Which physiological mechanism is responsible for the increase in blood pressure during leg crossing?

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**Objective** To determine which physiological mechanism is responsible for the blood pressure increase during leg crossing at knee-level in the sitting position.

**Methods** Finger blood pressure was measured with the Finometer in 102 participants (47 men) before and during leg crossing: 24 treated hypertensive patients, 50 diabetic individuals (25 with and 25 without antihypertensive medication) and 28 healthy volunteers. Mean age, 53 ± 15 years (range 21–82 years). All participants crossed their legs at knee-level, with the upper part of the popliteal fossa on the suprapatellar bursa, in the sitting position. Differences in mean blood pressure, cardiac output, stroke volume, heart rate and total peripheral resistance were assessed with legs crossed versus legs uncrossed.

**Results** Mean blood pressure (+3.3 ± 5.5 mmHg; 95% confidence interval (CI) = 2.7–3.8), stroke volume (+7.6 ± 5.4 ml; 95% CI = 6.7–8.6) and cardiac output (+0.4 ± 0.9 l/min; 95% CI = 0.3–0.5) were significantly higher with legs crossed than in the uncrossed position, while the heart rate (−1.8 ± 3.9 beats/min; 95% CI = −2.2 to −1.4) was significantly lower. Total peripheral resistance did not differ significantly (−0.01 ± 0.16 AU; 95% CI = −0.03 to 0.00). The largest differences occurred in the hypertensive diabetic individuals, the smallest in the healthy volunteers. The changes were similar in men and women. There were no significant correlations in the total group between the differences of the hemodynamic variables and sex, age, body mass index or leg circumference.

**Conclusion** The study shows that higher blood pressure with legs in the crossed position is due to higher cardiac output and not to a higher total peripheral resistance. J Hypertens 26:433–437 © 2008 Wolters Kluwer Health | Lippincott Williams & Wilkins.

**Keywords:** blood pressure measurement, leg crossing, physiological mechanism, stroke volume

Abbreviations: BMI, body mass index; CO, cardiac output; HR, heart rate; MAP, Mean arterial pressure; SV, stroke volume; TPR, total peripheral resistance

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**Introduction**

Blood pressure (BP) measurement is one of the most widely used diagnostic tests in medicine.

Numerous factors can affect the outcome of a BP measurement. An erroneously measured high BP may lead to a false diagnosis of hypertension. This error can be caused by an arm position below heart level, noise, talking and movements during BP measurement and by smoking or caffeine use before BP measurement [1–3].

Another potential interfering factor resulting in a falsely high BP is leg crossing at knee level in the sitting position during BP measurement. Several, but not all, studies have shown that BP is higher when the legs are crossed at knee level in the sitting position during BP measurement [4–9]. Some studies in patients with vasovagal syncope or autonomic failure [10–12] demonstrated that the higher BP in these patients during leg crossing in a standing position with active muscle tensing is due to a higher cardiac output (CO). The aim of the present study was to determine which physiological mechanism is responsible for the higher BP during leg crossing without active muscle tensing in the sitting position in healthy volunteers and in hypertensive and diabetic patients.

**Materials and methods**

**Sample** We included 108 participants – 28 healthy volunteers, 28 treated hypertensive patients, 52 diabetic individuals (type I and type II, 26 with and 26 without antihypertensive medication) – who visited the outpatient clinic at the University Medical Centre St Radboud in Nijmegen during a 4-week period in May–June 2006.

Individuals were excluded if they were unable to cross their legs or had a history of peripheral vascular disease. Other exclusion criteria were pregnancy and arrhythmias. There were no restrictions with regard to medication.
Informed consent was obtained from all participants before the start of this study. The local ethics committee approved the protocol.

**Measurement of blood pressure**

The age, height, weight, body mass index, sex, circumference of the calf (10 cm below the patella), and circumference of the thigh (15 cm above the patella) were noted in all individuals.

