Diameter-reflux relationship in perforating veins of patients with varicose veins

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Purpose: **Treatment of chronic venous valvular insufficiency requires understanding of the hemodynamics of perforating veins. To preserve normal veins or veins that can function normally once primary sources of valvular insufficiency are removed, a better understanding of the diameter-reflux relationship is desirable. We measured reflux and diameters in 500 perforating veins of patients with varicose veins ($C_2E_PA_{SP}P_R$).**

Methods: **Color flow duplex ultrasonography scanning was performed with the patient standing. Perforating veins were mapped medially in the thigh and medially, laterally, and posteriorly in the calf. Reflux was defined as reverse flow that lasted longer than 0.5 seconds. Diameters were measured on B-mode transverse projections at the crossing of the fascia. Competent versus incompetent vein diameters were compared by means of Student *t* test, one-way analysis of variance, and Bonferroni *t* test.**

Results: **Diameters of competent and incompetent perforators averaged 2.5 ± 0.9 mm (n = 17) and 4.7 ± 1.9 mm (n = 17) at the medial thigh ($P < .0002$), 2.2 ± 0.8 mm (n = 179) and 3.7 ± 1.0 mm (n = 210) at the medial calf ($P < .0001$), 2.2 ± 0.6 mm (n = 13) and 3.5 ± 0.8 mm (n = 37) at the posterior calf ($P < .0001$), and 2.1 ± 0.8 mm (n = 9) and 3.3 ± 0.7 mm (n = 18) at the lateral calf ($P < .003$), respectively. Perforating vein diameters of 3.5 mm or larger in the calf and thigh were associated with reflux in more than 90% of the cases.**

Conclusion: **An enlargement in the diameter of the perforating veins of 1 to 1.5 mm in the calf or 2 mm in the thigh of patients with varicose veins could be the difference between normal flow and reflux. Further studies are needed to confirm if elimination of reflux in patients with primary varicosity will transform incompetent perforators to competent ones. (J Vasc Surg 1999;30:867-75.)**

A variety of surgical procedures have been designed to correct varicose veins, including total or partial stripping of saphenous veins, selective ligation of sources of venous blood flow reflux, and segmental preservation of superficial veins.[1-10] Enlarged perforating veins have been ligated or stripped. The advent of subfascial endoscopic perforator surgery has reawakened discussion about the role of perforating veins in the pathogenesis of chronic venous insufficiency.[11-19] Ligation of perforating veins has been questioned.[13,20] In the treatment of varicose veins, aesthetic and functional considerations may be conflicting. Risk of an unacceptable aesthetic result is diminished when perforating vein ligation or stripping is avoided or limited. In contrast, presence of insufficient perforating veins increases the risk of varicose vein recurrence, particularly when the saphenous vein is preserved. This dilemma is accentuated when future cardiac or infrainguinal arterial bypass grafting with a saphenous vein is contemplated.

Color flow duplex ultrasonography (US) scanning has facilitated and amplified preoperative evaluation.[5,7,21-30] Not only the deep and superficial veins can be evaluated for thrombosis,[31] obstruction, or valvular insufficiency with significant reflux, but also patterns of reflux can be identified. The femoral junction, branches, tributaries, and perforating veins are being classified as sources for drainage of reflux.[7,32,33] Elimination of the principal sources of reflux has resulted in decreased diameters of preserved veins.[7,34] This decrease in diameter may revert valvular insufficiency. These observations must be further analyzed to improve anatomofunctional interpretation of the perforating venous system. The aim of this study was to correlate diameter

