73 The development of proximal tubular (PT) Na-K-ATPase has been studied by determining the enzyme in 100 μ sections from the outer cortex (consisting of approximately 95% PT) in 8, 20, and 40 days old male sprague Dawley rats. The ATPase concentration is related to the protein concentration (μ mol Pi/mg protein) and the values in the different age groups (n=10-12) were for 8 days old rats 2.3 ± 0.85 , for 20 days old rats 5.06 ± 1.11 and for 40 days old rats 8.46 ± 1.33 . Rats in the different age groups were then treated with aldosterone or oestriol for 3 days and thereafter sacrificed for determination of outer cortical ATPase. Both hormones significantly increased ATPase content in 20 d old rats and the increase was dependent on the hormone dose up to a certain level. When maximal hormone doses were given, aldosterone was found to be the most potent inductor. 40-80 μ g aldosterone/kg/day increased the ATPase content to adult levels in the 20 d old rats. In 10 d old rats maximal doses of aldosterone resulted in a 2-fold increase of ATPase, while oestriol had no significant effect in this age group. The results suggest that aldosterone is the specific inductor of the Na-K-ATPase of PT. This would explain why the aldosterone levels are so high during a fairly long period of postnatal life. In the rat the age of 20 d appears to be the most sensitive period for enzyme induction in the PT. The ultrastructural and functional characteristics of the PT cell at this stage of development will be specified.

74 HEMOGLOBIN A1c IN THE STREPTOZOTOCIN (STZ) TREATED OR PANCREATECTOMIZED (Px) RHESUS MONKEY (RM). John EG, Justice, P., Jonnasson O. Univ. of Ill. and Cook County Hosp., Dept. of Ped. and Surg. Chicago. U.S.A.
HbA1c, and glycosylated minor variant of HbA, is elevated in diabetic patients (D). HbA1c is used to monitor adequacy of D control. It has been suggested that elevated HbA1c in D may have adverse effects on tissue oxygenation and function. In our study, RM model with acquired glucose intolerance (STZ-Insulin (I) dependent, on I 2-7 U/day, or Px, 2-4 years; on I 2-7 U/day) was evaluated for the presence of HbA1c and for evidence of a possible influence on kidney (K) function and histology. B10 Rx 70 columns 1x15 were used for separation of Ala, Alb, Alc and A. Determination of GFR, RPF, and 24 hr, urinary protein (P) were done in 7 STZ, 8 Px and 29 control (C) animals. Kidney biopsy was evaluated by light microscopy (L). Fasting glucose in STZ varied from 200-400mg% and in Px 150-400mg%. GFR and RPF in both groups were not significantly different from C. There was significant P in STZ (118 ± 50 mg%)* and in Px (70 ± 20 mg%)* compared to C (13 ± 14 mg). HbA1c was also significantly elevated in STZ ($13 \pm 1\%$) and Px ($12 \pm 2\%$)* compared to C ($0.9 \pm 1\%$). In 4/5 STZ and 3/5 Px changes by L consisted of basement membrane thickening and increase in mesangial matrix. Our finding of elevated HbA1c in D RM offers a model for studying this Hb in a non human D primate and suggests inadequate D control. Structural changes in K and proteinuria may be the consequence of elevated HbA1c in the already vulnerable diabetic RM. Further histochemical studies are required to verify this hypothesis. *P < .05.

75 EFFECT OF SODIUM LOADING ON RENAL FUNCTION IN RATS WITH CONGENITAL UNILATERAL HYDRONEPHROSIS. Boineau, F. G., Meyer, J. and Lewy, J. E. Tulane University School of Medicine, New Orleans, LA, USA.
The mechanism(s) responsible for impaired renal function in chronic renal obstruction is unclear. A colony of Wistar rats with congenital unilateral hydronephrosis was studied before and after an acute sodium load. The Control observations were made while rats were mildly hydropenic. 5mEq/Kg BW NaCl was infused intravenously over sixty minutes and the observations repeated (After Na*). Renal function, urinary sodium and volume were measured in the right hydronephrotic (Hydro K) and left normal kidney (Nor K) of the same rat.

CIN	CPAH	F.F.	V	V/GFR	UNaV/GFR
μ l/min/gm Kidney			μ l/min	%	μ Eq/min/100 μ l GFR
Control*					
Hydro K 481	1230	0.35	3.2	1.51	0.051
Nor K 700†	1717±	0.41†	2.8	0.45†	0.007†
After Na**					
Hydro K 845	2414	0.35	9.0	1.06	0.067
Nor K 870	2399	0.37	8.4	1.06	0.046

*Mean values (5 rats); †p<0.05 by paired t test Hydro K vs. NorK.

These results indicate that the renal functional changes are reversible by acute sodium loading. Since acute sodium loading is known to alter blood levels of renin, angiotensin and prostaglandins, the reduction in renal function in the hydronephrotic kidneys may be due to an imbalance in renal vasoactive peptides produced within the hydronephrotic kidney.

