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Renal Function in Hyperbaric Environment

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Abstract. During mixed gas saturation diving (to 3–49.5 ATA) daily urine flow increases by about 500 ml/day, with no changes in fluid intake and glomerular filtration rate. The diuresis is accompanied by a significant decrease in urine osmolality and increase in excretion of such solutes as urea, K^+, Na^+, Ca^{2+} and inorganic phosphate (Pi). The fall in urine osmolality is mainly due to a reduction of free water reabsorption which is associated with a suppression of insensible water loss and the attendant inhibition of antidiuretic hormone (ADH) system. The increase in urea excretion may be associated with a reduction of urea reabsorption at the collecting duct as a consequence of ADH suppression. The rise in K^+ excretion is due to a facilitated K^+ secretion at the distal tubule as a result of increased aldosterone, urine flow and excretion of impermeable anions such as Pi. The activation of aldosterone system is partly attributed to a transient dehydration induced by early hyperbaric diuresis. The increase in Na^+ excretion in the face of enhanced aldosterone secretion indicates that the Na^+ transport in the proximal tubule is markedly inhibited (by unknown mechanism). The Pi excretion increases with no changes in plasma level of parathyroid hormone (PTH), thus it may be due to an inhibition of Na^+-Pi cotransport in the proximal tubule. The increase in Ca^{2+} excretion may be secondary to the inhibition of Na^+ transport at the proximal tubule. Precise information on the proximal tubular Na^+ transport is important to understand the mechanisms of impaired solute transport under hyperbaric conditions.

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Numerous studies have documented that renal function undergoes significant variations during mixed-gas saturation diving. In the present communication we will review some of these works in an attempt to characterize the renal response to hyperbaric exposure. Emphasis will be made on the mechanism underlying the hyperbaric diuresis. This topic has been reviewed previously by Hong (1975), Hong et al. (1983; 1995), Hong and Claybaugh (1989), Shiraki (1987), and Sagawa et al. (1996).

Characteristics of Hyperbaric Diuresis

Fig. 1 depicts time courses of urine flow in subjects exposed to 31 ATA He-O_2 atmosphere determined in three saturation dives, Seadragon IV (Nakayama et al., 1981), Seadragon VI (Shiraki et al., 1987), and New Seatopia (Sagawa et al., 1990), conducted in Japan Marine Science and Technology Center (JAMSTEC). The daily urine flow increased rapidly upon compression to a value 700–1000 ml/day above the predive level, then it dropped off slightly to a steady level of approximately 500 ml/day above the predive level. During decompression, the diuresis slowly disappeared and the urine flow returned to the control level towards the end of decompression. Similar changes in urine flow have been observed in many other saturation dives.

Fig. 2 (upper panel) summarizes changes in daily urine flow determined in 18 different saturation dives to various depths (3–49.5 ATA). The urine flow increased in all dives except one (Buhlmann et al., 1970) in which the urine flow decreased by about 16% during exposure to 31 ATA. The net increase in urine flow (hyperbaric diuresis) varied from 137 ml/day (Raymond et al., 1980) to 1300 ml/day (Hamilton et al., 1966), with an average of 560 ml/day (Fig. 2, lower panel). It appears that there is no apparent correlation between the degree of diuresis and the depth of dive.

The threshold pressure for hyperbaric diuresis appears to be around 3 ATA in N_2-O_2 and 7 ATA in He-O_2. Niu et al. (1990) observed no diuresis in subjects exposed to 2.5 ATA N_2-O_2 (at a density of 3.16 kg/m^3), but Sagawa et al. (1996) observed a marked diuresis at 3 ATA N_2-O_2 (3.79 kg/m^3). In He-O_2 atmosphere, no diuresis was observed at 4 ATA (1.82 kg/m^3) (Shiraki et al., 1982), but a significant diuresis was noted at 7 ATA (2.25 kg/m^3) (Matsuda et al., 1975). Apparently, the gas density plays an important role in generation of hyperbaric diuresis,

