# Chronic Venous Insufficiency

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**Chronic venous insufficiency has been known as a distinct pathologic entity at least since the time of Hippocrates, yet many pathologic features of this common ailment remain obscure. A recent resurgence of investigative effort in chronic venous insufficiency has occurred, spurred by the development of valve reconstruction surgery. The advent of newer diagnostic devices and techniques such as duplex scan and air plethysmography allow for a more detailed examination of the venous system than was possible previously.**

## PATHOPHYSIOLOGY

Hemodynamic forces in veins are distinctly different from arteries; veins function as collapsible tubes in the normal physiologic pressure ranges. This is especially evident during pressure changes that normally occur with ambulation. In a person standing erect and motionless with the calf muscles relaxed, the foot venous pressure is high (about 90 mm Hg) and the calf veins are full and distended ('stretching' regimen). With emptying of calf veins after ambulation, these veins collapse ('bending' regimen), resulting in a reduction of foot venous pressure. Collapse of the calf veins is primarily responsible for the foot venous pressure reduction even though column segmentation may occur concurrently.[1] The volume pressure relationship in collapsible tubes is nonlinear and asynchronous. This is apparent in volume pressure curves obtained from a mechanical model simulating calf venous pump action (Figure 1).

It is evident that most of the pressure reduction occurs during the initial phase of tube emptying in the stretching regimen from a relatively small initial volume reduction. In the collapsed stage ('bending' regimen), pressure remains relatively constant when large changes in volume take place with further ejection. By setting the mechanical model for varying degrees of reflux and ejection fractions, it can be shown that the tube in the collapsed mode functions as a buffer, keeping pressure relatively constant despite wide variations in reflux volume and ejection fraction. The primary pressure-regulating mechanism is *tube collapse* with valve closure having a secondary role by maintaining and prolonging tube collapse. Because of the nonlinear volume pressure relationship, a reduction in reflux volume following valve reconstruction may result in little change in ambulatory venous pressure, even though the duration of tube collapse (ie, recovery time) is usually prolonged.

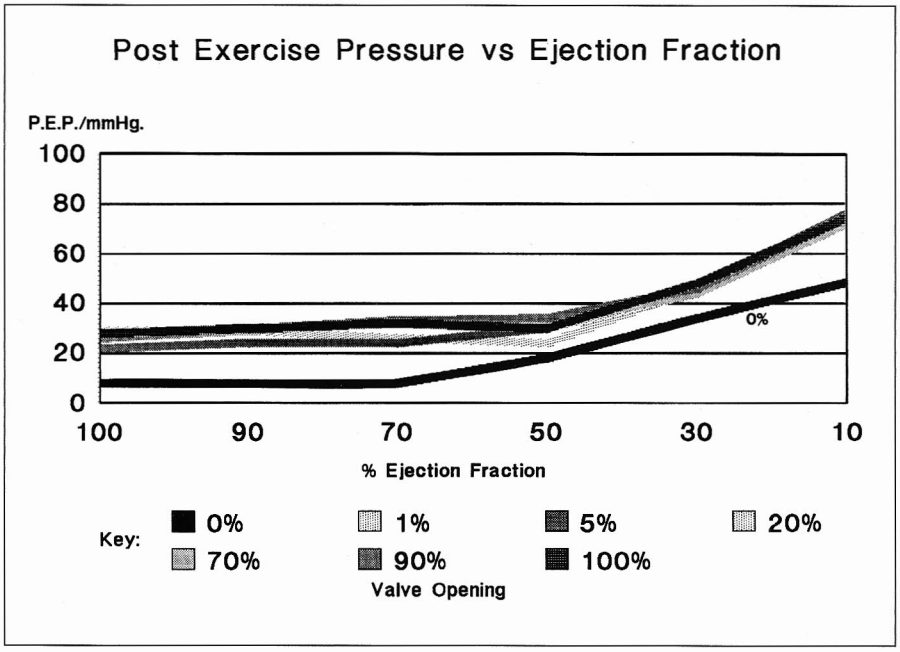


**Figure 1: Volume pressure curves derived from a mechanical model of calf venous pump. Note the rapid reduction of pressure up to about 30% ejection fraction after which the curve flattens out; pressure remains relatively constant around 30 mm Hg for a wide range of ejection fractions and valve opening (reflux). (With permission from: Raju S, Fredericks R, Lishman P, Neglén P, et al. Observations on the calf venous pump mechanism: Determinants of postexercise pressure. J Vasc Surg 1993; 17:459-69.)**

It is highly likely that changes in venous wall compliance, such as might occur in the postthrombotic extremity, will significantly alter calf venous function impeding its buffering capacity. This aspect of venous pathophysiology remains largely unexplored.

