

VOLUME REGULATION IN MAN DURING NECK-OUT IMMERSION

IN A MEDIUM WITH HIGH SPECIFIC GRAVITY (DEAD SEA WATER)

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ABSTRACT

The effect of immersion for 4 hours on arterial blood pressure and the rate of urinary sodium excretion was studied in the same five subjects both in fresh water and in Dead Sea water (specific gravity 1.19) at 34 C. Following 100 min of immersion, mean systolic and diastolic blood pressure decreased ($P < 0.01$) in subjects immersed in fresh water, and increased ($P < 0.05$) in those immersed in Dead Sea water. While immersion in fresh water led to hypotension, it was associated with an increased natriuresis. In contrast, the hypertensive response to immersion in Dead Sea water was not associated with an increased natriuresis. It is concluded that under the unique conditions of this experiment, urinary excretion of sodium becomes independent of systemic arterial blood pressure, and is presumably governed by neurohumoral influences originating in the baroreceptors of the low pressure system. Following completion of this study results from 2 independent investigations in Poland and in Canada showed that immersion in fresh water is associated with a distinct hypotension effect.

The pioneering extensive studies of Gauer and Henry (1) and Epstein (2) have shown that neck-out immersion in thermoneutral fresh water is associated with natriuresis and diuresis, which led to a negative salt and water balance. The afferent cause of this response to immersion is the redistribution of blood volume from the periphery into the great intrathoracic blood vessels. This central hypervolemia stimulates the cardiopulmonary baroreceptors, that in turn, initiate a neurohumoral efferent chain of events that signals the kidney to increase salt and water excretion (1,2).

The effect of the specific gravity (sp gr) of the immersion medium on volume regulation has not been previously reported. We describe here the effect of immersion in a hyperconcentrated medium on arterial blood pressure and sodium balance.

PATIENTS AND METHODS

Five healthy male volunteers ranging in age between 25 and 45 years participated in this study. Their diet throughout contained approximately 150 mmol sodium per day. All the experiments were conducted in the evening from 4.30 to 9.00 p.m. so as not to interfere with the daily work pattern. The participants had several training sessions before the definitive study began in order to familiarize them with the experimental procedure.

The following sessions were performed at 2 week intervals between the experiments: 1) immersion in a pool containing Dead Sea water kept thermostatically at 34 C; 2) immersion in fresh water under the same conditions; and 3) sitting in an empty pool.

At 4.30 p.m. each subject voided urine and drank 700 ml of tap water. They then sat outside the pool for 30 min (basal condition period). At the end of this period urine was collected and the subjects entered the water in the pool and sat immersed to the neck. Every hour they drank an additional 200 ml water throughout the 4 hours of immersion. Urine collections were obtained by voiding every hour, and venous blood was collected at the beginning and the end of the session. Systemic arterial blood pressure was taken by the same experienced observer, using a clinical cuff mercury manometer before, and at 100 and 160 min of immersion. During measurement of blood pressure the test subject rose from the immersion pool so that the water reached the level of the nipples. He extended his left arm to 90 so that an observer outside the pool could perform manometry on the arm held above water level. Subjects were weighed before and after immersion.

The immersion in Dead Sea water required the wearing of lead weights similar to those used during diving (approximately 6 kg per person). Unfortunately, due to an oversight, we did not place the weights on the subjects during the control procedure or during the immersion in fresh water. All subjects tolerated the procedure well, except for one who had an episode of orthostatic hypotension after 4 hours of immersion in Dead Sea water.

All blood specimens were analyzed for hematocrit, osmolality, potassium, sodium, calcium, magnesium, blood urea nitrogen and creatinine. All urine specimens were analyzed for osmolality and sodium. Blood and urine chemistry were performed according to standard techniques.

RESULTS

All results are presented as mean \pm SEM unless otherwise indicated.

Immersion for 4 hours in either fresh or Dead Sea water did not produce change in any of the following: hematocrit, blood urea nitrogen, blood creatinine, calcium, magnesium, sodium and potassium concentration and plasma osmolality.

The changes in weight during the three experimental procedures were (preimmersion weight minus postimmersion weight): 1) during immersion in fresh water 1.4 ± 0.4 kg; 2) during immersion in dead Sea water 0.3 ± 0.1 kg; 3) during sitting 0.0 kg.

Before the study began, 24 hours urinary sodium excretion was 155 ± 10 and 192 ± 24 mEq in Dead Sea immersion and fresh water, respectively.

Fig. 1 describes changes in systolic arterial blood pressure, the rate of urinary sodium excretion and free water clearance. The statistical significance refers to data obtained during immersion in fresh water, vs. data obtained during immersion in Dead Sea water (mean \pm SD). It can be seen that at 100 min of immersion in fresh water, systolic blood pressure falls, whereas urinary excretion of salt and water rises. In contrast, during immersion in Dead Sea water, systolic blood pressure rises, but there is no increased natriuresis and only modest diuresis.

