



# Wound Bed Preparation

## It's About TIME

Wound infection delays wound closure. It prolongs the inflammatory phase of healing and often causes distress and discomfort for patients. Wound infections also have considerable financial implications due to increased length of hospital stay, the additional cost of antimicrobial therapy, and a higher incidence of related wound management complications.

Increased bacterial burden, identified as “infection/inflammation” in the in the TIME model (T — Tissue, nonviable or deficient; I — Infection/Inflammation; M — Moisture Imbalance; E — Edge of wound non-advancing or undermined), may not be obvious in all wounds; in the chronic wound, signs of infection may be quite subtle and, therefore, overlooked. Prompt identification and management of infection is essential in the management of chronic wounds.

This is the sixth in a series of 12 articles that discuss various aspects of the TIME principle.



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## The Problem — Infection and Inflammation that Impair Wound Healing

The complete removal of bacteria from a wound is neither possible nor necessary in order to promote healing. All chronic wounds contain bacteria and their presence in the wound does not necessarily indicate that infection has occurred or has impaired wound healing.<sup>1,2</sup> Bacterial colonization alone is not clinically significant and should not be confused with a clinical diagnosis of wound infection. However, when bacteria are sufficiently virulent to compromise host response, wound healing is affected.

**Bacterial involvement.** Bacterial involvement within a wound can be divided into four categories. *Contamination* refers to the presence of non-multiplying bacteria within a wound and accounts for the majority of the micro-organisms present on the wound surface.<sup>3</sup> *Colonization* refers to the presence of bacteria that are multiplying but are producing no host reaction. *Critical colonization* may be defined as increased bacterial burden (multiplication of organisms) within the wound that initiates the body's immune response locally but not systemically and results in delayed healing. *Infection* is the presence of multiplying bacteria that cause an associated host reaction.

The term *host reaction* describes a variety of different signs and symptoms such as cellulitis and increased exudate levels that may occur in clinical practice once bacteria overwhelm the body's normal healing process. A pathogenic micro-organism may initially colonize a wound without inducing a host reaction. However, as the bacterial burden increases, the colonized wound gradually transforms into an infected wound.<sup>4</sup> Ultimately, with increasing bacterial burden, wound infection becomes more evident or systemic sepsis can occur.<sup>2</sup>

Pathogenic micro-organisms responsible for wound infection prolong wound healing, destroying cells by competing for available oxygen supplies within the wound; releasing toxins that damage tissue locally, causing necrosis and pus formation; and releasing toxins into the blood stream that may cause toxemia.

**Bacterial screening.** Wound swabs are a relatively unreliable method of identifying wound infection; all wounds contain bacteria and no international consensus exists regarding the best method to sample bacteria effectively. Bacteria within wounds are transient — a single bacteriological sample alone cannot indicate whether the bacterial count is rising or falling. Therefore, the established practice of diagnosing wound infection by isolating bacteria in the wound bed from a single bacteriological swab is now considered inadequate and misleading.<sup>5</sup>

In order to increase specimen collection accuracy for quantitative bacteriology, microbiologists recommend performing needle aspiration or biopsies utilizing several areas in the wound bed. However, this is an invasive procedure and rarely performed in clinical practice. The diagnosis of wound infection, therefore, is likely to be most accurate if based on the presence or absence of the objective clinical signs rather than by bacteriological analysis in isolation.

Most clinicians are aware of the “classic” signs and symptoms

Reduced perfusion	Metabolic disorders —
Large wound area/depth	diabetes mellitus
Chronicity	Alcohol abuse/smoking
Necrotic tissue	Corticosteroid medications
Foreign bodies	

**Figure 1.**  
**Systemic factors that increase infection risk.<sup>3</sup>**

of infection (advancing erythema, fever, warmth, edema, pain, purulence) often seen in the acute wound or the severe chronic wound. However, many clinicians often overlook the subtle “secondary” signs and symptoms associated with infection in the chronic wound. Secondary signs of infection include:

- Delayed healing
- Change in color of wound bed
- Friable granulation tissue
- Absent or abnormal granulation tissue
- Increased or abnormal odor
- Increased serous drainage
- Increased pain at wound site.<sup>6,7</sup>

A number of additional variables are known to affect the bacterial burden of a wound and increase the risk of wound infection. These include the amount of necrotic or slough tissue present in the wound bed, the number of organisms present, bacterial pathogenicity, and host factors.<sup>3</sup>

