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CRYOACTIVATION AND ACID ACTIVATION OF AN INACTIVE RENIN IN THE NEWBORN INFANT. S.R. Siegel, A. Hadeed* (Spon. by D.A. Fisher), Dept. of Pediatrics, UCLA Sch. of Med., Harbor General Hospital, Torrance, CA

An inactive large molecular form of renin which can be acid and cryoactivated has been described in human amniotic fluid and plasma. The present study was conducted to determine whether the concentration of inactive renin is high in the newborn at a time when circulating active renin levels are known to be elevated, and whether inactive newborn renin can be separated from active renin by chromatography. 78 samples of newborn plasma were acidified to ph 5.5-5.7, the optimal ph for generating angiotensin I (angio I) from active renin, and treated with Bal and 8-OH quinoline to prevent formation of angiotensin 2. The samples were divided into 2 equal parts, one control for circulating angio I and one generated to angio I at 37°C for 1 hr prior to RIA. The control and generated samples were placed in an ice bath (3-4°C) for 10 hrs. The control samples contained 6.7 ± 1.0 ng/ml/hr (M and SEM) of active renin, and 14.1 ± 1.8 of cryoactivated renin (as PRA). The (37°C, 1 hr) generated samples contained 22.8 ± 1.3 ng/ml/hr of active renin and 17.7 ± 1.7 of cryoactivated renin (as PRA). Chromatography on 1M G-75 Sephadex columns eluted with 0.1M Na Phosphate buffer, ph 6.9 showed active renin in one fraction, M.W. 45,000, and cryoactivated and acid activated (ph 3.0) renin in another fraction, M.W. 67,000. Conclusions: a) active and inactive renin are both in high concentration in newborn plasma, b) cryoactivated and acid activated renin are of similar M.W. and larger than active renin.

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EVIDENCE THAT IMMUNE COMPLEX GLOMERULONEPHRITIS ASSOCIATED WITH INDOLENT BACTERIAL SEPSIS MAY LEAD TO PROGRESSIVE RENAL DISEASE. Roger E. Spitzer, Ann E. Stitzel and Joan R. Urmson. State University of New York, Upstate Medical Center, Department of Pediatrics, Syracuse, NY

Glomerulonephritis (GN) associated with subacute bacterial endocarditis (SBE) or an infected ventricular jugular shunt (IVJS) is due to the development of immune complexes and complement fixation via the classical pathway. This process is thought to be self limiting once the infection is eradicated. Two children, one with SBE and one with IVJS, developed membranoproliferative glomerulonephritis (determined by biopsy) accompanied by azotemia, hematuria, proteinuria and hypocomplementemia (C1, C4, C2, C3). Serum levels of properdin, but not factor B, were also markedly depressed. After elimination of infection by antibiotics (and removal of the V-J shunt in the patient with hydrocephalus), C3 levels rapidly returned to normal. Serum levels of C4 and P, however, remained low for 6 months suggesting continued complement involvement. Hematuria, proteinuria, reduced GFR and hypertension also persisted. Both patients were normocomplementemic by 9 months but even after 2 years had residual proteinuria and hypertension. These data suggest that complement fixation may occur for long periods in the absence of new immune complex formation. During that time, glomerular inflammation apparently continues and may eventually result in irreparable renal damage. (Supported in part by USPHS grant #AI 12721 and New York State Health Research Council grant #352.)

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ROLE OF TUBULAR BASEMENT MEMBRANE AND PERITUBULAR PROTEIN CONCENTRATION IN THE DETERMINATION OF CELL VOLUME IN A HUMAN RENAL PROXIMAL TUBULE. F.Bruder Stapleton and Michael A.Linshaw (Spon.by A.M.Diehl), Univ.Ks.Med.Ctr. Dept.of Pediatrics, Kansas City, Kansas.

It is generally held that renal tubule cell volume is regulated by active cation transport. Ouabain,by inhibiting ATPase blocks Na and K transport with resultant cell swelling. However, the degree of swelling is less than would be expected were active cation transport completely inhibited. Using isolated rabbit proximal straight tubules(PST)we have shown that the transmembrane hydrostatic pressure developed from the constraining effect of the tubule basement membrane(TBM)and the colloid osmotic pressure exerted by bath protein limit the final degree of cell swelling in ouabain(Linshaw et al,AJP 233:325,1977). To evaluate these factors in human kidney,we isolated a single PST with a collapsed lumen from the normal portion of a nephrectomy specimen obtained from a 9 year old girl with unilateral pyelonephritis and hypertension. Both ends were tightly crimped in pipets and tubule viability was confirmed by observing fluid secretion when the tubule was exposed to 1mM PAH. In control serum (6 gm% protein)cell volume as assessed from outer tubule diameter increased 24% upon addition of 10-4M ouabain. Removing bath protein caused 11% more swelling. Removing the TBM with collagenase 500 u/ml caused further swelling and disruption of the tubule. This experiment suggests that in the human kidney hydrostatic and colloid osmotic forces determine at least in part the degree of cell swelling in PST after treatment with ouabain.