After 100 min of sitting, there was no significant change in blood pressure and natriuresis declined.

Mean diastolic blood pressure at 100 min decreased by 7.0 ± 2.0 mm Hg during immersion in fresh water and increased by 8.3 ± 0.3 mm Hg during immersion in Dead Sea water ($P < 0.05$). Thus, the changes in diastolic pressures during immersion paralleled those seen in systolic pressures.

During the immersion all subjects showed a tendency to bradycardia, which was greatest in the Dead Sea water (decrease in pulse rate of 16.0 ± 3.5). The difference between the means of the decreases in blood pressure among the three procedures was not, however, statistically significant.

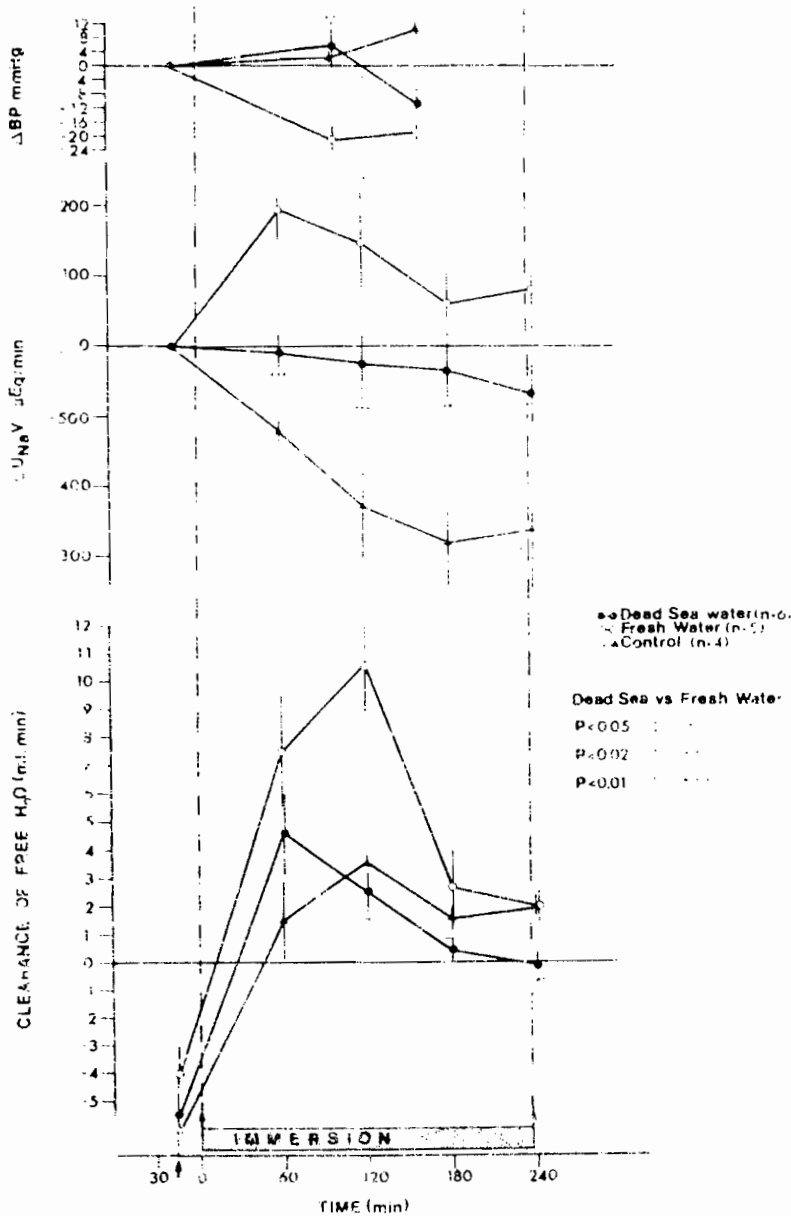


Fig 1 Influence of neck-out immersion on systolic blood pressure (ΔBP) (top panel), rate of urinary sodium excretion (U_{NaV}) (middle panel) and clearance of free water (bottom panel). Note a fall in blood pressure and an increase in natriuresis and diuresis during immersion in fresh water. In contrast, immersion in Dead Sea water results in an increase in blood pressure not associated with increased natriuresis, and is associated only with moderately increased diuresis. Quiet sitting is not associated with changes in blood pressure or in increased natriuresis. Results are expressed as mean \pm SD.

