Compression Bandaging Effects on Lower Extremity Peripheral and Sub–Bandage Skin Blood Perfusion

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Abstract: Laser–Doppler blood perfusion was simultaneously measured on both great toes and the lateral upper–calf before and during fore–foot–to–knee compression bandaging of one test–leg in ten vascularly healthy female volunteers. Two bandaging methods were sequentially used separated by a 30 minute interval. Bandage A consisted of a layer of zinc impregnated gauze and an elastic wrap; bandage B had the elastic wrap only. Sub–bandage pressures of the test–leg were measured at distal and proximal lateral below–knee standardized sites. The study purpose was to determine the effects of moderate compression pressure levels on skin microcirculation under and distal to bandaged regions. Initial (mean ± sem) sub–bandage pressures achieved for bandages A and B were similar, being respectively 32.9 ± 2.8 and 28.4 ± 3.9 mm Hg. Both bandage types were associated with significant reductions in test–leg toe blood perfusion amounting to 44.2 ± 13.1 percent and 27.5 ± 10.5 percent for bandages A and B respectively. Contrastingly, test–leg sub–bandage blood perfusion did not differ from its pre–bandage baseline mean level for either bandage type. These findings show that a widely used bandaging method and a slight variant each significantly reduces distal (toe) blood perfusion without reducing sub–bandage skin perfusion. Absence of sub–bandage perfusion decreases may be related to a partially compensating reflex vasodilatory response, but such effects if present are inadequate to prevent reductions in distal perfusion. These results reinforce the need for due care and risk–benefit considerations with respect to therapeutic compression levels.

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Introduction

Compression bandaging is a mainstay for treating venous ulcers although there is controversy as to the most efficacious level of sub–bandage pressure for timely ulcer healing.[1–3] Depending on bandage system type, sub–bandage pressures ranging from about 20 to 45 mm Hg can be imparted to the underlying tissue. Because at least 20 percent of patients with venous ulcers may also have coexisting lower extremity arterial disease,[4] the issue of the extent to which compression modifies microvascular blood perfusion is of considerable clinical interest. Though pressures high enough to compress

arteriolar blood vessels can produce ischemic effects on dependent tissue, plethysmographic measurements of mean blood flow under inflatable plastic splints suggest reductions at pressures as low as 30 to 40 mm Hg,[5] and compression to levels well below arterial pressures show decreases in leg mean flow.[6–8] Contrastingly, lower extremity compression, produced by elastic bandaging at these[9] and even lower[10] sub–bandage pressures, cause significant increases in leg pulsatile blood flow which is especially profound at more proximal below–knee sites. Recent work showed that using a four–layer bandaging system, which produced mean sub–bandage pressures near 40 mm Hg, caused significant decreases in lower extremity peripheral blood perfusion on non–compressed tissue as measured on toes.[11,12] However, it is unknown if such skin blood perfusion changes were unique to the specific bandage system used or were related either to the high–end sub–bandage pressures achieved or to yet other factors. The present study was undertaken to clarify these issues by using a different but widely used fore–foot–to–knee bandaging method (zinc impregnated gauze + one layer of elastic wrap) and a slight variant (elastic wrap alone). Laser–Doppler blood perfusion responses to both bandage types were sequentially assessed by simultaneous bilateral great toe and test leg sub–bandage perfusion measurements in ten vascularly normal female volunteers.

Methods

Subjects and preliminary evaluations. Healthy female volunteers (N = 10, age 42 ± 3.3 years) were evaluated 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[13–17] and ankle–brachial systolic pressure indices (ABI) obtained using standard Doppler ultrasound at the posterior tibia and dorsal pedis arteries. All subjects tested were well within normal ranges. Blood pressures measured with standard pressure cuffs also verified

that the group was normotensive (systolic 110 ± 4.5, diastolic 66 ± 2.7 mm Hg).

