The role of the nurse in wound bed preparation


Summary

This article explores the role of the nurse in wound bed preparation. Wounds cannot be treated in isolation – many patient factors will influence healing. The nurse needs to have an understanding of the process of wound healing and have undertaken a full patient assessment before focusing on the patient's wound. Recognising and managing problems at the wound bed, for example necrotic tissue and excess exudate, can result in a better prepared wound bed and optimal healing. If the concept is to be valuable to nurses, they need to be part of the debate that defines wound bed preparation, how it is being implemented in clinical practice and how patients can benefit. This debate needs to include all nurses involved in wound care.

Wound bed preparation is emerging as an essential element for gaining maximum benefits from advanced wound management products, such as growth factors and bio-engineered skin products. The aim is to create an optimal wound healing environment, by producing a well vascularised, stable wound bed with minimal exudate. The five key components of wound bed preparation that will be discussed in this article, include (Falanga 2000):

- Management of necrosis.
- Management of wound exudate.
- Restoring bacterial balance.
- Correcting cellular dysfunction.
- Restoring biochemical balance.

Some nurses might say that the concept of wound bed preparation is not new, but part of overall good wound care, others believe that it is a new way of looking at an existing problem of chronic wounds (Collier 2002). Whatever the nurse’s belief or understanding of the concept, his or her role in wound bed preparation involves:

- Knowledge and understanding of the wound healing process.
- Undertaking a full and detailed patient assessment, taking into consideration patient concerns and factors that might have an effect on wound healing.
- Assessing the wound and the tissue type at the wound bed, for example, necrotic or granulating.

- Having the knowledge and skills to implement treatments that move the wound bed along a continuum to healing. This might involve a variety of methods, for example, debridement, exudate reduction, elimination of infection and the use of advanced technologies.

Wound bed preparation aims to achieve optimal wound healing. To appreciate and apply the concept, nurses need to have an understanding of the wound healing process and how it relates to the patient. Through this knowledge and patient assessment, they can then recognise abnormalities and select the most appropriate treatment for the stage of healing.

Wound healing is a complex process with interdependent and overlapping stages. These include (Dealey 1999):

- Inflammation.
- Reepithelialisation.
- Maturation.
- Correcting cellular dysfunction.
- Restoring biochemical balance.

Stages of wound healing

**Inflammation** The inflammatory response is a non-specific local reaction to tissue damage and/or bacterial invasion. Any trauma to the skin that penetrates the dermis will result in bleeding. The injured blood vessels will constrict to minimise blood loss. Fibrinogen is released, which then converts to fibrin, resulting in clot formation. Tissue damage and activation of clotting factors stimulate the release of inflammatory mediators such as prostaglandins and histamine from mast cells. These mediators cause blood vessels adjacent to the injured area to become more permeable and vasodilate, resulting in an inflammatory response. This can be identified by the presence of localised heat, swelling, erythema, discomfort and functional disturbance (Tortora and Gabowski 1996).

It is important that this stage of wound healing is not confused with the presence of infection. During the inflammatory stage of healing, polymorphonuclear neutrophils migrate into the surrounding tissue to defend the body against microbial invasion. Inflammation lasts about four to five days, but this can be prolonged by the presence of infection, foreign bodies or inappropriate wound dressings, causing trauma to the wound (Dealey 1999).
Reconstruction The reconstruction phase is characterised by the development of granulation tissue. It consists of a loose matrix of fibrin, fibronectin, collagen and hyaluronic acid, and other glycosaminoglycans. This process stimulates synthesis of collagen matrix, which provides the framework into which new capillaries will grow. The growth of new blood vessels is termed angiogenesis and allows the wound to take on a red granular appearance, with the edges of the wound starting to contract inwards. The length of time needed for reconstruction depends on the type and size of the wound, but can take up to 24 days (Dealey 1999).

Epithelialisation During this stage of wound healing, the wound is covered with epithelial cells. Macrophages release epidermal growth factors, which stimulate proliferation and migration of epithelial cells. The cells move over the wound surface in a leapfrog fashion, the first cell remaining on the wound surface and forming a new basement membrane. This stage commences early in wound’s healing by primary closure, such as post-operative wounds, but cavity wounds need to be filled with granulation tissue before epithelialisation can begin.

Maturation The maturation, or remodelling, stage is the final stage of wound healing, during which the wound becomes less vascularised as there is a reduction in the need to bring cells to the wound site. The collagen fibres are reorganised so that they lie at right angles to the wound margins. The scar tissue present is gradually remodelled and tensile strength increases. This stage begins at approximately 21 days and can take from a few months to a year for wounds healing by primary closure. It takes much longer for those healing by secondary closure (Dealey 1999).

