Elevated Plasma Concentration of Arginine-Vasopressin in Renal Insufficiency Is Not Caused by Nonspecific Binding of Metabolic Breakdown Products

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Dear Sir,

Plasma concentrations of the antidiuretic hormone arginine-vasopressin (AVP) are elevated in renal insufficiency as we have recently reported in children [1, 2]. Reduced clearance of AVP in renal failure or nonosmotic mechanisms of AVP release have been suggested to cause this AVP elevation [3,4]. Metabolic breakdown products of AVP which circulate in plasma may nonspecifically bind to antibodies used in radioimmunoassay and cause higher AVP concentrations in renal insufficiency by nonspecific binding due to altered AVP metabolism [5]. We therefore examined the elution profile of plasma from 3 patients with end-stage renal failure after separation on high performance liquid chromatography (HPLC).

Blood was taken prior to regular hemodialysis. Plasma was separated immediately and was stored at -4°C until examination. 3–4 ml of plasma were extracted with acetone/petrolether and then separated on HPLC as recently reported for urine [6]. Fractions were collected in 1-min intervals, and the concentrations of AVP were measured in each fraction by radioimmunoassay [7].

Figure 1 shows the elution profiles of 3 patients. The immunoreactive AVP reveals nearly one single peak which corresponds to that of synthetic AVP. Therefore it is evident that our radioimmunoassay is specific for intact AVP, and metabolic fragments of AVP are not as-
Fig. 1. Elution profiles of AVP in plasma from 3 patients with chronic renal failure separated on HPLC. The ordinate shows the amount of AVP in each fraction, the abscissa the number of the fraction. The fraction in which synthetic AVP was eluted is indicated by the arrow.

40–30–20–10

TfT⁵%TfT¹ ! i⁵%ti ifT⁵%Mfl¹%%T⁵T¹

0 5 10 15 20 25 30 35

Fraction number

Arginine-Vasopressin in Renal Insufficiency

521

sayed. This is due to the high specificity of the antiserum used, which was also shown by cross-reaction studies [6]. Using the highly specific radioimmunoassay only intact AVP binds to the

antiserum even in chronic renal failure. The elevated concentrations of AVP in renal insufficiency are therefore not caused by nonspecific binding as consequence of altered metabolism. On the basis of the present results, the mechanism of elevated vasopressin in this disease is not explained and merits further study.

References

Rauh W, Hund E, Soh G, et al: Vasoactive hormones in children with chronic renal failure. *Kidney Int* 1983;24(suppl 15): S27-S33.

Rascher W, Bald M, Schärer K: Vasopressin and atrial natriu-retic peptide in children with chronic renal failure, in Schärer K (ed): *Growth and Endocrine Changes in Children and Adolescents with Chronic Renal Failure*. *Pediatr Adolesc Endocrinol*. Basel, Karger, 1989, vol 20, pp 136–146.

Ardailou R, Pruszczyński W, Benmansour M: Secretion and Catabolism of Antidiuretic Hormone in Renal Failure. *Contrib Nephrol*. Basel, Karger, 1986, vol 50, pp 46–53.

Bald M, Rascher W, Schärer K: Plasma concentration and urinary excretion of arginine vasopressin in children with moderately impaired renal function. *Child Nephrol Urol* 1989; 9:259–263.

Gross P, Rascher W: Vasopressin and Hyponatremia in Renal Insufficiency. *Contrib Nephrol*. Basel, Karger, 1986, vol 50, pp 54–63.

Bald M, Rascher W: Determination and characterization of arginine-vasopressin in extracted and unextracted urine and its urinary excretion in normal children and adolescents. *Horm Res* 1990;34:60–65.

Rascher W, Lang RE, Unger T, et al: Vasopressin in brain of spontaneously hypertensive rats. *Am J Physiol* 1982; 242: H296-H499.