BP measurements were carried out by one trained investigator in the outpatient clinic. Beat-to-beat systolic BP and diastolic BP were measured continuously and noninvasively by use of the Finometer Model 1.10 (TNO Biomedical Instrumentation, Amsterdam, The Netherlands). Finometer recordings accurately reflect BP changes [13–17]. The finger cuff was applied to the midphalanx of the left middle finger. To avoid hydrostatic level differences, the hand was continuously positioned at right atrial level in the midaxillary line.

Patients were seated in a comfortable chair with stable head, back and arm support. The individual was instructed to refrain from talking or movement during the procedure. The investigator left the room after the patient took the uncrossed leg position with both feet flat on the floor. After 7 min the investigator entered the room and the BP measurement by Finometer was started. After 3 min in the uncrossed position, the participants had to cross their legs at knee level for 4 min (the upper part of the popliteal fossa on the suprapatellar bursa). Subsequently there was another 4-min uncrossed period. After that, the participants had to cross their legs at knee level for 4 min again. Finally they took an uncrossed position for 4 min.

The sequence of leg crossing (right/left or left/right order) was randomized.

**Stroke volume and total peripheral resistance computation**

From the continuous BP measurement, the arterial pulse wave was analyzed by the pulse wave analysis method, which computes changes in left ventricular stroke volume (SV) from the pulsatile systolic area. We used the Modelflow program (described in [18]), which comes with the software package the manufacturer of the finometer provides with the instrument. Modelflow is a model-based algorithm that computes the aortic flow waveform from an arterial blood pressure pulsation by simulating a nonlinear, self-adaptive (three-element Windkessel) model of the aortic input impedance. Although Modelflow is less suitable to measure absolute values of SV, various studies have shown that Modelflow is a reliable method to assess changes in SV [19], which is the aim of our study. The CO was computed as the SV multiplied by the heart rate (HR). The total peripheral resistance (TPR) was calculated as the mean arterial pressure (MAP) divided by the CO and expressed in arbitrary units. The MAP was obtained as the integral of pressure over one beat divided by the corresponding interbeat interval.

**Analysis**

In the pilot phase of our study we observed that during the leg crossing itself and immediately thereafter the BP showed temporarily an extra increase for 1–2 min and then decreased to a lower steady state, but was still clearly above the BP before crossing. We therefore analyzed the last 2 min of each 4-min period of sitting with crossed or uncrossed legs. Changes for parameters are presented as the mean ± SD. Reproducibility was determined by the standard deviation of the difference between the first and the second period of crossing the legs.

For statistical analysis, we used SPSS version 14.0 for Windows (SPSS Inc., Chicago, Illinois, USA). The Kolmogorov–Smirnov test was used to determine the distribution of all hemodynamic variables.

The differences in hemodynamic variables between both positions were tested by Student’s t-test (paired). The correlations between the different variables were analyzed by Pearson’s linear correlation. To account for multiple testing, Bonferroni correction was used. The influence of age, gender, body mass index, leg circumference and the presence of hypertension or diabetes mellitus on the effects of the maneuvers on the hemodynamic variables was analyzed by univariate regression. Since diabetes mellitus and hypertension had a significant effect on the changes in hemodynamic parameters, we also investigated whether antihypertensive medication modified this response. To this end we introduced an interaction term of type of medication and the presence of diabetes mellitus and/or hypertension in the regression equation.

A P value less than 0.05 was considered significant (two-sided).

**Results**

Finally 102 participants were included in this study; six participants had to be excluded because of atrial flutter (n = 1) or an inability to cross legs for 2 × 4 min (n = 5). Patient characteristics are presented in Table 1. All hypertensive individuals used antihypertensive medication, with a mean of 2.9 antihypertensive drugs per patient in the hypertensive group and 2.4 antihypertensive drugs per patient in the hypertensive diabetic group.

All hemodynamic variables were normally distributed as demonstrated by the Kolmogorov–Smirnov test.

