External compression increases forearm perfusion

Rolf P. Bochmann¹, Woldemar Seibel¹, Elke Haase³, Volker Hietschold², Hartmut Rödel³ and Andreas Deussend¹

¹ Institute of Physiology and ² Department of Radiology and Diagnostics, Medical Faculty, Technical University Dresden, D-01307 Dresden, Germany; ³ Institute of Textile and Clothing Technology, Faculty of Mechanical Engineering, Technical University Dresden, D-01062 Dresden Germany.

External compression increases forearm perfusion

Dr. R.P. Bochmann, Institut für Physiologie, Medizinische Fakultät Carl Gustav Carus, Technische Universität Dresden, Fetscherstr. 74, D-01307 Dresden, Germany;

rbochman@res.urz.tu-dresden.de; phone +49 (0)351 458 6030, fax +49 (0)351 458 6301

Copyright © 2005 by the American Physiological Society.
Abstract

Application of compression stockings to the lower extremities is a widely used therapeutic intervention to improve venous return but there is only scarce information about the effects of compression on local arterial perfusion. Therefore, we tested the hypothesis that a positive external pressure increases forearm perfusion. The relation between increasing external pressure induced by standardized compression and the arterial inflow and arterial flow reserve of the forearm was critically evaluated in a group of healthy young males (n=9). Flow was measured with venous occlusion plethysmography after 10 min application of 6 different stockings with increasing compression pressure ranging 13 – 23 mm Hg. During compression the arterial inflow increased significantly from 3.7 ± 0.85 to 8.8 ± 2.01 ml/min per 100 ml tissue (p < 0.001) and the arterial flow reserve increased from 17.7 ± 4.7 to 28.3 ± 7.0 ml/min per 100 ml tissue. The flow increase was persistent after 3 hours of constant application of external pressure and also during simultaneous low intensity hand grip. Similar results as obtained with occlusion plethysmography were seen with MRI. During the interventions there was no change in forearm temperature and no report of discomfort by the volunteers. In conclusion, 1) arterial perfusion of the human forearm increases more than twofold during application of external compression for a pressure range of 13 – 23 mm Hg. 2) The result is interpreted as an autoregulatory response following the decrease of the vascular transmural pressure gradient.

Key words: flow autoregulation, transmural pressure, arterial inflow, venous occlusion plethysmography,
INTRODUCTION

Mechanical interventions such as bandages and compression stockings have found wide application in the treatment of venous insufficiency of the lower extremities /9, 24/. Mostly, compression stockings are applied over a long period of time in the range of weeks and have yielded improvements of venous return, edema reduction and pain relieve.

Improvement of venous return is optimised by applying an increasing compression from proximal to distal /3, 15, 18/. While the effect of limb compression on venous return is a well established fact, the effects on arterial flow regulation are unsettled. Changes of arterial flow could be brought about by oedema removal and, thus, a decrease of vessel compression. However, it is also possible that application of external pressure could affect arterial vessel tone regulation directly. An effect on arterial flow is indicated by a study on volunteers, in which during foot-to-knee compression bandaging an increase of lower leg pulsatile blood flow was observed by nuclear magnetic resonance flowmetry /22, 23/. Intermittent pneumatic foot and calf compression (IPFCC) is also an effective therapeutic means to reduce the symptoms of venous insufficiency /4, 17, 19, 26/. Labropoulos et al. (1998) found increased blood flow using colour flow duplex imaging of the popliteal artery in healthy volunteers during application of IPFCC /17/. Two studies /17, 22/ indicate that there is an influence of compression not only on venous outflow but also on arterial inflow. However, the relation between the compressive pressure and the change in blood flow remains unclear.

To assess the influence of external compression on local blood flow we tested the hypothesis that external compression increases forearm blood flow. The hypothesis is based on the following causal chain: external compression pressure increases tissue pressure which decreases transmural vascular pressure. This decreased transmural pressure may trigger a myogenic response resulting in vessel relaxation. If this response comprises the segments of small arteries and arterioles a flow rise might result. Thus, we applied compression sleeves to the forearms of healthy volunteers and measured the forearm blood flow and the compression
pressure simultaneously. The duration of the response was also studied. The flow measurements generally performed with occlusion plethysmography were complemented by measurements using MR. Both methods show that external compression equivalent to a skin surface pressure of 13-23 mm Hg doubles forearm blood flow.

