respiratory difficulties in the newborn period

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One of the major problems in paediatric practice is respiratory distress of the newborn. This problem is frequently met with; the causes are various and the mortality rate is considerably high.

The neonatal period covers the first four weeks of life. Depending upon its basic cause, respiratory distress may make its appearance immediately after birth or at any time thereafter. The severity varies from simple rapidity of respiratory rate (tachypnoea), to the most extreme air hunger. The main physical signs which are found in most moderate to severe cases are: increased respiratory rate, cyanosis, grunting respirations, intercostal and sternal retraction and seesawing of the abdominal wall. Silverman et al. evaluate severity of respiratory distress by assigning a score of O, 1, or 2, to 5 of these physical signs, the sum constituting the infant's score. Silverman score for assessing respiratory distress is used in most units in the United Kingdom and United States of America.

SILVERMAN SCORING

	Severe	Moderate	Nil
Grunting Respiration	2	1	0
Intercostal Recession	2	1	0
Sternal Recession	2	1	0
Seesawing of Chest Wall	2	1	0
Chin lag	2	1	0
1 — 3 mild respiratory distress			
4 — 6 moderate ,, ,,			
7 — 10 severe ,, ,,			

This scoring system has its faults in that it does not include two of the most important physical signs namely increased respiratory rate and cyanosis, but by and large holds good for quick assessment and for comparing series from various hospitals in different countries.

I would like now to discuss briefly some of the physical signs mentioned above.

Cyanosis is a blue colour of the skin or mucous membranes usually due to the presence of an excessive amount of reduced haemoglobin, and this in turn is due to insufficient oxygenation of the infant's blood. It is not present in mild cases of respiratory distress, as for cyanosis to appear, there must be about 5g. of reduced haemoglobin per 100 c.c. Lesser grades of cyanosis may clear completely in low concentration of O_2 but more severe ones may not disappear even when 100% oxygen is administered.

As intensity of respiratory difficulty increases, more and more of the chest wall retracts with each inspiration. At first the intercostal spaces alone are involved, then the suprasternal notch and eventually the sternum. In the most severe cases the whole chest wall retracts during inspiration whilst the abdomen is blown out *(seesawing* of the abdomen). In this phase all the accessory muscles of respiration are brought into play.

Expiration may be short and accompanied *by a grunt,* or may be prolonged and difficult, and may be accompanied by movement of the chin downwards *(chin lag).*

IDIOPATHIC RESPIRATORY DISTRESS SYNDROME OF NEWBORN (Hyaline Membrane Disease)

This syndrome is by far the commonest cause of death among premature infants, but is also seen with significant frequency in infants of diabetic mothers, in babies born by C.S. and occasionally among infants in whom no predisposing factor is present.

The syndrome consists of increasing respiratory distress at or very often a few hours after birth. Rapid respiratory rate is quickly followed by retraction, expiratory grunt and cyanosis. The disease progresses quickly in some infants, so that they become seriously ill with dyspnoea, deep cyanosis and profound retraction within 3 to 8 hours, but in most, progression to the severe phase takes 12 to 24 hours. Thereafter there is either improvement or deterioration with apnoeic and cyanotic attacks. The mortality rate is as high as 50% in most series and the smaller the infant at birth the higher the mortality rate.

CAUSES OF RESPIRATORY DISTRESS OF THE NEWBORN

The commonest causes are:

(1) Aspiration of amniotic, vaginal or oropharyngeal contents.
(2) Idiopathic Respiratory distress syndrome of the newborn (Hyaline membrane disease).
(3) Atelectasis.
(4) Cardiac failure.
(5) Cerebral Birth Injury.
(6) Pneumonia of the newborn.
(7) Congenital abnormalities e.g.
    Oesophageal atresia.
    Diaphragmatic hernia.
    Posterior choanal atresia.

Rarer causes include:

(1) Congenital cysts or tumours obstructing or pressing on pharynx, larynx, and lungs.
(2) Congenital laryngeal or tracheal stenosis.
(3) Emphysema.
(4) Pulmonary hypoplasia.
(5) Accesory and sequestrated lobes.
(6) Chylothorax and pleural effusion.
(7) Pulmonary haemorrhage.

By far the commonest causes of respiratory distress are idiopathic R.D.S. and massive aspiration.