Leg compression bandaging. Leg compression was produced first by wrapping one leg from fore–foot to knee with a widely used bandaging method. Bandage A, Tegapore™ dressing (3M Health Care, St. Paul, MN), was applied to the lateral supramalleolar region over which was placed one 4" x 4" cotton gauze which was folded in half. A zinc oxide coated gauze bandage (Viscopaste®, Smith & Nephew United, Inc., Largo, FL) was then wrapped around the limb, from fore–foot to knee using a spiral wrap. The final layer, which was the main pressure inducing component was Coban™, 3M Health Care, St. Paul, MN), wrapped at mid–stretch again from fore–foot to knee. The second procedure, denoted as bandage B, used a slight variant which differed from bandage A only by the elimination of the zinc gauze wrap component. Bandaging was done by the same wound care nurse who had extensive experience with the use of these bandage methods.

Initial preparatory sequence. At approximately 11:00 hours the subject assumed a supine position on an exam table in a testing laboratory which is temperature controlled to between 22 and 23° C. The leg length (L) between the lateral malleolus and the tibial turburcle was measured and recorded, and leg circumferences at 25 percent L and 75 percent L were measured. For the present group these circumferences were (mean ± sem) 23.3 ± 0.24 and 34.9 ± 0.27 cm respectively. A laser–Doppler probe (Vasamedics P–440 Soflex probe, Vasamedics Inc., St. Paul, MN) was placed on the lateral aspect of the left leg with the active sensing portion of the probe positioned at a standardized site. The location was determined in each subject by placing it at a distance from the lateral malleolus equal to 75 percent of the malleolus–knee length. The circumferential position was at or near to the center of the lateral 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. To monitor distal skin blood perfusion, two additional laser–Doppler probes

(P7 large area probes, Moor 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 Instruments, MBF3D, Instruments Ltd., UK). Laser–Doppler blood perfusion data, obtained from the distal toe sites and sub–bandage, represent signals obtained from a depth of about one 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 k HZ; 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.[18–20] Prior to the bandaging procedure, two interface pressure sensors connected to an automated pressure system (Talley Oxford Pressure Monitor, MKII, Progressive Medical, Lansing, MI), previously calibrated, were placed in contact with the skin on the lateral aspect, one under the Tegapore™ (at 25 percent L which is approximately ten cm proximal to the lateral malleolus) and the other about two cm distal to the sub–bandage laser–Doppler probe at the 75 percent L site. These sensors were used to measure sub–bandage pressures. The pressure registered by each sensor (which represents the counter compression pressure within the sensors' air–filled enclosure) was recorded in triplicate, and the average of each of the three readings from each sensor was 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, separate tests of the pressure sensors during calibration procedures, in which the sensors were placed on a cylindrical tube and compressed at known pressure levels using a blood pressure cuff, showed that sensor pressures were within ± 2 mm Hg of cuff pressure. It is also possible that the slight elevation produced by the placement of a 4 x 4 over the sensor might slightly increase pressure as measured by the sensor. However, as the purpose of the 4 x 4 was to partially simulate conditions of ulcer treatment, any such pressure elevation would also be present during such treatment.

Protocol. After completion of the preparatory sequence the subject was covered with a light blanket and the feet were tented by a sheet so as to diminish effects of environmental variables (room temperature, drafts, if any, etc.) on skin temperature and possibly blood flow between legs. The subject was left undisturbed for 15 minutes. At the conclusion of this rest interval, the first baseline blood perfusion recording was begun which consisted of simultaneous and continuous recordings of both toes and lateral leg perfusions for 10 minutes. The test leg was then bandaged (bandage A) as previously described and the contralateral control leg was similarly manipulated and a Kling™ (Johnson & Johnson Medical, Inc., Arlington, TX) non–compressive bandage used as a sham control wrap. Test–leg sub–bandage pressures were then recorded. Fifteen minutes after bandaging, the laser–Doppler blood perfusion measurements were repeated and followed by a second series of sub–bandage pressure measurements at about 30 minutes after its initial application. The bandage was then removed and the subject remained resting for an interval of 15 minutes. This entire sequence was then repeated using bandage B.

