

[Newborn Care Manual: Contents](#)**RESPIRATORY DISTRESS****25-1 WHAT IS RESPIRATORY DISTRESS?**

Respiratory distress in a newborn infant presents as a group of clinical signs which indicates that the infant has difficulty breathing. The 4 most important clinical signs of respiratory distress are:

1. TACHYPNOEA. A respiratory (breathing) rate of 60 or more breaths per minute (normal respiratory rate is less than 60).
2. CENTRAL CYANOSIS. A blue tongue in room air.
3. RECESSION. The in-drawing of the ribs and sternum during inspiration (also called retractions).
4. GRUNTING. A snoring noise made in the throat during expiration.

If an infant has 2 or more of the above clinical signs, the infant is said to have respiratory distress. Most infants with respiratory distress have central cyanosis.

AN INFANTS HAS RESPIRATORY DISTRESS IF TWO OR MORE OF THE IMPORTANT CLINICAL SIGNS OF RESPIRATORY DISTRESS ARE PRESENT

**** Respiratory distress (or respiratory distress syndrome) is not a complete diagnosis as there are many different causes.*

25-2 WHAT ARE THE IMPORTANT CAUSES OF RESPIRATORY DISTRESS?

Respiratory distress in newborn infants has many pulmonary (lung) as well as extra-pulmonary (outside the lungs) causes.

The most important PULMONARY CAUSES of respiratory distress are:

1. Hyaline membrane disease.
2. Wet lung syndrome.
3. Meconium aspiration.
4. Pneumonia.

The important EXTRA-PULMONARY CAUSES of respiratory distress are:

1. Pneumothorax.
2. Heart failure.
3. Hypothermia.
4. Metabolic acidosis.
5. Anaemia.
6. Polycythaemia.

**** Less common pulmonary causes of respiratory distress include pulmonary haemorrhage, hypoplastic lungs and chronic lung disease (bronchopulmonary dysplasia) while less common extra-pulmonary causes include diaphragmatic hernia and persistent pulmonary hypertension.*

THERE ARE MANY DIFFERENT CAUSES OF RESPIRATORY DISTRESS

Always look for the cause if an infant has respiratory distress. Simply saying that an infant has respiratory distress is not enough.

25-3 HOW SHOULD YOU MANAGE AN INFANT WITH RESPIRATORY DISTRESS?

The principles of care are the same, irrespective of the cause of the respiratory distress. Therefore, all infants with respiratory distress should receive the following general management:

1. Keep the infant warm, preferably in an incubator.
2. Handle the infant as little as possible, because stimulating the infant often increases the oxygen requirements. There is no need to routinely suction the airways.
3. Provide energy, preferably by giving an infusion of maintenance fluid (Neonatalyte).
4. Treat central cyanosis by giving head box or nasal catheter oxygen. An air/oxygen blender or venturi must be used. Monitor the percentage (fraction) of inspired air (F_iO_2) and arterial oxygen saturation (SaO_2) or arterial oxygen pressure (PaO_2). If this is not possible, give just enough oxygen to keep the infant's tongue pink.
5. Record the following important observations every hour and note any deterioration:
 - (i) Respiratory rate.
 - (ii) Presence or absence of recession and grunting.
 - (iii) Presence or absence of cyanosis.
 - (iv) Percentage of inspired oxygen (F_iO_2) if possible.
 - (v) Arterial oxygen saturation (SaO_2) if possible.
 - (vi) Heart rate.
 - (vii) Both the skin (or axilla) and incubator temperature.
6. Take a chest X-ray.
7. If possible measure the infant's arterial blood gasses (pH, oxygen and carbon dioxide).
8. Consult the nearest level 2 or 3 hospital as the infant may need to be transferred. This is particularly important in hyaline membrane disease.
9. Keep the infant pink in head box oxygen or continuous positive airways pressure (CPAP) via nasal prongs. With continuous positive airways pressure a mixture of air and oxygen is blown into the infant's lungs continuously while the infant breathes spontaneously. This helps to keep the alveoli expanded which improves oxygenation. Giving head box oxygen is easier but nasal prong oxygen may be more effective.
10. If the infant develops recurrent apnoea or if continuous positive airways pressure fails to keep the infant pink, then intubation and ventilation are indicated.

In addition to the general management of respiratory distress, any specific treatment of the cause of the respiratory distress must be given, e.g. antibiotics for pneumonia.

**** Spasm of the pulmonary arteries may be caused by excessive handling, hypothermia, acidosis or hypoxia. Pulmonary blood is then shunted away from the lungs via the foramen ovale and ductus arteriosus. This makes the hypoxia much worse. It is, therefore, essential to avoid these aggravating factors.*

25-4 WHAT IS HYALINE MEMBRANE DISEASE (HMD)?

