Role of the wet-to-dry phase of cleansing in preparing the chronic wound bed for dressing application

To achieve cleansing, fluid from a wrap is absorbed into the wound, resulting in the wrap becoming saturated with waste products and pathogens. Meanwhile, evaporation of excess moisture cools with the wound bed, reducing inflammation

cleansing; debridement; inflammation; infection

A delay in wound closure can result from poor perfusion, infection, pressure or chronic venous hypertension.[1] However, problem wounds are rarely affected by a single factor.[1] To facilitate wound healing, systemic and local factors that may impede healing need to be addressed.[1-8]

The University Hospital in Zurich, Switzerland have developed a wet-to-dry phase for wound cleansing prior to dressing application. This procedure promotes wound cleansing while also reducing inflammation. It was described by Kammerlander[9] in 1989 and has since been promoted by Wound Management Consulting, Wilhering/Linz, Austria. The wet-to-dry phase is based on empiric evidence[9] **please could you supply more references to support this - thanks** supported by more than 20 years of clinical practice.

The wound healing process

The model of 'normal' secondary wound healing has been much examined in both the laboratory setting and in practice. However, healing in chronic wounds is much more complex and is not yet fully understood.

Wound healing comprises a number of biochemical interactions and cascades, which in an acute wound appear to progress in a timely and ordered manner.[1,2,8,13-15] These interactions and cascades are influenced by several factors, such as the underlying pathophysiology and the presence of bacteria. These may up- or down-regulate the production of, for example, growth factors, enzymes, oxygen radicals and lactates.[8,10-12]

While it is known that disruptions to biochemical pathways in chronic wounds delay healing, there is debate as to the cause of and solutions to these problems.[2,4,8,14] For example, a wound may look 'healthy' and be granulating, yet does not heal. This could be due to non-viable (senescent) cells, or the presence in the wound bed of cells of the wrong phenotype. The cells are not responding to biochemical signals in a manner that is conducive to normal wound healing processes.[1-15]

Chronic wounds may demonstrate:[14]

- An enhanced number of pro-inflammatory cytokines
- A pathologically enhanced concentration of proteases
- Disturbed activity of growth factors
- Low concentration of tissue inhibitors of metalloproteinases (TIMPs)

Proteases

Studies investigating the contents of chronic and acute wound fluid have identified differences in their composition.[6-8,13-15] The major difference appears to relate to protease activity,[8] particularly the ability of chronic wound fluid to selectively inhibit cells that proliferate quickly.[8]

Fluid from acute wounds has been shown to stimulate cells in an *in vitro* environment.[6] In chronic wounds, however, the fluid components are unlikely to be in the correct concentrations at the appropriate time to support healing.[4,6-13,15-20] Some of these *in vitro* studies have used wound fluid from venous leg ulcers or post-surgical wounds.[6-8]

However, this area is still open to debate and significant further research is needed as there are many unanswered questions, such as which factors influence inflammation, and the mechanisms of the wound healing cascade are not yet understood, or are only partly understood.

The notion that chronic wound fluid contains elements that delay healing implies that maintaining this fluid in the wound over a period of time could delay healing. To support secondary closure, use of modern wound dressings that promote a moist enviroment by absorbing exudate in a controlled manner and keeping it separate from the wound bed is needed.[1,4,2]

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Matrix metalloproteinases

Matrix metalloproteinases (MMPs) play an important role in healing by:

- Remodelling connective tissue
- Enabling removal of non-vital tissue (autolytic debridement)
- Supporting epithelialisation.

In contrast to acute wounds, chronic wounds exhibit abnormally high levels of matrix metalloproteinases (MMPs),[16] and this imbalance disrupts healing.[16] Management strategies, therefore, include decreasing MMP levels to a concentration similar to that in normal healing wounds.

Tissue inhibitors of the MMPs (TIMPs) are the physiological opponents of MMPs. If their concentration is too small, the degradable characteristics of the MMPs predominate. The increased concentration of MMPs has a negative impact on local growth factors.[11,12]

Other factors that may delay healing

Chronic or problem wounds may develop due to an underlying disease such as chronic venous stasis, chronic lymphatic congestion, diabetes mellitus and arterial circulation disorders on a macro- or micro level.[4,9,13,16,17,22-30]

Bacterial burden and a necrotic layer will also compromise healing.[33-36] Chronic wounds that do not present signs of clinical infection are assumed to be colonised.

