

GUIDELINES DOCUMENT CONTROL DOCUMENT- 1

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All policies are copy controlled by date. When a revision is issued previous versions will be withdrawn. Uncontrolled copies are available but will not be updated on issue of a revision. An electronic copy will be posted on the Trust Intranet for information.

Executive Summary

This guidance should be used within Oxford Health Community Services, Oxfordshire, Oxfordshire Clinical Commissioning Group & Oxfordshire Care Homes

The effective diagnosis of wound bed colonization and the timely implementation of appropriate treatment is a challenging issue within clinical practice. Early recognition along with prompt, appropriate and effective intervention is considered paramount in optimizing patient outcomes whilst maximizing resource management within primary care.

These guidelines provide clear, evidence-based recommendations on the timely identification and management of patient-related risk factors towards reducing bacterial colonization within a wound bed. The content of this document has been carefully considered and formulated to provide safe guidance on holistic clinical assessment to identify the stages of wound colonization, optimize host resistance and select appropriate topical dressing or systemic therapies

This educational resource will be of interest to a range of disciplines within Oxfordshire primary care including: General Practitioners (GPs), Nursing Staff (District Nurses, Practice Nurses and Specialist Nurses), Podiatrists, Care Homes and Pharmacists.

The key messages within this document are:

- The diagnosis of wound bed colonization or local infection is a clinical diagnosis. The majority of wounds are contaminated with microorganisms such as MRSA, *Pseudomonas aeruginosa* and *Anaerobes* and yet most will progress through the normal phases of healing.
- Wound swabbing should NOT be undertaken unless systemic antimicrobials (antibiotics) are indicated. The diagnosis of wound bed colonization/wound bed infection is a decision based upon a thorough clinical assessment in which distinct symptoms have been identified. The use of wound swabbing is not advocated as a first line diagnostic tool and therefore should be avoided as routine practice.

- Wound colonization or local wound bed infection should be treated with a topical antimicrobial dressing in the first instance. The use of systemic antimicrobials (antibiotics) should be reserved for treating systemic or localised soft tissue infection. For those patients who are immunocompromised, systemic antimicrobials (antibiotics) may be considered in line with local prescribing guidelines.
- Topical antimicrobial dressings are indicated for bacterial colonization or local wound bed infection management. The maximum period for application is two weeks. Antimicrobial therapy can be stopped before this time if the colonized state is abated. Within primary care Honey-impregnated dressings are advocated as first line treatment with Iodine based dressings as second line.
- An evidence-based wound dressing formulary is available to guide topical dressing therapies and is available [here](#). It is important clinicians are familiar with the products recommendations for use and would be encouraged to seek support from tissue viability or the representatives from the dressing manufacturer if unsure regarding use.

Please contact the Tissue Viability Team if you require further support regarding the implementation of these guidelines into clinical practice.

Telephone: 01235 205786/01235 208755 Fax: 01235 206788

Email: tissueviability@oxfordhealth.nhs.uk

The service is available Monday to Friday (except bank holidays) from 8:30am to 4:30pm

Telephone calls and answerphone messages received after 4.00pm will be followed up the next working day.

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Introduction

The management of local wound infection continues to be a challenging problem and represents a considerable healthcare burden (WUWHS, 2008). The early recognition along with prompt, appropriate and effective intervention is considered crucial towards reducing the economic burden of inappropriate management strategies whilst improving patient outcomes (European Centre for Disease Prevention and Control (ECDC), 2009 and European Medicines Agency (EMA), 2009). This applied in the context of growing resistance to antimicrobial agents such as systemic oral antibiotics highlights this as nationally recognised focus in healthcare delivery.

The diagnosis of local wound bed infection is considered to be a clinical decision based upon clear symptom criteria in line with a holistic patient assessment. For this reason the routine procedure of wound swabbing is not deemed an effective tool towards diagnosis and therefore should be avoided. The majority of chronic wounds are colonised by microbial species and yet progressive wound healing can occur often in their presence (Angel et al, 2011).

In recent years the microbiology teams within the Oxford University Hospitals trust have been reinforcing this message through feedback on microbiological results and education of practitioners. As a result the trend over the last five years has been a gradual decline from 27,843 swabs submitted in 2005-6 to 20,746 in 2010-11. There is consensus within wound management research and Oxfordshire Health Trusts that formal guidelines should support practitioners in the constructive deployment of clinical diagnostic tools in order to continue to provide a cost-effective yet effective service.

Topical antimicrobial dressings and systemic antimicrobials (antibiotics) can often be the key to the management of local wound bed infection and systemic soft tissue infection. There is growing national and international concern regarding the increasing resistance among gram-positive and gram-negative bacteria which can cause serious infections within humans. Therefore prolonged use of antimicrobials is not advocated due to the increased host risk of acquiring a healthcare associated infection (HCAI) and in some products the potential of toxicity to new cell growth.

This document represents principles presented in scientific research, international consensus documents as well as the consensus opinion of experts from a wide disciplinary consortium and healthcare trusts. This has ensured the guidelines are both practical but adaptable to local community practice. It is recommended that community practitioners use this guidance as part of a quality improvement strategy to optimise patient care and safety through enhancing appropriate systemic (antibiotics) and topical antimicrobial dressing usage (ECDC and EMA, 2009)

The content of this document has been carefully considered to support community-based healthcare professionals in daily clinical practice. It aims to provide:

- Guidance on the effective diagnosis of wound bed colonisation and local wound bed infection.
- Guidance on when and how to take a wound swab for microbiological screening
- Support in the appropriate selection of topical antimicrobial dressings and advice on when to use systemic antimicrobials (antibiotics)
- A strategy for reducing the risk of local wound bed infection through optimising host resistance and reducing bacteria colonisation

With thanks to the following individuals for their contribution and expert support:-

- Dr Ian Bowler, Consultant Microbiologist, Oxford University Hospitals
- Joanne Stanney, Interface Medicines Management Lead, Oxfordshire Clinical Commissioning Group.
- Helen Bosley, Infection Prevention and Control Matron, Oxford Health NHS Foundation Trust
- Sarah Thorpe, Senior Infection Control Nurse, Oxford Health NHS Foundation Trust
- Andrew Kingsley, Clinical Manager Infection Control and Tissue Viability, North Devon Healthcare Trust
- Gill Wicks, Tissue Viability Consultant Nurse, Wiltshire Community Health Services.
- Rose Cooper, Professor of Microbiology, Cardiff Metropolitan University

The diagnosis of wound bed infection

Infection is the inevitable outcome following a chain of dynamic interactions between a host, a potential pathogen and the surrounding environment (Vowden and Cooper, 2006). Local wound bed infection is normally diagnosed on clinical criteria (table 3) rather than bacteriological criteria via a wound swab.

Most open wounds are colonised by microbial species and yet the majority are not infected and the progression of wound healing can often occur in their presence (Angel et al, 2011). This is often the result of an effective immune response which enables the body to maintain the bacterial burden within the wound bed at a safe level.

Microorganisms commonly found in wounds include: - *Pseudomonas aeruginosa*, *Staphylococci*, *Streptococci* *Pseudomonads* and *Anaerobes* (Table 6) (Collier, 2004)

The presence of infection within a wound can damage tissue, delay healing and occasionally cause systemic illness. The potential for bacteria to cause harmful effects is influenced by:-

Host Resistance	The ability of the patient's immune system to fight the bacteria
The virulence of the bacteria	Some have the ability to produce disease-related symptoms in very small numbers.
Number of bacteria introduced	A high proportion of bacteria is likely to overcome host resistance more easily

(World Union of Wound Healing Societies (WUWHS), 2008).

