Valve reconstruction procedures for nonobstructive venous insufficiency: Rationale, techniques, and results in 107 procedures with two- to eight-year follow-up

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Among 211 limbs with nonobstructive chronic venous insufficiency, valve reflux of the deep system was the predominant (more than 70%) pathologic condition. Superficial venous or perforator incompetence when present invariably occurred in combination with valve reflux of the deep veins, suggesting that the latter is a common denominator for symptom production. Single level—single system reflux was only occasionally symptomatic (10%), whereas the incidence of single level—multisystem reflux (25%) and multilevel-multisystem reflux (65%) in symptomatic limbs was much higher. Our experience with 107 venous valve reconstructions with a 2- to 8-year follow-up is described. Different techniques of valve reconstruction employed are detailed. The most common pathologic feature is a redundant valve with malcoaptation probably of nonthrombotic origin. Valsalva foot venous pressure elevation is a useful hemodynamic technique for assessing surgical results. Valvuloplasty may be superior to other reconstruction techniques in relieving symptoms of stasis, including stasis ulceration. (J VASC SURG 1988;7:301-10.)

Venous valve reconstruction for chronic venous insufficiency was pioneered by Kistner.^{1,2} Since then, considerable interest and controversy have surrounded the procedure with regard to indications, durability of results, and other aspects.3-5 Since perforator ligation was often combined with valve reconstruction by Kistner, some have questioned whether the symptomatic improvement noted in patients was attributable to the ancillary procedure or the valve reconstruction itself. In addition, ambulatory venous pressure failed to normalize after surgery in many patients, leading some to question the fundamental value of valvuloplasty in improving venous hemodynamics in chronic venous insufficiency. Nevertheless, relief of symptoms after valve reconstruction has now been confirmed by others.^{6,7} In this article we present arguments to support venous valve reconstruction in chronic venous insufficiency, detailing our own experience with more than 150 such procedures.

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MATERIAL

A total of 1378 limbs were evaluated for suspected venous insufficiency in the vascular laboratories of the University of Mississippi Medical Center from 1976 to 1987. Of these nonobstructed venous insufficiency caused by reflux was found in 56% (774 limbs). Surgery was performed on 153 limbs and a variety of valve reconstruction procedures were used, with valvuloplasty predominating. Follow-up greater than 2 years (range 2 to 8 years) was available in 107 cases. The details of venous investigation and the parameters defining venous reflux have been described elsewhere.8-10 Briefly, the venous profile included examination by Doppler probes, photoplethysmography, and a set of ambulatory and other pressure measurements. In the last category, arm-foot venous pressure differential and foot venous pressure induced by reactive hyperemia were used to rule out and grade obstruction.^{9,10} Valsalva-induced foot venous pressure and ambulatory venous pressure were indexes of reflux. In addition, ascending and descending venography, as well as nucleotide descending venography and lymphangiography,¹¹ were performed in 211 limbs in preparation for surgery.

Preoperative evaluation

Patients with obstructive venous insufficiency were not considered for valve reconstruction. How-

Fig. 1. Steps in superficial femoral valve reconstruction. A, Transverse venotomy should be placed over profunda orifice. It can be extended in a caudad direction if needed. B, Technique of valvuloplasty (see text). C, Strip test to confirm competence of repaired valve.

ever, two patients with compensated venous obstruction (grade I)¹⁰ underwent valve reconstruction procedures at the profunda and popliteal levels, respectively. Six cases of valve reconstruction with the use of miscellaneous techniques (segment transfer and saphenous valve transfer, among others) are not included in this report. Stasis ulceration was the primary indication for operation in 73 limbs (68%). Complaints of pain or swelling prompted surgery in the remaining patients.

TECHNIQUES Valvuloplasty

Through a longitudinal incision in the groin the common, profunda, and superficial femoral veins are dissected sharply to minimize venospasm. The location of the uppermost valve of the superficial femoral vein is indicated by a characteristic bulge. A strip test is performed to demonstrate valvular incompetence at this level (Fig. 1). After free valve reflux is confirmed, a transverse venotomy is made at the level of the orifice of the profunda vein in the common

femoral vein, with the patient given systemic heparin. Care is taken to avoid injury to the valve commissures, which can "ride high." Ten to twelve stay sutures of 5-0 polypropylene (Prolene) are placed on the lower lip of the venotomy and weighed down by rubber-shod hemostats, which help in the slow dilatation of the venotomy orifice. With the help of magnification $(2.5\times)$ and good lighting, the valve apparatus is examined for precise identification of the two cusps, the free edges, and the two commissures. In approximately 20% of the cases, a valve apparatus cannot be repaired because of varying degrees of destruction by previous thrombophlebitis. An axillary vein segment transfer should be performed in these instances.

