



Wound Bed Preparation

It's About TIME

Patients referred to a wound care clinic must be prepared to heal. Despite innovative, new technologies such as growth factors, bio-engineered skin products, and vacuum-assisted closure, chronic wounds will not progress to healthy closure without consideration of a number of patient and wound factors. Clinicians must address key initial components of wound management — treat the cause of the chronic wound, appraise patient-centered concerns, determine wound chronicity/location/dimensions/condition, and assess pain — and ensure the wound bed is appropriately prepared. Only then can the maximum benefits from advanced wound care products be derived and wound healing achieved.

The chronic wound is not the acute wound. Lessons learned regarding treatment of acute injury cannot readily be extrapolated to chronic wound healing. Wound bed preparation provides a coordinated, systematic approach specific to chronic, non-healing wounds that incorporates evidence-based best practices, allowing clinicians to more completely explore and initiate long-term yet efficacious strategies to their fullest advantage.

The wound bed preparation algorithm comprises four major care considerations: management of non-viable tissue (necrotic burden balance); management of inflammation or infection (bacterial balance); management of moisture imbalance (exudates management); and management of the edge of the wound. These concepts have been captured and organized into the TIME system for wound management. This series of outserts will provide information on implementing this approach. Clinicians can use the TIME principles to remove major obstacles to optimal closure of chronic wounds, intervening in four clinical areas to establish a wound bed environment that facilitates therapeutic measures — a wound that is prepared to heal.

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83 General Warren Boulevard, Suite 100
Malvern, PA 19355
Phone (800) 237-7285 FAX (610) 560-0502
www.hmpcommunications.com



The Problem — How to Correct Chronic Wound Abnormalities to Enable Healing

What is normal healing? Wound healing occurs in four dynamic, overlapping phases: the vascular response, the inflammatory response, the proliferative phase, and the maturation phase. Wounding initiates the vascular response: blood vessels constrict to staunch bleeding, clotting accelerates through a process called the coagulation cascade, fibrin mesh is formed to temporarily close the wound, and large amounts of blood and serous fluid help cleanse the wound surface. The inflammatory response is distinguished by vasodilation, increased capillary permeability, activation of polymorphonuclear leukocytes, and macrophage migration. Prostaglandins and histamines (inflammatory mediators) are released. The nutrients, growth factors, and enzymes in wound exudates perform antimicrobial, nutritional, and cleansing functions. Phagocytic cells (neutrophils) attracted to the injured area protect against micro-organisms. Macrophages, crucial throughout the healing process, initially cleanse the wound and produce regulating and complimentary substances, eventually inducing fibroblast and endothelial cell proliferation that assists in granulation tissue formation; in short, macrophages effectually control the transition between the inflammatory and proliferative phases.

In the proliferative phase, the wound fills with new connective tissue and decreases in size through granulation (creation of the scaffolding on which connective tissue will form), contraction (fibroblasts congregated around the wound pulling in the edges), and epithelialization (epithelial cells migrating across the granulation tissue to affect closure). The maturation phase is notable for increasing tensile strength and decreasing cellularity. Scar tissue matures and remodels; the wound has healed.

The chronic wound. Chronic wounds, for a variety of reasons, become “stuck” in the inflammatory and proliferative phases due to a variety of factors, including cellular dysfunction and dysregulation caused by abnormal over-expression of extracellular matrix molecules as well as trapped or sequestered growth factors. Chronic wounds are characterized by the presence of necrotic and unhealthy tissue, lack of adequate blood supply, absence of healthy granulation tissue, lack of re-epithelialization, and the presence of senescent cells, high levels of bacteria, and biofilms.

The Solution — the Wound Bed Preparation Model

The wound bed preparation approach enables the clinician to determine and address the reasons for delayed healing and to restart the healing process by removing barriers to healing.

Patient assessment. The wound bed preparation algorithm starts with patient assessment. The patient must be assessed for wound healability, including systemic and local factors. Systemic factors include metabolic disorders (eg, diabetes), respiratory disorders (eg, chronic obstructive pulmonary disease), circulatory disorders (eg, anemia), immune deficiency (eg, rheumatoid arthritis), immunosuppressive therapy, nutritional state, and medications. All must be corrected or modified to support healing and treatable causes of tissue damage remediated. Patient-centered concerns (ie, decision-making, pain, quality-of-life issues) must be considered and education and support provided. The general health of the patient will determine the ability to heal.

