WOUND AND LYMPHOEDEMA MANAGEMENT

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FOREWORD

This document is designed to assist health-care providers who manage chronic wounds and lymphoedema. The aim is to assist in achieving better outcomes. It describes methods that can be adapted to various levels of the health-care system depending on the country and available resources.

This document is not intended to serve as a standard textbook on wound care and lymphoedema management. Adherence to it will not ensure a successful outcome in every case, nor should it be construed as including or excluding proper methods of care. Ultimate judgement regarding a particular method and material to use must be made by the health-care provider in the light of the clinical findings in the patient and the available options for management.

ABBREVIATIONS

AAWC	ASSOCIATION FOR THE ADVANCEMENT OF WOUND CARE
ABPI	ANKLE BRACHIAL PRESSURE INDEX
BU	BURULI ULCER
CDP	COMPREHENSIVE DECONGESTIVE PHYSIOTHERAPY
CT	COMPUTED TOMOGRAPHY
CVI	CHRONIC VENOUS INSUFFICIENCY
CWATS	COMPREHENSIVE WOUND ASSESSMENT AND TREATMENT SYSTEM
DEC	DISEASE-ENDEMIC COUNTRY
DFU	DIABETIC FOOT ULCER
DIME	DEBRIDEMENT, INFECTION OR INFLAMMATION, MOISTURE BALANCE AND EDGE EFFECT
DVT	DEEP VEIN THROMBOSIS
EPUAP	EUROPEAN PRESSURE ULCER ADVISORY PANEL
GIEESC	GLOBAL INITIATIVE FOR EMERGENCY AND ESSENTIAL SURGICAL CARE (WHO)
HBOT	HYPERBARIC OXYGEN TREATMENT
LAS	LYMPHOSCINTIGRAPHY
LCD	LEAST COMMON DENOMINATOR
MLD	MANUAL LYMPHATIC DRAINAGE
MRA	MAGNETIC RESONANCE ANGIOGRAPHY
MRSA	METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS
NPUAP	NATIONAL PRESSURE ULCER ADVISORY PANEL (USA)
NPWT	NEGATIVE PRESSURE WOUND THERAPY
PU	PRESSURE ULCER
RA	RHEUMATOID ARTHRITIS
RNAO	REGISTERED NURSES ASSOCIATION OF ONTARIO
SSI	SURGICAL SITE INFECTION
VU	VENOUS ULCER
WHO	WORLD HEALTH ORGANIZATION

PREFACE

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Wound healing and lymphoedema have long histories, extending some thousands of years, in oral and written traditions. The reader of this document will find an enormous range of facts and concepts developed mostly during the last two or three decades. Significantly, these topics have been recognized as worthy of workshops, seminars, international congresses and inclusion in the curricula of schools of medicine and allied health professions. This attention reflects a better understanding of genetic and environmental research, as well as applied research, into dressings and medical devices.

The reader may find the range of topics covered somewhat overwhelming. No single discipline is expected to absorb the information contained herein. Indeed, throughout the discussions the writing group was aware that this document should be used at all levels of health care, and that at each level some of the information contained will be selected and some may be shelved. All authors were well aware that resource-poor countries may not be able to enact some specific aspects of best practices in this document.

In describing "best practice" in elite (usually urban-based) units, we are referring to "gold standards" and "evidence-based practice" (EBP). These are increasingly based upon randomized controlled trials (RCTs). We are identifying high technology, which is desirable but expensive. Ideally, such technology will be available in at least one centre in every nation or region, and access will extend, as much as possible, to all that are in need.

In making essential health care available to all, that which is common must be addressed at the most peripheral level. Self-help in the home is desirable, and the low technologies required should be available locally at low cost in a sustainable system of provision. Since patients use more than one system of medicine, this document acknowledges all local systems that are used. This effort maintains focus on safe and effective remedies, while eliminating the unsafe and reducing availability of ineffective remedies. Such an approach should contribute to poverty alleviation. Necessary knowledge must be taught. For example, remedying malnutrition by the growing of nutritional herbs at everyone's back door requires identification, optimal usage and preparation, dosage, and acceptance.

We intend that tertiary care will consist of a team of specialists providing their expertise within the focus of a vertical programme, thus providing an integrated approach in the hospital setting. The knowledge base of this team should filter down to lower levels at flexible rates of change and transfer from the vertical to

the horizontal. It should prevent overwhelming of the tertiary level by common problems that can be managed at a lower level. The primary aim of the tertiary team should be to enhance knowledge and practice at the lower level, receiving only what cannot be managed at that lower level. How far the rich knowledge base of a vertical programme penetrates to a lower level will depend on the prevalence of the disease covered by that knowledge.

The horizontal systems of delivery of care to the primary or general health services cannot cope with the receipt of packages of best practice from each discipline. For example, leprosy is rich in handbooks, but they will collect dust in a health centre seeing only one case every four years. However, if that same centre treats many diabetic foot ulcers, the patient with leprosy can receive the same treatment. It is clearly unnecessary for lymphoedema due to different causations to require separate instruction manuals from the lymphatic filariasis expert, the podoconiosis expert, and the post-surgery or radiotherapy experts. The scabies, pyoderma and fungal diseases that overwhelm all skin services are better managed at the lowest level with minimal instruction. Improved confidence in managing the commonplace allows rare diseases to be identified as uncertainties suitable for referral to a higher level.

Information in this document usable at low cost at the village and home levels should be made available with a mind to delivering it in simple one-day courses. We believe that a focus on the wound and all its complex features can be simplified, and common denominators can be selected. We believe that many of these common denominators have universal value as gems of public knowledge. Such coordination is a valuable topic in public health in an era threatened by increasing poverty, climate change, and the mobility of populations facing emerging epidemics and neglected diseases. If all people knew how to wash while conserving water and applied this low technology to wound care in an optimal way, it would provide a no-cost contribution to "Health for ALL".

Knowledge of wound and lymphoedema care is mostly new. The importance of patient concordance, adherence and sometimes compliance is also newly recognized. If we expect much of the knowledge in this document to be used in "home-based management", we should recognize the advances being made in the descriptions of these health problems. We should use terms such as "disabilities, impairments, and handicaps". Patients and those who provide for them appreciate such language. We also must recognize that patients are worthy members of the "TEAM". Indeed, without patient participation, optimal use of the knowledge in this document will not happen.

The Lymphatic Filariasis Workshop on Disability Prevention for Field Managers, sponsored by Handicap International in Accra, Ghana, 10–12 July 2007, brought together researchers, managers, and clinicians who reported on lessons learned in their respective programmes. Although limited resources were a general constraint in every country represented, the consensus held that integration of mass drug administration (MDA), parasite and vector elimination measures, and wound management services had positive potential. At a follow-up meeting held in Geneva on 13–14 September 2007, a working group explored the potential for wound and lymphoedema management to serve as a model for integration across diseases at both the service and system levels. Thus, the Working Group on Integration of Wound-Lymphoedema Management across Diseases in Resource-poor Settings was formed (WG-IWLM). Integration, in the context of the meeting, was generally defined as an alignment of concurrent activities, across and within organizations and medical specialties, in pursuit of a shared vision and common goals. Additionally, integration was interpreted as the development of coordinated interventions, by multiple organizations, to address targeted diseases at the clinical, community and point-of-care levels. In addition to identifying common services and process activities, another goal of this initial meeting was to develop funding strategies for planning and implementation.

This document is one of the by-products of that initiative-building meeting. The editor John M. Macdonald, who at that time was President of the American Association for the Advancement of Wound Care (AAWC), solicited contributions on best practices in aspects of wound and lymphoedema management from a panel of international experts. In attendance at the follow-up meeting in Geneva, Switzerland (6–7 March 2008) were the document authors, representatives of the World Health Organization (WHO), Health Volunteers Overseas, Médecins sans Frontières, Netherlands Leprosy Relief, Handicap International, The Royal Tropical Institute of Amsterdam, and Hôpitaux Universitaires de Genève. Revisions of the original draft were proposed during this meeting. A third meeting of the working group was held in October 2008.

This document is intended to serve as a resource for developing disease-specific guidelines and corresponding training curricula for use in resource-poor settings. It is intended to do the following:

- describe general principles for the clinical approach to treating chronic wounds and lymphoedema of all etiologies;
- following the basic principles of wound management:
  - — enhance systemic conditions;
  - — protect the wound from trauma;
  - — promote a clean wound base and control infection;
  - — maintain a moist wound environment;

— control peri-wound lymphoedema/oedema;
- consider basic clinical indicators for monitoring and evaluating wound and lymphoedema management interventions;
- serve as the foundation for the development of clinical practice guidelines in limited resource settings and curricula designed to promote the integrated management of wounds and lymphoedema at all levels within the health-care system utilizing the public health approach;
- describe the potential impact of wound and lymphoedema management integration across diseases targeted by WHO.

The document is not intended to be a thorough review of all related diseases or pain management, neuropathy, modern dressings and pharmaceuticals, nor is it intended to address acute wounds, surgical and traumatic, or burns. The document will not include in-depth recommendations for the WHO-targeted diseases of Buruli ulcer (BU), leprosy, or lymphatic filariasis (LF). Best practices for neglected tropical diseases (NTDs) are works in progress by definition. The document will be informed by continual input from disease-endemic countries (DECs), and addenda will provide opportunities for inclusion of approved novel drugs, diagnostic tests and other products developed in DECs. The clinical practice guidelines and curricula derived from the document are intended to serve:
- health-care and non-health-care personnel at multiple levels;
- programme managers;
- as a reference tool to aid policy-making at the ministries of health and education;
- as a general guide for programme developers;
- as a good practice guideline for allied health-care personnel at point of .

Admittedly, the best practices herein were developed in socioeconomic settings that do not exist in sub-Saharan Africa, rural Asia, and much of South America. The objectives are to assist in the development of approaches for preventing, diagnosing, and treating and managing chronic wounds and lymphoedema that are applicable, acceptable, and affordable by DECs, approaches that can be readily integrated into the existing health services. The organizations and programmes involved in this initiative focus on the health problems of the poor. The principles embodied in this document are expected to translate into good care practice guidelines for use at the district and community levels.

WOUND HEALING IN THE DEVELOPING WORLD

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The term "wound healing" embraces all types of wounds, burns, and ulcerations. Complete wound healing includes restoration of function hardly ever achieved in those disfigured by wounds, especially when one includes the appearance of the skin or absence of an appendage. Most texts on wound healing are becoming more complex with every edition ([1]) and emphasize gold standards that are achieved only in urban hospitals of major cities in the developed world. This is a small, elite and very expensive model. Despite rapid urbanization, much of the world is still rural, most wounds occur away from the elite centres, and most populations in the world are poor. The term "developing world" is sometimes equated with tropical countries, but in fact applies also to non-tropical countries and implies recovery from being resource-poor ([2]). This document addresses wound healing applicable to situations such as rural Oxfordshire (United Kingdom), communities of rural Africa, villages of India ([3]), the Australian outback, or the Indian villages of the American subcontinents, as well as mobile populations.

The primary health-care concept initiated at Alma-Ata and periodically reviewed by the World Health Assembly ([4]) is a system theoretically well suited to rural communities. It is a system supported by the local community to address its health-care needs using available resources and appropriate technology. Patients are treated in their homes or as close to them as possible. Treatment should be affordable and adequate. Because the occurrence of wounds is unpredictable, any resource for their management should be continually available. The health-care worker in the rural village should have appropriate knowledge, equipment, and transport. While delivering health care, he or she should be above all an educator. All useful sources of knowledge should be integrated in the programme to provide the principles of hygiene, including the knowledge of the traditional healer or major systems of medicine used in India and China. If a community can support a pharmacy, the staff should be well informed about the early management of wounds, and encouraged to advise their patrons as needed. It is important to understand local health problems in the light of local customs and enlighten them by bringing them to the attention of collaborations that use all the expertise available. That is the concept.

The challenges of wound healing in the developing world are connected to the reasons for the failure of health-care delivery, which are linked, in turn, to poverty and social unrest. Conflict and the excesses of climate change can ruin the best-made plans to manage with limited resources. Internal displacement and the general mobility of populations add considerable unreliability to follow-up.

Much of the world is resource poor, but every nation should aim to have widely distributed primary health care with access to secondary and tertiary levels of health care, all achieving good practice standards. These standards are based on evidence and carried out as a consequence of effective training and evaluated practice. Wound management has its place at every level from home-based care of wounds, to the district level of the health service and to the best equipped and staffed hospital in a nation's capital city.

It is our purpose to add to every community locally available, sustainable management of wounds at the lowest possible cost. The focus will be on educating carers whose role is to achieve tissue viability or optimal wound care. Such carers can be found near the patient's home, at the door of the village health centre, or tilling the herbal garden of the local healer. Ideally, their training will be about the best methods in wound healing, while being rich in understanding of local belief and culture.

Quality-of-life issues, personal preferences, priorities, and motivation are important considerations. Caregivers must want to reduce wound odour and relieve pain, and help the mobility of their patients, encouraging them to be well groomed, allowing them to maintain adequate hygiene by bathing and, above all, making them welcome so that they can be embraced, marry, and be employed.

To achieve these goals, complete wound healing is necessary, approaching normal colour, contour, and function. Management of wounds where poverty is unalleviated requires missionary zeal for some basic objectives. There must be food and drink for the patient, protection from flies, antisepsis, good surgical technique, a temperate environment, and a knowledge of the requirements for achieving one's potential in the community.

1.1 NECESSITY OF LOW-COST WOUND MANAGEMENT

Further developments are dependent on adequate research funding, an increasingly scarce resource. Yet many low technologies are undervalued, underfunded, and tend

to be neglected. It is these low technologies, available locally and sustainable at low cost, that form the foundation stones of wound management in resource-poor settings.

1.2 CAUSES OF DELAYED WOUND HEALING

Many wounds of healthy tissues heal at a predictable rate. However, there are three main groups of essential factors, without which healing is delayed. The first is the need for an adequate blood supply without the interposition of space-occupying clots, necrotic tissue, foreign bodies or soft tissue infection ([5]).

The second group of factors relates to the general health of the wounded. They are systemic factors such as anaemia. Malnutrition often is insufficiently understood or recorded. Malnourished skin has impaired function and can show signs of disease even when not wounded. Kwashiorkor, scurvy and pellagra are examples. Where wounds occur in a population with manifestations of malnutrition, it is likely that the whole population suffers from subclinical malnutrition.

The third group of factors contributing to non-healing relates to access. These factors include the distance and nature of the terrain travelled to seek help, and the kind of transport. When reached, how available, knowledgeable and skilled are the carers? To what extent are the wounded affected by issues of stigma that make them unwelcome in society and unable to participate? Do they have adequate resources? Is the equipment appropriate and sustainable? Is the treatment beyond the means of the patient?

A contrast between resource-poor and resource-rich people is the access to family. In the developing world the extended family and the generous time made available by family members is a rich resource because such carers are willing to learn caring skills. Unhappily, this resource is diminishing. One-child families and women involved in full-time work make care of the elderly sick by family members a major challenge even in crowded urban communities. The demographic changes of the HIV / AIDS epidemic can eliminate this resource in some communities.

1.3 EVIDENCE-BASED WOUND HEALING

Good standards of practice need a strong evidence base, which is what Archie Cochrane termed Effectiveness and Efficiency ([6]). Although many novel drugs began

as traditional wound-healing agents growing in a pot on the windowsill of the slum or gathered by relatives of the wounded in the forest ([7]), the safety of commonly used agents should be demonstrated before recommending them. The large populations of India and China rely on their traditional systems of medicine and export them widely. Both nations are applying modern biomedical tools to test the safety and effectiveness of traditional medicinal products. Training the prescriber is another challenge. This document provides a background for the design of future training manuals and adds weight to the growing field of public health skin care ([8]).

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INTEGRATING THE CONCEPT OF WOUND CARE, THE IMPORTANCE OF WOUND TREATMENT, WOUND TREATMENT EDUCATION, AND THE WOUND CARE "TEAM" IN THE DEVELOPING WORLD

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Since the first man injured himself, there has been debate over the best, most efficient, and most cost-effective way to treat a wound. Many years later the debate still rages. The treatment landscape of acute and chronic wounds worldwide varies so greatly that establishing a single, uniform, best way to manage wounds is not likely to be possible. Some approaches to managing wounds in countries where there are many resources and health-care providers will most likely not apply to resource-poor areas with few, if any, trained health-care 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 our most precious resource in every country, our 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 wound healing, based on better understanding 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, all of which influence the process. We recognize these multiple influences as co-morbidities that require control if proper healing is to occur. Increasing medical specialization has multiplied the numbers of medical and surgical specialties and of paramedical personnel involved in providing optimal care for patients with wounds ([1-6]).

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 assure appropriate footwear. 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 progress, and in helping the patient return to a productive life. Dieticians and diabetic 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 educating the patient or caregiver in 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 ([4]).

Evidence shows that patients with chronic venous wounds treated in a centre specializing 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 specialized clinics healed, compared with 12% of the patients treated at other locations. After 24 weeks of therapy, 68% of the patients in the specialized clinics were healed, compared with 29% of the patients treated at other locations. The studies also show that the recurrence rate of the ulcers was lower for patients treated in the specialized clinics. Recurrence rates were 23% and 43%, respectively, at 6 months post-healing, and 23% and 54% at 12 months ([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 that the recommended therapy can be accomplished by the available caregivers. If available 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 for diagnostic testing and follow-up appointments. Other special needs of the patient and family can best be met by a team of caring people with individual talents and abilities ([3, 5]).

In developing and resource-poor 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 of 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" in the treatment of wounds, having the knowledge and expertise to treat difficult wounds ([8]). These centres should become the educational centres spreading wound care education throughout the country and be the resource centres for treatment of the most difficult wounds.

Unfortunately, establishing centres of excellence in wound management may be difficult unless there is a change of attitude among the health-care providers. Many physicians view chronic wounds with disinterest and frustration and, when required, treat wounds with outdated and ineffective therapies or pass the patients on to anyone who will 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 very hard-working 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 at the centre and practically had to beg physicians to assist them with difficult patients. The importance of wound care must be stressed to the medical staff of each major health centre. Everyone, including health-care workers, leaders of the country, and community leaders, must understand how good wound care can improve the lives of their people and be aware of the fact that it will be economically advantageous to treat these people and return them to the workplace or, at least, to a productive, self-reliant life.

After one of my lectures in South America, a physician asked the question, "Why should we bother to treat patients with pressure ulcers? They all die anyway." To say the least, I was stunned by the question, but I quickly realized he was questioning the use of precious resources to treat patients who were most likely to die despite therapy. I explained to him that not all pressure ulcer patients were at the end of their lives, and many could recover if the ulcers were treated appropriately and not allowed to be the cause of the patient's death. I also tried to explain the concept of quality of life and of making a patient's last days more comfortable even if survival was not possible. I explained how this could be done without using many resources. He replied that he had never seen a pressure ulcer heal under any circumstance. One can see from this exchange that the need to educate caregivers in the current state of wound care and the benefits of good wound care is an important challenge.

What may be the best way to provide the 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. "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 health-care challenges in resource-poor settings" ([9]). The 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, evaluate carefully the ideas that may conflict with the current practices and values, consider the resources needed to implement the new ideas especially in resource-poor areas, and consider the educational and skill levels of the people providing the care in all locations ([9]). Without a thorough knowledge of the impact that the introduction of a new programme will have on the local people and the health-care providers, the programme will be predestined 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 five positive characteristics of the new programme, as described by Rogers ([10]).

1) The relative advantages of the new programme for the current practitioner must be made known.
2) The compatibility of the new programme with the current practices and beliefs must be stressed.
3) The simplicity of the new programme must be apparent.
4) The people adopting the new programme must be able to try all or part of the programme before committing to it completely.
5) The lack of hidden agendas and programme difficulties must be apparent to the people considering the programme.

Addressing these issues will increase the likelihood that the programme and its innovations will be embraced by the local people, whether in the city or in the country. After presenting the educational information, we must be able to bring together teams of people to discuss the new programmes 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 health-care workers to help formulate those plans and to spread the ideas and practices to their colleagues and patients ([11]).

In underserved areas, it will be necessary to identify the health-care provider for the village or area. Such providers most likely will not be a physician but a person who has been trained by the national health programme of the country. They might be 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 an expert in inguinal hernia repair is a former gardener at the hospital. He expressed an interest in helping a missionary surgeon in the operating room and became a proficient assistant. He subsequently was 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 ex-gardener and not the missionary surgeon. All it takes are people who are interested in learning about wound care and who have a heart for helping others.

Since the "multi-specialty wound team" in developing areas may be composed of people who have little formal education yet are willing to learn, the programmes we introduce must be understandable and easily implemented. We must work with them, training them, so that they can provide good wound care within their cultural and religious beliefs and with the resources available to them. We must work with them to "make do" with what is available. Wound care products used in some countries may be the only ones available. 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 all these issues, the programmes, no matter how well intentioned, will not succeed. Even teaching the people to help assume responsibility for their care (self-care) will make any of these efforts more successful ([13]). Attempts at major health initiatives using the skills of health-care 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.

The goal must be to establish at least one centre of excellence in wound treatment and wound treatment education within each country. Good wound management principles must be implemented at lower levels. This can only be done by identifying the local caregivers, involving them in wound care in an appropriate manner, and giving them the educational tools and resources to practise good wound care within the bounds of their religious and cultural boundaries. Monitoring and training evaluation must be integrated into every level if the objective is to make a sustainable difference in the lives of the people it is intended to help.

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GLOBAL IMPACT OF THE CHRONIC WOUND 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 health care 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 the chronic wound is well documented in North America. Each year, in North America, between 5 and 7 million chronic and/or complex wounds occur. International statistics giving the full picture of the prevalence, 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 American experience. This chapter aims to illustrate that chronic wound care is a great unmet need requiring government interventions and individual preparedness for significant improvement. Major textbooks now address these needs ([1]). This chapter will discuss the epidemiology and impact of the chronic wound in the USA and in selected resource-poor nations. The chapter will conclude with an overview of ulcers seen more commonly in resource-poor communities such as the Buruli ulcer and "tropical ulcers".

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, quality care intervention.

