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Venous pressure gradients in the lower extremity and the hemodynamic consequences

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Summary

Pressure differences play an important role in the hemodynamics of both arterial and venous circulation. Venous ambulatory pressure gradient of about 35 mm Hg arises during the activity of the calf muscle venous pump between the veins in the thigh and the lower leg; this is the initiator launching venous reflux in varicose vein patients. The hemodynamic consequence of venous reflux is interference with the physiological decrease in venous pressure in the lower leg and foot and the occurrence of ambulatory venous hypertension, the degree of which depends on the magnitude of refluxing blood. Pressure difference occurring between the femoral vein and the remnant of great saphenous vein after high ligation or crossectomy during calf pump activity may be the activator of the process leading to the building of new venous communicating channels, the consequence of which is recurrent reflux. Neovascularization is apparently triggered by this hemodynamic factor, not by the surgical procedure itself, because neovascularization does not occur after harvesting of the great saphenous vein in the groin in people without varicose veins. Venous pressure potentials developing in the lower leg during the calf pump activity force the blood to flow from deep into superficial veins during muscle contraction and in the opposite direction during muscle relaxation. An untoward event caused by venous pressure difference is presented – spontaneous bypassing of a competent valve in the saphenous remnant after crossectomy, which converted a favourable hemodynamic situation into a harmful one. Possible explanation of this undesirable event is offered.

Key words: Ambulatory pressure gradient, venous reflux, varicose vein recurrence, venous hemodynamics

Zusammenfassung

Venöse Druckunterschiede in der unteren Extremität und ihre hämodynamische Auswirkungen

Druckunterschiede spielen eine bedeutende Rolle in der Hämodynamik sowohl der arteriellen wie auch der venösen Zirkulation. Ein ambulatorischer venöser Druckgradient von etwa 35 mm Hg entsteht zwischen den Ober- und Unterschenkelvenen während der Betätigung der muskolovenösen Wadenpumpe. Der Druckgradient löst den venösen Reflux aus; dieser wirkt der physiologischen Drucksenkung in den Venen des Unterschenkels und des Fußes entgegen und fördert die Entstehung der ambulatorischen venösen Hypertonie, derer Höhe vom Ausmaß des zurückströmenden Blutvolumens abhängt. Jener Druckunterschied, welcher während der Betätigung der Wadenpumpe zwischen der V. femoralis und dem Reststamm der V. saphena am Oberschenkel nach Krossektomie entsteht, ist wahrscheinlich der Auslöser des Prozesses, der zur Bildung neuer venösen Verbindungen führt und für den Rezidivreflux sorgt. Nicht der chirurgische Eingriff selbst, sondern dieser hämodynamische Faktor ist wahrscheinlich für die Entstehung der Neovaskularisation verantwortlich; diese kommt nämlich bei Patienten, welche keine Varizen haben, nach der Entnahme der V. saphena magna in der Leiste nicht zustande. Venöse Druckpotentiale, die am Unterschenkel bei der Betätigung der Wadenpumpe entstehen, zwingen das venöse Blut während der Muskelkontraktion von den tiefen in die oberflächlichen Venen und während der Muskelrelaxation in umgekehrter Richtung zu fließen. Es wird über die spontane Überbrückung einer suffizienten Klappe im Reststamm der V. saphena magna nach Krossektomie berichtet, die durch den Druckunterschied ausgelöst wurde, und die günstige Lage in eine hämodynamisch störende umwandelte. Eine mögliche Erklärung dieses Vorgangs wird dargelegt.

Introduction

Direct venous pressure measurements discerned some important hemodynamic evidences concerning the behaviour of venous pressure in the superficial and deep veins of the lower extremity. Whereas in the upright standing position the gravitation force induces steady venous hypertension in superficial and deep veins of the lower extremity, calf muscular activity evokes pressure differences which guide the way of venous flow in the vertical direction (from lower leg toward thigh, against the gravitation force), as well as in the horizontal one (between deep and superficial veins). The basis and consequences of these pressure changes which play an important role in the venous hemodynamics under physiological and pathophysiological conditions, as well as some hemodynamic terms, such as hydrostatic venous hypertension, physiological decrease in pressure, venous ambu-

latory pressure gradient, ambulatory venous hypertension, are discussed and demonstrated in pictures in the following paragraphs.

