

NEONATAL RESPIRATORY DISTRESS SYNDROME

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NEONATAL RESPIRATORY DISTRESS SYNDROME

Hyaline membrane disease (glassy appearance of proteins and dead cells, hyalos in greek means transparent)

Disease of RS (alveoli)

Respiration; breath in and out

Distress; difficulty breathing

Syndrome; associated signs and symptoms of a disease (NRDS) - cyanosis (bluish discoloration), grunting (rough sound), nasal flaring, tachypnea (RR > 60), chest wall retraction, xiphoid retraction, hypoxia (spo2 < 90)

CAUSE

Inadequate surfactant

RISK FACTORS

Gynecological conditions that lead to preterm delivery i.e. born before 37 weeks (34 in this case because of surfactant)

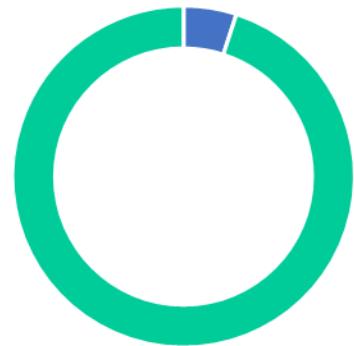
Besides prematures what reduces neonatal surfactant in older neonates;

Maternal - family hx, CS (labour increases surfactant, less lung fluid absorption -1/3 in SVD, diabetes (Insulin delays surfactant), perinatal sepsis (inflammation)

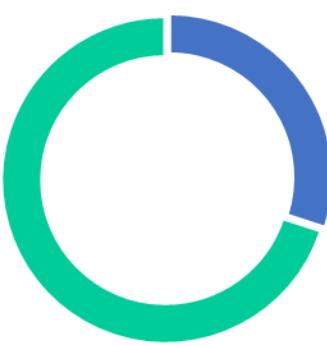
Foetal - twin gestation (birth weight - IUGR, hypocortisolemia, substrates), hypothermia (increased oxygen consumption and pulmonary vasoconstriction), congenital PDA, male (androgens vs estrogens in lung maturation)

RDS increases with decreasing gestational age

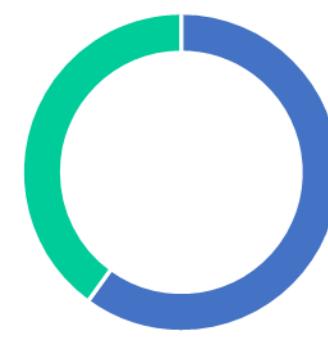
- The risk of RDS is inversely proportional to gestational age; occurs in approximately:



Near-term infants



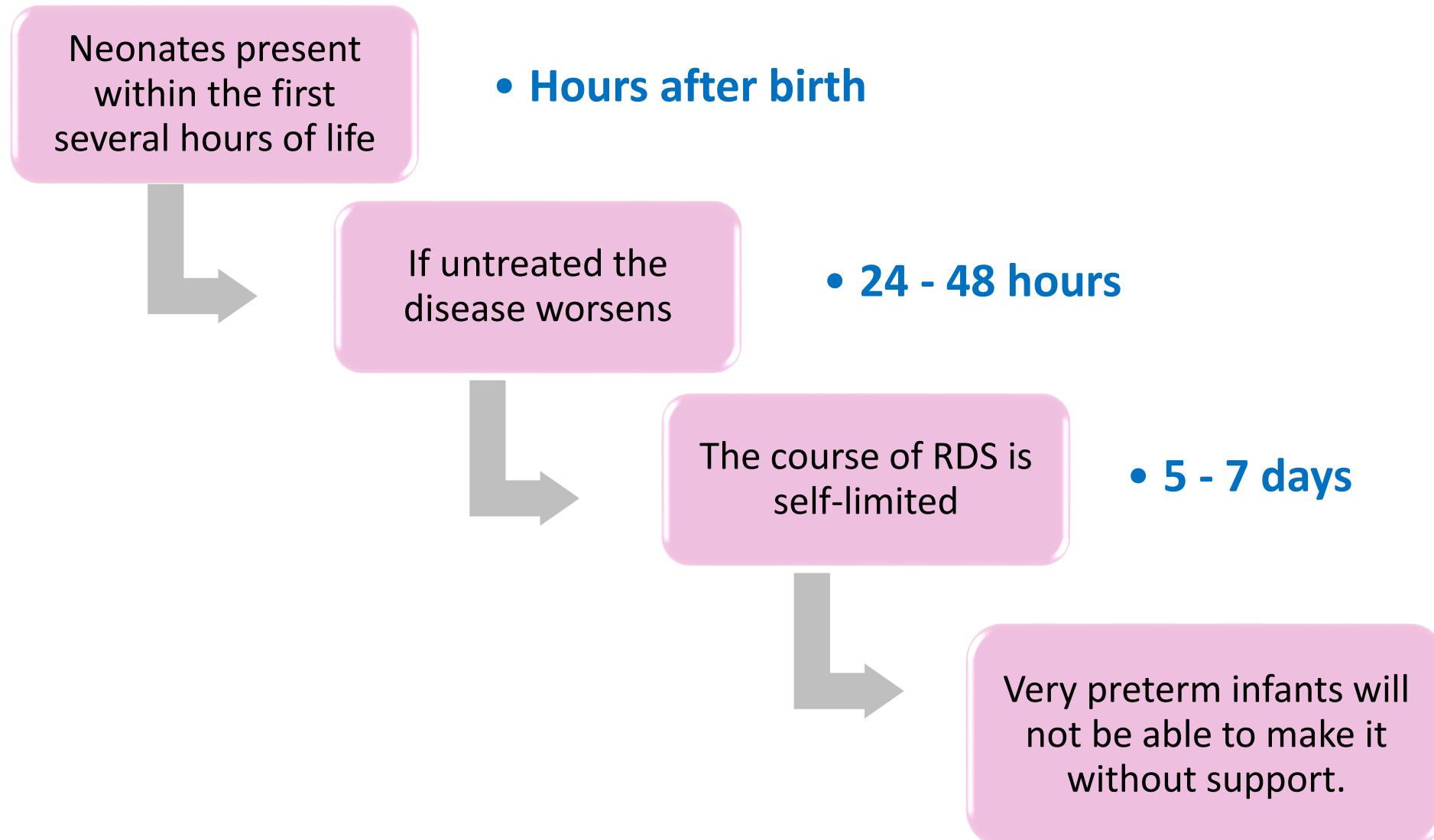
Infants <30 weeks



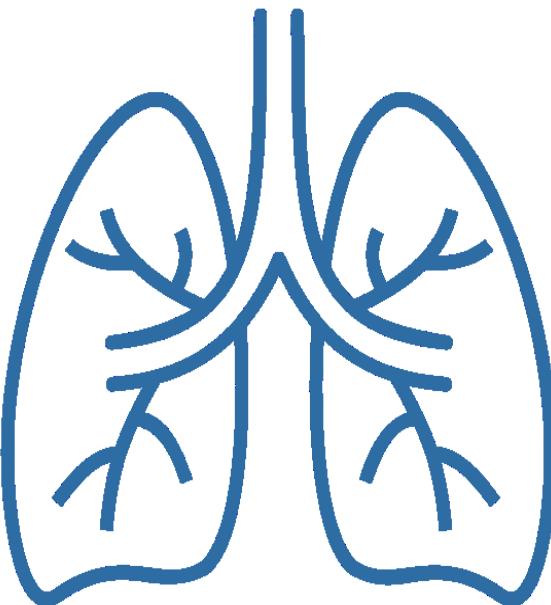
Infants <28 weeks

- RDS is seen soon after birth, worsens during the first few hours of life
- In contrast to Transient Tachypnea of the Newborn (TTN), worse at birth but improves within hours of birth

Clinical Course of RDS



Periods of Treatment for RDS



RDS was first described by Hochheim in 1903, who noted unusual membranes in the lungs of 2 infants who died shortly after birth

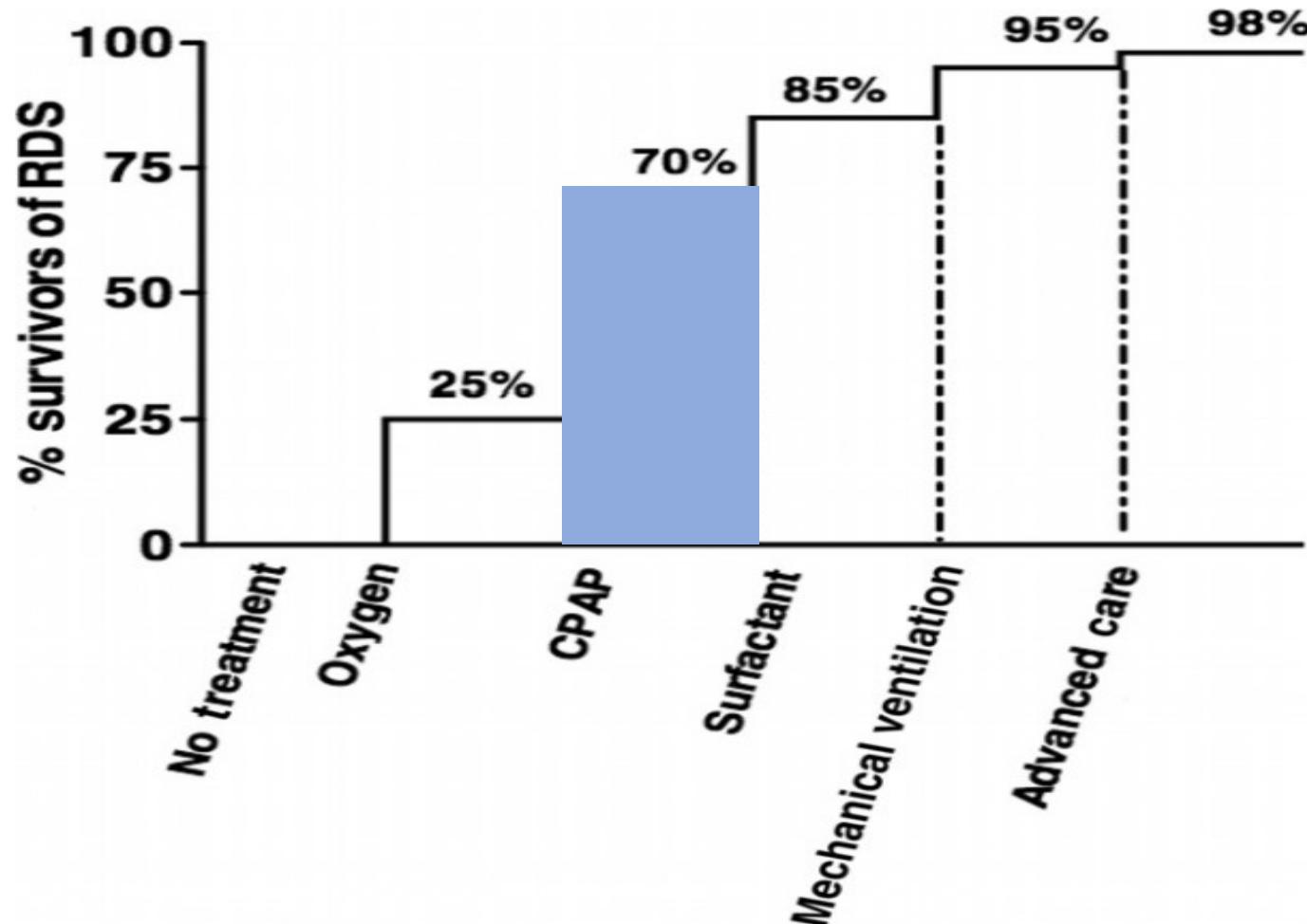
Period 1 - Before 1950s: No widely used treatment

