Measurement of Foot Artery Blood Pressure by 
Micromanometry in Normal Subjects and in Patients 
with Arterial Occlusive Disease

ALFRED BOLLINGER, M.D., JEAN PIERRE BARRAS, M.D., AND FELIX MAHLER, M.D.

SUMMARY  Blood pressure was measured continuously in the 
posterior tibial or dorsalis pedis arteries using an isovolumetric sys-
tem (steel cannulas of 0.18 mm, external diameter). The systolic 
values in the ankle arteries of 13 normal subjects at rest (154.3 ± 
22.3 mm Hg) exceeded the systolic arm pressure (128.9 ± 20.1 mm 
Hg, P < 0.001), while diastolic values (69.9 ± 8.7 mm Hg) were 
not significantly different from the arm pressure. In 13 patients 
with arterial occlusive disease (AOD) the dicrotic notch, normally situated 
close to the footpoint of the downslope, was either displaced upward 
or abolished. Both mean systolic and diastolic values (94.9 ± 35.9 
mm Hg and 62.5 ± 18.5 mm Hg, respectively) and also mean pressure amplitude were reduced compared to the corresponding arm 
values (158.5 ± 28.2 mm Hg and 87.7 ± 6.0 mm Hg, respectively, all 
P < 0.001). Comparison between the systolic pressure values 
measured by micromanometry and by an indirect technique using 
Doppler ultrasound showed a good correlation (r = 0.87). During 
postocclusive reactive hyperemia, the initial pressure values were 
markedly diminished in normal subjects and reached control values 
within 40 sec. In patients with AOD, however, this reduction in 
pressure was more pronounced and prolonged. Flow measurements 
using plethysmography showed flow diversion from the foot to the 
cafe as long as pressure values ranged below 40 mm Hg. This almost 
painless method appears useful for experimental and diagnostic 
studies in low pressure areas of the peripheral circulation.

INDIRECT SYSTOLIC BLOOD PRESSURE measurements 
in the low pressure area distal to arterial stenoses or 
occlusions have been introduced in routine clinical 
diagnosis. The methods currently used include mercury 
strain gauges,1,4 Doppler ultrasound,9,10 Xenon13,14 clearance,10 
spectroscopic procedures,11 and photoelectric devices.12 All these techniques depend on determination of 
the onset of blood flow at a given cuff pressure as an 
indicator for the intravascular pressure. To the present, direct measurements required traumatic approaches such as surgical 
exposure of the dorsalis pedis artery.13

A recently developed isovolumetric manometer connected 
to a steel cannula allows percutaneous determination of 
blood pressure in small blood vessels by an almost 
painless procedure.14 In the present study, pressure recordings 
were obtained not only in the foot arteries of normal subjects but also in poststenotic dorsalis pedis and posterior 
tibial arteries, previously not accessible to direct puncture. 
Since this method permits the continuous recording of 
pressure curves, systolic and diastolic values were studied at 
rest and during reactive hyperemia after three minutes of 
arterial occlusion. In order to evaluate the accuracy of an ind-
direct technique using Doppler ultrasound, the direct measurements were used for comparison. In some ex-
periments, the phenomenon of hemometakinesia was ex-
amined correlating ankle blood pressure during the course of 
reactive hyperemia to calf and foot blood flow measured 
during the same conditions.

Methods

Patients and Normal Subjects

Thirteen patients with arteriosclerosis obliterans of the 
lower limbs were included in the study (ten male and three 
female patients with a mean age of 58.2 years, range 35-81 
years). The main complaint was intermittent claudication. 
Patients with rest pain or gangrene were not included. In 
each patient a thorough clinical and laboratory examination 
was performed including electronic oscillography,15 post-
stenotic systolic blood pressure measurements with Doppler 
ultrasound9,9 and arteriography.

The findings of ankle pulse palpation and of arteriography 
are listed in table 1. In seven patients, both dorsalis 
pedis and posterior tibial pulses were absent and in the 
remaining six patients barely palpable. Even in the cases 
with absent ankle pulses, flow signals could be detected by 
Doppler ultrasound. One of these patients presented marked 
edema of the lower leg. In an additional patient, medial 
calcinosis of the calf and foot arteries was diagnosed (diffuse “pipe-stem”-calcifications on the roentgenogram). There 
were no arterial occlusions in this case. The control group 
consisted of 13 healthy male volunteers with a mean age of 
38.8 years (range 24-60 years). Their pedal pulses were 
easily palpable.

