VENOUS PATHOPHYSIOLOGY SHORT REVIEW

Historical view onto the contribution of phlebodynamometry to the knowledge of venous hemodynamics in the lower extremity

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Abstract Contribution of phlebodynamometry to the knowledge of the venous hemodynamics in the lower extremity is assessed from the historical point of view. Höjensgard, Stürup, and Arnoldi yielded in the 50's and 60's of the last century substantial information concerning changes of venous pressure in superficial and deep veins of the lower extremity arising during calf pump activity. They confirmed the previous finding that the venous pressure in the quiet standing position corresponds to the column of blood extending from the point of measurement to the heart. During calf pump activity, the systolic-diastolic amplitudes in lower leg veins are very marked; in contrast to that, they are only modest in the popliteal vein. The mentioned authors documented marked decrease in pressure in the posterior tibial vein as well as in the great saphenous vein arising during calf pump activity in healthy people. Contrary to that, the pressure in the popliteal vein did not decrease; it kept principally the value of the resting hydrostatic pressure. This difference of pressure between the thigh veins and the lower leg veins has been called ambulatory pressure gradient; its value in the examined patient cohort was 37.4±6.4 mm Hg. Ambulatory pressure gradient constitutes the suction force that triggers reflux in incompetent superficial and deep veins; this refluxing flow causes ambulatory venous hypertension. Simultaneous pressure recordings in the posterior tibial vein and great saphenous vein in healthy volunteers documented that the systolic and diastolic pressure amplitudes were similar in both veins. The systolic pressure in the posterior tibial vein increased on average by 75 mm Hg; this generates a strong impulse propelling the venous blood mainly into the popliteal vein, and partially into the great saphenous vein. Thus, there is a double-barreled outflow of venous blood from the lower extremity toward the heart. The steep increase in systolic pressure in the great saphenous vein gave evidence of the incompetence of the calf perforators system in healthy people. At present, plethysmography is able to yield similar information on the hemodynamic status of the lower limbs like phlebodynamometry.

Keywords phlebodynamometry; venous hemodynamics; ambulatory pressure gradient; ambulatory venous hypertension; incompetent calf perforators.

Introduction

Whereas the arterial hemodynamics is a relatively simple system corresponding to the physical laws that influence the flow in the water conduits, the venous hemodynamics in the lower extremity is a complicated matter with compound anatomic structure. The venous flow in the standing position flows toward the heart against the gravitation force within two systems: the deep and superficial veins, which are, in addition, interconnected through many connecting channels called perforators. The basic motive force propelling venous blood toward the heart is the vis a tergo, which is the remaining heart energy in the venules; its value is about 20 mm Hg. In addition, another important motive force is involved in propelling venous blood toward the heart: the calf muscle pump.

