Surgical Site Wounds and Suprasorb® X+PHMB
Infection, prevention and treatment
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- Leg ulcers
- Surgical site wounds
- Trauma wounds
- Pressure ulcers
- Diabetic foot ulcers
INTRODUCTION

Healthcare spending as a proportion of gross domestic product (GDP) will decrease over the next few years as the economic downturn continues. The prevalence of chronic wounds, especially diabetic foot wounds, pressure areas and venous ulcers, is set to rise and will be compounded by an increasingly obese, aging population. Clinicians will be tasked with reducing healthcare spend by clinical and cost-effectiveness. Innovations in wound care products will be interrogated by financial directors and business plans for implementation will need to justify spend and also deliver a cost saving.

Surgical site infections (SSIs) remain a problem despite investment in promoting hygiene and treatment protocols. SSIs remain a drain on healthcare resources and contribute to patient morbidity and mortality by delayed wound healing (DiPiro et al, 1998; Plowman et al, 1999; Leaper et al, 2004). Biofilms appear to contribute to SSIs, with complex synergies between bacterial colonies within a three-dimensional matrix. The combined colonies have different properties and sensitivities to the lone planktonic bacterium and are difficult to identify and isolate. The biofilm matrix appears to confer particular resistance to antibiotic therapy, which is of concern as antibiotic resistance is rising. In addition, in a healthcare community which has identified risk and harm associated with antibiotics and who are now culpable, we need to be aware of the rationale behind treatment options offered to patients.

Topical antimicrobial agents have come to represent the first line of treatment in the management of bacterial burden, particularly in chronic wound care, as they provide a high antimicrobial concentration at the site of infection. The use of the antiseptic polyhexamethylene biguanide (also known as polyhexanide or PHMB) demonstrates a reduction in overall SSI rate, meticillin resistant *Staphylococcus aureus* (MRSA) SSI rate and cost-effective care with demonstrable cost savings.

This document describes the relationship between optimal wound healing and SSIs, as well as an evidence-based approach to eradication of biofilms. Evidence for implementation of PHMB into current wound care practice is discussed, exemplified by case reports highlighting best practice. The authors suggest that PHMB offers a new method of bacterial control which has been proven safe, efficient and cost-effective.

This document will enable clinicians to realise benefits to patients and to provide alternative and additional tools to manage bacterial burden within the wound care environment.

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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], 2008).

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.

SURGICAL SITE WOUNDS

Infection at the site of surgery has been recognised as an issue probably for as long as man has been incising tissues to alleviate the symptoms of disease and injury. Historically, surgical interventions were nearly always associated with infection, patient mortality being accepted as almost an inevitability. Developments in infection control, surgical techniques, antiseptics, antibiotics and our understanding of the body’s response to bacterial infection have all resulted in surgery being a safer undertaking. However, postoperative wound infection still remains an issue.

Scale of the problem

In 1992, the US Centers for Disease Control (CDC) revised its definition of ‘wound infection’, creating the definition ‘surgical site infection’ (SSI) (Horan et al, 1992) to prevent confusion between the infection of a surgical incision and that of a traumatic wound. Although most SSIs are superficial, they contribute greatly to the morbidity and mortality associated with surgery (DiPiro et al, 1998; Leaper et al, 2004). A review of the incidence and economic burden of SSIs in Europe estimated that the mean length of extended stay attributable to SSIs was 9.8 days, at an average cost per day of €325 (DiPiro et al, 1998).

As far back as 2002 in the UK, it was estimated that hospital-acquired infections (HAIs) cost the NHS nearly one billion pounds (Nosocomial Infection National Surveillance System [NINSS], 2002). SSIs are one of the most important causes of healthcare-associated infections (HCAIs). A prevalence survey undertaken in 2006 suggested that approximately 8% of patients in hospital in the UK have an HCAI. SSIs accounted for 14% of these infections and nearly 5% of patients who had undergone a surgical procedure were found to have developed an SSI (Smyth et al, 2008). However, prevalence studies tend to underestimate SSIs because many of these infections occur after the patient has been discharged from hospital (National Institute for Health and Clinical Excellence [NIICE], 2008). Additional costs attributable to SSIs of between £814 and £6626 have been reported, depending on the type of surgery and the severity of the infection (Plowman et al, 2001; Coello et al,
The main additional costs are related to re-operation, extra nursing care and interventions, and drug treatment costs. The indirect costs due to loss of productivity, patient dissatisfaction and litigation, and reduced quality of life, are unknown. However, it is clear that there are serious implications for increased mortality, morbidity and costs associated with treatment of these infections (Reilly, 2002). These factors have additional significance for healthcare providers since the advent of publically accessible league tables.

