



Wound Bed Preparation

It's About TIME

An important aspect of the TIME principle is the need to address nonviable or deficient tissue and restore the wound base and extracellular matrix proteins. In acute wounds, wound debridement is an effective way to remove necrotic tissues and bacteria so the wound can heal with relative ease. This is not the case for chronic wounds, where much more than debridement needs to be addressed for optimal results. Chronic wounds, such as venous ulcers, have a “necrotic burden” consisting of both necrotic tissue and exudate — as such, these wounds can be intensely inflammatory. They produce substantial amounts of exudate that interfere with healing and the effectiveness of therapeutic products such as growth factors and bioengineered skin. Therefore, in the context of wound bed preparation, clinicians need to remove not only eschar and frankly nonviable tissue, but also wound exudate.

Management of nonviable tissue (necrotic burden barrier) through debridement is one key aspect of the TIME principles through which we remove the barriers to closure and provide an optimal wound environment. Expert opinion advocates the removal of nonviable tissue as essential to promoting healing and reducing the risk of local infection, provided adequate blood supply to the wound is present.

This is part 4 of a 12-part series of articles on Wound Bed Preparation and TIME.



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The Problem — What is Nonviable or Deficient Tissue?

Nonviable or deficient tissue is collectively termed *necrotic tissue* or *slough*. Necrotic tissue may appear black or brown — slough is yellow and fibrinous. When the tissue dries out and develops a thick, deficient, leathery texture, it is called eschar.

Necrotic burden is used to describe necrotic material, nonviable tissue, exudates, and high levels of bacteria. Necrotic burden tends to accumulate continually in chronic wounds because such wounds generally result from underlying and uncorrected pathogenic abnormalities such as diabetes mellitus or venous insufficiency. Fully resolving these systemic problems often is impossible; in such cases, wound bed preparation is even more crucial to help facilitate wound closure.

The Solution — Debridement

What is debridement and why is it important? Debridement is the removal of dead (necrotic), devitalized, or contaminated tissue and foreign material from a wound — a key initial step in wound bed preparation.

The removal of necrotic tissue by debridement is important for a number of reasons. First, devitalized tissue in the wound bed will reduce the clinician's ability to adequately assess the depth of the wound or the condition of the surrounding tissue. Concealed dead spaces can harbor bacteria and increase the risk of local infection. Second, necrotic tissue also may mask signs of local wound infection. Finally, the presence of necrotic tissue is a physical barrier to healing and it supports bacterial growth. Bacterial colonies can produce damaging proteases that can break down important constituents of the extra-cellular matrix and inhibit the formation of granulation tissue and re-epithelialization. Therefore, in addition to removing cell debris, debridement reduces wound contamination and tissue destruction.

Types of debridement. There are five methods of debridement: surgical (or sharp), enzymatic, autolytic, mechanical, and biologic. Several factors can influence the choice of debridement method used,¹² including the size, position, and type of wound; moisture level; pain management; time available for debridement; and the type of healthcare setting. It is also important to consider the patient's overall condition when choosing the debridement method. In some cases, the use of more than one debridement method may be appropriate (see Table 1 and Table 2).

Surgical (or sharp) debridement. Surgical, or sharp, debridement is the fastest way to remove debris and necrotic tissue from the wound bed. Surgical debridement is sometimes performed when an extensive amount of necrotic tissue is present, which is often the case when the depth of the wound cannot be judged or when widespread infection requires the removal of bone and infected material.³ In addition to its efficiency, surgical debridement causes minimal damage to surrounding tissues and the minor bleeding that follows the procedure can release inflammatory mediators, such as cytokines, that can assist the wound repair process.

However, surgical debridement has limitations. It cannot be used for patients with bleeding disorders or who are immunocompromised. The procedure may be painful and may cause transient bacteremia and damage to nerves and tendons.⁴ Newer technologies such as the VERSAJET Hydrosurgery

System enable surgeons to remove damaged tissue and contaminants precisely without the collateral trauma often associated with current surgical modalities. Debridement of traumatic wounds, chronic wounds, and other soft-tissue lesions is achieved in a single step while sparing healthy tissue and permitting the healing process to progress naturally.

