Water drinking improves orthostatic tolerance in patients with posturally related syncope

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ABSTRACT

Water drinking improves OT (orthostatic tolerance) in healthy volunteers; however, responses to water in patients with PRS (posturally related syncope) are unknown. Therefore the aim of the present study was to examine whether water would improve OT in patients with PRS. In a randomized controlled cross-over fashion, nine patients with PRS ingested 500 ml and 50 ml (control) of water 15 min before tilting on two separate days. OT was determined using a combined test of head-up tilting and lower body suction and expressed as the time required to induce presyncope. We measured blood pressure and heart rate (using Portapres®) and middle cerebral artery velocity (using transcranial Doppler). SV (stroke volume) and TPR (total peripheral resistance) were calculated using the Modelflow® method. OT was significantly ($P < 0.02$) greater after drinking 500 ml of water than after 50 ml (25.4 ± 1.5 compared with 19.8 ± 2.3 min respectively). After ingestion of 500 ml of water, blood pressure during tilting was higher, the tilt-induced reduction in SV was smaller and the increase in TPR was greater (all $P < 0.05$). The correlation coefficient of the relationship between cerebral blood flow velocity and pressure was lower after 500 ml of water (0.43 ± 0.1 compared with 0.73 ± 0.1; $P < 0.05$), indicating better autoregulation. In conclusion, drinking 500 ml of water increased OT and improved cardiovascular and cerebrovascular control during orthostasis. Patients with PRS should be encouraged to drink water before situations likely to precipitate a syncopal attack.

INTRODUCTION

Syncope is a common medical problem, responsible for 0.77 % of hospital admissions in the United States [1] and between 0.9–1.2 % of hospital admissions in Europe [2]. It does not have a uniform cause [3], although it is most commonly due to the ‘vasovagal’ phenomenon [4]. This response is characterized by bradycardia and vasodilatation, which leads to sudden hypotension and loss of consciousness. A vasovagal response can also be triggered in healthy volunteers subjected to a sufficiently severe orthostatic stress [5], and it seems that patients with recurrent vasovagal syncope [or PRS (posturally related syncope)] are merely more susceptible to orthostatic stress [6].

For patients with infrequent syncopal episodes, aggressive treatment is not usually needed, and they often respond well to advice regarding manoeuvres to prevent further episodes [7]. It is also known that a person’s plasma volume is a crucial determinant of their susceptibility to syncope [8] and advice concerning the maintenance of an adequate water intake may also be of benefit to such patients. Two previous studies [9,10] have demonstrated that water drinking itself was sufficient to

Key words: head-up tilting, haemodynamics, orthostatic tolerance, posturally related syncope, water.

Abbreviations: CBFV, cerebral blood flow velocity; DAP, diastolic pressure; HR, heart rate; HUT, head-up tilting; NS, not significant; OT, orthostatic tolerance; PRS, posturally related syncope; SAP, systolic pressure; SV, stroke volume; TPR, total peripheral resistance.

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cause an increase in OT (orthostatic tolerance) in healthy control volunteers. The mechanism(s) by which water has its beneficial effect is unknown. It is known that water evokes a pressor response in patients with autonomic failure [11–13] and improves orthostatic responses in the postural tachycardia syndrome [14]. Its effects, however, in patients suffering from uncomplicated PRS are unknown. The aim of the present study was to examine whether drinking water would improve OT, and orthostatic cardiovascular control, in patients with uncomplicated syncope. We used a combination of HUT (head-up tilting) and lower body suction to determine OT [5] and cardiovascular responses to an orthostatic stress sufficient to induce presyncope in patients suffering from recurrent syncopal episodes. This test has been shown previously [5] to be sensitive, specific and highly reproducible.

METHODS

Subjects

Studies were performed on otherwise healthy patients who were experiencing attacks of syncope of unknown cause and who fulfilled the criteria for entry into the study. These criteria were normal results of cardiological and neurological investigations, including resting and ambulatory ECG for all patients, and various other investigations requested by the referring physician, which included ECGs, ambulatory blood pressure monitoring and electroencephalography. All patients had experienced at least one attack of PRS in the 6 months prior to investigation (range, 1–36; mean, 6.7 ± 3.8 episodes/year). Patients were diagnosed as having PRS and were recruited for the study if their tolerance to an earlier investigation (range, 1–36; mean, 6.7 ± 3.8 episodes/year). Patients were diagnosed as having PRS and were recruited for the study if their tolerance to an earlier clinical orthostatic stress test had been less than that predicted on the basis of data published previously [5] and they experienced symptoms, associated with hypotension, that they recognized as being similar to their own spontaneous attacks. We use the term PRS, rather than vasovagal syncope, since the vagal component of this reflex is variable, and in some patients there was no significant bradycardia. We studied nine patients (mean age, 36.8 ± 4.2 years; four males) none of whom was taking any medication.

