ORIGINAL ARTICLE

The Combined Effects of Exercise and Post Dehydration Water Drinking on Plasma Argenine Vasopressin, Plasma Osmolality and Body Temperature in Healthy Males

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The purpose of this study was to investigate the effect of exercise and post dehydration water drinking on variations in plasma arginine vasopressin (PAVP), plasma osmolality (Posm) and tympanic temperature (Ttym) in healthy males.

Materials and Methods: Eight healthy young males (27.4+/-0.8 yrs old) volunteered for the study. They performed constant work exercise, at a rate of 60 rpm at 50% of individual work load for VO2 peak for 30 minutes. Six blood samples (at minutes 0, 15 and 30 during exercise and at minutes 3, 15 and 30 after termination of exercise) were obtained through an indwelling venous cannula. Plasma concentrations for Na+, and AVP were determined. P_{osm} was calculated using Na+ concentrations. Simultaneous variations in T_{tym} were also determined.

Results: Our results demonstrated a positive correlation between increase in PAVP and both P_{osm} and T_{tym} during exercise but not during the recovery period. When exercise was combined with water drinking a fast and significant suppression in PAVP occurred ($P<0.05$).

Conclusions: These results suggest that P_{osm} and T_{tym} have no significant effect on AVP secretion post exercise, and thus other factors must be involved in AVP secretion during post exercise recovery period.

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Introduction

Exercise is known to influence intravascular water and electrolyte balance in an exertion dependent manner, and to increase plasma osmolality (P_{osm}) and arginine vasopressin level (P_{AVP}).[1] During exercise, water loss through hyper-perspiration and hyperpnea results in an increase in P_{osm}. Alterations in plasma osmolality are sensed by osmoreceptors in the hypothalamus, which in turn initiate mechanisms that affect water intake (via thirst) and water excretion (via AVP secretion) to return plasma osmolality to normal.[2-9] Other factors motivated by exercise modify this response.[1] Increases in P_{osm} and core temperature are two significant factors of this kind.[15,16] On the other hand, post dehydration water drinking is another factor, which manipulates P_{AVP}.[16-18]

It has been shown that the increase in P_{AVP} per unit rise in P_{osm} tends to be higher than that induced by hypertonic saline infusion during exercise suggesting that mechanisms other than osmoregulation are involved.[10,11]

One possible mechanism is increased body temperature.[1] Several studies have suggested that heat exposure elevates plasma AVP concentration and reduces urinary excretion rate,[12-14] suggesting that increased body temperature, per se, can stimulate AVP secretion.[15] Takamata et al[16] reported that the effect of increased body core temperature on AVP secretion was P_{osm} dependent; i. e. , the effect of increased core temperature was greater at a higher P_{osm}. They also demonstrated that in humans the osmotic inhibition of thermal sweating and the associated reduction in AVP secretion were reversed by consumption of a small amount of water before occurrence of any change in plasma osmolality or volume. Similiar results have also been reported earlier by the authors.[17] Thrasher and colleagues have shown that the plasma AVP fall to basal levels within 15 min after water drinking (significant fall after 6 min) in dogs that had been deprived of water for 24 h.[18] Geelen et al demonstrated a rapid fall in P_{AVP} after drinking in dehydrated humans, in the absence of changes in serum Na^+, osmolality, or plasma volume. Therefore, they suggested that oropharyngeal-gastric stimuli mediated by the central nervous system are most likely responsible for the depressed AVP.[19] In further studies, Greenleaf et al showed that the act of drinking in dehydrated humans, alone or combined with gastric stimuli, independent of the composition and osmolality of the fluid consumed, leads to prompt inhibition of AVP secretion.[20]

Despite relatively extensive information regarding the effects of exercise and post dehydration water drinking on P_{AVP}, their combined effect has not been reported so far. In practice, drinking of water at cessation of exercise is common behavior, making the present issue an important research topic. The present study investigates the combined effect of exercise recovery and water drinking on AVP secretion.

Materials and Methods

Subjects

Eight healthy male subjects (27.4±0.8 years old) participated in the study. All volunteers were initially familiarized with the experimental procedures and written informed consent was obtained.

