PROTECTING THE INTEGRITY OF THE PERIWOUND SKIN

This article will explore some of the main factors that affect periwound integrity. Early identification of periwound skin damage and treatment options will also be discussed. Skin damage relating to urine and faeces has been comprehensively reported elsewhere (Cooper, 2011; Bianchi 2012), and will not be discussed in this article.

- ▶ Periwound skin
- ▶ Exudate
- ► Allergic reactions
- ➤ Adherence of dressings
- ➡ Skin stripping

JANICE BIANCHI is a Medical Education Specialist at JB Med Ed Ltd; Honorary Lecturer at University of Glasgow

The skin is the largest organ in the body and covers an area of approximately two square metres. It has an inherent ability to regenerate itself and, to a certain degree, repair any damage that is inflicted upon it through the course of daily living (Hughes, 2001). Given the right conditions, wounds will heal through tissue regeneration. During this process, the integrity of the periwound skin can be affected by both intrinsic and extrinsic factors. Clinicians must alert themselves to the key factors that may exacerbate the vulnerability of the skin surrounding a wound and how to prevent or reduce the risk of further damage.

What are the dangers? *Exudate*

Constant exposure to moisture can result in damage to the periwound skin. Wound exudate, particularly from chronic wounds, contains not only water, but often cellular debris and enzymes (Chen and Rogers, 1992), and this mixture can be very corrosive to the intact skin surrounding the wound (Coutts et al, 2001). Several studies have examined the impact of chronic wound fluid on the wound environment. Phillips et al (1998) used cultured fibroblasts from human neonatal foreskins in a plated laboratory model and treated them with either chronic wound fluid or a control (bovine serum albumin). The researchers found that chronic wound fluid dramatically inhibited the growth of fibroblasts. They concluded that this study gave some indication of how the microenvironment has a negative effect on wound healing.

Matrix metalloproteinases (MMPs) are enzymes that are responsible for regulation of many biological processes including regulation of the extracellular matrix proteins (substances in the extra cellular space that serve as a scaffolding to hold tissue together). Trengrove et al (1999) found differences in the levels of MMPs in chronic wounds, compared with acute wounds. In a further study, they identified that the chronic wound fluid also contained higher levels of proinflammatory cytokines, free oxygen radicals and proteases, such as MMPs and elastase (Trengrove et al, 2000). These studies help us to understand the effects of chronic wound fluid on the wound bed and on the periwound area.

Exposure to abnormal levels of MMPs can cause damage in the wound bed, such as breakdown of the extracellular matrix, as well as to the periwound area as the proteins enveloping the corneocytes, the outermost layer of skin cells, are destroyed, damaging epidermal barrier function (Langoen and Bianchi, 2012). This can result in a red, weeping surface. Additionally, the pro-inflammatory cytokines in chronic wound fluid cause additional damage to the stratum corneum (the skin's outer barrier) further reducing skin barrier function (Wolcott et al, 2008).

Adherence of dressings

Adherence of dressing material to the wound bed and periwound skin can damage newly forming cells and cause distress to the patient. Dykes and Heggie (2003) found repeated application and removal of adhesive dressings can lead to damage to the skin's surface and can strip the stratum corneum. This initiates an inflammatory skin reaction, oedema and pain (Langoen and Lawton, 2009). Although this type of damage can occur at any age, skin weakens naturally as it ages (Cooper et al, 2006), therefore, older patients' skin may be particularly susceptible to external trauma, such as the removal of adherent dressings (Bianchi and Gray, 2011).

Pre-term infants also have fragile skin that is susceptible to trauma, has poor barrier properties and is vulnerable to infection (Van Onselen, 2001). Other variables include skin pathology, the properties of the adhesive and frequency of tape or dressing removal (Fluhr et al, 2002).

Allergic reactions

Patients can develop allergic or irritant reactions to the components of dressings, such as adhesives, and to topical applications. Studies have identified that patients with venous disease are more likely to develop allergies to products than the population in general. *Table 1* identifies the most common allergens in a group of 200 patients where patch testing was performed. National guidelines recommend simple dressings for leg ulcer patients (SIGN, 2010).

How to identify periwound damage

When caring for a patient with a wound, healthcare professionals should compile a detailed history of the patient's skin and reassess it regularly at dressing changes, planning management according to the risk factors identified. There are no instruments currently available to aid clinicians in identifying periwound skin damage. Several instruments have been developed to assess the degree of incontinence-associated dermatitis, however.

One such instrument, developed by Kennedy and Lutz, rates skin damage in terms of area of skin breakdown (from none to large area [>50cm²]), skin redness (from no redness to severe redness) and erosion (none to extreme erosion of the epidermis and dermis with moderate volume persistent exudates) (Kennedy and Lutz, 1996).

