

Compression therapy in mixed ulcers increases venous output and arterial perfusion

Giovanni Mosti, MD,^a Maria Letizia Iabichella, MD,^a and Hugo Partsch, MD,^b *Lucca, Italy; and Vienna, Austria*

Objectives: This study was conducted to define bandage pressures that are safe and effective in treating leg ulcers of mixed arterial-venous etiology.

Methods: In 25 patients with mixed-etiology leg ulcers who received inelastic bandages applied with pressures from 20 to 30, 31 to 40, and 41 to 50 mm Hg, the following measurements were performed before and after bandage application to ensure patient safety throughout the investigation: laser Doppler fluxmetry (LDF) close to the ulcer under the bandage and at the great toe, transcutaneous oxygen pressure (TcPO₂) on the dorsum of the foot, and toe pressure. Ejection fraction (EF) of the venous pump was performed to assess efficacy on venous hemodynamics.

Results: LDF values under the bandages increased by 33% (95% confidence interval [CI], 17-48; $P < .01$), 28% (95% CI, 12-45; $P < .05$), and 10% (95% CI, -7 to 28), respectively, under the three pressure ranges applied. At toe level, a significant decrease in flux of -20% (95% CI, -48 to 9; $P < .05$) was seen when bandage pressure >41 mm Hg. Toe pressure values and TcPO₂ showed a moderate increase, excluding a restriction to arterial perfusion induced by the bandages. Inelastic bandages were highly efficient in improving venous pumping function, increasing the reduced ejection fraction by 72% (95% CI, 50%-95%; $P < .001$) under pressure of 21 to 30 mm Hg and by 103% (95% CI, 70%-128%; $P < .001$) at 31 to 40 mm Hg.

Conclusions: In patients with mixed ulceration, an ankle-brachial pressure index >0.5 and an absolute ankle pressure of >60 mm Hg, inelastic compression of up to 40 mm Hg does not impede arterial perfusion but may lead to a normalization of the highly reduced venous pumping function. Such bandages are therefore recommended in combination with walking exercises as the basic conservative management for patients with mixed leg ulcers. (*J Vasc Surg* 2012; 55:122-8.)

About 15% to 30% of patients with venous leg ulcers (VLU) have concomitant arterial disease presenting with a reduced ankle-brachial pressure index (ABPI).¹⁻⁵ In these patients, compression is still a controversial issue. On the one hand, compression is able to improve venous hemodynamics and reduce edema, but on the other hand, it could potentially restrict arterial perfusion. This is the reason why, in clinical studies on compression in VLU, an ABPI <0.8 is usually considered an exclusion criterion.⁶⁻¹⁰ In a recent Cochrane review on compression in VLU,⁸ an ABPI <0.8 was considered an exclusion criterion for compression therapy in 23 of 33 studies. Clinical studies^{1,10} and guidelines^{11,12} recommend “reduced,” “modified,” or “supervised modified” compression, with reduced pressure, in patients with arterial impairment and an ABPI of 0.5 to 0.8; however, sub-bandage pressures that can safely be used are not defined.^{1,10-14}

The aim of our work was to define a range of compression pressures that do not impair arterial flow but improve venous hemodynamics in patients with mixed leg ulcers.

MATERIAL AND METHODS

The study recruited 25 patients (10 men, 15 women) with mixed ulceration who were aged 76.3 ± 5.9 years (range, 62-80 years). Inclusion criteria were individuals aged between 18 and 80 years, affected by leg ulcers of proven arterial and venous pathophysiology, an ABPI between 0.5 and 0.8, systolic pressure at ankle level ≥ 60 mm Hg, systolic toe pressure >30 mm Hg, and axial venous reflux in the superficial or deep veins, or both, of the lower extremity. Toe pressure was measured to avoid a potential misdiagnosis due to severe media sclerosis because this parameter is much less influenced by this problem. The lowest value in our series was 32 mm Hg. Exclusion criteria were patients aged <18 years or >80 years, an ABPI <0.5 or >0.8, an ankle pressure <60 mm Hg, pain at rest, sensory loss (neuropathy), cardiac insufficiency, and media calcinosis.

Duplex ultrasound imaging (Esaote MyLab 60 Gold with linear probe 7.5-10 MHz; Esaote Spa, Genoa, Italy) was used to assess reflux >1-second duration after flow augmentation in the upright position in superficial and deep vein segments leading to the ulcer¹⁵ and to localize arterial occlusions. Ankle and arm pressures were measured by continuous-wave Doppler (PicoDop; Micro-labitalia, Padua, Italy), and ABPI was calculated by di-

From the Angiology Department, MD Barbantini Clinic, Lucca^a; and Private practice, Vienna.^b

Competition of interest: none.

Presented at the Twenty-third Annual Meeting of the American Venous Forum, San Diego, Calif, February 23-26, 2011.

Reprint requests: Dr Giovanni Mosti, Clinica Barbantini, Reparto di Angiologia, Via del Calcio 2, 55100 Lucca, Italy (e-mail: jmosti@tin.it).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a competition of interest.

