Leg crossing, muscle tensing, squatting, and the crash position are effective against vasovagal reactions solely through increases in cardiac output

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Krediet, C. T. Paul, Ivar G. J. M. de Bruin, Karin S. Ganzeboom, Mark Linzer, Johannes J. van Lieshout, and Wouter Wieling. Leg crossing, muscle tensing, squatting, and the crash position are effective against vasovagal reactions solely through increases in cardiac output. J Appl Physiol 99: 1697–1703, 2005; doi:10.1152/japplphysiol.01250.2004.—Tensing of lower body muscles without or with leg crossing (LBMT, LCMT), whole body tensing (WBT), squatting, and sitting with the head bent between the knees (“crash position,” HBK) are believed to abort vasovagal reactions. The underlying mechanisms are unknown. To study these interventions in patients with a clinical history of vasovagal syncope and a vasovagal reaction during routine tilt table testing, we measured blood pressure (BP) continuously with Finapres and derived heart rate, stroke volume, cardiac output (CO), and total peripheral resistance using Modelflow. In series A (n = 12) we compared LBMT to LCMT. In series B (n = 9), WBT was compared with LCMT. In series C (n = 14) and D (n = 9), we tested squatting and HBK. All maneuvers caused an increase in BP, varying from a systolic rise from 77 ± 8 to 104 ± 18 mmHg (P < 0.05) in series A during LBMT to a rise from 70 ± 10 to 123 ± 9 mmHg (P < 0.05) in series B during LCMT. In each maneuver, the BP increase started within 3–5 s from start of the maneuver. In all maneuvers, there was an increase in CO varying from 54 ± 12% of baseline to 94 ± 21% in WBT to a rise from 65 ± 17% to 110 ± 22% in LCMT in series A. No maneuver caused significant change in total peripheral resistance. We conclude that the mechanism underlying the effects of these maneuvers is exclusively an increase in CO.

Modelflow; muscle pump; stroke volume

Recurrent vasovagal syncope is a common medical problem, significantly affecting the quality of life (10). Therapeutic options are limited (2), but recently several physical countermeasures have been introduced that are effective in countering vasovagal faints. Leg crossing with tensing of leg, abdominal, and buttocx muscles was proven to be an effective maneuver to stabilize blood pressure (BP) during an impending vasovagal faint (9). Another study documented that isometric arm exercise was an effective countermeasure (3). Leg and buttock muscle tensing alone was documented in an elderly patient to prevent posture-induced syncope (15). Another case report showed such beneficial effects of isometric leg extensions in a young subject (18). Earlier, whole body tensing proved effective in controlling impending vasovagal syncope related to blood phobia (12). In patients with autonomic failure, squatting counteracts hypotension presumably through its action on preload (22). Traditionally sitting with the head lowered between the knees (“crash position”) is a maneuver against impending faints (25).

The presumed mechanism underlying the beneficial effect of physical countermaneuvers on systemic BP is that skeletal muscle tensing of the lower body reinfuses pooled venous blood back to the chest, thereby increasing cardiac filling pressure, stroke volume (SV), and cardiac output (CO) (22). This mechanism has, however, never been documented during vasovagal reactions. In addition, the possibility that physical countermaneuvers increase peripheral vascular resistance should also be considered (3). Skeletal muscle contractions are accompanied by an increase in central command, which is known to increase the sympathetic outflow (14, 17). Finally, isometric contractions up from 10% of maximal power can compress arteries in skeletal muscle (16, 19), and such mechanical increases in total peripheral resistance (TPR) have also been suggested to play a role in the efficacy of physical countermaneuvers (20, 28).

Hypotheses. The aims of this study were threefold: 1) to investigate the functional mechanisms underlying the effectiveness of the above mentioned physical countermaneuvers (Table 1), 2) to search for clinically relevant differences in BP changes between lower body muscle tensing with (LCMT, Table 1) and without leg crossing (LBMT), respectively, and whole body tensing (WBT), and 3) to document the efficacy of squatting and that of sitting with the head bent between the knees (HBK) as applied in vasovagal reactions.