Often in chronic wounds, the presence of bacteria may cause delayed healing despite lacking symptoms of active localised wound bed infection (Kingsley, 2001). The progressive stages of wound bed colonisation are described in table one and each will require individual considerations regarding treatment.

Wound Bed Contamination	The microbial loading of the skin and/or a wound bed with normal pathogens.
Wound Bed Colonisation	The presence of multiplying bacteria has the potential to tip the balance of host and wound defences. This requires close management to prevent the development of local wound bed infection. Effective wound bed preparation and management is required in order to suppress the bacterial bioburden. See section: the management of wound bed colonisation.
Local Wound Bed Infection	The bioburden of bacteria has disrupted the normal stages of wound healing and tissue at the wound bed and surrounding soft tissue are damaged. There is an acute onset of increased pain, levels of exudate and odour. A topical antimicrobial dressing should be first line treatment and the patient should be monitored for signs of systemic illness see section: selecting an appropriate antimicrobial treatment. The process of routine wound swabbing is not advocated as a first line approach to treating local wound infection
Systemic/Soft Tissue Infection	The presence of local wound bed infection and systemic illness and/or soft tissue infection. A topical antimicrobial dressing plus a systemic antimicrobial (antibiotic) should be considered in line with guidelines – see section: selecting an appropriate antimicrobial treatment.

Table One: The progressive stages of wound bed colonisation

(Adapted from The Sign Checker Tool with kind permission from Andrew Kinglsey) cited in (Young 2010)

Identifying Risks of Infection

Practitioners should be vigilant of those patients with underlying disease such as those with diabetes, on long-term steroidal therapy or patients receiving chemotherapy/radiotherapy as this may mask a normal inflammatory response.

Comorbidities	Diabetes Mellitus, immunocompromised status, hypoxia/poor tissue perfusion due to anaemia or arterial/cardiac/respiratory disease, renal impairment, malignancy, rheumatoid arthritis, obesity, malnutrition, smoking
Medication	Corticosteroids, cytotoxic agents, immunosuppressant
Psychosocial	Poor personal hygiene, unhealthy lifestyle choices, non-concordance, alcohol, drug abuse, sleep deprivation, stress

Table 2: Patient-related risk factors associated with localised wound bed infection

(WUWHS, 2008).

Clinical Assessment for Localised Wound Bed Infection

Complete the community wound assessment form as part of a nursing assessment of the wound. Refer to trust Wound Assessment and Management Guidelines for further information.

A holistic assessment of the patient should identify potential underlying causes for wound complications and potentially delayed wound healing e.g. sources of contamination, underlying disease processes, location of wound, nutritional status, medication regime, factors relating to delayed wound healing. Refer to Local Wound Assessment and Management guidelines for further information.

Consider whether the wound is within the normal inflammatory phase of healing. This process should subside within 3 days of initial trauma and does not require an anti-microbial dressing (table 3).

In chronic wounds the inflammatory process can become prolonged and exaggerated. Inflammation is one of the symptoms of a protracted process in which the overproduction of normally 'positive-to-healing' enzymes and proteases such as Matrix Metalloproteinases (MMP's) (see glossary in Appendix Two for definition of terms) combined with increased bacterial load can hinder tissue repair, new cell growth and angiogenesis (Vowden et al, 2008).

There is a close relationship between symptoms for soft tissue infection such as cellulitis, local wound bed infection, ischemia and inflammation. It is important that the correct diagnosis is achieved through a comprehensive nursing assessment as treatment options can vary considerably.

Identify and document the tissue type within the wound bed using the community wound assessment form. The presence of devitalised tissue, i.e. slough or necrosis can increase the risk or mask localised wound bed infection and inhibit the uptake of topical antimicrobial dressing therapy (O'Brien, 2002).

The decision to debride must be based upon a thorough clinical assessment and the formulation of a holistic treatment plan. Treatment aims should be in the best interests of the patient (Gray et al, 2011) and underlying risk factors (table 2) should remain a primary consideration towards constructive and safe wound bed preparation.

Clinical Indicators of Localised Wound Bed Infection

Many wounds may present with all or some of localised signs and symptoms.

Common signs of localised inflammation			
Erythema	Heat	Swelling	pain/tenderness
Diagnostic Criteria for local wound bed infection			
Discolouration of wound bed, ie. Dusky/raspberry jam colouration	Friable/Bleeding granulation tissue	Malodour	Pocketing/bridging of wound bed
Abscess	Cellulitis	Delayed healing	Peri-wound Oedema
Unusual or increase in pain/tenderness	Rapid deterioration of wound bed	Green/white/brown exudates	Increased or altered/purulent exudate

Table 3: comparison of inflammatory and localised wound bed infection symptoms

(WUWHS, 2008)

Erythema is not detectable in darkly pigmented skin and therefore additional indications of wound infection may be peri-wound induration ('woodiness') and possible changes in colour i.e. bluish purple.

The extent and severity of a wound infection will impact on management. It is important to recognise and differentiate the signs and symptoms of localised, spreading soft tissue infection and systemic infection (World Union of Wound Healing Societies (WUWHS), 2008). (see Table 4).

Documented Local Wound Infection	Pyrexia or Hypothermia
Tachycardia	Tachypnoea
Raised or Depressed white blood cell count	
NB: Other sites of infection should be excluded before assuming that systemic infection is related to wound infection	

Table 4: Diagnostic Criteria for Systemic Infection

(WUWHS, 2008)

Clinicians need to act promptly and seek an urgent medical assessment if a patient with a local wound bed infection shows signs of a systemic and/or potentially fatal infection, eg signs of sepsis or extensive tissue necrosis (necrotising fasciitis or gas gangrene) (WUWHS, 2008)

Soft Tissue Infection

Cellulitis

Cellulitis is a spreading infection of the skin and subcutaneous tissues. It is usually caused as a result of a break in the skin's defences which allows the entry of common pathogens (Beasley 2011). Presentation of symptoms may be a sudden onset of red tender swelling of the lower leg, warm and tender to touch, with a rapidly progressive demarcated margin, pyrexia. The affected area may present with blistering and possible leakage of serous exudates from the swollen limb, causing possible ulceration and skin maceration (Clinical Research Efficiency Support Team (CREST), 2005). Please refer to Oxfordshire Adult Antimicrobial Prescribing Guidelines for the use of Antimicrobial Agents in Primary Care (2012) regarding selecting appropriate systemic antibiotic treatment

Be aware that the occurrence of bilateral cellulitis is extremely rare. In the absence of typical clinical features consider deep vein thrombosis, varicose eczema or acute Lipodermatosclerosis as an alternative diagnosis (CREST, 2005). Also consider whether the symptoms are conducive to a protracted inflammatory process often seen in delayed wound healing. Please contact the Tissue Viability Team for further advice on 01235 205786 if required

Generally good skin care reduces the likelihood of cellulitis and consequently the need for antibiotics. However this condition can be resolved with a short course of oral systemic antibiotic therapy. Seek a medical assessment immediately and refer to Oxfordshire Adult Guidelines Antimicrobial Prescribing Guidelines for Primary Care 2012 regarding the selection of appropriate systemic antibiotic treatment. Monitor the patient closely for signs of sepsis and general deterioration.