Protocol

Written informed consent was obtained before entry to the study. Tests were carried out in the morning, following an overnight fast, in a temperature-controlled laboratory (22–24 °C). The study received approval from the Research Ethics Committee of the United Leeds Teaching Hospitals and was performed in accordance with the Declaration of Helsinki (2002) of the World Medical Association.

In a randomized controlled cross-over design, all subjects underwent the determination of OT, using the ‘Leeds’ protocol [5] on two consecutive days. Subjects were instructed to empty their bladders immediately prior to testing. They were positioned supine on a tilt table with the right arm supported at heart level. Throughout testing we monitored HR (heart rate; by ECG), brachial blood pressure (Hewlett Packard 78325C) and finger blood pressure on the right hand (Portapres® model 2; TNO-TPD Biomedical Instrumentation) continuously. Middle cerebral artery blood flow velocity was measured continuously using a 2 MHz ultrasound probe (Multi-Dop X4 TCD-8.01; DWL Elektronische System), which was fixed in position using a headband, with the angle of insonation held constant throughout. End-tidal CO₂ was recorded continuously using an infra-red analyser (Binos 1; Leybold-Heraeus).

Subjects rested in the supine position for 15 min, during which time baseline readings were taken. They were then tilted up by 30° to facilitate drinking, and drank either 50 ml (control) or 500 ml of non-sparkling mineral water (Northumbrian Spring) at room temperature. Subjects were then returned to supine for an additional 15 min. They then underwent HUT (time zero) to an angle of 60° for 20 min. After this time, while still tilted, lower body negative pressure was applied below the level of the iliac crest at – 20 mmHg and then – 40 mmHg for up to 10 min each. The test was terminated when the SAP (systolic pressure) fell below 80 mmHg and the subject experienced symptoms of presyncope. OT was defined as the time in minutes from the start of HUT until termination of the test. The decision to terminate the test was made by a ‘blinded’ investigator.

Data interpretation

Using the Portapres® Modelflow® technique, we calculated SV (stroke volume), cardiac output and TPR (total peripheral resistance) [15]. These data are presented as percentage changes from the initial supine variables. We also calculated cerebral blood pressure as: arterial pressure – (h/1.36) (where h is the vertical height difference in cm between the brachial cuff and the ultrasound probe). Changes in cerebrovascular resistance were calculated using the Ohmic relationship between pressure and flow. Cerebral autoregulation was assessed using the correlation coefficient describing the relationship between cerebral blood pressure and flow velocity as described previously [16]. A strong positive correlation indicates pressure-dependency of flow and thus poor autoregulation. During direct comparison between the two test conditions, data are only presented for the first 10 min of the tilting period, since after this time (particularly during the control study) several patients had become presyncopal and the test was terminated. After this time, responses would include data from presyncopal subjects with non-presyncopal subjects, which would skew the results. Data, however, are also presented.
Changes in time to presyncope (OT) after drinking 500 ml of water is represented in all cases as time zero from the period immediately before presyncope. HUT was no change in supine HR following drinking water on either test day. The orthostatic stress caused a significant increase in HR on both test days (P < 0.01); however, this increase was significantly smaller after drinking 500 ml of water (Figure 2).

SAP while supine was unaffected by drinking either 50 or 500 ml of water. Following tilting, SAP decreased after the 50 ml drink, but was unchanged after 500 ml of water. SAP, therefore, was significantly (P < 0.05) higher during tilting after 500 ml compared with that after 50 ml of water (Figure 2). DAP was unaffected by drinking 50 ml of water and did not change significantly during tilting. Drinking 500 ml of water did increase supine DAP and it remained higher during the tilting (Figure 2). DAP was significantly higher at 8–10 min of tilting following 500 ml compared with 50 ml of water.

