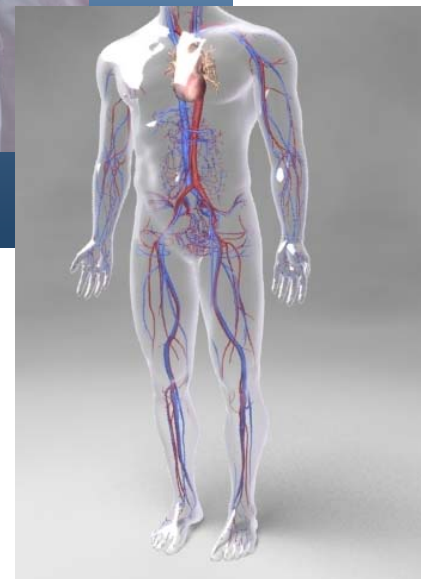


Chronic Venous Insufficiency and Venous Leg Ulceration Training Program



Produced by: Anne Somerville
Global Marketing
Jerry Hutchinson & John Chen
Global Research & Development
February 2009



CONFIDENTIAL – FOR INTERNAL USE ONLY.

This material is not to be used in sales presentations. This material is not to be left with any customers. This material is confidential and is for the use of ConvaTec Marketing Management and Sales Organization only.

Unless otherwise indicated ®/™ indicates trademarks of ConvaTec Inc.
©2009 ConvaTec Inc.

AP-000144-MM

Contents

Introduction

Chapter 1 – Skin and Wound Healing

Chapter 2 – The Impact of Leg Ulcers

Chapter 3 – Venous Leg Ulcers and Chronic Venous Insufficiency

Chapter 4 – The Care and Treatment of Venous Leg Ulcers

Chapter 5 – Glossary of Terms

Introduction

The aim of this training program is to provide you with a practical learning resource to develop your understanding of the disease state area of Chronic Venous Insufficiency (CVI) and Venous Leg Ulceration (VLU).

Learning objectives

On completion of the training program you should:

- Understand CVI and VLU at a level appropriate for a wound care professional
- Be able to communicate effectively with wound care professionals who provide care to venous leg ulcer patients
- Understand the phases of wound healing and be familiar with various factors that may disrupt or delay wound healing
- Recognize the key clinical features that distinguish between venous and arterial leg ulcers and understand the social, economic, and psychological burden of leg ulcers
- Know the characteristics and pathology of CVI, the common types of diagnostic evaluation and the relationship between CVI and venous leg ulcers
- Be familiar with the theory behind compression, understand why it is the cornerstone of therapy for venous leg ulcers and understand the limitations associated with different types of compression therapies available today

The training program consists of four chapters:

Chapter 1 introduces the skin, discussing its structure and function and the consequences for the skin when it is wounded. It details the stages of wound healing, the factors that may disrupt wound healing and general wound assessment. It also introduces the theory of moist wound healing.

Chapter 2 describes the most commonly occurring types of leg ulcers, that is, venous, arterial, and neuropathic leg ulcers and whom they affect within the population. It goes on to describe the specifics of ulcer diagnosis and the impact that leg ulcers have on the patient and the health system.

Chapter 3 looks at the causes of venous leg ulcers, the theories behind their formation, and explains their link with chronic venous insufficiency. It also describes the diagnostic tools that are used to determine the severity of underlying disease in patients with venous leg ulcers.

Chapter 4 discusses caring for venous leg ulcers and the treatment strategies that can be employed. Although the focus of this chapter is mainly on compression therapy, other management strategies are also discussed.

Chapter 5 is a glossary of terms.

At the end of each chapter there is a self assessment which will allow you to test your understanding of the information provided in the chapter.

Further reading

You may wish to read up on certain topics. Here are a few suggested references for further reading:

Chapter 1

- Tortora GJ, Grabowski SR. The Integumentary System. In: *Principles of Anatomy and Physiology* (7th edition). New York, NY: John Wiley and Sons; 1993:126-139.
- Quinn AG. Biology of the Skin and Dermatological Disease, *Medicine*. 2004; **32(12)**:1–3.
- Powell J. Skin Physiology. *Acute Care: The Foundation Years*. 2007; **3(5)**:193–196.
- Chettibi S, Ferguson MWJ. Wound Repair: An Overview. In: Gallin JI, Snyderman R, eds. *Inflammation: Basic Principles and Clinical Correlates* (3rd Edition). Philadelphia, PA: Lippincott, Williams and Wilkins; 1999:865–881.
- Grey JE, Enoch S, Harding KG. ABC of Wound Healing - Wound Assessment. *BMJ*. 2006; **332**:285-8.
- Hess CT. *Wound Care: Clinical Guide* (5th Edition). Ambler, PA: Lippincott Williams & Wilkins; 2005.
- Timmons J. Skin Function and Wound Healing Physiology. *Wound Essentials*. 2006; **1**:8-16.

Chapter 2

- Phillips TJ, Dover JS. Leg Ulcers. *J Am Acad Dermatol*. 1991;**25**:965–990.
- Phillips T, Stanton B, Provan A, Lew R. A Study of the Impact of Leg Ulcers on Quality of Life: Financial, Social and Psychosocial Implications. *J Am Acad Dermatol*. 1994;**31**:49–53.
- Etufugh CN, Phillips TJ. Venous Ulcers. *Clinics in Dermatology*. 2007;**25**:121–130.

Chapter 3

- Meissner MH. Acute and Chronic Venous Disease. Proceedings of the International Summit of the 5th Pacific Vascular Symposium. *J Vasc Surg*. 2007;**46**:**Supplement S**.
- Chronic Venous Insufficiency and Venous Ulceration – Aetiology and Treatment. ConvaTec Inc. 2008.
- Anderson I. Understanding Chronic Venous Hypertension. *Wound Essentials*. 2008;**3**:20-32.

Chapter 4

- Moffatt C, Martin R, Smithdale R. *Leg Ulcer management: Essential Clinical Skills for Nurses*. Oxford, UK: Blackwell Publishing; 2007.
- Moffat CJ. *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007.
- Partsch H, Clark M, Mosti G, et al. Classification of Compression Bandages: Practical Aspects. *Derm Surg*. 2008; **34 (5)**:600-609.
- European Wound Management Association (EWMA) Position Document: *Understanding Compression Therapy*. MEP Ltd.
- World Union of Wound Healing Societies (WUWHS). *Principles of Best Practice: Compression in Venous Leg Ulcers: A Consensus Document*. London: MEP Ltd, 2008.
- Anderson I. Aetiology, Assessment & Management of Leg Ulcers. *Wound Essentials*. 2006;**1**:20-37.

Chapter 1

Skin and Wound Healing

Welcome to the first chapter in your training program.

This chapter introduces the skin, discussing its structure and function and the consequences to the skin when it is wounded. It details the stages of wound healing, the factors which may disrupt wound healing and general wound assessment. It also introduces the theory of moist wound healing.

This chapter will be followed by three further chapters which will go into more depth on chronic wounds, specifically leg ulcers, chronic venous insufficiency (CVI) and the management of leg ulcers and CVI.

Topics to be discussed in this chapter include:

- The structure and function of the skin
- Phases of wound healing
 - Hemostasis and inflammation
 - Proliferation and granulation
 - Re-epithelialization
 - Maturation and remodeling
- Moist wound healing theory
- Factors which delay or disrupt healing
 - Systemic factors
 - Regional factors
 - Local factors
- Patient and wound assessment
 - Wound progression model
 - Wound classification
- Progression of wound to healing
 - Primary- versus secondary-intention healing
 - Wound management approaches
 - The extra challenges of managing chronic wounds

Upon completion of Chapter 1, you will:

- understand how wounds affect the structure and function of the skin and be familiar with the various overlapping phases of wound healing
- know the difference between different types of wounds and recognize the key clinical features between a wound that is healing well and one that is not
- have a basic knowledge of the benefits of moist wound healing
- be familiar with various factors that may disrupt or delay wound healing

Structure and Function of the Skin

The skin is the largest organ of the human body weighing approximately 4.5–5 kg and covering an average area of 2 m². It is a complex and multifunctional organ that contains many specialized cells that have adapted to perform a variety of different functions. The skin consists of three main layers. The surface layer that is exposed to the outside environment is called the epidermis. Underneath the epidermis is the dermis and beneath that is an underlying subcutaneous fat layer called the hypodermis. The dermis is well supplied with blood vessels and nerves. The skin also contains various functional units called skin appendages – sweat glands, sebaceous glands and hair follicles that contribute to the overall function of the skin.

Description of the structure and function of skin can be found in numerous articles. Here are a small number of references for further reading: Tortora & Grabowski (1993)¹; Quinn (2004)²; Powell (2007).³

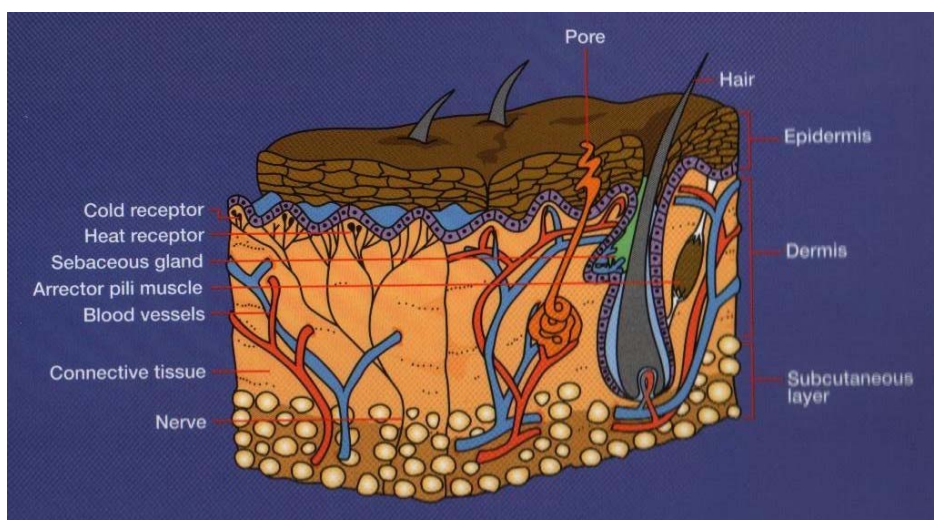


Figure 1. Schematic representation of the structure of skin

Structure of skin

The epidermis

The epidermis is made up of cells which waterproof and protect the skin and underlying tissues. The epidermis itself is divided into five layers. Each layer is filled with closely packed cells produced by cell division at the deepest layer. As more cells are created at this bottom layer, the cells are pushed up through the epidermis where they gradually flatten and die, finally reaching the uppermost layer called the stratum corneum from which they are shed. In normal skin it takes around 28 days from cell formation to shedding.

The thickness of the epidermis varies from one part of the body to the next. It measures only 0.1mm over the eyelids, whilst it is 2 mm thick over the soles of the feet.³

The dermis

The dermis is a complex structure, comprising many different cell types, which adapt to different functions. Fibroblasts are the predominant cell type in the dermis, and are mainly responsible for making and organizing the extracellular matrix that forms the bulk of the dermis. The major component of the dermal extracellular matrix is collagen, but it also contains many other proteins, proteoglycans and polysaccharides that collectively provide the stretch, bulk and shock absorption properties to the skin. The dermis also provides support and nutrients to the epidermis. The dermis itself is divided into two layers: the upper papillary layer and the thicker reticular layer below.³

Within the dermis:

Blood vessels form a superficial network in the papillary layer

Lymphatics perform drainage and form part of the immune defense system

Sensory nerves are used for touch, heat and pain

Skin appendages include hair follicles, sweat glands and sebaceous glands

The hypodermis or subcutaneous layer/tissue

Beneath the dermis sits the hypodermis, a layer of primarily fat and loose connective tissue which helps protect the internal structures of the body.

Function of the skin

The skin serves many vital functions for the body. A comprehensive overview of this subject can be found in Tortora & Grabowski (1993).¹

Regulation of body temperature – Adjustment of blood flow to the skin and levels of sweat production are the main ways the skin can contribute to regulating body temperature. Production of perspiration by sweat glands and subsequent evaporation from the skin in response to high temperatures or exercise helps to reduce an elevated temperature. Conversely, sweat production is reduced in response to a low environment temperature, just as blood flow to the skin is reduced in order to combat heat loss.

Protection – The skin provides a physical barrier that protects underlying tissues from physical abrasion, bacterial invasion, dehydration and UV radiation.

Perception of stimuli – The wealth of nerve endings and receptors located in the skin allow detection of stimuli related to temperature, touch, pressure and pain.

Excretion – Sweat plays a role not only in the regulation of body temperature, but also in the excretion of small amounts of water, salts and a few organic compounds.

Immunity – The skin contains a series of antigen-presenting cells, which process and present foreign antigens from intruder organisms and material to trigger the host immune response.

Blood reservoir – Extensive networks of blood vessels, which carry 8–10% of the body's blood flow, can be found in the dermis.

Synthesis of vitamin D – Upon exposure to sunlight (UV light).

Wound Healing

When the skin remains intact it is able to perform all of its required functions. When the skin is breached its functions are disrupted so it is important to heal the wound as quickly as possible in order to prevent infection and allow normal skin functioning to return.

The majority of wounds will heal without difficulty. When a wound becomes chronic and non-healing (for example, in the case of venous or arterial leg ulcers), the challenge is first to diagnose the cause(s) of non-healing correctly so as to allow appropriate treatment, in order to restore the healing capability. In addition, appropriate management needs to be applied to control symptoms and prevent complications, and to improve a patient's quality of life.⁴

Stages of healing

Wound healing results from a complex interaction of cells with their biochemical and physical environment. It is traditionally described in four sequential overlapping stages: hemostasis and inflammation, granulation, re-epithelialization, and remodeling [see Chettibi & Ferguson (1999)⁵ for a comprehensive review of this subject].

Hemostasis and inflammation

When living tissue is injured, platelets spilling from damaged blood vessels adhere to the damaged tissue and initiate hemostasis by releasing thrombin. This converts fibrinogen to fibrin, forming a clot which stops blood loss. Simultaneously, platelets release many chemical substances, including growth factors. Some of these cause vasoconstriction, whilst nearby intact blood vessels dilate and become more porous. As blood vessels dilate, elevating the local temperature and increasing blood flow leading to erythema, pores between the endothelial cells enlarge, and some of their contents leak into the surrounding tissues. This vasodilation and extravasation produces edema, pressing local nerve endings, thus causing pain. Clinically, inflammation is recognized by signs of erythema, edema, pain and elevated local temperature. Circulating white blood cells, called polymorphonuclear neutrophils (PMNs), are attracted by the chemical cocktail released by platelets. Within hours of wounding, they adhere to the endothelium of nearby blood vessels, and migrate into surrounding tissue to defend the body against microbial invasion by killing microorganisms and phagocytosing debris. They also set the stage for healing to begin by releasing enzymes and chemoattractants for macrophages which arrive within two days of wounding. They signal the beginning of the second healing stage, granulation. Both PMNs and macrophages release enzymes and engage in destruction and phagocytosis of debris and microorganisms.

Macrophages, as well as activated platelets and endothelial cells, also secrete their cocktail of growth factors and other intercellular signaling molecules which trigger healing. Macrophages, endothelial cells and platelets are therefore considered the key cells orchestrating the healing process.

Proliferation and granulation

In dermal wounds, growth factors and other intercellular signaling molecules from macrophages, endothelial cells and platelets attract dermal fibroblasts, stimulating their proliferation and synthesis of the collagen matrix through which the new blood vessels, formed by endothelial cells, will develop. Together, these elements form a new provisional dermis, called granulation tissue, until it is covered with a new epithelium. Many new tiny capillary loops cover the surface of this healthy new dermis, giving it a red, granular appearance. Some fibroblasts in granulation tissue respond to the cocktail of growth factors and other signaling mediators by organizing their actin and myosin bundles into contractile organs, thus drawing the wound edges inward, in a process known as wound contraction. These cells, called myofibroblasts, are particularly noticeable in full-thickness wounds.

Re-epithelialization

In shallow, partial-thickness wounds which do not extend beyond the dermis, and in late granulation of deep wounds, once the new dermis approaches the level of the original skin, nature uses another way to close wounds. Keratinocytes are stimulated by the growth factor cocktail from the inflammatory macrophages and fibroblasts, and also by the new dermis rich in collagen, hyaluronan and fibronectin. These keratinocytes, found at the base of the epidermis or any epidermal appendages, such as hair follicles, sebaceous glands or sweat glands, migrate over the dermal surface and proliferate, until the dermis is completely covered with a new epidermis. As they proliferate, they push their daughter cells upwards away from the dermis, where they gradually lose their nuclei and flatten, forming the outer epidermal barrier (the stratum corneum). This shields the body from external contaminants and provides an effective fluid and vapor barrier.

Maturation and remodeling

Granulation tissue, as the provisional dermis, is structurally fragile. After re-epithelialization, the extracellular matrix in the granulation tissue is gradually replaced by new, stronger extracellular matrix. This remodeling process, which takes place over many months, nevertheless cannot result in the skin completely regaining its original pre-injury strength. This remodeling process is mainly orchestrated by the fibroblasts and macrophages, and abnormalities in this phase can lead to pathological scarring, such as keloids and hypertrophic scars.

Moist Wound Healing Theory

Early accounts of moist wound healing principles were described as early as in Egyptian papyrus dating back to 1400 BC when a “paste of honey, grease, and lint” was applied to wounds.⁶ However, it was not until a British scientist, George Winter, who published a paper in 1962⁷ describing the moist healing benefits at the cellular level in swine, that the current interest in providing a moist environment for wound healing was triggered. Winter’s work, published in the prestigious journal *Nature*, sparked an era of research which has now confirmed that there is faster healing, autolytic debridement, fewer infections and less pain in moist compared with dry wounds.⁸

Field & Kerstein (1994) suggested that a moist environment, compared to a dry environment, is beneficial to the wound healing process for the following reasons:⁸

- prevention of tissue dehydration
- accelerated angiogenesis
- increased breakdown of dead tissue and fibrin
- reduction in pain
- faster healing time

Healing time with conventional dressings versus hydrocolloid occlusive dressings

Field & Kerstein (1994) carried out a survey of clinical data on healing time of acute partial-thickness wounds (skin graft donor sites and second-degree burns) and reported statistically significant faster healing times for wounds dressed with moisture-retentive hydrocolloid occlusive dressings in comparison to gauze-type conventional dressings.⁸

Reduction in pain

There are many clinical reports suggesting reduction in pain at rest and on dressing changes for patients with moisture-retentive dressings, in comparison with conventional dressings, as reviewed by Field & Kerstein (1994). The interface between the dressing and the wound that keeps nerve endings moist may be a contributory factor to pain reduction, and makes dressing changes more comfortable.⁸

Incidence of infection

Despite initial fears that wound infection would be promoted by occlusive dressings, the immediate application of an occlusive dressing does not increase the incidence of clinical infection, and may reduce the risk of infection compared to non-occlusive dressings.^{8, 9}

Factors which Delay or Disrupt Healing

Many factors can disrupt wound healing. These can be classified into systemic, regional and local factors.

Systemic factors

Systemic factors are either caused by disease, lifestyle or age. Those that affect the whole body can either compromise healing directly, or can lead to regional factors that compromise healing. The following are the important systemic factors that may disrupt healing:

Vascular and inflammatory diseases – It is now understood that chronic or uncontrolled inflammation is a key driver that leads to not only the creation of the wound but also its non-healing state. Circulatory diseases can lead to regional arterial and/or venous insufficiencies that in turn lead to local chronic inflammation and are recognized causes of arterial/venous/mixed etiology ulcers (see Chen & Rogers, 2007).¹⁰ There are also other types of inflammatory ulcers, e.g. pyoderma gangrenosum.¹¹

Metabolic disease – Metabolic diseases such as diabetes can lead to many factors that impair healing, including peripheral vascular disease, neuropathy and compromised immune response.¹²

Age – While it is accepted wisdom that wounds of aged patients heal slower than those of young patients, there is actually no strong clinical data to prove this relationship. Nevertheless this accepted wisdom can be considered a reasonable one, as there is substantial circumstantial evidence to suggest that many healing processes in aged subjects are compromised, including signal transduction, unchecked inflammation, and altered balance of protein synthesis and degradation that can affect the rate and quality of healing (see review of Ashcroft et al, 2002).¹³

General health, nutrition and lifestyle – Inadequate nutrition may influence the progression of healing because it affects the patient's general metabolism.¹⁴

Other systemic factors – Patients undergoing radiation therapy or chemotherapy may have compromised wound healing.¹⁵ Heavy smoking is also strongly associated with a large number of skin disorders including compromised wound healing.¹⁶

Regional factors

Regional factors are often consequences of the systemic factors described above. In particular, the consequences of vascular and metabolic diseases on regional blood supply and nerve function are well characterized [See Chen & Rogers (2007)¹⁰ and more details in subsequent chapters].

Arterial insufficiency – Arterial insufficiency as a consequence of vascular disease leads to tissue hypoxia and can also lead to regional tissue necrosis, which results eventually in infection and ulceration. Patients may sometimes require amputation to limit the injury from becoming life-threatening.

Venous insufficiency – Venous insufficiency can develop as a result of systemic vascular disease which leads to valvular dysfunction, venous hypertension, repeated tissue ischemia-reperfusion injury and ultimately skin ulceration (see Chapter 3).

Neuropathy – Neuropathy, brought on by diabetes, infectious diseases or nutritional disturbances, can lead to repeated trauma and ulcerations. Many patients with neuropathy will have poor circulation and will need extensive therapy to allow for adequate wound healing.

Local factors

Local factors that compromise healing can arise from systemic and regional factors as described above – e.g. ischemia, local inflammation, edema and excessive exudate. Local compromising factors also arise as a consequence of a wound too – the breach of skin lets in infecting organisms and foreign bodies.

Ischemia – Arterial and venous insufficiency, as well as prolonged inappropriate externally-applied pressure, can lead to local tissue ischemia that is known to activate inflammation leading to increase of tissue breakdown. Ischemia-reperfusion cycles are also known to be very potent in causing local tissue inflammation and tissue breakdown.¹⁰

Edema and excessive wound exudate – Edema is associated with tissue inflammation and leads to excessive wound exudate which can cause maceration of the wound and surrounding tissue. For chronic wounds, the proteolytic enzymes in the fluid can also cause direct tissue damage to the wound and the surrounding tissue.¹⁷

Infection – Substances such as proteolytic enzymes released from microorganisms can cause direct tissue damage. Indirectly, infection causes elevation of inflammation that is also known to cause tissue breakdown.¹⁸

Foreign bodies – Foreign bodies, particularly in large amounts such as in combat injury, can, in addition to being a recognized risk factor for infection, present a physical barrier to the healing process, delaying the healing process and meaning that tissue heals in a disordered manner.¹⁹

Patient and Wound Assessment

A wound should be assessed at first presentation and regularly thereafter. The frequency of wound assessment will depend on the patient's healthcare setting, and the wound characteristics observed at the previous dressing change. In addition, examples of when a wound should be assessed are:

- if the wound noticeably deteriorates
- if the wound develops an unpleasant odor or exudate
- if significant changes in the wound are observed
- on the patient's return from the operating theatre
- on the patient's return from another facility

Wound classification – acute versus chronic wounds

Wound healing involves a complicated series of events which leads to a 'healed' or 'closed wound', in which:

- connective tissue has been repaired
- re-epithelialization is complete
- anatomical structure and function have substantially returned to normal without the need for continued dressing or drainage

Many wounds are 'acute wounds' that will heal without difficulty in an orderly manner within an anticipated timeframe, but some wounds are resistant to all efforts to heal them. Enoch & Leaper (2007) described a chronic wound as one where *'...the normal process of healing is disrupted at one or more points in the phases of hemostasis, inflammation, proliferation or remodeling, resulting in a delay in healing beyond the anticipated time.'*²⁰

Many factors – systemic, regional and local as described above, all contribute to the wound remaining in a chronic state, and it is important that these contributing factors are diagnosed accurately so that the appropriate treatment regime is given. Any assessment should also include looking at a patient's medical history and lifestyle.