Patients with Lymphoedema are at increased risk of acute cellulitis due to swelling producing deep skin folds in which fungal and bacterial infections can develop. The antibiotic regimen used to treat patients with this condition will vary depending on the clinical situation but should be managed in line with local prescribing guidelines (Lymphoedema Framework, 2006)

Recurrent cellulitis is common in those patients with Lymphoedema. Therefore prophylactic antibiotic therapy is recommended for those individuals who have experienced two acute episodes of Cellulitis within a one year period (Lymphoedema Framework, 2006). Please refer to local prescribing guidelines for further information.

Consider whether the wound is within the normal inflammatory phase of healing. This process should subside within 3 days of initial trauma and does not require an anti-microbial dressing (table 3).

Necrotising Fasciitis

ALERT: This condition is life threatening and patient deterioration is rapid therefore if suspected should be treated as a medical emergency.

Necrotising Fasciitis (NF) is a rare but has a high mortality rate of approximately 50% (CREST, 2005). It is a rapidly progressive and destructive soft tissue infection commonly known as flesh-eating disease and affects the deeper layers of the skin and subcutaneous tissues (Health Protection Agency (HPA), 2010). The key organism involved in the development of NF is *A Streptococcus* although this can often occur due to range of microorganisms (CREST, 2005).

This condition can affect healthy individuals, however those patients with medical conditions that weaken the immune system, create skin lesions or require invasive treatment procedures for example: cancer, diabetes mellitus, kidney dialysis, alcohol abuse and injecting drug users are at increased risk. The condition can develop through direct skin contact with individuals carrying the organism (HPA, 2010). Therefore standard universal precautions should be observed at all times whilst in contact with the patient and their immediate care environment.

It is characterised by disproportionate pain, crepitus, blue discolouration or deep necrosis, systemic toxicity with hypotension and blistering, cutaneous numbness. The patient is often septic and without surgery to remove the affected tissue and urgent medical intervention NF will progress rapidly and can result in organ failure and death (CREST, 2005, HPA, 2010). If you suspect the presence of this infection seek medical advice immediately.

Human Faeces

Human faeces are predominately made up of water, remnants of digestion such as fibrous elements, proteins, proteases, dead and live bacteria. A question often raised in clinical practice relates to the ability of human faeces to cause infection should the wound bed become contaminated

The availability of scientific evidence specific to this particular question is lacking possibly due to the fact that wounds in this region are well supplied with blood bringing sufficient oxygen to be used as a substrate by immunological cells to kill bacteria.

The consensus opinion of microbiology specialists indicate that whilst faecal contamination is likely to contribute to the overall bacterial load within a wound bed, it is unlikely to be the sole cause of colonisation with delayed healing or local wound bed infection.

The Relevance of Exudate

It is important to differentiate between chronic exudate and acute wound exudate as the latter is good for wound healing (Table 6).

Exudate is the product of the normal wound healing process. The inflammatory process is often triggered as a result of trauma, surgery or through homeostatic imbalance (Adderley, 2010). If a wound is not healing as expected, exudate production may continue and be excessive due to a prolonged inflammatory process.

A prolonged inflammatory process can generate increased odorous wound exudate that contains high levels of bacteria and substances harmful to new cell growth (WUWHS, 2007). The presence of devitalised tissue is likely to increase odour and is therefore not always indicative of wound bed infection (WUWHS, 2007). The appearance and levels of exudates can be an indicator of abnormal localised inflammatory processes and in isolation, are not always a clinical indicator of localised wound bed infection (WUWHS, 2007).

Exudate Colour	Possible Cause
Clear, Amber	Serous Exudate, often considered normal
Cloudy, milky or creamy	May indicate the presence of fibrin strands (in inflammation) or infection (Purulent with white blood cells and bacteria)
Pink or Red	Presence of red blood cells indicating capillary damage
Green	May be indicative of bacterial infection e.g. Pseudomonas Aeruginosa
Grey or Blue	May be related to the use of silver-based dressings.

Table 5: Exudate Colour Classification

(WUWHS, 2007)

It is worth noting that some dressings such as hydrocolloids and hydrogel sheets may produce a characteristic odour due to their mode of action with wound exudates (WUWHS, 2007) and odour should be reassessed following appropriate wound cleansing.

Acute Wound Exudate	Chronic Wound Exudate
Rich in growth and immune factors which promote healing by stimulating cell proliferation and providing the nutrients for cell metabolism. Fluid supports cell proliferation. The levels of exudates tend to decrease in line with progressive wound healing.	Due to prolonged inflammatory processes often due to localised infection and/or underlying disease processes this fluid is more likely to contain dead white cells, high levels of inflammatory mediators and protein digesting enzymes which disrupt the normal healing process.

Table 6: Differences between acute and chronic wound exudates

(WUWHS, 2007)

Assess and document the colour, consistency, odour and levels of wound exudate as part of a formal nursing assessment (Table 6). An effective management plan should aim to contain exudates, maintain patient comfort and dignity whilst protecting vulnerable structures such as peri-wound skin and the wound bed from trauma, excoriation or maceration (Adderley, 2010).

Refer to the Community Wound Dressing Formulary for guidance on an appropriate absorbent dressing. Consider using the wound healing pathway as a tool towards effective diagnosis available via Tissue Viability.

Microbiological Investigations

Common Wound Pathogens

The products of certain microbial species are known to affect wound healing and are detailed in (Table 7). Often the presence of a community of microorganisms (a Biofilm) may inhibit wound healing by promoting a chronic inflammatory response (European Wound Management Association (EWMA), 2006).

Gram Positive Cocci	<i>Beta Haemolytic Streptococci (Streptococcus Pyogenes)</i> <i>Staphylococci (Staphylococcus aureus/MRSA)</i>
Gram-negative Aerobic Rods	<i>Pseudomonas aeruginosa</i>
Gram- positive Rods	<i>Anaerobes</i> (organisms surviving in the absence of oxygen)

Table 7: Common Wound Pathogens

Staphylococci (*Staphylococcus aureus*/MRSA)

Methicillin Resistant Staphylococcus (MRSA) is a strain of *Staphylococcus aureus* (SA). SA is the most common bacteria to affect humans and about a third of the population are colonised with the organism which can live harmlessly on the skin, in the nose, skin folds, hairline or navel (Royal College of Nursing (RCN), 2005).

Anaerobes

Anaerobic bacteria or *Anaerobes* are a common cause of wound infections and do not require oxygen for growth (Bowler et al, 2001). The clinical presentation of an anaerobic colonisation is often mistaken for necrotic tissue. There is often a pungent odour in the presence of anaerobes with a dark brown or sometimes black 'film' across the wound bed.

Pseudomonas aeruginosa

Pseudomonas is a gram-negative rod that belongs to the family *Pseudomonadaceae*. It has become an important cause of wound infection, especially in patients with compromised host defense mechanisms (Lessnau et al, accessed 12/12/2011). *Pseudomonas* flourishes in moist, aerobic (oxygenated) environments. These infections may have a characteristic blue-green exudate with a fruity produce and a characteristic sweet odour (Lessnau et al, accessed 12/12/2011).