A wound can significantly affect a person's life. Wounds can lead to prolonged periods of disability, in addition to causing pain and discomfort. Chronic wounds often prevent a person from performing everyday activities as basic 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 USA account for the estimated loss of 2 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-poor nations. 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 specialized 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 wounds have medical legal implications. Increasingly, this has become a major problem affecting resource-rich and resource-poor countries ([4]). Wound healing, in Europe and in North America, has developed into a library of "grey" literature relating to litigation from medical defence societies. 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 ([5]). Chinese traditional medicine and Indian herbalists sometimes are required by law to practise with single active principles and consistent dosage. Required investigations are ever more stringent, approaching advanced biomedicine. For example, bonesetters practising without X-rays face greater liability in an increasingly technological society.

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 ([6-7]). 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. ([8]), "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."

The treatment of chronic and complex wounds is a significant burden on the health-care system and on the economy as a whole. In 2003, in the USA, 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 ([9-10]).

3.1 ACCIDENT 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 because of government legislation. Occupational safety regulations, non-inflammable clothing, furniture and housing are controlling factors. Fireworks restrictions are important. The accumulation of litter in public buildings with minimal control on smoking is another factor.

Pressure ulcers have received significantly more publicity in recent years since the American Department of Health and Human Services introduced a National Pressure Ulcer 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. Quite a few governments have legislated to improve bedding quality. In New South Wales, Australia, no long-term care home is permitted 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 barrier ([17]).

3.2 WOUND TYPE, INCIDENCE AND PREVALENCE IN THE USA

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 the integrity of the skin itself. The most common types of chronic wound are leg ulcers, pressure ulcers, and diabetic foot ulcers. Underlying medical conditions often cause chronic wounds. 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 USA population ages, the incidence of chronic wounds is expected to rise significantly to an estimated 5–7 million ([19]).

3.2.1 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 6).

3.2.2 PRESSURE ULCERS

Over 2 million pressure ulcers occur each year in the USA. One third of patients admitted to a critical care unit develop a pressure ulcer. Approximately 15% of hospitalized patients aged 65 or older develop a pressure ulcer during a 5-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]).

3.2.3 DIABETES MELLITUS

According to the American Diabetic Association, 20.8 million children and adults or 7% of the USA population 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 American Indians, 12.8%; and Afro-Americans, 13.3%. Diabetic individuals 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-poor 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.

3.3 WOUND TYPE, INCIDENCE AND PREVENTION IN OTHER COUNTRIES

3.3.1 INDIA

Wounds, and particularly chronic wounds, are a major concern for the Indian patient and clinician. Chronic wounds affect a large number of patients and seriously reduce their quality of life. While there are few Indian studies on the epidemiology of chronic wounds, one study estimated the prevalence at 4.5 per 1000 population. The incidence of acute wounds was more than double at 10.5 per 1000 population ([31]). The etiology of these wounds included systemic conditions such as diabetes, atherosclerosis, tuberculosis and leprosy. Other major causes included venous ulcers, pressure ulcers, vasculitis and trauma. The study report stated that inappropriate treatment of acute traumatic wounds was the most common cause of the chronic wound. In India, as in other under-resourced nations, the problem of chronic wounds is compounded by other demographic factors, such as low literacy rates, poor access to health care, inadequate clinical manpower, and a poor health-care infrastructure. Inadequate education and clinical training in the fundamentals of basic wound care greatly magnify the problem in India. India has had its first wound healing programmes only in the last decade. Major textbooks on wound healing have only recently appeared on the shelves ([32]).

In India, as in many developed countries, diabetic foot disease now results in major debilitating complications with severe morbidity and increased amputations. A high prevalence of neuropathy promotes recurrence of foot lesions, more than 50% after three years. Unfortunately, these chronic wounds are often inadequately treated ([33]).

India remains the nation with the highest and virtually unchanged new case detection rate of leprosy. Severe disabilities and ulceration are common, and the custom of begging and the prevailing caste system do little to improve rates of healing.

3.3.2 CHINA AND VIET NAM

The prevention and management of chronic skin ulcers in lower extremities continues to be a severe problem in China ([34]). Wound healing in these injured tissues is a major health-care problem with considerable socioeconomic impact. According to data from epidemiological studies, the incidence of chronic ulcers in surgical hospitalized patients in China is 1.5% to 20.3%. The site distribution of these

wounds varies with etiology. In one study, of the 580 wound areas in 489 patients, 366 or 63% were ulcers on the lower extremities. The principle etiology (67%) of ulceration is trauma or traumatic wounds compounded by infection. This highlights the need for traumatic wound prevention programmes. Diabetic ulcers, venous ulcers and pressure ulcers accounted for 4.9%, 6.5%, and 9.2%, respectively. The majority of these wounds were seen in farmers and other agricultural workers ([35]). China with its 1.3 billion population will add to the global statistics on emerging epidemics. The age- standardized prevalence of metabolic syndrome and overweight was 9.8% in men and 17.8% in women in a 2005 study ([36]).

China is the country with the greatest number of burns. China's many, mostly military, burns units achieve a very high standard of care and there is distinguished research into skin equivalents and stem cells. Access to data is through two leading journals, *The Chinese Journal of Burns, Wounds and Surface Ulcers* and *Burns* ([37]). This high level of care is mostly available for the wealthy city population. There are 900 million people living outside the cities who have much less access to care due to distance and poverty.

The treatment of burn victims in Viet Nam has steadily improved since the National Society for Burn Injuries was formed two decades ago ([38]). For twenty years, they had little access to therapies from outside Viet Nam and their research into herbals and the application of frog skin reached an advanced state. They were pioneers in the training of caregivers in the General Health Services. As a result, in many cases, the poor appear to have more affordable care than in neighbouring China.

China has a residual population of patients affected by leprosy. In Yunnan province, there are 120 leprosy villages and the prevalence of untreated ulcers is very high ([39]).

3.3.3 MEXICO

Diabetes is the third leading cause of general mortality in Mexico. Between 8% and 12% of the general population in Mexico, 4–6 million people, currently have diabetes. The number rises to 21% in people above 65 years of age. It is estimated that by the year 2025, Mexico will have the highest incidence of diabetes in Latin America and the seventh highest incidence of diabetes worldwide. Only 200 000–300 000 of the people with diabetes are believed to have the disease under control. Approximately 30% of Mexicans with diabetes do not know they have the disease. Diabetes is the chief cause of lower extremity amputations in Mexico. More than

75 000 legs were amputated in the year 2000. No statistics are currently available on venous or pressure ulcers in Mexico ([40]).

3.3.4 CAMEROON

In a recent study of 300 diabetic patients, the incidence of diabetic foot ulceration ranged from 25.6% (inpatient) to 11.1%. The authors stated, "Diabetes mellitus, a non-transmissible disease, is a worldwide epidemic, especially in Africa and Asia, the diabetic foot being one of the most severe and frequent complications. Its cost is among the highest of the diabetic chronic complications. The struggle against that burden relies upon the prevention (education of patients and caregivers, early detection of the lesions) and upon a multidisciplinary approach and treatment. In sub-Saharan Africa and especially Cameroon, emphasis must be put on education of both patients and caregivers" ([41]).

3.3.5 UNITED REPUBLIC OF TANZANIA

In a literature review by Abbas & Archibald ([42]) covering 1960–2003, diabetic foot complications such as ulceration, infection or gangrene were associated with long-term disability and premature mortality. Rates of complications varied by African country: for foot ulcers, 4–19%; peripheral neuropathy, 4–84%; peripheral vascular disease, 2.9–78%; frequency of patients presenting with gangrenous foot ulcers, 0.6–69%; foot amputation rates, 0.3–45%. A study of diabetic patients in the United Republic of Tanzania showed mortality rates above 50% among patients with severe foot ulcers who did not undergo surgery ([42]).

3.3.6 MALAWI

In a prospective study conducted at Queen Elizabeth Central Hospital, Blantyre, by two surgeons Virich & Lavy, data from 200 consecutive patients with wounds were collected over a two-week period using a standard pro forma ([43]). Assaults were the principal cause of wounding (26.5%). Industrial injury and accidental self-injury also were common (17.5% and 12.5%, respectively). Lacerations were by far the most common type of wound encountered (67.5%). The most common anatomical site of injury was the hand (23.5%), while the trunk accounted for only 6% of injuries. Males were approximately four times more likely to present with a wound than females. The age of patients affected ranged from 2 to 76 years, with the commonest

group affected from 16 to 25 years: 35% of all wounds occurred in this group. In a study of people with leg ulcers attending the Central Hospital in Blantyre, Dutch authors found that venous and diabetic ulcers were rare but that infective causes and malignancy, perhaps resulting from HIV/AIDS, were common. They emphasized the value of biopsy and need for it, and therefore of histopathological services ([44]).

The above-mentioned countries addressed in this chapter are only a sample of available statistics. All of these countries have threats of increasing poverty and widening separation of economic opportunities as the middle class enlarges. The middle classes expect and receive better services. It is expected that every nation will have some capacity to adapt fundamental gold standards as the private sector contributes to the raising of standards of care. It is anticipated that these gold standards may trickle down to the impoverished majority who at present cannot afford them.

3.4 TROPICAL ULCERS

In western countries, most chronic wounds are due to venous insufficiency, arterial disease, diabetes, pressure or some combination of these factors. In tropical countries where few large series of leg ulcers have been reported, the prevalence and etiology of leg ulcers are largely unknown. A study based in one centre suggested that the chief causes of lower extremity wounds in the hospital were: leprosy (40%), diabetes (23%), venous disease (11%), and trauma (13%); 13% of the wounds were given no diagnostic etiology ([45]).

Buruli ulcer (BU) ([46]) has been reported from 30 countries in Africa, the Americas, Asia and the Western Pacific, mainly in tropical and subtropical regions. Approximately 24000 cases were recorded between 1978 and 2006 in Côte d'Ivoire. Nearly 7000 cases were recorded between 1989 and 2006 in Benin. More than 11000 cases have been recorded since 1993 in Ghana. Increasing numbers of cases of BU have been reported recently: 25 in 2004, 47 in 2005 and 72 in 2006 in Australia ([47]). Infection by *Mycobacterium ulcerans* begins as a dermal nodule. Lymphoedema may extend well beyond the nodule. Ulceration follows and is often extensive. Surgical management and physiotherapy to prevent scarring are demanding on hospital practice. Early BU is curable by appropriate antibiotic therapy and early nodule excision. When adipose tissue is perceived as part of the skin disorders, BU can be treated as skin failure ([48]).

There have been a few studies describing the epidemiology of the "tropical ulcer" ([49-50]). It seems apparent that a paucity of education and training in countries with poor health resources has resulted in a consensus view regarding the diagnosis as a mixed aerobic and anaerobic infection.

In reviewing the etiology, epidemiology, clinical findings and treatment of the common ulcers in the tropics, it is apparent that these lesions have much in common with complex wounds seen in western countries. Most of the ulcers have the characteristics of an infectious etiology: minor trauma, positive bacterial cultures, purulent exudates, pain, peri-wound oedema and tissue necrosis. Tropical phagedenic ulcer and Bazin's nodular vasculitis all have common findings. Each of these clinical findings can be addressed by the basic fundamentals of modern wound treatment: enhancing systemic factors, protecting the wound from trauma, debridement, control of infection, moist wound care, and control of oedema/lymphoedema ([51]). The Guidelines resulting from this document may include the concept of "Treatment of Skin Failure", which is sustainable, available at low cost at the local level, and appropriate for the majority of common conditions so classified.

Chronic wounds are a global epidemic. With wound care education intervention, it seems apparent that a new era is about to revolutionize global wound care. This will be a vitally important WHO initiative.

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COMPREHENSIVE WOUND ASSESSMENT AND TREATMENT SYSTEM (CWATS)

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The work-up of a patient with a non-healing wound can be complicated and, at times, all-consuming. In an effort to be thorough, many unnecessary laboratory and diagnostic tests are frequently ordered. The differential diagnosis of a patient with a leg ulcer includes disease states ranging from common conditions to esoteric syndromes. How can the wound care clinician navigate through this maze and arrive at a timely, accurate, cost-effective diagnosis and treatment plan? On average, 70% of all leg ulcers evaluated in an outpatient wound clinic are venous in origin ([1]). If the wound clinician adopts a systematic approach to all patients with non-healing wounds, regardless of etiology, the work-up becomes routine and fewer subtle signs, symptoms, and diagnoses will be overlooked. Medical error rates and hospital-based complications are becoming more common and the public is more aware of treatment inconsistencies ([2-3]). Standardizing a clinical approach to wound care can help limit a missed diagnosis or a delay in treatment.

It was thought that the creation of guidelines and protocols would eliminate practice variability and begin to standardize the practice of medicine. Failure of guideline implementation has severely thwarted those efforts ([4-5]). This chapter will outline a "work in progress" known as the Comprehensive Wound Assessment and Treatment System (CWATS). This system includes a concept known as the Least Common Denominator Model (LCD model), which attempts to take the patient work-up from the organism to the cellular level ([6]). Some of the concepts from the CWATS system and others from wound care clinicians were incorporated at a round table consensus meeting held in Hamburg, Germany, sponsored by Johnson and Johnson Wound Management Worldwide. That meeting generated a visual aide called the Core Healing Principles, a guideline for wound care clinicians to use when approaching the wound care patient ([7]).

4.1 COMPREHENSIVE WOUND ASSESSMENT AND TREATMENT SYSTEM (CWATS)

The physician who treats a recalcitrant wound must be skilled in communication, negotiation, and change management in order to coordinate an interdisciplinary team of specialists focused on a unified objective. The physician must possess an awareness and understanding of surgical concepts and treatment outcomes in plastic, orthopaedic, vascular, and general surgery in order to make appropriate, timely referrals during the continuum of care. Regardless of the physician's primary background, the wound clinician must incorporate knowledge and skills from both surgical and medical fields. The physician 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 paramount. An awareness of the social, psychological, spiritual or existential 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. Often a patient has already received conflicting therapeutic recommendations from other health-care professionals, leading to frustration easily misdirected towards the wound physician. Given the broad base of knowledge and skills required, coupled with a paucity of clinical experience that most physicians have in wound care, the practice of wound management is as challenging, demanding, and complex as that of any other field in medicine. Development of clinical training programmes at medical school and postgraduate levels is necessary to address such deficits ([8]).

4.2 HISTORY AND REVIEW OF SYSTEMS

Much information can be obtained simply by watching the patient walk into the clinic. Information concerning general appearance, body type, age, absence of a limb, ethnicity, and mobility can be evaluated before the formal examination begins. The patient may deny pain verbally, but the body position and facial expressions may indicate otherwise. The formal process begins with a comprehensive history and review of systems. Wound care is not unlike other specialties in that, for a majority of cases, a working diagnosis is achievable with a thorough history. 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 be venous in origin. The knowledge that the patient has RA will allow the clinician to consider a vasculitic wound in the differential diagnosis ([9]). A history of coronary disease in a patient with a venous ulcer should alert the clinician to the possibility of peripheral vascular disease and

a mixed etiology for the leg ulcer. This has implications for the work-up, expected time to healing, and the level of therapeutic compression to be used ([10]).

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, both 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 emphasize to the patient that all medications should be listed. It is well known that steroid use can negatively impact healing, and the commonly prescribed cox-2 inhibitors, ace inhibitors and calcium channel blockers have recently been implicated in slowing angiogenesis, a necessary component for normal healing ([11-12]). A family history will help identify risk factors and might shed light on the patient's diagnosis, as patients will often not recognize the correlation between a disease in their family history and their own current problem.

A complete past surgical history is important. Patients with prior venous surgery are known to have wounds that heal with more difficulty ([13]). This fact might influence the clinician to use advanced technologies earlier in the treatment regimen, in order to provide appropriate care beyond that considered standard of care for that wound type ([14]).

4.3 PATIENT EXPECTATIONS

Patients and their families desire outcomes from the treatment programme, which should be thoroughly understood and agreed upon from the onset of treatment. For example, an elderly female with a highly exudative venous ulcer may be most concerned with controlling the drainage. Although the patient might like the wound to heal, her primary concern for exudate management might explain why she becomes more non-compliant as the treatment programme progresses towards reaching "her" outcome. In addition, there is a growing population of patients who, through choice, terminal illness, or chronic conditions, have wounds that have reached a steady state and are not able to achieve healing. This group of patients might be better served with a palliative wound protocol that emphasizes the surrogate end-points of pain control, exudates and odour management, infection control, and quality of life outcomes, rather than total healing ([15]).

Finally, a history of present illness (wound history) should be recorded. This part of the history needs to occur after gathering all of the previously mentioned information. An interview beginning with the wound history requires an inordinate amount of time. The subsequent shorter, overall comprehensive history can frequently overlook potentially important details. The wound history should include the timing of ulcer occurrence, location, prior ulcer history, current and past treatments, size and appearance, complications, procedures performed, drainage, and pain.

4.4 PHYSICAL EXAMINATION

After the history, the clinician should begin a physical examination including 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 examination should be conducted using appropriate documentation as described in the American Medical Association's Coding for Procedural Terminology (CPT) manual for evaluation and management coding ([16]). This standard reference may not apply to other parts of the world, and a comparable guide should be used, based on individual local legal and regulatory policies. A complete examination frequently uncovers findings that aid in the final diagnosis and also ensures appropriate documentation and reimbursement for the work. After the complete history and physical examination, the clinician should concentrate on the wound itself. It is important to let the patient know, at the outset, that a complete examination will be conducted. 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 of the office consultation. Frequently they do not understand how important it is to get a complete medical picture before embarking on a wound-treatment programme. It is useful to send patients a welcome packet describing the entire process, either to their home prior to the visit or on arrival at the waiting area. Patients should fill in information concerning prior history and medications, which will expedite the visit and start them thinking about their wound on a more systemic level.

4.5 WOUND DOCUMENTATION

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 (i.e. presence of undermining, etc.) as well as standard measurements (length, width, depth).

4.6 LEAST COMMON DENOMINATOR (LCD)

The wound work-up next focuses on what has been termed the least common denominator (LCD) model ([6]). Composed of six subsections (tissue perfusion/oxygenation, infection, nutrition/immune status, psychosocial, pressure/neuropathy, and wound bed) this model ensures that the clinician consider all factors that affect healing in a wound of any etiology. Although initially covered in the history and physical examination, nutrition and psychosocial aspects are now revisited as they specifically relate to the wound and the potential for healing.

4.6.1 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 are not sufficient, however, to detect impaired arterial circulation, and additional testing is frequently required for the patient with leg ulcerations ([17]). Performing an ankle-arm index is useful for wound healing prediction and as an overall marker for cardiovascular health ([18]). Only about 10% of patients presenting to an outpatient wound clinic will have isolated arterial disease as an etiology for their leg ulcer ([19]). Arterial duplex scans, segmental pressures including toe pressures, pulse volume recordings, magnetic resonance angiography (MRA), and rapid sequence computed tomography (CT) scans are other non-invasive macro-circulation studies that may be ordered. An interventional angiogram is considered the gold standard at this time, although many facilities are using MRA as an alternative. 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.

If a patient has a leg ulcer and abnormal macro-vascular flow studies, but physiological studies (i.e. transcutaneous oximetry) indicate adequate values for healing, then a trial of aggressive local wound care is warranted. 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 wound patient in this manner will avoid unnecessary

high-risk procedures, and the limited treatment time will minimize potential harmful outcomes. If the micro-circulatory studies are abnormal, then a trial of treatments aimed at enhancing the micro-circulation (i.e. 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 ([20-21]).

4.6.2 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 (bio-burden) and the host's immune status. When equilibrium is reached there is no clinical infection. If the inoculum of bacteria is increased (>10^5 organism/gram tissue) or the host suffers a decrease in immunity, clinical infection occurs ([22]). Many examples are cited in the literature describing the failure of skin grafts, delayed closures, and overall wound healing problems when the bacterial bio-burden exceeds 10^5/gram tissue. This value is accepted by many as the quantitative definition of infection, except in the presence of beta-haemolytic streptococcus, where the value is somewhat lower (10^3/gram) ([23]). The bacteria compete for nutrients and oxygen with host repair cells in the granulating bed. Bacterial by-products of metabolism can be toxic to the host's normal cellular functioning. The presence of necrotic debris, foreign body, and the desiccation of the wound bed enhance bacterial growth. The colonization (the mere presence of organisms) and infection (the invasion of organisms into the tissue) are usually determined by the physical examination ([24]). The cardinal signs of inflammation (erythema, pain, swelling, and increased temperature) may be clues to impending infection. Many patients are clinically unable to mount an inflammatory response, and in those patients the use of quantitative culturing along with clinical intuition is necessary. There are numerous ways to obtain cultures of a wound but the quantitative biopsy remains the gold standard ([25]). Wound bio-burden and infection is a continuum, not a point in time when a specific number of organisms are present. This concept is important to emphasize, because the presence of granulation tissue depends on the presence of some bacteria ([26]).

Recently, the theoretical concept of "critical colonization" has been proposed as a point where colonization falls below classic quantitative values for infection but can negatively affect the patient's ability to heal by competing for nutrients and releasing toxic metabolic end-products ([27]).

4.6.3 NUTRITION AND IMMUNE STATUS

The third category within the LCD model focuses on the immunological state of the patient. Malnutrition is a major factor to consider. Although the literature fails to provide us with statistically significant relationships between healing and nutritional status, it is obvious that patients need to be nutritionally replete to maximize their chances of healing. Patients are not considered for surgical wound closure until they have achieved nutritional support and laboratory testing confirms success (i.e. pre-albumen level, serum transferrin level, total lymphocyte count, albumen level, etc.). Every attempt is 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.

4.6.4 PSYCHOSOCIAL

Psychosocial issues discussed earlier should be re-examined at this point. Chronic depression has been shown to affect healing and many wound patients suffer from psychiatric conditions if the clinician carefully probes during the history ([28]). Pain can exacerbate underlying psychiatric issues and can delay healing through the psycho-neuro-immunological connections ([29]). 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 will take priority over healing ([30]).

4.6.5 PRESSURE/NEUROPATHY

The LCD next addresses pressure on the wound and surrounding tissues. Pressure must be offloaded in order to maximize healing. 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.

4.6.6 WOUND BED AND PERI-WOUND TISSUE

The wound bed is the last aspect of the LCD model. The peri-wound tissue is assessed first. The skin can be painful to the touch, erythematous, macerated, dry and cracked or oedematous. A patient's continence status may play a pivotal role in wound healing and should be noted in the record. 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 more than 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 classic features on presentation. After a macroscopic view of the wound bed, the clinician should consider micro-environmental issues. The micro-circulation 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, resulting in delayed healing ([31-32]). The bio-burden 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 macro-vascular flow. Consider the patient with a venous ulcer, palpable pulses, but severe lipodermatosclerosis and relative dermal tissue hypoxia. The previously described wound would be difficult to heal and might require modifying the biochemical composition of the wound bed to accelerate healing ([33]).