Common chronic wounds include venous leg ulcers, arterial leg ulcers, diabetic foot ulcers (see Chapter 2), pressure ulcers, vasculitis and pyoderma gangrenosum.²¹ Each of these represents different combinations of systemic, regional and local factors that inhibit healing so that the treatment requirements for these chronic wounds can be very different.

Wound measurement

Wound measurement is an important part of the assessment and diagnosis of the wound because it enables clinicians to track changes in the wound over time and thereby direct treatment. Points of measurement include depth, area and circumference.

Clinical appearance

Careful assessment of the wound bed is vital as it gives clues as to how the wound is healing. Healing wounds are characterized by increasing amounts of granulation tissue (Figures 2a and 2b), and later, by epithelialization (Figure 3).²²

Wound bed – Healthy pink granulation tissue indicates healing. If the granulation tissue is dark red, or if it bleeds on contact, it may indicate wound infection. In addition, excess granulation may be down to infection or non-healing wounds.⁴



Figures 2a & 2b. Wound beds filled with healthy granulation tissue



Figure 3. Epithelialization of a wound

Necrotic tissue, slough and eschar – Necrotic tissue, slough (Figure 4) and eschar (Figure 5) can appear across the wound bed, impairing the healing process, and also serve as focal points for microbial colonization leading to infection. Such tissue should be removed.⁴



Figure 4. Wound with slough (dead tissue in the process of separating from viable living tissue)



Figure 5. Wound with eschar (thick, leathery, necrotic, devitalized tissue) across wound bed

Progression of Wound to Healing

Progress to wound healing requires a multi-disciplinary approach. A skin wound, irrespective of whether it is chronic or acute, will present a common set of general problems, such as cleansing, risk of infection and presence of exudate, that require local wound management. Chronic wounds are mostly symptoms of underlying diseases that translate systemic factors into regional and local factors that cause such wounds and prevent their healing, so additional targeting of these systemic/regional/local factors will be required in order to progress these chronic wounds to heal (see Figure 6).

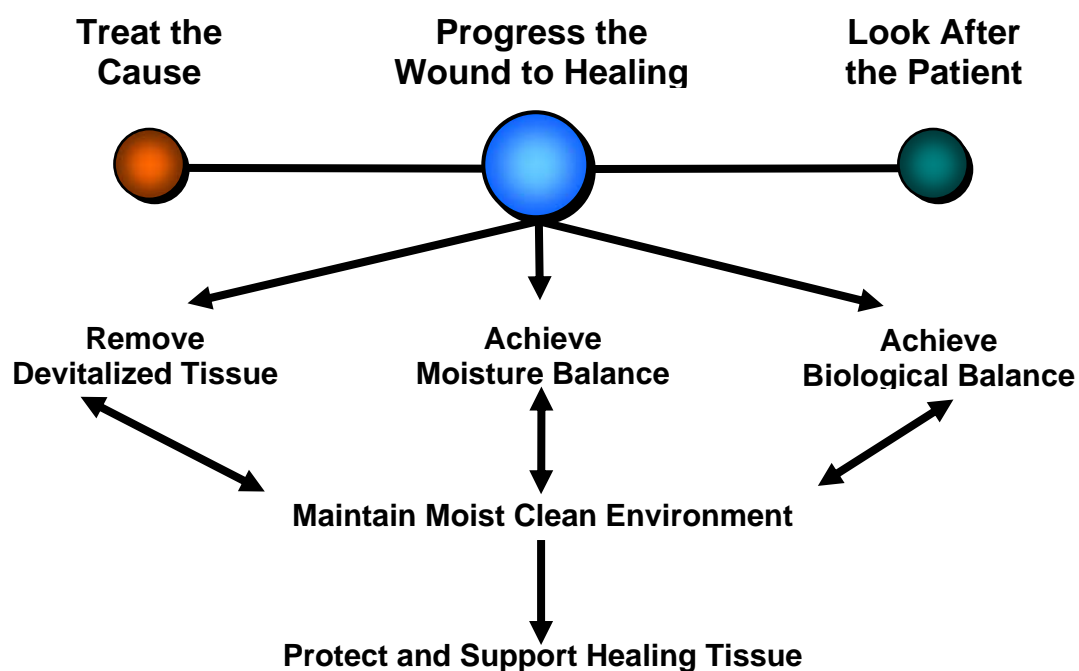


Figure 6. The wound progression model. Progress to wound healing requires a multi-disciplinary approach. Particularly for chronic or recalcitrant wounds, in addition to local wound management, those systemic, regional and local factors that cause healing problems will need to be addressed in order to progress healing.

Wound closure: primary- versus secondary-intention healing

This relates to how the wound is healed – whether the wound edges are physically brought together, or whether the wound is allowed to fill naturally. For more information see Enoch & Leaper (2007).²⁰

Primary healing (healing by first intention)

Primary healing is when the edges of the wound are brought together by suture, tape, staples, tissue glue or some mechanical devices.

Most wounds managed by primary intention are treated within 12-24 hours. Examples are clean surgical incisions and clean lacerations. The incision causes the death of only relatively few epithelial and underlying connective tissue cells, so epithelial regeneration predominates over fibrosis. A good balance between the healing phases means that wounds heal well and quickly.

There are occasions when primary healing has to be delayed – a procedure known as delayed primary healing. Candidates for delayed primary healing have mostly contaminated or poorly delineated wounds, e.g. bites and abdominal wounds after peritoneal soiling. They are intentionally left open for a few days to prevent infection, and wound edges are only approximated once the wound is clean of debris. After a few days phagocytic cells are recruited to the wound and inflammatory cells destroy contaminating bacteria.

Secondary healing (healing by second intention)

Secondary healing occurs when much of the soft tissue within the wounded area has been lost, for example, following severe trauma, burns and some surgical procedures. These wounds are healed mostly by tissue re-growth. Fibroblasts and blood vessels grow from the wound margin and from the underlying fatty layer to form the granulation tissue, and epithelial cells grow from the wound margin and from hair follicles to form the new epidermis. Wound closure is also facilitated by wound contraction – from contractile forces generated by fibroblasts in the granulation tissue.

On occasions where the capacity for tissue re-growth is not sufficient to close the wound in a timely manner, for example large burns, then grafting may be required.

Wound management approaches

A comprehensive review of good wound management approaches can be found in Schultz et al (2003).²³ Here is a summary of the general principles.

Wound exploration, wound cleansing and debridement

If a wound is contaminated it needs to be prepared before it is treated. A wound should be clinically explored to detect any associated injuries to the underlying neurovascular structures, tendons, joints and bones, and to locate and remove foreign bodies.

Wound cleansing refers to the cleansing of the wound with water, saline or antiseptics. Since bacteria rapidly multiply, this should be carried out at the first available opportunity, using a soft brush or sponge to clear away debris and up to several liters of isotonic fluid.

Debridement is a process to remove debris and non-viable tissue and can be achieved by surgical and non-surgical means.

Managing and treating infection

Infection control is vital to the wound management process: often more so with chronic than with acute wounds by virtue of the more long-standing nature of chronic wounds.

Infections of wounds are often polymicrobial. Where possible, clinically infected wounds should be cultured and microorganism sensitivities determined before systemic antimicrobial agents are prescribed. Elderly patients may have sub-optimal inflammatory responses, so signs of wound infection can be subtle. The same can be true for mycobacterial and fungal infections.

A number of antiseptic agents are indicated for managing the risk of infection. These include various forms of iodine and silver. For more heavily-colonized or infected wounds, the use of antibiotics may be required.

Although antibiotic use is controversial in clean wounds, it may be warranted when:

- the risk of infection is high
- there are other risk factors such as poorly controlled diabetes or morbid obesity
- the risk of infection for a particular procedure is high
- there are potentially disastrous consequences of an infection such as those involved in implants, incised bone leading to a cavity and prosthetic heart valves

Protection and management of exudate – use of dressings

Different types of wounds may require the use of dressings of different characteristics. For a comprehensive overview, see Fonder *et al* (2008).²¹

Gauzes are used for packing deep wounds. They are inexpensive but they dry out and need to be changed regularly, otherwise they may leave residue in the wound and cause damage on removal.

Films are used for wounds with minimal exudate and as secondary dressings. They are moisture-retentive and protect the wound from contamination but they can trap fluid and strip the skin.

Hydrogels are used on dry and painful wounds. They are moisture-retentive and easy to remove. Whilst they may over-hydrate, they also help with pain relief.

Hydrocolloids are appropriate for wounds with a light to moderate exudate. They are absorbent, occlusive and protective but can trap fluid and strip skin.

Alginates and Hydrofiber® dressings are used on wounds with moderate to heavy exudate, are highly absorbent and can be left on until soaked with exudate.

Foams are absorbent and provide thermal insulation for wounds with light to moderate exudate.

The extra challenges of managing chronic wounds

Most acute wounds will heal without complication if the general approach to good wound management is followed. Chronic wounds however present additional challenges because they are mostly symptoms of underlying diseases. So, in addition to the general principles of good wound management, aspects of the underlying diseases need to be targeted in order to bring about wound closure.

When a chronic wound is presented, it is first of all vital to make an accurate diagnosis of what type of chronic wound it is, so that a general approach to treating that type of wound can be decided upon. For example, in addition to good general wound care, venous leg ulcers will require compression, and diabetic foot ulcers, off-loading (taking pressure off the foot) and diabetes control. You will come across more details of these in relation to venous leg ulcers in the subsequent chapters.

Besides the general diagnosis, it is also important to look at the patient's health status and history, record all medications they might be taking and examine their nutritional intake and social history, as these may allow additional complicating factors to be identified and appropriate treatment/management to be given.

Summary

- The skin is made up of three main layers, the epidermis, the dermis and the hypodermis (subcutaneous tissue), each of which is responsible for a variety of functions, from protection and immunity, to regulating body temperature and perception of stimuli
- When the skin is breached, it heals itself in four overlapping 'phases' – hemostasis and inflammation, proliferation and granulation, re-epithelialization and maturation and remodeling
- Wound healing is dominated by cellular activity, collagen production and new tissue formation
- A moist environment can be beneficial to the wound healing process and has been found to reduce pain and healing time in comparison to wounds covered with dry dressings
- Various local, regional and systemic factors can delay and disrupt wound healing
- Whilst acute wounds will eventually heal, chronic wounds are resistant to healing without specialist and continued management and care tailored to the wound type and underlying pathology
- It is important to diagnose the type of chronic wound correctly in order that specialist management and care can be tailored to the wound type
- A wound can be assessed through its measurement and clinical appearance. A wound that is healing healthily is filled with pink granulation tissue
- Local wound management involves infection control, wound debridement and the use of a choice of wound dressings
- Primary healing refers to a wound which is closed by bringing the wound edges together using sutures, staples, tape, glue or mechanical devices. Secondary healing refers to a wound which is filled and covered by tissue growth

Chapter 1

Self Assessment Questions

Please circle the letter(s) to indicate the correct answer(s)

1. The following statements are true, except:

- A. The skin is made up of three layers: the dermis, the epidermis and the hypodermis
- B. In normal skin it takes 7 days from cell formation to shedding of cells
- C. Sweat production is reduced in response to a low environment temperature
- D. Skin appendages include hair follicles, sweat glands and sebaceous glands

2. As a wound heals, the following all happen, except:

- A. A blood clot forms at the site of injury to prevent hemorrhage and to regain hemostasis
- B. Inflammation protects the wound from infection, and acts as a bridge between tissue injury and new cell growth
- C. Fibroblasts migrate to the wound site and granulation tissue is formed
- D. Skin connective tissue eventually reverts to its original organization, function and strength

3. Which of the following statements is false? Moist wound healing is beneficial for the following reasons:

- A. Prevention of tissue dehydration
- B. Reduced angiogenesis
- C. Reduction in pain
- D. Reduced healing time

4. Which of the following can disrupt wound healing?

- A. Inappropriately applied compression
- B. Inadequate nutrition
- C. Metabolic diseases
- D. Venous insufficiency
- E. All of the above
- F. None of the above

5. Which of the following statements is false?

- A. Healing wounds are characterized by increasing amounts of granulation tissue, and later, by epithelialization
- B. Healthy granulation tissue is dark red
- C. Hydrogels can be used on dry and painful wounds
- D. In secondary healing a wound is filled and covered by tissue growth

Chapter 1

Self Assessment Answer Key

1. **B** – It takes 28 days from cell formation to shedding of cells
2. **D** – Even after maturation skin connective tissue does not regain its original organization, function or strength
3. **B** – Moist wound healing is beneficial due to accelerated angiogenesis
4. **E**
5. **B** – Healthy granulation tissue is pink

References

1. Tortora GJ, Grabowski SR. The Integumentary System. In: *Principles of Anatomy and Physiology* (7th edition). New York, NY: John Wiley and Sons; 1993:126-139.
2. Quinn AG. Biology of the Skin and Dermatological Disease. *Medicine*. 2004; **32(12)**:1-3.
3. Powell J. Skin Physiology. *Acute Care: The Foundation Years*. 2007; **3(5)**:193-196.
4. Grey J E, Enoch S, Harding KG. ABC of Wound Healing - Wound Assessment. *BMJ*. 2006; **332**:285-288.
5. Chettibi S, Ferguson MWJ. Wound Repair: An Overview. In: Gallin JI, Snyderman R, eds. *Inflammation: Basic Principles and Clinical Correlates* (3rd Edition). Philadelphia, PA: Lippincott, Williams and Wilkins; 1999:865-881.
6. Broughton G, Janis JE, Attinger CE. A Brief History of Wound Care. *Plast Reconstr Surg*. 2006; **117**:6S-11S.
7. Winter GD. Formation of the Scab and the Rate of Epithelialization of Superficial Wounds in the Skin of the Young Domestic Pig. *Nature*. 1962; **193**:293-294.
8. Field CK, Kerstein MD. Overview of Wound Healing in a Moist Environment. A Symposium: Wound Infection and Occlusion – Separating Fact from Fiction. *Am J Surg*. 1994; **167(1A)**:2S-6S.
9. Hutchinson JJ, Lawrence JC. Wound Infection Under Occlusive Dressings. *Journal of Hospital Infection*. 1991; **17**:83-94.
10. Chen WYJ, Rogers AA. Recent Insights into the Causes of Chronic Wound Ulceration in Venous Diseases and Implications on Other Types of Chronic Wounds. *Wound Rep Reg*. 2007; **15**:434-449.
11. Hoffman MD. Inflammatory Ulcers. *Clinics in Dermatology*. 2007; **25**:131-138.
12. Schaper NC, Nabuurs-Franssen MH, Huijberts MSP. Peripheral Vascular Disease and Type 2 Diabetes Mellitus. *Diabetes Metab Res Rev*. 2000; **16(1)**:S11-S15.
13. Ashcroft GS, Mills SJ, Ashworth JJ. Ageing and Wound Healing. *Biogerontology*. 2002; **3**:337-345.
14. Mandal A. Do Malnutrition and Nutritional Supplementation have an Effect on the Wound Healing Process? *J of Wound Care*. 2006; **15(6)**:254-257.
15. Payne WG, Naidu DK, Wheeler CK et al. Wound Healing in Patients with Cancer. *Open Access Journal of Plastic Surgery*. 2008; **8**:68-90.
16. Freiman A, Bird G, Metelitsa AI, Barankin B, Lauzon GJ. Cutaneous Effects of Smoking. *J Cutan Med Surg*. 2004; **8(6)**:415-423.
17. Bishop SM, Walker M, Rogers AA, Chen WYJ. Importance of Moisture Balance at the Wound-dressing Interface. *J of Wound Care*. 2003; **12(4)**:125-8.
18. Mims CA. Mechanisms of Cell and Tissue Damage. In: *The Pathogenesis of Infectious Diseases*. London, UK: Academic Press; 1987:179-225.
19. Sakorafas GH, Peros G. Principles of War Surgery: Current Concepts and Future Perspectives. *Am J of Emergency Medicine*. 2008; **26**:480-489.

20. Enoch S, Leaper DJ. Basic Science of Wound Healing. *Surgery*. 2007; **26(2)**:31–37.
21. Fonder MA, Lazarus GA, Cowan DA, Aronson-Cook B, Kohli AR, Mamelak AJ. Treating the Chronic Wound: A Practical Approach to Nonhealing Wounds and Wound Care Dressings. *J Am Acad Dermatol*. 2008; **58(2)**:185–206.
22. Doughty DB. Clinical Management Extra. Wound Assessment: Tips and Techniques. *Adv Skin Wound Care*. 2004; **17(7)**:369–372.
23. Schultz GS, Sibbald RG, Falanga V et al. Wound Bed Preparation: A Systematic Approach to Wound Management. *Wound Repair Regen*. 2003; **11(2)**, S1-S28.

Hydrofiber is a trademark of ConvaTec Inc.

Chapter 2

The Impact of Leg Ulcers

This chapter describes the most commonly occurring types of leg ulcers, that is, venous, arterial, and neuropathic leg ulcers and whom they affect within the population. It goes on to describe the specifics of ulcer diagnosis and the impact that leg ulcers have on the patient and the health system.

Chapter 1 discussed the structure and function of the skin and gave you an overview of wound healing and assessment.

Topics to be discussed in this chapter include:

- Leg ulcers
 - Venous leg ulcers
 - Arterial leg ulcers
 - Neuropathic ulcers (diabetic foot ulcers)
- Epidemiology of leg ulcers
 - How many people leg ulcers affect
 - Who leg ulcers affect
- Diagnosis of leg ulcers
 - Differential diagnosis
 - The differences between arterial and venous leg ulcers
- The impact of venous leg ulcers
 - What impact do venous leg ulcers have on patients?
 - What is the impact of venous leg ulcers on health service resources?

Upon completion of Chapter 2, you will:

- understand the social, economic, and psychological burden of leg ulcers with particular reference to venous leg ulcers
- know the difference between arterial and venous leg ulcers
- recognize the key clinical features that distinguish between venous and arterial leg ulcers.

Leg Ulcers

A leg ulcer, also referred to as leg ulceration, is a chronic leg wound that fails to heal.¹ The most common types of ulcer that occur on the leg or foot are:²

- venous leg ulcers
- arterial leg ulcers
- diabetic foot ulcers
- ulcers with other underlying causes.

Most leg ulcers are caused by problems in the veins (venous leg ulcers), but many have mixed or multifactorial causes.^{2, 3} About 5% of ulcers occur in patients with diabetes.⁴

Venous leg ulcers

Venous leg ulcers result from chronic venous insufficiency (CVI) - see Chapter 3, and occur mainly on the leg between the ankle and the knee.⁵ They require long-term care² and recurrence rates are as high as 59–67%.³

Arterial leg ulcers

Arterial leg ulcers are caused by reduced arterial blood supply to the legs and feet. The most common cause of this is atherosclerosis,⁶ which is a hardening and narrowing of the arteries that then restricts blood flow. Arterial ulcers are usually found on the toes, heels, and bony prominences of the foot.⁶

Diabetic foot ulcers

Patients with diabetes, particularly poorly controlled disease, are at risk of developing diabetic foot ulcers, resulting from vascular compromise or neuropathy. Diabetic foot ulcers can affect as many as 15% of people with diabetes.⁷

- **Neuropathic ulcers** – These ulcers usually occur on the sole of the foot particularly at pressure points such as the heel or base of the toes (metatarsal heads) because the patient has lost the protective pain sensation in their foot through nerve damage. The ulcer may be caused by repetitive rubbing of poorly fitting footwear, foreign bodies in the footwear, or standing on objects when walking barefoot.⁸
- **Ischemic ulcers** – These ulcers occur in feet with ischemia. If neuropathy is also present, they are known as neuroischemic ulcers and occur most often on the edges of the foot, especially on the medial surface of the first metatarsophalangeal joint and over the lateral aspect of the fifth metatarsophalangeal joint. They can also develop on the tips of the toes and, if the toe nails become thick, then ulcers can also form under the nails.⁸

Ulcers with other underlying causes

Although the types of leg ulcers above are the most commonly occurring, there are also other underlying causes that result in a variety of other leg ulcers. Some of these other causes include:³

- rheumatological disorders (e.g. rheumatoid arthritis)
- hematological disorders (e.g. sickle cell anemia, thalassemia)
- pressure sores
- traumatic ulcers
- dermatological disorders (e.g. malignancy)
- infections.

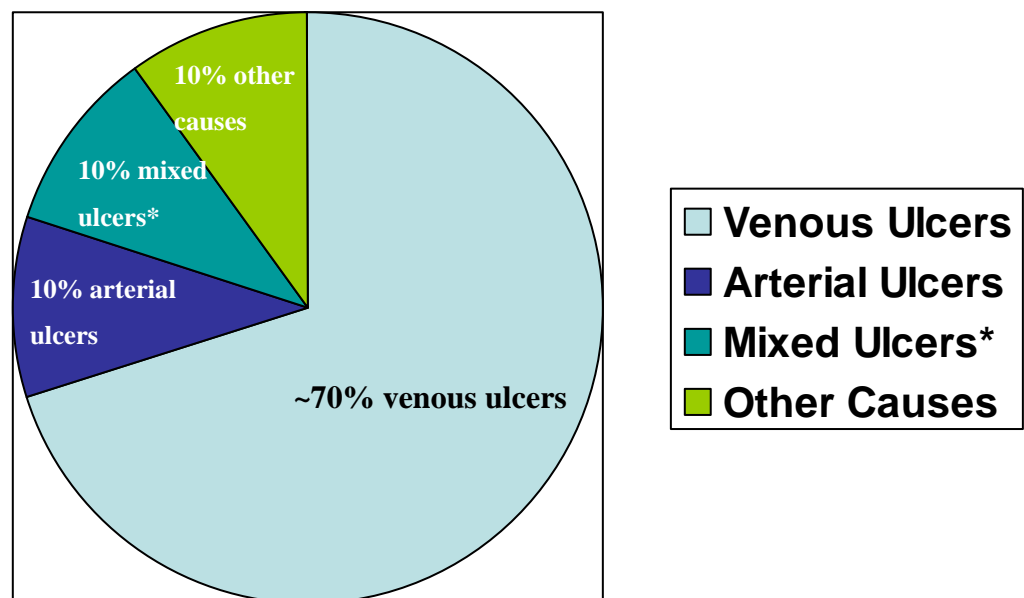
Epidemiology of Leg Ulcers

How many people do leg ulcers affect?

At any one point in time, 0.1–0.2% of the population have an open, active ulcer of some type (venous, arterial, or diabetic foot ulcer). Overall, 1–2% of the population will suffer a chronic leg ulcer with the prevalence increasing with increasing age.³

The distribution of ulcer cause (etiology) is shown in Figure 1.⁹

Figure 1. Ulcer etiology



*Arterial and venous etiology

Note: Prevalence/incidence data for ulcer etiology vary by source, estimates of ulcers of venous etiology range as high as 80% to 85%.¹⁰

Who do leg ulcers affect?

