1510 LONG TERM HEMODIALYSIS (HD) IN CHILDREN AND ADOLESCENTS. Valerie Johnson, Robert A. Weiss and Ira Greifer, Albert Einstein College of Medicine, Dept. of Pediatrics, Bronx, N.Y.

Despite agreement among pediatric nephrologists that renal transplantation is the therapy of choice, successful transplantation is often impossible due to cytotoxic antibodies. Ten pts (mean age at onset HD 10.8 yrs) have been on maintenance HD for more than 4 yrs (mean duration 5.3 yrs). 8 pts. have undergone successful transplantation with grafts functioning less than six months. Vascular access, even in children of 8 kg, has been by fistula exclusively. Mean fistula survival in 9 pts has been 59.7 months although a tenth patient has required 19 access procedures over 6 yrs. Manifestations of osteodystrophy such as epiphyseal slipping, genu valgum and brown tumors have been observed in 8/10, often due to poor compliance with the medication regimen. Linear growth has been poor even when corrected for skeletal age, despite biochemical control of uremia and nutritional counseling. An additional complication has been hemochromatosis (serum ferritin 11,000) in one adolescent and elevated serum ferritins in seven other pts. Promoting psychosocial maturation and rehabilitation through the use of "group therapy" sessions have been successful in achieving full school attendance and realistic career planning in all patients.

●1511 IDIOPATHIC HYPERCALCURIA (IHC) AND GROSS HEMATURIA (GH) IN CHILDREN. Alok Kalia and Luther B. Travis, Univ. of Texas Med. Br., Dept. of Pediatrics, Galveston, Tx.

Although onset of GH prior to calculus formation has been reported, the association of GH and IHC without clinical or radiological evidence of nephrolithiasis has not been described. Six children have been identified who presented with asymptomatic and recurrent GH in whom no calculus could be demonstrated radiographically. Investigations did not reveal any renal or urinary tract pathology which could account for the hematuria. ICH was documented in five; the sixth later passed a calcium oxalate calculus. Five had a family history of renal calculi. Three continued to have recurrent gross and microscopic hematuria with the initial renal calculus developing after 6 months, 5 years and 10 years. Two of the others were started on a thiazide diuretic and the sixth on chlorthalidone soon after onset of hematuria and confirmation of IHC. There was prompt cessation of hematuria without recurrence after 9, 9 and 24 months. We suggest that measurement of urinary calcium excretion as part of the initial evaluation of a child with GH may, in some cases, obviate invasive investigations and allow for effective therapy.

●1512 MONONUCLEAR CELL CHEMOTAXIS IN EXPERIMENTAL INTERSTITIAL NEPHRITIS. Thomas L. Kennedy and Martha Merrow, University of Connecticut Health Center, Department of Pediatrics, Farmington, Connecticut and Michael E. Norman, The Children's Hospital of Philadelphia, Philadelphia.

Because the inflammatory infiltrate in antitubular basement membrane nephritis in guinea pigs (Steblay nephritis) is predominantly mononuclear, the stimulus for monocyte and macrophage recruitment was studied. The tubulointerstitial inflammation begins 10-14 days following injection with heterologous tubular basement membrane antigen. Sera were obtained from the renal vein in order to avoid the difficulties of systemic inactivation or dilution encountered when trying to identify chemotactic activity (CA) from peripheral blood. These samples, as well as renal homogenates, were compared to renal arterial sera for CA using peritoneal mononuclear cells. Sera and homogenates were obtained from nephritic and control animals at several times in the course of the disease. CA is detected in samples obtained from day 7-15 and is maximal at day 10. The CA is heat stable and is most prominent in the renal venous serum suggesting an origin from the kidney. Use of the sera as a chemoattractant for guinea pig polymorphonuclear leukocytes produces no cell recruitment and suggests the CA is specific for mononuclear cells. The alternative pathway of complement has been implicated in the pathogenesis of Steblay nephritis and complement derived chemotactic factors may prove to be responsible for CA. However, measurable complement activation reflected in decreased hemolytic activity (CH50) or in depletion of complement proteins (C3, C4) does not occur.

1513 PERITONITIS IN CHILDHOOD NEPHROTIC SYNDROME. Alan M. Krensky, Warren E. Grupe, Julie R. Ingelfinger. Harvard Medical School. Department of Pediatrics. Children's Hospital Medical Center. Boston, Massachusetts.

