Dehydration and body fluid-regulating hormones during sweating in warm (38°C) fresh- and seawater immersion

ARVID HOPE,¹ LEIF AANDERUD,² AND ASBJØRN AAKVAAG²

¹NUI AS, 5848 Bergen; and ²Haukeland University Hospital, 5021 Bergen, Norway

Received 25 February 2000; accepted in final form 18 May 2001

Hope, Arvid, Leif Aanderud, and Asbjørn Aakvaag. Dehydration and body fluid-regulating hormones during sweating in warm (38°C) fresh- and seawater immersion. *J Appl Physiol* 91: 1529–1534, 2001.—Body weight (BW) reductions of more than 4 kg have been observed during diving with the open hot water suit, a technique in which heated seawater (SW) continuously floods the skin surface. To test the hypothesis that osmotic effects may be involved in these fluid-loss processes, head-out immersion experiments in 38°C freshwater (FW) and SW for 4 h were performed. Average BW reduction was 2.5 and 1.9 kg in SW and FW head-out immersion, respectively $(P < 0.01)$. Atrial natriuretic peptide increased during the first 30 min of SW immersion $(5.6-13.4 \text{ pmol/l}, P < 0.01)$ followed by a reduction to 7.6 pmol/l $(P < 0.01)$. This paralleled an initial decrease in aldosterone (from 427 to 306 pmol/l, $P < 0.05$) followed by an increase to 843 pmol/l ($P < 0.01$). The effects of temperature on fluid loss were studied in thermoneutral (34.5°C) and 38°C SW for 2 h. In thermoneutral SW, calculated sweat production was negligible (0.05 kg) compared with 1.2 kg in warm SW. We recommend that, if a dive is planned to last for more than 4 h, a mandatory break for fluid intake should be incorporated in the diving regulations.

body weight; rectal temperature; arginine vasopressin; atrial natriuretic peptide; aldosterone; osmolality

THE PHYSIOLOGICAL EFFECTS OF IMMERSION are rather well understood. Initially, plasma volume increases and atrial natriuretic peptide (ANP) is released (3, 6), arginine vasopressin (AVP) is unchanged, and diuresis and sodium excretion are both increased (6, 15). Saturation divers are, in addition to immersion effects on body fluid balance, exposed to warm hyperosmolar seawater (SW) when using the open hot water suit (HWS). With this technique, cooling of the diver is avoided by surface-heated SW delivered via "umbilicals" to perforated hoses sewed into the suit material. Warm SW $(\sim 38^{\circ}C)$ thereby continuously floods the skin surface and may even result in increased core temperature (G. Knudsen and B. Holand, unpublished observations).

We have previously reported that diving with HWS may result in severe sweat production and body fluid losses of up to 4–5 kg or 5–6% of body weight (BW) (10). We have also suggested that warm and strongly

hyperosmotic SW may have an osmotic effect on the body water (11) and thereby partly explain the large fluid losses observed in operational diving (10). This is in accordance with findings made by Hertig et al. (9), who observed that sweating in warm water increased significantly when salt was added to the water. Furthermore, because the diver's skin in the wet suit is 100% humid, the normal evaporative heat loss does not occur and hyperthermia may develop more readily. Thus, if body core temperature increases and body fluid losses equivalent to more than 3% of BW occur simultaneously, a working diver is at risk for markedly impaired performance underwater (1, 19, 20). If dehydration and hyperthermia have developed at the end of the dive and a critical situation should occur, divers might not react adequately and thereby endanger their safety. It should also be emphasized that, during a normal working dive lasting for 4–6 h, divers normally have no fluid replacement.

Surprisingly, no efforts have previously been made to obtain detailed information about body fluid loss during HWS diving. Better understanding of the physiological mechanisms may be useful to improve divers' safety and performance. Therefore, the major objectives of the present head-out immersion study were to *1*) test the hypothesis that osmotic effects of the hyperosmotic SW may add to the BW reduction observed during warm freshwater (FW) immersion and *2*) determine the effect of ambient temperature on this fluid loss.