The causal relationship between ambulatory venous hypertension and venous stasis ulceration has been considered nearly axiomatic, yet approximately 20% of patients with stasis ulceration have ambulatory venous pressures considered to be in the 'normal' range.[2] Little is known about the precise relationship between ambulatory venous hypertension and venous stasis changes. Plethysmographic studies indicate that the superficial and deep venous system function as a single, integrated, hemodynamic entity and the traditional separation between the two might be artificial.[3] Significant reflux that occurs in either system may result in pathologic venous stasis. The role of perforators and the concept of perfora-

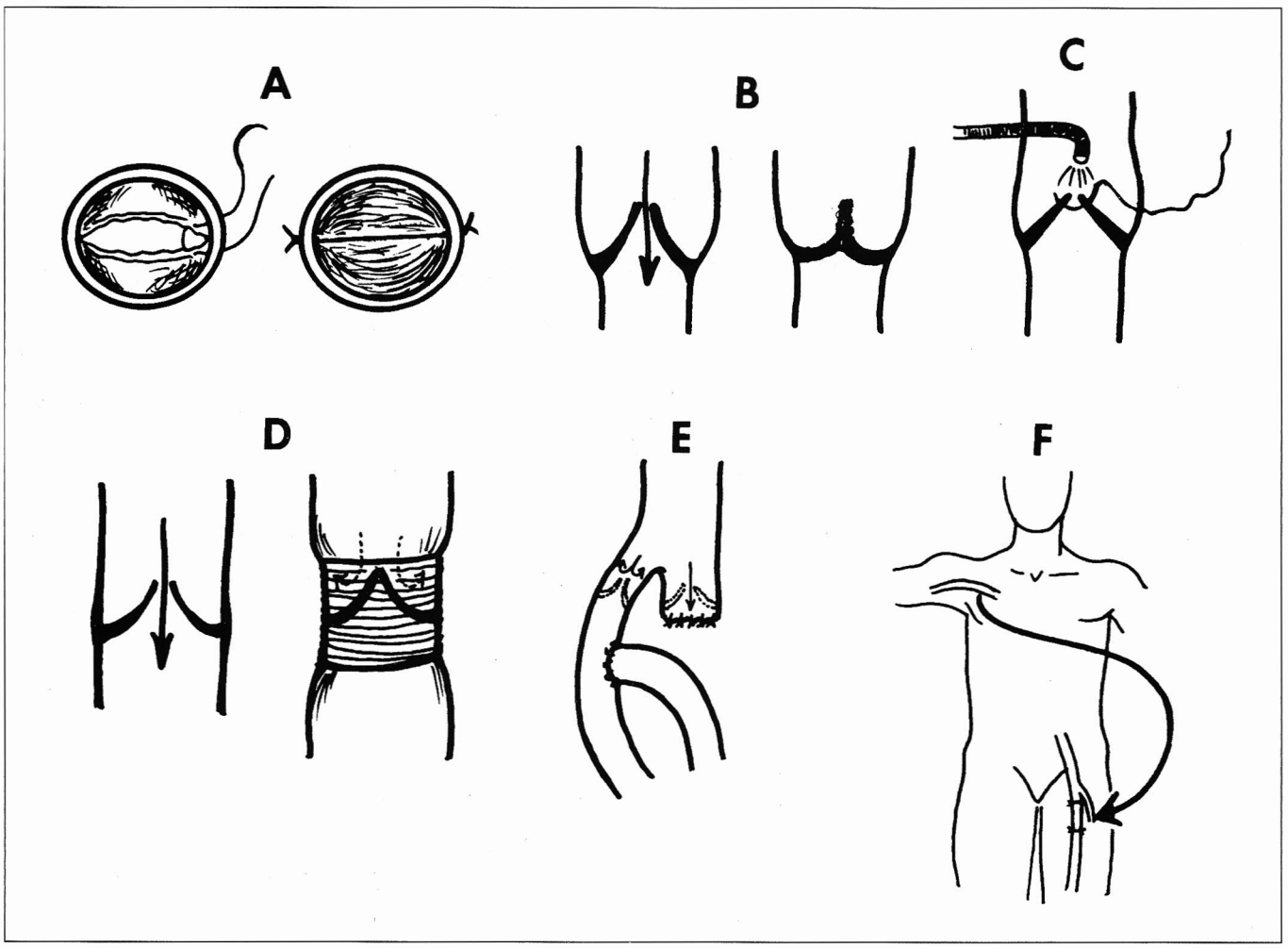


**Figure 2: Methods of venous valve reconstruction: A: Internal valvuloplasty. B: External valvuloplasty. C: Angioscopic valvuloplasty. D: Prosthetic sleeve *in situ*. E: Kistner segment transfer. F: Axillary vein valve transfer.**

tor incompetence as a major factor inducing venous stasis changes are undergoing reevaluation; duplex and contrast studies indicate that a bi-directional to-and-fro flow is normally present in perforators,[4] and prominent reverse flow is invariably the result of popliteal or tibial valve incompetence.[5]

Chronic venous insufficiency may result from obstruction, reflux, or a combination of these factors. Reflux may arise from a variety of *congenital* abnormalities involving aplasia or dysplasia of the venous valve and conduit. 'Primary' valve reflux, believed to be non-thrombotic in origin, is one of the major etiologic mechanisms in chronic venous insufficiency and accounts for about 30% of reflux cases encountered in modern hospital practice. The remainder result from postthrombotic syndrome.

An overwhelming proportion of cases exhibiting obstruction or a combination of obstruction and reflux is also the result of previous venous thrombosis. *Obstruction* is occasionally produced by non-thrombotic causes such as tumor, bands, or compression by the adjacent artery (eg, the left iliac vein compression by the right common iliac artery). Sequential duplex examination of extremities affected by deep venous thrombosis has documented the rapid resolution of thrombosed venous segments.[6]