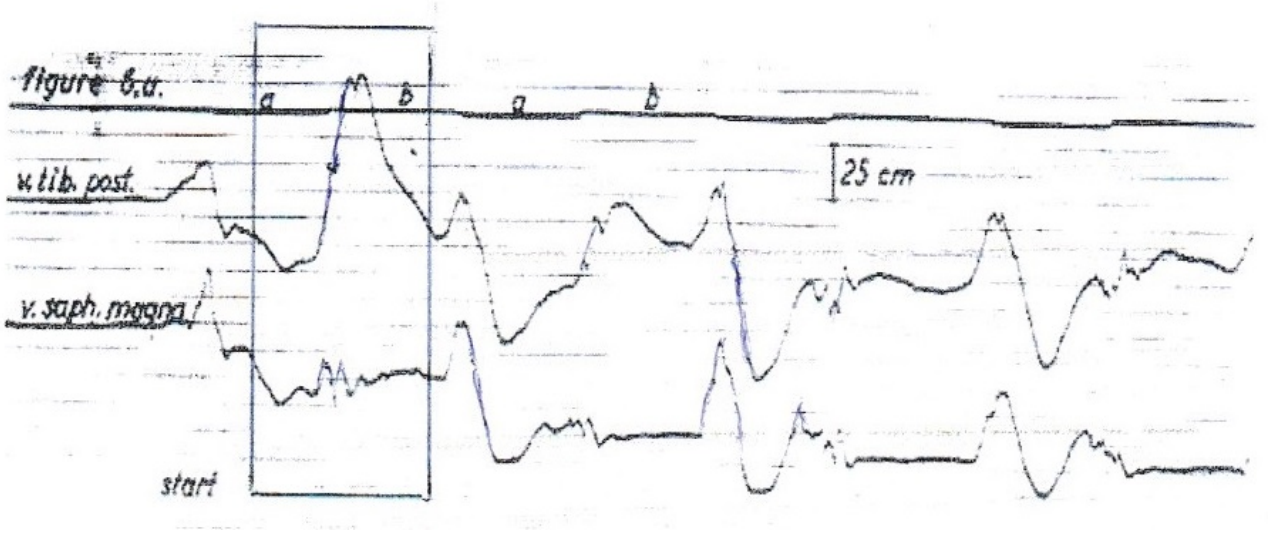

Figure 1 - Simultaneous pressure recordings in the PTV and GSV in a healthy person registered by Höjensgard and Stürup demonstrated among others a transient compression of calf perforators, induced very probably by contracting calf musculature. There was a simultaneous steep increase in pressure both in the PTV and in the GSV during calf muscle contractions, followed by decrease in pressure during the relaxations. The pressure curves in the PTV and the GSV are similar, which documents the presence of bidirectional flow within calf perforators. On one occasion (tagged), there was a high increase in pressure only in the PTV but not in the GSV; the calf perforator(s) behaved in this case as competent. The following contraction displayed also an interesting event: at the beginning of the contraction, calf perforators behaved as competent, at the end of contraction suddenly as incompetent. During the third contraction, the perforators behaved as unrestrained incompetent.

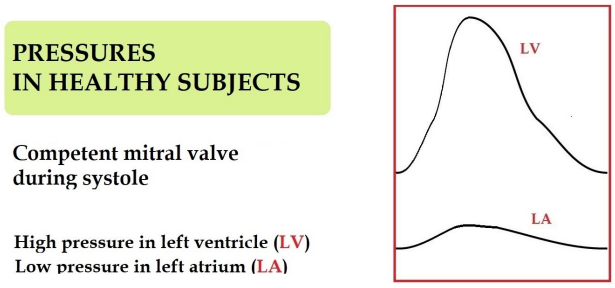

Figure 2 - Pressure differences across the competent mitral valve induced by the systolic contraction of the left ventricle. Whereas the pressure in the left ventricle increases distinctly, the pressure in the left atrium does not.

Calf pump activity produces marked systolic and diastolic venous pressure changes as well as changes in polarity of the venous pressure gradients, which determine the movement of venous blood both in the vertical and horizontal direction.

The correct understanding of the venous hemodynamics in the lower extremity was negatively impacted among others by incorrect theories, e.g. by the stubborn theory of incompetent calf perforators; these were based on subjective considerations, not on results of precise measurements. The results yielded by venous phlebodynamometry contradicted these false interpretations and rendered the first evidence based knowledge about the venous pressure changes and the hemodynamic behavior in the veins of the lower limbs.

Basic insights gained by phlebodynamometry

First attempts of venous pressure measurements were performed in the 20th years of the last century, as mentioned by Höjensgard and Stürup and by Arnoldi[1, 2]. It was stated that the pressure in the saphenous vein in the standing position was roughly equal to the pressure of the column of blood reaching from the point of measurement to the level of the heart. Furthermore, it was found that on muscular activity the pressure in the normal saphenous vein measured above the ankle joint decreased significantly below the value of the resting pressure of 60-90 cm of H_2O [2].

Höjensgard, Stürup, and Arnoldi[1-3] yielded essential contribution to the knowledge of the behavior of venous pressure in superficial and deep veins of the lower extremity under physiological and pathological conditions. Their findings are very important and have durable validity. A catheter was introduced into the vein to be examined, i.e. into great saphenous vein (GSV), posterior tibial vein

(PTV), and popliteal vein (PV), and the pressures in the quiet standing position and during calf pump activity were registered and evaluated.

Phlebodynamometry findings in healthy people

Höjensgard and Stürup[1] performed simultaneous measurements of venous pressure in the GSV, the PTV, and the PV of young healthy men in the motionless standing position and during standing-walking. Very interesting finding resulted from the simultaneous recording of the ambulatory pressure in the PTV and the GSV; it demonstrated inter alia the transient compression of calf perforators (figure 1). The systolic and diastolic pressure amplitudes in both veins were similar. Steep increase in pressure in both the PTV and the GSV was recorded, which documents incompetence of calf perforators enabling outward flow within them. On one occasion, the calf perforators behaved as competent: there was a high increase in pressure in the PTV but no increase in pressure in the GSV. The same pressure behavior is found across the competent mitral valve: contraction of the left ventricle induces marked increase in pressure only in the left ventricle but not in the left atrium (figure 2). The situation during the following contraction in figure 1 deserves also closer attention: at the beginning of the calf muscle contraction the calf perforators behaved as competent, at the end of contraction suddenly as incompetent. During the third contraction, the perforators behaved as unrestrained incompetent. These events might be explained by transient compression of calf perforators by the contracting calf musculature.

Arnoldi[2] performed simultaneous pressure measurements in the PTV, GSV, and the PV in 9 young healthy people. The value of the resting pressure in all three veins corresponded to the pressure of a column of blood reaching from the point of measurement to the level of the heart, which is in agreement with other reports in the literature. The mean systolic increase in pressure was 75 mm Hg in the PTV, 34 mm Hg in the GSV, and 29 mm Hg in the PV. The mean decrease in diastolic pressure was 48 mm in the PTV, 38 mm Hg in the GSV, and 8 mm Hg in the PV. Whereas the systolic-diastolic amplitudes in the PTV and the GSV were marked, they were modest in the popliteal vein; the mean ambulatory pressure in the PV remained practically unaltered during calf pump activity. The pressure curves registered in the PTV and the GSV were similar; the systolic-diastolic amplitudes in the GSV were a little smaller, especially the systolic ones.