METHODS

Subjects. Sixteen male subjects from the Norwegian Navy Diving School participated in two experimental series. In *series 1* ($n = 8$), average age and BW were 21.9 \pm 1.7 yr (range 20–25 yr) and 76.0 \pm 10.6 kg (range 58–97 kg), and in *series 2* ($n = 8$), they were 20.8 \pm 1.4 yr (range 20–24 yr) and 72.5 ± 5.2 kg (range 65–79 kg). The regional ethics committee at the University of Bergen approved the study, and the experiments were performed according to the Helsinki Declaration. The subjects signed a written, informed consent after the purpose of the study and the experimental procedures and risks had been explained to them.

Experimental design and measurements. The experiments were performed in a temperature-controlled (accuracy $=$

Address for reprint requests and other correspondence: A. Hope, NUI AS, PO Box 23 Ytre Laksevaag, 5848 Bergen, Norway (E-mail: ah@nui.no).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

 ± 0.2 °C) immersion pool (2 \times 2 \times 2 m). The pool was filled with either FW or SW. Two thermistors (EUS-U-V5-0, Grant Instruments, Cambridge, UK) were located at 0.3- and 1.2-m depths for thermostatic feedback control of the water temperature. All experiments started at 8:00 AM, and two subjects were immersed simultaneously. Before immersion, the subjects undressed and had a rectal probe inserted. Urine and venous blood sampling, BW measurement, and heart rate measurement were performed. The two subjects were immersed \sim 20 min after the venous blood sample was taken and \sim 10 min after weighing.

In *series 1*, eight subjects were, in randomized order, immersed to the neck for 4 h in 38°C FW and 38°C SW. The subjects wore bathing suits and were seated with no activity during the experiment. Before immersion, no alcoholic beverages were allowed during the previous 24 h and no coffee or tea for the last 12 h. No fluid was consumed during the immersion period.

A series of measurements were performed to characterize a possible dehydration resulting from the warm FW and SW immersion (Table 1). BW was measured before immersion, after 120 min of immersion, and postimmersion on a balance (Soehnle S10 2720, Soehnle-Waagen, Murrhardt, Germany) to the nearest 0.1 kg. A net sweat volume was calculated as the difference between the BW reduction and the sum of the measured urine and blood sample volumes and the estimated

The x indicates measurements that were made. *In *series 2* only before and after immersion. †Only during seawater (SW) immersion.

Fig. 1. *A*: rectal and water temperatures in freshwater (FW) (TrecFW and TempFW, respectively) and seawater (SW) (TrecSW and TempSW, respectively) immersion in *series 1*. SD indicated for rectal temperature values. *B*: rectal and water temperatures in thermoneutral (TrecTN and TempTN, respectively) and warm (TrecW and TempW, respectively) SW immersion in *series 2*. SD indicated for rectal and water temperatures.

weight losses from expiratory water and metabolism. From the ideal gas law ($PV = nRT$) and assuming resting ventilation volumes of \leq 10 l/min and a dry inspired air, respiratory water loss would maximally amount to 25 g/h. Correcting for the relative humidity in the laboratory of 60%, this water loss would be \sim 10 g/h, i.e., less than 50 g during the 4-h immersion period. With a metabolic weight loss of \sim 15 g/h (12), the total insensible fluid loss was estimated to be maximally 100 g in 4 h.

A rectal probe (REC-U-UV5-0, Grant Instruments) was protected with a condom and inserted \sim 6 cm into rectum. Data from the rectal probe and the two thermistors measuring water temperatures were sampled and stored with 1-min intervals in a data logger (Grant Squirrel Series 1200, Grant Instruments).

