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Unusual sodium and bicarbonate loss in an infant with obstructive uropathy.

The patient, a 50 days old boy, was admitted because severe hyponatremic dehydration and metabolic acidosis. Despite bilateral ureterostomy, chronic administration of high doses of sodium bicarbonate was required.

Functional evaluation was performed separately in left/right kidney at 5 mo. of age:  $\text{Cl}_{18}$ , 16.4/17.4 ml/min/1.73 m<sup>2</sup>. Minimal urine pH, 6.6/6.7.  $\text{E}_{\text{HCO}_3^-}$  at normal serum levels, 16.

9/15.7 %. Fractional distal sodium delivery,  $(\text{C}_{\text{H}_2\text{O}} + \text{C}_{\text{Na}})/100$  ml GFR, 24.7/29.1 ml vs 19.8

ml in controls. Sodium transport at the diluting segment,  $(\text{C}_{\text{H}_2\text{O}}/\text{C}_{\text{H}_2\text{O}} + \text{C}_{\text{Na}}) \times 100$ , 60.6/

56.5 vs 86.7 % in controls. Repeated studies at 17 mo. of age showed no improvement in GFR and in proximal sodium and bicarbonate rejection, but a moderate improvement in distal acidification and in sodium reabsorption at the diluting segment.

This case demonstrates that functional abnormalities in obstructive uropathy of infancy may attain both proximal and distal parts of the nephron, and also that recovery of tubular function may not obligatorily follow desobstruction.

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Studies on Na and K Balance in Bartter's Syndr.

This report concerns two children, one male and one female, fifteen and seven years old respectively, diagnosed as Bartter's Syndrome.

A sodium and potassium balance was done in each one of these two patients under the next conditions: 1) Basal situation; 2) overload of  $\text{ClNH}_4$  at a dose of 200 mEq/m<sup>2</sup>; 3) spironolactone at a dose of 3 mgrs/kg/24h; 4)  $\text{ClK}$  at 10 mEq/Kg/24h; 5)  $\text{ClK}$  at 10 mEq/Kg/24h (Case 1) and 7 mEq/Kg/24h (Case 2), plus  $\text{ClNa}$  at a dose of 4 mEq/Kg/24h; 6)  $\text{ClK}$ , 7 mEq/Kg/24h, plus  $\text{ClNa}$ , 4 mEq/Kg/24h plus Mg gluconate, 6 mEq/Kg/24h.

During each one of these periods, a daily control of plasma, urinary and intracerebrospinal Na and K concentration was done, as well as the daily food intake and faecal excretion; acid-base balance control was also done.

It has been possible to us, to demonstrate the existence of a Na loss syndrome in these two patients. 2.- Under  $\text{ClK}$  overload, these two patients reached the higher plasma K concentration with positive potassium balance, proving, although incompletely, the negative sodium balance existing previously. 3.- In our experience, it seems that the positivation of the Na balance benefits the potassium balance, in both patients. 4.- We can't prove clearly the biogenetic effect of Mg on Na and K metabolism; but we can suppose it, at least, in one of these two patients.

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DDAVP-test for estimation of renal concentration capacity in infants and children.

A new method for estimation of renal concentrating performance by intranasal administration of DDAVP ( 1-deamino-8-D-arginine-vasopressin ) has been tested in 79 children and 25 infants. By comparative studies of different doses of intravenous and intranasal DDAVP it has been possible to elaborate a standard procedure using 20 µg DDAVP in children and 10 µg DDAVP in infants by the intranasal route. The DDAVP-test with none or only moderate short-term fluidrestriction yields urine osmolality values equivalent to those after 22 hours of prolonged dehydration and significantly higher than those after combined pitressin and fluid deprivation test.

In a linear study of the urine concentrating performance postnatally in 28 infants as estimated by the DDAVP-test both lower maximum urine osmolality and shorter DDAVP-response curves are found in preterm and asphyxiated babies. The lastmentioned observation is in agreement with the nephronic heterogeneity with glomerular preponderance and small tubular mass at this age, but also indicates the effect of perinatal asphyxia upon tubular function.

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Circadian periodicity of serum proline in hereditary nephritis.

Circadian rhythm of serum proline was studied in three families with hereditary nephritis. In all members of the families EEG and audiometric investigation were carried out. Proline in the serum was estimated using O'Brien spectrophotometric method. In our laboratory, this method, compared with the method of chromatography on ion exchangers have given a good correlation. In persons with normal values of serum proline their circadian periodicity was displayed with peak values at 12-15<sup>h</sup> and 21<sup>h</sup>, small reversal peak at 18<sup>h</sup> and lowest values at 3<sup>h</sup>. Hyperprolinaemia has not always been easily marked in the members of the families with hereditary nephritis, if proline was estimated in the morning blood samples, but it was expressed in different time intervals during a day or night. Hyperprolinaemia coincides more with kidney disease, but in one family there was a frequent association of hyperprolinaemia and deafness, but without nephritis. These observations indicate that it is necessary to repeat the examinations of serum proline periodically during day and even at night in order to confirm hyperprolinaemia.