The distinct steep increase in pressure in the GSV during calf muscle contractions documents incompetence of the calf perforator system. Competent calf perforators would cause a hemodynamic disaster in case of acute popliteal and femoral vein thrombosis.

The mean systolic rise in pressure in the popliteal vein is caused by the inflow from the deep conductive veins (posterior tibial, anterior tibial, fibular veins) as well as by the inflow from the gastrocnemius veins; as mentioned above, it amounted by 29 mm Hg. In contrast to that, the diastolic decrease was small, only 8 mm Hg; further decrease in pressure is apparently blocked by the competent valves in deep lower leg veins and small saphenous vein. The presence or absence of competent valves in the popliteal vein has little influence upon the popliteal ambulatory pressure. Stürup and Höjensgard measured the pressure in normally valved popliteal veins and in cases, where the valves of the popliteal veins had been destroyed by thrombophlebitis. In both groups of patients the pressure remained almost unaltered during calf pump activity[3]. This finding proves that the conception of the so called “fractionation of hydrostatic pressure” emphasized in the CHIVA theory is at odds with the reality.

Kügler et al.[4] performed venous pressure measurements in 20 healthy young subjects (10 men, 10 women) under different walking conditions. They found that the venous pressure decreased more at the higher walking speed. Subjects with greater calf circumference showed greater venous pressure decrease; the refill time was prolonged. The differences were highly significant when compared with their counterparts. Females had smaller resting pressure. These effects largely result from differences in body height, weight, and calf circumference. Thus, higher degree of muscle activity and greater muscle mass enhanced venous emptying of the healthy human leg. Conversely, restricted ankle joint mobility caused feebler decrease in venous pressure; it reduces the efficacy of the calf muscle pump. Raju et al.[5] performed simultaneous pressure measurements in the foot vein and the GSV in 28 healthy volunteers. They stated significant difference concerning the drop in venous pressure between these veins; the pressure drop in the foot vein was more expressed. As mentioned above, at the end of calf muscle contractions the pressure in the GSV was by 10 mm Hg higher that in the PTV. It is possible that the pressure in the foot vein corresponds with the pressure in the PTV.

Systolic and diastolic pressure differences arising during calf pump activity

Calf pump activity produces pressure gradients both between the popliteal vein and lower leg veins and between the superficial and deep veins of the lower leg. These pressure gradients change their polarity during calf pump activity and influence the behavior of venous flow both in the vertical direction (toward the heart, away from the heart) and in the horizontal direction (outward, inward flow within calf perforators). Arnoldi[6] reported that the systolic-diastolic pressure difference between the PTV and the PV was plus 46 mm Hg and minus 40 mm Hg, and

between the PTV and the GSV +41 mm Hg and -10 mm Hg. Some calf perforators might be compressed during the contraction of the calf musculature, which increases the systolic pressure difference between the PTV and the GSV. In patients with varicose veins and larger calf perforators the systolic pressure difference between the PTV and the GSV came to modest +15 mm Hg. The marked increase in systolic pressure in the PTV (the top of the catheter was at the level of the greatest circumference of the calf) may be attributed to two factors: the suddenly increased inflow from the muscular tributaries and the compression of the deep central trunks.

As the blood must flow from the point with higher pressure to the point with lower pressure, the simultaneous pressure recordings in the three veins permit assessment of the venous flow direction during calf pump activity. During calf muscle contractions, the blood is expelled from the PTV predominantly into the PV (main outflow route), partially also into the GSV (collateral outflow route). Thus, there is a double-barreled systolic streaming in the centripetal/orthograde direction toward the heart. During the diastole, there is a short physiological retrograde flow lasting 200-300 milliseconds until the competent valves close[7]. There is a bidirectional systolic-diastolic flow within calf perforators.

Competent valves in the lower leg veins block the decrease of the diastolic pressure in the PV and enable the development of ambulatory pressure gradient.

Ambulatory pressure gradient

According to the measurements performed by Arnoldi[2, 6], the difference between the PV and the PTV after cessation of the calf pump activity came to 40 mm Hg in healthy people, to 33±11.8 mm Hg in varicose vein patiens and to 34±13.5 mm Hg in patiens with post-thrombotic syndrome. Recek and Pojer[8] found that after elimination of saphenous reflux in varicose veins patients this difference amounted to 37.4±6.4 mm Hg, and called it ambulatory pressure gradient. The dividing line of the ambulatory pressure gradient is situated at the beginning of the popliteal vein, i.e. just beneath the knee joint. The higher pole of the ambulatory pressure gradient lies in the thigh veins, the lower pole in lower leg veins. In incompetent superficial and/or deep veins the ambulatory pressure gradient triggers refluxing flow from the thigh veins into the lower leg veins. Figure 3 depicts the ambulatory pressure gradient by means of superposing the pressure curves registered in the popliteal vein and in the posterior tibial vein.