The cause of this syndrome is not known. At post-mortem pulmonary hyaline membranes with atelectasis are the principal findings, (hence the other name of this syndrome i.e. hyaline membrane disease). But it is not known whether this membrane is the cause of the symptoms. It is not always present in premature babies dying from this syndrome, and it is formed in the course of many disorders.

There are many theories on the cause of the idiopatic R.D.S. of the newborn including aspiration of amniotic fluid and alveolar effusion from the pulmonary circulation (left sided heart failure) with resulting transformation of the fibrinogin of the effusion into fibrin. This in turn covers the alveolar membrane and prevents exchange of gases.

The most recent theory is that the I.R.D.S. may result from a deficiency of a surface tension reducing film of lipoprotein (surfactin) normally present in the alveoli. This deficiency is presumably produced by an inhibiting substance derived from damaged pulmonary tissue or inhaled with amniotic fluids. This substance is believed to interfere with fibrinolysis resulting in the formation of the fibrin — containing hyaline membranes.

Treatment is strictly supportive and one must emphasise the importance of prevention of this syndrome by the prevention of prematurity, avoidance of unnecessary cesarean section and careful management of the diabetic mother.

Treatment include management in an incubator, gentle handling, oxygen, humidity, antibiotics (owing to frequency of complicating pneumonia) and intravenous glucose and bicarbonate to counteract resulting acidosis. Some of these infants develop a hyperkalaemia which in turn can cause hyperkalaemic E.C.G. conduction defects. The proper management of these infants must include repeated blood pH, serum K and serum bicarbonate levels and glucose + insulin is given to correct hyperkalaemia.

THE ASPIRATION SYNDROME

This is due to aspiration into lungs at birth of liquor amnii, meconium, blood or vaginal secretion. Aspiration of small quantities of liquor amnii is commonly met with, giving rise to very little discomfort. On the other hand massive aspiration fill the bronchi and bronchioles with resultant respiratory obstruction and severe distress. Conditions producing foetal asphyxia cause the foetus to breathe or gasp in the uterus and birth canal, these movements being caused by foetal anoxia or hypoxia in utero.

The most frequent cause of foetal aspiration is post-maturity (Peterson and Pendleton), other causes being placenta praevia, maternal haemorrhage, cord prolapse and other causes of compromised placental or foetal circulation. Another, often overlooked cause is administration of heavy sedatives to the mother shortly before delivery. These drugs may induce loss of the mechanisms calculated to prevent aspiration.

The aspirated fluid, by obstructing large airways, may give rise to segmental atelectasis (failure of segments to expand) and in turn emphysema. If the fluid is infected as in cases of prolonged rupture of membranes, it may lead to intrauterine pneumonia.

Respiratory difficulty of various severity occurs at or soon after birth. If there is massive aspiration the baby is shocked and apnoeic. These babies might have suffered intrauterine cerebal anoxia and may die soon after birth. The survivors develop quite marked respiratory distress when they recover from the initial state of shock.

The infants who have not suffered cerebal anoxia carry a good prognosis as the respiratory distress, after lasting a few hours to 2 or 3 days, is followed by rapid recovery unless complications such as gross atelectasis with emphysema, pneumonia or pneunothorax occurs.

Treatment consists of oropharyngeal and laryngeal toilet via an endotracheal tube, oxygen and antibiotics. the onset of feeding is delayed.

ATELECTASIS

Atelectasis means incomplete expansion of a lung or a portion of a lung. Atelectasis may be primary due to pulmonary immaturity (prematurity) or inadequacy of respiratory effort as occurs in oversedation or cerebral birth injury. In most cases it is secondary to other disease process as in massive inhalation and idiopathic respiratory distress syndrome of the newborn. It can also be caused by abnormal external pressure upon the lung as in congenital diaphragmatic hernia.

CARDIAC FAILURE

Congenital heart disease may present in the neonatal period as cardiac failure. There is dyspnoea, tachypnoea, tachycardia and cyanosis. Intercostal recession may also be present but it is rarely as great as in pulmonary disease. Enlargement of the liver may be present. Dyspnoea may occur in paroxysms in cyanotic infants who are not in heart failure but who suffer from anoxic spells. Among congenital heart defects giving rise to neonatal respiratory distress are transpositions of the great vessels, total anomalous pulmonary venous drainage, coarctation of the aorta, patent ductus arteriosus aortic atresia. Some of these defects are amenable to early surgery.