Blood Pressure Measurements

The direct determinations of blood pressure were 
performed by an isovolumetric manometer connected to a 
steel miconeedle with an external diameter of 180 µm 
and an internal diameter of 76 µm. The system contains a man-
ometer membrane and a mechanism of volume compensation 
which is operated by a feed-back circuit. Technical details 
have been described previously.14,16

Pressure curves of identical configuration and values 
within ±1 mm Hg were obtained when measured si-
taneously in a brachial artery with the micromanom-
eter method and a conventional transducer.16 Mean 
systolic ankle blood pressure evaluated by the indirect 
strain-gauge technique was 8.2 ± 9.1 mm Hg (N = 13) 
lower than mean intra-arterial systolic blood pressure.16

The patients were examined after resting in the supine 
position for 15–30 minutes. Location of the dorsalis pedis 
and posterior tibial arteries was made when a Doppler ultra-
sound flow velocity probe (Parks Electronics, model 806) in-
dicated pulsations were faint or absent. At the point where maximal arterial flow signals were detected, the microneedle was inserted through the intact skin after sterilizing the area. No anesthesia was necessary. The needle was slowly advanced in the direction of the vessel until an arterial pressure signal was obtained as visualized on the oscilloscope. As soon as stable conditions were reached, blood pressure was registered on UV-photographic paper (Siemens Oscillofot 16) or on a forced-ink pen recorder (Gould-Brush, model 480). In the normal subjects with palpable foot arteries the puncture by a trained observer required not more than five minutes. The time needed in patients with faint or absent pulsations varied from one to 20 minutes. In four cases with severe ischemic disease not included in this series pressure measurements were not possible.

In three healthy subjects and in 12 patients, immediately after registration of resting pressure, indirect measurements of systolic ankle pressure were performed by a trained observer. A standard pressure cuff around the ankle was inflated and the onset of blood flow velocity in the posterior tibial or the dorsalis pedis artery determined using a Doppler ultrasound flow detector (Parks Electronics, model 806). A mean of three to five determinations was used for the comparison with the average of the directly measured systolic pressure.

In three normal subjects and in six patients a previously applied pressure cuff placed above the knee was then suddenly inflated by a reservoir to 300 mm Hg. The arterial occlusion was maintained for three minutes. After the sudden release of cuff pressure, the changes in poststenotic blood pressure were continuously recorded until the resting values were attained or surpassed. For these registrations a paper speed of 1 mm/sec was used. At the end of the examination, the microneedle was withdrawn. Compression of the artery was not necessary, macroscopic bleeding did not occur. Arm pressure was measured by cuffs using the Korotkoff method.

In ten of the 13 subjects the intra-arterial pressure of the cubital artery was determined by micromanometry as described for the foot arteries.

**Blood Flow Measurements**

In three normal subjects and in four patients calf and foot blood flow were measured by venous occlusion plethysmography in a second experiment at intervals of 30 sec before and during reactive hyperemia, while the cannulae still remained in the artery for intermittent pressure measurement. Mercury-in-silastic-strain gauges were used to assess the limb volume changes (U.G. Gutmann, Eurasburg, Western Germany). One venous occlusion cuff was placed above the knee, a second above the ankle. The strain gauges were adapted to the calf at the site of maximal circumference and to the foot. The venous occlusion cuffs were suddenly inflated from a pressure reservoir to 40 mm Hg and the plethysmographic curves were registered on a two-channel recorder (Servogor Metrawatt). In the intervals

