Introduction

For many years, management of chronic wounds, e.g. pressure ulcers, has been based on an understanding of the physiology of acute wounds. Both acute and chronic wounds need to follow all four wound healing phases, namely haemostasis, inflammation, proliferation, and maturation or remodelling, in order to achieve wound closure. However, very often, the initiating mechanism of injury in chronic wounds does not require haemostasis.

Most chronic wounds are characterised by pro-inflammatory stimuli, e.g. local tissue ischaemia, repeated trauma, necrotic tissue, and an increased bacterial burden. These pro-inflammatory stimuli result in a pro-inflammatory wound environment, in which healing is less ordered and predictable than it would be in an acute wound environment.

The approach to chronic wound management needs to address the differences in wound healing which characterise this process. Therefore, this article will discuss the approach to chronic wound assessment, and focus specifically on the local wound environment.

In order to optimise the chronic wound treatment, it is important to properly assess the patient. The systemic environment will also impact on the local wound environment, and ultimately, healing of the wound.

To understand how to best manage chronic wounds and promote healing therein, attention will be given to potential local barriers to healing, and very briefly, to systemic factors.

Unlike the predictable acute wound healing process, chronic wounds do not always follow an orderly, or predictable, sequence of healing. A large percentage of pressure ulcers develop in the elderly and in gravely ill patients. Many of these patients have underlying co-morbidities, impaired immune systems, and are potentially malnourished. The risk of pressure ulcers developing is quite high, due to these underlying systemic factors.

Table 1 lists a number of systemic factors that may prolong the wound healing process (Flanagan).

Table I: Systemic factors that may prolong the wound healing process

<table>
<thead>
<tr>
<th>Systemic factors</th>
<th>Local factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic disorders: diabetes mellitus, renal failure</td>
<td>Necrosis</td>
</tr>
<tr>
<td>Respiratory disorders: chronic obstructive airways disease</td>
<td>Infection</td>
</tr>
<tr>
<td>Circulatory disorders: anaemia, congestive cardiac failure</td>
<td>Prolonged inflammation</td>
</tr>
<tr>
<td>Immune deficiency: HIV, rheumatoid arthritis, malignancies</td>
<td>Exudate</td>
</tr>
<tr>
<td>Immunosuppressive therapy</td>
<td>Cellular dysfunction</td>
</tr>
<tr>
<td>Nutritional state: dehydration, vitamin deficiency</td>
<td>Biochemical imbalance: pH</td>
</tr>
<tr>
<td>Medication: steroids, anticoagulants</td>
<td>Hypoxia</td>
</tr>
</tbody>
</table>

Managing the local wound environment in chronic wounds is vital to create the ideal environment in which to facilitate endogenous wound healing. As mentioned earlier, the acute wound model cannot be applied to the management of chronic wounds. The concept of wound bed preparation has gained recognition as a
basic framework within which to facilitate a structured approach to chronic wound management.

The concept of wound bed preparation focuses on three pillars (Figure 1), namely:
- Debridement
- Bacterial burden
- Exudate management.

![Figure 1: The three pillars of wound bed preparation](image)

**Assessing the need for debridement**

Necrotic tissue in the chronic wound creates a local barrier to wound healing. Many clinicians and healthcare workers will assess a wound with slough, and comment on the fact that the wound is "septic". This is not always an accurate assessment. In order to apprehend the concept of necrotic tissue, and how it contributes to non-healing within the local wound environment, it is important to understand what necrotic tissue is.

Necrotic tissue accumulates in a wound as healthy viable tissue dies. As the latter occurs, the colour and consistency of the tissue changes. Necrosis can begin as a white-grey diffuse covering in the wound, and progress to yellow and fibrinous, then to tan, brown or black. As the necrotic tissue dehydrates, the consistency changes from mucoid, which has a high water content, to desiccated, which has a leathery-to-hard appearance.

<table>
<thead>
<tr>
<th>Colour</th>
<th>Black or brown eschar</th>
<th>Tan or yellow slough</th>
<th>Yellow fibrinous</th>
<th>White or grey</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consistency</td>
<td>Hard</td>
<td>Soft or soggy</td>
<td>Soft or stringy</td>
<td>Mucinous</td>
</tr>
<tr>
<td>Adherence</td>
<td>Firmly attached base and edges</td>
<td>Attached to base</td>
<td>Loosely attached</td>
<td>Clumps</td>
</tr>
<tr>
<td>Appearance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table II shows the changes in the colour and consistency of necrotic tissue.