Creating an optimal wound environment

In most cases, providing an optimal healing environment — one that is free of debris and non-viable tissue[23,24,31] — and addressing the underlying disease will promote healing. Although there is no conclusive RCT evidence to support the notion that debridement plays a crucial role in effective wound closure,[9,50,51] it is widely accepted and has been practised for as long as there has been a systematic approach to wound treatment.

Necrosis

Tissue death, and thus necrosis, may occur because perfusion has been interrupted after injury. Necrotic tissue in a chronic wound presents morphologically as a mixture of non-vital cells, debris, exudate components (fibrin, collagen and elastin) as well as exogenous components (foreign bodies/contamination, particles left behind after dressing change and micro-organisms etc).[1,50,51]

The type of necrotic tissue present depends on the aetiology and influences the choice of debridement method.[1,23,33] It may present as a soft, permeable, whitish matter, or as a thick, black, leathery, rock-hard biological plug surrounded by an inflamed or healthy undisturbed tissue.[1,9]

Necrosis facilitates infection and the proliferation of bacteria, which produce 'toxins' that compete for oxygen and nutrients. The anaerobic environment within the devitalised tissue will decrease leucocyte movement and phagocytosis, and favour the growth of anaerobic organisms.[29,33,41] Leaving the eschar *in situ* extends and deepens the damage into neighbouring undamaged tissues, affecting the patient's local and general condition.[23,25]

Methods of debridement include surgical, biosurgical, enzymatic, mechanical and autolytic.[9,23,24,31,37] The choice of method relates to:

- The general goal of patient care
- The aetiology and nature of the wound and eschar
- Access to skilled health-care professionals
- Frequency of procedure
- Financial implications.[1]

The wet-to-dry phase

Wound cleansing and the application of dressings should be gentle but effective. Debridement and/or cleansing aims to:

- Reduce or remove foreign matter or devitalised, injured and infected tissue (eschar) if required alongside wound disinfection[50]
- Aid assessment of the extent of the injury when covered by devitalised eschar[1,51]
- Convert a necrotic wound into a clean acute wound[4,16,30,51]
- Protect the patient from micro-organisms and infection (local and systemic)
- Protect the wound edges and peri-wound skin from maceration and eczema.[9]

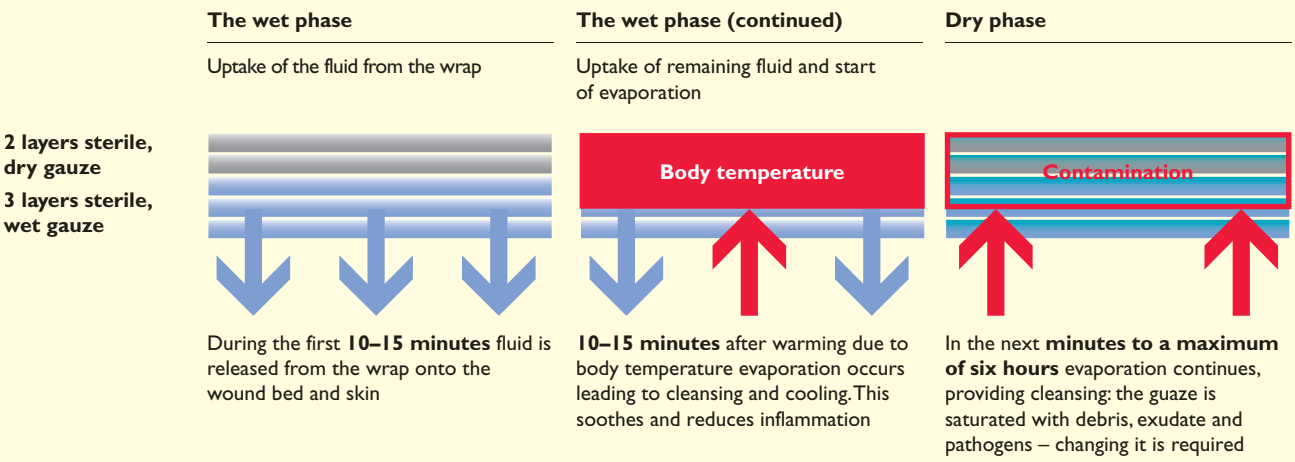
It is widely accepted that both dressings that promote a moist wound enviroment and active cleansing agents can be used to boost continuous physiological wound cleansing (autolytic debridement) if required.[38-49] This approach uses dressing changes as an active vechicle for wound cleansing.[9,36]

