ELSEVIER

REVIEW ARTICLE

A Primer on Wound Bed Preparation

Chuck Gokoo, MD, CMO CWS, FACCWS^{a,*}

^aChief Medical Officer for American Medical Technologies, Irving, Texas, USA

KEYWORDS:

Wound Bed;
Wound Healing

Abstract Successful wound closure and healing are a major concern for today's clinician. Determining if the wound will progress or not relies on a comprehensive assessment, recognition of wound characteristics that will promote or impede the healing process and preparing the wound bed such that pathological features are removed allowing the healing cascade to occur. When complications are no longer a roadblock the wound will achieve a stable microenvironment and progress through the normal reparative process.

© 2009 Elsevier Inc. All rights reserved.

When a patient arrives with an acute or chronic wound, the wound care specialist begins with a comprehensive patient assessment. This consists of identifying preexisting signs or conditions of wound development; previous history of wounds by etiology, severity, and duration; and risk of infection. Diagnosis and differentiation of wound type (ie, arterial, diabetic, venous, pressure, etc) should include visual wound and periwound characteristics and vascular testing because clinical treatment for each may vary with etiology. Based on this assessment, the clinician must address the patient's and family's anxieties and concerns.

The next step is wound bed preparation. Evidence-based techniques are employed to prepare the wound and propel it toward healing. As the wound progresses toward healing, we may add healing enhancers to the prepared wound. These enhancers can include grafts (ie, xenografts or allografts, which are generally used on a temporary basis to cover large denuded areas), wound healing factors, bioengineered products, and moist wound healing products. This article explores wound bed preparation in detail.

Wound bed preparation¹ should create a microenvironment promoting wound healing. When planning the care

of wound, the clinician should consider significant factors which can impede the healing cascade:

1. reduction of bioburden levels preventing colonization or infection
2. management or prevention of dead/devitalized tissue (necrosis)
3. providing a moist environment by management of exudate levels
4. correction of cellular dysfunction
5. restoration of biochemical imbalance

Wound care specialists have identified 2 distinct models for dealing with wounds,^{2,3} an acute wound care model and a chronic wound care model (Figure 1).

In the acute wound care model, wounds heal spontaneously without complications through the phases of wound healing. Partial-thickness wounds, repair by regeneration (replication of like cells), and full-thickness wounds proceeding through 3 well-defined phases: inflammation, proliferation, and remodeling resulting connective scar tissue (Figure 2).^{2,3}

Research has shown that the pattern and the time frame of events in chronic wounds are different from those in acute wounds.⁴ Certain extracellular components that are normally not present by the end of the relatively short proliferative

* Corresponding author.

E-mail address: chuck.gokoo@amtoundcare.com

The Two Models of Wound Care

Figure 1 The 2 Models of Wound Care.

stage of wound healing have been found in chronic wounds for up to 12 months. The presence of inflammatory cells at the wrong phase in chronic wounds indicates that these wounds are in the inflammatory phase of wound healing and will not progress to the next phase of wound healing.⁴

The goal of chronic wound care is to create a stable, nondraining wound that displays well-vascularized granulation tissue (Figure 3). Wound bed preparation removes barriers to wound healing. The most common barriers are necrotic tissue, excessive bacterial burden, and excessive exudate levels.⁵ The clinician should begin the preparation process focusing on the triumvirate, debridement, bacterial (bioburden) balance and exudate management (Figure 4).

Debridement, the First Element

Falanga^{6,7} stresses the importance of debridement. Debridement is the excision of dead and nonviable tissue (ie, biofilm-embedded bacteria), resulting in the acceleration of wound healing. Optimum debridement can achieve a balance between removing necrotic tissue, preserving healthy tissue, and maintaining a healing environment.⁸ This is important when it is suspected that a wound may be infected.

Nonviable tissue is collectively termed necrotic tissue and most commonly encompasses slough and eschar. Necrotic material may appear in different colors but predominates as

The Goal of Chronic Wound Care

Figure 3 The Goal of Chronic Wound Care.

black- or brown-colored tissue. Yellow and fibrinous necrotic tissue is called slough. Dried-out tissue, forming a thick and leathery texture, is called eschar. Removal of this dead or devitalized tissue by debridement is important for the following reasons^{5,8}:

1. It removes the bioburden levels at the ulcer bed.
2. It stimulates growth factor activity.
3. It removes of senescent cells.
4. It removes hyperproliferative nonmigratory tissue.