The valve cusp is generally a gossamer-thin membrane with no evidence of previous phlebitis. The edge presents a lettuce-like appearance with multiple folds resulting in eversion and malcoaptation forming the basis of reflux. The valvuloplasty procedure itself involves precise placement of plicating sutures of 7-0 Prolene at the peripheral edge of both valve

Table I. Incidence of superficial, deep, and combined venous insufficiency

	"Pure" superficial insufficiency	"Pure" deep insufficiency	Combination
All symptomatic limbs		<u> </u>	
Descending venography (n = 139)	1% (1)	71% (99)	28% (39)
Ambulatory venous pressure	. ,	, ,	, ,
recovery time* $(n = 211)$	2% (4)	90% (190)	8% (17)
Limbs with stasis ulceration	. ,	` '	, ,
Descending venography $(n = 60)$	0% (0)	88% (53)	12% (7)
Ambulatory venous pressure		` ,	` ',
recovery time $(n = 60)$	3% (2)	87% (52)	10% (6)
Doppler $(n = 80)$	0% (0)	75% (60)	25% (20)

^{*}Recovery time less than 16 seconds. A below-knee tourniquet was used to differentiate superficial, deep, and combined venous insufficiency.

cusps at both commissures. Plication of approximately 20% of the free valve length at either end is usually necessary to achieve a satisfactory result. After repair, the valve edges should appear as a sharp crescent with satisfactory coaptation that can be checked visually by filling the valve sinuses with cold irrigant. After the venotomy is closed by interrupted sutures, the strip test should be performed again to confirm restoration of complete competence to the valve. Occasionally, iatrogenic tears in the valve cusps, misapplication of sutures, or inadequate repair results in persistence of valve incompetence. Under the circumstances, consideration should be given to reopening the venotomy and improving the repair if considered feasible. Substantial competence with minimal leakage may be managed by placing a Dacron jacket around the valve segment (discussed later). Persistent massive leakage on strip test usually requires axillary vein segment transfer or repair of the second superficial femoral valve, if present. The saphenofemoral junction is ligated by 0 silk sutures in continuity before skin closure. Current anticoagulation protocol calls for perioperative minidose heparin, intraoperative heparin (15 U/kg), and warfarin (2.5 to 5 mg usually) started on the first postoperative day to provide for chronic prolongation of prothrombin time of 2 to 4 seconds.

Axillary vein transfer

The technique was initially described in 1981¹² and expanded on subsequently by our group8 and others.¹³ Briefly, a valve-bearing segment of axillary vein is transposed to the superficial femoral vein and ensheathed in a Dacron sleeve (8 or 10 mm) to prevent late dilatation. The valve should be checked in situ for competence by the strip test before transfer, as 30% to 40% of axillary valves in our experience are incompetent (discussed later).

Table II. Incidence of pure perforator incompetence

	Ascending \mathfrak{G} descending venography $(n = 134)$	AVPRT/ Doppler (n = 170)
"Pure" perforator disease Perforator incompetence	3% (4)	2% (3)
associated with deep venous insufficiency	97% (130)	98% (167)

AVPRT = ambulatory venous pressure recovery time.

Dacron sleeve in situ

In 12 cases, the strip test determined the superficial femoral valve to be competent after the vein had gone into spasm with handling imposed by the surgical exposure. Deepening of the valve sinuses and better cusp coaptation with luminal contraction were thought to be the mechanisms responsible for this phenomenon. In these instances, it was considered logical to wrap the valve with a Dacron sleeve (8 or 10 mm, 3 to 4 cm long) to fit the contracted vein and to maintain competence of the valve without subjecting it to a venotomy and valve repair. Migration of the sheath is avoided by securing it to the adventitia of the vein by a few interrupted sutures of 5-0 Prolene.

RESULTS

Rationale for valve reconstruction

"Pure" superficial venous insufficiency is rare in symptomatic patients. The incidence of "pure" superficial venous insufficiency, combined superficial and deep venous insufficiency, and "pure" deep venous insufficiency in the limbs in our study, which used different techniques including descending venography, is detailed in Table I. It is apparent that deep venous reflux either alone or in combination

Table IIIA. Level of reflux

		Descending venography (n = 189)*	Doppler (n = 645)*
Single level, single system		<u> </u>	
Saph		J 1	
SFV }	10%	} 8	Femoral 19% (122)
Prof		l 10	
Single level, multisystem			
SFV/Prof		35	Popliteal 12% (78)
Saph/Prof	25%	∤ 3	• • • • •
Saph/SFV		3	
Saph/SFV/Prof)		6. ا	
Multilevel, multisystem			
Saph/SFV/Pop		42	Femoral and
Prof/Pop		0	popliteal
1	65%	}	combination
SFV/Pop	•	1 1	69% (445)
Calf (via deep veins)		^{\ 80}	\

Saph = saphenous; SFV = superficial femoral vein; Prof = profunda; Pop = popliteal.