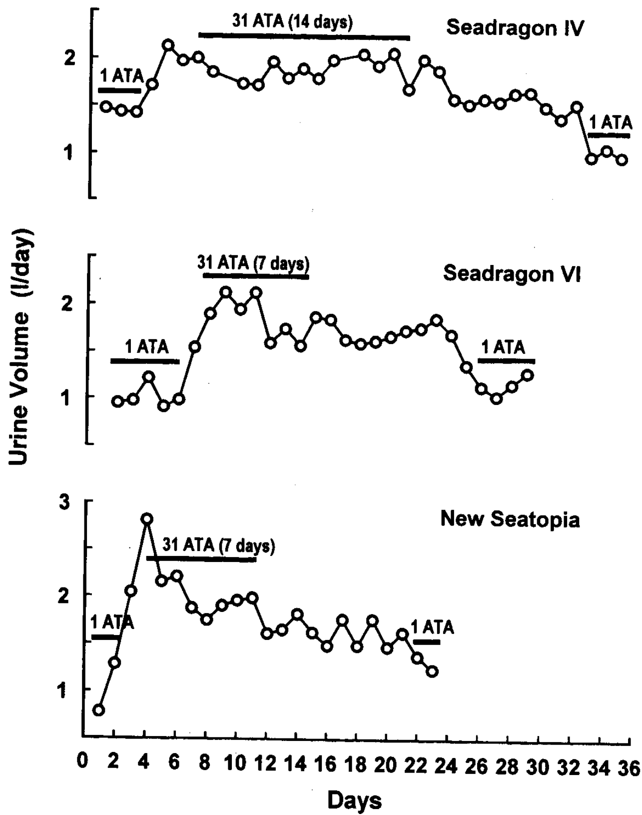


Fig. 1 Time courses of urine flow during saturation divings to 31 ATA. Data are from Seadragon IV (Nakayama et al., 1981), Seadragon VI (Shiraki et al., 1987), and New Seatopia (Sagawa et al., 1990) dives.

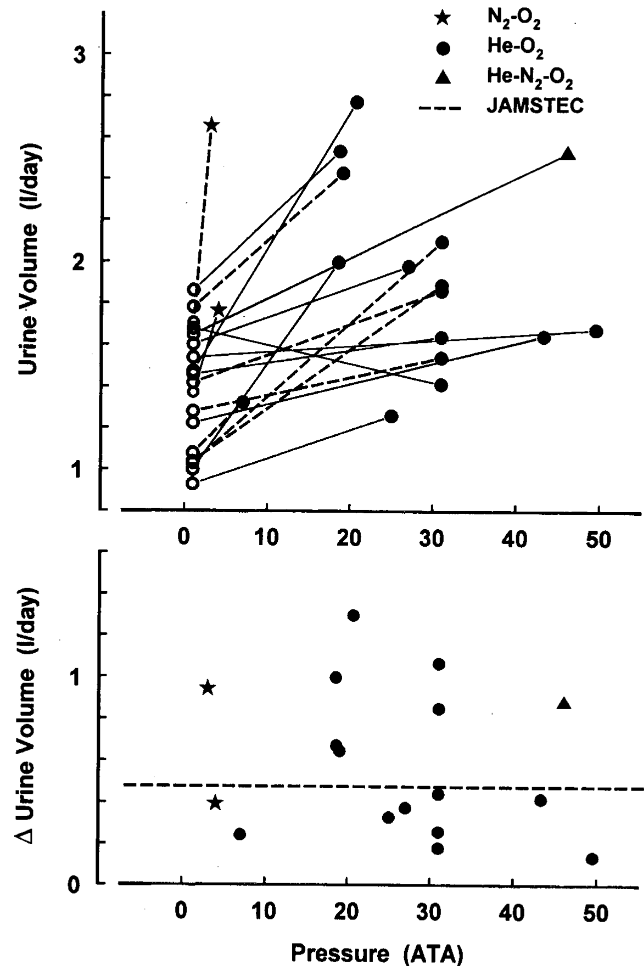


Fig. 2 Changes in daily urine volume in saturation divings to various depths. Data are based on Alexander et al. (1973), Buhlmann et al. (1970), Goldinger et al. (1992), Hamilton et al. (1966), Hong et al. (1977), Leach et al. (1978), Matsuda et al. (1975), Miyamoto et al. (1991), Nakayama et al. (1981), Neuman et al. (1979), Raymond et al. (1980), Sagawa et al. (1990, 1996), Schaefer et al. (1970), and Shiraki et al. (1984, 1987). Dashed lines in the upper panel represent experiments conducted in Japan Marine Science and Technology Center (JAMSTEC) and the dashed line in the lower panel represents the average value of urine volume change.

which may be related to the effects of gas density on water vapor diffusion and ventilatory mechanics, as will be described below.

