Wound healing and hyper-hydration: a counter intuitive model

Winters seminal work in the 1960s relating to providing an optimal level of moisture to aid wound healing (granulation and re-epithelialisation) has been the single most effective advance in wound care over many decades. As such the development of advanced wound dressings that manage the fluidic wound environment have provided significant benefits in terms of healing to both patient and clinician. Although moist wound healing provides the guiding management principle confusion may arise between what is deemed to be an adequate level of tissue hydration and the risk of developing maceration. In addition, the counter-intuitive model 'hyper-hydration' of tissue appears to frustrate the moist wound healing approach and advocate a course of intervention whereby tissue is hydrated beyond what is a normally acceptable therapeutic level. This paper discusses tissue hydration, the cause and effect of maceration and distinguishes these from hyper-hydration of tissue. The rationale is to provide the clinician with a knowledge base that allows optimisation of treatment and outcomes and explains the reasoning behind wound healing using hyper-hydration..

• **Declaration of interest:** K. Cutting is a clinical research consultant to Hartmann. M. Rippon is Medical marketing consultant for Hartmann, and K. Ousey provides consultancy for a range of companies through the University of Huddersfield including consultancy services for Hartmann on Hydrotherapy.

hyper-hydration; hydration; moisture balance; maceration; skin; wound dressings

A homeostatic moist wound environment is generally accepted as beneficial to the healing process and co-exists with an adequately hydrated wound. Conversely, maceration of the peri-wound skin is considered to have a far-reaching and negative influence which impacts adversely on the patient, clinician[1,2] as a result of putative excessive hydration. There is a lack of clarity regarding the optimal level of hydration required to support healing. Furthermore, the origin of fear associated with excessive hydration of the peri-wound skin (maceration) appears to be founded on anecdotal evidence.[2] Here we try to clarify tissue hydration in relation to wound healing, maceration and, rationalise the counter intuitive model of healing through hyper-hydration. Developing understanding, based on the available evidence, of wound/soft tissue hydration, peri-wound maceration and the nuances of hyper-hydration has the potential to improve not only patient outcomes but also clinicians' appreciation of topical wound dressings and the role they have to play in support of healing.-

Moisture and wound healing

Healing of the skin consists of four overlapping and integrated phases haemostasis, inflammation, proliferation and remodelling.[3] This initial haemostatic response is characterised by platelet activation and coagulation, ensuring that blood loss is minimised. The inflammatory phase consists of an influx of inflammatory cells and mediators that help to prevent infection through the open wound. This is then followed by periods of cellular proliferation, extracellular matrix (ECM) deposition and finally remodelling which leads to scar formation.[3] Underpinning these processes is angiogenesis, generally occurring in the proliferative phase of healing which leads to a temporary increase in the number of blood vessels at the site of injury.[4] Although remodelling is regarded as the final phase of the repair process it is important to remember that remodelling of tissue takes place throughout the repair process and is not isolated to the post-closure phase.[3]

Wound hydration has been the basis of modern wound care since the seminal papers of Winter[5–8] in the 1960s. Scientific research *in vitro* and *in vivo* has supported this original premise[9–15] and moist wound healing has become the recognised tenet of clinicians working in the wound care field.[16] Studies have highlighted that moisture retained over the wound prevents desiccation of the wound surface and/or deeper tissues allowing for an unimpeded migration of epithelial cells over the wound surface.[5,17–19] Cytokines and growth factors are also enabled to exert their beneficial effect on wound contracture and re-epithelialisation, through the maintenace of a moist enviroment.[20,21] Furthermore, there is improved cosmesis, the provision of an environment that supports autolysis[22,23] and a decrease in pain experienced for example in split-

