Effect of Physical Exercise on Renal Response to Head-Out Water Immersion

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Abstract. Head-out water immersion (HOI) induces various renal functional changes, such as diuresis, natriuresis, and kaliuresis. The present study was undertaken 1) to characterize the renal response to HOI in Koreans who routinely ingest high salt diet and 2) to evaluate the impact of exercise on the renal response to HOI. Six healthy male subjects (average Na^+ intake of $232 \text{ mEq} \cdot \text{day}^{-1}$) were immersed up to the neck in 34.5°C water and rested in a seated position or exercised on a bicycle ergometer for 3 hours. In resting subjects, we observed a reversible increase in urine flow and a decrease in urine osmolality, with no changes in creatinine clearance. The peak urine flow observed during the second hour of immersion was 4-fold greater than the pre-immersion level. The excretion of total osmotic substances rose progressively during the 3-hour immersion, which was accompanied by a similar change in Na^+ excretion. The K^+ excretion was slightly elevated. The major component of the immersion diuresis was a water diuresis in the early phase and an osmotic diuresis in the late phase of immersion. In exercising subjects, the diuretic and natriuretic responses to HOI were attenuated and the kaliuretic response was potentiated. Blood hemoglobin concentration and plasma levels of renin, ADH, and aldosterone decreased during immersion-rest, but they remained unchanged or increased during immersion-exercise. These results suggest that 1) the cardiac mechanoreceptor-mediated renal responses to HOI are not changed by chronic high salt diet, and 2) excessive urinary sodium and water losses are prevented by exercise during immersion.

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Introduction

Head-out water immersion (HOI) induces various renal functional changes, such as diuresis, natriuresis and kaliuresis (Epstein, 1978). These changes have been attributed to a redistribution of circulating blood volume. During HOI, hydrostatic compression of lower body elevates plasma volume and suppresses peripheral blood pooling (Krasney et al., 1989) and negative pressure breathing of approximately 20 cm H_2O (Agostoni et al., 1966; Craig and Dvorak, 1975) facilitates venous return, resulting in a central hypervolemia of approximately 700 ml in a normal adult (Arborelius et al., 1972). Such a cephalad shift in blood volume leads to activation of cardiac mechanoreceptors which bring about reflex hormonal and neural adjustments, including a suppression of antidiuretic hormone (ADH) (Epstein et al., 1975a, 1981b; Norsk and Epstein, 1988) and aldosterone (Epstein and Saruta, 1971; Epstein et al., 1975b) secretion, an inhibition of renal sympathetic nervous activity (Krishna et al., 1983; Miki et al., 1988b), and a stimulation of renal prostaglandin (Epstein et al., 1979; Lifschitz et al., 1985) and atrial natriuretic peptide (Epstein et al., 1987, 1989) release, ensuing a diuresis, natriuresis and kaliuresis.

The patterns of renal functional changes vary widely according to the state of hydration (Behn et al., 1969; Epstein et al., 1972, 1973, 1975a, Epstein, 1978) and salt intake (Epstein and Saruta, 1971; Epstein et al., 1972; Epstein, 1978). Furthermore, a study by Böning et al. (1988) suggested that they are also modified by physical activity. It was observed in the above study that the urine flow and urinary Na^+ and K^+ excretions did not undergo significant variations during one hour submaximal swimming exercise (about 70% $\dot{V}\text{O}_2\text{max}$ based on heart rate with assumed maximal heart rate $190 \text{ beats} \cdot \text{min}^{-1}$).

The present study was, therefore, undertaken 1) to characterize the renal response to HOI in Koreans who

routinely ingest high salt diet (Kim 1974), and 2) to systematically evaluate the impact of prolonged physical exercise on the renal response to HOI.

Methods

Six healthy male students (age 23.3 ± 1.8 [SE] yrs, height 172.3 ± 1.3 cm, weight 68.9 ± 2.8 kg, body surface area 1.80 ± 0.04 m²), who had no history of cardiorenal diseases, were recruited as subjects after informed consent. All of them were born and raised in South Korea. None of them were in rigorous physical training. At the time of study, all subjects were ingesting a regular Korean home diet. The 24-hour urinary Na⁺ excretion, determined for 3 consecutive days, was on the average $232 (\pm 51)$ mEq · day⁻¹, indicating that they were on a high salt diet.