Tauraginskij et al.[9] measured the acceleration of blood flow during venous reflux, peak velocity of venous reflux and time required to achieve the peak velocity in a clinical study using duplex ultrasonography in 80 patients. Physical models were used to demonstrate the difference in acceleration between the free-fall stream and the reflux flow in the saphenous vein. They concluded that the acceleration of blood flow during reflux exceeded the value of gravitational acceleration, suggesting the action of an additional non-gravitational force. They stated that the calf muscle pump might create suction force by decreased pressure during muscle diastole. As mentioned above, this suction force has been labeled ambulatory pressure gradient. Its lower pole is situated in deep lower leg veins.

Phlebodynamometry findings in varicose vein disease

Recek and Koudelka[10] performed simultaneous pressure measurements in the PTV and the GSV in 12 patients with primary varicose veins. The pressures were registered in the quiet standing position in the relaxed limb and during calf pump activity (toe stands). Thereafter, the same measurements were performed with a tourniquet applied in the thigh in order to suppress the saphenous reflux. During quiet standing, the pressure values both in the PTV and the GSV came to 70 mm Hg. In the presence of reflux, the pressure decreased at the end of pump activity by 38 mm Hg in the PTV, but by only 17 mm Hg in the GSV. After the elimination of saphenous reflux, the pressure decreased by 46 mm Hg in the PTV and by 37 mm Hg in the GSV. The difference between the condition with and without saphenous reflux was highly significant ($p < 0.001$), which documents the pathogenic role of saphenous reflux. Differences in the systolic and diastolic pressures between the PTV and the GSV were also determined. During muscle contractions, the pressure in the PTV was higher by 13 mm Hg than in the GSV; the presence or absence of saphenous reflux did not influence the difference. The pressure gradient in the systolic phase induces outward flow within calf perforators. During calf muscle relaxations, the pressure gradient turned round; the pressure in the GSV was higher than in the PTV. In addition, the presence of saphenous reflux increased significantly this difference from 9 mm Hg to 21 mm Hg ($p = 0.003$, figure 4). The recovery time, i.e. the time necessary to reach the resting pressure level, was prolonged after elimination of saphenous reflux from 4 to 11 seconds in the PTV and from 2 to 11 seconds in the GSV ($p < 0.001$).

Fukuoka et al.[11] performed foot venous pressure measurements in 257 legs of 196 patients with chronic venous insufficiency. They assessed the percentage decrease in pressure from the rest after manual calf compression, the rate of increase in pressure during 4 seconds after compression, and the time to 50% recovery of pressure after release of compression. They found that the incidence of skin changes increased with the pathological behavior of the foot pressure (lesser drop of pressure, shorter refilling times). Foot venous pressure parameters correlated well with the clinical manifestations

of venous reflux. Payne et al.[12] measured ambulatory venous pressure in 360 limbs with wide spectrum of venous disease and found that there was a linear trend toward more severe skin damage with increasing ambulatory venous pressure. Ulceration was associated with higher ambulatory venous pressure than were lipodermatosclerosis, eczema or pigmentation. Nicolaides et al.[13] performed venous pressure measurements in 153 limbs with reflux in superficial veins and in 83 limbs with deep venous disease. No ulceration occurred in limbs with ambulatory pressure < 30 mm Hg; there was a 100% incidence with ambulatory pressure > 90 mm Hg. In the groups studied, increased incidence of ulceration was associated with an increase in ambulatory venous pressure irrespective of whether the venous problem was the result of superficial or deep venous disease.

In varicose vein disease, the saphenous reflux is the most important pathological hemodynamic factor; it is released by the ambulatory pressure gradient and causes ambulatory venous hypertension. The route of saphenous reflux is as follows: iliac vein --> common femoral vein --> saphenous vein --> calf perforators (re-entry points) --> deep lower leg veins. The hemodynamic impact of saphenous reflux is demonstrated in figure 5. No decrease in ambulatory venous pressure during calf pump activity either in the PTV or in the GSV was registered in patients with very strong saphenous reflux. Elimination of saphenous reflux restored physiological decrease in pressure both in the PTV and the GSV. Figure 6 shows the schematic illustration of ambulatory pressure differences in healthy people (A), the ambulatory venous hypertension in varicose veins patients induced by saphenous reflux (B), and restoration of physiological pressure conditions after elimination of saphenous reflux (C).

In primary varicose veins, reflux within calf perforators is a **diastolic inward flow**. The outward flow within calf perforators is a **systolic**, centripetal, via the saphenous vein toward the heart (i.e. in the physiological direction) oriented streaming; it does not cause any hemodynamic disturbance nor induces ambulatory venous hypertension.

