Controlling wound infection in the hospital and home

The prevention of wound infection, whenever possible, is paramount in order to avoid a whole spectrum of potential diseases. This article examines the ways in which infection can be prevented or, at worst, controlled in the healthcare setting and the patient's home.

atients may experience a wide variety of wounds, many of which will heal without difficulty, however, some will become infected and may cause a spectrum of disease ranging from superficial infection to more serious deep infection — the latter may lead to septicaemia and even possible fatality. Wound infection may arise due to the complex interaction between a host and potentially pathogenic microorganisms, and can occur in any setting where patients are found to have a wound, both within the hospital and the community. Preventing wound infection, whenever possible, is therefore, vitally important.

Physiology of normal wound healing

Understanding the normal physiology of wound healing is central to understanding wound infection. Wound healing is a complex dynamic process occurring in sequential and overlapping phases - inflammation, reconstruction, epithelialisation and maturation (Dealey, 2012). The length of time taken to progress from one phase to another depends on the type, size and depth of the wound, along with patientspecific factors that can assist or impede the normal wound healing process. For example, diabetes mellitus is known to adversely affect wound healing due to reduced neutrophil (a type of white blood cell) activity (Wilson, 2006).

Once the protective barrier of the skin is broken, the normal physiologic process of wound healing is immediately initiated and within minutes of wounding platelets aggregate at the wound site to form a fibrin clot, in order to limit blood loss (Stadelmann et al. 1998). During the inflammatory phase, neutrophils rapidly congregate at the wound site (McCance and Huether, 2002) and macrophages (cells produced by the differentiation of monocytes in tissues) remove foreign matter and dead or dying cells through the process of phagocytosis (the ingestion of bacteria or other material by phagocytes and amoeboid protozoans), to prepare the wound for healing (Tortara and Grabowski, 2003).

Vasodilation increases the blood flow to the wound site, releasing chemical mediators, such as cytokines. This increases the permeability of the capillaries allowing neutrophils and fluid to pass into the surrounding tissues (Dealey, 2012).

The increased fluid in the tissues exerts further pressure on the nerve endings and produces pain around the wound site. In addition, increased vasodilation also produces redness, swelling and local heat at the wound site (Dealey, 2012). In some situations, healthcare workers may mistake the inflammatory phase as a sign of infection, as inflammation Understanding the normal physiology of wound healing is central to understanding wound infection

SHILA PATEL is a Lead Nurse, Infection Prevention and Control, at Epsom & St. Helier University Hospitals NHS Trust is also associated with wound infection. However, these signs are not indicative of wound infection at this stage of the healing process (Morrison et al, 1999).

The inflammatory phase acts as a trigger for the subsequent wound healing phases, but in some situations normal wound healing does not progress and this phase becomes prolonged. This may occur as a result of wound infection and is also seen in chronic and necrotic non-healing wounds. Prolonged inflammation increases macrophage activity and the release of cytokines, which, in turn, further stimulates the inflammatory process (Bryant, 2000).

To determine whether wound inflammation is part of the normal wound-healing process, chronic inflammation or a consequence of wound infection, it is important to monitor the progress of wounds. Looking at the wound regularly will help to determine which state it is in (although it is equally important to ensure that wounds are not disturbed unnecessarily, as this may increase the risk of introducing infection and interfere with wound healing). With chronic inflammation, the classic signs of redness, local heat, swelling and pain can be noted whereas in inflammation associated with wound infection there is also odour, discharge of pus and systemic signs, such as pyrexia, raised white blood cell count and raised serum C-reactive protein (CRP), which is normally not found in serum, but is present in many acute inflammatory conditions and with necrosis (see *Table 1*).

Individual vulnerability vs bacterial virulence

A number of factors influence the development of wound infection. Microbial characteristics, including the type and/or quantity of microorganisms present, will have an influence (Scanlon, 2005). All microorganisms have differing levels of virulence (ability to produce disease). While some microorganisms can be present in wounds in very large numbers without producing disease - for example, methicillin-resistant Staphylococcus aureus (MRSA) can be found in large numbers in chronic leg ulcers without causing infection - in contrast, other microorganisms, such as beta-haemolytic streptococci, can cause infection even in very low numbers due to their increased virulence factors (Robson, 1997).

