

# 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 ( $spo_2 < 90$ )

# 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)

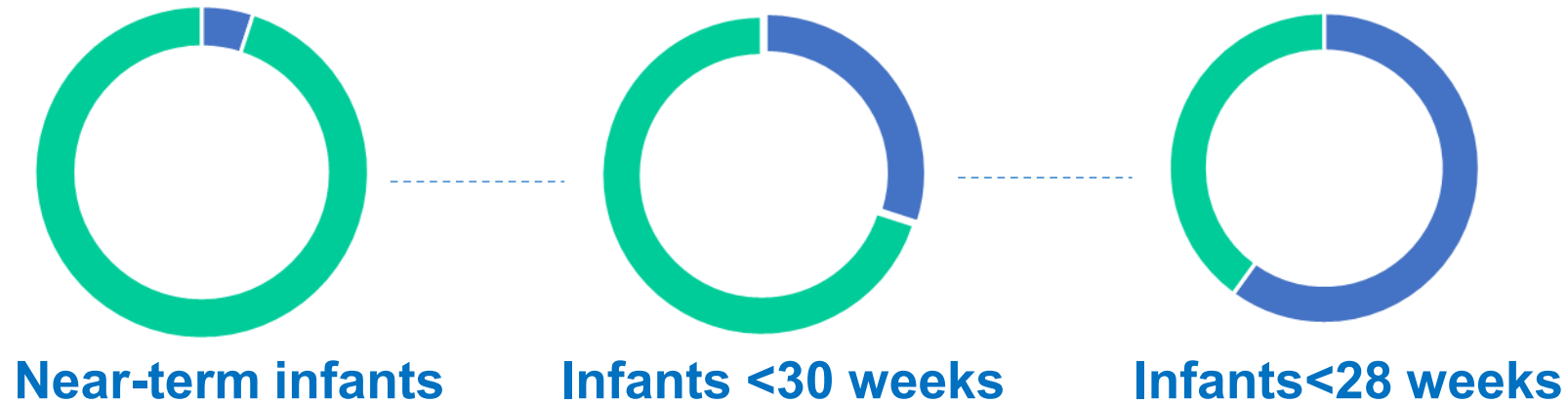
Besides prematures what reduces neonatal surfactant in older neonates;

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)

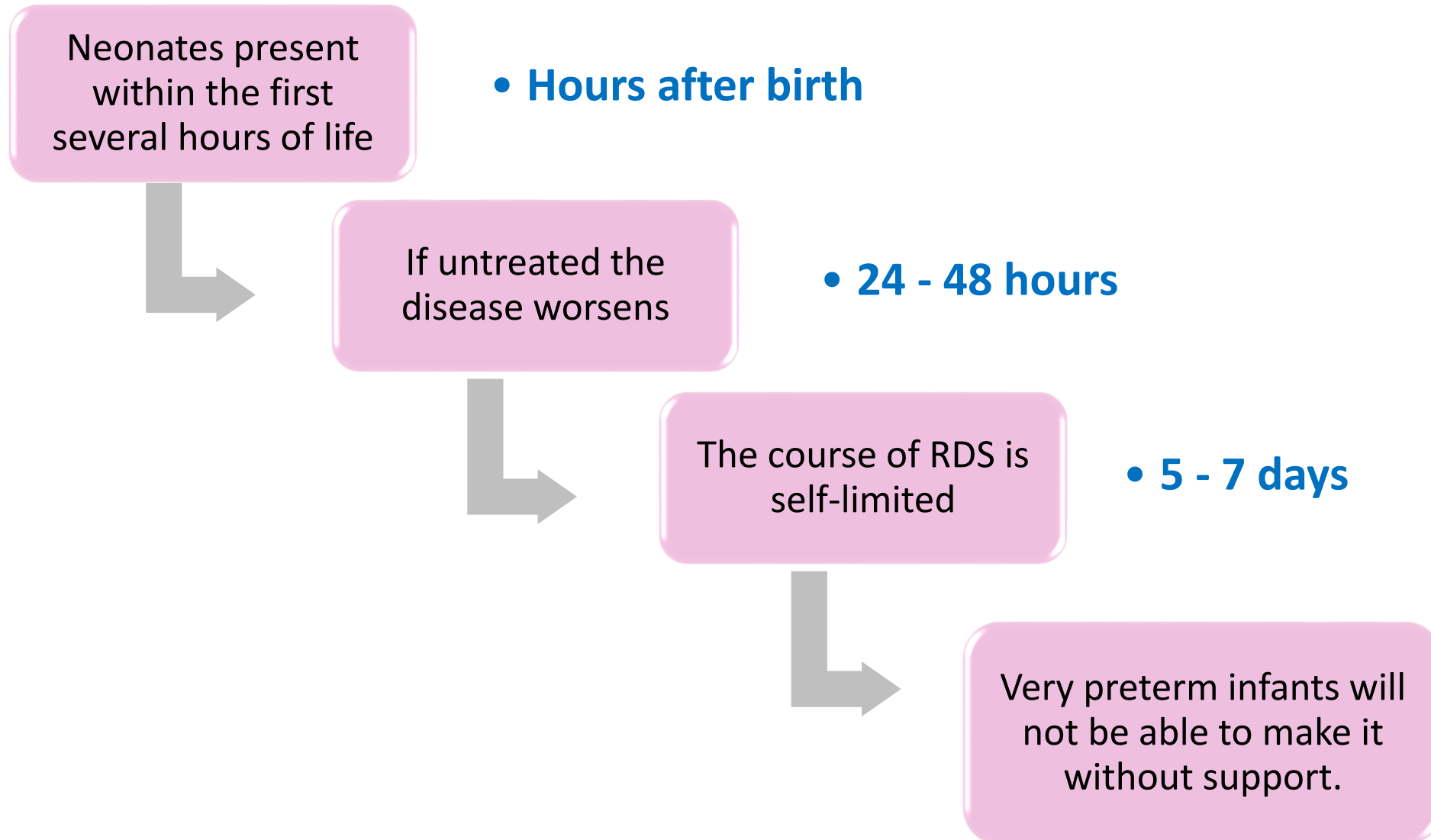
# RDS increases with decreasing gestational age

