Respiratory Distress Syndrome
or
Hyaline Membrane Disease

M.C.C of Respiratory failure disease in the [1st 2 days] HMD

RDS (2 types) <
1 (transient tachypnia of New born) TTN self limited benign ✓ – Not Related to surfactant ↓
2 [Hyaline memb. $] HMD

RDS ↓ surfactant Production
↳ function + Conc.

Full term ⇒ 5% of nn. at Risk ✓
GBS ← Can't differentiate 🫠

ex:

2 babys

- 35 w. → due to Prematurity
- 37 w. → due to GBS (Most severe)

both RDS

GBS:
- leading Cause for PPH ↳ severe hypoxia ↳ high frequency Counselation + Nitric oxide
- ↳ sepsis
- ↳ as Respiratory inf.

More خطورة

leading Cause of death [Vicious circle لذلك يدخل الطفل بـ

ex:

2 babys

- full term
- 34 w.

both RDS

Prognosis الـ بيكون أسوأ

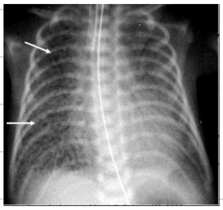
(infant of DM Mother) → لذلك بيكون عندهم Multiple causes of Respiratory distresses

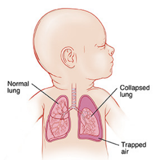
Good surfactant Concentration ✓

function بالـ مشكلة

1. ⇒ insuline ⊣ function of surfactant (inhibit)
2. ⇒ Polycythemia
3. ⇒ Meconium aspiration
   - ① → leading cause for PPHN (severe)
   - → Pneumothorax
   - → Pneumo mediastinum
   - ① air leak $
4. ⇒ Multiple CHD

HF ~





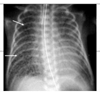
* RDS Not always from Premature #

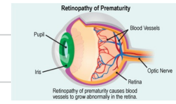
* single Most important factor for RDS → Prematurity, but the deficiency is → Relative

inCompatible w/ life ← absolute لو كذا *

≤ 22 w. don't resusstate

viability age → 24 w. } bad according to situation of NICU ↑

 (بيبين ١٤ علومات الجينة) Risk for bronch-Pulmonary dysplasia (BPD) 100% ① a ②

spastic cerebral CP ← < 500 g / 40% IVH / 

ROP 500 g

Incidence يعتمد ع شغلتين — GA / birth wt.

nn. diseases كل ال *
incidince نعتمد ع شغلتين ال
+ Prognosis

#

* extremely Premature Baby →	≤ 28	50%	دن
very Premature →	28 – 32	30%	↳ ROP Risk
Premature →	> 32		
Neo term →	36		
full-term →	37	5%	

decrease Incidence ال

due to vaccume ← ↑ Risk of RDS

very high

* extremely low Birth wt. →	< 500 g	71%
very Premature →	500 – 1000	54%
Premature →	1000 – 1250	36%
full-term →	1250 – 1500	22%

PBD 100%

decrease Incidence ال

* improvement in wt ≥ 1 kg → ↓ 22%

* factors Prevent RDS → steroid for high Risk mom

Recording

* RDS affect all Races [↓ in Developed] } Most in Caucasian

* ↓ developing (دول)?! لنجس ⇒ ↓ recording (to determine frequency)
⇒ in home تخلف

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* Contributing factors *

- Neonates younger than 37 weeks
- Weight less than 2500g
- Maternal diabetes
- Cesarean delivery without preceding labour elective
- Fetal asphyxia but emergency
- Second part of twins protective → Transient tachypnea of the newborn (TTN)
- White infants

another cause for PPHN * ← Meconium Aspiration (ttt: intubation)

* full term baby, but problem in function : asphyxia, hypoxic ischemic encephalopathy — the disease of full term baby

توضيح

Normal function — ① PH, ② temp

* PPHN → hypoxia → Resp. acidosis → PH ↓, PO_2 ↓, CO_2 ↑ → pulmonary vaso-constriction + systemic vasodilatation → Rt → Lt shunt → further hypoxia ↓ lactic acidosis
(ant. fontanell مفتوح ——)
+ (foramen ovale " ")
Trigger chemoreceptors → More tachycardia, More shunt

[Vicious Circle]

only treatment :- ⇐

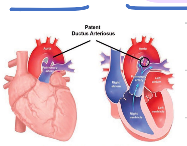
① selective vasodilation (Nitric oxide) — not available
NOT complete → systemic hypotension #
O_2 ← 90, 80, 70

② washing CO_2 (hyperventilation) — Mechanical → عن طريق ... high frequency (not available) ossilation 600-3000
RR in nn. 40-60 طبيعي

* conventional ventilation ≈ 80

③ ecmo Machine (not available)

* Complications of Prematurity :- ①
- 35% PDA ① + surfactant therapy



one of the causes of pulmonary Hge.