The differential diagnosis of leg ulcers is long and covers diverse disease states. Systematically performing a history and physical examination, 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 to reach a diagnosis and a treatment plan prescription. The patient should be seen weekly from weeks 1 to 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. The patient should cycle through the diagnostic process again. This will ensure nothing is missed and minimize 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 were no statistically significant differences in healing rates amongst the various wound etiologies in either site of care ([34]).

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BEST CLINICAL PRACTICES FOR PREPARING THE WOUND BED: UPDATE 2006

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EDITORS' NOTE

The clinical practice for preparing the wound bed describes several important principles that can be adapted for all levels of health care. It should be noted, however, that under Recommendation 9 (the section on advances in modern dressings and devices), because of cost and distribution the materials may not be available in resource-poor settings. It is intended that description of these materials will stimulate innovative substitution where clinically advised. JMM.

5.1 ABSTRACT

This chapter updates the concept of preparing the wound bed by considering the whole patient (treatment of the cause and patient-centred concerns) before treating the hole in the patient. Local wound care consists of tissue debridement, control of persistent inflammation or infection and moisture balance before considering advanced therapies for wounds failing to heal at the expected rate. The best practice recommendations are based on scientific evidence and expert opinion, and for translation into practice should include patient preference.

This update of preparing the wound bed has the benefit of connecting the recommendations to the evidence as identified through the Best Practice Guidelines of the Registered Nurses Association of Ontario (RNAO). To date, three guidelines related to the treatment of wounds (pressure, venous and diabetic) have been issued and the components related to local wound care have been considered.

5.2 INTRODUCTION

Preparing the wound bed was first described in 2000 by Sibbald et al. and Falanga ([1-2]). This approach to wound management stresses that successful diagnosis and treatment of patients with chronic wounds requires holistic care and a team approach. The whole patient (the underlying cause and patient-centred concerns) must be considered before looking at the wound itself (see Figure [5.1]). Wound bed preparation is the promotion of wound closure through diagnosis and appropriate treatment of the cause, attention to patient-centred concerns, and correction of systemic and local factors delaying healing. Local factors can be represented by DIME (Debridement, Infection or Inflammation, Moisture balance and Edge effect). A template is presented as a basis for the discussion of the evidence base and expert opinion corresponding to each step in the paradigm of preparing the wound bed (see Table [5.1]). The Canadian Association of Wound Care (CAWC) Best Practice articles are not comprehensive but are meant to provide a practical, easy-to-follow guide or bedside enabler for patient care. The recommendations are based on the best available evidence to support the wound care clinician and team in planning and delivering the best clinical practice. . The Quick Reference Guide summarises the recommendations in an easy to use format which can be used as pocket reminder (see Table 5.0). Interpretation of the Levels of Evidence is shown in Table 5.1. For more detailed information, to the reader should consult the RNAO Best Practice Guidelines or the designated references.

We have identified several guidelines that are important for local wound care, including:

1 • Registered Nurses Association of Ontario (RNAO) guideline – *Assessment and management of foot ulcers for people with diabetes* (2005) ([3]);

2 • Registered Nurses Association of Ontario (RNAO) guideline – *Assessment and management of venous leg ulcers* (2004) ([4]);

3 • *Anti–infective guidelines for community–acquired infections* (2005).

TABLE 5.0 QUICK REFERENCE GUIDE: PREPARING THE WOUND BED

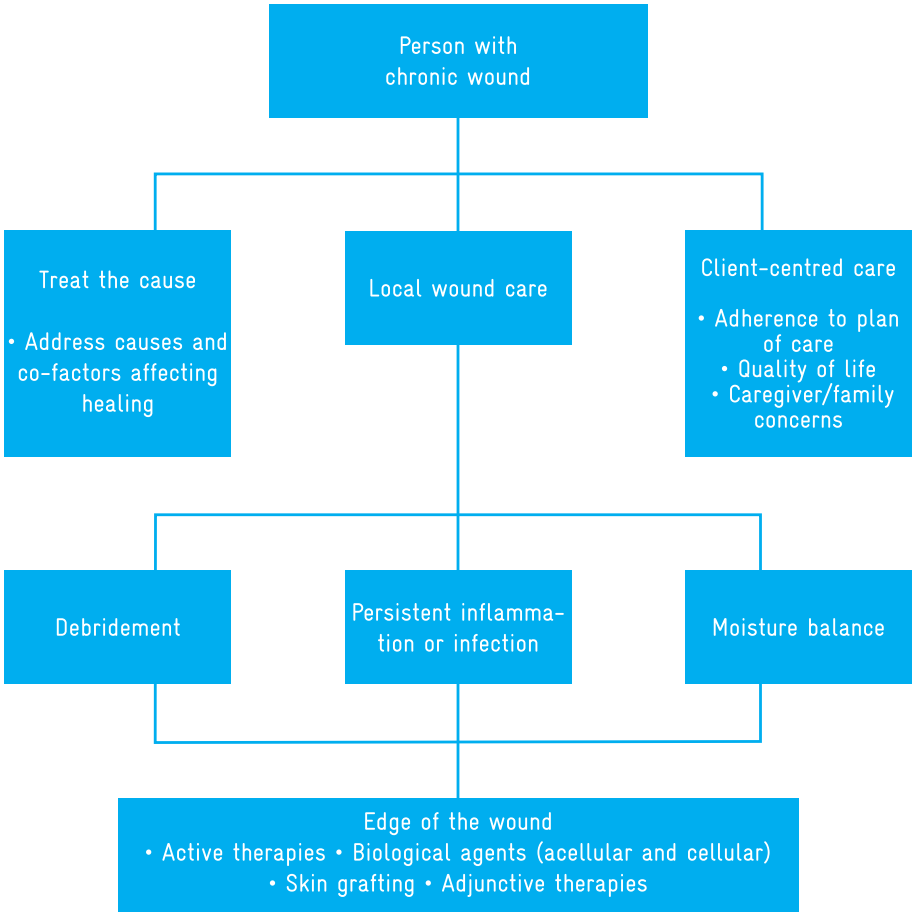
	RECOMMENDATIONS	LEVEL OF EVIDENCE
	IDENTIFY AND TREAT THE CAUSE	
1.	Assess the patient's ability to heal. Blood supply must be adequate and other important host factors corrected to support healing.	IV
2	Diagnose and correct or modify the treatable causes of tissue damage.	IV
	ADDRESS PATIENT-CENTRED CONCERNS	
3	Assess and support the management of patient-centred concerns to enable healing (pain and quality of life).	IV
4	Provide patient education and support to increase adherence to treatment plan.	IV
	LOCAL WOUND CARE	
5	Assess and monitor the wound's history and its physical characteristics (location + measure*).	IV
6	Debride healable wounds by removing non-viable, contaminated or infected tissue (surgical, autolytic, enzymatic, mechanical and larval). Non-healable wounds should have only non-viable tissue removed; active debridement to bleeding tissue is contraindicated.	Ib
7	Cleanse wounds with low-toxicity solutions (such as normal saline or water). Topical antiseptic solutions should be reserved for wounds that are non-healable or for those in which the local bacterial burden is of greater concern than the stimulation of healing.	III III
8	Assess and treat the wound for increased bacterial burden or infection (distinguish from persistent inflammation of non-bacterial origin).	IIa
9	Select a dressing that is appropriate to the needs of the wound, the patient and the caregiver, or clinical setting.	IV
10	Evaluate the expected rate of wound healing. If suboptimal, reassess recommendations 1 to 9.	III–IV
11	Use active wound therapies (biological agents, skin grafts, adjunctive therapies) when other factors have been corrected and if healing does not progress.	Ia–IV
	PROVIDE ORGANIZATIONAL SUPPORT	
12	For improved outcomes, education and evidence base must be tied to interprofessional teams with the cooperation of health-care systems.	IV

*Measure stands for Measure, Exudate, Appearance, Suffering, Undermining, Re-evaluate and Edge

TABLE 5.1 LEVELS OF EVIDENCE EMPLOYED BY RNAO GUIDELINE DEVELOPMENT PANELS (2005)

- Ia Evidence obtained from meta-analysis or systematic review of randomized controlled trials
- Ib Evidence obtained from at least one randomized controlled trial
- IIa Evidence obtained from at least one well-designed controlled study without randomization
- IIb Evidence obtained from at least one other type of well-designed quasi-experimental study, without randomization
- III Evidence obtained from well-designed non-experimental descriptive studies, such as comparative studies, correlation studies and case studies
- IV Evidence obtained from expert committee reports or opinions and/or clinical experiences of respected authorities

FIGURE 5.1 PREPARING THE WOUND BED PARADIGM



Source: adapted from: Sibbald RG et al. (5); reprinted with permission.

5.3 TREAT THE CAUSE

5.3.1 RECOMMENDATION 1

Assess the patient's ability to heal. Adequate blood supply must be present, as well as the correction of other important host factors to support healing. (Level of evidence = IV.)

Discussion

There are several important factors that determine the patient's ability to heal. The patient must be assessed to determine if the blood supply is adequate to support healing. If a regional pulse can be palpated, the local arterial flow will usually

support healing. If the dorsalis Pedis pulse is present, the pressure is approximately 80 mmHg or more, the radial pressure can be palpated at 70 mmHg and the carotid at 60 mmHg. If a pulse cannot be felt, special studies include the Doppler to assess ankle brachial pressure index (ABPI), toe pressures and, in specialized centres especially hyperbaric facilities, transcutaneous oxygen saturation equipment is often available (see Table 5.2). For healing to occur, blood flow studies often require a benchmark of: ABPI over 0.5 with a biphasic or triphasic pattern; toe pressure of 50 mmHg; transcutaneous oxygen pressure over 30 mmHg. Below these levels, healing can occur occasionally if all other contributing factors are optimized.

Clinicians must remember that in the presence of calcified arteries the ABPI may be falsely elevated, and any value over 1.2 is probably due to calcified vessels unless proven otherwise. The ability to heal and the criteria to apply compression are different. An ABPI will give information on arterial blood supply, but the diagnosis of venous disease must be based on clinical parameters and special duplex Doppler evaluation of the venous system.

TABLE 5.2 VASCULAR ASSESSMENT CRITERIA FOR HEALING

ABPI	TOE PRESSURE	TOE BRACHIAL INDEX	ANKLE DOPPLER WAVEFORM	$TCPO_2$ [A]	DIAGNOSIS
> 0.8	> 55 mmHg	> 0.6	Normal	> 40 mmHg	No significant arterial disease
> 0.6	> 40 mmHg	> 0.4	Biphasic/ monophasic	30–40 mmHg	Arterial disease, compression can be used with caution
> 0.4	> 20 mmHg	> 0.2	Biphasic/ Monophasic	20–30 mmHg	Arterial disease
< 0.4	< 20 mmHg	< 0.2	Monophasic	< 20 mmHg	High risk for critical limb ischaemia

Source: modified from Brown & Sibbald (6). A= $TcpO_2$ = transcutaneous partial pressure of oxygen.

Once adequate arterial flow is established, other criteria that may influence the ability of chronic ulcers to heal must be examined:

- A careful drug history and a history of known allergies should be obtained.
- Immunosuppressive agents and systemic steroids can impair healing.
- Uncontrolled oedema can impair healing. The area around the chronic wound should be examined for oedema and, if present, this should be corrected.
- Nutritional status can be screened. Serum albumin levels below 30 g/L delay healing and those below 20 g/L often represent non-healable wounds.

- Anaemia with haemoglobin levels below 100 g/L delay healing and levels below 70–80 g/L represent non-healing wounds or wounds that are very hard to heal.
- Cases with chronic diseases that impair immunity may also be a challenge for the wound care clinician; these include rheumatoid arthritis, collagen vascular diseases (lupus, scleroderma and dermatomyositis), people with organ transplants, and individuals receiving cancer chemotherapy or therapeutic radiation.

Remember the mnemonic: DOAAD = *Drugs, Oedema, Albumin, Anaemia, Diseases*.

5.3.2 RECOMMENDATION 2

Diagnose and correct or modify treatable cause of tissue damage. (Level of evidence = IV.)

Discussion

It is important to treat the cause of an ulcer as outlined in other articles of the Best Practice series published by Wound Care Canada in 2006 (www.cawc.net/open/wcc/4-1/index.html).

Pressure ulcers require pressure redistribution and other co-factors, such as friction, shear, mobility, nutrition. and control of external moisture including faeces.

Venous ulcers require oedema control with the cornerstone being compression therapy and activity modifications to activate calf muscle pump.

People with diabetic foot ulcers require pressure downloading and appropriate control of diabetes and it complications, including infection.

There are personal and health-care system factors that may prevent adequate correction of the cause. When it is not possible to provide best practice, clinicians may consider treating the wound to prevent complications and to improve quality of life, rather than having healing as the primary outcome. Enoch & Price ([7]) suggest considering alternative end-points to healing. This type of wound can be referred to as a maintenance wound and healing. If the goal is not wound healing, it is important to use resources to support alternative end-points, such as quality-of-life issues (care support) and prevention of complications (specialty surfaces) rather than wound-healing resources (dressings). RNAO Guidelines ([3-4]) outline the importance of using not only good practice recommendations but also recommendations relating to educational and operational needs.

5.4 PATIENT-CENTRED CONCERNS

5.4.1 RECOMMENDATION 3

Assess and support the management of patient-centred concerns to enable healing (pain and quality of life). (Level of evidence = IV.)

Discussion

Unresolved pain can negatively affect wound healing, which in turn has a negative effect on quality of life ([8]). Pain can cause activation of the sympathetic branch of the autonomic nervous system, leading to tissue hypoxia, and can also stimulate the hypothalamic-pituitary-adrenal axis causing a release of cortisol. Both of these affect wound healing.

Experienced clinicians need to take an initial full pain history to provide information about the patient's pain experience, with ongoing pain assessment occurring at each patient visit. There are two types of pain: nociceptive (an appropriate physiological response to painful stimuli; acute or chronic); and neuropathic (an inappropriate response caused by a primary lesion or dysfunction in the nervous system). The World Union of Wound Healing Societies (WUWHS) Consensus Panel on Pain identified categories related to the cause of pain (see Table [5.3]), which in turn support the development of management strategies for pain control. Psychological factors such as age, gender, culture, anxiety and depression, as well as environmental factors such as the timing of the procedure, resources and the setting, can all affect the patient's pain experience. Describing pain and monitoring the impact of management strategies for pain control begins by listening to how the patient describes the pain. Pain intensity can be measured using tools such as a visual faces scale or numerical rating scale. Pain frequency and intensity can be monitored using a pain diary.

TABLE 5.3 CAUSES AND MANAGEMENT OF PAIN

CAUSES OF PAIN	CHARACTERISTICS	MANAGEMENT STRATEGIES
Background pain	Pain at rest (related to wound etiology, infection, ischaemia)	Treat the underlying etiology of the wound and associated pathologies Analgesic and non-analgesic options as per WHO analgesic ladder
Incident pain	Pain during day-to-day activities (coughing, friction, dressing slippage)	
Procedural pain	Pain from routine procedures (dressing removal, application)	Preparation and planning of the procedure are key to preventing pain Analgesics as per WHO analgesic ladder should be administered before a procedure and may be required post-procedure Dressing selection is key to management of pain with dressing removal and application

WHO originally developed the pain ladder to simplify the management of cancer pain, and it is now used in more generalized applications ([9]) (see Figure [5.8]). The ladder provides a treatment algorithm that recommends a step-wise approach to alleviating persistent pain. Each progressive step on the ladder represents medications with higher potency for increased severity of pain. The WHO ladder, however, does not take into account neuropathic pain. These patients need to be referred to a specialist who is able to diagnose and treat neuropathic pain ([8]). Neuropathic pain is often identified with non-stimulus-dependent, burning, stinging, shooting and stabbing pain. It can be treated with tricyclic antidepressants, especially agents that have high anti-noradrenalin activity such as nortiptyline or desipramine. Gabapentin will also treat neuropathic pain, and these agents can be started at low dose, with gradual increase in dosage balancing therapeutic effect and side-effects. Chronic wound pain often benefits from combining treatment for nociceptive and neuropathic pain.

5.4.2 RECOMMENDATION 4

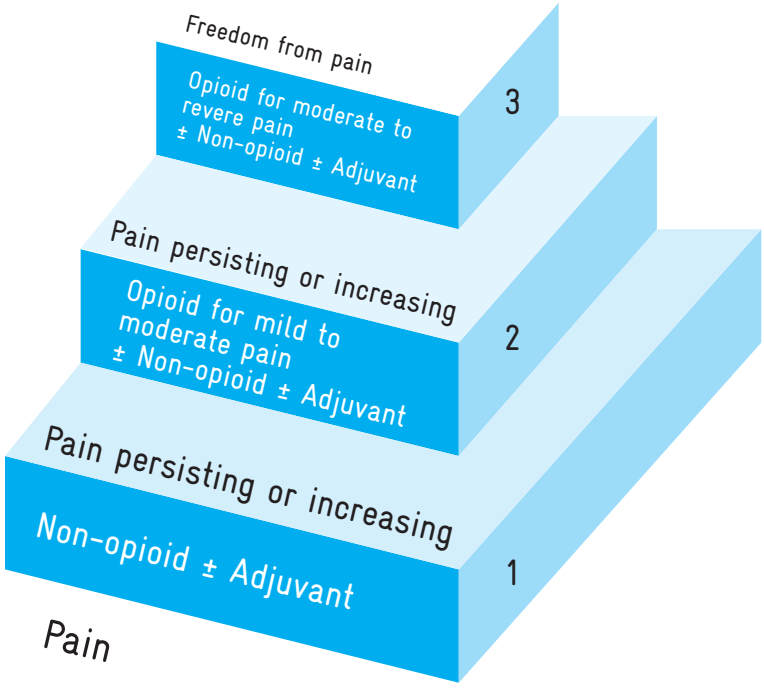
Provide education and support for patient-centred care to increase adherence with a treatment plan. (Level of evidence = IV.)

Discussion

In the original wound bed preparation article ([1]), the focus was on patient compliance with health-care provider recommendations, briefly touching on the term adherence. Adherence has become the cornerstone of patient-centred care, providing an open dialogue for patients and clinicians to discuss the rationale for care and its impact on the patient's life. The word "adherence" is preferred by many health-care providers, because "compliance" suggests that the patient is passively following the health-care provider's orders and that the treatment plan is not based on a therapeutic relationship established between the patient and the provider. Osterberg & Blaschke (2005) stated that "Poor adherence to medication regimens is common, contributing to substantial worsening of disease, death, and increased health-care costs" ([10]). They recommend that practitioners look for indications of poor adherence during patient visits, such as asking the patient how easy it has been to follow the treatment plan, assessing for clinical response to treatment, pill counts / rates of refill and physiological markers. Supporting adherence to treatment regimens can occur in several ways, but appears most effective when several strategies are used in combination.

- Emphasize the value of the patient's regimen and the positive effects of adherence.
- Make the regimen simple and give simple, clear instructions.
- Listen to patients and customize the regimen to their lifestyle.
- Enlist support from family, friends and community services when needed.

FIGURE 5.2 THE WHO ANALGESIC LADDER



Health-care interventions that incorporate a non-judgemental attitude as well as a collaborative approach to care augment patient adherence. Innovative methods of managing chronic diseases have had some success in improving adherence when a regimen has been difficult to follow. New technologies such as reminders through cell phones and personal digital assistants, as well as pillboxes with paging systems, may be needed to help patients who have the most difficulty meeting the goals of a regimen.

5.5 LOCAL WOUND CARE

The wound bed preparation paradigm in Figure 5.1 focused on a holistic approach to the person with a wound. Table 5.4 focuses on the components of local wound care and emphasizes the expected outcomes from clinical actions.

TABLE 5.4 PREPARING THE WOUND BED – CLINICAL AND PHYSIOLOGICAL MECHANISMS OF ACTION

CLINICAL OBSERVATIONS	MOLECULAR AND CELLULAR PROBLEMS	CLINICAL ACTIONS	EFFECT OF CLINICAL ACTIONS	CLINICAL OUTCOME
Debridement	Denatured matrix and cell debris impair healing	Debridement(episodic or continuous) Autolytic, sharp surgical, enzymatic, mechanical or biological	Intact, functional extra-cellular matrix proteins present in wound base	Viable wound base
Infection, inflammation	High bacteria concentration causes: ↑ inflammatory cytokines ↑ proteases ↓growth factor activity ↓ healing environment	Topical/systemic antimicrobials anti-inflammatories protease inhibitors growth factors	Low bacterial concentration causes: ↓ inflammatory cytokines ↓ proteases ↑growth factor activity ↑ healing environment	Bacterial balance and reduced inflammation
Moisture imbalance	Desiccation slows epithelial cell migration Excessive fluid causes maceration of wound base/margin	Apply moisture balancing dressings	Desiccation avoided Excessive fluid controlled	Moisture balance
Edgeofwound – non-advancing or undermined	Non-migrating keratinocytes Non-responsive wound cells, abnormalities in extracellular matrix or abnormal protease activity	Re-assess cause, refer or consider corrective advanced therapies bioengineered skin skin grafts vascular surgery	Responsive fibroblasts and keratinocytes present in wound	Advancing edge of wound

Source: International Wound Bed Advisory Board (copyright, used with permission).

5.5.1 RECOMMENDATION 5

Assess and monitor the wound history and physical characteristics (location + MEASURE). (Level of evidence = IV.)

Discussion

Consistent and reliable wound assessment remains a clinical challenge for wound-care clinicians. Wound assessment must include a global assessment of the patient and the environmental factors that may affect wound healing, as well as local assessment of the wound itself (see Figure [5.3]). The MEASURE ([11]) mnemonic presented in Table 5.5 is a simple conceptual framework that may act as a basis for a consistent approach to local wound assessment. The most common parameters evaluated include size, wound edges, wound bed appearance, presence or absence of undermining, exudate and pain. When assessed at an appropriate frequency, these parameters give the clinician important decision-making information, as well as

creating a comprehensive wound history. Clinicians are reminded that local wound assessment must occur in the context of a global assessment of the patient and of the environment.