Venous blood was sampled from the cubital vein 20 min before, at 30 and 210 min during, and 10 min after immer-

J Appl Physiol • VOL 91 • OCTOBER 2001 • www.jap.org

Fig. 2. Average values \pm SE of calculated sweat loss, urine volume, and the sum of blood sample volumes and estimated metabolic weight loss and respiratory fluid loss (MRB) during warm FW and SW immersion (38°C) for 4 h (series 1) and during thermoneutral (34.5°C) and warm SW immersion for2h(*series 2*). **Difference between sweat loss in warm FW and SW (*series 1*) and between urine production in thermoneutral and warm SW (*series 2*) was statistically significant $(P < 0.001)$.

sion. The pre- and postimmersion samples were taken after a 5-min rest in a seated position. Urine was voided and the bladder voluntarily emptied before, at a short interruption 120 min into, and 10 min after immersion.

The biochemical, hormonal, and thermal parameters are listed in Table 1. Hemoglobin, hematocrit, serum proteins, electrolytes, and osmolality were all analyzed by standard methods and instruments routinely used by the Laboratory for Clinical Biochemistry (Haukeland University Hospital, Bergen, Norway). Percent changes in blood and plasma volumes were calculated. The calculations are based on changes in hemoglobin and hematocrit as described by Dill and Costill (2). AVP was analyzed at the Hormone Laboratory (Aker University Hospital, Oslo, Norway) using a radioimmunoassay method that includes extraction on a SEP-PAK C18 column. The reference area is 0.9–9.2 pmol/l, and the sensitivity of the method is 0.5 pmol/l. Plasma level of ANP was measured as described by Omland et al. (16). The method utilizes a radioimmunoassay kit from Nycomed, Amersham, UK, and includes an extraction step on a C18 octadecyl minicolumn. The limit of detection is 5.0 pmol/l. The precision of the assay (the interassay coefficient of variation) is 12% at the level of 22 pmol/l and 6.2% at 82 pmol/l. Aldosterone was determined using a radioimmunoassay kit from Diagnostic Products (Los Angeles, CA). The method has a detection limit of 44 pmol/l and a sensitivity (interassay coefficient of variation) of 9.5% for the entire concentration range from 88 to 1,550 pmol/l.

In *series 2*, eight subjects were immersed for 2 h in thermoneutral (34.5°C) and warm (38°C) SW. The same procedures were followed as in *series 1*, but with sampling of blood and urine pre- and postimmersion only. The parameters measured are listed in Table 1.

Statistical analysis. Values are presented as means \pm SD or SE as indicated. A two-tailed *t*-test was used for paired comparisons between pre- and postimmersion measurements and between variables in the two different groups in each series. Differences were considered significant at the 5% probability level.

RESULTS

Series 1: 4-h immersion in SW and FW at 38°C. The mean rectal temperature leveled off after 90 min of immersion around 38°C, with no significant difference between the groups (Fig. 1*A*). Average BW reduction was 2.5 kg (range = $1.3-4.5$ kg) and 1.9 kg (range = 0.8–3.5 kg) in SW and FW, respectively $(P < 0.01,$ Fig. 2). The mean weight losses corresponded to 3.4% $(\text{range} = 1.7{\text -}5.9\%)$ and 2.6% (range = 1.1–4.6%) of BW during SW and FW immersion.

Calculated mean sweat volumes were 2.1 and 1.5 kg during SW and FW immersion, respectively $(P < 0.001$, Fig. 2). Urinary output was decreased to 50% in both SW and FW after 120 min of immersion (Table 2) with concomitant increased urine osmolality and decreased sodium and potassium excretion during the last 2 h. Electrolyte excretion and urine osmolality were not significantly different between SW and FW immersion (Table 2).

Pre- and post-SW immersion measurements showed significant changes in hematocrit, hemoglobin, albumin, serum osmolality, serum sodium $(P < 0.05)$, and percent changes in blood and plasma volume $(P < 0.01)$. In FW immersion, only blood and plasma volume and hemoglobin changed significantly $(P < 0.05$, Table 3). The differences in these changes between SW and FW immersion experiments were also significant (Table 3).

