

Contents

2.1	Physics Laws Governing Flow	19
2.2	Transmural Pressure and Venous Compliance	21
2.3	Anatomical and Physiological Pathways of Venous Drainage	22
2.4	Pathophysiological Venous Hemodynamics	24
2.4.1	Reflux Establishment and Definition	24
2.4.2	Reflux Pathogenesis in the Superficial Network: The Descending vs. Ascending Theories	25
2.4.3	Reflux Pathogenesis in the Deep Network	26
2.4.4	Shunts and Reflux Patterns	27
2.5	Hemodynamic Role of Perforators in Chronic Venous Disease	28
2.6	Reflux Hemodynamic Implications	28
2.7	Reflux Assessment	29
2.8	Hemodynamic Rationale of Reflux Suppression	30
	References	31

Abstract

Physiological Venous Hemodynamics Physics Laws Governing Flow

A review of the physical laws governing fluid motion is required to understand reflux pathophysiology. Venous blood flows not just because of a pressure gradient, as is commonly believed, but because of an energy gradient, in which pressure is only a single determinant. In accordance with the thermodynamics zero principle, there will be no energy exchange between systems presenting with the same energy values: no venous flow will occur. In accordance with the thermodynamics second principle, energy exchange will occur from a system presenting higher energy values to one at a lower energy state: venous flow will occur. Considering that reflux, like every physiological flow, needs an energy gradient to be generated, a simple but highly selective and reasoned therapeutic action against the escape, and in favor of the reentry, points will lead to a conservative but effective venous drainage restoration.

2.1 Physics Laws Governing Flow

In order to understand reflux pathophysiology deeply, a review of the physical laws governing fluid motion is required. The venous system presents several energy determinants making its physiology at least as intriguing as the arterial

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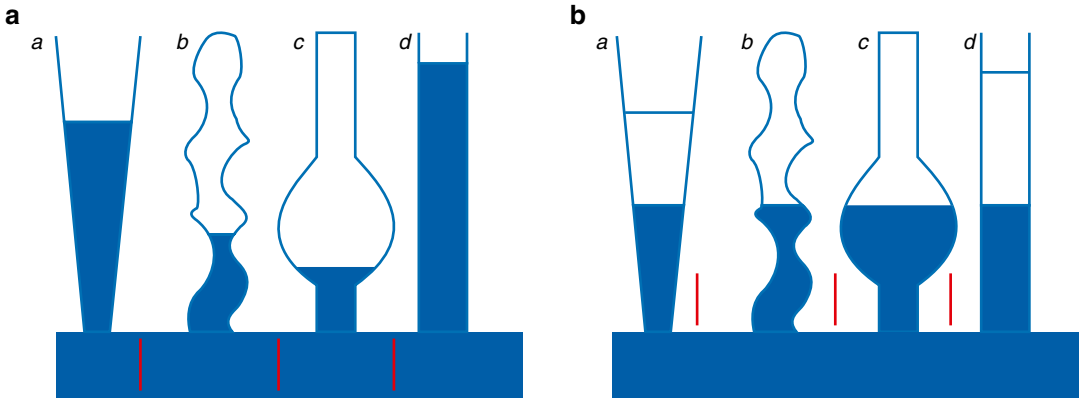


Fig. 2.1 The communicating vessel principle. (a) Noncommunicating hydrostatic columns presenting different heights, which lead to different energy states (column *d* presents the highest energy value).

(b) Communicating columns in which flow moves from the higher to the lower energy state systems, until a common energy balance is reached

one. Venous blood flows not just because of a pressure gradient, as is commonly believed, but because of an energy gradient, in which pressure is only a single determinant [1–4].

In accordance with the thermodynamics zero principle, there will be no energy exchange between systems presenting with the same energy values: no venous flow will occur. In accordance with the thermodynamics second principle, energy exchange will occur from a system presenting higher energy values to one at a lower energy state: venous flow will occur. The communicating vessel principle (Fig. 2.1) describes the result of these two phenomena: regardless of the vessel shape and volume, the fluid will flow from the system presenting a higher energy state to the one at lower energy value, until an energy balance is achieved [4].

If there is no communication between vessels (Fig. 2.1a), the columns present different energy states, which vary just according to the same column height. In fact, in this static situation, the only energy level determinant is the potential gravitational energy value, which is expressed by the following formula:

$$\text{Potential gravitational energy} = \rho gh$$

(ρ represents the fluid density, g the gravity constant, h the height above the surface)

Whenever the different systems are in communication (Fig. 2.1b), fluid flows from the higher to the lower column, thus settling into a

balanced common energy state, in which all the column heights are equal [5].

The venous system works both in stasis and in dynamics, so it is the Bernoulli's law which better describes the involved determinants. In ideal conditions, it states that the energy factors governing the venous hemodynamic are the kinetic energy ($\rho v^2/2$; ρ represents the fluid density, v the fluid velocity) together with the potential energy. The potential energy is constituted by the lateral pressure (p), linked to the vessel wall elastic properties, and gravitational pressure, produced by the blood column weight.

The sum of them ($\rho v^2/2 + p + \rho gh$) is constant at any point:

$$\text{Bernoulli's principle: } \rho v^2/2 + p + \rho gh = \text{constant}$$

This means that in the stasis condition, the potential energy will be at its maximum, while it will decrease proportionally to the flow velocity increase. The obvious but determinant consequence is that the lateral pressure, exerted on the venous wall, will decrease proportionally to the velocity reached by the fluid (Fig. 2.2).

According to Bernoulli's principle, in two communicating vessels, the one presenting a higher flow velocity will display a lower lateral pressure: a gradient will be created and the blood will flow from the slower to the faster vessel. The aspiration effect performed by the higher velocity vessel is universally known as the Venturi's principle. Venturi's principle is strictly linked to the

Castelli's law (Fig. 2.3) which states that flow velocity (v) is inversely proportional to the vessel cross-sectional area (A):

$$\text{Castelli's law: } A_1 v_1 = A_2 v_2 = \text{Flow } (Q)$$

The implication is that, whenever the vessel divides into several branches, if the sum of the areas of the branches is smaller than the original vessel, an increased flow velocity will be expected. The opposite will be realized if the total area of the branches increases.

