$\bigcirc SciMedCentral$ 

#### **Mini Review**

# The Hemodynamic Characteristic of the Popliteal Vein

# **Cestmir Recek\***

Department of Surgery, University Hospital Hradec Kralove, Czech Republic, Austria

#### Abstract

The hemodynamic characteristic of the popliteal vein is assessed. The hydrostatic pressure in the popliteal vein amounts to about 60 mm Hg at the knee level. Pressure changes are produced by the calf pump activity; they are much mitigated and short-lived in the popliteal vein, which contrasts distinctly with the situation in deep lower leg veins, where the pressure excursions are pronounced. The systolic pressure in the popliteal vein increases by about 25 mm Hg above the hydrostatic pressure during the first calf muscle contraction; the diastolic pressure decreases by about 8 mm Hg below the hydrostatic pressure.

The incompetence of the popliteal vein is hemodynamically not relevant; this statement is in contrast with the prevailing opinion. The state of affairs has been documented by plethysmographic examinations. Small saphenous vein incompetence is regularly accompanied by the incompetence of the femoropopliteal venous axis. Abolition of small saphenous vein reflux eliminates the hemodynamic disorders and restores physiological plethysmographic values in spite of the persistent popliteal vein incompetence. The refluxing flow in the popliteal vein disappears after interruption or abolition of small saphenous vein reflux.

From the functional point of view, the popliteal vein is the outflow pipe for the calf muscle pump, similarly to the aorta, which is the outflow pipe for the left ventricle.

#### **INTRODUCTION**

The hemodynamic significance of the popliteal vein incompetence continues to be a contentious issue. Whereas some authors regard the incompetence of the femoral and popliteal vein hemodynamically unimportant [1-3], many authors claim the opposite and advocate the femoral or popliteal valve repair and valve transplantation [4-12]. The hemodynamic changes occurring in the popliteal vein are predominantly induced by the calf pump activity, which is the motive force of the venous hemodynamics in the lower extremity. In this article, the systolic and diastolic pressures produced in the popliteal vein by the calf pump activity are presented, and the real importance of the popliteal vein incompetence is assessed. In addition, the hemodynamic similarity between the popliteal vein and the aorta is mentioned.

# Pressure changes in the popliteal vein produced by the calf pump activity

**Hydrostatic pressure:** The pressure in the deep and superficial veins of the lower extremity in the quiet standing position is determined by the hydrostatic pressure, i.e. by the distance between the point of the pressure measurement and the right atrium. The hydrostatic pressure is equal in deep and superficial veins at the same hydrostatic level [13-15]. The

# Annals of Vascular Medicine & Research

#### \*Corresponding author

Cestmir Recek, Department of Surgery, University Hospital Hradec Kralove, Czech Republic, Mantlergasse 24A-1130 Vienna, Austria, Email: cestmir@recek.at

Submitted: 25 March 2021

Accepted: 20 April 2021

Published: 21 April 2021

ISSN: 2378-9344

Copyright

© 2021 Recek C

OPEN ACCESS

#### **Keywords**

 Pressure changes in the popliteal vein, popliteal vein incompetence, small saphenous vein incompetence, calf pump activity, venous pressure.

hydrostatic pressure in the popliteal vein at the level of the knee joint amounts to about 60 mm Hg [15,16].

Systolic and diastolic pressure changes induced by the calf pump activity: According to the venous pressure measurements performed by Arnoldi, calf muscle contraction increases the systolic pressure in the popliteal vein by about 25 mm Hg above the level of the hydrostatic pressure; with the continuing calf pump activity the increase in systolic pressure diminishes [14]. For comparison: the systolic pressure in the posterior tibial vein increases by about 75 mm Hg over the hydrostatic pressure [14,15]. If the valve at the saphenopopliteal junction is incompetent, the increased systolic pressure in the popliteal vein causes evasion of blood from the popliteal vein into the small saphenous vein; the blood streams further via the Giacomini vein and the great saphenous vein in the centripetal direction toward the heart. In this way, an additional pathway for the systolic outflow toward the heart is formed. Even if the small saphenous vein itself is incompetent, the systolic flow cannot continue in the retrograde direction via small saphenous vein toward the deep lower leg veins, because the systolic pressure in these veins is much higher than in the popliteal vein; as mentioned above, it increases here by about 75 mm Hg. Thus, the systolic evasion of venous blood from the popliteal vein into the small saphenous vein cannot be labeled reflux; it is in reality a harmless orthograde/centripetal flow, which streams in

Cite this article: Recek C (2021) The Hemodynamic Characteristic of the Popliteal Vein. Ann Vasc Med Res 8(2): 1128.

