



**Leg ulceration in venous and arteriovenous insufficiency assessment and management with compression therapy as part of a holistic wound-healing strategy**



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# Introduction

This international consensus document presents the outcomes of an expert panel discussion, convened in October 2023. The discussion aimed to provide best-practice recommendations on the assessment and management of venous and arteriovenous leg ulcers. To this end, the panellists explored the accurate assessment of the venous and arterial aetiologies underlying leg ulceration, as well as the optimal safe and effective management of venous or arteriovenous ulceration using compression therapy as part of a holistic care plan. This consensus document is intended to complement existing published guidance on the management of venous ulceration and use of compression therapy,<sup>1-4</sup> primarily by filling gaps in earlier guidelines on the assessment and management of leg ulcers with a mixed aetiology caused by combined arterial and venous insufficiency (CAVI).

This consensus document has been written for a multidisciplinary readership of generalist and specialist health professionals, including physicians, podiatrists, nurses and allied health professionals, such as physical and occupational therapists. It aims to be inclusive and international, with a relevance to all healthcare settings and consideration for variations in practice; access to resources; and the way services are designed, provided and reimbursed in the medical systems throughout the world. It is hoped that the recommendations in this consensus document will provide health professionals with the skills and confidence to accurately assess chronic venous and/or arterial insufficiency and deliver compression therapy in a timely, safe and effective manner.

## Background

Chronic venous insufficiency (CVI), CAVI and resulting leg ulceration have a significant negative impact on quality of life. The global cost of CVI with and without ulceration is in the billions of dollars.<sup>5</sup> The reported global prevalence of CVI varies from <1 to 17% in men and <1 to 40% in women; 1–2% of the global adult population have a lower-extremity wound (leg ulcer), with the prevalence increasing to 3% for patients over 65 years old.<sup>5</sup> According to a 2023 meta-analysis, venous ulceration has an international pooled prevalence of 0.32% and incidence of 0.17%.<sup>6</sup>

The above statistics may be an underestimate of the real world burden due to small sample sizes, misdiagnosis and underreporting, especially of people outside of care, patients who self-treat their wounds and those in less-developed nations.<sup>6</sup> In addition, prevalence and incidence studies do not always include wounds that are treated without adequate

diagnosis.<sup>6</sup> Prevalence and incidence statistics may also be influenced by factors such as delays in detection and diagnosis, as identified by a 2022 study in primary care, where the median time after first appearance of a hard-to-heal wound was 8 days to first assessment but 41 days to diagnosis.<sup>7</sup> Real-world prevalence would be better understood with higher quality population studies using consistent methods for population data collection and database analysis, for example using a compliant commercial database such as the Blue Health Intelligence research database of administrative claims.<sup>8</sup>

## Structure

This international consensus document begins by summarising the potential for venous, arterial, arteriovenous and atypical aetiologies underlying leg ulceration. It then explains how these aetiologies can be assessed and diagnosed with a full holistic patient assessment. Recommendations are presented on how the results of a patient assessment should be used to indicate the safety and recommended application of compression therapy as part of a holistic wound-healing strategy. The document then describes the different types of compression systems with reference to the main functional characteristics of pressure, elasticity and stiffness. Guidance is offered on product selection, application technique and long-term maintenance for compression therapy. The last section surveys other aspects of the holistic management of patients receiving compression therapy in CVI or CAVI, including wound and skin care, exercise and supported self-management, as well as adjunctive therapies, revascularisation and patient and professional education.

Where possible, the recommendations presented in this document are informed and supported by citations of the best available published evidence. A narrative review of the literature was conducted on the electronic databases PubMed, ScienceDirect and Google Scholar, using keywords relating to the aetiologies, tools and interventions discussed. The cited literature includes level 1 (systematic reviews or meta-analyses) to level 4 (case-control or cohort studies) publications, alongside grey literature. Other recommendations based on the expert opinion, professional experience and clinical judgement of the consensus panel without reference to published literature have been presented under the label 'consensus statement'. The label 'consensus statement' denotes expert opinion and has no bearing on significance compared with the rest of the document. The full text has been read, discussed, edited and agreed by the panel prior to publication of the document.

# Aetiologies

Leg ulcers typically have a venous, arterial or mixed arteriovenous aetiology, although a smaller proportion have an atypical aetiology (*Box 1*).<sup>9-11</sup> A study of more than 31 000 patients with hard-to-heal (chronic) leg ulcers found that 47.6% were venous, 14.5% were arterial and 17.6% were arteriovenous.<sup>12</sup>

## Chronic venous insufficiency

Venous leg ulcers (VLUs) are caused by CVI, which refers to more severe, symptomatic classes of chronic venous disease (CVD).

Early-stage CVD commonly involves telangiectasia, which are small red vessels or spider veins on the lower leg as a result of

capillary dilation. Telangiectasia is a cosmetic feature that in isolation does not require treatment. Early progression of CVD may involve the appearance of varicose veins (enlarged, tortuous veins), which are often cosmetic and asymptomatic and thus do not require treatment. However, varicose veins may lead to symptoms such as aching, heaviness, itching or pain in the legs, which do require intervention and treatment.<sup>13</sup>

CVI involves lasting functional abnormalities of the veins that typically affect the leg, such as vein or valve incompetency or vein obstruction. These pathological changes in the venous structure commonly lead to venous reflux, inadequate venous return and, ultimately, venous hypertension. Inadequate venous return can also be as a result of an impairment or malfunctioning of the foot and calf pump.<sup>14-16</sup> The calf pump is a rhythmical contraction of the calf muscles during ambulation or movement of the ankle that facilitates upward blood flow (venous return). The muscle contraction applies pressure onto the veins, acting like bellows, forcing the blood to move from the superficial veins via the perforators into the deep venous system, aiding venous return (*Figure 1*). An impaired calf venous pump can contribute to oedema and venous hypertension, leading to venous ulceration and poor wound healing.<sup>17</sup>

Vein obstruction in CVI may result from deep vein thrombosis (DVT). DVT can result in post-thrombotic syndrome. Blockage and subsequent damage to the deeper veins lead to venous hypertension. Over time, this can result in leakage of proteins and fluid into the interstitial tissue spaces, which causes inflammation and eventually breaks down, leading to venous ulceration. There is a risk of a DVT when there is a change in mobility status or an underlying hypercoagulation disorder.

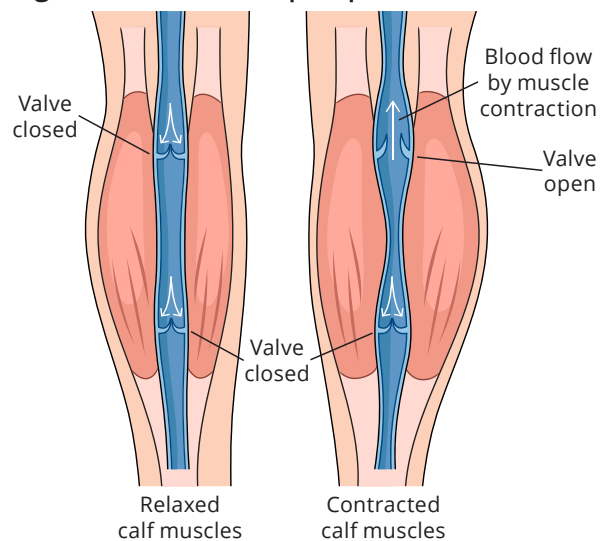
### Box 1. Atypical aetiologies for leg ulcers

Less common causes of wounds on the lower leg include (but are not limited to) the following:

- Autoimmune disorders
- Basal cell carcinoma
- Calciphylaxis
- Hydroxyurea ulcer
- Marjolin ulcer
- Martorell's ulcer
- Medication-induced ulceration
- Necrobiosis
- Post-COVID necrosis
- Pyoderma gangrenosum
- Scleroderma
- Sickle cell disease
- Squamous cell carcinoma
- Vasculitis

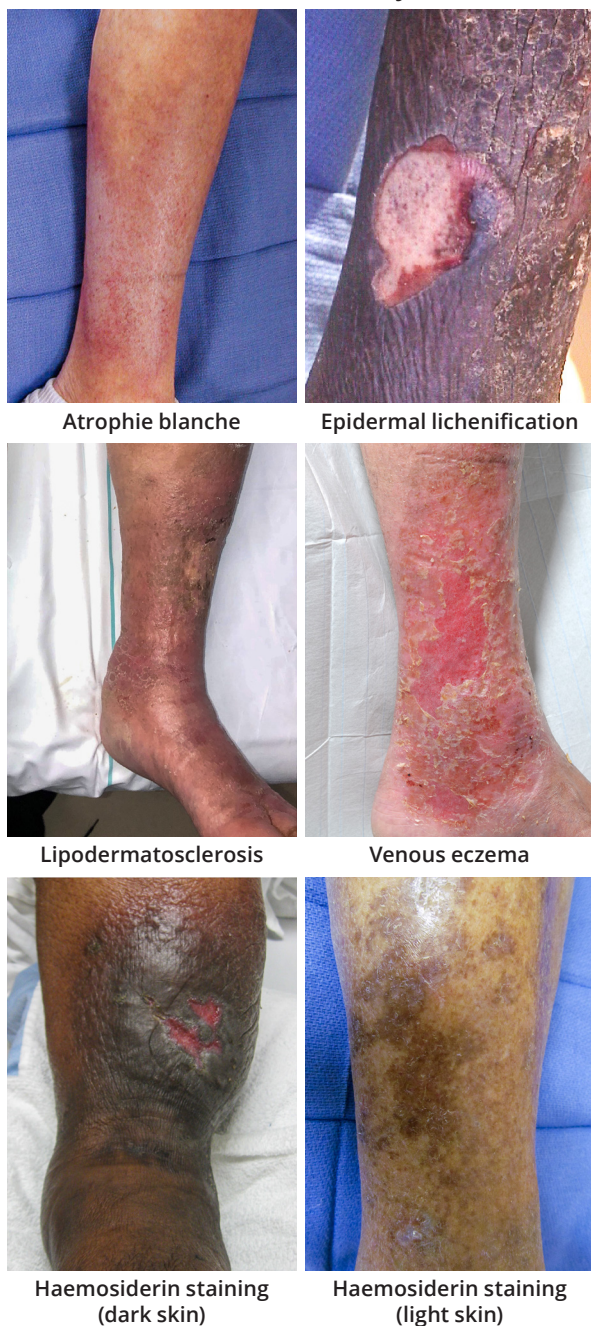
As with all leg ulcers, an accurate diagnosis is necessary to facilitate treatment of the underlying disease. Leg ulcers presenting as atypical still require vascular screening to rule out significant arterial involvement. Compression therapy may also still be beneficial, even if venous hypertension is not obvious. Leg ulcers with an atypical aetiology are hard to diagnose and are commonly misdiagnosed as venous, arterial or arteriovenous ulcers. Warning signs of an atypical aetiology include atypical presentation of the wound bed (e.g., depth, hypergranulation), wound location (e.g., calf, heel, malleoli) or periwound skin colour (e.g., purple edges or halo around the wound, which may be harder to see in dark skin tones), as well as a pain level disproportionate to the wound's appearance. Other signs where an atypical aetiology should be considered include failure to respond to best-practice care and red flags in the patient's history, such as frequent recurrence, wound history linked to a medical event (e.g., starting new medication) or systemic disorders/autoimmune conditions (e.g., rheumatoid arthritis, Crohn's disease or systemic lupus erythematosus).<sup>10</sup>

Figure 1. Calf muscle pump



CVI can overload the lymphatic system and lead to lymphatic insufficiency. In CVI, raised venous filtration initially increases lymphatic transport, which eventually overloads lymphatic capability, increasing fluid infiltration into the extracellular spaces and resulting in chronic secondary venous lymphoedema (phlebolymphoedema).<sup>18</sup> Phlebolymphoedema may be pitting or non-pitting, depending on the stage of lymphoedema and degree of tissue fibrosis (hardening of the skin and subcutaneous tissue where excess fluid accumulates).<sup>19</sup>

**Figure 2. Pathological skin changes linked to chronic venous insufficiency**



Untreated CVI is usually progressive and leads to a state of chronic inflammation, which is often more pronounced when combined with lymphatic insufficiency.

This can lead to visible pathological changes to the skin (Figure 2).<sup>20-24</sup>

- Atrophie blanche, blanched plaques in the skin due to occlusion of the small vessels in the middle and deep dermis – these plaques are frequently found at the base of the toes but can be found elsewhere – they are typically white in lighter skin and are paler but harder to see in dark skin (atrophie blanche may be misdiagnosed as livedoid vasculopathy, which is characterised by pain)<sup>25</sup>
- Epidermal lichenification, thick bark-like scaling as a result of hyperpigmentation due to keratin overproduction, which may have ulceration underneath<sup>26</sup>
- Haemosiderin staining, discolouration of the skin because of autolysis of red blood cells trapped in the interstitial spaces with resultant release of iron into the tissue
- Lipodermatosclerosis, subcutaneous fibrosis and skin induration caused by the chronic inflammation associated with venous hypertension
- Venous eczema, a non-infective inflammatory skin condition that affects the lower limbs and is a direct result of venous insufficiency (also known as venous dermatitis, varicose eczema, stasis eczema or gravitational eczema).

In the most severe cases, CVI results in venous ulceration. VLU are typically large, shallow wounds with sloped, uneven (serpentine) edges, and they are characteristically located on the gaiter area (lower third of leg above the malleoli). CVI is the main cause of venous leg ulcers (VLUs), with around 47.6% of all leg ulcers being caused by CVI alone.<sup>12</sup>

Venous hypertension can be significantly exacerbated by morbid obesity.<sup>27</sup> The risk factors for CVI are given in Box 2.

**Box 2. Risk factors for chronic venous insufficiency<sup>20,198,199</sup>**

- Advanced age
- Diminished ankle range of motion (e.g., history of ankle injury)
- Family history of venous disease, including varicose veins
- Flat feet
- Genetic conditions<sup>20</sup>
- History of deep vein thrombosis
- History of hernia surgery in men
- History of multiple pregnancies
- History of smoking
- Limited mobility or immobility
- Obesity and increased waist circumference
- Pro-thrombotic clotting disorders
- Vascular malformations

## Peripheral arterial disease

Arterial ulcers are the result of severe peripheral arterial disease (PAD). PAD is a condition caused by atherosclerosis or plaque buildup that reduces the flow of blood in peripheral arteries, with subsequent tissue hypoperfusion. Mild or moderate PAD may be asymptomatic. Moderate PAD has two main clinical features:

- Diminished or absent pedal pulses<sup>28</sup>
- Intermittent claudication, a sudden-onset pain, cramping or fatigue in the calf muscles that occurs with a predictable amount of physical activity (due to increased oxygen demand in the muscles) and subsides after cessation of exercise (as the oxygen requirement of the muscle reduces); it often forces the person to slow or stop after walking a distance relative to the severity of arterial insufficiency<sup>29</sup>

Severe PAD has the following main features:<sup>30,31</sup>

- Arterial ulceration, presenting as deep, punched-out wounds typically on the foot or toes
- Gangrene (tissue loss or necrosis) on the foot and especially the toes
- Ischaemic rest pain, defined as an often constant, severe pain in the distal extremity or foot, which is made worse by elevation of the leg and can be relieved slightly by lowering the limb into a dependent position.<sup>32</sup>

More-severe cases of PAD are more likely to involve the following additional symptoms:

- Cold limb, where the leg is relatively cold to the touch
- Hair loss, on the lower leg and foot
- Severe intermittent claudication (determined by impact on quality of life and treadmill walking distance, which may be less than 200 m)<sup>30</sup>
- Skin atrophy, with the distal limb having a thin, smooth and shiny appearance
- Thickened and discoloured toenails, caused by insufficient oxygen to the nail bed and often involving fungal infection (onychomycosis).<sup>33</sup>

The most severe chronic manifestation of PAD is known as chronic limb-threatening ischaemia (CLTI), defined by the diagnostic criteria given in *Box 3*.<sup>18,34,35</sup> In the US, CLTI has an annual incidence of 0.35% and prevalence of 1.33%. Up to 10% of patients with PAD may have CLTI, and 5–10% of patients with asymptomatic PAD or intermittent claudication will progress to CLTI over a 5-year period.<sup>36</sup>

### Box 3. Diagnostic criteria for chronic limb-threatening ischaemia<sup>34,35</sup>

**At least one of the following symptoms of more than 2 weeks' duration:**

- Ischaemic rest pain
- Tissue loss (gangrene or ulceration)

**At least one of the following objective signs of peripheral arterial disease:**

- Ankle pressure <50 mmHg
- Ankle brachial index <0.4
- Toe pressure <30 mmHg
- Transcutaneous partial pressure of oxygen (TcPO<sub>2</sub>) <30 mmHg
- Flat or minimal pulsatile waveforms

Risk factors for PAD include older age and smoking, as well as CVD, renal disease, hypertension and diabetes,<sup>31</sup> which is reported in up to 50% of people with PAD.<sup>34,37,38</sup>

## Combined arterial and venous insufficiency

Arteriovenous ulcers are defined by the simultaneous presence of both CVI and mild-to-moderate PAD, which are together termed combined arterial and venous insufficiency (CAVI). A 2016 study showed that 10–18% of all leg ulcers are a result of CAVI, 17.3% of patients with CVI had some degree of PAD, and PAD was more frequent in patients with severe CVI.<sup>39</sup> Another study found that 21% of patients with PAD had co-existing CVI.<sup>40</sup>

In CAVI (and arteriovenous ulceration), the arterial involvement is always mild-to-moderate, and the venous element is predominant.<sup>41</sup> These presentations must be distinguished from the combination of CVI and severe PAD, which should instead be understood as a predominantly arterial condition with a secondary venous involvement. This distinction will determine whether compression therapy can be safely used without prior successful revascularisation.

**Consensus statement:** The term 'CAVI' is preferable to the more widely used 'mixed-aetiology', which is less specific and may infer several other diagnoses, such as malignancy, vasculopathy or vasculitis.