Secondary surfactant deficiency may occur in infants with the following:

- Pulmonary infections e.g. GBS
- Pulmonary hemorrhage
- Meconium aspiration pneumonia
- Oxygen toxicity; barotrauma or volutrauma to the lungs
- Congenital diaphragmatic hernia → scaphoid abdomen + sever cyanosis → ttt: intubation

(Pulmonary infections, hemorrhage, meconium aspiration → affect the function of surfactant)

* maximum PO_2 → > 80 / 24 hours (O_2 sat → 100%)

ROP — bronchopulmonary

surfactant :-

↓ Surfactant- lertion at the end of expiration (↓complete cat-elactesis).

① large vent-elation / perfusion mismatch.

② respiratory acidosis + atelaciesis.

③ Dead space ↑ → cant maintain Functional residual capacity

④ ↓ compliance

⑤ massive hypoxia.

↳ metabolic acidosis → hypoxia.

بنجيب عن طريق ↓

→ Positive end expiratory pressure [peep]

* ④ type of protein in surfactant

A, B, C, D

A, B → hydrophilic [Head]

C, D → hydrophobic [tail]

Factors decrease the risk of RDS :

كل ما كانت الفترة ابكر بكون احسن

* 24-34 w.   (* 2 w. قبل) ##

* surfactant → Phospholipids, Proteins (A, B, C, D)

- Use of antenatal steroids ← (↑ Mitochondrial Messenger RNA → ↑ Ca^{2+} influx) → for any ↑ Risk mom
- Pregnancy-induced or chronic maternal hypertension → hypoxia ↓ ↑ surge steroid (IUGR) (Preeclampsia)
- Prolonged rupture of membranes → ↑ Risk of sepsis #
- Maternal narcotic addiction

chronic ROM ≈ oligohydraminous → ↑ Risk of RDS #

Pregnancy (stress) ↑ induced steroid surge

52- All the following are at increased risk of respiratory distress syndrome (RDS), Except:
a. Infant of diabetic mother
b. Premature baby
c. Low birth weight
d. Second-born twins
e. Maternal Preeclampsia

Pathophysiology :

* Oxygenation : oxygen
* ventilation : oxygen + pressure

- Lung surfactant deficiency is the primary cause
- Pulmonary surfactant synthesis, in type II pneumocytes
- surfactant production begins at 24-28 weeks of gestation, and gradually increases until full gestation

- The cause of respiratory distress syndrome is relative deficiency of surfactant, which decreases:

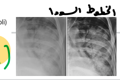
1st line of tt: PEEP
- lung compliance ↓
- functional residual ↓FRC
- increased dead space.

flaiable chest بكون لينا (Premature baby) ← need ↑ intra-thoracic negative pressure عشان افتحها
sub costal + intercostal contraction

• **Air bronchograms** (air-filled bronchi against collapsed alveoli)

No exchange (air bronchogram)

الخطوط السودا

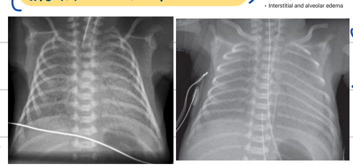


"Ground-glass appearance"

This is due to:

- Diffuse alveolar collapse (atelectasis)
- Poor lung inflation
- Interstitial and alveolar edema

(Ground-Glass appearance)



Atelectasis of Alveoli

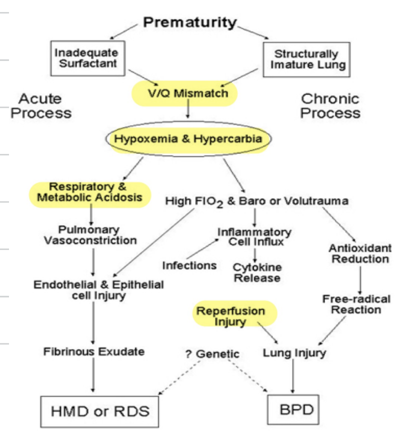
→ large ventilation–perfusion (V/Q) mismatch right–to–left shunt.

