49 AZOTEMIA, HEMOLYTIC ANEMIA, WITH MINIMAL RENAL MICROANGIOPATHY IN A PATIENT WITH COMBINED IMMUNODEFICIENCY (CID), AND RHEOVIRUS (RV) INFECTION. Bangash, I, John, E.: Univ. of Illinois Hospital, ALSM, Chicago.

Hemolytic uremic syndrome (HUS) has been rarely reported in CID. A 3 month old male infant presented with 3 day history of vomiting, diarrhea, cough and 24 hr. oliguria. Two weeks prior to this episode patient was (pt) admitted and treated for diarrhea, dehydration, oligurea and azotemia. On second admission pt. had 10% dehydration, acidosis and oral thrush. A normal platelet count, coombs negative hemolytic anemia (Hct 33%, retic count 25%, burr cells), elevated BUN (62mg%), and creatinine (12mg%), metabolic acidosis ($CO_2$12mm/l, BE-15), microscopic hematuria and proteinuria were observed on admission. Subsequently the patient developed severe thrombocytopenia, moderate anemia and decreased GFR. These changes normalized in two weeks. A chest roentgenogram revealed pneumonitis and absence of thymus gland. Immunoglobulin electrophoresis and T-cell function were consistent with CID. Stool, CSF fluid and bone marrow cultures were positive for RV. oliguria, azotemia and hyponatremia responded to conservative therapy. However, pt. subsequently developed extensive dermatitis (D), generalized edema and recurrent bouts of diarrhea. Ten weeks later despite hyperalimentation and gammaglobulin therapy pt. expired. Autopsy revealed generalized RV infection, parainfluenza pneumonitis, hypoplasia of thymus, spleen and lymph nodes, graft vs. host D, and minimal glomerular histological changes. Whether mild renal histological changes seen in this pt. with other evidence of HUS is in part due to concomitant CID, is a matter of conjuncture.

DEVELOPMENTAL NEPHROLOGY

50 THE RENAL RESPONSE TO CHRONIC ALKALI LOADING IN INFANTS. *Mathéová E.,Tischler V.,Pavkovčeková O.,Beňo P.,Fedorová E.* University Children's Hospital, Košice, CSSR.

The present observations were undertaken to establish the response of infants to the chronic alkali loading for the interpretation of the abnormalities in the maintenance of acid-base homeostasis in renal tubular alkalosis.

Twelve normal infants aged 3-12 months were studied before and during 5 days of $NaHCO_3$ administration, 157,0 mM/1,73 m^2/day. The blood acid-base parameters during alkalization showed the mild compensated metabolic alkalosis. Fractional Na reabsorption / T_{Na}/F_{Na}/ providing the indirect evaluation of the degree of ECF volum expansion remained unchanged during the alkali loading period. Urinary pH values increased to 7,278 ± 0,202 concomitantly with the increase of the mean fractional HCO_3^- excretion $FE_{HCO_3^-}$ = 4,16 ± 1,62%. The changes of the means of fractional HCO_3^- reabsorption from the control values $\Delta T_{HCO_3^-}/\Delta F_{HCO_3^-}$ were 95,1% in the first day and decreased to 69,9 and 65,2% in the 3th and 5th loading day resp. The renal excretion of H^+ in both forms as TA and NH_4^+ decreased during alkali loading from the mean control value of 119,5±21,9 to 56,8±12,5 uM/C_{cr}.100. The renal acid-base parameters interacted with the urinary excretion of the citric acid cycle intermediate, α-ketoglutarate. $U_{\alpha KG}V$ varied inversely with the $U_{H^+}V$. The index of the mean values of $U_{\alpha KG}V/U_{H^+}V$ rosed from 0,39 ± 0,12 in control to 2,80 ± 0,25% in alkali loading period.

The presented results showed the measure of the normal efficiency with which the kidney protected the organism against accumulation of excess base during chronic $NaHCO_3$ administration in infants.

51 NET ACID EXCRETION DURING FIRST WEEK OF LIFE. Chan, L.L., Balfe, J.W., Exeni, R., Cifuentes, R.F., Bryan, M.H. The Hospital for Sick Children, University of Toronto, Toronto, Canada.

Metabolic acidosis occurs frequently in newborns. We have studied 34 preterm and 12 term infants during their first week of life. Twenty preterm infants were nourished with breast milk or SMA (Wyeth^R); the remaining infants received total parenteral nutrition (TPN) - synthetic amino acids or casein hydrolysate solution. Net acid excretion (NAE) for breast milk vs. SMA fed infants was 5.4 ± 0.4 and 7.8 ± 0.6 uEq/min/m^2 (mean ± SEM). The corresponding values for the two TPN solutions in preterm infants were significantly higher at 12.5 ± 1.4 and 19.4 ± 3.5 uEq/min/m^2. The casein group of infants developed acidosis with depressed plasma bicarbonate 17.4 ± 2 mEq/l. Term infants treated with the TPN solution produced even greater amounts of net acid, 20.6 ± 2.9 for synthetic amino acids and 35 ± 3.7 uEq/min/m^2 for casein hydrolysate. Because of the net base generated from milk consumption, milk fed infants are less prone to develop metabolic acidosis. Due to its inherent acidogenic effect, TPN solutions induce acidosis more readily. Preterm infants receiving TPN are therefore required to generate a higher NAE rate compared to the milk fed infants to maintain acid-base balance. With a similar quantity of TPN intake, the term infants produced higher urinary ammonium, thus NAE.

