Effects of Ankle-to-Knee External Pressures on Skin Blood Perfusion Under and Distal to Compression

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ABSTRACT

OBJECTIVE: To compare the effects of select leg compression pressures on blood perfusion in the skin overlying bone and the skin distal to the leg compression.

DESIGN: Blood perfusion of skin overlying the tibia and the foot dorsum was simultaneously measured in 12 healthy subjects using laser Doppler. Each subject's calf was compressed from ankle to knee with an air cast that applied external pressure, ranging from 0 to 40 mm Hg in 10 mm Hg increments. All measurements were initiated after a 20-minute rest interval and were recorded with each subject in a supine position.

SETTING: University research center

RESULTS: The main findings show that compression causes a significant decrease in skin blood perfusion (SBF) at both sites, with greater reductions in SBF occurring with increased compression pressure. Greater SBF reductions occurred at the foot than at the directly compressed tibia site. Thus, at the highest pressure (40 mm Hg), the foot SBF reduction (61.6% ± 13.7%) was greater than at the directly compressed tibial site (33.3% ± 0.3%), both of which were significantly less than baseline (P <.01).

CONCLUSIONS: The present findings and considerable related evidence indicate that lower extremity external compression, within the range of commonly used therapeutic levels, reduces SBF by an amount that depends on the magnitude of the compression pressures. The quantitative difference between the effect of compression on SBF within the compression area, and distal to it, is likely a result of the way that the compression affects arteriolar and venous resistance to blood flow at the 2 sites. From a clinical perspective, it would seem prudent to anticipate that sub-bandage and distal SBF would be negatively affected by compression, with greater effects likely in tissue distal to compression.

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Compression bandaging is a primary treatment for lower extremity venous ulcers[1] and a principal component of treatment for peripheral edema and lymphedema.[2] Current concepts indicate that compression effectiveness for venous ulcers is partially linked to the amount of sub-bandage pressure exerted. Clinical practice, in part, is based on higher compression levels yielding a more favorable outcome on venous ulcers.[1,3,4] As a result, sustained sub-bandage pressures of 40 mm Hg and higher are selectively advocated.

Numerous investigators have considered the potential impact of various types of compression on lower extremity blood flow. The predominant pattern of noncompromised blood flow in lower extremities is flow pulsations that are synchronized with each heartbeat. The total or mean limb blood flow is considered to be the average value of these pulsations. The effects of foot-to-knee compression bandaging on pulsatile flow patterns were studied using nuclear magnetic resonance flowmetry,[5,6] which showed an increase in both the peak and duration of flow pulses measured in the bandaged leg. In contrast, midcalf pulsatile blood flow was progressively decreased with localized thigh compression pressures from 10 to 40 mm Hg.[7] The increased pulse flow within the compressed

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calf was thought to occur as a result of changes in effective arterial compliance and arterial transmural pressures associated with the bandaging. Based on the findings in that study, the authors speculated that pulse flow augmentation, independent of possible effects on mean blood flow, may play a positive role in the therapeutic efficacy of compression bandaging.

In contrast to pulsatile flow, lower extremity mean arterial blood flow has been studied using plethysmography, with mechanical[8,9] or electrical[10,11] transducers sensitive to limb volume changes. Although few studies have used this method to investigate the effects of leg compression on mean arterial blood flow, some data suggest that blood flow decreases with increasing ankle-to-knee compression, starting at compression pressures as low as 10 mm Hg, with a progressive flow reduction reaching 58% at a pressure of 40 mm Hg.[8] These investigators also described an increase in pulsations with increasing external pressure.

At the local tissue level, blood flow changes associated with external compression have been measured using radioactive tracer clearance and laser Doppler methods. Early work used the clearance of ^{133}Xe to assess subcutaneous and muscle blood flow changes due to external leg compression.[12-15] Findings relevant to the present study showed that midcalf regional blood flow remained unchanged at a compression pressure of 10 mm Hg, but progressively decreased thereafter. At a pressure of 40 mm Hg, the composite data indicated a flow reduction in the compressed region of about 40% in subcutaneous tissue and 35% in muscle tissue.

