

Angiography showing varicose veins in the lower leg.

# Chronic Venous Insufficiency

Glenn Buczkowski, RPA-C, Cary Munschauer, BA,  
Michael A. Vasquez, MD, FACS, RVT

Varicose veins, venous stasis ulcers—the pain and disability of chronic venous disease are finally gaining appropriate attention among both clinicians and patients. A national screening program is raising awareness of venous insufficiency, and new management options are emerging to relieve patients' symptoms and improve cosmetic appearance.

## CONTINUING EDUCATION INFORMATION

**TARGET AUDIENCE:** This activity has been designed to meet the educational needs of physicians, physician assistants, and nurse practitioners involved in the management of patients with signs and symptoms of venous disease.

- **Original Release Date:** March 2009
- **Expiration Date:** March 31, 2010
- **Estimated Time to Complete This Activity:** 1 hour
- **Medium:** Printed journal and online CME
- Sponsored by Postgraduate Institute for Medicine

**PROGRAM OVERVIEW:** The primary objective of this educational initiative is to provide clinicians in primary care with the most up-to-date information regarding evaluation for chronic venous insufficiency and key treatment options.

**EDUCATIONAL OBJECTIVES:** After completing this activity, the participant should be better able to:

- Describe the physiology of chronic venous insufficiency (CVI).
- Identify factors from the history and physical examination that help distinguish CVI from other conditions in the differential diagnosis.
- List medical and surgical strategies that address the goals of therapy for CVI.
- Explain two endovenous ablative procedures that may benefit patients with specific characteristics.

**FACULTY:** Glenn Buczkowski, RPA-C, practices at the Venous Institute of Buffalo in North Tonawanda, New York, and is an Adjunct Assistant Professor of Surgery at Daemen College in Amherst, New York. Cary Munschauer, BA, is Research Coordinator at the Venous Institute of Buffalo. Michael A. Vasquez, MD, FACS, RVT, is a vascular and general surgeon and founder of the Venous Institute of Buffalo.

### PHYSICIANS

**Accreditation Statement:** This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of Postgraduate Institute for Medicine (PIM) and Quadrant HealthCom Inc. PIM is accredited by the ACCME to provide continuing medical education for physicians.

**Credit Designation:** Postgraduate Institute for Medicine designates this educational activity for a maximum of 1.0 *AMA PRA Category 1 Credit™*. Physicians should only claim credit commensurate with the extent of their participation in the activity.

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The American Academy of Physician Assistants accepts AMA category 1 credit for the PRA from organizations accredited by ACCME.

### NURSE PRACTITIONERS

This program has been approved by the Nurse Practitioner Association New York State (The NPA) for 1.0 contact hour.

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**C**hronic venous insufficiency (CVI) of the superficial, deep, or perforating veins is a common clinical disorder, with as many as one in five US adults affected.<sup>1-3</sup> Varicose veins are seen in as many as 33% of women and 20% of men in the US, while CVI-related edema, hyperpigmentation, and eczema may affect 3% to 11%.<sup>4</sup> The incidence of venous ulcers, which occur in about 0.3% of adults in Europe and the Americas,<sup>4</sup> increases to 20% in the presence of CVI.<sup>5</sup>

Although pigmentation changes and venous ulcers are the most striking signs of CVI, some patients have no visible evidence and complain instead of fatigue, heaviness, or aching in the legs. The combination of physical examination and imaging, including duplex Doppler ultrasonography (US), should allow the thoughtful practitioner to differentiate venous valvular incompetence from deep venous thrombosis (DVT) and other serious venous conditions, such as venous outflow obstruction.<sup>1,6</sup>

### DISEASE MECHANISMS

CVI refers specifically to the presence of valvular incompetence with secondary venous hypertension in the lower extremity. Risk factors for CVI include prolonged standing, increased body weight, failed muscle pump function in the lower extremity, trauma, pregnancy, and genetic predisposition.<sup>2</sup> Typical arterial risk factors (eg, diabetes mellitus, hypertension, smoking) are not risk factors for CVI.

