

INTRODUCTION

Chronic venous disease (CVD) is a common pathology in the general population of adults in both industrialized and developing countries (***Rabe et al., 2012***).

Superficial venous incompetence is a common disorder affecting 25% of women and 15% of men presenting with varicose veins. Venous insufficiency increases with age and is most commonly caused by primary valvular incompetence. The most important factors appear to be heredity, female sex, and previous phlebitis, pregnancy, obesity and use of OCPs, deep venous reflux (***Callam, 1994***).

Poor deep venous function caused by venous reflux, obstruction, or calf muscle pump failure will ultimately lead to an increase in ambulatory venous pressure and recurrence of VV through incompetent perforators, resulting in chronic venous insufficiency. Incompetent perforator veins in patients with venous ulcerations were previously treated with ligation using the open Linton procedure, and now occasionally with subfascial endoscopic perforator surgery (SEPS). More commonly, endovenous thermal ablation or sclerotherapy is the treatment of choice for patients with venous ulcers who have failed conservative compression therapy and require ablation of incompetent perforator veins (***Arafa et al., 2020***).

The most severe form of CVD is venous ulceration with a prevalence of about 1%. Venous ulcers are often painful and affect quality of life negatively (*Evans et al., 1999*).

The standard treatment for active venous ulcer is compression stockings, but the recurrence rate is high (*Nelson and Harrison, 2014*).

In the ESCHAR study the chronic venous ulcer outcomes reported here after ultrasound guided foam sclerotherapy combined with compression appear to be at least as good as those reported after surgery. This suggests that ultrasound guided foam sclerotherapy combined with compression may be an attractive alternative to surgery in this group of patients who are often elderly, frail and refuse (or are refused) operative intervention (*Darvall et al., 2009*).

Ultrasound-guided foam sclerotherapy is a minimally invasive treatment option used for ablation of axial and perforator reflux for chronic venous ulceration (*Grover et al., 2014*).

AIM OF THE WORK

To assess the benefit of ultrasound-guided sclerotherapy (USGS) of incompetent perforator on healing of chronic venous ulcers with deep venous reflux.

Chapter 1

SURGICAL ANATOMY

Understanding of the anatomy of veins and the proper use of terminology are very important to correctly diagnose and treat underlying venous insufficiencies and related varicose veins (*Santler and Goerge, 2017*).

The muscular fascia surrounding the calf and leg muscles separates two compartments: the superficial compartment that consists of all tissues between the skin and the muscular fascia, and the deep compartment, which includes muscles and deep veins that lie beneath the muscular fascia (**Figure 1**). The veins of the lower extremities are divided into three systems: 1) The superficial veins that are in the superficial compartment above the muscular fascia and drain the cutaneous microcirculation. 2) The deep veins that lie beneath the muscular fascia and drain the lower extremity muscles. 3) The perforating veins that penetrate the muscular fascia and connect the superficial and deep veins (*Santler and Goerge, 2017*).

The saphenous fascia is the portion of the membranous layer of the subcutaneous tissue that overlies the saphenous veins. The saphenous fascia is thinner than muscular fascia. The two fascial layers, with the saphenous fascia above and muscularis fascia below, merge at each end to form a closed space, which is called the saphenous compartment (*Gianesini, 2018*).

