

Evaluation and Treatment of Lower Extremity Superficial Venous Insufficiency

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Abstract: *Chronic venous disease of the lower extremity is frequently not diagnosed, misdiagnosed or untreated. Clinical findings are often minimized as cosmetic or simply attributable to the aging process. This is one of the greatest misnomers among both medical providers and the lay public. This article will review the pathophysiology, clinical findings, diagnosis, and treatment of lower extremity venous insufficiency. (see color figures on p.51.)*

Introduction

Lower extremity chronic venous disease (CVD) is a condition whose spectrum ranges from abnormalities such as “spider veins” to severe edema, skin ulceration, and major disability. The importance of chronic venous disease is related to the large number of people with the disease and the socioeconomic impact of its more severe manifestations. More important is the significant adverse effect on an individual’s quality of life (QOL).¹ Chronic venous disease is often overlooked by providers because of an under appreciation of the magnitude and impact of the problem. These are the patients who repeatedly appear in the emergency room at 2 a.m. with leg pain yet have no evidence of deep venous thrombosis. These patients may present with moderate to severe chronic edema; however, the lack of a proper diagnosis leads to an incorrect treatment with chronic diuretics. Symptoms such as leg discomfort, fatigue, and restless legs, are often minimized by providers or relegated to vague diagnoses such as fibromyalgia or restless leg syndrome. Many patients do not seek or receive care until development of venous ulceration. The aim of this article is to update the clinician regarding current diagnosis and treatment.

Normal Venous Anatomy and Function

The veins of the lower extremity are divided into the superficial and deep venous systems connected by a series of perforator veins. The deep venous system is located below the muscular fascia and serves as the primary conduit for venous return. The deep veins follow the course of the major arteries and return greater than 90% of blood volume from the lower extremity. The superficial venous system is located above the muscular fascia layer and functions as a reservoir and conduit to the deep venous system. The principal veins of the superficial system are the great and small saphenous veins. By International Consensus, the word “great” replaces “greater” or “long.” The word “small” replaces “lessor” or “short.” The great saphenous vein runs from the medial ankle along the medial leg to join the common femoral vein. The small saphenous

vein runs along the midline of the calf between the medial and lateral gastrocnemius muscles and typically joins the popliteal vein. Innumerable small cutaneous veins arborize to join superficial branches, which drain into the truncal great, and small saphenous veins. The superficial veins are connected to the deep system by numerous perforating veins in the thigh and leg that pass through anatomic fascial spaces.²

In the erect position, venous blood return must rise against both gravity and fluctuating thoracoabdominal pressures. A large number of one-way bicuspid valves function in concert with the lower extremity muscle pump to facilitate venous return. The muscle pump, primarily the calf, forces blood out of the venous plexus and up the deep venous system. The valve system insures that blood moves only in the cephalic direction and blocks gravitational forces from pulling blood back to the feet. This prevents a significant increase in hydrostatic pressure within the distal veins of the lower leg and feet. Perforator valves prevent transmission of high pressure and flow from the deep system to the superficial system.

Venous Pathophysiology and Dysfunction

Venous pathology develops when the return of blood from the lower extremity is impaired and venous pressure is increased. In the majority of cases, this results from venous valve incompetence. When the bicuspid valves do not correctly oppose, retrograde flow of blood occurs. This is known as venous insufficiency or venous reflux and may occur in the deep, superficial and perforator veins. Venous reflux results in venous hypertension. Dysfunction of the deep venous valves is most often a consequence of deep venous thrombosis (DVT.) Incompetence of the valves in the superficial venous system may be Primary due to preexisting weakness of the vessel wall or valve leaflets, or Secondary due to direct injury, superficial phlebitis, or excessive venous distention.^{3,4}

When this occurs locally in small cutaneous veins, it can be manifested as telangiectasias (spider veins.) Visually, these appear as fine cutaneous red, purple or blue veins less than 1mm in diameter. These are often seen in relation to larger 1-3 mm subdermal blue or blue-green veins known as reticular veins. (Figure 1, p.51) While these entities are often considered cosmetic, patients may experience symptoms such as itching, burning or achiness. These symptoms are often exacerbated by factors promoting vasodilation such as heat or elevated hormone levels (menstrual cycle, pregnancy.)

