ORIGINAL ARTICLE

Acute Effects of Intermittent Pneumatic Compression on Popliteal Artery Blood Flow

Nicos Labropoulos, PhD; William C. Watson, BS; M. Ashraf Mansour, MD; Steven S. Kang, MD; Fred N. Littooy, MD; William H. Baker, MD

Objectives: To investigate the immediate effects of intermittent pneumatic foot and calf compression (IPFCC) on popliteal artery blood flow in symptom-free volunteers and to determine the reproducibility of color flow duplex imaging in the popliteal artery.

Design: Cohort study.

Setting: A university associated tertiary care hospital.

Patients: Forty lower limbs of 30 volunteers without symptoms or noteworthy risk factors of peripheral vascular disease.

Interventions: Popliteal artery blood flow was measured in the sitting position before, during, and after the application of IPFCC using color flow duplex imaging. The interobserver, intraobserver, and between occasion within-subject variability of the popliteal artery blood flow were evaluated in 5 symptom-free volunteers who had at least 5 color flow duplex imaging measurements taken at each of the above time points on 3 different days.

Main Outcome Measures: The arterial diameter, peak systolic, end diastolic, and reverse-flow velocities were measured, as well as the duration of forward flow during diastole before, during, and after IPFCC. The same variables were measured in 5 separate volunteers by 3 different observers, on 3 separate days, at 3 separate times to determine reproducibility.

Results: Including all types of variability, the popliteal artery blood flow varied from 8% to 39% with a mean value of 19%. Since the diameter of the artery was obtained with less than 5% variability, the time average mean velocity was responsible for the high variation in flow. During application of the IPFCC, the popliteal artery blood flow increased significantly in all subjects ($P<.001$). The mean increase in the flow was 2.4 times the baseline values. The diameter of the arteries remained unchanged while the time average velocity increased significantly ($P<.001$). This velocity increase was due to marked elevation in the peak systolic and end diastolic velocities and diminution of the reverse-flow component, as well as a prolongation of the forward flow during diastole. After cessation of the pump, flow returned to baseline levels ($P=.41$)

Conclusions: Ultrasound-derived popliteal artery blood flow measurements show moderate variability. The application of IPFCC greatly enhances popliteal artery blood flow. The flow increase is due to a dramatic drop in the peripheral vascular resistance as the peak systolic and end diastolic flow velocities increase and the reverse-flow component diminishes. Its role in the treatment of lower extremity occlusive arterial disease needs to be determined.

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APPROXIMATELY 10% of the US population older than 70 years is affected by lower extremity claudication secondary to arterial occlusive disease.[1] Traditionally, conservative measures to increase blood flow to relatively ischemic limbs have included exercise and modification of risk factors . However, conservative treatment is not always effective and symptomatic patients may require more aggressive therapy, including percutaneous transluminal angioplasty or bypass grafting. These treatments have proven effective but are themselves associated with several complications that include embolization, infection, thrombosis, dissection, and limb loss.[2]

Intermittent pneumatic compression has been widely used as a method for deep venous thrombosis prevention[3-11] and reduction of both lymphedema[3,12-14] and venous ulcer healing times.[15-18] Its use has been investigated, but not established, for therapy in patients with intermittent claudication secondary to arterial occlusive disease. Foot and/or calf compression has been shown to augment popliteal artery blood flow in both normal volunteers and patients with peripheral arterial disease.[19] While it has been hypothesized that the increase in blood flow is a result of a reduction in the arteriovenous pressure gradient,[8,10-13,15,16] there are no studies to our knowledge to confirm this hypothesis. Furthermore, previous studies have used duplex ultrasonography to determine the effects of intermittent pneumatic compression on popliteal artery blood flow, although the reproducibility

From the Department of Surgery, Loyola University Medical Center, Maywood, Ill.

PATIENTS AND METHODS

Forty limbs from 30 symptom-free volunteers (21 men, 9 women) with a mean (±SD) age of 33±6 years (age range, 21-44 years) with normal body mass indices were studied. All volunteers were free of risk factors, except for 1 man who was a mild smoker.