METHODS

Measurement protocol
The effect of compressive pressure on forearm blood flow was tested in a group of 9 healthy young males (age 25 ± 3 years). The study was approved by the ethical committee of the Medical Faculty of the Technical University of Dresden. No subject had diabetes, any history of venous or arterial disease or was taking any vasoactive medication. Upper arm blood pressures measured with standard blood pressure cuffs verified that subjects were normotensive (systolic: 117 ± 7 mm Hg, diastolic: 76 ± 5 mm Hg). Heart rate was 68 ± 8 bpm. During the experimental sessions the volunteers were seated in an armchair such that the forearms rested comfortably on wide arm rests at heart level with the wrist slightly elevated above elbow to promote venous drainage. During the entire session both forearms were covered with a light cloth to prevent cooling. The room temperature was regulated and held constant at 22 ± 2 °C.

Study 1. The purpose of the study 1 (n = 9) was to determine the forearm blood flow under the influence of 6 compression sleeves exerting an increasing compression pressure. The blood flow of both forearms was first measured under baseline conditions. Then, the compression sleeve was applied to the right forearm (in general the dominant arm) for 10 min. At the end of this intervention period (within the 10th minute) the blood flow of both arms was measured. In the same manner five other sleeves with increasing compression pressure were applied consecutively to the right forearm. Thus, the entire compression protocol lasted 60 min. After
removal of the strongest compression sleeve the blood flow of both arms was measured twice under baseline conditions again, once, immediately after the removal of the last compression sleeve and, secondly, after an additional 10 min.

**Study 2.** The purpose of study 2 (n = 9) was to determine the effect of an external compression pressure on the arterial flow reserve. To this end the study 1 was repeated and three measurements of the arterial flow reserve were inserted. The arterial flow reserve was determined after the second baseline measurement before the application of the first compression sleeve; after the application of the third compression sleeve (CS3) and at the end of the study, i.e. after the second baseline measurement. After the end of the arterial flow reserve measurement there was a resting interval of 10 min before the next measurement or before application of compression pressure.

**Study 3.** The purpose of the study (n = 5) was to test the duration of the effect of the compression on forearm blood flow. After a baseline measurement the compression sleeve CS3 was applied to the test arm and flow measurements were taken at 10 min, 30 min and then every half hour up to 180 min. After 180 min the compression sleeve was removed and forearm blood flow was determined immediately after removal and after 10 and 30 min, respectively. Between flow measurements, that is from 30 to 180 min the volunteers could move freely and do laboratory work.

**Study 4.** The purpose of the study (n = 5) was to test the hypothesis that the flow augmentation by compressive force was maintained during light exercise or movement. After a baseline measurement the compression sleeve CS3 was applied to the test arm. A first measurement of resting flow was undertaken after 10 min. Then, the volunteers were instructed to carry out rhythmic hand grip exercise with both hands (5-10% of maximal voluntary contraction in a 1/2–s contraction/relaxation duty cycle) for nearly 70 min. The low workload of 5-10% maximal voluntary contraction and the relaxation period twice as long as the contraction period was specially selected to avoid an increase in blood flow by the
exercise itself. During hand grip exercise blood flow was determined every 10 min. During the flow measurements (90 s) the subjects did not exercise.

**Study 5.** The purpose of the study (n = 3) was to test the flow augmentation under clinical conditions and to measure the forearm blood flow with MR technique. Using the MR technique the blood flow of the right forearm was determined under normal conditions without compression and 10 min after the application of a compression sleeve (CS3) the measurement was repeated. For comparison the blood flow of the same forearm was measured with venous occlusion plethysmography with and without compression sleeve. For technical reasons it was not possible to perform MR and plethysmography simultaneously. The time lag between both measurements was approximately 180 min. The temperature of both forearms was controlled using a multi channel measuring system ("ALMEMO-1", Ahlborn Mess- und Regelungstechnik GmbH, Holzkirchen, Germany).