Leg ulcers are often considered to affect primarily elderly people. However, one of the largest published population studies of leg ulcer patients found that over a third of patients experienced their first ulcer before the age of 50. The chronic nature of the condition does mean that the age distribution is heavily weighted towards the elderly, and with an ageing population, this trend is likely to continue.⁴

Diagnosis of Leg Ulcers

Differential diagnosis

Ulcers are usually diagnosed clinically by using differential diagnosis, which involves systematically excluding all other causes of leg ulceration by taking a thorough patient history and closely examining the ulcer and the affected leg. Each patient should also be assessed for concomitant arterial disease as this has implications for subsequent treatment.²

Differentiating between arterial and venous leg ulcers

Venous leg ulcers can be distinguished from arterial leg ulcers by their location, appearance, and their effect on the skin surrounding the wound (see Table 1).²

Table 1. Features of arterial and venous ulcers

	Arterial	Venous
Location	Toes, heels, and bony prominences of the foot ⁶	Gaiter region – medial malleolus ^{5,6,11}
Edema	Not common ⁶	Pitting or non-pitting ⁶
Depth	Deep, 'punched out' appearance ^{2,5,6}	Shallow ^{2,11}
Margins	Sharply defined ⁶	Irregular ²
Wound bed	Pale, necrosis or gangrene may be evident ^{2,6}	Deep red with yellow fibrin ⁶
Skin features	Blanched skin Shiny and taut around ulcer ²	Hyperpigmentation ⁶
Exudate	Minimal ⁶	Moderate to heavy ⁶
Skin temperature	Cool ^{2,6}	Normal or slightly warm ⁶
Pain	Aggravated by walking and exercise ^{2,6}	Can be associated with edema ⁶

Arterial leg ulcers

Arterial leg ulcers are commonly associated with poor arterial blood flow largely owing to constriction or blockage of the arteries in the leg and foot, the most common cause of which is atherosclerosis (hardening and narrowing of the arteries).^{6, 12} Tissues located distal to the blockage (tissues located away from the centre of the body) can become ischemic, leading to tissue damage and ulceration. Gangrene is a potential complication and amputation is quite common. Patients may complain of intermittent claudication (leg pain when walking) or, in severe cases, leg pain even when at rest.²

Arterial leg ulcers (Figure 2) are usually found between the toes or on the tips of the toes, over phalangeal heads (toe joints), around lateral malleolus, or on areas exposed to repetitive trauma, such as rubbing footwear.¹²



Figure 2. Arterial leg ulcer

Arterial ulcers have a 'punched out' appearance with well-defined edges and minimal levels of exudate. The wound base is generally pale or necrotic with little, if any, granulation. Gangrene, necrosis, or eschar (scabbing tissue) are common, although signs of inflammation may be subtle.¹²

Photograph courtesy of Professor Keith Harding, Cardiff University

The periwound skin (skin surrounding the wound) usually shows signs of erythema with possible hardening. Levels of edema surrounding the wound can vary, and the skin around the wound is usually cool to the touch, even in warm environments. Blanched or purpuric (small purples spots or patches) periwound tissue is a common sign that accompanies arterial leg ulceration.²

Examination of the arterial system may show a decreased or absent pulse in the dorsalis pedis and posterior tibial arteries (main arteries supplying the foot). Patients with arterial ulcers also have a reduced capillary refill time. After compression of the big toe or dorsum of the foot (upper surface of the foot; as opposed to the sole of the foot) for a few seconds, the skin color should return to normal in less than two to three seconds with normal capillary refill.⁶ Delay in the return of the normal color indicates vascular compromise. The ankle brachial pressure index or ABPI (sometimes referred to as the ankle brachial index or ABI) is helpful in identifying peripheral vascular disease, except if vessel calcification (for example, as is found in diabetes) or edema is present.⁶

ABPI

The ABPI is a non-invasive procedure that measures the level of arterial blood supply to the foot and as such is used routinely to identify the presence of peripheral vascular disease (arterial disease in the legs). It measures the difference in systolic blood pressure in the arm (brachial pressure) and the systolic blood pressure in the foot (ankle pressure) using a Doppler. The ABPI is then calculated by using the following equation:¹³

$$ABPI_l = \frac{P_l}{P_a}$$

$ABPI_l$ = ABPI for a leg

P_l = Highest pressure obtained from the ankle vessels for that leg

P_a = Highest brachial pressure of the two arms

In a normal subject the pressure at the ankle pulses is slightly higher than at the elbow so an ABPI of 1–1.1 is considered normal, whereas an ABPI of 0.5 or less indicates severe arterial disease (see Table 2 for further details).^{2,6}

Table 2. Explanation of the ABPI⁶

	Mild leg pain when walking or no symptoms	
	Varying degrees of intermittent claudication	Mild to moderate arterial disease
	Severe intermittent claudication and pain at rest	
	Pain at rest for more than two weeks with or without tissue loss	

Table created using data from Grey *et al.*⁶

Note: ABPIs may be problematic (give false readings or are difficult to measure) in patients with vessel calcification (for example in diabetic patients) or edema, thus alternative assessments may need to be performed to determine the adequacy of circulation

Venous leg ulcers

Venous leg ulcers generally occur in the 'gaiter' area (see Figure 3). This area lies between the ankle and the knee, with the medial and lateral malleoli (the bony part of the ankle that protrudes slightly) being the most common sites of ulcer formation.⁴

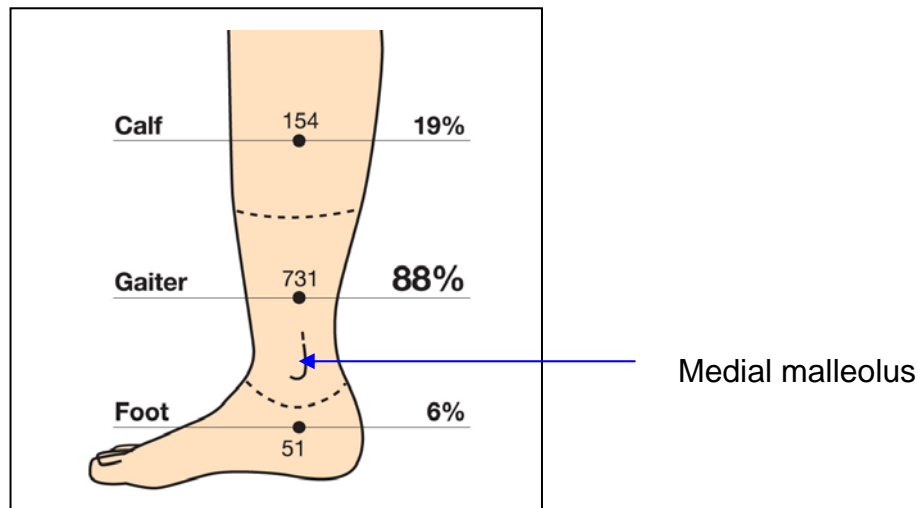


Figure 3. The majority of venous ulcers appear on the gaiter of the lower leg, above or just below the ankle. There may be more than one ulcer present. Adapted from Callam *et al.*⁴ (Amended with permission from the BMJ Publishing Group)



Figure 4. A venous leg ulcer

In the case of venous leg ulcers, the base of the ulcer is usually deep red, with granulation tissue and/or yellow fibrin being present. Calcification of the wound base is common, and the wound margins tend to be large and irregular with moderate to heavy levels of exudate (Figure 4).¹²

Photograph courtesy of Hugo Partsch, MD, Medical University of Vienna



Figure 5. Edema

One of the most characteristic features of venous leg ulcers is the presence of lower-leg edema (Figure 5). Edema can be either pitting or non-pitting with possible hardening of the skin and cellulitis (inflammation of the subcutaneous tissue).⁶

Photograph courtesy of Hugo Partsch, MD, Medical University of Vienna



Figure 6. Hyperpigmentation

Venous ulcers are often accompanied by hyperpigmentation (discoloration) of the skin caused by the deposition of hemosiderin¹⁴ – a breakdown product of extravasated red blood cells (see Figure 6).

Photograph courtesy of Hugo Partsch, MD, Medical University of Vienna

The skin surrounding the ulcer can often show signs of erythema (reddening of the skin) and venous or 'stasis' dermatitis.



Figure 7. Lipodermatosclerosis

Lipodermatosclerosis is common and is characterized by a painful hardening of the skin with the lack of pitting edema (see Figure7).⁶

Photograph courtesy of Hugo Partsch, MD, Medical University of Vienna



Figure 8. Champagne-bottle deformity of the leg

In the later stages, chronic lipodermatosclerosis may alter the shape of the leg, making it look like an inverted champagne bottle¹⁵ (see Figure 8). Lipodermatosclerosis may also lead to a loss of sweat glands and hair follicles as atrophy of the skin progresses.⁶ Skin temperature is normal, and skin remains warm to touch.⁶

Photograph courtesy of Professor Christine Moffatt CBE, Centre for Research & Implementation of Clinical Practice, London

Mixed arterial and venous leg ulcers

Mixed leg ulcers are more difficult to diagnose than venous or arterial leg ulcers. They are caused by a combination of venous and arterial problems, and therefore have symptoms of both venous and arterial leg ulcers (Figure 9).⁶ Patients with a mixed leg ulcer fall into one of two categories:

- Patients with venous disease and an ABPI 0.5–0.8 (moderate arterial disease)
- Patients with venous disease and an ABPI < 0.5 (advanced arterial disease)

Mixed ulcers can prove challenging to treat as the main treatment recommended for venous leg ulcers is sustained graduated compression (SGC) therapy at high pressure (see chapter 4 for further details). This, however, proves problematic in the case of mixed ulcers as SGC therapy is contraindicated in the presence of arterial disease⁶ as compression can impede arterial inflow even further. Low levels of compression, however, can be used safely in patients with an ABPI of 0.5–0.8 to help manage the venous component of their ulcer.^{16, 17} The arterial status of these patients can change rapidly, so a sudden increase in pain or an inability to tolerate compression may be a sign that the patients' arterial disease has advanced.¹⁸ Patients with an ABPI of less than 0.5 have advanced arterial disease and SGC is therefore not recommended in these cases.¹⁸



Figure 9. Mixed arterial and venous leg ulcer

The Impact of Venous Leg Ulcers

What impact do leg ulcers have on patients?

Chronic leg ulceration can be a debilitating condition¹⁹ and can substantially reduce many aspects of a patients' quality of life.^{20,21,22} Overall, the prognosis of venous leg ulcers is poor. Venous leg ulcers typically follow a pattern of remission and recurrence, with recurrence rates of up to 59–67%.³ The psychological impact of leg ulceration is considerable.²⁰

Quality of life

Chronic venous disease (CVD) and the more severe CVI have a major impact on a patients' quality of life.²³ Patients live with a variety of symptoms including aching, heaviness, swelling, cramps, itching, tingling, and restless legs.²⁴ Additionally, the clinical manifestations of CVD range from telangiectasia (spider veins) and varicose veins to active ulceration. The incidence of symptoms increases with increasing disease severity.²⁴ Table 3 summarizes some of the impacts that a venous leg ulcer has on general patient quality of life.

Table 3. Impact of leg ulceration on aspects of a patients' quality of life²¹

Activity	Psychological	Symptom distress
Activities outside the home restricted Hard to do regular activities (e.g., work) Do not go out Activities in the home restricted Difficult to move around Dependent on other people Cannot do things I enjoy	Feel embarrassed Feel self-conscious Feel angry Lack of self-confidence	Ulcer burns/stings Ulcer hurts Skin irritated Ulcer itches Skin sensitive Do not sleep well Difficult to relax

Table created using data from Hareendran *et al.*²¹

An emotional and financial burden

The swelling or edema that is associated with venous leg ulceration is a major predictor of impaired mobility, negative financial impact, impaired working capacity, and emotional impact.²⁰ The financial impact alone can be substantial. Loss of employment, time lost from work, and medication and dressings expenses all contribute to the financial burden and can result in anger and frustration.²⁰ A quality-of-life study with respect to patients with chronic venous ulcers published in 2005 found that not having a job had an impact on a patient's quality of life to an extent similar to that of pain.²⁵ The feeling of social isolation also had an impact on patients in this study. This isolated feeling was especially seen with patients who encountered difficulties at work as a result of their leg ulcer. These employment-related problems also had a subsequent economic impact.²⁵

Pain, odor, and exudate leakage

A study published in 2001 described the feelings and experiences of patients living with a chronic venous leg ulcer.¹⁹ Despite the common assumption that venous ulcers are not painful,² pain was an overwhelming feature that had a profound effect on patients' lives. Analgesia was frequently inadequate and patients were reported as saying that no one really understood their pain. Pain was also the cause of many sleepless nights and as the treatment itself can be painful, it was frequently quoted as a reason for non-compliance to treatment.¹⁹

A 2007 study investigating pain and patients' quality of life found that only 23% of patients were prescribed pain relief despite World Health Organization recommendations. This study also found that pain was strongly correlated with both the size and the duration of the ulcer, with patients with the longest ulcer duration reporting the highest pain scores.²⁶

Excess exudate leakage and odor were also a big issue for patients, with many patients expressing horror and dismay at these effects. Leakage was deemed unacceptable and patients found the effects of odor to be particularly distressing.¹⁹

"[I] used to try all sorts but in the end wore Wellington boots at work, anything to hide the leakage"

"The odor has been unbearable. I used to go to church but the person next to me could smell my leg, so I don't go now"

Another recurring theme from the study was personal hygiene. Participants reported that "they never felt clean" and that "they always smelt", which negatively impacted on their sense of wellbeing.¹⁹

"It's just so disgusting. I feel so dirty. [I] don't want anyone to see those awful bandages"

Many of the participants in the study were pessimistic when it came to talking about whether their ulcer would heal or not, regardless of whether they had previously experienced ulcer recurrence. Feelings of guilt, disappointment, and sadness at the persistence or recurrence of their ulcer were common (Box 1).¹⁹

Box 1. Emotions reported by patients with leg ulceration.²⁰

Lethargy
Anger/resentment
Worry
Depression
Fear
Social isolation

What is the impact of leg ulcers on health-service resources?

Leg ulcers are not only expensive to treat, but they also use a substantial amount of resources, particularly the time of the staff who perform the dressing changes.²⁷ The average cost of treating a first venous leg ulcer patient over the period of a year (data collected in the period of 1993–1997) varied between €1332–2585 (US \$2197.80–3489.75*) in Sweden and €814–1994 (US \$1098.90–2691.90*) in the UK. The most expensive venous ulcers to treat are first venous leg ulcers that are larger than 10 cm² and older than 6 months at diagnosis.²⁷ These difficult-to-heal ulcers can cost up to €181 (US \$244.35*) per week (see Table 4).²⁸ Unhealed ulcers are also expensive to treat, costing the UK National Health Service (NHS) £1067 (US \$1835.24*) per unhealed ulcer per year.²⁹

Table 4. Costs associated with difficult-to-heal ulcers²⁸

Total treatment time (months)	38 months
Dressing changes Frequency Time per dressing change†	3.8 per week 40 minutes
Weekly cost Staff costs – treatment time – travel time Wound-care-product costs – dressing products – compression Travel costs – for staff or patients Total weekly cost of dressing changes In-patient care cost‡	 €57.30 (US \$77.36*) €28.60 (US \$38.61*) €21.50 (US \$29.01*) €5.80 (US \$7.83*) €3.10 (US \$4.19*) €116.30 (US \$157.01*) €65.00 (US \$87.75*)
Total weekly cost	€181.30 (US \$244.76*)

Data created using data from Tennvall, Hjelmgren & Öien.²⁸

*Exchange rate based on January 20, 2009. 1 Euro=US\$1.35, 1 GBP=US \$1.72.

†Including time for arrangements before and after dressing changes. ‡Equivalent to 'hotel costs' in specialist clinic or other institutional care.

Overall, venous leg ulcers alone are thought to cost the UK NHS £400 million a year (US \$720 million; €600 million),³⁰ while in the US the annual cost has been estimated to be US \$1 billion.³¹

Summary

- The most commonly occurring types of leg ulcer are venous, arterial, diabetic and ulcers with other underlying causes
- Venous leg ulcers are the most common type of leg ulcer
- Venous leg ulcers can be clinically distinguished from arterial leg ulcers by their location, appearance, and underlying cause
- Patients with venous leg ulcers must always be examined for signs of concomitant arterial disease, the presence of which has significant impact on possible treatment options
- Venous leg ulcers are a psychological, emotional, and financial burden and can be debilitating for patients

Chapter 2

Self Assessment Questions

Please circle the letter(s) to indicate the correct answer(s)

1. Which of the following statements is FALSE?

- A. The majority of leg ulcers are venous, but many have mixed or multifactorial causes
- B. Contributing factors to the overall burden of venous ulcers include the impact on work productivity and impaired mobility
- C. Atherosclerosis is a common cause of venous leg ulcers
- D. Venous leg ulcers are more common with increasing age

2. Which of the following are not reported in relation to venous leg ulcers?

- A. Very little pain
- B. Significant impact on quality of life
- C. Common ulcer recurrence
- D. Odor and copious exudate from the wound

3. Which of the following is FALSE with respect to venous leg ulcers?

- A. There is very little exudate
- B. The base of the ulcer may be deep red with yellow fibrin slough
- C. The gaiter area lies between the ankle and the knee
- D. Chronic lipodermatosclerosis may result in the leg taking on a 'champagne-bottle' form
- E. Calcification of the wound base is common

4. Which of these is NOT a feature of arterial leg ulcers?

- A. The majority are located on the toes, heels, and bony prominences of the foot
- B. The periwound tissue is usually white
- C. Gangrene is a complication that often results in amputation
- D. Characteristically has shiny and taut skin around the edges of the wound
- E. Edema is quite uncommon

5. Which of the following statements is incorrect with respect to the various impacts of venous leg ulcers?
- A. Pain associated with venous ulcers has a huge impact on patients lives
 - B. Venous leg ulcers typically do not recur once they have healed
 - C. Loss of and inability adequately to perform employment not only has a financial, but also an emotional impact
 - D. Pain is strongly correlated with size and duration of ulcer
 - E. The overall emotional impact is huge
6. Look at the pictures below and identify which one is a typical venous leg ulcer and which one is an arterial leg ulcer:

A.



B.



Chapter 2

Self Assessment Answer Key

1. **C** – atherosclerosis is the most common cause of reduced **arterial** blood supply to the legs. It is therefore a common cause of arterial leg ulcers.
2. **A** – contrary to popular belief, pain is actually a common feature of venous as well as arterial ulcers
3. **A** – venous leg ulcers tend to have moderate to heavy levels of exudate
4. **B** – The periwound skin usually shows erythema and is blanched or purpuric
5. **B** – Venous leg ulcers have recurrence rates of up to 59–67%
6. **A** – arterial leg ulcer
B – venous leg ulcer

References

1. Moffatt CJ, Franks PJ, Doherty DC, Martin R, Blewett R, Ross F. Prevalence of Leg Ulceration in a London Population. *Q J Med*. 2004;**97**:431–437.
2. Valencia IC, Falabella A, Kirsner RS, Eaglstein WH. Chronic Venous Insufficiency and Venous Leg Ulceration. *J Am Acad Dermatol*. 2001;**44**:401–421.
3. Briggs M, Closs SJ. The Prevalence of Leg Ulceration: A Review of the Literature. *EWMA Journal*. 2003;**3**(2):14–20.
4. Callam MJ, Harper DR, Dale JJ, Ruckley CV. Chronic Ulcer of the Leg: Clinical History. *BMJ*. 1987;**294**:1389–1391.
5. Grey JE, Enoch S, Harding KG. ABC of Wound Healing - Wound Assessment. *BMJ*. 2006;**332**:285-288.
6. Grey JE, Enoch S, Harding KG. ABC of Wound Healing - Venous and Arterial Leg Ulcers. *BMJ*. 2006;**332**:347–350.
7. Boulton AJM. The Diabetic Foot: A Global View. *Diabetes Metab Res Rev*. 2000;**16**(Suppl 1):S2–S5.
8. Edmonds ME, Foster AVM. ABC of Wound Healing - Diabetic Foot Ulcers. *BMJ*. 2006;**332**:407-410.
9. Dissemmond J, Körber A, Grabbe S. Differential Diagnosis of Leg Ulcers. *J Dtsch Dermatol Ges*. 2006;**8**:627-634.
10. Simon DA, Dix FP, McCollum CN. Management of Venous Leg Ulcers. *BMJ*. 2004;**328**:1358–1362.
11. Royal College of Nursing. *Clinical Practice Guidelines. The Nursing Management of Patients with Venous Leg Ulcers. Recommendations* 2006.
http://www.rcn.org.uk/__data/assets/pdf_file/0003/107940/003020.pdf.
Accessed January 23, 2009.
12. Phillips TJ, Dover JS. Leg Ulcers. *J Am Acad Dermatol*. 1991;**25**(6):965–990.
13. Vowden P, Vowden K. Doppler Assessment and ABPI: Interpretation in the Management of Leg Ulceration. *World Wide Wounds*.
<http://www.worldwidewounds.com/2001/march/Vowden/Doppler-assessment-and-ABPI.html>.
March 2001. Accessed January 23, 2009.
14. Nicolaides AN. Chronic Venous Disease and the Leukocyte-Endothelium Interaction: From Symptoms to Ulceration. *Angiology*. 2005; **56**: S11-S19.
15. Etufugh CN, Phillips TJ. Venous Ulcers. *Clinics in Dermatology*. 2007;**25**:121–130.
16. Marston W, Vowden K. Compression Therapy: A Guide to Safe Practice. In: *EWMA Position Document Understanding Compression Therapy*. London, UK: MEP Ltd; 2003,11-17.
17. Guest M, Williams A, Greenhalgh R, Davies A. Mixed Leg Ulcers. *Eur J Vasc Endovasc Surg*. 1999;**18**(6):540–541.
18. Moffatt CJ. International Leg Ulcer Algorithm. In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007,62-73.

19. Douglas V. Living with a Chronic Leg Ulcer: An Insight into Patients' Experiences and Feelings. *J Wound Care* 2001;**10(9)**:355–360.
20. Philips T, Stanton B, Provan A, Lew R. A Study of the Impact of Leg Ulcers on Quality of Life: Financial, Social and Psychosocial Implications. *J Am Acad Dermatol.* 1994;**31**:49–53.
21. Hareendran A, Doll H, Wild DJ, et al. The Venous Leg Ulcer Quality of Life (VLU-QoL) Questionnaire: Development and Psychometric Validation. *Wound Rep Reg.* 2007;**15**:465–473.
22. Franks PJ, Moffatt CJ, Connolly M, et al. Community Leg Ulcer Clinics: Effect on Quality of Life. *Phlebology.* 1994;**9**:83–86.
23. Kahn SR, M'lan CE, Lamping DL, Kurz X, Bérard A, Abenhaim LA. Relationship between Clinical Classification of Chronic Venous Disease and Patient-Reported Quality of Life: Results from an International Cohort Study. *J Vasc Surg* 2004; **39**:823-828.
24. Carpentier PH, Cornu-Thénard A, Uhl JF, Partsch H, Antignani PL. Appraisal of the Information Content of the Classes of CEAP Clinical Classification of Chronic Venous Disorders: A Multicenter Evaluation of 872 Patients. *J Vasc Surg.* 2003; **37**:827-833.
25. Yamada BFA, Santos VLCG. Quality of Life of Individuals with Chronic Venous Ulcers. *Wounds.* 2005;**17(7)**:178–189.
26. Guarnera G, Tinelli G, Abeni D, Di Pietro C, Sampogna F, Tabolli S. Pain and Quality of Life in Patients with Vascular Leg Ulcers: An Italian Multicentre Study. *J Wound Care.* 2007;**16(8)**:347–351.
27. Tennvall GR, Hjelmgren J. Annual Costs of Treatment for Venous Leg Ulcers in Sweden and the United Kingdom. *Wound Repair and Regeneration.* 2005;**13(1)**:13–18.
28. Tennvall GR, Hjelmgren J, Öien R. The Cost of Treating Hard-to-Heal Venous Leg Ulcers: Results from a Swedish Survey. *World Wide Wounds.* www.worldwidewounds.com/2006/november/Tennvall/Cost-of-treating-hard-to-heal-venous-leg-ulcers.html. November 2006. Accessed February 12, 2009.
29. Bosanquet N. Costs of Venous Ulcers: From Maintenance Therapy to Investment Programmes. *Phlebology.* 1992;**7(suppl 1)**:44–46.
30. Ruckley CV. Socioeconomic Impact of Chronic Venous Insufficiency and Leg Ulcers. *Angiology.* 1997;**48**: 67–69.
31. Weingarten MS. State-of-the-Art Treatment of Chronic Venous Disease. *Clin Infect Dis.* 2001;**32(6)**:949–954.