Due to great individual scatter in AVP levels, an insignificant and inconsistent increase from 15.3 to

Table 2. *Body weight reduction, urine osmolality, diuresis, and sodium and potassium excretion during the first and second halves of 4-h immersion in FW and SW*

Immersion Period	ΔBW , kg	Osmolality, mosh/kgH ₂ O	V. ml/h	$U_{\text{Na}}V$, mmol/h	U_KV , mmol/h
FW I	0.98 ± 0.44	924 ± 158	40.8 ± 16.5	4.11 ± 1.14	5.73 ± 1.38
FW II	0.93 ± 0.39	1.045 ± 104	$17.8 \pm 5.8^*$	$1.63 \pm 0.70^*$	$2.89 \pm 1.32^*$
SW I	1.24 ± 0.80	814 ± 122	52.8 ± 19.0	6.57 ± 1.91	5.69 ± 2.03
SW II	1.25 ± 0.40 †	931 ± 77	25.4 ± 9.08	4.43 ± 2.69	$3.56 \pm 1.41\$

Values are means \pm SD. Δ BW, body weight reduction; V, diuresis; FW, freshwater. I, first half; II, second half. *Significant difference vs. FW I ($P < 0.01$). †Significant difference vs. FW II ($P < 0.05$). ‡Significant difference vs. SW I ($P < 0.05$). §Significant difference vs. SW I $(P < 0.01)$.

	SW Immersion		FW Immersion		$%$ Change	
Parameter	Pre	Post	Pre	Post	SW	FW
Hct, 1/1 full blood	0.43 ± 0.04	$0.46 \pm 0.03*$	0.44 ± 0.04	0.46 ± 0.03	8.1 ± 4.7	4.7 ± 5.7
Hb , g/dl	14.9 ± 1.3	$15.9 \pm 1.1^*$	15.4 ± 0.9	$16.0 \pm 0.9^*$	7.5 ± 4.0	$4.0 \pm 4.0^+$
$S-Alb, g/l$	45.3 ± 2.3	$48.8 \pm 2.6^*$	47.3 ± 2.5	49.3 ± 2.4	7.8 ± 4.7	$4.4 \pm 5.0^+$
S-Osm, mosmol/kg H_2O	289.6 ± 3.4	$296.8 \pm 6.5^*$	291.5 ± 3.9	295.5 ± 4.8	2.5 ± 1.6	1.4 ± 1.8
S-Na, mmol/l	139.5 ± 2.3	$143.0 \pm 3.6^*$	140.6 ± 3.4	141.4 ± 2.8	2.5 ± 2.7	0.6 ± 2.1
B-vol, $%$	100.0 ± 0.0	$93.1 \pm 3.4^*$	100.0 ± 0.0	$96.3 \pm 3.6^*$	6.9 ± 3.4	3.7 ± 3.6 †
P-vol, $%$	57.3 ± 3.8	$50.0 \pm 3.1^*$	55.9 ± 2.5	$51.9 \pm 3.7^*$	12.2 ± 5.8	7.0 ± 7.6 †

Table 3. *Hematocrit, Hb, S-Alb, S-Osm, and S-Na before and after SW and FW immersion, and percent change in calculated B-vol and P-vol*

Values are means \pm SD. Hct, hematocrit; Pre, before immersion; Post, after immersion. *Significant difference vs. Pre ($P < 0.05$). \dagger Significant difference vs. SW ($P < 0.05$).