Pathogenesis of surgical site infections

The development of a SSI depends on contamination of the wound site during or after a surgical procedure, and specifically relates to the numbers and virulence of the bacteria present, balanced against the individual’s ability to mount an appropriate immune response. Most SSIs are caused by bacteria that are present on the patient’s skin or gut, which gain entry to the tissues during surgery (endogenous infection). Exogenous infection occurs when microorganisms from instruments or the theatre environment contaminate the site at operation, or when bacteria gain access to the wound after surgery before the skin has sealed. Rarely, bacteria from a distant source of infection (such as through contaminated blood products) can cause an SSI by attaching to a prosthesis left in an operation site. Practices to prevent SSIs are therefore aimed at minimising the number of bacteria introduced into the wound, for example by:

- Reducing normal skin flora through preoperative showering, use of skin disinfectants and evidence-based depilatory methods if hair removal is required
- Preventing the multiplication of bacteria in the wound with prophylactic antimicrobial therapy
- Enhancing the patient’s defence against infection by minimising tissue trauma and maintaining body temperature
- Preventing entry of bacteria into the wound postoperatively by using a wound dressing instigating an appropriate infection control wound dressing procedure.

The most commonly encountered organism in SSIs is *Staphylococcus aureus*, however, when the bowel has been opened a large variety of bacteria may be present including anaerobes. In prosthetic surgery, the presence of the foreign body reduces the number of pathogenic organisms required to cause a SSI (NICE, 2008). In this environment, normally non-pathogenic organisms such as *Staphylococcus epidermidis* may also cause a SSI. Operations
on sites that are normally sterile (‘clean’) thus have relatively low rates of SSI (generally less than 2%). Such sites include planned surgery to the skin and muscle. However, after operations in ‘contaminated’ or ‘dirty’ sites (including where the lumen of the bowel is breached or where bacteria are known to be present, e.g. abscess drainage), rates may exceed 10% (Health Protection Agency [HPA], 2006).

General risk factors

Despite the negative effects of SSIs, data about their incidence and risk factors are incomplete. However, there are both intrinsic and extrinsic risk factors that are recognised as increasing the likelihood of the patient developing a SSI. Intrinsic factors include: age, active skin condition, smoking status, high body mass index (BMI), and comorbidity. Extrinsic factors include: poor or inappropriate pre-, peri- and postoperative patient care practices, such as inadequate preoperative skin preparation technique, inadequate hand washing and asepsis, inappropriate postoperative dressing selection and wound care (NICE, 2008).

While many of the intrinsic factors are largely outside the control of the clinician, measures can be taken to reduce the extrinsic risk factors in healthcare settings or practices. Positive changes in practice have occurred in recent years. Improvements in general infection control measures such as hand washing have seen the incidence of wound infections fall within specific patient populations, and guidance on the management of individuals undergoing surgical procedures is available from NICE (2008). However, without comprehensive national audit of healthcare practices and SSI rates, the full impact of these changes is difficult to quantify. Dressing choice and use is one area where improvements can still be realised.

The approach to dressing selection for surgical wounds depends to some extent on the type of surgery being undertaken, the closure technique employed, the objectives of wound management and the individual patient needs, including pre-existing comorbidities.

The majority of surgical wounds heal by primary intention, however, in some instances, delayed primary intention (sometimes referred to as ‘tertiary intention’), or secondary intention healing is the preferred route. In wounds managed by primary intention, the wound edges are apposed and then held in place by mechanical means (adhesive strips, tissue glue, staples or sutures), allowing the wound time to heal and develop enough strength to withstand stress without support. In delayed primary intention wounds, non-viable tissue is removed but the wound is not closed. Instead, it is dressed and the
wound edges are subsequently brought together after 4–6 days, generally before granulation tissue is visible (Gottrup, 1999). This method is often used where the wound is contaminated with bacteria or faecal matter, or when the complete removal of necrotic material cannot be assured. Another benefit of delayed closure is the cosmetic result after healing; the appearance of a wound closed after a delay of four to five days is comparable to that of a wound that underwent primary closure. A wider scar follows late closure (after 10–14 days), although this is cosmetically much better than the result obtained after the healing of an open, granulating wound (Gottrup et al, 2005).