Venous distention may lead to incompetence of the venous valves (see Figure 1) and chronically increased pressure in the lower extremity veins. Resulting overexpression of metallopro-

teinases can impair both the endothelium and the extracellular matrix, with possible damage to the vein wall.<sup>7</sup>

In addition, pressure-related injury to the epithelial cells leads to leukocyte migration and a generalized inflammatory response.<sup>8</sup> This prolonged hypertensive state induces circulatory changes that may result in hyperpigmentation, lipodermatosclerosis, and even ulceration.<sup>5</sup> Great saphenous vein reflux in the proximal lower extremity is responsible for possibly 60% of varicosities.<sup>1</sup>

### DIAGNOSTIC STEPS

The diagnosis of CVI encompasses a careful combination of patient history, physical examination, and findings on noninvasive duplex Doppler US (see table, page 20, and Figure 2, page 21).<sup>5</sup> The medical history should address the nature and severity of the patient's lower extremity symptoms, aggravating factors (including prolonged standing, history of lower extremity trauma, and prior vascular conditions or procedures), and mitigating factors (such as elevating the leg).<sup>4</sup>

The physical examination requires close inspection of the leg for evidence of visible or palpable varicosities and their origin, pigmentation irregularities, lipodermatosclerosis, stasis dermatitis, or atrophic blanche.<sup>4</sup> The limbs should be assessed for the presence of edema (unilateral or bilateral) and healed or active ulcers. The limbs should also be palpated for the presence of dilated saphenous veins.

The medical history should be reviewed for systemic disease, and conditions with similar presentation should be ruled out, including congestive heart

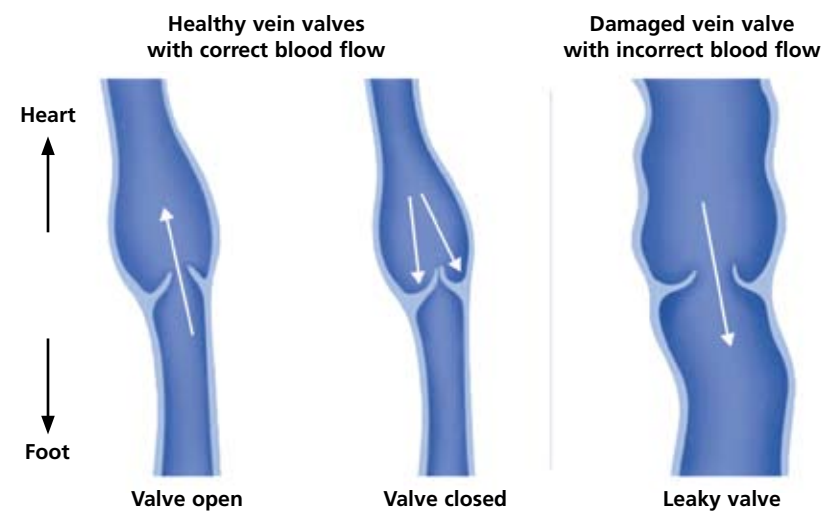
**Glenn Buczkowski** practices at the Venous Institute of Buffalo in North Tonawanda, New York, and is an Adjunct Assistant Professor of Surgery at Daemen College in Amherst, New York. **Cary Munschauer** is Research Coordinator at the Venous Institute of Buffalo. **Michael A. Vasquez** is a vascular and general surgeon and founder of the Venous Institute of Buffalo.

**FIGURE 1**

## Venous Reflux Disease

### Anatomic representation of the development of venous reflux

1. Vein valves become damaged or diseased, resulting in vein valve failure.
2. Reflux or backward flow occurs in the veins.
3. Pooling of blood causes pressure in the leg veins.
4. Increased pressure may cause surface veins to become varicose.