The hyperbaric diuresis consists of both osmotic and water diuresis components. As depicted in Fig. 3 which summarizes results of three JAMSTEC dives mentioned above (Seadragon IV and VI and New Seatopia), the increase in urine flow (V) at pressure (31 ATA) is accompanied by an increase in osmolal clearance (C_{osm}) and a decrease in the negative free water clearance-to-osmolal clearance ratio (T^{c}_{H2O}/C_{osm}). The T^{c}_{H2O}/C_{osm} represents the relative free water reabsorption at the medullary collecting duct. These changes in renal function generally occur with no significant changes in plasma osmolality and glomerular filtration rate, indicating that the renal tubular transport, not the filtered load, of salt and water is altered in hyperbaric environment.

Fig. 4 illustrates changes in urinary excretion of various solutes. The Na^+ excretion either increases or remains unchanged at pressure (31 ATA). The K^+ excretion is always found to increase markedly. Likewise, urinary excretion of urea and inorganic phosphate (Pi) appears to be significantly enhanced at pressure. However, the Ca^{2+} excretion remains unchanged or rises slightly. Overall, the osmotic diuresis observed at pressure is largely associated with increase in urea, K^+ and Pi excretions, and in some instances with Na^+ and Ca^{2+} excretions.

Mechanism of Hyperbaric Diuresis

One of the consistent features of hyperbaric diuresis is a fall in urine osmolality. As shown in Fig. 5, the hyperbaric diuresis is accompanied by a significant

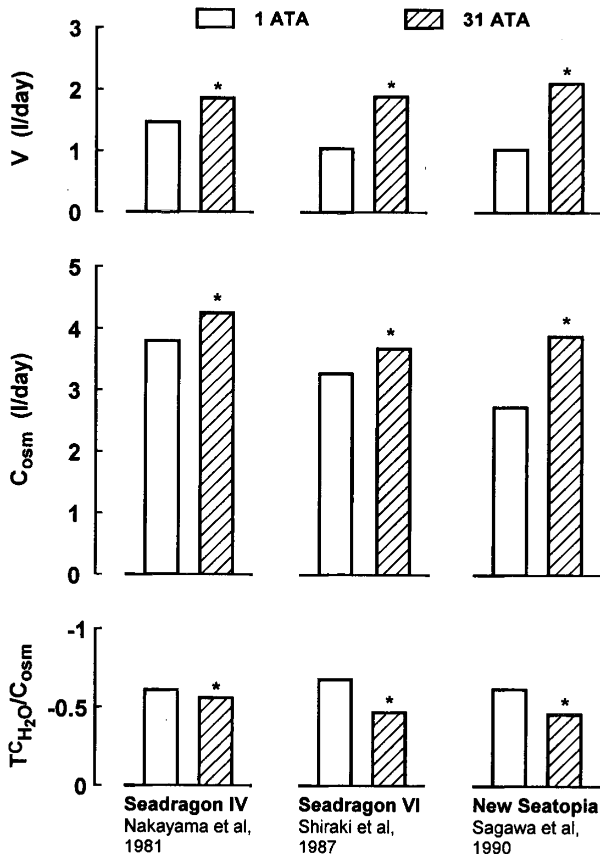


Fig. 3 Daily urine volume (V), osmolal clearance (Cosm) and the negative free water clearance-to-osmolal clearance ratio (T^{c}_{H2O}/Cosm) before and during exposure to 31 ATA. Data are from Seadragon IV (Nakayama et al., 1981), Seadragon VI (Shiraki et al., 1987), and New Seatopia (Sagawa et al., 1990) dives. *Significantly different from the pre-dive 1 ATA value.

