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WOUND MANAGEMENT

The new name in wound care

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Editorial policy

WHSA seeks to publish articles related to wound healing and wound care. This may take the form of original research (clinical or laboratory work), review papers for continued professional development (CPD), case reports, product reviews and letters (scientific and editorial). The target readership is specialists allied to wound care, general practitioners and nursing and allied professions with an interest in the field.

All material submitted for publication must be submitted exclusively to WHSA. All articles will be sent for peer review. WHSA will not accept material submitted to, or published by, other journals or books. Opinions expressed by authors are their own and not necessarily those of Medpharm Publications (Pty) Ltd, the editorial staff, or any member of the editorial advisory board. The publishers of WHSA accept no responsibility for statements made by contributors or claims made by advertisers nor does the publication of advertisements constitute or imply endorsement. The content of WHSA is protected by copyright.

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For a full version of the author guidelines, please visit
www.woundhealingsa.co.za
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**Drawtex** is a 3mm thick, three-layered "sandwich" wound dressing with superb capillary action that lifts, holds and transfers exudate, slough and necrotic debris away from the wound interface to create an optimal moist environment and significantly accelerate the healing process.

This makes it ideal to treat heavily exuding wounds such as venous ulcers, pressure ulcers, cavity wounds, stoma sites, burns, amputations, external cancer wounds, fungating wounds and difficult-to-heal and non-healing wounds.

- **Drawtex** saves you time and money.
  Using Drawtex as a fast line dressing together with the correct wound care procedures can result in major savings for state hospitals, clinics and patients and even medical aid funds. Owing to its accelerated healing quality, Drawtex reduces the healing period after surgery or injury, resulting in shorter hospital stays and less clinic visits.

  Since Drawtex is easy to use, it also makes convenient home-based care possible, allowing patients to change their own wound dressings daily without having to travel to a clinic each time. The result is both time and cost savings for state hospitals, clinics and patients.

- **Drawtex** can be effectively used on:
  Wounds of all shapes and sizes including burns, amputations, postoperative wounds, venous ulcers, pressure or bed sores, cavity wounds, stoma sites, external cancer wounds, diabetic ulcers, leg prosthesis related wounds, fungating wounds, burn ulcers, difficult-to-heal and non-healing wounds and for heavily excuding wounds.

- **Drawtex** has excellent fluid handling capabilities.
  The dressing has the ability to absorb and retain exudate, keeping it away from the edges of the wound and thereby reducing the risk of maceration. The rapid wicking action of Drawtex is invaluable in transferring exudate away from a cavity or sinus and enables it to be used on many different types of wounds.

- **Drawtex** is available in a wide range of sizes.
  Drawtex comes in rolls or flat sheets and can be cut to fit wounds of different shapes and sizes. Thanks to its versatility it can easily be used either as a flat dressing or as a drain for deeper wounds.

- **Drawtex** is ideal for wound bed preparation.
  Drawtex drains the deeper invaded tissue and creates a healthy wound bed that ensures rapid healing. When applied as a wound dressing, Drawtex promotes autolysis, assisting to maintain an optimal moist environment.

- **Drawtex** is easy to use and apply.
  Either side of Drawtex can be used and the dressing can be multi-layered onto a wound as necessary.

- **Drawtex** retains it’s integrity.
  As a wound dressing, Drawtex maintains its integrity, even when completely saturated, ensuring that it remains in situ and that it can be easily removed in one piece.

- **Drawtex** is safe to use with other dressings.
  Drawtex can confidently be used with over dressings and wound contact materials as part of a wound care regime on different skin types and wounds.

---

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- Limiting cross-contamination
- Promoting a healthy wound and insertion environments

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*As part of your infection control program.
A warm welcome to delegates of the 2010 UBUNTU Wound Healing Congress. WHSA is proud to be part of this initiative by publishing a special issue for the occasion.

Seeds for the UBUNTU Congress were planted at the WUWHS International meeting in Toronto in 2008 by WHASA representatives. The concept of ‘revealing the hidden/neglected wound’ was introduced at the time as it was felt that for too long, International Congresses had concentrated on ‘first world diseases’ without any mention of the prolific and deadly occurrence of a host of wounds related to malnourishment, poor hygiene, tropical diseases and the like.

Obviously the message was heard and here we are at the first Congress to simultaneously deal with first world science, first world diseases AND those of the developing world.

Much credit goes to Liezl Naude, Hiske Smart and their international partners for bringing the dream to fruition.

This edition of WHSA comprises a compilation of selected articles for the UBUNTU Congress.

Enjoy the reading and congratulations for being part of a brand new UBUNTU global initiative!

Alan Widgerow
WHSA Editor-in-Chief
e-mail: awidgerow@adarscience.com
Complications such as surgical site infection, dehiscence, or even hematoma and seroma may progress into problems for high-risk patients. The **PREVENA™ INCISION MANAGEMENT SYSTEM** is the first and only negative pressure product designed specifically for management of incisions at risk of post-operative complications. The Prevena™ System helps draw incision edges together and may reduce the likelihood of surgical site infection and dehiscence, while stimulating perfusion, and acting as a barrier to external infectious sources. For more information, visit [www.kci-medical.com](http://www.kci-medical.com).

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Wound bed preparation: DIM before DIME

Introduction

Chronic wounds are often recalcitrant to healing and they often do not follow the expected trajectory (30% smaller in 12 weeks).1,2 They are disabling and constitute a significant burden on patients’ activities of daily living and the healthcare system. Of persons with diabetes, 2–3% develop a foot ulcer annually, while the lifetime risk of a person with diabetes developing a foot ulcer is as high as 25 percent.3 It is estimated that venous leg ulcers affect 1% of the adult population and 3.6% of people over 65 years old.4 As our society continues to age, the problem of pressure ulcers is growing. To address this burgeoning problem, this article will incorporate the wound bed preparation model into a practical clinical guide for the treatment of chronic wound (see Figure 1).5,6,7 Central to this paradigm is the importance of treating the cause and addressing patient centered concerns prior to optimising local wound care. The three important components of local care are: debridement, infection and inflammation, moisture balance (DIM). If wound bed preparation is optimised and healing is stalled, the additional E or the edge of non-healing wounds represents the potential use of advanced active therapies to stimulate healing. Remember this, DIM before DIME.8

Key component analysis

The key components and relevant questions relating to chronic wound management are:

1. **Cause(s) of the wound**: What cause(s) this wound? Is the cause treatable or correctable?

2. **Patient centered concerns**: Is pain a concern? What are the psychological factors that can influence wound healing?

3. **Local wound factors**: Think DIM. Is there a need for debridement to remove necrotic tissue? (D) Is there an undiagnosed infection or inflammatory pathology? (I) Is there too much or too little moisture? (M)

4. **DIM before DIME**: What other treatment can be provided to promote faster wound edge migration after local wound care (DIM) has been optimised?

These components form the basis for the approach to wound bed preparation and will be used as an outline for this article.

1. **Cause(s) of the wound**

   It is important to make an accurate diagnosis and correct the cause as a first step. Clinicians must optimise compression in venous disease and plantar pressure redistribution for diabetic neurotrophic foot ulcers (where infection is kept under control and tissue perfusion is adequate for healing). For pressure ulcer management, the plan of care must involve individualised pressure relief or reduction, nutritional supplementation, friction and shear prevention, moisture control from incontinence of stool and urine, and mobility enhancement including frequent turning.
Table 1: Causes of wounds

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous disease</td>
<td>High compression in the absence of arterial disease:</td>
</tr>
<tr>
<td></td>
<td>Modified compression for Ankle Brachial Pressure Indexes (ABPIs) between 0.65-0.8</td>
</tr>
<tr>
<td>Diabetic neurotrophic foot ulcers</td>
<td>Vascular supply: adequate for healing</td>
</tr>
<tr>
<td></td>
<td>Infection: Bacterial balance</td>
</tr>
<tr>
<td></td>
<td>Pressure downloading</td>
</tr>
<tr>
<td></td>
<td>Sharp surgical debridement</td>
</tr>
<tr>
<td>Pressure ulcers</td>
<td>Pressure downloading, optimal nutrition, friction and shear, moisture control, increase mobility</td>
</tr>
</tbody>
</table>

However, not all wounds can be healed. To determine the healability of a wound (Table II), clinicians must ascertain if:

1. The cause is treatable
2. The blood supply is adequate
3. The coexisting conditions or drugs may prevent healing

Table II: Determining the healability of a wound

<table>
<thead>
<tr>
<th>Wound prognosis</th>
<th>Treat the cause</th>
<th>Blood supply</th>
<th>Coexisting medical condition/drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healable</td>
<td>Yes</td>
<td>adequate</td>
<td>Not prevent healing</td>
</tr>
<tr>
<td>Maintenance</td>
<td>No*</td>
<td>adequate</td>
<td>+/- prevent healing</td>
</tr>
<tr>
<td>Non-healable</td>
<td>No</td>
<td>Usually inadequate</td>
<td>May inhibit healing</td>
</tr>
</tbody>
</table>

* Cause not treated due to lack of adherence to treatment regimen or health system factors preventing access to resources

Based on the above criteria, it is useful to categorise a wound according to its ability to heal (healability). This classification helps clinicians to establish a realistic wound prognosis and to ensure that appropriate resources are allocated. Healable wounds have several requirements starting with adequate tissue perfusion. (See Table III) The presence of decreased vasculature will increase the risk of infection. For leg and foot ulcers, if there is a palpable pulse then the blood supply is usually adequate for healing (80 mm Hg and higher). If the pulse is not palpable, Doppler examination of the Ankle Brachial Pressure Index (ABPI) is necessary to determine healability.11-16 In addition, patients with leg and foot ulcers may have a coexisting cause to be treated, medical condition (e.g. advanced cancer) or prescribed medication (e.g. immunosuppressive drugs) that would prevent normal healing despite an adequate blood supply. A maintenance wound is a wound with the ability to heal but either the patient does not consistently adhere to the treatment or the healthcare system may restrict access to appropriate resources. Non-healable wound has either inadequate vasculature, a cause that is not treatable, coexisting medical conditions or medications that prohibit the healing process.