Source: Patient Presentation Slide VN20-87-8-08/07. San Jose, CA: VNUS Medical Technologies, Inc; 2008. Used with permission.

failure, renal dysfunction, spinal radiculopathies, lymphedema, soft-tissue mass, or DVT.<sup>5</sup> Possible adverse effects of certain medications (eg, calcium channel blockers, NSAIDs) should be considered. A history of recent or remote pelvic radiation can help distinguish the underlying cause of edema.<sup>9</sup>

Duplex Doppler US should be performed at a reputable facility by a technician familiar with the spectrum of venous disease. The deep venous system should be examined for the presence of reflux, and DVT should be ruled out. The presence or absence of reflux in perforating veins should also be noted. The superficial system should be evaluated for evidence of reflux or thrombophlebitis.<sup>1,4,5</sup> If the evaluation is negative for CVI, then other etiologies for the edema may be pursued.<sup>10</sup>

### Differential Diagnosis: Edema, Ulcers

The differential diagnosis for leg edema includes venous insuffi-

ciency, postphlebotic syndrome, lymphedema, edema resulting from systemic illnesses (eg, heart failure, renal failure), and idiopathic edema. The history and examination can help determine the cause; duration of the edema can help the clinician distinguish DVT (acute) from CVI or lymphedema (chronic).<sup>10</sup> If the patient reports the edema to be diminished or absent immediately after he or she wakes, a venous source rather than lymphedema may be indicated. Assessing the edematous limb for pain is important to distinguish between DVT (painful), CVI ("achy" or "full," in the patient's words), and lymphedema (generally painless).

In patients with lower extremity ulcers, the differential diagnosis can include arterial insufficiency, mixed arterial and venous disease, true venous stasis ulcers (see Figure 3, page 22), and ulcers of neuropathic origin.<sup>11</sup> Other causes of ulcers include infectious processes, rheumatoid dis-

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## TABLE

## Management of CVI: How to Proceed and When to Refer

### History

- Identify risk factors (family history of CVI; pregnancy, DVT, trauma)
- Signs and symptoms (right, left, bilateral): discomfort, swelling, varicose veins, pigmentation changes
- History of leg ulcer
- Presence of claudication
- Alleviating or aggravating factors

### Comprehensive vascular exam (arterial and venous)

- Carotid bruit
- Radial pulse
- Abdominal mass, AAA, groin nodes
- Legs (pulses, swelling, varicose veins, pigmentation changes, ulcer, induration, erythema, dilated/palpable GSV/SSV, phlebitis)

### Initiation of conservative care (following CVI studies):

- Weight loss as appropriate
- Elevation of affected leg
- Exercise/walking
- Compression hose: 20 to 30 mm Hg

If symptoms on initial presentation are minimal, a trial of conservative care, overseen by the primary care provider, is a reasonable option. If more significant symptoms are present or if symptoms persist despite conservative care, the patient should be referred to a vascular surgeon. In the presence of cellulitis or an ulcer, the patient should be referred expeditiously.

Abbreviations: CVI, chronic venous insufficiency; DVT, deep venous thrombosis; AAA, abdominal aortic aneurysm; GSV, greater saphenous vein; SSV, small saphenous veins.

Table courtesy of Glenn Buczkowski, RPA-C.

orders, hematologic syndromes (including clotting anomalies and sickle cell disease), vasculitis, and trauma.<sup>3</sup> It is important to be able to identify the various types of ulcers, as they require different therapies.

*Venous ulcers* typically appear with an irregular border and a shallow fibrinous wound bed. Eschar formation is rare, and underlying structures are usually not exposed.

In ulcers resulting from *arterial insufficiency*, the margins are regular, and the base is often covered with yellow fibrous material or necrotic eschar. Granulation tissue may be scant or absent, and exposure of underlying structures is more common. Exami-

nation may reveal hair loss, waxy skin that is cool to the touch, and absent distal pulses.<sup>3,11</sup>

*Neuropathic ulcers* are more often encountered in patients with diabetes mellitus. Diabetes may lead to paresthesia, lack of sensation, or burning in the feet. The neuropathic ulcer typically results from either a foot lesion that is poorly treated because sensation is lacking or callous breakdown resulting from improperly fitted shoes.<sup>3,11</sup>