From Angiolab and Associates.
Presented at the Eleventh Annual Meeting of the American Venous Forum, Dana Point, Calif, Feb 18–21, 1999.
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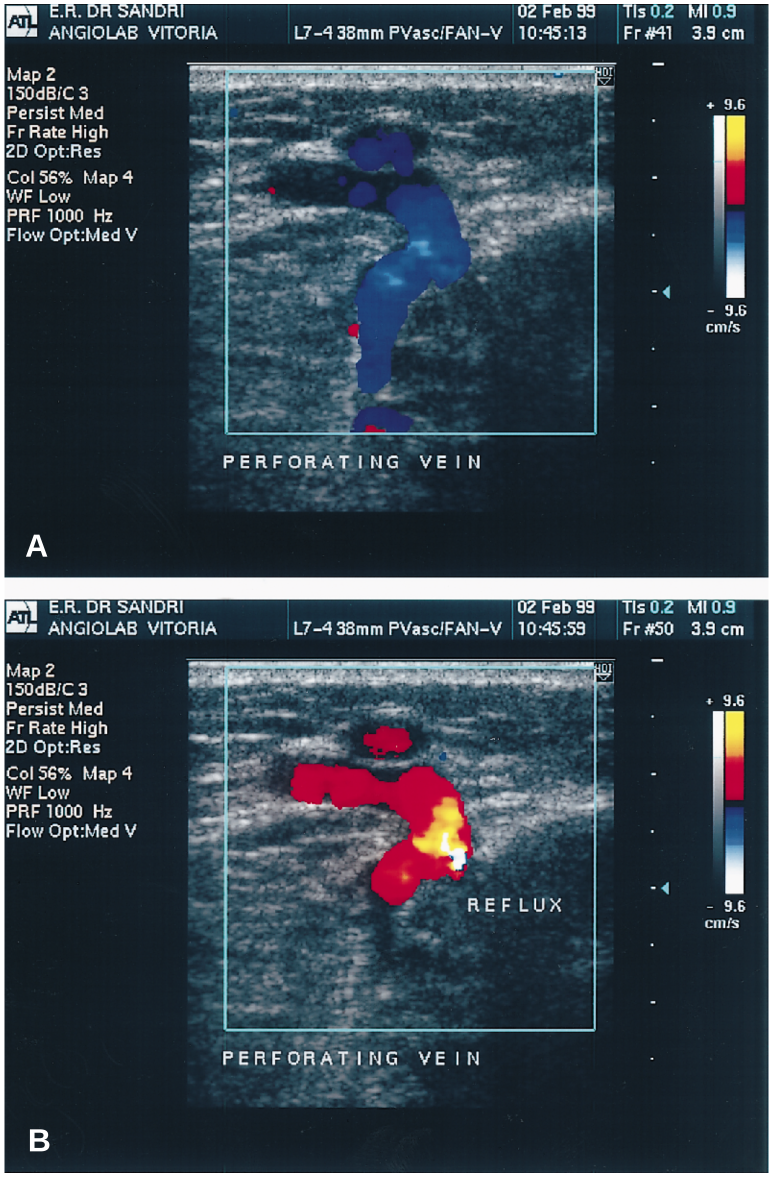

Fig 1. Color flow duplex ultrasound scan of a medial calf perforating vein. Diameter was measured as the vein traversed the fascia. Flow is in the superficial-to-deep vein direction on the top and deep-to-superficial vein direction on the bottom (reflux).

and functional status of perforating veins in a select patient population being treated for varicose veins. This information may improve selection of veins that should be treated or might be preserved.

METHODS

US was performed in patients who were candidates for surgical treatment of varicose veins. Patients were entered in the study until 500 perforating veins were evaluated. Data from 116 limbs of 78 patients, 63 women (81%) and 15 men (19%), were entered. According to the Clinical, Etiologic, Anatomic, and Pathophysiologic (CEAP) classification recommended by the American Venous Forum,[35-38] all limbs were in the clinical class C_2–varicose veins. Eleven limbs (9.5%) had edema–class C_3. No patients were class C_4–significant skin changes. No patients were class C_5–healed ulceration or class C_6–active venous ulceration. Etiologic classification placed all patients in the primary E_P group. There were no patients with congenital, known thrombotic, or traumatic causes of venous insufficiency. In all limbs, the greater saphenous vein was affected with reflux noted above the knee, below the knee, or both (A_S). Deep venous insufficiency (A_D) was noted in 11 limbs (9.5%). The A_P perforating vein anatomic classification is the primary objective of this work and is described in detail in the results section. The pathophysiologic classification was P_R, with all limbs having reflux but no venous obstruction. Complaints of moderate pain were associated with 60 limbs (51.7%) requiring analgesics (clinical score 2) and with 37 (31.8%) limbs not requiring analgesics (clinical score 1). Fifty-six limbs (48.2%) were asymptomatic, and 60 extremities (51.7%) were symptomatic but functioning without support device. In this series, there were no patients who were incapacitated for work.

Standard US techniques were used to evaluate deep, superficial, and perforating veins.[5,7,21,22,26,29,31] A US scanner equipped with a 7.5-MHZ transducer was used in all cases (ATL HDI 3000, Advanced Technology Laboratories, Bothell, Wash). The test was performed with the patient standing. Femoropopliteal veins were evaluated for thrombosis, chronic obstruction, or significant valvular insufficiency with reflux. In addition, sources and drainage of reflux were defined for the greater and lesser saphenous veins.[7,32,33] Furthermore, perforating veins were detected at the medial aspect of the thigh and at the medial, lateral, and posterior aspects of the leg. All thigh perforators included in this study had direct communication with the great saphenous vein. Distance from the base of the foot was determined for each perforator encountered. Measurement of the perforating vein diameter was performed in a B-mode transverse section at the site at which the vein perforated the fascia (Fig 1). Perforating flow was analyzed with color flow and Doppler spectral analysis in longitudinal section, during compression of the leg above and below the level of the perforator being examined. Manual compression was performed to provide flexibility in the evaluation of veins with varied anatomical configurations. Significant perforating vein reflux was defined as reverse flow lasting longer than 0.5 seconds.

