Augmentation of blood flow in limbs with occlusive arterial disease by intermittent calf compression

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Abstract

Purpose: This study was designed to investigate the effect of intermittent calf compression on popliteal arterial blood flow and to see how flow is influenced by position of the subject and by arterial blood pressure at the ankle.

Methods: Volume flow in the popliteal artery of subjects in the sitting and prone positions was measured with duplex ultrasonography before inflation and immediately after deflation of a pneumatic cuff placed around the calf. Eleven legs of control subjects and 41 legs of patients with symptoms (32% patients with diabetes) with decreased ankle pressure were studied. Cuffs were inflated for 2 seconds at pressures ranging from 20 to 120 mm Hg.

Results: An increase in arterial blood flow of two to eight times (mean 4.4 ± 2.0) was found on deflation of the cuff in seated control subjects. Little change in flow was observed when the subjects were in the prone position. In seated patients with arterial obstruction, the mean increase in arterial flow was 3.2 ± 1.6 times the resting flow. Little correlation was found between the maximum increase in flow and the ankle/brachial index.

Conclusions: An increased arteriovenous pressure gradient accounts for some but not all of the flow increase, much of which must be attributable to transient vasodilatation. Because the increase in flow does not depend on an increased inflow pressure and was not adversely affected by a low resting ankle-brachial pressure index or a low toe-pressure, intermittent external limb compression may deserve investigation as a possible adjunct to the nonoperative treatment of patients with severe arterial insufficiency. (J VASC SURG 1994;19:1052–8.)

Article Outline

Increasing blood flow in ischemic distal vascular beds is a universal objective of peripheral vascular surgical procedures. This goal, however, may be difficult to achieve in limbs that have been subjected to multiple revascularization procedures, in limbs with graft infections, or whenever the distal arterial bed offers no suitable site for an anastomosis. Although some patients with ischemic foot lesions respond to conservative therapy, [1] the chances for primary healing of ischemic ulcers, localized gangrene, or minor foot amputations are severely compromised in legs with low ankle or toe pressures unless something can be done to improve blood flow. [2], [3]

Little evidence exists that methods aimed at reducing peripheral resistance of the vascular bed are beneficial. Surgical sympathectomy increases total limb perfusion, but flow rates subside rapidly, approaching preoperative values within 1 or 2 weeks. [4] Moreover, experimental studies in animals indicate that most of the increased flow is diverted through arteriovenous communications with little or no improvement in nutritive flow through the capillaries [5] or in tissue oxygen delivery. [6], [7] Likewise, oral vasodilators have been shown to be ineffective and are no longer advocated for treating the manifestations of obstructive arterial disease. [8]

Another approach to the problem of exhausted or absent surgical possibilities may be to improve tissue perfusion by reducing venous pressure below the normal level. When the patient is in a seated or erect position, reduction of venous pressure by external limb compression leads to an increased arteriovenous pressure gradient without requiring any input of increased fluid energy on the arterial side of the circulation, other than that contributed by the hydrostatic effects of gravity. Variations on this approach have been advocated by a number of investigators, but the method has received little attention. [9], [10]

The purpose of this study was to investigate the effect of intermittent calf compression on blood flow in a major inflow artery and to see how flow is influenced by position of the subject and by arterial blood pressure at the ankle. To our knowledge only one previous report has documented changes in arterial inflow in response to intermittent limb compression. [11]

MATERIAL AND METHODS

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Eleven legs of six volunteers without symptoms (four men, two women; mean age 36 ± 4 years) and 41 legs of 38 patients (19 men, 19 women; mean age 69 ± 11 years) with ankle brachial indexes less than 0.85 were studied. An additional patient had undergone a lumbar sympathectomy for causalgia. Clinical manifestations of arterial insufficiency ranged from

claudication ($n = 29$ legs) to rest pain ($n = 7$) and localized tissue loss ($n = 5$). Thirty-two percent of the patients had diabetes. A continuous-wave Doppler flow detector was used to measure the ankle/brachial index (ABI) with the patient in a supine position. ABIs were calculated by division of the higher ankle pressure (obtained from the posterior tibial or dorsalis pedis arteries) by the higher of the two arm pressures. Mean ABI in normal legs was 1.20 ± 0.08. For the groups with claudication and rest pain, the mean ABIs were 0.58 ± 0.16 and 0.41 ± 0.20, respectively. In limbs with tissue loss the mean ABI was 0.51 ± 0.14. Toe pressure was measured in 29 legs by means of a photoplethysmographic transducer placed on the tip of the toe.