Initially, all subjects were rested in a sitting position for 10 minutes with their legs supported in a dependent, nonweight-bearing position with the knee flexed at a 60° angle from the horizontal. Inflatable 22- and 12-cm neoprene cuffs were placed on the dorsum of the foot and the muscle mass of the calf, respectively, and connected to a timed-pressure pump (Art Assist AA 1000, ACI Medical Inc, San Marcos, Calif) set at an inflation rate of 120 mm Hg for 3 seconds at 12-second intervals. Inflation of the foot cuff preceded that of the calf cuff by 2 seconds. Previous studies have determined that this timing and rate resulted in the maximal venous return in normal volunteers.[20] After a 10-minute baseline period, in which the popliteal artery blood flow was measured 5 times, IPFCC was applied for 10 minutes. During the IPFCC application, popliteal artery blood flow was measured an additional 5 times. The IPFCC was then switched off, and after 5 minutes of rest 5 final blood flow measurements were taken.

Blood flow measurements were assessed using color flow duplex imaging (HDI 3000, Advanced Technology Laboratories, Bothell, Wash) with a 4- to 7-MHz linear array transducer with software capable of calculating blood flow in milliliters per minute. Peak systolic velocity, reverse-flow velocity, end diastolic velocity, and duration of forward flow during diastole were measured. Time average mean velocity was then calculated as the mean of the mean velocities over 5 cardiac cycles. Assuming the popliteal artery is cylindrical, the blood flow was calculated as the time average mean velocity multiplied by the cross-sectional area of the vessel. All blood flow measurements were measured 1 cm above the popliteal skin crease and insonated at a 60° angle to standardize measurements.

The interobserver, intraobserver, and between occasion within-subject variability was assessed in 5 symptom-free volunteers who had at least 5 popliteal artery blood flow measurements taken on 3 different days. More than 50 data points were collected per volunteer.

of this method in this arterial segment has not been determined.

This study was designed to determine the reproducibility of popliteal artery blood flow by color flow duplex imaging and the acute effects of intermittent pneumatic foot and calf compression (IPFCC) on lower limb arterial hemodynamics in volunteers without symptoms of peripheral vascular disease.

RESULTS

Popliteal artery diameter did not vary significantly in measurements before, during, or after compression ($P=1.0$) (**Figure 1**). Interobserver, intraobserver, between occasion, and within-subject variability of popliteal artery diameter ranged from 2% to 5%. Including all types of variability, time average mean velocity varied from 5% to 36%. Therefore, time average mean was the most influential component for the overall variability of blood flow measurements (range, 8%-39%, mean = 19%). During IPFCC, popliteal artery blood flow increased significantly from baseline ($P<.01$) (**Figure 2**). After the 10-minute compression time, blood flow returned to baseline ($P=.41$) (Figure 2). Peak systolic and end diastolic velocities increased significantly ($P=.006$ and .004, respectively) during application of IPFCC (**Figure 3**, A and B; **Table 1**). The reverse-flow velocity decreased significantly ($P=.002$) or was abolished during that time (Figure 3, A and B) (Table 1). In addition, the duration of forward flow during diastole was significantly prolonged ($P<.001$) (**Table 2**). All the velocity parameters returned to baseline with termination of IPFCC (Figure 3, C).

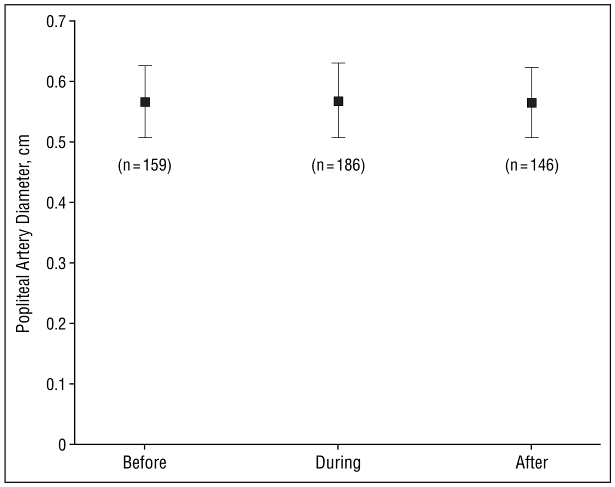

Figure 1. *Popliteal artery diameter (mean±SD) at the popliteal crease, before, during, and after application of intermittent pneumatic foot and calf compression in normal volunteers. P>.2 for all comparisons.*

