Eur J Vasc Endovasc Surg **19**, 270–277 (2000)
doi:10.1053/ejvs.1999.1028, available online at http://www.idealibrary.com on IDEAL®

Effect of Intermittent Pneumatic Foot Compression on Popliteal Artery Haemodynamics

K. T. Delis, N. Labropoulos, A. N. Nicolaides, B. Glenville and G. Stansby

Irvine Laboratory for Cardiovascular Investigation and Research, Academic Vascular Surgery, Imperial College School of Medicine, St Mary's Hospital, Paddington, London, U.K.

Purpose: *the aim was to investigate the effect of intermittent pneumatic foot compression (IPC_{foot}) on popliteal artery haemodynamics in normal individuals and in patients with intermittent claudication due to peripheral vascular disease (PVD) (Fontaine stage II).*
Material and methods: *popliteal artery volume flow [vFl], pulsatility index [PI], mean velocity [mV], peak systolic [PSV] and end diastolic velocity [EDV], in 25 limbs of 20 normal subjects and 40 limbs of 32 stable claudicants were obtained in the sitting position before, during and within 30 seconds after the application of IPC_{foot} (applied pressure: 120 mmHg; inflation time: 3 seconds; deflation time: 17 seconds) using colour-flow duplex imaging (CFDI). The reproducibility of flow velocity estimations using CFDI in the horizontal [hor] (recovery) and sitting [sit] positions was evaluated in 20 limbs of normal controls and 20 limbs of claudicants.*
Results: *popliteal artery vFl, mV, PSV and PI measurements were performed with a coefficient of variation (CV) of less than 14.6% among claudicants and of less than 13.3% in normal subjects. EDV is the least reproducible parameter with an overall CV range of 10.2–21.5% in normal controls and 9.1–18.6% in arteriopaths. On application of IPC_{foot} popliteal artery vFl increased by 111% in the control group ($p<0.001$) and by 51% in the claudicants ($p<0.001$). Within 30 seconds of the cessation of pump action flow decreased significantly in both groups ($p<0.001$), but maintained a significantly higher level than that at baseline ($p<0.001$, in both groups). The mV, PSV and EDV showed a similar pattern of significant changes. Both in normals and claudicants, the PI decreased with IPC_{foot} ($p<0.001$) and increased post-compression; however, it was significantly lower than baseline ($p<0.005$) within 30 seconds of impulse delivery.*
Conclusions: *current CFDI technology enables a reproducible estimation of popliteal artery flow velocities. IPC_{foot} can significantly augment arterial calf inflow on an acute basis both in normals and claudicants. The increase of EDV and decrease of PI indicate that attenuation of peripheral resistance to flow is the main mechanism underlying the popliteal artery vFl enhancement on application of IPC_{foot}. Prospective trials on the long-term effect of IPC_{foot} in the management of patients with PVD are indicated from the results of this study.*

Key Words: Pneumatic compression; Popliteal artery.

Introduction

Appreciation of the physiological role of foot and calf pumps in promoting the return of lower-limb venous blood[1–3] motivated the development of intermittent pneumatic limb compression (IPC) systems, which could activate these pumps artificially. Well documented effective clinical applications include prevention of deep-vein thrombosis (DVT),[4–7] management of leg oedema[8] and postsurgical rehabilitation of patients with leg fractures.[9] IPC systems have also been investigated with respect to their effect on distal arterial flow in patients with peripheral vascular disease (PVD). First studies have shown acute arterial calf inflow enhancement on application of IPC when used on patients with stable intermittent claudication[10–12] or critical ischaemia.[13] If prolonged periods of acutely improved arterial leg inflow, as produced by the frequent use of IPC, could augment distal tissue perfusion long-term, these systems might emerge as invaluable non-invasive therapeutic options in the management of PVD. The practical and cost-saving implications are vast, considering that an estimated 10% of the population over the age of 70 and 1–2% of individuals aged 37–69 are affected by lower extremity claudication secondary to PVD.[14]

The aim of this study was to investigate the direct effect of intermittent pneumatic foot compression (IPC_{foot}) on popliteal artery haemodynamics in normal

* Please address all correspondence to: K. T. Delis, Senior Registrar, Irvine Laboratory, 10th Floor QEQM Wing, Academic Vascular Surgery, Imperial College School of Medicine, St Mary's Hospital, Paddington, Praed Street, London, U.K. W2 1NY.

