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Characteristics of increased urine flow during a dry saturation dive at 31 ATA

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Sagawa S, Claybaugh JR, Shiraki K, Park YS, Mohri M, Hong SK. Characteristics of increased urine flow during a dry saturation dive at 31 ATA. Undersea Biomed Res 17(1):13–22.—Three male divers were subjected to a 7-day dry saturation dive at 31 ATA (New Seatopia). Urine samples were collected for measurements of electrolytes and creatinine for glomerular filtration rate (GFR) 5 times daily, at 3-h intervals during daytime (0700–2200 h) and once at night (2200–0700 h). Collections were taken for 2 days before (predive 1 ATA air), 7 days during 31 ATA exposure, and during 10 days of decompression and for 2 days at postdive 1 ATA air. Blood samples were taken after overnight fasting at each of the dive periods for measurements of atrial natriuretic factor (ANF), electrolytes, and other blood constituents. Compression to 31 ATA resulted in a twofold increase in urine flow accompanied by increases in excretion of osmotic substances (+40%) and sodium (+54%), and a reduction in urine osmolality (−32%). The increase in urine volume was greater ($P < 0.05$) at night than day with no change in GFR between day and night, confirming the earlier findings. However, no change in plasma ANF was observed in spite of a sustained increase in daily sodium and water excretion at high pressure. These results suggest that the hyperbaric diuresis-natriuresis may not be directly mediated by the ANF release.

diuresis
nocturia
natriuresis
atrial natriuretic factor

A significant increase in daily urine flow during multiday exposure to high pressure (hyperbaric diuresis) has been the focus of many investigations since its first description by Hamilton (1). Although this hyperbaric diuresis may be due to the subtle cold stress in some dives (2), it was observed even in the complete absence of cold stress (3). Moreover, neither the total body fluid volume nor the daily fluid intake changes significantly during the steady-state exposure to high pressure, despite the presence of sustained diuresis (3, 4). In general, the increase in urine flow is accompanied by a reduction in urine osmolality and insensible water loss (3, 4). The basic characteristics of this diuresis have been described as an increased osmolar clearance (Cosm) and a decreased negative free-water clearance. Certain progress has been accom-

plished regarding the mechanism of the free-water component of the diuresis. The urinary level of antidiuretic hormone (ADH) has been observed to decrease (4–7) during a sustained diuresis in the hyperbaric environment. A decreased plasma concentration of ADH has provided convincing evidence of this hormonal role in the hyperbaric diuresis (6, 8). In contrast, a hormonal involvement in the increased osmotic clearance has not been established. Early attempts to implicate the renin-angiotensin-aldosterone system and some subsequent studies have convincingly shown that this system is stimulated at hyperbaria (4–6), and therefore an increased aldosterone secretion has been proposed as a possible contributing mechanism for the increased potassium excretion. The accompanying natriuresis, however, remains unexplained.

An increase in thoracic blood volume has been proposed as an underlying mechanism of the hyperbaric diuresis at 18.6 ATA (9). If so, the distension of the atrium could release atrial natriuretic factor (ANF) (10) and possibly contribute to the well-documented natriuresis in hyperbaria. However, with a single exception (11), we know of no other report of ANF in connection with the hyperbaric diuresis and natriuresis. Moon et al. (11) concluded that increased secretion of ANF is not the only factor that mediates the diuresis seen during the compression phase of saturation dives at 46 and 61 ATA.

The present experiment, code-named New Seatopia and conducted at the Japan Marine Science and Technology Center (JAMSTEC), was designed to examine the role of ANF in the development of hyperbaric diuresis and natriuresis.

METHODS

Three healthy male volunteer divers were subjected to a 7-day dry saturation dive in a hyperbaric chamber in JAMSTEC according to a dive profile illustrated in Fig. 1. The physical characteristics of the subjects are shown in Table 1. All divers entered the chamber at 1000 h on dive Day 1. The temperature (T_a) and relative humidity (RH) inside the chamber were maintained at 26° ± 0.5°C, and 60 ± 10% during the pre- and postdive periods. At 31 ATA the chamber gas was composed of approximately 0.4 ATA O_2, 0.79 ATA N_2, less than 0.004 ATA CO_2, and helium making up the balance. The T_a and RH were maintained at 31° ± 0.5°C and 60% except for nighttime at Days 5, 6, and 7 in 31 ATA, when the T_a was lowered to 29° ± 0.5°C between 2200 and 0700 h for another experimental purpose.