Venous pressure in quiet standing and during ambulation

In the motionless standing position the hydrostatic pressure in the veins of the lower extremity is equivalent to the height of the blood column between the right atrium and the point of measurement, and causes hydrostatic venous hypertension with an average value of 90 mm Hg at the ankle level. The pressure is equal in superficial and deep veins at the same hydrostatic level [1], and no reflux occurs in quiet standing with relaxed calf musculature [3]. During the activity of the calf muscle venous pump the hydrostatic venous pressure decreases significantly in the veins of the lower leg and foot; this physiological decrease in pressure can be defined as a pressure drop to about 25 mm Hg at the ankle level in upright position. By contrast, the pressure in the popliteal and femoral vein exhibits slight ups and downs during calf pump activity, but the mean pressure does not decrease, it remains at the same level as in the motionless starting position. In this way, the potential for the retrograde flow arises.

Pressure behavior in the veins of the lower leg and in the femoral vein was discerned by Höjensgard and Stürup in 1952 [11]and later confirmed by Arnoldi in 1966 [2], but no lessons were drawn from these findings. Höjensgard and Stürup stated this fact, but did not mention the value of the pressure difference. In Arnoldi's paper the pressure difference between the popliteal and posterior tibial vein reached the value of 33 + –11,8 mm Hg; nevertheless, Arnoldi mentioned it only incidentally in one of a couple of tables among many other numbers and data, without any comment. Recek and Pojer [15] found a pressure difference of 37,4 + –6,4 mm Hg between the femoral and deep lower leg veins, pointed out the importance of this phenomenon and called it ambulatory pressure gradient.

