of lungs obtained at necropsy from 44 children at or above the 50th centile dying from non-pulmonary causes were described. In the present paper some results from the analysis of 92 further pressure-volume curves are described.

These were compared with the theoretically normal values according to total body length, by dividing the observed value by the theoretically normal one. This was done for both the maximum inflation volume and the low pressure air retention index. These indices were then plotted against each other to look for 'clusters'.

One cluster showed low maximum volume, but, in contrast to the IRDS group, an above normal low pressure air retention index. The deflation curve characteristics of this group closely resembled the normal curves of premature infants rather than those of the growth-matched controls.

Histological examination of these lungs fixed distended at a pressure of + 5 cm. H₂O showed only alveolar ducts and collapsed alveoli, whereas the controls showed alveolar ducts lined with open alveoli. Stains for elastic tissues showed that the alveoli were normally developed, but collapsed against the duct walls. At low pressures, alveolar morphology is almost certainly controlled by surfactant.

From these findings, it is suggested that, (1) the finding in an older child's lung of a deflation curve profile similar to that of a premature lung but with a severely diminished maximum inflation volume is characteristic of alveolar closure with surfactant malfunction in a more mature lung; (2) the reason for the different deflation profiles of surfactant malfunction in the newborn and the older child is that in the older child the elastic tissues around the alveolar ducts are sufficiently rigid to hold them open against the increased surface tension (due to surfactant malfunction); and (3) the similarity of deflation curve profile between the premature lung and abnormal older child's lung is because both are respiring with simple alveolar ducts without functioning definitive alveoli.

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Pulmonary Ultrastructure in Neonatal Hyaline Membrane Disease. J. M. LAUWERYS and N. BOURGEOIS (University of Louvain). Pulmonary tissues of 8 babies (gestational age: 29–34 weeks) dying of hyaline membrane disease were studied with the electron microscope (buffered osmium tetroxide fixation, epon embedding) and the results correlated with the findings by light microscopy.

Electron microscopy revealed the membranes to be essentially composed of degenerated epithelial cells and transudate of blood. Keratinized cells of amniotic fluid origin were frequently enmeshed within the membranes. Fibrin with its characteristic periodicity, however, was exceptional.

Lamellar bodies were repeatedly present in the granular pneumocytes. Quantitative studies should be undertaken to give more reliable information on their exact

incidence. The relation of these bodies to surfactant remains an open question, as they were observed infrequently in fetuses of 20 weeks' gestational age.

Concurrently a detailed analysis has been made of the ultrastructure of the terminal air sacs of these prematurely born babies. This indicates that though the inter-saccular septum of the premature lung is much broader than the interalveolar septum of the adult lung, the diffusion pathway for the blood gases, the so-called 'alveolo-capillary barrier', is identical in both as regards its ultrastructure.

An attempt has been made to integrate these findings with some earlier studies by light microscopy and by injection. It is concluded that in hyaline membrane disease hypoperfusion and hypoventilation (rather than a diffusion disturbance) seem to be of paramount importance. (Investigation supported by WHO R/00176 and FWGO-901 grants.)

Congenital Cystic Malformation of the Lung: its Relation to Hydrops and Hydramnios. H. C. KOHLER (Maternity Hospital, Leeds). According to severity, infants with congenital cystic malformation of the lung fall into 2 groups: those that are either stillborn or die immediately after delivery and those that survive and can be operated upon. The latter group is, of course, clinically more important but the former is also worth studying.

In the observed case pregnancy had been complicated by hydramnios; labour was premature; the infant was born hydropic and moribund, expiring within 1 hour. At necropsy a cystic hamartoma of the left lower lobe was seen to occupy most of the thoracic cavity, compressing the remaining normally formed lung tissue and displacing heart and great vessels.

Hydrops and hydramnios have a significantly high incidence in published case reports, but there is good reason to believe that hydramnios has been under-reported.

The explanation put forward by some authors that both hydramnios and hydrops are due to circulatory failure from mechanical interference, i.e. pressure in the great veins, is unattractive and lacks evidence, but there is no alternative explanation of the pathogenesis of hydrops. Hydramnios, however, can be attributed to failure of pulmonary absorption of liquor. There is strong evidence that the lungs under normal circumstances play a significant role in the disposal of amniotic fluid, but neither the abnormal lining epithelium of the cystic lesion nor the badly compressed 'normal' lung tissue would be able to absorb liquor.

Necropsy Findings in Six Cases of *Pneumocystis carinii* Pneumonia. J. VLACHOS (Athens). The first 6 cases of *Pneumocystis carinii* pneumonia, verified at necropsy in Greece, are presented as a contribution to the geographic pathology of the disease. They comprise 1.4% of a series of 420 paediatric necropsies performed during the past 4 years (1964–1968) in the Department of Pathology of Athens University. Their ages ranged up to 6.5 months. 3 cases had a well-

defined primary deficit of their immunological mechanisms, the other 3 cases were markedly dystrophic infants subjected to protracted chemotherapy. These last 3 cases may also have had an underlying but ill-defined impaired resistance; alternatively, however, the pneumocystis carinii pneumonia could have been the primary disease, which initiated a vicious circle in which both pathological processes were involved.

