

Wound Dressing Selection for Pressure Ulcers

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Every individual and every wound requires a holistic, individualized approach. Pressure ulcers present healthcare providers with a myriad of factors to consider in the assessment, treatment and dressing selection to attain a path to wound healing. This paper provides clinicians with an overview of the causes and characteristics of pressure-related wounds, with a practical approach to the assessment of individuals with pressure ulcers. A guide to the form and function of wound dressings by composition is offered to aid clinicians in appropriate dressing selection to match the characteristics of the wound for optimal healing outcomes.

Pressure ulcers, also known as pressure sores, bedsores or decubitus ulcers, continue to be a significant problem across the continuum of healthcare settings. According to results of nine international pressure ulcer prevalence surveys from 1989 to 2005, involving a total of 447,930 patients¹, pressure ulcer prevalence rates in acute care, long-term acute care, and long-term care facilities ranged from 9.2% in 1989 to 10% in 2004. The highest prevalence was estimated at 27.3% in long-term acute care. In Canada, the prevalence rates are estimated between 3.7% to 14% from 2010-2012 with the highest prevalence rate in chronic care settings.²

The majority of pressure ulcers were classified as stage I and II [see sidebar]. The burden of pressure ulcers as a chronic disease is far-reaching and onerous; the average cost associated with the treatment of stage IV pressure ulcers and related complications in the US was \$129,248 for one single episode of hospitalization.⁴ Living with pressure ulcers can be devastating, leading to social isolation, loss of independence, depression, anxiety, pain and financial encumbrance.⁵

CAUSES OF PRESSURE ULCERS

Pressure ulcers, as the name implies, are primarily caused by excessive or prolonged pressure to the skin, distorting and compressing underlying soft tissues, especially over bony prominences.^{3,6} Shear contributes to deep tissue injury and undermining as one layer of tissue slides over the deeper structure. The damaging effect of pressure and shear is often exacerbated by friction that is described as the resistance to movement created between two surfaces such as the superficial layers of skin and the adjoining support surface.⁷

Pressure Ulcer Staging Review

Stage I pressure ulcers are characterized by non-blanchable erythema of intact skin that may be coupled with alterations in skin temperature and tissue consistency.³

Stage II pressure ulcers are superficial lesions involving the erosion of epidermis with epidermal base or an ulcer with loss of epidermis and a dermal base.

Full-thickness tissue damage may extend to subcutaneous tissue as in **stage III** pressure ulcers and to deeper supporting structures such as muscle, fascia, joint capsule and bone that are classified as **stage IV** pressure ulcers. Deep tissue injuries (DTI) have the appearance of a purple or maroon bruise under intact skin that resembles and are often mistaken as a stage I pressure ulcer.³

Skin that is exposed to moisture due to incontinence or heavy perspiration has increased vulnerability to breakdown. Moisture not only potentiates friction and shear but also compromises normal barrier functions by causing the skin to swell, weakening intercellular bonds in the epidermal layer and damaging skin cells in the presence of chemical irritants and digestive enzymes. Increasing attention is being drawn to early recognition and management of moisture-associated skin damage, or MASD, which is evidenced by diffuse erythematous irritation and inflammation.⁸

Overall, tissue tolerance or the ability of the tissue to offset the damaging effect of pressure, shear, friction, and moisture will determine the extent of tissue injury and disruption to blood flow.⁹ Following persistent pressure injury is a cascade of physiological events characterized by ischemia, anaerobic metabolism, accumulation of toxic metabolites, acidosis, increased cell membrane permeability, cellular edema, cell death and tissue necrosis, which culminates in a pressure ulcer. Suffice to say, prudent risk assessment and early intervention is of utmost importance to preventing pressure ulcers.