Clot resolution/retraction occurs in 80% to 90% of thrombosed venous segments by four months after the initial onset. Complete resolution is seldom achieved, however, with partial obstruction being present in most venous segments and 14% remaining completely thrombosed. Collateral development is often rapid following venous thrombosis and frequently results in normalized venous pressures even when recanalization is partial or totally absent. When developed, collaterals appear to persist even when the initial thrombus is later completely resolved.[7]

Collateral reflux frequently accompanies collateral reflux and compounds the reflux that may occur through the recanalized axial vein itself. Postthrombotic compliance and capacitance changes in calf veins, resulting in calf venous pump dysfunction, add to the aforementioned reflux abnormality in producing the panoply of symptoms and signs generally described as postthrombotic syndrome. *Venous claudication* syndrome, in which the venous pressure rises above resting levels with calf exercise, is an extremely rare entity despite the attention it has received in the literature.

## DIAGNOSIS

The advent of duplex Doppler has supplanted earlier indirect diagnostic methods (photoplethysmography, transcutaneous oxygen measurement, etc.) and invasive diagnostic procedures (ambulatory venous pressure measurement) as the initial screening choice in assessing chronic venous insufficiency. Duplex is, however, largely a qualitative tool, but recent attempts have been made to extract quantitative information with this technique. These efforts involve measurement of reflux duration compared to the ejection phase,[8] or measuring actual valve closure times using standardized rapid inflation/deflation pneumatic cuffs applied to the calf and thigh muscles.[9] The development of air plethysmography (APG™, ACI Medical, Sunnyvale, CA) has facilitated easy clinical application of the plethysmographic technique, providing details of calf venous pump function and quantification of venous reflux.

