1504 ADRENAL RESPONSIVENESS TO ACTH IN PATIENTS(pts) WITH MINIMAL CHANGE NEPHROTIC SYNDROME(MCNS).

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An impaired response to a 2-hr ACTH test has been reported to predict pts with MCNS who will develop frequent relapses(FR). We have evaluated basal 8AM cortisols(B)(nl >8μg/dl) and 2 hr-post ACTH stimulation cortisols(S)(nl >30μg/dl, Ref., Leisti, 1978) in 28 pts with MCNS. Results of studies performed were: Prior to steroid therapy(Rx)(B:8±3.6*;S:18±2.9); Following alternate day Rx(QOD)(B:8±3.7;S:21.5±7.3); and 1-6 mo post Rx(B:11.8±4.5;S:21±7.5)(*mean±SD). Pts with nl responses:

	Normal	Nl Control	Pre-Pred	QOD Pred	Post-Pred(1-6mo)
B cortisol		16/17	2/5	4/15	6/8
S cortisol		2/2	0/5	2/15	0/8

There is no significant difference between Rx groups at P<.05. The control group is significantly different from all Rx groups except B-post-pred. Abnormal tests occurred in 13/14 pts with a history of FR and 5/6 infrequent R. No relationship was observed (P<.05) between length of remission (REM) and response to ACTH in 22 pts followed >1 yr.

	Length of Remission		
Normal Tests	<6mo	6-12mo	>12mo
B cortisol	7/14	1/3	2/5
S cortisol	1/14	1/3	0/5

Thus basal and stimulated cortisol levels were frequently abnormal in pts with MCNS and suggest that further studies should be done to define possible roles for adrenal cortical hyporesponsiveness in the pathogenesis of MCNS. Response to ACTH stimulation was not predictive of length of remission in our patients.

1505 LOCALIZATION OF THE SITE OF URINARY TRACT INFECTIONS USING SERUM C-REACTIVE PROTEIN (CRP).

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The CRP test has been used as an indication of the presence or absence of kidney inflammation during urinary tract infections. However, only Jodal's group has reported a study of this application. In our studies the concentration of serum CRP, measured by radial immunodiffusion, are compared with the site of infection determined by bladder washout (BWO) and by clinical criteria. The table shows that 9 of 16 children with upper tract infection by BWO had serum CRP levels >25 μg/ml while only 4 of 28 with lower tract infection had values in this range.

SITE OF INFECTION -BLADDER WASHOUT-	C-REACTIVE PROTEIN (μg/ml)			
	0	<10	10-25	>25
Upper	4	1	2	9
Undetermined	7	0	4	1
Lower	20	0	4	4

Four of 6 children with upper tract infection by BWO and clinical acute pyelonephritis had serum CRP levels >25 μg/ml. However, the CRP levels were <25 μg/ml in 5 of 7 children with upper tract infections by BWO and clinical diagnoses of cystitis or asymptomatic bacteriuria. This suggests that the CRP test may be unreliable for localization of the site of infection in the clinical situation in which this information is most needed.

●1506 SINGLE DOSE AMOXICILLIN (S) TREATMENT OF UNCOMPLICATED URINARY TRACT INFECTIONS (UTI) AS EFFECTIVE AS CONVENTIONAL THERAPY (C).

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Forty children, aged 2½ to 12 years, with acute, uncomplicated UTI's due to amoxicillin-sensitive organisms were randomized to receive either single oral dose (30 mg/kg) or 10 days of conventional dose amoxicillin. Urine cultures were performed X2 on entrance to the study, and 1 and 4 weeks after treatment. Sedimentation rates (SR), C-reactive protein (CRP) and antibody-coated bacteria testing (ACB) were performed prior to therapy; IVP's and VCUG's, 6 to 8 weeks after. Results were:

Treatment Regimen	Outcome	No. Patients	Abnormal Xrays
Single Dose (D)	Cure	17	0
Single D	Failure	5	80%
Conventional D	Cure	16	12.5%
Conventional D	Failure	2	0

SR, CRP, and ACB did not correlate with either radiology or treatment outcome. However, response to S did, with those failing S treatment having a high rate of xray abnormalities, while those responding having a low probability of abnormalities. The results suggest that S treatment is as effective as C. As well as being associated with better compliance and decreased cost, S may provide useful information for future patient management.