Biofilms

The term biofilm describes a diverse community of microorganisms which cannot be seen by the naked eye but have shown to contribute to the impaired healing of chronic wounds (Percival and Bowler, 2004). A biofilm has been described as bacteria embedded in a protective slimy barrier of sugars and proteins attached firmly to a living or non-living surface (Phillips et al, 2010).

Biofilms have a high tolerance level to topical antimicrobial dressings and are often the cause of or have often fallen into the category of wound bed colonisation with presenting symptoms being typical of a prolonged inflammatory response (Phillips et al, 2010). If despite appropriate wound management, healing fails progress in a timely manner, consider the presence of a biofilm within the wound bed. For further guidance on biofilm management see section: The Management of Wound Bed Colonisation.

Wound Swabbing

When to take a wound swab

The process of routine wound swabbing is not advocated as a first line approach to diagnosing or treating local wound bed infection or colonisation

As part of the national and local MRSA screening guidelines, those patients admitted to a community hospital will require a wound swab for skin lesions. Please refer to the MRSA Procedure on the Infection Control Portal for further guidance.

Local wound bed infection is normally diagnosed on clinical criteria (Table 3) rather than bacteriological criteria, as most open wounds are colonised by microbial species and yet the progression of wound healing can often occur in their presence (Angel et al, 2011). Therefore the use of a topical antimicrobial dressing is not indicated in the absence of localised wound bed infection or colonisation (refer to section: Diagnosis of Local Wound Bed Infection).

The diagnosis of wound bed infection is a clinical judgement and the information on wound-based micro-organisms provided by laboratories may have little value if not considered without reference to the patient (Vowden and Cooper, 2006).

The criteria for sending a microbiological specimen should be with the intention to treat with systemic antibiotic therapy, a clinically diagnosed wound bed infection that has progressed to a soft tissue or systemic sepsis.

If despite the appropriate application of a topical antimicrobial dressing a practitioner can demonstrate (in line with recommendations put forward in these guidelines) this has failed to manage the localised infection effectively a wound swab may be indicated to further guide a topical antimicrobial dressing or systemic antimicrobial (antibiotic) therapies.

It is important to consider that it is the interaction between the host and the bacteria which will determine how the organisms will influence wound healing, not the presence of the bacteria alone (Angel, et al, 2011).

For those patients who fall into the following categories and have clinically diagnosed wound bed infection, a wound swab may be required to guide antibiotic therapy (WUWHS, 2008; EWMA, 2006) (Table 8).

Compromised Immune System Function	
Long term or several systemic antibiotics in the past	Diabetes Mellitus
Not responding to current systemic antibiotics following patient re-assessment	Limb threatening infection

Table 8: Health indicators for the use of systemic antibiotic therapy

If a patient presents with Cellulitis or locally leg/foot ulceration and diabetic foot infections systemic antibiotic therapy is indicated. Please refer to the Oxfordshire Adult Antimicrobial Prescribing Guidelines for Primary Care 2012 for appropriate therapy.

The prescription of a systemic antimicrobial (antibiotic) or topical antimicrobial dressings should be in line with local and national protocols. The potential risk of progressive soft tissue infection and sepsis in this patient group versus potential host resistance should be a primary consideration (advisory committee on Antimicrobial Resistance and Healthcare Associated Infection (ARHAI) (2011).

Undertaking an effective wound swab

The process of routine wound swabbing is not advocated as a first line approach to diagnosing/treating local wound bed infection.

The tissue viability team have developed a procedure for wound swabbing. A video and step by step guide to support practitioners in conducting a successful wound swab has been devised and is available at (TBC)

The Levine swabbing technique is advocated in line with research evidence (Angel et al, 2011 and Gardner et al, 2006) and specialist consensus. It differs from the current Zig Zag technique in that it involves applying a firm pressure whilst rotating the swab over a 1 cm² area of the wound bed.

There is emerging evidence that good quality tap water can contain pseudomonas. In routine cleansing of chronic wounds this is unlikely to be detrimental to the overall healing process and therefore continues to be supported evidence based practice. However expert microbiology consensus highlights using tap water prior to wound swabbing may have the potential to skew the accuracy of swab results.

Due to the destructive nature of wound bed infection, the regeneration of new cell growth conducive to progressive wound healing is seriously compromised. For the purpose of wound swabbing the use of dry sterile gauze to remove obvious contaminating materials such as dried exudates and dressing residue is considered an acceptable alternative method. Please refer to the wound swabbing procedure (appendix three for further guidance on the safe execution of this method.

NB: This method of 'cleansing' is only advocated as part of the swabbing process and not as routine practice.

Put the swab back into the transport medium and send to the microbiology laboratories via your transport system. Ideally the wound swab should be sent the day of sampling however this may be stored overnight in the fridge if delayed.

Provide comprehensive information on the microbiology request form including:-

- Enter the request in the GP electronic requesting system (ICE) if possible, as this minimizes the chance of data entry errors and ensures the result is sent back electronically to the correct GP as soon as it is available.
- Patients full name (not initials), date of birth and NHS number.
- Ensure a clear contact and address for return of results.
- Include the name and practice address of registered GP.
- Please state any antimicrobial therapy the patient is about to start or has already been used.
- Please state the anatomical location from which the swab was taken.

Optimising Host Resistance

The effective management of local wound bed infection often requires a multidisciplinary approach and treatment aims should be to minimise the infecting microorganisms whilst optimising host resistance (WUWHS, 2008)

A holistic nursing assessment should identify systemic factors which may have contributed to the development of infection. The implementation of therapeutic measures that manage patient co-morbidities will aim to optimise patient ability to fight the infection whilst improving healing outcomes (WUWHS, 2008) (Table 2).

The immune system is highly reliant on good nutritional status. The cells within the immune system require nutrients such as amino acids, vitamins and lipids to function effectively (Royal College of Nursing (RCN), 2012). Malnutrition and dehydration can compromise immune function and therefore predispose the patient to local wound and skin as well as systemic infection (RCN, 2012).

The Malnutrition Universal Screening Tool (MUST) (British Association of Parenteral and Enteral Nutrition (BAPEN) is available for download at: http://www.bapen.org.uk/must_tool.html) and is a recommended community-based tool for performing an initial and on-going nutritional assessment as part of a holistic assessment. A clearly documented action plan should be implemented to address the nutritional needs. Consider a referral to the dietetics teams for timely support (RCN, 2012). Also refer to the local 'Guidelines for the Management of Undernutrition for Adults in Primary Care (2010). Available at www.oxfordshirepct.nhs.uk

Effective wound management should also encompass wound bed preparation to create an environment that optimises healing outcomes. This can be achieved through effective exudate management, debridement of devitalised tissue, appropriate dressing selection and in some cases early implementation of compression bandaging or hosiery. Refer to wound bed preparation within the management of wound bed colonisation section for guidance.

Local standard precautions should be followed to reduce the risk of exposure to potentially infective materials, to protect the healthcare worker and patient and to prevent the transmission of infection. Please refer to Standard Precautions and Personal Protective Equipment Procedure for comprehensive guidance on effective management in the community.

Selecting an appropriate antimicrobial treatment

Healthcare Associated Infections (HAIs) are infections that develop as a direct result of medical or surgical treatment or contact within a healthcare setting. They can occur in hospitals as well as health and social care settings in the community (Department of Health (DH), accessed 28/12/11).

