Wound and Lymphoedema Management

**2ND EDITION** 

# Focus on Resource-limited Settings

Editor, David H. Keast, BSc, MSc, MD, CCFP, FCFP(LM)





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#### World Alliance for Wound and Lymphedema Care Editor, David H. Keast, BSc, MSc, MD, CCFP, FCFP(LM)

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#### Foreword

This is not intended to be an academic text but rather practical information for those working in resource-limited settings. The focus of each chapter follows four key ideas:

- 1. Background/Introduction What is the extent of this problem? Why is it important?
- 2. Diagnosis How is this problem diagnosed in settings that may have limited investigative resources available?
- 3. Management How is this problem managed? How are barriers to implementing best practice overcome with limited resources?
- 4. Key tips

#### Dedication

This publication is dedicated to the vision and commitment of John Macdonald (USA) and Terry Treadwell (USA) to improving wound and lymphoedema care in resource-limited settings and key founders of the World Alliance for Wound and Lymphedema Care.

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Taken in Mbarara, Uganda.

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# Introduction: What is best practice in resource-limited settings?

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#### **Evidence-based Medicine**

As evidence accumulates regarding healthcare practices, doing things "the way we have always done them" is no longer acceptable. In the past, part of the art and necessity of practice in healthcare was making decisions on the basis of tradition and, in many cases, inadequate evidence. This often led to variances in practice, inappropriate care and uncontrolled costs. (1)

In 2000 Sackett et al. described the concept of evidence-based medicine as "The conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients and involves integrating individual clinical expertise with the best available external evidence from systemic research". (2) The Centre for Evidence-based Medicine in Oxford, England, promotes evidence-based healthcare and provides resources for clinicians interested in learning more about evidenced-based practice (available at http://www.cebm.net/index.aspx?o=1001).

Multiple organisations have developed and maintained clinical practice guidelines for the prevention and management of various medical conditions. These clinical practice guidelines undergo a rigorous process in their development, including a thorough review of the literature, accessing clinical practice norms and compiling expert opinion. These guidelines are readily available on the Internet through such agencies as the National Guideline Clearinghouse<sup>™</sup> (NGC), a publicly available database of evidence-based clinical practice guidelines, available at http://www.guideline.gov, or the National Institute Health and Care Excellence (NICE) in the UK, available at www.nice.org.uk. Many wound care organisations such as the European Wound Management Association (www. ewma.org) maintain wound-specific guidelines.

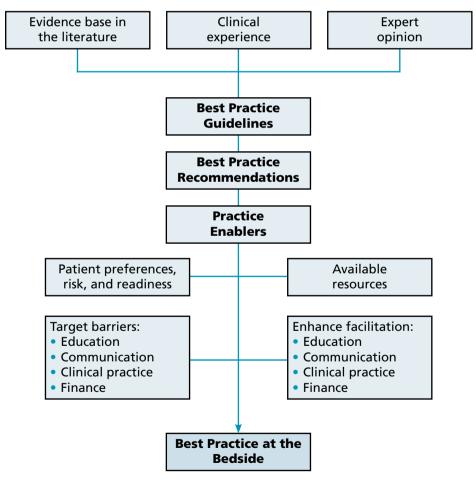
#### **Implementation of Evidence-based Medicine**

Guideline documents are often very large and implementation can be challenging, depending on the environment. Barriers may include lack of financial and other resources, inadequate or contradictory knowledge of evidence-based practice and an environment that does not support the implementation or sustainability of the guidelines. In resource-limited settings, implementation will require adapting guidelines to account for resources available, local cultural norms and availability of ongoing support. Many models for implementation of evidence-based, or best, practice have been developed. Kitson et al. (3) have developed a simple model, which has three components required for successful implementation of best practice:

- 1. Robust Evidence: Best practice requires robust scientific evidence such as that developed in clinical practice guidelines.
- 2. Context: The environment where the evidence is to be implemented needs to be prepared to receive the change in practice. Change strategies, such as assessing readiness for change, need to be employed. Barriers and facilitators for change must be identified, and strategies to reduce barriers and enhance facilitators must be sought.
- 3. Facilitation: Change is difficult in any environment. For change to be sustainable, ongoing facilitation is required. This is often the piece that is ignored. Ongoing facilitation includes the training and mentoring of local practice facilitators to avoid outsiders taking over the care and creating dependency.

Figure 1 outlines a pathway that illustrates the foundations of implementing evidence-based medicine.

Figure 1: The pathway to best practice



Adapted from Keast and Orsted (4)

This revision of *Wound and Lymphoedema Management (5)* is intended to take a practical approach to wound and lymphoedema care in resource-limited settings. Each chapter will include key practice points intended to enable best practice.

#### **Key Practice Points:**

Implementation of best practice in limited-resource settings must:

- Take into account resources and skills available
- Be culturally sensitive
- Be facilitated

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# Global Impact of Chronic Wounds and Lymphoedema

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Globally, every person will experience some type of wound in his or her lifetime. Most wounds are due to small injuries and heal quickly, with very little attention. However, many people suffer from chronic or complex wounds that can be very difficult to heal and cause severe pain and hardship. Most affected is the skin, an organ with well-understood functions that ideally should be restored. Restoration of skin function in primary healthcare can be simplified and provided at low cost without the impediment of the complex terminology of skin disease diagnosis.

The epidemiology and economic burden of chronic wounds are well documented in North America. Each year in North America between five and seven million chronic and/or complex wounds occur. (1) However, international statistics giving the full picture of the prevalence and resulting disability and impairment of wounds, burns and lymphoedema are difficult to acquire. The devastating effects of improperly treated chronic wounds can be inferred from the experience in the United States. This chapter aims to illustrate that the care of chronic wounds is a great, unmet need that requires government interventions and individual preparedness for significant improvement. Major textbooks now address these needs. (1) The chapter will discuss the epidemiology and impact of the chronic wound in the United States and in selected resource-limited nations.

The ultimate goals of wound management are the prevention of wounds, followed by the halting of wound deterioration to achieve more rapid healing, and the prevention of wound-related disability. These goals can be accomplished only by appropriate, timely, high-quality care interventions.

#### **Impact on Individuals and Families**

A wound can significantly affect a person's life. Wounds can lead to prolonged periods of disability and cause pain and discomfort. Chronic wounds often prevent a person from performing basic, everyday activities such as walking and bathing. This inactivity often leads to further co-morbidity.

Some wounds are associated with odour and excessive drainage, and require frequent attention that impedes social interactions. A non-healing wound may prevent a return to employment, which has attendant psychological and economic ramifications. Chronic leg wounds in the United States account for the estimated loss of two million workdays per year. (2) The impact of resulting loss of self-esteem, continued pain, and possible depression is difficult to quantify but is certainly real.

In addition to any loss of earnings, people may have to choose between a commitment to work and a commitment to medical management of their wound. This choice has increased significance in resource-limited nations because of lack of universally funded basic healthcare. In many cases, a disabling wound results in the loss of two or more people from the work force: the patient and the family member caring for the patient. A wound can control a life. People may have to cope with specialised devices or beds, lack of mobility, dressing changes, drainage, odour, clothing limitations, and sleep deprivation. Healing may take months or years, and unsuccessful wound treatment can lead to limb loss or even death. Sixty per cent of non-traumatic lower limb amputations are associated with diabetes. (3)

Chronic and complex wounds can lead to complications such as infection, pain, and limb amputation. The psychological problems that such patients and their families acquire are today better managed because of greater understanding of their needs, as a consequence of quality-of-life studies. Patients affected by these types of wounds often require assistance in performing common daily tasks. Neglect can lead to malnutrition, further morbidity, and, as with the diabetic foot, higher mortality rates. (4, 5) Patients need access to the best standard of care available to heal their wounds, prevent complications, and restore quality of life.

The management of burns has greatly advanced in the past few decades. Where medical gold standards are practised at the tertiary level, management of skin loss gives those affected a better chance of survival. As described in Africa by Stanley et al., "The major emphasis should be on identifying risk factors and preventing the burn happening in the first place. Education should be aimed specifically at children and the elderly. Messages can be delivered through schools, radio, television and newspapers. Teachers, nurses and members of religious communities can all play an important role." (6)

#### **Impact on Healthcare Systems**

The treatment of chronic and complex wounds is a significant burden on healthcare systems and on the economy as a whole. In 2003, in the United States, over US\$ 1.7 billion was spent on specialty dressings, devices, and topical treatments for chronic wounds. The annual cost for overall management of these wounds is greater than US\$ 20 billion, not including the additional costs to society in terms of lost workdays or productivity. (7, 8)

#### **Medical-legal Impact**

Chronic wounds have medical-legal implications. Increasingly, this has become a major problem affecting both resource-rich and resource-limited countries. (9) A library of "grey" literature relating to litigation from medical defence societies has developed in Europe and North America relating to wound healing. In most parts of the world, the law pertains only to orthodox or modern medicine. The majority of people with chronic conditions often use traditional medicine before seeking orthodox or modern medical treatment. (10) Traditional Chinese medicine practitioners and Indian herbalists sometimes are required by law to practise with single active principles and consistent dosage. Required investiga-

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tions are ever more stringent, approaching advanced biomedicine. For example, bonesetters practising without X-rays face greater liability in an increasingly technological society.

#### **Wound Prevention**

Landmine injuries head the list of demands for desirable legislation to effect accident prevention. (11)

India registers the highest number of road accidents in the world; the number of accidents per 1000 vehicles is as high as 35, most caused by human error. (12) This is a consequence of vehicle numbers and improved roads, without improved driver training. As the economy improves in every developing country the situation will magnify. There must be stringent enforcement of a highway code, wearing of helmets, separate lanes for heavy vehicles, and speed-control measures.

In China, burns are becoming a major cause of morbidity, with large social and economic implications. In contrast, burns are far less common in the United Kingdom than a few decades ago. The difference is due to government legislation. Occupational safety regulations, non-flammable clothing and furniture, and building standards are controlling factors. Fireworks restrictions are important. The accumulation of litter in public buildings with minimal control on smoking is another factor.

Pressure ulcers/injuries have received significantly more publicity in recent years since the Department of Health and Human Services in the United States introduced a National Pressure Injury Advisory Panel. (13) This was followed by a European Pressure Ulcer Advisory Panel. (14) These advisory bodies provide substantial advocacy for prevention. Patient, nurse, and physician awareness has improved. A number of governments have legislated to improve bedding quality. In New South Wales, Australia, no long-term care home is permitted to use a mattress less than 15 cm thick. This followed a comprehensive public health initiative (15) that produced usable guidelines. It is well documented that in countries where families in large numbers look after the bedridden, pressure ulceration is less prevalent. However, the increasing trend to one-child families and the reduced number of carers in the home pose a major threat to the enlarging elderly population. (16)

Malnutrition is common in the rural developing world, resulting in delayed wound healing in those who have vitamin A deficiency and where pellagra is common, especially in alcoholics. This contributes to healing delay and, with associated niacin deficiency, causes defects in the barrier function of the skin. (17)

#### Wound Type, Incidence, and Prevalence in the United States

Countless wounds occur each year, but chronic wounds require the most skill, time, and resources to heal. The Wound Healing Society (WHS) defines a chronic wound as one that has "failed to proceed through an orderly and timely repair process to produce anatomic and functional integrity". (18) Such wounds involve damage to underlying tissue and structures as well as to the integrity of the skin itself. The most common types of chronic wounds are leg ulcers, pressure ulcers, and diabetic foot ulcers. Underlying medical conditions often cause wounds to

become chronic. Older adults are more likely to develop chronic wounds. With ageing, the protective layers of the skin diminish, placing the patient at greater risk of injury. As the population in the United States ages, the annual incidence of chronic wounds is expected to rise significantly. (19)

#### Venous leg ulcers

Venous leg ulcers are the most common type of chronic wound, with an incidence of 2.5 million each year. (20) There have been several reviews of the epidemiology of venous insufficiency in the developing world. (21–23) In black skin, early venous insufficiency presenting as varicosities is more difficult to see. This is important because such insufficiencies are common in the lymphoedema population, and it can be a principal cause of leg swelling as well as of ulceration (see Chapter 10).

#### **Pressure ulcers (injuries)**

Over 2 million pressure ulcers occur each year in the United States. One third of patients admitted to a critical care unit develop a pressure ulcer. Approximately 15% of hospitalised patients aged 65 or older develop a pressure ulcer during a five-day stay or longer. (24–26) In Zimbabwe, the treatment of burns and pressure ulcers with plastic surgery is more likely to experience graft failure, and pressure ulcers are more common in people with AIDS. (27)

#### **Diabetes mellitus**

According to the American Diabetes Association, 20.8 million children and adults, or 7% of the population of the United States, have diabetes. Of significant concern is the higher incidence of diabetes in specific ethnic or age groups: age 20 years or older, 9.6%; age 60 or older, 20.9%; non-Hispanic whites, 8.7%; Hispanic Americans, 9.5%; Native Americans, 12.8%; and Afro-Americans, 13.3%. Individuals with diabetes are especially prone to foot ulcerations and chronic wounds that are difficult to heal. Diabetic foot ulcers affect approximately 15% of the diabetic population (28) and account for more than 82 000 amputations annually. (29) The statistics listed above do not include the incidence of complex wounds such as non-healing surgical wounds and burns.

Diabetes-related lower extremity chronic wounds are the most likely subject for epidemiological reporting in resource-limited countries. While the statistics are difficult to detail, certain generalizations can be inferred. For example, it is estimated that approximately 15% of the more than 150 million people with diabetes worldwide will at some stage develop diabetic foot ulceration. This situation is worsening as diabetes becomes an emerging epidemic. Foot problems are ubiquitous; all parts of the world report the development of foot lesions as a consequence mainly of neuropathy and peripheral vascular disease. The prevalence of active foot ulceration varies from approximately 1% in certain European and North American studies to more than 11% in reports from some African countries. (30) Compounding the problem is the fact that diabetes may not be treated because of insulin expense. In such cases, neuropathy and foot ulcers accelerate, and with poor foot care, the rate of amputation increases. Chronic wounds are a global epidemic. With wound care education intervention, it seems apparent that a new era is about to revolutionise global wound care. This will be a vitally important WHO initiative.

#### **Key Practice Points:**

- Wounds can have a significant impact on individuals, families, societies, and healthcare systems.
- Landmine injuries, road accidents, burns, and pressure injuries are key areas for prevention activities.
- Malnutrition has a negative impact on wound healing.
- Venous leg ulcers are the most common type of chronic wound.
- Diabetes-related foot ulcers are a growing problem worldwide.

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### **Wound Healing in Limited-resource Settings**

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Limited-resource settings are likely to be rural and located far away from teams of experts in advanced tertiary health centres. According to *Global Evidence on Inequities in Rural Health Protection*, published in April 2015, (1) 70% of the world's poorest people live in rural areas, where a majority do not access effective healthcare. The report makes depressing reading. In these areas, health worker shortages are extreme, in part because recruitment can be difficult as relatively few are able to leave the security of urban academic centres. Upgrading the medical practice of local inhabitants is possible but difficult as it requires incorporating government and funding agents to address the particular needs of the region.

Strong data will be required to counterbalance competing demands in any decision-making. The report emphasises that data about the rural-urban divide are weak and fragmented. Collecting data is time-consuming, but shortcuts can be taught. In the field of wound care in rural locations the author believes that education on accident prevention and first aid provide one of the most valuable shortcuts to gaining much-needed community involvement in healthcare provision. Organisations such as St John Ambulance, Die Johanniter-Unfall-Hilfe, and the Red Cross have great skills and teaching manuals for delivering health education to rural areas. Much can be learned from the way they have approached education in rural communities.

These organisations take with them medical experts and a well-filled medicine chest, but very soon they meet traditional health practice, whose practitioners will continue to be first in attendance of the wounded long after biomedical advances have been brought to primary healthcare centres.

#### **Causes of Wounds**

While road accidents are a significant cause of wounds in some limited-resource areas, armed conflicts and terrorist activities contribute as well. The dangers of land mines and damaged buildings persist long after the promoters of turmoil have departed.

Wounds result from carelessness in agricultural practices. The frailty of the very young and the elderly often contributes to wounds in the home.

It follows that what is most needed is a program of accident prevention and first aid training. This means that a well-informed ministry of health can supplement in support of repair and regeneration, disability impairment, handicap, and palliative care.

#### **Focus on Prevention and First Aid**

Education of communities to manage where and when wounds are caused includes very basic tenets such as: not getting oneself killed; not smoking in the presence of spilled inflammables; the ABC of airway, breathing and circulation; and the recovery position or how to manage a spinal injury on rough terrain, such as a mountainside.

Having accident prevention and first aid on the agenda creates a caring society, often with common sense, that can take up other low-cost self-help interventions, improve quality of life and increase participation. Most first aid manuals have pioneered effective teaching to communities, and for more than two centuries small communities have learned simple truths on how to care more effectively with minimal, inexpensive equipment.

#### **Treating Wounds**

The term *wound healing* embraces all types of wounds, burns, and ulcerations. Complete wound healing includes the level of restoration of function hardly ever achieved by those disfigured by wounds. There are numerous textbooks and journals focusing on wound management, and the future of wound healing in resource-limited locations will depend on making the knowledge they contain available beyond its usual readership.

Patients should be treated in their homes or as close to them as possible. To illustrate this point, consider snake bites. The future scenario may be a photo taken with a mobile phone that is transmitted to a distant anti-venom centre for identification and, if necessary, transfer of the appropriate anti-venom. In the meantime, the patient is immobilised in their home to prevent lymphatic spread of the venom and strongly reassured in a way that conforms to cultural beliefs as to why, when, and where the snake bite occurred.

This scenario replaces the current one of contact with a traditional health practitioner who delays best practice by time taken in exorcising the cause and scarifying the wound, followed by a thorough shakeup of the lymphatic system on a bumpy route to a health centre or hospital.

Another scenario familiar to experts visiting small peripheral hospitals is a ward full of the wounded with evident delay in healing of many of the wounds. Regardless of the initial cause, delay will occur when the following are not well managed: (2)

- Systemic illness such as anaemia, diabetes or HIV/AIDs, and an excess of steroid therapy
- Foreign bodies such as necrotic tissue, haematomas, pockets of pus, fragments of bone, all of which require debridement
- Unresolved tissue oedema
- Bacterial infections, an unclean wound base and too early closure of wounds that are so affected
- Trauma from patient interference, or pressure requiring offloading

To these can be added clinically significant arterial disease, maintenance of optimal temperature and pH, adequate nutrition, and the requirement to stop smoking.

#### **Supplies and Equipment**

It has only been a decade or two that wound healing has been well managed even in resource-rich countries. This has been aided by the interest of the pharmaceutical industry in the manufacture of dressings and devices and in their support of organisations providing training. This is gradually being extended to resource-limited countries.

One consequence of this collaboration has been the rising cost of interventions. One role for experts is to monitor the availability of cheap but effective wound dressings and devices that might be added to government provision through essential drug and dressings lists. As John Macdonald described (personal communication) after the earthquake in Haiti, centralisation of supplies determines the adequacy of provisions, reduces waste, and does not delay distribution to those in need.

Water is an example of what should be a low-cost, readily available agent for wound care. For cleansing purposes, it should be fit for drinking. (3)

Brookes describes how a small hospital short of local water used bottles of Ringer's solution for washing wounds. By using a humanitarian gift of a water purifier she was able to reduce the considerable wound care costs. (4) Where soaking is required, wet wrapping may reduce waste by spillage.

In Kenya, in one study large gaps in essential emergency care were identified, including anaesthetic provision and pain relieving drugs. Burke et al. appealed for "Advanced emergency care packages and pre-hospital care transportation planned from a designated facility". (5) Even in the last decade, I have visited hospitals in India treating several traffic accident victims each night that had run out of needles, sutures, and gloves. In Sierra Leone, I have observed surgeons operating on perforations of the intestine from typhoid where there were no anaesthetists, but ketamine was administered by untrained recruits volunteering from a local town. In light of these observations, there is a definite need for more research, education, and application of best practice.

#### **Overcoming Barriers to Best Practice**

Barriers to the implementation of best practice in wound care are many in resource-rich countries. Factors such as preference for personal experience and traditional knowledge and practice and difficulty in assimilating too much information are far worse in resource-limited locations. (6)

With some of these problems in mind The International Society of Dermatology created a taskforce called Skin Care for All: Community Dermatology (www.skincareforall.org). (7) It aims to tackle, especially in resource-limited countries, the need for all who care for the skin, such as doctors, nurses, podiatrists, and many other professions, to meet basic education and skills required to manage wounds, burns, lymphoedema, and neglected tropical diseases such as leprosy, lymphatic filariasis, and Buruli ulcer. It aims to support management of the skin functions of barrier, thermoregulation, sensation, and communication. In India, it has begun a policy of education of family practitioners on skin hygiene and wound healing, while in China it is encouraging hospitals that treat burns to add wound healing units and to develop an approach to other causes of absent skin such as managing the epidemic of obesity and diabetic foot ulcers as well as pressure ulcers/injuries in the elderly. The concept behind this is that the skills of managing skin regeneration and rehabilitation of the severely burned are applicable to all presentations of absent skin. To ensure sustainability, city units can educate town units and town units can educate village units. Minority ethnic groups and tribal communities can share in such benefits if well planned. Both China and India have strong alliances with other systems of medicine, and integrated medicine has government support.

Traditional healthcare has contributions to make. It often provides both technical skills and a helpful attitude to patient care. By organising traditional health practitioner associations, dangerous practices can be eliminated, and some basic skills can be added to the practice to this very large human resource. (8)

In the first edition of *Wound and Lymphoedema Management (2)*—which, judging from its presence on Internet searches currently has few rivals—there is a call for simple, low-cost interventions in resource-limited locations. A good start is to aim at prevention and to teach simple interventions as well as priorities, which, like those in first aid manuals, are easy to understand and put into practice. Because very often in resource-limited locations the first on call are traditional healthcare systems; those planning interventions must seek collaboration and education of all involved in care.

Wound management is a discipline that is a good start to community healthcare practice. (9, 10) When added to a regional dermatology centre in Tanzania it created more than 260 dermatologists with appropriate skills for 14 sub-Saharan English-speaking countries. When added to burn units in Beijing and Shanghai it has resulted in a better public understanding of two great epidemics: obesity with diabetic foot ulcers and pressure ulcers in the frail elderly.

#### **Key Practice Points:**

- Focus on prevention and first aid.
- Advocate for the availability of cheap but effective dressings and devices through each government's essential drug and dressings lists.
- Provide education to local traditional practitioners and incorporate them into prevention, first aid, and wound management systems.
- Provide skin hygiene education to primary caregivers and encourage the development of wound healing units in hospitals.

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## The Wound Prevention and Management Cycle: An Approach to Wound Care

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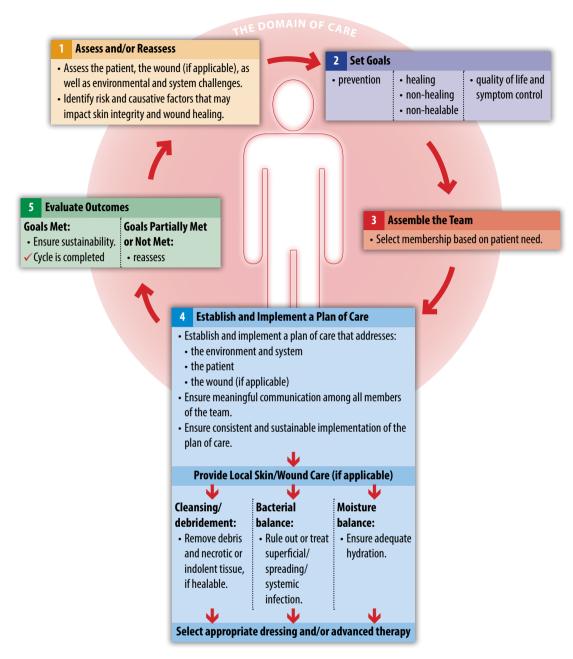
#### Introduction

Dr. Keith Harding is often guoted as saying that the problem with chronic wounds is that they are not a disease but the complication of disease. This makes the assessment and management of chronic wounds a complex process. In a hospital-based wound management clinic in Canada, a review of 326 lymphoedema patients showed that the patients had an average of 7.3 co-morbid conditions. (1) In 2000 the Canadian Association of Wound Care (now Wounds Canada) published an approach to wound bed preparation which provided a systematic model for chronic wound management. (2) The model proposed three key activities: assessing and treating the underlying cause, identifying and managing patient-centred concerns, and treating the wound through debridement of non-viable tissue, managing bioburden, and managing moisture balance through appropriate dressing selection. Since that time there have been several adaptations of this model, most notably the TIME concept. (3) The difficulty with these documents is that they focused primarily on the wound and paid little or no attention to patient and environmental factors or to prevention strategies. In 2017 Wounds Canada (www.woundscanada.ca) published a revised Best Practice Recommendations for the Prevention and Management of Wounds, (4) which includes the Wound Prevention and Management Cycle (Figure 1). This cycle provides a clear pathway through assessment and management which is adaptable to limited resource settings.

#### The Wound Prevention and Management Cycle

The Wound Prevention and Management Cycle contains five key steps which take the clinician through assessment to reassessment. The full document is free to download from the Wounds Canada website (https://www.woundscanada.ca/docman/public/health-care-professional/bpr-workshop/165-wc-bpr-prevention-and-management-of-wounds/file). The key statements in each step are summarised in Table 1: The Wound Prevention and Management Quick Reference Guide. In the full text the table also has levels of evidence for each step.

#### Figure 1: The Wound Prevention and Management Cycle (4)



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Table 1: Wound Prevention and Management Quick Reference Guide (4)

Step		Recommendation
1	Assess and/or Reassess	<ul> <li>1.1 Select and use validated patient assessment tools.</li> <li>1.2 Identify risk and causative factors that may impact skin integrity and wound healing.</li> <li>1.2.1 Patient: Physical, emotional and lifestyle</li> <li>1.2.2 Environmental: Socio-economic, care setting, potential for self-management</li> <li>1.2.3 Systems: Healthcare support and communication</li> <li>1.3 Complete a wound assessment, if applicable.</li> </ul>
2	Set Goals	<ul> <li>2.1 Set goals for prevention, healing, non-healing and non-healable wounds.</li> <li>2.1.1 Identify goals based on prevention or healability of wounds.</li> <li>2.1.2 Identify quality-of-life and symptom-control goals.</li> </ul>
3	Assemble the Team	<ul> <li>3.1 Identify appropriate healthcare professionals and service providers.</li> <li>3.2 Enlist the patient and their family and caregivers as part of the team.</li> <li>3.3 Ensure organisational and system support.</li> </ul>
4	Establish and Implement a Plan of Care	<ul> <li>4.1 Identify and implement an evidence-informed plan to correct the causes or co-factors that affect skin integrity, including patient needs (physical, emotional and social), the wound (if applicable) and environmental/system challenges.</li> <li>4.2 Optimise the local wound environment aided through 4.2.1 Cleansing 4.2.2 Debriding 4.2.3 Managing bacterial balance 4.2.4 Managing moisture balance</li> <li>4.3 Select the appropriate dressings and/or advanced therapy.</li> <li>4.4 Engage the team to ensure consistent implementation of the plan of care.</li> </ul>
5	Evaluate Outcomes	<ul> <li>5.1 Determine if the outcomes have met the goals of care.</li> <li>5.2 Reassess patient, wound, environment and system if goals are partially met or unmet.</li> <li>5.3 Ensure sustainability to support prevention and reduce risk of recurrence.</li> </ul>

Adapted from Wounds Canada 2016. Used with permission.

#### Step 1: Assess and/or Reassess

The wound is on the patient and the patient is in their environment. Therefore, any assessment must involve all three domains of care: wound, patient, patient's environment. Before the goals of care and the implementation of the care plan can take place, patient and environmental factors, which often have a significant impact in the case of chronic wounds, must be assessed.

In resource-limited settings there may be many barriers to care. Access to potable water may be a significant issue. Living conditions, nutritional status, the availability of informal caregivers, cultural norms, and distances to travel for formal care all may have significant impact on care plans. Prevention strategies may also be determined by these and other available resources or usual practices. For example, in many settings childhood burns are a significant problem, and prevention may require a change in cooking practices.

The wound itself must be assessed with standardised measures so that progress towards meeting the goals of care can be evaluated. A more complete discussion of wound assessment can be found in the Best Practice Recommendations (4) article or in Chapter 5 of this book.

#### Step 2: Set Goals

Wounds may be classified as healing, non-healing, or non-healable. Healing wounds are those where the patient has the physical capacity to heal, the resources are available to support best practice care, and the wound is progressing along a healing trajectory. Non-healing wounds are those where the patient has the physical capacity to heal but is not healing because the appropriate resources are not available or the patient is unable or unwilling to implement the care plan, as, for example, in the case of a neuropathic diabetic foot ulcer where no offloading devices are available. Non-healable wounds are those where the patient has no physical capacity to heal, due, for example, to malignancy or lack of vascular supply. (4)

To be effective, goals should be measurable and time sensitive and be based on the healability of the wound. Goals could include sourcing offloading devices or managing pain and other symptoms in non-healable wounds.

#### Step 3: Assemble a Team

Wound management is a complex process and may require the skills of many different disciplines. It is important to remember that the key members of the team are the patient and their informal caregivers. Particularly in resource-limited settings it may not be possible for all team members to be present at one time. The concept of the "team without walls" is one strategy that may be employed to build collaborative relationships. The key to effective team collaboration is timely communication. Please see Chapter 6 for an in-depth discussion.

#### Step 4: Establish and Implement a Plan of Care

This step incorporates the original wound bed preparation model. The care plan should address the causative factors, patient factors, and system factors identified in the assessment step. Local wound care is provided through cleansing with appropriate agents, debridement to remove non-viable tissue, identification and management of bioburden, and appropriate dressing selection to support moist, interactive wound healing. Care planning also includes identifying barriers and facilitators to the implementation of the plan as well as strategies to overcome the barriers and enhance facilitators.

Cleansing remains a challenge in resource-limited settings due to lack of potable water. Antiseptic solutions may be available but must be used at concentrations that minimise tissue damage. Cleansing does mean using appropriate volumes. A table of cleansing agents can be found in Chapter 8.

