



A Basic Approach to Wound Care

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Residents are faced with treating wounds all the time. In fact, it is a part of our job to make wounds either from biopsies or surgical excisions. We also have the sometimes-difficult task of promoting the healing of chronic wounds. Because of the aging population and the rise in chronic conditions such as diabetes mellitus and obesity, the global wound care market is projected to reach \$20.3 billion by 2015.¹ There are more than 6500 types of wound dressings in 30 categories,² and choosing a product can be a confusing task. With wounds playing such a large role in our practice of dermatology, it is important to have a clear understanding of why we treat wounds the way we do and how to treat wounds that are difficult to heal. To simplify the process, residents should ask 4 questions when treating a wound^{3,4}: (1) Is the wound acute or chronic, and is there a treatable underlying cause? (2) Does the wound need debridement? (3) Is the wound infected? (4) Is the wound dry or exudative?

Before approaching these 4 questions, I should examine why wound dressings are used. Acute wounds treated with occlusive dressings heal 40% to 50% more quickly than wounds that are exposed to air.⁵⁻⁷ Occlusion promotes keratinocyte migration, creates a hypoxic environment that stimulates angiogenesis,⁸ and allows retained water and proteolytic enzymes to facilitate wound debridement.⁴ Covering a wound also protects the wound bed from trauma and contamination with additional bacteria and/or foreign materials. Although occluded wounds have a higher bacterial load, studies show that they still heal faster.⁹⁻¹¹ By minimizing the loss of fluid and heat from the wound, occlusion creates a moist environment

that reduces pain, desiccation, epidermal necrosis, and eschar formation.^{12,13} Dressings also absorb wound drainage. Although a wound should be kept moist, it should not be wet and macerated.

Is the wound acute or chronic?

Acute wounds include surgical and traumatic wounds. The reparative process of acute wounds has been well studied and consists of 4 predictable stages: (1) coagulation; (2) inflammation; (3) granulation tissue formation (cell proliferation and repair of the matrix); and (4) remodeling (epithelialization and scar tissue formation).^{3,4} Fluid in acute wounds contains growth factors, neutrophil-derived autolytic enzymes (eg, elastase, collagenase, myeloperoxidase), and protease inhibitors that prevent damage to the intact tissue at the wound edge.³ Other types of acute wounds include those that are secondary to vasculitis, Stevens-Johnson syndrome, and pemphigus vulgaris. For all acute wounds it is imperative to address the primary cause of the wound.

Chronic wounds are defined as wounds that show no tendency to heal after 3 months of appropriate treatment or are not fully healed by 12 months and rarely are seen in healthy individuals.¹⁴ In the United States, chronic wounds affect 6.5 million patients¹⁵ and include venous stasis, diabetic foot ulcers, decubitus ulcers, and ulcers that develop from arterial disease. Compared to acute wounds, chronic wound progression is less predictable. It is largely accepted that the wound-healing process is prolonged due to an aberration in 1 or more of the stages of wound healing. For example, in patients with diabetes mellitus, venous and diabetic foot ulcers are thought to be stuck in the inflammatory and proliferative phases, respectively.^{3,16} Compared to acute wound fluid, which promotes wound healing, chronic wound fluid slows healing and prevents proliferation of keratinocytes, fibroblasts, and endothelial cells.¹⁷ Furthermore,

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fibroblasts derived from chronic wounds show poorer response to growth factors than fibroblasts derived from acute wounds.¹⁸ Similar to acute wounds, one also should investigate and address the source of a chronic wound. For example, in cases of venous insufficiency local edema must be reduced with compression therapy; for diabetic wounds, orthotics can be used to reduce pressure on the sole of the foot.

Does the wound need debridement?

Using knowledge of acute wound healing, physicians can utilize techniques to imitate a healthy wound as much as possible, one being debridement. In many wounds, especially chronic wounds, necrotic or hyperkeratotic tissue can accumulate and prevent appropriate healing. Debridement is the act of removing this tissue to expose healthy granulation tissue. For example, in diabetic foot ulcers hyperkeratosis can be addressed with sharp debridement. Debridement also reduces wound contamination. There are several forms of debridement: autolytic, surgical, enzymatic, mechanical, and biological therapy (eg, maggots). Debridement can be painful. If the wound is small enough, topical or local injection anesthetics can reduce pain to allow the physician to debride more aggressively.

Is the wound infected?

