Wound hydration versus maceration: understanding the differences

KEY WORDS
- Debridement
- De-sloughing
- Devitalised tissue
- Hydration
- Hyper-hydration
- Maceration

This article provides an explanation and visual demonstration of the differences between the pathology and presentation of hydration versus maceration in wounds. This is described in order that the clinician can distinguish between the two and optimise wound treatment.

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All biological processes require water and it is essential for maintaining homeostasis (El-Sharkawy et al, 2015). Water is a universal solvent, a mediator of life's chemical reactions, and has a structure unlike that of any other liquid (Pohorille and Pratt, 2012). From the time that primeval species ventured from the oceans to live on land, a major key to survival has been the maintenance of hydration. Without water, humans can only survive for a few days. Once living organisms ventured from the oceans onto land, the uptake and retention of water were key to their survival. As a consequence, the development of a barrier (the skin) to water loss was important in order to prevent tissue desiccation of the organism. Water is also essential for the normal functioning and maintenance of healthy skin (Verdier-Sévrain and Bonté, 2007). Damage to the skin requires an immediate and coordinated repair response to prevent further damage to the organism in terms of fluid loss, pathogenic ingress and functional re-establishment (Rosińczuk et al, 2016). This healing process is initiated to, firstly, physically plug the wound and then to remodel the damaged tissue via a series of closely coordinated steps; ultimately leading to the restoration of the barrier and physiological process that the skin undertakes (Bíró and Harder, 2016). The hydration balance of the skin is crucial for its normal functioning and, once breached, the breakdown of the skin barrier and the exposure of the sub-epidermal structures to the external environment presents challenges to these tissues to maintain a balanced hydration level (Kruse et al, 2015).

HYDRATION AND THE HEALING PROCESS

Hydration is important to the wound healing process, this was elegantly demonstrated by George Winter who identified that wounds exposed to the air and allowed to dry healed poorly, but that wounds managed in a moist environment showed better healing (Winter, 1962, 1963; Winter and Scales, 1963; Bishop et al, 2003). Winter's work was the basis for the concept of moist wound healing (Bryan, 2004; Jones, 2005). Subsequent to this early work, there has been growing, consistent evidence presented in the literature from then to the current date with numerous laboratory, preclinical and clinical studies providing evidence for the benefits of moist wound healing (*Table 1*) (Junker et al, 2013; Souliotis et al, 2016). As a consequence, woundcare clinicians have embraced the concept of moist wound healing that has been further developed to encompass wound bed preparation as a clinical concept evolved to aid healing (Butcher, 2010; Sibbald et al, 2015). Wound bed preparation is '*the management of a wound in order to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures*' (Schultz et al, 2003; Falanga, 2000). It enables clinicians to focus on optimising conditions at the wound bed in order to encourage the normal processes of healing (Deeth and Grothier, 2016; Snyder et al, 2016).

However, the benefits of hydration in enabling wound healing progression has been somewhat overshadowed by the fact that a hydrated environment accompanied by the

Table 1. Some advantages of moist wound treatment over dry wound treatment

Effect	Experimental evidence	Clinical evidence
Up to 50% faster wound healing	Winter, 1962; Dyson et al, 1988	Varghese et al, 1986; Falanga, 1988; Madden et al, 1989; Rubio, 1991; Beam, 2008
Faster wound contraction		Wigger-Alberti et al, 2009
Enhanced and faster reepithelialisation	Eaglstein, 2001; Triller et al, 2012	Jones and Harding, 2007
Generally increase cellular proliferation		Romanelli et al, 2004; Attinger et al, 2008; Harding, 2012
Prolonged presence of growth factors and cytokines	Svensjö et al, 2000; Powers et al, 2013; Hackl et al, 2014	
Keratinocyte proliferation, fibroblast growth		Korting et al, 2011
Promotes angiogenesis/ revascularisation	Svensjö et al, 2000; Rusak and Rybak, 2013	Field and Kerstein, 1994; Dowsett and Ayello, 2004
Greater quantity and quality of ECM	Dyson et al, 1992; Mosti, 2013	
Collagen synthesis	Chen et al, 1992; Leung et al, 2012	
Lower rate of infection		Hutchinson and Lawrence, 1991; Kannon and Garret, 1995; Kirsner et al, 2004; NICE, 2008
Cleansing/irrigation		Dulecki and Pieper, 2005; Hall, 2007; Tao et al, 2015
Painless removal of the dressing without destroying newly formed tissue		Wiechula, 2003; Metzger, 2004; Coutts et al, 2008; Leaper et al, 2012
Less scarring and better cosmetic results	Atiyeh et al, 2003; Tandara et al, 2007; O'Shaughnessy et al, 2009; Mustoe and Gurjala, 2011	Atiyeh et al, 2004; Metzger, 2004; Hoeksema et al, 2013
Enhance autolytic debridement		Gray et al, 2005; King et al, 2014
Decrease in initial donor site pain and improved donor site healing		Weber et al, 1995