2.2 Transmural Pressure and Venous Compliance

Transmural pressure (TMP) is a key factor in understanding venous hemodynamics. It is the difference between the internal venous pressure (IVP), acting on the internal vessel side to expand

it, and the external venous pressure (EVP), acting on the external parietal wall to collapse it (Fig. 2.4). TMP and vessel permeability represent the determinants of intravascular-extravascular exchanges (Starling's law).

Together with an energy gradient, another necessary element in producing a flow is the vessel capacity to receive a certain amount of fluid. As a collapsed, elliptical vein begins to fill, it can receive a large volume of fluid with little increase in pressure, a property conferring the blood reservoir function to the venous system. Much more pressure is required to stretch the vessel with additional fluid volume once it has become circular.

The physical property of a vessel to increase its volume with increasing TMP is known as compliance (C) and is expressed by the change in volume (ΔV) divided by the change in pressure (ΔP):

$$\text{Compliance} = \Delta V / \Delta P$$

Compliance is strictly linked not only to the filling degree but also to the geometric vessel properties (length and radius), together with its wall elasticity.

A pressure-diameter curve (Fig. 2.5) highlights the nonlinear relationship in the initial filling phase, which is due to the great increase in vessel caliber following tiny pressure augmentations. On the contrary, in a distension phase, starting from pressure values around 20 mmHg, a volume/pressure linearity has been demonstrated.

Up to this point, all of our physics law applications have been made considering the vessel and the blood as an ideal conduit and liquid,

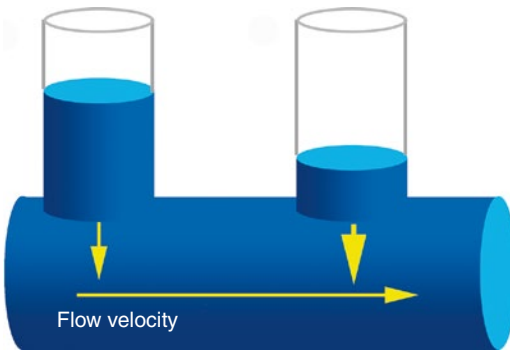


Fig. 2.2 Bernoulli's principle related lateral pressure (LP) drop. Decreasing lateral pressure values, according to flow velocity increase

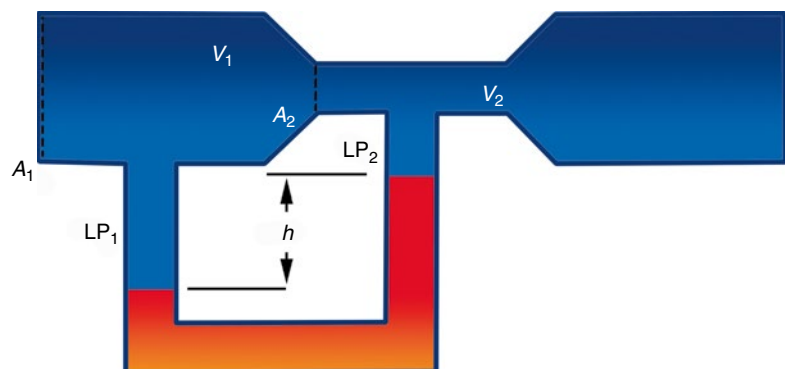


Fig. 2.3 Castelli's law and Venturi's effect. Flow velocity (v) inverse proportionality to vessel cross-sectional area (A) (Castelli's law) and consequent LP variation ($LP_1 > LP_2$) leading to the fluid aspiration determined by the Venturi's effect

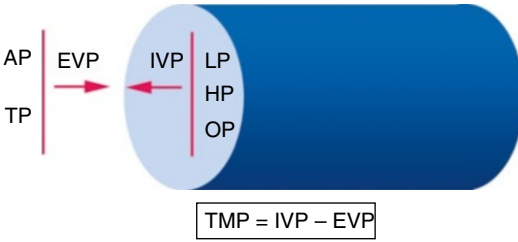


Fig. 2.4 Transmural pressure. *AP* atmospheric pressure, *TP* tissue pressure, *EVP* external venous pressure, *IVP* internal venous pressure, *LP* lateral pressure, *HP* hydrostatic pressure, *OP* oncotic pressure. TMP is the crucial parameter in tissue drainage and venous caliber regulation

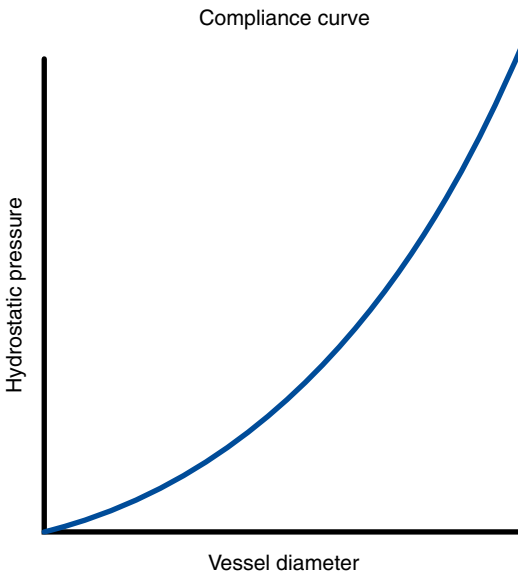


Fig. 2.5 Compliance curve. The pressure-diameter curve highlights an exponential pressure increase over a little volume amount in an initial filling phase. After the achievement of a certain distension phase, the volume–pressure ratio (compliance) shows a linear relationship: in the saphenous system, this happens around the pressure value of 20 mmHg

respectively. The human body, however, produces friction through blood contact. An extension of the thermodynamics second principle, the entropy law, states that in case of nonideal conduits or liquids, part of the energy is dissipated as heat generation, thus increasing the amount of unavailable energy (entropy). The human body solution to counteract this energy dissipation has

been the creation of the several pump mechanisms placed in series all along the cardiovascular system.