Table IIIB. Level of reflux associated with stasis ulcer

		Descending venography (n = 60)*	$Doppler \\ (n = 80) *$
Single level, single system			
Saphenous		(0	
SFV	3%	· { 0	Femoral 6% (5)
Profunda) 2	
Single level, multisystem		. (
Saph/SFV		[2	
SFV/Prof	28%) ₁₀	Popliteal 0% (0)
SFV/Prof/Saph		5	1
Multilevel, multisystem		('	
SFV/Prof/Pop/Calf	69%	41	94% (75)

For abbreviations see Table IIIA.

with superficial venous reflux is the predominant basis of symptoms, including stasis ulceration. Isolated superficial venous insufficiency was an uncommon to nonexistent cause in symptomatic limbs, including those with stasis ulceration.

Perforator incompetence is nearly always a result of deep valve reflux. Currently, there is no definitive test to detect "pure" perforator incompetence. "Pure" perforator incompetence was sought in an indirect fashion: among 232 limbs, perforator incompetence was identified in 73% (170 limbs) on ascending venography performed with an above-ankle tourniquet. The occurrence of associated deep valve reflux was analyzed in 134 of these limbs by means of descending venography. The results are shown in Table II. "Pure" perforator incompetence

could be identified in only 3% of limbs by this analysis.

In another analysis, it was assumed that a certain proportion of patients with abnormal ambulatory venous pressure (not normalized by below-knee tourniquet) had perforator incompetence. The incidence of deep valve reflux as detected by Doppler examination in such a group is shown in Table II. Even allowing that 100% of patients with abnormal ambulatory venous pressure could have had perforator incompetence, the incidence of "pure" perforator incompetence could not have exceeded 2%.

Taken together, these analyses suggest that deep valve incompetence is a common denominator of both superficial venous insufficiency and perforator incompetence. A hypothesis suggesting deep valve incompe-

^{*}No attempt has been made to correlate Doppler with descending venography in this analysis as nonidentical series of limbs were used for analysis of each technique.

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Table IVA. Ambulatory venous pressure measurements in stasis ulcer patients

Pressure after exercise (mm Hg)	No. of ulcer limbs	Total limbs tested	Incidence of ulcers (%)
< 30	0	15	0
30 - 40	8	103	8
40 - 50	14	206	7
50 - 60	30	198	15
60 - 70	16	131	12
70 - 80	10	56	18
80 - 90	6	45	13
90 - 100	3	9	33
> 100	0	0	0

tence as the predominant originator of superficial and perforator insufficiency is discussed later.

Multivalvular or multisystem involvement necessary for stasis symptoms

The distribution of valvular defects in the saphenous, superficial femoral, popliteal, and profunda femoral systems is shown in Tables IIIA and IIIB. In our series of symptomatic patients, multivalvular insufficiency or multisystem reflux appears to be generally necessary for the production of symptoms.

Ambulatory venous pressure and the Valsalva-induced foot venous pressure

The range of ambulatory venous pressure measurements and Valsalva foot venous pressure measurements in the patients operated on is shown in Tables IVA and IVB, respectively. A clear association between increasing pressure after exercise and increased incidence of stasis ulceration was not found. Although the incidence of stasis ulceration was generally lower at the low pressure ranges and higher at the opposite end of the spectrum, stasis ulceration occurred in nearly all the pressure ranges. This suggests that local and other factors may be as important as, or even more important than, venous hypertension in generation of stasis ulceration. The preoperative hemodynamic pressure measurements for Dacron sleeve in situ were better than for the other categories of surgery, suggesting a milder form of reflux. In the cohort of limbs in which stasis ulceration healed after surgery, the mean improvement on postoperative or postexercise pressure was $10.6 \pm 4.9 \text{ mm Hg (n = 29)}.$

A minimal follow-up of 2 years was available for 107 valve reconstruction procedures. Results at 1- and 2-year intervals for the various valve reconstruction procedures are shown in Table V according

Table IVB. Range of Valsalva-induced foot venous measurements in stasis ulcer patients

Normal		Abnor	mal	
0 - 4	5 - 10	11 - 15	16 - 20	> 20
10 18% (10)	25	15 82% (46)	5	1

to breakdown of symptoms. The healing or nonhealing of ulcer was confirmed objectively whereas pain and (to a lesser extent) swelling are of necessity subjective evaluations. Results of valvuloplasty were approximately 20% better than those of axillary vein transfer at all intervals of follow-up. However, this difference was not statistically significant given the small numbers in some groups. Patency of repair in all the patients operated on was monitored by periodic Doppler examination and pressure measurements¹⁰ during the follow-up period. All repairs were patent except as noted under complications.

