Best Practices for the Prevention and Treatment of Venous Leg Ulcers

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ABSTRACT

Chronic venous insufficiency is the most common cause of leg ulcers. Its incidence increases as the population ages. Managing venous leg ulcers involves treating the cause, optimizing local wound care, and addressing patient-centered concerns. The cornerstone of the diagnosis of chronic venous insufficiency includes demonstrating venous disease. The clinician must rule out significant coexisting arterial disease by performing a thorough clinical assessment and obtaining an ankle brachial pressure index. The most important aspect of treatment is resolving edema through high compression therapy for those individuals with an ankle brachial pressure index greater than or equal to 0.8. Other components of successful chronic venous insufficiency management include increasing mobility and medical management. Selected patients may respond to surgery, biologicals, adjunctive therapies, and lifestyle enhancements. Twelve recommendations are made incorporating current best clinical practices and expert opinion with available research. The approach to venous disease is best accomplished through a multidisciplinary team that revolves around the active participation of patients and their families. The authors' intent is to provide a practical, easy-to-follow guide to allow healthcare professionals to provide best clinical practices.

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In recent years, the Canadian Association of Wound Care (CAWC) has been involved in issues surrounding venous leg ulcer management, and has developed 12 recommendations for best clinical practices in patient care. This information is presented in a workable and effective protocol to address diagnosis, treatment, and prevention of venous leg ulcers (see Table 1 for a quick reference guide).

Recommendation 1

Obtain a Careful History to Determine the Venous Characteristics and to Rule Out Other Diagnoses. Assess Pain and Identify the Systemic and Local Factors that May Impair Wound Healing

Venous disease history. Several important risk factors can be elicited from the patient history. Numerous occupations involving standing and sitting for prolonged periods of time place patients at increased risk for developing venous hypertension.[1] Obesity, multiple pregnancies, and a previous history of major leg trauma are acquired risk factors for chronic venous disease.[2] A history of deep vein thrombosis (DVT) or congenital weakness can lead to valve damage and may be an inciting event in edema production, other signs of venous insufficiency, and subsequent venous ulcer risk. Varicose veins, previous vein stripping, and sclerotherapy all suggest the abnormalities of the venous system that may contribute to the develop-

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TABLE 1
QUICK REFERENCE GUIDE TO THE 12 RECOMMENDATIONS FOR BEST PRACTICES IN THE PREVENTION AND TREATMENT OF VENOUS LEG ULCERS

1. Obtain a careful history to determine the venous characteristics and rule out other diagnoses. Assess pain and identify the systemic and local factors that may impair wound healing.
2. Determine the cause(s) of chronic venous insufficiency (CVI) based on etiology: abnormal valves (reflux), obstruction, or calf muscle pump failure.
3. Perform the ankle-brachial pressure index (ABPI) test on all patients with venous ulcers to help rule out significant arterial disease.
4. Implement high compression bandaging for the management of venous edema if the ABPI is ≥ 0.8.
5. Use graduated compression stockings to manage and prevent venous leg edema. Wearing stockings to decrease the frequency of ulcer recurrence is important.
6. Implement intermittent pneumatic compression therapy and/or elevation of the leg as an added benefit in managing venous edema and venous leg ulcers.
7. Consult with rehabilitation experts to maximize activity and mobility. Consider appropriate adjunctive therapies.
8. Assess for infection and treat if indicated.
9. Optimize the local wound healing environment: debridement, bacterial balance, and moisture balance. Use biological agents when the cause has been corrected and healing does not proceed at an expected rate.
10. Implement medical therapy if indicated for CVI (superficial and deep thrombosis, woody fibrosis).
11. Consider surgical management if significant superficial or perforator vein disease exists in the absence of extensive deep disease.
12. Communicate with the patient, the family, and the caregivers to establish realistic expectations for (non)healing. The presence or absence of a social support system is important for treatment and prevention of venous leg ulcers.

ment of venous leg ulcers. Varicosities on the surface may be due to superficial disease alone or in combination with perforator and/or deep venous ulceration.

Establishing a chronology of events is important. The onset of the ulcer may be traumatic or secondary to edema, infection, or a combination of factors. Asking the patient if he or she experiences leg swelling by the end of the day, and for how long this has been occurring, is important. The local skin is often extremely itchy and may breakdown with fluid exudation. This may be the inciting event for local infection. With time, the inner aspect of the ankle may become hyperpigmented with leakage of red blood cells, leaving behind hemosiderin and melanin deposition. Long-standing changes also may include thickening of the ankle with nonpitting edema (woody fibrosis) that does not resolve with the recumbent position.

The clinician should ask about previous topical treatments. Patients with venous disease are very susceptible to irritant and allergic reactions. Common allergens include latex, perfumes, lanolin, neomycin, and other topical antibiotics, as well as home remedies (including alternative medicines). If an ulcer has developed, a chronology of local treatments and previous bandaging will help the clinician to determine what aspects of care have been helpful or harmful.

Pain. Pain is associated with the venous ulcer. Pain also may result from phlebitis (superficial or deep), infection, or local wound factors (debridement, dressing changes, or sensitivity to one of the components of a dressing). Cofactors such as arterial disease may be responsible for pain. Arterial pain may be aggravated by limb elevation and produce intermittent claudication of the calf or a local tightness with walking. Venous disease is often associated with pain at the end of the day,

Ostomy/Wound Management 2001;47(2):34–50

KEY POINTS

- ❑ Chances are that the number of individuals with peripheral vascular disease and venous ulcers will increase at the same rate as the proportion of older adults.
- ❑ This extensive review of the literature and its resultant recommendations for practice serve as a reminder that prompt diagnosis and appropriate care are crucial to preventing unnecessary pain and suffering.
- ❑ Wound care experts familiar with the principles of venous ulcer prevention and treatment detailed in this article are encouraged to share them with healthcare professionals who are not, thereby reducing the incidence of persons experiencing a delay in receiving optimal care.

