Chronic Venous Insufficiency

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Chronic venous disease (CVD) is often overlooked by healthcare providers because of an underappreciation of the magnitude and impact of the problem, as well as incomplete recognition of the various presenting manifestations of primary and secondary venous disorders. The importance of CVD is related to the number of persons afflicted and the socioeconomic impact of its more severe manifestations. CVD is a very common problem, with varicose veins affecting more than 25 million adults in the United States and more than 6 million with more advanced venous disease.[1] Because of this high prevalence of venous disease, the National Venous Screening Program was conducted by the American Venous Forum in the United States to increase awareness. The program identified varicose veins in >30% of participants and more advanced venous disease in >10%.[2] The most common manifestations of CVD are telangiectases, reticular veins, and varicose veins. Chronic venous insufficiency (CVI) describes a condition that affects the venous system of the lower extremities, with the *sine qua non* being persistent ambulatory venous hypertension causing various pathologies, including pain, edema, skin changes, and ulcerations. CVI often indicates the more advanced forms of venous disorders, including manifestations such as hyperpigmentation, venous eczema, lipodermatosclerosis, atrophie blanche, and healed or active ulcers. However, because varicose veins also have incompetent valves and increased venous pressure, we use the term "CVI" to represent the full spectrum of manifestations of CVD.[3]

Varicose veins have an estimated prevalence between 5% and 30% in the adult population, with a female:male predominance of 3:1, although a more recent study supports a higher male prevalence.[4] The Edinburgh Vein Study screened 1566 subjects with duplex ultrasound for reflux, finding CVI in 9.4% of men and 6.6% of women after age adjustment, which rose significantly with age (21% in men >50 years, and 12% in women >50 years).[5] The San Valentino Vascular Screening Project found among the 30 000 subjects evaluated by clinical assessment and duplex ultrasound a prevalence of 7% for varicose veins but <1% for symptomatic CVI.[6] CVI was more common with increasing age; however, there was no significant sex difference. The rate of varicose vein development may be estimated from the Framingham Study, which found an annual incidence of 2.6% in women and 1.9% in men.[7] The Vein Consult Program evaluated more than 91 000 subjects in various geographic regions and found a worldwide prevalence of clinically significant CVD of ≈60%.[8] The prevalence of varicose veins is higher in developed, industrial countries than in underdeveloped countries. Risk factors found to be associated with CVI include age, sex, a family history of varicose veins, obesity, pregnancy, phlebitis, and previous leg injury.[9,10] There are also environmental or behavioral factors associated with CVI, such as prolonged standing and perhaps a sitting posture at work.[10,11]

The more serious consequences of CVI, such as venous ulcers, have an estimated prevalence of ≈0.3%, although active or healed ulcers are seen in ≈1.0% of the adult population.[12] It has been estimated that approximately 2.5 million people experience CVI in the United States, and of those ≈20% develop venous ulcers.[13] The overall prognosis of venous ulcers is poor, because delayed healing and recurrent ulceration are very common.[14] The socioeconomic impact of venous ulceration is dramatic because of an impaired ability to engage in social and occupational activities, reducing the quality of life and imposing financial constraints. Disability related to venous ulcers leads to loss of productive work hours, estimated at 2 million workdays per year, and may cause early retirement, found in >12% of workers with venous ulcers.[15] The financial burden of venous ulcer disease on the healthcare system is readily apparent, with an estimated $1 billion spent annually on treatment of chronic wounds in the United States or ≤2% of the total healthcare budget in Western countries, and estimates placing the cost of venous ulcer care at $3 billion annually.[16,17]

Given the prevalence and socioeconomic impact of CVD, an understanding of the clinical manifestations, diagnostic modalities, and therapeutic options is warranted. This article reviews clinical aspects of CVI, with a focus on the diagnostic and therapeutic options, and places these in context of the Clinical Practice Guidelines (CPG) of the Society for Vascular Surgery and American Venous Forum of 2011, which used best evidence-based practice, and applied a grading system.[18]

Venous Pathophysiology

Normal Venous Anatomy and Function

To appreciate the pathophysiology of CVI, it is first useful to understand normal venous anatomy and function. The peripheral venous system functions as a reservoir to store blood and as a conduit to return blood to the heart. Proper functioning of the peripheral venous system depends on patency of vessels containing a series of 1-way valves and muscle pumps. In the erect position, blood that enters into the lower extremity venous system must travel against gravity and other pressures to return to the central circulation.

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The veins of the lower extremity are divided into superficial, deep, and perforator veins.[19,20] The superficial venous system is located above the muscular fascial layer. It is composed of an interconnecting network of veins and several truncal superficial veins, including the great saphenous vein (GSV) and small saphenous vein, as well as several accessory veins, which may develop pathology contributing to CVI. The deep venous system is located below the muscular fascia and serves as collecting veins and the outflow from the extremity. The deep veins of the lower extremity consist of axial veins, which follow the course of the major arteries, and intramuscular veins, including venous sinusoids and plexi. The perforating veins traverse the anatomic fascial layer to connect the superficial to the deep venous system.

The valves within the veins are essential in assuring that blood flows in the correct direction, particularly while in the upright posture.[19] There is a series of 1-way bicuspid valves located throughout the deep and superficial veins that open to allow flow toward the heart but close to prevent the return of blood toward the feet. There are 4 phases of valve function which include opening, equilibrium, closing, and closed. The critical factors to valve function are axial vortical of blood flow opening the valve and vertical velocity in the valve cups that increases mural pressure relative to the luminal pressure leading to valve closure.[21] The frequency of these venous valves increases from the proximal to distal leg to prevent an increase pressure within the distal veins because of gravitational effects. In addition, perforating veins also contain valves that only allow blood flow from the superficial to the deep veins.

The valves function in concert with venous muscle pumps to allow the return of blood against gravity to the heart.[22] Contraction of the muscle pumps, primarily in the calf, force blood out of the venous plexi to ascend up the deep venous system. The valves prevent blood from being forced more distally within the deep system or through perforator veins into the superficial system. Immediately after ambulation, the pressure within the veins of the lower extremity is normally low because the venous system has been emptied by the muscle pump action (Figure 1). Relaxation of the muscle pump then allows blood to refill to the deep venous system. With prolonged standing, the veins become distended as the vein fills via antegrade flow, allowing the valves to open and pressure to increase. Contraction of the muscle pump will again empty the veins and reduce venous pressure.

Venous Pathophysiology and Dysfunction

Venous pathology develops when venous pressure is increased and return of blood is impaired through several mechanisms.[23] This may result from valvular incompetence of axial deep or superficial veins, perforator veins, venous tributaries, or venous obstruction, or a combination of these mechanisms. These factors are exacerbated by muscle pump dysfunction, most notably of the calf muscles. These mechanisms serve to produce global or regional venous hypertension, particularly with standing or ambulation. Contributing to the macrocirculatory hemodynamic disturbances are alterations within the microcirculation.[24,25] Unabated venous hypertension may result in dermal changes with hyperpigmentation; subcutaneous tissue fibrosis, termed "lipodermatosclerosis"; and eventual ulceration.

