Leg crossing with muscle tensing, a physical counter-manoeuvre to prevent syncope, enhances leg blood flow

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ABSTRACT

In patients with orthostatic intolerance, the mechanisms to maintain BP (blood pressure) fail. A physical counter-manoeuvre to postpone or even prevent orthostatic intolerance in these patients is leg crossing combined with muscle tensing. Although the central haemodynamic effects of physical counter-manoeuvres are well documented, not much is known about the peripheral haemodynamic events. Therefore the purpose of the present study was to examine the peripheral haemodynamic effects of leg crossing combined with muscle tensing during 70° head-up tilt. Healthy subjects (n = 13) were monitored for 10 min in the supine position followed by 10 min in 70° head-up tilt and, finally, for 2 min of leg crossing with muscle tensing in 70° head-up tilt. MAP (mean arterial BP), heart rate, stroke volume, cardiac output and total peripheral resistance were measured continuously by Portapres. Leg blood flow was measured using Doppler ultrasound. Leg vascular conductance was calculated as leg blood flow/MAP. A significant increase in MAP (13 mmHg), stroke volume (27%) and cardiac output (18%), a significant decrease in heart rate (-5 beats/min) and no change in total peripheral resistance during the physical counter-manoeuvre were observed when compared with baseline 70° head-up tilt. A significant increase in leg blood flow (325 ml/min) and leg vascular conductance (2.9 arbitrary units) were seen during the physical counter-manoeuvre when compared with baseline 70° head-up tilt. In conclusion, the present study indicates that the physical counter-manoeuvre of leg crossing combined with muscle tensing clearly enhances leg blood flow and, at the same time, elevates MAP.

INTRODUCTION

Orthostatic stresses are common daily events in humans. In the upright position, a gravitational displacement of blood from the thorax to the venous vascular beds of the legs, buttock and abdomen occurs. During orthostasis, approx. 600–700 ml of blood is transferred to the regions below the diaphragm [1,2], which is known as ‘venous pooling’. This results in a reduced venous return to the heart and a fall in central venous pressure with a consequent decrease in cardiac filling, SV (stroke volume) and CO (cardiac output) [3]. The cardiovascular system in healthy humans is exquisitely adapted to maintain sufficient CO in the upright posture. Neural pathways of the autonomic nervous system mediate rapid short-term adjustments to orthostatic stress by increasing TPR (total peripheral resistance) and HR (heart rate). Concurrently, an increase in muscle tone in the legs prevents excessive...
pooling and promotes the venous return when upright [3]. In patients with orthostatic intolerance, these regulatory mechanisms fail to maintain BP (blood pressure) in the upright posture [1,4–6]. Patients with autonomic failure, who suffer from severe orthostatic hypotension due to failure of sympathetic neural vasomotor control [3], can promote venous return and raise CO by applying physical counter-manoeuvres that increase muscle skeletal tone, e.g. leg crossing and muscle tensing [3,7–10]. Also, patients with vasovagal syncope have successfully used these physical counter-manoeuvres to counteract their orthostatic intolerance [5,6,11]. Leg crossing and muscle tensing lead to an increase in MAP (mean arterial BP) and postpone or even prevent syncope [5,8,11–13].

It has been shown recently [12,13] that the BP-raising effect of physical counter-manoeuvres in patients with orthostatic intolerance is solely due to an increase in CO. However, leg vascular resistance itself could be affected in several ways during performance of the physical counter-manoeuvre. On the one hand, it has been suggested that leg vascular resistance would increase as a consequence of compression of the large arteries in the legs by the squeezing manoeuvre [6,11,14]. Some questions may be raised by this latter suggestion, since a very high pressure is needed to compress the large arteries and compression will only occur at the site of squeezing, i.e. a small part of the thigh and not the entire leg. On the other hand, vasodilatation is known to occur during muscle tensing [15–17] and could occur during performance of the physical counter-manoeuvre.

The purpose of the present study, therefore, was to examine the peripheral haemodynamic effects of leg crossing combined with muscle tensing during 70° head-up tilt. We tested the hypothesis that leg blood flow would increase during the physical counter-manoeuvre in the head-up tilt position.