Wound assessment. The wound must be assessed for chronicity, location (particularly with regard to blood supply), dimension, wound pain, condition of the wound bed (color is a simple, immediate indicator), exudates, and periwound skin. Wound assessment helps identify prolonged inflammatory response, infection, necrotic tissue, unhealthy granulation tissue, delayed epithelialization, skin maceration, and skin sensitivities and allergies to provide the tools necessary for appropriate treatment.

Preparing the Wound Bed

Once initial assessments have been completed and contributory obstacles addressed, actions taken to prepare the local wound bed can be more effective. The principles of wound bed preparation have been summarized by the acronym TIME — **T**: tissue, non-viable or deficient; **I**: infection or inflammation; **M**: moisture imbalance; **E**: edge of wound, non-advancing or undermined.

Tissue — non-viable or deficient. Non-viable or deficient tissue is referred to as necrotic tissue or slough. Necrotic (dead) tissue may appear black or brown; slough is yellow, fibrinous tissue. Eschar is tissue that is dried out, thick, deficient, and leathery. Necrotic material, non-viable tissue, exudates, and high bacteria levels are collectively termed *necrotic burden*. Because chronic wounds generally result from underlying and uncorrected pathogenic abnormalities (eg, diabetes or venous insufficiency), necrotic burden has a tendency to accumulate continually, underscoring the need for wound bed preparation.

Debridement. Removing necrotic, devitalized, or contaminated tissue and foreign material is a crucial first step in wound bed preparation. Debridement facilitates this process. Devitalized tissue in the wound bed reduces clinician ability to adequately assess wound depth or condition of the surrounding tissue — concealed dead spaces may harbor bacteria, increasing the risk of local infection. Bacterial colonies produce damaging proteases that can break down important components of the extracellular matrix and inhibit formation of granulation tissue and re-epithelialization. Necrotic tissue also may mask signs of local infection and represents a physical barrier to healing. In addition to removing cell debris, debridement reduces wound contamination and destruction. Debridement may be performed surgically (sharp), enzymatically, autolytically, mechanically, and biologically. Factors influencing choice of debridement include the size, position, and type of wound; moisture levels; pain management; time available for the procedure; healthcare setting; and overall patient condition. Additionally, some clinicians may choose to use more than one method for debridement. For example, enzymatic debridement with products such as GLADASE ◊ or GLADASE ◊ C from Smith & Nephew may be used between episodes of sharp or surgical debridement to facilitate the removal of necrotic tissue.

Part 1: An Introduction to TIME

Infection or inflammation. Because clinical studies have shown that wounds with high bacterial burden are less likely to heal, decreasing bacterial burden is a vital element of wound bed preparation. Chronic wounds exist along a bacterial continuum — determining where in the continuum the wound fits is important to appropriate management. The continuum ranges from contamination to infection:

Contamination — non-replicating organisms are present in the wound

Colonization — replicating organisms (eg, *Staphylococcus epidermidis* and the genus *Corynebacterium*) are present in the wound but do not cause injury to the host

Critical colonization — replicating organisms are beginning to cause local tissue damage and delay healing. The subtle (also referred to as “secondary”) signs and symptoms of this category include stalled healing, change of wound bed color, friable granulation tissue, absent or abnormal granulation tissue, abnormal odor, increased serous exudates, and increased wound site pain; these symptoms may be overlooked by the clinician

Infection — replicating organisms are capable of causing host injury. Classic signs and symptoms include advancing redness (erythema), fever, warmth at the wound site, edema (swelling), pain, foul odor, and pus.

Wound cleansing and appropriate dressing selection may support continued progress of the wound that is contaminated or colonized. However, once the wound reaches the point of critical colonization, topical antimicrobial products such as ACTICOAT ◊ Nanocrystalline Silver Dressings may be considered as a part of topical therapy. When infection moves into the surrounding tissue and beyond, the addition of systemic antibiotic treatment is important.

