THE HAEMODYNAMIC ROLE OF CALF PERFORATORS

Čestmir Reček

Retired, formerly Department of Surgery, University Hospital, Hradec Kralove, Czech Republic

REVIEW PAPER

Phlebological Review 2014; 22, 2: 45–48
DOI: 10.5114/pr.2014.48903

Submitted: 13.01.2015
Accepted: 17.01.2015

ADDRESS FOR CORRESPONDENCE:
Čestmir Reček, MD, PhD
Retired, formerly Department of Surgery, University Hospital
e-mail: cestmir@recek.at

ABSTRACT

The haemodynamic role of calf perforators under physiological and pathological conditions continues to be a controversial issue. According to the prevailing opinion, calf perforators in healthy people are competent, enable only one-way flow from superficial into deep veins and hinder escape of venous blood from deep into superficial veins, while under pathological conditions these perforators widen, become incompetent and make outward flow possible, induce venous hypertension and create preconditions for the development of chronic venous insufficiency and leg ulcers. In this review paper the evidence is presented, including the results of studies utilising direct venous pressure measurements, plethysmographic findings and electromagnetic flow measurements, that actually an outward flow through calf perforators should not be interpreted as reflux, does not cause ambulatory venous hypertension and that bidirectional flow through these perforators is found not only in pathological conditions but also in healthy people. Calf perforators enable free pressure transmission between the deep and superficial veins, enable flow in both directions, and make deep and superficial veins of the lower leg conjoined vessels. The genuine venous reflux within calf perforators is an inward and not outward flow.

Key words: calf perforators, venous reflux, ambulatory venous hypertension.

INTRODUCTION

The haemodynamic role of calf perforators has not yet been unanimously perceived. A generally accepted opinion claims that calf perforators in healthy people are competent, enable only one-way flow from superficial into deep veins, and prevent escape of venous blood from deep into superficial veins. If in the course of varicose vein disease calf perforators widen, they become incompetent and make outward flow possible. This outward flow within calf perforators is generally regarded as a pathological reflux causing ambulatory venous hypertension and leading to the development of chronic venous insufficiency, including venous/varicose ulcers. Unfortunately, this concept is based on subjective opinions and has never been backed by a body of evidence.

The objective of this article is to present an evidence-based view into the haemodynamic role of calf perforators, supported by the results of venous pressure measurements, plethysmographic findings, and electromagnetic flow assessment. Specifically, the goal of this paper is to answer the following questions:

1. Is the outward flow in calf perforators a reflux?
2. Does the outward flow within calf perforators cause ambulatory venous hypertension?
3. Do calf perforators in healthy people work as a one-way system?

IS THE OUTWARD FLOW IN CALF PERFORATORS A REFLUX?

As mentioned in the introduction, an outward flow within calf perforators is generally referred to as reflux. Because there is no consensus as to the real substance and characteristic attributes of venous reflux, the definition of venous reflux is given below to avoid misconceptions.

Venous reflux in the lower extremity is a diastolic (i.e. occurring during relaxation of calf muscles, and not during heart diastole), centrifugal (i.e. the flow directed away from the heart), flow of venous blood within incompetent venous channel(s) connecting both poles of the ambulatory pressure gradient that arises during the diastolic phase of the calf muscle pump activity (this pressure gradient is at the level of 37.4 ±6.4 mmHg).

The higher pole of this pressure gradient is located in the popliteal, femoral, or iliac vein, the lower pole in the deep lower leg veins (importantly, this significantly differs from the situation observed in a static, not moving leg, where the highest pressure is measured at the level of the ankle). Reflux takes place during relaxation of calf musculature, exceeds the duration of the physiological centrifugal flow lasting 200-300 milliseconds, and stops as soon as the pressure difference has been equalised. Such a reflux can also be evoked by an increased intra-abdominal pressure propagating into the iliac, femoral, and popliteal veins. Reflux interferes with the physiological drop of pressure arising in the deep and superficial veins of the lower leg and foot during calf pump activity. Such venous reflux results in the so-called ambulatory venous hypertension (i.e. hypertension present during walking activity). The clinical severity of venous ambulatory hypertension depends on the volume of such a centrifugal flow (expressed in ml/s).