Moist interactive healing is contraindicated in non-healable wounds. Instead, local wound care involves conservative debridement without cutting into living tissue and causing bleeding, bacterial reduction, and moisture reduction. Wounds are best treated with antiseptics if healing is not immediately achievable (uncontrolled deep infection) or where bacterial burden is more of a concern than tissue toxicity (maintenance or non-healable wounds).11-16 Topical antimicrobial and antiseptics will decrease local bacterial counts and subsequent invasion of organisms into proximal viable tissue. (See table IV)

Table III: Clinical criteria for healability

<table>
<thead>
<tr>
<th>Clinical measure</th>
<th>Cut-off values for healability</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palpable pulse</td>
<td>80 mmHg</td>
<td>Usually adequate for healing</td>
</tr>
<tr>
<td>Ankle brachial ratio</td>
<td>&gt; 0.5</td>
<td>False elevation in patients with calcified vessels</td>
</tr>
<tr>
<td>Transcutaneous oxygen tension</td>
<td>&gt; 30 mmHg</td>
<td>Expensive equipment and is labour intensive</td>
</tr>
<tr>
<td>Toe pressure</td>
<td>&gt; 55 mmHg</td>
<td>Large toe is of a small calibre without a fully developed adventitial layer to facilitate circumferential calcium deposits (always compressible)</td>
</tr>
</tbody>
</table>

Table IV: Topical antimicrobials and antiseptics

<table>
<thead>
<tr>
<th>Preferred agents</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorhexidine</td>
<td>Low toxicity</td>
</tr>
<tr>
<td>Povidone Iodine (0.9%)</td>
<td>Low toxicity Broad spectrum</td>
</tr>
<tr>
<td>Acetic acid (0.5-1%)</td>
<td>Pseudomonas</td>
</tr>
<tr>
<td>More toxicity than benefit</td>
<td>Concerns</td>
</tr>
<tr>
<td>Dyes-scarlet red, proflavine</td>
<td>High tissue toxicity Carcinogenic Select out gram positive organisms</td>
</tr>
<tr>
<td>Na-hypochlorite (Eusol®)</td>
<td>Toxic = bleach</td>
</tr>
<tr>
<td>Hydrogen peroxide</td>
<td>Action duration only few seconds with effervescence</td>
</tr>
<tr>
<td>Quaternary ammonias</td>
<td>Very high toxicity</td>
</tr>
</tbody>
</table>

2. Patient centered concerns

A chronic wound may exert a devastating effect on many activities of daily living including pain, limited physical mobility, decreased social functioning, disruption of work and leisure activity.13-16 The quality of life issues for patients with chronic wounds are multifaceted and complex (See Figure 2).

Persons with chronic wounds

Figure 2: Facets of quality of life in chronic wounds
Converging evidence invariably indicates that pain has a profound impact on patients’ quality of life and their abilities to perform activities of daily living. Patients describe wound associated pain (WAP) as all encompassing and one of the most devastating aspects of living with chronic wounds. However, the management of pain by healthcare professionals is often perceived to be of a lower priority. Healthcare providers should be cognisant of the potential but devastating effect of pain on an individual and provide emotional support as part of comprehensive wound care. Wound related pain is often under-treated with a reluctance of healthcare providers to prescribe narcotic agents and patient fears of addiction.

To mitigate pain, it is important to:

a. Engage patients by talking about their pain and their concern about debridement
b. Empathise about the impact of pain for individuals with chronic wounds
c. Educate patients by explaining procedures and how they are performed, and
d. Enlist patient participation by actively engaging them during the procedure and giving them permission to call time-outs. (Based on Keeler and Caroll’s suggestions)

The use of ‘atraumatic’ dressings was considered the most important strategy to avoid wound surface trauma and patient distress. Non-adherent layers can also be used effectively for reducing dressing adherence to the wound and preventing local damage and pain on removal. There are some reports of the successful use of topical analgesics (e.g. non-steroidal anti-inflammatory agents, opioids) to relieve pain.

3. Local wound factors

Debridement

The wound bed is optimally prepared by aggressive and regular debridement of any firm eschar or soft slough if the wound has the ability to heal. A firm eschar serves as a pro-inflammatory stimulus inhibiting healing while the slough acts a culture media for bacterial proliferation. Debridement may also promote healing by removing senescent cells that are deficient in cellular activities and biofilms that shield the bacteria colonies. Although sharp debridement is the most expeditious, this method of debridement may not always be feasible due to pain, bleeding potential, cost, and the lack of clinician expertise. Alternatively, autolytic debridement is most accepted by keeping a moist wound environment to enhance the activities of phagocytic cells and endogenous enzymes on non-viable tissues. Mechanical debridement with saline wet to dry can cause a lot of local trauma and pain. A randomised controlled trial (n = 42) compared the use of collagenase with a fabric dressing infused with Ringer’s solution-soaked particles for 21 days in patients with chronic leg ulcers. These particles bind to MMPs and bacterial cell walls assisting the removal of both of these harmful factors in healing. Wounds were evaluated weekly for the amount of eschar/slough, the area of healthy granulation and the re-epithelialised area. Sloughy materials were reduced by 19% with polyacrylate wet dressing and by 9% with collagenase during the first 14 days. Emerging technology using ultrasonic device has also been demonstrated to prepare wound beds without painful and traumatic scraping and cutting.

Infection

All chronic wounds contain bacteria and the presence of bacteria obtained from a surface swab does not define infection. In fact, the mean number of bacterial species per chronic ulcer has been found to range from 1.6 to 4.4. Critical to wound healing is whether bacterial balance is achieved (contamination or colonisation) or bacterial damage (critical colonisation or infection). The four S’s of bacteria and the skin is outlined in Figure 3.

The risk of infection is determined by the number and nature of invading bacteria as well as host resistance as outlined in the following equation:

\[
\text{Infection} = \text{Number of Organisms} \times \text{Organism virulence} \times \text{Host resistance}
\]

Host resistance is the most important factor and it refers to the host immune response to resist bacterial invasion and prevent bacterial damage. Systemically, we need an adequate blood supply to heal and a decreased or inadequate blood supply favours bacterial proliferation and damage that may prevent or delay healing. Uncontrolled oedema, smoking, poor nutrition, alcohol abuse, drugs that interfere with the immune system or immunodeficiency diseases may all delay or prevent wound healing. Infection is more prevalent in certain disease conditions. For example, individuals with diabetes have at least a 10-fold greater risk of being hospitalised for soft tissue and bone infections of the foot than those individuals without diabetes. Local factors inhibiting healing may include a large wound size, the presence of foreign bodies (prosthetic joints, a thread of gauze or a retained suture) and an untreated deeper infection such as osteomyelitis. External contamination of the wound bed by micro-organisms can occur from the environment, dressings, the patient’s secretions and hands along with the hands of healthcare provider.

By using this superficial and deep separation, the clinician can identify wounds with increased bacterial burden that may respond to topical antimicrobials and deep infection that usually requires the use of systemic antimicrobial agents. The mnemonic NERDS© and STONEES© represent the initials of the signs to categorise the two levels of bacterial damage or infection. (See table V and VI). Two or three of these signs should be sought for the diagnosis in each level. If increased exudate and odour are present, additional signs are needed to determine if the damage is superficial, deep, or both.
A plethora of antimicrobial products are available, not one product is going to be appropriate for all patients.

It is important to remember that the diagnosis of infection is made clinically with bacterial swabs used only to guide therapy and identify resistant organisms.

