



University of **HUDDERSFIELD**

University of Huddersfield Repository

Ousey, Karen

Pressure Ulcers and Suprasorb X+PHMB Infection, prevention and treatment

Original Citation

Ousey, Karen (2010) Pressure Ulcers and Suprasorb X+PHMB Infection, prevention and treatment. Wounds UK, Aberdeen.

This version is available at <http://eprints.hud.ac.uk/id/eprint/9905/>

The University Repository is a digital collection of the research output of the University, available on Open Access. Copyright and Moral Rights for the items on this site are retained by the individual author and/or other copyright owners. Users may access full items free of charge; copies of full text items generally can be reproduced, displayed or performed and given to third parties in any format or medium for personal research or study, educational or not-for-profit purposes without prior permission or charge, provided:

- The authors, title and full bibliographic details is credited in any copy;
- A hyperlink and/or URL is included for the original metadata page; and
- The content is not changed in any way.

For more information, including our policy and submission procedure, please contact the Repository Team at: E.mailbox@hud.ac.uk.

<http://eprints.hud.ac.uk/>

Pressure Ulcers and Suprasorb® X+PHMB

Infection, prevention and treatment

ACTIVA
HEALTHCARE

an  Company

In association with

Wounds UK

Part 4 of a series of 5 clinical booklets

Wounds UK Ltd, A Schofield Healthcare Media Company, Suite 3.1, 36 Upperkirkgate,
Aberdeen AB10 1BA

© Wounds UK Limited 2010

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without prior permission from the publishers

Printed in the UK by MDP, Wiltshire, UK

Pressure Ulcers and Suprasorb® X+PHMB

Infection, prevention and treatment

As a leading provider of education in the management of wounds, Activa Healthcare, an L&R Company, are delighted to sponsor this series of booklets, covering infection, prevention and treatment of:

- »» Leg ulcers
- »» Surgical site wounds
- »» Trauma wounds
- »» Pressure ulcers
- »» Diabetic foot ulcers.

INTRODUCTION

The last few years have witnessed the identification of the importance of effective pressure ulcer prevention through the publication of the Quality Agenda (Department of Health [DH], 2009a). Labour and the new coalition Government have made it clear that they want the NHS to provide a quality service for all those accessing health care, and that poor quality care will not be tolerated.

Pressure ulceration is not only a financial burden to health care, but also a significant cause of morbidity and mortality for patients (Posnett et al, 2009). The number of older people in the United Kingdom is rising, with the fastest growing age group in the population being those aged 80 years. The DH (2006) approximated that this age group had increased by over 1.1 million between 1981 and 2007, from 2.8% to 4.5% of the population. This suggests that the incidence of pressure ulcers may also rise and, as such, clear guidelines and policies must be developed and implemented to allow the effective use of scarce resources. Integral to these is education that can promote understanding of the cause and prevention of pressure ulceration, and present evidence-based strategies to treat pressure ulceration.

Healthcare spending will not rise over the next few years, indeed, cost-efficiency savings of £15–£20 billion need to be made by the end of 2013/14 which can be reinvested into the service to deliver year-on-year quality improvements (DH, 2010). The NHS will be required to concentrate on improving productivity and eliminating waste, while focusing on quality (DH, 2010).

Posnett and Franks (2007) estimated the cost of wound care to the NHS as being £2.3bn and £3.1 billion a year (2005–2006 prices). The DH (2009b) approximated that an average district general hospital spends between £600,000 to £3 million each year on treating pressure ulcers, and that this figure needs to be reduced. They maintained that the majority of pressure ulcers are entirely preventable through risk assessment and the implementation of pressure-relieving measures (DH, 2009b). The publication of *NHS 2010–2015: from good to great* (DH, 2009c) further identified that there would be 'safer care for patients who could be confident that they would be protected from avoidable harm', and highlighted pressure ulcers as an area that required addressing.

This document presents the practitioner with an overview of pressure ulceration, how to manage pressure ulcers and directs the reader to relevant guidelines that can underpin interventions.

Karen Ousey
Principal Lecturer, Department of Nursing and Health Studies,
Centre for Health and Social Care, University of Huddersfield

WOUND INFECTION

Intact skin provides an effective physical barrier to microbial invasion, while normal surface flora and epidermal lipids form a chemical barrier (Landis et al, 2007). However, when skin integrity is compromised, a pathway is opened for microorganisms to enter the body. Once present in a wound, bacteria have the resources and conditions available for their rapid multiplication; a process which ultimately leads to infection unless the body's defence mechanisms can overcome this assault. Bacteria within the wound actively compete with the host cells for oxygen and nutrients, and also release a wide range of enzymes and toxins which negatively affect the host cells within the area and cause systemic toxicity (White et al, 2001). All wounds become contaminated with bacteria at, or shortly after injury. Wounds that remain open are colonised with bacteria, and yet research shows that most of these wounds, even chronic wounds, can and do heal (Hansson et al, 1995).

In nature, communities of bacteria consisting of a single species are rare (Cooper and Okhiria, 2008); instead, bacteria exist in diverse communities, including anaerobes as well as the more commonly identified aerobes (Cutting and Harding, 1994). On occasions, these communities will also include fungi and viruses. Recently, there have been discussions about the possibility of biofilm development within wounds (Rhoads et al, 2008). Biofilms are communities of organisms living within a three-dimensional extracellular polysaccharide matrix, which form gradually over time. In order for a biofilm to develop, bacteria must be able to attach to a substrate, e.g. the wound bed. Once attached, the bacteria relinquish their planktonic state (free-floating) and recruit new members which can be of different species of bacteria (both aerobic and anaerobic species), fungi, or protozoa (Serralta et al, 2001). These biofilm colonies are dynamic, constantly changing and adapting to their environment. This adaptation requires that bacteria within biofilms communicate. Part of this communication process is known as quorum sensing (Mertz, 2003). This allows bacteria to access nutrients and dispose of waste rather than outgrow their resources or become poisoned by waste, giving a colony a unique ability to survive (Serralta et al, 2001).

Many biofilm-associated infections within the body have been shown to be unresponsive to antibiotic therapy. Comparisons of planktonic and biofilm *Staphylococcus aureus* has found that *S. aureus* biofilms may be 50 to 1000 times more resistant than planktonic or free-floating bacterial cells (Ceri et al, 1999).

The formation of biofilms is well established in industrial and dental research, but in the field of wound care, understanding of biofilms and their effect on wound healing is extremely limited. However, they seem to be a key component in resistant bacterial colonisation (Serralta et al, 2001). It certainly appears that

chronic wounds provide an environment capable of supporting the development of bacterial biofilms. However, further research is needed before it can be conclusively stated that biofilms are a threat to the wound healing process.

Wound infection is the result of a complex interaction between the individual's immune system, the wound conditions and the numbers and virulence of bacteria present (Thomson and Smith, 1994; Dow, 2001; Dowsett et al, 2004; Stotts, 2004; Best Practice Statement, 2010). If host defences are robust, bacterial proliferation is halted and the wound progresses to healing. However, if defences are weak, or bacterial virulence is high, proliferation continues, wound repair is halted, and eventually systemic sepsis occurs. Underlying medical problems such as poor blood supply, hypoxia and metabolic disorders are also contributing factors (Hunt and Hopf, 1997). The bacterial bioburden within the wound varies from simple contamination (where bacteria are present in the wound but are not multiplying and are held in check by the host's defence mechanisms), through colonisation to critical colonisation (where wound healing is interrupted), to local infection and finally systemic infection. This 'continuum of infection' (Kingsley, 2001; White et al, 2001) represents not only the establishment and proliferation of bacterial communities within the wound, but the ability of the host to mount a successful immune response to pathogenic ingress which is generally determined by clinical signs.

