Posturally induced microvascular constriction in patients with different stages of leg ischaemia: effect of local skin heating

DIRK TH. UBBINK*, M. J. H. M. JACOBS*, G. J. TANGELDER†, D. W. SLAAFS‡ AND R. S. RENEMAN†

Department of *Surgery, Academic Hospital Maastricht, and Departments of †Physiology and ‡Biophysics, Cardiovascular Research Institute, Maastricht, The Netherlands

(Received 30 July/27 December 1990; accepted 17 January 1991)

SUMMARY
1. Skin microcirculation was investigated in 12 asymptomatic subjects and 76 patients, grouped according to their ankle-to-brachial systolic blood pressure index, in order to evaluate to what extent posturally induced microvascular constriction is dependent on the stage of leg ischaemia at different local skin temperatures.
2. Skin microcirculation was assessed in the supine and sitting position by using laser Doppler fluxmetry at unheated skin temperature and at 36°C, and transcutaneous oximetry at 37°C and 44°C.
3. Skin perfusion and oxygenation diminished with decreasing ankle-to-brachial systolic blood pressure index. In healthy control subjects, perfusion and oxygenation were reduced when changing from the supine to the sitting position, but were enhanced in patients with severe leg ischaemia (ankle-to-brachial systolic blood pressure < 30%), indicating disturbed posturally induced vasoconstriction.
4. Increasing the local skin temperature resulted in a higher perfusion and masked the posturally induced vasoconstriction in healthy subjects. In patients with severe leg ischaemia, however, perfusion was unaltered by the temperature increase, apparently because the microvessels were already maximally dilated. The induction of reactive hyperaemia produced no additional increase in perfusion or oxygenation.
5. It is concluded that posturally induced microvascular constriction in the skin is disturbed in patients with severe leg ischaemia (ankle-to-brachial systolic blood pressure index < 30%). Disturbed microvascular constriction upon dependency was also seen in healthy subjects after local skin heating. This suggests that posturally induced vasoconstriction is mainly regulated by local mechanisms.

Key words: laser Doppler fluxmetry, leg ischaemia, local heating, posture, skin microcirculation, transcutaneous oximetry.

Abbreviations: ABI, ankle-to-brachial systolic blood pressure index; LDF, laser Doppler flux; tcPo2, partial pressure of oxygen at the skin surface.

INTRODUCTION
Patients suffering from severe arterial obstructive disease of the leg tend to lower the leg to alleviate nocturnal or rest pain. This seems paradoxical, because in normal limbs blood flow is reduced on dependency [1] by central sympathetic reflexes [2-5] and local myogenic or veno-arteriolar constriction mechanisms [6-8] in order to protect against oedema formation [8, 9].

The microvascular constriction, as seen after leg dependency in healthy subjects [3, 9-11], is abolished in patients with leg ischaemia [2, 12, 13], possibly owing to a disturbance of the veno-arteriolar response [14, 15], finally resulting in enhanced perfusion of the capillaries [16] and deeper vessels of the skin [17]. Local skin heating [18, 19], especially in combination with postural changes [20-22], is a useful test with which to determine the integrity of microvascular constriction mechanisms in leg ischaemia. However, whether local or central mechanisms prevail in the disturbance of these mechanisms has not been investigated. Furthermore, it is still unknown at which level of arterial pressure reduction microvascular constriction upon dependency will be impaired. To this end, we investigated skin microcirculation in patients with various degrees of arterial obstructive disease of the legs, by using laser Doppler fluxmetry and transcutaneous oximetry, before and after a change in posture. In each position the measurements were performed at rest and during reactive hyperaemia, and at different local skin temperatures.

Dr M. J. H. M. Jacobs, Dept. of Surgery, Academic Hospital, PO Box 5800, 6202 AZ Maastricht, The Netherlands.
METHODS

Patients and control subjects

Twelve asymptomatic control subjects and 76 patients suffering from atherosclerotic arterial obstructive disease of one or both legs, co-operated after giving their informed consent. The age of the 62 males and 26 females ranged from 43 to 95 years, with a mean value of 71 years. Clinically, their complaints varied between intermittent claudication, rest pain and ulceration. The patients were divided into five groups (Table 1), according to their ankle-to-brachial systolic blood pressure index (ABI). ABI varied from 0% to 123%, being lowest in group I and highest in group V.