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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;[5] Dyson 1988[90]	Falanga, 1988;[97] Beam et al. 2008;[98] Varghese et al. 1986;[99] Rubio et al. 1991;[100] Madden et al. 1989[101]
Faster wound contraction		Wigger-Alberti et al. 2009[102]
Enhanced and faster reepithelialisation	Eaglstein 2001;[17] Triller et al. 2012[91]	Jones et al., 2007[103]
Generally increase cellular proliferation		Romanelli et al 2004;[104] Attinger et al. 2007;[105] Harding, 2012[106]
Prolonged presence of growth factors and cytokines	Svensjö et al. 2000;[14] Hackl et al. 2014;[15] Powers et al. 2013[92]	
Keratinocyte proliferation, fibroblast growth		Korting et al. 2011[77]
Promotes angiogenesis/revascularisation	Svensjö et al. 2000;[14] Rusak et al. 2013[93]	Field and Kerstein 1994;[107] Dowsett and Ayello 2004[108]
Greater quantity and quality of extracellular matrix	Dyson, 1992;[94] Mosti et al. 2013[21]	
Collagen synthesis	Chen et al. 1992;[95] Leung et al. 2007[96]	
Lower rate of infection		Hutchinson and Lawrence 1991;[109] Kannon and Garret, 1995;[110] Thomas 2003;[111] NICE, 2008[112]
Cleansing/irrigation		Duleck et al. 2005;[71] Hall 2007;[72] Tao et al. 2015[73]
Painless removal of the dressing without destroying newly formed tissue		Wiechulaet al. 2003;[113] Metzger 2004;[116] Leaper et al. 2012; Coutts et al., 2008
Less scarring and better cosmetic results	Atiyeh et al. 2003;[18] O'Shaughnessy et al. 2009;[114] Mustoe and Gurjala 2012;[22] Tandara et al. 2007[115]	Metzger 2004;[116] Atiyeh et al. 2004;[117] Hoeksema et al. 2013[118]
Enhance autolytic debridement		Gray et al. 2005;[119] King et al. 2014[23]
Decrease in initial donor site pain and improved donor site healing		Weber et al. 1995[24]

thickness graft donor sites in a moist enviroment.[24] The benefits resulting from maintaining a moist healing environment can be seen in Table 1. Good hydration (of the wound) has been described as the single most important external factor responsible for optimal wound healing.[20]

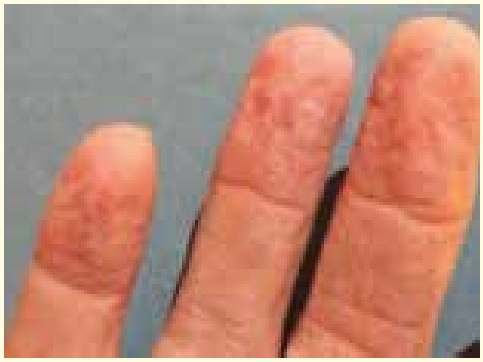
Tissue hydration

All biological processes require water, it is essential for maintaining homeostasis, a universal solvent, mediator of life's chemical reactions, and has a structure unlike that of any other liquid.[25] 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 survive only for days. In man, water content ranges from 75% body weight in infants, to 55% in the elderly[26] with the skin having a water content of approximately 30%. The outermost layer of the epidermis, the stratum corneum, prevents water loss. It forms a water-impermeable barrier. Any structural defect of its integrity will result in uncontrolled water loss such as in ruptured blisters or more dramatically in burns. The noticeable water 'loss' e.g. sweating is the result of active water transport in sweat glands regulated by ion and water channels. The epidermis provides a portal for insensible loss of water termed trans epidermal water loss (TEWL). Here fluid (vapour) is lost to the atmosphere via evaporation and diffusion over which the organism has little or no physiological control.

The skin therefore provides an interface between the body and the environment but does not maintain total control over water loss. Provided the skin remains intact and in conjunction with surface lipids and antimicrobial peptides the entry of potentially harmful environmental substances and microorganisms is prevented.[28,29]

In intact skin, the opposing forces of interstitial fluid pressure and capillary filtration pressure together with the rate of lymphatic drainage control fluid inflow from the local vasculature to the ECM[30] and thus maintain tissue hydration. However, when wounding occurs this mechanism is compromised and fluid inflow from the blood vessels increases due to vascular leakiness triggered by inflammation and is observed on the wound surface as exudate because of the missing epidermal barrier. Not all of the fluid that results from this decrease in interstitial fluid pressure resides on the wound surface as some ECM components such as hyaluronan absorb and hold this fluid at a capacity greater than that achieved by the skin.[27]

Fig 1. Transient, hyper-hydration of the skin (wrinkling) following prolonged immersion in water. This is quickly reversed on exposure to air



A number of definitions of hydration exist, but in relation to soft tissue, the most appropriate appears to be 'the process of providing an adequate amount of liquid to bodily tissues'.[31] Naturally, the question arises—what is adequate?

