J CARDIOVASC SURG 2006;47:629-35

Impact of the calf perforators on the venous hemodynamics in primary varicose veins

C. RECEK

Division of Vascular Surgery, Department of Surgery, University Hospital, Hradec Kralove, Czech Republic

The hemodynamic significance of the calf perforating veins continues to be the point of controversy. The conception that incompetent perforating veins cause hemodynamic disturbance and are responsible for the formation of leg ulceration has still many adherents prefering perforator surgery, whereas others reject any causal relation between large, incompetent perforators and severe forms of chronic venous insufficiency. In this study well documented facts concerning the impact of the calf perforators on the venous hemodynamics are reviewed. There is a bidirectional flow within calf perforators in healthy subjects enabling a quick equilibration of pressure changes produced during calf muscle contractions and relaxations, so that recordings of the mean pressure display identical values in superficial and deep veins of the lower leg, a feature typical of conjoined vessels. In cases with saphenous reflux, the bidirectional flow within calf perforators has a distinct inward vector directed to the deep veins; this inward component is the more pronounced, the larger the saphenous reflux is. Incompetent calf perforators do not cause ambulatory venous hypertension, exactly the opposite happens: the high hydrostatic pressure found in the quiet standing position drops significantly during ambulation, as soon as the saphenous reflux is interrupted. In primary varicose veins calf perforators can not become the source of reflux because they are situated at the lower pole of the ambulatory pressure gradient, which occurs between thigh and lower leg veins during ambulation. The size of the calf perforators is determined by the amount of saphenous reflux. When the saphenous reflux is abolished (*e.g.* by high ligation), the enlarged calf perforators diminish.

Key words: Veins - Hypertension - Venous insufficiency.

Linton, in 1938, and Cocquett and Jones, in 1953, pointed out the importance of incompetent lower leg perforators for the development of leg ulcers. Linton [1] drew his conclusions from 10 anatomical studies on bodies and from 50 operations. He reported on perforating veins connecting the posterior tibial, the anterior tibial, the peroneal, and the popliteal veins with the greater and lesser saphenous veins and proposed the subfascial ligation of perforators. Cocquett and Jones[2] argued on the basis of their anatomical studies on bodies (they did not precise the total number of studies, mentioned only two examinations), and of their 10 operated cases with a follow-up of several months that the perforating veins collect the venous drainage of the skin and subcutaneous tissue in the gaiter area, and when incompetent, they cause venous hypertension leading to ulcer formation. They believed that the perforating veins were supplied with valves enabling unidirectional flow only, namely, from superficial into deep veins. This opinion has since been widely accepted by most autors. As a consequence, perforating veins have been divided into competent ones found in healthy subjects, and incompetent ones found in pathological situations. According to this theory, if in the course of the disease the perforating veins of the lower leg become incompetent, a considerable amount of blood leaking during muscle contractions from deep to superficial veins produces venous hypertension in the skin and subcutaneous tissue and promotes formation of leg ulcers. However, this theoretical assumption was not

Address reprint requests to: C. Recek, Mantlergasse 24, A-1133 Vienna, Austria. E-mail: recek@aon.at

substanciated by functional tests and hard facts: no evidence has so far been offered that incompetent calf perforators really cause ambulatory venous hypertension and that significant improvement of venous hemodynamics or normalization of ambulatory venous pressure in the superficial veins occurs after selective interruption of incompetent calf perforators. On the contrary, many functional studies which are mentioned and discussed in the following chapters document that large calf perforators do not cause ambulatory venous hypertension and that there is a close relation between the amount of saphenous reflux and the size of calf perforating veins.

No reflux, but a physiological bidirectional flow takes place in calf perforators. Perforating veins make superficial and deep veins of the lower leg conjoined vessels

Reflux is defined as a retrograde, downward flow in an incompetent vein connecting both poles of the ambulatory pressure gradient that occurs between the veins of the thigh and the lower leg during ambulation in an erect position and under the influence of gravitation[3]. The activity of the calf muscle venous pump evokes a pronounced pressure drop in deep and superficial veins of the lower leg, whereas no pressure decrease occurs in the popliteal and femoral veins; this pressure difference has been called ambulatory pressure gradient[4] and is the prerequisite for the venous reflux to set in[3]. Because calf perforators are situated at the lower pole of the ambulatory pressure gradient, they can not become the source of reflux, they represent the mouth of saphenous reflux and are also called re-entry points. On the other hand, thigh perforators are connected with the higher pole of the gradient, and therefore, when incompetent, become the origin of retrograde flow. The reflux-carrying incompetent vein connects the iliac, femoral or popliteal vein with 1 of the deep veins of the lower leg[3].