Period 2 – 1950 – 1969: Oxygen therapy was the specific intervention

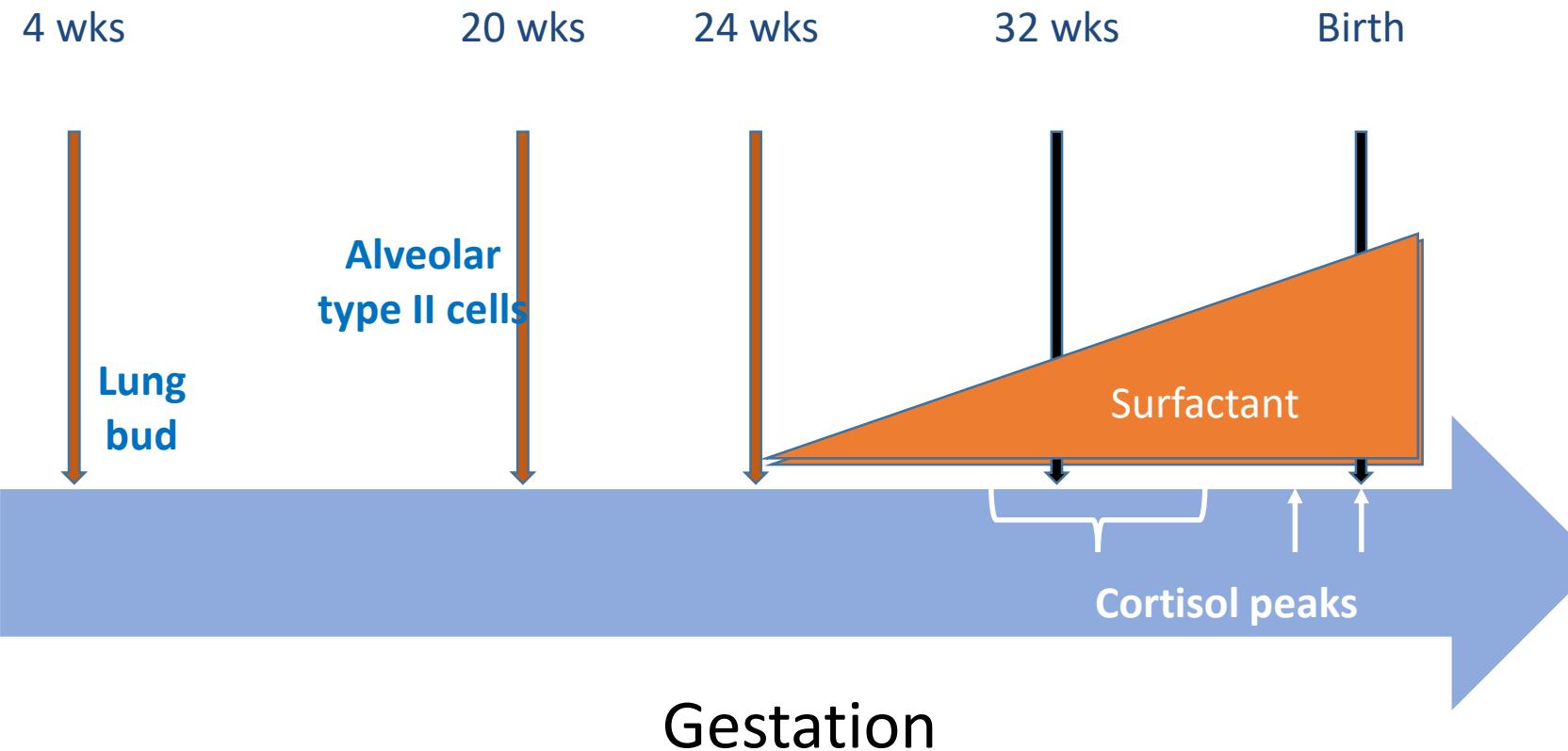
Period 3 – 1970 – 1989: CPAP therapy was the specific intervention. Later on use of mechanical ventilation was attempted

Period 4 – After 1990: Antenatal corticosteroids, surfactant, advanced care technologies e.g. high frequency oscillation

Increased % in RDS survivors with introduction of specific treatments



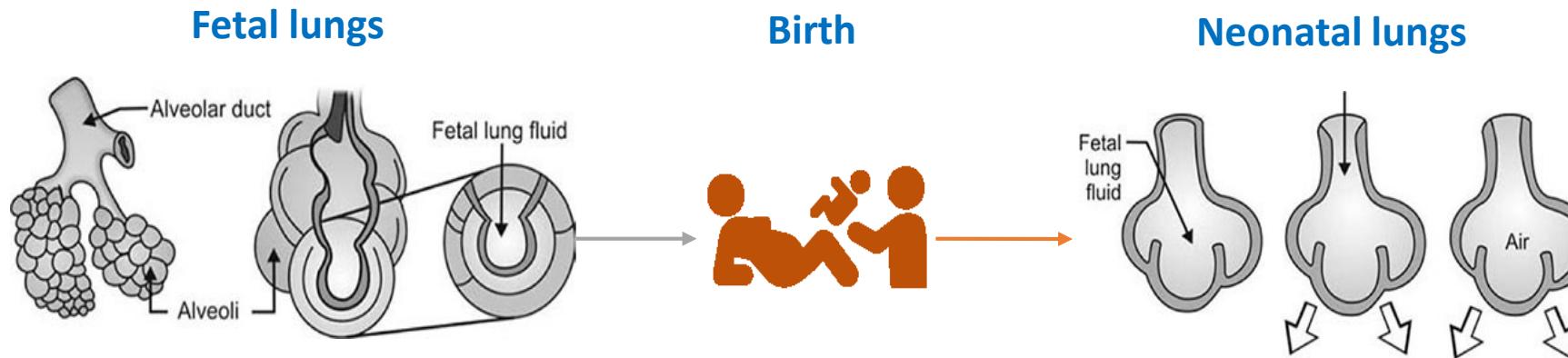
Lung growth and Development



Transition to extrauterine life

Cortisol

- ✓ Levels increase at **30 - 36 wks**, prior to term labor & peak **at labour**
- ✓ Regulates thyroid hormones and catecholamine release



- ✓ Fetal fluid secreted into lungs
- ✓ Promotes development
- ✓ Maintains distension
- ✓ Pressure = $2 - 4\text{cmH}_2\text{O}$
- ✓ Mechanical stretch stimulates surfactant production

- ✓ Fluid replaced by air (labour & delivery, first breath and cry)
- ✓ Reduced secretion; increased absorption (regulated by hormones)
- ✓ Surfactant coats alveoli

