Effect of Temperature on Digital Systolic Pressures in Lower Limb in Arterial Disease

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Background. Toe pressures correlate with the chances of healing of skin lesions and with the risk of amputation and mortality. They are not affected by incompressibility of the tibial vessels, which may render ankle pressures inaccurate and reflect the overall arterial obstruction down to the digits. Digital pressures, however, may be affected by temperature.

Methods and Results. Measurements under routine laboratory conditions were compared with those at controlled temperatures in 77 limbs with arteriosclerosis obliterans. The desired local temperatures were attained by perfusing water through the cuffs for 7 minutes before the measurements while the flow was interrupted by a tourniquet to allow equilibration of the toe and water temperatures. Mean toe pressure of 34±4 mm Hg at 10°C was significantly lower (p<0.001) than the values during routine measurements (57±4 mm Hg) and at 30°C (69±4 mm Hg). The pressure at 30°C was 10±3 mm Hg higher than during routine measurements when initial digit temperature was below 30°C (p<0.01) but not when it was higher. Measurements at 3°C increments from 27°C to 39°C showed progressive increase in pressure (p<0.01).

Conclusions. Pressures at 36°C and 39°C were the highest but were not significantly different from each other. When toe temperature is low under routine conditions, the measured pressure may be falsely low, probably because of an effect on the main digital arteries that leads to delayed opening during deflation of the cuffs. Measurements at warm local temperature will increase the accuracy of the assessment of the severity of the arterial obstruction and may improve prediction of spontaneous healing of skin lesions.

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Key Words • arteriosclerosis • arterial occlusive diseases • peripheral vasculature • blood pressure

Noninvasive measurements of systolic pressure at the ankles represent an established and valuable method for assessment of obstruction in the arteries of the lower limbs because they correlate well with the angiographic findings1-3 and with the severity of symptoms in patients with arteriosclerosis obliterans.1,4 However, pressures measured at the ankles have limitations. They may be inaccurate because of the incompressibility of the tibial vessels caused by medial calcification in some patients with diabetes mellitus or renal disease.1,3,5 Also, they usually do not detect obstruction if only one or two of the three tibial arteries are affected1,3 and do not reflect the occlusive process in the foot itself. These facts explain why distal skin ulcers or gangrene often do not heal despite the presence of a high ankle pressure.3,6 The limitations of the ankle pressures led to increased interest in measurements of systolic pressure in the toes, which reflect the overall obstruction of the limb arteries down to the digits and probably are not affected by the vessel incompressibility.1,7-12 Toe systolic pressures were also shown to correlate well with the angiographic evidence of arterial obstruction and with the severity of symptoms.7 They were found to be useful in predicting chances of spontaneous healing of skin lesions and of toe or foot foot amputations.6,10,12,13 Toe pressure of 30 mm Hg or greater is associated with spontaneous healing of lesions in over 90% of cases, but when it is lower, the chances of healing are uncertain.3,6,10,13 The reason healing occurs in some limbs with lower pressure is not clear, but it may be related to the fact that digital pressures are known to be affected by changes in the vasomotor state induced, for example, by body heating or cooling.14,15 More recently, it was shown that finger pressures fell precipitously during local cooling of the digits, especially in patients with vasospastic Raynaud's syndrome, and there was frequently a loss of measurable pressure.16,17 In some patients, body cooling resulted in the loss of measurable finger pressure without the application of local cooling; the presence of organic arterial obstruction exaggerated the effect of cooling.18

We recently demonstrated that toe systolic pressures were also significantly lower during body cooling in normal subjects and patients with Raynaud's syndrome in the toes and that this effect was increased by the addition of local cooling.19 The present study was undertaken to assess the effect of local temperature on measurements of systolic pressures in the toes in patients with arteriosclerotic occlusion. We demonstrated that when the toes were cold, local warming increased the measured pressure, a finding that has important implications for more accurate determination of the
severity of arterial obstruction and of the prognosis for healing of skin lesions.