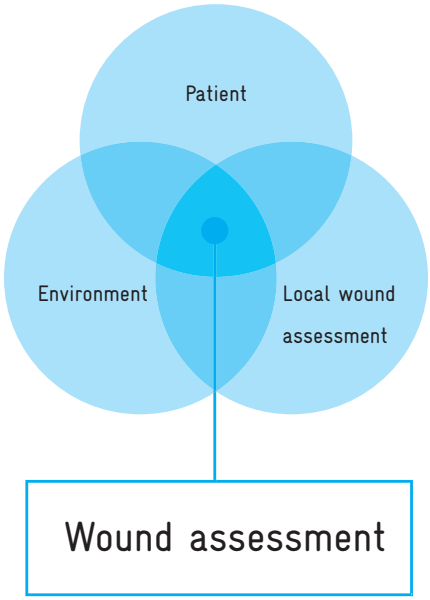
TABLE 5.5 MEASURE – A POCKET GUIDE FOR CLINICIANS

MEASUREMENT PARAMETER	CLINICAL OBSERVATION	INDICATOR
Measure	Length, width, depth, area	Reduction or increase in wound surface area and/or depth
Exudate	Amount, quality	• Decreased or increased amount • Decreased or increased purulence
Appearance	Wound bed appearance, tissue type and amount	• Increased or decreased % granulation-tissue • Increased or decreased % necrotic tissue • Friability of granulation tissue
Suffering	Patient pain level using validated pain scale	Improved or worsening wound-related pain
Undermining	Presence or absence	Decreased or increased amount
Re-evaluate	Monitor all parameters on a regular basis every 1–4 weeks	Parameters sequentially documented in patient record
Edge	Condition of wound edge and surrounding skin	• Presence or absence of attached edge with advancing border of epithelium • Presence or absence of erythema and/or induration • Presence or absence of maceration

Change in wound surface area is emerging as the most reliable predictor of outcomes in wound healing. The challenge is to measure wound surface areas in a valid and reliable manner. Consistently done, simple ruler methods may be adequate for most clinical practice settings, but for greater reliability acetate tracings or digitizing systems should be considered.

Wound assessments need to be consistently done and documented in the client/patient record. Multiple wound assessment tools have been developed to assist the clinician. The tool selected for use should be both valid and reliable and should detect change over time. In 1999, Woodbury et al. ([12]) critically appraised the tools existing at that time. The PSST (Pressure Sore Status Tool, now replaced by the Bates–Jensen Wound Assessment Tool) and Sessing tools showed the best evidence for their use with pressure ulcers. Since that time, further work on validation of the Pressure Ulcer Scale for Healing (PUSH) tool ([13]) has been completed and the tool can be recommended for use. The Photographic Wound Assessment Tool (PWAT) ([14]) is useful for all types of ulcers and can be scored reliably from 35 mm photographs. Most recently, the Leg Ulcer Measurement Tool (LUMT) ([15]) has been validated for use with leg ulcers. The tool used must be appropriate for the setting and the users.

FIGURE 5.3 THE CONTEXT OF WOUND ASSESSMENT



5.5.2 RECOMMENDATION 6

Debride healable wounds, removing non-viable, contaminated or infected tissue (surgical, autolytic, enzymatic, mechanical and larval). Non-healable wounds should have only non-viable tissue removed and active debridement to bleeding tissue is contraindicated. (Level of evidence = Ib.)

Discussion

The recommendation and discussion of appropriate debridement of chronic wounds from the 2000 Wound Bed Preparation article ([1]) remain remarkably valid. Review of the Medline, CINAHL and Cochrane databases yielded very little new literature on the debridement of chronic wounds. A Cochrane review of debridement in diabetic foot ulcers ([16]) found evidence to support hydrogels over standard gauze, but concluded that there was insufficient evidence for surgical or larval therapy. The Steed retrospective analysis ([17]), not considered in the Cochrane review, does however provide good evidence (Level Ib) for surgical debridement of neuropathic ulcers with adequate circulation to heal. Table 5.6 has been adapted from the one included in the original article to include larval (biological) debridement therapy. This table assists the clinician in choosing the appropriate method of debridement based on key clinical factors. Many clinicians are reluctant to perform debridement, especially in primary health-care settings, because of the perceived risks ([18]). Before clinicians embark on debridement of chronic wounds they must ensure that they

have the necessary skills to perform the task, the skill is within their scope of practice and there is agency or institutional policy in place to support them. The discussions of autolytic, mechanical and surgical debridement in the original article remain current.

TABLE 5.6 KEY FACTORS IN DECIDING METHOD OF DEBRIDEMENTA

	SURGICAL	ENZYMATIC	AUTOLYTIC	BIOLOGICAL	MECHANICAL
Speed	1	3	5	2	4
Tissue selectivity	3	1	4	2	5
Painful wound	5	2	1	3	4
Exudate	1	4	3	5	2
Infection	1	4	5	2	3
Cost	5	2	1	3	4

a A score of 1 is most desirable and 5 is least desirable.

Source: adapted from Sibbald et al. (1).

Enzymatic debridement uses proteolytic agents to breakdown necrotic tissue. Various commercial preparations are available in different countries; these contain agents such as collagenase, papain/urea, DNAse/fibrinolysin and trypsin. In general, these agents are safe and specific to necrotic tissue, but may cause local irritation due to pH changes. They may provide for faster removal of necrotic tissue than autolysis. Except for collagenase, there is very little literature on their efficacy. One study showed collagenase to be more cost effective than hydrocolloids in the treatment of Stage IV pressure ulcers ([19]). In another study, collagenase was shown to be more effective than other enzymatic debriding agents and mechanical debridement in the form of wet to dry dressings ([20]). In some countries, non-commercial preparations may be used ([21]). Only collagenase has been approved for use in Canada.

Larval debridement therapy or biological debridement is gaining in popularity in many clinical settings. In this therapy, sterile larvae of the greenbottle fly *Lucilia sericata* are used to remove non-viable tissue from the wound bed. Proteinases secreted by the larvae selectively digest non-viable tissue ([22]). Several recent studies have appeared in the literature supporting the use of larval debridement therapy ([23-24]). Concern remains regarding infection if non-sterile larvae are used ([25]). This method has yet to find general acceptance in Canada largely because of patient and clinician disgust, but when presented in an appropriate manner may find more acceptance ([26]).

5.5.3 RECOMMENDATION 7

Cleanse wounds with normal saline or water. The use of topical antiseptics should be reserved for wounds that are non-healable or those in which the local bacterial burden is of greater concern than the stimulation of healing. (Level of evidence = III.)

Discussion

In vitro studies have identified the toxicity of many of the topical antiseptic agents, as outlined in the 2000 article ([1]). To prevent tissue damage in wounds with the ability to heal, saline and water are recommended as cleansing agents. If a wound is non-healable and bacterial burden is more important than tissue toxicity, antiseptics may be used to dry the wound surface and decrease local bacterial proliferation. This strategy may also be important if deep infection or osteomyelitis is present. Once the deep infection has been controlled, toxic solutions should not be instituted. Moist interactive dressings will promote healing and optimal wound bed preparation. Cleansing agents and their effects are shown in Table 5.7.

TABLE 5.7 CLEANSING SOLUTIONS

AGENT	EFFECTS
Sodium hypochlorite solution	High pH causes irritation to skin. Dakin's solution and Eusol (buffered preparation) can select out Gram-negative microorganisms
Hydrogen peroxide	De-sloughing agent while effervescing; can harm healthy granulation tissue and may form air emboli if packed in deep sinuses
Mercuric chloride, crystal violet, Proflavine	Bacteriostatic agents active against Gram-positive species only; may be mutagens and can have systemic toxicity
Cetrimide (quaternary ammonium)	Good detergent, active against Gram-positive and -negative organisms, but high toxicity to tissue
Chlorhexidine	Active against Gram-positive and -negative organisms, with small effect on tissue
Acetic acid (0.5–5%)	Low pH, effective against Pseudomonas species, may select out Staphylococcus aureus
Povidone iodine	Broad spectrum of activity, although decreased in the presence of pus or exudate Toxic with prolonged use or over large areas

5.5.4 RECOMMENDATION 8

Assess and treat the wound for increased bacterial burden or infection. (Distinguish from persistent inflammation of non-bacterial origin.) (Level of evidence = IIa.)

Discussion

The diagnosis of infection is based on clinical criteria with bacterial swabs or deep cultures, and laboratory and radiological tests used as adjuncts for diagnosis and

treatment. All wounds contain bacteria at levels ranging from contamination through colonization, clinical colonization (also known as increased bacterial burden, occult or covert infection) to infection. Increased bacterial burden may be confined to the superficial wound bed or may be present in the deep compartment and surrounding tissue of the wound margin. Therefore, it becomes important to diagnose both the bacterial imbalance and the level of invasion in order to diagnose and treat infection properly (see Table [5.8]). Increased bacterial burden in pressure ulcers has been demonstrated to delay healing in patients with chronic ulceration ([27-28]).

TABLE 5.8 CLINICAL SIGNS AND SYMPTOMS OF WOUND INFECTION

SUPERFICIAL INCREASED BACTERIAL BURDEN (CRITICALLY COLONIZED)	DEEP WOUND INFECTION	SYSTEMIC INFECTION
Non-healing Bright red granulation tissue Friable and exuberant granulation New areas of breakdown or necrosis on the wound surface (slough) Increased exudate that may be translucent or clear before becoming purulent Foul odour	Pain Swelling, induration Erythema Increased temperature Wound breakdown Increased size or satellite areas Undermining Probing to bone	Fever Rigours Chills Hypotension Multiple organ failure

Source: adapted from Sibbald et al. (35).

Contamination is the presence of bacteria in the wound surface. Colonization is the presence of replicating bacteria attached to the wound tissue without causing injury to the host. Critical colonization occurs when bacteria delay or stop healing of the wound without classic symptoms and signs of infection. Infection is the presence of replicating microorganisms in a wound associated with host injury. The borders between these concepts are not clearly established. The clinician must assess the patient's symptoms and signs present in the wound to distinguish contamination, colonization and healing from critically colonized or infected wounds that are not healing or even endangering the life of the patient.

The classic signs of infection are: pain, erythema, oedema, purulent discharge and increased warmth. In chronic wounds, other signs should be added: delayed healing or new areas of breakdown, increased discharge (often initially serous or clear and watery before becoming pustular), bright red discoloration of granulation tissue, friable and exuberant granulation, new areas of slough on the wound surface, undermining and a foul odour ([29]). Serous exudate may be increased in a chronic wound with increasing bacterial burden before purulence is noted with the clinical signs usually recognized in infections. It has been suggested that chronic wounds should show some evidence of healing within four weeks to progress to healing by

Week 12. If this time limit is exceeded, then increased bacterial burden or infection should be suspected as one of the causes of delayed healing ([30]).

Discoloration of granulation tissue arises from loose, poorly formed granulation tissue, while friable granulation tissue that bleeds easily results from excessive angiogenesis stimulated by bacterial pathogens. Healthy granulation tissue is pink-red and moist, translucent in appearance. When infected, it will appear dull and may have patches of greenish or yellow discoloration. Certain anaerobic bacteria such as *Bacteroides fragilis* and streptococci produce a dullish, dark red hue, while *Pseudomonas* species produce green or blue patches that may fluoresce at 365 nm (Wood's) light. Undermining results from atrophic granulation tissue inhibited or digested by bacteria. Foul odour is usually produced by Gram-negative bacilli, especially *Pseudomonas* species, or anaerobes digesting granulation tissue ([31]).

Deep infection will often cause erythema and warmth extending 2 cm or more beyond the wound margin when the surrounding skin becomes involved. The bacterially stimulated increased inflammatory response is painful and causes the wound to increase in size or lead to satellite areas of tissue breakdown resulting in adjacent tissue ulceration. Deep infections, especially in ulcers of long duration, can often lead to underlying osteomyelitis. Probing to bone is a simple clinical test that may indicate osteomyelitis, especially in patients with neuropathic foot ulcers often associated with diabetes ([32]).

Gardner et al. examined the reliability and validity of clinical signs of infection in two recent papers ([33-34]). These studies identified various symptoms and signs of infection and compared the diagnoses made using these signs with results of quantitative cultures from tissue biopsies to correlate each sign or symptom with the stated criteria of infection. Increasing pain, friable granulation tissue, foul odour and wound breakdown all demonstrate the validity for the diagnosis of infection based on discriminatory power and positive predictive value. The symptoms that rated most highly were:

— increasing pain (1.0);
— oedema (0.93);
— wound breakdown (0.89);
— delayed healing (0.87);
— friable granulation (0.8);
— purulent exudate (0.78);
— serous exudate (0.74).

Often clinicians use a number of signs or symptoms on which to base a diagnosis of infection. Non-healing is often the first criterion. When managing bacterial

colonization in infection, we can examine the US Department of Health and Human Services Clinical Guideline No. 50, 1994. The modified recommendations state the following:

- Do not use swab cultures to diagnose infection.
- Consider a two-week trial of topical antimicrobials/antimicrobial dressings if the wound is not healing in spite of optimal care (increased bacterial burden, covert infection, critical colonization suspected).
- Perform bacterial cultures and evaluate for osteomyelitis if the wound fails to improve.
- Use systemic antibiotics if there is overt infection.

If topical antimicrobials are used, it is important to use non-sensitizing antibiotics with low tissue toxicity in those agents not used systemically so that resistant organisms will not breed on the surface of a wound (see Table [5.9]). Common sensitizers frequently misused in patients with chronic wounds, particularly leg ulcers, include neomycin, bacitracin agents containing lanolin, and perfumes ([36]).

TABLE 5.9 TOPICAL ANTIMICROBIALS USEFUL IN WOUNDS WITH OVERT AND COVERT INFECTION

Agent	Staphylococcus aureus	MRSA[a]	Streptococcus	Pseudomonas	Anaerobes	Comments	Summary
Cadexomeriodine	+	+	+	+	+	Also debride; low potential for resistance; caution with thyroid disease	Low risk and effective
Silver	+	+	+	+	+	Do not use with saline; low potential for resistance	
Silver sulfadiazine	+	+	+	+	+	Caution: sulfonamide sensitivity	
Polymyxin B sulfate/ Bacitracin zinc	+	+	+	+	+	Bacitracin in the ointment is an allergen; the cream formulation contains the less-sensitizing gramicidin	Use selectively
Mupirocin		+				Reserve for MRSA and other resistant Gram+ spp.	
Metronidazole					+	Reserve for anaerobes and odour control; low or no resistance of anaerobes despite systemic use	
Benzoyl peroxide	Weak	Weak	Weak		Weak	Large wounds; can cause irritation and allergy	
Gentamicin	+		+	+		Reserve for oral/intravenous use – topical use may encourage resistance	Use with caution
Fusidin ointment	+		+			Contains lanolin (except in the cream)	
Polymyxin B sulfate/ Bacitracin zinc Neomycin	+	+	+	+	+	Neomycin component causes allergies, and possibly cross-sensitizes to aminoglycosides	

a methicillin-resistant Staphylococcus aureus.

For systemic antibiotics, it is often wise to base these choices on culture once a diagnosis is made. In chronic wounds of less than a month's duration, the causative pathogen is often Gram-positive organisms, while in wounds lasting longer than a month or in patients who are immune-compromised, coverage for Gram-positive, Gram-negative, and anaerobic species is needed.

TABLE 5.10 TREATMENT OF WOUND INFECTION IN DIABETIC FOOT ULCER MANAGEMENT

NON-LIMB-THREATENING INFECTION	LIMB-THREATENING INFECTION	
Superficial infection	Deep wound infection	Systemic infection
• Support host defences • Requires a team approach • Cleanse and debride wound • May be monomicrobial • Topical antimicrobials • May require oral/IV antibiotics (based on host risk) • Offloading • Ongoing evaluation based on clinical findings • Patient education	• As in superficial infection Polymicrobial • Will require oral or IV antibiotics • May require surgical debridement • Non-weight bearing • Consider hospitalization • Consider Infectious Disease consultation • Ongoing evaluation based on clinical findings	• As in deep wound infection • Will require hospitalization • Will require IV antibiotics • Ongoing evaluation based on clinical findings • Bed rest

Source: RNAO Guideline Development Panel (3).

5.5.5 RECOMMENDATION 9 (SEE EDITOR'S INTRODUCTORY NOTE)

Select appropriate dressings for local moisture balance to stimulate granulation tissue and re-epithelialization. **(Level of evidence = IV.)**

Discussion

Clinicians should base the choice of dressing on patient history and assessment, cause of the wound, and evaluation of the wound bed and peri-wound skin. Each wound must be treated individually, as there is no "recipe" for a particular wound type. The selected dressing should provide moisture appropriate for the wound environment, not cause pain, prevent infection, and not cause damage to the wound or peri-wound area. The clinician needs to consider what the function of the dressing is in order to maximize the wound bed preparation. The form chosen needs to conform to the area of application, facilitate moisture balance, and prevent infection. Ongoing assessment of the dressing choice needs to be made along with the regular wound assessment. The clinician should become familiar with the different categories of dressings and their construction (see Table [5.11]). They should understand the mode of action of the dressing within the wound, and the indications and contraindications to use. The selection of the dressing should balance the goal of care with the cost to the payer in order to attain optimal, cost-effective care.

For systemic antibiotics, it is often wise to base these choices on culture once a diagnosis is made. In chronic wounds of less than a month's duration, the causative pathogen is often Gram-positive organisms, while in wounds lasting longer than a month or in patients who are immune-compromised, coverage for Gram-positive, Gram-negative, and anaerobic species is needed." Table 5.10 describes an approach to the management of infections in diabetic foot ulcers.

TABLE 5.II MODERN CLASSES OF DRESSING

GENERIC CATEGORIES		
	CLASS	DESCRIPTION
1	FILMS/MEMBRANES	Semi-permeable adhesive sheet Impermeable to H_2O molecules and bacteria
2	NON-ADHERENT	Sheets of low adherence to tissue Non-medicated tulles
3	HYDROGELS	Polymers with high H_2O content Available in gels, solid sheets or impregnated gauze
4	HYDROCOLLOIDS	May contain gelatin, sodium carboxymethylcellulose, polysaccharides and/or pectin Sheet dressings are occlusive with polyurethane film outer layer
5	CALCIUM ALGINATES	Sheets or fibrous ropes of calcium sodium alginate (seaweed derivative) Have haemostatic capabilities
6	COMPOSITE DRESSINGS	Multilayered, combination dressings to increase absorbency and autolysis
7	FOAMS	Non-adhesive or adhesive polyurethane foam May have occlusive backing Sheets or cavity packing Some have fluid lock
8	CHARCOAL	Contains odour-adsorbent charcoal within product
9	HYPERTONIC	Sheet, ribbon or gel impregnated with sodium concentrate
10	HYDROPHILIC FIBRES	Sheet or packing strip of sodium carboxymethylcellulose Converts to a solid gel when activated by moisture (fluid lock)
11	ANTIMICROBIALS	Silver or cadexomer iodine with vehicle for delivery: sheets, gels, alginates, foams or paste
12	DEVICES	Negative pressure wound therapy (NPWT) applies localized negative pressure to the surface and margins of the wound Dressings consist of polyurethane or polyvinyl alcohol materials
13	BIOLOGICALS	Living human fibroblasts provided in sheets at ambient or frozen temperatures Extracellular matrix Collagen-containing preparations Hyaluronic acid Platelet-derived growth factor

a Use with caution if critical colonization is suspected.

	LOCAL WOUND CARE			CARE CONSIDERATIONS
	TISSUE DEBRIDE-MENT	INFECTION	MOISTURE BALANCE	INDICATIONS/CONTRAINDICATIONS
	+	-	-	Moisture vapour transmission rate varies from film to film Should not be used on draining or infected wounds* Create occlusive barrier against infection
	-	-	-	Allow drainage to seep through pores to secondary dressing Facilitate application of topicals
	++	-	+	Should not be used on draining wounds Solid sheets should not be used on infected wounds
	+++	- / +	++	Should be used with care on fragile skin Should not be used on heavily draining or infected wounds* Create occlusive barrier to protect the wound from outside contamination Characteristic odour may accompany dressing change and should not be confused with infection
	++	+	+++	Should not be used on dry wounds Low tensile strength – avoid packing into narrow deep sinuses Bioreabsorbable
	+	-	+++	Use on wounds where dressing may stay in place for several days[a]
	-	-	+++	Use on moderate to heavily draining wounds Occlusive foams should not be used on heavily draining or infected wounds[a]
	-	-	+	Some charcoal products are inactivated by moisture Ensure that dressing edges are sealed
	+	+	++	Gauze ribbon should not be used on dry wounds May be painful on sensitive tissue Gel may be used on dry wounds
	+	-	+++	Best for moderate amount of exudate Should not be used on dry wounds Low tensile strength – avoid packing into narrow deep sinuses
	+	+++	+	Broad spectrum against bacteria Not to be used on clients with known hypersensitivities to any product components
	-	+	+++	This pressure distributing wound dressing actively removes fluid from the wound and promotes wound edge approximation Advanced skill required for patient selection for this therapy
	-	-	-	Should not be used on wounds with infection, sinus tracts, excessive exudate, or on clients known to have hypersensitivity to any of the product components Cultural issues related to source Advanced skill required for patient selection for this therapy

5.5.6 RECOMMENDATION 10

Evaluate expected rate of wound healing to determine if treatment is optimal. If sub-optimal healing is noted, re-assess the cause and patient-centred concerns. (Level of evidence = III–IV.)

Discussion

Flanagan ([37]) stated that a 20–40% reduction of wound area in two and four weeks is likely to be a reliable predictive indicator of healing. A clinical study demonstrated that a 50% reduction in ulcer area at 12 weeks of treatment is a good predictor of healing ([38]). If the edge is not migrating, and the wound is not getting smaller, a full reassessment of cause and corrective therapies needs to be made. If patient and wound are optimized and the edge is still not migrating, then a wound may need advanced therapies to kick-start the healing process. Maintenance wounds, which are unlikely to heal, need to have alternative end-points, such as wound stabilization, reduced pain, reduced bacterial load or decreased frequency of dressing changes.

5.5.7 RECOMMENDATION 11

Ruling out of other causes, such as unrecognized malignancy, is needed if healing does not progress.