Patients were examined in the sitting position with their legs dependent and their heels supported on a low stool. Volume flow in the popliteal artery was evaluated with a Quad 1 color duplex scanner that incorporated software necessary to calculate flow in milliliters per minute (Siemens-Quantum, Issaquah, Wash.). Arterial diameter was measured by electronic calipers, and a sample volume large enough to insonate the entire lumen was used. Recorded flow rates represent average data over four cardiac cycles. In three patients with popliteal occlusion, the peak systolic and diastolic flow velocities in centimeters per second were measured in the largest visible collateral artery in the area. Velocities rather than flow were measured in these vessels to avoid the inaccuracy of volume flow determinations in conduits smaller than 3.5 mm.

A brief (2-second) external compression was applied by rapid inflation of a large cuff (22 cm bladder) placed around the calf. The cuff was connected to an automatic air source (Hokanson, Bellevue, Wash.) that allowed inflation and deflation to be accomplished within 0.3 second. Inflation pressures ranged from 20 to 120 mm Hg and were increased at intervals of about 1 minute by 20 mm Hg increments.

Blood flow in the popliteal artery (or velocity in adjacent collateral arteries) was measured before and after each compression sequence. Special attention was given to maintaining the probe in the same position throughout each study and to ensuring that the same site in the arterial lumen was interrogated at a constant angle of insonation. Because absolute volume blood flow varies depending on limb size and muscle mass, blood flow measured after decompression was divided by the resting flow of the extremity being studied to obtain a volume flow ratio. Use of the volume flow ratio also compensates for errors inherent in measuring absolute blood flow by the duplex method in atherosclerotic arteries.

With the use of the same compression protocol a control experiment was performed on six normal legs, with the subjects lying prone on a table tilted to 20 degrees Trendelenburg position and with their feet slightly elevated to minimize extrinsic pressure on the calf.

This study was approved by the human subjects review board, and each participant signed an informed consent form.

RESULTS

In the popliteal arteries of volunteers without symptoms, both peak-systolic velocities and diastolic forward flow velocities increased markedly after decompression of the cuff (Fig. 1).
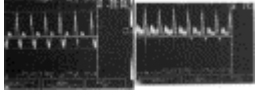

Fig. 1. **A**, Resting flow signal in popliteal artery of seated subject without symptoms. Peak-systolic velocity is 50.5 cm/sec. Early diastolic flow is retrograde with velocity of -23.5 cm/sec. **B**, Flow signal in same artery as in **A**. After brief compression of leg with 100 mm Hg, peak systolic velocity increased to 69.9 cm/sec. Diastolic flow is towards foot with velocity of 17 cm/sec.

Flow remained elevated for about 4 to 14 seconds and then gradually normalized within less than 1 minute. Unlike the flow pattern observed in normal limbs, the resting flow velocity signal in patients with significant arterial obstruction was usually monophasic, with flow confined to the antegrade direction. The effect of compression on the flow-velocity spectrum in a patient with severe arterial obstruction (ABI = 0.30) is shown in Fig. 2.
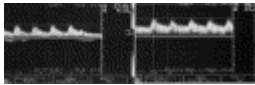

Fig. 2. **A**, Resting flow signal in popliteal artery of seated patient with rest-pain and foot ulcer, with ABI of 0.3 and absolute toe-pressure of 0 mm Hg. Flow signal is monophasic with peak systolic velocity of 27.6 cm/sec. End-diastolic velocity is 8 cm/sec. **B**, Flow signal in same artery as in **A**, after brief compression of leg with 120 mm Hg external pressure in sitting position. Peak-systolic velocity increased to 39.9 cm/sec, and end-diastolic velocity increased three times to 24 cm/sec.