Methods

Subjects

The results are based on measurements in 77 limbs of 39 patients with arteriosclerosis obliterans. All subjects gave informed consent, and the protocol was approved by the Faculty Committee on the Use of Human Subjects in Research, the Faculty of Medicine, University of Manitoba. The presence of peripheral arterial disease was determined by clinical assessment and by abnormal ankle and toe pressures measured by the routine methods previously described.1–3,7 No patients had skin lesions or pain at rest in the feet, but all had typical symptoms of intermittent claudication. All patients had toe systolic pressure index below 0.64 and ankle index below 0.97, the lower limits of normal established in our laboratory.1,7 The mean toe index of 0.27±0.04 and ankle index of 0.60±0.03 in the leg with more severe disease were grossly abnormal and similar to or slightly lower than the values reported previously in groups of patients with intermittent claudication.3,7,10 The average age was 68±1 year. Ten patients had diabetes mellitus, 16 were smokers, and 13 were taking vasodilator drugs such as calcium channel blockers.

Techniques and Experimental Protocols

Studies were carried out in supine subjects who had refrained from smoking and eating for at least 2 hours. They wore hospital gowns and rested for not less than 30 minutes. Room temperature was maintained relatively constant within ±1°C and averaged 21°C.

Determination of systolic pressures. Pressures were measured in the great toe during deflation of a 3-cm-wide pneumatic cuff applied to the base of the digit by previously described techniques.7,19 The pressure in the cuff at the time of resumption of arterial flow into the digit distal to the cuff was taken as the systolic pressure. The resumption of arterial inflow was indicated by an increase in volume of the digit using a photocell plethysmograph (Medasonics) attached to the plantar aspect of the distal phalanx of the toe with double-sided Scotch tape. The quality of the systolic end points was considerably improved by rendering distal parts of the toes relatively bloodless by the tight application of a rubber dam before the inflation of the cuffs and application of the sensors. The outputs from the plethysmograph and from the pressure transducer (Statham PM6TC) used to record pressure in the cuffs were connected to a multichannel recorder (Beckman R-611). During the measurements, skin temperature under the cuffs was monitored by insulated thermistors (Yellow Springs), and skin temperature of the toes adjacent to the hallux was measured by copper/constantan thermocouples. Brachial systolic pressure was measured by the auscultatory technique at the time of each measurement of the toe pressure.

Assessment of effect of local temperature. The technique previously used to study the effect on finger pressures was modified.18,19 Water at the desired temperature was perfused through a double-inlet, 3-cm-wide pneumatic cuff applied to the base of the hallux for 7 minutes before the pressure measurement. Water was circulated from a reservoir with a commercial sump pump. During the perfusion, blood flow to the digit was interrupted by a tourniquet applied to the base of the toe to allow toe temperature to equilibrate with that of the water. After 7 minutes, the cuff was pressurized, the tourniquet was released, and the measurements were carried out during deflation of the cuff at the rate of 2 mm Hg/sec. Toe pressure measurements were carried out at local temperatures from 10°C to 39°C as well as under routine conditions (the same cuff but no water perfusion) referred to as baseline measurements.

Three experimental protocols were used. 1) In 22 extremities, the effects of local cooling to 10°C and of local warming to 30°C were compared with baseline measurements with the subjects covered only by a light blanket and extremities exposed to room temperature. 2) In 28 extremities, the effects of local temperatures of 10°C and 30°C were compared with baseline measurements after the subjects and their limbs were covered for 20 minutes by a heating blanket, which is the routine condition under which toe systolic pressures have been measured in our laboratory.7 3) After routine baseline measurements were carried out as in protocol 2, pressure measurements in the toes were obtained at the local temperature of 27°C and at increments of 3°C up to 36°C in 27 extremities and up to 39°C in 19 of these limbs.

Statistical Analysis

Data are reported as mean±SEM. The results were examined by ANOVA.20 Differences in toe pressures at various temperatures were tested by one-way or two-way ANOVA, as appropriate. ANCOVA was used to examine a possible effect of varying brachial pressure at different temperatures. The potential effect of categorical variables (e.g., diabetes) on the effect of temperature on toe pressure was examined with split-unit ANOVA by testing for interaction between each categorical variable and temperature. The mean values shown in Table 1 are computed by least-squares regression. The probability values are given for two-tailed tests. A value of p<0.05 is considered significant.