Stockings

The use of stockings by patients declined postoperatively (Table VIA). Stockings were used postoperatively mostly by patients in whom surgery failed. This is especially clear in the group of limbs operated on for stasis ulceration (Table VIB).

Hemodynamics

Valsalva-induced foot venous pressure and ambulatory venous pressure measurements after surgery are presented in Tables VIIA and VIIB, respectively. There was significant improvement in postoperative values in most categories.

Correlation of hemodynamics with symptoms

Among stasis ulcer patients only, there was a 94% correlation between postoperative Valsalva-induced pressure improvement and healing of ulcer (n = 35). The corresponding figure for ambulatory venous pressure improvement and ulcer healing was 100% (n = 23). However, there was poor correlation (20%) between ambulatory venous pressure and nonhealing of ulcer, that is, patients had ulcer healing despite lack of improvement in ambulatory venous pressure after surgery. With Valsalva-induced foot venous pressure, this negative correlation was good (75%). It is apparent that the Valsalva-induced foot venous pressure had a better correlation with actual symptom status postoperatively. Seventy-five percent of operated limbs had profunda reflux and 25% did

Table V. Operative results

	Postop improvement*			
	Preoperative	0-12 mo	12-24 mo	> 24 mo to present
Valvuloplasty (n	= 61)			
Pain	98% (60/61)	90% (54/60)	87% (52/60)	87% (52/60)
Swelling	98% (60/61)	83% (50/60)	83% (50/60)	83% (50/60)
Ulcer	66% (40/61)	85% (34/40)	75% (30/40)	63% (25/40)
Valvuloplasty/Da	cron sleeve $(\hat{n} = 10)$, ,	, ,	, ,
Pain 1	100% (10/10)	80% (8/10)	60% (6/10)	60% (6/10)
Swelling	100% (10/10)	80% (8/10)	60% (6/10)	60% (6/10)
Ulcer	60% (6/10)	83% (5/6)	50% (3/6)	50% (3/6)
Axillary vein tran		` /	, ,	• • • • • • • • • • • • • • • • • • • •
Pain	89% (16/18)	75% (12/16)	56% (9/16)	50% (8/16)
Swelling	100% (18/18)	66% (12/18)	50% (9/18)	39% (7/18)
Ulcer	72% (13/18)	69% (9/13)	46% (6/13)	46% (6/13)
Axillary vein tran	sfer/Dacron sleeve $(n = 6)$,	, ,
Pain ´	100% (6/6)	83% (5/6)	50% (3/6)	50% (3/6)
Swelling	100% (6/6)	83% (5/6)	50% (3/6)	50% (3/6)
Ulcer	100% (6/6)	100% (6/6)	33% (2/6)	33% (2/6)
Dacron sleeve in			` '	, ,
Pain	100% (12/12)	100% (12/12)	83% (10/12)	83% (10/12)
Swelling	91% (11/12)	100% (11/11)	91% (10/11)	91% (10/11)
Ulcer	66% (8/12)	88% (7/8)	63% (5/8)	63% (5/8)

^{*}For stasis ulceration, improvement was defined as sustained and complete healing of the ulcer.

Table VIA. Stocking use in all operated patients

Preoperative	Postoperative
40/61	25/61
7/10	5/10
16/24	10/24
8/12	4/12
	40/61 7/10 16/24

not. There was no difference in Valsalva-induced and ambulatory venous pressure measurements between the two groups; surgical results were also similar.

Secondary procedures

Eleven patients in the reported group underwent secondary or repeat procedures for recurrence of symptoms during the follow-up period. Of these, seven were treated by a modified Linton procedure, two by axillary vein transfer, and two by saphenous vein stripping for recanalization of the previously ligated saphenofemoral junction.

Other observations

An effort was made during surgery to note signs of previous phlebitis, such as the presence of a fibrotic reaction, abnormal tributaries, recanalized lumen, perforated or dissolved cusps, and the like. Thirty-two percent (n = 63) of operated limbs in which

Table VIB. Postoperative stocking use and surgical outcome in stasis ulceration

Surgical procedure (n = 22)	% Postoperative stocking users with healed ulcers	% Postoperative stocking users with unhealed ulcers
Valvuloplasty (n = 14) Axillary vein transfer (n = 7) Dacron sleeve in situ (n = 1)	18% (4)	82% (18)

this observation was available were thought to have had previous thrombophlebitis. If the axillary vein was explored, a note was made on the status of competence (strip test) of the axillary valve. Forty-four percent of axillary vein valves observed (n=36) were incompetent.

