Infection or inflammation

Bacterial load directly impacts wound-bed preparation. Assess and treat the patient's wound for superficial or serious infection, persistent inflammation, extensive colonization, and cellulitis. With infection or inflammation, wound healing stalls because the extracellular matrix and growth factors degrade more rapidly than they synthesize, impeding progression toward the proliferative phase and ultimately affecting re-epithelialization. Managing the bacterial load with local or systemic therapy is crucial to wound-bed preparation.

M: Moisture balance

Appropriate moisture promotes the action of growth factors and cytokines and aids migration of cells, including fibroblasts and keratinocytes. However, attaining a moisture balance is challenging. Excessive moisture can damage the wound bed and surrounding skin, leading to maceration and skin breakdown. Inadequate moisture, on the other hand, can impede cellular activities and promote eschar, resulting in poor wound healing.

Normal wound healing

Wound healing involves a complex series of physiologic and pathologic events, including:

- cell division
- revascularization
- synthesis of new extracellular matrix (ECM)
- tissue formation and remodeling.

Soluble mediators, such as growth factors, cytokines, matrix metalloproteinase, and their regulators control many of these processes through their effects on various cell types and ECM.

Wound healing occurs in four overlapping but well-defined phases—hemostasis, inflammation, proliferation, and remodeling. In chronic wounds, this normal process is disrupted.

Be sure to evaluate the patient's nutritional status, cardiac and peripheral vascular status, and renal function. Check for risk factors that can cause moisture imbalance. Then identify an appropriate treatment plan. To increase compliance, explain planned interventions to patients and their caregivers. The typical plan includes medical wound management strategies, such as manual lymph drainage, compression devices and garments, absorptive dressings, and negative pressure wound therapy.

E: Epithelial advancement

Cellular dysfunction and biochemical imbalance can stall wound-bed progression by impeding epidermal cell and keratinocyte migration. Migration of epidermal cells and keratinocytes indicates the wound bed is adequately prepared. Wound contraction is another key sign of an adequately prepared wound bed.

Assessing the patient's overall health

Remember—you must treat the patient before you can treat the wound. Especially

Types of debridement

Autolytic, enzymatic, surgical, mechanical, or biological methods can be used to debride nonviable contaminated and infected tissues.

- *Autolytic debridement* uses the body's inherent ability to digest and remove necrotic tissues by promoting a moist wound environment that allows endogenous enzymes or phagocytic cells to liquefy such tissues. To some extent, all wounds go through autolytic debridement. Such debridement is slow, selective, painless, and noninvasive. However, it's not recommended for infected wounds and isn't the best choice for wounds with a large amount of necrotic tissue.
- *Enzymatic debridement* uses manufactured enzymes, such as collagenase, as debriding agents to dissolve necrotic tissue. Collagenase enzyme is safe on viable cells and can be combined effectively with moist wound healing. Like autolytic debridement, it's a slow method.
- *Surgical debridement* is indicated for wounds with extensive or adherent necrotic tissues. Chronic non-healing infected wounds may require surgical debridement down to the level of bone or muscle. The fastest way to create an acute healing phase, this method allows accurate assessment of wound severity and extent. It's particularly useful in life- or limb-threatening infections with necrotic eschar or gangrene.
- *Mechanical debridement* is a nonselective method that removes debris physically by separating nonviable tissues from the wound bed when the dressing is removed. A wet-to-dry dressing is the simplest form of mechanical debridement. Hydrotherapy (pressure irrigation) and whirlpool therapy loosen and help remove nonviable tissues, debris, and exudates. Mechanical debridement isn't recommended for wounds with fragile granulation.
- *Biological debridement* uses sterile maggots to digest slough and necrotic tissues and secrete bactericidal enzymes without damaging surrounding healthy tissue. Known for centuries to help heal wounds, maggots recently have made a comeback and are garnering renewed interest in biosurgical debridement. Biological debridement is a secondary debridement method for patients who aren't eligible for surgical debridement.

with difficult wounds, multiple comorbidities can delay or interrupt re-epithelialization. Adequate nutrition and smoking cessation are especially important. (See *How smoking and poor nutrition impair wound healing*.)

Checking for wound-specific problems

Assess the patient for two wound-specific problems—biofilms and abnormal matrix.

