

PRINCIPLES OF SKIN AND WOUND CARE: THE PALLIATIVE APPROACH

Jane McManus

The term 'palliative care' is used to describe care given to patients with advanced, life-limiting illness of any aetiology. It is a philosophy of care that is patient and family-centred, designed to meet the needs of the patient and family. Wound care for palliative care patients should be managed so that patient and family needs/concerns are the main focus of attention. Dressing products designed to heal acute wounds may not have the same effect on chronic, non-healing wounds. The palliative care goals of symptom control and psychosocial support can be transferred to palliative wound care for patients whose wounds will not heal. Nurses must become familiar with the concept of a stable non-healing wound when providing palliative wound care. This article will discuss the principles of wound management in relation to palliative care. *Declaration of interests: none*

KEY WORDS

Chronic wounds
Dry wound management
Moist wound management
Palliative wound care

The goals of palliative wound care include reducing pain, odour, exudate, bleeding and infection (McDonald and Lesage, 2006). Some authors have raised concerns with regard to the use of the term 'palliative wound care' as patients might be labelled 'palliative' if their wounds are too difficult or costly to heal, allowing 'palliative' to excuse poor outcomes (Ennis and Meneses, 2005).

It is important that patient comfort takes priority over preventing skin breakdown and care of the wound in palliative care (Langemo, 2006). However, when wounds and their symptoms worsen following implementation of measures designed

Jane McManus is Deputy Ward Manager; Rugby Ward, St Christopher's Hospice, London. Email: j.mcmanus@stchristophers.org.uk

to prioritise patient comfort, holding these opposing needs in balance becomes more challenging for clinicians and patients. This article will argue that current theories of moist wound management, with healing as the endpoint, are inappropriate for the needs of palliative patients with wounds (Grocott, 2005). It will also describe the principles of wound management and symptom control with regard to wounds commonly found in patients in the advanced stages of their disease.

Wound types

A wound is a breach in the epidermis or dermis resulting from trauma or pathological change that initiates a process of repair (Collins et al, 2002). An acute wound is a wound that occurs suddenly and has a short duration. Examples include surgical wounds and burns that heal easily with few complications (Dealey, 1999).

A chronic wound is a wound that remains unhealed for longer than 6 weeks, influenced by complex and multiple factors that impede healing (Collins et al, 2002). In chronic wounds, the pattern and timing of physiological and biochemical changes associated with healing are disrupted. Although chronic wounds have been defined as

'those that do not heal', and have been interpreted as the result of deficiencies in diagnosis or management, some chronic wounds appear resistant to all treatments aimed at them (Enoch and Price, 2004). Patients unable to eat a balanced diet or to digest and absorb nutrients, will be less likely to have a wound that heals and remains healed. Examples of commonly occurring chronic wounds in palliative care include pressure ulcers and fungating or malignant wounds.

Pressure ulcers

A pressure ulcer is an area of local necrosis developing when soft tissue is compressed between a bony prominence and a rigid external surface (McGrath and Breathnach, 2004). The mean capillary blood pressure in the skin of healthy individuals is 25–30mmHg. Damage to the subcutaneous tissue can occur after both prolonged exertion of pressure and shorter periods of high pressure (Langemo, 2006; Langemo and Brown, 2006).

Any severely ill patient may develop pressure ulcers. Immobility and prolonged pressure on a body part is the major risk factor, although reduced sensory perception, older age and neurological disability are also

important factors (Reifsnnyder and Magee, 2005) (Figure 1). Dehydration and hypotension compound tissue damage. Further risk factors include general ill health, ischaemic heart disease, peripheral vascular disease, raised body temperature, incontinence and poor nutritional state, especially hypoalbuminaemia and low vitamin and zinc levels (Dealey, 1999). Drugs that suppress sensation, mobility or blood flow and skin strength are also aggravating factors (McGrath and Breathnach, 2004), e.g. steroids (Dealey, 1999) and vasoactive drugs often used in cardiac care (Papantonio et al, 1994).

The occurrence of pressure ulcers varies according to patient group and care setting. One study of patients receiving home hospice care in the USA highlighted increased incidence of pressure ulcers in patients who had a history of pressure ulcers, were older, had a diagnosis of cancer, central nervous system disorders or dementia and had lower Karnofsky palliative performance scores (Reifsnnyder and Magee, 2005).

