



Haemodynamic Management of Deep Venous Insufficiency

9

Sergio Giancesini, Erika Mendoza,
and Paolo Zamboni

9.1 Introduction

Femoral vein (FV) duplication is a higher prevalent anatomical variant than what generally thought [1, 2]. In primary and post-thrombotic reflux patterns, the duplication feeds a closed circuit with one incompetent vessel constituting the leaking point and with the other conduit representing the antegrade draining route. The described pattern offers the opportunity of suppressing the deep venous reflux by means of a surgical closure of the leaking point, using haemodynamic principles to restore a physiological venous drainage.

S. Giancesini, M.D., Ph.D.
Vascular Diseases Center, University of Ferrara,
Ferrara, Italy

USUHS University, Bethesda, MD, USA

E. Mendoza, M.D., Ph.D.
Venenpraxis, Wunstorf, Germany
e-mail: erika.mendoza@t-online.de

P. Zamboni, M.D. (✉)
School of Vascular Surgery and Vascular
Diseases Center, University of Ferrara,
Ferrara, Italy

Unit of Translational Surgery, AOU Ferrara,
Ferrara, Italy
e-mail: zmp@unife.it

9.2 Applied Deep Venous Anatomy

Lower limb deep venous anatomy plays a major role in venous return, thanks to the interaction with the muscle masses creating the so-called calf venous pump [3]. Indeed, while the venous return in a supine position is mainly triggered by the pressure gradient created by the cardiac pump and by the thoraco-abdominal aspiration, in the standing position, the venous drainage counteracts the force of gravity by activating the lower limb muscle, thus squeezing the veins and propelling the blood.

It is interesting to notice that four-legged animals do not possess a calf pump mechanism: an evidence of the role of the gravitational force and of the synergistic action of lower limb veins and muscles. The interconnection among the deep and the superficial venous system is functionally separated by the two fascial layers: the deep and superficial fascia. While the first is a strong membrane with little elasticity, the latter is softer and provides less support to the saphenous system which lies in between the same two fascial structures (see Sect. 2.2).

The deep venous system below the knee is constituted by the anterior and posterior tibial veins, the peroneal and popliteal veins (see Fig. 2.1) [4]. The anterior tibial vein is the continuation of the venae concomitantes of the dorsalis pedis artery, and it drains the anterior part of the lower limb. It

runs just above the interosseous membrane between the tibia and the fibula.

The posterior tibial vein generates at the confluence of the medial and lateral plantar veins below the medial malleolus, and it drains the posterior lower part of the leg together with the plantar surface of the foot. This vein runs behind the tibia and joins the popliteal vein at the posterior knee.

The peroneal vein drains the lateral part of the lower leg while ascending along the posteromedial side of the fibula and joins the posterior tibial vein in the upper third of the lower leg. The anterior and posterior tibial veins unite together forming the tibio-peroneal trunk which then generates the popliteal vein at the lower aspect of the posterior knee.

The popliteal vein then ascends along the anteromedial part of the thigh, medial to the artery in the lower knee, superficial to the artery at the posterior knee and lateral to it above the knee. At the adductor hiatus, the popliteal vein becomes the femoral vein. The term superficial femoral vein is no longer in use considering the potential misunderstanding as the vein is deep [5].

In the lower part, the femoral vein is lateral to the artery, in the middle third behind and in the upper portion medially. The deep femoral vein joins the femoral vein running along the deep femoral artery, so forming the common femoral vein which is located medially to the common femoral artery. Once the common femoral vein has passed underneath the inguinal ligament, it becomes the external iliac vein.

From a structural point of view, the deep veins are thin walled and have little smooth muscle. They are present also as sinuses collecting significant amount of blood volume, such as in the soleal (draining into the posterior tibial vein) and gastrocnemius (draining into the popliteal vein) muscles. Deep veins are subfascial and are imbedded inside the muscle masses, and they play a fundamental role in venous return, particularly during standing.