Diagnostic techniques applicable to venous obstruction have lagged behind those developed for assessment of reflux. Ascending venography remains the mainstay of evaluating obstruction in many centers. Contrast ascending venography is an anatomic method that provides little useful physiologic information regarding the functional adequacy of recanalization and collateral development. In fact, ascending venography can be misleading in this respect.[10]

Plethysmographic techniques, such as measurement of outflow fraction, are cumbersome and prone to technical error, difficult to standardize, and may be affected solely by venous compliance changes independent of any obstruction impeding outflow. Measurement of

**VENOUS VALVE RECONSTRUCTION**

| Methods | Comments |
|---|---|
| *Internal Valvuloplasty* | Initial technique of valve reconstruction described by Kistner.[12] Precise and effective. Valve may be approached by way of a longitudinal, transverse or T-incision. Time-consuming. Currently, not applicable to valves in the crural veins. |
| *External Valvuloplasty* | Avoids venotomy. Valve attachment lines are brought together by external sutures, closing the wide commissural angle. Rapid. Applicable to valves in small-caliber veins. Durability is currently under assessment. |
| *Prosthetic Sleeve In Situ* | May be used when surgical manipulation results in venoconstriction while restoring valve competency by strip test. Indirect technique, rapid and applicable to small-caliber veins. Not recommended when gross reflux is present after venoconstriction. |
| *Angioscopic Valvuloplasty* | Uses combined internal/external technique. Minimal venotomy. Extent of valve tightening can be accurately assessed with angioscopic visualization. Function of the repaired valve can also be better assessed by angioscopic irrigation rather than the traditional strip test. |
| *Kistner Segment Transfer* | The superficial femoral vein is divided and anastomosed to the profunda vein below a competent profunda valve. Not applicable when the profunda valve is absent or incompetent. |
| *Axillary Vein Valve Transfer* | Useful option when the postthrombotic valve is beyond repair. Forty percent of axillary vein valves are refluxive *in situ*. Yields inferior results compared to valvuloplasty because transferred axillary vein valves frequently dilate (probably from compliance mismatch) and become refluxive. The restrictive prosthetic sleeve may aid in preventing such dilatation. |

**Table 1.**

arm/foot venous pressure differential combined with monitoring foot/venous pressure elevation following reactive hyperemia induction appear to be the most reliable method currently available for the diagnosis and grading of venous obstruction.[11]

In high-grade venous obstruction (grade 4 decompensated), expected foot/venous pressure elevation following a period of ischemic cuff occlusion is paradoxically absent, apparently because the reactive hyperemia response is either muted or absent entirely.[7] Application of this diagnostic technique in a large number of venographic obstructions illustrates the variable pattern of functional adaptation to the presence of axial venous obstruction. Axial venous obstruction in the pelvis and abdomen (ie, iliac veins and vena cava) was fully compensated by collateral development in more than half the cases studied. Thus, the venographic appearance of axial venous obstruction may belie the underlying functional adequacy. Five percent of crural obstructions may be high grade (grade 4) because of the inadequate recanalization/collateralization.

## CLINICAL FEATURES AND INDICATIONS FOR SURGERY

Pain, swelling, and stasis skin changes or combinations thereof are presenting features of chronic venous insufficiency. Stasis skin changes imply reflux either in its pure form ('primary' reflux) or in combination with obstruction in postthrombotic syndrome. Painful swelling without stasis changes is a feature of severe venous obstruction even though reflux may also be a causative factor in producing this symptom combination in some cases.

Recurrent cellulitis and recurrent venous thrombosis are less commonly recognized features of chronic venous insufficiency and are each present in about 5% of cases. The mainstay of treatment in chronic venous insufficiency remains conservative, consisting of compression therapy and support stockings combined with leg elevation. Failure of conservative therapy or recurrent complications are indications for surgical intervention. Socioeconomic factors, such as age, occupational requirements, or others, may preclude effective conservative therapy. Surgery may be considered on a selective basis in such instances.