Average diameters of competent and incompetent perforators at the medial aspect of the thigh and the medial, posterior, and lateral aspects of the leg were compared by means of Student *t* test, one-way analysis of variance (ANOVA), and Bonferroni *t* test. The Microsoft Excel program Student *t* test, two-tail, two-sample, unequal variance (heteroscedastic) was used to calculate the *P* values shown in Table I. These statistics were also used to compare average longitudinal positions of perforating veins.

RESULTS

There were 282 (56.4%) incompetent perforators in this sample population. Among the incompetent perforators, most were located medially in the calf (210; 74.5%); the others were located posteriorly in the calf (37; 13.1%), laterally in the calf (18; 6.4%), or medially in the thigh (17; 6.0%). Most competent perforators were also located medially in the calf (179; 82.1%); the remainder were located posteriorly in the calf (13; 6.0%), laterally in the calf (9; 4.1%), or medially in the thigh (17; 7.8%).

The average longitudinal location in the thigh, measured as the distance from the popliteal crease, was similar for competent (17.1 ± 9.9 [SD] cm) and incompetent (21.0 ± 16.4 cm) perforating veins (P = .63). Ranges for competent and incompetent perforating veins in the thigh were 8 to 51 cm and 5 to 60 cm, respectively. Average longitudinal location in the calf, measured as the distance from the base of the foot, was similar for competent posterior perforators (28.8 ± 6.2 cm) and incompetent posterior perforators (28.5 ± 6.6 cm; P = .88), competent lateral perforators (24.2 ± 4.0 cm) and incompetent lateral perforators (24.1 ± 6.2 cm; P = .93), and competent medial perforators (21.9 ± 6.4 cm) and incompetent medial perforators (22.7 ± 6.7 cm; P = .23). Medial perforators were found at a more distal location than posterior perforators by means of one-way ANOVA with Bonferroni *t* test (P < .001). Ranges for competent and incompetent perforating veins were 18 to 37 cm and 15 to 36 cm for posterior calf perforating veins, 18 to 30 cm and 15 to 35 cm for lateral calf perforating veins, and 10 to 39 cm and 7 to 42 cm for medial calf perforating veins, respectively.

Table I shows the average diameters obtained for competent and incompetent perforators in the thigh and calf. No statistically significant difference in the diameter of competent perforators in the four locations was shown by means of one-way ANOVA with Bonferroni *t* test (P = .49). By means of similar testing, incompetent thigh perforators were demonstrated to have larger diameters than calf perforators (P < .05). In the thigh, perforating vein diameter of 3.5 mm or larger was predictive of reflux in 92% of the cases (12 of 13 cases); diameters smaller than 3 mm predicted lack of reflux in 81% of the cases (13 of 16 cases), and all 10 perforating veins with a diameter smaller than 2.5 mm were competent. Figs 2 and 3 show the cumulative and interval reflux positive- and negative-predictive values as a function of calf perforating vein diameter. Calf perforating vein diameter of 3.5 mm or larger was predictive of reflux in 90% of the cases (137 of 152 cases); vein diame-

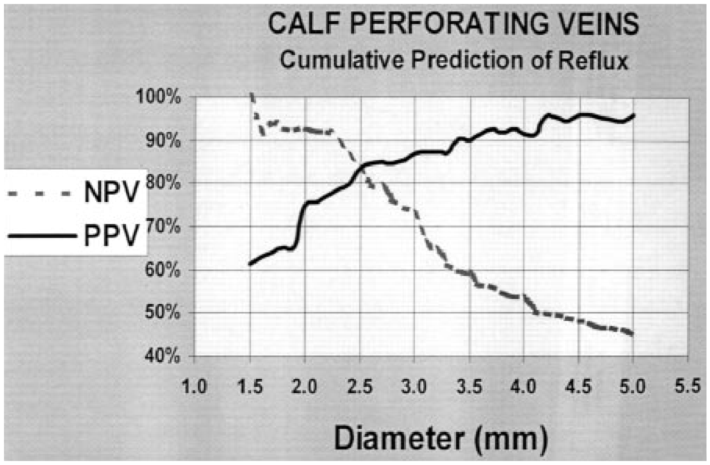

Fig 2. Cumulative predictive values for reflux in calf perforating veins. Cumulative negative- and positive-predictive values were calculated for diameters smaller than or larger than that plotted at the horizontal axis, respectively. *NPV,* negative-predictive value; *PPV,* positive predictive value.

