Isometric Arm Counter-Pressure Maneuvers to Abort Impending Vasovagal Syncope

Michele Brignole, MD,* Francesco Croci, MD,* Carlo Menozzi, MD,† Alberto Solano, MD,* Paolo Donateo, MD,* Daniele Oddone, MD,* Enrico Puggioni, MD,* Gino Lolli, MD†
Lavagna and Reggio Emilia, Italy

OBJECTIVES
We hypothesized that isometric arm exercises were able to increase blood pressure (BP) during the phase of impending vasovagal syncope and allow the patient to avoid losing consciousness.

BACKGROUND
Hypotension is always present during the prodromal phase of vasovagal syncope.

METHODS
We evaluated the effect of handgrip (HG) and arm-tensing in 19 patients affected by tilt-induced vasovagal syncope. The study consisted of an acute single-blind, placebo-controlled, randomized, cross-over tilt-table efficacy study and a clinical follow-up feasibility study.

RESULTS
In the acute tilt study, HG was administered for 2 min, starting at the time of onset of symptoms of impending syncope. In the active arm, HG caused an increase in systolic blood pressure (SBP) from 92/61 mm Hg to 105/38 mm Hg, whereas in the placebo arm SBP decreased from 91/61 mm Hg to 73/21 mm Hg (p = 0.008). Heart rate behavior was similar in the two arms. In the active arm, 63% of patients became asymptomatic, versus 11% in the control arm (p = 0.02); conversely, only 5% of patients developed syncope, versus 47% in the control arm (p = 0.01). The patients were trained to self-administer arm-tensing treatment as soon as symptoms of impending syncope occurred. During 9±3 months of follow-up, the treatment was actually performed in 95/97 episodes of impending syncope (98%) and was successful in 94/95 (99%). No patients suffered injury or other adverse morbidity related to the relapses.

CONCLUSIONS
Isometric arm contraction is able to abort impending vasovagal syncope by increasing systemic BP. Arm counter-pressure maneuvers can be proposed as a new, feasible, safe, and well accepted first-line treatment for vasovagal syncope. (J Am Coll Cardiol 2002;40:2053–9)

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Vasovagal syncope is preceded by prodromal symptoms in about two-thirds of cases (1). Prodromal symptoms are present in virtually all cases of tilt-induced vasovagal syncope, which occurs, on average, 1 min after the onset of prodromal symptoms (2). During the prodromal phase, blood pressure (BP) falls markedly; this fall usually precedes the decrease in heart rate (HR), which may be absent at least at the beginning of this phase (2,3). Hypotension is caused by vasodilation in the skeletal muscles due to inhibition of sympathetic vasoconstrictive activity (2–7). In normal and hypertensive subjects, isometric handgrip (HG) exercises are able to induce a significant BP increase, which is mediated largely by endogenous catecholamine release (8,9). Muscle sympathetic nerve discharge and vascular resistance increase during HG in healthy subjects (10). We hypothesized that isometric arm exercises were able to increase BP during the phase of impending vasovagal syncope and allow the patient to avoid losing consciousness.

METHODS
The study enrolled patients affected by vasovagal syncope and consisted of an acute tilt-table efficacy study and a clinical follow-up feasibility study. In addition, the physiological cardiovascular response to two isometric arm exercises was evaluated in healthy subjects.

We studied patients affected by vasovagal syncope who had the following: a history of ≥1 episode of syncope; one or more syncope episodes preceded by prodromal symptoms that were recognized by the patient as symptoms of impending syncope; syncope reproduced during two tilt tests performed on different days; age ≥18 years.