Morbidity and mortality

The mortality rate of the surgical cases was zero. There was a 7% incidence (eight limbs) of deep venous thrombosis with involvement and loss of valve repair in two of eight limbs. In two others the femoropopliteal segment was involved below the level of repair. The remainder (four limbs) had thrombosis limited to the tibiopopliteal area. In two limbs in the last category the site of thrombosis was the unoperated contralateral limb. Other complications included infection in four limbs (two superficial and

Table VIIA. Valsalva-induced foot venous pressure elevation

	$Mean \pm SD (mm Hg)$		
	Preop	Postop	p Value*
Valvuloplasty (n = 40)	7.5 ± 4.3	3.2 ± 1.9	< 0.001
Valvuloplasty with Dacron sleeve (n = 14)†	6.1 ± 3.4	3.1 ± 2.9	< 0.001
Axillary vein transfer $(n = 6)$	11.0 ± 6.2	3.67 ± 2.6	< 0.02
Dacron sleeve in situ $(n = 7)$	6.0 ± 2.6	4.0 ± 2.1	NS

^{*}Student's t test for paired data.

Table VIIB. Ambulatory venous pressure recovery time

	Mean ± SD (sec)		
	Preop	Postop	p Value
Valvuloplasty (n = 50)	9.0 ± 4.3	14.1 ± 4.7	< 0.001
Valvuloplasty with Dacron sleeve (n = 14)†	11.5 ± 5.9	13.3 ± 5.0	<0.02*
Axillary vein transfer $(n = 8)$	6.8 ± 2.9	11.0 ± 5.2	< 0.02
Dacron sleeve in situ $(n = 8)$	13.8 ± 5.0	16.6 ± 4.2	NS

^{*}Data from four limbs with normal preoperative time excluded from calculation of p value. †Includes data with follow-up of less than 2 yr.

two deep), wound seroma or hematoma in four, and lymphocele in one.

Lymphangiography

There were eight instances (n = 36) of abnormal lymphangiographic findings in patients who underwent valve reconstruction procedures. Two of these showed obstruction at the inguinal level, and the remainder showed delay of lymphatic drainage as denoted by late uptake of the nucleotide at the inguinal level. Successful relief of symptoms and cure of stasis ulceration in all limbs were apparently not jeopardized by this abnormal lymph drainage.

DISCUSSION

The pathophysiology of chronic venous insufficiency is poorly understood. Venous insufficiency is traditionally classified into superficial and deep varicosities according to the system involved. It is generally presumed that the former is of congenital origin and the latter is postthrombotic in origin. There has been a great deal of speculation regarding the origin of superficial incompetence, and many theories¹⁴⁻²⁰ have been proposed. The more popular ones14,16,17 postulate ascending or descending varicose formation from primary saphenofemoral or perforator incompetence although it appears implausible that a single or a localized group of valves should become primarily incompetent whereas other valves subjected to similar or worse hemodynamic loads should be unaffected.

It is widely believed that the deep system is normal in primary varicosity and plays no part in its etiology.²¹ Nevertheless, some features of varicosities have tended to implicate an abnormal deep system. It has been known for some time that ambulatory venous hypertension is often associated with primary varicosities and does not normalize despite saphenous vein stripping, or perforator ligation (Linton procedure). The high recurrence rates, despite extensive ablative procedures of the perforator and saphenous system, are also suggestive of an inherently abnormal deep system in these limbs. Our data clearly show that in symptomatic limbs examined by descending venography,22 the deep system is almost always abnormal (i.e., refluxive). Other authors^{9,23} have also recently emphasized the presence of abnormal deep systems in chronic venous insufficiency. The terms "primary" and "secondary" as well as "superficial" and "deep" venous insufficiency should perhaps be abandoned in view of the interdependent relationship between these entities. The occurrence of saphenous or perforator incompetence invariably in association with deep system reflux implies a leading and probably an etiologic role for deep valvu-

[†]Includes data with follow-up of less than 2 yr.

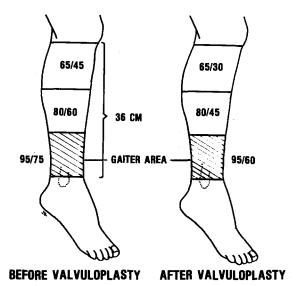


Fig. 2. This diagram illustrates how a modest decrease of 15 mm Hg in blood pressure after exercise reverts the ulcer bed to a pressure range that could have existed above the gaiter area preoperatively. Pressure calculations are based on hydrostatic pressure differences in a leg 36 cm in length.

lar reflux in the origination of superficial insufficiency. This concept supports the practice of venous valve reconstruction for most venous insufficiency syndromes.