Debridement is essential to remove non-viable tissue and manage bioburden. Different types of debridement are found in Table 2. Debridement requires that the practitioner have the training and skill to perform the selected mode and that it is within their scope of practice. For a more complete discussion see the Best Practice Recommendations for Prevention and Management of Wounds. (4)

Method	Description	Comments
Biological	Use of maggots that secrete digestive enzymes that specifically liquefy necrotic tissue	Can be used in most settings. Some concern about transmission of viral agents if not medical grade.
Mechanical	Use of mechanical forces to remove non-viable tissue and bacteria	Most common form is wet-to-dry dressings, but these are painful and often disturb healthy tissue and leave gauze fibres behind. Scrubbing with gauze is similarly problematic. Irrigation with safe irrigating fluids at safe irrigating pressure is acceptable.
Hydrosurgical	Pulsed lavage, ultrasound, or hydro surgical wand	Expensive. Requires power source. Difficulty with overspray.
Chemical	Use of sodium hypochlorite	Non-selective and may damage health tissue. Other methods preferred.
Autolytic	Uses body's own neutrophils, macrophages, and proteolytic enzymes	Safe and can be used in most settings. Dressings that maintain a moist environment must be used. Irrigation must be used to flush away loosened tissue. Eschar needs to be cross-hatched
Enzymatic	Application of exogenous collagenase	Faster than autolytic. Requires daily dressing changes and reapplication. Not available in all settings. Also see papaya, in Chapter 17.
Conservative sharp	Use of forceps, scalpel, scissors, or curettage to remove non-viable tissue	Requires skill, training, and mentoring and must be within scope of practice. Should not cause excessive bleeding. Need either single-use equipment or the ability to sterilise instruments.
Surgical	Use of surgical instruments to remove non-viable tissue but may extend into viable tissue	Requires specific skill and training. Usually requires analgesia. Requires ability to control bleeding and may require an operating room set-up.

Managing bioburden is discussed in more detail in Chapter 8.

Moisture balance is managed through selection of an appropriate dressing which supports moist interactive wound healing. When selecting a dressing, one needs to consider form, function, and properties of the dressing.

• Does it help to autolytically debride?

- Does it absorb exudate?
- Does it donate moisture to the wound bed?
- Does it maintain an effective barrier to external contaminants?
- Does it help control bleeding?
- Does it help manage bacterial burden?
- Is it available?

For information about the World Alliance for Wound and Lymphedema Care (WAWLC) Dressing Kit for resource-limited settings please see Chapter 18.

Successful implementation of any care plan requires all team members to be involved and, in particular, for patients and their informal caregivers to be invested in the plan.

#### Step 5: Evaluate Outcomes

On a regular basis, the prevention or wound care plan needs to be reassessed against the outcome measures established in the goals of care. Goals of care should be adjusted to account for unmet needs, followed by the implementation of a revised care plan. Prevention plans should be assessed for sustainability. Frequency of reassessment is under debate, but care plans need to be given a reasonable time to work. Every four to six weeks is usually acceptable. (5)

The Wound Prevention and Management Cycle provides a general framework to support clinical decision making. Specific information for various aetiologies is found in succeeding chapters.

#### **Key Practice Points:**

In the first edition, Dr. John Macdonald wrote:

- Enhance systemic conditions through assessment of the patient and their environment.
- Protect the wound from trauma, both physical or chemical.
- Promote a clean wound base and control infection.
- Maintain a moist wound environment.
- Control peri-wound lymphoedema/oedema.

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# Chronic Wound Assessment and Treatment System (CWATS)

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The work-up of a patient with a non-healing wound can be complicated. The differential diagnosis of a patient with a leg ulcer, for example, includes disease states ranging from common conditions to esoteric syndromes. Venous ulcers are the most common diagnosis in an outpatient clinical setting but are often complicated by the different anatomic levels of possible involvement, compounded by many potential comorbid conditions that create many variations, each with their own individual treatment plan. However, if the wound clinician adopts a systematic approach to all patients with non-healing wounds, regardless of aetiology, the work-up becomes routine, and fewer subtle signs, symptoms, and diagnoses will be overlooked.

The reality is that medical error rates and hospital-based complications are becoming more common, and the general public is more aware of treatment inconsistencies. (1) As well, tracking outcomes on both clinical and patient-centred parameters is becoming more important as healthcare resources are limited worldwide and healthcare providers are being graded and compared. It was thought that the creation of guidelines and protocols would begin to standardise the practice of medicine and minimise unnecessary practice variation and the chances of a missed diagnosis or a delay in treatment. Unfortunately, failure of guideline implementation has severely thwarted those efforts. (2)

This chapter will outline a protocol known as the Comprehensive Wound Assessment and Treatment System (CWATS). This system incorporates a concept known as the Least Common Denominator Model (LCD model), which attempts to standardise the wound care patient work-up. (3) Outcomes, using this method, have been validated in various care settings and in several publications. (4, 5) Efforts are now underway to utilise big data to assist clinicians by providing real-world outcome measures and offer predictive analytics to identify potential clinical and economic outliers earlier in the treatment plan.

#### **Comprehensive Wound Assessment and Treatment System (CWATS)**

The clinician who treats a recalcitrant wound must be skilled in communication, negotiation, and change management in order to coordinate an interprofessional team of providers focused on a unified objective. The clinician must possess an awareness and understanding of surgical concepts and treatment outcomes in plastic, orthopaedic, vascular, and general surgery in order to make appropriate and timely referrals during the continuum of care. Regardless of the clinician's primary educational background, the wound clinician must incorporate knowledge and skills from both surgical and medical fields. The clinician must also command knowledge of dermatology, rheumatology, endocrinology, and general medicine in order to integrate the management of chronic systemic disease states into local wound care therapy.

Communication with patients and family is of paramount importance. An awareness of the social, psychological, spiritual, and financial concerns that are often involved with the possibility of limb loss and/or loss of independence and functionality is required to approach the wound patient in a holistic way. Many times, a patient has already received conflicting therapeutic recommendations from other healthcare professionals, leading to frustration easily misdirected towards the current wound clinician.

Given the broad base of knowledge and skills required, coupled with the paucity of clinical experience most clinicians have in wound care, the practice of wound management is as challenging, demanding, and complex as that of any other field in medicine.

A movement towards the specialisation of wound care worldwide has arisen over the past 10 or so years. For example, the average physician in the United States receives only 9.2 hours of didactic information over a four-year medical school curriculum. (6) Wound care fellowships, graduate level education, courses and conferences have all been created to help solve this educational gap. (7) As more diverse providers begin to deliver wound care throughout the world, programs need to be developed that deliver both an agreed upon "core curriculum" that all providers would require and the degree-specific content that applies to the scope of practice determined by each provider's individual licensing body. These efforts are being spearheaded by the American College of Wound Healing and Tissue Repair. (8)

While there will always be an important role for a wound care physician, access to this type of care is limited in many parts of the world. Many programs are finding how important team care can be. Community health workers, patient navigators, and social services professionals often provide, monitor, assist, and actualise treatment plans for patients that were created by physicians and mid-level providers. One effort to bridge the knowledge gap and move knowledge instead of patients is Project ECHO. This novel approach, created by Dr. Sanjeev Arora at the University of New Mexico, is combining technology with medical expertise to diffuse knowledge to the areas it is needed most. (9) The University of Illinois at Chicago is preparing a Project ECHO for wounds that will attempt to diffuse knowledge in wound healing to areas in which access to expertise is lacking.

#### **History and Review of Systems**

Much information can be obtained by simply watching the patient walk into the clinic. Information concerning general appearance, body type, age, absence of a limb, ethnicity, and mobility can be evaluated as the provider walks into the exam room. The patient may deny pain verbally, but their body position and facial expressions may indicate otherwise. As more healthcare is being delivered via telemedicine it becomes important to use technologies that recreate the

traditional patient encounter so this valuable information is not lost. Recently, providers rounding remotely at a sub-acute wound unit were broadcast into a hotel conference room where over 400 providers could see the patient and the wound as well as interact with the providers. Wearable glass technology allows wound providers hands-free communication from the bedside to connect with experts located in remote academic settings. (10)

The patient assessment process begins with a comprehensive history and a review of systems. Wound care is not unlike other specialties in that a majority of cases can be diagnosed with a thorough history alone. Past medical history is important because many co-morbid illnesses exist in a patient with a complex wound. For example, in most patients with rheumatoid arthritis (RA) and a leg ulcer on the medial side of the leg, the ulcer will still be venous in origin due to the high prevalence rate of venous disease in patients with RA. The knowledge that the patient has RA, however, will allow the clinician to consider a vasculitic wound in the differential diagnosis. A history of coronary disease in a patient with a venous ulcer should alert the clinician to the possibility of concomitant peripheral vascular disease and a mixed arterial/venous aetiology for the leg ulcer. This has implications for the work-up, expected time to healing, and the level of therapeutic compression to be used.

The social history will address confounding variables such as alcohol use, tobacco use, illicit drug use, occupational history, and level of activities. The patient must be questioned about the presence of any allergies to medications, environmental factors, and foods. A detailed list of all medications taken by the patient is also important. With an increasing number of patients using herbals, vitamins, homeopathic preparations, and dietary supplements it is important to emphasise to the patient that all medications should be listed. It is well known that steroid use can negatively impact healing, but other common medications, such as COX-2 inhibitors, ace inhibitors, and calcium channel blockers, have been implicated in slowing angiogenesis, a necessary component for normal healing. (11) A family history will help identify risk factors and might shed light onto the patient's diagnosis, even though the patient will often not recognise the correlation between a medical condition in their family history with their own current problem.

A complete past surgical history is important. Patients with prior venous surgery are known to have wounds that are harder to heal, for example. Identifying these factors might influence a clinician to use advanced technologies earlier in the treatment regimen.

#### **Patient Expectations**

Patients and their families desire specific outcomes from a treatment program, and these should be thoroughly understood and agreed upon during the development of the care plan and before the onset of treatment. For example, an elderly female with a highly exudative venous ulcer may be most concerned with controlling the drainage or odour. Although the patient would like the wound to heal, their primary concern for exudate/odour management might explain why they become less adherent to the treatment protocol as "their" outcome goals are met. In addition, there is a growing population of patients who, by choice, terminal illness, or chronic conditions, have wounds that have reached a steady state and are not able to achieve healing or for which the treatment risks outweigh any possible clinical benefits. This group of patients might be better served with a palliative wound protocol that emphasises surrogate endpoints such as pain control, exudate and odour management, infection control, and quality of life outcomes rather than total healing. These concepts are in alignment with a patient-focused approach to wound care. (12)

Finally, a history of present illness (wound history) should be recorded. This part of the history should occur after gathering all the previously mentioned information. Timing of the ulcer, location, prior episodes, current and past treatments, pain, drainage, appearance, and alleviating factors should be recorded. The wound history can take a long time because patients often provide tremendous details about the prior treatment plans and how they responded. It is important to have already created a relationship with the patient and have some clinical insight into the overall patient condition before evaluating the actual wound. By looking at the wound initially, the clinician might use a pattern recognition reflex to diagnosis and miss subtle other findings that might be present.

#### **Physical Examination**

After the history, the clinician should begin a physical examination that includes vital signs, a pain scale score, height, weight, and a comment on general appearance. The blood pressure should be measured in both arms and legs at the initial visit in order to calculate an ankle-brachial index. A thorough exam should be conducted using appropriate documentation as described in the American Medical Association's Current Procedural Terminology (CPT) manual for evaluation and management coding. A complete exam frequently uncovers findings that aid in the final diagnosis.

Note: It is important to let the patient know that you intend to conduct a complete exam at the onset. Many patients assume they will simply have to remove their bandage and show the clinician their wound. They occasionally will become angry if much time is spent on what they perceive as "unnecessary" components to the office consultation. Frequently they do not understand how important it is to get a complete medical picture before embarking on a wound treatment program. It is useful to send the patients a welcome packet describing the entire process either to their home prior to the visit or on arrival to the waiting area. The patient should fill in information concerning prior history and medications to expedite the visit and start them thinking about their wound on a more systemic level.

#### **Wound Assessment and Documentation**

After the complete history and physical, the clinician should turn their attention to the wound itself. The examination of the wound takes into account the important issues of wound location, drainage quality and quantity, surrounding skin condition, quality of wound bed tissue (granular, fibrinous, eschar, etc.), pain, odour, condition of wound perimeter (e.g., presence of undermining), as well as standard measurements (length, width, depth).

#### Least Common Denominator (LCD)

The wound work-up next focuses on what has been termed the Least Common Denominator model (6). Composed of six subsections (tissue perfusion/oxygenation, infection, nutrition/immune status, psychosocial, pressure/neuropathy, and wound bed) this model, when followed by clinicians, ensures that all factors that affect healing in a wound of any aetiology are considered. Although initially covered in the history and physical exam, nutrition and psychosocial aspects are now revisited, as they specifically relate to the wound and the potential for healing.

#### **LCD: Perfusion-Oxygenation**

The most important aspect of the LCD model is the adequacy of tissue perfusion/ oxygenation. Tissue perfusion analysis within the paradigm of both the macro- and micro-vascular status is imperative. The initial evaluation focuses on the macro-circulation with the palpation of peripheral pulses. Ankle pulses, however, are not sufficient to detect impaired arterial circulation, and additional testing is frequently required for the patient with leg ulcerations. Performing an ankle brachial-index is useful for wound healing prediction and as an overall marker for cardiovascular health. (13)

Only about 10% of patients presenting to an outpatient wound clinic will have isolated arterial disease as an aetiology for their leg ulcer. Arterial duplex scans, segmental pressures, including toe pressures, pulse volume recordings, and Doppler wave forms scans are other non-invasive macro-circulation studies that may be ordered. An interventional angiogram can be done with a plan to intervene via an endovascular approach in many cases. At times, however, magnetic resonance angiography (MRA) or computerised tomography angiography (CTA) can be done to provide an arterial road map and allow for future planning of the best treatment option if revascularization is required.

After a complete assessment of the macro-circulation, the clinician's attention must be turned to the status of the micro-circulation. Adequacy of macro-vascular flow does not ensure healing will occur. Currently clinicians are turning to new technologies such as indocyanine green studies to assess the cutaneous circulation to determine healing capacity. (14, 15)

If a patient has a leg ulcer and abnormal macro-vascular flow studies, but physiological studies (e.g., transcutaneous oximetry) indicate adequate values for healing, then a trial of aggressive local wound care is warranted. This assumes the patient does not have severe rest pain or significant tissue loss. If, after a four-week treatment course, there is no significant improvement in either wound dimensions or quality of tissue, then further invasive studies followed by revascularization might be necessary. Approaching the patient in this manner will avoid unnecessary high-risk procedures, and the limited treatment time will minimise potential harmful outcomes. (16)

If the micro-circulatory studies are abnormal then a trial of treatments aimed at enhancing the micro-circulation (e.g., electrical stimulation, growth factors, bio-engineered tissue, and therapeutic ultrasound) could be used along with aggressive wound care for a short course. Systemic therapy along with lifestyle modifications should also be employed. (17) In general the provider should consider the named vessel that primarily supplies blood flow to the anatomical area in which the non-healing wound is located. This approach ensures that perfusion will not be overlooked—for example in the case where the wound is located on the trunk or abdomen. The venous circulation should also be evaluated when clinically relevant to the wound location. The use of the CEAP classification system will allow clinicians to describe the venous anatomy in a consistent manner. Recent guidelines have been developed to diagnose and treat venous ulcers. (18, 19)

#### **LCD: Infection**

The second component of the LCD model refers to the determination of infection. Bacteria are present in all chronic wounds. There is a natural balance between the quantity of bacteria present (bioburden) and the host's immune status. When equilibrium is reached there is no clinical infection. If the inoculum of bacteria is increased (>10<sup>5</sup> organism/gram tissue) or the host suffers a decrease in immunity, clinical infection may occur. (20) Many examples are cited in the literature describing the failure of skin grafts, delayed closures, and overall wound healing problems when the bacterial bioburden exceeds 10<sup>5</sup>. This value is accepted by many as the quantitative definition of infection except in the presence of beta-hemolytic streptococcus, where the value is somewhat lower (10<sup>3</sup>). (21)

The bacteria compete for nutrients and oxygen with host repair cells in the granulating bed. Bacterial byproducts of metabolism can be toxic to the host's normal cellular functioning. The presence of necrotic debris and foreign body, and the desiccation of the wound bed enhance bacterial growth. The contamination (the mere presence of organisms), colonisation (replicating bacteria) and infection (the invasion of organisms into the tissue) represent a continuum. Infection can usually be determined in the physical exam by looking for the cardinal signs of inflammation: erythema, pain, swelling, and increased temperature. Many patients, however, are clinically unable to mount an inflammatory response, and in those patients the use of quantitative culturing along with clinical intuition might be necessary. There are numerous ways to obtain cultures of a wound but the quantitative biopsy remains the gold standard. *(22)* 

#### **LCD: Nutrition/Immune Status**

The third category within the LCD model focuses on the immunological state of the patient. Malnutrition is a major factor to consider in this section. Although the literature fails to provide statistically significant relationships between healing and nutritional status, it is obvious that a patient needs to be nutritionally sound in order to maximise their chances for healing. Patients should not be considered for surgical wound closure until they have achieved optimal nutritional status and laboratory testing confirms success via tests for parameters such as pre-albumen level, serum transferrin level, total lymphocyte count, and albumen level. Every attempt should be made to support patients with multivitamins, minerals, and enteral or parenteral support. Patients should be evaluated for other forms of immunosuppression such as use of steroids, anti-metabolic agents, overwhelming infection, and chronic disease states such as diabetes and HIV.

#### **LCD: Psychosocial Issues**

Psychosocial issues discussed earlier in the paper should be re-examined at this point. Chronic depression has been shown to affect healing, and many wound patients suffer from psychiatric conditions which can be identified if the clinician carefully probes during the history. Pain can exacerbate underlying psychiatric issues and can delay healing through the psycho-neuro-immunological connections. (23) As society ages and more patients live with chronic diseases, we will be faced with patients for whom healing options are limited and for whom maintenance of the wound bed, prevention of infection, exudates management, odour control, and wound pain issues take priority over healing. (24)

#### LCD: Pressure/Neuropathy

The LCD next addresses pressure on the wound and surrounding tissues. Pressure must be offloaded in order to maximise healing in any wound location. This seems obvious when dealing with classic pressure ulcers located on the trunk but also applies to pressure from wheelchair leg rests, bed railings, oxygen tubing, and improperly fitting footwear.

There are many products available to offload the patient, including mattresses, orthotics and prosthetics, and foam padding for wheelchairs and beds.

#### LCD: Wound Bed and Periwound Tissue

The wound bed is the last aspect of the LCD model. The periwound tissue is assessed first. The skin can be painful to the touch, erythematous, macerated, dry and cracked, or edematous. A patient's continence status may play a pivotal role in wound healing and should be noted in the record. It is important to develop a consistent, reproducible method to measure the wound. Current technologies include planimetry and digital cameras that have the capacity to measure depth. Even if a simple disposable ruler and cotton swab are used, it is important that the technique is well described in a policy and that all providers perform the measurement the same way.

The wound bed must be assessed for the state of moisture balance, presence of necrotic tissue, and quality of granulation tissue. If the wound has been present for six months to a year and/or has an abnormal appearance, then strong consideration should be given to performing a biopsy. Histology can help achieve a diagnosis, rule out malignancy, or provide confirmation to a clinical suspicion. The threshold for biopsy should be low, as many wounds do not demonstrate classical features on presentation. After a macroscopic view of the wound bed, the clinician should consider micro-environmental issues. Microcirculation has already been reviewed, and those results need to be documented in the patient's record as well.

The biochemistry of the wound is becoming more important to wound healing as new concepts in healing have been elucidated. A hostile wound environment with excess matrix metalloproteases can lead to the destruction of both endogenous and exogenous growth factors, leading to delayed healing. As previously mentioned, the bioburden of the wound can lead to the presence of metabolic waste products in the wound bed. Inflammatory mediators and cytokines can create an environment that does not allow proliferation. Interestingly, this situation can develop with adequate macrovascular flow. Consider the patient with a venous ulcer, palpable pulses, but severe lipodermatosclerosis and relative dermal tissue hypoxia. Such a wound would be difficult to heal and might require modifying the biochemical composition of the wound bed to accelerate healing.

#### Summary

The differential diagnosis of leg ulcers is extensive and covers diverse disease states. Systematically performing a history and physical, and considering all the features from the LCD model should enable the clinician to narrow the list to a provisional diagnosis and two or three potential confounding conditions. At this point, laboratory testing can help arrive at a diagnosis and a treatment plan prescription. The patient should be seen weekly from weeks 1 through 4. If there is little change in either wound dimensions or quality of wound tissue after the fourth visit, then the diagnosis should be reconsidered. There is confirmatory data that progress in the first four weeks of treatment is a surrogate marker for overall healing outcomes. (25) The patient should cycle through the diagnostic process again. This will ensure nothing is missed and will minimise any time spent on an ineffective treatment protocol. Using this system, the author has been able to achieve similar healing rates at a community hospital and a tertiary care facility. There have been no statistically significant differences in healing rates among the various wound aetiologies in either site of care. (26)

The terminology used in wound care needs to be standardised as well. For example, in order to compare healing rates from one group to another, it is important to know which patients were included or excluded. Using terms such as *intention to treat* with a prescribed definition can assist in comparing outcomes and techniques throughout the world.

#### **Key Practice Points:**

- The practice of wound management is as challenging, demanding, and complex as that of any other field in medicine.
- The clinician who treats a recalcitrant wound must be skilled in communication, negotiation, and change management in order to coordinate an interprofessional team of providers focused on a unified objective.
- The use of the Comprehensive Wound Assessment and Treatment System (CWATS), which includes the Least Common Denominator Model (LCD model), is an approach clinicians can use to standardise their wound care patient work-up.

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# Integrating the Concept of Wound Care, the Importance of Wound Treatment, Wound Treatment Education, and the Wound Care "Team" in Resource-limited Areas Around the World

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Since the first person was injured, there has been a continuing debate over the best, most efficient, and most cost-effective way to treat a wound. Today, the treatment landscape of acute and chronic wounds worldwide varies so greatly that establishing a single, uniform "best" way to manage a wound is not likely to be possible. Managing wounds in countries where there are many resources and healthcare providers will differ from treatments in areas with few resources and few, if any, trained healthcare providers. Effective wound treatment that improves the quality of life of all people and returns them to productive, self-sufficient lives is a challenge that must be met to prevent further loss of every country's most precious resource: their people. Formulating ways to integrate the concept of wound care, the importance of wound treatment and wound treatment education, and the idea of a team approach to wound care is crucial if we are to be successful in the improvement of wound care in all parts of the world.

As our knowledge of the pathology and physiology of wounds has evolved, it has become apparent that the healing of a seemingly simple wound can be very complex and involve multiple body systems that influence the process. These multiple influences, called co-morbidities, require control if proper healing is to occur. Increasing medical specialisation has multiplied the number of medical and surgical specialties and paramedical personnel involved in providing optimal care for patients with wounds. (1–6)

#### The Team

Surgical specialists, including general surgeons, vascular surgeons, orthopaedic surgeons, and plastic surgeons, are needed if operative debridement, revascularization, skin grafting, or amputation is indicated. Podiatrists can provide basic foot care services, perform preventative and corrective operative procedures, and ensure appropriate foot wear. Other specialists are needed for patients with medical conditions that influence wound healing, such as diabetes mellitus, anaemia, heart disease, renal disease, and vasculitis. Infected wounds may require the attention of infectious disease specialists. Nurses and physical therapists are invaluable in overseeing the patient's care and helping the patient return to a productive life. Dietitians and diabetes educators can help manage nutritional problems and nutritional education. Social workers can provide needed access to resources for the patient and family. Pharmacists can help patients get the recommended medications and assist in providing education to the patient or caregiver regarding the appropriate use of the medication.

Each individual specialty involved in care of the patient with a wound brings a particular knowledge base about the wound and its treatment that strengthens the entire team and fills any knowledge gaps that might exist. (3)

In developed countries, wounds are best treated in an environment that brings together current knowledge of the care of wounds with physicians with multiple specialties interested in providing good care for these patients and the equipment to provide that care. That environment is a wound care centre that specialises in the treatment of patients with wounds. (4)

Evidence shows that patients with chronic wounds treated in a centre specialising in wound care have better outcomes than patients treated in physicians' offices or community clinics. The studies show that after 12 weeks of therapy, 53% of the patients treated in the specialised clinics healed versus 12% of the patients treated at other locations. After 24 weeks of therapy, 68% of the patients in the specialised clinics healed versus 29% of the patients treated at other locations. The studies also show that the recurrence rate of the ulcers treated in the specialised clinic was lower. Recurrence rates were 23% and 43% at six months post-healing, and 23% and 54% at 12 months respectively. (7)

A multi-specialty team makes treatment of the whole patient with a wound much easier. There are team members who can interact with the patient and family to ensure the recommended therapy can be accomplished by the available caregivers. If family members are unable to help with the care of the patient, outside help can be arranged for the patient. Team members can help the patient or family obtain the appropriate treatment medications and bandages, or arrange transport to diagnostic testing and follow-up appointments. Other special needs of the patient and family can best be met by a team of caring individuals with individual talents and abilities. (3, 5)

#### The Team in Resource-limited Countries

In developing and resource-limited countries, the approach should be the same but may be impossible when there is a lack of trained personnel, education, and resources. In health centres in developing countries where multiple specialties are available, all efforts should be made to involve each centre in the most up-to-date treatment of wounds possible for the resources available. Available specialists can come together as a team to become proficient in wound management. It is reasonable to expect at least one centre in each developing country be a "centre of excellence" containing personnel with the knowledge and expertise to treat difficult wounds. (8) These centres should become educational hubs that spread wound care education throughout the country and be the resource centres for treatment of the most difficult wounds.

#### **Overcoming Barriers**

Unfortunately, establishing centres of excellence in wound management may be difficult unless there is a change of attitude among the healthcare providers. Many physicians view chronic wounds with disinterest and frustration and may treat wounds with outdated and ineffective therapies or pass the patients on to anyone willing to manage the problem. (4)

I visited an excellently equipped, multi-specialty hospital in South America and found the wound patients relegated to a small office run by two diligent nurses who had very few resources at their disposal to treat a large number of wound patients each day. They had no physician in attendance and had to practically beg physicians to assist them with difficult cases. The importance of wound care must be stressed to the medical staff of each major health centre. Everyone, including healthcare workers, community leaders, and leaders of each country, must understand how good wound care can improve the lives of their people and recognise the fact that it is economically advantageous to treat these people and return them to the workplace or, at least, to a productive, self-reliant life.

What is the best way to provide information about the importance of wound care? Many times the adoption of important concepts and new ideas is unreliable and slow. If we depend on conferences, journals, recommendations from so-called experts in the field, guidelines, and word of mouth to spread new and important information, we shall be disappointed with the results. As McCannon and colleagues state: "Good ideas, even when their value is thoroughly demonstrated in one place, will not reliably spread into action through normal communication channels at a pace truly responsive to the enormous healthcare challenges in resource-poor settings." (9)

Existing public health structure must be used to spread new information in developing countries. The new information must be compatible with the social structure into which it is being sent. Educators must take into consideration the local politics of the people currently providing the care, carefully evaluate ideas that may conflict with the current practices and values, consider the resources needed to implement the new ideas—especially in resource-limited areas—and take into account the educational and skill level of the people providing the care in each location. (9) Without a thorough knowledge of the impact the introduction of a new program will have on the local people and the healthcare providers, the program is destined for failure.

The important issue is to remove any perception of risk on the part of the person adopting the new information. This can be done by showing them five positive characteristics of a new program, as described by Rogers. (10)

- 1. The relative advantages of the new program *for the current practitioner* must be made known.
- 2. The compatibility of the new program with the current practices and beliefs must be stressed.
- 3. The simplicity of the new program must be apparent.
- 4. The persons adopting the new program must be able to try all or part of the program before committing to it completely.

5. The lack of hidden agendas and program difficulties must be apparent to the persons considering the program.

Addressing these issues will increase the likelihood that the program and its innovations will be embraced by the local people, whether in the city or in rural areas. After presenting the educational information, the teams of people must be brought together to discuss the new programs and share ideas as to the best method of implementation in each area. The best results will occur when we are able to recruit local leaders and healthcare workers to help formulate those plans and to spread the ideas and practices to their colleagues and patients. (11)

#### **Key Personnel**

In underserved areas, it will be necessary to identify the healthcare provider for the village or area. Such a provider may not be a physician but a person who has been trained by the national health program of the country, a self-taught member of the community whom people respect as a healer or a leader in matters of health, or simply someone in the village who is kind and willing to help those with medical problems. (12)

In Togo, West Africa, a "surgeon" who is recognised as an expert in inguinal hernia repair is a former gardener for the hospital. He expressed an interest in helping a missionary surgeon in the operating room and became a proficient assistant. He was subsequently trained by the surgeon to repair inguinal hernias, which freed the missionary surgeon to do the more complicated procedures that required his advanced skills. Patients needing hernia repair in his region of Togo now ask to see the "gardener surgeon" and not the missionary surgeon. We just need people who are interested in learning about wound care and who have a heart for helping others.

#### **Implementation Challenges**

Since the "multi-specialty wound team" in developing areas may be composed of people who have little formal education, the programs we introduce must be understandable and easily implemented. We must train them so they can provide good wound care within their cultural and religious beliefs and with the resources available to them. We must teach them how to make do with what is available. Wound care products and treatments not used in some countries may be the only ones available. Even the basic need to wash one's hands and the patient's wound may be difficult if there is no water. The treatment of infection may be difficult if antibiotics are unavailable. If we fail to address these issues, the programs, no matter how well intentioned, will not succeed. In addition, teaching people to help assume responsibility for their care (self-care) will make these efforts more successful. (13) Attempts at major health initiatives using the skills of healthcare providers other than physicians, such as the Community-based Health Planning and Services project in Ghana, have proven successful in underserved areas with other diseases. (14–16) There is no reason to believe they would not be successful for the treatment of wounds.

### Summary

The goals for this project must be to establish at least one centre of excellence in wound treatment and wound education in each country. Good wound management principles can then be taken to the surrounding areas. This can only be done by identifying the local caregivers and giving them the educational tools and resources needed to practise good wound care within the bounds of their religious and cultural beliefs. Project monitoring and training evaluation must be integrated into every level if the project is to make a sustainable difference in the lives of the people it intends to help.

