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 mEq·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, 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.


Keywords: head-out water immersion, kidney function, diuresis, natriuresis, exercise

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 (Krasnay et al., 1989) and negative pressure breathing of approximately 20 cm H₂O (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; Norsd 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., 1986) 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% VO₂max based on heart rate with assumed maximal heart rate 190 beats·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 (± 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 ± 0.5°C) water bath, and rested in a seated position or exercised at a constant intensity using a bicycle ergometer submerged 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 (cyanmethemoglobin 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 ± 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 (± 0.7) ml · min⁻¹, which was approximately 4-fold greater than the pre-immersion value 1.5 (± 0.2). Upon completion of immersion, the V promptly reverted to the pre-immersion level. By contrast, the Uosm decreased markedly from 808 (± 77) mOsm · kg H₂O⁻¹ before immersion to 249 (± 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 ± 1 SE of 6 subjects. Solid symbols represent values significantly different (p<0.05) from the respective pre-immersion value.](image-url)
Figure 2 depicts changes in free water clearance (C\(\text{H}_2\text{O}\)). The average C\(\text{H}_2\text{O}\) changed from \(-2.5 \pm 0.4\) ml \(\cdot\) 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 C\(\text{H}_2\text{O}\) became a positive value (1.2 \pm 0.4 ml \(\cdot\) min\(^{-1}\)), indicating that there was a net free water excretion. During the third hour of immersion the C\(\text{H}_2\text{O}\) returned to a negative value (\(-1.6 \pm 0.3\) ml \(\cdot\) 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 (± 126) μOsm \(\cdot\) min\(^{-1}\) to 1645 (± 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 (± 26) μEq \(\cdot\) min\(^{-1}\) was about 56% higher than the pre-immersion level 334 (± 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

![Graph](image1)

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

![Graph](image2)

**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.

![Graph](image3)

**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.

![Graph](image4)

**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 VO₂, kcal·h⁻¹·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 urinary 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 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 (± 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 (Pₐdo) and ADH (PₐDH). In resting subjects,

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Plasma solute concentrations and creatinine clearance</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Rest</td>
</tr>
<tr>
<td>Pₙa</td>
<td>Pre-immersion</td>
</tr>
<tr>
<td>(mEq·l⁻¹)</td>
<td>Immersion</td>
</tr>
<tr>
<td></td>
<td>Post-immersion</td>
</tr>
<tr>
<td>Pₖ</td>
<td>Pre-immersion</td>
</tr>
<tr>
<td>(mEq·l⁻¹)</td>
<td>Immersion</td>
</tr>
<tr>
<td></td>
<td>Post-immersion</td>
</tr>
<tr>
<td>Pₒsm</td>
<td>Pre-immersion</td>
</tr>
<tr>
<td>(mOsm·kgH₂O⁻¹)</td>
<td>Immersion</td>
</tr>
<tr>
<td></td>
<td>Post-immersion</td>
</tr>
<tr>
<td>Ccr</td>
<td>Pre-immersion</td>
</tr>
<tr>
<td>(ml·min⁻¹)</td>
<td>Immersion</td>
</tr>
<tr>
<td></td>
<td>Post-immersion</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Hematocrit, hemoglobin concentration, and plasma levels of renin activity, aldosterone and ADH.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-immersion</td>
</tr>
<tr>
<td>Hct</td>
<td>Rest</td>
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<tr>
<td>(%)</td>
<td>Ex 2</td>
</tr>
<tr>
<td>Hb</td>
<td>Rest</td>
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<tr>
<td>(g·dl⁻¹)</td>
<td>Ex 2</td>
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<tr>
<td>PRA</td>
<td>Rest</td>
</tr>
<tr>
<td>(ngAl·ml⁻¹·h⁻¹)</td>
<td>Ex 2</td>
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<tr>
<td>Pₒdo</td>
<td>Rest</td>
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<tr>
<td>(pg·ml⁻¹)</td>
<td>Ex 2</td>
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<tr>
<td>PₒDH</td>
<td>Rest</td>
</tr>
<tr>
<td>(pg·ml⁻¹)</td>
<td>Ex 2</td>
</tr>
</tbody>
</table>

Data represent the mean ± SE of 4 subjects. Metabolic rate (kcal·h⁻¹·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

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

Mean 89 185 97 52 90 38 126 130 4

( ) number of subjects.

the PRA, P<sub>ald</sub> and P<sub>ADH</sub> all declined significantly during immersion. In subjects performing moderate exercise, however, P<sub>ADH</sub> increased and PRA and P<sub>ald</sub> 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<sup>-1</sup> before immersion to a peak value of 6.4 ml · min<sup>-1</sup> 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<sup>-1</sup> (Fig. 2); thus 76% of the increased urine flow (4.9 ml · min<sup>-1</sup>) was accounted for by a reduction in free water reabsorption (3.7 ml · min<sup>-1</sup>). 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<sup>+</sup> intake of the present subjects, as judged by 24-hour urinary Na<sup>+</sup> excretion (232 mEq · day<sup>-1</sup>), was more than 50% greater than that of Caucasians with normal salt intake (150 mEq · day<sup>-1</sup>) (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<sup>+</sup> excretion increases progressively during immersion, peaking after 3-4 hours. In accordance with these reports, the Na<sup>+</sup> 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<sub>Na</sub> · V) in the present subjects (116 μEq · min<sup>-1</sup>) was of similar magnitude to that in Caucasian subjects (average 97 μEq · min<sup>-1</sup>). The absolute amount of Na<sup>+</sup> excretion was much greater in the former (334 and 450 μEq · min<sup>-1</sup> before and during immersion) than in the latter (89 and 185 μEq · min<sup>-1</sup> 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 \( P_{\text{aldo}} \) 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 ± 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 levels 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, Bönig 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; Geyssant 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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