Effect of Compression Bandaging on Lower Extremity Skin Microcirculation

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WOUNDS 1996;8(6):200–207

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The research support of the Walter G. Ross Foundation and Smith & Nephew United is gratefully acknowledged.

Introduction

Compression bandaging is the mainstay for the treatment of venous ulcers. Recent reports have indicated that achieving sustained sub–bandage pressures near 40 mm Hg may be more efficacious in providing timely wound healing than lower pressure levels.[1–4] However, since at least 20 percent of patients with venous ulcers may also have some degree of co–existing lower extremity arterial disease,[5] it is important to clarify the possible impact of higher levels of compression bandaging on lower extremity skin circulation. Though it has long been recognized that pressures high enough to compress arteriolar blood vessels can produce ischemic effects on the

dependent tissue, plethysmographic measurements of blood flow under inflatable plastic splints indicate flow reductions at pressures as low as 30 to 40 mm Hg,[6] and other workers using various compression methods have demonstrated decreases in leg mean flow using compression levels well below arterial pressures.[7–9] Contrastingly, when lower extremity compression was produced by elastic bandaging at similar levels, significant increases in leg pulsatile blood flow were noted.[10,11] Reductions in leg muscle and subcutaneous oxygen tension accompanying leg external pressures have also been reported.[12] Recent studies have demonstrated that in supine subjects, regional compression of the gaiter area via cuff compression to 40 mm Hg significantly reduced skin microcirculation below the cuff and at non–compressed distal sites on foot dorsum.[13,14] It is unknown if the impact of regional compression as previously used differs from the flow effects associated with therapeutic compression bandaging. The present study was undertaken to clarify this issue by more closely approximating the clinical situation. This was accomplished by using laser–Doppler measurements to determine the sub–bandage and distal (great toe) microcirculatory response of foot–to–knee compression bandaging on vascularly normal legs in the horizontal (supine) and leg dependent (seated) positions.

Methods

Subjects and preliminary evaluations. Healthy volunteer subjects (N = 14, age 39 ± 4.3 years, seven female) were studied after reading and signing an Institutional Review Board approved informed consent. No subject had diabetes, had any history of venous or arterial disease or was taking any vasoactive medication. Two subjects were current cigarette smokers. Absence of lower extremity arterial disease was confirmed in each participant based on screening with bilateral nuclear magnetic resonance flowmetry[15–20] and ankle–brachial systolic pressure indices (ABI) obtained using standard Doppler ultrasound at the posterior tibia and dorsal pedis arteries. All subjects tested normal with mean blood perfusion at the knee and ankle of 1.72 ± 0.18 and 1.68 ± 0.14 ml/min/100 cc respectively and an average ankle systolic pressure and ABI of 129.8 ± 4.7 and 1.06 ± 0.03 respectively. Blood pressures measured with standard pressure cuffs also verified that the group was normotensive (systolic 122 ± 4.1, diastolic 83 ± 2.9 mm Hg).

Leg compression bandaging. Leg compression was produced by wrapping one leg from fore–front to knee with a four–layer bandaging system.[1,3] The compression system chosen for use in this study (Profore*, Smith & Nephew United, Inc., Largo, FL) is one of several available commercially designed and used therapeutically for treatment of venous ulcers. The four layers of the bandage system, referred to as layers 1 through 4, are applied sequentially to the leg with each subsequent layer overlapping the other. Layer 1 is lightly wrapped and produces no measurable compression; layer 2 secures layer 1 and produces little compression; layers 3 and 4 are the elastic components which produce roughly equal compression pressures. In all subjects, application of the bandage was done by the same wound care nurse who had extensive experience with the use and application of this bandage system.