Healing by secondary intention occurs when the wound is intentionally left to heal by a process of granulation, contraction and epithelial migration from the wound margins (Thomas, 1990; Foster and Moore, 1997). These wounds include abscesses and pilonidal sinuses, where the wound is considered ‘dirty’ or ‘contaminated’ either with pus or foreign bodies (Dowsett, 2002).

Objectives in surgical wound management

The objectives for wound management and dressing choice in surgical wounds can be summarised as:

- Protection from mechanical injury
- Protection from bacterial contamination
- Absorption of wound exudate
- Maintenance of the ideal wound healing environment.

Primary closure wounds

Generally, wounds which are closed at the time of procedure are protected from mechanical trauma and bacterial ingress during the first few days and heal on their own. Wound exudate tends to be limited to the first 24 hours following surgery, during which time some minor leakage of blood or serous exudate can be expected. In the majority of cases, initial postoperative dressings only need to absorb small amounts of fluid. After 48 hours, most wounds will be sealed with a plug of fibrin and congealed exudate and the dressing merely provides protection from mechanical trauma. As the wound edges are approximated, the wound bed is completely covered (or closed); maintenance of an ideal healing environment is therefore achieved by the body itself.

Protection of the wound from mechanical trauma and bacterial ingress is the main priority in the majority of situations. Appropriate dressings should
act as a barrier to inoculation of the wound from environmental bacteria. In many cases, simple adhesive dressings are sufficient, however, once wet, fabric-based dressings can easily form a direct route for bacterial spread (Aindow and Butcher, 2005). Dressing materials which adhere to the wound bed can increase the risk of mechanical damage to the wound when they are removed, leaving an entry site for bacterial contamination (Chrintz et al, 1989). Non-adherent dressings should therefore be used on surgical wounds (Phillips, 2001). Products with high adhesive ‘tack’ can cause disruption to the periwound skin on removal. This can lead to epidermal stripping, a leading surgical iatrogenic injury (White, 2005; 2008). This is easily managed in most cases, but is uncomfortable for the patient and can lead to increased risk of secondary infection.

For some individuals wound closure is not as straightforward. In patients with a compromised immune system, or who are known to carry high levels of potentially pathogenic organisms (either on the skin or on pre-existing wounds), the risk of wound infection is increased. In such cases, the use of a prophylactic antimicrobial dressing may be indicated. Similarly, if postoperative SSI occurs, there is a need to manage bacterial load within the wound. Serious wound infection (characterised by spreading cellulitis, erythema, oedema and pain, Cutting and Harding, 1994) is managed with systemic antibiotic therapy (EWMA, 2006). However, local wound infection may be managed by the use of topical antimicrobial products (EWMA, 2006; Best Practice Statement, 2010). Additionally, spreading infection may benefit from the use of topical antimicrobial adjuncts to reduce bacterial load (EWMA, 2006; Best Practice Statement, 2010).

**Secondary closure wounds**

In wounds healing by secondary intention, wound bed exposure is inevitable. Dressings must therefore manage the wound environment, controlling excess exudate by absorption and/or transpiration through the dressing, or by donating moisture to the wound bed to prevent desiccation. The time to affect closure is wound and patient dependent, and takes a number of weeks even in healthy individuals exhibiting normal wound healing physiology. Multiple dressing changes are needed before closure is achieved.

**Delayed primary closure (tertiary intention) wounds**

In tertiary intention healing the initial objective of wound management is the preparation of the wound for subsequent surgical closure. To facilitate this,
bacterial count needs to be kept to a minimum until closure can be achieved. The bacterial barrier function of the dressing chosen takes priority, an issue which is even more important when wound contamination is suspected. Dressings used to manage these wounds should be able to neutralise bacterial colonisation by preventing bacterial ingress and exerting an antimicrobial activity to the wound bed. As in secondary intention wounds, management of moisture balance should also be considered.

Management of open wounds

Ostensively open surgical wounds appear similar to chronic wounds. They heal by a process of granulation and, unless subsequently surgically closed, reepithelialise by gradual migration of cells from the wound margins over many weeks or months. However, there are two specific areas in which they differ; the bacterial bioburden within the wound and the physiological response to the healing process. Unlike chronic wounds, the acute open surgical wound has at the time of its inception a low bacterial bioburden. This condition may be transient, but does provide the clinician with a window of opportunity in which to ensure that the detrimental effects of high bacterial load can be avoided. There are subtle differences between acute and chronic wound healing, which a time-based description of the healing event does not adequately convey. Extensive wounds may take weeks or months to heal, however, physiologically they still heal using an acute healing model. 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). These changes may occur soon after injury, or may become established some time later.