61.7 pmol/l was observed during SW immersion. ANP levels were significantly increased throughout the immersion period (Fig. 3). ANP increased significantly during the first 30 min of SW immersion from 5.6 to 13.4 pmol/l ($P < 0.05$), followed by a reduction to 7.6 pmol/l postimmersion. This paralleled an initial decrease in aldosterone from 427 to 306 pmol/l at 30 min $(P < 0.05)$, followed by an increase to 843 pmol/l postimmersion $(P < 0.01,$ Fig. 3). Almost identical changes in aldosterone were observed during FW im-

Fig. 3. Average values \pm SE of atrial natriuretic peptide (ANP; *A*), aldosterone (*B*), and arginine vasopressin (AVP; *C*) in *series 1* before (-20 min) , during (30 and 210 min), and after (250 min) 4-h 38°C SW immersion. $*P < 0.05$ vs. -20 min. $**P < 0.01$ vs. -20 min. ***Not significant vs. -20 , 30, and 250 min. Significance levels between blood samples are also shown.

mersion (398, 289, and 867 pmol/l, respectively). AVP and ANP were not measured during FW immersion.

Series 2: 2-h immersion in thermoneutral (34.5°C) and warm (38.2°C) SW. As in *series 1*, rectal temperature leveled off around 38.1°C after 90 min of warm SW immersion (Fig. 1*B*). In thermoneutral conditions, an insignificant reduction in core temperature was observed. Calculated sweat volumes increased from 0.05 kg after 2 h in thermoneutral SW to 1.2 kg in warm SW (Fig. 2). The urine production in 2-h warm SW immersion fell to one-third of the values from thermoneutral immersion (Fig. 2).

DISCUSSION

Diving with the open HWS exposes the skin surface continuously to warm SW with a temperature of $\sim 38^{\circ}$ C. Hot water temperatures of \sim 40°C at the suit inlet, resulting in rectal temperatures of 38°C, have been observed in operational HWS diving (G. Knudsen and B. Holand, unpublished observations). BW losses >3 kg and 4% of BW have also been reported (10, 11). From this, it would be reasonable to conclude that many divers are passively heated and lose body fluids by sweating when using this technique for diving. The purpose of the present investigation was to study the effects of ambient water temperature and osmolality on the body fluid loss during head-out immersion and describe the compensatory hormonal reactions.

The significant difference, averaging 0.6 kg, in calculated sweat loss between SW and FW experiments $(P < 0.001,$ Fig. 2) supports our hypothesis that osmotic effects are involved in these fluid-loss processes. Furthermore, because the calculated sweat loss was zero in thermoneutral SW immersion, osmosis exerts its effects only when sweat is being produced and the sweat channels are open.

SW is strongly hyperosmotic $(\sim 1,000 \text{ mes})$ $kgH₂O$) and FW is strongly hypoosmotic ($<$ 20 mosmol/ $kgH₂O$) compared with plasma (290 mosmol/kg $H₂O$) and with hypotonic sweat (\sim 150 mosmol/kgH₂O). Thus the osmotic pressure difference between the immersion medium and body fluids will have opposite directions during immersion in SW vs. FW. This is in accordance with observations by Hertig et al. (9), who found that sweating in warm water increased significantly when 5% salt was added to the immersion water.

A greater hemoconcentration and dehydration is reflected by the 7.5 and 8.1% increases in hemoglobin and hematocrit, respectively, after SW, compared with 4.0 and 4.7% after FW immersion ($P < 0.05$, Table 3). The corresponding plasma volume reduction was 12.2 and 7% ($P < 0.05$) in SW and FW immersion, respectively. This difference is further illustrated by the significant changes in both plasma albumin and osmolality during SW but not FW immersion (Table 3). Previous immersion studies in thermoneutral water showed that initially increased plasma volume is followed by a decrease (8, 13, 14) and that these isotonic fluid shifts are of extracellular origin (7).

One could argue that a time period of 10 min from the end of immersion to postimmersion blood sampling, ending with 5 min in a seated position, is too short to restore fluid balance between central and peripheral tissues and between intra- and extracellular compartments. The changes observed from the end of immersion to postimmersion (Fig. 3), and the differences between pre- and postimmersion values (Table 3), would probably be even greater if extending these time periods. However, because the procedure was identical in FW and SW immersion, it would be proper to compare changes in blood parameters between the two groups (Table 3).