Urine collection

The subjects were requested to collect their urine at 3-h intervals during the daytime (0700–2200 h) and one collection during the nighttime (2200–0700 h) throughout the dive days. Every urine sample was collected in a separate container, which was locked out at the end of each interval. The volume of urine was recorded and aliquots for various analyses were obtained. One-tenth of each sample was pooled and kept in a refrigerator until 0700 h of the next day, when daily collections were terminated. Aliquots of the pooled samples for the previous 24-h period were then frozen for later analysis.

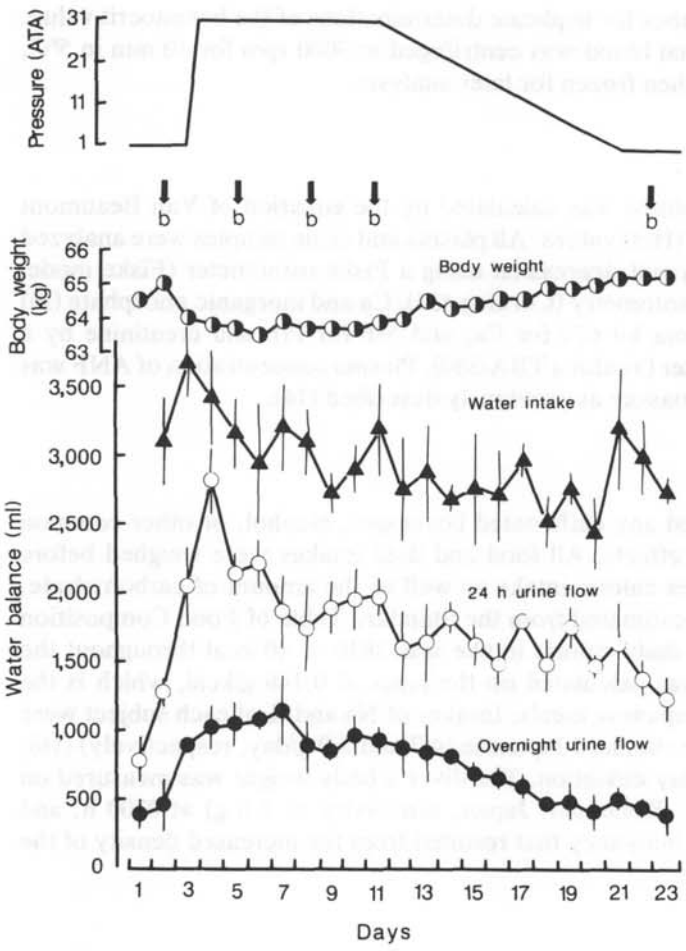

Fig. 1. Body weight, urine flow, and water intake together with dive profile. *Points* and *vertical bars* indicate means ± SE of corresponding values of body weight, daily water intake including calculated metabolic water, 24-h urine flow, and overnight urine flow in 3 subjects; *b* indicates blood sampling day.

TABLE 1
PHYSICAL CHARACTERISTICS OF THE SUBJECTS

Subject	Age, yr	Height, cm	Weight, kg	S.A., m^2
H.M.	37	166.5	63.5	1.73
T.S.	34	167.8	63.0	1.73
T.O.	21	166.5	67.5	1.77

Blood sampling

Venous blood (20 ml) was withdrawn from the antecubital vein of each subject under a fasting condition between 0700 and 0730 h of the 5 days, as shown in Fig 1. All sample tubes of 6 ml of EDTA blood, and 14 ml of heparinized blood were placed on ice in a small decompression chamber and locked out. The small chamber was decompressed slowly, as described previously (12). Portions of the heparinized blood

were drawn into capillary tubes for triplicate determinations of the hematocrit value. The remainder of the original blood was centrifuged at 3000 rpm for 10 min in 5°C, and separated plasma was then frozen for later analysis.

Chemical analysis

The change in plasma volume was calculated by the equation of Van Beaumont (13) by using the hematocrit (Hct) values. All plasma and urine samples were analyzed for osmolality by freezing point depression using a Fiske osmometer (Fiske model 2300), Na and K by flame photometry (Corning 435), Ca and inorganic phosphate (Pi) by spectrophotometer (Sigma kit 670 for Ca, and 586 for Pi), and creatinine by a Toshiba biochemical analyzer (Toshiba TBA-380). Plasma concentration of ANF was determined by radioimmunoassay as previously described (14).