2.3 Anatomical and Physiological Pathways of Venous Drainage

The venous drainage occurs from the superficial to the deep tissues and from the distal areas to the heart. The only two exceptions are represented by the foot sole venous system, where the blood is directed toward the dorsal network through marginal veins and the saphenofemoral junction tributaries, some of which drain reversely from the abdomen toward the groin.

Three anatomical compartments are identifiable in the venous system:

Anatomical compartment 1 (AC1) is located underneath the deep fascia and contains the deep venous system (femoral, popliteal, tibial, peroneal, gastrocnemius, and soleal veins).

Anatomical compartment 2 (AC2) is located between the superficial and the deep fascia and contains the saphenous system (great, accessory, small saphenous, and intersaphenous veins).

Anatomical compartment 3 (AC3) is located above the superficial fascia and contains the tributary veins.

One of the most important vein features is their being endowed with bicuspid unidirectional valves. These are thin but extremely strong structures lying at the base of a vein segment which is expanded into a sinus. This anatomical peculiarity allows the valve to open without completely touching the parietal wall, thus resulting in a fast closure with blood flow reversal [6].

Valve density is significant in determining drainage pathways. The density of AC2 valves is lower at the leg than at the thigh (Fig. 2.6), and it always remains lower than that belonging to the AC1. This anatomical arrangement puts into practice the communicating vessel principle

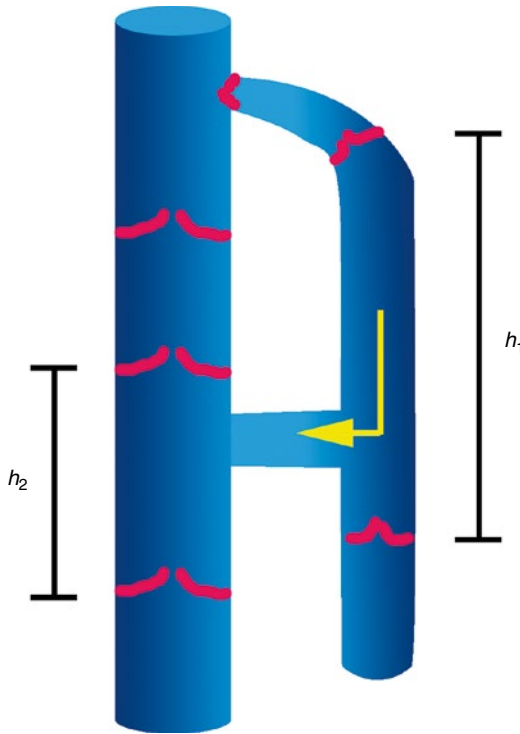


Fig. 2.6 Valve density in the deep and saphenous compartments. The valvular density of the saphenous compartment is lower than that of the deep venous system. This anatomical organization creates higher hydrostatic columns in the saphenous system. Following the communicating vessel principle, blood will flow from the superficial to the deep compartments, through the perforating veins

(Fig. 2.1), thus representing the first determinant in the hierarchical emptying order from the superficial to the deep venous system [7].

The different anatomical compartment locations confer the second determinant of the physiological lower limb drainage. In fact, the muscle pump, which is mainly developed in the calf, assumes the main antagonist role against the force of gravity. The soleal and gastrocnemial contractions exert an EVP between 40 and 200 mmHg, thus reducing the TMP and displacing the blood toward the heart. The generated pressure wave will be transmitted to the surrounding veins proportionally to their own proximity to the muscular fascia investments.

In AC1, all the veins are in direct contact with the muscle mass and are surrounded by the rigid counterforce provided by the deep fasciae. At this level, the calf muscle pump is able to exert its maximum activity in opposing the force of gravity. The saphenous system, being above the muscular compartment and banded between the superficial and the deep fasciae, receives from the calf systole a higher energetic amount than that of its tributaries but also a significantly smaller one than that received by AC1. The decreasing muscular pump effect from AC1 to AC3 is shown by the decrease of concomitant flow velocities: 20–40 cm/s in AC1, 10–20 cm/s in AC2, and 0.05 cm/s in capillary plexuses (Fig. 2.7). The above-described deceleration creates a Venturi's effect. This in turn governs the so-called physiological venous hierarchical order of emptying from the superficial to the deep compartments (from AC3 to AC2 to AC1) of the leg (Fig. 2.8).

Two others “pumps” have to be considered among venous flow determinants: the cardiac and thoracoabdominal. The heart is the main blood propeller providing volume, pressure, and flow to the system in the supine position when the hydrostatic pressure is null (in the standing position, the cardiac-created energy gradient needs to be integrated with the muscular pump because of the hydrostatic pressure increase).

Moreover, the right heart greatly influences venous hemodynamics, increasing the central venous pressure during its systole and the venous flow during its diastole. The close link between heart pump and venous circulation is shown by the evident cardiac pulsations in lower limb venous tracings and by the venous edema seen in congestive heart failure patients.