- *Biofilms* delay wound healing by creating a host-pathogen environment that promotes cohabitation of many bacterial species and anaerobes. These bacteria promote their own survival within the wound environment. Elderly patients and those with complex diseases, diabetic foot ulcers, venous leg ulcers, or pressure ulcers may develop extensive bacterial populations. That's why restoring bacterial balance is important in managing chronic wounds. Techniques for managing biofilms effectively include use of topical agents or systemic antibiotics and regular maintenance debridement.
- *Abnormal matrix* can develop in chronic wounds containing proteases that digest fibronectin and growth factors in the fibrin clot, causing a degraded matrix that no longer supports re-epithelialization or formation of granulation tissue.

Managing wounds unresponsive to treatment

Cells in chronic wounds become unresponsive, unable to divide or respond to such messengers as cytokines and growth factors. This results in phenotypic dysregulation. For successful wound-bed preparation, options may include bioengineered skin-cell therapy, stem-cell therapies, and platelet-rich plasma (PRP) for cutaneous wounds.

Bioengineered skin-cell therapy

Bioengineering treatments have provided viable therapeutic options, especially in managing chronic or difficult-to-heal wounds. Bioengineered skin and soft-tissue substitutes can be acellular or cellular.

Acellular products contain a matrix or scaffold composed of such materials as collagen, hyaluronic acid, and fibronectin. These products differ in various ways, including:

- species source (human, bovine, or porcine)
- tissue source (such as dermis, pericardium, or intestinal mucosa)
- additives (for instance, antibiotics or surfactants)
- hydration (wet or freeze-dried)
- required preparation (multiple rinses or rehydration).

Cellular products contain living cells, such as fibroblasts and keratinocytes within the matrix. Cells within the matrix may be autologous, allogeneic, or derived from other species (such as sheep or pigs). Skin substitutes also may be composed of dermal cells, epidermal cells, or a combination and may provide growth factors to stimulate healing. Topical growth factors are used as an adjuvant treatment to such therapies as debridement, off-loading, frequent dressing changes, and compression of wounds caused by vascular insufficiency

Stem-cell therapy

Use of stem cells in tissue regeneration has significant potential in cutaneous wound-bed preparation. Stem cells act in several ways to aid wound repair; in chronic wounds, they

How smoking and poor nutrition impair wound healing

Patients who smoke or have a poor nutritional status may experience poor wound healing.

Smoking

Smoking disrupts the normal healing process by damaging arterial endothelial function at many levels, decreasing cell proliferation and migration. Smoking also has a direct cutaneous effect, reducing cutaneous blood flow up to 40%; this can cause ischemia and impair healing. Smoking a single cigarette causes vasoconstriction for up to 90 minutes; smoking a pack leads to tissue hypoxia lasting an entire day.

To achieve positive outcomes, assist patients with smoking cessation and promote revascularization of ischemic wounds. The goal is to achieve a well-perfused wound bed and increase viability of active growth factors within the wound bed.

Poor nutritional status

Low protein intake can impede tissue granulation. To promote wound healing, optimize the patient's nutritional status by providing adequate protein.

Also, impaired glucose metabolism reduces the ability of erythrocytes to deliver oxygen to the wound bed—a fundamental step in collagen synthesis and tissue proliferation. A high level of hemoglobin glycosylation prolongs the inflammatory phase, decreases neutrophils, and reduces macrophage phagocytosis of bacteria, all of which directly affect wound-bed preparation. Encourage patients to eat a healthy diet. For those with diabetes, stress the importance of adhering to the treatment regimen.

could serve as an additional tool for therapeutic augmentation. This combined mode of repair and regeneration probably explains why cell therapies are more effective than simpler alternatives, such as direct growth-factor therapy treatment.

PRP

Multiple studies since the 1990s have shown promising results for PRP. For instance, researchers found PRP gel accelerates bone regeneration and studies confirmed the presence of specific platelet-derived growth factor receptors (PDGFs) in bone tissue. Research has uncovered strong evidence that these therapies are effective.

The PRP gel aids molecular and cellular induction of normal wound-healing responses, similar to platelet activation. In addition, PRP contains several growth factors and other cytokines that stimulate healing of bone and soft tissues. Autologous PRP is an advanced wound therapy used for hard-to-heal acute and chronic wounds.

Growth factors such as topical PDGF can help correct matrix abnormalities of growth factors. Also, studies have found that accelerated wound healing follows topical application of epidermal growth factor derived from the patient's own cultured keratinocytes, delivered in a fluid compress.

A systematic approach

When preparing the patient's wound bed and managing the wound, use a systematic approach that helps remove barriers to healing. Proper preparation helps ensure optimal patient outcomes. ■

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