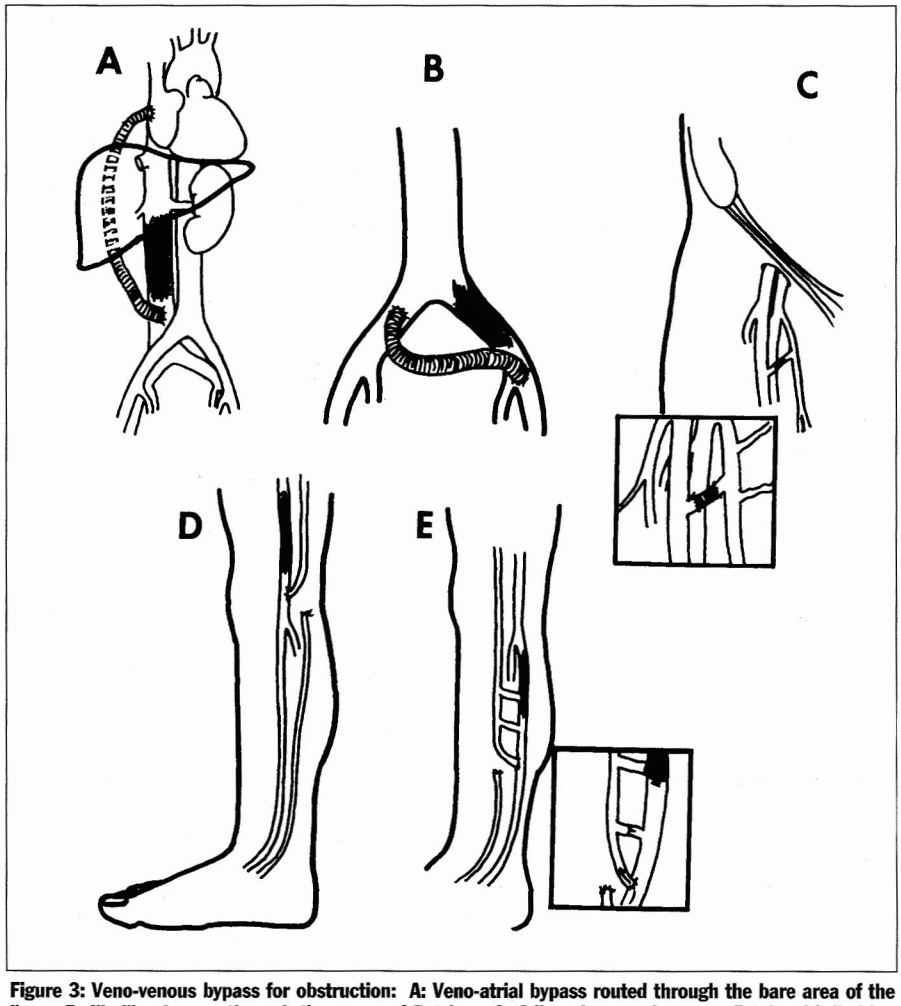


**Figure 3: Veno-venous bypass for obstruction: A: Veno-atrial bypass routed through the bare area of the liver. B: Ilio-iliac bypass through the space of Retzius. C: Adjunctive arteriovenous fistula with limiting prosthetic sleeve. D: Husni bypass for popliteal vein obstruction. E: Perforator bypass for crural vein obstruction. Inset in Figure 3E shows orientation of the perforator valves impeding the flow from deep to the superficial system in symptomatic patients.**

## TREATMENT: REFLUX

Ascending and descending venography should be performed before surgery to obtain the necessary anatomic detail for the surgical approach. The technique of valve repair by internal valvuloplasty as initially described by Kistner[12] has since evolved into a number of technical options, both direct and indirect, to reconstruct the refluxive valve (Figure 2; Table I). Repair of a single valve in the axial vein, either in the femoral area or distally in the adductor canal, is usually adequate in primary valve reflux. In postthrombotic syndrome when significant collateral reflux is present, multiple valve reconstruction is recommended to control reflux both at the inflow and outflow sites of the collateral.[13] Ligation of collaterals is not recommended because outflow obstruction may result from residual lesions present in the axial vein due to incomplete recanalization. The presence of residual obstructive lesions cannot be reliably assessed by ascending venography.

Valve reconstruction surgery yields excellent results, with 60% to 80% resolution of venous stasis.[5,14] These results appear to be durable, with decay of only 10% to 20% during the first three to four years and remaining stable from then on.

## VENOUS OBSTRUCTION

There is increasing awareness that a venous bypass should not be performed on the basis of venographic appearance alone without accompanying functional tests that confirm the presence of a significant gradient across the site of the obstruction. For iliac and vena cava obstruction, a large prosthesis (preferably 14 mm to 17 mm) stented to prevent collapse from higher abdominal pressure is recommended (Figure 3).

In most instances, the saphenous vein, as used originally in the Palma procedure, is inadequate in size to decompress an obstruction of the iliac or vena cava level. An adjunctive arteriovenous fistula between the saphenous vein or tributary and the adjoining superficial femoral vein increases bypass flow and maintains graft patency during the critical early postoperative period. A prosthetic sleeve of Dacron® or Gore-Tex®, which limits the fistula to about 4 mm, diminishes the chance of distal venous hypertension and assists in percutaneous closure of the fistula later, if necessary (Figure 3).

