# Effect of Sustained Regional Compression on Lower Extremity Skin Microcirculation

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**Abstract.** Laser–Doppler blood perfusion was measured on foot dorsum (lateral and medial) and medial lower calf before (10 minutes), during (40 minutes) and after (10 minutes) of lower leg regional external compression at 40 mm Hg using a blood pressure cuff in 15 healthy supine subjects. Dorsum transcutaneous oxygen tension (TcPO2) was measured simultaneously. The purpose was to determine the effect of controlled and sustained regional compression on skin microcirculation at and distal to the compression site using a pressure level routinely employed for treatment of venous ulcers. Significant ($p < 0.01$) reductions in laser–Doppler blood perfusion under the cuff and at both distal noncompressed sites occurred with compression and lasted the full 40 minutes. Under–cuff minimums (percent of pre–compression baseline) occurred during the first 5 minutes (55.3 percent +/- 5.7) and distally at the end of compression (60.7 percent +/- 3.9 and 63.9 percent +/- 5.2) for medial and lateral dorsum respectively. No perfusion trends during compression were noted. Dorsum TcPO2 gradually declined with a minimum (89.6 percent +/- 2.2) after 30 minutes of compression. After pressure relief, perfusion increased but no significant hyperemic response was noted in the dorsum (102.6 percent +/- 6.1) whereas below–cuff perfusion increased in the immediate 5 minute post–compression interval (122.2 percent +/- 19.3). All values were insignificantly different than baseline during the final five minutes. The sustained and significant blood perfusion decreases found in healthy subjects are directly applicable to the regional compression employed but are believed to represent an upper bound on that to be expected with full leg compression bandaging. The findings reinforce the need for caution regarding therapeutic compression levels in patients with reduced vascular function. Because the effects are manifested distally, appropriate and timely perfusion monitoring at distal un–compressed sites may be efficacious to assess patient–by–patient compression effects and help guide the choice of appropriate compression levels.

**WOUNDS 1996;8(4):111–117**

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*The research support of the Walter G. Ross Foundation is gratefully acknowledged.*

## Introduction

External compression of limbs is used therapeutically for a variety of conditions including the treatment of venous ulcers. In spite of its long–time usage, several aspects of the hemodynamic sequelae to externally applied pressures are incompletely characterized. It is well–recognized that external pressures high enough to compress arteriolar blood vessels result in blood flow decrements which if sustained may produce ischemic effects on the dependent tissue. However, it may be less widely realized that significantly lower pressures have been reported to have potential untoward effects. Data obtained

from plethysmographic measurements of blood flow under inflatable plastic splints indicate flow reductions at pressures as low as 30 to 40 mm Hg.[1] Other workers have demonstrated decreases in whole leg flow with short term compression levels well below arterial pressures.[2–9] Blood flow measured with $^{133}Xe$ clearance indicate that flow reductions may occur at 10 mm Hg of calculated local skin compression pressure,[6] and graded flow decreases with increasing levels of sub–arterial pressures have been reported.[10,11] Significant reductions in leg muscle and subcutaneous oxygen tension (TcPO2), accompanying whole leg external pressures as low as 20 mm Hg[12] with increased effects on elevated limbs,[13] have been described. The TcPO2, which is a reflection of micro–circulatory blood perfusion, was reduced after direct loading of calf muscle with as little as 20 mm Hg and over bone with near zero values at 40 mm Hg of applied pressure.[14] In forearm, similar TcPO2 findings had been reported.[15] However, there has been little systematic study of the impact of lower extremity extended compression directly on skin microcirculatory blood perfusion. In view of the composite evidence suggestive of potential clinically relevant sub–arterial compression impacts on limb circulation, an issue raised to increasing importance in view of a trend for the use of higher compression therapy in selected patients to achieve more effective ulcer healing,[16–20] the present study sought to better define the microcirculatory effects of sustained (40 minute), sub–arterial compression pressure (40 mm Hg) of a circumscribed region of the distal lower limb. The specific, albeit limited, initial aim was to determine and compare the magnitude and time course of the microcirculatory changes as measured with laser–Doppler simultaneously within the region of the compression (gaiter region) and at skin sites distal to this region (foot dorsum) both during the sustained compression and following its release.