Plethysmography is able to yield similar information on the venous hemodynamics like phlebodynamometry, so that phlebodynamometry is not necessary in the current clinical practice. Although the measured parameters are different, the curves registered by phlebodynamometry and plethysmography are similar (figure 7). Phlebodynamometry measures changes of venous pressure, plethysmography changes of venous volume. Both methods are able to quantify the hemodynamic status and the therapeutic effect.

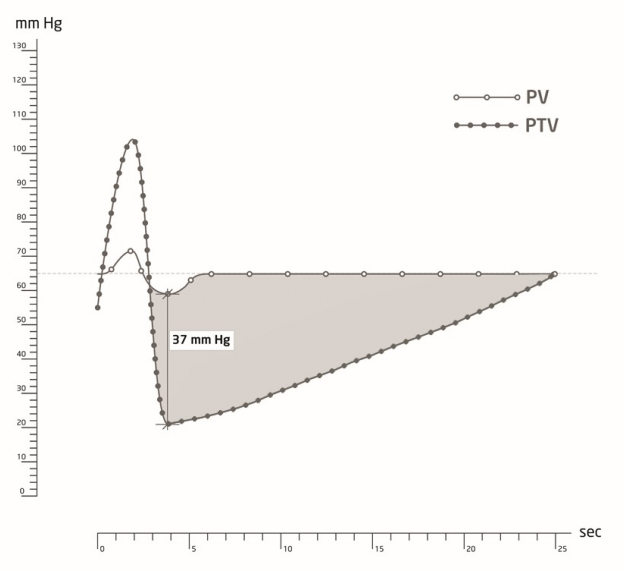

Figure 3 - Demonstration of the ambulatory pressure gradient by overlapping of pressure curves in the PTV and the PV at the end of calf pump activity. Whereas the pressure amplitudes in the PTV are very marked, the amplitudes in the PV are small. The shadow area showcases the ambulatory pressure gradient. PV = popliteal vein, PTV = posterior tibial vein.

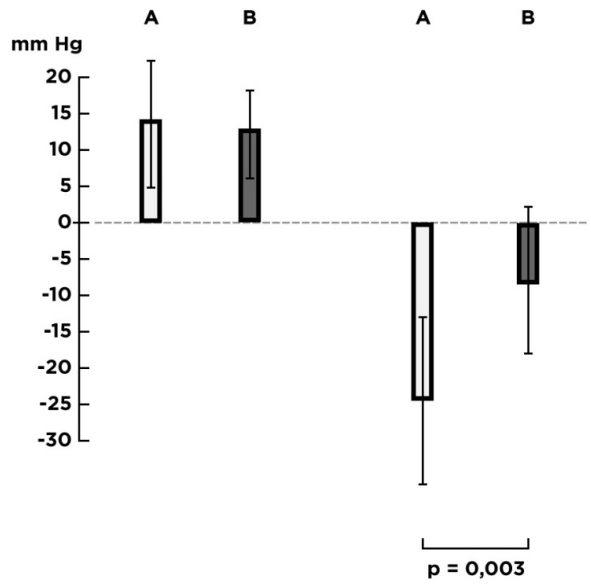

Figure 4 - Pressure differences between PTV and GSV arising during calf pump activity. During calf muscle contractions, the pressure in the PTV is higher than in the GSV; the difference comes to 13 mm Hg and is not influenced by the reflux. During calf muscle relaxations, the pressure in the GSV is higher than in the PTV; saphenous reflux increases significantly the pressure difference. A = with saphenous reflux, B = after elimination of reflux.