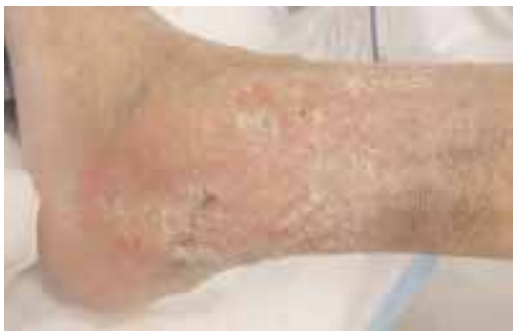

Figure 1
This photo demonstrates skin changes in a patient with chronic venous insufficiency.

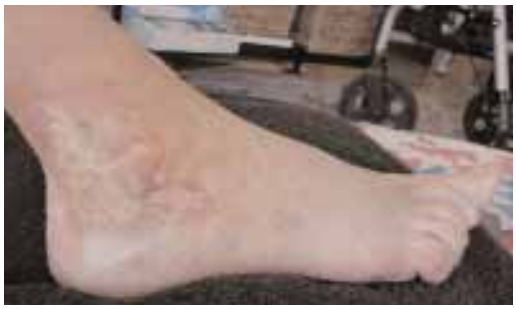

Figure 2
It is important to recognize atrophie blanche, which is shown above, because the skin is fragile and ulcers may develop after only minor trauma.

especially with prolonged standing or sitting. The resulting edema often is reduced by recumbency and elevation of the limb.

Factors that may affect wound healing. Margolis et al[3] defined several factors that were associated with an increased incidence of nonhealing venous leg ulcers. Four hundred thirty-three consecutive patients had a number of factors analyzed using a multiple variant logistic regression model. In order of odds ratio, the following six risk factors obtained statistical significance:

1. History of venous ligation or stripping – odds ratio: 4.58
2. Hip or knee surgery – odds ratio: 3.52
3. ABPI < 0.8 – odds ratio: 3.52
4. Fibrin (yellow > 50% of the ulcer base) – odds ratio: 3.42
5. Larger size (area) – odds ratio: 1.19
6. Longer duration in months – odds ratio: 1.09.

An odds ratio of 4.58 means that a patient with a history of venous ligation or stripping is 4.58 times less likely to heal by week 24 compared to patients who have not had this procedure done. In this analysis, diabetes mellitus, previous deep vein thrombosis, visible varicose veins, lipodermatosclerosis, and undermined wound margin did not make a significant difference.

Building blocks such as protein, vitamins, and trace metals are required to heal a wound. The functioning of the wound healing process requires an adequate supply of these building blocks. A nutritionist or dietitian should be consulted if nutritional deficiency is thought to be significant enough to possibly impair wound healing. Deficiencies in the intake of protein and vitamin intake are common in the elderly. Managing these deficiencies may make a difference between a healing and a nonhealing wound even in the presence of best clinical practices.

Drug history is important. Medications such as prednisone and immunosuppressive agents can have a profoundly negative influence. Many patients self-medicate with antiseptic cleaning agents that have been shown to be toxic to fibroblast cells. Similarly, patients may be applying potent topical steroids on the wound that are designed for the surrounding skin.

Lastly, patients and their caregivers may believe in the traditional dogma of leaving wounds open to the air allowing the formation of a dry crust that inhibits healing.

Periulcer assessment. The appearance of the surrounding skin is important in the clinical diagnosis of venous ulcers. Early venous insufficiency presents with pitting edema that should be distinguished from congestive heart failure and other systemic diseases. As a result of the deposition of iron and melanin in the skin, irregular pigmentation in the gaiter area (lower calf) develops as the disease progresses (see Figure 1). Lipodermatosclerosis (bound down appearance with atrophy, telangiectasia, hyperpigmentation, and hypopigmentation) often occurs later as the progressive deposition of fibrin in the deep dermis and fat results in a woody induration of the gaiter area of the calf. This may contribute to the appearance of the so-called "inverted champagne bottle leg." Often accompanying this is the appearance of atrophie blanche presenting as white areas of extremely thin skin dotted with tiny tortuous blood vessels. The recognition of atrophie blanche (see Figure 2) is important because the thin skin is fragile, and ulcers may develop after only minor trauma. Eczema, commonly known as "stasis dermatitis," may appear in the gaiter area. The "stasis" is, in fact, a misnomer, as true stasis of the blood does not exist, but pooling of fluid and extravasation of red blood cells is present in the underly-

TABLE 2
CLASSIFICATION OF CVI

Class	Type of CVI	Symptoms
0	Asymptomatic	Nil
1	Mild	Mild swelling, heaviness, local or generalized dilatation of subcutaneous veins
2	Moderate	Hyperpigmentation, moderate brawny edema, subcutaneous fibrosis
3	Severe	Chronic distal leg pain with ulceration or pre-ulcerative changes, eczematoid changes, and/or severe edema

From Iafrati M, Welch H, O'Donnell TF, Belkin M, Umphrey S, McLaughlin R.. Correlation of venous noninvasive tests with the Society for Vascular Surgery/International Society for Cardiovascular Surgery. Clinical classification of chronic venous insufficiency. Journal of Vascular Surgery; *1994;19(6):1001-1007.*

ing dermis that may lead to irritation of the skin. The characteristics of the skin and local surrounding structures may be grouped into four classes based on disease characteristics that often help define the strength of compression garments (see Table 2).

