morphological expression. We have, therefore, an interesting model of injury, the same for man and animals, in which an early lesion in an organelle (ribosome) is associated with a later lesion in tissue (metaplasia) and finally with disruption of organ function (emphysema). The model also suggests a reconsideration of the principles which guide the administration of high tensions of oxygen to newborn infants.

Morphology of Late Stages of Hyaline Membrane Disease. S. Ranström (*Sahlgrenska Hospital, Gothenberg, Sweden*). Beginning about the 5th day there is a proliferation of fibroblasts in the alveolar septa, but with rather scanty formation of collagen fibres. At the same time alveoli begin to disappear, with persistence of at least some of the capillaries. The hyaline material is gradually resorbed, partly in the form of fragmentation, partly as carnification, with formation of new capillaries in the lung tissue. The end result is a severe structural change with disappearance of alveoli, leaving only the alveolar ducts open and separated by broad septa of connective tissue rich in capillaries and poor in collagen. As the bulk of the lung capillaries are no longer in direct contact with the epithelium, a great deal of blood passing through the lung vessels is shunted past the much diminished gas-exchanging surface. These changes in the late stages of the respiratory distress syndrome seem too severe to make normal restoration of lung likely.

Perinatal Pathology of Costochondral Junction in Relation to Pulmonary Hyaline Membranes. B. I. Ivemark and B. Robertson (*Department of Paediatric Pathology, Karolinska Institute, Stockholm, Sweden*). Histological examination of the costochondral junction in a consecutive series (Robertson and Ivemark, 1969) of 100 neonatal necropsy cases showed a statistically significant correlation ($p < 0.001$) between a certain type of growth disturbance, characterized by rarefaction of the trabecular pattern in the metaphyses, and the presence of pulmonary hyaline membranes. In the same series, cases with trabecular rarefaction also showed a high incidence of abnormal dental development, with proliferation of the outer enamel epithelium and cord-like persistence of the dental ledge. The cause of this correlation is not clear. Possibly, the trabecular rarefaction and the dental lesions represent a non-specific pattern of growth arrest,

secondary to the severe metabolic derangement occurring in fatal cases of the idiopathic respiratory distress syndrome. An alternative explanation of the association between these particular metaphysal, dental, and pulmonary lesions would be a hitherto unrecognized common aetiological mechanism, and this is being studied.

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Dilatation of the Renal Tract in Children with Neurospinal Dysraphism. M. Forbes and A. R. Wilcock (*The Children's Hospital, Sheffield*). Wilcock and Emery (1970) in a recent necropsy study showed the predominance of dilatation lesions of the renal tract in children with meningocele. The incidence of these deformities increased steeply with the age at death, starting at 6% in stillbirths and rising to 35% in children dying at 5 years.

The aetiology of dilated ureters in these children is probably multifactorial, but some workers have suggested that faulty pelvic autonomic innervation may be responsible.

The intrinsic innervation of normal and dilated ureters was studied by the acetylcholine esterase method and formalin-induced catecholamine fluorescent technique. The histological findings of this study were described (Forbes, Underwood, and Emery, 1969, 1970). No qualitative difference was found between normal and dilated ureters in either their adrenergic or cholinergic innervation.

It was concluded that abnormalities of intrinsic innervation did not appear to contribute significantly to dilatation of the ureters in children with meningocele.

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