Comp. Biochem. Physiol. Vol. 86A, No. 1, pp. 49–55, 1987
Printed in Great Britain

0300-9629/87 $3.00 + 0.00
Pergamon Journals Ltd

METABOLIC EFFECTS IN RATS DRINKING INCREASING CONCENTRATIONS OF SEA-WATER

Z. ETZION and R. YAGIL*

Corob Medical Center, Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer Sheva, Israel

(*Received* 25 *March* 1986)

Abstract—1. Research on laboratory rats confirmed that drinking sea-water when dehydrated, was not beneficial and caused impaired renal function.

2. When the concentration of sea-water in the drinking water is gradually increased there is a gradual increase in water uptake and corresponding urine excretion.

3. At 50% sea-water the maximum uptake and excretion is reached. Following this there is a decline in appetite, water uptake and urine secretion.

4. When on 100% sea-water, the creatinine clearances were greater than on tap water, while urine/plasma osmolalities (U/P) averaged 7. The only higher U/P was found in animals drinking sea-water when dehydrated, i.e. a U/P of 11.

5. The urea metabolism appears to be suited to either the need to conserve body water, up to 50% sea-water, or to guarantee an adequate urine production, from 50% sea-water to pure sea-water.

6. It is suggested that when a man is stranded at sea it is not advisable to drink all the fresh water and then be compelled to drink sea-water when dehydrated.

7. It is better to slowly increase the sea-water uptake. This will prolong the time before sea-water needs to be drunk and result in only minor metabolic changes. Return to fresh water will be followed by an immediate return to normal homeostasis.

INTRODUCTION

A ship-wrecked person with no fresh drinking water could theoretically replenish his urinary losses by drinking sea-water (Bellamy *et al.*, 1975). In fact, a steady increase in body sodium would occur that eventually would be fatal. In order to excrete the additional absorbed sodium, so much water would be lost that dehydration would follow (Brown *et al.*, 1947). Thus, a man stranded at sea without drinking water is in a similar position to a man stranded in the desert without water (Smith, 1951). A man with the sea as his only source of drinking water is confronted with "water, water everywhere, nor any drop to drink" (Coleridge—*The Rime of the Ancient Mariner*).

There is a remarkable constancy of sodium content of the body fluids of nearly all vertebrates (Bellamy *et al.*, 1975). Mammals rely on their kidneys to regulate body sodium. The mammalian metanephros is a capable excretor of sodium. Not so the non-mammalian vertebrate mesonephros (Schmidt-Nielsen, 1979; Smith, 1951). Non-mammals are unable to excrete a hypertonic urine, so must rely on supplementary excretory organs, the salt glands. All birds and reptiles possess paired salt glands situated near their eyes (Schmidt-Hielsen, 1979). Non-active glands of inland birds became highly active when the animals were brought close to the sea (Holmes, 1977). Large amounts of salt were absorbed and necessitated selective excretion. The absorption and excretion were prevented when spironolactone was administered, demonstrating the role of aldosterone in absorbing salt and water from the intestines. Most fish living in the sea drink the water and excrete the unwanted salt via their gills leaving osmotically free water in their bodies (Schmidt-Nielsen, 1979; Smith, 1951). Marine birds and fish can therefore survive drinking sea-water when no fresh water is available. Marine sharks and rays, though, do not drink sea-water and rely on urea retention to maintain normal body water content, salt being excreted mainly from rectal glands.

Drinking sea-water, or even urine, is of no benefit to man or other mammals, who have no extra renal mechanism of excreting salts (Bellamy *et al.*, 1975). Accidentally swallowing large amounts of sea-water can cause transient renal failure (Grausz *et al.*, 1971; Oren *et al.*, 1982). Even imbibing brackish water causes metabolic changes, both acute and chronic (Yagil and Berlyne, 1975).

It is thus clear that drinking sea-water *per se* is not advisable for Man or other mammals. The rat survives drinking sea-water only slightly longer than Man (Smith, 1951). The problem is further aggravated by the fact that drinking sea-water is usually postponed for as long as possible and by this time Man will already be dehydrated.

This communication examines the metabolic effects of drinking increasing concentrations of sea-water compared with drinking sea-water prior to and following dehydration.

*All correspondence to be addressed to: Prof. R. Yagil, Unit of Physiology, Faculty of Health Sciences, Ben-Gurion University of the Negev, P.O. Box 653, 84105 Beer Sheva, Israel.