## **⊘**SciMedCentral

the physiological direction toward the heart. In contrast to that, reflux is a pathological flow occurring in the diastolic phase of the calf pump activity; it streams in the retrograde/centrifugal direction into the deep veins of the lower leg, where the lower pole of the ambulatory pressure gradient is situated.

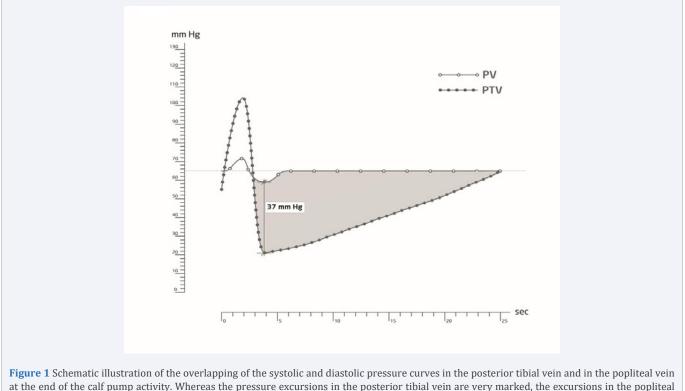
The diastolic pressure in the popliteal vein decreases only slightly by about 8 mm Hg and returns quickly to the resting value of the hydrostatic pressure [13-15]; for comparison, the decrease in diastolic pressure in the posterior tibial vein amounts to about 50 mm Hg and lasts more longer. The statistically evaluated difference in the diastolic pressure between the posterior tibial vein and the popliteal vein yielded the value of 33 + -11.8 mm, as documented by simultaneous pressure measurements in these veins [15]. The pressure difference between the femoropopliteal venous axis and the deep lower leg veins has been later termed ambulatory pressure gradient; its value is 37.4 + -6.4 mm Hg [16]. The schematic illustration of the overlapping of the pressure curves in the popliteal vein and the posterior tibial vein is shown in (Figure 1).

The hemodynamic consequence of popliteal vein incompetence: The significance of the competence and incompetence of the popliteal and femoral veins continues to be a contentious issue. The incompetence of the femoral and popliteal vein is mostly regarded as the main cause of pronounced venous disorders. Therefore, repair of femoral or popliteal vein incompetence has been performed, as mentioned in the introduction. However, these procedures either did not improve the hemodynamic conditions or brought only a slight and transitory improvement of several hemodynamic parameters [7-9]. From the hemodynamic point of view, in varicose vein disease the decisive factor is the competence or incompetence of deep lower leg veins, not the competence or incompetence of the femoropopliteal venous axis.

Superficial femoral and popliteal vein incompetence has been concurrently found in patients with small saphenous vein incompetence [3,17]. Similar situation exists in patients with great saphenous vein incompetence: the deep veins above the saphenofemoral junction (common femoral vein, iliac veins) are incompetent [18,19]. Trendelenburg stated as early as near the end of the 19<sup>th</sup> century that there was no competent valve between the varicose veins and the right atrium; in recumbent patients, the elevation and lowering of the limb emptied and filled quickly the varicose veins [18].

In fact, incompetence of the femoral and popliteal vein might be regarded as the prerequisite for the development of the small saphenous vein incompetence. Competent valves in the superficial femoral and popliteal vein impede the transmission of the increased intra-abdominal pressure to the saphenopopliteal junction and protect in this way the small saphenous vein from the untoward dilatation effect. Hereby it can be explained why the incompetence of the small saphenous vein occurs multiple times more rarely than the great saphenous vein incompetence, although the hydrostatic pressure at the saphenopopliteal junction is higher than at the saphenofemoral junction.

Thus, in patients with small saphenous vein incompetence, the deep veins above the saphenopopliteal junction are incompetent; the centrifugal/retrograde flow in the popliteal vein occurs



at the end of the calf pump activity. Whereas the pressure excursions in the posterior tibial vein are very marked, the excursions in the popliteal vein are minimal. The darker area showcases the ambulatory pressure gradient, which arises after the end of calf pump activity. PV = popliteal vein, PTV = posterior tibial vein.

