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THE EFFECT OF DRINKING ON PLASMA VASOPRESSIN
AND RENIN IN DEHYDRATED HUMAN SUBJECTSG. GEELLEN, L.C. KEIL, S.E. KRAVIK, C.E. WADE*, T.N. THRASHER**, P.R. BARNES***,
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Oropharyngeal mechanisms activated by drinking have been shown to induce a rapid decline in plasma vasopressin which precedes postabsorptive changes in plasma composition in the dehydrated dog. The present study was undertaken to determine what factor(s) inhibit(s) vasopressin secretion after rehydration in water deprived human subjects.

For this purpose, 8 volunteers (5 men and 3 women) underwent 24 hours of water deprivation during which they ate a standardized dry diet plus 4 g of NaCl. A catheter was then inserted in an antecubital vein and the subjects remained seated during the time-course of the experiment. After 15 minutes, three dehydration control blood samples were taken 30, 9, and 3 minutes before rehydration began. At time 0, the subjects drank 10 ml/kg of water (17.5 ± 0.5 °C) in 35 seconds to 4 minutes. Blood was then drawn 3, 6, 9, 12, 15, 30, and 60 minutes after drinking began. A control water replete sample was drawn before the start of the dehydration.

Hematocrit (Hct) and hemoglobin (Hb) were determined on the day of the experiment, together with electrolytes and osmolalities which were measured on freshly separated serum. Plasma was immediately frozen and further analyzed by radioimmunoassay for renin activity (PRA), vasopressin (AVP), and aldosterone.

The data were analyzed using an analysis of variance for repeated measurements and significant differences between the dehydrated control period and various time points after the start of rehydration were determined using a multiple-range test.

The results are depicted in figures 1 and 2. Compared to the water-replete state, 24 hours of dehydration plus salt caused serum sodium and osmolality and plasma AVP to rise significantly ($p < 0.05$, paired t test) from 139.6 ± 0.92 (mean \pm SE) to 143.4 ± 0.66 mEq/l, 291.4 ± 1.1 to 300.5 ± 1.22 mOsm/kg, and 1.70 ± 0.15 to 3.25 ± 0.51 pg/ml respectively. PRA, Hct, and Hb showed no change.

Plasma AVP showed a significant decrease ($p < 0.025$) within 3 minutes after drinking began and was not associated with a detectable decline in either serum sodium or osmolality. Plasma AVP continued to fall throughout the 60 minutes after drinking; however, a significant decline in serum sodium was not observed until 60 minutes after drinking. PRA, Hct, and Hb did not show consistent changes after drinking.

Since plasma AVP showed a significant fall 3 minutes after drinking began and reached water replete levels 15 minutes after drinking in the absence of any detectable decline in serum sodium or osmolality, we conclude that

oropharyngeal factors, alone or combined with gastric distension account for the extremely rapid inhibition of AVP secretion after drinking in the water-deprived human, as has been shown to be the case in dogs. Our findings are also in agreement with the recent demonstration that at the onset of drinking in the dehydrated monkey, there is an abrupt fall in plasma AVP concentration associated with a considerable decrease in the firing rate of the supraoptic neurosecretory neurons.

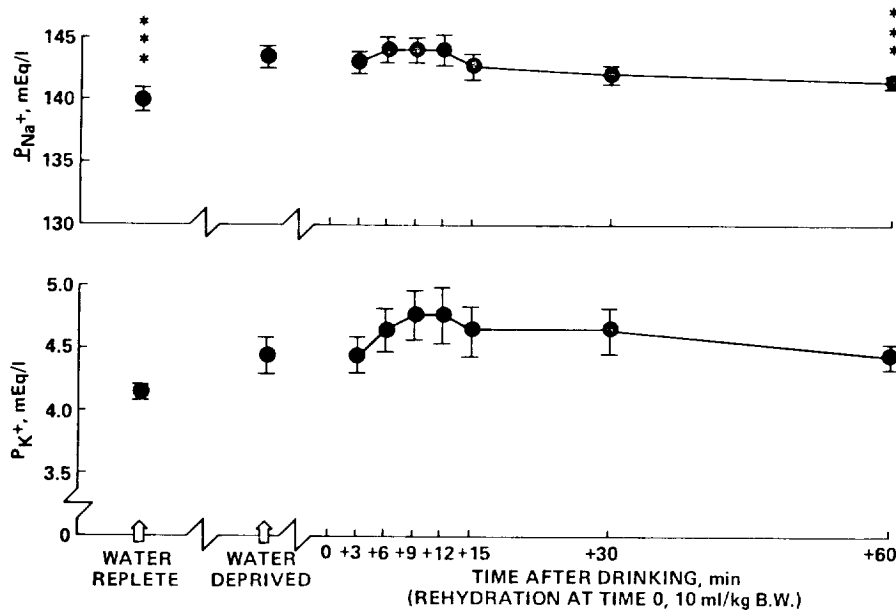


Fig. 1: Serum sodium and potassium after 24 hour water deprivation followed by rehydration with water (10 ml/kg BW) in 8 human subjects. Values are means \pm SE.