Resistance to systemic antimicrobial (antibiotic) therapies is of growing national concern due to the recognised detrimental effects on long-term patient health and treatment outcomes. Common pathogens such as MRSA, *Clostridium difficile* and *Escherichia coli* are now

resistant to commonly prescribed systemic antimicrobial (antibiotic) therapies (Advisory Committee on Antimicrobial Resistance and Healthcare Associated Infection (ARHAI), 2011).

Topical (dressings) and Systemic Antimicrobial (antibiotic) agents act by inactivating many aspects of microorganisms cell function and therefore can reduce the numbers of microorganisms within a host.

Antimicrobial	Antimicrobials are substances which are used in the treatment of infection caused by bacteria, fungi or viruses (Department of Health, 2008)
Antibiotic	Antibiotics are substances that kill or interfere with the growth of microorganisms, especially bacteria (Healthcare Protection Agency, 2010)
Antiseptic	A disinfectant substance that can be used on skin and on wounds that either kills or prevents the multiplication of potential pathogenic organisms (Vowden and Cooper, 2006)

Table 9: Definition of antimicrobial terms

Where localised wound bed infection has been diagnosed, use an appropriate topical antimicrobial dressing. Prior to use, the modality, indications for use and contraindications of the antimicrobial dressing should be considered.

The use of topical antimicrobial dressings and systemic antimicrobial (antibiotics) if indicated should be re-assessed at timely intervals during a two week period. Systemic therapy should be for one week initially, if systemic response is slow, therapy should be continued for a further week.

The prolonged use of topical antimicrobial dressings and systemic antimicrobial (antibiotic) therapy is not advocated due to the increased host risk of acquiring a healthcare associated infections (HCAI) and in some products the potential of cytotoxicity. However some patients, such as those with conditions such as cellulitis and diabetic foot infections may require system antibiotics for longer periods. Please refer to the Oxfordshire Adult Antimicrobial Prescribing Guidelines for Primary Care (2012) for guidance on duration for oral therapies.

The maximum time a single topical antimicrobial dressing should be used is two weeks (Wounds UK, 2010). Practitioners should be reassessing the patient and wound state at regular, agreed intervals during this period. If during this time, nursing assessments identify the local wound bed infection or colonisation state has abated and the wound bed is showing signs of improvement the antimicrobial dressing should be stopped and dressing choice should be in line with wound and patient needs (EWMA, 2006).

If the localised wound bed infection is not resolving within seven to ten days (Vowden and Cooper, 2006), re-assess to rule out underlying causes including systemic infection and consider host resistance to the topical antimicrobial dressing in use. Consider an alternative dressing in the first instance.

If despite the effective use of an topical antimicrobial dressing, the patient presents with symptoms of systemic sepsis or soft tissue infection consider taking a wound swab in line with local protocols to isolate key pathogens and seek medical advice immediately (Tyrell et al, 2000) (see section on When to take a wound swab for further guidance)

The use of Systemic Antibiotic Therapy

NB: Where a reference source is depicted by a number, please refer to Reference List Two in Appendix One for the supporting evidence.

The term **antibiotic** resistance describes those microorganisms which are not killed or inhibited by an antibiotic but continue to grow and multiply in its presence (Health Protection Agency (HPA), 2010). This can result in failure to respond to treatment and prolonged illness (HPA, 2010).

The emergence of systemic antibiotic resistance has been to some extent inevitable due to the increasing demand for such therapies (ARHAI, 2011). It is widely advocated that the use of systemic oral antibiotics should be reserved for treating invasive infection, while topical antimicrobial dressings should be used to treat superficial local infection on an open wound bed (EWMA, 2006; Wounds UK, 2010; WUWHS, 2008).

Inappropriate use of broad-spectrum antimicrobials (antibiotics) is associated with the acquisition of *Methicillin Resistant Staphylococcus aureus* (MRSA)³⁻⁶ and the induction of *Clostridium difficile* Infection (CDI)⁷⁻¹² as well as the selection of antimicrobial (antibiotic) resistant bacteria such as Extended-Spectrum Beta-Lactamase (ESBL)-producing Gram-negative bacteria^{13,14}.

Whilst all antimicrobials (antibiotics) are able to pre-dispose patients to CDI and MRSA, **quinolones, cephalosporins, and clindamycin** are particularly associated with a high risk of causing CDI and so should be avoided unless there are clear clinical indications for their use^{15,16}. **Co-amoxiclav** has also been associated with CDI cases both nationally^{15,16} and locally. Therefore, the above antimicrobials (antibiotics) have been restricted where possible within Oxfordshire primary care and secondary care antimicrobial guidelines.

Appropriate antimicrobial (antibiotics) prescribing is a key element in the reduction of healthcare associated infections¹⁷. The evidence that use of antimicrobial (antibiotics) agents (whether appropriate or not) causes resistance is overwhelming¹⁸; resistance is greatest where use of antibacterial agents is heaviest¹⁸. Prescribing a routine course of antimicrobials (antibiotics) significantly increases the likelihood of an individual carrying a resistant bacterial strain¹⁹

The appropriate use of systemic antibiotic therapy can play an important role in treating progressive soft tissue infection such as Cellulitis and Diabetic foot infections or in the presentation of systemic sepsis or bacteraemia (Wounds UK, 2010, EWMA, 2006). Where there is evidence of infection and advancing cellulitis around the wound or fever (Table 4) or there is no/limited response to topical dressing treatment, systemic antimicrobial (antibiotic) therapy should be considered and a medical assessment should be sought immediately.

Generally a narrow-spectrum antibiotic will be commenced (Oxfordshire Adult Antimicrobial Prescribing Guidelines for Primary Care, 2012). For severe infections requiring IV antibiotic therapy a referral will be necessary. Refer to wound swabbing section for guidance on nursing interventions to support the appropriate prescription of antibiotic therapy.

Clinical Management: Implementing an Effective Plan

The wound management care plan should form part of holistic assessment documentation and clearly document the formal diagnosis at the time of assessment, a clear rationale for anti-microbial dressing selection, specific treatment objectives, underlying risk factors, set realistic re-assessment dates (WUWHS, 2008). The selection of an anti-microbial dressing should be in line with the community dressing formulary.

The presence of multiplying bacteria has the potential to tip the balance of host and wound defences. This requires close management to prevent the development of local wound bed infection (Kingsley, 2001). Effective wound bed preparation and management is required in order to suppress the bacterial bioburden.

Establishing and maintaining ways of working which keep the level of potential cross contamination between patients to an absolute minimum is a major priority in Infection Control^{20,21}. The most effective way to do this is to decontaminate hands and equipment between patients²²

The importance in Wound Bed Preparation

Bacterial colonies are often contained within devitalised tissue, i.e. slough or necrosis and not only present an increased risk or mask the presence of local wound bed infection but can also inhibit the uptake of antimicrobial therapy donated by topical wound dressings (O'Brien, 2002).

The decision to debride must be based upon a thorough clinical assessment and formulation of a holistic treatment plan. Treatment aims should be in the best interests of the patient (Gray et al, 2011) and underlying risk factors (table 2) should remain a primary consideration towards constructive wound bed preparation.