Not all studies, however, have yielded consistent results. Studies using laser Doppler to investigate the effects of circumscribed regional leg cuff compressions in the gaiter area have reported essentially the opposite results. An increase or no change in sub-cuff SBF over the range of 10 to 60 mm Hg was reported in one study,[16] whereas another study reported significant decreases (40% to 50%) in both sub-cuff and distal foot dorsum SBF accompanying sustained 40 mm Hg compression.[17] Four-layer, foot-to-knee compression bandaging has reportedly reduced distal toe perfusion by 36%[18]; other bandaging has reduced distal toe perfusion by 27% to 44%, depending on bandage type used.[7] In other studies,[19,20] sub-bandage SBF overlying soft tissue was not significantly decreased.

Because external compression pressure of the leg potentially impacts circulation in several ways, it is important to clarify the relationship between compression pressure levels and local blood circulation in both compressed and noncompressed tissue. Previous work revealed that foot-to-knee compression bandaging of healthy legs, at an average sub-bandage pressure of 27 mm Hg, resulted in significant reductions of SBF at toe sites distal to compression, but had little detectible effect on SBF under the bandage in skin overlying soft tissue of the calf.[20] However, effects of higher compression pressures were not addressed in that study. An additional clinically relevant question, not previously investigated, is whether the negligible effect of compression on SBF overlying softer tissue can be reliably extrapolated to sub-bandage SBF effects in skin overlying bone. To answer this question, an ankle-to-knee air cast applied stepped compression between 0 to 40 mm Hg to the leg and simultaneously measured changes in SBF overlying the tibia and dorsum of the foot in 12 healthy volunteers.

METHODS

Subjects

Twelve healthy female subjects were randomly selected from 100 medical school students and staff volunteers; all signed an approved institutional review board informed consent. Females were selected to match the gender of the group previously studied at lower pressure.[20] The subjects (6 Caucasian, 3 Hispanic, and 3 Asian) were aged 25.5 ± 3.1 years, with an average height of 64.2 ± 1.7 inches and weight of 118.7 ± 5.0 pounds. None of the subjects had diabetes, history of hypertension, or known vascular problems, nor were any of them using a vasoactive medication. Ankle-brachial systolic pressure indices determined that all subjects had normal lower extremity arterial circulation (1.06 ± 0.01). Arm blood pressure (BP) measurements indicated systolic and diastolic BPs were within the normal range (systolic, 104 ± 4; diastolic, 69 ± 7); mean BP was 80 ± 8 mm Hg.

Initial procedures

Subjects, wearing shorts, arrived at the testing laboratory and assumed a supine position on a standard examination table that had been covered with a comfortable support surface. A BP cuff was placed around each subject's thigh, just proximal to the knee. Cuff inflation was later used to produce a suprasystolic compression to determine the biologic zeros of the laser Doppler apparatus.[21,22]

Leg length (L) was determined by measuring the distance between the midpoints of the lateral malleolus and the tibial tubercle. Limb circumference was measured with a tape measure at positions corresponding to 10%, 25%, 50%, 75%, and 90% of L. Two laser Doppler probes were then placed on the left limb. One probe was placed overlying a flat portion of the tibia at a standardized position located at 50% of L, and the other probe was placed on the distal foot dorsum between the great and second toes.

The tibial probe (P-440 Soflex; Vasamedics, Eden Prairie, MN) is relatively thin (1.3 mm), with a total surface contact

area of 9 cm^2. Made of a flexible silicone elastomer, it conforms to the skin surface and helps minimize focal pressure effects. Pressure measurements on the tibia, with the probe present and absent, were within 2 mm Hg of each other at a compression pressure of 40 mm Hg. The foot dorsum probe was a standard right-angle probe (Vasamedics).

Each probe was connected to a separate laser Doppler monitoring system (Laserflo BPM^2, Vasamedics). Signal outputs were recorded in real time on a dedicated data acquisition computer. All data were recorded using a system time constant of 1 second, with an analog output full-scale gain setting of 50. A small thermocouple was then placed on the foot dorsum and calf to monitor skin temperature. Finally, an open-toed, half-leg, double-walled, transparent air cast (40 cm long) was positioned under the calf in the open position. The lower edge of the air cast was placed slightly proximal to the malleolus and zipped closed. The cast circumference was sufficiently large in relation to the subject's limb girth to ensure that the zipped position caused no added pressure. After all preparations were made, the torso and legs were covered with a blanket, and an additional 10-minute interval was observed prior to starting data acquisition.