52 EFFECT OF INTRAVENOUS INDOMETHACIN ON RENAL FUNCTION OF PREMATURE INFANTS. John, E.G, Bhat, R., Vasan, J, Vidyasagar, D, Hastrieter, A.R. Univ. of Ill. and Pres.-St. Lukes Hospital, Chicago, Illinois, U.S.A.

Ten infants with respiratory distress syndrome and large patent ductus arteriosus gestational ages 27-35 weeks, birth weights 0.9 2.0 kg., were treated with 0.2 mg.kg intravenous Indomethacintrihydrate (IND) given 3 times at 12 hourly intervals. Renal function studies were performed before (T-0), 24 hours (T-24) and 48 hours (T-48) after the first dose of IND. As shown below, the urine volumes (Uv) and glomerular filtration rates (GFR) decreased at T-24 but returned to normal at T-48. The fractional excretion of sodium (FENa%), urine sodium (U_{Na}), and urine osmolarity (Uosm) were also reduced at T-24 but decreased even further at T-48, despite the normal GFR. There was no appreciable change in the values of serum sodium, serum osmolarity and free-water clearance (CH_2O).

	Uv	GFR	U_{Na}		Uosm	CH_2O	CH_2O+C_{Na}	CH_2O
	ml/min	ml/min/1.73m	mEq/L	FENa%	mosm/L	ml/min	100mlGFR	CH_2O+C_{Na}
T-0	.52	9.2	47	1.94	221	.12	29.0	45
T-24	.36*	6.6	29*	1.54	198	.12	14.0	65
T-48	.55	9.5	19*	.76	175*	.15	12.0	80

The calculated $CH_2O + C_{Na}$ decreased and the $CH_2O/CH_2O + C_{Na}$ increased appreciably; they measure proximal and distal tubular resorption of sodium, respectively. Intravenous IND appears to produce increased resorption of sodium, decreased GFR and Uv. Increased Na absorption would explain the low urine sodium, and urine osmolarity. * = $p < .05$.

53 Low GFR in the early postnatal period is to a large extent due to low renal plasma flow (RPF). Since the low RPF appears to be due mainly to active vaso-constriction, the question is raised whether renal vaso-constriction is needed to minimize the energy demands of the kidney in early postnatal life. The work load to the kidney is determined by the GFR. The most energy demanding process of the kidney is reabsorption of Na (TNa). In the present study the relationship between TNa and O_2 consumption are compared in the immature kidney of 24 days old rats (R24) and the mature kidney of 45 days old rats (R 45). Determinations are made of filtered Na (F_{Na}), T_{Na} and O_2 consumption by extraction and clearance techniques during hydropenia (HP) and volume expansion (VE). The two conditions are chosen since VE will cause vaso-dilatation of the immature kidney. This finding was confirmed in the present study and resulted (see table) in a marked increase of TNa in volume expanded R24.

	TNa µEkv/min/ 100 g BW	O_2 cons µmol/ min/100 g BW	Na/O_2 ratio µekv/ mol
R45 HP	68.7	2.05	15.74
R45 VE	73.4	4.69	17.49
R24 HP	36.8	3.39	13.02
R24 VE	76.4	4.49	19.48

Yet the increase in O_2 consumption during the transition from HP to VE was less pronounced in R24 than in R45. In volume expanded R24 the amount of T_{Na}/µmol oxygen consumed ($Na-O_2$) increased significantly. This suggests that in the volume expanded R24 the increased tubular load of sodium is reabsorbed by non oxidative pathways, either passive or anaerobic.

54 COMPENSATORY ADAPTATION TO REDUCED RENAL MASS IN THE NEWBORN GUINEA PIG (GP). Chevalier, R.L. Dept. Pediatrics, Univ. of Virginia, Charlottesville, VA., USA

In order to identify the physiologic adaptation to compensatory renal hypertrophy in the newborn, right uninephrectomy (NX) or sham (S) operation was performed in GP within the first 36 hrs. of life. At 3 wks. of age, they were studied using clearance and micropuncture techniques. Body weight (BW), left kidney weight (LKW), and arterial blood pressure (ABP) were measured. Left kidney glomerular filtration rate (LKGFR) and single nephron GFR (SNGFR) were measured by clearance of ^{14}C inulin. Proximal tubular pressure (P_T) and stop flow pressure (SFP) were measured using a servonulling device. Glomerular capillary pressure (P_{GC}) was estimated from the sum of SFP and colloid oncotic pressure. Results (mean±SE, n = Number of animals, NS = not significant):

	BW (g)	LKW (g)	LKGFR (ul/min)	SNGFR (nl/min)	ABP (mmHg)	P_{GC} (mmHg)	P_T (mmHg)
NX	247±14	1.91±0.09	936±54	12.6±1.1	54±4	31.6±0.8	9.5±0.2
n	(11)	(11)	(6)	(6)	(5)	(5)	(5)
S	250±12	1.31±0.05	558±56	7.9±0.5	56±2	30.8±0.7	9.6±0.2
n	(12)	(12)	(6)	(6)	(6)	(6)	(6)
P	NS	<0.001	<0.001	<0.005	NS	NS	NS

Conclusions: 1) Growth in the neonatal GP is unaffected by NX, 2) 3wks. following NX, there is a 41% increase in mass and a 68% increase in GFR of the renoprival kidney, 3) There is a parallel increase in SNGFR, indicating a significant contribution of immature superficial cortical nephrons to the adaptive process. 4) The increased SNGFR is not due to changes in ABP, P_{GC}, or P_T.