Wound Hydration versus Maceration: Understanding the Differences

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Abstract: 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.

Key Words: wound bed preparation, devitalised tissue, debridement, de-sloughing, hydration, hyper-hydration; maceration

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Background: 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 co-ordinated 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 co-ordinated 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 evidence in support of this idea with consistent supportive evidence presented in the literature from then to the current date with numerous laboratory, preclinical and clinical studies that provides evidence for the benefits of moist wound healing - see Table 1 (Junker et al, 2013; Souliotis et al, 2016). As a consequence wound care clinicians have embraced the

concept of moist wound healing which 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' (p. S1, Schultz et al, 2003; Falanga, 2000) and to enable 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 redolent inflammatory response occurs in chronic wounds and associated with high levels of 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

Wound healing and hydration: 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 (eg matrix metalloproteinases – 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 (Caley et al, 2015; Gibson and Schultz, 2013) and neutrophil elastase (McDaniel et al, 2013; Wilgus et al, 2013; McCarty and Percival, 2013) which results in protein degradation in parallel with over-synthesis of inflammatory mediators that now prolong the inflammatory phase to the detriment of healing. As a consequence of both over-hydration and this biochemical wound milieu maceration occurs as a result which is not only damaging but a significant management challenge.

It is apparent therefore that maceration impedes healing, but that in presentation these 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 main aim fluid management and limiting the exposure of tissues to these 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).

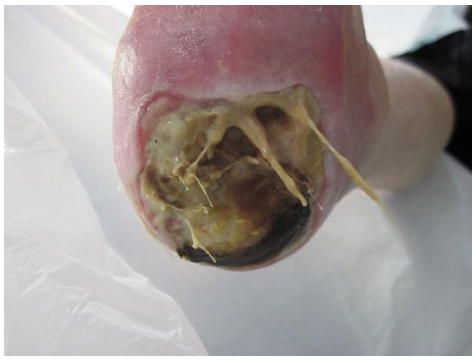
However some wound dressings are poorer at managing wound exudate and preventing maceration than others. Figures 1 – 4 present diagrams that are representative of the processes that occur when wounds are treated with a) a new Hydro Responsive Wound Dressing (HRWD) and b) a standard (eg. a foam, hydrocolloid 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.

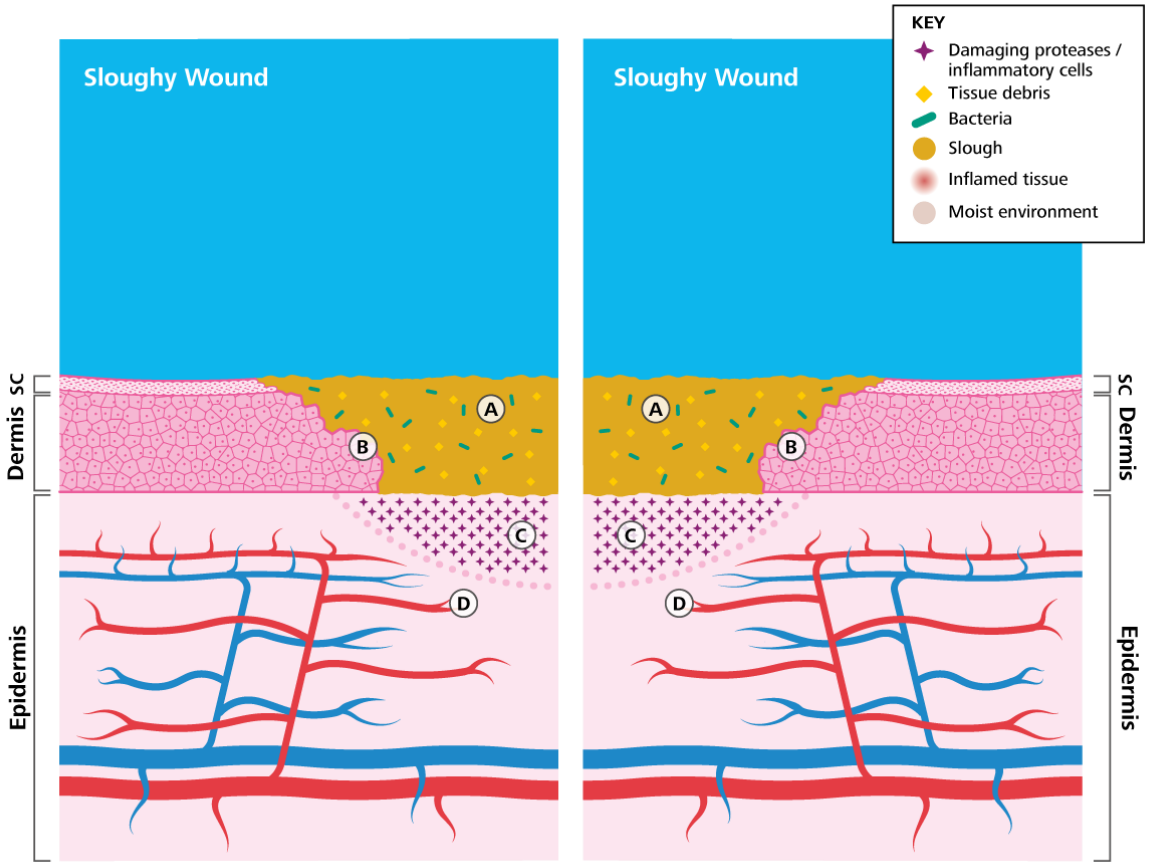
Conclusion. It is essential that clinical practitioners in wound care are able to understand and identify the differences between peri-wound maceration and that of 'normal' hydration in order to achieve optimal outcomes of healing. For example, newly formed (delicate) epithelial tissue can easily be mistaken for maceration as it often appears as pale white tissue at the wound edge. It is therefore important 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.