Falanga ([39]) designed a classification system (see Table [5.12]) to monitor the outcomes of bioengineered skin that is helpful in assessing the movement of the wound edge as a parameter for monitoring healing outcomes.

TABLE 5.12 CLINICAL CLASSIFICATION OF THE EARLY EFFECT OF THE WOUND EDGE

CLASS	EFFECT	EDGE DESCRIPTION
A	Full	Thin but widespread epidermal coverage; the edges have been activated
B	Edge effect only	Stimulation of the wound's edges; translucent epidermal outgrowth visible
C	Wound bed stimulation only	Stimulation of granulation tissue; wound bed is even with surrounding skin
D	No benefit	There is no stimulation of the wound edges or bed

Source: Falanga (39).

Clinicians need to remember that the edge of the wound is only one outcome parameter, and wound closure is not always the expected outcome. Active wound therapies (biological agents, skin grafts, adjunctive therapies) should be used when other factors have been corrected and if healing still does not progress. (Level of evidence = Ia–IV.)

Discussion

Adjunctive therapies should be considered as options for wound management when healing is recalcitrant. Adjunctive therapies such as Negative Pressure Wound Therapy (NPWT), also referred to as Topical Negative Pressure therapy (TNP), biologically active dressings, living skin tissue (grafts) or living skin equivalents, electrical stimulation, hyperbaric oxygen and therapeutic ultrasound may offer alternatives to stimulating healing when malignancy is ruled out. Some of these therapies are discussed in more detail under the appropriate ulcer etiology in other papers in the Best Practice series published by Wound Care Canada (www.cawc.net/open/wcc/4-1/index.html). The level of evidence for each therapy is dependent on the etiology of the ulcer.

The Canadian Consensus Group VAC Therapy (CCGVT) Report (2003) ([40]), and the Medical Advisory Secretariat (MAS) Ontario Ministry of Health and Long-term Care for the Ontario Health Technology Advisory Committee Report (2004) ([41]) have reviewed the use of NPWT in the Canadian context. Both reports were unable to find significant evidence to support the use of NPWT but did conclude that there were clear clinical situations where the use of NPWT might be beneficial. These included such benefits as earlier hospital discharge, fewer dressing changes, savings in nursing costs, and improved quality of life. The Canadian consensus panel also suggested appropriate criteria for implementing NPWT. These included appropriate assessment of the patient, the absence of fistulas and malignancy, the ability of the patient to adhere to the plan of care, and at least four weeks of prior first-line treatment without a reasonable decrease in wound size (<30%).

A 2004 Cochrane review by Kranke et al. ([42]) gave qualified support to the use of hyperbaric oxygen treatment (HBOT) for diabetic foot ulcers. HBOT significantly reduced the risk of major amputation, and may improve the chance of healing at one year. The authors commented on the high cost of the therapy and its limited availability. The review could find no evidence to support the use of HBOT in other etiologies.

Cochrane reviews of the use of both electromagnetic therapy ([43]) and low-level laser ([44]) in the treatment of venous leg ulcers could find no evidence to support these modalities. This is consistent with the findings regarding pressure ulcers discussed in the pressure ulcer paper in the Best Practice series published by Wound Care Canada (www.cawc.net/open/wcc/4-1/index.html).

The discussion of the use of living skin equivalents and of platelet-derived growth factor from the original 2000 article remains valid.

5.5.8 RECOMMENDATION 12

For improved outcomes, education and the evidence base must be tied to interdisciplinary teams with the cooperation of health-care systems. (Level of evidence = IV.)

Discussion

Wound healing can be a complex process once all the factors and co-factors that may affect healing are identified. Best practice care for people with chronic ulcers demands a systematic team approach from knowledgeable and skilled health-care professionals. These team members will vary based on the needs of the patients. The interdisciplinary team needs to work closely with patients and their families to address the complex lifestyle, self-care and multiple treatment demands of patients who have chronic wounds. Clinicians can facilitate and positively influence wound-healing outcomes by promoting, collaborating and participating in interdisciplinary care teams who follow best practice guidelines similar to those presented in this document and the other documents in the Best Practice series published by Wound Care Canada (www.cawc.net/open/wcc/4-1/index.html). Armstrong ([45]) demonstrated that a team approach to diabetic foot care resulted in significant savings to the health-care system. Implementation of best practice team-focused care in a study of 16 000 patients resulted in 66% fewer hospital admissions, a 74% decrease in hospital days and a 53% decrease in nursing home admissions.

The development and implementation of a successful wound management programme not only involves collaboration with good practice leaders but, as the RNAO guidelines demonstrate, there is also a need for collaboration with educators and administrators. Their support is required to ensure coordinated care with community and health-care agencies and the specialized knowledgeable interdisciplinary team of health-care professionals striving for improved wound-care outcomes. All the RNAO wound-care-related clinical practice guidelines contain multiple recommendations related to the value of inter-professional teams and the need for organizational support.

5.6 CONCLUSION

The concept of the wound bed preparation algorithm as a systematic clinical decision-making framework, first published in the 2000 article "Preparing the wound bed" ([1]), has stood the test of time. The key components of wound assessment and management, identifying and treating the cause of the wound, addressing patient-centred concerns, establishing goals for wound healing, optimizing local wound care, and collaborating with inter-professional team members remain valid today. To effect change and improve outcomes, clinicians need to move beyond the local to the global, learning to interact with, and effect change within, health-care systems.

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LYMPHOEDEMA AND THE CHRONIC WOUND: THE ROLE OF COMPRESSION AND OTHER INTERVENTIONS

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6.1 INTRODUCTION

The term lymphoedema entered common usage in the early 20th century, when systems of manual lymphatic drainage (MLD) and comprehensive decongestive physiotherapy (CDP) were developed to move excess fluid in the tissues. Before that time, for centuries, the term elephantiasis was used to describe gross swelling that was at least only in part due to lymphatic failure with excess fluid collection. Lymphoedema is a chronic condition characterized by an abnormal collection of fluid (lymph). However, as Hebra ([1]) and Unna ([2]) emphasized, elephantiasis is a gross hypertrophy of many components of the tissue. There is fibrosis and disintegration of elastic fibres. It seemed to Unna that the pathogenesis could be attributed to damage to the lymphatic system by bacterial infections and by venous congestion. Today we would describe this as a failing lymphatic system due to gross overload. This is often the result of bacterial infection and venous hypertension. In many cases, it is secondary to a lymphatic system that is anatomically either incompletely formed or disrupted by cancer and its surgical treatment. Other etiologies include radiotherapy, filariasis, and paralytic pathology. We now recognize a sequence of lymphatic failure causing a loss of barrier function of the epidermis that in turn allows entry of agents that are harmful to the lymphatic system. This allows invasion of irritants and bacteria through entry points combined with impaired immune surveillance. Both contribute to further lymphatic damage. The lymphatic system is part of the immune-surveillance system and thus plays a vital role in the wound healing process.

It is recognized that wound healing, with its phases of haemostasis, inflammation and remodelling, utilizes all three vascular systems at every phase. Different phenotypes of wound healing responses are determined by genetic interaction with the environment. Likewise, repair of damage to the arterial, venous and lymphatic systems interacts with the phases of wound healing. This may produce disorders such as lipodermatosclerosis and keloids. In elephantiasis, there may be a variable phenotypic response to the failure of the lymphatic system to clear lipid and proteins inclusive of growth factors and cytokines. The end-point may be severe hyperkeratosis, severe fibrotic nodularity or hypertrophy of adipose tissue. Most commonly, there is a mixture of tissue responses, which can be observed in lymphoedema. It is well recognized that damage to the lymphatic system impairs the healing of both venous and non-venous chronic wounds ([3]).

Estimates state that one person in 30 worldwide is afflicted with lymphoedema. This figure does not include the millions suffering from chronic venous disease nor the patients with chronic wounds and peri-wound lymphoedema ([4]). Until the past decade, lymphoedema received little clinical attention and has been termed "the hidden epidemic". Most recently, studies of a London population by Moffatt et al. reported a lymphoedema prevalence rate of 1.33/1000, rising to 5.4/1000 in the over 65-year age group ([5-6]). Williams & Mortimer (2007), reviewing the literature, found that groin dissection or gynaecological surgery for cancer causes lymphoedema in 40% of cases ([7]). Male cancers of the groin and pelvic region provide lower figures of around 20%. Mixed venous and lymphatic disease, as emphasized more than a century ago, continues to be reported ([1-4]). In India, it is estimated that filariasis may account for 23 million cases of lymphoedema ([8-9]). In China, the one-time epidemic of filariasis has been eliminated. However, lymphoedema secondary to malignancy has resulted in 60 000 new cases per annum in Shanghai alone ([10]).

Understanding the pathophysiology, diagnoses and management of lymphoedema is essential to the proper care of the wound care patient. This chapter is divided into two segments. The first will discuss the epidemiology, anatomy, pathophysiology, and therapy of lymphoedema. The second segment will discuss the unique relationship between the chronic wound and peri-wound lymphoedema.

6.2 ANATOMY OF THE LYMPHATIC SYSTEM

The lymphatic system consists of lymph vessels and regional and central lymph nodes. Initial lymph vessel is the collective name given to the valved lymphatic capillaries and the single and double valve pre-collectors. Lymphatic capillaries consist

of flat overlapping endothelial cells surrounded by a fibrous network. Filaments fix the lymphatic vessels to the surrounding connective tissue ([11-12]). They prevent the narrowing of the initial vessel system and enable increased fluid influx into the lumen in the presence of oedema. The initial lymphatics provide liquid reabsorption while the lymph collectors are transport vessels. Lymph collectors consist of three layers: intima, media and adventitia. The lymph collectors have valves consisting of fibrous stroma lined with endothelial cells. This lymphatic vascular unit is called a lymphangion. The rhythmic pumping action of the lymphangion smooth muscle is likened to cardiac muscle. These vessels lead to lymphatic ducts from the main parts of the transporting vessels and to the thoracic duct. The lymphatic ducts do not have resorbing function. The blood supply to the lymphatic vessel wall is provided by vasa vasorum originating in the adventitia.

Sympathetic nerve fibres innervate the adventitia. The extremities contain a superficial (above the fascia) and a deep (below the fascia) lymphatic system. The transport of lymph occurs not only from distal to proximal, but also from the superficial to the deep system and vice versa. The collectors generally follow the course of the blood vessels and lead to the regional lymph nodes. However, throughout the body, variations in the vessel course and the directional flow are of clinical importance. As an example, the lymphatic drainage in the hand flows from the palm to the dorsum, thus explaining the dorsal hand oedema in palmar infection.

Most of the fluid filtering from the arterial capillaries perfuses the interstitium and returns to the venous circulation via the venous section of the blood capillaries. An estimated 20% of this fluid returns to the blood vascular system via the lymphatic system ([13-14]). Almost all of the lymph from the lower extremities and lower body flows into the thoracic duct. From there, it empties into the left internal jugular and subclavian veins. Lymph from the left side of the head, left arm, and areas of the chest also empty into the thoracic duct prior to rejoining the venous circulation. Lymph from the right arm, right side of the neck and head, and parts of the thorax enter the right thoracic duct, which then joins the right subclavian and internal jugular vein. Thus, the upper great venous system receives this large volume of lymph. However, the actual entry point, though mostly as described above, shows significant variation in the point of entry. The large contribution, therefore, of lymph to cardiac output and to the filling of the thoracic great veins needs to be understood. The factors controlling emptying of the veins, such as breathing and posture, are well understood, but their implications for the lymphatic system have received less attention ([15]).

The body, both the deep organs and skin, is divided into a series of lymphatic drainage regions by "lymphatic watersheds". Watersheds are found primarily in the dermis and divide areas free of lymph collecting vessels that contain only initial lymph capillaries. On either side of these dividing lines, the flow of lymph drains in different and generally opposite directions.

These lymphatic drainage areas are called "lymphotomes". Variations in lymphatic course are frequent. Knowledge of the regional anatomy of the lymphatic system enables the therapist to redirect lymphatic flow into functioning collateral circulation after lymph node removal or destruction. For example, in drainage of the lower extremity, lymph collectors that course along the sciatic nerve, ending in the internal iliac lymph nodes, may circumvent the inguinal lymph nodes. Likewise, the cephalic lymph vessels that originate in the region of the wrist anastomose with nearby lymphatic pathways and end directly in supraclavicular lymph nodes. This allows use of the functioning collateral lymphatic circulation after the removal of the axillary lymph nodes during the management of breast cancer.

6.3 PHYSIOLOGY OF THE LYMPHATIC SYSTEM

The function of the lymphatic system is the transport of interstitial fluid and its components (lymph) from the interstitium back to the venous circulation. The lymph components consist of proteins, fat, cells, organic and inorganic cell products, viruses, growth factors, cytokines and bacteria. Water is a small part of the lymph composition. Water functions as a transport media for the lymph. The venous capillaries cannot reabsorb substances such as proteins with high molecular weight. The lymphatic capillary endothelial wall structure facilitates the absorption of these substances. Resorption of excess water occurs through the venous drainage system. The filtration and resorption of lymph are determined by capillary pressure, tissue pressure including the influence of tissue movement, and colloidal osmotic pressure in capillaries, and tissue fluid according to Starling's equilibrium. Lymph passing from the pre-lymphatic channels may have some low resistance and preferential pathways to the lymphatic capillaries, such as along elastin fibres ([16]). The continued mechanism of lymph uptake and flow is thought to be the result of several factors; the pumping action of lymphangions stimulated by vessel fluid dilatation, arterial pulsation, muscle contraction, body movements that involve stretching and relaxing the tissues, and the effect of respiratory pressure changes. External pressure from massage and compression bandages also assists in lymph transport.

6.3.1 LYMPHOEDEMA VERSUS OEDEMA

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.

Lymphoedema is the result of a low output failure of the lymphatic system. Simply stated, lymphatic transport is reduced. This derangement arises from either congenital lymphatic dysplasia (primary lymphoedema) or anatomical obliteration (secondary lymphoedema), such as operative dissection, repeated lymphangitis with lymphangiosclerosis or functional deficiency (e.g. lymphangiospasm and valvular insufficiency). The result is that lymphatic transport falls below the capacity needed to handle the presented micro-vascular filtrate. Oedema, on the other hand, is a high output failure of the blood vascular and lymph circulation (see Table [6.1]). This takes place when a normal or increased transport capacity of the intact lymphatics is overwhelmed by an excessive flow of filtrate. Common examples of oedema include congestive heart failure, chronic venous insufficiency, hepatic cirrhoses (ascites), nephritic syndrome and the oedema secondary to the inflammatory wound healing response.

In some disease states, swelling may be a mixed form of oedema and lymphoedema. In these cases, where high output failure is chronic, a gradual deterioration of the lymphatic anatomy takes place, resulting in decreased transport capacity (e.g. recurring infection).

TABLE 6.1 OEDEMA CLASSIFICATION

Passive hyperaemia	Malnutrition
Chronic venous insufficiency	Malabsorption
Congestive heart failure	Renal disease
Pregnancy	Active hyperaemia
Inactivity	Inflammation
Hypoproteinaemia	Allergy

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. Every tissue is stressed by common, long-standing lower extremity lymphoedema. Although rare, malignant changes are a concern. This is partly aggravated by a deficiency in immunosurveillance seen in the lymphoedematous limb (see Table 6.2).

The lymphoedema associated with acute and chronic wounds is sometimes called "post-traumatic lymphoedema". Perhaps "post-traumatic lymph stasis" would be more appropriate. Acute trauma is usually followed by a transudative "low protein oedema", the result of a prolonged inflammatory phase of wound healing. If the lymphatic collecting anatomy is initially damaged (i.e. in an open wound), true lymphoedema rapidly develops. Unless the lymphoedema is pre-existing, collectors proximal and distal to the lesion are normal ([4]).

TABLE 6.2 CONSEQUENCES OF CHRONIC LYMPHOEDEMA

Protein-rich interstitial oedema	Lymphatic arthropathy
Fibrosis, sclerosis, fat cell generation	Arterial and venous damage
Disturbance of local metabolism	Neuropathy
Increased occurrence of cellulitis – bacterial and fungal	Impairment of wound healing, resulting in chronic wounds
Progressive lymphatic damage	Malignant degeneration ([17])

6.4 LYMPHOEDEMA CLASSIFICATION

Lymphoedema is differentiated into "primary lymphoedema" and "secondary lymphoedema" (see Table 6.3). Primary lymphoedema is a hereditary malfunction of the lymph system resulting in impaired lymph node or lymph vessel development and accounts for 10% of all lymphoedema patients. The best known is Milroy's Disease (Nonne–Milroy), which is lymphoedema present at birth (congenital lymphoedema), but with the advances in genetics many new types are being recorded ([18]). The symptoms of primary lymphoedema may not be apparent until the second or third

decade of life. This form of primary lymphoedema may present as lymphoedema praecox, appearing in adolescence, and lymphoedema tarda, which begins after 35 years of age. The distribution in cases of primary lymphoedema between the sexes is reported to be 87% in women and 13% in men ([19]). Primary lymphoedema occurs most often in the legs. Primary lymphoedema of the upper extremities and face is rare.

TABLE 6.3 LYMPHOEDEMA CLASSIFICATION

PRIMARY	SECONDARY
Praecox: adolescent age Birth Tardum: age 30–35 years +	Surgery Infection Trauma Chronic wound Podoconiosis Malignancy Radiation Venous disease Neurological paralysis Filariasis

Secondary lymphoedema can be caused by many factors. The most recognizable are associated with recurrent cellulitis, lymphadenectomy, radiation, venous disease, and numerous post-surgical complications. Lymphoedema secondary to vascular reconstruction, joint replacement, and venous harvesting in conjunction with coronary by-pass comprises an ever-growing problem. Peri-wound (localized) lymphoedema in acute and chronic wounds is now recognized as a major inhibitory factor in wound healing ([3]). The most common tropical cause of lymphoedema is filariasis, a disorder caused by infection with larvae transmitted to humans by mosquitoes and infecting more than 125 million people worldwide. In the western hemisphere, filariasis is now confined to areas of the Caribbean and South America, predominantly Haiti and Brazil. However, it is most common in India, tropical Asia and Africa.

Podoconiosis, also known as non-filarial lymphoedema, is best described in Ethiopia and is currently being thoroughly researched there ([20]). It is found in communities that have no footwear and work in soils believed to contain silica. It is possibly much more common than previously described and will account for patients who are currently assumed to have suffered from filariasis but have neither genital involvement nor blood antigen from filaria.

Genital swelling is one of the most disabling presentations in lymphatic filariasis. It is especially a feature of lymphoedema due to *Wuchereria bancrofti* filarial infection from mosquito. Scrotal swelling is used to identify the prevalence of the disease in population surveys prior to elimination programmes by the WHO-led Global Programme to Eliminate Lymphatic Filariasis (GPELF). The lining sac surrounding the testis fills with fluid as in hydrocele but in this case it is a lymphocele or filariacele. The soft tissues of the scrotum or vulva develop oedema and overgrowth of tissues as in lymphoedema elsewhere. The lymphatics drain into the lymph nodes of the inguinal region but some drainage from the genitalia may pass through abdominal lymphatic networks and thus chyle may leak into these tissues.

6.5 Diagnosis of lymphoedema

Accurate diagnosis of lymphoedema, in most patients, can be made with a detailed history, physical examination, and volume measurements. Co-morbidities such as venous insufficiency, metastatic disease, morbid obesity, and repeated infections may affect the clinical presentation. Related diseases such as diabetes mellitus, congestive heart failure, and peripheral vascular occlusive disease will also influence the therapeutic approach. When presented with unilateral extremity lymphoedema, venous occlusion and occult visceral tumours must be considered. It should seem obvious that informed physician input is required prior to the start of lymphoedema therapy. Best practice guidelines are being formulated and will require international consensus ([21]).

Examination of the extremities, trunk, and neck provides information as to the extent of the oedematous changes and the potential areas of drainage. Papular lymph cysts, deepened natural skin creases, and lympho-cutaneous fistulas may be seen. Surgical or traumatic scars may suggest an etiology. Palpation reveals thickened skin and fibrous changes in the subcutaneous tissue. The Stemmer sign is an important diagnostic finding. This sign, considered pathognomonic in lymphoedema, shows thickened cutaneous folds on the dorsum of toes or fingers that cannot be lifted by pinching the skin between the fingers ([22]).

6.6 LIPOEDEMA

This is a chronic disease, possibly of local lipid metabolism. This results in the symmetrical impairment of fatty tissue distribution and storage, combined with hyperplasia of individual fat cells. This abnormal distribution of fat is usually seen between the pelvic crest and the ankle, so that unless the lymphatic system has been damaged, the feet appear normal. The swelling often progresses during the day as the

diminished tissue resistance of fatty tissue permits the accumulation of orthostatic oedema. A positive family history has been reported by early studies at the Mayo Clinic in 20% of cases ([23]). Eventually, in many patients, mechanical insufficiency of the lymph system occurs, leading to true lymphoedema. Lipo-lymphoedema is, therefore, a combination of impaired fat distribution and an impaired lymphatic system. Lipoedema is seen almost exclusively in women. Men develop this pattern often in association with feminization, i.e. hepatic cirrhosis, hormonal therapy for prostatic carcinoma, or Kleinfelter's syndrome. However, as awareness of the clinical presentation of lipoedema increases, more cases of male lipoedema of unknown etiology are being reported. In most cases, lipoedema develops during puberty. Heavy hips and thighs are obvious signs. Simultaneous incidence in the upper extremities is rare, but, when it is involved, there is often a large fold of loose hanging skin visible when the patient elevates her arm to the horizontal position.

Medical history and physical examination form the diagnoses of lipoedema. Special diagnostic procedures or additional laboratory tests are rarely necessary to establish the diagnosis. The main differential consideration is lymphoedema. In contrast to lymphoedema, lipoedema is symmetrical, often painful to palpation, susceptible to easy bruising, and the patients rarely develop cellulitis. The Stemmer sign is negative ([22]). The patient may show varying degrees of obesity and list many unsuccessful attempts at dieting. Often, the patient will relate that dieting resulted in weight loss only in the upper part of the body while the tissues of the lower body remained soft and rubbery, as opposed to the hard and fibrotic tissue seen with chronic lymphoedema. The pathogenesis of lipoedema and lymphoedema could possibly be related. In inflammatory conditions, adipose tissue is a source of fatty acids, which are known to be increased in all forms of lymphoedema ([24]).