Reflux can also be described as shunting of blood from thigh into lower leg veins.

Simultaneous pressure recordings in the saphenous and posterior tibial veins have shown that the mean pressure curves in these veins have been identical at rest, during ambulation, and in the recovery period[5-7]. The analysis of the instantaneous, undamped pressure has demonstrated light pressure differences between the saphenous and the posterior tibial vein during ambulation. During muscle contraction the vector of the pressure difference has been directed outward, *i.e.* to the saphenous vein; in this phase, some blood escapes *via* calf perforators from the posterior tibial into the saphenous vein and is drained at the saphenofemoral junction into the femoral vein[8]. During muscle relaxation the vector turns round and is directed toward the posterior tibial vein; in this phase, the blood is aspirated from the superficial into the deep veins of the lower leg[8]. This bidirectional flow within the calf perforators with a predominantly inward resultant net flow was clearly documented by electromagnetic flow measurements in primary varicose veins[9] and by color coded duplex ultrasonography in healthy subjects[10,11]. Sarin *et al.*[10] observed no significant difference in the number of perforators allowing outward flow between normal limbs and limbs with superficial or deep venous insufficiency. The flow within medial calf perforators was in either direction, depending on the instantaneous local pressure conditions. Labropoulos *et al.*[11] found a bidirectional flow within calf perforators in healthy subjects as well.

Bjordal[9] performed simultaneous pressure and flow recordings by using electromagnetic flowmeter in varicose vein patients. He demonstrated that during "walking in place" the flow in the incompetent saphenous vein as well as calf perforators had a time-varying characteristic, *i.e.*, a bidirectional pattern, with a predominant retrograde (distal) component in the saphenous vein and an inward component within the incompetent calf perforator. The average mean retrograde flow in the saphenous trunk in the thigh was 280 mL/min (range 175-500 mL/min); this amount of blood is drained through the calf perforators into the deep veins. The perforator size fashions to the amount of the drained blood, it enlarges with increasing rates of saphenous reflux and diminishes after elimination of reflux.

The bidirectional flow taking place in calf perforators makes the superficial and deep venous system of the lower leg conjoined vessels and allows a quick equilibration of pressure changes between deep and superficial veins during ambulation. Calf perforators are essentially incompetent, the bidirectional flow within them is a physiological feature. Large calf perforators are not the source of venous reflux, they represent the mouth of reflux.

Relation between the incidence of incompetent calf perforators and the clinical grade of chronic venous insufficiency

Stuart *et al.*[12] reported that the number of demonstrable perforators, the number of incompetent perforating veins, and the median diameters of perforators increased with deteriorating grade of CEAP classification. Delis *et al.*[13] draw the same conclusion in their report; the prevalence of incompetent perforating veins increased significantly with the clinical severity of chronic venous insufficiency. Niederle and Prerovsky[14] performed hemodynamic measurements with strain gauge plethysmography in patients with uncomplicated varicose veins and with chronic venous insufficiency. The incidence of incompetent perforators was significantly higher in the group with a more pronounced hemodynamic disturbance. Labropoulos *et al.*[15] investigated 103 limbs in 75 patients with different grades of chronic venous insufficiency by using color-flow duplex scanning, and reported that the total number of perforating veins and the number of incompetent perforating veins per limb increased significantly with the severity of chronic venous insufficiency.

