

Effect of Posture on Popliteal Artery Hemodynamics

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Hypothesis: Marked peripheral vasodilation and rubor characterize critically ischemic limbs on dependency. We believe that intermittent claudication is also associated with peripheral hemodynamic changes on postural alteration, which differ distinctly from normal. Evaluation of such differences and understanding of the underlying physiological derangements may be essential in the development of treatments for intermittent claudication. We comparatively assess the effect of posture on lower limb arterial hemodynamics in normal subjects and in patients with intermittent claudication (or Fontaine II) due to peripheral vascular disease, determined in the popliteal artery.

Design: A cohort study.

Setting: A university-associated tertiary care hospital.

Patients: Thirty-seven legs of 29 normal subjects (group A) and 50 legs of 36 patients with intermittent claudication (ankle-brachial index range, 0.39-0.76; median, 0.57) (group B).

Interventions: Popliteal artery volume flow (vFl), mean velocity, and luminal diameter were measured on (1) recumbency, (2) sitting, and (3) return to recumbency in groups A and B using color duplex imaging.

Main Outcome Measures: The pulsatility index, peak systolic velocity, and end diastolic velocity (EDV) were measured on (1) recumbency, (2) sitting, and (3) return to recumbency.

Results: Popliteal artery vFl in normal subjects decreased from 110 ± 43 mL/min on recumbency to 57 ± 27 mL/min on sitting ($P < .001$) and returned to 111 ± 46 mL/min on resumption of recumbency ($P < .001$). Similarly, in patients with intermittent claudication, vFl decreased from 113 ± 52 mL/min on recumbency to 76 ± 41 mL/min on sitting ($P < .001$) and increased on resumption of recumbency to 114 ± 53 mL/min ($P < .001$). There was no difference ($P = .97$) in the vFl between the study groups on recumbency, but sitting vFl in normal subjects was significantly lower than in patients with intermittent claudication ($P = .04$). The mean velocity, peak systolic velocity, and EDV displayed a similar pattern of change as vFl. The pulsatility index in both groups increased significantly on sitting ($P < .001$) and decreased on return to recumbency ($P < .001$). All data are given as mean \pm SD.

Conclusions: Lower limb arterial vFl, mean velocity, peak systolic velocity, and EDV decrease significantly ($P < .001$) when posture is altered from recumbency to sitting, in normal subjects and in patients with intermittent claudication. A decrease in the EDV and an increase in the pulsatility index on sitting indicate enhancement of arterial resistance to flow secondary to peripheral vasoconstriction. Quantitative differences between the groups in vFl ($P < .04$), EDV ($P < .01$), and pulsatility index ($P < .001$) on dependency indicate that the orthostatic vasoactive response in patients with intermittent claudication is significantly subdued, reflecting a marked derangement in venoarteriolar response.

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THE CLINICAL observation that patients with critical limb ischemia tend to find relief in the sitting position and the need to identify conditions that might affect the already impaired tissue perfusion have stimulated research into the effect of postural alteration on lower limb arterial flow. Arteriovenous oxygen difference,¹ venous occlusion plethysmography,^{2,3} xenon clearance,⁴⁻¹¹ and duplex ultrasonographic flowmetry¹² are among the methods that have been used by different investigators.

It is maintained that blood flow in the lower extremities of normal subjects or patients with peripheral vascular disease

(PVD) without critical limb ischemia decreases as body posture alters from recumbent to erect (and vice versa).^{2,4,5,7,8,12-15} This (venoarteriolar) response has been ascribed to the venoarteriolar reflex,^{8,9,13,16} a local sympathetic reflex that causes the precapillary resistance to increase in response to elevation of venous pressure, thus decreasing capillary flow.^{17,18} Whatever the mechanism of action, it is believed to function almost normally in patients with claudication but to be defective in patients with rest pain.^{9,12}

Recently, color flow duplex imaging supported by appropriate software technology has enabled the measurement of arterial volume flow (vFl) in an accu-

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PATIENTS AND METHODS

STUDY GROUPS

The effect of posture on lower limb arterial blood flow was evaluated in 37 legs of 29 normal individuals (group A) (15 women and 14 men) aged 31 to 79 years (median, 63.5 years) and 50 legs of 36 patients with stable intermittent claudication (group B) (20 women and 16 men) aged 47 to 76 years (median, 66 years). Resting ankle-brachial systolic pressure indexes of examined individuals (group A, >1.08 ; group B, range [median], $0.39-0.76$ [0.57]) were obtained by dividing the higher ankle pressure, obtained from either the dorsalis pedis or the posterior tibial artery, by the higher of the 2 brachial artery pressures. The maximal walking distance of these patients with intermittent claudication ranged from 95 to 250 m on a treadmill at 3.8 km/h and an inclination of 10%. Subjects included in group A had an ankle-brachial index of 1.0 or higher after exercise (1-minute treadmill test at 4 km/h and an inclination of 10%). The post-exercise ankle-brachial index in group B was 0.05 to 0.37 (median, 0.19).

