



# Wound Bed Preparation

## It's About TIME

Pressure ulcers are a costly, often unpredictable challenge for clinicians. Although these wounds have been managed successfully for years, increased awareness among governing bodies and healthcare providers regarding the need for prompt, appropriate pressure ulcer management has sparked a renewed interest in improving care. The National Pressure Ulcer Advisory Panel (NPUAP) is currently revisiting its understanding of pressure ulcer physiology — the results of its considerations potentially will affect the way pressure ulcers are staged and treated. Meanwhile, the Centers for Medicare and Medicaid Services (CMS) has increased scrutiny of pressure ulcer management in long-term care, introducing revisions to its pressure ulcer management guidelines; similar guideline implementation is anticipated for acute care. Pressure ulcers and their possible implication in substandard care litigation are fueling public awareness of the pressure ulcer conundrum. Obviously, diligent healthcare providers need to find the optimal balance between providing quality care and managing expenses.

The TIME model for Wound Bed Preparation proactively addresses the issues raised by the NPUAP and the CMS, as well as the concerns of patients and care providers. The TIME framework takes into account tissue condition, infection, moisture balance, and edge of wound, along with patient-centered concerns such as pain and appropriate product use. The TIME model is an enlightened approach to pressure ulcer management clinicians can confidently incorporate into their wound care regimens.

This is the eleventh in a series of 12 articles on TIME and wound bed preparation.



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## The Problem — How to Provide Quality, Cost-Effective Pressure Ulcer Management

**What is a pressure ulcer?** Pressure ulcers are defined as any lesions caused by unrelieved pressure, resulting in damage of underlying tissue.<sup>1</sup> Pressure ulcer healing time, like that of other often chronic wounds, is unpredictable because a variety of systemic and local factors, diseases, and medications may interfere with the normal phase sequence of wound healing. Subsequently, pressure ulcers often are defined as “wounds that have failed to proceed through an orderly and timely process to produce anatomic and functional integrity or proceeded through the repair process without establishing a sustained anatomic and functional result.”<sup>1</sup>

### Stage I

An observable, pressure-related alteration of intact skin whose indicators, as compared to an adjacent or opposite area in the body, may include changes in one or more of the following:

- skin temperature (warmth or coolness)
- tissue consistency (firm or boggy feel)
- sensation (pain, itching)

The ulcer appears as a defined area of persistent redness in lightly pigmented skin; whereas in darker-toned skin, the ulcer may appear with persistent red, blue, or purple hues.



### Stage II

Partial-thickness skin loss involving epidermis or dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.



### Stage III

Full-thickness skin loss involving damage or necrosis of subcutaneous tissue that may extend down to but not through the underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.



### Stage IV

Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structure (such as tendon or joint capsule).



**Figure 1.** The NPUAP classification for pressure ulcer staging.<sup>2</sup>

**Pressure ulcer classification.** Pressure ulcers are classified/staged according to NPUAP criteria, which incorporates a description of the extent of visible tissue destruction (see Figure 1). However, ulcers may not be able to be staged accurately when necrotic tissue is present. Also, pressure ulcers should not reverse-stage to describe healing owing to the fact that the body is unable to regenerate certain tissues (fat, fascia, muscle), rendering reverse staging an inaccurate parameter for wound healing.

**Incidence and impact.** According to the NPUAP,<sup>3</sup> the incidence rate for pressure ulcers in the US varies from 0.4% to 38% in acute care and from 2.2% to 23.9% in long-term care. Conservative estimates for the cost of pressure ulcer treatment range from \$500 to \$50,000 per pressure ulcer based on the severity of the ulcer and its complications. Regardless of admitting setting, diagnosis, and comorbidities, once a patient acquires a pressure ulcer, the cost of care and length of stay are significantly increased when compared to a patient with the same diagnosis but without a pressure ulcer.<sup>4,5</sup> The overall federal healthcare expenditure for Medicare and Medicaid beneficiaries in 2005 is estimated at \$648 billion; for 2011, projected costs are more than \$1 trillion.<sup>6</sup> Recent guideline revisions (some specific to pressure ulcers) in the long-term care industry aim to temper these costs as well as improve care and will eventually extend into the acute care setting.