Not all wounds pass from one stage of healing to the next and many chronic wounds, for example leg ulcers and pressure ulcers, can remain in the inflammatory stage of healing, resulting in delayed healing. Many factors will influence the wound healing process including:

- The patient’s general health and immunological status.
- Medication.
- Nutritional state.
- Wound type, site, duration and exudate levels.
- Bacterial status of the wound.

The concept of wound bed preparation cannot, therefore, be considered in isolation and many factors will need to be taken into account when assessing the patient. Only through a comprehensive patient assessment can the nurse identify the underlying cause of the wound and develop a treatment plan for the patient. Patient concerns and psychosocial circumstances need to be an integral part of the plan of care if it is to be successful.

The next focus of the assessment will be on the wound and how the wound bed can be prepared for optimal healing. A full and detailed wound assessment should include the following details:

- Wound history.
- Location.
- Size.
- Condition of the wound bed.
- Condition of surrounding skin.
- Wound pain.

This should be documented on a wound assessment and evaluation form, and some form of wound measurement should take place, for example, wound photography or tracing. This allows the patient and nurse to follow the progress of the wound and change treatment as necessary.

Assessment of the wound bed will take into account the five main components of wound bed preparation, as outlined (Falanga 2000). It will also enable the nurse to make a decision about treatment options to address identified problems. An example of this might be the removal of necrotic tissue through sharp debridement, and then the use of an absorbent dressing to manage exudate levels and allow for autolytic debridement.

Management of necrosis Debridement of devitalised tissue is an essential step to the success of wound management (Bergstrom et al 1994), provided that adequate blood supply to the wound is present. Necrotic tissue can be a focus for bacteria and a barrier to healing. There are a number of methods that nurses can use to debride a wound and these include:

- Sharp.
- Surgical.
- Enzymatic.
- Autolytic.
- Larval.
- Mechanical.

The method that the nurse selects will depend on many factors as outlined in Box 1.

The knowledge and skills of the nurse in the chosen method of wound debridement will also influence the choice, as will availability of equipment and products. These factors alone should not, however, be the main influence, as nurses should be able to access training and bid for funding for necessary equipment.

Box 1. Key factors in choosing a debridement

- Speed
- Tissue type
- Painful wound
- Exudate levels
- Presence of infection
- Acceptability to the patient
- Cost

(Adapted from Sibbald et al 2000)
Having decided the method, the nurse will need to:

I Define an aim – for example, the aim for a patient with peripheral vascular disease with a necrotic toe (Figure 1) will be different to the aim for a patient with a necrotic pressure ulcer (Figure 2). The former should be left dry and intact and the patient referred to a vascular surgeon. In some cases, necrotic digits will autodebride.

I Define a timescale – this involves balancing the risk to surrounding viable tissue from rapid debridement techniques against the danger of prolonged exposure to necrotic tissue and the risk of infection.

I Define a method – this will be determined by the factors listed in Box 1. The method chosen will need to be acceptable to the patient and informed consent should be obtained. More than one method might be used to debride the wound.

Surgical and sharp debridement Surgical and sharp debridement methods are the fastest and most effective way to remove debris and necrotic tissue. Surgical debridement will usually be carried out in a hospital environment by a surgeon. Nurses, however, are increasingly undertaking wound debridement in the hospital and community setting. It is essential that they have the necessary training in debridement and the equipment to perform the procedure, as well as a trust policy or guidelines. Following sharp debridement, a secondary debridement technique is usually necessary, such as autolytic or larval therapy. A list of recommendations for effective sharp debridement is outlined in Box 2.

Enzymatic This involves the application of proteolytic and other exogenous enzymes to the wound surface, such as variadase and collagenase (Tong 2000). These enzymes break down necrotic tissue when used within a moist wound healing environment. In the UK they are less favoured for wound debridement. If used, nurses need to be aware that these products are inactivated by heavy metals such as silver, which are present in other wound care products. It is essential that the manufacturer’s instructions are followed.

Autolytic This occurs naturally to some extent in all wounds. It is a highly selective process involving macrophages and endogenous proteolytic enzymes that liquify and spontaneously separate necrotic tissue and eschar from healthy tissue. This is often seen, particularly in necrotic digits (Figure 1). Moist interactive wound dressings also contribute to autolytic debridement, as they create an optimal environment for debridement by phagocytic cells and help to create an environment capable of liquifying slough and promoting granulation (Kennedy and Tritch 1997, Levenson 1996).