Smoking habits and drug treatment, especially that interfering with vasoconstriction, are shown in Table 2.

Of the 12 subjects in group V, two were receiving medication for cardiac angina, one for hypertension, one for a recent myocardial infarction and one for atherosclerotic symptoms of the leg not investigated here. The patients in this group had a presumed normal arterial system in their investigated leg, not only because their ABI was higher than 95%, but also because analysis of the Doppler signals of the major leg arteries showed no pathology, as opposed to the arterial Doppler signals of the patients in groups I–IV. The mean age in group V (67 years) did not differ significantly from the mean age in groups II–IV (69 years). The mean age in group I, however, was higher (82 years). Hence, patients in group V were considered as age-matched controls only for groups II–IV.

Patients with insulin-dependent diabetes mellitus were excluded from the study, since this disease per se influences microcirculatory autoregulation mechanisms [23]. However, 19 non-insulin-dependent diabetic patients did participate in this study (Table 2). They were all well-regulated and none of them had clinically manifest peripheral neuropathy. Their findings were interpreted separately to assess possible differences in microvascular reactivity between diabetic and non-diabetic patients.

Patients and control subjects refrained from smoking and did not consume caffeine or alcohol for at least 1 h before examination.

Experimental protocol

Measurements were performed after a 20 min period of acclimatization in a room with a temperature between 23 and 24°C. Laser Doppler fluxmetry and transcutaneous oximetry (see the Equipment sub-section) were performed simultaneously, as shown in Fig. 1.

Microvascular reactivity was determined by laser Doppler fluxmetry at unheated skin temperature (about 29°C, see the Results section) and at 36°C, and by transcutaneous oximetry at 37°C and at 44°C. Recordings of Laser doppler flux (LDF) at unheated skin temperature were made in order to assess the integrity of the constriction mechanisms under control conditions during the first part of the investigations (parts 1–4 in Fig. 1). Subsequently (parts 5–8 in Fig. 1), laser Doppler fluxmetry was performed at 36°C to determine the degree of microvascular constriction after limited impairment of local vasoconstricting mechanisms, as was the case with the transcutaneous oximetry performed at an electrode temperature of 37°C (parts 1–4 in Fig. 1). At this temperature, there is only slight dilatation of the superficial arterioles and the recorded partial pressure of oxygen at the skin surface (tcPo2) depends on arteriolar flow rather than arteriolar partial pressure of oxygen [24, 25]. Transcutaneous oximetry was performed at 44°C, to overrule local vasoconstriction completely (parts 5–8 in Fig. 1). In this state, the tcPo2 recorded depends only on the arteriolar partial pressure of oxygen and is flow-independent [25].

LDF and tcPo2 were assessed at rest, as well as during reactive hyperaemia after the release of a 3 min arterial occlusion, which was achieved by inflating a supra-

<table>
<thead>
<tr>
<th>Table 1. Patient groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>I</td>
</tr>
<tr>
<td>II</td>
</tr>
<tr>
<td>III</td>
</tr>
<tr>
<td>IV</td>
</tr>
<tr>
<td>V</td>
</tr>
</tbody>
</table>

Table 2. Characteristics of patients

The number of patients who experienced relief of pain on dependency, of non-insulin-dependent diabetic patients, of smokers and of patients using vasoactive drugs is shown. Vasodilator drugs: 1, arterial and venous dilators (one patient in group II and one in group IV used an angiotensin-converting-enzyme inhibitor); 2, β-adrenoceptor blocking agents; 3, calcium antagonists.

<table>
<thead>
<tr>
<th>Group</th>
<th>Diabetes</th>
<th>Smokers</th>
<th>Vasoactive drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>13</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>24</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>III</td>
<td>23</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>IV</td>
<td>16</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>V</td>
<td>12</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 2. Characteristics of patients

The number of patients who experienced relief of pain on dependency, of non-insulin-dependent diabetic patients, of smokers and of patients using vasoactive drugs is shown. Vasodilator drugs: 1, arterial and venous dilators (one patient in group II and one in group IV used an angiotensin-converting-enzyme inhibitor); 2, β-adrenoceptor blocking agents; 3, calcium antagonists.