The thoracoabdominal pump influences the venous return by means of the diaphragm movements, which in the end determines the intra-abdominal pressure. During inspiration, intra-abdominal pressure increases, thus compressing the inferior vena cava and reducing the venous blood flow; usually the venous outflow from the lower limbs can temporarily cease. During expiration, the intra-abdominal pressure falls again, the inferior vena cava expands, and the

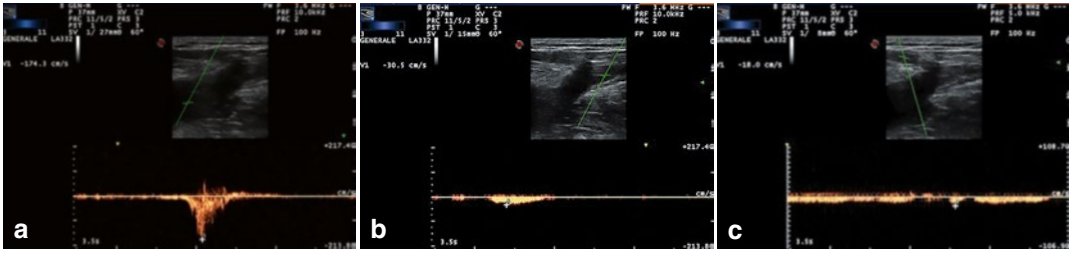


Fig. 2.7 Decreasing flow velocities from the deep to the superficial venous compartment. The phenomenon is mainly due to a muscular pump minor energy transfer to the superficial veins. The Doppler sample was placed in

the common femoral vein just above the saphenous confluence (a), in the great saphenous vein underneath the preterminal valve (b), in the superficial epigastric vein just before its saphenous confluence (c)

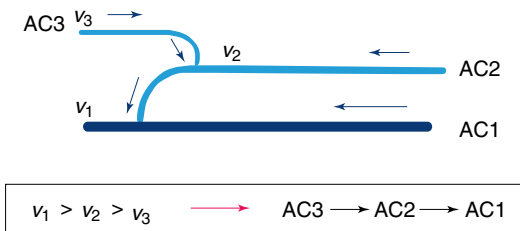


Fig. 2.8 The hierarchy of venous compartment emptying. The physiological venous emptying from the superficial to the deep tissues is made possible by the blood aspiration exerted by the Venturi's effect application

venous blood from the lower limbs can flow to the heart. In conclusion, in order to establish a flow, two factors are needed: an energy gradient (potential plus kinetic) and a system with a compliance capable of receiving a certain fluid amount [4].

2.4 Pathophysiological Venous Hemodynamics

2.4.1 Reflux Establishment and Definition

A retrograde segmental superficial and/or deep venous flow lasting less than 0.5 s (except in the femoropopliteal system, where 1.0 s is allowed) is considered physiological in the muscular diastolic phase. The communicating vessel principle predicts blood displacement from the higher hydrostatic columns to the lower ones, until an energy balance is reached.

The distance between two competent valvular planes is not long, even if greater in AC2 than in AC1. Thus, a physiological retrograde flow exhibits slow velocities, which render this blood movement undetectable by Doppler (Fig. 2.9a). On the other hand, in the case of valvular incontinence or absence, the height of the hydrostatic columns becomes progressively higher (Fig. 2.9b): if the system presents a compliance capable of receiving a blood overload, a diastolic flow at high velocity will be produced and revealed by Doppler.

A reflux is defined as a flow that is inverted with respect to the physiological direction and that lasts more than 0.5 s (except 1.0 s for the femoropopliteal system). Thus, it is a flow that displaces blood toward the distal areas of the lower limbs and from the deeper to the more superficial compartments; in this sense, reflux can be defined as a change in the hierarchical order of emptying (from AC1 to AC3).

Therefore, two main scenarios are possible in reflux characterization: it is a retrograde flow running down in the same conduit, or it is an inverted flow jumping into an anatomical compartment more superficially placed. The first situation is common in healthy but long-standing subjects, thus not strictly pathological. The second scenario is certainly a pathological one because of the loss in the hierarchical order of venous emptying. Unfortunately, the actual definition does not differentiate between the two different hemodynamic situations, thus offering an issue for a future-related consensus.

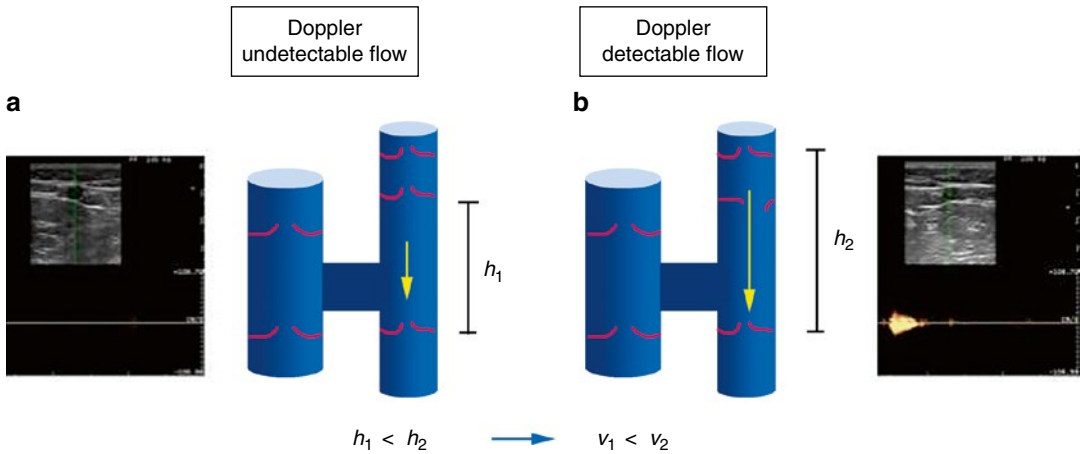


Fig. 2.9 Valvular derangement consequence on flow velocity. (a) In physiological conditions, the distance between two competent valves does not allow a significant retrograde flow velocity enhancement. No Doppler

signal will be revealed. (b) In case of valvular derangement, the distance between two competent valves becomes significant. The consequent refluxing flow velocity increase will permit a Doppler signal transmission

Reflux is a flow, so to be established, it needs an energy gradient and a system compliance capable to receive it. Moreover, a reflux needs a connection between the venous segment acting as the flow source (the escape point) and the vessel destined to receive the blood overload (the reentry point).