Recommendation 2

Determine the Cause(s) of Chronic Venous Insufficiency Based on Etiology: Abnormal Valves (Reflux), Obstruction, or Calf Muscle Pump Failure

Venous hypertension is primarily caused by one of three major pathologies, which include:

1. Reflux, known as valve dysfunction
2. Obstruction, which is either complete or partial blockage of the deep veins. Reflux and obstruction may co-exist.
3. Failure of calf muscle pump function related to decreased activity, paralysis, ankle joint deformity, or decreased range of motion.[4]

Chronic venous disease can be congenital or acquired. Recurrent thrombophlebitis may alert the clinician to the presence or protein C, S, or Factor 5 (Leiden) deficiency. Valve dysfunction may be due to a congenital weakness or acquired secondary to previous episodes of thrombophlebitis. Valves also can be damaged from previous trauma or infection. Outflow obstruction, such as increased local pressure, can result from obesity and pregnancy. Damage to the venous outflow system, especially in the pelvic region, may result from malignancy or radiotherapy.

The least reported cause of venous hypertension is musculoskeletal changes that can lead to calf muscle pump failure. The dynamics of the calf muscle pump are adversely affected by changes that often accompany major injuries, neurological disease, vascular insufficiency, myositis, and bone and joint pain. The calf muscles rapidly waste and weaken with disuse.[5] Even the change in gait related to a painful ulcer can exacerbate the venous hypertension and cause calf muscle disuse atrophy. Back et al[6] stated that a normal walking motion may be required for full functional activation of the calf muscle pump and that ankle dorsiflexion past the 90-degree position is needed for a normal walking motion.

Investigation of the venous system should start with a clinical examination. Many investigations are noninvasive and can assess venous function.

The simplest way to determine venous reflux is to have the patient lie flat and raise the leg 45 degrees to drain the blood from the long saphenous vein (approximately 30 seconds). A rubber tourniquet is then applied above the knee and the patient is asked to stand up. The tourniquet is released and if the long saphenous vein fills in less than 20 seconds, venous insufficiency is present (superficial or deep). A hand-held Doppler can be used to locate abnormal venous areas.[7] The patient should stand upright with weight on the other leg and the knee slightly flexed. The probe is placed over the saphenofemoral or saphenopopliteal junction, followed by manual squeezing and release of the calf muscle. A prolonged regurgitation indicates reflux. A similar procedure may be used as a rough indicator of perforator incompetence as well.[7]

Color duplex Dopplers are used in the vascular lab by combining pulsed ultrasound systems with real time D mode to examine flow patterns in precisely defined areas.[8] This technique can demonstrate the failure of a thrombosed vein to collapse under direct compression, as well as visualize the thrombus within the vessel wall and detect the absence of or abnormal venous pulsation on Doppler scanning. This technique is most accurate in identifying thrombi in the common femoral vein to the popliteal vein, but less reliable in the calf. Duplex systems

can be added to color frequency mapping, which allows instant visualization of blood flow and its direction to accurately assess reflux.[8]

One of the most common tests to determine the efficacy of the calf pump is air plethysmography (APG). Air plethysmography can differentiate between superficial and deep venous disease, assess the degree of valvular insufficiency and the efficiency of the calf muscle pump. This technique also can provide a noninvasive estimate of ambulatory venous pressure.[9]

Recommendation 3

Perform the Ankle-Brachial Pressure Index Test on All Patients with Venous Ulcers to Help Rule Out Significant Arterial Disease

A physical examination of the feet and legs will help to detect clinical signs of vascular compromise. Arterial disease is often marked by a vascular dilation (flush) that blanches with elevation. Loss of hair and thickened nails with decreased nail luster may be evident. On palpation, the foot is characteristically cold with a loss of pulses. Microcirculatory supply can be tested by pressing a finger on the dorsum of the dependent foot to produce a noticeable blanching. Normally, erythema should return within 5 seconds, and if a delay is noted, decreased local perfusion (microcirculation time) is present. Distal gangrene of the toes with a palpable pulse or adequate circulation may indicate microemboli from proximal atheromatous plaques. The ankle brachial pressure index (ABPI) is determined by a hand-held Doppler and blood pressure cuff (see Figure 3) and is important as part of the vascular assessment of the lower leg. The ABPI is an important component of lower leg assessment and should be performed only by a skilled healthcare practitioner (see Table 3).[10]

In the vascular lab, reasonable healability potential for patients requires a toe pressure of 30 mm Hg (in people with diabetes > 45 mm Hg) or a transcutaneous oxygen saturation of > 30% (see Table 4).[11] A palpable pedal pulse indicates approximately 80 mm Hg and should be sufficient for healing in most patients. Checking sequential arterial circulation for waveforms is important. A normal Doppler triphasic waveform will become blunted and biphasic/monophasic, and pressures will drop disproportionately distal to possible bipassable or dilatable lesions with angioplasty.

Any wound, acute or chronic, affected by ischemia as a result of severe arterial insufficiency, will not heal no matter what local measures are employed. If the arterial disease is considered uncorrectable or if the patient's general health precludes surgery, management becomes palliative.

Recommendation 4

Implement High Compression Bandaging for the Management of Venous Edema if the ABPI ≥ 0.8

All compression systems must create a pressure gradient from ankle to knee. The Law of Laplace (see Figure 4)

TABLE 3
ABPI PROCEDURE

Step 1
Ensure that the patient is lying flat and is comfortable.

Step 2
Secure the appropriate size blood pressure cuff around the arm (pediatric or oversized cuffs may be indicated).
Apply ultrasound gel over brachial pulse.
Slowly move the Doppler probe at a 45-degree angle to the flow over area until a good signal is obtained.
Inflate the cuff until Doppler signal disappears, then gradually release the pressure valve until the signal returns. This is the brachial systolic pressure.