# Assessment

The continuum of care begins with those patients who are at risk of developing venous and/or arterial disease. Patients with signs of potential venous and/or arterial disease, including undiagnosed ulceration on their leg, should undergo a full holistic clinical assessment of the individual, their limb and any ulceration.<sup>42</sup> Ideally, an initial assessment should be performed as promptly as possible, within 14 days of presentation and earlier in those with recurrent ulceration, severe oedema or high exudate levels.<sup>42</sup>

The primary aim of initial assessment is to accurately diagnose the aetiology underlying the patient's presentation. An accurate assessment of the underlying pathology and its severity is crucial to establishing an evidence-based plan of care and determining the safety and appropriateness of interventions.

There are several clinical assessments that can be used to compare the patient's presentations with the typical clinical features of a venous or arterial aetiology. Individual presentations often occur on a continuum between typically venous or arterial characteristics, with presentations exhibiting significant aspects of both conditions, suggesting a mixed aetiology. However, it should be noted that presentations often vary between individuals and may not precisely align with the

characteristics expected for their condition. Moreover, clinical features associated with venous or arterial insufficiency may be coincidental and result from other factors.

**Consensus statement:** Bedside examination and screening may provide a provisional working diagnosis, but a firm diagnosis of CVI, CAVI or PAD (and staging of severity) requires specialist assessment with a combination of relatively reliable and accurate metrics.

Assessment should be carried out by a health professional with the appropriate training, competency and experience. This may require referral to another service where specialist vascular personnel and equipment are available. Health services in resource-limited settings may not have access to all the diagnostic equipment, specialist training and recommended referrals; therefore, they may need to rely on the tools available to assess the patient and initiate therapy.

**Consensus statement:** Assessment is an ongoing process, and the patient's condition should be regularly assessed throughout treatment and follow up.

**Consensus statement:** During initial assessment and ongoing monitoring, consistent standardised tools and techniques for measurement, classification and documentation are essential to track progress and adjust the care plan as needed to obtain optimal patient outcomes (*Box 4*). CVD, including CVI, is most commonly classified using the CEAP Classification of Venous Disorders (*Table 1*)<sup>13</sup> and PAD using the Fontaine Classification (*Table 2*).<sup>30</sup>

## Box 4. Standardised assessment tools

### CEAP Classification of Venous Disorders

The CEAP Classification of Venous Disorders uses clinical, aetiological, anatomical and pathophysiological descriptors to classify the severity and type of venous disease. It serves as a basis for systematic investigations, orderly documentation and appropriate decisions on treatment and interventions, including the type and level of compression (*Table 1*).<sup>13,200,201</sup>

### Fontaine Classification

The Fontaine Classification categorises arterial insufficiency based on symptomatic presentation into four stages, with stage II divided into stage IIA and stage IIB (*Table 2*).<sup>30</sup>

### Venous Clinical Severity Score

The Venous Clinical Severity Score (VCSS) scores pain, varicose veins, venous oedema, skin pigmentation, inflammation and induration as 1 (mild), 2 (moderate) or 3 (severe), as well as the number, duration and size of ulcers and patient use of compression. The VCSS provides longitudinal information for ongoing assessment and prognosis of how venous disease is advancing towards ulceration or healing.<sup>202</sup>

### Wifl Classification

The Wifl Classification system for the threatened lower limb is based on the three main factors that impact limb amputation risk: wound, ischaemia and foot.<sup>203</sup>

**Table 1. CEAP Classification of Venous Disorders<sup>13</sup>**

Class	Description
C0	No visible or palpable signs of venous disease
C1	Telangiectasias or reticular veins
C2	Varicose veins
C2r	Recurrent varicose veins
C3	Oedema
C4	Changes in skin and subcutaneous tissue secondary to venous disease
C4a	Pigmentation or eczema
C4b	Lipodermatosclerosis or atrophie blanche
C4c	Corona phlebectatica
C5	Healed venous ulcer
C6	Active venous ulcer
C6r	Recurrent active venous ulcer



**Table 2. Fontaine Classification for peripheral arterial disease<sup>30</sup>**

Stage	Description
Stage I	Asymptomatic, incomplete blood vessel obstruction
Stage IIA	Mild claudication at a distance of >200m
Stage IIB	Claudication at a distance of <200m
Stage III	Rest pain, mostly in the feet
Stage IV	Necrosis and/or gangrene of the limb

## Vascular assessment

Patients presenting with leg ulceration should undergo arterial assessment within 14 days to screen for the presence of arterial insufficiency (Table 3).<sup>43</sup> Arterial assessment may begin with the following fast and accessible tests:

- Pedal pulse palpation, where a diminished or absent pulse in the anterior tibialis and/or dorsalis pedis can be indicative of arterial insufficiency
- Capillary refill, where skin that, after being sufficiently compressed to blanch, takes more than 3 seconds to return to normal may be indicative of arterial insufficiency.<sup>44</sup>

Pedal pulse palpation is subjective and may be obscured in patients with oedema, limiting its use in venous disease. Capillary refill is poorly evidenced and not always reliable, especially in patients with diabetes and autonomic dysregulation, and it is more difficult to detect in people who have dark skin tones, so it should have a limited clinical role. Therefore, confirmation of clinical diagnosis ideally requires objective, quantifiable and reliable measurements of arterial blood flow.<sup>45</sup> A hand-held Doppler ultrasound device and a blood pressure monitor (sphygmomanometer) should be used to calculate the following important metrics of arterial flow to the distal lower leg:

- Ankle pressure (absolute ankle systolic pressure), where systolic pressure in the 50–100 mmHg range can indicate mild-to-moderate arterial insufficiency and below 50 mmHg severe arterial insufficiency<sup>34</sup>
- Ankle brachial index (ABI), also known as the ankle brachial pressure index (ABPI), calculated by dividing the ankle pressure by the brachial systolic pressure, where an

ABI in the 0.4–0.8 range suggests moderate arterial insufficiency and an ABI below 0.4 is indicative of severe arterial insufficiency.

An ABI reading above 1.4 is not considered reliable because of arterial calcification that makes the arteries noncompressible, which is a condition associated with diabetes.<sup>34</sup> Patients with CVI alone should have a normal ABI of 0.9 and above, and as many as 30% of patients with a VLU have an ABI above 0.8.<sup>46</sup>

An 8 Mhz Doppler probe is usual, although a 5 Mhz Doppler probe may be used in patients with oedema, as this can help locate and interpret deeper pulses. The blood pressure cuff is used to measure the systolic pressure in the brachial artery and the highest pressure of the arteries in the leg, commonly the dorsalis pedis (or anterior tibial) and the posterior tibialis arteries, although the peroneal artery can also be used. In patients who have diabetes or cannot tolerate the inflation of the blood pressure cuff around the ankle, the first toe can be used instead to take the following measurements:

- Toe pressure (absolute toe systolic pressure), where systolic pressure in the 30–60 mmHg range is indicative of mild-to-moderate arterial insufficiency and below 30 mmHg of severe arterial insufficiency<sup>34</sup>
- Toe brachial index (TBI), also known as the toe brachial pressure index (TBPI), calculated by dividing the toe pressure by the brachial systolic pressure, where a TBI below 0.7 is indicative of arterial insufficiency, although guidelines do not specify an index threshold for distinguishing severe presentations.<sup>35</sup>

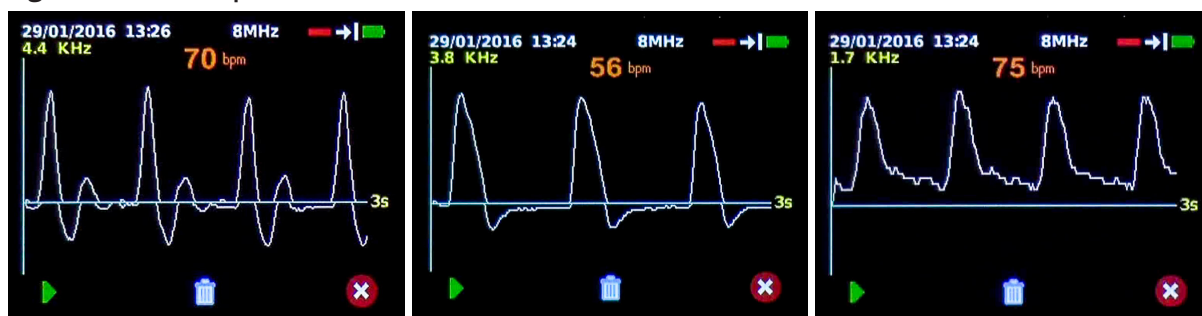
A handheld Doppler ultrasound device can also use reflected sound waves to assess the direction of arterial blood flow, either representing it as an audible sound or displaying it as a visual waveform. A triphasic or biphasic pulse is normal, while a monophasic pulse is indicative of arterial insufficiency. Accurate testing and interpretation of audible sound or visual waveforms requires specific specialised training and competency (Figure 3).<sup>47</sup>

**Consensus statement:** Where possible, it is best practice to obtain an objective measurement of arterial supply by calculating the ABI. Assessment of patients with suspected CAVI or PAD should also consider the absolute ankle pressure, as it is a determinant of safe use of compression therapy.

**Table 3. Objective instrumental assessments of arterial insufficiency**

Metric	Mild-to-moderate arterial insufficiency	Severe arterial insufficiency
Ankle brachial index	0.5–0.8	<0.5
Ankle pressure	No data	≤60 mmHg
Arterial waveform	Monophasic	Monophasic or absent
Partial pressure of oxygen (TcPO <sub>2</sub> )	30–50 mmHg	<30 mmHg
Toe brachial index	<0.7	No data
Toe pressure	30–75 mmHg	<30 mmHg
Skin perfusion	No data	<30 mmHg

**Figure 3. Arterial pulse waveforms**



- |  |  |  |
|--|--|--|
| <p><b>Triphasic</b></p> <ul style="list-style-type: none"> <li>• Systolic forward flow</li> <li>• Early diastolic reverse flow</li> <li>• Late diastolic forward flow</li> </ul> | <p><b>Biphasic</b></p> <ul style="list-style-type: none"> <li>• Systolic forward flow</li> <li>• Early diastolic reverse flow</li> <li>• Absent late diastolic forward flow</li> </ul> | <p><b>Monophasic</b></p> <ul style="list-style-type: none"> <li>• Unidirectional forward flow (delayed acceleration and deceleration)</li> </ul> |
|--|--|--|

If there is concern about arterial insufficiency because the objective measurements previously described either produce abnormal results or cannot be performed, further vascular assessment can include:

- Transcutaneous oximetry, where a transcutaneous partial pressure of oxygen (TcPO<sub>2</sub>) below 50 mmHg is indicative of arterial insufficiency and TcPO<sub>2</sub> below 30 mmHg is indicative of severe arterial insufficiency
- Skin perfusion pressure (SPP), usually measured by laser Doppler, where an SPP in the 30–50 mmHg range indicates mild-to-moderate arterial insufficiency and below 30 mmHg is indicative of severe arterial insufficiency<sup>48</sup>
- Pulse volume recordings, where a minimal pulse volume recording is indicative of severe arterial insufficiency
- Near-infrared spectroscopy, where infrared light is used to measure regional tissue oxygenation.<sup>49,50</sup>
- Arterial duplex ultrasound, which can help assess disease severity, stenosis locations (single or multiple) and intervention options (such as revascularisation).<sup>28,51,52</sup>

**Consensus statement:** In the presence of CLTI, computed tomography angiography (CTA) and magnetic resonance imaging (MRI) may also be useful in assessing severity and determine treatment options. Examination of the femoral, popliteal and pedal pulses may help determine the location of arterial disease.

In venous insufficiency, venous duplex ultrasound can be used to visualise superficial and deep vein compartments to detect venous insufficiency or obstruction and explore perforator veins to assess their competence.<sup>53–55</sup> CVI can also be assessed with the Trendelenburg test of vein incompetence, evaluating the competence of the venous valves by measuring the refill times of the superficial veins.<sup>56–58</sup>

**Consensus statement:** The full suite of diagnostic tests described in this document represents an ideal, and it may not always be realistic to perform all of them in real-world practice, although ankle/toe pressure and ABI/TBI should always be determined before initiating strong compression therapy.

## Pain assessment

The nature of pain felt in the leg or wound can indicate the aetiology. Pain severity can be documented with an established universal pain assessment tool, such as the 0–10 Visual Analogue Scale and the Wong-Baker Faces Pain Scale.

In venous insufficiency, pain is typically associated with inflammation or infection, and it decreases with elevation. CVI patients may also complain of heaviness, tingling or itching.<sup>59</sup> Patients with CAVI often experience more pain than would be expected with CVI, which can affect their tolerance of compression therapy. It should be noted that patients may report both nociceptive (pain from physical damage) and neuropathic pain (nerve pain) and, if left untreated, may develop central sensitisation, leading to symptoms such as allodynia (extreme sensitivity) on mild touch.<sup>60</sup>

Intermittent claudication can be assessed with a test of a patient's ability to walk set distances, such as the 2-minute step test or 6-minute walk test.<sup>30</sup> Intermittent claudication after walking a distance less than 200 m is a sign of severe arterial insufficiency. However, walking ability can be affected by factors including gait, general health and comorbidities, and measurements of intermittent claudication may be obscured by or confused with other causes of pain triggered by exercise, such as spinal stenosis.<sup>61</sup> The location of intermittent claudication is directly related to the anatomical location of the arterial disease.<sup>29</sup> More formal non-invasive flow studies may be obtained by recording ABI and the arterial pulse before and after a fixed amount of exercise. There are a variety of protocols available, such as 5 minutes of walking on an incline of 1.8%.<sup>62</sup>

Ischaemic rest pain, a sign of severe arterial insufficiency, can be assessed by asking a recumbent patient to move their leg between an elevated and dependent position. Increased pain on elevation is indicative of CLTI.

## Limb assessment

A full assessment of a limb with suspected venous or arterial disease can help determine the aetiology and what care is necessary and appropriate.

Lower-extremity oedema, especially phlebolympoedema, can be a visual indicator of a venous aetiology and is common in patients with CVI (*Figure 4*).<sup>63</sup> Presence of haemosiderin staining in the gaiter area is a clinical hallmark of underlying CVI.<sup>64</sup> However, there are many other potential causes of oedema, including use of medications such as calcium channel

**Figure 4. Phlebolympoedema**



blockers (*Box 5*).<sup>18,64–68</sup> These concomitant causes need to be ruled out with a full review of the medical and medication history before a wound on an oedematous leg can be diagnosed as a VLU, even in patients who present with the signs of phlebolympoedema. Progressing lymphoedema is associated with toe oedema and an inability to grab the dorsal skin between a thumb and finger over the second toe (Stemmer sign).<sup>69</sup> PAD in the absence of underlying venous disease does not usually present with oedema, although these patients may have oedema for other reasons.

Other visual signs can help distinguish venous and arterial disease and identify progression. The skin should be examined for skin changes, such as haemosiderin staining, atrophic blanche, epidermal lichenification, venous eczema and hyperkeratosis, which are suggestive of venous disease and can greatly affect the patient's skin integrity.<sup>24</sup> Skin atrophy and onychomycosis (fungal nail infection) may suggest arterial insufficiency.<sup>33</sup> More severe arterial insufficiency is indicated by changes in skin temperature and colour in particular positions. This includes red or purple discoloration when the leg is in a dependent position (dependent rubor), although this may be less visible in people with dark skin tones. Likewise, holding the foot in an elevated position will show pallor in light skin and an ashen hue in dark skin (elevated blanching). Arterial insufficiency can also be assessed with Buerger's test, an assessment of the angle to which the leg has to be raised in the supine position before it blanches. With normal arterial flow, the toes and sole retain their normal colour when raised to 90°; with mild-to-moderate PAD pallor will occur with elevation 15–30° for 30–60 seconds; and with moderate-to-severe PAD pallor will occur at less than 20°.<sup>70</sup>

**Consensus statement:** It is important to assess for factors that can impair the calf venous pump during ambulation, including limitations in the ankle's range of motion and strength, as well as absence of a heel-toe sequence during the gait cycle. If gait impairments are detected, corrective interventions and evaluation for assistive devices should be part of the care plan. If a patient has been using an assistive device, any alterations in the patient's gait pattern need to be identified and corrected.

**Box 5. Non-venous conditions that can cause lower-limb oedema**

- Cardiovascular disease and heart failure
- Dependent oedema
- Hyponatraemia
- Liver failure
- Lymphoedema (primary or secondary to cancer)
- Orthopaedic surgery
- Pelvic malignancy with inguinal lymph node obstruction
- Prolonged dependency due to impaired mobility (e.g., continual wheelchair use)<sup>204</sup>
- Renal failure
- Systemic disorders
- Trauma

**Wound assessment**

In patients with a leg ulcer, visual assessment of the wound can identify distinct differences between the typical appearance of venous, arterial and arteriovenous leg ulcers (*Figure 5*). Venous ulcers are typically located in the gaiter area and arterial ulcers typically on the foot, toe or (in severe cases) lower leg. Arterial ulcers are more likely to have fibrous or necrotic tissue in the wound bed. Venous ulcers are typically shallow, with irregular (serpentine) sloping edges, although depth may depend on degree of oedema. Arterial ulcers typically have steeper punched-out edges, and they are often deeper and, thus, more likely to have possible tendon or fascia exposure. Therefore, if a wound has exposed joint capsule or bone, there is always suspicion of arterial involvement. Wound drainage tends to be higher in venous ulcers and absent in arterial ulcers.<sup>46</sup>

**Figure 5. Leg ulcers by aetiology**



Arterial leg ulcer

Arterial leg ulcer

Arterial foot ulcer

Arteriovenous leg ulcer

Arteriovenous leg ulcer

Venous leg ulcer

Venous leg ulcer

Traumatic wound with signs of venous and arterial components

**Consensus statement:** The wound and periwound skin should undergo regular monitoring to track healing progress, with assessments at every dressing change. Vigilant monitoring of skin integrity is crucial, particularly for patients with arteriovenous ulcers. It is essential to provide meticulous care and follow up to ensure that treatments do not exacerbate skin loss.