We hypothesized that the external pressure to the lower extremities in leg crossing with lower body muscle tensing would cause an additional increase in venous return and thereby in more pronounced effects on CO. Thus we expected that leg crossing with lower body muscle tensing would be more effective in raising BP than lower body muscle tensing alone. Secondly, we hypothesized that, if central command elicits a reflex increase in peripheral resistance, more extensive skeletal muscle contractions would be accompanied by a more pronounced effect (17). Consequently, we expected that the increase in TPR would be higher in total body tensing compared with lower body muscle tensing. The third part of the study had a descriptive purpose, aiming to document the effects of squatting and sitting down with the head lowered between the knees on all above mentioned parameters during vasovagal syncope.
METHODS

At the Academic Medical Center Syncope Unit, patients with vasovagal syncope are instructed how and when to apply physical countermaneuvers during vasovagal reactions provoked by head-up tilting. Usually a symptomatic vasovagal reaction returns after termination of the maneuver. This gives the opportunity to have patients use more than one maneuver to combat a vasovagal reaction and to compare the effects. Table 1 summarizes the maneuvers.

Table 1. Manuevers

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Maneuver Description</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBMT</td>
<td>Lower body muscle tensing: tensing of muscles in legs, buttock, and abdomen at maximal voluntary power</td>
<td>15</td>
</tr>
<tr>
<td>LCMT</td>
<td>Leg crossing with muscle tensing, i.e., tensing of muscles in legs, buttock, and abdomen at maximal voluntary power</td>
<td>9, 22, 28</td>
</tr>
<tr>
<td>WBT</td>
<td>Whole body tensing: tensing of all skeletal muscles at maximal voluntary power, except those in the instrumented left hand</td>
<td>12</td>
</tr>
<tr>
<td>SQT</td>
<td>Squatting</td>
<td>11, 20, 22</td>
</tr>
<tr>
<td>HBK</td>
<td>Sitting on a bed side with the head bent between the knees (“crash position”)</td>
<td>25</td>
</tr>
</tbody>
</table>

METHODS

Inclusion. From a consecutive series of 66 patients who developed a vasovagal reaction during tilt table testing, 26 patients were selected who successfully had applied either of the next two combinations of maneuvers: LBMT and LCMT (series A, n = 9) or WBT and LCMT (series B, n = 12). Patients who had squatted or applied HBK constituted series C (n = 14) and series D (n = 9), respectively. Consequently, there was some overlap between the series of patients.

Medical history in all patients (conducted by the senior investigator W. Wieling) consisted of at least one episode of loss of consciousness, with prodromal symptoms such as diaphoresis, pallor, or visual disturbances (“seeing black spots”) in the presence of any of the following triggers: (prolonged) orthostatic stress, pain, or the experience of an unusually strong emotional circumstance. Exclusion criteria for tilt testing were pregnancy or the presence of structural or (possibly) arrhythmogenic heart disease that could lead to syncope.

Tilt protocol and maneuvers. Investigations took place in the morning, at least 1 h after breakfast, in a room with a temperature of 23°C. Patients were strapped by a single torso belt to a manually controlled tilt table with a foot board. After 5 min of supine rest, patients were 60° tilted head up for 20 min. If no vasovagal reaction developed, 0.4 mg of nitroglycerin was administrated sublingually before an additional 15-min tilt (2). Beat-to-beat systolic and diastolic BPs were measured continuously noninvasively by use of Finapres model 5 (TNO Biomedical Instrumentation, Amsterdam, The Netherlands). The finger cuff was applied to the midphalanx of the left
middle finger. To avoid hydrostatic level differences, the hand was held at right atrial level in the midaxillary line. Finapres recordings accurately reflect BP changes during orthostatic stress (7). One of the investigators operated a marker pulse to identify the onset and termination of each maneuver. For offline analysis, all signals were digitally stored in a personal computer at a sampling rate of 100 Hz, as well as real-time printed by a thermo-paper writer (Thermal array;nation of each maneuver. For offline analysis, all signals were digi-
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investigators operated a marker pulse to identify the onset and termi-
In effects of maneuvers in series A and B
Effects of maneuvers in series A and B
as well as real-time printed by a thermo-paper writer (Thermal array-
tally stored in a personal computer at a sampling rate of 100 Hz, as

Before the test patients had practiced the maneuvers that they

All maneuvers started at the moment of a rapid fall in BP in
association with symptoms of impending syncope (Fig. 1). Each
maneuver was sustained for 40–60 s. Half of patients in series A
started with LBMT and applied LCMT at faint recurrence; the other
half performed these in reverse order. In series B, maneuver order
was split similarly.

