SCIENTIFIC ARTICLES

Stents for Chronic Venous Insufficiency: Why, Where, How and When — A Review

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Venous stents deployed in the iliac-femoral veins have introduced an entirely new paradigm of treatment for a wide spectrum of patients with severe chronic venous insufficiency. The procedure is percutaneous, performed in an outpatient setting, is safe and efficacious and does not preclude traditional open operations in the event of stent failure. Therefore, venous stents are an attractive first and often definitive choice of treatment in this disease.

TYPES OF CHRONIC VENOUS INSUFFICIENCY AMENABLE TO STENTING

The etiology of chronic venous insufficiency is for the most part roughly divided equally between postthrombotic and 'primary' (idiopathic)[1] causes. The postthrombotic variety can manifest at variable times after initial DVT, sometimes decades later, when the patient may have even forgotten the original event. Some 30% of DVT events are estimated to be silent, thus absence of a positive history does not rule out the condition. Recurrent DVT often results in postthrombotic syndrome.[2] The 'primary' variety is indistinguishable from the postthrombotic variety in clinical features and presentation. Both types are selectively amenable to stent treatment.

STENTING IN COMBINED OBSTRUCTION/REFLUX

Hemodynamically, both postthrombotic and 'primary' varieties present with a combination of obstruction and reflux.[3] In the former, the obstructive component arises from postthrombotic strictures and occlusions of major venous pathways; the reflux arises from postthrombotic destruction of involved valve stations and late onset of reflux at valve sites spared from destruction.[4] The mechanism of such late onset of reflux is not known. In the 'primary' variety reflux arises from progressive redundancy of valve cusps, thought to be due to a degenerative process. The obstructive component in 'primary' venous insufficiency occurs at the iliac vein level at multiple naturally-occurring compression sites.[5] Such compressive lesions underlie arterial crossover points (iliac or hypogastric artery) and often develop internal webs or membranes generally thought to be the result of repeated pulsatile trauma, though there is some controversy as to the exact mechanism.[6] Large autopsy series[7-9] and modern imaging techniques[10] have established that such compressions and webs are present in 30-65% of the general population in silent form. The acronym NIVL (for 'non-thrombotic iliac vein lesion') is preferred, as it encompasses both compressive and intrinsic 'primary' lesions. Such lesions are thought to play a permissive role (similar to PFO and stroke, obesity and diabetes, or ureteral reflux and pyelonephritis) favoring the development of chronic venous insufficiency and resulting in eventual symptoms in perhaps 3-4% of the population. In any event these lesions are present in >90% of 'primary' patients with severe symptoms of venous insufficiency.[5] Venous stenting often remits symptoms in this class of patients.[11] A remarkable observation borne out of venous stent experience is the fact that venous stenting alone relieves symptoms in the majority of both postthrombotic and 'primary' patients *even when the associated reflux component is left untreated.*[12, 13] This core observation has allowed the application of the minimal-

ly invasive percutaneous stent technology to both classes of symptomatic patients as the initial and often the definitive choice of treatment. Correction of deep reflux that requires intricate open surgery[14-16] can be deferred and is required in only a minority of patients who do not adequately respond to the initial stent treatment.

Anatomy

In anatomical terms, venous outflow obstruction is confined to the iliac vein segment in 'primary' disease. In postthrombotic disease, morphologic stenoses and occlusions can be present in the crural, femoral and iliac veins or the inferior vena cava. All except the iliac vein are well collateralized owing to embryologic reasons[17, 18] and therefore do not generally pose symptoms even when the segment(s) are totally occluded. Iliac vein lesions are the basis of symptoms in most such patients and are therefore the primary target for stenting.[19-21]