The most frequent pathologic condition encountered in valve reconstruction is the redundant valve cusp and resultant lack of effective coaptation. It appears unlikely that previous phlebitis would be the etiologic basis for this type of structural abnormality. In most of the operated limbs there was no evidence of previous phlebitis in venography or during direct inspection at surgery. We have presented detailed evidence elsewhere8 to support the argument that the lesion is developmental or congenital rather than postphlebitic. Bauer²⁴ and Kistner² have made similar observations. Furthermore, we9 and others25 have noted that previous cases of proven deep venous thrombosis followed up to 7 years or more display a clinical and hemodynamic profile distinctly different from the experience reported here.

A major criticism of deep venous valve reconstruction has been the failure to normalize the extant ambulatory venous hypertension.³ In most patients who show symptomatic improvement, even by objective healing of stasis ulceration, a 10 mm Hg improvement in pressure after exercise is the norm. Fig. 2 illustrates how this modest improvement in post-exercise pressure may be adequate for ulcer healing. An alternative explanation may be that ambulatory venous hypertension may not be so important

as hitherto believed in the genesis of stasis ulceration. Certainly, in our material (Table IV) a wide range of exercise pressures from high to low was found in stasis ulceration. Other factors such as the velocity of reflux (the water hammer effect) and the extent of reflux through low-resistance large-bore veins may be relatively more important. A repaired superficial femoral valve providing impedance to the above-mentioned mechanisms may be responsible for ulcer healing. Apropos, the Valsalva-induced foot venous pressure measurement may be a better index of reflux than the traditional ambulatory venous pressure measurement.

It has been stated that grade I reflux by Kistner's criteria²² (reflux up to lower thigh) is not a symptomatic lesion. Our data indicate that this degree of femoral reflux is associated with significant symptoms if the profunda or saphenous systems are also refluxive. Eriksson and Almgren⁶ have recently pointed out the importance of profunda reflux. It is our experience that the profunda system assumes additional importance if the superficial femoral venous system is compromised by obstruction or recanalization. Implicit in our surgical approach is the concept that the dilated perforators will revert to a competent state with time if the underlying deep valve abnormality is corrected. Ferris and Kistner²⁶ have shown that concomitant perforator ligation with valvuloplasty can rapidly restore a symptomatic limb to near normalcy. Such a combined approach may be appropriate in some patients with extensive "blow-outs."

Some authors have suggested that the good results of valve reconstruction may be a result of the increased awareness of the patient and physician with regard to the subject limb and the resultant better conservative care bestowed on it postoperatively, such as the use of support stockings. Our data indicate that the proportion of patients using stockings is less postoperatively than before and the stocking-wearers are usually those in whom valve reconstruction had failed to relieve symptoms.

REFERENCES

- Kistner R. Surgical repair of a venous valve. Straub Clin Proc 1968:24:41.
- Kistner RL. Surgical repair of the incompetent femoral vein valve. Arch Surg 1975;110:1336.
- Strandness DE Jr, Thiele BL. Selected topics in venous disorders. New York: Futura, 1981:186.
- Moore DJ, Himmel PD, Sumner DS. Distribution of venous valvular incompetence in patients with the postphlebitic syndrome. J VASC SURG 1986;3:49.
- Raju S. Distribution of venous valvular incompetence in patients with the postphlebitic syndrome (Letter). J VASC SURG 1986;4:536.

- 6. Eriksson I, Almgren B. Influence of the profunda femoris vein on venous hemodynamics of the limb. J VASC SURG 1986:4:390.
- 7. Taheri SA, Pendergast DR, Lazar E, et al. Vein valve transplantation. Am J Surg 1985;150:201.
- 8. Raju S. Venous insufficiency of the lower limb and stasis ulceration. Ann Surg 1983;197:688.
- 9. Raju S, Fredericks RK. Late hemodynamic sequelae of deep venous thrombosis. J VASC SURG 1986;4:73.
- 10. Raju S. New approaches to the diagnosis and treatment of venous obstruction. J VASC SURG 1986;4:42.
- 11. Patel BR, Burkhalter JL, Patel RB, Raju S. Interstitial lymphoscintigraphy for diagnosis of lymphocele. Clin Nucl Med 1985:10:175.
- 12. Raju S. Discussion after Johnson ND, Queral LA, Flinn WR, Yao JST, Bergan JJ. Late objective assessment of venous valve surgery. Arch Surg 1981;116:1461.
- 13. Taheri SA, Lazar L, Elias S, et al. Surgical treatment of postphlebitic syndrome with vein valve transplant. Am J Surg 1982;144:221.
- 14. Hobbs JT. The treatment of venous disorders. Philadelphia: JB Lippincott, 1977:195.
- 15. Bjordal RI. Pressure patterns in the saphenous system in patients with venous leg ulcers. Acta Chir Scand 1971;137:495.
- 16. Linton RR. John Homans' impact on diseases of the veins of the lower extremity with special reference to deep throm-