The escape and reentry points are not to be considered just for their anatomical relevance. In fact, their first meaning is their hemodynamic significance. The energy gradient between the escape and the reentry points is the *conditio sine qua non* for the reflux creation. If the venous network receiving the refluxing blood (the reentry point) was not in a lower energy state as a consequence of its smaller hydrostatic columns created by competent valves, no flow motion would be developed. Every time a reflux is detected, a reentry point must be expected.

The pathological reflux compartment jump has two main causes: an increase in the deep pressure (following a Valsalva maneuver or a thrombotic occlusion) or a superficial pressure decrease. In the last case, the energy drop is linked to the aspiration caused by the reentry gradient, which in turn is responsible for an acceleration of the refluxing blood, thus creating a Venturi's effect which reduces the lateral pres-

sure, thus aspirating on the reflux source itself in a closed circuit (Fig. 2.10).

The vicious cycle of the previously described circuit is defined as a “private circulation.” This is a pathological blood recirculation which is established between two linked venous networks in which a certain amount of venous blood refluxes into the reentry point during diastole and then, during systole, flows back to the escape point, thus supplying the same shunt once again [8–10].

2.4.2 Reflux Pathogenesis in the Superficial Network: The Descending vs. Ascending Theories

The pathogenesis of superficial venous reflux is controversial [11, 12]. Descending and ascending theories are promoted. The descending or valvular hypothesis was first described by Trendelenburg in the nineteenth century. He proposed that reflux begins because of an incompetent terminal saphenofemoral valve, which is overwhelmed by the hydrostatic column pressing on it. Reflux then progresses in a retrograde direction, progressively causing incompetence of more distal valves.

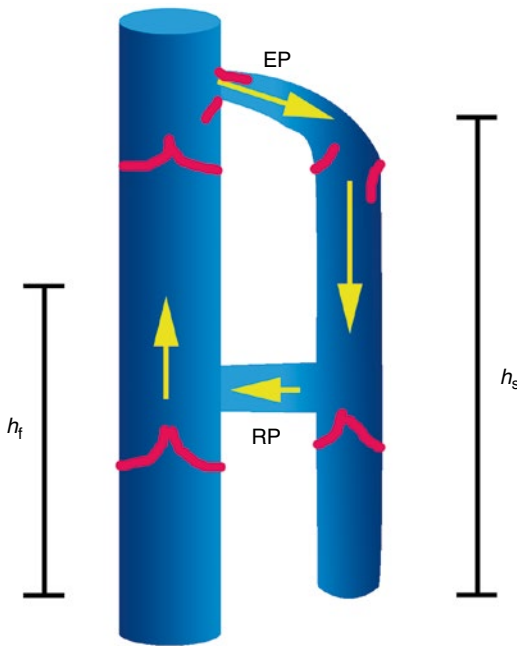


Fig. 2.10 Venturi's effect and communicating vessel principle application on the anatomical venous compartments jump. In this figure, the saphenofemoral junction represents the reflux source (escape point [EP]). The blood reversal takes place because of the aspiration effect exerted by the higher femoral flow velocity (v_f) through the reentry perforator (RP). The incompetent proximal saphenous vein valves allow a blood recirculation into the saphenous axis itself. Together with the communicating vessel principle, this causes a progressive increase in the reversed flow velocity (v_s). This leads to a reduction in the saphenofemoral confluence lateral pressure, which in turn promotes the establishment of a refluxing closed circuit (femoral hydrostatic column height [h_f], saphenous hydrostatic column height [h_s])

The ascending theory was proposed in the 1980s based on histological, biochemical, and functional investigations demonstrating how the venous wall can undergo pathological alterations in segmental localizations, irrespective of the site and functional state of the valves. In this pathophysiological explanation, reflux begins as a local alteration, possibly developing in any part of the lower limb, and valve failure progresses in an antegrade fashion [13].

Even if the location of reflux genesis is controversial, recent researchers have proposed a

unifying pathogenetic theory. Primary structural changes of the venous valve or wall lead to an initial reflux. Increased metalloproteinases (MMPs) activity, following high wall tension values, has been recently demonstrated. The consequent derangement of the endothelium and smooth muscle cells causes altered venous constriction/relaxation properties, together with the leukocyte chemotaxis. A vicious circle involving valvular incompetence, venous wall alteration, vessel dilation, and increasing reflux is created. Thus, parietal damage leading to vessel dilation seems to be antecedent to the valvular incompetence instauration; a cascade of events is then executed by the activation of MMPs, with consequent progressive venous drainage impairment. The unbalanced proteolytic activity in varicose vein tissues is nowadays fully documented in the literature: MMP 1, MMP 2, MMP 9, and MMP 13 upregulation has been observed in stasis dermatitis, ratios of tissue inhibitors of MMPs to activated MMPs have been found to be higher in varicose patients, and unregulated levels of inflammation-related TGF-beta have been assessed into the dilated vein wall [1]. All these molecular events become the final executor of the pathological cascade which, in the end, leads to the macroscopic varicose derangement.

2.4.3 Reflux Pathogenesis in the Deep Network

Agensis, malformations, and post-thrombotic damages are the most frequent causes of deep valvular incompetence linked to deep venous reflux. In past years, deep venous incompetence was considered to cause significant hemodynamic derangements. Several recent investigations have pointed out how different deep segments carry with them different hemodynamic impacts. Iliac axis incompetence is considered hemodynamically less relevant than the deep lower leg veins, whose alteration can lead to even irreversible muscle pump damage. The femoral vein is still a matter of debate. Some data suggested it is irrelevant, while other data highlight severe disturbances following its incompetence. Selective saphenous vein ablation in the case of combined saphenous