#### **Key Practice Points:**

- Establish a centre of excellence in each region.
- Identify local caregivers and support them in spreading the principles exercised at the centre of excellence to surrounding areas.
- Ensure new information is compatible with the social structure into which it is being sent, does not conflict with the current practices and values, and takes into account the educational and skill level of the people providing the care in each location and available resources.

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# **Wound Healing: The Role of Compression Therapy**

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Compression therapy in the treatment of lower extremity wounds is now recognised as an essential component of wound healing therapy. (1,2) It is therefore important that oedema and lymphoedema be defined.

#### Oedema vs. Lymphoedema

Traditionally, the terms oedema and oedematous are used to describe any limb or organ that becomes swollen. However, oedema should be differentiated from lymphoedema. Protein content defines the difference. Lymphoedema is high-protein oedema, which is the result of damage to, or absence of, the normal lymphatic system. Oedema, in contrast to lymphoedema, is mostly water. Common examples of causes of oedema include congestive heart failure, chronic venous insufficiency, hepatic cirrhosis (ascites), nephrotic syndrome, and the oedema secondary to the inflammatory wound healing response.

The aetiology of lymphoedema is divided between primary (congenital) lymphoedema (10–20%) and secondary lymphoedema (80–90%). The aetiology for secondary lymphoedema includes surgery, trauma, venous disease, filiariasis, and chronic wounds. (3)

The complications of lymphoedema are secondary to the high protein content in the interstitial fluid. The persistent increase of protein and its degradation products results in chronic inflammation. This is seen in the increased number of macrophages, fibroblasts, and lymphocytes. The inflammation and resulting fibrosis and sclerosis are seen in all affected tissues. Disruption of local metabolism and an increased rate of cellulitis lead to hemangio-lymphangiopathy and progressive lymph stasis.

Treatment for oedema differs from the treatment for lymphoedema. Oedema, consisting mostly of water, is treated with diuretics and, when necessary, compression. Standard treatment for lymphoedema is compression. In pure lymphoedema, diuretics are contraindicated. Water functions as media for lymph transport. Diuretics may induce fluid and electrolyte imbalance by decreasing the water content of the lymph fluid and increase the viscosity, thereby hindering the mobilization of lymphoedema. (4)

#### Lymph Stasis and Wound Healing

To appreciate the relationship of lymphoedema to wound healing, a review of the pathophysiology of chronic venous insufficiency and venous stasis ulceration is helpful. Chronic venous insufficiency leads to venous hypertension, which results in a high filtration pressure, causing increased fluid to appear in the tissues, i.e. increased lymphatic water load. When the lymphatic transport capacity is exceeded by the water load, a state of low-protein oedema occurs after this dynamic failure. Constant lymphatic hypertension causes infiltration of lymph into the peri-lymphatic tissue, resulting in fibrosclerosis and lymphangitis. The phlebology literature is rich in theory and observation of fibrin cuffing, white cell sequestration, and oxygen free radicals. These findings are additional indications of chronic inflammation tissue repair. Protein permeability increases and lymphatic damage follows. Subsequently, lymphoedema (high protein oedema) becomes the underlying pathology that contributes to the formation of venous ulcers.

Venous ulcers often exhibit many of the characteristics of the non-venous chronic wound: normal arterial blood supply and obstructed lymphatic drainage of the chronic wound fluid discharge. The chronic wound fluid is characterised by elevated inflammatory cytokines and matrix metalloproteases (MMPs). (5) With compression and reduction of the periwound swelling, these wounds will heal in the majority of cases. Given exactly the same parameters in non-venous, acute, and chronic wounds throughout the body, controlling the periwound lymphoedema will result in enhanced wound healing.

Removing excess chronic wound fluid is thought to remove inhibitory factors present in the fluids. Studies have shown that fluids removed from chronic wounds suppress the proliferation of keratinocytes, fibroblasts, and vascular endothelial cells *in vitro*. (6) Argenta and Morykwas, in their investigations related to vacuum-assisted closure of wounds, have provided valuable insight into the consequences of lymph stasis and the healing wound. (7) Their technique removes chronic wound fluid (lymph), which has been proven to dramatically enhance wound healing. By moving retained lymphatic fluid past obstructed lymphatics to patent lymphatics, compression therapy provides many of the positives attributed to negative pressure wound therapy (NPWT).

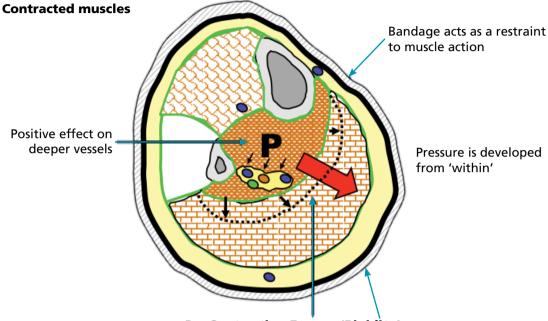
Until the past 10 years, compression bandaging was thought to be related only to venous wounds. We now recognise that the vast majority of all acute and chronic wounds exhibit some degree of lymph stasis.

#### The Physics of Compression

The application of external compression initiates a variety of complex physiological and biochemical effects involving the venous, arterial, and lymphatic systems. The bandage material used determines the compression effect. Short-stretch (reduced elasticity) bandages are the preferred bandage for the treatment of periwound oedema and lymph stasis. Short-stretch bandage compression more nearly imitates the dynamic, normal physics and physiology of extremity fluid mobilization. Short-stretch bandages cause a higher pressure during activity (working pressure) and relatively low pressure at rest (resting pressure). The Unna boot was the original template for non-elastic bandage compression.

Resting (static) pressure is the pressure produced by the compression bandage or device while the muscle is relaxed. The pressure is due to bandage tension projecting an inward radial pressure. The superficial vessels are affected the most. Laplace's Law states that, in general, the tension of the applied bandage on the extremity is inversely proportional to the tension of the application divided by the radius of the extremity. This is an important consideration in preventing tissue damage when applying a compression wrap to a small ankle, the malleolus or the tibial ridge (see Figure 1).

Figure 1: Working (dynamic) pressure



P = Contraction Force x 'Rigidity'

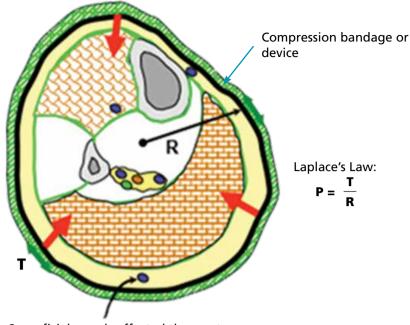
Illustration courtesy of Dr. H.N. Mayrovitz

Working (dynamic) pressure is the pressure produced when the muscle of the extremity contracts. The bandage acts as a restraint to muscle contraction. The pressure is developed from within and has a positive effect on the deeper vessels. The pressure achieved is equal to the contraction force times the rigidity of the bandage (see Figure 2).

#### Figure 2: Resting (static) pressure

Muscles relaxed

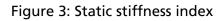
Pressure due to bandage tension (T) projecting an inward radial pressure (P)

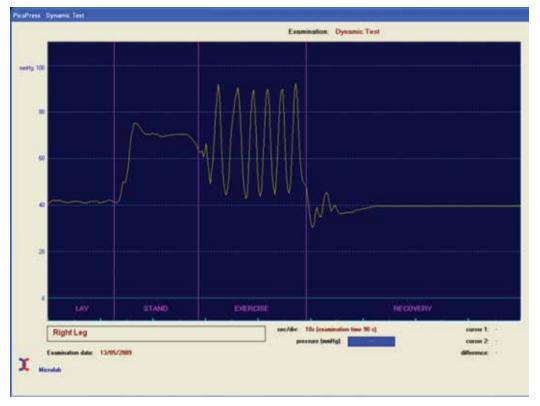


Superficial vessels affected the most

Illustration courtesy of Dr. H.N. Mayrovitz

It should be understood that if the bandage device moves with muscle contraction, the resulting tissue pressure is reduced and the mobilization of fluid is reduced. Therefore, the dynamic pressures depend upon the bandage material features. The higher stiffness of the short-stretch bandage is characterised by the higher pressure amplitudes during movement, but also by the higher increase of the pressure by standing up from the supine position. This is called the "Static stiffness index". A static stiffness index in the 30 mmHg range is the recommended index for short-stretch bandages (see Figure 3).





It is important to recognise that dynamic compression is intermittent compression rather than sustained compression. Intermittent compression as high as 80 mmHg can be tolerated in normotensive limbs. It is the dynamic properties of intermittent compression that most imitates normal physiology. Sustained pressure of 40 mmHg or more can create a tourniquet effect and is not tolerated.

# **Physiology of Compression**

Dynamic, intermittent compression improves the venous and lymphatic return. (8–10)

- Reduction in venous reflux
- Improvement of venous pumping function
- Reduction of ambulatory venous hypertension
- Increase of arterio-venous pressure gradient
- Improvement of lymphatic drainage

Compression affects arterial flow.

- Light sustained pressure < 40 mmHg enhances arterial flow.
- Higher sustained pressure reduces arterial flow.
- Intermittent, dynamic pressure enhances arterial flow.

The increase of arterial flow under light compression is secondary to:

- Myogenic relaxation of the arterial wall
- Release of vasodilating mediators

• Reduction of the arterio-venous gradient by improvement of venous return (11–14)

Studies have demonstrated in patients with mixed arterial-venous ulceration and arterial-brachial indices greater than 0.5 that dynamic compression up to 40 mmHg:

- Increases arterial flow under the bandage
- Does not reduce arterial flow distal to the bandage.
- Increased distal transcutaneous oxygen pressure (TcP0,)
- Significantly improves venous pumping (15)

## **Global Health Wound Care**

Effective wound care in resource-limited environments is a priority for global health. Adherence to the noted five principles of wound care form the basis is this therapy. Control of periwound oedema/lymphoedema is often the most difficult challenge. Clinician education, material supply availability, cultural adaptation, and environmental conditions must be considered.

## **Clinician Education**

All clinicians with wound care responsibilities must be trained in the safe application of bandage compression. While short-stretch bandaging is the first choice for compression, depending upon material availability, fundamental teaching states that, in the presence of detectable periwound oedema/lymphoedema, some compression is better than no compression. Bandage compression should only be applied by adequately trained clinicians. Post healing and long-term prevention with clinician instruction in the use of compression garments then becomes the responsibility of the patient.

## **Bandage Availability**

Material supply of bandages and cost vary dramatically in resource-limited nations. While non-elastic bandage material is preferable, multiple options exist with the use of both non-elastic and elastic bandages, roller gauze, and Unna boot variations. Reports from India and the use of elastic bandages in the successful compression of lymphatic filiariasis illustrate the many options. (16)

## **Cultural and Environmental Factors**

Depending on local environmental factors such as temperature, air pollution, contamination, and availability of clean water, adjustments in patient compliance and frequency of bandage change must be considered. Quite obviously, one size does not fit all! The clinician must be adaptable to whatever bandage system reduces periwound swelling. The clinician must act as the patient advocate and develop patient compliance that will assure healing.

### **Key Practice Points:**

- The key intervention in the management of oedema is compression therapy.
- A full lower limb assessment, including arterial flow, must be completed before implementing compression therapy.
- Inelastic or short-stretch systems are the most effective forms of compression.

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# **Wound Infection: Diagnosis and Management**

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#### Introduction

All open wounds contain microorganisms, yet the majority are not infected. Wound infection depends on the number of invading organisms present, their virulence, and the ability of the host to manage the bacterial load. The spectrum of interactions in the microbial communities is illustrated in Table 1. (1) The host may gradually reach a point at which wound healing is impaired. When this occurs, immediate intervention to pre-empt infection is indicated. (2)

Bacterial biofilms are now considered to be one of the key contributors to chronic wound pathogenesis, and "hard to heal" recalcitrance, alongside hypoxia, ischemia-reperfusion injury, and intrinsic host factors. (3) Biofilm development involves a cycle of attachment, growth involving persister cells, and detachment of planktonic phenotypes. The rate of biofilm formation in chronic wounds can be rapid.

Term	Clinical Interpretation	Clinical Intervention
Contaminated	Bacteria on surface only. No signs or symptoms.	Monitoring and risk reduction
Colonised	Bacteria attached to surface, starting to form colonies, minimally invasive. No local tissue damage.	Monitoring and risk reduction
Localised Infection	Bacteria more deeply invasive. Local wound bed involved. Healing compromised in healable wounds. Subtle signs of infection may be present including: • Friable bright red granulation tissue • Delayed healing • Bridging or pocketing • Increased or altered exudate • Increased odour • Increased pain • Localised oedema	Intervention required. Often can be managed with local measures such as topical antimicrobials or antimicrobial dressings in addition to effective debridement.

Table 1: Bacterial Burden in Chronic Wounds

cont'd.

Spreading Infection	<ul> <li>Bacteria now involve the surrounding tissues. In addition to the subtle signs described above, classic signs of infection such as pain, redness, heat, and swelling may be present. Other signs and symptoms include:</li> <li>wound breakdown with satellite lesions</li> <li>induration and redness extending well beyond the wound borders</li> <li>lymphangitis</li> <li>general malaise</li> </ul>	Intervention required. As for localised infection, plus systemic antibiotics.
Systemic In- fection	Classic signs of sepsis, including pyrexia or hypothermia, tachycardia, tachypnoea, elevated or depressed white call counts, and, in more severe cases, multi-organ system failure.	Intervention required. As for spreading infection. Other sources of infection need to be ruled out. Systemic and topical measures required.

Adapted from International Wound Infection Institute (1)

### **Risk Factors for Infection**

Any factor which impairs the ability of the host to mount a response to bacteria in an open wound increases the risk of infection. These factors may include:

- 1. Co-morbid conditions such as:
  - obesity
  - renal failure
  - diabetes
  - collagen vascular disorders
  - malignancy
  - aenemia
- 2. Medications which suppress immune function such as corticosteroids and chemotherapeutic agents also increase the risk of infection.
- 3. Poor tissue perfusion is a key risk factor for infection.
- 4. Wound-related factors may include: presence of necrotic tissue or a foreign body, prolonged duration, large size or depth, and anatomical location.
- 5. Patient factors such as poor hygiene and treatment choices must also be considered.
- 6. Caregiver factors such as poor hand hygiene or dressing techniques may put the patient at risk.

The clinician must consider all these factors and develop strategies to mitigate them to reduce the risk of infection.

## **Recognising an Infected Wound**

The diagnosis of wound infection is primarily clinical. The assessment should include evaluation of host factors, the surrounding skin, and the characteristics of the wound itself. Wound swabs, while helpful in directing treatment, do not in themselves diagnose infection and are not readily available in resource-limited settings. While there are no validated tools to assess for wound infection, the following simple checklist based on the international consensus document may be helpful in deciding on the level of bacterial burden in a chronic wound. (4, 5) At each assessment, the clinician checks the appropriate boxes if the signs or symptoms are present and leaves them blank if they are absent.

Table 2: Bioburden Checklist

Group	Signs and Symptoms	Date (yy/mm/dd)		
A	Stalled healing			
	Friable and bright red granulation tissue			
	Increasing or altered exudate			
	Increasing malodour			
	Localised oedema			
	Increased pain			
В	Increasing induration plus erythema extending well beyond wound borders			
	Wound breakdown and/ or satellite areas of breakdown			
	Lymphangitis			
	General malaise			
C	Fever			
	Rigors			
	Chills			
	Hypotension			
	Organ failure			

**Clinical Interpretations:** 

Level of Bioburden	Definition
Colonised: At Risk	No signs or symptoms from any group; clinical decision based on location of wound and co-morbid conditions
Localised Infection	Presence of two or more signs or symptoms from Group A
Spreading Infection	Presence of 2 or more signs or symptoms from Group A PLUS one or more from Group B
Systemic Infection	Presence of any sign or symptom from Groups A and B PLUS one or more from Group C

From Woo and Sibbald (6)

## **Techniques for Disrupting Infection (and Biofilms)**

In Table 1, clinical interventions are described for various levels of bacterial burden in wounds. Localised wound bioburden is managed through good cleansing, effective debridement and judicious use of antimicrobial dressings. For more deeply invasive infections or sepsis, systemic antibiotics are also required in addition to local measures. Recently discussion has focused on disrupting biofilms. To date the most effective intervention appears to be the "clean and cover" approach using effective debridement followed by application of an antiseptic dressing to prevent the biofilm from reoccurring.

## Cleansing

Much controversy has existed around which cleansing agents are appropriate. In general, potable water is considered safe for wound cleansing. In resource-limited settings obtaining potable water may be a challenge. Antiseptic cleansers were at one time considered toxic and were contraindicated. However, antiseptic wound cleansing agents which can disrupt biofilms have become increasingly used in clinical practice. In resource-limited settings antiseptic cleansers may be the safest choice. Surfactant-containing cleansing solutions have also been shown to disrupt biofilms. However, without repeated debridement, biofilm often rapidly reforms. Common antiseptic cleansing agents include sodium hypochlorite solutions, chlorhexidene, dilute acetic acid, and povidone iodine.

## Debridement

Traditional techniques for debridement have included supporting autolytic debridement through dressing choices, enzymatic debridement using exogenous collagenase applied to the wound, conservative sharp debridement using surgical instruments to remove non-viable tissue, or more aggressive surgical debridement. Mechanical debridement with saline wet-to-dry dressings is not considered an effective debridement modality, but irrigation with safe irrigating fluids can be used. In some settings maggots are used to reduce debris and bacterial burden. (7) Other advanced forms of debridement are available but are not common in resource-limited settings.

## **Antimicrobial Dressings**

Following proper wound cleansing and debridement, appropriate wound dressings should be selected. The selection of such dressings is dependent on wounds status and the treatment goals for the wound and the patient as well as on the resources available.

When a wound is assessed as locally infected, dressings containing topical antiseptics should be selected. The selection of such a dressing is dependent on wound condition, exudate level, adaptability of the dressing to suit the wound, patient comfort, associated pain, and the treatment goals for the wound and the patient. There is little advice to be obtained from systematic reviews regarding choice of topical antimicrobials, and most practice must be based on the results of research which has been performed *in vitro*. The specificity and efficacy of the agent, its cytotoxicity to human cells, its potential to select resistant strains, and its allergenicity must be considered. (1) Modern topical antiseptics

include polyhexanide (PHMB), (8) silver, (9) iodine, (10) and honey. (11) Table 3 provides a general overview of some of the common antimicrobial dressings available. These may have different trade names and are not universally available in all settings.

Antimicrobial Agent	Dressing Forms	Comments
Medicated Tulles	<ul> <li>Petrolatum gauze or other non-adherent vehicles impregnated with:</li> <li>Antibiotics such as framycetin, fucidic acid or bacitracin zinc</li> <li>Antiseptics such as chlorhexidene or iodine</li> </ul>	<ul> <li>Bacterial resistance may develop to antibiotics.</li> <li>Antibiotics may cause irritation or allergy.</li> <li>Antiseptic preparations are preferred.</li> </ul>
Silver Dressings	Vehicles may include: • Alginates • Foams • Hydrophilic fibres • Gels • Powders • Impregnated gauze • Combined with oxidised regenerated cellulose/ collagen • Combined with collagen • Coated polyethylene mesh • Impregnated hydrocolloids • Combined with charcoal in a sachet	<ul> <li>Silver may be atomic, oxysalt, or ionic form.</li> <li>Broad spectrum of activity against bacteria</li> <li>Debate about effectiveness of high- vs. low-release formulations</li> <li>Some formulations kill bacteria within dressing.</li> <li>May reduce inflammation through reduction in MMPs</li> <li>May be useful against biofilms in the "debride-and-cover" strategy</li> <li>Charcoal-containing preparation may be useful for odour control.</li> <li>Choose vehicle depending on other wound characteristics.</li> </ul>
lodine	<ul> <li>Three preparations:</li> <li>Iodophor-impregnated gauze</li> <li>Slow-release molecular iodine in cadexomer starch beads</li> <li>Povidone-iodine-impregnated non-adherent dressing</li> </ul>	<ul> <li>Broad-spectrum activity against gram negative, gram positive, anaerobes, viruses, and fungi</li> <li>Some evidence of effectiveness of the cadexomer form against biofilms but all may be useful in the "debride-and-cover" strategy.</li> <li>Cadexomer starch absorbs wound fluid (6x weight).</li> <li>Care with large amounts over long periods due to possible thyroid interaction</li> </ul>

cont'd.

Polyhex- amethyline Biguanide (PHMB) or Polyhexa- nide	Multiple preparations <ul> <li>Ribbon gauze</li> <li>Gauze squares</li> <li>Transfer foam</li> <li>Backed foam</li> <li>Non-adherent</li> </ul>	<ul> <li>Broad spectrum of activity</li> <li>Bacterial kill largely in dressing</li> <li>Choose vehicle based on wound characteristics.</li> <li>Ribbon gauzes are particularly useful for sinuses.</li> </ul>
Hypertonic Saline	Hypertonic saline in • Gauze • Gel	<ul> <li>Help to debride necrotic tissue</li> <li>Help to control bacterial loads</li> <li>May be painful</li> </ul>
Honey	Leptospermum honey in • Liquid form • Alginate pads • Hydrocolloids	<ul> <li>Biocidal effect is multifactorial.</li> <li>May assist with autolytic debridement</li> <li>Choose formulation based on wound characteristics.</li> </ul>

From Keast (12)

## Conclusion

Managing wound infections should always be made based on an assessment of the microbial burden in the wound, the host defence of the patient, the type of wound, and the location and condition of the wound. Modern antiseptics for wound management have proven to be safe and efficient, and should not be confused with old, cytotoxic preparations. Wounds in children, major wounds and some patients' specific sensitivities to components in the antiseptic require consideration when selecting topical antiseptics. Manufacturers' instructions should always be followed. Microbial resistance to antiseptics is rare. In the global threat of increasing antibiotic resistance and environmental hazards associated with antibiotics, prevention and treatment of locally infected wounds with topical antiseptics such as polyhexanide (PHMB), povidone iodine, silver, honey, and similar products is an attractive option.

#### **Key Practice Points:**

- The factors in wound infection are microbial number and virulence in the context of host defences.
- All wounds exist somewhere along a wound infection continuum from colonised to systemic infection.
- Diagnosis of wound infection is primarily a clinical judgement based on observable signs and symptoms.
- The key interventions in managing wound infections are good cleansing with safe fluids, appropriate debridement, and covering with an appropriate dressing.
- Systemic antibiotics should be reserved for spreading or systemic infections.

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# **Pressure Injuries**

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## Introduction

Classically, a pressure injury (PI), or pressure ulcer (PU), is localised injury to the skin and/or underlying tissue, usually over a bony prominence. It is a consequence of direct pressure and/or shear forces within the tissues (see Figure 1). Both modalities can act in combination. Just a few hours are necessary to produce a PI.

The application of long-term pressure can result in skin and subcutaneous hypoxia, cells lacking oxygen, which in turn will produce necrosis that can extend to the underlying bone, joints, tendons, etc.

Figure 1a: Pressure

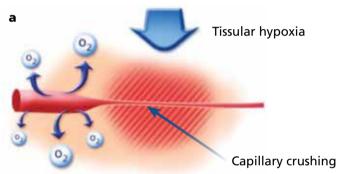
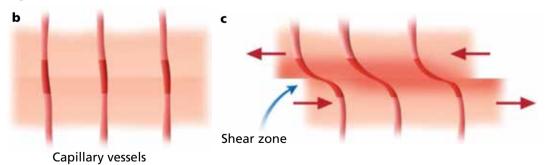


Figure 1b and c: Shear, before and after



From escarre.fr (5). Used with permission.

A number of contributing or confounding factors are also associated with PIs and have to be taken into account as risk factors:

- loss of sensory perception
- loss of motility

- advanced age
- poor perfusion and oxygenation
- poor nutritional status
- increased skin moisture
- increased body temperature
- low haematological measures
- poor general health status

Many of these risk factors are included in the numerous PI risk scales available, such as those developed by the European Pressure Ulcer Advisory Panel (EP-UAP) and National Pressure Injury Advisory Panel (NPIAP) (formerly the National Pressure Ulcer Advisory Panel) in the United States. (1)

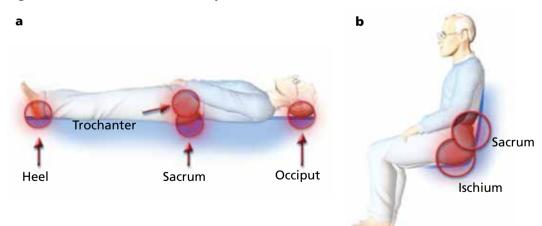
The consequences of pressure injuries may include pain, lengthened hospital stay, high treatment costs and death.

A very large prevalence study in healthcare facilities (mostly in the U.S.) shows that 15% of the patients suffered from PIs. (2) These figures can be much higher depending on setting and diagnosis. For example, 29.8% of patients in a South African spinal cord injury centre had a PI. (3) In two teaching hospitals in Cameroon, the global prevalence was 20% and the prevalence in the "at risk" population (assessed following Braden scale) was 50%. (4)

Pressure injuries occur over bony prominence (see Figure 2) so clinicians should be particularly observant of areas of the body that may be subject to pressure and shear, depending on their position. For example:

- if lying on the back: occiput, sacrum and tallus
- if on the side: shoulder, trochanter, condyle, malleolus
- if sitting: sacrum and ischium

Figures 2a and b: Areas of Bony Prominences



From escarre.fr (5). Used with permission.

It must be remembered that medical devices can also cause pressure injuries. Furthermore, pressure injuries can occur on mucosa.

## **Risk Evaluation and Diagnosis**

Two things are very important: 1) risk assessment for preventing the development of pressure injuries and 2) staging existing PIs to provide a basis for treatment plans.

- To identify patients at risk for developing PIs, risk scales (Braden, Waterloo, and Norton being the most used) are available and can provide a range of parameters that should be considered. But the bottom line is that patients who are unable to move (reposition) themselves and and/or who lack skin sensation are at highest risk.
- 2. In cases where a PI already exists, the clinician needs to evaluate the extent of the injury, treat it appropriately and prevent any further damage through prevention strategies. The consensus document of specialised societies (1) is a very useful tool for evaluating and documenting PI severity and zes four or six categories or stages of PI.

Table 1: National Pressure Injury Advisory Panel (NPIAP) Staging and Illustrations

Healthy Skin	Healthy Skin – Lightly Pigmented	Healthy Skin – Darkly Pigmented
<ul> <li>Category/Stage 1: Nonblanchable</li> <li>Erythema</li> <li>Intact skin with non-blanchable redness of a localised area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area.</li> <li>The area may be painful, firm, soft, warmer or cooler as compared to adjacent tissue. Category/Stage 1 may be difficult to detect in individuals with dark skin tones. May indicate "at risk" individuals (a heralding sign of risk).</li> </ul>	Stage 1 Pressure Injury – Lightly Pigmented	Stage 1 Pressure Injury – Darkly Pigmented

cont'd.

<ul> <li>Category/Stage 2: Partial-thickness</li> <li>Skin Loss</li> <li>Partial-thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister.</li> <li>Presents as a shiny or dry shallow ulcer without slough or bruising.* This Category/Stage should not be used to describe skin tears, tape burns, perineal dermatitis, maceration or excoriation.</li> <li>*Bruising indicates suspected deep tissue injury.</li> </ul>	Stage 2 Pressure Injury	
Category/Stage 3: Full-thickness Skin	Stage 3 Pressure Injury Stage 2 Press	
<ul> <li>Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscles are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling.</li> <li>The depth of a Category/Stage 3 pressure injury varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and Category/Stage 3 ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category/Stage 3 pressure injuries. Bone/tendon is not visible or directly palpable.</li> </ul>	with Epibole	
Category/Stage 4: Full-thickness	Stage 4 Pressure Injury	
<ul> <li>Tissue Loss</li> <li>Full-thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling.</li> <li>The depth of a Category/Stage 4 pressure injury varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Category/Stage 4 ulcers can extend into muscle and/ or supporting structures (e.g., fascia, tendon or joint capsule) making osteomyelitis possible. Exposed bone/ tendon is visible or directly palpable.</li> </ul>		

cont'd.

<ul> <li>Unstageable: Depth Unknown</li> <li>Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed.</li> <li>Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore category/stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as 'the body's natural (biological) cover' and should not be removed.</li> </ul>	Unstageable Pressure Injury – Dark Eschar	Unstageable Pressure Injury – Slough and Eschar
<ul> <li>Suspected Deep Tissue Injury: Depth Unknown</li> <li>Purple or maroon localised area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue.</li> <li>Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment.</li> </ul>	Deep Tissue Pressure Inj	

From National Pressure Injury Advisory Panel (6–7). Used with permission.

Additional items are important to record regularly for treatment and follow-up:

- Location identified by bones underlying the PI
- Size (recorded weekly): length, width, depth, undermining
- State of the surrounding skin: erythema, oedema, damage, maceration, rashes or bullas
- Ulcer edge: beveled, vertical, rolled hyperkeratosis or callus
- Wound bed tissue type: % necrotic, % fibrin, % granulation tissue, % epithelium
- Exudates: type, amount, odour
- Structure(s) visible in wound, e.g., tendon, bone, joint
- Pain (ideally patient-reported): visual analogue scale (0 to 10), during the day, during dressing changes

### **Prevention and Treatment**

Although there is some controversy about the issue (8–9) all these lesions are a consequence of excessive or prolonged pressure or shear, and prevention is aimed at two main objectives: mobilization and pressure redistribution.

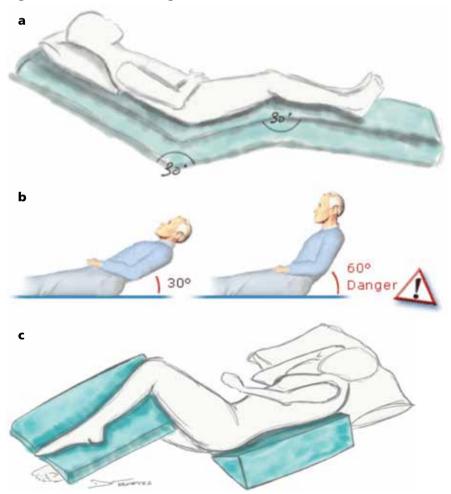
#### Mobilization

At-risk patients must be repositioned each two to four hours in alternate positions. Dorsal decubitus and ventral or dorsal 30° side positions can be used. Ventral decubitus is a possible but more difficult alternative.

