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Study of correlation between the volume of consumed water and the drinking – induced sweating, plasma levels of arginine vasopressin, epinephrine and norepinephrine in teakwondo elite athletics

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ABSTRACT

The purpose of this study was to investigate the effect of the volume of consumed water on sweating response and plasma levels of arginine vasopressin, epinephrine and norepinephrine in the first few minutes of water drinking. Materials and Methods: After 4-h water deprivation, six healthy teakwondo champion were exposed to heat and performed mild exercise under an ambient temperature (2 hours, 38-40°C, relative humidity<30%). Subjects were dehydrated by sweating. They were then allowed to drink water with volumes of 1, 3, and 5 ml/kg of body weight using three separate protocols. Sweat rate was measured by amount of sweat collected from the forehead area in grams during 3 minute periods before and after drinking. Blood samples were drawn before heat exposure, before drinking and then every 3 minutes up to the 15th minute after drinking. Results: Dehydration increased mean serum sodium ($p<0.001$). Sweating increased markedly just after the onset of drinking ($p<0.01$) and was greater when consumed water was 5ml/kg of body weight. The more the water volume consumed, the greater was the reduction in plasma arginine vasopressin 3 minutes after drinking. The reverse was true for plasma norepinephrine ($p<0.01$), whereas plasma epinephrine was essentially unchanged by drinking. Conclusion: These data suggest that oropharyngeal sensors that interfere with the activation of sweating response can also manipulate it by consumed water volume. Moreover, the amount of water received affected plasma arginine vasopressin and norepinephrine but not plasma epinephrine which suggests a drinking stimulated neural mechanism.

Keywords: Consumed water volume; Sweat rate; Arginine vasopressin; Epinephrine; Norepinephrine; Oropharyngeal receptors; Teakwondo elite athletic.

INTRODUCTION

When the ambient temperature is above body temperature, then radiation, conduction and convention all transfer heat into the body rather than out. The only mechanisms left to regulate body temperature are the evaporation of sweating from the skin and the evaporative cooling from exhaled moisture. In hot climates, a substantial volume of body water may be lost via sweating to

enable evaporative cooling. One study has shown that dehydrated humans in a warm environment begin to sweat within seconds to minutes after drinking (1). Another study demonstrated that when dehydrated goats were allowed to drink after 60 minutes of heat exposure, sweating began abruptly within 3 minutes of the start of drinking in every animal whether water or saline was drunk (2).

A rapid inhibitory effect of fluid ingestion on thirst and vasopressin (VP) secretion has been documented in studies using water-deprived dogs as experimental subjects (3).

Inhibition of VP secretion occurred within minutes after drinking began, before substantial amounts of the ingested water had been absorbed. These findings suggest that the swallowing of fluid provides an early signal that inhibits VP secretion in dehydrated dogs (4,5). A similar conclusion regarding the control of VP secretion has been drawn from studies of human (6,7) or nonhuman primates (8,9). Investigators also observed an increase in plasma norepinephrine (NE), which occurred immediately after onset of drinking which may suggest, as for arginine vasopressin (AVP), a drinking-stimulated neural mechanism (10).

The effects of volume of consumed water, following dehydration, on drinking induced sweating have not been yet studied. In the present study, we have tried to elucidate the effects of different volumes of consumed water following dehydration on the extent of sweating response and plasma levels of vasopressin, norepinephrine, and epinephrine.

MATERIALS AND METHODS

Subjects

Six healthy male Teakwondo elite athletics 22-26 (23.7 ± 0.6) years old, weight: 80.7 ± 5.7 kg, and height: 181 ± 2 cm participated in this study. They were physically active, routinely participate in sports and endurance exercise training. All volunteers were familiarized with all the experiment procedures and written informed consent was obtained

Procedure

Experiments started at 4 pm. Pretest instructions included eating a light lunch, refraining from drinking any beverage since 12 noon and abstaining from exercise on the day of an experiment. Before each experiment, subjects rested in the sitting position for 30 minutes at a thermoneutral temperature (28°C). After 8 ml of blood were drawn by venipuncture as the first control sample, subjects entered an environmental chamber (38-40°C, <30% relative humidity) and their body weights were measured. Subjects performed a mild physical activity by alternating 10-minutes rest and 20-minutes exercise periods for 60-minutes, and then exercise continued for the last 30-minute period to induce a reduction in total body water through sweating. Air temperature inside the chamber was controlled at 39 ± 1°C and relative humidity was measured at being between 20-28% during the experiment. Total heat exposure time was 120 minutes and subjects were under constant observation for indications of any inability to tolerate the experimental conditions (e.g., elevated heart rate, nausea or confusion).