Step 3
Examine the foot for posterior tibial and dorsalis pedis pulse using fingers and/or Doppler probe.

Step 4
Secure the blood pressure cuff just above the ankle.
Locate the posterior tibial and dorsalis pedis pulse using Doppler probe and gel.
Inflate the cuff until the signal disappears, then gradually release the pressure valve until the signal returns. Repeat with second pulse. This is the ankle systolic pressure

Step 5
To calculate the ABPI, divide the ankle systolic pressure by the brachial systolic pressure.
Caution: *Unusually high readings may be obtained in elderly or diabetic patients as the cuff may not fully compress the calcified vessels.*

Adapted from Moffatt C, O'Hare L. Ankle pulses are not sufficient to detect impaired arterial circulation in patients with leg ulcers. Journal of Wound Care.*1995;4(3):134–138.*

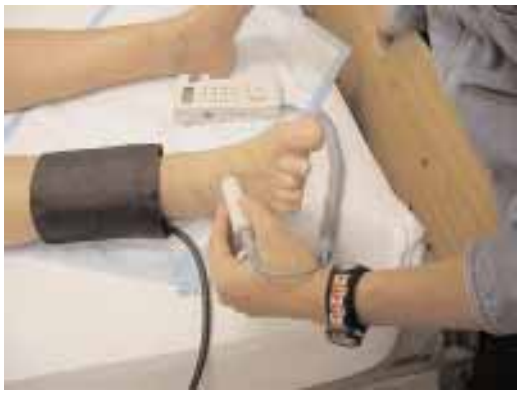

Figure 3
This photo demonstrates how to obtain an ankle brachial pressure index.

mathematically relates bandage tension and the number of layers to inverse of the radius of the leg and bandage width.

Thus, if the bandage tension is constant as one winds the bandage up the leg, a compression gradient will naturally develop because the smallest limb radius is at the ankle area just proximal to the ankle joint. Progressively larger radii are encountered up the leg, resulting in lesser degrees of compression given a constant bandage tension. This gradient of pressure provides support against venous hypertension that is greatest at the ankles when the patient is standing. A compression system must be capable of exerting at least 30 mm Hg of pressure at the level of the ankle to reliably prevent fluid exudation.[12–15] The Fletcher, Cullum, and Sheldon[16] systematic review of compression treatment for venous ulcers states:

1. Compression treatment increases the ulcer healing as compared to no compression.
2. High compression is more effective than low compression that should only be used in the absence of significant arterial disease.
3. No clear differences in effect from different types of compression system (multi-layer and short-stretch bandages, Unna's boot) have been shown. Since the publication of this article, however, the updated Cochrane Library website (www.update-software.com/cochrane/cochrane-frame.html) indicates that there may be an advantage to using elastic systems.
4. Intermittent and pneumatic compression appears to be a useful adjunct to bandaging.
5. Rather than advocate one particular system, the increased use of any correctly applied high compression treatment should be promoted.

Compression systems may be classified into three groups: short-stretch bandages (SSB), long-stretch bandages (LSB), and stockings. If the limb affected by the ulcer is acutely edematous, most experts believe that using a SSB system is preferred.[14,15] The SSB provides little or no elasticity. The contracting calf muscle exerts a high pressure against the fixed resistance of the SSB. This working pressure drives blood in the deep veins upwards. For the same reason, when the calf muscle relaxes, the bandage does not continue to exert pressure. This low "resting pressure" facilitates deep venous filling.[14,15] Short-stretch bandage systems require patients to be ambulatory. Without a calf muscle capable of contracting, the nonelastic bandage becomes less effective, but edema may fill a fixed volume and pressure exerted can prevent further fluid exudation. Patients who tend to shuffle around need to be trained to walk properly, making sure they push off with their toes. Similarly, those patients with ankle joints stiffened by arthritis or old injuries may not be good candidates for SSB systems.

Elastic or LSBs are more commonly used than SSB systems in North America and the United Kingdom. The "four-layer bandage" is popular because of its ability to

TABLE 4
VASCULAR SCREENING IN ASSESSMENT OF ISCHEMIC RISK

Ankle Brachial Pressure Index (ABPI)	Toe Pressure (mm Hg)	Ankle Doppler Waveform	Transcutaneous Oxygen Saturation	Risk
> 0.8	> 55 mg Hg	Normal	> 40%	Low
> 0.6–0.8	> 40 mm Hg	Biphasic or monophasic	30-40%	Low
> 0.4-0.6	> 20 mm Hg	Biphasic or monophasic	20-30%	High
< 0.4	< 20 mm Hg	Monophasic	< 20%	Severe

**Based on personal experience (R.G. Sibbald)*
Adapated with permission from Sykes MT, Godsey JB. Vascular evaluation of the diabetic foot. Clin Podiatr Med Surg. *1998; 15(1):49–83.*

$$\text{Sub-bandage} = \frac{\text{N (number of bandage layers)} \times \text{T (bandage tension)} \times \text{Constant}}{\text{R (radius of the leg)} \times \text{B (bandage width)}}$$

Figure 4
The Law of Laplace, demonstrated above, mathematically relates bandage tension and the number of layers to inverse of the radius of the leg and bandage width.

maintain high compression over several days up to a week. This reduces the frequency of dressing changes, a great advantage for home-care nursing. Because of elasticity, the four-layer bandage and other LSBs continue to exert compression even when the leg is elevated. This can be a problem if significant arterial insufficiency exists. As a result, the four-layer bandage is not recommended for use in patients with an ABPI of less than 0.8. All compression systems require trained personnel for application.