The prevalence of pressure ulcers in palliative care ranges from 13–47% (Langemo, 2006). A study in a UK hospice found a 24% prevalence of pressure ulcers (Bale et al, 1995). Research using an audit cycle to reduce pressure ulcer incidence in a UK hospice found the incidence did not reduce over 2 years. The author concluded that pressure damage at the end of life may be inevitable (Galvin, 2002). This has been termed 'skin failure' (Langemo and Brown, 2006).

Malignant or fungating wounds

A malignant or fungating wound occurs when tumour invades the epithelium and breaks through the skin surface (Dealey, 1999). The wound may either be ulcerative or proliferative, meaning that the wound forms ulcerating craters or raised, cauliflower-like nodules (Bridel-Nixon, 1997; Naylor 2001). Malignant wounds are commonly seen in breast and head/neck cancers (Naylor, 2002b). They also occur in cancers of the skin, vulva and bladder (Dealey, 1999). Fungating wounds

develop at the site of the primary cancer and also at affected lymph nodes of the axilla and groin (Dealey, 1999). A malignant wound is unlikely to improve, even if radiotherapy, chemotherapy or surgery offer short-term symptom reduction, because cancer cells continue to grow (Figure 2).

Bridel-Nixon (1997) notes that there is a dearth of published research on fungating wounds. Most articles discuss single cases. The UK incidence and prevalence is difficult to determine, as national cancer registries do not record this information (Thames Cancer Registry, 2005). Few published



Figure 1. A grade 4 sacral wound of a 38-year-old woman admitted to the hospice with cervical cancer. (The raised area in the middle of the wound is her coccyx.) The goals of care were to manage her pain levels, cushion the coccyx and prevent infection. The wound continued to deteriorate due to her condition but she was kept comfortable with a dressing regime of diamorphine mixed with Intrasite gel, Mepilex and Mefilm.

studies discuss the extent of the problem (Bridel-Nixon, 1997). The incidence of malignant wounds in patients with breast cancer appears to be between 2 and 5% (Fairbairn, 1993; Haisfield-Wolfe and Rund, 1997; Grocott, 1999).

A 10-year prevalence study at a cancer registry in the USA revealed that 367 (5%) of 7316 patients had cutaneous malignancies, of which 38 had wounds resulting from direct local invasion, 337 had metastatic wounds, and eight had both (Lookingbill et al, 1990). Malignant wounds affect a small group of people but provide major challenges that will be discussed.

How fungating wounds develop and the problems they generate is determined by a combination of factors. As cancerous cells multiply, blood and lymph vessels distort, affecting the flow of blood and lymph. This disrupts haemostasis, alters lymphatic, interstitial and cellular environments, causing tissue hypoxia and necrosis. This encourages infection by organisms that thrive in dead tissue. Eventually, blood vessels may be eroded by tumour, causing bleeding (Bridel-Nixon, 1997).

Wound management theories

Moist wound healing

Modern wound healing theory developed from the work of Winter (1962, 1963). Winter (1962) examined the rate of epithelialisation in experimental wounds cut into the skin of healthy pigs, comparing wounds with a natural scab exposed to the air against wounds that were covered with polythene film. He found that epithelialisation occurred more quickly in the latter: In exposed wounds, epidermal cells migrated from hair follicles and the wound edges, whereas in covered wounds, epidermal cells migrated through serous exudate, forming a new epidermal layer above the dermis (Winter, 1962).

The principle of moist wound healing led to the development of the first 'scientific' wound dressings



Figure 2. A fungating breast wound of a 75-year-old woman. The patient had refused a wound dressing, and made the wound bleed by picking at it regularly. She felt that the bleeding was indicative of the wound 'cleansing itself' so that it could heal. She refused to accept that the wound was composed of cancer cells and that it was unlikely to heal.

to support optimal healing processes that have revolutionised wound management (Benbow, 2005). These products include hydrogels to retain/bring moisture to the wound, hydrocolloids to absorb small amounts of excess moisture without drying the wound bed, absorbent foams, alginates, adhesive dressings, non-adhesive dressings and silicone-based low-adherent dressings.