Anatomically, venous reflux, whether 'primary' or postthrombotic, may involve the saphenous veins (superficial system), the perforators and/or the deep valves, particularly at the popliteal and femoral valves. In highly symptomatic patients, all three systems are frequently involved[22, 23] and the reflux may be straight line ("axial") from groin to the ankle. Valves above the inguinal ligament are rare and are not functionally significant when present. When necessary, partial correction of reflux (one system or one deep valve) is enough to relieve symptoms.[24-27] With the advent of percutaneous laser and radiofrequency techniques, saphenous ablation to abolish saphenous reflux has become simple and can be combined with venous stenting in one session.[28] Despite traditional teaching to the contrary, saphenous ablation is now known to be safe even in the presence of postthrombotic deep venous obstruction.[29] Perforator reflux is often secondary to deep reflux[27] and its correction is controversial[30] even though minimally invasive endoscopic techniques (SEPS) have now become available. SEPS is ineffective in postthrombotic syndrome.[31] Deep valve reconstruction can be performed safely and successfully in 'primary' as well as postthrombotic veins, even in trabeculated veins, using a variety of techniques in accordance with the pathology.[14, 16, 32-34] Perhaps because of the demanding technique, it has been confined to only a handful of centers worldwide. The industry has expended enormous resources in the development of a percutaneously implantable venous valve, but all such efforts have not borne fruit to date.

The excellent clinical response to stent correction of the obstructive component even in the presence of associated reflux has drastically reduced the need for deep valve reconstruction.

Table 1. Clinical Classification of Venous Disease

Clinical class	
C_0	No venous disease
C_1	Spiders
C_2	Varicose veins
C_3	Edema
C_4	Hyperpigmentation, Dermatitis
C_5	Healed ulceration
C_6	Active ulceration

Clinical Considerations

Clinical presentation and symptom severity are widely variable in chronic venous insufficiency[35] (Table 1). Spiders and varices may be asymptomatic, associated with cosmetic complaints or local pain. Local removal (stab phlebectomy/photoablation) or sclerotherapy is effective in such cases. When associated saphenous reflux is present, it should be eradicated by saphenous ablation to reduce chances of varix recurrence.[36] Pain extending beyond the spiders and varices to the calf muscle or more diffusely to the entire limb is often indicative of deep venous involvement and these patients may be candidates for stent therapy, depending upon the severity of pain. In about 10% of patients, pain may be the *only* clinical feature of chronic venous insufficiency present in an *otherwise normal-looking limb*.[5] Pain disproportionate to clinical signs such as varices or minimal swelling is also indicative of deep venous involvement. Venous pain is characteristically orthostatic, worse with sitting or standing, and relieved with leg elevation, ambulation (which reduces venous pressure) and stockings. Venous claudication mimicking arterial claudication is present in some patients; a treadmill test with ankle pressure measurement is required for differentiation. Restless legs and night cramps are common features of chronic venous insufficiency. Pain may be severe enough to require narcotics and some of these patients may easily be mistaken for drug addicts. Subjective pain description by the patient is unreliable and routine use of a simple visual analogue scale is recommended[37] (Fig. 1).

With some exceptions, leg edema, stasis skin changes or ulceration are indicative of deep venous involvement.[38] Venous ulcers are usually distinguish-