Antimicrobial/antibiotic agents

Antimicrobial is a term used to describe methods of eliminating or reducing bacterial load. Antimicrobial therapy includes the use of antibiotics and antiseptics.

The term 'antibiotic' is used to describe a substance or compound that kills bacteria or inhibits their growth and/or duplication. Most have a narrow band of effectiveness and, therefore, specific antibiotics are needed to treat particular bacteria species or strains. They can be administered orally, intravenously and, in some cases, topically.

Antiseptics are chemicals which are used to eliminate or reduce bacterial numbers on hard surfaces, on the skin and within wounds. They have an action on a broad spectrum of organisms including bacterium, protozoa, fungi and viruses. Some antiseptics can be toxic to human tissues (World Union of Wound Healing Societies [WUWHS], 2008a).

The presence of spreading infection has potential serious implications for patient well-being and appropriate systemic antibiotic therapy should be considered (European Wound Management Association [EWMA], 2006; WUWHS, 2008). The clinical diagnosis of wound infection was described by Cutting and Harding (1994) as:

- » Redness (erythema)
- » Swelling (oedema)
- » Localised heat
- » Pain
- » Limited function.

However, they expanded on this traditional view by stating that the following parameters should also be considered:

- » Discharge
- » Delayed healing
- » Wound breakdown
- » Pocketing at the base of the wound
- » Epithelial bridging
- » Unexpected pain or tenderness
- » Friable granulation tissue
- » Discolouration of the wound bed
- » Abscess formation.

This has been further refined within the WUWHS document (2008) to take into account the subtle differences in presentation that are observed between acute and chronic wounds of different aetiologies.

The presence of bacteria in acute or chronic wounds does not necessarily indicate that infection has occurred, or that it will lead to impaired wound healing (Kerstein, 1997; Dow et al, 1999). In many cases, identification of wound infection by laboratory methods can be inconclusive; the usefulness and significance of wound swabbing in the context of wound infection is still a subject of controversy. While a microbiological examination is indicated in the presence of 'classic signs' of infection (particularly in the acute wound), the results of these tests need to be considered within the context of a full clinical assessment before they are considered in therapeutic decision-making (WUWHS, 2008). Wound swabs can identify organisms present within wound fluid but may not identify the actual causative organism of infection, particularly in polymicrobial colonisation. Also, the accuracy of swabbing results depend on the techniques used and the speed with which samples are tested.

Managing wound bioburden

Wound infection is not just costly to the patient, it has serious financial implications for healthcare providers. The reduction of bacterial contamination to

the lowest level possible, along with the optimisation of healing potential through maintenance of an ideal wound environment and management of associated health-related issues, remain central to good wound care (WUWHS, 2008). For example, if the wound has a high necrotic burden, measures should be undertaken to facilitate wound debridement (EWMA, 2006; WUWHS, 2008).

Spreading infection can be life-threatening and so immediate action is required. Individuals should have blood cultures taken to identify the offending organism and to assess for differential diagnosis, and appropriate systemic antibiotic therapy should be implemented immediately (EWMA, 2006; WUWHS, 2008). Topical antimicrobial dressings should also be used to help reduce the wound bioburden (EWMA, 2006; WUWHS, 2008). Generally, systemic antibiotics are not recommended for wounds that only show signs of local infection (Bowler et al, 2001). In addition, topical antibiotics are linked to the development of bacterial resistance, therefore these should be avoided (EWMA, 2006; Melling et al, 2006).

Critical colonisation and localised, sub-clinical infection have also been recognised as significant factors in prolonged wound healing (Edwards and Harding, 2004; Warriner and Burrell, 2005), and effective management and treatment is identified as a central tenet when undertaking Wound Bed Preparation (WBP) (Schultz et al, 2003). In recent years, topical antimicrobial agents have come to represent the first line of treatment in the management of bacterial burden. This is particularly so in chronic wound care, as:

- ▶▶ They provide a high antimicrobial concentration at the site of infection (White et al, 2001; Cooper, 2004)
- ▶▶ They have bactericidal effects against multi-resistant organisms such as MRSA (Lawrence, 1998; Sibbald et al, 2001)
- ▶▶ They have the additional advantage that they do not interfere with the remainder of protective bacterial flora in other parts of the body
- ▶▶ They are less likely to produce an allergic reaction.

Once initiated, if the signs of infection subside and the patient shows no signs of systemic infection, the antimicrobial agent may be discontinued. If the wound continues to show signs of infection, a systemic antibiotic should be considered (EWMA, 2006). Similarly, a lack of a noticeable healing response within two weeks may necessitate the use of other topical or systemic agents (Bowler et al, 2001; Best Practice Statement, 2010). However, their use has to be targeted and measured, as widespread, inappropriate use increases healthcare costs with no outcome gain. The prophylactic use of antimicrobial preparations is controversial, and clinicians need to compare the clinical

benefit or treatment against the potential issues of increased cost and patient sensitivities/risk of systemic absorption. The use of these products can be justified in individuals whose immune capability is severely restricted, or where there is a high risk of infection, as the balance of risk swings strongly in favour of an active prophylactic management approach.

PRESSURE ULCERATION

Pressure ulcers are localised areas of tissue necrosis involving the skin and/or soft tissues (European Pressure Ulcer Advisory Panel [EPUAP], 2003) caused by the interplay of three mechanisms; pressure, friction and shear (Collier and Moore, 2006). Pressure on the skin and soft tissues leads to the compression of blood vessels and lymphatic drainage which results in build-up of metabolic waste and tissue ischaemia (Collier and Moore, 2006). If this is unrelieved, cell death occurs and necrotic material becomes evident as an ulcer. Friction to the surface of the skin strips away protective cornified epithelium, revealing delicate germinative cells unable to provide protection from bacterial ingress and moisture loss (Read, 2001). Friction is also central in shear damage; the high friction co-efficient that exists between the skin and most support surfaces, splints tissue preventing its movement. However, lateral, rotational and twisting forces distort tissues, stretching and damaging blood and lymphatic vessels which causes deep damage (Shear Force Initiative, 2006).

Although all individuals are faced with these forces, for most of us our body defence systems protect from damage, discomfort and pain by subconsciously prompting us to reposition, thereby relieving pressure on tissues. Lifting our bodies clear of support surfaces prevents rubbing and dragging, and our ability to maintain posture when sitting minimises drag on tissues. However, for some, reduced mobility places prolonged stress on tissues, absent or altered sensation means the warning signs of damage cannot be felt, and lack of muscle tone and function means self-initiated movement becomes difficult, if not impossible.