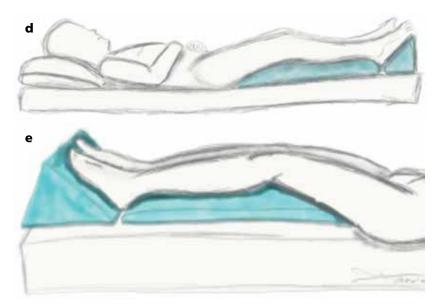
Patients can be stabilised by pillows, cushion, pieces of foam, and cotton balls. Heels must be protected or mildly elevated.

Side position at 90° is strictly forbidden as it can cause lesion of the trochanter.

Bed head must not have an angle of more than 45 degrees as it will induce shear forces. See Figure 3 for illustrations.



Figures 3a-e: Positioning



From escarre.fr (5) and Di Tommaso.\* Used with permission.

For patients in a wheelchair repositioning must be regular and frequent. It can be either active (push-up, lateral displacements) or done by a caregiver. Any movements must be done by lifting the buttocks, not sliding them, in order to avoid shear forces.

Figures 4a and b: Positioning for Patients in a Wheelchair



From escarre.fr (5). Used with permission.

At each repositioning, all bony prominence must be thoroughly protected. As soon as possible, the patient must be helped to walk and move independently.

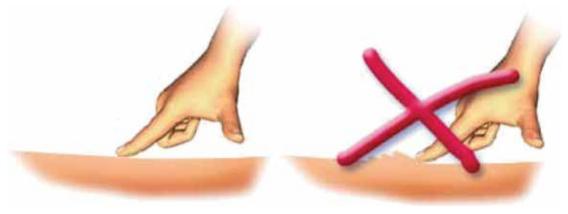
Skin must be protected from moisture (urine, feces, perspiration).

## **Pressure Redistribution**

Specialty beds are available to distribute pressure on the whole body and are recommended for patients at high risk for developing pressure injuries.

When patients are sitting, pressure can be lowered with a foam cushion but also by having the seat support the entire thigh and the chair back completely support the patient's back. Massaging the skin is PROHIBITED (Figure 5) as it can enhance microvascular lesions.

## Figure 5: Massaging of skin



From escarre.fr (5). Used with permission.

## **Tips for Pressure Injury Prevention and Treatment**

At all times, all other health issues presented by the patient must be corrected (first basic principle).

If a lesion is present, regardless of category/stage, all the prevention principles must be applied.

For Stage 1 pressure injuries, besides offloading, the skin must be protected from urine, feces, and perspiration through the gentle application of a barrier cream. It can also be protected with a hydrocolloid or a hydrocellular dressing if available.

For suspected deep tissue injury, the first step remains the implementation of prevention measures plus surveillance to determine the final ulcer stage.

For Stage 2 to 4 and unstageable pressure injuries, the prevention methods plus specific treatment (basic principles) must be applied.

As tobacco smoking has direct effects on tissue oxygenation and thrombosis, individuals at PI risk or with PI should quit smoking. (10)

#### **Key Practice Points:**

- Pressure ulcers are most frequent in bed- or chair-ridden individuals.
- Key risk factors are age, lack of mobility and no skin sensation.
- PUs result in pain, prolonged hospitalisation and increased costs.
- Just a few hours are necessary to produce a PU.
- Prevention is the primary consideration for all these individuals.
- Mobilization, offloading by positioning and improvement of the patient's general health conditions are key interventions.
- Skin must be protected from moisture (urine, feces, perspiration).
- In the case of an open wound, wound-specific treatment must be accompanied by prevention strategies.
- Smoking must be stopped as it impairs blood from normally oxygenating tissues.

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#### **Useful websites:**

- EPUAP http://www.epuap.org
- L'escarre http://www.escarre.fr
- NPIAP http://www.npiap.com
- Perse http://www.escarre-perse.com/escarres/

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# Chronic Venous Insufficiency, Venous Hypertension, and Venous (Stasis) Ulcers

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Venous (or stasis) ulcers occur around the ankle area of the leg (the gaiter area) and are the most frequent lower extremity ulcer. Venous leg ulcers can be seen in up to 3% of the population in the United States, (1) and are a significant problem worldwide. The cost of treatment of these ulcers is a major source of economic impact on the healthcare budget of any country. Additionally, many uncounted costs impair the patient's quality of life. Having a large, open, draining, foul-smelling hole in one's leg leads to social isolation, depression, and loss of self-image. Venous leg ulcers account for over two million days lost from work annually in the United States. (2) The reduced mobility and decreased productivity are sources of additional disability, requiring family or home healthcare provider involvement.

Venous ulcers are caused by venous insufficiency, which is due to damaged valves in the veins of the lower extremity. The damaged venous valves allow the blood to pool in the legs and go from the deep veins to the superficial veins when the calf muscles contract during walking. This malfunction of the calf-muscle pump and venous valve damage can result in the pressure in both the arterial and venous ends of the capillaries being elevated by over 100 mm Hg when standing. This is known as ambulatory venous hypertension. This increased venous pressure causes the signs and symptoms of chronic venous insufficiency or post-phlebitic syndrome. It has been suggested that up to 76% of venous ulcers are the result of ambulatory venous hypertension. (3)

The incidence of chronic venous insufficiency or post-phlebitic syndrome is twice as common in women as in men. Women may develop the problem at an earlier age than men (55 years compared to 61 years of age). In patients who have a history of thrombophlebitis, the chronic venous insufficiency or the post-phlebitic syndrome will develop in 20% to 50% of them within five years of having the disease. This is true even if the thrombophlebitis is appropriately treated with anticoagulation and/or thrombolytic therapy. If a venous ulcer develops, it will do so an average of seven years following the episode of thrombophlebitis; however, it may range from as short a period as two years to as long as several decades. (4)

#### **Specific Causes**

A well-known cause of damaged venous valves is thrombophlebitis, commonly occurring in the postpartum or post-operative periods. The inflammatory reac-

tion in the vein causes scarring and fibrosis of the valves, resulting in their becoming nonfunctional or incompetent. However, 20% to 40% of patients with venous insufficiency have no history of thrombophlebitis. Valve damage can occur following operations, trauma, and even diagnostic exams. Unfortunately, a number of patients with chronic venous insufficiency and valve incompetence have no cause that can be identified.

It is known that venous insufficiency is associated with the development of a venous ulcer. There are theories regarding the mechanism by which venous hypertension leads to the development of a venous ulcer, but the exact mechanism is still unclear.

An often-overlooked cause of venous ulcers may be ischemia of the tissues due to the prolonged venous hypertension. It is known that increased pressure can result in destruction of the capillaries, capillary thrombosis, and ischemia of the skin. Recent studies have shown a reduced density of the capillaries in patients with varying degrees of chronic venous hypertension. By the time an ulcer develops, the capillary density is very low and oxygen levels in the skin are very depressed. As patients are treated for the ulcers and begin healing, the capillary density and tissue oxygen levels return toward normal. (5)

The fibrin cuff theory involves the leakage of fibrinogen through endothelial pores in the small vessels that have enlarged due to the venous hypertension. The fibrinogen in the perivascular space becomes activated and is converted to fibrin, forming a thick cuff around the capillary. This fibrin cuff is thought to prevent the diffusion of oxygen and nutrients to the surrounding tissues, resulting in death of the cells and an ulcer. (6) Recent work has shown that these cuffs of fibrin may not be a continuous barrier around the dermal vessels. They may, however, act as a physiological barrier affecting oxygen and nutrient perfusion in the dermis. (7)

The "trap" theory involves macromolecules present in the tissues. With venous hypertension, the macromolecules become active and "trap" and inactivate growth factors and other cytokines essential for skin repair and maintenance of skin integrity. If the skin becomes damaged in any way, the factors necessary for repair are unavailable. The result is that the skin breaks down, causing an ulcer. (8)

The leukocyte trapping theory involves leukocytes and capillaries. Venous hypertension causes leukocytes to accumulate in capillaries and occlude them. The leukocytes then become activated and release toxic metabolites. Free radicals and proteolytic enzymes damage the capillary endothelium and spill out into the surrounding tissue, damaging cells there as well. The continued damage results in breakdown of the skin and formation of an ulcer. (9)

#### **Associations and Co-factors**

Genetic factors have been noted to increase the susceptibility of some patients to develop venous ulcers. Polymorphisms of certain genes are associated with the development of venous ulcers. (10) Abnormalities of gene expression may also account for the development of venous ulcers in other patients. (11,12)

Work is continuing on the clinical importance of these findings in patients with chronic venous ulcers.

Venous ulcers have been shown to be associated with HIV/AIDS, especially if the patient has had a history of intravenous drug abuse. (13) This would suggest that the evaluation for a venous ulcer that is not responding to therapy might need to include a test for HIV, especially if there is a history of intravenous drug abuse. None of the HIV-positive patients with venous ulcers that we have treated have had a history of intravenous drug abuse. In our experience these patients must have their primary disease treated and under control before the venous ulcer will respond to any therapy.

Interesting associations between venous ulcers and other conditions have been noted. In a study by Margolis and his associates of 44,195 postmenopausal women, the incidence of venous leg ulcers and pressure ulcers was 30-40% less if the patients were taking estrogen replacement. (14) It has been shown that estrogen therapy will decrease neutrophil chemotaxis and localisation in the wound bed, decrease the wound elastase activity, increase fibronectin and collagen in the wound, and increase the healing rate. (15) It has been suggested that treatment with intermittent topical estrogen therapy may be as effective as systemic therapy.

All of these theories and associations may play a role in the ultimate formation of the ulcer but, regardless of the mechanism, the clinical picture is the same. Patients with chronic venous insufficiency or post-phlebitic syndrome have oedema of the legs, pain in the legs (usually a tightness or bursting type of pain), dermatitis with discolouration of the skin of the lower legs, and subcutaneous fibrosis (lipodermatosclerosis). Many patients will have varicose veins.

#### Assessment

In the evaluation of any lower extremity ulcer it is most important to be sure of the aetiology, or treatment will not be successful. Ulcers of the leg can be due to vasculitis or causes other than venous hypertension. Even a skin cancer can masquerade as a lower leg ulcer. The clinician is encouraged to biopsy any wound that has been present for a long period of time, one that does not respond to therapy, or one that just does not "look right".

#### Treatment

Treatment of venous ulcers can be a challenge. The cornerstone of venous ulcer therapy involves compression therapy. Compression therapy can be done with compression bandages, compression hose (socks), or other compression devices. The reader is referred to Chapter 7: Wound Healing: The Role of Compression Therapy for the details of this treatment.

In addition to compression therapy there are other factors that must be addressed if any therapy is to be successful. There are numerous factors that predict whether or not treatment will be successful. Some of the conditions affecting the healing of a venous ulcer are found in Table 1. Among the more critical are the presence of necrotic tissue in the wound bed, non-functioning or senescent cells in the wound bed, elevated bacterial levels in the wound, and excess wound drainage and excess protease levels in the wound fluid. Debridement of the wound will help minimise the problems with necrotic tissue, non-functioning cells, and bacteria. Compression therapy can help manage the excess wound drainage and the wound fluid protease levels. There is good evidence that compression therapy and reduction of the oedema will improve the capillary density and tissue oxygen content in the wound bed. Any successful therapy of a venous ulcer must include compression therapy.

Table 1.	Predictors	of Non-healing	Venous Ulcers
TUDIC I.	1 I Calciol J	or non neuling	

Predictors
Advanced age
• Male gender
• Obesity
<ul> <li>History of congestive heart failure</li> </ul>
<ul> <li>Postmenopausal state (estrogen deficiency)</li> </ul>
• HIV infection
<ul> <li>Activated protein C resistance/factor V Leiden mutation</li> </ul>
Arterial insufficiency
<ul> <li>Senescent cells in the wound bed</li> </ul>
<ul> <li>Presence of necrotic tissue in the wound bed</li> </ul>
<ul> <li>History of deep vein thrombosis</li> </ul>
<ul> <li>History of serious leg injury</li> </ul>
<ul> <li>History of previous venous ulcer</li> </ul>
Large size of ulcer
Ulcer of long duration
<ul> <li>Venous ulcer with high exudate volume</li> </ul>
<ul> <li>Elevate proteases in the wound fluid</li> </ul>
<ul> <li>Elevated levels of bacteria in the wound bed</li> </ul>

Adapted from Taylor RJ, et al. (16)

# **In Conclusion**

As we continue to search for the best treatment for patients with venous ulcers, it is imperative to treat the patient and not just the wound. A quotation from Dr. James Peck emphasises this: "It is the individual patient who we treat, not the disease. It is the patient who recovers or dies, not the illness." (17) Treating the patient and not just the wound will mean that the patient is involved in deciding the best care for him/her. We, as caregivers, may not feel the patient has chosen wisely, but we must have the flexibility to manage the patient's wound within the best interests of the patient. We must also never forget the influence a chronic wound has on the entire family. Every therapeutic approach to the treatment of a chronic wound must be designed with the effect it will have on the entire family in mind. When we can approach a patient with a chronic wound and combine the best care for the patient with the best science available, then we shall be ready to face a world filled with wounds. (18)

#### **Key Practice Points:**

- Treatment of venous leg ulcers requires a full lower limb assessment to determine arterial status and the presence of treatable co-factors.
- The principles of wound bed preparation should be employed.
- Compression therapy is the key to resolving venous leg ulcers.

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# **Neuropathic Diabetic Foot Ulcers**

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The burden of neuropathic diabetic foot ulcers (DFUs) on the healthcare systems of developed countries is significant in terms of cost, mortality, and morbidity; the impact of DFUs on developing countries can be devastating. (1-4) The unique social determinants in developing countries provide additional obstacles that must be addressed. The application of basic evidence-based wound care principles provides a reliable methodology for successful outcomes. DFUs have been reported to have as high of a lifetime risk of 25%, with an annual incidence between 1% and 4%. (5)

A DFU is the precursor to approximately 85% of lower extremity amputations, and 7% to 20% of DFUs will progress to amputation. (6) The five-year mortality rate associated with amputation ranges from 50% to 68%, depending on amputation level. (7)

#### Diagnosis

The diagnosis of DFUs is based on history and physical examination supported by diagnostic testing. The risk factors for DFUs are listed in Table 1, and the recommended history components for a DFU are itemised in Table 2.

General or Systemic Factors	<ul> <li>Poor glycemic control</li> <li>Duration of diabetes</li> <li>Peripheral vascular disease</li> <li>Visual impairment</li> <li>Diabetic nephropathy (especially dialysis)</li> <li>Older age</li> <li>Cigarette smoking</li> </ul>
Local Factors	<ul> <li>Peripheral neuropathy</li> <li>Structural foot deformity</li> <li>Trauma and improperly fitted shoes</li> <li>Callus</li> <li>Prior history of ulceration/amputation</li> <li>Prolonged elevated pressure to specific areas</li> <li>Limited joint mobility</li> </ul>

Table 1. Risk Factors for DFUs

Adapted from Frykberg et al. (6) and Boulton et al. (8)

Table 2. Medical History for DFUs

Global History	<ul> <li>Diabetes duration</li> <li>Glycemic control</li> <li>Cardiovascular, renal and ophthalmic evaluations <ul> <li>Renal complications – dialysis, transplant</li> <li>Visual complications – visual impairment</li> </ul> </li> <li>Other comorbidities <ul> <li>Treating physicians</li> <li>Nutritional status</li> <li>Social habits – alcohol, tobacco, drugs</li> <li>Current medications</li> <li>Allergies</li> <li>Prior hospitalisations/surgeries</li> </ul> </li> </ul>
General Foot History	<ul> <li>Daily activities, including work</li> <li>Footwear</li> <li>Neuropathic symptoms <ul> <li>Positive – burning or shooting pain, electrical or sharp sensations, etc.</li> <li>Negative – numbness, feet feel dead, etc.</li> </ul> </li> <li>Vascular symptoms <ul> <li>Claudication</li> <li>Rest pain</li> <li>Non-healing ulcer</li> <li>Vascular surgery or angioplasty</li> <li>Foot deformities and calluses</li> <li>Previous foot infections</li> <li>Previous foot surgeries including amputations</li> <li>Previous DFU</li> <li>Previous trauma</li> </ul> </li> </ul>
DFU History	<ul> <li>Location</li> <li>Duration</li> <li>Inciting event or trauma</li> <li>Recurrence</li> <li>Infection</li> <li>Hospitalisation</li> <li>Wound care</li> <li>Offloading techniques</li> <li>Wound response</li> <li>Patient adherence</li> <li>Interference with wound care <ul> <li>Family</li> <li>Social</li> </ul> </li> <li>Presence of edema</li> <li>Unilateral vs. bilateral</li> <li>Charcot <ul> <li>Previous or active</li> </ul> </li> <li>Treatment</li> </ul>

Adapted from Frykberg et al. (6) and Boulton et al. (8)

The physical examination of a patient with a DFU requires a methodical, well-documented assessment that includes ulcer measurements at each office visit, with a comparative analysis performed every four weeks. Sheehan and

colleagues demonstrated the percent of change in an ulcer at four weeks is a strong predictor of healing at 12 weeks. (9) The key elements in a systematic physical examination are detailed in Table 3.

Table 3. Physical Examination for DFUs

Vascular Examination	<ul> <li>Palpation of pedal pulses, popliteal, femoral: present or absent</li> <li>Ankle-brachial index (ABI) if indicated: &gt; 0.9 = normal, &lt; 0.8 is associated with claudication, &lt; 0.4 ischemic rest pain, &gt; 1.3 vascular calcification</li> <li>Toe-brachial index</li> <li>Transcutaneous oxygen</li> <li>Skin colour changes: cyanosis, erythema, dependent rubor, elevational blanching</li> <li>Presence of edema</li> <li>Temperature gradient proximal to distal bilateral</li> <li>Integumentary changes: skin atrophy, absence of hair growth, nail dystrophy</li> </ul>
Neurological Examination	<ul> <li>10 g monofilament wire plus one of the following to diagnose loss of protective sensation (LOPS)</li> <li>One or more abnormal test indicates LOPS</li> <li>At least two normal tests indicate no LOPS</li> <li>Recommended to test plantar distal hallux, sub 1<sup>st</sup>/3<sup>rd</sup>/5<sup>th</sup> MTHs: one negative finding out of the four is diagnostic for LOPS</li> <li>Vibration using 128-Hz tuning fork: distal tip of hallux bilaterally, negative test is when the patient does not feel vibration but examiner does feel vibration</li> <li>Pinprick sensation: dorsal hallux just proximal to toenail, negative test is when the patient does not feel light pinprick on either hallux</li> <li>Ankle reflexes: total absence at rest or with reinforcement is considered a negative test</li> <li>Vibration perception threshold: biothesiometer/neurothesiometer tested over the pulp of the hallux and the detected amplitude is recorded with the mean value of three recordings used as the result, &gt; 25 V = abnormal test</li> <li>Other neurological examinations</li> <li>Patellar reflex</li> <li>Clonus</li> <li>Babinski</li> <li>Light touch</li> <li>Two-point discrimination</li> <li>Temperature perception</li> </ul>
Dermatologic Examination	<ul> <li>Skin status – colour, thickness, dryness, cracking</li> <li>Sweating</li> <li>Infection – abnormal erythema, temperature (differences locally or between limbs), interdigital fungal infection</li> <li>Ulceration</li> <li>Callus with or without subdermal bleeding, blisters, nail dystrophy, paronychia, etc.</li> </ul>

cont'd.

Musculoskeletal Examination	<ul> <li>Deformities: hammer digit syndrome, hallux valgus, hallux limitus, plantarflexed metatarsal, tailor's bunion deformity, Charcot deformity, equinus deformity, etc.</li> <li>Muscle strength testing and examination for muscle wasting (foot intrinsic muscles)</li> <li>Gait examination</li> <li>Joint range of motion</li> <li>Biomechanical examination – weight-bearing and non-weight-bearing</li> </ul>
Footwear Examination	<ul> <li>Type: athletic, dress, sandal, comfort, etc.</li> <li>Fit</li> <li>Depth of toe box</li> <li>Wear patterns</li> <li>Shoe lining</li> <li>Foreign bodies within shoes</li> <li>Orthoses</li> </ul>

Adapted from Frykberg et al. (6) and Boulton et al. (8)

## **Risk Classification**

Once a DFU has been diagnosed with a proper history and physical examination supported with appropriate diagnostic testing, the patient should be classified based on their risk level (see Table 4), and the DFU should be categorised using a zed system (see Tables 5 and 6 for two examples of commonly used DFU classification systems). Treatment plans can then be developed that reflect the level of identified risk.

Risk Category	Definition	Treatment Recommendations	Suggested Follow-up
0	No LOPS, no PAD, no deformity	<ul> <li>Patient education, including advice on appropriate footwear</li> </ul>	Annually by generalist and/or specialist
1	LOPS ± deformity	<ul> <li>Consider prescriptive or accommodative footwear</li> <li>Consider prophylactic surgery if deformity is not able to be safely accommodated in shoes</li> <li>Continue patient education</li> </ul>	Every 3–6 months by generalist and/or specialist
2	PAD ± LOPS	<ul> <li>Consider prescriptive or accommodative footwear</li> <li>Consider vascular consultation</li> </ul>	Every 2–3 months by specialist
3	History of ulcer or amputation	<ul> <li>Same as risk category 1</li> <li>Consider vascular consultation if peripheral artery disease is present</li> </ul>	Every 1–2 months by specialist

Table 4. Risk Classification Based on Comprehensive Examination

From Boulton et al. (8)

Table 5. Wagner Classification of DFUs

Grade	Lesion
0	No open lesion, may have deformity or callus, pre-ulcerative lesion
1	Superficial ulcer
2	Deep ulcer to tendon or joint capsule
3	Deep ulcer with abscess, osteomyelitis, or joint sepsis
4	Local gangrene forefoot or heel
5	Gangrene of the entire foot

From Frykberg et al. (6)

Table 6. University of Texas Health Science Center San Antonio Diabetic Wound Classification System

	Grade 0	Grade I	Grade II	Grade III
Stage A	Pre- or post- ulcerative lesion completely epithelized	Superficial wound, not involving tendon, capsule, or bone	Wound penetrating to joint or capsule	Wound penetrating to bone or joint
Stage B	With infection	With infection	With infection	With infection
Stage C	With ischemia	With ischemia	With ischemia	With ischemia
Stage D	With infection and ischemia	With infection and ischemia	With infection and ischemia	With infection and ischemia

From Lavery et al. (10)

#### Management

An evidence-based approach to the treatment of DFUs applies equally to developed and developing countries, allowing adaptation to each country's unique circumstance. The best method to treat a DFU is to prevent if from happening. The American Diabetes Association (ADA) issues position statements on standard of care for preventative diabetic foot care using an evidence-based grading system for recommendations. The grading system consists of Grades A, B, C, and E, ranging from highest levels of evidence to expert consensus or clinical experience. (11) The ADA foot care recommendations for preventative care are shown in Table 7.

#### Table 7. ADA Foot Care Recommendations with Grade

Recommendations	Grade
<ul> <li>For all patients with diabetes, perform an annual comprehensive foot examination to identify risk factors predictive of ulcers and amputations.</li> <li>Inspection</li> <li>Assessment of pulses</li> <li>Testing for LOPS – 10 g monofilament wire plus one of the following: vibration exam with a 128-Hz tuning fork, pinprick sensation, ankle reflexes, vibration perception threshold</li> </ul>	Grade B
Provide general foot care education for patients with diabetes.	Grade B
A multidisciplinary approach is recommended for individuals with foot ulcers and high-risk feet, especially those with a history of prior ulcer or amputation.	Grade B
Refer patients who smoke, have LOPS and structural abnormalities, or have history of prior lower-extremity complications to foot care specialists for ongoing preventive care and lifelong surveillance.	Grade C
Screen for PAD. • Claudication history • Assessment of pedal pulses • Possible ankle-brachial index	Grade C
Refer patients with significant claudication or a positive ABI for further vascular assessment and consider exercise, medications, and surgical options.	Grade C

Adapted from American Diabetes Association (11)

Developing countries face challenging circumstances in the treatment of DFUs that are unique for each country. For example, many developing countries lack reconstructive vascular surgery, necessitating an adaptable outcome metric that takes these limitations into account. Wrobel et al. described a ratio of high-level (transtibial or transfemoral) amputations to low-level (digital, ray, tarnsmetatarsal and Choparts) amputations. (12) Rogers and Bevilacqua used this outcome metric to evaluate a six-step amputation prevention program for DFUs, demonstrating a reduction of diabetic amputations by 72% and an eight-fold decrease in the high-low amputation ratio (see Table 8). (13)

Table 8. Six-step Protocol for Amputation Prevention

Process	Components
1. Identification and management of infection	<ul> <li>Diagnosis based on clinical signs and symptoms</li> <li>Treatment includes antibiotics</li> <li>Consideration of bacterial resistance</li> </ul>
2. Identification and management of ischemia	<ul> <li>Non-invasive vascular tests for macrovascular and microvascular flow</li> <li>Referral to vascular surgery for evidence of ischemia</li> </ul>
3. Offloading pressure (needs to be maintained throughout wound healing)	<ul> <li>Use of total contact cast, instant total contact cast, diabetic cast walker, wheelchair, crutches, bed rest</li> <li>Surgical correction of deformity, pressure foci</li> </ul>
4. Debridement of wound	<ul> <li>Preference given to surgical debridement, including hydrosurgical scalpels</li> <li>Enzymatic, mechanical, maggots</li> </ul>
5. Promotion of granulation tissue	<ul> <li>Use of negative-pressure wound therapy, growth factors, marrow-derived stem cell transplantation</li> </ul>
6. Closure of wound	<ul> <li>Split-thickness skin graft, bioengineered skin substitutes, surgical flaps</li> </ul>

Reprinted with permission from American Podiatric Medical Association (13)

Cychosz et al. evaluated non-operative and operative treatments for DFUs based on level of evidence and graded each treatment with either A, B, C or I (highest level of evidence to insufficient level of evidence to be recommended). (14) These recommendations for treatment can be used to implement the six-step program above. See Tables 9 and 10.

Intervention	Considerations/Comments	Level of Evidence
Custom Shoes and Footwear Modification	Diabetic shoe with wider, deeper, rounder toe box and custom orthotics; grade based on preventing recurrent ulceration instead of initial ulceration; compliance is critical	
Total Contact Casting	Safe and effective treatment for DFUs, may be limited by practicality and ease of use, especially in developing countries; lower cost alternatives such as football dressings may be viable alternatives (15)	
Debridement and Specialised Dressings	Converts chronic wound into acute wound, allowing normal stages of wound healing; lacking enough high-quality studies to receive a grade	Grade I
Hyperbaric Oxygen Therapy	Improves oxygenation to assist healing by increasing angiogenesis and fibroblast production, reducing inflammation, stimulating leukocyte activity; lacking supportive evidence for efficacy, cost-effectiveness, and indications; rarely an option in developing countries	Grade I
Negative Pressure Wound Therapy (NPWT)	Limited evidence reveals potential advantages for DFUs; disadvantages include costs, dressing changes required 2–3 times per week, potential non-weight-bearing depending on placement; possible option in developing countries especially with some of the newer, less costly NPWT units	Grade I
Advanced Biological Therapy	Specific advanced biological therapies have high-quality support for DFUs, but due to the large variety of products not all have been evaluated with equal levels of research; cost effectiveness and clinical significance are yet to be determined; application for developing countries is most likely limited due to cost	Grade A
Electrophysical Therapy	Ultrasound, light therapies, and electrical stimulation may aid DFU healing via wound perfusion and cell migration; evidence for this newer treatment is limited, with varied results; application for developing countries is most likely limited	Grade I

From Cychosz et al. (14)

Intervention	Considerations/Comments	Level of Evidence
Tendo-Achilles Lengthening (TAL)	Effective for forefoot and midfoot DFUs in patients with equinus deformity; definition of equinus is best described by DiGiovanni et al. as < 5° ankle joint dorsiflexion with the knee extended for gastrocnemius equinus and <10° with knee flexed for gastrosoleal equinus ( <i>16</i> ); care must be taken to prevent over-lengthening, resulting in a calcaneal gait and potential heel ulcerations; this is applicable to developing countries due to minimal instrumentation for a triple hemi-section TAL	Grade A
Gastrocnemius Recession	Indicated for forefoot or midfoot DFUs associated with gastrocnemius equinus as described above; more controlled lengthening with less risk of over-lengthening compared to TAL; current evidence is limited by small sample size and lack of controls; no consensus on technique (author's preference is the Baumann intramuscular lengthening)	Grade B
Toe Flexor Tenotomy	Effective for distal toe DFUs, indicated for flexible hammer toe, claw toe, and mallet toe deformities; used for both preventative and therapeutic treatment; percutaneous procedure with few complications	Grade B
Plantar Fascia Release	Indicated for plantar DFUs at distal tip of digits, interphalangeal joint, and metatarsal phalangeal joint; selective plantar fascia release; limited evidence	Grade I
Correction of Static Forefoot Deformity	Exostectomy, arthrodesis, and osteotomy; limited supporting evidence	Grade I

From Cychosz et al. (14)

# Conclusion

Despite the unique social determinants of health for developing countries, DFUs can be treated effectively with an evidence-based systematic approach producing outcomes similar to the treatment of DFUs in developed countries.

# **Key Practice Points:**

- A multidisciplinary approach is recommended for individuals with foot ulcers and high-risk feet, especially those with a history of prior ulcer or amputation.
- Interventions should be based on risk.
- Key interventions include screening, vascular assessment, appropriate debridement of nonviable tissue, management of infection, and plantar pressure redistribution.

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# Lymphatic Filariasis

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Historically, reports of lymphatic filariasis (LF) date back almost 4000 years. (1) Artifacts from ancient Egypt and the Nok civilization in West Africa show possible symptoms suggestive of LF. The first substantiated reference to the disease was noted in ancient Greek literature where scholars differentiated LF symptoms from those of leprosy. The first documented symptoms were reported in the 16<sup>th</sup> century by Jan Huyghen van Linschoten during an exploration of Goa. He noted that descendants of those who "had killed St. Thomas ... were all born with a lower limb, swollen from the knee down, similar to the leg of an elephant ..." Therefore, lymphatic filariasis became known as the "curse of St. Thomas". It was not until 1877, when Patrick Manson, a British physician, determined that mosquitoes were the vectors of transmission. This was the first time an insect had been implicated in the active transmission of an agent of any human or animal disease.