**Flow assessment**

The arterial inflow and arterial flow reserve of both forearms were measured with venous occlusion plethysmography ("COMPACTUS" Fa. Gutmann-Medizintechnik, D-82547 Eurasburg, Germany). This method is based on the measurement of volume changes under the condition of blocked venous outflow and by this way measures the arterial inflow. The strain gauge was placed on the forearm where the circumference is maximal and during the application of the compression sleeve the strain gauge was placed over the sleeve. The venous occlusion cuff around the upper arm was inflated for 6 s with a pressure of 60 mm Hg to ensure venous occlusion. During this period of time the arterial inflow was calculated from the increase of forearm circumference. The arterial inflow was measured 6 times consecutively every 15 s and averaged. The flow measurement lasted typically 90 s. The maximal arterial inflow or arterial flow reserve of the forearm was determined as the peak arterial inflow after a 5 min arterial occlusion. During the arterial occlusion the cuff around
the upper arm was inflated to a pressure of 180 mm Hg which was at least 50 mm Hg above the mean systolic pressure of the subjects.

*External pressure measurement*

The actual pressure under the sleeve was measured with a sensor using the piezoelectric effect ("MCDM-1", MIPM GmbH, Mammendorf, Germany). A piezoelectric crystal is characterised by that property under the application of a given pressure to induce a certain voltage. Such a sensor is suitable to measure the pressure in biological tissue or between different layers. The MCDM-1 is a device especially developed for the measurement of the pressure under a compression stocking. The technical details of the device /14/ and its application for measurement of the pressure exerted by the compression stocking /11/ have been reported by others. The measurement error in our experiments was ± 2 mm Hg. The pressure under the stocking was measured at two positions differing by the winding radius of the circumference of the forearm. According to the rule of Laplace the pressure inside a wall is inversely proportional to the winding radius at a given wall tension. Therefore, the two sensors were placed on the forearm at positions of greatest and smallest winding radii.

*Compression sleeves*

The force exerted on the skin surface critically depends on the dimensions and elastance of the sleeves as well as individual arm geometries. Thus, the compression sleeves were manufactured individually for each volunteer on the basis of 6 measurements of the circumference of the forearm between wrist and elbow. The increasing level of compression was realised by decreasing the equivalent circumferences of the textile. In this way the wall tension of the sleeves, when covered over the forearm, was increased according to the stress-strain relation. According to the rule of Laplace this increased wall tension exerted a certain pressure on the tissue of the forearm. Thus, for each volunteer a set of 6 sleeves (CS1 – CS6)
with decreasing circumferences was designed and manufactured using a knitted fabric which is based on an elastane yarn plated upon a cotton yarn. The used compression sleeves are similar to the sleeve of a sport tricot and their thickness is 1.022 ± 0.034 mm. The stress-strain relation of the knitted fabrics was determined and the textile parameters are summarized in Table 1.

The sleeves were produced in consideration of the textile-physical parameters of the knitted fabrics as usual in clothing manufacturing. The specific stretch-factor of the fabrics was determined at 6 different stresses in order to manufacture the sleeves. These factors were multiplied with the corresponding circumferences of the forearm at different cross sections as measured for each test person.

MR measurements

MR measurements were performed on a 1.5 Tesla whole body scanner (Siemens Magnetom Sonata, Siemens AG, Erlangen, Germany). The volunteers were positioned in a supine to lateral position in order to locate the forearm near to the isocenter of the magnet. A rectangular flexible surface coil was placed on the proximal end of the forearm. An ECG triggered FLASH 2D sequence with flow encoding perpendicular to the slice plain (max. velocity 100 cm/s) was applied (FOV 220 * 165 mm², matrix 256 * 192, slice thickness 5 mm, T_E = 3.9 ms, T_R triggered to every heart beat, 5 averages), giving modulus and velocity images every 32 ms. For quantification, the ARGUS software package was used (Siemens AG, Erlangen, Germany). Flow and velocity data were derived from regions which were fitted to the actual vessel size and position for each time point of the measurement.
Statistics

