Management of Vasovagal Syncope
Controlling or Aborting Faints by Leg Crossing and Muscle Tensing

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Background—Posture-related vasovagal syncope is by far the most frequent cause of transient loss of consciousness, and present pharmacological and cardiac pacing treatment remains unsatisfactory. A simple maneuver to prevent or diminish vasovagal reactions would be beneficial.

Methods and Results—Twenty-one patients with recurrent syncope (age 17 to 74 years, 11 males) who were referred for routine tilt-table testing and had a positive test were included. They were instructed to perform leg crossing and muscle tensing for at least 30 seconds at the onset of a tilt table–provoked impending faint. Continuously measured blood pressure and heart rate at nadir and during the maneuver were compared. Ten months after the test, a telephone follow-up was performed. The physical counter-maneuver, performed in 20 of 21 subjects, increased blood pressure and heart rate. Systolic blood pressure rose from 65±13 to 106±16 mm Hg (mean±SD, P<0.001), and diastolic blood pressure rose from 43±9 to 65±10 mm Hg (P<0.001). During the maneuver, prodromal symptoms disappeared in all patients, and none lost consciousness. After terminating the maneuver, symptoms did not return in 5 subjects during the test. At follow-up, 13 of 20 patients reported that they applied the maneuver in daily life and benefited from it.

Conclusions—Leg crossing combined with tensing muscles at the onset of prodromal symptoms can postpone and in some subjects prevent vasovagal syncope. (Circulation. 2002;106:1684-1689.)

Key Words: blood pressure ■ syncope ■ tests ■ hemodynamics ■ pacemakers

Vasovagal reflex syncope is the most frequent cause of transient loss of consciousness.1–3 The vasovagal reaction consists of vasodilatation and a heart rate decrease. During prolonged standing, this reaction is triggered by a reduction of the central blood volume because of pooling in the lower body veins, sometimes combined with other provocative factors.3–6 Patients with reflex syncope may suffer from recurrent loss of consciousness, varying from once a year to weekly or even daily episodes. Most of these patients also experience frequent presyncope, which can be just as incapacitating as syncope itself. Vasovagal syncope is usually not a dangerous condition, because episodes are self-limiting. However, the quality of life of patients with recurrences can be seriously affected.7 The rapid loss of consciousness and the possibility of trauma tax the patient’s sense of physical control and self-esteem.

The present management of vasovagal syncope consists of providing the patient with an explanation of the pathophysiology involved and advising him or her to avoid provocative situations and to increase salt intake. Various drugs have been proposed in the treatment of vasovagal syncope. In general, although the results have been satisfactory in uncontrolled or short-term controlled trials, several long-term prospective trials have been unable to show consistent benefit of the active drug over placebo.1 There is strong consensus of opinion that the role for pacing in the treatment of patients with vasovagal syncope is minor.1 Thus, a simple and effective interventional approach relevant to most patients suffering from vasovagal syncope and without side effects would be an important addition to present management and helpful for combating presyncope.

In patients with orthostatic hypotension attributable to autonomic failure, crossing legs increases orthostatic tolerance by decreasing blood pooling in the lower body veins.8–14 Muscle tensing enhances this effect.13 Inflation of an anti-gravity suit that promotes the return of pooled blood from the lower body and increases cardiac afterload can abort an impending vasovagal faint.15,16 Against this background information, we addressed the hypothesis that leg crossing combined with tensing of leg, abdominal, and buttock muscles can be applied as a natural means of improving venous return such that the vasovagal syncope is aborted or at least
temporarily controlled. We report results in 21 consecutive patients with vasovagal syncope that support this hypothesis.

Methods
We included consecutive patients who were referred to the Syncope Unit of the Academic Medical Center for routine tilt-table testing who developed a vasovagal reaction during the test. From March to September 2001, 58 patients underwent a tilt-table test at our laboratory for suspected vasovagal syncope. Twenty-seven developed a vasovagal reaction during the test. Six were excluded because of inability to perform the counter-maneuver. The remaining 21 patients constitute our study population, and their characteristics are given in Table 1. ECG and echocardiography, performed in all patients, revealed no structural heart disease of clinical relevance. Patients had no comorbidities of clinical relevance.

Before the test, subjects received oral instruction on how to perform the maneuver (Figure 1) and practiced it once. We used a manually controlled tilt table with a foot board. Subjects were not strapped to the tilt table, to provide the freedom of movement to perform the maneuver. Risk of falling and potential for injury were minimized by close observation of the patient by 2 experienced investigators (C.T.P.K. and W.W.) and continuous blood pressure monitoring. One of the investigators was ready to tilt the patient back to horizontal position immediately, in case of imminent syncope. The manually controlled tilt table allowed a tilt back in 1 second.