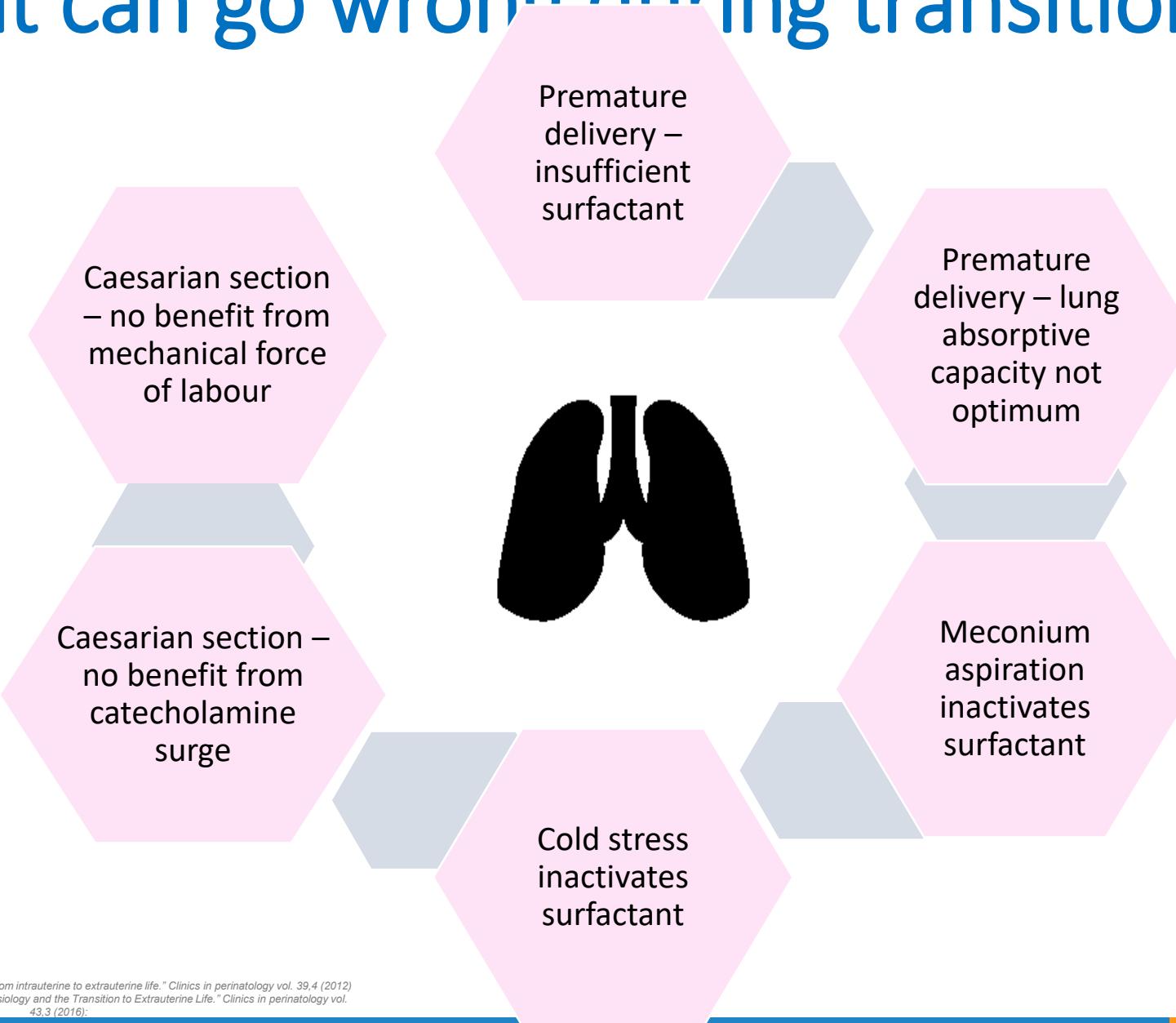
Surfactant



- A complex mixture of phospholipids and proteins
- Reduces surface tension at the air-liquid interface of the alveoli
- Prevents collapse of alveoli during end exhalation
- Secretion is stimulated by hormones like thyroxine as well as glucocorticoids
- Mechanical stretch (distension and hyperventilation) can stimulate secretion from Alveolar type II cells

Surfactant production can be hindered by inflammation, genetic defects, infection

What can go wrong during transition?



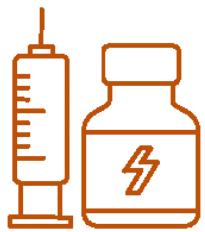
Antenatal steroids and the fetal lung

1

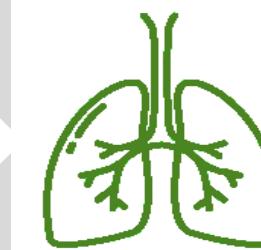
Improved antenatal care – to reduce risk of premature deliveries

2

Premature labour 24 - 34 weeks



Administration of antenatal corticosteroids (ACS)