0741-5214/\$36.00

Copyright © 2012 by the Society for Vascular Surgery.

doi:10.1016/j.jvs.2011.07.071

viding the higher pressure at ankle level (examining anterior or posterior tibial artery) of the affected leg by the higher pressure of the two arms measured simultaneously.

Patients were informed of the study details and gave their informed consent to be enrolled in the study. Ethical committee approval was not required because all procedures were noninvasive, of short duration, were performed under the surveillance of doctors, and with Conformité Européenne-marked materials and devices that have been used in clinical practice for many years.

Measurements. All measurements were performed in the morning in a quiet room with a constant temperature of $\sim 22^{\circ}$ at baseline and after the application of a multicomponent, multilayer bandage with high stiffness at different pressure ranges. The measurements were performed with the participant supine with intervals of 10 minutes after application of each bandage; the interval from application of one bandage strength to another was 30 minutes. A variety of measurements were obtained.

Laser Doppler fluxmetry (LDF) was measured at the periwound area and distally to the bandage on the plantar surface of the big toe. Two different probes were used for these measurements: a flat probe specially designed not to exert eccentric pressure when compressed by bandages in the periwound area and a standard probe at toe level (Periflux System 5000, Perimed, Järfälla, Stockholm, Sweden). The laser Doppler shift was analyzed, processed, and filtered in real time by an analog processor and transformed in a reproducible electrical signal that varied linearly with the blood flow.¹⁶⁻¹⁹ The LDF is measured in perfusion units (PUs).

Toe pressure was measured by using a pressure device (Perimed PF 5050) with a small toe cuff instantaneously inflated to 250 mm Hg and slowly deflated. Systolic pressure was considered to be the value at which arterial flow detected by the laser Doppler probe reappeared.^{20,21}

Transcutaneous oxygen pressure (TcPo₂) was measured on the dorsum of the foot, distal to the bandage, using a probe heated to 44°C (Periflux System 5000).

Ejection fraction (EF) from the lower leg was measured by strain gauge plethysmography (Angioflow 2; Microlabitalia, Padua, Italy) to quantify the venous pumping function following the method described by Poelkens et al.²² An indium-gallium alloy gauge (diameter of 1 mm) is placed around the leg in the supine position, 5 cm distally from the patella and proximally to the bandage applied with a pressure of 20 to 30 and 31 to 40 mm Hg. The placement of the transducer proximal to the bandage avoids artefacts that would occur by putting the strain gauge over the bandaged calf segment.²²

The investigation starts, after calibration of the device, by elevating the examined leg to empty the veins and record the minimal volume of the leg segment. Then, the patient stands and the volume increase of the calf segment encircled by the strain gauge probe, reflecting venous filling, is measured continuously. Venous volume (VV) is defined as the difference between empty and filled veins. During a

standardized exercise (walking on spot with 20 steps in 20 seconds), the amount of blood that is expelled toward the heart (EV [expelled volume]) reflects the quality of the venous pump. EF, calculated according to the formula $100 \times EV/VV$ is highly reduced in patients with venous incompetence.²³ As demonstrated in previous reports,²²⁻²⁶ this method is able to assess the hemodynamic efficacy of different compression devices in a completely noninvasive way.

Interface pressure of the compression bandage was continuously measured by means of a validated^{27,28} pneumatic compression device (Picopress; Microlabitalia, Padua, Italy), with the pressure probe placed next to the flat laser Doppler probe.

LDF, TcPo₂, and the toe pressure under different levels of compression pressures were considered as safety parameters. A decrease of the baseline TcPo₂ values to <30 mm Hg or of the toe pressure to <30 mm Hg would have fallen below the criteria for critical ischemia²⁹ and would have stopped the experiment. TcPo₂ measurements, which take considerable time for achieving a stable output under the heated probe, were only done before and after compression with 31 to 40 mm Hg, assuming from reported data that this was still a safe pressure range.^{30,31}

EF of the calf muscle pump was taken as a parameter to measure the hemodynamic efficacy of compression.

We applied a stiff bandage composed of an inelastic material, Mollelast Haft (Lohmann & Rauscher, Rengsdorf, Germany), wrapped from the base of the toes to the popliteal area, on top of a padding material (Cellona; Lohmann & Rauscher). Mollelast was wrapped with an overlap of 50% under surveillance of a pressure recorder, adjusting the number of layers to achieve pressures of 20 to 30, 31 to 40, and 41 to 50 mm Hg.

LDF and TcPo₂ measurements were performed at baseline without compression. After placing the pressure probe and the compression device, measurements were repeated at different bandage pressures according to the protocol. TcPo₂ was performed at a pressure range of 31 to 40 mm Hg.

After these measurements, EF was measured at baseline and after application of the bandage exerting a pressure range of 20 to 30 and 31 to 40 mm Hg.

All measuring probes stayed in place during the repeated measurements.

