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Urinary excretion of water and electrolytes during open-sea saturation diving to 850 fsw

T. S. NEUMAN, R. F. GOAD, D. HALL, R. M. SMITH, J. R. CLAYBAUGH, and S. K. HONG

Submarine Development Group One, San Diego, CA 92132; Departments of Physiology, University of Hawaii, Honolulu, HI 96822, and State University of New York at Buffalo, Buffalo, NY 14214; and Tripler Army Medical Center, Tripler AMC, HI 96859

Neuman, T. S., R. F. Goad, D. Hall, R. M. Smith, J. R. Claybaugh, and S. K. Hong. 1979. Urinary excretion of water and electrolytes during open-sea saturation diving to 850 fsw. Undersea Biomed. Res. 6(3):291–302.—The dive was carried out in the open sea to a depth of 850 fsw (26.7 ATA) for 6 days (DD 1–6) in the saturated mode, with personnel transfer capsule (PTC) excursions between 0 and 150 fsw and diver excursions between 0 and 50 fsw from the saturation base. Each diver had two excursion dives on alternate days. Although each PTC excursion lasted approximately 7 h, the actual time spent in the water averaged 10.5 min per diver. For 12 divers, daily excretion of water, electrolytes, aldosterone, and antidiuretic hormone (ADH) was studied, along with plasma composition (including prolactin), before, during, and after hyperbaric exposure. A significant increase in urine flow was observed on DD 2–4 (1604 ml/day predive vs. 2300 ml/day on DD 4; $P < 0.05$), after which the degree of diuresis decreased to about 1800 ml/day. Urine osmolality changed inversely with urine flow, with the lowest value of 532 mOsm/kg on DD 4. During the postdive period, both urine flow and urine osmolality returned to the predive level. The endogenous creatinine clearance was maintained at about 200 liters/day throughout the dive. The fractional excretion of Na^+ remained unchanged while that of K^+ increased significantly during hyperbaric exposure, thus decreasing the urinary Na^+/K^+ ratio. The fractional excretion of total osmotic substances showed a small but consistent increase. The plasma protein concentration consistently increased during hyperbaric exposure. Body weight decreased progressively during the initial 4 days of pressure exposure, equalling 2.6 kg on DD 4. These findings suggest that the observed diuresis may be accompanied by a net loss of body water. Neither the plasma prolactin level nor urinary excretion of aldosterone and ADH showed any consistent change throughout the dive. It thus appears that, although there is a small osmotic component, the observed diuresis is primarily due to the ADH-independent inhibition of free water reabsorption from the collecting duct by means of a mechanism yet to be identified.

aldosterone
antidiuretic hormone (ADH)
hyperbaric diuresis
prolactin

A significant increase in urine flow has been observed in the majority of saturation diving experiments carried out in the past (see the review by Hong (1975)). Although the mechanism(s) underlying this hyperbaric diuresis is not clearly understood, it is known that increased fluid intake is not responsible for this phenomenon. Based on comprehensive studies conducted during the Hana Kai II dive, Hong, Claybaugh, Frattali, Johnson, Kurata, Matsuda, McDonough, Paganelli, Smith, and Webb (1977) proposed that the primary mechanism for the hyperbaric diuresis is suppression of antidiuretic hormone (ADH) as a result of suppression of insensible water loss. However, these authors also recognized that secondary mechanisms such as psychological stress, the osmotic gas effect, and the effect of breathing a denser gas may alter the basic nature of diuresis during the early phase (first several days) of diving.

The present investigation was undertaken to study the pattern of diuresis during an open-sea saturation dive (6 days at 850 fsw), which involves several stress factors in addition to those found in dives. Particular emphasis was placed on the daily changes in serum composition and in urinary excretion of water, solutes, ADH, and aldosterone to reexamine the mechanism responsible for the diuresis during the early phase of diving.

