NEONATAL RESPIRATORY DISORDERS

Khalid Altirkawi, MD Notes by: Dania Alkelabi

Assistant Professor, Pediatrics
College of medicine
King Saud University
Riyadh, KSA

2017

Disclaimer

This presentation is to help medical students upon the start of their rotation in Pediatrics. It is NOT to replace the recommended textbook.

Khalid Altirkawi, M.D.

Objectives

By the end of this presentation, the student should be able to:

- Recognize the **SIGNS** of respiratory dysfunction in the neonate
- Describe the **MECHANISMS** underlying the respiratory illnesses discussed
- Mention the steps to **DIAGNOSE** and the strategies used to **MANAGE** these illnesses

Respiratory Signs

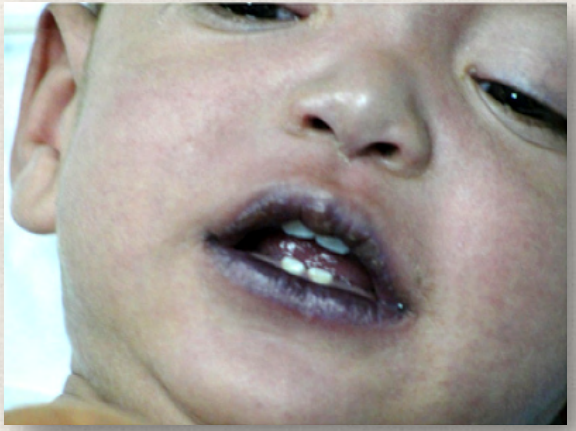
- Cyanosis: peripheral and central
- Tachypnea:
  - Normal RR for neonates (first 28 days): 40-60 breaths/minute
  - RR decreases with age
  - Grunting: a sound produced by baby during expiration because of partially closed airway between vocal cords (the baby tries to increase pressure at the end of the airway to prevent alveoli collapse at the end of expiration "natural PEEP, or natural Positive End Expiratory Pressure"), grunting is most commonly seen in RDS and surfactant deficiency
- Nasal flaring
- Chest recessions
- Feeding difficulties

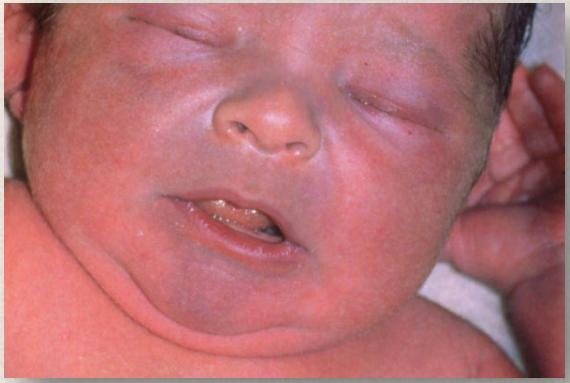
Central Cyanosis

- Affects lips and mucous membranes
- Most likely babies with central cyanosis has very low O2 saturation

Causes of central cyanosis:

- **Respiratory insufficiency**
- **Cyanotic cardiac disease**
- **CNS depression** (when respiratory center in brainstem is affected babies may have shallow breathing thus not have enough O2)
- **PPHN** (Persistent Pulmonary Hypertension), no enough oxygen because of blood shunting
- **Sepsis** - sepsis has very wide presentations (eg. reduced activity, vomiting, tachypnea or cyanosis) so **whenever you see any abnormal finding in neonates you should do septic work up and start empiric antibiotics waiting for result of culture** (for 2-3 days) if negative then stop antibiotics

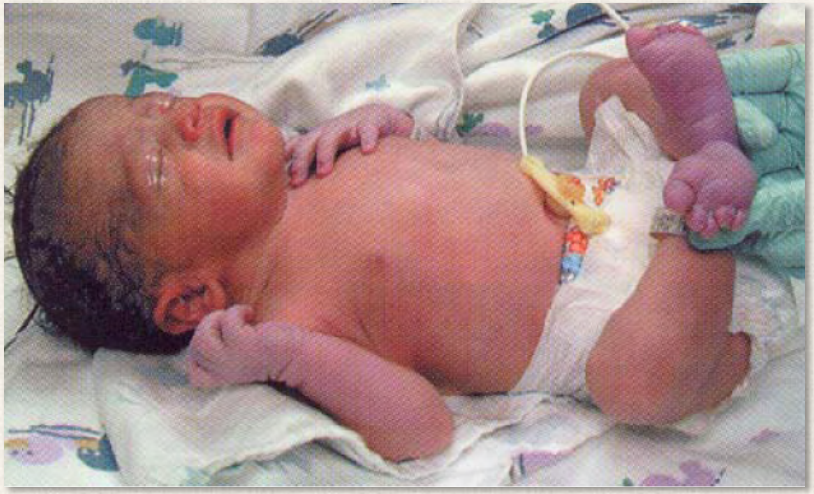


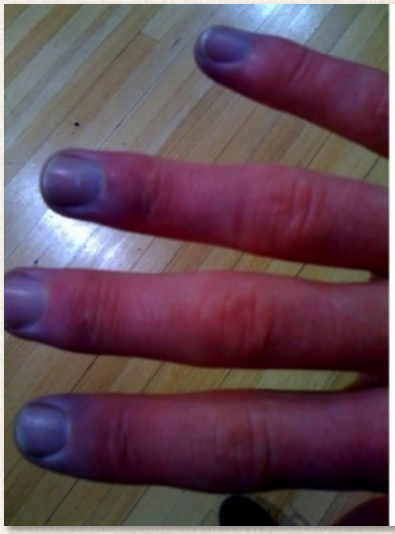


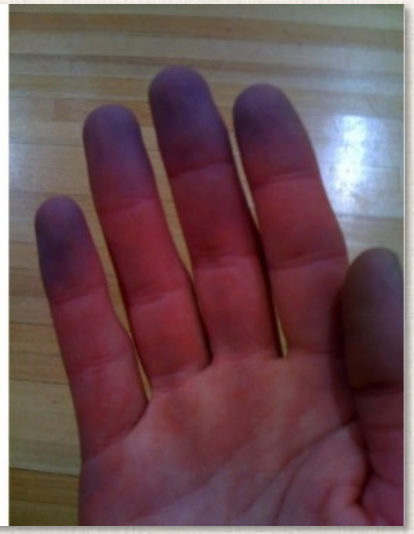
Peripheral Cyanosis

- Local phenomenon
- May take several days to resolve
- No special investigations
- No special therapy