Individual vulnerability to infection and in particular the ability of the host to mount an adequate immune response is often critical in determining the role of bacteria within wounds. Host resistance is influenced by a number of factors. Behavioural factors include smoking, and drug and alcohol abuse, both of which can adversely affect the normal wound healing process due to reduced tissue perfusion (Sibbald et al, 2003). Circulatory impairment, such as cardiovascular disease, may also result in reduced blood and oxygen supply within wounds, slowing the normal woundhealing process and increasing the risk of wound infection (Dealey, 2005).

A poor diet, in particular insufficient intake of vitamin B complex and vitamin C, can result in a poor immune response, in particular impaired phagocytosis, as well as an overall reduction in white blood cell production (Dealey, 2005). A decreased immune response is also associated with advancing age, diabetes mellitus, concurrent infections, stress and disease-related immunosuppression (this includes a variety of genetic disorders, autoimmune diseases and chemotherapy as well as some viral infections, such as the human immunodeficiency virus [HIV]) (Wilson, 2006).

Body size and obesity in particular acts as an important risk factor for surgical site infection (Cruse and Foord, 1973) as deep layers of adipose (fat) tissue can complicate the surgical procedure and reduce blood flow to the wound site during healing (Mangram et al, 1999). Wound characteristics, such as size, position, duration and presence of dead tissue or blood clots, can also play a significant role (White et al, 2001).

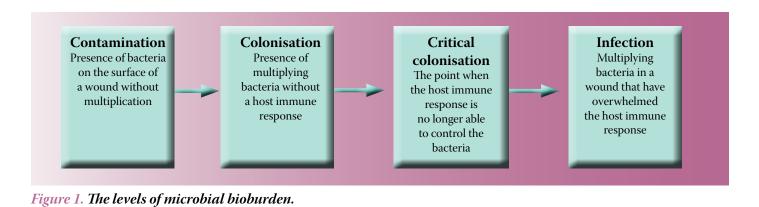
When all the above considerations are taken into account, wound infection arises when the microbial bioburden overcomes host immunity.

Recognising wound infection

All wounds contain a variety of microorganisms in varying numbers, dependant on how long a wound has existed, the wound type and its location on the body. However, the mere presence of microorganisms is not by itself indicative of wound infection — the level of microbial bioburden can range from contamination, colonisation, critical

Table 1 Signs and symptoms of wound infection

Classic signs	Additional signs
 Pyrexia Inflammation Oedema Pain Increase in exudate or pus 	 Delayed healing Bridging of skin across a wound Dark/discoloured granulation tissue Increased friability (tissue that bleeds easily) Painful/altered sensation to the wound site/ surrounding skin Altered odour Wound breakdown Pocketing at the base of the wound Increased watery/serous exudate rather than pus



colonisation and infection (White, 2003), (see *Figure 1*).

These definitions indicate that contamination and colonisation are not associated with ill health and can be regarded as normal states. In contrast, critical colonisation and infection can result in mild-to-severe disease and are, therefore, abnormal states. However, colonisation can act as a precursor to infection in vulnerable individuals, for example, colonisation with MRSA can lead to MRSA-related wound infection in those who are susceptible.

In order to accurately diagnose wound infection, relevant clinical signs and symptoms must be considered (see Table 1), along with a review of microbiology results from wound samples, such as wound aspirate or wound swabs. However, it is important to note that positive microbiology results are not by themselves indicative of wound infection — they only show whether microorganisms have been identified through wound sampling and, if so, which microorganisms. A number of bacteria may be isolated from wound sampling, but they may be harmlessly colonising the wound or surrounding skin (Wilson, 2006).

