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DELAYED PUBERTY IN MALES WITH CHRONIC RENAL FAILURE. Jorge Ferraris, Paul Saenger, Lenore S. Levine, Maria I. New, Brij Saxena, and John E. Lewy. Dept. of Peds., Cornell University Medical College, New York, New York.

The effects of chronic renal failure on the pituitary-testicular axis of 31 males, aged 11.7-20.0 years ($\bar{x}$ 16 y) were studied. Nine patients not on hemodialysis (GI) had serum creatinines between 2 and 10 mg%, 10 patients were on hemodialysis (GII) and 12 patients had received a renal transplant (GIII). Tanner stage of pubertal development was delayed >2SD relative to chronologic age but not to bone age in 79% of patients. Testosterone (T), Δ4-androstenedione (Δ4), dehydroepiandrosterone (DHEA), DHEA sulfate (DS), and urinary 17 keto steroids were normal when related to pubertal stage in Groups I and II. In Group III adrenal androgens (Δ4, DHEA,DS) were decreased while T was normal. Luteinizing hormone levels were normal in all. FSH (normal range 1-6 ng/ml) was significantly increased in GI (11.9 ± 1.5) and GII (30.3 ± 11.5). In GIII FSH was normal in patients with serum creatinines <2 mg%. FSH levels were uniformly elevated in Tanner I-V patients with Cr >5 mg% and also correlated positively with length of time on hemodialysis. Conclusion: FSH is elevated in patients with chronic renal failure even in early adolescence and may reflect damage to germinal epithelium prior to the advent of spermatogenesis, while Leydig cell function appears to remain intact.

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THE EFFECTS OF BODY BURDENS OF LEAD ON THE GROWING RAT KIDNEY. Fine, B.P., Jortner, B., Ty, A. and Gause, D. (Spon. by F. Behrle), New Jersey Medical School, Dept. of Pediatrics, Newark, New Jersey.

Lead induced chronic nephropathy is seen after prolonged occupational exposure to lead and has been produced in experimental animals. There is also suggestive clinical and experimental evidence that development of chronic lead nephropathy may be related to renal growth. Lead acetate was administered by three i.p. injections for a total dose of 125 mg/kg of body weight to 30 Sprague-Dawley rats during the fifth week of life. A control group received i.p. sodium acetate during the same period. One half of each group was studied after eight weeks and the remainder after 32 weeks. At 32 weeks there was a persistent elevation of blood lead concentrations (control 0.138±.04, exp. 0.367±.04 ug/gm; $p < .05$) and renal tissue lead concentrations (control 1.88±.19, exp. 8.2±.93 ug/gm; $p < .05$); however, body weight, kidney weight, and renal tissue RNA and DNA concentrations were not significantly different from the controls. Numerous renal tubular giant cells and intranuclear inclusions were found in the experimental group at 8 weeks but were reduced in number markedly by 32 weeks. There was no evidence of a generalized progressive nephropathy.

These results indicate that after an acute exposure to lead in the growing rat progressive morphologic changes of the kidney are not seen despite persistent elevations of renal lead concentrations.

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CHRONIC HYPONATREMIA AND MIDFACIAL HYPOPLASIA (MFH) Aaron L. Friedman, Russell W. Chesney, William E. Segar. University of Wisconsin School of Medicine, University Hospitals, Department of Pediatrics, Madison, Wisconson.

Persistent hyponatremia (Na 126 mEq/L) was found in a 30 month old girl with typical features of MFH. Hyponatremia, serum hypoosmolality (250 mOsm/L), urine hyperosmolality (U/P osm 3:1) and continued urinary sodium excretion suggested the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Renal, adrenal, pulmonary and drug etiologies for SIADH were excluded. 5 mEq/kg hypertonic Na Cl resulted in less than expected rise in serum Na (115 to 120 mEq/L) and marked increase in urine Na excretion (26 to 95 mEq/L). Water loading (25 ml/kg D_5W) leading to serum osmolality fall from 250 to 227 mOsm/L or volume expansion (25 ml/kg 0.9% Na Cl iv) did not lead to formation of dilute urine (lowest U osm 686 mOsm/L). Also plasma ADH measured by radioimmunoassay did not fall to 0 (lowest value 1.4 μIU/ml). These findings suggested resistance of hypothalamic center to afferent stimuli from volume and osmoreceptors which normally inhibit ADH release. Only fluid restriction (40 ml/kg/24h) successfully raised serum Na to normal. Serum Na returned to hyponatremic levels on 3 days ad lib diet. MFH has been associated with deficiencies of anterior and posterior pituitary hormones. This report documents persistent ADH excess and further suggests hypothalamic abnormalities in MFH.