## **⊘**SciMedCentral-

regularly during the diastolic phase of the calf pump activity. When the incompetent small saphenous vein is compressed so that the refluxing flow is eliminated, the retrograde flow in the popliteal vein disappears; it reoccurs immediately as soon as the compression of the incompetent small saphenous vein is loosened [3]. Hence, there is no retrograde flow in the incompetent popliteal vein without reflux in the small saphenous vein, provided that the deep veins of the lower leg are competent, which is the case in varicose veins disease.

Plethysmographic findings documented the hemodynamic insignificance of the popliteal vein incompetence. In patients with small saphenous vein incompetence, strain gauge plethysmographic parameters showed marked hemodynamic disorder before surgery. Abolition of reflux in the small saphenous vein eliminated the hemodynamic disturbance and restored physiological values of all plethysmographic parameters in spite of the persisting popliteal vein incompetence. The differences between preoperative and postoperative values were highly significant (p < 0.001) [3]. The retrograde flow in the popliteal vein was not detectable anymore after the surgical abolition of reflux in the small saphenous vein. These results document that the incompetence of the popliteal vein is not hemodynamically relevant, provided that the deep lower leg veins are competent and no incompetent venous channel connects both poles of the ambulatory pressure gradient.

Similar results documenting the hemodynamic irrelevance of the superficial femoral vein incompetence were presented in patients with combined great saphenous vein and femoral vein incompetence. Selective abolition of reflux in the great saphenous vein eliminated the distinctly disturbed hemodynamic conditions and restored normal plethysmographic values, in spite of the persistent incompetence of the superficial femoral vein. Reflux in the incompetent superficial femoral vein was not detectable anymore after stripping of the great saphenous vein [20-22].

Functional similarity between the popliteal vein and the aorta: The popliteal vein is de facto the outflow pipe of the calf muscle pump, similar to the aorta in relation to the left ventricle. The ambulatory pressure gradient arising during calf pump activity between the veins above and below the knee resembles the diastolic pressure difference between the aorta and the left ventricle; it is just a bit less marked. The venous reflux, which is the consequence of venous valve incompetence, has a similar impact like the regurgitation through the incompetent aortic valve; both events increase the pressure: the venous reflux in the venous reservoir of the calf musculature, and the "heart reflux", i.e. the flow regurgitating from the aorta increases the pressure in the left ventricle. In addition, there is another similarity between the popliteal vein and the aorta: at the beginning of the diastole, there is a retrograde flow in both these structures. In healthy people, the retrograde flow in the femoropopliteal venous axis lasts about 200-300 milliseconds until the competent valves close; thereafter the diastolic flow stops [23]. In the aorta, the retrograde flow continues even after the closure of the aortic valve during the whole diastole because it provides the coronary artery perfusion. So, similar events take place in the heart and in the "peripheral venous heart".

# **CONCLUSION**

Calf pump activity produces pressure changes in the popliteal vein. The systolic pressure increases by about 25 mm Hg, the diastolic pressure decreases by about 8 mm Hg in relation to the hydrostatic pressure. These pressure changes are very short-lived; the pressure in the popliteal vein tends to keep the level of the hydrostatic pressure of about 60 mm Hg.

In patients with small saphenous vein incompetence, the femoropopliteal venous axis is incompetent in most cases, if not in all, which induces retrograde flow in this venous segment during the diastolic phase of the calf pump activity. The retrograde flow in the popliteal vein disappears after interruption of the small saphenous vein reflux. Plethysmographic examinations confirmed the hemodynamic irrelevance of the incompetent popliteal and superficial femoral vein. These findings contradict the prevailing opinion that popliteal vein incompetence is hemodynamically important.

From the hemodynamic point of view, there is certain similarity between the popliteal vein and the aorta. The ambulatory pressure gradient, i.e. the pressure difference, which is produced during calf pump activity between the veins above and below the knee, is similar to the diastolic pressure difference between the aorta and the left ventricle. In the lower extremity, the dividing line of the pressure gradient lies at the beginning of the popliteal vein, in the aorta at the aortic valve. In addition, in the diastolic phase there is a retrograde flow in both vascular segments. In the femoropopliteal venous axis this retrograde flow lasts only 200-300 milliseconds until the competent valves close, whereas in the aorta it continues even after the closure of the aortic valve during the whole diastole, because it supplies the coronary circulation.