**Table V: The bacterial damage or infection mnemonic NERDS (© Sibbald, Ayello, Woo)**

<table>
<thead>
<tr>
<th>Letter</th>
<th>Description</th>
<th>Key information to know</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>Non-healing wound</td>
<td>• The wound is non-healing despite appropriate interventions (healable wound with the cause treated and patient centered concerns addressed)</td>
<td>• Wound size should decrease 30% after 4 weeks of appropriate treatment to heal by week 12.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Bacterial damage has caused an increased metabolic load in the chronic wound creating a pro-inflammatory wound environment that delays healing</td>
<td>• If the wound does not respond to topical antimicrobial therapy consider advice after 4 to 12 weeks to rule out an unsuspected diagnosis such as vasculitis, pyoderma gangrenosum or malignancy</td>
</tr>
<tr>
<td>E</td>
<td>Exudative wound</td>
<td>• An increase in wound exudate can be indicative of bacterial imbalance and leads to periwound maceration</td>
<td>• Increased exudate needs to trigger the clinician to assess for subtle signs of infections</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Exudate is often clear before it becomes purulent or sanguineous</td>
<td>• Protect periwound area using the LOWE© memory jogger (Liquid film forming acrylate; Ointments; Windowed dressings; External collection devices)</td>
</tr>
<tr>
<td>R</td>
<td>Red &amp; bleeding wound</td>
<td>• When the wound bed tissue is bright red with exuberant granulation tissues and bleeds easily bacterial imbalance can be suspected.</td>
<td>• Granulation tissue should be pink and firm. The exuberant granulation tissue that is loose and bleeds easily reflects bacterial damage to the forming collagen matrix and an increased vasculature of the tissue</td>
</tr>
<tr>
<td>D</td>
<td>Debris in the wound</td>
<td>• Necrotic tissue and debris in the wound is a food source for bacteria and can encourage a bacterial imbalance</td>
<td>• Necrotic tissue in the wound bed will require debridement</td>
</tr>
<tr>
<td>S</td>
<td>Smell from the wound</td>
<td>• Smell from bacterial byproducts caused by tissue necrosis associated with the inflammatory response is indicative of bacterial damage. Pseudomonas has a sweet characteristic smell /green colour and anaerobes have a putrid odour due to the breakdown of tissue.</td>
<td>• Clinicians need to differentiate the smell of bacterial damage from the odour associated with the interaction of exudate with different dressing materials particularly some hydrocolloids</td>
</tr>
</tbody>
</table>

**Table VI: The deeper infection mnemonic STONEES (© Sibbald and Ayello)**

<table>
<thead>
<tr>
<th>Letter</th>
<th>Description</th>
<th>Key information to know</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>S</td>
<td>Size is bigger</td>
<td>• An increased size from bacterial damage is due to the bacteria spreading from the surrounding tissue to the deeper compartment. This indicates that a combination of bacterial number and virulence has overwhelmed the host resistance.</td>
<td>• Size as measured by the longest length and the widest width at right angles to the longest length. Only very deep wounds need to have depth measured with a probe</td>
</tr>
<tr>
<td>T</td>
<td>Temperature increased</td>
<td>• With surrounding tissue infection there is an increased temperature. This may be performed crudely by touch with a gloved hand or by using an infrared thermometer or scanning device, there is &gt;3 degrees F difference in temperature between two mirror image sites.</td>
<td>Temperature differences can also be attributed to:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• A difference in vascular supply (decreased circulation is colder)</td>
<td>• Extensive deep tissue destruction (acute Charcot joint)</td>
</tr>
<tr>
<td>O</td>
<td>Os (probes to or exposed bone)</td>
<td>• There is a high incidence of osteomyelitis if there is exposed bone or you can probe to the bone in a person with a neurotrophic foot ulcer</td>
<td>• X-rays, bone scans and MRIs of underlying bone to confirm osteomyelitis</td>
</tr>
<tr>
<td>N</td>
<td>New areas of breakdown</td>
<td>• New satellite areas of skin breakdown that are separated from the main ulcer can be indicative of deep infection.</td>
<td>• Search for the cause of the satellite areas of breakdown and the need to correct the cause</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• It is important to remember this may also be due to the inability to correct the cause of the wound or local factor leading to persistent damage.</td>
<td>• Check for local damage and consider infection, increased exudate or other sources of trauma</td>
</tr>
<tr>
<td>E</td>
<td>Exudate Erythema and oedema</td>
<td>• Increased exudate, erythema and oedema are due to the inflammatory response. With increased bacterial burden, exudate often increases in quantity and transforms from a clear or serous texture to frank purulence and may have a haemorrhagic component. The inflammation leads to vasodilatation (erythema) and the leakage of fluid into the tissue will result in oedema.</td>
<td>• Match the absorbency of the dressing (none, low, moderate, heavy) to amount of exudate from the wound.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Assess surrounding skin to evaluate for maceration. Refer to the enabler LOWE© (Liquid film forming acrylate, Ointments; Windowed dressings; External collection devices)</td>
<td>• Assess surrounding skin to evaluate for maceration. Refer to the enabler LOWE© (Liquid film forming acrylate, Ointments; Windowed dressings; External collection devices)</td>
</tr>
<tr>
<td>S</td>
<td>Smell</td>
<td>• Bacteria that invade tissue have a “foul” odour. There is an unpleasant sweet odour from pseudomonas /gm. Negative organisms and anaerobe organisms can cause an putrid smell from the associated tissue damage.</td>
<td>• Systemic antimicrobial agents are indicated that will treat the causative organisms and devitalised tissue should be aggressively debrided in wounds with the ability to heal.</td>
</tr>
</tbody>
</table>

A plethora of antimicrobial products are available, not one product is going to be appropriate for all patients. Dressings with silver are one of the most popular choices of topical agents. Silver needs to have moisture for ionization and it is only the ionised form that is an effective antimicrobial agent (not appropriate for non-healing wounds). Clinicians need to be aware of the mechanism of moisture balance in the dressing to match appropriate characteristics with the clinical features of the wound bed.

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Wound Healing Southern Africa
Do not use topical or systemic antibacterial agents long-term without weighing the benefits and the risk. Discontinue antibacterial agents after the wound is in bacterial balance unless the patient is prone to re-infection due to local or systemic factors such as immune-compromise.

Deep tissue infection may penetrate to bone and produce osteomyelitis. Ulcers probing to bone or exposed bone in persons with diabetic foot ulcers is a reliable and valid sign of osteomyelitis. Ulcers probing to bone or exposed bone in persons with immune-compromise is prone to re-infection due to local or systemic factors such as drug agents after the wound is in bacterial balance unless the patient is not weighing the benefits and the risk. Discontinue antibacterial agents after the wound is in bacterial balance unless the patient is prone to re-infection due to local or systemic factors such as immune-compromise.

Systemic antimicrobial therapy depends on local practice. In general chronic wounds are first affected by gram positive bacteria in the first month and after that time gram negatives and anaerobes invade the tissue as well. The diagnosis of infection is made clinically and swab results are used to identify organisms and their antimicrobial sensitivities. The duration of therapy is dependent on the improvement in the benchmarked clinical parameters.

**Moisture balance**

Appropriate moisture is required to facilitate the action of growth factors, cytokines, and migration of cells including fibroblasts and keratinocytes. Moisture balance is delicate act. Excessive moisture can potentially cause damage to the surrounding skin of a wound leading to maceration and skin breakdown. Conversely, inadequate moisture in the wound environment can impede cellular activities and promote eschar formation resulting in poor wound healing. A moisture-balanced wound environment is maintained primarily by ‘modern dressings’ with occlusive, semi-occlusive, absorptive, hydrating and haemostatic characteristics, depending on the drainage of the wound bed. Choose a STAR topical agent that is:

- Not used Systemically
- Not high in Tissue toxicity,
- Not likely to induce Allergy,
- Not likely to be associated with bacterial Resistance

---

<table>
<thead>
<tr>
<th>Generic Categories</th>
<th>Local Wound Care</th>
<th>Care Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absorbent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class</td>
<td>Description / Mode of Action</td>
<td>Tissue Debridement</td>
</tr>
<tr>
<td>Foam</td>
<td>Bi-laminate opaque semi-occlusive dressings with polyurethane foam mesh inner layer and semi-occlusive outer layer.</td>
<td>+ + +</td>
</tr>
<tr>
<td>Hydrofibre</td>
<td>Non-adhesive dressing composed of sodium arboxy-methylcellulose gelling agent available in sheets or ropes.</td>
<td>+</td>
</tr>
<tr>
<td>Crystalline NaCl gauze</td>
<td>+ +</td>
<td>+</td>
</tr>
<tr>
<td>Calcium alginate</td>
<td>Non-adherent calcium alginate polysaccharide kelp derivative available in fiber or non-woven form. Sodium-calcium ion exchange between exudate and dressing promotes formation of sodium alginate gel. High content of mannuronic acid promotes gelling and high galuronic acid content promotes fiber integrity for packing.</td>
<td>+ + +</td>
</tr>
<tr>
<td>Hydrocolloid</td>
<td>Outer polyurethane foam bonded to a middle hydrocolloid gelling agent and inner adhesive layer.</td>
<td>+ +</td>
</tr>
</tbody>
</table>
Table VII: Generic categories of wound dressings

<table>
<thead>
<tr>
<th>Membrane</th>
<th>Semi-permeable (O2, CO2, gases) or synthetic polymer coated on one side with adhesive.</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. Adhesive film</td>
<td>A: Highly elastic and transparent allowing continuous inspection of wound. D: Difficult to apply; self-sticking. Reports of some increased rates of infection.</td>
</tr>
<tr>
<td>8. Non-adhesive film</td>
<td>A: Transparent allowing continuous inspection of wound; conforms to wound shape; non-self-adhesive; direct absorption of antimicrobial agents. D: Non-adherent; requires tape or secondary dressing to adhere.</td>
</tr>
</tbody>
</table>

4. DIM before DIME

It is noted that a 20 to 40% reduction in two and four weeks is likely to be a reliable predictor of healing.1,2 Sheehan3 noted a 50% reduction at week four was a good predictor for persons with diabetic foot ulcers. One measure of healing is the clinical observation of the edge of the wound. A non healing wound may have a cliff like edge between the upper epithelium and the lower granulation in comparison to a healing wound with tapered edges like the shore of a sandy beach. If the wound edge is not migrating after appropriate wound bed preparation (debridement, bacterial balance, moisture balance) and healing is stalled, then advanced therapies should be considered. The first step prior to initiating the edge effect therapies is a reassessment of the patient to rule out other causes and co-factors.4 Clinicians need to remember that wound healing is not always the primary outcome. Consider other wound related outcomes such as: reduced pain reduced bacterial load, reduced dressing changes or an improved quality of life. Several edge effect therapies support the addition of missing components: growth factors, fibroblasts or epithelial cells or matrix components.5

In conclusion

In summary, the concept of wound bed preparation includes the treatment of the whole patient before the hole in the patient (treat the cause and patient centered concerns). Local wound bed preparation includes the DIM: debridement, bacterial balance and moisture balance before DIME that includes the advanced edge effect therapies for wounds with the ability to heal.