**Host resistance.** Host monitoring is a critical aspect of wound assessment and management. According to Dow, Browne, and Sibbald,<sup>2</sup> “Host resistance is the single most important determinant of wound infection and must be meticulously assessed in every situation where a chronic wound fails to heal.” Local factors that increase the likelihood of wound infection include wound size, depth, and duration. A larger wound is associated with greater host impairment during wound healing and, consequently, a greater risk of infection. Vascular status is equally as important — wounds with a reduced arterial pressure will remain unhealed.<sup>8</sup> The extent of wound perfusion also must be considered because an inadequately perfused wound is unlikely to show the typical signs of inflammation. A number of systemic factors also increase infection risk (see Figure 1). In addition, immunosuppressive drug use may mask signs of localized or systemic infection.<sup>2</sup>

**Increased bacterial burden.** An increased bacterial load within chronic wounds can delay or inhibit healing. Experimental evidence indicates that, regardless of the type of organism, substantial impairment of wound healing occurs when the wound bed contains between  $10^5$  and  $10^6$  organisms per gram.<sup>2</sup> However, the number of organisms may not necessarily be as critical as the type and pathogenicity of the organisms in the wound. Furthermore, increased bacterial load often increases the amount of wound exudate, requiring effective management to avoid providing a venue for increased bacterial infection.

**Biofilms.** Sheets of soft adherent material (biofilms) are highly organized bacterial communities that allow individual organisms to interact with each other, exchanging nutrients and metabolites. Biofilms represent a foci of infection and

bacterial resistance within the wound that protect the bacteria from the effects of antimicrobial agents, especially antibiotics and antiseptics.<sup>9</sup>

## The Solution — Addressing Bacterial Burden as an Obstacle to Healing

Identifying and reducing the bacterial burden in the wound is an important component of preparing the wound for closure. Bacterial burden can be reduced by removing necrotic tissue, purulence, and excessive exudate — all of which act as mechanical barriers at the wound surface and provide an environment for bacterial proliferation. The early detection and treatment of wound infection limits the amount of local tissue damage and minimizes disruption of the healing process. Infected wound treatment objectives include:

- Identifying the infective organism
- Removing devitalized tissue and excess exudate from the wound bed
- Eliminating wound infection using appropriate antimicrobial agents
- Protecting the surrounding skin from the effects of maceration.

**Antimicrobials.** Since the introduction of topical antiseptics 150 years ago, a wide range of antiseptics has been used to prevent and treat wound infections. During the last 25 years, much conflicting research has emerged regarding the effectiveness of antiseptics on open wounds. Interpretation of findings is difficult, as many studies have been conducted on animals, sample sizes are often small, and methodologies are inconsistent. The clinical significance of these results for human wounds is still open to debate but an important goal has emerged: to balance the effects of bacteria against the potential cytotoxicity of antiseptics.

Currently, the literature lacks consensual findings. Some evidence supports the selective use of antiseptics to stimulate previously unresponsive chronic wounds, treat critically colonized or infected wounds, or eradicate methicillin-resistant *Staphylococcus aureus* (MRSA) from contaminated wounds.<sup>3,10</sup> If used correctly, topical antiseptic agents, despite their cytotoxic properties, can be effective antibacterial agents.<sup>3</sup>

As bacteria become more resistant to antibiotics, interest grows in the selective use of topical antimicrobial agents. Studies have demonstrated that some iodine and silver preparations have bacteriocidal effects, even against multiresistant organisms such as MRSA.<sup>10,11</sup> Furthermore, in contrast to antibiotics, which have a more specific mode of action and are effective against a narrower range of bacteria, broad spectrum antimicrobial agents provide action across three target areas — the cell membrane, cytoplasmic organelles, and the bacteria's nucleic acid.<sup>11</sup>

ACTICOAT® brand (Smith & Nephew, Largo, Fla.) is an example of an antimicrobial barrier dressing. The ACTICOAT® brand has the only wound care products to feature patented Silcryst® nanocrystalline silver technology. They have been shown by *in vitro* testing to be bactericidal against a broad range of pathogens, including MRSA and vancomycin-resistant Enterococcus (VRE), and are important components of burn and chronic wound care. ACTICOAT offers 3-Day, 7-Day, Burn, and Absorbent dressings and the new ACTICOAT Moisture Control.