Hyper-hydration of the skin

Following prolonged exposure to water, swelling and absorption occur in the dead corneocytes in the outermost layer of the stratum corneum.[32] These cells are stacked in layers like bricks and their swelling is the key process by which their permeability and the mechanics of fluid interactions within the skin is controlled.[32] These cells contain a network of keratin filaments that interlock to form a three-dimensional lattice—which can increase its volume by five times when the strands stretch out.[32] The interplay of these opposing forces ensures that the skin can only absorb a certain amount of water, limited by the skin's physical structure.[32]

Immersion in a moist/wet environment for prolonged periods of time results in the skin becoming white and wrinkly (Fig 1). It is thought that this wrinkling response may provide an evolutionary benefit in terms of providing improved grip in wet conditions[33] and a better grasp of wet objects.[34] This wrinkling mechanism was investigated by Lewis and Pickering (1936) who suggested that the phenomenon was not solely related to water absorption but that the nervous system was also implicated.[35] Recent findings support this view and indicate that sympathetic innervation is important in water-immersion skin wrinkling.[36]

Some authors have shown that prolonged exposure to water can lead to dermatitis[37,38] but that exposure of the skin to water for short periods of time is generally deemed to be innocuous.[39] This latter finding is supported by a study that evaluated the effect of continuous exposure of human skin to water for 72 hour and 144 hour. The results noted only a mild, transient dermatitis occurred in half of the test sites.[40] Other report have demonstrated that extended water exposure had effects, which in themselves were not considered overtly damaging, for example, swelling of stratum corneum with increased epidermal thickness and dilation of intracellular spaces[41,42,43,32] increased stratum corneum suppleness[44,45,46] enhanced mitotic rate,[47] and reduced cytokine IL-1α mRNA levels.[48,49]

From the literature, its appears that reports on clinical observations and investigations that hyper-hydration of the skin is biologically limited, and does not necessarily result in sustained damage to the skin. Prolonged exposure of the skin to water results in a whitened or pearlescent appearance and is termed maceration.[50]

Maceration of the skin

Maceration of peri-wound skin is defined as 'the softening and breaking down of skin resulting from prolonged exposure to moisture'.[51] Maceration is a common aversion and although many clinical guidelines contain preventative recommendations[2] the origin of this apprehension appears to be shrouded by history, although some consider it may be related to the time when corrosive or irritating agents were used on wounds.[2]

The term Moisture Associated Skin Damage (MASD) is now accepted as the general term for inflammation or skin erosion caused by prolonged exposure to a source of moisture such as urine, stool, sweat, wound exudate, saliva, or mucus.[52] Moreover in order for MASD to occur, complicating factors are required in addition to moisture exposure, such as mechanical (friction), chemical (irritants contained in the moisture source), microbial or in the case of chronic wounds a significant complicating factor is the presence of wound exudate.[50,53]

The synthesis of wound exudate is a normal part of wound healing, generally associated with the inflammatory stage.[54] It is an essential part of the wound healing process in that it provides a moist environment conducive to healing. Acute wound exudate is a mixture of biochemical (including, growth factors, cytokines, electrolytes, proteases and nutrients) cellular components (such as infiltrating white cells such as leucocytes) and proteins (including fibrinogen and fibrin) that enable healing to occur.[55] Chronic wounds, however, are characterised by non-resolving inflammation, underpinned by disruption to the 'normal' biochemistry and cellular activity.[56] Chronic wound exudate contains an excess of protein degrading enzymes such as serine proteases such as plasmin and elastase and matrix metalloproteases (MMPs) such as MMP-2 and MMP-9, increased numbers of neutrophils together with an elevated profile of pro-inflammatory cytokines.[57,56] In addition to endogenous proteases some bacterial species can produce powerful proteases and may contribute to the pro-

