Reversing Acidotic Hyperkalemia of Acute Renal Shut-Down by Artificial Hyperventilation. Ophelia James, Rupert Green, Abner Levkoff (Intr. by Mitchell I. Rubin), Med. Univ. of So. Carolina, Med. Univ. Hosp., Depts. of Pediatrics & Physiology, Charleston, South Carolina.

Typical EKG changes associated with hyperkalemia due to renal shut-down following cardiogenic shock in a neonate with coarctation were reversed by the inadvertent induction of respiratory alkalosis in a newborn on a respirator.

It therefore seemed reasonable to determine in dogs whether manipulation of acid base balance by respirator could reverse the hyperkalemia and acidosis associated with acute renal failure. Eight dogs anesthetized with pentobarbital were intubated with cuffed endotracheal tubes. Both ureters were then ligated and .15 N HCl was infused IV at 5 ml./min. until a partially compensated metabolic acidemia associated with a pH of approximately 7.0 and a PCO₂ of 20-24 mm Hg ensued. 90 minutes after the end of the infusion, serum potassium values had risen to 8 mEq/L from a pre-acid value of 5 mEq/L. A volume controlled respirator was then used to triple the dogs' existing respiratory minute volume. Within 120-180 minutes as the PCO₂ dropped, in general, to 10 mm Hg, or below, the blood pH returned to approximately 7.4 and the serum potassium concentration to pre-acid levels. Serum potassium concentration remained normal for the 3 to 6 hour duration of hyperventilation.

AUTOREGULATION IN THE IMMATURE KIDNEY. Pedro A. Jose, Lawrence M. Slotkoff, Stephen Montgomery, Lawrence S. Lilienfeld, Gilbert M. Eisner and Philip L. Calcagno, Georgetown University School of Medicine, Depts of Ped., Physiol. and Biophysics, Washington, D. C.

In the adult, renal autoregulation may be mediated by the renin-angiotensin system(RAS). Autoregulation has not been fully investigated in the puppy. Nine, 4-5wks. old puppies were studied. Renal blood flow(RBF) was measured by electromagnetic flow meter. Sar¹ala⁸angiotensin II(anti II)an antagonist of angiotensin II(AII)was administered into the renal artery(4ug/min)and subsequently intravenously(IV)(4ug/min). In both instances RBF(1.6+0.3ml/gm/min)was unchanged by anti II and mean arterial pressure(MAP)remained constant (78+4mmHg). In 6 puppies, during intrarenal infusion of anti II, AII given IV raised MAP to 100mmHg. RBF increased promptly from 1.6+0.3 to 2.0+0.6ml/gm/min but returned to control levels within 40sec. in spite of persistent MAP elevation. In 3 other puppies reduction of renal perfusion pressure by aortic constriction to 50mmHg did not cause a fall in RBF.

This model provides a useful new means of defining autoregulation. In these puppies, renal autoregulation, RBF and arterial pressure are not regulated by the RAS.

ANTICOAGULANT THERAPY IN "ACUTE PROGRESSIVE" GLOMERULONEPHRITIS: Randy A. Kienstra, Barbara R. Cole, Norma Alkjaersig, Anthony P. Fletcher and Alan M. Robson, Depts. Peds. & Med., Washington Univ. Sch. Med., St. Louis.

6 azotemic patients in whom renal biopsy showed crescent formation, focal necrosis and fibrin deposition ("acute progressive" glomerulonephritis), were examined by plasma fibrinogen chromatography, a technique for quantifying fibrinogen/fibrin complexes, monomeric fibrinogen, and smaller fibrinogen derivatives in plasma. Serially, pathological concentrations of fibrinogen/fibrin complexes (> 20%) were invariably demonstrated, a finding indicative of intravascular fibrin deposition, presumably intraglomerular. Heparin followed by phenindione/dipyridamole therapy resulted in return of plasma fibrinogen chromatographic findings to normal. Therapy was continued for 2 years until, on termination, chromatographic findings remained normal. Serial renal evaluation by C_{IN}, C_{PAH}, 24-hour protein excretion and urinary sediments demonstrated substantial improvement in all patients by two months. In all cases, C_{IN} was normal at one year and 2-year repeat biopsies showed apparent absence of active disease. In contrast, 2 patients with similar renal biopsy pathology did not demonstrate increased levels of fibrinogen complexes: these patients failed to respond to anticoagulant therapy. Our results suggest that "positive" plasma fibrinogen chromatographic findings are indicative of intrarenal fibrin deposition and that control of this process by long-term anticoagulant therapy is of clinical benefit.

