The First Year of Life

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BREATHING DIFFICULTIES IN THE NEWBORN

Respiratory distress syndrome

The respiratory distress syndrome is the commonest cause of respiratory problems in the newborn and a common cause of death in preterm infants. Although preterm babies are particularly susceptible, it also occurs in the obese infants of mothers with diabetes mellitus. Hypoxia before, during, or after birth is a predisposing factor.

The cause is a deficiency of pulmonary surfactant, a substance normally present on the alveolar walls. Surfactant lowers the surface tension in the alveoli so that during the first few breaths the same pressure is required to inflate them all. This produces uniform inflation of all alveoli. Surfactant also prevents the alveolar walls from collapsing during expiration. Without surfactant the surface tension is great in the smaller alveoli, causing them to collapse, while larger alveoli continue to expand easily. Thus there is uneven expansion, with increasingly widespread alveolar collapse. Surfactant production in the fetal lung may be poor before the 36th week of gestation and the level in the fetal lung correlates well with the lecithin:sphingomyelin ratio in the amniotic fluid.

The incidence of the respiratory distress syndrome is higher with decreasing gestation so that at 27-31 weeks 35-50% of all infants are affected. About three out of 1000 infants born alive die from the respiratory distress syndrome. Microscopy of postmortem specimens often shows the presence of amorphous material lining the terminal bronchioles and alveoli, which is called hyaline membrane. There are also multiple areas of alveolar collapse.

Clinical features and general management

An expiratory grunt, a respiratory rate over 60 per minute, and recession of the chest wall begin within three hours of birth. Central cyanosis may appear later. Usually auscultation of the lungs reveals only reduced breath sounds, but crepitations may be present. In most infants the chest radiograph shows normal lung fields in the early stages of the disease, but later a fine reticular pattern or generalised loss of translucency (the ground-glass appearance) may be present. The contrast between the air in the bronchi and the opaque lung fields produces an air bronchogram. The greatest value of the chest radiograph is in excluding other conditions (see below).

The object of management is to support the infant for a few days until further surfactant is produced. The infant should be handled as little as possible, as movement during frequent recording of rectal temperature or changing the sheet may make his condition deteriorate abruptly. The infant should be given oxygen, his blood oxygen concentrations should be monitored, and fluid and electrolyte balance maintained.

Oxygen treatment and positive airways pressure

Blood oxygen analyser

Catheter electrode

Transcutaneous electrode

Endotracheal tube

Face mask

Nasal prongs

During oxygen treatment the arterial oxygen tension should not fall below 6.7 kPa (50 mm Hg) or rise above 12 kPa (90 mm Hg). Below 6.7 kPa the risk of cerebral palsy and mental retardation increases, and above 12 kPa there is a possibility of retrolental fibroplasia and subsequent blindness. Some infants may need high concentrations of inhaled oxygen to prevent hypoxia. This can be given safely only if there are facilities for making frequent estimations of the blood oxygen concentrations.

There are several methods of measuring oxygen concentrations in the blood. Samples can be taken from an umbilical artery catheter at regular intervals or they can be obtained by repeated puncture of one of the radial arteries. An oxygen-sensitive electrode implanted into the tip of an umbilical artery catheter is used in many units. It measures oxygen levels continuously, but the electrode must be calibrated at regular intervals with samples of blood aspirated through the lumen of the same catheter. The transcutaneous oxygen electrode has recently been introduced. The heated electrode increases the blood flow to the skin, where the oxygen tension is measured. The electrode must be moved every four hours to avoid burns. The method is not invasive and the electrode can be calibrated with a gas mixture or special fluid.

Incubators are used to improve observation. There are two types—the conventional incubator and an open type. A perspex head box can be used to maintain an oxygen concentration above that in the air.

Continuous positive airways pressure maintains a low positive pressure in the alveoli during expiration thus preventing collapse. This positive pressure can be delivered to the infant by nasal prongs, a face mask, or an endotracheal tube. Some units have reported very low death rates for the respiratory distress syndrome when this treatment has been started at an early stage of the disease. Infants with repeated apnoeic attacks or evidence of increasing respiratory failure may require intermittent positive pressure ventilation. The results have improved considerably during the past few years. Infection and displacement of the endotracheal tube are constant hazards and this form of treatment can be undertaken only where there are adequate nursing and medical staff and enough patients for an effective service to be provided.