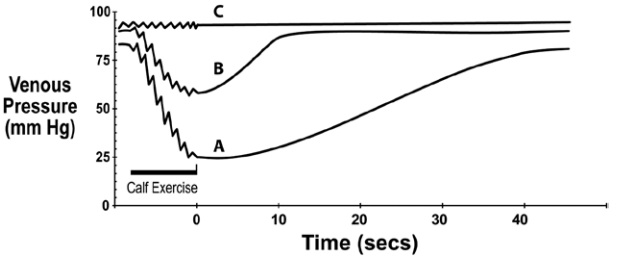

Figure 1. Illustrative ambulatory foot venous pressure measurements during exercise and at rest over time in the standing position. **A**, Normal venous pressure. The resting standing venous pressure in ≈80 to 90 mm Hg. The pressure drops to ≈20 to 30 mm Hg (or >50% decrease) with calf exercise. The return in pressure is gradual, with refill taking >20 seconds. **B**, Abnormal venous pressure with venous reflux. The resting standing pressure is usually higher than normal. The drop in pressure with exercise is blunted (<50% decrease). The return in venous pressure to the resting level is rapid because of a short refill time (<20 seconds). **C**, Abnormal venous pressure with venous obstruction. Resting standing venous pressure is usually higher than normal. There is minimal-to-no drop in pressure with exercise.

Dysfunction or incompetence of the valves in the superficial venous system also allows retrograde flow of blood, which is called "reflux" and serves to increase hydrostatic pressures. Valve failure of the superficial veins may be primarily because of pre-existing weakness in the vessel wall or valve leaflets or secondary to direct injury, superficial phlebitis, or excessive venous distention resulting from hormonal effects or high pressure.[23] Failure of valves located at the junctions of the deep and superficial systems, at the saphenofemoral and saphenopopliteal junctions, can be a source of reflux leading to CVI. Incompetence of the valves of the superficial veins with reflux has been shown in ≤90% of patient presenting the CVD with reflux in the GSV, accounting for 70% to 80%, and in ≈84% of those presenting with venous ulcers.[26,27]

Dysfunction of the valves of the deep system is most often a consequence of damage from previous deep vein thrombosis. The damage to the valves of the deep veins leads to rapid refilling by pathologic retrograde venous flow and may even reduce the blood volume exiting the limb. The venous pressure immediately after ambulation may be slightly elevated or even normal, but the veins refill quickly with the development of high venous pressure without ongoing muscle contraction (Figure 1).[28] Dysfunction of the deep vein valves has been shown to increase the rate of progression of venous disease with a higher rate of venous ulceration formation.[29,30]

Failure of the valves in the communicating perforator veins may also allow high pressure to enter into the superficial system.[31,32] Perforator valve incompetence allows blood to flow from deep veins backward into the superficial system with force because of the high pressures generated by the muscle pump action. This excessive local pressure can produce dilatation of the superficial veins and their valve cusps with secondary failure of the valves. This may result in a localized cluster of dilated veins that ascends up the leg.[33]

In addition, reflux may also occur in venous tributaries in the absence of any truncal superficial or deep vein or perforator vein reflux.[34] The most common tributaries with reflux are in communication with the GSV (≈60%), small saphenous vein (≈20%), or both (≈10%). This process of isolated

tributary reflux may contribute to progression of disease within the other superficial or deep venous segments.

Obstruction of the deep veins may limit the outflow of blood, causing increased venous pressure with muscle contraction, and lead to secondary muscle pump dysfunction. Venous obstruction may result from an intrinsic venous process, such as chronic deep vein thrombosis or venous stenosis, or because of extrinsic compression. Venous outflow obstruction plays a significant role in the pathogenesis of CVI.[35] Postthrombotic venous obstruction, as with deep vein valve dysfunction, has a high rate of venous ulceration development and more rapid progression of disease.[29]

Dysfunction of the muscle pumps may lead to ineffective emptying of venous blood from the distal lower extremity. This rarely occurs as a primary disorder with neuromuscular conditions or muscle-wasting syndromes. However, muscle pump dysfunction often occurs with severe reflux or obstruction. Because of ineffective venous emptying, the immediate postambulatory venous pressure will be nearly as high as the pressure after prolonged standing. Muscle pump dysfunction appears to be a significant mechanism for the development of complications such as venous ulcers.[36,37]

Changes in the hemodynamics of the large veins of the lower extremity are transmitted into the microcirculation and lead to the development of venous microangiopathy.[24] In addition, dysfunction of the microvenous valves seems to be play a key role and may occur independent of the macrovenous dysfunction.[38] These hemodynamic perturbations contribute to the development of microangiopathic findings, with elongation, dilation, and tortuosity of capillary beds; thickening of basement membranes with increased collagen and elastic fibers; endothelial damage with widening of interendothelial spaces; and increased pericapillary edema with halo formation; however, normal interendothelial junctions have also been found in advance CVI.[39] The abnormal capillaries with increased permeability and high venous pressure lead to the accumulation of fluid, macromolecules, and extravasated red blood cells into the interstitial space. There have been several postulated mechanisms for the development of venous microangiopathy, including fibrin cuff formation, growth factor trapping, and white blood cell trapping, but further work in this area is needed to better define this pathophysiology.[24]

Clinical Manifestations

CVD represents a spectrum of conditions ranging from simple telangiectases or reticular veins to more advanced stages, such as skin fibrosis and venous ulceration. It is important to realize that the same clinical manifestations may result from different pathogenic mechanisms, including incompetent valves, venous obstruction, muscle pump dysfunction, or a combination. The major clinical features of CVI are dilated veins, edema, leg pain, and cutaneous changes in the leg. Varicose veins are dilated superficial veins that become progressively more tortuous and enlarged (Figure 2A). They may develop bouts of superficial thrombophlebitis, recognized by painful, indurated, inflamed areas along the varicose vein. Edema begins in the perimalleolar region and ascends up the leg with dependent fluid accumulation. Leg discomfort is often described as heaviness or aching after prolonged standing and is relieved by elevation of the leg. This discomfort is thought to be produced by increased intracompartmental and subcutaneous volume and pressure. There may also be tenderness along varicose veins because of venous distention. Obstruction of the deep venous system may lead to venous claudication (or intense leg discomfort with ambulation). Cutaneous changes include skin hyperpigmentation because of hemosiderin deposition and eczematous dermatitis (Figure 2B). A fibrotic process may occur in the dermis and subcutaneous fat termed "lipodermatosclerosis." There is an increased risk of cellulitis, leg ulceration, and delayed wound healing (Figure 2C). In addition, protracted CVI may also contribute to the development of lymphedema, representing a combined process.

The manifestations of CVI may be viewed in terms of a well-established clinical classification scheme. The Clinical, Etiology, Anatomic, Pathophysiology (CEAP) classification was developed by an international consensus conference to provide a basis of uniformity in reporting, diagnosing, and treating CVI (Table 1).[40] The clinical classification has 7 categories (0–6) and is further categorized by the presence or absence of symptoms. The etiologic classification is basis on congenital, primary, and secondary causes of venous dysfunction. Congenital disorders are those that are present at birth but may be recognized later in life. These include the well-recognized syndromes of Klippel-Trenaunay (varicosities and venous malformations, capillary malformation, and limb hypertrophy) and Parkes-Weber (venous and lymphatic malformations, capillary malformations, and arteriovenous fistulas).[41] Primary venous insufficiency is of uncertain etiology, whereas secondary venous insufficiency is attributed to an acquired condition. The anatomic classification describes the superficial, deep, and perforating venous systems, with multiple venous segments that may be involved. The pathophysiologic classification describes the underlying mechanism resulting in CVI, including reflux, venous obstruction, or both. Validation of the CEAP classification system has often focused on the clinical classification.[42] The classification is a valuable tool in the objective evaluation of CVI, providing a system to standardize CVI classification with emphasis on the manifestations, cause, and distribution of the venous disease.[43] The use of the CEAP classification in the evaluation of CVD has a strong recommendation in the CPG (grade 1A).[18]