References

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Persistence of the chronic wound – implicating biofilm

Abstract

Chronic wounds by their very nature are recalcitrant and resistant to treatment. The pervading illness and pathology associated with the particular background disease, be it venous insufficiency, diabetes or the pathology underlying pressure ulcers have in the past been used as an explanation for non-healing and chronicity in these wounds. Thus managing poor perfusions, nutrition, sugar control, avoiding repetitive pressure have been and remain priorities in the overall treatment of these chronic wounds. It is apparent however, that in many cases, even when these processes are managed well, wounds still advance to non-healing and chronicity. Of late more and more authors are looking at biofilm formation and its behaviour characteristics as a possible explanation for chronicity in many wounds.1−13

The biofilm concept

Bacteria as we traditionally know them begin as single seeds of a (planktonic) bacterium. They express proteins and structures for motility (flagella) and attachment (fimbria). They aim to seed themselves and disperse to different areas thus exposing widespread areas to their presence and toxicity. In this form they are susceptible to antibiotics, some antiseptics and the immune system. In acute wounds they are usually rapidly destroyed or inactivated by neutrophils, antibodies and common wound bed preparations. They are also usually easily identified and cultured.

In the chronic wound however, the bacterium often takes on a different form. Small numbers of these single planktonic bacteria adhere to the surface of the wound by attaching to the exposed extracellular matrix; they multiply and develop over time into microcolonies. These colonies then aggregate into larger groups known as biofilms. The biofilm bacteria are encased in an extracellular polymeric matrix (EPS) which they manufacture themselves. Within 10 hours, each single-cell planktonic bacterium has differentiated into a complex community with defences and resistance to antibiotics.1,2,4,5,6,8

As the colony begins to grow signals are sent out amongst the cells – when the cells reach a certain density, known as a quorum (much the same as a minimum no of votes/people needed to pass a resolution in a meeting = a quorum) this density is sensed by the cells and they begin to elaborate virulence factors which are a potent defence against the body’s polymorphonuclear leukocytes. This process is known as quorum sensing (QS).1,19 (Figure 1).

Once the colonies of bacteria form a biofilm, individual bacteria can separate from the biofilm structure through a process called dispersion.

Prevalence

There is little doubt about the existence of biofilm now. Biopsies of 50 chronic wounds and 16 acute wounds by James2 showed that 60% of the chronic wound beds demonstrated definite biofilm. Of the 16 acute wounds, only one showed a small patch of biofilm on the wound bed. Current estimates assume that 99.9% of the total microbial biomass on earth exists as a biofilm.2,5,11 It has been estimated by the National Institutes of Health (United States) that more than 80% of persistent bacterial infections are likely to involve biofilms.2,5,11

Figure 1: The mechanism of biofilm formation
Specific areas where biofilms are known to be problematic have included hospital and portable water supplies, medical prosthetics and catheters, veterinary medicine, and cystic fibrosis.7 Biofilms have commonly been identified on inert surfaces such as medical devices or on dead tissue such as sequestra of dead bone, but they can also form on living tissues, as in the case of endocarditis. Tissue samples have identified biofilm in patients with dental caries, periodontitis, otitis media, biliary tract infections, and bacterial prostatitis (i.e., non-device-related chronic infections).13,14,15

One of the most commonly studied biofilm entities is oral biofilm. With progressive periodontal disease the patient is advised to brush and floss to remove their plaque and thus decrease the disease-causing biofilm. In the moist environment of the mouth, mature biofilm forms within 48 hours requiring brushing (debridement) at least twice a day.

A drier environment as in an ischaemic wound bed may not allow a biofilm to fully mature for 5–7 days (less frequent debridements needed).14 Among hospitalised patients, 8–10% are susceptible to infection by opportunistic pathogenic bacteria such as *P. aeruginosa* and *S. aureus*, which are notorious for forming chronic, biofilm-based infections in their hosts. Examining the chronic wound exudates and the Pseudomonas bacteria isolated typically from patients with cystic fibrosis, Bjarnsholdt et al found distinct microcolonies – the basal structures of bacterial biofilms.7 They hypothesise that the lack of proper wound healing is at least in part caused by inefficient eradication of infecting, opportunistic pathogens. They suggest that the biofilm also offers a shielding mechanism from the phagocytic activity of the polymorphonuclear neutrophils (PMNs), the backbone of the immune response in patients with cystic fibrosis.

Gjodsbol et al15 investigated the bacterial profile of chronic venous leg ulcers and found *Staphylococcus aureus* (in 93.5% of the investigated ulcers), *Enterococcus faecalis* (71.7%), *Pseudomonas aeruginosa* (52.2%), coagulase-negative staphylococci (45.7%), Proteus species (41.3%), and anaerobic bacteria (39.1%). Bacterial infections are generally considered to be treatable by administration of antibiotics; however, this is not always true. Thus more than half of the chronic wounds investigated in this study were colonised with *P. aeruginosa*. Furthermore, the *P. aeruginosa*-infected wounds appeared significantly larger in terms of area than wounds that did not contain *P. aeruginosa*.15,16 The presence of *P. aeruginosa* also seems to delay or even prevent the healing process – is this a biofilm phenomenon? Most authors seem convinced.

Biofilms continually release planktonic ‘seeds’ of bacteria from their biofilm matrix, which can bait the immune system to mount an inflammatory response. The biofilm can harvest nutrients from the host exudate that accompanies the inflammatory response. In this way, the sacrifice of a few bacteria promotes the survival of the community through continual nutrient acquisition. Suppressing the host response with steroids (antiinflammatorizes) or physically removing the bacterial load through debridement may reduce the nutrients available to the bacteria.17

**Anti-microbial resistance**

The EPS alluded to earlier offers structural stability and protection to the bacteria.1–9,11–13 In this composite state, the bacteria resist the action of a variety of antimicrobial measures. The ability of *P. aeruginosa* to form EPS-encapsulated biofilms is thought to be one of its main survival strategies in hostile environments. The EPS may contain polysaccharides, alginate (mucoid phenotype), extracellular DNA, and other components such as proteins and lipids. Alginate enhances the three-dimensional (3D) structure of the biofilm,10 acts as a scavenger of free oxygen radicals15 prevents phagocytosis, and binds many cationic antibiotics such as aminoglycosides.18,19

**Identifying biofilm**

As previously discussed biofilm bacteria are assembled in microcolonies and not evenly distributed within the wound. The implications are that cultures from a biopsy or swab are not likely to be representative for the total bacteriological load in the wound. The sample might be lacking the correct information of the colonising organisms.

Clinically, biofilms have been observed frequently as a translucent shiny/glazed manifestation on infected and nonhealing wound surfaces, often containing slough, not responding to traditional antimicrobial therapies and showing no signs of healing.3

**Biofilm models and treatment**

A major concern in the management of nonhealing and infected wounds is the fact that bacteria within a biofilm phenotypically become more tolerant and resistant to antimicrobial therapies when compared with their planktonic counterparts.

A few publications have appeared specifically looking at isolating and treating biofilm colonies. A porcine model was used by Davis et al.3 Using this model, partial thickness wounds were inoculated with a wound isolate *Staphylococcus aureus* strain. Wounds were then treated with either one of two topical antimicrobial agents (mupirocin cream or triple antibiotic ointment) within 15 minutes to target planktonic bacteria or 48 hours after initial inoculation to target biofilm-associated wound infection. Using light microscopy, scanning electron microscopy and epifluorescence microscopy, they were able to observe biofilm-like structures in wounds after 48 hours of inoculation and occlusion. Both mupirocin cream and the triple antibiotic ointment were effective in reducing planktonic *S. aureus* but had reduced efficacy against biofilm-embedded *S. aureus*. They demonstrated that *S. aureus* formed firmly attached microcolonies and colonies of bacteria encased in an extracellular matrix on the surface of the wounds. These biofilm-like communities also demonstrated increased antimicrobial resistance when compared with their planktonic phenotype in vivo.2

A second study using a LabTekt slide model was used by Percival et al12 in an effort to determine the antimicrobial efficacy of a silver-containing Hydrofiber® (SCH) dressing on bacteria growing in a biofilm state. The efficacy of silver dressings against biofilm had not been previously demonstrated. As antimicrobial dressings containing ionic silver are increasingly being used to help manage the microbial bioburden in infected or potentially infected chronic wounds, it is desirable that they are effective in preventing and breaking down biofilm formation, as microbes prefer to exist in a biofilm. This study showed that all strains of bacteria utilised readily formed biofilms after only 3 hours growth in the LabTektslide model. Following real-time long-term visualisation studies (up to 72 hours), the SCH dressing was found to be effective in killing the tested bacteria. For bacteria growing as a biofilm phenotype, kill did not begin until after three hours. Over 90% kill was, however, achieved after a 24-hour contact time with the SCH dressing. The results showed that SCH dressings are effective at inhibiting certain biofilms and killing certain bacteria within a biofilm. The results also showed that the LabTekt biofilm model proved effective as a test for potential biofilm treatments in vitro.
Concurrent with this study was a recent study undertaken by Chaw et al. who demonstrated that silver ions have the ability to break up and disrupt biofilm structures at concentrations above 50 ppb.

