1540 RELATIONSHIP OF ANGIOTENSIN 11 TO INACTIVE RENIN AND RENIN IN THE FETAL LAMB. Sharon R. Siegel and Terry Parkhill, UCLA Hospital and Clinics, Div. of Nephrology, Los Angeles, California.

An inactive form of renin has been found in human and animal blood, kidneys, brain, and amniotic fluid. This inactive renin increases renin activity in vitro upon exposure to acid pH 3.3 followed by alkaline pH 7.4, low temperature, the proteases trypsin and pepsin, and urinary kallikrein. The purpose of this study is to show the effects of angiotensin 11 and saralasin on inactive renin and renin in five fetal lambs 120-130 days gestation, and five lambs 115-138 days, using the chronic sheep preparation. Inactive renin (after cryoactivation and trypsin activation) and renin, after generation of angiotensin 1 are measured by radioimmunoassay. Cryorenin and trypsin activated renin decreased from 4.0 ± 1.0 ng/ml/hr of plasma renin activity (PRA) (M and SEM) and 7.3 ± 1.0 to 0.05 ± 0.01 and 1.7 ± 1.0 ($p<.01$) respectivly, after 15 min of angiotensin 11 infusion (0.25 ug/kg/min); renin did not statistically decrease. Cryoactivated and trypsin activated renin increased from 2.2 ± 0.5 ng/ml/hr and 4.9 ± 0.9 of PRA to 4.6 ± 1.0 and 8.5 ± 1.5, respectively after 30 min ($p<.05$) of the saralasin (10 ug/kg/min) infusion, while renin showed no change. Furosemide increased inactive renin in parallel to renin. In conclusion: 1) angiotensin 11 can inhibit inactive renin, but not renin; 2) a negative feedback loop exists between angiotensin 11 and inactive renin, but not renin; and 3) trypsin activates inactive renin more effectively than cryoactivation; in the fetal lamb.

● **1541** EXAMPLE OF THE EFFICACY OF PREDNISONE IN THE CONTROL OF TYPE I MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS. Roger E. Spitzer and Ann E. Stitzel. SUNY, Upstate Medical Center, Department of Pediatrics, Syracuse, NY.

Controversy exists as to the utility of prednisone in the treatment of membranoproliferative glomerulonephritis (MPGN). Two patients with Type I disease were treated at the onset of their disease with 2 mg/kg/day of prednisone in divided doses for 2 weeks and then maintained on every-other-day therapy in decreasing doses for 4 & 6 years. In the first month, serum levels of C3 and factor B became normal and C3NeF disappeared. In the first 2 years, creatinine clearances increased from 40 & 51 to 95 & 100 cc/min; proteinuria decreased from 8 and 11 to 0.5 and 0.75 grams/24 hrs; and total serum proteins rose from 4.4 & 4.1 to 6.8 and 7.8 g%. Initially, edema was minimized with lasix therapy while blood pressure was controlled with the aid of hydralazine, aldomet, and propranolol. After 3 & 4 years, all medications except prednisone were withdrawn without change in clinical or laboratory findings. After 4 & 6 years, prednisone therapy was tapered and discontinued. Within 30 and 60 days, C3 and factor B levels decreased by 50%, C3NeF returned, creatinine clearances decreased by 44%, proteinuria increased to 4 and 4.5 g/24 hrs, and blood pressure rose to levels greater than the 95th percentile for age. Prednisone was reinstituted on a daily schedule; in 3 months, all findings returned to "near normal" again. These data suggest that prednisone was important in controlling the activity of MPGN in these 2 patients. The possibility that both patients had spontaneous remissions and relapses coincident with the changes in prednisone is unlikely.

1542 TREATMENT OF THE HEMOLYTIC UREMIC SYNDROME (HUS) WITH PLASMA

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HUS is an important cause of acute renal failure in children and its pathogenesis remains unknown. Recently it has been suggested that in both HUS and Thrombotic Thrombocytopenic Purpura there is a defect of an endogenous inhibitor of platelet aggregation, prostacyclin. The infusion of plasma has been reported to be an effective means of reversing this defect. Ten consecutive children with HUS, anuric at the time of diagnosis and who required peritoneal dialysis were treated with daily plasma (5 ml/kg) for a mean of 7.8 days. Their outcome was compared retrospectively with ten consecutive patients, seen over the previous two years, who were also anuric and required peritoneal dialysis, but did not receive plasma. Recovery from the microangiopathic process, as judged by a rising platelet count, occurred after 6.5 days in the plasma treated group and after 6.6 days in those not given plasma. There were no deaths in either group but one patient in the no plasma group has required long term dialysis and two patients in each group have mild chronic renal failure. Six patients in the plasma treated group required dialysis for more than 14 days. These results do not suggest any beneficial effect from plasma infusion in anuric HUS.

1543 ORAL CALCIUM LOADING(OCL) STUDIES IN NORMAL CHILDREN AND IN CHILDREN WITH UROLITHIASIS. F. Bruder Stapleton, H. Norman Noe, Gerald R. Jerkins and Shane Roy, III, (Spon. by George Burghen). Depts. Peds. and Ped. Urology, Univ. Tenn. Ctr. Health Sciences, Memphis, TN.