In 1989 Kammerlander[9] reported clinical experiences with a multi-phase concept for dressing changes, which involves an active cleansing phase (a wet phase lasting 15 minutes to one hour) and a short resting phase (the 'dry' phase) that restores the peri-wound skin integrity.[9]

The overall aim is not to create an optimal physiological wound environment or temperature (although excessive cooling is prevented), but to cleanse the wound and reduce itching and inflammation.[9] Patients have reported that the cooling phase that occurs as part of this process is soothing when extensive inflammation is present.[9]

During the first phase (the wet phase) fluid is donated from a moist wrap into the wound bed and the peri-wound skin for 15 minutes to one hour, depending on the level of inflamation. The amount of time for which the wrap is left on the wound depends on the objective: if standard superficial cleansing is required, where there is non-vital tissue ▶

Fig 1. The wet-to-dry phase



in the wound, debris or a combination of slough and debris, then it is 10–15 minues; if there is severe inflammation, then a longer period will be required. The wrap is held in place with a tubular bandage or a fixation bandage.

The body temperature causes some of the fluid to evaporate, initiating wound cleansing and cooling, which in turns helps reduce any inflammation. This method of cleansing is an alternative to rinsing.

The cleansing fluid used should be clinically effective and non-toxic. Examples include saline, Ringer's solution or specific wound cleansing fluids such as applicable antiseptics like povidone-iodine.[23,35,36] Ideally, the fluid should be lukewarm.[9]

If local infection is present, an antiseptic solution may be used[35] as it will be quickly activated and reduce the number of pathogens.[52-55] Iodine and octenidine have similar effects against vegetative bacteria, although octenidine is ineffective against spores and protozoa.[52] Iodine is better tolerated by tissue than a combination of octenidine and phenoxyethanol or preparations that contain chlorhexidine, but is less well-tolerated than polihexanide and taurolidine.[57-59]

In the next phase (the dry phase) the wound is covered with sterile dry gauze dressing for about 15 minutes, to restore the peri-wound integrity.

After this, the wound is covered with a modern wound dressing suitable for the wound type and healing phase. Evaporation will continue and the dressing will be saturated with debris, exudate and pathogens, and so will require changing.[9]

The wet-to-dry phase is illustrated in Fig 1.

Figs 2 and 3 illustrate the clinical application of wet-to-dry cleansing. A moistened carrier consisting of two or three layers of gauze or non-woven material may be used, as can a hydrofibre dressing.

Fig 4 shows a technique where the wound is kept

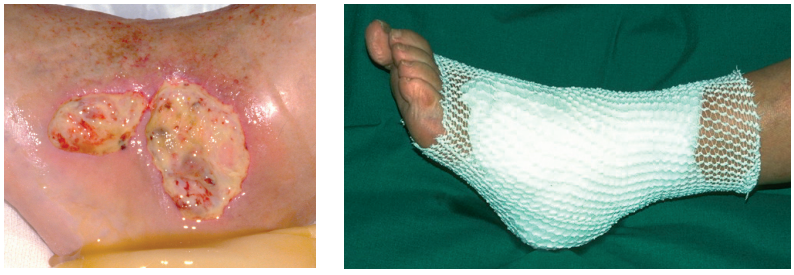


Fig 2. Clinical application of the wet-to-dry phase: a clinically infected leg ulcer (a); the wet wrap, which was fixed with a tubular bandage, was left in situ for 15 minutes (b)

