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Standard technique for H ion balances were carried out in 6 infants on TPN. Balance periods varied between 4 and 7 days. Blood chemistry and acid-base parameters remained in normal limits during the study period. The mean body input of H ions (1.05 mEq/kg/24 h.) was balanced with the mean net acid excretion (1.16 mEq/kg/24 h.) and SO4 excretion was correlated with N retention. The comparison of our results with the ones obtained in infants on enteral nutrition by other authors revealed that TPN increases the mean urinary organic acid excretion by nearly x 3.5 and that of SO4 by nearly x 6. But quantitatively urinary organic acid constituted the most important fraction of endogenous acid production (4.95 mEq/kg/24 h) and it was balanced with the metabolisable non measured anions in the perfusion (5.46 mEq/kg/24h). It is concluded that an adequate daily nitrogen and calorie intake (400 mg N/kg/24 h and 100 cal/gk/24 h respectively) the use of a well-balanced mixture of cathionic and anionic aminoacids (Vamin) and the intravenous supply of metabolisable non measured manions to compensate the endogenous organic acid production played the most important rôle in the satisfactory H ion balance of our infants.

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Plasma renin activity and aldosterone concentration in children.

Plasma renin activity (PRA) was measured by radio-immunoassay of Angiotensin I(AI) production; plasma aldosterone concentration (P_{Aldo}) was also measured by radio-immuno-assay.

It was found that in healthy children on free diets over the age of 1 year, the upper limit of PRA was about 500 pg AI/ml/hr, but in infants much higher levels were observed, up to 3000 pg AI/ml/hr. There was a positive correlation between PRA and sodium turnover, estimated from the urinary sodium/creatinine ratio. The upper limit of P_{Aldo} in normal

children aged over 1 year was 16 ng/100ml. but in the infants was 60 ng/100ml.

PRA was in the range from 1000-2000 pg AI/ml/ hr in several hypertensive children, without P_{Aldo} necessarily being above the upper limit

of normal. On the other hand, in children with salt-losing states PRA was much greater usually over 10,000 pg AI/ml/hr, and in the majority of these children P_{Aldo} was over

30 ng/100ml.

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Urinary Syclic AMP in renal polyuric disorders.

It is widely held that adenosine-3'-5'monophosphate (cyclic AMP) is a mediator of the hormonal effect of antidiuretic hormone (ADH) in renal tubules. A recent report (J. Clin.End.Metab. 1972,35,35-47) demonstrated a 70 percent increase in urinary cyclic AMP in normals after infusion of ADH. To see if the sensitivity of the method could be increased, we gave ADH together with theophyllamin, an inhibitor of cyclic AMP degradation, but urinary cyclic AMP remained the same as without theophyllamin. We studied normal children and children with renal polyuric disorders: urinary tract abnormalities, interstitial nephritis and inheritable nephrogenic diabetes insipidus (NDI). ADH (15 mU/min) was infused and urinary cyclic AMP and osmolality measured. Patients with NDI do not show increment in either parameter, whereas other patients show increased values in one or both. The measurement of urinary cyclic AMP is of some value in the differential dignosis of renal polyuric disorders. The etiologic diagnosis should, however, be confirmed by renal biopsi and renal X-ray.

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Thromboembolism in the Nephrotic Syndrome. Thromboembolism is a rare but often serious and sometimes fatal complication of the nephrotic syndrome in childhood. An inquiry amongst the members of the ESPN and other European paediatric nephrologists disclosed 60 patients (1.8%) with thromboembolic complications of 3377 children with a nephrotic syndrome during 1962 - 1971. We reviewed anamnestic and clinical as well as pathological-anatomical findings and laboratory data from 56 of these patients.

Thromboembolism may be localized in almost any part of the circulation. The histological examination of the kidneys of 48 patients showed predominantly minimal changes (26 cases) or focal glomerular lesions (15 cases). The children are especially susceptible to this complication during the active phase of the N.S.(22 patients with first manifestation of the disease, 33 cases during relapse) while having generalized massive edema (33 patients). As most of these children have received steroid treatment somewhere during the course of their disease, it is not possible to exclude the possibility, that steroids play an etiological role for this complication. Immobilization and secondary infections as well as hypertension and azotaemia do not seem to be an important predisposing factor.