Infection can cause a persistent inflammatory response that delays wound healing.³ Oral antibiotics may be needed, especially for deep wounds that have surrounding erythema, warmth, and are increasing size. All chronic wounds are colonized with bacteria, and it can be difficult to differentiate colonization from infection. Colonization is the presence of replicating bacteria with no evidence of tissue damage.^{3,19} Critical colonization describes a scenario in which the bacterial load is not high enough to show classic signs of infection but does delay wound healing. Studies have shown correlation between wounds that had greater than 100,000 to 1,000,000 organisms per gram of tissue and signs of poor wound healing.^{20,21} In more superficial wounds that do not have surrounding erythema or classic symptoms of infection, bright red, friable granulation tissue; exuberant granulation; increased exudates; new areas of sloughing; pain; and odor also can be indicative of underlying infection.^{3,20,22} Slow healing or failure to heal also can indicate infection. These wounds may benefit from topical antiseptic agents such as silver dressing and cadexomer iodine (hydrogen peroxide and povidone-iodine can be cytotoxic).^{19,23} Topical antibiotics such as mupirocin and gentamicin may be helpful. Keep in mind that neomycin and bacitracin may cause allergic dermatitis.¹⁹

Is the wound dry or exudative?

For acute wounds, occlusive dressings optimize healing, primarily because of the wound's ability to retain healthy fluid. However, the biochemical contents of chronic wound exudate differ from acute wounds and can be detrimental to healing. Thus after addressing the root cause of the wound and preparing the wound base, one must choose an appropriate occlusive dressing, balancing maintenance of wound moisture to encourage cell migration and avoidance of eschar formation without oversaturating the wound. Because the characteristics of wounds change over time and no dressing can perfectly accommodate the entire spectrum of wound types, a slew of products have been developed. One of the most practical ways to choose a dressing is by evaluating the amount of exudate a wound produces.

Highly Exudative Wounds—For wounds that are highly exudative, choose foam or hydrofiber dressings. Both of these materials can be kept on the wound for a week. Foam dressings, which are made of polyurethane,¹³ are highly absorbent and allow for gas permeability while still maintaining a moist environment; they also can be cut into different shapes.³ Foams do not harm viable tissue because they do not stick to the tissue. They also can be used in conjunction with compression stockings or bandages.¹³ Hydrofibers have good tensile strength,³ and they interact with wound exudates to form a soft gel. They can be used for bleeding wounds, as hydrofibers are 3 times more absorbent than calcium alginates and can hold up to 30 times their weight in fluid. The risk for maceration is lower because of vertical fluid absorption properties.²⁴

Moderately High Exudative Wounds—Calcium alginates also can be used to dress exudative wounds as well as deep wounds or cavities (eg, rope or ribbon forms).⁴ Calcium alginates also have hemostatic qualities.²⁵ They are biodegradable, hydrophilic, non-adherent, and absorbent.²⁵ Avoid calcium alginates in dry or mildly exudative wounds. Alginates can hold up to 20 times their weight in fluid.²⁴

Mild to Moderately Exudative Wounds—Hydrocolloid dressings were among the first occlusive dressings used.²⁶ They consist of a mixture of adhesive, absorbent, and elastomeric ingredients, making them useful in dressing mild to moderately exudative wounds.¹³ Carboxymethyl cellulose is a common absorptive ingredient. All hydrocolloid dressings have a top layer of film that makes them waterproof and impermeable to bacterial contaminants, allowing them to be left on the wound for several days.¹³

Dry Sloughy Wounds—Hydrogels often consist of 70% to 90% water and rehydrate wounds by donating water to the dry tissue.^{3,13} They are available in the

form of sheets or gels. They do not retain moisture well and cannot handle heavy exudate.¹³ Hydrogels also stimulate autolytic debridement and therefore promote granulation tissue formation.⁴ Hydrogels in the form of gel dressings require a secondary dressing and should be changed every 3 days for necrotic wounds and every 7 days for granulating wounds. Hydrogels are minimally anti-infective.³

Superficial Wounds With Minimal Exudate—Films are polyurethane-based dressings that can be used on shallow wounds or abrasions. They are transparent, thin, and retain moisture.¹³ Films are poor choices for exudative wounds because they have no absorptive capacity.

Final Thoughts

It is important for residents to develop a familiarity with the main categories of wound dressings and practice a systematic approach to treating wounds. Difficult cases will exist, but approaching wounds step-by-step with a basic algorithm can make the process less daunting and extremely rewarding when that impossible wound finally closes.