Indeed, the cardiac and thoraco-abdominal pumps are insufficient in the generation of the pressure gradient that drives the venous blood back to the heart against the force of gravity. Together with the valvular apparatus and the

muscle masses, the deep veins constitute the so-called peripheral heart: a main actor in venous drainage regulation. Like in the heart, the venous blood is collected in the deep venous network and then propelled by the muscles through a valvular apparatus towards against the force of gravity.

The deep veins receive the full transmission of the energy generated by the systolic contraction thanks to their anatomical location just inside the muscle masses. Thanks to the valvular apparatus the blood is propelled in one direction.

The most superficial network is solicited by a smaller pressure gradient because of its localization above the muscular fascia. Subsequently, an energy gradient differential is generated between the deep and superficial system, so favouring the drainage from the most superficial towards the deepest compartment, from the distal to the proximal parts. In case of valvular failure, a deep venous reflux can originate, presenting different possible networks of pathological drainage.

9.3 Rationale for the Haemodynamic Management and Clinical Scenario

Literature is clearly showing how descriptive anatomy must take into consideration the extremely frequent variations in the course and number of lower limb veins [6]. In particular, duplication of deep veins has been found in 42% of popliteal veins and in 31% of femoral veins (see Fig. 9.1).

The duplicated vessel has been called accessory femoral vein. It was found to be medial to the femoral vein in 46% of cases while lateral in 49% of patients. The remaining 5% of cases were triplications. The average length of the duplicated femoral vein was reported to be in between 6 and 15 cm. No correlation among gender, age and duplication was found, while the presence of a duplication on one leg was strongly associated with the presence of a duplication in the contralateral limb [7].

The same vessel duplication predisposes a network potentially developing a reflux through a closed circuit (Fig. 9.2).

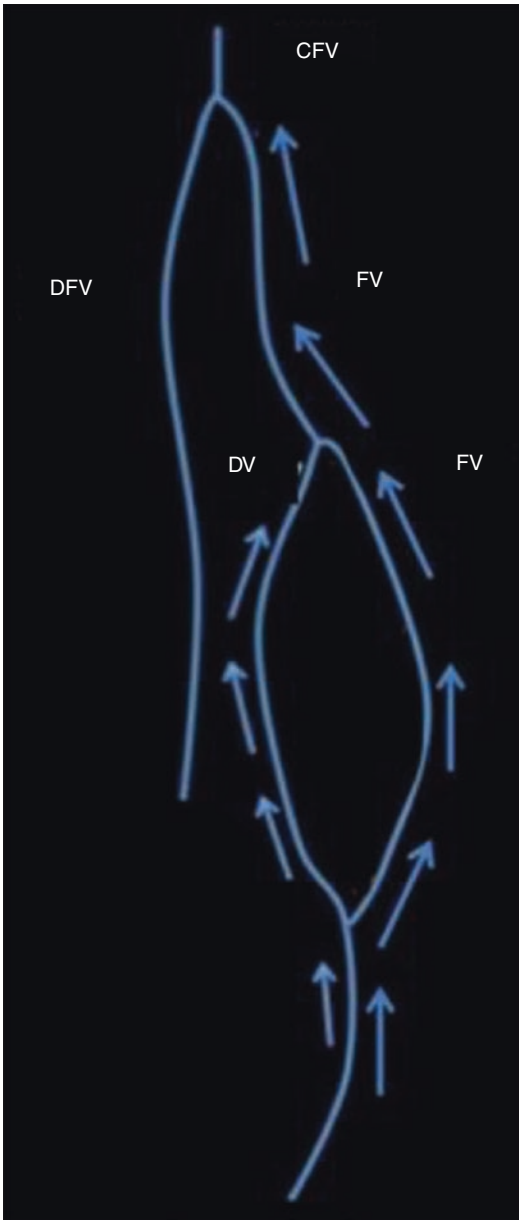


Fig. 9.1 Duplication (DV) of the femoral vein (FV). *DFV* deep femoral vein, *CFV* common femoral vein (With permission from [8])