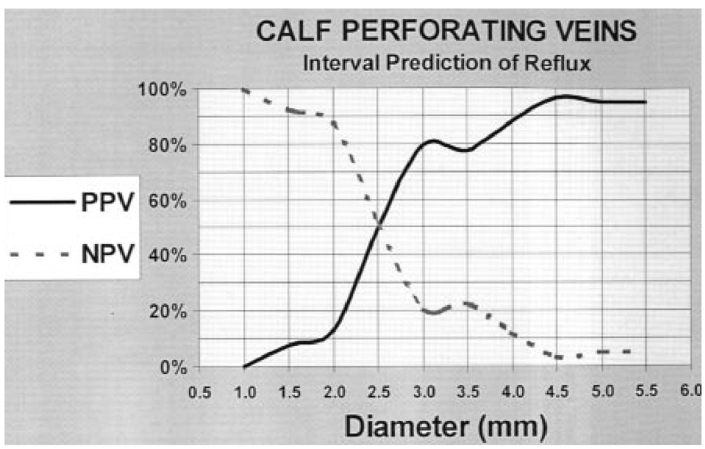

Fig 3. Interval predictive values for reflux in calf perforating veins. Interval positive- and negative-predictive values were calculated for 0.5 mm ranges around the center values plotted. *NPV,* negative-predictive value; *PPV,* positive-predictive value.

ter smaller than 2.2 mm was predictive of lack of reflux in 92% of the cases (119 of 130 cases).

DISCUSSION

Many reports have described the role of incompetent perforating veins as the cause of primary, persistent, or recurrent varicose veins.[39-48] Patients are concerned with the aesthetic results of varicose vein surgery. Better aesthetic results could be achieved if fewer perforating veins were ligated. In planning surgery for varicose veins, we also consider saphenous vein preservation for future coronary or infrainguinal artery bypass grafting procedures. Guidelines suggesting which perforators should be ligated are lacking. In an attempt to formulate quantitative guidelines, we considered that a significant number of perforating veins have reflux caused by venodilation with diameter enlargement that renders the valves incompetent. If the primary source of reflux is eliminated, perforating vein diameter may diminish and the valves may regain competency. Therefore, as a first step, we investigated the prevalence of reflux as a function of perforating vein diameter in a homogeneous patient population classified as clinical class C_2, according to the CEAP classification endorsed by the American Venous Forum.[35-38]

Table I. Average diameters obtained for competent and incompetent perforators in the thigh and calf

Location	*Diameter*		
	Competent perforators	*Incompetent perforators*	*P value*
Medial thigh	2.5 ± 0.9 mm (n = 17)	4.7 ± 1.9 mm (n = 17)	< .0002
Medial calf	2.2 ± 0.8 mm (n = 179)	3.7 ± 1.0 mm (n = 210)	< .0001
Posterior calf	2.2 ± 0.6 mm (n = 13)	3.5 ± 0.8 mm (n = 37)	< .0001
Lateral calf	2.1 ± 0.8 mm (n = 9)	3.3 ± 0.7 mm (n = 18)	< .003

We performed perforating vein diameter measurements and reflux detection by means of color flow duplex US scanning. This noninvasive test has become the practical standard for lower-extremity venous evaluation.[5,7,25-27,31-33,44-46,48-49] The diagnosis of deep venous thrombosis with US scanning is well established.[31] Many US studies have been directed toward the diagnosis of chronic venous insufficiency and the relation of perforating veins to venous stasis ulceration or less-severe complications. Unlike venography, the US test can be repeated without risk and readily provides functional flow evaluation, including the evaluation of perforating veins before varicose vein surgery or even subfascial endoscopic perforator surgery.

There were no unusual perforating vein anatomical findings in the patient population studied. Most perforating veins were found in the medial aspect of the calf. These veins communicated primarily to the posterior arch of the greater saphenous vein in the leg. Virtually all the posterior calf perforating veins communicated with the lesser saphenous vein. All the lesser saphenous vein perforators communicated either with a muscle vein or with one of the major deep veins. Lateral perforating veins had complex connections with either the greater or lesser saphenous veins. Thigh perforators communicated with the greater saphenous vein.

We opted for a commonly used criterion to determine reflux in perforating veins, based on a duration of reverse flow of greater than 0.5 seconds. Manual compression was used to elicit reflux. This maneuver improves flexibility in evaluating veins with different anatomical orientations. The appropriate quantification of reflux is still being scrutinized. Veins with abrupt closure of reverse flow were considered competent, regardless of reverse flow duration. Reflux severity may depend on a complex interaction involving time, blood velocity, and blood volume. The criterion used is simple and can be reproduced easily.

