

# Maceration and its effect on periwound margins

Alison Rodgers and Lynne Watret

## ARTICLE POINTS

**1** In the diabetic foot, loss of autonomic nerve supply can alter the vascular perfusion and nerve supply of the skin.

**2** Choosing the appropriate dressing and determining the frequency of dressing changes to cope with the levels of exudate remain common problems for clinicians involved in wound care.

**3** Evaluating the the periwound margins is an essential part of wound assessment.

**4** Knowledge of dressings and of the moisture vapour transmission rate is essential in deciding how long dressings may remain in situ.

## KEY WORDS

- Wound care
- Exudate
- Maceration
- Moisture vapour transmission rate
- Clinical knowledge

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## Introduction

**Diabetic foot ulceration continues to be synonymous with delayed healing, higher infection rates and an increased risk of lower extremity amputation (Frykberg, 1998). A number of factors can affect the local wound environment in diabetic foot ulceration including: hyperglycaemia; macrovascular and microvascular disease; polyneuropathy; and impaired host immunological defence (Kamal et al, 1996; Boulton, 2000). Maceration of the wound bed and surrounding skin in diabetic foot ulceration may be one of the least well recognised factors contributing to impaired healing (Cullum, 2000). The impact of maceration on skin integrity, its traditionally poor management and frequency make it an obvious contender for inclusion as a risk factor in wound care (Cutting and White, 2002). However, there is little research on the possible implications of maceration in diabetic foot ulceration (Bale et al, 2001). The focus of this article is on the problem and management of maceration of the periwound skin in the diabetic foot.**

**T**he aims of wound management are: to address patient concerns; correct intrinsic and extrinsic factors where possible; and to optimise the healing environment (Clark, 1995). It is also essential to include the periwound margins as an integral part of wound assessment (Cutting, 2002).

## Maceration in wound healing

Maceration of the epithelium has been described as the softening of the skin by exposure to excessive amounts of liquid for extended periods (Bowser et al, 1985). *Figure 1* shows an amputated toe with macerated surrounding skin.

Pathophysiologically, maceration reduces the physical and chemical integrity of the stratum corneum, thereby predisposing the periwound area to potential invasion by bacteria and fungi (Butcher, 2000). Studies of pressure ulcer development have also implicated maceration with a reduction in tensile strength of skin to axial pressure and shear forces (Thyagoragan and Silver, 1984).

In the diabetic foot, loss of autonomic nerve supply can alter the vascular perfusion and nerve supply of the skin. This affects the integrity of the skin and its resistance to mechanical and chemical trauma from pressure and wound exudate

(Faber et al, 1993). Maceration is a common problem, particularly in the management of chronic wounds (Thomas, 1997). Consideration should be given to the effects of the water content in wound exudate, and to the degree and nature of the inflammatory exudate in acute and chronic wounds (Cutting and White, 2002).

Various studies have suggested qualitative differences between acute and chronic wound exudate (Chen et al 1992; Trengrove et al, 1999). Chronic wound exudate has a potentially destructive composition of high levels of inflammatory mediators such as matrix metalloproteinases and plasminogen activators (Trengrove et al, 1999). This over elevation of inflammatory cytokines has been suggested as a causative factor in the degranulation of periulceration skin, and may result in a prolonged inflammatory response (Cullen, 2001). Nelson (1997)



*Figure 1. Maceration in wound healing*

suggests that prolonged contact with moisture may also increase the likelihood of an extension of the wound margin.

The use of dressings can be an effective method of managing excess exudate without damaging the periwound skin. However, choosing the appropriate dressing and determining the frequency of dressing changes to cope with the levels of exudate remain common problems for clinicians involved in wound care (Cutting and White, 2002).

The impaired inflammatory response in diabetes with the subsequent reduction in leucocyte infiltration, decreased phagocytosis and poor chemotaxis have a knock-on effect on the normal healing process and may potentially lead to an increased risk of clinical infection (Reiber et al, 1992; Kamal, 1996). Diabetic foot ulcers are often polymicrobial with gram-positive aerobes (such as *Staphylococcus*) often comprising the most commonly isolated microbiological species (Ge et al, 2002). When maceration is present and becomes infected it is more likely to be caused by organisms that prefer an environment with high water content such as *S. aureus* (Troller, 1978). Axelrod (1985) suggested that people with diabetes are five times more susceptible to fungal and bacterial infections. A retrospective study by Eneroth et al (1997) reported that in the diabetic foot, infection was the leading cause of admission to hospital, and subsequent amputation.

