RENAL BICARBONATE HANDLING IN NEONATAL PIGS DURING NORMOXIC RESPIRATORY ACIDOSIS AND ALKALOSIS, Kersti Thodenius, Gaston

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RESPIRATORY ACIOSIS AND ALKALOSIS, Kersti Thodenius, Gaston Zilieruelo, Cleida Suguihara, Eduardo Bancalari, and Josa Strauss Department of Pediatrics, University of Miami, Miami, Florida, Although the neonatal kidney seems to be able to reabsorb HCO2 during respiratory acidosis, it is not clear whether renal bicarbonate tubular reabsorption (HCO2TR) occurs in response to changes in hlood ph, PaCO2 or plasma HCO2, in an attempt to evaluate the relative importance of each of these factors, measurements of HCO2TR, fractional excretion of HCO2 (FEHCO2), glomerular filtration rate as endogenous creatinine clearance (Ccr) and urina flow (V) were done in 6 neonatal, enesthesized and mechanically ventilated plgs during four study periods: 1. Control, normal ventilation in room air (X pH 7.43, PaCO2 22, HCO2 22); il. Respiratory alkalosis induced by hyperventilation (X pH 7.62, PaCO2 22, HCO2 24); III. Respiratory acidosis induced by adding 10% CO2 to inspired gas (X pH 7.12, PaCO2 71, HCO222); and IV, Hypercapnea with normal pH, which was obtained by infusion of NaHCO3 (X pH 7.38, PaCO2 74, HCO3 48). Urine was collected anaerobically by catheter. Results (X+SEM) were:

PERIO	DD HCO;TR	FEHCO-	Ccr	٧
	(mEq/100mIGFR)	(%)	(mi/kg/min)	(ml/kg/mln)
1	2.29+0.13	0.08+0.03	1.84+0.46	0.036+0.016
1.1	2.28+0.14	0.38+0.12 *,**	1.75+0.38	0.027+0.007
111	2.51+0.14	0.12+0.07	2.90+0.54**	0.118+0.037*,**
IV	4 74+0 10* **	1 53+0 73* **	1 94+0 26	0 046+0 006**

*p < 0.05 compared to control. **p < 0.05 compared to previous period.

These data suggest: a) HCO $_{2}$ TR is clearly influenced by HCO $_{2}$ filtered load more than by PaCO, or blood pH changes; b) significant increase in FEHCO $_{2}$ in period IV, when pH normalized and PaCO $_{2}$ remained unchanged, indicates that pH exerts an important effect independent of PaCO $_{2}$; and c) acute respiratory acidosis significantly affects urine flow and Cor $_{2}$.

ULTRASONOGRAPHIC MEASUREMENT OF RENAL SIZE IN NEW-1646 BORN INFANTS. H.C. Tien, V. Kamtorn, S. Sun (Spon. by Richard Rapkin) UMD-NJ Med. Sch., Children's Hosp. of N.J., Dept of Neonatology, Newark, N.J.
Prior to introduction of ultrasound, radiography was the

only way of measuring renal size during life. This study reports the results of kidney length measured by realtime ultrasound system with 5 mHz transducer (Hewlett Packard 77020A) in 50 unselected neonates admitted to NICU. Gestational ages ranged from 24 to 43 wks, birth weight from 570 to 4540 gms. The infants were examined in supine position. The transducer was placed parallel to the spine and moved laterally. The longitudinal renal length was determined and laterally. The longitudinal renal length was determined and plotted against 3 independent parameters, gestational age, birth weight and crown-heel length. The results revealed; (1) kidney length (mm)= -1.35 + 0.82 x crown heel length (cm) r=0.887 p<0.001, (2) kidney length(mm)= -2.0+1.06xGA(wks) r=0.873, p<0.001, (3) kidney length(mm)=24.72+0.005xBW(gm) r=0.828, p<0.001. It appears that kidney length has the best correlation with crown-heel length among the 3 parameters studied. The left kidney is longer than the right in 58% of infants, and vice versa in 42%. SGA infants generally show prominence of calyces (echolucent areas), not to be confused with hydronephrosis or cystic malformation. Because of increasing application of realtime ultrasound in NICU, this information is important for the early diagnosis of deviant kidney size. Further study is underway to determine both the surface area and 3 dimensional volume of the kidney.