Analyses. Laser–Doppler data was analyzed by first computing the average skin blood perfusion during each of the following 10 minute data acquisition intervals: 1) baseline for bandage A, 2) with bandage A in place for 15 minutes, 3) baseline for bandage B and 4) with bandage B in place for 15 minutes. A data acquisition interval of 10 minutes was chosen so that·normal short–term physiological variations in blood perfusion could be suitably averaged. All laser–Doppler perfusion measurements were analyzed using raw values expressed in arbitrary units (a.u.) as is standard practice. For the non–compressed distal toe measurements, pre–bandage (baseline) and bandaged perfusion values were compared for each leg separately for each bandage type, and paired–leg perfusions (bandaged leg vs. control leg) were compared during corresponding protocol intervals for bandages A and B. Sub–bandage perfusions were compared on the basis of pre– and post–bandaging for each bandage type separately. Sub–bandage pressures were compared with respect to initial and follow–up measurements at each of the two measured sites for each bandage type. Statistical analyses were done using the Wilcoxon non–parametric test with a p–value <
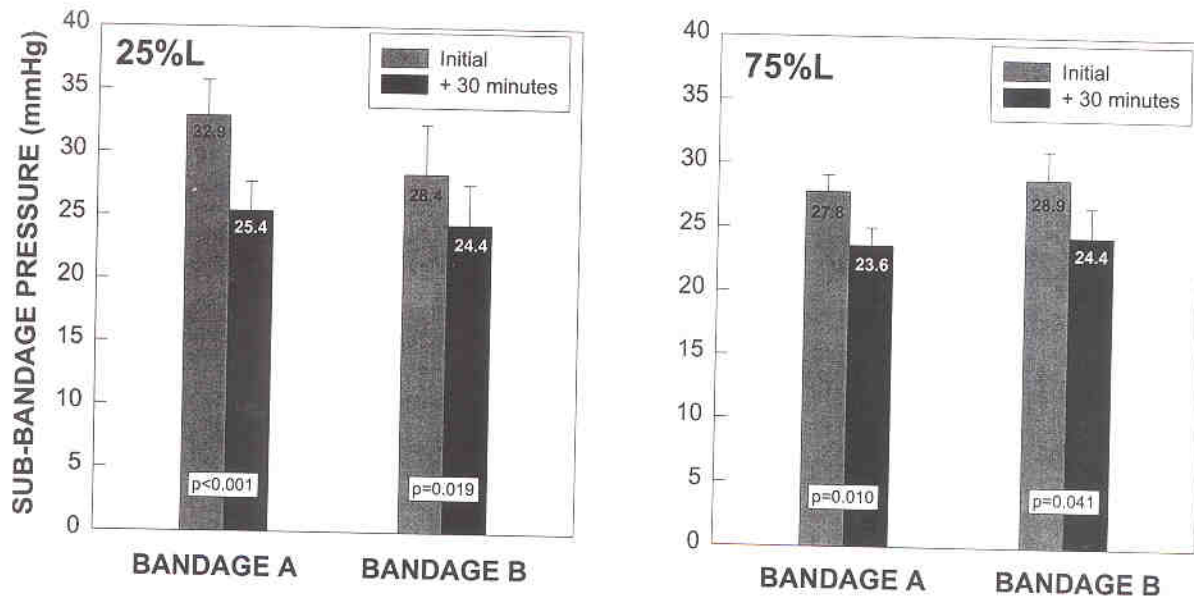
Figure 1. _Sub–bandage pressures._ Pressures measured under bandage A and bandage B when initially applied and 30 minutes later. Whether measured at the distal leg site (25 percent L) or proximal leg site (75 percent L) the sub–bandage pressures were significantly reduced after 30 minutes with both bandage types. There were no significant differences between bandage types in the mean initial or follow–up pressures. L is the distance between the lateral malleolus and the knee.

0.05 being accepted as statistically significant. All data within the text is reported as mean ± sem, unless otherwise indicated.

Results

Sub–bandage pressures. Applications of bandages A and B (Figure 1) produced initial mean sub–bandage pressures of 32.9 ± 2.8 and 28.4 ± 3.9 mm Hg respectively at the 25 percent leg site and 27.8 ± 1.3 and 28.9 ± 2.2 mm Hg at the 75 percent leg site. Initial pressures produced by bandages A and B did not significantly differ at either site. Follow–up pressure measurements about 30 minutes after bandaging showed that initial pressures at each site and for each bandage type were significantly reduced. At the 25 percent site sub–bandage pressures were reduced to 25.4 ± 2.3 and 24.4 ± 3.2 mm Hg for bandages A and B respectively; at the 75 percent site corresponding pressures were reduced to 23.6 ± 1.4 and 24.4 ± 2.3 mm Hg. Follow–up pressures did not significantly differ.