Preparatory sequence. Subjects, dressed in shorts or hospital gown, assumed a supine position on an examination table upon entry into the testing laboratory which is temperature controlled to between 22° and 23° C. 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 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 includes the imbedded transmitting and detecting fibers is 2.2 mm. Thereafter two pressure sensors were placed on the medial aspect of the leg near the laser–Doppler probe to record sub–bandage pressures using an automated monitoring system (Talley Oxford Pressure Monitor, MKII,

Table 1
Sub–Bandage Laser–Doppler Values

Parameter	Baseline	Bandaged	
	Supine	Supine	Dependent
Perfusion (a.u.)	0.87 ± 0.14	1.01 ± 0.21	1.38 ± 0.39
Volume (a.u.)	0.28 ± 0.04	0.30 ± 0.37	0.37 ± 0.07
Velocity (a.u.)	0.95 ± 0.09	0.88 ± 0.08	0.99 ± 0.09

Values are group means ± sem (N = 14) obtained from continuous 10 minute recording intervals during supine baseline (not bandaged) and bandaged with legs horizontal (supine) and dependent (seated).

Progressive Medical, Lansing, MI). The pressure sensors were located on either side of the laser–Doppler probe approximately 2 cm proximal and distal respectively. The pressure registered by each sensor was recorded in triplicate and the average of each of the three readings from each sensor used to specify sub–bandage pressure. Although the pressure sensors were thin, pliable and conformed to the contour of the skin it is possible that the recorded pressure might deviate from that which would be present in the absence of the measuring device. However, in separate tests of the pressure sensors in which they were compressed below a pressure cuff at a known pressure, the sensor pressure recorded values which were within a few mm Hg of the cuff pressure measured with a mercury gauge.

To monitor distal skin blood perfusion, two additional laser–Doppler probes (Moor, P7 large area probes, Instruments Ltd., UK) were placed on the pulp of the great toe of each foot. These probes were connected to a dual–channel laser–Doppler system (MOOR, MBF3D, Instruments Ltd., UK) Laser–Doppler perfusion data obtained from the distal toe sites and sub–bandage represent signals obtained from a depth of about 1 mm. The laser–Doppler perfusion data was acquired by computer and analyzed at the end of the procedure. All data was obtained using a time constant of one second and cutoff frequencies of 14.9 kHZ; blood perfusion is herein reported in arbitrary units (a.u.) as is standard practice for laser–Doppler measurements. Technical aspects, operating principles and features of the laser–Doppler blood perfusion measurements have previously been reported.[21–24]

Protocol. After placement of the probes, bandage layer 1 of the four–layer compression bandaging system was applied to both legs, the subject was covered with a light blanket and the feet were tented by a sheet. Covering both legs with layer 1 (non–compression) during baseline measurements and tenting the feet and exposed toes diminishes effects of environmental variables (room temperature, drafts, if any, etc.) on skin temperature and possibly blood flow between legs. Baseline measurements began 10 minutes after the final preparation and consisted of a 10 minute continuous recording of laser–Doppler signals from toe and sub–bandage probes. Thereafter, one leg was bandaged with the full compression system (layers 2, 3 and 4) and the subject re–evaluated after 20 minutes by a second 10 minute continuous recording. Finally the subject was assisted to a seated position with both legs dependent. After 20 minutes of leg dependency a final 10 minute data recording interval was initiated. Testing was done in a quiet, low lighted, temperature controlled room. Room temperature variation over the test interval did not exceed 1.6° C. In all subjects the bandage was applied by the same wound care nurse who has extensive experience with this compression bandaging system. The sub–bandage pressure recorded was 41.8 ± 2.0 mm Hg which is near the target level recommended for therapeutic use (40 mm Hg). At the end of the procedure, the bandage was removed and the skin site where the sub–bandage laser–Doppler measurement was made was visually examined for indications of probe related skin indentations, trauma or erythema. No evidence of erythema was detected.

Table 2
Toe Laser–Doppler Perfusion Values

Condition	Bandaged Leg	Control Leg	p–Value
Supine Baseline	52.6 ± 17.6	43.8 ± 15.0	0.730
Supine Bandaged	23.0 ± 6.0	41.0 ± 10.9	0.055
Dependent Bandaged	21.0 ± 7.7	13.5 ± 3.7	0.069

Values are laser–Doppler perfusion (a.u.) group means ± sem (N = 14) obtained from continuous 10 minute recording intervals. Statistics: paired–toe comparisons for bandaged leg vs. control leg, Wilcoxon test.

