THE EFFECT OF HYDRATION ON VASOPRESSIN AND NEUROPHYSIN RELEASE IN THE RAT

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Changes in the neurohypophysial content of arginine vasopressin (AVP) and neurosecretory material in different states of hydration have been reported by many authors (see Jones & Pickering, 1969; Vilhardt, 1970). The present paper reports the effect of hydration on pituitary and plasma levels of AVP and neurophysin in the rat, and on the release of these two peptides in response to haemorrhage.

Four groups of male Wistar rats were studied over a period of 1 week. One group was maintained on an unrestricted water intake (control), another on a restricted water intake, the third on 1-8 % sodium chloride solution and a fourth group was hydrated (water intake equivalent to 25 % body weight/24 h). The rats used weighed 200 g and at least six animals were included in each group. After 1 week the animals were anaesthetized with sodium pentobarbitone (3 mg/100 g) and 0·8 ml blood removed for the determination of neurophysin by a radioimmunoassay which detects both major bovine neurophysins (M. J. Martin, T. Chard & J. Landon, in preparation) and which also cross-reacts with neurophysin in the pituitary and plasma of the rat (Burton, Forsling & Martin, 1971). Subsequently, up to four blood samples, each of 2 ml, were removed at 5-min intervals. The plasma was assayed for neurophysin and for AVP by bioassay (Forsling, Jones & Lee, 1968). The posterior pituitary lobe was then removed and extracted with 0·25 % acetic acid. The extract was assayed for AVP by both bioassay and radioimmunoassay (Edwards, Chard, Kitau & Forsling, 1970), and for neurophysin after denaturation of proteolytic enzymes (Dean, Hollenberg & Hope, 1967). There was good correlation between the results obtained by the two assay techniques for AVP ($r = 0.92$, $n = 22$).

There was no significant difference between the basal levels of circulating neurophysin in the three test groups as compared with the control value of $6.2 \pm 0.7$ (s.e.m.) ng/ml. Neurophysin and AVP levels in the control rats showed a considerable increase after haemorrhage, approximately one molecule of neurophysin being released per molecule of AVP. A significantly reduced response to haemorrhage was found in the other three groups ($P < 0.005$), being least marked in the hydrated rats (Fig. 1). Maximum plasma concentrations with a mean of $75 \pm 24$ µu./ml for AVP and $8.4 \pm 0.3$ ng/ml for neurophysin were found in the saline-treated rats and $106 \pm 42$ µu./ml and $10.0 \pm 0.3$ ng/ml in the dehydrated rats.

The pituitary content of AVP and neurophysin in the controls was $490 \pm 112$ µu.
and 1.88 ± 0.32 µg respectively. In the saline-treated and dehydrated rats, pituitary levels of AVP were reduced by 95% and neurophysin by 60%, so that in these two groups the reduced release of peptides in response to haemorrhage seems merely to reflect the lowered pituitary content. The pituitaries of rats in the hydrated group contained 942 ± 200 µu. AVP and 2.07 ± 0.24 µg neurophysin, higher levels than were found in the controls. Plasma osmolality in this test group was 278 ± 4.5 mosm., as compared with the control value of 303 ± 1.8 mosm. It would therefore seem that release of AVP and neurophysin in response to a potent stimulus such as haemorrhage can be inhibited under conditions of reduced plasma osmotic pressure.

Fig. 1. Plasma concentration of (a) AVP and (b) neurophysin in blood drawn from the carotid artery during progressive haemorrhage in control (●—●) and hydrated (○—○) rats. The vertical bars indicate ± s.e.m.

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