* O_2 — Mask
  — C-PAP (cont. (+) Airway Pressure) ##) to prevent (atelactasis) (diffuse) (BPD)

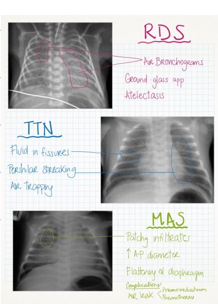
* surfactant → prevent collapse at end of expiration ### + end exp. pressure.

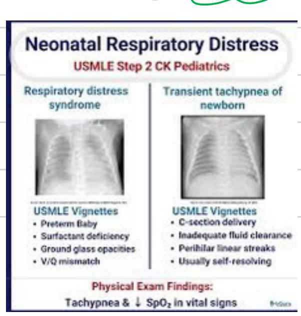
⟹ diffuse atelactasis → ↑ V/Q mismatch → ↑ PCO_2, ↓ PO_2, ↓ PH → pulmonary vaso-const. → opened → Rt-Lt shunt → hypoxic in tissues → accumulation → Metabolic Acidosis

Mixed acidemia w/ hypoxia ← Metabolic Acidosis, hypoxia, respiratory Acidosis

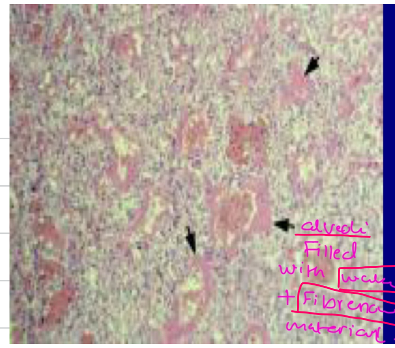


RDS vs TTN
↳ deteriorating (lung recruitment)





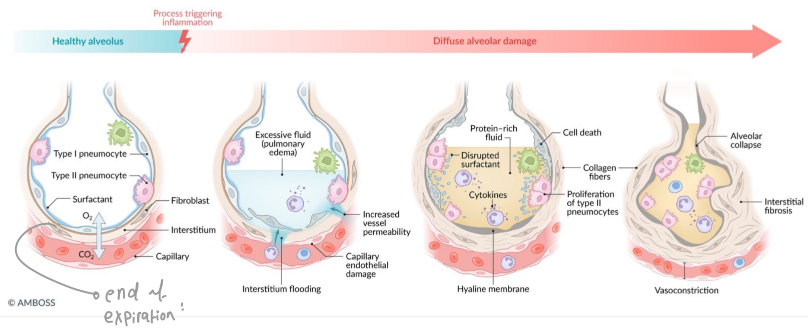
HMD
شبه ال liver



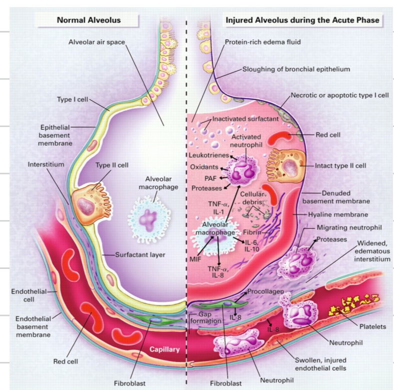
atopsy

macroscopic evaluation

- lungs appear airless and ruddy
- Diffuse atelectasis of distal airspaces
- Progressive atelectasis damages endothelial and epithelial cells lining distal airways, resulting in :
  - exudation of fibrinous matrix derived from blood.
  - Hyaline membranes that line the alveoli may form within a half hour after birth.



Complete بتلاقي بعض ما رح يصير collapse
يتحافظ عليه من ال



- Chemical mixture that decreases surface tension
- Lines the air-fluid interface in alveoli
- Prevents atelectasis at the end of expiration
- Synthesized in alveolar type II cells and stored in lamellar bodies

Pulmonary Surfactant

Function of lung surfactant

decreases surface tension during expiration (function) → B C → lung fibrosis (20s ثلاث)

allows the alveolus to remain partly expanded

maintains functional residual capacity

- Pulmonary surfactant deficiency
  - Atelectasis causes lung inflammation and respiratory epithelial cell injury
  - Decrease fluid absorption and lung edema
  - Accumulation of neutrophils in the lung

⇒ early in life

SP-B — Mandatory for water film
Required for normal pulmonary function → any Mutation incompatible w/ life
Mutation result in deficiency SP-B Can cause severe lung disease that is lethal in perinatal period

SP-C
Promotes formation of phospholipid film lining of alveoli
- Human with SP-C deficiency develop interstitial pulmonary fibrosis in early childhood
- SP-C deficiency do not cause respiratory distress at birth