RENAL TUBULAR RESPONSE OF THE NEWBORN DOG TO A SODIUM LOAD: Leonard I. Kleinman and Ning N. Hsieh, Univ. of Cinti. Col. of Med., Dept. of Ped., Cinti.

When equivalent I.V. saline loads were given to 30 newborn and 7 adult dogs, adults excreted 32% of the infused sodium after 2 hours compared to 10% for puppies (p<0.01). GFR increased equivalently (30%) for puppies and adults but the fraction of filtered sodium excreted (FeNa) was 6.4% for adults and 2.4% for puppies (p<0.01). In order to determine whether inability to adequately decrease tubular sodium reabsorption in the newborn dog had a proximal or distal locus, sodium reabsorption beyond the proximal tubule was blocked (D.B.) in 27 additional puppies with ethacrynic acid and chlorothiazide. Following D.B. in 9 puppies FeNa remained constant for 180 minutes with no sodium load and when dosage of diuretics varied over a two fold range. In 11 puppies FeNa was 26% following D.B. but FeNa increased to 46% (p<0.01) when a saline load was imposed on top of the D.B. In 7 puppies FeNa was 1.5% following a saline load but increased to 49% (p<0.01) when D.B. was imposed on top of the saline load. Since saline loading produced an increase in FeNa during D.B., proximal tubular Na reabsorption must have decreased during saline loading. The inability of the puppy to respond to a saline load therefore is due to increased distal sodium reabsorption.

HYPOTONIC SALINE AND FUROSEMIDE DIURESIS IN BARTTER'S SYNDROME Edward C. Kohaut, Carol J. Wilson, and L. Leighton Hill. Baylor College of Medicine, Dept. of Pediatrics, Houston.

Chamiovitz, et. al., recently demonstrated a defect in sodium (NA) reabsorption in the medullary diluting segment in a patient with Bartter's Syndrome (B.S.). We performed hypotonic saline diuresis studies on a patient (J.A.) with B.S. and followed this with furosemide (F) infusion of 1mg/kg. The following table illustrates our data compared with Chamiovitz's patient and his normal controls.

	*Vml	C _{IMl} 1.73m ²	*CH _{2O}	*C _{NA}	*CH _{2O} +C _{NA}	CH _{2O} CH _{2O} +C _{NA}	CH _{2O} V
pt. J.A.	20.36	133	9.17	10.42	19.59	46.8%	45%
J.A. - F.	24.06	134	3.74	20.37	24.11	15.5%	15%
Pt. (Cham)	16.9	128	4.2	9.1	13.1	32.0%	
Normals	13.7	129	9.0	2.1	11.1	80.0%	

*corrected to ml/100cc GFR

Although the actual free water clearance (CH_{2O}) in J.A. is comparable to established controls (9.2 vs. 9.0), it is reduced in relation to NA delivery to the ascending limb (CH_{2O} + C_{NA}) thereby supporting the contention of Chamiovitz, et. al., of defective sodium reabsorption in the diluting segment. The defect appears partial as evidenced by the change in CH_{2O} when NA delivery was increased. Since F. reduced CH_{2O} some NA reabsorption does occur in the medullary diluting segment in B.S. The increase in CH_{2O} + C_{NA} achievable as compared to controls treated similarly (19.6 vs. 11.1) might also suggest defective proximal tubular reabsorption of NA.

INTRAVENOUS DIAZOXIDE IN ACUTE GLOMERULONEPHRITIS. Edward C. Kohaut, Carol J. Wilson, and L. Leighton Hill. Baylor College of Medicine, Dept. of Pediatrics, Houston.

Diazoxide was used to treat 16 patients with hypertension secondary to post-streptococcal acute glomerulonephritis. The diagnosis of AGN was made clinically, however culture and/or serologic evidence of a recent strep infection was mandatory for retention in the study group.

Diazoxide was given by rapid intravenous injection at a dose of 5mg./kg. Since 11 patients required repeated injections, a total of 29 doses were given. The average pre-injection systolic pressure was 159 mm. of Hg. This fell by 23% within 5 minutes to an average post-injection pressure of 122 mm. of Hg. The average pre- and post-injection diastolic pressures were 104 and 71 mm. of Hg. respectively, an average decrease of 32%. There were no episodes of hypotension nor symptoms attributable to this rapid fall in blood pressure. Five patients maintained acceptable blood pressures throughout their hospital course. Eleven required repeat doses. The interval between doses averaged 41 hours (range 5-95).

Four patients had blood sugar elevations above 120mgm%, the highest was 155mgm%. No other adverse side effects were noted.

We feel diazoxide is effective in treating hypertension associated with AGN. It is faster acting than any of the currently used drugs. It does not produce CNS depression as seen with reserpine and is less likely to cause hypotension than intravenous hydralazine or methyl dopa.