<table>
<thead>
<tr>
<th>Patient/age/sex</th>
<th>Pedal pulses</th>
<th>Angiography</th>
<th>Main complaint</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/O.M. /81/F</td>
<td>Absent</td>
<td>Occlusion of right superficial femoral artery and posterior tibial artery</td>
<td>Intermittent claudication after 50 m</td>
</tr>
<tr>
<td>2/T.M. /41/M</td>
<td>Weakly palpable</td>
<td>Segmental occlusion of right common iliac artery</td>
<td>Intermittent claudication after 100-200 m</td>
</tr>
<tr>
<td>3/F.H. /35/M</td>
<td>Palpable</td>
<td>Segmental stenosis of right common iliac artery (2 mm of internal diameter)</td>
<td>Intermittent claudication after strenuous exercise only</td>
</tr>
<tr>
<td>4/M.H. /70/M</td>
<td>Absent</td>
<td>Occlusion of right femoro-popliteal segment</td>
<td>Intermittent claudication after 500 m</td>
</tr>
<tr>
<td>5/D.B. /47/M</td>
<td>Weakly palpable</td>
<td>Stenosis of right superficial femoral artery</td>
<td>Intermittent claudication after 450 m</td>
</tr>
<tr>
<td>6/C.M. /57/M</td>
<td>Absent</td>
<td>Stenoses of abdominal aorta and both iliac arteries, occlusion of right superficial femoral artery</td>
<td>Intermittent claudication after 200 m</td>
</tr>
<tr>
<td>7/D.R. /69/M</td>
<td>Weakly palpable</td>
<td>Stenosis of left superficial femoral artery</td>
<td>Intermittent claudication after 700 m</td>
</tr>
<tr>
<td>8/B.G. /41/M</td>
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<td>Occlusion of right common femoral artery</td>
<td>Intermittent claudication after 300 m</td>
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<td>9/E.A. /49/M</td>
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<td>Stenosis of left common iliac and left superficial femoral arteries</td>
<td>Intermittent claudication after 50 m</td>
</tr>
<tr>
<td>10/F.P. /56/M</td>
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<td>Stenosis of right superficial femoral artery</td>
<td>Intermittent claudication after 500 m</td>
</tr>
<tr>
<td>11/F.B. /75/F</td>
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<td>Occlusion of right superficial femoral artery</td>
<td>Intermittent claudication after 50 m</td>
</tr>
<tr>
<td>12/H.H. /66/M</td>
<td>Weakly palpable</td>
<td>Stenosis of left anterior tibial and fibular artery, occlusion of anterior tibial artery (proximal part)</td>
<td>No intermittent claudication because of concomitant coxarthrosis</td>
</tr>
<tr>
<td>13/S.E. /69/F</td>
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<td>Stenosis of right common iliac artery, occlusion of right superficial femoral artery</td>
<td>Intermittent claudication after 200 m</td>
</tr>
<tr>
<td>14/B.R. /75/M</td>
<td>Palpable</td>
<td>Calcification of calf and foot arteries (medial calcinosis). Peripheral arteries patent.</td>
<td>No intermittent claudiation</td>
</tr>
</tbody>
</table>
between the blood flow measurements direct blood pressure was monitored continuously with deflated occluding cuffs.

**Results**

**Blood Pressure Measurement at Rest**

Blood pressures of the ankle arteries were obtained in 13 healthy subjects without impairment of peripheral arterial circulation (table 2). The systolic values (154.3 ± 22.3 mm Hg) of the dorsalis pedis or the posterior tibial artery exceeded in each of the 13 subjects the values determined from the arm (average 128.9 ± 20.1 mm Hg, P < 0.001), while the diastolic ankle pressures (average 69.9 ± 8.7 mm Hg) were not significantly different from the arm values (71.3 ± 7.8 mm Hg). Thus, pressure amplitude in the normal subjects was larger in the ankle artery (84.4 ± 15.6 mm Hg) than in the brachial artery (57.6 ± 16.4 mm Hg, P < 0.001).

