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Drinking-Induced Plasma Vasopressin and Norepinephrine Changes in Dehydrated Humans*

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ABSTRACT

After 24-h water deprivation, five men (23–41 yr; 78 ± 3.6 kg) consumed, within 4.0–6.2 min, 12 mL/kg of one of six fluid formulations (16.5 C) once a week over a period of 6 weeks: water, hypotonic saline (0.045% Na^+), isotonic saline (0.36% Na^+), hypertonic glucose (9.7% glucose), and two commercial mildly hypertonic 9.7% carbohydrate drinks. Blood samples were drawn 5 min before and 3, 9, 15, 30, and 70 min after completion of drinking. Ingestion induced no significant change in plasma Na^+, K^+, osmotic, or protein concentrations; blood pressure; or heart rate. Plasma volume (PV) was increased ($P < 0.05$) between 30–70 min with isotonic saline and the two commercial drinks. Ingestion induced a decrease in plasma AVP (PAVP) at 3 min, which was maximal ($P < 0.05$) at 15 min with all drinks. Thus, the act of drinking, independent of the composition or osmolality of the fluid absorbed, leads to a prompt inhibition of PAVP secretion in man. With the exception of rehydration with isotonic saline, this prompt response was followed by a long lasting inhibition of PAVP. There was no change in PRA, plasma aldosterone, atrial natriuretic peptide, or epinephrine, but an increase in plasma norepinephrine occurred immediately after ingestion, which suggests, like that for PAVP depression, a drinking-stimulated neural mechanism. (*J Clin Endocrinol Metab* 81: 2131–2135, 1996)

EVEN THOUGH plasma arginine vasopressin (PAVP) is not required for the stimulation of normal thirst, AVP secretion and thirst are stimulated by similar mechanisms, mainly by an intracellular component linked to cellular osmolality via osmoreceptors and, to a lesser extent, by an extracellular component associated with blood volume (1, 2). Although relative osmotic thresholds for AVP secretion and thirst are still debatable (2), they are similar. Thus, vasopressin secretion almost correlates temporally with the onset of thirst, which is initiated in dehydrated subjects before urine has reached its maximal concentration, thus before renal mechanisms are saturated (2–4).

Because dehydration increases PAVP and aldosterone concentrations, which promote renal water and sodium conservation, respectively, as a first line of defense before or as fluid intake is stimulated, it was probable that termination of drinking would be associated with inhibition of their secretions. Elevated PAVP decreases to normal levels quickly after drinking water in humans (5–11) and animals (12–17), *i.e.* before any postabsorptive change in plasma volume (PV) or osmolality. When rehydration is achieved with isotonic saline or artificial extracellular fluid in dogs (12, 16) or with slightly hypertonic saline in man (10) and hypertonic saline solutions in dogs (12), a rapid, but transient, decrease in PAVP also occurs, suggesting that drinking is followed by a decrease in PAVP regardless of the osmolality of the solution consumed, triggered by reflexes related to the act of drinking *per se*. One recent study in which rehydration in dogs was achieved using a variety of saline and carbohydrate solutions confirmed and extended these findings (12).

The purpose of the present study was to test the hypotheses that as the inhibition of AVP secretion in response to drinking appears to occur as a neural mechanism: 1) it is likely to be independent of the composition or osmolality of the solution ingested, in man as in animals; and 2) it may possibly correlate with changes in other neurally regulated hormones involved in salt/water homeostasis.

Subjects and Methods

Subjects

Five men, 23–41 yr old (mean ± SE, 32 ± 3) and 66–87 kg (mean, 78.0 ± 3.6 kg), volunteered as test subjects. They gave written informed consent and completed a comprehensive medical examination, including history, blood and urine analyses, and a treadmill exercise test. They took no medication, were nonsmokers, and were requested to refrain from consuming alcohol or caffeine for 24 h before testing. This study was approved by Ames Research Center's human research institutional review board.