After the cessation of exercise, subjects dried their body with a towel, were weighed, and then sat on chairs and dried their foreheads. An indwelling cannula was inserted into a large superficial vein in the forearm to collect free-flowing blood samples. Second control blood sample was drawn through the cannula. The first control blood sample compares the plasma concentrations of sodium, arginine vasopressin (PAVP), epinephrine (PE) and, norepinephrine

(PNE) pre and post heat exposure while the second control blood sample was considered as a control to compare before and after drinking.

Sweat rate was measured before drinking for 3 minutes as a control, and then subjects were allowed to drink tap water at the volumes of 1, 3, and 5 ml/kg of body weight using three protocols. Blood samples (8 ml) were drawn through the indwelling cannula at the start of drinking (0 minute) and at 3, 6, 9, 12, and 15 minutes after drinking. Each sample was immediately divided, such that 6 ml were collected in 3 chilled tubes containing dry heparin for determination of PAVP, PE, and PNE, which after centrifugation for 15 minutes at 1,000 g and 4°C, aliquots of plasma were frozen and stored at -70°C until the hormone assays were performed. The reminder of the blood sample (2 ml), which had been transferred to a simple tube, was used to determine the plasma concentration of sodium.

Measurements

Forehead sweat rate was chosen to represent a localized area of sweating and was measured by the weight gain of a covered filter paper disk (96 cm^2) placed on the skin over the forehead. The disks were enclosed in a waterproof tape to prevent evaporation. Each time, the disk was left on the skin for 3 minutes. The weight of a filter paper disk was obtained using EK-500 G beam balance, accurate to ±0.01 g. body weight was measured using a Seca beam balance, accurate to ±100 g. Plasma sodium concentration determined by eppendorf flame photometry (model 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: Posm = 2.1 × Plasma sodium concentration. [PAVP], [PE], and [PNE] values were determined by radioimmunoassay (AVP-RIA Kit, Webster, Texas) and (Norepinephrine/Epinephrine-RIA Kit, KatCombi) from the samples mentioned above.

Statistical analysis

Data were analyzed by SPSS software, using one-way analysis of variance. The Paired-sample T test was used for within-group comparisons between control values and the values obtained after drinking. The variations in data were expressed in terms of the estimated 95% confidence interval of the individual differences relative to the mean of the repeated measurements. Values of $P<0.05$ were considered statistically significant, and all data are presented as means ± SE.

RESULTS AND DISCUSSION

Effects of heat exposure and exercise on plasma osmolality

Heat exposure and the performance of exercise significantly raised Posm in all subjects ($p<0.01$; Fig. 1). After heat exposure, just 3 minutes before the drinking, total mean Posm was increased to 312.2±1.2 mosmol/kg H2O from the baseline level of 304.8±1.0 mosmol/kg H2O. As it is shown in the Table 1, this was also significant for each of the three protocols. All subjects showed similar dehydration with losing 2.37±0.08% (24.3±1.2ml/kg water loss) of their respective preheat exposure body weight. There was significant difference between control and other conditions ($p<0.01$) but no significant difference after drinking at different times.

