



Insights into the Venous Hemodynamics of the Lower Extremity

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Abstract

Purpose: This review study pays attention to motive forces that influence the return of venous blood and venous hemodynamics in the lower extremity, deals with the occurrence and hemodynamic consequences of saphenous reflux, assesses the pathogenic significance of calf perforators and femoral and popliteal vein incompetence, and explains the term hemodynamic paradox.

Method: Information gained from PubMed and based on 28 articles published in the literature was used to compile the article.

Results: Calf pump activity is the most important motive force propelling venous blood from the lower extremity toward the heart against the gravitation force. It produces decrease in venous pressure below the knee, creates ambulatory pressure gradient and thereby preconditions for the occurrence of saphenous reflux. Saphenous reflux causes ambulatory venous hypertension. Calf perforators do not induce hemodynamic disturbance; the outward flow within them is no reflux but a physiological, toward the heart oriented flow. Similarly, femoral and popliteal vein incompetence per se does not cause hemodynamic disorders. The tenacious tendency of varicose veins to recurrence is caused by a curious hemodynamic phenomenon called hemodynamic paradox.

Conclusions: Saphenous reflux in primary varicose veins is responsible for the development of the hemodynamic disorders and chronic venous insufficiency, whereas incompetent calf perforators and incompetence of the femoral and popliteal vein are hemodynamically irrelevant. A curious phenomenon called hemodynamic paradox is apparently responsible for the tenacious tendency of varicose veins to recur.

Keywords: Venous reflux; Incompetence of the femoral/popliteal vein; Hemodynamic paradox

Introduction

Venous hemodynamics in the lower extremities and the pathophysiology of the venous return to the heart are a contentious issue in phlebology. This concerns especially the significance of saphenous reflux, the role of calf perforators, and the implication of femoral and popliteal vein incompetence. Based on the results of venous pressure and flow measurements as well as on plethysmographic findings published in the literature, this article discusses the contentious topics, attempts to delineate the factual hemodynamic conditions in the lower extremities, and mentions a hemodynamic phenomenon that might be responsible for the tenacious tendency of varicose veins to recur.

Motive Forces of the Venous Hemodynamics

At rest, the venous return to the heart is driven by *vis a tergo*, which is the resting energy produced by the heart and transmitted through the capillary bed into the venous system; its value — the pressure difference between the venules and the right atrium — is about 15 mmHg. In addition to this cardiac energy, calf muscle pump produces a very effective supplementary motive force that substantially enhances the venous return.

In the quiet standing position, the pressure in the veins of the lower extremity is determined by the gravitation; it increases by 0.8 mmHg per centimeter beneath the right atrium and, depending on the body height, reaches the value of 80 mmHg to 100 mmHg above the ankle. This condition has been called hydrostatic venous hypertension, and comes about both in varicose vein patients and in healthy people. In varicose vein disease, the effect of the hydrostatic pressure is further potentiated by increased intra-abdominal pressure propagating into the veins of the lower extremity. The

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combined effect of the hydrostatic and increased intra-abdominal pressure causes distention of the veins, induces incompetence of venous valves, and enables the occurrence of venous reflux. Moreover, vein walls in varicose vein patients are impaired by inherent defective structure and predisposed to increased distensibility and decreased contractility.

Calf pump activity influences considerably the venous hemodynamics. Calf muscle contraction increases the venous pressure by about 80 mmHg in deep lower leg veins [1], which propels the venous blood predominantly into the popliteal vein, but a part of the venous blood is expressed via calf perforators into the saphenous system and continues in the centripetal direction into the femoral vein and furthermore toward the heart. Thus, there is a double-barreled outflow tract for the venous return in the lower extremity.

During calf muscle relaxation and/or continued calf pump activity the high hydrostatic pressure in the veins below the knee decreases significantly to about 25 mmHg; this phenomenon has been called physiological decrease in ambulatory venous pressure. In contrast to that, the venous pressure in the popliteal and femoral veins does not decrease; it fluctuates during calf pump activity but keeps principally the level of high hydrostatic pressure. In this way, calf pump activity produces a pressure difference with higher pressure in thigh veins and lower pressure in lower leg veins; it has been called ambulatory pressure gradient, achieves the value of $37.4 \text{ mmHg} \pm 6.4 \text{ mmHg}$, and is the essential factor triggering venous reflux in incompetent veins [2].