redolent inflammatory response occurs in chronic wounds and, associated with high levels of matrix metalloproteinases (MMPs), causes maceration of the wound/peri-wound skin and interferes with the healing process. It is therefore important to note here that excessive fluid is not *per se* the cause of skin damage but it is the content of the fluid that is of major importance (Cutting and White, 2002; Rippon et al, 2016). The differences between the two are explained here.

WOUND/PERI-WOUND SKIN HYDRATION AS OPPOSED TO MACERATION

From the initial trauma, wounds are bathed in wound exudate that contains many components that enable the normal process of wound healing to proceed. Such components include water, electrolytes, nutrients, inflammatory mediators, white cells, protein-digesting enzymes, e.g. MMPs, growth factors and waste products (Schultz et al, 2003). Wound healing is very dependent upon the level of hydration (Bishop et al, 2003) and hydration is purported to be the single most important external factor responsible for optimal healing (Atiyeh and Hayek, 2005). *Table 1* summarises the evidence that has been obtained from literature that supports the use of moist wound treatment over dry. Ousey et al (2016) has recently undertaken a literature review that presents the case for wound hydration.

Wound healing and maceration

In chronic wounds, exudate appears to have the opposite effect resulting in an aberrant healing process whereby its components debilitate healing. For example, over-production of MMPs (Gibson and Schultz, 2013; Caley et al, 2015) and neutrophil elastase (McCarty and Percival, 2013; McDaniel et al, 2013; Wilgus et al, 2013) that results in protein degradation in parallel with over-synthesis of inflammatory mediators which now prolong the inflammatory phase to the detriment of healing. As a consequence of both, over-hydration and biochemical wound milieu maceration occurs, which is not only damaging but a significant management challenge.

There is a difference between hydration, which is beneficial and enables healing, and maceration, which exacerbates healing. However, in presentation both conditions appear very similar. *Table 2* compares the effects of hydration versus maceration on healing.

As a consequence, moisture control in terms of wound exudate is of paramount importance especially in terms of managing its potential for damage (Chamanga, 2015). Thus a balance between enabling moist wound healing and preventing exudate damage (maceration) is vital (Jones, 2014). To this end, advanced wound dressings have been designed specifically with the

Wound bed covered with devitalised tissue/slough that contains tissue debris including bacteria (A). The sloughy surface acts as a barrier to epidermal movement across the wound bed (B) and requires debridement in order for this barrier to be removed. Lack of healing due also to presence of excessive wound bed proteases (C) as a result of an elevated tissue inflammation brought on by the underlying wound aetiology and presence of high levels of tissue irritants (e.g. proteases) within the slough/devitalised tissue. The consequences of an immature wound bed are a lack of effective tissue/dermal responses, e.g. angiogenesis (D). SC = *stratum corneum*