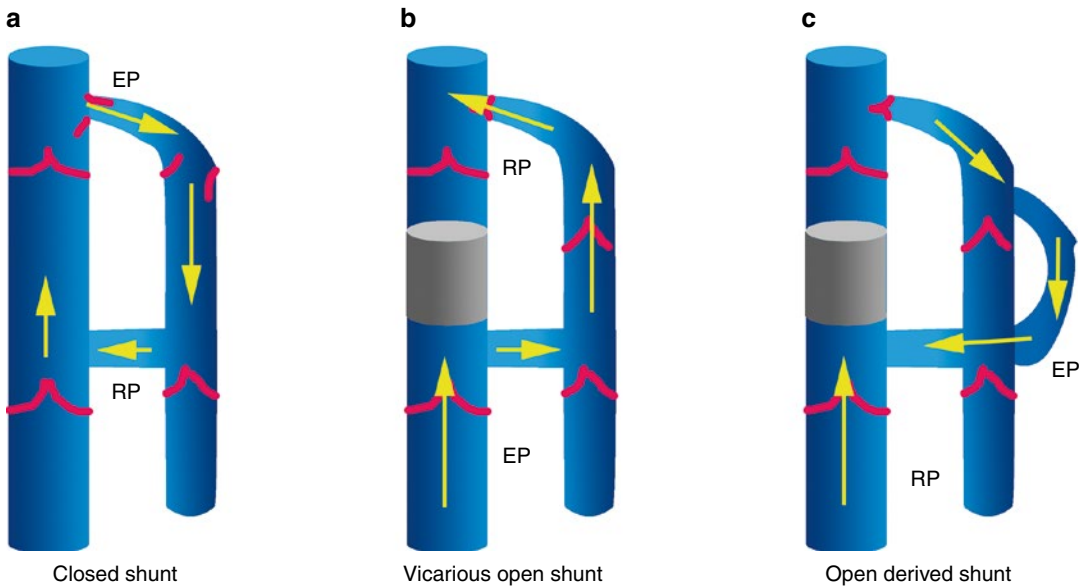


Fig. 2.11 (a) Closed shunt. Recirculation from an escape point (EP) through a reentry perforator (RP) during the diastole following the muscular pump activation. A private circulation is established and excluded by the remaining draining network. (b) Vicarious open shunt. Collateral circulations are activated in order to bypass an obstacle (e.g., thrombosis, vicarious varicose veins

following non-hemodynamic therapeutic approaches). (c) Open derived shunt. Diastolic retrograde flow overload from a competent confluent vein because of an incompetent collateral link to a reentry perforator which directly drains into a deeper compartment. No recirculation is established because the EP and the RP belong to different networks

and femoral vein refluxes usually restores normal venous hemodynamics despite persisting femoral vein insufficiency. Femoral vein incompetence seems not to cause severe hemodynamic derangements if the lower legs are competent [14, 15].

2.4.4 Shunts and Reflux Patterns

A venous shunt is a pathway carrying two different types of flow: the physiologically draining one and the pathologically deviated blood. Anatomically and hemodynamically, it starts in a refluxing (or escaping) point and terminates in the so-called reentry point. Three main shunt networks are described: closed, vicarious open, and open derived.

In *closed shunts* (Fig. 2.11a), a vicious circle is created between the escape and the reentry points. The deviated flow recirculates at each energy gradient inversion like in a closed electrical circuit. A classic example is an incompetent saphenofemoral junction (escape point) letting

the femoral blood drain along the saphenous trunk in a retrograde fashion toward a reentry perforator during muscular pump diastole. At the systolic energy gradient inversion, the femoral blood will flow back to the saphenofemoral junction and then will be deviated again along the saphenous compartment. In this way, a closed shunt will be established, and a certain amount of blood will be excluded by the systemic venous network because it is entrapped in the previously described private circulation [4].

A *bypassing open shunt* (Fig. 2.11b) is a natural bypass exploited by the venous network to go over an obstacle. The use of a collateral route to bypass what is usually a thrombotic occlusion is desirable, as it reduces the drainage resistance. In this type of shunt, there is no recirculation, and it is fed by the residual draining pressure together with the proximal cardiac and thoracoabdominal aspiration. It may be either antegrade or retrograde.

An *open derived shunt* (Fig. 2.11c) is a flow diversion into an incompetent vein caused by a

reversed energy gradient usually generated during muscular pump diastole. The blood overload is directed to a reentry perforator which drains directly into a network not linked to the escape point one: no recirculation occurs. A typical example is an incompetent saphenous tributary endowed with a reentry perforator draining into the deep venous system: the blood overload will “jump” from AC2 to the saphenous tributary AC3 and then will flow down directly toward AC1 [4].

2.5 Hemodynamic Role of Perforators in Chronic Venous Disease

Perforating veins connect the superficial to the deep compartments by piercing the muscular fascia. A physiological draining direction from surface inward is guaranteed by unidirectional valves. In the past, perforator incompetence was considered a main initial reflux trigger. Several ligation or disruption methods, including subcutaneous endoscopic surgery, have been in use for many years. Nowadays, the evidence suggests that nonselective ligation or ablation of calf perforators is ineffective. When they are dilated, these veins should be analyzed and treated, taking into consideration their hemodynamic significance (escape or reentry points). Large perforating veins are not always directly responsible for trophic disorders, and, even if dynamic tests highlight a systolic outflow, the net flow direction is usually toward the deep venous system. On the other hand, a perforating vein is considered pathological whenever refluxing during the diastolic phase of a dynamic test.

Thus, a perforating vein may be treated depending on its hemodynamic role and on the shunt type it constitutes. For example, treatment of a reentry perforator belonging to a closed (Fig. 2.12a) or open derived shunt would be erroneous because that impairs the physiological blood return to the heart (Fig. 2.12b). On the contrary, elimination of a refluxing perforator feeding the closed or open derived shunt (escape point) is mandatory to perform a hemodynamic correction of the venous return (Fig. 2.12c).

Bidirectional flow can take place into the perforating system afflicted by primary chronic venous disease (CVD). Moreover, most of the time, these dilated veins are not the cause of venous hypertension but the consequence of voluminous saphenous system refluxes, flowing into the deep venous network through the perforating system [16].

