●1591 SODIUM (Na) BALANCE (B) IN THE GROWING PRETERM INFANT. William D. Engle, Ronald R. Magness, Daniel J. Faucher, Billy S. Arant, Jr. and Charles R. Rosenfeld, Southwestern Med. Sch., Dept. Peds., Dallas, TX.

Negative NaB and hyponatremia have been reported for growing preterm infants. It has been suggested that less Na is required when fluid therapy is restricted. To examine this, we have measured NaB(total intake-urinary excretion/24hrs), serum Na and potassium(K) concentrations, and urinary aldosterone (U-A) in 11 neonates(BW=1378±43g; GA=30.6±0.5wks) during the 1st and 3rd wks of life. (Mean ± SEM; *p<0.05).

Age(days;d)	0.8±0.3	2.5±0.5	19.6±0.6
Wt (%Δ from birth)	-2.5±1.1	-6.2±1.5	3.7±3.2*
Fluid intake (ml/kg·d)	75.5±4.7	92.6±6.0	148.6±6.7*
U vol. (ml/kg·d)	51.7±7.3	54.9±7.5	58.1±7.9
Serum Na (mEq/L)	138±0.9	143±1.0	138±1.8
Na intake (mEq/kg·d)	1.44±0.67	2.73±0.53	2.55±0.28
NaB (mEq/kg·d)	-0.21±0.62	0.25±0.58	1.77±0.26*
Serum K (mEq/L)	5.8±0.3	4.8±0.2	5.2±0.2
U-A (ug/kg·d)	0.50±0.11	0.59±0.16	0.86±0.18*

Maximum wt. change(Δ) from birth was -13.5±1.4% at 8.4±0.8d. Adequate growth, positive NaB and normal serum Na can be achieved in rapidly growing preterm infants receiving moderate Na intake when fluid therapy is not excessive. The increase in U-A from 1 to 3wks was associated with a decrease in the U Na/K ratio. U Na/K did not correlate with U-A during the 1st wk(r=-0.10) but did during the 3rd wk(r=-0.43). We conclude that an appropriate renal tubular response to A was evident by the 3rd wk of life in these infants.

●1592 FUNCTION OF RENAL DOPAMINE-2 RECEPTORS. Christian C Felder, Robin A Felder, Robert R Holloway, Jean E Robillard, and Pedro A Jose. Univ of Iowa & Georgetown Univ Med Ctr, Dept of Peds, Iowa City & Washington, D.C.

We have previously reported presence of dopamine-2 receptors (D_2R) in canine intrarenal arteries (IRA) using radioligand binding. To determine the consequences of D_2R occupancy we measured the effect of intrarenal infusion of the D_2 agonist LY171555 (LY) (10^{-9} to 10^{-6}M) on renal blood flow (RBF, ml/gm/min), renal vascular resistance (RVR, units) and mean arterial pressure (MAP, mm Hg) during α and β adrenergic blockade. Group I(n=8) =dogs whose MAP was decreased by LY (10^{-7}M) and Group II(n=6)had Δ MAP<10%).

	BASAL			LY		
Group	RBF	MAP	RVR	RBF	MAP	RVR
I	4.0±.5	130.1±2.9	36.3±4.6	4.6±.4*	110.0±4.9*	24.7±2.3*
II	4.8±.6	120.8±6.2	27.4±3.8	5.8±.7*	114.6±4.0	21.3±2.6*

Adenylate cyclase (AC) linkage of D_2R occupancy was determined by examining the effects of 10^{-4}M forskolin(F) and the D agonists bromocryptine, 10^{-5}M(B), LY 10^{-4}M and (-)-apomorphine, 10^{-4}M(A)on basal or F stimulated AC activity (pmol/mg protein/min) in main renal artery (MRA) and IRA homogenates.