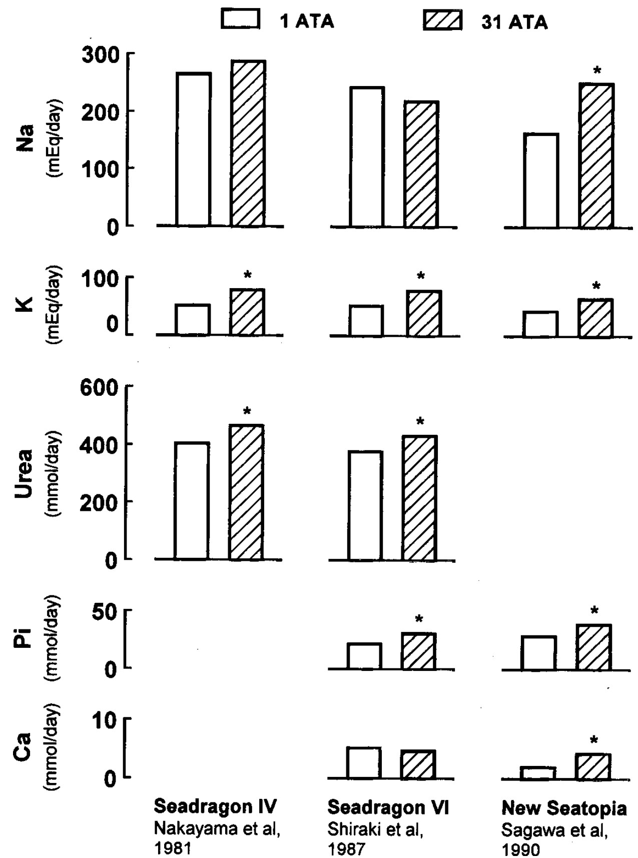


Fig. 4 Daily urinary excretion of Na^{+}, K^{+}, urea, inorganic phosphate (Pi), and Ca^{2+} before and during exposure to 31 ATA. Data are from Seadragon IV (Nakayama et al., 1981), Seadragon VI (Shiraki et al., 1987), and New Seatopia (Sagawa et al., 1990) dives. *Significantly different from the 1 ATA value.

reduction in urine osmolality (U_{osm}). The degree of U_{osm} change is approximately 200 mOsm/kg H_2O. Such a change may be attributed mainly to a reduction of free water reabsorption at pressure. The amount of net free water reabsorption is determined by the osmotic pressure gradient between the medullary interstitium (ISF_{osm}) and collecting duct urine (TF_{osm}) and water permeability of tubular membrane (k_{H2O}): $T^{c}_{H2O} = k_{H2O}$ ($ISF_{osm} - TF_{osm}$). Since the k_{H2O} is determined by the ADH action and the ($ISF_{osm} - TF_{osm}$) gradient by the sodium pump activity in the ascending Henle's loop and the medullary blood flow (Valtin, 1983), the change in T^{c}_{H2O} at pressure should be mediated by changes in one (or more) of these factors. Neither the sodium pump activity nor the medullary blood flow in hyperbaric environment has been directly assessed, but the water loading experiments at 16.1 ATA by Moore et al. (1975) and at 31 ATA by Takeuchi et al. (1995) imply that at least the sodium pump in the diluting segment is not significantly altered by hyperbaric

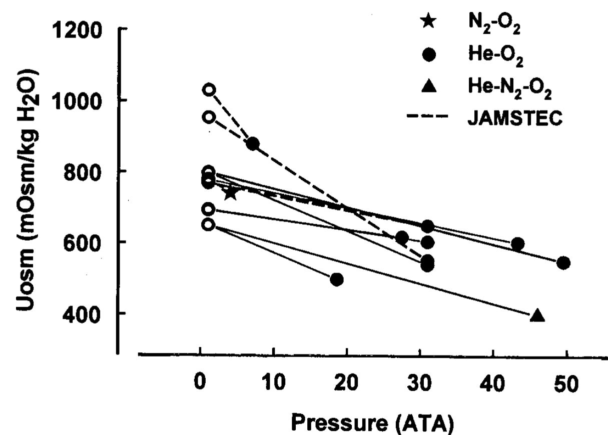
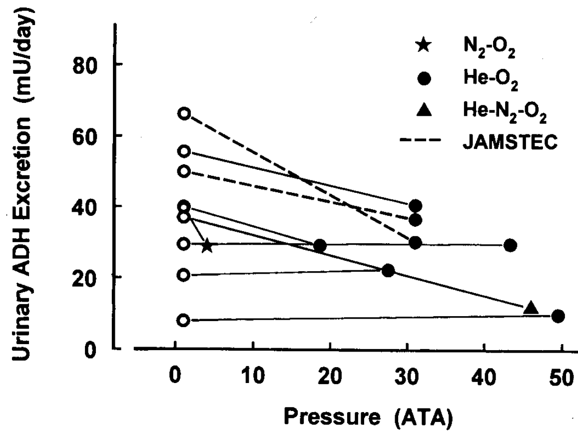


