Sodium Paradoxically Reduces the Gastropressor Response in Patients With Orthostatic Hypotension

Satish R. Raj, Italo Biaggioni, Bonnie K. Black, Aniket Rali, Jens Jordan, Indu Taneja, Paul A. Harris, David Robertson

Abstract—Orthostatic hypotension (OH) can cause syncope that is difficult to treat. We have found that 473 mL (16 oz) of water can increase systolic blood pressure (SBP) by >30 mm Hg in many OH patients (the gastropressor response). OH patients are routinely advised to increase their sodium intake to augment their blood volume. We tested the hypothesis that the ingestion of salt with water would increase the magnitude of the acute pressor response compared with water alone in patients with OH. Patients with OH (n=9; female=5; 65±3 years) underwent a randomized crossover trial of drinking water (H₂O) and salt water (NaCl-H₂O). Noninvasive heart rate and BP were measured with the patient seated for ≥60 minutes after ingestion. The area under the curve for SBP was greater with H₂O than NaCl-H₂O for the 30 minutes (714±388 mm Hg×min versus 364±369 mm Hg×min; P=0.002) and 60 minutes (1454±827 mm Hg×min versus 812±734 mm Hg×min; P=0.048) after ingestion. The increase in SBP with H₂O was greater than with NaCl-H₂O at 30 minutes (37±6 versus 18±5 mm Hg; P=0.006) but not at 60 minutes (17±6 versus 10±6 mm Hg; P=0.4). Norepinephrine increased after H₂O (P=0.018) but not after NaCl-H₂O (P=0.195). Both oral water and salt water increase BP in patients with OH. Instead of augmenting the gastropressor response, the additional salt paradoxically attenuates the pressor response to water. These data suggest a potentially important role for gastrointestinal osmolality in the activation of the sympathetic nervous system leading to cardiovascular reflexes responsible for the gastropressor response. (Hypertension. 2006;48:1-6.)

Key Words: blood pressure ▪ sympathetic nervous system ▪ sodium ▪ hypotension ▪ autonomic nervous system ▪ water-electrolyte balance

Orthostatic hypotension (OH) can be a debilitating condition that is difficult to treat.1-8 Oral water ingestion has been shown to effect a large pressor response in patients with neurogenic OH,4-8 with some patients experiencing increases of >40 mm Hg in their systolic blood pressure (SBP).2 This gastropressor response takes place over minutes rather than seconds, with a peak effect between 20 and 40 minutes after the water ingestion.9,10 The oral route is important, because the increase in SBP was only one-third as great with identical volumes of intravenous 5% dextrose in water (D₅W) versus oral water.10 Activation of the sympathetic nervous system seems to play an important role in the gastropressor response. In healthy individuals, water ingestion has been shown to increase muscle sympathetic nerve activity14 and levels of both plasma dopa15 and norepinephrine,10 which are respectively the key precursor and the primary neurotransmitter in the sympathetic nervous system. In addition, blockade of the autonomic ganglia with trimethaphan has completely abolished the pressor response to oral water, underlining the critical role played by the sympathetic nervous system in this response.10

Despite all that is known about the water response, the afferent signal is still poorly understood. Some have postulated that gastric distension9,11 is the trigger; others have argued that the low osmolality of water is the trigger;16 and still others postulate that the pressor response is because of an increase in blood volume.11

We often advise our patients to augment their blood volume by increasing their salt and water intake. Specifically, we may ask them to undertake a high-salt diet (8 to 10 g of sodium chloride [NaCl], a goal that cannot often be achieved without the use of supplemental NaCl tablets (1 g per tablet). In this study, we sought to compare the hemodynamic effects of the ingestion of 473 mL (16 fl oz) of water (H₂O) compared with a similar amount of water with 2 g of NaCl (NaCl-H₂O) in patients with OH. This dose was chosen as one that would realistically be consumed by our patients. We chose to study only patients with...
OH for 2 reasons: (1) we clinically use these therapies for patients with OH, and (2) physiological alterations because of the addition of salt might be more apparent in these patients because of their large amplitude blood pressure signal in response to water ingestion.