MATERIALS AND METHODS

Male white laboratory rats of the Wistar strain were randomly divided into nine groups. Each group consisted of 16 animals. Each animal was weighed at the beginning and at the end of the experiment. Food was the normal rat chow (Labena, Asia Maabarot) and was given *ad libitum*. Each group received a different water regimen *ad libitum*. The groups were:

1. Tap water (26 mOsm/kg);
2. 10% sea-water for 4 days (140 mOsm/kg);
3. 30% sea-water for 4 days (330 mOsm/kg);
4. 50% sea-water for 4 days (552 mOsm/kg);
5. 70% sea-water for 4 days (745 mOsm/kg);
6. 100% sea-water for 4 days (1100 mOsm/kg);
7. From pure sea-water (group 6) to tap water for 24 hr (Sea → Tap);
8. Acute sea, i.e. 24 hr on sea-water from tap water;
9. Following 2 days of dehydration at room temperature, sea-water for 24 hr (Dehydration → Sea).

Groups 3–7 were given gradually increasing sea-water content as drinking water, 4 days at each concentration, until the specific concentration was reached.

For the last 24 hr of each period, the animals were kept in metabolic cages, where water and food uptake, urine and faeces production, were recorded. The urine was collected with thymol in the collecting tubes. Following anaesthetization with chloral hydrate, blood was taken by direct cardiac puncture using heparinized venoject tubes.

The following parameters were determined: (a) Blood hematocrit using a hematocrit centrifuge (Wifug, Sweden); (b) Serum and urinary sodium on a flame photometer (No. 243, Instrumentation Labs. Italy); (c) Serum and urinary urea and creatinine on a SMA 6/60 Autoanalyzer (Technicon, Tarrytown, U.S.A.); (d) Water, serum and urine osmolalities on a Fiske O-M-osmometer (Fiske Associates, Uxbridge, Massachussets, U.S.A.); (e) Clearances were calculated using the formula

$$\frac{UV}{P},$$

where U and P = concentrations in urine and plasma respectively and V = volume of urine in 24 hr (f) The total urinary sodium excretion of substances was calculated from the concentration and the volume of urine. The results are given as mean ± standard error (SEM).

RESULTS

Body weights

All groups started with similar body wts (215 ± 20 g). Nearly all groups drinking increasing concentrations of sea-water had the same increases in body wt as the control animals. Only the group on 100% sea-water had 5% less gain in body wt, probably due to a decline in food uptake. Two days of dehydration at room temperature caused a loss of 12% in body wt, with a further loss of 4% when presented with sea-water.

Water uptake

The volumes of water drunk are given in Fig. 1. There was a highly significant increase in water uptake from 10% ($P < 0.001$) and the uptake increased further on 30% sea-water ($P < 0.005$) and 50% sea-water ($P < 0.001$). There was a 124% increase in drinking between tap water and 50% sea-water. Following this, on 70% sea-water there was a sharp drop in water uptake. There was a further decline in water uptake when 100% sea-water was drunk, to 45% less than control animals ($P < 0.05$).

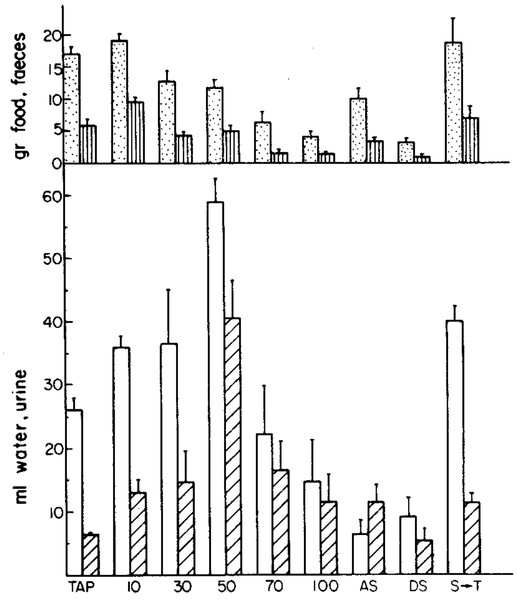

Fig. 1. Water, food, urine and faeces metabolism with increasing concentrations of sea water; * = 10, 30, 70, 100 = % of sea-water; AS = acute sea for 24 hr; DS = sea for 24 hr following dehydration; S–T = tap water for 24 hr after 100% sea-water. □ = water; ▨ = urine; ⬚ = food; ▥ = faeces.

