

Promoting Evidence-Based Nursing Practice: *Wound Bed Preparation*

Introduction

District Nurses, in collaboration with community dwelling clients, take responsibility for wound management (on average 39% of visits involve wound care). Guided by evidence, sound wound management practice involves: determination of wound aetiology; identification and control of factors impairing healing; and providing appropriate local wound care. However, even when these principles have been applied some wounds do not heal (Sibbald et al 2000). Wounds which do not heal in a timely and orderly manner are classified as chronic wounds. In this newsletter it is proposed that wound bed preparation provides the clinician with a systematic approach to the management of chronic wounds. The principles of wound bed preparation aim to address aspects of local wound care in the chronic wound which can be altered to overcome the barriers to healing (Pudner 2002).

Part of a comprehensive approach

Central is that clients are partners in wound management. All wound management should be based on a comprehensive client assessment carried out with the client. Whilst wound bed preparation can help the clinician understand and manage chronic wounds at a local level, it is only effective when the cause of the wound and client factors impairing healing are identified and addressed (Keast et al 2004). Wound aetiology, factors impairing healing, social and psychological issues and wound characteristics are considered. For example, management of venous insufficiency (through compression therapy) or control of diabetes mellitus will promote favourable systemic conditions for wound healing to occur. There will be some wounds where healing is not an expected outcome, for example, a person with a malignancy or when a person with severe, inoperable arterial disease has a wound. Management of non healable wounds involves different aims and strategies. Whilst comprehensive assessment and care of the whole person is the foundation of sound wound management, this paper will limit discussion to optimising the local wound environment in persons with chronic wounds where healing potential exists.

The pathophysiology of chronic wounds

Chronic wounds do not progress through the acute wound healing phases of haemostasis, inflammation, proliferation and maturation (Enoch & Harding 2003). This results in a number of features and processes occurring. Clinically, the wound may present with slough, necrotic tissue, unhealthy granulation tissue, lack of re-epithelialisation and recurrent wound breakdown (Enoch & Harding 2003). Microbiology often reveals high wound bacterial load, with multiple organisms. The environment and fluid of chronic wounds is very different from that found in acute wounds. Chronic wounds have elevated levels of matrix metalloproteinases, inflammatory cytokines, decreased growth factor activity, low levels of cell mitosis (division) and senescent (inactive) cells (Enoch & Harding 2003). It is often stated that chronic wounds are 'stuck' in the

inflammatory or proliferative phases of healing (Pudner 2002).

The role of wound bed preparation

Wound bed preparation aims to overcome factors at the wound site that are impairing healing. Initially wound bed preparation was developed in order to prepare wounds to maximise the efficacy of advanced products such as growth factors and skin substitutes. However, it became apparent that the principles of wound bed preparation provided a management model which addressed the particular characteristics of the chronic wound. Previously, acute wound healing techniques were applied to the management of chronic wounds, often unsuccessfully (Schultz et al 2004). The principles of wound bed preparation have been articulated as the acronym TIME (see table 1).

Table 1 TIME

	Framework terms	Application to practice
T	Tissue management	Removal of non viable tissue.
I	Inflammation and infection control	Control of bacterial load/burden.
M	Moisture balance	Management and control of exudate.
E	Epithelial (edge) advancement	Promotion of a healthy, advancing wound margin.

(Falanga 2004)

The extent to which each factor in wound bed preparation is relevant can be linked to the aetiology of the chronic wound. For example, the focus in managing a venous leg ulcer will usually be on **Moisture balance**, whilst the focus in managing a foot wound in a person with diabetes will be on **Tissue management** and **Inflammation and infection control** (Moffatt 2004).

Tissue management

Non viable tissue such as slough and necrosis in a wound will delay healing (Sibbald et al 2000). The term *necrotic burden* has been used to describe the load of non viable tissue, excess exudate and bacteria within dead tissue (Schultz et al 2004). In the presence of adequate blood supply, debridement of non viable tissue is essential as it will accelerate healing (Smith & Nephew 2001). There are five main methods of debridement: surgical/sharp, autolytic, mechanical, enzymatic and biological (Schultz et al 2000). Enzymatic and biological (larvae) debridement is rarely used in Australia and mechanical debridement (eg. wet to dry saline soaks) is avoided due to the associated pain and potential damage to healthy tissue (Pudner 2002). Whilst surgical wound debridement is fast and effective it is usually performed by skilled medical practitioners. Conservative sharp wound debridement can be performed by skilled clinicians in a variety of settings (Smith & Nephew 2001). Achievement of a bleeding wound base may revitalise the wound bed and promote healing by turning a chronic wound into an acute wound (Enoch & Harding 2003, Sibbald et al 2000). Autolytic debridement is most commonly used as it is gentle, effective and selective. Autolytic debridement is achieved through the use of moist wound healing dressings to provide an environment which liquefies slough and necrotic tissue (Pudner 2002). The

chosen dressing should be appropriate for the exudate level of the wound. Due to their high moisture content, hydrogels are efficient for debriding dry, necrotic wounds. However, they should not be used on wounds with moderate to large amounts of exudate as this will result in maceration and possible wound breakdown. When attempting to debride hard, dry eschar a product such as a hypertonic gel can be useful or, alternatively, the eschar can be scored with a scalpel to promote penetration of the hydrogel (Sibbald et al 2000). Chronic wounds may require repeated debridements (Falanga 2004).

