

Chronic Venous Insufficiency

Author: **Deron J Tessier, MD**, Staff Surgeon, Kaiser Permanente Medical Center, Fontana, CA

Deron J Tessier is a member of the following medical societies: [American College of Surgeons](#) and [American Medical Association](#)

Coauthor(s): **Russell A Williams, MBBS**, Program Director, Professor, Department of Surgery, University of California Medical Center at Irvine

Editors: **William H Pearce, MD**, Chief, Division of Vascular Surgery, Violet and Charles Baldwin Professor of Vascular Surgery, Department of Surgery, Northwestern University School of Medicine; **Francisco Talavera, PharmD, PhD**, Senior Pharmacy Editor, eMedicine; **Vincent Lopez Rowe, MD**, Assistant Professor of Surgery, Department of Surgery, Division of Vascular Surgery, University of Southern California Medical Center; **Paolo Zamboni, MD**, Professor of Surgery, Chief of Day Surgery Unit, Chair of Vascular Diseases Center, University of Ferrara, Italy; **William H Pearce, MD**, Chief, Division of Vascular Surgery, Violet and Charles Baldwin Professor of Vascular Surgery, Department of Surgery, Northwestern University School of Medicine

[Author and Editor Disclosure](#)

Synonyms and related keywords: CVI, superficial venous insufficiency, venous insufficiency, postphlebotic syndrome, postthrombotic syndrome lipodermatosclerosis, superficial venous incompetence, venous incompetence, valvular incompetence, hypercoagulability, Virchow triad, venous stasis, venous stasis ulcers, venous reflux, stasis dermatitis, stasis ulcer, venous ulcer, DVT, deep vein thrombosis, varicose veins, junctional high-pressure disease, perforator high-pressure disease, venous hypertension, varicosities, varicosity, telangiectasia, venectasia, spider vein, vein disease, venous disease, swollen veins, telangiectatic veins

Chronic venous insufficiency (CVI) is a common condition affecting 2-5% of Americans. Historically, CVI was known as postphlebotic syndrome and postthrombotic syndrome, both of which refer to the etiology of most cases. However, these names have been abandoned because they fail to recognize another common cause of the disease, the congenital absence of venous valves.

History of the Procedure

In 1914, Homans postulated that the relative hypoxia of static venous blood decreases the amount of oxygen reaching the skin, causing skin changes and ulcers characteristic of CVI.

In 1930, Landis et al demonstrated the direct relationship between venous hypertension in the legs and increased capillary intraluminal pressures.

In 1953, Piulacks et al theorized that arteriovenous fistulas in the skin of the lower extremities cause hypoxia, resulting in changes to the skin and tissues.

In 1982, Burnand et al presented the fibrin cuff hypothesis, which describes the primary problem as venous hypertension in the lower extremities causing leakage of plasma proteins, particularly fibrinogen. A fibrin cuff encircles affected capillaries, decreasing oxygen diffusion to surrounding tissues.

In 1988, Coleridge-Smith et al described the white-cell trapping theory, which hypothesizes that venous hypertension and resultant increased capillary pressures trap white blood cells in the capillaries, where they become activated and damage capillary beds. Increased capillary permeability allows seepage of plasma proteins and fibrinogen into the interstitium, where a fibrin cuff forms, thus decreasing oxygen diffusion to surrounding tissues.

Problem

In addition to poor cosmesis, CVI can lead to chronic life-threatening infections of the lower extremities. Pain, especially after ambulating, is a hallmark of the disease. CVI causes characteristic changes, called lipodermatosclerosis, to the skin of the lower extremities, which lead to eventual skin ulceration.

Frequency

CVI is a significant public health problem in the United States. Of all Americans, estimates indicate that 2-5% have some changes associated with CVI.

Approximately 24 million Americans have varicose veins. Approximately 6 million Americans have skin changes associated with CVI. Venous stasis ulcers affect approximately 500,000 people.

The mean incidence for hospital admission for CVI is 92 per 100,000 admissions.

Epidemiology:

Peak incidence occurs in women aged 40-49 years and in men aged 70-79 years.

Etiology

Congenital absence of or damage to venous valves in the superficial and communicating systems can cause CVI. Venous incompetence due to thrombi and formation of thrombi favored by the Virchow triad (venous stasis, hypercoagulability, endothelial trauma) also can cause CVI (see [Image 1](#)). Varicose veins rarely are associated with the development of CVI.