DISCUSSION

Under ordinary experimental conditions (in non-immersed subjects), the rate of urinary sodium excretion (U V) is directly correlated with arterial blood pressure (pressure natriuresis) (3). The unique conditions of our present study dissociate this relationship. Thus, the hypotensive response to immersion in fresh water is associated with an increase in U V, whereas the hypertensive response to immersion in Dead Sea water is not associated with increased U V.

Our study does not clarify the mechanism of the opposite pressor responses in the two immersion media used or their paradoxical influence on U V. However, previous studies by others (4-6) allow the following speculation: the central hypervolemia of immersion in fresh water activates the cardiopulmonary stretch receptors. This leads to suppression of the sympathetic nervous system, and the renin-angiotensin-aldosterone axis, and to a decrease in plasma arginine vasopressin levels. All these would tend to lower systemic blood pressure, and enhance diuresis and natriuresis.

It is interesting to note that a similar fall in systemic blood pressure with an increase in U V is seen in another example of central engorgement, namely, the inflation of a balloon in the left atrium of a dog (7). Also, the classic study of De Wardener et al. (8) in the dog clearly demonstrated that the U V may increase during volume expansion in the face of reduction of renal perfusion pressure and lowering of the glomerular filtration rate.

Information on the effect of immersion on arterial blood pressure is scant and conflicting. Epstein stated in 1978 that "immersion in fresh water was associated with significant albeit slight decrease in mean arterial blood pressure" (9). A later study by the same author (4) also showed a decrease in mean arterial pressure during immersion in fresh water in, at least, certain individuals. Arborelius et al. (5), utilizing intraarterial manometry, found that immersion caused a mean increase in arterial pressure of 10 mm Hg. This was, however, less than the increase of pressure of 15 to 20 mm Hg in the ambient water surrounding the immersed arm in which the brachial artery was cannulated for the manometry. Moreover, their study was short and did not last the 100 min of immersion, which is when hypotension was seen in the present study. Therefore, it may be concluded that immersion in fresh water may be associated with a decrease in blood pressure.

Why hypotension has not been regularly seen during immersion may depend on the sympathetic tonus of the test subjects, on subtle environmental conditions and on the methodology of blood pressure manometry.

Immersion in Dead Sea water differs from that in fresh water by providing a 20% greater head of pressure on the body. This would increase the pressure 1 m below the neck from approximately 76 mm Hg in fresh water to approximately 91.2 mm Hg in Dead Sea water. It is postulated that this hydrostatic pressure compromises venous return and increases arteriolar peripheral vascular resistance. This would have the dual effect of increasing systemic blood pressure and abolishing the central hypervolemia of immersion.

In the absence of central hypervolemia no change will occur in the basal activity of the cardiopulmonary baroreceptors, the sympathetic nervous system, the renin-angiotensin-aldosterone axis and the levels of plasma arginine vasopressin. The net result would be hypertension without natriuresis and diuresis during immersion in Dead Sea water.

We have shown that the specific gravity of the immersion medium has a profound influence on arterial blood pressure and on U V. During immersion, natriuresis and diuresis become independent of systemic arterial blood pressure. It is postulated that neurohumoral influences originating in the lesser circulation predominate over any effects of systemic blood pressure on urinary sodium excretion. The suggestion that the low pressure baroreceptors are more sensitive to moderate changes in blood volume than are high pressure baroreceptors is supported by theoretical considerations (1), as well as by human experiments, in which design and methodology were entirely different (6,10) from those employed in our study.

Since a preliminary report of the present study had been published (Ish-Shalom and Better, Isr. J. Med. Sci. 20: 109-112, 1984) additional information became available concerning the effects of immersion on systemic hemodynamics and of renal function. Thus it is now known that immersion increases both blood level of ANF as well as the urinary excretion of ANF second messenger cyclic guanosine monophosphate (cGMP) (reviewed in ref. 11) in parallel with the natriuresis MAP decreased during immersion in normal controls as well as in patients with cirrhosis (11). This immersion induced hypotension was statistically significant (11). These findings explain both the natriuresis of the hypotension of immersion since exogenous ANF is known to cause peripheral vasodilation even in non immersed persons and experimental animals. Such vasodepressor influence of ANF would be enhanced by the suppression of at least 3 major constrictor-antinatriuretic and anti diuretic systems during immersion.

In additional investigations in Poland on the hypotensive effects of immersion in near term pregnant women (12) 39 healthy women as well as 45 pregnant women with late toxemia, immersion caused a distinct and statistically significant decrease of both systolic and diastolic pressures within an hour of immersion. The absolute decrease in mean systolic and diastolic blood pressure during immersion in women with toxemia were 19 and 13 mm Hg respectively and this hypotension was associated with bradycardia (12).

Thus independent studies from Poland, Canada and Israel found that immersion decreased arterial blood pressure.

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