The availability of US testing before varicose vein surgery has fomented analysis of reflux patterns and diameter-reflux relations.[7,32,33,49] Engelhorn et al published data showing that the average diameter of incompetent greater saphenous veins was 2 mm wider than that of competent saphenous veins at the femoral junction (7.7 vs 5.7 mm) and midthigh (5.5 vs 3.3 mm) and 1 mm wider at midcalf (3.5 vs 2.5 mm).[49] These authors also indicated that a greater saphenous vein diameter wider than 8 mm, 6 mm, and 4 mm at the femoral junction, midthigh, and midcalf, respectively, was predictive of reflux with near certainty. On the other side of the spectrum, diameter smaller than 5 mm, 3 mm, and 2 mm were associated with lack of reflux at the same three respective levels. These guidelines may be used to help make the decision for stripping or segmental preservation of the greater saphenous vein.

The prevalence of incompetent perforating veins in class C_2 limbs in this series matched the data presented by Delis et al with one variance: we detected more incompetent perforators in the posterior and lateral aspects of the calf than medially in the thigh.[47] Otherwise, most incompetent perforators were detected medially in the calf in both investigations. Posterolateral thigh perforator vein incompetence must be recognized for the treatment of individual patients, but its prevalence is low;[46] therefore, cases with this condition were not included in this analysis.

The diameters of competent perforating veins measured in this series of patients with varicose veins, averaging 2 to 2.5 mm, were similar to those described by Labropoulos et al (average, 2 mm for class C_2; increasing to 2.6 mm for class C_6) and Hanrahan et al for competent perforating veins in patients with venous stasis ulcers.[48,23] These values were similar to the diameters of competent greater saphenous veins at midcalf.[49] Such diameters, however, may not be considered normal; in a control group of healthy volunteers studied by Hanrahan and co-workers, the average diameter of competent perforators was 1.4 mm.[23] Furthermore, the diameters of competent perforators increase with the severity of chronic venous disease.[48] Although valves may become incompetent when under physiological stress, they can still be functional within certain limits of venodilatation.[50] From this data, we raised the hypothesis that perforating veins can dilate at least 1 mm without becoming incompetent.

The average diameter of incompetent perforating veins was smaller in limbs with varicose veins, as described in this work, than in limbs with venous ulcers, as described by Hanrahan (3.5 vs 4.4 mm).[23] Our data confirmed those previously described by Labropoulos et al for class C_2; these authors also confirmed that incompetent perforators in limbs with skin changes and healed or active ulcers had diameters averaging 4.4 to 4.5 mm.[48] We demonstrated that incompetent perforating vein diameters were larger in the thigh than in the calf. Nevertheless, diameters larger than 3.5 mm were predictive of reflux with virtual certainty in the entire lower extremity. In a population that also included patients who had limbs with skin changes and ulcers, Labropoulos et al indicated that diameters larger than 3.9 mm was reliably predictive of reflux.[48] In a similar population of patients with varicose veins, the average diameter of incompetent greater saphenous veins also averaged 3.5 mm in the calf.[49] It is probable that perforating veins, acting either as the source or drainage of saphenous vein reflux, have matching diameters as a continuum.

A significant decrease in the diameter of greater saphenous veins after ligation at the femoral junction has been described.[7] The diameter of perforating veins can also be reduced postoperatively. Stuart et al demonstrated a 1-mm reduction of median perforating vein diameter, from 4 to 3 mm, after saphenous vein ligation and/or stripping and multiple phlebectomies.[34] These authors also showed that incompetent perforating veins continue to be detected after surgery in the presence of deep vein reflux or failure to abolish the main stem superficial venous reflux. In this study population, less than 10% of the limbs had detectable deep vein reflux. Anecdotally, as we have monitored patients after varicose vein surgery, we have observed that the diameter of deep veins can diminish and deep vein reflux can disappear. Patterns of reflux must be studied in detail before we can predict the appropriate link between deep vein and perforating vein refluxes in individual patients.

A relationship between patterns of reflux and the diameter of greater saphenous veins has been demonstrated.[32] Diffuse reflux from femoral junction to ankle formed a group of greater saphenous veins with the largest average diameter. Next in size were veins with proximal reflux from the femoral junction to a branch or perforator above or below the knee. Veins with segmental reflux starting distal to the femoral junction and ending proximal to the ankle had larger diameters than veins with reflux limited to their portion at the distal calf. All groups of veins with reflux had average diameters significantly wider than that of greater saphenous veins without reflux.

By design, after ligation at the femoral junction, a proximal segment of the saphenous vein may have reverse flow from a tributary source to a distal drainage vein.[7] Otherwise, the proximal saphenous vein may thrombose from the junction to the next branch. Therefore, a perforating vein may become a source of reverse flow, drainage of reverse flow, or drainage of normal, distal saphenous flow into the deep system. In these circumstances, the perforating vein may have an enlarged diameter that is wider than normal. Not only must we consider the diameter of perforating veins in the decision-making process of their ligation, but also we must understand the role played by such communicating veins before varicose vein surgery.