Winter's (1962) research focused only on acute, superficial wounds, but the results have been used to generate theory of moist wound healing for all types of wound of varying aetiologies. Moist wound healing has become the gold standard of current clinical care and product development. However, the theory of moist wound healing does not provide a basis for satisfactory management of every wound seen in palliative care practice. Whilst a moist environment at the wound site has been shown to aid the rate of epithelialisation in superficial wounds (Eaglstein et al, 1988; Agren et al, 2001; Parnham, 2002), excess moisture at the wound site also causes maceration of the periwound skin (Cutting and White, 2002). It is clear that wounds need a *balanced* level of moisture to heal.

Principles of wound bed preparation

Another contemporary approach to wound healing is 'wound bed preparation'. This involves priming the wound to create optimal conditions for healing and removing factors that impede healing such as necrotic tissue, infection and exudate (Falanga, 2000). Necrotic tissue needs to be removed (debrided) from the wound. This occurs naturally (autolysis), conducted by phagocytic cells in the wound bed, but can be accelerated by the use of enzymes, sterile maggots, Manuka honey or surgical techniques that cut away dead tissue (Romanelli and Mastronicola, 2002). Historically, debridement was considered a treatment to be given once for each wound, unless more necrotic tissue formed.

Following debridement, the wound would be expected to heal (Dealey, 1999). However, in chronic

wounds, where the healing process has faltered, necrotic tissue may recur. The acute wound debridement model must be tailored to fit chronic wound pathophysiology. Repeated debridement of necrotic tissue is likely to be necessary and has been termed 'maintenance debridement' (Falanga, 2000).

The damaging properties of chronic wound exudate have already been outlined. Managing exudate is a fundamental principle of wound bed preparation. However, containing large volumes of fluid in an effective, reliable and acceptable wound dressing over a wide variety of body locations remains an unmet challenge for dressing manufacturers (Grocott, 2000).

Moisture balance

A more recent concept of moist wound healing is 'moisture balance' (Bishop et al, 2003). This theory proposes that a balance must be struck between excessive wound fluid, particularly in chronic wounds which can cause maceration of the periwound skin destroying some of the beneficial processes occurring in the wound, and a wound surface exposed to air which then forms a dry, hard scab that can delay healing (Winter and Scales, 1963). Despite vast amounts of research undertaken, the optimal level of wound exudate, allowing healing, remains unestablished (Cutting, 1999).

Bishop et al (2003) propose that wound dressings need to be designed for moisture balance. Dressings must absorb and contain exudate away from the wound surface, while ensuring that the wound surface remains moist. These components should be available as a single dressing or a simple dressing system that is secure and effective. This theory and the associated features of dressings designed for exuding chronic wounds is appropriate where the wound has the potential for healing. However, in situations where healing is unlikely, such as malignant wounds and in patients with a short prognosis, the exudate needs to be managed in such a way that causes minimal impact upon the patient. If the wound surface can be dried to slow the rate and volume of

exudate produced, some reduction of the discomfort and distress associated with very wet wounds may be achieved.

Dry wound management: an alternative to moist wound healing in palliative care?

In palliative wound care, a wound that is maintained with a dry scab, allowing the wound bed underneath to remain dry (Winter and Scales, 1963), enables a patient with a prognosis too short to allow a wound to heal to have a wound managed without a complex dressing regime to absorb exudate, prevent maceration, odour and infection. If it were possible for patients who have dry wounds to have the dry scab maintained and protected without a dressing that creates a moist wound environment, dry wound management would be a viable alternative in palliative wound care.

Goals of chronic wound management at the end of life need to shift towards relieving suffering, maintaining function and enabling the patient to engage in activities that are important to them (Enoch and Price, 2004). This requires research to determine how palliative wounds can be managed to fulfil these goals without using healing as the endpoint of research study. There has been some interest in scabs that form naturally on exposed wounds. There is a need to investigate their properties to understand their role in wound healing and how that may be integrated into current wound management research (Nelson, 1995).

Wound risk assessment at the end of life

Regular assessment of patients and their skin is key to maintaining skin integrity or reducing skin damage. There are many risk-assessment tools available to measure patients' relative risk of pressure ulcer development. Although they are used in a wide variety of care settings, few have been validated for palliative care. For example, the Braden Scale, a six-part scale assessing risk of pressure ulcers, is commonly used in long-term elderly care in the USA (Van Rijswijk and Lyder, 2005). The Hunter's Hill tool, a variation of the Braden Scale, was developed at a hospice in

Scotland (Chaplin, 2000). While it has not been the subject of published validation research, consensus among nurses at the author's workplace is that it provides a broad guide to a patient's relative risk.