<table>
<thead>
<tr>
<th>Group</th>
<th>Diabetes</th>
<th>Smokers</th>
<th>Vasoactive drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>13</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>24</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>III</td>
<td>23</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>IV</td>
<td>16</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>V</td>
<td>12</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
malleolar cuff. The induction of reactive hyperaemia in the foot in this standard manner was applied not only to reduce measurement variability at rest, but also to assess whether the larger vessels between the ankle and the skin had any additional effect on microvascular reactivity upon dependency.

The laser Doppler probe holder was attached to the pulp of the great toe with double-sided adhesive tape. The probe itself was secured to the holder with adhesive tape to avoid flux variations due to rotation of the probe. The tcPo₂-measuring electrode was attached to the dorsum of the foot in the first intermetatarsal space by an adhesive fixation ring containing contact liquid.

Recordings started after the laser Doppler fluxmeter and the transcutaneous oximeter had reached a stable value, which took at least 10 min. Measurements were repeated in the other position after a 10 min adaptation period, during which the parameters again reached a steady state. The laser Doppler probe and the tcPo₂ electrode were kept in place while changing position.

Brachial systolic blood pressure was measured by means of a cuff and an 8 MHz Doppler probe, with the patients in the supine position. After the whole procedure, systolic ankle blood pressure was assessed in the same way. These data were used to calculate the ABI in the supine position. Brachial blood pressure was also measured using the Riva Rocci method to obtain mean arterial pressure (two-thirds diastolic blood pressure + one-third systolic blood pressure). Finally, systolic ankle blood pressure was measured in the sitting position in order to assess the orthostatic pressure increase after the change in posture.

**Equipment**

**Laser Doppler.** A laser Doppler instrument (Periflux PF3; Perimed, Stockholm, Sweden) produces a voltage signal directly proportional to the microvascular blood flux in superficial skin vessels. This signal is derived from the Doppler shift of the laser light, backscattered by moving blood cells in a skin sample volume of about 1 mm³ [26]. Hence, both the flow in superficial capillaries and in deeper skin vessels, such as the venous plexuses and arterio-venular anastomoses, contribute to the signal. The laser Doppler probe holder contains a heater and a temperature-measuring device. The temperature can be measured continuously and can be set at any level between 26 and 44°C. The instrument was calibrated on 250 perfusion units (equal to 2.5 V), using the Periflux Motility Standard. During the measurements, the low band pass filter was used to reduce movement artefacts and the time constant was set at 2 s to evade heart beat oscillations. The 0.07–12 kHz band was used, unless the perfusion was so low that switching to the 4 kHz band, and, hence, improving the signal-noise ratio, was necessary. The output was fed into a pen recorder. The recording speed was 3 cm/s.

**Transcutaneous oximeter.** The transcutaneous oximeter (Radiometer TCM2, Copenhagen, Denmark) measures tcPo₂. The electrode itself can be heated to increase local skin temperature. tcPo₂ is derived from the electrical potential changes due to a redox reaction that takes place in the electrode in the presence of oxygen. The instrument was calibrated against atmospheric Po₂ [27]. The pen recorder speed was set at 0.5 cm/min.

**Off-line analysis.** LDF and tcPo₂ recordings yielded the following parameters:

1. The flux (V) and tcPo₂ (mmHg) under resting conditions (rest flux and rest tcPo₂, respectively) and the peak flux and peak tcPo₂ during reactive hyperaemia (peak flux and peak tcPo₂, respectively). Biological zero flux was subtracted [28].
2. The time (s) required to reach peak flux and peak tcPo₂ after release of the occlusion (Tpfux and TpPo₂, respectively).
3. The time (s) required after release of the occlusion to reach half of the rest flux and rest tcPo₂ value (T50%flucx and T50%Po₂).
4. The individual supine to sitting flux and tcPo₂ ratios (at rest and during reactive hyperaemia), as an index of the effectiveness of the postural vasoconstrictive mechanisms (vasoconstriction index), e.g. the lower the ratio, the more these mechanisms are impaired. If the rest flux or rest tcPo₂ was zero in both the supine and the sitting position, the ratio was regarded to be zero.