At term the fetal alveoli are mature and ready to be inflated with air after delivery. These mature alveoli secrete a substance called SURFACTANT that prevents them collapsing completely at the end of expiration. This allows the infant to breathe air in and out with very little physical effort.

In contrast, infants with immature lungs do not have adequate amounts of surfactant at birth. As a result their alveoli collapse with expiration and the infant has difficulty expanding them again during inspiration. Collapsed alveoli, due to the lack of surfactant, result in respiratory distress. This condition is known as hyaline membrane disease (HMD).

HYALINE MEMBRANE DISEASE IS CAUSED BY THE LACK OF SURFACTANT IN IMMATURE LUNGS

*** *The lungs of infants with hyaline membrane disease have 3 major abnormalities:*

1. *Generalized alveolar collapse due to inadequate amounts of surfactant.*
2. *The collapsed alveoli fill with a protein-rich fluid that forms hyaline membranes, giving the condition its name.*
3. *Spasm of the pulmonary arteries which results in blood being shunted away from the lungs via the foramen ovale and ductus arteriosus. These abnormalities all result in respiratory failure with poor oxygenation of the blood.*

25-5 WHICH INFANTS DO NOT HAVE ADEQUATE SURFACTANT?

Preterm infants often have immature lungs with inadequate surfactant. Therefore, the more preterm the infant, the greater is the risk of hyaline membrane disease.

HYALINE MEMBRANE DISEASE IS A MAJOR CAUSE OF DEATH IN PRETERM INFANTS

25-6 HOW CAN YOU TELL WHETHER THE INFANTS HAS ADEQUATE AMOUNTS OF SURFACTANT?

1. The presence or absence of surfactant in the fetal lung can be determined before delivery by doing a BUBBLES TEST on a sample of amniotic fluid obtained by amniocentesis.
2. Similarly the SHAKE TEST on a sample of gastric aspirate obtained within 30 minutes after delivery will indicate whether adequate amounts of surfactant are present in the lungs of a newborn infant.

The method of doing a shake test is described in skills workshop 25 in this PEP manual.

*** *Fetal lung fluid is both swallowed and passed out of the mouth into the amniotic fluid during pregnancy. A sample of amniotic fluid before delivery, or gastric aspirate immediately after delivery can, therefore, be used to test whether surfactant is being produced by the alveoli.*

25-7 HOW DO YOU DIAGNOSE HYALINE MEMBRANE DISEASE?

1. The infant is almost always preterm. Only occasionally does a term infant develop hyaline membrane disease. Term infant with hyaline membrane disease are usually born to women with poorly controlled diabetes.
2. The bubbles test on amniotic fluid or the shake test on gastric aspirate is negative indicating inadequate surfactant.
3. The infant develops respiratory distress at or soon after delivery. The signs of respiratory distress gradually become worse during the first 48 hours after birth.
4. The infant is usually inactive and commonly develops peripheral oedema.
5. The chest X-ray is abnormal and shows small lungs with granular lung fields. These findings are the result of alveolar collapse. A typical chest X-ray is needed to make a definite diagnosis of hyaline membrane disease.

*** *The typical X-ray findings of hyaline membrane disease include small lung volume (best seen on the lateral view) with air bronchograms extending beyond the cardiothymic shadow, granular opacities extending out to the periphery of the lungs, poor distinction between the cardiothymic shadow and the lungs, and usually a large thymus. The X-ray features of hyaline membrane disease may not be typical in the first few hours after birth.*

25-8 WHAT IS THE CLINICAL COURSE IN HYALINE MEMBRANE DISEASE?

The degree of respiratory distress gets worse and the concentration of inspired oxygen needed to keep the infant pink increases for the first 2 to 3 days after birth. During this time some infants will die of hyaline membrane disease. Otherwise the respiratory distress gradually improves after 48 to 72 hours and the oxygen can usually be stopped after 5 to 10 days of age. Once fully recovered the infant's lungs are usually normal, although repeated episodes of bronchiolitis during the first year of life are common.

**** The clinical signs of respiratory distress get worse after delivery as the alveolar surfactant is gradually used up. However after 2 to 3 days of breathing air the lungs start to produce surfactant again and the signs of respiratory distress, therefore, improve.*

HYALINE MEMBRANE DISEASE GETS WORSE BEFORE IT GETS BETTER

25-9 HOW DO YOU PREVENT HYALINE MEMBRANE DISEASE?

1. If possible, preterm delivery should be prevented. Unfortunately this is often not possible.
2. If the patient is in preterm labour before 34 weeks, labour should be suppressed if there are no contra-indications.
4. In preterm labour, it is possible to assess whether the fetal lungs are mature by doing a bubbles test on a sample of amniotic fluid. However, this is usually no longer done.
3. Steroids (betamethasone) can be given to the mother to accelerate maturation of the fetal lungs if the gestational age is less than 34 weeks (or if the bubbles test is negative).
5. All preterm infants must be adequately resuscitated.
6. Prevent hypothermia, hypoglycaemia and hypoxia after birth as they can all decrease the production of surfactant.