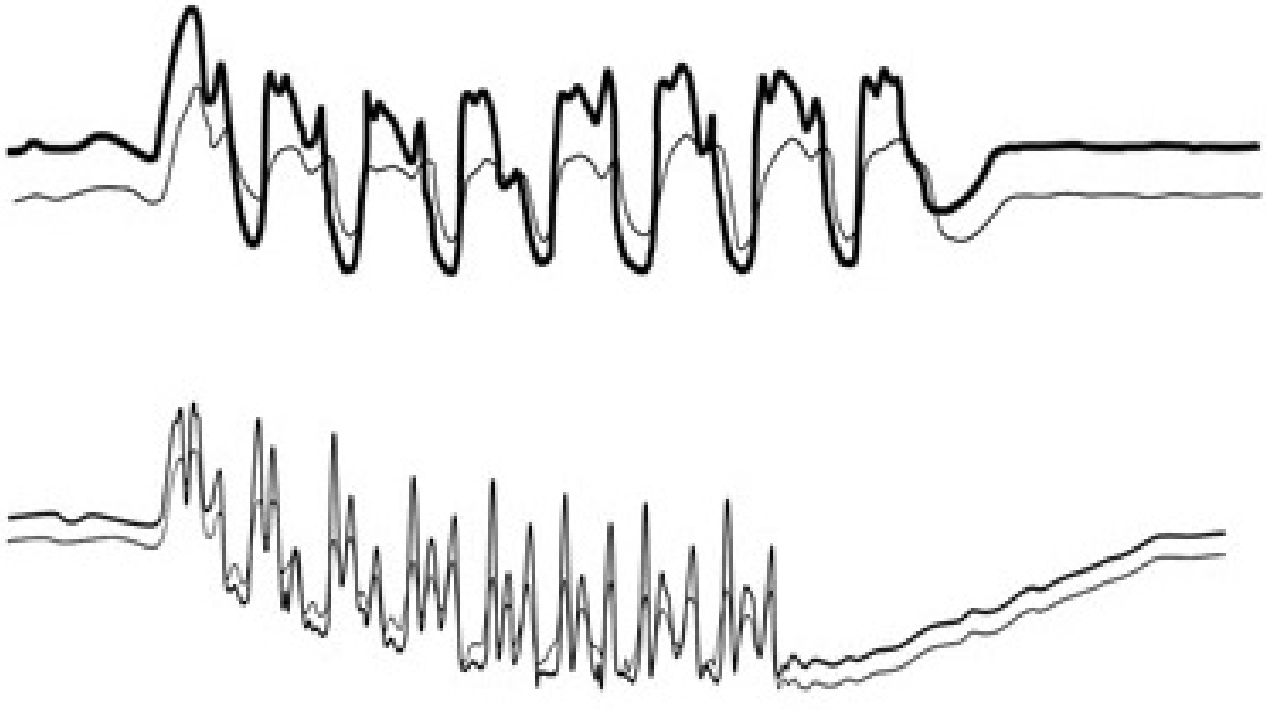

Figure 5 - Simultaneous recordings of ambulatory pressure in the PTV (upper thick curve) and GSV (lower thin curve). Above: No decrease in ambulatory venous pressure both in the PTV and in the GSV due to very strong saphenous reflux. Below: Physiological decrease in ambulatory venous pressure both in the PTV and the GSV after elimination of saphenous reflux.

Correlation of phlebodynamometric and plethysmographic findings

Payne et al.[14] compared air plethysmography and ambulatory venous pressure measurement in patients with venous disease. They found that the residual volume fraction did not correlate with ambulatory venous pressure measurements. The venous refilling time gained using air plethysmography correlated poorly with that one obtained by venous pressure measurements. Similarly, Raju et al.[15] found little correlation between air plethysmography and ambulatory venous pressure. Nonetheless, other authors found and presented the opposite. Tachibana et al.[16] investigated the relationship between air plethysmographic volume parameters and foot venous pressure measurements under exercise loading in patients with primary varicose veins. Venous filling index correlated most closely with ambulatory venous pressure. Carrel et al.[17] compared results obtained by quantitative photoplethysmography and venous pressure measurements in 20 normal subject, 20 patients with varicose veins, and 20 patients with chronic venous insufficiency. Quantitative photoplethysmography correlated closely with measurements of ambulatory venous pressure with respect to drop of intravenous pressure in normal subjects, in patients with varicose veins, and post-thrombosis patients. Similar results were presented by Norris et al.[18]. Further correlations between the phlebodynamometric and plethysmographic results were mentioned in another article[19]. They showed principally good correlation between these two methods.

Changes of venous pressure in post-thrombotic venous obstructions

Labropoulos et al.[20] performed foot-arm venous pressure differential measurements in patients with post-thrombotic venous outflow obstructions as well as in control subjects. Venous obstructions were localized from the popliteal to the iliac veins. With the patient in the

supine position, simultaneous venous pressure recordings were obtained from both the foot and arm before and after reactive hyperemia of the foot induced by a 3-minute arterial occlusion of the thigh. Normal values in healthy subjects were 1±0.6 mm Hg at rest and 2.7±1.2 mm Hg after reactive hyperemia. In patients with popliteal vein obstructions the values were 1.5±0.8 mm Hg at rest and 3.1±0.9 mm Hg after reactive hyperemia; in femoropopliteal obstructions the corresponding values were 4.4±1.3 mm Hg at rest and 7.3±1.1 mm Hg after reactive hyperemia, and in iliofemoral obstructions 6.3±2.6 mm Hg at rest and 8.9±1.8 after reactive hyperemia. Iliac vein obstructions induced the severest hemodynamic disturbance. As concerns the collateral venous circulation, the superficial venous system was of significant importance in only 10% of cases. Thus, the collaterals of the deep venous system seem to play an important role in bypassing venous obstructions. The anatomical extend of obstruction and the degree of collaterization determines the hemodynamic severity of chronic venous obstructions.