In series C and D, patients were tilted back or had applied one of
the muscle tensing maneuvers when a faint was imminent. After a
subsequent head-up tilt or a release of the maneuver, they stepped off
the tilt table. Standing next to the table, the patients waited until faint
recurrence and then squatted in the tilt table. Standing next to the

Before the test patients had practiced the maneuvers that they
performed during the vasovagal reaction. Patients were instructed to
avoid straining during performance of the maneuvers.

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association with symptoms of impending syncope (Fig. 1). Each
maneuver was sustained for 40–60 s. Half of patients in series A
started with LBMT and applied LCMT at faint recurrence; the other
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was split similarly.

In series C and D, patients were tilted back or had applied one of
the muscle tensing maneuvers when a faint was imminent. After a
subsequent head-up tilt or a release of the maneuver, they stepped off
the tilt table. Standing next to the table, the patients waited until faint
recurrence and then squatted in series C and in series D performed
HBK. For safety reasons (i.e., risk of collapse when stepping off the
table and consequences), in our tilt lab patients aged >55 years are
never taught HBK or squatting when a vasovagal reaction is present.

During all testing, patients were closely monitored by two experi-
enced investigators. In case of syncope during or after a maneuver, the
tilt table was tilted down immediately or if the patient was standing
they were laid down on the bed.

SV and TPR computation. From the continuous BP measurement,
the arterial pulse wave was analyzed by a pulse wave analysis method,
which computes changes in left ventricular SV from the pulsatile
systolic area. We used the improved method of Wesseling, as de-
scribed in detail previously, using the Modelflow program (model-
based measurement method based on a nonlinear, 3-element model of
the input impedance of the aorta) (8). This methodology tracks rapid
changes in SV accurately (compared with gas rebreathing) during leg
crossing with and without muscle tensing (23). During conditions with
a low systemic BP, the technique provides accurate values for SV and
CO (6, 8).

CO was computed as SV times heart rate (HR), and TPR was
calculated as mean BP (MBP) divided by CO. MBP was obtained as

Table 2. Patient characteristics and baseline values per series

<table>
<thead>
<tr>
<th>Patient characteristics</th>
<th>Series A</th>
<th>Series B</th>
<th>Series C</th>
<th>Series D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LBMT vs. LCMT</td>
<td>WBT vs. LCMT</td>
<td>SQT</td>
<td>HBK</td>
</tr>
<tr>
<td></td>
<td>n = 12</td>
<td>n = 9</td>
<td>n = 14</td>
<td>n = 9</td>
</tr>
<tr>
<td>Age, yr</td>
<td>46 (18–80)</td>
<td>37 (16–71)</td>
<td>27 (16–52)</td>
<td>28 (16–38)</td>
</tr>
<tr>
<td>Sex</td>
<td>f/m</td>
<td>f/m</td>
<td>f/m</td>
<td>f/m</td>
</tr>
<tr>
<td>Height, meters</td>
<td>7 ±5 m</td>
<td>4 ±5 m</td>
<td>9 ±5 m</td>
<td>7 ±2 m</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>1.75 ±0.12</td>
<td>1.76 ±0.89</td>
<td>1.74 ±0.11</td>
<td>1.73 ±0.63</td>
</tr>
<tr>
<td>No. of syncopal episodes in 1 year</td>
<td>(2–10)</td>
<td>(2–20)</td>
<td>(3–30)</td>
<td>(5–30)</td>
</tr>
<tr>
<td>No. of syncopal episodes, life time</td>
<td>6 (1–40)</td>
<td>3 (1–30)</td>
<td>10 (1–40)</td>
<td>10 (1–35)</td>
</tr>
<tr>
<td>5-Min supine rest</td>
<td>116±16</td>
<td>105±12</td>
<td>120±17</td>
<td>123±14</td>
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<td>SBP, mmHg</td>
<td>63±9</td>
<td>56±8</td>
<td>63±10</td>
<td>66±8</td>
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<tr>
<td>DBP, mmHg</td>
<td>81±9</td>
<td>72±8</td>
<td>81±11</td>
<td>84±10</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>73±10</td>
<td>73±11</td>
<td>71±11</td>
<td>73±14</td>
</tr>
<tr>
<td>3-min head-up tilt</td>
<td>118±15</td>
<td>113±18</td>
<td>123±18</td>
<td>124±15</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>73±11</td>
<td>67±14</td>
<td>75±11</td>
<td>74±12</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>88±11</td>
<td>80±14</td>
<td>89±12</td>
<td>89±12</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>88±19</td>
<td>91±21</td>
<td>89±12</td>
<td>89±15</td>
</tr>
</tbody>
</table>

Age and number of syncopal episodes are given as median and range. Systolic (SBP), diastolic (DBP), and mean (MBP) blood pressure and heart rate (HR) are given as means and SD.