Each of the 3 possible responses for the pressor response to salt water compared with water might offer some insight into the physiology of the gastropressor response. If an augmentation in blood volume were the primary mode underlying the acute pressor response, one would expect that with the addition of salt, the NaCl-H2O would lead to a larger magnitude response. Conversely, if the hypo-osmolality of the fluid were the primaryafferent signal, then the NaCl-H2O would have a smaller response. If gastric distension was the primary afferent signal, then the 2 interventions would produce similar responses. We prospectively tested the hypothesis that the ingestion of salt with water would increase the acute magnitude of the pressor response compared with water alone in patients with OH.

**Methods**

**Subjects**

Patients referred to the Vanderbilt University Autonomic Dysfunction Center with OH between October 2003 and October 2005 were candidates for inclusion in this study. OH was defined as a decrease in blood pressure ≥20/10 mm Hg within the first 5 minutes of standing from a supine position. All of the patients had at least a 6-month history of orthostatic symptoms and were ≥18 years of age. All of the medications that could impair blood pressure regulation were withdrawn for ≥3 half-lives before testing. The Vanderbilt University Institutional Review Board approved this study, and written informed consent was obtained from each subject before initiating the study.

**Study Diet and Baseline Characterization**

Study investigations were performed at the Elliott V. Newman Clinical Research Center at Vanderbilt University. For ≥3 days before testing, subjects consumed a diet containing 150 mEq of sodium per day and 70 mEq of potassium per day. The subject diets were free of caffeine-containing beverages. Heart rate (HR) and blood pressure were assessed after overnight rest in the supine position and again after standing ≥5 minutes (as tolerated) as part of baseline characterization. All but 1 patient underwent autonomic reflex testing: continuous HR and blood pressure monitoring that included controlled breathing fluid ingestion in the fasting and postvoid state. On separate days, patients with OH admitted to our inpatient research unit. The mean age of OH patients was 60.9 years (range, 41-81 years).

**Fluid Trials**

Water and salt water trials were started in the morning with subjects in a fasting and postvoid state. On separate days, patients with OH were asked to rapidly drink (>2 to 3 minutes) 473 mL (16 fl oz) of distilled water (H2O) or 473 mL of distilled water mixed with 2 g of NaCl added (NaCl-H2O; 0.423% NaCl solution) in a randomized, crossover fashion. The patients were seated comfortably in a chair for the duration of the data collection. If their blood pressure fell excessively when initially seated with their legs down, their legs were elevated to re-establish an acceptable blood pressure before the formal study was commenced. The leg positions remained fixed for the duration of the study. Brachial cuff blood pressures and HRs were measured using an automated vital signs monitor (Dinamap Vital Signs Monitor, Critikon Corp) and digitally acquired into a customized database (Microsoft Access, Microsoft Corporation). Seated HRs and blood pressures were measured every 5 minutes for ≥20 minutes before and for 80 minutes after the administration of the study fluid.

Blood samples were drawn for plasma catecholamine levels and serum osmolality from an indwelling antecubital intravenous catheter before fluid ingestion (baseline), 30 minutes after fluid ingestion (30 minutes), and 60 minutes after fluid ingestion (60 minutes). For catecholamine samples, blood was collected in plastic syringes, immediately transferred to chilled heparinized vacuum tubes, and immediately placed on ice. Plasma was separated by centrifugation at −4°C, transferred to collection tubes with 40 μL/mL plasma of 6% glutathione (Sigma Scientific), and stored at −70°C until the assay was performed. Concentrations of norepinephrine and epinephrine were measured by batch alumina extraction followed by high-performance liquid chromatography for separation with electrochemical detection and quantification. Both norepinephrine and epinephrine concentrations are reported in SI units (nanomoles per liter). To convert to the more conventional units of picograms per milliliter, one should multiple the SI value by a conversion factor of 169.18 for norepinephrine and a conversion factor of 183.2 for epinephrine. Serum osmolality samples were collected in plain tubes and the serum analyzed using a freezing point osmometer.