The subject who has eaten a light breakfast 2-3 hours earlier reported to the laboratory at 10:00 a.m. on the day of experiment. They were instructed not to drink after breakfast. The subject voided and rested for 1 hour, sitting on a chair (pre-immersion period). The subject was then clothed in a swimming trunk, immersed up to the neck in a constant temperature ($34.5 \pm 0.5^\circ\text{C}$) water bath, and rested in a seated position or exercised at a constant intensity using a bicycle ergometer submersed in water bath for 3 hours. The exercise intensity was adjusted to be light (Ex 1), moderate (Ex 2) or heavy (Ex 3) by modifying the frequency of pedaling (see Results for the metabolic rates for each level of exercise). Upon completion of immersion, the subject climbed out of the tank, dressed and rested on a chair for another 1 hour (post-immersion period). The same time schedule was applied to the time control studies in which the subject sat (Air-Rest) or exercised (Air-Ex) outside the water bath. The subject was not previously dehydrated and was not given any fluid during the 5-hour experimental period.

Hourly urine samples were collected during 1 hour before immersion, 3-hour immersion and 1 hour following immersion. At the end of each hour, the subject was raised to the level above the water for urination. Venous blood samples were collected in the middle of pre-immersion, immersion and post-immersion periods. The oxygen consumption (open circuit spirometry) was determined for 10 min at 30-min intervals throughout the experiment. Urine and plasma samples were analyzed for creatinine (Wako Technical Bulletin, No. 271-10509, Wako Pure Chemical Ind., Osaka, Japan), osmolality (Advanced Osmometer 3D2), and Na⁺/K⁺ (Radiometer Flame Photometer FLM3). In some experiments hematocrit (microcentrifugation), hemoglobin concentration (cyanomethemoglobin method, Sigma Chemical, St Louis, MO), and plasma levels of renin (Lee et al., 1995), aldosterone (Radioimmunoassay kit, Diagnostic Inc.) and ADH (Lee et al., 1995) were also determined.

All results were presented as the mean $\pm$ SE and statistical evaluation of the data was done using the Student's t-test (paired comparison).

Results

Renal function during immersion-rest

Figure 1 illustrates average time courses of urine flow rate (V), urine osmolality (Uosm) and creatinine clearance (Ccr) of 6 subjects quietly immersed in water at thermoneutral temperature (34.5°C) with the heads out. The V increased markedly during the first 2 hours of immersion, and then declined. The peak urine flow observed during the second hour of immersion was on the average $6.4 (\pm 0.7)$ ml · min⁻¹, which was approximately 4-fold greater than the pre-immersion value $1.5 (\pm 0.2)$. Upon completion of immersion, the V promptly reverted to the pre-immersion level. By contrast, the Uosm decreased markedly from $808 (\pm 77)$ mOsm · kg H₂O⁻¹ before immersion to $249 (\pm 17)$ at the second hour of immersion, and it quickly reversed during the post-immersion period. The Ccr was maintained constant at about 140 ml · min⁻¹ during the entire course of experiment, indicating that the glomerular filtration rate (GFR) remained unchanged.

Fig. 1 Changes in urine flow rate (V), urine osmolality (Uosm) and creatinine clearance (Ccr) during head-out water immersion at rest. In this and other figures in the paper, each point and vertical bar represent the mean and ± 1 SE of 6 subjects. Solid symbols represent values significantly different ($p < 0.05$) from the respective pre-immersion value.

Figure 2 depicts changes in free water clearance (CH_2O). The average CH_2O changed from $-2.5 (\pm 0.4) \text{ ml} \cdot \text{min}^{-1}$ to $-0.3 (\pm 1.0)$ during the first 1-hour immersion, thus the free water reabsorption was reduced by 90%. During the second hour of immersion the CH_2O became a positive value ($1.2 \pm 0.4 \text{ ml} \cdot \text{min}^{-1}$), indicating that there was a net free water excretion. During the third hour of immersion the CH_2O returned to a negative value ($-1.6 \pm 0.3 \text{ ml} \cdot \text{min}^{-1}$), but the amount of free water reabsorption was considerably lower (by 36%) than the pre-immersion level. These data indicate that the immersion-induced polyuria was associated with a reduction in free water reabsorption.

Figure 3 presents changes in solute excretion. The excretion of the total osmotic substances ($U_{\text{osm}} \cdot V$) increased progressively during the 3-hour immersion from $1187 (\pm 126) \mu\text{Osm} \cdot \text{min}^{-1}$ to $1645 (\pm 98)$, and it promptly declined during the post-immersion period. This change in osmolar excretion was accompanied by a similar change in sodium excretion ($U_{\text{Na}} \cdot V$). The value of $U_{\text{Na}} \cdot V$ during the third hour of immersion $521 (\pm 26) \mu\text{Eq} \cdot \text{min}^{-1}$ was about 56% higher than the pre-immersion level $334 (\pm 45)$. The potassium excretion ($U_{\text{K}} \cdot V$) increased slightly, but the magnitude of increase was relatively insignificant. These data indicate that the immersion-induced polyuria involved an osmotic diuresis as well, which was mainly associated with a natriuresis.