Fig. 5 Changes in urine osmolality (Uosm) in saturation divings to various depths. Data are based on Alexander et al. (1973), Goldinger et al. (1992), Hong et al. (1977), Leach et al. (1978), Matsuda et al. (1975), Miyamoto et al. (1991), Nakayama et al. (1981), Neuman et al. (1979), Raymond et al. (1980), Sagawa et al. (1990), and Shiraki et al. (1987).



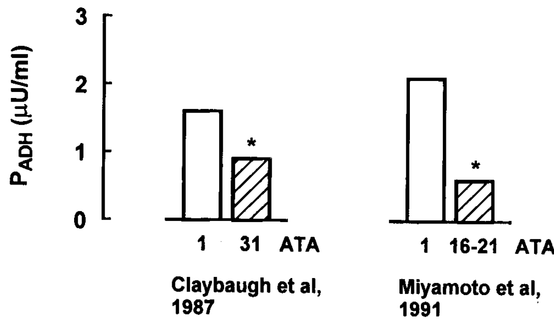


Fig. 6 Changes in urinary excretion (Upper panel) and plasma level (Lower panel) of ADH in saturation divings to various depths. Data are based on Claybaugh et al. (1984, 1987, 1992), Hong et al. (1977), Leach et al. (1973, 1978), Neuman et al. (1979), Raymond et al. (1980) and Miyamoto et al. (1991). *Significantly different from the 1 ATA value.

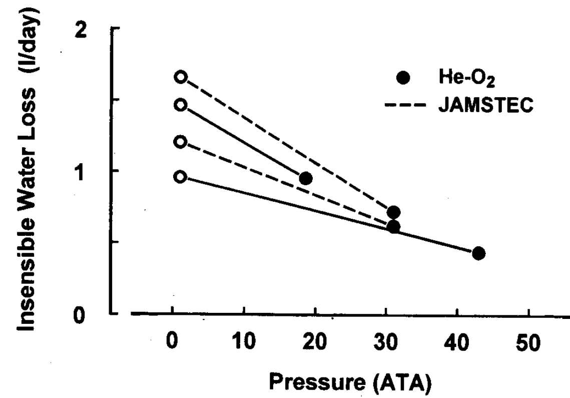


Fig. 7 Changes in insensible water loss in saturation divings to various depths. Data are based on Hong et al. (1977), Nakayama et al. (1981), Shiraki et al. (1987), and Raymond et al. (1975).

exposure. The above experiments have shown that the minimum U_{osm} achieved after 1 liter of water ingestion is not different between 1 ATA and hyperbaric environment. This suggests that the urine diluting capacity of the kidney, which depends on the ascending Henle's loop sodium pump activity, is not impaired by high pressure.

In the case of ADH system, a number of studies have shown that it is attenuated under high pressure. Fig. 6 (upper panel) summarizes changes in 24-h urinary ADH excretion determined in 9 different saturation dives. In 6 dives the ADH excretion decreased at pressure. In 3 dives the hormone excretion appeared to be unaltered, but in these cases the predive level of the hormone excretion was rather low. Plasma level of ADH was also found to decrease at pressure (16–31 ATA) by 40–60% (Fig. 6, lower panel). It is, therefore, likely that the reduction in free water reabsorption in hyperbaric environment is primarily attributed to a suppression of ADH system.