**MATERIALS AND METHODS**

**Subjects**
Healthy normotensive subjects \(n = 17\), nine males and eight females, volunteered to participate in this study. The subjects’ medical history did not reveal any episodes of vasovagal syncope, cardiovascular diseases or any other morbidity. None of the subjects used any medication, and all subjects were non-smokers. All subjects gave written informed consent. The study has been carried out in accordance with the Declaration of Helsinki (2000) and was approved by the Medical Ethical Committee of our institution.

**Experimental procedures and protocol**
Subjects refrained from caffeine, alcohol and nicotine for at least 12 h and did not eat for 2 h prior to testing. The experiment took place in the morning in a quiet temperature-controlled room (between 22 and 24 °C). Before the experiment, the subjects received instructions on how to perform the physical counter-manoeuvre. They practised leg crossing combined with muscle tensing standing on the tilt table. This physical counter-manoeuvre consisted of crossing the right leg in front of the left leg at thigh level and pressing the legs firmly against each other. Simultaneously, the subjects were asked to tense the muscles of the legs and buttocks to their best ability and to maintain this tension during the measurement. The physical counter-manoeuvre was performed while standing on both legs, distributing the body weight equally on both legs. Subjects were instructed to avoid straining during the physical counter-manoeuvre [5,13].

Subjects were then placed in the supine position on a manually controlled tilt table with footboard in such a manner that they could stand on the footboard in the head-up tilt position. The subjects were supported with a chest belt to prevent them from falling down during the experiment in case of vasovagal syncope. When presyncopal symptoms occurred, the experiment was aborted and the subject was immediately tilted back to the supine position.

During an acclimatization period of a minimum of 20 min in the supine position, the measurement apparatus was connected, after which the experimental protocol was started with 10 min in the supine position in order to establish steady-state values. Subjects were subsequently tilted manually within 5 s to 70° head-up tilt. In this position, subjects were instructed to relax, standing with the weight of the body equally distributed on both legs. After 10 min in the head-up tilt position, the subjects were asked to perform the physical counter-manoeuvre. The physical counter-manoeuvre was executed within 2 s. The physical counter-manoeuvre was continued for 2 min. Then, the subjects were tilted back to the supine position and the experiment was ended.

**Measurements**
Arterial BP and HR were measured continuously and non-invasively using Portapres (TNO), a portable BP device. A finger cuff was attached to the middle phalanx of the third finger of the right hand in order to measure finger arterial BP and HR. Finger arterial BP measurements accurately reflect intra-arterial BP changes during orthostatic stress [18]. Data were collected during the experiment at a rate of 200 Hz. A built-in expert system, physiocal, was in operation during the entire experiment to establish and adjust the proper volume clamp set point. MAP values were derived beat-to-beat. HR was the inverse of the interbeat interval. SV was determined by a three-element model of arterial input impedance, using the Modelflow program, that provides accurate estimates of changes in SV during orthostatic stress [19]. CO was calculated as SV × HR, and TPR was calculated as MAP/CO. Monitoring of CO requires calibration.
against a gold standard if accurate absolute values are required [6,12]. Because the Modelflow measurement was not calibrated in the present study, SV, CO and TPR were expressed as relative changes from steady-state values in the head-up tilt position [12,20]. Changes in SV, CO and TPR were calculated only in the head-up tilt position, because there is some debate on using Modelflow with change of posture [21]. Changes in Modelflow CO during leg crossing with muscle tensing are in excellent agreement with CO changes measured by inert gas breathing [12].

