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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 nomeostasis, 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 1, 111 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 1 + 15, Group 11 + 16, Group 111 -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 or 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/ spectively.

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.

IDIOPATHIC RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS 1057 WITH C3NeF AND HYPOCOMPLEMENTEMIA. Charles A. Davis Robert J. Wyatt, A. James McAdams, Paul T. McEnery West. University of Cincinnati College of Mediand <u>Clark D. West</u>. cine, Children's Hospital Research Foundation, Department of Pediatrics, Cincinnati.

Hypocomplementemia with circulating C3NeF occurs in membranoproliferative glomerulonephritis and occasionally in acute post-streptococcal and lupus nephritis. A 7 year old boy with idioathic rapidly progressive glomerulonephritis (IRPGN), with a serum C3 level of 16 mg/d1, a circulating 7S factor splitting C3 with the classical pathway blocked (C3NeF), and circulating gG-containing complexes reactive with Clq, is reported. Crescents were present in 50% of the glomeruli. As in normocomple-mentemic 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 the Initiation of therapy with corticosteroid, renal function greatly improved with concurrent fall in the levels of C3NeF and of Clq reactive complexes and rise in C3 concentration. At months, 45% of glomeruli were globally sclerotic and IgG, C3, 5 and properdin could not be demonstrated. The observations mphasize that C3NeF can be present in many types of CN and that hypocomplementemia in RPGN is not necessarily indicative of a streptococcal origin.

1058 INTRARENAL BLOOD FLOW (RBF) AFTER ENDOTOXIC SHOCK (ES). L.B. deLeon, L.S. Weiss, E.S. Moore, M. Goto, B. Bernheim, B.J. McMann, D.A. Vitullo and P. Chiem-mongkoltip. Dept. of Pediatrics, Pritzker School of Med., Uni-versity 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 perform-ed in 18 mongrel puppies age 10-20 days. Cardiac output (CO), total renal blood flow (RBF) and IRBF was determined by dye di-lution and the injection of 25 µ radiolabeled microspheres into the left ventricle. ES was produced by IV injection of 10 mgm/ kg of E coli 0111 B4. kg of E coli 0111 B4. Control $\frac{5 \text{ min}}{72}$ 20 min MAP∆ 70 69 266 131 167 C0* SAR# 0.85 0.47 0.84 7.88 **RBF**o 4.89+ 6.00+ 0C o 4.54 4.34 3.61+ ICo 1.39 1.36 1.52 +OC/IC 3.27 3.19 2.38 AMean 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. ES and begins to increase by 20 min. Of and IC flow is initial-ly 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.

INTRARENAL BLOOD FLOW (RBF) AFTER ENDOTOXIC SHOCK

URINARY LACTIC DEHYDROGENASE (LDH) ISOENZYME IV AND 1059 V IN THE DIFFERENTIAL DIAGNOSIS OF PYELONEPHRITIS AND CYSTITIS. <u>Udayakumar P. Devaskar, William C.</u> (Sponsored by Joan E. Hodgman). Mount Carmel Mercy Montgomery. 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 Taboratory 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 11 patients with cystitis, one had isoenzyme IV and V in 2.6% and 1.3% concentrations. However, in all 9 pa-tients with pyelonephritis, there was a significant percentage concentration of LDH IV (13.8 \pm 8.08) and V (15.1 \pm 9.8) activ-ity in the urine. It was concluded that patients with pyelo-nephritis had significant concentrations of LDH IV and V iso-enzymes in their urine. unlike normal children or those with 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.

DEFECTIVE RENAL TUBULAR FUNCTION IN DIABETIC CHILDREN 1060 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 $(0.21\pm0.11, \text{mean} \pm \text{SD})$ differed (p<.001) from that of the normo-calciuric group $(\overline{0.08\pm0.06})$, also the urinary pH of the hypercal siuric 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 respec-tively) in the hypercalciuric group but not the urinary calcium/ creatinine ratio. Indomethacin lowered (p<.02) the calcium/crea-tinine ratio in fasting urine samples of 10 hypercalciuric chil-iren from 0.32+0.18 to 0.17+0.15 but not that of the normocalciu 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.

PULMONARY FUNCTION STUDIES IN CHILDREN WITH JEUNE 1061 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 Univ. Gaines-(asphyxiating thoracic dystrophy) and end stage renal failure underwent successful kidney transplantation using parents as donors. They were evaluated by pulmonary function studies 5, and 30 months following their renal transplantation. The patients, 2 males and 1 female, exhibited the typical physical and radiologic findings of the syndrome consisting of congenital 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 meters² and serum creatinines ranging from 0.4 to 1.2mg% Pulmonary function studies including lung volumes and flow olume 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.