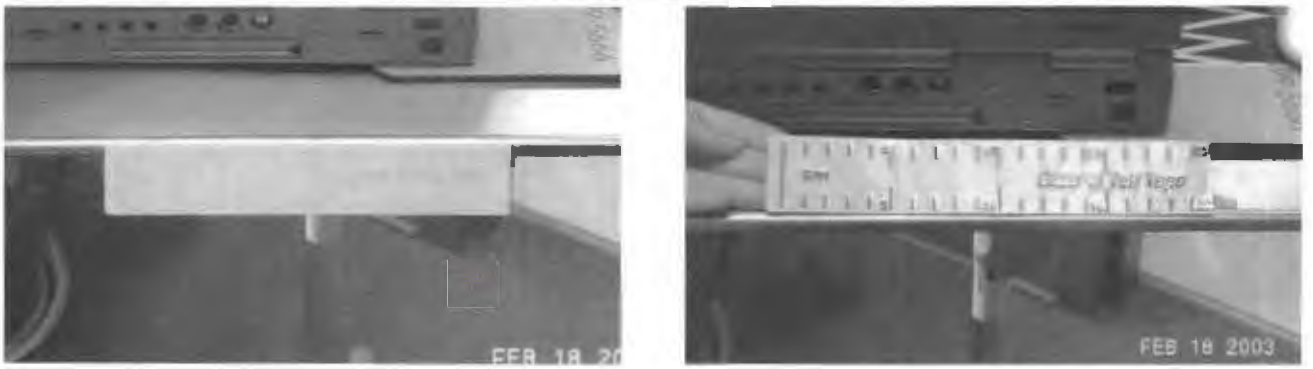

Figure 1. A simple visual analogue scale constructed from a 10" strip of paper tape. Patient indicates level of pain on the blank side which then is converted to numbers, 0-10 from the corresponding point on the scale side. Level 10 should be described in concrete fashion, such as for example "suicidal pain," and used consistently thereafter.

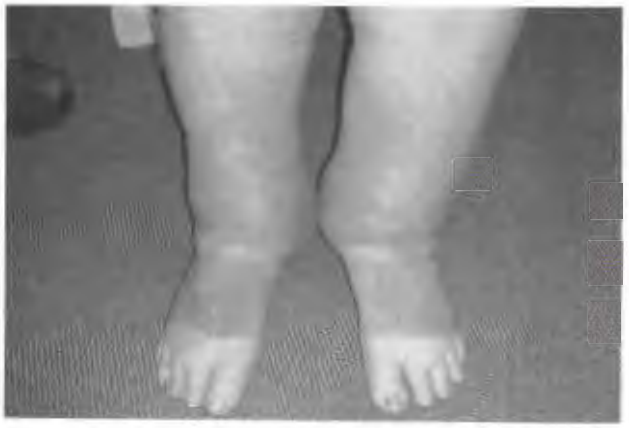

Figure 2. Seventy-eight-year-old lady with bilateral limb swelling which was a source of considerable morbidity. Such swelling is often empirically treated with diuretics (ineffective) and seldom investigated. Venous outflow obstruction may underlie such presentation. Bilaterality does not rule out such a diagnois.

able from arterial ulcers by clinical features such as associated stasis skin changes. The presence of palpable pedal pulses virtually rules out an arterial etiology. Pedal pulses may be absent, however, in some elderly patients despite a venous etiology, and combined arterial venous ulcers do occur in the elderly. Detailed vascular laboratory studies are required for proper diagnosis in such cases.

Extensive varicosities and saphenous reflux may cause mild forms of limb edema; more severe swelling is usually indicative of deep venous obstruction/reflux. All patients with leg swelling whether mild or severe should undergo a comprehensive venous examination. If a deep venous lesion is not investigated or is undetected before superficial venous surgery, leg swelling may worsen afterwards to the dismay of the patient and the treating physician. Many elderly patients, particularly women, develop leg edema in the sixth and later decades of life, limiting their mobility and their ability to take care of themselves (Fig. 2). This can be a serious quality of life issue. "Fluid overload," "a touch of congestive failure" or "hormonal imbalance" is often blamed; diuretics are often prescribed to no avail. Patients in this age group are often unable to apply or tolerate tight-fitting stockings. Increasing leg dependency (such as while watching TV), and venosclerosis combine with previously incipient iliac venous compression to render the limb edematous in many such cases.[5] All such patients, even if the involvement is bilateral, deserve a thorough venous investigation. Swelling relief and the improvement in quality of life following stent placement can be impressive. The procedure has been safely performed even in octogenarians who are otherwise reasonably healthy.[39]

Venous edema is often confused with lymphedema. There are no clinical features that can reliably distinguish one from other, including age of onset, family history and bilaterality. All such cases should undergo a proper venous investigation (IVUS) before being labeled as lymphedema and prescribed lifelong conservative treatment, which is often ineffective. Neither can abnormal lymphangiography be relied upon, as about 30% of venous obstruction is associated with secondary (due to lymphatic overload and subsequent damage) lymphedema.[40,41] Timely venous stenting to correct the obstruction may reverse the abnormal lymphangiogram (Fig. 3).[42]