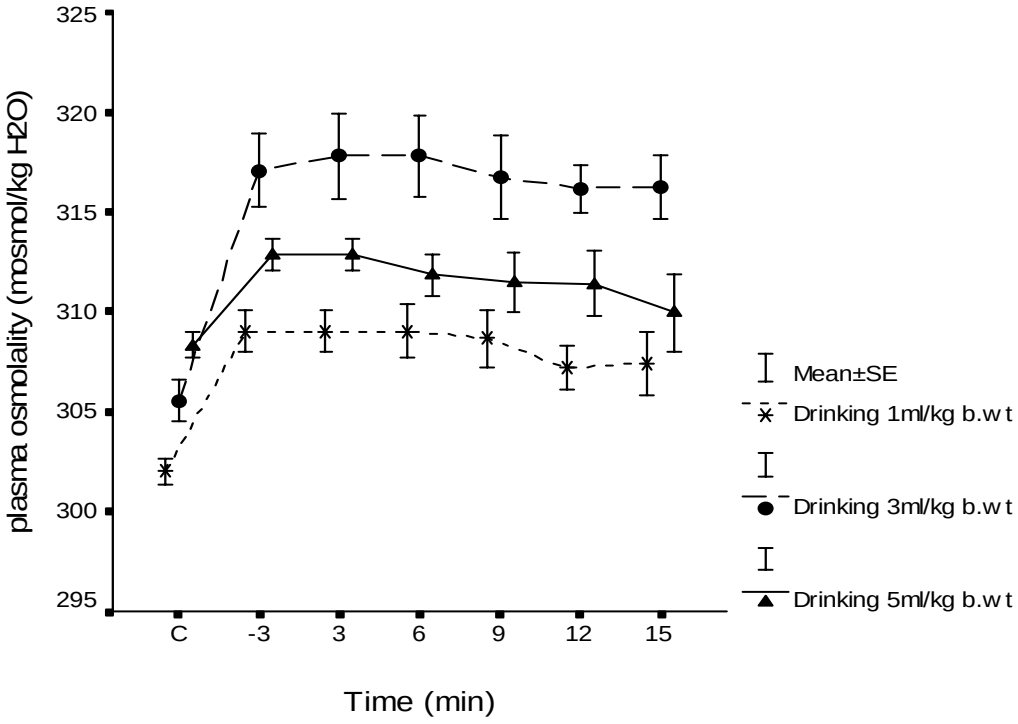

Figure 1 Changes in plasma osmolality pre and post heat exposure and after drinking. Significant differences were observed for dehydrated (-3 minutes) and rehydrated conditions (3, 6, 9, 12, and 15 minutes) with that of control ($p<0.01$). Values are means±SE in six subjects.

Note: In order to avoid overlaps in standard error (SE) bars coincident mean values are shifted a little automatically by the plotting program.

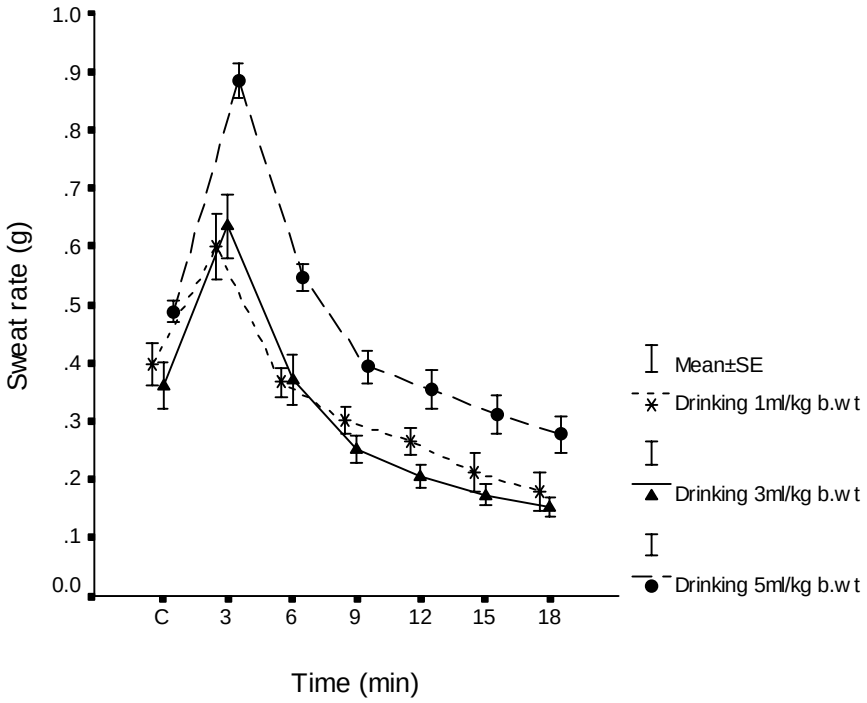

Figure 2 Effect of drinking water volume on sweat rate. Subjects started drinking at 0 min. Significant differences were observed for all protocols just after drinking ($p<0.01$). This difference was greatest in water volume of 5ml/kg of body weight. Values are means ±SE of six subjects.