To complement the CEAP classification and further define the severity of CVI, a venous clinical severity score was developed.[44,45] The revised venous clinical severity score provides clarification of the terms and better definition of the descriptors and has further clinical applicability (Table 2).[46] The venous clinical severity score consists of 10 attributes with 4 grades (absent, mild, moderate, and severe). It has been shown to be useful in evaluation of the response to treatment in CVD and is recommended for routine clinical use in the CPG (grade 1B).[18,45] To better evaluate patient-perceived success, severity of disease, and treatment outcome in CVI, the use of validated disease-specific quality-of-life questionnaires is also encouraged by the CPG (grade 1B).[18]

Diagnosis of CVI

A complete history and physical examination are important to establish a proper diagnosis of CVI and may be assisted by noninvasive testing. Invasive testing may also be used to establish the diagnosis but is typically reserved for assessing disease

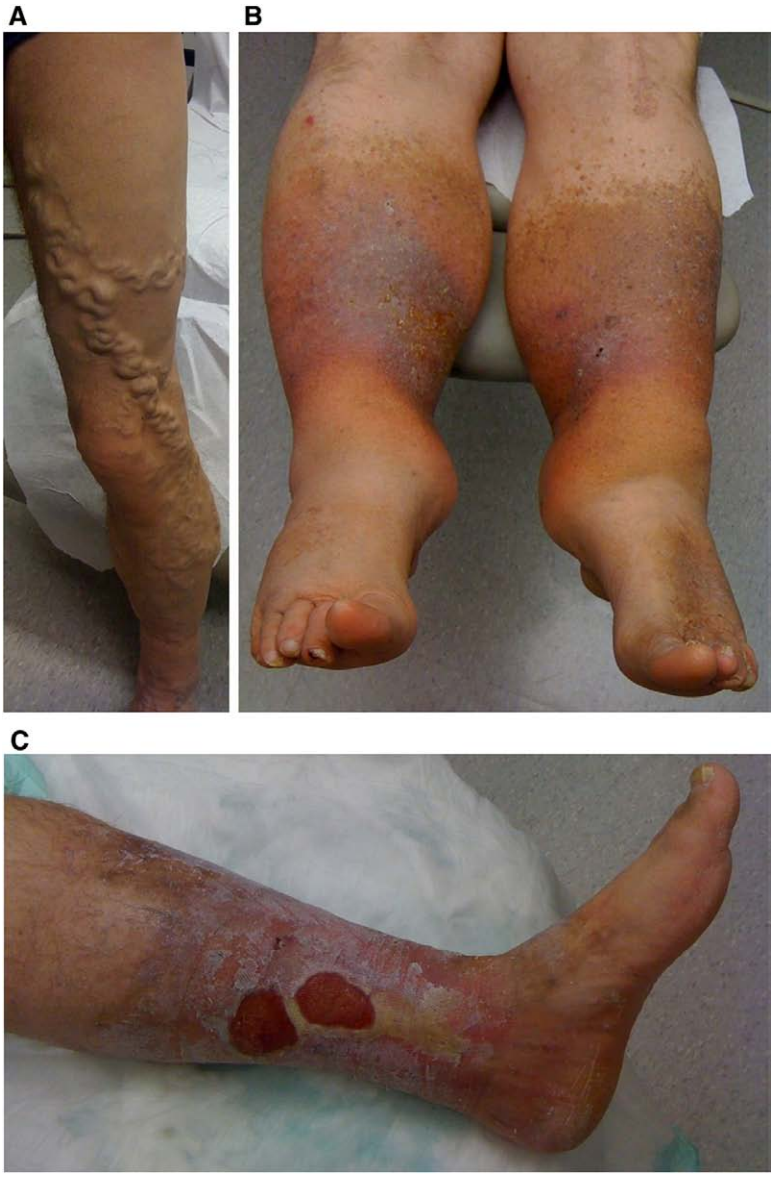

Figure 2. Manifestations of chronic venous insufficiency. **A**, Extensive varicose veins involving the thigh and leg. **B**, Hyperpigmentation and severe lipodermatosclerosis with leg edema. Notice healed ulcers in the gaiter region of the medial leg. **C**, Medial malleolar venous ulcers. Notice concomitant eczema and lipodermatosclerotic skin.

severity or if surgical intervention is being contemplated. The methods used to assess CVI are described below, but comprehensive overviews have been published previously.[47]

Physical Examination

The physical examination not only aids in establishing a diagnosis but plays an important role in helping to guide therapy in CVI. Visual inspection and palpation may reveal evidence of venous disorders. The skin is examined for prominent, dilated superficial venous abnormalities, such as telangiectasis, reticular veins, or varicose veins. The surface is inspected for irregularities or bulges to suggest the presence of dilated tortuous veins. The distribution of these varicose veins may follow the course of the affected superficial vein, such as the GSV and small saphenous vein. This evaluation should include positioning in the upright posture to allow for maximal distention of the veins. Additional skin findings may be seen, such as hyperpigmentation, stasis dermatitis, atrophie blanche (or white scarring with a paucity of capillaries), or lipodermatosclerosis. The presence of edema and its severity is assessed. The edema seen in CVI is dependent and usually pitting; however, it may become more resilient to palpation if protracted. There is often relative sparing of the forefoot to help distinguish the etiology of other causes of edema, such as lymphedema. An early finding of venous congestion includes calf fullness or increased limb girth, so the calf muscle consistency should be assessed, and measurement of the limb girth should be performed. There is no universally agreed on scale for grading the severity of edema. The venous clinical severity score graded edema on the basis of the level of the most proximal involvement in the limb (see Table 2). Other scales (typically range from 0 to 4+) use the presence of visual distortion of the limb, the depth of indentation, and the duration for rebound of the pitting. Palpation along the course of dilated veins may reveal tenderness. The presence of active or healed ulcers, typically in a distribution near the medial aspect of the ankle with GSV reflux or lateral aspects of the ankle with small saphenous vein reflux, may be seen with more advanced disease.[48]

A classic tourniquet (eg, the Brodie-Trendelenburg) test may be performed at bedside to help distinguish deep from

Table 1. CEAP Classification of Chronic Venous Disease

CEAP Classification
Clinical classification (C)*†
C_0 No visible sign of venous disease
C_1 Telangiectases or reticular veins
C_2 Varicose veins
C_3 Edema
C_4 Changes in skin and subcutaneous tissue‡
A Pigmentation or eczema
B Lipodermatosclerosis or atrophie blanche
C_5 Healed ulcer
C_6 Active ulcer
Etiologic classification (E)
E_c Congenital (eg, Klippel-Trenaunay syndrome)
E_p Primary
E_s Secondary (eg, postthrombotic syndrome, trauma)
E_n No venous cause identified
Anatomic classification (A)
A_s Superficial
A_d Deep
A_p Perforator
A_n No venous location identified
Pathophysiologic classification (P)
P_r Reflux
P_o Obstruction, thrombosis
$P_{r,o}$ Reflux and obstruction
P_n No venous pathophysiology identified

*Telangiectases are <1 mm, reticular veins are between 1 and 3 mm, and varicose veins are >3 mm measured in the upright position; however, in the Venous Clinical Severity Score, varicose veins are considered to be >4 mm. The Revised Venous Clinical Severity Score considers varicose veins to be ≥3 mm in the standing position.

†The descriptor A (asymptomatic) or S (symptomatic) is placed after the C clinical class.