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

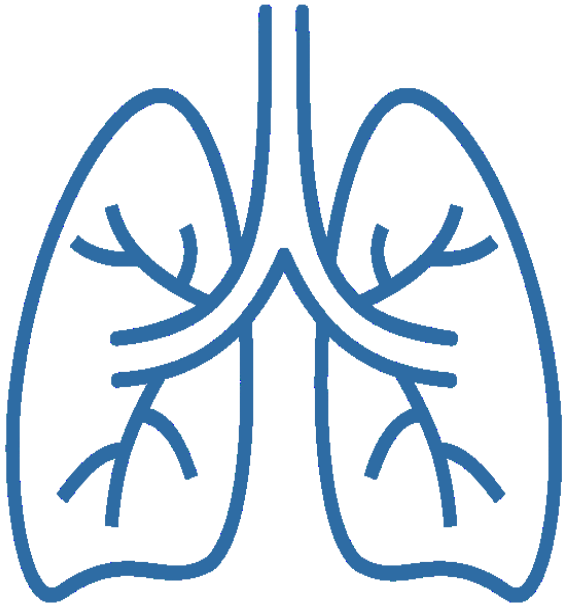


- RDS is seen soon after birth, worsens during the first few hours of life
- In contrast to Transient Tachypnea of the Newborn (TTN), worse at birth but improves within hours of birth

# Clinical Course of RDS



# Periods of Treatment for RDS



RDS was first described by Hochheim 14 in **1903**, who noted unusual membranes in the lungs of 2 infants who died shortly after birth