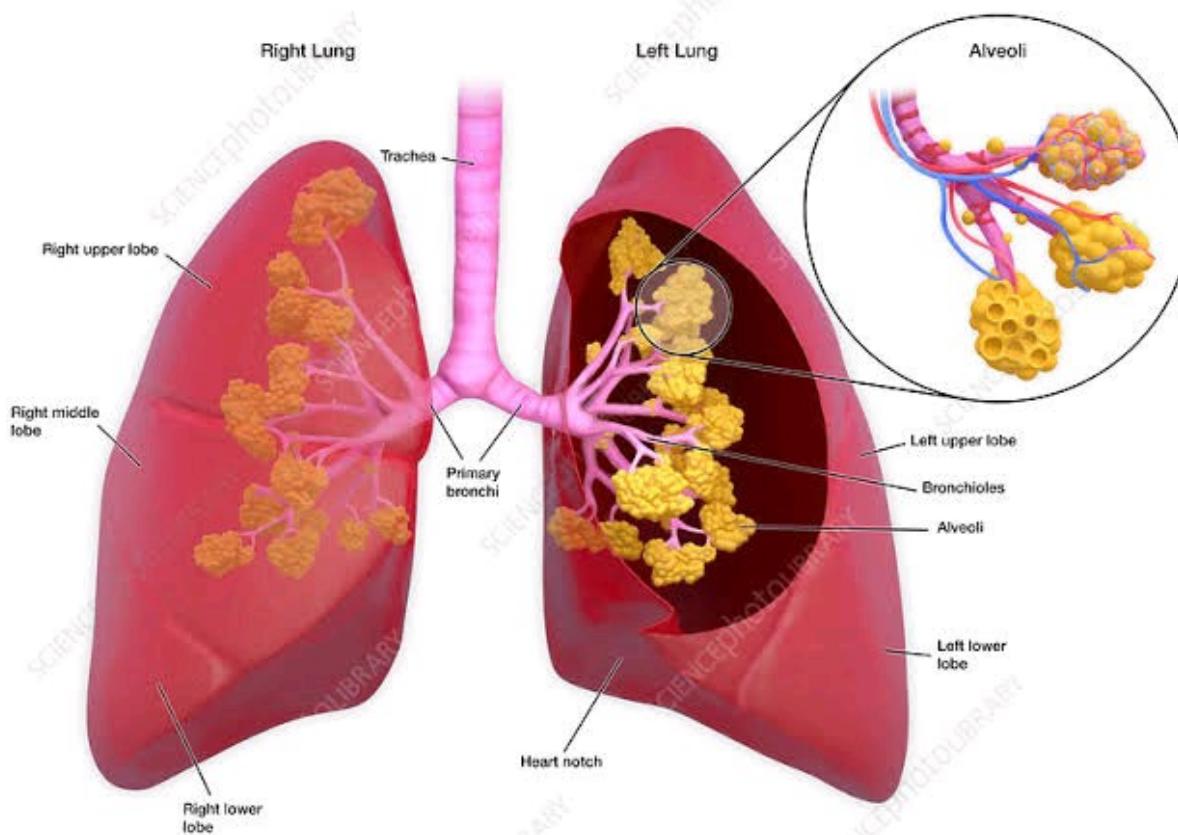


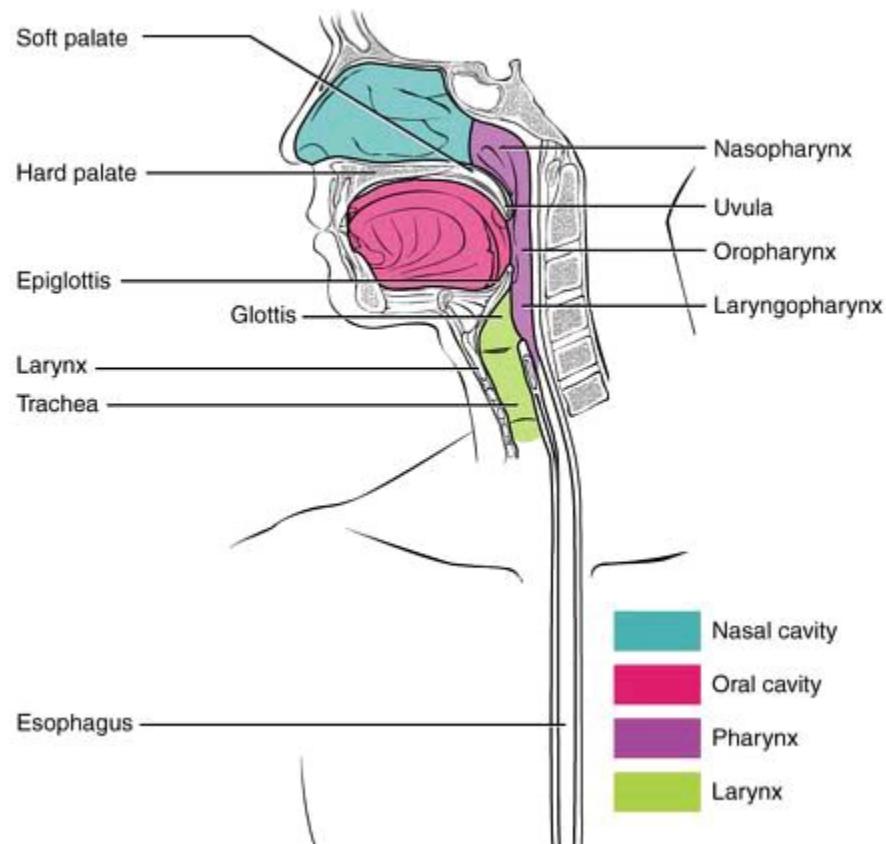
Fetal lung maturation

ACS not recommended for those with chorioamnionitis and those undergoing c/s for late preterms

ANATOMY

Nose/mouth, laryngopharynx (Epiglottis) Trachea, Bronchus, brochioles, alveoli (wet balloons)





ALVEOLI (WET BALLOONS)

Has alveolar fluid inside and interalveolar septum outside (elastic fibre containing capillaries and pores of kohn - balance interalveolar pressure)

Alveolar fluid: water, wbc, complement, electrolytes and surfactant,

Type 1 pneumocytes - alveolar epithelium and continuous with capillary endothelium (for gaseous diffusion)

Type 2- Make surfactant, replace type 1

ALVEOLI



SURFACTANT

Phospholipoprotein - DPPC mainly (90% phospholipids, 10% surfactant proteins)

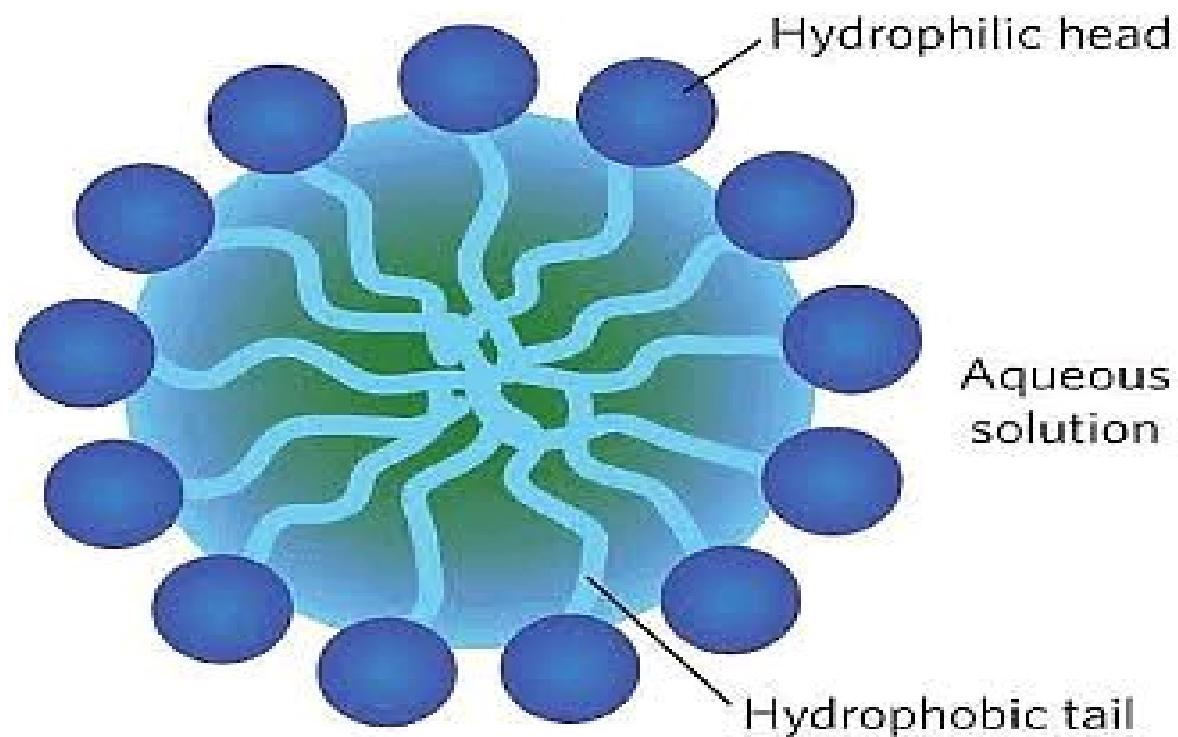
Hydrophilic head (phosphate and protein AD) faces alveolar epithelium and hydrophobic tail (lipid and proteins BC) tail faces the air forming micelle.

