

Chronic Venous Insufficiency



CONTEMPORARY ENDOVASCULAR MANAGEMENT

Edited by Mark G. Davies and Alan B. Lumsden

The Contemporary Endovascular Management series is designed to be a focused, relevant, and timely review of the modern aspects of imaging and intervention in specific vascular beds. Each volume in the series addresses one vascular bed and equips readers with the current information necessary for vascular practice using a clear and easy-to-access format.

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Forthcoming

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Forthcoming

Chronic Venous Insufficiency

CONTEMPORARY ENDOVASCULAR MANAGEMENT

Volume 1

editors

Mark G. Davies MD, PhD, MBA

Vice Chairman, Finance and Administration, Department of Cardiovascular Surgery; Program Director, Vascular Surgery Fellowship and Integrated Vascular Surgery Residency; Director of Research and Education, Methodist DeBakey Heart & Vascular Center, The Methodist Hospital, Houston, Texas; Senior Investigator, The Methodist Hospital Research Institute; Professor of Cardiovascular Surgery, Weill Cornell Medical College, New York, New York

Alan B. Lumsden MD

Chairman, Department of Cardiovascular Surgery; Medical Director, Methodist DeBakey Heart & Vascular Center, The Methodist Hospital, Houston, Texas; Professor of Cardiovascular Surgery, Weill Cornell Medical College, New York, New York

Daynene Vykoukal PhD **assistant editor**

Scientific Editor, The Methodist Hospital Research Institute, Methodist DeBakey Heart & Vascular Center, The Methodist Hospital, Houston, Texas



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Introduction

Mark G. Davies

Recognition of venous disorders as a significant public health issue has increased awareness of venous problems among both the medical professions and the lay population. This awareness has resulted in greater attention to prevention, introduction of quality performance measures, and enhanced operative and pharmacological interventions, which have transformed the treatment of venous disease from a stepchild of several specialties into a robust subspecialty field in medicine. Venous problems may be divided into two categories, acute venous disorders and chronic venous disorders. This volume in the Contemporary Endovascular Management series provides a focused and timely review of the current state of the field in chronic venous disorders.

Chronic venous disease continues to be a common medical problem in Western Europe and the United States.¹ Reported prevalence ranges from 1% to 40% in females and from 1% to 17% in males. Esti-

mates for varicose veins are higher, ranging from 1% to 73% in females and 2% to 56% in males. While many risk factors have been associated with chronic venous disease, the ones most commonly quoted are older age, female gender, pregnancy, family history of venous disease, obesity, and occupations associated with orthostasis. Chronic venous disease can be divided into primary and secondary disorders.

Primary chronic venous disorders, or those not associated with an identifiable mechanism of venous dysfunction, are among the most common venous problems.² Varicose veins without skin changes are present in about 20% of the population, while active ulcers may be present in as many as 0.5%. These problems arise from intrinsic structural and biochemical abnormalities of the vein wall that with progression lead to skin changes and ulceration. This progression arises from extravasation of macromolecules and red blood cells,

leading to endothelial cell activation, leukocyte diapedesis, and altered tissue remodeling with intense collagen deposition. Patients may remain asymptomatic, or they may develop pain or swelling, skin changes, or a complication (thrombophlebitis, bleeding, ulceration). While physical exam allows for the identification of varicosities and saphenofemoral incompetence, venous duplex ultrasonography is the most common study used to define the function of the superficial and venous systems in the lower extremities. Primary venous disease is most often associated with truncal saphenous insufficiency. Until recently, these veins were treated with stripping of the saphenous vein and interruption and removal of major tributary and perforating veins. Currently, endovenous ablation techniques with radiofrequency, laser, or foam sclerotherapy are the norm. Sclerotherapy and cutaneous laser treatment are used in the management of telangiectasias; primary, residual, or recurrent varicosities without connection to incompetent venous trunks; and congenital venous malformations.

