The Role of Posture in the Natriuresis of Water Immersion in Normal Man (36972)

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(Introduced by William J. Harrington)

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". . . if the blood be thus driven (by the bath) from both the external and internal parts, what *becomes* of the blood? The heart and great vessels, it would seem, must be burdened. Such is to a degree the case; and it is perhaps the stimulus of this fullness and distention or its action on the elasticity of those great vessels and the heart that constitutes the *reaction* (which leads forth the urine in abundant effusion). Such overloading of the heart and great organs would be dangerous in every case if the volume of blood remained the same." Henry Hartshorne, 1847 (1).

Previous studies from this laboratory have demonstrated that water immersion has profound effects on the renin–aldosterone system and renal sodium handling in normal seated subjects (2–4). Although these changes are thought to be mediated in part by the redistribution of blood volume induced by immersion (5), the precise mechanism whereby water immersion exerts this effect remains unclear. Since the pressure exerted on body surfaces increases with increasing depth of immersion, it has been suggested that during water immersion in an upright posture, a significant hydrostatic pressure gradient is induced between various levels of the body which acts on the vascular beds of the body differentially (5, 6). Conversely, water immersion to the neck in the supine position would tend to minimize this hydrostatic pressure gradient effect.

Recently, several studies have reported that water immersion during recumbency was associated with a significant natriuresis (7, 8), thus challenging this formulation. The current study was undertaken to assess the effects of water immersion on renal sodium handling while supine, under carefully controlled conditions. It was anticipated that such studies would further elucidate the mechanism involved in the natriuresis of water immersion.

Materials and Methods. Seven healthy male subjects between the ages of 18 and 25 yr were studied. None had a history of hypertension, cardiovascular disease or diabetes. Significant renal disease was excluded by documenting a normal urine sediment and creatinine clearance and negative urine cultures. The subjects were housed during the study in an environmentally controlled metabolic ward at a constant temperature. Each consumed a diet containing 150 mEq sodium, 60 mEq potassium and 2000 ml water whose composition remained unchanged throughout the study. Daily 24-hr urine collections were made for determination of sodium, potassium and creatinine.

Control and immersion studies were carried out in each subject on the third and fifth days of dietary equilibration, respectively, by which time all subjects had achieved sodium balance. On study days, identical protocols were carried out as follows:

The subject was awakened 0730 and instructed to void. After completely emptying his bladder, he was given a 700 ml oral water load and then assumed a seated position for 1 hr. During control studies, the subject then assumed the supine position on a cot for a 5-hr period. During immersion, the subject assumed the supine position on a cot placed in the study tank such that his whole body was under water except for his head and neck.

Each subject stood briefly every hour to void spontaneously. To maintain an adequate urine flow, 200 ml water was administered orally every hour during each study. Sodium,

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potassium and creatinine were measured in aliquots of the hourly urine collections. Blood was collected at 2-hr intervals throughout the study. All subjects were weighed every morning at 0730 after voiding, and before and after each study.

Immersion was carried out in a waterproof tank described in detail in a previous communication (4). A constant water temperature of 34 ± 0.5° was maintained by two heat exchangers with a combined output of 13,500 BTU/hr controlled by an adjustable temperature-calibrated control meter, with input derived from two thermistors immersed at different water levels.

Sodium and potassium were analyzed with a IL flame photometer. Creatinine was measured by an automated adaptation of Jaffee's picric acid reaction (9). In the presentation of the data, mean values are followed by the standard error of the mean as an index of dispersion. Tests of statistical significance were calculated by means of a paired *t* test.

Permission for the study was obtained from each subject after a detailed description of the procedures and their potential complications was given. The protocol was approved by the Human Experimentation Committees of the University of Miami School of Medicine and the Miami Veterans Administration Hospital. No complications occurred.

Results. Urinary sodium and potassium. The effects of 5 hr of water immersion on sodium and potassium excretion are shown in Table I. During control, the change in posture from the seated to the supine position was accompanied by a gradual increase in the rate of sodium excretion from 68 to 153 μEq/min. When an identical protocol was carried out while the subjects were immersed to the neck (immersion), the resultant increase in $U_{Na}V$ from 70 to 180 μEq/min did not differ from control during any hour of study ($p > 0.1$). The absolute quantity of sodium excreted during 5 hr of immersion was 41.3 ± 6.6 mEq, not different from the 37.3 ± 6.8 mEq during control ($p > 0.4$).