**The role of wound dressings in the management of maceration**

Winter (1962) discussed the effect of moist wound healing using an animal model and suggested that epithelialisation occurs optimally in a moist wound environment. Although this work was originally carried out on acute superficial wounds, the research has been transposed effectively to acute and compromised wounds in humans. Turner (1985) developed the criteria for the ideal dressing; maintaining a moist warm environment and absorbing excess exudate were viewed as favourable characteristics.

Dressings with these characteristics were considered to be a major advance in wound management (Jones, 1998) with additional criteria cited for diabetic foot lesions (Foster, 2002; Watret and Rodgers, 2003). A range of smaller sizes and shapes for foot lesions is important when considering the

effects of moisture retention and absorption. Cutting the dressings may compromise their ability to retain moisture, so it is valuable to have smaller sized dressings or those which can be cut to size, to not affect the integrity of the dressing.

Traditionally, wound care products, such as alginates as primary dressings and hydrocolloids as secondary dressings, were viewed as having the ability to absorb excess exudate and create a gel which provided a moist interface between the dressing and granulation tissue, which helps to optimise epithelialisation (Bale et al, 2001). Alginates remain widely used. However, additional dressings have been developed which help minimise maceration, such as hydrofibre dressings which do not cause lateral wicking and lock exudate into the dressing (McInnes, 1998). Alternatively, the patients' footwear or orthotic devices may affect the moisture vapour transmission rate (MVTR). High loading plantar pressure may also have the potential to force exudate laterally to surrounding tissue, increasing the risk of maceration (Sharman and Kerr, 2000).

Dressings such as soft silicone foam are of value in the management of diabetic foot lesions (Jones, 1998; Watret and Rodgers, 2003). Soft silicone does not adhere to the wound bed and ensures atraumatic removal at dressing changes. The risk of maceration is reduced by the properties of the soft silicone dressing which absorbs large amounts of exudate into the dressing whilst maintaining a moist interface at the wound bed. The absorbent layer of the dressing has a wicking effect on exudate, drawing the excess exudate from the wound bed through an absorbent layer to a vapour-permeable backing film which allows

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**1** When maceration is present and becomes infected it is more likely to be caused by organisms that prefer an environment with high water content such as *S. aureus*.

**2** Cutting dressings may compromise their ability to retain moisture, so it is valuable to have smaller sized dressings or those which can be cut to size, which do not affect the integrity of the dressing.

**3** Patients' footwear or orthotic devices may affect the moisture vapour transmission rate.

**4** Dressings such as soft silicone foam are of value as they do not adhere to the wound bed and ensure atraumatic removal.

**5** The risk of maceration is reduced by the properties of the soft silicone dressing which absorbs large amounts of exudate into the dressing whilst maintaining a moist interface at the wound bed.



Figure 1. Mepilex dressing

**PAGE POINTS**

**1** Dressings designed to remain in situ for longer periods of time may require more frequent changes and could result in unrealistic expectations due to alteration of the fluid handling ability of the dressing.

**2** A primary contact dressing with a high MVTR is of value when managing a highly exuding wound, by limiting the amount of exudate in contact with the skin.

**3** Whenever possible, multiple dressings should be avoided, with the exception of hydrogels or topical antimicrobials.

**4** When debridement of slough or necrotic tissue is required, surgical or sharp debridement should be carried out to minimise the need for autolytic debridement whenever possible.

**5** Sound clinical judgment of the practitioner is necessary to ensure that the balance between hydration of slough at the wound bed is not at the expense of destruction of surrounding tissue.

moisture to evaporate through the back (Thomas, 2003). Thomas (2003) argues that a soft silicone adhesion layer also inhibits lateral movement of exudate, thereby preventing maceration to surrounding skin.

The occlusive nature of dressings can exacerbate maceration by minimising transepidermal water loss (Van der Walk and Maibach, 1990). Conway (2002) argues that occlusive dressings have the potential to trap exudate resulting in maceration. However, this may occur in occlusive dressings with a low MVTR that have been left in situ for extended periods.

Hydrocolloid dressings are still used in the care of diabetic ulcers (Jones and Gill, 1998). Foster et al (1997) raised concern over the deterioration of wounds under hydrocolloid dressings on diabetic foot lesions. However, dressings were in situ for prolonged periods of time (5–7 days) and in some cases infection was already apparent. Jones and Gill (1998) discussed the value of hydrocolloids in optimising the healing environment by maintaining a moist warm environment. However, they also noted that there remain major concerns over maceration and frequency of dressing changes. Dressings designed to remain in situ for longer periods of time may therefore require more frequent changes and could result in unrealistic expectations due to alteration of the fluid handling ability of the dressing under these circumstances. This is not necessarily a fault with the dressing; careful assessment and knowledge of the structure and function of individual dressings is important to ensure best practice.