Inflammation and infection control

Bacterial load or burden follows a continuum which includes: contamination → colonisation → critical colonisation → local infection → systemic infection (Sibbald et al 2000). Of particular importance to clinicians is the stage of critical colonisation, as these wounds fail to heal, yet do not exhibit the 'classic' signs of infection (Enoch & Harding 2003). Managing bacteria is dependent on the type/s of bacteria, level of the load and host reaction. Critical colonisation and local infection are usually effectively managed through the use of topical antimicrobial agents such as cadexomer iodine and slow release silver dressings (Schultz et al 2004). Systemic antibiotics should only be utilised when absolutely necessary due to the risk of bacterial resistance (Smith & Nephew 2001). When using a topical antimicrobial agent the clinician needs to understand its mode of action, how to apply it correctly and any precautions. If the wound does not respond to a topical antimicrobial in 2-3 weeks its use should be re-evaluated and consideration given to other factors which may be impairing healing (Sibbald et al 2000).

Moisture balance

In an acute wound exudate contains substances which promote healing. However, the amount and composition of chronic wound exudate can impair healing (Enoch & Harding 2003). Determining the exudate level of a wound allows the clinician to choose an appropriate dressing which will control exudate to produce a moist wound environment. If the wound environment is too moist maceration will occur. If the wound environment is too dry desiccation will occur (Sibbald et al 2000). Whilst there are many products on the market today, the following is a basic guide:

Table 2 basic guide to choosing a dressing product

Exudate level	Suitable product
Dry wound/no exudate	Hydrogel
Low exudate	Hydrocolloid
Moderate exudate	Foam
High exudate	Alginate or hydrofibre

(Schultz et al 2004)

Before applying any dressing the clinician should be aware of: its indications for use (including what level of exudate it is appropriate for); how to apply it (eg. amount of overlap, which side is against the wound, need for fixation or secondary dressings); expected wear time; and any precautions or contraindications (eg. suitability for use on infected wounds). In the management of

chronic wounds dressings form only one component of an overall management regime. Dressings alone are unlikely to heal chronic wounds.

Epithelial (edge) advancement

The epidermis in chronic wounds fails to advance across the wound, thereby inhibiting wound closure, and the margin often hyperproliferates (Smith & Nephew 2001). This results in chronic wounds with undermined, distinct, raised or rolled edges. If a wound has not decreased in size by 30% within four weeks, it is unlikely to heal within twelve weeks (Sibbald et al 2000). Utilising sound principles of wound bed preparation to control bacterial load, reduce necrotic burden and manage exudate may result in the conditions to allow advancement of the epithelial edge and ultimately, wound closure (Schultz et al 2004). If the wound fails to respond, despite control of physiological and local factors impairing healing, consideration may be given to using advanced therapies (Sibbald et al 2000). Advanced therapies currently available include: negative pressure therapy (VAC), bio-engineered skin substitutes, growth factor/cytokine preparations and matrix metalloproteinase inhibitors (Schultz et al 2004, Sibbald et al 2000).

Conclusion

Optimal management of chronic wounds is based on understanding the altered wound healing processes involved. A regime for management of chronic wounds to promote optimal client outcomes is:

- delivered by knowledgeable and skilled clinicians;
- based on comprehensive assessment;
- implemented according to sound principles which address underlying physiological problems and adequately prepares the wound bed; and
- undertaken in collaboration with the client.

TIME forms a foundation upon which to base the management of chronic wounds. The pathophysiology of chronic wounds is becoming better understood. With research and technological advancements, innovative strategies to manage these challenging wounds will be developed and clients will continue to benefit from our evidence based interventions

References

Enoch, S. & Harding, K. 2003, 'Wound bed preparation: The science behind the removal of barriers to healing', *WOUNDS*, Vol. 15, No. 7, pp. 213-229.

Falanga, V. 2004, 'Wound bed preparation: science applied to practice', *European Wound Management Association (EWMA) Position Document: Wound bed preparation in practice*, MEP Ltd, London.

Keast, D.H., Bowering, C.K., Evans, A.W., MacKean, G.L., Burrows, C. & D'Souza, L. 2004, 'MEASURE: A proposed assessment framework for developing best practice recommendations for wound assessment', *Wound Repair and Regeneration*, Vol. 12, No. 3, pp. S1-S17 (supplement).

Moffatt, C.J. 2004, 'Wound bed preparation in practice', *European Wound Management Association (EWMA) Position Document: Wound bed preparation in practice*, MEP Ltd, London.

Pudner, R. 2002, 'Wound bed preparation', *Journal of Community Nursing*, Vol. 16, Issue 5, p. 35.

Schultz, G.S., Barillo, D.J., Mozingo, D.W. & The Wound Bed Advisory Board Members, 2004, 'Wound bed preparation and a brief history of TIME', *International Wound Journal*, Vol. 1, No. 1, pp. 19-32.

Sibbald, R.G., Williamson, D., Orsted, H.L., Campbell, K., Keast, D., Krasner, D. & Sibbald, D. 2000, 'Preparing the wound bed -Debridement, bacterial balance and moisture balance', *OstomyWound Management*, Vol. 46, No. 11, pp. 14-35.

Smith & Nephew, 2001, *Science of Wound Management: Wound Bed Preparation*, Smith & Nephew Medical Ltd, Hull, England.

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