When confronted with this definition, an outward flow within calf perforators does not fulfil the criteria characteristic for venous reflux; on the contrary, it turns out to be the exact opposite of such a reflux. Whereas venous reflux (according to the above-presented definition) is a diastolic, centrifugal, pathological flow, the outward flow in calf perforators is a systolic (i.e. occurring during contraction of calf muscles), centripetal (i.e. directed towards heart), physiological flow evoked by the contraction of the "peripheral heart" and directed via the great saphenous vein (GSV) or other superficial veins toward the heart. Such a flow does not interfere with physiological decrease of venous pressure during muscle pump activity and does not result in ambulatory venous hypertension or other haemodynamic disorders. Thus, it should not be defined as venous reflux. According to the above-presented definition, the genuine venous reflux within calf perforators is an inward, not outward, flow, which drains venous blood from the iliac veins via an incompetent saphenous system into the deep veins of the lower leg. Bjordal [1] has documented this phenomenon using electromagnetic flow measurements in the GSV and in incompetent calf perforators (ICP). The flow within ICPs exhibited a bidirectional pattern with a clearly prevailing inward component. This bidirectional flow in calf perforators was evoked by pressure differences between the deep and superficial veins of the lower leg arising during calf muscle pump activity, which was documented by simultaneous pressure measurements in the posterior tibial vein (PTV) and the GSV in varicose vein patients during calf muscle contractions and relaxations [2]. During muscle contraction, pressure in the PTV was about 14 mmHg higher than in the GSV, which promoted outward flow via calf perforators from the deep veins of the lower leg into the saphenous system. Then the blood flowed through the GSV into the femoral vein. As such, it could be described as centripetal flow directed physiologically toward the heart. During muscle relaxation, the pressure gradient was reversed: the pressure in the GSV was higher than in the PTV, which triggered an inward flow. It was shown that reflux in the GSV significantly enhanced this pressure gradient during calf muscle relaxation, in comparison with legs without saphenous reflux ($p < 0.005$) [2]. An increased pressure gradient evoked by saphenous reflux augments the amount of inward flow into the deep veins of the lower leg.

DOES THE OUTWARD FLOW WITHIN CALF PERFORATORS CAUSE AMBULATORY VENOUS HYPERTENSION?

Cockett and Jones [3], on the basis of anatomical cadaver studies, described calf perforators located on the medial side of the lower leg above the ankle. These perforators have since been coined "Cockett's perforators". In the above-mentioned article, Cockett did not specify the total number of preformed studies, mentioning only two anatomical examinations and 10 patients operated on, with a follow-up of several months after ligation of the perforators. He stated that perforating veins drained venous blood from the skin and subcutaneous tissue in the gaiter area exclusively into the deep veins of the lower leg, and believed that these calf perforators in healthy people were equipped with competent valves enabling only unidirectional flow: from the superficial into the deep veins. He also believed that in the course of venous disease these perforating veins of the lower leg become incompetent and during calf muscle contractions a considerable volume of blood escapes from the deep into the superficial veins, which in turn induces venous hypertension in the veins of skin and subcutaneous tissue and creates a precondition for the development of leg ulcers. Cockett labelled this pathological situation "blow-out syndrome". However, his concept concerning the role of calf perforators has never been validated, especially by precise functional measurements. Moreover, systolic (i.e. occurring during muscle contractions) pressure is physiologically higher in the deep veins of the lower leg than in the saphenous system, both in varicose vein patients and in healthy individuals, without causing any harm to the subfascial tissue. Nevertheless, in the second half of the 20th century this pathomechanism has been generally accepted.