Failure of valves located at the saphenous-femoral and saphenous-popliteal junctions substantially increases hydrostatic pressure within the superficial veins. There is usually

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concomitant valve failure in larger secondary and tertiary branches of the saphenous vein. The markedly elevated venous pressures may result in progressive venous dilation and thickening of large venous branches, which manifest visually as varicose veins. “Varicose veins” are defined as any vein abnormally dilated greater than 3 mm.

The effects of venous hypertension are transmitted to the microcirculation and eventually result in venous microangiopathy. The normal reabsorption of perivascular fluids by osmotic and pressure gradients is impaired, resulting in accumulation of perivascular and lymphatic fluid. This leads to edema, fibrosis, and impaired oxygenation of surrounding tissue.⁵ The disruption of normal vascular and lymphatic flow of the lower extremities may result in the symptoms of chronic venous insufficiency and lymphedema.

Venous obstruction, deep vein reflux, muscle pump failure and congenital abnormalities are less common causes. Venous obstruction and deep venous reflux are the most common secondary causes of CVD and are almost always the result of DVT.⁶ Iliac venous stenosis or obstruction may be detected in 10-30% of patients with lower extremity swelling. Venous insufficiency of ovarian, parauterine, and internal iliac veins (Pelvic Congestive Syndrome) may contribute significantly to lower extremity symptoms.

Clinical Manifestations

Chronic venous insufficiency (CVI) is the clinical entity that results from chronic venous hypertension. Numerous risk factors contribute to the development of venous insufficiency (Table 1).⁷⁻⁹ Hereditary factors play a major role. If a single parent experienced venous disease, there is a 50% likelihood of the child presenting with a similar condition and nearly 100% probability if both parents had CVD. Many patients report their first telangiectasia as a teenager. The incidence of venous insufficiency increases with age. Varicose veins occur in 8 % of women aged 20-29 years, increasing to 41% in the fifth decade and 72% in the seventh decade of life. Prolonged hormonal influences related to female gender, pregnancy, and birth control pills increase the incidence. It has been estimated that almost 70% of women develop telangiectasias during pregnancy. Men are affected less than women (41% versus 72% in the seventh decade.) Prolonged standing/sitting, obesity, limb trauma and limb surgery are other risk factors.

Patients often present with concerns regarding the cosmetic appearance of their leg veins. They frequently do not recognize CVI symptoms (Table 2) due to the insidious progression of venous insufficiency. They have acclimated to their condition, incorrectly attribute their symptoms to another cause, or consider them part of the aging process. Directed specific questioning is required.

Complaints of lower extremity fatigue or heaviness are common. Ankle or calf edema is also common and may be severe (Table 3, p.25); however, early evidence may only be calf fullness and is often not recognized by the patient. Edema

Table 1 Risk Factors for Chronic Venous Insufficiency

Heredity
Female gender
Pregnancy
Hormone therapy
Age
History of prolonged standing or sitting
Obesity
Leg trauma or surgery

Table 2 Leg Symptoms Associated with Chronic Venous Insufficiency

Arching
Throbbing
Cramping/Night Cramps
Heaviness
Fatigue
Swelling
Restless legs
Itching, Burning, Numbness, Tingling

is typically described as completely resolved upon awakening and progressively worsens throughout the day. Patients may report cramping or pain that often awakens them. They may describe chronic aching or throbbing. Other complaints include skin itching, burning, tingling, and numbness. Patients frequently complain of restless legs and venous insufficiency symptoms are these are often mischaracterized as “restless leg syndrome.” Generally all these symptoms are worse with prolonged standing or sitting and are most noticeable at the end of the day. Leg elevation, walking and exercise improve symptoms.