A primary contact dressing with a high MVTR is of value when managing a highly exuding wound, by limiting the amount of exudate in contact with the skin (Thomas, 1997). However, the MVTR may be altered due to the use of multiple layered dressings being used beneath this in an attempt to extend wear time, which may in turn reduce the efficacy of the original dressing, which has now become a secondary dressing. This may also increase the likelihood of maceration to surrounding skin as the primary contact dressing exceeds its maximum capacity for absorption.

Whenever possible, multiple dressings should be avoided, with the exception of hydrogels or topical antimicrobials. The advantages of using one dressing are that it:

- Allows the dressing to carry out its fluid handling function.
- Reduces bulk
- Is easily applied and removed.

This allows the cost effective use of products and reduction in time taken to carry out the procedure. On removal of wet dressings, the patient may detect an odour, which can be distressing and affect patient compliance with dressing regimens (Foster, 1997). Pain at dressing changes, often attributed to a prolonged inflammatory response following maceration may not be a concern in the neuropathic foot (Butcher, 2000).

**Getting the balance right**

When debridement of slough or necrotic tissue is required, surgical or sharp debridement should be carried out to minimise the need for autolytic debridement whenever possible (Steed et al, 1996). However, if this is not possible, promoting autolytic debridement by hydration is effective. Hydrogels absorb their own volume in exudate and in the process of hydration may cause some maceration to the periwound margins. This may be a particular problem in patients with ischaemic foot lesions (Knowles and Jackson, 1997). It is therefore important to ensure that the minimum amount of hydrogel is used, which will assist in hydration of slough whilst not spreading laterally to surrounding tissue. Secondary dressings should promote wicking excess exudate away from the wound bed, whilst allowing the hydrogel to hydrate slough but not dehydrate the wound bed (Jones, 1998). If maceration still occurs, it may be necessary to increase dressing changes to prevent the potential destructive effect of the components of exudate on the periwound margins. Sound clinical judgment of the practitioner is necessary to ensure that the balance between hydration of slough at the wound bed is not at the expense of destruction of surrounding tissue.

**Conclusion**

Effective wound management remains difficult, despite increased awareness of the underlying causative factors (Steed,1998). Various studies use maceration as an outcome measure of the role of wound dressings in retaining exudate away from the healthy skin. However, few have examined the effects of maceration on the diabetic foot (Bale et al, 2001). There is a need for further research into the appropriate use of dressings and their role in reducing maceration in diabetic foot lesions. Knowledge of dressings and of the MVTR are essential in deciding how long the dressings may remain in situ dependent on patient needs and preferences. The fundamental cornerstone for the treatment of maceration is good skin care based on sound clinical knowledge about the detrimental effects of exudate on periwound margins and the attributes of available dressings, combined with skill. ■

Axelrod S (1985) Infections in the diabetic patient. *Clinical Diabetes* 3: 98

Bale S, Baker N, Crook H et al (2001) Exploring the use of an alginate dressing for diabetic foot ulcers. *Journal of Wound Care* 10(3): 81–84

Boulton AJ (2000) The pathway to ulceration: aetiopathogenesis. *The Foot in Diabetes* 3rd Edn. (Eds. Boulton A, Connor H, Cavanagh PR) Wiley & Sons Ltd

Bowser PA, White RJ (1985) Isolation, barrier properties and lipid analysis of stratum compactum, a discrete region of the stratum corneum. *British Journal of Dermatology* 112: 1–14

Butcher M (2000) The management of skin maceration. *Nursing Times* 96(45): 35–36

Chen WY, Rogers AA, Lydon MJ (1992) Characteristics of biologic properties of wound fluid collection during early stages of wound healing. *Journal of Investigative Dermatology* 99(5): 559–64

Clark RA (1995) Wound repair: overview and general considerations. In: Clark, RA (Ed) *The molecular and cellular biology of wound repair* 2nd ed. Plenum Press, New York

Conway J (2002) Adverse reactions to wound dressings. *Nursing Standard* 16(44): 52–60

Cullen B (2001) Chronic wound healing. Wound Care Device Expert Meeting. *The Diabetic Foot* 4(3): S4–S5

Cullum N, Najid M, O'Meara S, Sheldon T (2000) Use of dressings: is there an evidence base? In: Boulton AJM, Connor H, Cavanagh PR (Eds.) *The Foot in Diabetes* 3rd Edn. Wiley, Chichester. 153–68

Cutting KF (2002) Avoidance and management of per-wound maceration of the skin. *Professional Nurse* 18(1): 33–36