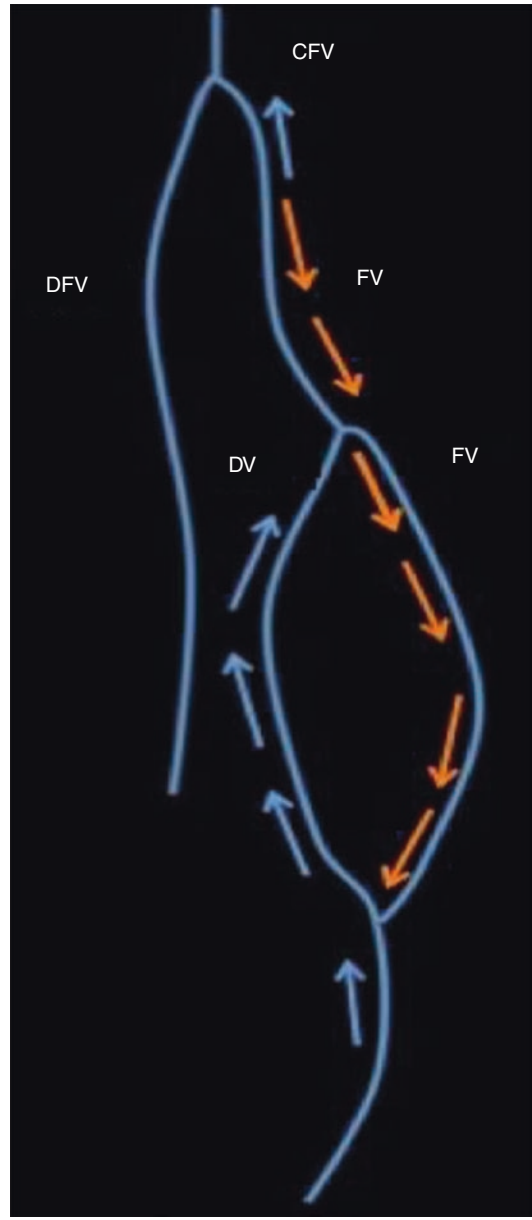


Fig. 9.2 Closed refluxing network generated by the incompetence at the confluence among the femoral vein (FV) and its duplication (DV). *CFV* common femoral vein, *DFV* deep femoral vein (With permission from [8])

As previously described in Sect. 3.7.1, dedicated to the superficial system, a reflux generated inside a closed circuit (closed shunt) can be suppressed by adequately changing the pressure gradient through a selective ligation of the leaking point.

In the case described in Fig. 9.2, for example, a simple titanium clip apposition at the

confluence among the femoral vein and its duplication can restore the venous drainage by creating a favourable pressure gradient, thanks to the suppression of the leaking point (Fig. 9.3).

Published data show the feasibility and effectiveness of the technique [8].