Statistics. Medians with interquartile ranges (IQR) and maximal and minimal values are presented. Mean percentage changes and 95% confidence intervals (CI) show the differences compared with baseline. Repeated measures analysis of variance was used to compare the leg perfusion under the bandage, toe perfusion, and toe pressure. To compare the effect of bandages on the TcPo₂ the nonparametric Wilcoxon test was used. Differences with a *P* value $<.05$ were considered statistically significant. A retrospective power calculation based on our EF measurements showed that the experiments had a 95% power to detect the smallest average differences between pairs with a significance level (α) of 0.05 (two-tailed). The graphs and the

Table. Baseline characteristics of the examined 25 patients

Variable	Value ^a
Age, years	75.9 ± 10.3
Sex	
Male	10
Female	15
Diabetes	5
Smoking	12
Arterial disease	
Iliofemoral	18
Femoropopliteal	7
Venous insufficiency	
Superficial	21
Superficial and deep	4
Claudication	
Walking distance >100 m	9
Walking distance <100 m	16
Ankle arterial pressure, mm Hg	95 (75-115)
Ankle-brachial pressure index	0.58 (0.5-0.65)
Ejection fraction, % ^b	33.95 (26.6-37.5)

^aContinuous data are presented as mean ± standard deviation or median and interquartile range; categorical data are number of patients.

^bEjection fraction % is calculated according to the formula: $100 \times EV/VV$.

statistical evaluations were generated with Prism 5 software (Graph Pad, San Diego, Calif).

RESULTS

The relevant baseline characteristics of 25 patients enrolled in this study are summarized in the Table. LDF in the periwound skin area increased under external pressure of 20 to 30 mm Hg from a median value of 4 PU (IQR, 3-8.5 PU) by 33% (95% CI, 17%-48%; $P < .01$) and under 31 to 40 mm Hg by 28% (95% CI, 12%-45%; $P < .05$). In the pressure range of 41 to 50 mm Hg, there was still a nonsignificant increase in flux of 10% (95% CI, -7% to 28%; Fig 1).

At toe level, only minor changes in LDF occurred when the leg was compressed. From a baseline value of 65 PU (IQR, 33-147.5 PU), there was an increase of 3% (95% CI, -14% to 20%) with a pressure of 20 to 30 mm Hg and a decrease of 4% (95% CI, -27% to 18%) with a pressure of 31 to 40 mm Hg. Only when the bandage pressure exceeded 41 mm Hg did a significant decrease in flux of 20% (95% CI, -48% to 9%; $P < .05$) occur (Fig 2). Under increasing compression, toe pressure showed a small increase, from a median of 62 (IQR, 50-70) up to 68 mm Hg (IQR 57-75.5 mm Hg) with each pressure step. The mean percent differences are 6% (95% CI, 3%-8%) for the compression range of 20 to 30 mm Hg, 9% (95% CI, 4%-14%) for 31 to 40 mm Hg, and 13% (95% CI, 5%-20%; $P < .001$) for 41 to 50 mm Hg (Fig 3).

Under compression of 31 to 40 mm Hg, the TcPo₂ on the dorsum of the foot, distal to the bandage, showed a small but significant increase of 7% (95% CI, 5%-10%) from 45 (IQR, 40-53) to 49 (IQR, 40.5-55) mm Hg ($P < .001$; Fig 4).

Increasing compression on the venous pump caused an improvement of EF from 33.9% (IQR, 28.1%-36.1%) to 54.3% (IQR 52%-56%), which could be demonstrated by applying pressure of 20 to 30 mm Hg. Under a compression pressure of 31 to 40 mm Hg, EF increased to 62.6% (IQR, 58.6%-69.9%), approaching the normal value of 64.6% (IQR, 63%-71%).¹⁹ This corresponds to a mean increase of 72% (95% CI, 50%-95%; $P < .001$) and 103% (95% CI, 70%-128%; $P < .001$), respectively (Fig 5).

DISCUSSION

Our usual treatment in patients with mixed ulceration who do not fulfill the criteria of critical ischemia²⁹ is to start conservative therapy using supervised modified inelastic bandages and to consider revascularization if the ulcers do not show improvement or healing after several weeks.¹ All patients in this case series tolerated the bandages not only for the short period of the experiments but also when they continued compression treatment following our routine regimen of "supervised modified compression."¹ After bandaging, they are asked to return immediately in case pain increases. Bandages are routinely changed an average of twice weekly.

However, compression using bandages or stockings is still widely considered a contraindication in patients affected by any arterial occlusive disease, even when a concomitant venous disease could benefit from such management. In fact, several guidelines supported more by clinical experience than by clinical outcome data recommend avoiding compression when the ABPI is <0.8 or applying "reduced," "moderate," or "supervised modified" compression to treat mixed ulcers.¹⁰⁻¹⁴

We consider the absolute value of the systolic ankle pressure to be of higher practical relevance than the ABPI because this parameter characterizes the perfusion pressure of the distal leg independent from the systemic blood pressure. Our patients, in fact, presented a low ABPI, often considered a contraindication for compression therapy, simultaneously with a systolic pressure at the ankle >60 mm Hg. Although ABPI is generally used in daily practice to assess the severity of arterial occlusive disease, the absolute values of the ankle pressure give a very clear warning signal when a bandage is applied because it is evident that any sustained external compression pressure should never exceed this perfusion pressure.