Secondary chronic venous disorders are those venous problems that occur following an episode of acute deep venous thrombosis.³ Most occluded venous segments recanalize over the first 6 to 12 months after an episode of acute deep venous thrombosis, leading to chronic luminal changes and a combination of partial obstruction and reflux. These intrinsic changes lead to venous hypertension. The clinical manifestations of secondary chronic venous disease, including pain, venous claudication, edema, skin changes, and ulceration, are commonly referred to

as postthrombotic syndrome. The diagnostic evaluation of secondary chronic venous disorders is similar to primary chronic venous disorders, although greater use of imaging and intervention to correct structural changes in veins is more common. In general, however, compression remains the primary treatment of chronic venous disorders and is essential for healing ulcers and minimizing recurrence. The efficacy of various adjuncts to ulcer treatment, including complex wound dressings and medications, have been variable. Although superficial venous surgery has not been demonstrated to improve ulcer-healing rates, it does decrease ulcer recurrence. Treatment of incompetent perforating veins remains controversial and appears to be appropriate for venous ulceration. With respect to venous obstruction, ilio caval angioplasty and stenting has emerged as the primary treatment for proximal iliofemoral venous obstruction, with surgical bypass assuming a secondary role.

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Varicose Veins

A lack of rigor exists in the degree to which interventions for venous disease have been scientifically evaluated and evidence-based guidelines developed and followed. The professional societies have begun to tackle this problem, and a growing body of scientific investigation and practical classification systems now exist for developing best-practice protocols for the treatment of chronic venous insufficiency. With the recent expansion in venous therapies, evidence-based medicine will be necessary to allow appropriate delivery of care with a shrinking healthcare budget and resources. The rapid evolution and expansion of treatment in varicose veins has changed the field from an open inpatient procedure to a percutaneous therapy suitable for the office environment. This change in practice has been driven by the widespread introduction and competency in duplex ultrasonography and by a better understanding of the pathology of varicose veins. While duplex scanning has been effective in the extremities, the development of CTV and MRV has increased our understanding of both peripheral and central venous diseases and

their association with venous incompetency. Both radiofrequency and laser-based systems provide equivalent results, and it appears that operator experience and patient selection are key to success. Sclerotherapy is evolving as an alternative to many of the catheter-based techniques but is still reserved for nonaxial varicose veins in the majority of practices. There is a remarkably low rate of true adverse reactions following all percutaneous therapies. However, many venous studies continue to suffer from short-term follow-up and small sample size, as well as an observational nature. These limitations tend to decrease the validity of assessments of outcomes for varicose vein recurrence and are the basis of the determinations of most insurance carriers. The evolution in the understanding of and therapy for venous perforator has had a fluctuating course. Open perforator surgery has evolved to SEPS and now to RFA or sclerotherapy. While restricted to therapies for advanced CVI, its widespread adoption is relatively low due to the conflicting reports and a general lack of evidence-based support to validate its use.

The understanding of and ability to image venous incompetence and the efforts to link these to clinical disease have advanced the field. Coupled to the introduction of less invasive therapies, the field is emerging as a patient-specific and disease-oriented intervention. An increase in evidence-based data is required to consolidate these initial positive advances.

Endovenous Radiofrequency Ablation of the Saphenous Vein

Mechanism of Action, Procedural Technique, and Review of Clinical Outcomes

Elizabeth Acquista and Alan M. Dietzek

Venous reflux produces hemodynamic changes including venous hypertension in the lower limb. This in turn can cause surface veins to distend and press against cutaneous sensory nerves, causing significant pain and discomfort for patients. Compression therapy, although effective at reducing short-term pain and discomfort, does not provide a cure for chronic venous reflux. Thus, the fundamental element of successful treatment of symptomatic superficial venous insufficiency in the lower extremities is to eliminate the sources of venous reflux. Treatments for chronic venous insufficiency are documented in historic, surgical literature dated from 450 BC in the writings of Hippocrates through Galen, Cel-