‡C_4 is subdivided into A and B, with B indicating higher severity of disease and having a higher risk for ulcer development.

superficial reflux.[49] The test involves applying a tourniquet or manual compression over the superficial veins after the patient lies down to empty the veins. The veins are observed with resumption of an upright posture; in the presence of superficial reflux, the varicose veins will take >20 seconds to dilate; in contrast, in the presence of deep (or combined) venous reflux, the varicose veins will rapidly dilate. Similarly, the use of handheld continuous-wave Doppler may also be used to assist in the bedside evaluation.[50] Although these adjunctive bedside techniques are potentially useful to help determine the presence and location of CVI, this is typically performed with noninvasive testing using venous duplex imaging.

Differential Diagnosis

There is a broad differential for the most common presenting symptoms of limb swelling and discomfort that are seen in CVI. The initial task is to exclude an acute venous problem, such as deep vein thrombosis. Then, systemic causes of edema need to be considered, such as heart failure, nephrosis, liver disease, or endocrine disorders. Importantly, adverse effects of medication should be considered, such as those with calcium channel blockers, nonsteroidal anti-inflammatory agents, or oral hypoglycemic agents. Critical disorders to consider are lymphedema, lipedema, and the combined disorder of lipolymphedema. Lymphedema because of obstruction of lymphatic drainage leads to fluid accumulation that extends into the foot and toes, in contrast with CVI, which relatively spares the foot. The edema may be pitting early in the course of the disease but as the disease progresses becomes nonpitting. In contrast, lipedema is characterized by fatty tissue accumulation rather than fluid, thus, it is not pitting. It usually spares involvement of the feet, often with a cuff of tissue at the ankle. Finally, other regional considerations should be made, such as ruptured popliteal cyst, soft tissue hematoma or mass, exertional compartment syndrome, or gastrocnemius tear. The use of examination findings and noninvasive testing should allow for the proper diagnosis to be established.

Noninvasive Testing

Venous Duplex Imaging

Venous duplex imaging is currently the most common technique used to confirm the diagnosis of CVI and assess its etiology and anatomy and is highly recommended in the CPG (grade 1A).[18,51,52] Venous duplex imaging combines B-mode imaging of the deep and superficial veins with pulsed Doppler assessment of flow direction with provocative maneuvers. The presence of venous obstruction because of chronic deep vein thrombosis or venous stenosis may be directly visualized or inferred from alteration in spontaneous flow characteristics. The direction of flow may also be assessed in the reverse Trendelenburg position during a Valsalva maneuver or after augmenting flow with limb compression. However, the preferred method to assess for reflux involves the use of a cuff inflation-deflation technique with rapid cuff deflation in the standing position.[53] This provides information about the anatomic distribution of reflux disease involving the deep and superficial venous systems, as well as perforator veins.

The presence of reflux is determined by the direction of flow, because any significant flow toward the feet is suggestive of reflux (Figure 3). The duration of reflux is known as the reflux time. A reflux time of >0.5 seconds for superficial veins and 1.0 second for deep veins is typically used to diagnose the presence of reflux.[54] A longer duration of reflux implies more severe disease but does not correlate well with clinical manifestations.[55] Venous duplex imaging provides information about local valve function to construct an anatomic map of disease in terms of the systems and levels of involvement. This is often sufficient data to help guide therapy, but if the contribution of the reflux to global hemodynamics is required, then further testing, such as plethysmographic techniques, should be considered.

Air Plethysmography

Air plethysmography (APG) has the ability to measure each potential component of the pathophysiologic mechanisms of CVI, including reflux, obstruction, and muscle pump dysfunction.[56,57] Changes in limb volume are measured by air displacement in a cuff surrounding the calf during maneuvers to empty

Table 2. Venous Clinical Severity Score

	Venous Clinical Severity Score			
Attribute	Absent = 0	Mild = 1	Moderate = 2	Severe = 3
Pain	None	Occasional, not restricting daily activity	Daily, interfering but not preventing daily activity	Daily, limits most daily activity
Varicose veins	None	Few, isolated branch varices, or clusters, includes ankle flare	Confined to calf or thigh	Involves calf and thigh
Venous edema	None	Limited to foot and ankle	Extends above the ankle but below knee	Extends to knee and above
Skin pigmentation	None or focal	Limited to perimalleolar	Diffuse, over lower third of calf	Wider distribution above lower third of calf
Inflammation	None	Mild cellulitis, ulcer margin limited to perimalleolar	Diffuse over lower third of calf	Wider distribution above lower third of calf
Induration	None	Limited to perimalleolar	Diffuse over lower third of calf	Wider distribution above lower third of calf
Ulcer number	0	1	2	≥3
Ulcer duration	NA	<3 mo	>3 mo but <1 y	Not healed >1 y
Ulcer size	NA	Diameter <2 cm	Diameter 2–6 cm	Diameter >6 cm
Compressive therapy	Not used	Intermittent	Most days	Full compliance

NA indicates not applicable.

and fill the venous system (Figure 4). Venous outflow is assessed during rapid cuff deflation on an elevated limb that has a proximal venous occlusion cuff applied. The venous outflow at 1 second, expressed as a percentage of the total venous volume, is used to evaluate the adequacy of outflow. The limb is then placed in the dependent position to evaluate venous filling. The rate of refill, expressed as the venous filling index, is used to determine the presence and severity of reflux. A normal venous filling index is <2 mL/s, whereas higher levels (>4 mL/s) are abnormal.[59,60] Abnormal venous filling indices have excellent test characteristics to detect reflux (with sensitivity of 70% to 80% and positive predictive value of 99%) and have been found to correlate with the severity of CVI.[57–60] The function of the calf muscle pump in ejecting blood is determined after 1 and 10 repetitive contractions during toe raises. The volume of blood ejected with 1 tiptoe maneuver divided by the venous volume is called the ejection fraction. Complications of CVI, including ulceration, have been shown to correlate with the severity of venous disease assessed with the venous filling index and ejection capacity.[47,56,59] This technique provides quantitative information about several aspects of global venous function and may be used in the selection of intervention and assessment of the response to intervention.[61,62] APG may be clinically useful when the venous duplex does not provide definitive information on the pathophysiology of CVI, especially in C_3 through C_6, as recommended in the CPG (grade 1B).[18]

Computed Tomographic or Magnetic Resonance Venography

Advances in imaging with computed tomography and magnetic resonance have allowed for their use in the evaluation of venous disease.[63–66] These techniques are most useful to evaluate more proximal veins and their surrounding structures to assess for intrinsic obstruction or extrinsic compression. Optimal imaging of the venous system requires the use of intravenous contrast material, with appropriate timing of image acquisition on the basis of venous filling to obtain a venogram. Proper technique is required to avoid artifacts with inadequate venous opacification, with refinements allowing for better visualization to assess for obstructive disease, varicose veins, perforating veins, and other venous malformations. Both computed tomography and magnetic resonance venography may be used to define complex venous anatomy, such as ileofemoral venous obstruction, before intervention as recommended in the CPG (grade 1B).[18,63–66]

Other Techniques

Other techniques, such as photoplethysmography, strain gauge plethysmography, and foot volumetry, may also be used. These techniques provide information about global venous function. Photoplethysmography may be used to assess the time

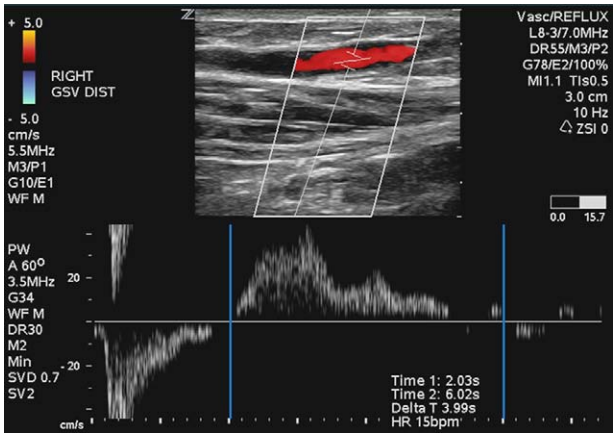

Figure 3. Venous duplex ultrasound demonstrating reflux in the great saphenous vein. Blood flow direction is determined after increasing central venous return with rapid cuff inflation then deflation. Flow in the direction of the feet is because of incompetent valves, as shown in red in the color image and above the baseline in the pulse Doppler. The Doppler spectrum quantifies the duration of reflux, and in the example above it is ≈4 seconds.