Owing to their structural properties, inelastic bandages are sometimes preferred^{12,32} because they provide a comfortable, relatively "safe" pressure during rest and, at the same time, high pressure and efficacy during standing and walking; furthermore, they produce an improvement in the otherwise reduced venous pumping function. Even with relative low pressures²⁴ and after some days of wear, when the initial bandage pressure has dropped,²⁵ inelastic bandages are quite effective in improving the EF of the venous pump. Inelastic, short, stretch materials exert an intermittent compression similar, to some extent, to intermittent pneumatic compression (IPC) delivered by special pumps. For IPC, there is growing evidence of improvement in

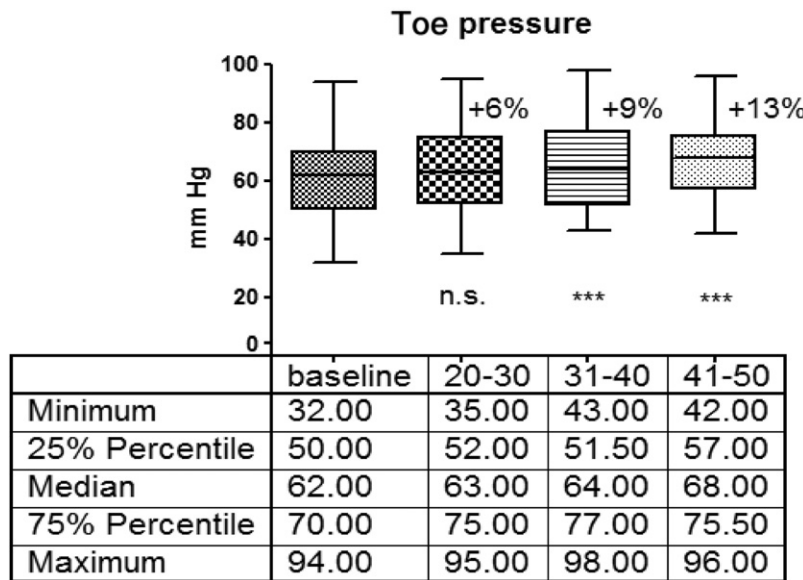


Fig 1. Laser Doppler flux values in periwound skin. Compared with baseline, flux increases under a bandage pressure of 20 to 30 and 31 to 40 mm Hg and starts to decrease only when the pressure exceeds 41 to 50 mm Hg. $**P < .01$; $*P < .05$; n.s., not significant. The *horizontal line* in the middle of each box indicates the median; the *top and bottom borders* of the box mark the 75th and 25th percentiles, respectively, and the *whiskers* mark the 90th and 10th percentiles.

Laser Doppler flux distal to the bandages (toe)

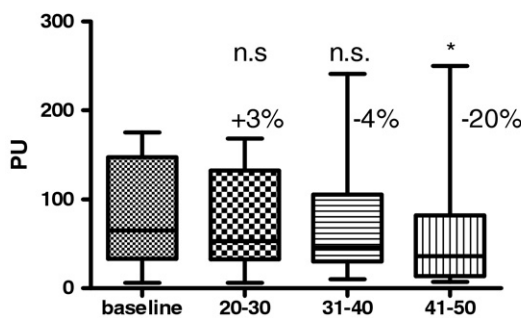


Fig 2. Flux values are slightly reduced by compression at toe level. Only with a bandage pressure >41 mm Hg is flux reduction significant ($*P < .05$). n.s., not significant. The *horizontal line* in the middle of each box indicates the median; the *top and bottom borders* of the box mark the 75th and 25th percentiles, respectively, and the *whiskers* mark the 90th and 10th percentiles.

arterial blood flow and subsequent beneficial clinical effects, even in patients with severe arterial occlusive disease.^{33,34}

In contrast to IPC, little is known regarding the safety and efficacy of inelastic bandages according to the sub-bandage pressure. In our study, we used experimental methods to check the safety and efficacy of inelastic bandages applied with different pressure ranges in patients with mixed ulcers. Long-term effects of compression are beyond the spectrum of this study. We know from previous examinations that the sub-bandage pressure of inelastic bandages will decrease by about 30% after 2 hours, so that a