Several mechanisms have been advanced to account for the ADH suppression in high pressure environment. During hyperbaric exposure the water intake is generally changed little. However, insensible water loss falls progressively as the pressure increases (Fig. 7). This may result in water retention and consequently lead to a suppression of ADH secretion. Hong et al. (1977), therefore, proposed that the primary mechanism for the hyperbaric diuresis is the suppression of insensible water loss. The reduction in insensible water loss has been explained by the fact that the diffusivity of the water vapor is inversely proportional to the ambient pressure or gas density (Paganelli and Kurata, 1977). Another mechanism which could account for ADH suppression is the blood redistribution associated with breathing a high density gas mixture (Hong et al., 1977). According to this notion, the negative intrathoracic pressure during breathing a high density gas facilitates venous return and increases thoracic blood volume, with a consequent suppression of ADH release via Gauer-Henry reflex (1976). This hypothesis, however, has not been verified. In fact, experiments involving 60-min negative pressure breathing of − 15 cm H_2O (Hebden et al., 1992) or − 11 mm Hg (Tanaka et al., 1991) have shown that the plasma ADH level does not undergo significant variations, the results inconsistent with the above notion.

The mechanism(s) underlying the osmotic diuresis observed in hyperbaric environment may be multiple. The osmotic diuresis is accompanied by increased excretion of urea, K^+, and Pi in most cases and Na^+ and Ca^{2+} as well in some cases (see Fig. 4).

The increase in urea excretion is probably associated with a reduction of ADH secretion. Urinary excretion of urea is determined by its reabsorption in the late collecting duct. This urea reabsorption (T_{urea}) is a

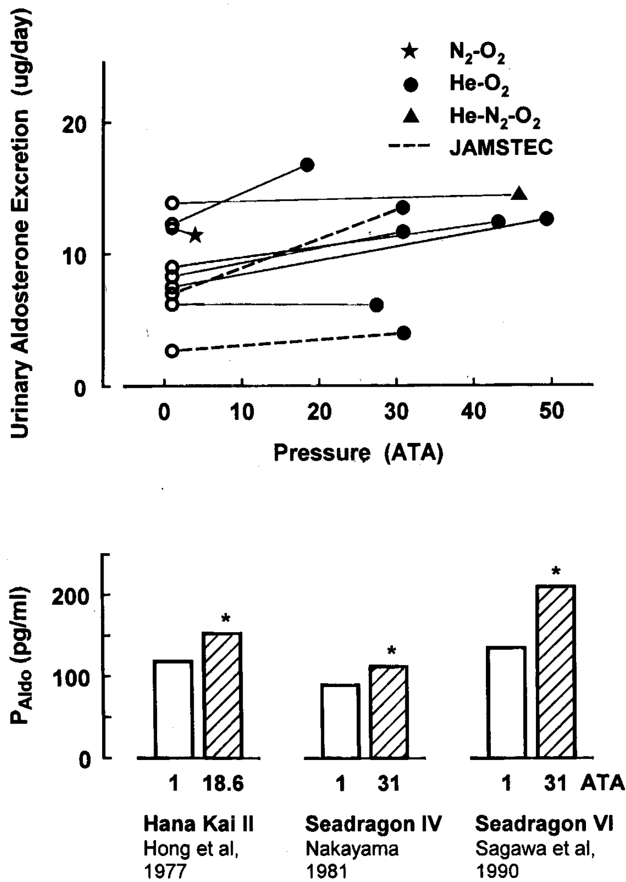


Fig. 8 Changes in urinary excretion (Upper panel) and plasma level (Lower panel) of aldosterone in saturation divings to various depths. Data are based on Claybaugh et al. (1984, 1987, 1992), Hong et al. (1977), Leach et al. (1973, 1978), Neuman et al. (1979), Raymond et al. (1980), Nakayama et al. (1981), and Sagawa et al. (1990). *Significantly different from the 1 ATA value.

passive transport process, and thus is proportional to the urea concentration gradient between the late collecting duct urine (TF_{urea}) and papillary interstitium (ISF_{urea}) and the permeability of membrane to urea (k_{urea}): $T_{urea} = k_{urea} (TF_{urea} - ISF_{urea})$. ADH facilitates the T_{urea} by increasing both the k_{urea} and TF_{urea} (Valtin, 1983). The later effect is due to a differential effect of ADH on the urea and water permeabilities of the distal tubule and collecting ducts. ADH increases water permeability of the distal tubule and the entire collecting duct; however, it increases the urea permeability only in the late collecting duct. Consequently, as water is withdrawn from the distal tubules and early collecting ducts, the urea, unable to diffuse out of the lumen as readily as water, is progressively concentrated until it reaches the late collecting duct where it is reabsorbed. Thus, a reduction of ADH secretion, as in hyperbaric environment, would impair the driving force, as well as the membrane permeability, for urea reabsorption.