Results

Effect of Local Cooling

The effects of local cooling did not differ significantly in the experiments using protocols 1 and 2; therefore, the data were pooled for the analysis. Figure 1 shows that the mean toe systolic pressure of 34±4 mm Hg at 10°C was significantly lower than the mean baseline value of 57±4 mm Hg and the value of 69±4 mm Hg at 30°C (p<0.001). Toe systolic pressure at 10°C was lower than during the baseline measurements in 42 of the 50 limbs and lower than at 30°C in 48 limbs.

Effect of Local Warming

In the studies using protocol 1, a tendency toward peripheral vasoconstriction was evidenced by the skin temperature of the adjacent toes of 24±1°C (range, 21–31°C) during baseline measurements. Local warming to 30°C resulted in an increase of toe systolic pressure from the baseline values in 21 of 22 limbs. The difference between the mean value of 61±6 mm Hg at baseline and 86±6 mm Hg at 30°C was highly significant (p<0.01). In the studies using protocol 2, there was no significant
difference between mean systolic pressure of 53±5 mm Hg during baseline measurements and 56±5 mm Hg after warming to 30°C. The average skin temperature of the toes in these experiments was 27±1°C (range, 21–34°C). Different results were obtained, however, when the effect of local warming to 30°C was compared separately in limbs with under-cuff skin temperatures below and above 30°C during baseline measurements. Figure 2 shows that pressures measured during warming to 30°C were significantly higher than during baseline measurements when local temperature at baseline was below 30°C but not when it was higher. The pressure during local warming increased in 11 of 13 limbs in which the temperature at baseline was below 30°C and in five of 15 limbs in which it was 30°C or higher.

Effect of Progressive Increase in Local Temperature

Figure 3 shows the results of baseline measurements and of toe systolic pressures measured at local temperatures from 27°C to 39°C. Mean pressures increased with digital temperature (p < 0.01). The pressures at 36°C and 39°C were significantly greater than at baseline but did not differ significantly from each other. They were higher than during baseline measurements in 16 of 27 limbs at 36°C and in 14 of 19 limbs at 39°C.

The mean brachial pressure during various experimental conditions varied by no more than 5 mm Hg, and the effect of local temperature on toe pressures could not be explained by differing brachial systolic pressures. Also, the same results were obtained when toe systolic pressure was expressed as a ratio of the toe-to-brachial systolic pressures. Table 1 shows a breakdown of the data by patient subgroups. The severity of arterial disease, the presence of diabetes, the smoking status, and therapy with vasodilators did not show any significant interactions with the effect of temperature on toe pressure (probability values, 0.96, 0.20, 0.57, and 0.51, respectively). In all subgroups, the largest increase in pressure occurred between 30°C and 33°C, and the changes in pressure over the range of temperature were similar in limbs with more and less severe arterial obstruction. The patients on vasodilators (six limbs) were all smokers, and two of these patients (four limbs) had diabetes. Because of the small numbers of limbs, especially in the subgroups of smokers and of those on vasodilators, the possibility of some differences in the effect of higher temperatures cannot be ruled out.

Discussion

To best apply the measurements of distal blood pressure to the assessment of arterial disease, it is helpful to consider two important factors that affect them: 1) the degree to which the blood pressure falls during flow through the vessels proximal to the site of the measurements and 2) the behavior of the walls of the arteries under the blood pressure cuffs during measurements. The degree to which the blood pressure falls during flow depends on the geometric dimensions of the vessels, including the severity of the obstruction by the pathological process, and, importantly, on the existing blood flow, which in turn depends on the peripheral resistance in the microcirculation. Peripheral vasodilation, induced, for example, by exercise or by body heating, increases blood flow and thereby results in greater frictional pressure energy losses during flow through a pathological resistance as well as through normal vessels, especially in the hands and feet, where the arteries are narrower. Greater energy losses result in a larger pressure drop across a pathological resistance and in lower digital pressures in normal subjects. Conversely, when vasoconstriction in the microcirculation results in lower blood flow, the pressure distal to an occlusive process and the digital pressures increase.