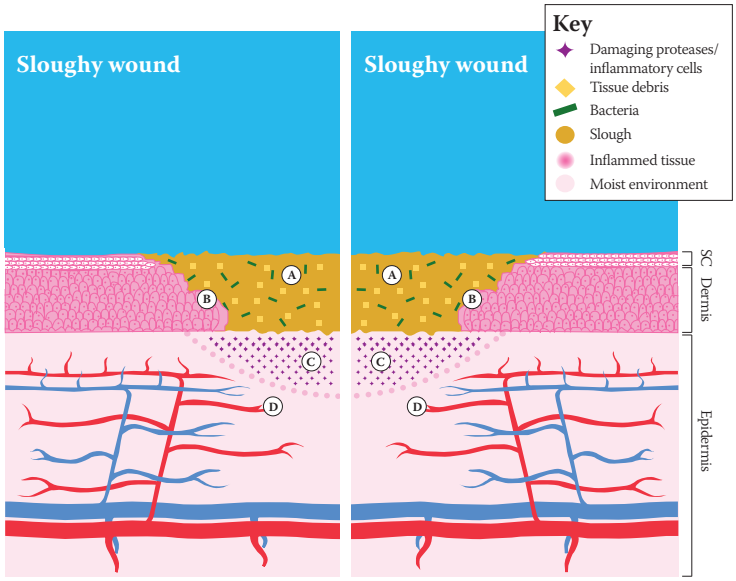
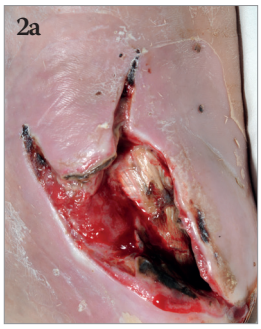
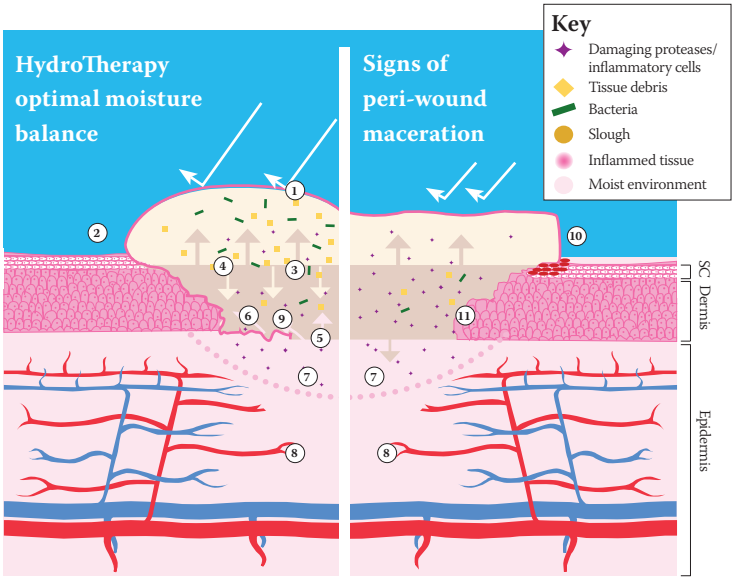


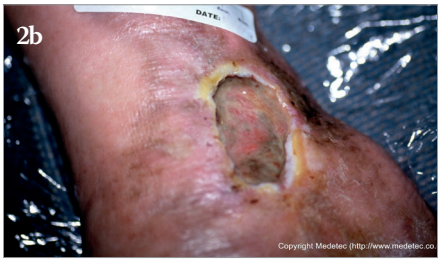
Figure 1. A photograph of a wound in need of debridement correlating with a diagrammatic representation and explanation of pathological components of that wound



HydroTherapy – optimal moisture balance

HRWD cover dressing protects the fragile tissue from contamination from the environment (1) and promotes hydration (2). HRWD's absorbing/rinsing action and fluid uptake characteristics removes tissue debris and damaging components (including proteases) (3). HRWD rinsing effect donates Ringer's solution (4) and establishment of moist wound environment encourages softening of slough (5, 6). Optimal moisture levels encourages wound progression (7), healthy granulation tissue formation (8), and epithelialisation (9). SC = *stratum corneum*

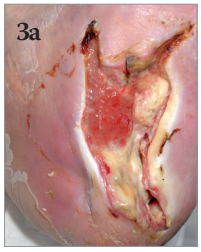




Signs of peri-wound maceration

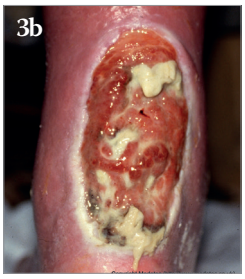
Presence of a modern wound dressing protects the fragile tissue from contamination from the environment (1) and softening of the slough (6). The establishment of a moist environment encourages some wound progression in the deeper tissues (7, 8). However, suboptimal hydration management results in deficiencies in exudate management leading to wound/peri-wound tissue damage due to excessive proteases (e.g. MMPs). Evidence of peri-wound skin maceration (10) with maceration-induced epidermal irritation hindering effective epidermal migration (11). SC = *stratum corneum*