Due to the difficulty in distinguishing between bacteria colonising or infecting a wound, microbiology results should be interpreted with caution (Wilson, 2006). In order to determine microbial impact on a wound, a thorough clinical assessment of the patient and their wound should be performed. Many patients may present with frank signs and symptoms of infection, however, making an accurate diagnosis can be harder when wounds display few signs of wound infection or they are hard to distinguish. Sibbald et al (2003) indicates that this may occur when the patient is harmed enough to delay normal wound healing but not enough to cause the typical inflammatory symptoms; or where the patient is unable to mount an adequate immune response due to other comorbidities.

Preventing wound infection

Standard infection control precautions should be practised during all aspects of healthcare and with all patients. As it is often not known which patients are colonised with potentially pathogenic microorganisms, the precautions should be implemented at all times to combat this unknown risk. While it is not possible to prevent all infections (particularly in patients with high intrinsic vulnerability), the precautions aim to reduce the risk of infection as much as possible, by breaking the chain of infection. The following precautions are applicable in all healthcare settings, including hospitals, clinics and surgeries, as well as non-clinical settings, such as the patient's own home, where healthcare is delivered (Department of Health [DoH] 2006; Pratt et al, 2007).

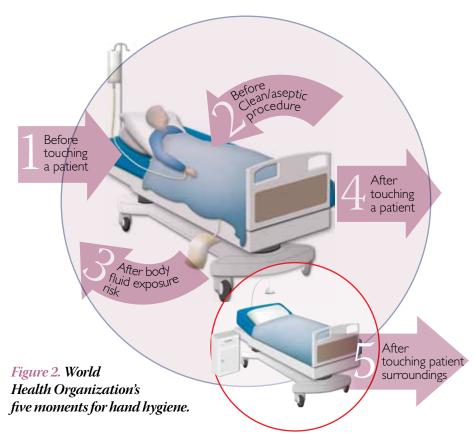
Standard infection control precautions Hand hygiene

Hands can commonly become contaminated during contact with patients or the environment and act as a vehicle for further transmission. The point of care is the time and place at which there is the greatest likelihood of transmitting potentially pathogenic microorganisms that may result in infection (National Patient Safety Agency [NPSA], 2008).

Clinicians should apply the World Health Organization's 'Five Moments for Hand Hygiene' at the point of care — see *Figure* 2 (Sax et al, 2007).

In clinical settings, both in primary and secondary care, hand washing should be undertaken at a clinical hand wash basin, using running water, liquid soap and disposable paper hand towels (NPSA, 2008). Alternatively, alcohol hand rub/gel can also be used to decontaminate hands, providing they are not visibly soiled alcohol hand rubs/gels do not work in the presence of physical soiling, such as body fluids, and are also ineffective for norovirus, Clostridium difficile and other diarrhoeal infections (NPSA, 2008). Irrespective of the product used, staff should always use the correct hand hygiene technique, ensuring all surfaces of the hands, including thumbs and wrist areas, are fully covered to achieve a high standard of hand decontamination (Wilson, 2006).

For practitioners working in patients' own homes, hand washing should be undertaken when there is access to a clean basin or sink, liquid soap in a pump action dispenser, paper hand towels, absorbent paper roll, or clean fabric towels. Where these facilities are unavailable or are not ideal, staff should



ensure that they have access to alcohol hand rub/gel by carrying a supply with them when visiting patients at home (Health Protection Agency South West, 2007).

Patients should also be educated about the importance of good hand hygiene at appropriate points in time, such as after using the toilet, in order to reduce the risk of the spread of microorganisms from areas of their body to more vulnerable sites, such as wounds.

Personal protective equipment

Personal protective equipment (PPE), such as disposable plastic aprons and disposable gloves, should be worn when there is a risk of infection to the patient and to minimise the risk of crosscontamination to the healthcare worker's clothing (National Institute for Clinical Excellence [NICE], 2012).

Wound care is an indication for the use of PPE, which should be single use and always changed between patients and procedures (even procedures on the same patient) (Pratt et al, 2007). PPE should always be put on immediately before and removed immediately after the task for which it is required — in this case any aspect of wound care. Failure to do so may increase, rather than decrease, the risk of infection, because gloved hands may acquire pathogenic microorganisms from environmental surfaces and spread to the patient or vice versa.