sus, and de Chauliac and later recorded by Harvey and Weisman, who all advocated for compression of the lower extremity to cure ulcers. The sources of reflux most commonly emanate from the great and small saphenous veins, and it was Trendelenbourg who first published on the ligation of the great saphenous vein (GSV). Later, Moore developed the contemporary procedure for GSV ligation just distal to the saphenofemoral junction (SFJ). Most recently, these veins, when incompetent, were entirely removed from the venous circulation primarily with surgical stripping. Presently, endovenous radiofrequency ablation (RFA), also referred to as the closure procedure, a catheter-based endovascular intervention, offers a more effective, less morbid alternative for the treatment of superficial venous reflux. The indications for intervention and the imaging to document reflux have been illustrated in previous chapters.

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Radiofrequency Vein Ablation Procedure

Although surgical removal of the saphenous vein is an effective, time-honored surgical procedure, it causes postoperative morbidity and carries a significant psychological burden to the patient. Endovenous RFA was first introduced into clinical practice in Europe in 1998 and in the United States little more than a year later. The objective of the less invasive endovenous radiofrequency vein ablation is to remove the refluxing saphenous vein from the circulation in a manner that minimizes patient discomfort, decreases postoperative morbidity, provides a good long-term outcome, and allows rapid return to normal activities. Since elimination of the refluxing saphenous vein from the circulation is the only goal of the procedure, judgment of RFA success can be assessed by duplex ultrasonography at any time postoperatively. Relief of patient symptoms, time to resume normal activities, postprocedure adverse events, and improvement in health-related quality of life are additional relevant measures of the procedure outcome, and these are linked to successful elimination of saphenous vein reflux.

The RFA procedure does not require a general anesthetic and can be performed using local tumescent anesthesia with or without conscious sedation. In the United States, it is usually performed in a physician's office or minor procedure room, although some surgeons prefer to perform the procedure on an outpatient basis in a day surgery center. While there are anecdotal reports of saphenous vein stripping being performed in an office setting, it is most commonly performed with general or regional anesthesia in a more formal operating room environment. The technical methodology of the

endovenous RFA procedure is described in numerous publications and will be further elaborated upon here.^{1,2}

The Closure system (VNUS Medical Technologies, San Jose, CA) consists of a radiofrequency (RF) generator and disposable catheters. The first-generation catheter (Closure PLUS) has collapsible bipolar electrodes (Figure 5.1). When deployed, the electrodes make direct contact with the luminal surface of the vein wall. RF energy is delivered through the electrodes into the vein, and the vein wall tissue resistance converts RF energy into thermal energy. With the RF energy activated, the catheter is slowly withdrawn to treat the desired length of vein. A thermocouple located on the electrodes monitors the temperature and provides feedback to the generator, which, in turn, controls the power delivery and the treatment temperature; the latter is set at either 85°C or 90°C. The thermocouple



FIGURE 5.1 VNUS Closure PLUS catheter electrodes. VNUS product image copyright ©Covidien. Used with permission.

Chronic Venous Reconstructions

In contrast to superficial venous incompetence, the understanding of and interventions for chronic venous occlusive disease have progressed at a slower pace with a lower penetration of new technologies. Many ideas and therapies have been borrowed from the equivalent obstructive scenarios in arterial disease, although the pathophysiology is markedly different since obstruction alone causes symptoms in about one-third of postthrombotic limbs, while the remainder have a combination of reflux and obstruction. This transfer of techniques and knowledge may have underserved the development of therapy in the field. The increasing use of CTV/MRV and the resurgence of venography have increased the awareness of iliofemoral obstruction in patients with chronic venous disease. Introduction of intravascular ultrasonography has further enhanced the three-dimensional understanding of occlusive and nonocclusive iliac venous obstructive disease. The adoption of both recannulization and stenting techniques is altering perceptions of therapy in these conditions. May-Thurner syndrome is now considered a treatable condition.

