Neonatal Cyanosis & Respiratory Distress

A baby is born at 32 /52’ gestation following a placental abruption. He requires bag/mask ventilation & following this, is noted to have marked sternal recession. An arterial blood gas shows high $PaCO_2$ & low PaO_2. A chest radiograph shows bilateral diffuse opacifications

1-Recognise the Presentation
2-Analyse the Presentation
3-Suggest a DDX
4-Manage the patient .

Common causes of Acute Respiratory Distress in Premature Infants.

1-Respiratory distress syndrome(RDS)

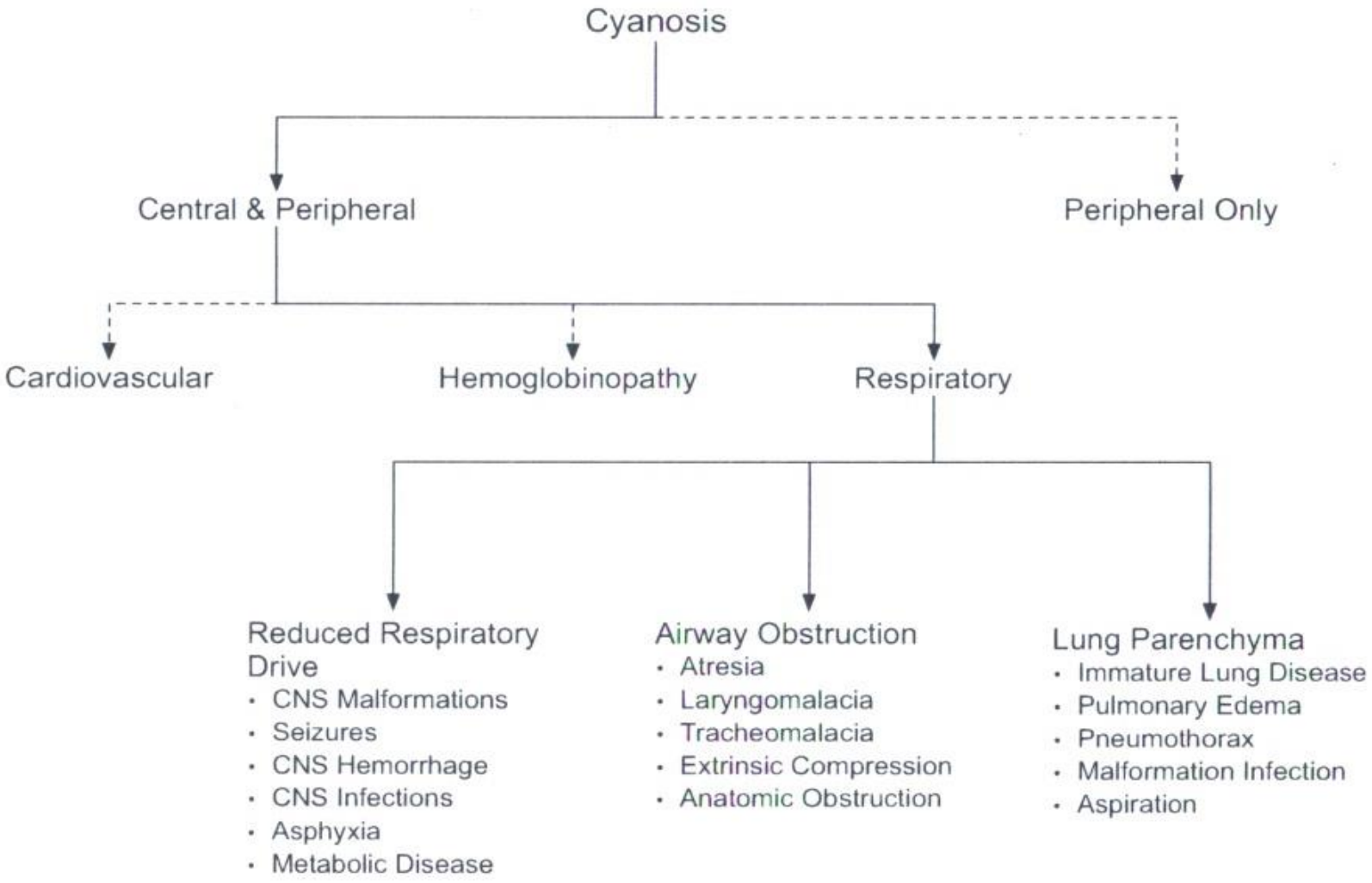
2-Pneumonia (congenital or acquired)