#### **Background/Introduction**

Lymphatic filariasis has been rated as the leading cause of permanent disability worldwide. (2) Lymphatic filariasis is a parasitic disease caused by nematodes of the *Filarioidea* type. The parasites are spread by the bites of infected mosquitos (multiple species), which are the sole vector of transmission. There are three types of nematodes that cause the disease: *Wucherieria bancrofti* (most common), *Brugia malayi*, and *Brugia timori*. The parasites have an affinity for the human lymphatic system, where they live, breed and die, resulting in damage to the lymphatic system over time. The disease manifests as a spectrum of clinical presentations, including hydrocele (scrotum), lymphoedema/elephantiasis of the extremities, and sub-clinical lymphatic damage even in young children. About 1.4 billion people are at risk of the disease in 73 countries throughout the tropics and sub-tropics of Asia, Africa, the Western Pacific, parts of the Caribbean and South America. (3) With more than 120 million people infected with LF, lymphatic filariasis is now classified among the neglected tropical diseases.

Repeated mosquito bites over months to years in endemic areas are needed to contract LF. When a mosquito bites a person who has LF, microscopic worms or filariae, circulating in the person's blood enter and infect the mosquito. Filariae go through a multiple-stage development cycle within the mosquito. During a blood meal, the larvae enter the human host at the puncture site and migrate to the lymphatic system. Once in the lymphatic system, a few filariae mature and develop into adult worms whose life cycle is about five to seven years. The adult worms mate and release millions of microfilariae into the blood, which are sub-sequently ingested by mosquitos, continuing the transmission cycle.

Most infected individuals remain asymptomatic and never develop clinical symptoms despite damage to the lymphatic system. Those who do develop clinical symptoms manifest lymphoedema caused by impairments to the collecting lymphatic system from the parasites. These impairments, such as gross dilatation of the vessels rather than blockage, combined with the loss of skin barrier function, allow bacteria and soil irritants to enter the skin, damaging the initial superficial lymphatics in the upper dermis. Repeated infections and increased vascular permeability from inflammation are often exacerbated by venous overload, which can lead to more advanced stages or exacerbations of lymphoedema. Such recurrent infections, with pain and swelling over the lymph nodes, especially in the groin, were at one time thought to be due to a reaction to the filarial worm. Now, these acute attacks are attributed to bacteria such as Streptococcus entering opportunistically due to disruption of the skin barrier function. The resulting lymphoedema is a protein-rich fluid collection in the interstitial tissues that can lead to significant swelling of the extremities, breasts, and genitalia. Unlike the transudate associated with cardiac failure, the protein-rich fluid with lymphoedema encourages the fibroblasts to lay down an excess of collagen. This, in turn, results in significant fibrotic changes of the skin. Usually the lower limbs are involved, either unilaterally or sometimes bilaterally, in which case the swelling tends to be asymmetrical. The upper limbs, male genitalia (hydrocele) and rarely breasts in females may also be affected. The swelling is not uniform, and there are a variety of protuberances and deep crevasses in the skin. Repeated bacterial infections can lead to significant progressive changes in the skin and subcutaneous tissues. *Elephantiasis* is an ancient term describing the late hardening and thickening of the tissues and often-bizarre surface changes. with some areas of hyperkeratosis and other areas described as "mossy", while others may be smooth. (4)

There has been a long debate about grading and staging of lymphoedema. (5) Readers looking for updates on current thinking should examine publications on this topic by The International Society of Lymphology or The International Lymphedema Framework.

As described in Brazil, lymphoedema of the limbs due to LF is graded according to five or seven clinical presentations. Some who are used to more common patterns seen in Europe, usually due to cancer and its treatments, say this Brazilian staging is too complex. Others, especially those using investigative techniques picking up changes in lymph flow or tissue fluid accumulation before clinical signs develop, want a grading system that zes preclinical changes. One area of a limb may show a stage that is unlike that of another affected part of the body. All would agree that it is useful to classify the early stage of reversibility of swelling occurring with nocturnal elevation compared to those with no overnight changes. The great variety of physical signs developing in late stages can be recorded separately or simply as "late stage". (3–5) A minimal appearance of mossy foot, fibrotic nodules, fissuring, crevasses, and gross hyperkeratosis is not always confined to late stages, nor is it related to degree of edema.

# International Society of Lymphology (ISL) Staging System

# Grade 0

• Lymph flow is impaired and tissue fluid is accumulating without clinical signs or symptoms

#### Grade I

- Pitting edema, reversible on elevation of the affected limb
- Skin folds absent
- Normal, smooth appearance of skin

# Grade II

- Pitting or non-pitting edema, does not reverse on elevation of the affected limb
- Significant edema of the subcutaneous tissue
- Swelling not reversible at night

There may be early but mild signs of the grosser skin changes observed in grade III

# Grade III

- Non-pitting edema that is not reversible, with thickening of the skin
- Hypertrophy of subcutaneous adipose tissue
- Variable to severe nodularity, hyperkeratosis, and deep folds or crevasses

In advanced stages of lymphoedema, the skin is thickened and often presents with deep skin folds, frequently with hyperkeratosis, black pigmentation, nodules, warty growth, intertrigo, and maceration in the webs of toes or chronic non-healing ulcers. (4) Fungal infections in the interdigital region and in deep folds are a common finding in advanced lymphoedema. The swelling may be so significant that the patient is incapacitated, requiring help even for personal needs.

# Diagnosis

Lymphatic filariasis is typically diagnosed by a blood test using thick or thin blood smears stained with Giemsa or hematoxylin and eosin. A blood test can identify active infection by determining the presence of microfilariae. The microfilariae that cause LF circulate in the peripheral blood stream of the human host at night, typically between the hours of 10 p.m. and 2 a.m. This phenomenon is known as nocturnal periodicity.

Another option is the use of antigen detection using an immunoassay for circulating filarial antigens. Unlike microfilariae with nocturnal periodicity, filarial antigens can be detected in blood samples collected at any time of day. A rapid format immunochromatographic test (ICT) has been shown to be a useful and sensitive tool for the detection of the *Wucherieria bancrofti* antigen and is being widely used in LF elimination programs worldwide.

Over time these tests become negative, and it may be difficult to identify cause in patients who have lymphoedema long after the initial exposure to the mosquito. Additional information can be found in Chapter 13: Podoconiosis and Paskett et al. (6) A key consideration to remember in tropical climates is that filariasis is not the only cause of lymphoedema. In the elderly, immobility, heart failure, and other degenerative problems can also be determinants of lymphoedema.

#### Management

Management of LF consists of several key components broadly divided into:

- 1. breaking transmission with the mosquito and eradication using microfilaricidal agents
- 2. morbidity control through prevention and treatment of acute dermato-lymphangio-adenitis (ADLA) and bacterial infections such as cellulitis
- 3. interventions for lymphoedema for those with clinical signs resulting from damage to the lymphatic system. (7)

The goal is to prevent new cases of LF from developing. This is achieved through a two-fold approach involving education about LF for people in endemic areas on topics such as mode of transmission, methods of mosquito control, and how to provide better sanitation—all in an effort to prevent the transmission of LF. Coupled with education is mass drug administration (MDA), which involves giving entire communities medicine that kills the microfilariae. At the present time, dual-drug regimens of albendazole and either diethylcarbamazine (DEC) or ivermectin are utilised for MDA efforts. Typically, these medications are given annually for five years, omitting children under the age of five and pregnant women. DEC can also be added to salt used by the entire population for one to two years to interrupt transmission. (8) For individuals with active infection from W. bancrofti, B. Malayi, and B. timori, DEC is the drug of choice. DEC is an effective microfilaricidal agent; however it only kills about 50% of adult worms. (5) It is important to note that the anti-parasitic medications cannot reverse the lymphatic damage, nor do these medications have a role in the management of ADLA or lymphoedema.

#### **Bacterial Infections**

Patients with LF are at risk of bacterial infection entering through the many breaks in the skin, which can lead to further disability and exacerbation of lymphoedema. Prevention of acute attacks is paramount, and the most universally accepted method of prevention is proper skin hygiene and foot care. The hygiene and foot-care regimen to prevent these infections consists of: washing the affected area (with special attention to deep skin folds and interdigital spaces) with soap and water and gently drying with a clean cloth, proper nail care, promptly treating any infections of the skin using antibiotic and antifungal ointments, and consistent use of comfortable foot wear. If an attack does occur, most can be managed with bed rest, elevation of the involved limb, and simple medications. Moderate or severe attacks require oral or parenteral antibiotics coupled with analgesics and anti-inflammatory agents. Penicillin, doxycycline, ampicillin, amoxicillin, or cotrimoxazole may be given until infection resolves. (5)

# **Complete Decongestive Therapy (CDT)**

Lymphoedema resulting from LF cannot be cured, but it can be managed successfully. The gold standard for managing lymphoedema was developed in Europe and is named Complete Decongestive Therapy (CDT). CDT includes patient education, skin and nail care, manual lymph drainage, compression, and basic exercises. Even in resource-challenged nations, modified CDT can be implemented effectively.

Modified CDT to manage lymphoedema involves: (1, 3, 6, 8)

- Meticulous skin and nail care maintained by washing affected areas twice daily with soap and clean, tepid water followed by careful drying of the skin and interdigital spaces
- Avoiding immobility and dependency by encouraging a full range of body movements and use of the affected limb throughout the day, with elevation of the affected limb(s) during the night. (Please refer to Chapter 19, where a description of activities in Kerala includes yoga)
- Education and training of self-manual lymph drainage and diaphragmatic breathing
- Implementation of basic compression with bandages or hosiery to contain and maintain fluid reduction
- Prompt attention to any skin lesions or infections through the use of medicated creams (antibacterial or antifungal)
- Comfortable shoes that protect the feet

As the world nears eradication of LF, it is important to remember that lymphoedema, regardless of cause, cannot be cured, but it can be successfully managed. For the millions of individuals worldwide with lymphoedema, modified CDT and approaches to management adapted to the local environment can provide a renewed sense of hope and reduced social stigma from this debilitating disease.

#### **Key Practice Points:**

- Prevention strategies must focus on breaking the transmission cycle through vector control and mass drug administration.
- Morbidity must be managed through meticulous skin care, exercise, compression therapy, and an adapted form of manual lymphatic drainage.

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# Podoconiosis

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# **Definition and Aetiology**

Podoconiosis, a term coined by Ernest Price, (1) is derived from the Greek terms podos and konos, meaning foot and dust. It is a non-infectious, familial, geochemical lymphoedema of the lower legs caused by exposure of feet to irritant red clay soils of volcanic areas with an altitude of over 1000 metres, annual rainfall above 1000 mm, and average temperatures of ~20 deg C. (2) Cultivated by barefoot subsistence farmers, the alkaline clay is slippery when wet and very adhesive if allowed to dry on the skin. (3) Mineral particles are absorbed through the skin and taken up by the lymphatics of the foot and lower leg, inducing an inflammatory response in lymphatic vessels that leads to fibrosis and obstruction of the vessel lumen. (4) Early electron microscopy showed the presence of silicates in femoral lymph nodes harvested from Ethiopians with and without podoconiosis. (5) The inflammatory reaction to the colloid size minerals has been linked to a genetic predisposition. (6, 7)

# **Prevalence and Burden**

Podoconiosis (non-filarial elephantiasis) was identified by the World Health Organization as a neglected tropical disease in 2011, and elimination is now on the global health agenda. (8) Globally, it is estimated that there are at least four million people with podoconiosis, and the disease has been reported in 32 countries, 18 from the African Region, 11 from Latin America and three from Asia. (9) In Ethiopia alone, the estimated national annual cost of podoconiosis is US\$ 200 million, with 45% of economically productive time lost to morbidity associated with the disease. (10)

# Age and Sex Distribution

The onset of podoconiosis is common in the second and third decades of life, with the disease being common up to the sixth decade. (6) The prevalence in the first decade is almost zero. A study from Uganda reported no cases among those <10 years old, (11) and in southern Ethiopia, only 6.8% of the patients were  $\leq$ 15 years old. (12)

Reports on gender ratio in podoconiosis vary. A study conducted in southern Ethiopia found a male:female ratio among those with podoconiosis of 1:0.98. This was not significantly different from the zonal gender ratio (1:1.02). (12) A study conducted in Northern Ethiopia recorded a gender ratio of 0.98:1, (13) two others in Western Ethiopia documented gender ratios of 0.7:1 (14) and 0.5:1, (15) and another in Central Ethiopia demonstrated a gender ratio of 1.2:1. (16) In Cameroon, a gender ratio of 0.5:1 was recorded, (17) while that in Uganda was 1:1. (11) Some studies have suggested that mild forms of the disease are more common among women than men, possibly due to differences in treatment-seeking behaviour. (16)

# Stigma

Podoconiosis is highly stigmatized, which often makes early intervention difficult. A standardised podoconiosis stigma assessment scale (18) revealed that over half of patients considered suicide in response to discrimination and prejudice, particularly in interpersonal interactions. (19) Forced divorce, insults, and exclusion at social events were commonly reported. The Kessler-10 scale was used to document a high burden of mental distress among people with podoconiosis, (20) and more recent use of the PHQ-9 scale demonstrated high levels of depression. (21) A study in 2009 revealed that around half of health professionals in Southern Ethiopia incorrectly considered podoconiosis to be an infectious disease and were afraid of acquiring it while providing care, and 100% held one or more stigmatizing attitudes towards people with podoconiosis. (22)

# **Differential Diagnosis**

The common differential diagnoses of podoconiosis are filarial elephantiasis, lymphoedema of systemic disease and leprotic lymphoedema. Although there are point-of-care diagnostic tests for lymphatic filariasis infection, these are not very sensitive in established filarial infection among advanced cases. The differentiation of podoconiosis from filarial elephantiasis uses a panel approach, including clinical history, physical examination, antigen and antibody tests. The swelling of podoconiosis starts in the foot and progresses upwards, (9) where-as the swelling in filarial elephantiasis starts elsewhere in the leg. Podoconiosis lymphoedema is bilateral, asymmetric, usually confined to below the level of the knees, and unlikely to involve the groin. (23) In contrast, filarial elephantiasis is commonly unilateral and extends above the knee, usually with groin involvement. In addition to the clinical history and physical examination, an antigen-based ICT test can also help to further distinguish between the two lymphoedemas, although the majority of patients with filarial elephantiasis are also negative for the antigen-based test.

# **Clinical Presentation**

Early symptoms of podoconiosis include burning and itching of the dorsum of the distal foot or the first or second web spaces, particularly after a long day in the field. (24) Repetitive scratching may lead to entry portals for infection, which exacerbates the condition. (1) Initially, foot and lower leg oedema increases throughout the day and resolves with elevation overnight. The swelling may be pitting and may cause splaying of the forefoot and sausage-like appearance of the toes; plantar foot oedema may lift the toes off the ground. (24) A mismatched and asymmetrical enlargement of the second toe on the affected foot is a common finding.

As the lymphoedema progresses, the volume of the leg can reach massive proportions, with large lymphoedematous lobes. Oedematous tissues are described as "water bag", "rubbery", or "woody", corresponding to the level of fibrosis. Price speculated that the type of oedema and degree of fibrotic reaction is genetically determined, and the degree of fibrosis in lymph nodes has been found to parallel that of the leg. (25) According to him, the texture of the oedema is established at the onset and persists throughout the disease, unless it is complicated by secondary bacterial infections. (25) The fibrosis and thickening of the subcutaneous tissues and dermis can proliferate to the extent that web spaces between the toes are completely engulfed, and ankle and foot mobility is lost, leaving the limb looking like that of an elephant (hence "elephantiasis"). (26, 27)

Dermatological changes associated with podoconiosis are similar to other forms of lymphoedema, including thickening of the skin, a positive Stemmer's sign, lichenification, hyperkeratosis, and papillomatosis which ranges from cobblestone appearing skin to wart-like projections, nodules, or knobs. Stratum corneum hydration has been shown to be significantly lower in patients with podoconiosis than in controls. (28) Verrucous hyperplasia results in upward projections of the epithelium that give a moss-like appearance to the skin ("mossy foot"); it is not associated with known papilloma viruses. (26)

According to a 2012 survey, 97.9% of 1704 cases had mossy lesions. (13) Mossy lesions can present in three variations (author's observation):

- Fluid-filled vesicular projections with fragile walls that are easily disrupted, resulting in portals for secondary infections. The vesicles appear translucent when illuminated with a small flashlight.
- Dry, coarse mossy-appearing skin which tends to absorb and hold moisture similar to a diabetic callus.
- Tightly packed projections of the epithelium resembling elongated versions of filiform papillae on the surface of the tongue.

A simple staging system (see Table 1) taking into account the presence and position of knobs, bumps, and moss has been developed for podoconiosis with good inter-observer agreement and repeatability (29).

Table 1: Podoconiosis Staging Sheet

Stage 1	Swelling reversible overnight
Stage 2	Below-knee swelling that is not completely reversible overnight; if present, knobs/bumps are below the ankle ONLY
Stage 3	Below-knee swelling that is not completely reversible overnight; knobs/bumps present above the ankle
Stage 4	Above-knee swelling that is not completely reversible overnight; knobs/bumps present at any location
Stage 5	Joint fixation; swelling at any place in the foot or leg
Largest below-knee circumference	(in centimetres)
Presence of mossy changes	M+
Absence of mossy changes	M-
Example	Stage 2, M+, 48 cm

From Tekola et al. (29)

Complications of podoconiosis include susceptibility to recurrent and chronic infections. Leaking of lymph fluid, and skin-on-skin contact between oedematous toes or deep folds, creates the ideal environment for moisture-associated lesions and secondary fungal and bacterial infections. A range of studies has documented that 77.4–97% of patients have experienced acute dermatolymphangioadenitis (ADLA) at least once per year. (13–15) These attacks necessitate 4–5 days off work each episode. (13, 15) Acute attacks result in a painful, red-dened, swollen limb, and malaise and fever. The inflammatory process accelerates fibrosis formation in the affected areas; antibiotics and pain medication may be necessary. More than half (54%) of patients with podoconiosis also develop open wounds. (13) Many of these wounds become chronic. Healed areas result in scarring and depigmentation.

# **Hygiene and Footwear**

Nationwide mapping in Ethiopia has identified 34.9 million people living in at-risk areas. (2, 30) The best intervention against podoconiosis is prevention. Community-based education and screening are thought to help reduce local stigma and identify patients in early stages of the disease. Avoidance of the chronic irritant triggering lymphoedema is achieved through wearing socks and enclosed shoes, use of floor coverings, and washing the feet at least daily with soap and clean water.

Podoconiosis can be eliminated from countries by wearing shoes. However, in rural, undeveloped areas, solving the footwear problem is challenging. A 2013 study found that, although norms were changing so that going barefoot in public was considered "shameful", limited resources prevent families from buying shoes for every member. (31) Affordable footwear is generally not enclosed and therefore inadequate protection from the soil. Cheap, plastic footwear is common but becomes sweaty and is slippery on clay during the rainy season. Considered a luxury, shoes are reserved for special occasions, not everyday use. (31)

Footwear is also reported to be unsuitable for local agricultural practices. The sticky clay builds up on the outside of the shoes and becomes very heavy; dry clay that gets into shoes is painful. (31, 32) Mass-produced, moulded rubber shoes distributed for children in high-risk families may become associated with podoconiosis, resulting in stigmatization. Many patients with podoconiosis are unable to fit into standard footwear, so custom leather footwear must be made locally.

#### Treatment

In Ethiopia, daily soaking for 10 minutes in an antiseptic solution, such as salt, dilute bleach or potassium permanganate, is practised. This is particularly effective to clean the crevices within mossy lesions, in skin creases, and between toes. However, prolonged soaking of dry mossy lesions, particularly during rainy seasons, can macerate the tissue and increase its vulnerability to infections. Potassium permanganate is not available in all areas, and dilute bleach can dry out the skin and impair wound healing. A recommended alternate is locally available eucalyptus leaf soak.

Filtration systems or PUR packets can transform contaminated water into clean drinking water that is also suitable for hygienic care and soaking. Special attention must be paid to cleaning and drying between the toes with soft strips of cloth. Unscented and pH-neutral soap is best for patients with lymphoedema. Groups in Ethiopia train patients to make soap using local neem extract, and neem oil can be used as an emollient to soften dry skin and prevent cracking. Antifungal ointments help prevent and treat fungal infections between toes. Antifungal powders work best for crevices within mossy lesions and between folds. Patients using a simple lymphoedema treatment regimen such as this, who were followed for one year, showed significant clinical improvements and positive changes in quality of life. (33)

A pragmatic randomised controlled trial of a simple lymphoedema management package comprising foot hygiene, ointment, bandaging where indicated, exercise, elevation, and use of socks and shoes demonstrated a significant decrease in incidence of ADLA among patients randomised to this intervention. (34) Another trial demonstrated improvements in skin barrier function with the addition of glycerol to reduced soaking water volumes. (35)

Comprehensive treatment for podoconiosis is the same as treatment for other forms of lymphoedema, and implementation of modified complete decongestive therapy (CDT) is possible even in resource-limited settings (author experience). Central decongestion of the lymphatic system and opening of alternate and redundant pathways to facilitate lymphatic drainage of the involved areas can be achieved through group self-manual lymphatic drainage and deep breathing exercises. Range of motion exercises can help preserve ankle and foot mobility. Although short-stretch bandages are optimal, elastic bandages are more readily available in resource-limited areas and can be used judiciously to reduce limb volume in "water bag" forms of podoconiosis, in combination with limb elevation during the night and as much as possible during the day. More fibrotic presentations of podoconiosis, described as "woody", also respond well to more advanced clinical treatments, such as manual lymphatic drainage therapy combined with fibrosis techniques, and multi-layered, shortstretch compression bandaging with strategic placement of foam inserts (author experience). Over time, this can result in limb volume reduction, normalization of dermatological changes, reversal of fibrosis, and regeneration of the lymphatics. Figure 1 shows a fibrotic "woody" area on the anterior ankle of a young man who was excluded from local treatment because it was thought nothing could be done for him. One can see the dramatic difference in pliability of the tissue after 17 hours of compression with multi-layered, short-stretch bandaging with custom foam inserts. Figure 2 shows the reduction of oedema and improved pliability of the tissues after 10 days of short-stretch compression bandaging with foam inserts, and manual lymphatic drainage using fibrosis techniques.

Figure 1: Changes in Pliability of Tissue after 17 Hours of Compression



Before treatment. © Robyn Bjork



After 17 hrs compression using multi-layered, short-stretch bandaging with foam insert over anterior ankle lobe. © Robyn Bjork

#### Figure 2: Reduction of Oedema after 10 days of Compression



Before treatment. © Robyn Bjork



Volume reduction and softening of tissues after 10 days of multi-layered, short-stretch bandaging with foam inserts, and manual lymphatic drainage therapy. © Robyn Bjork

Gortex overboots can be used to protect bandaging systems. Local bandage covers can be constructed using any waterproof, breathable material coupled with gluing or spraying a sole onto it. Short-stretch bandages are expensive and generally unavailable in endemic areas; however, strips of fabrics with similar short-stretch properties could be explored. Shredded foam can be purchased through local foam manufacturers, or locally available foam sleeping pads can be used to create it. Complex bandaging systems should be used for short-term, intensive treatment (1–3 weeks), followed by transitioning into long-term use of short-stretch wraps with hook-and-loop closures, compression garments, or custom, lace-up knee-high boots that serve as both footwear and compression.

Compression bandaging is paramount to effective wound healing in patients with podoconiosis. Potable water combined with very small amounts of sea salt can be used to create normal saline to clean wounds, and periwound skin can be protected with locally found zinc oxide paste. In Ethiopia, indigenous honey impregnated in hygienic cotton is readily available and an excellent natural resource to use as a primary dressing. Additional layers of hygienic cotton can be used to absorb drainage, and compression effectively applied over the top (author experience).

Judicious surgical excision of nodules (nodulectomy) has been successfully practised in Ethiopia and has enabled patients to fit into custom footwear. (26, 27) Although treatment is steadily becoming available in more endemic areas,

the vast majority of patients globally are still untreated. This is a result of misconceptions and stigma related to the disease, lack of awareness among policy makers and the very practical challenges of patient access. (36, 37)

## Summary

Podoconiosis is a preventable and treatable form of lymphoedema. Although challenges exist in resource-limited nations, effective management can be achieved through education, stigma reduction, prevention, early intervention, community programs, and skilled medical care.

## **Key Practice Points:**

- The best intervention for podoconiosis is prevention through community education, screening, elimination of the chronic irritants triggering lymphoedema, wearing socks and enclosed shoes, use of floor coverings, and washing feet at least daily.
- Treatment involves meticulous skin care, compression bandaging and modified manual lymphatic drainage.

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# **Buruli Ulcer**

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#### Introduction

Buruli ulcer (BU) is a disease characterised by necrotizing infections of skin and soft tissue caused by *Mycobacterium ulcerans*. (1) BU has been reported in 33 countries worldwide, with more than 6000 cases reported each year. The greatest burden of disease is found in West and Central Africa. However, cases are also reported from South America, Asia, and the Pacific. It is predominantly found in tropical and subtropical climates, apart from southeastern Australia and Japan. In endemic areas, the disease is highly focal and is usually associated with wetlands, especially those with slow-flowing or stagnant waters. However, despite extensive research the environmental reservoir and mode of transmission remain unknown.

The disease pathogenesis is mediated through the production of a potent exotoxin called mycolactone. It is toxic to tissues and impairs local and systemic immune responses to the infection. Infection begins in the skin and subcutaneous tissue and can spread to contiguous structures such as tendon or bone or, less frequently, to more distant sites by hematogenous spread. The incubation period has been estimated to be a median of 4.5 months (range one to nine months).

In Africa Buruli ulcer affects mainly children under 15 years of age but can occur in all age groups, is found in males and females equally, and commonly affects those living in remote areas with limited access to healthcare. If left untreated, lesions usually progress and can lead to severe tissue destruction, limb deformity, and a high level of long-term disability. The majority of people affected are overtly immunocompetent; however the HIV prevalence in patients with BU disease is increased and BU disease is more severe in those co-infected with HIV.

#### **Clinical Features**

*M. ulcerans* presents as two different clinical forms: ulcerative and non-ulcerative lesions. (2) Non-ulcerative lesions include those classified as papules, a plaque, nodules, and edematous lesions. The ulcerative form is classically a deep ulcer extending into the subcutaneous fatty tissue with undermined edges. The floor of the ulcer may have a white, cotton-wool-like or yellow appearance due to necrotic tissue or fat. Untreated ulcers are painless, unless there is secondary bacterial infection. There is usually no local lymphadenopathy.

A papule is a painless, raised skin lesion less than 1 cm in diameter. The surrounding skin is reddened and may be confused with an insect bite. A nodule is a lesion less than 3 cm in diameter that extends from the skin into the subcutaneous tissue. It is usually firm and painless but may be itchy, and the surrounding skin may be discoloured in comparison with adjacent areas. A plaque is a firm, painless, elevated lesion larger than 3 cm in diameter with ill-defined edges. The skin over the lesion may be reddened or otherwise discoloured. The oedematous form is a diffuse, extensive, usually non-pitting swelling. The affected area has ill-defined margins, is firm, and involves part or all of a limb or other part of the body. All non-ulcerative forms may progress to ulcers after a variable time, which may be as short as four weeks. Most lesions are ulcerative, but a significant proportion are non-ulcerative at diagnosis.

Osteomyelitis can occur, usually as a complication of severe cases, with an estimated frequency of 6–10% of cases in Africa. It usually results from contiguous spread of infection from overlying non-ulcerative or ulcerative disease, especially on the forearm or lower leg.

The World Health Organization (WHO) developed a classification system to catagorise lesions according to their severity. There are three WHO categories of disease: Category I involves a single lesion < 5 cm in diameter, Category II involves a single lesion > 15 cm in diameter, multiple lesions, lesion(s) at a critical site (eye, breast, genitalia), and osteomyelitis. The size of a lesion is determined by the amount of induration surrounding a lesion (not the amount of ulceration). The majority of lesions are advanced (WHO category II or III) at presentation, and there is only one lesion in 95% of cases.

#### Diagnosis

Any person with a non-healing ulcer (longer than two weeks' duration) or a plaque, nodule, or oedematous skin lesion living in a BU-endemic area should be immediately referred for assessment for the presence of BU. (3) Referral should be performed as a matter of urgency, as some lesions can progress rapidly. With early diagnosis the feasibility, cost, and complexity of treatment reduce and outcomes of treatment, including time to healing and presence of long-term deformities, significantly improve.

Initial screening includes a clinical assessment by a clinician—and for lesions suspicious of BU appropriate diagnostic tests should be performed.

The following diagnostic samples can be obtained:

a. Swabs: These should be taken from the undermined edges of a clinically diagnosed Buruli ulcer. They should not be used for non-ulcerative lesions.

- b. Fine-needle aspiration: This is mainly used to obtain samples from clinically diagnosed non-ulcerative lesions. If possible, it is preferred over tissue biopsy. Only physicians or experienced health workers should perform this technique in order to minimise any unintended damage to important organs or structures.
- c. Biopsy (punch or surgical): This may be used in the following situations:
  - When swabs and fine-needle aspiration have been tried, abandoned, or are not suitable
  - To establish a differential diagnosis (e.g., cancer)
  - To confirm the diagnosis of a paradoxical reaction
  - To investigate the possibility of disease recurrence or treatment failure

Note: Punch biopsy should be performed in a setting where the risk of infection can be minimised and where facilities are available to manage any profuse bleeding.