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INFANTILE SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) ASSOCIATED WITH CHROMOSOMAL ABNORMALITIES PRESENTING AS NEPHROTIC SYNDROME. Ty, A. and Fine, B.P. (Spon. by F. Behrle), New Jersey Med. Sch., Dept. Pediat., Newark, New Jersey.

SLE in very young infants is rare. Isolated discoid skin lesions, hematologic presentations and cardiac abnormalities have been some of the reported presenting manifestations in SLE in infants and neonates. There has been no report of renal expression as a presentation of the disease in such young infants. The youngest SLE patient who had lupus nephritis on postmortem examination presented with mucocutaneous lesions at 11 mos. of age without any urinary abnormalities. Nonspecific chromosomal aberrations have been reported in 2 cases of neonatal SLE, but without any urinary abnormalities.

We are presenting an unusual case of a young female infant who presented with hematuria and proteinuria at 3 mos. of age and progressed to develop a nephrotic syndrome, failure to gain weight, hepatosplenomegaly, thrombocytopenia, leukopenia, severe anemia and hyperbilirubinemia with positive ANA titres and low serum complement by age 6 mos. She also had a choanal atresia and a peculiar fascies with chromosomal abnormalities characterized by translocation (6q- , 16p+) with some deletion of the transferred #6 chromosome material. Her renal biopsy showed some mesangeal hypercellularity, coarse granular deposits of IgM, IgG and C' on the glomerular basement membranes and epimembranous electrodense deposits with some focal basement membrane thickening.

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AMELIORATION OF COMPENSATORY CHANGES AFTER RELEASE OF UNILATERAL URETERAL OCCLUSION. Kirti Upadhyaya and Norman J. Siegel, Yale Univ. Sch. of Medicine, Dept. of Pediatrics, New Haven, Conn.

Following release of unilateral ureteral occlusion (UUO), the adaptive changes which occur in the contralateral kidney have been poorly documented and the mechanisms involved in these compensatory changes are unknown. In the present study, rats were subjected to 24 hrs of UUO, and adaptive changes in the contralateral kidney were studied after relief of UUO.

Twenty-four hrs after release of UUO, the contralateral kidney had a massive increase in GFR (1020±63 µl/min/100gmBW) and RBF (4552±490 µl/min/100gmBW) compared to sham-operated controls (GFR 595±18; RBF 2745±275, P<0.001 for both). Similar but less impressive compensatory changes occurred in rats with unilateral nephrectomy or unrelieved UUO (GFR 712±39, RBF 3330±227, P<0.05). The infusion of indomethacin (10 mg/kg) completely eliminated the adaptive changes in rats with relieved UUO (GFR 592±63, RBF 2892±245, P<0.001) but had no effect on sham-operated controls (GFR 578±18, RBF 2801±313, P = NS).

These data indicate that impressive adaptive changes in GFR and RBF occur in the contralateral kidney 24 hrs after release of UUO. The inhibition of these compensatory changes by infusion of indomethacin suggests that the increase in GFR and RBF may be mediated via release of prostaglandins from the previously obstructed kidney.

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HYPOPHOSPHATEMIA (HP) IN PEDIATRIC RENAL ALLOGRAFT RECIPIENTS(RAR) Barry Warshaw, Mohammad H. Malekzadeh, Alfred J. Pennisi, Robert B. Ettenger, Christel H. Uittenbogaart & Richard N. Fine, Dept. Pediatrics, USC Sch.Med.& Dialysis & Transplant Program, Childrens Hospital of Los Angeles.

HP following successful renal transplantation has been attributed to either antacid therapy or persistent hyperparathyroidism. The data of 115 RAR were reviewed to assess the relationship between serum phosphate levels and circulating PTH. No patient routinely received antacid therapy. HP (serum PO4 <2.3 mg% >16 years of age; <2.5 mg% 12-16 years; <3.0 mg% 6-12 years; <3.5 mg% <6 years) was common during the initial 2 months posttransplant (PT); 46 of 87 RAR (53%) had HP in the first PT month, and 43 of 87 (51%) in the 2nd month. Between the 2nd and 6th month 10 of 88 (11%) had HP. Transient HP was observed on one or more occasions in 45 of 93 (48%) 6 months to 7 years PT. No patient in this group had persistent HP. PTH levels for the early PT period (<6 months) were available on 53 occasions in 34 RAR (serum creatinine <2.0 mg%), and are as follows:

PTH (Normal <90 µl EQ/ml)	Total No.	No.with low PO4	P	
<90	8	4	NS	NS
90-180	31	21		
>180	14	8	NS	

The incidence of HP did not correlate with the PTH level, and occurred on 4 occasions with normal levels. This data suggests that factors other than PTH and antacids must be implicated in the pathogenesis of HP PT. Since HP >6 months PT was always transient, a direct role for HP in PT osteopenia and growth impairment seems unlikely.