All patients had superficial femoral artery occlusion or tandem stenoses and no severe atherosclerotic aortoiliac disease as confirmed by a recent intravenous digital subtraction angiogram (28 legs) or duplex scan (22 legs). All had a history of symptomatic PVD in excess of 2 years (range, 2.1-16 years), and their intermittent claudication had been clinically stable during the past 6 months. Patients with coexisting symptomatic venous disease (clinical, etiologic, anatomic, and pathologic classes 2-6),²² leg ulcers, trauma, edema, or infection, extensive arteriosclerotic popliteal artery disease or occlusion, diabetic peripheral neuropathy, previous aortoiliac or infrainguinal reconstruction, and congestive cardiac failure and those taking vasoactive medication (eg, nifedipine) were excluded.

EXAMINATION AND SCANNING PROTOCOL

Lower limb arterial hemodynamic values were obtained from the popliteal artery using color flow duplex imaging. In both groups, the investigation commenced in the horizontal position after a resting period of 30 minutes, to ensure flow stabilization. Popliteal artery flow measurements were performed with the subjects in the recovery position facing the examiner (K.T.D.). The examined limb was uppermost

and flexed at the knee. Measurements were repeated in the sitting position (after a further 15-minute period) with the legs dependent, knees flexed at 45°, and feet resting on a low stool. Finally, popliteal artery flow was measured again in the horizontal position following a second 15-minute period.

The popliteal artery was imaged with a duplex scanner (Sonos 2500; Hewlett Packard, Palo Alto, Calif) fitted with a 7.5- or 5.5-MHz multifrequency linear array probe. The diameter of the arteries was obtained by longitudinal imaging, placing the tracker ball-guided calipers across the intimal-luminal interphases of the near and far walls. Measurements were repeated 3 times and were then averaged. Spectral analysis of pulsed Doppler signals insonating the entire lumen enabled determination of the mean velocity (mV). The sample volume gate was adjusted to encompass the entire lumen of the vessel, and the angle of insonation was maintained at 60°. The mV was the time average of the mVs of each of the velocity spectra occurring during an interval of 6 cardiac cycles and was calculated on line using a specially developed software package.

Mean vFl was calculated by multiplying the mV by the cross-sectional area and is expressed in milliliters per minute. Additional information obtained through computerized analysis of the profiles of the velocity waveforms included the peak systolic velocity (PSV), end diastolic velocity (EDV), and pulsatility index (PI). Pulsed Doppler spectral waveforms containing *aliasing*, noise due to venous flow or wall motion, were discarded. The mean of 3 different sets of flow estimations was obtained per posture, per subject.

The reproducibility of flow velocities using the previously mentioned method in the popliteal artery has been previously reported²¹; vFl, mV, PSV, and EDV are measured with a coefficient of variation of 3% to 9%, 3% to 10%, 4% to 12%, and 5% to 19%, respectively.

Popliteal artery flow velocity measurements were obtained in a quiet, temperature-controlled room (21°C-23°C) specially prepared for circulation investigations.

Statistical analysis was performed using the nonparametric paired 2 sample for means, the Wilcoxon signed rank test for intragroup paired comparisons, and the Mann-Whitney test for intergroup comparisons (Minitab statistical package; Minitab Inc, State College, Pa). Ninety-five percent confidence intervals of the estimated median difference are provided. Data are expressed as mean \pm 1 SD.

rate^{19,20(pp1027-1032)} and reproducible way.²¹ This study determines the postural effect on lower limb arterial hemodynamics in normal subjects and in patients with intermittent claudication (or Fontaine II), measured in the popliteal artery using color flow duplex imaging.