During the second baseline tilt test, the patients were instructed to recognize the onset of prodromal symptoms. The Italian tilt protocol (11), namely 60° passive tilting followed by 0.4 mg nitroglycerine challenge when the passive phase fails to induce syncope, was used in this test and for those of the acute study. Continuous recording of electrocardiogram (ECG) tracing and noninvasive beat-to-beat arterial BP was performed by means of the Finapres method (12). The average value of six consecutive beats was considered for analysis. The new VASIS classification was used to stratify positive responses (3).
Acute tilt-table study. The acute tilt study was a single-blind, placebo-controlled, randomized, cross-over study which was designed to evaluate the ability of HG to abort vasovagal syncope induced during tilt testing. The patients underwent two tilt tests, at least 1 h apart, on the same day. During one test, active HG treatment (using a Vigorimeter Martin cuff manometer, according to the standard protocol) (8, 9) was administered for 2 min at 50% of maximal voluntary contraction. During the other test, the placebo HG treatment was administered for 2 min without contraction. The sequential order of the treatments was randomized. In both cases, treatment was started at the time of onset of symptoms of impending syncope, while the patient was standing on the tilt table. The treatment—and the test—was interrupted in the event of syncope occurrence. If syncope did not occur, the patient was tilted for a further 2 min after the end of treatment (recovery phase). Again, the recovery phase—and the test—was interrupted in the event of syncope occurrence.

Impending syncope was defined as the onset of one or more of the following symptoms: weakness, dizziness, abdominal discomfort, nausea, sweating, sighing, and blurred vision, associated with marked systolic blood pressure (SBP) drop.

Cardiovascular response to HG and arm-tensing in healthy subjects. In 32 healthy volunteers (mean age 44 ± 12, 16 males), we evaluated the physiological response to 2 min of isometric contraction during standard HG (as described earlier) and during arm-muscle tensing exercise. Arm-tensing consisted of the maximum tolerated isometric contraction of the two arms achieved by gripping one hand with the other and contemporarily abducting (pushing away) the arms. The tests were performed on a tilt table at 60°; ECG tracing and noninvasive beat-to-beat arterial BP were continuously recorded. The sequential order of the maneuvers was randomized.

This study had two aims: to evaluate the cardiovascular response during HG tilting in healthy subjects and to compare HG with arm-tensing. Indeed, because arm-tensing does not require the use of any equipment, it seems more suitable, in that it can be performed during daily life to abort spontaneous attacks.

Follow-up. Irrespective of the results of the acute tilt phase, all patients were trained to perform the arm-tensing maneuver and were discharged with the recommendation to self-administer it at the maximum tolerated voluntary contraction as soon as symptoms of impending syncope impending.

Table 1. Characteristics of the Patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>19</td>
</tr>
<tr>
<td>Mean age, yrs</td>
<td>55 ± 20</td>
</tr>
<tr>
<td>Males</td>
<td>12 (63%)</td>
</tr>
<tr>
<td>History of syncope</td>
<td></td>
</tr>
<tr>
<td>Median number of syncopes (interquartile range)</td>
<td>3 (1–11)</td>
</tr>
<tr>
<td>Duration (interquartile range), yrs</td>
<td>3 (1–10)</td>
</tr>
<tr>
<td>Typical vasovagal/situational triggers</td>
<td>7 (37%)</td>
</tr>
<tr>
<td>Postprandial</td>
<td>2 (11%)</td>
</tr>
<tr>
<td>Secondary trauma</td>
<td>4 (21%)</td>
</tr>
<tr>
<td>Number of patients with pre-syncope</td>
<td>11 (58%)</td>
</tr>
<tr>
<td>Associated conditions</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>3 (16%)</td>
</tr>
<tr>
<td>Cardiac abnormalities</td>
<td>3 (16%)</td>
</tr>
<tr>
<td>Vasoactive therapy</td>
<td>5 (26%)</td>
</tr>
<tr>
<td>Baseline tilt-test responses (during repeat tilt)</td>
<td></td>
</tr>
<tr>
<td>Positive during passive/nitroglycerin phase</td>
<td>2/17</td>
</tr>
<tr>
<td>Cardiocinhibitory, mixed, vasodepressor type</td>
<td>5/12/2</td>
</tr>
<tr>
<td>Classic/dysautonomic hemodynamic pattern</td>
<td>12/7</td>
</tr>
<tr>
<td>Systolic blood pressure at time of impending syncope, mm Hg</td>
<td>88 ± 9</td>
</tr>
<tr>
<td>Heart rate at time of impending syncope, beats/min</td>
<td>82 ± 21</td>
</tr>
</tbody>
</table>
respectively, in the two arms. At that time, treatment was undertaken for 2 min or until syncope occurred. In the active arm, BP rose very quickly, then stabilized, whereas in the control arm BP continued to fall; the difference was significant. Heart rate decreased to the same extent in the two arms. Figures 2 and 3 show two illustrative cases. In the active arm, 12 (63%) patients became asymptomatic versus only 2 (11%) in the control arm (p = 0.02); conversely, only 1 patient (5%) developed syncope (after 70 s) in the active arm, whereas 9 (47%) patients developed syncope in the control arm (after 66 ± 36 s) (p = 0.01). During the recovery phase, syncope occurred in 3 and 2 patients, respectively. Overall, four patients had syncope induced during both active and control studies and seven had syncope induced during the control study but not during the active study (21% vs. 58%, p = 0.02).