\( V_{\text{mean}} \) (mean red blood cell velocity) and systolic and diastolic vessel diameter of the right common femoral artery were measured with a Doppler ultrasound device (Megas; ESAOTE) with a 5–7.5 MHz broadband linear array transducer. Doppler ultrasound measurements were performed on the right leg, which is the actively crossed leg. The sample volume was placed in the middle of the vessel, approx. 2 cm proximal of the bifurcation of the common femoral artery into the deep and superficial femoral artery. The angle of inclination for the red blood cell velocity measurements was consistently below 60°, which is visible online on the screen, and the vessel area was adjusted parallel to the transducer. A single analyst manually traced the Doppler waveforms thereafter. \( V_{\text{mean}} \) was calculated as the average of ten Doppler waveforms every 15 s in the supine and head-up tilt positions. At the start of the physical counter-maneuver (Time = 0 in Figures 2 and 3), \( V_{\text{mean}} \) was calculated every 5 s for a 20-s period and thereafter every 2 s. For diameter measurements at 5 min in the supine and head-up tilt positions, two consecutive longitudinal vessel images were analysed at the peak-systolic and end-diastolic phase. The diameter of the common femoral artery was calculated as 1/3 · systolic diameter + 2/3 · diastolic diameter [22]. Leg blood flow was calculated with the following formula: 

\[
(\pi \cdot r^2 \cdot V_{\text{mean}}) \cdot 60 \text{ (mmHg s)}
\]

Leg blood flow was calculated in the supine position using the supine diameter and in the upright position using the upright diameter. For the calculations, the assumption that the femoral artery diameter did not change is crucial. This assumption was confirmed by a pilot study performed in our laboratory in five healthy young men (22–28 years). Right common femoral artery diameter was 9.6 ± 0.6 cm in the supine position, 9.8 ± 0.8 cm in the head-up tilt position and 10.1 ± 0.7 cm after the physical counter-manoeuvre (non-significant differences within the error margin of the ultrasound device (coefficient of variation = 1.5% [22]).

Leg vascular conductance was calculated as leg blood flow/MAP. We choose to use leg vascular conductance as it is the most appropriate parameter to quantify the functional importance of local responses in the regulation of BP when large changes in blood flow are present [23]. For these calculations, we assumed that central venous pressure was near zero in the supine position and that hydrostatic pressure made an identical contribution to leg venous pressure as to leg arterial pressure.

### Data analysis

Steady-state values of MAP, HR, SV, CO, TPR, \( V_{\text{mean}} \), leg blood flow and leg vascular conductance were averaged over the intervals 7–9.5 min in supine and head-up tilt positions and 1.5–2 min in the head-up tilt position combined with the physical counter-manoeuvre. SV, CO and TPR in the head-up tilt position combined with the physical counter-manoeuvre (interval, 1.5–2 min) were also expressed as percentage changes from steady-state values in the head-up tilt position.

### Statistical analyses

Results are means ± S.E.M. or means [95% CI (confidence interval)]. Differences in steady-state values of MAP, HR, \( V_{\text{mean}} \), leg blood flow and leg vascular conductance between the supine and head-up tilt positions and differences in MAP, HR, SV, CO, TPR, \( V_{\text{mean}} \), leg blood flow and leg vascular conductance between the head-up tilt position and head-up tilt position combined with the physical counter-manoeuvre were tested with a two-tailed paired Student’s \( t \) test. Differences in diameter of the common femoral artery between the supine and head-up tilt positions were tested with a two-tailed paired Student’s \( t \) test. Gender differences in leg blood flow and leg vascular conductance between the head-up tilt position and head-up tilt position combined with the physical counter-manoeuvre were tested with a two-tailed unpaired Student’s \( t \) test. A two-sided probability value of < 0.05 was considered to be statistically significant.

### RESULTS

Four subjects, one male and three females, did not complete the protocol, due to presyncopal symptoms, and had to be returned to the supine position before the physical counter-manoeuvre was performed. This left 13 subjects for analysis, eight males and five females. General characteristics of the 13 subjects are shown in Table 1.