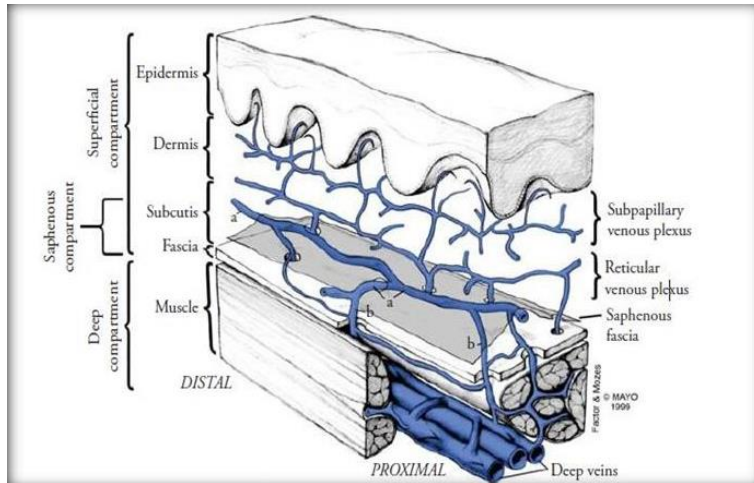


Figure (1): Relationship between fascia and veins of the lower extremity. The fascia is covering the muscle and separating the superficial compartment from the deep one. Superficial veins drain the reticular and subpapillary venous plexuses, and perforating veins connect them to the deep veins (*Mozes and Gloviczki, 2004*).

The Great saphenous vein (GSV)

The GSV is the longest vein in the body; this vein begins at the medial marginal vein of the foot and terminates at the saphenofemoral junction (SFJ) approximately 4 cm below the inguinal ligament. The GSV has a constant terminal valve 1–2mm distal to the SFJ in 94%–100% of the cases (*Yeh et al., 2017*) (**Figure 2**).

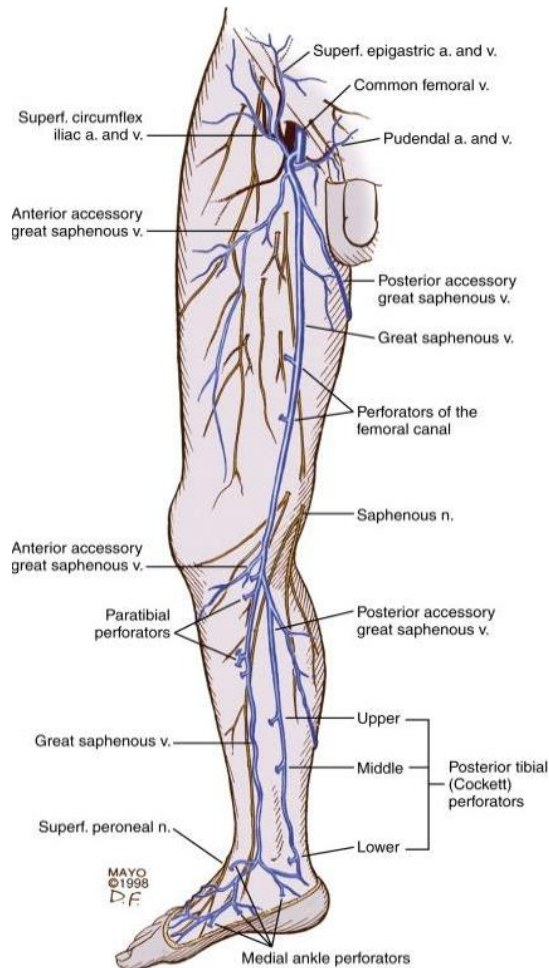


Figure (2): Course of great saphenous vein and its major tributaries (Mozes and Gloviczki, 2004).

Tributary veins to the GSV (Figure 3)

Tributaries are defined as veins that run obliquely or beside the tract of the associated saphenous vein but that are not situated within the saphenous compartment. It includes:

- a. Near termination: the confluence of superficial inguinal veins formed by superficial circumflex iliac vein,

superficial epigastric and external pudendal veins and the distal GSV (*Gianesini, 2018*).

- b.** At the thigh: Anterior accessory and posterior accessory GSV of the thigh (*Gianesini, 2018*).
- c.** At the leg: Anterior accessory and posterior accessory GSV of the leg. The posterior accessory GSV of the leg known as posterior arch vein or Leonardo's vein (*Gianesini, 2018*).
- d.** At the saphenofemoral junction, there could be a variable number of other unnamed tributaries (*Gianesini, 2018*).

The small saphenous vein (SSV)

The SSV begins behind the lateral malleolus as a continuation of the lateral marginal foot vein. It usually has another tributary from the posteromedial side of the ankle. It is close to the sural nerve (*Gianesini, 2018*).