**Wound care and lawsuits.** Healthcare providers face a constant threat of litigation related to the development of pressure ulcers. Clinicians know that not all pressure ulcers are preventable; however, general public perception is that all institution-acquired pressure ulcers are an indication of poor quality of care. Hospitals and long-term care facilities face increasing scrutiny as patients and their families have access to more and more information, including federal government statistics on the occurrence of pressure ulcers. The federal government, through the CMS, has established quality indicators intended to guide clinical practice (often referred to as evidence-based best practice), a strategy that demands more accountability on the part of providers.

## The Solution – Incorporating the TIME Model into CMS Guideline-Shaped Care Protocols

The CMS guideline revision provides expectations for long-term care facilities for pressure ulcer management. Although designed to assist surveyors in long-term care, the guidelines may be helpful for clinicians managing pressure ulcers within various healthcare settings. It is especially important for the staff of wound centers and other settings where residents of long-term care facilities may be treated, to understand these guidelines and the standards to which long-term care providers are accountable.

The TIME model for Wound Bed Preparation offers a viable approach for proactively addressing the issues raised within the revisions of the CMS guidance document, including debridement, infection related to pressure ulcers, dressings and treatment, and pain.<sup>7</sup>

**Debridement.** Tissue – nonviable or deficient (the T in the TIME acronym), reminds the clinician to assess for and remove nonviable tissue within the pressure ulcer.<sup>8</sup> Debriding nonviable tissue allows the clinician to better assess and stage the pressure ulcer. Additionally, removing nonviable tissue reduces the risk of sepsis from bacteria found in the wound. Debridement options (discussed in detail in Part 4 of this series) include sharp/surgical, mechanical, autolytic, enzymatic, and biological (larvae). Although each method

**Table 1. Classic and Secondary Signs of Infection**

Classic Signs of Infection	Secondary Signs of Infection
Erythema	Delayed healing
Fever	Change in color of the wound bed
Warmth	Friable granulation (bleeds easily)
Edema/swelling	Absent or abnormal granulation
Purulent exudate	Increased odor
Pain	Increased serous exudate
	Increased pain

has its advantages and disadvantages, the CMS guideline states, “Wet-to-dry gauze dressings or irrigation (with chemical solutions) may be appropriate in limited circumstances, but repeated use may damage healthy granulation tissue in healing ulcers and may lead to excessive bleeding and increased resident pain.”<sup>9</sup>

**Infection related to pressure ulcers.** The clinician should assess for and manage wound infection and inflammation (the I in TIME). Infection in pressure ulcers can delay healing and, as with other chronic wounds, may not be easy to identify. (Infection is addressed in detail in Part 6 of this series). Stage II, Stage III, and Stage IV pressure ulcers may be colonized but not infected with bacteria — critical colonization, the point at which bacteria have reached a level that stimulates a host response, affects wound healing. The number of bacteria and the balance between bacterial quantity, virulence, and the host’s ability to respond are all cause for concern.<sup>8</sup> The CMS guideline suggests that wound infection may be identified by clinical observation or from a quantitative wound culture containing  $10^5$  or more microbes per gram of tissue. A superficial swab culture may ascertain the presence of bacteria but it is not a reliable method for confirming infection.<sup>7</sup>

Assessing classic clinical signs and symptoms is not always a reliable means to determine the presence of infection. In chronic wounds such as pressure ulcers, secondary signs and symptoms of infection, often overlooked even by experienced clinicians, are more common than the classic signs (see Table 1).<sup>9</sup> Learning to recognize the secondary signs of infection in pressure ulcers allows for quicker bacterial burden management.

**Bacterial burden management.** Bacterial burden management in pressure ulcers may be achieved with thorough cleansing, debridement, and the use of antimicrobial barrier dressings such as nanocrystalline silver or cadexomer iodine. These dressings have demonstrated effectiveness against clinically relevant bacteria, including methicillin-resistant *Staphylococcus aureus* (MRSA), without causing toxicity to the wound. Systemic antibiotics play an important role in treating patients with infection that has moved beyond the wound margins and caused cellulitis or sepsis but they may not be necessary when the bacterial burden is confined to the wound. Topical antimicrobial agents should be continued as part of wound care while patients are treated with systemic antibiotics; systemic antibiotics may not be delivered to the wound bed.

**Dressings.** Moisture imbalance (the M in TIME) also must be addressed within the pressure ulcer (see Part 5 of this series). Wound care experts commonly use dressings that promote a moist wound bed. However, excessive amounts of exudate, common in the chronic wound, also can cause complications (eg, maceration).