The patient should be evaluated while standing to allow maximal distention of the veins and elicit dependent venous congestion. Calf edema is best evaluated viewing the posterior calves from a distance of 4-5 feet. (Figure 2, p.51) Direct careful attention to the skin of the medial ankle as this region is the most dependent and vulnerable to the effects of long-term venous hypertension. If saphenous reflux is severe, there may be focal or diffuse increased pigmentation from hemosiderin deposition. (Figure 3, p.51) There may be erythema, rubor or a dusky appearance to the feet. Long-standing venous hypertension and distal venous stasis leads to more advanced skin changes within the distal leg, ankle, and foot. These include eczema, lipodermatosclerosis, atrophe blanche and

Table 3 Differential Diagnosis of Lower Extremity Edema

Venous obstruction
Chronic right-sided heart failure
Pericardial effusion
Venous insufficiency
Tricuspid stenosis
Tricuspid regurgitation
Pericarditis
Cirrhosis
Cellulitis
Premenstrual fluid accumulation
Low albumin
Medications (Calcium channel blockers)
Lymphatic obstruction
Preeclampsia–eclampsia
Myxedema
Compartment syndrome
Malignancy
Pelvic tumor
Pregnancy
Deep venous thrombosis
Fluid overload

ulceration. (*Figure 4, p.51*) Lipodermatosclerosis is a dark leather-like fibrotic thickening of the skin. Atrope blanche is focal hypopigmentation resulting from skin ischemia/infarct.^{10,11} Uncorrected, advanced CVI patients are at risk for ulceration and non-healing wounds as well infection and lymphedema.

Venous ulcers from great saphenous vein insufficiency usually occur in the medial supramalleolar area and in the lateral malleolar area from small saphenous vein insufficiency. Long-standing CVI may also lead to the development of secondary lymphedema, representing a combined disease process. Lymphatic dysfunction is present in up to one third of patients with chronic venous insufficiency and may resolve with correction of the venous abnormalities.¹²

Large tortuous bulging varicose veins are not present in most patients with venous insufficiency. Scattered spider veins or reticular veins may be the only cutaneous finding in 30–40 percent of patients. While the history and physical exam may be suggestive, diagnosis requires a lower extremity venous duplex examination.

Duplex Ultrasound

Advances in affordable venous duplex ultrasound technology have revolutionized the treatment of venous disease. A properly performed duplex ultrasound is the primary diagnostic test to identify and characterize venous insufficiency.¹³⁻¹⁶ This has replaced other modalities such as hand-held Doppler, photoplethysmography, air plethysmography, and contrast venography. Venous duplex imaging combines grey scale imaging of the deep and superficial veins with pulsed Doppler assessment of venous flow. The aim of duplex ultrasound is to define all the incompetent superficial venous pathways. The deep venous system is assessed to exclude DVT or venous obstruction.

It is very important to recognize that a duplex ultrasound for CVD is very different than a study of the lower extremity for DVT. The patient must be examined while standing which requires a platform or a tilt table. Duplex studies for DVT are performed with the patient supine and venous reflux is usually not detected. A thorough knowledge of the superficial venous system anatomy and common variants is necessary. Unfortunately, most imaging centers and hospitals lack these capabilities. Many patients experience significant worsening of their lower extremity pain, cramping, and swelling in the late evening. They present to the emergency room where a venous duplex is performed with the patient supine. DVT is excluded; however, the true diagnosis is missed. Even most vascular technologists lack sufficient training and familiarity with duplex testing of the superficial venous system. One credentialing body, Cardiovascular Credentialing International (CCI), has addressed this deficiency by offering an examination to vascular ultrasound technologists to be certified as a Registered Phlebology Sonographer (RPhS.)

Any significant venous flow toward the feet is diagnostic of venous insufficiency. Venous reflux is diagnosed when there is reversal of flow from the expected cephalic direction for more than 0.5 seconds following a provocative maneuver such as calf/foot compression by the examiner, dorsiflexion by the patient, or a Valsalva maneuver.¹⁶ Significant reflux is identified in the great saphenous vein or one of its primary tributaries in 70-80% of patient's with CVI symptoms. Small saphenous vein reflux is found in 10-20% of patients and tributary non-saphenous superficial reflux is identified in 10-15%.^{17,18}

Duplex ultrasonography cannot reliably assess the iliac veins and the inferior vena cava. Additional imaging such as computed tomography venography, magnetic resonance venography, or contrast venography may be required to characterize venous obstruction, stenosis, or a venous anomaly in the pelvis. The iliac veins should be assessed in patients with post-thrombotic disease. It should also be considered in patients with non-thrombotic disease if the clinical presentation is incongruent with the duplex findings.^{19,20}

