

Eur J Vasc Endovasc Surg 28, 257–261 (2004)

doi: 10.1016/j.jevs.2004.06.002, available online at <http://www.sciencedirect.com> on 

Segmental Hypoplasia of the Great Saphenous Vein and Varicose Disease

A. Caggiati^{1*} and E. Mendoza²¹Department of Anatomy, University "La Sapienza", Rome, Italy; and ²Private Office, Wunstorf, Germany

Objectives. Primitive narrowing of great saphenous vein segments (saphenous hypoplasia) has been described in healthy limbs. The aim of the present study was to detect great saphenous vein segmental hypoplasia in limbs with varicose veins and to evaluate the local anatomical and haemodynamic patterns.

Materials and Methods. The incidence of saphenous hypoplasia and the local haemodynamic rearrangement were evaluated by duplex ultrasonography in 676 normal limbs and in 320 limbs with varicose veins.

Results. Segmental hypoplasia was demonstrated in 84 normal limbs and in 79 limbs with saphenous reflux. In the latter, the retrograde flow leaves the GSV at the proximal end of the hypoplastic segment to feed tributary veins.

Conclusions. Saphenous hypoplasia occurs in varicose limbs more frequently than in healthy ones ($p = > 0.001$). It greatly influences the path of the reflux and the anatomy of the varicose veins. GSV segmental hypoplasia can be detected preoperatively by duplex ultrasonography. Its occurrence may influence surgical management for two main reasons: in about 68% of varicose limbs with segmental hypoplasia, the distal GSV is competent. If the distal GSV is varicose, its size and flow direction is normalised by treating the accessory vein that bypasses the hypoplastic segment.

Key Words: Saphenous vein; Aplasia; Varicose disease; Anatomy; Surgery.

Introduction

The calibre of the healthy great saphenous vein (GSV) is extremely variable,¹ depending upon physiological factors (age, volume of the subcutaneous layer, sympathetic vasoconstrictor activity, muscular exercise) and ill-defined constitutional circumstances. In addition, the calibre of the GSV mildly and progressively increases from the ankle to the groin due to the confluence of tributaries.

Despite these individual physiological differences, drastic reduction in calibre of GSV segments was demonstrated in normal limbs by stereomicroscopy and light microscopy.² These segments coursed between normally sized tracts of the GSV, which were also connected by a parallel longitudinal vein (saphenous accessory vein).³ This segmental abnormality of GSV calibre was named 'aplasia',⁴ because histology revealed that the thin wall of these segments was similar in structure to that of the embryonic GSV.⁵ In this article we have used the term 'hypoplasia' to emphasise the point that the vein is present but of very small dimensions.

The aim of the present study was to assess the frequency of segmental GSV hypoplasia in varicose limbs using duplex ultrasonography. In addition, the anatomical and haemodynamic rearrangement occurring at the level of the hypoplastic saphenous vein was also studied.

Materials and Methods

Venous anatomy and flow in the GSV system was evaluated in 996 limbs by duplex ultrasonography. All subjects gave their informed consent to participate to the study, which was approved by the local ethics committees.

Control group

Six hundred and seventy-six limbs (441 limbs in women; median age 51, range 18–79, 235 limbs in men; median age 41, range 18–78) with no reflux in the GSV and no clinical sign of venous insufficiency (C0) were included in the control group. We excluded from this group: (i) healthy limbs from patients with unilateral varicose veins. (ii) Limbs with duplex or clinical evidence of any kind of vascular disorder of

*Corresponding author. Dr A. Caggiati, MD, PhD, Department of Anatomy, University "La Sapienza", Via Borelli 50, 00161 Rome, Italy. Email address: alberto.caggiati@uniroma1.it

the lower limbs. (iii) Limbs previously having undergone orthopaedic or plastic. (iiii) Limbs with post traumatic trophic changes of the skin and subcutaneous layers.

Varicose limbs

Five hundred and forty-eight limbs with varicose veins in 458 consecutive patients were considered for inclusion in our study. Eighty-three limbs were excluded on the basis of a preliminary questionnaire, which showed previous surgical treatment or sclerotherapy, superficial phlebitis or deep vein thrombosis. One hundred and forty-five limbs were excluded on the basis of duplex ultrasonography, which showed unexpected previous venous thrombosis (eight limbs) or competence of the sapheno-femoral junction (SFJ) (137 limbs). In the latter, varicose veins were due to pelvic reflux (32 limbs), an incompetent saphenopopliteal junction (47 limbs), an incompetent perforating vein (nine limbs) or varicose changes were limited to saphenous accessories (49 limbs).⁶

The remaining 320 varicose limbs with GSV reflux due to SFJ incompetence (246 women; median age 53, range 21–78. Seventy-four men; median age 52, range 19–76. CEAP score ranged between C1 and C4) were finally included in our study.