An increase was seen in both peak systolic and end-diastolic flow velocity. Because the increase in end-diastolic velocity was larger than the increase in peak-systolic velocity, flow after compression became more steady and less pulsatile.

Without exception, all control subjects experienced an increase in popliteal arterial blood flow. The mean ratio of the highest attainable postcompression flow to precompression flow in normal legs was 4.41 ± 2.0, with a range of 2.0 to 8.0. Fig. 3 correlates the postcompression/precompression (resting) flow ratios in seated subjects without symptoms with the various cuff pressures used.
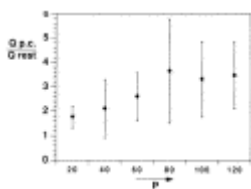
Fig. 3. Graph depicting ratio of mean postcompression (Qpc) to precompression (resting) blood flow (Qrest) versus cuff pressure (P in mm Hg) during compression in 11 legs of subjects without symptoms.

Although, as is evident from the large standard deviations, a great deal of variability was seen in the individual results, the ratios tended to increase with increasing cuff pressures until a pressure of 80 mm Hg was reached, at which point a plateau appeared to have been attained.

Six of the control subjects underwent the same compression protocol in a prone position, which resulted in a substantially lower maximal increase in flow of 1.30 ± 0.36 times resting flow and an average flow ratio of only 1.05 ± 0.52 at a cuff pressure of 60 mm Hg and 0.90 ± 0.42 at 80 mm Hg.

Again, without exception, compression increased popliteal arterial flow in all 38 patients with decreased ABIs. The mean ratio of the highest attainable postcompression flow to resting flow in seated patients was 3.17 ± 1.59, with a range of 1.1 to 8.0. Although the mean ratios of postcompression volume flow to resting flow increased with cuff pressure until a pressure of 100 mm Hg was reached, little increase occurred beyond 60 mm Hg (Fig. 4).
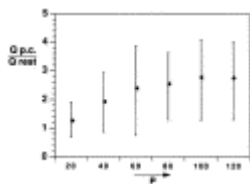

Fig. 4. Graph depicting ratio of mean postcompression (Qpc) to precompression (resting) blood flow (Qrest) versus cuff pressure (P in mm Hg) during compression in 38 legs with decreased ankle pressures.

In three legs of patients with pain on elevation of the leg and with ABIs of 0.0, 0.32, and 0.44, it was not possible to obtain flow signals in the popliteal artery. In these cases flow velocity was measured in the largest visible collateral vessel at the level of the knee. The maximal increase in peak systolic flow velocity in these collaterals was 1.8, 1.5, and 1.4 times that measured at rest, respectively. The maximal increase in end-diastolic velocity was 2.5, 2.9, and 3.8 times that recorded at rest.

One patient with an ABI of 1.10 who had undergone a sympathectomy for causalgia was also studied. A maximum increase in popliteal flow of 4.3 times the resting value was observed at a compression pressure of 80 mm Hg.

ABIs varied from 1.10 to 1.30 in normal limbs and from 0.21 to 0.82 in limbs with arterial obstruction. Little correlation ($r = 0.292$) was seen between the maximal postcompression/precompression flow ratios and the resting ABI. Likewise, flow ratios

correlated poorly ($r = 0.279$) with toe pressures, which ranged from 15 to 95 mm Hg in 29 limbs with arterial disease.

DISCUSSION

This study of control subjects and patients with a wide range of ankle pressures demonstrates that intermittent calf compression increases popliteal arterial blood flow even in limbs with arterial insufficiency in which the arterioles are already partially vasodilated. To avoid the confounding effect of variations in limb size, we used postcompression/precompression flow ratios rather than absolute volume flow measurements. Although volume flow rates in animals measured with the instrument used in this study [12] and similar duplex scanners [13] have been shown to be accurate, results in atherosclerotic arteries may be subject to errors related to the angle of insonation and the irregular lumen of the vessels. Every effort was made to minimize these errors by maintaining the angle and position of the probe constant between measurements.