RESULTS

In normal subjects (group A), the resting popliteal artery vFl decreased from 110 ± 43 mL/min in the horizontal position to 57 ± 27 mL/min in the sitting position ($P < .001$) and increased to 111 ± 46 mL/min ($P < .001$) when the horizontal position was resumed. In patients with intermittent claudication (group B), the resting popliteal artery vFl decreased from 113 ± 52 mL/min in the horizontal position to 76 ± 41 mL/min in the sitting position ($P < .001$)

and increased to 114 ± 53 mL/min ($P < .001$) when the horizontal position was resumed (**Figure 1**).

In both groups, the mV (**Figure 2**), PSV (**Figure 3**), and EDV (**Figure 4**) had a similar pattern of change with postural alteration as that described for popliteal artery vFl, all being higher in the horizontal position and lower in the sitting one ($P < .001$). The PSV in the popliteal artery of normal subjects was always significantly higher than that of patients with arteriopathy ($P < .001$) in the same positions.

Conversely, the PI in normal subjects (group A) increased from 6.0 ± 1.8 in the recumbent position to 8.4 ± 2.7 in the sitting position ($P < .001$) and decreased to 6.2 ± 2.1 ($P < .001$) when the horizontal position was resumed (**Figure 5**). Similarly, the PI in patients with intermittent claudication (group B) increased from 1.9 ± 0.8 in the recumbent position to 2.5 ± 1.3 in the

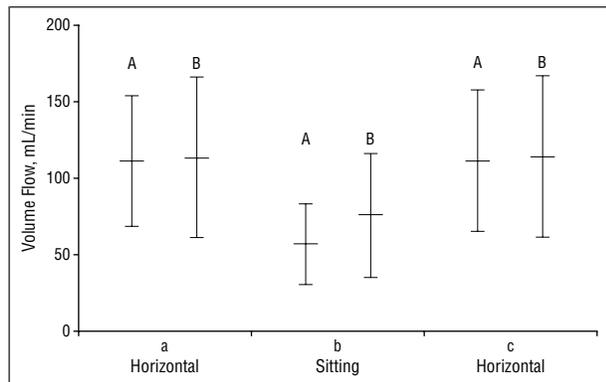


Figure 1. A, Mean \pm SD popliteal artery volume flow in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 37 normal limbs (a vs b: $P < .001$, 95% confidence interval [CI], 41.4 to 63.4 mL/min; a vs c: $P = .84$; b vs c: $P < .001$, 95% CI, -40.2 to -65.3 mL/min; Wilcoxon signed rank test). B, Mean \pm SD popliteal artery volume flow in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 50 limbs of patients with stable intermittent claudication (a vs b: $P < .001$, 95% CI, 30.2 to 42.3 mL/min; a vs c: $P = .84$; b vs c: $P < .001$, 95% CI, -29.1 to -43.5 mL/min; Wilcoxon signed rank test).

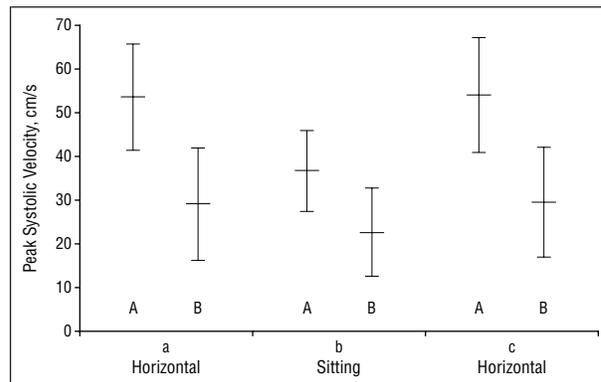


Figure 3. A, Mean \pm SD popliteal artery peak systolic velocity in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 37 normal limbs (a vs b: $P < .001$, 95% confidence interval [CI], 13.80 to 19.57 cm/s; a vs c: $P = .70$; b vs c: $P < .001$, 95% CI, -14.30 to -19.15 cm/s; Wilcoxon signed rank test). B, Mean \pm SD popliteal artery peak systolic velocity in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 50 limbs of patients with stable intermittent claudication (a vs b: $P < .001$, 95% CI, 4.85 to 7.45 cm/s; a vs c: $P = .88$; b vs c: $P < .001$, 95% CI, -4.7 to -7.8 cm/s; Wilcoxon signed rank test).