	MRA(n=7)	IRA(n=6)		MRA(n=7)	IRA(n=6)
Basal	10.4±2.6	8.7±1.3	F	41.1±9.5*	26.2±4.6
Basal + B	8.9±2.2	8.5±2.0	F+B	35.9±8.9	26.5±6.5
Basal + LY	9.2±2.2	8.2±1.9	F+LY	40.6±7.7	28.6±7.4
Basal + A	10.4±3.2	8.4±1.9	F+A	31.5±13.3	31.3±7.9

Conclusion: Occupation of renal vascular D_2R is associated with increase in RBF without any effect on basal or F stimulated AC. (*p<.05 drug vs. basal, paired t test).

1593 THE USE OF THE RENAL BIOPSY TO PREDICT ALLOGRAPH REJECTION IN A PEDIATRIC KIDNEY TRANSPLANT RECIPIENT. Robert S. Fennell, William H. Donnelly, Clifford A. Purcell. Dept. of Ped., Univ. of Florida, Gainesville, Florida.

During an eight year period, 127 kidney transplants were performed on children and adolescents at the University of Florida. Seventy-three kidney biopsies were performed on 52 of these recipients (mean age 13 years) to elucidate deterioration of allograft function. Specimens were stained for routine light microscopy and immunofluorescence. Glomerular, tubular, interstitial, arteriole and arterial abnormalities were assessed by a 117 item survey of renal pathology. Fifty-six items described the distribution of pathologic changes and the remainder were graded observations of histologic abnormalities. The results were statistically evaluated in search of features that predicted the loss of the allograft within one year of the biopsy. Glomerular hypercellularity, swelling of the mesangial, endothelial and epithelial cells, encroachment on glomerular vascular space, basement membrane splitting, arteriolar wall hyperplasia, and arterial luminal compromise were associated with allograft rejection. Related items such as luminal compromise and cell swelling were strongly correlated in individual biopsy specimens. When two or three of these features were present in a specimen, the allograft was almost certain to be rejected within a year. Tubular changes, interstitial fibrosis or mononuclear cell infiltrates did not predict allograft rejection. Only half of the specimens had positive immunofluorescence reactions and none predicted allograft loss.

1594 INAPPROPRIATELY LOW PLASMA CALCITRIOL (CL) IN CHILDREN WITH NEPHROTIC SYNDROME (NS) AND NORMAL GLOMERULAR FILTRATION RATE(GFR). Michael Freundlich, Jacques J. Bourgoignie, Gaston Zilleruelo, Carolyn Abitbol, Jose Strauss. Depts. of Pediatrics and Medicine, Univ. of Miami School of Medicine, Miami, Florida.

Although plasma CL levels in NS have been found to be low or normal, their interpretation remains ambiguous. To evaluate plasma CL in relation to its regulatory factors during both relapse (RL) and remission (RM), children with NS and normal GFR were studied. Data were compiled in 58 patients (39 males, 19 females) ages 2 to 20 yr (x̄ 10.1) during 79 RL and 82 RM. Results were:

	Relapse	Remission	p
Calcidiol (ng/ml)	9.0±7.0	30.4±14.6	< 0.001
Calcitriol (pg/ml)	45.8±32	53.4±28	NS
Ca (mg/dl)	8.3±0.9	9.7±0.5	< 0.001
P (mg/dl)	5.2±0.8	4.9±0.7	< 0.05
PTH (uLEq/ml)	105±57	92±51	NS
Albumin (g/dl)	3.3±1.5	4.2±0.6	< 0.001
Proteinuria (g/day)	2.4±2.2	0.04±0.12	< 0.001
GFR (ml/min/1.73m²)	109±34	100±26	NS

While, as expected, in RM Ca correlated inversely (r = -0.04) and PTH directly (r = 0.12) with CL, in RL these correlations were direct (r = 0.48, p < 0.001) and inverse (r = -0.05), respectively. Serum albumin and CD correlated both directly with plasma CL; proteinuria correlated inversely better with CD (r = -0.26, p < 0.01) than with CL (r = -0.15, p <0.2). We conclude that in nephrotic children, important abnormalities of mineral metabolism present in RL normalize during RM, daily proteinuria has a greater effect on plasma CD than on CL, and that circulating CL is normal in RL. The latter, however, is inappropriately low for the prevailing hypocalcemia and suggests inadequate synthesis of CL by the nephrotic kidney even with normal GFR.