Procedure

The subjects were instructed to dehydrate for 24 h before testing by restricting all fluid intake, eating dry food, and eliminating food after 2300 h on the evening before their test. They were tested once a week over a period of 6 weeks. Two subjects were tested per day and reported to the laboratory (temperature, 22 ± 0.1 C; relative humidity, 47 ± 6%) at 0730 and 0930 h, respectively, where they urinated and were weighed in shorts (model 5780 digital scale, National Controls, San Carlos, CA). A Teflon catheter (Quick-Cath Baxter Healthcare Corp., Deerfield, IL) was then inserted into an antecubital vein, and the subjects remained seated quietly for the duration of the experiment.

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Rehydration solutions

After a 45-min sitting control period, a blood sample from the dehydrated subject was drawn 5 min before the onset of rehydration at time zero. The subject then drank slowly one of six formulations in a random order. Drink I was water (30 mosmol/kg); drink II was hypotonic saline (0.045% Na^+; 70 mosmol/kg); drink III was isotonic saline (0.36% Na^+; 270 mosmol/kg) in the form of sodium chloride and sodium citrate to ameliorate the unpleasant taste of NaCl and prevent the well known diarrhetic effect of a large volume of isotonic saline orally; drink IV was hypertonic glucose (9.7% glucose and 0.045% Na^+; 650 mosmol/kg). In addition, two commercial solutions were tested: drink V, Performance (Shaklee U.S., San Francisco, CA); and drink VI, Power Surge (Pere Products, Moscow, PA); both were mildly hypertonic (280 and 390 mosmol/kg, respectively), with 9.7% total carbohydrates, mainly in the form of maltodextrins, and some Na^+ citrate. The noncaloric sweetener aspartame and annetto (natural orange coloring) were added so all solutions would appear and taste essentially the same, but the high salt content (157 mEq Na^+) of drink III was evident to the subjects. Drink volume was 12 mL/kg (789–1044 mL), temperature was 16 ± 0.5 C, and mean drinking time was 4.0–6.2 min.

Cardiovascular measurements

Heart rate (Cardiotachometer, model 78203C, Hewlett-Packard, Waltham, MA) and arm blood pressure (Digital sphygmomanometer UA-101, Somatronix, Bristol, CT) were measured at the heart level at −15, −10, and −5 min during the predrinking period, and at 0, 2, 4, 7, and 10 min and every 5 min thereafter during the postdrinking period.

Blood measurements

Blood samples (18 mL) were drawn and placed into chilled heparinized tubes 5 min before and 3, 9, 15, 30, and 70 min after drinking was concluded. For atrial natriuretic peptide (ANP) measurement, blood was drawn into chilled tubes containing 5% ethylenediamine tetraacetate (20 μL/mL blood) (18). Plasma was separated by centrifugation at 4 C, and aliquots for vasoactive hormone measurements were stored at −80 C.

Quadruplicate microhematocrits were spun for 10 min in a centrifuge at 11,500 rpm (model MB, International Equipment Co., Needham Heights, MA) and read on a modified microcapillary tube reader (model CR, International Equipment Co.). Raw hematocrit values were corrected (×0.874) for trapped plasma and whole body hematocrit. Blood hemoglobin was determined with the cyanmethemoglobin method (Coulter Electronics, Hialeah, FL). The percent change in plasma volume (%ΔPV) was calculated with the hematocrit and hemoglobin transformation equation (19). Plasma Na^+ and K^+ concentrations were measured with specific ion electrodes (system E2A, Beckman Instruments, Brea, CA), and osmolality was determined by freezing point depression (model 3DII, Advanced Instruments Digital Osmometer, Needham Heights, MA) in fresh plasma. Proteins were measured with the Bradford colorimetric assay reagent method (Pierce Chemical Co., Rockford, IL).

Vasopressin was extracted with bentonite and eluted with acidified acetone (80% acetone-20% 1 N HCl). The mean recovery of AVP-free plasma was 66 ± 3%. The intra- and interassay coefficients of variance were 7.5% and 14.5%, respectively. Plasma ANP (PANP) was extracted using Sep-Pack cartridges (Waters Associates, Milford, MA). Elution was performed with 60% acetonitrile in ammonium acetate. The intra- and interassay coefficients of variance were 10% and 12%, respectively. Vasopressin and ANP were then measured with sensitive RIAs (18, 20).