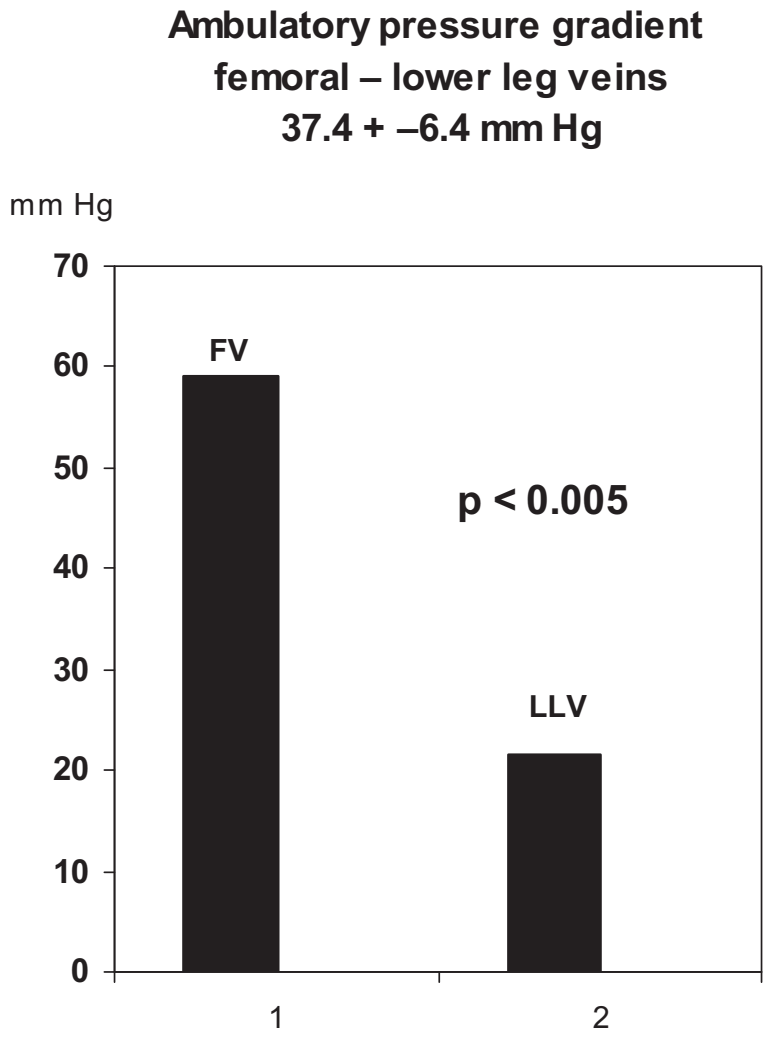

Figure 1: Ambulatory pressure gradient between the femoral and lower leg veins after stopping the activity of the calf muscle venous pump. FV = femoral vein, LLV = lower leg veins.

The pressure difference between the femoral and lower leg veins is depicted in fig. 1, and the pressures curves in the popliteal and posterior tibial vein registered during calf pump activity are displayed in fig. 2.

The pressure difference between the femoral vein and the veins of the lower leg arises also during straining (strain pressure, Valsalva manoeuvre), which increases the pressure in the femoral vein over the hydrostatic value.

Ambulatory pressure gradient triggers retrograde flow

In the upright position, ambulatory pressure gradient represents a power potential, produces tension between

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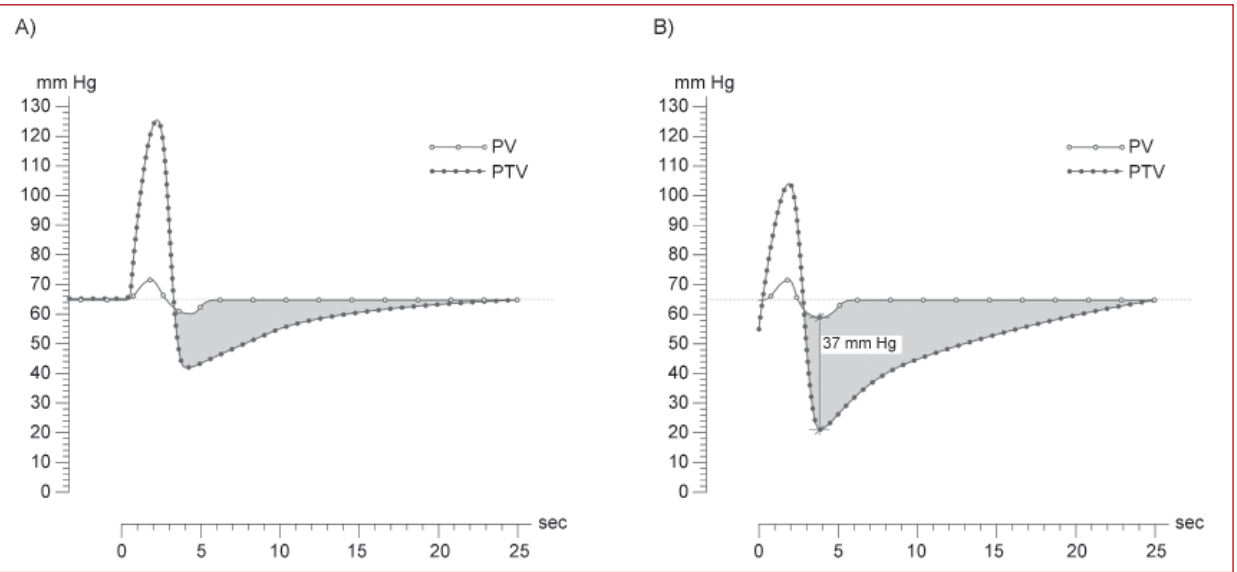

Figure 2: Schematic illustration of overlapping pressures in the popliteal and posterior tibial vein, based on pressure registrations during the first calf muscle contraction and relaxation (A), and during the last one of a series of contractions and relaxations before stopping the muscular activity and reassuming the motionless position (B). The dark area illustrates the ambulatory pressure gradient. PV = popliteal vein, PTV = posterior tibial vein.