Sample size was estimated a priori for the primary hypothesis that external compression would result in a vasodilatory response. These calculations showed that a sample size of 9 subjects would allow detection of a 50% increase in forearm blood flow under external compression with a power of 80% at $\alpha=0.05$. Statistical comparisons were performed by Student’s t test and by ANOVAs for repeated measures. Forearm blood flow responses of the test arm to external pressure were compared to that of the control arm. All calculated probability values were 2-tailed, and a p-value $< 0.05$ was considered to indicate statistical significance. All group data were reported as means $\pm$ SD.
RESULTS

The effect of a compression pressure of $16 \pm 5$ mm Hg applied to the forearm surface is shown in Fig. 1. Baseline forearm blood flow averaged $2.3 \pm 0.2$ and $1.9 \pm 0.6$ ml/min per 100 ml tissue for the test arm and the control arm, respectively (n. s.). After application of pressure blood flow of the test arm increased within 3 min to a new steady state 115% above control ($p< 0.001$), while blood flow of the control arm remained unaffected. Following removal of the compression blood flow of the test arm returned to the baseline level within 1 min.

Study 1: The effect of increasing compressive force on forearm perfusion is shown in Fig. 2. All six compression sleeves enhanced blood flow of the test arm specifically. The flow increases were significant ($p< 0.001$) as compared with the control arm for CS2, CS3, CS4, CS5 and CS6 respectively. The flow increases were completely reversible after 1 min of removal of the compressive force.

Calibration curves for the different constructions of compression sleeves with respect to the surface pressure applied are summarized in Fig. 3. Data is presented with respect to the smallest and the largest winding radius. An almost linear relationship was found for the largest winding radius, while the relationship was less consistent for the smaller winding radius. From the data shown in Fig.2 and Fig.3 the relationship of forearm perfusion versus compression pressure was calculated (Fig. 4). The largest flow increase was found for an arm surface compression pressure of $19.7 \pm 5.8$ mm Hg. In 3 subjects an additional flow measurement was taken at a compression pressure around 50 mm Hg by moving the sleeve slightly further upward. Pressures above 50 mm Hg reduced forearm blood flow considerably.

Study 2: Similar to baseline blood flow the flow reserve was enhanced during application of compressive force. Fig. 5 shows that peak flow after 5 min of occlusion was nearly 5-fold of baseline blood flow. During application of compression sleeve CS3 peak flow was significantly ($p<0.01$) increased to $28.0 \pm 6.0$ ml/min per 100 ml tissue as compared to $17.4 \pm$
6.0 ml/min per 100 ml tissue without compression. These effects were completely reversed after removal of the compression sleeve. In contrast, baseline flow and flow reserve of the control arm remained unchanged over the entire duration of the experiment (Fig. 5B).

**Study 3:** The effects of compression force on forearm blood flow were also studied for a more extended period of time (Fig. 6). In 5 volunteers forearm blood flow remained increased over the entire period of pressure application (3 hours). After a slightly higher initial increase of blood flow, the flow response reached a steady state after 60 min. Following removal of the compression sleeves the flow changes were largely reversible after 1 min and fully reversible after 30 min. The forearm blood flow of the control arm remained unchanged during the entire study period.

**Study 4:** In a further set of experiments we tested whether the flow augmentation by compressive force was maintained during light exercise. As shown in Fig. 7 light hand exercise during continuous application of compression sleeves (CS3) was associated with a trend to a further slight elevation of forearm blood flow in the test arm but not in the control arm.

**Study 5:** In a final set of experiments forearm blood flow in response to compressive force (CS3) was determined consecutively by venous occlusion plethysmography and MR in 3 volunteers. As evident from Fig. 8, both methods revealed increases of blood flow during the application of compression pressure.

The skin temperatures of the forearms remained unchanged throughout the measurement periods. No differences were found between the test arm and the control arm before (34.1 ± 0.98 °C and 34.1 ± 1.29 °C) and after test series (34.3 ± 1.03 °C and 34.5 ± 1.26 °C), respectively.
DISCUSSION

The application of increased tissue pressure known as compression therapy is the mainstay of physical treatment of venous insufficiency of the legs and the prevention of deep vein thrombosis /15,16/. The application of compression therapy increases the healing rate in venous leg ulcer and improves deep vein hemodynamics. However, little is known about the impact of external compression on arterial flow regulation. Using a forearm model the present study has revealed two major findings. Firstly, the external pressure application significantly increases forearm blood flow. A clear relationship between the increase of external pressure and the increase of blood flow could be established with a maximal flow effect at a forearm surface pressure increase around 20 mm Hg. The present study extends previous reports which suggested that external compression may not only augment venous return but also affect arterial flow regulation separately. Secondly, the external compression significantly increases hyperaemic peak flow after 5 min circulatory arrest.