Duplex examination was performed in the standing position. Competence of the GSV was evaluated by the Valsalva manoeuvre, manual calf squeeze—release manoeuvres and during movements of the forefoot ('Wunstorfer' manoeuvre).⁷ Reflux was defined as a retrograde flow lasting longer than 0.5 sec⁶ in response to any of the above tests.

The χ^2 -test was used to compare the incidence of GSV hypoplasia in healthy and varicose limbs.

Discrimination of GSV and accessory veins—identification of GSV hypoplasia

The GSV and its accessories were identified on the basis of their different planar anatomy and fascial relationships according with the recommendations of an International joint Committee of Anatomists, Phlebologists and Surgeons.³ The GSV runs deeply in the subcutaneous tissue, close to the muscular fascia (Fig. 1(A)). It is enclosed by a hyperechoic connective tissue lamina, the saphenous fascia. The saphenous and muscle fasciae delimit a flat space, the saphenous compartment, in which the saphenous vein is found accompanied by related nerves.⁸ Accessory saphenous veins course parallel to the GSV in a more superficial

plane of the subcutaneous tissue, out of the saphenous compartment.⁹

Segmental GSV hypoplasia was diagnosed when no ascending vein could be clearly identified within the saphenous compartment.² The haemodynamic rearrangement occurring at the level of the hypoplastic segments was accurately evaluated by duplex ultrasonography in both normal and varicose systems.

Results

Normal limbs

Hypoplastic segments were found in 84/676 limbs with a competent GSV (12%). In these cases, blood flow bypassed the hypoplastic segments through large accessory veins (Fig. 1). These started at the distal end of the hypoplastic segment and reached the proximal and normally sized tract of the GSV (Fig. 2(A)). In three of these limbs (3.5%), the distal GSV ended in a perforating vein (Fig. 2(B)), and the proximal normally sized GSV was fed by one or more accessory veins.

The distribution of hypoplastic segments along the GSV was as follows: in four limbs only the calf portion of the GSV was hypoplastic (Fig. 2(C)). In 78 limbs the hypoplasia extended from the upper portion of the calf over the knee, ending at any point in the thigh. In two cases, hypoplasia was found only in the thigh. In two limbs, hypoplasia extended up to the groin, where the ascending flow reached the femoral vein by the anterior accessory great saphenous vein (AAGSV) (Fig. 2(D)).

Varicose limbs

Segmental hypoplasia was diagnosed in 79 of 320 varicose limbs with incompetent GSV (25%). The distribution of hypoplastic segments along the GSV was as follows: In seven limbs, only the leg portion of the GSV was hypoplastic. In 71 limbs the hypoplasia extended from the upper portion of the calf over the knee, ending at any point of the thigh. In one case, hypoplasia was found only in the thigh.

Path of the reflux

In one case (1.2%), the upper portion of the GSV was hypoplastic and the reflux descended from the SFJ along the AAGSV to join the GSV at the mid thigh. In all other cases, the reflux originating from the SFJ descended along the GSV to be diverted in an accessory vein at the cranial end of the hypoplastic

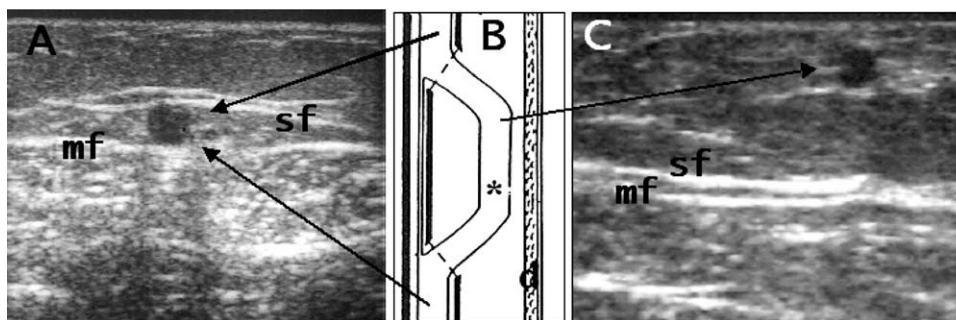


Fig. 1. Saphenous hypoplasia in healthy GSV. (A) The normal GSV courses between the muscular fascia (mf) and the saphenous fascia (sf). (B) At the level of an hypoplastic segment, the flow is shunted in a parallel accessory saphenous vein (*) which runs just below the dermis (d). (C) US at the level of an hypoplastic segment. The space between the saphenous fascia (sf) and the muscular fascia (mf) is thin and a large accessory vein runs more superficially.