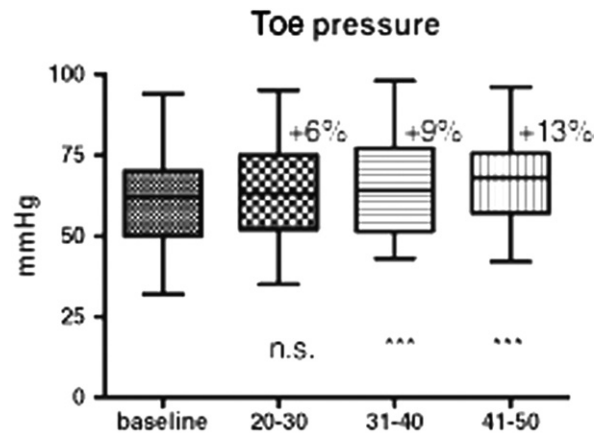


Fig 3. Toe pressure increases slightly with compression of the leg. The increase is not significant at a pressure range of 20 to 30 mm Hg but is significant at a pressure range of 31 to 40 and 41 to 50 mm Hg. $***P < .001$. n.s., not significant. The *horizontal line* in the middle of each box indicates the median; the *top and bottom borders* of the box mark the 75th and 25th percentiles, respectively, and the *whiskers* mark the 90th and 10th percentiles.

negative effect of such bandages on the arterial perfusion starting only after some time can be excluded. Clinical trials have shown that compression bandages applied with moderate pressure and staying on the leg for several days are able to heal mixed ulcers, which takes longer than healing of purely venous ulcers.¹ Safety was assessed by examining blood flow under and distal to the bandage and efficacy by measuring the improvement in venous pumping function.

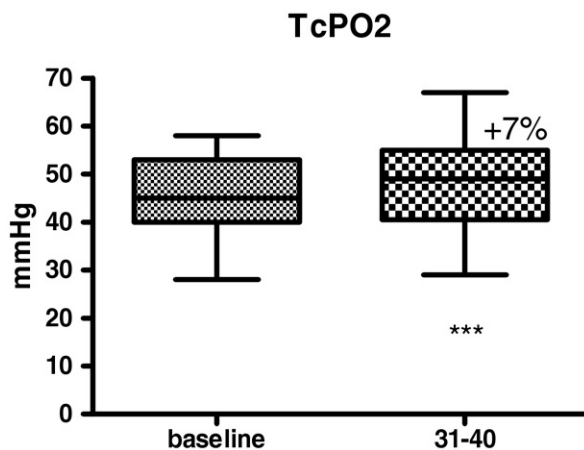


Fig 4. Transcutaneous oxygen pressure ($TcPO_2$) on the forefoot increases significantly with a proximal bandage exerting 30 to 40 mm Hg. *** $P < .001$. The horizontal line in the middle of each box indicates the median; the top and bottom borders of the box mark the 75th and 25th percentiles, respectively, and the whiskers mark the 90th and 10th percentiles.

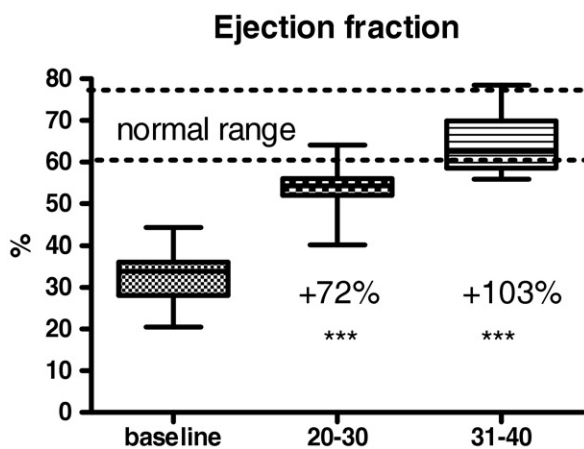


Fig 5. Ejection fraction increases significantly with inelastic compression exerting a pressure of 20 to 30 mm Hg. Normal values can be reached even with pressures between 31 and 40 mm Hg. *** $P < .001$. The horizontal line in the middle of each box indicates the median; the top and bottom borders of the box mark the 75th and 25th percentiles, respectively, and the whiskers mark the 90th and 10th percentiles.

LDF was used to study the effect of a bandage on the arterial perfusion underneath and distal to the bandage. Although this method gives us only a restricted insight into the microcirculation in a pinpoint skin area, changes in the LDF, measured in PU, can be used at least as a determinant of safety, excluding a decrease in the limb's arterial perfusion under and distal to the bandage. When measuring LDF, it is important to notice that the perfusion values are proportional to the product of amount and speed of blood cells moving in the subpapillary vascular plexus mainly from

the deeper, thermoregulatory microvessels and minimally (5%-10%) from the nutritional capillaries.³⁵ Furthermore, LDF does not allow the differentiation of healthy individuals from patients with arterial occlusive disease. This is especially true for acral regions, such as the toes, which contain large amounts of shunting vessels and therefore should be kept in mind when analyzing data. However, a linear correlation has been shown between the volume fraction of moving blood cells in the tissue and the LDF output, which suggests that the measured percentage changes can be taken as an indicator for an increase or a decrease of flow in the investigated skin area.¹⁶⁻¹⁸

On the basis of our measurements distal to the bandage (LDF at toe level, toe-pressure and $TcPO_2$ on the dorsum of the foot), inelastic compression up to a pressure of 40 mm Hg may be considered safe because it does not cause any negative effects on the arterial perfusion. Surprisingly, we even observed an increase of LDF under compression in the periwound skin. The highest increase, up to 33%, was measured under a moderate bandage pressure of 20 to 30 mm Hg, but LDF was also significantly increased by 28% with a compression pressure in the higher range of 31 to 40 mm Hg. These findings are in agreement with LDF data under compression reported by Abu-Own et al.^{30,36} They also showed a similar flux increase in skin areas of the distal lower leg up to a pressure of 50 to 60 mm Hg, both in healthy individuals and in patients with lipodermatosclerosis, in the sitting position.