A few previous studies have addressed the effects of moderate leg compression on arterial inflow /18, 22, 23, 25/. In one of these studies an unchanged calf muscle blood flow was reported for application of graded compression pressure /18/. In this study, muscle blood flow was determined by measuring xenon-133 clearance. The maximally applied compression pressure was 30 mm Hg. However, in another study /25/ conducted on male distance runners moderate compression (40 mm Hg) reduced the muscle blood flow in the thigh by approximately 50%. The reason for the discrepancy between these two studies is not readily apparent, because the method to assess changes in leg blood flow was identical. Another previous study comprising 14 healthy subjects reported the effects of leg compression by bandaging on leg pulsatile blood flow using nuclear magnetic resonance flow meter /18,19/. A flow increase of 29% was reported for a pressure increase of approximately 40 mm Hg. Leg
compression was produced by wrapping one leg from forefoot to knee with a four-layer bandaging system. The effect of lower compression pressures was not studied.

In the present study an individually fitted set of compression sleeves was used to obtain flow measurements over a wider pressure range. To the best of our knowledge this is the first systematic study performed on the human forearm. The use of elastic sleeves to induce an external compression pressure has two advantages. Firstly, blood flow measurement with strain gauge plethysmography can be performed at the forearm with and without the elastic sleeves. The strain gauge has to be placed around the outside of the sleeve to detect the expansion of the forearm when the venous outflow is blocked. Secondly, using compression sleeves it is possible to change or superimpose other conditions like hand exercise or free move. To address the limitation of strain gauge utilisation under simultaneous application of a compression sleeve the amount of volume expansion during a single measuring cycle under resting conditions is estimated: supposing that the resting blood flow to the forearm is 3 ml/min per 100 ml tissue then the additional blood volume which is pooled in the forearm during the short venous occlusion amounts to 0.3 ml per 100 ml tissue. In other words, the additional volume is in the range of 0.3% of the unaffected volume. By design the compression sleeves are stretched by 20% when applied to the forearm to produce the compression pressure. The additional stretch in the range of 0.3% is sufficiently small and can therefore be neglected. Under the increased flow conditions when the compressive sleeve is applied to the forearm the additional volume is in the range of 0.6% of the unaffected volume. This volume change is also sufficiently small for this effect to be neglected.

The elastic sleeve covers the forearm from elbow to wrist and it is generally assumed that the pressure exerted at the surface of the forearm is transmitted into the depth of the tissue to the arterial vessels as under every pneumatic cuff. In a methodological study the transmission of a negative surface pressure (2-50 mmHg) into an arm segment of only 8 cm length was demonstrated /21/. The forearm model was chosen, because it is well established for studies
on blood flow in humans. Furthermore, the human forearm is not subject to hydrostatic pressure differences to such a degree as it occurs in human leg. It is presently unknown whether there exist systematic differences between the upper and lower extremities with respect to the pressure range that enhances blood flow or to the mechanisms that might be involved. However, from the results obtained in the present study and the majority of the previous studies conducted on the human leg, it seems that external pressure application may increase blood flow in a similar pressure range and to a similar relative extent. The reason for the discrepancies between previous leg studies is not readily apparent. It should be kept in mind that the study which reported a decrease of leg flow in response to external pressure was performed in athletes. Whether there exist systematic differences of flow control between athletes and non-athletes, that may explain differences of flow adjustment in response to external compression, needs to be clarified.

The relationship between compression pressure and flow increase was more consistent for the pressure measured under the sleeve for the larger winding radius (Fig. 3). The larger winding radius was determined over the group of hand flexors, while the minimal winding radius was measured over the region of the hand extensors. Both muscle groups differ considerably with respect to their masses. The group of flexors dominates by mass considerably over that of the extensors. In addition, both muscle groups are compartmentalized and separated from each other by bone and ligament structures. Thus, it seems that the true relationship between compressive surface pressure and blood flow change of the forearm is best expressed by that determined for the larger winding radius. Additionally, scanning the human forearm shows that most of the cross sectional area is characterized by a large winding radius. This relationship between compressive pressure and blood flow change was almost linear for the range of 0 to 20 mm Hg.

