PAEDIATRIC PATHOLOGY CLUB

Proceedings of the Third Annual Meeting

The third meeting of the Paediatric Pathology Club took place at the Children’s Hospital, Sheffield, on November 8 and 9, 1957. Sixty members and guests attended.

Dinner was taken at the George Hotel, Hathersage, on the evening of Friday, November 8. There followed a discussion on pyelonephritis at which the principal speakers were W. W. Payne (London), R. C. B. Pugh (London), R. B. Zachary (Sheffield), A. M. MacDonald (Glasgow), B. I. Ivemark (Stockholm), K. B. Rogers (Birmingham), and A. G. Marshall (Wolverhampton).

The afternoon of Saturday, November 9, was taken up with a discussion of the National Perinatal Mortality Survey and a report by Dr. N. Brown on the pilot survey at Bristol.

Summaries of Original Papers

P. S. MacFarlane (Glasgow). ‘Haemolytic Anaemia associated with Acute Renal Damage in Infancy.’

Eight cases, in which a severe haemolytic anaemia was associated with renal damage, were described. The recent frequent association of what are rare diseases in infancy, may be of significance and the possibility that this is a new syndrome must be kept in mind.

The striking red cell morphology with frequent ‘burr’ cells was demonstrated.

Seven cases progressed to fatal uraemia in spite of adequate treatment of the haemolysis. At necropsy one case showed confluent cortical necrosis, five cases acute necrotizing glomerulitis and one case a severe tubular lesion with less severe glomerular damage. One case recovered.

This series suggests that the haemolytic anaemia does not cause the renal damage or vice versa, but rather that the two are concomitants and that a common aetiology is to be sought. The ‘burr’ cell phenomenon was present in two cases before a significant rise in blood urea had taken place.

There is no convincing evidence of an antigen-antibody reaction and the fundamental aetiology remains obscure.

R. C. B. Pugh (London). ‘Pyelonephritis.’ The term ‘pyelitis’ is probably unjustified as in nearly every case of so-called pyelitis there is also involvement of the renal parenchyma.

In a series of 150 necropsies in children, pyelonephritis was found in ten cases. Of these, six children had an underlying deformity of the urinary tract and in two cases pyelonephritis complicated tuberculosis.

An analysis of eight fatal cases of posterior urethral valves showed that in all there was pyelonephritis, which led to the development of renal rickets in two cases and to hypertension in the four older children.

When considering the morbid anatomical changes in pyelonephritis, the description of Weiss and Parker cannot be bettered, although a distinction should be made between the disease in neonates and in slightly older children, in whom the changes are very similar to those seen in adults.

The appearance of vascular lesions in pyelonephritis is also important especially as they resemble intrinsically those seen in hypertension. However, their distribution is different as they are confined to the scarred areas in pyelonephritis but occur in both the scarred and non-scarred areas in pyelonephritis complicated by hypertension.

The cases of glomerulonephritis, nephritis, pyelonephritis and nephrosis that occur in the neonatal period are a complex and as yet incompletely understood group.

N. O. Ericsson and B. I. Ivemark (Karolinska Institutet, Stockholm). ‘Renal Dysplasia and Pyelonephritis in Infants and Children.’ Clinical, radiological and pathological examinations were performed on 34 children, 30 of whom showed infections of the urinary tract. Eight were under 1 year of age, and 26 of them from 1 to 11 years. Malformations of the urinary tract predominated, 16 patients having ureterocele and nine ectopic ureters, an inevitable composition of paediatric material, as cases with no such malformations rarely undergo operation. Thirty-one females, and three males were encountered, a sex distribution due to the female predominance in pyelonephritis and ureteric malformations rather than in renal dysplasia. No preponderance of left-sided abnormalities was found.

The specimens were grouped in three categories. (1) Dysplastic (15 specimens) malformed kidneys with numerous primitive ducts resembling the mesonephric duct. Thirteen showed pyelonephritis. (2) Presumably dysplastic (16 specimens) mostly hydronephrotic kidneys with few medullary ducts (‘medullary dysplasia’) and primitive cortical ‘ductules’, resembling mesonephric ductules. All specimens displayed severe pyelonephritis. (3) No obvious renal dysplasia (three specimens). Ductules: no medullary dysplasia. Striking inflammatory changes.