Table 3. Effects of maneuvers in series A and B

<table>
<thead>
<tr>
<th></th>
<th>LBMT vs. LCMT</th>
<th>LCMT</th>
<th>Series</th>
<th>WBT vs. LCMT</th>
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<tr>
<td></td>
<td>Start</td>
<td>10–40 s</td>
<td>Start</td>
<td>10–40 s</td>
<td>Start</td>
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<tr>
<td></td>
<td>maneuver</td>
<td>after start</td>
<td>maneuver</td>
<td>after start</td>
<td>maneuver</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>77±8</td>
<td>104±18</td>
<td>75±14</td>
<td>120±13</td>
<td>73±6</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>54±8</td>
<td>69±12</td>
<td>53±11</td>
<td>76±11</td>
<td>51±8</td>
</tr>
<tr>
<td>MBP, mmHg</td>
<td>61±7</td>
<td>79±13</td>
<td>60±12</td>
<td>89±11</td>
<td>56±7</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>96±28</td>
<td>103±20</td>
<td>93±23</td>
<td>97±16</td>
<td>94±27</td>
</tr>
<tr>
<td>SV, %bl</td>
<td>66±19</td>
<td>83±25</td>
<td>64±24</td>
<td>100±24</td>
<td>53±16</td>
</tr>
<tr>
<td>CO, %bl</td>
<td>69±12</td>
<td>95±21</td>
<td>65±17</td>
<td>110±22</td>
<td>54±12</td>
</tr>
<tr>
<td>TPR, %bl</td>
<td>104±22</td>
<td>99±20</td>
<td>62±30</td>
<td>96±21</td>
<td>137±38</td>
</tr>
</tbody>
</table>

Values are means and SD. Stroke volume (SV), cardiac output (CO), and total peripheral resistance (TPR) are given as percentage baseline (%bl), i.e., over the interval 2½–3 min after head-up tilt. *Significant difference between compared maneuvers, P < 0.05.
before the start of the maneuver over an interval of 3–5 beats and 2) over an interval after onset of each maneuver of 30 s when a stable BP was reached (Fig. 1). SV, CO, and TPR were expressed in percentage of head-up tilted baseline values (%bl).

For each maneuver we compared all parameters before and during the maneuver, using Student’s paired $t$-tests. In series A and B, we compared the parameters during each of the two maneuvers in each series, also using paired $t$-tests. $P$ values $<0.05$ were considered statistically significant.

**RESULTS**

Patient characteristics and hemodynamic base line values are given in Table 2.

On the basis of the nadir values and the presence of prodromal symptoms, at the start of each maneuver, all patients had a vasovagal reaction with imminent syncope if no measures were taken. BP, HR, and derived parameters before start of each maneuver are given in Tables 3 and 4.

All maneuvers caused an increase in BP (Tables 3 and 4, Fig. 2) varying from a rise in systolic blood pressure (SBP) from $77\pm8$ to $104\pm18$ mmHg ($P<0.05$) in series A during LBMT to a rise from $70\pm10$ to $123\pm9$ mmHg ($P<0.05$) in series B during LCMT. With the onset of each maneuver, BP started to increase after $\approx3$ s and in all patients stabilizing BP took less than 15 s. HR increased in LBMT, LCMT, and WBT ($P<0.05$). In HBK HR decreased from $99\pm14$ to $78\pm12$.