76 I.V. SALINE LOADING AFTER ACUTE POST-STREPTOCOCCAL GLOMERULONEPHRITIS (PSAGN), Drukker, A., Shauli, S., Erlichman, M., Stankiewicz, H. and Mor, J., Pediatric Renal Unit, Shaare Zedek Medical Center, Jerusalem, Israel.
The aim of this study was to verify whether exaggerated natriuresis is always present after PSAGN, as previously reported. Nine patients (7 children, 2 adults) two years after PSAGN and four healthy control subjects (3 adults, 1 child) were studied. All subjects had normal urinalysis, GFR and BP. Following two control periods of 30 min (I, II) an i.v. saline load ($2.5\% \text{ NaCl}$, $1\text{L}/1.73\text{m}^2$) was given over 45 min (III). GFR, UNaV and venous HTC were measured during these periods as well as during four 30 min collection periods following completion of the saline load (IV, V, VI, VII).

PERIOD	GFR (cc/min/1.73m ²)		UNaV (μ Eq/min/1.73m ²)	
	Control	Patient	Control	Patient
I	105	115	116	145
II	96	112	108	139
III	103	100	224	342
IV	95	105	592	468
V	93	117	524	538
VI	86	88	518	402
VII	92	99	419	396

GFR was comparable in the two groups. No significant differences were found in absolute or fractional excretion of sodium. We thus found no evidence for exaggerated natriuresis two years after PSAGN. We conclude that PSAGN as seen by us in Israel, carries a good (short-term) clinical and renal functional prognosis.

77 EFFECT OF DIAZOXIDE ON PROXIMAL TUBULAR SODIUM REABSORPTION. Allen W.R., Brouhard B.H. and Lynch R.E. Department of Pediatrics, The University of Texas Medical Branch, Galveston, Texas, U.S.A.

Diazoxide, a potent antihypertensive, causes sodium retention when given systemically. However, renal artery (RA) infusion causes a natriuresis. The mechanism of this effect was investigated by recollection micropuncture of the superficial proximal tubule (SPT) in anesthetized dogs. During RA infusion of saline (NS) or diazoxide, initial and recollection samples were obtained from the late SPT. Renal blood flow (RBF) was monitored by electromagnetic flowmeter. In 4 control dogs the RA was infused with NS throughout. Glomerular filtration rate (GFR), urine flow rate (V), Na⁺ excretion (UNaV), Hct, BP and SPT fractional Na reabsorption (FR_{Na}) did not change (p>0.05) from initial to recollection periods.

In 7 dogs initial collections were made during NS infusion. Diazoxide was then begun at 0.04 mg/kg/min and adjusted to increase RBF during recollection.

	RBF	UNaV	GFR	V	TF/PIN	FR _{Na}
initial	145 ml	5.6 meq	25.9 ml	.13 ml	1.65	37.6%
recoll	166*min	37.3*min	31.8 min	.34*min	1.51*	31.1*

*p<0.05

Intrarenal Diazoxide causes increased Na⁺ delivery out of the SPT. This may result from altered peritubular physical forces or direct tubular action and may contribute to the natriuresis, although distal nephron effects are not excluded.

78 DOPAMINE (D) BLOCKADE DURING SALINE LOADING (SL) IN RATS. Pelayo, JC, Jose, PA, Slotkoff, LM & Eisner, GM. Georgetown Univ. School of Medicine, Wash. D.C.

D may participate in the natriuresis which follows SL. To determine the role of D as a natriuretic factor in rats, micropuncture of the proximal tubule (PT) and saline loading (SL) was performed in 5 Wistar rats (I) during hydropenia (H) and SL. 5 rats (II) received IV Cis-flupenthixol, a D blocking agent, and 5 (III) received IV Trans-flupenthixol, an isomer nearly devoid of anti-D activity. No significant difference between I and III was found.

GROUP	SNRFR*	TF/P In	GFR ml/min	UNaV uEq/min	FENa %
I H	32±2.9	2.3±0.1	2.0±0.3	0.07±0.002	0.03±0.006
SL	46±3.0a	1.4±0.1a	2.7±0.1	6.40±1.800a	1.70±0.300a
II H	30±2.8	2.3±0.3	1.4±0.1	0.05±0.009	0.03±0.007
SL	40±4.0	1.4±0.1a	1.9±0.1	1.20±0.300ab	0.50±0.100ab

Mean±SE, a = P<0.05 H vs. SL, b = P<0.05 I vs. II. * = ml/min.

TF/P In = tubular fluid to plasma inulin. TF/P In were similar in all groups during H or acute SL, indicating a lack of action of D at PT. V, UNaV and FENa were significantly lower after SL in II as compared to I and III.

In conclusion: 1) D blockade does not have an effect on PT or whole kidney function in the H state, and 2) following SL, D blockade attenuated the diuretic response without affecting GFR, SNRFR or PT reabsorption. Thus D may contribute to the natriuresis of SL by direct tubular action beyond the PT or in deeper nephrons.