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; National Institute for Health and Care Excellence (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

Table 2. Comparative effects Hydration vs 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

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





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 is a lack of effective tissue/dermal responses (e.g., angiogenesis, (D)). *SC, stratum corneum*

Figures 2a and 2b showing a wound with optimal moisture balance with some hyper-hydration versus a wound with low level maceration respectively

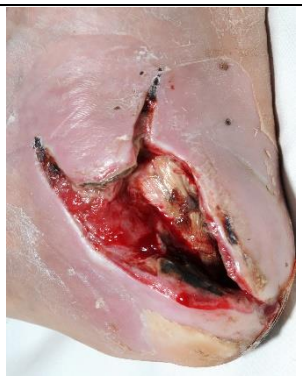


Figure 2a

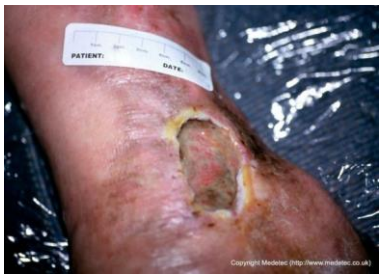
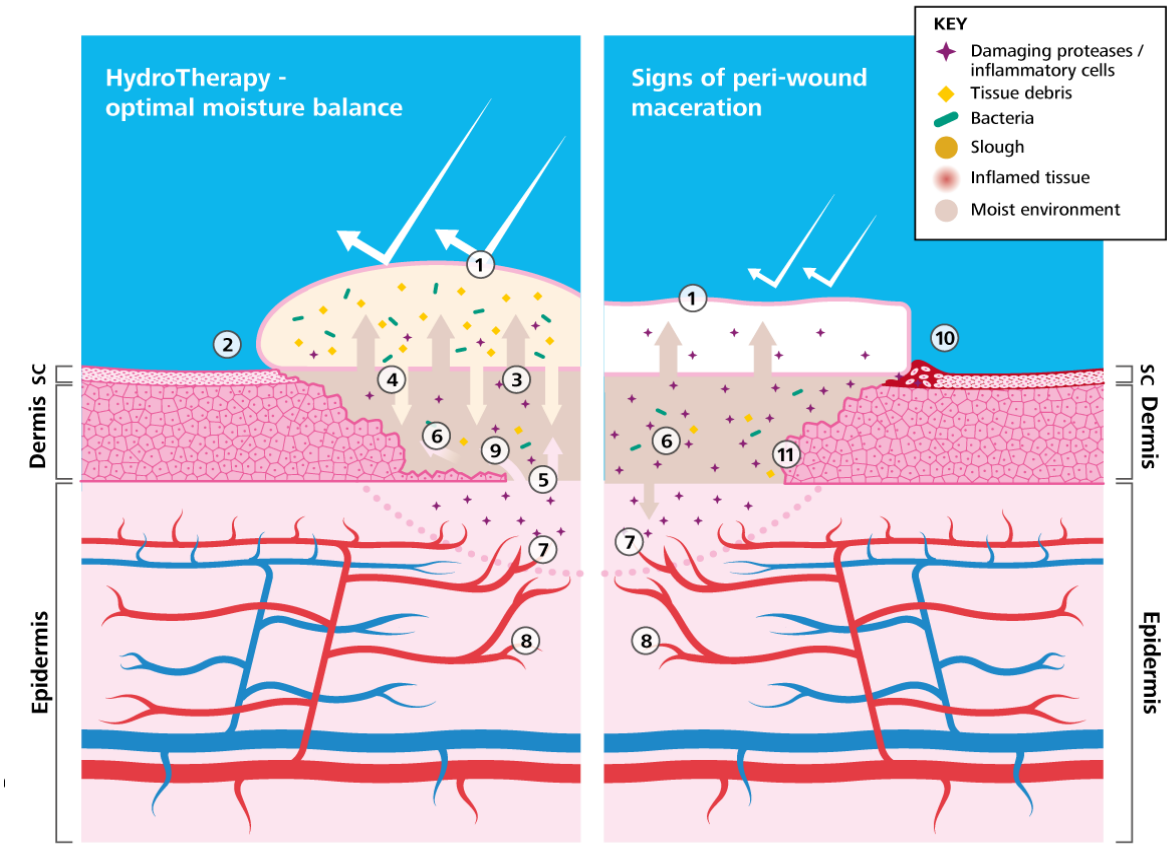