Other LSB systems are capable of lower levels of compression. These systems may be used with caution in the presence of moderate arterial insufficiency (ABPI > 0.5). Like other bandages, they may be left on for a week at a time. In the presence of severe arterial disease (ABPI < 0.5), lower compression systems are contraindicated.

Recommendation 5

Use Graduated Compression Stockings to Manage and Prevent Venous Leg Edema. Wearing Stockings to Decrease the Frequency of Ulcer Recurrence is Important

Once the venous ulcer has healed, the focus must shift toward prevention. Graduated compression stockings (GCS) are of proven value in managing venous hypertension.[17–19] Adherence to GCS has been shown to decrease the frequency of recurrent ulcers.[20,21]

Many experts in the field suggest compression levels of 20 mm Hg to 40 mm Hg for venous insufficiency. Evidence suggests that stockings need to extend higher than the knee in the majority of patients.

Patient compliance with the use of GCS is a major issue. Patients should be told that stockings must be applied first thing in the morning and removed in the evening. Several mechanical devices are available that facilitate the application of the garments even if a small ulcer is present. These can aid patients who suffer from arthritis of the hands or poor flexibility of major joints. Most stockings have a usable life of about 4 to 6 months. Adequate compression beyond this time cannot be guaranteed. The cost of the stockings should be justified to the patient as they offset the greater costs and decreased quality of life associated with managing recurrences.

Some experts on the CAWC panel use a nylon or cotton undersleeve to increase the compression of lighter dress support hose (15 mm Hg to 20 mm Hg is effectively increased to 25 mm Hg to 30 mm Hg). Alternatively, two dress support hose knee-highs can be put on top of each other to achieve higher compression or one pair can be mid-thigh length to decrease edema above the knee. Zippered stockings are helpful to some patients, while others may need a program of leg elevation and intermittent pneumatic compression if they cannot tolerate stockings. Patients must recognize that venous ulcer prevention means "compression for life."

Recommendation 6

Implement Intermittent Pneumatic Compression Therapy and/or Elevation of the Leg as an Added Benefit in Managing Venous Edema and Venous Leg Ulcers

More than occasionally, patients may require higher levels of compression than they can comfortably tolerate. Intolerable patient discomfort with the bandage system makes compliance an issue. The pneumatic compression (PC) device may be useful as an adjunct to compression bandaging whether used alone or as an alternative to compression bandaging or stockings in patients who are relatively immobile and, therefore, unable to activate the calf muscle pump. The elevation of the affected leg above the level of the heart also will help reduce edema.

A PC device consists of an inflatable sleeve that is placed around the limb and inflated to a preset pressure (30 mm Hg to 60 mm Hg). One type of PC, intermittent pneumatic compression (IPC), involves one chamber that is inflated intermittently; the other type, sequential pneumatic compression (SPC), involves a series of chambers that are inflated sequentially in a distal to proximal direction. This may be used to quickly reduce the volume of the leg prior to applying the compression bandages or graduated compression stockings. It is a useful alternative to compression bandages in patients who lack good mobility and cannot walk around to activate the calf muscle pump. It is also useful in managing lymphedema.

TABLE 5
AN APPROACH TO CLASSIFICATION AND TREATMENT OF ANKLE JOINT MOBILITY

1. If ankle joint mobility is present, and an SSB system is being used, the patient should be encouraged to raise both heels off the ground while in a standing position. The shuffling gait that is so common among the elderly should be discouraged.
2. If joint mobility is reduced but potential for improvement is evident, a physical therapist may be able to loosen soft-tissue contractures through the use of physiotherapy.
3. In patients with reduced or no ankle joint mobility, physical therapy and intermittent pneumatic compression should be considered.

One randomized, controlled study compared healing rates for 24 patients using moist occlusive dressings and graduated compression stockings (30 mm Hg to 40 mm Hg) with 21 patients using the same treatment plus sequential PC for a total of 4 hours per day. The treatment period lasted 3 months. Only one patient in the control group completely healed compared to 10 of the 21 in the PC group (P = 0.009, Fischer's exact probability test).[22] Pneumatic compression may be a useful adjunct that complements compression bandaging or stocking therapy for difficult-to-control edema.[23] Randomized controlled trials confirm the efficacy of both SPC or IPC therapy as an adjunct in managing venous leg ulcers. Pneumatic compression units are expensive, although rentals are available. Intermittent pneumatic compression therapy is contraindicated in the presence of significant arterial insufficiency, edema due to congestive heart failure, active phlebitis, deep vein thrombosis, or the presence of localized wound infection or cellulitis.

Recommendation 7

Consult with Rehabilitation Experts to Maximize Activity and Mobility. Consider Appropriate Adjunctive Therapies.

Activity and mobility. Altered or inefficient gait patterns, resulting in a walking disability, occur more frequently with advancing age. Alexander states that 8% to 19% of noninstitutionalized older adults and 63% of institutionalized older adults have difficulty walking or require assistance in order to ambulate.[24]

The ankle joint is equivalent to the hinge component of the calf muscle pump, and therefore, ankle dorsiflexion and plantar flexion are essential for the muscle pump to function efficiently. Limited ankle range of motion is known to exacerbate venous congestion and edema formation in patients with chronic venous insufficiency. Individuals lacking ankle mobility tend to externally rotate the hip and shuffle around, barely lifting their feet off the floor. Furthermore, the ability of compression bandages, such as the SSB system, to enhance venous return is compromised when ankle flexion is restricted. Chronic venous insufficiency itself may contribute to ankle immobility through the deposition of fibrotic tissue.