Leg crossing at knee level caused a significant rise in the MAP (+3.5 mmHg, +3.6%), systolic BP (+6.6 mmHg, +6.3%),
5.4%) and diastolic BP (+1.4 mmHg, +2.0%) in the total study group. This rise in BP was accompanied by a significant increase in SV and CO by, respectively, +7.4 ml (8.5%) and 0.4 l/min (6.7%). The HR decreased significantly (/C0 1.8 beats/min, /C0 2.4%). TPR did not change significantly (/C0 0.01 AU, /C0 1%) (Figs 1 and 2).

All subgroups showed the same trend in hemodynamic differences. Although the differences in MAP, SV, CO and HR were largest in the hypertensive diabetic individuals (+4.0 mmHg, +10.6 ml, +0.7 l/min and -1.0 beats/min, respectively) and lowest in the healthy volunteers (respectively +2.2 mmHg, +4.7 ml, +0.1 l/min and -2.9 beats/min), the differences in all subgroups were significant. All results are presented in Tables 2 and 3. Men and women did not differ with regard to the increase in BP or any other hemodynamic variables. There was no significant difference in the MAP either if the sequence was left/right or right/left. The difference in the MAP between the first and the second leg crossings was 0.6 ± 1.2 mmHg. The difference in CO between the first and the second leg crossings was 0.02 ± 0.6 l/min, indicating adequate reproducibility of the test. Both in the total study group and in the subgroups we observed a positive correlation between the difference in MAP and the difference in CO, varying from $r = 0.17$ ($P = 0.36$) in the normotensive individuals to $r = 0.67$ ($P < 0.05$) in the hypertensive diabetic participants. For the total study group we observed no correlation between the differences in CO and their baseline value, body mass index, age or leg circumference. By regression analysis with the difference in CO, the difference in HR or the difference in SV as dependent variables, we found a significant influence of the diagnosis hypertension (for changes in CO and HR), of diabetes mellitus (for changes in CO and SV) and of diabetes and/or hypertension (for changes in all three variables). The use of different antihypertensive drugs, however, studied as an interaction term with the presence of hypertension, diabetes mellitus or their combination, could not explain the hemodynamic changes in a model that included the presence of hypertension, diabetes mellitus or their combination (results not shown).

### Table 1 Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Total group</th>
<th>Healthy volunteers</th>
<th>Treated hypertensive patients</th>
<th>Normotensive diabetic individuals</th>
<th>Treated hypertensive diabetic individuals</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>102</td>
<td>28</td>
<td>24</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Men/women</td>
<td>46/56</td>
<td>12/16</td>
<td>11/13</td>
<td>12/13</td>
<td>11/14</td>
</tr>
<tr>
<td>Age (years)</td>
<td>53 ± 15 (21–82)</td>
<td>48 ± 18 (21–67)</td>
<td>60 ± 12 (35–82)</td>
<td>47 ± 15 (22–79)</td>
<td>58 ± 11 (41–81)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>80 ± 15</td>
<td>76 ± 10</td>
<td>81 ± 12</td>
<td>78 ± 9</td>
<td>87 ± 23</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174 ± 9</td>
<td>174 ± 8</td>
<td>173 ± 9</td>
<td>176 ± 10</td>
<td>173 ± 9</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>27 ± 5 (20–41)</td>
<td>25 ± 3 (20–32)</td>
<td>27 ± 4 (22–31)</td>
<td>25 ± 4 (20–31)</td>
<td>29 ± 6 (20–41)</td>
</tr>
<tr>
<td>Leg circumference, calf (cm)</td>
<td>37 ± 4</td>
<td>37 ± 2</td>
<td>36 ± 3</td>
<td>38 ± 2</td>
<td>37 ± 4</td>
</tr>
<tr>
<td>Leg circumference, thigh (cm)</td>
<td>47 ± 4</td>
<td>47 ± 4</td>
<td>47 ± 4</td>
<td>48 ± 3</td>
<td>48 ± 5</td>
</tr>
<tr>
<td>Baseline SBP/DBP (mmHg)*</td>
<td>122/69 ± 20/11</td>
<td>118/69 ± 17/10</td>
<td>130/72 ± 25/13</td>
<td>120/66 ± 17/7</td>
<td>121/66 ± 18/11</td>
</tr>
</tbody>
</table>

