to differentiate this enzyme deficiency from debranching enzyme deficiency. The amount of collagen present in the liver tissue varied considerably in these two disorders. In cases of debranching enzyme deficiency, collagen fibres grossly disturbed the liver architecture. The degree of fibrosis varied considerably in the liver in phosphorylase deficiency. By light microscopy the presence of collagen fibres was not impressive in glucose-6-phosphatase deficiency, though by electron microscopy collagen was shown in the spaces of Disse.

Localized obliteration of bile ducts. H. B. Marsden. Department of Pathology, Royal Manchester Children's Hospital, Pendlebury, Manchester M13 0JH.

Limitation of growth potential of kidney by injury in early life. C. L. Berry, G. W. Slocombe, and P. R. Freeman. Department of Morbid Anatomy, Guy's Hospital Medical School, St. Thomas's Street, London SE1.

There is evidence to suggest that ureteric reflux and urinary tract infections in early childhood may affect the growth potential of the kidney. Since tubular growth is mainly a postnatal event in man, it is possible that permanent damage may result from a depletion of the tubular cell pool. This hypothesis was examined in the developing rat kidney, whose growth is similar to that of man. Tubular cell numbers were reduced using mercuric chloride. The effect of unilateral nephrectomy was also examined. DNA and RNA estimations were used to assess renal growth in terms of cell numbers, mean cell size, and RNA/DNA ratio. It was found that the kidney was capable of repairing extensive necrosis within 10 days. Kidneys stressed by unilateral nephrectomy showed evidence of hyperplasia and hypertrophy. Unilateral nephrectomy in addition to mercuric chloride was followed by a hypertrophic response alone and cell numbers failed to increase. These findings suggest that while the developing kidney can repair tubular injury, a sufficiently gross depletion of tubular cells during a period of rapid growth may result in a permanent reduction in kidney size.

Primary causes of neonatal mortality: prospective study of 100 cases. V. V. Joshi. Department of Pathology, Medical College of Virginia, Virginia Commonwealth University, Richmond, Virginia.

Many of the previous reports on causes of neonatal mortality have the following faults. They are retrospective studies, they mix primary and secondary causes of death, they do not breakdown causes of extrinsic perinatal hypoxia (EPH), and such important causes as intrauterine growth retardation and neonatal necrotizing enterocolitis are excluded. The list of primary causes of death used by the Perinatal Mortality Committee of Quebec Province, Canada, was modified to avoid the above criticisms. The methodology and results of 100 consecutive necropsies, excluding macerated stillbirths, are reported. Respiratory distress syndrome (RDS) 31%, congenital anomalies 28%, and EPH 17% were the three commonest causes of death. In the low birthweight infants (<2500 g) RDS was the most frequent cause (46%), and in full birthweight infants (>2500 g) congenital anomalies were the most frequent (57%). Maternal, placental, or labour and delivery problems were associated with about half the cases of EPH. In 2 cases the cause of death remained unexplained, though sudden infant death syndrome may be a possibility.

Postperinatal deaths and unexpected deaths in infancy: prospective study. J. L. Emery. The Children's Hospital, Sheffield.

Home unexpected deaths now constitute the largest group of postperinatal deaths. The state of babies at the time of birth, who had later been unexpected deaths, has been compared with control children and a scoring system devised in order to select children at increased risk of unexpected death (Protestos *et al.*, 1973).

A prospective study using this criteria has been carried out in Sheffield which suggests that the method is functioning, and also identifies children having a doubled risk of requiring hospitalization (Carpenter and Emery, 1974). The study will be continued for a further year before a final report is made.

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Fatty change in brains of perinatal and unexpected deaths. D. R. Gadson and J. L. Emery. Children's Hospital, Sheffield.

Earlier work showed that the presence of fat-laden cells in the CSF of children with hydrocephalus is an indicator of severe brain damage (Chester, Emery, and Penny, 1971; Chester, Penny, and Emery, 1971). The findings of such cells in CSF of some unexpected cot deaths and perinatal deaths stimulated a survey of the brains of such children to ascertain the likely site of origin. This was found to be in the region of the corpus callosum, the septum pelucidum, fornix, and related regions of the lateral ventricles. A survey carried out of 200 brains of children dying in the postperinatal period indicated that the fatty change is principally related to two groups of children—early perinatal deaths associated with severe hypoxia and a number of the unexplained, unexpected deaths in infancy. Evidence was presented which indicated that this fatty change is pathological and suggests that it is a reaction to hypoxia in actively myelinating brain tissue.

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