Treatment

Conservative Treatment-Compression Leg Garments - The initial treatment of CVI involves conservative measures to reduce symptoms and help prevent the development of secondary complications. Elevating the legs, exercise or walking, flexion/extension of the feet helps reduce edema and symptoms. Graded compression garments have been the mainstay of conservative treatment.²¹

Most 3rd party payers require 90 days of compression therapy prior to authorizing more definitive treatment. There is no data supporting this requirement; It has not been proven that stockings will prevent progression of venous disease. Compression hose treatment is palliative. The patient's chronic venous disease will remain and the clinical benefits are only realized while wearing the garment. Symptomatic relief is incomplete and patient compliance is very poor. The reported rates of non-compliance range from 30-65%. The stockings are hot, difficult to put on, difficult to remove, may be uncomfortable, may fall down the leg, or may cause skin irritation. The elderly, who usually present with the most advanced venous disease, are often unable to adequately utilize standard compression garments due to frailty and arthritis. It is even more difficult to obtain adequate fitting in the obese patient. It is the rare patient who will comply with compression hose for more than a few days or weeks during a hot Florida summer.

Endovenous Thermal Ablation - For many years, definitive treatment required saphenous vein stripping and high ligation. Recovery from this treatment was long and painful. Consequently, many patients who required intervention avoided surgery. The second revolution in venous disease treatment was venous ablation. Endovenous thermal ablation (EVTA) of the great and small saphenous veins was FDA approved in 1999 and has replaced routine surgical stripping. This procedure uses either a radiofrequency catheter or a laser fiber to create a full-thickness burn of the incompetent vein wall. This results in irreversible occlusion and fibrosis of the vein.

The first device approved was a catheter that creates radiofrequency (RF) energy to heat the vein wall. Patents of the procedure make Covidien (formerly VNUS, Mansfield, MA) the sole vendor of RF technology in venous ablation. Laser fibers were subsequently developed to administer thermal energy to the vein wall and there are numerous laser vendors. Successful ablation rates of the great saphenous vein have been reported between 85% and 100%. Most recent studies report greater than 95% successful occlusion with both technologies. There are no prospective randomized comparisons between RF and laser, although several retrospective analyses have demonstrated similar occlusion and complication rates. Initially, the RF technique had significantly less post-procedure pain and bruising; however, this advantage has been eliminated with newer laser wavelengths. Endoluminal laser closure has a lower disposable cost and the fibers may be sterilized for reuse. Both technologies apply heat; it becomes a matter of preference and cost.

Procedures are performed in the physician's office with local anesthetic and require no sedation. Relative contraindications for EVTA are outlined in *Table 4*. It is not necessary to discontinue therapeutic anticoagulation.²² Following a sterile preparation and drape of the extremity, intravenous access into the mid-calf saphenous vein is obtained using ultrasound guidance. The RF catheter requires a 7-French hemostatic vascular introducer. The laser only requires a 4-French catheter. The RF catheter or a laser fiber is inserted into the great or small saphenous vein and advanced under ultrasound-guidance. The device is positioned 2cm proximal to the saphenous junction.

Table 4 Relative Contraindications for EVTA

Pregnancy or nursing
Obstructed deep venous system inadequate to support venous return after EVTA
Liver dysfunction or allergy limiting local anesthetic use.
Severe uncorrectable coagulopathy or hypercoagulability syndromes
Inability to wear compression stockings secondary to inadequate arterial circulation, hypersensitivity to the compressive materials, or musculoskeletal or neurologic limitations to donning the stocking.
Inability to adequately ambulate after the procedure
Sciatic vein reflux

A mixture of 50 cc of 1% lidocaine with epinephrine in 500 cc normal saline is utilized as a tumescent anesthesia. This solution is instilled into the venous perivascular space under ultrasound guidance. The tumescent provides local anesthesia, insulates the adjacent soft tissues, and compresses the vein lumen. When activated, the RF catheter uses a standard protocol to heat the vein wall to 120 degrees Celsius for 20 seconds. The RF catheter treats the vein sequentially in 7 cm segments. Lasers treatment protocols and wavelengths vary; in general, the laser fiber is withdrawn continuously at 3-20 cm/minute depending upon the laser wavelength and wattage. The goal is to apply at least 70 J/cm. The entire procedure is performed in less than 30 minutes.