Effects of drinking water volumes on sweating

Table 1 and Figure 2 show mean sweat rates (Msw) in three water volumes (1, 3, and 5 ml/kg of body weight). The increase in sweating response was evident immediately after drinking started and reached a maximum within 3 min ($p<0.01$). Then it fell gradually became significantly below baseline at final stages ($p<0.001$). The extra sweat just after drinking high volume water (5ml/kg of body weight) was the greatest. Elevated percentage of Msw 3 min after the onset of were 51.6±3.7, 75.5±3.9, and 79.7±3.1% regarding the order of the consumed water volume mentioned above. Comparing Msw within 3 min after drinking in the different consumed water volumes showed a significant difference between 1 and 5 ml/kg of body weight ($p<0.02$) but there was no significant difference between 3 ml/kg of body weight and others.

Table 1. Effects of volumes of consumed water on mean sweat rate

Water volume	Msw before drinking (g)	Msw after drinking (g)					
	-3 min	3 min	6 min	9 min	12 min	15 min	18 min
1 ml/kg b.wt	0.40±0.04	0.60±0.05*	0.37±0.02	0.30±0.02	0.26±0.02	0.21±0.03	0.18±0.03
3 ml/kg b.wt	0.36±0.04	0.63±0.05*	0.37±0.04	0.25±0.02	0.20±0.02	0.17±0.02	0.15±0.02
5 ml/kg b.wt	0.48±0.02	0.88±0.03*	0.55±0.02	0.39±0.03	0.35±0.03	0.31±0.03	0.28±0.03

Values are means±SE.
Msw: Mean sweat rate; b.wt: body weight
* *Significantly different from baseline ($p<0.01$).*

Effects of water drinking on plasma Vasopressin, Epinephrine, and Norepinephrine

The 2-h period of heat exposure and exercise induced significant increase in plasma levels of AVP, epinephrine, and norepinephrine ($p<0.001$). These were 1.0 ± 0.1, 41.6 ± 2.2, and 98.6 ± 6.7 pg/ml, respectively (Figs. 3, 4, and 5). Within 3 min following drinking, plasma AVP faced a significant decrease ($p<0.001$) being greater in 5 ml/kg of body weight consumed water. PAVP continued to fall and reaching to preheat exposure levels by 9 min in three protocols (table 2). Comparing plasma levels of AVP just 3 min after drinking in the different consumed water volumes showed a significant difference between the volume of 1 and 5 ml/kg of body weight ($p<0.03$) but there was no significant difference between 1 and 3 and also between 3 and 5 ml/kg of body weight.

Plasma levels of epinephrine increased significantly after heat exposure ($p<0.001$) and remained approximately unchanged after drinking (Fig.5). There was a significant increase in plasma NE within 3 min after drinking in consumed water volumes of 3 and 5 ml/kg of body weight ($p<0.01$) but this accretion was not significant in volume of 1 ml/kg of body weight. Three minutes after the onset of drinking, PNE increased by 12.1±5.1, 29.6±5.2, and 49.2±3.9% in consumed water volumes of 1, 3, and 5 ml/kg of body weight, respectively. This difference between 1 and 5 ml/kg of body weight was significant ($p<0.02$) but there was no significant difference between 1 and 3 and also between 3 and 5 ml/kg of body weight.

