Paediatric Pathology Society

Proceedings of the Fourteenth Annual Meeting

The Fourteenth Annual Meeting was held, by the kind invitation at the Sophia Kinderziekenhuis in Rotterdam on Thursday, 24 October, Friday, 25 October, and Saturday, 26 October. This was the first occasion on which the Society had met on the Continent. The President of the meeting was Dr. C. B. F. Daamen, and the chair at the scientific sessions was taken by Professors H. K. A. Visser and J. M. Lauweryns, and Drs. J. Huber, H. Kohler, and W. Aherne.

On Thursday, Dr. Daamen and Dr. and Mrs. Huber welcomed members and guests at a reception at the Sophia Kinderziekenhuis, and on Friday Professors H. E. Schornagel and J. M. de Vries held a sherry party at the Academisch Ziekenhuis Dijkzigt. On Friday evening the annual dinner held at the Atlanta Hotel. The social events concluded with a tour of Rotterdam on Saturday afternoon.

Twenty-eight papers were given, and there were 13 demonstrations. Some 40 members and 50 guests attended the scientific programme.

The next meeting of the Society will be held in Newcastle on Tyne on 24 and 25 October 1969, under the presidency of Dr. W. Aherne.

Scientific Communications

New Alveoli—Where and How? J. L. EMERY and D. G. FAGAN (The Children's Hospital, Sheffield). The number of alveoli in the adult lung is approximately six times that present in the lungs of newborn infants, and the method by which alveoli are formed during childhood is still not certain.

During postnatal life, there is an increase in the number of alveoli between the terminal respiratory tubules and the periphery of the terminal respiratory unit and also a diminution in the total number of fully epithelialized respiratory passages.

While studying the elastic tissue in a series of lungs that had been inflated with saline before fixation, the formation of consolidated masses of elastic tissue around the mouths of alveoli suggested that new alveoli were formed in two ways. Within alveolar walls, the elastic tissue becomes consolidated in such a way that parts of alveolar walls are increasingly held away from the air sacs, and as the lung expands the alveoli become subdivided. There is thus, after birth, a progressive development of a 'fishnet' type of elastic structure, the apertures of which form the mouths of alveoli. The second method consists of a breaking up of the muscle and elastic tissue of the terminal bronchioles with fenestration of the walls by new alveoli.

These processes are described under the headings of: (1) peripheral alveolar segmentation; (2) septal compoundment; (3) fragmentation of terminal respiratory passages.

Hyaline Membrane and Lung Stability in Babies with Respiratory Distress. J. S. WIGGLESWORTH (Hammersmith Hospital, London). (To be published.)

Changes in Pulmonary Structure in Neonatal Hyaline Membrane Disease Treated with High-pressure Artificial Respiration. M. J. BECKER-BLOEMKOLK (University of Amsterdam). 8 babies with hyaline membrane disease were treated with high pressure artificial respiration. At present 3 children are in good health. 5 babies died, respectively 3-5, 3-5, 6, and 6 days, and 5-5 weeks after the start of treatment. In these children high pressures (averaging 50 cm. H₂O) were used and high O₂ values (up to 100%) were reached.

Severe pulmonary changes were found. The lungs were heavy and non-aerated. There was emphysema only in the child treated for 5-5 weeks. Bronchi and bronchioli showed a marked epithelial hyperplasia as well as squamous cell metaplasia, whereas at other sites epithelial necrosis was apparent. The muscular layer was hypertrophic and mucous glands appeared hyperplastic.

Hyaline membranes containing bilirubin pigment were found in 4 of the 5 cases. In the child treated for 5-5 weeks no hyaline membranes were detected. The alveolar epithelium was extremely atypical. The interstitium showed a proliferation of fibroblasts which resulted in a pronounced interstitial fibrosis in the child treated for 5-5 weeks.

It is suggested that these changes are primarily provoked by the high pressures used.

Pulmonary Inflation: A Correlation between Histological Appearances and Abnormal Pressure-volume Curves. D. G. FAGAN and J. L. EMERY (The Children's Hospital, Sheffield). In a previous communication to this Society (Fagan, 1968), the growth-related changes in pressure-volume characteristics.
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**


**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 the fetus of 20 weeks’ gestational age.

Concurrently a detailed analysis has been made of the ultrastructure of the terminal air sacs of these premature babies. This indicates that though the interalveolar 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. G. 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 higher 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 on 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