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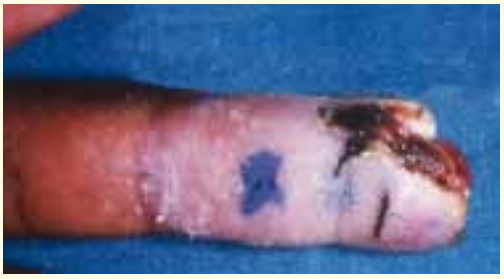
Fig 2. Maceration of a finger as a result of inhibition of trans epidermal water loss. This is promptly reversed on removal of occlusion



teolytic damage of chronic wound exudate.[58] This results in a state that is not conducive with homeostasis as proteases degrade growth factors and fibroblasts resulting in defective remodelling of the ECM.[59] Nearly two decades ago, chronic wound exudate was described as 'a wounding agent in its own right'.[60] Thus, excessive fluid is not per se the cause of skin damage but it is the content of the fluid that is of major importance.[27] The corrosive effect of chronic wound exudate leads to breakdown of the peri-wound skin, which in turn, can lead to wound enlargement, delayed healing, a higher risk of infection and increased pain and discomfort that results in a reduction in quality of life for the patient.[52,61] Patient morbidity and cost of treatment will inevitably increase with the potential for hospitalisation.[62]

Maceration that occurs as a result of both over-hydration and the biochemical wound composition is not only damaging but a challenging to manage.[63,64] Clinical practitioners need to be able to identify the differences between peri-wound maceration and that of 'normal' hydration in order to achieve optimal outcomes. For example, newly formed (delicate) epithelial tissue can be mistaken for maceration as it often appears as pale

Fig 3. Chronic wound with a moderate/heavy level of exudate with whitened skin, swelling and where the surface of the skin is laced with multiple networks of fine grooves called sulci cutis

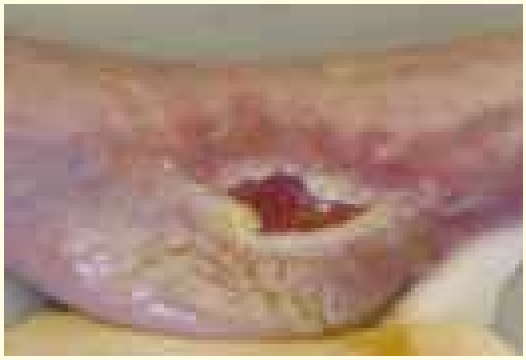


Fig 4. Grossly macerated peri-ulcer skin as a result of the combination of chronic wound exudate containing proteases and prolonged intervals between dressing change

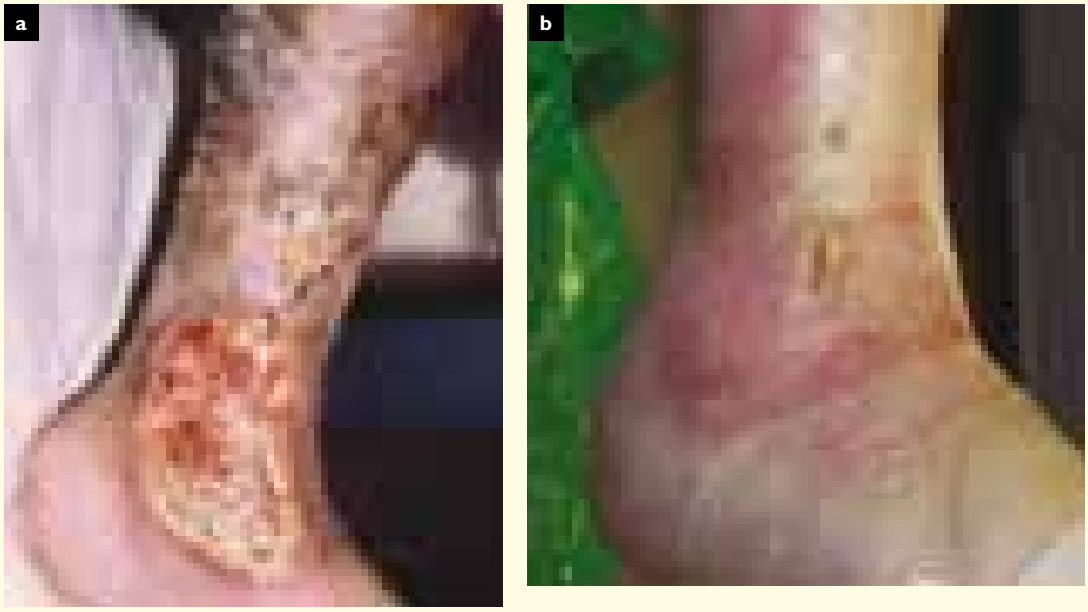
white tissue. It is therefore important that the clinician takes into account the context in which suspected maceration occurs so that an accurate diagnosis is made.w