Investigations

Ascending venography by pedal injection of contrast frequently does not opacify the iliac veins adequately for proper evaluation; transfemoral injection of contrast is required. Even with adequate opacification, diagnostic sensitivity of single plane venography is

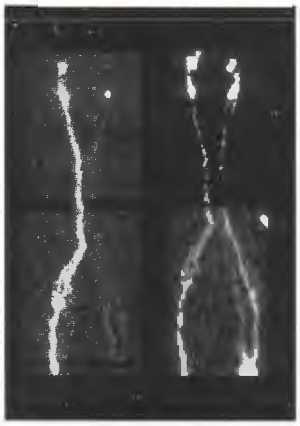

Figure 3. Absent lymph flow in the left lower limb with isotope lymphangiography (left) with recovery of normal flow after venous stenting. Secondary lymphatic dysfunction from venous disease is more common than 'primary' lymphedema. (By permission, *J Vasc Surg.*)

only about 50% as compressive iliac vein lesions present in a single plane, either sagittal or coronal, and intraluminal lesions are easily obscured by contrast[5,43] (Fig.4). Intravascular ultrasound (IVUS) has become the standard in iliac vein assessment as it does not have such projectional limitations, is free of radiation exposure and offers additional detail such as wall fibrosis and trabeculae.[44] The diagnostic accuracy of IVUS is >90% and has become essential for proper stent placement.

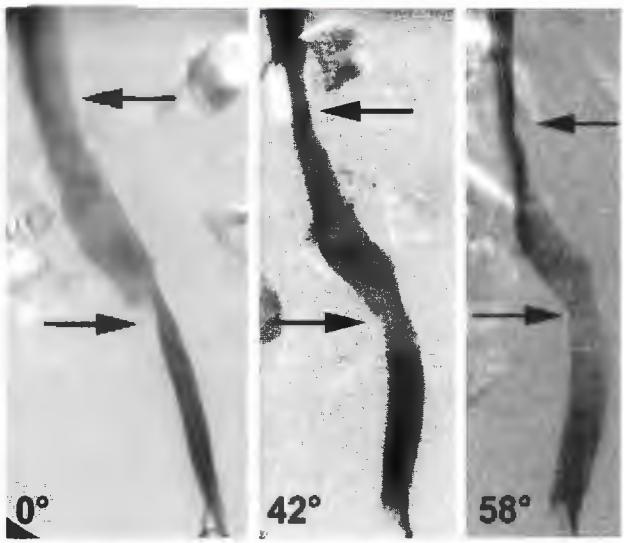

Figure 4. NIVL: the proximal lesion is coronal and the distal saggittal, resulting in appearance and disappearance of lesion from frontal to lateral projection. (By permission, *J Vasc Surg.*)

Compression stockings in chronic venous disease

Compression stockings are effective in the short term if properly prescribed and used *regularly*. Even under these ideal conditions, about 30% of patients will fail compression therapy short term and even a higher percentage long term (> 1 year).[45] Antithrombotic stockings commonly used in hospitals (e.g., TED) do not provide adequate compression to relieve symptoms of chronic venous insufficiency. Class 1 stockings (20-30 mm Hg pressure) are required to relieve mild edema;[46] and even higher elastic compression (Class 2; 30-40 mm Hg) or semi-rigid support (Circaid TM) are required to control gross edema and stasis skin changes/ulcers.[47] Semi-rigid/rigid bandaging such as the Unna boot or four-layer compressive wraps applied by the physician or wound care centers are effective in healing ulcers and stasis dermatitis but recurrence is the rule if the underlying pathology is left uncorrected and the patient does not follow with regular use of high compression stockings or semi-rigid legging afterwards.[48] Unfortunately, nearly two-thirds of patients are unable or unwilling to wear compression for a variety of reasons: physical frailty or arthritis, extreme general obesity and that of the limb, precarious condition of the skin, exudative edema, poor fit or binding at the upper edge, contact dermatitis and intolerance to the device ("too hot" or "cuts off the circulation").[49] About 20 to 30% of patients without such apparent limitations or reasons become non-compliant because of a sense of restriction of life style. Patient education and motivational sessions have not improved compliance in this group despite the presence of advanced and often disabling symptoms. Non-use/non-compliance with compression is the most common and the major cause of persistence of symptoms. Venous stenting offers an alternative to these patients as use of stockings can be altogether abandoned or reduced to occasional usage on "heavy days" on the legs. Specific correction of pathology with a chance of long-term cure has some attraction over empiric and continuous use of stockings, which are effective only as long as they are worn.