Fractional excretion of sodium ($C_{Na}/C_{Cr} \times 100$) during immersion did not differ from control ($p > 0.1$), mirroring the absence of change of $U_{Na}V$.

TABLE I. Effects of Immersion on Urinary Excretory Patterns (Results are Mean ± SE of 7 subjects).

	Group	(hr): Preimmersion	1	2	3	4	5
V	Control	2.5 ± 0.5	5.8 ± 0.6	5.9 ± 0.4	4.4 ± 0.7	5.2 ± 0.5	4.2 ± 0.6
(ml/min)	Immersion	3.1 ± 0.8	5.4 ± 0.4	10.7 ± 0.5[a]	3.9 ± 0.6	6.5 ± 0.8	4.3 ± 0.7
$U_{Na}V$	Control	68 ± 8	93 ± 23	98 ± 20	126 ± 28	153 ± 25	153 ± 24
(μEq/min)	Immersion	70 ± 18	89 ± 19	120 ± 22	134 ± 29	180 ± 24	167 ± 25
U_KV	Control	58 ± 6	85 ± 22	91 ± 14	93 ± 13	93 ± 10	72 ± 9
(μEq/min)	Immersion	84 ± 33	80 ± 14	91 ± 14	89 ± 17	87 ± 11	63 ± 7
C_{Cr}	Control	144 ± 17	139 ± 17	126 ± 6	123 ± 12	138 ± 12	123 ± 16
(ml/min)	Immersion	121 ± 10	134 ± 9	104 ± 10	114 ± 13	117 ± 10	106 ± 10
$C_{Na}/C_{Cr} \times 100$	Control	0.37 ± 0.08	0.45 ± 0.07	0.59 ± 0.09	0.75 ± 0.14	0.81 ± 0.11	0.93 ± 0.08
	Immersion	0.43 ± 0.12	0.45 ± 0.08	0.75 ± 0.14	0.77 ± 0.11	1.06 ± 0.10	1.06 ± 0.10
C_{H_2O}	Control	0.3 ± 0.5	3.3 ± 0.3	3.4 ± 0.5	2.1 ± 0.7	2.5 ± 0.5	2.0 ± 0.6
(ml/min)	Immersion	1.2 ± 0.8	3.0 ± 0.4	8.5 ± 0.6[a]	1.7 ± 0.4	4.1 ± 1.0	2.3 ± 0.5

[a] $p < .05$ differences from control.

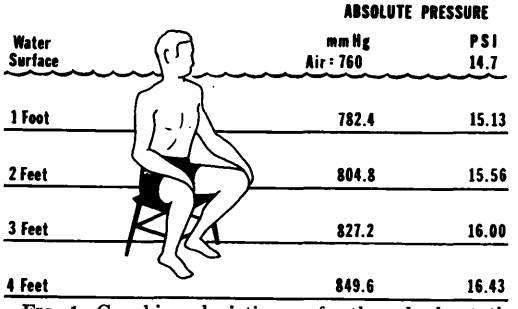

FIG. 1. Graphic depiction of the hydrostatic pressure gradient induced by water immersion to the neck in the upright posture. The pressure exerted on body surfaces increases by 22.4 mm Hg for each foot of water depth.

The rate of potassium excretion (U_KV) during immersion did not differ from control during any hour of study. Similarly, the absolute quantity of potassium excreted during 5 hr of immersion was 24.4 ± 3.4 mEq, not different from the 26.0 ± 3.3 mEq during control ($p > 0.1$).

Urine volume, C_{H_2O} and C_{Cr} (Table I). Urine flow during control ranged from 4.2 to 5.9 ml/min following assumption of recumbency. Immersion did not alter urine flow rates except for an increase during Hour 2 of recumbency which was attributable primarily to an increase in C_{H_2O}.

Creatinine clearance (C_{Cr}) during each hour of immersion was not different from the preimmersion hour. Furthermore, C_{Cr} during each hour of immersion did not differ from the comparable control hours.

C_{H_2O} during control ranged from 2.0 to 3.3 ml/min. Immersion did not significantly alter C_{H_2O}, except for an isolated increase during Hour 2. Factoring of C_{H_2O} values for the C_{Cr} of the corresponding clearance period did not disclose additional differences.