The results generated in the study by Newman et al. also suggest that *S. aureus* is less susceptible to ionic silver than *P. aeruginosa* — longer silver exposure time was required to induce cell death in *S. aureus* (24 hours) compared to *P. aeruginosa*.

Ideally, agents used in biofilm treatment should be able to disrupt its structure. Traditional antibiotics are better at destroying individual bacteria than colonies as seen in biofilm. Many agents are currently being investigated for use against biofilm — these agents mark a shift in traditional antibiotic mechanistic killing of bacteria. Rather they interfere with the formation of biofilm (*lysophorin B, gallium*), the attachment to the matrix (iron scavengers deferoxamine, lactoferrin, ethylene diamine tetraacetic acid (EDTA)), degrade EPS (dispersin B, alginate, phygo depolymerases) or inhibit the QS virulence producing mechanism described above (RIP — RNA III inhibiting peptide; furanone C30).

Strict adherence to wound management (especially repeated and adequate debridement) should not be underestimated in overcoming biofilm infection. These principles together with local agents (lactoferrin and xylitol) were successfully used in patients with critical limb ischaemia (CLI). In a report of their results Wolcott and Roads showed an impressive 77% rate of healing in these difficult-to-heal groups of patients. They attributed their excellent results to strict adherence to principles of wound healing (debridement, offloading, perfusion etc) causing a decrease in matrix metalloproteinases (MMPs) and elastase and decreased exudates in the wound environment. Secondly, by targeting biofilm specifically, they felt the effects of their antibiotic and hyperbaric oxygen (HBO) therapies were markedly improved.

The hypothesis

It would appear that more and more investigators are convinced that chronicity of a wound may be related in a large part to the presence of biofilm. The eradication of this virulent phenotype of bacterium is thus becoming an imperative in the treatment of wounds. Multiple authors have looked at biofilm in the context of patients with cystic fibrosis (CF). They point out the obvious similarities with respect to the bacterial infection found in CF and chronic wounds. They propose that the conditions are kept chronic by the bacterial burden especially that related to *P. aeruginosa*, a common pathogen in CF. They propose that presence of this bacterium in the form of a biofilm and its excretion of damaging virulence factors including an efficient PMN shield, encourages bacterial persistence and may explain the extreme tolerance to antibiotics and the diminished capacity of the immune defence.

This view, in relation to chronic wound healing, is one now shared by a large number of wound care researchers.

Future work and perspectives

Successful treatment of a non-healing wound depends on identifying and targeting factors that impede the healing process. Until now, it has not been fully explained why chronic wounds have elevated MMPs or why PMNs exist in vast numbers in the chronic wound. It now seems apparent that the chronicity of the wound as manifested by the biochemistry just described may be related to the formation of biofilm. Our thinking about bacteria and its causation in infection needs to change — no longer can bacteria be considered unicellular independent pathogens, rather it is now important to understand that they form multicellular tissue-like structures with major virulent capacity and antibiotic resistance. Treatment scenarios are within reach. Lately, a number of studies have proven the concept of OS inhibitors (QSI) as a relevant antimicrobial treatment. Effective QSI blockers have been designed, but further clinical tests including their relevance in wound-healing therapy are required.

In this regard Bjarnsholt et al conclude: “It is now generally accepted that all wounds are colonised; however, we believe that it is unrealistic to keep the wound sterile. By the use of QSI-based drugs, we propose that it might be possible to affect the wound to such an extent that the host itself is able to eliminate the infecting bacteria and recreate the normal healing process”.

By intervening in the process of biofilm formation, we are once again facilitating the body’s capacity to heal and tilting the odds toward an acute wound milieu with adequate defences for healing.

References

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A new look at venous ulceration

Introduction

Chronic venous disease is a widespread problem that impacts greatly on the health care system both in terms of time and costs involved. Varicose veins are extremely common among the general population (Figure 1). Of these hundreds of thousands of patients, it is estimated that one in every hundred will suffer from venous ulceration – out of these patients many endure ongoing ulceration for periods of 1-5 years (occasionally even longer) and recurrent ulceration after healing is not uncommon. Thus the magnitude of the problem associated with chronic venous disease is apparent.

The common background pathophysiologic process responsible for the manifestation of disease is venous hypertension, which is sustained and usually irreversible. It is proposed that this hypertension results in margination of white blood cells in the blood vessels, release of inflammatory mediators by these cells, capillary leakage of fibrin and cells out of the vessels and formation of fibrin cuffs surrounding the vessels. These fibrin cuffs then restrict the passage of nutrients and oxygen to the surrounding areas with resultant tissue damage and ulceration (Figure 2).1,2

The above theory has been expounded for many years but falls short in explaining the broad spectrum of abnormalities seen associated with chronic venous insufficiency. Of late, new pathophysiologic processes, previously overlooked, have been highlighted as contributors to tissue damage and venous ulceration. These include nerve and muscle disturbances and gait changes that may significantly affect the calf muscle pump and venous pooling.3

Background pathophysiology

Irrespective of which theory one accepts as the cause of venous ulceration, the background pathology is undoubtedly that of venous hypertension. This sustained increased pressure is a result of incompetent valves failing to alleviate this pressure. However, valves do not work in isolation. Aside from the heart pump working efficiently, venous return to the heart is reliant on a competent peripheral muscle pump and a maintained pressure gradient between the venous systems.4,5

The muscle pump from calf muscle contraction functions to shift blood from the superficial venous system to the deep one via perforator veins.6 During contraction, pressure increases in the thigh veins and decreases in the leg veins. The valve system plays an important role in maintaining the blood in the thigh area preventing reflux of blood into the superficial leg veins which would cause venous blood build up and resultant hypertension. As the pressure decreases in the deep veins as blood returns to the heart, the valves relax and re-open.4,5

It is evident that in the sequence of events outlined above, a number of factors could contribute to venous hypertension and its complications. This is extremely important when considering treatment alternatives for chronic venous insufficiency. Analysing the different components, Raju et al named the possible contributing factors as follows: poor calf muscle pump; reflux; increased arterial inflow; reduced venous capacitance, poor ejection fraction and a combination of the above.7
Accepted treatment modalities

Compression therapy for chronic venous insufficiency (CVI) is the accepted norm for managing this disease. The dressing type and ancillary therapies are minor additions in comparison to graded compression when considering efficacious treatment forms. Compression is applied with the purpose of providing graded pressure increases from the ankle upwards (Figure 3). Many reviews and randomised studies have confirmed the effectiveness of multilayer compression and long term stockings in confirmed uncomplicated venous leg ulcers.8,9

- Medications such as aspirin, pentoxyfylline10
- Autologous skin grafting11
- Biologic skin equivalents12
- Venous surgery10

Although the treatments noted above are accepted and standardised, there are still differences in response to these treatments. Various series have attempted to anticipate responses to treatment.13-15

Summarising these papers, it would appear that significant negative prognostic factors relate to the size of the ulcer at presentation and the length of time that the ulcer was present prior to consultation. An ulcer greater than 5 cm in diameter, present for longer than six months prior to presentation, would be expected to heal after 24 weeks of compression in only 13% of cases. Conversely 93% of cases with smaller sized ulcers present for less than six months would be expected to heal.15

Peripheral neuropathy in CVI

It is commonly accepted that peripheral neuropathy is part and parcel of the diabetic foot, contributing to the pathology and healing problems seen with this condition. What is less recognised is that peripheral neuropathy may be an integral part of the CVI pathological process too. Microangiopathy causes ischaemic polineuropathy mainly due to hypoxic tissue and nerve damage.16 CVI may lead to microangiopathy and may cause extensive tissue damage and ulceration.17

Reinhardt et al18 studied peripheral nerve function in patients with CVI because such patients describe symptoms similar to those of patients with peripheral neuropathies: cramps in 79%, dysaesthesia in 38%, painful sensations in 37% and paraesthesiae in 37%. If CVI leads to peripheral nerve damage, a neurotrophic disturbance might contribute to the pathogenesis of venous ulceration, much like that seen in diabetic ulceration. Of the 30 patients with CVI examined in this series, approximately one third showed signs of peripheral neuropathy.18 Nerve studies showed prolongation of distal motor latency, diminished vibration perception and lessened warm and cold perception thresholds. These findings clearly demonstrated disturbances of myelinated A-delta fibres and unmyelinated thermoafferent-C fibres.

Guest et al19 confirmed the findings of abnormal peripheral nerves using immunohistochemistry. Punch biopsies (4 mm) were taken from sites adjacent to venous ulcers: (1) from nonhealing ulcers (n = 15), (2) from healing ulcers (n = 14), and (3) from control patients with varicose veins (n = 12). Total nerve fibres were decreased in the dermis adjacent to healing ulcers but not in nonhealing group. The total nerve fibres of the epidermis were decreased in the non-healing group but not in the healing group. Nerve fibres were shorter in the dermis in both groups but more in the non-healer. The epidermal fibres were shorter in both groups. Reduced dermal and epidermal innervations may contribute to the initial development of a venous ulcer. The decrease in epidermal fibres in chronic ulcers suggests
The research suggests that epidermal innervations may be important in healing. Thus both healing and non-healing ulcers have reduced nerve fibres in the dermis. In addition, non-healing ulcers have reduced epidermal nerves which may indicate that epidermal nerve disturbance may relate to chronic non-healing. This study confirms the clinical studies with immunohistochemical evidence of peripheral neuropathy in venous ulceration.