### Table 4. Effects of maneuvers in series C and D

<table>
<thead>
<tr>
<th></th>
<th>SQT</th>
<th></th>
<th></th>
<th>HBK</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Start maneuver</td>
<td>10–40 s after start</td>
<td>Start maneuver</td>
<td>10–40 s after start</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>76±13</td>
<td>122±15*</td>
<td>69±8</td>
<td>115±16*</td>
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<td></td>
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<tr>
<td>DBP, mmHg</td>
<td>50±10</td>
<td>74±10*</td>
<td>48±7</td>
<td>70±16*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>57±12</td>
<td>88±11*</td>
<td>53±8</td>
<td>82±15*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>90±21</td>
<td>80±8</td>
<td>99±14</td>
<td>78±12*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SV, %bl</td>
<td>65±19</td>
<td>115±15*</td>
<td>50±12</td>
<td>98±30*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CO, %bl</td>
<td>66±23</td>
<td>104±15*</td>
<td>57±16</td>
<td>84±15*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TPR, %bl</td>
<td>107±33</td>
<td>99±16</td>
<td>127±51</td>
<td>117±38*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means and SD. SV, CO, and TPR are given as percentage baseline, i.e. after 2.5–3 min head up-tilt. *Significant difference, $P<0.05$. 

Fig. 2. Effects of the various maneuvers on mean blood pressure (MBP), HR, SV, CO, and TPR. SV, CO, and TPR are represented as percentage from baseline (%bl, i.e. mean over the interval 2.5 to 3 min after head-up tilt). Each line represents the indexes at the start of and during the maneuver. The dashed lines with the white plotting-dot represent the group means. The graphs on LCMT combine all subjects in series A and B. *Significant change at $P<0.05$; NS, nonsignificant. WBT, whole body tensing; HBK, head bent between knees. See Table 1 for further description of maneuvers.
beats/min ($P < 0.05$). All maneuvers elicited an increase in CO (54 ± 12% baseline to 94 ± 21 in WBT to a rise from 65 ± 17 to 110 ± 22 in LCMT in series A) without significant changes in TPR. The hemodynamic effects of maneuver per group are given in Tables 3 and 4 and Fig. 2.

In series A, SBP rose to 104 ± 18 mmHg in LBMT, whereas in LCMT SBP stabilized at 120 ± 13 mmHg ($P < 0.05$). In series B WBT showed slightly less effective on BP than LCMT: SBP was 123 ± 19 vs. 115 ± 23 mmHg ($P < 0.05$). CO was lower during LBMT than during LCMT (95 ± 17 to 110 ± 22%bl, $P < 0.05$). All other values (including HR) within both series did not differ. In Fig. 3, MBP, HR, SV, CO, and TPR during the two compared maneuvers in series A and B are plotted.

**DISCUSSION**

This study shows that the circulatory effect of various physical countermaneuvers during vasovagal reactions is exclusively due to an increase in CO rather than any consistent effect on TPR. The small differences between the BP effects of LCMT and LBMT, respectively, WBT do not seem clinically relevant. The study also documents the efficacy of squatting and the crash position as countermaneuvers to increase BP immediately during vasovagal reactions.

Effects of physical countermaneuvers on CO and peripheral resistance. There are remarkable resemblances between the effects of the physical countermaneuvers in the present study on CO and those when inflating an antigravity suit at the onset of an impending vasovagal faint (26). CO rises by a factor of 1.3–1.7 in our maneuvers (Tables 3 and 4) and 1.4 during suit inflation (26). In both experiments, CO increased to ~100% of baseline upright values (Tables 3 and 4, Ref. 26). In another study in two healthy individuals, squatting caused a factor 1.6 increase in CO measured by dye dilution (11).

In the classical study by Weissler and coworkers (26), inflation of an antigravity suit during an impending vasovagal faint induced an instantaneous increase in central venous pressure. More recently, it was shown that leg muscle tensing during upright free standing also increased central venous pressure instantaneously (24). These observations document the rapid effect of these interventions on right ventricular filling pressure. However, an effect on systemic BP was observed with a latency of ~3 s (9, 26). The explanation for this latency is straightforward and was likely due to the time it took to translocate the venous blood from the right ventricle through the pulmonary circulation to the left ventricle and the systemic circulation (27).