The increasing use of endoluminal techniques has decreased the utilization of venous bypasses, but these techniques remain a viable alternative in the appropriately selected patient. Expansion of the evidence from a few select centers and development of better evidence-based protocols are necessary for this field to advance further. However, while these modalities can change the outlook for occlusive disease and lessen the clinical impact of the combination of reflux and obstruction, treatment of deep venous reflux will require both refinements in autologous valve transfers and the development of a viable artificial venous valve. This is the next frontier in chronic venous disease.

Stenting of Chronic Obstruction

Indications, Techniques, and Outcomes

Peter Neglén

Chronic venous disease (CVD) is still among many physicians equated with venous valve incompetence, and diagnosis and therapy have been focused on venous reflux. The main reason for this is poor understanding of the importance of venous outflow obstruction in the pathophysiology of primary and secondary chronic venous disease. The lack of accurate tests and the sole treatment being invasive surgery also explain the lack of previous enthusiasm. However, it has been well known for some time that obstruction alone causes the symptoms in more than one-third of post-thrombotic limbs, that the most symptomatic limbs have a combination of reflux and obstruction,^{1,2} and that persistent obstruction of proximal veins is associated with progres-

sive distal vein incompetence.³ It has also been pointed out that the obstruction of the iliofemoral vein, that is, the final pathway for the venous outflow above the confluence of the profunda and femoral veins, more frequently results in leg complaints, especially venous claudication and leg ulcers. Studies have also shown that cumulative incidence of the postthrombotic syndrome is more common than previously realized (all symptoms 27% and ulcer rate 4% at 20 years following acute deep vein thrombosis [DVT]).⁴ Even without thrombosis, the existence of iliac vein compressions with or without intraluminal lesions are in themselves more pathogenic than previously thought, although they have been considered a common finding of little clinical importance.

Percutaneous endovenous stenting was introduced more than a decade ago and has emerged as the method of choice for the treatment of chronic obstruction of the femoro-ilio-caval venous outflow. It has replaced

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bypass surgery as the primary treatment. Stenting is a relatively simple and effective endovenous treatment with low morbidity and mortality. Venous stenting has been slow to be widely adopted despite these advantages. The key is to increase awareness among treating physicians of the possible presence of iliofemoral obstruction in patients with CVD, and, consequently, the need for more extensive evaluation of the pelvic venous outflow and less reliance on infrainguinal duplex Doppler investigation alone. Patients complaining of significant symptoms of chronic venous insufficiency should make the physician think obstruction rather than reflux.

Chronic Postthrombotic Obstruction

Poor recanalization following acute deep vein thrombosis is presently thought to be the most common cause of chronic venous blockage.¹ Most symptomatic outflow obstruction occurs following deep vein thrombosis involving the iliac segment. It may be limited to the iliofemoral segment or contiguous from the calf to the iliac veins. Only approximately 20% of these iliac veins will completely recanalize on anticoagulation treatment, while the remaining veins recanalize only partially and develop varying degrees of obstruction and collateral formation.^{5,6} In the 1960s, Cockett and his associates performed groundbreaking studies and published numerous “classic” reports on venous outflow obstruction, compression of the iliac veins, and its relationship to thrombosis.⁷⁻¹¹ They observed that the obstructive lesion that precipitated the thrombosis impeded its resolution, and the postthrombotic perivenous fibrosis appeared to develop excessively at the site of the initiating lesion.

Recently, this observation has been confirmed by serial spiral computed tomography venography (CTV) studies, which showed inhibited and incomplete recanalization in the presence of an external compression, eg, left iliac vein compression.¹² This finding is of great importance, as it has been reported that 80% of limbs with iliofemoral DVT have underlying extrinsic iliac compression-type lesions revealed by the same technique (67% and 84%, right and left iliofemoral veins, respectively).¹³

The typical postthrombotic iliofemoral lesion often involves both common and external iliac veins with irregular stenosis or occlusions and axial, transpelvic, and ascending lumbar collaterals are present (Figure 10.1). Infrequently, a diffusely narrowed long segment of the iliac vein with no collateral formation is found. We have designated this entity a “Rokitansky” stenosis, from the 19th-century pathologist who described the phenomenon. As the severe inflammation of the wall (phlebitis) subsides, a fibrotic cylinder is formed, which impedes any collateral development and expansion of the vein. Thus, significant outflow obstruction cannot be excluded because of lack of collaterals.

