Pediatrics

AIRWAY OBSTRUCTION Congenital airway obstruction may result from total extrinsic obstruction of a mainstem bronchus by a bronchogenic cyst or atresia of a bronchus. In either case, the obstructed lung tissue will appear as a large airless mass on the chest roentgenogram and at thoracotomy. The lung tissue is normal except for mild distention caused by fluid retention and, in the case of the bronchial atresia, lymphatic dilation. Bronchial obstruction in the newborn may manifest not as emphysema or atelectasis but as a pulmonary infiltrate. (*Griscom, N. T., and others: Fluid-filled Lung Due to Airway Obstruction in the Newborn, Pediatrics 43: 383 (March) 1969.*)

RESPIRATORY DISTRESS SYNDROME Histopathologic and surface tension studies were made of the lungs of 93 premature and mature infants dying of respiratory distress. Surface activity in the lungs was studied by three methods: 1) the volume/pressure relation; 2) the "bubbles test," in which, in the presence of active surface material, bubbles squeezed out from the cut surface of the lung retain their original size for more than 20 minutes; 3) dynamic measurement of the surface tension of lung extract with the Wilhelmy balance. In mature stillborn infants, results of all three tests appeared normal although the lung did not expand spontaneously at any time. Surface-active material must have been present, however, since the volume–pressure diagram showed a normal hysteresis. In premature (six months' gestation) stillborn infants, the tests were grossly abnormal in that extraordinarily high opening pressures were required to unfold the lungs, and minimal hysteresis was found in the ventilation/pressure diagram as well as on the Wilhelmy balance. The bubbles were few in number and less than a fifth the original size. Microscopically, the lungs were immature, the alveoli were lined with cuboid epithelium, and they retained a "glandular" structure. Hyaline membranes were not formed. In a two-kg infant who died of respiratory distress syndrome 12 hours after birth, high opening pressures were required to unfold the atelectatic lung. Once expanded, the collapse was rapid, with minimal hysteresis. The bubbles test showed minimal persistence, with no bubbles left after 20 minutes. Microscopically, there was the well-known picture of hyaline membrane disease, with atelectasis and wide homogenous bands lining the unfolded alveoli. Such membranes do not appear before two to 12 hours of respiratory activity. The first appearance of hyaline membrane material coincides with the change of the alveolar cells from cuboid epithelium to flattened cells. Lambs delivered prematurely by Cesarean section showed the anatomic and functional picture of hyaline membrane disease, with abnormally high surface tension being responsible for atelectasis and transudate. Logically, the treatment is positive-pressure breathing (PPB), provided circulatory depression does not supervene. It was shown in lambs that circulatory collapse could be prevented by wide bilateral thoracotomy, thereby permitting continuation of positive-pressure breathing long enough to allow formation of surfactant material, and thereby assuring survival of the animals. Formation of adequate amounts of surfactant material takes four to five days. If PPB breathing could be maintained in premature infants for this interval, they might survive. (*Benzer, H., Lempert, J., and Regele, H.: Surface Tension in the Lung and Hyaline Membrane Disease, Wien. Klin. Wschr. 81: 145 (Feb.) 1969.*)