SP-A and SP-D
- They are host defense of the lung (against bacteria + viruses)
  - Kill bacteria
  - Kill viruses
- They have carbohydrate recognition domain allows coating, and phagocytosis of virus and bacteria

Surfactant — Phospholipid + Apoproteins } Mixture lipoprotein
Phospholipid → lecithine & sphingeomyeline
lecithine → Dipalmitoylphosphatidylc holine DPPC (Primary functionally phospholipid)
sphingeomyeline: Constant
lecithine: keep increasing in pregnancy
90% → lipid → lecithin.
10% → proteins → hydrophilic / hydrophobic

Ratio نعتمد ع ال
lung maturation نقدر من خلالها نقيم ال

في ال 3 حالات :
① Meconium aspiration ↓ function
② Pulmonary Hge. (1-3)
Not sense ← Not all the time

>2 Mature ✓ delivery
immature <2 delay delivery

23 w. ← Viability age يرتبط بال

amniotic fluid → 34 w.

Timing of lung surfactant :

Good Conc. on Alveoli → 36 w.

- The timing of lung surfactant or (Lecithin) production :
- At 32-34 weeks fetal cortisol increase ——>> Stimulate Type II pneumocyte cells
- By 34-36 weeks sufficient amount of Lecithin secreted into alveolar lumen & Excreted into the amniotic fluids
- Lecithin concentration in amniotic fluid indicate lung maturity #

DDx ??

① TTN

→ doesn't correlate w/ severity #

- → improving / we don't need X-ray to confirm diagnosis. or for surfactant
- → need low conc. of O2 to be improved
- → بعطيهم : Just Nasal cannula or C-PAP
- Rarely → Mechanical ventilation #

- Prominent perihilar streaking
- Fluid in the fissures
- Small pleural effusions may be seen
- Patchy infiltrates have also been described ?

surfactant therapy Room + intubation ← ‹ 30w اي حالة *
(Risky therapy)
NICU ما بضيعوها لوقت ال

lung derecruitment
if collapsed → lung high pressure #
high volume
high O2
BPD

غلط يخرج ↗ ok !

GBS (can't differentiate by X-Ray)

① Bacterial Pneumonia
(3-5 d.) anti-biotics كلهم نعطيهم

② Anemia
④ hypoglycemia
⑤ meconium asp.
⑥ hypothermia
*⑦ Pulmonary air leak (Pneumothorax)
*⑧ Diaphragmatic hernia
⑨ Cardiac anomaly

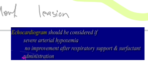
↳ cyanosis after delivery
مباشرة !

spot diagnosis by auscultation.

← 32 - 31 w. *

Not indication for غرفة الولادة
NICU ← تعطيه C-PAP بغرفة الولادة، بعدها بنقله ع
(Portable C-PAP)

① Transposition w/o VSD
② Tricuspid atresia w/ very small VSD & ASD
③ total anomalous Return the obst. type
→ Dx ? hyperbaric O2 (*)→ Prostaglandine E1 if ductal dependent lesion



* severe cyanosis
+
scaphoid abdomen → diaphragmatic hernia) untill proven other-wise

① intubation
② O2 قبل ال
③ (decompression) NG tube

Not by Positive Pr. vent. ✗ 2nd line of Mx → O2 #

ABC بالاول secure the airway

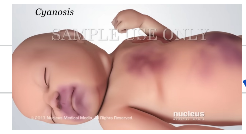
Diagnosis

→ Deteriorating the improvement (Progressive)

* No test can confirm RDS.

* completely so
① Clinically — X-Ray
— Blood gases
— lung function
— others

② S&S :- Soon after Birth



flaring ↓ ↑ resistance
$\frac{T_1}{(R)^4}$ ↓ Radius
small air way

- **Progressive signs of respiratory distress are noted soon after birth and include the following**
  - ✓ Tachypnea
  - ✓ Hypoxia → very bad sign
  - Cyanosis (central) — bluish ← deoxy hemoglobin >25% with normal Hb [75%] — * 4 types of cyanosis: acrocyanosis, differential [at coarctation of aorta], central, peripheral
  - Expiratory grunting (from partial closure of glottis)
  - Subcostal and intercostal retractions ? flexible chest
  - Nasal flaring ?
  - Extremely immature neonates may develop apnea and hypothermia — bad prognostic sign

* DDx: of central cyanosis — [with cardiovascular manifestation] breathing? if no, how to

* >6 time [in whatever]

* anemia → masks the cyanosis
* polycythemia → agrivated the cyanosis

Respiratory Distress Syndrome

breathing comfortable but cyanotic. — defferentiate of cardiac or RS?