When evaluating a *nonhealing* or *atypical ulcer*, the clinician must rule out any underlying infectious process that may be impairing normal healing mechanisms. Tissue biopsy is useful to exclude carcinoma and obtain

a sample for bacterial culture.<sup>11</sup> US is a mainstay in identifying an ulcer's underlying cause. CVI studies will reveal venous valvular incompetence within the deep, superficial, or perforator veins. Arterial Doppler US and ankle-brachial indexes will help the clinician evaluate distal perfusion and the extent of arterial disease.<sup>3,11</sup>

Though useful in planning interventions, venography and arteriography are invasive studies that have been largely replaced as initial diagnostic methods by noninvasive duplex Doppler US.<sup>4,5</sup>

### TREATMENT OF VENOUS STASIS ULCERS

Several medical and surgical strategies are currently used to treat venous stasis ulcers. The goals of therapy are to heal existing ulcers and to prevent new ulcers from forming. Minimizing increased venous pressure will reduce lower extremity edema and allow ulcers to heal.<sup>8</sup>

Among conservative treatment options, compression hose serve as a compensatory mechanism for lower extremity venous hypertension; they should be used only if arterial insufficiency has been ruled out. Elevating the leg at regular intervals throughout the day and overnight may reduce pressure and swelling. Recreational walking is recommended to improve muscle tone and intrinsic calf compression.<sup>11</sup>

Legendre et al<sup>12</sup> recommend a complete laboratory evaluation to assess nutritional status in patients with leg ulcers. In patients with serum albumin levels below 35 g/L, they found an increased likelihood for ulcers to become enlarged over 12 weeks. They also found a correlation between lower serum albumin values and the presence of an inflammatory process involving the ulcer.<sup>12</sup>

If medical hosiery proves insufficient to improve ulcers, sim-

ple elastic wrap dressings or commercial multilayered inelastic dressing systems, such as Unna boots, may help.<sup>13</sup> Unna boots provide maximum compression during activity and muscle contraction but comfortable compression during rest<sup>3</sup>—both important features for ambulatory patients. Because Unna boots do not stretch to accommodate increased edema, they must be removed frequently for inspection of the limb, then replaced with a new boot, to maintain proper compression.<sup>11</sup>

If these strategies are insufficient, refractory edema can be reduced with additional compression applied by sequential pump therapy.<sup>3,11</sup> If an ulcer has not improved within four weeks, the consensus is to evaluate it through biopsy for cancerous changes.<sup>3</sup>