Enzymatic debridement. Enzymatic debridement uses manufactured proteolytic enzymes to remove necrotic tissue and cell debris from the wound. When these exogenous enzymes are applied directly to the wound surface, they work with naturally occurring enzymes to degrade necrotic tissue. One of the oldest types of enzymatic debriding agents, used for more than half a century, comprises a combination of papain and urea. Papain-urea products such as GLADASE Ointment provide debridement by first degrading the surface necrotic tissue and then debriding the surface of the wound.

Papain-urea chlorophyllin products such as GLADASE-C Ointment offer a combination of papain urea and sodium copper chlorophyllin that can be applied continuously throughout the treatment period to remove necrotic tissue and liquefy slough; thereby, preparing the wound bed for healthy tissue granulation and healing. Sodium copper chlorophyllin, a chlorophyll derivative, is an anti-agglutinin that may reduce inflammation in the wound. Chlorophyll is known for its ability to reduce odors.

Autolytic debridement. Autolytic debridement occurs naturally, to some extent, in all wounds. A highly selective process, it involves macrophages and endogenous proteolytic enzymes that liquefy and spontaneously separate necrotic tissue and eschar from healthy tissue. Wound dressings that maintain a moist wound bed can provide an optimal environment for autolytic debridement because they allow the phagocytic cells to liquefy necrotic tissue; thereby, promoting granulation tissue. Smaller areas of slough or necrotic tissue can be quickly and safely removed using interactive dressing products that enhance the body's ability to debride devitalized tissue by the process of autolysis. Maintaining a moist wound surface helps promote rehydration of slough and necrotic tissue while allowing leukocytes and enzymes present in exudates to break down avascular tissue. The speed of this process depends on a number of factors, including the size of the wound and the general physical condition of the patient. In many instances, significant improvement can be observed with 3 to 4 days. For dry wounds, autolytic debridement can be facilitated through the use of hydrogel, transparent film, or hydrocolloid dressings. For exuding wounds, absorptive dressings, such as a foam dressing like Allevyn, or alginate dressings can be used.

Autolytic debridement requires limited technical skill, is easy to perform, and does not damage healthy tissue surrounding the wound. Furthermore, the patient experiences minimal pain with this method. However, it is a slower method of debridement and may be contraindicated if a high bacterial burden is present in the wound.

Mechanical debridement. Mechanical debridement is a non-selective method that physically removes debris from wounds. Examples of mechanical debridement include wound irrigation, whirlpool therapy, and wet-to-dry dressings.

Wet-to-dry dressings are the simplest form of mechanical debridement.⁵ These dressings cause mechanical separation of

Part 4: Debridement and the Role of Enzymes

Table 1. Comparison of Debridement Methods

Type	Mechanisms	Precautions	Comments
Surgical/Sharp	Removal of devitalized tissue using curved scissors, curette, scalpel, laser, hydro-surgical	Make sure there is potential for healing and enough blood supply to support healing. In lower extremity ulcers: Ankle-brachial index >0.5m: Toe pressure >50 mm Hg, Transcutaneous oxygen saturation >30 mm Hg	Fastest and most effective way to remove debris and necrotic tissue that can serve as a nidus for infection. Local anesthetic — topical
Enzymatic	Exogenous topically applied chemical agents: papain-urea, papain-urea with chlorophyllin	A transient burning sensation may be experienced by a small percentage of patients	Enzymatic debridement can be facilitated by scoring the wound (cross-hatching of eschar without causing bleeding)
Autolytic	The use of moist interactive dressings to rehydrate eschar and help remove slough. Dressings include: transparent film dressings and hydrogels	Loose debris should be removed when changing dressings to avoid infection. Dressing changes are usually required every 24 to 48 hours initially	Occlusive dressings can relieve pain
Mechanical	The removal of necrotic tissue by means of force. Performed using: wet-to-dry dressings, hydrotherapy (whirlpool), pulsed lavage	Excessive force will cause tissue damage. Use an 18- to 20-gauge angiocath on a 30- to 60-cc syringe	Whirlpool or saline wet-to-dry (painful if bleeding induced with dressing change) are alternate mechanical debriding methods

