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DOUBLE-MARKED LYMPHOCYTES IN MINIMAL CHANGE NEPHROTIC SYNDROME (MCNS) J.G. Bhat, H.O. Kerpen, R.G. Schacht, E.A. Gombos, B. Spinowitz, S. Handelsman, C. Rai, B. Gauthier, C. Kantor, and D.S. Baldwin. N.Y.U. Schl. of Med. and L.I.J. Hillside Med. Ctr. N.Y.

Indirect evidence supports a role for cell mediated immunity in the pathogenesis of MCNS. In the present study, the morphology and surface receptor characteristics of lymphocytes from 16 patients with MCNS and 14 controls were examined.

	MCNS		CONTROLS		P
	Mean	(SEM)	Mean	(SEM)	
% lymphocytes	35.8	(3.3)	48.6	(5.5)	<0.02
% T cells	71.3	(2.0)	67.5	(2.2)	NS
% Bsig	21.5	(2.2)	16.2	(1.0)	NS
% Beac	33.4	(2.5)	14.8	(1.3)	<0.0001

MCNS was characterized by a relative lymphocytopenia, a normal T cell count, and a discordance between B cells enumerated by the presence of surface immunoglobulins (Bsig) and by C3B receptors (Beac). By either technique of B cell count, the total number of T and B lymphocytes in MCNS exceeded that of controls, suggesting the presence of "double-marked" cells or decreased null cell population. This alteration in surface receptor characteristics of lymphocytes which was observed both in relapse and in remission, documents an abnormality, in MCNS, possibly of cell mediated immunity, which may be relavent to its pathogenesis .

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LACK OF A ROLE FOR RENAL RENIN (RCRC) IN DETERMINING SUSCEPTIBILITY TO MYOGLOBINURIC RENAL FAILURE (ARF) IN THE ABSENCE OF CHANGES IN Na INTAKE. Anil Bidani, Paul Churchill & Larry Fleischmann. (Spon. by Sanford Cohen), Dept. of Ped. & Physiol., Wayne State U. & Children's Hosp. of Mich., Det.

We have investigated the role of changes in RCRC (independent of Na intake) in determining susceptibility to ARF. RCRC was altered in either direction from control (Grp A - normal Na intake) in S.D. rats by 1% NaCl drinking + S.Q. DOCA for 4 wks (Grp B) or by a low Na diet for 6 wks (Grp C). For the next 5 days the animals were continued on the previous Na intake (Grp B1 & C1), normal Na intake (Grp B2 & C2), or 1% NaCl (Grp C3). On the 5th day the 24 hour urinary Na, Cl and Osm excretions were determined and random members from each group were sacrificed for RCRC determination. ARF was induced in the rest by injection of 10 mg/kg of 50% glycerol. Tail vein BUN's were done; results are shown (mean ± SEM). The number of animals in parenthesis.

Grp.	$U_{Na}V$ mEq/24 hr/100 gm (Pre-Glycerol)	BUN @ 48 hr mg% (Post-Glycerol)	RCRC Ng Angio I/hr mg wet wt.
A (19)	.29 ± .04	95 ± 17 (13)	457 ± 39 (6)
B1 (25)	5.46 ± .32*	43 ± 8 (14)*	132 ± 31 (11)*
C1 (19	.006 ± .001*	150 ± 21 (11)*	1480 ± 82 (8)*
B2 (24)	.64 ± .04*	78 ± 12 (13)	144 ± 40 (11)*
C2 (21)	.42 ± .04*	67 ± 15 (14)*	1702 ± 88 (7)*
C3 (21)	3.37 ± .35	27 ± 2 (15)	1238 ± 115 (6)*

*Significantly different from Control Grp A) p = < .05)

This study clearly excludes a role for RCRC in the pathogenesis of ARF since changes in Na intake, not RCRC, determine susceptibility.

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GLOMERULAR FILTRATION RATE(GFR) IN CHILDREN WITH VESICO-URETHRAL(V-U) REFLUX: Cr^{51} EDTA CLEARANCE(C) VERSUS INULIN AND ENDOGENOUS CREATININE CLEARANCE (ECC). Y.Blachar, E.H.Eliahou, A.Iana and H.Boichis. (Spon. by B.S. Kaplan) Kaplan Hosp., Dept. of Pediatrics, Rehovoth, and Sheba Medical Center, Dept. of Nephrology and Pediatric Nephrology Unit, Tel-Hashomer, Israel.

Determination of GFR by ECC, although widely used, correlates poorly with true GFR. V-U reflux occurs commonly in children with recurrent urinary tract infections and may impair accurate urine collection because of residual volume. GFR determination by single injection of Cr^{51} EDTA eliminates urine collections and is simple and reliable.

We studied GFR by 3 methods: 1) ECC with 24 and 2 hr collections; 2) inulin C; 3) Cr^{51} EDTA C calculated from 8 or 12 blood samples. The following correlations were found(r):

	Reflux	Control
ECC 24 hr vs. inulin C	-0.04	0.34
ECC 24 hr vs. Cr^{51} EDTA C	0.03	0.10
ECC 2 hr vs. inulin C	0.53	0.54
ECC 2 hr vs. Cr^{51} EDTA C	0.57	0.59
inulin C vs. Cr^{51} EDTA C	0.93	0.94
Cr^{51} EDTA 8 samples vs. 2 samples	0.97	

We conclude that: 1) ECC is a poor index of GFR in both situations; 2) Cr^{51} EDTA C is a simple and reliable method for GFR determination; 3) 2 blood samples (60 & 90 min post-injection) are sufficient for a good estimation of GFR.