Mechanism;

Reduce surface tension of alveolar fluid

Prevent alveolar collapse and stabilize size, increase lung compliance (expansion and contraction) hence decrease work of breathing, prevent fluid accumulation (pulmonary edema).

MICELLE



Micelle Structure & Formation

DIAGNOSIS

History and Physical exam (risk factors, signs and symptoms)

Silverman Anderson Score (SAS)

0; No distress

1-3; Mild distress

4-6; Moderate distress

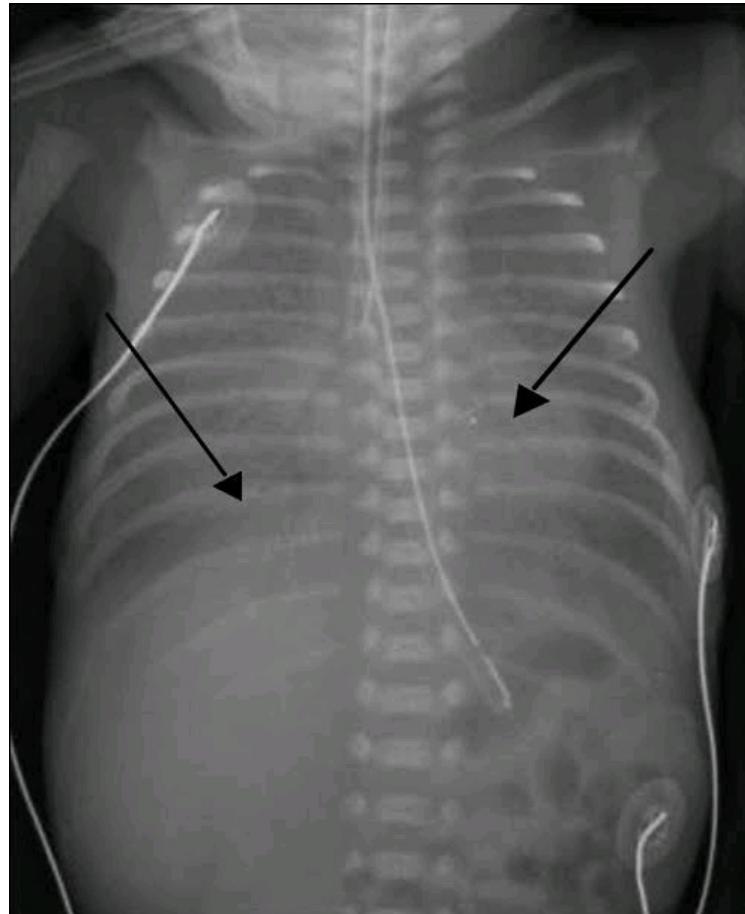
7-10; Severe distress

Feature observed	Score		
	0	1	2
Chest movement			
Intercostal retraction			
Xiphoid retraction			
Nares dilation			
Expiratory grunt			

INVESTIGATIONS

Spo2 (<90), rbs (hypoglycemia), temperature (hypothermia) fhg (leukocytosis, anemia), uec (aki), cxr (ground glass), BGA (acidosis), cranial ultrasound (IVH)

CXR-ground glass appearance of RDS



TREATMENT

ABC, resuscitate, oxygen, CPAP/ mechanical vent (higher PIP 20cm of water), synthetic surfactant, antibiotics, parenteral fluids and feeds, radiant warmer, caffeine citrate (reduce need for oxygen and cpap, prevents pda by better pulmonary mechanics, anti-inflammatory, increase angiogenesis and alveolarization, neuroprotective - myelination, treat AOP - stimulates respiratory center, sensitivity to co₂, muscle tone, oral or IV loading dose 20mg/kg, 5-10mg/kg maintenance). Methylxanthines?

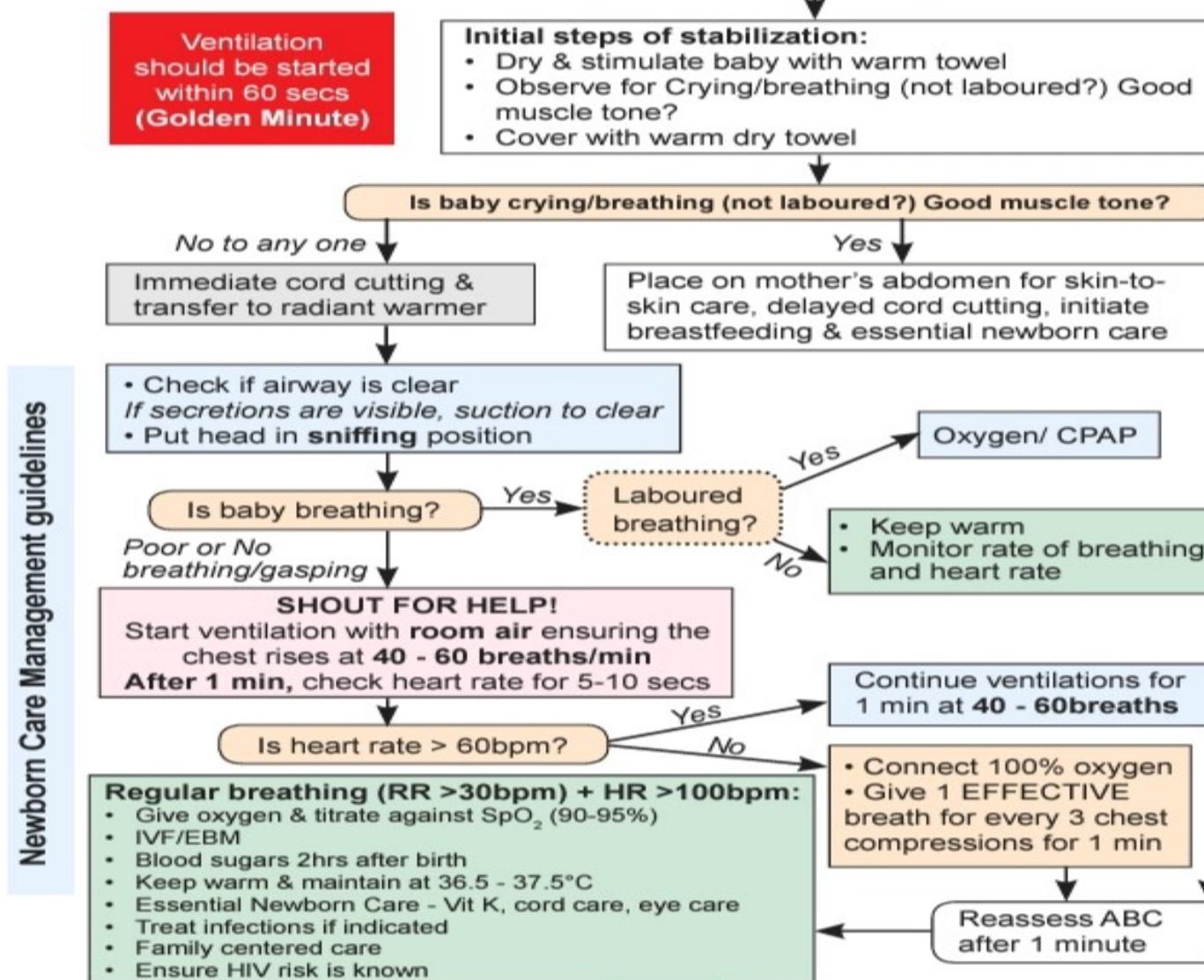
Neonatal Resuscitation

For trained health workers - Anticipate and prepare

Note for all newborns:

- Practice immediate cord clamping and cutting for newborns requiring resuscitation
- For newborns with good heart rate and spontaneous breathing, practice delayed cord clamping and cutting (1-3 mins)

PREPARE BEFORE DELIVERY - EQUIPMENT, WARMTH, GETTING HELP



VAYU bCPAP SYSTEM IN NBU HBCTRH



COMPLICATIONS OF RDS

Death, ROP (excess oxygen disrupts growth factors), IVH (cerebral perfusion and inflammation), PH (inflammation), BPD (excess o₂ causing dysregulated inflammation, proteases and fibrosis- chronic rds), CP (cerebral perfusion - motor development), AOP

Management of RDS

Approach to management



Keep warm and Maintain neutral thermal environment - Reduce oxygen consumption

Airway patency should be ensured

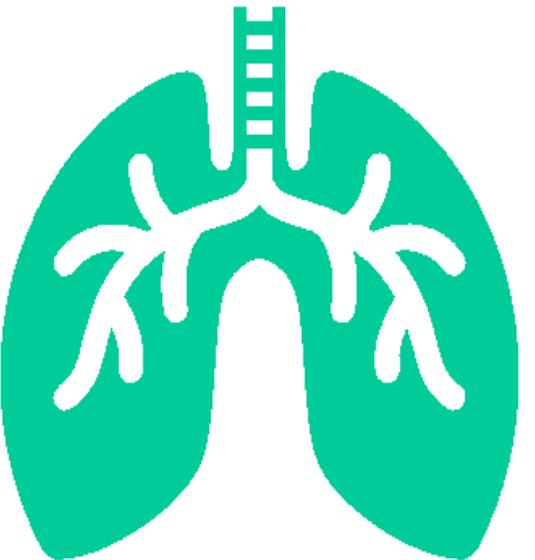
Breathing - Specific management; Surfactant use and Respiratory Support (CPAP)

Circulation- feeds and fluids; Initiation of early feeds & Maintenance fluids

D - Close monitoring of vitals; Blood sugars, Hypotension common in early RDS, Antibiotics, Caffeine

Continuous Positive Airway Pressure (CPAP)

- Non invasive method of **oxygen delivery**



Provides **continuous distending pressure** that's keeps alveoli open during expiration

Reduces work of breathing therefore improves oxygenation

Decreases atelectasis and respiratory fatigue

Why use CPAP?

In-utero



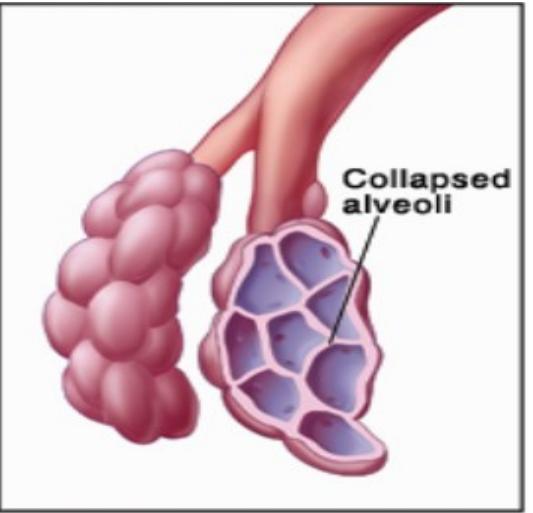
- Fetal lungs in utero remain distended due to the 2-4 cm H₂O maintained by the fluid in the fetal lungs

CPAP

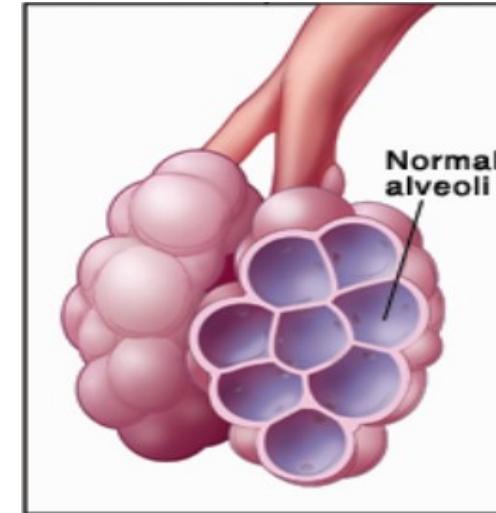


- CPAP mimics normal physiology.
- Constant distending pressure at 2-3cm H₂O.

Benefits of using CPAP



CPAP



1. Improves oxygenation
2. Continuous distending pressure keeps alveoli open which maintains functional residue capacity
3. Promotes lung growth and development.
4. Promote surfactant production