The mechanism postulated to explain the increased flow is roughly analogous to the pumping action of the calf muscle in normal walking. With the patient in the seated position, arterial and venous pressures in the calf are increased by an equal amount, commensurate with the elevated hydrostatic pressure. External compression elevates tissue pressure briefly and to a varying degree evacuates the underlying veins. Thus external compression produces a transient reduction in venous pressure but has little or no effect on arterial pressure. [9] Because of the increased arteriovenous pressure gradient that results from this action, arterial inflow is temporarily augmented. It is critical that the compression phase be short compared with the decompression phase to allow time for a flow increase to occur. Prolonged compression would elevate venous pressure and impede blood flow. [14], [15]

Our observation that calf compression failed to affect popliteal arterial blood flow in control subjects in the prone position is compatible with the mechanism outlined previously. With the subject in the horizontal position, venous evacuation produces little change in venous pressure and therefore has essentially no effect on the arteriovenous pressure gradient. The hypothesis also explains why the average postcompression/precompression flow ratios rose in seated subjects as cuff pressures approached the venous hydrostatic pressure (approximately 50 mm Hg [16]) but showed little further change beyond a cuff pressure of 60 to 80 mm Hg (Fig. 3, Fig. 4). Reduction in venous pressure is proportional to the extent of venous evacuation, which is incomplete until the pressure transmitted to the outside of the venous wall exceeds that in the lumen. Because part of the external pressure may be dissipated in the underlying tissues, cuff pressures higher than the venous hydrostatic pressure may be required in some limbs. Once the veins are completely collapsed by cuff compression, further increases in cuff pressure have little effect on venous pressure or on the arteriovenous pressure gradient.

Although reduction in the arteriovenous pressure gradient affords a plausible explanation for the increase in blood flow, the possibility that there may also be a direct reduction in peripheral resistance must be considered. Because blood flow through the calf should equal the arteriovenous pressure gradient divided by the resistance, the anticipated

postocclusion/preocclusion ratio can be calculated by dividing the postocclusion by the preocclusion arteriovenous pressure gradient, provided the resistance remains constant. [17] For example, if one assumes a hydrostatic pressure of 50 mm Hg and that compression temporarily reduces the total venous pressure from about 60 to 10 mm Hg, the expected postocclusion/preocclusion flow ratio would approximate 1.5 to 1.7 in limbs with normal (mean supine) arterial pressures (~80 to 120 mm Hg) and might range from about 1.7 to 3.0 or greater in limbs with reduced arterial pressures (~35 to 80 mm Hg). On the basis of these and similar estimates, it seems that a large part of the flow increases in control subjects (mean flow ratio 4.4 ± 2.0), and at least some of that observed in patients with arterial disease (flow ratio 3.2 ± 1.6) must be attributed to vasodilation. This disparity is consistent with the already partially dilated state of the microvasculature in limbs with arterial obstruction.

That limb compression produces a greater flow increase than predicted on the basis of the arteriovenous pressure gradient was also noted by early investigators. [18], [19] However, the mechanism responsible for the decreased resistance remains unclear. Because the duration of compression in our study was brief (2 seconds) and the highest compression pressures used were lower than arterial pressures (except in the most seriously diseased extremities), it is unlikely that vasodilation was due to postocclusion reactive hyperemia. Reactive hyperemia occurs only after a prolonged period of ischemia (minutes) in response to accumulation of metabolic byproducts and oxygen deficit. [17] In our study there was no ischemic period and the duration of the flow increase was less than that characteristic of reactive hyperemia. A fourfold increase in blood flow also occurred in the patient we studied 6 weeks after a lumbar sympathectomy, making a neural mechanism unlikely. Others have noted a similar response to limb compression after sympathectomy. [9], [16], [18]

The suggestion has been made that endothelium-derived relaxing factor released by the mechanical stimulation of leg compression could be responsible for increased postcompression blood flow. [11], [20], [21] Endothelium-derived relaxing factor acts on vascular smooth muscle within seconds and has a half-life of only 6 seconds, [22] a time course compatible with the observed vasodilation. We noted, however, that flow increases without compression when, after a brief elevation of the leg, the subject quickly shifts from a supine to a sitting position. This is difficult to explain on the basis of a humoral vasodilator.