A myriad of risk assessment tools and documents have been developed to help identify those most at risk and offer guidance on strategies to prevent pressure ulceration. However, its occurrence is still commonplace. It is estimated that despite the efforts of healthcare providers in the UK, one in five patients will be affected by some form of pressure damage. Bennett et al (2004) estimated that pressure ulcers cost the NHS £2.1 billion per year; equivalent to the budget of the entire mental health service. This is a cost which cannot be tolerated.

The implementation of appropriate pressure-relieving surface strategies, maintenance of skin integrity and hygiene, optimisation of nutrition, and management of urinary and faecal incontinence all assist and are key to

preventing the development of pressure ulcers. However, prevention is all too often overlooked and treatment of damage needs to be implemented. This makes the assumption that all pressure ulceration can be avoided, yet changes within healthcare funding in the US have led clinicians to challenge this argument. Since 2009, US healthcare establishments no longer receive funding for the treatment of pressure ulcers which have developed while the patient has been in their care (Bergquist-Beringer et al, 2009). For many clinicians, not just in the US, damage occurs before hospitalisation due to deterioration in the patient's health status and failures in care provision from lack of specialist knowledge and expertise among the general population (Cox-Martin and Shaw, 2010). In addition, some patients experience multiple system-failure as part of their disease process. In such cases, prevention of all damage may be impossible to implement (Wound, Ostomy and Continence Nurses Society [WOCNS], 2009).

In an effort to control the growing problem of pressure ulcer development and to provide an evidence-based approach to prevention and treatment, collaboration between clinicians, academics and researchers across the globe has resulted in the development of the International Pressure Ulcer Guidelines (European Pressure Ulcer Advisory Panel-National Pressure Ulcer Advisory Panel [EPUAP-NPUAP], 2009). This four-year project was undertaken by a joint Guideline Development Group with representatives from both the US NPUAP and EPUAP who planned the guideline development process and reviewed all the documentation. To simplify the logistics, the EPUAP took the lead on the pressure ulcer prevention recommendations, and the NPUAP on the pressure ulcer treatment recommendations. Together, these documents form the basis of a structured approach to care and prevention which enables the sharing of good practice across the international healthcare community. As with all guidelines, there are limitations. These are expressed as:

- ▶▶ Guidelines are systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific clinical conditions. The recommendations may not be appropriate for use in all circumstances
- ▶▶ The decision to adopt any particular recommendation must be made by the healthcare professional in light of available resources and circumstances. Nothing contained in the guidelines is to be considered medical advice for specific cases
- ▶▶ As a result of the rigorous methodology used to develop the guidelines, the NPUAP and EPUAP believe that the research supporting these recommendations is reliable and accurate. However, the guideline development group do not guarantee the reliability and accuracy of individual studies referenced in the documents

- ▶▶ The guidelines and any recommendations they contain are intended for educational and informational purposes only
- ▶▶ The guidelines contain information that was accurate at the time of publication. Research and technology change rapidly and the recommendations contained in the guidelines may be inconsistent with future advances. Healthcare professionals are responsible for maintaining a working knowledge of research/technological advances that may affect their practice
- ▶▶ Generic names of products are provided. Nothing in the guidelines is intended as an endorsement of a specific product
- ▶▶ Nothing in the guidelines is intended as advice regarding coding standards or reimbursement regulations.

(Adapted from EPUAP/NPUAP guideline statement, 2009)

These guidelines build on previous work undertaken in wound care, including the EWMA Position documents: 'Wound bed preparation in practice' (2004), 'Identifying criteria for wound infection' (2005), 'Management of wound infection' (2006), 'Hard-to-heal wounds: a holistic approach' (2008) and the WUWHS 'Principles of best practice: Wound Infection in Clinical Practice: An International Consensus' (2008), and form the basis of the recommendations within this booklet.

Managing pressure ulcers

Once damage has occurred, the aims of care are to prevent further tissue breakdown, to optimise general health status and to provide the optimum wound environment to facilitate healing. Such remedial action is rarely as effective as prevention, as tissue has already been compromised and the extent of damage can seldom be fully assessed. However, if managed appropriately, steps can be taken to minimise damage and provide a wound environment where effective healing can take place.

Clinical presentation of pressure damage

The mechanisms of pressure ulcer damage affect different structures within the soft tissues and so the appearance of lesions can differ. Various systems have been introduced to grade tissue damage based on the clinical features of the wound. These assist in documenting damage but can also steer the clinician into identifying wound-related needs and prioritising interventions. Although no mandatory standard for grading pressure ulcers exists, the recent EPUAP/NPUAP document has led to broad acceptance of a I-IV system (EPUAP/NPUAP, 2009).

Table 1: EPUAP/NPUAP Classification 2009

Grade	Short description	Definition
Category/ Grade I	Non-blanchable erythema of intact skin	Intact skin with non-blanchable redness of a localised area, usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area. The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue. Category I may be difficult to detect in individuals with dark skin tones. May indicate 'at risk' persons
Category/ Grade II	Blister	Partial-thickness loss of dermis presenting as a shallow, open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled or serosanguinous filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising (indicating deep tissue injury). This category should not be used to describe skin tears, tape burns, incontinence-associated dermatitis, maceration or excoriation
Category/ Grade III	Superficial ulcer	Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunnelling. The depth of a category/stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have (adipose) subcutaneous tissue and category/stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep category/stage III pressure ulcers. Bone/ tendon is not visible or directly palpable
Category/ Grade IV	Deep ulcer	Full-thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present. Often includes undermining and tunnelling. The depth of a category/stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Category/stage IV ulcers can extend into muscle and/or supporting structures (e.g. fascia, tendon or joint capsule) making osteomyelitis or osteitis likely to occur. Exposed bone/muscle is visible or directly palpable

Management of established pressure damage

The recent international collaboration between clinicians, academics and researchers in pressure ulcer prevention, management and treatment has extended beyond ulcer classification. If real change is to be achieved,

standardisation of terminology and approaches to pressure ulcer care needs to be agreed upon. Best practice is reached through assessment of the available evidence and the implementation of care regimens based on proven efficacy.

Wound dressings are a central component of pressure ulcer care. Selection of dressings should be based on the tissue in the ulcer bed, the condition of the skin around the ulcer bed, and the goals of the person with the ulcer.

Patients invariably have a number of comorbidities which have contributed to the development of their pressure ulcers. Although the initial damage may have resulted from an acute episode, these individuals are likely to exhibit the signs of chronic wound healing and, unless correctly managed, bacterial bioburden is likely to be high, as are proteases. Research has shown that in the chronic wound we see changes within wound exudate which have a detrimental effect on the healing process, decreasing mitogenic activity, increasing inflammation and accelerating protease activity when compared to acute wounds (Staiano-Coico et al, 2000).