Food and fluid intakes

Subjects were not allowed any caffeinated beverages, alcohol, or other common drugs with known diuretic effects. All food and fluid intakes were weighed before and after each meal, and net caloric intake as well as the amount of carbohydrate, fat, and protein intake was estimated from the Standard Table of Food Composition in Japan (15). The average daily caloric intake was 2830 ± 40 kcal throughout the dive. Water of oxidation was calculated on the basis of 0.146 g/kcal, which is the average value of standard Japanese meals. Intakes of Na and K of each subject were estimated to be those of the standard Japanese (4.7 and 2.9 g/day, respectively) (16), without significant day-to-day deviation. The diver's body weight was measured on a platform scale (type K-5, Kobekoki, Japan, sensitivity ± 1.0 g) at 0700 h, and corrected for the change in buoyancy that resulted from the increased density of the hyperbaric environment (3).

Statistical analysis

The mean and standard error for all measurements were calculated for various phases of the dive: predive, 31 ATA, decompression, and postdive. Since we used pooled urine data of 2 days for predive, 7 days for 31 ATA, 2 days for 31–25 decompression, and 2 days for postdive periods, Student's t test was used for comparing mean values of the urine data among various phases of the dive. Comparison between groups of the data for hormones and plasma compositions was performed by one-way analysis of variance followed by multiple comparisons at various time points. Significant differences for the analysis of variance were further tested using the method of Bonferroni simultaneous multiple comparisons (17). In all statistical tests a value of $P < 0.05$ was accepted as indicating significance.

RESULTS

Daily urine flow and solutes excretion

The 24-h and overnight urine flows and daily water intake during the course of the entire dive are shown in Fig. 1. Urine flow increased rapidly from 1032 ml/24 h on

average during 2-day predive, 1 ATA period to 2053 ml/24 h ($P < 0.05$) during compression and to 2827 ml/24 h ($P < 0.05$) on Day 1 at 31 ATA. The urine flow decreased slightly at Day 2 at 31 ATA, but the level was sustained at ~2000 ml/24 h during the rest period at 31 ATA. During the decompression period (31–25 ATA) urine flow was sustained at an elevated level (1700 ml/24 h, $P < 0.05$), and then returned to the predive level during postdive. The overnight urine flow markedly increased at 31 ATA and returned to predive levels gradually during decompression. Daily water intake was not increased during the stay at high pressure, ruling out increased fluid intake as a causative factor of the diuresis. Although a constant caloric intake was maintained, the average body weight decreased slightly (by ~1 kg) at 31 ATA and was restored gradually during postdive.

During the night on Days 5, 6, and 7 at 31 ATA, T_a was lowered to 29°C to examine behavioral responses of the subjects to cold stress. During these days, additional blankets were supplied. No further increase in urine flow was observed during these nights. The overnight rectal and mean skin temperatures on the nights when chamber temperature was set to 29°C were identical to those of other nights when chamber temperature was fixed to 31°C (data not shown). The results are not surprising because the subjects were sleeping under blankets and could protect themselves from the cold stress by appropriate behavioral responses.

As shown in Table 2, concomitant increases in urine flow ($P < 0.05$) and Cosm ($P < 0.05$) with a reduction of osmolality (Uosm, $P < 0.05$) were observed at 31 ATA, but there was no change in negative free water clearance and creatinine clearance (Ccr), resulting in a decrease in negative free water clearance relative to Cosm. These findings indicate that the diuresis observed in the present dive had both free water and osmotic components. The increased urinary excretion of total osmotic substances at 31 ATA was accompanied by an increase in excretion of Na ($P < 0.05$),

TABLE 2
URINARY EXCRETION OF WATER AND SOLUTES DURING VARIOUS PHASES OF DIVE[a]