**Statistical Analysis**

Our primary end point was the area under the curve (AUC) for the change in SBP over the first 30 minutes after study fluid administration (AUC30). AUC was chosen as the primary statistical method because it integrates information over time, without resorting to a repeated-measures analysis. The 30-minute time point was chosen because this is when the peak effect was seen in earlier water studies. The null hypothesis was that AUC30 would not be statistically different between the water day and the salt water day. The primary statistical analysis involved a 2-tailed paired t test that compared AUC30 after water versus salt water. Secondary analyses were performed to compare the AUC for change in SBP over the first 60 minutes after study fluid administration and the changes in SBP, diastolic blood pressure (DBP), and HR between the 2 interventions at 30 minutes and 60 minutes after fluid ingestion. The SBP, DBP, and HR were also compared within each intervention (H2O or NaCl-H2O) between baseline values and each of 30 minutes and 60 minutes. The plasma norepinephrine and epinephrine levels were compared from baseline to 30 minutes and from baseline to 60 minutes after fluid ingestion within each intervention. The increase in norepinephrine and epinephrine from baseline to 30 minutes were compared between the 2 groups.

To ensure a stable point of measurement, the baseline blood pressure (and HR) value was calculated as the mean of the last 3 readings before fluid administration. For the comparisons of the point estimates, the SBP, DBP, and HR values assigned to 30 minutes and 60 minutes were the mean of the 3 data points surrounding the time point of interest (eg, the 30-minute data point was the mean of the 25-, 30-, and 35-minute values). All of the values are reported as means and SEMs unless otherwise noted. A data reduction analysis strategy was used in preference over repeated-measures ANOVA because of concerns about overfitting error introduced with multiple repeated measures given our small sample size. Comparisons between the 2 interventions or between 2 time points were made using a paired t test and confirmed with the nonparametric Wilcoxon signed rank test. In all but 1 case (norepinephrine level after water ingestion), the results with the parametric and nonparametric statistical tests were concordant. P values of <0.05 were considered statistically significant, and all of the tests were 2-tailed. Statistical analyses were performed with SPSS for Windows (version 14.0, SPSS). Prism for Windows 4 (version 4.03, GraphPad Software Inc) was used for graphical presentation.

Our required sample size estimate was based on an expected greater increase in SBP in response to salt water ingestion with an effect size of 1.0. A sample size of 10 would have 80% power to detect a difference using a paired t test with a 0.05 two-sided significance level.

**Results**

**Patient Characterization**

The protocol was undertaken by 9 subjects (5 women) with OH admitted to our inpatient research unit. The mean age of
the subjects was 65±3 years, with a height of 167±4 cm, a mass of 67.9±5.1 kg, and a body mass index of 24.1±1.2 kg/m². The orthostatic decrease in SBP ranged from 43 to 105 mm Hg, with a mean decrease in SBP of 69±9 mm Hg. The subjects were able to remain standing for just over 2 minutes (128±41 seconds) before resuming a supine position. All of the subjects tolerated both fluid interventions without nausea, vomiting, or diarrhea.

Autonomic reflex tests were consistent with significant autonomic failure. During the recovery phase of the Valsalva maneuver (phase IV), all of the subjects had a decrease in their SBP compared with baseline, instead of the usual overshoot (increase) of SBP.17 The decrease in SBP ranged from 31 to 96 mm Hg with a mean decrease of 51±8 mm Hg. In response to hyperventilation for 30 seconds, the SBP decreased 37±5 mm Hg (range: 23 to 61 mm Hg). This has been reported in patients with autonomic failure.21 The sinus arrhythmia ratio in response to controlled breathing was blunted in our subjects (1.05±0.01; normal, >1.2).

Hemodynamic Response to Water
The SBP responses to the ingestion of H₂O and H₂O-NaCl are shown in Figure 1. Ingestion of H₂O caused the SBP to increase from 92±8 mm Hg at baseline to 129±9 mm Hg 30 minutes after ingestion (P<0.001). There was a similar increase in the DBP from 57±3 to 75±3 mm Hg (P<0.001). The HR decreased after H₂O from 76±4 bpm at baseline to 70±4 bpm 30 minutes after ingestion (P=0.043). By 60 minutes after ingestion of H₂O, the SBP (110±12 mm Hg) and DBP (66±4 mm Hg) had decreased from the 30-minute time point but were still significantly greater than baseline (P=0.022 and P=0.035, respectively). The HR at 60 minutes after ingestion of H₂O had returned to baseline (75±3 bpm; P=0.976).