Therapy for the most part is palliative or directed at co-morbidities. Adipose tissue is not easy to compress and manual lymphatic drainage is of diminished effectiveness. At present, lipectomy and liposuction, though promoted in some centres that demonstrate improvement, would seem to be contraindicated for fear of damage to normal lymphatic drainage ([25]). Nevertheless, leading practitioners of liposuction, experienced in the management of lymphoedema and arguing in favour of this technique, are supported by larger series and longer follow-up periods ([26]). Bariatric surgery has not been found to affect significantly the abnormal fat distribution and metabolism seen in lipoedema *(personal observation JM)*.

6.7 LYMPHOEDEMA DIAGNOSTIC STUDIES

In benign forms of lymphoedema, extensive laboratory testing is usually not necessary. Most laboratory testing evaluates co-morbidities. Thyroid hormone levels are examined when hypothyroidism is suspected. Blood chemistry may help to evaluate the degree of oedema versus lymphoedema. Imaging for diagnoses can be helpful if further definition is required. Many of the higher technologies have limited access and the best are to be found only in tertiary care centres. Non-invasive duplex-Doppler studies and, rarely, phlebography may be required if venous disease is suspected. CT, magnetic resonance imaging (MRI), and ultrasonography are also useful in selected patients. In filariasis, ultrasound is helpful preoperatively in the treatment of hydrocele in order to locate filarial nests or other causations. Lymphoscintigraphy (LAS) is very useful in demonstrating the detailed lymphatic pathology. LAS provides images of lymphatics and lymph nodes, as well as semi-quantitative data on radiotracer (lymph) transport. However, the limited availability of experienced specialists in nuclear medicine and the ability of LAS to influence therapy would seem, at this time, to limit its usefulness to research application.

Genetic testing has been shown to define a limited number of hereditary syndromes. In the future, such testing may become routine and could hold promise for specific gene therapy. Biopsy of enlarged regional lymph nodes in the presence of chronic lymphoedema is rarely helpful and should be discouraged. Fine needle aspiration is a useful alternative if malignancy is suspected. Any invasive procedure on a lymphoedematous limb has the potential to aggravate the swelling or lead to cellulitis.

6.8 LYMPHOEDEMA STAGING

Clinical staging has proven useful for the classification of lymphoedema ([27-28]).

Stage I: spontaneous, reversible tissue swelling leaving indentations, negative or borderline Stemmer sign, no palpable fibrous tissue.

Stage II: spontaneous, irreversible tissue swelling with moderate or pronounced fibrosis. Indentations are difficult to produce. Stemmer sign positive, lymphostatic dermatosis.

Stage III: lymphatic elephantiasis, usually with pronounced skin alterations and gross shape changes due to overgrowth of epidermal, dermal and subcutaneous

tissue. Severity based on differences in limb volume are assessed as minimal (<20% increase), moderate (20% increase), or severe (>40% increase).

Clinical staging has not reached complete consensus, since some would argue for a stage that identifies known impairment of lymphatic function, as after surgery or radiotherapy, identifiable by LAS but not yet causing swelling. Others would like to subclassify the grosser changes of elephantiasis and complications such as may be detected in the genitalia. The legal profession assessing impairment calls for such subclassifications.

6.9 NON-OPERATIVE (CONSERVATIVE) LYMPHOEDEMA THERAPY

Therapy for peripheral lymphoedema is divided into non-operative and operative methods. This discussion does not apply directly to wound-related lymphoedema. The principles of modern lymphoedema treatment, however, have direct application to the lymphoedema / wound healing equation.

If one takes an earlier view that elephantiasis is a response to infection and venous overload, one may, as a priority, concentrate on restoring the barrier function of the skin. The venous element is addressed by encouraging elevation and mobility. This can be seen in Indian systems of medicine that encourage, through yoga, improved posture, breathing and movement ([29]). Simultaneously, this system stresses the need for support and compression in order to reduce the tension and expansion of the tissues caused by excess lymph. An intensive regimen to clear central overload within the thorax and abdomen of both the venous and lymphatic system is also required ([15]).

6.9.1 COMPREHENSIVE DECONGESTIVE PHYSIOTHERAPY

Comprehensive decongestive physiotherapy (CDP), a combination of physical therapy modes, is the gold standard for the treatment of primary and secondary lymphoedema ([30–32]). In the 1930s, Emil Vodder, a Danish physician, used a type of therapy known as manual lymphatic drainage (MLD) to treat lymphoedema. This massage is a light, circular, superficial tissue stretching performed with varying degrees of pressure. The effect is to increase the transport through the lymph collectors and the development of new routes for lymph drainage. Decongesting lymphotomes "upstream" from the lymphoedema areas and utilizing the watershed anatomy for directional flow enhance treatment efficiency. Michael & Ethel Foeldi

in Germany introduced, applied and refined CDP as it is practised today ([30-31]). A further modification of this method by the Casley-Smiths in Australia is now widely used ([32]). A modified form using biomedicine integrated with Ayurvedic medicine (yoga and herbals) is currently promoted in Kerala, southern India ([29]).

CDP involves four therapy modes: MLD, compression bandaging, decongestive exercises, and patient education in hygiene and self-treatment. CDP is usually divided into a two-phase treatment programme. Phase one consists of daily therapy sessions of specialized manual lymph drainage/massage, a range of motion exercises, and compression wrapping applied with multilayered short stretch bandages. The use of short stretch bandages enables the contraction of muscles to apply pressure to a resistant force provided by stiff bandages, thus mobilizing lymph with intermittent pulsations. The patient wears the bandages during the interval between therapy sessions. These sessions vary from one to two times per day and may continue from two to four weeks or longer, depending upon the severity of the disease. Phase two, initiated immediately at the completion of phase one, is programmed to conserve and optimize the benefits achieved from the start of therapy.

Phase two is a lifetime commitment by the patient. Fitted, low stretch elastic compression garments are used daily. Exercises designed to improve systemic lymphatic flow and patient-administered MLD enable the patient to maintain the achieved lymphoedema reduction. Compliant patients are very often able to improve upon the initial reduction ([33]). The success of CDP depends upon the compliance of the patient and the availability of therapists trained and certified in this exacting technique.

The Foeldi Klinik in Germany has treated 2500 patients annually with CDP. Limb volume reductions averaged 50% after the completion of therapy. More than 50% of patients maintained their reduction during phase two ([28]). Casley-Smith reported volume reductions of over 60% in 618 lymphoedematous limbs ([34]). The pioneering work of Boris et al. introduced CDP to North America in the 1980s ([33-35]). They studied 119 consecutive patients, and the affected limbs included both arms and legs. Lymphoedema reduction averaged 62.6% in 56 patients with one affected arm and 68.6% in 38 patients with one affected leg. After 36 months of follow-up, the average volume reduction in the arms increased to 63.8% and remained at 62.7% in the affected legs.

6.9.2 PNEUMATIC COMPRESSION

Intermittent pneumatic compression by a sequential gradient pump, if prescribed, must be used with caution and under strict supervision. Inappropriately high pressures and variable time intervals must be closely monitored. The use of pneumatic pumps without the added resource of MLD and the failure to first empty the abdomen and thorax of excess lymph can result in further lymphatic damage as well as significant pelvic, genital and opposite limb swelling ([36]). MLD is used to decrease fluid volume proximal to the obstructing lesion, thereby improving the efficiency of lymph mobilization toward the thoracic duct and venous circulation. The role of thermal therapy and pulsed radio frequency energy in the treatment of lymphoedema remains unclear.

6.9.3 ELEVATION

Simple elevation is an obvious first step in the treatment of the lymphoedematous limb. Its main effect is probably to reduce venous overload ([15]). This is especially helpful in the early stage of lymphoedema. The effect of swelling reduced by this means should be maintained by wearing a low stretch, elastic sleeve/stocking accompanied by ankle flexion and extension exercise.

6.9.4 DRUG THERAPY

Diuretic agents may sometimes be useful during the early phases of CDP, especially in the elderly who may have some impairment of cardiac output. However, long-term administration of diuretics in the treatment of lymphoedema is of little benefit. Diuretics may induce fluid and electrolyte imbalance by decreasing the water content of the lymph fluid and increasing the viscosity, thereby hindering the mobilization of lymph ([27]).

Oral benzopyrones (coumarin) are thought to hydrolyse tissue proteins and facilitate absorption while stimulating lymphatic collectors ([34]). They are effective only at long term and high dosage. They have not been approved for use in the USA and many other countries as they have been linked to liver toxicity. Long-term high-dosage regimens are still in question.

Antibiotics should be administered for superimposed infection (cellulitis-lymphangitis). Skin erythema without systemic signs of infection does not

necessarily imply infection, and needs no antibiotics. It may be due to spread of lymph containing an excess of inflammatory cytokines, which in a fully functioning system would be cleared as fast as they are produced. Appropriate prophylactic antibiotics are often indicated in severe chronic lymphoedema with repeated episodes of cellulitis. Studies in Brazil and India demonstrate how local hygiene can be effective in quickly reducing inflammatory episodes ([37-38]). Antimycotic drugs can treat fungal infections.

In the treatment of filariasis, drugs are used to remove microfilariae from the bloodstream. Diethylcarbamazine, ivermectin, and albendazole are recommended. They are contraindicated in children under the age of five years and during pregnancy. Eradication of the adult nematodes by these drugs is variable and may be associated with significant side-effects. These drugs have no direct effect on the limb swelling. Usually, such swelling is a late event. Whole populations identified as having filarial infection are treated once a year for five years.

6.9.5 GARMENT AND BANDAGE COMPRESSION

Garment and bandage compression are essential components in the continuous therapy for lymphoedema. When therapy has resulted in volume reduction, compression prevents recurrence. The effect of continued compression results in a reduction of abnormally increased ultrafiltration and improved fluid reabsorption. Joint and muscle pump function is improved and there is a reduction of fibrosis in the limb. The studies of Mayrovitz & Larson suggest that compression, within defined parameters, may actually increase arterial perfusion ([39]).

The bandage material used determines the compression effect. Short stretch bandages are the preferred bandage for the treatment of primary and secondary lymphoedema. Short stretch bandages cause a higher pressure during activity (working pressure) and relatively low pressure at rest (resting pressure). Padding using cotton and selective use of foam rubber can protect protruding bones and reduce fibrosis. Padding should also protect kinking and closure of lymphatics by compression of deep folds and crevasses. The selection of compression grade and proper size is critical to the success of garment control in lymphoedema. Likewise, strict attention to patient adherence and garment compatibility determines the long-term success of therapy. Bandages and compression garments can be very expensive. Inexpensive, durable bandages and garments need to be developed if this important therapy is to become available in resource-poor areas.

6.10 OPERATIVE TREATMENT OF LYMPHOEDEMA

Surgical approaches to alleviate extremity lymphoedema have not been widely accepted. In selected patients, surgical procedures are used in combination with CDP. Surgery alone is used when CDP has been unsuccessful. Operative treatment for lymphoedema falls within four areas: resection, drainage, reconstruction, and liposuction. A number of reported cases have been remarkably successful but significant data on the long-term results are lacking. By contrast, severe scarring is frequently seen especially in those so predisposed. With fibrosis dominating, keloid formation is not unusual.

Resection or "debulking" is the most direct approach. This involves complete removal of cutaneous and subcutaneous tissue including muscle fascia. Skin from the resected area is then grafted over the resulting defect. The efficacy of this procedure is hampered by the obliteration of skin lymphatic channels, ulceration, and scar tissue. New oedema peripheral to the resected and grafted regions is common. After successful CDP in selected cases, redundant folds may require resection in advanced elephantiasis.

Surgical drainage methods include enteromesenteric bridge procedures, skin flaps, omental transposition, and the implantation of thread or tubes. None of these procedures has demonstrated long-term benefit in more than a small minority of cases.

Autogenous lymph vessel transplantation or interposition vein segments to restore lymphatic flow have been attempted. Lympho-venous and lympho-nodal venous shunts have yet to confirm long-term patency. Liposuction recently has been modified to treat successfully non-fibrotic upper extremity lymphoedema ([25]). Short-term results seem encouraging but strict patient compliance is required with continued use of low-stretch compression garments and supportive CDP.

6.11 MANAGEMENT OF LYMPHOEDEMA OF THE GENITALIA

As with all other sites, lymphoedema responds to skin hygiene and other general principles of lymphoedema management. In the early stages, swelling may vary from day to day and resolve partially when prone at night. However, lymph drainage proximally should be encouraged by attention to abdominal and thoracic emptying of dilated lymphatics. Often a venous overload component also responds to

central emptying. Treatment of the lower limb without first clearing central overload of lymphatic and venous systems leads to diversion of lymph into the genitalia.

The most common manoeuvres employed for genital swelling are surgical. More studies are needed, with long-term follow-up, to assess at what level of health systems this is best done. Huge numbers of affected people are waiting for surgery often at small community hospitals where complications of surgery are not easily addressed. Removal of an inflamed lining of the testis by the standard hydrocelectomy is common. While successful debulking of huge scrotal swelling is highly regarded by those affected, recent surveys in West Africa indicate the need for longer follow-up of such procedures and more detailed analysis.

6.12 LYMPHOEDEMA 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 perilymphatic 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, colonized bacterial contamination, and healthy granulation tissue. With compression and control of the lymphoedema, 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 peri-wound lymphoedema will result in enhanced wound healing ([3]).

In the author's clinic, more than 80% of patients presenting with lower extremity, non-venous chronic wounds have demonstrated generalized or peri-wound lymphoedema (data accumulated April 2000 to March 2001). The degree varied

from a trace to 4+ pitting. These findings were seen in multiple types of wounds, i.e. ischaemic, diabetic, and traumatic. In many instances in long-standing wounds, the elimination of the lymphoedema enhanced the rate of healing dramatically.

6.13 PATHOPHYSIOLOGY OF WOUND-RELATED LYMPHOEDEMA

The most obvious effect from lymphoedema is swelling. This can result in abnormal function at both the tissue and cellular level. The distance between tissue channels can affect metabolic exchange, causing a shift toward anaerobic metabolism. Because cells are more widely separated, the exchange of gases between plasma membranes is likely to be affected. In chronic venous insufficiency, the removal of lymphoedema results in a significant increase in intracutaneous oxygen tension ([40]). Capillaroscopy has shown that oedema reduction increases the density of skin capillaries ([41]).

Alterations in tissue produced by simple injections of protein are almost identical with the changes observed in subacute and chronic lymphoedema ([42]). Mani & Ross stated: "The chronic effects of edema on the visco-elastic properties of connective tissue are unknown. It is reasonable to assume that pools of edema will squash, squeeze, or stretch the crimping and orientation of dermal collagen bundles" ([43]). Unna originally described this event and emphasized elastin destruction ([2]).

Open wounds studied by the injection of dye have demonstrated significant reduction in lymphatic channel regeneration as compared with arterial and venous angiogenesis ([44]). Trauma increases lymphatic flow, and outflow obstruction with the accumulation of waste products generated in the wound healing process is a likely inhibitory factor in wound healing ([45]). Collections of interstitial or third space fluid characterize tissues surrounding acute and chronic wounds. This collection of fluid mechanically compromises the micro-vascular and lymphatic system, thereby increasing capillary and venous after-load. Consequently, the delivery of oxygen and nutrients and the discharge of toxins and inhibitory factors are affected ([46]).

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 ([47-48]). Argenta & Morykwas (1997), in their investigations related to vacuum-assisted closure (VAC) of wounds, have provided valuable insight into the consequences of lymph stasis and the healing wound ([49]). Their technique removes chronic lymphoedema, which contributes to increased blood flow and enhanced formation of granulation tissue.

6.14 LYMPHOEDEMA THERAPY AND THE OPEN WOUND

MLD and continued compression are the cornerstones of treatment for non-wound-related lymphoedema. However, using MLD to alleviate lymphoedema associated with the open wound is neither time nor cost efficient. After the wound has healed, CDP may be indicated if persistent swelling is a problem. In the treatment of chronic venous leg ulcers, improving the efficiency of the calf muscle pump by using compression bandages and encouraging ankle flexion and extension is widely accepted as essential for proper care. It is reasonable, then, to postulate that the therapy for lymphoedema, lymph stasis associated with the open non-venous wound, is also compression. Limb elevation, when practical, is obviously helpful. In addition to the accepted dicta of modern wound care, the reasoned use of compression with short stretch, long stretch or combinations of such bandages is designed to create a dynamic wound dressing. Diuretics are rarely indicated as primary therapy, and they can impair fluid mobilization by extracting water from the lymph ([26]). Diuretics are useful in treating limb swelling when a significant degree of oedema superimposed on the underlying lymphoedema is evident, as in chronic congestive failure.

6.15 FAMILY AND COMMUNITY SUPPORT

Self-management for life is a significant burden. The daily regimen and the list of guidelines provided to those affected by lymphoedema require a high level of concordance. The world's leading clinics, such as those initiated by the Foeldi Clinic in Bavaria, Gerusa Dreyer in Brazil, and the Institute of Applied Dermatology in Kerala, provide the encouragement that helps to sustain compliance. Often, as in India, the family and members of the patient's community are trained to give the necessary support; the Mossy Foot Association of Ethiopia and the Patient Support Peer Group Cooperatives of India are examples.

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INFECTED WOUNDS

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7.1 DEFINITION OF AN INFECTED WOUND

Infection is "invasion and multiplication of microorganisms in body tissues, which may be clinically apparent or result in local cellular injury because of competitive metabolism, toxins, intracellular replication or antigen-antibody response" ([1]). Contamination or colonization of a wound without signs or symptoms of clinical infection is not a wound infection ([2-3]). Chronic ([4]) or acute ([5]) wounds densely colonized with organisms often heal without clinical signs of infection.

7.2 EPIDEMIOLOGY: THE BURDEN OF INFECTED WOUNDS

A wound infection may develop in the community or any care setting in acute or chronic wounds, increasing morbidity and mortality. Though costs differ in different health-care systems, wound infections add significantly to health-care costs. One Canadian chart review in a tertiary care hospital reported additional hospital costs for inpatient care of US$ 3937 per wound infection, mainly nursing and hotel costs, and an additional 10.2 days per case, all directly attributable to wound infections ([6]).

A USA study of matched paediatric patients with and without surgical site infections (SSIs) reported that wound infections increased the hospital stay by an average of 10.6 days and costs of patient care by US$ 27 288 for each patient with a potentially preventable surgical site infection (SSI) ([7]). A study of 242 704 patients over 65 years of age hospitalized in the USA reported that those with an SSI were more than three times more likely to die and experienced more than twice the length of stay, drug expenses and laboratory charges compared to those without an SSI ([8]).

Reported incidence or prevalence of example clinical wound infections in Table 7.1 vary widely depending on contaminating organism types and levels, wound etiology or surgical procedures, patient variables, and varying definitions and methods of reporting clinical wound infection.

Hospital-acquired SSI post-operative surveillance extends to 30 days post-surgery

or up to 1 year if an implant is involved to track SSI occurring after early hospital discharge or in the outpatient surgery setting. An SSI can be classified ([9]) as:

- superficial incisional SSI: inflammation of the skin and subcutaneous tissue of the incision;
- deep incisional SSI: involving deep soft tissues of the incision;
- organ/space SSI: involving any part of the anatomy that is not the incision.

TABLE 7.1 REPORTED EPIDEMIOLOGY OF EXAMPLE WOUND INFECTIONS

WOUND ETIOLOGY	PERCENTAGE REPORTEDLY INFECTED	COMMENT OR COMPARATOR
Burn wounds (2nd intention)	4.7% with gauze primary dressings ([10])	3.9%: occlusive dressings
Skin graft donor sites	6.4% with gauze primary dressings ([7])	2.7%: occlusive dressings
Chronic ulcers (general)	6.5% with gauze primary dressings ([7])	1.1%: occlusive dressings
Diabetic foot ulcers	6.0% with gauze primary dressing ([3])	2-2.5%: hydrocolloid dressed ([3])
Pressure ulcers	25% ([3])	estimated % osteomyelitis
Venous ulcers	< 5% ([3])	often polymicrobial ([3])
Mixed etiology or other	9.3% with gauze primary dressings ([7])	4.3%: occlusive dressings ([7])
HOSPITAL-ACQUIRED INFECTION (HAI)		
CVC-related septicaemia ([11])	10.9%	non-surgical patients: 11.6%
HAI per 1000 bed-days	21% in uninfected surgical patients	23.4% with surgical infection
Surgical site infections (SSI)	With prophylactic antibiotics	No or unspecified antibiotics
Clean	2.1% ([24])	1.5–5.9% ([3, 24])
Clean-contaminated	3.3% ([24])	6–9% ([24])
Contaminated	6.4% ([24])	13–20% ([24])
Dirty	7.1% ([24])	27% ([12])–40% ([24])
General		3.4–9.4% ([3])
Orthopaedic		2–6.8% ([3])
TRAUMATIC WOUND INFECTIONS		
Abscesses	30–50% ([3])	polymicrobial ([3])
Bites: cat or dog/human	30–50%([3]) / 20% ([3])	
Necrotizing infections	100% by definition	47% polymicrobial ([3])
Varied etiologies	50% ([3])	polymicrobial ([3])

7.3 PATHOGENESIS (2, 13)

Bacteria, fungi or viruses may enter a wound through a break in the skin or local circulation. If the host immune, circulatory or other systems are compromised or challenged, for example with extensive injury, immunosuppression or foreign matter, these organisms may establish colonies, proliferate and release enzymes and toxins that initiate local inflammation. This situation presents clinically with the classic signs and symptoms of infection: erythema, oedema, elevated temperature, pain, unusual wound odour and/or purulent exudate ([13]). If unchecked by either host defences or appropriate therapy, local infection may escalate into regional, organic or visceral infection or systemic sepsis ([13]).

7.4 RISK FACTORS FOR DEVELOPING A WOUND INFECTION (13, 14)

Common infection precursors for all wounds include prior antibiotic usage predisposing to proliferation of resistant microorganisms, current or prior use of invasive procedures or devices, and decreased host immune competence.

Open wounds such as chronic ulcers, traumatic wounds or burns healing by second intention may have different risk factors than primarily closed wounds, and risk factors vary with wound etiology. Diagnostic biopsies were reported to be more likely to become infected if performed on the ward as compared to the outpatient operating theatre, if elliptical incisions were not sutured, if the biopsy site was below the waist or in patients who smoked or were taking corticosteroids ([15]). In burn patients, the greater the proportion of body surface area that was burnt, the greater the risk of a wound infection. Patients with burns, often the very young in developing countries, have a higher mortality rate if their wound becomes infected ([12, 16]) and a lower likelihood of infection if the burn is located on the face ([17]). One Nigerian cohort study reported that 40% of 102 successive infected wounds identified during six months in two institutions resulted from trauma, mainly to the extremities ([18]).