METHODS

Dive profile and environmental parameters

The present investigation was carried out in June, 1976 during the U.S. Navy-sponsored Elk River (IX-501) DDS MK2 MOD O Dual Complex Saturation Dive. The dive was carried out in the open sea to a depth of 850 fsw (257 m) in the saturated mode with personnel transfer capsule (PTC) excursions between 0 and 150 fsw and diver excursions between 0 and 50 fsw from the saturation base. After compression to 26.7 ATA (850 fsw) on Dive Day (DD) 1, the divers spent the next 5 days (DD 2–6) inside the chamber (26.7 ATA) with PTC/diver excursion dives to 950 fsw (29.8 ATA) in the water column. The overall dive profile is shown in the bottom panel of Fig. 1. Each diver had two excursion dives on alternate days. Although it took approximately 7 h for each PTC excursion, the actual time spent in the water averaged 10.5 min per diver per excursion.

Compression was initiated with air at a rate of 60 ft/min to 1.6 ATA, at which point all instruments were checked. Compression was resumed with 100% He at a rate of 5 ft/min to approximately 2.5 ATA, where another systems check was performed. Compression with 100% He then continued until the chamber pressure reached 26.7 ATA. The compression phase required 15 h (0700–2155 h of DD 1). The chamber atmosphere at 26.7 ATA consisted of 0.35–0.40 ATA O_2, 1.28 ATA N_2, and the balance He. The partial pressure of CO_2 was less than 0.005 ATA (3.8 torr). The chamber temperature was maintained at 29–30°C, with a relative humidity of 50–70%.

Subjects

Twelve U.S. Navy divers served as subjects; their physical and some physiological characteristics are shown in Table 1.

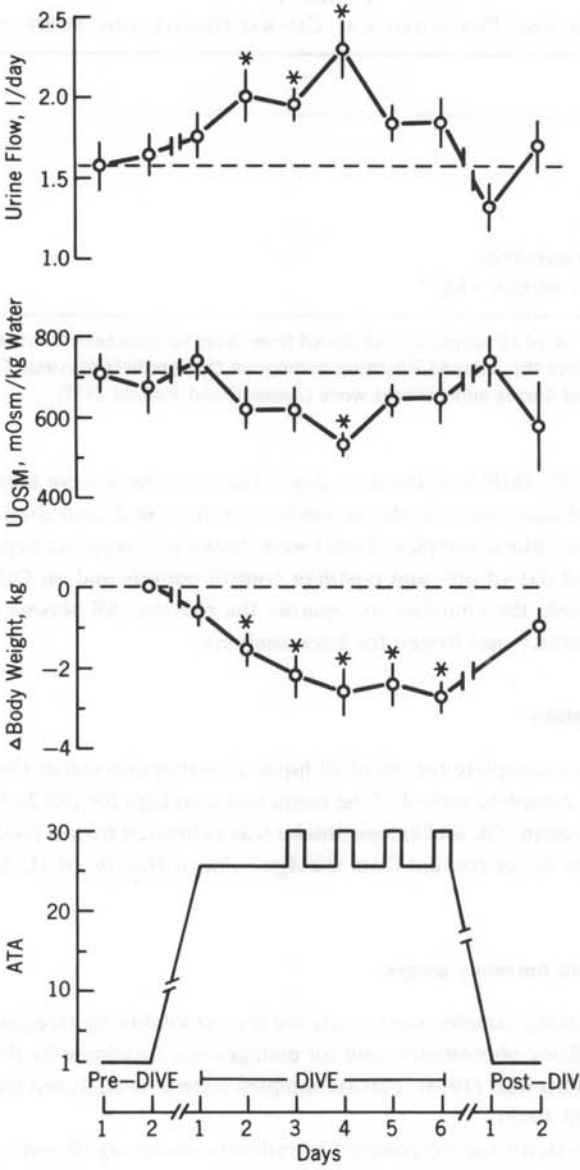

Fig. 1. Dive profile and daily urine flow, urine osmolality, and changes in body weight before, during, and after the dive. In the figures in this paper, each point represents mean from 12 subjects, and vertical bars indicate ± 1 SE; asterisks indicate significant differences from corresponding pre-dive control values ($P < 0.05$).