Figure 4 compares net changes in urine flow (ΔV) and osmolar clearance (ΔCosm) during immersion. The magnitude of ΔCosm was approximately 18% of that of ΔV during the first hour, 24% during the second hour,

Fig. 2 Changes in free water clearance during head-out water immersion at rest. Solid symbols represent values significantly different ($p < 0.05$) from the pre-immersion level.

Fig. 3 Changes in urinary excretions of osmotic substances ($U_{\text{osm}} \cdot V$), sodium ($U_{\text{Na}} \cdot V$) and potassium ($U_{\text{K}} \cdot V$) during head-out water immersion at rest. Solid symbols represent values significantly different at 5% level ($p < 0.05$) and half solid symbols at 10% level ($p < 0.10$) from the corresponding control value.

Fig. 4 Comparison of the increases in urine flow rate (ΔV) and osmolar clearance (ΔCosm) during head-out water immersion at rest. Data are based on Figs. 1 and 3. Solid symbols represent significant changes ($p < 0.05$).

and 60% during the third hour of immersion. Thus, the major component of immersion diuresis appeared to be a water diuresis in the early phase (less than 2 hours of immersion) and an osmotic diuresis in the late phase of

immersion.

Renal functions during immersion-exercise

Having established patterns of renal response to HOI

Fig. 5 Average time courses of urine flow rate during immersion-rest, immersion-exercise, air-rest and air-exercise.

Fig. 7 Total osmolar excretion during 3-h immersion as a function of exercise intensity. Solid symbol represents the value significantly different ($p<0.05$) from the corresponding value for immersion-rest.

Fig. 6 Total urine output during 3-h immersion as a function of exercise intensity. Solid symbols represent values significantly different ($p<0.05$) from the corresponding value for immersion-rest.

Fig. 8 Total sodium and potassium excretions during 3-h immersion as a function of exercise intensity. Solid symbols represent significantly different ($p<0.05$) from the corresponding value for immersion-rest.

in resting subjects, we next studied the renal response to HOI in exercising subjects. The subject performed three different levels of 3-hour leg exercise using a bicycle ergometer submerged in water. The average metabolic rates ($4.83 \dot{V}O_2$, $\text{kcal} \cdot \text{h}^{-1} \cdot \text{m}^{-2}$) for Exercise 1 (81 ± 4), 2 (122 ± 3) and 3 (153 ± 3) were 1.8, 2.7 and 3.8 times respectively higher than that for immersion-rest (46 ± 1).

Figure 5 illustrates time courses of urine flow rate (V) at each levels of exercise. With light exercise (Ex 1), the V changed similarly to that of immersion-rest during the whole course of experiment. With moderate (Ex 2) and heavy (Ex 3) exercises, however, the V changed identically to immersion-rest during the first 1 hour, but thereafter it declined quickly. Exercise in air induced no significant changes in V, as compared with Air-Rest.

Figures 6-8 summarize total urinary excretions of water and solute during 3-hour immersion as a function of exercise intensity. The exercise intensity was expressed as the average metabolic rate during 3-h immersion. As shown in Fig. 6, the total urine output during 3-hour

immersion was on the average $884 (\pm 70)$ ml in resting subjects. The value for subjects performing a light exercise (Ex 1, 848 ± 103) was similar to that above but those for subjects performing moderate (Ex 2, 592 ± 104) and heavy (Ex 3, 501 ± 91) exercises were significantly reduced ($p < 0.05$). Likewise, the urinary excretion of the total osmotic substances (Fig. 7) was reduced with exercise, especially at a high intensity (Ex 3), although the relative reduction was smaller than that in urine volume. The total Na^+ excretion progressively declined as the exercise intensity increased, whereas the total K^+ excretion was significantly enhanced by exercise of light (Ex 1) and moderate (Ex 2) intensities (Fig. 8).

In all the cases, the plasma levels of Na^+ , K^+ and osmolality, and creatinine clearance did not undergo significant variations during immersion (Table 1), thus the filtered loads of water and solute remained unaltered.