After drinking 50 ml of water, mean arterial pressures were not significantly changed during supine or tilting. However, after drinking 500 ml of water, there was a significant increase in supine mean pressure from 82.4 ± 1.5 to 87.1 ± 1.5 mmHg (P < 0.05). Tilted mean pressure also remained higher than before drinking 500 ml of water (pressure after 10 min of tilting was 87.8 ± 2.2 mmHg; P < 0.01). Mean pressures were significantly higher on the 500 ml test day compared with the 50 ml test, both during supine (87.1 ± 1.5 compared with 82.8 ± 1.9 mmHg respectively; P < 0.05) and following 10 min of tilting (87.8 ± 2.2 compared with 80.4 ± 3.9 mmHg respectively; P < 0.01).

Figure 1  Changes in time to presyncope (OT) after drinking 50 and 500 ml of water
Data show OT during each test for every subject. There was a significant increase in OT after drinking 500 ml of water. Mean OT is represented by the thick solid line, and indicates an improvement after drinking 500 ml of water of 5.6 ± 1.9 min. **P < 0.02 compared with the response to 50 ml of water.

from the period immediately before presyncope. HUT is represented in all cases as time zero.

Statistics
All statistical analyses were performed using GraphPad InStat version 3.00 for Windows 95. Data are represented as means ± S.E.M. Comparisons between the two test conditions were made using paired Student's t tests. ANOVA for repeated measures was used for multiple comparisons within tests. Correlations between variables were examined using the Spearman ranked correlation coefficient. Statistical significance was assumed when P < 0.05. In all of the Figures, asterisks are used to denote differences in responses between the two test days.

RESULTS

OT
The SAP and DAP (diastolic pressure) at which the test was terminated were not different on the two test days (77.4 ± 3.5/47.4 ± 2.9 mmHg and 75.0 ± 4.3/54.4 ± 4.3 mmHg for 50 and 500 ml of water respectively). After drinking 50 ml of water, the mean time to presyncope was 19.8 ± 2.3 min (range, 7–27 min). Drinking 500 ml of water increased the time to presyncope in eight subjects. One subject had a reduction in OT from 27 to 25 min. The mean time to presyncope increased to 25.4 ± 1.5 min (range, 18–34 min; P < 0.02; Figure 1).

Effects on HR and blood pressure
Supine HRs were 68.3 ± 5.5 beats/min before drinking 50 ml of water and 69.3 ± 4.3 beats/min before drinking 500 ml of water ([P = NS (not significant)]. There was no change in supine HR following drinking water on either test day. The orthostatic stress caused a significant increase in HR on both test days (P < 0.01); however, this increase was significantly smaller after drinking 500 ml of water (Figure 2).

SAP while supine was unaffected by drinking either 50 or 500 ml of water. Following tilting, SAP decreased after the 50 ml drink, but was unchanged after 500 ml of water. SAP, therefore, was significantly (P < 0.05) higher during tilting after 500 ml compared with that after 50 ml of water (Figure 2). DAP was unaffected by drinking 50 ml of water and did not change significantly during tilting. Drinking 500 ml of water did increase supine DAP and it remained higher during the tilting (Figure 2). DAP was significantly higher at 8–10 min of tilting following 500 ml compared with 50 ml of water.

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Effects on SV, cardiac output and peripheral vascular resistance
Figure 3 shows changes in SV, expressed as a percentage change from the supine period prior to water drinking on both test days. There was no effect of drinking either quantity of water on supine SV. However, after drinking either quantity there was a significant (P < 0.01) reduction in SV during tilting. This reduction in SV was significantly smaller after drinking 500 ml of water compared with that after 50 ml (P < 0.05).

Responses of TPR, expressed as percentage changes from the supine period prior to water drinking on both test days, are also shown in Figure 3. There was no effect of drinking either quantity of water on supine SV. However, after drinking either quantity there was a significant (P < 0.01) reduction in SV during tilting. This reduction in SV was significantly smaller after drinking 500 ml of water compared with that after 50 ml (P < 0.05).

Cardiac output was not significantly affected in the supine position by drinking either 50 or 500 ml of water. During tilting, cardiac output tended to decrease on both test days, with the initial fall in cardiac output after assumption of the upright posture (minute 2) being –12.4 ± 3.0% and –12.1 ± 4.7% (P = NS) after 50 and 500 ml of water respectively. There was no difference in
cardiac output following the 50 or 500 ml drink at any corresponding time.