1078–5884/00/030270+08 $35.00/0

individuals and in patients with intermittent claudication due to PVD (Fontaine stage II) using colour-flow duplex imaging (CFDI).

Material and Methods

Part I

The reproducibility of popliteal artery flow velocity measurements using CFDI was evaluated in 20 limbs of normal individuals and 20 limbs of patients with intermittent claudication, in the horizontal and sitting positions. Patients examined in part I had resting ankle–brachial systolic pressure indices (ABI) ranging from 0.41 to 0.72.

Eighteen estimations (three on six different occasions during the same day) were obtained in each position (horizontal or sitting) from every leg examined. The coefficient of variation (CV) (s.d./mean) was then calculated separately for each parameter, per position, per leg. The higher and lower CV values thus obtained within each study group defined the upper and lower limits of the CV ranges.

Part II

The effect of IPC_{foot} on popliteal artery flow velocities was investigated in 25 limbs of normal volunteers ($n=20$) (Group A), and 40 limbs of stable claudicants ($n=32$) (Group B) using CFDI. Resting ABIs in group B ranged from 0.46 to 0.76. Demographic data for patients in both parts (I and II) are provided in Table 1(a and b).

The ABIs were determined by dividing the higher ankle systolic pressure (obtained from either the dorsalis pedis or the posterior tibial arteries) by the higher of the two brachial artery systolic pressures. A continuous wave Doppler flow detector was used.

For uniformity, only patients with superficial femoral artery (SFA) occlusion or severe disease (tandem stenoses), and aortoiliac segments free of haemodynamically significant disease, as confirmed by a recent (3 months) angiogram ($n=24$) or CFDI ($n=8$), were included in the study. All claudicants had a disease history of 2 or more years. Patients with congestive cardiac failure; leg ulcers, trauma, swelling or pain; chronic venous disease (CEAP classes 2–6),[15] diabetic peripheral neuropathy; a severely atherosclerotic or occluded popliteal artery and those on vasoactive medication (e.g. nifedipine) were excluded.

Table 1a. Demographics of subjects involved in the reproducibility.

		Normal controls		Arteriopaths	
		Sitting	Horizontal	Sitting	Horizon
Males	Subjects	8	9	6	8
	Limbs	8	9	6	8
Females	Subjects	2	1	4	2
	Limbs	2	1	4	2
Age (range in years)		20–67		60–81	
r-ABI (range)		>1.05		0.41–0.72	

Table 1b. Demographics of subjects involved in the study of the effect of IPC_{foot} on popliteal artery flow.

		Normal controls	Arteriopaths
Males	Subjects	16	23
	Limbs	19	27
Females	Subjects	4	9
	Limbs	6	13
Age (range in years)		20–76	60–86
r-ABI (range)		>1.13	0.46–0.76

Also, claudicants with very distal SFA occlusion or stenoses and obvious collateral circulation in the distal thigh bypassing the popliteal artery, noted on angiography or CFDI, and confirmed on cross-sectional colour duplex scanning of the distal thigh and proximal calf, were excluded from the study.

Examination and scanning protocol

A resting period of 15 min was allowed at the beginning of the investigation for flow stabilisation purposes. In part I investigation commenced with the subjects in the recovery position, facing the examiner with the evaluated limb uppermost and slightly flexed. This position provided excellent access to the popliteal fossa. Patients in both parts (I and II) were scanned in the sitting position with the legs dependent and the feet resting on a low stool. IPC_{foot} (part II) was applied only in the sitting position. Two or more flow measurements were obtained from the popliteal artery first without the pump, ipsilateral to the delivery of impulses, then after 10 min of pump action, and finally within 30 s of cessation of pump action.