If ankle joint mobility is normal, and an SSB system is being used, the patient should be encouraged to contract the calf muscle while in a standing position, causing both heels to raise off the ground. Rehabilitation should include gait re-education that promotes proper heel-to-toe gait pattern and discourages the shuffling gait that is so common in this elderly patient population. If joint mobility is reduced due to soft tissue contractures, a physical therapist may perform manual and thermal joint mobilization techniques to optimize the range of motion of ankle dorsiflexion and plantar flexion (see Table 5).

Walking involves passive and active ankle joint motion as the weight of the body generates the force that drives the ankle joint pump. The power of the moving ankle joint generates the pumping force by virtue of the anatomic relations of the leg and ankle.[25] The power of the moving ankle joint and the competency of the veins work together and the calf pump moves venous blood back up to the heart.

Fiatarone et al[26] showed that exercise can increase muscle strength in frail men and women up to 96 years of age. The increase in lower extremity strength ranged from 61% to 374% over baseline. However, these gains were not maintained in the absence of continued training. Certain products (ie, Thera-Band, Hygenic Corporation, Akron, Ohio) promote dorsi and plantar flexion in patients with decreased mobility.

In his editorial in the *Journal of the American Geriatric Society*, Ettinger[27] states "a walk a day keeps the doctor away." He says it is the task of every physician and healthcare professional to encourage older patients to be more physically active to help lower rates of adverse health outcome.

Adjunctive therapies. The adjunctive modalities – ultrasound, pulsed electromagnetic fields, and electrical stimulation – may be beneficial in treating chronic venous ulcers that have failed to close despite good conventional wound care and compression therapy. The healing effects of therapeutic ultrasound on chronic leg ulcers has been the topic of at least seven properly designed clinical studies that exist in the research literature.[28] Three of these reports represent prospective, controlled, clinical trials that have examined the effectiveness of ultrasound specifically on chronic venous ulcers.[29–31] Although these clinical studies have produced both positive and negative findings, a recent meta-analysis that compiled the results of six well-designed clinical trials reported a significant beneficial effect of ultrasound on chronic leg ulcers.[32] This clinical research evidence is supplemented with extensive research performed in both animal and cellular experiments demonstrating that ultrasound stimulates release of inflammatory cell mediators, which, in turn, activate many important reparative cell processes such as fibroplasia and angiogenesis. For a summary of cellular and molecular mechanisms of ultrasound, consult a recent review.[33]

At least two clinical reports demonstrated accelerated healing rates of venous ulcers treated with pulsed electromagnetic fields compared to either standard wound care or placebo treatments.[34,35] Significant improvements were evident in local blood flow, skin temperature, and subcutaneous tissue oxygenation, and even in human subject tissue edema.[36,37]

Stimulating the wound bed using low level electrical currents has been

shown in numerous clinical reports to accelerate closure of several different types of chronic wounds.[28] Although the benefits of this modality on chronic pressure ulcers have clearly been established, few reports have specifically examined the effects of this modality on chronic leg ulcers. A prospective, placebo-controlled clinical trial that was recently completed by Houghton et al reported the benefits of electrical stimulation of chronic venous ulcers.[38]

Recommendation 8

Assess for Infection and Treat If Indicated

Bacteria potentially can become successful competitors for the natural resources needed by the wound healing machinery. If a sufficiently large population of a pathogenic species of bacteria is multiplying in the living tissue of the ulcer, healing will be severely impaired.[39] Bacterial quantitation may not tell the whole story. Bacterial virulence varies depending on the bacterial species in the wound. If host resistance is deficient, bacteria will thrive and markedly impair the healing process. Host resistance consists of systemic and local factors. Systemic factors include immune response and wound vascularity. Many systemic conditions such as diabetes and malnutrition also can contribute to reduced immune responses. Some examples of local factors that may promote bacterial growth include necrotic debris and foreign bodies. The combination of these factors determines the risk of significant bacterial influence on healing. If bacterial influence is considered sufficient to abolish good healing, antimicrobial intervention is necessary.

The decision to use antibiotics becomes important in several clinical situations. The patient who develops periulcer erythema, swelling, cellulitis, purulence, tenderness and pain, and sometimes fever and malaise, will benefit from systemic antibiotic therapy. In cases where infection results in septicemia, intravenous therapy is necessary. In these situations, taking a culture swab of the wound base after cleansing, as well as debriding, is justified. The main reason to obtain information is to make the antibiotic decision-making process more accurate. The patient likely will be put on antibiotics before results are available, and changes can be made later if sensitivity results are not favorable and the wound is not responding. Care must be taken in interpreting culture results, especially if multiple organisms are identified. Deciding which of the bacteria is the pathogen can be very difficult.[39]

Infection is only one cause of periulcer inflammation. Cellulitis and unusual infections must be differentiated from other causes. Leg ulcers may result from vasculitis, pyoderma gangrenosum, and malignancies (basal cell carcinoma, squamous cell carcinoma, and the like). Venous dermatitis occurs much more frequently than wound infection. Allergic contact dermatitis also must be considered. This type of eczema presents with erythema, scaling, erosions, and excoriations. Deep swelling characteristic of cellulitis is not seen. Another noninfective cause of periulcer eczema is the irritant contact dermatitis that occurs under the wound dressing if excessive moisture drains from the ulcer. Wound fluid contains many proteolytic enzymes that can be very irritating to the surrounding skin.

Using topical antibiotics is common in clinical practice, but their clinical use needs to be clarified by well-controlled studies.[39] The choice of systemic antibacterials is most often empirical and occurs prior to bacterial species identification. Chronic ulcers of less than 1-month duration usually are colonized by Gram-positive organisms (*Staphylococcus aureus*, and group A streptococcus). Cephalexin is an ideal choice of antibiotic, although cloxacillin is often used as well but does not adequately cover streptococcus species. Gram-negative and anerobic organisms may co-exist in ulcers of longer duration, and a broad spectrum of antibacterial coverage should be considered.