**Cerebrovascular responses**

There was no significant change in supine CBFV (cerebral blood flow velocity) after drinking either quantity of water. However, the significant ($P < 0.01$) decrease in velocity immediately following the tilting manoeuvre (minute 2) after the 50 ml drink ($-20.3 \pm 2.6\%$) was not seen after 500 ml of water ($-6.7 \pm 3.6\%; P = NS$). The decrease in mean cerebral arterial pressure with tilting was similar on both days (minute 2 of tilting, $-24.0 \pm 2.4$ and $-22.4 \pm 1.5$ mmHg for the 50 and 500 ml drinks respectively). However, the responses of cerebrovascular resistance at this time tended to be greater after 500 ml
Changes in SV and TPR are expressed as percentage changes from the supine period prior to water drinking. There was no effect of drinking water on supine SV on either day. On both days there was a significant reduction in SV during HUT (P < 0.01; significance not shown on the Figure). This reduction in SV was significantly (P < 0.05) smaller after drinking 500 ml compared with 50 ml of water. There was no effect of drinking 50 ml of water on supine TPR. Drinking 500 ml of water increased supine TPR. HUT increased TPR on both test days (P < 0.05). The response of TPR after water drinking, during both the supine and tilted phases, was significantly greater after drinking 500 ml compared with 50 ml of water. *P < 0.05 compared with the response to 50 ml of water.

We assessed the efficiency of autoregulation from the correlation coefficient (R) between cerebral blood pressure and CBFV during each test for each subject. An example of the derivation of this index in one representative subject is shown in Figure 4(A). Figure 4(B) shows the data obtained for each patient on the two test days. During the 50 ml test, the correlation coefficient was statistically significant in seven out of nine patients. During the 500 ml test, this was significant only in two out of the nine patients. In all but one subject, the correlation coefficient was less after drinking 500 ml than after 50 ml of water. The mean correlation coefficients after drinking 500 and 50 ml of water were 0.427 ± 0.09 and 0.731 ± 0.06 respectively (P < 0.01).

There were no differences in end-tidal CO₂ either during supine or tilting between the two test conditions. End-tidal CO₂ tended to decrease with tilting (P = NS), but this reduction was the same on both test days. End-tidal CO₂ levels during the baseline supine period and after 2 and 10 min of HUT were 4.9 ± 0.2, 4.6 ± 0.1 and 4.6 ± 0.2 % on the 50 ml test day. After drinking 500 ml of water, these values were 4.8 ± 0.2, 4.6 ± 0.1 and 4.5 ± 0.1 % respectively (P = NS for each).

**Effects on the haemodynamic profile preceding presyncope**

Figure 5 shows the blood pressure, HR, TPR and CBFV data on the two test days in the 6 min immediately prior to termination of the test due to presyncope. SAP was
We have demonstrated that ingestion of 500 ml of water in patients with poor tolerance to orthostatic stress results in a significant improvement in their OT. The improvement of approx. 6 min that we observed may seem small; however, this represents almost an entire phase of tilting and lower body suction, and this is a considerable orthostatic challenge. In fact, in two patients, their OT was increased sufficiently to place their test results within the normal predicted range for healthy volunteers [5]. Previous studies evaluating treatments for OT using an identical protocol demonstrated increases in OT of 4 min with exercise training [17], 5 min with water drinking in control volunteers [9], and between 5 and 12 min with salt supplementation (depending on the usual dietary salt intake of the subjects) [18–20]. The improvement in OT seen in the present study cannot be attributed to ‘tilt training’, as we conducted the tests in a randomized cross-over manner. In addition, the open design of the present study is not likely to have contributed to this improvement. Although the patients could not have been blinded as to how much water they were drinking, they were not informed as to the expected responses to the two conditions. Furthermore, all tests were terminated by a blinded investigator, who was unaware of the treatment status. Criteria to terminate the tests were for SAP to decline rapidly to 80 mmHg or less, associated with presyncopal symptoms. Blood pressures at termination of the test under the two conditions were not different. This means that there was no bias in terms of test termination on the two test days. Furthermore, analysis of the cardiovascular parameters immediately prior to termination of the test suggests that the presyncopal events that occurred on the two test days were haemodynamically similar and merely took longer to initiate after drinking 500 ml of water.

It should be noted that in the present study we use the term ‘PRS’ rather than vasovagal syncope, since the vagal component of this reflex is variable and in some patients there was no significant bradycardia. This implies that the hypotension seen is due primarily to vasodilatation and not bradycardia. This may seem at odds with the concept of the so-called Bezold-Jarisch reflex that was, until recently, felt to underlie the syncopal response, whereby a paradoxical stimulation of ventricular mechanoreceptors was thought to trigger bradycardia and vasodilatation. However, we feel that the evidence against this as the mechanism for the onset of syncope is now overwhelming [21], and it is our belief that the trigger for syncope lies within cerebral centres, rather than within the heart [21].

