ences, non-proliferative sclerosing lesions ('focal glomerulosclerosis') in 12 instances. Its recognition is important since it is a progressive condition showing almost total lack of response to therapy. The fully developed lesion is characterized by partly and completely sclerosed glomeruli as well as normal glomeruli, with tubular atrophy and interstitial fibrosis. Though early lesions may not be distinguishable on light microscopy from minimal changes, this latter condition can usually be ruled out on clinical grounds, and by the appearance of localized thinning as well as thickening of the capillary basement membrane, on electron microscopy. We have now observed the lesion in 18 children, aged 2 months to 14 years at onset; 13 were girls. Thirteen had the nephrotic syndrome, 3 a mixed nephritic-nephrotic presentation, and 2 symptomless proteinuria. Thirteen had haematuria and 8 hypertension. Proteinuria selectivity was impaired in 16 out of 17 cases; serum β1-globulin levels were normal. One child out of 16 responded to corticosteroids and is still in remission. None responded to either cyclophosphamide or azathioprine. Three have died, one is on dialysis and three have renal insufficiency. The remaining 10 have persistent proteinuria.

REFERENCE


P. M. Dunn (Bristol). 'Congenital Dislocation of the Hips and Congenital Renal Anomalies.'

Since Potter's first report in 1946 there have been many publications concerned with the presence at birth of various facial and musculoskeletal deformities in babies with congenital renal anomalies. Clinicopathological and statistical studies made by the writer over a 10-year period strongly support the widely held view that these various deformities occur as the result of pressure because of oligohydramnios due to fetal oliguria or anuria. Among the cases studied were 12 infants that were also noted to have congenital dislocation of the hips (CDH) at birth. All these infants died soon after birth. Postmortem examination revealed a wide range of renal and urinary tract malformation. Dissection of the hips joints confirmed the clinical diagnosis in every case and displayed a spectrum of pathology which illustrates the progression from mild hip-joint instability to the 'late' CDH changes normally associated with cases that remain untreated for two or more years. This association between anomalies of the kidneys and urinary tract and CDH does not appear to have been noted in previous reports. Its importance lies particularly on the light it throws on the aetiology of congenital dislocation of the hips.

W. Hamilton (Glasgow). 'Re-appraisal of Salt-Losing and Non-Salt-Losing Variants of C21-Hydroxylase Deficiency.' To be published elsewhere.
Congenital dislocation of the hips and congenital renal anomalies.

P M Dunn

Arch Dis Child 1971 46: 878
doi: 10.1136/adc.46.250.878

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