Autolytic Debridement describes the process of using the body's own enzymes and moistures to rehydrate and therefore soften devitalised tissue i.e. hard necrosis or slough (Gray et al, 2011). This ordinarily can be assisted using a hydrogel-based or hydrocolloid topical dressing (refer to the Wound Dressing Formulary for further guidance)

The routine use of topical antimicrobial dressings for wound bed preparation and tissue debridement is not advocated and relates back to previous sections regarding host resistance and cell cytotoxicity.

However where a patient has been identified as at high risk to local wound bed infection (Table 2) or bacterial colonisation has been clinically diagnosed, the **controlled** use of a honey impregnated topical dressing (Section 18) maybe a consideration for active debridement of devitalised tissue whilst suppressing bacterial bioburden and odour (Gray et al, 2011; Vowden and Vowden, 2011). **Please seek advice from Tissue Viability for further guidance on wound management prior to instigating this type of treatment.**

The Role of Biofilms

The term biofilm describes a diverse, microscopic community of microorganisms which have shown to contribute to the impaired healing of chronic wounds (Percival and Bowler, 2004). A biofilm has been described as bacteria embedded in a protective slimy barrier of sugars and proteins attached firmly to a living or non-living surface (Phillips et al, 2010).

Biofilms have a high tolerance level to topical antimicrobial dressings and are often the cause of or have often fallen into the category of colonisation with presenting symptoms being typical of a prolonged inflammatory response (Phillips et al, 2010).

The presence of a biofilm in a wound bed can stimulate a persistent inflammatory process which leads to the production of inflammatory mediators and proteolytic enzymes. This can have many affects however most poignant is the destruction of new cell matrixes and inhibition of re-epithelialisation (Gray et al, 2011).

In order to maximise the success of therapeutic measures the bacterial colonisation must be suppressed and the wound bed made viable through effective wound bed preparation (Mofatt, 2004).

Topical Antimicrobial Dressing Selection

Antimicrobial dressings act by inactivating many aspects of microorganisms cell function and therefore can reduce the numbers of microorganisms within a host. Where localised wound bed infection has been clinically diagnosed, use an appropriate topical antimicrobial dressing.

Optimising conditions to promote progressive wound healing should be the overriding objective to selecting an antimicrobial dressing. In order to successfully reduce microorganisms, dressing choice should be based upon the mode of action and efficacy of the agent, cytotoxicity to human cells and its potential to select resistant bacterial strains (Vowden and Cooper, 2006).

The careful assessment, appropriate care planning and documentation and clear reassessment dates are central to the success of antimicrobial dressings use (Moore and Romaneklli, 2008). The choice of dressing should be based upon a holistic patient and wound assessment and care plans should clearly state specific treatment objectives, frequency of dressing changes, set timeframes for antimicrobial dressing use and wound reassessment dates.

The desired frequency of dressing changes, the size of the wound and the proposed timeframe planned for use of the product will influence dressing choice (Table 6). It is important practitioners are familiar with the products recommendations for use and would be encouraged to seek support from tissue viability or the representatives from the dressing manufacturer (Moore and Romanelli, 2006) if unsure regarding use.

A [dressing selection](#) algorithm is available to assist practitioners in making informed clinical decisions regarding antimicrobial dressing choices.

Frequency of Dressing Changes	Wound Size and shape	Wound Anatomical Location
Pain	Patient Preference	Level of exudate (table 5)
Does the dressing?		Is the dressing?
Stay intact/In place throughout wear time		Comfortable, conformable, flexible
Kill bacteria or inhibit the action of bacteria		Suitable to be left for a long duration of time
Cause skin maceration/allergy/sensitivities		Easy to remove without causing pain or trauma
Reduce pain/odour		Easy to Use
Manage exudates		Cost-effective
Dump antimicrobial into the wound bed or absorb and retain microbes in the dressing		

Table 10: Considerations for Antimicrobial Dressing Choice

(WUWHS, 2007; Moore and Romanelli, 2006)

Introduction to Topical Antimicrobial Dressing Agents

This to be formalised once formulary decisions are made

Iodine

Iodine can be an effective broad spectrum antibacterial agent. There are two types of dressing types:

Cadexomder Iodine

The antibacterial agent is held in highly absorbent microbeads, the iodine is slowly released when it comes into contact with wound exudate.

- Often available in powder, paste or ointment modalities and are indicated in moderate to high exudating wounds, including cavity wounds.
- Maximum single application should be 50g – weekly application should not exceed 150g and treatment should not exceed two weeks in a single course of treatment (British National Formulary (BNF), 2011).

Povidone Iodine

Usually a single knitted viscous dressing impregnated with 10% povidone iodine used for superficial skin loss injuries.

- The iodine agent can be quickly de-activated by pus or high exudates levels. Generally the iodine is absorbed within twenty four hours and is indicated by dressing colour changes from brown to white.

Contraindications

Iodine can be absorbed systemically especially in larger wounds, therefore should be used with caution in those patients with thyroid disorders, significant renal disease, pregnancy/breast feeding and children (BNF, 2011).

Honey

Honey has been used for many centuries to accelerate wound healing. Honey promotes a highly concentrated sugar solution which reduces the amount of water available for bacterial growth.

This broad-spectrum topical antimicrobial has different therapeutic properties which include:

- Bacterial cells require water to survive. Through the osmotic effects of honey water is drawn from the bacteria cells and therefore damages their infrastructure.
- Honey produces Hydrogen Peroxide, the components of which decomposes bacteria and renders them ineffective.
- Honey supports moist wound healing and therefore can create an environment for autolytic debridement of devitalised tissue and reduce wound odour. It should be noted this should only be considered as part of an antimicrobial treatment plan for treating local wound bed infection or colonisation (BNF, 2011)

Honey is effective against multi-resistant organisms including MRSA and gram-negative *organisms* such as *Pseudomonas aeruginosa*. The product is indicated for use on locally infected cavity or superficial wounds requiring assistance with debridement of devitalised tissue

Generally Honey dressings consist of either honey impregnated tulle or alginate dressings or a mixture of substances to form a honey-gel consistency (Table 11 for guidance on modality selection). The choice of dressing should consider the depth and the location of the wound as well as the level of exudates. Refer to the Wound Dressing Formulary for further guidance on product availability.

Dressing Modality	Wound Type	Exudate Levels
Honey Gel or Ointment		
Presented in a tube of either pure honey or a composition of wax to increase adhesion. This product is generally recommended for use on low exuding wounds due to the risk of the honey being washed into secondary dressings by high exudates levels. However the gel can be useful in cavity or sinus wounds where alginate or fibrous dressings are difficult to place.	superficial or cavity	Low exudates
Honey Impregnated Tulle dressing		
Consists of a synthetic, fine weave non-adherent dressing impregnated with honey. This product has little absorbent capacity and therefore is better suited to low exuding and superficial wounds.	superficial	Low to moderate
Honey Impregnated Calcium alginate dressings		
An alginate impregnated with honey, approximately 10cm x 10cm contains 25mg-30mg of honey. In deep cavities, several layers of alginate may be required to increase absorbency and allows release of honey over an extended period of time.	Superficial or cavity	Moderate to high
Honey Gel Sheet		
The gel sheet consists of a mix of honey and sodium alginate. Honey is released into the wound bed as exudates are absorbed into the dressing. The dressing becomes a gel therefore does not leave residue or adhere the wound bed. The gel sheet is also pliable and conforms well to uneven wound surfaces. Recommended for light to moderately exuding superficial or cavity wounds	Superficial or Cavity Wounds	Light to moderate

Table 11: descriptions of honey dressing modalities

(Betts, 2010)

The use of honey in the Diabetic Patient

There is a general assumption in practice that honey should not be used due to the increased risks of hyperglycaemia and inconsistent blood sugar levels. In order for honey to influence blood sugar levels it would require absorption via the wound bed into the blood stream via the capillary network (Betts, 2010).