MATERNAL STEROIDS DURING PRETERM LABOUR CAN PREVENT HYALINE MEMBRANE DISEASE IN MANY INFANTS

25-10 HOW DO YOU MANAGE HYALINE MEMBRANE DISEASE?

Unfortunately artificial surfactant is expensive and, therefore, it is not possible to correct the deficiency of surfactant in many infants. The management of hyaline membrane disease consists of giving oxygen and providing general supportive management in most infants. If the infant can be kept alive for the first 72 hours, recovery usually occurs when spontaneous surfactant production increases. It is important to diagnose hyaline membrane disease as soon as possible after birth because these infants need to be transferred to a level 2 hospital with a newborn intensive care unit, or a level 3 hospital. Whenever possible, all infants at high risk of hyaline membrane disease should be delivered in a level 2 or 3 hospital.

Giving oxygen with nasal prongs (continuous positive airways pressure) is the best method in infants with hyaline membrane disease. If this fails to keep the infant pink, intubation and ventilation are needed. However, head box oxygen is usually adequate in many infants with mild hyaline membrane disease.

**** Artificial surfactant is instilled down an endotracheal tube. It is usually given within the first few hours to infants with hyaline membrane disease who cannot be adequately oxygenated with nasal prong CPAP. The use of artificial surfactant is often restricted to level 3 units.*

25-11 WHAT ARE THE COMPLICATIONS OF HYALINE MEMBRANE DISEASE?

1. All the other problems of the preterm infant are common in these infants, especially jaundice, apnoea of immaturity, hypothermia and hypoglycaemia.
2. Hypoxic brain damage if the infant cannot be kept pink.
3. Secondary bacterial pneumonia if the infant is intubated.
4. Intraventricular haemorrhage.
5. Pneumothorax.
6. Patent ductus arteriosus.
7. Chronic lung disease (bronchopulmonary dysplasia).

25-12 WHAT IS THE WET LUNG SYNDROME?

Before delivery the fetal lungs are not collapsed but the alveoli and bronchi are filled with lung fluid. At vaginal delivery, most of this fluid is squeezed out of the lungs as the chest is compressed in the birth canal. After birth the remaining fluid is coughed up or is absorbed into the capillaries and lymphatics of the lung within a few minutes. In some infants this rapid removal of fetal lung fluid does not take place resulting in the wet lung syndrome which presents as respiratory distress. The wet lung syndrome (also called "wet lungs" or transient tachypnoea of the newborn) is the commonest cause of respiratory distress. It is also important because during the first day of life it can easily be confused with hyaline membrane disease.

THE WET LUNG SYNDROME IS THE COMMONEST CAUSE OF RESPIRATORY DISTRESS**25-13 WHICH INFANTS COMMONLY DEVELOP THE WET LUNG SYNDROME?**

In the following conditions the normal clearance of lung fluid is often delayed for many hours resulting in the wet lung syndrome:

1. Caesarean section, especially if the mother has not been in labour and the membranes have not been ruptured before delivery (elective caesarean section).
2. Fetal hypoxia or severe birth asphyxia.
3. Maternal sedation.
4. Polyhydramnios.

In some infants, however, the above risk factors are not present and the cause of the wet lung syndrome is not known.

**** Excessive secretion of pulmonary fluid, poor respiratory efforts, damaged pulmonary capillaries and poor contraction of the left ventricle probable all can result in the wet lung syndrome.*

25-14 HOW CAN YOU DIAGNOSE THE WET LUNG SYNDROME?

1. These infants may be born at or before term.
2. They develop respiratory distress soon after delivery.
3. They often have an overinflated chest and usually do not need more than 50% oxygen to correct the central cyanosis.
4. Their clinical signs gradually improve after birth and usually disappear by 72 hours.
5. The shake test on the gastric aspirate is positive, which excludes hyaline membrane disease.
6. The chest X-ray in the wet lung syndrome shows hyperinflated (large) lungs, which is different from the small lungs seen in hyaline membrane disease.

THE WET LUNG SYNDROME IS IMPORTANT BECAUSE IT CAN BE CONFUSED WITH HYALINE MEMBRANE DISEASE

**** After 6 hours of age the chest X-ray in the wet lung syndrome is typical with hyperexpanded lungs (due to air trapping caused by oedematous small airways), increased parahilar vascular markings (due to dilated lymphatic and capillaries) and clear peripheral lung fields. However, the chest X-ray in the first few hours after delivery may be similar to that of hyaline membrane disease due to the presence of alveolar fluid. It is, therefore, best to wait a few hours before taking a chest X-ray if hyaline membrane disease and wet lung syndrome are to be differentiated.*

25-15 WHAT IS THE CLINICAL COURSE OF THE WET LUNG SYNDROME?

