



Wound Bed Preparation: It's About TIME

Chronic wounds present a unique set of challenges for wound care clinicians. Unlike acute wounds that progress through four phases to closure, chronic wounds become stuck in the intermediate phases and require intervention to remove the barriers to healing and to restart the healing process. Specific wound types, such as venous ulcers, require particular approaches within the realm of wound bed preparation to remove major obstacles to optimal closure of the chronic wound.

This is the second of 12 supplements that will discuss various aspects of the TIME principle.



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The Problem — How to Address Venous Insufficiency to Manage and Prevent Venous Ulcers

Venous insufficiency and related ulcers affect as much as 1% of the population — 22% of patients experience their first ulcer by age 40 and more than 70% by age 60. Venous ulcers are associated with pain, decreased mobility, negative self-image, and feelings of anger, fear, isolation and depression.^{1,2}

Managing patients with venous insufficiency and venous ulcers can be challenging for the staff in the outpatient setting. Venous ulcers are often highly colonized, with moderate to large amounts of exudate. The chronic nature of venous ulcers and recurrence rates as high as 72%² may result in high treatment costs. Additionally, care between visits must be provided by the patient, significant others, or home health care providers. For the best clinical and financial outcomes and to improve the quality of life for patients with venous ulcers, healthcare providers must act promptly to initiate appropriate intervention.

Pathophysiology. The venous system of the lower extremities consists of superficial, perforator, and deep veins. When a person walks, the calf muscle contracts, forcing blood in the deep veins to flow proximally toward the heart. During calf muscle relaxation, blood flows from the superficial veins through the perforator veins into the deep veins where muscle contraction forces it toward the heart. One-way valves in a healthy venous system prevent a reverse direction of the blood flow during contraction. However, in patients with incompetent valves, a weak calf muscle pump (from limited activity, paralysis or diminished range of ankle motion), or an occlusion such as a deep vein thrombosis, blood flow follows the direction of gravity, resulting in venous hypertension or high pressure in the venous system.^{1,3}

With increased venous hypertension, the capillaries expand and blood cells leak into the tissue, causing edema and ultimately ulceration. Several theories support the pathogenesis of venous ulceration. One hypothesis is that fibrinogen leaks through the distended blood vessels and is polymerized to fibrin, which forms a cuff around the capillaries and prevents delivery of oxygen and other nutrients to the tissues. Others have suggested that neutrophils become trapped within the capillaries, resulting in ischemia and the release of tissue-damaging toxic metabolites and proteolytic enzymes.^{1,3} Falanga and Eaglstein⁴ suggested that fibrinogen and other macromolecules sequester growth factors, rendering them unavailable for normal epidermal tissue repair.

Characteristics. Characteristics of venous insufficiency include edema, hemosiderosis (brown staining of the lower extremity), lipodermatosclerosis (hardening of the dermis and subcutaneous tissue), and dermatitis. Ulceration commonly occurs in the gaiter area (between the ankle and mid-calf). Ulcers are often granular with irregular margins.¹

The Solution — the Wound Bed Preparation Model using TIME

Although surgical intervention and adjunct therapies are management options, compression therapy is the cornerstone of treatment for venous ulcers.¹ However, compression therapy alone may not be enough. Clinicians must conduct a comprehensive assessment of the patient and address systemic as well as local factors that may affect healing. The TIME principles of Wound Bed Preparation (see Wound Bed Preparation: It's

About TIME, an outsert to the January 2005 issue of WOUNDS) should be considered as a model for comprehensive management of patients with venous ulcers.⁵

Preparing the Wound Bed

Systemic and local factors that can impede wound healing should be identified and corrected or modified and patient concerns (including education and support issues) should be considered and addressed. Attention then should focus on wound assessment in order to secure the tools necessary for appropriate treatment of the venous wound and management of the venous insufficiency. Completing initial assessments and addressing contributory obstacles help ensure that actions taken to prepare the local wound bed will be effective. The acronym TIME (**T**: tissue, non-viable or deficient; **I**: infection or inflammation; **M**: moisture imbalance; **E**: edge of wound, non-advancing or undermined) provides the paradigm for wound bed preparation — in this case, the specifics of venous ulcer management.

Tissue — non-viable or deficient. The venous wound is typically shallow with irregular edges and a ruddy, granular appearance. In venous ulcers, non-viable tissue often presents as shallow, fibrinous slough. The presence of eschar may indicate additional arterial insufficiency. Methods for debriding non-viable tissue include sharp/surgical (the fastest but potentially most painful debridement option for the venous ulcer patient), autolytic (dressing selection based on the amount of exudate), and enzymatic (slower than other methods but accomplished in conjunction with compression therapy). Enzymatic debridement often involves daily dressing changes. However, using papain-urea with sodium copper chlorophyllin (eg, GLADASE C[®]) offers clinicians the choice to leave the enzyme in place for up to 3 days; thus, reducing the frequency of compression bandage changes.