Water uptake of animals receiving sea-water acutely and those who were dehydrated first and then received sea-water was significantly depressed ($P < 0.001$). The animals acutely presented with sea-water drank the least, 76% less than tap water. Animals slowly brought to drinking sea-water and then returned to tap water drank significantly more than control animals ($P < 0.001$).

Osmol uptake

The osmol uptake in 24 hr by each group is given in Table 1. The greatest osmol uptake from water was by the group drinking 50% sea-water. Although animals drank less on 70% than 100% sea-water, their osmol uptake was the same. Although animals subjected acutely to drinking sea-water drank less, their osmol uptake was relatively high.

Urine excretion

This is given in Fig. 1. There were significant increases in urine excretion when the animals drank

Table 1. Osmol uptake from drinking water and osmol clearances when drinking varying concentrations of sea-water

	Osmol uptake in each group (mOsm/24 hr)	Osmolar clearances$^{\Delta}$ (ml/hr)
Tap	0.7	1.3
10% sea	5	3.1
30% sea	12	3.9
50% sea	32	7.0
70% sea	17	3.9
100% sea	16	3.9
Acute sea	7	3.1
Dehydrated → Sea	10	2.4
Sea → Tap	1.0	3.1

Δ = calculated from the averages.

10% and 30% sea-water ($P < 0.005$) and highly significant increases with 50% sea-water ($P < 0.001$). The greatest volume of urine excreted by any group was with 50% sea-water, then there was a sharp decline in urine excretion when drinking 70% sea-water and a further decline with 100% sea-water, acute drinking of sea-water and sea-water following dehydration. The differences in urine production between the latter cases and control animals were non-significant ($P > 0.05$). The animals presented with sea-water following dehydration were the only ones who excreted urine volumes below those of animals drinking tap water. When animals were returned to tap water from sea-water, there were no changes in urine volumes in the first 24 hr.

Food uptake and faeces production

These are given in Fig. 1. There was an increase in food uptake and faeces excretion when the animals drank 10% sea-water ($P < 0.01$). On 30, 50, 70 and 100% sea-water and sea-water following dehydration, there were steady decreases in food uptake and faeces production. On 70% and 100% and sea-water following dehydration, the decreases were highly significant compared to control animals ($P < 0.001$). The smallest food uptake and faeces production was found in the animals who drank sea-water after being dehydrated.

It is noteworthy that the animals who drank the most water, those on 50% sea-water, were the only animals with diarrhoea.

Hematocrit (Table 2)

There was a non-significant increase in hematocrits in animals drinking 10, 30 and 50% sea-water. The increase persisted and was highly significant with 70 and 100% sea-water ($P < 0.001$), as it was on acute sea-water ($P < 0.005$). The highest hematocrit was found in the dehydrated animals drinking sea-water, 28% higher than control values ($P < 0.001$). The

Table 2. Blood hematocrit of animals drinking varying concentrations of sea-water

Water	Hematocrit (%)
Tap	43 ± 1.5
10% sea	45 ± 1.7^{NS}
30% sea	45 ± 1.5^{NS}
50% sea	47 ± 1.7^{NS}
70% sea	$50 \pm 0.4^{***}$
100% sea	$52 \pm 0.9^{***}$
Acute sea	$49 \pm 0.9^{**}$
Dehydrated → Sea	$55 \pm 0.8^{***}$
Sea → Tap	$48 \pm 0.5^{**}$

NS = non-significant. ** = $P < 0.005$. *** = $P < 0.001$.

animals returned to tap water had lower hematocrits than when on sea-water, but still significantly higher than normal ($P < 0.005$).

Osmolalities (Fig. 2)

(i) *Plasma.* On 10% sea-water there was a slight increase in plasma osmolalitites ($P < 0.05$). Further increases in the concentration of sea-water caused highly significantly elevated osmolalities ($P < 0.001$). Acute drinking of sea-water increased osmolalities to the same level as when gradually brought to sea-water. Sea-water after dehydration did not cause as large an increase in plasma osmolalities ($P < 0.01$). Returning to tap water from sea-water caused a decline in osmolalities, but they were still higher than normal.

(ii) *Urine.* Except for the group getting 50% sea-water, all animals drinking sea-water in any concentration, had increased urine osmolalities ($P < 0.01$ to $P < 0.001$). The highest urine osmolalities were found in the animals drinking sea-water after being dehydrated ($P < 0.001$). Returning to drinking tap water did not change the urine osmolality.