Table 2 shows changes in hematocrit, hemoglobin concentration, and plasma levels of renin activity (PRA), aldosterone (P_{aldo}) and ADH (P_{ADH}). In resting subjects,

Table 1 Plasma solute concentrations and creatinine clearance

		Rest	Ex 1	Ex 2	Ex 3
P_{Na} ($\text{mEq} \cdot \text{l}^{-1}$)	Pre-immersion	158 ± 1.8	160 ± 3.3	157 ± 1.3	158 ± 2.3
	Immersion	162 ± 1.2	159 ± 3.3	156 ± 1.8	159 ± 2.7
	Post-immersion	161 ± 1.2	159 ± 3.1	157 ± 1.4	159 ± 2.2
P_{K} ($\text{mEq} \cdot \text{l}^{-1}$)	Pre-immersion	4.4 ± 0.11	4.4 ± 0.19	4.2 ± 0.11	4.4 ± 0.10
	Immersion	4.3 ± 0.11	4.3 ± 0.15	4.0 ± 0.08	4.3 ± 0.18
	Post-immersion	4.4 ± 0.10	4.4 ± 0.17	4.0 ± 0.14	4.4 ± 0.13
P_{osm} ($\text{mOsm} \cdot \text{kg H}_2\text{O}^{-1}$)	Pre-immersion	294 ± 4.3	297 ± 1.9	293 ± 1.5	295 ± 1.7
	Immersion	299 ± 3.2	296 ± 2.3	290 ± 2.0	293 ± 3.4
	Post-immersion	296 ± 3.2	297 ± 1.6	291 ± 1.8	294 ± 2.0
C_{cr} ($\text{ml} \cdot \text{min}^{-1}$)	Pre-immersion	144 ± 8	155 ± 16	144 ± 12	138 ± 6
	Immersion	145 ± 6	147 ± 10	148 ± 4	148 ± 12
	Post-immersion	133 ± 16	$110 \pm 5^*$	125 ± 4	120 ± 6

Data represent the mean $\pm$ SE of 6 subjects. *significantly different from the corresponding pre-immersion value ($p < 0.05$).

Table 2 Hematocrit, hemoglobin concentration, and plasma levels of renin activity, aldosterone and ADH.

		Pre-immersion	Immersion	Post-immersion
Hct (%)	Rest	47.1 ± 0.7	46.1 ± 1.3	49.2 ± 0.9
	Ex 2	45.4 ± 1.1	43.2 ± 1.6	46.7 ± 0.8
Hb ($\text{g} \cdot \text{dl}^{-1}$)	Rest	17.3 ± 0.3	$15.9 \pm 0.3^*$	17.6 ± 0.5
	Ex 2	15.8 ± 0.3	15.0 ± 0.4	16.3 ± 0.3
PRA ($\text{ngAI} \cdot \text{ml}^{-1} \cdot \text{h}^{-1}$)	Rest	4.78 ± 1.02	$1.81 \pm 0.09^*$	3.53 ± 0.62
	Ex 2	3.59 ± 0.67	3.55 ± 1.02	5.85 ± 1.78
P_{aldo} ($\text{pg} \cdot \text{ml}^{-1}$)	Rest	152 ± 25	$67 \pm 3^*$	$87 \pm 18^*$
	Ex 2	102 ± 15	79 ± 10	99 ± 25
P_{ADH} ($\text{pg} \cdot \text{ml}^{-1}$)	Rest	1.08 ± 0.27	$0.63 \pm 0.15^*$	0.97 ± 0.22
	Ex 2	0.95 ± 0.24	$1.49 \pm 0.25^*$	$2.98 \pm 0.96^*$

Data represent the mean $\pm$ SE of 4 subjects. Metabolic rate ($\text{kcal} \cdot \text{h}^{-1} \cdot \text{m}^{-2}$): Rest, 43; Ex 2, 120. *significantly different from the corresponding pre-immersion value ($p < 0.05$).

Table 3 Comparison of immersion-induced changes in sodium and potassium excretions and creatinine clearance between Koreans and Caucasians

Group	24-h Excretion (mEq · day ⁻¹)		U _{Na} · V (mEq · min ⁻¹)			U _K · V (mEq · min ⁻¹)			Ccr (ml · min ⁻¹)		
	Na ⁺	K ⁺	Pre	Imm	Δ	Pre	Imm	Δ	Pre	Imm	Δ
Koreans											
Present Study (6)	232	40	334	450	116	77	100	23	141	145	4
Kim (70)	264-343	41-52									
Caucasians											
Epstein et al., 1978 (12)	Regular diet		104	210	106	44	101	57	132	141	9
Epstein et al., 1981a (6)	Regular diet		114	209	95	71	92	21	132	134	2
Epstein et al., 1972 (13)	150	100	59	168	109	51	105	54	117	133	16
Epstein et al., 1983 (8)			82	154	72	51	85	34	107	115	8
Epstein et al., 1973 (6)	150	100	92	207	115	38	66	28	163	131	-32
Epstein et al., 1979 (9)	Regular diet		87	146	59	40	68	28	133	134	1
Epstein et al., 1975a (10)			64	181	117	48	99	51	111	124	13
Epstein et al., 1981b (12)			73	159	86	59	87	28	123	133	10
Epstein et al., 1974 (10)	150	80	55	147	92	56	82	26	120	133	13
Krishna & Danovitch, 1983 (10)	Regular diet		155	273	118	61	114	53	124	122	-2
Mean			89	185	97	52	90	38	126	130	4