Urine and blood collection

Twenty-four hour urine samples were collected during 2-day predive control periods, 6 day hyperbaric (DD 1–6) periods and 2-day postdive control periods. Each subject freely voided into a separate container during and at the end of each of four intervals (0601–1000, 1001–

TABLE 1
PHYSICAL AND PHYSIOLOGICAL CHARACTERISTICS OF SUBJECTS

Characteristic	Value
Age, years	29.4 ± 1.1
Height, cm	174.7 ± 1.9
Weight, kg	84.5 ± 2.5
Body fat, % weight*	22.5 ± 1.3
Resting heart rate, beats/min	73.1 ± 3.1
Maximal O_2 uptake, ml/min •kg**	36.9 ± 2.3

Values are means ± SE of 12 subjects; *calculated from skinfold thickness (Durnin and Rahaman 1967); **determined from the Åstrand-Rhyming nomogram that predicts maximal O_2 uptake from the heart rate measured during submaximal work (Åstrand and Rodahl 1977).

1500, 1501–2300, 2301–0600 h of the next day). The containers were then transferred to the surface at the end of each interval, the volumes were recorded, and aliquots were frozen for later analyses. Venous blood samples (20 ml) were drawn into separate heparinized syringes at 0600 h on the second day of pre- and postdive control periods and on DD 2–6. The samples were centrifuged inside the chamber to separate the plasma. All plasma samples were then transferred to the surface and frozen for later analyses.

Water and caloric intake

Each subject kept a complete record of all liquid consumption (other than that contained in food) at pressure. A complete record of the menu was also kept for DD 2–5. From this record, the carbohydrate, protein, fat, and caloric intake was estimated from a food table (Church and Church 1975) and the water content from the *Agricultural Handbook* (U.S. Dept. of Agriculture 1963).

Chemical analysis and hormone assays

Both urine and plasma samples were analyzed for osmolality by freezing point depression, for Na^+ and K^+ by flame photometry, and for endogenous creatinine by the method of Owen, Iggo, Scandrett, and Stewart (1954). Plasma samples were also analyzed for proteins (Gornall, Bardawill, and David 1949).

Plasma level of prolactin was determined by radioimmunoassay (Bryant, Siler, Greenwood, Pasteels, Robyn, and Hubinont 1971). Urinary aldosterone was determined by using a New England Nuclear Aldosterone (^3H) Radioimmunoassay Pak. For each assay a recovery was calculated (means 65%). Aldosterone values were then corrected for the corresponding recovery data. Urinary ADH was determined by the extraction method and radioimmunoassay technique of Miller and Moses (1972). Both the sensitivity of the method and the preparation and specificity of the antibody have been reported (Hong et al. 1977).

Statistical analysis

The mean and standard error were calculated for each day for all measurements, and all data were compared by nonpaired t tests. A difference was considered significant if $P < 0.05$.

RESULTS

Urine flow and solute excretion

The average urine flow during the predive control period was 1604 ml/day; average urine osmolality was about 700 mOsm/kg. On the compression day (DD 1), urine flow increased to 1753 ml/day, which was not significantly different from the predive control level. A significant increase in urine flow was observed on DD 2–4, with the peak level of 2300 ± 176 ml/day on DD 4 (Fig. 1). The degree of diuresis decreased to about 1830 ml/day on DD 5 and 6. In general, urine osmolality changed inversely with urine flow, with the lowest value of 532 ± 32 mOsm/kg occurring on DD 4 ($P < 0.01$ compared to the predive value). During the postdive control period, both urine flow and urine osmolality returned to the predive control level.

Note the progressive decrease in body weight during the initial four days of pressure exposure, equalling 2.6 kg (5.7 lbs) on DD 4, after which there was no further reduction (Fig. 1). Body weight returned to the predive level by the end of the decompression period.

The plasma clearance of endogenous creatinine (C_{cr}) was maintained at about 200 liters/day throughout the dive (Fig. 2). A significant increase ($P < 0.05$) of the daily excretion of solutes was observed on DD 1 (Fig. 2). In general, the daily excretion of Na^+ was maintained at about 200 mEq/day, without any consistent pattern, whereas the daily excretion of K^+ showed a significant increase at pressure and a significant decrease during the postdive period (Fig. 2). Therefore, the urinary Na^+/K^+ ratio decreased from the predive control value of 3.0 to approximately 2.0 during most of the hyperbaric exposure (Fig. 3). Although the osmolal clearance (C_{osm}) increased slightly at depth ($P < 0.05$ on DD 4), the negative free water clearance ($T^c_{H_2O}$) remained largely unchanged throughout the dive (Table 2). Hence, as significant reduction in standardized negative freewater ($C_{osm} T^c_{H_2O}$) was noted on DD 4 ($P < 0.05$).[1]