For closed surgical wounds, a simple index adding 1 for each of the four major risk factors listed below had predictive validity of 0.67 to 0.70 for identifying patients at risk of an SSI in large populations, with a score of 2 to 4 indicating high risk ([19]). Wound contamination levels alone had predictive validity of 0.36 ($P<0.0001$). The four most important risk factors for an SSI are:

- an operation involving the abdomen;
- operation lasting more than two hours;
- a patient with three or more underlying diagnoses;

- contaminated surgery or a dirty operation according to the following standard classification ([13]):
  — clean: aseptic elective non-traumatic surgery of primarily closed uninfected and uninflamed tissue, not drained or penetrating the respiratory, alimentary, genitourinary or oropharyngeal tract;
  — clean-contaminated: minor break in technique, mechanical drainage; above tracts are entered without yielding significant positive cultures;
  — contaminated: major break in technique (e.g. open cardiac massage); open fresh traumatic wounds; acute, non-purulent inflammation present, contacting fluid from one or more above tracts;
  — dirty and infected: old traumatic wound involving clinical infection or perforated viscera; contact with acute bacterial inflammation and pus.

Clean, technically excellent operating technique is the most effective way to reduce risk of an SSI, ([13]), which is further reduced by giving appropriate peri-operative antibiotics and special attention to additional risk factors including:

- patient factors such as morbid obesity, advanced age, disease severity, protein-calorie malnutrition and probably systemic infection, diabetes or cancer;
- technical problems with the operation such as bleeding, increasing devitalized tissue or the need for a drain in the wound;
- presence of such virulent organisms as *Staphylococcus aureus* and *Streptococcus pyogenes*.

7.5 DIAGNOSING AN INFECTED WOUND

Early proper diagnosis and treatment of a wound infection can prevent pain, morbidity, mortality or amputation, and significantly reduce costs of care. Biologically, a wound is infected if microorganisms are invading surrounding healthy tissue, but it is not always easy to discern early signs of a wound infection before it becomes serious. One systematic review ([20]) of validity and reliability of diagnostic criteria for wound infection reported that the most commonly used symptoms of wound infection were: wound discharge (purulent or otherwise); redness or erythema; swelling or oedema; pain; tenderness; heat; pyrexia; dehiscence; and separation of wound edges.

The numbers of organisms in either swab or biopsy samples of burns are poor predictors of clinical outcomes ([21]). The numbers of microorganism isolated from wounds lack diagnostic capability in the absence of clinical symptoms ([2, 14, 22]). For example, wounds richly populated with bacteria may heal without clinical signs of

infection and, conversely, bacteria are often not isolated when cultured from early wound infections.

To diagnose infection in chronic wounds possibly inflamed due to repeated tissue damage, independent of microbial burden, Cutting & Harding ([23]) suggested using secondary wound infection symptoms. Such symptoms include serous drainage with concurrent inflammation, delayed healing (e.g. failure to decrease in area by at least 20% in four weeks), discoloured granulation tissue, pocketing at the wound base, foul odour, and wound breakdown as diagnostic signs of infection.

Reliably measured clinical symptoms in chronic wounds with highest sensitivity in identifying those with $>10^5$ microorganisms per gram of soft tissue ([24]) were delayed healing and friable (easily caused bleeding) granulation tissue. Symptoms with the best power to discriminate between wounds with or without high bio-burdens were, in decreasing order, friable granulation tissue, foul odour, oedema, delayed healing and serous exudate. Wound breakdown and increasing pain had the highest positive predictive value of high bio-burdens.

To plan appropriate therapy for a wound with clinical symptoms of infection, the infecting organism and its spectrum of antibiotic, antifungal or antiviral sensitivity should be identified. Infecting organism(s) may be sampled either by biopsy or by rotating a sterile swab over 1 cm^2 in the cleansed wound centre with sufficient pressure on the wound surface to extract fluid from inside the wound (Levine's ([25]) technique) using quantitative swabs or irrigation aspiration. Quantitative swab techniques are less invasive options for isolating organisms in clinically infected chronic or acute wounds with high sensitivity in detecting infecting microorganisms ([2, 26]). Those without access to a microbiology laboratory must diagnose wound infection based on clinical symptoms alone and risk giving inappropriate antimicrobial therapy.

7.6 PREVENTING WOUND INFECTIONS (3, 27)

Several specific activities may be used to prevent infection in wounds of specific etiologies.

- Burn wound infections are less likely with early aggressive debridement of devitalized tissue and serial burn excision and grafting ([28]) while optimizing patient immune competence, fluid intake and nutrition.
- To prevent chronic wound infection one alleviates causes of tissue damage or ischaemia, debrides necrotic tissue, optimizes patient immune competence and nutrition.

- To prevent an SSI:
- initiate prophylactic antibiotic treatment 1 hour prior to surgery and discontinue the use by 24 hours post-surgery;
  — avoid shaving; clip if hair removal is needed;
  — use aseptic good surgical technique, avoiding excess bleeding or tissue damage;
  — all contacting professionals should wash or disinfect hands thoroughly;
  — debride necrotic tissue;
  — restore tissue perfusion;
  — optimize patient immunity and nutrition;
  — engage in infection surveillance with feedback to operating clinicians and staff;
  — delay closure of the wound if it is heavily contaminated.
- For traumatic wounds, debride dead tissue, drain or irrigate the wound to reduce microbial load, and apply an appropriate antibiotic for high-risk wounds such as punctures.

7.7 INFECTED WOUND TREATMENT

The treatment of choice for a wound infection is systemic administration of an antibiotic or antimicrobial agent to which the infecting organisms are susceptible. In open wounds, broad spectrum topical antimicrobial agents, such as silver dressings applied to diabetic foot ulcers ([29]), slow-release iodine dressings applied to venous ulcers ([30]) or topical honey, reported to have antimicrobial properties ([31]), have significantly improved chronically delayed healing. Infection rates in minor wounds were reduced by topical first-aid use of a combination antibiotic gel as compared to placebo and povidone iodine cream ([32]). Careful attention to preventing transmission of antibiotic-resistant yeasts and bacteria ([13]), such as methicillin-resistant *Staphylococcus aureus* (MRSA) or vancomycin-resistant enterococci (VRE), is of utmost importance in all health-care settings and the community. Patients are more likely to have a drug-resistant strain of an organism if they experienced antibiotic use in the past month, had an MRSA infection, or had recent close contact with a person having a similar infection ([33]). Early identification and appropriate treatment of antibiotic-resistant organisms is vital, because destruction of the body's normal flora increases the risk of colonization by antibiotic-resistant organisms. Conclusive evidence on techniques to restore normal body flora to combat overgrowth by drug-resistant organisms is lacking.

Antiseptics as introduced by Lister and others, such as povidone iodine, hypochlorite and acetic acid, help sterilize instruments and operating surfaces, and

have contributed to the survival of millions. This is especially true for those wounded by trauma. In this situation, antiseptics are unquestionably useful for the control of bacterial infection. However, their use in open wounds should be carefully weighed against their capacity to damage local tissue and delay healing ([34]), adding to local necrotic tissue that serves as a focus for infection. The use of such antiseptics has been discouraged when healthy tissue is present in a wound.

7.8 RESEARCH PRIORITIES

Priorities for infected wound research to address global patient needs include:

- identify and implement techniques appropriate for under-resourced countries to reduce SSI and trauma wound infections so that years of healthy life saved by the WHO Global Initiative for Emergency and Essential Surgical Care (GIEESC) in improving access to life-saving surgery in developing countries are not lost to wound infection;
- determine more precisely risk factors for wound infection in chronic ulcers and other wounds healing by secondary intention;
- identify and develop strategies to deter the rise in antibiotic-resistant strains of organisms infecting wounds;
- develop simple, rapid, inexpensive techniques for identifying infecting organisms and their profiles of sensitivity to antimicrobial agents in order to improve therapeutic decisions for infected wounds.

7.9 ROLE OF WHO AND COLLABORATING ORGANIZATIONS

Working together to reduce wound infections of all types in resource-poor settings, WHO and collaborating organizations can:

- Implement infection surveillance and reporting programmes for acute and chronic wounds in resource-poor settings, including monitoring antibiotic-resistant strains of organisms, while engaging those in resource-poor settings to identify and address:
  - — key risk factors for wound infection;
  - — simple, effective inexpensive processes and modalities to meet those needs;
  - — causes and cures for wound infection across the community and care settings;
- implement pilot wound infection prevention and management programmes supporting the GIEESC;
- learn from successes and challenges how to improve wound infection outcomes in resource-poor settings;

- progressively improve the pilot programmes until quality clinical outcomes are consistently achieved;
- communicate globally those aspects of wound infection management that are effective in resource-poor settings.

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PRESSURE ULCERS

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EDITORS' NOTE

Data on pressure ulcers in developing nations are not readily available.

8.1 EPIDEMIOLOGY: THE BURDEN OF PRESSURE ULCERS

A pressure ulcer (PU), also known as *Decubitus ulcer*, pressure sore or bed sore, is a localized area of soft tissue damage that results from external pressure, friction or shear forces applied to the skin. PUs increase the length of hospital stays, morbidity and mortality and reduce quality of life. The likelihood of developing a PU increases with length of stay in the care setting, increasing age, or years after spinal cord injury. Sixty-six per cent of patients with orthopaedic fractures develop a PU during hospitalization ([1]). In the USA, PU-related hospital stays increased from 280 000 cases in 1993 to 455 000 cases in 2003. The point prevalence of a PU in home care patients is 3–10%. In the United Kingdom in general practice, the annual prevalence among patients at least 65 years of age was 0.31–0.70%, with PU incidence increasing sharply in patients over 85 years of age to 3.3 per 100 person years for people over 95 years of age ([2]). During hospitalization, about 10% of the elderly are likely to develop a PU. The costs of treating PUs in the USA grew from an estimated US$ 1.3 billion in 1992 to US$ 17.2 billion in 2003, with average charges of US$ 21 675 per PU treated ([3]). Prevalence, incidence and cost reporting methods vary, as do cost components within different health-care systems, so the true magnitude of the global burden of PU is difficult to estimate.

8.2 PATHOGENESIS (1, 3)

A PU can be caused by direct pressure, friction or shear stress singly or in combination. Direct external pressure exceeding the local skin capillary pressure can occlude local circulation. Friction, such as that caused by dragging a patient across a bed or chair surface may stretch or damage local blood vessels. Shear stresses arise when skin is held in contact with the supporting surface, but subcutaneous bone and tissue move, deforming and occluding local blood vessels. In all three cases, local circulation is impaired, depriving cells of oxygen and nutrients, so they die.

Generally the amount of tissue damage increases in proportion to the product of pressure x time applied, although less time is required to produce the same damage in patients who are at high risk due to poor health, damaged, fragile or macerated skin, or limited sensation, responsiveness or mobility.

8.3 PRESSURE ULCER DIAGNOSIS: PATIENT AND ULCER ASSESSMENT AND CLASSIFICATION

Assessing the individual with a PU is as important as assessing the ulcer, providing important clues to why ulcer tissue is breaking down or not healing. There is a need to carefully assess and plan appropriate management for patient factors that may delay healing or predispose tissue to further deterioration ([4]):

- history, e.g. alcohol, nicotine or other drug use;
- physical examination, including sensory or motor neural impairment and incontinence;
- co-morbidities such as diabetes or complications such as infection or prior scars;
- nutritional status, including fluid, protein, calorie, vitamin or trace element deficiencies;
- sources and extent of pain;
- psychosocial situation, including training home caregivers in turning or moving the patient;
- environmental factors possibly causing tissue breakdown, such as unsafe restraining practices.

Accurate reliable classification of a PU helps clinicians, staff and researchers document changes in PU status and communicate about its progress. Table 8.1 summarizes the defining characteristics of classification schemes available at the web sites ([5]) of the European Pressure Ulcer Advisory Panel (EPUAP) and USA-based National Pressure Ulcer Advisory Panel (NPUAP).

Consistent PU assessment can guide care decisions, track PU progress over time or alert caregivers that a PU is not healing expediently ([6]). Commonly used PU assessment parameters ([4, 7, 8]) include:

- Location identified by bony prominence underlying the PU, using anatomical descriptors.
- Size (recorded weekly): length, width, depth, undermining; for optimal accuracy in estimating area and % reduction in area to identify slow healing, use maximum length x maximum width, not head-toe length or side-side width ([4]).
- Stage or Grade: highest ever recorded; benchmark for the deepest extent of tissue damage.

- Surrounding skin: erythema, oedema, damage, maceration, rashes or bullae.
- Ulcer edge: bevelled, vertical, rolled, hyperkeratotic or calloused.
- Wound bed tissue type: % necrotic, % granulation tissue, % epithelium.
- Exudate: e.g. serous, serosanguinous or purulent.
- Exudate amount: e.g. none, scant, minimal, moderate, copious.
- Wound odour: presence or absence; onset of odour may signal infection or further tissue damage.
- Structure(s) visible in wound, e.g. hip prosthesis, tendon, bone.
- Pain (ideally patient reported): visual analogue scale or rating scale, e.g. none, mild, moderate, severe.

8.4 WHO IS AT RISK OF DEVELOPING A PRESSURE ULCER?

Several scales are available identifying individuals at risk of developing a PU ([9]). Those with the best documented validity and reliability include the Braden, Norton and Waterlow Scales in decreasing order ([10]). Most PU risk scales rate some form of the following Braden Scale parameters as high risk:

- Sensory perception or mental status: unresponsive or with responses only to painful stimuli.
- Moisture exposure of the skin: constantly or most of the time due to perspiration or incontinence.
- Activity, the degree of physical movement: bedfast or chairfast.
- Mobility: unable or with limited ability to change or control body position.
- Nutritional status: rarely or never eats a full meal; receives inadequate diet and fluids.
- Friction and shear: requires assistance to move or reposition the body.

8.5 PREVENTING PRESSURE ULCER DEVELOPMENT (9–13)

Ideally, a multidisciplinary team is needed to address all PU risk factors well. More frequent and more intense prevention interventions are required for patients at greater risk of developing a PU. When PU risk is assessed regularly and interventions are focused on addressing risk factors of high-risk patients, pressure ulcer prevalence, incidence and costs of care are reduced.

Effective PU prevention interventions include:

- use of appropriate support surfaces, reserving mobility-enhancing surfaces for low-motion patients:

— ideal support surfaces redistribute pressure, manage moisture, temperature, and microorganisms, reduce friction, last long with minimal service and are fail - safe ([12]);
- turning schedules with up to 2-hour frequency for the highest-risk patients:
  — using 30° lateral turns with foam wedges to facilitate turning;
- heel protection and frequent checking;
- avoid elevating the head of the bed more than 30 degrees:
  — avoid prone positions for tube-fed patients to prevent regurgitation or aspiration;
- chair positioning with the back tilted slightly backward, legs supported on a rest with heels free ([13]):
  — use special cushions or support surfaces with evidence of efficacy if available;
- protect the skin from moisture by minimizing episodes of incontinence:
  — when incontinence occurs, gently clean soiled skin, pat dry and apply a commercial skin barrier;
- prevent friction and shear by appropriate use of a trapeze or turning sheet, ankle or heel protectors or hydrocolloid dressings over bony prominences to prevent friction/shear during repositioning;
- identify and address all nutritional and fluid deficiencies;
- record and post PU incidence and prevalence, providing feedback to all involved in PU prevention.

8.6 PRESSURE ULCER TREATMENT AND PREVENTING RECURRENCE

Many of the same multidisciplinary team principles used to prevent a PU are useful in healing it and preventing its recurrence. In addition to those above:
- measure and post healing rates or percentage of PU of each Grade or Stage healed within 12 weeks;
- appropriate use of surgical interventions ([14]);
- avoid gauze dressings to reduce costs and maintain a physiological environment for healing ([15]);
- manage excess wound fluid with absorbent primary dressings, such as alginate or other gelling fibre dressings that do not damage the ulcer on removal ([16]).

Patients can help the healing process by: avoiding use of nicotine or other agents that restrict circulation; improving nutrition; observing and reporting early signs of deterioration or infection such as unusual inflammation; and assisting with pressure redistribution on schedule when feasible.

Multidisciplinary skin-care teams implementing evidence-based plans of PU prevention and treatment such as those described can improve PU prevention and treatment costs and outcomes ([9, 11, 14, 15]).

TABLE 8.1 EPUAP AND NPUAP PRESSURE ULCER CLASSIFICATION SCHEMES

EPUAP[A]	NPUAP[B]	DEFINING CHARACTERISTICS
May be included in Grade 4	Suspected deep tissue injury	NPUAP: Localized area of discoloured (purple or maroon) intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear; may progress from tissue that is painful, or unusually firm, mushy, boggy, or warmer or cooler than adjacent tissue
Grade 1	Stage I	EPUAP: Non-blanchable intact skin erythema; also discoloration, warmth, oedema, induration or hardness of the skin are helpful indicators on individuals with darker skin NPUAP: Localized area of red, non-blanchable intact skin, usually over a bony prominence. Evaluating blanching may be difficult in darkly pigmented skin, which may differ in colour from surrounding skin
Grade 2	Stage II	EPUAP: Partial-thickness skin loss involving dermis and/or epidermis. Ulcer is superficial, presenting clinically as an abrasion or blister NPUAP: Partial-thickness loss of dermis. Presents as a shallow open ulcer with a red or pink wound bed, without slough, or as an intact or broken serum-filled blister
Grade 3	Stage III	EPUAP: Full-thickness skin loss; damage or necrosis of subcutaneous tissue extends down to but not through underlying fascia NPUAP: Full-thickness tissue loss not exposing bone, tendon or muscle. Subcutaneous fat may be visible. Slough, undermining and tunnelling may be present but do not obscure depth of tissue loss
Grade 4	Stage IV	EPUAP: Extensive destruction, necrosis or damage to muscle, bone or supporting structures with or without full-thickness skin loss NPUAP: Full-thickness tissue loss with exposed bone, tendon or muscle. Ulcer bed may have some areas of slough or eschar Undermining and/or tunnelling may be present.
No similar grade	Unstageable	NPUAP: Full-thickness tissue loss. Ulcer bed is covered by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black), sufficient to prevent accurate staging

A European Pressure Ulcer Advisory Panel.

B National Pressure Ulcer Advisory Panel (USA).

8.7 RESEARCH PRIORITIES

Key research priorities that can help address global PU patient needs include:

- psychological, sociological, epidemiological and economic studies to determine the incidence, prevalence and burden of PU in under-resourced countries;
- developing and exploring efficacy of simple, convenient, reusable, inexpensive modes of pressure redistribution;
- developing and exploring efficacy of PU dressing materials and skin-care formulations that optimize healing while protecting PU from foreign body contamination or invasion by microbial or parasitic organisms.

8.8 ROLE OF WHO AND COLLABORATING ORGANIZATIONS

Working together to integrate pressure ulcer management across diseases in resource-poor settings, WHO and collaborating organizations can:

- assess the global burden of pressure ulcers and other chronic wounds in resource-poor settings, while engaging those in resource-poor settings to identify:
  - — local needs;
  - — acceptable modalities to meet those needs;
  - — issues to address in order to meet those needs effectively;
- implement pilot PU management programmes and assess clinical, social and economic outcomes;
- learn from successes and challenges how to improve PU outcomes in resource-poor settings;
- progressively improve the pilot programmes until quality clinical outcomes are consistently achieved;
- communicate globally aspects of PU management that are effective in resource-poor settings.

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VENOUS ULCERS

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9.1 EPIDEMIOLOGY: THE GLOBAL BURDEN OF VENOUS ULCERS

Venous ulcers (VUS), also known as *Ulcus crurus*, venous insufficiency ulcers or venous leg ulcers, are severe clinical manifestations of chronic venous insufficiency (CVI). They are thought to be the most common chronic wound among the ambulatory elderly, afflicting an estimated 0.2–1% of the total population ([1]) and 1–3% of those over 60 years of age at any given time ([1–3]), accounting for 60–70% of chronic ulcers of the lower limb ([3, 4]), with prevalence rising in western countries as the population ages ([4]). A VU profoundly decrease a person's quality of life ([2, 5, 6]) causing social isolation ([1]) and necessitating medical care and lost work time ([3, 7]). A VU occurs on average about 13 years after the onset of chronic venous insufficiency ([7]) and recurs after healing in 54–78% of patients ([2, 6]). A study in Brazil reported VU to be more prevalent in individuals with less economic resources and fewer years of education ([7]). Annual costs of VU management have been estimated at £200 million in the United Kingdom, US$ 1 billion the United States ([4]) and $AU 550–650 million in Australia ([8]), with each VU costing an estimated US$ 27 500 ([4]) to heal, and monthly home-care costs estimated at US$ 2500 ([9]).

9.2 PATHOGENESIS, SIGNS AND SYMPTOMS (4, 9)

CVI is the most common cause of a VU, typically associated with earlier deep vein thrombosis (DVT), congenital weakness of or repetitive stresses on venous valves or vasculitis, inflammation of the small vessels. A VU may also occur in combination with diabetes, peripheral arterial disease (PAD) or infection.

As walking flexes the calf muscle, venous blood is normally propelled upward towards the heart. Inside each large vein, valves act like tiny dams temporarily holding the blood until it is propelled further towards the heart by the next pump of the calf muscle. Damaged venous valves leak so that the lower leg bears more and more of the weight of the column of venous blood between heart and ankle. This increased pressure, called venous hypertension in the lower leg forces fluid out of the vascular system into interstitial space, resulting in lower leg oedema

when the patient stands or sits for prolonged periods. Oedema further impairs circulation and causes the itching, painful, inflamed skin called "venous dermatitis". If the oedema is not corrected or prevented, continued extravasation of fluid and cells cause hyperpigmentation, lipodermatosclerosis, white atrophy (*atrophie blanche*) and hyperkeratosis, ultimately resulting in a VU.