Discussion. Water immersion to the neck has previously been shown to produce a profound natriuresis and suppression of the renin–aldosterone system in normal seated subjects (2–4). Several lines of evidence have suggested that these effects are mediated by a redistribution of blood volume with a relative increase in central blood volume (5, 10). Gauer has suggested that this redistribution may be mediated by an immersion-induced hydrostatic pressure gradient acting on the vascular columns of the body (5, 6). As shown in Fig. 1, the pressure exerted on body surfaces increases by 22.4 mm Hg for each foot of water depth. The net effect of this gradient is to force blood from the capacitance vessels of the lower extremities with the result that more blood returns to occupy the vessels of the heart and lungs. Recognition by the intrathoracic volume receptors of such an increase in central blood volume would result in a decrease in renin–aldosterone release and a natriuretic response (2, 4, 5). Recent reports of increase in $U_{Na}V$ in subjects undergoing water immersion in the supine position are at variance with such a formulation (7, 8).

The present studies demonstrate that water immersion in supine subjects did not significantly alter renal sodium handling compared to an identical supine posture without immersion. Although small decreases in creatinine clearance might have masked an increase in $U_{Na}V$, the demonstration that fractional excretion of sodium did not increase during immersion excludes this possibility. Thus, while these observations do not exclude the possibility that mechanisms other than a hydrostatic pressure gradient participate in producing the increase in central blood volume during immersion and resulting natriuresis, they do suggest that such other mechanisms are unlikely to be a predominant factor in this phenomenon.

Previous investigators examining renal sodium handling during head-out water immersion in the supine position have reported a natriuresis. However, in the studies reported, either the experimental design or methodologic differences might explain their results. Thus although Behn *et al.* (7) reported a 53–127% increase in $U_{Na}V$ during an 8-hr period of immersion compared to control, it should be noted that the "control" period which they utilized for comparison consisted of an 8-hr period of normal upright activity on the day preceding immersion. Since upright posture is known to induce an antinatriuresis (11, 12), the smaller $U_{Na}V$ during "control" probably reflects the antinatriuretic effect of upright posture, and the progressive increase in $U_{Na}V$ may merely reflect the assumption of recumbency. This formulation is

consistent with the demonstration that in our laboratory, normal subjects in balance on an identical diet excrete 14.9 ± 2.2 mEq Na ($n = 6$) during a 5-hr period of normal activity during an identical time of day (unpublished observations), compared to 37.3 ± 6.8 mEq/5 hr during the supine control in the present study.

Boening *et al.* (8) reported an increase in $U_{Na}V$ compared to recumbent control. However, Boening and co-workers' study protocol differed from ours in that the subjects ate breakfast and were given a supplemental feeding of powdered milk at hourly intervals. In addition, the subjects reclined in a deck chair during immersion, with a resultant elevation of the upper half of the body relative to the buttocks. Thus, it is conceivable that a small albeit significant hydrostatic gradient was operative.

The transition from the upright to the supine posture is associated with a redistribution of blood volume with an increase in central blood volume of approximately 400 ml (13). Aborelius *et al.* (10) have recently demonstrated an increase in central blood volume of 700 ml in seated subjects undergoing water immersion, compared to seated controls. The increase in central blood volume induced by assumption of the supine position *per se* may preclude any further increase in central blood volume by the superimposition of water immersion. Thus, further increases in sodium excretion would not occur during supine immersion. Finally, it is possible that the interaction of additional variables including a relatively lessened negative pressure breathing situation contributes to the absence of a significant natriuresis during supine immersion.

Summary. Water immersion to the neck has been demonstrated to produce a profound natriuresis in seated and standing subjects. Since an immersion-induced hydrostatic pressure gradient with a resultant redistribution of blood volume has been postulated to produce this natriuresis, it was of interest to examine this postulate by assessing renal sodium handling during immersion in supine subjects. Renal sodium, potassium and water handling was assessed in seven normal subjects during a control period and during water immersion under identical conditions of diet, supine posture and time of day. Although assumption of the recumbent position during immersion was associated with a gradual increase in $U_{Na}V$, the resultant increase did not differ from the increase in $U_{Na}V$ following the assumption of recumbency during control. These data support the hypothesis that the natriuresis of water immersion is mediated by an immersion-induced hydrostatic pressure gradient acting on the vascular beds of the lower extremities and body trunk.

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