Popliteal artery scanning was performed with a Hewlett Packard Sonos 2500 scanner fitted with a linear array probe featuring a 7.5/5.5 MHz transducer for B-mode distance measurements (diameter) and a 5 MHz pulsed Doppler for Doppler velocity estimations. All popliteal artery investigations were performed 2–3 cm distal to the medial condyle. The

internal diameter was measured by imaging the vessel longitudinally (real time B-mode) and using the tracker-ball guided callipers. Measurements were repeated three to four times and then were averaged. The mean velocity (mV) was obtained by spectral analysis of pulsed Doppler signals insonating the entire lumen. For this purpose the gate of the sample volume was adjusted to the lumen of the vessel, the sample site was held constant and a 60-degree angle of insonation was strictly maintained. The mV is the time average of the mean velocities of each of the velocity spectra occurring during an interval defined by the operator. In this study these intervals typically included five to seven cardiac cycles, with the starting and ending points always placed at the start of a waveform. A software package, specially developed for arterial volume flow measurement, enabled calculation of mV over the selected time, by tracing the profiles of the corresponding waveforms.

The mean volume flow (vFl) was obtained from the mV multiplied by the cross-sectional area of the popliteal artery and is expressed in ml/min. Data retrieved through computerised analysis of the spectral waveforms included the peak systolic (PSV) and end diastolic (EDV) velocities and the pulsatility index (PI). Pulsed Doppler spectral waveforms with aliasing, excessive noise from the popliteal vein or wall motion were discarded and measurements were repeated.

All popliteal flow measurements were performed in a quiet, draught-free temperature controlled room (21–23 °C).

Statistical analysis of paired comparisons within the same group of subjects was performed using the non-parametric Wilcoxon signed-rank test. For intergroup comparisons the Mann–Whitney test was applied (Minitab). Statistical significances are supported by 95% confidence intervals (95% CI) of the difference. Data are expressed as mean ± one standard deviation (S.D.).

Impulse unit

IPC_{foot} was delivered using the arteriovenous (AV) impulse system of Novamedix. The AV impulse system is a mechanical pneumatic pump, consisting of a pneumatic impulse generator and a plastic inflatable pad, which is applied firmly to the foot by means of a specially developed slipper. The generator is comprised of an electrically driven air compressor and an air reservoir which vents intermittently into the pneumatic foot pad. Investigation thoughout the study was conducted with the pump operating at the following presets: maximum inflation compression 120 mmHg; minimum deflation pressure 0 mmHg; inflation time 3 s and deflation time 17 s.

Table 2. The coefficient of variation of popliteal artery flow velocities in healthy subjects and claudicants.

	Normal controls		Arteriopaths	
	Sitting	Horizontal	Sitting	Horizontal
MV	5.9–11.2	5.8–10.15	2.8–10.3	2.69–6.6
PSV	3.9–13.6	4.1–8.7	3.2–12	3.6–9.4
EDV	10.2–16.5	11.7–21.5	9.1–18.6	4.7–15.4
PI	8.6–11.6	6.1–12.7	4.4–14.6	4.8–10.8
Diameter	1.08–1.99	1–1.8	1–3	1–3
vFl	5.7–10.8	5.7–9.59	2.9–6.6	2.9–6.6

Results

Part I

The range of coefficient of variation (CV) of the vFl, mV, PSV, EDV, PI and diameter in the popliteal artery is depicted in Table 2. Data are provided separately for the horizontal and sitting positions in both study groups.

Part II

On application of IPC_{foot}, mean vFl increased by 111% (from 58.7 to 124 ml/min) ($p<0.001$, 95% CI: 52, 77 ml/min) in Group A and by 51% (from 78 to 118 ml/min) ($p<0.001$, 95% CI: 32, 44 ml/min) in Group B. Within 30 seconds of the cessation of pump action vFl decreased to 86 ml/min in Group A ($p<0.001$) and to 95 ml/min in Group B ($p<0.001$). This level was significantly higher than the baseline reading by 47% ($p<0.001$, 95% CI: 19, 32 ml/min) in Group A and by 22% ($p<0.003$, 95% CI: 12, 20 ml/min) in Group B (Fig. 1).