To evaluate tabular handling of solutes, the C_{osm}/C_{cr} ratio (the fractional excretion of filtered osmotic particles) was calculated for each day, and the results are shown in Fig. 3. These data indicate small but consistent increases in the fractional excretion of filtered osmotic particles from the predive control value of 1.80 ± 0.08% to 2% or above during hyperbaric exposure. The increase achieved statistical significance on DD 6. The fractional excretion of the filtered K^+ increased from 9.1 ± 0.42% during the predive control period to 13.0 ± 0.87% on DD 2 ($P < 0.005$), after which it decreased continuously, reaching the control level on DD 6. The fractional excretion of the filtered Na^+ was constant at about 0.7% throughout the dive.

Plasma chemistry

The changes in plasma osmolality as well as in concentrations of Na^+, K^+, and protein are shown in Fig. 4. The plasma osmolality at depth was significantly lower than the predive value but was not different from the postdive level. Reductions in plasma Na^+ concentration were seen on DD 4–6. No consistent trend was noted in plasma K^+ concentration. In contrast, the plasma protein concentration consistently increased during hyperbaric exposure, with the peak value of 8.1 g % on DD 3 ($P < 0.05$).

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[1] This reduction in standardized negative free water clearance should be interpreted with caution, since the measurement was made under conditions where concentration of the urine was not maximal. In case of dehydration (or in the presence of exogenous ADH), an increase in urine flow is accompanied by an increase in $T^c_{H_2O}$ until the latter reaches a maximum at a urine flow of approximately 8 liters/day (Smith 1956). In the present study, the absolute values of $T^c_{H_2O}$ remained unchanged (Table 2) during the dive despite the increase in urine flow from 1.6 liter/day (pre-dive) to about 2 liter/day (at depth). Since the urinary ADH excretion also remained unchanged throughout the study, it is reasonable to conclude that the free water reabsorption is decreased at depth.

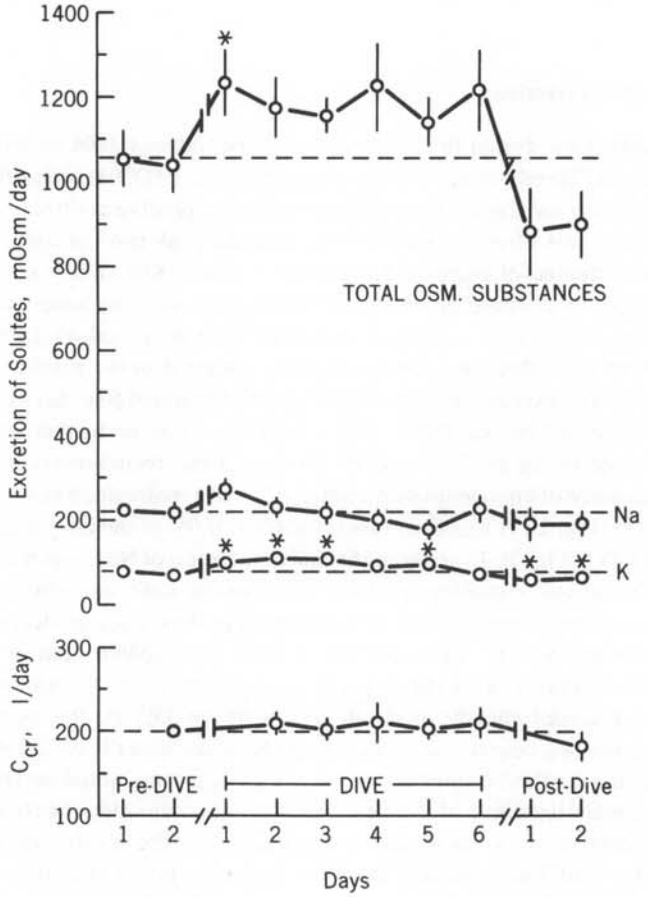

Fig. 2. Endogenous creatinine clearance (C_{cr}) and daily excretion of solutes before, during, and after the dive. (See Fig. 1 legend for details.)