It is critically important to remove bacteria from the wound.⁸ Necrotic tissue supports the growth of bacterial organisms. Infected or highly colonized wounds feature friable and hemorrhagic granulation tissue and decreased tensile strength. Biofilms (communities of bacteria and other organisms that are embedded within an extrapolymeric matrix) demonstrate increased adherence to the wound bed and increased resistance to antimicrobial agents and to the immune system of the host. Prolonged exposure to bacteria in chronic wounds prolongs the inflammatory response, resulting in lytic enzymes' damaging tissues and causing tissue hypoxia.⁹ Proteases released by bacteria attack growth factors vital to healing. Excessive exudate, which is a consequence of increased bacterial burden, degrades growth factors and matrix proteins, inhibiting cell proliferation.

The Acute Wound Model

Figure 2 The Acute Wound Model.

The Three Elements of Wound Bed Preparation

Figure 4 The 3 Elements of Wound Bed Preparation.

Debridement stimulates growth factor activity. Chronic wounds are deficient in and have reduced availability of growth factors. Necrotic tissue is unreceptive to growth factors and acts as a physical barrier for growth factor receptor interaction. Surgical or sharp debridement often results in bleeding, which stimulates production of blood-borne growth factors.¹⁰

Senescent cells (aged cells) are cells that have markedly reduced proliferation and protein production even though they remain viable.⁵ Senescent cells impair healing in chronic wounds and must be removed from the wound bed. Chronic wounds have fibroblasts that are less responsive to growth factors because of age. Debridement removes these senescent fibroblasts.⁵

Debridement also removes callus or hyperkeratotic tissue surrounding chronic wounds.⁹ The edge of a chronic wound is hyperproliferative and thickened. Hyperproliferative epithelium is nonmigratory and delays healing. Debridement allows normal cells to undergo normal proliferation, migration, and healing. Hyperproliferative tissue hides the depth of the wound.⁹

The following factors influence the choice of the method of debridement^{11,12}:

1. Size of wound
2. Position of wound
3. Wound etiology
4. Wound bed tissue involved
5. Moisture levels (exudate)
6. Pain management
7. Time available
8. Patient's overall condition

Debridement types and methods include surgical and sharp, enzymatic, autolytic, mechanical, and biological (Figure 5).⁸ Sharp debridement is often performed at the patient's bedside; is a conservative, selective measure; and may leave a thin margin of necrotic tissue in the wound. Surgical debridement is more extensive, often requires either general anesthesia or local infiltrate, and is performed in the operating room. Surgical debridement is

done when the patient's medical condition(s) or the wound characteristics are such that bedside debridement cannot be performed. Sharp and surgical debridement may convert a chronic wound into an acute wound by removing the eschar and the bacterial burden. Following sharp debridement a secondary debridement technique may be necessary.⁸

Enzymes are another method of debridement.¹³ The addition of proteolytic and other exogenous enzymes to the wound surface can break down necrotic tissue. Devitalized tissue will be digested and dissolved. Enzymatic debridement requires a good delivery system, a prolonged period of enzyme activity, and the correct wound environment, which includes moisture, appropriate wound pH, and temperature. Enzymes are inactivated by heavy metals.¹⁴ There are generally 2 classes of enzymatic agents¹⁴:

1. Papain and urea agents in a cream base
2. Collagenase agents in petrolatum base

To determine which should be used, it is important to determine the nature of the necrotic tissue and the pathophysiology of the wound bed.¹⁴ Collagenase is a water-soluble enzyme that specifically breaks down collagen. At a physiological pH (6–8), it will break down necrotic tissue. Papain is a proteolytic substance that digests fibrolytic necrotic tissue and is not active against collagen.

Autolytic debridement uses the body's own proteolytic enzymes and defense mechanisms to liquefy and digest necrotic tissue.⁸ Autolytic debridement relies on enhancing the natural process—selective liquefaction, separation, and digestion of necrotic tissue and eschar from healthy tissue—that occurs in wounds because of macrophage and endogenous proteolytic activity in a moist environment. The use of occlusive, semioclusive, or moist interactive dressings can promote both phagocytic activity and the formation of granulation tissue. While autolytic debridement is recognized as an effective means of debridement, it is a slow process, requiring multiple dressing applications, periwound prevention of maceration, and irrigations over a prolonged period of time. Autolytic debridement is often used in combination with other debridement techniques and is contraindicated where bacterial loads may be heavy or the wound is clinically infected.