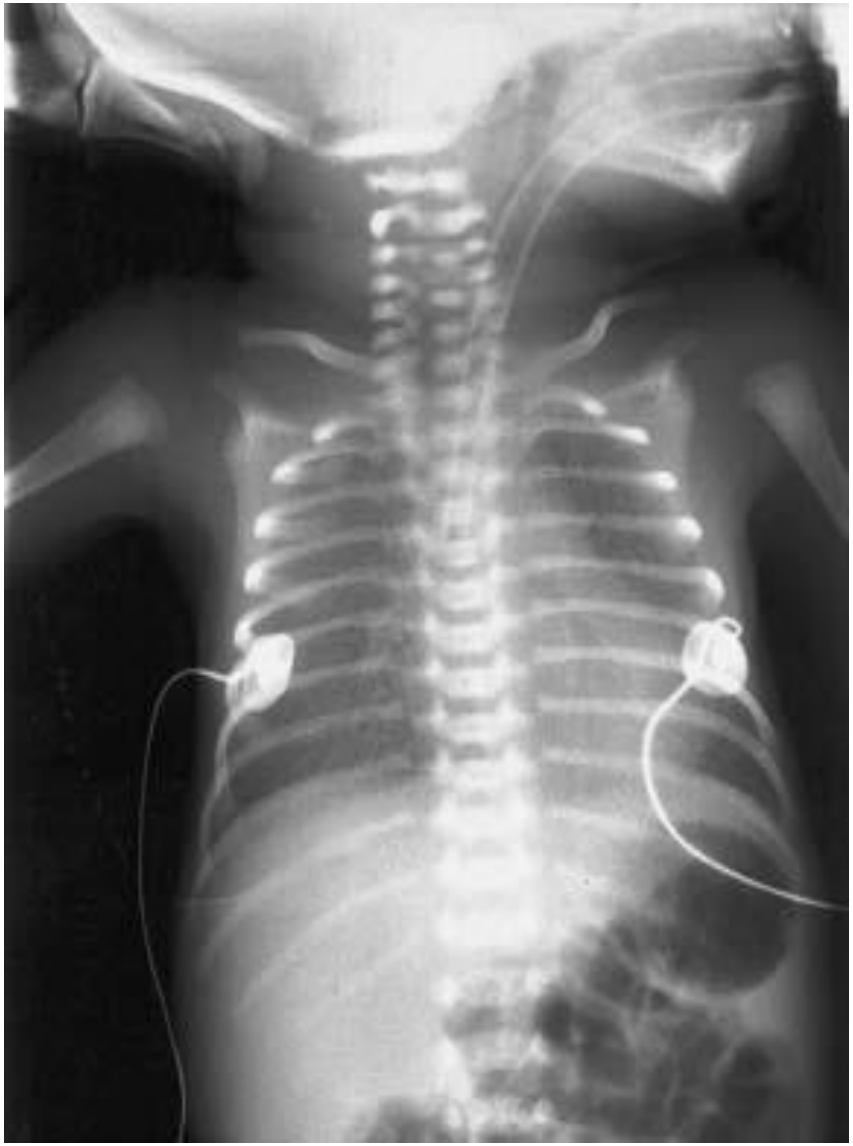
3-Pneumothorax

4-Surgical conditions (diaphragmatic hernia)

5-Cardiac causes

CYANOSIS IN THE NEWBORN: Respiratory





X-ray showing the features of respiratory distress syndrome (RDS)

The clinical features of respiratory distress syndrome (RDS) include the following:

*** Tachypnea**

*** Recession (subcostal, intercostal, sternal)**

***Cyanosis**

***Expiratory grunting.**

CXR is the best method to distinguish between the various causes & to make a definitive Dx.

RDS has a characteristic radiological appearance & an X-ray will distinguish it from a no. of other common causes.

Physical Exam.

-Cyanosis&/Or Resp. Distress starting within 4 hr. of birth DDX-

**TTN*

**Cong. Pneumonia*

**MAS*

**Cong. Lung anomalies*

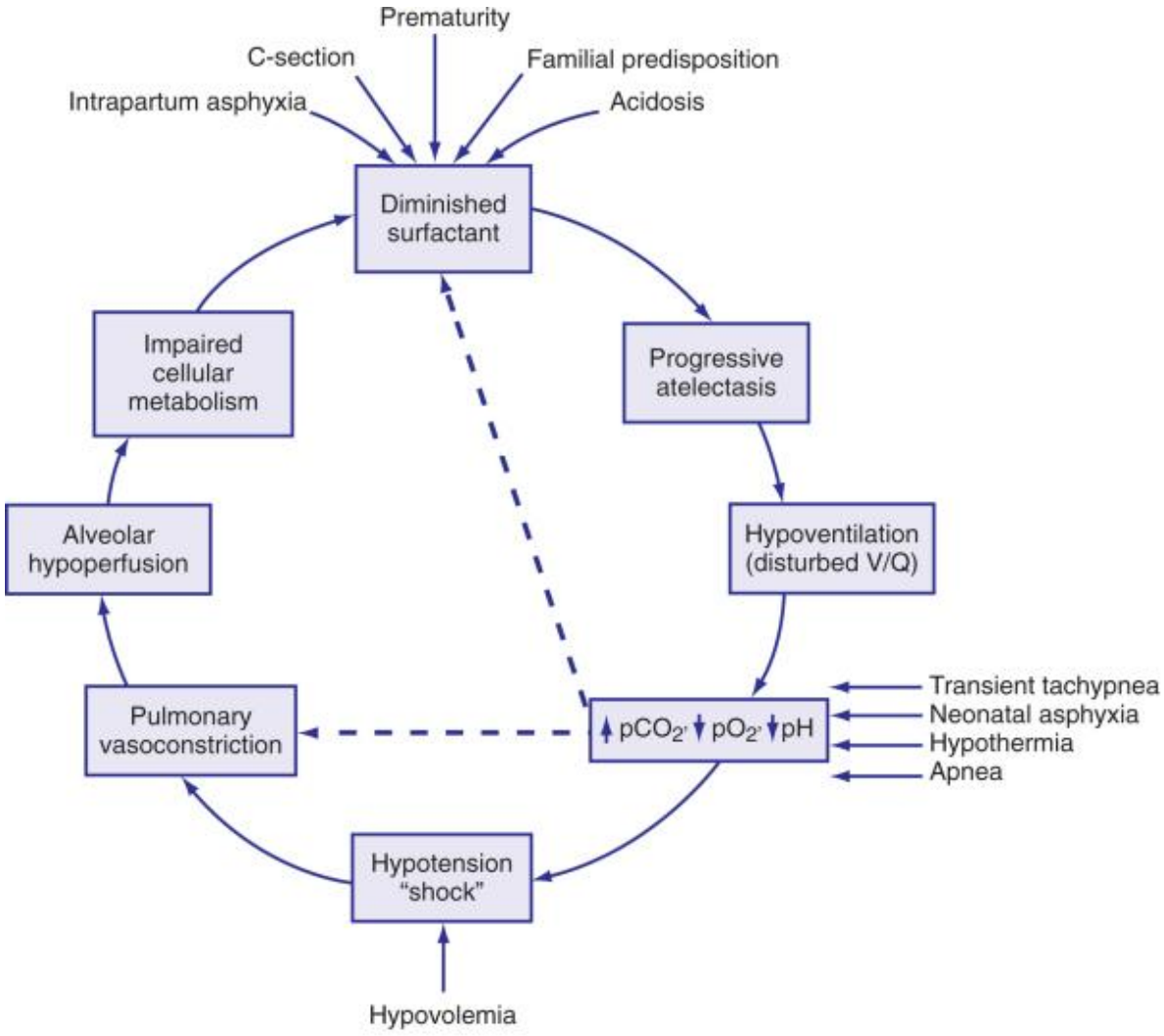
if no surfactant is given the condition deteriorates over 48-72 hr.