Beat-to-beat systolic and diastolic blood pressures and heart rate were measured continuously and noninvasively using Finapres model 5 (TNO Biomedical Instrumentation, Amsterdam, the Netherlands).

The tilt-table test started with 5 minutes of supine rest. The subjects were then tilted head up (60 degrees) for 20 minutes. If no vasovagal faint developed, nitroglycerin was administrated sublingually (0.4 mg) before an additional 15-minute tilt. At the moment of a relentless fall in blood pressure accompanied by prodromal symptoms indicating an impending faint, subjects were instructed on verbal command to start the physical counter-manuever. They were asked to uncross their legs after at least 30 seconds following the disappearance of prodromal symptoms. If symptoms returned, subjects resumed the counter-maneuver until symptoms disappeared. In case syncope appeared imminent in spite of the maneuver, subjects were tilted back within 1 second.

An example of the blood pressure and heart rate tracings during the maneuver is given in Figure 2. Averaged systolic and diastolic blood pressure and heart rate were determined between 4.5 and 5 minutes of supine rest, between 2.5 and 3 minutes of head-up tilt, between 2 and 1.5 minutes before the first episode of leg crossing, and during the 30 seconds immediately after blood pressure was stabilized by the physical counter-maneuver. The lowest blood pressure and heart rate values of the impending faint were determined (Figure 2). The following latencies (in seconds) were determined between the start of the counter-maneuver and the increase of blood pressure and the blood pressure nadir and a stabilized blood pressure.

Data fit a normal distribution. The differences of blood pressure and heart rate at nadir and during the maneuver were examined by paired t test.

A telephone follow-up was performed 10 months (median; range, 7 to 14 months) after the tilt-table test. Patients were asked if they had experienced any syncopal or presyncopal events in the period after the test and whether they had used the counter-maneuver and, if so, benefited from it.

We performed an additional experiment to assess the contribution of a central nervous drive (central command) to the cardiovascular events induced by the physical counter-maneuver. We compared the effects of leg crossing and lower body muscle tensing with those of hand gripping. Three consecutive tilt-positive patients performed isometric handgrip exercise at maximal voluntary at the moment of an impending faint.

The study was approved by the Medical Ethical Committee of the Academic Medical Center, University of Amsterdam, the Netherlands.

Results
Values of supine and orthostatic blood pressure and heart rate are given in Table 2. Four subjects developed a vasovagal reaction without and 17 of 21 after the addition of nitroglycerine.

During the first vasovagal episode, systolic blood pressure decreased to 65±13 mm Hg (mean±SD) and diastolic blood pressure to 43±9 mm Hg. A total of 14 of 21 subjects had a systolic blood pressure <75 mm Hg and 7 of 21 <60 mm Hg. In 10 of 21 subjects, heart rate decreased >10 bpm in the 30 seconds before the maneuver. Prodromal symptoms were present in all patients. On the basis of these observations, we concluded that at the moment they started the maneuver, all patients were experiencing a vasovagal reaction with development of syncope if no counter measures were instituted.
One subject developed a pronounced tachycardia (heart rate supine, 62 bpm; just before maneuver, 130 bpm). The other subjects showed a stable or slightly increased heart rate. One subject was near to unconsciousness before performing the physical counter-maneuver and was tilted back. The remaining 20 subjects performed the physical counter-maneuver 1 to 4 times. Performing the maneuver stabilized blood pressure (Figure 3) and heart rate in all subjects. Prodromal symptoms vanished during the performance of the maneuver in all subjects shortly after stabilization of blood pressure. None of the subjects lost consciousness during performance of the maneuver.

In 5 of 20 subjects, the vasovagal reaction was averted by the maneuver (Figure 3A through 3E). The remaining 15 subjects could not avert the faint or requested to be tilted back after having performed the maneuver but did postpone the faint by on average 2.5 minutes (range, 30 seconds to 11 minutes) (Figure 3F through 3T).

During the first episode of leg crossing, systolic blood pressure rose from 65±13 to 106±16 mm Hg (P<0.001) and diastolic blood pressure rose from 43±9 to 65±10 mm Hg (P<0.001). Heart rate increased from 73±22 to 82±15 bpm (P<0.01). In the subject with postural tachycardia (heart rate, 130 bpm), heart rate decreased during the physical counter-maneuver to 108 bpm, whereas blood pressure rose from 54/41 to 100/71 mm Hg.