Cutting KF, White RJ (2002) Maceration of the skin and wound bed I: its nature and causes. *Journal of Wound Care* 11(7): 275–78

Eneroth M, Apelqvist J, Stenstrom A (1997) Clinical characteristics and outcome in 223 patients with deep foot infections. *Foot Ankle International* 18: 716–22

Faber WR, Michels PPJ, Naafs B (1993) The neuropathic foot. In: Westerhof W (Ed) *Leg ulcers*

*diagnosis and treatment* Elsevier, Amsterdam Chapter10

Foster A (1997) Psychosocial aspects of treating the diabetic foot. *Practical Diabetes International* 1(2): 56–58

Foster AVM (2002) Is there any evidence base for diabetic foot care? *Journal of Tissue Viability* 12(3): 347–52

Frykberg R (1998) Diabetic foot ulcers: current concepts. *Journal of Foot and Ankle Surgery*. 37(5):440–46

Ge Y, MacDonald D, Hait H et al (2002) Microbiological profile of infected diabetic foot ulcers. *Diabetic Medicine* 19(12):1032–34

Jones V, Gill D (1998) Hydrocolloid dressings and diabetic foot lesions. *The Diabetic Foot* 1(4):127–34

Jones V (1998) Selecting a dressing for the diabetic foot: factors to consider. *The Diabetic Foot*. 1(2):48–52

Kamal K, Powell RJ, Sumpio BE (1996) The pathology of diabetes mellitus: implications for surgeons. *Journal American College of Surgeons* 183(3): 271–89

Knowles EA, Jackson NJ (1997) Care of the diabetic foot. *Journal of Wound Care* 6(5): 227–30

McInnes A (1998) Aquacel in the management of the diabetic foot. In: *Proceedings of the satellite symposium of the European Academy of Dermatology and Venereology: the next step in wound care dressings*. Kreig T, Harding KG (Eds) Churchill Communications, London

Nelson A (1997) Is exudate a clinical problem? In: *Proceedings, joint meeting, European Wound Management Association and European Tissue Repair Society: management of wound exudate*. Cherry G, Harding K (Eds) Churchill Communications, London

Reiber GE, Pecoraro RE, Koepsell TD (1992) Risk factors for amputation in patients with diabetes mellitus. A case controlled study. *Annals of Internal Medicine* 117: 97–105

Sharman DL, Kerr D (2000) Practical management of neuropathic diabetic foot ulcers. *The Diabetic Foot* 3(2): 49–54

Steed DL, Donohoe D, Webster MW, Lindsley L (1996) Diabetic ulcer study group. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. *Journal of the American College of Surgeons* 183: 61–64

Steed DL (1998) Foundations of good ulcer care. *The American Journal of Surgery* 176 (Supp 2A): 20–25

Thomas S (1997) Assessment and management of wound exudate. *Journal of Wound Care* 6(7): 327–30

Thomas S (2003) *Atraumatic dressings*. [www.worldwidewounds.com](http://www.worldwidewounds.com)

Thyagaragan C, Silver JR (1984) Aetiology of pressure sores in patients with spinal cord injury. *British Medical Journal* 289: 1487–89

Trengove NJ, Stacey MC, McAuley S et al (1999) Analysis of acute and chronic wound environments: the role of proteases and their inhibitors. *Wound Repair Regeneration* 7(6): 422–52

Troller JA, Stinson JV (1978) Influence of water activity on the production of extracellular enzymes by *Staphylococcus aureus*. *Applied and Environmental Microbiology* 35(3): 521–26

Turner TD (1985) Which dressing and why? In: *Wound care London*. Westaby S (Ed) Heinemann

Van der Walk P, Maibach H (1990) A functional study of the skin barrier to evaporative water loss by means of repeated cellophane stripping. *Clinical and Experimental Dermatology* 15(3): 180–82

Watret L, Rodgers A (2002) Effectiveness of foam dressings on diabetic foot lesions. *The Diabetic Foot* 5(3): 115–24

Winter GD (1962) Formation of the scab and rate of epithelialisation of superficial wounds in the skin of the young domestic pig. *Nature* 193: 293–94

**PAGE POINTS**

- 1 There is a need for further research into the appropriate use of dressings and their role in reducing maceration in diabetic foot lesions.
- 2 Knowledge of dressings and of the MVTR are essential in deciding how long the dressings may remain in situ dependent on patient needs and preferences.
- 3 The fundamental cornerstone for the treatment of maceration is good skin care based on sound clinical knowledge about the detrimental effects of exudate on periwound margins and the attributes of available dressings, combined with skill.