**Cardiovascular response to HG and arm-tensing in healthy subjects.** In healthy subjects SBP, diastolic blood pressure (DBP), and HR increased to a similar extent during HG and arm-tensing (Fig. 4). Indeed, SBP increased from 125 ± 18 mm Hg to 156 ± 26 mm Hg during HG and from 123 ± 15 mm Hg to 155 ± 24 during arm-tensing; DBP increased from 72 ± 10 mm Hg to 94 ± 16 mm Hg during HG and from 73 ± 11 mm Hg to 97 ± 17 mm Hg during arm-tensing; HR increased from 76 ± 14 beats/min to 84 ± 16 during HG and from 75 ± 13 to 86 ± 15 during arm-tensing.

The maximum increase in SBP was +31 ± 16 mm Hg during HG and +32 ± 18 mm Hg during arm-tensing; these values were only slightly higher than the maximum increase observed in the active arm of the syncope patients, which was +29 ± 41 mm Hg (not statistically different). On the contrary, HR behavior showed opposite patterns, increasing in healthy subjects (+9 ± 11 beats/min and +11 ± 11 beats/min during HG and arm-tensing, respectively) and decreasing in syncope patients (-8 ± 17 beats/min); this difference was highly significant (p = 0.000).

**Follow-up.** Follow-up data on 18 patients are available. During a mean follow-up of 9 ± 3 months, 11 patients experienced a total of 97 symptoms of impending syncope (median 3, interquartile range 2 to 5.5). The patients were able to self-administer the treatment 95/97 times (98%); on two occasions two patients had a syncopal relapse but were unable to perform the maneuver. The treatment was successful 94/95 times (99%); in one case syncope developed despite treatment. No patients had injury or other adverse morbidity related to the relapses. Patient satisfaction was very good: 64% of the patients were very satisfied with the treatment and 36% were moderately satisfied.

### DISCUSSION

This study shows that isometric arm contraction is able to abort impending vasovagal syncope by increasing systemic BP. Long-term treatment based on self-administered arm counter-pressure maneuvers is feasible, safe, and well accepted by the patient. Arm counter-pressure maneuvers can be proposed as a new first-line treatment for those patients who are able to recognize prodromal symptoms before vasovagal syncope.
Some physical counter-maneuvers have been proposed in the management of orthostatic hypotension (13–16). These mainly involve the muscles of the legs (leg-pumping and tensing, leg-crossing) or legs and abdomen (abdominal contraction, squatting). The increase in orthostatic BP is presumed to be due both to mechanical compression of the venous vascular bed in the legs and to a reflex increase in systemic vascular resistances caused by the activation of muscle mechanosensitive receptors. In a very recent uncontrolled study (17), leg crossing combined with tensing muscles at the onset of prodromal symptoms, performed in 20/21 subjects, increased SBP from 65 ± 13 mm Hg to 106 ± 16 mm Hg, postponed the faint by on average 2.5 min and in five subjects prevented vasovagal syncope; during the maneuver, symptoms disappeared in all patients. Cardiovascular responses during HG have been studied in healthy and in hypertensive patients (8–10), but not in fainting patients. Arm-tensing maneuvers have not been previously devel-