Respiratory distress caused by the wet lung syndrome presents at or soon after birth and can mimic hyaline membrane disease for the first few hours after delivery. However, infants with the wet lung syndrome gradually improve during the first 24 hours, and oxygen is usually needed for 2 to 3 days only. The clinical course of the wet lung syndrome is very different from that of hyaline membrane disease.

WET LUNG SYNDROME STEADILY IMPROVES AFTER DELIVERY

25-16 HOW SHOULD YOU MANAGE INFANTS WITH THE WET LUNG SYNDROME?

The management of the wet lung syndrome is the same as that for respiratory distress in general. However, oxygen alone is usually all that is needed to prevent cyanosis, and only rarely is there a need to transfer the infant to a level 2 or 3 unit as the condition can be expected to steadily improve. Three hourly feeds by nasogastric tube, rather than an intravenous infusion, can usually be given.

25-17 WHAT IS THE MECONIUM ASPIRATION SYNDROME?

If the fetus is hypoxic in utero it may pass meconium and make gasping movements which suck the meconium stained liquor into the larynx and trachea. If the airways are not well suctioned after the head is delivered, the meconium can be inhaled into the smaller airways and alveoli with the onset of breathing, resulting in the following lung damage:

1. Meconium contains enzymes (from the fetal pancreas) which damage the epithelial lining of the bronchi and bronchioles.
2. The enzymes in meconium also cause severe alveolar damage.
3. Meconium plugs partially or completely block the airways resulting in some areas of collapsed lung and other areas of over expanded lung.

**** Severe meconium aspiration causes a chemical pneumonitis and almost always results in pulmonary hypertension with shunting of blood away from the lungs via the foramen ovale and ductus arteriosus. This causes severe hypoxaemia.*

25-18 HOW DO YOU DIAGNOSE THE MECONIUM ASPIRATION SYNDROME?

1. The infant is usually born at term or post term but only rarely preterm.
2. The amniotic fluid is meconium stained.
3. Meconium may be suctioned from the mouth and upper airways at birth and the infant is usually meconium stained.
4. Respiratory distress is present and the chest usually appears hyperinflated (over expanded).
5. The chest X-ray shows hyperinflation with many white areas of collapsed lung.

**** The contradictory combination of marked hyperinflation (due to partially blocked airways) together with diffuse patches of collapsed lung (due to completely blocked airways and chemical pneumonitis) is typical of the meconium aspiration syndrome.*

25-19 WHAT IS THE CLINICAL COURSE OF THE MECONIUM ASPIRATION SYNDROME?

From birth the meconium stained infant has respiratory distress which, in severe cases, gets progressively worse and may kill the infant. Milder cases will gradually recover over days or weeks. Infants who survive severe meconium aspiration often have damaged lungs that may take months to recover.

25-20 CAN YOU PREVENT THE MECONIUM ASPIRATION SYNDROME?

Yes, most cases of severe meconium aspiration syndrome can be prevented by carefully suctioning the upper airways of meconium stained infants BEFORE they breathe at birth. Therefore, it is essential to clear the airways before the infant's shoulders are delivered.

MECONIUM ASPIRATION CAN USUALLY BE PREVENTED BY SUCTIONING THE UPPER AIRWAYS IMMEDIATELY AFTER THE INFANT'S HEAD HAS BEEN DELIVERED

Meconium is usually passed and sucked into the large airways by a fetus who suffers hypoxia during labour. Because the fetal alveoli are filled with lung fluid before delivery, very little meconium can get into the small airways and alveoli until the infant inhales air at delivery.

25-21 HOW SHOULD YOU MANAGE AN INFANT WITH THE MECONIUM ASPIRATION SYNDROME?

1. Management consists of the supportive care needed by any infant with respiratory distress.
2. Unfortunately there is no specific treatment for the infant with respiratory distress caused by meconium aspiration. The value of steroids, to decrease the inflammation, and prophylactic antibiotics remains unproved and, therefore, they are usually not given.
3. Continuous positive airways pressure or mechanical ventilation may be needed to correct hypoxaemia.
4. A stomach washout with 2% sodium bicarbonate or half normal saline helps to prevent gastritis caused by meconium. The phagocytes in colostrum feeds also help in the removal of meconium from the stomach.
5. Look for complications.