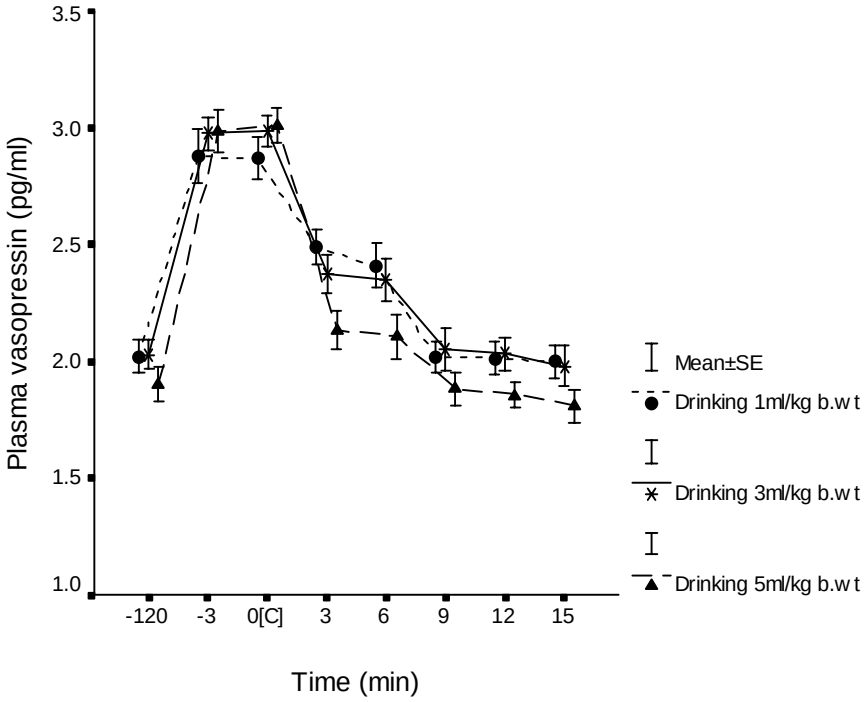

Figure 3 Effect consumed water volume on plasma levels of AVP following dehydration. Subjects started to drink at 0 time which is considered as control. There was a significant increase between before (-120 min) and after (-3 min) heat exposure for all protocols ($p<0.001$). Plasma AVP decreased abruptly at 3 min after drinking in all volumes ($p<0.001$). This reduction was significantly greater in the volume of 5 than 1 ml /kg b.wt ($p<0.03$) but there was not significant difference between 3 ml/kg b.wt and other volumes ($p<0.05$). Values are means ± SE of six subjects.

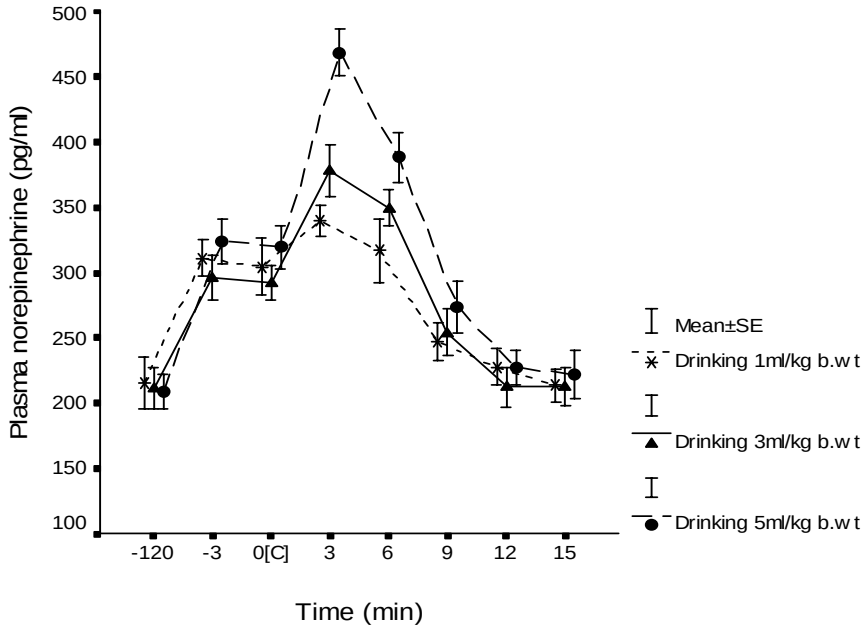

Figure 4 Effect of consumed water volume plasma levels of norepinephrine. Subjects started to drink at 0 time which is considered as control. Plasma NE increased significantly at 3 min after drinking in consumed water volumes of 3 and 5ml/kg b.wt ($p<0.01$) but this increase was not significant in volume of 1ml/kg b.wt. Then it fell gradually and reached to about baseline levels by 15 min. However, the increase was greater in the volume of 5 than 3 ml/kg b.wt. Values are means ± SE of six subjects.