Infection or Inflammation. Venous ulcers often are colonized with bacteria such as *Staphylococcus aureus* and *Pseudomonas*.⁶ When bacteria reach the point of critical colonization — where replicating organisms overwhelm the host's defenses and begin to cause local tissue damage — wound progress may be delayed.⁵ The signs of critical colonization may be subtle and frequently are overlooked. Secondary signs of infection occur more often than classic signs in the chronic wound⁷ (see Table 1) and should prompt the clinician to consider local treatment to reduce bacterial burden; if infection has advanced into soft tissue, systemic antibiotics, in addition to local treatment, should be considered. One example of products used to manage bacterial burden is Acticoat* from Smith & Nephew. Acticoat*, with SILCRYST™ Nanocrystals, is a unique antimicrobial barrier dressing that kills bacteria two to five times faster than other forms of silver. Plus, the antimicrobial barrier of Acticoat 7* remains effective for up to 7 days — convenient for use as a bacterial barrier under compression bandages that may be changed at the same 7-day interval.

Moisture imbalance. Venous ulcers often present with moderate to large amounts of exudate that may lead to periwound maceration and further breakdown. Chronic wound fluid also is known to contribute to the pro-inflammatory state of the wound, further delaying healing.⁵ Exudate resulting from edema may be managed with compression therapy and, when appropriate, absorptive dressings. Clinicians must ensure adequate arterial blood flow to the lower extremities before initiating compression therapy.

Table 1. Classic and Secondary Signs of Infection

Classic Signs of Infection	Secondary Signs of Infection
Advancing erythema	Delayed healing
Fever	Change in color of wound bed
Warmth	Friable granulation tissue
Edema/swelling	Absent or abnormal granulation tissue
Pain	Abnormal odor
Purulence	Serous drainage
	Pain at wound site

Adapted from Gardner SE, Frantz RA, Doebbeling BN. The validity of the clinical signs and symptoms used to identify localized wound infection. Wound Repair and Regeneration 2001;9(3):178-186.

A variety of compression products exist: elastic, inelastic, single-layer and multilayer bandages; compression stockings; and intermittent pneumatic compression (IPC) pumps. Elastic (long-stretch) bandages provide high compression (35 mm Hg to 40 mm Hg at the ankle) during exercise and at rest. Inelastic (short-stretch) bandages, like Una's Boot, provide compression with exercise but, as edema is reduced, offer little compression at rest.¹⁸ According to the International Leg Ulcer Advisory Board,⁹ sustained multilayer high compression bandaging that can maintain compression for up to 7 days improves healing, cost outcomes, and quality of life for patients.

Examples of multilayer, high compression products include ProFore* and ProGuide™. ProFore, a four-layer, high-compression system, was the first of its kind marketed in the US and has been used for years by clinicians who manage patients with venous ulcers. ProGuide™, a relatively new and unique product from Smith & Nephew, features revolutionary Vari-Stretch™ technology that utilizes a breathable outer, latex-free, compression layer marked with indicators to help the user determine a 50% stretch. Three-component, multilayer, high-compression technology provides high compression via an absorbent wound contact layer, an absorbent padding layer, and the Vari-Stretch™ technology outer compression layer to help ensure the right amount of compression. The product can be stretched 30% to 70% and still achieve an appropriate level of compression.

Depending on the amount of exudates, additional dressings for added absorption may be necessary during the initial phase of edema management. The ideal dressing should keep the wound bed moist and the periwound skin dry, absorbing without drying the wound. The Allevyn* family of hydrocellular foam dressings offers an array of products for a variety of exudate levels and wound depths. Allevyn Compression contains a highly absorbent material embedded in a polyurethane matrix that allows it to work well under compression bandages. The dressing confines and holds up to 99% of the exudate to help reduce the risk of maceration.

Edge of wound (epidermal margin): non-advancing or undermined. Wounds may fail to progress even after the clinician has addressed wound etiology, comorbidities, and other underlying factors affecting wound bed preparation. In this case, senescent cells in the wound may not respond to wound healing signals or the hyperproliferation of epidermal cells at the wound margin. In these scenarios, clinicians may consider intervention using biologicals, skin grafts, growth factors, negative pressure, or other adjunct therapies.⁵

Conclusion

Managing patients with venous ulcers is challenging. Successful, cost-effective outcomes depend on early and appropriate interventions. The initial assessment — identifying signs of mixed arterial disease or other factors that can affect healing — is crucial. Clinicians must choose compression wisely — sustained, multilayer, high compression is the gold standard for the management of patients with venous ulcers and, depending on circumstances, may be left in place for up to 7 days. Following the principles of wound bed preparation and re-assessing the patient and the wound frequently, changing care when indicated, are important management principles. By utilizing evidence-based treatment modalities, providing patient education, and continuously assessing progress, clinicians and patients should reap the rewards of improved clinical, financial, and emotional outcomes.





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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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