Following the procedure, the leg is wrapped with a compressive elastic bandage. The patient is fully ambulatory and may immediately return to routine activities. Elderly patients easily tolerate the minimally invasive treatment and the most difficult component of treatment is 20-30 mmHg compression hose for 1-3 weeks post-procedure.

Complications from EVTA are relatively minor.²³ There may be minor ecchymosis. Approximately one week after EVTA, the treated vein may develop a feeling of tightness and there may be transient discomfort related to inflammation in the treated vein segment. The latter is easily ameliorated with

non-steroidal anti-inflammatory drugs. Superficial phlebitis of branch varicosities may occur in 5% of patients. Irritation or injury to sensory nerves may result in paresthesia or dysesthesia. This has been reported in 0-15% of cases and is usually temporary. Skin burns have been reported but are rare and avoidable. DVT following EVTA is unusual. The reported rates vary widely; pooled data indicates a 0.3% to 0.4% incidence. A duplex ultrasound is performed 2-7 days after a procedure to exclude extension of saphenous thrombus into the deep system

Several studies have documented significant and durable improvements in QOL following EVTA.²⁴⁻²⁹ Many patients enjoy prompt symptomatic relief. In all reported series, most treatment failures occurred within the first 6 months and all have occurred within the first 12 months. Late clinical recurrence of venous insufficiency is due to progression of CVD. Treatment can only address current incompetent veins. The patient's individual risk factors will determine progression with advancing age.

Reflux in the truncal saphenous veins must be treated before addressing any visible abnormality. Clinical success after EVTA is predicated by the ability of the treating physician to identify and eliminate all incompetent pathways with adjunctive procedures. Unfortunately, many physicians do not provide comprehensive treatment and treat with only EVTA. While there may be an initial symptomatic improvement, the patient is unlikely to achieve lasting benefit and distal venous stasis will likely persist.

Sclerotherapy - Patients require adjuvant measures to eliminate incompetent tributaries.³⁰ Eradication of residual incompetent saphenous branches is important to complete the hemodynamic correction, maximize symptomatic improvement, eliminate incompetent reservoirs that could facilitate the development of new incompetent pathways, and complete cosmetic improvement of the treated limb. Elimination may also improve the success of EVTA and possibly slow the progression of venous disease in parallel venous trunks. Therefore associated varicose tributaries, reticular veins, and telangiectasias are treated with adjuvant sclerotherapy and ambulatory phlebectomy .

Sclerotherapy is the injections of a medication into a vein to irreversibly occlude the vein. This is usually done with a 1cc or 3 cc syringe and a 27 or 30-gauge needle. The preferred sclerosant agents are polidocanol, sodium tetradecyl sulfate and 50% glycerin. These agents are FDA approved, have an excellent safety profile, and have been in use for over 50 years. Some practitioners still use hypertonic saline (23.4%); however, this is painful and has higher risk of skin ulceration.

The size of the vein determines the sclerosant type and concentration. Higher concentrations (1.5-5%) of either sodium tetradecyl sulfate or polidocanol are used for obliteration of varicose veins and residual great or small saphenous vein remnants. The latter are treated with ultrasound guidance. The concentration is progressively and significantly reduced with decreasing vein size. For example, 0.5% to 0.75% of

polidocanol is used for reticular veins while a concentration of 0.25% is used for spider veins. And 50% glycerin is popular for the treatment of telangiectasias, particularly when they involve the thin skin of the ankle and foot.

Injecting the liquid agent into the vein is known as "liquid sclerotherapy." Agitating a sclerosant medication with air or physiologic gas creates a foam for "foam sclerotherapy." The foam promotes prolonged contact with the vein wall and is more effective; however, the FDA has not approved this method. Twenty-six percent of patients have a residual patent foramen ovale and there are reports of transient ischemic attacks presumably due to the embolization of foam bubbles.

Some authors have used sclerotherapy for the treatment of truncal saphenous vein insufficiency however, there is a high rate of recannulization. Sclerotherapy is the primary treatment of recurring neovascularization following surgical ligation and stripping.