Kurstjens et al.[21] measured simultaneously venous pressure in the common femoral vein and the dorsal foot vein in 22 patients with unilateral post-thrombotic obstructions of the iliofemoral veins. They compared the values of ambulatory pressure in the afflicted and unaffected limb. After quiet standing the patients underwent a standardized treadmill test. After walking, the post-thrombotic limbs showed a significantly higher mean common femoral vein pressure increase of 28.1±21.0 mm Hg, compared with only 2.1±6.2 mm Hg in control limbs. For comparison: in the popliteal vein the systolic pressure increases in healthy people by 29 mm Hg after calf muscle contraction. Less difference was observed in the dorsal foot vein: net drop of 36.8±22.7 mm Hg in affected limbs vs. 48.7±23.1 mm Hg in non-affected limbs. Changes in common femoral vein pressure after walking yielded the best discrimination between affected and non-affected limbs. In another study, Kurstjens et al. assessed the significance of collateral blood flow in chronic venous obstruction[22]. Resting intravenous pressure in the common femoral vein was measured bilaterally in the supine position in 14 patients with unilateral post-thrombotic obstruction of the iliac vein. In addition, pressure in control limbs was also measured in the common femoral vein after sudden balloon occlusion of the external iliac vein. Median common femoral vein pressure was 17.0 mm Hg in diseased limbs compared to 12.8 mm Hg in controls ($p = 0.001$), and 23.5 mm Hg in controls after sudden balloon occlusion of the external iliac vein ($p = 0.009$). The study showed the beneficial impact of the venous collateral circulation around the iliac vein obstruction.

Neglen et al.[23] reported on results of balloon angioplasty and stenting of common femoral and iliac veins. In addition to other diagnostic methods, venous pressure measurements were also performed. The patients were divided into two groups: with venous occlusion alone, and with combined occlusion and reflux. Patients with reflux and obstruction had more severe disease, as compared with obstruction alone. The rate of active ulcers was low (3%) in limbs with obstruction only, compared with 24% in limbs with combined obstruction and reflux. Foot-arm pressure differential, dorsal foot hyperemia pressure and ambulatory dorsal foot venous pressure were measured before intervention and repeated at follow up. In patients with obstruction only, the ambulatory pressure drop was 71±17% before treatment and 70±15% post-stent. In patients with obstruction plus reflux, the pressure drop was 58±19% and 57±20%, respectively.

The presence of reflux caused higher ambulatory venous pressure and shorter venous refilling time. As concerns values for foot-hand pressure differentials, significant improvement after stenting was registered ($p < 0.0001$), which hints at the hemodynamic impact of the venous obstruction. In addition, successful stenting relieved the patients of pain and swelling, and induced healing of leg ulcers. Nonetheless, the obstruction in the iliac veins did not influence the decrease in ambulatory venous pressure, albeit the resting venous pressure in the common femoral vein might be increased, as mentioned in the paper by Kurstjens et al.[22].

The degree at which venous stenosis is hemodynamically “critical” is not known. It has been perceived that when proximal outflow obstruction is relieved, reflux intensity will increase. Interestingly, ambulatory venous pressure, venous refilling time, and venous filling index in mL/s did not worsen after stent insertion in patients with preoperative reflux. The capacity of the venous collateral circulation was apparently sufficient enough for feeding the reflux. Phlebography performed before stenting demonstrated collateral vessels; they disappeared after stenting.

Raju et al.[24] presented results of saphenectomy in patients with deep venous obstructions confirmed on ascending venography and compared them with those in patients without obstruction. The deep venous obstruction was localized below the groin. Foot-arm venous pressure differential, reactive hyperemia foot pressure elevation, ambulatory foot venous pressure measurements, and air plethysmography were performed. The foot-arm pressure differential technique was the mainstay in functional assessment of obstruction. In most cases, the venous obstruction was adequately collateralized and functionally well compensated. In patients with femoral vein occlusion, the saphenous trunk in the thigh serves in the quiet condition and during calf pump contractions as a part of collateral circulation.

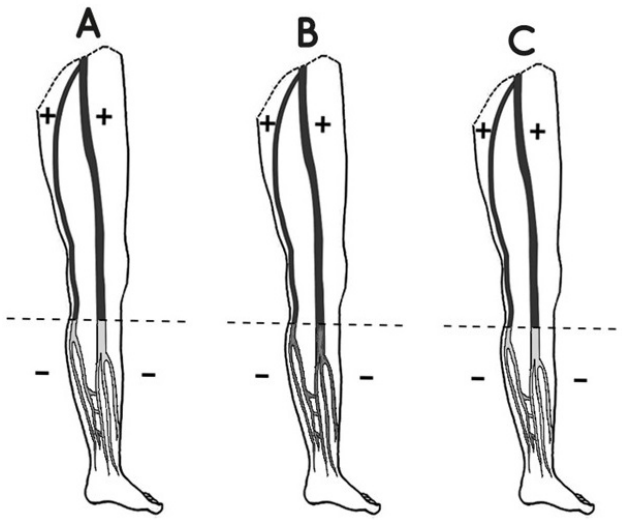

Figure 6 - Illustration of ambulatory pressure differences after stopping the calf pump activity in healthy people (A), in varicose vein patients with saphenous reflux (B), and after elimination of saphenous reflux (C). During calf pump activity, substantial pressure changes occur only in the lower leg veins. In healthy people (A) ambulatory pressure gradient arises between the thigh veins and lower leg veins. The pressure in the superficial veins below the knee is by 10 mm Hg higher than in the deep veins. B: Saphenous reflux induces ambulatory venous hypertension, which diminishes the ambulatory pressure gradient depending on the intensity of saphenous reflux (dark lower leg veins). C: Restoration of physiological venous pressure conditions and of normal ambulatory pressure gradient after elimination of saphenous reflux.
+ means higher pressure, - means lower pressure. The dotted line illustrates the dividing line of the ambulatory pressure gradient; it is situated at the beginning of the popliteal vein.

