automatically generated, and certain statistics which monitor the study to be recovered. Some 12,000 names have so far been filed.

Two cases of neuroblastoma with marrow findings mimicking acute leukaemia. T. E. Parry. Department of Pathology, Llandough Hospital, Penarth, Glamorgan CF6 1XX.

Two cases of neuroblastoma with multiple bone metastases in which marrow biopsy on first hospital admission presented findings indistinguishable from those of acute leukaemia occurred in a boy of 7 and a girl of 3 years, in both of whom the correct diagnosis was established at necropsy after an illness of some weeks. The features which may enable the two conditions to be differentiated during life are the absence of malignant cells from the peripheral circulation and the characteristic clumping of cells in the marrow with the formation of syncytial masses and of pseudorosettes in neuroblastoma. Though well documented (Piney, Mallarmé, and Ross, 1950; Gaffuey, Hansman, and Fetterman, 1959), the difficulty this differential diagnosis may present receives little attention in the standard textbooks of haematology.

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Diagnosis of immune deficiency states. J. Huber. Department of Pathology, Hospital for Sick Children, 555 University Avenue, Toronto 101, Ontario, Canada.

Significance of 'focal sclerosis' in lipoid nephrosis. J. D. Elema. Department of Pathology, University of Groningen, Holland.

Renal focal sclerosis is characterized by the presence in some glomeruli of adhesions of Bowman's capsule with thickening of the mesangial matrix and capillary eosinophilic sclerosis in the absence of a glomerular proliferative lesion. Immunofluorescence shows the presence of IgM and C3, and the increase in mesangial matrix and deposits of heterogenous material has been confirmed by electron microscopy. There is a definite relation between the histopathology and proteinuria in the nephrotic syndrome, though in some patients increased protein excretion has been shown to precede the development of focal sclerosis.

A comparison may be drawn with the focal glomerulosclerosis sometimes observed in aging rats. The light and electronmicroscopical features and immunohistology are similar to human focal sclerosis and these changes may also be shown to be preceded by increased protein excretion. It is suggested that focal sclerosis in nephrotic patients resistant to therapy is the result rather than the cause of persistent proteinuria.

Interstitial pulmonary emphysema in the newborn. J. F. Saltet. Morbid Anatomy Laboratory, St. Joseph-Ziekenhuis, Graaf Florisweg 77, Gouda, Holland.

Without measuring the intrathoracic air pressure by means of a manometer, followed by embedding the suspected lung tissue in nitrocellulose, many cases of ectopic air in the thorax will escape detection. This is unfortunate because the finding of air 'in the wrong place' can help to establish the cause of death. In the present study, interstitial lung emphysema occurred in 7% of all the babies who died in the first week of life, and it is concluded that this precedes most of the cases of mediastinal emphysema and pneumothorax seen in the newborn.

A sudden rise in intrathoracic pressure applies a shearing force to the alveolar margins in contact with the periarterial sheaths which may then rupture, allowing alveolar air to enter the interstitium.

Predisposing diseases in the newborn were hyaline membrane disease, intracranial haemorrhage, renal dysgenesis, and adrenal hypoplasia.

Forced respiration in an attempt to recover from anoxia is thought to be the commonest cause, but in this study of 100 cases of interstitial pulmonary emphysema in the newborn, mouth-to-mouth respiration as a cause of the lung pneumatosis was seen in only 4%, which is much lower than expected.

Necrosis of vocal cords in infants. G. J. Cullity and J. L. Emery. Department of Pathology, Children's Hospital, Western Bank, Sheffield S10 2TH.

Vocal cord lesions in infants have been reported at necropsy in association with sudden unexpected death (SUD) by 4 different groups of observers in the last 20 years. The lesions are generally described as deep-seated foci of necrosis or inflammation in the true vocal cord. Some have described them in up to 90% of cases of unexplained SUD, and most believe them to be pathognomonic. The aetiology and pathogenesis are unknown. Similar ulcerated lesions were described in 1962 by Osborn and Flett in newborn infants dying with respiratory distress syndrome and believed by them to result from vocal cord spasm. We have studied the vocal cords in an unselected group of 117 infants and children, comprising 55 cases of SUD by history, 55 hospital deaths in infants and children, and 7 stillbirths. The lesions seen have been classified histologically into 7 groups. When allowance is made for the effects of intubation, the same pattern of lesions is present in all but the stillbirth group. This argues against a specific pathogenetic mechanism confined to the cot death situation. The lesions certainly occur before death, and suggest the existence of a laryngeal disorder which merits further study.

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