Mechanical debridement involves the use of nondiscriminatory physical force to remove necrotic tissue and debris from the wound surface.⁸ Wet-to-dry dressing usage is now considered a means of nonselective remove tissue. That is, both healthy and necrotic tissue is removed at dressing change, and the process can be painful as well as damaging to healthy tissue. Alternative methods of mechanical debridement include wound irrigation, ranging from cleansing to pressure irrigation; whirlpool therapy; ultrasonic therapy; and cavitation.

Biodebridement (maggots) uses the sterile larvae of *Phaenicia sericata*, the greenbottle fly.¹⁵ These larvae secrete chymotrypsin, which softens the dead tissue and allows the larvae to devour the necrotic tissue and bacteria

Elements of Wound Bed Preparation Debridement

Figure 5 Elements of Wound Bed Preparation Debridement.

in a wound.¹⁵ Maggot debridement is rapid, selective, and painless. Thirty larvae consume approximately 1 g of tissue per day. This type of debridement is contraindicated with wounds that are tunneling, undermining, or may involve bone tissue. This type of debridement is often reserved for specialized wound cases requiring extensive but rapid debridement.

Bacterial Balance

Bacterial balance is the second element of wound bed preparation.¹ The variables that affect the bacterial burden of a wound are the amount of necrotic tissue, the number of microorganisms, the bacterial virulence, and the host resistance. Biofilms are highly organized bacterial colonies contained within an extracellular polysaccharide matrix in which the bacteria become embedded for protection (a barrier to microbial agents).

Of all the factors controlling infection, host resistance is probably the most important determinant of wound infection. Factors influencing host resistance include vascular disease, edema, diabetes mellitus, alcohol abuse, poor nutritional status, smoking, and immunosuppression (use of steroid medications). The formula for infection clearly demonstrates the influence of host resistance.¹⁶

$$\text{Infection} = \frac{\text{Dose} \times \text{Virulence}}{\text{Host Resistance}}$$

The bacterial status of a wound varies over time. Changes that indicate a wound is deteriorating include increases in exudates, pain, odor, bleeding, and tissue friability.

A clinical infection is determined by bacteria load, and a load greater than 100,000, or 10^5 , bacteria per gram of tissue or milliliter of fluid is indicative of infection.^{16,17} Tissue biopsy or punch biopsy is the most definitive method of quantifying the bacteria type. Often when this cannot be done, a swab culture (semiquantitative method) that uses the Levine method may provide an accurate culture. The Levine collection is performed by rotating a swab culture over a 1-cm² area of the wound with sufficient pressure to extract fluid from within the wound tissue. This swab is then sent off for laboratory analysis. It is important that prior to a swab's being performed, the wound is cleaned with normal saline and the swab is taken from viable tissue (bacteria growing in the tissue) and not from dead or devitalized tissue, because this tissue harbors exogenous bacteria and may produce a false finding.¹⁷

Local signs of infection often include erythema, warmth, edema, induration, and pain. These signs are also seen during the inflammatory and proliferative phase of wound healing, which makes it difficult at times to make the determination of infection. When any of these signs, plus purulent drainage, crepitation, foul odor, pocketing at the base of the wound, discolored or friable granulation tissue, or tissue breakdown,

are seen in concert, it is safe to say that bacterial infection at the wound site should be ruled out.¹⁷

Exudate Management

The third element of wound bed preparation is the management of exudate levels at the wound bed.¹ Excessive exudate has an adverse effect on wound healing.⁵ Chronic wounds may produce large amounts of exudate as a by-product of inflammation. Chronic wound fluid is biochemically different from acute wound fluid (elevated levels of metalloproteinases break down matrix proteins; increased macromolecules bind growth factors inhibiting cell proliferation).¹⁸ Exudate from chronic wounds has been shown to impede or block the proliferation of key cells in the wound healing process, such as keratinocytes, fibroblasts, and endothelial cells. This action interferes with growth factor availability and inactivates essential matrix material. Exudate results in a loss of protein to the host, can damage the surrounding healthy skin, and is an excellent culture medium for bacterial growth.¹⁸

In chronic wounds it is not possible to remove the underlying pathogenic abnormalities in a single therapeutic step, and the "necrotic burden" continues to accumulate. Therefore, debridement should be achieved when it is necessary to remove necrotic tissue and should not be done at each and every wound observation and dressing change but rather as needed.

Cellular Dysfunction and Biochemical Imbalances

Cellular dysfunction and biochemical imbalances are the fourth and fifth elements of wound bed preparation.^{1,10} Both of these elements are directly related to the general nutritional status of the patient, a factor that needs to be addressed as part of the patient's initial assessment.