On application of IPC_{foot}, mV increased by 115% (from 4.88 cm/s to 10.5 cm/s) in Group A ($p<0.001$, 95% CI: 4.6, 6.6 cm/s) and by 49% (from 9 cm/s to 13.4 cm/s) in Group B ($p<0.001$, 95% CI: 3.7, 4.9 cm/s). Within 30 seconds of the cessation of pump action mV decreased to 7.2 cm/s in Group A ($p<0.001$) and to 11.1 cm/s in Group B ($p<0.001$). This level was significantly higher than the baseline, reading by 48% ($p<0.001$, 95% CI: 1.7, 2.9 cm/s) in Group A and by 23% ($p<0.001$, 95% CI: 1.5, 2.6 cm/s) in Group B (Fig. 2).

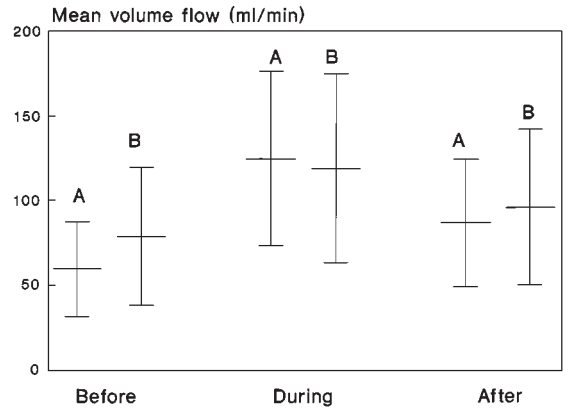

Fig. 1. Mean (±S.D.) popliteal artery vFl (ml/min) in 25 normal limbs (Group A) and 40 limbs with stable intermittent claudication (Group B) (a) before IPC_{foot}, (b) during and (c) within 30 s of cessation of pump action. Group A b vs. a: $p<0.001$ [95% CI of median difference (M.D.) 52–77 ml/min]; c vs. a: $p<0.001$ [95% CI of M.D. 19 to 32 ml/min]; b vs. c: $p<0.001$ [95% CI of M.D. 27 to 46 ml/min]. Group B b vs. a: $p<0.001$ [95% CI of M.D. 32 to 44 ml/min); c vs. a: $p<0.001$ [95% CI of M.D. 12 to 20 ml/min]; b vs. c: $p<0.001$ [95% CI 17–27 ml/min] (Wilcoxon signed-rank test).

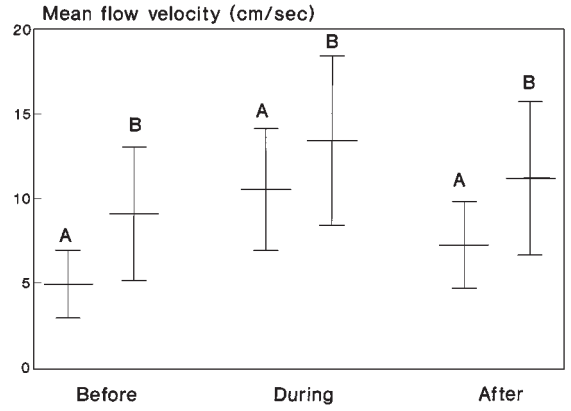

Fig. 2. Mean (±S.D.) popliteal artery flow velocity (cm/s) in 25 normal limbs (Group A) and 40 limbs with stable intermittent claudication (Group B) (a) before IPC_{foot}, (b) during and (c) within 30 s of cessation of pump action. Group A b vs. a $p<0.001$ [95% CI of M.D. 4.6–6.6 cm/s][c vs. a: $p<0.001$ [95% CI of M.D. 1.7–2.9 cm/s]; b vs. c: $p<0.001$ [95% CI of M.D. 2.5–3.9 cm/s]. Group B b vs. a: $p<0.001$ [95% CI of M.D. 3.7–4.9 cm/s]; c vs. a: $p<0.001$ [95% CI 1.5–2.6 cm/s]; b vs. c: $p<0.001$ [95% CI of M.D. 1.8–2.7 cm/s] (Wilcoxon signed-rank test).

Popliteal artery diameter did not change with IPC_{foot} in both groups (Fig. 3). Changes in PSV, EDV and PI during IPC_{foot} and on cessation of pump action are shown in Figs 4–6.