Technique of iliac vein stenting

Endovenous stent technique is markedly different from arterial stent technique.[11,19,50] Most arterial stenotic lesions respond well to balloon dilatation alone as recoil is minimal. Recoil is universal in veins, mandating stent placement after balloon dilatation. Large stents (14 to 16 mm) approximating normal size are required for iliac veins. Smaller sized stents may not provide adequate decompression of the limb and may thrombose. Extension into the inferior vena cava for 2 to 3 cm is necessary as the iliac-IVC junction is a choke point, tending to push the stent distally. If the stent is placed at the mouth of the iliac vein, recurrent stenosis can be expected with distal migration of the

stent. Vena cava extension does not compromise contralateral flow. The stent can be extended across the groin crease to correct retroinguinal lesions, as stent erosions and fractures well known in the arterial system do not occur among venous stents. All venous stenotic lesions should be corrected for successful decompression of the limb and long stent lengths (40 cm or more) are not thrombogenic in and of themselves; uncorrected residual lesions may lead to stent malfunction and thrombosis. Total iliac vein occlusions and even longstanding complete vena caval occlusions can be recanalized and stented with a high degree of procedural and long-term clinical success[18, 51] (Fig. 5).

Absent thrombophilia, only aspirin is required after stent placement. In coagulopathic patients either short-term or long-term anticoagulation is desirable, depending on the severity of the coagulopathy.

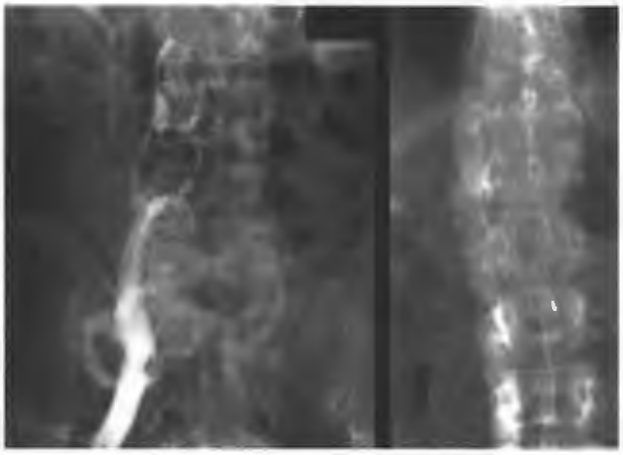

Figure 5. Even totally occluded veins can be successfully recannalized by stent technique. Recannalization of a totally occluded inferior vena cava is shown. The stent extends to the right atrium. (By permission, *J Vasc Surg.*)

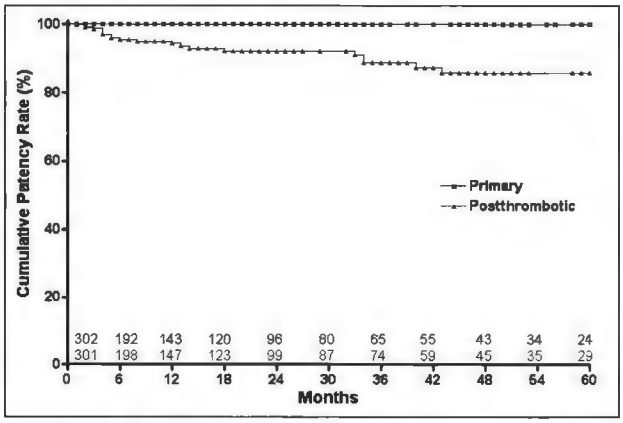

Figure 6. Cumulative rate of secondary stent patency in 'primary' and postthrombotic disease.