The kaliuria observed in hyperbaric environment is probably related to several factors. Urinary excretion of K^+ is determined by K^+ secretion in the distal tubule (Giebisch, 1983). K^+ in the blood first moves into the distal tubular cell by active transport mechanism at the basolateral membrane and then diffuses across the luminal membrane into the lumen. The first process is activated by aldosterone, and the second process by factors affecting the K^+ electrochemical potential gradient across the luminal membrane. Numerous studies have shown that the urinary excretion and the plasma level of aldosterone increase during hyperbaric exposure (Fig. 8). Also, the excretion of impermeable anion, such as Pi, is found to rise at pressure (Fig. 4). The latter phenomenon together with the increased urine flow would increase the K^+ electrochemical potential gradient across the distal tubular luminal membrane. Thus, both the active and passive steps of K^+ secretion could be facilitated under high pressure.

The mechanism by which aldosterone system is activated in hyperbaria is not entirely clear, but it may be related in part to the transient dehydration induced by early hyperbaric diuresis. The diuresis observed during the early phase of hyperbaric exposure is greater in magnitude than the subsequent steady-state diuresis (Fig. 1) and is accompanied by an increase in hematocrit and plasma protein concentration (Fig. 9). This, together with a slight reduction in body weight is indicative of a mild net loss of body fluid during the early phase of hyperbaric exposure. In fact, the plasma volume loss estimated from hematocrit change using Van Beaumont's formula (1972) appears to increase progressively during the early hyperbaric phase, and this is accompanied by a marked increase in aldosterone excretion (Fig. 9).

The mechanism of the hyperbaric natriuresis observed in some dives is not forthcoming. Since the aldosterone level is increased at pressure, the Na^+ reabsorption in the distal nephron must have been stimulated. Thus, an increase in urinary Na^+ excretion at pressure would indicate that the Na^+ reabsorption in the proximal nephron is markedly suppressed. Several studies have examined atrial natriuretic peptide (ANP), which alters Na^+ transport in the renal tubule, as a factor inducing hyperbaric natriuresis. Miyamoto et al. (1991) observed a significant elevation of ANP in subjects exposed to 16 ATA. On the other hand, Sagawa et al. (1990) and Moon et al. (1987) observed no significant changes in ANP level at 31 and 46 ATA, respectively, despite the marked increase in Na^+ excretion. Taken together, the ANP results may suggest that the hyperbaric natriuresis is not mediated by ANP secretion. An alternative possibility is the direct effect of high pressure on Na^+ transport system. Goldinger et al. (1980) showed that active Na^+ efflux from human

erythrocytes is significantly inhibited by a modest hydrostatic pressure of 30–50 ATA. Later studies using toad skin epithelia also indicated that the transepithelial active Na^+ transport is progressively inhibited with the increase in hydrostatic pressure (Hong et al., 1984; Goldinger et al., 1986). If such an inhibitory effect of high hydrostatic pressure exists for the active Na^+ transport in renal tubular epithelia, it could account for the hyperbaric natriuresis.

Regardless of the mechanism, an inhibition of proximal tubular Na^+ reabsorption would exert a profound effect on the excretion of passively transported substances. Rejection of Na^+ in the proximal tubule would retard water reabsorption, and hence increase fluid delivery to the distal nephron. This would increase driving force for K^+ secretion in the distal tubule (Giebisch, 1983) and decrease urea reabsorption in the collecting duct (Valtin, 1983). It is, therefore, possible that the increased excretion of K^+ and urea in hyperbaric environment is partly accounted for by the inhibition of proximal tubular Na^+ transport.