### Table 1 General characteristics of the subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All subjects</th>
<th>Male subjects</th>
<th>Female subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>( n )</td>
<td>13</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Age (years)</td>
<td>23.6 ± 1.0</td>
<td>22.4 ± 0.7</td>
<td>25.2 ± 2.3</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>184.3 ± 3.1</td>
<td>191.5 ± 2.6</td>
<td>173.0 ± 2.0*</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>75.4 ± 3.3</td>
<td>82.3 ± 3.0</td>
<td>64.4 ± 3.5*</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>22.0 ± 0.5</td>
<td>22.3 ± 0.4</td>
<td>21.5 ± 1.2</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>70.9 ± 1.3</td>
<td>71.0 ± 1.7</td>
<td>70.8 ± 2.1</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>120.1 ± 2.0</td>
<td>120.3 ± 3.0</td>
<td>120.0 ± 3.0</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>87.3 ± 1.0</td>
<td>87.4 ± 1.8</td>
<td>87.2 ± 1.2</td>
</tr>
</tbody>
</table>

*Significantly different from male subjects.
Steady-state values of central and peripheral haemodynamic parameters during supine, head-up tilt and head-up tilt with physical counter-manoeuvre

Values are means ± S.E.M., n = 13. SV, CO and TPR are absolute values.

*Significantly different from supine; †significantly different from head-up tilt.

<table>
<thead>
<tr>
<th>Haemodynamic parameter</th>
<th>Supine</th>
<th>Head-up tilt</th>
<th>Leg crossing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Central parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>90.4 ± 3.2</td>
<td>89.3 ± 2.5</td>
<td>102.5 ± 3.4†</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>67.3 ± 2.3</td>
<td>91.8 ± 3.2†</td>
<td>86.4 ± 3.3†</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>92.4 ± 6.6</td>
<td>65.3 ± 5.1</td>
<td>82.6 ± 5.6</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>6.12 ± 0.47</td>
<td>5.93 ± 0.52</td>
<td>7.02 ± 0.50</td>
</tr>
<tr>
<td>TPR (AU)</td>
<td>0.960 ± 0.089</td>
<td>0.991 ± 0.089</td>
<td>0.941 ± 0.081</td>
</tr>
<tr>
<td><strong>Peripheral parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Femoral artery Vmean (cm/s)</td>
<td>7.9 ± 0.8</td>
<td>3.5 ± 0.3†</td>
<td>10.8 ± 1.2†</td>
</tr>
<tr>
<td>Leg blood flow (ml/min)</td>
<td>318.5 ± 25.2</td>
<td>153.7 ± 12.8†</td>
<td>478.7 ± 57.4†</td>
</tr>
<tr>
<td>Leg vascular conductance (AU)</td>
<td>3.58 ± 0.32</td>
<td>1.75 ± 0.16†</td>
<td>4.40 ± 0.48†</td>
</tr>
</tbody>
</table>

**Central parameters**

A significant increase in HR [24 (20–29) beats/min; value is mean (95% CI)] was observed between the supine and head-up tilt positions with no change in MAP [1 (−2 to 4) mmHg] (Table 2 and Figure 1). An immediate rise in MAP, SV and CO, and a decline in TPR, were observed at the start of the physical counter-manoeuvre (Figure 1). After the rapid initial changes, significant increases in steady-state values were observed in MAP [13 (8–18) mmHg], SV [28 (17–39)%] and CO [21 (10–31)%], a significant decrease in HR [−5 (−10 to −1) beats/min], but no change in TPR [−3 (−12 to 6)%] (Table 2 and Figure 1).

**Peripheral parameters**

A significant increase in diameter of the right common femoral artery [0.03 (0.00–0.07) cm] was observed between the supine and head-up tilt positions, whereas significant decreases were seen in $V_{\text{mean}}$ [−4.4 (−9.3 to −1.6) cm/s], leg blood flow [−165 (−291 to −30) ml/min] and leg vascular conductance [−1.8 (−3.7 to −0.2) AU (arbitrary units)] (Table 2 and Figure 2). An immediate rise in leg blood flow and leg vascular conductance in the right, actively crossed, leg was observed at the start of the physical counter-manoeuvre (Figure 2). The physical counter-manoeuvre in the head-up tilt position resulted in significant increases in steady-state values of $V_{\text{mean}}$ [7.4 (1.8–15.5) cm/s], leg blood flow [325 (82–691) ml/min] and leg vascular conductance [2.9 (0.8–6.1) AU] in the right common femoral artery (Table 2 and Figure 2).