segment (Fig. 3). In 25/79 of these limbs (32%), the accessory vein rejoined the distal GSV, which appeared varicose and showed reflux on duplex ultrasonography (Figs. 4(A) and 5). In contrast, in 54/79 of these limbs (68%), the GSV portion distal to the hypoplastic segment was healthy because the reflux passed from the accessory vein to a perforator (27 limbs, 34%) (Fig. 4(B)), to other non-saphenous veins (18 limbs, 23%) (Fig. 4(C)), or, in nine limbs (11%), to the short saphenous vein (SSV) trunk (Fig. 4(D)).

Discussion

The findings presented above demonstrate that segmental GSV hypoplasia occurs in limbs with varicose

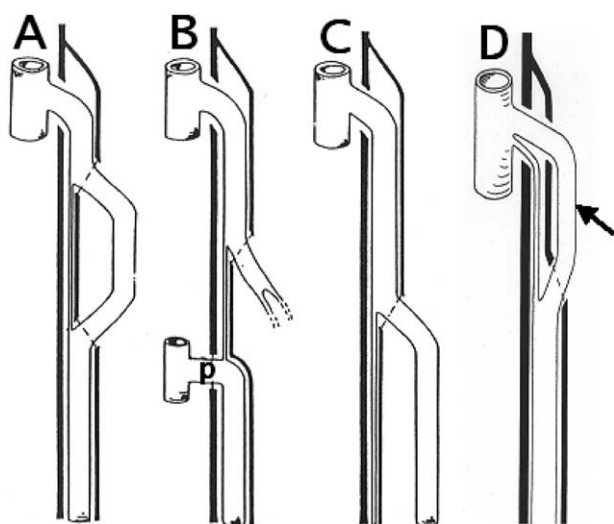


Fig. 2. Saphenous hypoplasia. (A) the hypoplastic segment interconnects normally sized tracts of the GSV. (B) The distal GSV drains into a perforator (p). (C) Hypoplasia of the leg portion of the GSV. (D) Hypoplasia of the upper thigh and groin portions of the GSV, which are bypassed by the anterior accessory great saphenous vein (arrow).

veins with SFJ incompetence more frequently than in healthy ones (25% vs. 12%), ($p = > 0.001$, Chi-squared). In addition, segmental GSV hypoplasia greatly influences the path of the reflux and, consequently, the gross anatomy of the resulting varicose veins.

In fact, the hypoplastic segments do not permit venous reflux. At the proximal limit of the hypoplastic segment reflux passes into superficial saphenous tributaries. In 17% of all varicose limbs (68% of varicose limbs with hypoplasia), diversion of venous reflux preserves the distal GSV from varicose changes, appearing competent and undilated. In 7.8% of all varicose limbs (32% of varicose limbs with hypoplasia), the distal GSV was varicose because it was reached by the reflux descending along the incompetent accessory vein. Finally, in 2.2% of all limbs with varicose veins (11% of varicose limbs with GSV hypoplasia), reflux originating at the SFJ reached the SSV trunk.

The higher incidence of segmental GSV hypoplasia in the patients with clinically apparent varicose veins suggests a possible role of this phenomenon in the pathogenesis of varices. The only mechanism that could be hypothesised on the basis of these preliminary report, is the haemodynamic overload of saphenous accessories occurring at the level of hypoplastic segments. In fact, the wall of saphenous accessory veins is thinner and contains less muscle than the GSV.⁹ In addition, these veins are surrounded only by a yielding layer of fat that cannot counteract vessel dilation. In contrast, the GSV runs closely encased by the two unyielding connective tissue layers of the saphenous compartment that work as a shield against vessel dilation.^{8,10}

In normal limbs segmental hypoplasia of the GSV leads to haemodynamic overload of saphenous accessories but the result is only a physiological compensatory dilation in the tributaries. In limbs predisposed to varicose disease, the overload in accessory saphenous veins is greater and results in varicose changes