These findings seem to support the perforator theory. But there is another factor involved in the pathophysiology of the venous hemodynamic disorder, namely the saphenous reflux, which influences both the severity of chronic venous insufficiency and the size of calf perforators. Some hemodynamic studies have discovered a close correlation between the amount of saphenous reflux and the clinical grade of chronic venous insufficiency. Christopoulos *et al.*[16] examined 24 normal volunteers (24 limbs), 18 patients (21 limbs) with primary varicose veins but without sequelae of venous disease, 24 patients (31 limbs) with primary varicose veins and sequelae of venous disease (chronic swelling, skin changes, ulcerations), and 25 patients (28 limbs) with popliteal reflux by use of air plethysmography and venous pressure measurements. The parameter venous fillig index (VFI) quantifies reflux in ml/sec. In normal limbs it was less than 1.7 mL/s, in limbs with uncomplicated varicose veins between 2 and 9.5 mL/s, in limbs with sequelae (pigmentation, liposclerosis, ulceration) between 3 and 30 mL/s, and in limbs with popliteal reflux between 7 and 28 mL/s. The incidence of skin changes was low for a VFI less than 5 mL/s, it increased with an increase of VFI. The incidence of sequelae in relation to increasing rates of reflux occured irrespective of whether the reflux was in superficial or deep veins. In the trial of Welkie *et al.*[17] 270 lower limb extremities from 149 patients with varying degrees of chronic venous insufficiency and 56 limbs from 28 symptom-free volunteers were examined by using air plethysmography. Extremities were classified according to the guidelines established by the *Ad hoc* Committee For Reporting Standards of the Society for Vascular Surgery and the International Society for Vascular Surgery. VFI was 1.52±0.11 mL/s in healthy subjects, 2.74±0.26 mL/s in class 1, 7.86±0.80 mL/s in class 2, and 8.28±0.73 mL/s in class 3. Similar results and a good correlation between the VFI and the clinical severity of chronic venous insufficiency were published by Neglen and Raju[18]. Nicolaides *et al.*[19] examined 336 limbs in 220 successive patients with venous disease by using duplex scanning and venous pressure measurements. They found a linear increase in the incidence of leg ulcerations in dependence of the level of ambulatory hypertension. Payne *et al.*[20] examined 360 limbs with a broad spectrum of venous disease and found a good correlation between the level of ambulatory venous hypertension and the clinical grade of chronic venous insufficiency. Labropoulos *et al.*[11] reported that the venous volume, venous filling index and residual volume fraction determined by air plethysmography worsened with the progression of chronic venous insufficiency.

Although the incidence and the size of incompetent calf perforators correlate with the severity of chronic venous insufficiency, the decisive factor for the development of pathological changes in primary varicose veins is the amount of saphenous reflux affecting both the incidence of chronic venous insufficiency and the size of calf perforators.

Relation between the saphenous reflux and the size of incompetent calf perforators

Large amounts of retrograde flow running downward the incompetent saphenous vein are drained through the calf perforators into the deep veins of the lower leg. Although it has so far not been examined whether a correlation exists between the amount of saphenous reflux and the size of calf perforators, there is an indirect evidence of a close relation between them, and it can be expressed as follows: the more voluminous the saphenous downward flow is, the

more blood runs through the calf perforators inward into the deep veins of the lower leg, the larger is the size of calf perforators. This presumption is further corroborated by the evidence that the size of large calf perforators as well as of other enlarged venous segments involved in the "private circulation" defined by Trendelenburg[21] diminished after the saphenous reflux had been eliminated[22-25]. Stuart *et al.*[22] examined 62 limbs of 47 patients undergoing superficial vein surgery (saphenofemoral and/or saphenopopliteal junction ligation, stripping of the long saphenous vein in the thigh, and multiple phlebectomies) with color flow duplex ultrasound scan immediately before and a median of 14 weeks after operation. Surgery resulted in a significant reduction of the total number of limbs in which incompetent perforating veins were imaged, in a reduction of median perforator diameter, and significantly reduced the proportion of perforators imaged that were incompetent. A statistically significant reduction in incompetent lower leg perforators after superficial vein surgery was recently reported by Gohel *et al.*[23] Similar results were published by Al-Mulhim *et al.*[24] 74.5% of preoperatively incompetent perforators regained competence after isolated superficial vein surgery. The diameter of mid-calf and lower-calf perforators diminished significantly from 5.5±1.4 mm and 4.4±0.5 mm preoperatively to 3.9±1.7 mm and 3±0.4 mm after surgery, respectively. Recek *et al.*[25] found that the phlebographically demonstrated diameter of 31 lower leg perforators in patients with primary varicose veins and with a marked saphenous reflux was significantly reduced 6 months after superficial vein surgery (7.2±1.9 mm preoperatively, 6.4±1.8 mm after surgery).

A close relation obviously exists between the amount of saphenous reflux and the size of calf perforators. Although no study confirming a statistically significant correlation between them has so far been presented, it can be reasonably presumed that the amount of retrograde saphenous flow determines the size of calf perforators. In any case, the diameter of calf perforators significantly diminishes after elimination of saphenous reflux.