These findings pointing to a local increase of arterial perfusion by using LDF are in accordance with results that were obtained by measuring the global pulsatile flow of the leg by using nuclear magnetic resonance flowmetry. In two studies of healthy participants, Mayrovitz et al^{31,37,38} were able to demonstrate an increase of arterial perfusion under stiff bandages exerting a mean pressure in the supine position of 28 and of 40 mm Hg, respectively.

LDF in the great toe revealed a moderate reduction in the flux values with increasing sub-bandage pressures being significant in the pressure range of 41 to 50 mm Hg. Mayrovitz et al³⁹ reported similar results in healthy volunteers. These findings cannot be taken as a sign of severe arterial perfusion reduction to the foot, because our toe pressure measurements demonstrate, with increasing sub-bandage pressure, a slight increase of toe pressure by 6% to 13%.

The phenomenon that there is even a blood flow augmentation under sustained compression is difficult to understand. Mayrovitz and Larsen³¹ hypothesize that arteriolar vasodilation is responsible for this phenomenon. Arteriolar vasodilation would be induced myogenically, by reduced transmural pressure, or by vasodilatory substances release triggered by increased venous shear stress. In our investigation, an increase of arterial LDF in the calf was also observed in the resting position under sustained compression.

In contrast to LDF, measurement of the toe pressure is a reliable and generally accepted method to assess arterial macrocirculation and to quantify the severity of arterial

occlusive disease. The obtained values depend on the size of the toe cuff and are always lower than the systolic ankle pressure.^{21,40} Values <30 mm Hg characterize critical ischemia.²⁹ The maintenance and even the slight increase of pressure at toe level under increasing compression are in agreement with data from 33 limbs with mixed ulceration reported from Top et al⁴¹ showing no significant difference between toe pressures before and after application of short stretch bandages. Together with our data, all the reported studies have clearly disproved the widespread theoretic prejudice that sustained compression would always reduce arterial perfusion.

Besides determining safety, the second objective of our investigations was to establish the efficacy of compression by demonstrating an improvement of venous return during movement.

Under normal conditions, one important mechanism for the augmentation of physiologic arterial blood flow during walking is the emptying of the venous blood from the leg, leading to a pressure reduction in normal peripheral veins and an increase in the arteriovenous pressure gradient. This physiologic pressure fall in peripheral veins does not occur in patients with severe venous incompetence, a situation referred to as “ambulatory venous hypertension.” It may be assumed that one important factor for the poor healing of venous leg ulcers in general (in contrast to wounds in other localizations) is the decrease in tissue perfusion due to this reduced arteriovenous pressure gradient caused by ambulatory venous hypertension. In patients with mixed ulcers, additional arterial impairment will worsen this situation.

Therefore, the reported findings of a severely reduced venous pumping function in such patients and its dramatic improvement by inelastic compression seem of utmost importance. As we could demonstrate, the decreased EF of the venous pump, which is in the same order as that in venous patients without arterial occlusions,²³ can even be normalized by applying an inelastic bandage with a pressure between 31 and 40 mm Hg. Therefore, the recommendation to include walking exercises with the use of inelastic bandages for patients with mixed ulcers has now a solid base and reinforces the need to train patients with arterial claudication.

In previous publications “reduced compression,” “supervised, modified compression,”¹ or “moderate compression”⁴² have been recommended as compression treatment for mixed ulcers. On the basis of our data, we propose that these terms imply a pressure range of 30 to 40 mm Hg.

The concept that venous pathophysiology in mixed ulceration may even be more relevant and easier to tackle than the arterial occlusions is also endorsed by recent reports showing beneficial results in ulcer healing by abolishment of venous reflux together with ulcer excision and mesh grafting before or even instead of reopening the arterial occlusions.⁴³

Another mechanism of action to improve arterial flow during walking with an inelastic bandage is the so-called massaging effect. With every muscle contraction, inelastic

bandages produce pressure peaks of 10 to 20 mm Hg higher than the resting pressure.²³ This massaging effect of inelastic bandages may be compared with the massaging cycles of an IPC, for which the release of various vasoactive mediators, such as nitric oxygen and prostacyclin from venular endothelial cells, has been demonstrated.⁴⁴ This effect, which is assumed to have a major effect on flow augmentation by IPC,³⁴ seems also to be important in explaining the beneficial effect of inelastic compression in mixed ulceration.