If baby SURVIVE ,THERE IS RESOLUTION& MARKED DIURESIS OVER NEXT 7 DAYS

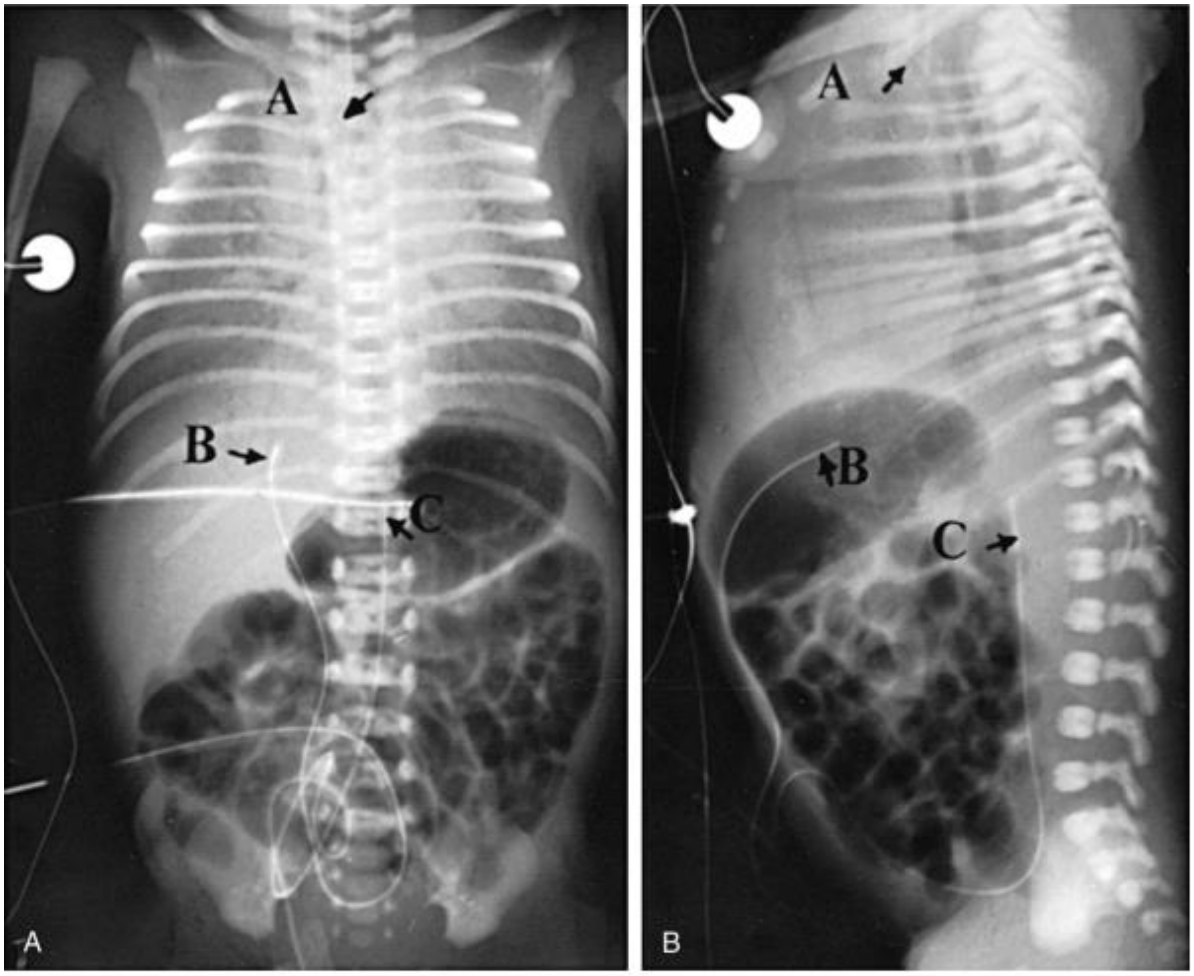
Specific causes of respiratory distress

Respiratory distress syndrome (RDS) has in the past been referred to as ***hyaline membrane disease (HMD)***, a condition recognized on *histological examination*

The fetal lung is a stiff structure as a result of lack of surfactant. At birth, the baby takes a deep breath which expands the lungs with air, but if surfactant is not present the stiff alveoli collapse down to their fetal size& the next breath is another massive inspiratory effort



Contributing factors in the pathogenesis of IRDS The potential "vicious circle" perpetuated hypoxia & pulmonary insufficiency.