In summary, treatment of chronic venous valvular insufficiency is evolving toward definite, predictable outcomes.[51] The CEAP classification is forcing a unified description of patients. It is not enough, however, to simply describe the patients studied. Investigation should be focused on very specific patient populations. We limited our study to a select group of patients with varicose veins, CEAP clinical class C_2. We demonstrated that the presence of reflux in perforating veins may be dependent on diameter differences of approximately 1 to 2 mm. Calf perforating veins are probably normal, abnormal but competent, incompetent, or severely incompetent with diameters in the 1.5-, 2.5-, 3.5-, and 4.5-mm range, respectively. These diameter observations were consistent with published data about the greater saphenous vein, particularly at the calf level, obtained in similar populations. The hypothesis that perforating veins could become competent after elimination of primary sources of reflux has been raised. Such cases have been described in the literature. In contrast, there is little evidence to support leaving an incompetent perforating vein with a diameter wider than 4.5 mm in the calf. Further research is recommended to determine if postoperative diameter reduction is a function of original diameter and/or patterns of flow in the deep, superficial, communicating, and/or perforating veins of the lower extremities. To better understand the fate of perforating veins after varicose vein surgery, the effects on diameter and reflux patterns of greater saphenous vein valvuloplasty or high ligation, alone or with limited or complete stripping, should be evaluated in detail.

REFERENCES

1. Meyers TT. Results and technique of stripping operation for varicose veins. JAMA 1957;163:87-92.
2. Lange J. Surgical treatment of saphenous varices with preservation of the main great saphenous trunk. J Vasc Surg 1985;2:886-91.
3. Hammersten J, Pederson P, Cederlund CG, Camparello M. Long saphenous vein saving surgery for varicose veins: A long-term follow-up. Eur J Vasc Surg 1990;4:361-4.
4. Rutherford RB, Saywer JD, Jones DN. The fate of residual saphenous vein after partial removal or ligation. J Vasc Surg 1990;12:422-8.
5. Engelhorn C, Picheth F, Castro N, et al. Color flow localization of insufficient communicating or perforating veins prior to surgical ligation. J Vasc Technol 1993;17:251-3.
6. Schanzer H, Skladany M. Varicose vein surgery with preservation of the saphenous vein: A comparison between high ligation-avulsion versus saphenofemoral banding valvuloplasty-avulsion. J Vasc Surg 1994;20:684-7.
7. Fonseca FP, Sarquis AL, Evangelista SS. Surgery for primary troncular varicose veins without stripping saphenous veins: Pre- and postoperative evaluation by duplex scan and photoplethysmography. Phlebology 1995;Suppl 1:419-21.
8. Villavicencio JL, Pfeifer JR, Lohr JM, et al. Sclerotherapy for varicose veins: Practice guidelines and sclerotherapy procedures. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p.337-54.
9. Bergan JJ. Surgical Management of primary and recurrent varicose veins. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p.394-415.
10. Queral LA, Criado FJ. Miniincisional ligation of incompetent perforating veins of the legs. J Vasc Surg 1997;25:437-41.
11. Linton RR. The communicating veins of the lower leg and the operative technique for their ligation. Ann Surg 1938;107:582-93.
12. Dodd H. The diagnosis and ligation of incompetent perforating veins. Ann R Coll Surg Engl 1964;34:186-96.
13. Stacey MC, Burnand KG, Layer GT, Pattison M. Calf pump function in patients with healed venous ulcers is not improved by surgery to the communicating veins or by elastic stockings. Br J Surg 1988;75:436-9.
14. Pierik EG, Wittens CH, van Urk H, et al. Subfascial endoscopic ligation in the treatment of incompetent perforating veins. Eur J Vasc Endovasc Surg 1995;9:38-41.
15. Gloviczki P, Cambria RA, Rhee RY, et al. Surgical technique and primary results of endoscopic subfacial division of perforating veins. J Vasc Surg 1996;23:517-23.
16. DePalma RG. Management of incompetent perforators: Conventional techniques. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p. 471-81.
17. Sullivan TR, O'Donnell TF Jr. Endoscopic division of incompetent perforating veins. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p.482-93.
18. Gloviczki P, Bergan JJ, Menawatt SS, et al. Safety, feasibility, early efficacy of subfacial endoscopic perforator surgery: A preliminary report from the North American registry. J Vasc Surg 1997;25:94-105.
19. Pierik EG, van Urk H, Wittens CH. Efficacy of subfascial endoscopy in eradicating perforating veins of the lower leg and its reaction with venous ulcer healing. J Vasc Surg 1997;26:255-9.
20. Gloviczki P, Burnand KG. Breakfast section. Proceedings of the 10th Annual Meeting of the American Venous Forum; 1998 Feb 18–21; Lake Buena Vista, Fla.
21. van Bemmelen PS, Bedford G, Beach K, Strandness DE Jr. Quantitative segmental evaluation of venous valvular reflux with duplex ultrasound scanning. J Vasc Surg 1989;10:425-31.
22. van Bemmelen PS, Beach K, Bedford G, Strandness DE Jr. The mechanism of venous valve closure. Its relationship to the velocity of reverse flow. Arch Surg 1990;125:617-9.
23. Hanrahan LM, Araki CT, Fisher JB, et al. Evaluation of the perforating veins of the lower extremity using high resolution duplex imaging. J Cardiovasc Surg (Torino) 1991;32:87-97.
24. Miller SC, Foote AV. The ultrasonic detection of incompetent perforating veins. Br J Surg 1991;61:653-6.
25. 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 1992:16:687-93.
26. Rizzo JL, Baker LA, Boyajian RA, et al. Accuracy of B-mode ultrasound vein mapping for vessel size. J Vasc Technol 1993;17:287-9.
27. Weingarten MS, Branas CC, Czeredarczuk M, et al. Distribution and quantification of venous reflux in lower extremity chronic venous stasis disease with duplex scanning. J Vasc Surg 1993;18:753-9.
28. Myers KA, Ziegenbein RW, Zeng GH, Mathews PG. Duplex ultrasonography scanning for chronic venous disease: Patterns of venous reflux. J Vasc Surg 1995;21:605-12.
29. Sumner DS. Direct noninvasive tests for the evaluation of chronic venous obstruction and valvular incompetence. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p. 130-51.
30. Pierik EG, Toonder IM, van Urk H, Wittens CH. Validation of duplex ultrasonography in detecting competent and incompetent perforating veins in patients with venous ulceration of the lower leg. J Vasc Surg 1997;26:49-52.
31. Salles-Cunha SX, Beebe HG. Direct noninvasive tests (duplex scan) for evaluation of acute venous disease. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p.112-29.
32. Engelhorn C, Salles-Cunha SX, Picheth FS, et al. Greater saphenous vein diameters as a function of reflux patterns. Proceedings of the 9th Annual Meeting of the American Venous Forum; 1997 Feb 20–23; San Antonio, Texas. p. 67.
33. Engelhorn CA, Engelhorn AL, Casagrande C, Salles-Cunha SX. Sources and drainage of saphenous vein reflux in patients with primary varicose veins. Proceedings of the 11th Annual Meeting of the American Venous Forum; 1999 Feb 18–21; Dana Point, Calif.
34. Stuart WP, Adam DJ, Allan PL, et al. Saphenous surgery does not correct perforator incompetence in the presence of deep venous reflux. J Vasc Surg 1998;28:834-8.
35. Porter JM, Moneta GL, International Consensus Committee on Chronic Venous Disease. Reporting standards in venous disease: An update. J Vasc Surg 1995;21:635-45.