Risk factors associated with chronic venous insufficiency

- Age: Incidence of CVI rises substantially with age.
- Family history: History of deep vein thrombosis (DVT), which renders venous valves incompetent, causing backflow and increased venous pressure, is a risk factor.
- Lifestyle: A sedentary lifestyle minimizes the pump action of calf muscles on venous return, causing higher venous pressure. CVI

occurs more frequently in women who are obese. Vocations that involve standing for long periods predispose individuals to increased venous pressure in dependent lower extremities. A higher incidence of CVI is observed in men who smoke.

Pathophysiology

Two major mechanisms in the body prevent venous hypertension. First, bicuspid valves in the veins prevent backflow and venous pooling. DVTs commonly occur at these valves, causing irreversible damage to the valve. Second, during normal ambulation, calf muscles decrease venous pressures by approximately 70% in the lower extremities. With rest, pressures return to normal in approximately 30 seconds. In diseased veins, ambulation decreases venous pressures by only 20%. When ambulation is stopped, pressure in the vein lumen increases slowly, returning to normal over a period of minutes (see [Image 2](#)).

Venous hypertension in diseased veins is thought to cause CVI by the following sequence of events. Increased venous pressure transcends the venules to the capillaries, impeding flow. Low-flow states within the capillaries cause leukocyte trapping. Trapped leukocytes release proteolytic enzymes and oxygen free radicals, which damage capillary basement membranes. Plasma proteins, such as fibrinogen, leak into the surrounding tissues, forming a fibrin cuff. Interstitial fibrin and resultant edema decrease oxygen delivery to the tissues, resulting in local hypoxia. Inflammation and tissue loss result.

Clinical

Clinical manifestations include the following:

- Varicose veins: In addition to poor cosmesis, varicose veins serve as indicators of venous hypertension, the most common reason for patient complaints regarding CVI (see [Image 1](#)).
- Leg discomfort: Venous hypertension in muscles and fascial compartments of the lower leg from exercise and prolonged standing results in the characteristic ache of CVI. The discomfort is described as pain,

pressure, burning, itching, dull ache, or heaviness in affected calves or legs.

- Nonhealing ulcers: Typically, these lesions occur around the medial malleolus, where venous pressure is maximal due to the presence of large perforating veins (see [Image 2](#)).
- Leg edema: Damage done to capillary basement membranes by white blood cells results in leg edema.
- Lipodermatosclerosis: These characteristic skin changes in the lower extremities include capillary proliferation, fat necrosis, and fibrosis of skin and subcutaneous tissues. Skin becomes reddish or brown because of the deposition of hemosiderin from red blood cells (see [Image 3](#)).

INDICATIONS

Surgical treatment is reserved for those with discomfort or ulcers refractory to medical management.

Indications for vein ligation: This technique is reserved for cases of CVI that include reflux in the saphenous system causing severe symptoms. For this reason, a diagnosis of reflux must be established preoperatively, usually with photoplethysmography or duplex imaging.

RELEVANT ANATOMY

The venous network in the lower extremities commonly affected by CVI is divided into 3 systems. The first is superficial veins, which include the lesser and greater saphenous veins and their tributaries (see [Image 7](#)). The second is deep veins, which include the anterior tibial, posterior tibial, peroneal, popliteal, deep femoral, superficial femoral, and iliac veins. The third is perforating or communicating veins (see [Images 8 and 9](#)).

CONTRAINDICATIONS

In patients with symptomatic greater saphenous varicosities, the presence of an occluded deep system must be ruled out. Deep occlusion is an absolute contraindication to vein ligation. Obtaining venographic studies of the deep venous system prior to superficial vein ligation is imperative.

WORKUP

Imaging Studies

- Doppler bidirectional-flow studies and Doppler color-flow studies are used to assess venous flow, its direction, and the presence of thrombus.

Other Tests

- Photoplethysmography uses infrared light to assess capillary filling during exercise. Increased capillary filling is indicative of venous reflux and, consequently, incompetent veins.
- Outflow plethysmography involves placing and subsequently releasing a tourniquet on the lower extremity; the veins should quickly return to baseline pressures. Failure to do so indicates reflux.

Medical therapy

Nonsurgical treatments for CVI include the following:

Leg elevation

By keeping the legs elevated, venous flow is augmented by gravity, lowering venous pressures and ameliorating edema. While sitting, the legs should be above the thighs. Supine, the legs should be above the level of the heart.