Endocrine changes

The plasma level of prolactin and the daily excretion of aldosterone and ADH showed only random changes that are not correlated with the diuresis. During the early phase of the dive, there were significant increases in plasma prolactin (from predive value of 3.49 ± 0.42 ng/ml to 5.22 ± 0.50 on DD 3, $P < 0.05$) and urinary excretion of ADH (from predive value of 20.75 ± 2.11 mU/day to 32.05 ± 4.29 on DD 2, $P < 0.05$), and during the postdive period, the urinary excretion of aldosterone showed a significant decrease (from predive value of 6.24 μg/day to 4.29 ± 0.56 postdive, $P < 0.05$).

Water intake

The average daily intake of liquid recorded by each subject during hyperbaric exposure is shown in Table 3, along with the water content of food and the water of oxidation, based on the menu. Since no correction was made for the non-ingested portion of the food, the latter values are approximations only. Nevertheless, it should be noted that the average total water

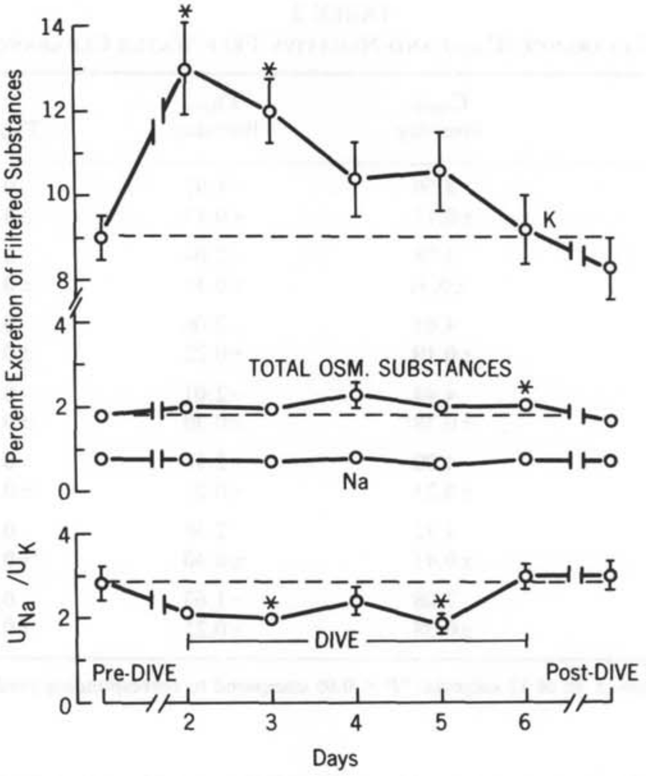

Fig. 3. Fractional excretion of filtered solutes and urinary Na^+/K^+ ratio before, during, and after the dive. (See Fig. 1 legend for details.)

intake (for DD 2–5) of 2947 ml/day is remarkably similar to the value obtained in very carefully carried out studies such as the Hana Kai II dive (Hong et al. 1977).

DISCUSSION

An open-sea saturation dive has many logistical limitations for scientific experiments, and certain scientific information needed to interpret the basic data is therefore lacking in the present work. Despite these limitations, the information obtained is extremely valuable because the experimental conditions represent a real diving situation, which may be quite different from simulated diving.

In the present dive, 12 divers spent most of their time inside the chamber at 850 fsw for six days before decompression. Although each diver participated in two PTC/diver excursion dives to a depth of 950 fsw in the open sea, the actual time spent in the wet sea environment averaged only about 10 min. Therefore, the results obtained in the present study may be primarily attributable to both the dry, hyperbaric condition and an unknown degree of additional stress (physiological and psychological) imposed by actual open ocean diving.

As in most previous saturation diving experiments, a hyperbaric diuresis was observed in the present dive (Fig. 1). However, in contrast to an earlier simulated dive (Hana Kai II, Hong et al. 1977) that showed a marked increase in urine flow 6 h after the start of compression (at about 10 ATA), no significant diuresis was observed until the second day of hyperbaric exposure in the present dive.