The Hemodynamic Impact of Venous Reflux

Venous reflux is a very important pathogenic factor. It comes into effect during calf pump activity, streams in incompetent veins of the lower extremity in the centrifugal direction (away from the heart), interferes with the physiological decrease in pressure in the veins below the knee and causes here ambulatory venous hypertension. It is necessary to differentiate between the hydrostatic venous hypertension, which is induced by the gravitation force, and ambulatory venous hypertension, which is evoked by venous reflux during calf pump activity. Saphenous reflux in primary varicose veins, i.e., centrifugal streaming in the incompetent great and/or small saphenous vein is responsible for the occurrence of the hemodynamic disorders. The blood volume ejected from deep lower leg veins during calf muscle contraction is replaced more or less during calf muscle relaxation (diastole) by the blood refluxing through the incompetent saphenous system, which counteracts the physiological decrease in venous pressure in the veins below the knee and induces, depending on reflux intensity, various degrees of ambulatory venous hypertension. Plethysmographic methods are able to quantify the hemodynamic disorders and the reflux intensity by means of several parameters. Venous filling index indicates reflux intensity in ml/s; it is gained using air plethysmography. If the ejected and refluxing blood volumes equal, no decrease of pressure occurs during calf pump activity and the severest grade of ambulatory venous hypertension arises.

Saphenous reflux is characterized by drainage of blood from superficial thigh veins into deep lower leg veins; this pathological phenomenon is distinctive of primary varicose veins; no such drainage occurs in healthy people.

The Pathogenic Significance of Calf Perforators

The pathogenic significance of calf perforators continues to be

a contentious issue. According to the generally accepted opinion established since the publications by Linton [3] and Cockett and Jones [4], calf perforators in healthy people are allegedly fitted with competent valves enabling exclusively unilateral flow from superficial into deep lower leg veins. It has been asserted that if these valves become incompetent, they enable outward flow from deep lower leg veins into the saphenous system; this outward flow has been denoted reflux and considered to be the main factor causing ambulatory venous hypertension. This theory continues to be still acknowledged, although it has never been confirmed by conclusive proofs. On the contrary, it has been proven that this conception is at odds with the reality. First, the outward flow within calf perforators is no reflux; it is the exact opposite of reflux. Whereas venous reflux is a centrifugal, diastolic flow streaming inward within calf perforators and causing ambulatory venous hypertension, the outward flow within calf perforators is a centripetal, systolic flow streaming in the physiological direction toward the heart and causing no hemodynamic disturbance. Bjordal [5] measured venous pressure directly in incompetent calf perforators during calf pump activity; he documented that the high hydrostatic pressure decreased and reached the value of the low physiological ambulatory pressure, once the saphenous reflux was interrupted. Thus, no ambulatory venous hypertension occurred in incompetent calf perforators but a physiological decrease in venous pressure took place here during calf pump activity. Second, there are many calf perforators; as a whole, they do not behave as a competent system, neither in healthy people. That was documented by simultaneous venous pressure recordings in the posterior tibial vein and the great saphenous vein in healthy people [6]. The pressure curves were nearly identical showing the same increase in pressure in both veins during calf muscle contractions and decrease during calf muscle relaxations, which documented free pressure transmission between deep and superficial veins of the lower leg. Competent calf perforators would hinder the increase of systolic pressure in the great saphenous vein. This is evident from the pressure/flow behavior across the mitral valve in the heart. Whereas the systolic pressure in the left ventricle increases to about 120 mmHg, competent mitral valve precludes increase in pressure in the left atrium. Incompetent mitral valve enables regurgitant flow, which is accompanied by increase in pressure in the left atrium. Deep and superficial veins of the lower leg form conjoined vessels enabling free pressure transmission and bidirectional flow not only in varicose vein patients but also in healthy people. Bjordal [5] performed, in addition, electromagnetic flow measurements in incompetent calf perforators; he evidenced that the refluxing flow in calf perforators was directed inward, into deep lower leg veins.