Chapter 3

Venous Leg Ulcers and Chronic Venous Insufficiency

This chapter looks at the causes of venous leg ulcers, the theories behind their formation, and explains their link with chronic venous insufficiency. It also describes the diagnostic tools that are used to determine the severity of underlying disease in patients with venous leg ulcers.

Chapter 2 provided the foundations for learning about venous leg ulcers. It discussed the different types of leg ulcers, the features and characteristics of each, and how they are differentiated.

Topics to be discussed in this chapter include:

- An overview of a healthy venous system
- Structure and function of veins
- Venous pressure in the healthy venous system
- Venous pressure in the diseased venous system
- Pathophysiological basis of venous hypertension
- Chronic Venous Insufficiency (CVI) and Chronic Venous Disease (CVD)
- Signs and symptoms of CVI
- Diagnosis of CVI
- CEAP classification of CVI
- Theories of ulcer formation

Upon completion of Chapter 3, you will:

- be able to describe normal venous function and how it compares with that of CVI
- have knowledge of the predisposing factors for CVI
- know the characteristics and pathology of CVI and the common types of diagnoses
- understand the CEAP classification system
- understand the relation between CVI and venous leg ulcers.

Overview of the Cardiovascular System

To understand the disease mechanisms that accompany leg ulcers, an understanding of how blood circulates around the lower limbs is useful. The next few sections discuss veins, arteries, and normal venous function.

The blood vessels in our bodies are split into two main types: arteries and veins (Figure 1). Arteries carry oxygenated blood away from the heart, whereas veins *return* the deoxygenated blood back to the heart (an easy way to remember it is, **A**rteries carry blood **A**way from the heart). Another difference between veins and arteries is that arteries have thick, muscular walls, whereas veins have thin walls with special one-way valves.¹



Figure 1. Schematic representation of human cardiovascular system showing arteries in red and veins in blue

Overview of the Venous System

Structure and function of veins

The primary purpose of veins is to return blood to the heart to be reoxygenated and recirculated. Starting at the heart, blood first travels through a network of arteries to reach the organs and tissues that require oxygen and nutrients. Arteries originate from the heart and divide like the branches of a tree, diminishing in size (arteries to arterioles), and eventually terminating in tiny single-cell-thick vessels called capillaries, which are distributed lavishly in every tissue in the body. At the capillary level, nutrients and oxygen are supplied to the muscles, bones, tendons, joints, other soft tissues, and skin. Capillaries also absorb carbon dioxide (CO₂) and waste products, which are carried in the blood back to the heart and lungs by another treelike network of vessels called veins that increase in size the nearer they are to the heart (capillaries to venules to veins). The veins essentially transport the blood so that it can be reoxygenated and cleaned of waste products and then pumped around the body again.

In the leg, the hierarchy of veins relates to their size and distribution within the limb. In ascending order of size, the veins of the leg are known as:^{2,3}

1. Superficial or saphenous veins
2. Perforator veins
3. Deep veins

Superficial veins

The superficial veins are also known as the saphenous veins and are located in the subcutaneous tissue of the leg. They form a network that eventually connects with the deep veins via the perforator veins. The main superficial veins are divided up into the small saphenous and the great saphenous veins. The great saphenous vein is the longest vein in the body and starts in the dorsum (sole) of the foot whereas the small saphenous vein begins in the lateral aspect of the foot.²

Perforator veins

Perforator veins are the vessels that connect the superficial venous system to the deep venous system. These veins are distributed at several locations in the leg from the medial ankle to the knee area, and are often named after their discoverer, for example, Cockett or Boyd.

Deep veins

Deep veins carry most of the blood out of the leg and generally follow the course of the associated arteries.⁴ There are 5 major branches of deep veins:

- 1) Anterior and posterior tibial veins – drain the foot and pass upward through the calf
- 2) Peroneal veins – originate in distal third of the calf and ascend deep in the muscle layer
- 3) Popliteal vein – located behind the knee, formed by the joining of the tibial and peroneal veins, continues upward through the adductor canal
- 4) Femoral vein – located in the thigh, is the continuation of the popliteal vein

- 5) **Common femoral vein** – is the result of the joining of the profunda femoral vein (draining the thigh muscle) and the femoral vein. After passing under the inguinal ligament, the common femoral vein becomes the external iliac.

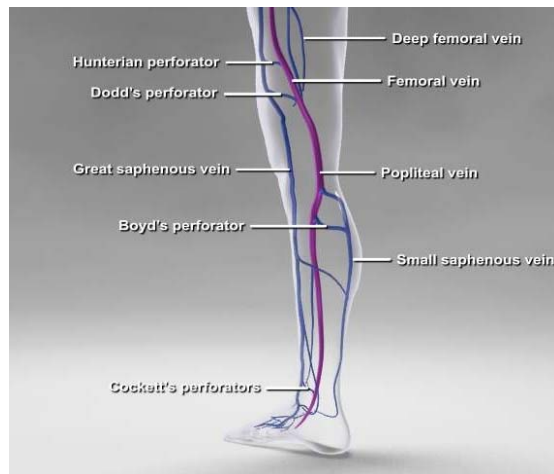


Figure 2. A schematic diagram of the veins of the leg showing superficial veins (include the saphenous veins and their branches), deep veins and the connecting perforator veins

Venous valves

On the pathway back to the heart and other organs, blood flows from the superficial veins, through the perforator veins, and to the deep veins through a system of venous valves (Figure 3a). Unidirectional blood flow is controlled by these valves that close in response to an increase in pressure from above, thereby preventing blood flowing backwards down the vein. Backward venous flow is known as reflux and retrograde flow.³ The muscles and tendons surrounding the deep veins work in concert with the venous valves, acting as a pump to force blood out of the leg and toward the heart during exercise (Figure 3b).

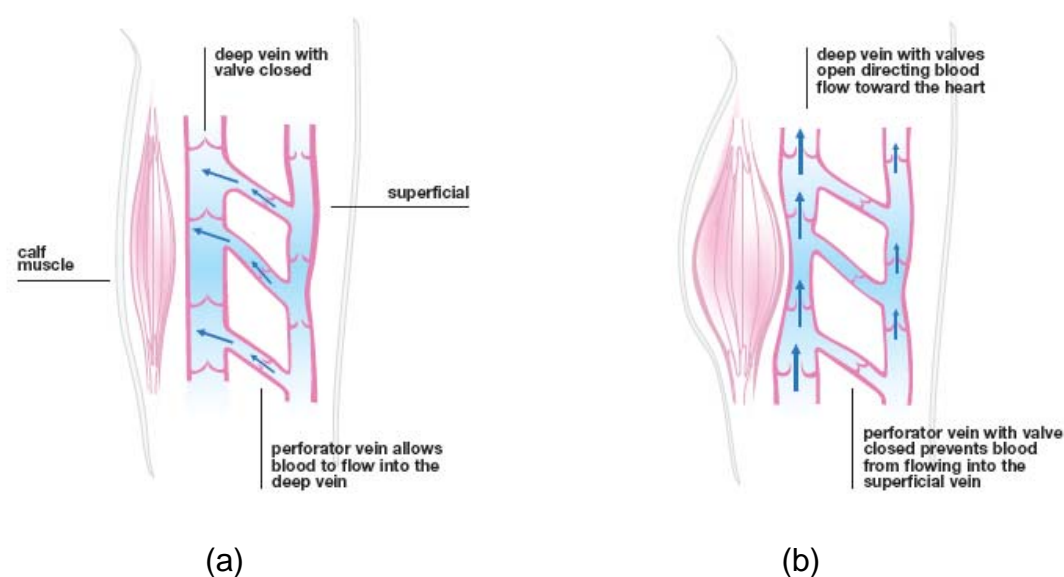


Figure 3. Normal venous function with the calf muscle relaxed (a) and with the calf muscle contracted (b)

Venous pressure in a healthy venous system

Venous pressure is determined by two components: the weight of the column of blood in the veins and the pressure change that can be generated by contraction of the leg muscles. The weight of the column of blood is correlated with the volume of blood in the leg.

When a patient is lying down, the heart is located on a horizontal plane with the feet and, as such, there is enough pressure remaining from the initial force of the heart pumping the blood into the legs to return the blood back to the heart. However, when the patient is standing, the heart is raised above the level of the feet, with a resulting increase in the pressure of the blood caused by the weight of the column of blood from the heart to the feet. When a patient stands without moving their feet or legs, venous pressure in the distal part of the leg can reach 80–90 mmHg (millimeters of mercury) and sometimes 100 mmHg in very tall people.

Calf muscle contraction generates intermittent peaks of venous pressure (i.e. temporary increases in pressure within the deep veins of the leg) by compressing the vein. The high pressure in the leg caused by this muscle movement creates a pressure gradient (i.e. a higher pressure in the lower part of the leg) that forces blood up the leg towards the heart⁵ emptying the deep and superficial venous system. Proximal valves (above the high pressure) open to allow blood to flow in this upward direction (Figure 4a). When the calf muscle relaxes, the pressure gradient is reversed (i.e. a higher pressure higher up the leg) and the valves close to prevent reflux (Figure 4b).³ This mechanism is collectively known as the calf muscle pump, and is the principle mechanism that pumps blood out of the leg.⁶



Figure 4a: Competent valve

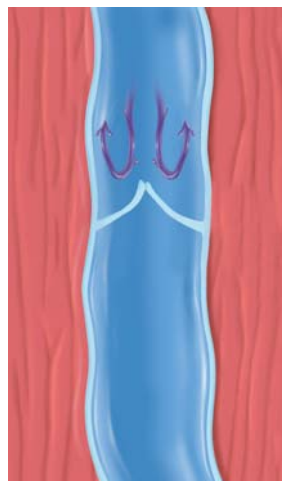


Figure 4b: Competent valve preventing reflux blood flow

During exercise the blood volume in the leg veins is reduced, thereby reducing the weight of the column of blood and the resultant venous pressure (see Figure 5). While walking at a constant pace, venous pressure, (known as ambulatory venous pressure (AVP)) in a healthy venous system is reduced to less than 30 mmHg.

When the subject stops walking, the blood volume of the leg increases through the arterial supply, resulting in an increase in the venous pressure. Eventually, a steady-state pressure will be reached again in the static standing patient.

In addition to the calf muscle pump, there is a pump mechanism in the dorsal structure of the foot that helps remove blood from the foot.⁶

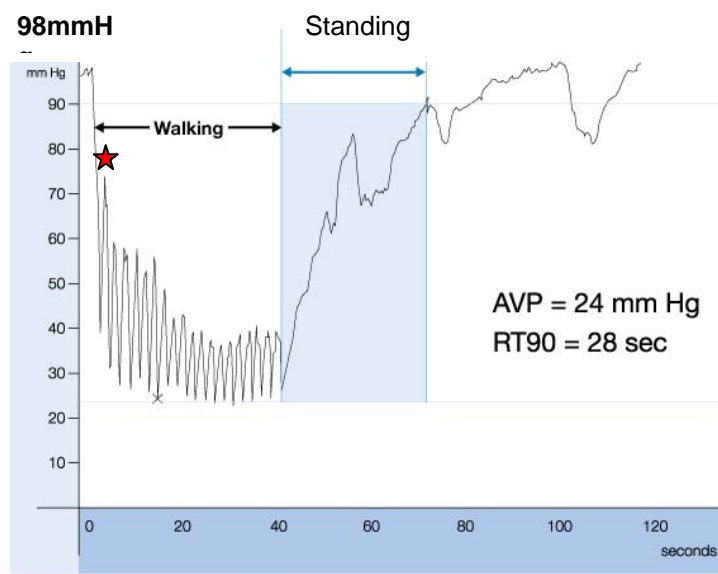


Figure 5. Pressure in the dorsal foot vein of a normal, healthy leg. Venous pressure at rest in this subject is 98mmHg. The asterisk indicates a pressure peak associated with contraction of the calf muscle. Walking leads to a reduction on venous pressure to 24mmHg. AVP (ambulatory venous pressure) is normal.

Venous pressure in the diseased venous system

In patients with CVI, this series of muscle-pumping events fails to occur, efficient emptying of the deep and superficial venous system does not take place during exercise, and gravity forces large volumes of blood to reflux into the superficial venous system. As a result, the subsequent reduction in venous pressure does not occur (Figure 6) and an abnormally high intravenous pressure, known as ambulatory venous hypertension (AVH), builds up.⁵ Thus, AVH is defined as the condition in which the venous pressure which equals the weight of the blood column between the foot and right atrium of the heart (expressed as mmHg)⁷ is higher than normal when an individual exercises.⁸

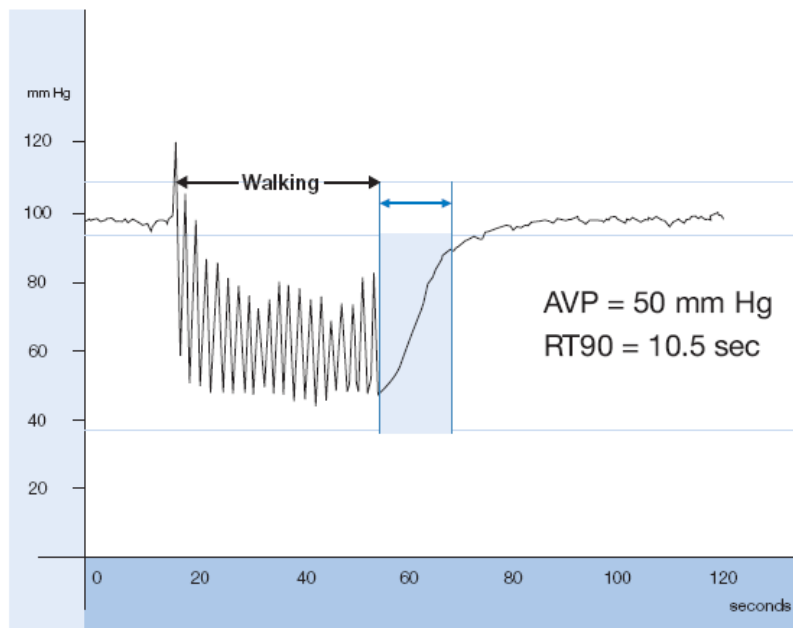


Figure 6. Pressure in a dorsal foot vein in a subject with chronic venous insufficiency. The pressure in a standing position is the same compared to a normal leg (98mmHg). During walking the deepest pressure values (AVP) are around 50mmHg, reflecting ambulatory venous hypertension.

Pathophysiological Basis of Venous Hypertension

Venous hypertension can be the result of one of a number of abnormalities. The two main causes are:⁹

- **Venous reflux** as a result of incompetent (“failed”) valves (accounts for approximately 90%)
- **Venous obstruction** (blockage: accounts for approximately 10%), which is usually post-thrombotic (following a blood clot), for example, deep vein thrombosis (DVT)

Other factors that can lead to venous hypertension include dysfunction of the calf muscle pump, immobility, prolonged standing by healthy or venous disease patients, trauma, or congenital absence of valves (congenital avalvulia).^{2, 3, 5}

Why do valves fail?

Valves may fail because of damage or incompetence present at birth or acquired later in life. Valves in the deep veins may suffer damage because of inflammation secondary to thrombosis (i.e. caused by a blood clot).^{2, 9, 10} Similarly, the perforating and superficial veins may become incompetent in a primary manner or they may develop incompetence secondary to a deep venous obstruction. An incompetent valve, as can be seen in Figure 7, does not prevent the reflux of blood.



Figure 7. A schematic representation of an incompetent valve and the resultant reflux of blood that occurs

CVI and Chronic Venous Disease (CVD)

What is CVI and CVD?

CVI is part of a broadly defined condition called chronic venous disease (CVD), which includes telangiectases, varicose veins, and edema without any other accompanying skin changes. CVI describes more-severe CVD that may be characterized by edema, lipodermatosclerosis, and ulceration of the leg.⁵

Despite the wide range of signs and symptoms associated with CVI, all are related to venous hypertension.

Signs and symptoms of CVI and CVD

CVI and CVD are characterized by a number of signs (Table 1) and symptoms (Box 1) that relate to the structural and functional abnormalities of the veins.¹⁰

Table 1. Clinical signs of CVI and CVD¹¹

Clinical sign	Visual symptom	
Telangiectasia	Dilated venules within the skin less than 1 mm in diameter. Also known as spider veins, thread veins, and hyphen veins	CVD
Reticular veins	Dilated veins below the skin 1–3 mm in diameter	
Varicose veins	Subcutaneous dilated vein 3 mm in diameter or larger	
Edema	Noticeable increase in volume of fluid in the skin and subcutaneous tissues; swelling	CVI
Pigmentation	Brownish darkening of the skin	
Venous eczema	Superficial reddening of the skin that may progress to blistering, weeping, or scaling. Most often located near varicose veins	
Lipodermatosclerosis	Localized chronic inflammation and hardening of the skin	
Atrophie blanche	Localized areas of skin atrophy, often whitish with surrounding dilated capillaries	
Active venous ulceration	Full-skin-thickness wound, usually in the gaiter region of the leg, that fails to heal spontaneously	

Box 1. Symptoms associated with structural and functional abnormalities of the veins**Symptoms**

- Aching legs^{9,11}
- Heavy legs^{9,11}
- Leg cramps^{9,11}
- Itching⁹
- Swelling⁹
- Restless leg syndrome⁹
- Skin changes¹²
- Dilation or prominence of superficial veins¹¹

CVI is characterized by leg swelling, pain, darkened skin color, hardened skin, and ulceration. Swelling is usually worse when the leg remains below the level of the heart known as “dependent” and improves after prolonged leg elevation. Leg pain is commonly described as heaviness or aching and is often worse in warmer weather.¹²

As the condition progresses, the skin may develop a tan or reddish-brown coloring, which is caused by leakage of red blood cells into the tissue and metabolism of hemoglobin, particularly at the ankle where AVH is highest. This can be followed by lipodermatosclerosis, which in severe cases can cover the circumference of the leg up to the mid calf. In these circumstances, it is common for the leg to resemble an upended champagne bottle, with the foot and leg above the lipodermatosclerosis swollen with edema.¹³

In between areas of heavy pigmentation, small areas of skin that have lost all pigmentation may often be found. These areas are known as atrophie blanche.¹³

Diagnosis of CVI

As CVI has various causes and a wide range of signs and symptoms, and because clinical examination will not always indicate the nature and extent of the underlying problem, a number of diagnostic investigations have been developed.¹⁰ It is vital that early diagnosis takes place as long-term CVI can lead to complications such as venous leg ulcers. Diagnosis should first entail obtaining the patient history followed by a clinical examination (including determination of the ankle brachial pressure index (ABPI)). If these tests are not conclusive, a series of invasive and noninvasive tests can then be performed.¹⁰ Diagnosis takes place by using techniques that are categorized by the degree of invasiveness (Levels I to III) as described below.

Level 1 assessment –history and clinical examination

The first step in diagnosing CVI is a thorough clinical history and examination to assess the patient's signs and symptoms (leg pain – whether on walking or resting, swelling, changes in skin color, hardening of the skin, etc.). This is referred to as “Level 1” assessment.

A clinical history, which should include the risk factors for CVI, is taken. These risk factors include:^{12, 14, 15}

- family history
- female sex
- decreased elasticity of the veins and valves caused by aging
- varicose veins
- pregnancy
- prolonged sitting, standing, or bed rest
- obesity
- previous history of major leg trauma
- conditions affecting the cardiovascular system including diabetes, hypertension, and high cholesterol
- smoking

There is a well-established association between DVT and chronic leg edema, pigmentation, and ulceration. Together, this is often referred to as the ‘postphlebotic’ or post-thrombotic syndrome. One third of all patients with DVT will develop CVI within five years.¹²

Every patient should also be assessed for concomitant arterial disease as this has implications for the treatment given to the patient.^{2, 16} The ABPI is helpful to identify the presence of peripheral vascular disease (arterial disease in the legs). See chapter 2 for further details.

Level 2 assessment – noninvasive diagnostic procedures/vascular lab testing

Duplex Doppler ultrasonography

Considered to be the “gold standard” for venous assessment, duplex Doppler ultrasonography, or duplex scanning is a noninvasive test that uses ultrasound waves to obtain a picture of the veins and determine the speed and direction of blood flow within the vein.

It provides an immediate picture of the veins and information on whether they are essentially normal or whether the valves allow an abnormal reflux of blood.¹⁷ This is achieved by using a color scanner that reveals blood flow down the leg in red and up the leg in blue. Duplex ultrasonography can also detect obstructions within the veins and has been used to diagnose DVT since the 1980s.¹⁰

Duplex ultrasonography is considered to be an extremely sensitive diagnostic test and is capable of detecting the degree of reflux by measuring valve-closure times. A valve-closure time of less than 0.5 seconds or less than 1.0 second (depending upon the position and technique of release of backflow) is considered normal.¹⁸

Plethysmographic techniques

These techniques measure volume changes within segments of the leg. One technique that is frequently used is air plethysmography in which a polyurethane air-filled chamber is wrapped around the leg and then inflated to a pressure of around 6mmHg. A measurement is made with the leg placed in an upward position to empty the veins and the changes in the pressure of the air chamber are measured and recorded when the leg is re-positioned and moved. These changes in pressure correspond to changes in the volume of the leg as a result of the veins emptying and filling. The patient is asked to place their leg in different positions to achieve this, for example, at a 45-degree angle or on their tiptoes.¹⁰

Another plethysmographic technique is photoelectric plethysmography, which can be used to detect changes in cutaneous blood volume. It consists of a sensor that transmits infrared light into the skin and senses its return. Blood contains red blood cells which absorb some of the infrared light in proportion to the volume of red cells. The infrared light reflected (not absorbed) is inversely proportional to the volume of red cells and is measured to calculate blood volume.¹⁹ This method can be used to measure arterial pulsations, transient venous volume, changes in venous volume, and changes in tissue venous filling when the patient performs physical movements.²⁰

As with the other plethysmographic techniques, strain-gauge plethysmography also assesses volume change. It involves wrapping a strain gauge (an elastic tube that expresses any change in volume as a change in voltage) around the calf.²⁰ The readings change as the calf volume expands and contracts and can be used for both arterial and venous applications.

Level 3 assessment – invasive diagnostic procedures

AVP

AVH is the most important factor in causing venous disease and its presence can be demonstrated by this diagnostic procedure. AVH is defined as a continued high AVP that is higher than that seen in a patient with competent veins (<30mm Hg) when the patient walks or activates their calf muscle pump.⁵

The AVP test is the best measurement of venous hypertension. It is based on the observation that venous pressure in the foot decreases during walking and gradually returns to the resting value when walking stops.¹⁰ Venous pressure is measured by inserting a needle (connected to a pressure transducer and amplifier) into a vein on the dorsum on the foot (upper surface of foot when standing). The patient is then asked to perform a series of up to 20 tiptoe movements or knee bends and venous-pressure measurements are recorded. Once the pressure has returned to baseline, the patient is asked to repeat the exercise after the inflation of a 2.5cm ankle cuff, which then occludes the superficial veins.