## Methods

**Subjects.** Fifteen healthy volunteer subjects (7 female) participated after being informed as to the details of the study and signing an informed consent approved by the Institutional Review Board. Subjects ages ranged from 21 to 52 years. All subjects had normal lower extremity circulations as determined by pretest measurements of systolic ankle–brachial indices, below–knee pulsatile blood flow and arterial status index.[21,22] No subject had diabetes or evidence or history of skin or venous disease in the tested limb. No subject was taking vasoactive anti–inflammatory medication. One subject was a current cigarette smoker.

**Compression and Below–Cuff Laser–Doppler.** The subject, dressed in loose fitting street clothing, assumed a supine position on an examination table upon entry into the testing laboratory which was temperature controlled to between 22 and 23° C. Compression was produced with a standard adult blood pressure cuff (13 cm width) connected to a mercury manometer. Prior to placement of the cuff on the leg, a laser–Doppler probe (Vasamedics P–440 Soflex probe, Vasamedics Inc., St. Paul, MN) was placed on the medial aspect of the lower left leg with the active sensing portion of the probe positioned at a standardized site. The location was determined in each subject by measuring the distance between the medial malleolus and the knee and then placing the probe at a distance from the medial malleolus equal to 25 percent of the malleolus–knee length. Typically this distance was about 8 to 10 cm. The circumferential position was at or near the center of the medial surface and on near–flat soft tissue. The probe encapsulation, which is a soft flexible silicone elastomer, conforms gently to the skin surface and minimizes potential tissue trauma subsequent to compression because of its softness and its uniform surface area which minimizes pressure concentration effects. The encapsulation thickness, which included the imbedded transmitting and detecting fibers, was 2.2 mm. The cuff was then placed around the leg with the center of the bladder positioned over the active portion of the probe and the probe cable connected to the laser–Doppler system (Vasamedics BPM2, Vasamedics Inc., St. Paul, MN).

**Microcurculatory Measurements Distal to Cuff.** In addition to the laser–Doppler probe at the cuff site, two additional probes (Moor MBF3D) were placed on the foot dorsum, one just proximal to the junction of the first and second toes and the other just proximal to the junction of the fourth and fifth toes. Both of the foot dorsum probes were connected to a dual–channel laser–Doppler system (MOOR, Instruments Ltd., UK). Laser–Doppler blood perfusion data obtained from both systems was acquired in real time by computer and analyzed at the end of the procedure. All data was obtained using a time

**Table 1**
**Supine resting leg parameter values**

| Flow (ml/min) | | Perfusion (ml/min/100 cc) | | | Pressures mm Hg | mm Hg/mm Hg | Geometry cm | cm³ |
|---|---|---|---|---|---|---|---|---|
| $Q_{90}$ | $Q_{25}$ | $PP_{90}$ | $PP_{25}$ | ASI | $P_{ankle}$ | ABI | $C_{25}$ | Volume |
| 48.3 | 21.5 | 1.68 | 1.71 | 7.62 | 130.8 | 1.02 | 21.6 | 1954 |
| (4.8) | (1.7) | (0.20) | (0.16) | (0.88) | (5.5) | (0.02) | (0.7) | (122) |

constant of one second and cutoff frequencies of 14.9 kHZ; blood perfusion was herein reported in arbitrary units (a.u.) as is standard practice for laser–Doppler measurements. Transcutaneous oxygen tension (TcPO2) measurements were obtained at 45° C using a TcPO2 probe placed between the two dorsum laser–Doppler probes (Novametrix model 811, Novametrix Medical Systems Inc., Wallingford, CT). Following probe placement, the lower leg and foot were lightly draped to minimize possible effects of convective air currents. Technical aspects, operating principles and features of the laser–Doppler blood perfusion measurements in our hands have previously been reported.[23–26]