Protocol

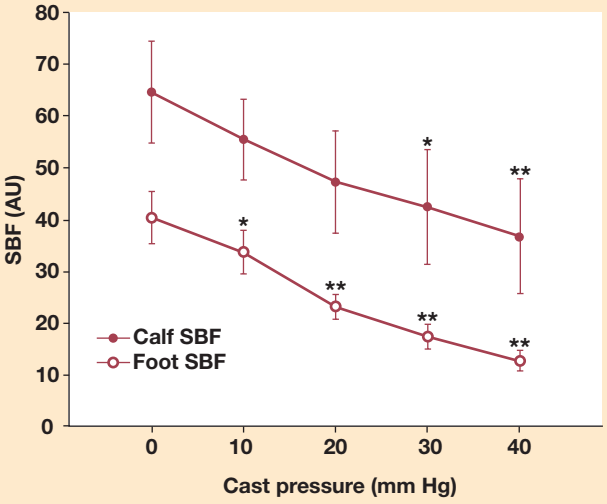
Laser Doppler SBF was continuously recorded from the leg and foot sites for 30 minutes. The air cast pressure during this time was increased in steps of 10 mm Hg, from 0 to 40 mm Hg, with each pressure level being held for 5 minutes. Air cast pressure was changed by manually increasing air volume with a BP bulb; pressure was continuously monitored with a mercury manometer.

Prior preliminary experiments using pressure sensors (Oxford Pressure Monitor; Talley Medical, UK) placed between the air cast and skin surface showed that the pressure exerted by the air cast on the tibia site was within 2 mm Hg of that recorded by the manometer. In other experiments, pressure variation was tested at 10% L, 50% L, and 90% L along the lateral and medial aspects of the leg. At a cast pressure of 40 mm Hg, the mean and standard error of the mean (SEM) of the measured pressures among these sites was 42 ± 1.5 mm Hg, with no indication of a pressure gradient from distal to proximal leg sites.

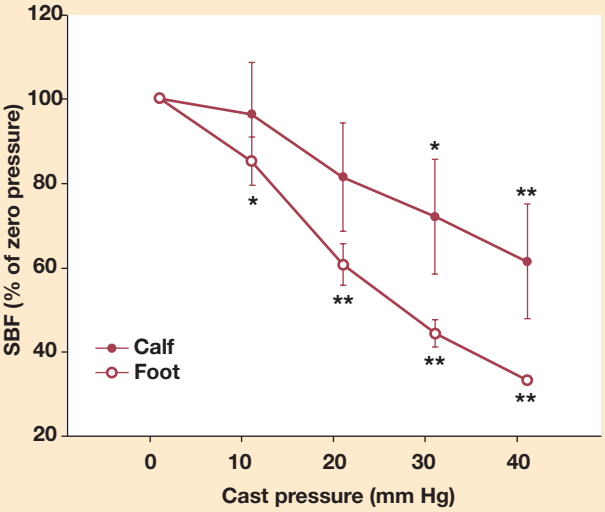
Pressure increases from one level to the next required less than 5 seconds. At the end of the stepwise pressure increases (40 mm Hg), the pressure was rapidly released and recording was continued for an additional 10 minutes. The thigh cuff was then inflated to 40 mm Hg above systolic pressure and maintained for 2 minutes as the biologic zero for both laser Doppler probes was recorded. Skin and room temperatures were measured and recorded every 5 minutes.

Figure 1.
SKIN BLOOD PERFUSION CHANGES WITH INCREASING COMPRESSION PRESSURE

A. Absolute SBF in arbitrary laser Doppler units (AU).



B. SBF expressed as a percentage of the zero pressure value. Data are mean values; bars are SEM. * and ** denote SBF significantly different than at zero pressure at $P < .05$ and $P < .01$ levels, respectively.



Calculations and analyses

The average SBF was determined during each interval by off-line computer processing of the raw data, recorded in volts. Overall effects of compression pressure on SBF were initially

tested using a general linear analysis of variance model for repeated measures (v6.1; SPSS, Inc, Chicago, IL). Follow-up tests compared the average SBF in each pressurized interval with the baseline zero pressure SBF. Changes from baseline were statistically assessed using nonparametric comparisons for related samples (Wilcoxon) with a *P* value of .05 being considered a statistically significant threshold. Mean arterial pressure (MAP) was calculated as the diastolic pressure plus one-third of the pulse pressure. During pressurization, the effective leg perfusion pressure was estimated as the difference between each subject's MAP and the compression pressure.