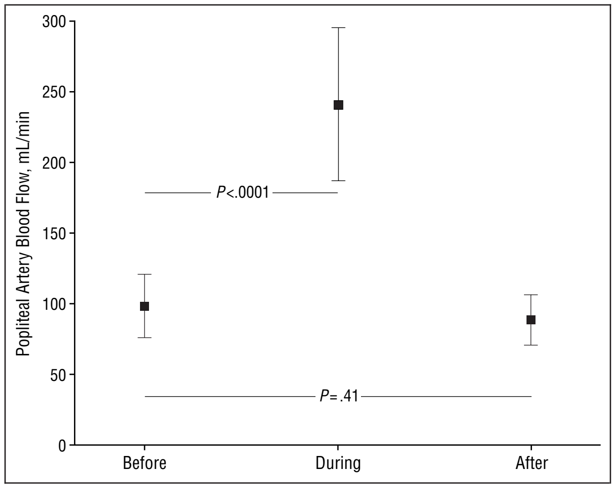

Figure 2. *Popliteal artery blood flow (mean±SD), before, during, and after application of intermittent pneumatic foot and calf compression.*

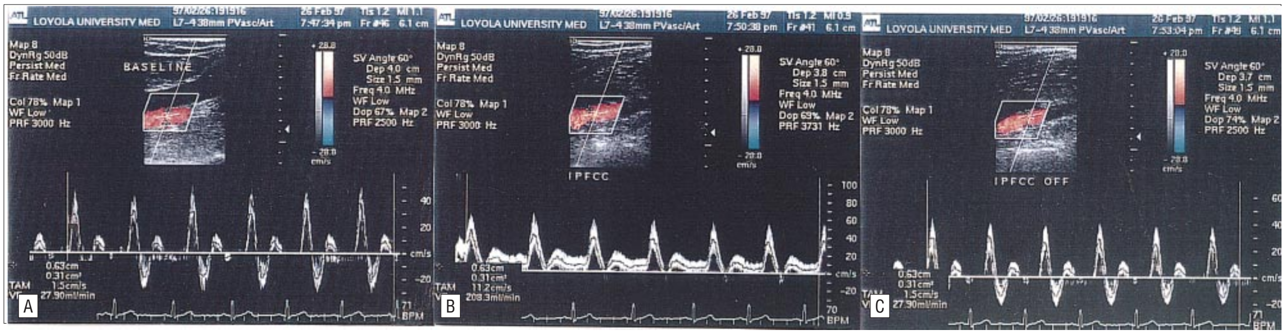

Figure 3. *Color flow duplex imaging of the popliteal artery velocities, before (A), during (B), and after (C) intermittent pneumatic foot and calf compression (IPFCC).*

Table 1. Change in Blood Velocities Within the Popliteal Artery During Intermittent Pneumatic Compression Using Color Flow Duplex Imaging

Velocity,* %Δ	Mean ± SD	Range	*P*
PSV	24 ± 21	9-62	.006
EDV	63 ± 46	4-185	.004
RFV	72 ± 53	8-100	.002

**PSV indicates peak systolic velocity; EDV, end diastolic velocity; and RFV, reverse-flow velocity.*

Table 2. Change in the Duration of End Diastolic Velocity During Application of Intermittent Pneumatic Foot and Calf Compression (IPFCC)*

	Mean ± SD	Range
Rest, ms	190 ± 86	95-260
IPFCC, ms	420 ± 170	160-520

*$P<.001$, *rest vs IPFCC.*

COMMENT

Intermittent pneumatic compression was originally used in the treatment of venous disorders. Gardner and Fox[12] first described the use of foot and ankle compression in the prevention of postsurgical edema. They found that artificial activation of the physiologic venous pump in nonambulatory patients prevented venous stasis and reduced the rate of formation of deep venous thrombosis. Subsequent studies showed that the rate of formation of deep venous thrombosis and postsurgical edema in patients with hip fractures,[3,4,7] knee replacement,[5] foot fracture surgery,[6,14] and laparoscopic cholecystectomies[9] was also dramatically reduced with intermittent foot compression. The compression of the pump, similar to the results of weight-bearing, collapses the venous plexus on the plantar surface in the foot and thereby increases venous return, which would normally be lost in nonambulatory patients.[21]

Further studies have demonstrated that the use of foot or calf compression devices notably increases the rate of venous ulcer healing.[16-18] Activation of the calf muscles, similar to weight-bearing of the foot venous plexus, directs blood toward the central circulation. The venous valves prevent reflux of blood and the result is a response identical to that seen in the foot.