Hemodynamic Response to Salt Water
Ingestion of NaCl-H₂O also elicited an increase in the SBP from baseline (94±9 mm Hg) to 30 minutes after ingestion (112±9 mm Hg; P=0.005). There was a similar increase in the DBP with NaCl-H₂O from 58±3 to 67±2 mm Hg (P=0.004). The HR decreased after NaCl-H₂O from baseline (76±4 bpm) to 30 minutes after ingestion (71±3 bpm; P=0.008). By 60 minutes after ingestion of NaCl-H₂O, the increases in SBP (104±9 mm Hg) and DBP (62±2 mm Hg) were no longer significantly greater than baseline (P=0.139 and P=0.210, respectively). Conversely, the HR at 60 minutes after ingestion of NaCl-H₂O was still lower than at baseline (72±4 bpm; P=0.040).

Comparison Between Water and Salt Water
The primary outcome for this study was a comparison of the AUC30 between the H₂O and NaCl-H₂O interventions. The AUC30 in response to H₂O (714±388 mm Hg×min) was double that which was seen with NaCl-H₂O (364±369 mm Hg×min; P=0.002). The AUC for the change in SBP over the first 60 minutes from fluid ingestion was greater with H₂O than NaCl-H₂O (1454±827 mm Hg×min versus 812±734 mm Hg×min; P=0.048). Individual data for AUC30 and AUC for the change in SBP over the first 60 minutes from fluid ingestion are shown in Figure 2.

The change in SBP in the first 30 minutes from baseline in response to H₂O (37±6 mm Hg) was more than double that which was seen with NaCl-H₂O (18±5 mm Hg; P=0.006). Similarly, there was a greater change in DBP in the first 30 minutes from baseline seen in response to the H₂O (18±3 mm Hg) than with the NaCl-H₂O (10±2 mm Hg; P=0.028). There was no difference in the decrease in HR over the first 30 minutes between the H₂O (−5±2 bpm) and the NaCl-H₂O (−5±1 bpm; P=0.924).

Figure 1. SBP response to water and salt water ingestion. The SBPs are shown at baseline and every 5 minutes for 80 minutes after the ingestion of 16 fl oz (473 mL) of distilled water (●) and salt water (○). Error bars, SEM.

Figure 2. The AUC shown in Figure 1 after the ingestion of 16 fl oz (473 mL) of distilled water (●) and salt water (○) are shown for each individual for the first 30 minutes after fluid ingestion (AUC30; top) and the first 60 minutes after fluid ingestion (AUC60; bottom). Individual patient data between the 2 interventions are linked by lines. P values were calculated using a paired t test.
At 60 minutes after ingestion, there was no significant difference between the H2O compared with NaCl-H2O for the change in SBP in the first 60 minutes from baseline (17±6 mm Hg versus 10±6 mm Hg; P=0.378) nor for the change in DBP in the first 60 minutes from baseline (8±3 mm Hg versus 4±3 mm Hg; P=0.439). There was similarly no significant difference between the interventions in the change in HR at 60 minutes (0±2 bpm versus −3±1 bpm; P=0.277).

Cathecolamines in Response to Fluid Ingestion

Plasma catecholamine assays were performed on samples from 7 subjects. In the remaining 2 subjects, inadequate intravenous access precluded the collection of blood for catecholamine assays. The plasma norepinephrine value (NE; Figure 3) increased after the ingestion of H2O from baseline (1.55±0.46 nmol/L; P=0.018) [the Wilcoxon signed rank test was used because of distribution of the data; P=0.087 with paired t test)]. It is noteworthy that this elevated norepinephrine coincided with the time of greatest blood pressure elevation. By 60 minutes after ingestion, the NE was slightly lower but still significantly greater than baseline (1.96±0.57 nmol/L). The plasma epinephrine value (EPI) did not change from baseline (0.20±0.08 nmol/L) in response to H2O at either 30 minutes (0.21±0.08 nmol/L; P=0.685) or 60 minutes (0.21±0.07 nmol/L; P=0.847).

Compared with baseline (1.25±0.35 nmol/L), NE did not increase significantly after the ingestion of NaCl-H2O at either 30 minutes (1.53±0.50 nmol/L; P=0.195) or 60 minutes (1.77±0.64 nmol/L; P=0.178). Similarly, EPI did not increase from the baseline value (0.17±0.06 nmol/L) either 30 minutes (0.16±0.05 nmol/L; P=0.165) or 60 minutes (0.17±0.05 nmol/L; P=0.803) after NaCl-H2O ingestion.