TABLE 2

OSMOLAL CLEARANCE (C_{OSM}) AND NEGATIVE FREE WATER CLEARANCE ($T^c_{H_2O}$)

Dive Period	C_{OSM}, liter/day	$T^c_{H_2O}$, liter/day	$T^c_{H_2O}/C_{osm}$
Predive	3.56 ±0.17	−1.97 ±0.17	0.55 ±0.035
DD 2	3.98 ±0.31	−2.04 ±0.30	0.49 ±0.058
DD 3	4.01 ±0.19	−2.06 ±0.22	0.50 ±0.042
DD 4	4.44 ±0.38*	−2.01 ±0.30	0.44 ±0.040*
DD 5	4.00 ±0.23	−2.17 ±0.23	0.52 ±0.036
DD 6	4.32 ±0.41	−2.38 ±0.40	0.53 ±0.055
Postdive	3.08 ±0.28	−1.63 ±0.25	0.51 ±0.041

Values are means ± SE of 12 subjects; *$P < 0.05$ compared to corresponding predive control.

In the Hana Kai II dive, the sudden appearance of diuresis during the early phase of compression was accompanied by a reduction of the thoracic conductive volume (an index of thoracic fluid volume), which had been continuously increasing since the onset of compression (Smith, Hong, Dressendorfer, Dwyer, Hayashi, and Yelverton 1977). Based on this observation, Smith et al. (1977) speculated that an increase in the central blood volume somehow triggers the diuresis. In the absence of data on the thoracic conductive volume in the present work, we cannot contest this speculation.

The diuresis reached a peak on DD 4, after which it attenuated (Fig. 1). Moreover, body weight decreased progressively until DD 4 and leveled off thereafter. During the first 4 days at pressure, the body weight decreased by 2.6 kg while the cumulative urinary water loss over and above the predive level amounted to 1.6 liters. Since the daily water intake appeared to be reasonably constant during this period (Table 3), the observed reduction in body weight may reflect a net loss of body water and fat. There was indeed a consistent increase in plasma protein concentration (Fig. 4), suggesting a state of dehydration. Such a greater diuresis accompanied by a dehydration was also observed during the early phase of the Hana Kai II dive (Hong et al. 1977). The mean daily caloric intake provided by the menu for DD 2–4 was calculated to be 2430 kcal. It is possible that the daily energy expenditure is higher than the energy intake during this period, resulting in a negative energy balance. Even when the daily caloric intake was greater than the daily energy expenditure, a small loss of body mass apart from water loss was observed in the Hana Kai II dive (Webb, Troutman, Frattali, Dressendorfer, Dwyer, Moore, Morlock, Smith, Ohta, and Hong 1977).

There are also other similarities between the characteristics of the diuresis observed in the present dive and those seen during the early phase (first 4 days) of the Hana Kai II dive. These include:

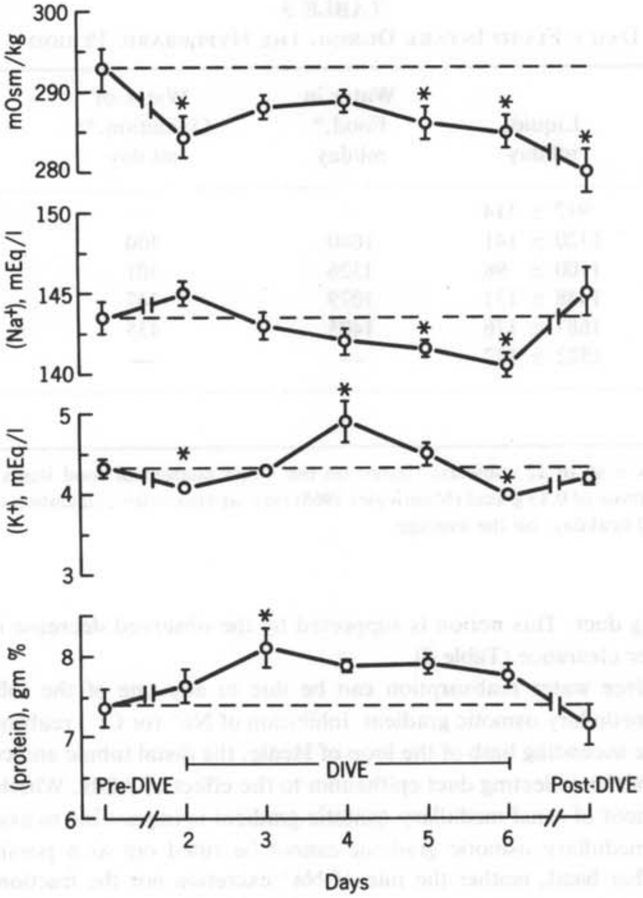

Fig. 4. Plasma composition before, during, and after the dive. (See Fig. 1 legend for details.)

