1092

PRIMARY HYPERTENSION (PH) IN CHILDREN AND ADOLESCENTS: A SUB-GROUP WITH BLUNTED PLASMA RENIN (PRA) AND NOREPINEPHRINE (NE) RESPONSE TO POSTURAL STRESS. M.Mentser, E. Lieberman, V. DeQuattro, USC School of Medicine, Depts. of Pediatrics and Medicine, Childrens Hospital of LA, Los Angeles.

PRA, plasma NE, plasma volume (PV) and extracellular fluid volume (ECF) were measured in the supine position and after 3 hours upright posture (PRA and NE) in 13 patients with PH aged 9 to 19 years. Patients were studied in the Clinical Research Center, on regular diets, off antihypertensive medications with simultaneously measured 24 hour urinary sodium. Results expressed as means ± S.E.M.

	no.	SUP PRA (ng/ml/hr)	(% Δ PRA) SUP→UP	SUP NE (ng/l)	ΔNE (ng/l)	PV (ml/kg)	ECF %body wt.
NR	8	0.95±.22	152±24	240±33	+27±13	38±3	25±.9
BR	5	0.85±.21	13±10	206±33	-20±15	50±3	25±1.5
p		NS	<.001	NS	<.05	<.05	NS

8 patients exhibited a normal renin response (NR) to postural stress; 5 patients had a significantly blunted renin response (BR). Age, mean arterial pressure, supine PRA, and supine NE did not differ significantly between the two groups. However, the NE response to postural stress (ΔNE) was significantly less in the BR group. While ECF and 24 hour urinary sodium did not differ significantly between the NR and BR groups; PV was significantly expanded in the latter. These results suggest that some children with PH have a blunted renin and sympathetic response to postural stress. The expanded PV in these patients may contribute to the blunted response and to the maintenance of their hypertension.

1093

NEONATAL NEPHROGENIC DIABETES INSIPIDUS INDUCED BY MATERNAL LITHIUM (Li) CARBONATE USE. Eli M. Mizrahi, Jean F. Hobbs, and David I. Goldsmith. (Spon. by Adrian Spitzer) Albert Einstein Col. of Med. and Bronx Municipal Hospital Center, Department of Pediatrics, Bronx, NY

There are few reports of transplacental Li intoxication none of which describe renal manifestations, although this is well documented in adults. We studied a 3510 gm male neonate born at 35 weeks gestation to a 38 year old woman who ingested 1.8 gm/day of Li carbonate throughout her pregnancy. Polyhydraminos was noted at delivery. At birth the serum Li concentration was 1.0 mEq/L. By the second day the urinary output exceeded 6.4 ml/kg/hr; polyuria persisted even after serum Li concentration fell to zero at four days of age. C_{cr} was normal (4.8 ml/min). Serum and urinary osmolalities were 294 and 100 mOsm/kg respectively; urea provided 17% of the total urinary solute. At 5 days of age the child was subjected to a vasopressin test (50 mU/kg, I.V.). This resulted in an increase in Uosm from 87 to 156 mOsm/kg and a slight decrease in plasma osmolality from 293 to 287 mOsm/kg; urea accounted for 6.1% of the urinary solute. Cyclic AMP excretion rose from 0.25 before to 0.75 nM/min after vasopressin. A similar response was encountered at one month of age. By 2 months, Uosm rose to 597 mOsm/kg following 6 hours of water deprivation. These data indicate that Li intoxication results in similar but longer lasting effects on the kidney of the newborn than that of the adult. The duration of the concentrating defect may be due to enhanced sensitivity of the newborn tubule to Li or to interference with the development of the renal concentrating mechanisms.

1094

IDIOPATHIC HYPERCALCIURIA (IH) IN CHILDREN. E.S. Moore, F.L. Coe, B.J. McMann, and M.J. Favus, Dept. of Pediatrics and Medicine, Pritzker School of Med., University of Chicago at Michael Reese Medical Center, Chicago.

IH occurs in 3-14% of adults and is a major cause of calcium (Ca) renal stones. We studied the prevalence of IH in an unselected population of 273 children and metabolic characteristics of IH in 7 children with symptomatic and asymptomatic IH. Random urine samples from 130 girls and 143 boys were analyzed for Ca/Cr concentration ratios. In 7 children, 3 with Ca stones, additional studies were done. 24-hour urine collections for Ca were obtained while the child was ingesting an ambient diet. After a 15-hour fast, first and second a.m. urine specimens were analyzed for Ca/Cr and cyclic-AMP. Blood was obtained in the fasting state and analyzed for Ca, immunoreactive parathyroid hormone (iPTH) and 25(OH)D_3. Values of urine Ca/Cr ranged from 0.01 to 0.73 mgm/mgm; mean 0.06, 0.062 S.D. Mean Ca/Cr was 0.078 in boys and 0.059 in girls (p<.03). Ca/Cr was not correlated with age for either sex. All 7 children studied in detail were of appropriate height and weight. The blood studies were normal in these children. Serum iPTH and 25(OH)D_3 levels were within normal range. 24-hour urine Ca ranged from 3.25 to 8.65 mgm/kg/24 hrs; mean 5.34. Fasting Ca/Cr decreased substantially in 4 patients but remained greatly elevated in 3. Urine cAMP was slightly elevated in 2 children. These data demonstrate that asymptomatic IH occurs in 2.9-6.2% of children and that IH in children can result from either intestinal overabsorption of Ca or from a renal leak, as is true for adults.

