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SILENT LUPUS NEPHRITIS (LN) IN CHILDREN. Thomas L. Kennedy, William Schwartz, David Cornfeld, Camillus L. Witzleben, Michael E. Norman (Spon. Mary Ann South). Univ. of Pa., Dept. of Pediatr., Children's Hospital, Phila., Pa.

Silent LN has been reported in adults (Med. 56:493, 1977). In order to determine its occurrence in children with SLE, 10 who were diagnosed during the past 18 months underwent renal biopsy at the time of diagnosis. Six of 10 had no evidence of renal disease by urinalysis, creatinine clearance, or 24-hour protein excretion, and they are the basis of this report. The mean age was 12 years. The mean duration from onset of symptoms to biopsy was 4 months. Four of 6 had low C3, 5 of 6 had elevated DNA binding, and 3 of 6 received prednisone prior to biopsy. All biopsies had > 10 glomeruli. All had histologic changes on light microscopy: 5 had mesangial proliferation and one had diffuse proliferative LN. On fluorescent microscopy, 4 of 5 had IgG and 5 of 5 had C3 along capillary loops; 2 of 5 had IgG and 3 of 5 had C3 in the mesangium. On electron microscopy, 4 of 6 had mesangial deposits, one had subepithelial and 2 had subendothelial deposits. Mean follow-up was 7 months. Renal function has been preserved in all, but 2 have developed overt nephritis. Analyses of clinical and laboratory data do not distinguish these 6 children from the other 4 presenting with overt nephritis.

Conclusions: 1) Silent LN occurs in childhood as well as in adult SLE. 2) The commonest histologic appearance is diffuse mesangial proliferation with a varying pattern of immune deposits. 3) Findings from early biopsy may be important in planning management and assessing prognosis.

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NEUTROPHIL (PMNS) CHEMOTACTION BY URINE CORRELATED WITH UPPER URINARY TRACT INFECTION (UTI). Abdul J. Khan, Kusum Kumar, Parvin Khan and Hugh E. Evans. Dept. of Ped., Jewish Hosp. & Med. Ctr. of Brooklyn, N.Y.

Infected urine specimens (IU) collected during 13 episodes of UTI (colony count $>10^5$/ml) from girls (mean age 5 1/2 years) and an equal number of sterile normal urine (NU) specimens from uninfected subjects were studied for chemotactic properties. Diagnosis of upper UTI was based on positive antibody coated bacteria (ACB) test. Chemotaxis was performed by modified Boyden's technique utilizing PMNS from healthy volunteers. Cells placed on 3μ micropore filter in the upper chamber were simultaneously tested against 1) Hanks' solution (HS). 2) HS containing 25, 50 and 100μl/ml of urinary supernatant filtrates. 3) Endotoxin activated normal serum (EAS) placed in the lower chamber. The mean (±1SD) chemotactic indices (CI) are presented in the table. IU in all concentrations were more chemotactic than NU

Groups (#)	25/ml	50/ml	100/ml	HS	EAS
ACB + (6)	139 (20)	191 (40)	236 (55)	60 (10)	263 (38)
ACB - (7)	88 (13)	103 (15)	132 (15)	64 (13)	252 (52)
NU - (13)	63 (7)	61 (10)	61 (8)	--	--

and HS ($P < 0.005$). ACB + IU showed higher CIs ($P < 0.005$) than ACB negative IU in each concentration. CIs with 100μl/ml of ACB + IU were comparable to CIs obtained with EAS. PMNS chemotaction may aid in diagnosis of UTI and in differentiation of upper from lower UTI. It may be speculated that higher CIs in upper UTI may be due to unknown chemotactic factor(s) elaborated by renal tissue in addition to those produced by the organisms.

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OXYTOCIN INDUCED NATRIURESIS IN NEWBORN DOGS, Leonard I. Kleinman, Robert O. Banks, University of Cincinnati Cincinnati, Ohio 45267.

Newborns have an attenuated natriuretic response to salt loading, due to the inability of the newborn kidney to adequately reduce tubular sodium reabsorption. When newborn dogs were saline loaded (2 ml/min/kg for 15' then 0.5 ml/min/kg for 2 hours), fractional sodium excretion (F_{ENa}) averaged less than 2% compared to similarly saline loaded adults who had F_{ENa} more than 6% ($p<.01$). Saline loaded hypophysectomized (HPx) adult dogs (n=11), however, had F_{ENa} of only 1.3%, similar to the intact puppy. When given 8mU oxytocin (oxy) IV, these HPx saline loaded dogs increased F_{ENa} to 5.9%, ($p<.01$) similar to intact saline expanded adult controls. Growth hormone, ACTH & ADH had no such effect. Oxy infusion to 7 saline expanded puppies resulted in a 5.9% increase of F_{ENa}, similar to the expanded HPx adult. However, oxy produced <1% increase in F_{ENa} from 7 nonexpanded intact puppies and 3 HPx adult dogs. In both saline expanded and non expanded puppies, oxy produced no consistent effect on K excretion, GFR & blood pressure, but did increase chloride and osmolar excretion. Oxy had no effect in the intact adult dog. The results suggest that a) oxy is essential for an appropriate natriuretic response to saline loading b) The natriuretic effect of oxy occurs only during saline loading and c) a lack of oxy may be responsible for the attenuated natriuretic response to saline loading in the newborn.