- We care more of central cyanosis because peripheral cyanosis can be normal at the first few hours of life ومو بالضرورة يدل على إن الاكسجين يتناقص
- example: after 5 minutes of delivery the baby developed peripheral cyanosis and his O2 sat is 95%, your management: No need to do any action, it is acceptable. No need to give oxygen







Hyaline Membrane Disease (HMD) (old name)

Respiratory Distress Syndrome (RDS) the new name

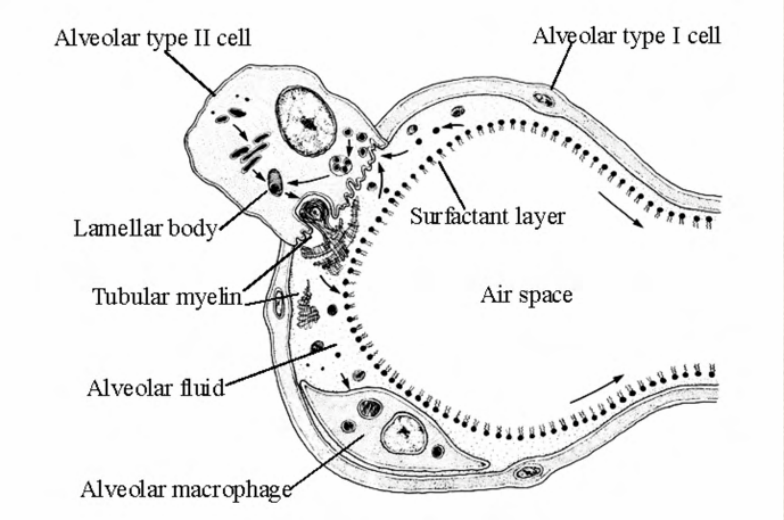
RDS

- Course: 3-4 days (must be admitted to ICU)
- Prevention: antenatal corticosteroids to mother that will deliver premature babies , control of maternal diabetes (risk factor RDS even of mature baby)
- Diagnosis:
  - ***Clinical signs:***

    Grunting, Retractions (intercostal, subcostal, and substernal depression during breathing تدخل على جوا), Nasal flaring يحاول يوسع لدخول الهواء, Cyanosis
  - ***Radiographic signs:***

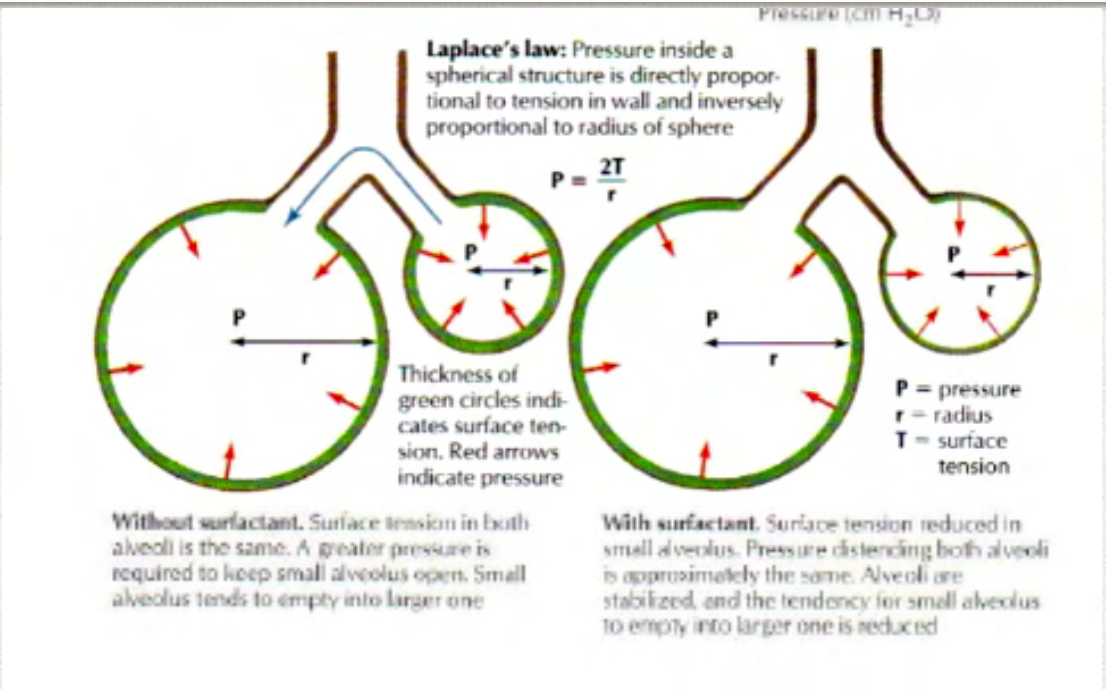
    Diffuse, Ground-glass opacities, Air bronchogram, Low lung volumes (if not ventilated)
    - These three signs in X-ray are important to differentiate RDS from TTN (transient tachypnea in newborn).
    - **Low lung volume is the most important sign to differentiate RDS from other disease** (less than eight to nine ribs)

Alviolus structure



- Surfactants are important to maintain alveolar volume at the end of expiration
- If alveoli are collapsed then lung has less volume
- Premature babies have less surfactant (the more premature -as 24-26 weeks- the less surfactant, in comparison to babies with born with older gestational age (above 30-34 weeks).
- If alveolar space is collapsed during expiration, the baby needs too much effort to open it. If the baby doesn't have enough surfactant he will start grunting (increasing pressure at larynx) to maintain alveolar volume at end of expiration