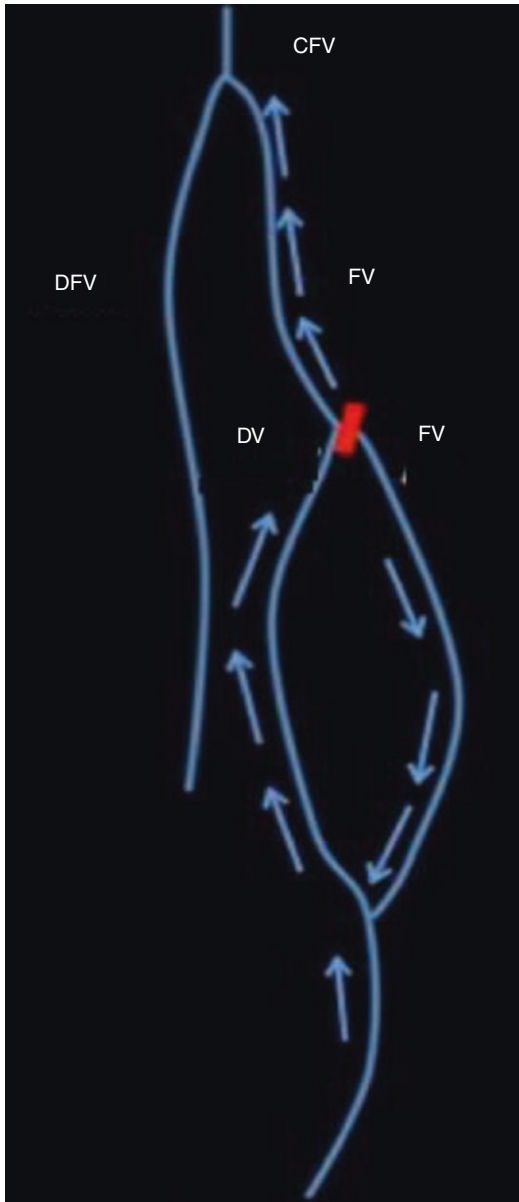


Fig. 9.3 Suppression of the leaking point by titanium clip apposition (red line). Breaking the close circuit established by the femoral vein (FV) duplication (DV) leads to the reflux suppression. *DFV* deep femoral vein, *CFV* common femoral vein (With permission from [8])

As initially postulated back in the early twentieth century by Linton and Bauer, femoral ligation seems to be not only safe but even effective in deep venous reflux control [9].

The frequent presence of deep venous duplication creates an anatomical premise for suppressing refluxes that are originated by closed circuits while maintaining the upward flow in the competent segment. Lower limbs' deep venous system constitutes one of the most fascinating but challenging topic in the vascular disease field.

Despite the high frequency of venous reflux occurrence, the related pathophysiology is still lacking of a clear interpretation [10–14].

Nowadays, therapeutic options such as valvuloplasty, transpositions and translocations are utilized in a limited number of cases and just in highly specialized centres [15, 16].

The feasibility and performance of a haemodynamic approach to deep venous insufficiency allow a potential widening of the indication to treatment, thanks to both the venous duplication frequency and the surgical easy feasibility. As it was demonstrated in case of haemodynamic restoration in the superficial venous insufficiency, the suppression of the leaking point of a closed refluxing circuit reduces the ambulatory venous pressure and improves the plethysmographic parameters [17].

The same haemodynamic improvement follows also in the deep venous system. An active muscle pump action is required to generate the post-operative retrograde draining flow. For this reason, this haemodynamic procedure is suggested just in the case of not bedridden patients, who have previously failed conservative measure and with a severe deep reflux affecting the possible ulcer healing.

9.4 Instructions for Users

9.4.1 Preoperative Diagnostics

The ultrasound assessment in colour identifies a deep venous reflux along a duplicated vein, with the leaking point at the vessels bifurcation (Fig. 9.4).

A magnetic resonance venography (MRV) protocol can be customized in order to detect the

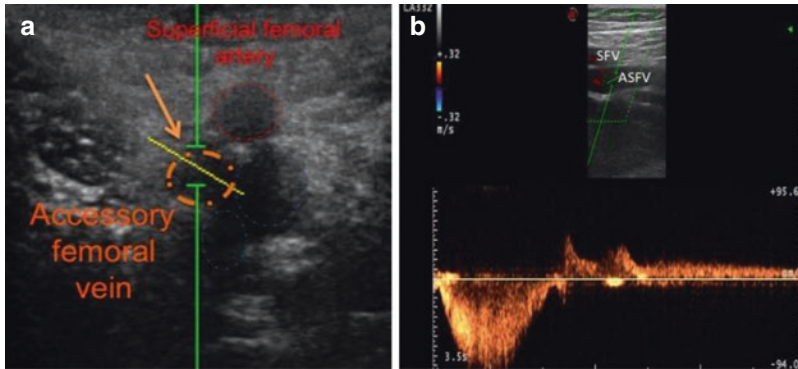


Fig. 9.4 (a) Duplicated femoral vein (orange dotted line, accessory femoral vein) at the confluence with the femoral vein. (b) Reflux documentation with the ultrasound in PW mode (With permission from [8])

slow flow of the deep venous system, providing further evidence of this anatomical bifurcation.

9.4.2 Surgical Technique

The patient lies supine with a leg flexed at the knee and in abduction (frog-legged), in order to facilitate the access to the medial side of the thigh.