PRA and plasma aldosterone were measured by RIA using commercial kits [New England Nuclear Corp. (Boston, MA) and Coat-a-Count kit, Diagnostic Products (Los Angeles, CA), respectively]. Plasma norepinephrine (NE) and epinephrine (E) were measured by high performance liquid chromatography with electrochemical detection (21). The intraassay coefficients of variance were 5% and 3% for NE and E, respectively. The detection limit was 5 pg/mL for both.

Statistical analyses

Data are expressed as the mean ± SE. For each test solution the data were analyzed using the Kruskall-Wallis test for nonparametric distribution. Significant differences between the dehydration control (−5 min) and the postdrinking data as well as between treatments were determined with Wilcoxon's matched pairs, signed rank test. The null hypothesis was rejected when $P < 0.05$, and nonsignificant differences were denoted NS.

Results

After rehydration and regardless of drink composition, there was no significant change in plasma Na^+, K^+, or protein concentrations in the 70-min postdrinking period. Plasma osmolality (P_{osm}), which ranged from 298 ± 2 to 305 ± 1 mosmol/kg just before drinking (−5 min), decreased upon drinking water and hypotonic saline and remained unchanged 70 min after rehydration with the isotonic and hypertonic solutions. P_{osm} decreased by 6 and 5 mosmol/kg 30 and 70 min after drinking water and by 3 mosmol/kg 70 min after drinking hypotonic saline, but due to the small number of subjects, the decrease did not reach statistical significance.

All plasma volumes tended to increase 30 min after drinking. Thereafter, water, hypotonic saline, and hypertonic glucose induced no change (0–1%) in PV, whereas isotonic Na^+ increased PV by 7.6%, Performance increased PV by 4.6%, and Power Surge increased PV by 1.8% ($P < 0.05$) at 70 min, with no significant change in mean corpuscular hemoglobin concentrations. The increase in %ΔPV 70 min after drinking isotonic saline was greater ($P < 0.05$) than that after drinking water, hypotonic saline, and Power Surge.

Heart rate and blood pressures were essentially unchanged and unaffected by drink composition during the 70-min postdrinking period. Blood pressures averaged 120/75 mm Hg, and heart rate was about 70 beats/min.

PAVP decreased with all drinks by 3 min after drinking began; the decrease was significant with hypotonic saline, Performance, and Power Surge. The decrease in PAVP became both maximal and significant for all drinks 15 min after drinking, from dehydration control concentrations of 1.7–3.7 to 0.1–0.7 pg/mL ($P < 0.05$); PAVP fell by 70–85% of dehydration control values (Fig. 1). Plasma AVP remained de-

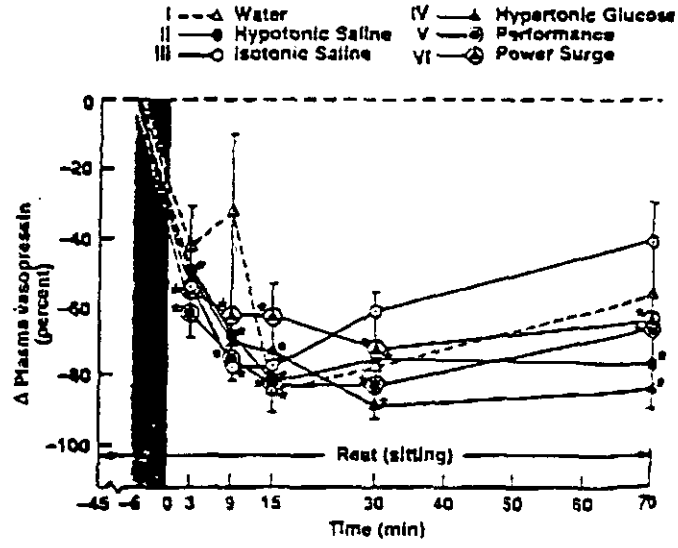

FIG. 1. The %ΔPAVP in dehydrated men 3–70 min after drinking six rehydration solutions. *, $P < 0.05$ vs. the −5 min concentrati