No gender differences were detected in leg blood flow and leg vascular conductance steady-state values or in changes in leg blood flow and leg vascular conductance between the head-up tilt position and head-up tilt position combined with the physical counter-manoeuvre (Figure 3).

**DISCUSSION**

The major findings of the present study are that the physical counter-manoeuvre, leg crossing combined with muscle tensing, enhances leg blood flow and leg vascular conductance. Concurrently, the physical counter-manoeuvre increases MAP and CO with an unchanged TPR. Thus the hypothesis that leg blood flow would increase during the physical counter-manoeuvre was confirmed; however, the increase in leg vascular conductance was not hypothesized.

**Haemodynamic effects**

The head-up tilt position causes a gravitational displacement of blood from the thorax to the venous vascular beds of the legs, buttock and abdomen, known as ‘venous pooling’. SV decreases, resulting in a decrease in CO and MAP [1,6,8,24,25]. In order to maintain sufficient CO, sympathetic activation results in an increase in peripheral vasoconstriction and HR [1,6,8,24–27]. In the present study, no change in MAP with an increase in HR was seen between supine and the head-up tilt positions (Table 2 and Figure 1), and leg blood flow and leg vascular conductance decreased (Table 2 and Figure 2). Supine and head-up tilt leg blood flow values found in the present study are in agreement with previous studies [1,24,26–28].

In parallel with previous studies [6,8], we have shown that leg crossing with muscle tensing during head-up tilting enhanced SV as well as CO and MAP, whereas HR decreased (Table 2 and Figure 1). We attribute the increase in CO to the fact that leg crossing with muscle tensing raises intramuscular pressure of the skeletal muscles in the legs, which causes mechanical compression of the venous vascular beds. This will counteract the ‘venous pooling’ and translocate venous blood back to the thorax, resulting in an increase in cardiac filling, SV and CO [5–9,12,13]. This translocation of venous blood occurs in the first 30 s after the onset of the physical counter-manoeuvre [20].

The present study is the first to report that leg crossing with muscle tensing during head-up tilting increases leg blood flow (Table 2 and Figure 2). Several factors could be involved. First, blood flow increases instantly at the onset of dynamic exercise [29] and peaks within 30 s (Figure 2) [20,30–32]. However, in the present study, subjects did not perform sustained dynamic exercise but maintained isometric muscle tensing after the dynamic initial bout. Isometric exercise also increases blood flow by dilatation of the vasculature, but the dilatation is less pronounced compared with dynamic exercise. Femoral artery blood flow increases slightly during isometric
contractions of the quadriceps muscle at 5–50% of MVC (maximal voluntary contraction) [15–17]. Sjøgaard et al. [16] reported that the increase in blood flow fell with increasing MVC, due to the increase in intramuscular pressure. MVC was not measured in the present study, but young healthy individuals can maintain 25–50%
of MVC during a 2-min period before fatigue occurs. Femoral artery blood flow increases during this period by approx. 40% [15–17]. Hence only a minor part of the increase in leg blood flow in the present study (> 200% increase) could be explained by maintained muscle tensing.

A second explanation may be that leg blood flow increases as a result of an increase in the arterio–venous pressure gradient (\(P_{\text{arterial}} - P_{\text{venous}}\)), due to venous emptying and increase in MAP. Hydrostatic pressure, caused by the upright position, can be assumed to be equal on the venous and arterial side of the leg circulation. Therefore leg blood flow (pressure difference/resistance) will increase due to an increase in MAP and a decrease in venous pressure [8,29,31].

Thirdly, as a result of the instant venous emptying induced by leg crossing with muscle tensing, withdrawal of the veno–arteriolar axon ‘reflex’ could occur. If so, the consequent increase in leg vascular conductance [28,33,34] will contribute to the increase in leg blood flow.