The wound and periwound area should be assessed for clinical signs of infection (Box 6).<sup>71-73</sup> If infection is suspected and does not resolve as expected with standard treatment according to guidelines, a swab culture using the Levine technique should be taken. This will determine if the infection is bacterial, viral or fungal to guide a targeted antimicrobial approach, as well as sensitivity to antibiotics to help minimise antibiotic over-prescription.<sup>73-77</sup> A biopsy should be taken if the wound fails to progress in the expected timeframe and meets the criteria given in Table 4. If available, fluorescence imaging can assist in determining the location of pathogenic activity; the species and amount of bacteria; and the presence of biofilm, thus facilitating earlier and more complete debridement of biofilm and non-viable tissue.<sup>78-81</sup> The non-invasive nature of fluorescence imaging makes this technology particularly helpful for those diagnoses where a biopsy may be detrimental, such as in pyoderma gangrenosum, as this can trigger an exaggerated inflammatory response and worsening of the wound.<sup>82</sup>

**Box 6. Signs of infection in a wound<sup>60,71-73</sup>**

- New or increasing wound pain
- New or increasing oedema
- Periwound erythema
- Local warmth
- Purulent exudate
- Increasing malodour
- Increasing wound size
- New satellite wounds
- Slough on wound bed

**Table 4. Indications for wound biopsy (panel consensus)**

<b>Aetiology</b>	Indication of atypical underlying cause, such as history of burn or trauma
<b>Appearance</b>	Inconsistent appearance, suggestive of malignancy, vasculitis or vasculopathy
<b>Behaviour</b>	Failure to heal as expected (e.g., <25% size reduction in 4 weeks)
<b>Consistency</b>	Unusual tissue quality (e.g., hypergranulation or different viscosities of exudate)
<b>Duration</b>	Wound present for 12 weeks or more
<b>Edge</b>	Raised edges (epibole) or dark/ discoloured edges
<b>Location</b>	Unusual location for wound type
<b>Pain</b>	Persistent or exquisite pain

**Consensus statement:** Innovative detection and diagnostic technologies, such as fluorescence imaging for pathogenic activity and near-infrared spectroscopy for tissue oxygenation, may be useful for specific assessments. Despite the growing evidence base for these technologies, they may not be available in all relevant settings.

Leg ulcers with notably atypical or inconsistent characteristics for their suspected type (e.g., symptoms, presentation or history) should undergo a wound biopsy. A biopsy may also be indicated for a hard-to-heal wound that has failed to heal as expected with administration of evidence-based care, defined for example as a 25% size reduction in 4 weeks.<sup>83,84</sup>

## Holistic assessment

Initial and ongoing assessment should be holistic, incorporating a thorough and subjective medical history, noting current medication and any psychosocial issues that may affect the patient's education, adherence to the treatment plan and capacity for self-care. It is also recommended to assess holistic health factors that can be a risk for arterial insufficiency, such as smoking and obesity.<sup>85</sup> Quality of life can be measured with the tools specific to patients with venous disease listed in *Box 7*. For example, the Venous Disability Score (VDS) evaluates the effect of venous disease by quantifying the level of work-based disability, based on the ability to work an 8-hour day with or without provisions for external support and including the presence and size of leg ulcers. A systematic review by Launois found that the most extensively validated scales were the Chronic Venous Insufficiency Questionnaire and Venous Insufficiency Epidemiological and Economic Study on Quality of Life (VEINES-QoL).<sup>86</sup> A study by Gonzalez-Consuegra reported the most commonly used tools for people with VLUs were the Short Form-36, the Nottingham Health Profile and the EuroQol-5 (EQ-ED-5L) (EQ-ED-5L), although the author concluded that the most appropriate tool was the Charing Cross Venous Leg Ulcer Questionnaire because of its disease-specific

## Box 7. Tools for holistic assessment of patients with venous insufficiency or venous leg ulcers

- Aberdeen Varicose Vein Questionnaire (AVVQ)
- Cardiff Wound Impact Schedule
- Charing Cross Venous Leg Ulcer Questionnaire
- Chronic Venous Insufficiency Questionnaire (CIVIQ)
- EuroQol-5 (EQ-ED-5L)
- Freiburg Life Quality Assessment for Chronic Venous Disease (FLQA-VS-10)
- Nottingham Health Profile
- Questionnaire on Quality of Life with Chronic Wounds (Wound-QoL)
- Sheffield Preference-based Venous Leg Ulcer SD
- Short Form-36
- Venous Disability Score (VDS)
- Venous Insufficiency Epidemiological and Economic Study on Quality of Life (VEINES-QoL)
- Wuerzburg Wound Score

psychometric characteristics.<sup>87</sup> The VDS has four categories for how the patient perceives the impact of venous disease on their ability to perform usual activities (with and without compression therapy). These quality-of-life tools and the VDS can help health professionals determine treatment goals and facilitate patient adherence.<sup>88</sup> Functional and holistic assessment will also help determine a patient's capacity for supported self-care.

Patient-reported outcome measurements (PROMs), such as pain scales, quality-of-life scores and disability scores, are essential for setting and measuring clinical objectives. A systematic review by Gethin emphasised the need for validated PROMs for patients with VLUs.<sup>89</sup>

# Compression therapy

Compression therapy refers to the clinical application of therapeutic pressure to the affected limb using one of a variety of compression systems. This pressure can improve venous function and thus address underlying venous insufficiency. Compression therapy can also improve lymphatic function, and so it is also used to treat lymphoedema, including phlebolymphoedema. Compression can also benefit arterial function by enhancing venous return, which can help reduce back pressure on the arteries. Compression therapy has anti-inflammatory properties, and, in suitable patients, it is known to reduce pain, exudate and associated skin problems, as well as decrease healing times and risk of recurrence.<sup>90–92</sup> Compression therapy has been linked with more frequent and faster complete healing of VLU in two systematic reviews.<sup>93,94</sup>

Compression therapy is the mainstay of evidence-based treatment for venous insufficiency and venous ulcers. Additionally, compression is beneficial for managing most cases of oedema.<sup>95,96</sup> For patients with CAVI, compression remains safe and effective in improving venous insufficiency and, at the same time, in enhancing arterial perfusion.<sup>97</sup>

## Indications, contraindications and cautions

Compression therapy must be applied at a degree of pressure that is high enough to be therapeutically effective but not so high that it causes risk. In VLUs without arterial involvement, compression therapy with a pressure of at least 40 mmHg is recommended to provide the maximum clinical benefit. However, in CAVI, compression pressure must be limited to a maximum of 40 mmHg.<sup>98–101</sup>

**Consensus statement:** Patients with CAVI have both venous disease that may benefit from compression therapy and arterial disease that can present a significant risk. Therefore, compression therapy may be indicated in CAVI with or without ulceration. However, compression in CAVI must be used with greater caution and reduced pressure than in CVI alone. Compression where there is significant suspicion of arterial involvement should only be initiated after extensive vascular assessment; it must be applied in clinic by a suitably trained and skilled health professional, and the degree of arterial insufficiency should be frequently assessed.

In an ideal setting, the safety, suitability and strength of compression therapy are determined by an ABI and absolute ankle pressure (*Figure 6*). According to guidelines from the European Society for Vascular Surgery (ESVS), compression therapy is contraindicated with an ABI below 0.6 (or absolute ankle pressure  $\leq 60$  mmHg); moderate compression is indicated with an ABI in the 0.6–0.8 range; and strong compression is indicated with an ABI above 0.8.<sup>3</sup> The ESVS ankle pressure and ABI thresholds for contraindicating compression

( $\leq 60$  mmHg and  $< 0.5$ ) are higher than those for diagnosing CLTI ( $< 50$  mmHg and 0.4). When ABI readings are unreliable, such as in diabetes or severe oedema, TBI and toe pressure should be considered instead,<sup>102</sup> with reference to relevant guidelines.<sup>35</sup>

**Consensus statement:** Safe use of moderate compression in patients with an arteriovenous ulcer requires separate evaluations for both ABI/TBI and ankle/toe pressure, even though ankle/toe pressure is used to calculate the ABI/TBI. This is because, for example, the same ABI of 0.5 would be calculated for a patient with 50 mmHg ankle and 100 mmHg brachial pressures (unsafe for compression) as for a patient with 90 mmHg ankle and 180 mmHg brachial pressures (safe for compression).

## Figure 6. When to use compression therapy

- Signs of chronic venous insufficiency
- Ankle brachial index  $> 0.8$
- Ankle pressure  $\geq 60$  mmHg
- Toe pressure  $\geq 30$  mmHg
- No or very few signs of arterial insufficiency

Chronic venous insufficiency without arterial involvement

**Strong compression indicated  
(40–60 mmHg)**

- Signs of chronic venous insufficiency
- Ankle brachial index 0.6–0.8
- Ankle pressure  $\geq 60$  mmHg
- Toe pressure  $\geq 30$  mmHg
- No or very few signs of severe arterial insufficiency

Combined arterial and venous insufficiency

**Moderate compression indicated  
(20–40 mmHg)**

- Ankle brachial index  $< 0.6$
- Ankle pressure  $< 60$  mmHg
- Toe pressure  $< 30$  mmHg
- Significant signs of severe arterial insufficiency
- Signs of chronic limb-threatening ischaemia
- Signs of decompensated heart failure (inability to carry out physical activity without discomfort or symptoms of cardiac insufficiency at rest)
- Extra-anatomical bypass
- Severe neuropathy with sensory loss

Severe peripheral arterial disease / decompensated heart failure

**Compression contraindicated\***

\*Mild compression may be considered outside of guidelines in exceptional circumstances under strict expert surveillance (panel consensus)

ABI/TBI and ankle/toe pressure should be sufficient to determine the safety of compression therapy. However, decision-making can be supported by other signs of venous or arterial involvement identified during a comprehensive patient assessment (*Box 8* and *Box 9a*).<sup>2,3,102-105</sup>

**Consensus statement:** Where key instrumental measures such as ABI/TBI and ankle/toe pressure are unreliable or unavailable, such as in resource-limited settings, trained and experienced health professionals should make informed decisions on prescribing or modifying the strength of compression using the best information that is available. However, a full and accurate vascular assessment should be completed if and as soon as possible.

Compression therapy is generally contraindicated in the following conditions:

- Severe neuropathy with sensory loss
- Extra-anatomical bypass, as subcutaneous bypass cannot be compressed
- Severe PAD/CLTI (*Box 9b*), where it may cause further skin breakdown and impede wound healing.<sup>45,106,107</sup>

**Consensus statement:** Patients with the general aforementioned contraindications may be considered for mild compression therapy (<20 mmHg) in exceptional individual circumstances, such as problematic oedema. This use of compression is outside of existing guidelines and should only be undertaken under the strict surveillance of an expert health professional with considerable proven experience and competence in compression application.

Compression therapy at any strength is strictly contraindicated in patients with decompensated heart failure of New York Heart Association (NYHA) class IV, as it may further overload the heart.<sup>108</sup> NYHA class IV is defined as severe limitations, with increased discomfort during any physical activity and symptoms of cardiac insufficiency at rest.<sup>109,110</sup>

Compression therapy can be considered to manage leg oedema in patients with less-severe (compensated) chronic heart failure where cardiac conditions have been stabilised with appropriate treatment.<sup>111</sup>

**Consensus statement:** In patients with chronic heart failure, compression should be initiated in just one leg, and only once oedema has been removed from this leg should the other leg be treated with the same modality. This will limit how much fluid is progressively shifted from legs into the general circulation, reducing the risk of worsening the cardiac condition. The strength of compression should be determined by clinical assessment of the severity of heart failure versus the need for compression. If there is concern about the stability or severity of heart failure, then a staged approach to compression therapy is recommended, initially using a mild strength and then increasing in strength in line with clinical assessment. Multidisciplinary collaboration with heart-failure specialists is required to monitor symptoms and signs of cardiac failure.

### Box 8. Signs of chronic venous insufficiency

- Phlebolympoedema
- Pathologic skin changes
  - Atrophie blanche
  - Epidermal lichenification
  - Haemosiderin staining
  - Lipodermatosclerosis
  - Venous eczema
- Predominantly venous ulceration
  - High drainage
  - Uneven (serpentine) wound edges
  - Shallow wound
  - Sloped wound edges
  - Wound on gaiter area

### Box 9a. Signs and symptoms of arterial insufficiency of any severity

- Absent pedal pulse
- Intermittent claudication
- Abnormal vascular readings
  - Ankle brachial index <0.8
  - Capillary refill >3 seconds
  - Monophasic arterial waveform
  - Partial pressure of oxygen (TcPO<sub>2</sub>) ≤60 mmHg
  - Toe brachial index <0.7
  - Toe pressure ≤60 mmHg
- Pathological skin changes
  - Cold limb
  - Hair loss
  - Onychomycosis
  - Skin atrophy

### Box 9b. Signs and symptoms of severe arterial insufficiency

- Dependent rubor
- Elevation blanching
- Gangrene
- Ischaemic rest pain
- Severe abnormal vascular readings
  - Absent arterial waveform
  - Ankle brachial index <0.4
  - Ankle pressure ≤50 mmHg
  - Minimal pulse volume recording
  - Skin perfusion <30 mmHg
  - Partial pressure of oxygen (TcPO<sub>2</sub>) <30 mmHg
  - Toe pressure <30 mmHg
- Predominantly arterial ulceration
  - Deep wound
  - Punched-out wound edges
  - Wound on foot or toe

## Types of compression system

There are several different systems available for compression therapy of venous or arteriovenous leg ulcers and to prevent recurrence, including compression bandages, adjustable wraps and compression stockings (Figure 7).

Compression bandages are bands of fabric that are wrapped around the limb in two, three or four overlapping layers, with different patterns of application. In wound care, bandages are typically applied as multicomponent bandage kits comprising several layers of different materials to produce different effects, such as increased pressure, stiffness and comfort.<sup>112,113</sup> For example, some bandage kits include padding or components that provide the additional benefits of topical treatment, such as gauze or foam impregnated with zinc or calamine. Multicomponent bandage kits may include a cohesive (self-adhesive) outer layer to provide additional pressure and ensure retention of the inner kit layers.<sup>113</sup>

**Consensus statement:** Compression bandages are globally the most widely used compression system in leg ulceration and in the reduction of oedema and exudate, and they are suited to many patient presentations.

Adjustable wraps are garments that have hook-and-loop fastening straps for relatively simple application, readjustment and removal, along with an inner sleeve or liner to provide skin protection and dressing retention if needed.

**Consensus statement:** Adjustable wraps are suited to patients who may benefit from greater self-care or caregiver involvement, and they can be a cost-effective option.

Hosiery kits (also known as leg ulcer hosiery kits) comprise two components that are designed to be worn together. Each component serves a different purpose, such as a stocking to deliver the majority of the compression needed and a liner for retention, comfort and ease of application (over a wound dressing), as well as light compression. The combination of materials may produce an effect that could not be achieved with any single component. For example, an inner layer may provide low pressure suitable for night-time wear and be easier to apply, while the second layer brings the combined pressure to a higher therapeutic dosage for daytime use.<sup>114</sup>

**Consensus statement:** Hosiery kits are suited to patients where wound exudate is contained within a dressing, without significant oedema or major limb distortion and who can self-care or have carer support, although prolonged failure to heal with a hosiery kit should prompt a re-evaluation of treatment.

Compression stockings (single-layer hosiery) are unobtrusive garments that are suited to patients without active ulceration to minimise the risk of occurrence or recurrence. Compression stockings are made from a variety of different fabrics and weaves, which can vary in their therapeutic functions.<sup>115</sup> Stockings are available in different compression classes that exert different degrees of pressure.

Figure 7. Example types of compression system





Specific compression systems are contraindicated if there is an allergy to their material components, although this can usually be resolved by changing to a compression system made from different materials.

## Functional characteristics of compression systems

Compression systems are further categorised according to their functional characteristics, particularly pressure, stiffness and elasticity.<sup>116</sup> The characteristics of compression systems have been systematised in the acronyms PLaCE (pressure, layers, components and elasticity)<sup>116</sup> and STRIDE (shape, texture, refill, issues, dosage and [a]etiology).<sup>115</sup>

Compression pressure (interface pressure) is the amount of pressure provided by a particular compression system, measured in millimetres of mercury (mmHg).<sup>117</sup> For safe application of compression, pressure must not exceed the arterial pressure.<sup>45</sup> Pressures that exceed this level are unsafe and can cause complications. However, when appropriately applied, pressures at the higher end of a patient's safe therapeutic range are likely to have a greater therapeutic effect than lower pressures. Maintenance of optimal and safe pressure requires appropriate device selection, limb measurement and application.

Compression pressure is often measured at the B1 area, in the medial aspect of the leg where the Achilles tendon turns into the gastrocnemius muscle. However, compression pressure can be measured at any part of a leg.

Manufacturers typically state an expected pressure range given by a particular compression system. However, in hosiery kits and compression stockings, the actual pressure provided by the material components and overlapping layers will be modified by patient factors, such as limb dimensions. Various pressures are often reported for the same hosiery in different randomised controlled trials.<sup>118</sup> In compression bandages and adjustable wraps, the pressure exerted is ultimately determined by the stretch given by the user to the material on application, the limb's dimensions and, for bandages, the number of applied turns (layers).<sup>119,120</sup>

Pressure forms the basis of many classification systems, including the World Union of Wound Healing Societies classification of compression systems (Table 5)<sup>121</sup>. Classification standards for elastic stockings vary between countries; for example, the RAL standard is established in German-speaking countries and Italy but not France, the UK or the US (Table 6).<sup>115</sup> Hosiery garments are standardised according to criteria including testing methods, fabric used in construction and the pressure delivered.