When the sweat glands are inactive, as in thermoneutral immersion in *series 2*, body fluid loss due to the hyperosmotic SW does not occur (Fig. 2). During thermoneutral immersion for 2 h, the urine volume produced was 600 ml $(\sim 4.4 \text{ m/min})$ and approximately three times that produced during 4-h warm SW immersion (200 ml or 0.7 ml/min). This corresponds to $\langle 10\%$ of the total fluid loss (Fig. 2) and clearly indicates that warm SW immersion prevents the normally occurring immersion diuresis.

The initial increases in ANP illustrated in Fig. 3 are in accordance with previous reports (3, 21). ANP release during the first 30 min, stimulated by an increase in central blood volume and atrial distension (4), results in a diuresis with diluted urine (21). This is compatible with the simultaneous, unaltered AVP level (Fig. 3). This difference in ANP and AVP release in euhydrated subjects indicates that, during the initial phase of warm water immersion, ANP is the primary regulator of water excretion. During the next 3 h in the SW experiments, ANP and electrolyte excretion decreased (Fig. 3, Table 2). This is in contrast to reports from thermoneutral immersion in which high ANP levels are maintained because of persistently increased central blood volume (17, 21). Simultaneously, an increased AVP level was observed (Fig. 3), resulting in decreased urine production (Table 2).

After a 3-h thermoneutral water immersion, several studies conclude that the serum aldosterone level is decreased as a result of the normally occurring hemodilution followed by an immersion diuresis (5, 18, 22). After an initial and significant decrease in aldosterone,

we observed increasing aldosterone levels, with maximum values postimmersion (Fig. 3). This increase in aldosterone correlated with the decreased electrolyte excretion (Table 2).

From the present investigation, we conclude that fluid loss by sweating in warm water is greater in hyperosmotic SW $(1,000 \text{ mosh}/\text{kgH}_2\text{O})$ than in hypoosmotic FW $(20 \text{ mosmol/kgH}_2O)$. Because the fluid loss across the skin surface is negligible in thermoneutral SW, fluid loss in warm water must be related to open sweat channels. As suggested by Hertig et al. (9), a reduced sweat fluid loss in FW may be explained by a mechanical occlusion of the distal parts of the sweat ducts because of swelling of the stratum corneum. This suppression of sweat exudation is counteracted in hyperosmotic immersion water. An additional effect may be caused by an osmotic water flux across the epithelial lining of the sweat channels.

The present findings may have implications for fluid balance in occupational diving because fluid losses $>3\%$ may reduce mental as well as physical performance (1, 20). Until a fluid supply system for divers in water is developed, and if a dive is planned to last for more than 4 h, we recommend that a mandatory break for fluid intake should be incorporated in the diving regulations. We also suggest that intake of fluids with diuretic effects should be restricted before diving.

The skillful technical assistance of Harald Sundland, Rune Pettersen, Janne Mo, Anne Gurd Lindrup, Egil Jensen, and Martha Forland is gratefully acknowledged.

The Norwegian Space Center, Norwegian Petroleum Directorate, Norwegian Board of Health, Confederation of Norwegian Business and Industry, Statoil, Norsk Hydro, and Saga Petroleum financially supported this study.

