1105 RENAL CLEARANCE STUDIES WITH INULIN AND PARA-AMINO HIPPURIC ACID IN CYSTIC FIBROSIS PATIENTS.

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Cystic fibrosis (CF) patients (pts) have been reported to have increased capability for renal clearance (C) of antibiotics (A) i e. dicloxacillin. To gain more knowledge of renal function of CF, inulin (I) and para-amino hippurate (PAH) were measured in 12 CF pts with a mean (M) age of 15.6 yrs ± 0.7, range 12-21 yrs, M body surface area 1.21m^2 ± 0.05, range 0.94-1.48m^2. The pts were in 2 groups; i no A, Shwachman and Kulczycki (S) 91-96; ii A S 20-65. I and PAH were given as a load followed by a continuous infusion to maintain constant plasma (P) levels. After equilibration at least 3 C periods were obtained. U was collected by timed spontaneous voidings. P and U were analyzed by specific methodology for I and PAH.

	$C_I$ml/min	$C_I$ml/min/1.73m^2	C_{PAH}ml/min	C_{PAH}ml/min/1.73m^2
(12)	115±8.0	163±7.0	(11) 493±29.9	698±24.3
range	62-156	107-194	299-612	512-774
i	(4)	165±9.5	(4)	729±7.2
ii	(8)	162±6.9	(7)	680±38.2

() number of pts; values mean and SEM. W.H. Smith (The Kidney, 1951) reports for mixed sexes C_I 118±28.1SD (50), C_{PAH} 640±164 SD (40) ml/min/1.73m^2. By non-paired t when each of the 3 groups was compared with Smith's values there was a significant increase $p < 0.001$ in all cases. By non-paired t there was no significant difference between groups i and ii. These investigations suggest that currently recommended drug regimens for CF need re-evaluation

1106 POSTNATAL RENAL MATURATION IN LOW BIRTH WEIGHT INFANTS.

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The postnatal contraction of extracellular fluid in low birth weight (LBW) infants is associated with increased solute excretion. Patterns of postnatal renal maturation were assessed with the assumption that changes in body composition are mediated in part by the developing kidney. Twenty-two appropriate for gestational age, LBW infants, (birth weight m = 1380 gms, gestational ages m=31 wks) were studied between 12 hours and 61 days to evaluate simultaneously glomerular and tubular functional maturation. Since most LBW infants have respiratory morbidities (respiratory distress followed by chronic lung disease) the infants were grouped into: Group I, (13 infants) transient or absent respiratory morbidities and Group II, (9 infants) persistent and severe respiratory morbidities. Creatinine clearance correlated directly with postnatal age in both groups, (Group I r=0.868 p<0.001 and Group II r=0.646 p<0.01) Sodium intake did not vary significantly with age in either group. Percent fractional sodium excretion was inversely related to postnatal age, (Group I r=-0.613 p<0.01 and Group II r=-0.646 p<0.01) and no significant difference was observed between the two regression lines. Increased % fractional sodium excretion in the first 10 days of life may reflect extracellular solute losses and persistent respiratory morbidities did not alter this homeostatic regulation.

1107 EFFECT OF FUROSEMIDE THERAPY ON RENAL FUNCTION IN LOW BIRTH WEIGHT (LBW) INFANT.

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Furosemide is frequently used in LBW infants. We studied its pharmacologic effects in 4 infants (mean gestation 32 wks, birth weight 1.4 Kg) at ages 10-57 days. Furosemide (1 mg/Kg as required clinically) was given intravenously over 1 minute and data collected over the ensuing 24 hours.

	Control	Hours post furosemide therapy				
		0-1	1-3	3-6	6-18	18-24
Ccr*	17±4	30±13	19±6	17±5	23±8	37±17
CH_2O*	0.3±0.2	0.7±0.3	0.7±0.3	0.1±0.1	0.05±0.4	0.5±0.7
COSM*	0.7±0.1	4.1±2**	2.9±0.8**	1.1±0.2**	0.9±0.3	0.8±0.1
V*	0.4±0.1	1.9±0.6**	1.3±0.3**	0.4±0.1	0.4±0.1	0.6±0.3
%FxNa	1.7±0.6	11.5±3.5**	12±2.6**	4.5±1.7	2.9±1.6	0.8±0.4
NaE+	1.7±0.6	21.5±6.7**	14±3.8**	4.2±1.3	2.8±1.5	1.1±0.5
KE+	2.0±0.6	4.6±1.6	4.4±2	5.4±2.8	1.7±0.1	2.5±0.8

±SEM; *ml/min/1.73 m^2, +μEq/Kg/min, **p<0.05.