Figure 2b



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

Figures 3a and 3b showing 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

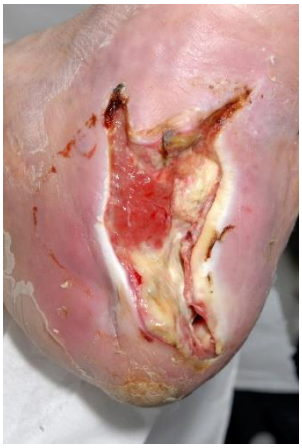


Figure 3a

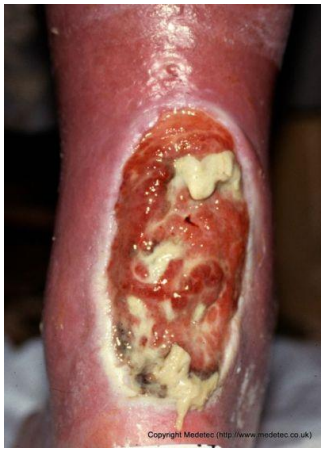
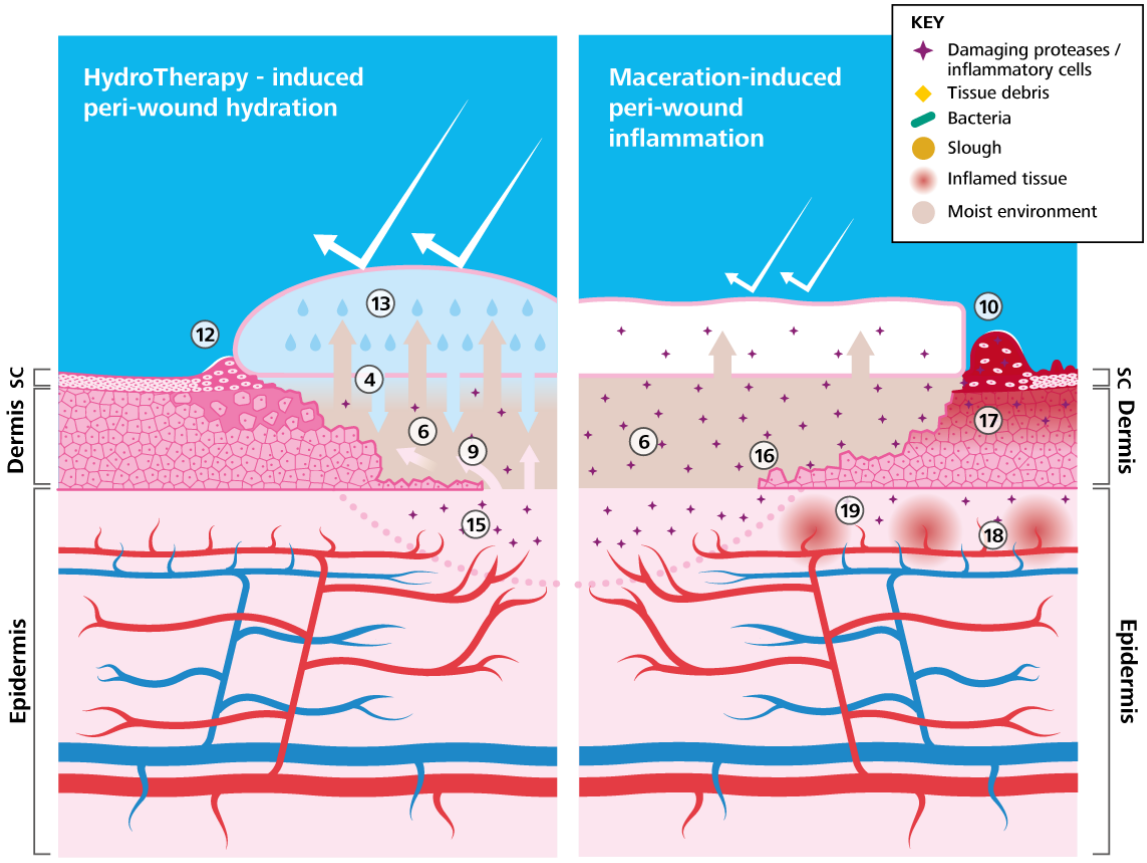