It is important for clinicians to manage the wound if progression to healing is to be accomplished. This can be achieved by the adoption of a Wound Bed Preparation (WBP) approach to wound management. The concept of WBP has gained international recognition as a framework that can provide a structured approach to wound management. By definition, WBP is the management of a wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures (Falanga, 2000; Schultz et al, 2003; EWMA, 2004). The concept focuses the clinician on optimising conditions at the wound bed so as to encourage normal endogenous healing (Dowsett, 2008). The mnemonic TIME is frequently used as a summary of the main focus within WBP:

- T** – represents the tissue types in the wound itself. Is it non-viable or healthy?
- I** – refers to the presence or absence of infection or inflammation
- M** – addresses the issue of moisture balance, and avoiding dessication or maceration
- E** – is epithelial (edge) advancement. Is this non-advancing or non-migrating? The aim is to promote wound closure and therefore it is essential to remove the barriers to this. Source: Schultz et al, 2003

T — Tissue

Within the pressure ulcer significant levels of necrotic tissue may be present in the wound both from the breakdown of tissues damaged by the original injury, and from subsequent bacterial colonisation and infection. The aim of management is to debride non-viable tissue which may act as a focus for bacterial proliferation and chronic inflammation. This may be present within

I — Infection/inflammation

Bacteria are present on all skin surfaces, however; when the skin is breached, bacteria can enter the body through the wound and, dependent on the numbers and/or virulence of the organism, cause damage both within the wound and systemically, resulting in serious systemic infections such as cellulitis, fasciitis, osteomyelitis, or sepsis. As said, many individuals who develop pressure ulcers have multiple comorbidities which increase their risk of infection. Systemic infection is uncommon in category/stage I or II ulcers, although the development of a critically colonised state can seriously delay healing in these patients. Infection is more likely to occur in individuals with category/stage III and IV ulcers. To avoid the serious consequences of infection, healthcare professionals should focus on identification of high-risk individuals, prevention, early detection, and prompt, effective treatment of pressure ulcer infection. The clinician should suspect infection in pressure ulcers that:

- » Have a high necrotic tissue burden or a foreign body present
- » Have been present for a long period of time
- » Are large in size or deep
- » Are likely to be repetitively contaminated (e.g. near the anus).

Patients particularly at risk include those with:

- » Diabetes mellitus
- » Malnourishment
- » Hypoxia or poor tissue perfusion
- » Auto-immune disease
- » Immune-suppression.

Careful assessment of the wound should be undertaken to identify signs of local infection and/or critical colonisation. As previously stated, the taking of wound swabs in the absence of clinical signs of infection may lead to the inappropriate use of systemic antibiotics.

Wherever possible, the patient's general health should be stabilised and optimised to maximise the host's immune response. The risk of infection should be reduced by:

- » Preventing contamination of the pressure ulcer
- » Debriding necrotic tissue, slough, eschar (and biofilm) as required
- » Minimising bacterial load in the ulcer.

Assess pressure ulcers at every dressing change to confirm the appropriateness of the current dressing regimen and to evaluate the effectiveness of the intervention. The plan of care should guide usual dressing wear times and contain plans for dressing changes, as needed, due to soiling, loosening, etc. Select dressings that keep the wound bed moist, keep the periwound dry and prevent maceration, and which remain in contact with the wound bed or skin barrier product.

If exudate levels are low, dressings capable of donating moisture are indicated to prevent desiccation of the wound bed. Hydrogel dressings may be used on cavity wounds or shallow, minimally exudating or dry pressure ulcers to facilitate autolysis. Consider the use of hydrogel sheet dressings for pressure ulcers without depth and contours, and/or on body areas that are at risk of dressing migration. Amorphous hydrogels are better suited for the treatment of pressure ulcers with depth and contours, and which are not infected. Other 'moisture-donating' dressings may also be suitable for use.

Dressings with higher absorption capacity should be used to prevent maceration and leakage in moderate to highly exudating wounds, as these can compromise the periwound environment. Alginate and Hydrofiber® dressings may be of assistance in managing exudate. Foam dressings may be beneficial in managing exudating category/stage II and shallow category/stage III pressure ulcers. Avoid using small pieces of foam in exudating cavity ulcers due to the risk of accidentally leaving them within the wound at dressing change.

Collagen matrix dressings and matrix metalloproteinases (MMP) modulating dressings may be considered for the treatment of non-healing category/stage III and IV pressure ulcers.

Consider amending the planned dressing-change interval or changing the type of dressing if the absorbent dressing is still dry at the scheduled time for dressing change, or if leakage occurs. Absorbent dressings can be used in infected pressure ulcers when there is concurrent treatment of infection. Foam dressings can be used on painful pressure ulcers. Some foam dressings may be of use in protecting body areas and pressure ulcers at risk of shear injury.

Gauze dressings should be avoided for clean, open pressure ulcers because they are labour-intensive, cause pain when removed and lead to desiccation of viable tissue if they dry out. Practice varies widely in relation to gauze dressings. Increased infection rates, retained dressing particles and pain have led professionals to avoid their use for open chronic wounds, such as pressure ulcers, in favour of advanced wound dressings. Gauze dressings today are primarily used as surgical dressings. Due to the need for frequent changes they have been shown to be costly in professional time. In the rare event

individuals. Urgent surgical debridement and drainage may be required for those with life-threatening spreading infection. The surgical technique employed will depend on the unique needs of the patient, and will be determined by the surgeon in consultation with the patient and the wider care team. The surgical technique employed will take into consideration the potential for osteomyelitis and will be planned to facilitate good postoperative recovery and rehabilitation.

CASE REPORTS

Managing a sacral pressure ulcer with an antimicrobial dressing

Pam Cooper, David Gray, Fional Russell and Sandra Stringfellow are Clinical Nurse Specialists; Melvyn Bertram, Kristin Duguid and Gail Pirie are Tissue Viability Nurses all at the Department of Tissue Viability, NHS Grampian, Aberdeen



Figure 1. Pressure damage has occurred but the wound has still to demarcate.

This case report features a 76-year-old male patient who presented to the tissue viability department with a large pressure ulcer on his sacrum. He was admitted to the intensive care unit having collapsed at home, where he lay overnight. The patient had a history of cardiac disease, respiratory failure and sepsis.

On initial assessment, the patient was on artificial ventilation. The ulcer was on artificial ventilation. The ulcer covered an area measuring 13x7cm and comprised red, hard non-blanching tissue with a central plug of black yellow tissue. The wound

needed to demarcate clearly in order to determine the extent of underlying tissue damage (*Figure 1*). As the ulcer was being reviewed immediately post trauma, the full extent and degree of tissue necrosis that would occur was still unclear. The team were also having difficulty in maintaining any dressing regimen as the patient was incontinent of faeces.

Assessment

Over a period of five months the patient's overall medical condition stabilised and improved. However, due to the extent of underlying tissue involvement the ulcer debrided centrally, revealing a wound measuring 3.5x3x0.2cm. It presented as 100% granulation tissue, which was pale in colour and failed to respond to

the treatment regimens being used (Figure 2). The decision was taken to start Suprasorb® X+PHMB (Activa Healthcare, an L&R Company) secured with a secondary foam dressing to absorb any excess exudate. It was hoped that the antimicrobial properties of Suprasorb X+PHMB would kick start the healing phase and encourage the promotion of granulation tissue.

Over a period of two weeks the dressing was changed every 3–4 days. On review, the wound dimensions had reduced slightly to 3.2x3x0.1cm, but of more clinical significance was the presence of friable, red granular tissue (Figure 3), which was actively responding to treatment.

During this time the patient was also diagnosed with *Clostridium difficile*, for which he was being treated. However, this led to an increased frequency of dressing changes and a number of episodes of wound contamination.