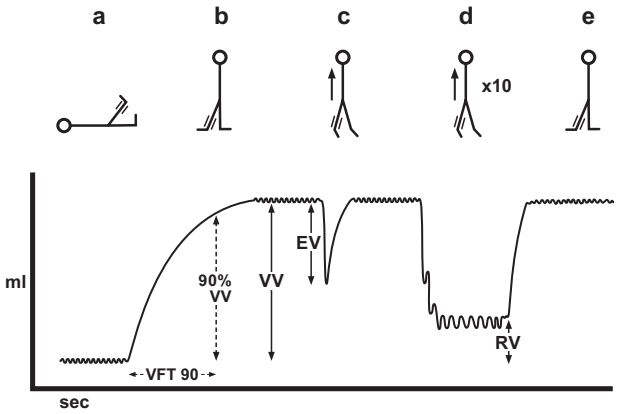

Figure 4. Air plethysmography (APG) measure changes in venous volume of the lower extremity over time. The ejection fraction (EF) is the amount of ejected volume (EV) after 1 tiptoe over the total venous volume (VV). The EF is a measure of the calf muscle pump function. The residual volume fraction (RVF) is the residual volume (RV) after 10 tiptoes over the venous volume (VV) and is the noninvasive measure of ambulatory venous pressure. The venous filling index (VFI) is 90% of the venous volume (VV90%) that fills within the venous filling time (VFT90) at 90% of the VV. The VFI measures venous valvular function and severity of global reflux. Reprinted with permission from Christopoulos et al.[58]

required to refill the veins within the dermis or the venous refill time. This is most useful to determine the absence or presence of reflux disease but correlates poorly with disease severity.[47,67] In contrast, strain gauge plethysmography and foot volumetry seem to correlate better with clinical severity of disease than duplex imaging.[68]

Invasive Testing

Contrast Venography

Venography may be used to directly visualize the venous system by either an ascending or descending approach (Figure 5).[69] Ascending venography involves injection of contrast in the dorsum of the foot with visualization of contrast-traveling cephalad in the deep venous system of the limb. Ascending venography provides details of venous anatomy that may be useful with surgical interventions and can help to distinguish primary from secondary disease. Descending venography involves proximal injection of contrast in a semivertical posture on a tilt table with the use of the Valsalva maneuver. It is most useful to identify reflux in the common femoral vein and at the saphenofemoral junction but may be used to evaluate other locations. A grading scheme has been developed on the basis of the anatomic extent of reflux. These modalities have been largely replaced by venous duplex imaging but may be performed if venous reconstruction is being contemplated.

Intravascular Ultrasound

Intravascular ultrasound is rapidly gaining acceptance in the management of venous disease and is increasingly being used to help guide interventions (Figure 6).[70] The technique uses a catheter-based ultrasound probe to visualize periluminal vascular anatomy to assess for obstructive or stenotic disease of the venous system. Intravascular ultrasound appears to be superior to venography in estimating the morphology

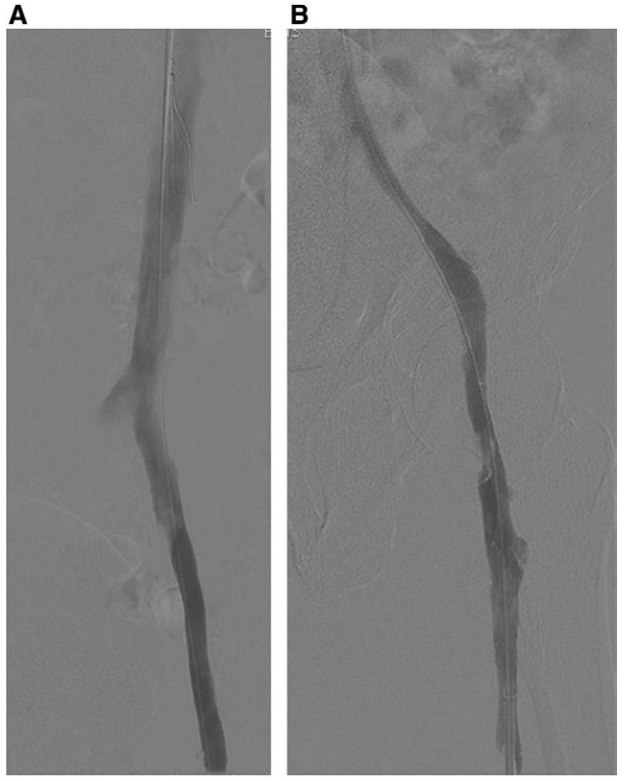

Figure 5. A, Venography of the iliocaval segment to assess for patency. **B**, Descending venography of the left lower extremity demonstrating reflux into the femoral vein in a postthrombotic vein.

and severity of central venous stenosis and in visualizing the details of intraluminal anatomy. This improvement in detection of the severity and significance of stenosis has led to greater consideration of venous percutaneous interventions in the treatment of CVI.[71]

Ambulatory Venous Pressure

Ambulatory venous pressure monitoring is the gold standard in assessing the hemodynamics of CVI.[72,73] The technique involves insertion of a needle into the dorsal foot vein with connection to a pressure transducer. The pressure is determined in the upright posture at rest and after exercise, such as during toe raises. The pressure is also monitored before and after placement of an ankle cuff to help distinguish deep from superficial reflux. Ambulatory venous pressure has been shown to be valuable in assessing the severity and clinical outcomes in CVI.[74] The mean ambulatory venous pressure and refill time are the most useful parameters. This technique provides information on global competence of the venous system. However, there is some concern that this pressure may not accurately reflect the pressure within the deep system.[75] This technique is seldom used in clinical practice because of its invasive nature, potential limitations, and alternative diagnostic modalities.

Selection of Studies

The purpose of the testing needs to be considered when selecting a diagnostic modality. Most of the noninvasive modalities may be performed to establish a diagnosis of CVI (Table 3). However, venous duplex imaging is the modality that is most

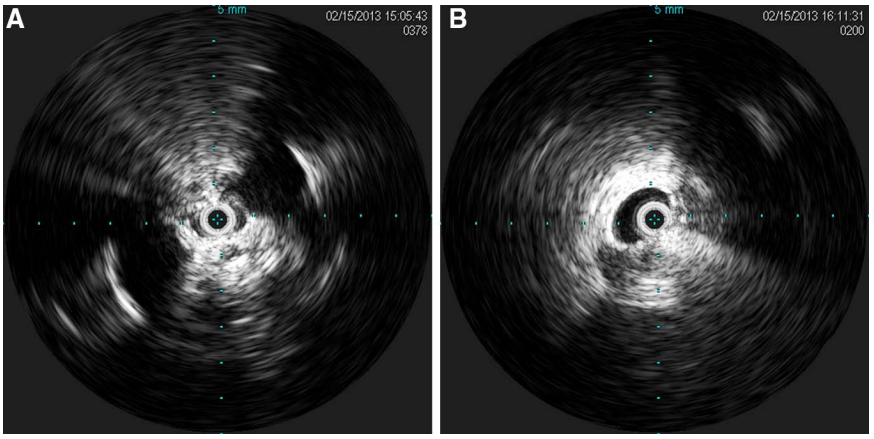

Figure 6. A, Intravascular ultrasound of compressed left common iliac vein with minimal lumen diameter. **B**, After angioplasty and stent of the left common iliac vein, there is significant improvement of the lumen. The hyperechoic Doppler spectra in both images is indicative of severe chronic fibrosis of the vein wall.

commonly used and provides information about the anatomic site of reflux that is required to plan an intervention. Other modalities, primarily APG, may be required if further information regarding the hemodynamic importance is needed to guide and monitor the response to therapy.