HOW TO ASSESS PRESSURE ULCERS

Center to the mainstay treatment of pressure ulcers is pressure redistribution.¹⁰ A comprehensive assessment must be conducted to evaluate the source of pressure, shear, friction, and any underlying conditions that impede healing including: poor nutritional intake, low body mass index (<18.5), hypoproteinemia, low systolic blood pressure, anemia, poor tissue perfusion, contractures and bony prominences, neuropathy, infection, and the presence of a foreign body (e.g., prosthetic joint, retained suture).¹¹

Over the course of a pressure ulcer's existence, several tissue types noted in the wound characteristic assessment can be identified. In order to properly evaluate and monitor the status of the pressure ulcer, it is important for the clinician to be able to determine the tissue type that is present. The following represent the most commonly identified tissue types seen in pressure ulcers, and also in other open wounds:

EPITHELIUM- Epithelial tissue often appears lighter than surrounding tissue (i.e., light pink in color). Epithelialization occurs when the epidermis regenerates over a wound surface. Basal keratinocytes travel from the wound edges, where they multiply until they meet in the middle. The basal lamina is a scaffolding secreted by the epithelial cells as they travel outwards from the wound edges. When a new epithelial layer is created, this new layer is only a few cell layers in thickness and appears translucent. It is extremely vulnerable to damage from friction, shear, and pressure. It generally takes 2 to 3 weeks for the new cells to become keratinized (i.e., waterproof).

GRANULATION TISSUE- The formation of granulation tissue is thought to be an intermediate step in the healing process of full-thickness wounds. Healthy granulation tissue is shiny red and granular in appearance. The process of granulation provides the early scaffolding necessary to promote healing from the edges of the wound. Granulation tissue is very fragile and especially prone to injury by mechanical forces, including dry/adherent dressings, pressure, high-intensity irrigation of the wound, and overzealous wound packing. Wound healing may stall in the granulation phase when nutrients are inadequate, infection is present, or blood flow is impaired.

SLOUGH- Slough is indicative of full-thickness stage III pressure ulcers or stage IV pressure ulcers. Slough can easily be confused with normal anatomical tissues such as tendons or ligaments because of their frequently yellowish coloration. Slough can be identified as a stringy mass that may or may not be firmly attached to surrounding tissue. Slough can range in color from white (scant bacterial colonization) to yellow or green (larger bacterial counts) to brown (hemoglobin is present). When a large amount of slough is present and obscures the wound bed, the wound is unstageable.

ESCHAR- Eschar is composed of dead granulation tissue, muscle, fat, tendon, or skin. The term stable eschar is used to describe leathery, dry, hard eschar tissue, such as the eschar that commonly forms on the heels or other bony prominences of the lower leg of patients with ischemic limbs. The term unstable eschar is used to describe tissue that is undergoing a softening process caused by proteolytic enzyme production from bacteria present in the tissues. The affected area is described as spongy, boggy or slimy. The presence of unstable eschar raises the risk of infection as evidenced by pain, redness, purulent discharge, warmth, and edema. Wet gangrene should be ruled out when fluctuance, crepitation or purulent drainage is present. Sometimes autolytic debridement or topical enzymes are used on eschar tissue, and this deliberate softening should not be confused with unstable eschar. To prepare a wound bed for healing, devitalized and damaged tissue such as firm eschar or sloughy materials that promote bacteria growth should be removed or debrided.

HOW TO SELECT THE RIGHT WOUND DRESSING FOR INDIVIDUALS WITH PRESSURE ULCERS

For wounds that have the potential to heal, moisture balance is essential for all phases of wound repair.¹² A moist wound environment is crucial to granulation and to promoting autolytic debridement through the activities of phagocytic cells and endogenous enzymes. However, excessive wound exudate accompanied with high levels of oxidative enzymes, cytokines, leukocytes, and proteases (e.g., metalloproteinases [MMPs]) due to protracted inflammatory response can be deleterious to the healing process.¹² The main goal of moist wound healing (MWH) is to maintain optimal levels of moisture in the wound bed through the use of appropriate dressings to avoid wound dessication and yet absorb excess exudate. To contain and remove excess exudate from the wound, a plethora of absorbent dressings have been developed.