Discussion

Blood flow estimation is feasible using contemporary duplex scanners.[16] The method has been extensively

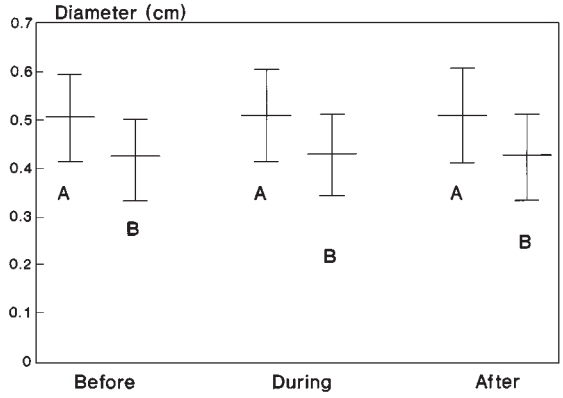

Fig. 3. Mean (±S.D.) popliteal artery diameter (cm) in 25 normal limbs (Group A) and 40 limbs with stable intermittent claudication (Group B) (a) before IPC_{foot}, (b) during and (c) within 30 s of cessation of pump action. Group A b vs. a: $p=0.271$; c vs. a: $p=0.39$; b vs. c: $p=0.459$. Group B b vs. a: $p=0.31$; c vs. a: $p=0.857$; b vs. c: $p=0.21$ (Wilcoxon signed-rank test).

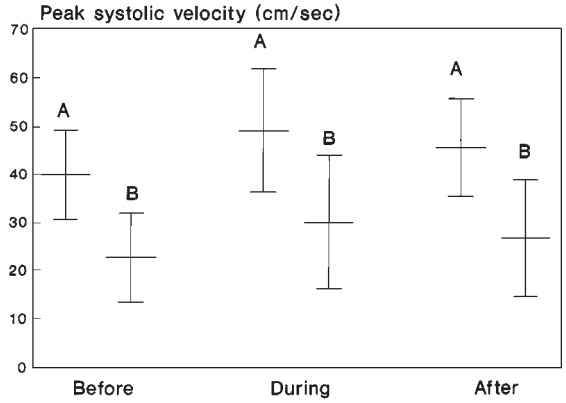

Fig. 4. Mean (±S.D.) popliteal artery PSV (cm/s) in 25 normal limbs (Group A) and 40 limbs with stable intermittent claudication (Group B) (a) before IPC_{foot}, (b) during and (c) within 30 s of cessation of pump action. Group A b vs. a: $p<0.001$ [95% CI of median difference (M.D.) 5.8–11.3 cm/s]; c vs. a: $p<0.001$ [95% CI of M.D. 2.97.7 cm/s]; b vs. c: $p<0.001$ [95% CI of M.D. 1.7–4.4 cm/s]. Group B b vs. a: $p<0.001$ [95% CI of M.D. 5.1–7.3 cm/s]; c vs. a: $p<0.001$ [95% CI of M.D. 1.8–4.7 cm/s]; b vs. c: $p<0.001$ [95% CI of M.D. 2 to 4 cm/s] (Wilcoxon signed-rank test).

validated with respect to cardiac output, both in animal and clinical studies,[17] but knowledge of its accuracy in assessing flow in peripheral vessels is limited. In the absence of a gold standard it is difficult to determine the accuracy of duplex ultrasonography in estimating peripheral arterial blood flow under physiological conditions. Lewis *et al.*[18] in their attempt to validate this method *in vitro* against a calibrated fluorometer demonstrated a volume flow error ranging from 5 to 18 ml/min yielding a correlation coefficient (r) of 0.99 ($p<0.01$). Common femoral artery (CFA) volume flow in resting humans, as estimated by duplex, was also reproduced with a coefficient of variation (CV) of 12%. A mean CFA flow of 350 ± 141 ml/