The fascial relationships of the SSV are more stable. The fascia is constant throughout the course of the SSV (*Gianesini, 2018*).

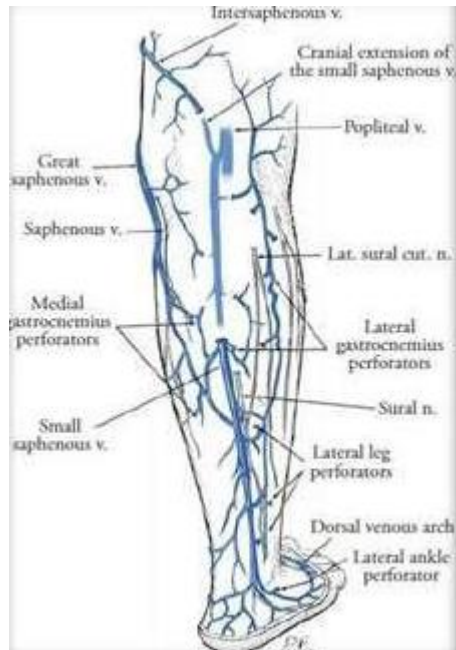


Figure (3): The short saphenous vein and lateral venous system of the calf (*Mozes and Gloviczki, 2004*)

Giacomini vein

The Giacomini vein is a thigh extension of the SSV that extends up to the posterior thigh, travels medially and joins the posterior thigh circumflex vein before draining into the GSV. This is an intersaphenous vein. Intersaphenous veins (*vena intersaphena*) are tributaries to the saphenous veins that course obliquely in the leg or thigh to connect the SSV and GSV (*Natsis et al., 2015*) (**Figure 4**)

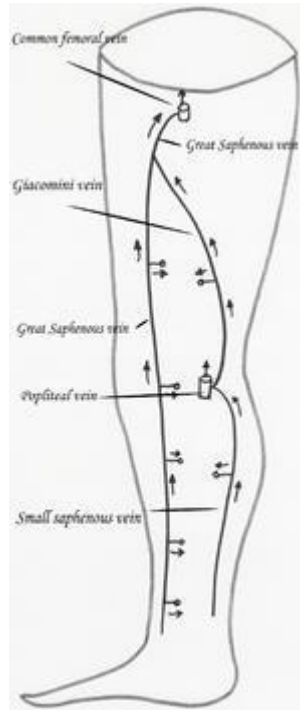


Figure (4): The **Giacomini vein** is a communicant vein between the great saphenous vein (GSV) and the small saphenous vein (SSV). It is named after the Italian anatomist Carlo Giacomini (1840–1898). The Giacomini vein courses the posterior thigh as either a trunk projection, or tributary of the SSV. In one study it was found in over two-thirds of limbs (*Khodabakhsh et al., 2004*).

Perforating veins

Small anatomic series in cadavers have reported an average of 64 perforating veins between the ankle and the groin. They may empty either into the axial deep veins (**direct perforators**) or into the venous sinuses of the calf (**indirect perforators**). Although numerous and variable, perforating veins can be grouped into four groups of clinical significance—those of **the foot**, **the medial and lateral calf**, and **the thigh**. The foot

perforators are unique in that they normally direct flow toward the superficial veins, while all others normally direct flow to the deep system. The major perforators of the medial calf and thigh have one to three valves that direct flow from the superficial to the deep veins (*Gianesini, 2018*) (**Figure 5**).