IT IS FAR BETTER TO PREVENT THAN HAVE TO TREAT MECONIUM ASPIRATION SYNDROME**25-22 WHAT ARE THE COMPLICATIONS OF MECONIUM ASPIRATION?**

1. Pneumothorax and pneumomediastinum are common due to rupture of areas of over expanded lung.
2. Hypoxic damage to other organs, such as the brain, due to the hypoxia that caused the fetus to pass meconium.
3. Meconium gastritis which presents with repeated vomiting of meconium stained mucus.

**** Persistent pulmonary hypertension (damage and spasm of the pulmonary arteries) often complicated meconium aspiration syndrome and causes severe hypoxia.*

25-23 WHAT IS THE CAUSE AND TREATMENT OF PNEUMONIA?

An infant may be born with pneumonia (congenital pneumonia) as a complication of chorioamnionitis. The diagnosis of congenital pneumonia resulting from chorioamnionitis is suggested by seeing pus cells and bacteria in a Gram stain of the gastric aspirate after delivery in an infant with respiratory distress. Infants, especially preterm infants, may also develop pneumonia in the days or weeks after birth (acquired pneumonia) due to the spread of bacteria by the hands of staff or parents (nosocomial infection). Pneumonia is common in infants receiving ventilation. The clinical diagnosis of pneumonia can be confirmed by a chest X-ray which usually shows areas of collapsed or consolidated lung. Every effort must be made to prevent pneumonia in ventilated infants by practicing good aseptic techniques (clean hands). Treatment of pneumonia is supportive care plus parenteral antibiotics, e.g. penicillin and gentamicin, or ceftriaxone.

CLEAN HANDS CAN PREVENT MANY CASES OF PNEUMONIA IN A NEWBORN CARE UNIT**25-24 WHAT IS A PNEUMOTHORAX?**

A pneumothorax (pneumo=air; thorax=chest) is a collection of air in the pleural cavity surrounding the lung. It is caused by the rupture of one or more alveoli which allows air to escape from the lung. The pneumothorax compresses the lung and prevents normal lung expansion during inspiration. Usually a pneumothorax occurs on one side only but it may be bilateral (pneumothoraces).

25-25 WHO IS AT RISK OF PNEUMOTHORAX?

1. All infants with respiratory distress, whatever the cause.
2. Infants with meconium aspiration are at increased risk.
3. Infants that need intubation and ventilation at resuscitation.
4. Infants that are intubated and are ventilated in the nursery.

25-26 HOW DO YOU DIAGNOSE A PNEUMOTHORAX?

The clinical diagnosis of pneumothorax in the newborn is often very difficult as the classical signs may not be present. The following signs are helpful however:

1. Sudden unexpected collapse.
2. Rapidly increasing oxygen needs in respiratory distress.
3. Poor breath sounds with little movement on one side of the chest.
4. An easily palpable liver in a right sided pneumothorax.
5. Poor heart sounds or heart sounds best heard on the right of the sternum in a left sided pneumothorax (the heart is pushed to the right).

The suspected clinical diagnosis can be confirmed by:

1. Transillumination of the chest. The chest wall on the side of the pneumothorax transilluminates well while the chest wall on the normal side does not.
2. A chest X-ray which will show air in the pleural space.

The method of transilluminating an infant's chest is described in skills workshop 25 of this PEP manual.

25-27 HOW DO YOU TREAT A PNEUMOTHORAX?

1. If the infant has mild respiratory distress with a small pneumothorax and is not cyanosed in headbox oxygen, the infant can be closely observed. Many pneumothoraces will reabsorb without drainage. However, infants requiring oxygen should be transferred to a level 2 or 3 hospital where a chest drain can be inserted, if necessary.
2. If the infant develops severe respiratory distress, is receiving continuous positive airways pressure or is on a ventilator, a chest drain must be inserted immediately.
3. If a chest drain cannot be inserted due to lack of equipment or a trained person, the pleural space can be aspirated with a needle and syringe as an emergency procedure. This is a first aid measure only and must be followed as soon as possible with a chest drain.

*** The methods of aspirating a pneumothorax and inserting a chest drain are described in skills workshop 25.

25-28 WHAT ARE THE COMMON CAUSES OF HEART FAILURE IN THE NEWBORN INFANT?

There are many different causes of heart failure. The common causes in the newborn infant are:

1. Patent ductus arteriosus.
2. Congenital malformation of the heart.
3. Infusion of excessive amounts of intravenous fluid.
4. Hypoxia.
5. Anaemia.

Heart failure in most of these conditions presents as respiratory distress due to pulmonary oedema.

25-29 WHAT IS A PATENT DUCTUS ARTERIOSUS (PDA)?

