To drain or not to drain? – That is the question

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Abstract
With regard to the management of partial thickness burns blisters, confusion continues in the literature as to whether to drain the blister, drain and deroof the blister, or leave the blister intact. This in turn creates difference of opinion between medical and nursing colleagues with little in the way of evidence-based recommendations. This paper highlights some of the conflicting research in an attempt to assist in the development of policies of best practice.

Introduction
The question as to whether burn blisters should be drained and deroofed has been debated for many years, with differing opinions expressed in texts and by medical and nursing colleagues.

The main arguments are whether to:

• Retain the blister fluid inside its cap so that it acts as a natural biological dressing.
• Remove the fluid, which can act as a medium for microbial proliferation.

These conflicting opinions create variation in the treatment of minor burn blisters resulting in confusion for staff and patients. This paper will examine the conflicting research so that it may assist in the formulation of evidence-based guidelines to best manage superficial partial thickness burn blisters.

Burns blisters
Burns are defined as tissue damage caused by radiation, heat, friction, electricity or chemicals, and can range from superficial, partial thickness, deep dermal or full thickness:

• A superficial burn is destruction of the epidermis only.
• A partial thickness burn affects the epidermis and some dermis.
• A deep dermal burn is destruction of epidermis and dermis leaving only skin appendages.
• A full thickness burn is destruction of the epidermis and underlying subcutaneous tissue.

Burns are comprised of three zones:
1. The coagulation zone consisting of dead tissue that has been destroyed by heat.
2. The stasis zone (often the most fragile area) consists of damaged tissue but is viable and may recover.
3. The hyperaemic zone, which is the area immediately surrounding the injury, has minimal damage and can recover spontaneously.

Although some burn injuries are made up of several zones of tissue damage due to difference in heat transfer, the hallmark of a superficial partial thickness burn is the formation of intact blisters at the site of heat exposure. There is a paucity of information in the literature as to what type of burn would predispose more to blister formation.

Burn blisters form when the epidermis separates from the underlying dermis, and can occur during the inflammatory stage as a physiological response to a burn injury. During the blister formation there is increased permeability by the damaged capillaries, which leak large amounts of plasma into the interstitial space, and it is the force of this fluid...
shift that disrupts and separates the epidermis from the dermis, creating a vesicle containing fluid. Superficial partial thickness blisters are typically thin walled, red, with weeping skin and intact sensation, whereas deep partial thickness blisters are thick walled and have white skin and either intact or impaired sensation.

Biochemical analysis of burn blister fluid demonstrates high levels of plasma protein containing thromboxane and arachidonic acid. Thromboxane causes vasoconstriction and has been thought to add to the risk of possible development of wound ischaemia. Arachidonic acid metabolites are released following an anti-inflammatory response mediated by cytokines and in contrast to thromboxane, causes vasodilatation. It is proposed that the first aid treatment of burn cooling is vital in maintaining normal homoeostasis between arachidonic acid and excess thromboxane production.

Proponents of draining burn blisters focus their arguments on the following:

Rockwell and Ehrlich evaluated a number of studies from 1970 to 1990, predominantly on the bactericidal activity of burn blister fluid and reduced amounts of immunoglobulins. In follow-up studies they conclude that there is an absence of complement levels in blister fluid, which in turn “affect the bactericidal and metabolic activities of normal lymphocytes and neutrophils”. They report that blister fluid contains acid metabolites which increase the inflammatory response thereby deepening the burn, and conclude that it causes several deleterious effects on wound healing, recommending that blister fluid be evacuated.

Garner et al studied the effects of burn blister fluid on the keratinocytes responsible for the protein keratin, which gives the epidermis its toughness and is essential for the healing of all burns. Their studies demonstrating an average of 40% reduction of keratinocyte formation in blister fluid, lead them to conclude that re-epithelialisation may be inhibited beneath burn blisters and to advocate debridement.

This was contradicted in 1994, when Wilson et al studied a calcium binding protein in human burn blister fluid called calmodulin and its role in the mitogencity of blister fluid in the culture of human keratinocytes, fibroblasts and mouse 3T3 fibroblasts. Calmodulin levels were found to be three times greater in fluid up to 48 hours old, acting as an autocrine growth factor for cultured keratinocytes, leading the researchers to conclude that calmodulin is partially responsible for cell proliferation in wound healing. These studies led Wilson et al to report that burn...
blister fluid favoured wound healing and advise leaving blisters intact, but if in danger of bursting, to retain under an occlusive dressing.

Further studies on cultured keratinocytes by Reagan et al showed only marginal effects on keratinocyte proliferation and differentiation. They conclude that the effect of burn blister fluid on re-epithelialisation is “neither salutary nor detrimental and should not dictate the clinical management of burn blisters” 10.

Wilson et al 11 observed that burns to the hands seemed to be followed by more stiffness and contracture, and questioned whether burn exudate contributes to this difference. They exposed fibroblasts to burn blister fluid which contracted 30% – 90% more than control cells not exposed to the fluid. This consistent finding led them to discuss possible future pharmacologic preparations that would antagonise the stimulatory effect of burn fluid on fibroblasts. They did not comment on whether or not to debride.