Figure 3b



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

Figures 4a and 4b showing a wound with optimal moisture balance with good healing progression versus a wound with a severe level of maceration respectively

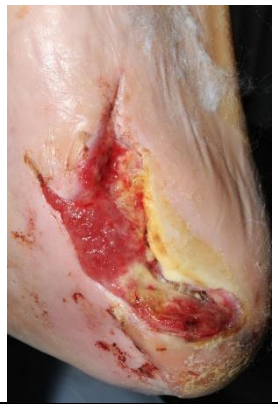


Figure 4a

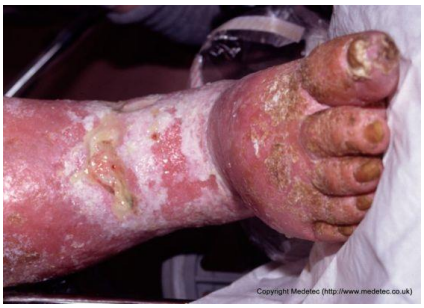
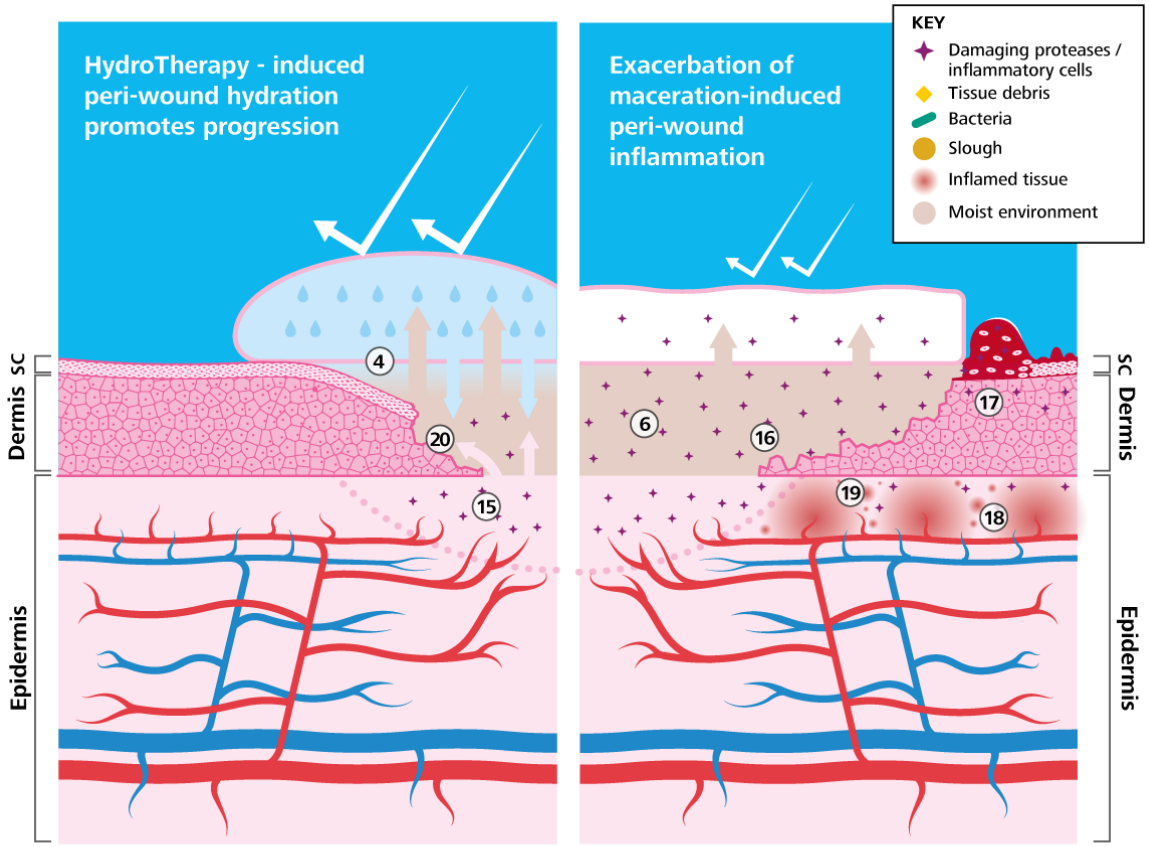


Figure 4b



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 resultof 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

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