Laplace's Law



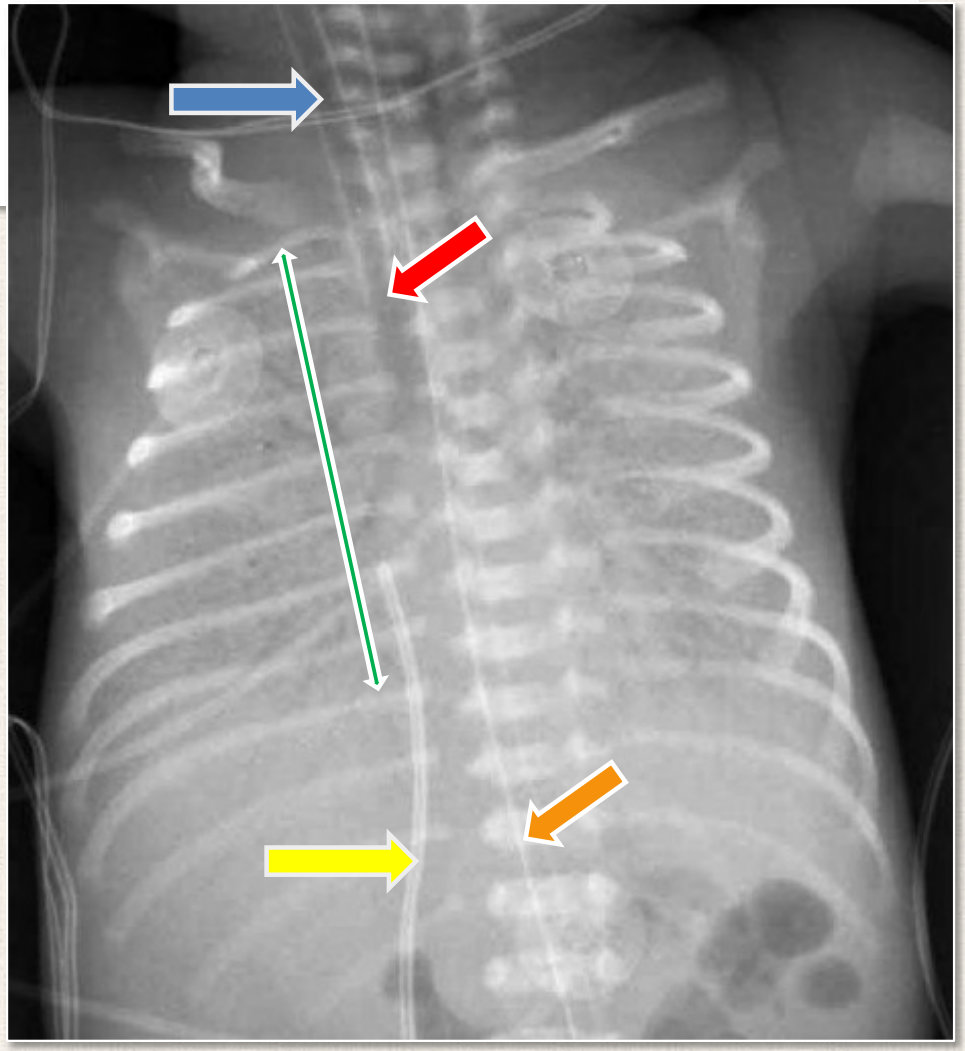
- Laplace law indicates that if there is two alveoli one big and one small then the pressure will go to the big one, this can lead to problems to the baby

RDS

PA view of chest radiograph of an infant with RDS.

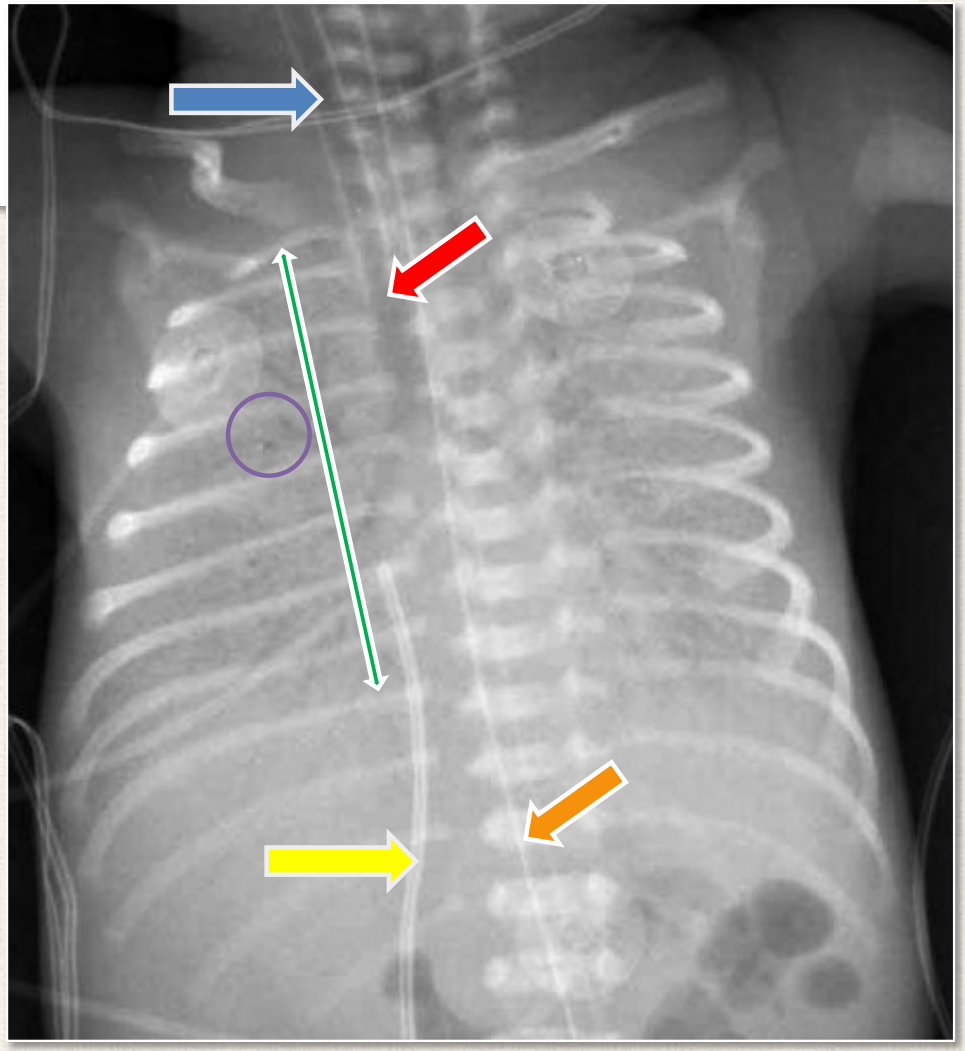
Notice the following:

- Air bronchogram (**Red**)
- Ground glass appearance e of both lungs
- Decreased lung expansion (**Green**)
- Endotracheal tube (**Blue**)
- Umbilical venous catheter (**Yellow**)
- Umbilical arterial catheter (**Orange**)