Although this tool has not been validated for use in assessing periwound skin, similar skin changes will take place. Skin breakdown, erythema and erosion commonly occur in skin that has been damaged by wound exudate (*Figure 1*).



Figure 1: Skin breakdown, erythema and erosion commonly occur in skin that has been damaged by wound exudate, as is evident here.

Table 1

The most frequent allergens found in venous leg ulcer patients (Tavadia et al, 2003).

Fragrance	30.5%
Antimicrobials	19.5%
Topical excipients	19.5%
Rubber	13.5%
accelerators	
Intrasite gel	9%
x x: 1	
Hioxyl cream	8%
	1000/
Total	100%

Maceration can also appear as a white margin around the wound. This occurs when moisture is trapped against the skin for a prolonged period. The skin will turn white or grey (*Figure 2*) and will soften and wrinkle (Langoen and Lawton, 2009).

This is a process that is purely moisturedependent and occurs as a result of over-hydration (Thomas, 2008). This change in the skin can lead to a breakdown of the periwound area (Cameron, 2004).

In instances of allergic reactions to dressings, there is often a clearly demarcated area where the dressing or topical treatment has been applied.

How to protect the periwound area

Periwound skin management should begin with protection against mechanical and chemical injury (Langoen and Lawton, 2009). A detailed assessment of the patient, the wound and the periwound skin with meticulous observation of the skin is key. This will provide clues to diagnosis, management and care of any existing or potential problems.

REVIEW



Figure 2: Examples of periwound maceration.

It is essential to work with the interprofessional team in order to correct the underlying factors where possible.

Exudate

If the wound is producing high levels of exudate, several factors should be taken into consideration. Infected wounds usually have an increased level of exudate and the exudate may have an offensive odour and can be the first indication of infection (Dealey, 2006). If infection does occur, the other clinical signs will usually be present. They include: pain, heat, swelling and erythema (Dealey, 2006).

Where a clinical diagnosis of infection has been made, treatment with antimicrobial therapy should be initiated. Clinicians should refer to their local protocol/guideline on the care of an infected wound. Comorbidities, such as venous disease, lymphoedema, cardiac failure and prolonged immobility, can lead to oedema, particularly in the lower leg (Williams and Moritmer, 2007).

If patients suffering from medical conditions that cause oedema also have a wound, exudate levels may be increased due to fluid overload. Compression is an effective method of controlling wound exudate in patients with venous disease as it reduces venous hypertension (SIGN, 2010). Patients with lymphoedema will also benefit from compression therapy (Lymphoedema Framework, 2006). Autolytic debridement occurs when necrotic tissue is softened by enzymes in the wound fluid (Vuolo, 2009).

This natural process can also cause an increase in exudate levels. Local treatment to avoid or treat periwound maceration includes the use of highly absorbent dressings. Dressings with a super-absorbent component provide effective protection (Langoen and Lawton, 2009).

Other technologies, such as topical negative pressure, should also be considered as they actively remove fluid from the wound bed. Skin barriers in the form of films and creams can also help by sealing healthy skin from bodily fluids (Coutts et al, 2001).

Skin stripping

Repeated application and removal of adhesive dressings and tapes can result in the stripping of the skin in both the wound and the periwound area (Cooper, 2011). This can cause pain, irritation and tissue breakdown. Minimising dressing changes can, therefore, be helpful. Identifying 'at risk' skin as previously described and avoiding adhesive products in vulnerable skin is essential. Silicone and lipocolloid-based dressings have been developed to minimise damage to the wound bed and surrounding skin and, as such, are suitable for friable skin.

Non-adherent dressings can also be used, particularly in patients who are having compression bandages or stockings applied as these will keep the dressing in place. Tubular bandages can also be used to secure a non adherent dressing.



Figure 3: Allergic reaction to paste bandage.

Allergic reactions

If an allergy is suspected, then it is important to remove the potential allergens from the patient's treatment. It is quite common for clinicians to mistakenly diagnose an allergic reaction in periwound tissue as an infection. Being meticulous when it comes to logging patient history, as well as when examining the skin, will aid diagnosis.

Applying topical corticosteroids to the periwound skin is the treatment of choice for allergic reactions. Clinicians should also seek to refer the patient to a dermatologist or dermatology department, where further investigations, such as patch testing, will be undertaken. The chosen dressing should have a proven low risk of contact reaction and good absorbency capacity. Dressings with adhesive borders should be avoided.

