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ASSESSING RENAL FUNCTION IN CHILDREN WITH MYELOMENINGOCELE. E.B. Charney, J.W. Foreman, A.T. Mazur, J.M. Egler (Spon. by J.R. Hoyer). Dept. Pediatrics, Children's Hospital of Philadelphia, PA.

Because children with myelomeningocele (MM) are at increased risk of renal insufficiency, a simple method of estimating GFR from the plasma creatinine (P_{Cr}) and height would be useful. However, this estimate is predicated on a normal muscle mass and creatinine production for weight. Muscle mass may be diminished in children with paralysis. We therefore compared the estimate of GFR/1.73M² from the formula 0.55 height (cm) ÷ P_{Cr} with that determined with inulin in 14 children with MM ranging in age from 4-17 years. Seven had severe paralysis requiring wheelchair (WC) mobility. IVPs were abnormal in 9/14; 7/14 had inulin GFR < 80 ml/min/1.73M². In 8 patients (57%) the estimated GFR averaged 85% higher than the inulin GFR. Of these 8 patients, 6 were WC-bound. In 5 patients (36%), there was close correlation between the two GFR methods, with an average difference of 11%. Only 1 of these was WC-bound. In 1 child, the estimated GFR was 64% lower than inulin GFR. All but 1 of the patients had P_{Cr} < 1 mg/dl. Our data suggest that an estimate of GFR determined from the serum creatinine and height in children with MM and significant paralysis is not reliable. This probably results from a diminished muscle mass and creatinine production per unit of body weight. Further studies are necessary to establish whether or not there is a reliable method for estimating GFR in this population.

Supported in part by NIH Grant RR 00240.

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RENAL CORTEX TAURINE (T) POOL SIZE REGULATES RENAL ADAPTIVE RESPONSE TO ALTERED DIETARY INTAKE OF SULFUR AMINO ACIDS (SAA). Russell W. Chesney, Sherman Dabbagh, Naomi Gusowski. University of Wisconsin Hospitals, Department of Pediatrics, Madison, Wisconsin.

Rats ingesting a low or high SAA diet show renal adaptation with low or high urinary T excretion. Brush border membrane vesicle (BBMV) studies indicate expression of this adaptive response at the brush border surface (Chesney et al, Kidney Int 24:588, 1983). The signal for this adaptive response, which increases the V_{max} of T transport at the BBMV, is unknown but may involve a change in plasma or renal cortex T. We used two maneuvers to deplete renal cortex T: 1) β-alanine feeding in drinking H₂O; 2) β-alanine feeding plus 72-hr fast, which reduced cortex T without greatly changing plasma T. As compared to rats fed a low, normal or high SAA diet, an accentuation of BBMV uptake was evident. Renal cortex T fell by 45-75% after β-alanine feeding (p < .001). Uptake of T by BBMV was increased significantly (p < .05 to .005) and was related to an increase in V_{max} of the initial rate of uptake by more than 100 pmoles/mg protein/60 sec (p < .01 to .001). The combination of fasting and β-alanine feeding reduces cortex T content even further and is again associated with higher BBMV uptake values. Plasma levels of T are not significantly altered as compared to each dietary group's control level. Change in renal cortex T is inversely correlated with change in BBMV initial uptake (r = -0.547, p < .05). Feeding 3% glycine or methionine does not alter T uptake. These data suggest that renal cortex pool size regulates the renal adaptive response to altered dietary intake of SAA.

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EFFECTS OF CHRONIC PARTIAL URETERAL OBSTRUCTION (CPUO) AND UNINEPHRECTOMY (NX) ON RENAL AND SOMATIC GROWTH IN THE NEWBORN GUINEA PIG. Robert L. Chevalier and Anthony V. Broccoli (Spon. by Alan D. Rogol), University of Virginia Medical Center, Dept. of Pediatrics, Charlottesville.

Although CPUO is one of the major causes of renal insufficiency in infancy, the relationship between renal impairment and growth remains poorly understood. Within the first 48 h of life, 63 guinea pigs were subjected to left (L) CPUO, 31 of which also underwent right (R) NX. 16 sham-operated and 15 NX animals were controls. At 23±2 days of age, body weight (BW), dry kidney weight (DKW), ureteral diameter (UD) and BUN were measured. Resulting UD was variable, and each group was divided into Mild (UD < 3 mm) and Severe obstruction (UD ≥ 3 mm). Control UD = 1 mm. Results were as follows (mean ± SE, *p < 0.05 vs Control):

	R Kidney Intact			R Kidney NX		
	Control	Mild	Severe	Control	Mild	Severe
BW g	245±8	217±11*	219±6*	235±9	209±8*	175±5*
LDKW mg	256±9	249±14	224±10*	453±22	318±16*	404±18
RDKW mg	259±9	254±15	359±16*	-----	-----	-----
BUN mg/dl	22±1	24±4	26±2	28±2	32±5	67±11*

We conclude that during Mild CPUO, compensatory renal growth due to NX is impaired, but normal renal growth is not. Severe CPUO with intact R kidney results in decreased growth of the affected kidney and compensatory hypertrophy of the other, while Severe CPUO and NX results in azotemia despite compensatory hypertrophy of the obstructed kidney. Somatic growth during CPUO is limited by unidentified factors in addition to functional renal mass.

