Intermittent Foot and Calf Compression: Effects on Arterial Blood Flow and Value in the Treatment of Intermittent Claudication

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Twelve percent of the adult population and 20% of people older than 70 suffer peripheral arterial disease. The majority of patients with intermittent claudication who have occlusive lesions involving the superficial femoral or more distal arteries are not suitable for angioplasty. They rely on medications and supervised training programs to modify their pathophysiology in order to mitigate their disability and improve their quality of life.

A new approach has been to increase blood flow in the lower limb by improving collateral circulation using intermittent pneumatic compression of the foot, of the calf or of both.

Gaskel and Parrot\(^1\) were the first to demonstrate in 1978 using \(^{133}\)Xe that intermittent impulses to the foot and ankle could increase blood flow in the limbs of sitting arterio-paths by 80%. It has recently been documented that popliteal artery flow increases by 300% on intermittent pneumatic calf compression\(^2\) and by 52-84% when intermittent pneumatic compression of the foot is applied.\(^3,4\)

Recent experimental work by our team and others has demonstrated that modern duplex scanning equipment and software can measure popliteal artery volume flow with a high reproducibility.\(^5,6\) In the horizontal position the popliteal artery flow is the same in normal individuals (110 ± 43 ml/min) and patients with superficial femoral artery occlusions (113 ± 52 ml/min). This flow decreases in the sitting position by 40 to 50% and returns to the original values when the horizontal position is resumed. A similar pattern is displayed by the peak velocity, end diastolic velocity and mean velocity. The pulsatility index is increased markedly on sitting and decreases on return to recumbency. These changes indicate that there is an increase in peripheral resistance in the sitting position even though the arteriovenous pressure gradient remains the same. (Both arterial and venous pressure increase by the same amount in the sitting position). The increase in the peripheral resistance is the result of the venoarteriolar reflex first demonstrated by Henriksen in 1994. This reflex consists of arteriolar vasoconstriction elicited by an increase in the venous pressure in the lower limbs by 40 mmHg or more. It is a protective mechanism that minimizes capillary perfusion and orthostatic oedema.

We have demonstrated that the application of intermittent foot, calf or foot and calf compression on the limbs of sitting normal individuals and patients with claudication increases popliteal artery flow. There is a 100% increase with foot compression, a 190% increase with calf compression and a 270% increase when both foot and calf compression are applied progressively in normal limbs.
The higher increase in popliteal artery flow with compression of foot and calf and its associated increase in foot skin perfusion has been demonstrated by another team. In the arteriopaths, the corresponding increases in flow are 50%, 130% and 170% respectively. Peak systolic and end-diastolic velocities increase with a corresponding decrease in the pulsatility index. These changes can be explained by a double mechanism: (a) a reduction in the venous pressure (pressure of competent venous valves) producing an increased arteriovenous pressure gradient and (b) abolition of the venoarteriolar reflux.

A randomized controlled study was performed to determine whether the daily use of pneumatic compression delivered simultaneously to the foot and calf (IPFCC) can improve long-term claudication distance and arterial hemodynamics of the leg in patients with intermittent claudication due to PVD (Fontaine II).

Eighty-five patients were investigated in total but only 41 were randomized. (Exclusions: Cardiorespiratory Problems 55%, Orthopedic Problems 18%, Poor Walking Ability 9%, Inconsistent Walking Ability 7%, and Walking Ability Exceeding the Requirements of the Study 11%). Forty-one patients with stable intermittent claudication due to PVD were randomly allotted in two groups to receive:

**Group 1**: IPFCC and salicylic acid (75 mg/day aspirin).

**Group 2 (Control)**: Salicylic acid (75 mg/day aspirin) alone

Both groups were encouraged to exercise.

The two groups were matched for age, sex, claudication distances and arterial hemodynamics (Resting-Ankle Brachial Index (ABI) and Post Exercise-ABI), smoking and diabetes mellitus. Medium duration of symptoms: Group 1: 1.9 yrs., Group 2: 2.1 yrs, with onset of claudication on exercise being clinically unchanged, in terms of walking distance or recovery time, for at least 12 months prior to their inclusion into this trial. The ABI was measured by the higher ankle pressure from dorsalis pedis or posterior tibial arteries divided by the higher of the two brachial artery pressures, with the patient in a horizontal position, using a continuous-wave Doppler.

Group 1 returned their IPFCC pumps at 5 months and followed Group 2 protocol (salicylic acid 75 mg/day aspirin and unsupervised exercise) for 12 more months. Both groups were followed for 5 + 12 (=17) months in order to evaluate the long-term effect of the lower limb impulse treatment.

**Results**

**Initial Claudication Distances (ICD)**

Group 1: Increased from 77.5m to 225m (=2.90 times) after 3 months. (p<.001)
Increased from 225m to 230m (=2.97 times) after 5 months.

Group 2: No significant increase in ICD. (p>.05)

Group 1: 12 months post treatment was not statistically different from that at the end of treatment period.
**Absolute Claudication Distances (ACD)**
Group 1: Increased from 137.5m to 380.5m (=2.76 times) after 3 months.  
Increased from 380.5m to 429m (=3.12 times) after 5 months.

**Resting ABI**
Group 1: Increased from .59 to .69 (=14%) after 3 months.  
Increased from .69 to .69 after 5 months. (Plateau in ~ 2 months).
Group 2: No significant changes.

Group 1: 12 months after trial, r-ABI was unchanged.

**Post Exercise ABI**
Group 1: Increased from .217 to .327 (=51%) after 2 months.  
Increased from .217 to .355 (=64%) after 5 months.
Group 2: No significant changes.

Group 1: 12 months after trial, P-eABI no significant changes.

**Resting Popliteal Artery Volume Flow (vFl)**
Group 1: Increased from 100 ml/min to 136 ml/min (=36%) after 5 months. (Peaked after 2^{nd} and 3rd months).
Group 2: No significant changes.

**Conclusion**
This study clearly demonstrates that IPFCC is an effective treatment in the management of intermittent claudication producing significant improvements both in the walking ability (ICD and ACD) and the arterial hemodynamics in the calf. Follow-up investigation one year after this treatment was completed has shown that the benefits gained are well sustainable.

**References**