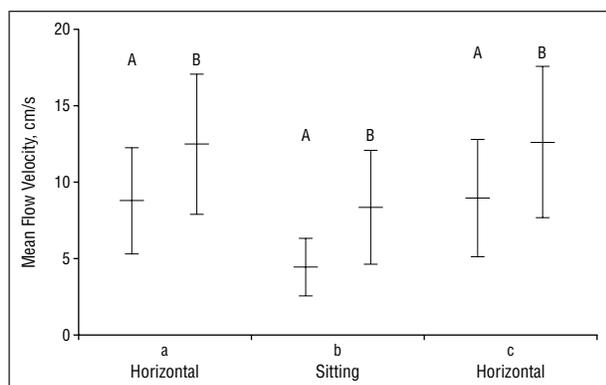


Figure 2. A, Mean \pm SD popliteal artery flow velocity in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 37 normal limbs (a vs b: $P < .001$, 95% confidence interval [CI], 3.15 to 4.70 cm/s; a vs c: $P = .76$; b vs c: $P < .001$, 95% CI, -3.19 to -5.11 cm/s; Wilcoxon signed rank test). B, Mean \pm SD popliteal artery flow velocity in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 50 limbs of patients with stable intermittent claudication (a vs b: $P < .001$, 95% CI, 3.42 to 4.63 cm/s; a vs c: $P = .92$; b vs c: $P < .001$, 95% CI, -3.13 to -4.75 cm/s; Wilcoxon signed rank test).

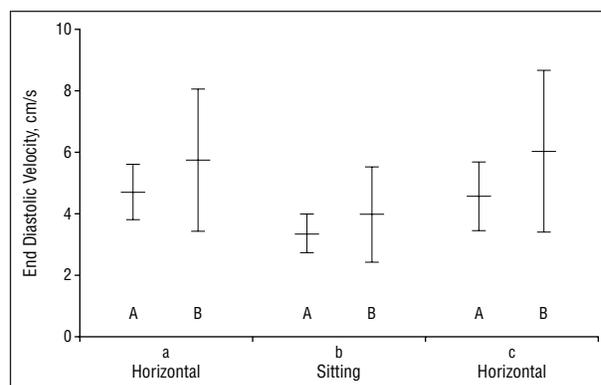


Figure 4. A, Mean \pm SD popliteal artery end diastolic velocity in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 37 normal limbs (a vs b: $P < .001$, 95% confidence interval [CI], 0.98 to 1.66 cm/s; a vs c: $P = .18$; b vs c: $P < .001$, 95% CI, -0.74 to -1.53 cm/s; Wilcoxon signed rank test). B, Mean \pm SD popliteal artery end diastolic velocity in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 50 limbs of patients with stable intermittent claudication (a vs b: $P < .001$, 95% CI, 1.13 to 2.16 cm/s; a vs c: $P = .36$; b vs c: $P < .001$, 95% CI, -1.29 to -2.55 cm/s; Wilcoxon signed rank test).

sitting position ($P < .001$) and decreased to 2.0 ± 1.1 ($P < .001$) on lying down (Figure 5).

The postural effect on the diameter of the popliteal artery was small and insignificant in both groups ($P > .10$). However, in all 3 investigated positions, popliteal artery diameters were significantly smaller in patients with intermittent claudication ($P < .01$).

COMMENT

Early studies,¹ based on the arteriovenous oxygen differential, indicated that lower limb blood flow in healthy subjects lying recumbent was about 15% greater with the legs hanging over the couch than in the horizontal position. Soon, Beaconsfield and Ginsburg² were able to contradict this using plethysmography. They demonstrated that when a normal leg is lowered while the body remains in a constant recumbent position, blood flow in the calf de-

creases. Rendering the lower limb dependent was shown to reduce the rate of blood flow in the toes,¹³ and when normal subjects were tilted from the recumbent position to a 45° foot-down position, flow in the inferior vena cava, just proximal to the confluence of the common iliac veins, was found to decline.¹⁴ Despite some recovery with time, the flow rate remained lower than when the subject was supine. Using isotopes (xenon 133 [¹³³Xe] clearance), Amery et al² were able to demonstrate that muscle blood flow in the tibialis anterior muscle after maximum ischemic exercise decreased in normal and hypertensive subjects on assuming the erect position (sitting or standing).