As shown in table, creatinine clearance (Ccr) was unchanged, urine volume (V), free water clearance (CH_2O), osmolar clearance (COSM), fractional Na excretion (%FxNa), urinary Na and K excretion (NaE, KE) increased with furosemide treatment. The onset of action occurred within 60 minutes, the peak action lasted 3 hrs. and the duration of increased COSM was 6 hrs. Solute loss appeared to be in excess of water loss and postnatal age did not alter this effect. The mechanism, peak and duration of action of furosemide in LBW infants is similar to adult (Cannon et al. Prog. Cardvasc. Dis. 12:99, 1969).

1108 SUGAR TRANSPORT IN ISOLATED NEWBORN RAT RENAL TUBULES: A POSSIBLE EXPLANATION FOR THE ABSENCE OF NEONATAL GLUCOSURIA.

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Although glycosuria is absent in the newborn rat, uptake of galactose and alpha-methyl-D-glucoside (AMG) a non-metabolized transport model for glucose by newborn cortical slices is known to be impaired. We have shown concentration-dependent active uptake of AMG by isolated renal tubules from the newborn Sprague-Dawley rat and have confirmed the validity of this in vitro uptake phenomenon by an in vivo demonstration of AMG uptake by the newborn kidney. A kinetic analysis of the entry phenomenon for AMG in the newborn tubule reveals two distinct membrane transport systems, only one of which is present in the adult tubule. Glucose was shown to competitively inhibit uptake on the shared, high-capacity system, with a K_i of 2 mM in both newborn and adult tubules. The low-capacity system in the newborn (K_m = 0.533 mM) was not affected by either 10 mM fructose or complete anoxia, although both glucose and galactose (10 mM) exerted inhibition on uptake by this system.

These data suggest that the newborn rat shares a sugar-transport system with the adult, but is able to transport less on this carrier per unit time. This deficit is offset by the presence of an additional system with a very high affinity, not present in the adult. The net result is a relatively efficient transport of sugar, impervious to anoxic insult, which approximates that of an adult.

1109 NEONATAL LACTIC ACIDOSIS AND ACUTE RENAL FAILURE: THE ROLE OF PERITONEAL DIALYSIS.

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The treatment of hypoxic lactic acidosis with acute renal failure was studied in 2 neonates requiring peritoneal dialysis. In infant I pre-dialysis blood pH was 7.23, serum HCO_3^- 13 mEq/L, and R fraction $[(\text{serum Na}-(Cl^- + HCO_3^-)]$ 39 mEq/L representing accumulation of lactate, 35 mM/L. After 24 hours of peritoneal dialysis with a commercial solution containing 35 mEq/L sodium lactate, acidosis failed to improve and in fact worsened. HCO_3^- fell to 8 mEq/L while R rose to 47 mEq/L and plasma lactate to 40 mM/L. Increased acidosis was presumably due to loss of bicarbonate into dialysate. After dialysis with a solution containing 40 mEq/L of bicarbonate instead of lactate, acidosis was corrected with blood pH 7.45, serum HCO_3^- 24 mEq/L, and R 22 mEq/L. In infant II predialysis blood pH was 7.18, serum HCO_3^- 12 mEq/L, R 53 mEq/L, and plasma lactate 50 mM/L. Dialysate containing 40 mEq/L of sodium bicarbonate and no lactate was used. With concentration gradient favoring movement of HCO_3^- into and lactate out of extracellular fluid, serum HCO_3^- rose to 19 mEq/L and R fell to 39 mEq/L, blood pH 7.36. Improved circulation and oxygenation permitted metabolism of remaining lactate, producing a transient metabolic alkalosis. In acute renal failure with lactic acidosis, a peritoneal dialysate should be prepared containing sodium bicarbonate rather than lactate. Likewise acetate or other organic anions would not be metabolized effectively in hypoxic states.

1110 ANTIBODY RESPONSE TO A GLOMERULONEPHRITIS RELATED STREPTOCOCCAL ANTIGEN.

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An antigenic protein can be isolated from the water soluble supernatant of disrupted group A streptococci. This antigen (Endostreptosin: ESS) can be detected in the glomeruli of patients with acute glomerulonephritis in the early days of the disease. ESS is not related to known streptococcal exozymes and is not excreted by intact streptococci. Antibody levels to this antigen were studied in 400 individuals of various ages using a microcomplement fixation test. Infants had usually negative titers. 41% of normal children were found to have moderately elevated (above 1:16) ESS antibody although 14% still had no ESS antibody. 90% of normal adults have low (less than 1:16) titers; only 3% have no detectable ESS antibody titer. Symptomatic and asymptomatic patients with acute glomerulonephritis have very high titers (up to 1:258). Patients with recent group A streptococcal infections without signs of nephritis also had consistently elevated titers, but titers were usually not as high as titers found in patients with acute glomerulonephritis. 61% of patients with lipoid nephrosis (minimal change disease) have no ESS antibody independent of activity of disease, steroid therapy, absence of proteinuria and years of remission. ASLO and streptozyme titers do not parallel ESS antibody levels. Penicillin therapy does not influence the emergence of titer elevation.