The British National Formulary (BNF) (2011) advises that diabetic patients should be monitored for changes in blood-glucose concentrations during a course of treatment involving the use of honey. The scientific evidence to support this as standard practice is limited (Betts, 2010; McIntosh and Frykberg, 2010). However in those patients with multiple wounds clinical judgement in line with a risk assessment should determine blood glucose monitoring practice (Betts, 2010).

The use of honey in patients' with an allergy to bee products

The BNF (2011) advises that honey dressings should not be used on patients with extreme sensitivity to bee stings or bee products. There is limited scientific evidence reporting known sensitivities and reactions to honey-related products (McIntosh and Krykberg, 2010). However practitioners should be cautious with patients who may have a known allergy to any component of the dressings used (Betts, 2010). Please seek advice from Tissue Viability if you are concerned.

Silver

When a silver-dressing is applied to a wound, silver ion is released. In order for silver to kill bacteria it must take on the form of charged particles - ions (Hampton, 2010). The silver ions are absorbed by bacteria which in turn bind to the DNA structures to affect cell function and respiration. The overall effect is bacterial replication is halted and colonies are reduced (Graham, 2005).

The range of silver dressings is large and their mode of antimicrobial action is diverse. This varies from those that actively deliver silver to the wound surface either in low or high concentrations (Nanocrystalline Technology) or others which retain and kill bacteria within the structures of the dressing.

The common types of silver used in wound dressings are:-

Nanocrystals– very small particles of silver ions usually contained within knitted fabric, non-contact layer, activated charcoal, foam or alginate dressings. **Nanocrystalline** dressings are generally able to sustain a higher level of silver activity for shorter periods of time due to the controlled coverage of the wound surface area (Maillard and Denyer, 2006; Leaper 2011)

Silver Sulphadiazine (SSD) – a combination of silver ions with a sulphonamide antimicrobial providing a moderated topical aqueous agent. SSD works slowly under the action of sodium chloride originating from wound exudates. This releases two antimicrobial agents at concentrations that are selective to a broad spectrum of bacteria on the wound surface (White and Cooper, 2003).

Absorption of Silver depends on the:

- Type of silver used within the dressing
- Depth and surface area of the wound
- Frequency of dressing application
- The amount of silver incorporated into the dressing
- Levels of wound exudate secreted from the wound

There is widespread debate regarding the safety of silver in relation to systemic absorption and toxicity to human cells. It is thought this occurs when silver ions are released into the wound bed too quickly over a sustained period of time. Most modern dressings are now designed to modulate the release of free silver ions available to penetrate the wound bed therefore hopefully reducing this eventuality (Leaper, 2003).

Argyria is the blue-black discoloration often seen at the wound bed following a period of silver application. It is one of the common contraindications of using silver compounds in wound dressings. There seems to be no evidence to indicate long-term side effects or skin staining (Landsdown, 2002). The discoloration is often mistaken for anaerobe infection, necrosis, or ischemic influences at the wound bed. Practitioners should consider the recent use of a silver-based dressing as part of the holistic nursing and wound assessment.

Topical dressing products vary in their structure, formulation and concentration of available silver. Due to their conformability to the wound surface (which allows for slow but sustained release of silver), topical dressings have been found to be more effective as an antimicrobial over silver sulfadiazine preparations (Maillard and Denyer (2006).

The activation of silver at the wound bed in Silver Sulfadiazine preparations is guided by the availability of sodium chloride in wound exudate. Conversely the presence of organic properties such as phosphates, sulphides and pus can also affect the efficacy of silver (Maillard and Denyer (2006). It is important to consider the preparation in line with wound management objectives. For example in a highly exuding wound, practitioners may consider a Nanocrystalline dressing which may address exudate levels as well as release silver into the wound effectively. Please seek advice from Tissue Viability if you are unsure as to which product to select.

Chlorhexidine Gluconate 0.05%

This product is produced in two forms: a 0.05% dilution for wound cleansing and a 4% solution for use as a surgical skin preparation and hand scrub (Main, 2008). The 4% solution is not licensed for wound cleansing.

It has been suggested it is the antibacterial properties of Chlorhexidine Gluconate 0.05% that can decrease the bacterial load in wounds by creating an environment that promotes cell migration and growth (Drosou et al, 2003). Some authors have argued it is in fact the action of cleansing the wound bed of debris and exudates rather than the antimicrobial effects of the product which can enhance wound healing outcomes therefore quality tap water should be sufficient (Lawrence, 1997; Main, 2008). Some studies focusing on Chlorhexidine 0.05% as an effective antimicrobial cleansing agent have argued that success would depend on the product being in contact with the wound bed for at least 20 minutes during which the product could be deactivated by body fluids and skin maceration an increase risk.

Earlier studies reported toxicity to new cell growth and researchers have raised caution when weighing up the perceived benefits of Chlorhexidine 0.05% as an antimicrobial wound cleansing agent against possible detrimental effects of the product.

Much of the existing evidence on the use of Chlorhexidine 0.05% in wound care dates back to the early 1990s. There is emerging consensus within research that the use of Chlorhexidine 0.05% as a wound cleansing agent is ritualistic practice (Main 2008). With so many variables in research outcomes, the use of this product should be reconsidered unless further evidence can demonstrate the benefits of use outweigh the potential toxic effects (Main, 2008)

At present the Tissue Viability Team are unable to advocate the use of Chlorhexidine 0.05% for wound cleansing within the community until further evidence can demonstrate improved efficacy and safety over other techniques such as the use of quality tap water and effective wound bed management. **Chlorhexidine 0.05% is therefore not recommended for use as a wound cleansing agent within the primary care setting in Oxfordshire**

Trimovate Topical Steroid Cream

Trimovate® is a cream licensed for the treatment of inflammatory skin conditions where there is a risk of/or infection present (BNF, 2011). Trimovate contains three therapeutic agents: -

- Nystatin: - antifungal element
- Oxytetracycline Calcium – antibiotic element
- Clobetasone Butyrate: - Steroid element, helps reduce inflammation and relieve symptoms of inflammatory skin conditions.

(EMC. 2007)

There appears to be an increasing use of Trimovate by clinicians in the community, mainly for leg ulcers that are failing to progress or for wounds that are overgranulating. Any reports of an improvement to wound healing status can be believed as it is likely this is due to the steroidal agent reducing local wound bed inflammation and the antimicrobial agents within the product reducing the bacterial colonisation that can often be responsible for delayed healing.

Topical steroids can increase cytokines, such as platelet derived growth factor (required for epidermal proliferation), and T cell growth factor (required for keratinocyte migration in the maturation phase of healing). However, using topical steroids directly on the wound bed for long periods and in large doses can cause the same side effects as systemic preparations and the sensitisation and resistance to antibiotics following prolonged use of Trimovate is also a concern.