Diagnostic tests (3) that can be performed include the following:

- a. IS2404 PCR test: This can be performed on a swab, aspirate, or tissue from a lesion. This is the most important diagnostic test as it has a high sensitivity and very high specificity. Importantly, swabs of non-ulcerative lesions can be falsely negative, and therefore a swab for PCR should not be relied upon as a diagnostic test for non-ulcerative lesions. These require a fine-needle aspiration or punch or incisional or excisional biopsy to obtain tissue fluid or fresh tissue. As the technology required to perform PCR is complex, it requires a sophisticated laboratory with good quality control to ensure its accuracy. It is expensive, meaning it is often not available in many endemic areas, or, if it is available, testing results can be substantially delayed. Nevertheless, due to its high sensitivity and specificity, it should be performed for all suspected BU lesions when possible.
- b. Ziehl-Neelson stain looking for AFB: *M. ulcerans* organisms are acid-fast on staining and can be identified on an aspirate or tissue from a lesion, or, if not available, from a swab of a lesion. Sensitivity is low, at about 60%, and it is not specific, as other mycobacteria such as *M. tuberculosis* can cause skin lesions. Therefore it is a useful initial test to be performed in clinically suspicious lesions in places where the PCR result is delayed or not available.
- c. Histopathology: This requires a tissue biopsy. Classical findings reveal tissue necrosis with few inflammatory cells (mainly neutrophils), dead adipose cells (ghost cells), and large numbers of extracellular AFB. Sensitivity is high, at about 90%, but it is often not available. It can be useful in situations where the PCR is not available or is delayed by providing supportive evidence to the findings of an AFB smear test.

- d. *Mycobacterium ulcerans* culture: This is best performed on an aspirate or tissue from a lesion, but a swab of a lesion can be used if an aspirate or tissue is not available. This requires a referral laboratory with specific culture media. It has a lower sensitivity (about 50%) and the result will not be available for between six and 12 weeks. Therefore, it is not useful as an initial diagnostic test. However, it may be particularly useful if considering a diagnosis of treatment failure, disease recurrence, or drug resistance.
- e. Thin-layer chromatography: This is a new, innovative, simple, low-cost technique of fluorescent thin-layer chromatography (f-TLC) that could be used as a point-of-care test at a district hospital level with results available within one hour. In a preliminary study, f-TLC had a sensitivity of 73.2% and a specificity of 85.7% when compared with PCR whether the skin sample was a swab, a biopsy or a fine-needle aspirate. (4)

If the clinician is confident that the lesion is BU but diagnostic confirmatory tests are negative, the diagnostic tests should be repeated. Lesions where a definitive diagnosis cannot be made initially should be reassessed after an interval of one to two weeks.

If the clinician is confident the lesion is BU and facilities for laboratory confirmation are not readily available or a significant delay in receiving the results is expected, treatment with antibiotics can be commenced. However, samples should be taken for laboratory confirmation later on. If confirmed to be BU, then antibiotic treatment should be continued. If the diagnosis is not confirmed to be BU the antibiotics should be ceased and the lesion managed as appropriate for the alternative diagnosis.

There are many other causes of non-healing ulcers in BU-endemic areas that need to be considered in the differential diagnosis of BU, (5) including the following:

- Venous ulcers (look for evidence of venous disease; often on medial aspect of leg, chronic)
- Tropical phagedenic ulcer (painful)
- Tuberculosis (look for evidence of TB elsewhere in the body)
- Carcinoma (often are exfoliative, fungating; may be a regional lymphadenopathy; X-ray may reveal destruction of nearby bone)
- Yaws
- Trauma (often lack of undermining edge, prior history of trauma/burn)
- Ischemic ulcer (absent pulses, diabetes)
- Sickle-cell ulcer
- Cutaneous leishmaniasis
- Osteomyelitis with fistula formation
- Vasculitis (e.g., pyoderma gangrenosum)

#### Management

It is now known that treatment with antibiotics alone, without surgery, will sterilise BU lesions and lead to healing without recurrence. Antibiotics are now recommended for all active BU lesions as first-line treatment to achieve sterilisation and cure and to prevent recurrences, which occur in up to 30% of cases treated with surgical excision alone. (2, 6, 7)

Surgery still has an important complementary role to antibiotics in the management of BU. However, surgery is no longer required for cure of *Mycobacterium ulcerans* lesions, and extensive "curative" excisional surgery for BU with wide margins through uninfected tissue is no longer routinely recommended. The aim now is to use minimal surgery to excise necrotic tissue when antibiotics have arrested progression of the disease. This improves the speed of wound healing and helps prevent deformity/scarring in lesions with significant skin/soft-tissue necrosis. Extensive curative excisional surgery may be required where antibiotics are refused, contraindicated, or not tolerated. It may also be considered for lesions with a low risk of recurrence if treated surgically alone (i.e. small uncomplicated lesions where the histological margins are clear).

Additionally, appropriate dressing protocols need to be implemented to optimise wound healing. (7) Adapted modern wound care base on optimal wound bed preparation and wound healing in moist environment will help, even with limited means, to reduce healing time, pain and disability.

Physiotherapy is vital to prevent permanent disability and deformity. (8) These actions must be thought of early and not only for extensive lesions, or those crossing a joint or vital structure.

Furthermore, attention needs to be paid to co-morbidities that may affect response to treatment (TB, HIV, diabetes, malnutrition), and appropriate pain relief should be provided where necessary.

Ideally, care should be decentralised to community settings to allow provision closer to patient's homes, improve adherence to treatment, and reduce loss to follow-up, as well as minimise the economic and social impact of treatment. This may be more feasible if all oral antibiotic regimens are utilised.

#### Antibiotics

First-line regimen:

• Rifampicin 10 mg/kg daily up to a maximum of 600 mg/day plus streptomycin 15 mg/kg daily. Duration of treatment: 8 weeks

Alternative regimens: (9, 10)

- Rifampicin 10 mg/kg daily up to a maximum of 600 mg/day plus clarithromycin 7.5 mg/kg twice daily (up to a maximum of 1000 mg/day). Duration of treatment: 8 weeks
- Rifampicin 10mg/kg daily up to a maximum of 600 mg/day plus moxifloxacin 400 mg daily. Duration of treatment: 8 weeks

In pregnancy:

• Rifampicin 10 mg/kg daily up to a maximum of 600 mg/day plus clarithromycin 7.5 mg/kg twice daily (up to a maximum of 1000 mg/day). Duration of treatment: 8 weeks

In children:

• Moxifloxacin is not recommended in children younger than 18 years of age unless no alternatives are available.

In patients with HIV infection:

- BU-HIV co-infection is as an important challenge for the management of BU disease. (11) There is an increased prevalence of HIV in BU patients, and HIV is associated with more severe BU disease. Furthermore, HIV adversely affects BU outcomes, with increased mortality rates and delayed healing times. Finally, there are significant drug interactions between drugs used to treat BU and TB.
- All BU patients should receive high-quality provider-initiated HIV testing and counselling at their initial contact with the BU treatment centre. Those who are BU/HIV co-infected should be referred to health providers trained in HIV management, ideally integrated within the BU treatment centres to facilitate timely antiretroviral therapy initiation and avoid loss to follow-up. Guidance on management of BU/HIV co-infection is available from the WHO BU-HIV technical update.

In patients with tuberculosis:

• Co-infection of active BU and active TB appears to be uncommon, but as most BU endemic regions are also highly endemic for TB infection, TB should always be strongly considered in any BU patient, especially if also infected with HIV. Active clinical screening to exclude TB should be performed prior to beginning, and at each review during, treatment with antibiotics for BU, as these antibiotics will not adequately treat TB and may lead to the development of drug resistance against important TB drugs. If available, a chest X-ray to screen for TB should be performed in all patients prior to commencing BU treatment. If co-infection with tuberculosis is present, then TB treatment according to WHO guidelines should be immediately commenced.

# Osteomyelitis

The duration of antibiotic treatment should be eight weeks. In the case of bony sequestra, tissue necrosis, or an abscess, surgical treatment is required. The progress of the infection should be monitored regularly through clinical examination and, if available, blood tests (FBE, CRP) and X-ray.

# **Paradoxical Reactions**

These are important complications of BU antibiotic treatment occurring in 10–20% of cases that need to be distinguished from treatment failure. Their pathogenesis likely involves reversal of the immune-inhibitory state induced by the mycolactone toxin due to antibiotic-mediated killing of the organism.

A paradoxical reaction has the following features:

- a. Clinical: an initial improvement on antibiotic treatment in the appearance of a BU lesion followed by deterioration of the lesion or its surrounding tissues, or the appearance of a new lesion(s), either during or after antibiotic treatment
- b. Histopathology: tissue showing evidence of an intense inflammatory reaction

c. Microbiology: cultures of tissue or pus are usually sterile; AFB stains may often be positive on microscopy, although usually only a few or reduced numbers of AFB are seen, and the PCR usually remains positive; AFB stains and PCR cannot be used to distinguish between a paradoxical reaction and treatment failure

The management of paradoxical reactions depends upon the severity. Mild-moderate reactions can be managed with observation only. Severe reactions may require the addition of prednisolone to prevent further tissue necrosis. Ensure active screening and exclusion of TB prior to the use of prednisolone and consider albendazole 400 mg/day for three days at the beginning of prednisolone treatment to prevent strongyloides hyperinfection.

## **Prevention of Disability**

In all instances of joint involvement or limitation of movement, good positioning and frequent movement to maintain joint mobility and reduce limb edema until healing is complete are essential in preventing long-term deformity of joints. (8) In order to prevent permanent impairment, movement should start at the time of diagnosis and be continued long after antibiotic treatment has been completed and skin closure has occurred, as scar maturation may take longer than a year. Splints and compression bandages can be useful. Exercise and movement should cease for seven to 10 days after skin grafting to allow the graft to take.

#### Analgesia

Apart from oedematous forms, BU lesions are usually painless. However, they can become painful if they are complicated by infection, if they progress to involve deeper structures (e.g., bone), during re-enervation of healing tissues, or if they become pressurised secondary to tissue edema such as with paradoxical reactions. Pain can also be a problem when dressings are changed during treatment, post-operatively in those requiring surgical procedures, or during physiotherapy manipulations. Therefore understanding the treatment of, and having access to, drug treatments for pain are important components of BU care.

# **Key Practice Points:**

- 1. Any person with a non-healing ulcer (>2 weeks' duration) or a plaque, nodule, or edematous skin lesion living in a BU-endemic area should be immediately referred for assessment for the presence of BU.
- 2. If the clinician is confident that the lesion is BU but diagnostic confirmatory tests are negative, the diagnostic tests should be repeated.
- 3. All BU cases should be confirmed with a PCR test if available.
- 4. Antibiotics are now recommended for all active BU lesions as first-line treatment.
- 5. In some cases, surgery can have a role to improve the speed of wound healing and help prevent deformity/scarring in lesions with significant skin/soft-tissue necrosis.
- 6. All BU patients should be tested for HIV and actively screened for tuberculosis prior to treatment.
- 7. In all instances of joint involvement or limitation of movement, good positioning and frequent movement to maintain joint mobility until healing is complete are essential in preventing long-term deformity of joints.

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# Burns

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# **Definition of Burns**

A burn is an injury to the skin or other organic tissue primarily caused by heat, radiation, radioactivity, electricity, friction, or contact with chemicals. (1)

# **Epidemiology: The Global Burden of Burns**

Burns are a global public health problem, accounting for an estimated 265,000 deaths annually. The majority of these occur in low- and middle-income countries, with almost half in the WHO South-East Asia Region. (1)

Globally, in 2004 the incidence of burns severe enough to require medical attention was nearly 11 million people and ranked fourth in all injuries. (2) The majority (over 95%) occurred in economically developing countries where medical care resources are limited. (3)

With improvements in socio-economic status, accident prevention measures, and advances of burn medicine, the incidence and mortality of burns are on the decline.

# **Evaluating a Burn Injury**

As with any other kinds of trauma, initial assessment and emergency treatment are extremely important, especially in severe burns.

- The primary survey begins with the ABCs (airway, breathing and circulation), and interventions for life-threatening problems should be taken.
- The initial assessment should include the mechanism and time of the injury and the surrounding environment, such as an enclosed space, noxious chemicals and any related trauma.
- Secondary assessment should examine a patient's past medical history, medications, and allergies, and a thorough head-to-toe evaluation of a patient should be done.
- Basic treatments after initial assessment include:
  - Catheterisation, nasal gastric intubation, tracheotomy; secure venous access must be accomplished when necessary
  - Fluid resuscitation when needed

- Wrapping the patient in a clean sheet and blanket to minimise heat loss
- Apply pain control and sedative treatment

# Assessing area and depth

Timely and accurate estimation of the surface area and depth of a burn injury is essential for determining appropriate management, such as fluid resuscitation, ensuring rapid healing and preventing complications.

The size of the burned area is expressed as a percentage of the total body surface area (TBSA). The larger the percent of body surface area involved by the burn, the worse the prognosis. For the past eight decades, medical professionals have relied on hand-drawn diagrams and other methods or formulas to determine the % TBSA based on numerous reported comparative clinical practices and research projects. (4)

Three commonly used methods are Lund and Browder chart, rule of nines and palmar surface (rule of palm). (4)

The chart revised by Lund and Browder in 1944 was based on previous methods and is commonly used today because of its simplicity and practicability. (5) The rule of nines divides the body surface into areas that each represents 9% of the TBSA. (6) The rule of palm is defined as the projection (apparent) area of the hand, which represents 1% TBSA. (7)

The initial 24-hour fluid resuscitation (half within 8 hours of injury and thereafter according to urine output 0.5-1.0 ml/kg/hr) is calculated following these formulae:

- Parkland formula:
  - With lactated Ringer's solution only
  - 4 ml x body weight in kilograms x % of total burned surface
- General formula in China:
  - Crystalloid solution/colloidal solution = 2:1
  - 1.5 ml × body weight in kilograms × % of total burned surface + 2000 ml (5% glucose solution)

The depth of a burn is related to the anatomical structure injured. Burn injury may involve one or both layers of the skin, and may extend into the subcutaneous fat, muscle, and even bony structures. The classification of burn depth is summarised in Table 1.

A number of techniques have been trialled based on the physiology of the skin and alterations produced by burning. However, none of these techniques have been proven superior to serial clinical assessments by an experienced burn doctor. Up until now, clinical observation of the burn wound and its evolution remains the primary basis of wound-depth assessment.

# **Pathophysiology of Burns**

The skin is the largest organ in the human body. The skin serves as protection against fluid and electrolyte loss, infection, radiation, and provides thermal regulation. Burn wounds—the breakdown of skin barrier function—lead to immune dysfunction and nutrition-related metabolic disorders in the process of disease

development. Wound care, to restore skin's protective function, is the key process in the treatment of burn injury.

Unlike many other forms of trauma, the understanding of the pathophysiology of burn wounds is a slowly evolving process. (8)

Whatever the mechanism, burn injuries cause a local response and, in complex burns, a systemic response. The local response to a burn injury consists of inflammation, regeneration, and repair. (8) In complex burns of more than 20– 30% TBSA, there is also a systemic response due to the extensive release of inflammatory mediators at the injury site. The effects are far reaching and include systemic hypotension (burn shock), bronchoconstriction, a threefold increase in basal metabolic rate, and a reduced immune response. (8) Although this chapter addresses only the burn wound, treatment of burns is a systemic issue, particularly in severe cases.

Classification	Layers Involved	Clinical Manifestation	Wound Healing and Treatment
Epidermis burns (superficial/first degree)	Epidermis only	<ul> <li>Erythematous</li> <li>Painful</li> <li>Without blisters</li> </ul>	The dead epidermis is replaced by regenerating keratinocytes within 3–7days • Apply soothing gels (e.g., aloe vera)/moisturising creams)
Superficial dermal burns (superficial partial- thickness burns/lla degree)	Extends into superficial (papillary) dermis	<ul> <li>Blistered</li> <li>Pink and moist underneath blisters</li> <li>Very painful</li> <li>Wound blanches under pressure</li> <li>Sensation intact</li> </ul>	Usually heal within 2–3 weeks without risk of scarring Cleaning • Topical agents • Dressing

Table 1: The Classification of Burn Depth and Management Principles

cont'd.

Deep dermal burns (deep partial- thickness burns/IIb degree)	Extends into deep (reticular) dermis	<ul> <li>Some blistering</li> <li>Wound moist or dry</li> <li>Wound white or fixed staining (red)</li> <li>Less blanching or capillary refill</li> <li>Sensation diminished</li> </ul>	Conservative management leading to spontaneous healing usually involves prolonged and painful dressing changes and the resultant scar is invariably hypertrophic, leading to cosmetic and functional debility • As for superficial burns • Wound excision and grafting an option
Full-thickness burns (third degree)	Extends through entire dermis	<ul> <li>No blisters</li> <li>Wound dry</li> <li>Leathery appearance to eschar—feels hard</li> <li>Charring</li> <li>No blanching</li> <li>Painless</li> </ul>	<ul> <li>Will not heal spontaneously unless very small</li> <li>As for superficial burns</li> <li>Wound excision and grafting at earliest feasible time</li> </ul>
Deep/severe full- thickness burns (fourth degree)	Extends through entire skin, and into underlying fat, muscle, and bone	<ul> <li>As full-thickness burns</li> <li>Black</li> <li>Charred with eschar</li> </ul>	<ul> <li>Will not heal</li> <li>spontaneously</li> <li>May require</li> <li>elaborate</li> <li>debridement, skin</li> <li>grafting or flap</li> <li>reconstruction,</li> <li>even amputation</li> </ul>

### Criteria for Referring Burn Injuries to a Specialised Burn Unit

Once the burn injury has been evaluated, the healthcare provider must decide whether the patient should be transferred to a specialty burn unit or can be managed properly in local medical facilities.

The decision should be based on size and depth of the burn, age of the patient, cause of the burn, distribution of the burn, premorbid diseases, and co-morbid factors such as associated trauma. (8) The availability of the local medical services is another factor to consider. Table 2 outlines the criteria for referral decision-making.

# Table 2: Criteria for Referring Burn Injuries to a Specialised Burn Unit

• A complex burn injury comprises and is likely to be associated with:

- Large size
- >10% TBSA in children (>5% in children younger than 1 year)
- >15% TBSA in adults

• All full-thickness burns in any age group and any extent

• Deep dermal burns > 5% TBSA in adults and all deep dermal burns in children

#### Mechanism of injury:

- All chemical and electrical burns
- Exposure to ionizing radiation
- High-pressure steam injury
- Suspected non-accidental injury
- Age (<10 or >49 years)

• Site of injury (there are no absolute criteria, but the following should be considered):

- Face, hands, genitals or perineum
- Any flexural surface such as neck, axilla, front of elbows OR back of the knee
- Circumferential deep burns in any age group
- Burns with a suspicion of inhalation injury
- Co-existing conditions that could complicate burn management, prolong recovery or affect mortality
- Associated injuries (fractures, head injury or crush injuries)
- Septic burn wounds

• Burn patients who require special social, emotional or long-term rehabilitation support

From Wounds International (8)

# Burn Wound Treatment (8, 9)

### First aid

First aid and initial management of the burn site can limit tissue damage and subsequent mortality. Emergency management continues to be effective for up to three hours after the initial burn injury.

The priorities are to:

- Make sure that it is safe when approaching the patient.
- Remove the patient from the source of injury and stop the burning process, attend to airway, breathing, and circulation.
- Apply immediate active cooling of the wound to dissipate heat in small burns (less than 10% TBSA). The widely used method is to apply cool tap water in any practical manner (e.g., compress, lavage, or immersion).
- Cover the burn with appropriate dressing.

### Healing process and treatment principle for burn wounds

The healing process and treatment principles of burn wounds are summarised in Table 1. Additional tips for wound care include:

- Wound debridement can only be implemented when the patient is in a stable condition.
- Urgent escharotomy of annular eschar of neck, chest and limbs is necessary to release constriction, relieve chest wall restriction, and improve ventilation.

- A common approach to burn wound care consists of wound cleansing and debridement daily or twice daily and reapplication of the dressing.
- Operative wound care should be scheduled according to the general condition of the patient, the area of deep wounds, and the availability of donor sites and techniques of wound closure.

### **Research Priorities**

There are at least four main priorities for burn research to address global burn patient needs:

- 1. Psychological, sociological, epidemiological, and economic studies to determine the incidence, prevalence, and burden of burns in under-resourced countries
- 2. Developing and exploring the efficacy of burn dressing materials, techniques, and wound care formulations which are affordable and acceptable to and consistently used by individuals with burns
- 3. A better understanding of the pathogenesis of post-burn hypertrophic scarring and contracture, and developing effective methods for their prevention
- 4. Developing and exploring multidisciplinary rehabilitation methods to improve the emotional and psychological support and quality of life of burn survivors. (10) Planning return to home and employment requires a well-developed occupational health and social service.

# **Role Of WHO And Collaborating Organisations**

Working together to integrate burn wound management in resource-limited settings, WHO and collaborating organisations can:

- Implement burn prevention projects and assess their clinical, social, and economic outcomes
- Assess the global burden of burns in resource-limited settings
- Communicate globally aspects of burn wound management that are effective and inexpensive in resource-limited settings

### **Key Practice Points:**

- First aid and initial management of the burn site can limit tissue damage and subsequent mortality.
- Basic treatments include:
  - Catheterisation, nasal gastric intubation, tracheotomy; secure venous access must be accomplished when necessary
  - Fluid resuscitation when needed
  - Wrapping the patient in a clean sheet and blanket to minimise heat loss
  - Applying pain control and sedative treatment
- The decision on whether the patient should be transferred to a specialty burn unit or can be managed properly in local medical facilities should be based a variety of factors.

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# Hansen's Disease (Leprosy)

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# Introduction

Hansen's disease (HD), also known as leprosy, is a chronic disease caused by a slowly multiplying bacillus, *Mycobacterium leprae*, mainly affecting skin, peripheral nerves, mucosa of the upper respiratory tract, and eyes. (1) Primary nerve impairments and disability can be prevented or minimised when disease is diagnosed early and managed adequately. (1–3) The incubation period of the disease is about five years, but symptoms can take as long as 20 years to appear. (1) The World Health Organization (WHO) recommends MDT (multi-drug therapy) consisting of two or three drugs: dapsone and rifampicin for all plus clofazimine for multibacillary disease. (1)

# Impairments and Disability in Hansen's Disease

Visible impairments (wounds, clawed fingers, lagophthalmos, drop foot) at time of diagnosis are classified as a WHO disability grade (DG) 2 and indicate that diagnosis of the disease is late. (3–8) WHO DG1 leads to DG2; both identify protective sensory loss and higher risk for injuries and wounds due to nerve function impairment (NFI). (7–9) See Table 1 for WHO disability grading. (10, 11) An estimated three million persons currently live with HD-related impairments and disability. Greater stigma is associated with visible impairments affecting physical, psychological, social, and economic well-being. (7, 8, 12, 13, 14)

### Epidemiology

In 2017, 150 countries reported 210 671 new HD cases to WHO, and within the past 20 years more than 16 million persons have been treated with MDT (1, 3, 4) (see Table 2). Clinically detectable nerve impairment is found in 33–56% of newly registered HD patients, of which 2–37% present with secondary impairments. (2, 7, 8) An estimated 30% are expected to develop nerve damage of eyes, hands and/or feet. (7, 8) Integration of HD services into general health services has facilitated diagnosis and treatment. However, stigma associated with the disease can be an obstacle to self-reporting and early treatment. (1)

# Risks for Cracks, Blisters, Injuries and Ulcers in Hansen's Disease

If there is neuropathy, there is a high risk for cornea dryness and ulceration, hand and foot cracks, blisters, injuries and plantar ulcers. (16) If not managed, secondary infection, osteomyelitis, and cellulitis can follow. (16, 17)

# **Risks for Plantar Ulceration**

An assessment using a 10 g monofilament or light touch with ball-point pen can indicate whether patients are at risk for plantar ulceration (they are at high risk if the test is not felt by the patient. (9, 18, 21–25) In a study by Seboka and Alert poor-fitting footwear was implicated in 21% of foot ulcer cases with sensory loss. (25) Further identification of feet with pronated or hyperpronated subtalar joints (STJ) can identify those needing specific accommodative orthotics to reduce forefoot pressures and further reduce wounds (see Tables 3 and 4). (18, 19, 20, 26)

# Early Diagnosis of the Disease and Nerve Function Impairment

Health professionals and persons affected by HD play an important role in identifying the disease early and assuring MDT is completed, contacts are examined, and nerve function of eyes, hands and feet is monitored regularly. Nerve function assessment (NFA) is crucial for both disease diagnosis and prevention as well as early treatment of nerve function impairment (NFI). (2, 7, 8, 27–30) Nerve damage in HD is caused by three distinct mechanisms: direct effects of *M. leprae*, mediated by inflammatory and immune mediated processes, oedema, and mechanical processes. (7)

### **Clinical Exam: Skin Lesions and Anaesthetic Areas**

Diagnosis is made through a good history and clinical exam. Laboratory skin smears are negative for paucibacillary (PB) forms of the disease. Talk to the person about how their skin lesion and anaesthetic area started, if they have changed or have sensory loss. (7, 27) Lesions usually appear slowly, do not itch and are not painful. Examine the whole body looking for skin lesions that are usually lighter in colour from the rest of the skin but may also be reddish and have raised borders. (7, 27) HD lesions will not sweat (feel dry) and will have hair and sensory loss. Test sensation with cotton, a feather, disposible pin, toothpick, warm/cold temperatures (32°C baseline) or other and compare with normal skin. (7, 27, 29, 30) Multibacillary (MB) forms may have no skin patches but only skin thickening, lumps or bumps on limbs, face and earlobes with little or no sensory loss. (27) This skin is shiny, dry and redder than surrounding skin and is a sign of a serious HD infection. (27) MB forms with NFI at diagnosis are at highest risk for developing permanent NFI. (7) Skin smears will confirm a large number of leprosy bacilli. (27)

# **Clinical Exam: Nerve Palpation and Nerve Function Assessment (NFA)**

The clinical exam will further investigate recent change in vision, unusual hand or foot sensation (pain, numbness, tingling, burning), or if the person has burned or injured hands and feet without feeling it. Persons may state they lose footwear while walking, drop things from hands, have difficulty buttoning, holding a pen or picking up coins. NFA (motor and sensory) for eyes, hands, and feet will confirm NFI. Key nerves should be palpated for enlargement and pain (27, 28, 29, 31) (see Figure 1). NFI (motor and/or sensory) can occur with or without nerve pain. (31) NFA and nerve palpation is done at time of diagnosis and at a minimum of every three months during MDT. (5, 19) If NFI is present at diagnosis, nerve function should be monitored every visit and continued every three months for two years after MDT completion. (2, 7, 9)

Routine nerve palpation and monitoring of nerve function (sensory and motor) identify NFI early and also identify persons at risk for ulcers. NFI of less than six months' duration can improve with adequate corticoid steroid interventions. (2, 7, 9, 27–30)

Surgical decompression can lessen pain, (2, 7) and clinical observation suggests that nerve function can improve and the level of corticoid steroid dosage can be reduced with surgery. (7, 8, 29) Table 5 shows early primary and late secondary impairments as a result of NFI. (31)

Eye sensory and motor function are evaluated by observing blink frequency, eye closure and by testing corneal sensation. (32) Hand and foot sensory and motor function are best monitored by using a standardised 5 or 6 g monofilament test (MFT) kit (monofilaments bending at 0.05 g, 0.2 g, 2.0 g, 4.0 g, 10 g and 300 g) and a 6-grade voluntary muscle test (VMT) as both monitor nerve function and are more sensitive to detecting change in clinical and field conditions. (2,7, 9, 30) A simplified test is shown in Table 6, with results used to determine DG of 0, 1 or 2 (Table 1). (31)

# Identification of Persons at Risk of Injury and Ulceration

Persons with decreased corneal sensation or those not feeling a 4 g monofilament on the hands or 10 g monofilament of the feet are at risk of mechanical, thermal or chemical injury. (5, 7, 9, 11, 32) Monofilaments are not always available, so a light touch with a ball-point pen may be used; but remember a light touch is usually greater than the pressure of a 10 g monofilament and varies between examiners and exams. (35)

### **Impairment Management**

Persons with permanent NFI require lifelong daily self-care practices to preserve vision, prevent contractures, cracks, blisters, wounds, secondary infections, and ulcers. (5, 12, 17, 33, 34) The utilisation of self-care groups is a key strategy for managing impairments in addition to providing psychological and social support. (17, 33, 34) Preventive and reconstructive surgery for eyes, hands, and feet can make significant contributions to maintain or improve function and wellbeing.

### **Eye Care and Corneal Ulcers**

Preserving vision is a vital objective in HD as self-care will depend upon it. (32) Persons using prednisolone should be monitored for glaucoma and cataracts. (32, 35) Rarely, type 2 HD reactions cause inflammation inside the eye (sudden change in vision, slow-reacting pupil, pain, and eye redness). (32, 35) It is an emergency that is treated with atropine and cortisone drops or ointment. Surgery to the eyelids may be needed to help prevent corneal damage from trichiasis (eyelashes turning inward and rubbing on the cornea) or reduce exposure of the cornea from lagophthalmos (lid gap from muscle weakness making it difficult to close the eyes). In addition, removing cataracts can restore vision that is essential for independent daily function when there is sensory loss to hands and feet. (32, 35)

Daily care involves checking eyes and vision. Face and eyes are washed with clean water with care not to scratch the eye when drying. Eyes are observed daily for redness and eye lashes turning in and touching the cornea. Looking at the same object at 6 m with one eye shut and than the other checks vision. If vision has changed, the subject must blink for five minutes and recheck. If it improves, the eye is too dry, requiring more blinking. One must learn to "think blink" to protect and provide moisture to the eyes. (32, 34, 35)

Eyes with lagophthalmos can be kept moist by using artificial tears frequently during the day and bland oil such as castor oil at night. (32, 35) Eyes must not be rubbed with dirty clothes or rough, insensitive hands. A hat, cap, scarf, or shawl and eyeglasses can reduce the drying. A fan can keep flies away. At night the eye(s) should be covered to maintain moisture and keep dust and insects away. (32–34) A simple elevated eye patch can be made from a 1 cm thick sponge with the centre cut out and covered with a clear plastic and held into place by an elastic or tie head band. A cone-shaped eye shield can also be made from cardboard.

Corneal ulceration is an emergency and best referred to a professional trained in eye healthcare. The eye should be washed with clean water or saline, antibiotic eye ointment applied, and the eye closed. If the person has a lagoph-thalmos, the elevated eye patch should be used. (32, 35)

#### Prevention and Care of Dry Skin, Cracks and Calluses on Hands and Feet

Skin becomes very dry (due to lesions of the autonomous fibres of the peripheral nerves and clofazimine used in MDT), commonly causing cracks in creases of "clawed" fingers and toes, and heels. (33) Healing cracks can result in soft tissue contractures and joint stiffness if not managed adequately. Callus builds up around the edges of cracks and in areas of high pressure or stress. Daily search of cracks and calluses must be made.