Procedure

Subjects underwent two exercise protocols (Prot A. without water drinking and Prot B. with water drinking) on separated days. Experiments started at 15:00 hours. Pretest instructions included eating a light lunch, refraining from drinking any beverage after 1:00 pm and any exercise on the day of experiment.

During the first session, each subject had his peak oxygen uptake (VO2 peak) measured. This was determined by an incremental work rate protocol on a bicycle ergometer (ergo-metrics 800S Sensormedics, USA) interfaced with a personal computer (V max 29C Sensormedics, USA). It was preprogrammed to begin at a load of 25 w for an initial 2 minute warm up stage, increase to 50 w for the second stage, and increases by 20 w each minute until the subject achieved volitional fatigue. Subjects were requested to maintain a pedal cadence of 60 rpm for the duration of the test. In the second and third sessions, the protocols conducted were as follows:

Protocol A. An indwelling cannula was inserted into a large superficial vein in the forearm initially. Then subjects rested in the sitting position for 30 minutes in the laboratory (28.4±0.4 ambient temperature, 45.5±0.03 relative humidity, and natural convection). 9mL of blood sample was obtained through the indwelling cannula at the end of resting period, after which they performed a constant work rate exercise test (60 rpm at 50% of their determined VO2 peak) for 30 minutes. Second and third blood samples were collected at 15 and 30 minutes respectively after the onset of the exercise. During the recovery

period, blood samples were taken at 3, 15 and 30 minutes. The samples were immediately centrifuged at 3000 g and 4°c for 5 minutes and then plasma concentrations of sodium and AVP were measured.

Tympanic temperature was measured at 0, 15 and 30 minutes during exercise and again every consecutive 3 minutes, for 30 minutes into the recovery.

Protocol B. The same procedure, described above, was followed except that subjects consumed a desired amount of water (425±25 ml) just after exercise cessation.

Measurements

Change in tympanic temperatures was measured using the Ear Thermometer (Braun Pro 3000, Ireland).

Plasma sodium concentration was determined by Eppendorf flame photometr (Efox 5054, instrumentation laboratory). Because sodium and its associated anions account for about 94 percent of the solute in the extracellular compartment, plasma osmolality could be roughly approximated as P_{osm}=2.1× plasma sodium concentration.[21]

P_{AVP} was determined by radioimmunoassay (vasopressin RIA, IBL Hamburg RIA kit).

Statistical analysis

All values are presented as means±SE. The average response of the different physiological variables was compared using ANOVA with repeated measures. In the event of statistical significance ($P < 0.05$), a Tukey's test was used to identify significant differences. Independent-sample t-test was used for differences between Protocols A and B. Results obtained in the two groups were assessed for statistically significant differences by the Pearson correlation test ($P<0.05$).

Results

Inter- and intraassay coefficients of variation were 3.4% and 2.3%, respectively. Changes in plasma osmolality, AVP and tympanic temperature during exercise and subsequent recovery are given below:

Arginine vasopressin: Before the onset of exercise, mean P_{AVP} was 2.26±0.21 pg/mL in Prot. A and 2.50±0.12 pg/mL in Prot B. In both protocols, significant increases in P_{AVP} were observed during exercise (Table 1). In

Table 1. Plasma concentration of arginine vasopressin (P_{AVP}), plasma osmolality (P_{osm}) and tympanic temperature (T_{tym}) before, during and after cessation of exercise (50% of VO2peak) with and without after cessation water drinking (protocols A and B, respectively) in eight subjects. P values for significant differences with control (0 time) are shown under mean values.

	Protocols	Baseline	Exercise		Recovery		
Samples (min)		0	15	30	3	15	30
P_{AVP} (pg/mL)	A	2.26±0.21	2.50±0.15	3.45±0.07 0.00	3.73±0.20 0.001	2.88±0.13	2.33±0.06
	B	2.50±0.12	2.95±0.17	3.62±0.11 0.00	2.40±0.15	1.93±0.14 0.04	2.65±0.08
P_{osm} (mosm/kg H2O)	A	298.7±1.76	307.6±2.0 0.04	311.5±2.4 0.001	290.3±1.8 0.01	295.8±2.4	301.3±2.1
	B	300.3±0.9	311.3±1.6 0.001	309.4±2.0 0.02	290.3±2.9 0.01	297.1±1.1	294.0±2.0
T_{tym} (°C)	A	37. 08±0.05	37.18±0.05	37.36±0.05 0.03	37.38±0.06 0.02	37.25±0.08	37.0±0.08
	B	37. 05±0.08	37. 09±0.07	37.34±0.05 0.01	37.45±0.03 0.00	37. 08±0.04	37. 03±0.03