Data presented as the mean ± SD (range). * Mean of the systolic blood pressure (SBP) and the diastolic blood pressure (DBP) in the three uncrossed positions measured by Finometer.
Table 2 Differences in hemodynamic parameters between uncrossed and crossed leg positions in the total study group

<table>
<thead>
<tr>
<th>Hemodynamic parameter</th>
<th>Uncrossed leg position</th>
<th>Crossed leg position</th>
<th>Difference absolute (relative)</th>
<th>(95% confidence interval of absolute difference)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>123.1 ± 19.5</td>
<td>129.7 ± 19.2</td>
<td>+6.6 (+5.4%)</td>
<td>(5.8/7.5)</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>89.1 ± 10.6</td>
<td>70.3 ± 9.9</td>
<td>-16.8 (-20.0%)</td>
<td>(-21.2/-16.4)</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>90.1 ± 13.0</td>
<td>93.4 ± 12.4</td>
<td>+3.3 (+3.6%)</td>
<td>(2.7/3.8)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>72.1 ± 12.6</td>
<td>70.3 ± 12.1</td>
<td>-1.8 (-2.4%)</td>
<td>(-2.2/-1.4)</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>88.6 ± 24.3</td>
<td>96.4 ± 26.5</td>
<td>+7.8 (8.5%)</td>
<td>(6.7/8.6)</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>6.3 ± 1.7</td>
<td>6.7 ± 1.9</td>
<td>+0.4 (+6.3%)</td>
<td>(0.3/0.5)</td>
</tr>
<tr>
<td>Total peripheral resistance (AU)</td>
<td>0.94 ± 0.37</td>
<td>0.93 ± 0.36</td>
<td>-0.01 (-1%)</td>
<td>(-0.03/0.00)</td>
</tr>
</tbody>
</table>

Leg position data presented as the mean ± SD.

Discussion
The present study shows that, both in healthy individuals and in patients with hypertension and diabetes mellitus, the higher BP during leg crossing at knee level is due to a higher CO and not to a higher TPR. The rise in CO is due to an increase in the SV. An explanation of this increase in SV and reaching a steady state is the translocation of blood to the central thoracic compartment by continuous mechanical compression of venous capacitance vessels in the leg. The significant decrease in the HR is due to a baroreceptor reflex activation as a consequence of the increase in blood pressure. A smaller decrease in the HR is seen in the hypertensive and hypertensive-diabetic subgroups.

Our results are in agreement with the conclusions from Krediet et al. [10] and van Dijk et al. [11] in patients with vasovagal syncope and autonomic dysfunction in the standing position with active muscle tensing. In these studies the BP measurement was started directly after performing the maneuver and took 2 min. In a previous pilot study [9] we observed that BP stabilized 2 min after changing leg position. For this reason, and because Wieling et al. [20] showed a blood pressure response immediately after starting dynamic leg exercise in humans, we only analyzed the BP values of the last 2 min of each position.

Groothuis et al. [21] suggested that leg crossing increases TPR mechanically. Their study, however, reports leg crossing with muscle tensing in a standing position. In our study, participants had to cross their legs in a sitting position without muscle tensing. Comparing the results of these studies is therefore not possible because of the study design differences.

Continuous beat-to-beat BP measurement is a strong point of the present study, because changes in hemodynamic variables were calculated based on a great number of measurements. We examined a large number of participants with different cardiovascular risk factors. The reason why we examined four subgroups is inspired by the potential influence of both underlying disease and the medication on the changes in hemodynamic variables. The randomization of leg order is important, because when there is no randomization participants take their ‘preferential position’.

We did not consider or correct for the influence of meals, coffee, smoking and the time of medication intake. Possibly there is a relation between the time of meals, coffee, smoking or medication intake and the time of BP measurement.

Changes in CO were related to the presence of hypertension, diabetes mellitus and their combination, but were not explained by the use or the type of the antihypertensive drug. Further analysis was limited by the fact that the patients were on many different combinations of antihypertensive drugs we could not adequately test for interactions of drugs.