Peripheral perfusion effects. As illustrated in Figure 2, prior to bandage A application, baseline toe skin blood perfusions of test and control legs were not significantly different (115.9 ± 30.7 vs.

132.0 ± 39.8 a.u. respectively). Bandage A application was associated with a significant decrease in test leg toe perfusion to 63.5 ± 24.1 a.u., $p = 0.016$ and a slight but insignificant decrease in the control toe perfusion to 96.3 ± 30.5 a.u., $p = 0.169$. Although baseline toe perfusions prior to application of bandage B did not differ significantly from each other (65.3 ± 29.8 and 88.6 ± 21.9 a.u.), both tended to be reduced as compared with their initial baselines. The baseline perfusion reduction as measured prior to applying bandage B to the test leg was highly significant ($p = 0.012$) whereas the baseline difference for the control leg was not significant ($p = 0.571$). After application of bandage B, there was a further significant decrease in the test leg toe perfusion to 40.3 ± 18.1 a.u., $p = 0.028$, but no significant reduction in control toe perfusion (81.0 ± 28.3 a.u.). With bandage B in place, test toe perfusion was significantly less than the control toe ($p = 0.028$). Expressed in terms of percentage changes from corresponding baselines, application of bandage A to the test leg produced a toe perfusion decrease of 44.2 ± 13.1 percent, and bandage B resulted in an overall reduction of 27.5 ± 10.5 percent. When bandage A was used, nine of the tested subjects demonstrated a decrease which ranged from 14.7 to 85.4 percent
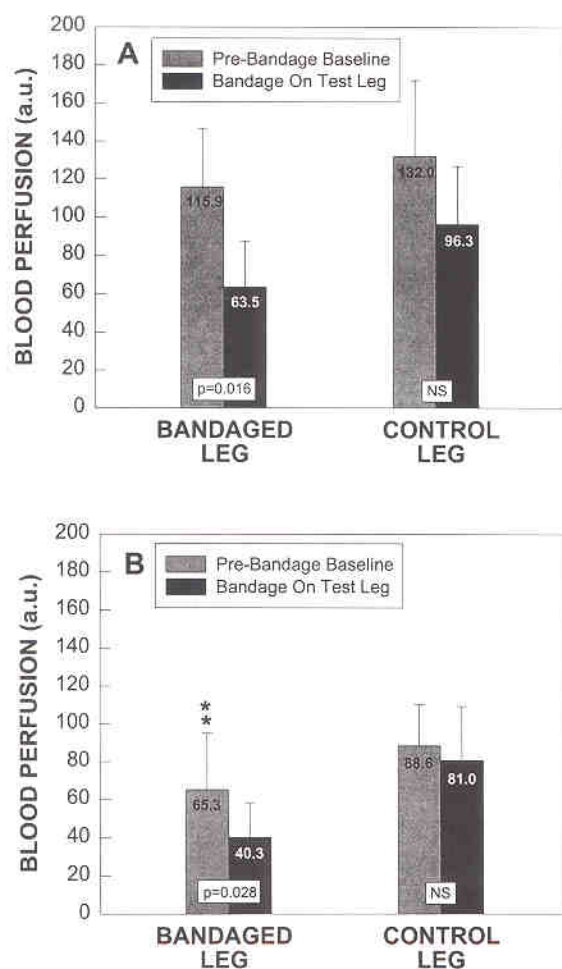
*Figure 2. Impact of compression bandaging on toe blood perfusion. Panels A and B correspond to laser–Doppler blood perfusion measured on the great toe pulp using bandage A and bandage B respectively. Both bandage types caused a significant decrease in the toe blood perfusion of the bandaged leg. ** denotes a significantly decreased pre–bandage B baseline perfusion of the test–leg toe as compared with its value prior to bandaging with bandage A.*

(53.9 ± 8.6 percent, n = 9). When bandage B was used, perfusion decreased in eight of the tested subjects with the reductions ranging from 9.1 to 67.7 percent (40.5 ± 6.2 percent, n = 8).

