RESPIRATORY DISTRESS IN THE NEWBORN

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R. K. CHANDRA: Respiratory distress in the newborn is a challenging problem. It makes a large contribution to neonatal morbidity and mortality, especially among the premature. While a host of pathological conditions may give rise to respiratory distress at this age, we are concerned chiefly with what has often been termed ‘idiopathic respiratory distress syndrome’. It is also known as ‘hyaline membrane disease’, and ‘pulmonary syndrome’. It may be defined as respiratory distress in newborn infants in whom specific causes of abnormal breathing, such as pulmonary haemorrhage, intrauterine pneumonia, pneumothorax, diaphragmatic hernia, congenital heart disease, intracranial pathology, have been excluded.

A typical case of the respiratory distress syndrome (R. D. S.) is characterized clinically by onset of symptoms within an hour or two of birth, rapid breathing, use of accessory muscles of respiration, indrawing of intercostal and subcostal spaces an an expiratory grunt. With progression of the disorder, the respiratory rate falls with apneic spells, periodic breathing and intense central cyanosis. The reflexes are lost and there may be signs of congestive cardiac failure. Auscultation reveals areas of reduced air entry with fine crepitations. Typically, X-ray of the chest shows a generalized fine mottling. The mortality rate is about 50 per cent. It is responsible for 15—25 per cent of all deaths during the first week of life.

We would discuss some important aspects of this well known clinical syndrome. Dr. Usha Oberoi would first tell us about applied aspects of neonatal pulmonary physiology.

USHA OBEROI: In order to obtain an insight into disturbances of pulmonary function in newborn infants, we should be aware of the physiological changes which occur in the lungs at birth. When the fetus is delivered, its lungs are functionally inert, the alveoli are filled with liquid and they are perfused by only 10 per cent of the cardiac output. For acquiring the function of gas exchange, the lungs undergo a number of changes.

Initiation of alveolar ventilation and formation of gas-fluid interface. Initiation and maintenance of adequate alveolar ventilation is an important sequential step in functional development of the lung. Distribution of air among the alveoli is as important as the amount of air inhaled. The formation

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of a gas-fluid interface enables the gas molecules to diffuse through the separating membranes.

It is well known that the first breath of a newborn infant requires higher pressure than the subsequent ones. A pressure of 18—20 cm. of water is required for the alveoli to open up. In a mature foetus, a stable alveolar volume (Functional Residual Capacity or F. R. C.) is formed after the first effort at inflation and normal ventilation can proceed thereafter. In the premature or ‘immature’ baby, however, the physical characters of the lung require abnormally large ventilatory pressure to open up the alveoli with each breath and the gaseous exchange takes place mainly during expiration.

The formation of a stable F. R. C. in the mature lung can be explained by properties of the surface film of fluid which lines the alveoli. It is lipoprotein in nature and has a very low surface tension, preventing the alveoli from collapsing due to pressure exerted by their walls. It is also called ‘surfactant’ or ‘anti-atelectasis factor’. Two forces act on the alveoli—the intra-thoracic pressure, and the pressure (P) within the alveolus produced by surface tension (T) of the lining liquid, tending to contract the alveolus and expel air. This is expressed in LaPlace formula as \( P = 2 \times \frac{T}{\text{radius}} \). If T were to remain constant, the smaller alveoli would tend to collapse into bigger ones, resulting in an unstable alveolar function. However, T varies according to the size of the alveolus, since it also depends upon the surface area.

In some infants, notably the premature, there is an absence or a reduced quantity of surfactant. A more recent view regards it as a developmental immaturity, which would correct itself given enough time. This deficiency of surfactant makes the lungs stiff and less compliant, and the muscles of respiration have to exert much more in order to produce enough negative intra-pleural pressure so as to overcome the elastic recoil of pulmonary tissues. This seems to be the key pathogenetic factor in production of R. D. S.

**Removal of liquid from the lungs.** Foetal lungs contain significant amount of clear fluid, most of which is squeezed out during the process of birth. The remaining fluid is absorbed through lymphatics. Lymphatic flow increases as soon as ventilation starts. Babies born after caesarian section have a relatively more quantity of liquid and it may predispose them to the development of R. D. S.

**Increase in pulmonary blood flow.** In the normal newborn, changes in partial pressure of \( p \text{CO}_2 \) and \( p \text{O}_2 \) produce a reduction in the pulmonary vascular resistance, through chemoreceptors. This results in increased blood flow through the lungs. In the face of chemical and physical stresses like hypoxemia, acidemia, hypovolemia and hypothermia, the pulmonary vessels undergo further vasoconstriction, excluding blood from the alveoli, the cells of which normally form the surfactant. This results in atelectasis and further anoxia and acidemia, which intensify the vaso-constriction. The latter increases the right to left shunt, and causes more anoxia. Thus a vicious circle is set up, which propagates itself.

**Pulmonary functions in R. D. S.** It is amazing that modern techniques are able to estimate various parameters of pulmonary function at this delicate