Under local anaesthesia and after an accurate preoperative echo-guided mapping, a longitudinal incision is performed along the medial aspect of the upper thigh over the anterior border of the sartorius muscle, and dissection between sartorius and the medial edge of the vastus medialis muscle is performed to expose the femoral vein, the duplicated (accessory femoral) vein and the femoral artery. The vessels are isolated and controlled, mobilizing the femoral artery additionally in order to get a better exposure of the femoral venous bifurcation. A titanium clip (large size) is flush applied at the confluence among the femoral vein and its duplication (Fig. 9.5).

The dissection layers are closed by running 3–0 absorbable sutures, while the skin by a subcuticular 4–0 absorbable monofilament.

Neither antibiotics nor LMWH prophylaxis is considered mandatory. Only in post-thrombotic cases an anticoagulation therapy is suggested because of increased deep venous thrombosis risk.

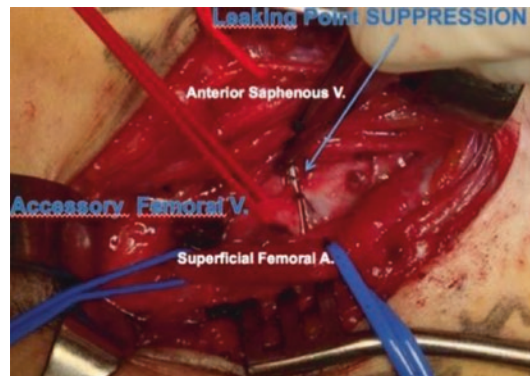


Fig. 9.5 Titanium clip application at the duplicated femoral vein confluence with the femoral vein

9.5 Haemodynamic Management of Deep Venous Insufficiency at the Calf

The same rationale as described in Fig. 9.3 can be applied to a selective reflux situation at the calf. The most often observed situations are:

- Refluxing muscle veins with connection to the distal small saphenous vein. The treatment is to interrupt the muscle vein at its junction with the popliteal vein in the popliteal fossa (see Fig. 9.6a).
- Refluxing posterior tibial vein with connection to the distal great saphenous vein. The

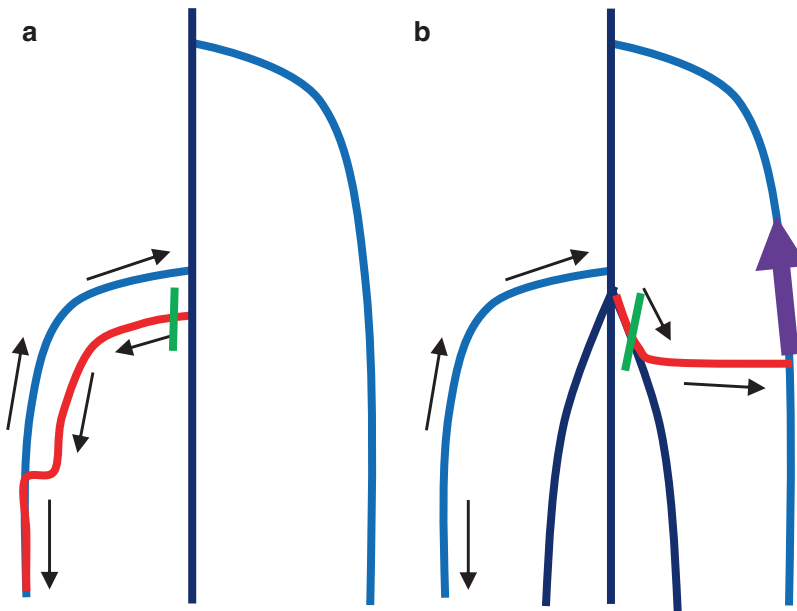


Fig. 9.6 (a) Reflux via a muscle vein, joining the distal small saphenous vein (compare Fig. 4.31). The treatment is to interrupt the refluxing muscle vein (red) at its junction with the popliteal vein in the popliteal fossa (green line). (b) Reflux in the posterior tibial vein, filling the

great saphenous vein via a paratibial perforator (Boyd). The deep reflux is drained via an antegrade but overloaded GSV (violet arrow) (compare Fig. 4.34). The treatment is to interrupt the posterior tibial vein at the junction with the popliteal vein

treatment is to interrupt the posterior tibial vein at its junction with the popliteal vein in the popliteal fossa (see Fig. 9.6b).