Gaskel and Becker⁶ were among the first to understand that postural changes influenced patients with severe atherosclerotic occlusive disease in a different way than normal subjects. They demonstrated that on standing the arterial pressure at the ankle of patients with femoral artery occlusion increased more than would have been pre-

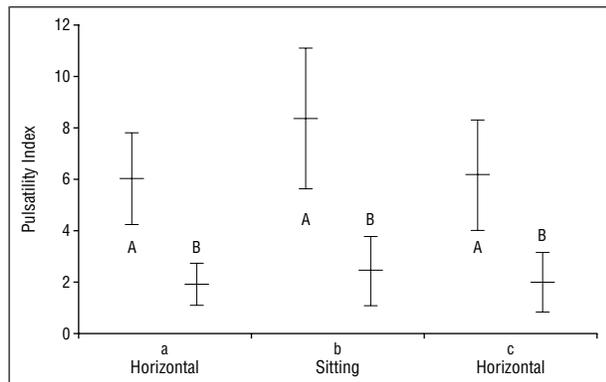


Figure 5. A, Mean \pm SD popliteal artery pulsatility index in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 37 normal limbs (a vs b: $P < .001$, 95% confidence interval [CI], -1.51 to -3.09 ; a vs c: $P = .96$; b vs c: $P < .001$, 95% CI, 1.32 to 2.88 ; Wilcoxon signed rank test). B, Mean \pm SD popliteal artery pulsatility index in the horizontal (a), sitting (b), and on return to the horizontal (c) position in 50 limbs of patients with stable intermittent claudication (a vs b: $P < .001$, 95% CI, -0.25 to -0.56 ; a vs c: $P = .64$; b vs c: $P < .001$, 95% CI, 0.21 to 0.60 ; Wilcoxon signed rank test).

dicted by consideration of the hydrostatic factors and was associated with a substantial (44%) elevation of blood flow in the subdermal tissue of the forefoot. On the contrary, subdermal blood flow in normal volunteers was on average 20% less when sitting than when supine. Two years later, Amery et al,⁵ investigating patients with PVD using ¹³³Xe, showed that muscle blood flow, measured at rest or after ischemia provoked by cuff compression, with or without exercise, was significantly lower in the sitting or standing position than in the recumbent position.

Henriksen,⁸ elaborating on the orthostatic changes of blood flow in the subcutaneous tissue, confirmed that patients with arteriopathy without rest pain displayed a similar vascular response to that seen in normal subjects (decrease of blood flow on dependency and maintenance of flow on elevation). In contrast, in patients with rest pain, he found that lower limb blood flow increased on sitting and decreased on limb elevation, explaining the frequent clinical observation that patients with rest pain due to PVD find relief in the upright position.⁹ He hypothesized that the vessels in ischemic areas might act passively, and as nonischemic areas respond normally, blood tends to divert from nonischemic to ischemic tissues (inverse steal syndrome) on dependency, improving perfusion and relieving rest pain.

Our study has demonstrated that lower limb blood flow in the popliteal artery is markedly affected by posture, being significantly higher in the recumbent than the sitting position in normal individuals and in stable patients with intermittent claudication. Despite their similar pattern of blood flow response, the 2 groups displayed a consistently different level of flow decrease on assuming the sitting position; popliteal artery flow decreased by 49% (from 110 to 57 mL/min) in normal subjects and by 33% (from 113 to 76 mL/min) in patients with intermittent claudication (Figure 1). The pattern of these findings is in agreement with previous investigations.^{2,4,5,7-9,12-15} However, the level of flow decrease found in our study differs from that presented in all previous pertinent publications, such as the 3 following ones. Morgan et al¹² noted that common

femoral artery flow, determined by duplex ultrasound flowmetry, decreased by 17% on dependency (supine with legs hanging down) and by 47% on standing in normal subjects, and by 22% and 24% in patients with intermittent claudication, respectively. Based on clearance rates of ¹³³Xe injected under the skin, Gaskell and Parrott⁷ reported that blood flow decreased by 37.5% (range, 14%-60%) among normal subjects, and increased by $4.60\% \pm 20.84\%$ in patients with arteriopathy with a systolic ankle pressure higher than 60 mm Hg (ankle-brachial index range, 0.39-0.65; median, 0.56), when the posture was altered from supine to sitting. However, our analysis of their data shows that patients with arteriopathy with an ankle-brachial index exceeding 0.55 had a blood flow decrease of 27.5% (range, 16%-34%) on changing to the sitting position. Finally, based on the local ¹³³Xe washout technique and direct (\pm indirect) determinations of lower limb venous and arterial blood pressures, Henriksen and Serjen^{16,23} demonstrated that lowering the legs of normal subjects to a level of 44 cm below the midaxillary line caused blood flow in the subcutaneous adipose tissue over the lateral malleolus to decrease by 52% and total vascular resistance to increase by 136%.

