1098

MATURATION OF THE GLUCOSE TRANSPORT PROCESS BY THE FETAL KIDNEY. Jean E. Robillard and Fred G. Smith, Jr. Univ. of Iowa Hosp., Dept. of Peds., Iowa City, Iowa.

Maturation of the glucose reabsorptive process by the fetal kidney was studied in 9 chronic fetal lamb preparations (0.66 to 3.72 kg) and values compared to data obtained from 4 nonpregnant ewes. Blood and urine control values showed a close relationship between fetal glomerular filtration rate (GFR ml/min) and fetal body weight ($r=0.78$, $p<0.02$). The mean value for blood glucose observed at threshold was higher in fetuses (200 ± 13.3 mg/100 ml) than in adult ewes (177 ± 2.8 mg/100 ml). Fetal glucose threshold was correlated to fetal body weight ($r=0.71$, $p<0.05$) and fetal GFR ($r=0.74$, $p<0.025$). The maximum amount of glucose reabsorbed expressed per unit of GFR (Tmg/GFR) was reached in only 1 fetus and was 4.73. In all other fetuses, Tmg was not reached since metabolic acidosis developed during the course of glucose infusion. However, in those fetuses, the highest amount of glucose reabsorbed, expressed per unit of GFR (Tg/GFR), before glucose infusion was stopped, varied $2.32\text{--}3.85$ with a mean of 2.83 ± 0.26 . In the adult ewes Tmg/GFR varied $1.94\text{--}2.47$ and the mean value (2.21 ± 0.12) was significantly lower ($p<0.05$) than the mean Tmg/GFR and Tg/GFR values found in fetuses. These data indicate plasma threshold values for glucose reabsorption by the fetal kidney increased with fetal GFR, suggesting a parallel development in fetal tubular and glomerular function. Moreover, the fact that fetal Tmg/GFR and Tg/GFR were higher than adult Tmg/GFR suggests a non-functional correlate to the documented anatomical glomerular preponderance during fetal life.

1099

PRESENCE OF FUNCTIONING VASOPRESSIN RECEPTORS IN THE FETAL KIDNEY. Jean E. Robillard, Richard E. Weitzman and Fred G. Smith, Jr., Univ Iowa Hosp, Dept Peds, Iowa City, Iowa & UCLA/Harbor Gen Hosp, Dept Med, Torrance, CA.

Since there is no direct evidence that the fetal kidney is responsive to arginine vasopressin (AVP), the effects of AVP infusion (600 μ U/min/kg) were studied in 13 chronic fetal lamb preparations (106–142 days gestation). During infusion, AVP increased from a mean of 1.18 ± 0.35 to 25.97 ± 2.58 μ U/ml ($p<0.001$), free water excretion (CH_2O) decreased, and both osmolality (Uosm) mOsm/KgH₂O and sodium excretion (UNaV) μ eq/min increased. There was no change in GFR ml/min nor total renal blood flow when measured by microspheres. A significant decrease in plasma renin activity (PRA) ng/ml/hr was also observed during AVP infusion.

	GFR	RBF	Uosm	CH ₂ O	UNaV	PRA
Control	3.24	66.7	222	0.17	37.8	10.5
	± 0.93	± 7.1	± 32	± 0.03	± 7.5	± 3.5
AVP	3.59	61.7	341	-0.08	57.6	6.24
	± 0.49	± 9.0	± 22	± 0.02	± 7.7	± 1.9
2P	NS	NS	0.001	0.01	0.05	0.05

Looking at factors modulating the fetal AVP response, a positive correlation was found between Uosm and fetal age ($r=0.85$, $p<0.001$) during AVP infusion. Significant negative correlations were shown between AVP metabolic clearance rate (AVP-MCR) and both fetal age ($r=-0.88$, $p<0.005$) and Uosm ($r=-0.85$, $p<0.01$). These observations suggest the presence of functioning vasopressin receptors in fetal kidneys and that fetal AVP-MCR may modulate the fetal kidney response to AVP.

1100

RENAL GLUCOSE HANDLING IN THE LOW BIRTH WEIGHT (LBW) INFANTS. B.S. Ross, A. Pollak, L. Rubin, R.M. Cowett W. Oh., Brown Univ. Program in Med., Women & Infants Hosp., Dept. of Ped., Providence, R.I.

Glycosuria (GU) which may lead to an osmotic diuresis is often seen in LBW infants receiving glucose infusion during parenteral alimentation presumably due to low renal threshold (RTG) and tubular maximum (TmG) for glucose. This study was designed to determine 1) RTG and TmG in LBW infants and 2) the effects of glycosuria on renal functions. In 9 2–14 day old infants with mean birth weight (bw) of 1125 gms and gestation of 29 wks, plasma glucose titration curves (PGTC) were obtained by stepwise increase in plasma glucose levels (by hourly increments in glucose infusion rate at 10, 12, 14, and 16 mg/kg/min) along with data obtained by timed uncatheterized urine collections. The splays of the PGTC were used to derive RTG (164 ± 6.7 mg/dl) and TmG (0.86 ± 0.1 mg/kg/min). Neither correlated with gestational, conceptual or postnatal age. In parallel studies, 24 LBW infants (similar bw and gestation) developed GU of increasing magnitude during the graded increase in plasma glucose levels. Urine flow, creatinine, osmolar and free water clearances were unchanged but the fractional Na excretion was significantly reduced ($p<0.05$) during increasing degrees of GU (trace to 4+ by Labstix). Our data show that RTG and TmG are low in LBW infants (approximately 1/2 and 1/5 of the reported adult values, respectively) but with the exception of Na handling, glycosuria was not accompanied by alteration in renal functions and in our experiment, osmotic diuresis did not occur.