Infant with RDS. Note the granular lungs, air bronchogram, &air-filled esophagus. AP *(A)* & lateral *(B)* CXR are needed to distinguish the umbilical artery from the vein catheter &to determine the appropriate level of insertion. B view clearly shows that the catheter has been inserted into an umbilical vein & is lying in the portal system of the liver. *A* is the endotracheal tube; *B* is the umbilical venous catheter at the junction of the umbilical vein, ductus venosus, &portal vein; *C* is the umbilical artery catheter passed up the aorta to T_{12}.

There is no specific clinical feature of RDS, but it is rare in mature infants &common in the severely premature baby. A major feature of RDS is recession,

Grunting is uncommon in premature infants & is mainly a feature of full-term babies.

Dx of RDS is confirmed by CXR which is usually diagnostic .It shows two characteristic features.

1 An air bronchogram (radiolucent air in the bronchi seen against the airless lung).

2 Ground glass appearance of the lung fields as a result of *alveoli collapse*

Investigations

1-CXR **(*Bilateral diffuse ground glass appearance (generalized atelactasis) airway bronchogram ,reduced lung volume).***

2-Continuous Pulse Oximetry Transcutaneous O_2 & CO_2

3-Frequent blood gas analysis(pH, PaO_2, $PaCO_2$)

Management depends on the Dx,

General principles of Mx include:

- ***Monitoring vital signs.***

•Babies with resp. distress are potentially if not actually very ill. Early deterioration may be detected by monitoring RR , HR & BP . Maintenance of normal BP is esp. important in avoiding cerebral complications.

- ***Monitoring blood gases.*** This is essential in all infants with resp. distress. It is only possible to determine what the appropriate **O_2** conc. is for an individual baby by measuring the arterial blood (*PaO_2) & titrating the inspired O_2* to maintain the arterial O_2 conc. in the normal range. The decision as to whether a baby requires resp. support is determined by the blood pH & by the arterial blood (*$PaCO_2$).*

***Respiratory support** Babies with deteriorating lung disease, esp. those who are very small &weak, require resp. support. This takes the form of either (CPAP) or (IPPV).

* ***Treat infection.*** The possibility of infection must always be considered. Blood cultures & AB are given until the cultures are known to be negative.

Management for this condition includes the following:

* ***Titration of the inspired O_2 level against partial*** (*PaO_2) in arterial blood. If the baby is* breathing spontaneously, the O_2 can be given via a headbox.

* ***Continuous positive airway pressure.(CPAP)*** Resp. support can be given in a spontaneously breathing baby by applying CPAP either via a face mask or a nasal prong. ***This technique maintains constant +ve distending pressure on expiration, which prevents alveolar collapse.***

* ***Mechanical ventilation.***

Very small babies will require immediate mechanical ventilation because they are too weak to breathe spontaneously. Larger babies who do not improve on CPAP, or those having severe apneic episodes, will also require mechanical ventilation (IPPV). ***Mechanical ventilation is applied by intubating the baby & connecting the ventilator to an endotracheal tube.***

* ***Administration of exogenous surfactant.***

Natural or synthetic surfactant. This must be given directly into the baby's lungs by instillation through an ETT . This therapy has ↓ the mortality of RDS by 40%.

Complications of IRDS

*Pneumothorax
*Pneumonia
*Intracranial hemorrhage
*Hydrocephalus
*Patent ductus arteriosus
*Necrotizing enterocolitis
*Retinopathy of prematurity
*Chronic lung disease
*Cerebral palsy