Plus, chronic wound fluid is biochemically different from acute wound fluid and has been shown to delay wound healing. The CMS guideline states that Stage II, Stage III, and Stage IV pressure ulcers should be covered, dressing selection should be based upon the wound characteristics, and no single dressing is appropriate for all pressure ulcers.

**Edge of wound.** If a pressure ulcer shows no evidence of healing after 2 to 4 weeks, the patient/resident and the pressure ulcer should be reassessed. The clinician also should review the components of the TIME model. If the T, I, and M have been addressed and no other factors that delay healing are evident, the clinician may consider the E in TIME — edge of wound non-advancing or undermined. Adjunct therapies to stimulate cellular migration and wound closure may need to be implemented appropriately.

**Pain.** Pain associated with pressure ulcers may be addressed with analgesia and by eliminating the cause,<sup>9</sup> which can include selecting a dressing capable of eliminating pain at the wound site and not causing pain when removed.<sup>10</sup>

## Conclusion

New care guidelines stress the importance of providing pressure ulcer treatment that is in accordance with patient needs, overall goals of care, and recognized standards of practice. The TIME model for Wound Bed Preparation serves as an excellent platform for staff education and for developing interventions for pressure ulcer management. Clinicians should rely on manufacturers such as Smith & Nephew for products and information to address the components of wound bed preparation.





## References

- Lazarus GS, Cooper DM, Knighton DR, et al. Definitions and guidelines for assessment of wounds and evaluation of healing. *Arch Dermatol*. 1994;130:489–493.
- Panel for the Prediction and Prevention of Pressure Ulcers in Adults. *Pressure Ulcers in Adults: Prediction and Prevention. Clinical Practice Guideline, Number 3*. AHRQ Publication No. 92-0047. Rockville, Md.: Agency for Health Care Research and Quality, Public Health Service, US Department of Health and Human Services. May 1992.
- National Pressure Ulcer Advisory Monograph. Pressure ulcers in America; prevalence, incidence, and implications for the future. *Adv Skin Wound Care*. 2001;14:208–215.
- Lyder C, Shannon R, Empleo-Frazier O, McGehee D, White C. A comprehensive program to prevent pressure ulcers in long-term care: cost and outcomes. *Ostomy Wound Manage*. 2002;48(4):53–60.
- Allman R, Goode P, Burst N, Bartolucci A, Thomas D. Pressure ulcers, hospital complications, and disease severity: impact on hospital costs and length of stay. *Adv Wound Care*. 1999;12(1):22–30.
- Department of Health and Human Services, Centers for Medicare and Medicaid Services, Office of the Actuary. National Health Expenditures Projections: 2004 – 2014. Available at: <http://www.cms.hhs.gov/statistics/nhe/projections-2004/proj2004.pdf>. Accessed September 1, 2005.
- Guidance to Surveyors for Long Term Care Facilities. Centers for Medicare and Medicaid Services (CMS) Manual System. Pub.100-07 State Operations. Provider Certification. Transmittal 4. November, 2004.
- Schultz G, Sibbald RG, Falanga V, et al. Wound bed preparation: a systematic approach to wound management. *Wound Rep Regen*. 2003;11(1):1–28.
- Gardner SE, Frantz RA, Doebbeling BN. The validity of the clinical signs and symptoms used to identify localized wound infection. *Wound Rep Regen*. 2001;9(3):178–186.
- Baranoski S, Ayello A. Wound treatment options. In: Baranoski S, Ayello A (eds.). *Wound Care Essentials: Practice Principles*. Philadelphia, Pa.: Lippincott, Williams & Wilkins;2004:134.

# WOUND BED PREPARATION

Removing the barriers

## TIME<sup>†</sup> - Principles of Wound Bed Preparation

Clinical Observations	Proposed Pathophysiology	WBP Clinical Actions	Effect of WBP Actions	Clinical Outcome	SOLUTIONS
<b>T</b> 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	<b>GLADASE<sup>®</sup></b> Papain-Urea Debriding Ointment**  <b>GLADASE<sup>®</sup> C</b> Debriding, Deodorizing and Healing Ointment**
<b>I</b> 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	 <b>ACTICOAT<sup>®</sup></b> (with SILCRYST Nanocrystals)†
<b>M</b> 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	 <b>ALLEVYN<sup>®</sup></b>
<b>E</b> 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	 <b>DERMAGRAFT<sup>®</sup></b> Human Fibroblast-Derived Dermal Substitute**

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