Blood pressure measurements using pneumatic cuffs can also be influenced by the behavior of the walls of the arteries under the cuffs, and this was shown to be affected by temperature. The studies of Lassen and coworkers demonstrated that local cooling markedly decreased measured values of the finger systolic pres-
TABLE 1. Effect of Temperature on Mean Toe Systolic Pressure (mm Hg) in Groups of Patients With Peripheral Arterial Disease

<table>
<thead>
<tr>
<th>Group</th>
<th>Limbs (n)</th>
<th>Baseline</th>
<th>27°C</th>
<th>30°C</th>
<th>33°C</th>
<th>36°C</th>
<th>39°C</th>
</tr>
</thead>
<tbody>
<tr>
<td>All limbs</td>
<td>27</td>
<td>33.1±1.7</td>
<td>32.3±1.9</td>
<td>33.1±1.7</td>
<td>38.3±1.8</td>
<td>39.6±1.7</td>
<td>41.0±2.2</td>
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<tr>
<td>Baseline toe systolic pressure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Above 30 mm Hg</td>
<td>13</td>
<td>47.6±2.6</td>
<td>45.5±2.6</td>
<td>47.0±2.6</td>
<td>53.2±2.6</td>
<td>54.9±2.6</td>
<td>56.0±2.8</td>
</tr>
<tr>
<td>Below 30 mm Hg</td>
<td>14</td>
<td>19.7±2.5</td>
<td>20.6±3.0</td>
<td>20.1±2.5</td>
<td>24.5±2.7</td>
<td>25.4±2.5</td>
<td>27.0±3.4</td>
</tr>
<tr>
<td>No diabetes</td>
<td>16</td>
<td>33.4±2.2</td>
<td>31.4±2.5</td>
<td>30.4±2.2</td>
<td>33.8±2.5</td>
<td>35.7±2.2</td>
<td>39.1±2.5</td>
</tr>
<tr>
<td>Diabetes</td>
<td>11</td>
<td>32.7±2.7</td>
<td>33.3±3.1</td>
<td>36.9±2.7</td>
<td>44.5±2.7</td>
<td>45.3±2.7</td>
<td>43.2±4.2</td>
</tr>
<tr>
<td>Nonsmokers</td>
<td>19</td>
<td>35.5±2.1</td>
<td>33.7±2.2</td>
<td>34.5±2.1</td>
<td>39.5±2.1</td>
<td>41.6±2.1</td>
<td>44.8±2.4</td>
</tr>
<tr>
<td>Smokers</td>
<td>8</td>
<td>27.6±3.2</td>
<td>29.4±3.9</td>
<td>29.6±3.2</td>
<td>36.1±3.9</td>
<td>34.8±3.2</td>
<td>29.5±4.8</td>
</tr>
<tr>
<td>No vasodilators*</td>
<td>19</td>
<td>35.5±2.1</td>
<td>34.0±2.5</td>
<td>34.3±2.2</td>
<td>39.4±2.3</td>
<td>42.2±2.2</td>
<td>45.7±2.7</td>
</tr>
<tr>
<td>Vasodilators</td>
<td>6</td>
<td>30.2±3.8</td>
<td>30.8±3.8</td>
<td>31.5±3.8</td>
<td>37.5±3.8</td>
<td>34.7±3.8</td>
<td>30.9±4.9</td>
</tr>
</tbody>
</table>

*One patient was taking a β-blocker and was excluded from the group.

Vasoconstriction is Raynaud's syndrome. The frequent loss of measureable pressure indicated the occurrence of critical closure in the main digital arteries. The effect of local cooling was exaggerated by sympathetic vasoconstriction induced by body cooling,23 and body cooling without local cooling often led to the loss of measureable pressure in patients with Raynaud's syndrome.18 High local temperature (30°C) protected fingers from vasospasm, whereas low pressure pressure related to proximal arterial obstruction predisposed to vasospasm.18 The effect of local cold on the apparent digital systolic pressure is probably related to a high tone or altered viscoelastic properties of the smooth muscle of the main digital arteries under the pressure-measuring cuff. These changes result in a delayed opening of the vessels during or after deflation of the cuffs.18 Therefore, local cold, whether produced by local cooling or by low blood flow related to intense sympathetic vasoconstriction induced by body cooling, may result in decreases of the measured digital pressure in some subjects because of the effect on the smooth muscle of the main digital arteries. Such decreases represent a measurement artifact.