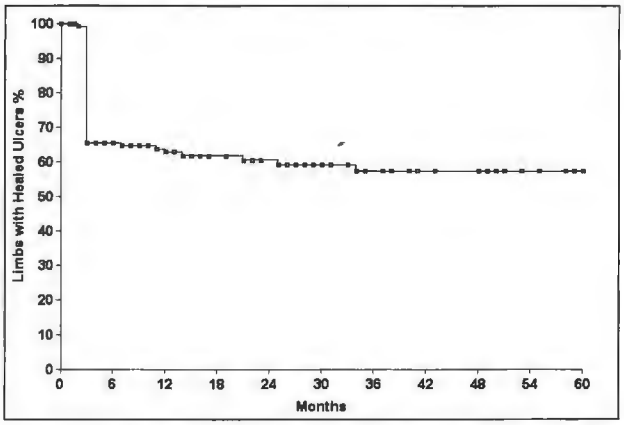

Figure 7. Cumulative rate of stasis ulcer healing after venous stenting; associated reflux was not corrected in the group with such reflux. (By permission, *J Vasc Surg.*)

Results

Venous stenting is a safe procedure with negligible mortality and low morbidity.[39] Both short-term and long-term rates of DVT are low and are similar to natural rate in patients with venous pathology of the type being treated. Long-term stent patency is astonishingly high in 'primary' disease and only slightly less in postthrombotic disease (Fig. 6). Clinical results correspond, with excellent *complete* relief of pain; *complete* relief of swelling occurs less often (62% and 30% cumulative at 6 years, respectively). Others receive partial relief of swelling which is clinically beneficial, especially when the pain component is eliminated. Notably, 58% of ulcers heal and remain healed long-term with little degradation of the curve even when associated reflux is left uncorrected (Fig. 7). Quality of life is significantly improved.

Summary

Venous stenting has introduced a minimally invasive and safe technique that can be performed on an outpatient basis with little downtime for the patient. It is applicable in a wide spectrum of patients with chronic venous disease with disabling symptoms. Long-term patency and clinical outcome are excellent even when associated reflux is present and left untreated. The need for open surgery to correct reflux or obstruction has been drastically reduced; however, such procedures can still be carried out later in the event of stent occlusion or failure to relieve symptoms.

References

1. Kistner RL. Primary venous valve incompetence of the leg. *Am J Surg.* 1980; 140(2):218-24.
2. Prandoni P, Lensing AW, Prins MR. Long-term outcomes