9.3 WHO IS AT RISK OF DEVELOPING A VENOUS ULCER? (9, 10)

Individuals at risk of developing a VU most commonly include those with:

- a family history of maternal CVI;
- thrombophilia, increased likelihood of blood clotting;
- personal history of DVT, diabetes mellitus, chronic heart failure or recent oedema;
- obesity or other conditions that prevent ankle flexing or calf muscle contraction;
- severe trauma of the lower leg;
- unusually vigorous exercise;
- a greater number of pregnancies for women.

9.4 DIAGNOSING A VENOUS ULCER (4, 9–11)

To plan appropriate therapy, the caregiver should determine if a patient's leg ulcer is caused by CVI as opposed to an arterial or other condition. The simplest way to check is to look for signs of:

- lower leg oedema, reduced by leg elevation;
- with the ulcer usually located on the medial aspect of the lower leg;
- palpable popliteal, dorsalis pedis and posterior tibial pulses help rule out arterial disease:
  — if oedema is severe, Doppler ultrasound may help assess the pulse;
- the ratio of ankle to brachial systolic blood pressure is usually between 0.9 and 1.0;
- increasing lower leg ache or pain as oedema increases during prolonged standing or sitting;
- capillary refill in the toes takes < 3 seconds for a VU or > 3 seconds for an arterial ulcer;
- the leg appears and feels normal in colour and temperature for a VU or cool for an arterial ulcer.

Other ulcer-causing conditions that should be ruled out through differential diagnosis include:

- neoplastic disease such as epitheliomas, sarcomas or lymphoma;
- metabolic disorders including gout, diabetes and deficiencies;
- haematological diseases such as sickle cell anaemia, thalassaemia, leukaemia and dysproteinaemias;
- topical chemical, thermal or physical insults, such as burns, frostbite, allergy or radiation;
- microbial challenges including bacterial, fungal, viral or protozoan infection;
- other conditions such as panniculitis or pyoderma granulosum.

9.5 VENOUS ULCER TREATMENT (4, 9–11)

Even a long-duration VU will usually heal within 12 weeks if consistently provided with:

- graduated, high (35 mmHg at the ankle decreasing to 10–15 mmHg at the infrapatellar notch) multi-layer elastic compression sustained for the full time it is worn;
- a physiologically moist environment for healing ([11]);
- if needed to manage excess wound fluid, absorbent primary dressings such as alginate ([12]) or other gelling fibre dressings that do not damage the VU on removal;
- management of surrounding venous dermatitis with non-sensitizing topical agents ([11]).

Patients can help the healing process by:

- stopping use of nicotine or other agents that restrict circulation;
- improving nutrition;
- recognizing and reporting early signs of deterioration or infection such as unusual inflammation;
- adhering to the prescribed compression, dressing and skin management regimens;
- avoiding scratching or other trauma to the VU or surrounding skin;
- regularly engaging in:
  — lower leg elevation above the heart periodically throughout the day until oedema recedes;
  — calf muscle contractions such as those associated with walking or ankle flexes.

9.6 PREVENTION OF VENOUS ULCER RECURRENCE (8–11)

VUs recur after healing in 67–90% of patients with CVI ([8]) because the underlying venous damage is permanent. Education about VU etiology, nutrition, infection signs, lower leg exercise, elevation and compression helps patients develop a sense of personal control over their VU and prevent VU recurrence by encouraging them to continue with their leg elevation and calf muscle exercise programmes, while consistently wearing appropriate lower leg compression, such as washable multilayer stockings sufficiently easy to apply and economical to encourage consistent use.

9.7 RESEARCH PRIORITIES

There are at least three main priorities for VU research that can address global VU patient needs:

- psychological, sociological, epidemiological and economic studies to determine the incidence, prevalence and burden of VUs in under-resourced countries;
- developing and exploring efficacy of simple, reusable, inexpensive modes of compression which are acceptable to and consistently used by individuals with CVI;
- developing and exploring efficacy of VU dressing materials and skin-care formulations that optimize healing while protecting VUs from foreign body contamination or invasion by microbial or parasitic organisms.

9.8 ROLE OF WHO AND COLLABORATING ORGANIZATIONS

Working together to integrate wound-lymphoedema management across diseases in resource-poor settings, WHO and collaborating organizations can:

- assess the global burden of venous ulcers and other chronic wounds in resource-poor settings, while engaging those in resource-poor settings to identify:
  - — local needs;
  - — acceptable modalities to meet those needs;
  - — issues to address in order to meet those needs effectively;
- implement pilot VU management programmes and assess clinical, social and economic outcomes;
- learn from successes and challenges how to improve VU outcomes in resource-poor settings;
- progressively improve the pilot programmes until quality clinical outcomes are consistently achieved;

- communicate globally aspects of VU management that are effective in resource-poor settings.

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DIABETIC FOOT ULCERS

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EDITORS' NOTE

Discussion of the neuropathic characteristics evident in the morbidity of the diabetic is applicable to other common entities, e.g. leprosy, spina bifida, paraplegia, quadriplegia, and progressive demyelinizing pathology.

10.1 DEFINITION OF THE DIABETIC FOOT

Various foot abnormalities result from peripheral neuropathy, macro-angiopathy, and other consequences of metabolic disturbances in patients with diabetes. Important clinical manifestations are foot ulcers, Charcot foot deformity, and amputation of parts of the foot or of the lower leg ([1]).

10.2 EPIDEMIOLOGY: THE GLOBAL BURDEN OF DIABETIC FOOT ULCERS

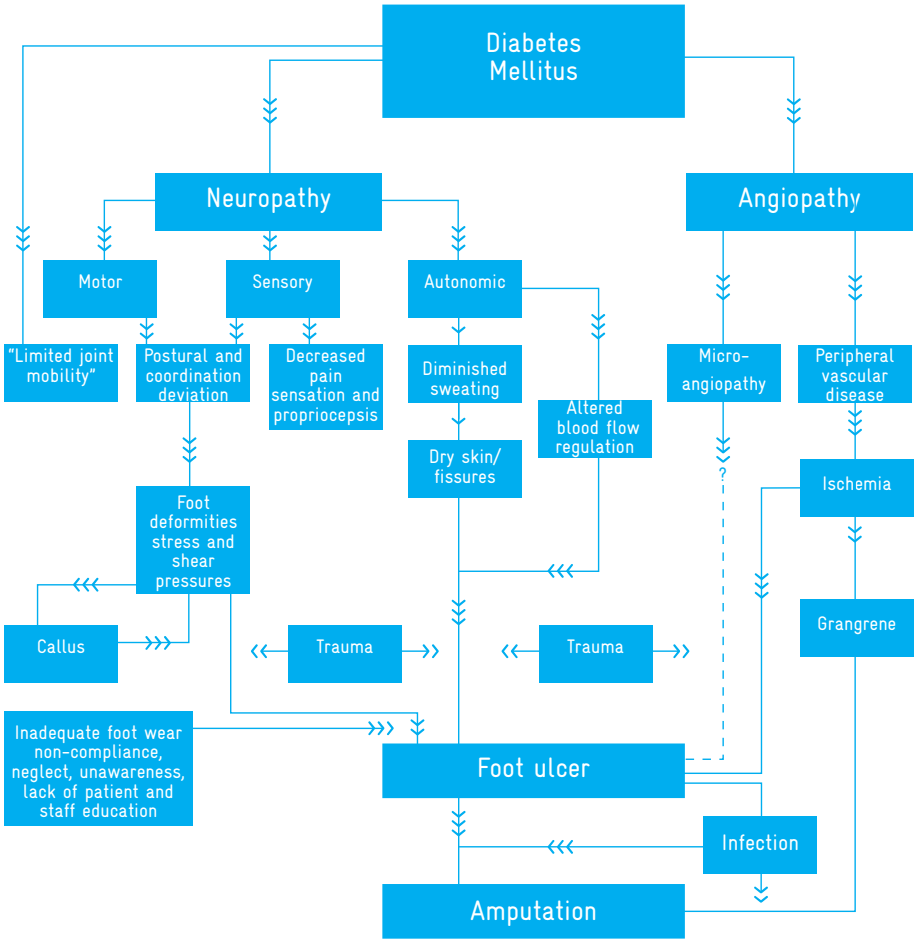
Diabetic foot problems occur in both type 1 and type 2 diabetes mellitus. They are more common in men and in patients over 60 years of age. A recent population-based study of more than 10000 patients in the north-west of England reported that 5% had past or present foot ulceration and almost 67% had one or more risk factors ([2]).

Ulceration is much more common in patients with predisposing risk factors; annual incidence rates in neuropathic individuals vary from 5% to over 7% ([3]). It is likely that the cumulative lifetime incidence of foot ulcers may be as high as 25% ([3]).

As up to 85% of amputations are preceded by foot ulcers, it can therefore be presumed that any successes in reducing foot ulcer incidence will be followed by a reduction in the number of amputations. To date, studies in Europe, with the exception of studies in Sweden and the Netherlands, have been disappointing in this respect ([4]). Studies from Germany have shown no evidence of a decrease in amputation in the last decade ([5]), whereas one report from the United Kingdom actually reported an increase ([6]).

In India and the United Republic of Tanzania, the estimated prevalence of diabetes for the urban areas is between 12% and 14% and in rural areas about 1–2% ([1]). Diabetic foot complications have a large impact on the quality of life of patients, and diabetic foot complications are a large economic problem, particularly if amputation results in prolonged hospitalization, rehabilitation, and an increased need for home care and social services ([1]). In these countries, approximately 3–4% of all diabetic patients have foot problems and use 12–15% of the health-care resources for diabetes. In some developing countries, foot problems may account for up to 40% of all available resources ([9]).

FIGURE 10.1 PATHOGENESIS OF DIABETIC FOOT ULCER



Source: International Consensus on the Diabetic Foot 2007 (1).

10.3 PATHOGENESIS, SIGNS AND SYMPTOMS

In the pathogenesis of diabetic foot ulcers (DFUs), neuropathy, angiopathy (ischaemia), foot deformity and limited joint mobility are central risk factors, as shown in Figure 10.1.

With regard to the etiology of foot ulceration, 45–60% of ulceration is thought to be purely neuropathic, 10% purely ischaemic and 25–40% mixed neuroischaemic. People in developed countries tend to be more often neuroischaemic ([7]). Because of the presence of diabetic neuropathy, many patients do not observe classic signs and symptoms such as pain. It has been observed that the local temperature of the foot increases prior to ulceration, as a result of inflammation thought to be most likely related to repetitive trauma ([8]). The underlying etiology of the ulcer is of great importance for ulcer treatment. In Table 10.1, some differentiating factors are mentioned.

TABLE 10.1 SIGNS AND SYMPTOMS RELATED TO THE ETIOLOGY OF DIABETIC FOOT ULCERS

NEUROPATHIC	ISCHAEMIC
Related to pressure	Related to ischaemia
Located at high-pressure areas	Located at end-arteries
Painless or burning pain	Painful
Callus	Gangrene

10.4 WHO IS AT RISK OF DEVELOPING A DIABETIC FOOT ULCER?

In addition to elderly patients, individuals at risk of developing a DFU most commonly include those with:

- diabetic neuropathy;
- peripheral vascular disease;
- foot deformity;
- previous history of foot ulceration;
- other micro-vascular complications;
- lower socioeconomic status.

10.5 RISK PROFILE OF THE DIABETIC FOOT

In Table 10.2 the risk profile and frequency of control as stated by the International Consensus on the Diabetic Foot 2007 ([1]) is shown.

TABLE 10.2 RISK PROFILE

RELATIVE RISK	PROTECTIVE SENSIBILITY	PERIPHERAL VASCULAR DISEASE	ELEVATED PRESSURE	ULCER, FOOT DEFORMITY
0	-	-	-	-
1	-/+	-/+	-	-
2	+	-/+	-/+	-
3				+

This risk profile has been evaluated and showed high predictive values in ulcer development.

10.6 DIAGNOSING A DIABETIC FOOT ULCER

The most important action that a health-care provider can take is to examine the foot. This requires the patient to remove shoes and socks so that the feet can be examined in detail for evidence of neuropathy, vascular disease, deformities, and trauma such as callus formation. If an ulcer is found, which means a complete absence of the epithelium, the caregiver should determine if a patient's foot ulcer is caused by diabetes, as opposed to other conditions. Location on pressures of the foot or heel is consistent with ulcers due to neuropathy and distally for ulcers due to vascular disease. Neurological testing with a Semmes–Weinstein monofilament and with a 128 Hz tuning fork ([1, 10]) confirms the presence of neuropathy. The presence of diabetes mellitus should be confirmed and other causes of neuropathy should be excluded.

Other ulcer-causing conditions that should be ruled out through differential diagnosis include:

- neoplastic disease such as epitheliomas, sarcomas or lymphoma;
- metabolic disorders including gout, diabetes and deficiencies;
- haematological diseases such as sickle cell anaemia, thalassaemia, leukaemia and dysproteinaemias;
- topical chemical, thermal or physical insults, such as burns, frostbite, allergy or radiation;
- microbial challenges including bacterial, fungal, viral or protozoan infection;
- other conditions such as panniculitis or pyoderma granulosum.

10.7 DIABETIC ULCER TREATMENT

The presence of vascular disease and of bone infection should be assessed. Patients can help the healing process by offloading the foot where the ulcer is present. This can be accomplished through the use of a variety of shoes or other offloading devices. The wound should be debrided and a moisture retentive dressing should be applied.

10.8 PREVENTION OF DIABETIC FOOT ULCER RECURRENCE

Because of the impact on quality of life and the high costs of DFU treatment, prevention is important and has proven to be effective. Preventive screening and education programmes are cost effective.

It is important to prevent diabetic complications through first achieving optimal glucose regulation, regulation of blood pressure and lipids, and smoking cessation. Specific tactics for DFU prevention are regular inspection and examination of both feet, identification of the foot at risk, education of patients, family and health-care providers, appropriate footwear and treatment of non-ulcerative pathology.

DFUs often recur after healing. Neuropathic foot ulceration often occurs because of the use of inappropriate footwear, and appropriate footwear with adequate depth and width is recommended to protect the feet. Examination of the feet by both the at-risk person and the caregiver should be performed regularly.

10.9 RESEARCH PRIORITIES

There are at least three main priorities for DFU research, in order to address global DFU patient needs:

- psychological, sociological, epidemiological and economic studies to determine the incidence, prevalence and burden of DFU in under-resourced countries;
- developing and exploring the efficacy of simple, reusable, inexpensive modes of offloading that are acceptable to, and consistently used by, individuals with DFUs;
- developing and exploring the efficacy of DFU dressing materials and skin-care formulations that optimize healing while protecting DFUs from foreign body contamination or invasion by microbial or parasitic organisms;
- evaluating the effectiveness of a screening and educational programme for diabetic foot complications, as for example already operational for leprosy.

10.10 ROLE OF WHO AND COLLABORATING ORGANIZATIONS

Working together to integrate wound-lymphoedema management across diseases in resource-poor settings, WHO and collaborating organizations can:

- assess the global burden of DFUs and other chronic wounds in resource-poor settings, while engaging those in resource-poor settings to identify:
  - — local needs;
  - — acceptable modalities to meet those needs;
  - — issues to address in order to meet those needs effectively;
- implement pilot DFU management programmes and assess clinical, social and economic outcomes;
- learn from successes and challenges how to improve DFU outcomes in resource-poor settings;
- progressively improve the pilot programmes until quality clinical outcomes are consistently achieved;

- communicate globally aspects of DFU management that are effective in resource-poor settings.

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ATYPICAL WOUNDS

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11.1 DEFINITION OF AN ATYPICAL WOUND

Wounds, especially non-healing wounds, can have several different causes. The most common causes of chronic wounds are long-standing diabetes mellitus (diabetic foot ulcers), poor arterial supply (arterial ulcers), venous insufficiency (venous leg ulcers), neurological deficit (neuropathic ulcers), and prolonged pressure (pressure ulcers or bed sores) ([1]). Wounds secondary to more unusual sources are called atypical wounds, and as a result some are rare and intricate in nature. Atypical wounds have a spectrum of etiologies that include inflammatory processes, vasculopathies, infectious disease, metabolic disorder, genetic disease, neuropathy, neoplastic origination, and external trauma or injury ([2]).

EPIDEMIOLOGY: THE BURDEN OF ATYPICAL WOUNDS

Out of 500 000 leg ulcers in the USA alone, an estimated 10% are caused by atypical or unusual etiologies ([1]). Examples of atypical causes of ulcerations are given in Table 11.1 and vary widely depending on wound etiology, patient variables and varying definitions and methods of reporting.

11.3 PATHOGENESIS

The pathogenesis of atypical wounds differs according to the etiology of the wound. One of the unifying features of atypical wounds is the presence of skin necrosis. Skin necrosis is a clinical manifestation of tissue death, most commonly due to alteration of cutaneous blood flow. This may occur from either blockage or destruction of vessels supplying the skin, either large vessels (peripheral arterial disease) or small vessels (thrombi or emboli). Destruction may be direct, as in vasculitis, or sickle cell anaemia, or indirect, as in pyoderma gangrenosum. Skin necrosis may be associated with a reticulated pattern called *livedo reticularis*, which outlines the involved cutaneous vasculature. Infectious disease and malignancy may

also present with skin necrosis. Figure 11.1 highlights a simple diagnostic algorithm. Vascular studies, biopsy for histology, and tissue cultures and laboratory tests are important in rendering a diagnosis.

11.4 DIAGNOSING AN ATYPICAL WOUND

An atypical etiology for a wound must be considered within one's differential diagnosis when: ([1]) the location of the wound varies from that of a common or chronic wound; ([2]) the clinical presentation is unique from that of a common or chronic wound; or ([3]) the suspicious wound fails to respond to standard treatment regimens ([1, 2]). Since many atypical wounds tend to present clinically as each other, a diagnosis based on visualization is nearly impossible and lesional biopsies are warranted and crucial for identification. Histopathological evaluation with adjunct studies such as special staining, tissue cultures, or immunofluorescence is essential to evaluate a wound properly once it is suspected to be of an atypical nature. It is also without question that a comprehensive history and physical examination be undertaken to diagnose accurately the cause of the wound. Factors to be taken into considerable account during the work-up are the following: epidemiological exposure, family history, personal or unusual habits, recreational and hobby activities, employment history, recent travel, sexual history, substance abuse history, known history of systemic illnesses, immunosuppression status, and laboratory blood testing ([3–5]).

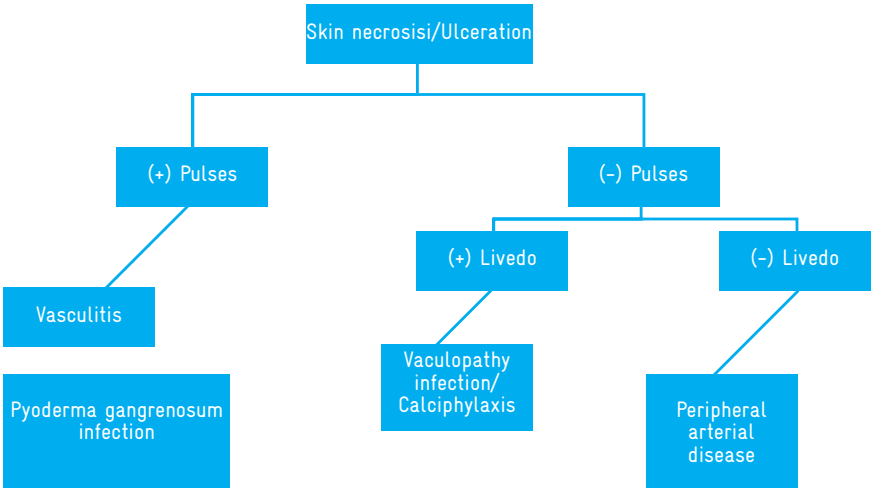
TABLE 11.1 EXAMPLES OF ATYPICAL WOUNDS

WOUND ETIOLOGY	EXAMPLES
Inflammatory ulcers ([3–12])	Vasculitis, pyoderma gangrenosum, lichen planus, sarcoid
Vasculopathic ulcers ([13–16])	Cryofibrinogenaemia,mixedcryoglobulinaemia,antiphospholipid syndrome
Haematological ([17, 18])	Sickle cell disease
INFECTIOUS DISEASE	
Atypical Mycobacteria ([19–21])	«M. ulcerans, M. abscessus, M. fortuitum, M. chelonae, M. marinum»
Deep or systemic fungus ([22–26])	Chromomycosis,pheohyphomycosis,sporotrichosis,rhinosporidiosis (deep fungi) Paracoccidioidomycosis, coccidioidomycosis, histoplasmosis, cryptococcosis (systemic fungi)
Bacterial/viral ([27–30])	«Vibrio vulnificus Bacteroides, Peptococcus, Fusobacterium, Clostridium/ Herpes simplex»
Metabolic ([31, 32])	Calciphylaxis
Malignancy ([33, 34])	Squamous cell carcinoma

11.5 ATYPICAL WOUND TREATMENT

Treatment of atypical wounds is based on the etiology of the wound. For example, inflammatory wounds are given anti-inflammatory treatment ([3–12]). Infectious wounds are treated with the appropriate antimicrobial therapy ([19–30]). Cancers are treated with surgical excision and/or chemotherapy agents.

FIGURE 11.1 DIAGNOSTIC ALGORITHM FOR ATYPICAL WOUNDS



11.6 RESEARCH PRIORITIES

Priorities for atypical wound research to address global patient needs include:

- determining more precisely risk factors for wound infection in chronic ulcers and other wounds healing by secondary intention;
- identifying and developing strategies to deter the increase in atypical wounds due to infectious causes;
- developing simple, rapid, and inexpensive techniques for identifying atypical wounds will improve therapeutic decisions.

11.7 ROLE OF WHO AND COLLABORATING ORGANIZATIONS

Working together to reduce wound infections of all types in resource-poor settings, WHO and collaborating organizations can:

- implement atypical wound surveillance and reporting programmes for acute and chronic wounds in resource-poor settings, while engaging those in resource-poor settings to identify and address:
  — key risk factors for atypical wounds;
  — simple, effective inexpensive processes and modalities to meet those needs;
  — causes and cures for atypical wounds across the community and care settings;
- implement pilot atypical wound prevention and management programmes supporting the WHO/GIEESC;
- learn from successes and challenges how to improve atypical wound outcomes in resource-poor settings;
- progressively improve the pilot programmes until quality clinical outcomes are consistently achieved;
- communicate globally aspects of atypical wound management that are effective in resource-poor settings.

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