In addition to moisture balance, the location of the wound should be taken into great consideration. The sacrum, coccyx, and heels are the most common areas where pressure ulcers develop, but the contour and high shear forces make these areas the most challenging areas to treat. Dressings tend to slip, bunch up, or get soiled (especially wounds that are in close proximity to the anal orifice). The heel has a pointed shape with a limited surface area of contact to redistribute pressure and when this is combined with low subcutaneous tissue volume, this area is prone to pressure damage. The heel tissue is enveloped within the fibrous septa that allows pressure to build up easily and occlude vascular supply.¹³

WOUND DRESSINGS BY CATEGORY

Thorough assessment of the individual and pressure ulcer, paired with and a strong knowledge of wound dressing technologies will ensure the appropriate choice is made to meet the requirements of the wound. The major categories of dressings are foams, alginates, hydrofibers, hydrogels, hydrocolloids, and films.¹⁴ The choice of which type of advanced dressing to use is a clinical decision based largely on:

- Dressing features such as conformability, cushioning, ease of removal, and ability to minimize pain at the wound site and during dressing changes, all of which contribute to patient comfort;
- Performance measures, such as ease of application, absorbency, wear-time, barrier properties, and the ability to protect the periwound area and control wound odor;
- And cost.

FOAM DRESSINGS

Foam dressings are designed to wick up a large volume of exudate with minimal lateral movement to prevent periwound skin maceration. The fluid handling capacity of various foams can be affected by the polyurethane film backing and its ability to transfer moisture vapor out of the dressing but form a barrier to bacterial contamination. Depending on the level of wound exudate, foams have a wear time of one to seven days.

Increasing attention has been drawn to the role of soft silicone foam composite dressings to prevent pressure ulcers. It is hypothesized that the multi-layer material inside the foam dressing will help minimize shear as the dressing materials slide against each other, reduce friction at the interface between the skin and the support surface, and protect the skin from mechanical damage. The other potential advantage is the ability of a breathable foam dressing to minimize heat and moisture accumulation that tends to render the skin more vulnerable to pressure damage.

Are foam dressings appropriate for use in the prevention of pressure ulcers? There are a number of new studies indicating that incidence of pressure ulcers is reduced by the introduction of silicone foam composite dressings in critical care settings.¹⁵⁻¹⁸ However, more evidence is needed to confirm the efficacy of using a dressing to prevent pressure ulcers and it appears, at least at this point in time, that the evidence may not be directly transferable between products because of variations in design and components.

ALGINATE DRESSINGS

Alginate dressings are also capable of handling copious exudate while the gelling effect of these materials will keep the wound base moist. Alginates are derived from brown seaweed or kelp. Depending on the species and the origin of the calcium alginate (leaf or stem), they may have more gelling (high manuronic acid concentration) or a higher fiber strength (high galuronic acid concentration). The calcium component of the alginate may also trigger the coagulation cascade to facilitate hemostasis.

HYDROFIBER DRESSINGS

Hydrofiber dressings consist of carboxymethyl cellulose and have a water-repelling (hydrophobic) component (methylcellulose) that gives this dressing its tensile strength and a water-absorbing (hydrophilic) component (carboxy) that acts as a fluid lock. As the dressing absorbs fluid, the hydrofiber is converted into a gel consistency.

HYDROGEL DRESSINGS

Hydrogel dressings are usually indicated for dry wounds. The major ingredient of hydrogels is water (70 to 90%) that donates moisture into the wound base. The backbone for a hydrogel may be a hydrocolloid, propylene glycol, sodium salt, or other substance.

HYDROCOLLOID DRESSINGS

Hydrocolloid dressings consist of a backing (often a film or polyurethane) with carboxymethyl cellulose, water absorptive components (such as gelatin and pectin) and an adhesive. Hydrocolloids are able to handle low to moderate amounts of fluid.

FILM DRESSINGS

Film dressings are semi-occlusive with varying degrees of permeability, referred to as the Moisture Vapor Transmission Rate (MVTR). The permeability of these dressings allows water molecules to pass through them and evaporate into the ambient environment at a variable rate depending on the MVTR of the dressing.

Thorough assessment of the pressure ulcer and its characteristics, combined with an understanding of the properties and performance measures of the different types of wound dressings, will ensure the appropriate selection of a dressing for optimal healing. When healing is not the realistic objective, moist wound healing is contraindicated; instead, conservative debridement without cutting into living tissue, bacterial reduction, and moisture reduction should be considered.