(iii) *The total urinary osmol excretion.* This was great in all groups compared with tap water. The increases were highly significant ($P < 0.001$). The

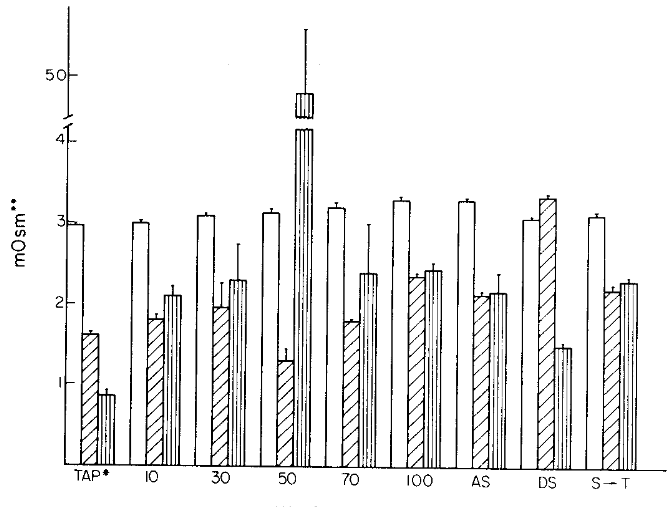

Fig. 2. Osmolalities of plasma and urine and total excretion with increasing concentrations of sea-water; * = 10, 30, 50, 70, 100 = % of sea-water; AS = acute sea for 24 hr; DS = sea for 24 hr following dehydration; S–T = tap water for 24 hr after 100% sea-water. **□ = mOsm/kg × 10^2 in plasma; ▨ = mOsm/kg × 10^3 in urine; ▥ = total excretion mOsm × 10^1.

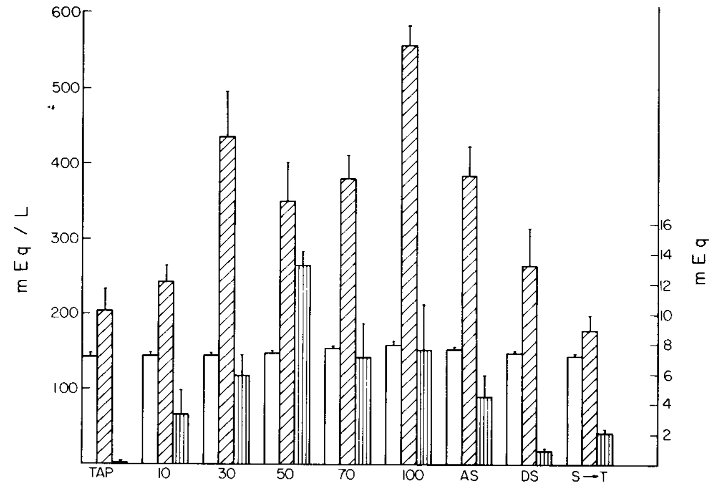

Fig. 3. Sodium concentrations of plasma and urine and total excretion with increasing concentrations of sea-water; * = 10, 30, 50, 70, 100 = % of sea-water; AS = acute sea for 24 hr; DS = sea for 24 hr following dehydration; S–T = tap water for 24 hr after 100% sea-water. □ = plasma (mEq/l); ▨ = urine concentration (mEq/l); ▥ total urine excretion (mEq).

greatest osmotic excretion was found in the animals drinking the most water, i.e. 50% sea-water, even though the urine osmolality was low. Then the excretion of osmotic substances decreased with decreased water consumption on 70 and 100% sea-water, but was remarkably constant. Animals drinking sea-water after dehydration had relatively small osmol excretion even though urine osmolalities were high.

(iv) *Osmol clearances.* These are given in Table 1. All groups, except the animals getting sea-water following dehydration, had more than double the osmol clearances when compared to the group drinking tap water. The post-dehydrated group had an 84% increase in osmol clearances. The highest osmol clearances were found in the animals drinking the most water, i.e. 50% sea-water, an increase of 418%. Although returning to tap water led to a decline in osmol clearances, they were still higher than normal.

(v) *Concentration capability of the kidneys.* This was calculated from urine osmolality/plasma osmolality (U/P) and are as follows: Tap water 4.9; 10% sea 5.8; 30% sea 6.2; 50% sea 4.1; 70% sea 5.6; 100% sea 7.8; acute sea 6.4; sea-water after dehydration 11; tap water after 100% sea-water 1.5.