1101

THE RENAL HANDLING OF PHOSPHATE (P) IN THE NEWBORN CANINE KIDNEY. Janita C. Russo and Martin A. Nash, (Spon. by William Heird), Dept. Ped., College of

Physicians and Surgeons, Columbia University, New York, N.Y. Newborn animals of many species demonstrate hyperphosphatemia (HP) relative to adult animals. We studied the renal response to this relative HP in beagle puppies age 5–10 days. Animals were divided into 2 groups: phosphate loaded group (PL, n=7) received 320 mg phosphorus daily as a neutral P solution by nasogastric feeding; phosphate restricted group (PR, n=8) received aluminum hydroxide gel. After 3 days P titration studies were performed. Control plasma phosphate concentration (PP) was not significantly different between the 2 groups (PL 12.2mg/dl, PR 8.6mg/dl), although the highest values were in the PL group. GFR (PL 1.9, PR 1.4ml/min) and the rate of phosphate reabsorption (PL 0.077, PR 0.085mg/min) were not different and were not correlated with PP. At comparable filtered loads, however, P reabsorption was greater in PR. There were striking differences in P excretion (PL 0.118, PR 0.030mg/min), fractional excretion P (PL 0.605, PR 0.250), and percent reabsorption P (PL 39.8, PR 74.0%). Maximal rates of phosphate reabsorption were similar (PL 15.3, PR 18.1 mg/100ml GFR). Negative values for P reabsorption were seen in 4 animals at very high PP, suggesting tubular secretion of P. The previously described decrease in TmP with continued P infusion may represent decreased net reabsorption due to P secretion. These data demonstrate that the young beagle kidney alters tubular P reabsorption in response to changes in dietary P so as to maintain PP higher than that in the adult. This represents a physiologic response and not renal functional immaturity.

1102

FAILURE OF THE KIDNEY TO VASODILATE IN DIABETES MELLITUS. Robert G. Schacht and David S. Baldwin, New York University School of Medicine, New York, N.Y.

To determine whether the elevated glomerular filtration rates (GFR) and renal plasma flow (RPF) described early in the course of diabetes results from renal vasodilation or an increase in functioning mass, we examined the renal and systemic responses to a renal vasodilator, dopamine, in 10 juvenile diabetic (JDM) (ages 10–23 years), and compared the results to those observed in 10 patients (ages 11–30) who had recovered from an episode of acute poststreptococcal glomerulonephritis (PSGN). The mean pre-infusion values for GFR in JDM were 146 ml/min, RPF 763 ml/min, mean arterial pressure (MAP) 89 mm Hg and renal resistance (R_p) 5563 d/sec/cm⁵. In PSGN mean values for GFR were 124 ml/min, RPF 645 ml/min, MAP 88 mmHg and R_p 6797 d/sec/cm⁵.

In response to dopamine, the diabetics sustained a lesser increase in CIN and CpAH and a greater increase in MAP. No change in R_p occurred in the diabetics, while a decrease of 25% was induced in the PSGN group. Our data confirm that renal hemodynamics are enhanced in young diabetics. Failure of renal resistance to decrease during administration of dopamine supports the thesis that an already vasodilated kidney is responsible for supernormal renal perfusion in these diabetics. The greater increase in MAP during dopamine suggest also a lesser vasodilatory response of the systemic resistance vessels in juvenile diabetes.

1103

INFLUENZA VIRUS IMMUNIZATION IN CHILDREN WITH RENAL DISEASE.

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Thirty-six children (ages 4–15 yr) with renal disease were separated into 3 groups of 12 - Group I, Idiopathic Nephrotic Syndrome (INS) in remission and no therapy; Group II, INS in remission with alternate day maintenance steroid therapy and Group III, nephritides. All were immunized with a single dose of 0.5 ml Influenza virus vaccine, bivalent type A subvirion (Park-Davis & Co). Before and 4 weeks after immunization, sera were tested for serum hemagglutination inhibition (HI) antibody titers to A/New Jersey/76 (Hsw1N1), A/Victoria/75 (H3N2) and A/Port Chalmers/73 (H3N2). Pre-immunization, serum HI antibody titers (1:40) to A/New Jersey was present in one child, to A/Victoria in 10 children (27%) and to A/Port Chalmers in 25 of 34 children (68%). After immunization 31 of 36 children (86%) had 4-fold or more rise in serum HI titers ($p<0.01$). Neither the type of the renal disease nor therapy with prednisone had any effect on the rise of serum HI titers ($p>0.05$). Sixteen of 36 children (44%) had local tenderness and 3 children (8%) had a fever of 39°C. Of the 7 children with pre-immunization proteinuria, 4 children had transient rise in proteinuria after immunization. None required an increased prednisone dose for exacerbation of INS. Children with chronic renal problems should be protected against influenza.