Analyses. Laser–Doppler data was analyzed by first computing the average skin blood perfusion during each of the following 10 minute intervals: 1) non–compressed supine baseline, 2) full compression bandage in place with subject in a supine position and 3) full compression bandage in place with subject in the seated position with both legs dependent. An interval of 10 minutes was chosen so that normal short–term physiological variations in blood perfusion could be suitably averaged. For the bandaged leg, sub–bandage laser–Doppler perfusion, rbc concentration and speed were compared with respect to their raw values in arbitrary units (a.u.). For the non–compressed distal toe measurements, paired–leg values of perfusion (bandaged leg vs. control leg) were compared on the basis of raw perfusion values (a.u.) and the percentage changes produced by bandaging and dependency. Statistical analyses were done using the Wilcoxon non–parametric test with a p–value < 0.05 being accepted as statistically significant.

Results

Sub–bandage effects. Neither compression nor leg dependency produced an overall statistically significant decrease in sub–bandage laser–Doppler values. The data shown in Table 1 hint of a trend for the mean perfusion to increase with leg bandaging and dependency. Analyses of individual responses showed that bandaging decreased perfusion (> 20 percent decrease) in four of the 14 subjects with a mean percentage decrease of 35.0 ± 9.9 percent and increased perfusion in six of the 14 subjects with a mean increase of 106 ± 34 percent. A clear explanation of these individual variations is not yet available.

Distal effects. Baseline toe skin blood perfusions (a.u.) were not significantly different between the bandaged and control legs (Table 2). During compression bandaging the mean perfusion of the bandaged leg decreased to less than half the baseline mean whereas the control toe perfusion remained near baseline levels. When the legs were placed in a dependent position there was a further decease in perfusion only in the control leg. Neither compression bandaging nor dependency resulted in a statistically significant difference between paired–leg raw perfusion values. However, when the responses are expressed as percentage changes, clear and significant differences are observed (Figure 1). As depicted in the figure, the effect of compression bandaging on the distal perfusion of the bandaged leg resulted in a mean decrease of 35.6 percent ± 12.8 whereas, during the same interval, the control toe mean perfusion increased to 28.5 percent ± 24.1. The paired–toe difference was highly significant ($p < 0.001$). The transition from the supine to leg dependent position resulted in a slight perfusion decrease in the bandaged leg as compared with the non–compressed baseline, but was accompanied by a significantly greater perfusion decrease in the control toe (7.8 percent ± 18.1 vs. 55.8 percent ± 9.9, $p < 0.001$). Expressing the response to dependency relative to compressed supine values shows no difference in the percentage decrease between bandaged and control legs (36.7 percent ± 19.1 vs. 38.8 percent ± 17.8). When legs were in the dependent position, perfusion ratios between paired–toes (bandaged/control) were essentially identical to those present during the supine baseline interval (1.87 ± 0.47 vs. 1.89 ± 0.46). But during the supine compression interval, this ratio was significantly reduced as compared

