SCANNING ELECTRON MICROSCOPY (SEM) OF THE MAMMALIAN URINARY TRACT. Joseph A. Burke, Univ. of Kentucky Col. Med., Dept. of Ped., Lexington, Kentucky. (Intr. by Nancy H. Holland).

SEM permits characterization of the topography and individual cell surface of the urinary tract at the ultrastructural level. We have examined the entire urinary tract of the normal monkey, and the fusion of podocyte terminal processes in aminonucleoside-nephrotic rats using SEM.

The podocyte has a central perikaryal region that branches into radiating processes, which give rise to smaller terminal processes that interdigitate with terminal processes of adjacent podocytes. In the proximal convoluted tubule the epithelial cells are covered with elongated closely packed microvilli. In contrast, stub-like microvilli are seen on the cells of the large collecting tubules. Microplications and microvilli are interwoven on the surface of the transitional epithelium of the renal pelvis, the ureters, and the urinary bladder. These cells are polyhedral in shape, variable in size, and demarcated by distinct cell boundaries. The stratified columnar epithelium of the membranous portion of the male urethra is covered with short, stubby microvilli. Partially interwoven microplications characterize the surface of the stratified squamous lining of the female urethra and the distal male urethra. The podocyte alteration in nephrotic rats is seen as a loss of the terminal process interdigitations.

This study demonstrates that SEM provides data on surface topography and ultrastructural morphology that is difficult to obtain using light and transmission electron microscopy.

DEVELOPMENTAL CHARACTERISTICS OF ISOLATED RABBIT RENAL CORTEX MEMBRANES. Russell W. Chesney and Bertram Tacktor. (Intr. by Keith N. Drummond) Nat. Insts. of Health, Nat. Inst. of Child Health & Human Development, Lab. of Molecular Ageing, Baltimore.

Lumen membranes isolated from renal cortex of fetal, 3-5 day, 3-4 week and adult rabbits were compared for trehalase specific activity (SA), D-glucose uptake and by electron microscopy (EM). Brush border membranes were immature in fetal and 3-5 day rabbits but mature in 3-4 week ones by EM. Trehalase, a disaccharidase, is a marker enzyme for tubule brush borders. Its SA expressed as μmoles/min/mg protein X 10^{-2}, and membrane D-glucose uptake at 5 mM expressed as nmoles/min/mg protein, were:

	Fetal	3-5 day	3-4 week	Adult
Trehalase SA	1.67±.04	6.12±1.89	65.33±7.46	73.35±5.6
Glucose uptake		1.77±.06		2.88±.17
	n=6	n=6	n=6	n=6

D-glucose uptake by membranes was examined over the range 0.5 mM-50 mM. Although the Km of glucose uptake by membranes from 3-5 day and adults was similar (24 mM & 21 mM), the Vmax was lower, 8 nmoles vs. 21 nmoles. Membrane glucose uptake was inhibited by phloridzin and N-ethyl malemide. Urine glucose fell from .67 mM in 3 day to .09 mM in 4 week rabbits. These studies indicate that: 1) trehalase SA increases 50X as brush borders develop with age, 2) glucose transport increases as brush borders mature, 3) Vmax increases as urine glucose decreases, 4) trehalase is a marker of these changes in the development of cortex luminal membranes.

URINARY LDH ISOENZYMES IN THE DIFFERENTIAL DIAGNOSIS OF KIDNEY AND BLADDER INFECTIONS. Hugo F. Carvajal, Richard B. Passey, Michael Berger, Luther B. Travis, and William B. Lorentz, Univ. of Texas Med. Br. and Shriner Burns Inst., Depts. of Ped. and Clin. Path., Galveston, Texas.

Urinary LDH isoenzyme assays were performed in patients with proven kidney (N-15) and bladder (N-15) infections as well as normal controls (N-24). Documentation of bladder and kidney infection was accomplished by means of the Bladder Washout Test, culture of ureteric urine (urinary diversion), maximal urine concentration test, clinical symptomatology and radiologic appearance of the urinary tract. U-LDH in normal children (10.8±1.0uU/ml) was significantly lower than in patients with bladder (23.1± 4.4uU/ml), or kidney (231.2±72.2uU/ml) infections ($p<0.05$). In the normal population most of the enzyme activity was comprised among isoenzymes I and II (fast zone pattern). In patients with bladder infection both fast and slow zone patterns were observed, but the actual level of isoenzyme V (4 ± 3.8) was significantly lower than in patients with kidney infections (116±165) where a slow zone pattern was invariably seen ($p<0.05$). Since overlap occured in only one patient, a correct differential diagnosis of kidney and bladder infections would have been possible in 96.6% of the cases.