The relative importance of various known risk factors is not known (Lyder, 2005), so cannot be prioritised in the care of the palliative care patient. *Table 1* outlines wound risk-reduction strategies for end-of-life care.

Effects and symptoms of chronic wounds in palliative care

The presence of a wound has consequences for the patient, his/her family and health care providers. There are also financial consequences of managing a wound. As has been shown above, wounds occur in some patients as one of the consequences of advanced disease of all aetiologies. Studies have assessed the symptoms experienced by patients in the last year of life and wounds contribute to a significant number of these symptoms. One well-known study is the Regional Study of Care for the Dying, which found that 28% of dying people had 'bedsores' in the last year of life and for 55% of those it was 'very distressing' (Addington-Hall and McCarthy, 1995).

In a Danish qualitative study into the impact of malignant breast wounds on the femininity, sexuality, emotions and feelings of 12 women with advanced breast cancer, the wounds and symptoms of malodour and exudate caused the women to feel less feminine and less self-confident (Lund-Nielsen et al, 2005). When wounds in palliative care are assessed, the assessment should include psychological assessment of the patient and his/her family. Care must focus on the maximisation of coping strategies. Patients often express their needs as fears (Langemo, 2006).

Exudate

When the skin is injured causing a wound, fluid accumulates more readily in the wounded tissues. Histamine is released from wounded cells causing plasma to leak from blood vessels, resulting in local oedema (Thomas,

1997). It becomes more difficult for interstitial fluid pressure to be maintained, affecting the control of fluid in blood and lymph systems (Bishop et al, 2003). Granulation tissue, which may be present in the wound, can absorb more water than normal skin tissue, also increasing tissue fluid accumulation at the wound site.

Inflammation, which occurs as part of the post-injury process, results in dilation of capillaries, increasing their permeability to fluid (Dealey, 1999). An excess of leaking fluid from the wound is known as exudate. It is normally a pale, straw-coloured, watery fluid, which becomes more viscous and opaque because of leucocytes, albumin,

Table 1

Risk-reduction strategies for end-of-life skin and wound care

Skin care

Where skin is fragile and at risk of breakdown, keeping skin clean and dry is paramount. Emollients moisturise and maintain a balanced skin pH (Dealey, 1999). Soap is drying to the skin and should be discouraged (Langemo, 2006).

Maceration

Maceration decreases the ability of the skin to withstand pressure, shearing or friction (Langemo, 2006). Incontinence of urine or faeces makes skin maceration more likely (Dealey, 1999). Urinary incontinence increases the hydration of the skin, making it more permeable, reducing its barrier functioning and increasing its pH. Faecal incontinence contributes to skin damage because enzymes in the faeces convert urea in urine to ammonia, irritating the skin further (Van Rijswijk and Lyder, 2005).

Immobility

Regular repositioning of the body to redistribute pressure is essential but challenging when the patient has symptoms of pain, nausea or dyspnoea and cannot tolerate many variations in position. Repositioning must be balanced against the patient's holistic needs.

Friction and shear forces

Whilst elevating the head of the bed may help a patient who has dyspnoea, increased pressure on blood vessels and the skin increases the risk of skin damage (Langemo, 2006). Methods of protecting the skin include protective film or hydrocolloid dressings covering bony prominences (Langemo, 2006). However, dressings can cause pain and damage on removal and, if not transparent, can make skin observation difficult.

Patient support surfaces

Specialist mattresses and seat cushions that distribute pressure more evenly across the body surface in contact with the product are recommended for patients at risk of pressure damage, although their use must be balanced against comfort at the end of life. There is a lack of evidence to demonstrate that a particular seat cushion performs better than any other, or that low-tech pressure-relieving mattresses are more effective than high-specification foam mattresses at reducing pressure damage (National Institute for Health and Clinical Excellence, 2003).

Nutrition and hydration

As patients become weaker and less alert, they are often unable to maintain their oral intake. Additionally, as a result of symptoms or the medication to manage them, absorption of nutrients is often reduced, increasing the risk of skin damage and delaying skin healing (McLaren, 1997).

macrophages and cell debris (Cutting and White, 2002). Exudate volume has traditionally been described using the subjective terms of 'light', 'moderate' or 'heavy' (Cutting, 1999). This subjectivity may hinder appropriate dressing selection as imprecise assessments will result in dressings being chosen on a 'best guess' basis. Dressings may not perform effectively as a result of inappropriate selection rather than inferior dressing properties.