RDS

- This is a typical x-ray of RDS, lungs here are more whitish because alveoli are collapsed, no air inside alveoli (normal is black), black dots (purple circle) represent air inside alveoli… but many alveoli are collapsed and cause the lungs to appear whitish, this is called ground glass or الزجاج المغشّى which you can't see clearly through it
- Lung volume is decreased because alveoli are collapsed (if you count the number of ribs less than eight)
- Trachea and bronchi are very clear (black) because the the lungs around them is whitish resulting in air bronchogram (clear bronchi)

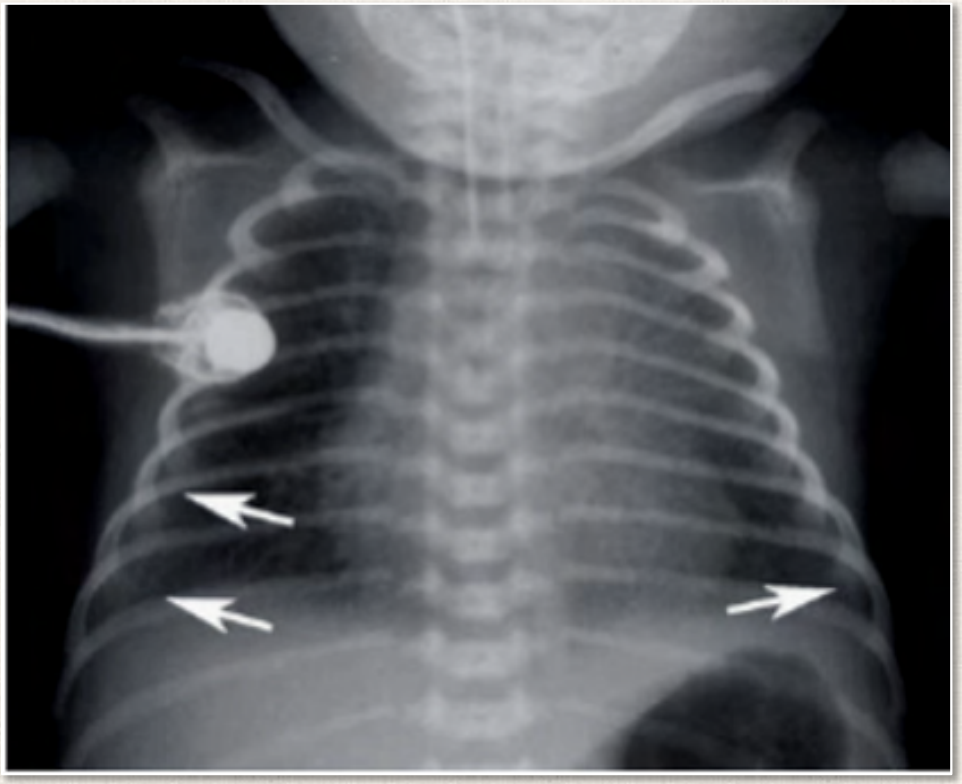


GBS group B streptococcus pneumonia

Chest radiography of infant with congenital pneumonia.

Virtually indistinguishable from one of RDS patients

- Confused with RDS because both of them have diffuse lung whitish opacities
- These patients have pneumonia following sepsis with GBS (coming from colonized mother and transferred to child) leading to *early onset sepsis* (first three days of life) presenting with respiratory symptoms (pneumonia)
- That's why **any baby with RDS is started on antibiotics!** Because we don't want to miss any sepsis induced pneumonia (bad disease) and can cause shock and kill baby within few hours



General note: If sepsis is *late onset* (after 3 days) we suspect blood stream infection or meningitis

Transient Tachypnea of the Newborn

Chest radiograph of an infant with TTNB.

Notice the fluids in the fissure (Red arrows) Confused with RDS sometimes

We differentiate by two signs:

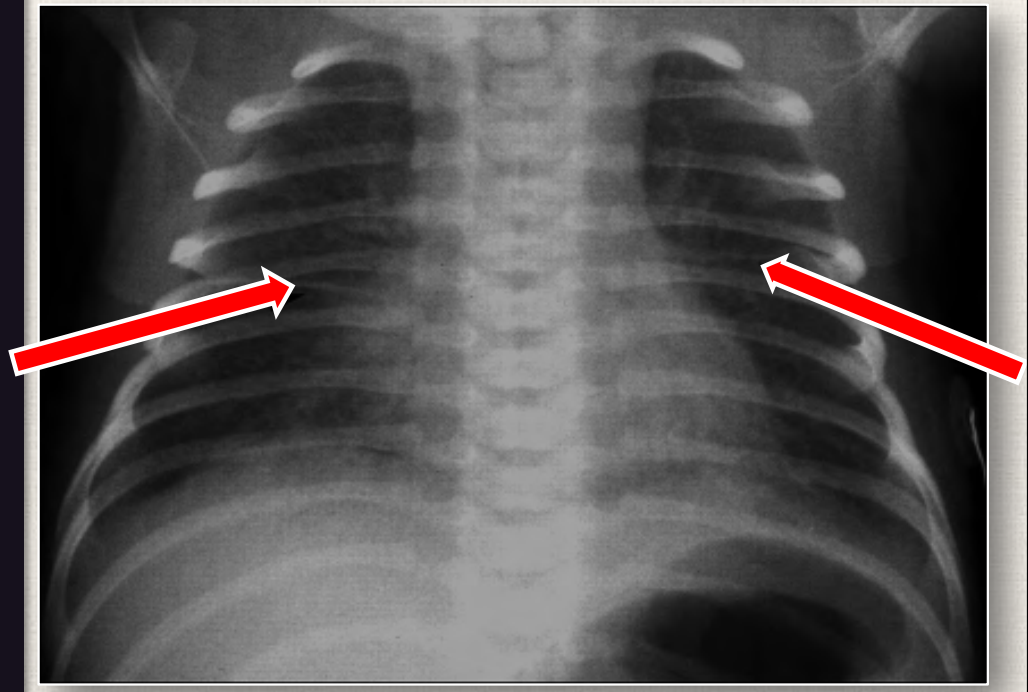
1- lung volume in TTNB is normal **(most important sign)**

2- lung fissure filled with fluid (whitish)

- TTNB is common in more mature babies (close to term), the baby will not be as sick as RDS

- The cause of TTNB is the remnant fluid in lung (usually babies get rid of most fluid inside their lungs during delivery)