	Predive 1 ATA	Saturation 31 ATA	Decompression 31–25 ATA	Postdive 1 ATA
Urine vol, ml/24h	1032 ± 140	2100 ± 105*	1797 ± 161*	1326 ± 101
Uosm, mosmol/kg	799 ± 78	541 ± 29*	611 ± 60	723 ± 83
Cosm, ml/24h	2722 ± 288	3787 ± 158*	–	3470 ± 245
Negative free water clearance, ml/24h	−1685 ± 230	−1685 ± 158	–	−2146 ± 274
Ccr, 1/24 h	154 ± 8	135 ± 4	–	166 ± 20
TOS, mosmol/24 h	782 ± 82	1097 ± 46*	1077 ± 98*	935 ± 86
Na, meq/24 h	163 ± 24	251 ± 14*	207 ± 23	228 ± 27
K, meq/24 h	43 ± 4	65 ± 3*	58 ± 4*	37 ± 1
Ca, mmol/24 h	2.0 ± 0.4	4.3 ± 0.4*	5.0 ± 1.0*	6.1 ± 0.3*
Pi, mmol/24 h	29.2 ± 2.2	39.1 ± 3.3*	30.7 ± 2.5	28.4 ± 1.9

[a]Values are means ± SE. Uosm, Cosm, Ccr, and TOS represent urine osmolality, osmolal clearance, creatinine clearance, and total osmotic substances, respectively. * $P<0.05$ vs. corresponding predive 1 ATA values.

K ($P < 0.05$), Ca ($P < 0.05$), and Pi ($P < 0.05$). Urinary excretion of these ions returned to predive levels at the postdive period; however, Ca excretion kept rising to reach a peak value in the postdive period.

Diurnal and nocturnal urine flow and solute excretion

The 24-h urine was analyzed for daytime (0700–2200 h) and nighttime (2200–0700 h) components as shown in Table 3. A significant increase in urine volume with a concomitant decrease in osmolality ($P < 0.05$) was observed at 31 ATA in both day and night. In contrast to the nocturnal increase in Na and K excretion at 31 ATA, Pi and Ca excretion increased during both day and night. The creatinine excretion did not change throughout the dive during either day or night. The nocturnal urine flow rate was greater ($P < 0.05$) than the diurnal rate at 31 ATA (Fig. 2). The Cosm at 31 ATA increased significantly ($P < 0.05$) at night compared with predive control value, but there was no significant difference between day and night. The negative free water clearance remained unchanged throughout dives at both day and night, although it tended to decrease at night.

Plasma composition

The Hct values tended to increase during the early period at 31 ATA with a return to control value during the rest of dive periods (Table 4). The increase in Hct and

TABLE 3
URINARY EXCRETIONS OF SOLUTES DURING DAY (0700–2200 H) AND NIGHT (2200–0700 H)[a]

		Predive 1 ATA	Saturation 31 ATA	Decompression 31–25 ATA	Postdive 1 ATA
Urine vol, ml	Day	609 ± 120	1098 ± 88*	888 ± 83	896 ± 98
	Night	423 ± 97	1002 ± 56*	909 ± 109*	431 ± 69
Uosm, mosmol/kg	Day	852 ± 55	644 ± 39*	695 ± 66	730 ± 87
	Night	809 ± 128	477 ± 33*	540 ± 65	767 ± 86
TOS, mosmol	Day	491 ± 73	656 ± 36	605 ± 59	624 ± 50
	Night	281 ± 16	449 ± 18*	474 ± 51*	312 ± 46
Creatinine, mg	Day	1033 ± 53	1005 ± 42	954 ± 27	1034 ± 44
	Night	641 ± 27	621 ± 26	645 ± 27	563 ± 32
Na, meq	Day	113 ± 23	150 ± 12	107 ± 13	154 ± 15
	Night	53 ± 6	101 ± 5*	107 ± 14*	84 ± 16
K, meq	Day	31 ± 5	39 ± 2	29 ± 2	23 ± 1
	Night	12 ± 2	25 ± 2*	28 ± 3*	14 ± 2
Ca, mmol	Day	1.2 ± 0.2	2.8 ± 0.2*	3.0 ± 0.4*	4.5 ± 0.2*
	Night	0.8 ± 0.1	1.2 ± 0.2*	2.2 ± 0.6*	1.6 ± 0.2*
Pi, mmol	Day	15.4 ± 1.4	23.0 ± 1.6*	18.7 ± 1.7	16.6 ± 1.0
	Night	13.1 ± 0.6	15.9 ± 1.3	12.1 ± 1.9	12.0 ± 0.9

[a]Values are means ± SE. Abbreviations as in Table 2. * $P < 0.05$ vs. corresponding predive 1 ATA values.