**Statistics**

Data are plotted as a function of ABI. To facilitate comparison, the patient data were handled per ABI group. Because in each group the results of all parameters obtained were not symmetrically distributed, medians are presented to characterize group values. The non-parametric Kruskal–Wallis test was used to test for signifi-
significant differences between the five groups. For the analysis of differences between the sitting and the supine position the Wilcoxon signed-ranks test was used. Differences were regarded as significant when $P < 0.05$.

RESULTS

In general, mean arterial blood pressure was slightly higher in groups II and III. Differences in systolic ankle blood pressure between the sitting and the supine position were on average 48 mmHg, being higher in the sitting position. This was a consistent finding, not related to the ABI in the supine position. In each group LDF and tcPO$_2$ indices in both positions did not significantly differ between non-insulin-dependent diabetic patients and non-diabetic subjects and, therefore, the data were pooled.

Laser Doppler fluxmetry

Fig. 2 presents the individual laser Doppler rest flux data, including group medians, at unheated skin temperature (Fig. 2a) and at 36°C (Fig. 2b), for both positions. Vasoconstriction indices at these temperatures are shown in Figs. 2(c) and 2(d), respectively. The median unheated skin temperature in both positions (29°C, range 27–29°C) differed significantly between the five groups ($P < 0.005$) and was lowest in the supine position in group I (27°C). Changing from the sitting to the supine position caused a non-significant decrease in unheated skin temperature. At this temperature, median rest fluxes (Fig. 2a) appeared to decrease with decreasing ABI (supine: from 0.3 V in group V to 0 V in group I; sitting: from 0.18 to 0.01 V). This decrease was significant in the supine position ($P < 0.001$). In groups IV and V, rest fluxes in the supine position were higher than those in the sitting position ($P < 0.01$), but the opposite was seen in groups II and I ($P < 0.05$). In these groups, vasoconstriction indices were generally below unity (Fig. 2c). All patients (except one) with an ABI less than 30% had an index below 1. All patients (except one) with an ABI above 55% had indices above 1. At an ABI of between 30% and 55%, it could be described on the basis of LDF measurements whether or not vasoconstrictive mechanisms were intact, according to the vasoconstriction indices.

Local skin heating to 36°C enhanced rest flux in the higher-ABI groups ($P < 0.05$ in groups II–V) in both positions (Fig. 2b). However, the flux differences between the supine and sitting position, as seen at unheated skin temperature, were now reduced. This is illustrated in Fig. 2(d), showing vasoconstriction indices only slightly exceeding unity in groups IV and V. Rest fluxes as well as flux indices in groups I and II persisted to be very low and were virtually unaltered by skin heating: the median rest flux in the sitting position remained higher than in the supine position ($P < 0.05$, groups I–III) and again below an ABI of 30%, every patient had a vasoconstriction index lower than 1.

Peak flux and rest flux data were similarly related to the ABI: at both temperatures, median peak flux decreased

![Fig. 2](image-url). Scatterplots of rest flux (a and b) in the supine (●) and sitting (○) positions, and the supine to sitting flux ratios (c and d), at unheated skin temperature and at 36°C, respectively. The error bars up and down indicate the 75th and 25th percentile intervals, respectively. The broken lines at a ratio of 1 in (c) and (d) indicate equal flux in both positions. Group I, ABI < 20%; group II, ABI 20–39%; group III, ABI 40–59%; group IV, ABI 60–95%; group V, ABI 96–123%.
significantly ($P<0.001$) with a reduction of the ABI (at unheated skin temperature from about 0.85 V in group V to 0.03 V in group I, and at 36°C from 1.15 to 0.03 V). A difference, however, was found between rest and peak flux data in groups IV and V at unheated skin temperature: median peak flux values in the sitting position were virtually identical with those in the supine position, resulting in vasoconstriction indices of about 1. In patients with lower ABIs, the median vasoconstriction indices were lower than 1, indicating that peak flux was higher while sitting. In group I, local skin heating to 36°C did not have any influence on peak flux. Also, the vasoconstriction indices did not show a significant change upon increasing temperature in any of the groups.

Ttpflux and T50%flux increased progressively with decreasing ABI at both temperatures and in both positions ($P<0.005$). At unheated skin temperature, Ttpflux in the sitting position rose from 8 s in group V to 165 s in group I and in the supine position from 32 s in group V to 237 s in group II. In group I, Ttpflux could not be assessed, because of the no-flow state. After local skin heating, Ttpflux values rose from 11 to 131 s while sitting and from 59 to 126 s while supine.