Table 2 Effect of 2-hour heat exposure followed by water drinking with different volumes on plasma osmolality and plasma levels of Arginine vasopressin, Norepinephrine, and epinephrine.

	Pre-heat exposure	Post-heat exposure	Start of drinking	After drinking			
	-120 min	-3 min	0 min	3 min	6 min	9 min	12 min	15 min
Drinking 1ml/kg body weight								
Posm (mosmol /kgH2O)	302±0.6	309 ± 1.0	309.4±0.9	309± 1.0	309±1.4	308.7±1.4	307.4±1.4	306.6±1.5
PAVP (pg/ml)	2.02±0.07	2.88±0.12	2.88±0.08	2.49±0.07†	2.40±0.09	2.02±0.07	2.01±0.07	1.99±0.07
PNE (pg/ml)	215 ± 19	310 ± 13	304 ± 21	339 ± 11	316 ± 24	247 ± 14	227 ± 13	213 ± 13
PE (pg/ml)	39.8±4.0	90.7±1.3	88 ± 3.6	87.7±2.4	83.8±4.9	69.3±5.3	52 ± 5.7	48.2±3.6
Drinking 3ml/kg body weight								
Posm (mosmol /kg H2O)	305.5±1.0	317.1±1.8	317.4±1.6	317.8±2.1	317.8±2.0	316.7±2.1	316.3±1.7	316.2±1.1
PAVP (pg/ml)	2.03±0.03	2.97±0.07	2.98±0.07	2.37±0.08†	2.34±0.09	2.03±0.09	2.04±0.07	1.98±0.08
PNE (pg/ml)	211 ± 15	296 ± 17	292 ±13	378 ± 20‡	349 ±14	253±18	212±15	213 ± 14
PE (pg/ml)	40.3±3.7	73.2 ± 4.0	72.3± 6.2	72.5±5.2	69.2± 4.4	60.3± 4.8	48.8± 3.9	45.2 ± 2.0
Drinking 5ml/kg body weight								
Posm (mosmol /kg H2O)	308.3±0.6	312.9±0.8	313.2±1.4	312.9±0.8	311.8±1.0	311.5±1.5	311.2±1.7	310.5±1.8
PAVP (pg/ml)	1.90±0.07	2.98±0.09	3.01±0.07	2.13± 0.08†	2.10±0.09	1.90±0.07	1.85±0.06	1.80±0.07
PNE (pg/ml)	209 ± 13	324 ± 17	319 ± 16	468 ± 18‡	388 ± 19	273 ± 19	227 ± 13	222 ± 18
PE (pg/ml)	37.2 ± 3.0	78.2±2.7	78.2±1.8	76.8 ± 2.3	72.5± 3.2	68.3± 4.3	54.2±4.8	48.5±2.1

Values are means±SE.
Posm: plasma osmolalityY PAVP: plasma arginine vasopressin; PNE: plasma norepinephrine; PE: plasma epinephrine
Values obtained in -120 minutes are baseline levels for the minute of -3 while 0 minute acts as baseline for the values