The microscopic findings suggest abnormal budding of the ureter to be the underlying cause of the dysplasia. The cortical ductules described may be evidence of renal dysplasia—possibly residual mesonephric tubules or abnormal proliferation of ureteric buds. Concomitant
inflammation in all patients surviving more than seven days is an indication that renal dysplasia is an important predisposing element in the pyelonephritis of infancy and childhood.

C. G. Paine (Sheffield). 'Changes in the Placenta with Particular Reference to Essential Hypertension and Pre-eclamptic Toxaemia.' A description of the normal ageing changes in placental tissue was given. This related to the villous stroma which was converted from primitive mesodermal tissue to adult fibrous tissue, the arterioles of the villous stalks which changed from primitive blood spaces to relatively thick, muscular walled tubes to deal with the increasing volume of foetal blood, the villous blood vessels with special reference to subtrophoblastic sinusoid formation in the late weeks of pregnancy, and the syncitiotrophoblast which started as a thick plastic multinucleus structure, and finished as a thin membrane capable of rapid metabolite exchange at full term. A description was given of the distribution of lipoid with normal syncitium.

The normal changes were compared with those seen in essential hypertension affecting pregnancy, and in pre-eclamptic toxemia. In the former condition, thromboses and fibrin deposition in the overlying decidua were associated with local intervillosus fibrin deposition and infarct formation, and with premature ageing of the villous blood vessels and stroma, such that subtrophoblastic sinusoid formation was limited. In pre-eclamptic toxemia, syncitial atrophy was accompanied by a diminution in lipoid. It was considered that this was related to the diminution in pregnandiol excretion which occurs in pre-eclamptic toxemia.

J. Beverley (Sheffield). 'Stress and Pathogenicity of Toxoplasma.' Evidence was produced that most toxoplasma infection in animals and humans remains subclinical. Instances from the literature of clinical grade infections in various species were cited to illustrate that such factors as cold, concomitant infections, captivity, infancy, pregnancy, injury and endocrine imbalances may have contributed to the severity.

A strain of toxoplasma, isolated from a rabbit, was found to be only feebly pathogenic for non-pregnant mice, causing them to lose weight from the ninth to the 15th days after inoculation, but they completely recovered. When the same strain, in the same doses, was injected into other mice so that the peak of the illness occurred at different stages of pregnancy it was found that injection 11 to three days before pregnancy caused abortion with 17% of mothers dying; injection during the first 12 days of pregnancy caused abortion or full term still-births, or live births which soon died, and the maternal mortality was 55%; injection between the 13th and 16th day resulted in live litters which were not reared and no maternal mortality; and inoculation between the second and 12th day of lactation rarely caused neglect of the litters but a 92% maternal mortality around the time of weaning.

J. H. Allan (Sheffield). 'The Mineralization of Enamel in Developing Teeth.' Ground sections of human deciduous teeth, ranging in development from 26 weeks in utero to 18 months after birth, were examined in plane polarized light, and the following pattern of mineral accretion deduced.

1. Following the commencement of enamel organic matrix formation, a primary, fairly diffuse deposition of mineral matter occurs along a narrow zone at the incisal amelo-dental junction. From this latter area, the amount of mineral matter falls in a regular manner towards the periphery of the enamel.

2. In the peripheral enamel a secondary deposit then occurs, producing slightly better mineralized zones in the cervical region and just deep to the enamel surface. This secondary deposit results in an area of slightly lower mineral content being present in the centre of both labial and lingual enamel.

3. A final deposition then starts on the lingual side of the incisal amelo-dental junction and expands cervically and peripherally, gradually obliterating the primary and secondary deposition patterns. This final deposition, which initially advances parallel to the organic matrix incremental lines, gradually alters its direction until it is almost parallel to the enamel surface.

These results support the view that all the main phases of enamel mineralization proceed peripherally from the amelo-dental junction, not cervically from the incisal surface.

J. L. Emery, G. H. Jowett and Morag S. Macdonald (Sheffield). 'Post Natal Changes in the Glomeruli of the Kidney.' Kidneys from 235 children aged 26 weeks in utero to 13½ years were described.

The glomeruli were classified according to six stages of maturity. The percentages of these six stages were related to age.

A reproducibility survey showed that there had been a subconscious change in standards of classification during the period of the survey, but the following conclusions appear valid.

1. Primitive forms may persist until 4 to 6 months of age, but some of these may be abnormal.

2. The majority of glomeruli are of mature type from 6 years onwards, but it is not until after 12 years of age that all are of adult form.

3. A very wide individual variation in the rate of glomerular maturation was found.

In addition, various case reports were given.