Conclusion

Over the 15 days of treatment with Suprasorb X+PHMB the team saw the wound positively respond and develop healthy granulation tissue. The dimensions of the wound reduced slightly, and the treatment also prevented any deterioration or infection developing in a wound that was frequently contaminated with faecal enzymes.

Management of a grade III pressure ulcer on the foot of an 89-year-old female patient

Gill Wicks is Nurse Consultant, Tissue Viability, Wiltshire Primary Care Trust and Lecturer, University of the West of England, Bristol

An 89-year-old patient had a grade III pressure ulcer of eight weeks' duration



Figure 2. Granulation tissue appears pale in nature and is not actively improving.



Figure 3. The wound presented with red granular tissue.



Figure 4. Grade III pressure ulcer of eight weeks' duration at initial presentation.




Figure 5. Wound after 18 days of treatment with Suprasorb X+PHMB. The wound had filled with granulation tissue and reduced in size.

on her foot (Figure 4). The granulation tissue appeared healthy but was not progressing. The patient was bed bound, nursed on an alternating mattress and her foot was fully offloaded. She had no concurrent illnesses but was taking supplementary nutrition. She was unable to state a pain score, but made clear verbal indications that dressing changes were painful.

A variety of dressings had been used previously, and a hydrogel had initially succeeded in debriding the wound. However, the wound had not progressed for four weeks, despite the use of Hydrofiber and cadexomer iodine dressings for two weeks respectively.

Suprasorb X+PHMB was considered because Moore and Gray (2007) suggested that PHMB reduces wound bioburden. A foam was used as the secondary dressing. After 18 days, the wound bed had filled with granulation tissue and the wound reduced in width (Figure 5). Dressing

change frequency reduced from three times a week to twice-weekly. In addition, dressing changes appeared painless and stress free. The evaluation stopped at this point as this patient was admitted to hospital with an unrelated illness.

This case report first appeared in Glover D, Wicks G (2009) Suprasorb® X+PHMB: the clinical evidence. In: *A new solution to managing wound pain and infection: Suprasorb® X+PHMB*. *Journal of Wound Care* 18(Suppl 11): 1–24. It is reproduced by kind permission of the *Journal of Wound Care*. 

SUPRASORB® X+PHMB

Moisture management and bacterial control are two of the fundamental issues in wound management. The new dressing, Suprasorb® X+PHMB

(Activa Healthcare, an L&R Company) has been specifically designed to deal with these two issues simultaneously. Suprasorb X+PHMB is made up of a unique structure composed of biosynthetic HydroBalance fibres. These fibres are the products of a cellulose fermentation process using *Acetobacter xylinum*. The bacteria produce a mesh structure of cellulose fibrils which are 200 times finer than cotton, giving the material an exceptionally high surface area with enhanced moisture-handling capabilities and tensile strength. As a result of the biosynthetic HydroBalance fibres, the dressing is able to regulate the absorption and donation of moisture at the wound-dressing interface. Depending on the status of the wound, surplus exudate can be absorbed by the dressing, or moisture donated to provide an ideal moist wound healing environment.

This ability to balance moisture levels can occur within the same wound dressing, removing exudate from one area and donating moisture to others. In addition, the dressing contains the potent antimicrobial PHMB 0.3%. The PHMB component exerts its antimicrobial effects both within the dressing and also at the wound-dressing interface. As the PHMB is not bound to the HydroBalance fibres of the dressing, it is released into the wound fluid along a concentration gradient. The presence of fluid in the dressing means that antimicrobial activity is possible even on dry wounds (unlike silver-based antimicrobial dressings).

Mosti et al (2008) and Galitz et al (2009) found that use of Suprasorb X+PHMB saw a decrease in patient-reported pain at dressing change. This was matched by a reduction of background pain following use. Galitz et al (2009) showed this to be significant ($p < 0.05$) after the first day of use, and considered this to be a notable feature of the dressing's performance.

Suprasorb X+PHMB dressings are indicated for use on lightly to moderately exuding, superficial and deep, critically colonised and infected wounds in all stages of wound healing (Kingsley et al, 2009).

What is PHMB?

The antiseptic polyhexamethylene biguanide is a mixture of polymers, structurally similar to the naturally-occurring antimicrobial peptides which support the innate immune response and protect against infection. While the precise action of PHMB on bacteria is unclear, the primary targets appear to be the outer and cytoplasmic membranes of bacterial cells. PHMB adheres to bacterial cell membranes, causing them to leak potassium ions and other cytosolic components which results in cell death. There is evidence that once in the bacterial cell, PHMB also binds to DNA and other nucleic acids, damaging or

inactivating them. As PHMB changes the bacterial cell membrane, once inside it cannot be removed by the bacterial defence system (Kingsley et al, 2009). PHMB is also effective at controlling fungal colonies (Shah, 2000; Lee et al, 2004), but does not adhere to healthy cell membranes and has shown no evidence of toxic effect on human cells (Ikeda et al, 1983; Moore and Gray, 2007).

Use of PHMB

PHMB has been in use as an antiseptic and disinfectant for approximately 60 years, with proven effectiveness against a broad number of bacterial and fungal species (Moore and Gray, 2007) and rapid and sustained action. It has been demonstrated to be effective at biofilm management with no evidence of bacterial resistance or systemic absorption. Comparative tests of PHMB's biocompatibility (the measurement of an antiseptic agent's activity in relation to its cytotoxicity) against other commonly used therapies have demonstrated its superiority to chlorhexidine, povidone-iodine, triclosan, silver and sulfadiazine (Müller and Kramer, 2008). Studies have shown that skin sensitising to PHMB is very low even in high concentration (Schnuch et al, 2000; 2007).

Recently, PHMB has been introduced into wound management within a range of wound care products. In some cases, the PHMB molecule is chemically bound to the base material, providing dressings with antimicrobial properties when in contact with wound moisture. These products protect against the development of wound infection by decreasing the bacterial load in the dressing and preventing bacterial ingress. In other products, the active component is free to be delivered into the wound and periwound tissues; the dressing in this case being a carrier for a wider antimicrobial activity by donating PHMB to the wound itself.

PHMB has also been shown to have positive effects on wound healing. *In vitro* and *in vivo* studies have shown that PHMB:

- ▶▶ Reduces wound pain rapidly and effectively (Daeschlein et al, 2007; Galitz et al, 2009)
- ▶▶ Reduces wound odour (Daeschlein et al, 2007)
- ▶▶ Increases granulation tissue formation (Mueller and Krebsbach, 2008)
- ▶▶ Increases keratinocyte and fibroblast activity (Wiegand et al, 2008a)
- ▶▶ Reduces slough within the wound (Mueller and Krebsbach, 2008)
- ▶▶ Reduces MMP-induced periwound breakdown (Cazzaniga et al, 2002; Werthen et al, 2004)
- ▶▶ Assists in removing non-viable tissue (Kaehn, 2009).

PHMB is indicated for the control of bacterial burden within wounds.

Specifically, it is used to reduce bacterial burden in the critically colonised wound and may be indicated as infection prophylaxis in immunocompromised individuals. Adjunct therapy with PHMB should also be considered to systemic treatment when treating serious wound sepsis. As with all topical antimicrobial therapies, if the wound is unchanged after ten days or deteriorates, alternative antimicrobial strategies should be considered (including systemic antibiotics). In most cases, treatment should not extend beyond 14 days unless previously agreed by a local specialist (Best Practice Statement, 2010).

