Decreased urine formation in sodium depleted rats[1]

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Sodium deficient rats show a marked antidiuresis in response to an intragastric water load thus invalidating the hypothesis that increased water intake of hyposalemic animals results from an increased urine formation.

Sodium depletion leads to an increased water intake in man (Gregerson et al, 1951; McCance, 1936), the dog (Cizek et al, 1951; Holmes, 1940; Holmes & Cizek, 1951), the rabbit (Huang, 1955), and the rat (Semple, 1952). The mechanism of this increase remains obscure, although both Verney (1947), and Wolf (1953) have offered explanations in terms of the "osmoreceptor" mechanism assumed to be responsible for normal thirst and the thirst of hypernatremia. Hypernatremic thirst, however, appears to depend on the integrity of the olfactory bulbs (Vance, 1967a), while olfactory bulb resection does not affect hyposalemic thirst (Vance, 1967b), thus implying different mechanisms for the increased water intake observed in hypo- and hypernatremic states.

It is well known that an increase in the extracellular osmotic pressure is a powerful stimulus for the secretion of antidiuretic hormone (Verney, 1947, Woods et al, 1966), and since hyponatremia leads to a reduction of the tonicity of the extracellular fluids, it seems reasonable to suppose that the increased water intake of salt depleted animals results from a reduced antidiuretic hormone secretion with a consequent increase in urine formation leading to greater drinking. The present experiment tests this assumption by examining the rate of urine formation in salt depleted, water loaded rats.

Method

Eight, male, Sprague-Dawley, rats served as Ss, four experimental and four control. The animals weighed approximately 300 g at the start of the experiment, and were housed individually in wire mesh cages fitted with graduated cylinders and drinking spouts for recording water intake. The experimental animals were given an intraperitoneal injection of 5% glucose (10% of body weight) which was removed through a 13 gauge needle 4 h later. A second dialysis was performed six days later following an identical procedure. After the first dialysis, the experimental animals were maintained on a sodium deficient diet (General Biochemicals) for the balance of the experiment. Three weeks following the second dialysis, the animals were transferred to a metabolism cage, and given a 3 ml/100 g body weight water load by intragastric tube under light ether anesthesia. Urine volume and specific gravity was then determined each hour for 3 h following the load. The animals were food and water deprived for 10 h prior to the load, and no food or water was present during the test.

Results

For the last five days preceding the water load, the average daily water intake (ml/100 g body weight) was 14.1 for the experimentals vs 7.9 for the controls, the difference being significant (t-test, $p > .01$). Figure 1 presents the cumulative urine output for the two groups over the 3 h post load period. The mean differences are significantly higher (t-test, $p > .01$) at all points for the controls. In addition, the controls excreted the entire load in less than 2 h while at the end of 3 h the experimentals retained better than 1/3 of the original load. The specific gravity is also everywhere less for the controls than for the experimentals. There was no significant difference in the urine volumes collected over a 24-h period of food and water deprivation for the two groups.

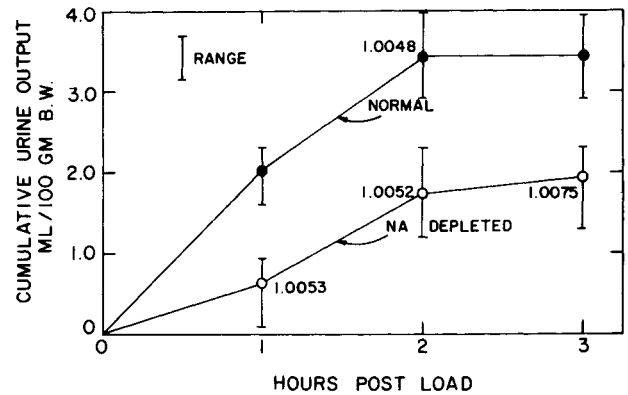

Fig. 1. Cumulative urine output (ml/100 g body weight) for normal and hyponatremic animals following a 3 ml/100 g water load. Numbers near data points indicate urine specific gravity.

Discussion

The results demonstrate that hyposalemic thirst is definitely not due to an increased urine formation and indeed there is a significant impairment in the excretion of a water load by hyposalemic animals. Whether the antidiuresis is a result of an increased antidiuretic hormone production or due to other factors is not clear, although it should be noted that in rats with diabetes insipidus with little or no antidiuretic hormone, an antidiuretic effect is produced by natriuretic drugs and the effect is proportional to the amount of sodium depletion (Skadhague, 1966).

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NOTE

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