The surgical access in both cases is the same as to perform an interruption of the sapheno-popliteal junction. In the hands of an experienced surgeon, the intervention is safe.

Literature

1. Casella IB, Presti C, Yamazaki Y, Vassoler AA, Furuya LA, Sabbag CD. A duplex scan-based morphologic study of the femoral vein: incidence and patterns of duplication. *Vasc Med*. 2010;15:197–203.
2. Dona E, Fletcher JP, Hughes TM, Saker K, Batiste P, Ramanathan I. Duplicated popliteal and superficial femoral veins: incidence and potential significance. *Aust N Z J Surg*. 2000;70:438–40.
3. Goldman MP. Anatomy and pathophysiology of varicose veins. *J Dermatol Surg Oncol*. 1989;15:138–45.
4. Meissner M. Lower extremity venous anatomy. *Semin Intervent Radiol*. 2005;22(3):147–56.
5. Caggiati A, Bergan JJ, Gloviczki P, Eklof B, Allegra C, Partsch H, et al. Nomenclature of the veins of the lower limb: extensions, refinements, and clinical application. *J Vasc Surg*. 2005;41:719–24.
6. Park EA, Chung JW, Lee W, Yin YH, Ha J, Kim SJ, Park JH. Three-dimensional evaluation of the anatomic variations of the femoral vein and popliteal vein in relation to the accompanying artery by using CT venography. *Korean J Radiol*. 2011;12(3):327–40. <https://doi.org/10.3348/kjr.2011.12.3.327>. Epub 2011 Apr 25
7. Quinlan DJ, Alikhan R, Gishen P, Sidhu PS. Variations in lower limb venous anatomy: implications for US diagnosis of deep vein thrombosis. *Radiology*. 2003;228(2):443–8. Epub 2003 Jun 23
8. Zamboni P, Giancesini S. Surgical technique for deep venous reflux suppression in femoral vein duplication. *EJVES Short Rep*. 2016;30:10–12.
9. Glasser ST. Ligation of the femoral vein for chronic occlusive arterial disease. A review of one hundred and eighteen ligations. *Arch Surg*. 1945;50:56–62.
10. Labropoulos N, Tassiopoulos AK, Kang SS, Mansour MA, Littooy FN, Baker WH. Prevalence of deep venous reflux in patients with primary superficial vein incompetence. *J Vasc Surg*. 2000;32:663–8.
11. Robertson LA, Evans CJ, Lee AJ, Allan PL, Ruckley CV, Fowkes FG. Incidence and risk factors for venous

- reflux in the general population: Edinburgh vein study. *Eur J Vasc Endovasc Surg.* 2014;48:208–14.
12. Meissner MH, Manzo RA, Bergelin RO, Markel A, Strandness DE Jr. Deep venous insufficiency: the relationship between lysis and subsequent reflux. *J Vasc Surg.* 1993;18:596–605.
 13. Meissner MH, Moneta G, Burnand K, Gloviczki P, Lohr JM, Lurie F, et al. The hemodynamics and diagnosis of venous disease. *J Vasc Surg.* 2007;46 Suppl S:4S–24S.
 14. Meissner MH. Pathophysiology of varicose veins and chronic venous insufficiency. In: *Comprehensive vascular and endovascular surgery.* Edinburgh: Mosby; 2009. p. 729–48.
 15. Lurie F, Kistner R, Perrin M, Raju S, Neglen P, Maleti O. Invasive treatment of deep venous disease. A UIP consensus. *Int Angiol.* 2010;29(3):199–204.
 16. Goel RR, Abidia A, Hardy SC. Surgery for deep venous incompetence. *Cochrane Database Syst Rev.* 2015;2:CD001097.
 17. Zamboni P. Reflux elimination without any ablation or disconnection of the saphenous vein: a hemodynamic model for venous surgery. *Eur J Vasc Endovasc Surg.* 2001;21(4):361–9.