Sodium metabolism

See Fig. 3. Plasma sodium was relatively unchanged when 10% sea-water was drunk. On 30 and 50% sea-water, there was a non-significant increase ($P > 0.05$), but the plasma sodium increased significantly on 70 and 100% sea-water ($P < 0.001$ and $P < 0.005$, respectively). Acute drinking of sea-water and sea-water following dehydration also caused increased plasma sodium concentrations ($P < 0.01$ and $P < 0.05$ respectively). The plasma sodium levels of animals returned to tap water were back to normal.

There was an increase in urinary sodium concentrations when sea-water in any concentration was drunk ($P < 0.001$). On 100% sea-water the increase was the greatest, 185% more than that excreted when drinking tap water. Returning to tap water had only a slight effect on urine sodium concentrations.

Total urinary excretion of sodium was greater than tap water in all but one group drinking sea-water. Only animals presented with sea-water following dehydration did not excrete more sodium in their urine. The biggest load of sodium excreted was found in animals drinking 50% sea-water. When animals were returned to tap water there was a decline in urinary excretion of sodium, but still significantly more was secreted than normal ($P < 0.005$).

Creatinine in plasma, urine and clearances

These are given in Table 3. There were fluctuations in plasma creatinine levels. Animals drinking sea-water, after slowly increasing the concentrations of sea-water, showed a decrease, though insignificant, in plasma creatinine concentrations. Drinking 30% sea-water and sea-water after dehydration caused significantly increased plasma creatinine ($P < 0.01$ and $P < 0.005$ respectively).

Urinary creatinine concentrations were all lower when sea-water was provided instead of tap water. The lowest urinary creatinine concentration was following drinking 50% sea-water, a decline of 60% ($P < 0.001$). As the actual urine volume is of prime importance, the actual creatinine clearances are of far greater value than the urinary concentrations.

The clearances were increased when 10% sea-water was drunk. Drinking 50% sea-water led to the highest increases in creatinine clearances, by 120% compared with tap water. Clearances of the animals drinking 100% sea-water were twice those of animals on tap water. Acute drinking of sea-water led to the smallest increase in creatinine clearances, while drinking sea-water following dehydration led to a decreased creatinine clearance. Return to tap water from sea-water led to a return to normal creatinine clearances, although the urinary creatinine concentrations were significantly low ($P < 0.001$).

Table 3. Creatinine handling by animals drinking increasing concentrations of sea-water

	Plasma (mg%)	Urine (mg%)	Clearance (ml/hr)$^{\Delta}$
Tap	0.43 ± 0.04	147 ± 5.9	91
10% sea	0.43 ± 0.04^{NS}	94 ± 10.7***	118
30% sea	0.53 ± 0.02*	106 ± 26.1*	121
50% sea	0.50 ± 0.03^{NS}	59 ± 18.3^{NS}	200
70% sea	0.45 ± 0.03^{NS}	85 ± 23**	130
100% sea	0.28 ± 0.07^{NS}	104 ± 40.4^{NS}	183
Acute sea	0.48 ± 0.1^{NS}	106 ± 20.0^{NS}	107
Dehydrated → Sea	0.65 ± 0.05**	130 ± 23.1^{NS}	43
Sea water → Tap	0.47 ± 0.05^{NS}	92 ± 8.9***	89

Δ = calculated from mean. NS = non-significant. * = significant (when compared with tap water). ** = very significant. *** = highly significant.

Urea handling

This is given in Table 4. Sea-water in any concentration caused a relative decline in plasma urea, although non-significant. Animals drinking increasing concentrations of sea-water had lowered urinary concentrations of urea until 100% sea-water and acute sea-water were drunk. These animals had normal concentrations. Taking into account the volume of urine excreted (total excretion), then it can be seen that only on 10 and 30% sea-water was there a decline in urea excretion, in all other groups there was increased excretion of urea including the animals which were returning to tap water. The urea clearances were higher in all groups drinking sea-water compared with tap water, except on 30% sea-water, which was the same as the control group.

DISCUSSION

When would Man, and presumably other non-marine mammals, drink sea-water? Only when no other source of water is available and then only when the thirst drive is so great that one is compelled to drink sea-water. Normally man will avoid drinking sea-water for as long as possible (Brown *et al.*, 1947; Aubert *et al.*, 1980). Therefore, the dehydrated man driven to drink sea-water will not only have to deal with the salt load in the sea-water, but is already at a disadvantage because of renal changes caused by the loss of water. This was adequately demonstrated in the present communication: Dehydrated animals, who lost 12% of their body wt, drank more sea-water than those acutely exposed to sea-water, but still far less than control animals. As the amount of urine that they produced was almost normal they become even more dehydrated. This resulted in the highest hematocrits and the only group to have severely reduced glomerular function (creatinine clearances). It must be noted that the urea clearance of the dehydrated animals was one of the highest of all the groups.

