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SERIAL MYOGLOBIN DETERMINATIONS IN NEONATES.

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Rhabdomyolysis resulting in myoglobinemia is associated with renal failure in adults. We examined the hypothesis that birth asphyxia in neonates results in rhabdomyolysis with subsequent release of myoglobin. Serial serum samples were obtained on a population of 20 neonates (9 asphyxiated and 11 non-asphyxiated) during the first four days of life. Serum samples were analyzed by radioimmunoassay for myoglobin (NMS Pharmaceuticals, Inc., Newport Beach, CA). Asphyxia (5 minute Apgar ≤ 5) predicted an elevation of myoglobin (> 200 ng/ml) at 12 and 24 hours (Chi square, $p < .005$). Serum values comparable to those reported in adults with myoglobinemic renal failure were seen in some neonates. High serum myoglobin values (> 1000 ng/ml) at 12 hours were associated with severe oliguria at 24 and 48 hours (24 hrs = $0.27 \pm .28$ cc/kg/hr, 48 hrs = $0.70 \pm .46$ cc/kg/hr, $n = 3$). Myoglobinemia may be associated with poor urine output during the neonatal period and serum myoglobin determination may have a value as a marker for birth asphyxia. This preliminary study suggests that birth asphyxia results in rhabdomyolysis with the subsequent release of myoglobin in some neonates.

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EVALUATION OF SINGLE DOSE GENTAMICIN THERAPY (ST) IN COMPLICATED URINARY TRACT INFECTIONS (UTI).

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ST is effective in uncomplicated lower UTI but its efficacy in complicated cases has not been studied. Eighteen (18) consecutive cases (all girls) of UTI associated with confirmed Pyelonephritis and/or structurally abnormal urinary tract (AUT) were treated with ST. An equal number of similar cases (all girls) were treated alternately with a 10 day conventional therapy (CT) as control. Each patient was examined at least 4 times during 1 month follow-up for recurrence and cure (defined as absence of recurrence). The two groups were comparable in age, sex and incidence of AUTs. Cure and recurrence rates 2-4 days and 1 month after therapy are presented (table).

Group (N)	Age		Urinary Tract		#(%) Recurred at		#(%) Cured at	
	Mean	Normal	Abnormal		2-4 days	1 Mth.	2-4 days	1 Mth.
ST (18)	7.5	9	9	0(0)	7(40)	18(100)	11(60)	
CT (18)	8.0	8	10	1(6)	8(44)	17(94)	11(56)	

Urine was sterilized at 2-4 days in almost every case in both groups. Recurrence and cure rates were comparable ($P > 0.5$). All recurrences were reinfections except one in ST group which was a relapse. ST was as effective as CT. Further studies would help confirm the role of ST in complicated UTIs.

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ULTRASOUND DIAGNOSIS OF NEPHROCALCINOSIS DURING TREATMENT OF HEREDITARY RICKETS. Jonathan Kronick, Paul Goodyer, Sigrid Jegquier, and Terry Reade,

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Patients with autosomal recessive vitamin D dependency, type I (ARVDD) are treated with vitamin D (Vit. D); those with X-linked hypophosphatemia (XLH) also receive oral phosphate. Since episodic hypercalcemia and hypercalciuria are complications of therapy, we performed renal ultrasounds to detect nephrocalcinosis (NC). Ten patients with ARVDD were treated from infancy with Vit. D or its analogs for a mean of 12 years (range 3-20). Two patients (treated 4.5 and 9 years) had NC (echodense renal pyramids). Mean serum calcium (9.2 , 9.7 mg/dl) and UCa/creat ratios (0.15 , 0.17) did not differ from patients without NC. Among 17 patients with XLH, 8 had NC. Duration of treatment in affected and unaffected patients was not significantly different ($p=0.09$). Therapy began before age 3 in all affected XLH patients, whereas in unaffected patients treatment began after 4 years (mean 18 years of age, $p=0.001$). Hypercalciuria during treatment was more frequent in XLH than ARVDD. NC was not found in untreated XLH patients despite longstanding severe disease. Estimated creatinine clearance was normal in all patients except one with XLH and severe 2° hyperparathyroidism. Water deprivation tests suggested a modest deficit of concentrating ability in many patients with XLH. Vit. D treatment of inherited rickets is associated with NC, especially in patients with hypophosphatemia treated from infancy.

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CONTINUOUS ARTERIAL-VENOUS HEMOFILTRATION (CAVH) IN PEDIATRICS. Kenneth V. Lieberman (Spon. by Ed Brown). Mt. Sinai School of Med., Dept. of Ped., NY, NY.

CAVH is a new extracorporeal technique for the treatment of renal failure. Slow continuous ultrafiltration, the driving force for blood flow being provided by the patient's own systemic blood pressure, results in the gradual removal of fluid and solutes. CAVH is especially applicable in the hemodynamically unstable, critically ill patient. Herein is described the first use of this technique in pediatric patients.