Prenatal Diagnosis

- ✓ History of premature delivery
- ✓ Ratio of lecithin/sphingomyelin
  - L/S ratio 2:1 indicate lung maturity
  - *Lecithin :* indicate lung maturity
  - *Sphingomyelin :* remains constant during pregnancy

Lecithen/sphingomyeline — >2 → mature lung → delivery; <2 → delay delivery

* → need intubation
* hernia
* meconium
* >6 time apnea
* 1 apnea need ambubaging

Rt → Lt shunt

- **Blood gases** →
  - Blood gases show respiratory and metabolic acidosis along with hypoxia. ← Rt-Lt shunt.
  - **Respiratory acidosis** occurs because :
    - alveolar atelectasis
    - overdistension of terminal airways
  - **Metabolic acidosis,** is primarily lactic acidosis, results from
    - poor tissue perfusion
    - anaerobic metabolism.
  - **Hypoxia**: occurs from right-to-left shunting of blood through the pulmonary vessels, patent ductus arteriosus (PDA), patent foramen ovale
- **Pulse Oximetry**

Diagnosis

↓ compliance
↑ Dead space
↓ FRC

- Full blood count
- Cultures to rule out sepsis
- Electrolytes, glucose, renal and liver function
- Echocardiogram:
  - *diagnosing PDA*
  - *determine the direction and degree of shunting*
  - *making the diagnosis of pulmonary hypertension*
  - *excluding structural heart disease*

- **CXR**
  - Bilateral, diffuse reticular granular or ground-glass appearances
  - Air bronchograms
  - Poor lung expansion
  - *Prominent air bronchograms represent aerated bronchioles superimposed on a background of collapsed alveoli.*
  - Heart is normal or enlarged.
  - ✓ Cardiomegaly may be the result of: (DDx)
    - *Prenatal asphyxia*
    - *Maternal diabetes*
    - *PDA*
    - *Congenital heart anomaly*
    - *poor lung expansion.*

+ hypoventilation

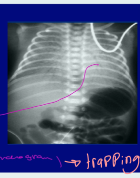
- ⇒ Radiologic findings of RDS can be similar to pneumonia
  - Group B beta-hemolytic streptococci.
- If streaky opacities, the diagnosis of: ***Ureaplasma* or *Mycoplasma*** pneumonia should be considered and confirmed by means of tracheal aspirate cultures grown in the appropriate medium.

Pulmonary Function

- ↓ Compliance decrease
- ↓ Functional residual capacity is reduced
- Hypoxemia secondary to mismatch of ventilation
  - PDA and foramen ovale plays rule in hypoxemia due to R-L shunting
- Alveolar ventilation is decreased

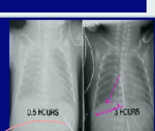
CXR of RDS

- Ground glass appearance
- Air bronchogram
- Poor lung expansion



ground glass — air in the bronchiole [air-bronchogram] → trapping

- **Left: Initial radiograph shows**
  - poor lung expansion
  - air bronchogram
  - reticular granular appearance.
- **Right: At 3 hours**
  - after surfactant therapy
  - marked improvement



endotracheal tube (to open lung) high volume → Rapid weaning → Prevent cyanosis (Pneumothorax)

ground glass, white lung — abnormal a'Pleu. surfactant (true lab)

Treatment

Need supportive ttt #

- Oxygenation
- Surfactant (Ventilation)
- Infection Control

* Prematurity → Most important RF.

→ Goals :- S.E ... ttt ↓ RDS ↓ 4x

① Oxygenation :-

* SpO2 → 88 - 92%
* O2 → 50-80
* PCO2 → 35-45

oxygen Radicals → BPD
over ventilation → ROP
↓ PCO2
<35 — Apnea
PVL — Chorio amnionitis
↑ hypocarbia
HIE

accepted if low PCO2 < 35

* ↑ failure ⇐ extubation ↓ hypocarbia ↳ Risk for Apnea

- Nasal cannula
  - if >25% oxygen is required

<88% hypoxia
>92% hyperventilation
PO_2 <55 ↓ intubation
✓ failure of CPAP

C-PAP — surfactant ↓ → Not improve → CPAP failure.
— Mechanical vent.

FiO2 → 21 - 60% or 80%
deep → 5-7

∴ Next step ??

surfactant + intubation

* < 30 week → intubation + surfactant [by endotracheal tube]
✓ [don't wait for x-ray]

8 indications for intubation

Respiratory Treatment of RDS

- ✓ Nasal CPAP [oxygen + peep] continuous + airway pressure
  - Using CPAP soon after delivery *reduces the number of babies requires ventilation*
- Indication of CPAP
  - In delivery room for babies at risk of RDS
  - Babies on low flow oxygen with respiratory distress
- Mechanical ventilation ?