2.6 Reflux Hemodynamic Implications

The first consequence of venous hypertension, linked to the refluxing blood overload, is an increase in the IVP and thus a rise in TMP values. As previously described, TMP is a key element in balancing the liquid compartments and so in ruling the tissue drainage. An excessive TMP results in an accumulation of toxic metabolites, which in turn becomes responsible for pathognomonic CVD skin changes.

The high TMP assessed in CVD patients leads to extravasation of red blood cells through the capillary sinusoids. Hemoglobin degradation in the interstitial compartment becomes the source of the iron-mediated oxidative reactions that are the final executor of this pathological tissue damage. Clinically, even with genetic and molecular individual peculiarities, the previously described hemodynamic phenomenon causes the typical edema, hyperpigmentation, lipodermatosclerosis, necrosis, and ulcer.

Another main reflux consequence is represented by the changes in flow physical characteristics. Physiologically, blood flow is laminar. It is comparable to several concentric cylinders sliding one over the other at decreasing velocities from the vessel central axis to the parietal layers. The cylinder adjacent to the vessel wall is the one most greatly involved by the parietal friction, thus the one presenting the lowest velocity.

The kinematics of fluid motion is significantly affected by blood viscosity, wall friction, geometric conduit characteristics, and flow velocities. All these factors are summarized in a dimensional parameter known as Reynolds number, which, if higher than 2,000, is predictive of

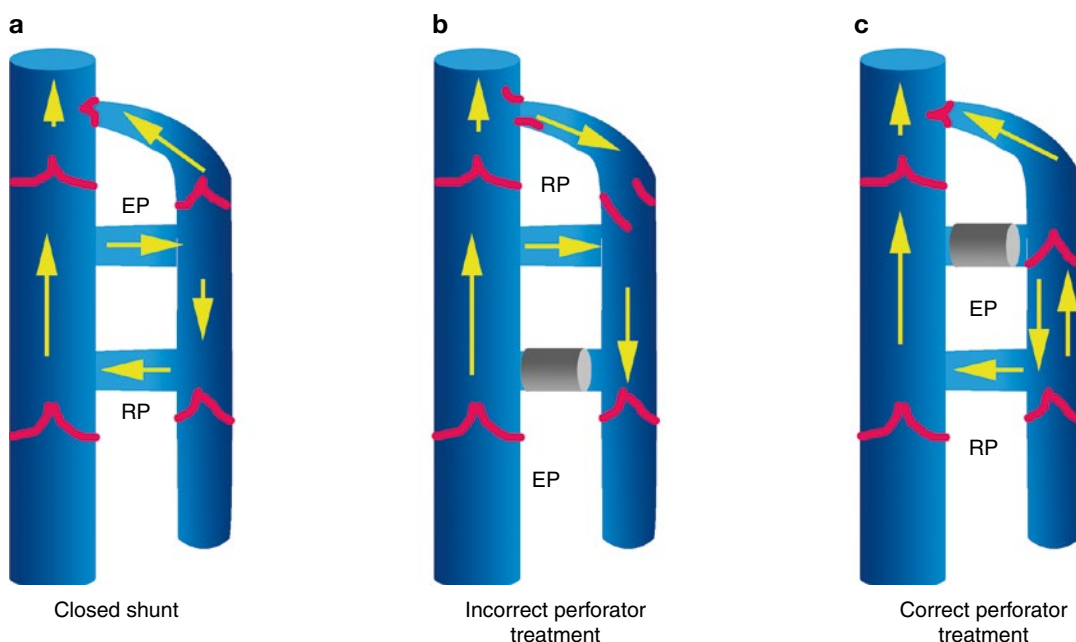


Fig. 2.12 (a) Closed shunt. Recirculation from an EP represented by a Hunterian perforator through a more distal RP. (b) Incorrect perforator treatment. Suppressing the RP energy gradient leads to a lateral pressure increase in the more superficial network which will suffer the consequent dilation progressively undergoing valves incompetence.

This phenomenon will cause an increased hydrostatic overload, which will worsen the hemodynamic impairment. (c) Correct perforator treatment. Suppressing the EP will lead to an abolition of the shunted blood overload. The lightened superficial network will possibly decrease its diameter, assuring valvular competence and physiological drainage

turbulent flow. Turbulent refluxing flow is a flow presenting a derangement of the kinetic energy vectors, which are no longer linear but chaotically orientated in all the directions. Thus, an energy waste is produced together with a tendency toward vascular wall dilation.

Dilation is more related to the energy gradient alteration than to just a pressure increase. The importance of turbulence and not just pressure is suggested by the use of the saphenous vein for arterial bypasses. Despite the increased pressure, the graft does not develop varicosities because of the arterial laminar flow (Fig. 2.13).

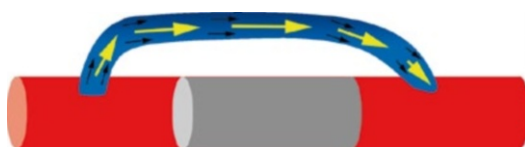


Fig. 2.13 Turbulence and vessel dilation. A vein graft in an arterial bypass does not develop varicosities despite the high-pressure values. This phenomenon is explained by the laminar flow draining into the vein and offers evidence of the importance of turbulence in the pathophysiology of varicose veins

2.7 Reflux Assessment

Understanding the pathophysiological background of the various reflux assessment techniques is mandatory for a correct interpretation of the performed investigations. A deeper description of the different diagnostic tools will

follow in Sect. 2.2. Nowadays, invasive ascending and descending venography has been largely replaced by ultrasound scans [17]. The noninvasive Doppler evaluation is proved to be comparable to venography in providing deep and superficial venous reflux staging. Descending contrast venography remains important in cases of severe deep venous occlusive disease leading to venous bypasses, valve repair, or transplantation procedures.

The ideal reflux assessment tool should provide both anatomical and hemodynamic data in a dynamic evaluation. Venography, photoplethysmography, and air plethysmography assessments, although particularly useful in providing standardized data on global venous hemodynamics, are unable to detect those isolated segmental refluxes and hemodynamic changes that are related to the muscular pump and gravitational energy gradient activation.