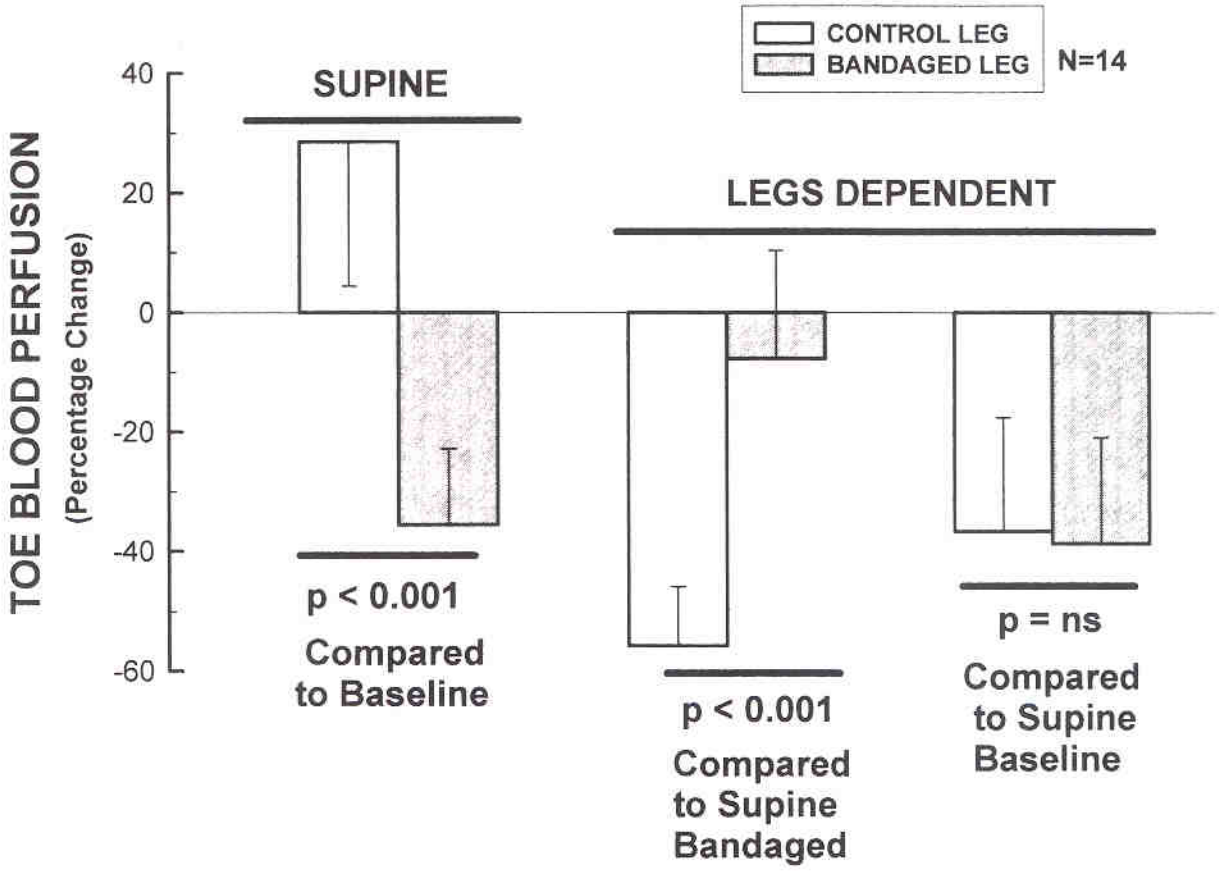

Figure 1. Percentage changes in toe skin blood perfusion due to compression bandaging and leg dependency. Bandaging is associated with a significant decrease in perfusion in the supine position but little further decrease during dependency. Overall percentage change in perfusion from baseline is the same for bandaged and control limbs.

to either of these values (0.93 ± 0.28, $p < 0.001$). It is noted that toe perfusion on the bandaged leg was decreased by at least 20 percent of its baseline value in 13 of the 14 subjects under supine conditions.

Discussion

The present findings indicate that skin blood perfusion responses to four–layer compression bandaging depend on both site and posture. Under supine and leg dependent conditions, bandaging did not reduce sub–bandage laser–Doppler blood perfusion. Contrastingly, under supine conditions bandaging clearly caused a decrease in toe perfusion of the bandaged limb as compared with the control limb. Furthermore, perfusion decreases normally seen with postural change from supine to seated were considerably blunted by the bandaging, causing a posturally related perfusion decrease that was significantly less than that measured in the control limb during the same interval.