1) no significant change in the glomerular filtration rate (as estimated by endogenous creatinine clearance);

2) the diuresis was accompanied by a reduction in urine osmolality (but was still hypertonic);

3) a tendency for a small increase in the fractional excretion of total osmotic substance;

4) the fractional excretion of Na^+ was unchanged while that of K^+ increased significantly; and

5) no significant change was found in the urinary excretion of ADH. In the Hana Kai II dive, there was a significant reduction in plasma prolactin level, which was suggested to be responsible for the early diuresis (Hong et al. 1977). In contrast, the plasma prolactin level in the present dive remained largely unchanged during the hyperbaric exposure. This implies that prolactin does not play a role in the development of early diuresis in the hyperbaric environment. Since the diuresis was not accompanied by an increase in the plasma clearance of Na (the major osmotic component of extracellular fluid), the observed increase in C_{osm} is probably due to an inhibition of urea reabsorption secondary to inhibition of free water reabsorption

TABLE 3
DAILY FLUID INTAKE DURING THE HYPERBARIC PERIOD

Dive Days	Liquid, ml/day	Water in Food,* ml/day	Water of Oxidation,** ml/day	Total, ml/day
1	917 ± 114	—	—	—
2	1320 ± 141	1040	300	2660
3	1100 ± 98	1326	301	2727
4	1448 ± 131	1079	347	2874
5	1687 ± 176	1405	435	3527
6	1522 ± 137	—	—	—
Average				2947

Values are means ± SE of 12 subjects; *based on the water content of food items (see METHODS); **calculated on the basis of 0.13 g/kcal (Muntwyler 1968) (see METHODS for calculation of caloric intake). Menu provided 2660 kcal/day, on the average.

from the collecting duct. This notion is supported by the observed decrease in standardized negative free water clearance (Table 2).

A decrease in free water reabsorption can be due to any one of the following causes: washout of renal medullary osmotic gradient, inhibition of Na^+ (or Cl^-) reabsorption from the distal nephron (the ascending limb of the loop of Henle, the distal tubule and collecting duct), or refractoriness of the collecting duct epithelium to the effect of ADH. Whether or not there was a partial washout of renal medullary osmotic gradient is impossible to assess, and therefore washout of medullary osmotic gradient cannot be ruled out as a possible factor (see below). On the other hand, neither the rate of Na^+ excretion nor the fractional excretion of filtered Na^+ showed any consistent change throughout the dive, and hence it is not likely that the inhibition of Na^+ (or Cl^-) reabsorption from the distal nephron is responsible for a decrease in free water reabsorption. There are at least three factors known to interfere with the hydrosmotic action of ADH at the collecting duct level: catecholamines, cortisol, and prostaglandin E (PGE) (Schrier, Berl, Anderson, and McDonald 1977). In fact, Raymond, Sode, and Leach (1975) suggested that the diuresis they observed during a saturation dive at 50 ATA may be due to the increased release of catecholamines. An increase in the urinary excretion of cortisol has also been observed during exposure to 4 ATA (Leach, Alexander, Fischer, Lambertsen, and Johnson 1973). However, PGE excretion during hyperbaric exposure has never been studied, to our knowledge. If indeed the hydrosmotic action of ADH is inhibited, this would secondarily lead to an increase in urea excretion and a washout of the medullary osmotic gradient.

The plasma osmolality decreased at depth compared to the predive level, but failed to return to the predive level after the dive (Fig. 4). In contrast, in Hana Kai II, a significant reduction in plasma osmolality, which returned to the predive level after the dive (Hong et al. 1977), was noted after 10 days at depth. Such a slow reversible reduction in plasma osmolality in the Hana Kai dive was attributed to the suppression of insensible water loss in a hyperbaric environment (Hong et al. 1977; Paganelli and Kurata 1977) in the face of a constant water intake. However, the observed reduction in plasma osmolality in the present dive cannot be attributed to the suppression of insensible water loss because it not only appeared too early but was also irreversible. It is likely that the predive level of plasma osmolality was too high for some unknown reason(s), and that the postdive plasma osmolality represents a more acceptable control level. We therefore interpret the present data to mean that there was no significant change in plasma osmolality.