Nonthrombotic Iliac Vein Lesion

Even without previous thrombosis, studies have shown frequent findings of intraluminal lesions and varying degrees of external compression of the iliac vein in the general population (22%–33%¹⁴⁻¹⁶ and 66%–88%,^{9,11,17} respectively). They have often been considered a common finding of little clinical importance. Symptomatic nonthrombotic iliac vein lesions (NIVL) have previously been described as May-Thurner syndrome¹⁴ or Cockett’s or “iliac vein com-

Special Circumstances

The conditions of Nutcracker syndrome and pelvic congestion syndrome were relative rarities until the widespread introduction of cross-sectional imaging. In the case of Nutcracker syndrome surgical intervention is the norm; however, the few reports on endovascular therapy raise hopes for a percutaneous option in this condition. In the arena of chronic pelvic pain, pelvic congestion syndrome is treatable endoluminally and is anatomically successful in most cases. Coils currently are the treatment of choice, but whether sclerosants or glue will be of use is unknown. Both syndromes are now more commonly reported; however, reports on therapy remain observational and further work to develop evidence-based approaches is warranted.

Nutcracker Syndrome

Joseph J. Naoum

Vascular compression of the left renal vein (LRV) between the superior mesenteric artery (SMA) and the aorta is an uncommon condition. Diagnosis requires a high degree of suspicion. Helical computed tomography (CT) scans, with the administration of intravenous contrast, and 3-dimensional reconstruction have proven to be a powerful diagnostic tool. Left renal vein venography with pressure gradient measurements is important to confirm the diagnosis. The presence of severe or classic symptoms requires operation. The operative treatment of choice consists of transposition of the LRV; however, left renal autotransplantation has been used with success.

History

Obstruction of the LRV by the SMA against the aorta was first identified and described in 1944 in autopsy studies.¹ In 1950, El-Sadr and Mina² described the first patient with this pathology. However, it was not until 1971 that Chait and colleagues³ reported the use of venography to demonstrate the vascular impression on the compressed LRV and described the aorta and the SMA as 2 arms of a nutcracker. It appears that in 1972, de Schepper⁴ identified the “Nutcracker” compression of the LRV as a syndrome and noted that it resulted in the extensive development of perirenal varicosities. Numerous alternative names to describe this syndrome include Nutcracker syndrome, Nutcracker phenomenon, renal vein entrapment syndrome, vascular compression of the LRV, aortomesenteric compression of the LRV, and mesoaortic compression of the LRV. The latter term seems to best and most accurately describe this disease.

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Anatomy

Vascular compression of the LRV between the SMA and the aorta is a real disease with a specific anatomic basis (Figure 15.1). In humans, the SMA arises from the abdominal aorta at the level of the first lumbar vertebra at an angle that averages 42.4° , with a wide range of variation from 18° to 70° . Normally, the LRV crosses the abdomen over the aorta at the level of the second lumbar vertebra to join the inferior vena cava (IVC). Thus, the LRV is fixed at 2 points by the left kidney and the IVC.

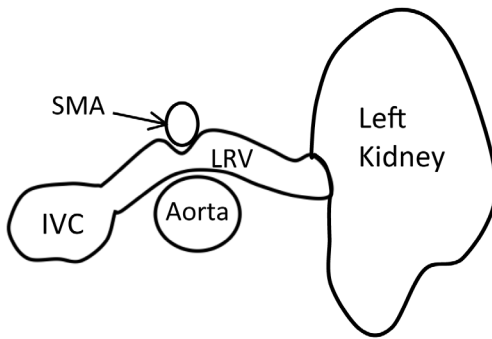


FIGURE 15.1 Axial schematic demonstrating the LRV compressed by the SMA against the aorta.