Incompetent calf perforators and ambulatory venous hypertension

As mentioned in the introduction, the theory of the blow-out syndrome asserts that ambulatory venous hypertension in superficial veins of the ankle region is due to a large volume of blood leaking from the deep veins of the lower leg into the superficial system *via* incompetent perforators during muscle contractions. Many studies claimed favorable results after perforator vein surgery. However, the interpretation of these results is confounded by the fact that perforator surgery has usually been performed simultaneously with the abolition of saphenous reflux, precluding in this way separate analysis of the influence on venous function of each procedure[26-33]. In addition, the interpretation of clinical outcome and ulcer healing was further hampered by additional therapeutic procedures, including prolonged postoperative bed rest, elevation and compression treatment[33].

The simple and well known Perthes test documents that incompetent calf perforators in primary varicose veins do not produce ambulatory venous hypertension. Perthes, a disciple of Trendelenburg, examined most patients with severe forms of chronic venous insufficiency and with strongly filled varicose veins in the leg. When in these patients the retrograde flow in the incompetent saphenous vein was interrupted by compression and the patient activated the muscle venous pump (*e.g.* by walking, tiptoe movements or knee bending), the bulging varicose veins evidently emptied[34]. This is, of course, in total discrepancy with the theory of incompetent calf perforators: according to this theory the varicose veins should yet more fill up in such cases.

Simultaneous recordings of flow and pressure curves in an incompetent saphenous vein and incompetent calf perforator have brought the direct evidence that ambulatory venous hypertension has not been caused by blood evasion through incompetent calf perforators. First, the flow in the incompetent calf perforator has been predominantly inward, not outward, and second, the high ambulatory venous pressure measured either in the incompetent saphenous vein or directly in the incompetent calf perforator has distinctly dropped and normalized, as soon as the retrograde flow in the saphenous trunk has been stopped[9].

So, no ambulatory venous hypertension due to the blood evasion occurs in incompetent calf perforators and in superficial veins in the lower leg during ambulation, exactly the opposite happens: the venous pressure drops considerably as soon as the saphenous reflux is eliminated. Incompetent calf perforators do not produce ambulatory venous hypertension.

Interruption of incompetent calf perforators does not improve venous hemodynamics

Burnand *et al.*[35] measured venous pressure in 20 patients with incompetent calf perforators before operation and 3 months after selective surgical perforator ligation. The ambulatory venous pressure improved very little and remained far below normal postoperatively. Akesson *et al.*[36] reported on 25 patients (30 limbs treated) with chronic venous insufficiency. Deep venous insufficiency was seen on descending phlebography in all cases; in 12 limbs a mild reflux limited to above the knee level, and in 18 limbs a severe reflux extending below the knee level was diagnosed. The patients were examined by use of foot volumetry and venous pressure measurements. Twelve limbs with a clinical evidence of superficial insufficiency underwent saphenous stripping and excision of varicosities; 3 months later extensive subfascial ligation of all incompetent perforating veins was performed on all 30 limbs. Venous pressure was measured inicially and 3 months after each surgical procedure. Ambulatory venous pressure improved significantly after superficial vein surgery, but did not change after ensuing subfascial ligation of incompetent perforators. Foot volumetry parameters behaved in a similar manner: they improved after superficial vein surgery, but did not change after subsequent perforator ligation. In the trial of Scriven *et al.*[37] concerning patients with combined deep and perforating vein incompetence but without saphenous reflux the preoperative photoplethysmography demonstrated a global abnormality of venous function at all sites examined that persisted after perforating vein surgery. Likewise, no further improvement of venous hemodynamics was achieved by additional perforator ligation in the study of Fitridge *et al.*[38]

The quoted papers evidenced that selective interruption of incompetent calf perforators did not improve ambulatory venous hypertension and/or venous hemodynamics both in primary varicose veins and in deep venous insufficiency.