Pain is relieved and foot perfusion is improved in patients with severe arterial insufficiency when the legs are dependent. [23], [24], [25] The enhanced perfusion is attributed to passive dilation of the peripheral blood vessels in response to increased hydrostatic pressure. Although Scheinberg et al. [18] noted that flow rates measured by water plethysmography increased more than 200% in patients examined in an erect position after compression of the foot veins, their study did not separate the effect of the erect posture itself from that of venous emptying of the foot. Later investigators, however, have avoided this pitfall by obtaining baseline measurements in the sitting position.

Gaskell et al. [9] measured blood flow in the skin of the forefoot with the xenon clearance technique in seated patients before and after inflating a mechanical pump applied to the foot and ankle for 2 seconds every 15 seconds. In patients with an ankle pressure equal to or less than 60 mm Hg, shifting from a supine to a sitting position increased blood flow by 82% ± 19%.

Pumping increased blood flow by an additional 104% ± 44% for a combined effect of sitting and pumping of 186% ± 50%.

Experiments reported by Morgan et al. [11] in 1991 showed that intermittent foot compression increased mean popliteal arterial blood flow by 93% in seated subjects without symptoms and by 84% in patients with arterial obstruction (ABIs of 0.33 to 0.74). More recently, Abu-Own et al. [21] reported that intermittent foot compression increased laser Doppler flux measured on the big toe by 66% in the control group and by 57% in patients with claudication who had ABIs ranging from 0.31 to 0.68. A smaller but statistically significant rise in the toe transcutaneous oxygen tension was also observed in the control group (10%) and in patients with arterial disease (8%). Clearly limb compression seems to have a positive effect on arterial blood flow in patients with a low ankle pressure, an observation that is in accordance with our findings.

Other authors have shown that intermittent brief insufflation of pneumatic cuffs applied to the calf increases foot blood flow in patients without arterial obstruction and in patients with arterial obstruction. [16], [19], [26] Of particular interest are Dillon's observations. [10], [27] In his studies cardiosynchronous (every second QRS complex) inflation of pneumatic boots enclosing the leg from the groin or calf to the toes increased pulse volume in the calf, subcutaneous oxygen tension in the foot, and Doppler signals in the pedal arteries of normal limbs and limbs with severe symptomatic arterial obstruction. Together with local antibiotic injections, repeated treatments were believed to have been successful in preventing amputation in 31 of 34 legs of patients with diabetes who had soft-tissue infections, osteomyelitis, ischemic ulcers, or localized gangrene caused by peripheral arterial insufficiency. [27] Although Dillon's method differs in some respects from that used in this study, it is probable that the mechanism underlying the increase in flow is similar. [10]

Our observation that intermittent calf compression augments popliteal arterial blood flow may have potential clinical implications. Increased pressure gradients, flow rates, and shear stresses might serve as a stimulus for collateral development. [28] The response therefore may extend beyond the immediate period of compression. Although we did not study the effect of foot compression on blood flow in the dorsalis pedis or posterior tibial arteries, it is logical to assume that the response would be analogous to that observed in the calf. The results of previous investigations suggest that foot compression augments blood flow, improves oxygen tension, and may be beneficial in patients with marginal distal perfusion. Detrimental effects are possible but have not been reported.

Although proposals to use limb compression as a method for augmenting blood flow have been made sporadically for decades, the method has received scant attention and has never been subjected to critical study. Thus at this point a healthy skepticism is appropriate. Further work is required to ascertain whether compression devices specifically designed for the purpose of improving arterial inflow in patients with unreconstructible arterial disease might have a role in alleviating rest pain or in promoting healing of ischemic foot lesions.

Acknowledgements

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