WHEN ANTIMICROBIAL DRESSINGS SHOULD BE USED FOR PRESSURE ULCERS

All chronic wounds, such as pressure ulcers, are colonized by bacteria.¹¹ If bacteria are allowed to proliferate beyond a critical threshold, local tissue damage can lead to delayed healing. Many modern wound dressings contain active antimicrobial ingredients (e.g., silver, iodine, honey, polyhexamethylene biguanide) that can reduce bacterial damage in the surface compartment. While there is no evidence that prophylactic use of antimicrobial dressings can prevent infection, early detection and treatment of localized wound infection may prevent deep compartment infection and minimize the use of systemic antibiotics.¹¹

Based on literature review and previous studies, the UPPER and LOWER wound infection checklist was developed incorporating a total of 10 signs and symptoms (Table 1) that are associated with critical colonization (upper compartment) and deep infection (lower compartment).¹⁹ The list of criteria to evaluate wound infection has been validated in previous studies.^{19,20} There is no one individual sign or symptom that will accurately confirm the diagnosis of wound infection, but a combination of two or three of these possible signs should be sought for the diagnosis in each level shown in the table.

Common Topical Antimicrobial Agents

Common Topical Antimicrobial Agents Include iodine, polyhexamethylene biguanide (PHMB), topical silver dressings, dressings with fatty acids and honey-based dressings. For more information on Antimicrobial Dressings, [click here](#).

TABLE 1. UPPER AND LOWER WOUND INFECTION CHECKLIST

U	UNHEALTHY TISSUE	L	LARGER
P	PAIN	O	OSSEOUS TISSUE
P	POOR HEALING	W	WARMTH
E	EXUDATE	E	EDEMA
R	REEK	R	REDNESS

To reduce superficial and localized bacterial burden in chronic wounds, topical antimicrobial dressings have been demonstrated to be effective.¹¹ Once bacterial burden has been rectified based on clinical assessment, antimicrobial dressings should be transitioned to moisture balance dressings to prevent potential cytotoxicity. Systemic antibiotics should be considered if there is any concern about deep tissue infection or osteomyelitis (especially in patients with diabetes and immunosuppression).

ALTERNATIVE WOUND THERAPIES

For certain types of acute wounds and chronic wounds that exhibit delayed healing, there may be a need to consider an alternative therapy to local dressings in order to achieve more cost-effective wound healing. The number of related dressings for negative pressure wound therapy (NPWT) is burgeoning. During NPWT, subatmospheric pressure or suction is applied across the wound bed. Specialized dressings (e.g., interfaces), which are cut to fill the wound bed, have historically been made of open-cell foam or gauze that remain porous during suction and equalize the pressure that is applied across the wound surface. The healing mechanism of NPWT is based on the assumption that uniform negative pressure exerts three-dimensional mechanical stress on the wound bed. This stress is then transmitted down to cellular and cytoskeletal levels, resulting in the activation of signal transduction pathways, which trigger cell recruitment, angiogenesis, growth factor expression, and cell proliferation.²¹ The growth of granulation tissue is stimulated as a result and wound healing proceeds at a faster rate than with the application of moist wound healing dressings.

While the focus of this discussion has been wound dressing selection, it is crucial to remember that treatment of pressure ulcers requires comprehensive assessment and interprofessional interventions. Moist wound healing (MWH) therapies and other treatment modalities have been successfully utilized in the management of wounds such as pressure ulcers. However, it is important to understand where these advanced therapies fit within the spectrum of wound management, taking into consideration the cost-effectiveness of care and optimal patient outcomes.