**Table 2. Direct and Indirect Blood Pressure in the Ankle Arteries of Patients and Normal Subjects**

<table>
<thead>
<tr>
<th>Arterial Occlusive Disease</th>
<th>Blood pressure (mm Hg)</th>
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<tr>
<td></td>
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<tr>
<td></td>
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</tr>
<tr>
<td>1 Arterial Occlusive Disease</td>
<td>1 81 F</td>
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<tr>
<td>2</td>
<td>41 M</td>
</tr>
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<td>3</td>
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<td>14</td>
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<td>sd</td>
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<tr>
<td>16</td>
<td>P</td>
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<table>
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<th>Mediasclerosis (Patent Arteries)</th>
<th>Blood pressure (mm Hg)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
</tr>
<tr>
<td>Normal Subjects</td>
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</tr>
<tr>
<td>18</td>
<td>24 M</td>
</tr>
<tr>
<td>19</td>
<td>26 M</td>
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<td>34 M</td>
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<td>24</td>
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</tr>
<tr>
<td>25</td>
<td>36 M</td>
</tr>
<tr>
<td>26</td>
<td>35 M</td>
</tr>
<tr>
<td>27</td>
<td>60 M</td>
</tr>
<tr>
<td>28</td>
<td>57 F</td>
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<tr>
<td>29</td>
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<td>M</td>
</tr>
<tr>
<td>31</td>
<td>sd</td>
</tr>
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<td>32</td>
<td>P</td>
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</tbody>
</table>

Abbreviations: M = mean; d.p. = dorsalis pedis artery; p.t. = posterior tibial artery; sd = standard deviation; NS = not significant.

Figure 1 shows an original recording of a pressure curve from a normal subject and one patient in whom media-sclerosis of the lower leg arteries was diagnosed by the roentgenogram, but the ankle pulses were vigorously palpable and the pulse tracings registered from the big toe by electronic oscillography were normal. The difference between arm and ankle pressure and the pressure amplitude (205/72 mm Hg, fig. 1) are remarkable in this case with rigid lower leg arteries.

Poststenotic blood pressure was measured in 13 patients with arterial occlusive disease (table 2). In every instance, the systolic and, with one exception, also diastolic values were lower than the corresponding arm artery pressures. The mean systolic (158.5 ± 28.2 mm Hg) and diastolic values (87.7 ± 6.0 mm Hg) at the arm were significantly higher than in the poststenotic ankle arteries (94.9 ± 35.9 mm Hg systolic and 62.5 ± 18.5 mm Hg diastolic, P < 0.001). Furthermore, the pressure amplitude of the ankle arteries (32.5 ± 20.1 mm Hg) was significantly reduced compared with the arm values (70.8 ± 28.3 mm Hg, P < 0.002). There were considerable inter-individual differences in these measures, reflecting the differences in severity of arterial occlusive disease among the patients. The lowest value measured in the dorsalis pedis artery was 39/34 mm Hg. The arteriogram of this patient showed a complete occlusion of the common femoral artery including the proximal part of the profunda femoris artery. The other extreme was represented by a patient with a stenosis of the left common iliac artery causing intermittent claudication only after strenuous exercise. His blood pressure in the posterior tibial artery amounted to 166/98 mm Hg (arm pressure 185/85 mm Hg). Figure 2 shows the original tracings of three patients with different degrees of arterial insufficiency. In most of the patients, no diacrotism was present. A dicrotic wave was found only in cases with very well compensated occlusions or stenoses. The dicrotic wave, however, was not located near the diastolic base line as in the normal subjects, but at about half pressure amplitude level.

In order to evaluate the accuracy of indirect ankle blood pressure determinations, directly and indirectly measured systolic ankle blood pressures were compared. Figure 3 shows the correlation between direct and indirect measurements. The agreement is good (r = 0.87) and the differences between the two methods within the same artery did not exceed 15 mm Hg in general. In two instances, however, the systolic ankle blood pressure was markedly overestimated by the Doppler ultrasound technique (cases 11 and 14, table 2). The first of these patients had severe edema of the lower leg and the second patient had "pipe-stem" calcifications of the anterior and posterior tibial arteries due to medial calcinosis. At values above 100 mm Hg the pressures obtained by the indirect method were underestimated compared to those determined by micromanometry, while at lower pressures overestimation predominated.

**Blood Pressure during Reactive Hyperemia**

In three normal subjects, blood pressure in the ankle arteries fell slowly after application of the suprasystolic pressure and finally reached values that were near zero. Within the first heart cycle after the release of the cuff, positive pressures with a reduced pressure amplitude were
recorded. The systolic pressure values measured at the beginning of reactive hyperemia in the normal subjects were reduced by 37% or more of resting values (table 3). The dicrotic wave was absent during this initial period. Pressure increased steadily, and within about 40 seconds the initial values of pressure and amplitude were reached again and dicrotism was restored. Figure 4, upper panel, shows original tracings from a normal subject before and during reactive hyperemia.