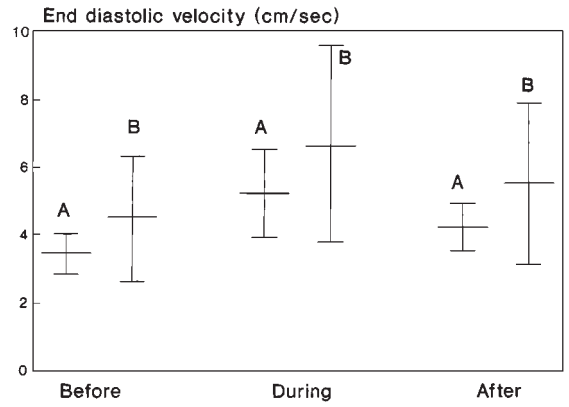

Fig. 5. Mean (±S.D.) popliteal artery EDV (cm/s) in 25 normal limbs (Group A) and 40 limbs with stable intermittent claudication (Group B) (a) before IPC_{foot} (b) during and (c) within 30 s of cessation of pump action. Group A b vs. a: $p<0.001$ [95% CI of median difference 1.2–2.3 cm/s]; c vs. a: $p<0.001$ [95% CI of M.D. 0.487–1.04 cm/s]; b vs. c: $p=0.001$ [95% CI of M.D. 0.51–1.45 cm/s]. Group B b vs. a: $p<0.001$ [95% CI of M.D. 1.5–2.8 cm/s]; c vs. a: $p<0.001$ [95% CI of M.D. 0.5–1.8 cm/s]; b vs. c: $p<0.001$ [95% CI of M.D. 0.8–1.6 cm/s] (Wilcoxon signed-rank test).

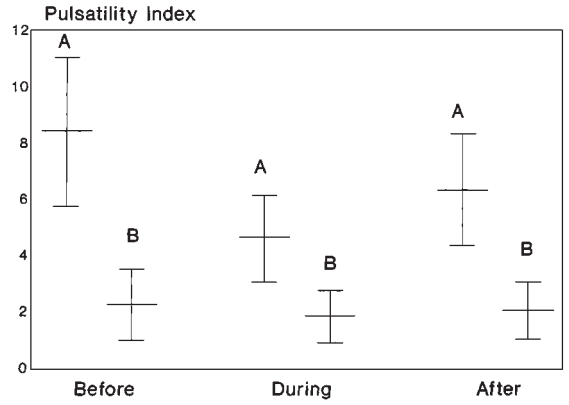

Fig. 6. Mean (±S.D.) popliteal artery PI in 25 normal limbs (Group A) and 40 limbs with stable intermittent claudication (Group B) (a) before IPC_{foot}, (b) during and (c) within 30 s of cessation of pump action. Group A a vs. b: $p<0.001$ [95% CI of median difference (M.D.) 3–4.7]; a vs. c: $p<0.001$ [95% CI 1.3–2.7]; c vs. b: $p<0.001$ [95% CI of M.D. 1.3 to 2]. Group B a vs. b: $p<0.001$ [95% CI of M.D. 0.2–0.5]; a vs. c: $p<0.001$ [95% CI of M.D. 0.06–0.3]; c vs. b: $p<0.001$ [95% CI of M.D. 0.1–0.3] (Wilcoxon signed-rank test).

min was obtained, but the reported broad range (125–819 ml/min) indicates that a number of factors such as the limb size, fat content, age and sex most likely affected lower-limb blood flow and should have been taken into account to allow for a flow normalisation. In another study by Oates *et al.*[19] duplex was shown to reproduce brachial artery blood flow with a CV of 12%. In a pulsatile model, with tubes of different diameter, the mean error of duplex in measuring the actual volume flow ranged from 12 to 31%.[18] Absolute flow estimations from the internal diameter of canine aorta (8.0 mm) and femoral artery (3.7 mm) using duplex ultrasonography were favourably compared ($r_{Aorta}=0.94$, $r_{Fem\ Art}=0.84$) to those obtained with an electromagnetic flow probe.[20]

Zierler *et al.*[21,22] evaluated the accuracy of duplex ultrasound for measuring blood flow in the external iliac artery (baboon model) and reported a high correlation with timed blood collections obtained through a cannula inserted into the common femoral artery (CFA). An absolute percentage error of $13\pm8\%$ was documented, which was mainly attributed to ultrasonographic discrepancies in determining the arterial diameter.