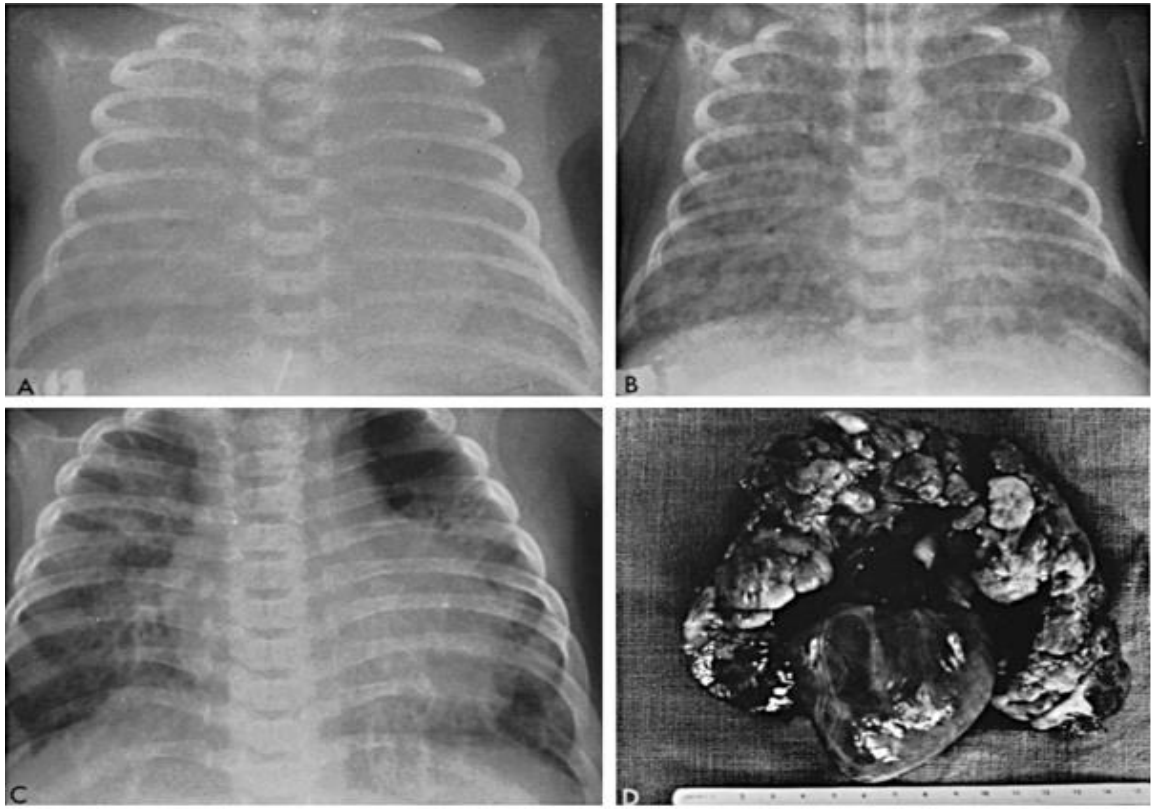
Bronchopulmonary dysplasia (BPD)

lung injury in infants ,requiring mechanical ventilation & supplemental O_2 . The use of antenatal steroids & postnatal surfactant ↓ its incidence & severity .

BPD is a disease primarily of infants <1,000 g born at less < 28 /52 , many of whom have little or no lung disease at birth, but develop progressive respiratory failure over the 1st few wks of life.

***It is inversely related to gestational age*. Instead of showing improvement on the 3rd–4th day, consistent with the natural course of RDS, some infants develop an ↑need for O_2& Ventilatory support. Resp. distress persists or worsens & is characterized by hypoxia, ↑CO_2, O_2 dependence, &, in severe cases, the development of right-sided HF**

Treatment includes: *Nutritional support, fluid restriction, drug therapy(Diuretic therapy , Inhaled bronchodilators , Cromolyn sodium, Methylxanthines,) maintenance of adequate oxygenation, &prompt Rx of infection .*



Pulmonary changes in infants treated with prolonged, IPPV with air containing 80–100% O_2 in the immediate postnatal period for the clinical syndrome of RDS .

***A,* A 5-day-old NN with nearly complete opacification of the lungs.**

***B,* A 13-day-old infant with "bubbly lungs"**

***C,* A 7-mo-old infant with irregular, dense strands in both lungs, hyperinflation ,&cardiomegaly suggestive of CLD ,**

***D,* Large RV & a cobbly, irregular aerated lung of an infant who died at 11 mo of age. This infant also had a PDA**

PDA is a common complication of infants who require mechanical ventilation. During **fetal life**, the DA shunts blood from R---L away from the unexpanded lungs. In premature infants with lung disease, particularly RDS, the ↑ pulmonary resistance & hypoxia makes it more likely that the ductus will reopen. In **neonatal life**, the direction of the blood shunt is reversed &is more likely to be L---R, so ↑ the work of breathing. This may make it difficult to wean the baby from the ventilator.

Clinical features **The baby has a systolic murmur & collapsing pulses .If the left to right shunt is large, the CXR shows plethoric lung fields. Dx is confirmed by echocardiography.**

Management Fluid restriction & diuretics may encourage the ductus to close. Indomethacin is a prostaglandin synthetase inhibitor & is used to close the ductus if standard treatment fails. **Rarely, surgical ligation** may be required.

Pneumonia **develops shortly after birth if infection is acquired from the mother during passage down the genital tract (perinatal infection). Group B β-HS is the causative organism. It may also occur in mechanically ventilated babies who acquire nosocomial infection from their carers.** ***Pseudomonas is the most likely infectious agent for late pneumonia.***