The urinary excretion of aldosterone remained unchanged throughout the dive despite the diuresis, which led to a state of dehydration. However, a significant increase in both the plasma aldosterone level and the urinary excretion of aldosterone was observed during the first week of the Hana Kai II dive (Hong et al. 1977). Note, however, that the magnitude of diuresis was much greater in the latter dive compared to the present dive. It is thus possible that the degree of dehydration in the present dive may not be large enough to induce a significant stimulation of the renin-angiotensin-aldosterone system.

A slight but significant increase in the urinary excretion of K^+ was observed during the hyperbaric period (Fig. 2), in agreement with the results obtained in most previous dives (Hong 1975). This phenomenon appears to be due to an increase in the tubular secretion of K^+, indicated by the corresponding change in the fractional excretion of K^+ (Fig. 3). In view of the fact that the urinary excretion of aldosterone did not change during the hyperbaric period (Table 2), the permeability of the distal tubule to K^+ may be increased in a hyperbaric environment.

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Neuman, T. S., R. F. Goad, D. Hall, R. M. Smith, J. R. Claybaugh, and S. K. Hong. 1979. L'excrétion urinaire de l'eau et des électrolytes pendant une plongée à saturation en pleine mer à 850 fsw. Undersea Biomed. Res. 6(3):291–302.—La plongée a été mise à exécution en pleine mer à une profondeur de 850 fsw (26,7 ATA) pour 6 jours (DD 1–6) d'une mode saturée, avec les sorties de la capsule de transfer personel (CTP) entre 0 et 150 fsw et les sorties de plongeur entre 0 et 150 fsw de la base de saturation. Chaque plongeur a fait deux plongées excursionaires sur les jours alternatifs. Bien que chaque excursion de CTP ait duré environ 7 h, le temps actuel passé dans l'eau a été en moyenne de 10,5 min pour chaque plongeur. Pour les 12 plongeurs, l'excrétion journalière d'eau, d'électrolytes, d'aldosterone, et d'hormone antidiuretique (HAD) ont été examinés, et aussi la composition du plasma (y compris le prolactin), avant, durant, et après l'éxposition hyperbare. Une augmentation significante du flux de l'urine a été observé le DD 1–4 (1604 ml/jour avant-plongée vs. 2300 ml/jour le DD 4; $P < 0.05$), après que la degré du diurèse a diminué à environ 1800 ml/jour. L'osmolalité de l'urine a changé inversement avec le flux de l'urine, avec la valeur la plus basse de 532 mOsm/kg le DD 4. Pendant la durée après-plongée, le flux de l'urine et l'osmolalité de l'urine sont revenus, tous les deux, aux niveaux de l'avant-plongée. Le dégagement endogène creatinin a été maintenu environ 200 litres/jour pendant la durée de la plongée. L'excrétion fractionelle de Na^+ est resté la même, pendant que celle de K^+ a augmenté significativement durant l'éxposition hyperbare, ainsi diminuant le ratio urinaire Na^+/K^+. L'excrétion fractionelle des substances osmotiques totales a montré une augmentation petite, mais consistante. La concentration de la protéine du plasma a augmenté en consequence pendant l'éxposition hyperbare. La pesanteur du corps a diminué progressivement durant les 4 jours initials de l'éxposition à une pression égalant 2–6 kg le DD 4. Ces résultats suggérent que le diurèse observé peut être accompagné d'une perte nette de l'eau corporelle. Ni le niveau du plasma prolactin ni l'excrétion urinaire d'aldosterone de HAD a montré un changement consistant pendant la duration de la plongée. Il semble ainsi que, bien qu'il y ait un petit composant osmotique, le diurèse observé vient sûrtout de l'inhibition

HAD-indépendant de la réabsorption de l'eau douce à partir du vaisseau collecteur par un mécanisme jusqu'à présent non identifié.

aldosterone	diurèse hyperbare
hormone antidiuretique (HAD)	prolactin

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