Relation between saphenous reflux, ambulatory venous hypertension, and venous hemodynamic disturbance

Hypertension in the veins of the lower leg found in a quiet standing position is equivalent to the hydrostatic pressure induced by gravitation. Whereas in healthy subjects the hydrostatic pressure drops considerably during ambulation, the venous reflux interferes with the effectivity of the calf muscle venous pump. As a consequence, depending on the amount of the retrograde flow, the pressure drop is more or less attenuated, and when the expelled and refluxing volumes get in equilibration, no pressure decrease occurs during ambulation. If large enough, the saphenous reflux itself may cause the most severe form of chronic venous insufficiency[39-45]. Interruption of retrograde flow in the incompetent saphenous vein restores physiological pressure drop indicating normalization of hemodynamic situation; release of the saphenous compression results in an immediate increase of ambulatory venous pressure, evidencing a close relation between the saphenous reflux and ambulatory venous hypertension[9]. Burnand *et al.*[35] measured venous pressure in a vein on the dorsum of the foot at rest and during maximal heel raising without and with a tourniquet on the thigh in 37 cases. The pathological ambulatory venous pressure was restored to normal limits with applied tourniquet. Recek[46] examined 30 patients (37 limbs) with severe chronic venous insufficiency by use of strain gauge plethysmography. The marked venous disturbance in these patients, as documented by low values of refill time and refill volume, disappeared after elimination of saphenous reflux by crossectomy, and normal hemodynamic conditions were restored one week after surgery, although large incompetent perforators were left intact. Fitridge *et al.*[38] examined 35 patients (38 limbs) with uncomplicated varicose veins by using air plethysmography and found that superficial venous surgery significantly improved venous volume, venous filling index, and ejection fraction. Perforator surgery performed in addition to superficial vein surgery brought no further improvement. Padberg *et al.*[47] and Ting *et al.*[48] showed that selective abolition of saphenous reflux in patients with combined saphenous and femoral vein incompetence restored normal venous hemodynamics in spite of persisting femoral vein incompetence.

In primary varicose veins, the ambulatory venous hypertension is caused by retrograde flow in the incompetent saphenous vein. The interruption of saphenous reflux normalizes ambulatory venous pressure and restores normal venous hemodynamics in spite of persisting large incompetent lower leg perforators.

Conclusions

Following facts can be considered to be proved:

1) Bidirectional flow within calf perforating veins occurs during ambulation in healthy subjects and in primary varicose veins enabling a quick equilibration of pressure changes produced during the activity of the calf muscle venous pump. The mean venous pressure in the superficial and deep veins of the lower leg is identical during quiet standing, ambulation, and the recovery period, indicating that calf perforators make superficial and deep venous system of the lower leg conjoined vessels.

2) In primary varicose veins, the diastolic inward flow within calf perforators is larger than the systolic outward one, resulting in a net inward flow during the ambulation period, irrespective of whether the perforating veins are competent or not.

3) The high ambulatory venous pressure at the ankle region in primary varicose veins is caused by saphenous reflux; abolition of reflux results in a marked drop of ambulatory venous pressure signalizing normalization of venous hemodynamics in spite of the presence of incompetent calf perforators. Incompetent calf perforators neither produce nor influence the ambulatory venous hypertension.

4) A close relation exists between the saphenous reflux and the size of calf perforating veins. Large perforating veins are obviously the consequence of a voluminous saphenous reflux. The diameter of the enlarged perforators diminishes after elimination of the saphenous retrograde flow.

5) In view of the fact that calf perforators are situated at the lower pole of the ambulatory pressure gradient, they can not became the source of venous reflux in primary varicose veins. They represent the mouth of saphenous reflux (re-entry points).