REFERENCES

- 1. **Åstrand PO and Rodahl K.** *Textbook of Work Physiology*. New York: McGraw-Hill, 1986.
- 2. **Dill DB and Costill DL.** Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* 37: 247–248, 1974.
- 3. **Epstein M.** Renal effects of head-out water immersion in humans: a 15-year update. *Physiol Rev* 72: 563–621, 1992.
- 4. **Epstein M, Norsk P, and Loutzenhiser R.** Effect of water immersion on atrial natriuretic peptide release in humans. *Am J Nephrol* 9: 1–24, 1989.
- 5. **Farrow S, Banta G, Schallhorn S, May R, Mers A, Cadaret L, Rydstedt L, and Lockette W.** Vasopressin inhibits diuresis induced by water immersion in humans. *J Appl Physiol* 73: 932–936, 1992.
- 6. **Greenleaf JE, Morse JT, Barnes PR, Silver J, and Keil LC.** Hypervolemia and plasma vasopressin response during water immersion in men. *J Appl Physiol* 55: 1688–1693, 1983.
- 7. **Greenleaf JE, Shevartz E, and Keil LC.** Hemodilution, vasopressin suppression, and diuresis during water immersion in man. *Aviat Space Environ Med* 52: 329–336, 1981.
- 8. **Harrison MH, Keil LC, Wade CA, Silver JE, Geelen G, and Greenleaf JE.** Effect of hydration on plasma volume and endocrine responses to water immersion. *J Appl Physiol* 61: 1410– 1417, 1986.
- 9. **Hertig BA, Riedesel ML, and Belding HS.** Time course of sweating in warm baths. *Adv Biol Skin* 3: 213–229, 1962.
- 10. **Hope A.** Loss of body fluid during operational diving with the open hot water suit. In: *XXIst Annual EUBS Meeting on Diving* and Hyperbaric Medicine, edited by Sipinen SA and Leiniö MA.

Helsinki, Finland: European Underwater and Baromed. Soc., 1995, p. 170.

- 11. **Hope A, Hjelle JO, and Reed RK.** Effects on body fluid balance of repeated daily diving for one week (Abstract). *XIVth Annual EUBS Meeting on Diving and Hyperbaric Medicine*. Aberdeen, Scotland: European Undersea Biomed. Soc., 1988, abstr. no. 16.
- 12. **Keele CA and Neil E.** *Samson Wright's Applied Physiology*. London: Oxford University Press, 1971, p. 204–205.
- 13. **Khosla SS and DuBois AB.** Osmoregulation and interstitial fluid pressure changes in humans during water immersion. *J Appl Physiol* 51: 686–692, 1981.
- 14. **Kravik SE, Keil LC, Silver JE, Wong N, Spaul WA, and Greenleaf JE.** Immersion diuresis without expected suppression of vasopressin. *J Appl Physiol* 57: 123–128, 1984.
- 15. **Norsk P, Drummer C, Johansen LB, and Gerzer R.** Effect of water immersion on renal natriuretic peptide (urodilatin) excretion in humans. *J Appl Physiol* 74: 2881–2885, 1993.
- 16. **Omland T, Bonarjee VVS, Aakvaag A, Aarsland T, and Dickstein K.** The relationship between early plasma atrial natriuretic factor levels and exercise performance after myocardial infarction. *Eur Heart J* 14: 1022–1026, 1993.
- 17. **Pendergast DR, De Bold AJ, Pazik M, and Hong SK.** Effect of head-out immersion on plasma atrial natriuretic factor in man. *Proc Soc Exp Biol Med* 184: 429–435, 1987.
- 18. **Rim H, Yun YM, Lee KM, Kwak JT, Ahn DW, Choi JK, Kim KR, Joh YD, Kim JY, and Park YS.** Effect of physical exercise on renal response to head-out water immersion. *Appl Human Sci* 16: 35–43, 1997.
- 19. **Saltin B.** Circulatory responses to submaximal and maximal exercise after thermal dehydration. *J Appl Physiol* 19: 1125– 1132, 1964.
- 20. **Sharma VM, Sridharan K, Pichan G, and Panwar MR.** Influence of heat-stress induced dehydration on mental functions. *Ergonomics* 29: 791–799, 1986.
- 21. **Stadeager C, Johansen LB, Warberg J, Christensen NJ, Foldager N, Bie P, and Norsk P.** Circulation, kidney function, and volume-regulating hormones during prolonged water immersion in humans. *J Appl Physiol* 73: 530–538, 1992.
- 22. **Warner LC, Morali GA, Miller JA, Logan AG, Skorecki KL, and Blendis LM.** Aldosterone, atrial natriuretic factor and sodium intake as determinants of the natriuretic response to head-out water immersion in healthy subjects. *Clin Sci (Lond)* 80: 475–480, 1991.