CEREBRAL BIRTH INJURY

This has already been mentioned as a cause of massive aspiration and atelectasis. Intracranial haemorrhage whether due to trauma or to anoxia is very often present. There is bleeding in the area of the respiratory centre which in turn leads to failure of respiratory centre resulting in weakness of respiratory effort and apnoeic attacks.

PNEUMONIA OF THE NEWBORN

Pneumonia is one of the important causes of perinatal death. It may be acquired in utero, during labour and after birth.

Pneumonia can be acquired in utero either transplacentally secondary to maternal septicaemia, or an ascending infection in cases of prolonged rupture of the membranes.

It can be acquired during labour if baby inhales maternal faecal matter or infected liquor.

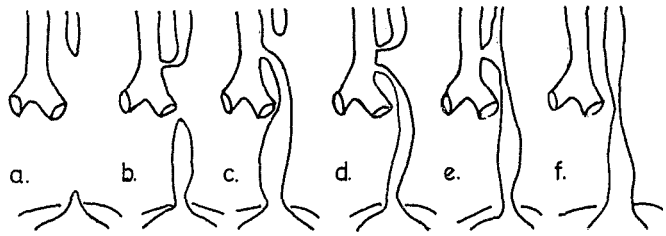
Post-natally it can be due to aspiration of food or gastric juice, it can be due to airborne bacterial, viral and fungal infections, and it can be secondary to a septicaemia. Respiratory distress may be present at birth in cases of intra-uterine pneumonia but usually develops later. Oxygen and antibiotics are the main lines of treatment.

CONGENITAL ABNORMALITIES

In addition to the illnesses described above, there are a number of important surgically remediable congenital abnormalities which can give rise to severe respiratory distress. They are less common than the illnesses already described but are by no means rare. The following conditions should be borne in mind in any infant with respiratory illness. They are the commonest of this group of illness.

(1) Oesophageal Atresia with Tracheo-oesophageal fistula.
(2) Congenital Diaphragmatic Hernia.
(3) Congenital Lobar Emphysema.
(4) Choanal Atresia.

A chest X-Ray is an important help to the diagnosis in all cases of respiratory distress of the newborn but more so if one of the above conditions is suspected.



Six types of congenital anomalies of the oesophagus as described by R.E. Gross (Surgery of Infancy and childhood).
a - d=oesophageal ateresia, b - e=tracheo-oesophageal fistula, f=oesphageal stenosis.

(a) *Oesophageal atresia with Tracheo-oesophageal fistula.*

Recurrent collection of mucus in baby's throat should give rise to immediate suspicion of atresia and diagnosis is confirmed clinically if a small gastric catheter cannot be passed via the oesophagus into the stomach.

Once diagnosis is made early surgery by closing the tracheo-oesophageal fistula and gastrostomy is essential as a life-saving procedure. Reconstructive surgery on the oesophagus is performed at a later date.

(b) *Congential Diaphragmatic Hernia,*

This is another condition in which surgery is essential as a life-saving procedure as soon as diagnosis is made. The small bowels and sometimes parts of the large bowel and spleen are within the chest cavity, the lung on the affected side being completely collapsed. The mediastinum is displaced to the opposite side so the lung on the opposite side may also not be fully expanded due to this pressure. Because of this there is severe dyspnoea and cyanosis which are exacerbated by attempted feeding. A clue to the diagnosis may be a bulging hemithorax (due to the increased contents) and a fairly flat abdomen. Chest X-Ray is diagnostic and only surgery can save an infant's life.

(c) *Congenital Lobar Emphysema* occurs as the result of a congenital deficiency of the bronchial cartilage. The affected lobe expands enormously and compresses the remaining lobe on the same side, and also due to shifting the mediastinum compresses the lung on the other side. Chest X-Ray shows marked translucency of the affected side. Lobectomy will be necessary.

(d) *Posterior choanal atresia.*

In this condition the openings of the nose into the nasopharynx are closed by a septum containing bone. A bilateral case will present with cyanosis and violent inspiratory effort. If the mouth is opened and tongue drawn forward air can be inhaled easily. A newborn baby is unable to mouth breath and some form of tube over the back of the tongue may be used to allow mouth breathing until the choanae can be opened by operation.

For further information about the conditions mentioned in this article the reader is instructed to look up standard works of paediatrics such as *Nelson;* 'Text Book of Paediatrics' and *Schaffes,* 'Disease of the Newborn'.