Aside from the vulnerability to tissue damage that results from nerve disturbance, other factors may also impact. Thus growth factors that are normally elaborated by nerve endings will be reduced; exaggerated pain from disturbed nerve fibres may be a real problem—a correlation has been shown between ulcer size and duration to the amount of pain and quality of life.3

Thus consideration must be given to the protection of tissue due to the neuropathy; control of pain which may be exaggerated; a possible role for therapeutic use of growth factors (once matrix metalloproteinases are controlled) which are shown to be decreased.

**Ankle mobility, range of motion and gait**

Calf pump failure, thought to be a primary cause of venous pooling, may be intimately associated with decreased ankle mobility.1 Dix et al examined ankle mobility in patients with venous disease. They found a direct correlation between the severity of the venous disease and the lack of motion at the ankle joint.20 As the venous hypertension increased, the range of ankle motion decreased.

Another important aspect examined was that of calf muscle strength, which was found to be reduced in patients with venous disease.21 Padberg et al introduced a programme of graded exercise in which was found to be improved and maintained for three patients with venous disease. After six months of exercise, muscle pump function was found to be improved and maintained for three months following the programme.22 Osteoarthritis was a further factor compounding lack of mobility and reduced calf muscle function.

Additionally patients with CVI were shown to have alterations in their gait—the distribution of foot pressure was altered, with higher midfoot and lower big toe pressures.26

Thus, taken together, there is more than a suggestion that an exercise programme, gait supervision and treatment of any concomitant osteoarthritis may be of great benefit to these patients.3

**CVI management suggestions**

In an effort to improve outcomes in difficult disease processes, it is always worth looking at new avenues of possible therapeutic intervention. When one considers that the pathophysiology of CVI and venous ulceration is multifactorial in nature, it seems logical that we should consider factors allied to the pathophysiology, other than those that are obvious. Thus consideration of pain control, calf muscle exercising, gait supervision and arthritic treatment could possibly influence outcomes positively and contribute to the holistic management of patients with CVI.

**Pain**

Pain has a protective function in nature, warning of damage, and promoting careful treatment of the affected area. However, pain can be destructive too: by heightening the cellular stress response, the autonomic, somatic and endocrine reflexes are diminished, resulting in protein breakdown, platelet aggregation, nausea, ileus and a suppressed immune system.24-27

Tissue damage or inflammation sensitises nerve endings, termed ‘nociceptors’ that transmit pain signals. Goodwin25 terms the complex mix of macrophages and lymphocytes, alongside chemical mediators, such as histamine, serotonin, bradykinin, substance P, prostaglandins and cytokines in the peripheral tissues, as an ‘inflammatory soup’. This ‘peripheral sensitisation’ decreases the firing threshold and increases the responsiveness of A delta and C nerve fibres, which conduct signals at different rates. Their respective signals are perceived differently. A delta fibres conduct rapidly and give rise to a sharp shocking pain; C fibres signal a more diffuse, constant ache.25

Once the signals reach the spinal cord they terminate in the dorsal horn, where another ‘soup’ of chemical mediators, lead to ‘central sensitisation’ of spinal neurons and the transmission of the pain signals onward to the brain. Repeated stimulation, such as from a surgical wound, can give rise to a state of ‘hyperalgesia’ where normally benign sensations can now be perceived as being painful,26 and an increased tenderness extends to a wider area around the actual tissue damage.27 Thus the pain is exaggerated and out of proportion to the stimulus. This often surprises and frustrates the wound care specialist – control of pain is important not only to alleviate these frustrations but to aid in ultimate wound healing too.

It is known that the type of wound dressing can play a role in the generation of localised pain.24 Maintaining a moist wound surface may bathe nerve endings in fluid and reduce their stimulation.24 If a wound dries out, particularly to the point where the dressing adheres to it, there will be increased sensitivity on movement and greater pain during dressing changes. This is an area where research is ongoing, and is highlighting many factors that impact on the patients’ experiences. The distinction between discomfort and pain, and what this means to any individual patient is a highly personal thing.

The first line pharmacologic options to treat chronic pain are nonopioids (e.g. acetaminophen, nonsteroidal anti-inflammatory agents), opioids, and co-analgesics. Generally speaking, the nonopioids are unlikely to provide any significant degree of pain relief in patients with neuropathy.28 Unfortunately neuropathic pain is difficult to treat – responses to opioid or co-analgesic agent will result in a 30% reduction in the pain severity rating at best, although these patients often report even this small improvement as ‘much improved’.29 Careful analgesic selection and dosage titration are required, as many patients with neuropathic pain are elderly, take multiple medications, and have numerous comorbid conditions.25,30

At present there are only five co-analgesic agents that carry US Food and Drug Administration (FDA)-approved indications for neuropathic pain. These are carbamazepine (Tegretol [Novartis]) for trigeminal neuralgia, gabapentin (*Neurontin* [Parke-Davis]) and transdermal...
Supervised and unsupervised components of the study. Orthotics exercise training logs that were completed for each session in both one hour of individualised therapy. Patients were taught the principles of calf muscle exercise and gait control.

Calf muscle exercise and gait control

Physiotherapy and graded exercise programmes have been shown to be of long term benefit to strengthen calf musculature and enhance joint mobility. The exercise programme as described by Padberg et al22 consisted of lower limb and trunk stretching and strengthening, with active gravity strengthening and resistive weights in two sessions per week. Physical training focused on leg strengthening, primarily of the calf musculature, and progressed in repetitions, sets, and weights throughout the three months. Uphill treadmill walking was included in each session of the supervised component of the intervention, to further strengthen the calf, and participants were encouraged to walk uphill while maintaining their exercise programme during the unsupervised component. Each session consisted of approximately one hour of individualised therapy. Patients were taught the principles of exercise progression, and were asked to continue the progression for an additional three months unsupervised. Participants submitted exercise training logs that were completed for each session in both supervised and unsupervised components of the study.22 Orthotists should also be involved in unloading, weight distribution, foot wear where applicable to improve on gait disturbances.

Conclusion

Although CVI is the background to venous ulceration, we need to divert our attention somewhat from the vascular pathology. From cited studies it is apparent that other areas can be considered as therapeutic approaches to venous ulceration. These ancillary approaches may not only facilitate healing, but may have a real impact on long term control and recurrent ulceration. Thus neuropathic pain control, graded exercise programmes, gait attention and osteoarthritic control may all contribute to improved outcome in the treatment of venous ulceration and CVI.

References

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Offloading the diabetic foot ulcer

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Ulcers develop on insensate feet due to trauma the patient does not feel, and it makes sense that ulcers cannot heal if mechanical trauma is ongoing.1 Andrew Boulton3 has repeatedly stated that it is not what you put on a diabetic foot ulcer that heals it but rather what you take off it. Ideally then, ulcers must be managed with rest and avoidance of all pressure. However, total non-weight bearing is rarely practical and is difficult to achieve. In the neuropathic foot, the overall aim is to redistribute plantar pressures evenly, thus avoiding areas of high pressure that will prevent or delay healing. In the neuro-ischaemic foot, the aim is to protect the vulnerable margins of the foot.2 Patients usually prefer devices that are light and easy to walk with, but in reality the most effective treatment strategy requires a device that will severely disrupt normal activity for 6–8 weeks.2 De Block and colleagues found that if a plantar foot ulcer fails to heal by approximately 8 weeks, either it is being ineffectively treated or the patient is not being compliant with the treatment regimen.1 It must always be remembered that heel raisers should be applied to the contralateral limb when using any device that raises the heel of the ulcerated limb to avoid limb length discrepancy that may result in postural insecurity and lower back pain. This article will discuss the variety of offloading options, keeping patient adherence in mind.

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Total contact cast (TCC)

The total contact cast is a close fitting plaster of Paris and/or fibreglass cast applied over minimal padding.2 This is considered the gold standard of offloading for the management of the diabetic foot.3 The total contact cast works according to the following mechanisms:

- It transfers 30% of the load from the leg directly to the cast walls
- 28% of the pressure is relieved due to the ‘void’ created under the ulcer area with soft foam in the construction of the device
- It reduces midfoot pressures by 28% and heel pressure by 49%, but increases overall heel impulse by 52% because of the increased time of loading1

The TCC should be removed every week or two. Where the extremity is severely swollen, a compression dressing should be applied for 2–5 days before reaplication of the cast.3 The use of the TCC is contraindicated in the acutely infected or ischaemic foot.4 This should not be applied by an inexperienced person due to the complications that may occur.2 The most common mistakes applying the TCC are:2

- The cast comes too high up on the leg, so that when the knee is bent the cast presses on the back of the thigh
- The cast is wrapped too tightly around the toes and borders of the foot, causing pressure lesions
- Rough fibreglass outside layer is not covered with stockinet and rubs occur on the contralateral limb
- The cast is too lightweight for heavier patients and collapses
- Fibreglass is dented by pressure from fingertips (The cast should only be handled with the flat of the hands)
- The foot is insufficiently dorsiflexed. If the cast is applied to a rigid plantar flexed foot there should be compensatory building up of the heel area of the cast.