As mentioned above, the hemodynamic disturbance in varicose vein disease is evoked by the saphenous reflux [7]. The greater the intensity of saphenous reflux, the severer the clinical staging, the more pronounced the ambulatory venous hypertension and the hemodynamic disturbance, and the larger the diameter of calf perforators and of other segments of superficial and deep veins, in which the Trendelenburg's "private circulation" takes place. The diameter of these enlarged venous segments diminishes after abolition of saphenous reflux [8,9].

The Hemodynamic Impact of Superficial Femoral and Popliteal Vein Incompetence

Another controversy concerns the hemodynamic impact of superficial femoral vein incompetence. Whereas some authors

assert that the superficial femoral vein incompetence does not produce hemodynamic disturbance [10-13], the current opinion argues that incompetence of the femoral vein causes pronounced venous derangement [14-18]. It must be taken into account that various segments of the deep veins possess a different hemodynamic significance. It is generally assumed that iliac vein incompetence is hemodynamically irrelevant. On the other hand, incompetence of all deep lower leg veins (posterior tibial, anterior tibial and fibular veins) causes an irreparable damage to the calf muscle venous pump and induces severe hemodynamic disorder. As concerns femoral vein incompetence, in most studies presenting results after femoral valve repair the saphenous reflux was also abolished, so that the real hemodynamic improvement attributable to the femoral valve repair could not be evaluated. When only selective femoral valve repair was performed, either no improvement or only a light and transitory improvement of several hemodynamic parameters was achieved [19-22]. On the other hand, selective abolition of the great saphenous vein reflux in cases with combined saphenous and femoral vein incompetence restored normal venous hemodynamics in spite of persisting femoral vein incompetence [23,24]. It follows that femoral vein incompetence does not induce hemodynamic derangement in cases with competent saphenous system and competent deep lower leg veins.

Notwithstanding, the superficial femoral and popliteal vein incompetence has some hemodynamic relevance. It apparently promotes the development of small saphenous vein incompetence. Small saphenous vein incompetence occurs 3-4 times less frequently than great saphenous vein incompetence, although the hydrostatic pressure is distinctly higher at the sapheno-popliteal junction than at the sapheno-femoral junction. Competent valves in the superficial femoral vein protect the small saphenous vein from the dilatation effect of the increased intra-abdominal pressure. It was found out that small saphenous vein incompetence was accompanied with incompetence of the deep venous axis (popliteal vein, superficial femoral vein) in the vast majority of patients [12,25]. In a similar way, in patients with great saphenous vein incompetence the deep veins above the sapheno-femoral junction (common femoral, iliac) are incompetent as well [26,27], so that enough blood volume is available in this venous reservoir to counteract the physiological decrease in pressure in the veins below the knee.

The Hemodynamic Paradox

Treatment of varicose veins generates a curious phenomenon. Abolition of saphenous reflux removes the ambulatory venous hypertension of any degree of severity and restores normal hemodynamic values, but at the same time it generates preconditions for reflux recurrence. This chain of events starting the same trouble while fixing the problem has been called hemodynamic paradox [28]. In healthy people, the dividing line of the ambulatory pressure gradient is located at the beginning of the popliteal vein below the knee. After abolition of saphenous reflux in varicose vein patients, the blood contained in incompetent superficial thigh veins is drained during calf pump activity into deep lower leg veins, and the low ambulatory pressure extends from deep lower leg veins into the remainder of the saphenous system in the thigh. As a consequence, a pressure difference arises between the femoral vein and the superficial veins in the thigh and triggers the following run of events: pressure gradient → increased flow through preformed tiny communicating venous channels between the femoral vein and incompetent superficial veins

→ enhanced fluid shear stress on the endothelium → release of nitride oxide and vascular endothelial growth factor → progressive dilatation of the communicating channels → recurrent reflux. The relocation of the dividing line of the ambulatory pressure gradient from below the knee into the thigh might be the reason for the tenacious tendency of varicose veins to recur.

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