**Antibiotics.** Antibiotic resistance is the result of bacteria's genetic adaptability to develop enzymes that can dismantle antibiotics either before or after they enter the organism. Some bacteria can actively pump the antibiotics out of the cell or alter the shapes of the molecule to which the antibiotic binds. Thus, the actual or potential increase in the occurrence of bacterial resistance has become a major concern when using antibiotics for treating wound infections. Consequently, the use of antibiotics has been restricted to situations for which they are absolutely necessary. Systemic antimicrobial therapy should be used when active infection cannot be managed with local therapy (eg, fever, underlying deep structure infection, and spreading cellulites).<sup>3</sup>

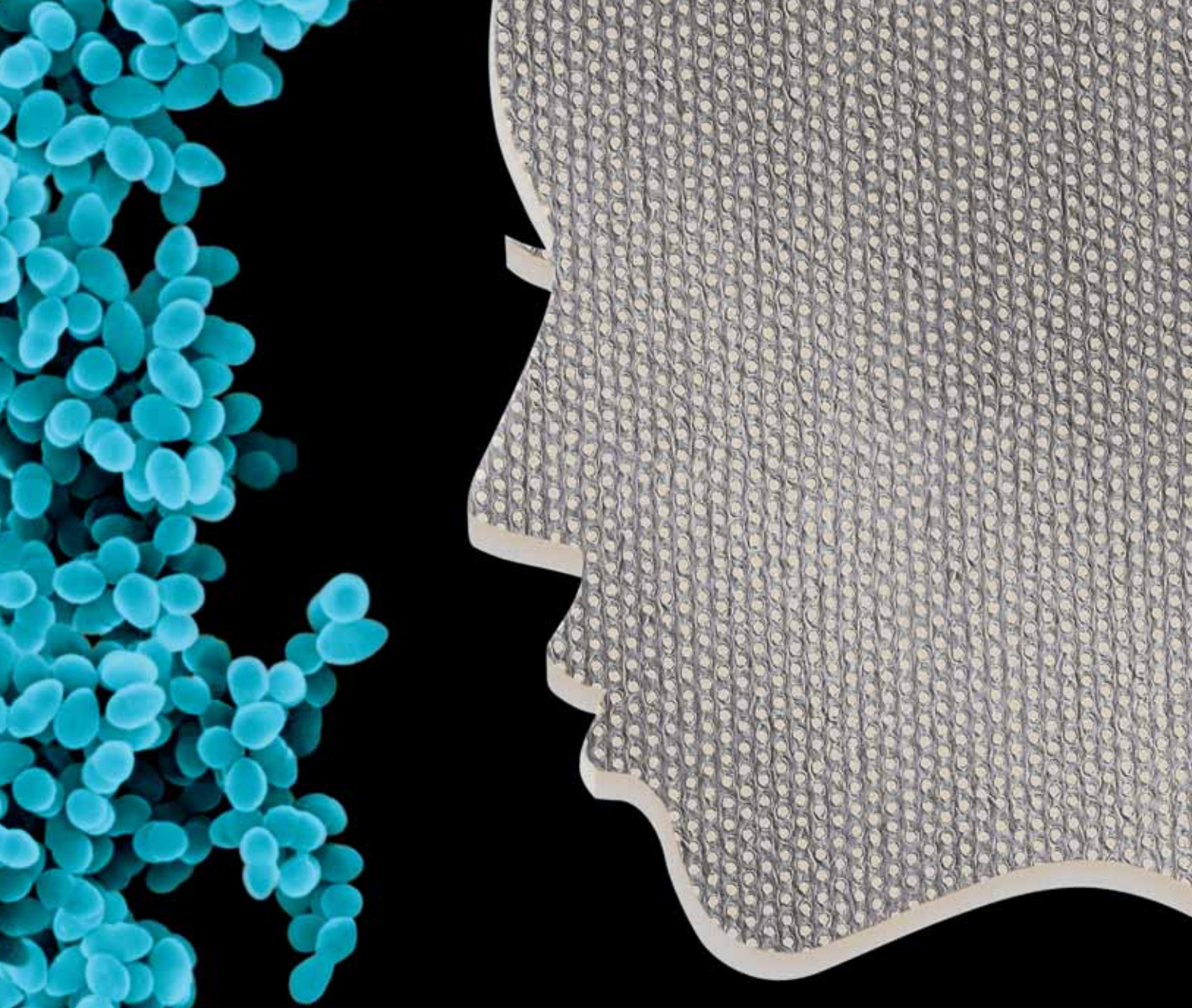
Topical antibiotics have no place in the management of wounds. No controlled trials demonstrate the superiority of topical antibiotics over antiseptics.<sup>12</sup> Using topical antibiotic powders on wounds is unjustified and in many cases has led to the development of bacterial resistance. Allergic reactions to topical antibiotics such as neomycin, framycetin, gentamicin, and sodium fusidate are common.<sup>13</sup>

## Conclusion

The risks of toxicity, sensitization, and bacterial resistance far outweigh any potential benefits of topical antibiotics in the treatment of wound infection. Without appropriate use of antibacterial agents, wound bed preparation will remain inadequate and bacteria will continue to thrive in the wound and delay healing.

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