The nature of acute wound exudate has been found to be substantially different to that of chronic wound exudate. The difference lies predominantly in the presence of tissue-destructive enzymes (proteinases) in the latter (Tregrove et al, 1999). These proteinases serve a useful purpose in wound healing in small amounts (Bishop et al, 2003), but damage chronic wounds and their surrounding skin (Barrick et al, 1999). They contribute directly to chronic wound enlargement (Cutting and White, 2002). Periwound skin is known to be at increased risk of damage compared to healthy skin as it is affected by tissue inflammation (Walker et al, 1997). Chronic wound exudate poses serious problems.

There is a growing body of research evidence suggesting that it is not excessive fluid at the wound, per se, that causes healing problems and maceration, rather that it is the nature of the fluid and its components that cause problems (Bishop et al, 2003). Breuing et al (1992) used special 'wound chambers' on the skin to maintain a wet environment on the wound using saline, demonstrating that healing was unimpaired and possibly improved, with no maceration and reduced scarring, compared with uncovered wounds. Other work has shown that moist or wet wounds heal as effectively as each other compared with a dry environment (Vogt et al, 1995), so long as the composition of the exudate is not detrimental to the wound itself.

The use of moist wound healing can be problematic in some clinical

situations, in particular wounds that are being managed at the end of life and are not going to heal, where the volume of moisture produced by the wound is absorbed with varying success by dressing products. In patients with advanced disease who develop wounds, healing is often unrealistic. For these patients, the moist wound environment may be a disadvantage. The moisture must be contained using dressing products, which provide a good fit that does not leak (Grocott, 2000). Dressings require regular reapplication. The serous fluid, proteins and sugars present in the moist wound provide a growing medium for microbes that compound the difficulties of managing non-healing wounds.

Odour

Malodorous wounds can be very distressing for the patient. Patients' relationships may be affected (Douglas, 2001; Lund-Nielsen et al, 2005), contributing to social isolation (Douglas, 2001; Naylor, 2002a). Wound odour is produced by bacteria that are present in necrotic tissue of the wound (McDonald and Lesage, 2006). In a wound that is expected to heal, debridement of the necrotic tissue is a priority (McDonald and Lesage, 2006). In fungating wounds, debridement must be done carefully using non-surgical techniques, as the wounds are likely to be friable and have a tendency to bleed (Naylor, 2002a).

The use of systemic and topical antibiotics (McDonald and Lesage, 2006), as well as silver dressings as an antimicrobial, charcoal dressings to bind the odour molecules, and topical honey (Naylor, 2002a), may assist in the management of odour. Any creams or ointments applied topically may increase the volume of exudate produced, which may cause further wound management problems.

Infection

Not all chronic wounds will be infected with micro-organisms, but they will all contain microbes. The point at which microbial colonisation becomes problematic for the

wound depends on the numbers and virulence of the microbes and the extent of the immune response triggered (Cutting and Harding, 1994). There are four categories of microbial presence in a wound: contamination, colonisation, critical colonisation and infection. Infection of a chronic wound produces an enhanced and prolonged inflammatory response (Romanelli and Mastronicola, 2002). This causes more damage to the wound and reduces the immune response of the patient (Dow and Ronald, 1994). This masks some of the symptoms of chronic wound infection (Romanelli and Mastronicola, 2002), making the presence of infection less obvious and, therefore, less likely to be investigated, diagnosed and treated.

A recently discovered element of chronic wound infection is 'biofilm' (Sibbald et al, 2000). Bacteria exist in wounds in microcolonies, which attach to the wound bed and secrete a biochemical liquid that helps to protect them from antiseptics and antibiotics (Davey and O'Toole, 2000).

The presence of infection or critical colonisation will prevent a wound from healing (Romanelli and Mastronicola, 2002); therefore, reducing microbial numbers to minimise their impact is an important component of encouraging a chronic wound to heal. Additionally, symptoms of odour, exudate and pain can be reduced if the microbial cause is treated (Naylor, 2005).

