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

MARY L. FORSLING, MARION J. MARTIN 
AND ANGELA M. BURTON

Departments of Chemical Pathology and Physiology, St Bartholomew's 
Hospital Medical College, London E.C.1

(Received 15 June 1971)

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 neuro¬
physin in the three test groups as compared with the control value of 6·2 ± 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 ± 24 µu./ml for AVP and 
8·4 ± 0·3 ng/ml for neurophysin were found in the saline-treated rats and 106 ± 42 µu./ 
ml and 10·0 ± 3·3 ng/ml in the dehydrated rats.

The pituitary content of AVP and neurophysin in the controls was 490 ± 112 µu.
and $1.88 \pm 0.32 \mu g$ 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 $942 \pm 200$ mu. AVP and $2.07 \pm 0.24 \mu g$ neurophysin, higher levels than were found in the controls. Plasma osmolality in this test group was $278 \pm 4.5$ mosm., as compared with the control value of $303 \pm 1.8$ 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.

A.M.B. was supported by a grant from the S.R.C. and M.J.M. by the B.E.C.C. The authors are grateful to Miss Linda Woollard for technical assistance during part of this work.

REFERENCES