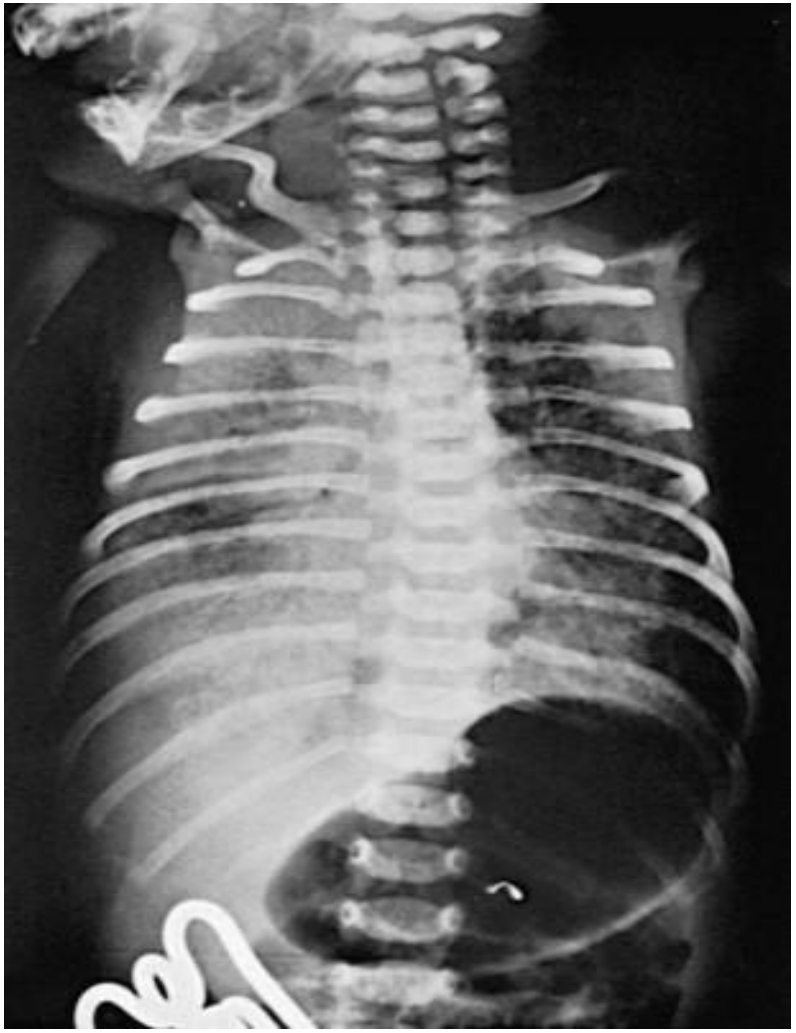
Clinical features The baby presents early with respiratory distress &shock. The CXR may show an identical appearance to that of RDS, so **pneumonia** must always be considered as a DDX in RDS. Group B β-HS is identified in blood cultures.

Pneumonia Must always be considered in any infant who deteriorates on mechanical ventilation.

Management An appropriate AB is curative if given early enough. Group B β-HS is mostly sensitive to penicillin. Supportive care is necessary to manage the circulation until the baby recovers.

Prognosis is good with early DX .

Death occurs in rapidly progressive cases



Fetal aspiration syndrome (aspiration pneumonia). Note the coarse granular pattern with irregular aeration typical of fetal distress from the aspiration of material contained in amniotic fluid, such as *vernix caseosa, epithelial cells, & meconium*

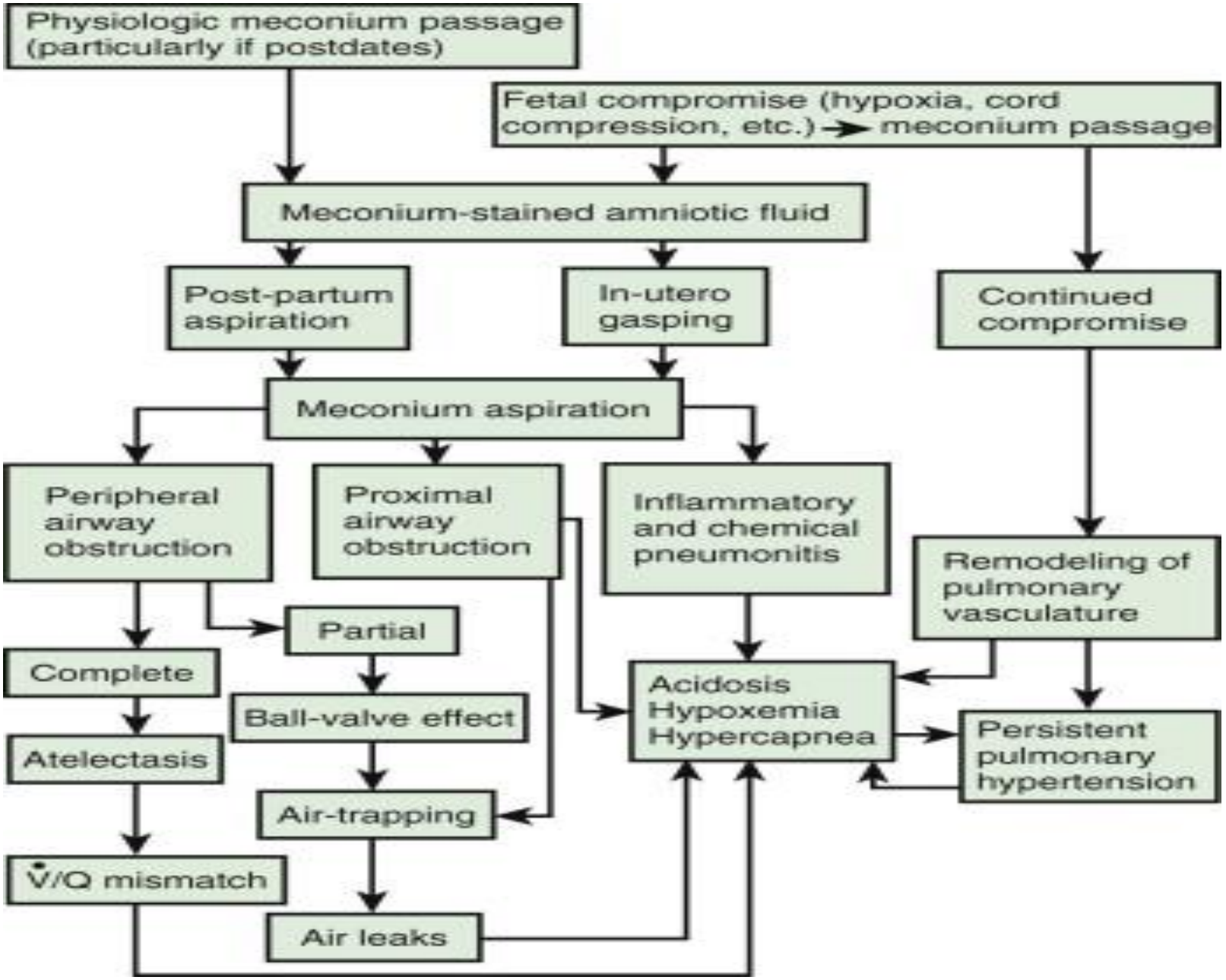
Meconium aspiration syndrome(MAS)

Acute or chronic hypoxia *in utero may cause the meconium* to be passed before birth. Infants may aspirate meconium either *in utero or at the time of delivery due* to the **deep gasping breaths** that occur from hypoxia. The majority of meconium aspiration is mild but in severe cases it causes marked respiratory distress.