Larval The value of the Lucilia sericata larvae (the greenbottle fly) in the treatment of infected or necrotic wounds has been recognised for centuries.
and numerous reviews have been published that describe the history and use of this technique (Knowles et al. 2001, Thomas 1996, Thomas et al. 1999a, Trudgian 2002). Larval therapy has the ability to remove necrotic and sloughy tissue rapidly because of secretion by the larvae of a number of proteolytic enzymes. There are also indications that larval therapy might influence the bacterial burden within the wound, and should be considered for debridement of infected necrotic wounds (Thomas et al. 1999b).

In the UK, sterile larvae can be purchased from the Surgical Materials Testing Laboratory at Bridgend, Wales and delivered the following day. The skin surrounding the wound is protected by a hydrocolloid dressing before the larvae are carefully placed in the wound. A close-net dressing is supplied to cover the maggots. This is secured with tape before applying any secondary dressings or padding. Therapy can be repeated as necessary, depending on the amount of necrotic tissue present. There are, however, certain aesthetic factors to be considered and not all patients are happy to have this form of treatment. The role of the nurse is to ensure that the patient has given informed consent and to ensure that other nurses in the team have the required knowledge and skills in the technique and that they are comfortable with carrying out care. Box 3 outlines the indications for use of larvae.

**Mechanical** This is the use of physical force to remove sloughy or necrotic tissue. Saline wound-irrigation or the use of aerosol cans of saline under pressure can achieve a degree of mechanical debridement. Applying wet packs or saline-soaked gauze and allowing it to dry out so that sloughy tissue is removed at dressing change is perhaps the most traditional form of mechanical debridement. However, this method is painful for the patient and is not recommended by the National Institute for Clinical Excellence (NICE 2001). Other methods include ultrasound therapy (Nussbaum et al. 1994).

The elimination or reduction of exudate is an important part of wound management and wound bed preparation. Even when granulation tissue is present in the wound, it will not heal if there are copious amounts of exudate (Falanga 2000). Exudate from chronic wounds has been shown to be detrimental to wound healing as it leads to the breakdown of extracellular matrix proteins and growth factors, and the inhibition of cell proliferation (Ennis and Meneses 2000, Falanga et al. 1994).

Excess wound exudate can also lead to maceration of the surrounding skin and is a culture medium for bacterial growth. For the nurse, wound exudate can be managed directly – by the use of absorbent dressings, for example, alginates or foams, compression therapy, and mechanical devices such as vacuum-assisted closure (VAC) (Ballard and Baxter 2000); or indirectly – by alleviation of the underlying cause of excess fluid, which might be due to bacterial colonization oedema. The nurse needs to ensure that the underlying cause has been identified before deciding on direct management. An example of this is excess exudate associated with venous ulceration (Figure 3), which should be managed by the use of compression therapy (RCN 1999). For other wounds, such as pyoderma gangrenosum which is an inflammatory type of ulcer, the use of immunosuppressives or steroids will be required to control inflammatory exudate (Figure 4).

**Management of wound exudate**

All chronic wounds contain bacteria and routine wound swabbing is not recommended (Gilchrist 1996). The role of the nurse is to recognise the factors associated with increased risk of infection (Box 4) and

### Box 3. Indications for the use of larvae
- Necrotic or infected wounds
- Where sharp debridement might expose bone or joint
- Where autolytic debridement has failed or was contraindicated
- Before skin grafting

### Box 4. Factors associated with increased risk of infection
- Age
- Obesity
- Smoking
- Presence of infection at another site
- Severity and number of concurrent diseases
- Other factors, for example, poor nutrition, vascular disease, diabetes mellitus, immunosuppressive therapy or presence of necrotic tissue at the wound bed
Correcting cellular dysfunction and restoring biochemical balance

During the normal process of wound healing, a series of rapid increases in specific cell populations is responsible for preparing the wound for repair, deposition of the new extracellular matrix and eventually complete wound closure. This process is, however, impaired in chronic wounds. There is evidence that cells within chronic wounds become senescent (old) and are unable to respond to messengers such as cytokines and growth factors (Agren et al 1999). Falanga (2000) suggests that the epidermis in chronic wounds fails to migrate across the wound tissue, and that there is widespread hyperproliferation associated with chronic wounds, which delays the healing.

The nurse cannot determine this aspect of wound bed preparation by clinical examination alone. However, the nurse’s role is to recognise when wounds are not progressing through the wound healing process and refer the patient for biopsy or further therapies.

The other significant contribution that nurses can make is to recognise that a red wound does not always indicate a healthy wound, therefore wound classifications can be misleading.

Conclusion

Wound bed preparation is not a new concept and it incorporates the elements that constitute good wound management. It is not a one-off treatment, nor it is solely about debriding the wound. Nurses need to be aware of the many patient factors and wound bed factors that influence wound healing, and the many and varied methods of achieving a well-prepared wound bed.