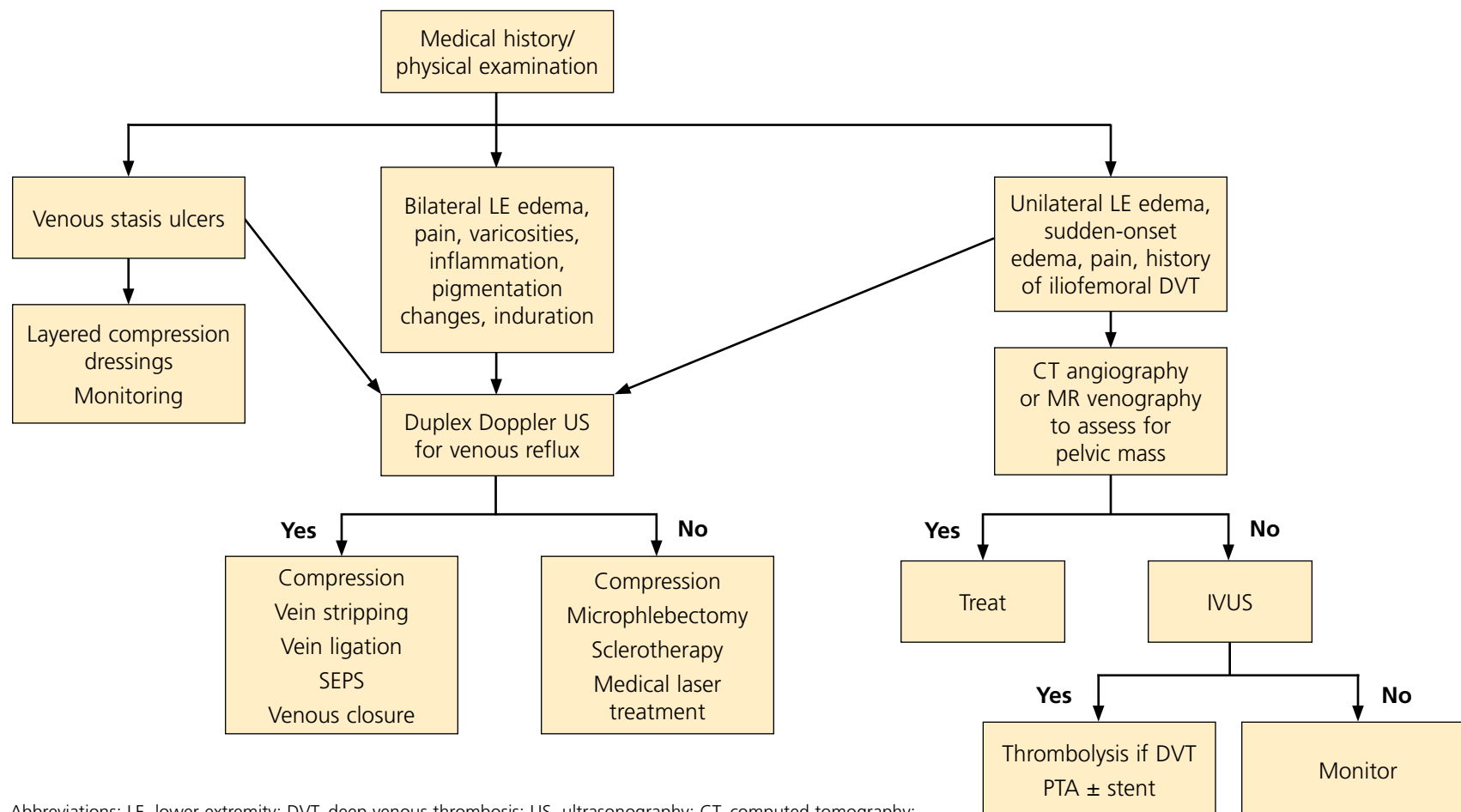
Venous stasis ulcers may be treated surgically by direct or indirect means. Direct excision of the ulcerated tissue with subsequent flap coverage may be beneficial if the contributing venous disease is also addressed.<sup>11</sup> Indirect treatment through endovenous ablation, stripping, ligation, or avulsion of implicated superficial veins, as well as increased pressure in the affected extremity, often achieves healing of the ulcer without excision. A combination of compression and venous ablation has been shown to produce healing in 86% of venous stasis ulcers.<sup>14</sup>

Incompetent perforating veins, which contribute to the development of venous stasis ulcers, can be effectively treated with subfascial endoscopic perforator vein surgery (SEPS), endovenous ablation, US-guided sclerotherapy, or ligation.<sup>3</sup>

To prevent recurrence of venous stasis ulcers, patients should be encouraged to continue their compression therapy after surgical procedures. They should also continue leg elevation and

**FIGURE 2**

**Evaluation for Chronic Venous Insufficiency**



Abbreviations: LE, lower extremity; DVT, deep venous thrombosis; US, ultrasonography; CT, computed tomography; MR, magnetic resonance; SEPS, subfascial endoscopic perforator vein surgery; IVUS, intravascular ultrasonography; PTA, percutaneous transluminal angioplasty.