- More common in babies delivered by cesarean section



RDS Treatment

- Not all cases need surfactant
- First: keep alveoli open by nasal CPAP + give O2
- Then if RDS is severe (require >30% O2 on CPAP) consider administering exogenous intratracheal surfactant
  - Lowers ***surface tension*** at air-fluid interface
  - Improves ***oxygenation*** and increases FRC at lower airway pressures
  - Single treatment is enough for most newborns because type II pneumocytes ***recycle*** surfactant
  - Second dose may be needed in > 6 hours if surfactant ***inhibition*** occurs (e.g. in MAS)
- How to give surfactant? There are many ways available including intubation, small catheter (Elisa), and minimally invasive approach

Meconium Aspiration Syndrome (MAS)

- Fetus is surrounded by embryonic fluid, this fluid goes inside lungs
- Babies usually pass meconium in first days of life (after birth), but if they pass it before birth they might aspirate it into respiratory tract
- Doctors in delivery room will call if they noticed the amniotic fluid (liquor) stained by color of meconium
- If the baby developed respiratory distress and with history of meconium staining then most likely this is a case of meconium aspiration syndrome, admit to NICU
- Difficult case and have significant mortality rates
- May present with pulmonary hypertension
- Need high support (mechanical ventilation, nitric oxide administration)

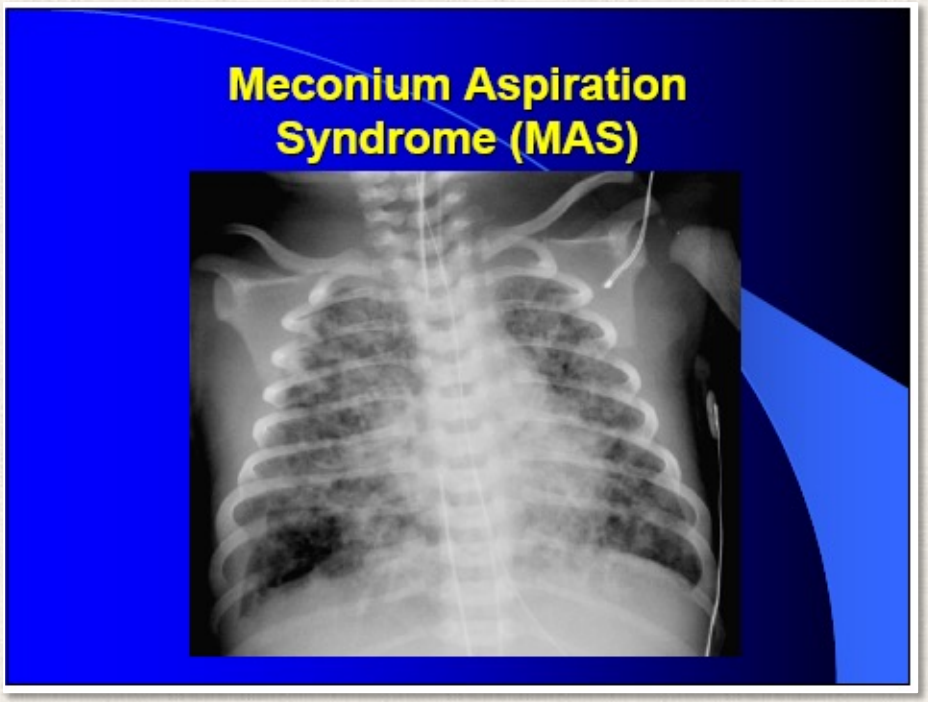
Meconium Aspiration Syndrome

Chest radiograph of an infant with MAS

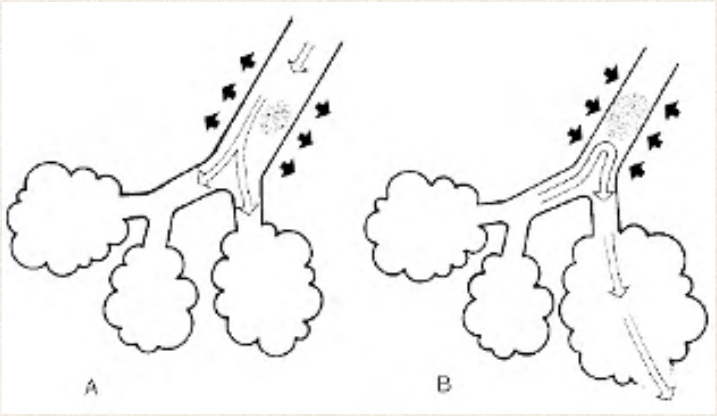
Notice the bilateral **patchy** opacities

Black areas are hyper inflated whereas whitish areas are collapsed, why?

- Because of partial obstruction (forming one way valve like effect)
- Whitish collapsed areas may be completely obstructed so no gas inside
- Or Sometimes meconium is very distant in the airway (beside alveoli) and meconium deactivates surfactant



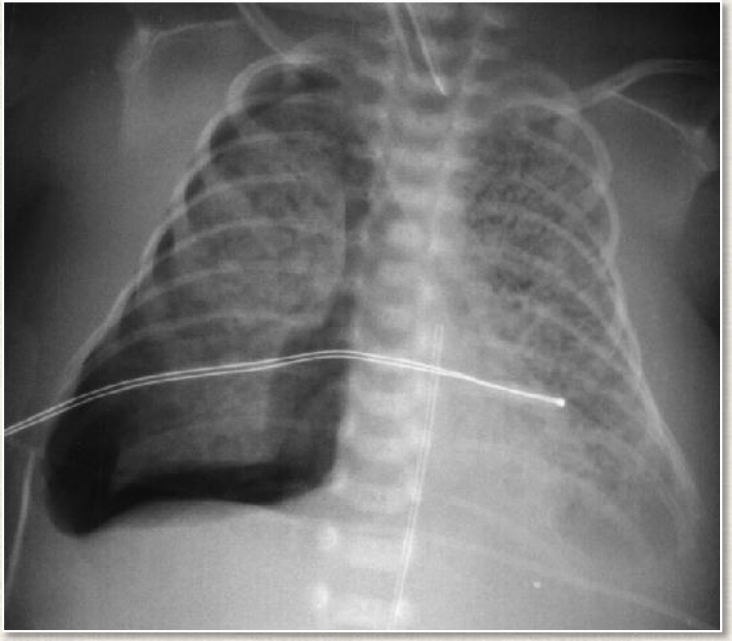
One-way valve Mechanism



- during inspiration airways are bigger (active pressure) so meconium can reach alveoli
- While during expiration the airway narrows (passive pressure) so there is no chance for the meconium to go out
- Air trapped inside causing hyperinflation of alveoli (black areas on x-ray), and sometimes rupture causing pneumothorax
- Management: good oxygenation and CPAP make airway big enough to allow ,eco imm to go out with expiration, also CPAP keeps alveoli open at end of expiration… n severe cases we might give surfactant (because inactivated surfactant by meconium) make lungs behave as same as if it was premature
- In severe cases it is associated with pulmonary hypertension thus nitric oxide given to dilate pulmonary vasculature

