

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.

REFERENCES

- Fagan, D. G. (1968). Lung compliance in the postnatal period. *Archives of Disease in Childhood*, **43**, 498.

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 in fetus of 20 weeks' gestational age.

Concurrently a detailed analysis has been made of the ultrastructure of the terminal air sacs of these premature 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. 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 underreported.

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-

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