References

1. Linton RR. The communicating veins of the lower leg and the operative technic for their ligation. Ann Surg 1938;107:582-93.
2. Cockett FB, Jones EE. The ankle blow-out syndrome. A new approach to the varicose ulcer problem. Lancet 1953;1:17-23.
3. Recek C. The venous reflux. Angiology 2004;55:541-8.
4. Recek C, Pojer H. Ambulatory pressure gradient in the veins of the lower extremity. VASA 2000;29:187-90.
5. Höjensgard IC, Stürup H. Static and dynamic pressures in superficial and deep veins of the lower extremity in man. Acta Physiol Scand 1952;27:49-62.
6. Recek C. A critical appraisal of the role of ankle perforators for the genesis of venous ulcers in the lower leg. J Cardiovasc Surg 1971;12:45-9.
7. Recek C, Koudelka V. Calf perforators and their role in the pathogenesis of varicose veins (Czech). Rozhl Chir 1973;52:14-20.
8. Recek C. Venöse Hämodynamik in den Beinen bei Gesunden und bei primärer Varikose. Phlebologie 2001;30:107-14.
9. Bjordal RI. Circulation patterns in incompetent perforating veins in the calf and the saphenous system in primary varicose veins. Acta Chir Scand 1972;138:251-61.
10. Sarin S, Scurr JH, Coleridge Smith PD. Medial calf perforators in the venous disease: the significance of outward flow. J Vasc Surg 1992;16:40-6.
11. Labropoulos N, Mansour MA, Kang SS, Gloviczki P, Baker WH. New insight into perforator vein incompetence. Eur J Vasc Endovasc Surg 1999;1:228-34.
12. Stuart WP, Adam DJ, Allan PL, Ruckley CV, Bradbury AW. The relationship between number, competence, and diameter of medial calf perforating veins and the clinical status in healthy subjects and patients with lower-limb venous disease. J Vasc Surg 2000;32:138-43.
13. Delis KT, Ibegbuna V, Nicolaides AN, Lauro A, Hafez H. Prevalence and distribution of incompetent perforating veins in chronic venous insufficiency. J Vasc Surg 1998;28:815-25.
14. Niederle P, Prerovsky I. The evaluation of venous muscle pump disorder in chronic venous insufficiency. VASA 1976;5:24-7.
15. Labropoulos N, Giannoukas AD, Nicolaides AN, Veller M, Leon M, Volteas N. The role of venous reflux and calf muscle venous pump function in nonthrombotic chronic venous insufficiency. Correlation with severity of signs and symptoms. Arch Surg 1996;131:403-6.
16. Christopoulos D, Nicolaides AN, Szendro G. Venous reflux: quantification and correlation with clinical severity of chronic venous insufficiency. Br J Surg 1988;75:352-6.
17. Welkie JF, Comerota AJ, Katz ML, Aldridge CS, Kerr RD, White JV. Hemodynamic deterioration in chronic venous disease. J Vasc Surg 1992;16:733-40.
18. Neglen P, Raju S. A rational approach to detection of significant reflux with duplex Doppler scanning and air plethysmography. J Vasc Surg 1993;17:590-5.
19. Nicolaides AN, Hussein MK, Szendro G, Christopoulos D, Vasdekis S, Clarke H. The relation of venous ulceration with ambulatory pressure measurements. J Vasc Surg 1993;17:414-9.
20. Payne SP, London NJ, Newland CJ, Thrush AJ, Barrie WW, Bell PR. Ambulatory venous pressure: correlation with skin condition and the role of identifying surgically correctable disease. Eur J Vasc Endovasc Surg 1996;11:195-200.
21. Trendelenburg F. Über die Unterbindung der V. saphena magna bei Unterschenkelvarizen. Beitr Klin Chir 1891;7:195-210.
22. Stuart WP, Adam DJ, Allan PL, Ruckley CV, Bradbury AW. Saphenous surgery does not correct perforator incompetence in the presence of deep venous reflux. J Vasc Surg 1998;28:834-8.
23. Gohel MS, Barwell JR, Wakely C, Minor J, Harvey K, Earnshow JJ *et al.* The influence of superficial venous surgery and compression on incompetent calf perforators in chronic venous leg ulceration. Eur J Vasc Endovasc Surg 2005;29:78-82.
24. Al-Mulhim AS, El-Hoseiny H, Al-Mulhim FM, Bayameen O, Sammi MM, Abulaziz K el al. Surgical correction of main stem reflux in the superficial venous system: does it improve the blood flow of incompetent perforating veins? World J Surg 2003;27:793-6.
25. Recek C, Karisch E, Gruber J. Veränderungen der Perforansvenen und tiefen Unterschenkelvenen nach Beseitigung des Saphena-Refluxes. Phlebologie 2000;29:37-40.
26. Cockett FB. Die Rolle der Vv. Perforantes des Knöchels beim postthrombotischen Syndrom und bei primärer, hereditärer Varikose. In: May R, Partsch H, Staubesand J, eds. Venae perforantes. München, Wien, Baltimore: Urban u. Schwarzenberg; 1981.p.107-9.
27. Fischer R, Schwahn-Schreiber CH, Sattler G. Ergebnisse der Konsensuskonferenz über die subfasciale Endoskopie der Vv. Perforantes des medialen Unterschenkels. Phlebologie 1997;26:60-5.
28. Gloviczki P, Bergan JJ, Rhodes JM, Canto LG, Harmsen S, Ilstrup DM. Mid-term results of endoscopic perforator vein interruption

