1111 FOOD SENSITIVITY AND THE NEPHROTIC SYNDROME INCLUDING MEMBRANOUS GLOMERULOPATHY. Douglas H. Sandberg, Rawle M. McIntosh, Charles W. Bernstein, Jose Strauss Dept. of Pediatrics, University of Miami, Miami, Florida and the University of Colorado, Denver, Colorado.

We have reported previously that sensitivity to foods played an important role in six children with frequently relapsing steroid sensitive minimal change nephrotic syndrome (Lancet, in press). These investigations have been extended to include 4 more children with the same disorder and to 2 children, a 15-yr-old WM and 13-yr-old WF with membranous glomerulopathy and the nephrotic syndrome. The latter two patients had not received prednisone. Evaluation of use of food extract injections for diagnostic testing and therapy also was performed. All patients, off prednisone, were admitted to the Clinical Research Center for study. All patients showed decrease in 24 hr. protein excretion on restricted diets, exacerbation of proteinuria with ingestion of specific foods and decreased proteinuria with return to the previous limited diet. Oral challenge with milk produced alteration of plasma C3 in 6 of 7 patients. Serial measurements of serum proteins and immunoglobulins were also made. Two patients were again given milk and after onset of proteinuria, were tested intradermally with serial dilutions of milk extract. A treatment dose was determined and given daily s.q. while ingestion of milk continued. This produced rapid decrease in proteinuria toward normal amounts. Subsequently these patients and 3 previous patients have been treated with food extract injection therapy with improved control of their nephrosis.

1112 PRESSURE NATRIURESIS IN EXPERIMENTAL GLOMERULO NEPHRITIS. Robert G. Schacht, M. Sajjad Iqbal, Gerald Schulman and David S. Baldwin. New York University Schl. of Med. Depts. of Med. and Ped. New York, N.Y. (Spon. by J. Dancis).

To examine possible adaptive mechanisms involved in the maintenance of glomerular filtration and sodium balance in glomerulonephritis, an in situ perfused kidney was compared in 7 normal mongrels (N) and in 6 mongrels 2 to 4 months following the production of mild anti glomerular basement membrane nephritis (GN). The left kidney was perfused via a carotid-renal artery pump at systemic pressure levels and then at an elevated pressure. The average mean systemic pressure was higher in GN (140 mm Hg) than in N (121 mm Hg). Filtration rates (GFR) and renal plasma flows (RPF) were similar, averaging 17.3 and 88 ml/min in GN and 18.6 and 70.3 ml/min in N. When perfusion pressure was elevated to 195 mm Hg mean sodium excretion more than doubled in GN and in N while GFR and RPF decreased by 30% in GN and remained constant in N. The data demonstrate that pressure natriuresis occurs in GN as well as in N and that the GN kidney responds to increased perfusion pressure with exaggerated vasoconstriction as compared to N which autoregulates. The excessive vasoconstrictor response of the GN kidney may be revealing an adaptive vasodilatation which occurs as a mechanism for the maintenance of renal perfusion and sodium homeostasis.

1113 PAH TRANSPORT IN THE PROXIMAL STRAIGHT TUBULES (PST) OF DEVELOPING RABBITS. George J. Schwartz, David I. Goldsmith and Leon G. Fine. (Spon. by Adrian Spitzer). Albert Einstein College of Medicine, Departments of Pediatrics and Medicine, Bronx, New York.

PAH secretion in developing rabbits has been shown to increase as a function of age and to be enhanced by pretreatment with penicillin. This may result from increases in tubular mass, transport capacity/mm (TC) or both. Complete (PST) were dissected from 17 rabbits 8-21 days old, and from 8 penicillin pretreated rabbits 10-13 days old. The tubules were measured within an eye piece micrometer, perfused with simulated late proximal tubular fluid, and bathed in rabbit serum containing 2×10^{-4}M ^{3}H-PAH. PAH secretion was calculated from the appearance of ^{3}H in the collected fluid. The length (L) of PST increased with age: L (μm) = 51.2x (age in days) - 28.4, r = .84, p<.001. Likewise, TC for PAH increased with age: TC (10^{-15} moles/min/mm) = 89.2x -559, r = .59, p<.005. Absolute secretion of PAH/tubule increased 30 fold, 75% of the increment due to TC and 25% to L. Pretreatment with penicillin resulted in a TC of 684±88 (SE) which was 89% greater than the 361 ±84 10^{-15} moles/min/mm found in age matched controls, p<.02; tubular length was not significantly affected, p>.4. Thus, the bulk of the increase in PAH secretion/tubule with age in rabbits results from enhanced TC; this can be nearly doubled by penicillin pretreatment. Changes in tubular length contribute to the development of PAH secretion but play no role in the phenomenon of substrate stimulation.