The application of external pressure increases also the peak flow after a 5 min occlusion of the upper arm (Fig. 5). The increase in peak flow is greater than the increase in resting blood
flow. In addition to resting blood flow the present study evaluated flow under stimulated conditions induced by either moderate handgrip exercise lasting 1 hour (Fig. 6) or during 3 hours when the subjects could move freely in the laboratory (Fig. 7). For both conditions, the enhancement of blood flow could be assessed suggesting that the mechanism that leads to the flow augmentation is robust with respect to the particular condition.

The somewhat surprising finding that static compression consistently increases, rather than decreases, blood flow of the human forearm gives rise to the question of the underlying mechanism(s). Based on known vascular control features three mechanisms may envisioned that account for this: 1) myogenic response, 2) venular-arteriolar communication, and 3) skin vasomotor reflexes. These potential mechanisms are discussed below.

**Myogenic response.** It is well established that arteriolar vessels constrict in response to an increase of the vessel transmural pressure gradient and dilate in response to a fall of the pressure gradient (Bayliss effect) /2, 6, 12/. The increased external arm compression by the sleeve is transmitted into the tissue and reduces the transmural pressure gradient of the arterial vessels and arterioles in the forearm /21/. The potential importance of transmural vascular pressure is supported by the early study of Greenfield and Patterson /8/. These authors applied subatmospheric pressure (-100 mm Hg) to the forearm for up to 1 min. Immediately after pressure release venous occlusion plethysmography indicated a fall of blood flow, which may have reflected a vasoconstriction in a response to the increase of the vessel transmural pressure during external application of the subatmospheric pressure.

In a more detailed study Lott et al. /20/ have applied a positive or negative pressure of 50 mm Hg to the forearm with an onset time and reset time of less then 0,2 s. During the first two heart beats after the onset of +50 mm Hg pressure there was a rapid transient decrease in blood flow followed by a steady increase in flow. This steady increase of flow after a positive pressure step and the equivalent decrease of flow after a negative pressure step provide
evidence that the myogenic response is normally operative in the forearm circulation of humans.

Our study clearly demonstrates that external pressure not only increases resting blood flow in the forearm but also the peak flow after a 5 min occlusion. The role of NO-mediated dilatation in the hyperaemic flow response was investigated in two studies /4, 11/ and revealed that hyperaemic peak flow was not influenced by NO-synthase blockade. Joannides et al. /11/ measured blood flow and diameter of the radial artery and the occlusion cuff was placed distal around the wrist. Under conditions of NO-synthase inhibition (L-NMMA) the blood flow increase was unchanged compared to control conditions but the diameter of the radial artery was reduced. In the second study /4/ the brachial artery was imaged for diameter measurements 7-10 cm distal the puncture site for L-NMMA infusion and the hyperaemic response was provoked with occlusion cuffs at two positions: distal to the ultrasound probe for imaging and proximal to the puncture site. When NO production was blocked with L-NMMA there was no dilatation of the brachial artery with occlusion cuff around the wrist, but with occlusion cuff around the upper arm there was the same initial dilatation of the brachial artery as under control conditions. These two studies have demonstrated that the lowering of the transmural pressure alone as it occurs during 3-5 min circulatory arrest produces a local dilatation. This dilatation does not require local NO production. In this study we have demonstrated that external compression not only increases resting blood flow in the forearm but also the hyperaemic peak flow. These two facts point to the key role of the transmural pressure and the myogenic response in the underlying processes.