Further support for burn blister removal is cited by Haycock et al 12 who published a complex study in 1997, showing evidence that oxygen free radicals may contribute to further tissue damage following cutaneous thermal injury. They cite a number of studies supporting the involvement of reactive oxygen species in the process of thermal injury, and suggest further studies to investigate the benefit of giving antioxidants to people with burns.

Ortega et al 13 examined blister fluid for human beta-2 defensin. Defensins are a family of antimicrobial peptides produced by human keratinocytes, with a potent bactericidal activity. Ortega et al found human defensin beta-2 to be absent in burn blister fluid, and conclude that this suggested a predisposition to infection, advocating antimicrobial peptide application to prevent and treat burn sepsis.

Proponents of keeping blisters intact have used different arguments and studies:

The role of cytokines and growth factors which stimulate the wound healing process were studied by Ono et al 14 who report that blister fluid contains large amounts of platelet derived growth factor, interleukin, and transforming growth factor both alpha and beta, all of which suggests a cytokine network leading to a more rapid epithelialisation.

Singer et al 15 question the recommendation to routinely debride all burn blisters following a study that compared rates of infection in debrided and non-debrided pigs. They demonstrated that debrided partial thickness burns are three times more likely to become infected than non-debrided. They admitted that the efficacy of their study could be questioned because although porcine and human skin is very similar, pigs do not form blisters and comparison with humans would be difficult to prove.

**Discussion**

Reading the available literature makes it difficult to formulate an evidence-based opinion of burn blister management. Evidence in the literature is limited by lack of randomised clinical trials based on clinical (patient) outcomes. The work that has been done is important, but it is untested in the clinical setting and so it is difficult to draw any conclusion. There is little discussion with regard to burns of varying depth, or mixed burns that have both deeper partial thickness and superficial burn areas.

In practical terms, Banwell states that intact blisters indicate a superficial burn only, and that these burns usually heal by conservative means within one to two weeks in a moist wound environment provided by most dressing regimens 16. In regard to initial assessment and treatment of burns of varying degrees of dermal destruction, Banwell goes on to point out that intact blisters are usually not a feature of these burns, but it is the depth of dermal injury which determines if these burns will heal conservatively or require surgery. If these burns are admitted for surgery, any blisters will usually become deroofed in the cleansing and debridement processes 16.

Whether studies advocate retention or debridement of burn blisters, almost without exception all texts acknowledge that this is a contentious issue and further research is required. Therefore, when searching for clear guidelines that can be used with confidence in the practical setting, it is the tertiary burns centres that can assist in forming protocols.

The Royal Childrens Hospital Melbourne (Burns Unit: Clinical Information <www.rch.org.au/burns/clinical/> and Clinical Practice Guidelines <www.rch.org.au/clinicalguide/cpg.cf?doc_id=5158>) advocates a closed dressing technique for small blisters, which are often very painful and hypersensitive to touch. It is recommended that these blisters should be left intact and covered with an occlusive dressing whereas large blisters should be aspirated but not deroofed until a few days later, unless infection is suspected.
A similar protocol is advocated by duKamp, Wardrope and Flanagan. Richard et al advocate draining blisters with the exception of finger tips and the palm of the hand, which can lead to increased pain.

Most discrepancies in the literature apply to the management of larger blisters. Hudspith et al recommend large blisters be deroofed and dead skin removed, and duKamp discusses the argument that blister fluid may increase pressure on the wound bed and slow healing by reduction of blood supply, thereby causing necrosis.

At the Burns Unit at Royal Perth Hospital they routinely deroof blisters to facilitate joint movement, assessment of burn depth, removal of non-viable tissue, and reducing the risk of necrosis and infection (pers. comm.).

When considering all of the previous references and recommendations, the single overriding principle must be that all burns be kept clean and infection free and that pain management be effective. On balance it would seem good practice to leave small (<6 mm) or thick walled blisters intact to allow gradual absorption of blister fluid to maximise healing and reduce discomfort.

If blisters are large, thin walled and confluent, they can be drained and deroofed. duKamp recommends that the blister roof be left intact for 72 hours, allowing the inflammatory response to be completed, following which blistered skin should be excised, ensuring the zone of hyperaemia is not exceeded. If shearing forces become an issue or the blister is over a joint, aspiration with a needle and syringe is recommended. Wardrope et al recommend that if a blister has burst at initial assessment and almost detached, it should be removed. Most texts highlight the impact of blisters on hands and joints and necessitate intervention for functional purposes, depending on the anatomical position of injury and institutional preference.

Conclusion

Although the references advocating burn blister drainage and debridement are associated with more scientific studies, the majority of similar references are now over ten years old. The text most often quoted by Rockwell and Ehrlich is fifteen years old, and that paper quotes literature that was then twenty years old. The field of burns treatment and dressing products has advanced significantly and some of the arguments put forward over the past thirty-five years may not be as relevant today. This is reinforced by the advances taking place in burns dressings, bioengineering and a greater understanding of burns and their pathophysiology.

The principles of superficial partial thickness burns that could be used to compile an evidence-based policy with confidence are highlighted by Sargent who discusses the conflicting recommendations and clinical applications, but advocates that the management of burn blisters be supported by evidence across the six categories of burn wound management: healing, functional outcome, aesthetic outcome, patient comfort, ease of dressing and cost efficiency.

Revisiting the question – To drain or not to drain? – the above information may be the foundation for answering the question with more confidence.

References