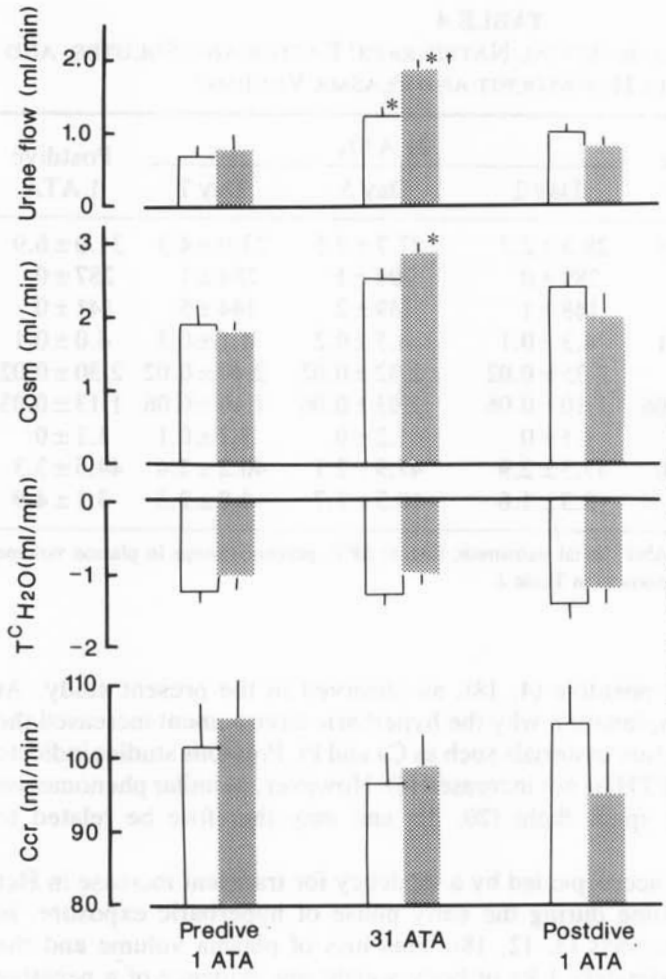

Fig. 2. Diurnal and nocturnal urine flow rate and osmolal and negative free water clearances (T^cH_2O) during various dive periods. *Open columns* and *vertical bars* represent means ± SE during daytime (0700–2200 h) and *dotted columns* and *vertical bars* represent the means ± SE during nighttime. *$P < 0.05$ vs. corresponding predive 1 ATA values, †$P < 0.05$ vs. corresponding daytime values. Abbreviations as in Table 2.

hence a decrease in plasma volume were not statistically significant because of the small number of subjects; however, these changes indicated a tendency toward dehydration at 31 ATA. No significant change in plasma electrolytes was observed throughout dive days.

Plasma hormones

Plasma levels of ANF concentration were not affected by the pressure and remained constant throughout the dive period (Table 4).

DISCUSSION

The renal responses in the present dive were qualitatively similar to those of previous reports. Thus, the hyperbaric diuresis in this study was characterized by a reduction in urine osmolality and clear evidence of a natriuresis, kaliuresis, and phosphaturia (4, 5, 18, 19). Also, increased Ca excretion has been frequently observed

TABLE 4

PLASMA CONCENTRATIONS OF ATRIAL NATRIURETIC FACTOR AND SOLUTES, AND CHANGES IN HEMATOCRIT AND PLASMA VOLUME[a]

	Predive	31 ATA			Postdive
	1 ATA	Day 2	Day 5	Day 7	1 ATA
ANF, pg/ml	29.1 ± 5.9	29.3 ± 2.7	27.7 ± 2.5	23.0 ± 4.3	31.0 ± 6.9
TOS, mosmol/kg	288 ± 2	289 ± 0	294 ± 1	284 ± 1	287 ± 0
Na, meq/liter	142 ± 0	148 ± 1	149 ± 2	144 ± 5	141 ± 0
K, meq/liter	4.2 ± 0.1	4.3 ± 0.1	4.5 ± 0.2	4.1 ± 0.3	4.0 ± 0.1
Ca, mmol/liter	–	2.35 ± 0.02	2.32 ± 0.02	2.40 ± 0.02	2.30 ± 0.02
Pi, mmol/liter	1.03 ± 0.06	1.10 ± 0.06	1.03 ± 0.06	1.10 ± 0.06	1.13 ± 0.03
Cr, mg/100 ml	1.1 ± 0	1.1 ± 0	1.2 ± 0	1.2 ± 0.1	1.1 ± 0
Hct, %	45.1 ± 3.0	47.3 ± 2.9	47.9 ± 2.1	46.2 ± 2.4	44.5 ± 3.3
ΔPV, %	0	−8.3 ± 1.6	−10.3 ± 3.7	−4.0 ± 2.3	3.1 ± 4.9