Prot A, it increased to an even higher level in the first sample taken 3 minutes after exercise (3.73±0.20 pg/mL, P=0.00) and then fell to lower levels recovering to values not significantly different from the baseline. In contrast, in Prot. B, P_{AVP} decreased rapidly during recovery period and became significantly lower than baseline in the second sample taken 15 minutes after the termination of exercise (1.93±0.14 pg/mL, P=0.04). There was also a significant difference in P_{AVP} between the two protocols during the recovery period (P<0.05) (Table 1).

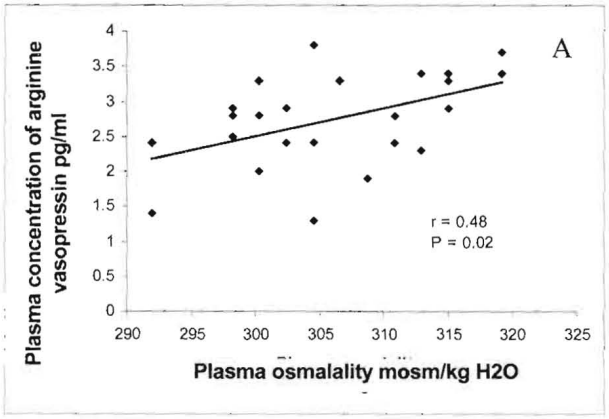
Plasma osmolality: Before the onset of exercise, P_{osm} was 298.7±1.8 mosm/kg H2O in Prot. A and 300.3±1 mosm/kg H2O in Prot. B, which were not significantly different. In both protocols, there were significant increases in P_{osm} during exercise (311.6±2.5 mosm/kg H2O, P=0.00 in Prot. A and 309.5±2.1 mosm/kg H2O, P=0.02 in Prot. B), followed by a significant decrease by exercise termination (290.3±1.9 mosm/kg H2O, P=0.01 in Prot. A and 290.3±2.9 mosm/kg H2O, P=0.01 in Prot. B), and then recovered to baseline values. There was no significant difference in P_{osm} between counterpart values of the two protocols during recovery period. As shown in figure 1A and 1C, there were significant positive correlations between P_{AVP} and P_{osm} (r = 0.48, P=0.02 in Prot A and r = 0.42, P= 0.04 in Prot B) during exercise but not during recovery (Table 1 and Fig.1 and 2), (Figs 2A and 2C).

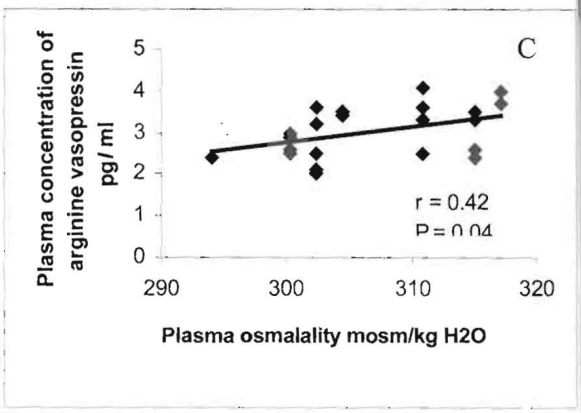
Tympanic temperature: Baseline values for T_{tym} were 37. 08±0.05 °C and 37. 05±0.08 °C for Prot. A and Prot. B, respectively, which were not significantly different. In both protocols, these values increased significantly during exercise, (37.36±0.05 °C, P=0.03 in Prot. A and 37.34±0.05 °C, P=0.01 in Prot. B) and remained elevated up to the first post exercise measurement (37.38±0.06 °C, P=0.02 in Prot. A and 37.45±0.03 °C, P=0.00 in Prot. B), and then recovered to the baseline values. There were significant correlations between P_{AVP} and T_{tym} in both protocols during exercise (r = 0.56, P=0.01 in Prot. A and r=0.35, P= 0.04 in Prot. B) (Fig 1B and 1D); this was not the case during recovery in either of the protocols (Figs 2B and 2D) (Fig.1 and 2 and Table 1).