REFERENCES

1. Vangilder C, Macfarlane GD, Meyer S. Results of nine international pressure ulcer prevalence surveys: 1989 to 2005. *Ostomy Wound Manage* 2008 Feb;54(2):40-54.
2. Woo KY. Emerging Health System Use Analysis Using HOBIC Data: pressure ulcers. 2013 February 7th 2nd Annual HOBIC Symposium on February 7th at The Old Mill Inn & Spa.
3. Black J, Baharestani MM, Cuddigan J, Dorner B, Edsberg L, Langemo D, et al. National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system. *Adv Skin Wound Care* 2007 May;20(5):269-274.
4. Brem H, Maggi J, Nierman D, Rolnitzky L, Bell D, Rennett R, et al. High cost of stage IV pressure ulcers. *Am J Surg* 2010 Oct;200(4):473-477.
5. Gorecki C, Brown JM, Nelson EA, Briggs M, Schoonhoven L, Dealey C, et al. Impact of pressure ulcers on quality of life in older patients: a systematic review. *J Am Geriatr Soc* 2009 Jul;57(7):1175-1183.
6. Registered Nurses' Association of Ontario. Risk assessment and prevention of pressure ulcers (Revised) Nursing Best Practice Guideline 2005.
7. Sibbald RG, Krasner DL, Woo KY. Pressure ulcer staging revisited: superficial skin changes & Deep Pressure Ulcer Framework(c). *Adv Skin Wound Care* 2011 Dec;24(12):571-80; quiz 581-2.
8. Gray M, Black JM, Baharestani MM, Bliss DZ, Colwell JC, Goldberg M, et al. Moisture-associated skin damage: overview and pathophysiology. *J Wound Ostomy Continence Nurs* 2011 May-Jun;38(3):233-241.
9. Sibbald RG, Orsted H, Schultz GS, Coutts P, Keast D, International Wound Bed Preparation Advisory Board, et al. Preparing the wound bed 2003: focus on infection and inflammation. *Ostomy Wound Manage* 2003 Nov;49(11):23-51.
10. Norton L, Coutts P, Sibbald RG. Beds: practical pressure management for surfaces/mattresses. *Adv Skin Wound Care* 2011 Jul;24(7):324-32; quiz 333-4.
11. Woo KY. The use of antimicrobial dressings in chronic wounds: NERDS and STONEES principles. *Surg Technol Int* 2010 Oct;20:73-82.
12. Sibbald RG, Woo KY, Ayello EA. Increased bacterial burden and infection: the story of NERDS and STONES. *Adv Skin Wound Care* 2006 Oct;19(8):447-61; quiz 461-3.
13. Cichowitz A, Pan WR, Ashton M. The heel: anatomy, blood supply, and the pathophysiology of pressure ulcers. *Ann Plast Surg* 2009 Apr;62(4):423-429.
14. Woo KY, Abbott LK, Librach L. Evidence-based approach to manage persistent wound-related pain. *Curr Opin Support Palliat Care* 2013 Mar;7(1):86-94.
15. Brindle CT, Wegelin JA. Prophylactic dressing application to reduce pressure ulcer formation in cardiac surgery patients. *J Wound Ostomy Continence Nurs* 2012 Mar-Apr;39(2):133-142.
16. Chaiken N. Reduction of sacral pressure ulcers in the intensive care unit using a silicone border foam dressing. *J Wound Ostomy Continence Nurs* 2012 Mar-Apr;39(2):143-145.
17. Doughty D. Studies on the use of silicone foam dressing for prevention of sacrococcygeal breakdown in high-risk patients. *J Wound Ostomy Continence Nurs* 2012 Mar-Apr;39(2):150-151.
18. Walsh NS, Blanck AW, Smith L, Cross M, Andersson L, Polito C. Use of a sacral silicone border foam dressing as one component of a pressure ulcer prevention program in an intensive care unit setting. *J Wound Ostomy Continence Nurs* 2012 Mar-Apr;39(2):146-149.
19. Woo KY, Coutts PM, Sibbald RG. A randomized controlled trial to evaluate an antimicrobial dressing with silver alginate powder for the management of chronic wounds exhibiting signs of critical colonization. *Adv Skin Wound Care* 2012 Nov;25(11):503-508.
20. Woo KY, Sibbald RG. A cross-sectional validation study of using NERDS and STONEES to assess bacterial burden. *Ostomy Wound Manage* 2009 Aug 1;55(8):40-48.
21. Morykwas MJ, Simpson J, Punger K, Argenta A, Kremers L, Argenta J. Vacuum-assisted closure: state of basic research and physiologic foundation. *Plast Reconstr Surg* 2006 Jun;117(7 Suppl):121S-126S.

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