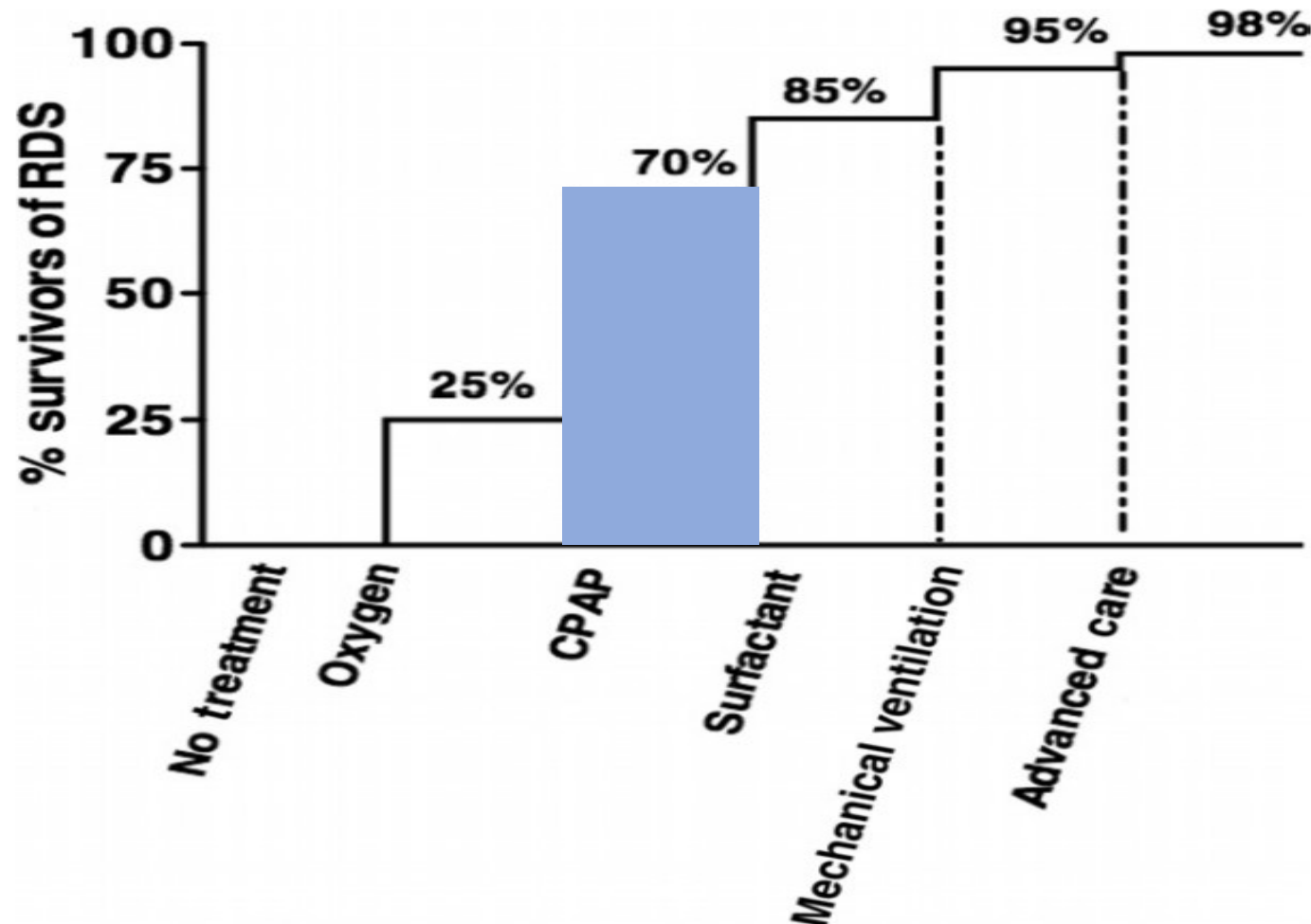
**Period 1 - Before 1950s:** No widely used treatment