The pathogenetic role of renal hypercalciuria and absorptive hypercalciuria in children with urolithiasis has not been fully evaluated. Therefore, we examined urine calcium(UCa), creatinine (UCr) and uric acid(UUa) excretion after an overnight fast and following oral calcium(1.0gm/1.73m^2 body surface area) in 47 normal children and in 16 children with calcareous renal stones which were not secondary to infection, immobilization or obstruction. Milk products and supplemental vitamins were restricted for 5 days prior to the study. Urine was collected for 2 hours before and 4 hours after the calcium load and a standardized breakfast. Fasting UCa/UCr was unrelated to sex, race or age in normal children. Results are:

	Normal	Renal Hypercalciuria	Absorptive Hypercalciuria
No. pts.	47	10	3
Age, yrs	3-16	2-15	10-14
UCa/UCr, pre OCL	0.08±0.06*	0.36(0.21-0.58)Δ	0.13(0.08-0.16)Δ
UCa/UCr, post OCL	0.12±0.08*	0.60(0.23-1.09)Δ	0.32(0.3 -0.33)Δ
UUa/UCr, fasting	0.43±0.03*	0.96(0.33-2.11)Δ	0.66(0.2 -0.62)Δ

Serum CO_2 and PTH levels were normal in all patients with hypercalciuria. OCL studies were normal in 3 children with calculi. This OCL study appears to be extremely useful in categorizing children with calcium oxalate urolithiasis and allows specific therapeutic intervention to prevent hypercalciuria in most patients. (mean;*±SD; Δrange)

1544 EFFECTS OF ASPHYXIA ON RENAL FUNCTION IN TERM AND PRETERM LAMBS. B.S. Stonestreet, A. Laptook, S.R. Siegel, and W. Oh, Brown Univ. Prog. in Med., Women & Infants Hosp., Dept. of Ped., Providence, RI

To assess the role of maturity on the renal responses to asphyxia (Asph)16 spontaneously born term (T) and 5 preterm (PT)experimental and 12 control (C) lambs were studied between 1-6 days of age. We procured surviving PT lambs by induction of delivery at 133 days gestation with a fetal glucocorticoid injection. Asphyxia was induced by adding 38 ml/kg of dead space to the airway for 20 min. changing the arterial blood pH to 7.05 in the T, 7.17 in the PT, pO2 to 36 in the T and 29 torr in the PT and pCO_2 to 66 in the T and 62 torr in the PT groups. The studies continued for 2 hrs. following the removal of the dead space. Plasma glucose concentrations increased significantly (peak:M±SE 285±50ml/dl) from baseline in the T during and for 15 min. after Asph. Excreted glucose increased and the percentage of tubular reabsorption of glucose decreased significantly ($p<0.05$), after Asph. Urine volumes, fractional and absolute sodium excretions, and osmolar clearances increased in the PT and T groups during Asph. and significantly ($p<0.05$) 30 min. after the removal of the dead space. No differences were observed between the PT and T groups when these changes were compared. GFR,total renal blood flow (microsphere technique),and plasma renin values did not change significantly during or after Asph. in either the T or PT groups. Changes were not seen in the C lambs. The data indicate that irrespective of maturity, Asph. produces significant hyperglycemia with concomitant natriuresis and osmotic diuresis.

1545 PRIMARY HEMATURIA (PH): PREDICTIVE VALUE OF PERIPHERAL LYMPHOCYTE MARKERS, SERUM IMMUNOGLOBULINS AND RENAL HISTOPATHOLOGY. José Strauss, Michael Freundlich, Violet Esquenazi, Thelma Pardo, Gastón Zilleruelo, Rafael Galindez Helen Gorman, Victoriano Pardo. Univ. of Miami Sch. of Med., Depts. of Pediatrics, Surgery and Pathology, Miami, Fla.

P.H. has been reported as having a good prognosis except in some cases of IgA mesangial deposition (MGN-A) with focal segmental sclerosis and proliferation (FSS) which may progress to ESRD. HLA, lymphocytes bearing surface IgA (SIgA), serum immunoglobulins (Ig) and renal histopathology were evaluated in patients with P.H. persisting $>$ 6 months. HLA-A, B and C were determined in 49 patients (29 MGN-A and 20 MGN-non A) and in 524 controls. In P.H. patients the frequency of Bw35 was 43% (similar in MGN-A and MGN-non A) and 23% in controls ($p<0.01$). Dr7 was present in 8/22 MGN-A (36%) but only in 19/109 (17%) control subjects. SIgA was elevated in 41/42 (98%) patients but only in 3/52 (6%) controls ($p<0.001$). Mean serum IgA and IgM levels were higher in P.H. patients compared to controls. Of 10 Bw35 patients with MGN-A, 9 had FSS (3 of the 9 evolving to ESRD) compared to 8/19 withoug Bw35 ($p<0.01$). It is concluded that 1) HLA-Bw35 is present and SIgA elevated more frequently in patients than in controls, and may be useful in separating P.H. from other hematurias, 2) a positive correlation of these findings with FSS and/or progression to ESRD exists only in MGN-A patients, 3) Dr7 seems increased in MGN-A patients, and 4) some P.H. patients require renal biopsy for better assessment of prognosis.