Sub–bandage perfusion effects. Neither bandage A nor bandage B resulted in any significant sub–bandage perfusion changes as illustrated in Figure 3. Pre– and post–bandage group mean perfusions were identical for bandage A (0.40 ± 0.06 vs. 0.40 ± 0.08) and very close for bandage B (0.35 ± 0.05 vs. 0.33 ± 0.05).

Discussion

A primary goal of the present study was to determine if previously observed[11,12] peripheral blood perfusion reductions secondary to compression bandaging were bandage–system unique and/or dependent upon associated high–end sub–bandage pressures [41.8 ± 2.0 mm Hg]. The present findings show that significant distal blood perfusion reductions are present even at considerably lower sub–bandage pressures which, at (lateral) distal leg sites comparable to those previously measured, ranged from 32.9 to 28.4 mm Hg for the two different bandage methods here employed. The associated distal (toe) perfusion mean reductions of 44.2 and 27.5 percent bracket the previously measured reduction of 35.6 percent, thereby indicating a rather robust response over this sub–bandage pressure range. The fact that different skin contact materials and different numbers of bandage layers produce similar reductions in distal perfusion suggests that these components have small impacts on perfusion decrement differences.

Another interesting aspect emanating from the present study was the demonstration that in spite of significant peripheral blood perfusion reductions, test leg sub–bandage perfusion was essentially unaffected by either bandage system used. With regard to this finding, it should be noted that since two different laser–Doppler systems were used (one a dual–channel for the toes and the other a single–channel for sub–bandage perfusions), comparisons or inferences regarding the absolute perfusions (in a.u.) registered by the different units should not be made because the effective signal gains may be different. In the present study, for example, sub–bandage perfusions (before compression) are generally less than 1.0 a.u. whereas toe perfusions are greater than 100 a.u. This wide difference is due in part to the naturally occurring much higher flow at the toe–pulp and in part to different system sensitivities and settings. For the sub–bandage measurements the ability of the system to detect perfusion reductions, if and when present, was verified using suprasystolic thigh cuff compressions which invariably reduced the perfusion to zero. Thus the low (relative to toe) values registered by the sub–bandage perfusion monitoring system are well within the range necessary to detect sub–bandage perfusion changes if present.
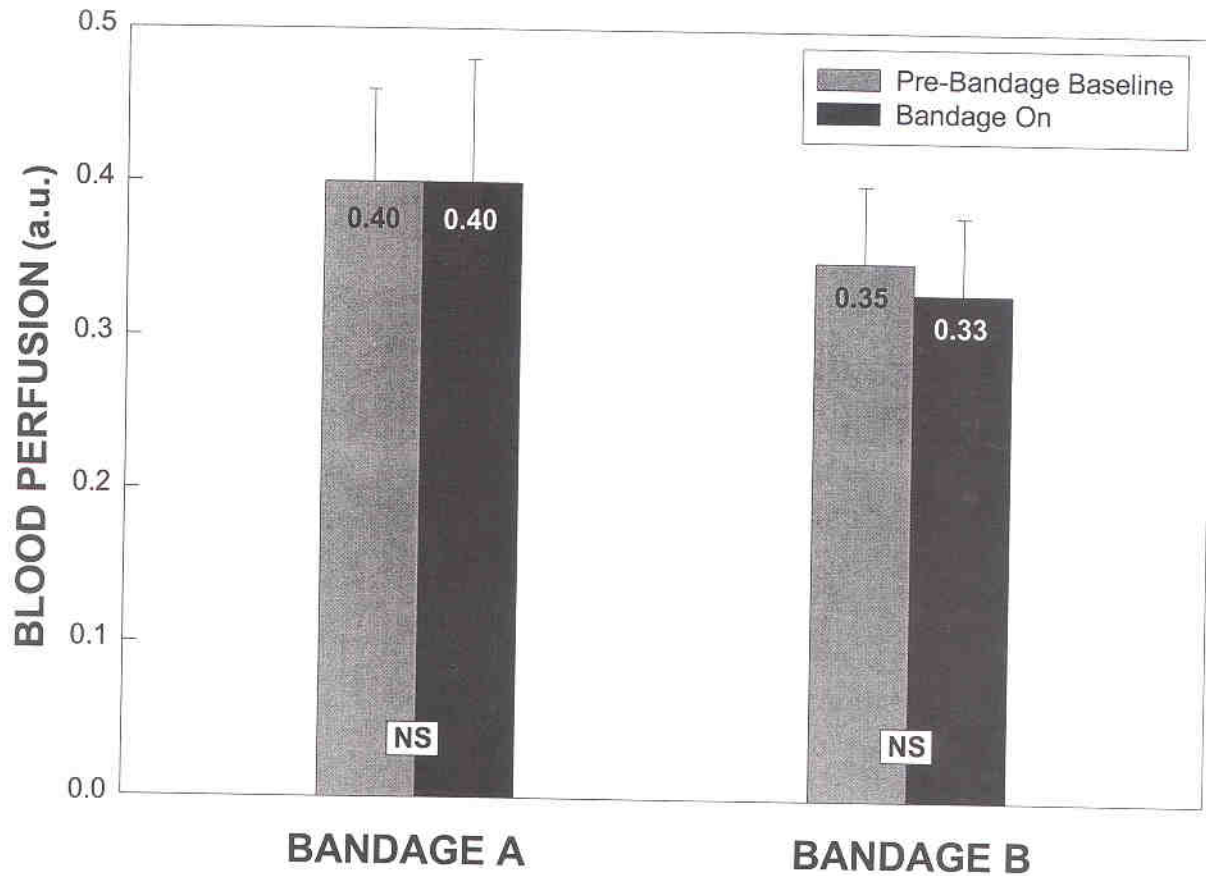
Figure 3. _Sub–bandage blood perfusion._ Neither bandage A nor bandage B produced a significant decrease in sub–bandage blood perfusion.