❖ What is your O_2 targets ?
- SpO_2: 88-92%
- P_{CO2}: 45-55 mmHg
- Maintain PaO_2 at 50-80 mmHg
- PH :

1)Less than 30 weeks
If more than 30 weeks 2) 6 apnea need tectile stimulation
3) 1 apnea need ambu bag
4) congenital diaphragmatic hernia
5) muconeum aspiration
On blood gases 6) PaO2 less than 55
7)PaCo2 more than 55
8) failure of CPAP

→ $\uparrow O_2$ / $\downarrow CO_2$

No Hypervent
↳ PVL, RoB, BPD

→ What is your target? PaO_2 50–80

If PaO_2 > 100 for 24 hrs → Hypervent

→ How Can I maintain that?

- Continous monit of ABG ... there is a Device مش موجود عندنا
- بالعادة → By O_2 Sat ($PaO_2 = O_2 Sat - 30$)   88–30 = 58 PaO_2
  ↳ 88–92   92–30 = 62 PaO_2

→ Normal PCO_2 in neonate = 45–55 in first 7 days

if after 7 days neonate on Vent, accept PCO_2 up to 70

"Permisive Hypercarpia"

→ How to maintain O_2 Sat at the level i want?

- <30 week → Intubation and surfactant (Mech Vent)

  ↲ تخلص منه في اقرب فرصة (يعني اول ما يتحسن المريض)

  ↳ Baby w/ stiff lung need certain amount of pressure provided by Mech vent.

  ↳ But the pressure required is decreased after giving surfactant so we need to lower the pressure provided by the ventilator as soon as possible.

  ↳ If we don't lower the pressure (from Vent) after giving surfactant, Pneumothorax will be the result

Eg. 30 week-old baby w/ RDS, you start Mech Vent and gave him Surfactant, After 2 hrs the baby is status is detorierating with Hypotension; Cyanosis and retractions ....

↳ The baby has pneumothorax (due to high pressure)

↲ حتى لا تنتظر Transillumination بتعمله لما بتستنى الـ X ray

→ Tidal Volume = 7–14 /Kg

- >30 weeks → Nasal Canula or CPAP
  - Nasal Canula:
    - PEEP: ~2
      ↳ So use it if Mild case
  - CPAP:
    - PEEP: 5–10   NL in neonate ↳ 6–8
    - FiO_2: up to 100%
    - Continous pressure
    - Apnea
      ↳ firstly give Caffeine Citrate, if no response, Put the Baby on CPAP.
      ↳ Agitation/Stimulation of the baby

∴ >30 week

1- Nasal Canula
2- CPAP
3- Intubation and Surfactant

طيب ليش الكل ما نعطيهم Surfactant ???
هذا ما سنعرفه بالسلايد الآت القادمة

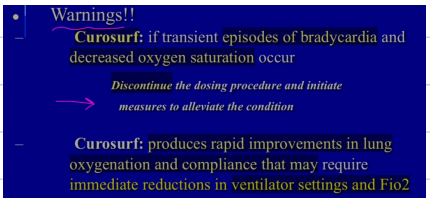
* أنواع الـ surfactant :-

- **Natural lung surfactant**
  - Alveofact - extracted from cow lung lavage fluid
  - Curosurf - extracted from material derived from minced pig lung
  - Infasurf - extracted from calf lung lavage fluid

- **Survanta-** extracted from minced cow lung with additional DPPC, palmitic acid and tripalmitin → weaning بعد ما نستزفهما
  in Jordan هذا اللي بنستخدمه

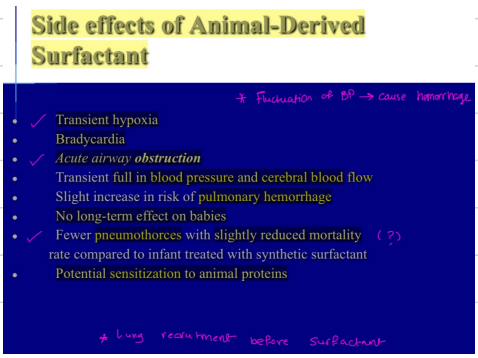
- Curosurf (Poractant alfa)
  Dosage: Intratracheal: Initial: 2.5 mL/kg/dose (200mg/kg/dose); may repeat 1.25 mL/kg/dose (100mg/kg/dose) at 12-hour intervals for up to 2 additional doses; maximum total dose: 5 mL/kg

- Precautions:
  Correction of acidosis, hypotension, anemia, hypoglycemia, and hypothermia is recommended prior to administration.