Figure 2a and 2b. A wound with optimal moisture balance with some hyper-hydration versus a wound with low level maceration respectively



HydroTherapy-induced peri-wound hydration

Alongside the beneficial softening of the sloughy material (6) and migration of epidermis across the wound bed (9), optimising hydration levels leads to non-irritant hydration of the peri-wound epidermis. Donation of fluid (in the form of Ringer's solution) (4) from the reservoir of the HRWD dressing core (13), wound bed protease levels are modulated (decreased) via uptake and wound cleansing actions (15). SC = *stratum corneum*



Maceration-induced peri-wound inflammation

Sub-optimal hydration balance through limited exudate management results in worsening peri-wound maceration (10) alongside softening of slough (6). Peri-wound maceration accompanied by poor epidermal migration across wound bed (16). Poor exudate management of damaging wound exudate leads to elevation of peri-wound inflammatory irritation, both in the deeper layers of the epidermis (17) and in the subepidermal/dermal region (18). Additional irritation due to elevated and uncontrolled inflammatory cell-derived proteases (19). SC = *stratum corneum*

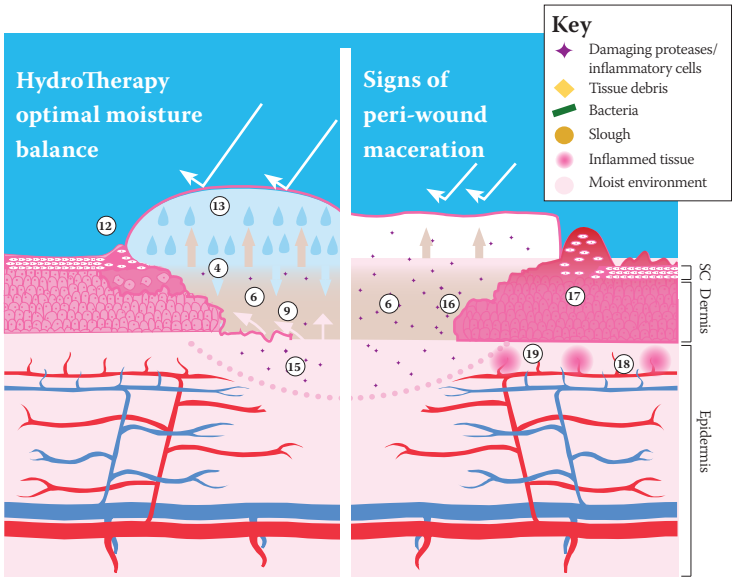
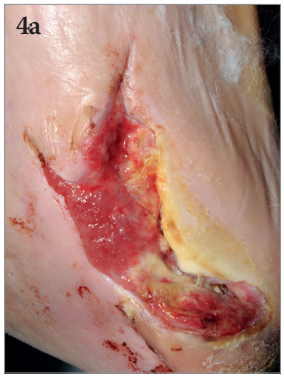
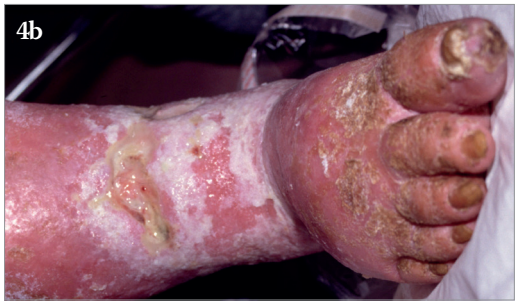


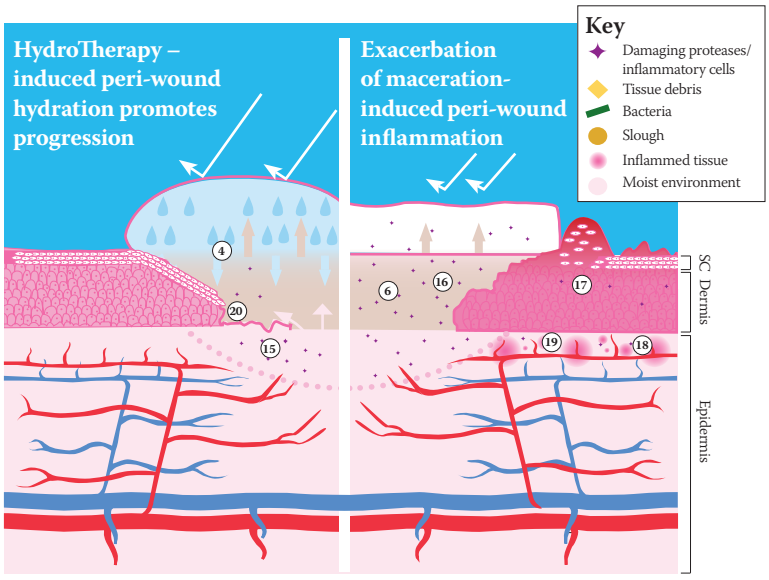
Figure 3a and 3b. A wound with optimal moisture balance with some hyper-hydration and healing/reepithelialisation versus a wound with erythema around wound and tissue damage/maceration at wound edges respectively