RENAL RESPONSE OF THE FETAL LAMB TO ASPHYXIA - S.S.Daniel, M.Yeh, E.T.Bowe, L.S.James, Divisions of Perinatology, College of Physicians & Surgeons, Columbia University, Babies Hospital, NY, NY. Fetal lambs chronically catheterized & intact in utero, were asphyxiated by partial occlusion of the umbilical cord. A standard asphyxial insult was given in 4 experiments by inflating the occluder sufficiently to lower fetal heart rate 35±5 BPM, raise blood pressure 18±2mmHg, & lower pH by 0.15 units; these changes were maintained for $\bar{1}$ hour. This asphyxial stress caused a marked fall in urine output from a control rate of 0.17 to 0.03 ml/kg/min; release of the cord was followed by a diuresis. The fall in urine output was accompanied by a rise in solute concentration which continued for 1 to 2 hours following the release of the cord: osmolality rose from 133 to a maximum of 462 mOsm/kg, sodium from 21.5 to 66.8mEq/L, potassium from 6.8 to 35.8 mEq/l, chloride from 17.0 to 52.5 mEq/L, urea nitrogen from 35.5 to 125.0 mg%, ammonia from 1.2 to 4.2 mEq/L & titratable acid from 3.4 to 17.4mEq/L. Because of the low urine output, there was a fall in solute, electrolyte & acid excretion during the occlusion; however, the increase in excretion of these components with diuresis during recovery exceeded the amount retained during occlusion. These experiments show that while the kidney is capable of contributing to acid elimination, this is accompanied by a net loss of water & electrolytes in the urine during recovery from asphyxia. These observations provide an explanation for the hyponatremia seen in infants recovering from severe intrapartum asphyxia.

USE OF 1 ALPHA-HYDROXYVITAMIN D_3 ON CHILDREN WITH CHRONIC RENAL FAILURE. James C M Chan, Susan B Oldham, Michael F Holick & Hector F DeLuca. George Washington Univ Children's Hosp Nat Med Ctr, Dept of Nephrology; Univ of Southern Calif, Dept of Medicine and Children's Hosp of Los Angeles; Univ of Wisconsin, Dept of Biochemistry, Madison, Wisconsin. (Intr by Wellington Hung).

It is now well accepted that the kidney activates 25-hydroxyvitamin D_3 to the highly potent metabolite, 1,25-dihydroxyvitamin D_3. Impaired activation in uremia may contribute to renal osteodystrophy.

Recently, we tested the short-term effects of a highly potent analog, 1 alpha-hydroxyvitamin D_3 (1α-OH-D_3) which is relatively inexpensive to synthesize. Thirty-two mineral balance studies, each comprising a 3 day period, were performed before, during and after oral administration of 1α-OH-D_3 (1-4 micrograms/day) to four children, aged 11 to 16 years, with renal osteodystrophy and stable chronic uremia (BUN > 60mg% and creatinine > 5mg%). Serial determinations of iPTH were measured. The results showed that physiological amounts of 1α-OH-D_3 stimulated increased absorption of calcium in the gastrointestinal tract and promoted positive calcium balances. In addition, serum calcium levels returned to normal and iPTH secretion was suppressed. No complications were encountered.

These data would infer that 1α-OH-D_3 has major therapeutic value in the treatment of renal osteodystrophy.

RECURRENT HEMOLYTIC UREMIC SYNDROME WITH HYPOCOMPLEMENTEMIA. Alfred Drukker, Michael Winterborn, Boyce Bennett, Jacob Churg and Ira Greifer (Intr. by Henry L. Barnett), Dept. of Ped. and Path., Albert Einstein Col. of Med., Bronx, N.Y.

The hemolytic uremic syndrome (HUS) usually occurs as a single, acute episode. Recurrences with intervening periods of complete recovery have rarely been reported. Serum complement levels are stated to be normal. A girl who developed HUS at the age of 2 years had four recurrences during the next 2½ years. Each time she had microangiopathic anemia, thrombocytopenia and oliguric renal failure. During recovery from the third episode, renal biopsy showed focal and segmental thickening of the glomerular capillary walls due to endothelial edema, mesangial proliferation and thickening of the lamina interna rara. Between the acute episodes there were no detectable renal or hematological abnormalities. A transient fall in serum B1C (C3) complement was observed during each of the last three episodes; C1q was depressed during the third, while during the last one, C1q and C4 as well as components of the alternate pathway (C3PA and properdin) were normal. Enquiries to three other clinics revealed that the serum level of C3 was low in 8 of the 17 patients with HUS in whom it was measured. No correlation with course or outcome could be established. We postulate that hypocomplementemia results from non-specific consumption of complement during the acute process of intravascular coagulation. However, the possibility that the complement system is involved in the pathogenesis of certain forms of HUS cannot be excluded.