**Protocol.** Testing was done in a quiet, low–lit temperature–controlled room. Room temperature variation over the test interval did not exceed 1.6° C. The subject was placed in a fully supine position on an exam table and the probes and cuff applied. The TcPO2 probe was then brought to operating temperature and recordings made every 5 minutes for 30 minutes. At 25 minutes, brachial arterial blood pressure was measured and repeated at the end of the protocol. At 30 minutes after activating the TcPO2 measurement, laser–Doppler data acquisition began and continued for the duration of the experiment; TcPO2 was recorded at 5 minute intervals. After obtaining 10 minutes of baseline data at zero cuff pressure, the cuff was inflated to 40 mm Hg and maintained (+/- 1 mm Hg) for 40 minutes. The cuff pressure was then rapidly released and the recovery response followed for 10 minutes. The cuff was removed and the skin site, where the laser–Doppler measurement was made, was visually examined for indications of probe related skin indentations, trauma or erythema. No evidence of erythema was detected.

**Analysis.** Laser–Doppler data was analyzed as follows: for the zero–pressure baseline, the average of the 10 minute interval was computed (denoted as Qo); for the 40 minute compression interval, the averages of each of the eight contiguous five minute intervals were computed (denoted as Q1 through Q8); and for the 10 minute recovery interval, two contiguous 5 minute intervals were computed (denoted as Q9 and Q10). Relative changes from baseline of each measured parameter for each subject were calculated as the ratio of the value in each interval divided by the baseline average and expressed as a percentage. Statistical evaluations with regard to the effect of compression were made first by comparing laser–Doppler data (raw data in a.u.) in intervals 1 through 8 using the nonparametric Friedman two–way ANOVA for dependent samples for overall differences. Thereafter, the baseline interval was compared with interval 1 (first compression interval) and interval 8 (last compression interval). Similar methods were used for TcPO2 comparisons. For graphical presentation purposes overall results are shown as changes relative to baseline.

## Results

**Baseline Leg Parameters.** Table 1 summarizes pertinent baseline leg functional and anatomic parameters of the studied subject group. Blood flow and perfusion parameters, as well as pressure indices, were all well within the normal range. The ASI value (Arterial Status Index) is calculated from the flow pulse waveform as the ratio of the leg average blood perfusion divided by the flow pulse with the width measured at 50 percent of the pulse amplitude and expressed as a fraction of the cardiac cycle. ASI values > 4.5 are considered normal.[22]

**Response to Compression.** Reductions in

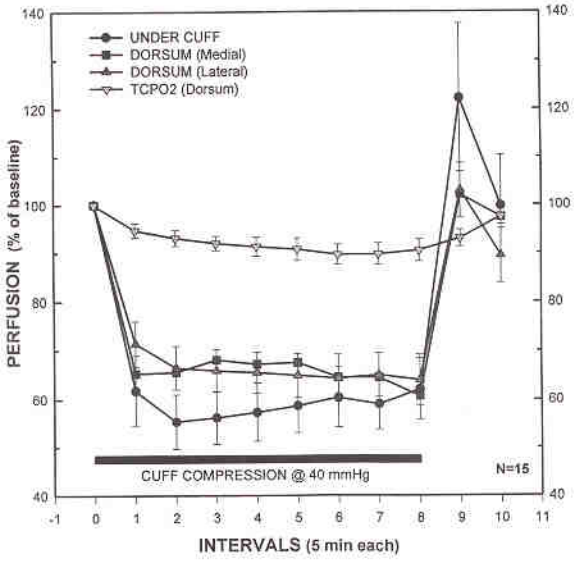


*Figure 1. Skin microcirculatory changes due to lower leg regional external compression for 40 minutes at 40 mm Hg pressure. Each laser–Doppler perfusion data point following the baseline pre–compression interval (0) is a five minute average; the baseline interval is a 10 minute average. TCPO2 values are those at the end of each interval. All values are given as a percentage of the baseline average. Bars are sem. All perfusion values during the compression intervals (108) are significantly less than baseline (p,0.01).*