Hyper-hydration of the skin (such as spending too long in the bath) can present as white wrinkly skin (Fig 1). A similar situation occurs when a wound dressing has not managed to maintain wound moisture balance such as when TEWL is inhibited or when wound exudate remains in contact for extended periods of time with the peri-wound skin. Fig 2 shows over-hydrated skin on a finger due to inhibition of TEWL. In these two examples the 'maceration' is quickly and easily reversed. In contrast, Fig 3 demonstrates a chronic wound with a moderate to heavy level of exudate presenting as whitened skin with swelling and where the surface of the skin is not smooth, but is laced with multiple networks of fine grooves called sulci cutis.[50,65] Fig 4 shows grossly macerated peri-ulcer skin as a result of a combination of chronic wound exudate and prolonged dressing change intervals. Maceration may also be associated with dermatitis/eczema (Fig 5 and 6) and may present with associated erythema, sloughy/necrotic tissue and extensive tissue breakdown of the wound/peri-wound skin.

Reversal of over-hydration of the skin

It is important to note that some authors have inferred that skin damage caused by excessive hydration is reversible.[61] In support of this premise is data from a recent study[62] that investigated the hydration effects on skin microstructure and its implications in relation to enhancing transcutaneous delivery of biomacromolecules. In this study cryo-scanning electron microscopy was used to investigate how hydration changes to the stratum corneum allowed penetration of macromolecules. The results showed that extended hydration (>8 hour) caused swelling of the corneocytes, created inter-corneocyte rupture, and caused micro-

Fig 5. Maceration of venous leg ulcers with associated dermatitis/venous eczema.



structural changes in lipid self-assembly. These disruptions allowed penetration (of biomacromolecules) through the barrier of the stratum corneum, importantly the disruptions were reversible, as removing the hydration source enabled restoration of the barrier.[66] This is supported by data presented in a study in which the skin membrane electrical impedance properties under the influence of a varying water gradient was investigated. The results from this study concluded that hydration/dehydration induced reversible changes of membrane resistance and effective capacitance.[67]

Healing and hyper-hydration

Although the term hyper-hydration is contemporary the underlying principle has a notable historical provenance. Junker (2013), records how Hebra, who published in 1861 his experience of patients with extensive burns and how they were treated by immersion in a bath using 'continuous baths' for months or years.[10] Hebra claimed that the treatment, using water, reduced patients' pain, limited their weight loss and ensured their survival. When the continuous baths were stopped none of the patients survived. Later, during the Second World War John Bun-

Table 2. Comparative effects hydration versus maceration

Hydration	References	Maceration	References
Beneficial to healing	Kruse et al. 2015[9]	Delays healing	Cutting and White 2002[92,120]
Aids debridement/cleansing	Powers et al. 2013[87]	Increases slough and tissue damage	Mugita et al. 2015;[121] Ichikawa-Shigeta et al. 2014[50]
Lowers risk of infection	Sarabahi 2012[122]	Increased tissue necrosis—higher risk of infection	Benbow and Stephens, 2010;[123] Charlesworth et al. 2014[62]
Transient low grade dermatitis	Rietschel and Allen 1977[40]	High grade dermatitis, wet eczema	Gray and Weir 2007;[52] Colwell et al. 2011[124]
Less pain	Morgan 2000;[125] Metzger et al. 2004[116]	Increased discomfort, irritation pain and reduced quality of life	Butcher 2000;[126] Dini et al. 2014[88]
Less scarring	Bolton et al.2000;[2] Benbow 2008[123]	Long term physiological changes in skin with associated tissue degradation	Mugita et al. 2015[121]
Lower cost	Kerstein 1995;[128] Metzger 2004[116]	Increased cost	Charlesworth et al. 2014[62]