for chronic venous insufficiency: lessons learned from the Nord American Subfascial Endoscopic Perforator Surgery Registry. The North American Study Group. J Vasc Surg 1999;29:489-502.
29. Hauer G, Bergan JJ, Werner A, Mitterhusen M, Nasralla F. Development of endoscopic dissection of perforating veins and fasciotomy for treatment of chronic venous insufficiency. Ann Vasc Surg 1999;13:357-64.
30. Illig KA, Shortell CK, Ouriel K, Greenberg RK, Waldman D, Green RM. Photoplethysmography and calf muscle pump function after subfascial endoscopic perforator ligation. J Vasc Surg 1999;30:1067-76.
31. Nelzen O. Prospective study of safety, patient satisfaction and leg ulcer healing following saphenous and subfascial endoscopic perforator surgery. Br J Surg 2000;87:86-91.
32. Rhodes JM, Gloviczki P, Canton L, Heaser TV, Rooke TW. Endoscopic perforator vein division with ablation of superficial reflux improves venous hemodynamics. J Vasc Surg 1998;28:839-47.
33. Bradbury AW, Ruckley CV. Foot volumetry can predict recurrent ulceration after subfascial ligation of perforators and saphenous ligation. J Vasc Surg 1993;18:789-95.
34. Perthes G. Über die Operation der Unterschenkelvarizen nach Trendelenburg. Dtsch Med Wschr 1895;21:253-7.
35. Burnand KG, O'Donnell Jr. TF, Thomas ML, Browse NL. The relative importance of incompetent communicating veins in the production of varicose veins and venous ulcers. Surgery 1977;82:9-13.
36. Akesson H, Brudin L, Cwikiel M, Ohlin P, Plate G. Does the correction of insufficient superficial and perforating veins improve venous function in patients with deep venous insufficiency? Phlebology 1990;5:113-23.
37. Scriven JM, Bianchi V, Hartshorne T, Bell PR, Naylor AR, London NJ. A clinical and hemodynamic investigation into the role of calf perforating vein surgery in patients with venous ulceration and deep venous incompetence. Eur J Vasc Endovasc Surg 1998;16:148-52.
38. Fitridge RA, Dunlop C, Raptis S, Thompson MM, Leppard P, Quigley F. A prospective randomized trial evaluating the hemodynamic role of incompetent calf perforating veins. Aus NZJ Surg 1999; 69:214-6.
39. Sarin S, Scurr JH, Coleridge Smith PD. Stripping of the long saphenous vein in the treatment of primary varicose veins. Br J Surg 1994;81:1455-8.
40. Recek C, Auswirkung der Krossektomie auf die venöse Hämodynamik bei primärer Varikose. Phlebologie 1996;25:11-8.
41. McMullin GM, Coleridge Smith PD, Scurr JH. Objective assessment of high ligation without stripping the long saphenous vein. Br J Surg 1991;78:1139-42.
42. Neglen P, Einarsson E, Eklof B. The functional long-term value of different types of treatment for saphenous vein incompetence. J Cardiovasc Surg 1993;34:295-301.
43. Ik Kim D, Boong Lee B, Bergan JJ. Venous hemodynamic changes after external banding valvuloplasty with varicosectomy in the treatment of primary varicose veins. J Cardiovasc Surg 1999;40:567-70.
44. Koyano K, Sacaguchi S. Selective stripping operation based on Doppler ultrasonic findings for primary varicose veins of the lower extremities. Surgery 1988;103:615-9.
45. Christopoulos D, Nicolaides AN, Galloway JM, Wilkinson A. Objective noninvasive evaluation of venous surgical results. J Vasc Surg 1988; 8:683-7.
46. Recek C. Saphenareflux als Ursache der venösen Zirkulationsstörung bei primärer Varikose mit chronischer Veneninsuffizienz. Acta Chir Austriaca 1998;2:76-7.
47. Padberg FT Jr., Pappas PJ, Araki CT, Back TL, Hobson RW. Hemodynamic and clinical improvement after superficial vein ablation in primary combined venous insufficiency with ulceration. J Vasc Surg 1996;26:169-71.
48. Ting AC, Cheng SW, Wu LL, Cheung GC. Changes in venous hemodynamics after superficial vein surgery for mixed superficial and deep venous insufficiency. World J Surg 2001; 25:122-5.