Nevertheless, the relative contribution of the saphenous vein trunk in collateral compensation is minor; non-saphenous venous collaterals, e.g. deep collaterals, compensate easily and rapidly the elimination of collateral function provided by the dilated saphenous vein.

Saphenectomy was clinically well tolerated in patiens with venous obstruction; no significant worsening of objective measures of obstruction was documented after saphenectomy. Improvement in reflux and calf venous pump function was also largely similar in both groups, with and without venous obstruction. In cases with venous obstruction the saphenous reflux seems to be the main factor determining the aggregate severity of the hemodynamic disorder.

During the diastole, the saphenous trunk is the main conduit for saphenous reflux. Therefore, the calf pump must convey, in addition to the normal circulating blood, the reflux volume against the venous blockade. When the saphenous trunk and the saphenous refluxing volume are eliminated, the capacity of the remaining collateral circulation seems to be sufficient for transporting the circulating blood toward the heart.

Conclusion

Calf pump activity produces venous pressure gradients with changing polarity and promotes the complexity of the venous hemodynamics in the lower extremity. Phlebodynamometry yields evidence based information about these events. Höjensgard, Stürup, and Arnoldi presented in the 50's and 60's of the last century information on the systolic and diastolic venous pressures induced during calf pump activity under physiological and pathological conditions in superficial and deep veins of the lower limb. Their findings are substantial and have durable validity.

Very important was the discovery that whereas the ambulatory pressure fell considerably in the veins below the knee, it did not decrease in the popliteal and femoral veins, where it kept the resting hydrostatic pressure value. In the examined patient cohort, this difference in ambulatory pressure had the value of 37.4±6.4 mm Hg; it has been called ambulatory pressure gradient. The suction force of the ambulatory pressure gradient triggers reflux in incompetent superficial and/or deep veins of the lower extremity. Saphenous reflux induces ambulatory venous hypertension. Good correlation has been found between the ambulatory venous hypertension and the clinical symptoms.

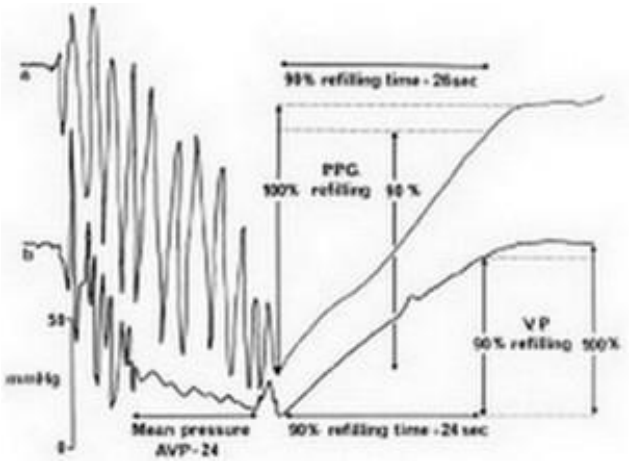

Figure 7 - Plethysmographic curve displaying volume changes (above) and phlebodynamometric curve displaying pressure changes (below) during calf pump activity are similar. Both methods are able to quantify the hemodynamic status and the therapeutic effect.

Simultaneous pressure recordings in the PTV and GSV in healthy people registered during calf muscle contractions displayed steep increase in pressure not only in the PTV, but also in the GSV, which documented that systolic outward flow takes place within calf perforators. Calf perforator system in healthy people is incompetent. Competent calf perforators would augment the hemodynamic disorder in case of acute popliteal and femoral vein thrombosis.

The systolic pressure in the PTV increases by 75 mm Hg. This is a strong impulse propelling the venous blood toward the heart. The venous blood is expelled mostly into the popliteal vein, and partially into the GSV. Thus, there is double-barreled outflow of venous blood from the lower extremity.

In patients with post-thrombotic obstructions, iliac vein occlusions are accompanied with the most untoward impact on the venous hemodynamics. Ambulatory pressure in such cases increases mainly in the femoral vein, whereas in the lower leg veins the net drop was only slightly less or even similar like in healthy people. Femoral vein obstructions tend to be well compensated by deep venous collaterals. In patients with combined obstruction and reflux, the reflux intensity is the main factor disturbing the venous hemodynamics. Saphenectomy was clinically well tolerated in patients with femoral vein obstructions.

Expression of thanks

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