Treatment of CVI

Conservative Management

The initial management of CVI involves conservative measures to reduce symptoms and prevent development of secondary complications and progression of disease. The use of compressive stockings is the mainstay of conservative management and is further described below. If conservative measures fail or provide an unsatisfactory response, further treatment should be considered on the basis of anatomic and pathophysiologic features (Figure 7).

Regarding the management of CVI, the practitioner should be able to recognize the manifestations of CVI and use confirmatory testing, such as venous duplex reflux studies and perhaps APG for advanced and recurrent disease. Specific treatment is based on severity of disease with CEAP classes C_4 to C_6 often requiring invasive treatment. A referral to a vascular specialist should be made for patients with CEAP classes C_4 to C_6 (and probably for CEAP class C_3 for extensive edema). These patients with uncorrected advanced CVI are at risk for ulceration, recurrent ulceration, and nonhealing ulcers with progressive infection and even veno-lymphedema.

A healthy lifestyle including maintaining an ideal body weight or weight reduction if overweight may improve manifestations of CVI. Obesity is a well-established risk factor for the development of CVI and its complications. Weight reduction after bariatric surgery has been shown to improve manifestations of CVI, including edema and ulcers.[76] It may be extrapolated that weight reduction by other means might also assist in the treatment of CVI.

Table 3. Relative Value of Noninvasive Testing Modalities in CVI Based on Clinical Indication

	Duplex	APG	MRV/CTV
To establish a diagnosis	+++	++	++
To assess severity	+/–	+++	+/–
To determine anatomy	+++	–	+++
To determine hemodynamic significance	–	+++	–

APG indicates air plethysmography; CTV, computed tomography venography; and MRV, magnetic resonance venography.

Compressive Leg Garments

A therapeutic consideration for all of the CEAP clinical classes is compression therapy. The objective is to provide graded external compression to the leg and oppose the hydrostatic forces of venous hypertension. A number of compression garments are available, including graded elastic compressive stockings, paste gauze boots, layered bandaging, and adjustable layered compression garments. The use of graded elastic compressive stockings (between 20 and 50 mm Hg of tension) is a mainstay in the treatment of CVI (Table 4). Treatment with a 30- to 40-mm Hg compression stocking seems to result in significant improvement in pain, swelling, skin pigmentation, activity, and well being if compliance of 70% to 80% is achieved.[77] In patients with venous ulcers, graded compression stockings and other compressive bandaging modalities are effective in both healing and preventing recurrences of ulceration. With a structured regimen of compression therapy, complete ulcer healing can be achieved in >90% of patient with ulcers at a mean of 5.3 months.[78] Several studies have investigated the hemodynamic benefits of compression therapy in patients with CVI. Compression stockings have been shown to reduce the residual volume fraction, which is an indicator of improving the calf muscle pump function, and reduce reflux in vein segments.[79,80] Recently, studies evaluating reverse compression (antigraduated stockings or progressive elastic compression with higher pressure in the calf than the ankle), found improved calf muscle pump function on ambulation and reduced edema in severe CVI.[81,82] Further studies are required to determine whether reverse compression has a similar effectiveness to graduated compression stockings.

Compression therapy is considered first-line therapy for those with symptomatic varicose veins or greater (grade 2C) but not in candidates for great saphenous ablation. Compression therapy is recommended for both patients with venous ulcers (grade 1B) and as an adjunct to superficial venous ablation to reduce the risk of ulcer recurrence (grade 1A).[18] The prescription for elastic compression stockings in

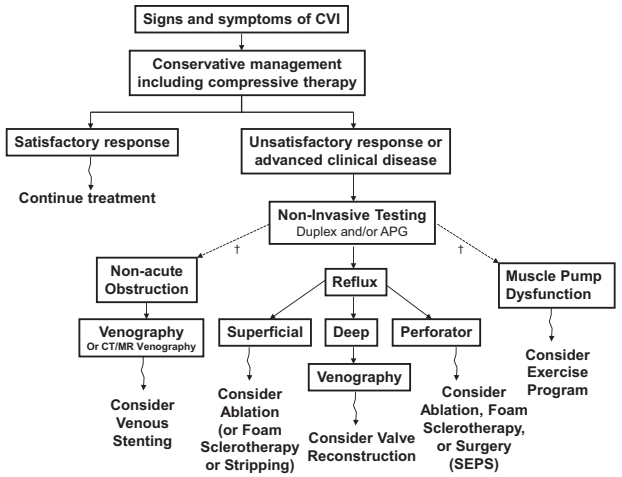

Figure 7. A simplified overview for the diagnosis and treatment of chronic venous insufficiency (CVI) on the basis of pathophysiologic mechanism. Multiple pathophysiologic mechanisms may contribute to CVI within the same patient and require a combination of treatment options. † indicates better assessment by air plethysmography (APG) as more limited information obtained from venous duplex imaging.

CVI includes information about the tension and length. The tensions is based on the clinical severity with 20 to 30 mm Hg for CEAP class C_2 to C_3, 30 to 40 mm Hg for CEAP class C_4 to C_6, and 40 to 50 mm Hg for recurrent ulcers. The most common-length stockings are knee length, because patient adherence is greater and symptom relief adequate. Stockings need to be changed every 6 to 9 months if worn daily with an alternate pair to avoid loss of the tension that the stocking exerts.

Wound and Skin Care

Advanced CVI may lead to compromised skin integrity; thus, it is important to maintain skin health and prevent infection. The use of topical moisturizers, often with lanolin, for the excessive dry skin helps to reduce fissuring and skin breakdown. The development of stasis dermatitis may need to be treated sparingly with topical steroid. Bacterial overgrowth may ensue with venous ulcers; thus, aggressive wound care is required to minimize infectious complications. A variety of hydrocolloids and foam dressings are available to control wound fluid drainage and maceration of the adjacent skin.[83] Silver impregnated dressings have been used to control infection and restore tissue integrity for infected ulcers, but the benefit is controversial.[84–86]

Biologic skin substitutes derived from tissue engineering have been used in treating difficult-to-heal ulcers with some success. There seems to be an improvement in ulcer healing when added to standard compression therapy but no difference in the rate of recurrent ulcers at 1 year.[87,88]

Table 4. Level of Compression Based on the Clinical Manifestation

Clinical Manifestation	Tension, mm Hg
Varicose veins with or without edema (C_2–C_3)	20–30
Advanced venous skin changes, or ulcers (C_4–C_6)	30–40
Recurrent ulcers	40–50

