changes were greater in the mature brain but this appeared to be due to difficulties of recognizing degeneration in the immature cerebellum rather than to any correlation with brain swelling. Necropsy delay was also related to an increased incidence of Purkinje cell degeneration.

Brain water content showed an inverse relation to maturity and brain swelling: it was highest in a case of overhydration and possibly in hydropic fetuses, but lowest in cases with herniation phenomena and small-for-dates babies. No consistent relation was recognized between high brain water levels and histological evidence of oedema. Gyral flattening was, however, associated with an unexplained vacuolated inner cerebral cortical picture, but such changes were related to a wide range of brain water content.

The main histological feature related to maturity and low water levels appeared to be vascular congestion. Dehydration of cord blood specimens showed that fetal blood contained twice the solid content of whole brain. It is suggested that perinatal brain swelling is basically due to increased blood volume. Vascular congestion is a marked feature of an asphyxial fetal death and this may be part of a vicious circle involving hypoxia, acidosis, vascular dilatation, and brain swelling with raised intracranial pressure. This may be lethal or, in survivors, may play a part in the development of the early stages of cerebral birth injury.

**Thymoma associated with leukaemoid infiltrates in the brain.** J. J. Puitinen. Koupio 8 D 11, Finland.

**Familial lymphohistiocytosis—20 cases in three family groups.** A. J. Barson. University Department of Pathology, Williamson Building, Brunswick Street, Manchester 13.

**Effect of tracheal deposition of surfactant on air expansion of lungs—study on premature rabbit fetuses.** G. Enhorning, Gertie Grossman, and B. Robertson. Department of Paediatric Pathology, Karolinska Sjukhuset, Stockholm 60, Sweden.

Premature rabbit fetuses (gestational age 28 days) were tracheostomized immediately after delivery, the respiratory movements of the fetuses being prevented by compression of the thorax during operation. Consequently, the fetuses had to take their first breath through a tracheal cannula, which in 20 experimental animals contained 50 μl of a concentrated suspension of pulmonary surfactant, prepared by centrifugation of alveolar wash from adult rabbit for 1 hour at 1000 × g and 4 °C. In 20 control fetuses, the tracheal tubing contained an equal amount of saline, or it was empty. 13 of the surfactant-treated fetuses survived the operation by 3 to 40 hours, whereas all but one of the control fetuses died within 45 minutes after tracheostomy (P < 0.001). The unopened thorax of the fetuses was fixed by immersion in formalin, and the air expansion of the lungs was evaluated histologically. Alveolar air expansion, varying in degree from slight to prominent, was apparent in all surfactant-treated fetuses. Among controls, slight alveolar air expansion was observed in 3 cases, whereas in 17 fetuses the lungs were unexpanded. Our findings thus suggest that tracheal deposition of surfactant increases the survival time of the fetuses by enhancing the air expansion of the lungs. Possibly a modification of this treatment might be adopted as a prophylactic measure against neonatal respiratory distress due to prematurity.

**Relation of bronchopulmonary dysplasia to oxygen and ventilator therapy in the newborn.** J. S. Wigglesworth. Nuffield Neonatal Research Unit, Institute of Child Health, Hammersmith Hospital, Du Cane Road, London W.12.

**Scanning electron microscopy of human placenta.** B. Ivemark and B. Sandstedt. Department of Paediatric Pathology, Karolinska Sjukhuset, Stockholm 60, Sweden.

**Intrathoracic tracheal collapsibility—a cineradiographical study.** I. Claesson. Department of Radiology, The Children's Hospital, Barnsjukhuset, Göteborg, Sweden.

The trachea in infants with respiratory distress and stridor, especially during prolonged cannulation, was examined by biplane cineradiography. The examination was performed without any contrast medium in the trachea. In some cases intrapleural and intratracheal pressure was recorded together with respiratory flow and lung volume. When no pressure measurements were made, thoracic impedance was registered so that each single frame could be timed to the respiratory phase.

In cases with increased mobility of the tracheal walls there was a marked increase of inspiratory widening and expiratory narrowing of the intrathoracic trachea or part of it. The tracheal collapse was considerably more marked in the lateral than in the anteroposterior projection, indicating the major role of the membranous part of the trachea. Causes of an increased weakness of the tracheal wall and the significance of the tracheal cross-sectional shape in increased collapsibility were discussed.

**Intrathoracic tracheal collapse—a pathophysiological study.** O. Hjalmarson. Department of Paediatrics, The Children's Hospital, Barnsjukhuset, Göteborg, Sweden.

It can be shown that pronounced expiratory narrowing or collapse of the intrathoracic trachea can be seen as a normal phenomenon at very high flow rates or in pathological conditions in forced expiration if the static lung pressure or the stability of the tracheal wall is reduced, or if the resistance 'upstream' to the collapsing site is increased.

Eight infants, 2 to 25 months of age, all with a history of tracheal cannulation of long duration, and on clinical grounds believed to have tracheomalacia, were examined with simultaneous cineradiography of the trachea and measurements of tracheal and oesophageal pressures, tidal flows, and volumes. ‘Upstream’ resistance, lung