Previous work in which supine sub–bandage perfusion was measured at the medial supra–malleolar region also showed an absence of a significant decrease in leg skin blood perfusion.[11] Direct comparison of the present findings with these is not appropriate since in the present study laser–Doppler perfusion was measured at a proximal lateral–leg site. This location was expressly chosen based on recent data indicating that arterial pulsatile blood flow, secondary to compression bandaging, is significantly augmented there.[9,10] Although corresponding skin blood perfusion increases were not found in the present study it is possible that the absence of sub–bandage perfusion decreases, as one might expect to have occurred, are influenced by sub–bandage augmented arterial pulsatile flow. Thus based on the facts that distal but not sub–bandage perfusion is significantly reduced but sub–bandage arterial flow is increased, it has been speculated that compensatory sub–bandage arteriolar vasodilation may partially off–set mechanical effects which do impact on distal, non–compressed tissue.

The present results regarding ankle–knee bandaging are consistent with other studies which have shown significant reductions in toe pulses at proximal pressures greater than about 30 mm Hg[21] and with findings based on proximal regional leg compression effects.[22] As previously noted, the fact that compression bandaging, even at the nominal sub–bandage pressures here used, may lead to a blood flow decrease distal to the bandaged region, may have clinical implications, especially with regard to patients with compromised lower extremity circulation estimated to be at least 20 percent.[4] However, the present data is strictly applicable to healthy limbs, and induced perfusion decrements that might accompany compression bandaging of limbs with arterial disease are unknown. However, as recently pointed out, such findings reinforce the need for due care

and risk–benefit considerations with respect to therapeutic compression levels in patients with reduced vascular function.[11]

Summary

Compression bandaging with a widely used bandaging method and a slight variant significantly reduces distal (toe) blood perfusion but does not reduce sub–bandage proximal below–knee skin perfusion. Absence of perfusion decreases under the bandage may be related to a partially compensating reflex vasodilatory response, seen previously as an increase in arterial pulsatile flow at sub–bandage pressures ranging from about 25 to 40 mm Hg. Though such possible flow increases may be adequate to compensate sub–bandage effects, they are inadequate to prevent reductions in distal perfusion ranging from about 45 to 27 percent.

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