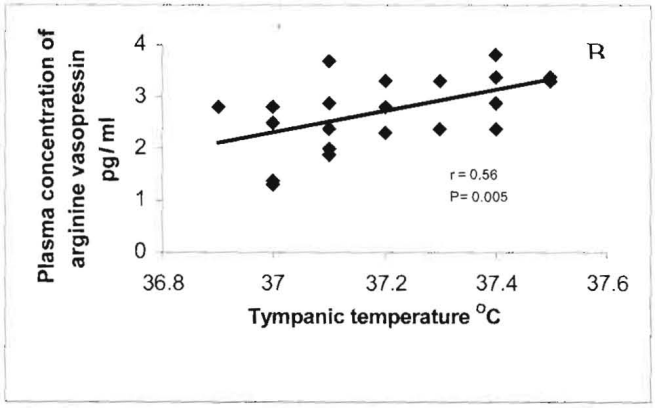
Discussion

Drinking water on termination of exercise is a common behavior. In the present study, we examined the combined effect of exercise and post dehydration water drinking on P_{AVP} variations. Our results demonstrate that in the face of similar changes in P_{osm} and T_{tym}, when exercise is combined with water drinking a fast and significant suppression in P_{AVP} occurs. (Table 1).

Several researchers[1-4] have reported that the primary stimulus for AVP release during exercise is increased P_{osm} resulting from enhanced sweat production and respiratory water loss. Alterations in P_{osm} are sensed by osmoreceptors in the hypothalamus, which initiate the thirst mechanism and AVP secretion, thereby affecting water intake and urinary water excretion.[7-9] Takamata et al[1] have suggested that the main cause of the increased P_{osm} during moderate to heavy exercise of short duration is the hypotonic fluid movement from intra to extra vascular space. This may explain the rapid improvement in P_{osm} seen during the recovery period in the present study (Table 1), which seems to be due to the redistribution of water and retrievement of hypotonic fluid lost into interstitial compartments. More reductions in P_{osm} 30 minutes after exercise in Prot. B can be attributed to absorption of the water consumed by the subjects. In agreement with these reports, results obtained in the present study indicate a positive correlation between P_{AVP} and P_{osm} during exercise (r= 0.48, P=0.02 and r= 0.42, P=0.04 in Prot. A and B, respectively) (Fig.1). During recovery, however, our results failed to show any significant correlation between P_{AVP} and P_{osm} in either of the two protocols (Fig.2).







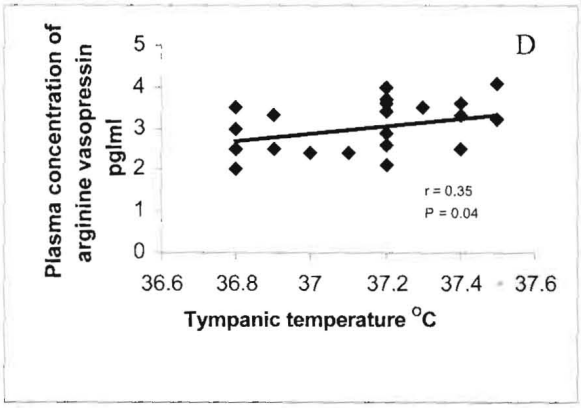
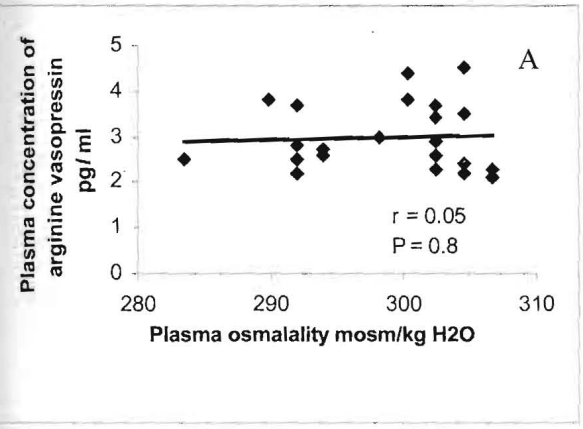
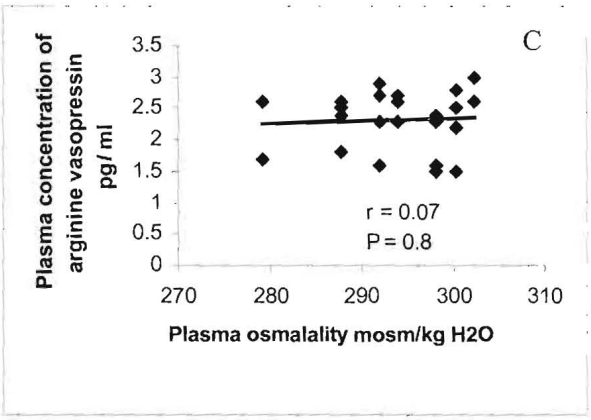