PHMB's ability to effectively bind to proteins is a key feature of its success as an environmental disinfectant. In wound care, clinicians should choose wound care products which are appropriate to patient needs, be they as barriers to bacterial spread (preventing bacterial ingress or cross-contamination from colonised wounds), or as 'donating' dressings, which are also able to disperse PHMB into the wound.

In addition to wound dressings containing PHMB, wound irrigation fluid containing PHMB is also available, however, studies indicate that solution concentration should be between 0.01%–0.04% (depending on clinical need) (Dissemond et al, 2010), and contact between the bacterium and PHMB needs to be maintained for 10–15 minutes to ensure maximum antibacterial action. Continuous irrigation is possible, although clinicians need to be aware of the technical and practical issues that might arise, particularly in community settings.

The use of PHMB has specific contraindications. PHMB must not be used:

- ▶▶ For peritoneal lavage
- ▶▶ For antiseptic joint lavage (cartilage toxicity)
- ▶▶ In applications involving any part of the central nervous system (CNS), including the meninges and intralumbal applications
- ▶▶ For applications involving the middle or inner ear, or intraocular applications
- ▶▶ During the first four months of pregnancy (at any time thereafter, a strict benefit/risk assessment has to be performed),
- ▶▶ In patients allergic to PHMB (Dissemond et al, 2010).

As can be seen, apart from a very small minority of patients who fall within the last two groups, PHMB does not have any contraindications for application within the pressure ulcer population.

Health economics and cost-effectiveness

Cost-effectiveness of treatments has to be taken into account when implementing new therapies. There has been considerable concern raised over the cost of

pressure ulceration in the UK (Bennett et al, 2004) and it is essential that steps are taken to bring this expenditure under control. Ideally, all pressure ulcers would be prevented by early intervention, but as already stated, a number of patients present to clinicians with damage already present. In addition, some individuals will develop damage despite clinicians' best endeavours due to worsening health status. In calculating total costs of pressure ulceration it is essential to consider the cost of treating existing pressure damage.

The health economics of pressure ulcer treatment is currently in its infancy but is set to take a higher priority due to financial constraints on public sector expenditure. In June 2010, the recently elected coalition Government's Health Secretary, Andrew Lansley, announced an intention to reduce the half million annual readmissions to hospitals. Under the new plans, hospitals would receive funding for the first hospital stay plus treatment for the 30 days post discharge, thus giving hospitals the responsibility for a patient's health and wellbeing following hospital treatment, not the GPs and PCTs as is currently the case. Therefore, the management of hospital-acquired infections arising within 30 days of admission is likely to have a much greater impact on hospital funding with readmission within 30 days resulting in financial penalties for acute trusts. Readmission due to infected pressure ulcers does occur, although figures on the incidence of this are unavailable. However, in the wider context of pressure ulcer costs, it appears likely that further targeting of 'avoidable' expense is set to be announced — given the attention that pressure ulcers attract, this is likely to be one of those key areas.

Health economics assesses the cost of treatment implementation against measurable health gains, identifying where cost savings can be realised. If pressure ulceration is seen as avoidable, focus will be placed on this domain. For clinicians, speedy healing of pressure ulcers is essential if reductions in health costs are to be realised. Wound care interventions need to demonstrate that they can manage the side-effects of pressure ulceration, such as exudation and pain, and also prevent wound complications which will extend the healing process. A key factor in healing rates is the presence of infection. Local infection and critical colonisation lead to reduction in healing potential and the development of chronic wound stasis. It is therefore essential to manage bacterial wound burden.

In other situations, such as in the management of surgical site infection (SSI), the use of PHMB-based wound care products has been shown to have a marked impact on reducing infection rates and costs to healthcare providers (Gilliver, 2009). Although empirical evidence is currently unavailable in pressure ulcer care, it would seem reasonable to conclude from the reduction in pain found in studies, the increased healing rates and effectiveness in bacterial

management in pressure ulcer care and in other situations, that PHMB offers a cost-effective form of treatment. It is predicted that such opinion will be supported by firm data in the near future.

In summary, PHMB has a number of properties and characteristics which make it particularly appropriate for use in critically colonised and locally infected acute and chronic wounds, namely:

- ▶▶ Proven broad antimicrobial action (Cazzaniga et al, 2002; Wright et al, 2003; Eberlein and Wild, 2008; Mosti et al, 2008; Müller and Kramer, 2008; Mueller and Krebsbach, 2008; Kaehn, 2009; Wild et al, 2009)
- ▶▶ Anti-fungal activity (Shah, 2000; Lee et al, 2004)
- ▶▶ Minimum blood/protein inactivation (reduction of effect on mucous membranes due to presence of mucin) (Ansorg et al, 2002)
- ▶▶ Sustained, post-application effect (Rosin et al, 2002)
- ▶▶ Established promotion of wound healing (depending on concentration) (Davies and Field, 1969; Kramer et al, 2004; Daeschlein et al, 2007; Wiegand et al, 2008a, b)
- ▶▶ Additional anti-inflammatory properties
- ▶▶ No development of resistance reported to date (Gilliver, 2009; Weigand et al, 2009)
- ▶▶ Reduction of biofilm (Harbs and Siebert, 2007) and fibrin (Körber et al, 2008)
- ▶▶ Good clinical safety (Disch et al, 2007; Mulder et al, 2007; Bruckner et al, 2008)
- ▶▶ Targeted action on bacterial cells (Ikeda et al, 1983; 1984)
- ▶▶ Biocompatibility index > I (Müller and Kramer, 2008)
- ▶▶ No known risks of adsorption (Kramer and Roth, 2008)
- ▶▶ No known toxic risks (Moore and Gray, 2007)
- ▶▶ Low risk of contact sensitisation (Schnuch et al, 2000; 2007).

PHMB offers a new method of bacterial control which has been proven safe, efficient and cost-effective. This will provide benefits to patients and clinicians in providing alternative and additional tools to manage bacterial burden within the wound care environment.

CONCLUSION

Clinicians are frequently faced with complex wounds in patients with poor healing potential, reduced mobility and compromised immune status. Wounds in these circumstances not only take time to heal and are costly to healthcare providers, but pose a real threat to the patient when wound infection occurs.

When faced with pressure ulcers, clinicians need to adopt treatment strategies which are able to provide the optimum moist wound healing environment and reduce both pain and bacterial load, thus lessening the risk of bioburden interfering with the healing process or precipitating life-threatening sepsis.

Suprasorb X+PHMB offers the clinician a product which is safe and effective; which can modulate wound moisture, has proven ability to reduce pain and can deliver antimicrobial activity to the wound.