Pain

Many patients with pressure ulcers experience moderate to severe pain during wound care, dressing changes and debridement. Dressing removal is often the most painful part of the wound management regime (Langemo, 2006). Analgesia should be given systemically or topically before the dressing procedure (Naylor, 2001). Sensitive psychological care, i.e. relaxation, music, distraction and gentle conversation, may also help pain management (Langemo, 2006). Pain is limited by the use of dressings that are minimally traumatic to remove and cleansing of the wound by gentle

irrigation with warmed normal saline (Langemo, 2006). There is a lack of data about the pain experienced by patients with fungating wounds, although one small study ($n=13$) found that 38% of patients with fungating wounds experienced pain (Haisfield-Wolfe and Baxendale-Cox, 1999).

Dressing fit problems

Dressings that have been developed in response to Winter's (1962, 1963) theory maintain moist conditions by ensuring low moisture-vapour loss from the wound. They tend to have a poor capacity to manage large volumes of exudate satisfactorily. They exhibit problems with fit and fixation to a moving body (Grocott, 2000). A radical revision of dressing products in terms of size, presentation, thickness of materials and fixation methods is needed in the light of substantial case study evidence of exuding wounds in palliative care (Grocott, 2000; Seaman, 2006). Evidence from dressings research for heavily exuding wounds demonstrates the benefits of a two-layer dressing system, with the primary contact layer drawing exudate from the wound into a secondary absorbent layer (Grocott, 2000).

Alternative endpoints in palliative care

The goals in palliative care of symptom control and psychosocial support can be transferred into wound care for palliative patients whose wounds will not heal. This concept of a stable, non-healing wound as a goal of care is relatively new (Ennis, 2001). However, it is important that, if healing stops being the goal and 'palliative wound care' becomes the focus, palliation follows careful, holistic assessment of the wound and patient, so that it does not become the easier or cheaper option for resource-poor health services (Ennis and Meneses, 2005).

Alternative treatment approaches may prove beneficial to relieve suffering and manage symptoms (Enoch and Price, 2004). Dressing products that are designed to heal acute wounds may not have the same effect on chronic, non-healing wounds (Enoch and Price, 2004).

Conclusion

Wounds are a symptom of advanced disease, as demonstrated in symptom prevalence studies in palliative care. Wound incidence and prevalence data are not commonly available. The data that exist come mainly from small-scale surveys predominantly of pressure ulcers. Chronic wounds in palliative care are mainly of the pressure ulcer and malignant type.

Modern wound healing theory developed from the work of Winter (1962, 1963) who demonstrated faster epithelialisation in superficial pig wounds. Winter's theory has been translated to the management of all wounds at all stages of healing. Moist wound healing is the gold standard of current clinical care and product development. Two theories have evolved from moist wound healing: wound bed preparation and moisture balance. The risks of developing wounds have been widely researched and risk-assessment tools have been formulated, although their validity in palliative care is not established. Effective palliative wound care relies on attention to maceration, friction and shear forces, nutrition and hydration, as well as a focus on patient mobility and the support surfaces they are nursed on, although the relative risks of each factor are not known.

Evidence shows that the effects of chronic wounds are physical (exudate, malodour, infection, pain and dressing fit problems), psychological, emotional and social. In palliative care, alternative surrogate or intermediate endpoints, rather than wound healing, may be appropriate goals, with patient comfort and choice being key to success. The inappropriateness of moist wound healing in palliative care suggests the need for alternative practices. **EOLC**

References

- Addington-Hall J, McCarthy M (1995) Dying from cancer: results of a national population-based investigation. *Palliative Medicine* 9(4): 295–305
- Agren MS, Karlsmark T, Hansen JB, Rygaard J (2001) Occlusion versus air