The ductus arteriosus is a large artery that joins the aorta and pulmonary artery in the fetus. Because the lungs do not function before birth, blood from the pulmonary artery by-passes the fetal lungs via the ductus arteriosus to the aorta. After delivery the ductus arteriosus normally closes and blood then passes from the pulmonary artery to the lungs. In preterm infants the ductus often does not close normally but remains open (patent) for a few weeks. As a result, blood flows backwards from the aorta into the pulmonary artery, flooding the lungs with blood and causing heart failure. This usually presents 5 or more days after delivery and may be precipitated by increasing the infant's feeds to more than 150 ml/kg/day. If the ductus is large then it will cause pulmonary oedema and present with signs of respiratory distress.

The diagnosis of a patent (open) ductus arteriosus can be made by observing the following signs:

1. A heart murmur.
2. Collapsing pulses (the pulses are very easy to feel).
3. In severe cases the infant will also have signs of respiratory distress.

**** The heart murmur is typically pansystolic and heard best under the left clavicle or to the left of the sternum. Usually the heart beat can also be easily felt by placing your hand over the infant's lower sternum. On chest X-ray the lungs appear congested. The clinical diagnosis can be easily confirmed by cardiac ultrasonography.*

25-30 HOW SHOULD YOU TREAT A PATIENT WITH A PATENT DUCTUS ARTERIOSUS?

If the infant has no signs of heart failure, then the feeds should not exceed 150 ml/kg/day and the infant should be carefully observed. In most cases further treatment is not needed and the ductus closes spontaneously when term is reached.

However, if the infant has signs of respiratory distress:

1. The infant must be referred to a level 2 or 3 hospital.
2. Restrict fluid intake to 120 ml/kg/day.
3. Furosemide (Lasix) 1 mg/kg must be given orally or by intramuscular or intravenous injection.
4. Transfuse very slowly with packed cells (10 ml/kg) if the PCV is below 30% (Hb below 10 g/dl).

If the infant fails to respond to this management then the infant must be transferred to a level 3 hospital for ultrasound examination to confirm the clinical diagnosis. Oral or intravenous treatment with indomethacin (Indocid) is used to close the patent ductus arteriosus. Rarely surgical closure may be needed.

**** Indomethacin blocks the synthesis of prostaglandins which keep the ductus arteriosus patent. Indomethacin has many side effects, however, including renal failure.*

25-31 HOW CAN YOU DIFFERENTIATE BETWEEN THE COMMON CAUSES OF RESPIRATORY DISTRESS?

Factors in the history, physical examination and investigations may suggest a particular cause for the respiratory distress:

1. HISTORY:
 - (i) Hyaline membrane disease in a preterm infant or an infant of a diabetic mother.
 - (ii) Wet lung syndrome in an infant born by elective caesarean section.
 - (iii) Meconium aspiration if the infant is meconium stained.
 - (iv) Congenital pneumonia if there has been maternal pyrexia or offensive liquor.
 - (v) Patent ductus arteriosus in a two week old preterm infant.
2. EXAMINATION:
 - (i) Preterm infant in hyaline membrane disease.
 - (ii) Hyperexpanded chest in the wet lung syndrome.
 - (iii) Meconium staining in the meconium aspiration syndrome.
 - (iv) Offensive smell at birth in congenital pneumonia.
 - (v) Asymmetrical chest movement and breath sounds in pneumothorax.
 - (vi) Murmur in an infant with a patent ductus arteriosus.

3. INVESTIGATIONS:

- (i) No surfactant on shake test in hyaline membrane disease.
- (ii) Pus cells and bacteria in the gastric aspirate in congenital pneumonia.
- (iii) Typical chest X-ray in hyaline membrane disease, the wet lung syndrome, the meconium aspiration syndrome, pneumonia and pneumothorax.
- (iv) Transillumination in pneumothorax.

APNOEA

25-32 WHAT IS APNOEA?

APNOEA is the arrest (stopping) of respiration for long enough to cause bradycardia together with cyanosis or pallor. The oxygen saturation falls with apnoea. Usually apnoea for 20 seconds or longer is needed to produce these clinical signs. The patient may have a single apnoeic attack but usually the episodes of apnoea are repeated.

INFANTS WITH APNOEA STOP BREATHING FOR LONG ENOUGH TO RESULT IN BRADYCARDIA AND CYANOSIS OR PALLOR

Apnoea should not be confused with PERIODIC BREATHING, which is a normal pattern of breathing in preterm and some term infants. These infants have frequent short pauses (less than 20 seconds each) in their respiration. With periodic breathing, the arrest of breathing movements does not last long enough to cause bradycardia, cyanosis or pallor.

PERIODIC BREATHING DOES NOT CAUSE BRADYCARDIA, CYANOSIS OR PALLOR

25-33 HOW SHOULD YOU DIAGNOSE APNOEA?