REFERENCES

- Ansorg RA, Azem T, Fabry WH, Rath PM (2002) Influence of mucin on the activity of the antiseptic Lavasept against *Staphylococcus aureus*. *Chemotherapy* 48(3): 129–33
- Bennett G, Dealey C, Posnett J (2004) The cost of pressure ulcers in the UK. *Age Ageing* 33: 230–5
- Best Practice Statement: The use of topical antiseptic/antimicrobial agents in wound management (2010). Wounds UK, Aberdeen
- Bergquist-Beringer S, Davidson J, Agosto C, et al (2009) Pressure ulcer treatment. Clinical Practice Guideline No 15 (AHCPR Publication no. 95-0652) Rockville, MD. US Dept. Of Health and Human Services, Public Health Services, Agency for Health Care Policy and Research
- Bowler PG, Duerden BI, Armstrong DG (2001) Wound microbiology and associated approaches to wound management. *Clin Microbiol Rev* 14: 244–69
- Bruckner M, Schwarz C, Otto F, Heillinger J, Wild T (2008) Evaluation of cellulose and polyhexamethylene biguanide (Suprasorb X+PHMB) in therapy of infected wounds. Poster presentation, Wounds UK Conference, Harrogate. Available online at: www.activahealthcare.co.uk/pdf/SXP006.pdf (last accessed April 2010)
- Cazzaniga A, Serralta V, Davis S, Orr R, Eaglstein W, Mertz PM (2002) The effect of an antimicrobial gauze dressing impregnated with 0.2-percent polyhexamethylene biguanide as a barrier to prevent *Pseudomonas aeruginosa* wound invasion. *WOUNDS* 14(5): 169–76
- Ceri H, Olson ME, Stremick C, et al (1999) The Calgary biofilm device: New technology for rapid determination of antibiotic susceptibilities of bacterial biofilms. *J Clin Microbiol* 37(6): 1771–6
- Collier M, Moore Z (2006) Etiology and risk factors. In: Romanelli M, Clark M, Cherry G, Colin D, Defloor T, eds *Science and Practice of Pressure Ulcer Management*. Springer-Verlag, London: 4: 27–36

- RG, eds. *Chronic wound care: a clinical source book for healthcare professionals*. 3rd edn. HMP communications Wayne, PA: 385–6
- Dow G, Browne A, Sibbald RG (1999) Infection in chronic wounds: controversies in diagnosis and treatment. *Ostomy Wound Manage* 45: 23–40
- Dowsett C (2008) Using the TIME framework in wound bed preparation. *Br J Community Nurs* 13(6 Suppl): S15–S22
- Dowsett C, Edwards-Jones V, Davies S (2004) Infection control for wound bed preparation. *Br J Community Nurs* 9(9s): 12–17
- Eberlein Th, Wild Th (2008) Approaches to therapy and an outlook on improving the clinical data situation, with a special focus on the polihexanide-containing Suprasorb X+PHMB Hydrobalance dressing. EWMA conference extended abstract. Available online at: www.activahealthcare.co.uk/pdf/SXP009.pdf (last accessed April 2010)
- Edwards R, Harding KG (2004) Bacteria and wound healing. *Curr Opin Infect Dis* 17(2): 91–6
- European Pressure Ulcer Advisory Panel (2003) European Pressure Ulcer Advisory Panel. Report from the guideline development group. *EPUAP Rev* 5(3): 80–2
- European Pressure Ulcer Advisory Panel/National Pressure Ulcer Advisory Panel (2009) *Clinical Practice Guidelines: Pressure Ulcer Prevention and Treatment*. Washington DC
- European Wound Management Association (2004) Position Document: *Wound bed preparation in practice*. MEP Ltd, London.
- European Wound Management Association (2005) Position Document: *Identifying criteria for wound infection*. MEP Ltd, London
- European Wound Management Association (2006) Position Document: *Management of wound infection*. MEP Ltd, London
- European Wound Management Association (2008) Position Document: *Hard-to-heal wounds: a holistic approach*. MEP Ltd, London
- Falanga V (2000) Classification for wound bed preparation and stimulation of chronic wounds. *Wound Rep Regen* 8(5): 347–52
- Galitz C, Hämmerle G, Signer M, Traber J, et al (2009) Polihexanide versus silver wound dressings — first interim results of a controlled, randomized, prospective, multicentric study. Poster. European Wound Management Association (EWMA) Helsinki/FIN, 20–22 May 2009. *EWMA Journal Supplement* 9(2): 171

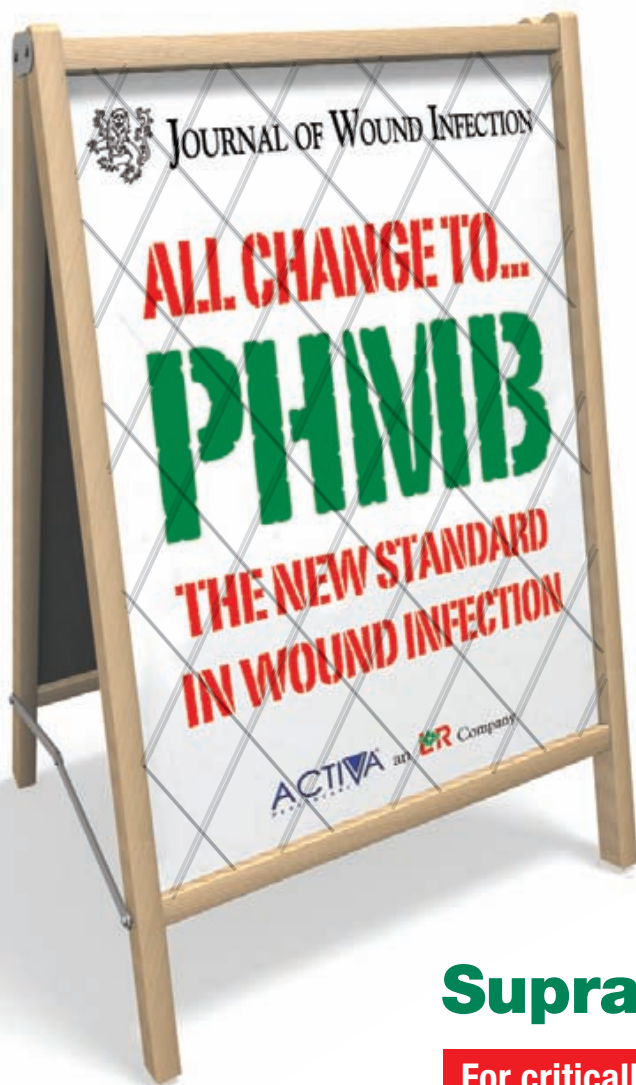
- Gilliver S (2009) PHMB: a well-tolerated antiseptic with no reported toxic effects. *J Wound Care* Activa Healthcare Supplement: S9–14
- Gray D, Barrett S, Battacharyya S, et al (2010) *PHMB and its potential contribution to wound management*. Wounds UK, Aberdeen
- Grocott P (1998) Exudate Management in fungating wounds. *J Wound Care* 7(9): 445–8
- Hansson C, Hoborn J, Möller A, Swanbeck G (1995) The microbial flora in venous leg ulcers without clinical signs of infection: repeated culture using a validated standardised microbiological technique. *Acta Derm Venereol* 75(1): 24–30
- Harbs N, Siebert J (2007) *In vitro* efficacy of octenidine and polihexanide against biofilms composed of *Pseudomonas aeruginosa*. *GMS Krankenhaushyg Interdiszpl* 2(2): Doc.45 (20071228)
- Hunt TK, Hopf HW (1997) Wound healing and wound infection. What surgeons and anaesthesiologists can do. *Surg Clin North Am* 77: 587–606
- Ikeda T, Ledwith A, Bamford CH, Hann RA (1984) Interaction of a polymeric biguanide biocide with phospholipid membranes. *Biochim Biophys Acta* 769(1): 57–66
- Ikeda T, Tazuke S, Watanabe M (1983) Interaction of biologically active molecules with phospholipid membranes. I. Fluorescence depolarization studies on the effect of polymeric biocide bearing biguanide groups in the main chain. *Biochim Biophys Acta* 735(3): 380–6
- Kaehn K (2009) An *in-vitro* model for comparing the efficiency of wound rinsing solutions. *J Wound Care* 18(6): 229–36
- Kerstein MD (1997) The scientific basis of healing. *Adv Wound Care* 10: 30–6
- Kingsley A (2001) A proactive approach to wound infection. *Nurs Standard* 15(30): 50–8
- Kingsley A, Tradej M, Colbourn A, Kerr A, Bree-Aslan C (2009) Suprasorb X + PHMB: antimicrobial and HydroBalance action in a new wound dressing. *Wounds UK* 5(1): 72–7
- Körber A, Seipp H-M, Hofmann S (2008) Efficacy of different wound dressings on artificial plaques of fibrin. *EWMA J (Suppl)* 8(2): 261
- Kramer A, Roth B, Muller G, Rudolph P, Klocker N (2004) Influence of the antiseptic agents polihexanide and octenidine on FL-cells and on healing of experimental superficial aseptic wounds in piglets. A double-blind, randomised, stratified, controlled, parallel-group study. *Skin Pharmacol Physiol* 17: 141–6