after deep venous thrombosis of the lower extremities. *Vasc Med.* 1998;3(1):57-60.
3. Johnson BF, Manzo RA, Bergelin RO, Strandness DE Jr. Relationship between changes in the deep venous system and the development of the postthrombotic syndrome after an acute episode of lower limb deep vein thrombosis: a one- to six-year follow-up. *J Vasc Surg.* 1995; 21(2):307-12; discussion 313.
4. Raju S, Fredericks RK, Hudson CA, Fountain T, Neglen PN, Devidas M. Venous valve station changes in "primary" and postthrombotic reflux: an analysis of 149 cases. *Ann Vasc Surg.* 2000;14(3):193-9.
5. 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(1):136-43; discussion 144.
6. Ehrich WE, Krumbhaar EB. A frequent obstructive anomaly of the mouth of the left common ilia vein. *Am Heart J.* 1943;26:737-50.
7. Cockett FB, Thomas ML. The iliac compression syndrome. *Br J Surg.* 1965;52(10):816-21.
8. May R, Thurner J. The cause of the predominantly sinistral occurrence of thrombosis of the pelvic veins. *Angiology* 1957;8:419-27.
9. McMurrich JP. The occurence of congenital adhesions in the common iliac veins and their relation to thrombosis of the femoral and iliac veins. *Am J M Sc.* 1908;135:342-6.
10. Kibbe MR, Ujiki M, Goodwin AL, Eskandari M, Yao J, Matsumura J. Iliac vein compression in an asymptomatic patient population. *J Vasc Surg.* 2004;39(5):937-43.
11. Raju S, Owen S Jr, Neglen P. The clinical impact of iliac venous stents in the management of chronic venous insufficiency. *J Vasc Surg.* 2002;35(1):8-15.
12. Neglen PN, Thrasher TL, Raju S. Venous Outflow Obstruction - An Underestimated Contributor to Chronic Venous Disease. *J Vasc Surg.* 2003;38(5):879-85.
13. 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(1):136-43; discussion 144.
14. Raju S, Hardy JD. Technical options in venous valve reconstruction. *Am J Surg.* 1997;173(4):301-7.
15. Kistner RL EBME. Deep venous valve reconstruction. *Cardiovasc Surg.* 1995;3:129-40.
16. Raju S, Neglen P, Doolittle J, Meydrech EF. Axillary vein transfer in trabeculated postthrombotic veins. *J Vasc Surg.* 1999;29(6):1050-62; discussion 1062-4.
17. Raju S, Fountain T, Neglen P, Devidas M. Axial transformation of the profunda femoris vein. *J Vasc Surg.* 1998; 27(4):651-9.
18. Raju S, Hollis K, Neglen P. Obstructive lesions of the inferior vena cava: clinical features and endovenous treatment. *J Vasc Surg.* 2006;44(4):820-7.
19. Neglen P, Raju S. Proximal lower extremity chronic venous outflow obstruction: recognition and treatment. *Semin Vasc Surg.* 2002;15(1):57-64.
20. Neglen P, Berry MA, Raju S. Endovascular surgery in the treatment of chronic primary and post- thrombotic iliac vein obstruction. *Eur J Vasc Endovasc Surg.* 2000; 20(6):560-71.
21. Neglen P, Thrasher TL, Raju S. Venous outflow obstruction: An underestimated contributor to chronic venous disease. *J Vasc Surg.* 2003;38(5):879-85.
22. Neglen P, Raju S. A rational approach to detection of significant reflux with duplex Doppler scanning and air plethysmography. *J Vasc Surg.* 1993;17(3):590-5.
23. 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.* 1992;16(5):687-93.
24. Raju S, Fredericks R. Valve reconstruction procedures for nonobstructive venous insufficiency: rationale, techniques, and results in 107 procedures with two- to eight-year follow-up. *J Vasc Surg.* 1988;7(2):301-10.
25. Raju S, Fredericks RK, Neglen PN, Bass JD. Durability of venous valve reconstruction techniques for "primary" and postthrombotic reflux. *J Vasc Surg.* 1996;23(2):357-66; discussion 366-7.
26. Kistner RL, Eklof B, Masuda EM. Deep venous valve reconstruction. *Cardiovasc Surg.* 1995;3(2):129-40.
27. Raju S. Venous insufficiency of the lower limb and stasis ulceration. Changing concepts and management. *Ann Surg.* 1983;197(6):688-97.
28. Neglen P, Hollis KC, Raju S. Combined saphenous ablation and iliac stent placement for complex severe chronic venous disease. J Vasc Surg 2006; 44(4):828-33.
29. Raju S, Easterwood L, Fountain T, Fredericks RK, Neglen PN, Devidas M. Saphenectomy in the presence of chronic venous obstruction. *Surgery* 1998;123(6):637-44.
30. Burnand KG, O'Donnell TF Jr, Thomas ML, Browse NL. The relative importance of incompetent communicating veins in the production of varicose veins and venous ulcers. *Surgery* 1977;82(1):9-14.
31. Gloviczki P, Bergan JJ, Rhodes JM, Canton LG, Harmsen S, Ilstrup DM. Mid-term results of endoscopic perforator vein interruption for chronic venous insufficiency: lessons learned from the North American subfascial endoscopic perforator surgery registry. The North American Study Group. *J Vasc Surg.* 1999;29(3):489-502.
32. Raju S, Berry MA, Neglen P. Transcommissural valvuloplasty: technique and results. *J Vasc Surg.* 2000; 32(5):969-76.
33. Puggioni A, Kistner RL, Eklof B, Lurie F. Surgical disobliteration of postthrombotic deep veins--endophlebectomy--is feasible. *J Vasc Surg.* 2004; 39(5):1048-52; discussion 52.
34. Maleti O, Lugli M. Neovalve construction in postthrombotic syndrome. *J Vasc Surg.* 2006; 43(4):794-9.
35. Beebe HG, Bergan JJ, Bergqvist D et al. Classification and grading of chronic venous disease in the lower limbs. A consensus statement. *Eur J Vasc Endovasc Surg.* 1996; 12(4):487-91; discussion 491-2.
36. Sarin S, Scurr JH, Coleridge Smith PD. Stripping of the long saphenous vein in the treatment of primary varicose veins. *Br J Surg.* 1994;81(10):1455-8.
37. Scott J, Huskisson EC. Graphic representation of pain. *Pain* 1976;2(2):175-84.
38. Shami SK, Sarin S, Cheatle TR, Scurr JH, Smith PD. Venous ulcers and the superficial venous system. *J Vasc*