Drinking sea-water (acute) before being dehydrated was not much better than drinking it only when dehydrated. Because of the taste little water was drunk, while a normal volume of urine was produced. The glomerular filtration and osmol clearances were higher, however, so the salt load was more or less neutralized. This is in contrast to the acute renal failure experienced in near-dry drownings when large amounts of sea-water were swallowed (Grausz *et al.*, 1971; Oren *et al.*, 1982). The difference in metabolism is due to the fact that in dry drownings, the organism is suddenly confronted by a large salt load in the gastrointestinal tract. This exerts osmotic pressure on the body fluids causing a temporary renal shut down. Drinking sea-water involved much smaller volumes being imbibed over a period of 24 hr and so the osmotic stress was less drastic. The food consumption was also far less affected than when drinking after being dehydrated.

When drinking increasing concentrations of sea-water there were two specific modes of reaction. The first type of reaction was found in the groups drinking 10, 30 and 50% sea-water. More and more water was drunk as the concentration increased. Concomitantly the osmol load increased, which necessitated a large volume of urine. On 50% sea-water the urine volumes were over 20% of the animals' body wt. These animals excreted by far the most sodium in the urine and so the plasma sodium levels were virtually unchanged. Therefore, up to this stage the kidneys were able to handle the salt load in the same way as marine birds do with the aid of their nasal glands (Schmidt-Nielsen, 1979). Marine birds force-fed with large volumes of sea-water excreted the entire load

Table 4. Urea handling of animals given varying concentrations of sea-water to drink

	Plasma (mg%)	Urine (mg%)	Urine Excretion (mg)	Clearance$^{\Delta}$ (ml/hr)
Tap	64 ± 16.3	4795 ± 517	278 ± 22	20
10% sea	33 ± 2.9^{NS}	2892 ± 519*	185 ± 40**	47
30% sea	40 ± 3.2^{NS}	1317 ± 334***	133 ± 15***	20
50% sea	38 ± 3.6^{NS}	2325 ± 924*	881 ± 437*	104
70% sea	46 ± 6.1^{NS}	3267 ± 992^{NS}	316 ± 53^{NS}	49
100% sea	54 ± 3.9^{NS}	4920 ± 1489^{NS}	421 ± 99^{NS}	51
Acute sea	41 ± 4.9^{NS}	4292 ± 691^{NS}	439 ± 77*	51
Dehydrated → Sea	49 ± 4.0^{NS}	13140 ± 2204***	543 ± 84***	58
Sea → Tap	56 ± 8.4^{NS}	6600 ± 573*	703 ± 100***	54

Δ = calculated from means. NS = non-significant. * = $P < 0.01$. ** = $P < 0.005$. *** = $P < 0.001$.

through their nasal glands which, in comparison with Man, would have entailed double the urine production that is excreted during maximal water diuresis. Sheep fed fodder containing 30% of the dry wt as salt, also excreted large volumes of diluted urine (unpublished data).

The animals drinking 50% sea-water were the only ones who had diarrhoea. The possible causes of the diarrhoea were either the inability to absorb adequately the large volumes of water or the fact that the large amounts of magnesium and sulphates caused irritation of the bowel. It is highly probable that both factors together led to the diarrhoea.

Urine osmolalities are not always a good indication of renal function. Animals drinking 50% sea-water had the largest osmol uptake. Nevertheless, there was a decline in urine osmolalities, while plasma osmolalities were elevated. This suggests a renal malfunction, but this was not the case. These animals excreted the most osmotically active substances with the largest osmol clearances. This was due to the extremely large urine volumes i.e. osmotic diuresis. Animals imbibing sea-water after being dehydrated had the highest urine osmolalities, but had only a relatively low osmol excretion compared with the amount of osmols taken up. This was due to the small urine volume. Their concentrating capability however, was outstanding; the U/P of 11 is far greater than the capabilities of Man (Pitts, 1968). It would account for the ability of the rat to survive on sea-water longer than Man (Smith, 1951).