() number of subjects.

the PRA, P_{aldo} and P_{ADH} all declined significantly during immersion. In subjects performing moderate exercise, however, P_{ADH} increased and PRA and P_{aldo} remained unchanged during immersion. The hematocrit change during immersion was not significant both in resting and exercising subjects. However, the hemoglobin concentration appeared to be significantly reduced during immersion in resting subjects, but not in exercising subjects.

Discussion

Immersion-rest

The immersion-induced diuresis in the present study was comparable to those observed in previous studies under a similar condition. The urine flow increased from 1.5 ml · min⁻¹ before immersion to a peak value of 6.4 ml · min⁻¹ in the second hour of immersion (Fig. 1). This diuresis was accompanied by a change in free water clearance from -2.5 to 1.2 ml · min⁻¹ (Fig. 2); thus 76% of the increased urine flow (4.9 ml · min⁻¹) was accounted for by a reduction in free water reabsorption (3.7 ml · min⁻¹). A very similar phenomenon has been observed by Behn et al. (1969) and Epstein et al. (1972, 1973) in normally hydrated subjects (probably Caucasians). Such a change in free water balance may be resulted from a suppression of ADH secretion. The plasma ADH level determined during immersion was approximately 40% lower than the pre-immersion level (Table 2). According to previous reports (Epstein et al., 1981b, Ameln et al., 1985), ADH secretion decreases within 30 min of immersion. This change has been attributed to a

stimulation of cardiac volume receptor by an increased intrathoracic blood volume (Gauer and Henry, 1976; Epstein, 1978). The present results may, therefore, imply that the sensitivity of this volume receptor to HOI is not changed by chronic high salt diet. The amount of daily Na⁺ intake of the present subjects, as judged by 24-hour urinary Na⁺ excretion (232 mEq · day⁻¹), was more than 50% greater than that of Caucasians with normal salt intake (150 mEq · day⁻¹) (Table 3).

The natriuretic response to HOI in the present study was also similar to those observed in subjects ingesting normal salt diet. Epstein et al. (1972, 1973, 1975a) have reported that, unlike water excretion, the Na⁺ excretion increases progressively during immersion, peaking after 3-4 hours. In accordance with these reports, the Na⁺ excretion in the present study increased gradually during 3-hour immersion (Fig. 3). Furthermore, as shown in Table 3 which compares results obtained in the present and 10 other studies, the immersion-induced natriuresis (ΔU_{Na} · V) in the present subjects (116 μEq · min⁻¹) was of similar magnitude to that in Caucasian subjects (average 97 μEq · min⁻¹). The absolute amount of Na⁺ excretion was much greater in the former (334 and 450 μEq · min⁻¹ before and during immersion) than in the latter (89 and 185 mEq · min⁻¹ before and during immersion), probably due to higher salt intake. The mechanism(s) mediating the natriuresis may be multifactorial, including a suppression of renin-aldosterone system, alterations in renal prostaglandin release, an inhibition of renal sympathetic nerve activity, secretion of a humoral natriuretic factor, and alterations in intrarenal blood flow distribution (Epstein, 1978, Epstein et al., 1989). In the

present study, both PRA and P_{aldo} appeared to be significantly decreased during immersion (Table 3), as observed in previous studies (Epstein and Saruta, 1971; Epstein et al., 1975b). We, therefore, presume that at least the mechanism for aldosterone regulation during immersion is not changed by a chronic high salt diet.

The kaliuretic response to immersion was somewhat different between the present and other studies. In the present study, the K^+ excretion increased slightly (by about 35%, $p < 0.1$) during the first 2 hours (from 77 ± 14 to $104 \pm 16 \mu\text{Eq} \cdot \text{min}^{-1}$), and then it declined (Fig. 3). Such a pattern of kaliuresis is qualitatively similar to those observed in Caucasians (Epstein, 1978). Quantitatively, however, the net increase in K^+ excretion was considerably smaller in the present subjects ($23 \mu\text{Eq} \cdot \text{min}^{-1}$) than in Caucasians ($38 \mu\text{Eq} \cdot \text{min}^{-1}$) (Table 3). The reason for this difference is not apparent. The basal level of K^+ excretion of the present subjects ($77 \mu\text{Eq} \cdot \text{min}^{-1}$) was approximately 48% higher than those of Caucasians ($52 \mu\text{Eq} \cdot \text{min}^{-1}$) (Table 3). This may be due to a relatively high Na^+ excretion in the present subjects. High Na^+ excretion facilitates K^+ excretion by increasing the electrochemical potential gradient for K^+ secretion in the distal nephron (Wright and Giebisch, 1985).