Compression stockings

First described by Jobst in 1940, compression stockings produce graded pressures from the foot to the knee or thigh to decrease edema and minimize venous hypertension.

Unna boots

First described by Unna in 1854, the Unna boot now is the mainstay of treatment for people with venous ulcers. Unna boots are rolled bandages that contain a combination of calamine lotion, glycerin, zinc oxide, and gelatin.

Injection sclerotherapy

Injection of sclerosing agent directly into veins usually is reserved for telangiectatic lesions rather than CVI.

Phlebotonics have not been proven to be beneficial for CVI.

Surgical therapy

Approximately 8% of patients require surgical intervention for CVI. Surgical treatment is reserved for those with discomfort or ulcers refractory to medical management. Below are several conditions and the surgical options considered appropriate for each.

Chronic venous insufficiency resulting from superficial vein disorders

Vein ligation is the treatment of choice for superficial vein disorders. Historically, the entire greater saphenous vein system was removed; this has been replaced by the stab evulsion technique.

Several 2- to 3-mm incisions are made overlying the greater saphenous at various levels. The vein is dissected from the underlying tissues and any perforators are ligated. A small hook or blunt needle is used to extract as much of the vein as possible.

Typically, stab evulsion is limited to areas above the knee in the greater saphenous system to avoid damage to the saphenous nerve or sural nerve. This technique is reserved for CVI in which reflux in the saphenous system occurs and causes severe symptoms. For this reason, a diagnosis (usually accomplished with photoplethysmography or duplex imaging of reflux) must be established preoperatively. Hematoma, sural or saphenous nerve damage, and infection are possible complications of vein ligation.

Chronic venous insufficiency resulting from deep vein disorders

The decision to operate on a patient with venous obstruction in the deep veins should be made only after a careful assessment of symptom severity and direct measurement of both arm and foot venous

pressures. Venography alone is not sufficient because many patients with occlusive disease have extensive collateral circulation, rendering them less symptomatic. Clot lysis (eg, tissue plasminogen activator [TPA], urokinase) and thrombectomy have been tried but have largely been abandoned owing to extremely high recurrence rates.

For iliofemoral disease, the operation of choice is a saphenous vein crossover graft. In the procedure, the contralateral saphenous vein is mobilized and divided at its distal end. It then is tunneled suprapubically and anastomosed to the femoral vein on the diseased side. The result is the diversion of venous blood through the graft and into the intact contralateral venous system (see [Image 4](#)).

Because of a relatively high failure rate of 20%, ringed polytetrafluoroethylene (PTFE) grafts are used. The long-term patency is unknown.

Superficial femoral vein occlusion

Described by Warren in 1954 and Husni in 1983, the Husni bypass (as it has come to be called) is used to treat occlusion of the superficial femoral vein. The ipsilateral greater saphenous vein is harvested and used as an in situ popliteal-femoral vein bypass. This surgery is performed infrequently due to the high failure rate (approximately 40%).

Deep vein incompetence

Valvuloplasty is reserved for patients with a congenital absence of functional valves. A venotomy is performed, and the valve cusps are plicated. To ensure an adequate result, plicating 20-25% of each cusp is recommended. The addition of a PTFE sleeve around the operative site is used routinely to maintain valve integrity. When combined with the ligation of perforating veins, valvuloplasty has a superior outcome in 80% of cases after 5 years.

With vein segment transposition, a vein with normal function in close proximity to the diseased vessel is identified. The incompetent vein then is dissected, mobilized, and transposed on to the normal vein distal to a functional valve.

With vein valve transplantation, a valve-containing segment of a competent axillary or brachial vein is mobilized and inserted into either the popliteal or the femoral systems. The incompetent segment of the

leg vein is excised and replaced with the transplant segment. Allograft or cadaveric vein transplants are being tested, with long-term results pending.

Preoperative details

Both invasive and noninvasive studies are conducted.

Invasive studies

Contrast venography is the criterion standard for assessing venous reflux, vein abnormalities, and the presence of valves. Ambulatory venous pressure is measured by placing a catheter in a vein on the dorsum of the foot during exercise.

Noninvasive studies

Commonly, both Doppler bidirectional-flow studies and Doppler color-flow studies are used to assess venous flow, its direction, and the presence of thrombus.