The beneficial effect of intermittent pneumatic compression was first described in 2 reports in 1934[22,23]; however, favorable clinical results were not attributed to increased tissue perfusion because blood flow could not be measured. Using xenon 133 clearance rates, Gaskell and Parrott[24] demonstrated that pneumatic foot and ankle compression increased the blood flow 80% in the limbs of sitting patients with peripheral arterial disease. More recently, Morgan and associates[20] showed that foot compression not only increased venous return but also produced a postcompression arterial hyperemia. Similar results were found in another study in which calf compression was applied.[19] It has also been demonstrated that foot compression increased both trancutaneous oxygen tension and skin perfusion.[25] Combination of foot and calf compression also significantly increased popliteal artery blood flow and skin perfusion.[26] Delis et al[27] demonstrated that by combining foot and calf compression (with a 1-second delay in calf compression) a significantly lower venous pressure could be produced than with foot or calf compression alone. These studies[20-24] suggested that the increased blood flow was partially due to the reduction of peripheral vascular resistance. Since vascular resistance is dependent on arterioles, vasodilation of these vessels may be responsible for the hyperemia.

Our study is one of the few to date applying both intermittent foot and calf compression in normal volunteers, and it is the first to determine observer and temporal variability of the ultrasound-derived popliteal artery blood flow. Color flow duplex imaging showed moderate variability (8%-39%) in the measurement of popliteal artery blood flow. The increase in blood flow (240%) during application of IPFCC in the volunteers was significantly higher than the variation of the measurement, thus indicating that the IPFCC effects were independent of the variation. Similar increases in popliteal blood flow have been reported with IPFCC.[26]

In our study, there were no significant changes in the arterial diameter during IPFCC. Therefore, the marked changes in the time average mean velocity, which is a result of the mean of the mean blood velocities, must be responsible for the increased blood flow. The elevations in the time average mean velocity was due to an increase in the peak systolic and end diastolic velocities, as well as the abolishment of the reverse-flow velocity. These velocity changes, together with the prolonged phase of the end diastolic velocity, suggest a reduction in the vascular resistance of the arterial tree distal to the popliteal artery, supporting previous findings.[20-24]

The increase in the arteriovenous pressure gradient alone cannot explain the significant changes in blood flow seen in our and other recent studies. An additional mechanism for the blood flow increase may be the local release of vasodilatory substances, such as nitric oxide and prostacyclin, from the increased shear stress during IPFCC.

CONCLUSIONS

The role of IPFCC in arterial occlusive disease has yet to be elucidated. We demonstrated that in normal volunteers, without significant risk factors, the application of IPFCC increases popliteal artery blood flow well above the variability of repeated-flow measurements. Because the diameter remains unchanged, the increase in the blood flow can be explained by the considerable elevation in the peak systolic and end diastolic velocities, the reduction of reverse flow, and the extended duration of the forward flow during diastole. Decreased local vascular resistance appears to play an important role in the action of IPFCC in increasing blood flow in normal volunteers and may have an impact in the treatment of arterial occlusive disease.

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Corresponding author: Nicos Labropoulos, PhD, Department of Surgery, Loyola University Medical Center, 2160 S First Ave, Maywood, IL 60153 (e-mail: nlabrop@luc.edu).

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Surgical Anatomy

The blood supply to the adrenal gland originates from three sources to converge on each gland: (a) from the **adrenal artery**, which springs from the aorta; (b) from the **inferior phrenic artery** above; and (c) from the **renal artery** below. A single large adrenal vein leaves the anterior surface of each gland: the right vein ascends to the inferior vena cava while the left vein descends to the left renal vein.

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