[a]Values are means ± SE. ANF, atrial natriuretic factor; ΔPV, percent change in plasma volume from predive value. Abbreviations as in Table 2.

during decompression and postdive (4, 18), as observed in the present study. At present we have no clear explanation why the hyperbaric environment increased the excretion rate of bone-structure materials such as Ca and Pi. Previous studies indicate that parathyroid hormone (PTH) is not increased (6). However, a similar phenomenon has been observed during space flight (20, 21) and may therefore be related to inactivity.

Hyperbaric diuresis was accompanied by a tendency for transient increase in Hct and decreased plasma volume during the early phase of hyperbaric exposure, in agreement with previous reports (3, 12, 18). This loss of plasma volume and the concomitant loss of approximately 1 kg of body weight are evidence of a negative water balance, most likely due to the increased urine output at pressure in the face of unchanged fluid intake.

A marked and sustained nocturia was also observed at 31 ATA, in agreement with previous reports (3, 5, 18). This nocturia was characterized by an increased excretion of osmotic substances and Cosm. Previous dives (5, 18) have indicated that hyperbaric nocturia is primarily induced by an inhibition of tubular reabsorption of certain solutes during the night.

The increased airway resistance associated with breathing high-density gas increases intrathoracic negative pressure, resulting in an increase in central blood volume. However, we have no evidence of increased atrial distension at depth. The previous study at 31 ATA either failed to demonstrate an increase or showed no changes in cardiac output during a dry saturation dive (22). Thus, atrial distension may not be causally related to the development of hyperbaric diuresis. A well-documented suppression of insensible water loss due to the reduction of diffusivity of water vapor (23) may contribute in part to a development of the diuresis. However, the question remains why the diuresis is more dominant at night. It has been reported that renal response to central volume expansion induced by the supine posture is attenuated

during sleep (24), resulting in no increase in urine flow by recumbency during sleep at normal pressure.

DiBona (25) has reported that a diminished renal sympathetic activity induces a marked diuresis, and indeed a previous study (26) has indirectly demonstrated a low sympathetic tone in supine position at high pressure. Sympathetic fibers are known to be distributed to the basolateral aspect of tubular cells and to directly modulate the Na reabsorption process (27). Thus, a possibility remains that the diuresis can be induced by a lower sympathetic tone during sleeping at night in the hyperbaric environment by inhibiting tubular reabsorption of Na.

The present experiment revealed a null response of ANF to hyperbaria, in agreement with another report (11). However, our present observation does not exclusively deny an involvement of ANF for the diuresis for the following reason. The urine flow in the morning between 0700 and 1000 h did not significantly increase even in hyperbaria, and the blood was withdrawn at 0700 h in the present study. Thus, no increase in the morning urine flow was concurrent with the lack of ANF change in the morning blood sample. Similar reasoning may be used to argue that no central volume expansion has occurred during the first 3 h of the daytime.

In the present dive we had no plasma aldosterone (Paldo) data; however, an increased Paldo was likely in the present case, as has been well documented in the earlier dives (5, 6). Increased Paldo may cause the kaliuresis, but other factor(s) must play a role in modulating the excretion of Na at high pressure.

In conclusion, a lack of ANF response to hyperbaria observed in the present study may indicate no cephalad blood shift at 31 ATA; however, it is possible that the sampling time of the blood was not appropriate to detect the ANF increase in the hyperbaric condition. Further examinations are needed for ANF by frequent blood sampling, preferably during the evening and night. It is obvious that an accumulation of more data is necessary before concluding whether the central volume expansion and/or ANF are causative factors in hyperbaric diuresis. One of the interesting observations in the present experiment was an increased excretion of Ca and Pi accompanying the hyperbaric diuresis. Previous studies indicate that this is not due to an increase in PTH (6). We cannot explain the physiologic mechanisms responsible for these observations. The present study did not provide evidence to indicate that the hyperbaric diuresis-natriuresis is mediated by ANF release.

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