Prophylactic versus Rescue CPAP

Prophylactic CPAP



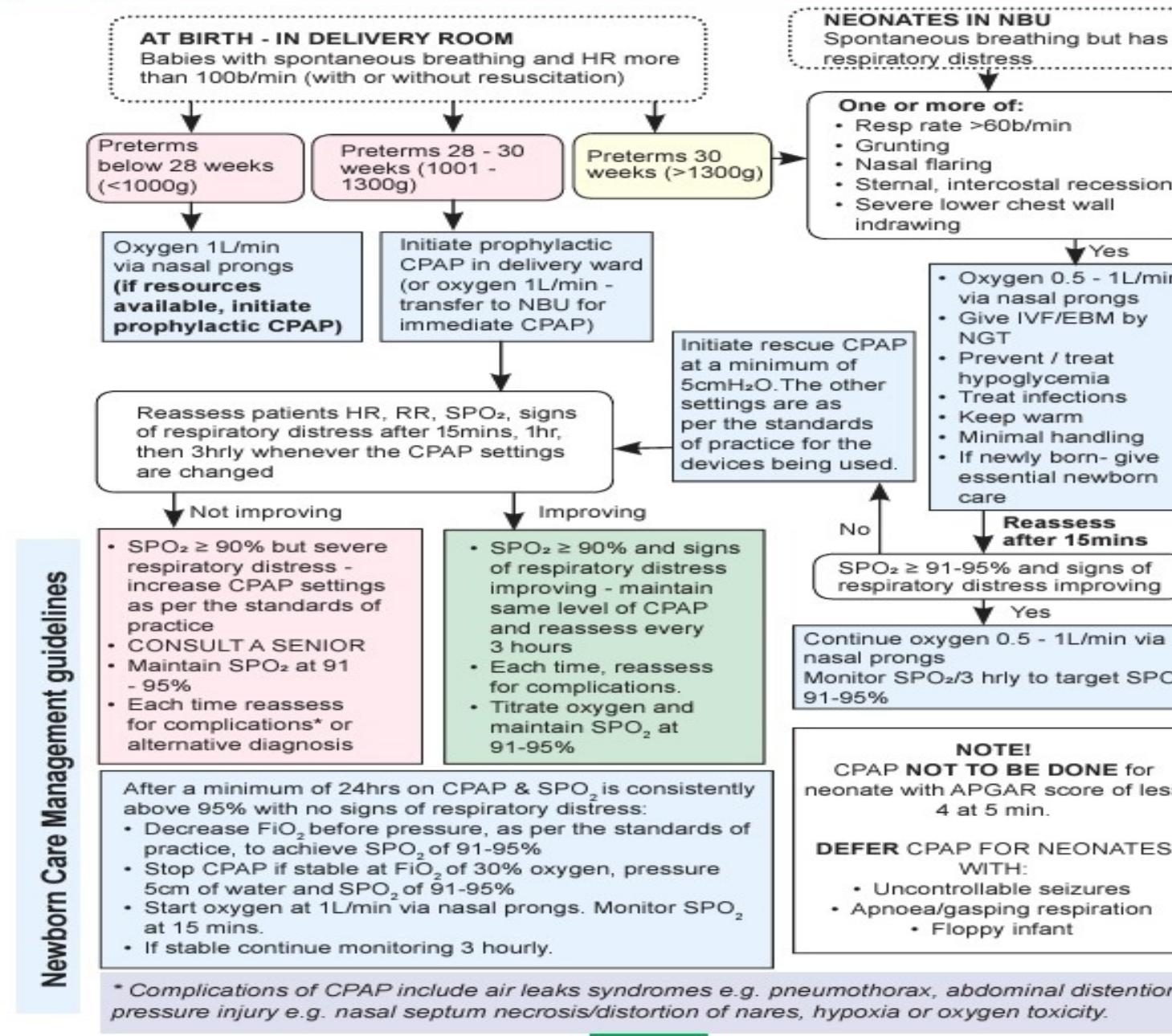
- **28 - 30 weeks (1-1.3kgs)**
- Initiated as soon as possible within the delivery room
- For the newly born with good cardiac activity and breathing spontaneously
- Not in respiratory distress
- Intended to avoid mechanical ventilation



Rescue CPAP

- **Above 30 weeks (>1.3kgs)**
- Initiated after trial of oxygen therapy
- Neonate with increased work of breathing and $\text{SpO}_2 < 90\%$ on nasal prongs at 1L/min

Continuous Positive Airway Pressure (CPAP)



CPAP

- Complications

Skin Complications

- Constant pressure on nares, ears, head and forehead can lead to reduced skin integrity and injury causing pressure ulcers.



First sign of skin breakdown is nasal erythema.

Prevention

- Frequent observation**
- Minimize points of contact**
- Keep skin dry and clean**
- Avoid tight fitting hat over forehead, ears and bony prominences**

Nasal Complications



Nasal septal injury



Nasal flaring



Nasal snubbing



Prevention

- a) Maintain 2mm distance between columella and nasal prongs**
- b) The prongs should fill the entire nare without blanching the external nare**
- c) Ensure appropriate size of the nasal prongs and positioning of the whole interface**

Lung Complications



Pneumothorax

CPAP increases risk of air leaks.

Prevention

- a) Always check CPAP pressure
- b) Do not exceed pressures of 8cm H₂O.
- c) Check for any air leaks in circuit

Lung Complications



Hyperinflation of lungs

- Occurs due to high CPAP pressures.
- Results in reduced cardiac output secondary to reduced venous return.

Prevention

- a) Always check CPAP pressure
- b) Do not exceed pressures of 8cm H₂O.

Abdominal Complications



Abdominal distention

- Excessive swallowed air
- Feeding intolerance and desaturation episodes.

Prevention

- a) Insert an OGT
- b) Leave OGT open
- c) If OGT is used for feeding, close for 30mins after feeding the baby then open OGT

Oxygen Therapy Complications

Hypoxia

SPO₂ - 85 - 89%

- Increases mortality
- Does not alter rates of developing;
 - a) Chronic lung disease- BPD
 - b) Blindness
 - c) Neurodevelopmental impairment.

Hyperoxia

SPO₂ - > 95%

- Free radicals that cannot be metabolized by immature antioxidant systems.
- Chronic lung disease - BPD
- Eye Injury - RoP

Prevention

- a) Monitor SpO₂
- b) Aim for O₂ saturation of 90-95%
- c) Titrate the FiO₂ based on SpO₂

CPAP Failure

CPAP leads to a 35% reduction in death and use of assisted ventilation¹.

It however can fail

A diagnosis of CPAP failure is made on any baby who has been on correctly applied CPAP for 72 hours and who continues to have;

1. Moderate to severe retractions and grunting
2. $\text{SpO}_2 < 90\%$
3. Recurrent apneas even on a maximum CPAP pressure of 8cm H₂O

Risk factors for CPAP failure



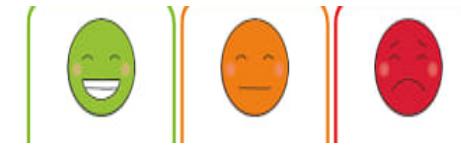
Newborn Characteristics

- Weight <1000g
- Gestation <28 weeks
- Sex - Male



Maternal Factors

- Poor antenatal steroid coverage
- PPROM



Severity of the Disease

- Moderate or Severe RDS
- Delayed onset of treatment

Summary

1. CPAP promotes lung growth/development and protects lung – all babies deserves the best care.
2. CPAP should be initiated at an FiO₂ of 50%, which then is titrated upwards or downwards to achieve oxygen saturation targets of 90-95%
3. Regularly monitor patient to optimize CPAP benefits and reduce risk of complications