Hypertensive and/or diabetic individuals show the most pronounced hemodynamic response to changing leg position, whereas healthy volunteers show the smallest response. This difference cannot be attributed to different

Table 3 Absolute differences in hemodynamic variables (95% confidence interval) due to crossing the legs at knee level in the different subgroups

<table>
<thead>
<tr>
<th>Hemodynamic variable</th>
<th>Healthy volunteers</th>
<th>Treated hypertensive patients</th>
<th>Normotensive diabetic individuals</th>
<th>Treated hypertensive diabetic individuals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difference in systolic blood pressure (mmHg)</td>
<td>+4.1 ± 6.8 (2.8–5.4)</td>
<td>+7.0 ± 7.4 (5.5–8.5)</td>
<td>-7.8 ± 7.2 (6.4–9.3)</td>
<td>+8.2 ± 10.7 (6.1–10.4)</td>
</tr>
<tr>
<td>Difference in diastolic blood pressure (mmHg)</td>
<td>+1.2 ± 3.2 (0.6–1.8)</td>
<td>+1.4 ± 3.3 (0.8–2.1)</td>
<td>+2.1 ± 3.4 (1.4–2.8)</td>
<td>+2.2 ± 4.3 (1.4–3.1)</td>
</tr>
<tr>
<td>Difference in mean arterial pressure (mmHg)</td>
<td>+2.2 ± 4.1 (1.4–2.9)</td>
<td>+3.6 ± 4.6 (2.6–4.5)</td>
<td>+3.9 ± 4.7 (2.9–4.8)</td>
<td>+4.0 ± 6.4 (2.7–5.2)</td>
</tr>
<tr>
<td>Difference in heart rate (beats/min)</td>
<td>-2.9 ± 5.5 (-3.6 to -2.3)</td>
<td>-1.1 ± 4.2 (-1.9 to -0.2)</td>
<td>-2.9 ± 3.7 (-2.7 to -1.2)</td>
<td>-1.0 ± 3.7 (-1.8 to 0.3)</td>
</tr>
<tr>
<td>Difference in stroke volume (ml)</td>
<td>+4.7 ± 6.8 (3.4–5.9)</td>
<td>+6.8 ± 8.4 (5.1–8.5)</td>
<td>+8.5 ± 8.1 (6.1–10.1)</td>
<td>+10.8 ± 10.3 (8.5–12.6)</td>
</tr>
<tr>
<td>Difference in cardiac output (l/min)</td>
<td>+0.1 ± 0.4 (0.0–0.2)</td>
<td>+0.4 ± 0.5 (0.3–0.5)</td>
<td>+0.4 ± 0.4 (0.3–0.5)</td>
<td>+0.7 ± 0.7 (0.5–0.8)</td>
</tr>
<tr>
<td>Difference in total peripheral resistance (AU)</td>
<td>+0.01 ± 0.07 (0.00–0.02)</td>
<td>0.03 ± 0.15 (0.00–0.06)</td>
<td>-0.01 ± 0.07 (-0.02 to 0.01)</td>
<td>-0.02 ± 0.35 (-0.09 to 0.05)</td>
</tr>
</tbody>
</table>

Data presented as the mean ± SD.
Blood pressure increase and leg crossing van Groningen et al. 437

subgroup characteristics or to the use of antihypertensive treatments. The subgroups may have been too small, however, to rule out an effect of antihypertensive drugs. A physiological explanation for the pronounced response in hypertensive and/or diabetic individuals could be the decreased venous distensibility that is already present in borderline hypertensive patients [22]. If crossing the legs causes compression of the capacitance vessels, the ensuing mobilization of blood from the legs can be contained to a lesser extent in the relatively stiffer venous vessels, leading to increased venous return and, hence, to increased CO.

In conclusion, the present study shows that a higher BP during leg crossing at knee level is due to the higher CO and not to a higher TPR. The results of this study suggest the importance of standardizing the leg position during BP measurements. The position of the legs should therefore be explicitly reported in the guidelines about hypertension and in all publications about BP measurement.

Acknowledgements
There are no conflicts of interest.

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