Even though the prosthetic venovenous bypass enjoys a surprisingly good short-term patency up to six months,[15] information regarding long-term outlook is scant. Using a preperitoneal route through the space of Retzius (Figure 3), Gruss has reported excellent long-term patency in venovenous bypasses for iliac vein obstruction.[16] Axial venous obstruction in the femoral vein or crural veins is usually well-compensated and a bypass is generally not required. As indicated previously, high-grade venous obstruction may be present occasionally, however, and a bypass may be considered in such cases (Figure 3). The Husni[17] and perforator bypasses[18] use the saphenous vein *in situ* for direct anastomosis to the popliteal or crural veins to bypass obstruction (Figure 3).

Despite the high incidence of early postoperative thrombosis, perforator bypasses curiously recanalize to yield an excellent 100% long-term patency rate with resolution of obstructed symptoms. Reported results of the Husni bypass are also similarly excellent. STI

## REFERENCES

1. Raju S, Fredericks R, Lishman P, et al. Observations on the calf venous pump mechanism: Determinants of postexercise pressure. J Vasc Surg 1993; 17:459-69.
2. Raju S, Fredericks R. Hemodynamic basis of stasis ulceration: A hypothesis. J Vasc Surg 1991; 13:491-5.
3. Nicolaides AN, Sumner DS (eds). *Investigation of Patients with Deep Vein Thrombosis and Chronic Venous Insufficiency*. Los Angeles: Med-Orion, 1991:49.
4. Sarin S, Scurr JH, Smith C. Medial calf perforators in venous disease: The significance of outward flow. J Vasc Surg 1992; 16:40-6.
5. 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; 7:301-10.
6. Killewich LA, Bedford GR, Beach KW, et al. Spontaneous lysis of deep venous thrombi: Rate and outcome. J Vasc Surg 1989; 9:89-97.
7. Raju S, Fredericks R. Venous obstruction: An analysis of one hundred thirty-seven cases with hemodynamic, venographic, and clinical correlations. J Vasc Surg 1991; 14:305-13.
8. Beckwith TC, Richardson GD, Sheldon M, et al. A correlation between blood flow volume and ultrasonic Doppler wave forms in the study of valve efficiency. Phlebology 1993; 8:12-6.
9. van Bemmelen PS, Beach K, Bedford G, et al. The mechanism of venous valve closure. Arch Surg 1990; 125:617-9.
10. Raju S. A pressure-based technique for the detection of acute and chronic venous obstruction. Phlebology 1988; 3:207-16.
11. Neglén P, Raju S. Detection of outflow obstruction in chronic venous insufficiency. J Vasc Surg 1993; 17:583-9.
12. Kistner RL. Surgical repair of the incompetent femoral vein valve. Arch Surg 1975; 110:1336-45.
13. Raju S. Multiple-valve reconstruction for venous insufficiency: Indications, optimal technique, and results. In: Veith FJ, ed. *Current Critical Problems in Vascular Surgery, Vol. 4*. St. Louis: Quality Medical Publishing, Inc, 1992:122-6.
14. Masuda EM, Kistner RL. Long-term results of venous valve reconstruction: A four- to twenty-one-year follow-up. J Vasc Surg 1994; 19:391-403.
15. Raju S. New approaches to the diagnosis and treatment of venous obstruction. J Vasc Surg 1986; 4:42-54.
16. Gruss J. Bypass procedures for venous obstruction: Palma and Husni bypasses, Raju perforator bypass, prosthetic bypasses, primary and adjunctive AV fistulae. In: Raju S, Villavicencio L, eds. *Surgical Management of Venous Disorders*. Baltimore: Williams & Wilkins (In Press), 1994.
17. Husni EA. Venous reconstruction in postphlebitic disease. Circulation 1971; 43 (Suppl 1):147-50.
18. Raju S, Fredericks R. Venous obstruction: Diagnosis and treatment. In: Veith FJ, ed. *Current Critical Problems in Vascular Surgery. Vol. 3*. St Louis: Quality Medical Publishing, Inc, 1991:151-5.