Ultrasonography, even if operator dependent, not only allows precise reflux anatomic localization and hemodynamic quantification but also evaluates the venous system under conditions that simulate the normal venous system pump function (Valsalva, compression/relaxation, Paranà, oscillation, toe-flexion, active foot dorsiflexion, rising-on-tiptoes maneuvers). Moreover, echo and color Doppler scanning represents the ideal tool to quantify reflux characterizing parameters, such as reflux time (RT), Psathakis (PI), and dynamic reflux indexes (DRI).

The exact cutoff time for reflux definition remains an actual topic of debate. Recent consensus opinion defines reflux as abnormal

flow lasting over 0.5 s for all veins except the femoropopliteal system, where the value is 1.0 s [18]. Independently by its duration, RT is a parameter not necessarily proportional to the reflux severity. The smaller the leaking valvular hole is, the longer lasting RT will be. Moreover, RT has been proved not to be proportional to the clinical severity class. It also changes according with the type of assessment maneuver utilized.

Psathakis (PI) and dynamic reflux indexes (DRI) also offer a potential means to quantify the hemodynamic impairment. PI is a parameter assessed in the deep network during compression/relaxation maneuver and is expressed by the following formula:

$$PI = \frac{(\text{diastolic reflux velocity surface})}{(\text{systolic velocity flow surface})}$$

This parameter is considered pathological when greater than 0.40, but it takes into account the reflux volume without assessing the flow. The DRI expresses both the reflux volume and the flow rate, through the following formula:

$$DRI = \frac{\left[(\text{diastolic reflux mean velocity})^2 \times (\text{diastolic reflux time}) \right]}{\left[(\text{systolic reflux mean velocity})^2 \times (\text{diastolic flow time}) \right]}$$

DRI varies according to the reflux flow rate, and it expresses the reflux severity degree more completely than PI or RT.

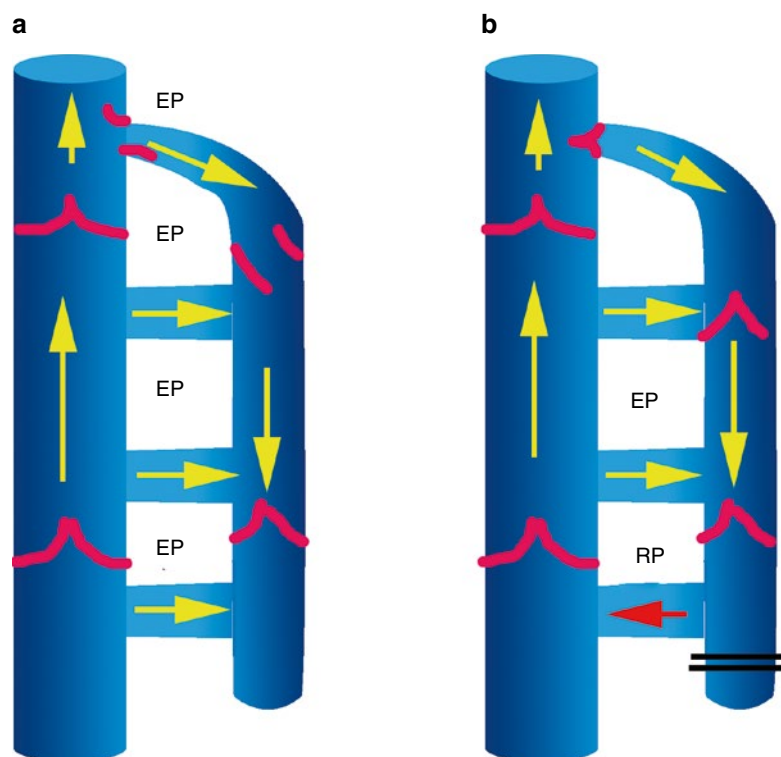
2.8 Hemodynamic Rationale of Reflux Suppression

As previously described, venous reflux leads to a venous hypertension related to an excessive TMP. The therapeutic aim becomes either a reduction of the IVP or an increase in EVP. When IVP is moderately high, the EVP increase caused

by elastic stocking compression can counteract the hypertension caused by the reflux. The elastic compression stocking assists the muscular fasciae and induces blood acceleration. Compression is responsible for a lateral pressure drop (Bernoulli's principle), thus favoring the hierarchical order of venous drainage by means of a blood aspiration toward the deepest networks (Venturi's effect).

Following valvular incompetence and venous wall degeneration, the hydrostatic columns can significantly increase their values. In this case, the therapeutic strategy needs to be addressed by

Fig. 2.14 Perforating vein terminalization. A simple ligation, or even a finger compression, below a perforating vein originally representing an EP of reflux can break off the private circulation. The consequent reflux disappearance is caused by the reduced diastolic reflux velocity, together with the private circulation compliance reduction. The result is an energy gradient inversion, which favors blood drainage toward the deep compartment. A perforator would then be transformed from an EP (a) to an RP (b)



fragmentation of such valve distances. Surgical correction can be offered by means of valve repair or permanent hydrostatic pressure fractionation through perforating veins “terminalization” [19].

Hydrostatic pressure fractionation is a conservative hemodynamic approach consisting in a ligation below a perforator belonging to a private circulation. The consequent reduction in the hydrostatic column leads to a decrease of the reflux velocity and system compliance, which in turn is associated to an increased lateral pressure. The consequence is an inversion of the refluxing gradient in favor of its aspiration into the deep circulation (Fig. 2.14).

Applying the physics laws, the strategy to conservatively achieve a reflux abolishment becomes understandable. Considering that reflux, like every physiological flow, needs an energy gradient to be generated, a simple but highly selective and reasoned therapeutic action against the escape, and in favor of the reentry points, will lead to a conservative but effective venous drainage restoration [20].

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