Transcutaneous oximetry

Fig. 3 shows the individual and group median tcPO$_2$ data at rest at 37°C (Fig. 3a) and at 44°C (Fig. 3b), for both positions. Figs 3(c) and 3(d) present the vasoconstriction indices at rest at 37°C and 44°C, respectively. Tissue oxygenation (37°C) was higher in the supine than in the sitting position in the patients with the highest ABIs (see Fig. 3a). Below an ABI of 70%, oxygenation decreased gradually towards median values of 1 mmHg (supine) and 2 mmHg (sitting) in group I. In this group, tcPO$_2$ was higher in the sitting position ($P<0.05$), as can be seen more clearly in Fig. 3(c), which shows ratios below unity in group I. Again, all patients having an ABI below 30% had a vasoconstriction index lower than 1.

Heating of the skin (Fig. 3b) caused a four- to seven-fold increase in oxygenation in the patients with higher ABIs ($P<0.005$), but the oxygenation in group I was virtually unaltered. In all groups oxygenation was now found to be persistently lower in the supine than in the sitting position ($P<0.005$, except group I): Fig. 3(d) shows that all individual ratios, which were above unity in Fig. 3(c), are now close to, or below, unity.

The peak tcPO$_2$ results were very similar to the rest tcPO$_2$ results. Median values at 37°C were comparable: in groups II–V about 20 mmHg in the supine position and about 16 mmHg in the sitting position and in group I decreasing to 1 mmHg and 9 mmHg, respectively. Local skin heating induced an increase in peak tcPO$_2$ (to 60 mmHg) only in the upper four ABI groups ($P<0.005$). The vasoconstriction indices were also similar.

TtpPO$_2$ and T50%PO$_2$ were prolonged at lower perfusion pressures: the median TtpPO$_2$ in the supine position at 37°C varied from 106 s (group V) to 212 s

![Fig. 3](image-url)

*Fig. 3.* Scatterplots of rest tcPO$_2$ (a and b) in supine (●) and sitting (○) positions, and the supine to sitting tcPO$_2$ ratios (c and d), at 37°C and 44°C, respectively. The error bars up and down indicate the 75th and 25th percentile intervals, respectively. The broken lines at a ratio of 1 in (c) and (d) indicate equal tcPO$_2$ in both positions. Group I, ABI <20%; group II, ABI 20–39%; group III, ABI 40–59%; group IV, ABI 60–95%; group V, ABI 96–123%.
(group I) and at 44°C from 180 s (group V) to 366 s (group II). In the sitting position at 37°C, these values varied from 120 s in group V to 284 s in group I and at 44°C from 141 s in group V to 250 s in group I.

Finally, LDF and tcPO₂ data were combined in order to compare both techniques and to obtain an overall view of the influence of local skin heating (Fig. 4). LDF indices tended to decrease before the tcPO₂ indices with decreasing ABI. A clear overlap was found in the highest ABI groups between the vasoconstriction indices of LDF at 36°C and tcPO₂ at 37°C, although these instruments measure quite different parameters.

**DISCUSSION**

Skin perfusion and oxygenation are markedly reduced and posturally induced microvascular constriction mechanisms cease to function when arterial disease of the legs progresses beyond a certain limit, as was found in all patients with an ABI below approximately 30%. In these patients, local vasoconstriction mechanisms are overruled, as an increase in local skin temperature does not improve skin perfusion and oxygenation at rest, while these parameters increase upon dependency.

Microvascular reactivity was similar in patients with mild ischaemic disease of the legs and in asymptomatic control legs. In all patients with an ABI of 55% or more, skin perfusion and oxygenation were reduced after changing from the supine to the sitting position. Thus, disturbances of posturally induced vasoconstriction are confined to critically low blood pressure levels. In the present study, ABI could not predict the effectiveness of microvascular constriction mechanisms below a value of 60%. In this range, laser Doppler fluxmetry, especially when performed without local heating of the skin, is a useful tool with which to assess the presence or absence of microvascular constriction upon dependency by calculating the vasoconstriction index (i.e. the supine to sitting ratio) from measurements in the supine and the sitting position. Transcutaneous oximetry at 37°C also appeared to be able to be tested the effectiveness of postural vasoconstriction. However, using this technique, the transition from intact to disturbed vasoconstriction is not so clear-cut, because vasoconstriction mechanisms are already suppressed by the required heating of the skin.