The refilling time (RT) is an important measure of reflux severity. It measures the amount of time taken for the venous system to refill with blood as indicated by a return to the baseline venous-pressure level. The refilling curve is exponential, which means that as the system approaches the fully filled level, the rate of filling decreases. This makes it difficult to establish exactly when the system is fully filled and so it is considered more accurate to define the 50% or the 90% refill time, rather than the 100% refill time. A normal value of venous pressure 90% RT (RT₉₀) is 18–40 seconds, with or without an inflated ankle cuff (Figure 8a).¹⁰ In a patient with venous disease, the RT₉₀ is considerably shortened, indicating that refilling is taking place from the venous system rather than the arterial system (Figure 8b). The RT₉₀ can be as short as 5 seconds or less. The drawback of this test is that it is an invasive procedure that requires patient mobility.

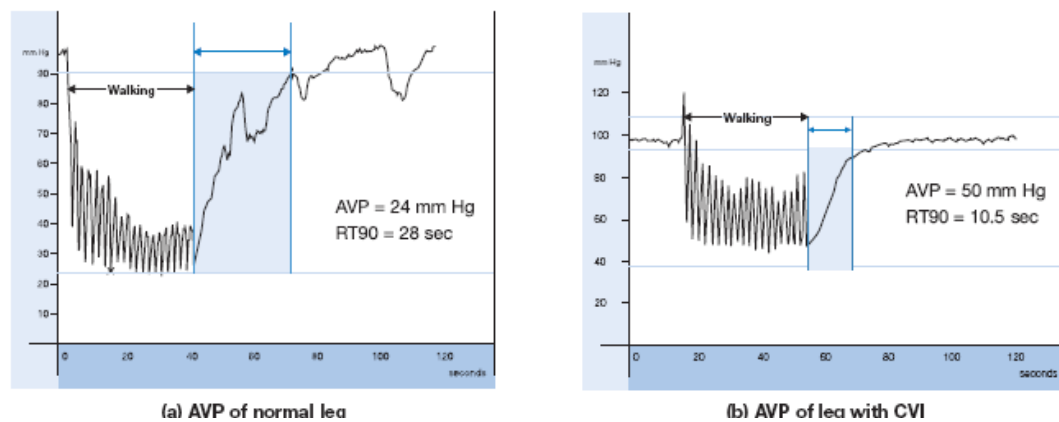


Figure 8. The continuous registration of the pressure in a dorsal foot vein on both legs in a patient with unilateral CVI. a) Pressure curve in normal leg. The starting resting pressure during standing is 98 mmHg and subsequent walking on the treadmill leads to a reduction in the venous pressure to 24 mmHg AVP. The RT₉₀ after standing still is 28 sec. b) Pressure curve from the contralateral leg with signs of venous insufficiency. The pressure in the standing position is the same as that of the normal leg (98 mmHg). During walking, however, the deepest pressure values (i.e. AVP) are around 50 mmHg, indicating AVH. As blood flows retrograde in the vein owing to valvular incompetence, the RT₉₀ is shortened to 10.5 sec.

Ascending venography

Ascending venography is also called ascending phlebography and is a standard test for assessing acute DVT or an obstruction. A radiologic contrast dye is injected into a vein in the foot and directed towards the deep veins by using an ankle tourniquet. Although this was once the method of choice for assessing the degree of obstruction within veins, the development of duplex scanning has made it unnecessary in most cases¹⁰

Descending venography

This test is used to assess the valves in the leg and examine reflux. It involves the injection of contrast media into the external iliac vein while the patient is lying on a tilting table. Again, this method has been superseded by duplex ultrasonography¹⁰

How are CVI and CVD Classified?

CVD may be categorized into primary or secondary disorders. Primary CVD is not associated with an identifiable mechanism of venous dysfunction. Although the direct cause is unknown, it is thought the CVD may arise from intrinsic structural and biochemical abnormalities of the vein wall. Primary CVD encompasses varicose veins and can also include skin changes and ulceration. Secondary CVD usually follows an episode of DVT, which, over time, recanalizes (the clot partially dissolves creating a channel). This leads to partial obstruction, reflux, and AVH. Clinically, secondary CVD includes pain, venous claudication (pain on walking), edema, skin changes, and ulcers. These symptoms are collectively called post-thrombotic syndrome.²¹

CEAP classification

With such a variety of symptoms and causes included in the grouping of CVD and CVI, a classification system to accurately describe each patient and their symptoms was developed. This is known as the CEAP classification system. The classification, which is qualitative, is based on **C**linical signs (C; 7 levels – see Table 2b), **E**tiologic factors (E; congenital, primary, or secondary), **A**natomic distribution of disease (A; superficial, deep, or perforator, alone or in combination), and underlying **P**athophysiologic cause (P; reflux, obstruction, or both).¹¹ Table 2a gives an overview of the CEAP system.

Table 2a. Overview of the CEAP system¹¹

CEAP Classification	
C	Clinical signs (grades 0 to 6 – see Table 2b), supplemented by ‘a’ for asymptomatic and ‘s’ for symptomatic (e.g. pain, heaviness, itching. etc.)
E	Etiologic classification E _c – congenital E _p – primary E _s – secondary E _n – no venous cause identified
A	Anatomic distribution A _s – superficial veins A _p – perforator veins A _d – deep veins A _n – no venous location identified
P	Pathophysiologic dysfunction P _r – reflux P _o – obstruction P _{r,o} – reflux and obstruction P _n – no venous pathophysiology identifiable

Table 2b. CEAP clinical classification¹¹

Class	Clinical signs
C ₀	No visible or palpable signs of venous disease
C ₁	Telangiectases or reticular veins
C ₂	Varicose veins
C ₃	Edema
C _{4a}	Skin and subcutaneous changes – pigmentation of eczema
C _{4b}	Skin and subcutaneous changes – lipodermatosclerosis or atrophie blanche
C ₅	Skin changes as defined above with a healed venous ulcer
C ₆	Skin changes as defined above with an active ulcer

CVI:
C3 to
C6

Example of basic CEAP classification

A patient has painful (s) swelling of the leg (C₃), varicose veins (C₂), lipodermatosclerosis (C_{4b}), and active ulceration (C₆). Duplex scanning showed axial reflux (P_r) of the great saphenous vein above and below the knee (A_s), incompetent calf perforator veins (A_p), and axial reflux in the femoral and popliteal veins (A_d, P_r). There are no signs of post-thrombotic obstruction.

The classification according to basic CEAP is therefore: C_{6,s}E_pA_{s,p,d}P_r.

Note: As the single highest descriptor can be used for clinical classification in basic CEAP, in this case active ulceration (C₆), this is what is then used in the classification.¹¹ The subscript 's' next to the C classification refers to the pain symptom reported for this patient. This should not be confused with the 's' applied to the A classification, which means "superficial".

In practice, many health care professionals only use the "C" classification, so, in the example here, this is a "C₆" patient.

How are CVI and Venous Leg Ulcers Related?

Venous leg ulcers are a consequence of long-term CVI.²¹ There is no general agreement on the sequence of events that leads from venous hypertension to venous ulceration,² although a number of theories do exist. Two early mechanistic models were the fibrin-cuff hypothesis²² and the growth-factor-trap hypothesis.² The former hypothesis proposed that fibrin, which is observed around blood vessels in ulcer patients, forms a physical barrier that oxygen and nutrients cannot penetrate, whereas the second hypothesis proposed that fibrin trapped vital growth factors so that they were unavailable to participate in the healing process. However, both of these theories have since fallen out of favor.²³

Recent theories have concentrated on changes in the microcirculation (the body's smallest blood vessels such as capillaries and venules) and inflammation. The white-cell-trapping hypothesis²⁴ proposed that venous hypertension leads to reduced capillary flow, which in turn traps white blood cells (leukocytes). The trapped cells release reactive oxygen metabolites and proteolytic enzymes (enzymes that breakdown proteins) that damage the capillaries, making them more permeable to large molecules and therefore more likely to trap additional white cells. The result is an inadequate blood supply to some areas of tissue, an inflammatory cascade, and tissue breakdown, which ultimately leads to ulceration.

Subsequent theories have expanded and refined Coleridge Smith's proposal. Herrick and colleagues linked the white-cell-trapping hypothesis with CVI, suggesting that trapped leukocytes release a cocktail of cytokines that trigger the surrounding tissue to synthesize a complex cuff of fibrin, collagen, and other molecules. The disappearance of the cuff as ulcers heal may reflect a lower level of inflammation.²⁵

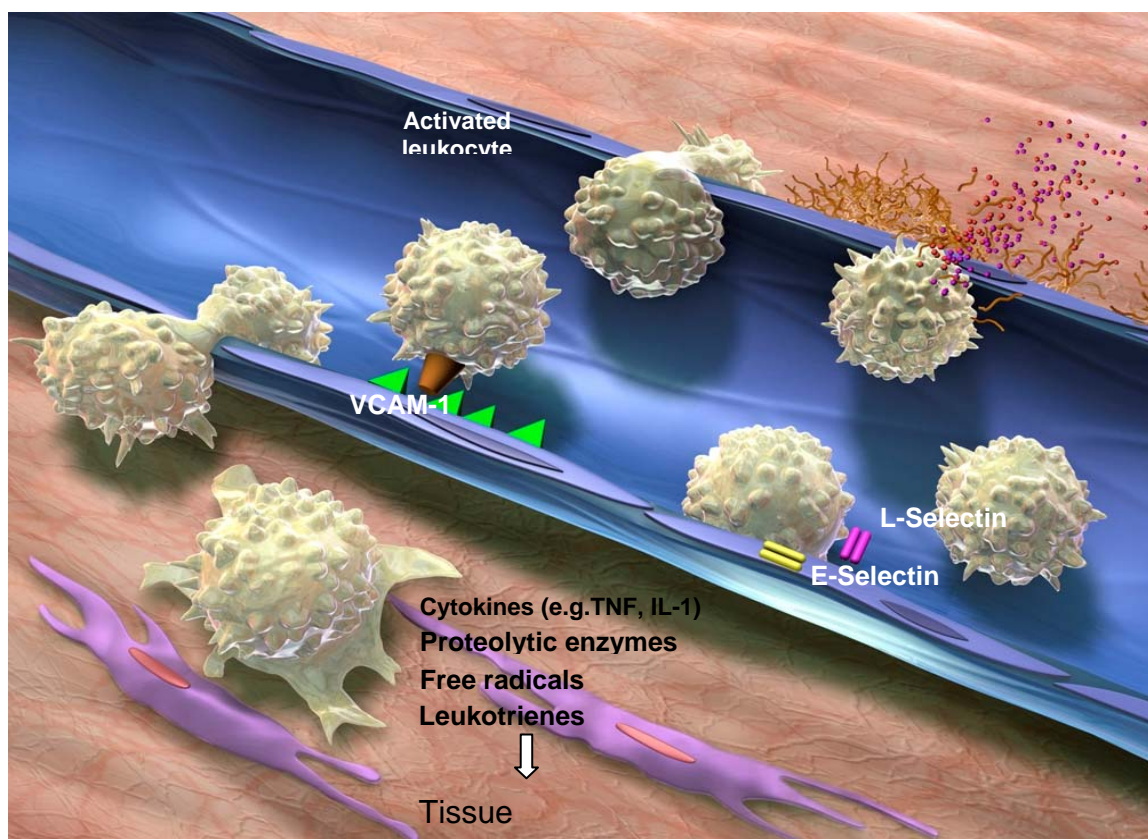


Figure 9. Schematic diagram representing leukocyte endothelium interaction and subsequent cell changes. Within the blood vessel or capillary, leucocytes “roll” along the endothelium aided by the expression of selectin molecules on both the leukocyte and the endothelium (leukocyte margination).²⁶ Abnormal hemodynamic forces, such as increased blood pressure and altered fluid shear stress, hypoxia and inflammatory events affect the endothelium and attract and activate large numbers of leukocytes.²⁶ These events trigger the expression of adhesion molecules such as vascular cell adhesion molecule (VCAM-1) on the surface of leukocytes; VCAM-1 firmly binds the leucocytes to the endothelial wall (leukocyte adhesion).²⁶ In the microcirculation of the lower leg, the accumulation of activated leukocytes under conditions of high venous pressure causes the release of inflammatory mediators (e.g. tumor necrosis factor [TNF], interleukin-1 [IL-1], and leukotrienes), proteolytic enzymes, and free radicals that have been associated with capillary damage, local ischemia and tissue breakdown.^{27, 28, 29}

Experts agree that inflammation plays a major role in the sequence of events that lead from CVI to leg ulceration (Figure 10).⁵ In 1995, Greenwood proposed that patients with CVI may also have low-level, long-term ischemia-reperfusion injury, which may predate the ulcer by months or even years.^{23, 30} Ischemia-reperfusion injury is considered to be the underlying pathology in many cardiovascular and inflammatory diseases. Indeed, many of the pathophysiological features of venous ulceration and CVI mirror those seen in other types of ischemia-reperfusion injury.²³

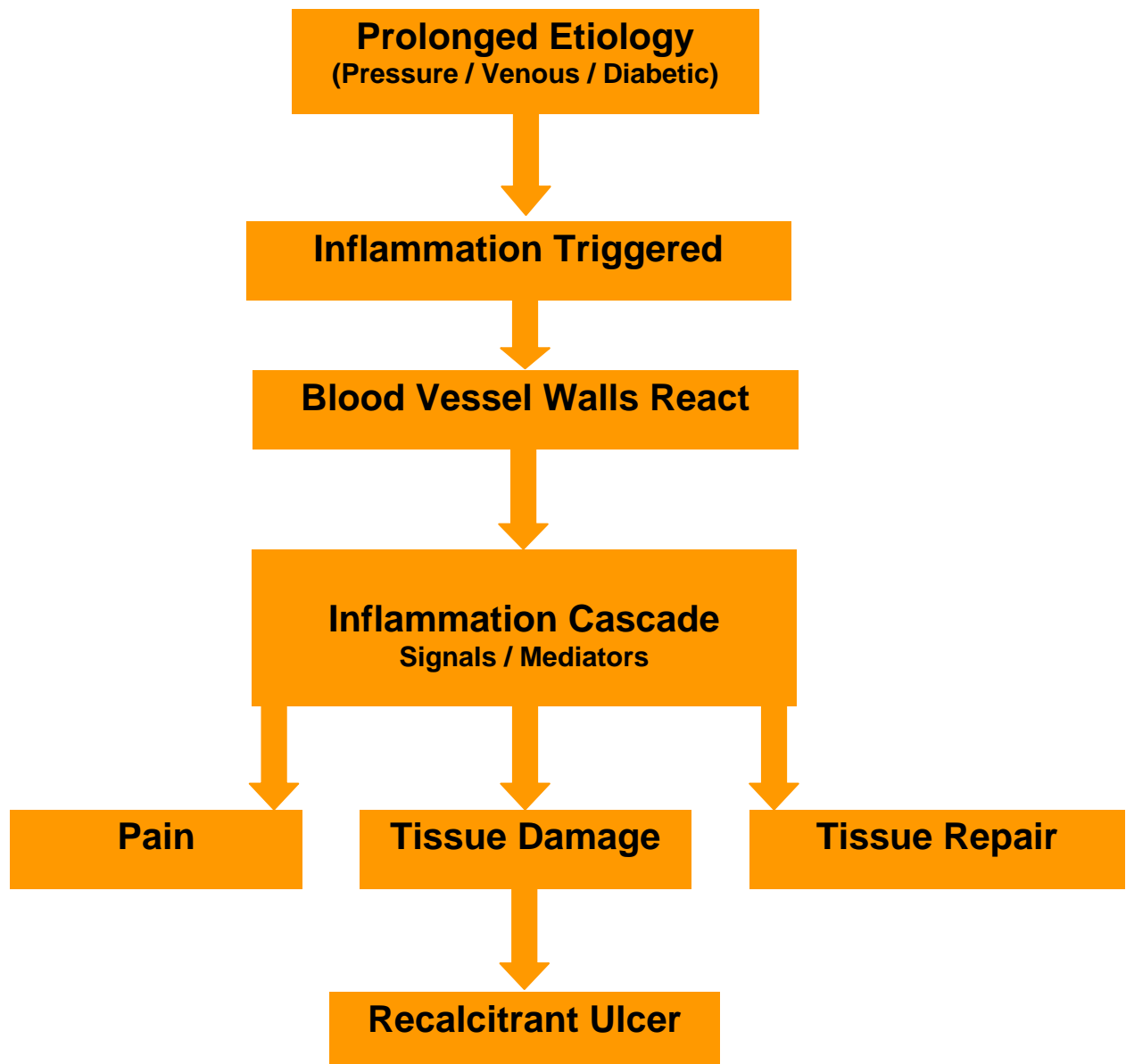


Figure 10. CVI: Pathophysiological events leading to venous ulceration

Summary

- The venous network of superficial, perforator, and deep veins return blood from the leg to the heart
- In a person with a healthy venous system, the muscles of the leg and foot work together with venous valves to pump blood out of the leg
- In a person with CVD, the calf muscle pump does not function properly, resulting in AVH
- Venous hypertension can be caused by a reflux of blood owing to incompetent or absent valves or venous obstruction
- AVH is the underlying cause of the varied signs and symptoms of chronic venous disease
- CVD describes a broad range of conditions including telangiectases, varicose veins, and edema
- CVI is a severe form of chronic venous disorder, in which venous ulcers are the most severe consequence
- The ABPI is used to detect the presence or absence of arterial disease
- Duplex Doppler ultrasonography is the gold-standard method of diagnosing venous insufficiency
- The CEAP classification system is a qualitative scale that is used to describe each patient and the symptoms that they exhibit
- The currently accepted theory to explain how CVI leads to the formation of a leg ulcer focuses on inflammation and the presence of ischemia–reperfusion injury

Chapter 3

Self Assessment Questions

Please circle the letter(s) to indicate the correct answer(s)

1. Which of the following statements is NOT TRUE?

- A. The venous system is divided into three components: the superficial, perforating, and deep systems.
- B. Blood is moved from the leg toward the heart primarily by the pumping action of the leg muscles.
- C. When standing still, the pressure in the superficial and deep systems is about 30 mmHg
- D. An intact venous system and calf muscle pump are essential in avoiding retrograde flow to the superficial system.

2. The following are risk factors for CVI, except:

- A. Varicose veins
- B. Pregnancy
- C. Obesity
- D. Skin ulceration
- E. Smoking

3. Which of the following is FALSE?

- A. The normal function of venous valves is to prevent the reflux of blood
- B. Valves normally close in response to changes in pressure
- C. Incompetent valves only allow the flow of blood in one direction
- D. Incompetent valves result in venous hypertension

4. Which of the following statements is NOT TRUE?

- A. Venous hypertension resulting from incompetent valves accounts for 90% of chronic venous insufficiency
- B. Incompetent venous valves are always the result of post-thrombotic damage
- C. Venous obstruction is a cause of venous hypertension
- D. One third of all patients with DVT will develop CVI within 5 years

5. Which of the following statements is FALSE?

- A. White blood cells release toxic oxygen metabolites and proteolytic enzymes when they are trapped
- B. Inflammation plays a major role in leg ulceration
- C. Proteolytic damage makes capillaries less permeable, which results in edema
- D. Ischemia-reperfusion injury is considered to be the underlying pathology in many cardiovascular and inflammatory diseases

6. A patient with a history of DVT and venous disease secondary to post-phlebotic syndrome has a venous leg ulcer and a healed ulcer and lipodermatosclerosis of the right leg. Venous investigations show reflux in the great saphenous vein and Cockett perforator.

Is the CEAP classification:

- A. C₆E_sA_{s,p}P_r
- B. C₅E_pA_{p,d}P_r
- C. C₆E_sA_sP_o
- D. C_{6,s}E_sA_{s,p,d}P_r

Chapter 3

Self Assessment Answer key

1. **C** – the venous pressure when standing still is 80–90 mm Hg
2. **D** – not a risk factor
3. **C** – incompetent valves allow blood to flow in both directions, instead of just in one direction
4. **B** – not always, weakened vein walls can also result in incompetent valves
5. **C** – Capillaries become more permeable
6. **A**

References

1. Warren D, Attridge E, Goodman N. Keeping Healthy. In: Skinner R, ed. *OCR GCSE Science*. Carnforth, UK: Lonsdale; 2006, 33-42.
2. Valencia I, Falabella A, Kirsner RS, Eaglstein WH. Chronic Venous Insufficiency and Venous Leg Ulceration. *J Am Acad Dermatol*. 2001;**44**:401–421.
3. Etufugh CN, Philips TJ. Venous Ulcers. *Clin Dermatol*. 2007;**25**:121–130.
4. Gray H. *Anatomy of the Human Body* (20th Edition). Philadelphia, PA: Lea & Febiger, 1918; Published New York: Bartleby.com, 2000.www.bartleby.com/107/173.html. Accessed January 27, 2009.
5. Bergan JJ, Schmid-Schönbein GW, Coleridge Smith PD, Nicolaides AN, Boisseau MR, Eklof B. Chronic Venous Disease. *NEJM*. 2006;**355**:488–498.
6. Meissner MH, Moneta G, Burnand K et al. The Hemodynamics and Diagnosis of Venous Disease. *J Vasc Surg*. 2007; **46**:4S–24S.
7. Partsch B, Partsch H. Calf Compression Pressure Required to Achieve Venous Closure from Supine to Standing Positions. *J Vasc Surg*. 2005; **42**:734-738.
8. Partsch H, Clark M, Bassez S. Measurement of Lower Leg Compression In Vivo: Recommendations for the Performance of Measurements of Interface Pressure and Stiffness. *Dermatol Surg*. 2006; **32**:224–233.
9. Bradbury A, Ruckley CV. Clinical Assessment of Patients with Venous Disease. In: Gloviczki P, Yao JST, eds. *Handbook of venous disorders - Guidelines of the American Venous Forum* 2nd ed. New York, NY: Oxford University Press Inc; 2001:71–82.
10. Nicolaides AN. Investigation of Chronic Venous Insufficiency. A Consensus Statement. *Circulation*. 2000;**102**:103–163.
11. Eklof B, Rutherford RB, Bergan JJ, Carpentier PH et al. Revision of the CEAP Classification for Chronic Venous Disorders: Consensus Statement. *J Vasc Surg*. 2004; **40**(6):1248–1252.
12. Beckman JA. Diseases of the Veins. *Circulation*. 2002;**106**:2170–2172.
13. Alguire PC, Mathes BM. Chronic Venous Insufficiency and Venous Ulceration. *J Gen Intern Med*. 1997;**12**:374–383.
14. Staffa R. Chronic Venous Insufficiency – Epidemiology. *Bratisl Lek Listy*. 2002;**103**:166–168.
15. Berard A, Abenheim L, Platt R, Khan SR, Steinmetz O. Risk Factors for the First-Time Development of Venous Ulcers of the Lower Limbs: The Influence of Heredity and Physical Activity. *Angiology*. 2002; **53**(6):647–657.
16. Moffatt CJ. How Compression Works In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007,1-15.
17. Nachreiner RD, Bhuller AS, Dalsing MC. Surgical Repair of Incompetent Venous Valves. In: Dalsing MC, ed. On-line resource. *The Vein Handbook. A Layman's Version of Venous Disorders*. <http://veinforum.org/index.php?page=hbk16b>. Accessed January 27, 2009.
18. Rutherford RB, Padberg FT, Comerota AJ, Kistner RI, Meissner MH, Moneta GL. Venous Outcomes Assessment. In: Gloviczki P, Yao JST, eds. *Handbook of venous disorders - Guidelines of the American Venous Forum* 2nd ed. New York, NY: Oxford University Press Inc; 2001: 498–507.