It is important for the clinician to attempt to prevent the wound developing a chronic status if progression to healing is to be accomplished. This can be achieved by adopting the Wound Bed Preparation (WBP) approach to wound management. This approach has traditionally been utilised to manage chronic wounds, however, the tenets apply equally to the acute wound scenario. 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 in order 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). It is an approach...
that should be considered for all wounds that are not progressing to normal wound healing, or which have a risk of developing a chronic status. The mnemonic TIME is frequently used as a summary of the main focus within WBP.

**T** — **Tissue**
Within the acute, open surgical wound the clinician is generally faced with healthy tissue. The aim of management is therefore to maintain wound health and provide an environment in which granulation and re-epithelialisation can occur, or where non-viable tissue can be identified and removed (in the case of tertiary intention healing and the infected wound). The presence of friable granulation tissue in the wound bed increases the risk of dressing adherence, wound bed trauma, and secondary bleeding. Non-adherent dressings thus become essential. Any non-viable tissue present should be debrided to prevent it acting as a focus for infection.

**I** — **Infection/inflammation**
Wounds healing by secondary intention initially have low levels of bacterial colonisation, however, they are ideal environments for bacterial growth and their bioburden can quickly increase. If bacterial colonisation reaches a crucial point (critical colonisation), and/or is made up of highly virulent organisms such as Streptococci, wound healing is delayed and wound breakdown may occur. Close observation of the wound is important. If the wound shows signs of local infection or critical colonisation, the use of antimicrobial products is indicated (EWMA, 2006; Best Practice Statement, 2010). Ideally, such products should incorporate additional dressing characteristics which can manage other wound-related issues such as exudate management or moisture donation, and periwound skin protection. Regular and possibly frequent exposure of the wound bed to environmental bacteria increases the risk of infection, and so appropriate dressings to minimise the frequency of dressing changes should be
selected. In the immunocompromised individual, the risks of infection are also increased. In such cases a prophylactic antimicrobial may be indicated at an early stage of wound management.

**M — Moisture balance**
Moisture management is essential as open wounds lack the protection of epidermal cover. If exudate levels are low, moist dressings are indicated to prevent desiccation of the wound bed. In moderate to highly exudating wounds, dressings with a higher absorption capacity should be used to prevent maceration and leakage, both of which can compromise the periwound environment. Dressings should be able to balance the need to minimise wound bed disruption (with its increased risk of desiccation and bacterial ingress), and also to prevent the build-up of moisture (which increases the risk of maceration and infection). This may require the dressing to handle larger volumes of fluid and incorporate fluid-handling technology. Granulation tissue needs to be protected by the use of non-adherent wound contact materials. The health of the periwound skin should also be considered, as regular dressing changes with high-tack adhesive dressings can cause trauma. Dressings which are designed to elicit minimal skin damage should be chosen, together with good skin care regimens and barrier creams if required.

**E — Epithelial (edge) advancement**
In the acute wound, rapid re-epithelialisation should be expected if wound trauma at dressing change is avoided and bacterial colonisation is controlled. Failure to elicit epithelial migration within an expected time period may indicate the presence of a previously unrecognised delaying factor and will require a reappraisal of the wound management regimen. At wound level, the clinician should question if critical colonisation is having a detrimental effect on wound healing and intervention, and consider if the use of antimicrobial interventions is required.

**Special considerations**

**Infected surgical wounds**
Clinicians need to adopt an aggressive approach to the bacterial management of infected wounds. Local wound infection may be managed with topical antimicrobial dressing materials (EWMA, 2006; WUWHS, 2008; Best Practice Statement, 2010), with or without the use of systemic antibiotics. Dressing materials which facilitate debridement of non-viable tissue, management of exudate, and protection of periwound skin should also be selected. Spreading or
systemic infection requires rapid intervention and the initiation of appropriate systemic antibiotics in addition to local wound care measures.