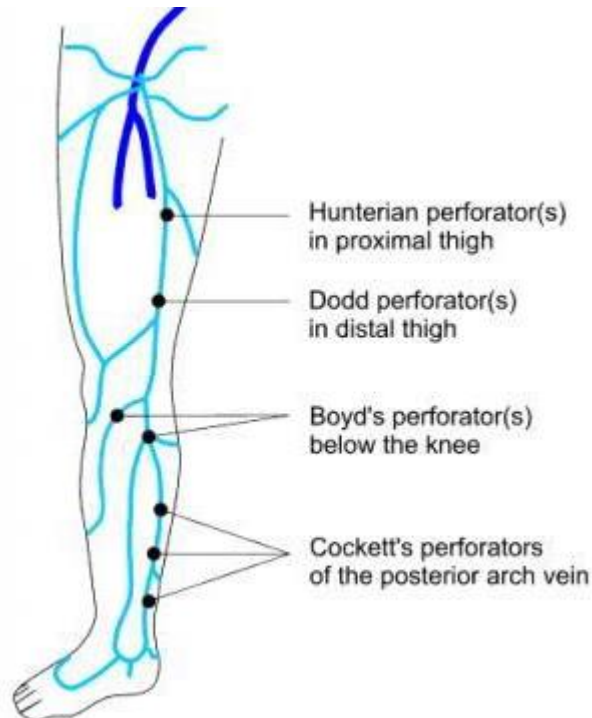


Figure (5): Hunterian perforator connects GSV to femoral vein in mid-thigh, **Dodd perforator** connects GSV to femoral vein in lower thigh, **Boyd's perforator** connects GSV or posterior arch vein of Leonardo to posterior tibial vein while three **Cockett's ankle perforators** (6 cm, 13 cm, 18 cm above medial malleolus) connects posterior arch vein to posterior tibial vein (*Mozes and Gloviczki, 2004*).

Anterior Accessory saphenous vein (Figure 2)

The **anterior accessory saphenous vein** is a special anterior tributary of the great saphenous vein (GSV), draining

the antero-lateral aspect of the thigh (**Franceschi and Zamboni, 2009**). It joins GSV near the saphenous-femoral junction at the saphenous arch or can drain directly in the femoral vein. It can drain below the saphenous arch or in a GSV tributary.

Sometimes it can drain in the external pudendal vein (which can communicate with an ovarian vein) and be the reason of a varicose disease of the thigh secondary to pelvic varicose disease (**Franceschi and Zamboni, 2009**). In contrast with other tributaries, its wall is histologically saphenous type with a thick media, running parallel and external to the GSV (**Franceschi and Zamboni, 2009**).

Venous Valves (Figure 6)

Bicuspid valves are important structures allowing unidirectional flow in the normal venous system. The GSV has at least 6 valves (range 6-24) with a constant valve present within 2 to 3 cm of the SFJ in 85% of cases and the SSV has a median of 7 to 10 valves (range 4-13). There are valves in the deep veins, but the common femoral and external iliac vein has only one valve in 63% of cases. The internal iliac vein has a valve in 10% of cases (**Vincent et al., 2011**).

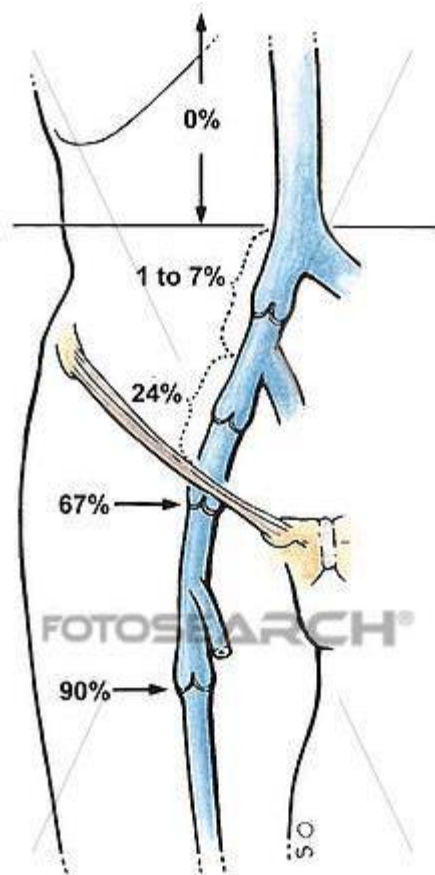


Figure (6): Valves in different venous segments (*Vincent et al., 2011*).