RESULTS

Distal foot dorsum

The zero pressure baseline at the foot dorsum (40.4 ± 5.0) decreased progressively as leg compression pressure increased (Figure 1A). Analyses, using a general linear model for repeated measures, showed an overall significant difference within pressures ($P < .001$). Follow-up tests showed that SBF is already significantly reduced ($P < .05$) at 10 mm Hg and that the significance was higher for higher pressures ($P < .01$). At the highest compression pressure (40 mm Hg), foot SBF was reduced to 12.8 ± 1.8 arbitrary units (AU), which is 33.3 ± 0.3% of the nonpressurized baseline (Figure 1B).

Calf tibial skin

When tissue was directly pressurized at the tibial site, the zero pressure baseline was 64.5 ± 9.9 AU. An overall decrease in SBF was found for the stepwise pressure increase sequence ($P < .001$) (Figure 1B); however, differences from baseline were not individually significant for pressures lower than 30 mm Hg. At a cast pressure of 30 mm Hg, SBF was reduced to 42.5 ± 11.0 ($P < .05$) and, at the highest compression pressure (40 mm Hg), tibia SBF was reduced to 36.7 ± 11.1 AU ($P < .01$), which is 61.6 ± 13.7% of the nonpressurized baseline level.

Temperature

Starting foot and calf temperatures of 28.9°C ± 0.5° and 31.1°C ± 0.7°, respectively, were slightly decreased by the end of the experiment to 28.2°C ± 0.7° and 30.6°C ± 0.8°, respectively. Starting and ending room temperatures, 22.1°C ± 0.3° and 22.3°C ± 0.3°, were not significantly different.

DISCUSSION

Main findings

The results demonstrate that SBF distal to and within a region of leg compression is increasingly compromised in direct relation to applied compression pressure. The magnitude of SBF decline is more pronounced distal to the leg compression region than directly under the compression site. The absolute magnitude of SBF reduction at the highest compression pressure used (40 mm Hg) was 66.6% at the distal foot dorsum site and 38.6% at the directly compressed tibia site. The quantitative difference is largely due to the differential effects of compression on the effective perfusion pressure that drives blood flow through the 2 sites and on opposite effects on vascular resistance, as discussed below.

Distal flow reduction

The effective perfusion pressure for the distal site may be approximated as the difference between MAP and compression pressure. Based on the measured average MAP for this study group, the average, noncompressed overall perfusion pressure was 80 mm Hg. As an approximation, a compression pressure of 40 mm Hg would reduce this to 40 mm Hg (80 to 40 mm Hg). This represents a 50% reduction in effective perfusion pressure and, by extension, results in a similar reduction of blood flow. Because the data indicate a mean SBF reduction of 66.6%, other factors must be involved. The other main factor is thought to be a calf compression-induced increase in venous resistance between the foot dorsum and the venous outflow from the leg. A reduction in perfusion pressure driving blood through the foot, combined with the increased outflow resistance due to the proximal compression could, therefore, explain the significant blood flow reduction.

Compressed region flow reduction

For the tibial site, which is directly exposed to compression pressure, the local perfusion pressure is also related to the difference between MAP and local venous pressure. However, because this site receives a direct external pressure, underlying arteriolar blood vessels experience a net decrease in transmural pressure that triggers a myogenically dependent vasodilation.[23-25] The associated reduction in effective arteriolar vascular resistance explains the lesser blood flow deficit compared with the distal dorsum site. This concept is supported by an observed increase in arteriole diameter with external compression[26] and observed blood flow autoregulation associated with both intravascular hypotension and increased tissue pressure.[18]

Clinical significance

The present findings and the large number of related works indicate that lower extremity external compression reduces SBF in the foot and digits by an amount determined by compression pressure levels, within the range of commonly used therapeutic compression levels. The effects within the compressed leg region are less clear and more variable; however, the present results clearly show a significant graded blood perfusion reduction over

the range of 20 to 40 mm Hg. The fact that such localized reductions have not been previously reported in all studies using laser Doppler measures but have been observed with tracer methods may be related to the sites of previous laser Doppler measurement, which have all been made on the relatively softer tissue in the calf and gaiter areas. Compression in these areas may cause the laser Doppler probe to be displaced inward and sample from a deeper depth with a generally greater perfusion level. This is not the case for probe placement on skin overlying the tibia, which should register perfusion changes from approximately the same tissue depth and vessels over the full compression range. From a clinical perspective, it would seem prudent to anticipate that sub-bandage SBF is negatively affected by compression, although the effect is not as great as in tissue distal to the bandage edge. ●

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