1. The diagnosis of apnoea is usually made by observing the breathing pattern, colour and heart rate of an infant.
2. Apnoea can also be diagnosed with the aid of an apnoea monitor which is usually set to trigger if the infant does not breathe for 20 seconds. A solid sensor pad is placed under the infant, or electrodes are attached to the infant's chest. The sensor pad or electrodes are attached via a connecting lead to the monitor unit.
3. A cardiorespiratory monitor, that measures and displays both the respiratory and heart rate, can also be used to detect apnoea.
4. An oxygen saturation monitor will indicate a fall in oxygen saturation when the infant has apnoea.

The use of an apnoea monitor is described in skills workshop 25 of this PEP manual.

25-34 WHAT ARE THE CAUSES OF APNOEA?

Apnoea is a clinical sign that has many causes:

1. The commonest cause is apnoea of immaturity.
2. Respiratory distress of any cause may result in apnoea.
3. Infection, especially pneumonia, septicaemia and meningitis may cause apnoea.
4. Hypoxia, hypothermia or hypoglycaemia.
5. Hyperthermia due to over heating in an incubator is an important and easily correctable cause of apnoea.
6. Intraventricular haemorrhage.
7. A large feed, vomit or gastro-oesophageal reflux.
8. Convulsions may present with recurrent apnoea only.
9. Maternal analgesia or sedation during labour, e.g. pethidine, morphine or diazepam (Valium).
10. Anaemia, especially if the infant also has a patent ductus arteriosus.

25-35 WHAT IS APNOEA OF IMMATURITY?

In some preterm infants the respiratory centre in the brain stem is immature and this results in repeated attacks of apnoea. These infants are usually under 34 weeks of gestation. The more preterm the infant, the greater is the risk of apnoea of immaturity. Apnoeic attacks usually start after 48 hours of age, and occur especially after a feed.

25-36 HOW SHOULD YOU MANAGE APNOEA?

1. Always look for a cause of the apnoea and treat the cause if possible.
2. Apnoea of immaturity can be largely prevented and treated with the use of oral theophylline or caffeine.
3. Nursing infants slightly head up on their abdomen (the prone position) decreases the incidence of apnoea.
4. Keeping the infant's skin temperature strictly between 36 and 36,5°C helps to prevent apnoea.
5. Monitor infants for apnoea, or infants with a high risk of apnoea, using an apnoea monitor, cardiorespiratory monitor or oxygen saturation monitor.
6. During an attack of apnoea, breathing can be restarted in most cases by simply stimulating the infant, provided the apnoea has only lasted a few seconds. Flicking the feet is usually adequate to restart breathing.
7. Headbox oxygen, at a concentration not higher than 25% (FiO₂ 0,25), may prevent repeated apnoea. Giving a higher concentration of oxygen to prevent apnoea is extremely dangerous as it can cause retinopathy of prematurity.
8. Continuous positive airways pressure via nasal prongs is used to prevent repeated apnoea if theophylline fails.
9. In more severe apnoea, mask ventilation is needed and occasionally the infant will require intubation and ventilation. If oxygen is used, the concentration must be reduced to 25% or less as soon as breathing is established and the cyanosis corrected.
10. Apnoea needing ventilation usually is not due to immaturity but is caused by some other more serious problem.

APNOEA OF IMMATURITY CAN BE PREVENTED BY ORAL THEOPHYLLINE**25-37 HOW IS THEOPHYLLINE ADMINISTERED?**

Theophylline is usually given via a nasogastric tube as Nuelin liquid. With oral theophylline, a loading dose of 5 mg/kg is given, followed by a maintenance dose of 2,5 mg/kg every 12 hours. Prophylactic theophylline is given routinely to all infants born before 35 weeks of gestation. It can usually be stopped at 35 weeks or when a weight of 1800 g is reached. When an infant receives an overdose of theophylline, the infant presents with tachycardia, vomiting or convulsions.

Oral caffeine is very effective but has to be made up by the hospital pharmacy. A loading dose of 10 mg/kg is followed by a daily dose of 2,5 mg/kg.

**** Theophylline can also be given intravenously at the same dose as oral theophylline. The serum concentration of theophylline can be measured to determine whether the correct dose is being given. The correct range to prevent apnoea of immaturity is 5-10 µg/ml.*

CASE PROBLEMS**CASE 1**

A male infant is born at 32 weeks gestation in a level 1 hospital. Soon after delivery his respiratory rate is 80 breaths per minute with recession and expiratory grunting. The infant's tongue is blue in room air. The gastric aspirate collected 10 minutes after delivery contains no pus cells or bacteria on Gram stain but the shake test is negative.

1. What are the infant's clinical signs which indicate that he has respiratory distress.

Tachypnoea, recession, grunting and central cyanosis in room air.

2. What is the probable cause of the respiratory distress? Give reasons for your answer.

The infant probably has hyaline membrane disease due to immature lungs. This is common in infants born preterm. The diagnosis is supported by the negative shake test. The normal Gram stain suggests that the infant does not have congenital pneumonia as a complication of chorioamnionitis.

