1585 HEMODYNAMIC EFFECTS OF ANGIOTENSIN CONVERTING ENZYME INHIBITOR (CEI) IN CHRONIC PARTIAL URETERAL OBSTRUCTION (CPUO) OF THE NEWBORN GUINEA PIG. Robert L. Chevalier and Anthony V. Broccoli, Univ. of Virginia, Department of Pediatrics, Charlottesville, Virginia.

Unilateral CPUO results in significant vasoconstriction of the ipsilateral kidney (Pediatr Res 18:359A, 1984). To evaluate the role of endogenous angiotensin II (A II) in this vasoconstriction, guinea pigs subjected to constriction of the left ureter during the first 2 days of life were given enalapril, a CEI, 6 mg/day for 7-12 days prior to study. These were compared to a control group of littermates receiving no drug. Renal blood flow (RBF, ml/min), number of perfused glomeruli (NPG, thousands), glomerular perfusion rate (GPR, nl/min) and glomerular filtration rate (GFR, ml/min) were measured at 21-26 days of age (mean $\pm$ SE, ns = $P > 0.05$):

	Left (obstructed) Kidney				Right (intact) Kidney			
	RBF	NPG	GPR	GFR	RBF	NPG	GPR	GFR
Enalapril	4.7	79.6	61.0	0.37	9.9	83.4	119.8	
$\pm$ SE	0.5	2.7	9.0	0.10	0.9	2.4	14.3	
Control	2.6	63.1	41.7	0.13	8.0	79.3	107.2	
$\pm$ SE	0.3	3.7	3.7	0.02	0.7	2.2	8.8	
P<	0.001	0.01	0.05	0.05	ns	ns	ns	

While the intact kidney was unaffected by enalapril administration, a 26% increase in NPG and 46% increase in GPR were associated with marked increase in RBF and GFR of the left kidney. We conclude that A II plays a significant role in reduction of renal function during CPUO, and acts in part by diminishing the proportion of perfused glomeruli.

1586 PLASMA CREATININE (Pcr) IN CHILDREN: INTER-LABORATORY VARIATION. Marie J. Clermont, Luc Brion and George J. Schwartz, Albert Einstein College of Medicine, Dept. of Ped., Div. Nephrol. and Neonatol., Bronx, N.Y.

Pcr is used to estimate glomerular filtration rate (GFR) in children: $GFR = 0.55L/Pcr$, where L is body length in cm. The validity of this formula depends on accurately measuring Pcr, especially near the maximum sensitivity of the method. In our lab, we have used the Auto-Analyzer modified Jaffe end-point method, running an air blank before each sample to minimize the "carry-over" effect. Because this method is too cumbersome to be used routinely, we compared our values for a given sample to those obtained on the same day in two hospital labs: a pediatric lab using the kinetic Jaffe reaction with an automated system (Beckman Astra 8), and a busy general lab using the end-point technique on a Technicon SMAc.

For every range of Pcr (0.3-2 mg%) there was no significant difference between our measurements and those from the pediatric laboratory (mean diff. = 0.01 ± 0.02 (SE), $n=143$). Mean values from the general lab, however, were significantly higher ($*p < 0.05$) than ours (mg%):

Pcr Range mg%	n	Our Lab	General Lab	% Error
0.30-0.50	21	0.42 ± 0.01	$0.50 \pm 0.02^*$	22 ± 5
0.50-0.75	44	0.66 ± 0.01	$0.75 \pm 0.01^*$	18 ± 1
0.75-1.00	42	0.89 ± 0.01	$0.96 \pm 0.02^*$	12 ± 2
1.00-2.00	62	1.24 ± 0.03	$1.31 \pm 0.03^*$	11 ± 1

Both our end-point and the kinetic methods are equally accurate. In the general lab, the "carry-over" effect may account for higher Pcr levels (0.08 ± 0.01 mg%). The relative error is higher for low Pcr, decreasing its reliability, especially in small children. Minimization of "carry over" should improve the accuracy of this measurement.