laser–Doppler blood perfusion under the cuff and on both of the distal dorsum sites were found in every subject after initiation of cuff compression. As shown by the mean values in Figure 1, the perfusion was significantly reduced during the first compression interval and remained reduced throughout the 40 minute duration of sustained cuff compression. Minimum measured perfusion values occurred under the cuff during the first compression interval (55.3 percent +/- 5.7) whereas distal perfusion was found to be minimum at the end of the compression interval (60.7 percent +/- 3.9 and 63.9 percent +/- 5.2) for the medial and lateral dorsum respectively. Analysis of the baseline and the eight compression intervals shows an overall statistically significant decrease ($p < 0.01$) during compression as compared with baseline for each laser–Doppler measurement site. At no measured site was there a significant perfusion difference among the compression intervals and there was no evidence of a trend during the 40 minute compression. The foot dorsum TcPO2 also showed a decrement from its baseline value. As shown in Figure 1, its percentage change was much less than the blood perfusion decrement with a gradual decline during the compression interval to a minimum level slightly less than 90 percent of baseline (89.6 percent +/- 2.2) after 30 minutes of compression. Upon pressure relief, a rapid increase in perfusion was noted as perfusion increased toward or above baseline. Distal dorsum mean perfusion in the post–cuff release intervals did not differ between the dorsum sites and averaged 102.6 percent +/- 6.1 and 93.4 percent +/- 6.1 for intervals 9 and 10 respectively. These were not significantly different with respect to the pre–compression baseline. Post–compression perfusion increase at the under–cuff site was greater than at the dorsum sites, amounting to 122.2 percent +/- 19.3 during interval 9 and reaching baseline levels (99.7 percent +/- 10.7) during the final interval. No changes in pre– vs. post–compression systolic blood pressure (128.1 +/- 4.8 vs. 127.0 +/- 4.8 mm Hg) or diastolic blood pressure (78.2 +/- 3.5 vs. 88.5 +/- 3.5 mm Hg) were noted.

## Discussion

The present findings clearly demonstrate a significant decrement in skin microvascular blood perfusion subsequent to lower leg external compression of a circumscribed limited region when compressed at a level well below arterial pressure. The 40 mm Hg pressure used at the distal part of the leg is similar to pressures used with some elastic compression stockings and bandages.[16–20,27,28] It differs from these pressures in that herein only the distal part of the leg was compressed rather than the entire below–knee area as is done with most standard compression therapy. Because of this difference, the perfusion changes herein measured likely represent an upper–bound on those which may occur with full leg bandage compression. However, for the initial purposes of the present study this method provided a well controlled and uniform procedure for the estimation of local and distal compression effects. The percentage reductions in blood perfusion due to this compression, though slightly greater under the cuff, were similar whether measured directly under the compression cuff or distally and, depending on time interval, ranged between 55 and 70 percent of baseline. There was no evidence of a "vascular escape" phenomenon during the compression interval.