Pharmacologic Therapy

Four groups of drugs have been evaluated in the treatment of CVI, including coumarins (α-benzopyrenes), flavonoids (γ-benzopyrenes), saponosides (horse chestnut extracts), and other plant extracts. These drugs with venoactive properties are widely used in Europe but are not approved in the United States. The principle for using these venoactive drugs is to improve venous tone and capillary permeability, although a precise mechanism of action for these drugs has not been full elucidated. It is thought that the flavonoids act on leukocytes and endothelium to modify the degree of inflammation and reduce edema. A micronized purified flavonoid fraction has been shown to reduce edema-related symptoms as either primary treatment or in conjunction with surgical therapy.[89] A combination of coumarin and troxerutin (a flavonoid), with compression garments, was shown to reduced edema and pain compared with placebo at 12 weeks.[90] In addition, a meta-analysis found that micronized purified flavonoid fraction added to compression therapies aided in venous ulcer healing. Horse chestnut seed extract has been found to be as effective as compression stockings in the short term at reducing leg edema and pain from CVI, but the long-term safety and efficacy have not been established.[91,92] In general, venoactive drugs, which provide relief of pain and swelling or accelerate venous ulcer healing, carry a moderate recommendation in the CPG (grade 2B) when used in conjunction with compression therapy but are not approved by the US Food and Drug Administration.[18,83]

There have been several trials to suggest that pentoxifylline may improve healing rates of venous ulcers, although the magnitude of the effect appears to be small, and its role in patient management is unclear.[93,94] The use of pentoxifylline in conjunction with compression therapy is also recommended in the CPG (grade 2B), although it is not approved by the US Food and Drug Administration for this purpose.[18,83] The use of other agents, such as aspirin and platelet-derived growth factor, has been reported to promote healing or prevent the recurrence of venous ulceration, but no large randomized studies have been conducted. It should also be mentioned that there have been data to support the use of aspirin in the prevention of recurrent thromboembolic events in those with unprovoked thrombosis.[95] Sulodexide has been used in Europe to treat venous ulcers with relative success and has a modest recommendation by the CPG (grade 2B), but further studies are required to determine its clinical efficacy long term.[96]

Exercise Therapy

Abnormalities in calf muscle pump functions play a significant role in the pathophysiology of CVI. Graded exercise programs have been used in CVI to rehabilitate the muscle pump action and improve symptoms. A small controlled study randomly assigned patients with advance venous disease (CEAP class C_4–C_6) to structured calf muscle exercise or routine daily activities.[97] Venous hemodynamics were assessed with duplex ultrasound and APG, and muscle strength was assessed with a dynamometer. After 6 months, patients receiving a calf muscle exercise regimen normalized calf muscle pump function parameters but had no change in the amount of reflux or severity scores. It appears that structured exercise to re-establish calf muscle pump function in CVI may prove beneficial as

supplemental therapy to medical and surgical treatment in advanced disease.

Interventional Management

Sclerotherapy

Venous sclerotherapy is a treatment modality for obliterating telangiectases, varicose veins, and venous segments with reflux. Sclerotherapy may be used as a primary treatment or in conjunction with surgical procedures in the correction of CVI. Sclerotherapy is indicated for a variety of conditions, including spider veins (<1 mm), venous lakes, varicose veins of 1- to 4-mm diameter, bleeding varicosities, and small cavernous hemangiomas (vascular malformation). There are a number of sclerosing agents, including hypertonic solution of sodium chloride (23.4%); detergents such as sodium tetradecyl sulfate, polidocanol, and sodium morrhuate; and others such as sodium iodide and chromated glycerin. In the United States, sodium tetradecyl sulfate, polidocanol, glycerin, and sodium morrhuate are approved for use in treating varicosities.[18] In general for smaller-diameter veins the sclerosing agent needs to be diluted to avoid tissue inflammation and tissue necrosis. Sclerosing agents such as polidocanol have been shown to be superior to normal saline in both obliterating incompetent varicose veins and improving venous hemodynamics at 12 weeks.[98] In Europe, sclerotherapy with polidocanol foam with duplex ultrasound guidance has become standard in the treatment of intracutaneous telangiectasis, subcutaneous varicose veins, transfascial perforating veins, and venous malformations.[99] In a randomized, controlled trial, 430 patients with varicose veins with an incompetent GSV were treated with either ultrasound guided foam sclerotherapy or surgical ligation and stripping. Varicose vein recurrence was similar in both groups (11% versus 9% at 2 years); however, reflux was significantly higher in the foam group (35% versus 21% at 2 years).[100] Use of these agents for foam sclerotherapy is not currently approved by the US Food and Drug Administration; however, such therapy is used routinely to treat CVI. Sclerotherapy carries a high recommendation as a treatment option for varicose tributaries (grade 1B), whereas foam sclerotherapy carries a moderate recommendation as a treatment option for incompetent GSV (grade 2C) in the CPG.[18]

A common complication of sclerotherapy is hyperpigmentation of the surrounding skin from hemosiderin degradation. Attempts to minimize complications of sclerotherapy have been undertaken with microthrombectomy using multiple small incisions directly over the thrombosed varicosity to extrude the thrombus. A randomized, multicenter study found that microthrombectomy 1 to 3 weeks after injection of small varicose veins (≤1 mm) resulted in less hyperpigmentation and in varicose veins ≤3 mm resulted in less pain and inflammation.[101]

Endovenous Ablative Therapy

Thermal energy in the form of radiofrequency or laser is used to ablate incompetent veins. This technique is frequently used for GSV reflux as an alternative to stripping. The heat generated causes a local thermal injury to the vein wall leading to thrombosis and fibrosis. With radiofrequency ablation of the GSV there is complete obliteration in 85% after 2 years with recanalization in ≈11%.[102] However, 90% of patients treated with radiofrequency ablation are free from saphenous vein reflux, and 95% of patients report satisfaction and improvement of symptoms (regardless of the technical success).[102] Laser treatment with either an 810-nm or 940-nm diode has provided excellent results, with saphenous vein obliteration in 93% at 2 years and no cases of deep vein thrombosis.[103] Both radiofrequency and laser treatment are performed with tumescent anesthesia to prevent skin burns and reduce pain with earlier return to normal activities. A potential complication of ablation remains deep venous thrombosis and pulmonary embolism, although with a very low frequency. Several studies comparing endovenous ablation with conventional ligation and stripping found that the short-term efficacy and safety of ablation and surgery are comparable, with improved quality of life and earlier return to normal activity and work with ablation.[104,105] A randomized trial comparing laser ablation, radiofrequency ablation, foam sclerotherapy, and surgical stripping for GSV reflux found similar efficacy but higher technical failure after foam sclerotherapy and faster recovery after radiofrequency ablation and foam sclerotherapy compared with laser ablation and surgery.[106]

Endovenous ablation can also be applied to combined superficial and perforator reflux. A study of 140 consecutive endovenous ablation procedures (74 superficial and 66 perforator) preformed for ulcers that failed to heal with compression therapy (with a mean duration of 71±6 months) found a healing rate of 76% in 142±14 days, with an ulcer recurrence rate of 7%.[107]

On the basis of these studies, the CPG recommend endovenous thermal ablation over chemical ablation with foam sclerotherapy (grade 1B), and the treatment of pathologic perforator veins should be offered to patients with C_5 and C_6 disease (grade 2B).[18] Many studies have demonstrated the safety of thermal ablative techniques with low complications rates and a <1% venous thromboembolic rate.[18,107]

New ablative technologies are aimed at not using tumescent anesthesia to reduce pain and nerve injuries that can occur with thermal energy. A new method of mechanochemical ablation provides both mechanical injury and then a sclerosing agent to the endothelium to cause occlusion. In a small trial of 29 patients with GSV insufficiency, the primary occlusion rate at 6 months was 96.7%, with no adverse events reported.[108] Further studies with long-term follow-up in a variety of CEAP clinical classes are necessary to assess the equivalency of these tumescentless methods compared with other ablative methods.