Both effects of inelastic bandages—the augmentation of arterial flow (mediated by an increased release of vasoactive mediators in the microcirculation together with a reduction of proinflammatory cytokines) and the increase in venous pumping function, leading to a higher arteriovenous pressure gradient—may explain the clinical efficacy of inelastic compression in the healing of mixed ulcers.^{1,45}

One limitation in our study was that only short-term measurements were performed, so that no conclusions may be drawn concerning effects of the applied bandages over a longer period. This and the question concerning the influence of mild compression in purely arterial ulcers should be clarified by future investigations.

CONCLUSIONS

Patients with mixed ulcers presenting a systolic ankle pressure of >60 and toe pressures >30 mm Hg show a significant improvement of reduced venous pumping function when inelastic bandages with a pressure up to 40 mm Hg are applied. In the short-term experiment, an increase of the arterial perfusion of the compressed part of the leg and no deteriorating effect on the areas distal to the bandage could be demonstrated.

AUTHOR CONTRIBUTIONS

Conception and design: GM, HP, MI
Analysis and interpretation: GM, HP, MI
Data collection: GM, MI
Writing the article: GM, HP
Critical revision of the article: GM, HP, MI
Final approval of the article: GM, HP, MI
Statistical analysis: HP, GM
Obtained funding: Not applicable
Overall responsibility: GM

REFERENCES

1. Humphreys ML, Stewart AH, Gohel MS, Taylor M, Whyman MR, Poskitt KR. Management of mixed arterial and venous leg ulcers. *Br J Surg* 2007;94:1104-7.
2. Cornwall JV, Doré CJ, Lewis JD. Leg ulcers: epidemiology and aetiology. *Br J Surg* 1986;73:693-6.
3. Callam MJ, Harper DR, Dale JJ, Ruckley CV. Arterial disease in chronic leg ulceration: an underestimated hazard? Lothian and Forth Valley leg ulcer study. *Br Med J (Clin Res Ed)* 1987;294:929-31.
4. Andersson E, Hansson C, Swanbeck G. Leg and foot ulcer prevalence and investigation of the peripheral arterial and venous circulation in a randomised elderly population. An epidemiological survey and clinical investigation. *Acta Derm Venereol* 1993;73:57-61.