The mechanism underlying the normal vasoactive reaction to postural changes is unknown.^{8,12} It is postulated that this might be due to an autonomic control response mediated via sympathetic outflow into the lower limb.¹² The venoarteriolar reflex and the myogenic hypothesis²⁴ have been given extensive consideration in the literature as possible autoregulatory mechanisms of vasoconstrictor response to vascular transmural pressure elevation.¹⁶

The increase of precapillary resistance to blood flow with subsequent fall of capillary flow, in response to elevation of venous pressure, is called venoarteriolar response.^{17,18} Venous stasis, application of external negative pressure, and lowering of the limb below the level of the heart are conditions capable of eliciting this response.¹⁶ Physiologically, the venoarteriolar response is important in that it minimizes the increase of microvascular pressure when the venous pressure increases (on standing), protecting the capillary bed from the consequences of a raised hydrostatic load.^{18,25,26}

It has been accepted knowledge that the orthostatic peripheral vascular responses in patients with intermittent claudication do not differ from those of normal subjects.^{8,9,12,15} Yet, in the present study, the lower level of popliteal artery flow attenuation seen among patients with arteriopathy changing from the reclining to the sitting position when compared with the normal group ($P = .04$) indicates a derangement of the venoarteriolar reflex in the former.

Our data show that popliteal artery flow in patients with stable intermittent claudication in the horizontal position is not different from that of normal subjects ($P = .97$). This is in accord with data by Lewis et al,²⁷ who reported that the supine common femoral artery vFl in limbs of patients with intermittent claudication (390 mL/min) does not differ significantly from that of normal subjects (344 mL/min). However, as popliteal artery flow in the present study, as in most previous pertinent ones, has not been normalized for limb size, direct intergroup comparisons should be interpreted with caution. The lack of normalization does not affect intragroup comparisons of flow variables, as each limb is its own control.

We were able to demonstrate that the sitting position was invariably associated with significantly lower EDVs and higher PIs in both groups. Considering that the relative diastolic flow velocity changes with outflow resistance, increasing as downstream resistance is lowered,^{20(pp249-268),28} and that the PI varies with the impedance of the receiving circulation, decreasing with peripheral vasodilation, the previously mentioned findings support the presence of a vasoactive response increasing peripheral resistance to flow on dependency. The marked difference in the PI in both examined postures between the 2 study groups could be viewed as a severe derangement of the vasoactive mechanisms controlling outflow impedance in patients with arteriopathy. The PSV was significantly lower in the sitting than in the horizontal position in the normal subjects and in the patients. In view of the increase in peripheral resistance on leg dependency (denoted by the EDV and PI), this was not an unexpected finding. The amplitude of the pulsation at any point in the arterial tree is the summation of forward travelling and reflected waves (dispersion reflection).²⁹ With the increase of vasoconstriction on dependency, more energy is reflected, leading to a decrease in the amplitude of pulsation³⁰ and similarly in the PSV in the popliteal artery. In our study, the significantly lower level of PSV among the patients with arteriopathy, confirming earlier Doppler flowmetry investigations,³¹ should be attributed to the lower distensibility of peripheral vessels in patients with PVD, causing higher pulsatile energy expenditure.³²

Understanding these postural changes is key to the development of therapeutic measures to improve flow and collateral circulation in patients with PVD. It has recently been found that mechanical means, such as intermittent pneumatic compression of the foot, generating a hemodynamic effect similar to that produced by a postural change from sitting to reclining,³³ and reducing peripheral resistance to flow,^{21,34-36} can directly enhance arterial calf inflow and, if used for several consecutive weeks, improve the lower limb hemodynamics and walking ability of patients with stable intermittent claudication.³⁷

In conclusion, popliteal artery vFl decreases significantly when posture alters from the horizontal to the sitting position, in normal subjects and in patients with intermittent claudication. Similarly, the mV, PSV, and EDV are all attenuated on sitting and the PI increases, indicating enhancement of the arterial resistance to flow secondary to peripheral vasoconstriction. The orthostatic vasoactive response in patients with intermittent claudication, however, is significantly attenuated compared with normal subjects, reflecting a marked derangement in the function of the venoarteriolar reflex. Understanding these postural changes is key to the development of therapeutic measures to improve flow and collateral circulation in patients with PVD.

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