and frequent desmosomes. The good prognosis of these tumours tends to support a diagnosis of haemangioendothelioma rather than Ewing's sarcoma.

Necropsy findings in a case of Lesch-Nyhan syndrome. V. Mahnovski, S. Dozic, D. Vulovic, B. Marjanovic, and G. Tasic. Institute of Mothers and Child Health of SR Serbia, 8, Radoja Dakica, 11070 Novi Beograd, Yugoslavia.

A case of self-mutilation in an infant was first apparent at the end of the second year of age and resulted in death at 3 years 8 months. During life he was physically and mentally retarded with polydypsia, polyuria, choreoathetosis, and spasticity of the legs. EEG showed diffuse moderate cortical damage. Serum uric acid was >30 mg/100 ml and urinary uric acid was 50–130 mg/kg per 24 h. Renal function was impaired, but no calculi or haematuria was observed. At necropsy there were numerous excoriations and scars in the perioral and infraorbital regions and over both hands. The fingers and toes were plump and short and of similar size. The brain weight was slightly reduced (960 g, normal 1154 g) and there were moderate focal degenerative changes. The kidneys showed interstitial nephritis and focal glomerulosclerosis. There were numerous granulomata containing uric acid crystals, predominantly in the medulla. These granulomata were not seen in other organs though they have been described in the liver, spleen, and bone marrow. Additional findings were focal degenerative changes in some skeletal muscles, chronic enteritis with hyperplasia of mucosecretory glands, prominent pancreatic periductular fibrosis, and lamellation of the adventitial tissue of the periadrenal blood vessels. There was also focal chronic pneumonitis and diffuse chronic bronchitis.

Oedema of umbilical cord and respiratory distress in the newborn. J. M. Scott and J. B. S. Coulter. Departments of Pathology and Neonatal Paediatrics, Glasgow Royal Maternity Hospital, Rottenrow, Glasgow G4 0NA.

Oedema of the umbilical cord (defined as visible oedema in a cord with a minimal cross-sectional area of 1·3 cm) was found in 11·5% of deliveries. It was seen more frequently in certain complications of pregnancy such as abruptio placentae, maternal diabetes, macerated intrauterine death, and conditions such as prematurity, Rhesus isoimmunization, respiratory distress syndrome, and transient respiratory distress. There was a higher incidence in infants delivered by caesarean section. There was no association between cord oedema and either fetal distress or neonatal asphyxia, nor any correlation with maternal hyper tension or oedema.

Some factors involved in the production of oedema included low osmotic pressure, raised hydrostatic pressure in the placenta and umbilical cord, or an increase in total water in the fetoplacental unit. It is suggested that oedema of the cord may reflect similar changes in the lungs which prenatally predispose an infant, whose pathway for production of surfactant is immature, to develop respiratory distress syndrome, and the mature infant to develop transient respiratory distress.

Pathogenetic implications of the lesion complex of hyaline membrane disease. D. R. Shanklin. Laboratory of Pathology, Chicago Lying-in Hospital, Chicago, Illinois 60637.

The elements of the lesion can be divided into principal and ancillary. The principal elements are (a) partial collapse with centrolobular air space distension ('air bronchogram'); (b) vascular congestion, especially in capillaries and venules; (c) pulmonary oedema and lymphatic dilatation; (d) membranes. Ancillary elements include (e) necrosis of bronchiolar epithelium, especially in early cases and in very small fetuses; (f) focal haemorrhage, both interstitial and in air spaces; (g) polymorphonuclear leucocytosis, especially at about 20 to 30 hours; (h) later macrophagic response; (i) swelling of interstitium with possible increased cellularity and increased matrix.

These changes represent phases in a classic form of injury, accomodation, and repair. The stability of the lung and alterations of permeability which are so striking provide evidence for disruption of the expected mechanisms for integration of perfusion-ventilation interaction. Disturbance of the ventilatory action could arise either from initiatory or from regulatory phenomena. Changes in permeability must mean profound injury to the usual vascular defenses, and the full range of factors that have to do with that integrity. These points suggest either a multifactorial aetiology and pathogenesis, or a sufficiently diverse agent or event to promote a wide range of physicochemical and physiological changes. The promptness of onset of clinically observable disease and the occasional severity of lesions in short-lived infants speak for the importance of events surrounding the onset of breathing, and the lack of development of defences in the prematurely born.

Wilson-Mikity syndrome. O. Braun. Niederosterreichisches Landeskrankenhaus, Pathologisches Institut, A-2340 Modling bei Wien, Austria.

A preterm infant with a birthweight of 1350 g and length 38 cm lived for 80 days, in spite of progressively increasing respiratory distress and cyanosis. Oxygen was given from birth but could not control hypoxia and acidosis. Radiologically, increased striation in the perihilar fields was observed and at the age of 5 weeks the *x*-ray picture was typical of Wilson-Mikity syndrome. At necropsy the lungs were firm and dark red, with numerous small emphysematous bullae in the subpleural tissue. There was no histological evidence of infection either in the present case or in published reports. Special emphasis was given to the changes found in the fibrous scaffolding of the lungs. Collagenous fibres were increased, especially in the thickened septa and alveolar walls. Elastic fibres were irregularly distributed and in some areas at least were split, rolled up, and protruding into the alveolar lumen. Argyrophil fibres were similarly fragmented. Conversion of