5. Nelzén O, Bergqvist D, Lindhagen A. Venous and non-venous leg ulcers: clinical history and appearance in a population study. *Br J Surg* 1994;81:182-7.
6. Morrell CJ, Walters SJ, Dixon S, Collins KA, Brereton LM, Peters J, et al. Cost effectiveness of community leg ulcer clinics: randomised controlled trial. *BMJ* 1998;316:1487-91.
7. Zimmet SE. Venous leg ulcers: modern evaluation and management. *Dermatol Surg* 1999;25:236-41.
8. O'Meara S, Cullum NA, Nelson EA. Compression for venous leg ulcers. *Cochrane Database Syst Rev* 2009;CD000265.
9. Gohel MS, Barwell JR, Taylor M, Chant T, Foy C, Earnshaw JJ, et al. Long term results of compression therapy alone versus compression plus surgery in chronic venous ulceration (ESCHAR): randomised controlled trial. *BMJ* 2007;335:83.
10. Grey JE, Enoch S, Harding KG. Venous and arterial leg ulcers. *BMJ* 2006;332:347-50.
11. Hopf HW, Ueno C, Aslam R, Burnand K, Fife C, Grant L, et al. Guidelines for the treatment of arterial insufficiency ulcers. *Wound Repair Regen* 2006;14:693-710.
12. World Union of Wound Healing Societies' Initiative (WUWHS) Compression in venous leg ulcers. A consensus document. London: MEP Ltd; 2008.
13. Neglen P, Writing Group II of the Pacific Vascular Symposium 6; Eklöf B, Kulwicki A, Davies A, Deschamps T, Garcia M, Gloviczki P, et al. Prevention and treatment of venous ulcers in primary chronic venous insufficiency. *J Vasc Surg* 2010;52(5 Suppl):15S-20S.
14. Stacey M, Falanga V, Marston W, Moffatt C, Phillips T, Sibbald RG, et al. The use of compression therapy in the treatment of venous leg ulcers: a recommended management pathway. *EWMA J* 2002;2:9-13.
15. Obermayer A, Garzon K. Identifying the source of superficial reflux in venous leg ulcers using duplex ultrasound. *J Vasc Surg* 2010;52:1255-61.
16. Nilsson GE, Tenland T, Oberg PA. A new instrument for continuous measurement of tissue blood flow by light beating spectroscopy. *IEEE Transact Biomed Engin* 1980;27:12-9.
17. Nilsson GE, Tenland T, Oberg PA. Evaluation of a laser Doppler flowmeter for measurement of tissue blood flow. *IEEE Transact Biomed Engin* 1980;27:597-604.
18. Oberg PA, Tenland T, Nilsson GE. Laser-Doppler flowmetry—a non-invasive and continuous method for blood flow evaluation in microvascular studies. *Acta Med Scand Suppl* 1984;687:17-24.
19. Fronck A. Noninvasive evaluation of the cutaneous circulation. In: Bernstein EF, editor. *Vascular diagnosis*. 4th ed. St Louis: Mosby Year Book; 1993, p. 269-79.
20. Ubbink DT. Toe blood pressure measurements in patients suspected of leg ischaemia: a new laser Doppler device compared with photoplethysmography. *Eur J Vasc Endovasc Surg* 2004;27:629-34.
21. de Graaff JC, Ubbink DT, Legemate DA, de Haan RJ, Jacobs MJ. The usefulness of a laser Doppler in the measurement of toe blood pressures. *J Vasc Surg* 2000;32:1172-9.
22. Poelkens F, Thijssen DH, Kersten B, Scheurwater H, van Laarhoven EW, Hopman MT. Counteracting venous stasis during acute lower leg immobilization. *Acta Physiol* 2006;186:111-8.
23. Mosti G, Mattaliano V, Partsch H. Inelastic compression increases venous ejection fraction more than elastic bandages in patients with superficial venous reflux. *Phlebology* 2008;23:287-94.
24. Mosti G, Partsch H. Is low compression pressure able to improve venous pumping function in patients with venous insufficiency? *Phlebology* 2010;25:145-50.
25. Mosti G, Partsch H. Inelastic bandages maintain their hemodynamic effectiveness over time despite significant pressure loss. *J Vasc Surg* 2010;52:925-31.
26. Mosti G, Partsch H. Measuring venous pumping function by strain-gauge plethysmography. *Int Angiol* 2010;29:421-5.
27. Mosti G, Rossari S. L'importanza della misurazione della pressione sottobendaggio e presentazione di un nuovo strumento di misura. *Acta Vulnol* 2008;6:31-6.
28. Partsch H, Mosti G. Comparison of three portable instruments to measure compression pressure. *Int Angiol* 2010;29:426-30.
29. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, et al. Inter-society consensus for the management of peripheral arterial disease. *Int Angiol* 2007;26:81-157.
30. Abu-Own AA, Scurr JH, Coleridge-Smith PD. Effects of compression stockings on cutaneous microcirculation. *Phlebologie* 1993;46:671-2.
31. Mayrovitz HN, Larsen PB. Effects of compression bandaging on leg pulsatile blood flow. *Clin Physiol* 1997;17:105-17.
32. Schuren J, Vos A, Allen JO. Venous leg ulcer patients with low ABPIs: how much pressure is safe and tolerable? *EWMA J* 2010;10:29-34.
33. Labropoulos N, Wierks C, Suffoletto B. Intermittent pneumatic compression for the treatment of lower extremity arterial disease: a systematic review. *Vasc Med* 2002;7:141-8.
34. Comerota AJ. Intermittent pneumatic compression: physiologic and clinical basis to improve management of venous leg ulcers. *J Vasc Surg* 2011;53:1121-9.
35. Fagrell B. Problems using laser Doppler on the skin in clinical practice, In Belcaro GV, Hoffmann U, Bollinger A, Nicolaidis AN, editors. *Laser Doppler*. London: Med-Orion Publishing Company; 1994, p 49-54.
36. Abu-Own A, Shami SK, Chittenden SJ, Farrah J, Scurr JH, Smith PD. Microangiopathy of the skin and the effect of leg compression in patients with chronic venous insufficiency. *J Vasc Surg* 1994;19:1074-83.
37. Mayrovitz HN. Compression-induced pulsatile blood flow changes in human legs. *Clin Physiol* 1998;18:117-24.
38. Mayrovitz HN, Macdonald JM. Medical compression: effects on pulsatile leg blood flow. *Int Angiol* 2010;29:436-41.
39. Mayrovitz HN, Sims N. Effects of ankle-to-knee external pressures on skin blood perfusion under and distal to compression. *Adv Skin Wound Care* 2003;16:198-202.
40. Carter SA. Role of pressure measurements, In Bernstein EF, editor. *Vascular diagnosis*. 4th ed. St Louis: Mosby Year Book; 1993, p. 494-5.
41. Top S, Arveschoug AK, Fogh K. Do short-stretch bandages affect distal blood pressure in patients with mixed aetiology leg ulcers? *J Wound Care* 2009;18:439-42.
42. Partsch H, Clark M, Mosti G, Steinlechner E, Schuren J, Abel M, et al. Classification of compression bandages: practical aspects. *Dermatol Surg* 2008;34:600-9.
43. Obermayer A, Göstl K, Partsch H, Benesch T. Venous reflux surgery promotes venous leg ulcer healing despite reduced ankle brachial pressure index. *Int Angiol* 2008;27:239-46.
44. Chen AH, Frangos SG, Kilaru S, Sumpio BE. Intermittent pneumatic compression devices—physiological mechanisms of action. *Eur J Vasc Endovasc Surg* 2001;21:383-92.
45. Beidler SK, Douillet CD, Berndt DF, Keagy BA, Rich PB, Marston WA. Inflammatory cytokine levels in chronic venous insufficiency ulcer tissue before and after compression therapy. *J Vasc Surg* 2009;49:1013-20.

Submitted May 23, 2011; accepted Jul 13, 2011.