Immersion-exercise

The present study clearly demonstrated that the renal response to HOI is modified by physical exercise. Exercise attenuated the diuretic and natriuretic responses, and potentiated the kaliuretic response (Figs. 6-8). These changes occurred without altering GFR (Ccr, Table 1), thus the renal tubular transports, not the filtered loads, of water and solutes were modified by exercise. The precise mechanism for this modification is not forthcoming, but it may be related, in part, to changes in blood volume regulating hormones such as renin, aldosterone and ADH. Unlike immersion at rest, the plasma levels of these hormone did not fall, but remained unchanged (renin and aldosterone) or increased significantly (ADH) during exercise with immersion (Table 2). These results are consistent with those observed by others in swimmers. Lenz et al. (1988) have reported that the plasma renin, aldosterone and vasopressin levels determined during 1000-m fin swimming were significantly higher than those measured during immersion at rest. Also, Böning et al. (1988) have observed no significant changes in plasma ADH and aldosterone concentrations during 60-min swimming as compared with the pre-immersion level. Since muscular exercise stimulates ADH and aldosterone secretion (Melin et al., 1980; Convertino et al., 1981), we presume that the effects of immersion on these hormone secretion may be offset by the exercise effect.

Such effect, however, may not fully account for the exercise-induced attenuation of immersion diuresis and

natriuresis, as the renal response to HOI is often observed not to be directly correlated with hormonal changes. For instance, Shiraki et al. (1986) have shown that the diuretic and natriuretic responses to HOI was significantly attenuated at night, despite the suppression of plasma ADH, renin and aldosterone was similar to that in daytime. Also Miki et al. (1988b) observed that plasma level of atrial natriuretic peptide (ANP) rose similarly during immersion at night and in daytime, yet the immersion natriuresis was significantly attenuated at night. In both studies, the rise in cardiac output measured by impedance cardiography was identical during the day and night periods, indicating that the input from cardiac mechanoreceptors was similar in both situations. These results suggest that the hormonal adjustments may not be the predominant mechanism mediating the diuresis and natriuresis of immersion.

In studies on conscious dogs, Miki et al. (1988a) have shown that HOI leads to a depression of renal sympathetic nervous activity and denervation of the kidneys in these dogs largely abolishes both the diuresis and the natriuresis. These results suggest that the renal sympathetic nerves play a major role in immersion diuresis and natriuresis in dogs. If this is also true in humans, it is possible that sympathetic activation evoked by exercise can overlie the mechanoreceptor-mediated suppression of renal sympathetic nerve activity during immersion; consequently, the renal response to immersion is attenuated.

Another mechanism for the suppression of immersion diuresis by exercise would be associated with the plasma volume change. Several studies on humans (Greenleaf et al., 1983; Khosla and DuBois, 1981) and dogs (Davis and DuBois, 1977; Miki et al., 1986, 1987) have demonstrated that a fluid shift into the vascular compartment occurs during immersion. The diuretic response acts to minimize the degree of vascular hypervolemia which would otherwise be quite large (Miki et al., 1987). If so, then the diuretic response should disappear if the fluid shift into the vascular compartment is prevented during immersion. Since, during exercise, cholinergic sympathetic vasodilator activity preferentially relieves the tone of precapillary sphincters, but not of postcapillary sphincters, the capillary pressure in the active muscles increases, facilitating fluid shift to the extravascular space (Little, 1981). Furthermore, intracellular hyperosmolality in working muscle induces an osmotic water shift into the cell (Lundvall, 1972; Costill, 1977). Consequently, the circulating plasma volume is reduced by as much as 10-13% at moderate or greater levels of exercise (Costill, 1977; Convertino et al., 1981; Geysant et al., 1981). Such an effect of exercise would counteract the effect of immersion on the plasma volume. Although we have not directly assessed plasma volume changes in the present study, the blood

hemoglobin data support this notion. The hemoglobin concentration appeared to be decreased during immersion-rest, suggesting hemodilution, but showed no significant change during immersion-exercise (Table 2). Also, Böning et al. (1988) have observed that the plasma volume loss during exercise with immersion (swimming) was very small compared to that for exercise on land, and they speculated that this was because interstitial fluid entered the vascular space during the initial phase of immersion.