thigh and lower leg veins and forces the blood to flow in the retrograde direction from the thigh into the lower leg, both in healthy persons and in varicose vein patients. Competent vein valves prevent retrograde flow and ensure low pressure in lower leg veins in healthy persons. In primary varicose veins, the retrograde flow arises in incompetent venous conduits connecting both poles of the pressure gradient: the higher pole of the pressure gradient, the source of reflux lies in the iliac-femoral-popliteal vein, the lower pole, the issue or re-entry point of reflux lies in one of the deep lower leg veins. Reflux stops when the decreased pressure in the lower leg veins is equalized, i.e. when the value of hydrostatic pressure is reached. The most common source of reflux in primary varicose veins is the sapheno-femoral or sapheno-popliteal junction, so that reflux can be considered as shunting of venous blood from the thigh into the lower leg.

There is no competent valve in the deep veins above the sapheno-femoral junction in the vast majority of varicose vein patients with great saphenous vein incompetence, as documented by Trendelenburg [20] and Ludbrook and Beale [12]. Similar situation exists in patients with small saphenous vein incompetence, where incompetence of the deep venous axis above the sapheno-popliteal junction was found in most cases [10, 16]. Hence, enough blood volume is available in cases with great and small saphenous vein incompetence for a vigorous reflux to cause even the severe form of chronic venous insufficiency. The possible explanation why ambulatory pressure gradient triggers the retrograde flow in varicose veins patients, but not in healthy persons lies in the inherent defective biochemical vein wall structure in varicose vein disease, characterized among others by increased synthesis of type I collagen, decreased synthesis of type III collagen, disproportion of collagen/elastin ratio, lack of elastin, fragmented elastin, abnormally shaped and degenerated smooth muscle cells [6, 13]. This structural deficiency lowers the resistance of the vein wall to the dilating effect of the synergistic force of hydrostatic and strain pressures. The consequence is progressive vein dilation, vein incompetence, and pathological drainage of blood in superficial veins from the thigh into the lower leg. Thus, the coincidental effect of physical laws and of inherent defective vein wall structure may explain the occurrence of venous reflux in varicose vein patients.

Hemodynamic consequence of reflux

The hemodynamic consequence of reflux is ambulatory venous hypertension, which can be defined as aberrancy from the physiological decrease in pressure towards higher pressure values. Reflux may cause any degree of ambulatory venous hypertension from mild to severe forms, depending

only on the amount of refluxing blood volume, and irrespective of the localization in deep or superficial veins [5]. The physiological decrease in pressure shrinks; when the expelled and refluxing volumes equilibrate, no decrease in pressure occurs and the severest form of ambulatory venous hypertension develops.
Fig. 3 displays a schematic illustration of pressure pattern in the great saphenous vein above the ankle during calf pump activity and digital compression of the incompetent trunk of great saphenous vein in the thigh. It demonstrates the physiological decrease in pressure, the ambulatory venous hypertension, and the effect of the saphenous reflux evoking ambulatory venous hypertension.