Skin is kept soft and flexible by soaking approximately 15–20 minutes in clean water. Hard, dry skin and callus should be rubbed or scraped off with something rough such as a pumice stone, coconut shell or sandpaper. (33, 34) Wet-dry sandpaper of grit 80 (medium) for feet and 100 (fine) for hands can be glued or taped to wooden tongue blades, small round dowels, or blocks. After that, hands and feet should be rinsed with clean water and have oil rubbed into the skin. Common local oils can be used (shea butter, cocoa butter, coconut butter, petroleum jelly). Mineral oils are best, as vegetable oils can attract rats and insects. Drying between the toes and within fixed claw fingers and toes reduces the chance of fungal infection and skin maceration. (33, 36)

Joint flexibility of clawed fingers and toes is maintained by gentle stretching. A small gutter splint (made from smooth, hard material such as plaster, rubber tubing, plastic hoses, wood, bamboo, or other) can be applied to clawed fingers with cracks so that the skin heals in the most extended position possible. The splint should be worn 23 hours each day, removed only for care and movement. (33)

# **Prevention and Care of Blisters and Ulcers**

The most important first step in care is discovering the cause so that future blisters, injuries, or ulcers can be prevented. The health worker, person affected, and family should work together to find solutions. The two main causes of blisters are from heat (cigarettes, hot cups or glasses, hot liquids, pots and pans, cooking and heating fires, and barefoot walking on hot sand) and friction from skin rubbing back and forth over hard surfaces (unpadded tools and poorly fitting footwear). (33)

Blisters should not be opened but gently washed with clean water and soap, covered, and protected until they heal. (33)

The cause of tissue stress must then be removed and the injured part rested so damaged tissue can repair itself. (20, 33, 34, 36) Long wooden cooking tools or protective gloves can be used. Padded work tools can reduce shear stress and improve grip.

The gold standard for offloading pressures in plantar ulcers is the total contact cast (TCC). However, in resource-limited environments, technical training and materials are not easily available or accessible. The next best option for plantar ulcers is to spend as much time as possible lying down with the foot raised above heart level. In practice, this is difficult, as people will walk to take care of personal and family needs. Alternative options may be using crutches or walking sticks along with using protective footwear, walking more slowly, and resting more often. It may temporarily involve swapping work with another and using other transportation options such as riding a bicycle or donkey. (33) Rest and good hygiene prevent infections. Remember that bathing and latrine areas can be infection reservoirs. Routine cleaning with bleach (hypochlorite) will reduce risks. (33)

Following wound care principles facilitates wound healing. (33, 36) The clinician should do a quick check of key local resources (see Table 7). Figure 2 summarises care and monitoring considerations for simple and complicated ulcers. (36)

### **Protective Footwear**

Learning to buy, repair, and replace worn footwear is important for ulcer prevention. (10, 26, 33, 34, 36) Proper-fitting footwear can frequently be found commercially for feet without severe structural changes. Seboka found a 75% reduction in ulcers when subjects used canvas shoes but no significant change for those using plastazote sandals. (25) Identification of insensate feet with pronated or hyperpronated subtalar joints (STJ) identifies those needing specific accommodative orthotics to reduce forefoot pressures and further reduce wounds (18–20) (See Tables 5 and 6).

### **Adaptations and Assistive Devices**

Adaptations and assistive devices are important adjuncts to wound management as they can both protect and improve function. In drop foot, an ankle dorsi-flexion assist with a dynamic elastic strap attached to distal shoelaces of the shoe will protect from ankle sprains and toe injuries (10) (see Figure 3).

# **Chronic Venous Leg Ulcers (VLU)**

VLU can also be a present, especially among the older HD population. Compression bandaging, elevation, and activity are important components of wound management. An Unna boot is very effective as it provides both compression and protection. (36) After healing, daily use of compression bandages or stockings decreases VLU reoccurrence. Unna paste can be prepared in the field. Ingredients and preparation are described in Table 8. (36)

# **Local Support and Referral**

Local nutritional, psychological, physical and social supports are important. In many cases, these may not be available and may need to be developed within the local health system. Surgical debridement, nerve decompression, wound grafts and flaps, correction of clawed toes, and amputation can be important but may not be available or accessible.

Chronic ulcers can turn malignant. (33) An unusual raised border or "cauliflower growth" is typical of squamous cell carcinoma and can be confirmed with a biopsy. (33) Amputation is recommended. (33) Discussions are needed with person affected and their families to obtain approval and plan for the needed support following surgery.

# Self-care Groups

To reduce secondary lesions, self-care and self-care empowerment groups such as the STEP (Stigma Elimination Programme) in southern Nepal have proved successful. (12, 17)

# **Key Practice Points:**

- Suspect, confirm and treat HD early with MDT before Grade 1 and Grade 2 disabilities.
- Suspect, confirm and treat nerve function impairment (NFI) to preserve nerve function.
- Identify persons with protective sensory loss of eyes, hands, and feet at risk of ulcers; involve the person in self-care.
- Preserve vision to enable self-care and prevent injury during daily activities.
- Protect and offload high pressures on sensory loss skin areas by using protective footwear and padding work tools.
- Clean, moisturise, cover, rest, and protect wounds until healed, continue after for prevention.
- The person affected and families are central to wound care planning and problem solving.
- Improve self-esteem and provide physical, psychological, and social support with self-care groups.

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Table 1: WHO Hansen's Disease Disability Grade for Eye, Hand, Foot Score (EHF Score)

Grade	Eyes	R	L	Hands	R	L	Feet	R	L
0 Grade	No severe visual impairment (can count fingers at 6 m; visual acuity > 6:60 No visible impairments Normal blink <b>Eyes</b>		ag nd	Touch is felt on the palm of the hand No muscle weakness or visible impairment <b>Hands</b>	Di Er		Touch is felt on the soles of feet No muscle weakness or visible impairment <b>Feet</b>		ag nd
	Loss of blink reflex, and/or inability to hold the eyelids closed against moderate force to open them No severe visual	Di	ag	At least 2 points on the hands where touch is not felt (4 g monofilament or light touch with ball point pen) and/ or	Di	ag	At least 2 points on the foot where touch is not felt (10 g monofilament or light touch with ball point pen) and/ or		ag
1	1 impairment (can count fingers at 6 m; visual acuity > 6:60		nd	Muscle weakness is present on testing but there is no visible impairment	Er	nd	Muscle weakness is present (on testing) but there is no high-stepping gait when the patient walks and there is no other visible impairment	Er	nd
Grade	Eyes	R	L	Hands	R	L	Feet	R	L
	Visible impairments to eye due to leprosy For example: iridocyclitis, lagophthalmos, corneal ulcer or		ag	Visible impairments to the hand if it has occurred since the onset or loss of sensation and/ or loss of muscle function due to	Di		Visible impairments to the foot if it has occurred since the onset or loss of sensation and/ or loss of muscle function due to		ag
2	scars, corneal opacity, ectropion, entropian, trichiasis, nodules on the sclera, irregular shaped or pin point pupil Severe visual impairment (cannot count fingers at 6 m, visual acuity <6:60 regardless of cause)	Er		leprosy For example: any bone loss, claw finger(s), muscle wasting, wrist drop, wounds(s), deep cracks	Er		leprosy For example: any bone loss, claw toe(s), high stepping gait (obvious foot drop), wounds(s), deep cracks	Er	

<b>Results of EHF Sc</b>	Results of EHF Score and Disability Grade at Diagnosis and End of MDT								
At Diagnosis Date					Grade for Feet		EHF Score a+b+c+d+e+f	Maximum Grade	Signature
//	R-a	L-b	R-a	L-b	R-a	L-b			
End of MDT									
Date	R-a	L-b	R-a	L-b	R-a	L-b			
// (dd/mm/yy)									

From Lehman, Geyer and Bolton (10)

Table 2: Registered Prevalence of Leprosy at End of 2017 and Number of New Cases of Leprosy Detected during 2017, by WHO Region (received from 150 countries)

WHO Region	<b>Registered</b> Pr	evalence	Number of New Cases			
	Number Rate per 10 000 population		Number	Rate per 1 000 000 population		
African	30 654	0.28	20 416	1.90		
Americas	31 527	0.31	29 101	2.86		
Eastern Mediterranean	4 405	0.06	3 550	0.51		
European	32	0	33	0		
South-East Asia	119 055	0.60	153 487	7.72		
Western Pacific	7 040	0.04	4 084	0.21		
Total	192 713	0.25	210 671	2.77		

From World Health Organization (3)

Table 3: Subtalar Joint Position and Relative Risk of Ulceration among Persons with Leprosy with Sensory Loss Greater than 10 g, Ulcers or Scars

(n=110)	No Ulcers	Ulcer	Relative Risk (95% confidence)
Neutral	16	2	
Hyperpronated	20	22	5.3 (C.I. 1.3 – 21.1)
Pronated	21	11	2.8 (C.I. 0.7 – 10.5)
Supinated	14	4	1.6 (C.I. 0.5 – 5.2)

From Cross and Rendall (18)

Table 4: Comparison of Foot Posture and Peak Pressures in Group with Sensory Loss and Group with Sensory Loss Plus Plantar Ulcer or Scar in Persons with Leprosy

(n=110)	Group (n=71)	with Sensory Loss Only	Group with Sensory Loss Plus Ulcer or Scar (n=39)			
	Feet	Median Peak Pressure	Feet	Median Peak Pressure		
Neutral	16	2.1 (29 PSI)	2	2.3 (32 PSI)		
Supinated	14	2.5 (35 PSI)	3	2.8 (40 PSI)		
Pronated	21	2.4 (34 PSI)	11	3.8 (54 PSI		
Hyperpronated	20	2.9 (41 PSI)	22	3.6 (52 PSI)		

From Cross and Rendall (18)

Figure 1: Common Peripheral Nerves affected in Hansen's Disease for Palpation and Abbreviated Sensory and Motor Nerve Function Assessment

#### 1. Trigeminal Nerve

- Observe blink
- Test corneal sensation

#### 2. Facial Nerve

- Check eye closure by closing as in sleep
- Check resistance when closing eyes tightly

#### 3. Auricular Nerve

### 4. Radial Nerve

Check force of "wrist up"

- 5. Radial Cutaneous Nerve
  - Check dorsal web space sensation(optional)

#### 6. Median Nerve

- Check sensation on palmer side of thumb, index finger and hand
- Check force of "thumb up" in abduction

#### 7. Ulnar Nerve

- Check sensation on palmer side of little finger and hand
- Check force of "little finger out" in abduction

#### 8. Peroneal Nerve

- Check "foot up" in dorsiflexion
- Check "big" toe up
- 9. Sural Nerve (lateral, dorsal side of foot)
  - Check dorsal later side of foot (optional)
- 10. Tibial Nerve (medial side of ankle)

 Check sensation on plantar surface of foot

Adapted by Lehman, 2006, from Lehman et al., 1997 and 2006 (31)

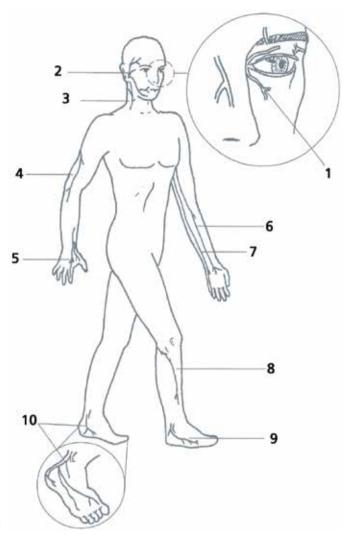


Table 5: Peripheral Nerve Lesions in Hansen's disease

Effects of *M. leprae*, inflammatory and immune-mediated processes and oedema and mechanical processes on peripheral nerves

Autonomic Fibres	Sensory Fibres	Motor Fibres
<ul> <li>Impaired vasomotor reflexes</li> <li>Inability to sweat normally in area affected</li> <li>Loss of hair in area affected</li> </ul>	<ul> <li>Decrease corneal sensation</li> <li>Decrease in feeling light touch, pressure, temperature, pain</li> </ul>	Decrease strength
Common observations and	d complaints	
<ul> <li>Area is dry, no sweating and with hair loss</li> </ul>	<ul> <li>Forgets to blink</li> <li>Complaint that specific areas feel strange, tingly, different or less</li> <li>Complaint of difficulty identifying and removing coins from pocket</li> <li>Complaint that sandals fall off feet while walking</li> </ul>	<ul> <li>Difficulty closing eyes, turning keys, writing, picking up feet</li> <li>Hands look thinner (atrophy)</li> <li>Calf muscle and feet look thinner (atrophy)</li> </ul>
Identify who is at risk for crac	cks, blisters, injuries and/or wour	nds. Practise daily self-care,
Protect eyes, hands and feet. <b>Eyes:</b> Dry eye (risk for corneal ulceration) <b>Hands and Feet:</b> Dry skin	cks, blisters, injuries and/or wour Treat complications (wound car <b>Eyes:</b> Loss of corneal sensation (risk for corneal ulceration – dry eye from	e, surgery, therapy, etc). Muscle paralysis increases risk for contractures and increases areas of high
Protect eyes, hands and feet. <b>Eyes:</b> Dry eye (risk for corneal ulceration)	Treat complications (wound care <b>Eyes:</b> Loss of corneal sensation (risk for corneal	e, surgery, therapy, etc). Muscle paralysis increases risk for contractures and
Protect eyes, hands and feet. <b>Eyes:</b> Dry eye (risk for corneal ulceration) <b>Hands and Feet:</b> Dry skin	Treat complications (wound care <b>Eyes:</b> Loss of corneal sensation (risk for corneal ulceration – dry eye from exposure and dryness from not blinking sufficient) <b>Hands and Feet:</b> Loss of sensation (risk for blisters, injuries, ulcers when doing	e, surgery, therapy, etc). Muscle paralysis increases risk for contractures and increases areas of high pressure Eyes: Lagophthalmos (risk of corneal ulceration from cornea exposure and dryness from not being able to close
Protect eyes, hands and feet. <b>Eyes:</b> Dry eye (risk for corneal ulceration) <b>Hands and Feet:</b> Dry skin	Treat complications (wound care <b>Eyes:</b> Loss of corneal sensation (risk for corneal ulceration – dry eye from exposure and dryness from not blinking sufficient) <b>Hands and Feet:</b> Loss of sensation (risk for blisters, injuries, ulcers when doing	e, surgery, therapy, etc). Muscle paralysis increases risk for contractures and increases areas of high pressure Eyes: Lagophthalmos (risk of corneal ulceration from cornea exposure and dryness from not being able to close the eye) Hands: Clawing of fingers (risk for contractures and
Protect eyes, hands and feet. <b>Eyes:</b> Dry eye (risk for corneal ulceration) <b>Hands and Feet:</b> Dry skin (risk for cracks)	Treat complications (wound care <b>Eyes:</b> Loss of corneal sensation (risk for corneal ulceration – dry eye from exposure and dryness from not blinking sufficient) <b>Hands and Feet:</b> Loss of sensation (risk for blisters, injuries, ulcers when doing	e, surgery, therapy, etc). Muscle paralysis increases risk for contractures and increases areas of high pressure Eyes: Lagophthalmos (risk of corneal ulceration from cornea exposure and dryness from not being able to close the eye) Hands: Clawing of fingers (risk for contractures and injury) Feet: Foot drop, clawing of toes (risk for contractures and ulcers)
Protect eyes, hands and feet. Eyes: Dry eye (risk for corneal ulceration) Hands and Feet: Dry skin (risk for cracks)	Treat complications (wound card Eyes: Loss of corneal sensation (risk for corneal ulceration – dry eye from exposure and dryness from not blinking sufficient) Hands and Feet: Loss of sensation (risk for blisters, injuries, ulcers when doing daily activities) ters, injuries, ulcers and seconda	e, surgery, therapy, etc). Muscle paralysis increases risk for contractures and increases areas of high pressure <b>Eyes:</b> Lagophthalmos (risk of corneal ulceration from cornea exposure and dryness from not being able to close the eye) <b>Hands:</b> Clawing of fingers (risk for contractures and injury) <b>Feet:</b> Foot drop, clawing of toes (risk for contractures and ulcers)

Adapted by Lehman from van Brakel et al. (8) and Lehman et al. (31)

Table 6: Simplified Vision and Nerve Function Assessment for Monitoring Change

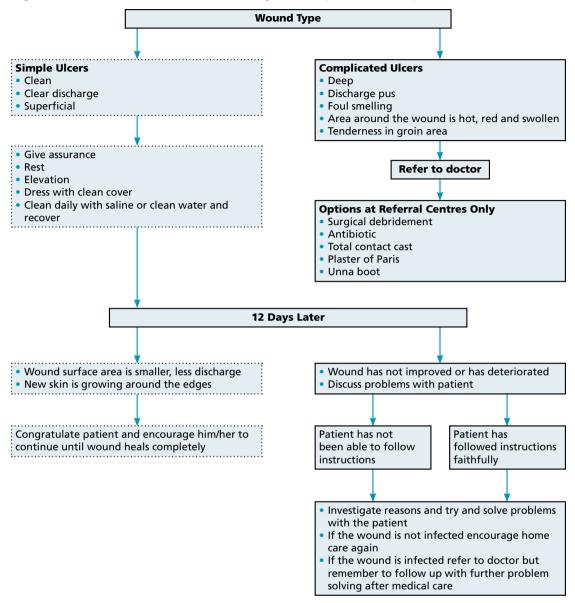
Name:				Age:	Occupatio	n:
Date (1)	Date (2)	Date (3)	Vision and Neurological	Date (1)	Date (2)	Date (3)
			Exam			
Right			EYES		Left	
m SC	m SC	m SC	Visual Acuity Note finger count in metres 0–6 or number on Snellen Chart (SC)	m SC	m SC	m SC
Yes No	Yes No	Yes No	Cornea: Loss of Sensation Blink decreased or decrease sensation with 5 mm length dental floss	Yes No	Yes No	Yes No
P W S	P W S	P W S	Loss of Muscle Strength Eye Closure P=Paralyzed, W=Weak, S=Strong	P W S	P W S	P W S
mm	mm	mm	Lid Gap: Light closure of eyes Measure lid gap in mm	mm	mm	mm
mm	mm	mm	Lid Gap: Tight closure of eyes Measure lid gap in mm	mm	mm	mm
Yes No	Yes No	Yes No	Visible Impairments of the Eyes	Yes No	Yes No	Yes No
	Right		HANDS		Left	
ΡΕΝ	ΡΕΝ	ΡΕΝ	Nerve Palpation: Ulnar P=Painful, E=Enlarged, N=Normal	ΡΕΝ	ΡΕΝ	ΡΕΝ
		Eval	uate Loss of Muscle Strength in Ha P=Paralyzed, W=Weak, S=Strong	ands		
PWS	PWS	PWS	Little Finger Out (abduction)	PWS	PWS	PWS
PWS	PWS	PWS	Thumb Up (abduction)	PWS	PWS	PWS
PWS	PWS	PWS	Wrist Up (extension)	PWS	PWS	PWS
			Protective Sensory Loss to Palm of Hands Light touch with ballpoint pen or 4 g monofilament X = Loss of sensation O = Feels touch			
Yes No	Yes No	Yes No	Wounds on Hands	Yes No	Yes No	Yes No
Yes No	Yes No	Yes No	Visible Impairments of the Hands	Yes No	Yes No	Yes No
			nanus			

cont'd.

	Right		FEET		Left	
ΡΕΝ	ΡΕΝ	ΡΕΝ	Nerve Palpation: Peroneal P=Painful, E=Enlarged, N=Normal	ΡΕΝ	ΡΕΝ	PEN
ΡΕΝ	ΡΕΝ	ΡΕΝ	Nerve Palpation: Tibial P=Painful, E=Enlarged, N=Normal	ΡΕΝ	ΡΕΝ	PEN
		Eva	luate Loss of Muscle Strength of F P=Paralyzed, W=Weak, S=Strong	eet		
PWS	PWS	PWS	Foot Up (dorsiflexion)	P W S	P W S	PWS
P W S	PWS	PWS	Large Toe Up (extension)	PWS	PWS	PWS
0	0 0	0 0	Sensory Loss to Sole of Feet Light touch with ballpoint pen or 10 g monofilament X = Loss of sensation O = Feels touch	° °	° °	0 0 0
Yes No	Yes No	Yes No	Wounds on Soles of Feet	Yes No	Yes No	Yes No
Yes No	Yes No	Yes No	Visible Impairments of the Feet	Yes No	Yes No	Yes No
Signature	Signature	Signature		Signature	Signature	Signature

Adapted by Lehman, from Lehman, Geyer and Bolton (10)

### Figure 2: Wound Care and Monitoring for Simple and Complicated Ulcers



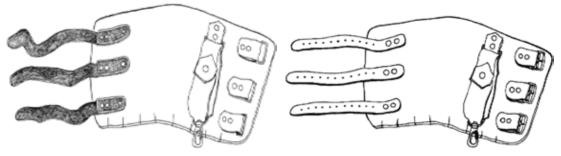
#### Adapted from Cross (36)

# Table 7: Checklist for wound care (place X if present or done)

· · · · · ·	
1	. Clean water is available and accessible
2	. Materials and supplies to do wound care are available and accessible
3	. Organises materials before starting wound care
4	. Washes hands before wound care procedure
5	. Uses gloves appropriately
6	. Removes gauze and bandages without damaging new skin
7	. Cleans wound with clean water or saline solution to remove debris and dead tissue without damaging new skin
8	. Checks to see if wound is improving, getting worse or the same
g	. Moves joints near or at the wound before new dressing and bandage is applied
1	0. Applies clean Vaseline gauze or other moisture-retentive dressing
1	1. Bandages with light compression distal to proximal
1	2. Bandages without restricting circulation or movement
1	3. Tapes end of bandage, does not tie a knot to secure bandage
1	4. Follows special care procedures for skin grafts under 10 days old
1	5. Disposes of contaminated material safely
1	6. Rests the affected part
1	7. Protects the affected part
1	8. Keeps bandage clean and dry
1	9. Changes bandage if outer bandage becomes wet
2	0. Asks for help if not improving or getting worse
	2016 from Lehman Gever and Bolton (10)

Adapted 2016 from Lehman, Geyer and Bolton (10)

Figure 3: Dorsi-flexion Assist with Dynamic Elastic Strap that Attaches to Shoe Laces



Ankle straps with Velcro closures From Lehman, Geyer and Bolton (10) Ankle straps with buckle closures

Tabe 8: Unna Paste Preparation for Unna Boot Used for Venous Leg Ulcers

Ingredients (36)	Preparation
100 g of gelatine powder, no colour or flavour	<ol> <li>Prior to application wash the leg with clean water and elevate the leg while preparing the paste.</li> <li>Mix gelatine powder and distilled water together and set aside</li> </ol>
350 g of distilled water	for approximately 5 minutes. 3. While waiting, Mix together zinc oxide and glycerine in
100 g of zinc oxide	separate container
400 g of glycerine	<ol> <li>Heat up gelatine mixture until the gelatine dissolves but does not boil.</li> <li>Once dissolved, add zinc oxide mixture</li> <li>Soak bandages (gauze or preferably elastic) in paste</li> <li>Remove soaked bandage rolls from paste and apply bandage from toes up to the knee.</li> <li>Repeat process to complete 3–4 layers</li> <li>Cover with dry bandage</li> <li>Encourage the person to walk, to do daily activities and keep Unna boot dry</li> <li>Remove Unna boot carefully with blunt-ended scissors</li> </ol>
<ul> <li>The Unna boot provid</li> </ul>	es protection of fragile skin and light compression to reduce

oedema

• Initially the Unna boot may be changed more frequently due to reduction of oedema or excessive exudate.

• As oedema and exudate reduce, the Unna boot can be changed weekly.

• Have compression stocking available to put on when the last Unna boot is removed.

Good skin care is done daily.

# **Using Local Resources for Wound Care**

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### Introduction

Access to contemporary wound care products is limited in most resource-limited communities. In the absence of access to expensive modern products, clinicians and informal caregivers continue to use traditional wound management strategies that are often locally developed. While the use of some traditional management strategies is supported by a documented evidence base, other interventions have limited or no formal research to supporting their efficacy. The primary goal of this chapter is to promote quality of care by providing guidance on the use of traditional wound care products and care strategies and detailing known adverse events.

### **Management Strategies**

This chapter provides brief summaries on the efficacy of traditional wound management strategies. The Wound Healing and Management (WHAM) Node within the Joanna Briggs Institute (JBI) and the Australian Wound Management Association have undertaken more extensive literature reviews and evidence summaries regarding some of these practices. They are available through HINARI in the Joanna Briggs Institute EBP database (OVID).

# **Topical Antimicrobial Agents**

# Polyhexamethylene biguanide (PHMB)

Polyhexamethylene biguanide (PHMB) has a long history as an anti-microbial and is used as an alternative to contemporary topical antiseptics, particularly in the management of biofilm. *In-vitro* studies have demonstrated the efficacy of PHMB 2% solutions in eradicating *P. aeruginosa* biofilms and *S. aureus. In-vivo* studies demonstrate effectiveness of PHMB-impregnated dressings in reducing clinical signs of infection, as well as bacterial levels, in wounds without heavy exudate. In numerous studies, PHMB-impregnated dressings are associated with faster wound healing (compared with gauze or foam dressings) and reduction in wound pain. No adverse effects have been reported from use of PHMB in wound care, although its use is not recommended in wounds of the central nervous system (including lumbar wounds) or during pregnancy. *(1)* 

### Acetic acid

Acetic acid is a traditional antiseptic with an ancient history that was revived in World War I when it was found that treating wounds with a 1% solution for two weeks resulted in elimination of *B. pyocyaneus. In-vitro* and *in-vivo* studies have shown efficacy of 3–5% acetic acid in eliminating gram-negative and gram-positive bacteria (particularly *Pseudomonas* strains), although wound healing may be slowed. Acetic acid 3% can be applied to a wound or burn via a gauze-soaked dressing, replaced at least twice (preferably four times) daily. At concentrations of 3%, no local or systemic side effects are reported; however, pain, stinging, and itching are associated with stronger concentrations, and application of non-diluted (pure) acetic acid results in severe burns. Therefore, clear labelling of products and development of local guidelines for safe preparation are essential. (2)

### Iodine preparations (iodophors)

lodine preparations are topical antimicrobial options for superficial and shallow-depth wounds. Iodine is generally administered via an iodophor (surfactant) that stabilises the iodine and allows a sustained slow release of iodine to the wound bed while reducing potential side effects (e.g., allergic reactions, pain upon application to open wounds, and irritation to tissue). The two iodophors commonly used in wound management are povidone iodine (PVP-I) and cadexomer iodine. Although there is conflicting evidence on their effectiveness, most in-vitro studies have shown PVP-I is active against S. aureus at concentrations of 0.001% and 0.005%, and that both PVP-I and cadexomer iodine inhibit biofilm arowth. However, evidence in clinical settings is lacking. There is some evidence that iodine enhances angiogenesis and modulates white cell activity. Iodophors are applied as topical solutions or impregnated dressings, and in some countries PVP-I is mixed with sugar and stirred vigorously to form a paste. No substantial difference in adverse reaction is reported between iodophors and other local wound care strategies; however, care should be taken to avoid use of iodine in individuals with known sensitivities, thyroid or renal dysfunction, and in management of extensive burns. (3)

# Citric acid

Evidence that is generally of low quality suggests that citric acid is effective for promoting wound healing and eradicating or reducing *S. aureus* and *E.coli* from chronic wounds. Complete wound healing has been achieved with daily application of 3% citric acid and histological findings suggest that citric acid does not necessarily create an environment that is toxic to a healing wound. Patients report mild to severe itching and burning associated with application of citric acid. In general wound care, 3% citric acid can be used as an irrigation solution or to soak gauze and apply directly to the wound (change dressing daily). Where resources are available, a 3% citric acid ointment can be prepared by a trained technician by mixing pure, monohydrate citric acid with soft white paraffin to attain a pH of 2.0–2.5. *(4)* 

# Hypochlorite solutions

A hypochlorite contains ions of oxygen and chlorine that combine with salts (e.g., sodium or calcium) to form a solution with an alkaline pH (around 7.5 to 8.5). The most commonly known hypochlorites are Dakin's solution (sodium) and Edinburgh University Solution of Lime (EUSOL [calcium]). Although *in-vitro* studies have demonstrated anti-bacterial effects of sodium hypochlorite at 0.025% concentration and lower, these effects are short-lived, and clinical evidence has failed to confidently support *in-vitro* findings. Studies on toxicity of calcium hypochlorite to cells are sparse and conflicting, and its use is not recommended. In addition, increased wound pain is often reported due to the alkaline nature of hypochlorites. Sodium hypochlorite should be used at concentrations no greater than 0.025% and only when there is no alternative antimicrobial option. *(5)* 

# **Topical Agents Traditionally Used to Promote Wound Healing**

# Turmeric

Turmeric, the active ingredient of which is curcumin (difeurloylmethane), is a spice that is traditionally used in Indian and Asian medicine for wound treatment due to perceived anti-inflammatory, antioxidant and antimicrobial effects. *In-vitro* studies demonstrate a protective effect on wound regeneration cells, and animal studies show superior wound healing associated with curcumin; however, there is no clinical evidence in human wounds to support these findings. In its traditional form, turmeric is applied to wounds as a paste prepared from the turmeric rhizome mixed with mustard oil and water. Turmeric-impregnated bandages are also available in India. Turmeric/curcumin are generally well tolerated; however, local inflammation is reported with application of topical curcumin. *(6)* 

# Aloe vera

Aloe vera gel is obtained from the inner part of the succulent leaf of the perennial plant. The gel has been traditionally used to promote healing of burns and minor skin irritations. Although the plant is 99% water, aloe gel contains glycoproteins, polysaccharides, and monosaccharides that have been shown in *in-vitro* studies to bind to growth factor receptors on the surface of fibroblasts, thereby enhancing wound healing activity. However, clinical trials with burns and chronic wounds fail to demonstrate enhanced healing, and in acute wounds aloe vera has been associated with delayed healing. (7) In traditional use aloe gel is applied to the wound daily or every other day, preferably in the acute phase only. Prolonged use may lead to contact dermatitis.