Compression systems can also be classified according to their elastic properties (Table 7).<sup>116</sup> Elasticity refers to a material's elastic recoil or ability to regain its initial length when stretched under a particular force. Elasticity can be expressed as a static stiffness index (SSI), calculated in vivo as the difference between standing and supine pressures exerted by a compression system at point B1.<sup>122,123</sup>

Elasticity can have a significant impact on the haemodynamic effect and comfort of a compression system (Figure 8). The less elastic (or stiffer) the compression material, the greater the haemodynamic effect in creating a fixed resistance on the calf muscle. This is related to how intravenous pressure changes with the body position, from below 20 mmHg in a supine

**Table 5. Example system for classification of compression systems by pressure**

Pressure class	mmHg
Mild	<20
Moderate	20–40
Strong	40–60
Very strong	≥60

Note: This example classification system from the World Union of Wound Healing Societies may differ from other classifications systems in use<sup>121</sup>

**Table 6. Classification standards for elastic compression stockings, mmHg**

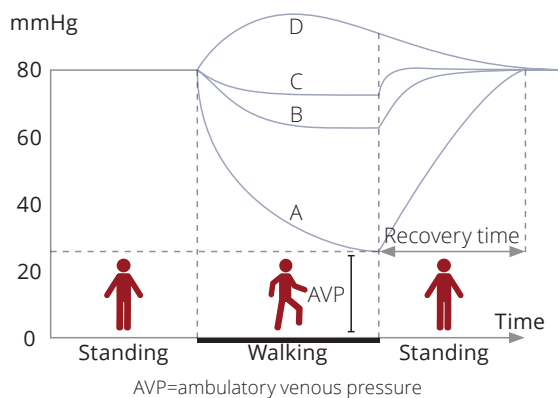
Pressure class	RAL	UK	FR	US
I	15–21	14–17	10–15	20–30
II	23–32	18–24	15–20	30–40
III	34–46	25–35	20–36	40–50
IV	>49	>35	>36	

**Table 7. Classification of compression bandage material by elastic properties<sup>116</sup>**

Elasticity class	Elongation	SSI*
Long-stretch	Over 100% of original length	≤10
Short-stretch	10–90% of original length	>10
Inelastic	<10% of original length	>10

SSI=static stiffness index; \*SSI values proposed by consensus panel

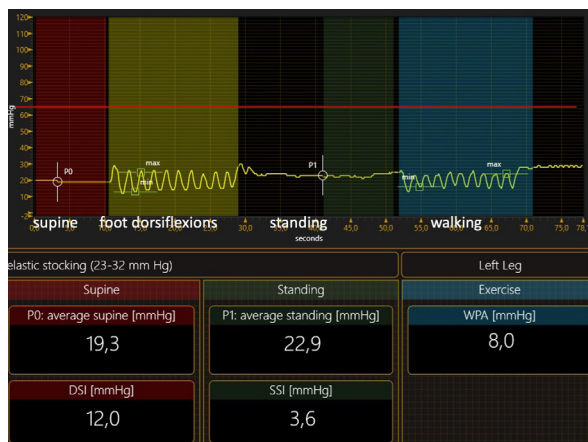
**Figure 8. Ambulatory venous pressure in (A) healthy subjects and patients with (B) superficial and perforator dysfunction, (C) additional deep venous dysfunction and (D) deep venous outflow obstruction<sup>152</sup>**



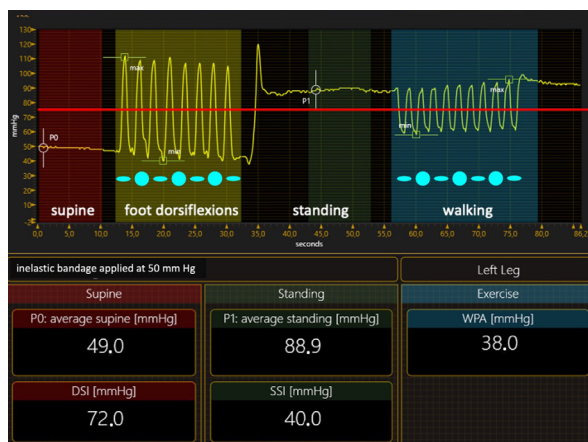
position, to around 40–50 mmHg in a sitting position and around 70–80 mmHg in a standing position. An ideal compression material for VLU treatment should exert a light pressure in a supine position when a strong pressure is not necessary to overcome the intravenous pressure, as well as a strong pressure in a standing position when it is necessary to approach or overcome the increased intravenous pressure. Elasticity also impacts pressure difference while the patient walks (pressure amplitude) and thus the massage effect on the calf muscle pump. Elastic materials are composed of elastic threads and are very stretchable (long-stretch) and may be characterised by an

### Figure 9. Pressure curves of elastic (left) and stiff (right) compression systems

These pressure curves show how standing pressure is minimally increased by an elastic system (stocking) and significantly increased by a stiff system (multicomponent bandage/ adjustable wraps). The static stiffness index (SSI) is 3.6 with the elastic system and 40 with the stiff system. Pressure amplitudes (peaks) during dorsiflexions are considerably narrower with the elastic system and wider with the stiff system, which thus exerts a greater ‘massaging’ effect on muscles and veins. The stiff system is effective in an intravenous standing pressure of up to around 70–80 mmHg, so occluding the veins and restoring a kind of valve mechanism. This is not the case with an elastic system, which would never be able to overcome this intravenous pressure.



Elastic material



Inelastic material

SSI up to 10. Elastic compression systems (e.g., elastic bandages and elastic stockings) follow the muscle expansion when the patient stands up or walks. As a consequence, standing pressures are only slightly higher than supine pressures and thus unable to overcome the intravenous pressure, while the massage effect on the calf muscle pump is minimal.

Stiff materials do not have elastic threads in the warp and weft, and they are much less stretchable (short-stretch) or not extensible at all (inelastic) and may be characterised by an SSI over 10. Stiff compression systems (e.g., inelastic bandages, zinc paste and adjustable wraps) resist muscle expansion when standing or walking. Consequently, standing pressures are significantly higher than supine pressures to overcome the intravenous pressure and exert an increased haemodynamic effect (>80 mmHg). During walking, a wide pressure amplitude exerts a strong massaging effect on the calf muscle pump. This adaptation of pressure to body position (low supine pressure and high standing pressure) can also make stiffer systems considerably more comfortable than elastic systems, which may be important for patient adherence (Figure 9).

Inelastic bandages have also been found to be significantly more effective than elastic bandages in decreasing venous reflux, increasing calf muscle pump function<sup>124,125</sup> and thus reducing ambulatory venous hypertension.<sup>126</sup> Inelastic compression at 40 mmHg of pressure has been shown to produce significantly higher peak pressures in the sitting and standing positions, as well as working effectively during passive and active exercise, making this well suited to patients with restricted mobility.<sup>127</sup> Different compression systems vary in their stiffness and elasticity.<sup>128–130</sup> Inelastic bandages, multicomponent bandages and adjustable wraps can all potentially be used to apply stiff compression.

Pressure maintenance is a relevant metric for predicting the clinical efficacy of a compression system.<sup>131</sup> Nevertheless, it has been demonstrated that inelastic materials maintain haemodynamic effectiveness despite significant pressure loss, mainly due to the maintenance of their massaging effect that is likely a major factor in the improvement of calf pump function.<sup>132</sup>

### Product selection

The compression system selected for an individual patient should provide the optimal therapeutic pressure and elasticity to treat the underlying pathology without significant risk.

**Consensus statement:** Patient input can help determine the most appropriate compression system for their needs and can be critical for patient satisfaction, adherence and thus treatment efficacy. Thus, patients should be asked how well they (or their carer) can apply, tolerate and remove different removable compression systems. If a patient is self-managing, their compression system should have characteristics and ease of use to suit their preferences, lifestyle and ability to apply and remove the system, which may be influenced by their age, occupation and dexterity. To ensure adherence, it is crucial that the patient understands the rationale for the compression system selected and that the system is manageable by the patient. This should be discussed at the earliest opportunity.

Product selection should be based on the best available evidence. Studies have evaluated the pressure (both at rest and with activity) and elasticity of different compression systems and their constituent materials *in vivo*, as well as their impact on patient outcomes.<sup>133–135</sup> A 2021 Cochrane review of 14 studies and 1391 participants reported that compression bandages or stockings, compared with no compression, in patients with a VLU reduced leg pain improved quality of life and enabled more frequent and faster complete healing, although the study did not compare different systems.<sup>94</sup> A 2012 Cochrane review suggested that multi-layer systems were more effective in healing wounds than single-layer systems and that systems containing an elastic component were more effective than those that contained only inelastic components.<sup>93</sup> A 2015 randomised controlled trial suggested that compression wraps are more effective than inelastic compression bandages in maintaining stable pressure, controlling venous oedema and promoting wound healing.<sup>131</sup> The 2014 VenUS IV study found that two-layer compression hosiery systems were as effective as four-layer compression systems in healing time for VLUs; additional benefits were reduced recurrence over 12 months and reduced costs associated with the supported self-care or caregiver involvement.<sup>114</sup>

However, these studies are limited by several major methodological flaws:

- Who applied the bandage and their experience of bandage application are never reported, so it cannot be known if the bandages were applied correctly.<sup>136</sup> Likewise, pressure and stiffness are almost never reported, also making it difficult to know if the bandages were correctly and consistently applied in all the patients. These limitations allow for situations such as a study in which a poorly applied elastic kit exerted lower pressures than an inelastic bandage, when it should have exerted a higher pressure if applied appropriately.<sup>137</sup>
- In almost all studies comparing inelastic and elastic materials, the elastic system used is almost always the same four-layer multicomponent bandage kit. This kit has been assumed to be elastic because it is made up of four elastic components. However, when the stiffness of the kit was assessed, it was shown to have the same stiffness as widely used inelastic devices. Consequently, these studies purporting to compare elastic and inelastic systems have actually compared two inelastic systems, significantly limiting the value of their conclusions.
- Inclusion of these limited studies in consensus papers and meta-analyses limits the validity of their conclusions.

The lack of conclusive data makes it difficult to recommend a best compression system for the treatment of venous and arteriovenous ulcers. However, some assumptions can be made based on expert opinion and what data are available.

From the very few studies reporting compression pressure, higher compression pressures are known to be associated with improved wound healing.<sup>99,137–139</sup> Inelastic systems are able to exert higher pressures than elastic systems (and with greater comfort). Moreover, inelastic compression systems are more

effective than elastic systems in improving the haemodynamic impairment underlying venous ulceration. For these reasons, venous and arteriovenous ulceration should generally be treated with inelastic compression systems, such as compression bandages, adjustable wraps or, after reduction of oedema, hosiery kits. However, elastic (long-stretch) systems can be used in small and recent ulcers, where they have proved effective in improving wound healing at 3 months.<sup>137,140</sup>

**Consensus statement:** Health professionals should aim to select a compression system that exerts a comfortable pressure at rest and a strong standing pressure to approach/overcome the intravenous pressure and exert the necessary haemodynamic effect. It should provide sufficient comfort and facilitate easy application, as well as protect the malleoli, Achilles tendon and friable skin at risk of skin tears. Inelastic materials seem to respond to these requirements and can be used in patients with venous or arteriovenous ulcers. However, based on the limited evidence available, it is not possible to make a decisive recommendation on which combination and characteristics of compression systems should be used in managing all venous or arteriovenous ulcers.

**Consensus statement:** Health systems in resource-limited settings may not have access to all commercially available compression systems. In these settings, health professionals can still achieve adequate compression by improvising with the supplies available, such as surgical bias wrap, washable and reusable bandages, three-layer tubular bandaging or gauze rolls, cast padding and self-adherent bandages. However, improvised systems made from layers of generic materials, similar to those in commercial compression products, cannot be guaranteed to provide the same interface pressure and the same effectiveness, and evidence is limited.

**Consensus statement:** As a patient's condition improves or deteriorates over time, the optimal type of compression system may also change, and so their compression needs should be assessed at each clinical assessment. Even as wound healing progresses and dressing needs change, the pressure required generally remains the same throughout treatment. Progress and anticipated outcomes need to be discussed with the patient after every assessment. A lack of progress should prompt a review of the intervention as part of the wider care plan and, ultimately, the diagnosis. Patients not responding to best-practice care, as evidenced by an improvement in symptoms, should be referred to a vascular or lower-limb specialist.

## Application technique

The actual interface pressure applied by a compression system is significantly influenced by the degree of tension with which a compression bandage or adjustable wrap is applied, as well as the number of layers used and the amount of overlap between these layers in compression bandages.

Leg ulcers may occur in the retromalleolar area (the space behind the malleolus), where the concave shape of the anatomy can leave a void between the limb and compression system.<sup>141</sup> This can critically reduce the pressure applied to the wound site,

which does not increase when standing or during muscle activity.<sup>142</sup> Pressure and stiffness in this area can be increased by applying local, focused compression.<sup>143</sup> This may be achieved either by proactively preparing and applying a small pad made from rolled wadding or foam to fill the void or by applying local compression straps to increase stiffness and pressure.<sup>42,143</sup> These straps can be applied in a variety of ways, such as a fan or chevron technique.<sup>143</sup> Health professionals should be trained to undertake these additional techniques safely and effectively using the correct adhesive inelastic materials.

**Consensus statement:** When applying compression in venous and arteriovenous ulceration, particular attention should be paid to ensuring appropriate therapeutic pressure on retromalleolar areas to prevent accumulation of fluid that may result in further skin damage.

With bandages, the application pattern helps determine the overlap and number of layers (*Box 10, Figure 10 and Figure 11*).<sup>143–145</sup> For example, a single bandage applied with the spiral technique and 50% overlap results in two layers of material, whereas a single bandage applied with the figure-of-eight technique and 50% overlap results in four layers of material, with correspondingly greater pressure and stiffness. The recommended application pattern, number of layers and components can significantly impact the complexity, accessibility and training requirements of different compression systems. For example, four-component compression bandage systems form 10 layers when applied according to

### Box 10. Application patterns for compression bandages<sup>205</sup>

#### Spiral technique

The bandage is applied with a spiral rotation from the base of the toes to just below the knee, usually with 50% overlap to produce equal pressure on all parts of the limb.

#### Figure-of-eight technique

The bandage is applied in oblique or diagonal turns that alternate between going up and down in the front, creating a figure-of-eight or herringbone pattern. Care should be taken to produce the same overlap and number of layers throughout.

#### Reverse-spiral or Pütter technique

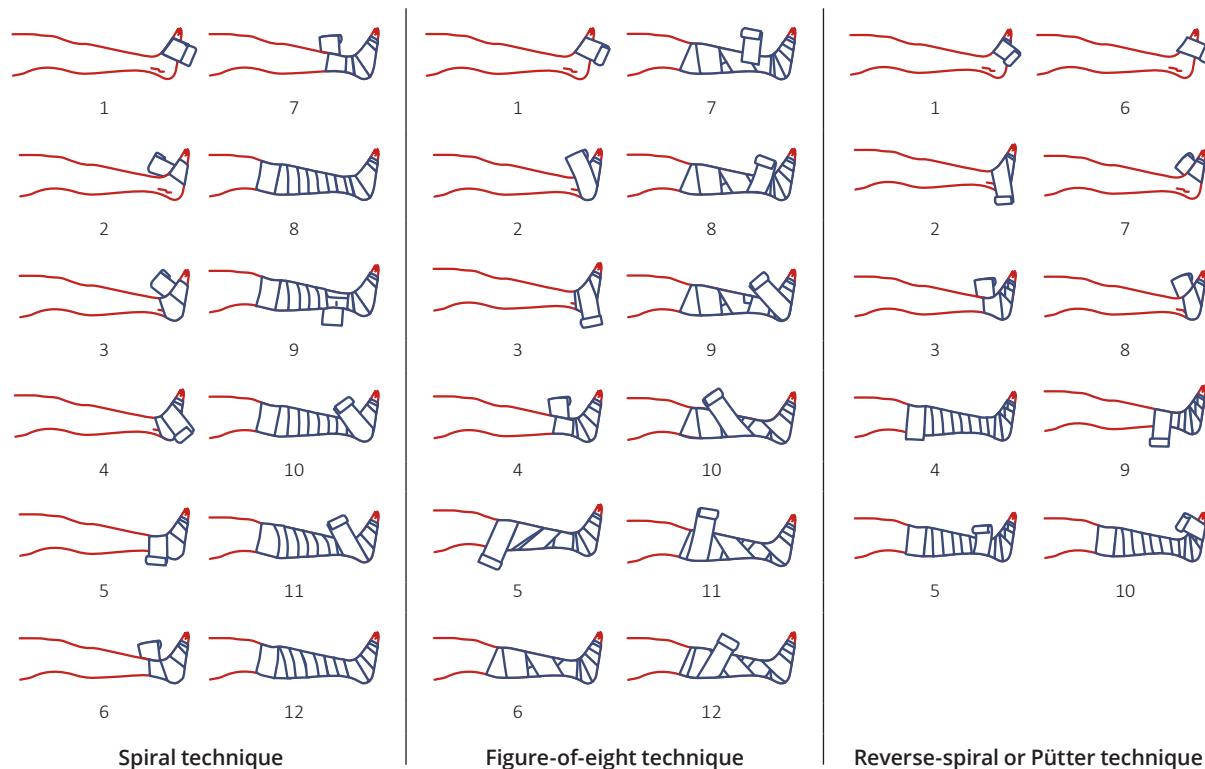
Two bandages are used. The first bandage is applied from the malleolus to the base of the toes, and then in a spiral rotation up the leg. The second bandage is applied starting at the malleolus in rotation down to the base of the toes and then up the leg, in a spiral rotation opposite to that used in the first layer. A variation of the Pütter technique, recommended for patients with a leg shaped like an inverted champagne bottle, is to wrap in a spiral up the leg and then a spiral down the leg.

#### Fischer technique

The bandage is applied starting at the heel, proceeding to the base of the toes and then in a spiral rotation up the leg to just below the knee.