REFERENCES

1. Global wound care products market to reach \$20.3 billion by 2015, according to new report by Global Industry Analysts, Inc [press release]. San Jose, CA: Global Industry Analysts; September 6, 2010.
2. Metzger S. Clinical and financial advantages of moist wound management. *Home Health Nurse*. 2004;22:586-590.
3. Schultz GS, Sibbald RG, Falanga V, et al. Wound bed preparation: a systematic approach to wound management. *Wound Repair Regen*. 2003;11(suppl 1):S1-S28.
4. Robinson JK, Hanke CW, Siegal D, Fratila A, eds. *Surgery of the Skin: Procedural Dermatology*. 2nd ed. London, England: Mosby; 2010.
5. Winter GD. Formation of the scab and the rate of epithelialization of superficial wounds in the skin of the young domestic pig. *Nature*. 1962;193:293-294.
6. Hinman CD, Maibach H. Effect of air exposure and occlusion on experimental human skin wounds. *Nature*. 1963;200:377-378.
7. Hasan A, Murata H, Falabella A, et al. Dermal fibroblasts from venous ulcers are unresponsive to the action of transforming growth factor-beta 1. *J Dermatol Sci*. 1997;16:59-66.
8. Agren MS, Karlsmark T, Hansen JB, et al. Occlusion versus air exposure on full-thickness biopsy wounds. *J Wound Care*. 2001;10:301-304.
9. Mertz PM, Eaglstein WH. The effect of a semioclusive dressing on the microbial population in superficial wounds. *Arch Surg*. 1984;119:287-289.
10. Mertz PM, Marshall DA, Eaglstein WH. Occlusive wound dressings to prevent bacterial invasion and wound infection. *J Am Acad Dermatol*. 1985;12:662-668.
11. Helfman T, Ovington L, Falanga V. Occlusive dressings and wound healing. *Clin Dermatol*. 1994;12:121-127.
12. Kannon GA, Garrett AB. Moist wound healing with occlusive dressings. a clinical review. *Dermatol Surg*. 1995;21:583-590.
13. Eaglstein WH. Moist wound healing with occlusive dressings: a clinical focus. *Dermatol Surg*. 2001;27:175-181.
14. Kahle B, Hermanns HJ, Gallenkemper G. Evidence-based treatment of chronic leg ulcers [published online ahead of print April 8, 2011]. *Dtsch Arztebl Int*. 2011;108:231-237.
15. Sen CK, Gordillo GM, Roy S, et al. Human skin wounds: a major and snowballing threat to public health and the economy. *Wound Repair Regen*. 2009;17:763-771.
16. Falanga V. Classifications for wound bed preparation and stimulation of chronic wounds. *Wound Repair Regen*. 2000;8:347-352.
17. Shah JM, Omar E, Pai DR, et al. Cellular events and biomarkers of wound healing. *Indian J Plast Surg*. 2012;45:220-228.
18. Mendez MV, Stanley A, Park HY, et al. Fibroblasts cultured from venous ulcers display cellular characteristics of senescence. *J Vasc Surg*. 1998;28:876-883.
19. Hafner A, Sprecher E. Ulcers. In: Bologna JL, Jorizzo JL, Schaffer JV, eds. *Dermatology*. 3rd ed. New York, NY: Elsevier Saunders; 2012:1729-1746.
20. Gardner SE, Frantz RA, Doebbeling BN. The validity of the clinical signs and symptoms used to identify localized chronic wound infection. *Wound Repair Regen*. 2001;9:178-186.
21. Panuncialman J, Falanga V. The science of wound bed preparation. *Surg Clin North Am*. 2009;89:611-626.
22. Cutting KF, White R. Defined and refined: criteria for identifying wound infection revisited. *Br J Community Nurs*. 2004;9:S6-S15.
23. Sibbald RG, Browne AC, Coutts P, et al. Screening evaluation of an ionized nanocrystalline silver dressing in chronic wound care. *Ostomy Wound Manage*. 2001;47:38-43.
24. Gibbs KA. Absorptive dressings: alginates and hydrofibers. In: Sen CK, ed. *Advances in Wound Care*. Vol 1. New Rochelle, NY: Mary Ann Liebert; 2010:142-147.
25. O'Meara S, Martyn-St James M. Alginate dressings for venous leg ulcers. *Cochrane Database Syst Rev*. 2013;4:CD010182.
26. Baum ME. Flexible decubitus treatment. *Nurs Care*. 1976;9:24-25.