A common complication (10-40%) of sclerotherapy is hyperpigmentation of the surrounding skin from hemosiderin degradation. This is more common in individuals with darker skin pigmentation and usually resolves within 6 months to one year. Inadvertent backwash into a small arteriole will result in focal skin ulceration. Inadvertent direct injection of an artery will cause acute arterial occlusion. Allergic reactions occur infrequently.³⁰

Ambulatory Phlebectomy - Ambulatory phlebectomy (AP), also known as microsurgical phlebectomy or stab avulsion phlebectomy, is a procedure by which small segments of veins are removed with minimal skin damage. This procedure is performed for incompetent tributary branches of the great or small saphenous veins, reticular veins and varicose veins. Pudendal and labial varicose veins are also appropriate for AP. As with EVTA, the procedure is performed as an outpatient in the office.^{30,31}

The procedure is performed either in conjunction with or shortly after EVTA. The veins to be treated are marked both upright and supine. Following a sterile preparation and drape, the tissue surrounding the veins is infiltrated with the tumescent. Cutaneous 1-3 mm incisions are made and the vein is snared with various vein hooks. The vein is brought to the skin surface, grasped with a clamp and progressively freed from the subcutaneous tissues until avulsed. This is repeated along the entire course of the marked veins at 1cm-5cm intervals depending upon the size, length, tortuosity, and type of vein. Long courses of veins often require greater than 20 or more individual stabs.

Absolute contraindications for AP include infectious dermatitis or cellulitis in the area to be treated, severe peripheral edema, and lymphedema. The vast majority of complications are minor. There may be skin blisters from dressings, changes in skin pigmentation, minor scarring, infections, rare allergy to the local anesthetic, post-procedure venous bleeding, and sensory nerve paresthesia.³¹

Conclusion

The prevalence of symptomatic CVD may be as high as 60-70% in the elderly population. CVI can severely impair an individual's activities of daily living and quality of life. Painful surgical stripping has been replaced by endovenous ablation, which is a minimally invasive venous procedure performed in the physician's office and allows an immediate return to daily activity. Additional treatment with adjuvant methods is extremely important to eliminate all incompetent venous disease and obtain lasting clinical benefit.