Note: Always refer to manufacturer's instructions for use

Figure 10. Compression bandage application techniques<sup>205</sup>



**Figure 11. Compression bandage application**



manufacturer's recommendations and require considerable practice to apply correctly.

**Consensus statement:** Health professionals using compression bandages should be trained in appropriate application techniques, and users of a compression system should seek information from the manufacturer on the correct application technique to achieve the intended interface pressure. It is also imperative for health professionals to continue to practise and perfect their application technique to ensure the goals of compression therapy are achieved. This includes applying interface pressure appropriate for the pathology, while ensuring that calf-pump mobility is maintained and skin integrity is not compromised. The impact of application technique is of particular importance when applying moderate compression when there is arterial involvement and excessive pressure could present greater risks.

Although interface pressure is usually measured at the medial aspect of the lower leg, health professionals should be aware that pressure measurements elsewhere on the limb may be equally important. The impact of varying compression gradients and the corresponding haemodynamic function relative to varying oedema presentations is still under evaluation. It has been suggested that, despite a traditional focus on graduated compression, compression application is not always graduated and that other types of compression may be beneficial for the ambulatory population.<sup>146-148</sup> In which case, for such patients, health professionals may aim for even pressure distribution around and up the limb rather than focusing on engineering a compression gradient using excess padding. This discussion is ongoing and inconclusive.

**Consensus statement:** Bony prominences under compression are subject to greater interface pressures and thus should be protected from the risk of pressure injury with padding or wadding. Padding is especially relevant for patients with CAVI. Padding is not recommended for changing the shape of an inverted-champagne or pencil-shaped leg because the padding will decrease the amount of pressure delivered to that area resulting in less anti-inflammatory effect and possible skin damage. In addition, the padding should not be used to absorb exudate from a wound; that is the purpose of the wound dressing(s). Thus, it is recommended that extra padding should only be used to protect bony prominences that may be vulnerable to skin breakdown under pressure.

### **Box 11. Key factors to prevent recurrence of venous and arteriovenous ulceration**

- Effective treatment of the underlying aetiology
- Appropriate specialist referrals
- Appropriate superficial venous reflux correction
- Adherence to maintenance compression after wound healing
- Patient education on the need for follow-up care and impact of lifestyle modifications on comorbidities
- Professional education of health professionals on the need for compression after wound closure

### **Long-term maintenance**

Appropriate use of strong or moderate compression therapy should allow a venous or arteriovenous ulcer to fully heal (close) and oedema to be controlled. After wound closure (and oedema reduction) it is critical that compression therapy is continued, and an alternative compression device may be required to maintain long-term skin health and prevent recurrence of ulceration. The pressure provided by the maintenance device will depend on the underlying disease and other patient-specific criteria.<sup>115</sup> Health professionals should help minimise the risk of recurrence of ulceration by addressing the key factors in *Box 11*.

Compression stockings are recommended once the affected area is sufficiently remodelled to withstand putting on and removing a garment. Patients need to be trained in the correct method of putting on and taking off the stockings, with competent return demonstration of their competence in the technique.

Compression stockings have been shown to be safe and effective in oedema management for patients with moderate PAD.<sup>149</sup> During the remodelling phase of wound healing, the epithelium is fragile and vulnerable to breakdown, and so the transition from bandages to stockings needs to be closely monitored for skin breakdown or recurrence of ulceration, as well as for management of oedema.

Compression stockings undergo textile fatigue with continuous use and therefore need to be replaced approximately every 6 months. Patients should be informed that, if they find their stocking easier to put on, or if they note increased swelling of their limb, they may need to check with their health professional for replacement compression stockings or remeasurement for new compression stockings.

Long-term adherence to maintenance compression is critical to prevent ulcer recurrence. Patients in the maintenance phase need periodic follow-up assessments to monitor skin integrity and review stocking strength. This is a critical part of preventing recurrence and improving long-term self-management. Reassessments are best performed by a health professional with experience in assessment of oedema and skin integrity who can recognise when compression stockings need to be replaced. The Venous Clinical Severity Score (VCSS) is a valuable tool for determining if a patient is regressing on the CEAP classification, for example from a C5 back to a C6r ulcer.<sup>150</sup>

Beyond limb volume status, health professionals should monitor for changes in skin texture. Compression with insufficient pressure or stiffness may result in progressive tissue changes unrelated to limb volume. For example, a limb that remains the same size but becomes more fibrotic may have different compression needs.<sup>115</sup>

**Consensus statement:** Patients working in professions that require prolonged standing or more frequent washing of their garments may need to replace their compression stockings more often than normally recommended. In patients who cannot apply compression stockings, an application aid can be helpful, otherwise an alternative such as an adjustable compression wrap should be considered.

**Consensus statement:** Government agencies and third-party providers who may be reluctant to pay for compression stockings should be made aware of their cost-effectiveness and critical importance for ulcer prevention in patients of all ages.

# Holistic management

## Wound and skin care

In patients with a venous or arteriovenous ulcer, compression therapy must be accompanied by ongoing wound care, carried out according to the best-practice guidelines.<sup>151,152</sup> The details of this care are outside the scope of this document. Application of any compression system in venous insufficiency with or without ulceration must be preceded by cleansing, which consists of washing the wound and periwound skin with water or non-toxic solutions and potentially applying a hypoallergenic moisturiser to the periwound skin. It is essential that patients with ulceration undergo local wound bed preparation, including debridement of non-viable tissue to remove debris and biofilm,<sup>153</sup> treatment of local or deep tissue infection<sup>73</sup> and facilitation of epithelialisation at the wound edges, as well as management of exudate and protection of the periwound skin from maceration.<sup>152,154–156</sup> Care of a venous or arteriovenous leg ulcer generally requires selection of a wound dressing to be worn under the compression system that is appropriate to the patient's needs, such as absorptive dressings for exudate management or antimicrobial dressings for infection control.<sup>151,152</sup> Wound care should be carried out according to a structured, personalised and holistic strategy that addresses all the elements covered by the acronym TIMERS.<sup>151</sup>

- T: Tissue management
- I: Inflammation and infection
- M: Moisture balance
- E: Epithelial edge
- R: Regeneration and repair of tissue
- S: Social and patient-related factors.

Skin care is an important aspect for the management and treatment of patients with CVI, VLU and CAVI, both around the ulcer (if present) and the entire lower leg. The patient's skin integrity can be affected by factors including CVI-related skin changes, oedema and exudate pooling from the ulcer.<sup>24</sup> Treatment plans should aim to address skin changes. Some compression solutions may not be suitable for patients with compromised skin integrity, such as fragile skin or allergies to the system's components.<sup>157</sup>

The components of some compression systems may also cause dry skin or itching (pruritus). Pruritus has been found to affect one-in-three patients and potentially results in one-in-five non-adherent patients, which is important when successful treatment is highly dependent on adherence.<sup>158</sup> In venous eczema, when red blood cells and proteins leak from the enlarged veins into the tissues, resulting in skin irritation, successful management requires rehydration of the skin with prescribed topical emollients, in addition to treating the underlying venous insufficiency.<sup>159</sup> Both pruritus and venous eczema can be treated with a compression system impregnated with zinc oxide with or without ferric oxide.<sup>158,159</sup>

Skin changes can occur with both CVI and CAVI and require appropriate treatment and skin hygiene to regain or maintain skin integrity. Treatment of skin changes as a result of both the disease and compression usage is paramount for successful patient outcomes.<sup>157</sup> Good skin-care practice should continue after wound healing.

## Exercise

Stretching and strengthening exercises should be an integral part of the treatment plan for patients with CVI or CAVI who are receiving compression therapy. Exercises to increase the ankle range of motion and strengthen the gastrocnemius muscle group can support calf-pump function, which is essential for wound healing (*Figure 12*).<sup>160–162</sup> Patients should be taught and encouraged to perform exercises with the compression system in place. In addition, patient balance must be assessed, and if there is concern for safety, especially when doing unilateral exercises, ankle pumps and toe raises should be initiated in the sitting position and progress to standing only when safe.<sup>163</sup>

**Consensus statement:** Patients with venous or arteriovenous leg ulcers should benefit from exercise and elevation of the lower extremity in the supine position. However, patients with CAVI need to be monitored for reduction in arterial perfusion.

Supervised exercise therapy (SET) has been shown to be more effective than non-supervised programs.<sup>164,165</sup> Exercise that activates the gastrocnemius increases the girth of the calf. A stiffer compression system resists that expansion, creating an inward, intermittent force that compresses the deep veins and increases the velocity of blood flow.<sup>115,160,166,167</sup> This pumping action may aid in oedema management and wound healing; however, more randomised control trials with larger sample sizes are needed to confirm the effect of SET on healing of VLUs.<sup>160,168</sup> SET has been shown to be effective in improving walking distance and quality of life for patients with intermittent claudication. The exercise program consists of either treadmill walking with incline sufficient to elicit pain or exercise with a leg ergometer.<sup>169–171</sup>

**Figure 12. Heel-toe raises**



Exercise programmes and nutritional consultation aimed at optimising nutrition and controlling bodyweight are also recommended as part of the holistic treatment for patients with CVI or CAVI (moderate PAD). Obesity is a risk factor for venous insufficiency, and obese patients with CVI are more likely to be symptomatic.<sup>27,172</sup> Increased body mass index (BMI) is associated with limited ankle mobility in CVI.<sup>173</sup> Waist circumference is associated with elevated levels of reported diabetes, hypertension, lipid disorders and cardiovascular disease.<sup>174,175</sup> Patients with inadequate exercise and nutrition may benefit from referral to a physical therapist and/or nutritionist.<sup>176–178</sup>

**Consensus statement:** Waist–hip ratio is preferable to BMI as a guide to bodyweight and weight loss.

## Adjunctive therapies

Patients receiving compression therapy for VLU are likely to benefit from adjunctive therapies for oedema reduction, including negative pressure wound therapy (NPWT) and intermittent pneumatic compression (IPC).<sup>179–181</sup> IPC can also benefit patients with CAVI by increasing arterial blood flow for treatment and, in some instances, maintenance.<sup>182–184</sup> Unlike compression therapy, there are IPC units intended to treat arterial disease that may be used on patients with severe PAD or CLTI.<sup>182,185,186</sup>

Anti-embolism stockings should only be used for prophylaxis of venous embolism, as they are neither appropriate nor effective in applying therapeutic levels of compression for leg ulcers.<sup>187,188</sup>

Patients with symptomatic CVI should undergo a venous duplex ultrasound scan to assess for venous reflux and the suitability for venous intervention. Intervention for superficial venous incompetence should be carried out immediately after healing if not before healing.<sup>1,2</sup> The ESCHAR study was pivotal in showing that surgical treatment of superficial venous reflux and continued compression therapy are effective in reducing the recurrence of VLUs at 4 years and increasing ulcer-free time after healing.<sup>189</sup> Further studies (the EVRA trial) by Gohel et al. showed that endovenous ablation of superficial venous reflux within 2 weeks of initiating care resulted in faster wound-healing rates than compression alone with deferred surgical intervention and reduced the overall incidence of ulcer recurrence.<sup>58,190</sup> Although it can be presumed that wounds heal better and stay closed longer with better arterial supply, many patients are not receiving this care because of reluctance to have surgery and lack of access to vascular services. A recent study on treating incompetent perforators and surrounding veins in the presence of lipodermatosclerosis saw wounds reduce in size or completely heal within 1 month in 108 (81%) patients.<sup>191</sup>

## Revascularisation

Revascularisation, a surgical or endovascular procedure to address underlying arterial insufficiency, is the mainstay of the treatment for PAD with arterial ulceration/CLTI, with or without venous involvement. Patients with PAD with venous involvement may benefit from a combination of revascularisation to treat the arterial component and compression therapy to treat

the venous (and oedematous) component of their condition, which should shorten the wound healing time and minimise the risk of recurrence.

A study by Lantis et al. found that percutaneous revascularisation significantly improved the wound-healing trajectory of 27 patients with arteriovenous leg ulcers and ABI <0.7 when treated with ambulatory compression therapy. Prior to revascularisation, the wounds had remained open for an average of 17 weeks. After revascularisation, 100% of the wounds fully healed, taking an average of 10 weeks. At 10 weeks, the closure rate was 75%, and the average ABI was 0.97. The authors concluded that attaining a near-normal ABI with revascularisation accelerated wound healing and thus advocated an aggressive approach to revascularisation in arteriovenous ulcers.<sup>192</sup>

**Consensus statement:** Patients with signs of moderate-to-severe PAD should be referred to a vascular specialist for evaluation and potential revascularisation. Specialist vascular referral is especially critical for patients with progressive arterial disease and ulceration, to accelerate wound healing, prevent development of new arterial wounds and improve adherence to compression. Should revascularisation be unavailable for these patients, carefully monitored compression therapy is even more critical for wound healing and oedema management.

## Supported self-management

Patients with CVI or CAVI should be encouraged to participate in their care as appropriate. Patient empowerment (support and education) can be crucial to successful long-term outcomes. Patient-centred care should involve patient and/or caregiver engagement in all aspects of their care plan.

Health professionals first need to identify an individual patient's capacity for self care (where they are on the self-care continuum from being unconscious to being able to manage compression therapy independently) and then recommend the extent of self-care accordingly.<sup>193</sup> Once self-care has been initiated, health professionals are responsible for supporting patients with self-management strategies.<sup>194</sup>

Patient engagement can be encouraged with the application of validated models of behaviour change, such as the transtheoretical model of change, self-determination theory or motivational interviewing. These models aim to effect lasting health behaviour by placing more accountability on the patient/caregiver, whereby they are given the opportunity to decide on their own terms what changes to make to their health behaviour.<sup>195</sup> Barriers and facilitators to a patient's ability to improve their lifestyle and health behaviour can be identified using the International Classification of Functioning, Disability, and Health.<sup>195</sup>

**Consensus statement:** These models can be used to enhance patient self-management of their disease; to improve adherence to treatment plans including compression therapy; and to implement lifestyle behaviours that will promote healing, prevent recurrence and improve quality of life.



**Consensus statement:** Self-care delivery can be supported by establishing a wider carer strategy, including family members and/or friends.

## Patient education

Patients with venous and/or arterial insufficiency need to be educated about the aetiology of their condition and how it should be managed. Education is particularly essential for patients who are prescribed compression therapy. They need to be informed about the essential benefits of compression and why it is necessary for healing and long-term management, as this will help promote lasting adherence. They also need to be educated on the correct use of their prescribed compression system, such as how to readjust adjustable wraps to maintain the recommended level of pressure or how to recognise when stockings need replacing. Patients should also be taught how long the product can be expected to remain effective according to the manufacturer's recommendations, as well as any recommended exercises and leg elevation. This practical education should be confirmed with return demonstrations.

**Consensus statement:** To avoid the devastating effects of gravitational oedema, patients should be strongly encouraged to sleep in a supine position at night and to avoid prolonged periods with their feet and lower legs in a dependent position.

Patients also should be educated about appropriate skin care. This includes the use of high-quality hypoallergenic moisturisers that contain no perfumes or preservatives; the avoidance of aqueous creams and soap; and the application of topical steroids for eczema (depending on the wetness/dryness of the skin). Patients need to be instructed on proper footwear that will permit adequate ankle range of motion and calf muscle pump action during ambulation. It can be beneficial to incorporate models to facilitate behavioural change within patient education. Key points to patient education are highlighted in *Box 12*.

A key component of the education is the fostering of two-way communication.<sup>196</sup> Patients must understand the necessity of recognising changes in skin, pain and wound presentation that may occur during treatment and report these to the health professional. They should also be encouraged to provide feedback on the fit and comfort of their compression system.

Education needs to be provided in a form that is appropriate and accessible for each individual patient, whether that is paper handouts or links to online resources and video presentations. Patients may prefer to be part of a class or one-on-one instruction, as well as have remote or in-person contact. Help can be sought from support groups and patient ambassadors.

## Box 12. The 7-E model of patient education

- Education in an accessible form
- Engagement through personalised care
- Empowerment by supportive self-management and patient-reported outcome measures
- Evaluation as continuum, with patient-reporting a key component of the process
- Edification of patient and health literacy
- Endurance – development of patient maintenance of compression and good skin hygiene
- Encouragement of diagnosis-appropriate exercise

## Professional education

A study by Protz et al. measured the competency of 55 nurses in applying compression bandages before and after one training session and found a significant improvement (based on 6 control parameters) at 1 and 3 months.<sup>197</sup>

**Consensus statement:** It is imperative that all health professionals expected to deliver compression therapy receive the robust specialist training necessary to achieve competency and confidence in its use. This training should be facilitated by their employer and cover the following areas of clinical competency:

- Assessment of arterial and venous insufficiency, such as pulse palpation and Doppler interpretation
- Holistic assessment of the wound, limb and individual, including medical history and lifestyle factors such as smoking
- Selection of an appropriate compression system for the patient's needs
- Application of consistent pressures
- Patient education and motivation.

For health professionals in all regions and settings, an understanding of the theory underpinning practical skills is more valuable than access to expensive diagnostic equipment. Truly effective application of compression therapy that goes beyond simple delivery of instructions requires a deeper understanding of the patient, the diagnosis and the treatment plan. Trained and competent health professionals are better able to improve patient outcomes, adherence and satisfaction and thus raise staff morale. Professional education can be particularly impactful in settings where specialist care is less available.

# Conclusion

Compression therapy is universally accepted as an integral part of the best-practice treatment of CVI (with or without ulceration and lower-limb oedema) and CAVI. However, it can present serious risks for patients with severe arterial involvement.

Compression therapy can counteract the underlying venous aetiology and thus reduce pain, exudate and skin problems, as well as reduce healing times and risk of recurrence.

Compression therapy should be part of a wider care strategy that includes wound management, such as the use of dressings appropriate to the presentation and stage of wound healing.