1587 ATRIOPEPTIN: A POWERFUL NATRIURETIC IN RATS WITH RENAL FAILURE. Barbara R. Cole, Mary A. Kuhlman, Mark G. Currie, and Philip Needleman, Washington Univ. Sch. of Med., Depts of Peds. and Pharmacol., St. Louis.

Atriopeptins are recently described peptides in cardiac atria which are natriuretic and vasodilatory. We have found that a 30 min infusion of Atriopeptin III (AP III), a 24 amino acid peptide, produces a 9-fold increase in the fractional excretion of sodium (FE_{Na}), with initial increases in both glomerular filtration rate (GFR) and renal plasma flow.

To determine whether atriopeptins might be efficacious in the volume expansion of chronic renal failure, 250 g Sprague-Dawley rats underwent 5/6 nephrectomy. Clearance studies were performed after 4 days or 4 weeks of uremia. After a baseline period of 20 min, AP III was infused for 30 min at $0.5 \mu\text{g/kg/min}$. Responses in GFR and FE_{Na} are shown below. (mean $\pm$ SEM)

	(Baseline)	10'	20'	30'	Recovery
GFR(ml/min)					
4 d	$.45 \pm .05$	$1.72 \pm .15$	$.75 \pm .05$	$.75 \pm .05$	$.32 \pm .07$
4 wk	$.28 \pm .05$	$.60 \pm .06$	$.48 \pm .12$	$.83 \pm .08$	$.25 \pm .02$
$FE_{Na}(\%)$					
4 d	$.4 \pm .2$	$1.0 \pm .2$	13.8 ± 1	13.1 ± 1.6	$3.6 \pm .1$
4 wk	5.3 ± 2	12.4 ± 2.5	24.4 ± 4	$8.7 \pm .7$	7.1 ± 2.1

The responses to atriopeptins in rats with chronic renal failure far exceed those in the normal rat. Not only is there an increase in GFR that remains elevated throughout the infusion, but the magnitude of the natriuresis ($FE_{Na}=24\%$) is striking. This peptide promises to be helpful in managing the volume overload of patients with chronic renal failure.

1588 THE RENAL REABSORPTION OF INORGANIC SULFATE (SO_4) IS INCREASED IN PREGNANT WOMEN. David E.C. Cole, Lesley S. Baldwin and Linda J. Stirk. (Spon. by J. Crocker) Dalhousie Univ., I.W. Killam Hospital for Children, and Grace Maternity Hospital, Depts of Pediatrics, and Obstetrics and Gynecology, Halifax, NS.

Renal regulation of SO_4 reabsorption permits the excretion of the excess SO_4 generated by sulfur amino acid oxidation, while maintaining a supply of substrate for sulfation reactions that is sufficient to support growth. In the adult, increasing serum SO_4 concentrations usually signal a decline in renal filtration. In pregnancy, however, maternal glomerular filtration (GF) is enhanced and serum creatinine (Cr) falls, but serum SO_4 does not. To understand why, we measured serum SO_4 by controlled-flow ion chromatography in 144 pregnancies of 7 to 43 wks duration. SO_4 concentrations rose steadily throughout pregnancy and were significantly elevated by the second trimester; the correlation between serum SO_4 and length of gestation was highly significant ($r=.53$, $p<.001$). In 32 third-trimester pregnancies, we also collected 24hr urines, measured SO_4 and Cr clearances, and compared them with results from 22 controls. While serum SO_4 was higher in this group [434 ± 6 vs $328 \pm 10 \mu\text{mol/L}$ ($x \pm SE$, $p<.001$)], 24hr excretion of SO_4 was not (221 ± 13 vs $225 \pm 13 \mu\text{mol/kg body wt}$). Based on creatinine clearances, absolute SO_4 reabsorption was significantly increased in the pregnant women [28 ± 2 vs $21 \pm 2 \mu\text{mol/100ml GF}$, $p<.02$]. This increase may be the result of other changes in renal solute handling during pregnancy, but a specific adaptive response of the maternal kidney to increasing fetal-placental needs cannot be ruled out.