The presence of necrotic tissue does not immediately imply wound infection in the absence of corroborating clinical signs and symptoms. Necrotic tissue retards wound healing, as it creates a physical barrier to wound contraction and re-epithelialisation. Due to the avascularity of necrotic tissue, it is an ideal medium for bacterial growth. The resultant enzymes and production of proteases further delay wound healing.

The type of necrotic tissue in a pressure ulcer may be indicative of the degree of tissue destruction. Deep tissue injury in pressure ulcers can initially appear as a blue or purplish discoloration, but as this evolves, the necrosis can, or will, usually demarcate. In order to facilitate wound healing, the pro-inflammatory stimuli, i.e. the necrotic tissue, needs to be removed (debrided).

**Common methods of wound debridement**

The most common accepted methods of wound debridement are surgical or sharp, mechanical, autolytic, and enzymatic.

**Surgical or sharp debridement**

This is the fastest way to achieve a clean and viable wound bed, especially if the depth of the wound cannot be assessed due to the presence of thick adherent necrotic tissue. Should there be signs or symptoms of a spreading infection, the removal of the necrotic tissue will immediately decrease the bacterial burden in the wound. Surgical debridement also removes any senescent cells often found in the chronic wound, and the wound environment can momentarily mimic that of an acute wound.

**Mechanical debridement**

This refers to any method that mechanically removes necrotic tissue, or debris, from the wound environment. Examples of these are wet-to-dry dressings, whirlpool
therapy, and wound irrigation, if performed with pressure.

**Autolytic debridement**

To a large extent, autolytic debridement occurs naturally in the wound in the presence of appropriate levels of wound exudate. Wound exudate contains phagocytic cells (macrophages) and proteolytic enzymes that can liquefy and separate non-viable tissue from viable tissue. Dressings that maintain, or facilitate, a moist wound environment, will facilitate or enhance the natural process of wound debridement.

**Enzymatic debridement**

The application of an enzymatic debriding ointment is essentially the equivalent to an application of exogenous proteolytic enzymes. When applied to the surface of the wound, enzymatic debridement works synergistically with the naturally occurring proteolytic enzymes in the wound, to degrade necrotic tissue from the wound bed. Enzymatic debridement essentially uses proteolytic enzymes to cleave the dead collagen from the viable collagen, thereby creating a viable wound bed.

The method of debridement will be dependent on several factors. These include:

- Size, shape and location of the wound
- The amount of exudate
- The patient's overall condition
- Cost
- Efficacy.

Due to their chronicity, more than one method of debridement may need to be utilised when managing necrosis in chronic wounds (Figure 2). For this reason, the concept of maintenance debridement needs to be explored in light of the fact that chronic wounds have an increased bacterial burden and biofilm formation.

Essentially, maintenance debridement refers to the need to continue with a process of wound debridement, even when the wound bed appears to be healthy and viable. In light of the above, especially because of the presence of biofilm in chronic wounds, maintenance debridement is often beneficial to maintain a clean wound bed to ensure wound healing.

Example: A patient with a Grade 3 or 4 pressure ulcer requires a surgical debridement. Once the wound has been surgically debrided, dressings need to be applied, e.g. with hydrogel and an enzymatic debriding agent, to continue debriding to keep the wound clean, so as to prevent the reaccumulation of necrotic tissue in the

Figure 2: Algorithm for the debridement of chronic wounds (Falanga et al)
Wound postoperatively.

Assessing wound infection

All chronic wounds contain micro-organisms. However, the presence of micro-organisms does not automatically indicate that the wound is infected, and requires treatment with antimicrobials. In fact, wounds are considered to be polymicrobial, i.e. when there is more than one type of micro-organism present. Whether or not these micro-organisms proliferate, and result in infection, is largely dependent of the local wound factors, e.g. necrotic tissue and poor perfusion, and underlying systemic pathologies.

Wound infection occurs when the micro-organisms overcome the natural host defences, and the local wound environment is favourable to microbial proliferation. This results in the invasion of proliferating micro-organisms into viable tissue, which elicits a local and systemic host reaction. At this point, wound healing will be delayed. The patient may exhibit systemic signs and symptoms, and require treatment with both a topical, as well as a systemic, antimicrobial.

Not all chronic wounds require treatment with antimicrobials. Chronic wounds exist along a continuum of infection. Where the chronic wound falls on this continuum will dictate if treatment with a topical, or systemic, antimicrobial, is required.

The bacterial continuum in wounds can be divided into four categories (Figure 3):

- **Wound contamination** micro-organisms are present in the wound. However, they do not replicate, and are non-adherent to the wound. They do not elicit a host reaction, and wound healing will occur.