Drinking 70 and 100% sea-water led to the second type of reaction to the increasing salt load. There was a decline in water uptake to volumes below those found when drinking tap water in order to limit the salt intake. When confronted with salt water, the marsh mouse, *Peromyscus ribius*, decreased its water uptake by 55% (Fisher, 1962). In contrast, the mountain species of mice, *Peromysus ganbelii*, increased the water intake by almost four-fold, with fatal results. The osmol uptake in the groups of rats getting 70 and 100% sea-water was the same and was more than that of the animals drinking 30% sea-water. There was no diarrhoea. The urine volumes were greater than normal, suggesting a tendency to dehydration. The decline in drinking could have been due to the taste of the water or to a resetting of the thirst mechanism. The latter seems more plausible. The excessive water uptake when drinking 50% sea-water would be self-defeating if the trend continued when drinking 70 and 100% sea-water.

Another factor affecting the drinking mechanism is satiation. Satiation of drinking in the rat, similar to Man, is determined by tissue hydration (Adolph, 1982). This is normally a slow process and therefore water losses are not replaced at once, but over a period of time. Hypernatremia and increased hematocrits suggest a tissue dehydration which would cause an unsatiated thirst. This would also explain the large volumes of water imbibed when drinking 50% sea-water. However, drinking-water is not entirely genetically determined (Adolph, 1982). There are adaptative regulations and it is probable that the decline in water uptake on 70 and 100% sea-water is an attempt to achieve osmoregulation without severely taxing the kidneys or tissue hydration. The excretion of salts is the task of the kidneys alone, as unlike birds, reptiles and marine fishes, no secondary organs are available to selectively excrete the salts. Unlimited water uptake would not improve tissue hydration or hypernatremia. Marine fishes that drink sea-water excrete salt from their gills, but water retention is also enhanced by aglomerular nephrons (Schmidt-Nielsen, 1979). The decline in glomerular filtration when plasma volume declined could be an attempt to conserve water in the same manner. The natriuresis in all groups, except for the animals drinking sea-water following dehydration, was in accordance with what was found in dogs and rats infused with large amounts of sodium (De Wardener *et al.*, 1961; Landwehr *et al.*, 1968).

Marine mammals also rely entirely on their kidneys to excrete any salt load and so avoid drinking sea-water as far as possible (Schmidt-Nielsen, 1979). Sea-water is, however, taken up involuntarily when eating (Depocas *et al.*, 1971). Whales would be able to excrete salts from sea-water even with a water gain, but apparently still do not drink sea-water (Schmidt-Nielsen, 1979).

During osmotic diuresis urine is more dilute than with oliguria (Smith, 1951) due to the osmotic properties of the agent that reduces water reabsorption. The chief osmotically active constituent of urine is urea. Marine animals that do not rely on drinking sea-water and salt excretion for homeostasis, i.e. sharks and rays, utilize urea retention to guarantee body water content (Schmidt-Nielsen, 1979). This mechanism is not specific to sharks and is also found in a salt water crab (Schmidt-Nielsen, 1979), in the lung-fish in the cocoon stage (Forster and Goldstein, 1966), in the dehydrated camel (Yagil and Etzion, 1979), and even in the lactating goat which needs water for milk production (Maltz *et al.*, 1981). The urinary urea loss was 25% lower in lactating goats than non-lactating goats. Rats given salt orally had a more concentrated urine when urea concentration was increased (Smith, 1951). The question is whether rats drinking sea-water in any concentration utilize urea for water retention or for concentrating urine. Rats drinking up to 30% sea-water excreted less urea. When the salt load became greater, increasing the urine volume became paramount and therefore large amounts of urea were excreted.

One of the dangers accompanying sea-water drinking is brain dehydration (Holliday *et al.*, 1968; Arieff and Guisado, 1976). It was shown that it was the short-duration hypernatremia which caused brain dehydration. After 7 days of hypernatremia brain water was normal. The gradual increase in sea-water concentration would probably not lead to brain dehydration as the changes in sodium are gradual.

To date, the only advice given to the ship-wrecked or cast-away on the sea has been to use sea-water only for cooling the body (Brown *et al.*, 1947; Aubert *et al.*, 1980). Although the data presented in the present communication concerns rats, with a higher tolerance for salt than Man, it is suggested that better use can be made of any freshwater sources, namely a gradual increase in sea-water concentration. This will allow for a much longer period before pure sea-water is drunk and then it will be with only minor metabolic discomfort. When returning to fresh

water there wil
metabolism.