The increase in NE over the first 30 minutes after fluid ingestion was not significantly different between the H2O (0.65±0.32 pg/mL) and the NaCl-H2O (0.28±0.19 nmol/L; P=0.196). The difference was even less pronounced when looking at the increase in NE over the first 60 minutes after fluid ingestion (0.41±0.15 nmol/L versus 0.51±0.34 nmol/L; P=0.769). There were no differences in EPI in response to the 2 types of fluid ingestions at either 30 or 60 minutes (data not shown).

Serum Osmolality in Response to Fluid Ingestion

It is noteworthy that the estimated osmolality of the 16-oz water with 2 g of sodium was ~153 mosmol (milliosmol)/kg H2O, which is less than the osmolality of blood; hence, both H2O and NaCl-H2O, if fully absorbed, would be expected to lower serum osmolality. The baseline serum osmolality was not different before the water and salt water interventions (292±2 mosmol/kg H2O versus 295±2 mosmol/kg H2O; P=0.085). Both the water and salt water interventions decreased the serum osmolality by an equal amount at 30 minutes (3±1 mosmol/kg H2O versus 3±1 mosmol/kg H2O; P=0.795) and 60 minutes (4±1 mosmol/kg H2O versus 4±1 mosmol/kg H2O; P=0.813).

Discussion

Oral water ingestion greatly increases acute blood pressure in patients with autonomic failure and OH,9,10 and we have termed this the gastropressor response. This pressor response is most likely because of a combination of an increase in sympathetic tone and an increase in blood volume. The relative importance of these 2 potential mechanisms for the pressor response is not known, nor is the mechanism of sympathetic nervous system activation. To better understand the underlying mechanism of the pressor response to water, we compared the acute hemodynamic response to the oral ingestion of distilled water with that of salt water (approximately half that of normal saline).

Blood Volume and the Gastropressor Response

Given that salt ingestion has been shown to increase blood volume,22 one would expect that if an increase in blood volume were the mechanism of the gastropressor response, then the pressor response to salt water would be greater than that seen with water alone. We found that both water and salt water resulted in a prominent pressor response that was maximal at 30 minutes after ingestion and decreased by 60 minutes after ingestion. The increase in SBP seen in response to water was twice the increase in SBP that was seen in response to salt water (Figures 1 and 2). These data argue strongly against a “blood volume” explanation for the gastropressor response.

Sympathetic Nervous System Activation and the Gastropressor Response

The plasma norepinephrine level is often used as a biochemical marker of sympathetic nervous system activity. We found that the level of plasma norepinephrine increased significantly after water ingestion (Figure 3) but did not increase...
after salt water ingestion. This is the first report to show this increase in plasma norepinephrine after water ingestion in patients with autonomic nervous system failure, although this has been reported previously in healthy subjects. These data strongly suggest activation of the sympathetic nervous system as the putative mechanism of the pressor response that occurs in response to acute water ingestion. It is noteworthy that the magnitude of this increase in norepinephrine is about the same as is observed in response to drinking 2 to 3 cups of coffee. The increase in plasma norepinephrine could result from either an increase in norepinephrine release because of sympathetic nerve activity or because of a decrease in the clearance of norepinephrine. Without either a norepinephrine spillover study or direct sympathetic nerve recordings, we cannot be certain that the increase in plasma norepinephrine is because of sympathetic activation. The plasma epinephrine did not increase, suggesting that the sympathoadrenal system is not activated.

The blunted pressor response to salt water offers some insights into the mechanisms underlying the putative activation of the sympathetic nervous system induced by water ingestion. Both fluid loads were of similar volume (16 fl oz), so they would both be expected to induce a similar degree of gastric distension. If gastric distension were the trigger for the activation of the sympathetic nervous system, then one would expect that the 2 interventions would result in similar pressor responses. Given the difference in blood pressure responses for water and salt water seen in this study, however, it is unlikely that gastric distension is the afferent signal triggering the sympathetic activation.

Gastropressor Response: The Role of Salt and Osmolality
A major difference between the 2 interventions was the osmolality of the ingested fluids. Distilled water has a distinctly lower osmolality than does salt water. We measured the serum osmolality and found that the changes in serum osmolality after the water or salt water ingestion were similar. It is, thus, unlikely that altered serum osmolality is acting at a central nervous system receptor to trigger the sympathetic nervous system activation.