- exposure on full-thickness biopsy wounds. *Journal of Wound Care* 10(8): 301–4
- Bale S, Finlay I, Harding KG (1995) Pressure sore prevention in a hospice. *Journal of Wound Care* 4(10): 465–8
- Barrick B, Campbell EJ, Owen CA (1999) Leukocyte proteinases in wound healing: roles in physiologic and pathologic processes. *Wound Repair and Regeneration* 7(6): 410–22
- Bishop SM, Walker M, Rogers AA, Chen WY (2003) Importance of moisture balance at the wound-dressing interface. *Journal of Wound Care* 12(4): 125–8
- Benbow M (2005) *Evidence-based Wound Management*. Whurr Publishers, London
- Bridel-Nixon J (1997) Other chronic wounds. In: Morison M, Moffatt C, Bridel-Nixon J, Bale S, eds. *The Nursing Management of Chronic Wounds*. 2nd edn. Mosby, London: 221–44
- Breuing K, Eriksson E, Liu P, Miller DR (1992) Healing of partial thickness porcine skin wounds in a liquid environment. *Journal of Surgical Research* 52(1): 50–8
- Chaplin J (2000) Pressure sore risk assessment in palliative care. *Journal of Tissue Viability* 10(1): 27–31
- Collins F, Hampton S, White R (2002) *A–Z Dictionary of Wound Care*. Quay Books, London
- Cutting KF (1999) The causes and prevention of maceration of the skin. *Journal of Wound Care* 8(4): 200–1
- Cutting KF, Harding KG (1994) Criteria for identifying wound infection. *Journal of Wound Care* 3(4): 198–201
- Cutting KF, White RJ (2002) Maceration of the skin and wound bed. 1: Its nature and causes. *Journal of Wound Care* 11(7): 275–8
- Davey ME, O'Toole GA (2000) Microbial biofilms: from ecology to molecular genetics. *Microbiology and Molecular Biology Reviews* 64(4): 847–67
- Dealey C, ed (1999) *The Care of Wounds: A Guide for Nurses*. 2nd edn. Blackwell Science, London
- Douglas V (2001) Living with a chronic leg ulcer: an insight into patients' experiences and feelings. *Journal of Wound Care* 10(9): 355–60
- Dow G, Ronald A (1994) Skin tests. In: Hoeprich PD, Jordan MC, Ronald AR, eds. *Infectious Diseases: Treatise of Infectious Processes*. JB Lippincott, Philadelphia
- Eaglstein WH, Davis SC, Mehle AL, Mertz PM (1988) Optimal use of an occlusive dressing to enhance healing. Effect of delayed application and early removal on wound healing. *Archives of Dermatology* 124(3): 392–5
- Ennis WJ (2001) Healing: can we? Must we? Should we? *Ostomy Wound Management* 47(9): 6–8

Ennis WJ, Meneses P (2005) Palliative care and wound care: two emerging fields with similar needs for outcome data. *Wounds* 17(4): 99–104

Enoch S, Price P (2004) Should alternative endpoints be considered to evaluate outcomes in chronic recalcitrant wounds? *World Wide Wounds*. www.worldwidewounds.com/2004/october/Enoch-Part2/Alternative-Endpoints-To-Healing.html (last accessed 2 March 2007)

Fairbairn K (1993) Towards better care for women: understanding fungating breast lesions. *Professional Nurse* 9(3): 204–8

Falanga V (2000) Classifications for wound bed preparation and stimulation of chronic wounds. *Wound Repair and Regeneration* 8(5): 347–52

Galvin J (2002) An audit of pressure ulcer incidence in a palliative care setting. *International Journal of Palliative Nursing* 8(5): 214–21

Grocott P (1999) The management of fungating wounds. *Journal of Wound Care* 8(5): 232–4

Grocott P (2000) The palliative management of fungating malignant wounds. *Journal of Wound Care* 9(1): 4–9

Grocott P (2005) Commentary: A guide to wound management in palliative care.

International Journal of Palliative Nursing 11(11): 579

Haisfield-Wolfe ME, Baxendale-Cox LM (1999) Staging of malignant cutaneous wounds: a pilot study. *Oncology Nursing Forum* 26(6): 1055–64

Haisfield-Wolfe ME, Rund C (1997) Malignant cutaneous wounds: a management protocol. *Ostomy Wound Management* 43(1): 56–66

Langemo DK (2006) When the goal is palliative care. *Advances in Skin and Wound Care* 19(3): 148–54

Langemo DK, Brown G (2006) Skin fails too: acute, chronic and end-stage skin failure. *Advances in Skin and Wound Care* 19(4): 206–11

Lookingbill DP, Spangler N, Sexton FM (1990) Skin involvement as the presenting sign of internal carcinoma. A retrospective study of 7316 cancer patients. *Journal of the American Academy of Dermatology* 22(1): 19–26

Lund-Nielsen B, Muller K, Adamsen L (2005) Malignant wounds in women with breast cancer: feminine and sexual perspectives. *Journal of Clinical Nursing* 14(1): 56–64

Lyder CH (2005) Is pressure ulcer care evidence based or evidence linked? *Journal of Wound Care* 14(6; Suppl): 2–3