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EFFECT OF ALBUMIN ADMINISTRATION ON RENAL FUNCTION IN PREMATURE INFANTS. Khin Swe Lay, Eduardo Bancalari, Herbert Malkus, Rex Baker, and Jose Strauss. Depts. Pediatr. and Pathol., Univ. Miami Sch. Med., Miami.

This study was done to determine effect of salt-poor albumin on blood volume (BV) and renal function in ten premature infants (GA 28-36 weeks, body weight 1.43 ± 0.15 SE kg) with total serum protein (TSP) < 4.5 g/dl. They were given 1 g/kg albumin as 25% salt-poor solution iv in 5-10 min. Urine was obtained in a bag applied to the infant; the bladder was creded after each voiding and when collection periods ended. Initial BV was measured using Evan's blue; changes over a 40-min period were estimated from changes in hematocrit (Hct). Mean arterial blood pressure (MABP) was continuously monitored through an umbilical arterial catheter. Serum and urine osmolarities and creatinines, TSP, colloid osmotic pressure (COP), BV, and MABP were measured and creatinine clearance (C_{Cr}) calculated before and after albumin infusion. An increase in BV from 88 ± 5 to 100 ± 8 ml/kg ($P<.0005$) was first observed 10 min after infusion and then remained unchanged during the study; this was paralleled by increases in TSP from 4.4 ± 0.1 to 4.8 ± 0.1 g/dl ($P<.0005$) and COP from 20 ± 1 to 25 ± 1 cm H_2O ($P<.0005$). However, MABP was not significantly changed. There was an increase in C_{Cr} from .42 ± .09 to .89 ± .17 ml/min ($P<.0005$), but C_{H2O} and U_{osm}/P_{osm} did not significantly change. Apparently, observed changes in MABP, BV, and Hct had a greater influence in increasing GFR than increases in COP had in decreasing it. In conclusion, albumin infusion may help improve GFR in hypoproteinemic premature infants.

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PRESENTATION OF CYSTINOSIS AS BARTTER'S SYNDROME AND CONVERSION TO FANCONI SYNDROME ON INDOMETHACIN TREATMENT. Jacques Lemire, Bernard S. Kaplan and Charles R. Scriver. McGill Univ.-Montreal Children's Hosp. Research Inst., Dept. of Nephrology, and deBelle Lab. for Biochemical Genetics, Montreal, Canada.

A 4 yr girl presented with profound polyuria, clinical features of cystinosis and electrolyte abnormalities of Bartter's syndrome. Phenotypic findings included flaxen hair, growth retardation, polydipsia, polyphagia and salt craving, BP 100/65, photophobia, crystals in conjunctiva, cornea and iris by slit lamp, pigmentary changes in macula, and peripheral retina. She had hypokalemic metabolic alkalosis (K 2.0, Cl 84, HCO_3 24 mEq/L), inappropriate K loss in urine (14 mEq/L), polyuria (up to 1700 ml/24 hr), low urine SG (1.002), serum PO_4 3.2 mg/dl. TRP 72%, generalized aminoaciduria but no glucosuria. WBC free-cystine content 1.1-2.8 ng/mg protein. Peripheral renin activity (PRA) > 50 ng/ml/hr. Indomethacin treatment (2 mg/kg/day) restored serum K to normal, reduced polyuria by 50% and allowed weight gain. After 1 mo, PRA decreased to 3.7 with emergence of hypertension, acidosis and hyperchloremia; serum PO_4 fell to 2.0 mg/dl. Heavy glucosuria and aminoaciduria became constant findings. Electronmicroscopy of renal biopsy revealed cystine in tubular and interstitial cells; unfortunately JGA were not observed in the specimen. The full phenotype of the nephropathic form of cystinosis was thus unmasked by treatment of secondary Bartter's syndrome with indomethacin.

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ESTIMATION OF INTRACELLULAR MACROMOLECULAR OSMOTIC PRESSURE OF RENAL PROXIMAL CONVOLUTED TUBULES (PCT). Michael A. Linshaw and F. Bruder Stapleton (Spon. by Cheng T. Cho), Univ. Ks. Med. Ctr., Dept. of Peds., Kansas City, Kansas.

Renal tubule cell volume is thought to be kept constant by a cation pump. When active transport is blocked by ouabain, cells swell due to intracellular impermeant solutes (π_i) and reach equilibrium when a sufficient transmembrane hydrostatic pressure develops from the constraining effect of the tubule basement membrane (TBM). At equilibrium, $\pi_i = \pi_o + P_i$ (π_o = extracellular oncotic, P_i = intracellular hydrostatic pressure). Since the TBM provides the principal structural support of the tubule, P_i can be estimated because pressure needed to extend outer tubule diameter would be the same whether transmitted against the TBM from the lumen across cells or from within swollen cells. We indirectly estimated π_i in rabbit PCT. Tubules were tightly crimped at one end. Applied intraluminal hydrostatic pressure was set by varying the height of a column of perfusate at the other end. A graph was made to relate applied lumen pressure to outer tubule diameter. We interpolated the pressure necessary to extend outer diameter to that observed in swollen ouabain-treated tubules with collapsed lumens. In 6 gm% protein (π_o = 35 cm H_2O) tubule diameter increased 20% above control corresponding to an intratubular pressure of 6 cm H_2O (P_i). π_i = 41 cm H_2O and would be even higher in normal cells before swelling in ouabain. From these findings, we conclude that effective intracellular macromolecular pressure in normal PCT is probably greater than 41 cm H_2O, a relatively large driving force across mammalian plasma membranes.