Studies based on popliteal artery flow using duplex have been performed only by three investigators to date[10–12] none of whom addressed the reproducibility of their findings. Our study demonstrates that the latest generation of CFDI, supported with appropriate software packages, can be safely used for the determination of arterial haemodynamics in mid-calibre vessels. The vFl, mV, PSV, PI and diameter measurements can be obtained both in the recumbent and dependent positions with a CV of less than 14.6% among patients with PVD, and of less than 13.6% in normal subjects. Compared to the above, EDV is currently the least reproducible with an overall CV range (in all positions) of 10.2–21.5% in healthy subjects and 9.1–18.6% in those with PVD.

In determining peripheral arterial flow by CFDI, errors are caused primarily by inaccuracy in measuring the angle of insonation.[21,22] Errors ranging from 20 to 60% should be expected when the PSV is measured at an insonation angle of 70 degrees.[23] Overestimation of actual values may be caused by incomplete vessel insonation, use of a high pass filter and attenuation of duplex sensitivity as regards high velocities in the vicinity of the Nyquist limit.[21,22] Throughout our investigation the wall filter was set at minimum and an insonation angle of 60 degrees was meticulously used, as this yields the most accurate velocity measurement.[21,22] Adjustment of the Doppler sample volume to encompass the entire vessel ensured a uniform insonation.

The arterial diameter changes over a cardiac cycle could be viewed as another potential source of error in estimating volume flow. A diameter change of 6.7% for the common carotid artery (CCA) and 2.8% for the CFA have been measured over a cardiac cycle.[24] It might be assumed that the popliteal artery, which is more distal to the CFA and whose walls contain less elastic and more muscular fibres, would display an even smaller diameter variation over the cardiac cycle.

Depth should not be disregarded as a possible cause

of error in volume flow determination, in view of the fact that the quality both of stationary resolution and motion information deteriorates with increasing depth of ultrasonic imaging at a certain frequency, wavelength ultrasound.[25] The mean popliteal artery depth in this series was 3.5 cm (range 2–6 cm). Subjects with the popliteal artery at 5 cm or more would be more likely to be susceptible to depth-related volume flow errors than those with a more superficial vessel location, but the former comprised less than 10% of the total.

This study demonstrates that IPC_{foot} is an effective means of increasing popliteal artery flow both in the limbs of claudicants (51%) and normal individuals (111%). To our knowledge there are two studies published to date[10,12] evaluating the effect of IPC_{foot} on popliteal artery flow using duplex imaging. Morgan *et al.*[10] showed a similar degree of flow augmentation in normal subjects (93%), but a much higher level in arteriopaths (84%). On the other hand, Eze *et al.*[12] reported small levels of flow enhancement with IPC_{foot} both in normals (54%) and in arteriopaths (13%). The discrepancy in published results may be explicable by differences in the composition of patient groups and small sample volumes investigated;[10,12] duplex technology hardware and software;[10,12] pneumatic compression equipment utilised (and thus delivered impulses).[12]

Post-compression flow enhancement has been attributed to augmentation of the arteriovenous pressure gradient,[11,12,26] and decrease of peripheral arterial resistance to flow.[8,10,12] Application of intermittent external compression causes tissue pressure to elevate and underlying veins to empty, until they are refilled by the forward flow of blood from the arteries. As veins empty, venous pressure decreases and the increased arteriovenous pressure gradient results in blood flow elevation.[8,11,12] However, increase of the arteriovenous pressure gradient on intermittent compression cannot solely explain the high level of flow augmentation observed in several individuals in this study. A direct reduction of peripheral resistance has been suggested[10–12] but the mechanism involved is currently unknown. It is postulated that pressure changes in the smaller venous radicles may induce nitric oxide release and its action on the adjacent arteriolar resistance vessels by local diffusion causes them to dilate temporarily with subsequent increase in flow.[8,10–12,27]

Another proposed method by which peripheral resistance may be lowered by IPC_{foot} involves the autoregulatory reflexes. As veins empty, venous pressure decreases to less than 25 mmHg and remains so for most of the deflation time (17 s).[26] Under these conditions, it could be assumed that the venoarteriolar and myogenic reflex is suspended with a subsequent decrease of peripheral resistance.[28,29] The presence of a venoarteriolar reflex inducing arteriolar vasoconstriction as a result of an increase in venous pressure has been documented.[30] However, at low venous pressure precapillary sphincters do not constrict, possibly because the venoarteriolar reflex is not stimulated.[31]