Other aspects of treatment

Adequate fluids and electrolytes must be given. The methods will vary with the severity of the respiratory problem. Milk can be given by continuous gastric or intrajejunal infusion. Alternatively, small volumes of milk can be given intermittently at frequent intervals. Some infants need intravenous fluids.

Antibiotics have no place in the treatment of the respiratory distress syndrome, but some infants may need antibiotics as the group B *Streptococcus* may produce a clinical picture similar to that of severe respiratory distress syndrome.

Other causes of respiratory problems

Transient tachypnoea of the newborn is found in full-term infants and usually resolves within 48 hours. The chest radiograph often shows a streaky appearance of the lung fields but may be normal. This syndrome may be due to failure of normal reabsorption of the lung fluid at birth or it may be a mild form of respiratory distress syndrome.

Pneumothorax may follow intermittent positive pressure ventilation during resuscitation of the newborn but it may also occur as a result of the vigorous spontaneous respiratory efforts of a normal infant or during treatment with continuous positive airways pressure. A pneumothorax should be suspected in any infant who deteriorates rapidly for no obvious reason. A chest radiograph confirms the condition, but the diagnosis may be made quickly by transilluminating each side of the chest with a powerful fibreoptic light source. If symptoms are severe a disposable

Apnoea

cannula should be inserted into the pleural space and connected to an underwater seal or a Heimlich disposable valve.

A *diaphragmatic hernia* is usually confirmed by a chest radiograph, which may show loops of small gut or solid organs in the thorax.

The first symptoms of *congenital heart disease* are often noticed by the nurse or mother when the infant has dyspnoea during feeding or is reluctant to feed. The respiratory rate is raised and there may be recession of the chest wall. Excessive weight gain and enlargement of the liver are early confirmatory signs. The edge of the liver is normally about 2 cm below the costal margin in the right midclavicular line in the full-term newborn. There may be no murmurs with some lesions.

Atelectasis is failure of the lungs to expand after birth. It occurs particularly in preterm infants after cerebral birth trauma. Difficult resuscitation after birth is immediately followed by a rise in respiratory rate and recession, and breath sounds are reduced on auscultation.

Pneumonia may occur if there has been rupture of the membranes for longer than 24 hours: the infant may inhale infected liquor before birth and so develop pneumonia. If the mother has had ruptured membranes for over 24 hours before delivery antibiotics should be considered for the mother to prevent or treat pneumonia in the fetus.

Group B streptococcal infection may present with a raised respiratory rate, and a chest radiograph may show extensive areas of consolidation in both lungs or may appear normal.

Meconium aspiration usually occurs in an infant who has become anoxic before delivery. He may start respiratory movements before his mouth and pharynx have been cleared. Aspiration of meconium may cause bronchial obstruction, secondary collapse, and subsequent infection of the distal segments of the lungs. In some units the high mortality has been reduced by bronchial lavage at birth but this needs considerable skill. Oxygen and antibiotics are often needed and mechanical ventilation may be required for a few days.

Preterm infants have a poor cough reflex, and material such as regurgitated milk in the pharynx is easily aspirated into the lungs and may cause pneumonia. Signs may be minimal and are often limited to a small increase in respiratory rate, but a chest radiograph may show extensive changes.

Severe anaemia may cause a raised respiratory rate.

Choanal atresia or stenosis—a congenital posterior nasal obstruction makes it difficult for a newborn infant to breathe as he depends on a clear nasal airway. An oropharyngeal airway produces immediate improvement, and an ENT surgeon should be consulted.

Morphine or pethidine given to the mother before delivery may cause apnoea in the newborn. Cerebral birth trauma, particularly hypoxia, may inhibit the onset of spontaneous respiratory movements after birth so that the baby may need to be intubated and undergo positive-pressure ventilation.

Apnoeic attacks, which occur when respiratory movements stop for over 10 seconds, may be due to cerebral hypoxia during delivery, hypoxaemia due to the respiratory distress syndrome, hypoglycaemia, or meningitis. An ultrasound scan or computerised axial tomography of the brain may help in determining the presence and site of any intracranial haemorrhage.

Apnoea of prematurity occurs in infants of very short gestational age. Several attacks may occur daily for the first month of life. These may be difficult to manage and may be followed by cerebral palsy at a later date if the episodes are prolonged. The various forms of treatment include stimulation of a limb during attacks or prophylaxis with aminophylline or continuous positive airways pressure.

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