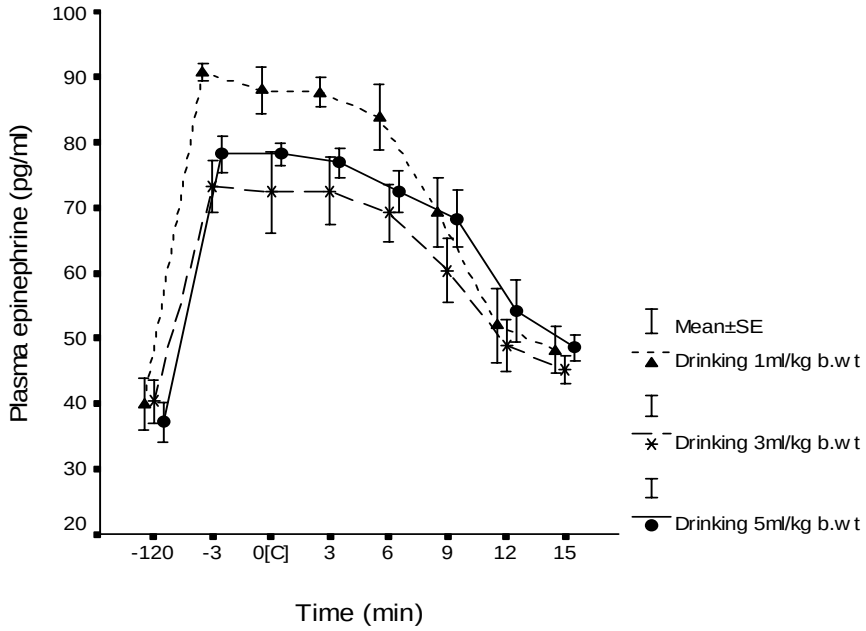

Figure 5 Effect consumed water volume on plasma levels of epinephrine. Subjects started to drink at 0 time which is considered as control. Significant differences were observed between before(-120 min)and after (-3 min)heat exposure for three protocols ($p<0.001$)and remained about unchanged after drinking, then it fell downward baseline levels. Values are means±SE of six subjects.

CONCLUSION

Previous studies have established what is called drinking-induced sweating in dehydrated human (1) and animal (2). In the present study we tested the effect of consumed water volume on local sweating response following heat exposure and mild exercise.

Salata et al (1987) have demonstrated that water temperature of 25°C have no significant effect on PAVP (6). This way, by using water at room temperature we tried to avoid the effect of water temperature on PAVP and investigate the effect of volume per se.

The results indicated that the first 3 minutes after drinking local sweating is aggravated significantly for all three water volumes. This was transient and later on sweating faced a gradual decrease. As for the sweating response was more or less proportional to consumed water volume and became greater when water volume increased. These results elucidate that not only water passing through oropharynx and upper gastrointestinal (GI) itself but also the amount work on the sweating response post dehydration.

Secretion of AVP is in close contact with Posm. Four hours of water deprivation increased Posm and PAVP, which both effects were then intensified by 2 hr of heat exposure. PAVP concentration started to fall significantly within 3 min of water intake in all volumes and reached pre-heat exposure levels the 9th min after drinking (Table 2 and Fig 3). Similar results have been reported by other researchers (3,11,12), but according to our results the immediate changes in PAVP concentration was more prominent in higher volumes ($p<0.03$ when 5 ml/kg was compared with 1 ml/kg of body weight). At the same time plasma osmolality was almost unchanged. This response which is also reported by others (5,13), can be attributed to the delay in water absorption coincident with the continuation of perspiration.

PE and PNE were increased by dehydration but a clear dissociation in the relevant profiles of changes occurred following drinking. PE was initially almost unchanged and then faced a gradual decrease, while PNE had an abrupt increase in the first 3 min prompted by a sharp decrease following. Similar results were also reported by Ghislaine et al (1996) (13). Changes in consumed water volume affected PNE changes proportionally, while no clear effect was evident in the case of PE.

The increase in PNE, which occurs immediately after drinking may suggest, as for AVP, a drinking stimulated neural mechanism. However, this increase may be related to stomach distension. A reflex increase in sympathetic tone in response to stomach distension has been shown repeatedly in controlled laboratory animal experiments (14-19).

In summary, we have shown that changing the volume of water alters the sweating response to drinking in dehydrated hyperthermic man. Drinking water volume had a positive effect on sweating response i.e the more consumed water volume, the more was sweating response.

PAVP, PNE and PE which increase by dehydration respond differently to drinking; PAVP is decreased, PNE firstly is increased then decreased while PE shows no significant change by 6 min and then starts to decrease.

Results also demonstrated that drinking water with high volume cause to decrease more in PAVP and a greater increase in PNE but PE was not affected.

It is concluded that the oropharynx and upper GI appears to have a receptor system that can discriminate between the volumes of consumed water. This factor in turn takes part in the integrated response of sweating following drinking.

In order to determine the location of receptor which triggers a neuronal reflex, leading to alterations in sweating and relevant hormonal secretion, we suggest to use nasoesophageal and nasogastric tube occurs to introduce water selectively and study these changes in dehydrated man. The central locations and neurotransmitter involved in this reflex can be more clarified by devising invasive animal studies that involve dialysis of interstitial fluid in different nuclei of brain. If it is tolerable by subjects, the effect of water with different osmolalities on sweating response is worth study.

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