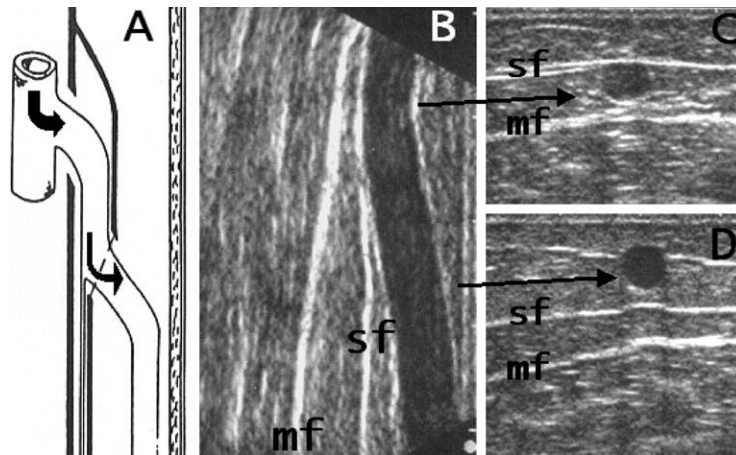


Fig. 3. Saphenous hypoplasia in a varicose limb. (A) The reflux originated at the SFJ descends along the GSV down to the upper end of the hypoplastic segment where it is shunted in a more superficial accessory vein. (B) the corresponding US. (C) and (D) Transverse US scanning clearly demonstrates the different planes in which the GSV (C) and its accessory vein (D) run; sf, saphenous fascia; mf, muscular fascia.

(dilation and tortuosity), that are greater, earlier and clinically more evident than those occurring in the incompetent GSV.¹¹

The aetiology of GSV hypoplasia is still unknown. However, histological findings² and the fact that GSV segmental hypoplasia occurs in normal limbs without history of previous phlebitis or treatment indicate that it is due to a primitive defect of development. The pathogenesis of GSV hypoplasia could be attributed to a mechanism similar to that responsible for the disappearing of the thigh portion of the small saphenous vein.¹⁰

Despite its high incidence, GSV hypoplasia has been reported infrequently in literature. This is probably due to two main facts: (i) hypoplastic segments are not visible on duplex ultrasonography; (ii) the accessory vein that

accompanies and bypasses the hypoplastic segment is often erroneously considered to be the GSV if the unless rigorous anatomical criteria for GSV identification (topography and fascial relationships) are taken into account.³ However, different degrees of abnormality of the GSV calibre have been reported by ultrasonography and venography with an incidence ranging between 16% and 42%.^{2,12-15}

Clinical implications

GSV segmental hypoplasia can be demonstrated pre-operatively by duplex ultrasonography. Its occurrence may influence surgical decisions for two main reasons: Firstly, in about 17% of limbs with saphenous reflux

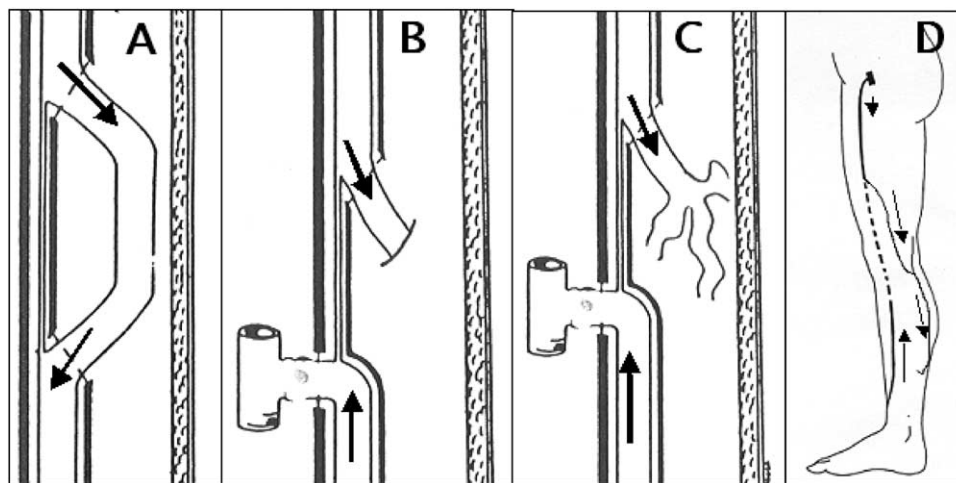


Fig. 4. The fate of the reflux. Once shunted in the accessory vein that bypasses the hypoplastic GSV, the reflux is transmitted to the distal GSV (A), to a perforator (B), to other superficial veins (C), or, finally, to the Small Saphenous Vein by a Giacomini's anastomosis (D). In B, C and D the distal GSV is competent.



Fig. 5. Saphenous hypoplasia in a varicose limb. (A) 3D CT-venography. The hypoplastic segment of the GSV is clearly visible (arrow). The accessory vein that bypasses the hypoplastic segment is markedly tortuous. (B) The correspondent US. The hypoplastic segment of the GSV (arrow) can be visualized by high frequency probes (14 MHz) between the muscular fascia (mf) and the saphenous fascia (sf).