In patients with severe leg ischaemia (ABI < 30%), skin perfusion was low, but always slightly enhanced when changing to the sitting position, indicating disturbed microvascular constriction mechanisms. This is in accordance with previous investigations using laser Doppler fluxmetry and transcutaneous oximetry [17]. By means of capillary video-microscopy, vasoconstriction responses at the capillary level were found to be disturbed below an ABI of 20% [16]. This suggests that in patients with an ABI below 30%, capillary perfusion is sustained at the cost of the perfusion in deeper skin layers, in an attempt to preserve nutritional blood flow. These findings may explain the relief of pain on dependency as is seen in some of these patients.

Stepwise local skin heating [unheated (29°C) to 36°C/37°C to 44°C] in patients without or with mild ischaemia causes a stepwise counteraction of microvascular constriction on dependency, resulting in a progressively higher perfusion and oxygenation in the sitting than in the supine position, a situation similar to that in severely diseased patients before heating of the skin. In these patients perfusion and oxygenation at rest cannot be improved anymore by a temperature increase or the induction of reactive hyperaemia, obviously because the microvessels are already completely dilated. This is supported by the finding that local skin heating did not enhance reactive hyperaemia in these patients.

The larger arteries (e.g. > 500 µm) of the foot between the ankle and the toe are not likely to have any regulatory influence on microvascular perfusion, because the induction of reactive hyperaemia showed no additional increase in perfusion or oxygenation as compared with the increase observed during local skin heating, indicating that local mechanisms prevail in this regulation.

In this study, the small number of diabetic patients per ABI group apparently did not influence microcirculatory reactivity differently. It is also conceivable that in both types of patients atherosclerosis is the dominating disease.

tcPO₂ measurements were performed on the dorsum of the foot, whereas the laser Doppler instrument was applied to the pulp of the great toe, in which arteriovenous anastomoses are more numerous. Despite this difference, there is a close resemblance between the results obtained with laser Doppler fluxmetry at 36°C and transcutaneous oximetry at 37°C in the highest ABI groups. This suggests that tcPO₂ measurements, even at this temperature, do not only provide information about the capillary perfusion [20, 29], but also about the perfusion of deeper layers of the skin [30]. Reduction of skin perfusion, however, occurred at a higher ABI than the reduction of skin oxygenation, which is more in accordance with capillary microscopy findings [16]. This indicates that transcutaneous oximetry does not measure tcPO₂ in the skin layers that can be reached by laser Doppler fluxmetry.
Because of the clear inverse correlation between the time to peak, as measured by both instruments, and the ABI, these parameters appear to be more informative about the macrocirculation than the microcirculation.

In conclusion, the ABI is widely used in clinical vascular practice as an objective parameter with which to assess peripheral vascular disease. The findings in the present study show that an ABI higher than 55% concurs with intact microvascular reactivity in patients with mild leg ischaemia. However, below 55%, the ABI can not portend the existence of disturbed constriction mechanisms. Therefore, in more severe leg ischaemia, only the exemplified comparison of skin microcirculatory perfusion in the supine versus the sitting position using laser Doppler fluxmetry (at unheated skin temperature) and transcutaneous oximetry (at 37°C) is useful to investigate to what extent microvascular constriction responses upon dependency are disturbed. Therapeutically, this disturbance indicates that the limb is in jeopardy of becoming critically ischaemic and therefore urges the necessity of improving arterial circulation. In future longitudinal studies, it remains to be proven whether disturbed posturally induced microcirculatory reactivity is of prognostic value. The possibility of counteracting microvascular constriction by only a local stimulus suggests that these constriction mechanisms are mainly local phenomena.

REFERENCES

31. Fontaine, R., Riveaux, R., Kim, M. & Kieny, B. Results of the observations hyperaemiantes (sympathectomies lobomaries et artériectomies) dans les oblitérations artérielles chroniques spontanées des membranes. Rev. Chr. 1953; 72, 204–30.