Further light has been shed on the riddle of the small scarred contracted kidney with the fashionable term "reflux nephropathy". At the last IPNS meeting in Finland, a great amount of interest was aroused about the subject, resulting in a work shop conference which convened in June, 1978 in Bermuda. A monograph of these proceedings was published, edited by Hodson and Kincaid-Smith entitled Reflux Nephropathy. This volume presents a variety of controversial subjects that involve urologists, nephrologists, radiologists, pathologists, pediatricians, and other specialists. Unfortunately, emotional and anecdotal arguments are often injected into these debates. In this seminar we hope to offer an update on these subjects and focus on the issues of greatest disagreement. May I first introduce our distinguished panel:

Prof. George A. Richard, University of Florida, Gainesville, FL
Prof. Jan Winberg, Karolinska Hospital, Stockholm, Sweden
Dr. Jean Smellie, University College Hospital, London, UK
Prof. Hermann Olbing, Universitatskinderklinik, Essen, W. Germany
Prof. Robert Jeffs, Johns Hopkins University, Baltimore, MD
Mr. Phillip Ransley, Institute of Child Health, London, UK

This brief introduction and update of the subject may indeed be confusing and is intentionally so.

Is VUR normal? We used to think that it was distinctly abnormal; however, a study done by Kollermann and Ludwig in 1967 showed that 30% of children under three years of age have mild reflux. Animal experiments, by Lenaghan and Cussen in 1968 showed that 80% of pups under six months of age reflux and Roberts in 1974 showed a high incidence of reflux in monkeys under three years of age. The concept of "marginal
competence" of the antireflux mechanism in normal children may be valid.

What is the pathogenesis of VUR? Most of us feel it is a congenital anomaly of the UV junction with a laterally displaced abnormal ureteric orifice and a short intramural ureter. If the marginal competence concept is valid, uninhibited contractions seen in young children at the time of bladder training with increased bladder pressures may stress the UV junction and contribute to the development of VUR. Chronic cystitis has been felt to cause reflux, however, there is little evidence that cystograms done with the bladder infected have any higher incidence of VUR than those carried out in uninfected bladders (Leibowitz).

Is reflux nephropathy a useful term? RN is defined as calyceal clubbing, adjacent cortical scarring, and arrest of renal growth. This term, coined by Baily in 1973, as an alternative to chronic pyelonephritis was intended to stress that VUR is essential to the pathogenesis of this lesion. Habib has called this lesion "congenital segmental hypoplasia"; the Ask-Upmark kidney may be the same renal lesion.

How does intrarenal reflux fit into the picture? Ransley's "big bang" theory postulates that areas of renal scarring are drained by flat or concave renal papillae which allow IRR. This permits pyelo-tubular backflow with pyelo-interstitial extravasation. While IRR may occur in 5-15% of newborns and infants with reflux, it is rare after four years of age (Rolleston). As the distribution of non-convex papillae is the same in all age groups, the relatively larger size of collecting ducts in infants is thought to be responsible for their higher incidence of IRR (Tamminen). Hodson and Rolleston feel that