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PATHOPHYSIOLOGY OF CHRONIC HEREDITARY HYDRONEPHROSIS IN THE RAT. Jeffrey Friedman and John E. Lewy, Dept. of Peds., Cornell University Medical College, New York, New York.

We have developed and reported on a colony of rats with congenital, unilateral (right sided) hydronephrosis (Ped. Res., 11:550, 1977). The hydronephrotic kidneys (HNK) of these animals have decreased clearances of inulin and para-aminohippurate, renal blood flow and filtration fraction (ff), and increased fractional sodium and water excretion. Radiolabelled microspheres (S) were utilized to determine intrarenal blood flow distribution (ID). Ten animals with unilateral HNK and 5 controls were given intraaortic S injections. Clearance studies were similar to our previous report. ID are expressed as % of total renal blood flow for the outer (OC) and inner (IC) halves of the renal cortex adjusted for tissue weight. Control animals had an OC of 69.8 ± 2.1% for left kidneys and an OC of 70.4 ± 3.1% for right kidneys ($p > .2$). HNK animals had a right HNK OC of 76.9 ± 2.3% and a left non-HNK OC of 70.7 ± 2.7% ($p < .025$). This redistribution of renal cortical blood flow toward the OC may be responsible for the decrease in ff previously observed. The resulting diminution of peritubular oncotic pressure might contribute to the increased salt and water excretion from the obstructed kidney relative to its non-hydronephrotic mate.

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CRYOPROTEINS IN PRIMARY GLOMERULOPATHIES. Eduardo H. Garin, Sandra G. Austin and George A. Richard. (Spon. by Elia M. Ayoub), Univ. of Fla., College of Med., Dept. of Pediatrics, Gainesville.

Serial serum samples from 85 children with glomerulopathies and 14 healthy controls were examined for the presence of cryoproteins.

The patients were divided in six groups according to their clinical and glomerular pathologic findings. The incidence and the protein concentration of the cryoprecipitates were determined

Group	# of patients	Incidence	Mean cryoprotein concentration (mg%)
Control	14	10/14	1.75
Minimal lesion			
a) Hematuria	22	21/22	1.46
b) Nephrotic syndrome	25	25/25	2.62
Focal glomerulosclerosis	11	10/11	1.81
Membranous nephropathy	8	8/8	3.58
Acute glomerulonephritis	11	9/11	0.77
Membranoproliferative	9	7/9	2.46

The incidence and concentration of serum cryoprotein in patients with glomerulopathies were not different from the control group, regardless of the immunopathologic findings. No correlation was found between the clinical course and the presence of cryoproteins.

Cryoproteins in patients with glomerulopathies are not indicators of an immunologic mediated disease and do not have a prognostic value.

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PATTERN OF RESPONSE TO PREDNISONE AND IMMUNOPATHOLOGIC FINDINGS IN PATIENTS WITH IDIOPATHIC, MINIMAL LESION, NEPHROTIC SYNDROME. Eduardo H. Garin, William H. Donnelly, and George A. Richard. (Spon. by Elia M. Ayoub), Univ. of Fla. College of Med., Dept. of Pediatrics, Gainesville.

Sixty-seven patients with idiopathic, nephrotic syndrome underwent renal biopsy. All had minimal lesion by light microscopy. Immunopathologic studies for complement (C3) and immunoglobulins (IgG, IgM and IgA) were carried out in all the specimens.

The patients were divided according to their response to prednisone into steroid resistant (11), frequent relapser (39) and infrequent relapser (17). Immunofluorescent findings on renal biopsy were the following:

Category	# of specimens	C3 only	C3 + Ig	Ig only
Resistant	11	1	3	2
Frequent relapser	39	3	4	6
Infrequent relapser	17	-	-	6

There was no correlation between the deposition of immunoglobulins and the pattern of response to prednisone. However, complement (C3) was found only in patients with a steroid resistant or frequent relapsing nephrotic syndrome ($p < 0.05$).