The latency between the start of the physical counter-maneuver and the start of the increase of blood pressure ranged from 3 to 6 seconds. In some subjects, an almost instantaneous increase in blood pressure was observed, whereas in others blood pressure rose slowly (Figure 3).

Patients who could completely abort the faint started the maneuver at a significantly higher blood pressure level than patients who could not (79/51 versus 61/41 mm Hg, P<0.01). The latencies between the blood pressure nadir and stabilization of blood pressure were on average 9 seconds (range, 3 to 18 seconds).

For the follow-up interview, 19 of 20 subjects who had performed the maneuver on the tilt table were contacted. Their number of recurrences is given in Table 3. In one subject, Addison’s disease was diagnosed during follow-up. Three subjects had had no syncopal complaints since the test. Two subjects who still suffered from faints did not use the maneuver; one of them reported a too short prodromal period to apply. The remaining 13 patients used the counter-maneuver in daily life for preventing or controlling syncope. Ten patients who, apart from syncope, had suffered from presyncope as well, indicated that they also benefited from the maneuver to alleviate presyncopal complaints.

The results of isometric handgrip exercise at the moment of an impending faint are given in Figure 4. With hand gripping there was some stabilizing effect on blood pressure but far less pronounced than during leg crossing and tensing. Hand gripping could not abort the faint, and all 3 patients had to be tilted back to horizontal within 1 minute.

**Discussion**

The main finding of this study is that crossing legs can abort or delay impending faints in subjects prone to vasovagal reactions. Previous case reports have indicated a beneficial effect of skeletal muscle pumping and tensing on blood pressure and heart rate in patients with vasovagal syncope. This is the first report that documents the efficacy of aborting a vasovagal faint by leg crossing and muscle tensing.

### Table 2. Blood Pressure and Heart Rate Responses During the Tilt-Table Test in 20 Subjects Who Performed the Physical Counter Maneuver

<table>
<thead>
<tr>
<th>Condition</th>
<th>Systolic Blood Pressure, mm Hg (SD)</th>
<th>Diastolic Blood Pressure, mm Hg (SD)</th>
<th>Heart Rate,* bpm (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>After 5 minutes of supine rest</td>
<td>120 (16)</td>
<td>62 (9)</td>
<td>72 (14)</td>
</tr>
<tr>
<td>After 3 minutes of head-up tilt</td>
<td>117 (11)</td>
<td>69 (7)</td>
<td>84 (15)</td>
</tr>
<tr>
<td>90 seconds before BP nadir</td>
<td>105 (13)</td>
<td>68 (8)</td>
<td>91 (18)</td>
</tr>
<tr>
<td>At nadir</td>
<td>65 (13)</td>
<td>43 (9)</td>
<td>73 (22)</td>
</tr>
<tr>
<td>During maneuver</td>
<td>106 (16)</td>
<td>65 (10)</td>
<td>82 (15)</td>
</tr>
</tbody>
</table>

*n=19; subject with postural tachycardia (130 bpm) is excluded from calculation.
in a series of consecutive patients and the effectiveness of the maneuver in daily life. The maneuver also seems to be effective in combating presyncopal complaints.

The posture-related vasovagal reaction is thought to be elicited in response to a postural reduction of the central blood volume. The effects of leg crossing are explained by breaking the vicious cycle that maintains the ongoing vasovagal reflex. Weissler et al demonstrated that an impending vasovagal faint could be aborted by inflation of antigravity suit. They observed rapid increases in central venous pressure and cardiac output, indicating reinfusion of pooled blood. Previous studies have shown an increase in central venous pressure and cardiac output during muscle tensing. Changes in peripheral resistance should also be considered as a contribution to the stabilization of blood pressure. Sustained tensing of skeletal muscles is associated with activation of a central nervous drive (central command) and of the muscle chemoreflex. These mechanisms induce an increase in sympathetic outflow and thereby in peripheral resistance with stabilization of blood pressure. In addition to these neurogenic effects, mechanical effects of muscle tensing on peripheral conductance could be involved. The muscle chemoreflex is not likely to play an important role in the instantaneous blood pressure–raising effect of the physical counter-maneuver, because muscle chemo-aффerents are activated only after ~1 minute of sustained muscle contractions. The trivial effect of hand gripping on the fainting response (Figure 4) suggests that central command plays a minor role only. Thus, the mechanical effects of the combination of leg crossing and muscle tensing alone seem to explain almost all of the blood pressure–raising effect. In contrast, the instantaneous increase in heart rate observed at the onset of the physical counter-maneuver (Figure 1) is likely to be of neurogenic origin. It may be attributed to withdrawal of vagal outflow to the heart related to the muscle-heart reflex or central command.