Algorithm courtesy of Glenn Buczkowski, RPA-C.

exercise to reduce edema and improve function of the calf muscle pump.<sup>11</sup>

**Symptom Relief and Other Benefits**

In the absence of great or small saphenous vein reflux, several therapies may be considered to relieve symptoms and improve cosmetic appearance. Sclerotherapy obliterates veins by introducing an agent that irritates and scars the vein walls, leading to venous thrombosis; it is effective to treat smaller veins.<sup>5</sup> Microphlebectomy involves selective excision of individual veins or interruption of clusters of superficial veins.<sup>3-5,12,15,16</sup>

Incompetent perforating veins may improve with use of isolated radiofrequency ablation (RFA), foam sclerotherapy, or SEPS.<sup>4,5,17-21</sup>

Eliminating perforating veins leads to decreased pressure between the superficial and deep systems and helps decompress the leg.<sup>4,18-21</sup> Compression, too, may help relieve symptoms associated with perforator disease.<sup>11</sup>

If superficial reflux is present—*saphenofemoral* (involving the great saphenous vein) or *saphenopopliteal* (involving the small saphenous vein)—associated symptoms can be relieved by one of several methodologies. Traditionally, the gold standard for treatment has been saphenous vein stripping.<sup>15,16</sup> However, the healing time can be long and associated morbidity rates are high. Additionally, residual varicosities or varicosities fed by still-intact veins can produce persistent symptoms.<sup>15</sup>

**New Procedures for Saphenous Vein Reflux**

In recent years, two endovenous ablation procedures to treat saphenous vein reflux have increased in use: endovenous laser treatment (EVLT) and RFA therapy. EVLT involves medical laser-generated heat to thermally injure the vein wall, producing early thrombosis and late fibrotic changes.<sup>5</sup> Though successful in causing long-term occlusion of the great saphenous vein (with two-year closure rates nearing 100%<sup>22</sup>), EVLT is not frequently used in below-knee procedures because of the increased possibility of nerve injury.<sup>1</sup>

RFA catheters heat the vein lumen by way of alternating current. This effect causes intimal scarring and thrombosis, result-

ing in venous occlusion.<sup>14,22</sup> This procedure has also been used successfully to treat CVI, with two-year occlusion rates approaching 90% for the first-generation catheter.<sup>22</sup>

Contraindications to EVLT and RFA therapy include isolated deep reflux, DVT in the ipsilateral limb, venous outflow obstruction, arterial insufficiency (ankle-brachial index, < 0.8), intention to become pregnant, and obesity that obscures visualization of the saphenofemoral junction.<sup>14</sup> Both procedures are generally performed on an outpatient basis using local and tumescent anesthesia to provide adequate pain relief.<sup>14,23</sup>

The catheter system is introduced percutaneously and

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FIGURE 3

## Venous Stasis Ulcer



©Watney Collection/Phototake

The classic appearance of an ulcer resulting from chronic venous insufficiency, with an irregular border and shallow wound bed. No eschar is present, and underlying structures are not exposed.

advanced to a safe distance (2.0 cm for RFA or EVLT) from the saphenofemoral junction. In either procedure, correct positioning is confirmed by duplex Doppler US, which also guides circumferential placement of the tumescent anesthesia mixture, a dilute lidocaine solution (30 mL of 1% lidocaine with epinephrine in 500 mL of saline).<sup>23</sup> The mixture creates the effect of a local anesthetic, providing a heat sink to guard against thermal injury to surrounding tissue and circumferential compression of the treated vein. The catheter is then activated and pulled back at specific time and distance intervals.

Finally, US is performed to ensure closure of the vein and patency of the junction with the deep system. The limb is wrapped in layered compressive dressings.

At discharge, the patient is advised to resume normal activities with three exceptions: heavy exercise or other activities that would cause the treated vein to dilate; immersion in water before the puncture site is fully healed;

and air travel before the first postprocedure US. The patient is encouraged to walk daily and to wear compression hose consistently during waking hours once the surgical bandage is removed (24 hours after the procedure) for at least several months.<sup>14</sup>