Two forms of lung pathology can result:

- **Emphysema** resulting from partial obstruction of the airway causing a **ball valve effect.**
- **Atelectasis** resulting from total obstruction of the airway.

Both are usually present in the same case making the Mx of these babies difficult.



Pathophysiology of meconium passage & meconium aspiration syndrome.

Initial management of MAS

***Consists of nursing the baby in high-inspired O2**

to ↓the hypoxia caused by (V/Q) mismatching & to prevent the development of PPHN. Higher pCO_2 levels are acceptable in an effort to avoid intubation & ventilation.

***If a baby needs ventilation** then sedation &/or paralysis should be used to enable the use of slow rates & long inspiratory/expiratory times.

***Meconium inactivates surfactant** & frequent dosing with surfactant has been shown to be helpful. If a baby is failing, standard ventilation high-frequency oscillatory ventilation can be used, but is often unsuccessful due to the non-homogeneous nature of MAS.

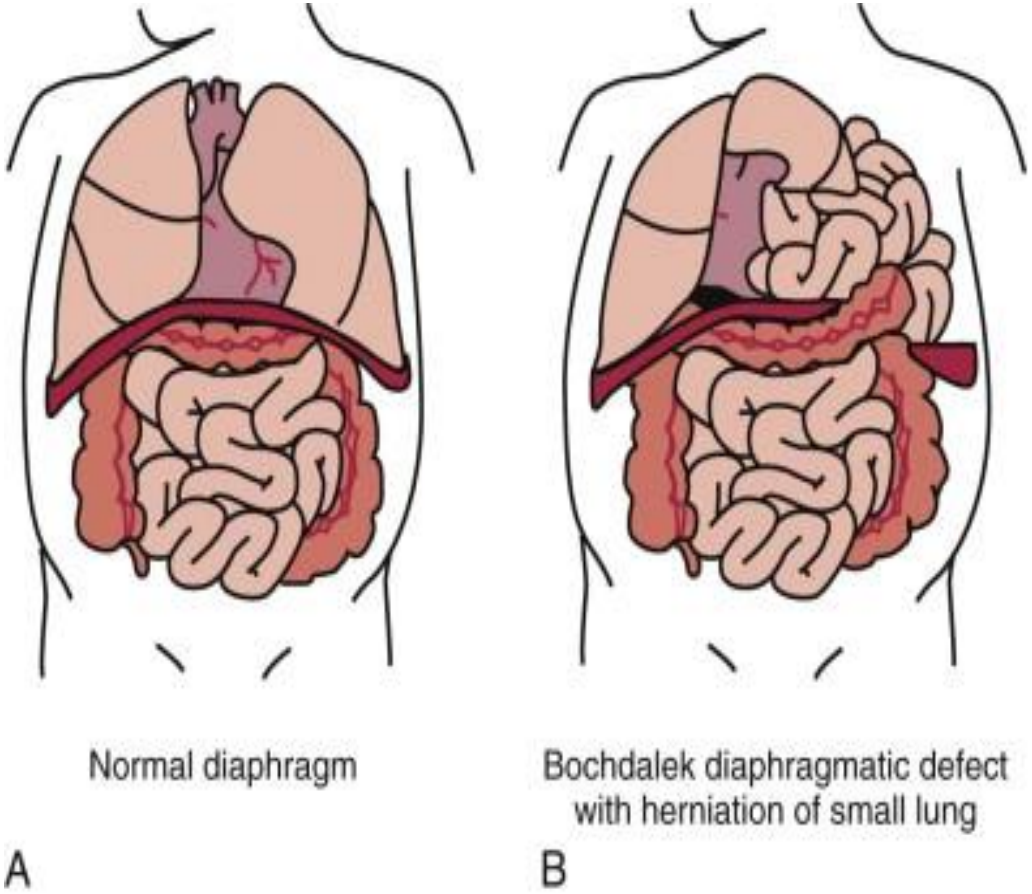
***Nitric oxide** may ↓ the need for (ECMO). If a baby does require ECMO the survival in most units is 90%, so transfer to an ECMO centre should be considered early in the NN failing conventional therapy