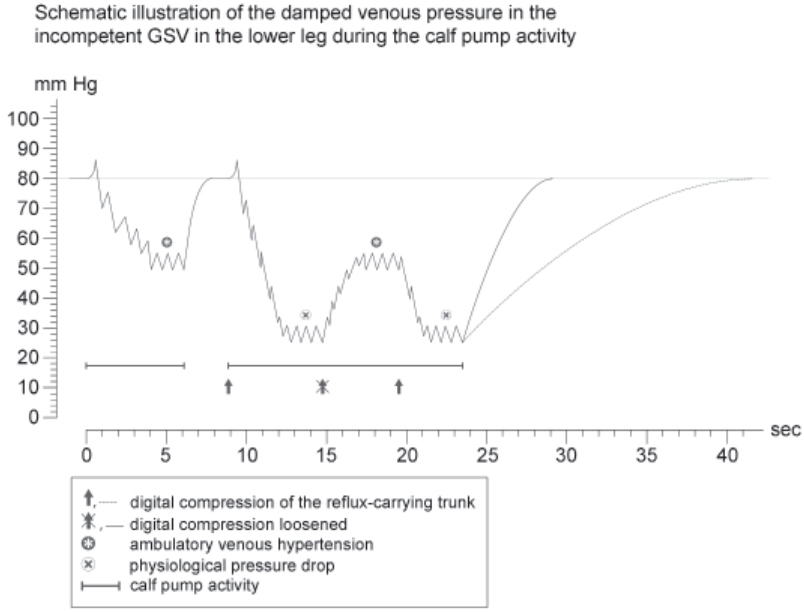

Figure 3: Schematic illustration of the damped pressure in the incompetent GSV in the lower leg during calf pump activity demonstrating physiological decrease in pressure and the influence of saphenous reflux causing ambulatory venous hypertension.

Possible impact of the pressure gradient on varicose vein recurrence

Venous pressure measurements in the incompetent saphenous vein in the thigh completed with the occlusive test [15] showed that the pressure in the proximal part of the great saphenous vein above the tourniquet remained uninfluenced by the calf pump activity, reflecting the pressure in the femoral vein. The pressure in the lower part of the great saphenous vein below the tourniquet decreased due to draining of blood from this segment into the deep lower leg veins, and reflected the pressure in deep lower leg veins. Hence, a pressure difference develops between the femoral vein and the saphenous segment in the thigh below the point of reflux interruption (be it high ligation, crossectomy, or tourniquet); this is apparently the trigger activating the process leading to opening of new channels between the deep and superficial venous system in the thigh.
Neovascularization or neoangiogenesis is considered to be a major cause of recurrent varicosities and was put into causal connection with surgery [7–9, 19]. Nevertheless, neovascularization does not occur in patients with normal veins after harvesting great saphenous vein in the groin for bypass graft; it occurs only in varicose vein patients in connection with recurrences. That means that neovascularization, i.e. building of new venous communications between deep and superficial veins is not triggered by the surgical procedure itself, but by conditions specific to varicose veins. Pressure difference between the femoral and superficial veins in the thigh, a situation arising in primary varicose veins, but not in non-refluxing superficial veins in the thigh, may play a significant triggering role in this event.
Fig. 4 shows retrograde phlebography performed several years after crossectomy. Letter "A" indicates the original site of saphenofemoral junction. A new large meandering venous channel developed between the femoral vein and the saphenous remnant, and caused recurrent reflux.

Pressure differences occurring between deep and superficial veins in the lower leg and causing displacement of venous blood

Simultaneous recordings of the instantaneous, non damped venous pressure in the posterior tibial vein (PTV) and great saphenous vein (GSV) detected pressure differences between both veins during calf muscle contractions and relaxations. In a study performed by Arnoldi [1] on healthy volunteers, the peak systolic pressure in the PTV was higher than in the GSV, the difference reached an average value of +52 mm Hg (range +28 to +89 mm Hg). During muscle relaxation the pressure difference turned round: the pressure in the PTV was lower than in the GSV, the peak

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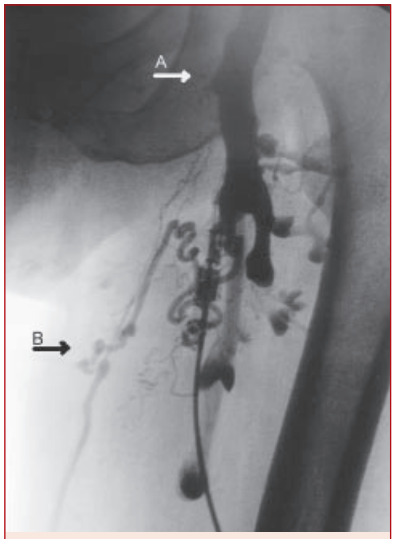