The patients showed qualitatively the same reaction. The pressure reduction during reactive hyperemia, however, was quantitatively more pronounced and prolonged (table 3). After release of the arterial occlusion cuff, the poststenotic pressure rose only slowly. In some cases, blood pressure amplitude was initially abolished and developed progressively toward the initial values. Figure 4, lower panel, shows an example for the pressure reaction during reactive hyperemia in a patient with arterial occlusive disease.

Comparison of Poststenotic Blood Pressure and Flow

Alternated measurements of poststenotic pressure and of foot and calf blood flow were performed to analyze the
pressure-flow relationship during reactive hyperemia. Measurements were obtained in three normal subjects and in four patients. The results are listed in table 3. In the patients no. 4, 5 and 7, no or very low foot flow values were measured as long as the ankle pressures were below 40 mm Hg, while increased flow was recorded simultaneously at the calves. Typical examples are represented in figure 5. The decrease in blood pressure during the reactive hyperemia phase was associated with an increase in calf blood flow. As expected reactive hyperemia flow values in patients did not reach the level attained by the normal subjects. Peak flow was reduced and the time course of reactive hyperemia delayed. Immediately after release of the arterial occlusion cuff, the foot blood flow did not show a uniform pattern. In patient 2 the foot peak flow was measured within the first 10 sec, whereas in patient 4 there was no measurable flow at that time. Blood pressure in the ankle arteries was reduced to 52/47 mm Hg in the former patient and to 17/14 mm Hg in the latter during the early phase of reactive hyperemia (fig. 5, lower panels).

Discussion

In normal subjects, pressure curves of the dorsalis pedis artery have been recorded by means of direct puncture. Using micromanometry it was possible to measure blood pressure even in foot arteries with absent or faint pulsations, as was shown in 13 patients (table 2, fig. 2) and reported in two preliminary examples. The formal appearance of the pressure curves in these patients reflected the hemodynamic significance of the leg artery stenoses or occlusions. In cases with mild ischemia, the dicrotic notch, which is normally located at the base of the pressure curve of the foot arteries was less prominent and displaced upward on the descending limb of the pressure pulse. In the pressure curves of patients with severe ischemia, however, the dicrotic notch was not discernible and the pressure amplitude was markedly decreased or nearly abolished (lowest pressure values encountered at rest: 39/34 mm Hg), a finding which confirms the results of measurements after surgical exposure of the pedal arteries.

Measurements after a circulatory arrest of specific duration or after exercise are more sensitive than measurements at rest for the evaluation of the poststenotic circulatory dynamics, as is known from studies using indirect methods. In agreement with the results of Wallace and Stead and of Rowell and coworkers, who directly recorded radial artery pressure after arterial occlusion, an initial decrease of pulsatile blood pressure was found during reactive hyperemia even in healthy volunteers (table 3, fig. 4, upper panel). The pressure reduction lasted no more than 40 sec after three minutes of circulatory arrest in normal subjects. In the patients with arterial occlusive disease, however, the pressure fell to lower absolute values than in normals and the initial postocclusive pulse excursions were small or even lacking (fig. 4, lower panel). The resting values
were not reached 45 sec after release of the occlusion cuff. The duration of the pressure response increased with increasing severity of ischemia. Although in a group of patients (nos. 2, 4, 7) the resting pressure values were comparable, their reaction to arterial occlusion showed considerable variability. This observation is consistent with the fact that the functional importance of arterial stenoses is better discriminated by measurements during provoked sub-

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**FIGURE 5.** Diagrammatic representation of direct systolic ($P_s$) and diastolic ($P_d$) ankle pressure and blood flow in the calf ($Q_c$) and in the foot ($Q_f$) during resting state and during reactive hyperemia. Upper left panel shows the course of an experiment in a normal subject (No. 15, arm blood pressure 110/60 mm Hg), lower left panel in a patient with moderate arterial occlusive disease (No. 2, arm blood pressure 130/85 mm Hg), and lower right panel in a patient with more severe arterial occlusive disease (No. 4, arm blood pressure 190/80 mm Hg). In the last patient reactive hyperemia during the initial phase was confined to the calf. Foot blood flow rose only after the peak flow at the calf was reached (phenomenon of hemometakinesia).