A retrospective review (1970-1980) of 310 children with idiopathic nephrotic syndrome revealed 24 episodes of peritonitis in 19 patients (6.9 ± 3.7 years old). None were on cytotoxic drugs and only 64% were on steroids at the onset of peritonitis. All had proteinuria; 23 occurred during a relapse and one was at presentation of nephrosis. No morphologic subtype was at significantly greater risk for peritonitis. Abdominal pain, tenderness, edema, ascites, leukocytosis (22,300 ± 7,700 wbc/mm^3) were universal. Other signs and symptoms included: fever (96%), anorexia (65%), vomiting (65%), diarrhea (52%), and abdominal wall cellulitis (22%); 17/19 had > 1000 wbc/mm^3 of peritoneal fluid. Gram stain was positive in only 7/18. Organisms isolated included pneumococcus (13), E. coli (5), E. coli and B. fragilis (1-with appendicitis), and α-streptococcus (1); 4 were culture negative. Only 1/8 pneumococci typed is not included in the commercial vaccine (type 33). All pneumococci and E. coli were sensitive to all antibiotics routinely tested. Serum IgG levels in 13 episodes measured were dramatically reduced (116±77 mg%) compared to 43 control nephrotics (579±293; $p < 0.001$). Thus, peritonitis is still common in patients with nephrosis (6%); second episodes are frequent (26%); pneumococcus is the most frequent agent (54%) with 7/8 types represented in available vaccine. Patients in relapse, with ascites, edema, and severe hypogammaglobulinemia appear at greatest risk.

1514 ELEVATION OF NEPHROGENOUS CYCLIC ADENOSINE MONOPHOSPHATE (Neph cAMP) AS EVIDENCE OF EARLY RENAL OSTEODYSTROPHY. Alan M. Krensky, Warren E. Grupe, William E. Harmon, Julie R. Ingelfinger, John A. Kirkpatrick. Harvard Medical School, Children's Hospital Medical Center, Departments of Pediatrics and Radiology, Boston, Massachusetts.

To determine at which point in chronic renal insufficiency (CRI) the physiologic conditions for altered bone metabolism appear, radiographs, serum chemistries, parathyroid hormone (PTH), and neph cAMP were evaluated in 25 children with CRI compared to 7 children with benign renal disease and normal renal function:

PATIENT GROUP	n	Cr mg/dl	Ca mg/dl	Phos mg/dl	Alk P mU/ml	PTH µlEq/ml	Neph cAMP nmol/100mlGF
Normal	7	0.6	9.7	3.9	9	50	1.4
CRI	10	5.7	8.6	5.3	160	370	5.4
Hemodialysis	5	9.0	9.5	3.4	94	181	4.6
Transplant	10	2.4	9.7	4.6	76	155	3.3
Normal range		.3-.95	9-11	3.5-4.5	50-125	20-60	0-4

Neph cAMP increases linearly with creatinine (Cr) (r=0.81) and PTH (r=0.89) except for patients on chronic hemodialysis, in whom a metabolic steady state did not exist, or for patients with serum Cr > 8.0mg/dl. Serum Cr appropriate for age and height was universally associated with neph cAMP< 4.0nmol/100mlGF, while neph cAMP was elevated in 9/15 patients with Cr > 1.0mg/dl and all patients with Cr > 3.5mg/dl. Both PTH and neph cAMP were elevated in asymptomatic patients with Cr as low as 1.45mg/dl. Neph cAMP > 4.0nmol/100mlGF is a reliable, non-invasive measure of early changes consistent with the development of renal osteodystrophy even before routine changes are evident.

●1515 SUSTAINED IMPROVEMENT IN GROWTH VELOCITY (GV) & BONE HISTOLOGY (BH) WITH 25OHD$_3$ (25D) THERAPY (RX) IN CHRONIC RENAL FAILURE (CRF). Craig B. Langman, Alice T. Mazur, Roland Baron, Michael E. Norman. Univ. of Pa. Sch. Med. Children's Hosp. of Phila. & Yale Univ. Sch. Med., New Haven. Depts. of Ped. Neph. & Med.

Linear growth failure is a major complication of CRF unique to children and is related to renal osteodystrophy (ROD). 19 children, ages 2.8-16.4 yrs with CRF and BH of osteomalacia (OM), osteitis fibrosa (OF) or both (OFM) were given 1-2 mcg/kg/d of 25D to evaluate effects on ROD & GV. GV was defined by standard deviation scores on Tanner growth charts, the preferred method of expressing growth in pts with CRF. Group GV (mean±SEM) was -1.75±.45 in the pre-Rx year, increasing to +.2±.31 after the 1st Rx yr, and correlated with an increase in serum 25D of 42±8 ng/ml to 244±20 ng/ml (r=.37, p<.002). Pre-Rx BH showed 7 pts with OM, 9 with OFM, & 3 with OF. Pre-Rx GV was similar in the 3 groups, but increased in OM (p<.002) while remaining unchanged in OFM (p=NS) after the 1st Rx year. Data were insufficient to analyze OF pts. BH after the 1st Rx year normalized in 4/7 OM, 2/9 OFM, and 2/3 OF. GV remained unchanged (p=NS) after 1st Rx year over the subsequent 3 Rx yrs in all BH groups despite 7 pts progressing to ESRF. Changes in GV & BH did not correlate with CO_2, PO_4, Ca, iPTH or GFR when pre-Rx levels were compared to 1st or later Rx years. In conclusion: 25D often heals abnormal BH, especially OM; is associated with an increase in GV after 1st Rx year and its preservation in ensuing Rx years. Supported in part by The Upjohn Company.