Paediatric Pathology Society
Proceedings of the Fourteenth Annual Meeting

The Fourteenth Annual Meeting was held, by the kind invitation of Professor H. K. A. Visser, 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 was 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) septic compound; (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 bronchioles 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

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