Health professionals should feel confident in safely prescribing compression at therapeutic pressures so long as they are able to distinguish the signs of a venous, arterial or arteriovenous aetiology. In best practice, the key minimum requirements for prescribing compression therapy are an ABI of at least 0.6, an ankle pressure of at least 60 mmHg and a toe pressure of at least 30 mmHg, in addition to absence of signs of severe PAD or decompensated heart failure. In patients with an ABI over 0.8 and no or very few signs of PAD, health professionals should be confident in prescribing strong compression for the optimal therapeutic benefit. Where compression is generally contraindicated, experienced health professionals can consider mild compression in exceptional circumstances and with careful observation. Where key instrumental measures are unreliable or unavailable, as in resource-limited settings, experienced health professionals should make informed decisions on prescribing or modifying the strength of compression with the best information available, and a full vascular assessment should be completed if and as soon as possible. Clinical decisions on compression can be supported by other aspects of a full and holistic clinical assessment, and, where appropriate, patients should be encouraged to be actively involved in their care.

Treatment must be holistic and, in patients with leg ulcers, involve best-practice wound care. Patients with severe PAD with venous involvement should be referred to a vascular specialist for evaluation for revascularisation as soon as possible. Health professionals may be reluctant to begin

compression therapy before the referral is complete out of fear of causing harm to the patient. However, earlier initiation of compression therapy is associated with better outcomes, including shorter healing times, and a trained and competent health professional should be able to initiate compression at a safe and effective therapeutic pressure before referral (provided there are no absolute contraindications).

There is no single compression system appropriate for every patient, and health professionals need to select and apply the optimal system for the patient's clinical presentation and needs. The ideal compression system should deliver the appropriate therapeutic pressure to address the underlying pathology throughout the patient's activities of daily living. Moreover, a compression system needs to facilitate a normal gait and ankle mobility to maintain calf pump function and skin integrity. Likewise, it must not contribute to further integumentary dysfunction or oedema under the system or in adjacent areas, such as the toe or thigh. To promote lasting adherence, the most appropriate compression system should be recommended for each patient's individual needs, lifestyle and health status. Patients should be informed about the treatment options and the importance of continuing adherence to compression, skin care and exercise.

Health professionals providing compression therapy for patients with CVI or CAVI would benefit from more research and resources to assist with the optimal selection and application of compression systems. There is also a need for more accurate measurements of the actual interface pressure applied in practice, as well as the efficacy of wound healing.

Health services around the world should work together to overcome barriers to the initiation and success of an integrated strategy combining wound care and compression therapy, including misdiagnosis of the underlying aetiology on the lower extremity; poor patient adherence due to inadequate education or comfort; varying clinical competency in compression application technique; and lack of accessible diagnostic equipment and compression supplies.<sup>101</sup> **JWC**

# References

1. Rabe E, Partsch H, Hafner J et al. Indications for medical compression stockings in venous and lymphatic disorders: An evidence-based consensus statement. *Phlebology*. 2018;33(3):163–184. <https://doi.org/10.1177/0268355516689631>
2. O'Donnell TF, Passman MA, Marston WA et al. Management of venous leg ulcers: clinical practice guidelines of the Society for Vascular Surgery and the American Venous Forum. *J Vasc Surg*. 2014;60(2):35–59S. <https://doi.org/10.1016/j.jvs.2014.04.049>
3. De Maeseneer MG, Kakkos SK, Aherne T et al. European Society for Vascular Surgery (ESVS) 2022 clinical practice guidelines on the management of chronic venous disease of the lower limbs. *Eur J Vasc Endovasc Surg*. 2022;63(2):184–267. <https://doi.org/10.1016/j.ejvs.2021.12.024>
4. Harding K, Dowsett C, Fias L et al. Simplifying venous leg ulcer management: consensus recommendations. *Wounds International* 2015. <https://woundsinternational.com/consensus-documents/simplifying-venous-leg-ulcer-management-consensus-recommendations/> (accessed April 2024)
5. Azar J, Rao A, Oropallo A. Chronic venous insufficiency: a comprehensive review of management. *J Wound Care*. 2022;31(6):510–519. <https://doi.org/10.12968/jowc.2022.31.6.510>
6. Probst S, Saini C, Gschwind G et al. Prevalence and incidence of venous leg ulcers: a systematic review and meta-analysis. *Int Wound J*. 2023;20(9):3906–3921. <https://doi.org/10.1111/iwj.14272>
7. Ahmajärvi K, Isoherranen K, Venermo M. Cohort study of diagnostic delay in the clinical pathway of patients with chronic wounds in the primary care setting. *BMJ Open*. 2022;12(11):e062673. <https://doi.org/10.1136/bmjopen-2022-062673>
8. Son A, O'Donnell TF, Izhakoff J et al. Lymphedema-associated comorbidities and treatment gap. *J Vasc Surg Venous Lymphat Disord*. 2019;7(5):724–730. <https://doi.org/10.1016/j.jvs.2019.02.015>
9. Hamm R, Carey J. *Essential elements of wound diagnosis*. New York: McGraw Hill; 2021
10. European Wound Management Association. *Atypical wounds 2024*. <https://ewma.org/what-we-do/projects/atypical-wounds> (accessed April 2024)
11. Mitchell A, Ritchie G, Hopkins A, editors. *Lower limb and leg ulcer assessment and management*. Hoboken (NJ): Wiley-Blackwell; 2024
12. Körber A, Klode J, Al-Benna S et al. Etiology of chronic leg ulcers in 31,619 patients in Germany analyzed by an expert survey. *J Deutsche Dermatol Gesell*. 2011;9(2):116–121. <https://doi.org/10.1111/j.1610-0387.2010.07535.x>
13. Lurie F, Passman M, Meisner M et al. The 2020 update of the CEAP classification system and reporting standards. *J Vasc Surg Venous Lymphat Disord*. 2020;8(3):342–352. <https://doi.org/10.1016/j.jvs.2019.12.075>
14. Raju S, Lucas M, Thaggard D et al. Plethysmographic features of calf pump failure in chronic venous obstruction and reflux. *J Vasc Surg*. 2023;11(2):262–269. <https://doi.org/10.1016/j.jvs.2022.10.013>
15. Horwood A. The biomechanical function of the foot pump in venous return from the lower extremity during the human gait cycle: An expansion of the gait model of the foot pump. *Med Hypoth*. 2019;129:109220. <https://doi.org/10.1016/j.mehy.2019.05.006>
16. Tauraginskii RA, Lurie F, Simakov S et al. Calf muscle pump pressure-flow cycle during ambulation. *J Vasc Surg*. 2023;11(4):783–792.e7. <https://doi.org/10.1016/j.jvs.2023.04.002>
17. Milic DJ, Zivic SS, Bogdanovic DC et al. Risk factors related to the failure of venous leg ulcers to heal with compression treatment. *J Vasc Surg*. 2009;49(5):1242–1247. <https://doi.org/10.1016/j.jvs.2008.11.069>
18. Lurie F, Malgor RD, Carman T et al. The American Venous Forum, American Vein and Lymphatic Society and the Society for Vascular Medicine expert opinion consensus on lymphedema diagnosis and treatment. *Phlebology*. 2022;37(4):252–266. <https://doi.org/10.1177/02683555211053532>
19. Duhon BH, Phan TT, Taylor SL et al. Current Mechanistic Understandings of Lymphedema and Lipedema: Tales of Fluid, Fat, and Fibrosis. *IJMS*. 2022;23(12):6621. <https://doi.org/10.3390/ijms23126621>
20. Patel SK, Surowiec SM. *Venous insufficiency*. StatPearls, Treasure Island (FL): StatPearls Publishing; 2024
21. Raffetto JD. Pathophysiology of chronic venous disease and venous ulcers. *Surg Clin N Am*. 2018;98(2):337–347. <https://doi.org/10.1016/j.suc.2017.11.002>
22. Eberhardt RT, Raffetto JD. Chronic venous insufficiency. *Circulation*. 2014;130(4):333–346. <https://doi.org/10.1161/CIRCULATIONAHA.113.006898>
23. Chuback JA, Melin MM, Massey HT et al. Congestive lower extremity failure: An educational model for improved understanding of phlebolympheidema. *J Vasc Surg Venous Lymphat Disord*. 2024;12(2):101737. <https://doi.org/10.1016/j.jvs.2023.101737>
24. Cameron J. Dermatological changes associated with venous leg ulcers. *Wounds UK*. YumpuCom. 2007. <https://www.yumpu.com/en/document/view/23335978/dermatological-changes-associated-with-venous-leg-wounds-uk> (accessed April 2024)
25. Majmundar VD, Syed HA, Baxi K. *Livedoid vasculopathy*. StatPearls, Treasure Island (FL): StatPearls Publishing; 2024
26. Fife CE, Farrow W, Hebert AA et al. Skin and wound care in lymphedema patients: a taxonomy, primer, and literature review. *Adv Skin Wound Care*. 2017;30(7):305–318. <https://doi.org/10.1097/01.ASW.0000520501.23702.82>
27. Davies HO, Popplewell M, Singhal R et al. Obesity and lower limb venous disease: the epidemic of phlebesity. *Phlebology*. 2017;32(4):227–233. <https://doi.org/10.1177/0268355516649333>
28. Criqui MH, Matsushita K, Aboyans V et al. Lower extremity peripheral artery disease: contemporary epidemiology, management gaps, and future directions: a scientific statement from the American Heart Association. *Circulation*. 2021;144(9). <https://doi.org/10.1161/CIR.0000000000001005>
29. Zemaitis MR, Boll JM, Dreyer MA. *Peripheral Arterial Disease*. StatPearls. 2024. [www.ncbi.nlm.nih.gov/books/NBK430745/](https://www.ncbi.nlm.nih.gov/books/NBK430745/) (accessed April 2024)
30. Hardman R, Jazaeri O, Yi J et al. Overview of classification systems in peripheral artery disease. *Semin Intervent Radiol*. 2014;31(04):378–388. <https://doi.org/10.1055/s-0034-1393976>
31. Nordanstig J, Behrendt C-A, Baumgartner I et al. European Society for Vascular Surgery (ESVS) 2024 clinical practice guidelines on the management of asymptomatic lower limb peripheral arterial disease and intermittent claudication. *Eur J Vasc Endovasc Surg*. 2024;67(1):9–96. <https://doi.org/10.1016/j.ejvs.2023.08.067>
32. Santilli JD, Santilli SM. Chronic critical limb ischemia: diagnosis, treatment and prognosis. *Am Fam Phys*. 1999;59(7):1899–1908
33. Marin JA, Woo KY. Diagnostic accuracy of a fluorescence imaging device in diabetic wounds: a pilot study using a tissue culture system. *J Wound Ostomy Continence Nurs*. 2017;44(1):41–47. <https://doi.org/10.1097/WON.0000000000000294>
34. Conte MS, Bradbury AW, Kolh P et al. Global vascular guidelines on the management of chronic limb-threatening ischemia. *J Vasc Surg*. 2019;69(6):3S-125S.e40. <https://doi.org/10.1016/j.jvs.2019.02.016>
35. Fitridge R, Chuter V, Mills J et al. The intersocietal IWGDF, ESVS, SVS guidelines on peripheral artery disease in people with diabetes and a foot ulcer. *Diabetes Metab Res Rev*. 2024;40(3):e3686. <https://doi.org/10.1002/dmrr.3686>
36. Fereydooni A, Gorecka J, Dardik A. Using the epidemiology of critical limb ischemia to estimate the number of patients amenable to endovascular therapy. *Vasc Med*. 2020;25(1):78–87. <https://doi.org/10.1177/1358863X19878271>
37. Conte SM, Vale PR. Peripheral arterial disease. *Heart Lung Circ*. 2018;27(4):427–432. <https://doi.org/10.1016/j.hlc.2017.10.014>
38. Campia U, Gerhard-Herman M, Piazza G et al. Peripheral artery disease: past, present, and future. *Am J Med*. 2019;132(10):1133–1141. <https://doi.org/10.1016/j.amjmed.2019.04.043>
39. Matic M, Matic A, Djuran V et al. Frequency of peripheral arterial disease in patients with chronic venous insufficiency. *Iran Red Crescent Med J*. 2016;18(1). <https://doi.org/10.5812/ircmj.20781>
40. Ammermann F, Meinel FG, Beller E et al. Concomitant chronic venous insufficiency in patients with peripheral artery disease: insights from MR angiography. *Eur Radiol*. 2020;30(7):3908–3914. <https://doi.org/10.1007/s00330-020-06696-x>

41. Ghauri AS, Nyamekye I, Grabs AJ et al. The diagnosis and management of mixed arterial/venous leg ulcers in community-based clinics. *Eur J Vasc Endovasc Surg*. 1998;16(4):350–355. [https://doi.org/10.1016/s1078-5884\(98\)80056-7](https://doi.org/10.1016/s1078-5884(98)80056-7)
42. Hopkins A, Kerr A, Clarke C et al. Holistic management of venous leg ulceration (second edition). *Wounds UK* 2022. <https://wounds-uk.com/best-practice-statements/holistic-management-venous-leg-ulceration-second-edition/> (accessed April 2024)
43. National Institute for Health and Care Excellence. Venous leg ulcers 2024. <https://cks.nice.org.uk/topics/leg-ulcer-venous/management/venous-leg-ulcers/> (accessed July 2024)
44. Bachour RP de S, Dias EL, Cardoso GC. Skin-color-independent robust assessment of capillary refill time. *J Biophotonics*. 2023;16(11):e202300063. <https://doi.org/10.1002/jbio.202300063>
45. Andriessen A, Apelqvist J, Mosti G et al. Compression therapy for venous leg ulcers: risk factors for adverse events and complications, contraindications – a review of present guidelines. *Acad Dermatol Venereol*. 2017;31(9):1562–1568. <https://doi.org/10.1111/jdv.14390>
46. Stanek A, Mosti G, Nematillaevich T et al. No more venous ulcers—what more can we do? *J Clin Med*. 2023;12(19):6153. <https://doi.org/10.3390/jcm12196153>
47. Kim ES, Sharma AM, Scissons R et al. Interpretation of peripheral arterial and venous Doppler waveforms: a consensus statement from the Society for Vascular Medicine and Society for Vascular Ultrasound. *Vasc Med*. 2020;25(5):484–506. <https://doi.org/10.1177/1358863X20937665>
48. Kim HJ, Kim WJ, Lee HS et al. Clinical utility of skin perfusion pressure measurement in diabetic foot wounds: An observational study. *Medicine*. 2022;101(36):e30454. <https://doi.org/10.1097/MD.00000000000030454>
49. Baltrūnas T, Mosenko V, Mackevičius A et al. The use of near-infrared spectroscopy in the diagnosis of peripheral artery disease: a systematic review. *Vascular*. 2022;30(4):715–727. <https://doi.org/10.1177/17085381211025174>
50. Joseph S, Munshi B, Agarini R et al. Near infrared spectroscopy in peripheral artery disease and the diabetic foot: A systematic review. *Diabetes Metab Res Rev*. 2022;38(7):e3571. <https://doi.org/10.1002/dmrr.3571>
51. Martinelli O, Alunno A, Drudi FM et al. Duplex ultrasound versus CT angiography for the treatment planning of lower-limb arterial disease. *J Ultrasound*. 2021;24(4):471–479. <https://doi.org/10.1007/s40477-020-00534-y>
52. Huthart S, Oates C, Allen J et al. Validation of a standardised duplex ultrasound classification system for the reporting and grading of peripheral arterial disease. *Eur J Vasc Endovasc Surg*. 2022;64(2–3):210–216. <https://doi.org/10.1016/j.ejvs.2022.04.013>
53. Konoeda H, Yamaki T, Hamahata A et al. Quantification of superficial venous reflux by duplex ultrasound—role of reflux velocity in the assessment the clinical stage of chronic venous insufficiency. *Annals of Vascular Diseases*. 2014;7(4):376–382. <https://doi.org/10.3400/avd.0a.14-00047>
54. Zygmunt JA. Duplex ultrasound for chronic venous insufficiency. *J Invasive Cardiol*. 2014;26(11):E149–155
55. Garcia R, Labropoulos N. Duplex ultrasound for the diagnosis of acute and chronic venous diseases. *Surg Clin N Am*. 2018;98(2):201–218. <https://doi.org/10.1016/j.suc.2017.11.007>
56. National Institute for Health and Care Excellence. Varicose veins: diagnosis and management 2013. [www.nice.org.uk/guidance/cg168](http://www.nice.org.uk/guidance/cg168) (accessed April 2024)
57. Wound Educators. Vascular evaluation 2013. <https://woundeducators.com/vascular-evaluation-venous-insufficiency/> (accessed April 2024)
58. Gohel MS, Heatley F, Liu X et al. A randomized trial of early endovenous ablation in venous ulceration. *N Engl J Med*. 2018;378(22):2105–2114. <https://doi.org/10.1056/NEJMoa1801214>
59. Davies AH. The seriousness of chronic venous disease: a review of real-world evidence. *Adv Ther*. 2019;36(Suppl 1):5–12. <https://doi.org/10.1007/s12325-019-0881-7>
60. Holloway S, Ahmajärvi K, Frescos N et al. Holistic management of wound-related pain: an overview of the evidence and recommendations for clinical practice. *J Wound Manage*. 2024;25(1). <https://doi.org/10.35279/jowm2024.25.01.sup01>
61. Erdal ES, Demirgüç A, Kabalci M et al. Evaluation of physical activity level and exercise capacity in patients with varicose veins and chronic venous insufficiency. *Phlebology*. 2021;36(8):636–643. <https://doi.org/10.1177/02683555211002339>
62. Stonko DP, Hicks CW. Current management of intermittent claudication. *Adv Surg*. 2023;57(1):103–113. <https://doi.org/10.1016/j.yasu.2023.04.009>
63. Australasian Lymphology Association. What is lymphoedema? 2024
64. Urbaneck T, Juško M, Kuczmik WB. Compression therapy for leg oedema in patients with heart failure. *ESC Heart Failure*. 2020;7(5):2012–2020. <https://doi.org/10.1002/ehf2.12848>
65. Clark AL, Cleland JGF. Causes and treatment of oedema in patients with heart failure. *Nat Rev Cardiol*. 2013;10(3):156–170. <https://doi.org/10.1038/nrcardio.2012.191>
66. Dineen R, Thompson CJ, Sherlock M. Hyponatraemia - presentations and management. *Clin Med*. 2017;17(3):263–269. <https://doi.org/10.7861/clinmedicine.17-3-263>
67. Dessources K, Aviki E, Leita MM. Lower extremity lymphedema in patients with gynecologic malignancies. *Int J Gynecol Cancer*. 2020;30(2):252–260. <https://doi.org/10.1136/ijgc-2019-001032>
68. Largeau B, Cracowski J, Lengellé C et al. Drug-induced peripheral oedema: an aetiology-based review. *Brit J Clinical Pharma*. 2021;87(8):3043–3055. <https://doi.org/10.1111/bcp.14752>
69. Goss JA, Greene AK. Sensitivity and specificity of the stemmer sign for lymphedema: a clinical lymphoscintigraphic study. *Plast Reconstr Surg Glob Open*. 2019;7(6):e2295. <https://doi.org/10.1097/GOX.00000000000002295>
70. Star A. Differentiating lower extremity wounds: arterial, venous, neurotrophic. *Semin Intervent Radiol*. 2018;35(05):399–405. <https://doi.org/10.1055/s-0038-1676362>
71. Sibbald RG, Woo K, Ayello EA. Increased bacterial burden and infection: the story of NERDS and STONES. *Adv Skin Wound Care*. 2006;19(8):447–461. <https://doi.org/10.1097/00129334-200610000-00012>
72. Dissemmond J, Gerber V, Lobmann R et al. Therapeutic index for local infections score (TILI): a new diagnostic tool. *J Wound Care*. 2020;29(12):720–726. <https://doi.org/10.12968/jowc.2020.29.12.720>
73. Swanson T, Ousey K, Haesler E et al. IWII wound infection in clinical practice consensus document: 2022 update. *J Wound Care*. 2022;31(Sup12):S10–S21. <https://doi.org/10.12968/jowc.2022.31.Sup12.S10>
74. Bonham PA. Swab cultures for diagnosing wound infections: a literature review and clinical guideline. *J Wound Care*. 2009;36(4):389–395. <https://doi.org/10.1097/WON.0b013e3181aaef7f>
75. Smith ME, Robinowitz N, Chauk P et al. Comparison of chronic wound culture techniques: swab versus curetted tissue for microbial recovery. *Br J Community Nurs*. 2014;19(Sup9):S22–S26. <https://doi.org/10.12968/bjcn.2014.19.Sup9.S22>
76. Copeland-Halperin LR, Kaminsky AJ, Bluefield N et al. Sample procurement for cultures of infected wounds: a systematic review. *J Wound Care*. 2016;25(Sup4):S4–S10. <https://doi.org/10.12968/jowc.2016.25.Sup4.S4>
77. Haalboom M, Blokhuis Arkes MHE, Beuk RJ et al. Wound swab and wound biopsy yield similar culture results. *Wound Rep Regen*. 2018;26(2):192–199. <https://doi.org/10.1111/wrr.12629>
78. Moelleken M, Jockenhöfer F, Benson S et al. Prospective clinical study on the efficacy of bacterial removal with mechanical debridement in and around chronic leg ulcers assessed with fluorescence imaging. *Int Wound J*. 2020;17(4):1011–1018. <https://doi.org/10.1111/iwj.13345>
79. Sandy-Hodgetts K, Andersen CA, Al-Jalodi O et al. Uncovering the high prevalence of bacterial burden in surgical site wounds with point-of-care fluorescence imaging. *Int Wound J*. 2022;19(6):1438–1448. <https://doi.org/10.1111/iwj.13737>
80. Koo D-Y, Namgoong S, Han S-K et al. Diagnostic accuracy of a fluorescence imaging device in diabetic wounds: a pilot study using a tissue culture system. *Wounds*. 2023;35(7):e218–e223. <https://doi.org/10.25270/wnds/23002>
81. Serena TE, Snyder RJ, Bowler PG. Use of fluorescence imaging to optimize location of tissue sampling in hard-to-heal wounds. *Front Cell Infect Microbiol*. 2023;12:1070311. <https://doi.org/10.3389/fcimb.2022.1070311>
82. MacLeod BG, Klarich CS, Wessman LL et al. Fluorescent imaging as a component of diagnosing pyoderma gangrenosum: a case report. *Adv Skin Wound Care*. 2022;35(6):1–6. <https://doi.org/10.1097/01.ASW.0000820248.26138.bc>
83. Tang JC, Vivas A, Rey A et al. Atypical ulcers: wound biopsy results from a university wound pathology service. *Ostomy Wound Manage*. 2012;58(6):20–22, 24, 26–29
84. Ansert E, Tickner A, Cohen D et al. Understanding the zebras of wound care: an overview of atypical wounds. *Wounds*. 2022;34(5):124–134. <https://doi.org/10.25270/wnds/2022.124134>
85. Schahab N, Sudan S, Schaefer C et al. Sleep apnoea is common in severe peripheral arterial disease. *PLoS One*. 2017;12(7):e0181733. <https://doi.org/10.1371/journal.pone.0181733>