Still, the actual behaviour of calf perforators is very different. Outward flow in these perforating veins does not result in ambulatory venous hypertension. Direct venous pressure measurements in ICPs performed by Bjordal demonstrated that high resting pressure lowered during calf muscle pump activation once the saphenous reflux was interrupted [1]. This was direct proof that ICPs do not induce ambulatory venous hypertension. In primary varicose veins haemodynamic disorders are evoked by saphenous reflux and not by an outward flow in calf perforators. In the patients with severe saphenous reflux and large ICPs (Fig. 1 shows a typical example) simultaneous venous pressure measurements were performed in the PTV and GSV. These patients presented with very severe ambulatory venous hypertension, which was manifested by no pressure decrease during calf pump activity. After interruption of saphenous reflux, a physiological decrease of pressure during muscle pump activation was restored, despite the presence of large ICPs [4, 5]. Similarly, the irrelevance of ICPs was proven by strain gauge plethysmography. The abolition of saphenous reflux at the saphenofemoral junction eliminated severe pre-treatment haemodynamic disturbances and restored normal plethysmographic values one week after the crossectomy, in spite of the presence of large ICPs [6]. On the other hand, selective interruption of ICP did not improve the haemodynamic situation, as has been demonstrated in several studies [7-10]. All of these findings unequivocally prove the blow-out theory to be false.

DO CALF PERFORATORS IN HEALTHY PEOPLE WORK AS A ONE-WAY SYSTEM?

The pressure gradient arising between the deep and superficial veins of the lower leg during calf muscle pump activity constitutes the main mechanism driving the flow in calf perforators. Simultaneous pressure measurements in the PTV and GSV in healthy individuals demonstrates that during calf muscle contraction the pressure in the PTV is higher than in the GSV [11], which creates a precondition for an outward flow. This systolic outward flow though calf perforators in healthy people was revealed by Sarin *et al.* [12]. However, as yet nobody has provided conclusive proof that calf-perforating veins as a whole system enable exclusively unidirectional inward flow from superficial veins into deep veins.

Importantly, there are many venous channels connecting the deep and superficial veins below the knee, and these communicating veins form a complex system. Therefore, examining a single calf perforator by means of Doppler ultrasonography does not seem to be an appropriate diagnostic tool to evaluate the competence of the whole system. On the contrary, simultaneous invasive pressure measurements in the PTV and GSV are much more reliable in resolving this controversial issue. Höjensgard and Stürup [13] performed such a study in healthy people and demonstrated that pressure curves in both veins were nearly identical, with an increase of pressure during each muscle contraction and decrease during muscle relaxation. This phenomenon has confirmed that blood could flow freely in both directions through calf perforators, thus equalising the pressure in such conjoined blood vessels. The same situation exists in varicose vein patients with clear ICPs. These findings published by Höjensgard and Stürup demonstrate that either perforating veins in healthy people are valveless, or that such valves are functionally incompetent. If the calf perforators as a whole system were equipped with competent valves, transmission of pressure from the deep to superficial veins would not be possible. Interestingly, in the same experiment during one of the muscle contractions, the pressure increased in the PTV but did not change in the GSV, which was a pattern of a competent valve. Irrespective of the actual background of this single observation, during this, but not other muscle contractions, calf perforators behaved as competent. A similar situation occurs in the heart across the competent mitral valve: whereas the systolic pressure in the left ventricle rises to about 120 mmHg, there is no pressure increase in the left atrium, where the pressure remains at the level of 10 mmHg. On the other hand, regurgitation of blood from the left ventricle into the left atrium through an incompetent mitral valve is accompanied by increased pressure in the left atrium. In a similar manner, an increase of pressure in the GSV during muscle contraction is caused by inflow of blood from the deep into the superficial veins through calf perforators. Thus, an unrestricted transmission of pressure and flow in both directions within the calf perforators occurs not only in varicose vein patients but also in healthy individuals. This bidirectional flow through calf perforators enables quick pressure equalisation and makes the deep and superficial veins of the lower leg into conjoined vessels.