Pneumothorax ??? NOT safe surfactant + intubation
* ليش مش كلهم نعطيهم
air way obstruction → Tube وبال → hypoxia
PBHL اذا كان صغير
bleeding (Pul. Hge)

Trauma Vocal cord
Trachea Perforation
bleeding
infection
2 complications



BPD — Mechanical ventilation
Pneumothorax
Pneumomediastinal
subsurgical emphysema

Supportive ttt :-

Supportive treatment (premature).

- Temperature regulation: prevent hypothermia. (because ↑ surface area.)
- Fluids, metabolism, and nutrition: closely monitor and maintain blood glucose, electrolytes, acid balance, calcium, phosphorous, renal function, and hydration.
- Once the infant is stable, intravenous nutrition with amino acids and lipid.
- After the respiratory status is stable, initiate small volume gastric feeds (preferably breast milk) via a tube to initially stimulate gut development

✓ (immature skin).

Supportive treatment

- ***Circulation and anemia***: monitor heart rate, peripheral perfusion, and blood pressure. Blood or volume expanders may be required.
- ***Antibiotics*** : start antibiotics in all infants who present with respiratory distress at birth after obtaining blood cultures.
  - Discontinue antibiotics after three to five days if blood cultures are negative.
- Support of parents and family: keep the parents well informed. Encourage parents to frequently visit and stay with their baby

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How to prevent :-

Prevention of RDS

* <30 week → intubation + surfactant [Don't wait for X-ray] ✓

- American Academy of Pediatrics recommendation 2008
  - Intubation of infant born at or before 30 weeks gestation in the delivery
  - Prophylactic natural surfactant therapy is administrated through the ET as soon as the infant is stable after intubation
- Do not delay surfactant for CXR
- No CXR is necessary to confirm proper tube placement

- ✓ Antenatal Steroids should be given to any pregnant women at 24 to 34 weeks of gestation with intact membranes at high risk for preterm delivery. (DM, short, pre-eclampsia) high risk mom ✓
- After administration of surfactant and if the infant is active and exhibit spontaneous respiratory effort :
  - extubation and stabilization on CPAP rather than continued intubation and M.V

- Prophylactic surfactant therapy is not recommended in infant greater than 30 weeks gestation
- Delaying premature birth.
  - Tocolytics may delay delivery by 48 hours and therefore enable time for antenatal corticosteroids to be given.
- Good control of maternal diabetes
- Avoid hypothermia in the neonate

Complications :-

acute

Acute complications (Killer)

- ① Alveolar rupture
- Infection
- ✓ Intracranial hemorrhage and periventricular leukomalacia
- ✓ Patent ductus arteriosus (PDA)
- ✓ Pulmonary hemorrhage
- ✓ Necrotizing enterocolitis (NEC) and/or GI perforation
- ✓ Apnea of prematurity

• ICH → grades → U/S

- Alveolar rupture: → resistant
  - when an infant with respiratory distress syndrome suddenly deteriorates with hypotension, apnea, or bradycardia or when metabolic acidosis is persistent. + cyanosis
  - → pneumomediastinum, pneumopericardium, interstitial emphysema, pneumothorax
- Infection
- Intracranial hemorrhage and periventricular leukomalacia:
  - is observed in 20-40% of premature.
  - Cranial ultrasonography is performed in the first week and thereafter as indicated
  - Prophylactic indomethacin therapy and antenatal steroids have decreased the frequency
  - periventricular leukomalacia associated with Hypocarbia and chorioamnionitis

*sudden onset pneumothorax → midsternum shifting [aorta + SVC kinking] + tracheal shifting → Rapid cardiogenic shock.

→ transillumination test → ⊕ needle aspiration

Ductus arteriosus physiological closed 48 hours // anatomically 2 weeks.

- 35% [Patent ductus arteriosus (PDA) with increasing left-to-right shunt: machinery murmur [systolic + diastolic]
- Suspect PDA in any infant who deteriorates after initial improvement or who has bloody tracheal secretions.. (C/P → Pulmonary edema ... tachypnea, cyanosis, crepitation + machinery murmur syst. diast.)
- Pulmonary hemorrhage: especially after surfactant therapy.
- Necrotizing enterocolitis (NEC) and/or GI perforation

systemic circul. ... NPO ... endomethacine → prostaglandine (VC) ... complication ① V(VC) → NEC ② Na⁺ & water retention

IVH ↓ endomethacin

+ shiny abdomen
* ↑ residual volume
* oligourea.
* tacypnea
* V/P mismatched
* machinary murmur
+ crepitation

premature: T: endomethacin (to block prostaglandin) ↳ * sei: 1. renal shutdown (KFT) ② vasoconstriction [NEC] → NPO ③ Na-water retention [accumulation of fluid].