Endovenous Deep System Therapy

Endovenous deep system therapy has become an increasingly important therapy in CVI to restore venous outflow and to provide relief from obstruction. Abnormalities in venous outflow, involving iliac veins, contribute to symptoms in 10% to 30% of patients with severe CVI.[35] Before endovascular therapy, iliac vein stenosis and obstruction causing CVI were treated with surgical procedures such as crossfemoral venous bypass or iliac vein reconstructions using prosthetic materials.[109] However, because of the success of venous stenting, surgical venous bypass is infrequently

performed. Several series of patients with CVI and outflow obstruction have shown that iliac vein stenting resulted in significant clinical improvement with complete pain relief in ≈50%, complete resolution of edema in ≈30%, and complete healing of ulcers in ≈50%.[110] The patency of iliac vein stents appears good, with primary patency of 75% to 80% at 3 to 6 years for nonthrombotic disease but ≈60% for thrombotic disease. Close follow-up is advocated to ensure that stent patency is maintained, because intervention for in-stent restenosis or reocclusion may be required in ≈25% of patients, particular those with thrombotic disease.[111] It should be noted that in-stent restenosis of >50% is relatively rare (≈5%). The success for iliac stenting on clinical outcomes appears to be durable, with 85% to 90% remaining free of recurrent ulceration at 5 years.[112]

Surgical Management

Surgical management of CVI may be considered to complement the compressive stocking in those refractory to medical and endovenous therapy. This includes patients with persistent discomfort with disability and nonhealing venous ulcers. Surgical options may also be considered in those unable to comply with compression therapy or those have recurrences of varicose veins. The potential procedures to be considered are based on the venous territories and underlying pathophysiologic mechanisms.

Surgery for Truncal Vein or Venous Tributaries

Surgery for truncal veins is directed toward preventing the consequences of reflux in the superficial venous system by interruption and removal, whereas surgery on vein tributaries is directed toward removal of the resulting varicosed veins. Excision of the GSV with high ligation of the saphenofemoral junction is considered durable and can be applied to all of the CEAP clinical classes (C_2–C_6) with GSV reflux.[113] Ligation and stripping have been shown to improve venous hemodynamics, to provide symptomatic relief, and possibly to assist in ulcer healing.[114,115] In a study evaluating 500 patients with venous ulcer and reflux of superficial and deep venous systems, random assignment to surgery (only to the superficial venous segments) plus compression demonstrated a significant reduction in ulcer recurrence at 12 months compared with compression alone (12% versus 28%) but failed to accelerate ulcer healing.[116] This finding supports the benefit of correcting the incompetent superficial venous system on prevention of ulcer recurrence, leading to a high level of recommendation (grade 1A) in the CPG; but this favored endovenous ablation over surgery to prevent ulcer recurrence (grade 2B).[18] In addition, venous tributaries that communicate with the incompetent saphenous vein and form large varicose vein clusters can be avulsed either at the same time or in a separate setting by stab or transilluminated power phlebectomy.[117] Concomitant or staged ambulatory phlebectomies of varicose veins during saphenous vein procedures (specifically ablation) are recommended in the CPG (grade 1B).[18]

Perforator Vein Surgery

Incompetent perforator veins may contribute to the pathophysiology of CVI and its advanced manifestations, primarily ulceration. In this situation, surgery may be performed to ligate perforator veins that may be contributing to the focal high pressure within the superficial veins at the site of an ulcer. However, this often presents difficulties with traditional surgical techniques because of the pre-existing tissue damage. Subfascial endoscopic perforator surgery provides a means to ligate incompetent perforator veins by gaining access from a site that is remote from the affected skin area. Several studies have shown that subfascial endoscopic perforator surgery is associated with a high rate of ulcer healing (≈90% at 3–12 months) and low rate of ulcer recurrence (<30% at 2 years), either alone or in conjunction with a procedure on GSV (ligation and stripping or ablation).[118–120] These studies are contrasted by a randomized, controlled study of 75 patients with class C_5 to C_6 who underwent saphenous surgery alone or in combination with subfascial endoscopic perforator surgery and found no short-term clinical benefit from adding subfascial endoscopic perforator surgery to saphenous surgery in patients with varicose ulcers and incompetent perforators. Current recommendations (grade 2B) in the CPG are to treat pathologic perforator veins (outward flow duration ≥500 ms, vein diameter ≥3.5 mm) located underneath healed or active ulcers in CEAP class C_5 to C_6 patients.[18] The choice of techniques to disrupt pathologic perforator veins remains an area of investigation but has been favoring less invasive methods, such as endovenous ablation and foam sclerotherapy.

Valve Reconstruction

Venous valve injury or dysfunction may contribute to the development and progression of CVI. Venous valve reconstruction of the deep vein valves has been performed selectively in advanced CVI with recurrent ulceration and disabling symptoms. An open technique for repairing the femoral vein valve has been described that renders the valve leaflets competent.[121] This technique of open valvuloplasty has been refined and closed techniques for venous repair developed with transcommissural valvuloplasty.[122] Venous valvuloplasty has been reported to provide competency in ≈60% and ulcer-free recurrence in ≈60% at 30 months. Complications from valvuloplasty include bleeding, deep venous thrombosis, pulmonary embolism, ulcer recurrence, and wound infections. Other procedures for reconstructing nonfunctioning venous valves resulting from postthrombotic valve destruction, which is not amenable to valvuloplasty, include transposition, transplant, cryopreserved vein valve allografts, and neovalve construction. Transposition of the profunda femoris vein or saphenous vein valve and axillary vein valve transplantation to the popliteal or femoral vein segments may be performed. Cryopreserved vein valve allografts have also been used but are limited because of frequent complications, such as early thrombosis, and poor patency and competency.[123] A novel technique involves neovalve construction of monocuspid or bicuspid valves from the intima and media of the thickened venous wall. Preliminary studies using this technique have been encouraging, with ulcer healing in 90% to 95% of patients with postthrombotic syndrome and active ulcers, but the studies are small.[124,125] The neovalve was competent in 95% to 100% of treated limbs at ≈2 years. Postoperative duplex scanning and APG showed significant improvement

in hemodynamic parameters and muscle pump function. The work on percutaneous placement of cryopreserved valves had some enthusiasm; however, the high rate of failure of most of these percutaneous valves in the treatment of valve dysfunction has limited their clinical use to date.

Summary

CVD is a common problem with a significant impact on both afflicted individuals and the healthcare system. Normal venous function requires the axial veins with a series of venous valves, perforating veins to allow communication of the superficial to the deep venous system, and the venous muscle pumps. Dysfunction of any of the normal structures may lead to venous hypertension and development of CVI. There is a spectrum of manifestations of CVI with an emphasis on more serious consequences, such as skin changes and venous ulceration. There are a number of noninvasive and invasive techniques to assist in the diagnosis and management. The most commonly used is the venous duplex ultrasound to confirm the diagnosis and provide anatomic detail. APG may be used to assist in assessing the severity of disease and response to treatment. The treatment of CVI will be based on the severity of disease and guided by anatomic and pathophysiologic considerations. Compressive garments have been a mainstay in the management of CVI. Traditional surgical techniques and newer interventional methods are often reserved for unsatisfactory response to conservative measures, although earlier use of venous ablation should be considered in symptomatic patients.

Disclosures

None.

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KEY WORD: Varicose ■ venous insufficiency ■ venous stasis syndrome ■ venous ulcer ■ venous valves