McDonald A, Lesage P (2006) Palliative management of pressure ulcers and malignant wounds in patients with advanced illness. *Journal of Palliative Medicine* 9(2): 285–95

McGrath JA, Breathnach SM (2004) Wound healing. In: Burns T, Breathnach S, Cox N, Griffiths C, eds. *Rook's Textbook of Dermatology*. 7th edn. Blackwell Science, Oxford

McLaren S (1997) Nutritional factors in wound healing. In: Morison M, Moffat C, Bridel-Nixon J, Bale S, eds. *The Nursing Management of Chronic Wounds*. 2nd edn. Mosby, London: 27–51

National Institute for Health and Clinical Excellence (2003) *Pressure Ulcer Prevention. Clinical Guideline 7*. NICE, London

Naylor W (2001) Assessment and management of pain in fungating wounds. *British Journal of Nursing* 10(22; Suppl): S33–S40

Naylor W (2002a) Part 1: Symptom control in the management of fungating wounds. *World Wide Wounds*. <http://www.worldwidewounds.com/2002/march/Naylor/Symptom-Control-Fungating-Wounds.html> (last accessed 2 March 2007)

Naylor W (2002b) Malignant wounds: aetiology and principles of management. *Nursing Standard* 16(52): 45–53

Naylor WA (2005) A guide to wound management in palliative care.

International Journal of Palliative Nursing 11(11): 572–9

Nelson EA (1995) Moist wound healing: critique II. *Journal of Wound Care* 4: 370–1

Papantonio CT, Wallop JM, Kolodner KB (1994) Sacral ulcers following cardiac surgery: incidence and risks. *Advances in Wound Care* 7(2): 24–36

Parnham A (2002) Moist wound healing: does the theory apply to chronic wounds? *Journal of Wound Care* 11(4): 143–6

Reifsnnyder J, Magee H (2005) Development of pressure ulcers in patients receiving home hospice care. *Wounds* 17(4): 74–9

Romanelli M, Mastronicola D (2002) The role of wound-bed preparation in managing chronic pressure ulcers. *Journal of Wound Care* 11(8): 305–10

Seaman S (2006) Management of malignant fungating wounds in advanced cancer. *Seminars in Oncology Nursing* 22(3): 185–93

Sibbald RG, Williamson D, Orsted HL, Campbell K, Keast D, Krasner D, Sibbald D (2000) Preparing the wound bed — debridement, bacterial balance and moisture balance. *Ostomy Wound Management* 46(11): 14–35

Thames Cancer Registry (2005) *Cancer in South East England 2003*. Thames Cancer Registry, King's College London

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

Trengove NJ, Stacey MC, MacAuley S et al (1999) Analysis of the acute and chronic wound environments: the role of proteases and their inhibitors. *Wound Repair and Regeneration* 7(6): 442–52

Van Rijswijk L, Lyder CH (2005) Pressure ulcer prevention and care: implementing the revised guidance to surveyors for long-term care facilities. *Ostomy Wound Management April(Suppl):* S7–S19

Vogt PM, Andree C, Breuing K, Liu PY, Slama J, Helo G, Eriksson E (1995) Dry, moist and wet skin wound repair. *Annals of Plastic Surgery* 34(5): 493–9

Walker M, Hulme TA, Rippon MG, Walmsley RS, Gunnigle S, Lewin M, Winsey S (1997) In vitro model(s) for the percutaneous delivery of active tissue repair agents. *Journal of Pharmaceutical Sciences* 86(12): 1379–84

Winter GD (1962) Formation of the scab and the rate of epithelialisation of superficial wounds in the skin of the young domestic pig. *Nature* 193(4812): 293–4

Winter GD (1963) Effect of air exposure and occlusion on experimental human skin wounds. *Nature* 200: 378–9

Winter GD, Scales JT (1963) Effect of air drying and dressings on the surface of a wound. *Nature* 197: 91–2

Key Points

- ▶ Dressing products designed to heal acute wounds may not have the same effect on chronic, non-healing wounds.
- ▶ The principle of moist wound healing led to the development of the first 'scientific' wound dressings to support optimal healing processes.
- ▶ Moist wound healing may not be appropriate for palliative wound care.
- ▶ A radical revision of dressing products in terms of size, presentation, thickness of materials, and fixation methods is needed for the management of chronic, exuding wounds.
- ▶ Nurses must work towards alternative goals in palliative wound care; healing may not be the endpoint.