**DISCUSSION**

It is a commonly held belief that people who faint should be given a glass of water. Although we did not investigate the effects of water drinking after a syncopal event, it seems this may be a sensible response since, if patients who are prone to fainting consume water in situations likely to predispose to an attack, they may be able to reduce the likelihood of fainting.

Cardiovascular adjustments following water drinking

Drinking 500 ml of water led to an increase in peripheral vascular resistance and a consequent increase in blood pressure during the orthostatic stress. The finding of
Water drinking in patients with syncope

Increased vascular resistance during orthostasis after water drinking has been noted previously in healthy volunteers [9,22]. However, unlike in volunteers, in patients there was also an increase in both vascular resistance and blood pressure in the supine position after drinking 500 ml of water. This response is more like that seen in patients with autonomic failure [11–14]. This may suggest that there is some subclinical impairment of autonomic control in these individuals. In support of this suggestion, it has been shown previously [23,24] that vascular resistance responses in these individuals are smaller than in asymptomatic controls. Also these syncopal patients have greater fluctuations in blood pressure during postural changes [25]; however, it is important to emphasize that these patients showed no other evidence of autonomic deficiency.

HR responses to the orthostatic stress were blunted by drinking 500 ml of water. This finding has also been seen in healthy volunteers [9]. HR increases during tilting are mainly due to a baroreflex-mediated decrease in parasympathetic tone [26,27]. Following ingestion of 500 ml of water, the change in blood pressure during tilting was smaller, thus providing a smaller change in the stimulus to baroreceptors. The smaller reduction in SV during tilting that was seen after drinking 500 ml of water can be explained by a positive inotropic response.
to increased sympathetic activity, as seen from the higher blood pressure and vascular resistance. This is also likely to affect capacitance vessels, thereby resulting in a smaller reduction in venous return. It might be expected, therefore, that the cardiac output during orthostasis would also be increased after drinking 500 ml of water. However, this was not the case and, in fact, cardiac output did not change. This is due to the larger SV after drinking 500 ml of water being offset by the smaller HR responses to orthostasis.

Cerebrovascular responses after water drinking

In the present study, we have provided evidence that there was an improvement in the control of the cerebral circulation after drinking 500 ml of water. The reduction in CBFV following tilting was less after 500 ml of water and there was less pressure dependency of flow. We have seen a similar effect in healthy volunteers after drinking 500 ml of water [9]. This improvement in cerebral blood flow may contribute to the improvement in OT seen after drinking 500 ml of water, and it has been demonstrated that OT is correlated with the index of autoregulation used in the present study [9,16]. However, it is also possible that the higher pressures and improved cardiovascular stability following drinking 500 ml of water represented less of a challenge to autoregulatory control. We do not consider this explanation to be very likely, since the range of pressure changes that occurred during the orthostatic stress were similar on both test days and merely took longer to evoke after drinking 500 ml of water.

Mechanisms of action of water ingestion

The mechanisms underlying the increased blood pressure and vascular resistance response following the ingestion of 500 ml of water are unknown [28], but they are likely to be related to increased sympathetic activation [12,22,29]. The effect of water drinking is known to be dose-dependent and is not related to the temperature of the ingested water [12]. One possible stimulus for the increased sympathetic activity may be as a direct result of stomach distension. Indeed, gastric distension using a swallowed intragastric bag is known to increase blood pressure and muscle sympathetic nerve activity [30]. Furthermore, comparison of oral water ingestion with an equal volume of intravenous dextrose revealed much smaller pressor responses to the intravenously administered fluids, again suggesting that stomach distension may be important in the mechanisms of action of water drinking [12]. However, the role of a potential hypotensive effect of dextrose in masking any pressor effect from the fluid administration cannot be excluded. It seems that stomach distension is likely to underlie the initial response to water drinking, and yet it should be expected that this effect would diminish over time as the water is absorbed and the stomach distension stimulus fades. Some other, as yet unknown, factor is likely to underlie the more prolonged responses to water ingestion seen in the present study and other studies [9,12,28]. This could be related to the secondary release of systemic or local vasoconstrictor substances (although this has yet to be determined) or the correction of fluid depletion [28]. In our subjects, fluid depletion is unlikely to be a major factor, as none had any disorder likely to affect fluid balance. The pressor response to water ingestion is also unlikely to be related to a direct expansion of the plasma volume after drinking water since, even if one assumes that a water load of 500 ml is absorbed and not excreted, total body water would only change by 1% [29] and, in fact, a substantial amount of the ingested water would be expected to be excreted at the time of the maximal pressor response. Furthermore, fluid expansion would be likely to be associated with decreases in vascular resistance, rather than the increases seen in the present study. Thus it is unlikely that the responses seen in the present study are related to a volume effect.