Diaphragmatic hernia

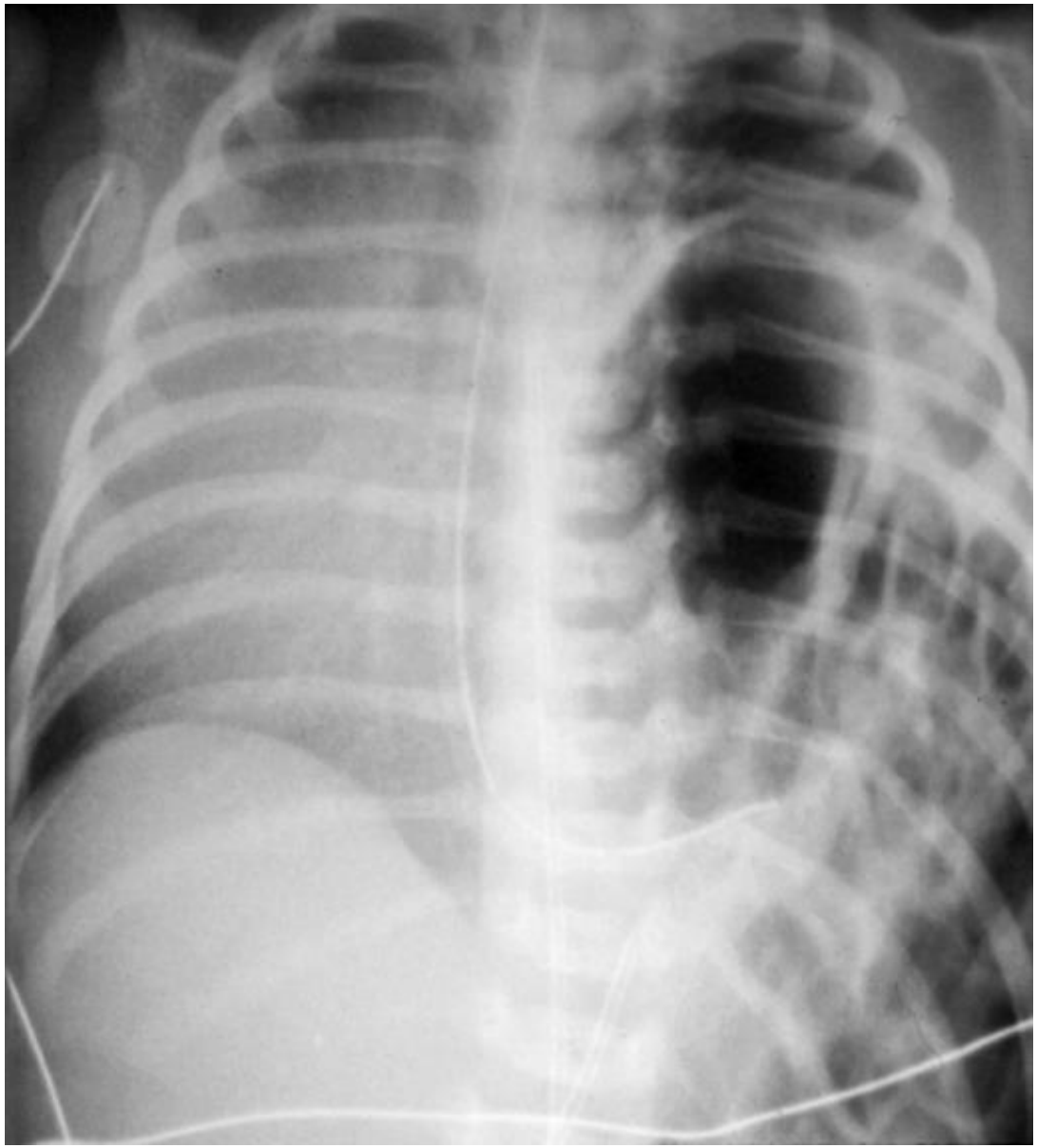
This is a congenital defect in the left hemi-diaphragm that allows bowel to herniate into the Lt chest with compression of the lungs & deviation of the heart to the Rt.

If it occurs early in pregnancy, severe lung hypoplasia develops as a result of the compression &the baby dies rapidly in the NN period.

Mx is directed towards resp. support, &surgical repair of the hernia is performed once the lung is stabilized.



A, **A normal diaphragm separating the abdominal and thoracic cavity.**
B, **Diaphragmatic hernia with a small lung and abdominal contents in the thoracic cavity**



This CXR shows a stomach, NGT , & small bowel contents in the thoracic cavity consistent with a congenital diaphragmatic hernia (CDH).

A neonate with respiratory distress must be treated with antibiotics.

- Early surfactant use is advantageous in IRDS.
- Severe MAS should be referred to an ECMO centre.

Neonatal Cyanosis & Respiratory Distress

Objectvies:

Key Objective(s)

Differentiate between peripheral & central cyanosis ,generalized cyanosis is more consistent with primary heart or lung disease, then distinguish between them .

Explain that if the process causing peripheral cyanosis is severe enough (e.g., sepsis), generalized cyanosis may occur.

Objectives:

Through efficient, focused, data gathering:

1-Elicit maternal Hx of illness or sepsis in pregnancy, GA , delivery complications, presence of meconium, suction of infant, Apgar score, family Hx of CHD

2-Determine the vital signs, age of infant (PDA usually closes by 3rd day), whether the infant is alert & active, if infant is able to feed, & the presence of resp. distress (tachypnea, grunting, flaring,retracting).

3-Perform P/E of the NN for evidence of resp. distress, CHF or shock, signs of CNS depression, whether the cyanosis is central or peripheral .

4-Select appropriate investigations including diagnostic imaging, ECG, & blood tests.

5- Explain the hyperoxia test (arterial blood gas from a site distal to the PDA on room air & 100% oxygen).

6-Conduct an effective plan of Mx for a NN with cyanosis/hypoxia

7-Outline initial Mx including cardio-respiratory monitoring.

8-Select premature infants with hemodynamically significant PDA to refer for NSAID therapy