### FOLLOW-UP

Follow-up duplex Doppler US is performed in the early postprocedural period to rule out development of DVT and evaluate the treated vein for closure.<sup>1,14</sup> Although no universal guidelines have been established for follow-up visits or US screening, it is important for each center where endovenous ablation is performed to implement a protocol to evaluate patient outcomes. Doppler US may be performed at each visit in conjunction with an outcome assessment measurement; the Venous Clinical Severity Score (VCSS),<sup>14</sup> for example, is a symptom-scoring questionnaire comprising severity ratings for pain, varicosities, edema, pigmentation, inflammation, induration, and the presence, size,

and duration of stasis ulcers, as well as the use of compression. The VCSS, scored at baseline and three times during follow-up evaluations (ie, three to four days, three to four weeks, and three to four months postprocedure), can provide important information about CVI signs and symptoms and the degree of resolution after EVLT or RFA.<sup>14</sup>

### Potential Concerns

Complications after endovenous ablation are infrequent, but they may include hyperpigmentation, phlebitis, paresthesia, erythema, ecchymosis, infection, thermal injury, bleeding, pain, treatment failure, and DVT.<sup>14,22,24</sup> The most potentially serious complication is DVT, which can occur at several sites following ablation: the saphenofemoral junction, the saphenopopliteal junction, and the tibial, popliteal, and femoral vessels. Observed rates of postprocedure DVT range from less than 1% to more than 15%,<sup>14,22,24</sup> although investigations reporting higher DVT rates may have involved treatment of the vein closer to the junction with the deep system than is recommended.<sup>22</sup>

Erythema and infection may be attributable to breaks in sterile technique, while thermal injury may be explained by inadequate infiltration of tumescent anesthesia to shield perivenous structures from heat.<sup>25</sup> Postprocedure bleeding may be a result of inadequate compression or misapplication of dressings.

Possible causes for treatment failure include inappropriate patient selection, failure to follow catheter protocol, and device malfunction. Paresthesia, ecchymosis, phlebitis, and pain are generally mild and transient, and phlebitis may result from the presence of large or superficial varicosities. Phlebitis responds well to anti-inflammatory agents, compression, and topically applied heat. Paresthesia and ec-

chymosis are generally self-limiting, and postprocedural pain usually subsides within a short period.<sup>22,24,25</sup>

In long-term follow-up, some patients may report symptoms that return or worsen, despite a persistently occluded great saphenous vein. These patients should be evaluated for the presence of below-knee great saphenous or small saphenous reflux or incompetent perforating veins.<sup>14,24,26</sup> Only with further treatment will symptom relief be complete.

### ADDITIONAL CONSIDERATION: ILIAC VEIN LESIONS

In the course of evaluation for CVI, it is important to rule out proximal venous lesions as a cause of lower extremity edema.<sup>27</sup> *Iliac vein lesions*, whether postthrombotic, intrinsic, stenotic, or resulting from external compression, increase venous hypertension when combined with venous reflux and contribute to more pronounced symptoms than does reflux alone.<sup>28</sup> In addition, intra-abdominal pathologic conditions should be excluded as the cause of outflow obstruction in the lower extremity.

Iliac vein lesions present across a spectrum, ranging from an incidental finding of asymptomatic lesions to unilateral or bilateral edema with or without sudden-onset leg pain.<sup>29,30</sup> Postthrombotic lesions, resulting from previous DVT, manifest as distal edema, pain, venous claudication, and ulcer formation.<sup>31</sup> Despite adequate anticoagulation at the initial event, almost half of all patients treated for iliofemoral DVT experience late venous claudication.<sup>28,31</sup>

Nonthrombotic iliac vein lesions include intraluminal web formations and stenoses.<sup>30,31</sup> Although these lesions may be incidental findings in asymptomatic patients, they can manifest as a clinical syndrome called *iliac vein*

compression syndrome, also called *May-Thurner syndrome* or *Cockett syndrome*. Affected patients generally present in one of three ways:

- First, sudden-onset, left-sided iliofemoral thrombosis with pain and unilateral leg swelling. This presentation is most often seen in women. Following thrombectomy, an anatomic anomaly is detected in which the iliac vein crosses under the iliac artery.

- Second, long-standing symptoms of CVI in patients who are discovered to have iliac vein stenosis related to arterial compression.