In the above-mentioned antigravity suit experiment, the effects of the intervention were solely explained by an increase in CO (determined by the dye-dilution method) (26). No effects were observed on peripheral vascular resistance, and the vasovagal faints reoccurred after deflation of the antigravity suit. Accordingly, none of the physical countermaneuvers in the present study had any significant effect on TPR, and vasovagal reactions reoccurred after release of muscle tensing. The lack of an effect of physical countermaneuvers on vascular resistance may be surprising, but it is not when the time course of the effects of skeletal muscle tensing on sympathetic outflow and systemic vascular resistance under similar circumstances is considered. During preparation and initiation (first minute) of upright leg-cycling exercise in healthy volunteers, muscle sympathetic nerve activity is decreased, suggesting facilitation of increased muscle blood flow (4). During the first minute of sitting static leg exercise (at 30% of maximal voluntary power), muscle sympathetic nerve traffic was also found to decrease. One of the explanations given by the authors is loading of the cardiopulmonary baroreceptors due to the rapidly increased thoracic blood volume (13). In their study, muscle sympathetic activity only started to increase after 1 min of exercise (13). This increase in sympathetic nerve activity is mediated by the muscle chemoreflex, which is activated after ~1–2 min of static muscle exercise (14).
In our study, skeletal muscle tensing was only sustained for 40–60 s. Thus an increase in muscle sympathetic outflow and peripheral resistance is not to be expected. Previous observations that show that lower body muscle tensing during orthostatic stress increases orthostatic tolerance but does not increase peripheral vascular resistance (21) and that isolated hand gripping has only a trivial effect on orthostatic tolerance (9) are in excellent agreement with this explanation of the event.

The observation that isometric handgrip does not increase orthostatic tolerance (9) seems in conflict with the observation by Brignole et al. (3) that forceful isometric arm counterpressure maneuvers during free standing are effective to combat vasovagal reactions. We attribute this difference to leg and abdominal contractions that are inevitable during maximal isometric arm exercise in the upright position to stabilize the body.

So far we have attributed the rise in BP during muscle tensing maneuvers to mechanical effects on CO and not to reflex effects. The HR response, however, documents that reflex effects are involved as well. The instantaneous increase in HR at the onset of muscle tensing (Fig. 1, Ref. 9) is a reflex effect by a combination of central command and the muscle-heart reflex (1, 14). Reflex sympathetic stimulation of cardiac contractility at the onset of exercise may have contributed to the increased CO. In contrast to the muscle tensing maneuvers, the crash position (where no skeletal muscle tensing is involved but only passive abdominal compression) subsequently does not increase HR.

In squatting, where compression of arteries would be expected most prominently, TPR also did not change significantly, indicating that mechanical effects on arterial conductance play a minor role in the efficacy of the maneuvers. This corresponds to an earlier study that was unable to show an effect of squatting on TPR (11).

Limitations. A potential limitation of our study is that we did not objectify levels of skeletal muscle tension. Although we took care that the different maneuvers were performed with comparable effort in all subjects at all times, the lack of its measurement makes muscle tension an uncontrolled variable in the study. We also did not measure possible changes in intraabdominal and intrathoracic pressure. Especially in whole body tensing, unintentionally increasing intrathoracic pressure (closing the glottis when commencing whole body tensing) may have impaired venous return, thereby not fully effecting the maneuver’s potential.

A potential limitation of generalizability lays in our protocol in which we used nitroglycerine to induce a vasovagal reaction. Although this method is widely used and considered by many as a satisfactory model for vasovagal episodes outside the laboratory (2) there are indications that this provocation leads to predominantly CO (i.e., decreased cardiac filling)-mediated vasovagal reactions (5). Among vasovagal episodes outside the laboratory, the bradycardic and peripheral vasodilatory effects may be more prevalent. This study may thus overestimate the effects of physical countermaneuvers on CO, compared with vasovagal reactions without nitroglycerine provocation.

Clinical use of physical countermaneuvers. Although there are differences in the BP effects of the investigated maneuvers, they are small and, because all maneuvers prevented syncope, they do not seem clinically relevant. Thus, when advising patients, the daily applicability of each maneuver should be taken into account, and the advice can be customized to the individual patient’s ability to perform the maneuver.

Leg crossing (without muscle tensing) could be used in provocative situations as a preventive measure and either lower or whole body muscle tensing seems a reasonable next step when symptoms develop. If symptoms persist, patients could squat, which may be less embarrassing socially than the formerly advocated laying down. The crash position is especially appropriate when syncope occurs while sitting or for patients who have motor disabilities that would prevent squatting or make them more vulnerable in case of collapse. Recognition of prodromal signs is pivotal in the timely institution of any of the maneuvers, and thus the maneuvers do not apply to the relatively small subset of patients who experience syncope without warning. The range of maneuvers in this study could, however, offer physicians and the mainstay of their patients a means of tailoring therapy to the individual patient’s needs. By having a short list of readily accomplishable maneuvers, patients could increase control over their symptoms and potentially improve the quality of their lives.

REFERENCES


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