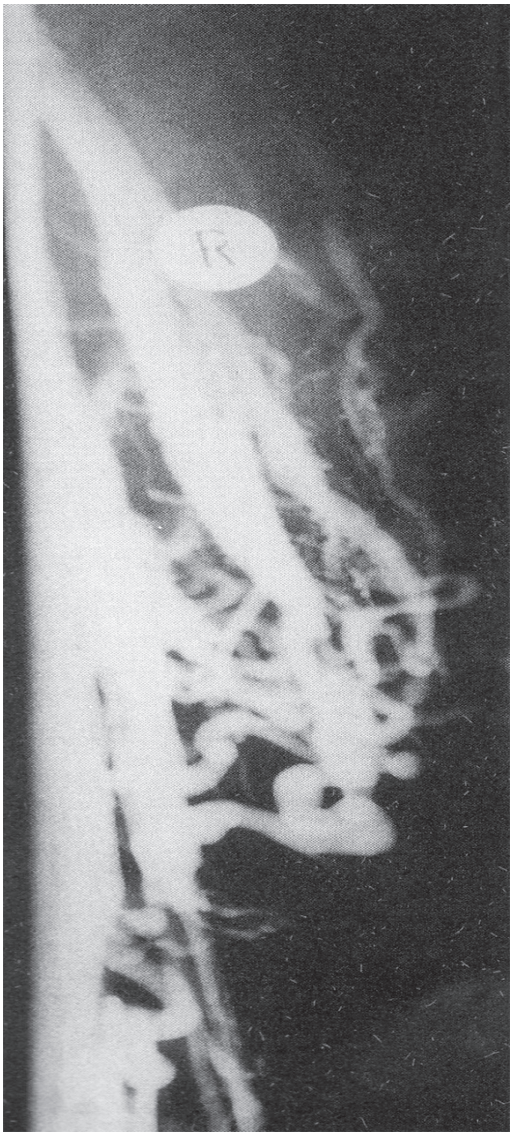

Fig. 1. Phlebography displays a dilated great saphenous vein, enlarged calf perforators, and enlarged posterior tibial vein, forming the so-called Trendelenburg's "private circulation". The enlargement of these venous segments was induced by huge saphenous reflux. Note the presence of a normally sized pair of posterior tibial veins below enlarged calf perforators, as well as thin calf perforators with apparently outward flow

The saphenofemoral junction (SFJ) and the saphenopopliteal junction (SPJ) can also be regarded as "perforators" connecting superficial with the deep veins, similarly to numerous communicating veins below the knee. Still, from the haemodynamic point of view there

is a substantial difference between the perforators located above and below the knee. Thigh perforators (such as the SFJ, the SPJ, and mid-thigh perforators) are connected to the higher pole of the ambulatory pressure gradient [14]. When incompetent, they enable pathological centrifugal flow resulting in ambulatory venous hypertension. However, calf perforators are connected to the lower pole of the ambulatory pressure gradient. Therefore, they cannot become a source of centrifugal (refluxing) flow [15]. As previously mentioned, an outward flow in calf perforators should not be interpreted as reflux.

Calf muscle pump can be considered an auxiliary peripheral heart enhancing return of venous blood to the heart. There are two outflow routes from the leg: the main one through the deep venous system and an accessory route through the GSV. Both routes join in the common femoral vein.

Competent valves located in the deep veins of the lower leg and in the saphenous system (they resemble an 'aortic' valve of the peripheral heart) preclude centrifugal (backward) flow during calf muscle relaxation and enable a decrease in pressure in the veins below the knee from about 90 mmHg (equivalent to the hydrostatic pressure during quiet standing) to about 25 mmHg. Nonetheless, competent valves in calf perforators (they can be compared to a 'mitral' valve of the peripheral heart) are not indispensable. An outward flow within calf perforators is not a pathological 'regurgitation' as in the case of incompetence of the mitral valve in the heart, since such a flow continues via the GSV in the physiological centripetal direction toward the heart, without causing any haemodynamic harm to the superficial veins. In addition, in a case of thrombotic occlusion of the femoral and/or popliteal veins competent valves in calf perforators would compromise venous return from the lower leg. Nevertheless, these perforators enable bidirectional flow, thus protecting the leg from severe venous congestion in the setting of deep venous thrombosis.