Adolph E. F. (
Fedn Proc. Fe
Arieff A. I. and
nervous syste
states. *Kidney*
Aubert M., Papy
physiopatholo
Revue Int. Oc
Bellamy D., Gol
W. and Philli
pp. 53. Black
Brown A. H., G
losses of men
Desert (Edite
science, New
Depocas F., Ha
drinking and
seals, *Phoca v*
De Vries A., F
(1967) Nephr
Congr. Life A
De Wardener H
C. J. (1961)
sodium diures
venous saline
Fisher G. F. (
maniculatus.
Forster R. P. an
lungfish: relat
pathways. *Sc*

water there will be a rapid return to normal renal metabolism.

REFERENCES

Adolph E. F. (1982) Termination of drinking: satiation. *Fedn Proc. Fedn Am. socs exp. Biol.* **41,** 2533–2535.

Arieff A. I. and Guisado R. (1976) Effects on the central nervous system of hypernatremic and hyponatremic states. *Kidney Intern.* **10,** 104–116.

Aubert M., Papy J. J., Aubert J. and Gillet E. (1980) Etude physiopathologique de le survie diun naufrage volontaire. *Revue Int. Oceanogr. Med.* **LVIII,** 81–95.

Bellamy D., Goldsworthy G. J., Highnam K. C., Mordue W. and Phillips J. G. (1975) *Environmental Physiology,* pp. 53. Blackwell Scientific, Oxford.

Brown A. H., Gosselin R. E. and Adolph E. F. (1947) Water losses of men on life rafts. In *Physiology of Man in the Desert* (Edited by Adolph E. F.), pp. 280–314. Interscience, New York.

Depocas F., Hart J. S. and Fisher H. D. (1971) Sea water drinking and water flux in starved and in fed Harbor seals, *Phoca vitulina. Can. J. Physiol. Pharmac.* **49,** 53–16.

De Vries A., Frank M., Sperling O. and Atsmon U. R. (1967) Nephrolithiasis in a subtropical climate. *Proc. Int. Congr. Life Assce Med.,* pp. 132–143. Karger, Basel.

De Wardener H. H., Mills I. H., Clapham W. F. and Hayter C. J. (1961) Studies on the efferent mechanism of the sodium diuresis which follows the administration of intravenous saline in the dog. *Clin. Sci.* **21,** 249–258.

Fisher G. F. (1962) Ingestion of sea water by *Peromysus maniculatus. J. Mammal.* **43,** 416–417.

Forster R. P. and Goldstein L. (1966) Urea synthesis in the lungfish: relative importance of purine and ornithine cycle pathways. *Science* **153,** 1650–1652.

Grausz T., William J. C. and Early E. (1971) Acute renal failure complicating submersion in sea water. *J. Am. Med. Ass.* **217,** 207–209.

Holliday M. A., Kalayci M. N. and Harrah J. (1968) Factors that limit volume changes in response to acute and sustained hyper- and hyponatremia. *J. clin. Invest.* **47,** 1916–1928.

Holmes W. N. (1977) Regulation of nasal gland functions in marine birds. *Proc. Int. Congr. Physiol. Sci., Paris.* **767.**

Landwehr D. M., Klose R. M. and Giebisch G. (1968) Effect of reduction in filtration rate on renal tubular sodium and water reabsorption. *Am. J. Physiol.* **215,** 687–695.

Maltz E., Silanikova N. and Shkolnik A. (1981) Renal performance in relation to water and nitrogen metabolism in Bedouin goats during lactation. *Comp. Biochem. Physiol.* **70A,** 145–147.

Oren A., Etzion Z., Broitman D. and Yagil R. (1982) Renal involvement following near drowning in the sea. *Comp. Biochem. Physiol.* **73A,** 175–179.

Pitts R. F. (1968) *Physiology of the Kidney and Body Fluids,* 2nd edn., pp. 213–226. Year Book, Med. Publ., Chicago.

Schmidt-Nielsen K. (1979) *Animal Physiology. Adaptation and Environment,* 2nd edn., pp. 300–332. Cambridge University Press, Cambridge.

Smith H. W. (1951) *The Kidney. Structure and Function in Health and Disease,* pp. 613–635. Oxford University Press, New York.

Yagil R. and Berlyne G. M. (1975) Health aspects of drinking brackish water. In *Brackish Water as a Factor in Development* (Edited by Issar A.), pp. 247–267. Ben-Gurion University Press, Beer Sheva.

Yagil R. and Etzion Z. (1979) The role of antidiuretic hormone and aldosterone in the dehydrated and rehydrated camel. *Comp. Biochem. Physiol.* **63A,** 275–278.