The main pathology of pneumocystis carinii infection and the parasites were limited to the lungs in all 6 cases, despite the generalized character of the underlying condition which predisposed to the disease.

Neonatal Meningitis and Pneumonia due to Lancefield Group B Streptococci. K. B. ROGERS (The Children's Hospital, Birmingham). 4 newborn infants with pneumonia and 3 with meningitis were all infected with Lancefield Group B streptococci. Three of the infants with pneumonia were stillborn and the fourth only lived 18 hours: all four showed histological evidence of pneumonia in the lungs obtained at necropsy. Lancefield Group B streptococci were grown from vaginal swabs of all the mothers.

One of the 3 infants with meningitis was admitted at the age of 1 day, and a vaginal swab from this baby's mother grew Lancefield Group B streptococci: the other 2 were admitted at the ages of 10 and 11 days. All 3 only survived for a day after the onset of meningitis. Group B streptococci were isolated from the umbilicus of 2 of the 3 babies. Necropsy showed that 2 of the 3 babies had pneumonia as well as meningitis.

Group B streptococci were grown from 7 other babies; 2 were stillborn and the other 5 died a few days after birth. The streptococci were isolated from the throat of a 1-day-old infant on admission to hospital, from the lungs of 5 babies at necropsy, and from a cut-down wound in the seventh baby. Vaginal swabs from 3 of these babies' mothers grew Group B streptococci.

It is suggested that the Lancefield Group B streptococci were commensals in the mother's vagina, and that the infants were infected from this source: if vaginal swabs had been taken during pregnancy the streptococci could have been eliminated before the onset of labour. So many of these babies had been infected during or before labour that swabs taken when the mother was admitted for delivery would diagnose vaginal carriage too late for effective chemotherapy.

Birth Trauma and the Cervical Spine. E. LYNN JONES (Birmingham). The findings of Yates (1959) that distortional trauma to the cervical spine can occur at birth, and result in damage to the cervical portions of the vertebral artery was investigated further in a series of perinatal deaths.

The cases were selected during 1967 from 78 stillbirths and 114 neonatal deaths amongst a total of 320 necropsies performed at the Birmingham Children's Hospital.

The intact cervical spine was removed from 30 fetuses selected at random, fixed in 4% formaldehyde

in saline, and decalcified in 25% formic acid. The specimens were divided in horizontal planes and double-embedded in paraffin-wax and 2% celoidin. 5 μ thick sections cut on a sledge microtome were stained by a wide variety of methods.

Evidence of distortional trauma to the cervical spine was seen in 25 cases. The lesions were considered under 4 main groups: (1) extradural, dural, subdural, and subarachnoid haemorrhage; (2) tears and haemorrhages in nerve roots and spinal ganglia; (3) in 19 cases evidence of haemorrhage around one or both of the vertebral arteries was seen either in the form of a crescentic adventitial haematoma or massive haemorrhage encircling the vessel; (4) spinal cord lesions: these were seen in 2 cases, a precipitate and a breech delivery, and consisted of contusion in one case and bilateral necrosis of the lateral columns in the other.

The vertebral artery haemorrhages found in 19 fetuses may be important causes of perinatal mortality and morbidity. Many recorded cases of cerebral palsy could be explained on the basis of vertebral artery trauma and ischaemic cerebral damage at birth.

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Intrauterine Causes of Neonatal Asphyxia. K. F. KLOOS (Berlin). The gross and histological study of the placenta is essential not only in the investigation of stillbirths, but also of neonatal deaths (and infantile disorders). Disturbance of implantation generally leads to abnormal shape of the placenta or to abnormal nidation: this can occasionally be the cause of fetal death and/or of maternal complications.

Of greater importance—and so far very little appreciated—are disorders of maturation, affecting the placental vascularity and hence the exchange of gases and metabolites. The fetus is especially vulnerable during labour and even a relatively mild degree of hypoxia can lead to damage to the central nervous system, depression of regulatory function, and failure of spontaneous respiration on delivery. This in turn can start a vicious circle ending in metabolic acidosis.

Collapse of the Trachea—a Possible Cause of Asphyxia and Death? S. RANSTROM (Gothenberg). It seems probable that a preparatory dilatation of the trachea with air precedes the start of respiration after delivery, and that collapse of the trachea may be an obstacle to this. Such a collapse may be caused by foreign material in the laryngeal aditus, or by anomalies of the trachea such as hypoplasia with defective thickness or elasticity of the cartilage, a broad membranous part of the tracheal wall, or doubling of the posterior wall, the edges of the cartilage bows lapping over each other. Collapse of the trachea in the newborn may be maintained by respiratory effort, the walls of the trachea being held together more tightly the greater the strength of the inspiratory movement. During expiration there is no material in the respiratory tract capable of dilating the trachea.