- **Wound colonisation:** This represents the presence of micro-organisms within the wound environment. They replicate and adhere to the wound surface. However, the micro-organisms are usually skin contaminants, and do not interfere with wound healing, due to the fact that they are not yet invasive.

- **Critical colonisation:** At this stage, the micro-organisms in the wound replicate, are adherent, and invade the superficial tissue of the wound. Wound healing will be delayed. The wound exhibits subtle signs and symptoms of increased bacterial burden. Treatment with a topical antimicrobial is required, as well as optimisation of the local wound environment, e.g. debridement of the necrotic tissue.

- **Wound infection (local or systemic):** This refers to the presence of replicating and invasive micro-organisms within the wound. Wound healing is delayed, and the presence of the bacteria elicits a host reaction. If this local infection is left untreated, systemic dissemination can occur, with a resultant systemic reaction. At this stage, treatment with a topical, as well as a systemic, antimicrobial, is required.

Signs and symptoms of a critically colonised and clinically infected wound are listed in Figure 4.


**Figure 3:** The infection continuum in wound care

**Figure 4:** Signs and symptoms of a critically colonised and clinically infected wound

Due to the increased risk of resistant microflora developing in the wound with the use of topical antibiotics, generally, the use of topical antibiotics is not
advised in wound management. For this reason, there is renewed interest in the use of certain liquid antiseptics. Liquids such as acetic acid, iodine, and chlorhexidine, are only advocated for use in wound management at low concentrations. They should not be used as dressings in their liquid form, as they are quickly inactivated by proteins within the wound. However, they can be used as an irrigating solution in clinically infected wounds, which should be followed by the use of an antimicrobial dressing.

The development of infection in Stage 1 and 2 pressure ulcers, which are partial-thickness wounds, remains low. However, the probability of infection in Stage 3 and 4 pressure ulcers, which are full-thickness wounds, is relatively common.

The question needs to be asked: why are Stage 3 and 4 pressure ulcers more likely to result in clinical infection? The reason is that Stage 3 and 4 pressure ulcers are classified as full-thickness injuries that can very often involve muscle, joint capsules, or bone. Exposure of these structures in a wound increases the risk for invasion by pathogenic micro-organisms. Also, a large proportion of

Table III highlights some of the commonly used antimicrobials in wound care

<table>
<thead>
<tr>
<th>Antiseptic</th>
<th>Formulations</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic acid</td>
<td>Solution</td>
<td>Considered for its effect against <em>Pseudomonas aeruginosa</em>. Consider protecting periwound skin during use.</td>
</tr>
<tr>
<td>Chlorhexidine</td>
<td>Solution, powder, impregnated dressings</td>
<td>May be used as an alternative in patients who are allergic to iodine preparations</td>
</tr>
<tr>
<td>Honey</td>
<td>Available for direct application, impregnated dressings</td>
<td>Antimicrobial effects have been attributed to some components and physical properties. However, composition (and hence antimicrobial activity) is highly variable, making comparison of clinical trials difficult.</td>
</tr>
<tr>
<td>Hydrogen peroxide</td>
<td>Solution, cream</td>
<td>Caution is advised when using the solution because causes of gas embolism have been described.</td>
</tr>
<tr>
<td>Iodine</td>
<td>PVP-1, solution, cream, ointment, spray, impregnated dressings, cadexomer iodone: paste, ointment, powder, impregnated dressings</td>
<td>Modern products slowly release relatively low levels of iodine, reducing the likelihood of toxicity and staining. Povodine iodone (PVP-1) is an iodine-surfactant complex Cadexomer iodine releases iodine from highly absorbent beads.</td>
</tr>
<tr>
<td>Polyhexamethyl biguanide (PHMB)</td>
<td>Solution, impregnated dressings</td>
<td>Also known as polyhexanide and polyaminopropyl buguanide. Related to chlorhexidine. Currently used mainly for burns.</td>
</tr>
<tr>
<td>Silver</td>
<td>Silver sulfadiazine: cream, impregnated dressings Ionic silver: impregnated dressings, nanocrystalline silver</td>
<td>Available in several forms, including silver sulfadiazine (silver-antibiotic combination). More recently, dressings have become available that release charged silver atoms (ionic silver- Ag⁺) on contact with wound fluid. The amount or rate of ionic silver release from different dressings is variable. Initial release of high levels, followed by sustained release, appears to aid reduction in bacterial numbers, and a wide spectrum of activity. Staining of the wound bed or surrounding skin by ionic silver dressings may occur occasionally, and is usually reversible.</td>
</tr>
<tr>
<td>Sodium hypochlorite</td>
<td>Solution, impregnated dressings</td>
<td>Not usually recommended, unless suitable alternative are unavailable.</td>
</tr>
</tbody>
</table>

*a polyvinylpyrrolidone iodine
pressure ulcers are located in the sacral area, which poses a high risk for faecal contamination. Faecal matter has a high concentration of bacteria, and when it comes into contact with a wound, the result is often a high bacterial burden developing within it.