Pneumothorax

- Asymptomatic (1-2% of all newborn infants)
- Respiratory distress signs with/without CVS compromise
- Drainage of the pleural air by chest tube under water seal.
- Observation if no CVS compromise.
- You can find pneumothorax as incidental finding (asymptomatic) and usually that's spontaneous pneumothorax
- X-ray: collapsed lungs and a absent lung marking at periphery
- Pneumothorax is a result of RDS for this X-ray! Because look for signs of RDS (ground glass appearance (right lung), air bronchogram…)
- Clinical signs of pneumothorax: sudden deterioration and desaturation, bradycardia (in tension pneumothorax)
- Management: needle decompression (2nd intercostal space) then chest tube



Causes of sudden desaturation: mnemonic (DOPI):

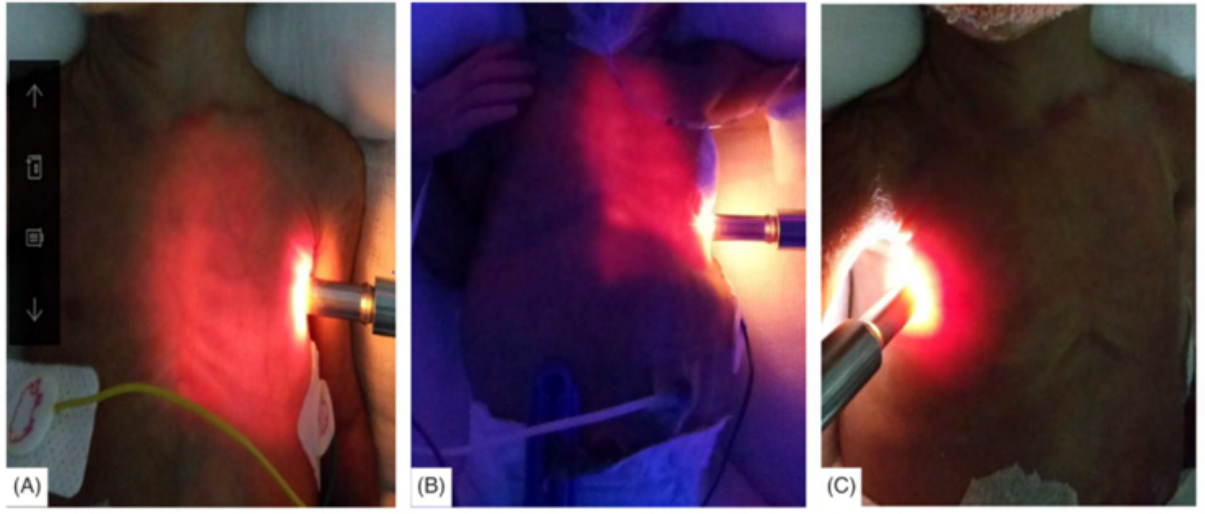
D: displacement of tube (tube out or tube very deep)

O: obstruction of tube (as by secretion)

