1056

RESPONSE OF THE FETAL LAMB KIDNEY TO SOLUTE LOADING. S.S.Daniel, M-N Yeh, L.S.James, Div. of Perinatal Med., Coll. of P & S, Columbia Univ., N.Y.

In order to study the role of the fetal kidney in water and electrolyte homeostasis, the effect of a solute or salt load was examined in the chronically instrumented fetal lamb 113-137 days gestation. Four groups, each of 6 fetuses, received 0.3M NaCl (Group I); 0.6M glucose (Group II) 0.075M NaCl(Group III); or 0.15M glucose (Group IV) administered in a volume of 50 ml/kg over 90 minutes.

Fetuses in Groups I, III and IV excreted 30-40% of the water load while those in Group II conserved water. Changes in urine osmolality, Na and Cl concentrations were much smaller than the corresponding change in urine volume, hence excretion of electrolyte load depended mainly on changes in urine output. The average changes in urine output from control were Groups I and III + 0.06 ml/kg/min, Group IV +0.07, while there was no significant change in Group II. At the end of the infusion changes in plasma osmolality were Group I + 15, Group II+16, Group III-9, and Group IV -6 mOsm/kg. The corresponding changes in Na were +8, -7, -9 and -5 mEq/L respectively. No significant changes in urine osmolality r Na concentration were observed in Groups II and III while changes in Groups I and IV were +32.6 and 37.4 mOsm/kg and +11.8 and -17.0 mEq/ $\ell$ espectively.

These studies show that the kidney of the fetal lamb during the last triester can respond to both a volume and osmolar stimulus and thus make a substantial contribution to both electrolyte and water homeostasis.

1057

IDIOPATHIC RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS WITH C3NeF AND HYPOCOMPLEMENTEMIA. Charles A. Davis

Robert J. Wyatt, A. James McAdams, Paul T. McEnery and Clark D. West. University of Cincinnati College of Medicine, Children's Hospital Research Foundation, Department of Pediatrics, Cincinnati.

Hypocomplementemia with circulating C3NeF occurs in membranoproliferative glomerulonephritis and occasionally in acute poststreptococcal and lupus nephritis. A 7 year old boy with idio-pathic rapidly progressive glomerulonephritis (IRPGN), with a serum C3 level of 16 mg/dl, a circulating 7S factor splitting C3 with the classical pathway blocked (C3NeF), and circulating IgG-containing complexes reactive with Clq, is reported. Cres-cents were present in 50% of the glomeruli. As in normocomplementemic IRPGN, ultrastructure showed subepithelial deposits located exclusively in that part of the basement membrane in apposition to the mesangium (capillary waist) and composed, by immunofluorescence, of C3, C5, and properdin. Following Initiation of therapy with corticosteroid, renal function Following the greatly improved with concurrent fall in the levels of C3NeF and of Clq reactive complexes and rise in C3 concentration. At 8 months, 45% of glomeruli were globally sclerotic and IgG, C3, C5 and properdin could not be demonstrated. The observations emphasize that C3NeF can be present in many types of GN and that hypocomplementemia in RPGN is not necessarily indicative of a streptococcal origin.

INTRARENAL BLOOD FLOW (RBF) AFTER ENDOTOXIC SHOCK 1058 (ES). L.B. deLeon, L.S. Weiss, E.S. Moore, M. Goto, B. Bernheim, B.J. McMann, D.A. Vitullo and P. Chiem-Dept. of Pediatrics, Pritzker School of Med., Uni-

B. Bernheim, B.J. McMann, D.A. Vitulio and P. Chiemmongkoltip. Dept. of Pediatrics, Pritzker School of Med., University of Chicago at Michael Reese Med. Center, Chicago.

The purpose of this study was to determine distribution of IRBF in infant puppies in response to ES. Studies were performed in 18 mongrel puppies age 10-20 days. Cardiac output (CO), total renal blood flow (RBF) and IRBF was determined by dye dilution and the injection of 25 µ radiolabeled microspheres into the left weething. the left ventricle. ES was produced by IV injection of 10 mgm/kg of E coli 0111 B4.

Control	5 min.	20 min.
70	<del>- 72</del>	69
266	131	167
0.47	0.85	0.84
7.88	4.89+	6.00+
4.54	4.34	3.61+
1.39	1.36	1.52+
3.27	3.19	2.38
	70 266 0.47 7.88 4.54 1.39	70 72 266 131 0.47 0.85 7.88 4.89+ 4.54 4.34 1.39 1.36

∆Mean Art. Press. mmHg; \*ml/min/kgBW; #Systemic Art Resistance; oml/min/gm; OC-outer cortex; IC-inner cortex; +statistically significant.

These data demonstrate that RBF falls significantly 5 min. after ES and begins to increase by 20 min. OC and IC flow is initially unchanged; in late ES, IC flow is maintained as a result of a significant fall in OC flow. This response of IRBF may play a role in renal functional response to ES.