**Wound care decision-making with percutaneous devices**

One specific area within the realm of surgical treatment is the use of percutaneous devices such as orthopaedic pins, external fixators, and other devices. These breech the integrity of the skin and remain anchored to underlying structures often for weeks or months. They pose particular challenges as they provide a route to deeper tissues through which pathogens can travel. Consequently, the way they are cared for may affect infection incidence. Infection prevention and management is important to prevent pin loosening, deep infection requiring surgical treatment (and increased morbidity), revisional surgery, and hospital stay (Egol et al, 2006; Wu et al, 2008). Additionally, pin tract infection reduces pin stability, which adversely affects fixation leading to loss of alignment of the fracture or fusion site. It can also cause osteomyelitis and systemic infection, which may be both costly and difficult to treat, as well as limb and life-threatening (Campbell et al, 2000; Temple and Santy, 2004).

Despite advances in the design and use of external fixators, pin tract infections are still common (Steckelberg and Osmon, 2000). Bacterial cultures have demonstrated that most pins are colonised, predominantly with aerobic Gram-positive cocci but also yielding Gram-negative and Candida species (Mahan et al, 1991; Dahl and Toksvig-Larsen, 2004).

Despite the frequency of infections in pin sites, a recent Cochrane Collaboration review (Lethaby et al, 2008) concluded that there is a complete absence of any particular strategy for pin site care. However, it seems appropriate that interventions should be initiated to remove debris which can act as a focus for bacterial colonisation, and reduce the risk of bacterial ingress through good wound hygiene, asepsis and prophylactic antimicrobial intervention.

**CASE REPORTS**

Management and treatment of a patient with a non-healing surgical wound

_David Gray, Clinical Nurse Specialist, Department of Tissue Viability, NHS Grampian_

A 64-year-old man with a history of bowel cancer presented with a non-healing surgical wound. He had undergone extensive surgery for his cancer, which led to a laparotomy wound. He also presented with a large abdominal
hernia, which could not be operated upon due to his underlying condition. He was advised that because of the extent of surgery undertaken, he would not be a candidate for further surgical intervention but would require ongoing chemotherapy.

The patient initially presented in April 2008 with a wound measuring 4.5x2.5cm that undermined by 1.5cm around the circumference.

On examination, surgical mesh was evident on the wound bed from previous surgery, with buds of granulation tissue forming over the mesh. The wound was colonised and there was medium volume low viscosity exudate.

The wound was initially treated with topical negative pressure therapy (TNPT), both in the hospital and by the district nurses in the community. Over a period of 10 weeks the wound improved significantly, reducing in size to 5.5x5cm and 0.3cm in depth.

No undermining was evident, there was 100% granulation tissue present on the wound bed, with no infection and a moderate volume of low viscosity exudate. The patient was then treated with a number of interventions including PolyMem Foam (Aspen Medical), Versiva® XC® gelling foam dressing (Convatec) and Promogran Prisma™ (Systagenix), with the wound almost healing. By December 2008, the dimensions of the wound were 0.2x0.2cm. During this period the patient underwent a number of treatments of chemotherapy, with multiple blood transfusions. At this point he was discharged from the department of tissue viability.

The patient returned in April 2009 with a non-healing wound (Figure 1). He had been treated at home with Versiva XC gelling foam dressing. The wound now measured 2.6x2.6cm with no depth and was pale and fibrinous. There was no evidence of infection or exudate.

The dressing was changed to Suprasorb® X+PHMB (Activa Healthcare, an L&R Company), as its moisture-donating ability provided a moist wound healing environment coupled with antimicrobial properties to reduce bioburden. This was secured with Versiva XC. The patient felt comfortable with this secondary dressing, as no surrounding skin issues had arisen while using it previously. Dressing changes were recommended every 3–4 days. The patient was also
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having fortnightly chemotherapy treatments.

During this time the patient went on holiday with his wife. He returned for review three weeks later and for more chemotherapy, reporting that dressing changes had taken place every 3–4 days. The wound measured 2.5x1.8cm with no depth. The wound presented with 50% granulation tissue and 50% delayed fibrous tissue (Figure 2). No infection was evident and there was minimal exudate. He continued with the regimen and was reviewed two weeks later while admitted for chemotherapy. His wound measured 1.4x2cm with no depth, no infection and no exudate. The wound bed consisted of 100% granulation tissue (Figure 3).