P: pneumothorax

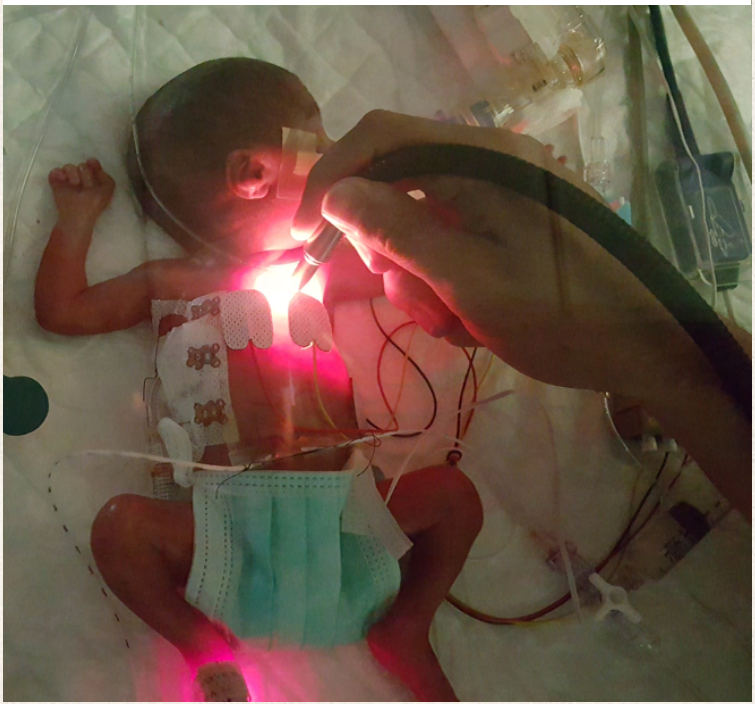
E: equipment failure

Transillumination

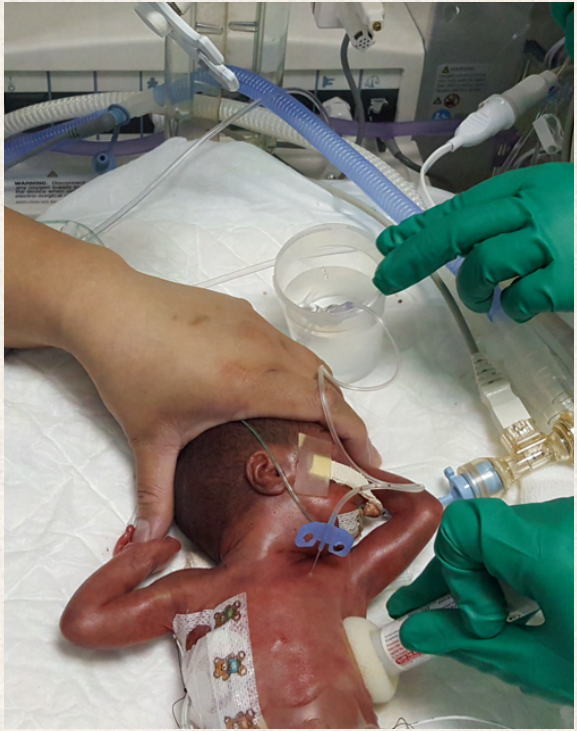


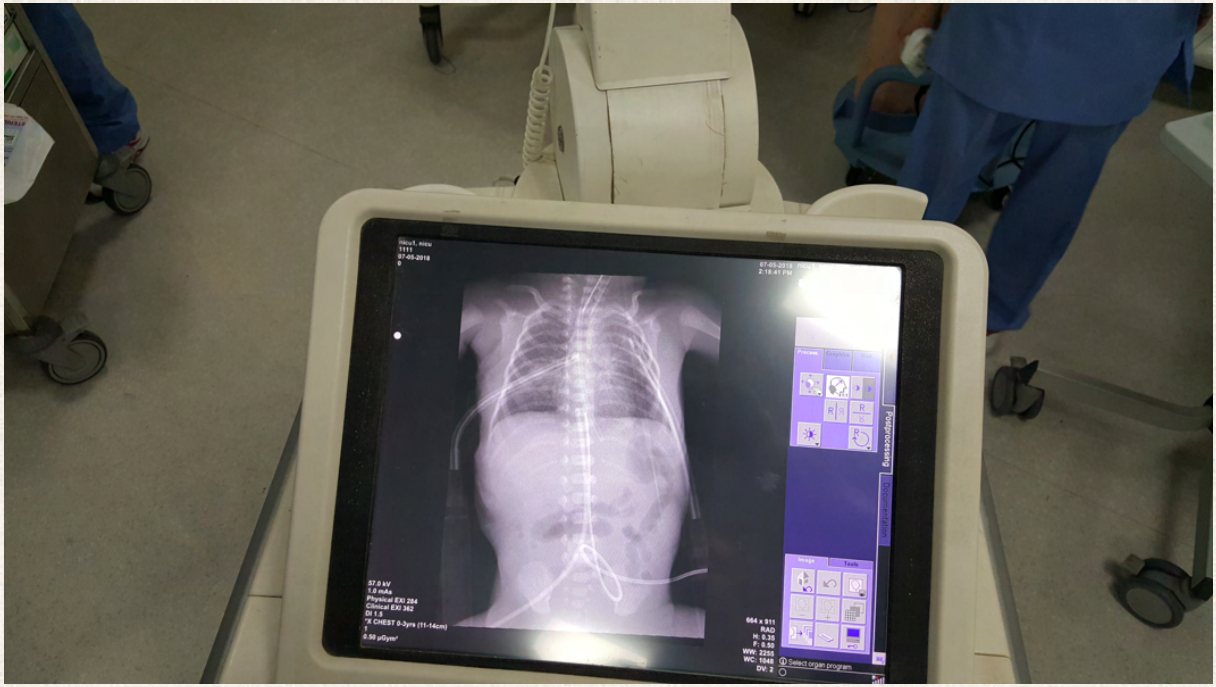
- Photo (A,B) show left side pneumothorax (light all over left side) while picture (C) is normal
- Transillumination is a quick way to check (more quick than x-ray, you need every minute to save the baby), just make the room dark and put light, you should put needle decompression as soon as possible until chest tube is ready
- If you suspect pneumothorax but transillumination is not available then TREAT IMMEDIATELY DO NOT wait for x-ray to prevent brain damage
- transillumination only works for neonates (especially premature), but you cannot use this in adults and sometimes also not work in term babies (fat is thick, you cannot see through)

Transillumination +ve so treat immediately



Needle aspiration





Don’t wait for CXRay to confirm pneumothorax in critical patient, here x-ray arrived after needle decompression done

Congenital Diaphragmatic Herina (CDH)

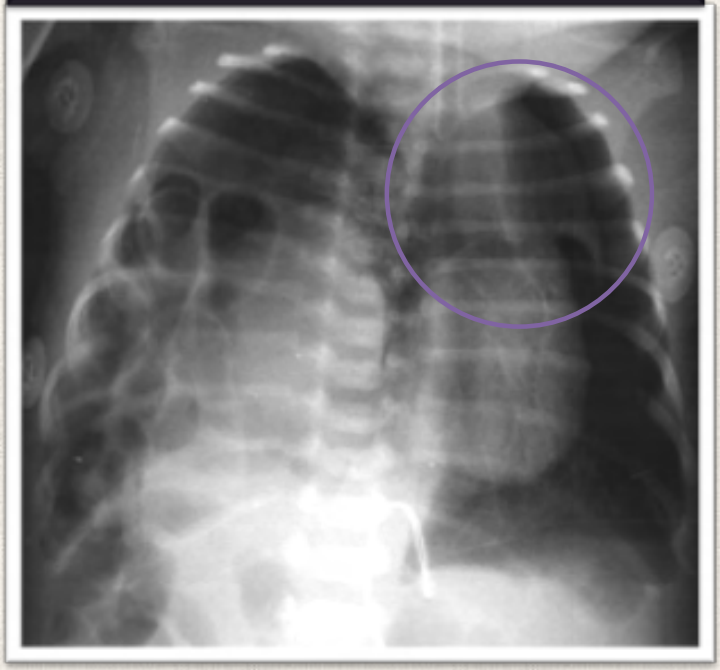
- Difficult to manage with high mortality and long ICU stay

Diaphragmatic Hernia

- Congenital vs. acquired
- Most often left (85%) so heart pushed to right side and through the poster-lateral segment of diaphragm.
- Respiratory Distress (usually severe), cyanosis, bradycardia, scaphoid abdomen
- Diagnosis: signs and imaging
- Management: stabilization then surgery
- If seen in prenatal ultrasound then IMMEDIATE intubation and mechanical ventilation, and NGT to decompress stomach, and admit to NICU
- If not noticed in prenatal ultrasound: after birth baby have scaphoid empty abdomen, chest diameter increased, heart sounds found in right side (if pushed from hernia on left side but if right side hernia heart sounds on left as normal), then same incubate and NGT and transfer to NICU