- 86.** Launois R. Health-related quality-of-life scales specific for chronic venous disorders of the lower limbs. *J Vasc Surg.* 2015;3(2):219-227.e3. <https://doi.org/10.1016/j.jvs.2014.08.005>
- 87.** González-Consuegra RV, Verdú J. Quality of life in people with venous leg ulcers: an integrative review. *Quality of life in people with venous leg ulcers.* *J Adv Nurs.* 2011;67(5):926-944. <https://doi.org/10.1111/j.1365-2648.2010.05568.x>
- 88.** National Institute for Health and Care Excellence. Multimorbidity: clinical assessment and management 2016. [www.nice.org.uk/guidance/ng56](http://www.nice.org.uk/guidance/ng56) (accessed April 2024)
- 89.** Gethin G, Killeen F, Devane D. Heterogeneity of wound outcome measures in RCTs of treatments for VLUs: a systematic review. *J Wound Care.* 2015;24(5):211-226. <https://doi.org/10.12968/jowc.2015.24.5.211>
- 90.** Murphy MA, Joyce WP, Condrón C et al. A reduction in serum cytokine levels parallels healing of venous ulcers in patients undergoing compression therapy. *Eur J Vas Endovas Surg.* 2002;23(4):349-352. <https://doi.org/10.1053/ejvs.2002.1597>
- 91.** Beidler SK, Douillet CD, Berndt DF et al. Multiplexed analysis of matrix metalloproteinases in leg ulcer tissue of patients with chronic venous insufficiency before and after compression therapy. *Wound Rep Regen.* 2008;16(5):642-648. <https://doi.org/10.1111/j.1524-475X.2008.00415.x>
- 92.** Beidler SK, Douillet CD, Berndt DF et al. Inflammatory cytokine levels in chronic venous insufficiency ulcer tissue before and after compression therapy. *J Vasc Surg.* 2009;49(4):1013-1020. <https://doi.org/10.1016/j.jvs.2008.11.049>
- 93.** O'Meara S, Cullum N, Nelson EA et al. Compression for venous leg ulcers. *Cochrane Dat Syst Rev.* 2012. <https://doi.org/10.1002/14651858.CD000265.pub3>
- 94.** Shi C, Dumville JC, Cullum N et al. Compression bandages or stockings versus no compression for treating venous leg ulcers. *Cochrane Dat Syst Rev.* 2021;2021(7). <https://doi.org/10.1002/14651858.CD013397.pub2>
- 95.** Levick JR, Michel CC. Microvascular fluid exchange and the revised Starling principle. *Cardiovasc Res.* 2010;87(2):198-210. <https://doi.org/10.1093/cvr/cvq062>
- 96.** Mosti G, Partsch H. Bandages or double stockings for the initial therapy of venous oedema? A randomized, controlled pilot study. *Eur J Vasc Endovasc Surg.* 2013;46(1):142-148. <https://doi.org/10.1016/j.ejvs.2013.04.015>
- 97.** Mosti G, Iabichella ML, Partsch H. Compression therapy in mixed ulcers increases venous output and arterial perfusion. *J Vasc Surg.* 2012;55(1):122-128. <https://doi.org/10.1016/j.jvs.2011.07.071>
- 98.** Blair SD, Wright DD, Backhouse CM et al. Sustained compression and healing of chronic venous ulcers. *BMJ.* 1988;297(6657):1159-1161. <https://doi.org/10.1136/bmj.297.6657.1159>
- 99.** Milic DJ, Zivic SS, Bogdanovic DC et al. The influence of different sub-bandage pressure values on venous leg ulcers healing when treated with compression therapy. *J Vasc Surg.* 2010;51(3):655-661. <https://doi.org/10.1016/j.jvs.2009.10.042>
- 100.** Wittens C, Davies AH, Bækgaard N et al. Management of chronic venous disease. *Eur J Vasc Endovasc Surg.* 2015;49(6):678-737. <https://doi.org/10.1016/j.ejvs.2015.02.007>
- 101.** Harding K. Challenging passivity in venous leg ulcer care – the ABC model of management. *Int Wound J.* 2016;13(6):1378-1384. <https://doi.org/10.1111/iwj.12608>
- 102.** Hinchliffe RJ, Forsythe RO, Apelqvist J et al. Guidelines on diagnosis, prognosis, and management of peripheral artery disease in patients with foot ulcers and diabetes (IWGDF 2019 update). *Diabetes Metab Res Rev.* 2020;36 Suppl 1:e3276. <https://doi.org/10.1002/dmrr.3276>
- 103.** Bonham PA, Flemister BG, Droste LR et al. 2014 guideline for management of wounds in patients with lower-extremity arterial disease (LEAD): an executive summary. *J Wound Care.* 2016;43(1):23-31. <https://doi.org/10.1097/WON.0000000000000193>
- 104.** Lim SLX, Chung RE, Holloway S et al. Modified compression therapy in mixed arterial-venous leg ulcers: An integrative review. *Int Wound J.* 2021;18(6):822-842. <https://doi.org/10.1111/iwj.13585>
- 105.** Chuter V, Schaper N, Mills J et al. Effectiveness of bedside investigations to diagnose peripheral artery disease among people with diabetes mellitus: A systematic review. *Diabetes Metab Res Rev.* 2024;40(3):e3683. <https://doi.org/10.1002/dmrr.3683>
- 106.** Rabe E, Partsch H, Morrison N et al. Risks and contraindications of medical compression treatment: critical reappraisal (an international consensus statement). *Phlebology.* 2020;35(7):447-460. <https://doi.org/10.1177/02683555200909066>
- 107.** Mosti G, Namislo A, Benigni JP et al. Indications, contraindications, side effects, general assessment, and outlook for the future: an International Compression Club survey. *Phlebology.* 2024;02683555241228899. <https://doi.org/10.1177/02683555241228899>
- 108.** Cooper KL. Care of the lower extremities in patients with acute decompensated heart failure. *Crit Care Nurs.* 2011;31(4):21-29. <https://doi.org/10.4037/ccn2011337>
- 109.** Niklasson A, Maher J, Patil R et al. Living with heart failure: patient experiences and implications for physical activity and daily living. *ESC Heart Fail.* 2022;9(2):1206-1215. <https://doi.org/10.1002/ehf2.13795>
- 110.** New York Heart Association. New York Heart Association (NYHA) Classification 2018. <https://manual.jointcommission.org/releases/TJC2018A/DataElem0439.html> (accessed April 2024)
- 111.** Nasu T, Matsumoto S, Fujimoto W et al. The safety and efficacy of compression therapy in patients with stable heart failure. *Int J Cardiol Heart Vasc.* 2024;50:101343. <https://doi.org/10.1016/j.ijcha.2024.101343>
- 112.** Chassagne F, Helouin-Desenne C, Molimard J et al. Superimposition of elastic and nonelastic compression bandages. *J Vasc Surg Venous Lymphat Disord.* 2017;5(6):851-858. <https://doi.org/10.1016/j.jvs.2017.07.006>
- 113.** Ehmann S, Ortega AE, Hettrick H. Textile composition, not number of layers, impacts interphase pressure and static stiffness index: A pragmatic, comparative analysis of the in vivo interphase pressure of 7 different 2-layer cohesive bandage kits in healthy volunteers. *Wound Manag Prev.* 2023;69(2):14-25
- 114.** Ashby RL, Gabe R, Ali S et al. Clinical and cost-effectiveness of compression hosiery versus compression bandages in treatment of venous leg ulcers (Venous leg Ulcer Study IV, VenUS IV): a randomised controlled trial. *Lancet.* 2014;383(9920):871-879. [https://doi.org/10.1016/S0140-6736\(13\)62368-5](https://doi.org/10.1016/S0140-6736(13)62368-5)
- 115.** Bjork R, Ehmann S. S.T.R.I.D.E. professional guide to compression garment selection for the lower extremity. *J Wound Care.* 2019;28(Sup6a):1-44. <https://doi.org/10.12968/jowc.2019.28.Sup6a.51>
- 116.** Partsch H, Clark M, Mosti G et al. Classification of compression bandages: practical aspects. *Dermatol Surg.* 2008;34(5):600-609. <https://doi.org/10.1111/j.1524-4725.2007.34116.x>
- 117.** Mosti GB, Mattaliano V. Simultaneous changes of leg circumference and interface pressure under different compression bandages. *Eur J Vasc Endovasc Surg.* 2007;33(4):476-482. <https://doi.org/10.1016/j.ejvs.2006.11.035>
- 118.** Lurie F, Kistner R. Variability of interface pressure produced by ready-to-wear compression stockings. *Phlebology.* 2014;29(2):105-108. <https://doi.org/10.1258/phleb.2012.012045>
- 119.** Thomas S. *Wound Management and Dressings.* London: Pharmaceutical Press; 1990
- 120.** Hopkins A, Bull R, Worboys F. Needing more: the case for extra high compression for tall men in UK leg ulcer management. *Vein Lymphatic.* 2017;6(1). <https://doi.org/10.4081/vl.2017.6630>
- 121.** Calne S, Martin R, Day K et al. Compression in venous leg ulcers: a WUWH consensus document. *Wounds International* 2009. <https://woundsinternational.com/consensus-documents/compression-venous-leg-ulcers-wuwhs-consensus-document/> (accessed April 2024)
- 122.** Partsch H. The use of pressure change on standing as a surrogate measure of the stiffness of a compression bandage. *Eur J Vasc Endovasc Surg.* 2005;30(4):415-421. <https://doi.org/10.1016/j.ejvs.2005.06.002>
- 123.** Partsch H. The Static Stiffness Index: a simple method to assess the elastic property of compression material in vivo. *Dermatol Surg.* 2006;31(6):625-630. <https://doi.org/10.1111/j.1524-4725.2005.31604>
- 124.** Partsch H, Menzinger G, Mostbeck A. Inelastic compression increases venous ejection fraction more than elastic bandages in patients with superficial venous reflux. *Dermatologic Surgery.* 1999;25(9):695-700. <https://doi.org/10.1046/j.1524-4725.1999.98040.x>
- 125.** 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(6):287-294. <https://doi.org/10.1258/phleb.2008.008009>
- 126.** Partsch B, Mayer W, Partsch H. Improvement of ambulatory venous hypertension by narrowing of the femoral vein in congenital absence of venous valves. *Phlebology.* 1992;7(3):101-104. <https://doi.org/10.1177/026835559200700304>