Preliminary data from a recent study [31] suggest that the osmolarity of the ingested liquid may be important, as water ingestion elicits a greater pressor response than ingestion of the same volume of normal saline. This suggestion is also supported by earlier animal work in which intragastric infusion of saline was found to elicit smaller pressor responses than water in dogs [32].

Limitation of the study

It should be noted that the SV, cardiac output and TPR data were calculated using the Portapres® Modelflow® technique [15]. As with any non-invasive measurement of these variables, responses have to be treated with caution. In the present study, we examined the changes that occurred relative to baseline measurements and this should counteract the inaccuracies in the absolute values, particularly since the group mean baselines were similar on both test days. Moreover, it has recently been demonstrated that the Modelflow® technique is valid to examine group-averaged changes in these variables [33,34]. Our own experience has shown similar changes in cardiac output and SV between the Portapres® Modelflow® data and impedance cardiography (V. E. Claydon and R. Hainsworth, unpublished work).

Cerebral blood flow changes were inferred from changes in cerebral velocity using the Doppler shift technique. This technique is dependent on both the angle of insonation of the vessel and the diameter of the artery remaining constant. The transducer was carefully fixed and clamped in position and, as such, values of flow can be reliably compared within tests. However, it cannot be assumed that the transducer position was identical on the two test days. For this reason, comparisons between tests were performed on changes in CBFV data. However,
it should be noted that the baseline data from which these changes were calculated were similar on both test days. In addition, it has been shown that the diameter of the middle cerebral artery does not change measurably [35,36]. For these reasons, it seems appropriate to assume that cerebral velocity changes are proportional to flow changes and that the change in flow velocity can be reliably compared on the two test days.

**Clinical relevance**

We propose that individuals suffering from recurrent syncopal events be encouraged to drink water in situations likely to predispose to a syncopal attack. This may initially seem to be at odds with the nature of syncopal events, which often occur suddenly and with little warning [21]. However, it is our experience that patients who had not previously noticed the early signs of a syncopal event, such as a feeling of warmth or sweating, can be taught to do so. In addition, careful history taking can usually identify specific triggers for each patient. Common examples are prolonged standing (especially in warm crowded places), episodes occurring within the first hour of waking (e.g. standing in a hot shower in the morning) or episodes occurring after physical exercise. The identification of such triggers can be utilized to encourage water drinking prior to these circumstances as a preventative measure. Although 500 ml of water may seem quite a lot of water to drink in real life, it is our experience that if patients are informed as to the mechanisms underlying syncope and the effects of drinking water, they are willing to drink water prior to specific known triggers. Furthermore, although in the present study we examined the effects of 500 ml of water, we do not know how much water is actually required to be drunk in order to convey the beneficial haemodynamic responses seen. It may be that a smaller volume would also convey some degree of protection against syncope. It would be reasonable to suggest that patients ‘titrate’ the volume drunk to determine the required minimum amount. Finally, although we advocate that patients be encouraged to drink water in order to prevent syncopal events, it should be emphasized that this is not to be undertaken in place of other lifestyle changes, but in addition to them. Patients with syncope should also be advised regarding the use of muscle-tensing manoeuvres [7], increased dietary sodium intake [18,19] and physical training [17] in addition to water drinking, as all of these techniques are known to be beneficial in preventing syncopal events.

**Conclusions**

In the present study, we have shown that drinking 500 ml of water significantly improves OT in patients with recurrent PRS. The mechanisms underlying this response are uncertain, but may be related to improved control of cerebral and peripheral haemodynamics. These responses are unlikely to be mediated purely by volume expansion following water ingestion and are more likely to be related to enhanced sympathetic control of the vasculature. We advocate that water drinking be utilized as an adjunct to other methods of treatment for patients with postural syncope and suggest that these patients be encouraged to drink water, particularly prior to situations likely to precipitate a syncopal event.

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