Systemic Hemodynamic Changes in Older Hypertensive Patients After Drinking Water or Eating a Meal

To the Editor:

Raj et al recently showed an increase in norepinephrine blood serum levels in patients with autonomic system failure after drinking water. This alteration had been reported previously in healthy subjects. Water ingestion rapidly raises sympathetic response and substantially increases the blood pressure levels, which is an important factor often unrecognized in clinical studies of pressor agents and antihypertensive medications. On the other hand, older hypertensive patients have greater reductions in blood pressure levels after a meal or after oral glucose ingestion than do age matched normotenive people. Postprandial decreases in systolic blood pressure have been found in the elderly patients without overt cardiovascular disease.

We studied the systolic heart function of the 17 elderly hypertensive people after drinking water and eating a meal (10 men and 7 women, mean age: 69 years). All of them had a history of high blood pressure levels for ≥10 years, and they did not have postural hypotension and did not report other comorbidities. Patients with creatinine serum levels <2.5 mg% were included. Heart rate and blood pressure levels were measured by a semiautomated oscillometric device (Dixtal Instruments). The blood pressure values were considered to be the average of 3 sequential readings. Cardiac output (CO) and stroke volume were obtained by a thoracic bioimpedance instrument (NCVO-3, Bomed Instruments). The total peripheral resistance was obtained using the equation (MAP−CVP)/CO×80, where CVP is the central venous pressure and MAP is the mean arterial pressure. Total peripheral resistance was expressed by dyne s/cm².

In the morning, after an overnight fast (8:00 PM to 10:00 AM), the patients arrived at the laboratory, and anthropometric measurements were taken in all of them. After that, they rested for 30 minutes in a sit down position, and during this time the bioimpedance electrodes were attached to their thoraxes. At the end of the resting time, we gave the patients 500 mL of fresh water (2 glasses, 250 mL each) or a meal of 700 kcal, with a total volume of 500 g. The meal had 40% proteins, 30% lipids, and 30% carbohydrates. The average time for drinking or eating was 30% carbohydrates. The average time for drinking or eating was 30 minutes in a sit down position, and during this time the bioimpedance electrodes were attached to their thoraxes. At the end of the resting time, we gave the patients 500 mL of fresh water (2 glasses, 250 mL each) or a meal of 700 kcal, with a total volume of 500 g. The meal had 40% proteins, 30% lipids, and 30% carbohydrates. The average time for drinking or eating was 10 minutes. The patients who were randomly chosen to drink water the first day were invited to eat a meal the following day and vice versa. After ingestion of food or water, at the end of the resting time, we started to register the cardiac output, systolic volume, blood pressure levels, and heart rate. After fasting time, the patients were observed for 60 minutes, and we registered the average hemodynamic values at 5, 15, 30, 45, and 60 minutes. ANOVA and Tukey test were used to locate the differences.

After ingestion of H₂O, the systolic pressure levels increased at 30, 45, and 60 minutes. It increased from 164±2.7 to 175±3.6 mm Hg (the highest significant value was observed at 45 minutes; P<0.05). The DBP did not change significantly, from 90±3.3 to 95±3.9 mm Hg (at 45 and 60 minutes; not significant, P>0.05). The MAP increased at 30, 45, and 60 minutes, and its highest value was observed at 45 minutes (from 112±2.7 to 122±3.6 mm Hg; P<0.05). Neither the HR nor the CO changed after water ingestion. Both retained their fasting-time values of ≈3.5±0.28 L/min. Stroke volume also maintained its fasting-time values, which were ≈62±5.1 mL. Compared with values during the fasting period, total peripheral resistance manifested a significant increase at 45 minutes after water ingestion. It changed from 2847±239 to 3229±298 dyne s−2 (P<0.05).

After food intake, the SBP decreased from 163±2.2 to 156±2.6 mm Hg at 60 minutes (P<0.05). DBP reduced significantly at 15, 30, 45, and 60 minutes. The lowest value was obtained at 15 minutes (from 89±2.0 to 82±2.4 mm Hg; P<0.05). MAP initially reduced at 15 minutes, it maintained these levels until the end of the protocol, and the lowest level happened at 60 minutes (from 114±1.5 to 108±1.4 mm Hg; P<0.05). After food intake, an increase in HR was observed at 5, and this change was maintained throughout the rest of the protocol. The HR increased from 60±2.4 to 66±2.4 ppm (P<0.05). The CO increased after a meal, from 3.6±0.26 to 4.5±0.33 L/min at 45 minutes (P<0.05), and this increase in CO remained constant throughout the remaining research period. The stroke volume changed from 61±5.1 to 69±6.0 mL, PE an beat at 15 and 30 minutes (P<0.05). The TRP reduced from 2799±200 to 2177±179 dyne s−2, at 15 minutes (the lower total peripheral resistance value; P<0.05).

Our study manifested 2 distinct responses. The first 1 occurred after the patients drank water and the second after they ate a meal. After water intake, there were no increases in CO, stroke volume, or HR, but the same did not happen after food intake. These systemic hemodynamic effects in the systolic function of the heart appeared to be in response to digestive processes. The blood pressure levels increased if the patients drank water or decreased if they ate a meal. If the patient drinks and eats at the same time, these effects could theoretically cancel each other out and could, thus, abolish the therapeutic use of water in the treatment of orthostatic hypotension or reduce the vasoconstrictor effect after water ingestion. Data from this study would suggest that the water pressor effect could be eliminated by food ingestion and that the fall in the blood pressor levels after food intake could be reduced by the ingestion of water. Careful attention must be paid to the timing of the water and food administrations to the patients with autonomic failure diseases to prevent additional decreases in their blood pressure levels.

Disclosures

None.

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