Minor alterations of the normal anatomic relationships of the SMA, the aorta, the LRV, and the left kidney may lead to this syndrome. A narrow aortomesenteric angle produces extrinsic compression of the LRV by the SMA. Using ultrasonography, Arima and colleagues⁵ demonstrated symptomatic compression of the LRV when the angle was less than 6° to 16° compared with 38° to 56° in controls. On the other hand, Ali-El-Dein and associates⁶ used magnetic resonance angiography (MRA) to show that symptomatic patients in their series had an average

aortomesenteric angle of 54° as compared to 91° in healthy controls. A low origin of the SMA or a high LRV position can also lead to compression. An extensive loss or decrease of retroperitoneal fat pad can contribute to a decrease in the aortomesenteric distance and angle responsible for the development of vascular compression of the LRV. Posterior renal ptosis with concomitant stretching of the left renal vein over the aorta may also be a contributing cause.⁷

Clinical Presentation

Mesoaortic compression of the LRV is an uncommon condition. It can affect children, especially with low body mass index,⁸ it is more common in young adults between the ages of 20 and 40 years than in older individuals,⁹ and it affects women more often than men.

Symptoms are nonspecific and may include abdominal or left flank pain, pelvic pain of variable intensity that can be aggravated by prolonged standing, and fatigue. Compression of the LRV leads to venous hypertension, which can cause perirenal varicosities and incompetence of the left ovarian or testicular vein. Therefore, men can present with left-sided varicocele while women can have symptoms of pelvic congestion and labial varicosities. It is believed that LRV hypertension may lead to rupture of the thin-walled septum separating the veins and the urinary collecting system and cause unilateral micro- or macroscopic hematuria and proteinuria.^{6,10}

Diagnosis

Confirmation of the diagnosis requires a high degree of suspicion and meticulous evaluation. The usual causes for abdominal pain and hematuria need to be considered

Venous Malformations

There is a growing realization of the impact that venous malformations have on patients and the complexity of their treatment. Cross-sectional imaging has markedly improved the ability to diagnose and classify these lesions. Considerations are different from interventions for arterial malformations. Venous malformations are low-flow lesions and often truncular in form, with involvement of the lower extremity, and treatment is aimed at controlling the signs and symptoms of chronic venous hypertension. Multimodality imaging and a multidisciplinary management strategy are the norm. Embolization is technically more difficult since the embolization materials are prone to enter the systemic circulation in many variants of venous malformation, and surgery is only reserved for focal lesions and limb salvage situations. Conservative therapy is more common given the anatomic patterns, the relatively benign pathological course of these lesions, and the technical challenges of intervention.

Arteriovenous Malformations

Alan B. Lumsden

Arteriovenous malformations (AVMs) are congenital lesions, which consist of a complex of blood vessels and abnormal arteriovenous communications. Although not hereditary, they represent inborn defects in vascular morphogenesis and consist of feeding arteries and draining veins, which communicate through a nidus. The nidus represents a direct communication between the arteries and veins with absence of intervening capillaries. Blood shunts directly through the nidus from the higher-pressure arterial system to the lower-pressure venous system.

AVMs may be mixed (arteriovenous) or predominantly venous (65% of total) in composition, and determination of the major component is an important guide in choice

of therapy. Occurring in 1.5% of the population, they are usually isolated but occasionally are associated with identifiable syndromes such as Klippel-Trenaunay or Parkes-Weber syndromes.

The natural history of AVMs is unpredictable. Growth can be stimulated by trauma or hormone exposure, or may become more progressive for no obvious reason. AVMs may be multifocal. Progressive growth requiring multiple interventions is necessary.

Classification of congenital Malformations is confusing because of the multitude of terms. The Hamburg classification of vascular malformations uses criteria that take into account the underlying anatomical, histological, pathophysiological, and hemodynamic status of each malformation (Table 18.1). Congenital vascular malformations (CVMs) include hemangiomas and arteriovenous malformations. The latter behave more aggressively, pursuing a more destruc-

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