36. Porter JM, Moneta GL, International Consensus Committee on Chronic Venous Disease. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p. 629-51.
37. Ad Hoc Committee of the American Venous Forum. Classification and grading of chronic venous disease in the lower limbs: A consensus statement. In: Gloviczki P, Yao JST, editors. Handbook of venous disorders. London: Chapman and Hall; 1996. p. 652-60.
38. 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;12:487-91.
39. Cockett FB. The pathology and treatment of venous ulcers of the leg. Br J Surg 1955;43:260-78.
40. Bjordal RI. Circulation patterns in incompetent perforating veins in the calf and in the saphenous system in primary varicose veins. Acta Chir Scand 1972;138:251-61.
41. Burnard KG, O'Donnell TF, Thomas ML, Browse NL. The relative importance of incompetent communicating veins in the production of varicose veins and venous ulcers. Surgery 1977;82:9-14.
42. Zukowski AJ, Nicolaides AN. Hemodynamic significance of incompetent calf perforating veins. Br J Surg 1991;78:625-9.
43. Sarin S, Scurr JH, Smith PD. Medial calf perforators in venous disease: The significance of outward flow. J Vasc Surg 1992;16:40-6.
44. Labropoulos N, Leon M, Nicolaides NA, et al. Superficial venous insufficiency: Correlation of anatomic extent of reflux with clinical symptoms and signs. J Vasc Surg 1994;20: 953-8.
45. Labropoulos N, Delis K, Nicolaides AN. Venous reflux in symptom-free vascular surgeons. J Vasc Surg 1995;22:150-4.
46. Labropoulos N, Delis K, Mansour A, et al. Prevalence and clinical significance of posterolateral thigh perforator vein incompetence. J Vasc Surg 1997;26:743-8.
47. Delis KT, Ibegbuna V, Nicolaides A, et al. Prevalence and distribution of incompetent perforating veins in chronic venous insufficiency. J Vasc Surg 1998;28:815-25.
48. Labropoulos N, Mansour A, Kang SS, et al. New insights into perforator vein incompetence. Eur J Vasc Endovasc Surg. In press 1999.
49. Engelhorn C, Engelhorn A, Salles-Cunha SX, et al. Relationship between reflux and greater saphenous vein diameter. J Vasc Technol 1997;21:167-72.
50. Criado E, Daniel PF, Marston W, et al. Physiologic variations in lower extremity venous valvular function. Ann Vasc Surg 1995;9:102-8.
51. Kistner RL. Definitive treatment in chronic venous disease: A concept whose time has come. J Vas Surg 1996;24:703-10.