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HEMODYNAMIC EFFECTS OF CHRONIC PARTIAL URETERAL OBSTRUCTION (CPUO) IN THE NEWBORN GUINEA PIG. Robert L. Chevalier and Anthony V. Broccoli (Spon. by Alan D. Rogol), University of Virginia Medical Center, Department of Pediatrics, Charlottesville.

To evaluate the hemodynamic effects of CPUO and reduced renal mass in the neonate, 38 guinea pigs were subjected to left (L) CPUO within the first 48 h of life, 17 of which underwent concurrent right (R) uninephrectomy (NX). Eight sham-operated and 8 NX animals were controls. At 23±2 days of age, L renal blood flow (RBF, ml/min), filtration fraction (FF, %), glomerular filtration rate (GFR, ml/min) and percent of RBF to the outer third of renal cortex (OCRBF) were measured using microsphere and clearance techniques. Resulting hydronephrosis was variable in animals with CPUO, and each group was divided into Mild and Severe obstruction for ureteral diameter < 3 mm and ≥ 3 mm respectively. Results were as follows (mean ± SE, *p < 0.05 vs Control):

	R Kidney Intact			R Kidney NX		
	Control	Mild	Severe	Control	Mild	Severe
RBF	5.0±0.7	6.2±1.0	2.2±0.4*	6.5±0.7	5.6±1.1	5.3±0.9
FF	22.2±0.9	20.5±2.0	11.5±1.5*	24.6±1.5	24.8±1.8	15.9±2.6*
GFR	0.65±0.10	0.71±0.12	0.13±0.02*	0.91±0.11	0.88±0.14	0.48±0.10*
OCRBF	47.7±1.9	52.0±2.2	52.2±2.8	52.3±1.9	49.3±1.2	45.7±1.4*

We conclude that reduced GFR during Severe CPUO with intact R kidney is due primarily to decreased RBF. R NX prevents this vasoconstriction and causes redistribution of RBF from outer to inner cortex. As Severe CPUO results in lower FF, factors other than RBF also contribute to reduced GFR, particularly following removal of the contralateral kidney.

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SEVERE HYPONATREMIA IN INFANTS TREATED WITH CONTINUOUS AMBULATORY PERITONEAL DIALYSIS (CAPD). Richard A. Cohn, Larry Misiewicz, and Donald I. Moel, (Spon. by Carl Hunt). Northwestern University, Children's Memorial Hospital, Department of Pediatrics, Chicago.

With improvements in technology, successful treatment of end-stage renal disease in infants by CAPD is possible. After achieving optimal dialysis and reversal of uremia, four consecutive infants developed severe hyponatremia (Na < 120 mEq/L) during their first month on CAPD. All 4 infants were males, 7-14 months old, weighing 4.5 - 7.8 kg. Two had obstructive uropathy as the underlying renal disease and one each had polycystic kidney disease and hemolytic uremic syndrome. A typical daily balance study follows:

	volume (ml)	Na (mEq/L)	Na (mEq/24 hr)	
Intake: Diet (PM 60/40 ^R)	1170	7	8	TOTAL 59
NaCl tabs (17 mEq x 3)	-	-	51	
Output: Urine	402	67	27	TOTAL 58
Dialysate (net neg. balance)	235	131	31	

Salt balance was easily maintained with oral salt tablets; blood pressure values did not change significantly with salt supplementation. We conclude that due to dialysate Na losses and infant formulas that may be low in sodium concentration, infants on CAPD may require salt supplements, especially during intercurrent illnesses when oral intake may be limited; these infants are at great risk of developing severe life-threatening hyponatremia. Older children and adults who have free access to dietary salt maintain salt balance with greater ease.

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ATRIOPEPTINS: NATRIURESIS OCCURS INDEPENDENT OF VASCULAR EFFECTS. Barbara R. Cole, John G. Boylan, Mary A. Kuhnline, Mark G. Currie, David M. Geller, and Philip Needleman. Washington U. Medical School, Depts. of Pediatrics and Pharmacology, St. Louis, MO

Atriopeptins I and II (AP-I, AP-II), 2 low mol. wt. peptides isolated from rat atria, cause specific relaxation of only intestinal smooth muscle (the 21-amino acid peptide AP-I) or both intestinal and vascular smooth muscle (the 23-AA peptide AP-II). Renal arterial injection of AP-II, but not AP-I, produces renal vasodilation. Both substances, as well as a high mol. wt. precursor, cause dose-related natriuresis and general increases in GFR when injected IV into anesthetized rats. The data below compare control (C) and experimental (E) periods of 10 min.

	U _{Na} V (μEq/min)		GFR (ml/min)	
	C	E	C	E
AP-I				
2 μg(N=4)	.23±0.06	1.34±.39	2.56±1.1	4.41±1.2
3 μg(N=2)	.24	5.19	3.46	5.18
AP-II				
1 μg(N=3)	.24±0.05	5.15±2.8	2.48±.72	5.83±1.3
1.5 μg(N=3)	.62±0.03	11.64±.83	3.02±.58	5.60±1.8

Partial purification of rabbit atrial homogenate yields high and low mol. wt. peptides which cause natriuresis. Low dose trypsin (1U/ml) activation of HMW produces a substance which is spasmolytic and natriuretic but does not increase GFR. HMW (N=8) 1.15±.27 4.61±1.13 1.62±.11 3.45±.59 HMW-tryp(N=5) 1.30±.58 5.61±2.13 1.41±.42 1.75±.40 The data suggest that the atriopeptins exert a direct effect on the kidney tubule in addition to the vasodilatory effects.