3. Should this infant remain at the level 1 hospital?

No, he should be moved as soon as possible to a level 2 or 3 hospital with staff and facilities to care for sick infants. Hyaline membrane disease deteriorates for 2 to 3 days before improving. Therefore, this infant will need more intensive care during the next 72 hours.

4. What would you expect to see on a chest X-ray of this infant?

The lungs will appear small and granular due to collapsed alveoli.

5. How would you manage this infant before transfer to a larger hospital?

Keep the infant warm and give just enough oxygen via a head box to keep the tongue pink. Handle the infant as little as possible after starting an intravenous infusion of maintenance fluid (Neonatalyte). Carefully observe his respiration rate and pattern, colour, heart rate and temperature. The oxygen requirements are likely to increase over the first 48 to 72 hours. CPAP via nasal prongs is a very effective way of managing the respiratory distress in an infant with hyaline membrane disease. Ventilate via an endotracheal tube if the infant develops apnoea or remains cyanosed in 100% oxygen.

6. What is the best way to determine whether this infant is receiving the correct amount of oxygen?

By measuring his oxygen saturation with an oxygen saturation monitor.

CASE 2

A preterm infant with hyaline membrane disease is treated with head box oxygen in the intensive care unit of a level 2 hospital. On day 5 the respiratory distress becomes much worse and the amount of oxygen (FiO₂) has to be increased.

1. Give 3 important conditions that may complicate hyaline membrane disease on day 5.

Pneumonia, pneumothorax and a patent ductus arteriosus.

2. How would you diagnose a pneumothorax?

The chest may move poorly with decreased breath sounds on the side of the pneumothorax. An easily palpable liver suggests a right sided pneumothorax while poorly heard heart sounds suggest a left sided pneumothorax. However, the clinical diagnosis is difficult and transilluminating the chest is the quickest way to diagnose a pneumothorax. A chest X-ray will also confirm the diagnosis.

3. How is a pneumothorax treated?

Usually a chest drain must be inserted for a few days. In an emergency, the air in the pleural space can be aspirated with a syringe and needle while waiting for staff and equipment to insert a chest drain.

4. What clinical signs would suggest a patent ductus arteriosus?

A heart murmur and collapsing pulses (i.e. very easy to feel).

CASE 3

A 2900 g infant is delivered in a clinic and appears normal at birth. However at 30 minutes of age the infant has tachypnoea and mild central cyanosis. There is no meconium staining. The infant improves markedly in oxygen and is transferred to a level 1 hospital.

1. Does this infant have enough clinical signs to diagnose respiratory distress?

Yes, as the infant has 2 of the 4 important signs of respiratory distress, i.e. tachypnoea and central cyanosis in room air.

2. What is the probable cause of the respiratory distress?

Wet lung syndrome. The birth weight suggests that the infant is not preterm while the lack of meconium staining makes meconium aspiration unlikely. Congenital pneumonia cannot be excluded.

3. What test on the gastric aspirate after birth would help to diagnose congenital pneumonia?

A Gram stain showing pus cells and bacteria.

4. Why is a patent ductus arteriosus unlikely to be the cause of the respiratory distress in this infant?

Because a patent ductus arteriosus rarely causes respiratory distress in a term infant and usually does not present the first few days of life.

5. Should this infant be transferred to a level 1 or 2 hospital?

Yes. As the wet lung syndrome usually resolves in 48 hours, this infant need only be transferred to a level 1 hospital, provided that there are adequate facilities to give and monitor oxygen via a headbox. Careful observations are essential. The infant must be transferred to a level 2 or 3 hospital if the signs of respiratory distress become worse as this would suggest that the cause is not wet lung syndrome.

CASE 4

A 30 week infant has 3 apnoeic attacks on day 3. Clinically the infant is well with no signs of respiratory distress or infection. The infant is nursed in a closed incubator and fed by nasogastric tube.

1. What are the likely causes of the apnoea?

Apnoea of immaturity, big volume feeds, or the incubator temperature being too high.

2. What is apnoea of immaturity?

Apnoea which is common in healthy preterm infants, due to immaturity of the respiratory centre.

3. How do you differentiate apnoea from periodic breathing?

In periodic breathing the infant stops breathing for less than 20 seconds and does not develop bradycardia, cyanosis or pallor. The oxygen saturation does not drop.

4. How would you treat this infant?

Give the infant theophylline (Nuelin liquid) 5 mg/kg as a loading dose via a nasogastric tube then 2,5 mg/kg every 12 hours. The theophylline can usually be stopped when the infant reaches 1800 g or 35 weeks. Observe the infant carefully with an apnoea monitor.