**Conclusion**
The wound had been non-healing due to the patient’s underlying cancer, ongoing chemotherapy and the presence of a large abdominal hernia. However, the wound improved against all the odds with the wound bed covered in granulation tissue. The ongoing treatment of Suprasorb X+PHMB with a secondary dressing of Versiva XC resulted in the wound fully healing.

**Use of a HydroBalance dressing with PHMB on an infected amputation wound**

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This case reports on a 72-year-old patient who was hospitalised in July 2008 for an infected stump wound. He had had insulin controlled type 2 diabetes and macrovascular disease for six years. He had a forefoot amputation in February 2008.
Materials and methods
On the stump there was a deep cavity (Figure 4) not reaching to the bone (Figure 5), with some exudate production. Previously, various dressings had been unsuccessfully used, such as alginates and silver dressings. The patient had received systemic antibiotics before being admitted to hospital. His blood tests showed normal markers for inflammation and moderate low albumin levels.

A puncture-biopsy taken from the stump showed positive for MRSA. The magnetic resonance imaging (MRI) scan taken of the foot did not indicate any infection of the underlying bone. On 26 July 2008, treatment with a HydroBalance dressing containing polyhexamethylene biguanide (Suprasorb X+PHMB) was started (Figure 6). PHMB is an antimicrobial shown to be effective against MRSA.

The HydroBalance dressing, Suprasorb X, is composed of biocellulose and water. The dressing is able to absorb exudate, donate moisture and maintain a moist environment, and is ideally conducive to support wound healing.

Suprasorb X+PHMB was used as a wound filler in the cavity (Figure 7) and covered with a surgical pad, fixed with a retention bandage. Dressing changes took place every second day.

Results
After 14 days of treatment, marked improvement was noted (Figure 8). The cavity was now filled with granulation tissue. The use of Suprasorb X+PHMB
was continued, now being employed as a primary cover for the superficial wound.

After 28 days of treatment the wound had closed. The blood markers for inflammation remained within the normal range.

**Conclusion**

Treatment outcome was unsuccessful for this patient’s stump wound with systemic antibiotics and various dressings which were applied over a period of five months. However, after one month of treatment with Suprasorb X+PHMB, the infected stump wound was closed.

**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 xylinium*. 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 dressings 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).

**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 intraluminal applications
- For applications involving the middle or inner ear, or for 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 SSI population.

**Health economics and cost-effectiveness**

The targeted use of antimicrobial dressings has repeatedly been reported to reduce surgical site infection (SSI) rates, thereby yielding substantial cost-savings. Gilliver (2009) identifies four US-based studies which consider the health economics consideration of the introduction of PHMB-based products. They demonstrate that following introduction of PHMB:

- Overall SSI rate was reduced by 24%
- MRSA SSI rate was reduced by 47%
- This delivered a $508,605 net saving during the one-year evaluation period (Mueller and Krebsbach, 2008)
- Vascular surgery had a progressive year-on-year fall in SSIs from 4.6% in 2000 to 0.4% in 2005, with an overall estimated saving of $876,176 (Penn et al, 2006)
- Hospital-wide introduction resulted in a reduction in the incidence of infections from 23 to 11 (both reported in separate six-month observation periods)
- Calculated net savings were $171,537 (Beneke and Doner, 2005)
- Treatment of recalcitrant wounds was more cost-effective; calculations
(based on material costs) averaged $5.99–9.01 per patient per day and were as low as $2.14 per day in one patient (Mulder et al, 2007).

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 >1 (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**

Surgical wounds present clinicians with an acute injury which, in the absence of underlying disease, should heal without incident. However, for many patients the development of infection can delay closure, lead to chronicity and herald systemic sepsis. Bacterial management is therefore of great importance,
particularly for those patients with a compromised immune response. While the implementation of infection control measures before and during surgical procedures can reduce the risk of infection, a key aspect of a proactive approach to SSIs is the management of bacterial load following surgery.

Topical antimicrobial preparations provide clinicians with a method of reducing bacterial load, and thus lessen the burden on the individual’s immune system which greatly increases the opportunity for positive wound healing outcomes.

PHMB is an antimicrobial compound which is new to wound care in the UK. It has a proven record of clinical efficacy, cost-effectiveness, and most importantly patient safety. Judicial use of this antimicrobial product can enhance care provision, particularly in those at risk of SSIs. By carefully combining this compound with a delivery vehicle that is easy to use and is structured to manage moisture balance, such as Suprasorb X+PHMB, a new approach to prevention and treatment of SSIs has emerged.

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