The fact that external compression at the pres-

sure levels herein used leads to a relative blood flow deficit in the compressed region, as well as distally, is in accord with previous studies which were primarily concerned with short–term compression effects on arterial inflow. Ankle–knee cuff–compression over a range of pressures showed that 40 mm Hg compression pressure reduced mid–calf muscle tissue blood flow under the cuff to about 58 percent of the non–compressed flow.[11] This flow reduction is similar to the below–cuff laser–Doppler derived microcirculatory perfusion percentage reduction herein found. Other studies with full leg (ankle–knee) and local calf compression[11,29] indicate a monotonic reduction in flow with increasing short duration compression levels with near–zero flow occurring when the compression pressure approaches arterial diastolic pressure. Since in these studies pressures were maintained for short time intervals as part of the flow measurement protocol ($^{133}$Xe clearance), inferences regarding possible flow adjustments during sustained compression were not studied by these workers. A possible adaptive vasodilatory response associated with compression is, in part, related to the concept that arteriolar transmural pressure is reduced by an amount proportional to the external compression, thereby eliciting a myogenically stimulated vasodilation. Some experimental evidence for this occurring locally at external pressures between 10 and 30 mm Hg has been reported.[10] There are older reports alluding to therapeutic intermittent venous occlusion as a method to increase arterial flow.[30–31] Macrovascular blood flow assessments using venous occlusion plethysmography suggest contrarily that limb flow decreases with external compression.[2–9] The present results are generally in agreement with this latter view in that the microcirculatory perfusion under the cuff did not significantly increase from its initially reduced value throughout the duration of the 40 minute sustained compression. By following the response over this duration yet observing no flow increases, one can tentatively also suggest that the possibility of slower acting vasodilatory mechanisms, at least over this time frame, are unlikely. However, the possibility that very brief–impulse–like compressions might induce a transient vasodilatory response and/or flow increase cannot be discounted. Indeed, recent measurement of popliteal blood flow before and after intermittent calf compression demonstrate a flow augmentation,[32] and a biphasic response to venous congestion has been previously reported.[33]

Blood perfusion changes distal to sites of sustained limb compression have not previously been intensively studied. Using ankle–knee bandages with sub–bandage pressure monitoring, a significant reduction in plethysmographically determined toe pulse amplitude was demonstrated when proximal pressures exceeded 30 mm Hg,[34] and after 40 minutes of 50 mm Hg compression the toe pulse amplitude was reduced to about 60 percent of its pre–compression value. The present findings indicate an accompanying decrease in distal foot dorsum skin microvascular perfusion amounting to a 30 to 40 percent decrease from pre–compression values and a 10 percent decrease in skin transcutaneous oxygen tension.

This reduction in microvascular perfusion does not necessarily signal an ischemic risk to the distal tissue. The absence of a significant hyperemic response upon release of the 40 minute regional compression suggests that in this healthy subject group the amount of flow reduction did not significantly produce a functionally important flow deficit. As noted, since the compression was only over a limited leg extent whereas most therapies encompass the region from near–toe to near–knee, it is presently unknown whether such applications would give rise to similar perfusion changes as herein demonstrated. However, the present findings do reinforce the need for caution with respect to therapeutic compression levels in patients with reduced vascular function.

The specific thresholds and types of preexisting vascular deficits which should preclude the use of "higher" levels of compression have not been addressed in the present work, nor are such thresholds clear in the literature. The demonstrated efficacy and benefits of compression therapy need to be weighed with respect to overall risks. The need for preliminary vascular assessments in patients targeted for such compression is a well–accepted premise by most practitioners. An ankle–brachial pressure index of 0.9 or less has been used to exclude patients from compression use[35,36] and this ABI level has been advanced as a cautionary flag for compression therapy.[37,38] Some workers have employed an ABI value of 0.8 as a suitable threshold.[17–20] With respect to direct compression therapy for venous ulcers, current practice guidelines clearly point out that dangerously high pressures over bony prominences may

result in some aged patients, especially those with thin legs.[39]

The present initial findings which are directly applicable to regional compression effects indicate that significant microcirculatory perfusion reductions occur at lower pressures even at non–bony prominences, both at regions under compression and distally. As noted, the magnitude of these reductions likely represent upper bounds on the deficit to be expected with full leg compression bandaging. Extensions of the present regional compression findings to full leg compression effects are currently underway. However, the present findings renew clinical questions regarding appropriate criteria for withholding use of adequate compression therapy and, when used, the need for timely monitoring of the effects of compression. Recent data indicates that at least 20 percent of patients with lower extremity ulcers have mixed venous and arterial components as judged by venous testing and ABI values.[40] The prevention of compression induced microcirculatory impairments in this patient group, as well as in others with less obviously present risk, may be approachable by using laser–Doppler perfusion monitoring of distal un–compressed tissue (dorsum or toe) to assess patient–by–patient compression effects and to help guide the choice of appropriate compression levels. Research on this aspect is currently underway.

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