Complications include:2

- Iatrogenic lesions
- Muscle wasting
- Osteopenia
- Leg length disparity
- Danger of fracture and the development of a Charcot foot when the cast is discontinued without careful rehabilitation
- Increased risk of falls in frail patients
- Complications arising from patients failing to care for the cast

The above listed complications are appropriate to all irremovable casts used for the management of diabetic ulcers.

Nabuurs-Franssen and colleagues reported 90% of patients without infection or ischaemia healed and 87% of infected ulcers healed within 6–8 weeks. In patients with vascular impairment 69% healed. However in patients with peripheral vascular disease (PVD) and infection only 36% healed.3

(Application of the total contact cast can be seen at http://diabeticfootonline.com/CLEAR/Clinicians.html).

Irremovable or instant total contact cast (ITCC)

This is a prefabricated pneumatic walking brace (removable cast walker (RCW) or aircast) which has been ‘locked’ onto the affected limb. (Methods of locking the cast on may be seen at http://diabeticfootonline.com/CLEAR/Clinicians.html) It is used in conjunction with a protective innersole. The advantages are that
the compliance levels are similar to those of the TCC and therefore the healing times are comparable. As the RCW is locked onto the foot, care must be taken to prevent some of the complications i.e. over inflating the bladders. The advantage is that it is relatively inexpensive and less labour intensive to use. It also does not require a skilled technician to apply a new plaster cast weekly. It is also easy to remove to examine the wound. The mean healing time in the ITCC is 5.1 weeks.3

Removable cast walker (RCW)
The removable cast walker is essentially the same as the TCC, but it is not locked on. Research has shown that patients remove the device for 72% of their daily activity on average. Although a total of 30% of the patients in the study recorded more daily activity while wearing the device, this subgroup wore the RCW only for 60% of their total daily activity. Those patients who increased their activity levels with the RCW probably did so due to a false sense of security with regards to the ulcer. This highlights the need for patient education; reduction in activity is an integral part of wound healing.

Charcot restraint orthotic walker (CROW), Patellar tendon weight relieving orthoses and/or pressure relief ankle-foot orthosis (PRAFO)
These devices transfer the weight bearing from the foot to the knee where it is transferred through the cast walls to the ground. They allow for a moderate degree of mobility while still allowing non-weight bearing status to the limb.2 The primary draw back of the CROW walker is the manufacture time and the expense. Should the limb size change due to oedema or muscle atrophy the device will no longer fit properly and cannot be used. The PRAFO is used in heel offloading. It has a washable fleeced liner with an aluminium and polypropylene adjustable frame and a non-slip walking neoprene base. The patient can wear this orthosis both lying down and walking to avoid pressure on the back of the heel.2

Healing sandals and half shoes
There are many types of healing sandals, half shoes and wedged shoes available to reduce pressure on the forefoot. Half shoes are not very well accepted by patients as they are difficult to walk in.2 When compared with TCC, ITCC and RCW they had the lowest proportion of healed wounds and slowest rates of healing with an average healing time of 61 days compared to 33.5 days in the TCC group and 50.4 days in the RCW group.3

Felted foam dressings
Padding techniques that use adhesive paddings to secure a pad around or over an ulcer on the sole of the foot have also been reported in the medical literature as being successful. This type of technique can be used in the patients’ shoes if there is adequate space in the toe box, with healing sandals or in IRCW/RCW. The technique involves two materials: orthopaedic felt (i.e. Paragon Felt) and foam (i.e. Reston foam) (These products are also available from podiatry suppliers.) The non-ulcerated skin is usually prepared with a standard skin adherent for protection and to assist in keeping the felt in place. The felt is cut to support the areas around the ulcer site taking into account the bony prominences. Additional pieces of felt can be added as needed to affect a flat plantar surface.2 The wound dressing material may be used in place of the foam over the ulcer site. Care must always be taken to ensure that the wound dressing material is not as thick as the protective felt. Foam is then cut to cover the entire dressing including the felt and ulcer site. A hypoallergenic fabric tape is then use to secure the edges of the dressing.

Healing with this offloading approach is usually less successful than with more aggressive immobilisations. Healing time was on average 79.6 days.3

Therapeutic shoes and innersoles
Therapeutic shoes and innersoles provide only a fraction of the pressure reduction at the site of ulceration provided by casts, RCW or even padded dressings or healing sandals. In randomised clinical trials of Dermagraft, only 18% of patients healed in 12 weeks in a control group that was offloaded with extra depth shoes with custom made innersoles. When they were compared to TCCs, only 32 % of subjects treated with therapeutic shoes and innersoles healed compared to 90% treated with TCCs.3

Crutches, walking sticks, Zimmer frame walkers and wheelchairs
All of these may be used as an adjunct to other pressure relieving techniques. Care must be taken when using crutches in patients with impaired joint position sense which often occurs in the patient with neuropathy. It is important to check for Romberg’s sign before dispensing crutches. This is performed by asking the patient to stand
with a narrow base of support and then close their eyes. Neuropathic patients may lose their sense of balance. If this is the case the test is positive and the patient should not be given crutches. In addition, patients with neuropathy of the hands or Dupuytren’s contracture may find hand-held crutches difficult to manage. Diabetic patients need to be monitored for nerve compression injuries to the arms. Walking sticks must be measured to determine that they are of the correct length. Wheelchairs are often unavoidable in those patients with ulcerations on both limbs. Leg rests are available which are useful to prevent oedema but care must be taken that the patient does not place the foot rest under the Achilles tendon. Occupational therapists are useful for determining the viability of using the wheelchair in the home and can help with adjusting the home and providing skills to enable the patient to perform their daily tasks.

Pressure relieving socks

Socks should be considered as part of the cushioning system. This is especially appropriate once the wound is healed and prevention of further ulceration on the same site is important. Veves and colleagues have shown that special padded socks can significantly reduce plantar pressures in patients with diabetes. Ten neuropathic subjects with plantar pressures greater than 980 kPa wore specially padded socks for 6 months. Reductions of 31.3% in peak forefoot pressures were found when the socks were new. After 3 and 6 months the socks had lost some of their efficacy and offered reductions in peak pressure of only 15.5% and 17.6% respectively. The socks were well accepted by the patients and no patients developed wounds during this time. Padded sports socks only offered 17% reduction of plantar pressures when new.1

Surgical interventions and foot ulcerations

Metatarsal head resection may be used to accelerate wound healing in the forefoot. Although pressure reduction was evident in a series of 16 cases at 6–8 weeks following surgery, it is not known whether this procedure may result in a transfer of peak pressure to other areas in the foot in the long term. This may result in higher ulceration risk in those areas. Dorsiflexion metatarsal osteotomy has been suggested as an alternative to metatarsal head resection as this procedure does not violate the metatarsal phalangeal joint (MTPJ). It elevates prominent metatarsal heads, thereby balancing the metatarsals, and redistributes weight bearing forces more evenly across the forefoot. Unfortunately no pressure data is available to confirm the theory. Offloading of the hallux is often achieved using a metatarsal phalangeal arthroplasty. Recently Achilles tendon lengthening procedures have been performed to increase the dorsiflexion range of motion. Comprehensive gait analysis has shown that the initial decrease in forefoot pressure is caused by reduced plantar flexion power during gait rather than increased range of ankle motion. The most important complication of the Achilles tendon-lengthening procedure is the development of transfer lesions to the heel. This procedure has currently fallen into disfavour as heel ulcerations are more difficult to offload and therefore heal.

Offloading the ulcerated patient in hospital

It is often forgotten that whilst the patient is hospitalised to manage the complications of foot ulceration offloading must still occur. It is important not only to protect the ulcerated area but to prevent new ulceration sites. All too often new pressure wounds develop in the insensitive foot due to misplaced pillows and lack of attention to the heels and margins of the foot. Using air mattresses, foam foot protectors (Limbo), heel guards (Allevyn, Biatain and many others) and correct skin care are all critical in the management of these patients. The patient’s family need to be educated regarding the dangers before they leave the hospital.

Conclusion

There are many ways to offload the diabetic foot ulceration but all of them require ongoing negotiation with the patient. Offloading has the tendency to reduce social interaction and productivity in the work place and at home. The patient’s perceptions of quality of life need to be constantly monitored and encouragement regarding the benefits of the offloading devices highlighted to prevent patient rebellion to the offloading measures. In highly exudating wounds the offloading devices can become malodorous which patients find humiliating. Management of this is important through providing washable coverings etc. It has been my experience that all offloading devices must be used for some time after wound healing to prevent ulceration over the same site. They may also be used to alternate with offloading innersoles and new extra depth shoes in the early stages after healing whilst the ability of the new devices in offloading is evaluated.

References

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Background

Many of the complications of diabetes are well studied but robust research documenting the cutaneous effects of the disease remains scarce. Very little literature is available on blisters in diabetes but they are often the first sign of underlying ulceration. In the neuropathic diabetic patient determining the cause of blistering can be challenging as these patients often have no recall of how the incident occurred. Blisters may be filled with either serous fluid, blood (blood blister) or pus (septic blisters).

They may be caused by:
- Excessive heat
- Excessive cold
- Friction (shear stress)
- Contact with chemicals i.e. detergent, solvents
- Allergic reactions to bites and stings
- Fungal infections
- Pressure sores

In the diabetic patient they may also occur spontaneously, so-called diabetic bullae. Typically these lesions develop most frequently over the toes, heels and occasionally on the anterior aspect of the shins. There is general consensus that it is most often adult males who are afflicted. The aetiology is unclear but it has been suggested that some patients may have a reduced threshold to frictional forces. An association with neuropathy and retinopathy has been found.