- bophlebitis and the post thrombotic syndrome with ulceration. Surgery 1977;81:1.
- 17. Fegan WG, Kline AL. The cause of varicosity in superficial veins of the lower limb. Br J Surg 1972;59:798.
- 18. May R, Partsch H, Straubesand J. Perforating veins. Baltimore: Urban and Schwarzenberg, 1981:3-58.
- 19. Ludbrook J. Primary great saphenous varicose veins revisited. World J Surg 1986;10:954.
- Rose SS, Ahmed A. Some thoughts on the aetiology of varicose veins. J Cardiovasc Surg 1986;27:534.
- 21. Dood H, Cockett FB. The pathology and surgery of the veins of the lower limb. Edinburgh: Churchill Livingstone, 1976.
- 22. Kistner RL, Ferris EB, Randhawa G, Kamida O. A method of performing descending phlebography. J VASC SURG 1986;4:464.
- 23. Johnson G Jr. In: Dale WA, ed. Management of vascular surgical problems. Invited commentary. New York: McGraw-Hill, Inc, 1985:458.
- 24. Bauer G. The etiology of leg ulcers and their treatment by resection of the popliteal vein. J Int Chir 1948;8:937.
- 25. Lindner DJ, Edwards JM, Phinney ES, et al. Long-term hemodynamic and clinical sequelae of lower extremitiy deep vein thrombosis. J VASC SURG 1986;4:436.
- 26. Ferris EB, Kistner RL. Femoral vein reconstruction in the management of chronic venous insufficiency. Arch Surg 1982;117:1571.

DISCUSSION

Dr. Robert L. Kistner (Honolulu, Hawaii). This is a large series of patients with chronic venous insufficiency; 1378 limbs were evaluated. Eleven percent of the total series ultimately had surgical deep vein reconstruction, whereas 75% of those with severe reflux disease were operated on.

The extensive venous pressure studies that were reported here have shown a poor correlation between venous pressure studies and the clinical state. There is a small but statistically significant improvement in the venous pressure recovery time after proximal valve repair. This correlation is not very strong. The venous pressure Valsalva test as described by Dr. Raju bears corroboration because it appears to correlate well with the results of deep venous repair.

Dr. Raju's supravalvular approach to valvuloplasty is a good approach and I commend it as an alternative technique for those who plan to do this type of surgery.

The design of this series tests the authors' hypothesis that deep system incompetence is basic to all incompetence in the leg, including saphenous, perforator, and distal deep veins. There are six series in the literature but they are not completely comparable. This series looks at treating deep veins and greater saphenous veins but not the perforating veins. The series of Johnson did not treat perforator or greater saphenous veins at all. Eriksson in Sweden treated deep veins only after the perforator and greater saphenous

veins had been treated and that treatment failed; the same for Hughes in Boston. The series of Taheri and ours treated the perforator and greater saphenous veins selectively in conjunction with deep vein repair.

This study then is unique and provides the opportunity to see whether deep valve repair with greater saphenous vein ligation will suffice for treating patients with complicated degrees of deep vein disease.

Dr. Raju, can you elaborate on the cause of recurrence in those patients whose ulcers did return? Would the earlier use of peripheral varicose vein procedures or perforator interruptions have made a material difference in your results? In addition, your results are best with valvuloplasty. Under what circumstances would you advocate axillary vein transplantation?

Dr. David S. Sumner (Springfield, Ill.). Of the physiologic tests that you employed for evaluating venous valvular incompetence, the Valsalva-induced foot pressures correlated most closely with the operative results. As I understand this measurement, it is performed with the patient supine. I submit that the results of this test bear little relationship to the severity of the physiologic aberrations responsible for severe stasis changes, which are generally thought to be a function of ambulatory venous hypertension. Only in the upright position do the adverse effects of hydrostatic pressure come into play.

One would surmise that Valsalva-induced foot pressures would give normal values anytime there is a competent valve anywhere in the leg. In other words, the test would fail to detect below-knee valvular incompetence if one or more proximal valves were competent. Could you clarify the rationale for using this measurement? In addition, did you evaluate the patients in other ways for below-knee valvular incompetence or make an effort to segregate limbs with below-knee disease from those with incompetence confined to the proximal veins? I ask these questions because there is some evidence that distal valvular incompetence is more important than proximal valvular incompetence in the genesis of stasis changes.