Finally, the potentiation of kaliuresis by exercise during immersion (Fig. 8) may reflect synergism between exercise and immersion on renal K^+ excretion. Although the underlying mechanism may be distinct, both exercise (Castenfors, 1977) and immersion (Epstein, 1978) are known to facilitate the K^+ excretion.

Regardless of the mechanism, the present study emphasizes that an extensive urinary loss of water and Na^+ may be prevented by physical exercise during immersion. Such an effect may serve to preserve body fluid volume during prolonged underwater activity, such as long-distance swimming and skin diving, although an increase in K^+ loss may require attention.

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References

- Ameln H von, Laniado M, Röcker L, Kirsch KA (1985) Effects of dehydration on the vasopressin response to immersion. *J Appl Physiol* 58: 114-120
- Agostoni E, Gurtner G, Torri G, Rahn H (1966) Respiratory mechanics during submersion and negative-pressure breathing. *J Appl Physiol* 21: 251-258
- Arborelius M Jr, Balldin UI, Lilja B, Lungren CEC (1972) Hemodynamic changes in man during immersion with the head above water. *Aerospace Med* 43: 592-598
- Behn C, Gauer OH, Kirsch K, Eckert P (1969) Effects of sustained intrathoracic vascular distension on body fluid distribution and renal excretion in man. *Pflügers Arch* 313: 123-135
- Böning MM, Massen N, Busse M, Wagner TO (1988) Exercise versus immersion: antagonistic effects on water and electrolyte metabolism during swimming. *Eur J Physiol* 57: 248-253
- Castenfors J (1977) Renal function during prolonged exercise. *Ann NY Acad Sci* 301: 151-159
- Convertino VA, Keill LC, Bernauer EM, Greenleaf JE (1981) Plasma volume, osmolality, vasopressin, and renin activity during graded exercise in man. *J Appl Physiol* 50 (1): 123-128
- Costil DL (1977) Sweating: its composition and effects on body fluid. *Ann NY Acad Sci* 301: 160-170
- Craig AB Jr, Dvorak M (1975) Expiratory reserve volume and vital capacity of the lungs during immersion in water. *J Appl Physiol* 38: 5-9
- Davis JT and DuBois AB (1977) Immersion diuresis in dogs. *J Appl Physiol (Respir Environ Exerc Physiol)* 42: 915-922
- Epstein M (1978) Renal effects of head-out water immersion in man: Implications for an understanding of volume homeostasis. *Physiol Rev* 58: 529-581
- Epstein M, Saruta T (1971) Effect of water immersion on renin-aldosterone and renal sodium handling in normal man. *J Appl Physiol* 31: 368-374
- Epstein M, Duncan D, Fishman LM (1972) Characterization of the natriuresis caused in normal man by immersion in water. *Clin Sci* 43: 275-287
- Epstein M, Katsikas JL, Duncan DC (1973) Role of mineralocorticoids in the natriuresis of water immersion in normal man. *Circulation Res* 32: 228-286
- Epstein M, Schneider NS, Vaamonde CA (1974) Alterations in acid-base homeostasis during water immersion in normal man. *J Lab Clin Med* 84: 777-791
- Epstein M, Pins DS, Miller M (1975a) Suppression of ADH during water immersion in normal man. *J Appl Physiol* 38: 1038-1044
- Epstein M, Pins DS, Sancho J, Harber E (1975b) Suppression of plasma renin and plasma aldosterone during water immersion in normal man. *J Clin Endocr Metab* 41: 618-625
- Epstein M, Briker NS, Bourgoignie JJ (1978) The presence of a natriuretic factor in urine of normal men undergoing water immersion. *Kidney Intern* 13: 152-158
- Epstein M, Lifschitz MD, Hoffman DS, Stein JH (1979) Relationship between renal prostaglandin E and renal sodium handling during water immersion in normal man. *Circ Res* 45: 71-80
- Epstein M, DeNunzio AG, Loutzenhiser RD (1981a) Effects of vasopressin administration on diuresis of water immersion in normal humans. *J Appl Physiol* 51: 1384-1387
- Epstein M, Preston S, Weitzman RE (1981b) Isosmotic central blood volume expansion suppresses plasma arginine vasopressin in normal man. *J Clin Endocr Metab* 52: 256-262
- Epstein M, Johnson G, DeNunzio AG (1983) Effects of water immersion on plasma catecholamines in normal humans. *J Appl Physiol* 54: 244-248
- Epstein M, Loutzenhiser R, Friedland E, Aceto RM, Camargo MJF, Atlas SA (1987) Relationship of increased plasma natriuretic factor and renal sodium handling during immersion-induced central hypervolemia in