Combined leg compression and dependency resulted in toe perfusion values in bandaged and control limbs which were insignificantly different from each other. In fact, during dependency the paired–leg perfusion ratios (bandaged/control) were the same as during the baseline interval. These bandage effects were obtained at an average sub–bandage pressure 41.8 ± 2.0 mm Hg which is close to that used and/or recommended.[1,2,25–27]

Previous studies in which sustained regional cuff–compression (40 mm Hg) of the gaiter area was used to evaluate sub–cuff and distal microcirculatory effects[14] showed similar effects on the foot dorsum which was distal to the compression. Blood perfusion was noted to decrease by about 35 percent which is close to the 35.8 percent

decrease in toe perfusion here reported. Contrastingly, whereas cuff compression also caused a decrease in sub–cuff perfusion, sub–bandage perfusion was not reduced with the present fore–foot–to–knee bandaging. This difference suggests that perfusion at sites distal to compression are generally reduced at pressure levels near 40 mm Hg. It also suggests that the way compression is applied can significantly affect perfusion at the compression sites.

Ankle–knee bandaging studies showing a significant reduction in toe pulse amplitude at proximal pressures greater than 30 mm Hg[28] are consistent with the present microcirculatory findings. Other work showing that compression bandaging increases leg arterial pulsatile blood flow[10,11] may indicate the presence of a reflex arteriolar vasodilation in response to the external pressure. Because these flow changes occur in the bandaged region but not distally, the present finding of a differentially directed response distally as compared with sub–bandage may partly be explained on this basis.

The finding that compression bandaging appears to reduce the sub–bandage and distal responses to leg dependency is also interesting. Normally, leg dependency is associated with a significant decrease in distal limb skin blood perfusion due to arteriolar vasoconstriction.[29–32] The shift in blood volume with leg dependency is thought to be the myogenic stimulus triggering this response which serves to partially buffer the capillary network from pressure overload. Recent work showed that the vasoconstriction is maintained even after 40 minutes of sustained leg dependency[33] although there is some evidence of a deficit in diabetes.[34] The reason that the bandaging reduces this vasoconstrictor response may be because it restricts the blood volume shift to the dependent limb.

The fact that compression bandaging may lead to a blood flow decrease distal to the bandaged region may have clinical implications for patients with compromised lower extremity circulations since at least 20 percent of patients with lower extremity ulcers have mixed venous and arterial components.[5] However, because the present data is strictly applicable to healthy limbs, induced perfusion deficits that might accompany compression bandaging of limbs with arterial disease is not yet known. Furthermore, although the reduction in blood perfusion here reported does not necessarily signal an ischemic risk it raises the possibility that monitoring toe perfusion to detect changes subsequent to bandaging may be a useful indicator of impending problems. It is not yet clear if such measurements would best be made in the supine or leg dependent position or what level of microvascular perfusion reduction would be an acceptable threshold. However, the findings do reinforce the need for caution with respect to therapeutic compression levels in patients with reduced vascular function and emphasize that benefits of compression therapy need to be weighed with respect to overall risks. Minimum vascular assessments using ankle–brachial pressure indices (ABI) are now well accepted procedures, with ABI values ranging from 0.8 to 0.9 used to exclude patients from compression or raise cautionary flags.[1–4,35–37] Prudent application of these concepts together with good clinical judgement as well as use of other indicated and available vascular tests are important defenses against injurious compression related effects. However, since even in the vascularly normal subjects supine blood perfusion was diminished more than 35 percent on the average and in some subjects significantly more, ABI screening may not always be adequate. An adjunctive procedure of selective perfusion monitoring may assist in assessing patient–by–patient compression effects to help guide the choice of appropriate compression levels.

Conclusions

Compression bandaging as here used does not reduce sub–bandage microvascular blood perfusion but does cause a decrease in blood perfusion distal to the compression when supine. The absence of a perfusion decrease under the bandage may be related to a partially compensating reflex vasodilatory response, seen previously as an increase in arterial pulsatile flow. However, such possible flow increases are inadequate to compensate the reduced distal microcirculation which is reduced by about 35 percent. When the legs are in a dependent position, however, there is no longer a difference in toe perfusions between bandaged and non–bandaged legs. The equivalence of distal perfusion under dependent but not supine conditions is likely explained by a blunting of the normal vasoconstrictor response to leg dependency by the bandage.

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