pressed throughout the 70-min experiment. However, at 70 min, with water and isotonic saline, PAVP reached concentrations (0.8 ± 0.3 and 1.0 ± 0.5 pg/mL, respectively) lower than but no longer significantly different from those during dehydration (2.2 ± 1.0 and 1.7 ± 0.5 pg/mL, respectively). The kinetics of the change in PAVP after drinking were similar with all solutions and independent of the control PAVP concentration (2.2 ± 1.0, 2.8 ± 0.3, 1.7 ± 0.5, 2.8 ± 0.9, 3.7 ± 1.1, and 2.2 ± 0.6 pg/mL before drinking solutions I, II, III, IV, V, and VI, respectively) or the solution osmolality. These immediate and long lasting decreases in PAVP occurred with no concomitant significant changes in PRA or in PA (Fig. 2). PANP varied between 27.2–35.2 pg/mL after 24 h of dehydration and showed only decreasing trends 15 and 30 min after drinking water, 9 min after drinking hypotonic saline, and 9 and 15 min after drinking isotonic saline (Fig. 2).

Although PE concentrations were essentially unchanged after drinking (Fig. 2), there were significant increases in PNE concentrations between 3–9 min with all solutions, except water, that lasted for 30 min, from concentrations of 288 ± 85, 269 ± 9, 264 ± 48, 252 ± 40, 247 ± 61, and 189 ± 24 pg/mL before drinking solutions I, II, III, IV, V, and VI. Elevated PNE concentrations with hypertonic glucose and Power Surge continued for 70 min. The average PNE increased by 30% 3 min after the onset of drinking and by 50–70% 9–30 min after drinking hypertonic glucose and the two commercial drinks (Fig. 3).

Analysis of hormonal responses after drinking showed no difference between treatments for PRA, PNE, and PE, but occasional differences at various time points for plasma ANP.

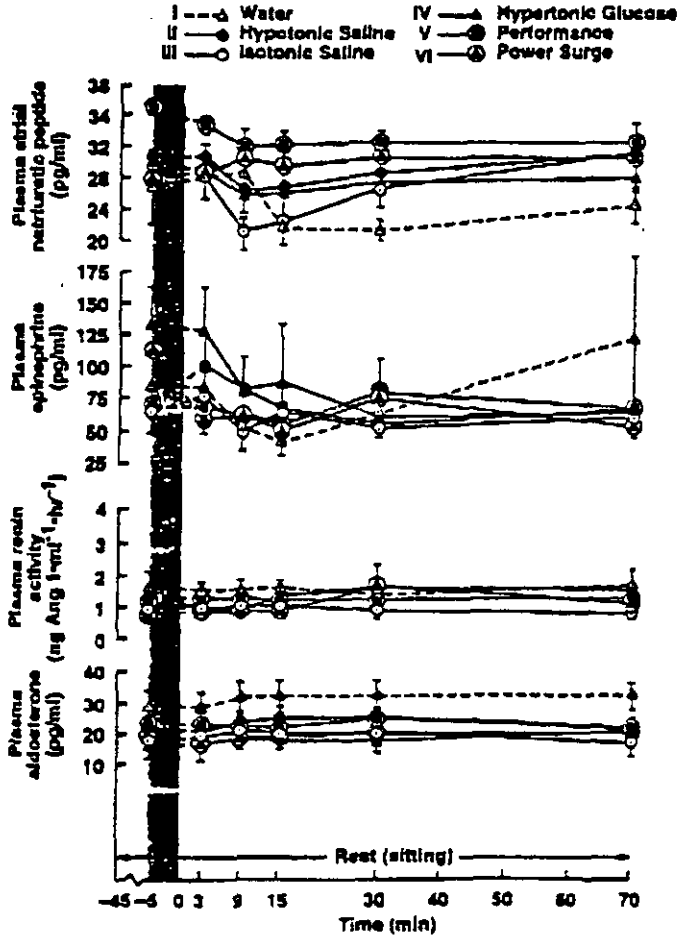

FIG. 2. Mean (±SE) PANP, PE, PRA, and plasma aldosterone in dehydrated men 3–70 min after drinking the six rehydration solutions. *, $P < 0.05$ vs. the −5 min concentration.