### REFERENCES

- Gooley Na, Sumner DS. Relationship of venous reflux to the site of venous valvular incompetence: implications for venous reconstructive. J Vasc Surg. 1988; 7: 50-59.
- 2. Pearce WH, Ricco JB, L A Queral LA, et al. Hemodynamic assessment of venous problems. Surgery. 1983; 93: 715-721.
- 3. Recek C, Hammerschlag A. What hemodynamic significance has the incompetence of the femoral and popliteal vein? (German). Phlebologie. 1998; 27: 15-18.
- 4. Kistner RL. Surgícal repair of the incompetent femoral vein valve. Arch Surg. 1975; 110: 1336-1342.
- Raju S, Fredericks R. Valve reconstruction procedures for nonobstructive venous insufficiency: Rationale, techniques and results in 107 procedures with two-to eight-year follow-up. J Vasc Surg. 1988; 7: 301-309.
- Wilson NM, Rutt DL, Browse NL. Repair and replacement of deep valves in the treatment of venous insufficiency. Br J Surg. 1991; 78: 388-394.
- 7. Taheri SA, Prendergast DR, Lazar E, et al. Vein valve transplantation. Am J Surg. 1985; 150: 201-202.
- Welch HJ, Mclaughlin RL, O'Donnell TF Jr. Femoral vein valvuloplasty: Intraoperative angioscopic evaluation and hemodynamic improvement. J Vasc Surg. 1992; 76: 694-700.
- 9. Jamieson WG, Chinnick B: Clinical results of deep venous valvular

# **⊘**SciMedCentral-

repair for chronic venous insufficiency. Can J Surg. 1997; 40: 294-299.

- 10.Bry JD, Muto PA, O'Donnell TF, et al: The clinical and hemodynamic results after axillary-to-poplíteal vein valve transplantation. J Vasc Surg. 1995; 21: 110-119.
- 11. Eriksson I, Almgren B: Surgical reconstruction of incompetent deep vein valves. Ups J Med Sci. 1998; 93:139-143.
- 12. Johnson ND, Queral LA, Flinn WR, et al: Late objektive assessment of venous valve surgery. Arch Surg. 1981; 116: 1461-1466.
- 13. Höjensgard IC, 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. Arnoldi CC. Venous pressure in the leg of healthy human subjects at rest and during muscular exercise in nearly erect position. Acta Chir Scand. 1965; 130: 573-583.
- 15. Arnoldi CC. Venous pressure in patients with valvular incompetence of the veins of the lower extremity. Acta Chir Scand. 1966; 132:628-645.
- 16. Recek C, Pojer H: Ambulatory pressure gradient in the veins of the lower extremity. Vasa. 2000; 29: 187-190.
- 17. Hauser H, Brunner U. New pathophysiologic and functional viewpoints

about the insufficiency of the vena saphena parva. Preliminary report. Vasa. 1993; 22: 338-341.

- 18. Trendelenburg F. The ligation of the great saphenous vein in varicose vein disease (German). Beitr Klin Chir. 1891; 7: 195-210.
- 19. Ludbrook L, Beale G. Femoral venous valves in relation to varicose veins. Lancet. 1962; 1: 79-81.
- 20.Padberg FT Jr, Pappas PJ, Araki CT, et al. Hemodynamic and clinical improvement after superficial vein ablation in primary combined venous insufficiency with ulceration. J Vasc Surg. 1996; 24: 711-718.
- 21. Ting AC, Cheng SW, WU LL, et al. Changes in venous hemodynamics after superficial vein surgery for mixed superficial and deep venous insufficiency. World J Surg. 2001; 25: 122-125.
- 22.Walsh JC, Bergan JJ, Beeman S, Comer TP. Femoral venous reflux abolished by greater saphenous vein stripping. Ann Vasc Surg. 1994; 8: 566-570.
- 23.Jeanneret C, Labs KH, Aschwanden M, et al. Physiological reflux and venous diameter change in the proximal lower limb veins during a standardised Valsalva manoeuvre. Eur J Vasc Endovasc Surg. 1999; 17: 398-403.

#### **Cite this article**

Recek C (2021) The Hemodynamic Characteristic of the Popliteal Vein. Ann Vasc Med Res 8(2): 1128.