Thus, changes in temperature may exert two opposing effects on the pressure measured in the digits. Vasoconstriction in the microcirculation in response to cold decreases flow and frictional pressure energy losses and thus increases digital blood pressure.15 Local cold, however, can result in delayed opening of the digital arteries after they are compressed by the pressure-measuring cuffs and thus in underestimation of the measured pressure. The net effect of temperature in an individual subject probably depends on the relative balance between these two opposing effects, including the actual level of the temperature and the sensitivity of the digital vessels to temperature. Marked sensitivity to low temperature was demonstrated previously in the fingers of patients with Raynaud's phenomenon and in some normal subjects.16-18 Our recent demonstration that local as well as body cooling resulted in a marked decrease or loss of measureable toe pressure in patients with Raynaud's syndrome in the toes and in a proportion of normal subjects19 led to the present study of patients with arterial occlusive disease.

The effect of temperature on toe pressure has not been studied extensively in patients with arterial disease. Direct heating of the legs and feet resulted in small decreases in toe pressures compared with direct cooling, which were not considered to be practically important.22 These changes were in the same direction as the results of body heating in normal subjects14,15 and were probably a result of vasodilation in the tissues of the legs and feet and greater pressure energy losses in flow proximal to the toes. Local heating of the feet to 30°C for 10 minutes, however, increased toe pressures in limbs of patients without diabetes mellitus and with different degrees of severity of the arterial obstruction.23 The measurements in that study were carried out at a warm room temperature of 26°C. Despite the high ambient temperature, however, the toes were cold, as indicated by the mean toe temperature of only about 22°C during the measurements before warming of the feet, which was 2°C lower than the toe temperature in our experiments using protocol 1. The low temperature likely led to apparently low initial pressures caused by a delayed opening of the digital arteries before warming of the feet. Warming then eliminated the effect of the initially low toe temperature and resulted in a significant increase in the measured pressure.

Our findings indicate that local temperature of the digits has a significant effect on the value of the toe systolic pressure measured in limbs with arterial disease. When the temperature is low during routine laboratory measurements, the resulting lower pressures may suggest a greater severity of arterial obstruction and worse prognosis than may actually be the case. Toe pressures are used increasingly as a valid index of the severity of arterial obstruction and of the presence of critical limb ischemia.7-13,24-27 They reflect the overall involvement of the arterial tree by the occlusive process down to the digits3,7,12 and correlate with the risk of amputation and with mortality.24-26 The value of the toe pressure is important in the assessment of the chances of healing of skin lesions and of distal amputations.6,12,13 The prognosis for healing is very good if the pressure is above 30 mm Hg but uncertain in the extremities with lower pressures. Our results suggest that maintaining digital temperature at 30°C would result in an increase in toe systolic pressure from below to above 30 mm Hg in 20-25% of cases (protocols 1 and 2) and that this proportion may be greater with warming to a higher temperature (protocol 3). Also, measurements at 10°C indicate that when the toes are very cold, a much larger incidence of falsely low apparent pressures, indicative of
more severe arterial obstruction and worse prognosis, is likely to occur.

The effects of temperature demonstrated in this study have important practical implications for routine measurements of toe pressures, which are used in making decisions concerning management of individual patients, and in the evaluation of the results of surgical interventions. In our routine testing, we cover the patients, including the limbs and the feet, by a heating blanket for at least 20 minutes before the measurements, and if the toes are still cold, the period of heating is prolonged. The results of this study indicate that such a protocol is necessary to obtain results that reflect as accurately as possible the severity of arterial disease in routine laboratory measurements. A study of toe pressures measured under controlled local temperatures in limbs with severe ischemia and skin lesions is needed to demonstrate whether or not such measurements will improve accuracy of predicting spontaneous healing of skin lesions.

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References

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