# **Wound Dressings**

### Tea tree oil dressings

Tea tree oil (TTO) is an essential oil traditionally used for its antibacterial and anti-inflammatory properties. A range of evidence demonstrates its effectiveness in managing common skin conditions (e.g., dermatitis, acne, and tinea), and *in-vitro* studies support its activity against bacteria, fungi, and herpes simplex virus. One *in-vitro* study reported effectiveness in decreasing methicillin-resistant *S. aureus* (MRSA) biofilm activity at concentrations of 1%. Further research is required to determine if there is a sustained effect or ability to completely eradicate biofilm. A small body of evidence from clinical trials suggests that TTO in concentrations from 1–10% may promote reduction in chronic wound sizes and increase granulation and healthy epithelialisation. In some studies it has been used effectively to facilitate wound debridement, reduce local inflammation, and as a topical antimicrobial. Tea tree oil is applied to wounds through impregnation in common wound dressings. Mild skin irritation is reported in studies investigating TTO for dermatological conditions, but no adverse effects are reported from direct application to wounds. *(8)* 

# Banana leaf dressings

Banana leaves have a waxy surface that prevents the dressing adhering to the wound and, although impervious to water, they allow exudate to drain from the wound due to slits made in the leaves before application. Banana leaf dressings (BLD) are associated with rapid healing of skin graft donor sites and a low incidence of wound infection. Concurrent antiseptics (e.g., PVP-I ointment) are sometimes used in conjunction with BLD. Patients report that BLD are comfortable to wear and are associated with lower levels of pain, including on dressing removal. Studies have been conducted in India, Uganda, and Thailand (including details on local methods of BLD preparation), where supplies are easily accessible at a very low cost. (9)

### Potato peel dressings

Potato peel dressings (PPDs) provide a low-cost, traditional wound dressing option for the management of a variety of wound types. PPD provide a wound healing environment in which desiccation of the wound surface is prevented and optimal epithelial regeneration can occur. Although PPD has no inherent antibacterial properties, wounds treated with PPD show no greater incidence of bacterial colonisation compared with gauze dressings. Clinical studies (some of which detail local methods of PPD preparation) have demonstrated faster wound healing compared with petroleum gauze or BLD. Patients also report that PPD relieve pain. No studies have reported adverse effects associated with PPD. (10)

# **Wound Debridement Strategies**

### Larval therapy (myiasis)

It has been long established that the use of larvae in aiding wound debridement (i.e. removal of non-viable tissue and foreign debris) has merit. Larval debridement involves applying medical-grade sterile (non-breeding) maggots to the wound bed. Caution is advised that not all flies are the same. The green bottle fly (Lucilia sericata) is the most efficacious species due to its exclusive diet of necrotic tissue. The digestive juices secreted by the active larvae contain proteolvtic enzymes that selectively debride necrotic tissue while leaving non-viable tissue unharmed. Many studies provide clinical evidence that larval debridement therapy reduces necrotic wound tissue more rapidly than conventional therapy alone, often with reduced wound odour and exudate. The larvae are applied to the wound, covered with gauze and taped securely at the edges to contain the larvae. Peri-wound skin can be protected with application of gauze or film dressing prior to applying larvae to the wound bed. Wound dressings are removed after three days. Larval therapy should not be applied close to major blood vessels, and as minor bleeding is a side effect, caution is required in patients taking antiplatelet medication or with bleeding disorders. Mild to severe pain has been reported. (11)

### Carica papaya

*Carica papaya* is a tropical plant, the fruit of which is traditionally used for a variety of medicinal purposes, including as a wound debriding agent. Studies conducted in animals suggest that protease enzyme (papain and chymopapain) activity assists in removal of slough, necrosis, and debris, thereby allowing faster epithelialisation. Clinical studies from West Africa and India conducted in full-thickness burns, infected wounds, and pressure ulcers provide evidence for the effectiveness of *Carica papaya* in promotion of wound granulation, and a possible role in reducing wound bioburden. The fruit pulp is either mashed and applied directly to the wound bed or grated and blended to a paste that is applied to gauze. Wounds dressings are changed daily or twice daily. Enzymatic content is reduced as the fruit ripens, suggesting better efficacy of raw papaya. *(12)* No clinical studies report on adverse effects; however, *Carica papaya* is an allergen that may cause reaction.

### Assistive Devices Used in Conjunction with Wound Care

# Overshoes to protect lower-limb wound dressings

Large rice bags are found in many resource-limited communities. Apart from using the empty bags for storage, these bags are waterproof, thus providing an excellent resource for protecting wound dressings on lower limbs. On a trip to the Solomon Islands, nursing students produced many overshoe designs that can be made by informal caregivers and brought to the clinic for waterproofing after clean wound dressings are applied (see Figure 1).

### Figure 1: Protective overshoe



### Softened bamboo support splints

On a trip to remote Indonesia, where hand and arm splints were required for patients following tendon surgery over flexor regions, we reviewed the cost of the normal casting materials and realised the local bamboo would be suitable as it is quite pliable once softened in water for a period of time. These splints can be made in advance and used with burns victims to help reduce contractures over flexor areas.

### Leather splints following hand surgery

Another product often found in resource-limited communities is leather. Although it is much sought after and often expensive in developed nations, in many countries animal hide is found in abundance. While in Indonesia splints were required for patients who had undergone tendon surgery. Local therapists indicated that patient needs could be met with splints made using leather. The splints were kept after use for assignment to future patients requiring similar sizing.

### **Key Practice Points:**

- Clinicians and informal caregivers can use traditional wound management strategies using locally available resources.
- Evidence exists to support some of these strategies.
- Wound care clinicians should work to integrate traditional practices with modern wound healing strategies.

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# The WAWLC Wound Care Kit: A Tool for Modern Wound Care in Limited-resource Settings

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Chronic wounds are a public health problem too often ignored in some regions with limited resources such as in Africa. The main aetiologies of these wounds are similar to those encountered in Europe (such as vascular or diabetic ulcers, traumatic wounds, burns) but to which are added tropical pathologies of infectious origin (such as leprosy, Buruli ulcer, phagedenic ulcer, sickle cell ulcers, and wounds from trauma with secondary infection). In emergency situations such as displacements of populations, armed conflicts, or natural disasters, wounds resulting from physical trauma are one of the primary reasons for demand for care.

The treatment of these wounds sometimes requires long and expensive care. The patient often needs to remain in hospital, sometimes accompanied by someone else when a person of limited independence is involved (an elderly person or a child, for example). Even when the care is provided free of charge, the indirect costs—such as purchase of food, loss of ability to work, interruption to schooling for a child as well as social limitations—are a burden that is difficult to bear.

Existing kits tend to focus on on large but poor use of antiseptics and drying of the wound, resulting in long, expensive, and painful care. Serious physical consequences, like limb contractures and amputations can occur if the wounds are not managed effectively. Material for dressings is often not available, being limited to various bandages and compresses.

Some decades ago the approach to wound care transformed, notably thanks to a better understanding of the physiological processes of healing. The principles that were traditionally based on frequent disinfection and drying of the wound are no longer in current use. There is an international consensus in favour of healing conducted in a moist environment. These principles have been adopted by WHO as the benchmark approach for wound care. (1)

Healing conducted in a moist environment has largely benefitted from the arrival of new dressings on the market (such as hydrocolloid, hydrogel, hydrocellular, and alginate dressings), which, unfortunately, remain expensive and rarely available in regions with limited resources.

# **Principles Behind the Humanitarian Kits**

International organisations such as CICR, HCR, UNICEF, and NGOs like MSF, MDM, CARE, and Oxfam are often called to respond to emergency situations due to conflicts or natural disasters.

In the 1980s, standardisation of medicines and medical devices was sought to rationally and effectively respond to health needs in these emergencies. This initial work led to provision of standardised and pre-packaged kits that were immediately available and easy to transport.

These kits meet the guiding principles of WHO (2) for administration of medicines so they are of actual use to the beneficiaries of care. The kits are generally composed of a base unit and complementary units, enabling the response to be better suited to the variety of scenarios encountered on the ground.

The establishment of these kits is the object of consensus from the major emergency organisations and WHO. They are regularly updated according to feedback, development of medical products, and protocols.

The concept of emergency health kits is considered as reliable, standardised, inexpensive, and capable of quickly providing essential medicines and medical devices (renewable supplies and equipment) necessary in an emergency.

Different kits are developed according to the situations they must address. The most useful kits are, for example, the cholera kit, (3) the Interagency Emergency Health Kit 2011, (4) and the Reproductive Health Kit from UNFPA. (5)

# **Updating the Wound Care Kit**

At present a dressing kit (6) exists for nursing staff. It contains the renewable supplies necessary for 50 dressings. It is largely used by international organisations but was created more than 30 years ago and does not take into account progress made in dealing with wounds. It is mainly composed of dry gauze and iodine. Based on the principle of drying and disinfecting, it does not enable a modern approach to wound care to be taken, and thus it is necessary to update it.

This wound care kit, as with all the kits, makes it possible to:

- Provide basic equipment in an emergency situation but also for primary healthcare
- Enable distribution of standardised material, known in advance
- Quickly order the appropriate material
- Facilitate management of stocks and health programs
- Facilitate management of health programs by better controlling orders and their budgets

However, any update to the wound care kit must also enable a dynamic to be created for:

- Standardising care protocols by using the same materials
- Harmonising training on modern wound care based on a standard list of materials
- Facilitating WHO in establishing a list of essential materials for wound care
- Facilitating negotiations with industry to obtain better commercial conditions

• Facilitating development of materials for wound care in regions with limited resources

This kit mainly concerns:

- Organisations acting in the field of care in emergency situations (e.g., UNICEF, UNHCR, CICR, MSF, MDM, CARE, Oxfam, Save the Children)
- National health programs related to care of wound such as Buruli ulcer, yaws, leprosy, lymphatic filariasis
- Organisations acting in the domain of primary or secondary healthcare (health centres, district hospitals)

The wound care kit, described here, should be at the base of the update to the kits provided by specialist distributers in humanitarian crises. (7) This kit can also be a simple reference list to help NGOs or local authorities to organise their own pharmacy from local purchases. Furthermore many, if not all, of its components can either be bought in the limited-resource countries or switched to local products (e.g., shea butter instead of petroleum jelly).

On the initiative of the World Alliance for Wound and Lymphedema Care (WAWLC) and WHO, consensual work was carried out to define the content of this new kit. This task group, bringing together people experienced in the field of wounds and emergency care in regions with limited resources, was brought together notably by Internet consultations and in workshops organised at conferences of the EWMA (European Wound Management Association) in Copenhagen (May 2013), in Madrid (May 2014) and the CAWC (Canadian Association of Wound Care) in Toronto (October 2014).

# **General Description of the Wound Care Kit**

# Specifications

The kit brings together several features. The main objective is to realise quality wound care in an emergency situation or in a situation with limited resources in outlying health centres for first aid or even district hospitals. Therefore, the kit should:

- Contain the necessary renewable materials to make up to a hundred standard size (10 cm x 10 cm) wound dressings
- Be easy to use for nursing or medical staff
- Enable the principles of modern wound care to be met
- Be transportable by plane, ship, or road
- Be able to be transportable, in case of emergency, by plane in hand baggage and be able to easily pass through customs
- Be easy to store and quick to identify (expiry date, origin of the materials, name of the kit, list of contents)
- Be able to be stored stably in humid conditions and high temperatures (50°C) and have a shelf life of more than two years
- Respect the laws on medical materials, notably directives 93/42/EEC (7) concerning medical equipment

# Features of the kit

- It is presented in the form of a cardboard box that can be easily transported and stored. It weighs around 16 kg and has a volume of 0.1 m<sup>3</sup>.
- It is composed of a base unit (see Table 1) to which complementary units can be added, such as the "modern dressing 01" unit, defined later (see Table 2).
- The base unit comprises classic materials known by most carers and that can be found in most countries. It could thus be easily used locally. Furthermore, with its simplicity, if needed it could also be made up on site.
- The additional unit "modern dressing 01" comprises three so-called modern dressings that are not always available locally in countries. In addition, their use, although very simple, necessitates specific training.
- According to the resources available in the health structure, the wound care kit could be complemented by the disinfection kit and the dressing equipment kit, which already exist. (8)
- The wound care material necessitates, in part, sterilisation. If the structure does not have the necessary means of sterilisation, it is recommended that a sterilisation kit be procured. (9)
- The kit contains no substance that could be considered narcotic or psychotropic. The medicines that are included are not, therefore, subject to international inspection and there is no requirement for extra formalities in order to transport them.

# What is not included in the kit

- The kit is tied in with wound care itself. It is clear, however, that one of the basic principles of modern wound care is to take a holistic interest in the patient. Some items should be sought outside the kit, as the latter needs to be used in the context of a health structure.
- The battle against pain is most important, and analgesics use—minor or major—will need to be evaluated in each situation.
- Preventing localised or general secondary infections could sometimes necessitate oral or enteral antibiotics; however, it should be pointed out that most wounds do not need antibiotics. Simply cleaning the wound with clean water or possibly using an antiseptic is often sufficient.
- The kit does not contain items necessary for diagnosis and aetiological treatment of wounds.
- The kit contains neither anti-tetanus vaccine nor serum that could be recommended according to the state of the wound and its context.
- Having a PEP (Post-Exposure Prophylaxis) kit for staff members who may be accidentally exposed to blood is also recommended.

# Calculation of the quantities of items

- The benchmark measurement is a wound needing to be covered by a 10 cm x 10 cm dressing.
- The estimation of the quantities of material to include in the kit is based on the use of standard therapeutic directives and on the figures provided by organisations with experience on the ground.

• The quantities of medicines provided will only be sufficient if the consultants follow the standard therapeutic directives.

However, it should be stated that the wound care kit was not conceived for the treatment of severe wounds or wounds from major surgery, which require recourse to second-level hospitals.

# Contents of the kit

WHO has defined a list of essential medicines, including medicines that respond to the priority health needs of a population. They are selected according to the prevalence of diseases, safety, efficacy, and a comparison of the cost-efficiency ratios. They should be permanently available in the context of operational health systems, in sufficient quantity, in a suitable galenic formulation, with quality assured and at an affordable price at an individual level such as at that of the community. (10) This list of essential medicines, revised every two years, is an effective tool for rationalisation of the distribution of medicines.

Unfortunately, there is no similar list for medical materials. Initially the group thus established a list of essential medical materials for wound care containing 42 items (Tables 1, 2, and 3).

The objective of including these essential materials is to ensure wound care meets the modern principles of wound care that we could summarise in six points:

- 1. Protect the wound from trauma
- 2. Perform debridement in case of necrosis or fibrous tissues
- 3. Manage moisture balance of the wound
- 4. Manage the infection when present
- 5. Control oedema and lymphoedema
- 6. Prevent disability

This list of essential materials has been reduced to 26 to make the kit more realistic. This limitation of the number of items has been done principally for two reasons:

- A pedagogic will to promote simple protocols
- Ease of management

With regard to the kit currently used by humanitarian organisations, 19 new items have been introduced:

- Non-sterile, non-woven gauze
- Thick, sterile tulle
- Polyurethane transparent film
- Cohesive bandage
- Under-plaster bandage, wadding
- Petroleum jelly
- Silver sulfadiazine
- Sterile ophthalmic saline solution
- Liquid soap
- Hydro alcoholic solution for the hands
- Scalpel blade

- Scalpel with sheath, disposable
- Single-use drapes

In addition, some items enabling management of waste and good patient follow-up have been added:

- Pen
- Basic, easy-to-use wound care procedure information
- Patient follow-up chart
- Paper rulers
- Box for disposing of sharps
- Plastic rubbish bag

The complementary "modern dressings 01" unit for the wound care kit comprises three items:

- Alginate
- Hydrogel
- Polyurethane foam (hydrocellular)

The kit contains a reminder of the therapeutic directives regarding wound care, a list of kits available as complement to the wound care kit, and a list of addresses of kit suppliers.

To understand the convenience of the new kit, we have noted in the tables the use of each item with respect to the six basic principles of wound care as highlighted in the introduction of this chapter (see Tables 1, 2 and 3). Table 1: Composition of the basic wound care kit

	Essential materials	Items present in the former dressing kit	Protect the wound from trauma	Perform debriding in case of necrosis or fibrous tissues	Manage moisture balance of the wound	Manage infection when present	Control oedema and lymphoedema	Other (e.g., hygiene, immobilisation, analgesics)
	Gauze and dressings							
1	Sterile, non-woven gauze	Х	X		X			
2	Non-sterile, non-woven gauze Thick, sterile tulle		X	Y	X			
3 4	Polyurethane transparent film		x x	X	x x			
-	Bandages		~		~			
5	Cohesive bandage						х	
6	Under plaster bandage, wadding						X	
7	Crêpe bandage	х	х					
	Creams and ointments							
8	Petroleum jelly			х	х			
9	Silver sulfadiazine			х	х	х		
	Таре							
10	Plaster 2 cm	х	х					
11	Plaster, roll 10 cm	х	х					
	Hygiene and disinfectants							
12	Sterile saline solution			Х				Х
13	Liquid soap					Х		
14	Povidone iodine, 10%, solution	Х				Х		
15	Hydroalcoholic solution for the hands							х
	Materials							
16	Single-use non-sterile gloves	Х						х
17	Scalpel blade			х		х		
18	Scalpel with sheath, disposable			Х		Х		
19 20	Syringe 20 ml Single-use drapes	Х		Х				х
								Х

cont'd.

	Stationery				
21	Marker				х
22	Easy-to-follow procedure				x
23	Patient follow-up card				х
24	Ruled paper				х
	Waste management				
25	Sharp-edged box				х
26	Plastic rubbish bag				x

# Table 2: Supplementary "modern dressings 01" wound care kit

Essential materials	Items present in the former dressings kit	Protect the wound from trauma	Perform debriding in case of necrosis or fibrous tissues	Manage moisture balance of the wound	Manage infection when present	Control oedema and lymphoedema	Other (e.g., hygiene, immobilisation, analgesics)	
Gauze and dressings								
Alginate		x	x	x				
Hydrogel		х	х	х				
Polyurethane foam (hydrocellular)		х	х	х				

Table 3: Supplementary items in the lists of essential materials for wound care but not contained in the wound care kit

Essential materials	Items present in the former dressings kit	Protect the wound from trauma	Perform debriding in case of necrosis or fibrous tissues	Manage moisture balance of the wound	Manage infection when present	Control oedema and lymphoedema	Other (e.g., hygiene, immobilisation, analgesics)
Gauze and dressings							
Non-adherent dressing		х					
Super-absorbent dressing		х		х			
Charcoal dressing		х	х				х
Dressing with PHMB		х	х		х		
Bandage				_			
Light cotton bandages		х					х
Short-stretch bandage		х				х	х
Tubular bandage		х					х
Creams and ointments							
Corticosteroid cream						x	
Hygiene and disinfectants							
5% aqueous chlorhexidine solution					х		
Povidone iodine ointment					х		
Disinfection of surfaces							х
Materials							
Lidocaine injection							х
Lidocaine spray							х

## What is the cost for the new kit?

The cost of the former dressing kit was around 200 euros for up to 100 dressings—about 2 euros per dressing. The cost of the new wound care kit is currently estimated at between 220 and 240 euros but would need to be reduced for mass production.

In an emergency situation a high cost of 4 euros per dressing is acceptable, but for routine scenarios, in a context of limited resources, a lower cost is desirable. A cost of 1 or 2 euros per dressing would be an attractive objective. Several routes for reducing the cost can be envisaged and are under discussion:

- Agreement with producers of differentiated policies of price for countries with limited resources
- Purchase of kits in part subsidised by specific funds
- Work with pharmaceutical companies to have cheaper packaging
- Fostering competition between producers
- Procurement from producers in emerging countries

# **Development of the Wound Care Kit**

Work within WALWC is underway to test the wound care kit. After the production of a first prototype, 18 preseries kits will be produced and sent to three reference hospitals. Once tested, and based on these observations, the necessary adaptation of the kit will be made before a dialogue with the medical device and/or pharmaceutical industry takes place for industrial production.

The kit contains a form that will enable users to give their comments. Distributors and users will be invited to give their comments and recommendations with a view to the next edition.

To optimise diffusion and use of the kit and a modern approach to wound care suited to contexts of limited resources, various actions are underway:

- Training sessions, both face-to-face and over the Internet
- Guidelines currently under publication, notably within MSF
- Validation request regarding the list of essential materials for wound care with WHO
- Definition of quality criteria for materials for wound care in order to facilitate choice of procurement sources

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# **Case Studies**

# Cases Study 1: From Disaster Arises Hope: Wound Care in Haiti After the Earthquake

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January 12, 2010, marked a day of disaster in Haiti. A massive earthquake killed over 300,000 people, injured many more, and destroyed most of the infrastructure of the island. The country was overwhelmed by the devastation and unable to manage the injured. The hospitals were destroyed or significantly damaged, and many of the healthcare providers were killed, injured, or managing family members injured in the event. The world responded by sending healthcare teams to treat the injured and help in any way possible. Through their efforts, many were treated and saved. The many wound care volunteers were organized into a cohesive, effective team of medical professionals providing good care for the many patients with wounds.

As the situation seemed to stabilise after the disaster and volunteers began going home—leaving more and more of the healthcare to be provided by the local providers—the Minister of Health in Haiti and the Medical Association of Haiti felt that Haitian providers were in need of additional training in wound care to be able to manage the huge number of patients with wounds. They contacted the World Alliance for Wound and Lymphedema Care (WAWLC) to provide that training. (1) A program was provided the first week in July 2010.

After the training was done, questions about where that care could be provided arose. The Mevs Hospital and Clinic, with the help of the University of Miami and Project Medshare, stepped forward, and a small wound clinic started to be the centre of wound care in the area. Developing a wound treatment program that included training physicians and other healthcare professionals to work in the clinic, finding sustainable sources of supplies, and securing a source of funding seemed to be major obstacles. Could this even be accomplished under the circumstances?

Several of the general surgical residents at the Hôpital de L'Universite D'Etat D'Haïti in Port-au-Prince under the direction of Dr Franck Telemaque expressed interest in having additional training in wound care. It was felt that training could be accomplished with Dr Terry Treadwell at the Institute for Advanced Wound Care in Montgomery, Alabama. Dr Adler Francius was selected to be the first trainee. After spending six weeks training in Montgomery, Dr Francius returned to Port-au-Prince and became the medical director of the wound clinic at the Mevs Hospital. Additional residents have been trained since then and are waiting to take their place in wound care programs in Haiti.

Finding a source of sustainable supplies for the clinic was the next big task. Members of industry had been extremely generous in providing wound care supplies after the earthquake, and this generosity continued for the clinic; however, we knew there would be a limit on what could be provided. Major financial support was needed for the long-term sustainability of the wound program. Dr John Macdonald, through contacts in Port-au-Prince, Haiti, and Ft Lauderdale, Florida, was able to find initial sponsors who have been extremely generous in supporting the wound clinic at the Mevs Hospital. As with everything, there is a continued search for funding since it cannot be expected that one or two very generous people can fund the program over the long term.

The wound clinic at the Mevs Hospital in Port-au-Prince has been a success due to the hard work of many local people and volunteers. As many as 60 patients per day are being treated at the clinic. Unfortunately, the work is not done. As long-term support for Mevs is being sought, other wound clinics are needed. A wound clinic has been established at the main teaching hospital, Hôpital de L'Universite D'Etat D'Haïti in Port-au-Prince under the direction of Dr Franck Telemaque and Dr Jean Fritz Jacques, but support is severely lacking. The Minister of Health has requested the establishment of wound centres and teaching programs at all of the government hospitals in Haiti. As we begin to help with this project, the major issue will be sustainable support and funding.

The establishment of wound centres in countries with few resources can be done but, as can be seen from the experience in Haiti, there must be local people interested in supporting them. There must be support and participation by the healthcare providers to be educated in modern wound care and to be the "champions" for wound treatment in their facilities. There must be support by the hospital, the administrators, the government, and the community if these projects are to be sustainable. If we all pitch in to get the job done, these are only bumps in the road and not mountains to climb!

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# Case Study 2: Setting up an Integrative Medicine Clinic for Self Care of Lymphoedema in Indian Villages

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## Introduction

The morbidity of lymphoedema makes family wage earners both poor and dependent. The only public health program of proven effectiveness, in any endemic country, is self-care integrative medicine (IM) for lymphoedema of any aetiology, including lymphatic filariasis (LF). The following describes our experience of treating 4000 patients in a medical centre at Kasaragod, Kerala, and in Indian endemic villages. (1)

## **Conduct a Rapid Survey to Estimate the Prevalence**

Begin with a strategic liaison with government health departments to obtain an estimate of disease prevalence. In most developing nations exact data on incidence and prevalence are difficult to obtain due to prevailing disorganisation; village homes are often not registered for house numbers, there are owners of the same name, and there is internal migration. A rapid survey for secondary data can be conducted in collaboration with district-level health officers, malar-ia-filaria officers and public health managers. One easy method to develop the initial contact with these unskilled public health workers (2) is by attending their monthly review meeting of Accredited Social Health Activists (ASHA) and office holders of community-based organisations.

# Establish Community-based Integrative Medicine Clinic (2)

We recommend forming a lymphoedema care team in each centre led by an Ayurvedic doctor, who has a first degree at one of the few Ayurveda universities or health universities, and with a general nurse and midwife. A graduate in medical social work or a local social worker can support field activities. Healthcare assistants should also be locally recruited and then trained as community lymphoedema care workers. The treatment centre can be established in a minimum area of 800 square feet partitioned to provide different components of the IM. In resource-limited centres this can be established in vacant buildings or with little renovation in those attached to primary health centres.

# Create Lymphoedema Camps to Target Community Awareness (3)

Non-residential medical camps should be organised in endemic villages near the patient clusters. Each camp should have an inaugural public function involving bureaucrats, health inspectors, social and people's representatives of the panchayath or municipality, a patient education class (preferably as part of inaugural function), a skin care demonstration, and a demonstration of simple yoga exercises designed for lymphoedema. (4) Patient information may include details of aetiology, clinical leads that differentiate LF from other forms of lymphoedema, associated co-morbidities, and environmental factors that complicate lymphoedema. A quality-of-life tool specific for LF should be administered where possible. (5) Nurses and patient peer volunteers using audio-visual tools help to explain bacterial entry points (BEP) and self-care techniques. All healthcare workers—especially ASHA and patients and their attendants—should be encouraged to practise and learn simple yoga exercises and other self-care activities demonstrated during lymphoedema camps. A check-list-based guideline may be used to identify patients with recurrent cellulitis and other associated comorbidities that may lead to complications such as septicaemia requiring supervised care. Such patients should be referred to higher centres.

# Treatment Protocol (1)

In our centre lymphoedema is treated as an outpatient procedure. Patients attend a counselling session before deciding on treatment. During treatment, at discharge, and during every follow-up attendance the counsellor conducts similar sessions to emphasise the treatment procedures and improve the adherence and care of BEP.

The initial intense phase of treatment and coaching for self-care is followed by three monthly follow-ups. The protocol is an integrated treatment combining the therapies of both biomedicine and the Indian Ayurveda system of medicine and yoga, delivered in such a way as to be acceptable to local communities. Passive exercises coordinated with deep breathing techniques of yoga and modified traditional ayurvedic oil massages (nalpamardhi thailam is commonly used) as local skin treatment measures replace physiotherapy, manual lymph drainage and commercial biomedical emollients. BEP are treated using modern dermatology drugs (ointments for fungus infections or eczema) when necessary.

Data collection includes weekly measurement of limb circumference or water displacement. Patients with a smart phone make use of a lymphoedema-and-wound-care-specific mobile app to give feedback and get treatment guidance.

Once the treatment plan is decided patients receive an initial two weeks of supervised treatment. The sequence of treatment components that each patient follows is: skin wash with soap, soaking the limb in an herbal phanta solution, a set of yoga exercises (called pre-IMLD yoga), Indian manual lymph drainage (IMLD) (see Figure 1), compression bandaging, and post IMLD yoga. The steps of

treatment are the same for all grades of patients although its duration may vary as limb size decreases.

Figure 1: Indian manual lymph drainage, a modification of traditional Ayurvedic oil massage, is done for 20 minutes until 50–70 ml of medicinal oil is absorbed by the skin.



## **Patient Education**

Repeated emphasis on adherence to treatment and care of BEP is done through counselling sessions. Focused group discussions on how to do the multiple selfcare steps at home are conducted during the first 14-day outpatient care session. Patients are taught how to use the low-cost, locally available materials for self care. The locally available materials include Ayurvedic oils available in nearby Ayurvedic pharmacies, cold-cure foam, cotton cloths, finger bandages and micro cellular rubber or moulds available in local markets, and biomedical drugs for BEP care available in local medical stores or primary health centres. Compression bandaging (long stretch) is the most expensive part of treatment.

## Outcomes

The International Society of Lymphology consensus outcomes should be aimed for to determine the success of lymphoedema treatment. Several studies with evidence grade II-1 (of US Preventive Services Task Force) showed that low-cost IM is effective as self-care treatment in endemic communities. A quality-of-life tool for Indian LF patients (5) was shown to be superior to the World Health Organization's WHODAS.

In a community-based lymphoedema treatment clinic at Gulbarga of Karnataka and Alappuzha of Kerala provinces of south India, QoL changes showed highly significant results in mobility, pain and discomfort, psychological health, social relationships and disease burden. Overall QoL scores improved from 77.8 to 91. 8 with 99% confidence interval range of 9.4–12.4. The concordant limbs showed volume reduction of 45.6% at the third follow-up (see Figure 2), while non-concordant limbs reduced by 8.7%. Inflammatory episodes at the interval of three months before recruitment to treatment were present in 37.5% patients and reduced to 16% at the end of 3.5 months of treatment. Although reduced significantly, there was a continued presence of BEP-induced inflammatory episodes. All results were significant at 1% level. (2) Further, Aggithaya et al. have shown that simplified integrative treatment improved the overall QoL of their concordant patients in three endemic districts of Kerala. (3)

Figure 2: The volume change after treatment in a community-based integrative medicine lymphoedema treatment clinic in Gulbarga district of Karnataka, India.



This cost-effective integrative treatment protocol incorporated all the recommendations of an international consensus statement on care of lymphoedema. It adapted all the principles of primary healthcare: community participation, intersectoral coordination, appropriate technology and equitable distribution and can be adapted globally. Lymphoedema care is not part of health insurance systems anywhere. Being low-cost, this low-technology self-care treatment protocol ideally suits government-subsidised insurance systems such as Ayushman Bharat of the Indian Government.

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