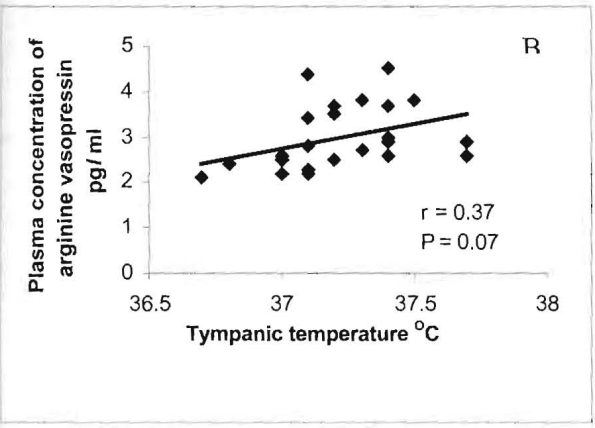
Fig.1. Relations between plasma concentration for arginine vasopressin and plasma osmolality and tympanic temperature during exercise (50% of VO2peak) in Prot. A (panels A and B) and Prot. B (panels C and D).

Another possible mechanism involved in AVP secretion is increased body temperature. Takamata et al[16] demonstrated that AVP secretion is enhanced by increased body core temperature. Our conclusion drawn from the result, obtained in the present study was similar to that of P_{osm}, i. e. ; P_{AVP} and T_{tym} were significantly correlated during exercise only, a correlation that later became non significant.

According to the results of this study, it seems that P_{osm} and T_{tym} which are assumed to be two first line factors involved in AVP secretion, although effective during exercise, do not seem to have a significant role in P_{AVP} changes during its recovery.







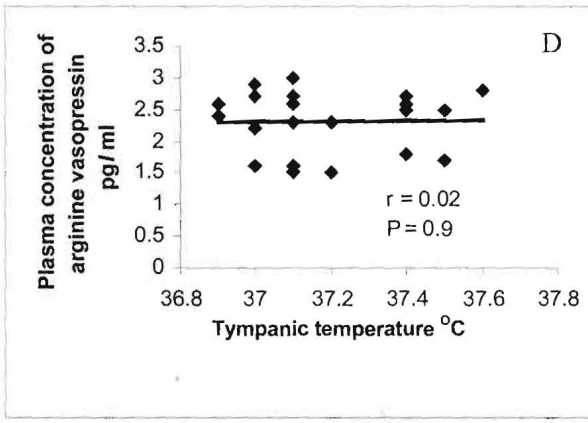


Fig.2. Relations between plasma concentration for arginine vasopressin and plasma osmolality and tympanic temperature during recovery from exercise (50% of VO2peak) in Prot. A (panels A and B) and Prot. B (panels C and D).

The sharp reduction in P_{AVP} in Prot B by initiation of recovery period suggests possible contribution of oropharyngeal[18] and gastric[19] receptors in AVP secretion. These receptors are assumed to be stimulated by water drinking and studies performed on dehydrated human[19] and animals.[22-24] In other words, it seems that the inhibitory effect of water drinking on AVP secretion gains dominance on the stimulatory effects elicited by increase in P_{osm} and T_{tym} seen at the off-transient of exercise. More confirmatory data particularly from animal models are required for the inference made above.

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