1507 PARADOXIC HYPERTENSION (PH) IN RATS EXPOSED TO BOTH CLONIDINE (CL) AND PROPRANOLOL (P).

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To assess PH seen clinically in our patients, either CL (.1mg/kg/24h) or P (200mg/kg/24h) was given in drinking water singly and then together to normal (N) or spontaneously hypertensive (SHR) rats and blood pressure followed by tail plethysmography. (Letter after Group denotes first drug given; values=Δ BP (mmHg); *p < 0.01 P or C vs.control; ** p < 0.01 P + C vs. control.)

Group	n	Control BP	CL or P 7 days (d)	CL and P 2d	CL and P 7d
N-P	9	120mmHg	-12*	+6**	-2
N-CL	9	120	-11*	+21**	0
SHR-P	8	190	-20*	+5	+15**
SHR-CL	9	170	-13*	+7	+25**

Subsequently SHR or N rats on oral CL or P were anesthetized with pentobarbital and given graded infusions of P or CL while BP responses to low-dose drug (CL 6 ug/kg or P .075mg/kg); for 5 minutes BP rose 2-10 mmHg (p < 0.02) prior to sustained fall of 5-15mmHg. Graded doses of Phentolamine did not influence response. We have shown that PH seen in patients can be produced in both N and SHR rats. We conclude that PH is probably not due to increased alpha stimulation (from CL) in the presence of beta-blockade with P.

●1508 GLIADIN-INDUCED HYPOCOMPLEMENTEMIC MEMBRANOUS GLOMERULONEPHRITIS.

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A 13-month-old presented with intractable diarrhea, marked proteinuria (8 g/24 hrs) and severe nephrotic syndrome. Small bowel biopsy showed celiac disease. Renal biopsy demonstrated epimembranous glomerulonephritis with deposition of IgG, IgA, C3, properdin and substantial amounts of gliadin. Serum complement studies showed marked depression of CH_{50} titers and classical pathway (CP) proteins C1, C2, C3 and C3-C9. Alternative pathway (AP) proteins factor B, properdin, β1H and C3bINA were equally depressed as was the lysis of unsensitized rabbit erythrocytes by the patient's serum. In all cases, values were more than 3 S.D. from the age-corrected mean. No C3NeF or circulating immune complexes by Raji cell assay were detectable. Treatment with prednisone, restricted diet, and hyperalimentation allowed for a reduction in diarrhea and proteinuria (400 mg/24 hrs) as well as an increase in all CP proteins within 2 months. Serum levels of β1H and C3bINA also rose but the remainder of the AP abnormalities persisted for several more months before returning to normal. These data suggest that gliadin, adsorbed via the GI tract and deposited within the glomerulus, may have induced the development of membranous glomerulonephritis in this child. Further, the bound gliadin may also have been responsible for the IgG-mediated CP activation and, either alone or with IgA, the more persistent AP activation.

●1509 REABSORPTION OF PHOSPHATE (Pi) DURING DEVELOPMENT BY THE ISOLATED PERFUSED KIDNEY.

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It is generally accepted that the renal transport rate of Pi is low in the developing animal and human. This conclusion is difficult to reconcile with the fact that growing subjects are able to maintain a positive external balance and a high plasma concentration of Pi even when the intake is low. Kidneys obtained from guinea pigs of <7 days and >30 days of age were perfused with Krebs-Henseleit bicarbonate buffer solution containing amounts of fraction V bovine serum albumin in concentrations appropriate to the age of the animal and Na or KH_2PO_4 in concentrations varying between 3.0 and 15.0 mg%. The results obtained:

Age (days)	n	GFR (ml/min)	FeNa (%)	FePi (%)	p
< 7	6	0.21±0.07	2.74±1.2	8.34±5.7	<0.01
>30	8	0.47±0.07	3.32±1.8	15.34±5.7	

demonstrate a lower fractional excretion (Fe) of Pi in the younger than in the older animals. The regression equations representing the relationship between filtered load (x) and amount reabsorbed (y) were: $y = 0.90x - 0.007$, ($r = 0.98$) for the <7 days and $y = 0.86x - 0.06$, ($r = 0.99$) for the >30 day old guinea pigs indicating that the absolute reabsorption rate of Pi (μg/min) was higher in the newborn than in the more mature animals over the entire range of filtered loads ($p<0.001$) and that the reabsorption rate increased more in the newborn than in the mature animals for any given increment in filtered load. Thus, contrary to previous claims, the renal transport mechanism for Pi is more efficient in the newborn than it is later in life.