- * significantly different from dehydration control period ($p < 0.025$)
- ** significantly different from dehydration control period ($p < 0.01$)
- *** significantly different from dehydration control period ($p < 0.05$)

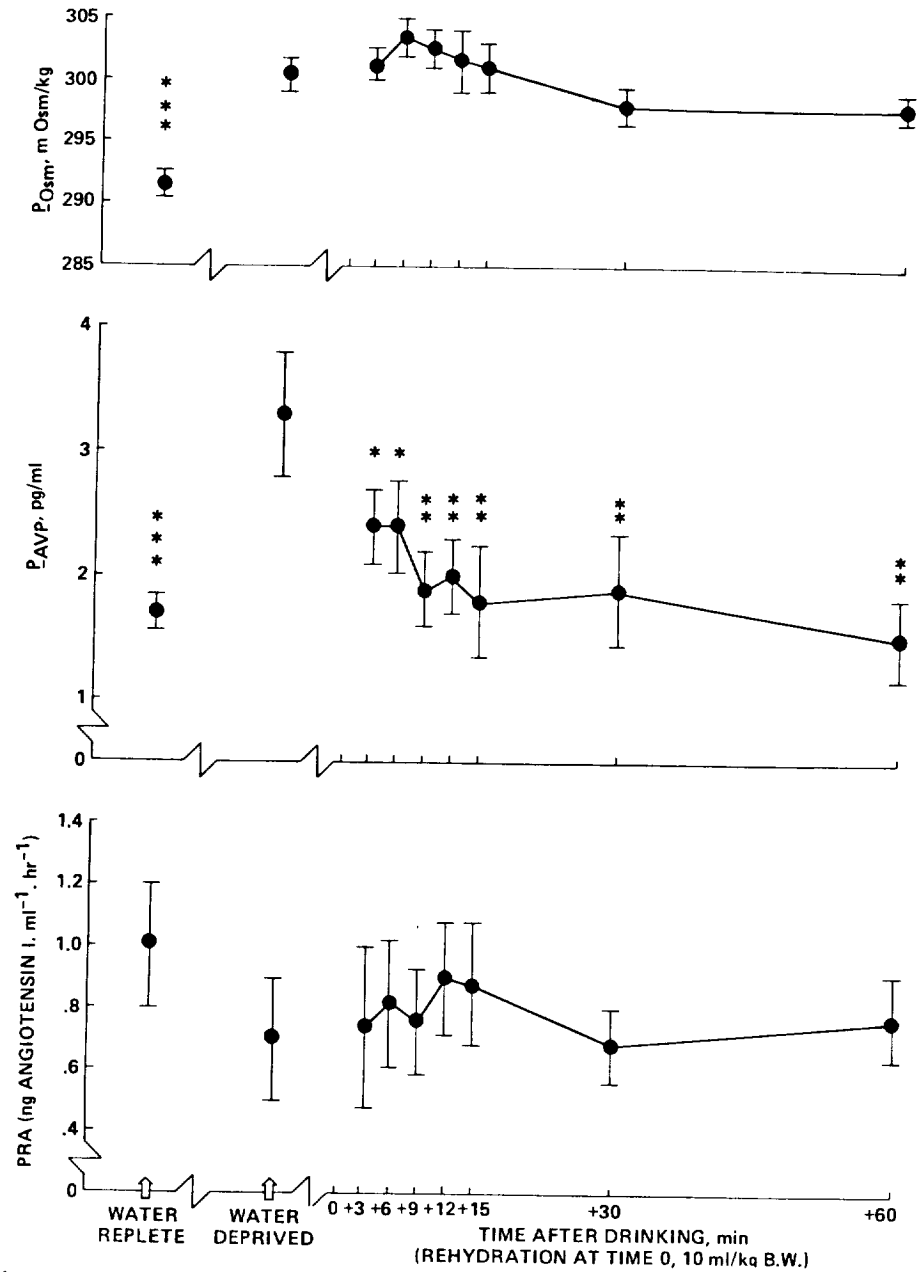


Fig. 2: Serum osmolality, plasma AVP and PRA after 24 hour water deprivation followed by rehydration with water (10 ml/kg BW) in 8 human subjects. Values are means \pm SE.
 * significantly different from dehydration control period ($p < 0.025$)
 ** significantly different from dehydration control period ($p < 0.01$)
 *** significantly different from dehydration control period ($p < 0.05$)