A 1300 gram premature female, delivered by emergency Caesarian section because of abruptio placenta and fetal distress, developed renal failure secondary to intrapartum shock and asphyxia (APGARs 1/5). On day 3 of life CAVH was initiated by connecting the umbilical artery line to the blood inlet port of a miniature Amicon diafilter; the umbilical venous line was connected to the blood outlet port. Over 24 hours 68 cc of ultrafiltrate was generated, the patient's edema markedly diminished.

A 10 year old boy with infantile polycystic kidney disease and congenital hepatic fibrosis had been suffering steadily declining renal and hepatic function over about 8 months. Diuretic resistant anasarca and uremia developed. CAVH was performed with an Amicon Diafilter - 20 connected via a Scribner shunt. Ultrafiltrate formed at 500-700 ml/hour necessitating partial replacement with a modified Ringer's solution. After 24 hours creatinine and BUN fell and the edema was markedly reduced.

CAVH solute clearance is by convection. Measured transplant kinetics: Na, K, HCO_3 , BUN, creatinine, phosphorus-unrestricted; bilirubin-negligible; calcium-limited to free (ionized) moiety.

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MATURATIONAL CHARACTERISTICS OF RABBIT BASOLATERAL MEMBRANE (BLM). Michael A. Linshaw, Cathryn Bauman and Larry Welling, Univ. of Ks. Med. Ctr., Dept. of

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Overall renal function is thought to increase during development, but it is unclear if a continuum of changes occurs during as well as after nephrogenesis (2½-3 weeks in rabbit). In previous studies (AJP, 1983) we found no change in BLM characteristics in outer cortical proximal convoluted tubules (PCT) during nephrogenesis. This finding is not surprising since tubules from the outer nephrogenic zone are of similar maturity even with advancing age. We therefore studied juxtamedullary (JM) tubules to look for developmental changes in the BLM from more mature tubules. 65 early collapsed JMPCT were tightly crimped with micropipets. Changes in cell volume, assessed by measuring outer diameter with an image splitting eyepiece, were used to measure rate of trans BLM fluid movement as tubule volume increased in 10^{-6} M ouabain. Results are $\text{M} \pm \text{SE}$.

Age-days	Outer diameter- μm	Swelling rate- $\text{nl}/\text{min}/\text{mm}$
2-7 (N,19)	27.5 ± 0.7	$.016 \pm .002$
14-18 (N,20)	28.4 ± 0.7	$.018 \pm .002$
28-43 (N,15)	34.2 ± 0.7	$.028 \pm .004$
Adult (N,11)	37.8 ± 0.7	$.040 \pm .007$

Tubule size and swelling rate were constant in the first 17 days of life but increased by 4-6 weeks. Early changes in BLM surface area were also inapparent. We suggest from this data and that of Arant (J.Peds, 1978) and Schwartz (AJP, 1983) that nephron function during nephrogenesis is relatively dormant and increases only after nephrogenesis is completed.

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RESPONSE TO FUROSEMIDE IN HYPERKALEMIC RENAL TUBULAR ACIDOSIS (RTA). Kimo C. Stine and Michael A. Linshaw

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Hyperkalemic RTA in young children usually indicates renal or adrenal insufficiency, adrenal hyperplasia or a form of tubular resistance to mineralocorticoid. NH_4Cl used to evaluate acidifying ability may augment pre-existing hyperkalemia to potentially dangerous cardiotoxic levels. An infant with profound neonatal metabolic alkalosis secondary to maternal psychogenic vomiting was found to have hyperkalemic RTA at two months of age. Lab values included, in mEq/L : Na 138, Cl 105, K 5.8, HCO_3 18, BUN, blood sugar, serum creatinine and cortisol levels and BP were normal. Serum aldosterone (225 ng/ml) and plasma renin activity (20.1 ng/ml/hr) were high. Neither Florinef .1 mg/day nor Florinef + 4 days of supplemental NaCl 6 mEq/kg/day corrected the hyperkalemia, but renin and aldosterone levels dropped during this time. After Furosemide (F) 1 mg/kg IV:

	Serum K mEq/L	Serum HCO_3 mEq/L	Urine pH	FE _K
Baseline	6.4	16	6.23	14%
1 hr Post F			4.5	49.5%
4 hr Post F	4.0	26	4.5	24.3%

Fractional excretion of K rose and urine pH dropped within one hour after Furosemide and serum K and HCO_3 were normal after 4 hours. Twelve mEq/kg/day of NaHCO_3 were sufficient to correct the hyperkalemic acidosis. We conclude that, as in adults (Kurtzman KI, 1983 and Arruda KI, 1983), Furosemide stimulates acid secretion in the infant and is useful in the evaluation of RTA without the risk of worsening hyperkalemia.