- 127.** Mosti G. Elastic or inelastic compression in patients with leg ulcer and restricted mobility? *Vein Lymphatic*. 2013;2(2):20. <https://doi.org/10.4081/vl.2013.e20>
- 128.** Dale JJ, Ruckley CV, Gibson B et al. Multi-layer Compression: Comparison of Four Different Four-layer Bandage Systems Applied to the Leg. *Euro J Vasc Endovasc Surg*. 2004;27(1):94–99. <https://doi.org/10.1016/j.ejvs.2003.10.014>
- 129.** Benigni J-P, Uhl J-F, Filori P et al. Adjustable compression wraps: stretch, interface pressures and static stiffness indices. *Int Angiol*. 2023;42(3). <https://doi.org/10.23736/S0392-9590.23.04957-X>
- 130.** Ehmann S, Ortega AE, Hettrick H. Textile composition, not number of layers, impacts interphase pressure and static stiffness index: A pragmatic, comparative analysis of the in vivo interphase pressure of 7 different 2-layer cohesive bandage kits in healthy volunteers. *Wound Manag Prev*. 2023;69(2):14–25
- 131.** Mosti G, Cavezzi A, Partsch H et al. Adjustable velcro compression devices are more effective than inelastic bandages in reducing venous edema in the initial treatment phase: a randomized controlled trial. *Eur J Vasc Endovasc Surg*. 2015;50(3):368–374. <https://doi.org/10.1016/j.ejvs.2015.05.014>
- 132.** Mosti G, Partsch H. Inelastic bandages maintain their hemodynamic effectiveness over time despite significant pressure loss. *J Vasc Surg*. 2010;52(4):925–931. <https://doi.org/10.1016/j.jvs.2010.04.081>
- 133.** Wong IKY, Man MBL, Chan OSH et al. Comparison of the interface pressure and stiffness of four types of compression systems. *J Wound Care*. 2012;21(4):161–167. <https://doi.org/10.12968/jowc.2012.21.4.161>
- 134.** Partsch H, Schuren J, Mosti G et al. The Static Stiffness Index: an important parameter to characterise compression therapy in vivo. *J Wound Care*. 2016;25 Suppl 9:S4–S10. <https://doi.org/10.12968/jowc.2016.25.Sup9.S4>
- 135.** Ehmann S, Ortega AE, Hettrick H. Textile composition, not number of layers, impacts interphase pressure and static stiffness index: A pragmatic, comparative analysis of the in vivo interphase pressure of 7 different 2-layer cohesive bandage kits in healthy volunteers. *Wound Manag Prev*. 2023;69(2):14–25
- 136.** Protz K, Heyer K, Dörler M et al. Compression therapy: scientific background and practical applications. *J Deutsche Dermat Gesell*. 2014;12(9):794–801. <https://doi.org/10.1111/ddg.12405>
- 137.** Partsch H, Horakova MA. [Compression stockings in treatment of lower leg venous ulcer] *Wien Med Wochenschr*. 1994;144(10–11):242–249
- 138.** Mosti G, Mattaliano V, Partsch H. The influence of different sub-bandage pressure values on venous leg ulcers healing when treated with compression therapy. *Dermatol Surg*. 2008;34(5):631–639. <https://doi.org/10.1111/j.1524-4725.2007.34119.x>
- 139.** Brizzio E, Amsler F, Lun B et al. Comparison of low-strength compression stockings with bandages for the treatment of recalcitrant venous ulcers. *J Vasc Surg*. 2010;51(2):410–416. <https://doi.org/10.1016/j.jvs.2009.08.048>
- 140.** Junger M, Partsch H, Ramelet A et al. Efficacy of a ready-made tubular compression device versus short-stretch compression bandages in the treatment of venous leg ulcers. *Wounds*. 2004;106(10):313–320
- 141.** Rosell M, Haynes S, Hall T. Adopting compression strapping technique to enhance compression therapy in hard-to-heal leg ulcers. *Wounds UK* 2019. <https://wounds-uk.com/journal-articles/adopting-compression-strapping-technique-enhance-compression-therapy-hard-heal-leg-ulcers/> (accessed April 2024)
- 142.** Mosti G. Stiffness of compression devices. *Veins and Lymphatics*. 2013;2(1):e1–e1. <https://doi.org/10.4081/vl.2013.e1>
- 143.** Hopkins A, Worboys F, Bull R et al. Compression strapping: the development of a novel compression technique to enhance compression therapy and healing for 'hard-to-heal' leg ulcers. *Int Wound J*. 2011;8(5):474–483. <https://doi.org/10.1111/j.1742-481X.2011.00819.x>
- 144.** Charles H. Using compression bandages in the treatment of venous leg ulceration. *Prof Nurse*. 2001;17(2):123–125
- 145.** Finnie A. Bandages and bandaging techniques for compression therapy. *Br J Community Nurs*. 2002;7(3):134–142. <https://doi.org/10.12968/bjcn.2002.7.3.10212>
- 146.** Schuren J, Mohr K. Pascal's law and the dynamics of compression therapy: a study on healthy volunteers. *Int Angiol*. 2010;29(5):431–435
- 147.** Mosti G, Partsch H. Compression stockings with a negative pressure gradient have a more pronounced effect on venous pumping function than graduated elastic compression stockings. *Eur J Vasc Endovasc Surg*. 2011;42(2):261–266. <https://doi.org/10.1016/j.ejvs.2011.04.023>
- 148.** Couzan S, Leizorovicz A, Laporte S et al. A randomized double-blind trial of upward progressive versus degenerative compression stockings in patients with moderate to severe chronic venous insufficiency. *J Vasc Surg*. 2012;56(5):1344–1350.e1. <https://doi.org/10.1016/j.jvs.2012.02.060>
- 149.** Rother U, Grussler A, Griesbach C et al. Safety of medical compression stockings in patients with diabetes mellitus or peripheral arterial disease. *BMJ Open Diab Res Care*. 2020;8(1):e001316. <https://doi.org/10.1136/bmjdr-2020-001316>
- 150.** Passman MA, McLafferty RB, Lentz MF et al. Validation of Venous Clinical Severity Score (VCSS) with other venous severity assessment tools from the American Venous Forum, National Venous Screening Program. *J Vasc Surg*. 2011;54(6):2S–9S. <https://doi.org/10.1016/j.jvs.2011.05.117>
- 151.** Atkin L, Bučko Z, Montero EC et al. Implementing TIMERS: the race against hard-to-heal wounds. *J Wound Care*. 2019;28(S3a):S1–S50. <https://doi.org/10.12968/jowc.2019.28.Sup3a.S1>
- 152.** Isoherranen K, Conde E, Atkin L et al. Lower leg ulcer diagnosis and principles of treatment. *JOWM*. 2023. <https://doi.org/10.35279/jowm2023.24.02.sup01>
- 153.** Mayer D, Tettelbach WH, Ciprandi G et al. Best practice for wound debridement. *J Wound Care*. 2024;33(S6c). <https://doi.org/10.12968/jowc.2024.33.Sup6b.S1>
- 154.** Sibbald RG, Elliott JA, Persaud-Jaimangal R et al. Wound bed preparation 2021. *Adv Skin Wound Care*. 2021;34(4):183–195. <https://doi.org/10.1097/01.ASW.0000733724.87630.d6>
- 155.** Stiehl JB. Early wound bed preparation: irrigation and debridement. *J Wound Care*. 2021;30(Sup9):S8–S16. <https://doi.org/10.12968/jowc.2021.30.Sup9.S8>
- 156.** Thomas DC, Tsu CL, Nain RA et al. The role of debridement in wound bed preparation in chronic wound: A narrative review. *Ann Med Surg*. 2021;71:102876. <https://doi.org/10.1016/j.amsu.2021.102876>
- 157.** Todd M. Compression therapy for chronic oedema and venous leg ulcers: CoFlex TLC Calamine. *Br J Nurs*. 2019;28(12):S32–S37. <https://doi.org/10.12968/bjon.2019.28.12.S32>
- 158.** Jonker L, Todhunter J, Robinson L et al. Open-label, randomised, multicentre crossover trial assessing two-layer compression bandaging for chronic venous insufficiency: results of the APRICOT trial. *Br J Community Nurs*. 2020;25(Sup6):S6–S13. <https://doi.org/10.12968/bjcn.2020.25.Sup6.S6>
- 159.** Atkin L. Zinc: the benefits to venous eczema and ulceration. *Wounds UK* 2016. <https://wounds-uk.com/journal-articles/zinc-the-benefits-to-venous-eczema-and-ulceration/> (accessed April 2024)
- 160.** Orr L, Klement KA, McCrossin L et al. A systematic review and meta-analysis of exercise intervention for the treatment of calf muscle pump impairment in individuals with chronic venous insufficiency. *Ostomy Wound Manage*. 2017;63(8):30–43. <https://doi.org/10.25270/owm.2017.08.3043>
- 161.** Kirsner RS. Exercise for leg ulcers: "working out" the nature of venous ulcers. *JAMA Dermatol*. 2018;154(11):1257. <https://doi.org/10.1001/jamadermatol.2018.2926>
- 162.** Zhang Q, Lu L, Song JL et al. Effects of exercise in treating patients with venous leg ulcers: a systematic review and meta-analysis. *Int Wound J*. 2023;20(5):1776–1783. <https://doi.org/10.1111/iwj.14020>
- 163.** Davies JA, Bull RH, Farrelly JJ et al. A home-based exercise programme improves ankle range of motion in long-term venous ulcer patients. *Phlebology*. 2007;22(2):86–89. <https://doi.org/10.1258/026835507780346178>
- 164.** Bendermacher BL, Willigendael EM, Teijink JA et al. Supervised exercise therapy versus non-supervised exercise therapy for intermittent claudication. *Cochrane Dat Syst Rev*. 2006;CD005263. <https://doi.org/10.1002/14651858.CD005263.pub2>
- 165.** Hageman D, Marijn M, Houten VD et al. Supervised exercise therapy: it does work, but how to set up a program? *J Cardiovasc Surg*. 2017;58(2). <https://doi.org/10.23736/S0021-9509-16.09825-6>
- 166.** Li T, Yang S, Hu F et al. Effects of ankle pump exercise frequency on venous hemodynamics of the lower limb. *Clin Hemorheol Microcirc*. 2020;76(1):111–120. <https://doi.org/10.3233/CH-200860>

- 167.** Da Matta ES, Mosti G, Corralo VDS et al. Effects of lower limb muscle strengthening on interface pressure in older adults undergoing inelastic compression: Randomized controlled clinical trial. *Phlebology*. 2024;02683555241235042. <https://doi.org/10.1177/02683555241235042>
- 168.** Yim E, Kirsner RS, Gailey RS et al. Effect of physical therapy on wound healing and quality of life in patients with venous leg ulcers: a systematic review. *JAMA Dermatol*. 2015;151(3):320. <https://doi.org/10.1001/jamadermatol.2014.3459>
- 169.** Lane R, Harwood A, Watson L et al. Exercise for intermittent claudication. *Cochrane Dat Syst Rev*. 2017;2017(12). <https://doi.org/10.1002/14651858.CD000990.pub4>
- 170.** McDermott MM. Exercise training for intermittent claudication. *J Vasc Surg*. 2017;66(5):1612–1620. <https://doi.org/10.1016/j.jvs.2017.05.111>
- 171.** Hageman D, Fokkenrood HJ, Gommans LN et al. Supervised exercise therapy versus home-based exercise therapy versus walking advice for intermittent claudication. *Cochrane Dat Syst Rev*. 2018;2018(4). <https://doi.org/10.1002/14651858.CD005263.pub4>
- 172.** Allman-Farinelli M. Obesity and venous thrombosis: a review. *Semin Thromb Hemost*. 2011;37(08):903–907. <https://doi.org/10.1055/s-0031-1297369>
- 173.** Belczak SQ, Neves Ramos R, Pereira De Godoy JM. Association between obesity and the aggravation of limited range of ankle mobility in chronic venous disease. *Phlebology*. 2022;37(3):196–199. <https://doi.org/10.1177/02683555211055350>
- 174.** Morrell J, Fox KAA. Prevalence of abdominal obesity in primary care: the IDEA UK study. *Int J Clin Pract*. 2009;63(9):1301–1307. <https://doi.org/10.1111/j.1742-1241.2009.02126.x>
- 175.** Ruiz AJ, Aschner PJ, Puerta MF et al. [IDEA study (International Day for the Evaluation of Abdominal Obesity): primary care study of the prevalence of abdominal obesity and associated risk factors in Colombia]. *Biomedica*. 2012;32(4):610–616. <https://doi.org/10.1590/S0120-41572012000400016>
- 176.** Belczak CEQ, De Godoy JMP, Belczak SQ et al. Obesity and worsening of chronic venous disease and joint mobility. *Phlebology*. 2014;29(8):500–504. <https://doi.org/10.1177/0268355513492510>
- 177.** Meulendijks AM, Franssen WMA, Schoonhoven L et al. A scoping review on chronic venous disease and the development of a venous leg ulcer: the role of obesity and mobility. *J Vasc Surg*. 2020;29(3):190–196. <https://doi.org/10.1016/j.jtv.2019.10.002>
- 178.** Millen RN, Thomas KN, Versteeg MPT et al. Popliteal vein compression, obesity, and chronic venous disease. *J Vasc Surg*. 2022;10(1):200–208.e2. <https://doi.org/10.1016/j.jvsv.2021.05.013>
- 179.** Comerota AJ. Intermittent pneumatic compression: physiologic and clinical basis to improve management of venous leg ulcers. *J Vasc Surg*. 2011;53(4):1121–1129. <https://doi.org/10.1016/j.jvs.2010.08.059>
- 180.** Nelson EA, Hillman A, Thomas K. Intermittent pneumatic compression for treating venous leg ulcers. *Cochrane Dat Syst Rev*. 2014;2014(6). <https://doi.org/10.1002/14651858.CD001899.pub4>
- 181.** Horn C, Fierro A, Lantis IJ. Use of negative pressure wound therapy for the treatment of venous leg ulcers. *Wounds*. 2023;35(6):117–125. <https://doi.org/10.25270/wnds/23035>
- 182.** Abu-Own A, Cheate T, Scurr JH et al. Effects of intermittent pneumatic compression of the foot on the microcirculatory function in arterial disease. *Eur J Vasc Endovasc Surg*. 1993;7(5):488–492. [https://doi.org/10.1016/S0950-821X\(05\)80358-5](https://doi.org/10.1016/S0950-821X(05)80358-5)
- 183.** Swedish Council on Health Technology Assessment. Peripheral arterial disease – diagnosis and treatment: a systematic review. Stockholm: Swedish Council on Health Technology Assessment (SBU); 2008
- 184.** 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. <https://doi.org/10.1016/j.jvs.2005.06.004>
- 185.** Oresanya L, Mazzei M, Bashir R et al. Systematic review and meta-analysis of high-pressure intermittent limb compression for the treatment of intermittent claudication. *J Vasc Surg*. 2018;67(2):620–628.e2. <https://doi.org/10.1016/j.jvs.2017.11.044>
- 186.** Zaleska MT, Olszewski WL, Ross J. The long-term arterial assist intermittent pneumatic compression generating venous flow obstruction is responsible for improvement of arterial flow in ischemic legs. *PLoS ONE*. 2019;14(12):e0225950. <https://doi.org/10.1371/journal.pone.0225950>
- 187.** Patel N, Khakha R, Gibbs J. Review article: anti-embolism stockings. *J Orthop Surg (Hong Kong)*. 2013;21(3):361–364. <https://doi.org/10.1177/230949901302100319>
- 188.** National Institute for Health and Care Excellence. Venous thromboembolism in over 16s: reducing the risk of hospital-acquired deep vein thrombosis or pulmonary embolism 2018. [www.nice.org.uk/guidance/NG89/chapter/recommendations](http://www.nice.org.uk/guidance/NG89/chapter/recommendations) (accessed April 2024)
- 189.** Gohel MS, Barwell JR, Taylor M et al. Long term results of compression therapy alone versus compression plus surgery in chronic venous ulceration (ESCHAR): randomised controlled trial. *BMJ*. 2007;335(7610):83. <https://doi.org/10.1136/bmj.39216.542442.BE>
- 190.** Davies HOB, Bradbury AW. The EVRA trial: new hope for people with venous leg ulcers? *Eur J Vasc Endovasc Surg*. 2019;57(2):163–164. <https://doi.org/10.1016/j.ejvs.2018.07.030>
- 191.** Pihlaja T, Kosunen E, Ohtonen P et al. Sub-ulcer foam sclerotherapy in patients with venous leg ulcer, analysis and technical aspects of 134 consecutive patients. *Int J Low Extrem Wound*. 2024. <https://doi.org/10.1177/15347346241245765>
- 192.** Lantis JC, Boone D, Lee L et al. The effect of percutaneous intervention on wound healing in patients with mixed arterial venous disease. *Ann Vasc Surg*. 2011;25(1):79–86. <https://doi.org/10.1016/j.avsg.2010.09.006>
- 193.** Self Care Forum. The self-care continuum 2020. [www.selfcareforum.org/wp-content/uploads/2012/08/The-self-care-continuum.pdf](http://www.selfcareforum.org/wp-content/uploads/2012/08/The-self-care-continuum.pdf) (accessed July 2024)
- 194.** Parfitt G, Blackburn J, Ousey K. Exploring concepts and current evidence of shared and self-care in the management of lower limb wounds. *Wounds UK* 2021. <https://wounds-uk.com/journal-articles/exploring-concepts-and-current-evidence-shared-and-self-care-management-lower-limb-wounds/> (accessed July 2024)
- 195.** Miller KL. Patient centered care: A path to better health outcomes through engagement and activation. *NRE*. 2016;39(4):465–470. <https://doi.org/10.3233/NRE-161378>
- 196.** Street RL, Makoul G, Arora NK et al. How does communication heal? Pathways linking clinician–patient communication to health outcomes. *Pat Ed Counseling*. 2009;74(3):295–301. <https://doi.org/10.1016/j.pec.2008.11.015>
- 197.** Protz K, Dissemmond J, Karbe D et al. Increasing competence in compression therapy for venous leg ulcers through training and exercise measured by a newly developed score-Results of a randomised controlled intervention study. *Wound Repair Regen*. 2021;29(2):261–269. <https://doi.org/10.1111/wrr.12899>
- 198.** Criqui MH, Denenberg JO, Bergan J et al. Risk factors for chronic venous disease: the San Diego Population Study. *J Vasc Surg*. 2007;46(2):331–337. <https://doi.org/10.1016/j.jvs.2007.03.052>
- 199.** Van Cott EM, Khor B, Zehnder JL, Factor V L Eiden. *American J Hematol*. 2016;91(1):46–49. <https://doi.org/10.1002/ajh.24222>
- 200.** Eklöf B, Rutherford RB, Bergan JJ et al. Revision of the CEAP classification for chronic venous disorders: Consensus statement. *J Vasc Surg*. 2004;40(6):1248–1252. <https://doi.org/10.1016/j.jvs.2004.09.027>
- 201.** Almeida JJ, Wakefield T, Kabnick LS et al. Use of the clinical, etiologic, anatomic, and pathophysiologic classification and venous clinical severity score to establish a treatment plan for chronic venous disorders. *J Vasc Surg Venous Lymphatic Dis*. 2015;3(4):456–460. <https://doi.org/10.1016/j.jvsv.2015.05.007>
- 202.** Vasquez MA, Rabe E, McLafferty RB et al. Revision of the venous clinical severity score: venous outcomes consensus statement: special communication of the American Venous Forum Ad Hoc Outcomes Working Group. *J Vasc Surg*. 2010;52(5):1387–1396. <https://doi.org/10.1016/j.jvs.2010.06.161>
- 203.** Cerqueira L de O, Duarte EG, Barros AL de S et al. Wifl classification: the Society for Vascular Surgery lower extremity threatened limb classification system, a literature review. *J Vasc Bras*. 2020;19:e20190070. <https://doi.org/10.1590/1677-5449.190070>
- 204.** Burian EA, Karlsmark T, Nørregaard S et al. Wounds in chronic leg oedema. *Int Wound J*. 2022;19(2):411–425. <https://doi.org/10.1111/iwj.13642>
- 205.** Lee AJ, Dale JJ, Ruckley CV et al. Compression therapy: effects of posture and application techniques on initial pressures delivered by bandages of different physical properties. *Eur J Vasc Endovasc Surg*. 2006;31(5):542–552. <https://doi.org/10.1016/j.ejvs.2005.10.023>