Dr. Syde A. Taheri (Buffalo, N.Y.). Our findings during phlebography have shown that the incidence of primary superficial venous insufficiency is extremely low and is usually due to deep venous incompetence. Although noninvasive tests are used for screening, we heavily depend on ambulatory venous pressure as well as ascending and descending phlebography.

In 40 patients with venous insufficiency, ambulatory venous pressure and recovery time were abnormal. The close relationship of venous hypertension and muscle atrophy and abnormal nerve conduction velocity has also been an interesting finding.

It is postulated that with increased capillary and compartment pressure the resulting edema will cause a diffusion barrier for oxygen transport and ensuing chronic ischemia. This will generate oxygen-derived free radicals, which are known to play a prominent role in several aspects of the pathophysiology of neuromyopathy.

In a 7-year follow-up of 40 patients with vein valve transplant or reconstruction, three patients had dilatation and one graft has been occluded. The postoperative venous pressure reduction has been only 15%. Dr. Raju, do you think the low reduction of venous pressure may be due to severe neuromyopathy in these patients?

Dr. Harry Schanzer (New York, N.Y.). We have analyzed all our cases, more than 600, with ascending and retrograde phlebography and surgery of the deep veins and have come to the same conclusions as Dr. Raju. About 66% of our cases with chronic venous stasis do not have any evidence of previous phlebitis in their deep veins. The valves are completely free of chronic inflammation or other evidence of phlebitis. On this basis, I strongly believe that the denomination "postphlebitic syndrome" is a misnomer. This entity should be called "chronic venous stasis syndrome." Only one third of them are postphlebitic in origin.

The second comment has to do with the pathophysiology of this so-called primary valvular incompetence. Dr. Kistner postulated that this was due to very elongated or lax leaflets in the valves. I have an alternate explanation. It is possible that the venous wall is weak and tends to become dilated; this produces a dilatation of the valvular ring and secondary incompetence. On the basis of this hypothesis we have begun to do some repairs of the valves by splinting the vein with a rigid membrane such as polytetrafluoroethylene. Postoperative long-term phlebography has shown competence and patency. This procedure seems to

be similar to the 10 cases that had the incompetent valve surrounded by Dacron reported by Dr. Raju.

What were the indications for your surgery? Second, I would like to know how many of your patients had a postoperative phlebogram to demonstrate competence of the repaired valve. Third, I would like to know a little more about the results of your Dacron cuffing of the valve.

Dr. Raju (closing). Dr. Kistner, I agree with you that the perforator component, although secondary to deep valve reflux, may require separate treatment either at the same time or at a later time. Implicit in our approach is the belief that correction of primary deep system reflux will in time resolve secondary perforator reflux, but this may not be true. We really do not know the answer, and perforator surgery may be required in some of these patients.

We studied the causes of surgical failure with venography and with various laboratory parameters. In two instances, the cause of recurrence was a recanalized saphenous vein after saphenofemoral ligation even though the valve repair was intact. The saphenous vein was stripped and the ulcer healed in both instances. Other causes of failure have included inadequate valvuloplasty, recurrent reflux through the repaired valve, venous obstruction caused by deep vein thrombosis, or a small axillary vein segment. In some instances, despite detailed investigation, the cause of failure was not apparent. Our approach to these cases is to correct the specific elements of recurrent reflux identified. The Linton procedure, which in my view temporarily disconnects the deep system from the superficial system, has been used when a repeat valvuloplasty or axillary vein transfer has not been possible. In our initial experience, poor case selection was likely a factor in ulcer recurrence after valve reconstruction. Our present ability to exclude cases of venous obstruction on the basis of arm-foot pressure differential and reactive hyperemia techniques has resulted in better case selection.

Dr. Sumner, the Valsalva-induced foot venous pressure certainly improves with single valve repair. In addition, it tends to correlate well with the postoperative symptom status of the patient. Ambulatory venous pressure has not correlated well with surgical results. In fact, there is not a good correlation between stasis ulceration and ambulatory venous hypertension in our material preoperatively. I know of one other series published by Dr. Nicolaides in which he showed a correlation between ambulatory venous pressure and ulceration; but his study comprised both obstruction and reflux. Surgical procedures such as saphenous vein stripping and perforator ligation do not materially change ambulatory venous pressure, despite symptomatic improvement postoperatively. The precise relationship between ambulatory venous pressure and stasis ulceration is not clear. There is much work to be done in this area.

Dr. Taheri, we are of course closely following your work with neuromyopathy and various effects of ambulatory venous pressure on muscle function.

Dr. Schanzer, the Dacron sleeve is an appropriately sized sleeve around the vein to provide competence without inducing obstruction.