Discussion

A first finding of the present study is that in man, drinking solutions with a wide range of tonicity regardless of the composition induces a prompt acute decrease in PAVP that is independent of P_{osm}, as it occurs at a time where such an AVP response is entirely inappropriate with regard to the concomitant P_{osm}.

We and others have suggested that it is the act of drinking *per se* that appears to activate the afferent limb of a reflex causing the immediate inhibition of vasopressin secretion (5, 9, 10, 12, 16, 17). Although drinking water or hypertonic saline decreases PAVP, gargling with water for 2 min without swallowing does not (10), nor does holding a hypertonic saline solution in the mouth for 30 min (9). In addition, oropharyngeal motor activity and swallowing do not induce inhibition of AVP secretion in dogs unless liquid, not solid, is swallowed (17). Thus, the oropharynx appears to have a system that can both discriminate between solid and liquid food and meter the intake, and swallowing plays a major role in the initiation of the reflex. Of note is the recent description of water-sensitive receptors in the pharyngolaryngeal area in man. They appear to initiate a swallowing reflex, the latency of which increases as a function of the concentration of NaCl added to the solution, whereas added carbohydrates have a limited effect (22).

Our data also provide evidence of a long lasting inhibition

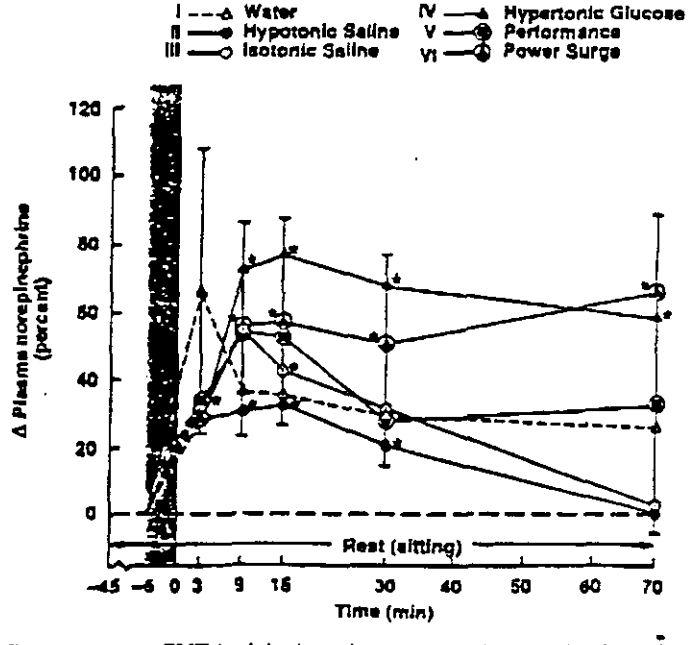

FIG. 3. The % ΔPNE in dehydrated men 3–70 min after drinking the six rehydration solutions. *, $P < 0.05$ vs. the −5 min concentration.

of PAVP after drinking in man, as PAVP was still decreased significantly 30 and 70 min postdrinking with all solutions except water, where it was at the limit of significance, and isotonic saline, where the decrease was present but no longer significant. Seventy minutes after drinking water or hypotonic saline, PV was not significantly increased, but a decrease in P_{osmv}, although nonsignificant, occurred, and this postabsorptive change may have explained the sustained decrease in PAVP. The faster return of PAVP to the predrinking concentration upon drinking isotonic saline despite a 7.6% increase in PV agrees with the observation that the higher the osmolality of the saline solutions drunk by dehydrated dogs, the shorter the time PAVP was suppressed (12). Interestingly, PAVP remained significantly depressed 30 and 70 min after drinking the three hypertonic carbohydrate solutions, a response different from that after drinking hypertonic saline or hypertonic mannitol (12). Absorption of hypertonic saline (12) results in expansion of extracellular fluid volume, which may not be the case with hypertonic mannitol (isotonic mannitol tends to contract PV) (23), but in both cases, there is a rise in P_{osmv}; the latter is a strong stimulus for AVP secretion and can override the suppressive effect on AVP secretion of oropharyngeal receptor stimulation (9). Seventy minutes after drinking in our study, PV had increased modestly, but significantly, with the mildly hypertonic carbohydrate solutions (+4.6% with Performance and +1.8% with Power Surge) and nonsignificantly with hypertonic glucose. Meanwhile, P_{osmv} increased transiently and nonsignificantly at 9 and 15 min of rehydration, suggesting that most of the carbohydrate content in our hypertonic solutions was metabolized during the 70-min period, so that their osmotic effect was progressively diminished and thus no longer a factor of stimulation of AVP. This would explain why the duration of the AVP inhibition was not reduced in our study by increasing the osmolality of the glucose solutions, in contrast to the observations made in dogs drinking mannitol solutions of increasing osmolality (12). On the other hand, it should be noted that infusion of hypertonic glucose has been shown to induce in man a paradoxical small, but significant, decrease in PAVP, despite the increase in P_{osmv} that has been ascribed to a concomitant decrease in plasma Na^+, which did not occur in the present study (24).