HydroTherapy-induced peri-wound hydration promotes wound progression

Continued donation of Ringer's solution promoted continued wound cleansing (4). Sustained modulation of protease levels via wound cleansing action (15) and maintained wound closure via migration and maturation of peri-wound epidermis (20). Together, HydroTherapy treatment promotes healing response via optimal wound environment at all phases of healing. SC = stratum corneum





Exacerbation of maceration-induced peri-wound inflammation

'Stalling' of re-epithelialisation as a result of sub-optimal epidermal migration (16) despite reduced epidermal barrier via softening of slough (6). Persistent poor exudate management results in spread of epidermal inflammation/irritation to deeper epidermal layers (17) and the spread of sub-epidermal/dermal inflammation due to protease-containing exudate (18,19). SC = *stratum corneum*

Figure 4a and 4b. A wound with optimal moisture balance with good healing progression versus a wound with a severe level of maceration respectively

Table 2. Comparative effects hydration versus maceration

Hydration	References	Maceration	References
Beneficial to healing	Kruse et al, 2015	Delays healing	Cutting and White, 2002
Aids debridement/cleansing	Powers et al, 2013	Increases slough and tissue damage	Ichikawa-Shigeta et al, 2014; Mugita et al, 2015
Lowers risk of infection	Sarabahi, 2012	Increased tissue necrosis — higher risk of infection	Benbow and Stephens, 2010; Charlesworth et al, 2014
Transient low grade dermatitis	Rietschel and Allen, 1977	High grade dermatitis, wet eczema	Gray and Weir, 2007; Colwell et al, 2011
Less pain	Morgan and Hoelscher, 2000; Metzger, 2004	Increased discomfort, irritation pain and reduced QoL	Butcher, 2010; Dini et al, 2014
Less scarring	Bolton et al, 2000; Benbow, 2008	Long term physiological changes in skin with associated tissue degradation	Mugita et al, 2015
Lower cost	Kerstein, 1995; Metzger, 2004	Increased cost	Charlesworth et al, 2014

main aim of fluid management and limiting the exposure of tissues to destructive wound fluids (Sibbald et al, 2015; Vasconcelos and Cavaco-Paulo, 2011; Wiegand and Hipler, 2013; Wiegand et al, 2011; Edwards and Caston-Pierre, 2013).

However, some wound dressings are poorer at managing wound exudate and preventing maceration than others. *Figures 1–4* present diagrams that are representative of the processes that occur when wounds are treated with a) a new hydro-responsive wound dressing (HRWD) and b) a standard, e.g. a foam, hydrocolloid or hydrofibre, wound dressing that is not managing wound exudate to the detriment of healing and therefore contribute to wound/peri-wound maceration. A detailed account of the mechanisms of either hydration (aiding healing) or maceration (exacerbating healing) supports the diagrams supplemented by images exemplifying the different states of hydration and maceration. It is anticipated these diagrams will assist clinicians in being able to differentiate between hydration and maceration when used in conjunction with the standard wound assessment procedures.

CONCLUSION.

It is essential that clinical practitioners in wound care are able to understand and identify the differences between peri-wound maceration and that of 'normal' hydration in order to achieve optimal outcomes of healing. For example, newly formed (delicate) epithelial tissue can easily be mistaken for maceration as it often appears as pale white tissue at the wound edge. It is important therefore that the clinician takes into account the context in which suspected maceration occurs so that an accurate and differential diagnosis can be undertaken. This article has aimed to support this differentiation using clinical examples and diagrammatic representations of hydration/hyper-hydration versus maceration.

WUK

Conflict of interest

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