CONCLUSIONS

The opinion that an outward flow in calf perforators should be regarded as pathological reflux is incorrect. Actually, an inward flow through these perforators should be interpreted as pathological reflux inducing ambulatory venous hypertension. A systolic outward flow though calf perforators enables a centripetal outflow toward the heart via the GSV, which is propelled by the calf muscle pump and is utterly harmless. Also, it does not interfere with the physiological decrease in venous pressure or cause ambulatory venous hypertension.

Calf perforators enable free pressure transmission between the deep and superficial veins, and enable flow in both directions, which results in quick pressure equalisation and makes deep and superficial veins of the lower leg into conjoined vessels – both in healthy people and in the setting of venous disease. Considering these facts, calf perforators, even if enlarged, are rather innocent structures, and in most of the cases their ligation seems to be a baseless procedure, carrying at least an unnecessary harm (not to mention potential complications that may occur during such a procedure).

References

1. Bjordal R.I. Circulation patterns in incompetent perforating veins in the calf and the saphenous system in primary varicose veins. Acta Chir Scand 1972; 138: 251-261.
2. Recek C., Koudelka V. Effect circulatoire du reflux saphénien dans la varicose essentielle. Phlebologie 1979; 32: 407-414.
3. Cockett F.B., Jones D.E. The ankle blow-out syndrome. A new approach to the varicose ulcer problem. Lancet 1953; 1: 17-23.
4. Recek C. A critical appraisal of the role of ankle perforators for the genesis of venous ulcers in the lower leg. J Cardiovasc Surg (Torino) 1971; 12: 45-49.
5. Recek C., Koudelka V. Perforating veins of the leg and their role in the pathogenesis of varicose veins (Czech). Rozhl Chir 1973; 52: 14-20.
6. Recek C. Saphenous reflux causing venous circulatory disturbance in patients with primary varicose veins and chronic venous insufficiency (German). Acta Chir Austriaca 1998; 30: 76-77.
7. Burnand K.G., O'Donnell Jr. T.F., Thomas M.I., Browse N.L. The relative importance of incompetent communicating veins in the production of varicose veins and venous ulcers. Surgery 1997; 82: 9-13.
8. Akesson H., Brudin L., Cwikiel M., et al. Does the correction of insufficient superficial and perforating veins improve venous function in patients with deep venous insufficiency? Phlebology 1990; 5: 113-123.
9. Scriven J.M., Bianchi V., Hartshorne T., Bell P.R., Naylor A.R., London N.J. A clinical and hemodynamic investigation into the role of calf perforating vein surgery in patients with venous ulceration and deep venous incompetence. Eur J Vasc Endovasc Surg 1998; 16: 148-152.
10. Fitridge R.A., Dunlop C., Raptis S., Thompson M.M., Leppard P., Quigley F. A prospective randomized trial evaluating the hemodynamic role of incompetent calf perforating veins. Aust N Z J Surg 1999; 69: 214-216.
11. Arnoldi C.C. Venous pressure in the legs of healthy human subjects at rest and during muscular exercise in nearly erect position. Acta Chir Scand 1965; 130: 570-583.
12. Sarin S., Scurr J.H., Smith P.D. Medial calf perforators in venous disease: The significance of outward flow. J Vasc Surg 1992; 16: 40-46.
13. Höjensgard I.C., Stürup H. Static and dynamic pressures in superficial and deep veins of the lower extremity in man. Acta Physiol Scand 1952; 27: 49-67.
14. Recek C., Pojer H. Ambulatory pressure gradient in the veins of the lower extremity. Vasa 2000; 29: 187-190.
15. Recek C. The venous reflux. Angiology 2004; 55: 541-548.