The rapid fall in PAVP after drinking was dissociated from the other enzyme-hormonal responses. Chronic water deprivation causes both cellular and extracellular dehydration, and hypovolemia due to the latter increases PRA (16, 17). Because PRA is unchanged by drinking water or isotonic or even slightly hypertonic saline (Refs. 5 and 10 and the present study), correction of cellular and extracellular fluid deficits may not have occurred (16). Indeed, rehydration with water after 24-h deprivation decreases PRA only when an increase in PV occurred after fluid ingestion (7, 25).

Our data corroborate the finding that PANP decreases in dehydrated humans (26). PANP in all dehydration controls averaged 30 pg/mL, which was substantially lower than the concentration of 47.9 ± 3.3 pg/mL found in 26 normal euhydrated sitting subjects (18). In man, PANP increases significantly only when oral rehydration is sufficient to induce significant hypervolemia (6, 7), even in the presence of a reduction in P_{osmv} (25). In the present study none of the PV or osmolality changes was of sufficient magnitude to induce a significant change in PANP during the 70-min postdrinking period. Thus, ANP does not appear to play a role in AVP suppression after drinking.

Few data are available on the influence of hydration status on plasma catecholamines taken as an index of sympathetic activation, even though there appears to be both inhibitory and facilitatory effects of noradrenaline on fluid intake. A possible dual role for central NE in the control of hydromineral fluid intake was recently proposed (27). Our data show a clear dissociation between PE and PNE responses to drinking; although PE was essentially unchanged by rehydration, PNE increased by 3 min after rehydration and remained significantly elevated with hypertonic glucose and Power Surge. The kinetics of the PNE response were not significantly different between solutions even though the increase tended to be of greater magnitude and duration with the three carbohydrate solutions. There has been indication of an increase in PNE in man after rehydration (28). Our observation together with previous findings in rats suggest that insulin-mediated glucose metabolism within neurons in the ventromedial portion of the hypothalamus may be involved in initiating changes in sympathetic activity in response to changes in diet because fasting suppresses the sympathetic nervous system activation, whereas overfeeding with sucrose has a stimulatory effect (29). On the other hand, seeing a drink and/or getting mentally prepared for imminent rehydration may induce a state of general arousal, leading to activation of the sympathetic nervous system, but the unchanged PE concentration would negate this hypothesis. Thus, this early increase in PNE associated with the early decrease in PAVP suggests a neural mechanism activated by oropharyngeal (and perhaps gastric) factors that would trigger both reflex inhibition of vasopressin secretion and reflex activation of the sympathetic system.

In summary, the act of drinking, alone or combined with gastric stimuli and independent of the composition and osmolality of the fluid absorbed, leads to prompt inhibition of vasopressin secretion in man. In addition, with the exception of rehydration with isotonic saline, this first rapid response is followed by long lasting inhibition of plasma AVP. The vasopressin response is dissociated from the other hormonal responses, which are characterized by no change in PRA, plasma aldosterone, ANP, or epinephrine, together with an increase in plasma NE, which immediate occurrence after the onset of drinking may suggest, as for AVP, a drinking-stimulated neural mechanism.

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