Waste disposal

All healthcare workers have a duty of care to correctly segregate and dispose of any waste they produce. Soiled wound dressings, along with used gloves and aprons should be assessed for infection risk and disposed of appropriately, while assessments should be carried out on a patient-specific basis (DoH, 2011). Where wound infection is suspected, i.e. the patient has clinical signs and symptoms, soiled dressings and used PPE should be disposed of as hazardous infectious waste (DoH, 2011).

In healthcare settings, such as hospitals or nursing homes, staff should dispose of infectious waste into bins with orange colour-coded waste bags and these will be consigned for terminal disposal as hazardous infectious waste by the healthcare organisation.

For healthcare workers visiting patients in their own homes, soiled wound dressings should be tied securely into an orange waste bag, placed in a rigid secure container and transported back to base for terminal disposal via the employer's infectious waste disposal systems. Waste transported in an individual's car will not pose a risk of infection to others in the vehicle, providing the waste is transported in this safe manner.

Alternatively, where wounds require frequent redressing and a large quantity of infectious waste is generated, the healthcare worker may arrange for the waste to be collected by a waste contractor or the local authority (where this service may be available). In this situation, the healthcare worker has responsibility for ensuring the waste is stored safely while awaiting collection and should discuss this with the patient.

Environment and equipment cleanliness

In general, it is considered that the environment plays a relatively minor role in the transmission of infection, however, surfaces that accumulate dust and liquid residues, along with frequent touch sites, may act as vectors for transmission (Fraise and Bradley, 2009). Therefore, in the healthcare setting, it is important that environmental surfaces are cleaned frequently enough to minimise dust accumulation and liquid residues. Environmental cleanliness is also important in the patient's own home, particularly when delivering wound care, however, tact is needed when discussing this with patients, as they may feel their home is already clean enough (Health Protection Agency South West, 2007).

Patient equipment or equipment used within the patient area, may also act as vectors for transmission of microorganisms. Therefore, all equipment, such as procedure trolleys, should be cleaned following each use (Pratt et al, 2007). In addition, a robust system should be put in place for cleaning all equipment thoroughly and on a regular basis, including stored equipment, so that it does not accumulate dust or dirt. Dust found inside buildings poses a risk of crossinfection. It is largely made up of shed skin squames and lint fibres from clothing and other fabric furnishings (skin squames are flat flakes of dead skin that are shed at a rate of 300 million per day and approximately 10% carry microorganisms) (Wilson, 2006).

Wound care

The aim with any wound is to achieve healing as quickly as possible, as intact skin acts as a natural barrier to potentially pathogenic microorganisms. Accurate wound assessment is, therefore, imperative, in order to facilitate selection of an appropriate wound dressing product, which is most suited to the wound type and will promote rapid wound healing.

In addition, aseptic non-touch technique (ANTT) should be used (Rowley et al, 2010), with a view to maintaining asepsis, which involves keeping the clinical area free from pathogenic microorganisms. Although chronic wounds are already colonised with a variety of microorganisms, the aim is to avoid introducing new microorganisms/ pathogens into a vulnerable site, as polymicrobial communities can gain virulence and give rise to wound infection (Kingsley, 2008). While nonsterile gloves can be used for redressing a chronic wound, such as leg ulcers, the aim nevertheless remains to maintain asepsis.

Healthcare workers should, therefore, have a good understanding of the principles of ANTT, which focuses on keeping key sites free from contamination, in order to prevent introduction of pathogens that may lead to infection. Patients should also be educated about avoiding unnecessary wound handling and what to do in the event of dressing strikethrough, such as safe application of a secondary dressing.

Conclusion

Wound healing and infection are complex processes. Healthcare workers need to have a good understanding of each process, whether working in the hospital or community setting. Standard infection control precautions must be applied to reduce the risk of wound infection whenever possible and patient education also plays an integral role in reducing this risk. WE

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