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yan, a medical officer in the Royal Navy treated wounded soldiers using the 'envelope' method.[68] A coated silk sheeting was used to envelope a large burn wound area which acted as a trough into which a solution of electrolytically produced sodium hypochlorite was placed for 20 minutes three times each day. Bunyan claimed that this method improved healing, cosmesis and avoided the use of painful dressing changes.[68]

In 1985 Stenn and Yan investigated the effect of a liquid covering for superficial skin wounds and its effect on wound closure in a guinea pig wound model.[69] The results of this study showed that the animals tolerated the liquid bandage well and that no bacterial contamination or wound maceration was evident. The extent of re-epithelialisation with time was measured histologically under three separate conditions: wound exposed to air, wound covered and kept moist, and wound covered with liquid and the results showed that the liquid cover enhanced the rate of wound closure significantly.[69] In another experimental study, that investigated the healing of partial-thickness porcine skin wounds, in a liquid environment, the healing of fluid-treated wounds occurred without tissue maceration and showed less inflammation and less scar formation than healing of wounds exposed to air.[70]

Topical wound irrigation (intermittent or continuous) with either saline, water or isotonic solutions is a technique that provides the wound with a fluidic environment and has been used successfully as an aid to healing in many different wound types.[71,72] A pilot study evaluated the effect of irrigation on two groups of patients, with severely infected wounds, treated with either a) continuous topical irrigation (n=17) or b) with standard of care (no irrigation; n=15). Results showed that irrigation improved severely infected wound healing through inhibition of pro-inflammatory cytokines and improving tissue regeneration when compared with the control group.[73] Fluidic therapy such as instillation combined with the use of adjunctive negative pressure wound therapy (NPWTi) has been shown to enhance exudate and debris removal, provide regular cleansing of the wound bed, and add moisture to the wound. Positive results have been demonstrated with this technique in assisting healing of static and painful wounds.[74]

These studies, uphold the view that innocuous fluid that remains in contact with the wound bed for extended periods of time supports healing and is well tolerated. Note—Hebra claimed his electrolytic sodium hypochlorite solution was non-toxic.[68]

Wound dressings that provide part or complete occlusion and retain a degree of moisture content over the wound surface are de rigueur in the treatment of most acute and chronic wounds. These have been developed in many forms for example, films,[75,76] hydrocolloids,[77–79] foams[80,81] and hydrofibres[82,83] and new dressings and their components are still being developed with this aim in mind.[84]

The use of a dressing that provides a high fluid content was introduced over ten years ago and has since been further developed. The main characteristic of this wound dressing is that it maintains the wound in a fluidic environment of isotonic Ringers +solution. This dressing technology has been shown to be highly successfully in the treatment of acute[76,85] and chronic wounds.[86,87]

Maintaining a balanced moist environment for wounds is highly important. Advanced dressings are now able to cope with a full range of exudate levels. It is this ability to effectively manage the balance between excessive moisture/exudate presence at the wound surface yet ensure a correct level of hydration that can otherwise complicate clinical practice.[88,89] However an imbalance of moisture in conjunction with an acidic wound fluid will cause tissue damage and maceration of the peri-wound skin.

Bolton[2] eloquently listed the dressing variables that require investigation to clarify clinical practice in relation to maceration:

- Wound dressings absorbency and adsorbency
- Wound dressing wicking characteristics
- Wound dressing capacity to retain fluid.

These variables cannot be effectively examined in isolation of:

- The type and amount of exudate
- Pathology of peri-wound skin
- Potential sources of physical, chemical, metabolic, or vascular damage.

Conclusion

Hydration is highly beneficial to wound healing but needs to be clearly differentiated from maceration (and the corrosive nature of chronic wound exudate). This is because, the negative physiological and clinical implications of maceration and its treatment/prevention is far removed from that of hydration. Similarities in presentation may cause confusion and unwarranted intervention that can lead to a wrong treatment pathway and ultimately be detrimental to the patient and the healing outcome of their wound. Healing through hyper-hydration is a counter-intuitive model that at first sight may appear incongruent with the more familiar moist healing paradigm. However, the isotonic nature of the fluid used in hyper-hydration together with the homeostatic mechanism of soft tissue, ensure that this approach to healing remains tenable. ■

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