Chronic wounds have several distinctive features that affect their ability to heal in a timely manner.¹⁰ Alterations in cellular function and biochemical balances can keep a chronic wound "stuck" in the inflammatory, proliferative phase of healing the wound. Cells in a chronic wound become senescent. They become old too early. They do not respond to chemical messengers. Cytokines and growth factors, essential to healing of the wound, become ineffective in senescent cells.

Chronic wound fluid contains chronic inflammatory cells as a result of the prolonged inflammatory proliferative phase. The effect is dramatic. Apoptosis, the genetic code to normal cell death, is inhibited and delayed. There is defective extracellular matrix remodeling, reepithelialization failure, failure of wound edge migration, increase in senescent fibroblasts becoming unresponsive to growth factors, and a further increase in senescent cells.¹⁰

Without addressing underlying cellular dysfunction and biochemical balances, topical agents and treatments may not have the anticipated effect on wound healing.¹⁰ Cellular dysfunction and biochemical imbalances compromise the overall anatomic and functional integrity of the wound. Most chronic wounds are complicated by underlying physiological issues, such as diabetes, vascular insufficiency, ischemia, and nutritional deficiencies that also contribute to wound failure. Healing of the wound requires addressing these issues as well as the underlying pathology.

Conclusion

In summary, as a dynamic process, wound bed preparation must be continued on a maintenance level to keep all the elements of wound bed directed toward healing. It requires an in-depth understanding of the patient's history, the underlying medical conditions, as well as the etiology of the wound and its environment.

References

1. Vowden K, Vowden C: Wound bed preparation. Available at: <http://www.worldwidewounds.com/2002/april/Vowden/Wound-Bed-Preparation.html>. Accessed October 27, 2008.
2. Ayello E, Cuddigan J: Conquer chronic wounds with wound bed preparation. *Nurs Pract*. 2004;29(3):8–25.
3. Sussman C, Bates-Jensen BM: *Wound Care: A Collaborative Practice Manual for Physical Therapists and Nurses*. (2nd ed.). Gaithersburg, Maryland: Aspen Publishers; 2001.
4. Menke N, Ward K, Witten T, et al: Impaired wound healing. *Clin Dermatol*. 2007;25(1):19–25.
5. Kirsner R: Wound bed preparation. Available at: <http://www.o-wm.com/article/1347>. Accessed October 27, 2008.
6. Falanga V: Classifications for wound bed preparation and stimulation of chronic wounds. *Wound Repair Regen*. 2000;8(5):347–352.
7. Falanga V, Brem H, Ennis WJ, Wolcott R, Gould LJ, Ayello EA: Maintenance debridement in the treatment of difficult-to-heal chronic wounds. *Ostomy Wound Management* (supplement) 1-13, June, 2008.
8. Dolynchuk KN: Debridement. In: Krasner D, Kane D, editors. *Chronic Wound Care: A Clinical Source Book for Healthcare Professionals*. 3rd ed. Wayne, PA: HMP Inc., 2001. p. 385–390.
9. Leaper D: Sharp technique for wound debridement. Available at: <http://www.worldwidewounds.com/2002/december/Leaper/Sharp-Debridement.html>. Accessed October 27, 2008.
10. Enoch S, Harding K: Wound bed preparation: The science behind the removal of barriers to healing. *Wounds*. 2003;15(7):213–229.
11. Vowden KR, Vowden P: Wound debridement, Part 1: Non-sharp techniques. *J Wound Care*. 1999;8(5):237–240.
12. Vowden KR, Vowden P: Wound debridement, Part 2: Sharp techniques. *J Wound Care*. 1999;8(6):291–294.
13. Salcido R: Enzymatic debridement: A tried and tested method. *Adv Skin Wound Care*. 2002;13(3 Pt 1):92.
14. Falanga V: Wound bed preparation and the role of enzymes: A case for multiple actions of therapeutic agents. *Wounds*. 2002;14(2):47–57.
15. Armstrong D, Mossel J, Short B, et al: Maggot debridement therapy: A primer. *J Med Podiatr Med Assoc*. 2002;92(7):398–401.
16. Robson MC: Wound infection: A failure of wound healing caused by an imbalance of bacteria. *Surg Clin North Am*. 1997;77(3):637–650.
17. Dow G: Infection in chronic wounds. In: Krasner D, Kane D, editors. *Chronic Wound Care: A Clinical Source Book for Healthcare Professionals*. 3rd ed. Wayne, PA: HMP Inc., 2001. p. 343–356.
18. White R, Kutting KF: Modern exudate management: a review of wound treatments. Available at: <http://www.worldwidewounds.com/2006/september/White/Modern-Exudate-Mgt.html#ref4>