THE UTILITY OF TRANSPLANT RENAL BIOPSY (TRB), Tc-99th SULFUR COLLOID SCAN (SCS), AND CYCLOSPORINE (CS) DRUG LEVELS IN DISTINGUISHING ACUTE REJECTION (AR) VERSUS CYCLOSPORINE NEPHROTOXICITY (NT). H. Trachtman, M. Khawar, K. Phadke, A. Tejani, (Intr: L. Finberg). DMC-SUNY, Brooklyn, N.Y. The use of CS immunosuppression in renal transplantation has made it increasingly difficult to ascertain the etiology of acute

deteriorations in allograft function because of direct drug NT. We compared the utility of three diagnostic modalities-TRB, SCS and serum CS levels - to distinguish AR from CS NT in 19 children who received a primary transplant with CS therapy and 13 who were switched to CS from conventional treatment during the period 9/83-10/84

The children (18M:14F) received 15 living-related and 17 cadaveric allografts. The three tests were performed whenever possible in 22/32 patients with suspected AR (mean interval of 3.2 months post-initiation of CS). Clinical response to antirejection therapy was the operational criterion for AR. A TRB was considered positive for AR if lymphocytic infiltration was noted, SCS was deemed positive if there was colloid uptake, and CS serum levels were considered positive if subtherapeutic (<100ng/ml) or in the toxic range (>500ng/ml).

Test	N	Sensitivity(%)	Specificity(%)
TRB	36	27/29 (93)	5/7 (71)
SCS	33	19/27 (70)	3/6 (50)
CS Level	23	8/21 (38)	1/2 (50)

We conclude that: 1)TRB is the most useful test for the diagnosis of AR episodes defined by response to anti-rejection therapy; and 2)SCS and CS drug levels correlate poorly with the TRB histological findings and the patient's clinical course.

LEUKOCYTES IN CLINICAL AND EXPERIMENTAL 1648 HEMOLYTIC UREMIC SYNDROME (HUS). V.V

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Although leukocytosis is a prominent feature of HUS, an elevated
leukocyte count has not been recognized as an important feature of this
syndrome. Ten consecutive patients with HUS admitted to our Hospital syndrome. Ten consecutive patients with HOS admitted to our mospital were studied. During the acute phase, the highest serum BUN was 101 ± 42 mg/dl and creatinine 4.9 ± 2.6 mg/dl. Total leukocyte count was at 16600 ± 5300/ul with differential counts of polymorphonuclear leukocytes at 54 ± 16%, band neutrophils 8 ± 5%, lymphocytes 31 ± 16% and monocytes 6 ± 5%. The role of leukocytes in this syndrome was studied in our model of HUS (J Lab Clin Med 102: 847, 1983) in rabbits given a single intravenous dose of endotoxin (LPS) over 5 hours. Leukocyte functions were studied with assessments of plasma and leukocyte B. functions were studied with assessments of plasma and leukocyte B-glucuronidase (primary granules), plasma and leukocyte lysozyme (secondary granules), chemotaxis, C5a mediated leukocyte aggregation and leukocyte procoagulant contents. Our results showed elevated plasma levels and reduced leukocyte contents of enzymes immediately following LPS infusion. Similarly, leukocyte reactivities plummeted at the same time period. At the time of the striking leukocytosis noted at 24 hours, plasma enzymes, leukocyte enzymes and leukocyte reactivities returned to normal values. At this time, leukocytes were found to have exaggerated amounts of cytoplasmic procoagulants. By

day 2, both leukocytosis and procoagulant contents returned to normal.