**Exudate management**

There are many reasons for an increase in exudate in chronic wounds (autolysis, increased bacterial burden or an underlying pathology). Assessment of the colour and consistency of the exudate can give an indication of what is happening in the wound e.g. purulent exudate can be indicative of wound infection.

Table IV provides some exudate or wound fluid descriptors (Mulder et al).

In order for wound exudate to be managed effectively, direct, as well as indirect, management of exudate, must occur. The wound is characterised by copious exudate that requires direct management through the application of an absorptive dressing. Indirect management would be through treatment with a topical antimicrobial, as the wound will have an increase in exudate, as a result of an increased bacterial burden.

If wound exudate is only treated directly, and the underlying reason for the increase in wound exudate is not managed, regardless of what dressing type is used, management of the exudate will often prove unsuccessful.

Although wound bed preparation represents a scientific and structured approach to wound assessment and management, the acronym, TIME, combines both the cellular, as well as the clinical, aspects of wound healing. The acronym, TIME, stands for:

**T:** Tissue non-viable or deficient  
**I:** Infection or inflammation  
**M:** Moisture imbalance  
**E:** Edge of the wound, non-advancing or undermined.

Table V integrates the knowledge gained from performing an appropriate holistic wound assessment, with the clinical application of the acronym, TIME, in the management of a Stage 3 pressure ulcer.

**Conclusion**

In conclusion, advances in the management of chronic wounds have taken place rapidly. Our understanding of the different biology of the acute and chronic wound environment has led to a more appropriate and structured approach to assessment and treatment. This is especially true in the context of chronic wounds.

However, in order to take advantage of these new treatment modalities, a proper and thorough holistic wound assessment is required. Often, healthcare workers are pressured to use the latest and most advanced therapeutic wound treatments, and in the process, neglect the basics of wound assessment. Unless local barriers to healing are removed, and unless contributing systemic pathologies are identified and managed, neither the patient nor the healthcare worker will benefit from advances in chronic wound management.

**Bibliography**

2. Association for the Advancement of Wound Care; 2008.
Table V: TIME assessment of a stage 3 pressure ulcer

<table>
<thead>
<tr>
<th>Description</th>
<th>T</th>
<th>I</th>
<th>M</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yellow fibrinous slough</td>
<td>Non-healing</td>
<td>Increasing exudate</td>
<td>Friable granulating tissue</td>
<td>Non-advancing</td>
</tr>
<tr>
<td>Increasing exudate</td>
<td></td>
<td>Friable granulating tissue</td>
<td>Moderate volume of slightly seropurulent exudate</td>
<td>No undermining</td>
</tr>
<tr>
<td>Friable granulating tissue</td>
<td></td>
<td>Malodour</td>
<td></td>
<td>Previous areas of re-epithelialisation</td>
</tr>
</tbody>
</table>

The wound was irrigated with ± 200ml sterile water. The periwound areas were also cleaned, using a mild chlorhexidine soap.

**Treatment**

Maintenance debridement was carried out, using a combination of an enzymatic ointment and a hydrogel, applied in a thin layer to the wound (Intrasite®, Iruxol®, Iodosorb®).

An antimicrobial was required, as the wound exhibited signs and symptoms of being critically colonised. An antimicrobial dressing (silver impregnated foam) was also used, due to the fact that the wound was located in a high-risk area. The silver impregnated foam was used to manage the wound exudate. The dressing was windowed with a polyurethane dressing, in order to maintain the integrity of the dressing due to friction and shear, and due to the fact that the patient had faecal incontinence.

**Additional management**

The patient was a type 2 diabetic on oral medication. His blood glucose needed careful monitoring and maintenance.

The patient was not mobile, and was unable to shift his own weight, due to cervical myopathy. Therefore, the patient was placed on a pressure-relieving mattress, and a strict turning schedule (every 2-3 hours) was followed. Care was taken to ensure that the patient never lay on the wound directly. The patient had urinary and faecal incontinence, and had a Foley's catheter in situ.

The patient had a poor appetite. A dietician was called in to give advice regarding nutrition. Supplemental protein shakes were ordered.