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**TABLE 3. Blood Pressure and Flow Measurements During Reactive Hyperemia in Patients and Normal Subjects**

<table>
<thead>
<tr>
<th>No.</th>
<th>P</th>
<th>C/F</th>
<th>Q (ml/min)</th>
<th>P</th>
<th>C/F</th>
<th>Q (ml/min)</th>
<th>P</th>
<th>C/F</th>
<th>Q (ml/min)</th>
<th>P</th>
<th>C/F</th>
<th>Q (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>102/62</td>
<td>3.5</td>
<td>1.3</td>
<td>52/47</td>
<td>14.0</td>
<td>4.3</td>
<td>74/54</td>
<td>4.8</td>
<td>3.2</td>
<td>104/72</td>
<td>3.3</td>
<td>2.3</td>
</tr>
<tr>
<td>4</td>
<td>98/67</td>
<td>1.9</td>
<td>0.7</td>
<td>17/14</td>
<td>11.2</td>
<td>n.m.</td>
<td>20/17</td>
<td>—</td>
<td>—</td>
<td>54/49</td>
<td>12.8</td>
<td>1.7</td>
</tr>
<tr>
<td>5</td>
<td>83/60</td>
<td>2.0</td>
<td>1.2</td>
<td>25/22</td>
<td>14.0</td>
<td>n.m.</td>
<td>30/26</td>
<td>9.3</td>
<td>0.9</td>
<td>54/46</td>
<td>3.4</td>
<td>2.2</td>
</tr>
<tr>
<td>7</td>
<td>107/65</td>
<td>4.0</td>
<td>0.9</td>
<td>20/22</td>
<td>12.7</td>
<td>n.m.</td>
<td>36/29</td>
<td>—</td>
<td>—</td>
<td>65/58</td>
<td>6.8</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Abbreviations: P = direct pressure measurement in mm Hg; Q = flow measured by venous occlusion plethysmography in ml/100 ml min; C = calf blood flow; F = foot blood flow; n.m. = not measurable.
maximal vasodilatation than during resting conditions.7,8
Similar conclusions were drawn from a study in patients with aortoiliac disease evaluating femoral artery pressure during reactive hyperemia.21
The correlation between blood pressure of the ankle arteries measured by micromanometry and by an indirect technique using Doppler ultrasound showed a good agreement of the systolic values with exception of two cases presenting with the marked edema or Moenckeberg's sclerosis of the lower leg arteries (fig. 3). The last two conditions do not permit an appropriate hemodynamic evaluation by indirect methods. Medial calcinosis may be severe enough to prevent compression by a standard blood pressure cuff (pseudohypertension).22,23 Direct measurements as used in this study are necessary to avoid falsely high readings.
In three normal subjects and in four patients, calf and foot blood flow during reactive hyperemia were measured by venous occlusion plethysmography alternating with measurements of the ankle blood pressure. In agreement with earlier results,24-26 the postocclusive calf flow increase was reduced and delayed in the patients with arterial occlusive disease. In the cases with a pressure fall to 30 mm Hg or more, low or not measurable foot blood flow was observed, while hyperemic values were recorded simultaneously at the calf. This phenomenon has been described as hemometakinesia27 or blood flow diversion.28 Although flow measurements using venous occlusion plethysmography are open to criticism when venous occluding pressure is within the range of the arterial pressure and therefore possibly influencing the arterial inflow to the foot, the presence of such low pressure values offers a physiological explanation for the phenomenon of hemometakinesia. If local perfusion pressure is reduced initially during reactive hyperemia to the range of the capillary or venular pressure, the arterio-venous pressure gradient is abolished and blood flow ceases. In the postocclusive pressure range of about 30-40 mm Hg, hemometakinesia could also be explained by a critical closure of the resistance vessels.29
The measurement of poststenotic blood pressure by transcutaneous micromanometry offers several advantages as compared to indirect methods. It permits continuous registration of blood pressure curves suitable for formal analysis. Systolic as well as diastolic values are obtained accurately even in very low pressure ranges, where indirect methods are questionable. The method described appears to be a promising investigative tool for use in pathophysiological problems in the low pressure areas distal to arterial occlusions and for evaluation of pharmacological agents. In addition, it also proved useful in clinical diagnosis, when local conditions such as edema or mediasclerosis render the indirect measurements unreliable.

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