(68% of those with hypoplasia), the distal GSV is healthy. Secondly, due to the small calibre, hypoplastic segments may stop the progression of any kind of endoluminal device (traditional strippers, endovascular laser or radiofrequency probes).¹⁶ Surgical removal of varicose veins will be achieved more easily by introducing the stripper directly into the refluxing accessory vein, limiting the treatment to this vein and to the proximal incompetent GSV. In these cases, the distal GSV can be left untreated because the surgical or endovascular treatment of the refluxing accessory vein allows the main saphenous trunk to normalise its size and flow direction.¹⁷

When laser or radiofrequency treatments are

performed in saphenous accessories that bypass hypoplastic GSV segments, particular care must be taken in order to avoid thermal skin injury due to their close relationship with the dermis. Treatment of saphenous accessory veins is less likely to result in neurological damage, because the saphenous nerve always lies deeply in the saphenous compartment,³ even when the GSV is hypoplastic.²

References

- 1 KUPINSKI AM, EVANS SM, KHAN AM, ZORN TJ, DARLING III RC, CHANG BB, LEATHER RP, SHAH DM. Ultrasonic characterization of the saphenous vein. *Cardiovasc Surg* 1993; **1**:513–517.
- 2 CAGGIATI A, RICCI S. The caliber of the human long saphenous vein and its congenital variations. *Anat Anz* 2000; **182**:195–201.
- 3 CAGGIATI A, BERGAN JJ, GLOVICZKI P, JANTET G, WENDELL-SMITH CP, PARTSCH H. Nomenclature of the veins of the lower limbs: an international interdisciplinary consensus statement. *J Vasc Surg* 2002; **36**:416–422.
- 4 Churchill's Medical Illustrated Dictionary. New York: Churchill Livingstone Inc; 1994.
- 5 KOKOVA J, HORAKOVA M, HORAKOVA MA. The development of pre- and post-natal veins. *Phlebologie* 1993; **46**:241–251.
- 6 LABROPOULOS N, TIONGSON J, LANDON PRYOR BS, TASSIOPOULOS AK, KANG SS, MANSOUR MA, BAKER WH. Nonsaphenous superficial vein reflux. *J Vasc Surg* 2001; **34**:871–877.
- 7 MENDOZA E, BERGER HA. Provokationsmanöver für die duplexsonographische Diagnostik der Varikosis. *Gefässchirurgie* 2001; **6**: 43–46.
- 8 CAGGIATI A. Fascial relationships of the long saphenous vein. *Circulation* 1999; **100**:2547–2549.
- 9 CAGGIATI A. Fascial relations and structure of the tributaries of the saphenous veins. *Surg Radiol Anat* 2000; **22**:191–196.
- 10 CAGGIATI A. Fascial relationships of the short saphenous vein. *J Vasc Surg* 2001; **34**:241–246.
- 11 COTTON LT. Varicose veins. Gross anatomy and development. *Br J Surg* 1961; **48**:589–598.
- 12 RUOFF BA, CRANLEY JJ, HANNAN LA, ASEFFA N, KARKOW WS, STEDJE KG, CRANLEY RD. Real-time duplex ultrasound mapping of the greater saphenous vein before *in situ* infrainguinal revascularization. *J Vasc Surg* 1987; **6**:107–113.
- 13 LEOPOLD PW, SHANDALL A, KUPINSKI AM, CHANG BB, KAUFMAN J, SHAH DM, LEATHER RP. Role of B-mode venous mapping in infrainguinal *in situ* vein-arterial bypasses. *Br J Surg* 1989; **76**: 305–307.
- 14 VAN DIJK LC, WITTENS CHA, PIETERMAN H, VAN URK H. The value of pre-operative ultrasound mapping of the greater saphenous vein prior to closed *in situ* bypass operations. *Eur J Radiol* 1996; **23**:235–237.
- 15 VEITH FJ, MOSS CM, SPRAYREGEN S, MONTEFUSCO C. Preoperative saphenous venography in arterial reconstructive surgery of the lower extremity. *Surgery* 1979; **85**:253–256.
- 16 BERGAN JJ, KUMINS NH, OWENS EL, SPARKS SR. Surgical and endovascular treatment of lower extremity venous insufficiency. *J Vasc Interv Radiol* 2002; **13**:563–568.
- 17 CRIADO E, LUJAN S, IZQUIERDO L, PURAS E, GUTIERREZ M, FONTCUBERTA J. Conservative hemodynamic surgery for varicose veins. *Semin Vasc Surg* 2002; **15**:27–33.

Accepted 2 June 2004