1095

COMPENSATORY RENAL HYPERTROPHY (CRH) IN FETAL LAMBS. E.S. Moore, B.J. McMann, L.B. deLeon, L.S. Weiss and M. Ocampo. Dept. of Pediatrics, Pritzker School Med. University of Chicago at Michael Reese Medical Center, Chicago.

Postnatal response to unilateral nephrectomy (UN) is age related and is greater in the young. We studied CRH in fetal lambs at 85-95 days gestation (term-150). RNA and DNA content in renal cortical slices (C) was measured in fetal kidneys from twin pregnancies, single fetuses after UN, and fetuses after maternal UN. Control studies were in 6 sets of twins; one fetus was delivered and the remaining fetus delivered 72 hrs. later. Left UN in 6 single fetuses was followed 72 hrs. later by renal function studies and removal of the right kidney. The weight of the remaining twin fetus increased by 3.3%; kidney weight (KW) increased 11% (p<.001). RNA and DNA in μgm/mgm C increased by 1.48 and 2.31 respectively (p<.005; p<.001). RNA/DNA fell by 0.09. 72 hrs. after left UN, mean right KW was 8.8 gms compared to 6.2 for the left (p<.005). Mean increase in right KW was significantly higher than that in the control twins (p<.001). Mean RNA and DNA in the left kidney was 2.88 and 7.08 μgm/mgm C compared to 4.28 and 9.75 for the right kidney (p<.005). RNA/DNA rose from 0.41 to 0.49. Control mean urine flow increased significantly after CRH (p<.001). Mean GFR increased from 1.43 to 1.53 ml/min. Mean C_{PAH} increased from 1.74 to 1.85 ml/min. Mean FEphos increased significantly from 1.27 to 3.66 μgm/min (p<.001). These data suggest that significant CRH occurs in utero by both cell proliferation and an increase in cell size, and a significant increase in renal function parallels CRH in the fetus.

1096

COMPENSATED HYPOTHYROIDISM IN CONGENITAL NEPHROTIC SYNDROME (CNS). Madjid Rasoulpour, Robert H. McLean, Norman J. Siegel, Arieh Kauschansky and Myron Genel, Depts of Pediat., Univ. of Conn Med. Sch., Farmington, and Yale Univ. Sch. of Med., New Haven.

Two neonates (M.B. & J.C.) with typical features and histological evidence of Finnish-type CNS had low serum T4 (<6μg/dl) and normal serum TSH (<20μIU/dl) detected by the New England Regional Hypothyroid Screening Program. At 6 and 4 weeks of age serum TSH was elevated (M.B.>300μIU/dl;J.C. 71μIU/dl), T4 was low and FT4 was normal (M.B.3.1 ng/dl;J.C. 4.4 ng/dl). Technetium scan showed normally located thyroid gland in both. The half-life of parenterally administered sodium levothyroxine (150μgIM) was accelerated (M.B.19.8 hrs; J.C. 22.6 hrs) compared to reported T4 half-life of 6-8 days. Urinary thyroxine was elevated in both patients (M.B.114-203μg/gm creat;J.C. 293μg/gm creat; normal <10μg/gm creat). Administration of 20-25μg/Kg/day of oral sodium levothyroxine suppressed TSH values in both. We conclude that despite low serum T4, which may be related to elevated urinary loss of hormonal iodine, normal free T4 is maintained by compensatory hypersecretion of TSH.

1097

ZINC EXCRETION AND PLASMA ZINC LEVEL IN CHILDREN WITH NEPHROTIC SYNDROME. Ekkehard W. Reimold, Univ. of Tex. Health Science Center at Dallas, Dept. of Pediatrics.

According to earlier reports albuminuria is accompanied by an increased excretion of zinc and corticosteroid treatment causes a precipitous fall in serum zinc level. These questions were examined in 92 children with nephrotic syndrome and in 33 controls in whom zinc studies were performed at time of initial diagnosis, during Prednisone treatment and after treatment was terminated. The plasma zinc content was low in all children with active nephrosis, mean 51.7 μg/100 ml, with values as low as 22 μg/100 ml. A slow increase was observed with improvement of the disease. It remained low, however, even in cases of complete and long-lasting remission (69 μg/100 ml).

For all groups the quantitative zinc excretion was not significantly different from the controls except for patients undergoing massive diuresis and those in long-lasting remission. During the polyuric phase our patients excreted up to 2500 μg zinc/24 hrs (mean 1770 μg/24 hrs). The zinc excretion of patients in remission was twice the control value (mean 860 μg/24 hrs). The correlation between plasma and urinary zinc and total protein, albumin and α_2-globulin concentrations was studied also. The hair zinc level was low in all cases of nephrotic syndrome.

A significant urinary zinc loss is not observed in nephrotic syndrome. The low plasma zinc level is not caused by either zinc excretion or corticosteroid administration and can not be explained by changes in serum protein or its fractions. Additional factors will have to be investigated.