* Maximum intra-uterine PO2 → 55

ttt:-
- Apnea of prematurity: → 1st day always Pathology / 2nd day ↓ Physiological
  - Apnea of prematurity is common in immature infants
  - Manage apnea of prematurity with
    - methylxanthines (caffeine)
    - (CPAP)
    - assisted ventilation in refractory incidents.
  - Exclude
    - ✓ Septicemia
    - ✓ seizures
    - ✓ gastroesophageal reflux
    - ✓ metabolic causes
    - ✓ ICH / ✓ electrolytes disturbances

✓ ↓ Resp centre: aminophiline, caffiene ... ① tactile stimulation, CPAP, Mechanical ventilation

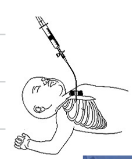
* Complete Maturation of Respiratory centre ≈ 34 w.

✓ *apnea 1st day → pathological cause.
✓ * apnea 2nd day → due to prematurity → immature of Respiratory

* How can I confirm the diagnosis :-

PDA

(very small baby :-
- Rapid transillumination
- NOT X-Ray
- needle decompression ??
  - ABC
  - butterfly needle at 2nd ICS (under seal water)



Kit includes two sets of the above

Chronic

Chronic complications

- → Bronchopulmonary dysplasia (BPD) to prevent it the best T
- → Retinopathy of prematurity (ROP): T: no treatment.
- → Neurologic impairment

2-5 years ... screening to prevent blindness.

- → Bronchopulmonary dysplasia (BPD)
  - Defined as a requirement for oxygen at 36 weeks CGA. → corrected gestational age
  - BPD is related directly to the high volume and/or pressures used for mechanical ventilation.
  - BPD increases with decreasing gestational age.
  - Rx PBD by
    - Postnatal use of surfactant therapy ?
    - gentle ventilation
    - vitamin A
    - low dose steroids and inhaled nitric oxide

(over distension of alveoli ... collapse + over distension) shearing force on alveoli → inflammatory process → fibrosis → O2 dependence

✓ *no treatment for fibrosis

O2 ... 36 w ①
... 28 ... ②

Pathophysiology [barotrauma, volutrauma, hemotrauma, O2 radical]

NO ttt / best Prevention → to know underlying pathology

... oxygen radical
... baro-trauma ... over ventilation — alveoli

✓ * immature retina

- Retinopathy of prematurity (ROP): → T: laser photocoagulation
  - Infants with respiratory distress syndrome and a PaO2 >100 mm Hg are at increased risk for ROP.
  - An ophthalmologist examines the eyes of all premature infants at 34 weeks' gestation and thereafter as indicated. ↳ Follow up by opthalmoscope
- Neurologic impairment occurs in approximately 10-70%.

Advanced → interest / good ttt if early diagnosed & 34 w. ↳ retinal detachment → blindness (screening & laser)

Prognosis (weight, gestational age)

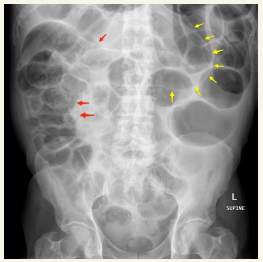
- → Very low birth weight <501 grams survival rate is 10% and 100% risk of BPD and very high risk of ROP
- Birth weight between 1001-1500 grams survival rate is ~ 96% and few develop BPD and ROP as well

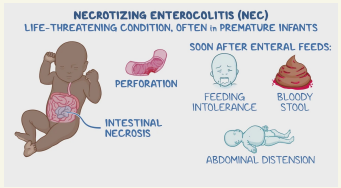
* 24 week → viable age
>24 → resuscitation

NEC

Risk factors for NEC :-

→ Prematurity , PDA , ischemia , infection





C/P :-

abd. distention , bleeding (rectum) , X-Ray → air intra-Peritoneum → Pneumatosis intestinalis #
→ thickening bowel #

MX :- 1 Medical : triple antibiotic ✓

Correction acid-base balance ✓

mechanical ventilation ✓

IV fluids ✓

Respiratory support ✓

2 surgical

if there is Biliary duct Perforation ✓
or
Failure of Medical ✓