Figure 4: Retrograde phlebography in a patient with recurrent saphenous reflux several years after crossectomy. The contrast dye was injected into the common femoral vein. Retrograde flow was induced by squeezing-relaxation of the calf and simultaneous Valsalva maneuver. A indicates the site of original saphenofemoral junction. The saphenous remnant in the thigh remained open. A new large meandering venous channel developed between the femoral vein and the saphenous remnant and caused recurrent reflux. The diameter of the saphenous vein diminished after crossectomy, and a valve became competent. Nevertheless, a new venous collateral bypassed this competent valve and rendered the saphenous remnant incompetent (B).

diastolic difference came at about –12 mm Hg (range –4 to –21 mm Hg). In another study Arnoldi [82] performed pressure measurements in 7 varicose veins patients and reported a peak systolic difference of +15 + –9.4 mm Hg; the diastolic difference was nearly the same as in healthy persons (–14 + –9.6 mm Hg). Recek and Koudelka [14] found out under similar conditions (simultaneous pressure recordings in the PTV and GSV in 12 varicose vein patients) an average peak systolic pressure difference of +13 mm Hg, and a diastolic difference ranging from –10 to –25 mm Hg depending on the presence or absence of reflux. Whereas reflux did not influence the systolic peak pressure difference, it generated a significantly greater peak diastolic pressure difference; the explanation may be that the retrograde flow rate capacity was greater in the incompetent saphenous vein than in the calf perforators, so that some kind of congestion developed in the GSV during muscle relaxation. Saphenous reflux produces greater driving force pressing the blood from the GSV into the PTV during muscle relaxation. Hence, during muscle contraction a part of the blood escapes from the PTV into the GSV via calf perforators; this blood is further transported within the GSV into the femoral vein in the groin (can be documented with duplex ultrasonography). The pressure rise registered in the GSV during each muscle contraction is the result of the blood afflux into this vein. During muscle relaxation the pressure condition forces the blood to flow from the GSV into the PTV. This bidirectional flow within calf perforating veins during the calf pump activity was documented with duplex ultrasonography in healthy persons [18] and with electromagnetic flow meter in primary varicose veins [4]. The vector of this bidirectional flow is directed into the deep veins; saphenous reflux augments this inward component: larger blood volume streams from the superficial into the deep veins via calf perforators in primary varicose veins than in healthy persons [4]; the larger the saphenous reflux, the larger the flow in inward direction into the deep lower leg veins. The bidirectional flow enables a quick equilibration of pressure changes between the deep and superficial venous systems of the lower leg; simultaneous recordings of the mean pressure in the PTV and GSV showed that the pressure curves were identical [11, 17], a sign typical of conjoined vessels.

Untoward effect of a venous pressure gradient

Retrograde phlebography (fig. 4, letter "B") demonstrates a curious event related to recurrent reflux after crossectomy. A newly formed collateral channel bypassed a single competent valve in the otherwise incompetent saphenous remnant, which rendered the entire saphenous vein incompetent and converted a beneficial situation into a hemodynamically harmful one. Interestingly, both the artery and the saphenous remnant have a common feature: the centrifugal flow. The saphenous remnant was apparently perceived to be an artery, the competent valve an obstacle to centrifugal flow, and it was ultimately bypassed. The trigger of this surprising undesirable event was apparently the pressure difference between the venous segments above and below the competent valve. The case might be considered as a derailment of the repair property of the human organism. The following four quite different situations have a common hemodynamic denominator: a pressure gradient:

1) Occlusion of the arterial stem;
2) sequel of crossectomy or high ligation in varicose veins;
3) inherent a-v. fistulae with pronounced tendency to recur after treatment;
4) the mentioned isolated competent valve in the otherwise incompetent saphenous remnant after crossectomy.

A striking inclination to discharge the pressure difference by building new collateral vessels is typical of all four situations. From this point of view, there is a conspicuous as well as sur-

prising similarity between the arterial occlusion and the situation after crossectomy, namely the tendency to restore the centrifugal flow in both so different situations, with a beneficial effect in arterial occlusions, but an untoward effect devaluating the therapeutic result in primary varicose veins.
Thus, pressure differences apparently play an important role in the pathophysiology of both arterial as well as venous circulation.

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Conflicts of interest

There are no conflicts of interest existing

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