With this in mind, the community tissue viability service do not currently advocate the routine use of Trimovate cream or any other topical steroid in wound management due to the lack of scientific evidence concerning its use directly to the wound bed. The products are not licensed for this purpose and until there is a local agreement in place on the role of topical steroids in wound care, individual practitioners should be mindful of their professional accountability when considering this within a treatment plan. **Trimovate® cream and other topical steroids are therefore not recommended for use as a wound management agent within the primary care setting in Oxfordshire.**

There are many reasons why wounds fail to progress and the development of tools to help clinicians identify and manage the biological causes are currently in place together with guidance on alternative therapies to address any abnormalities. There are licensed topical wound dressings available on local formularies that have the potential to achieve similar results in a timely and safe manner. Practitioners should be considering these products as first line and should contact the tissue viability team for further guidance on chronic wound management to promote a more scientific and successful approach to wound care

Potassium Permanganate

This is an oxidising agent which is often used as a disinfectant and contains properties which aid vasoconstriction and is sometimes called Condy's Crystals. As a raw substance, Potassium Permanganate presents as an odourless dark purple or almost black crystal or granular powder. In practice, the tablets are most convenient and are dissolved in water to a concentration of 1 in 1000. This is usually issued for the treatment of infected eczema or blistering wounds such as ulcers or abscesses once all standard treatments have been exhausted (DermNet NZ, 2006, accessed 06/02/2012).

Potassium Permanganate is advocated for weeping, exuding skin or for blistered areas due to the vasoconstriction action of this product can help dry out blistering and prepare the wound bed for further treatment.

Due to the caustic nature of this product and the potential to burn or irritate skin prolonged use is not advocated. Treatment implementation should follow a comprehensive patient assessment with clear rationale for use (DermNet NZ, 2006, accessed 06/02/2012). This treatment should be stopped and patient reassessed after a two week period or if there is a visible improvement during this time.

Practitioners should seek advice from the tissue viability team to discuss treatment objectives prior to implementation. A clinical procedure for the safe use and disposal of Potassium Permanganate is available should this treatment be indicated. Practitioners should request this on contacting the team.

Appendix One: -

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Appendix Two

Glossary of Terms

Term	Description
Antibiotics	Antibiotics are substances that kill or interfere with the growth of microorganisms, especially bacteria (Healthcare Protection Agency, 2010)
Antimicrobial	Antimicrobials are substances which are used in the treatment of infection caused by bacteria, fungi or viruses (Department of Health, 2008)
Antiseptic	A disinfectant substance that can be used on skin and on wounds that either kills or prevents the multiplication of potential pathogenic organisms (Vowden and Cooper, 2006)
Colonisation	Multiplication of <i>organisms</i> in the wound without host reaction
Crepitus	A crackling sensation on tissue and joint palpation. May be felt in infection.
Cytotoxic	The destruction of new forming cells within the wound bed. This can delay healing.
Debridement	The removal of dead or contaminated tissue through a variety of methods.
Devitalised tissue	Tissue that is no longer viable.
Induration	The abnormal firmness or 'woodiness' of skin around the wound edges.
MMP	Matrix Metallproteinases are enzymes that break down collagens and help to remodel extracellular matrix in healing.
Pathogen	Any micro-organism that is capable of causing disease. E.g. bacteria, virus
Proliferation	The rapid multiplication or reproduction of cells.
Prostaglandins	Enzymes synthesised within the cell membrane and promote the inflammatory process
Purulent	Exudate containing pus.
Resistance	Micro-organisms such as bacteria develop resistance to systemic antibiotic/antimicrobial dressings
Sepsis	The presence of pathogenic <i>organisms</i> in the bloodstream or tissues.
Topical	An antimicrobial agent incorporated into a dressing, powder or cream for direct application to the wound

Appendix Three

Primary Care Wound Swabbing Procedure

The process of routine wound swabbing is not advocated as a first line approach to diagnosing or treating local wound bed infection or colonisation.

Wound swabs should only be taken when there are clinical symptoms and signs of infection **and** where systemic antimicrobial (antibiotic) therapy is being considered. This procedure has been developed in conjunction with the local Oxfordshire Adult Antimicrobial Prescribing Guidelines for Primary Care (2012) guidelines approved by the Area Prescribing Committee Oxfordshire and Oxfordshire Clinical Commissioning Group with local antimicrobial guidelines endorsed by the Oxfordshire PCT/GP Consortium.

Please refer to the guidelines which state the clinical criteria for taking wound swab. The Levine swabbing technique is advocated in line with research evidence (Angel et al, 2011 and Gardner et al, 2006) and specialist consensus.

Before a wound swab is taken any contaminating materials such as slough or necrotic tissue should be removed with a short course of an appropriate topical wound dressing for rapid debridement.

Equipment Required:-

- A sterile or clean field to work from as appropriate for the environment and type of wound.
- Appropriate microbiology request form.
- Warmed good quality tap water if required
- Wound swab and correct transport medium – one per wound
- Softdrape Dressing Pack
- Appropriate primary dressing.
- Bag/clinical waste bag for disposal for soiled waste

Taking a wound swab:-

- Aim to gain informed consent to carry out the wound swab. Ensure the patient has received effective, prescribed pain analgesia prior to procedure if indicated.
- Decontaminate hands using soap and water or alcohol gel - see infection control hand hygiene procedure for guidance.
- Apply personal protective equipment: apron and non-sterile gloves.
- Remove soiled primary and secondary dressing from the wound bed and place in the bag provided in the dressing pack. Avoid wound cleansing with tap water prior to swabbing.
- Remove any visible debris or exudates from the wound bed using one gentle swipe of the wound bed with dry sterile gauze.

- Decontaminate hands using soap and water or alcohol gel - see infection control hand hygiene procedure for guidance and apply new non-sterile gloves.
- Remove the wound swab from the protective, sterile transporting container avoiding cross contamination of the cotton tip and inside the transporting container.
- Whilst holding the cotton tip horizontal to the wound bed, rotate between fingers and apply a sufficient pressure to express fluid from approximately 1 cm² of the central wound bed, avoiding contact with wound edges (WUWHS, 2008)
- Put the swab into the transport medium and send to the microbiology laboratories as soon as possible via your laboratory transport system. If there is an unexpected delay the sample, although not ideal, maybe stored in the fridge over night.
- Decontaminate hands using soap and water or alcohol gel - see infection control hand hygiene procedure for guidance and re-apply gloves provided within the Softdape dressing pack.
- Re-dress the wound bed as per nursing wound management plan as soon as possible after the wound swab has been taken to prevent temperature changes at the wound bed. Wounds can lose heat very quickly and this can affect the healing process.
- Place any soiled wound dressing materials, including soiled gloves and aprons into the clinical bag and dispose of this as per local clinical waste disposal protocols.
- Decontaminate hands using soap and water or alcohol gel - see infection control hand hygiene procedure for guidance.
- Complete the microbiology request form with comprehensive patient information such as:
 - Enter the request in the GP electronic requesting system (ICE) if possible, as this minimizes the chance of data entry errors and ensures the result is sent back electronically to the correct GP as soon as it is available.
 - patients full name (not initials), date of birth and NHS number
 - Ensure there is a clear contact and address for return of results.
 - Include the name and practice address of registered GP
 - Please state any antimicrobial dressing or systemic antibiotic the patient is about to start or has already used.
 - Please state the anatomical location from which the swab was taken.

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