19. Fronek HS. Functional Testing for Venous Disease. In: Fronek HS, ed. *The Fundamentals of Phlebology: Venous Disease for Clinicians*. Oakland CA: American College of Phlebology; 2004:17-22.
20. Araki CT, Hobson RW. Indirect Non-Invasive Tests (Plethysmography). In: Gloviczki P, Yao JST, eds. *Handbook of venous disorders* 2nd ed. New York, NY: Oxford University Press Inc; 2001:104-109.
21. Meissner MH, Eklof B, Coleridge Smith P et al. Secondary Chronic Venous Disorders. *J Vasc Surg*. 2007; **46**:68S-83S.
22. Burnand KG, Whimster I, Naidoo A, Browse NL. Pericapillary Fibrin in the Ulcer-Bearing Skin of the Leg: The Cause of Lipodermatosclerosis and Venous Ulceration. *BMJ*. 1982; **285**:1071-1072.
23. Chen WYJ, Rogers AA. Recent Insights into the Causes of Chronic Leg Ulceration in Venous Diseases and Implications on other Types of Chronic Wounds. *Wound Rep Regen*. 2007;**15**:434-449.
24. Coleridge Smith PD, Thomas P, Scurr JH, Dormandy JA. Causes of Venous Ulceration: A New Hypothesis. *BMJ*. 1988; **296**:1726-1727.
25. Herrick SE, Sloan P, McGurk M, Freak L, McCollum CN, Ferguson MW. Sequential Changes in Histologic Pattern and Extracellular Matrix Deposition During the Healing of Chronic Venous Ulcers. *Am J Pathol*. 1992;**141**(5):1085-1095.
26. Boisseau MR. Leukocyte Involvement in the Signs and Symptoms of Chronic Venous Disease. Perspectives for Therapy. *Clinical Hemorheology and Microcirculation*. 2007;**37**:277-290.
27. Eberhardt RT, Raffetto JD. Chronic Venous Insufficiency. *Circulation*. 2005;**111**:2398-2409.
28. Abbade LP, Lastória S. Venous Ulcer: Epidemiology, Physiopathology, Diagnosis and Treatment. *Int J Dermatol*. 2005;**44**:449-456.
29. Trent JT, Falabella A, Eaglstein WH, Kirsner RS. Venous Ulcers: Pathophysiology and Treatment Options. *Ostomy Wound Manage*. 2005;**51**(5):38-54.
30. Greenwood JE, Edwards AT, McCollum CN. The Possible Role of Ischaemia-Reperfusion in the Pathogenesis of Chronic Venous Ulceration. *Wounds*. 1995;**7**:211-219.

Chapter 4

The Care and Treatment of Venous Leg Ulcers

This chapter discusses caring for venous leg ulcers and the treatment strategies that can be employed. Although the focus of this chapter is mainly on compression therapy, other management strategies are also discussed.

Chapter 2 discussed leg ulcer etiology and the burden of leg ulcers on both the patient and health services. Chapter 3 expanded on this by describing the pathology behind venous leg ulcers and chronic venous insufficiency. Chapter 4 now continues your training with a discussion of the different approaches to managing a venous leg ulcer and the treatment options currently available for chronic venous insufficiency.

Topics to be discussed in this chapter include:

- Compression therapy for venous leg ulcers
 - Theory behind compression therapy
 - Types of compression therapy
 - Bandages
 - Hosiery
 - Intermittent pneumatic compression (IPC)
 - Limitations of compression therapy
- Management considerations for venous leg ulcers
 - Treatment algorithms and guidelines
 - Outcomes and factors that influence healing
 - Post-healing and long-term management
 - Assessment and education
- Other treatment strategies for chronic venous insufficiency (CVI)

Upon completion of Chapter 4, you will:

- understand why compression is the cornerstone of therapy for venous leg ulcers
- be familiar with the theory behind compression
- be aware of the different types of compression available
- understand the limitations associated with current compression therapies
- appreciate the role of treatment algorithms
- recognize the factors that can affect the successful healing of a venous leg ulcer
- gain a basic knowledge of other treatment strategies

Compression Therapy for Venous Leg Ulcers

What is compression therapy?

Any treatment strategy to heal a venous leg ulcer must directly address the underlying cause of venous disease, ambulatory venous hypertension (AVH). To achieve this, venous reflux needs to be reduced. One way to prevent venous reflux is through compression therapy.¹

Compression therapy is the application of external pressure (measured in mmHg) to the leg at a level that will influence the intravenous pressure. Compression is commonly applied by a range of compression devices, such as, hosiery (stockings) and bandages.

“One of the main intentions of adequate compression therapy of the lower extremities is to counteract gravity.”¹

How does compression therapy work?

The application of external compression through compression therapy results in a variety of complex physiological and biochemical effects that involve the venous, arterial, and lymphatic systems.²

The effects of compression therapy, when performed correctly, can be dramatic, reducing and suppressing superficial and deep venous reflux, restoring valvular competence, reducing edema, softening lipodermatosclerotic skin, aiding venous return, increasing arterial flow, enhancing lymph drainage, improving the microcirculation, and promoting ulcer healing.^{3,4}

Interface pressure (also known as sub-bandage pressure) is the term used to describe the level of compression delivered by a device. In a patient with venous leg ulcers and ABPI above 0.8, the clinician aims to apply between 30 and 45mmHg at the ankle graduated with decreasing pressure up the leg to below the knee.^{5,6} The ankle pressure is measured with the patient lying down (“supine”) at the point on the leg where the Achilles tendon joins the calf muscle, and this point is called the “B1 position”.⁷ With some compression products, this pressure level can reach values of 60–80 mmHg during walking as the calf muscle expands. These higher peaks of pressure are sometimes referred to as working pressure (an intermittent pressure exerted by a combination of the active muscle working against the resistance of the bandage).⁸ The pressure peaks can create intermittent venous occlusions, which are thought to be helpful in reducing reflux and decreasing AVH.⁶

When the calf muscle pump is at rest, as is the case when the patient is sitting or lying down, the exerted interface pressure is lower and is sometimes referred to as the resting pressure (a continuous force exerted externally from a bandage

towards the tissue in a resting patient).⁸ When a patient is lying down, there is minimal gravitational pressure to work against,⁹ especially if the legs are elevated above the heart, and as such there is a reduced need for compression.¹⁰ The calf muscle pump and the working pressure both affect the venous circulation and the level of edema in the leg.

Effects of compression on venous circulation

Hemodynamic effect

To counteract the effects of gravity and incompetent valves (see Chapter 3), a level of compression that exerts a pressure higher than the intravenous pressure is required.¹ Compression must also create a pressure gradient between the ankle and the knee, with the greatest pressure applied at the ankle (see Figure 1). This pressure gradient aids in the movement of fluids against the gravitational force and towards the heart.¹¹ The amount of blood ejected by the calf muscle pump (so-called “ejection fraction”) is increased under compression.¹²

Applying an adequate level of compression will:^{2,10}

- reduce the diameter of major veins
- accelerate blood flow back to the heart
- reduce blood volume in the leg by redistributing blood towards the center or trunk of the body

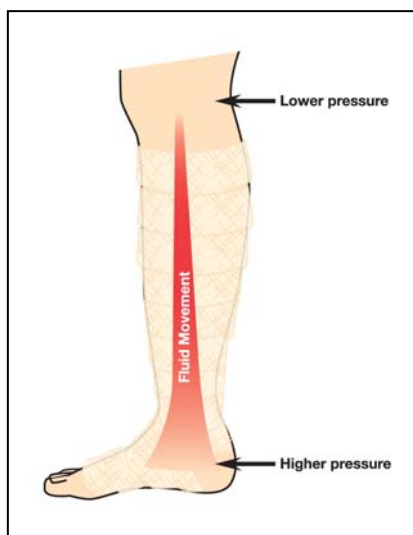


Figure 1. Pressure gradient extended from ankle to knee will achieve fluid movement against the gravitational force.

Administration of interface pressures of 35–45 mmHg at the ankle while the patient is in a supine position is effective in the management of patients with venous leg ulcers.⁶

Reducing venous reflux

Applying compression on a leg that has incompetent venous valves reduces the venous reflux and produces an increase in orthograde blood flow.² This increase in blood flow reduces edema (accumulation of fluid in extravascular tissue)¹³ and lipodermatosclerosis, and the application of compression bandages has been shown to increase ulcer-healing rates more effectively than no compression.⁴

Improving edema

Edema is described as the accumulation of fluid in extravascular tissue.¹³ Complex mechanisms are involved in the formation of edema, including an increase in the permeability of the capillaries without a concomitant increase in fluid clearance rate, which then allows fluid to accumulate.¹⁰ Edema formation is described by Starling's equation, which shows the relationship between tissue and fluid pressures and the filtration rate. The increased pressure that compression provides on local tissue encourages the reabsorption of fluid into the venous and lymphatic systems and prevents filtration from the capillaries.^{5, 10}

Types of Compression

Sustained graduated compression (SGC), which is the gold standard in venous leg ulcer management, is typically achieved with compression bandaging, which creates a pressure gradient in which the pressure is highest at the ankle and diminishes up the leg. Other compression systems are also available including hosiery (or stockings) and orthostatic band devices.

Intermittent pneumatic compression (IPC) involves the application of intermittent cycles of compression administered by a 'boot' or sleeve inflated with air by an electrical pump. The inflatable sleeves can have single or multiple chambers. Multiple-chamber devices can provide sequential compression. Intermittent pumping through inflation and deflation of the chambers creates intermittent pressure peaks, thereby mimicking the effect of the calf muscle pump.^{14, 15} The different types of compression systems are summarized in Table 1.

Table 1. Types of compression systems³

Type	Details
Compression bandaging – Long-stretch (or elastic) bandages – Short-stretch (or inelastic) bandages – Rigid compression – Multi-component systems Note: these are often referred to as multilayer systems, but multi-component is the recommended terminology ⁷	Highly elastic bandage, popular in the UK. Inelastic or minimally elastic bandage, widely used in Europe. Unna's boot – inextensible bandage impregnated with a zinc paste. Popular in the US. Orthostatic band device – system with multiple adjustable Velcro® straps. Based on the principals of the original four-component Charing Cross system. Combination of short- and long-stretch bandages and padding. Also includes 2- and 3-component systems.
Elastic hosiery or stockings	Often used to prevent recurrence. Some systems use two components.
Intermittent pneumatic compression (IPC)	Systems that consist of an air pump and 'boot' that fits around the leg and foot that intermittently inflates and deflates with air.

Compression bandages

Compression bandaging is the gold-standard therapy in the management of venous leg ulcers. The degree of compression produced by any bandage system is determined by the interactions between four major factors:¹³

- The physical structure and elasticity of the bandage
- The size and shape of the limb
- The application of the bandage
- The nature of physical activity undertaken by the patient.

Factors affecting performance

1) Laplace's law

The resting pressure generated by a bandage immediately after its application is determined by the tension in the bandage itself, the width of the bandage, the number of layers applied, and the limb circumference. The relationship between these factors is governed by Laplace's law:

$$\text{Pressure} = \frac{N \times T \times 4620}{C \times W}$$

Where:

N=Number of layers applied – *the more layers, the greater the pressure*

T=Bandage tension – *the greater the force applied, the greater the pressure*

C=Limb circumference/shape – *the smaller the circumference at any given point, the greater the pressure*

W=Bandage width – *the narrower the bandage, the greater the pressure*

“[Laplace's law] states that sub-bandage pressure is directly proportional to bandage tension, but inversely proportional to the radius of curvature of the limb to which it is applied.”¹⁶

This relationship governed by Laplace's law indicates that any bandage applied with constant tension to a limb of normal proportions (the circumference of the ankle is smaller than that of the calf) will automatically produce a sub-bandage pressure gradient from the ankle to the calf (in which the pressure produced by the bandage will decrease as the circumference of the limb increases.)¹³

A way to understand Laplace's law is to stretch a piece of cloth over the edge of a table. Trap one piece of paper under the flat portion on the table top and another under the portion stretched over the edge. Now try to pull out each piece of paper. The piece under the flat portion will pull out easily whereas the other piece will not. How does this relate to Laplace's law? The radius on the flat portion is infinite whereas the radius under the edge portion is small. Where the radius is small the difficulty in removing the paper is high. This is because the pressure is high at the edge portion. The same principle applies with bandaging, other wrapped compression products or orthostatic devices. The smaller the radius, the higher the pressure.

2) Physical properties and materials

The physical properties of the bandage also have an influence on the performance of the bandage. The physical properties of a bandage are summarized in Table 2.

Table 2. Physical bandage properties

Bandage property	Description
Tension	Determined by: ⁷ <ul style="list-style-type: none"> - force applied during application - ability of the bandage to sustain applied tension over time
Extensibility	Ability of a bandage to stretch when a force is applied – bandages can be described as short-stretch or long-stretch bandages based on their extensibility ¹³
Elasticity	The ability to return to the original unstretched length when tension is removed* ¹³
Stiffness	Defined as the increase in sub-bandage pressure per centimeter increase in the circumference of the leg. ⁸ Measures how resistant to stretching the fabric is. The stiffer the fabric is, the better it can function to bridge skin folds and the less likely it is to cut into the skin

* Note: the term “elastic” is often used interchangeably with terms that describe extensibility. Thus an elastic bandage is a long-stretch bandage, and an inelastic bandage is a short-stretch bandage.¹³

Bandage terminology and classification

Describing the different bandaging systems available is complex as there are no international or European standards to classify compression bandages by their performance. Table 3 illustrates the bandage classification and corresponding pressures in the UK where compression bandages are classified according to their ability to apply a specified sub-bandage pressure to an ankle circumference of 23cm when the bandage is applied with a 50% overlap.

Table 3. Compression levels of compression bandages according to the British standard (BS 7505) classification systems^{7,17}

Compression level	Classification	MmHg
Light	3A	<20
Medium	3B	21–30
High	3C	31–40
Extra high	3D	41–60

In 2008, the International Compression Club (ICC) published a consensus document to help standardize the classification of compression bandages.⁷ The recommendations aim to describe compression bandages in a way that informs healthcare practitioners of the likely effect of the bandage on a patient's leg. The recommendations are set out in Table 4.

Table 4. Current sub-bandage pressure ranges (mmHg) in the British standard (BS 7505) and ICC recommended recalibration to match *in vivo* pressure measurements in the medial gaiter area in the supine position

British Standard	ICC Recommendation
<20 ("light")	<20 ("mild")
21–30 ("medium")	20–<40 ("medium")
31–40 ("high")	40–<60 ("strong")
41–60 ("extra high")	≥60 ("very strong")

Table adapted from Partsch *et al* (2008).⁷

The lack of adopted international standards, ambiguous terminology, and limited understanding of bandage properties presents a practical problem in delivering effective compression.¹⁴ The term 'multilayer' is often used by practitioners to describe the individual products that make up a bandage system (e.g. wool padding, elastic bandages etc.). The term 'components' is a better way to describe the different elements of a bandage system.⁷ The term 'layer' should

describe the overlapping technique when a single bandage is applied to a leg, that is, the overlapping layers create several layers of bandage at specific points along a leg. The ICC points out that a single-layer bandage does not actually exist as there will always be some level of overlap. An overlap of 50% gives at least two layers of material at any point on a bandaged leg.⁷

Categories of compression bandages

Compression bandages can be broadly categorized into the following groups:

- Long-stretch (elastic) bandages
- Short-stretch (inelastic) bandages
- Rigid compression
- Multi-component systems

1) Long-stretch (elastic) bandages

Widely used in the UK, long-stretch (elastic) bandages contain elastomers that allow extension of 90–140% and will return to almost their original size once force is released. A category 3C bandage (British standard BS 7505; see Table 3) can be applied as a single component over a layer of padding. Elastic single-component bandages are designed to be capable of applying the total amount of compression required when the bandage is applied as a spiral with a 50% extension and a 50% overlap.^{3, 8}

Long-stretch (elastic) bandages are able to adapt to changes in calf dimension, thereby maintaining the sub-bandage pressure. In ambulatory patients, the rise in interface pressure is smaller (i.e., lower working pressure) with long-stretch (elastic) bandages than the stiffer short-stretch ones. As a result, there is a reduced hemodynamic effect,¹² however, patient comfort is increased. Conversely, reduction in edema and the resultant decrease in leg dimensions do not cause as great a drop in the pressure exerted by the compression bandage as that which occurs with short-stretch bandages. Long-stretch (elastic) bandages are therefore good at sustaining compression on a limb for up to a week. However, overextension of a single-component long-stretch bandage, carries the danger of pressure damage to the limb.^{3, 8} Table 5 summarizes the advantages and limitations of elastic bandaging.

Table 5. Advantages and limitations of elastic bandaging

Advantages	Limitations
Accommodates changes in limb circumference and provides sustained pressure during walking and resting	Provides a lower working pressure on ambulation – i.e. reduced hemodynamic effect
Patient comfort	Risk of pressure damage with over extension



Figure 2. Long-stretch bandage (Surepress® High Compression Bandage) being applied to a patient. Note the symbol on the bandage that guides the practitioner how much to extend the bandage. In this case it is a rectangle that extends to a square.

2) Short-stretch (inelastic) bandages

Short-stretch (inelastic) bandages are used extensively across Europe and Australia. They are applied at full stretch to create a rigid casing around the limb. Short-stretch bandages are generally made of cotton, without elastomers, and have an extension range of 40–90 %, whereas inelastic bandages do not stretch at all. This degree of stretch means that the short-stretch bandages produce a high working sub-bandage pressure when the calf muscle is active, and low sub-bandage pressure when the calf muscle is at rest (i.e., high working pressure and low resting pressure).

Short-stretch (inelastic) bandages cannot accommodate changes in limb circumference that are caused by a reduction in edema and therefore the bandage can become loose and slip down the leg and may lead to further skin trauma and ulceration.¹⁸ Regular reapplication is required especially in the early stages of treatment when changes in edema may be greatest.^{3, 8}

Short-stretch or inelastic bandages have the advantage that they remain rigid and thereby resist changes in the geometry of the calf muscle during exercise. High pressure peaks of 60–80 mmHg (the working pressure) may be achieved. Mosti et al (2008) has demonstrated that, even when both bandages are applied at the same standing pressure, inelastic material shows a significantly higher pressure increase during exercise compared with elastic materials and concludes that inelastic compression is more effective at normalizing venous pumping function than elastic compression in subjects with venous insufficiency.¹²

Stiffness can be measured using the static stiffness index (SSI), which describes the change in sub-bandage pressure that occurs when a patient moves from a lying to a standing position.^{1, 14}

Table 6. Advantages and limitations of inelastic bandaging

Advantages	Limitations
Strong hemodynamic effect i.e. produce a high, working sub-bandage pressure when the calf muscle is active	Does not accommodate changes in limb circumference with edema reduction
	Regular reapplication required especially in the early stages of treatment
	Can be uncomfortable and adversely effect patient compliance

3) Rigid compression

Rigid compression systems are more popular in the US than in Europe or the UK. Unna's boot is an example of a rigid compression system that consists of a bandage impregnated with zinc paste. Upon application, the bandage hardens to form a "molded boot" that provides a high working pressure but very little resting pressure, which is similar to the pressure provided by short-stretch bandages (Figure 3). Rigid compression is really only useful in ambulatory patients as it relies on a working calf muscle to apply compression and, as is the case with short-stretch bandages, cannot accommodate any reduction in edema.³



Figure 3. Unna boot (Unna-Flex™ ConvaTec Inc. Unna Boot dressing) applied to a patient

Although not strictly classified as bandages, orthostatic band devices, for example, CircAid® (CircAid Medical Products Inc.), are another form of rigid

compression available in the US. They consist of a completely inelastic, non-stretch wrap with multiple Velcro® (Velcro Industries B.V.) straps that can be adjusted to provide a high working pressure.³

4) Multi-component systems

The original multi-component (or 'multilayer') bandage system was developed in the 1980's at Charing Cross Hospital in London. It was designed to apply a high level of compression (35-40mmHg) by using four layers of weak elastic bandages applied to give an accumulated pressure that could be maintained for one week.⁸ More modern variations consist of a combination of:

- Cohesive/adhesive layers
- Short-stretch/inelastic layers
- Long-stretch/elastic layers
- Crepe bandages
- Padding

The properties of each component contribute to the overall effectiveness of the system and different systems use different numbers of components (usually two, three, or four).

A Cochrane review of compression techniques in venous leg ulcers⁴ concluded that:

- Any level of compression is more effective than no compression at all
- High compression (35–45 mmHg at the ankle) is more effective than low compression (15–25 mmHg at the ankle)
- Elastic or inelastic multi-component systems are more effective than single-component compression

While multi-component systems can be made up of elastic components, they perform as an inelastic bandage with a high working pressure during exercise.² This illustrates the current difficulties in the way that bandages are classified, and the particular challenges of describing multi-component systems that may combine inelastic and elastic materials.

Limitations of compression bandages

One of the major drawbacks of compression bandaging is that a high level of skill and training are required to ensure that the bandages are applied correctly so that they deliver the appropriate level of compression. This means that with most current compression systems, patients are not encouraged to apply the compression bandages themselves. This can affect the way in which the patient is treated and it can place them in a passive position, which can in turn affect patient concordance.

The tension used when applying compression bandages can vary depending on the healthcare professional that applies the bandage.¹⁹ Bandage companies have attempted to address this issue to some degree with the introduction of geometric pattern designs on bandages that change to indicate when the correct level of tension is being used.²⁰ There are also products with at least one

component applied at full-stretch to minimize variability (e.g. 3M Coban™ 2 layer). Despite these attempts to improve compression bandages, the application of bandaging still results in variable interface pressures.¹⁹

The applied tension is very important. If the tension is too low, an insufficient pressure will be applied to counteract venous hypertension. However, if the tension is too high, pressure damage, such as tissue damage and necrosis, could occur.²⁰ Bandages that are too tight can be difficult for patients to tolerate and this can lead to problems with concordance.

In addition to these issues, the application of compression bandages can reduce ankle movement, thereby reducing patient mobility. Patients quite often suffer from malformed limbs and feet and as such bandaging the area can be problematic for the doctor or nurse applying the bandage and painful for the patient.²¹

Other compression systems

Compression can also be applied by methods other than bandaging. Compression hosiery and IPC are two such methods:

Compression hosiery (stockings)

Compression hosiery is usually used to prevent ulcer recurrence once healing has occurred, although strong hosiery is also used as first-line treatment for venous ulcers in some markets, particularly for patients with small, uncomplicated ulcers.¹⁴ There are two main types available:²²

- 1) Circular knit stockings – available in nylon and cotton yarn, unfortunately, however, they lack stretch, making them difficult to put on.
- 2) Flat knit stockings – available in nylon, cotton, and nylon-plated varieties. These are more flexible than circular knit stockings, making them easier to get on and off.

Hosiery is available in both single- and two-component systems. With two-component systems (Figure 4) the first stocking delivers a light compression (10–24 mmHg) and a second stocking sits on top, providing a stronger compression (20–30 mmHg). For some patients, the two-component hosiery systems may be easier to apply than traditional compression hosiery, thereby enabling the patient to benefit from continued wear owing to improved concordance.²³



Figure 4. A two component stocking system (SurePress® Comfort™ Pro Stocking System)

Standards are of considerable value in ensuring that specific compression hosiery products will apply a known approximate level of compression and in providing the basis for national reimbursement strategies. However there is no international standard for classifying compression hosiery and only a few national standards under which compression hosiery is classified exist. Each standard sets out different test methods (Table 7), making comparison and practical application cumbersome. Testing is conducted under standard conditions in a laboratory, and is used to classify hosiery products.