Distinguishing the rapidly healing wound from the clinically infected one often is not difficult. Between these two extremes lies a gray area where the wound stops healing because of significant bacterial numbers, yet obvious signs of infection may not have developed (critical colonization, increased bacterial burden). In these cases, despite optimal management of venous insufficiency, using appropriate dressings, and appropriate use of wound debridement, the wound will show no improvement for several weeks. Wound deterioration may be noted as healthy-looking granulation tissue turns into dusky dark red, friable (bleeds easily) tissue and is often hypertrophic or is replaced by yellow slough or necrotic eschar. The inflammatory response associated with increased bacteria in the wound may cause an increase in clear exudate prior to frank purulence and odor. An empirical trial of topical or oral antibacterials may be indicated. With the emergence of resistant bacteria, topical antimicrobials that have multiple targets of action in the bacterial cell have become popular. Two topical agents

TABLE 6
CLASSIFICATION OF WOUND DRESSINGS AND BIOLOGIC AGENTS

Dressing Type	Main Uses	Absorption	Contraindications
Plastic films	• Epithelialization of wounds • Support autolysis • Protection of new epithelial tissue	Δ	• Draining wounds • Infected wounds • Poorly granulated wounds
Hydrogels **amorphous** **wafer**	• Hydration of dry wounds • Support autolysis • Donor sites (grafts) • Epithelialization	+	• Excessively draining wounds • Infected wounds (wafer)
Hydrocolloids	• Granulation tissue formation • Support autolysis	+	• Infected wounds • Excessively draining wounds
Calcium alginates	• Absorption of exudate • Hemostasis • Infected wounds	++	• Superficial wounds • Epithelializing wounds
Hydrofibers	• Absorption of exudate into the fibers (dynamic) • Infected wounds	+++	• Epithelializing wounds
Foams **adhesive** **nonadhesive**	• Excessively draining wounds • Nondynamic absorption	+++	• Infected wounds (adhesive)
Biological agents	• Difficult cases • Dormant wounds	Δ	• Infected wounds • Necrotic wounds

Δ = 0
+ = small amount
++ = moderate amount
+++ = large amount

that have recently received attention are cadexomer iodine and nanocrystalline silver dressings.[40,41] These agents may support autolytic debridement, decrease surface bacteria, and help achieve moisture balance without excessive toxicity to the cells in the wound base.

Recommendation 9

Optimize the Local Wound Healing Environment: Debridement, Bacterial Balance, and Moisture Balance. Use Biological Agents when the Cause Has Been Corrected and Healing Does Not Proceed at an Expected Rate

Moist wound healing is accepted as the best practice by most advanced wound care practitioners. It is contraindicated in nonhealable wounds with inadequate vasculature. The three major components of local wound care are debridement, moisture balance, and bacterial balance.[42] Debridement of slough (soft, devitalized tissue) and necrotic, hard eschar are primarily surgical and autolytic (hydrocolloids or hydrogels). Moisture balance is achieved largely through the use of moist interactive dressings. A review of wound dressings is covered in excellent current reviews and is summarized in Table 6.[42,43]

When treating the edematous leg, the patient should be warned that the ulcer will initially produce a great deal of drainage. During this early stage of ulcer management, the absorbent dressing may have to be changed frequently to avoid developing contact irritant dermatitis of the surrounding skin. Appropriate dressing types for this situation include absorbent foam dressings, hydrofibers, and calcium alginates. As the edema subsides, absorbent dressings can be left on for up to 1 week with appropriate compression therapy.

Once the granulation tissue has filled in the defect, facilitating re-epithelialization becomes important. At this time, dressing changes should be kept to a minimum. Keratinocytes begin to migrate across the wound and do not anchor to the wound bed until it is covered.

Too frequent dressing changes tend to tear off the neoepithelium before it gets a chance to establish itself. Plastic film dressings (adherent, nonadherent, fenestrated) may be a good option for this stage of healing.

A recent advance in managing venous leg ulcers has been the development of biological skin equivalents. In 1997, a cultured, allogeneic, bilayered human skin equivalent, Apligraf™ (Organogenesis, Canton, Mass.; Novartis Pharmaceuticals Canada Inc., Dorval, Quebec, Canada), was first released in Canada. This skin equivalent has made treating some nonhealing venous ulcers and other difficult-to-treat ulcers possible. By 1999, considerable Canadian experience had accumulated, resulting in a consensus paper.[44] The pivotal study that confirmed the efficacy of Apligraf™ was a randomized, multicentered, prospective study involving 275 patients with venous leg ulcers.[45] Falanga et al concluded that treatment with human skin equivalent (HSE), combined with compression therapy, was more effective than compression therapy alone (63% healed at 6 months versus 49% in the control group). Dermagraft (Advanced Tissue Science, La Jolla, Calif., and Smith and Nephew, Largo, Fla.), living fibroblasts on a vicryl mesh, also has been successful in healing venous leg ulcers refractory to optimum four-layer compression therapy alone.[46]

Recommendation 10

Implement Medical Therapy If Indicated for CVI (Superficial and Deep Thrombosis, Woody Fibrosis)

Deep venous thrombosis commonly presents with an acute swollen painful leg. Individuals with this condition have excruciating pain on deep palpation or walking. Homans sign (exquisite pain with ankle flexion of the outstretched leg) is often misleading as a diagnostic test. In the past, clinicians had to rely on invasive venograms; however, duplex Doppler studies in skilled hands are just as accurate in making this diagnosis. If DVT occurs in a young person or is recurrent, investigations should be carried out to look for Factor 5 (Leiden factor), protein C, or protein S deficiencies. Individuals also may have a family history of recurrent DVTs. The treatment is anticoagulation, often with heparin (or low-molecular weight heparin), followed by oral anticoagulation. Recent evidence indicates that anticoagulation may need to be continued for initial episodes lasting longer than 6 months and indefinitely for recurrence. Other risk factors for DVT include smoking and using birth control pills.