Venular-arteriolar communication. Because of the much thicker wall, arterial vessels are considerably more rigid than venous vessels. External pressure application may therefore reduce venous vessel lumen considerably, whereas the arterial segments dilate, because of the above mentioned myogenic response. As a consequence of the reduced lumen blood flow may then exert an enhanced shear stress in venous vessel segments, which may trigger the release
of endothelial dilators in the venous segments. Besides acting locally, and thereby tending to normalize venous segment resistance, in the microcirculation these compounds (e.g. NO or EDHF’s) may reach neighbouring arterial vessel segments by diffusion and reduce arteriolar resistance /10/. In the arteriolar vessel segments this might act synergistically with the myogenic response in reducing vessel resistance. Within our experimental setup we can not exclude the influence of the venular-arteriolar communication under resting conditions. However, as pointed out above treatment of volunteers with L-NMMA did not result in a diminution of the reactive hyperaemic response following transient ischemia. This argues against this mechanism being of importance.

Skin vasomotor reflexes. The application of pressure to the skin surface may activate non-nociceptive skin receptors or proprioceptors in the tissue layers beneath. The potential importance of such reflexes has been stressed recently /1, 7/. These authors applied increasing (5 mm Hg min⁻¹) external pressure locally (1 cm²) and measured microcirculatory flow in the same region by laser Doppler flow meter. In the range of high compression pressure (above 50 mm Hg) they found a steadily decreasing LDF signal, whereas in the pressure range of 20 mm Hg there was a transient increase in LDF signal above baseline. It should be pointed out, however, that this result might also be evoked by a myogenic response. To conclusively discern between these two causal factors needs to be addressed in future studies.

In conclusion, the present study demonstrates that systematic elevation of forearm external compression results in an increase of resting blood flow and hyperaemic peak flow in that region. The maximal flow effect is induced by a pressure increase of 20 mm Hg. The likely mechanism that may mediate the flow increase in response to external pressure application is the myogenic response. Future studies have to delimitate the involvement of venous-arterial communication and skin vasomotor reflexes.
GRANTS

The study was supported by grants from the Deutsche Forschungsgemeinschaft DE 360/ 7-1 and RO 1303/2-1.
References


List of figure captions

Fig. 1
Time course of forearm perfusion during 10 min application of compression sleeve CS3 (3 subjects). Closed symbols represent measurements on the test arm, open symbols those of the control arm.

Fig. 2
Arterial inflow of control arm (open bars) and test arm (closed bars) during consecutive application of different compression sleeves (CS1 – CS6) to the test arm. Each compression level was held constant for 10 min (n=9). * p<0.001 vs. control arm.

Fig. 3
Effects of compressive force on skin surface pressure as measured under conditions reported in the legend of Fig. 2. Surface pressures were measured for positions of smallest (A) and largest winding radii (B).

Fig. 4
Arterial inflow as a function of compression pressure as measured for the largest winding radius (n=9). The symbol close to a pressure of 50 mm Hg is the mean of 3 subjects.

Fig. 5
Arterial inflow (closed bars) and arterial flow reserve (open bars) measured as peak flow after 5 min occlusion. The 3 consecutive measurements were 40 min apart as described under study
2 section methods. Compression sleeve applied to the test arm (A) was CS3, (B) - control arm, (n=9). * p<0.01 vs. 1.measurement.

Fig. 6
Effects of external compression on forearm perfusion over 3 hours (n=5). Compressive sleeve (CS3) was applied from 5 to 180 min. Baseline measurements are set to 100%; control arm – open symbols, test arm – closed symbols. * p<0.01 vs. control arm.

Fig. 7
Forearm perfusion of control arm (open symbols) and test arm (closed symbols) during compression and rhythmic hand grip (exercise). Compression sleeve was CS3 applied from 5 to 80 min (n=5). For further information see text (section methods). * p<0.01 vs. control arm.

Fig. 8
Flow increase under compression (CS3) measured with venous occlusion plethysmography and MR in percent of baseline (n=3; median and range).
Table 1. Textile parameters of the circular knitted fabric

<table>
<thead>
<tr>
<th>Fibre composition</th>
<th>Yarn fineness</th>
<th>Construction</th>
<th>Cover factor in ( \sqrt{\text{tex}} )/mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>88 % CO; 12 % EL</td>
<td>70/1</td>
<td>circular knitted fabric, cross tuck 1x1</td>
<td>1.33</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td></td>
<td>2.83</td>
</tr>
</tbody>
</table>

CO – cotton; EL - elastane
Figures

Fig. 1

Fig. 2
Fig. 5

A

B

ml/min per 100 ml tissue


ml/min per 100 ml tissue


*