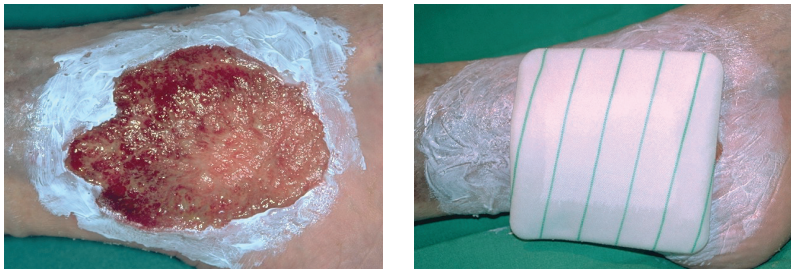


Fig 3. Clinical application of the wet-to-dry phase: venous leg ulcer (a); the ulcer is covered with a polyacrylate pad and the peri-wound skin is protected. The dressing is kept moist and stayed in place for 24 hours (b)

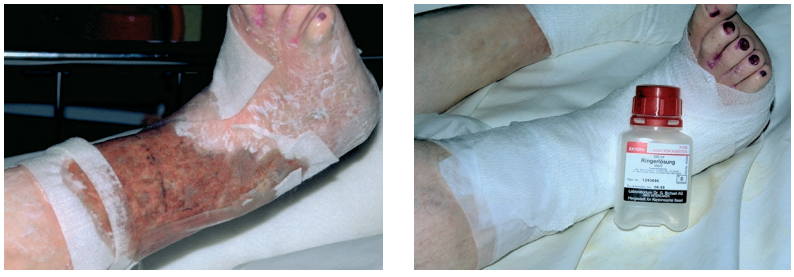


Fig 4. Clinical application of the wet-to-dry phase: the ulcer is covered with a moist hydrofibre dressing (a); the dressing is moistened with Ringers solution and secured with a fixation dressing (b)

▶

moist for a prolonged period. This may be required in specific cases such as dermatitis.

The venous leg ulcer shown in Fig 3 was of more than six months' duration. The ulcer bed and peri-wound skin showed signs of enhanced inflammation. The wound was kept moist with a polyacrylate pad, and the peri-wound skin was protected with a zinc cream. The dressing (applied in the final phase **which dressing? the wrap or the modern wound dressing?**) was left in place for 24 hours and moistened before application.

For a short cleansing phase, which can last for minutes to a maximum of one hour, the cleansing fluid may be applied at room temperature. McGuinness et al.,[60] in a study examining 44 patients and 133 dressing changes, found that wound cleansing with saline at 20°C (room temperature) did not have a significant influence on the temperature of the wound bed, which stayed at a mean of 27°C during cleansing (or the wet phase). Moreover, the dressing applied as a wound cover has little influence on wound bed temperature: on dressing removal the temperature was 32.6°C.

This suggests that the choice of carrier for a wet wrap does influence the temperature of the wound bed. Furthermore, the risk of cooling the wound too much when applying a rinsing fluid appears to be low. Body temperature is the key factor influencing wound bed temperature.[1,23,24]

Conclusion

Moist wound healing in chronic wounds requires that the wound bed be kept damp to enable cell movement, but not so wet as to delay healing or cause maceration.[1] Leaving a moist wrap *in situ* for more than 30 minutes will therefore protect the peri-wound skin.

The wet-to-dry phase for wound cleansing is thought to support the removal of non-vital tissue and exudate.[9] In addition, this method will help reduce a high bacterial burden if antiseptics are used. Reported clinical experience suggests that wound cleansing benefits from the application of wraps, using the wet-to-dry phase.[9]

Other advantages include a reduction in inflammation and that it aids treatment of wound infection. It is suggested that using wraps for cleansing may have advantages over just rinsing the wound as fluids used may take time to become activated.[1,5,9,24]

The wet-to-dry phase may be applied during dress-

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ing changes to reducte the bacterial burden and remove debris and exudate.

In cases of skin irritation, the method may be soothing and cooling, calming down the inflammation. Gauze moistened with saline may be replaced with modern moist wound healing dressings such as hydrofibres and polyacrylate pads moistened with Ringer's solution or a disinfectant. These carriers stay moist for a prolonged period of time, saving nursing time. ■

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