Effect of Intermittent Pneumatic Foot Compression on Popliteal Artery Haemodynamics


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Purpose: The aim was to investigate the effect of intermittent pneumatic foot compression (IPCfoot) 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 IPCfoot (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 arteriopathics. On application of IPCfoot, 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 \((p<0.001, \text{in both groups})\). The \( mV \), \( PSV \) and \( EDV \) showed a similar pattern of significant changes. Both in normals and claudicants, the \( PI \) decreased with IPCfoot \((p<0.001)\) 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. IPCfoot 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 IPCfoot. Prospective trials on the long-term effect of IPCfoot 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 (IPCfoot) on popliteal artery haemodynamics in normal
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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 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.

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

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<tr>
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<th>Normal controls</th>
<th>Arteriopathes</th>
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<tr>
<td></td>
<td>Sitting</td>
<td>Horizontal</td>
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<tr>
<td>Males Subjects</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Limbs</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Females Subjects</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Limbs</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Age (range in years)</td>
<td>20–67</td>
<td>60–81</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th></th>
<th>Normal controls</th>
<th>Arteriopathys</th>
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<tbody>
<tr>
<td></td>
<td>Sitting</td>
<td>Horizontal</td>
</tr>
<tr>
<td><strong>MV</strong></td>
<td>5.9–11.2</td>
<td>5.8–10.15</td>
</tr>
<tr>
<td><strong>PSV</strong></td>
<td>3.9–13.6</td>
<td>4.1–8.7</td>
</tr>
<tr>
<td><strong>EDV</strong></td>
<td>10.2–16.5</td>
<td>11.7–21.5</td>
</tr>
<tr>
<td><strong>PI</strong></td>
<td>8.6–11.6</td>
<td>6.1–12.7</td>
</tr>
<tr>
<td><strong>Diameter</strong></td>
<td>1.08–1.99</td>
<td>1–1.8</td>
</tr>
<tr>
<td><strong>vFl</strong></td>
<td>5.7–10.8</td>
<td>5.7–9.59</td>
</tr>
</tbody>
</table>

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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.

**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).
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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 IPCfoot, (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 IPCfoot, (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 of M.D. 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).

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 IPCfoot, (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 IPCfoot, (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. 2.9–7.7 cm/s]; b vs. c: p<0.001 [95% CI of M.D. 1.5–7.3 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).

Popliteal artery diameter did not change with IPCfoot in both groups (Fig. 3). Changes in PSV, EDV and PI during IPCfoot 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 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/
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. 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.\textsuperscript{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\textsubscript{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\textsuperscript{10,12} evaluating the effect of IPC\textsubscript{foot} on popliteal artery flow using duplex imaging. Morgan et al.\textsuperscript{10} showed a similar degree of flow augmentation in normal subjects (93%), but a much higher level in arteriopathies (84%). On the other hand, Eze et al.\textsuperscript{12} reported small levels of flow enhancement with IPC\textsubscript{foot} both in normals (54%) and in arteriopathies (13%). The discrepancy in published results may be explicable by differences in the composition of patient groups and small sample volumes investigated;\textsuperscript{10,12} duplex technology hardware and software\textsuperscript{10,12} pneumatic compression equipment utilised (and thus delivered impulses).\textsuperscript{12}

Post-compression flow enhancement has been attributed to augmentation of the arteriovenous pressure gradient,\textsuperscript{11,12,26} and decrease of peripheral arterial resistance to flow.\textsuperscript{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.\textsuperscript{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\textsuperscript{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.\textsuperscript{8,10–12,27}

Another proposed method by which peripheral resistance may be lowered by IPC\textsubscript{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).\textsuperscript{28} Under these conditions, it could be assumed that the venoarteriolar and myogenic reflex is suspended with a subsequent decrease of peripheral resistance.\textsuperscript{28,29} The presence of a venoarteriolar reflex inducing arteriolar vasconstriction as a result of an increase in venous pressure has been documented.\textsuperscript{30} However, at low venous pressure precapillary sphincters do not constrict, possibly because the venoarteriolar reflex is not stimulated.\textsuperscript{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 pre-compression 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,\textsuperscript{32,33} elevation of EDV on application of IPC\textsubscript{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 vasodilatation.\textsuperscript{34}

The diameter of popliteal artery did not change during the compression phase of the foot pump either among the control individuals or the arteriopathies. 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\textsubscript{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\textsubscript{foot}.

Popliteal artery resting flow on dependency was higher (33%) in arteriopathies (78 ml/min) than normal controls (58.9 ml/min). This, and the much lower PI values in arteriopathies, 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 IPCfoot. 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, flow discrepancies in the sitting position (favouring higher flow in arteriopathies) could be interpreted as a derangement in the venoarteriolar response. This is in agreement with the findings of Morgan 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 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 IPCfoot 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. IPCfoot 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 IPCfoot. 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.

References

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