Table 7. UK, German, French, and US classification of compression hosiery

	Support level	British standard BS:6612;1985 ³	German standard RAL-GZ 387;1987 ²⁵	French standard ^{3,26}	US pressure ranges ²⁴
Testing method		HATRA	HOSY	ASQUAL	
Class 1	Light	14–17 mmHg	18–22 mmHg	10–15 mmHg	20–30 mmHg
Class 2	Medium	18–24 mmHg	23–33 mmHg	15–20 mmHg	30–40 mmHg
Class 3	Strong	25–35 mmHg	34–46 mmHg	20–36 mmHg	40–50 mmHg
Class 4	Heavy	Not reported	>49 mmHg	>36 mmHg	50–60 mmHg

Limitations of compression hosiery

There are a number of concordance issues with compression hosiery as they can be difficult to apply, especially for patients with limited mobility and dexterity, even with the use of an application aid. In warmer climates in particular, hosiery can be uncomfortable to wear. Hosiery must also be replaced after several months as it loses the ability to maintain compression.²⁷ Owing to lack of padding, it is not always practical to use hosiery with patients at high risk of pressure damage.¹⁴ Hosiery may be inappropriate for very large or wet wounds as it is difficult to apply an adequate underlayer, and for painful ulcers due to the action of pulling hosiery over the wound.¹⁵ Furthermore any edema in a limb needs to be reduced prior to fitting with hosiery.¹⁵

Intermittent Pneumatic Compression (IPC)

Intermittent Pneumatic Compression (IPC) devices consist of a 'boot' (or sleeve) of chambers that are inflated with air by an electronic pump. The inflation and deflation of the air chambers is designed to create pressure peaks that mimic the effect of the calf muscle pump.^{14, 28} There is a wide variety of IPC devices on the market, some deliver uniform pressure over the whole leg through a single air chamber, others deliver graduated pressure through sleeves with multiple chambers. Other variations include pumps that exert short bursts of high pressure to improve arterial flow²⁹ and IPC 'shoes' that are designed to mimic the foot muscle pump for patients who cannot tolerate compression over painful ulceration.

IPC has been shown to improve tissue oxygenation in patients with arterial disease,³⁰ accelerate venous return³¹, aid edema reduction³², and increase healing rates.³³ Evidence suggests that IPC can shorten the duration of therapy for vascular disease and hasten the return of a patient to active life, especially in patients who are not as mobile and as such cannot use the calf muscle pump or who cannot tolerate SGC.^{30, 34-36}

The combination of IPC and compression bandages may be more effective than bandaging alone. Fletcher *et al.* (1997) concluded that studies combining the two showed that the overall odds of healing increased when IPC was combined with sustained graduated compression (SGC).³⁷ Improved healing in VLU by using combined SGC and IPC have been reported.^{28, 33, 34}

Limitations of IPC

IPC devices, however, are perceived to be expensive and are most often used at home with the initial support of a nurse who must train the patient in the application of optimal compression pressure, inflation/deflation time, and cycle frequency. There is no single identified pressure and cycle time for treatment of venous leg ulcers. Studies conducted to date have shown positive outcomes but have used a variety of values.^{33, 34} Furthermore application of IPC can be inconvenient, leaving the patient immobile during the treatment, all of which can result in low patient concordance.³⁸

Complications of the practical application of compression therapy

While compression is considered the gold-standard treatment for venous ulceration, it is not without complications. Pain may increase in the first few weeks of compression treatment and its presence often affects patient concordance, quality of life, and sleep patterns. The WUWHS Consensus on Compression highlighted that although compression therapy is designed to reduce pain, this may not occur immediately and as such, it may cause anxiety in the patient.¹⁴ Pain control should be an aim of the treatment program despite the complexity of the patients' ulcer. In addition, it is vital that the patient is prepared for compression therapy and understands that it is the most important part of their ulcer treatment. One strategy to combat the initial adjustment to compression includes introducing the compression over a number of weeks until the patient can tolerate a high level of compression.³⁹

Compression therapy can result in pressure damage, wasting of the calf muscle, and skin problems. Patients with impaired peripheral perfusion, thin or altered limb shape, foot deformities, or dependent edema (caused by sitting for long periods of time) have an increased risk of pressure damage and action should be taken to minimize this risk. Although calf muscle wasting can occur, it is usually caused by co-morbidities and reduced mobility of the patient as opposed to a direct result of the compression therapy. Skin problems, such as maceration/excoriation owing to heavy exudate levels, dryness, itching, allergic or irritant eczema, and erosive pustular dermatosis, are associated with the application of compression therapy, topical preparations, or chronic inflammation caused by CVI.¹⁴

The ideal compression system

According to the European Wound Management Association (EWMA), an ideal compression system is one that:⁴⁰

- provides and maintains clinically effective levels of compression
- enhances the function of the calf muscle pump
- is non-allergic
- is easy to apply
- facilitates ease of training
- is conformable and comfortable
- is durable

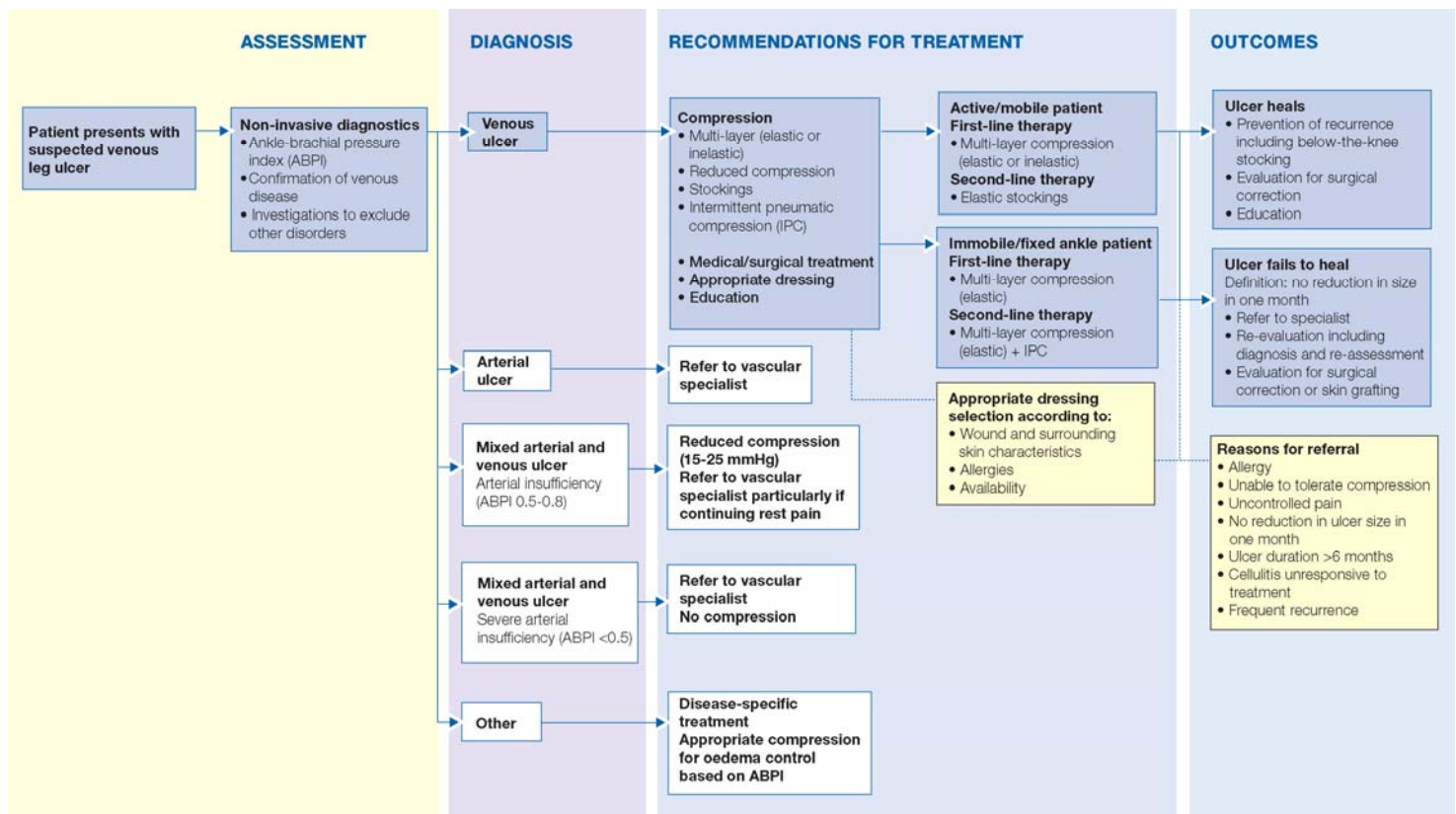
The ideal compression device would also exert a low resting pressure when a patient is lying down. When the patient then stands up, the interface pressure would be increased to counteract the increasing venous pressure. Ideally, intermittent narrowing of the veins induced by these pressure changes should occur while walking, which reduces venous reflux.¹

Furthermore, the compression system would actively involve the patient in their treatment, enhancing concordance and aiding healing.⁴¹

Treatment Algorithms for Venous Leg Ulcers

A number of treatment algorithms and guidelines exist for leg ulcers on a local and national level but, until recently, there was little international consensus on the management of venous leg ulcers. One area of agreement, however, is that compression is the cornerstone of therapy. The lack of agreement in other aspects of venous leg ulcer management prompted the development of the International Leg Ulcer algorithm by the International Leg Ulcer Advisory Panel (presented in a European Wound Management Association (EWMA) position document entitled 'Understanding Compression Therapy'). The algorithm provides a clear treatment pathway and illustrates where and when compression should be used.^{40,42} An overview of the International leg ulcer algorithm is shown in Figure 4.

Figure 4. International leg ulcer algorithm



Reproduced with permission from: European Wound Management Association. Position Document. Understanding Compression Therapy. London: MEP Ltd, 2003

The key recommendations from the International Leg Ulcer algorithm are summarized as follows:

- High compression therapy is the cornerstone for the management of venous leg ulcers
- The recommended treatment pathway highlights the accurate assessment and detailed diagnosis, in particular the assessment of the extent of arterial involvement (using ABPI) before commencing compression
- In patients with uncomplicated venous leg ulcers, decisions about which compression system to use should be based on whether the patient is mobile or immobile
- Patients require life-long compression therapy to prevent ulcer recurrence
- Patient-related and social factors, which may include treatment costs, must be taken into consideration when recommending compression therapy to achieve the best healing rates

Additionally, a group of international experts who met in June 2007 under the auspices of the World Union of Wound Healing Societies (WUWHS) has devised a management plan to assist in the clinical decision making associated with compression therapy in the treatment of venous leg ulcers. The principles presented in the *WUWHS Principles of Best Practice* document are designed to help clinicians around the world make a real difference to patient quality of life and their clinical outcomes. The document describes the factors that affect compression bandages, such as stiffness, tension, extensibility, the variety of compression bandages available, how to assess CVI, and goal setting and problem solving.¹⁴

Outcomes and Factors that Influence Healing

Venous leg ulcers can be notoriously difficult to heal and typically follow a pattern of remission and recurrence, with recurrence rates of up to 59–67%.⁴³

Randomized controlled trials designed to test the efficacy of treatments often report efficacy based upon ‘best-case scenarios’, that is, selecting patients with new venous ulcers and without other health problems. In such cases, it is possible to achieve healing results of over 70% of venous ulcers in 12 weeks ⁴⁴. When treatments are used within routine clinical practice, the outcome may be very different. For example, some studies report healing rates in the region of 30% and lower.^{45,46} Some of the reasons for this low rate of healing are discussed below.

The factors that influence healing can be broadly separated into three categories:

1. Disease related factors
2. Patient behavior related factors
3. Health service related factors

Disease related factors

Disease related factors that influence healing include the severity of the underlying pathology, size, and age of the ulcer.

Ulcers that have been present for more than 6 months and are larger than 10cm² have reduced healing rates compared with newer, smaller ulcers.^{47, 48} The increasing duration of an unhealed ulcer is also an important factor and may indicate important issues such as chronic infection, poor wound-bed preparation, or lack of appropriate treatment.

Other factors that can influence ulcer healing include poor general mobility, fixed ankle joint, history of DVT.¹⁵

Patient behavior related factors – patient concordance

Patient behavior related factors include many of the risk factors that predispose a person to developing an ulcer, concordance with compression therapy, and other social and psychology issues. Some of the factors that influence patient concordance include:

- *The patient’s knowledge, attitudes, and beliefs.* Many patients know little about their ulcer or their treatment and feel they are being “kept in the dark”.⁴⁹ A patient is more likely to persevere with treatment if they know why and how the treatment works⁵⁰
- *Level of pain and control of symptoms.* Pain control should be considered a goal of treatment⁵⁰

- *Emotional status.* Research has shown that patients with high levels of pain or clinical depression may suffer delayed healing⁵⁰
- *Desire for self-management.* The ability of patients to self-manage their treatment is highly desirable and can have a positive effect on concordance⁵⁰
- *Social pressures.* The practicalities of living with a leg ulcer and coping with compression, as well as the impact this can have on a patient's working and social life, should be handled sympathetically⁵⁰
- *Belief in treatment.* Patients that have experienced prolonged bouts of ulceration that have failed to respond to treatment will often lose faith in the treatment that is being offered.⁵¹
- *Locus of control.* Patients with an internal locus of control, which describes the way in which an individual perceives the causes behind the major events in their life, believe that they can influence their own health and will strive to use strategies that will help their ulcer to heal⁵⁰
- *Social isolation.* Social isolation can affect a patients' ability to cope with their treatment and put them at risk of developing depression⁵⁰
- *Personal relationships.* Uncontrolled odor of leg ulcers can have an impact on personal relationships and lead to social isolation⁵⁰

When properly applied, compression is an effective tool in the management of venous leg ulcers. However, inappropriate bandage selection and poor application are the most commonly reported factors that influence concordance. Concordance is a complex issue, but if a patient feels generally better about the treatment and themselves, they are more likely to adhere to the treatment.⁵⁰ It is clear that dealing with a leg ulcer involves significant suffering and psychological distress. Furthermore, health care professionals may have a negative attitude to how patients cope with leg ulcers and use blaming behavior in patients whose leg ulcer fails to heal.⁵² Practitioners can label patients as non-concordant, avoid continuity of care, and fail to address factors that are causing patient suffering. These reactions and behavior ultimately increases the level of distress that the patient feels and may reduce the rate of healing.⁵⁰

Health service related factors

The type of care a patient with a leg ulcer receives can also affect the outcome of the ulcer. A study published in 1998 compared the outcomes of ulcer treatment in UK community-based clinics treating patients with four component bandaging to those receiving usual home-based care provided by nursing services with limited access to four-component bandaging. The median time to healing was 20 and 43 weeks respectively.⁴⁵

Healing rates were higher for ulcers receiving systemic care with high compression (first ulcers healed at 12 weeks 34% vs. 24% in control), which suggests that the care provided by the community clinics resulted in better outcomes than the usual care provided by home care nursing services at the time of the study.⁴⁵

This study reflects a general move towards an increase in specialist wound centers in North America and Europe. These multidisciplinary centers can provide both out-patient clinics and inpatient ward facilities. They are recognized as experts in wound care often deliver improved rates of healing in patients with leg ulcers.⁵³ These centers are able to provide standardized treatment plans, access to relevant objective investigative assessments and surgical approaches, a high of continuity of treatment, increased patient satisfaction, potential for education and training and opportunities for clinical research.⁵³

It is important to remember that care must be taken when comparing healing rates across different sites of care and healthcare systems. It is not uncommon for patients to enter 'in and out of the system' over an extended episode of care which may effect outcome values. It is important to understand the site of care when analyzing wound healing data. ⁵⁴

In summary, although the outcome for venous leg ulcers can be improved when treated by multi-disciplinary teams from specialist centers,⁵³ there is still a high rate of ulcer recurrence,⁴³ and healing depends on many factors.⁵⁵

Post-healing and Long-Term Management of Venous Leg Ulcers

Venous leg ulcers are a chronic condition that typically follow a pattern of healing and recurrence.⁴³ To prevent ulcer recurrence, the use of compression hosiery and life-long compression therapy is recommended⁴⁰ along with regular assessment⁵⁶ and patient education.⁵⁷ Compression hosiery provides compression of 35–45 mmHg at the ankle and is the mainstay of ulcer prevention. This pressure can, however, be lowered to 25–35 mmHg for patients who struggle to apply the hosiery.⁴⁰

Assessment and education

Once an ulcer heals, it is important that a patient is still regularly assessed for changes in their medical condition or arterial status.⁵⁸ Regular assessment should be combined with education to ensure that the patient fully understands the importance of life-long compression and the early warning signs that their ulcer may be recurring.

Patient education should inform and advise patients of:⁵⁸

- the importance of compression hosiery
- appropriate skin care
- limitations and potential damage of self-treatment with over-the-counter preparations
- the need to avoid accidents or trauma to legs
- early self-referral at signs of possible skin breakdown
- benefits of mobility and exercise
- the need to elevate legs when immobile.

Other Treatment Strategies for CVI

Surgery

There are several surgical options that can be used in the management of CVI, however, they are not the treatment of choice for the disease. Surgical procedures are usually limited to the treatment of varicose veins, the treatment of ulcers caused by superficial reflux, or when non-operative attempts for VLU healing have failed in the presence of deep venous incompetence. Surgical treatment options are summarized in Table 9 below.

Table 9. Surgical treatment strategies

Procedure	Description
Surgical stripping	For treatment of varicose veins: great saphenous vein and its branches are 'stripped out' or surgically removed
Stab avulsion	For treatment of varicose veins: branch veins or affected tributary veins are surgically removed
Ligation	For treatment of varicose veins: the vein is tied off to prevent it from contributing to the general circulation
Radiofrequency ablation	For treatment of varicose veins: closure of the vein is achieved by using microwaves or an endovenous laser
Sclerotherapy	For treatment of varicose veins and telangiectases: Injection of a chemical irritant (sclerosing agent), which will eventually destroy the lumen of the vein
Valve reconstruction (valvuloplasty)	Surgery to change the shape or tightness of the valves to prevent reflux. A high degree of precision is required to repair valves that are structurally intact but incompetent

Pharmacotherapy

In some countries, venoactive drugs are used to manage common symptoms of varicose veins, such as aching, edema, and discomfort in the lower limbs. However, the mode of action of many of the drugs is unclear.⁵⁹ One example is pentoxifylline, which is used to improve blood flow in patients with circulation problems to reduce aching, cramping, and tiredness in the hands and feet. It works by decreasing the viscosity (thickness) of blood. There have been some studies conducted into the efficacy of venoactive drugs in treating leg ulceration. In a recent systematic review, pentoxifylline was reported to be more effective than placebo in achieving complete ulcer healing. Furthermore, a substantial improvement was found when used in conjunction with compression.⁶⁰ Another small study demonstrated that a flavonoid (Daflon® 500 mg Biofarma) achieved shorter healing times compared with a placebo.⁶¹

Summary

- Compression therapy is the gold-standard treatment for venous leg ulcers and is recommended as the first-line treatment strategy by the International Leg Ulcer Advisory Board and the World Union of Wound Healing Societies.
- To counteract venous hypertension, interface pressures of 35–45 mmHg at the ankle while the patient is in a supine position is recommended
- There are several different compression systems available
- Short-stretch (inelastic) compression bandages deliver high pressure when a patient is active (working pressure) and a low pressure when a patient is sitting or lying down (resting pressure)
- Long-stretch (elastic) compression bandages provide sustained compression that is not influenced by the patient's position or activity and can accommodate changes in calf shape
- Compression therapy, however, has a number of limitations
- There are several treatment algorithms that exist to guide clinicians
- Ulcer size, duration, patient mobility, and the type of care received all influence the outcome of a venous leg ulcer
- Patient concordance is affected by a number of different factors, such as pain, social pressures, and their belief in the treatment
- Compression therapy is also recommended after an ulcer has healed to prevent recurrence
- Although surgery is an option for CVI treatment, it is recommended only for a small sub-set of patients

Chapter 4

Self Assessment Questions

Please circle the letter(s) to indicate the correct answer(s)

1. Which of the following is not an outcome of compression therapy?
 - A. Reduction in venous reflux
 - B. A calf interface pressure of 60–80 mmHg when walking
 - C. Accumulation of edema
 - D. Restoration of valvular competence
2. Which of the following is incorrect with respect to edema?
 - A. Edema is a major complication in the treatment of venous leg ulcers
 - B. Starlings equation describes the relationship between fluid pressure and venous reflux
 - C. Increased pressure as a result of compression encourages reduced movement of fluid into the tissues
3. How is the pressure gradient described in a person with a venous leg ulcer using sustained graduated compression?
 - A. With the pressure highest at the ankle and then decreasing up the leg
 - B. With the pressure highest at the calf and decreasing down the leg
 - C. With pressures equal at all points
4. Which of these statements is FALSE?
 - A. Multi-component bandages are a combination of short- and long-stretch bandages and padding
 - B. An Unna boot uses an air pump
 - C. Elastic hosiery is often used to prevent recurrence
 - D. Long- and short-stretch bandages, rigid compression, and multilayer multi-component systems are all types of compression bandaging
5. When assessing effectiveness of compression bandaging, which factors need to be taken into account?
 - A. Laplace's law and venous return
 - B. Physical bandage properties and venous return
 - C. Laplace's law and physical bandage properties

6. Which of the following is FALSE when describing an ideal compression system for venous ulcer care?
- A. It should provide and maintain effective levels of compression
 - B. It should enhance calf muscle pump function
 - C. Is easy to apply
 - D. It should enhance arterial inflow
7. Which of the following is FALSE with respect to factors that effect wound healing?
- A. Patients prefer that the healthcare professional manages their treatment
 - B. The positive/negative attitude of the clinician towards the patient
 - C. The level of pain management and control of symptoms
 - D. The size and age of the patients' ulcer
8. Which surgical treatment involves removal of the branch veins or affected tributary veins?
- A. Surgical stripping
 - B. Stab avulsion
 - C. Ligation
 - D. Valve reconstruction
 - E. Radiofrequency ablation

Chapter 4

Self Assessment Answer Key

1. **C** – edema is reduced with the application of compression therapy
2. **B** – Starlings equation links tissue and fluid pressure
3. **A** – this is the case so that there is reduced pooling of blood in the limb
4. **B** – an intermittent pneumatic compression system uses an air pump
5. **C** – this ensures that properties of the compression system (as well as size and shape of limb, application of bandage, and activity undertaken) are taken into account to achieve the most effective pressure gradient in the limb
6. **D** – compression does not enhance arterial flow
7. **A**
8. **B**

References:

1. Partsch H. Compression Therapy of Venous Ulcers. *EWMA Journal*. 2006; **6(2)**:16–20.
2. Partsch H. Understanding the Pathophysiological Effects of Compression. In: European Wound Management Association (EWMA) Position Document *Understanding Compression Therapy*. London, UK: MEP Ltd; 2003.
3. Rajendran S, Rigby AJ, Anand SC. Venous Leg Ulcer Treatment and Practice – Part 3: The Use of Compression Therapy Systems. *J Wound Care*. 2007; **16(3)**:107–109.
4. Cullum N, Nelson EA, Fletcher AW, Sheldon TA. Compression for Venous Leg Ulcers. *Cochrane Database of Systematic Reviews*. 2001, Issue 2.
5. Meissner MH, Eklof B, Coleridge Smith Pet *et al*. Secondary Chronic Venous Disorders. *J Vasc Surg*. 2007;**46**:68S-83S.
6. Partsch B, Partsch H. Calf Compression Pressure Required to Achieve Venous Closure from Supine to Standing Positions. *J Vasc Surg*. 2005; **42(4)**:734–738.
7. Partsch H, Clark M, Mosti G *et al*. Classification of Compression Bandages: Practical Aspects. *Derm Surg*. 2008; **34 (5)**:600-609.
8. Moffatt CJ. Understanding Different Bandages. In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007, 32-48.
9. Meissner MH, Moneta G, Burnand K *et al*. The Hemodynamics and Diagnosis of Venous Disease. *J Vasc Surg*. 2007; **46**:4S–24S.
10. Moffatt CJ. How Compression Works. In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007,1-15.
11. Kunimoto B, Cooling M, Gulliver W, Houghton P, Orsted H, Sibbald RG. Best Practices for the Prevention and Treatment of Venous Leg Ulcers. *Ostomy Wound Management*. 2001; **47(2)**:34–50.
12. Mosti G, Mattaliano V, Partsch H. Inelastic compression increases venous ejection fraction more than elastic bandages in patients with superficial venous reflux. *Phlebology* 2008;**23**:287-294.
13. Clark M. Compression Bandages: Principles and Definitions. In: European Wound Management Association (EWMA) Position Document: *Understanding Compression Therapy*. London, UK: MEP Ltd; 2003.
14. World Union of Wound Healing Societies (WUWHS). *Principles of Best Practice: Compression in Venous Leg Ulcers. A Consensus Document*. London, UK: MEP Ltd; 2008.
15. Moffatt CJ, Martin R, Smithdale R. Bandaging and Compression Therapy. In: *Leg Ulcer Management. Essential Clinical Skills for Nurses*. Oxford, UK: Blackwell Publishing; 2007, 338-379.
16. Thomas S. The Use of the Laplace Equation in the Calculation of Sub-Bandage Pressure. *EWMA Journal*. 2003; **3(1)**:21–23.
17. Partsch H. Evidence Based Compression Therapy. An Initiative of the International Union of Phlebology (IUP). *VASA*. 2004; **34(Suppl 63)**:3–39.
18. Cornwall J. Treating Leg Ulcers. *J Dist Nurs*.1985:4-6.