- Kramer A, Roth B (2008) Polihexanid. In: Kramer A, Assadian O (Hrsg) *Wallhäußers Praxis der Sterilisation, Desinfektion, Antiseptik und Konservierung*. Thieme, Stuttgart: 789–93
- Landis S, Ryan S, Woo K, Sibbald RG (2007) Infections in chronic wounds. In: *Chronic wound care: a clinical source book for healthcare professionals*. 4th edn. HMP communications Wayne, PA: 299–32
- Lawrence JC (1998) The use of iodine as an antiseptic agent. *J Wound Care* 7: 421–5
- Lee WR, Tobias KM, Bemis DA, Rohrbach BW (2004) *In vitro* efficacy of a polyhexamethylene biguanide-impregnated gauze dressing against bacteria found in veterinary patients. *Vet Surg* 33(4): 404–11
- Melling AC, Gould FK, Gottrup F (2006) Topical antimicrobials and surgical site infection. In: EWMA Position Document: *Management of wound infection*. MEP, London
- Mertz P (2003) Cutaneous biofilms: friend or foe? *Wounds* 15(5): 129–32
- Moore K, Gray D (2007) Using PHMB antimicrobial to prevent wound infection. *Wounds UK* 3(2): 96–102
- Mosti G, Mattaliano V, Schmitz M, Abel M (2008) Successful therapy of critically-colonised or locally infected wounds with a new HydroBalanced biocellulose-based wound dressing with polihexanide on out-patients. Poster presentation, Wounds UK Conference, Harrogate. Available online at: www.activahealthcare.co.uk/pdf/SXP003.pdf (last accessed April 2010)
- Mueller SW, Krebsbach LE (2008) Impact of an antimicrobial-impregnated gauze dressing on surgical site infections including methicillin-resistant *Staphylococcus aureus* infections. *Am J Infect Control* 36(9): 651–5
- Mulder GD, Cavorsi JP, Lee DK (2007) Polyhexamethylene biguanide (PHMB): an addendum to current topical antimicrobials. *WOUNDS* 19(7): 173–82
- Müller G, Kramer A (2008) Biocompatibility index of antiseptic agents by parallel assessment of antimicrobial biguanide mouthrinse compared to an essential oil rinse and a chlorhexidine rinse on bacterial counts and 4-day plaque regrowth. *J Clin Periodontol* 98: 392–9
- Phillips P, Taylor L, Hastings R (2009) Silver ion antimicrobial technology: decontamination in a nursing home. *Br J Community Nurs* 14(6): S25–S29
- Posnett J, Franks PJ (2007) *The costs of skin breakdown and ulceration in the UK. The silent epidemic*. The Smith & Nephew Foundation, Hull
- Posnett J, Gottrup F, Lundgren H, Saal G (2009) The resource impact of wounds on

- Weigand C, Abel M, Ruth P, Hipler UC (2009) No resistance against polihexanide after repetitive incubation with *Staphylococcus aureus* for 100 days. Paper presented at 5th Joint Meeting of the European Tissue Repair Society (ETRS) and Wound Healing Society 2009. Available online at: www.activahealthcare.co.uk/pdf/SXP013.pdf
- Werthen M, Davoudi M, Sonesson A, et al (2004) *Pseudomonas aeruginosa*-induced infection and degradation of human wound fluid and skin proteins *ex vivo* are eradicated by a synthetic cationic polymer. *J Antimicrob Chemother* 54(4): 772–9
- White RJ, Cooper RA, Kingsley A (2001) Wound colonization and infection: the role of topical antibacterials. *Br J Nurs* 10: 563–78
- White RJ, Cutting KF (2003) Interventions to avoid maceration of the skin and wound bed. *Br J Nurs* 12(20): 1186–1203
- Wiegand C, Abel M, Kramer A, Müller G, Ruth P, Hipler U-C (2008a) Viability and proliferation of fibroblasts, keratinocytes and HaCaT-cells influenced by polihexanide. Poster presentation Wounds UK Conference, Harrogate. Available online at: www.activahealthcare.co.uk/pdf/SXP004.pdf (last accessed April 2010)
- Wiegand C, Abel M, Ruth P, Hipler U-C (2008b) Protective effect of polihexanide on HaCaT keratinocytes in co-culture with *Staphylococcus aureus*. Poster presentation at Wounds UK Conference, Harrogate. Available online at: www.activahealthcare.co.uk/pdf/SXP008.pdf (last accessed April 2010)
- Wild Th, Bruckner M, Payrich M, Schwarz Ch, Eberlein Th (2009) Prospective randomized study for eradication of MRSA with polihexanide containing biocellulose dressing compared with polihexanide containing wound solution. Poster. European Wound Management Association (EWMA) Helsinki/FIN, 20–22 May 2009. *EWMA Journal Supplement* 9(2): 170
- Wright JB, Lam K, Olson ME, Burrell RE (2003) Is antimicrobial efficacy sufficient? A question concerning the benefits of new dressings. *Wounds* 15(3) Available online at: www.woundsresearch.com/article/1583 (accessed April 2010)
- World Union of Wound Healing Societies (2008) *Principles of best practice: Wound Infection in Clinical Practice: An International Consensus*. MEP Ltd, London
- Wound, Ostomy and Continence Nurses Society (2009) Position paper: Avoidable versus unavoidable pressure ulcers. Available online at: www.wocn.org/AboutUs/News/37/

XTRA! - XTRA!
Effective against MRSA & VRE
read all about it...

Powerful and Gentle
reduces wound pain
Does not contain silver



Suprasorb® X + PHMB

For critically colonised & infected wounds



For more information log onto www.activahealthcare.co.uk
or call our customer care line: 08450 606707

Available on
Drug Tariff

M444 V1.1 - July 2010