The latency between the start of the physical counter-maneuver and the subsequent stabilization of blood pressure can be explained by various factors. One variable is the time needed by the presyncopal subject to perform the maneuver effectively. Other factors include unintentional straining during the onset of the maneuver and transit delay of the venous return through the pulmonary circulation.

For patients in whom the vasovagal episode cannot be aborted by the maneuver, the maneuver was useful in postponing the syncpe. The subsequent fall in blood pressure

### TABLE 3. Follow-up

| Patient No.* | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O | P | Q | R | S | T |
| No. of faints last year | 3 | 2 | 1 | 3 | 10 | 20 | 30 | 2 | 1 | 2 | 1 | 1 | 1 | 0† | 5 | 0† | 24 | 6 | 1 | 13 |
| No. of faints during lifetime | 10 | 3 | 2 | 6 | 20 | 50 | 100 | 2 | 1 | 11 | 2 | 1 | 10 | 3 | 6 | 3 | 30 | 22 | 1 | 13 |
| Follow-up time, mo | 9 | 11 | 9 | 10 | ‡ | 12 | 7 | 9 | 11 | 10 | 11 | 12 | 13 | 11 | 10 | 10 | 13 | 13 | 9 | 8 |
| Application of maneuver during follow-up | Yes | Yes | No | Yes | ‡ | No§ | Yes | Yes | Yes | No | No | Yes | Yes | Yes | Yes | Yes | Yes | Yes | No | Yes |
| No. of faints during follow-up | 0 | 0 | 0 | 0 | ‡ | 20 | 0 | 0 | 1 | 1‖ | 0 | 3 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 |

*Numbers correspond to Figure 3.
†Last year: Severe presyncopal complaints.
‡Patient could not be contacted.
§Patient reports: too short prodromal period to apply maneuver.
‖During follow-up, diagnosis of Addison’s disease.
after uncrossing may be counteracted by maintaining the muscles tensed after uncrossing. They can then sit or lay down controlled, keeping their blood pressure stabilized by tensing.

Sheldon and colleagues\textsuperscript{31,32} followed up a large group of patients with vasovagal syncope induced by head-up tilt-table testing for up to 3 years and developed a predictive model for recurrence of syncope after a positive tilt-table test. They found that the frequency of syncopal events decreased substantially after head-up tilt-table testing.

It has been suggested that the diagnostic procedure of head-up tilt-table testing and the associated clinical encounter, including counseling on avoidance of situational provocation, has the effect of a positive therapeutic intervention.\textsuperscript{3} This would be associated with a reduction in the number of events in follow-up, and therefore the absence of a control group is a potential limitation of our study.

On the basis of Sheldon’s predictive model, we estimated the recurrence risk in the follow-up cohort without intervention at 0.30. The observed recurrence rate of 0.15 after 10 months of follow-up supports that, apart from the effect of tilt-table testing itself, application of the physical counter-maneuver has contributed to the reduced frequency of events.

Laboratory studies show that pacing during the onset of a vasovagal faint has a modest stabilizing effect on blood pressure by increased heart rate supporting cardiac output.\textsuperscript{33} However, it does not counteract the vasodilatation. Therefore, cardiac pacing has in general been proven to be successful in prolonging the premonitory warning phase of vasovagal syncope.\textsuperscript{1} The combination of leg crossing and muscle tensing at the onset of a vasovagal faint seems to have a greater blood pressure–raising effect than cardiac pacing and overall at least the same beneficial effect.

We therefore propose that the physical counter-maneuver should be considered in patients with vasovagal syncope before cardiac pacing treatment because it offers a safe, inexpensive, and effective alternative. This easy-to-perform maneuver has a significant clinical effect, is without any side effects or additional patient burden, and may be equally effective in combating presyncope and syncope. The only limitations to the use of the maneuver are motor handicaps and absence of warning time. The observation that the patients who aborted the fainted started the maneuver at a significant higher blood pressure than the patients who could not emphasized the importance of an early commencement of the maneuver.

Because early patient recognition of prodromal symptoms is the key to adequately performing the physical counter-maneuver, the tilt-table test provides patients with a safe setting to become familiar with their prodromal symptoms so they can use them as a cue to apply the physical counter-maneuver.

**Conclusion**

Leg crossing combined with muscle tensing applied as a simple physiological measure at the onset of prodromal symptoms can prolong the time to or prevent vasovagal syncope. By aborting or delaying syncope, this maneuver can increase patients’ sense of control over their symptoms and thereby improve their quality of life.

**References**


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