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PSEUDOHYPOALDOSTERONISM (pseudoHA): SIMILAR TO BARTTER'S SYNDROME? Y.Blachar, B.Griffel, S.Levin and B.S. Kaplan. Kaplan Hosp., Dept. of Pediatrics, Rehovoth, Israel, and Montreal Children's Hosp., Dept. of Nephrology, Montreal, Canada.

Cardinal features of pseudoHA include growth failure, hyperkalemia, metabolic acidosis, salt wasting and elevated peripheral renin activity (PRA) and plasma aldosterone concentrations.

Three patients were studied. Two were sibs: R.G., 7 mo girl, PRA 242.58 ng angiotensin I/ml/hr (normal 0.96-1.67), aldosterone secretion rate 10680 ng/24 hr (normal < 300) and plasma aldosterone > 80 ng/dl (normal 5.16-10.2); the brother's cord blood PRA 18.7 and plasma aldosterone 96. Patient J.H., 4½ mo, PRA 28, plasma aldosterone 360. Treatment with mineralocorticoids failed to normalize serum electrolytes in R.G. and J.H. but this was achieved with large quantities of oral NaCl. Features of pseudoHA were prevented in the sib with NaCl supplement.

Light microscopy of renal biopsy (R.G.): many immature glomeruli, hyperplasia and hypertrophy of the JGA, arteriolar wall thickening, and, on electronmicroscopy, fusion of podocyte foot processes, hypergranularity of JGA cells.

Similarities between pseudoHA and Bartter's syndrome include growth failure, hyponatremia, salt wasting, elevated PRA and aldosterone, JGA hyperplasia and response to indomethacin. Major differences are hyperkalemia, acidosis in pseudoHA in contrast to hypokalemia, alkalosis in Bartter's syndrome.

This appears to be the first description of changes in the kidney in pseudoHA.

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IN VITRO IMMUNOGLOBULIN SYNTHESIS IN IDIOPATHIC NEPHROTIC SYNDROME (INS). Ben H. Brouhard, Randall M. Goldblum and Robert J. Cunningham. University of Texas Medical Branch, Department of Pediatrics, Galveston, Texas 77550

On the basis of serum immunoglobulin levels it has been suggested (NEJM, 293:8, 1975) that INS is caused by an immunologic defect similar to dysgammaglobulinemia with elevated IgM (DYS-M). We therefore compared the in vitro immunoglobulin production of pokeweed mitogen stimulated blood lymphocytes from 7 patients with INS (biopsy or a compatible clinical course) and 2 with DYS-M. All patients with INS were in relapse but had not taken immunosuppressants for the previous 6 months. Serum IgG was low in 6/7 patients with INS, but IgA was normal in all and IgM elevated in only one. Patients with DYS-M had low serum IgG, absent IgA but normal or high IgM. The in vitro immunoglobulin synthesis results were ($\bar{x}$ ± S.D.):

Group	IgG	IgM	IgA
Normal Adults	2639 ± 1766	3336 ± 1472	617 ± 494
INS	3587 ± 1389	6374 ± 6041	657 ± 222
DYS-M	42 ± 34	100 ± 53	6 ± 9

Patients with DYS-M but not INS produced significantly ($p < .05$) less immunoglobulins of each class than did normals. No suppressor cells were demonstrated. These results indicate that patients with INS do not have the same immunologic defect as patients with DYS-M. The role of soluble inhibitors of synthesis or increased catabolism in the reduction of serum IgG levels remains unknown.

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WHOLE BODY PROTEIN TURNOVER IN CHRONICALLY HEMODIALYZED CHILDREN. Susan B. Conley, Gilbert M. Rose, and Dennis M. Bier. Washington University School of Medicine, St. Louis Children's Hospital, Depts. of Pediatrics and Medicine, St. Louis.

Growth failure remains a concern in children on chronic hemodialysis. Inadequate intake of protein and calories is one cause. However little is known about the dynamic aspects of protein metabolism in these children. We investigated this by measuring whole body protein turnover from lysine-^{15}N enrichment during 14 hr infusions of ^{15}N labelled lysine, using newly developed gas chromatography-mass spectrometry micromethods. The patients had been on dialysis for 11.7 ± 10.7 months. All were below the 10th percentile for ht and wt. Prior to dialysis when BUN averaged 64.3 ± 38.8 mg/dl and serum creatinine 9.7 ± 5.4 mg/dl, the mean protein turnover rate was 2.36 ± .34 gm protein/kg body wt/day, a value significantly below the normal of 3.8. Protein turnover was reduced even in children ingesting 2 gm protein/kg body wt/day. To determine whether decreasing the degree of azotemia would improve protein turnover rates, studies were repeated after dialysis (average BUN 23.0 ± 15.7, serum creatinine 4.2 ± 2.6 mg/dl). Protein turnover remained identical 2.36 ± .33 gm protein/kg body wt/day. This study illustrates that even with adequate dietary protein intake, children on hemodialysis have decreased protein turnover. This is not improved by dialysis. Identification of the mechanisms responsible for this decrease should help the development of methods to increase growth rates in uremic children.