●1595 MILK FORMULA CAUSES ALUMINUM (AL) TOXICITY IN UREMIC INFANTS. Michael Freundlich, Marie C. Faugere, Gaston Zilleruelo, Carolyn Abitbol, Charles J. Bradac, Jose Strauss, Hartmut H. Malluche Dept. of Peds. Univ. Miami, Miami, Fl., and Div. of Nephrol., Bone & Min. Metab.; Univ. of Ky. Med. Ctr., Lexington, Ky.

Young children, particularly infants with chronic renal failure, are prone to develop Al accumulation in brain and bone. Ingestion of Al containing phosphate binders ($Al(OH)_3$) and Al in dialysis water were identified as sources for Al. Daily intake of elemental Al > 100 mg/kg b.w. was shown to be toxic. We report Al toxicity in 2 infants with congenital uremia and no intake of $Al(OH)_3$. The 1 and 2-1/2 month old infants were fed Similac PM 60/40. The first patient expired after one month of conservative management without dialysis or therapy with $Al(OH)_3$. Brain Al was high (47.4 ng/mg) and bone Al within normal limits. Bone histology did not reveal pathologic changes of bone structure, bone formation or resorption. The other patient began continuous peritoneal dialysis at two weeks of age, never received $Al(OH)_3$ and expired during the third month of life, following sudden onset of CNS-related symptoms. Al was high in brain (6.4 ng/mg) yet normal in bone. To explore possible sources of Al intake, powder of milk and ready-to-use formulae administered to the patients were assayed. Concentrations of Al in milk were consistently elevated (232±60, range 126-316 ng/ml). In addition, water used for preparation of milk formulae had Al concentrations of 130-280 ng/ml. Al concentrations in dialysate and peritoneal removal of Al were insignificant.

Our findings of Al accumulation in brain with normal Al content in bone and absence of stainable bone Al indicate a high susceptibility of the developing infant's CNS to Al toxicity. Since the infants reported here did not ingest $Al(OH)_3$, proprietary milk formulae and drinking water, identified as sources, must have played an important role. Availability of Al-free milk and of Al-free drinking water should help to prevent Al related toxicity in infants with renal failure.

† 1596 HYPERTENSION IN BABIES FOLLOWING DISCHARGE FROM NEONATAL INTENSIVE CARE UNIT (ICU). Aaron L. Friedman and Virginia Hustead. Univ. of Wisconsin Hospital, Department of Pediatrics, Madison, WI.

We followed 17 babies who were found to have elevated blood pressure (BP) at or after discharge from neonatal ICU (systolic BP > 115 mm Hg, repeated 3 times under quiet conditions); babies with hypertension during their stay were excluded. All 17 babies had nursery stays longer than 48 hr. Follow-up was 6 months to 3½ years. Five babies had the following secondary diagnoses: 2 UPJ obstruction, 1 coarctation of the aorta, 1 neuroblastoma and 1 renal artery thrombosis. Five babies had umbilical artery catheters (UAC) for more than 24 hours; three of these babies are included in the group with secondary diagnoses. Thus, 10 babies developed hypertension without obvious cause. Sex, age of gestation, type of feeding, ventilator use or antibiotic use, proved insignificant. Sixteen of 17 babies were treated with 1-2 mg/kg/day of propranolol and/or 10-20 mg/kg/day of chlorthiazide. All children older than 2 years were off medication and were normotensive.

We conclude: 1) follow-up of babies discharged from neonatal ICU should include careful BP measurements; 2) high BP may develop in babies who have not had prolonged use of UAC; 3) even when no secondary cause can be elucidated, hypertension responds to medication; 4) further study is needed to determine if babies discharged from neonatal ICU are at high risk for elevated BP and to determine the natural history, prognosis and best form of treatment for these babies.