- Third, chronic occlusion of the left common and external iliac veins and collateral formation from the common femoral vein.<sup>31</sup>

### Quantifying and Managing Iliac Venous Stenosis

What constitutes a hemodynamically significant stenosis? In arterial investigations, narrowing of the vessel by more than 75% in cross section has been determined to represent significant stenosis.<sup>32</sup> However, venous elasticity makes it more difficult to quantify venous narrowing. In recent research, patients with the greatest symptom improvement after venoplasty originally had lesions measuring at least 50% by intravascular ultrasonography (IVUS).<sup>28</sup>

Diagnosis of iliac vein compression syndrome with conventional duplex Doppler US is difficult because of overlying pelvic structures and the likely presence of collateral outflow vessels. CT angiography, magnetic resonance venography, and IVUS are useful methods for pinpointing an iliac vein lesion related to overlying arterial compression.<sup>31</sup>

IVUS may provide the most accurate means of evaluation because it images the weblike structures seen in iliac lesions and reveals anomalies in the vein wall.<sup>30,31</sup> This minimally invasive

## The National Venous Screening Program<sup>6,34</sup>

Launched in 2005-2006 by the American Venous Forum to increase clinician and patient awareness of venous disease, the National Venous Screening Program<sup>34</sup> initially involved 17 centers, 83 clinicians, and 2,234 patients. It comprised evaluation of demographic data, assessments of venous thromboembolism risk and quality of life, duplex Doppler US for venous reflux and obstruction, and clinical inspection.

Patients were given the opportunity to meet with a clinician to review assessment results. They received educational materials and a "report card" that could be shared with the primary care provider at additional follow-up.

The results of the first screening program were dramatic: 77% of patients were found to be at high or very high risk for venous thromboembolism; US revealed venous reflux in 40% and venous obstruction in 6%; 20% of patients had clinical severity class scores (ie, CEAP) of 3 or higher (range, 0 to 6).

Despite the program's efforts and a subsequent expansion from 17 to 83 participating centers, McLafferty et al<sup>6</sup> reported in 2008 that the presence of venous disease in a large screening group remained significant (eg, 37% with venous reflux, 5% with venous obstruction, and 21% with CEAP scores  $\geq$  3).

Abbreviations: US, ultrasonography; CEAP, clinical, etiologic, anatomic, pathophysiologic measures.

Data extracted from: McLafferty et al. *J Vasc Surg*. 2008<sup>6</sup>; McLafferty et al. *J Vasc Surg*. 2007.<sup>34</sup>

percutaneous procedure has a high rate of success and a low incidence of complications.<sup>21</sup>

Once an iliac vein lesion has been identified, the treatment algorithm indicates venoplasty with or without stenting.<sup>28-31</sup> The high rate of restenosis (attributed to the overlying compressive effect of the iliac artery) has led some investigators to recommend stenting in all cases.<sup>30,31</sup> The overall consensus is that stenting is safe, with minimal complications encountered, and durable, with low reported rates of stent occlusion or refractory stenosis.<sup>27-31</sup>

In a recent follow-up study on iliac vein compression, Neglén<sup>33</sup> reports lasting poststenting improvements in swelling, pain, and ulcer healing, whether or not residual reflux occurs or additional saphenous vein procedures are performed, and regardless of the underlying cause of the obstruction. Neglén<sup>33</sup> suggests that patients who do not respond to conservative therapy, those with a history of DVT, and those who have symptoms without identifi-

able pathology are good candidates for stenting. The researcher recommends stenting when IVUS evaluation reveals morphologic obstruction exceeding 50%.

### CONCLUSION

Venous disease is a significant issue in clinical medicine, and efforts are ongoing to increase clinician and patient awareness of CVI and its medical ramifications (see "The National Venous Screening Program,"<sup>6,34</sup> above). The scope of the problem includes chronic pain and disability, as well as the presence of varicose veins. Diagnostic efforts must take into consideration the full spectrum of symptoms and causality. Treatment of venous disease should originate from a broad-based approach, and outcomes should be followed. Clinicians should offer various tailored treatments with options to address commonly encountered problems. The desired end result is resolution of symptoms and reduction in sequelae, with minimal morbidity. **CR**

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