1057 USE OF SARALASIN IN THE DIAGNOSTIC EVALUATION OF HYPERTENSION IN CHILDREN. Robert C. Boerth, Laurent P. Favre, John W. Hollifield and Victor Braren. (Spon. by Thomas P. Graham, Jr.) Vanderbilt Univ. Med. Ctr., Depts. of Peds., Pharm., Med. and Urology, Nashville, Tennessee

Saralasin (S), a competetive antagonist of angiotensin II, has been used to evaluate renin-angiotensin dependent hypertension in adults. The purposes of this study were to determine 1) if S might be useful in the evaluation of hypertension in children and 2) if it would provide information about the involvement of the renin-angiotensin system in various forms of hypertension in the young. An IV infusion of S (0.4 to 0.8mg/min) was given to 21 children (4 to 18 years) with sustained hypertension (mean admission BP=162/114). All pats had complete diagnostic evaluation including renal arteriography. Pats were divided into 3 groups: hypertension secondary to renal parenchymal disease (RPH), renovascular hypertension (RVH) and essential hypertension (EH). The S test was considered to be positive when systolic BP decreased 10mmHg and diastolic BP decreased 6mmHg. A positive S test was obtained in 4 of 8 pats with RPH, 4 of 7 pats with RVH and 1 of 6 pats with EH. All pats with a positive S test had increased peripheral renin activity (PRA). Two pats with EH and 1 with RVH had increased PRA but negative S tests. Four pats with RPH (3+ and 1- S test) and all 7 pats with RVH have had a surgical procedure to help control their hypertension. These results suggest that S may be useful in the diagnostic evaluation of hypertension in children. More experience and time are required to determine whether S can help predict surgical success for RVH and RPH in the young.

1058 BARTER'S SYNDROME: EFFECTS OF LONG-TERM TREATMENT WITH PROPRANOLOL (P) AND IBUPROFREN (I). Ben H. Brouhard, Robert J. Cunningham, L. B. Travis, Dept. of Pediatrics, The University of Texas Medical Branch, Galveston, Texas, 77550.

Two brothers (RB age 19, JB age 9) with a three-year history of hypokalemic alkalosis, increased peripheral renin activity (PRA), normotension, and decreased growth rate (GR) were treated with P (2 mg/kg). This resulted in a marked decrease in PRA and an 8-20% increase in plasma volume as measured by ^{125}I-tagged albumin. Serum K was unaffected but when spironolactone and K supplements (600 meq/day) were added, it was maintained above 3.0 meq/l. One year later I was added to this regimen resulting in a further decrease in PRA. K supplements were discontinued but serum K remained above 3.0 meq/l. Prior to therapy with P and I, growth rates were 3.0-5.0 cm/year. Both have grown at 7.0-12.0 cm/year since therapy was initiated. Results are shown below:

	PRA ng/ml/1hr		Growth/cm/yr		Serum K meq/l	
	RB	JB	RB	JB	RB	JB
Control	309	>600	5.0	5.0	2.9	2.3
P	147	223	7.2	6.8	3.1	3.1
I	7.0	31	--	9.5	3.3	3.2

It is concluded that I is useful in the long-term therapy of Barter's syndrome and that the growth rate can be normalized if K balance is restored.

1059 FAMILIAL C4 DEFICIENCY WITH PROLIFERATIVE GLOMERULONEPHRITIS AND ELEVATED SWEAT ELECTROLYTE CONCENTRATIONS. Russell W. Chesney, Sheldon Horowitz, A. Vishnu Moorthy, and Henry Gewurz. The University of Wisconsin Medical School, Dept. of Pediatrics, Madison and Rush Medical College, Presbyterian-St. Lukes Hospital, Dept. of Immunology, Chicago.

Within a sibship of 5 having elevated sweat sodium (65-108 mEq/L) and chloride (33-84 mEq/L) concentrations, an 11 year old boy was found to have erythrocyte casts, proteinuria, hypertension, and isolated C4 deficiency. He has no skin rashes, angioedema or evidence of SLE Renal biopsy revealed diffuse mesangial hypercellularity and focal capillary wall thickening; immunofluorescent studies demonstrated mesangial and peripheral C3 deposition but no C4, IgG, IgM or IgA

C4 was extremely low by functional (<0.1% normal) and immunochemical (42 µg/ml) assays; CH50 < 5U/ml. All other complement components of the classical and alternate pathway were normal by immunochemical and functional assays. Preliminary family studies indicate that the C4 deficiency was inherited as an autosomal recessive trait and was carried on the HLA-A1, B8 (Maternal) and HLA-A2, B5 (Paternal) haplotypes. Further study of the linkage of this deficiency to other HLA loci (HLA-D and B cell alloantigens) and sweat chloride levels is in progress.