1059

URINARY LACTIC DEHYDROGENASE (LDH) ISOENZYME IV AND V IN THE DIFFERENTIAL DIAGNOSIS OF PYELONEPHRITIS

AND CYSTITIS. <u>Udayakumar P. Devaskar</u>, <u>William C. Montgomery</u>. (Sponsored by Joan E. Hodgman). Mount Carmel Mercy Hospital and Medical Center, Dept. of Pediatrics, Detroit, Michigan.

A prospective study was undertaken to determine if urinary LDH isoenzyme assays could differentiate site of infection in patients with urinary tract infection. Thirty children, with a mean age of 6.1 years (23 female and 7 male), were assigned to control, cystitis or pyelonephritis categories based on clinical laboratory and radiologic criteria. LDH isoenzyme concentrations were measured by a thin film agarose electrophoresis (Pol-E-Stret, Pfizer Co.) and expressed as % of I, II, III, IV and V. Of the 10 controls, one had isoenzyme IV and V in 1.2% and 2.3% concentrations. Of Il patients with cystitis, one had isoenzyme IV and V in 2.6% and 1.3% concentrations. However, in all 9 patients with pyelonephritis, there was a significant percentage concentration of LDH IV (13.8  $\pm$  8.08) and V (15.1  $\pm$  9.8) activity in the urine. It was concluded that patients with pyelonephritis had significant concentrations of LDH IV and V iso-enzymes in their urine, unlike normal children or those with cystitis. The source of these enzymes is postulated to be the renal parenchyma itself as modified by the disease process. The diagnosis of site of infection could be made by measuring LDH isoenzyme IV and V concentration in all the patients with urinary tract infection.

1060

DEFECTIVE RENAL TUBULAR FUNCTION IN DIABETIC CHILDREN CAUSING HYPERCALCIURIA AND AN INCREASED URINARY PH. Nick M. Drayer, Rita van Damme-Lombaerts, Catrienus

Rouwé (Spon. by Keith Drummond), Univ. of Groningen, Univ. Hospi tal, Dept. Peds., Groningen, the Netherlands.

In a group of 47 diabetic children 19 had a urinary calcium

excretion above 5.2 mg/kg/24 hr or 2 SD above the mean (2.4 mg/kg/24 hr) of a control group of 58 healthy children. In 13 of these 19 children the hypercalciuria was independent of the degree of glucose excretion and persisted on follow-up. In fasting urine samples the calcium/creatinine ratio of the hypercalciuric group (0.21±0.11, mean ± SD) differed (p<.001) from that of the normo-calciuric group (0.08±0.06), also the urinary pH of the hypercal ciuric group (6.27±0.74) differed (p<.05) from that of the normo calciuric group (5.57+0.40). The blood pH, bicarbonate, glucose and the serum fosfate, ionised calcium, alkaline fosfatase, iPTH, hCT, hGH were not different. After an oral calcium load the urinary cAMP and the TmP/GFR increased (p<.05 and p<.01 respectively) in the hypercalciuric group but not the urinary calcium/ creatinine ratio. Indomethacin lowered (p<.02) the calcium/creatinine ratio in fasting urine samples of 10 hypercalciuric chiliren from 0.32±0.18 to 0.17±0.15 but not that of the normocalciuren ric children. The correlation between the excretion of sodium and the calcium/creatinine ratio was significant in the normocalciuric group, but became only so in the hypercalciuric group after indomethacin administration. It is suggested that prostaglandins contribute to a defective renal tubular function causing hypercalciuria in these diabetic children.

1061

PULMONARY FUNCTION STUDIES IN CHILDREN WITH JEUNE SYNDROME POST-RENAL TRANSPLANTATION. Robert S. Fennell, Gerald M. Loughlin, Eduardo H. Garin,

Abdollah Iravani, William W. Pfaff, R. Dixon Walker, George A. Richard. Univ. of Fla., Col. of Med., Dept. of Peds., Gaines-ville. (Intr. by John A. Mangos)

Three children ages 6 through 10 years with Jeune Syndrome (asphyxiating thoracic dystrophy) and end stage renal failure underwent successful kidney transplantation using parents as donors. They were evaluated by pulmonary function studies 5, 14 and 30 months following their renal transplantation. patients, 2 males and 1 female, exhibited the typical physical and radiologic findings of the syndrome consisting of congenita narrowing of the thorax, shortened ribs and limbs, and abnormalities of the bones of the pelvis and extremities. The patients weighed 14, 16 and 22kg at the time of transplantation. Renal function, post-transplantation, was good to excellent with creatinine clearances ranging from 70cc to 190cc/min/1.73 meters<sup>2</sup> and serum creatinines ranging from 0.4 to 1.2mg%. Pulmonary function studies including lung volumes and flow volume loops were consistent with a pure restrictive pulmonary defect. The patients were followed serially with pulmonary function studies and as somatic growth occurred lung volumes increased proportionately to growth in height. Preliminary data would indicate that renal transplantation is practical and desirable in this syndrome since the restrictive pulmonary disease appears not to be a limiting factor in children surviving infancy.