Diaphragmatic Hernia

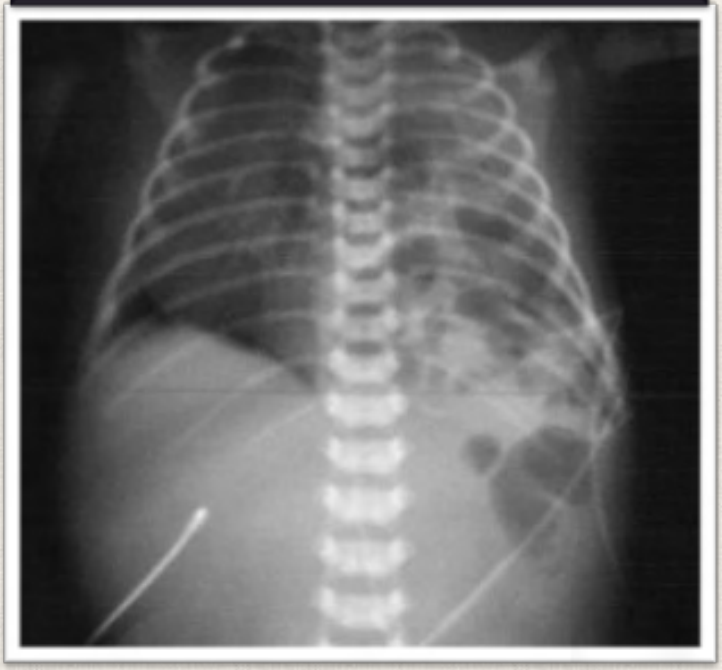
Right



Worse than left side, gas presence around thymus (sail sign الشراع)

Thymus usually is in center of mediastinum (it's the reason mediastinum is big in neonates) but here it's pushed

Left



No gas in abdomen, Intestinal loops in chest, pushed heart

In premature babies, when we use mechanical ventilation it can cause complication to the lung that make the baby need oxygen for longer time and complicate his course of treatment
(we call this chronic lung disease or bronchopulmonary dysplasia)

Chronic lung disease (CLD)

Broncho-pulmonary Dysplasia (BPD)

BPD

- Lung injury due to:
  - Barotrauma **the most serious** (pressure-related) when we give high pressure by mechanical ventilation and cause over stretch of the lung
  - Volutrauma (volume/expansion-related)
  - Oxygen toxicity too much O2 can damage lung, and cause retinopathy of prematurity, and damage brain and other tissues (O2 free radical injury)
- Defined by the need for oxygen therapy or respiratory support at 36 weeks post-menstrual age (PMA)*
- Management options ??? No definite treatment, each doctor approach it differently, some will support by CPAP others will give systemic steroids (especially if we are not able to extubate) or diuretics

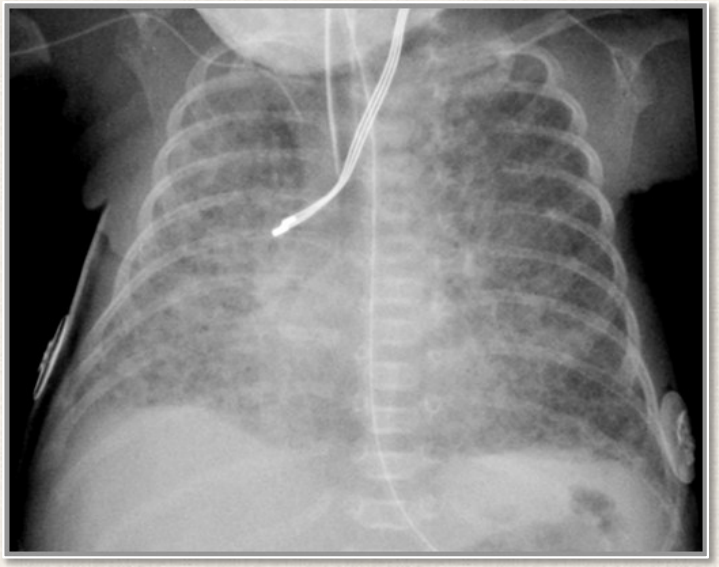
* in premature babies there is a way to calculate the "corrected age" called PMA

BPD

Chest radiograph of an infant with BPD

Notice the widespread haziness of both lung fields alternating with hyper-lucent areas.

- It looks like early stage of RDS but it is completely different, the whitish spots are fibrosis, if you did CT will see too much damage
- Black areas are hyperinflation



Apnea of prematurity (AOP)

Causes of Apnea in general

- Prematurity/immaturity center of breathing in medulla is not matured yet (if <34 weeks)
- Hypoglycemia (measure, give sugars to baby if low, will improve)
- Drugs (phenobarbital… etc)
- Seizures (sometimes the only sign of seizure in babies is apnea)
- CNS injury
- Sepsis, sepsis, sepsis!!! As we said before, think of sepsis if you find respiratory symptoms in first 3 days of life, give antibiotics waiting for culture, if all cultures negative within 2-4 days then stop antibiotics (might be another cause, look for it)
- ……
- Periodic breathing (not a true apnea) it is normal, the baby breaths very fast then stop then breaths back again (don't persist for more than 20 seconds), **not associated with bradycardia or desaturation** (هذا فرقها عن apnea of prematurity)

Apnea Of Prematurity

- Cessation of respiration for 20 seconds, or for 15 seconds associated with cyanosis, pallor or bradycardia
- Respiratory drive in preterm infants is
  - Less developed in response to hypercarbia
  - Transiently increased then decreased by hypoxia
- Preterm infants are at 3-4 increased risk of SIDS than term infants

AOP

- More common during sleep
- Uncommon if birth after 34 weeks of gestation (if happens after 34wks we give caffeine to stimulate respiratory center)
- May persist in VLBW infants until 44 weeks postmenstrual age.
- May recur following general anesthesia:
  - Preterms < 44 weeks PMA who receive GA require 24 hour monitoring

Types of AOP

- Central apnea
  - Lack of respiratory drive and effort, Typically brief
- Obstructive apnea
  - Presence of central drive and respiratory efforts
  - Cessation of airflow due to airway obstruction gets exhausted and stop
- Mixed apnea
  - Most common, Can be quite prolonged
  - Central apnea is a response to hypoxia of obstructive apnea
  - Both airway and respiratory center affected, even changing head position of baby can narrow his small airway

Treatment of severe AOP

- Methylxanthine drugs (e.g. Caffeine) IV if premature or oral if baby feeds
  - Central stimulation
- Nasal CPAP
  - Splints upper airway obstruction
  - Maintains FRC $\rightarrow$ stabilized oxygenation
- Low flow nasal oxygen
  - Stabilizes oxygenation
  - Be careful not to hyper-oxygenate!

Please provide me with your feedback at:

kaltirkawi@ksu.edu.sa