Chapter 2

ETIOLOGY & PATHOPHYSIOLOGY

Etiology and Pathophysiology of lower limb varicose veins

At rest, in the erect position, the pressure in the saphenous vein is primarily derived by that of a column of blood from the right atrium to the site of measurement of the leg (about 120 mm Hg at the ankle). With muscular activity, the pressure in the normal saphenous vein at the level of the malleoli falls 45-68 Hg below the resting level and is reduced from 80 to 40 mm Hg in the posterior tibial vein (**Arnoldi, 2005**) (**Figure 7**).

Due to the one-way valvular system in these veins and calf muscle pump, blood flow is thus directed from the superficial venous system to the deep venous system via communicating or perforating vessels (which also contain one-way valves). The venous blood then flows towards the heart (**Dodd and Cockett, 1976**).

The pathophysiology of varicose veins can be divided into four categories that may overlap with one another: increased deep venous pressure, primary valvular incompetence, secondary valvular incompetence, and fascial weakness (**Santler & Goerge, 2017**).

This may result from valvular incompetence of axial deep or superficial veins, perforator veins, venous tributaries, or venous obstruction, or a combination of these mechanisms. These factors are exacerbated by muscle pump dysfunction especially the calf muscles. These mechanisms produce venous hypertension, particularly with standing or ambulation (**Santler and Goerge, 2017**).

The superficial veins respond to increased pressure by dilating to accommodate the increased blood flow. Valvular incompetence occurs and varicosities appear (**Farber and Bates, 2011**).

In addition, with movement of the lower limbs, the high venous pressure that normally occurs within the calf is transmitted straight to the superficial veins and subcutaneous tissues drained by these communicating veins (**Negus and Friedgood, 2010**).

The venous pressure in the cuticular venules may reach 100 mm Hg in the erect position. This causes venular dilation over the whole area and results in capillary dilation, increased permeability and an increased blood volume in the subcutaneous capillary bed (**Burland et al., 2013**).

The perforating veins at the ankle are not surrounded by deep fascia or muscles. Therefore, the increased deep venous pressure is directly transmitted through the perforating vein to

its superficial connecting vein. This leads to high cutaneous pressures with a resulting transudation of extracellular fluid (*Burland et al., 2013*).

This has been shown to lead to perivascular fibrin deposition, which probably plays a significant role in decreased oxygenation of cutaneous and supporting tissues, thereby leading to cutaneous ulceration (*Lotti et al., 2007*).

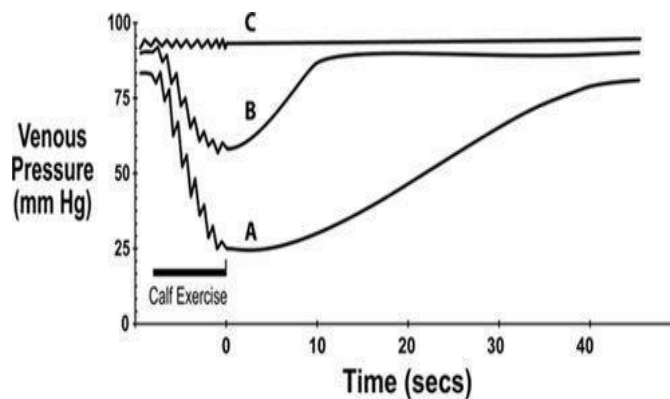


Figure (7): Ambulatory foot venous pressure measurements during exercise and at rest over time in the standing position. A, Normal venous pressure. The resting standing venous pressure is ≈ 80 to 90 mm Hg. The pressure drops to ≈ 20 to 30 mm Hg (or $>50\%$ decrease) with calf exercise. The return in pressure is gradual, with refill taking >20 seconds. B, Abnormal venous pressure with venous reflux. The resting standing pressure is usually higher than normal. The drop-in pressure with exercise is blunted ($<50\%$ decrease). The return in venous pressure to the resting level is rapid because of a short refill time (<20 seconds). C, Abnormal venous pressure with venous obstruction. Resting standing venous pressure is usually higher than normal. There is minimal-to-no drop-in pressure with exercise (*Padberg, 2001*).