On the basis of epidermal and dermal cleavage levels there are three types of blisters:
- Spontaneous and non-scarring where there is intra-epidermal cleavage without acantholysis. These resemble blisters from burning and are several millimeters to several centimeters in size, contain serous fluid and are not surrounded by erythema
- The second type may be haemorrhagic and may heal with scarring and atrophy. The cleavage plane is below the dermo-epidermal junction.
- The third type consists of multiple non-scarring blisters on sun exposed and deeply tanned skin on the feet, legs and arms. Electron microscopy has shown the cleavage plane at the lamina lucida.

In addition bullous dermatosis may be seen in those patients with diabetic nephropathy.

When assessing a blister the following should always be ascertained before treatment:
- Is it tense or flaccid?
- What colour is it?
- What does it contain?
- What was the probable cause?

Small flaccid blisters may be wiped with an antiseptic and covered with a sterile non-adherent dressing. It is important that no adhesive is applied to the roof of the blister otherwise the blister roof will be removed together with the dressing whilst checking the progress of the lesion.

Large blisters (over 1 cm in diameter and all tense blisters) need to be drained. There is some debate regarding the best method of doing this. Whilst some of the literature suggests draining with a sterile syringe other sources feel that this is of limited value as the blister quickly reseals and refills. They suggest lancing with a scalpel, draining and applying an antimicrobial dressing. At present at our clinic such wounds are treated similarly to burns, the main objective being the prevention of infection.

To illustrate the difficulties of determining the pathology of the blisters three cases from the Multidisciplinary Diabetic Foot & Ulcer Clinic, Montana Hospital in Gauteng will be discussed.

Case history 1

Diabetic male, 60 years of age with uncontrolled glucose levels, developed blisters over the dorsum of the foot. The blisters completely covered all toes and extending 1 cm on to the planter aspect of the foot. The patient reported that the blisters occurred spontaneously.
He became aware of the situation after seeing the dog lick his foot. Treatment comprised admission for intravenous antibiotics and drainage of the blisters. Two weeks later he underwent a balloon angioplasty and stent, and a surgical debridement was done. In addition, both the third and fourth toes required amputation. He is currently receiving Cuticell® Sorbact® dressings every two days. The wounds on the dorsum of the foot have almost completely healed whilst those over the toes are still in the process of healing. The team believes that these blisters were due to a third degree burn despite the patient having no recollection of such an incident.

Case history 2

A diabetic male 60 years of age, currently with controlled s-glucose, but with a long history of hyperglycaemia and peripheral neuropathy developed a large blister over the second, third and fourth metatarsal heads. When he arrived at the clinic the blister had burst and there was evidence of a deeper blood blister beneath the superficial blister. On discussing the development of the ulcer it was found that the patient had worn new innersoles provided by an orthotist prosthetist after using a gait analysis system. A plantar metatarsal pad had been placed in the shoe to improve toe function. Unfortunately the interphalangeal joints of this patient were no longer mobile and the plantar metatarsal pad created excessive shear stress. Initially treatment consisted of regular sharp debridement of the necrosed tissue followed by applications of iruxol® and Askina® sorb. Once the base of the wound started granulating he received treatment with Contreet®.

Case history 3

A diabetic female 40 years of age, with poor glucose control, in renal failure, spontaneously developed large blisters over the second, third and fourth metatarsal heads. On discussing the development of the ulcer it was found that the patient had worn new innersoles provided by an orthotist prosthetist after using a gait analysis system. A plantar metatarsal pad had been placed in the shoe to improve toe function. Unfortunately the interphalangeal joints of this patient were no longer mobile and the plantar metatarsal pad created excessive shear stress. Initially treatment consisted of regular sharp debridement of the necrosed tissue followed by applications of iruxol® and Askina® sorb. Once the base of the wound started granulating he received treatment with Contreet®.

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Since 2002, Medika SA has concentrated on introducing and establishing honey-based formulations as wound care applications in the local market. Their commitment and perseverance have paid off, as honey is now acknowledged and respected for its healing properties in South Africa.

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Medika SA is continuously evolving and expanding their current wound care offering. They have introduced the first locally manufactured skin substitute for the treatment of burns, and are proud of the fact that they can present a quality product that is also much more cost-effective than imported equivalents.

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Wound management in burns often involves a more complex wound healing continuum that must consider healing the burned skin while ensuring a good graft take, but must also consider the impact of higher incidence of scarring that often accompanies these injuries during the maturation process. Considering the delicate nature of re-epithelialisation and skin graft take, utilising a medium that closely mimics the breathability, optimises the moisture vapour transpiration rate and inhibits granulation ingrowths is paramount to successful healing.

To meet the demands of these wound requirements, a composite membrane has been developed: Silon-TSR®. This membrane is composed of a patented silicone dressing with a polytetrafluoroethylene interpenetrating polymer network. The dressing is non-adherent, can be repositioned and clings to the wound surface. The dressing allows oxygen influx but is resistant to water, creating a moist environment to promote wound healing that allows water vapour and carbon dioxide to leave the wound. The dressing is completely transparent, which allows the surgeon to directly visualise the wound bed without having to remove it, further allowing a speedy re-epithelialisation without disruption.

The clinical use and outcomes of the Silon-TSR® material has shown significant success across a range of superficial and partial thickness wound types. In the laser resurfacing industry, ensuring a successful, quick healing time is essential to creating significant cosmetic outcomes. Barta et al found that this membrane application immediately decreases postoperative morbidity and significantly reduces the severity and duration of erythema. The use of the Silon-TSR® in experimental conditions reduced the erythema and swelling in half the time, as compared to the control group using a traditional wound care techniques. Weiss et al also found similar results that showed significantly improved rates of re-epithelialisation, decreased rates of erythema and decreased postoperative discomfort. Additionally, in vivo research with the use of delicate autologous keratinocytes, Magnusson et al found that this membrane worked well to support the facilitation of the medium on the wound bed without disruption.

In deeper dermal injuries, Dillon and Okunski found that the membrane significantly reduced pain and decreased wound integration in split thickness skin grafting procedures, as well as increasing the rate of re-epithelialisation in the donor site. In skin sloughing syndromes, Campisi showed that there was significant reduction in pain and material cost, and an increase in comfort and durability, when comparing this membrane to a conventional biosynthetic dressing material.

Further expanding the role of this unique technology, incorporation of this Silon® membrane into open cell hydrophobic foam has resulted in another application for wound care in larger skin grafting procedures. Silon Dual-Dress 50® has been shown to maintain a high absorbency (8.2 grams of water per gram of foam), while maintaining minimal (less than 20%) swelling or expansion, further providing a consistent graft contact dressing.

Due to the high durability of the Silon® outer membrane, the borders of the dressing can be stapled onto itself and the wound margin to ensure complete graft coverage. Roarabough and Blome-Eberwein found that this structure provides for a bolster effect, ensuring graft take and helping to minimise graft loss, which is essential in larger total body surface injuries that often require increased patient mobilisation to prevent further secondary complications. This material works well with other artificial skin substitutes and in conjunction with other antimicrobial wound contact systems, further facilitating effective patient outcomes and minimising difficult wound management and postoperative care.

In summary, the use of Silon® materials further helps to facilitate the continuum of wound care and provides multiple applications to meet the needs of the unique facets required by challenging wound types.

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Please type or print clearly and fax or post to above address.

Surname : __________________________________________________________________________
First Names : _______________________________________________________________________
Identity number: _____________________________________________________________________
Qualifications : _____________________________________________________________________
Occupation : ________________________________________________________________________
Postal Address : _____________________________________________________________________
___________________________________________ Code: __________________

E-mail : (please print clearly)____________________________________________________________________

Tel No (H) : (            ) ________________________ Fax: (           ) _________________________
Tel No (W) : (            )_________________________ Cell: (          ) _________________________

My choice of communication:       cellphone - sms     email     post

WHASA Region(select province by marking appropriate block)

<table>
<thead>
<tr>
<th>Eastern Cape</th>
<th>Free State</th>
<th>Gauteng</th>
<th>Kwazulu-Natal</th>
<th>Mpumalanga</th>
<th>North West</th>
<th>Western Cape</th>
</tr>
</thead>
</table>

NURSING PRACTITIONERS ONLY: SANC Reg. No________________________________________

MEDICAL PRACTITIONERS ONLY: HPCSA Reg. No _______________________________________

MEMBERSHIP:

Please register me for (select your option in the allocated blocks on left side of table):

<table>
<thead>
<tr>
<th>INDIVIDUAL MEMBERSHIP</th>
<th>1 Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>FULL MEMBER (Please supply required SANC/HPCSA Registration number)</td>
<td>R 350.00</td>
</tr>
<tr>
<td>STUDENT MEMBER (Please supply proof of student registration)</td>
<td>R 150.00</td>
</tr>
</tbody>
</table>

I the undersigned hereby declare that I am eligible for membership of the Association and have included my subscription for one year. I am prepared to associate myself with the objectives of the Wound Healing Association of Southern Africa.

_______________________________                                        _____________________
SIGNATURE                                                                                    DATE

PAYMENT DETAILS:

Please deposit the amount relevant to your selection into the WHASA bank account and include the proof of payment with your application form.

WHASA Banking Details

Bank : Standard Bank
Branch : Brooklyn
Branch Code: 011245
Account No : 012966622

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