Photoplethysmography uses infrared light to assess capillary filling during exercise. Increased capillary filling is indicative of venous reflux and, consequently, incompetent veins.

Outflow plethysmography involves placing and subsequently releasing a tourniquet on the lower extremity; the veins should quickly return to baseline pressures, and failure to do so indicates reflux.

Intraoperative details

Careful monitoring of a patient's cardiac status and vital signs is extremely important. In addition, periodic monitoring of hemoglobin and hematocrit levels yields essential intraoperative data.

Postoperative details

Anticoagulation with heparin (or low molecular weight heparin) in the immediate postoperative period and long-term prophylaxis with Coumadin are recommended.

Follow-up

Patients should be observed frequently for wound infection after discharge, beginning 1 week

postoperatively. Sutures or staples typically stay in 2-4 weeks, depending on the health of the skin at the operative site.

For excellent patient education resources, visit eMedicine's [Circulatory Problems Center](#). Also, see eMedicine's patient education article [Varicose Veins](#).

OUTCOME AND PROGNOSIS

Hematoma, sural or saphenous nerve damage, and infection are possible complications of lower-extremity vein ligation.

Clot lysis (eg, TPA, urokinase) and thrombectomy have been tried but generally have been abandoned due to extremely high recurrence rates.

For iliofemoral disease, the operation of choice is a saphenous vein crossover graft. Due to a relatively high failure rate of 20%, ringed PTFE grafts are being used. The long-term patency is unknown.

The Husni bypass for superficial femoral vein occlusion is performed infrequently due to the high failure rate (approximately 40%).

Surgery for CVI resulting from deep vein incompetence includes valvuloplasty and allograft or cadaveric vein transplant. Valvuloplasty for patients with congenital absence of functional valves, when combined with the ligation of perforating veins, has a superior outcome in 80% of cases after 5 years. Allograft or cadaveric vein transplants are being tested, with long-term results pending.

Tsai et al examined the National Inpatient Sample from 1988-2000 and found that mean hospital charges were \$13,900 and did not change over the time period examined. They also found that deep venous thrombosis affected 1.3% of patients and amputation was necessary in 1.2%, with an overall mortality of 1.6%.

FUTURE AND CONTROVERSIES

Subfascial endoscopic perforator surgery (SEPS) is gaining in popularity as a means of treating CVI. Endoscopic techniques are used to find and ligate perforating veins. Preliminary reports are

encouraging. The 1997 North American Subfascial Endoscopic Perforator Surgery Registry showed that after SEPS, the average healing time for ulcers was 42 days, with a recurrence rate of 3%. Ulcers treated with SEPS heal 4 times faster than ulcers treated conventionally. In addition, morbidity of SEPS is significantly lower than traditional operations. Long-term results are pending.

ACKNOWLEDGMENTS

The authors and editors of eMedicine gratefully acknowledge the contributions of previous author Yale D Podnos, MD, MPH to the development and writing of this article.

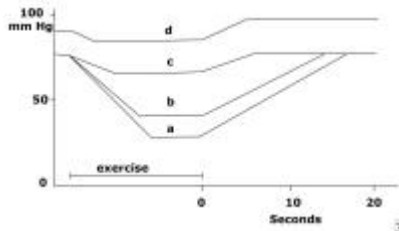
MULTIMEDIA

Media file 1: **Picture of venous valve: Thrombosis can begin as blood flow becomes turbulent, permitting platelets to remain in the valve sinus. This forms the nidus of a thrombus.**



 [View Full Size Image](#)

Media file 2: **Hemodynamic charting of (a) healthy patients, (b) patients with only varicose veins, (c) patients with incompetent perforator veins, and (d) patients with deep and perforator incompetence.**



 [View Full Size Image](#)

Media file 3: **Perforator vein bulging into subcutaneous tissue.**



 [View Full Size Image](#)

Media file 4: **Chronic venous stasis ulcer.**



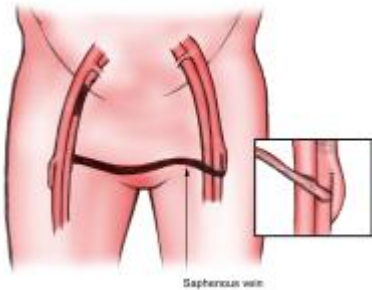
 [View Full Size Image](#)