19. Moffatt CJ. Variability of Pressure Provided by Sustained Compression. *Int Wound Journal*. 2008; **5**(2):259-265.
20. Thomas S. Compression Bandaging in the Treatment of Venous Leg Ulcers. *World Wide Wounds*. September 1997. <http://www.worldwidewounds.com/1997/september/Thomas-Bandaging/bandage-paper.html>. Accessed January 30, 2009.
21. Moffatt CJ. Managing the Disproportionate Limb. In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007,101-113.
22. Johnson S. Compression Hosiery in the Prevention and Treatment of Venous Leg Ulcers. *World Wide Wounds*. September 2002. <http://www.worldwidewounds.com/2002/september/Johnson/Compression-Hosiery-Leg-Ulcers.html>. Accessed January 30, 2009.
23. Ham S, Padmore J. Two-Layer Compression Hosiery for Patients with Venous Leg Ulceration. *Nurs Stand*. 2006; **20**(45):68–76.
24. Johnson JJ, Paustain C. Guideline for Management of Wounds in Patients with Lower-Extremity Venous Disease. Glenview IL: Wound Ostomy and Continence Nurses Society; 2005.
25. *Deutsches Institut für Gütesicherung und Medizinische Kompressionsstrümpfe RAL-GZ 387*. Berlin, Germany: Beuth-Verlag; 1987.
26. Certificat de Qualite-Produits. *Referentiel Technique Prescript pour les Ortheseselastiques de Contention des Membres*. Paris, France: ASQUAL; 1999.
27. *Chronic Venous Insufficiency and Venous Ulceration – Aetiology and Treatment*. ConvaTec Inc; 2008.
28. Comerota AJ. Intermittent Pneumatic Compression: Physiologics to Improve Management of Venous Leg Ulcers. *J Vasc Surg*. In press.
29. Delis KT, Knaggs AL. Duration and Amplitude Decay of Acute Arterial Leg Inflow Enhancement with Intermittent Pneumatic Leg Compression: An Insight into the Implicated Physiologic Mechanisms. *J Vasc Surg*. 2005;**42**(4):717-725.
30. Kolari PJ, Pekanmäki K, Pohjola RT. Transcutaneous Oxygen Tension in Patients with Post-thrombotic Leg Ulcers: Treatment with Intermittent Pneumatic Compression. *Cardiovasc Res*. 1988;**22**(2):138–141.
31. Labropoulos N, Cunningham J, Kang SS, Mansour MA, Baker WH. Optimising the Performance of Intermittent Pneumatic Compression Devices. *Eur J Vasc Endovasc Surg*. 2000; **19**:593–597.
32. Miranda F Jr, Perez MC, Castiglioni ML et al. Effect of Sequential Intermittent Pneumatic Compression on Both Leg Lymphedema Volume and on Lymph Transport as Semi-quantitatively Evaluated by Lymphoscintigraphy. *Lymphology*. 2001; **34**:135-141.
33. Coleridge-Smith P, Sarin S, Hasty J, Scurr JH. Sequential Gradient Pneumatic Compression Enhances Venous Ulcer Healing: A Randomised Trial. *Surgery* 1990;**108**:871-875.
34. McCulloch JM, Marlet KC, Phifer TJ. Intermittent Pneumatic Compression Improves Venous Ulcer Healing. *Adv Wound Care*. 1994;**7**:22-26.
35. Alpagut U, Dayioglu E. Importance and Advantages of Intermittent External Pneumatic Compression Therapy in Venous Stasis Ulceration. *Angiology*. 2005;**56**(1):19–23.

36. Arcelus JJ, Caprini JA, Sehgal LR, Reyna JJ. Home Use of Impulse Compression of the Foot and Compression Stockings in the Treatment of Chronic Venous Insufficiency. *J Vasc Surg.* 2001; **34(5)**:805–811.
37. Fletcher A, Cullum N, Sheldon TA. A Systematic Review of Compression Treatment for Venous Leg Ulcers. *BMJ.* 1997;**315(7108)**:576–80.
38. Allsup DJ. Use of the Intermittent Pneumatic Compression Device in Venous Ulcer Disease. *J Vasc Nurs.* 1994;**12(4)**:106–111.
39. Moffatt CJ. International Leg Ulcer Algorithm. In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007, 62-73.
40. Marston M, Vowden K. Compression Therapy: A Guide to Safe Practice. In: European Wound Management Association (EWMA) Position Document: *Understanding Compression Therapy*. London, UK: MEP Ltd; 2003.
41. Buchmann 1997. Adherence: A Matter of Self-Efficacy and Power. *J Adv Nursing.* 1997; **26**:132–137.
42. Stacey M, Falanga V, Marston W et al. The Use of Compression Therapy in the Treatment of Venous Leg Ulcers: A Recommended Management Pathway. *EWMA Journal.* 2002;**2(1)**:9–13.
43. Briggs M, Closs SJ. The Prevalence of Leg Ulceration: a Review of the Literature. *EWMA Journal.* 2003;**3(2)**:14–20.
44. Moffatt CJ, McCullagh L, O'Conner T et al. Randomized Trial of Four- Layer and Two-Layer Bandage Systems in the Management of Chronic Venous Ulceration. *Wound Repair and Regeneration.* 2003;**11**:166-171.
45. Morrell CJ, Walters SJ, Dixon S et al. Cost Effectiveness of Community Leg Ulcer Clinics: Randomised Controlled Trial. *BMJ.* 1998;**316**:1487–1491.
46. Dereure O, Vin F, Lazareth I, Bohbot S. Compression and Peri-ulcer Skin in Outpatients Venous Leg Ulcers: Results of a French Survey. *J Wound Care.* 2005;**14(6)**:265-271.
47. Franks PJ, Moffatt CJ, Connolly M et al. Factors Associated with Healing Leg Ulceration with High Compression. *Age & Ageing.* 1995; **24**:407-410.
48. Tallman P, Muscare E, Carson P, Eaglstein WH, Falanga V. Initial Rate of Healing Predicts Complete Healing of Venous Ulcers. *Arch Derm.* 1997; **133(10)**:1231-1234.
49. Douglas V. Living with a Chronic Leg Ulcer: An Insight into Patients' Experiences and Feelings. *J Wound Care.* 2001;**10(9)**:355–360.
50. Moffatt CJ. Improving Concordance with Compression Therapy. In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007, 21-31.
51. Charles H. The Impact of Leg Ulcers on Patients' Quality of Life. *Prof Nurse.* 1995; **10(9)**:571–574.
52. Moffatt CJ, Martin R, Smithdale R. Improving Concordance. In: *Leg Ulcer Management. Essential Clinical Skills for Nurses*. Oxford, UK: Blackwell Publishing; 2007, 260-280.
53. Gottrup F. A Specialized Wound-Healing Center Concept: Importance of a Multidisciplinary Department Structure and Surgical Treatment Facilities in the Treatment of Chronic Wounds. *Am J Surg.* 2004;**187**:38S-43S.

54. Ennis WJ, Fibeger E, Messner K, Meneses P. Wound Healing Outcomes: The Impact of Site of Care and Patient Stratification. *Wounds*. 2007;**19(11)**:286-293.
55. Moffatt CJ, Martin R, Smithdale R. Evaluating Outcomes of a Leg Ulcer Service. In: *Leg Ulcer Management. Essential Clinical Skills for Nurses*. Oxford, UK: Blackwell Publishing; 2007,338-379.
56. Moffatt CJ. Preventing Ulcer Recurrence with Compression Hosiery. In: *Compression Therapy in Practice*. Aberdeen, UK: Wounds UK Publishing; 2007, 21-31.
57. Moffatt CJ, Martin R, Smithdale R. Prevention of Recurrence and Patient Evaluation. In: *Leg Ulcer Management. Essential Clinical Skills for Nurses*. Oxford, UK: Blackwell Publishing; 2007, 397-415.
58. Royal College of Nursing. *Clinical Practice Guidelines. The Nursing Management of Patients with Venous Leg Ulcers. Recommendations*. www.rcn.org.uk/__data/assets/pdf_file/0003/107940/003020.pdf. September 2006. Accessed January 23, 2009.
59. Katsenis K. Micronized Purified Flavonoid Fraction (MPFF): A Review of its Pharmacological Effects, Therapeutic Efficacy and Benefits in the Management of Chronic Venous Insufficiency. *Curr Vasc Pharmacol*. 2005; 3:1–9.
60. Jull A, Waters J, Arroll B. Pentoxifylline for Treatment of Venous Leg Ulcers: A Systematic Review. *Lancet*. 2002; **359**:1550–54.
61. Coleridge Smith PD. The Drug Treatment of Chronic Venous Insufficiency and Venous Ulceration. In: Gloviczki P, Yao JST, eds. *Handbook of Venous Disorders*. 2nd ed. New York, NY: Oxford University Press Inc; 2001:309-318.

Surepress Comfort is a trademark of ConvaTec Inc.
All other trademarks are the property of their respective owners

Chapter 5

Glossary of Terms

actin	A protein found in cell structures and active in muscular contraction, cellular movement, and maintenance of cell shape.
ambulatory venous hypertension (AVH)	An abnormally high intravenous pressure when an individual exercises.
ambulatory venous pressure (AVP)	Blood pressure in the veins during exercise.
angiogenesis	Growth of blood vessels.
ankle brachial pressure index (ABPI)	Non-invasive test used routinely to identify the extent of arterial disease in the legs. Also known as the ABI (Ankle Brachial Index).
antigen	Any substance recognized by the body as foreign that evokes an immune response either alone or after forming a complex with a larger molecule.
arteries	Blood vessels that carry oxygenated blood away from the heart.
arterioles	Small arteries that branch off main arteries. Connect to the capillaries.
ascending venography	A standard test for assessing acute DVT or an obstruction.
atherosclerosis	A hardening and narrowing of the arteries that leads to restriction of blood flow.
atrophie blanche	A condition where small smooth ivory-white areas of tissue with reduced blood supply develop. Typically have hyperpigmented borders with telangectases.
atrophy	Decrease in size or wasting away of a body part or tissue.
autolytic debridement	Removal of lacerated, devitalized, or contaminated tissue by self-produced enzymes.
blanched skin	Pale skin due to lack of blood flow or supply.

calcification	Calcium deposit on tissue (usually resulting in tissue hardening).
capillaries	Very small blood vessels that connect the arteries to the veins.
CEAP Classification	Classification for chronic venous disease (CVD), originally developed in 1994 by an international <i>ad hoc</i> committee of the American Venous Forum, endorsed by the Society for Vascular Surgery, and incorporated into "Reporting Standards in Venous Disease" in 1995. A consensus report revising some of the classification criteria was published in 2004.
cellulitis	Inflammation of the subcutaneous tissue.
chemoattractants	Small molecules made by body cells to attract the migration of other cells.
chronic venous disease (CVD), chronic venous insufficiency (CVI)	Disease of veins in the legs that reduces the return of blood to the heart. Mainly caused by incompetent venous valves and/or vein blockage ("obstruction") by blood clot (deep vein thrombosis). CVD is characterized by varicosities, skin changes and edema. Severe CVD/CVI is characterized by damage to the skin and ulceration, usually in the lower limb.
deep vein thrombosis (DVT)	Blood clot in the deep veins.
dermis	The inner layer of skin immediately beneath the epidermis.
distal	Farthest from the trunk of the body.
dorsalis pedis artery	Dorsal (upper aspect) artery of the foot. One of the main arteries supplying blood to the foot.
duplex scanning, or ultrasonography	A noninvasive test that uses ultrasound waves to obtain a picture of the veins and determine the speed and direction of blood flow within the vein.
edema (or oedema)	Swelling from excessive accumulation of fluid in body tissue. C3 in the CEAP Classification.
endothelial cells, endothelium	Cells that line the internal surface of blood or lymph vessels, forming the endothelium. The inner lining of blood and lymph vessels.
epidermis	The surface cellular layer (epithelium) of skin.
epithelialization	The process of forming the epithelium by growth and migration of epithelial cells.

epithelial regeneration	The process of reforming the epithelium after loss or damage.
epithelium	A membranous cellular tissue consisting mostly of epithelial cells that covers a free surface or lines a tube or cavity of an animal body.
erythema	Abnormal redness of the skin.
eschar	A scab formed over a wound.
extravasation	The process of substances or cells forced out or caused to escape from a proper vessel or channel, such as a blood vessel.
extravascular	Outside of the blood vessels.
exudate	The material composed primarily of serum, fibrin and white blood cells that escapes from blood vessels into a superficial lesion, area of inflammation or a wound. May also contain cellular debris.
fibrin	Insoluble fibrous protein formed from fibrinogen by the action of thrombin especially in the clotting of blood.
fibroblasts	One of the main populations of cells in connective tissue that contribute to synthesis and organisation of the protein and carbohydrate materials found in connective tissue.
fibronectin	A group of glycoproteins (proteins with carbohydrate attachments) of cell surfaces, blood plasma, and connective tissue that promote cellular adhesion and migration.
fibrosis	A condition marked by increase of interstitial ("between parts or components") fibrous tissue.
gaiter area	The area of the leg that lies between the ankle and the knee.
gangrene	Local death of soft tissues due to loss of blood supply.
growth factors	Small protein molecules made by various body cells to stimulate growth or migration of other cells.
hemostasis	Stoppage of bleeding.
hyaluronan	Large carbohydrate molecule found in extracellular matrix, known to have major functions in many biological processes (also known as hyaluronic acid).
hyperpigmentation	Discoloring (darkening) of the skin. C4a in the CEAP Classification

hypertrophic Scar	An elevated scar resembling a keloid but which does not spread into surrounding tissues, is rarely painful, and regresses spontaneously. Often associated with burn injury (see also keloid).
hypoxia	A state of insufficient oxygen supply to tissue to support the normal function of that tissue.
interface pressure (or sub-bandage pressure)	The term used to describe the level of compression delivered by a device. Pressure between the compression device and the skin.
intermittent claudication	Leg pain when walking, usually indicative of arterial disease.
intermittent pneumatic compression (IPC)	A type of compression system which consists of an inflatable boot or sleeve connected to a pump. Chambers in the sleeve sequentially fill with air and apply pressure cyclically, essentially mimicking the action of the foot and calf muscle pump.
intermittent venous occlusion	Temporary complete vein lumen closure brought about by active calf muscle pump on exercise.
intravenous pressure	The pressure of the blood within a vein, the venous pressure.
ischemia	A low oxygen state usually due to obstruction of the arterial blood supply or inadequate blood flow leading to hypoxia in the tissue.
ischemic	Relating to or affected by ischemia.
ischemic Ulcers	Ulcers that occur (usually in feet) due to ischemia.
isotonic fluid	Fluid having equal osmotic pressure (in this context, in relation to blood).
keloid	A thick scar resulting from excessive growth of fibrous tissue and occurring especially after burns or radiation injury, likely to spread and does not regress spontaneously.
keratinocytes	A population of skin cells that make up the top layer of skin (epidermis). Epithelial cells.
lateral	Away from the center of the body on its vertical axis (see also medial).
lateral malleolus	The process at the lateral side of the lower end of the fibula, forming the projection of the lateral part of the ankle.
leg ulcer or leg ulceration	A chronic leg wound that fails to heal. C6 in the CEAP Classification

leukocytes	White blood cells.
ligation	Tying off a blood vessel preventing it from contributing to the general circulation (e.g. in a procedure to treat varicose veins whereby the vein is tied off).
lipodermatosclerosis	A state of chronic venous insufficiency characterized by induration (hardening) of the skin from fibrosis of subcutaneous fat. Hyperpigmentation often accompanies lipodermatosclerosis. C4b in the CEAP Classification
macrophages	A population of white blood cells important in inflammation and body defense (see also neutrophils).
malleolus	A projection at the distal end of each bone of the leg at the ankle joint. The bony part of the ankle that protrudes slightly.
medial	Towards the center of the body on its vertical axis.
medial malleolus	The process at the medial side of the lower end of the tibia, forming the projection of the medial side of the ankle.
metatarsophalangeal joint	The articulation between a metatarsal bone and a phalanx in the foot.
mycobacterial	Relating to a genus of bacteria of the family <i>Mycobacteriaceae</i> that are usually slender, difficult to culture and stain and difficult to detect by conventional microscopy.
myofibroblasts	Fibroblasts that have developed some of the functional and structural characteristics (as the presence of actomyosin structures) of smooth muscle cells.
myosin	A fibrous protein that reacts with actin to form actomyosin and provides the mechanical force for muscular contraction, cellular movement, and maintenance of cell shape.
necrosis	Cell or tissue death.
necrotic tissue	Dead or non-viable tissue.
neuropathy	Disease of nerve tissue.
neutrophils	A population of white blood cells important in inflammation and body defense (see also macrophages). Also known as polymorphonuclear neutrophils (PMNs) because of the irregular shape of the nucleus. Play a key role in defense against infection.
non-pitting (o)edema	Edema that cannot easily be indented by pressure.

occlusive dressings	A dressing that seals a wound from fluid loss and limits gas/vapor transmission.
orthograde blood flow	Forwards blood flow. In a vein, this is towards the heart.
periwound skin	Skin surrounding the wound.
phagocytic cells	Cells in the process of (or have the ability to) phagocytosing particles such as bacteria, other microorganisms, aged red blood cells, foreign matter, etc.
phagocytosing	The process by which a cell engulfs particles such as bacteria, other microorganisms, aged red blood cells, foreign matter, etc.
phalangeal heads	Toe joints.
pitting (o)edema	Edema that, for a time, retains the indentation produced by pressure.
platelets	Tiny cells that circulate in the blood whose main function is to take part in the clotting process.
plethysmography	Method for determining and registering variations in the size of an organ or limb resulting from changes in the amount of blood present or passing through it. Measurements can be achieved by various means, e.g. photoelectric plethysmography, air plethysmography.
polymicrobial	Involving multiple types of bacteria.
polymorphonuclear neutrophils (PMNs)	See neutrophils .
polysaccharides	Chemical compounds with many sugar molecules joined together in a linear fashion.
posterior tibial artery	One of the main arteries supplying the foot.
postphlebotic or post-thrombotic syndrome (PTS)	A chronic condition that develops in 20% to 50% of patients after deep venous thrombosis. Patients with PTS experience pain, heaviness, swelling, cramps, itching, or tingling in the affected limb, and may ultimately suffer ulceration. Combinations of symptoms may vary from patient to patient.
pressure gradient	The rate of decrease (gradient) of pressure applied by a compression system between the ankle and the knee.

pressure ulcers	Ulceration of tissue deprived of adequate blood supply by prolonged pressure, called also <i>decubitus</i> , <i>decubitus ulcer</i> , <i>pressure sore</i> . Note that other factors such as shear stress and maceration are also recognized contributors to pressure ulcer formation.
proteoglycan	Large molecule typically found in extracellular matrix composed of both protein (proteo-) and carbohydrate (glycan) components, with the latter as the major component. (Glycoprotein – similar but with protein as the major component)
proteolytic enzymes	Enzymes that catalyze the breakdown of proteins.
purpuric	Pertaining to small purple spots or patches on skin caused by small areas of hemorrhagia.
pyoderma gangrenosum	A chronic noninfectious condition that is marked by the formation of purplish nodules and pustules which tend to coalesce and form ulcers and that is associated with various underlying systemic or malignant diseases.
radiofrequency ablation	A procedure to treat of varicose veins whereby the vein is closed by using microwaves or an endovenous laser.
re-epithelialization	The process in which keratinocytes migrate and proliferate over the wound bed to form the new epithelium (epidermis), typically signaling the healing of the wound.
refilling time (RT)	Time (seconds) that it takes for a vein to refill with blood after muscular contraction - an important measure of reflux severity.
resting pressure	A continuous force exerted externally from a bandage towards the tissue in a resting patient.
retrograde flow or reflux	Venous blood flow backwards (the wrong way; away from the heart).
rheumatoid arthritis	An autoimmune disease which causes chronic inflammation of the joints, the tissue around the joints, as well as other organs in the body.
sclerotherapy	A procedure to treat of varicose veins whereby the vein is injected with a chemical irritant (sclerosing agent), which will eventually destroy the lumen of the vein.
sebaceous glands	Glands in the skin that produce sebum (fatty lubricant matter).

sickle cell anemia	A genetic disease involving abnormally shaped red blood cells, which then have difficulty circulating properly through the body.
slough	Dead tissue separating from living tissue.
stab avulsion	Surgical procedure whereby the branch veins or affected tributary veins are surgically removed - for treatment of varicose veins.
static stiffness index (SSI)	The change in sub-bandage interface pressure that occurs when a patient moves from a lying to a standing position.
stratum corneum	The outer part of the epidermis consisting mostly of layers of dead flattened non-nucleated cells filled with keratin.
surgical stripping	Surgical procedure whereby the great saphenous vein and its branches are 'stripped out' or surgically removed - for the treatment of varicose veins.
sustained graduated compression (SGC)	Compression of the limb which creates a pressure gradient where the pressure is highest at the ankle and diminishes up the leg. Sustained over a prolonged period, over several days.
telangiectasia	Spider or web veins – an early sign of chronic venous insufficiency. C1 in the CEAP Classification.
thalassemia	An inherited form of anemia caused by faulty synthesis of haemoglobin.
thrombosis	Blood clotting within blood vessels.
ultrasonography, or duplex scanning	A noninvasive test that uses ultrasound waves to obtain a picture of the veins and determine the speed and direction of blood flow within the vein.
valvuloplasty	Valve reconstruction surgery to change the shape or tightness of the valves to prevent reflux.
varicose veins	Dilated, snake-like segments of superficial veins appearing as twisted, dark blue vessels just below the skin. C2 in the CEAP Classification
vasculitis	Inflammation of a blood or lymph vessel.
vasoconstriction	Narrowing of blood vessels by contraction of the blood vessel wall.
veins	Blood vessels that <i>return</i> deoxygenated blood to the heart.
venous hypertension	Abnormally high blood pressure in the vein.

venous pressure	The weight of the blood column between the foot and right atrium of the heart (expressed as mm Hg).
venules	Small veins connecting the capillaries with the larger systemic veins.
viscosity	The property of resistance to flow in a fluid or semifluid.
working pressure	An intermittent pressure exerted by a combination of the active muscle working against the resistance of the bandage.