Table 2. Debridement Guidelines

Description	Debridement Decision
Majority of wound is covered with necrotic tissue and suspected soft tissue or an especially large wound is covered with necrotic tissue	Surgical debridement is the fastest method of wound debridement
Majority of wound is covered with necrotic tissue (slough or eschar)	Apply a papain-urea ointment such as GLADASE or a papain-urea chlorophyllin ointment such as GLADASE-C to necrotic tissue.
Majority of wound is clean and/or granulating. Necrotic tissue (slough or eschar) present. Wound is dry or has a small amount of exudate	Apply a papain-urea chlorophyllin such as GLADASE-C Ointment to wound. Change dressing daily or as needed to avoid compromised healing
Necrotic tissue on feet and/or toes. Arterial insufficiency	Debridement not recommended. Refer to vascular specialist

necrotic tissue from the wound bed when the dressing is removed. However, this can cause the patient significant discomfort and damage newly formed tissue.⁶

Wound irrigation utilizes a pressurized stream of normal saline. High-pressure irrigation removes bacteria and necrotic debris from wounds but also can drive bacteria into soft tissue.⁷ Whirlpool therapy, another form of powered irrigation, loosens and removes necrotic tissue, debris, and exudates. Wound irrigation is suitable for inflammatory wounds but not for wounds that have fragile granulation tissue.⁸

Biologic debridement. In some care settings, maggot larvae facilitate removal of necrotic tissue.

Maintenance debridement: Why is an extended phase of debridement more appropriate than single-intervention therapy? Until recently, debridement — regardless of the method used — has been considered a single therapeutic step within defined timelines. While a single episode of debridement may be appropriate for acute wounds, chronic wounds require more frequent debridement because of accumulating necrotic burden that results from underlying pathogenic conditions. Likely, the accumulation of necrotic burden could, in itself, cause wound repair failure; therefore, continuous removal of necrotic burden is an important part of wound bed preparation.

For these reasons, when treating chronic wounds, debridement should be viewed as an ongoing wound treatment process

rather than a single intervention. An extended “maintenance” phase of debridement, which will offer distinct advantages in wound management, has been proposed. Because autolytic and enzymatic debridement are more selective and generally less painful for the patient, these options are the recommended methods of treatment when extended periods of debridement are required.





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WOUND BED PREPARATION

Removing the barriers

TIME[†] - Principles of Wound Bed Preparation

Clinical Observations	Proposed Pathophysiology	WBP Clinical Actions	Effect of WBP Actions	Clinical Outcome	SOLUTIONS
T issue Non-viable or Deficient	Defective matrix and cell debris impair healing	Debridement (episodic or continuous): - Autolytic, sharp surgical, enzymatic, mechanical or biological - Biological agents	Restoration of wound base and functional extra-cellular matrix proteins	Viable wound base	GLADASE[®] Papain-Urea Debriding Ointment**  GLADASE[®] C Debriding, Deodorizing and Healing Ointment**
I nfection or Inflammation	High bacterial counts or prolonged inflammation: + Inflammatory cytokines + Protease activity - Growth factor activity	Remove infected foci Topical/systemic - Antimicrobials - Anti-inflammatories - Protease inhibition	Low bacterial counts or controlled inflammation: + Inflammatory cytokines + Protease activity - Growth factor activity	Bacterial balance and reduced inflammation	 ACTICOAT[®] (with SILCRYST Nanocrystals)†
M oisture Imbalance	Dessication slows epithelial cell migration Excessive fluid causes maceration of wound margin	Apply moisture balancing dressings Compression, negative pressure or other methods of removing fluid	Restored epithelial cell migration, dessication avoided Edema, excessive fluid controlled, maceration avoided	Moisture balance	 ALLEVYN[®]
E dge of Wound Non Advancing or Undermined	Non-migrating keratinocytes Non-responsive wound cells and abnormalities in protease activity	Reassess cause or consider corrective therapies: - Debridement - Skin grafts - Biological agents - Adjunctive therapies	Migrating keratinocytes and responsive wound cells Restoration of appropriate protease profile	Advancing epidermal margin	 DERMAGRAFT[®] Human Fibroblast-Derived Dermal Substitute**

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