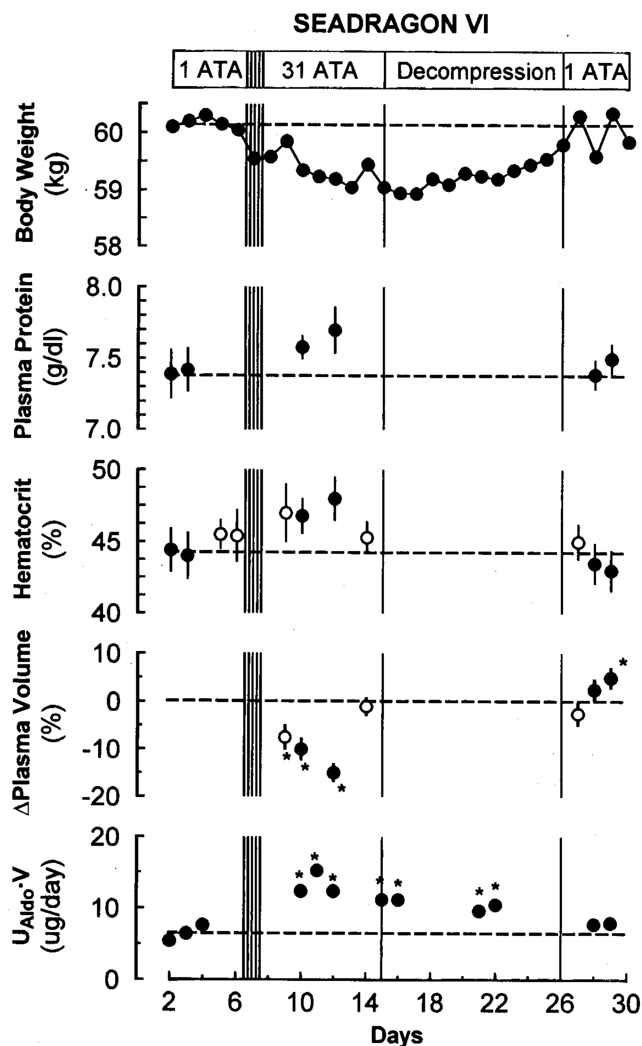


Fig. 9 Changes in body weight, plasma protein, hematocrit, plasma volume, and urinary aldosterone excretion during the course of a saturation diving to 31 ATA (Seadragon VI). Data are based on Shiraki et al. (1987) and Claybaugh et al (1987). *Significantly different from the 1 ATA value.

The mechanisms mediating the phosphaturia and occasional hypercalciuria observed in hyperbaric environment have not been clearly elucidated. Since the changes occur with no apparent variations in plasma parathyroid hormone (PTH) level (Claybaugh et al., 1987), alterations of their transport activities at the distal nephron may not be involved. Micropuncture studies in animal models have shown that 80–90% of the Pi filtered through glomeruli is reabsorbed in the renal tubules, almost all of which occur in the proximal tubule (Greger et al., 1977). Filtered Pi initially moves from the lumen into the tubular cell via a Na^+-Pi cotransport process at the apical membrane, then it diffuses across the basolateral membrane into the peritubular capillary (Hammerman, 1986). Since the rate-limiting step of Pi reabsorption is known to be the Na^+-Pi cotransport (Gmaj and Murer, 1986), it is speculated that the Na^+-Pi cotransport mechanism at the proximal tubular luminal membrane is attenuated during hyperbaric exposure. In this respect, it is important to note that the apical membrane Na^+ conductance is significantly reduced in toad skin epithelia exposed to high hydrostatic pressure (Wilkinson et al., 1987). Such a reduction in Na^+ conductance, if it occurs in the renal proximal tubular epithelia, would retard Na^+-Pi cotransport.

The inhibition of proximal tubular Na^+ transport would also affect Ca^{2+} reabsorption. Animal studies have indicated that reabsorption of filtered Ca^{2+} closely parallels that of Na^+, occurring predominantly in the proximal tubule (Lassiter et al., 1963). Thus, one may expect that Ca^{2+} excretion would rise when transport of Na^+ is retarded in the proximal tubule. Precise information on the proximal tubular Na^+ transport is, therefore, important in understanding the mechanisms of impaired renal tubular transport of various solutes under hyperbaric conditions.

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