Serum osmolality might not reflect important differential actions resulting from the different fluid osmolalities. The hypo-osmolar water might induce greater changes in the gastric or portal osmolality than the salt water, and this hypo-osmolar fluid might then act on osmoreceptors in the proximal gut or in the portal circulation. These splanchnic osmoreceptors could then modulate systemic responses similar to those seen in this study.

It is possible that splanchnic osmolality is not a key modulator of the gastropressor response. An alternate possible explanation for the lower blood pressure response to the salt water than plain water ingestion is that the salt itself is acting as the signal for a depressor response. This sodium-induced depressor response might then directly cancel some or all of the pressor response induced by the water. To date, however, neither we nor others have been able to show a pure depressor response to salt.

HR in the Gastropressor Response
The HR decreased in response to both fluid interventions. Although the reasons for the decrease in HR are not entirely clear, this pattern has been noted in previous studies with water. One possibility is that the HR is pushed lower by baroreflex-mediated vagal stimulation in response to the increase in blood pressure. Arguing against this mechanism is the fact that this water-induced decrease in HR has also been seen in young healthy volunteers, even when the blood pressure does not increase. Another possible mechanism of the HR lowering is via gastrointestinal luminal activation of vagal afferents by water ingestion. Wu et al have shown recently that these luminal vagal afferent neurons respond to osmotic stimuli and contain Substance P. We found that the HR recovered more slowly with the salt water intervention, suggesting that there may be a lingering signal from salt ingestion.

Consistency of Findings
Our data are concordant with and build on the recent report of Lipp et al. They studied 10 patients with multiple system atrophy and gave them 500 mL of water and 500 mL of normal saline (0.9%) through a nasogastric tube. After 20 minutes, the water increased the SBP in their patients by 8±9 mm Hg, whereas the normal saline did not alter the SBP (Δ±1±11 mm Hg). They did not find as dramatic a pressor response to the water as in the current study, nor did they find any pressor response to the salt water. The most likely explanation for the differences in the response to water is that their patients may have had a different degree of autonomic failure than did our patients. Another possibility for the larger pressor response in the current study is that deglutition or esophageal receptors, both of which Lipp et al bypass with the use of a nasogastric tube, might contribute to activation of the pressor response to water. Crossover studies with oral ingestions versus nasogastric ingestion in the same patients will be required to tease out these differences. Lipp et al also used a stronger saline concentration than was used in this study. It is possible that if our patients had drunk normal saline (and tolerated it), they might have shown an absent pressor response (demonstrating a dose–response relationship).

Clinical Impact of this Study
We chose to use an intervention that our patients might plausibly use for their clinical care. We often advise patients to consume water (both in bolus form and generally) and follow a high-salt diet (8 to 10 g per day). We have shown previously that a very low–salt diet can decrease the SBP 4 to 5 mm Hg over several days. Thus, despite our current findings, a chronic high-salt diet does likely increase blood pressure. The latter often requires the use of supplemental NaCl tablets. Our patients likely often consume 2 g of NaCl with 16 oz of water. Data from this study would suggest that, if the salt and water were consumed together, much of the beneficial acute pressor response of water in our patients with OH might be lost. Consuming a chronic high-salt diet in this patient population might still be therapeutically useful, but careful attention must be paid to the timing of the water interventions.
Conclusions
We conclude that the oral ingestion of water can induce a large pressor response in patients with chronic OH and that this occurs at least in part as a result of activation of the sympathetic nervous system. Adding sodium to the water, likely via an increase in fluid osmolality, decreases the magnitude of the gastropressor response. This suggests an important role for gut or portal osmoreceptors in the afferent signaling of the pressor response to oral water.

Perspectives
The gastropressor response is a recently described cardiovascular control mechanism that can lead to a marked increase in blood pressure after ingestion of water. This acute response peaks within 20 to 40 minutes and resolves within 60 to 90 minutes. In this report, we show that the pressor response after water ingestion correlates with an increase in plasma norepinephrine, which strongly suggests that the pressor response is mediated by the sympathetic nervous system. The ingestion of salt water, instead of plain water, decreases the magnitude of the pressor response, which suggests that a low gastrointestinal osmolality might be required to trigger the gastropressor response. These data further our understanding of this blood pressure control mechanism. Additional research is required to determine the afferent signaling pathway for the gastropressor response.

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Disclosures
None.

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