Superficial phlebitis often is localized, possibly with a discrete visible red streak with associated pain on palpation over the involved vein. Superficial phlebitis frequently is aggravated by surrounding edema. Compression therapy may not be tolerated due to increased local pain. Treatment includes the use of nonsteroidal anti-inflammatories or ASA. This treatment should be tapered as the patient improves, because these agents may impair wound healing. When compression therapy is used on painful legs, short-stretch compression (low pressure at rest) is often better tolerated.

Long-standing venous disease can be detected in patients by noting the development of woody fibrosis around the ankle region. Clinically, loss of tissue flexibility is evident, and the surface is hard and nonmoveable on palpation. These fibrin deposits may be improved with long-term pentoxifylline. Many of the studies have shown a benefit over compression therapy alone when used for periods of 3 to 6 months,[47] but these studies have been criticized for their lack of consistency of the compression and for using less than the optimal current gold standard high compression therapy. Falanga et al[47] documented that 800 mg tid rather than the usual 400 mg tid of pentoxyphylline, when combined with the Duke boot, provided statistically significant healing over similar compression combined with oral placebo or low-dose therapy. However, such doses are likely to be associated with significant gastrointestinal upset. Pentoxifylline is a xanthine derivative with many of the same side effects as caffeine. Normally, it is not given with anticoagulation therapy and must be used with caution in the very elderly as it may potentiate the action of antihypertensive agents.

Recommendation 11

Consider Surgical Management if Significant Superficial or Perforator Vein Disease Exists in the Absence of Extensive Deep Disease

In some venous ulcer patients, after systemic factors have been managed, the wound bed optimized, and compression therapy instituted, the wound stubbornly refuses to heal. In this situation, the venous system should be examined systematically to consider surgical procedures. Venous incompetence in the deep system is unlikely to be corrected surgically. Perforator incompetence and disease of the superficial venous system can be managed using new surgical techniques that are associated with

only mild morbidity. Significant perforator incompetence may cause severe localized venous hypertension, and the local edema may not be adequately managed by the use of a compression bandage system.

Specially trained vascular surgeons may be able to ligate incompetent perforator veins using fiber-optic instruments to perform the subfascial, endoscopic perforator surgical procedure.[46] In the past, Linton's procedure,[46] the definitive venous surgical procedure, involved opening the entire leg, exposing incompetent perforators. This was major surgery with marked morbidity and prolonged convalescence.

Historically, venous obstruction was thought to be the major etiological factor in CVI. More recently, only a small minority of patients was found to have isolated venous clot or obstruction. In 90% to 95% of venous ulcer patients, venous insufficiency is caused by valvular incompetence and not by blockage of the deep veins.[48] Recently, duplex ultrasound scanning has improved the measurement of reflux in deep, superficial, and perforators.[49] Surgical management of superficial disease may be of benefit in healing ulcers and for prevention. More research needs to be done to determine if any circumstances exist in which the surgery of the superficial system might benefit the deeper venous circulation. What still must be determined is the long-term sequelae of the new venous procedures and the frequency of ulcer recurrence 5 to 25 years later. At the present time, venous surgery should be reserved for patients who are compliant and do not respond to more conservative treatment programs.

Recommendation 12

Communicate with the Patient, the Family, and the Caregivers to Establish Realistic Expectations for (Non)Healing. The Presence or Absence of a Social Support System is Important for Treatment and Prevention of Venous Leg Ulcers

Phillips et al[50] calculated that 68% of patients with venous ulceration reported unpleasant emotions such as fear, depression, social isolation, anger, anxiety, and a negative self-image. Physical attributes such as the ability to walk, accomplish everyday tasks, and maintain mobility were of utmost concern. Chase et al[51] explored four themes that emerged during their 1-year study on patients' experiences with venous leg ulcers. The first theme was a "forever healing process" that discussed the patients' reports of the lengthy healing process, ulcer recurrences, and chronic pain. The second theme was the limitations that resulted in a loss of mobility, inconveniences of therapy, the change in body image, and the threat of amputation. Feeling powerless and resigned to the uncertainty of healing was the third theme. Unfortunately, the fourth theme was "who cares?" Patients displayed this attitude when they did not have active involvement or ownership of the venous ulcer treatment. These findings are very similar to Krasner's report on chronic pain and decreased quality of life in 14 patients with venous leg ulcers.[52]

As Price and Harding conclude, "The impact of living with the wound and its concomitant treatment can be seen in different ways by different people, but only the patient genuinely understands the significance of that experience."[53] Hayes[54] states there are two issues to be considered and to be addressed: "the consequences of doing nothing or the consequences of intervention." Education concerning the disease process and adherence to treatment are pivotal to the prevention and treatment of venous leg ulcers. The patient needs to know why compression is part of the treatment. A person with venous ulcers is facing a lifestyle change that requires mutual goal setting with the patient taking charge and maintaining independence. Using an interdisciplinary approach and carefully examining the patient's socioeconomic situation, a support system can be constructed that is vital to the success of a treatment program. The patient should be given the opportunity to verbalize or express his or her concerns and frustrations about how the venous ulceration has affected his or her life. Increased understanding between the patient and healthcare team is likely to increase adherence to treatment protocols and improve patient care outcomes.

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