Conclusion

Any patient with a wound is vulnerable to periwound skin damage. This damage can lead to increased wound size, delayed healing and increased levels of pain. Consequently, healthcare costs may increase and the patient's quality of life may be reduced. Careful monitoring and ongoing assessment of both the wound and periwound skin will aid in identifying skin changes and treatment options. Clinicians need to have a good understanding of different causes of periwound damage to better understand these changes and initiate appropriate treatment strategies or onward referral. $W_{\rm E}$

References

Bianchi J (2012) Causes and strategies for moisture lesions. *Nurs Times* 108(5): 20–22

Bianchi J, Gray D (2011) Adaptic Touch non-adherent dressing. *Wounds UK* 7(1): 120–23

Cameron J (2004) Exudate and careof periwound skin. *Nurs Standard* 19(7): 62–66

Chen WYJ, Rogers AA (1992) Char-

acterisation of biological properties of wound fluid collected during the ealy stages of wound healing. J Invest Dermatol 99(5): 559–64

Coutts P, Queen D, Sibbald RG (2001) Periwound Skin Protection: A comparison of a new skin barrier vs. traditional therapies in wound management. Poster presentation CAWC, London

Cooper P (2011) Skin care: managing the skin of incontinent patients. *Wound Essentials* 6: 69–74

Cooper P, Clark M, Bale S (2006) Best Practice Statement: care of the older person's skin. *Wounds UK*, Aberdeen

Dealey (2006) The physiology of wound healing. *In The Care of Wounds: A Guide for Nurses* 3rd Edition. Blackwell Publishing, Oxford

Dykes PJ, Heggie R (2003) The link between the peel forces of adhesive dressings and the subjective discomfort in volunteer subjects. *J Wound Care* 2(7): 260–62

Fluhr JW, Dickel H, Kuss O et al (2002) Impact of anatomical location on barrier recovery, surface pH, and stratum corneum hydration after acute barrier disruption. *BJ Dermatol* 146(5):770–76

Hughes E (2001) Skin: its structure, function and related pathology. *In Hughes E, Van Onselen (ed): Dermatology Nursing A Practical Guide.* Churchill Livingstone, London

Kennedy KL, Lutz L (1996) Comparison of the efficacy and cost effectiveness of three skin protectants in the in the management of incontinence dermatitis. In: Proceedings of the European Conferenceon Advances in Wound Management. Amsterdam

Langoen A, Bianchi J (2012) Integrity and Skin Barrier Function: in Flanagan M (Ed) *Skin Integrity and Wound Healing: principles and practice*. Wiley-Blackwell

Langoen A, Lawton S (2009) Assessing and Managing vulnerable periwound skin. World Wide Wounds . Available at: *http://www.worldwidewounds. org/2009/October/Lawton-langoen/* *vulnerable-skin-2.html* (accessed 31 May 2012)

Lymphoedema Framework (2006) Best Practice for the Management of Lymphoedema. MEP, London

Phillips TJ, Al-Amoudi HO, Leverkus M, Park H-Y (1998) Effect of chronic wound fluid on fibroblasts. *J Wound Care* 7(10): 527–32

SIGN (2010) Management of Chronic Venous Leg Ulcers. Available at: *http:// sign.ac.uk/guidelines/fulltext/120/references.html* (accessed 28 March, 2012)

Tavadia S, Bianchi J, Dawe R et al (2003) Allergic contact dermatitis in venous leg ulcer patients. *Contact Dermatitis* 48: 261–65.

Thomas S (2008) The role of dressings in the treatment of moisture-related skin damage. Available at: *http://www. worldwidewounds.com/2008/march/ Thomas/Maceration-and-the-roleof-dressings.html* (accessed 28 March, 2012)

Trengrove MK, Stacey MC, McCauley S et al (1999) Analysis of the acute and chronic wound environments: the role of proteases and their inhibitors. *Wound Repair Regen* 7(6): 442–52

Trengrove NJ, Bielefeldt-Ohmann H,Stacey MC (2000) Mitogenic activity and cytokine levels in non-healing and healing chronic leg ulcers. *Wound Repair and Regen* 8(1): 13–25.

Van Onselen (2001) Age-specific issues in dermatology. *In Hughes E, Van Onselen (ed): Dermatology Nursing A Practical Guide.* Churchill Livingstone, London

Vuolo J (2009) Basic wound care procedures. *In Wound Care Made Incredibly Easy.* Lippincott Williams & Wilkins, London

Williams AF, Mortimer P (2007) Lymphoedema of the lower limb: causation, assessment and management. *In Morrison MJ, Moffatt CJ, Franls PJ (eds) Leg Ulcers A Problem Based Learning Approach*. Mosby Elsevier, London

Wolcott RD, Rhoads DD, Dowd SE (2008) Biofilms and chronic wound infection. *J Wound Care* 17: 333–41