A fourth possible explanation is related to baroreflex activity. In response to a rise in MAP upon leg crossing with muscle tensing, arterial baroreceptor activity increases, resulting in a decrease in HR, CO and TPR [35]. In the present study, the physical counter-manoeuvre caused an increase in MAP with a slight decrease in HR. TPR, however, did not change (Table 2 and Figure 1). If baroreceptor activity explains the increase in leg blood flow during leg crossing with muscle tensing, by causing a reflex increase in leg vascular conductance, then this should have been reflected in a decrease in TPR.

Leg vascular conductance increases approx. 2.5-fold when the steady-state values of head-up tilting without and with the physical counter-manoeuvre were compared; however, the decrease in peripheral resistance is not reflected in the TPR change between head-up tilting and head-up tilting with the physical counter-manoeuvre.
Leg crossing with muscle tensing enhances blood flow

(Table 2). TPR is a computed central cardiovascular parameter (MAP divided by CO). It expresses total body peripheral resistance, which is the sum of vasodilatation in the active muscles of the legs and, apparently, vasoconstriction in other areas of the body, such as the non-working muscles, kidneys and splanchnic vascular bed.

Gender differences

In accordance with the study of van Dijk et al. [12], who reported no gender differences in central haemodynamic effects, except for a larger increase in systolic BP and pulse pressure in male individuals, when a physical counter-manoeuvre was applied in the head-up tilt position in patients with vasovagal syncope, we found no gender differences in peripheral haemodynamic effects, such as leg blood flow and leg vascular conductance changes induced by the physical counter-manoeuvre (Figure 3). However, our small sample size (eight males and five females) must be taken into account.

Limitations

Doppler ultrasound measurements were performed on the right common femoral artery, the leg that was actively crossed-over the left leg. One may argue that the results obtained in the present study do not represent left leg peripheral haemodynamics directly. The physical counter-manoeuvre involved crossing of the right leg in front of the left leg at thigh level and pressing them firmly against each other. Simultaneously, the muscles of both legs and buttocks were tensed. We assume therefore that the pressure applied was similar in both legs, resulting in partial compression of the thigh vascular bed. Thus, although the anatomical site of pressure was different in the two legs, i.e. medial-dorsal in the right leg and medial-ventral in the left leg, it is likely that the results obtained for the right common femoral artery apply to the left common femoral artery as well.

For calculation of the leg blood flow during the physical counter-manoeuvre, we used the diameter of the common femoral artery in the head-up tilt position. We assumed that the diameter would not change during the entire experiment [24]. Although we found a significant difference in diameter of the common femoral artery between supine and head-up tilt positions, this difference is within the error margin of the ultrasound method used (coefficient of variation = 1.5% [22]). It is not likely that the physical counter-manoeuvre affected the diameter of the common femoral artery because (i) crossing of the legs occurred at thigh level and the ultrasound measurements were performed well above this level in the inguinal area, and (ii) to affect the diameter of a large conduit artery a very high pressure would be needed and would probably induce more deformation rather than an actual change in diameter.

We studied healthy normotensive subjects, whereas physical counter-manoeuvres are usually recommended to patients with orthostatic intolerance [9–13]. Nevertheless, patients with orthostatic intolerance have similar initial central and peripheral haemodynamic effects of moving from supine to the upright posture as in healthy subjects [5,8,12,13,25,36–38], but these patients have a fall in CO before the onset of syncope, starting at the onset of the prodromal symptoms. If a physical counter-manoeuvre is applied at the moment that prodromal symptoms occur, as is recommended, the central haemodynamic effects are similar to healthy subjects [8,13]. We therefore suggest that the changes recorded in the present study are also likely to apply to patients with orthostatic intolerance.

(362x740)
Conclusions

The present study demonstrates that the physical counter-manoeuvre of leg crossing combined with muscle tensing clearly enhances leg blood flow and, at the same time, elevates MAP. The largest and immediate effect appears to be achieved by the compression of the venous vascular bed at the onset of the physical counter-manoeuvre, translocating blood back to the thorax and, simultaneously with this effect, an enhanced arterio-venous pressure gradient and possibly withdrawal of the veno-arterial reflex may occur, explaining the observed increase in leg blood flow.

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