1060 ORAL 1α, 25-DIHYDROXYCHOLECALCIFEROL (1,25 DHCC) IN THE TREATMENT OF CHILDHOOD RENAL OSTEODYSTROPHY Russell W. Chesney, A. Vishnu Moorthy, John E. Eiseman, Diane K. Jax, and Henry F. DeLuca, The University of Wisconsin School of Medicine, Wisconsin University Hospitals, Department of Pediatrics and Biochemistry, Madison, Wisconsin.

Central to the development of renal osteodystrophy is the failure of the kidney to produce sufficient amounts of the most active natural vitamin D metabolite 1,25 DHCC. We have initiated oral therapy with 1,25 DHCC at 10-30 ngm/kg daily in 6 uremic children attempting to reverse bone disease or prevent further change. Patients with hypocalcemia or X-ray evidence of osteodystrophy while on high dose vitamin D_2 or DHT were included in this study; none had primary glomerular disease. An improvement in gait and subjective improvement in muscle strength was noted in 4 patients. Mean serum calcium concentration rose from 7.5 ± 1.6 to 10 4 ± 3 mg/dl (P<.01) in all; serum phosphate rose from 5.1 ± 2.6 to 6.8 ± 2.0 mg/dl (N.S.) and serum alkaline phosphatase fell from 527 ± 214 to 250 ± 57 IU/L (P< 02). Serum immunoreactive PTH levels fell from 864 ± 525 to 320 ± 51 pEq/ml (P<.05). X-ray evidence of osteodystrophy improved in 5. Bone mineral content, measured by single photon absorptiometry, improved by 10% in 3 patients. Serum levels of 1,25 DHCC rose in 3/3 patients. Although the follow-up period has been short, there has been an increase in growth rate. The complications found were hypercalcemia in younger patients, mild hypermagnesemia and hyperphosphatemia possibly related to the reduction in circulating PTH levels. The oral administration of 1,25 DHCC appears to be useful in the treatment of childhood renal osteodystrophy.

1061 PERITONEAL DIALYSIS IN ACUTE COPPER SULPHATE POISONING. David E. C. Cole and David S. Lirenman (Spon. by S. Segal). Univ. of British Columbia, Health Centre for Children, Dept. of Pediatrics, Vancouver, BC

A 2-year-old boy was admitted to hospital with acute hemolytic anemia, acute renal failure and cerebral dysfunction 16 hours after ingesting 20-30g supersaturated solution of copper sulphate. He was drowsy, febrile and anuric. Serum copper was 201µg/dl, ceruloplasmin 40mg/dl. Peritoneal dialysis was started and antibiotic coverage was initiated.

57 hours after ingestion with no change in serum copper levels or clinical status, exchange transfusion was carried out and salt-poor albumin (2.5g/dl) was added to the dialysate. Serum copper following dialysis was 123µg/dl and his clinical status improved. Dialysate copper concentrations were measured:

Period *	Albumin?	Concentration (µg/dl)
17-57	No	4.0
57-118	Yes	38.0
118-184	No	3.9

* hours after ingestion

Diuresis occurred on day 22. BUN and creatinine are normal 255 days following ingestion although copper (176µg/dl) and ceruloplasmin (42µg/dl) remain elevated.

These data suggest a beneficial role of exchange transfusion and peritoneal dialysis with albumin in the treatment of copper sulphate poisoning with acute renal failure.

1062 MECHANISMS OF ACIDOSIS-INDUCED PROTEINURIA. Susan B. Conley, Brenda N. Murray, Edward R. Root, Alan M. Robson. Wash Univ Sch of Med, Dept of Ped, St. Louis

For more than 50 years it has been noted that systemic acidosis induces proteinuria. Proteinuria was found to increase with increasing acidosis (Gardner, JCI,1961,40:525) and was attributed to an increase in filtered load. The present study was designed to delineate the mechanisms for this increase. Acute (<6 hr) and chronic (10-20 d) acidosis was produced in rats by oral feeding of NH_4Cl. Acute and chronic acidosis was associated with an increase in protein excretion (41.2±15.8 vs 22.0±5.1 (SD) µgm/min p<.02). This was predominantly due to an increase in albuminuria (12.90±8.50 vs 3.26±1.63 µgm/min p<.02), so that in acidosis albumin represented a greater proportion of total urine protein. Non-albumin proteinuria increased to a lesser extent. Acidosis was not associated with any significant change in GFR (2.29±0.52 vs 2.66±0.63 ml/min), filtration fraction (32.8±10.2 vs 36.0± 9.14%), or total serum protein (6.73±0.74 vs 6.73±0.18 gm/dl). Glomerular polyanion as determined by staining with both colloidal iron and alcian blue was not altered in acute acidosis but appeared to decrease with chronic acidosis. Glomerular permeability to inert polydisperse polyvinyl pyrrolidone (mol.size 20-45 Å) was increased significantly in acidotic rats.

We postulate that the proteinuria of acidosis results from the documented alteration in glomerular permeability. The conformational changes in protein molecules associated with pH changes may also contribute. Both of these mechanisms would increase the filtered protein load resulting in proteinuria.