![Figure 1. Acute tilt-table study results. Systolic blood pressure (SBP) (A) and heart rate (HR) (B) in the active treatment arm (continuous line) and control arm (dotted line). Values are expressed as mean ± 1 SE. During the test, some patients had syncope and the test was interrupted; numbers at the top refer to patients free of syncope at that time. (A) Handgrip (HG) started at the onset of impending syncope; compared with placebo, active HG caused a significant increase in SBP, which was already significant after 10 s. (B) Heart rate behavior was similar in the two arms. There was an initial compensatory HR increase, which peaked at the time of impending syncope. *p < 0.05.](image-url)
Figure 2. A case of handgrip (HG) tilting. The top trace shows the heart rate (HR) curve; the bottom trace shows systolic, diastolic, and mean blood pressure (BP) curves. Symptoms of impending syncope occurred when systolic BP fell to 80 mm Hg with an abrupt drop in HR from 95 beats/min to 65 beats/min. During treatment, BP progressively increased and symptoms disappeared. The vagal bradycardic reflex was also quickly interrupted, and HR rose in an oscillatory manner to baseline values. During the recovery phase, systolic BP decreased but remained at values higher than before and symptoms did not recur.

Figure 3. A case of control and active handgrip (HG) tilting in the same patient. The top trace shows the heart rate (HR) curve; the bottom trace shows systolic, diastolic, and mean blood pressure (BP) curves. Right panel, control. The pattern was that of a typical tilt-induced vasovagal reaction, with hypotension and bradycardia. The arrow indicates the time of onset of symptoms of impending syncope, when placebo HG was administered. Subsequently, BP and HR continued to fall and the patient suffered syncope after 45 s. Left panel, active treatment. Initially, the pattern of BP and HR was similar to that observed in the control study. The start of HG caused a rapid rise in BP, which persisted as long as the contraction was maintained; initially, HR slightly increased and then slightly decreased; symptoms disappeared. During the recovery phase, SBP fell again to 90 mm Hg and symptoms reappeared.
cular adjustments elicited by separate limbs are not simply additive, but rather exhibit an inhibitory interaction. We observed similar responses between one-arm HG and two-arm arm-tensing.

During tilt-induced vasovagal reaction, HG caused an abrupt rise in systemic BP, which was already evident after 10 s. Consequently, symptoms of impending syncope disappeared in many patients and remained unchanged in others, and syncope was aborted. Conversely, in the control arm, BP continued to fall slightly and approximately half of the patients developed syncope after a mean of 66 s. The benefits were maintained during the recovery phase, and only 20% of patients ultimately developed syncope (vs. 58% in the control arm). This finding means that isometric arm contraction is able to abort syncope in most cases, even when the patient remains in the standing position. The practical consequence is that when symptoms of impending syncope occur, the patient will have enough time to apply the counter-pressure treatment before losing consciousness. In some cases, the treatment will definitely abort the vasovagal reaction, in others it will be able to delay syncope for the duration of the maneuver, thus allowing enough time to initiate other maneuvers to abort syncope (e.g., supine posture). This approach seems to be very helpful in real life. Indeed, during follow-up our patients were able to enact a counter-pressure maneuver in 98% of cases and to relieve symptoms in 99% of these. The treatment is therefore easy to perform, reliable, safe and well accepted by the patients, who expressed good satisfaction. Admittedly, most of these episodes would have resolved spontaneously without leading to syncope, even in the absence of the counter-pressure treatment. Owing to the open design of the follow-up study, we are unable to establish the exact benefit of the treatment. A randomized trial should address this question. In any case, counter-pressure maneuvers can be regarded as a first-line treatment in association with the other conventional measures usually recommended for vasovagal syncope: namely, reassurance regarding the benign nature of the condition, training in the recognition of premonitory symptoms, avoidance of triggering events, the adoption of maneuvers to abort the episode (e.g., supine posture), and avoidance of volume depletion and prolonged upright posture. With regard to these latter treatment concepts, formal randomized studies are also lacking, but physiological evidence and clinical experience have been sufficient to warrant their inclusion in the available guidelines (21).

Reprint requests and correspondence: Dr. Michele Brignole, Department of Cardiology, Ospedali del Tigullio, Via don Bobbio, 16033 Lavagna, Italy. E-mail: mbrignole@ASL4.liguria.it.

REFERENCES