Variables. To determine the effect of infection variables on the wound, the following formula may be applied:

$$\text{Risk of wound infection} = \frac{\text{bacterial dose} \times \text{virulence}}{\text{host resistance}}$$

Along with bacterial quantity and virulence of the organism, host factors are important — immunosuppression, diabetes mellitus, and certain medications can affect bacteria in a wound and impair healing.

Biofilms have a recognized influence in wound infection. Proliferating bacteria form microcolonies that attach to the wound bed and secrete a glycocalyx — biofilm — that protects the organisms. These bacterial colonies undergo numerous changes, expressing different genes that can alter organism sensitivity to antimicrobial agents such as antibiotics and antiseptics, inhibiting agent effectiveness.

Moisture imbalance. Wound exudate production is a normal part of the inflammatory phase of wound healing; blood capillary permeability increases, allowing protein-rich fluid to flow into the interstitial spaces. Increased fluid production facilitates cleansing of the wound surface and helps provide a moist local wound environment to enhance healing. Too much wound fluid, however, can inhibit wound healing; chronic wound fluid can break down extracellular matrix proteins and growth factors and inhibit cell proliferation, leading to the degradation of the tissue matrix. In addition, excessive moisture can be associated with bacterial burden, breakdown of necrotic tissue, or edema of the lower extremities as in cases of venous insufficiency and venous leg ulcers —

compression therapy may be needed to reverse the effects of lower extremity edema. A moisture-balanced wound-dressing interface must be maintained as part of wound bed preparation. Dressing selection is key to the effectiveness of any chronic wound healing regimen.

Although there are many dressings available for managing wound exudate, Smith & Nephew's ALLEVYN ◊ products offer a wide variety of products designed for wounds with low, moderate and heavy exudate levels. The ALLEVYN family also offers a number of specialty dressings specifically made for heels, sacrum, tracheostomy/tube sites and wound cavities.

Edge of the wound (epidermal margin): non-advancing or undermined. The epidermis in chronic wounds fails to migrate across the wound bed. Widespread hyperproliferation associated with the wound margin results from the inhibition of apoptosis (normal programmed cell death) within the fibroblast and keratinocyte cell populations and interferes with normal cellular migration over the wound bed. Other phenotypic abnormalities associated with chronic wound fibroblasts include an altered morphology, a reduced rate of proliferation, and a decreased response to exogenous application of growth factors, perhaps because fibroblasts from chronic wounds are senescent. Failure of the wound margin to migrate across the wound bed also may indicate that the margin may be undermined, often a sign of critical colonization or infection.

There are many ways of assessing advancing wound margin (or wound size). The VISITRAK ◊ system from Smith & Nephew is a new technology that accomplishes wound measurement in a quick, accurate, and repeatable manner. As opposed to the approximation of size, which results from L x W and counting squares methods, VISITRAK is a portable planimetry-based device that provides instant data on several wound size metrics.

If the principles of wound bed preparation have been successfully applied to this point, the patient should be re-assessed to ensure that tissue perfusion is adequate and that patient abnormalities/co-existing conditions (ie, ischemia, diabetes mellitus, malnutrition, alcoholism, cardiac failure, hepatic dysfunction, and immunosuppressive drug use) have been addressed. DERMAGRAFT ◊ human fibroblast-derived skin substitute, skin grafting, biological, or other adjunctive therapies may need to be implemented.

Conclusion





Wound bed preparation following the TIME approach enables clinicians to address the key obstacles to healing chronic wounds. The maximum benefit from evolving therapies and technologies cannot be achieved without a greater understanding of the often neglected basic principles of wound management inherent in the wound bed preparation model. This series comprises the research and experience of wound care experts who have studied and implemented evidence-based protocols to accomplish the goal of wound bed preparation — wound healing that leads to complete wound closure naturally or through the use of technologically advanced skin/wound products. It's all about TIME.

A clinical concept led by **smith&nephew**

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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* Courtesy of International Advisory Board on Wound Bed Preparation
† Schultz, Sibbald, Falanga, et al. (2003) Wound Repair and Regeneration Supplement, Table 6