1114 LATE METABOLIC ACIDOSIS (LMA): A REASSESSMENT George J. Schwartz, George B. Haycock, Chester M. Edelmann, Jr. and Adrian Spitzer, Albert Einstein College of Medicine, Department of Pediatrics, Bronx, New York.

There is substantial controversy regarding the definition and the clinical consequences of LMA. Well, low birth weight (LBW) infants fed standard formula were screened for blood tCO_2 q. 3-4 days, (n=114). Values for the entire group were normally distributed (19.1±2.6 (SD) mmol/l). From 1-21 days of age the mean rose linearly from 18.2 to 19.7 mmol/l and then plateaued. 16/74 infants had "acidosis" arbitrarily defined as tCO_2 <18 mmol/l. Eight of them were matched for maturity and weight and randomly allocated to treatment either with 5% $NaHCO_3$ p.o. to raise tCO_2 to >21 mmol/l (E) or 0.9% NaCl (1 ml/feed) (C). The weight gain was 16.0±3.8 (SE) g/kg/day in E, 17.2±1.4 in C, (p>.7), and 14.4±1.21 in non-acidotic babies. Values for titratable acidity, ammonium excretion, net acid excretion (NAE) or minimum urinary pH attained during ammonium chloride loading were not different in E or C and were similar to those previously reported for non-acidotic infants. Thus: a) values of tCO_2 as low as 14 mmol/l during the first month of life fall within 2 SD for age and cannot be considered abnormal; b) the appropriate NAE for age suggests that blood tCO_2 in LBW infants reflects their HCO_3 threshold; c) LMA did not result in an increased capacity to excrete H^+; d) since no association was found between weight gain and tCO_2, the failure to thrive of LBW infants might be spuriously attributed to low tCO_2.

1115 RENAL BLOOD FLOW AND GLOMERULAR PERFUSION DURING URETERAL OCCLUSION. Norman J. Siegel, Sonia Gunstream, Michael Kashgarian. Depts. of Ped. & Path. Yale School of Medicine, New Haven, Conn.

Although previous studies suggested that decreased cortical perfusion occurred in obstructive nephropathy, technical limitations had prevented the measurement of renal blood flow (RBF) and glomerular perfusion during complete ureteral occlusion in the rat. Using a recent modification of the radioactive microsphere technique for the determination of RBF and Damadian's method for the determination of glomerular counts, both of these parameters were measured after 24 hrs of bilateral ureteral occlusion and after sham operation. In sham operated control animals (n=10), RBF was 5.468±0.241 ml/min/100gmBW (Mean±SEM), and glomerular perfusion in the superficial cortex (SNGP) was 225.8±9.7 nl/min; while in the deep cortex, glomerular perfusion (DNGP) was 199.7± 10.0 nl/min. After 24 hrs of ureteral occlusion (n=9), RBF had fallen 57% to 3.120±0.274 ml/min/100gmBW (P<0.001). SNGP was only 85.1±6.7 nl/min and DNGP was 112.7±8.2 nl/min, 38% and 56% of control values, respectively, (P<0.001 for both).

These data demonstrate that after 24 hrs of ureteral occlusion a) total renal blood flow is markedly reduced, b) glomerular perfusion in the superficial cortex is even more strikingly diminished, and c) the glomeruli in the deep cortex are comparatively better perfused than those in the superficial cortex. These findings would support the hypothesis that cortical ischemia and glomerular hypoperfusion contribute to the renal injury in obstructive nephropathy.

1116 SEQUENTIAL HISTOLOGIC CHANGES IN UNTREATED MESANGIAL-CAPILLARY GLOMERULONEPHRITIS (MCGN). Norman J. Siegel, and Michael Kashgarian, Depts. of Ped. & Path., Yale School of Medicine, New Haven, Conn.

Since recent data have suggested that histologic improvement in MCGN may be associated with longterm steroid therapy, 4 patients with well documented MCGN who had not received steroids or immunosuppressive agents were studied. A second renal biopsy (bx) was performed 3, 3, 4, and 5 years after the initial diagnosis (1st bx). On the 1st bx, all patients had diffuse mesangial proliferation, lobulation, and splitting of the basement membrane: one patient had subendothelial deposits, one patient had no deposits, and 2 patients had mesangial/subepithelial deposits.

On the second bx, there were progressive changes only in the one patient with subendothelial deposits (i.e., increased proliferation and nodular sclerosis). In the other 3 patients, there were either no significant changes (one patient) or a decrease in the degree of mesangial hypercellularity/proliferation (2 patients). Interstitial fibrosis and mesangial sclerosis were seen on the 2nd bx in all 4 patients.

These data suggest that renal histologic improvement (i.e., decreased cellularity/proliferation) may occur in patients with MCGN who are untreated. However, the presence of interstitial fibrosis and mesangial sclerosis suggests that some irreversible changes have occurred.