Media file 5: **Venous stasis ulcer and surrounding dystrophic tissue.**



 [View Full Size Image](#)

Media file 6: **Venous insufficiency iliofemoral obstruction (Palma operation). Saphenous vein from contralateral leg tunneled subcutaneously to the femoral vein of the affected limb. Cumulative patency of 75% at 5 years. Relieves venous claudication but may not heal ulcers or relieve swelling.**



 [View Full Size Image](#)

Media file 7: **Lower leg venous anatomy.**



 [View Full Size Image](#)

Media file 8: **Perforating veins of the lower leg.**



[View Full Size Image](#)

Media file 9: **Venogram demonstrating incompetent perforating veins.**



[View Full Size Image](#)

AUTHOR AND EDITOR INFORMATION

Section 1 of 12

- [Authors and Editors](#)
- [Introduction](#)
- [Indications](#)
- [Relevant Anatomy](#)
- [Contraindications](#)
- [Workup](#)
- [Treatment](#)
- [Outcome and Prognosis](#)
- [Future and Controversies](#)
- [Acknowledgments](#)
- [Multimedia](#)
- [References](#)

REFERENCES

- Brand FN, Dannenberg AL, Abbott RD. The epidemiology of varicose veins: the Framingham Study. *Am J Prev Med*. Mar-Apr 1988;4(2):96-101. [\[Medline\]](#).
- Burnand KG, Whimster I, Naidoo A. Pericapillary fibrin in the ulcer-bearing skin of the leg: the cause of lipodermatosclerosis and venous ulceration. *Br Med J (Clin Res Ed)*. Oct 16 1982;285(6348):1071-2. [\[Medline\]](#).
- Coleridge Smith PD, Thomas P, Scurr JH. Causes of venous ulceration: a new hypothesis. *Br Med J (Clin Res Ed)*. Jun 18 1988;296(6638):1726-7. [\[Medline\]](#).
- Elder DM, Greer KE. Venous disease: how to heal and prevent chronic leg ulcers. *Geriatrics*. Aug 1995;50(8):30-6. [\[Medline\]](#).
- Gloviczki P, Bergan JJ, Menawat SS. Safety, feasibility, and early efficacy of subfascial endoscopic perforator surgery: a preliminary report from the North American registry. *J Vasc Surg*. Jan 1997;25(1):94-105. [\[Medline\]](#).
- Homans J. The etiology and treatment of varicose ulcers of the leg. *Surg Gynecol Obstet*. 1917;24:300-11.
- Husni EA. Reconstruction of veins: the need for objectivity. *J Cardiovasc Surg (Torino)*. Sep-Oct 1983;24(5):525-8. [\[Medline\]](#).
- Ibrahim S, MacPherson DR, Goldhaber SZ. Chronic venous insufficiency: mechanisms and management. *Am Heart J*. Oct 1996;132(4):856-60. [\[Medline\]](#).
- Martinez MJ, Bonfill X, Moreno RM. Phlebtonic for venous insufficiency. *Cochrane database of Systematic Reviews*. 2005;3.
- Neglen P, Raju S. A comparison between descending phlebography and duplex Doppler investigation in the evaluation of reflux in chronic venous insufficiency: a challenge to phlebography as the "gold standard". *J Vasc Surg*. Nov 1992;16(5):687-93. [\[Medline\]](#).
- Nyhus LIM, Barker RB, eds. *Mastery of Surgery*. 2nd ed. Boston, Mass: Little Brown; 1992.: 2133-9.
- Piulacks P. Pathogenic study of varicose veins. *Angiology*. 1953;4:59-100.
- Puggioni A, Kalra M, Gloviczki P. Superficial vein surgery and SEPS for chronic venous insufficiency. *Seminars Vasc Surg*. 2005;18:41-8.
- Tsai S, Dubovoy A, Wainess R. Severe chronic venous insufficiency: magnitude of the problem and consequences. *Ann Vasc Surg*. 2005;19:705-11.
- Weiss VJ, Surowiec SM, Lumsden AB. Surgical management of chronic venous insufficiency. *Ann Vasc Surg*. Sep 1998;12(5):504-8. [\[Medline\]](#).
- Wilson SE, Veith FJ, Williams RA. Chronic Venous Insufficiency. *Vascular Surgery: Principles and Practice*. 1978;723-61.

[Chronic Venous Insufficiency excerpt](#)

Article Last Updated: Oct 5, 2006