1589 VITAMIN D-DEFICIENT PROLINURIA IS EXPRESSED AT THE RENAL BRUSH BORDER MEMBRANE (BBM) INDEPENDENT OF PARATHYROID HORMONE (PTH). Shermine Dabbagh, Russell W. Chesney and Naomi Gusowski. University of Wisconsin, University Hospitals, Dept. of Pediatrics, Madison, WI.

Vitamin D deficiency is associated with a generalized aminoaciduria and phosphaturia. To study this tubulopathy weanling rats were fed several vitamin D deficient diets for 4-6 wks. 1) Very low Ca, 0.02% (VLC). 2) Low Ca, 0.47% (LC). 3) High Ca, 2.2%. and 4) Very Low P, 0.1% (VLP). Rats on VLC were given 500 pmoles $1,25(OH)_2D_3$ for 2 days.

Rats on VLC diet had 10 X elevated PTH and 2 X elevated urinary cAMP and 10 X prolinuria which did not change with $1,25(OH)_2D_3$. Rats on LC and HC diets had attenuated changes 8 and 5 X in PTH and 1.7 and 1.8 X cAMP and 5 X and 11 X elevated prolinuria. Rats on VLP had normocalcemia ($Ca=8.6-0.5$) hypophosphatemia ($PO_4=6.4-0.6$) normal PTH and urinary cAMP, but a 12 X elevated prolinuria, hypercalcuria and no PO_4 excretion.

The V_{max} & K_m of proline uptake by BBMVs on each diet was similar but a reduction in the peak of the overshoot at 30 sec. in all the vitamin D deficient diets was statistically lower than normal. $1,25(OH)_2D_3$ corrects this defect in the VLC diet along with phosphaturia, and Ca excretion without affecting PTH or urinary cAMP. The peak overshoot correlated with the plasma PO_4 ($r=0.536$, $P<0.02$) but not Ca, cAMP, PTH or urinary proline. Thus, prolinuria of D-deficiency is expressed at the brush border membrane surface. H $^+$ -proline uptake by BBMVs is independent of parathyroid status and appears to involve "leaky" membranes.

1590 ABSENT FACTOR VIII RESPONSE TO DDAVP IN NEPHROGENIC DIABETES INSIPIDUS (NDI). John J. Doyle, Nathan L. Kobrinsky, Esther D. Israels, Jeremy S.D. Winter. Department of Pediatrics, University of Manitoba, Winnipeg, Can.

Patients with NDI are resistant to the anti-diuretic effects of ADH. If this resistance is due to a generalized defect of the ADH receptor, then other end-organ responses should be absent. DDAVP, a synthetic analog of ADH, increases factor VIII coagulant activity (FVIIIc) and factor VIII related antigen (FVIII:Ag) in normal subjects. Therefore, if NDI is due to a generalized receptor defect, the factor VIII response to DDAVP should be absent in affected patients and decreased in obligate carriers. Two unrelated NDI patients, three obligate carriers and twenty controls were studied. Each received $0.30 \mu\text{g/kg}$ DDAVP. Pre and one-hour post infusion FVIIIc and FVIII:Ag levels were determined:

SUBJECTS	FVIIIc (%)		FVIII:Ag (%)	
	Pre	Post	Pre	Post
patient #1	117	103	145	135
patient #2	128	126	140	125
carrier #1	106	174	180	240
carrier #2	72	144	87	125
carrier #3	113	253	99	118
controls (20)	98 $\pm$ 21	260 $\pm$ 57	92 $\pm$ 33	165 $\pm$ 21

FVIIIc and FVIII:Ag responses were absent in both patients ($P<.01$), and were decreased in two of three obligate carriers. These data confirm that NDI is a generalized defect of the ADH receptor. Further, a decreased factor VIII response may aid in identifying carriers in kindreds at risk.