- normal humans. *J Clin Invest* 79: 738-745
- Epstein M, Norsk P, Loutzenhiser R (1989) Effects of water immersion on atrial natriuretic peptide release in humans. *Am J Nephrol* 9: 1-24
- Gauer OH, Henry JP (1976) Neurohumoral control of plasma volume. In Guyton AC, Cowley AW eds. *Cardiovascular physiology II*. Vol 9. University Park Press, Baltimore, 145-189
- Geysant AG, Geelen G, Denis C, Allevard AM, Vincent M, Jarsaillon E, Bizollon CA, Lacour JR, Charib CI (1981) Plasma vasopressin, renin activity, and aldosterone: Effect of exercise and training. *Eur J Appl Physiol* 46: 21-30
- Greenleaf JE, Morese JT, Baines PR, Silver J, Keil LD (1983) Hypervolemia and plasma vasopressin response during water immersion in man. *J Appl Physiol (Respir Environ Exerc Physiol)* 55: 1688-1693
- Khosla SS, DuBois AB (1981) Osmoregulation and interstitial fluid pressure changes in humans during water immersion. *J Appl Physiol (Respir Environ Exerc Physiol)* 51: 686-692
- Kim KJ (1974) Studies on the correlation between sodium chloride metabolism and blood pressure of Koreans. *Kor J Physiol* 8: 105-117
- Krasney JA, Hajduczuk G, Miki K, Claybaugh JR, Sondeen JL, Pendergast DR, Hong SK (1989) Head-out water immersion: A critical evaluation of the Gauer-Henry hypothesis. In Claybaugh JR and Wade CE, eds. *Hormonal regulation of fluid and electrolytes*. Plenum Pub Co. 147-185
- Krishna GG, Danovitch GM (1983) Renal response to central volume expansion in humans is attenuated at night. *Am J Physiol (Regul Integr Comp Physiol)* 13: R481-R486
- Krishna GG, Donovan GM, Sowers JR (1983) Catecholamine responses to central volume expansion produced by head-out water immersion and saline infusion. *J Clin Endocr Metabol* 56: 998-1002
- Lee WJ, Yang EK, Park YY, Park JS, Kim HJ (1995) Central ANG II-receptor antagonists impair cardiovascular and vasopressin response to hemorrhage in rats. *Am J Physiol* 268: R1500-R1506
- Lenz T, Weiss M, Werle E, Walz U, Kohler U, Pinther J, Weicker H (1988) Influence of exercise in water on hormonal, metabolic and adrenergic receptor changes in man. *Int J Sports Med* 9 (Suppl 2): S125-S131
- Lifschitz MD, Epstein M, Larios O (1985) Relationship between urine flow rate and prostaglandin E excretion in human beings. *J Lab Clin Med* 105: 234-238
- Little RC (1981) *Physiology of the heart and circulation*, 2nd ed. Year Book Medical Pub, INC, Chicago, 323-331
- Lundvall J (1972) Tissue hyperosmolality as a mediator of vasodilation and transcapillary fluid flux in exercising skeletal muscle. *Acta Physiol Scand (Suppl 379)*: 1-142
- Melin B, Eclache JP, Geelen G, Annat G, Allevard AM, Jarsaillon E, Zebidi A, Legros JJ, Gharib CL (1980) Plasma AVP, neurophysin renin activity and aldosterone during submaximal exercise performed until exhaustion in trained and untrained men. *Eur J Appl Physiol* 14: 141-151
- Miki K, Hajduczuk G, Hong SK, Krasney JA (1986) Plasma volume changes during head-out water immersion in the conscious dog. *Am J Physiol* 251: R582-R590
- Miki K, Hajduczuk G, Hong SK, Krasney JA (1987) Extracellular fluid and plasma volumes during water immersion in nephrectomized dogs. *Am J Physiol* 252: R972-R978
- Miki K, Hayashida Y, Sagawa S, Shiraki K (1988a) Role of sympathetic nerve activity in renal responses during head-out water immersion in conscious dogs. *FASEB J* 2: A1319
- Miki K, Shiraki K, Sagawa S, Devold AJ, Hong SK (1988b) Atrial natriuretic factor during head-out immersion at night. *Am J Physiol* 254: R235-R241
- Norsk P, Epstein M (1988) Effect of water immersion on arginine vasopressin release in humans. *J Appl Physiol* 64: 1-10
- Shiraki K, Konda K, Sagawa S, Claybaugh JR, Hong SK (1986) Cardiorenal- endocrine responses to head-out immersion at night. *J Appl Physiol* 60: 176-183
- Wright FS, Giebisch G (1985) Regulation of potassium excretion. In Seldin DW, Giebisch G eds. *The kidney. physiology and pathophysiology*. Vol 2. Raven Press, New York, 1223-1249

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