lated to the dosage of prednisone used for immunosuppression, the absence of growth potential at the time of transplantation and the development of chronic rejection.

57 *Nocturnal Enuresis: Correlation with Bacteriuria, Proteinuria and Dysuria.* WARREN F. DODGE, EVELYN F. WEST and LUTHER B. TRAVIS, Univ. of Texas Med. Branch, Dept. of Ped., Galveston, Texas.

Nocturnal enuresis has troubled parents and physicians at least since the 16th century. Theories as to its cause and cure have varied with the fad and fashion of the times and, until recent years, most studies have been remarkable principally for sampling bias and lack of controls. Myths which have been recently discarded include those relating it to deep sleep, lack of early toilet training, spina bifida occulta, low intelligence or common behavior problems. The present investigations of renal disease in school children presented a unique opportunity to evaluate the role of abnormal urinary findings in enuretic children and their non-enuretic classmates as well as re-examination of its association with sex, ethnic group, socio-economic level, birth order and mother's level of education and marital status.

The prevalence of current bed-wetting for 6- to 10-year-old children was noted to be 18% for girls and 24% for boys ($p < 0.001$); and to differ significantly by ethnic group for girls but not for boys; by socio-economic level and family size for boys but not for girls; and for both sexes by mother's education but not by the other demographic characteristics examined. For girls, prevalence of significant bacteriuria increased in association with both presence and frequency of current enuresis ($p < 0.005$) and past complaint of dysuria ($p < 0.025$). No significant difference for prevalence of proteinuria in boys or girls was noted when analyzed by current enuresis or past complaint of dysuria.

58 *Measurement of Renal Function by Radionuclide Disappearance Curves.* GERALDINE I. SILKALNS, DONALD L. JECK, ADRIAN SPITZER, CHESTER M. EDELMANN, Jr. and M. DONALD BLAUFOX, Albert Einstein Coll. of Med., Dept. of Ped. and Radiol., Bronx, New York.

Reports based on adult patients suggest that either GFR or RPF can be measured accurately from the rate of disappearance of a radionuclide from the blood following a single intravenous injection. Although the advantage of avoiding urine collections in children is obvious, few attempts have been made to validate the method in this age group. Inulin, creatinine, and PAH clearances were performed and compared with simultaneous measurements obtained from iothalamate ^{125}I or orthoiodohippurate ^{131}I disappearance curves calculated as clearances by compartmental analysis (open, two compartment system). The results obtained in 31 patients ranging in age from 18 months to 16 years, with levels of renal function as estimated by C_{IN} between 20 and 158 ml/min/1.73 m^2, were:

		n	ratio	r
^{125} I vs.	Inulin	12	0.96	0.93
	Creatinine	16	1.05	0.94
^{131}I vs.	PAH	6	0.81	0.97

No differences attributable to age or level of function were observed. It appears, therefore, that GFR or RPF can be estimated validly in children using the disappearance of a radionuclide from the blood.

59 *Hypersarcosinemia: New Observations.* F. H. GLORIEUX, F. MOHYUDDIN, D. T. WHELAN and C. R. SCRIVER, McGill Univ., Children's Hosp. Res Inst., Montreal.

Sarcosinuria was found in a 10-year-old French-Canadian boy with normal IQ, small stature (< 3rd percentile) and bilateral contractures of lower limb muscles. Coexistent hypersarcosinemia (0.18–0.34 μM; normal < 0.02 μM) was unmodified by large supplements of folic acid, the coenzyme for the presumably abnormal apoenzyme, sarcosine dehydrogenase. It is appropriate to screen urine for this trait, since sarcosine is cleared rapidly by kidney (8–35 ml/min/1.73 m^2). The Tm sarcosine is about 160 μmoles/min/1.73 m^2 in the 'blocked catabolic mutant'. Sarcosine appears to interact weakly with a system shared by imino-acids and glycine. Sarcosine loading (100 mg/kg p.o.) produced 3 different responses in subjects. Normal: rapid disappearance of sarcosine from plasma ($t\,½ \sim 30$ min) with a fall in plasma glycine. Proband: greatly delayed disappearance of plasma sarcosine ($t\,½ \sim 153$ min) with no change in plasma glycine. Parents of proband: initial plasma sarcosine concentration normal but delayed disappearance of plasma sarcosine ($t\,½ \sim 68$ min) with a rising plasma glycine level. These findings suggest the trait is autosomal recessive in this pedigree. We were unable to detect sarcosine dehydrogenase activity in normal skin fibroblasts sub-cultured for 14 regenerations; consequently lack of activity in the proband's fibroblasts is of no significance. (Research supported by grants from Quebec MRC and MRC, Canada.)

60 *Studies on Alkaline Phosphatase in Hypophosphatasia.* YOUNG J. KIM and LYMAN A. PAGE, Dept. of Ped., Stanford Univ. Sch. of Med.

Central to an understanding of the pathogenesis of hypophosphatasia is the question of whether or not there is a deficiency of alkaline phosphatase (AP) activity in bone. A recently reported patient with the full syndrome had consistently normal total AP activity in serum and the suggestion was made that deficiency of phosphatase was not of primary pathogenic importance [SCRIVER and CAMERON, New Engl. J. Med. *281:* 684, 1969].

We have studied two children who have the typical osseous and dental lesions of hypophosphatasia, markedly increased excretion of phosphoethanolamine, and total activity of serum AP in the normal adult range. AP was undetectable histochemically in leukocytes of the patients and was normal in family members. Extraction of the patients' leukocytes yielded extremely low activity. Electrophoresis of the patients' sera on polyacrylamide gel consistently yielded a single major band of phosphatase activity with a rate of migration intermediate between the rates of the two fast bands seen in normal sera. The two fast bands in normal sera have been identified as hepatic and osseous AP by comparison with extracts of tissues. Although the dominant band in the patients appeared to migrate differently from any seen in normal sera, there were no unusual isozyme patterns of AP from sera and leukocytes of family members. Studies of sensitivities of various AP to heat, *l*-phenylalanine, and urea did