Surg. 1993; 17(3):487-90.

39. Neglen P, Hollis KC, Olivier J, Raju S. Stenting of the Venous Outflow in Chronic Venous Disease: Long-term Stent-related Outcome, Clinical and Hemodynamic Result. *J Vasc Surg.* 2007;46(5):979-990.
40. Collins PS, Villavicencio JL, Abreu SH GE et al. Abnormalities of lymphatic drainage in lower extremities: a lymphoscintigraphic study. *J Vasc Surg.* 1989; 9:145-52.
41. Gloviczki P, Calcagno D, Schirger A et al. Noninvasive evaluation of the swollen extremity: experiences with 190 lymphoscintigraphic examinations. *J Vasc Surg.* 1989; 9:683-89.
42. 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(5):779-84.
43. Negus D, Fletcher EW, Cockett FB, Thomas ML. Compression and band formation at the mouth of the left common iliac vein. *Br J Surg* 1968;55(5):369-74.
44. Neglen P, Raju S. Intravascular ultrasound scan evaluation of the obstructed vein. *J Vasc Surg.* 2002;35(4):694-700.
45. Barwell JR, Davies CE, Deacon J et al. Comparison of surgery and compression with compression alone in chronic venous ulceration (ESCHAR study): randomised controlled trial. *Lancet* 2004;363(9424):1854-9.
46. Partsch H, Winiger J, Lun B. Compression stockings reduce occupational leg swelling. *Dermatol Surg.* 2004; 30(5):737-43; discussion 743.
47. Cullum N, Nelson EA, Fletcher AW, Sheldon TA. Compression for venous leg ulcers. *Cochrane Database Syst Rev.* 2001;(2):CD000265.
48. Mayberry JC, Moneta GL, Taylor LM Jr, Porter JM. Fifteen-year results of ambulatory compression therapy for chronic venous ulcers. *Surgery* 1991; 109(5):575-81.
49. Raju S, Hollis KC, Neglen P. Compression Stockings in Chronic Venous Disease: Patient Compliance and Efficacy. *Ann Vasc Surg.* 2007;21(6):790-5.
50. Neglen P, Raju S. Balloon dilation and stenting of chronic iliac vein obstruction: technical aspects and early clinical outcome. *J Endovasc Ther.* 2000;7(2):79-91.
51. Raju S, McAllister S, Neglen P. Recanalization of totally occluded iliac and adjacent venous segments. *J Vasc Surg.* 2002;36(5):903-11.

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