We conclude that leukocytosis is a common feature of various forms of HUS. Perturbation of leukocytes may play a role in and also reflect the process of inflammatory injury in this syndrome.

PERMEABILITY OF SYSTEMIC CAPILLARIES TO ALBUMIN IN

PERMEABILITY OF SYSTEMIC CAPILLARIES TO ALBUMIN IN
PUROMYCIN AMINONUCLEOSIDE (PA) NEPHROTIC SYNDROME.

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We have shown that polycation injection induces albumin leakage out of systemic microvasculature, probably by neutralizing an
electric charge barrier (JCI 73:1053,1984). PA causes proteinuria
by reducing the glomerular charge barrier in rats. To investigate whether it has similar effects on systemic capillaries, rats were studied 2 weeks after a single IV injection of 15 mg/kg PA when they were hypoalbuminemic and mildly edematous. The animals were nephrectomized, allowed to recover from anesthesia and then injected with the polycation protamine sulfate (PS), 40 mg/kg. Blood samples were drawn prior to and 2,5,8 and 11 min after the infusion. Control rats not given PA were studied similarly. Plasma albumin concentration remained unchanged in PA rats but decreased in controls. Total intravascular albumin was calculated from albumin concentration and hct, and expressed as % of base-2 min 85.6±14.7* 8 min 45.0±11.3** 11 min

5 min 53.2±11.4** 116.0±18.8 Control 38.0± 9.1 108.4±18.1* ** P<.001 PA * P< .05 110.1±25.7

In control, but not in PA rats, hot increased after PS indicating concomitant fluid loss from vascular space. The data are consistent with the thesis that both PA and PS cause loss of charge barrier in capillary walls. Thus, pretreatment with PA eliminated the effects of PS seen in the control animals. The data provide further evidence that loss of peripheral charge sites may be important in the pathophysiology of edema formation.

THE EFFECT OF CHRONIC CAPD/CCPD TREATMENT AND PERITORIA TONITIS ON PERITONEAL CLEARANCES IN PEDIATRIC PATTENTS. Tassilo von Lilien, Isidro B. Salusky, John C. Alliapoulos, Heinz E. Leichter, Marcy Wilson, Teresa L. Hall, Richard N. Fine. UCLA Sch. Med., Div. Pediatric Nephrology, Los Angeles, California.

Sixteen CAPD/CCPD pts. aged 1 4/12 to 19 2/12 underwent serial peritoneal clearance studies. After drainage, a two hour dwell (1.5% Dianeal PD solution) was performed and a peritoneal fluid sample was obtained at the end of this period. A blood sample was taken at 60 minutes. The initial study was performed at 11.1 + 10.5 mos., the second study at 19.6 + 14.8 mos. and the third study at 31.9 + 13.3 mos. The creatinine and BUN clearances per 70Kg BW at the initial, second, third study were 11 + 3.6 and 14.2 + 4.0, 10.5 + 3.3 and 13.8 + 3.5, 10.8 + 4.1 and 13.4 + 2.6 ml/min. Of the 16 pts., only 3 (18%) had more than 20% decline in the creatinine and BUN clearances over time. Two pts. (12.5%) had more than a 20% increase in the creatinine clearance and one pt. in the BUN clearance over time. The effect of peritonitis on peritoneal clearance was studied by comparing the results of 14 peritonitis free intervals in 10 pts. The range in the change of the creatinine clearance in the group of pts. with peritonitis was -3.6 to +2.8 (mean -0.582) and without peritonitis -3.6 to +6.4 (mean -0.286) ml/mn/70kg (P=NS).

These data indicate that there is no loss of peritoneal

peritonitis -3.6 to +6.4 (mean -0.286) ml/mn/70kg (P=NS).

These data indicate that there is no loss of peritoneal clearance in pediatric pts. undergoing CAPD/CCPD of periods up to 54 mos. and that episodes of peritonitis did not adversely affect peritoneal clearance in these pts.