Comparative analysis of data from all groups showed that the mV, PSV and EDV increased significantly during foot pump activation. In spite of their decrease soon after cessation of pump action, they were all maintained at a higher level than the precompression baseline, for a period of 30 seconds. In view of the facts that relative diastolic flow velocity changes with outflow resistance (for a given pressure waveform at the entrance of an arterial segment) and that time–velocity waveforms with higher diastolic run-off accompany a lower down-stream resistance and *vice versa*,[32,33] elevation of EDV on application of IPC_{foot} should be viewed as indicative of a corresponding decrease in peripheral resistance. This is also supported by corresponding changes in the PI, which decreased on foot pump activation and recovered on its cessation. For a certain pressure waveform at the entrance of an arterial segment, the PI varies with the impedance of the receiving circulation, increasing during peripheral vasoconstriction and decreasing during peripheral vasodilation.[34]

The diameter of popliteal artery did not change during the compression phase of the foot pump either among the control individuals or the arteriopaths. This could partially be attributed to resolution limitations imposed by the transducer (7.5/5.5 MHz: 0.2 mm) and the mandatory use of B-mode throughout the process of flow determination. The M-mode would probably have enabled a more pedantic approach, but its application in the context of these measurements was not feasible. Considering that the vessel diameter would tend to increase rather than decrease with elevation of the mean, peak systolic and end diastolic velocities generated with IPC_{foot}, similar to arterial diameter increase documented with peak systolic velocity, our inability to demonstrate it, if it really occurred, would only mean that our flow estimations are not less significant or erroneous compared with the actual ones, but they constitute a conservative estimate of the flow enhancement benefit offered with IPC_{foot}.

Popliteal artery resting flow on dependency was higher (33%) in arteriopaths (78 ml/min) than normal controls (58.9 ml/min). This, and the much lower PI values in arteriopaths, indicate a lower level of

peripheral resistance to flow, suggesting that autoregulatory mechanisms in the latter may have been reset to maximise flow, thereby reducing the margin of flow enhancement with IPC_{foot}. Provided that lower-limb arterial volume flow at rest is not significantly different between normal subjects and patients with peripheral vascular disease, irrespective of the presence of non-critical leg ischaemia or intermittent claudication,[35] flow discrepancies in the sitting position (favouring higher flow in arteriopaths) could be interpreted as a derangement in the venoarteriolar response. This is in agreement with the findings of Morgan[36] who reported that CFA vFl in stable claudicants exhibits a less marked peripheral response on dependency than that of normal individuals. It differs, however, from Henriksen's data[37] demonstrating that the vascular response to orthostatic pressure changes is almost identical in normal individuals and in patients with intermittent claudication.

In this study, like most previous ones examining lower-limb arterial haemodynamics, popliteal artery flow was not normalised for limb size. Inaccuracies introduced by the limitation of this technique should be taken into account when comparisons between small sized groups are attempted. However, these do not affect the comparative results of studies, like the present one, which use each limb as its own control.

In some patients with SFA occlusion or stenoses, most often those with very distal SFA disease, the naturally developed collateral vessels bypassing the luminal block may re-enter the axial vessels of the calf distal to the popliteal artery or may bypass a substantial segment of it. In such disease pattern, flow estimations obtained from the popliteal artery, particularly the most proximal portion of it, may not reflect the actual arterial calf inflow. In order that this should not compromise our data, patients with that disease pattern, identified from the angiograms or CFDI, and confirmed by cross-sectional duplex scanning of the lower thigh and upper calf at rest and on application of IPC_{foot}, were excluded from the study.

In conclusion, the current CFDI technology, supported by specially designed flow software packages, enables a reproducible estimation of popliteal artery and mid-calibre artery haemodynamics. IPC_{foot} can significantly augment arterial calf inflow on an acute basis both in the legs of stable claudicants and normal individuals. Elevation of EDV with a concomitant decrease in PI indicate that attenuation of peripheral resistance to flow is the main mechanism underlying flow augmentation on application of IPC_{foot}. Prospective trials on the long-term effect of intermittent pneumatic limb compression in the management of patients with PVD are indicated from the results of this study.

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Accepted 12 November 1999