References

1. Van Korlarr I, Vossen C, Rosendaal F, Cameron L, Bovill E, Kaptein A. Quality of life in venous disease. *Thromb Haemost* 2003;90:27-35.
2. Caggiati A, Bergan JJ, Gloviczki P, Jantet G, Wendell-Smith CP, Partsch H. International Interdisciplinary Consensus Committee on Venous Anatomical Terminology. Nomenclature of the veins of the lower limbs: an international interdisciplinary consensus statement. *J Vasc Surg*. 2002;36:416-422.
3. Raju S, Neglen P. Chronic Venous Insufficiency and Varicose Veins. *N Engl J Med* 2009; 360:2319-27.
4. Eberhardt RT, Raffetto JD. Chronic Venous Insufficiency. *Circulation*. 2005;111:2398-2409.
5. Bergan JJ, Schmid-schonbein GW, Coleridge Smith PD, Nicolaides AN, Boisseau MR, Eklof B. Chronic Venous Disease. *N Engl J Med*. 2006;355:488-98
6. Neglen P, Thrasher TL, Raju S. Venous outflow obstruction: an underestimated contributor to chronic venous disease. *J Vasc Surg*. 2003;38:879-885.
7. Scott TE, LaMorte WW, Borin DR, Menzoian JO. Risk factors for chronic venous insufficiency: a dual case-control study. *J Vasc Surg*. 1995;22:622-628.
8. Jawien A. The influence of environmental factors in chronic venous insufficiency. *Angiology*. 2003;54:S19-S31.
9. Lacroix P, Aboyans V, Preux PM, Houles MB, Laskar M. Epidemiology of venous insufficiency in an occupational population. *Int Angiol*. 2003;27:172-176.
10. Labropoulos N. Hemodynamic changes according to the CEAP classification. *Phlebology* 2003;40:130-6.
11. Kistner RL, Eklof B, Masuda EM. Diagnosis of chronic venous disease of the lower extremities: the "CEAP" classification. *Mayo Proc*. 1996;71:338-45.
12. Raju S, Owen S Jr, Neglen P. Reversal of abnormal lymphoscintigraphy after placement of venous stents for correction of associated venous obstruction. *J Vasc Surg* 2001; 34:779-84.
13. Nicolaides A. Quantification of venous reflux by means of duplex scanning. *J Vasc Surg* 1990; 10:670-677.
14. Cavezzi N, Labropoulos H, Partsch S et al. 2006 Duplex Ultrasound investigation of the veins in chronic venous disease of the lower limbs--UIP consensus document. Part II. Anatomy. *European Journal Vascular and Endovascular Surg* 31:288-299.
15. Codege-Smith P, Labropoulos N, Partsch H et al. 2006 Duplex Ultrasound investigation of the veins in chronic venous disease of the lower limbs--UIP consensus document. Part I. Basic principles. *European Journal Vascular and Endovascular Surg* 31:83-92.
16. Labropoulos N, Tiongson J, Pryor L et al. 2003 Definition of venous reflux in lower extremity veins. *J Vasc Surg* 38:793-798.
17. Labropoulos N, Delis K, Nicolaides AN, Leon M, Ramaswami G, Volteas N. The role of the distribution and anatomic extent of reflux in the development of signs and symptoms in chronic venous insufficiency. *J Vasc Surg* 1996;23:504-510
18. Labropoulos N. Clinical correlation to various patterns of reflux. *J Vasc Surg* 1998;28:826-833.
19. Meissner M, Caps MT, Zierler B, et al. Determinants of chronic venous disease after acute deep vein thrombosis. *J Vasc Surg* 1998;28:826-833.
20. Raju S, Neglen P. High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: a permissive role in pathogenicity. *J Vasc Surg* 2006; 44:136-43.
21. Motykie GD, Caprini JA, Arcelus JI, Reyna JJ, Overom E, Mokhtee D. Evaluation of therapeutic compression stockings in the treatment of chronic venous insufficiency. *Dermatol Surg*. 1999;25:116-120.
22. Theivacumar NS, Gough MJ. Influence of warfarin on the success of endovenous laser ablation (EVLA) of the great saphenous vein (GSV). *Eur J Vasc Endovasc Surg* 2009;38:506-510.
23. Khilnani NM, Grassi CJ, Kundu S, Agostino HR, et al. Multi-disciplinary quality improvement guidelines for the treatment of lower extremity superficial venous insufficiency with endovenous thermal ablation from the Society of Interventional Radiology, Cardiovascular Interventional Radiological Society of Europe, American College of Phlebology and Canadian Interventional Radiology Association. *J Vasc Interv Radiol* 2010; 21:14-31.
24. Proebstle TM, Gul D, Lehr HA, et al. Infrequent early recanalization of greater saphenous vein after endovenous laser treatment. *J Vasc Surg* 2003;38:511-516.
25. Almeida JI, Raines JK. Radiofrequency ablation and laser ablation in the treatment of varicose veins. *Ann Vasc Surg* 2006;20:547-552.
26. Nicolini P, the Closure Group. Treatment of primary varicose veins by endovenous obliteration by the Closure system: results of a prospective multicenter study. *Eur J Vasc Endovasc Surg* 2005;29:433-439.
27. Ravi R, Rodriguez-Lopez JA, Trayler EA, Barrett DA, Ramaiah V, Diethrich EB. Endovenous ablation of incompetent saphenous vein: a large single-center experience. *J Endovasc Ther* 2006;13:244-248.
28. Marston WA, Owens LV, Davies S, Mendes RR, Farber MA, Eeagy BA. Endovenous saphenous ablation corrects the hemodynamic abnormality in patients with CEAP class 3-6 CVI due to superficial reflux. *Vasc Endovasc Surg* 2006; 40:125-130.
29. Min RJ, Khilnani NM, Zimmet SE. Endovenous laser treatment of saphenous vein reflux: long-term results. *J Vasc Interv Radiol* 2003;14:991-996.
30. Goldman MP, Guex JJ, Weiss RA. *Sclerotherapy Treatment of Varicose and Telangiectatic Leg Veins* 5th Edition. Elsevier/Saunders, Philadelphia, PA. 2012.
31. Kundu A, Grassi CJ, Khilnani NM, Fanelli F et al. Multi-disciplinary quality improvement guidelines for the treatment of lower extremity superficial venous insufficiency with ambulatory phlebectomy from the Society of Interventional Radiology, Cardiovascular Interventional Radiological Society of Europe, American College of Phlebology and Canadian Interventional Radiology Association. *J Vasc Interv Radiol* 2010; 21:1-13.