Submitted Feb 23, 1999; accepted May 12, 1999.

DISCUSSION

Dr Bo Eklof (Honolulu, Hawaii). Sergio Salles-Cunha and his international group present an interesting hypothesis: if the primary source of reflux mainly in the saphenous system is eliminated, perforating vein diameter may diminish and the valves may regain competency. Thank you very much for the manuscript.

Guidelines suggesting which perforators would be ligated are lacking. At the breakfast debate at the last American Venous Forum meeting, Peter Gloviczki and Kevin Burnand were battling about the importance of incompetent perforators and the role of subfascial endoscopic perforator surgery. To gain more information, the authors have investigated the prevalence of reflux as a function of perforating vein diameter in a homogenous patient population classified as clinical class C_2–varicose veins, according to CEAP. There were no class C_4–skin changes, C_5–previous ulcer, or C_6–active ulcers in their group. All patients had primary venous disease. Anatomically, there was incompetence of the long saphenous vein and perforators in all patients, combined with deep venous incompetence in approximately 10% of patients. Pathophysiologically, all patients had reflux. There were no obstructions, and 51.7% of the limbs were symptomatic, with pain. That is, a group of patients without complications of their primary varicose veins. There were 500 perforators in 116 limbs in 78 patients; 282 perforators, more than 50%, were incompetent. Most perforators, 75%, were located medially in the calf, 22.7 cm from the base of the foot. In the thigh, a perforating vein diameter larger than 3.5 mm indicated reflux in 92% of the cases. Calf perforating vein diameter larger than 3.5 was predictive of reflux in 90% of the cases.

Do perforators above the knee connect with the long saphenous vein? Below the knee, do they connect to the long saphenous vein, the posterior arch, or the short saphenous vein? To test your hypothesis, how would you design further studies? Would you do a valvuloplasty at the proximal long saphenous vein? Would you do high ligation? Would you combine high ligation with stripping below the knee or down to the ankle? Would you leave a perforator that is more than 4.5 mm in diameter in this group? What is your opinion on the Chiva theory on the role of thigh perforators as reentry vessels for the incompetent long saphenous vein? Because I am a believer in the significant role of the incompetent perforator in chronic venous disease, I always take part in marking all the perforators in the vascular laboratory. The time has come for us to have a duplex scanner in the office, so that we can learn how to diagnose and mark the incompetent perforators, among other things. We have tried to stimulate the industry to produce a cheaper, simpler scanner for routine use in the office, an extension of the hand-held vascular surgery Doppler.

I thank the president and the Forum for the privilege of discussing this important paper. Thank you.

Dr João Luis Sandri. I want to thank Dr Eklof for the questions.

Your first question was, do the perforators above the knee connect with the long saphenous vein? Yes, they do connect; however, perforators were a direct source of reflux in only 3% to 4% of cases, according to Engelhorn's poster outside.

Your second question was whether perforators below the knee connect to the long saphenous vein, posterior arch, or short saphenous vein. We observed that the perforators communicate mostly with the posterior arch. Once more, according to Engelhorn's poster, perforating veins were the direct source of calf greater-saphenous-vein reflux in only 7% of cases and lesser-saphenous-vein reflux in 12% of cases.

With your third question, you have given us good ideas to test our hypothesis and design of further studies. We are now in the first step of our study, that is, just disconnection, high ligation of the saphenous femoral junction to abolish reflux source and resection of varicose veins.

We do not leave perforators larger than 4.5 mm. We do ligate them because it is a severely abnormal incompetence. This is the size that links to complications, such as induration and ulceration. When the larger perforator is connected to a branch that is going to be avulsed, however, we don't go after the perforator to avoid a possibly larger scar incision.

We don't use the classic French method, but we pay attention to the sources of reflux, rather than drainage. If we have to intervene, we would ligate or strip the saphenous vein, ligation when the diameter is 6 to 8 mm and stripping when the diameter is more than 10 to 11 mm. Today, we know that approximately 70% of disconnected saphenous veins show a significant reduction in size after ligation, but larger sizes fail to do so.

I would once more like to, in the name of my colleagues, thank you for the opportunity to present this paper.