**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

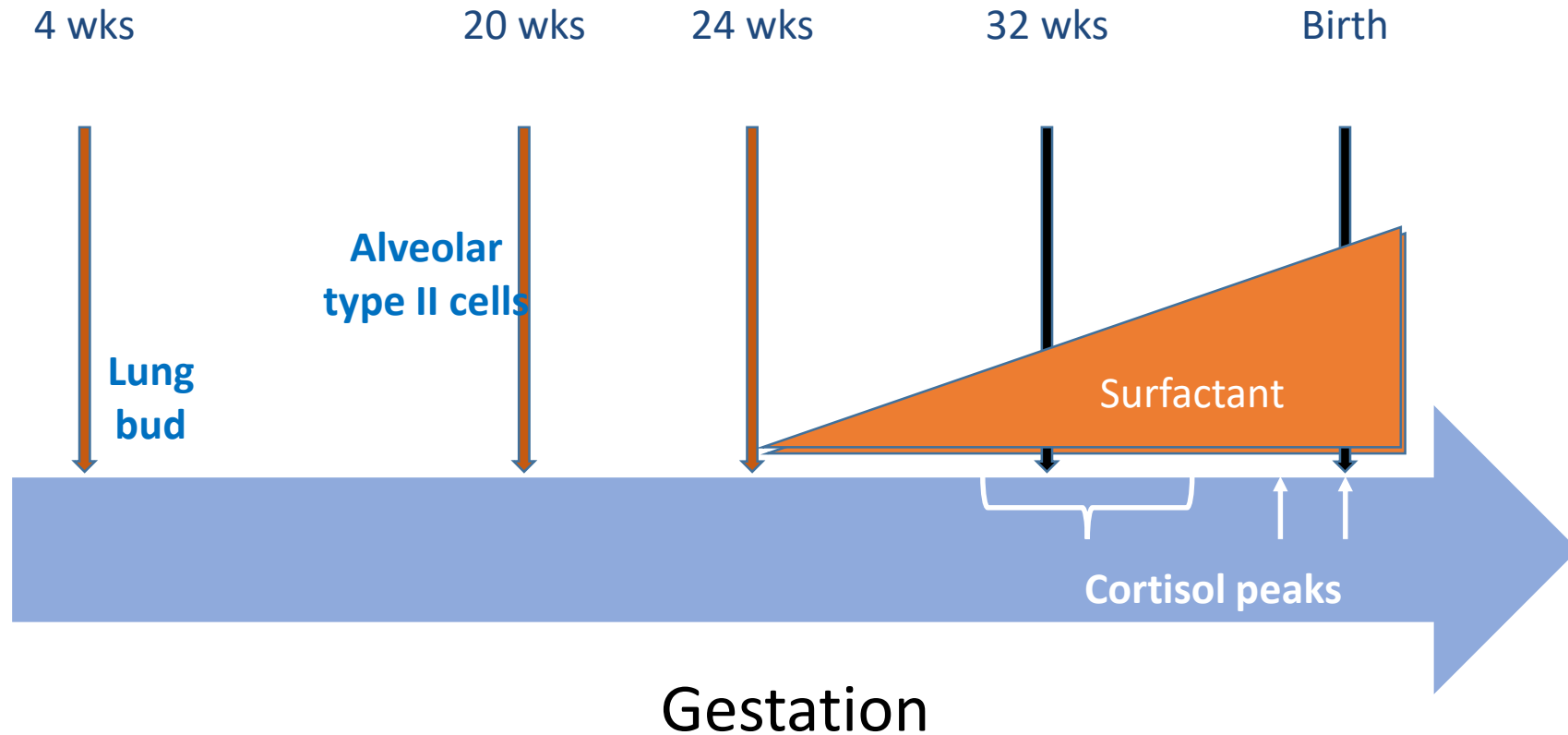
**Period 4 – After 1990:** Antenatal corticosteroids, surfactant, advanced care technologies e.g. high frequency oscillation

# Increased % in RDS survivors with introduction of specific treatments





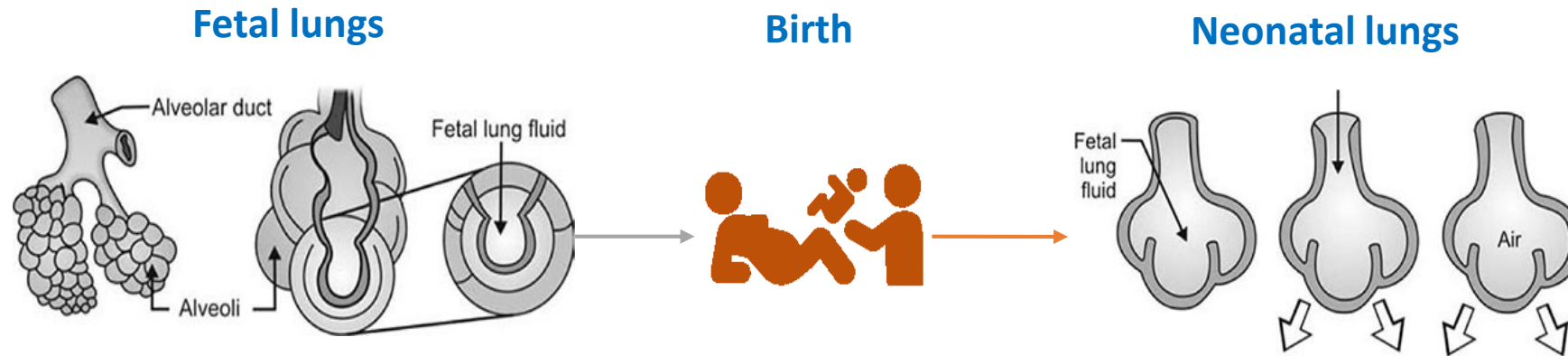
# Lung growth and Development



# 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 - 4cmH<sub>2</sub>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

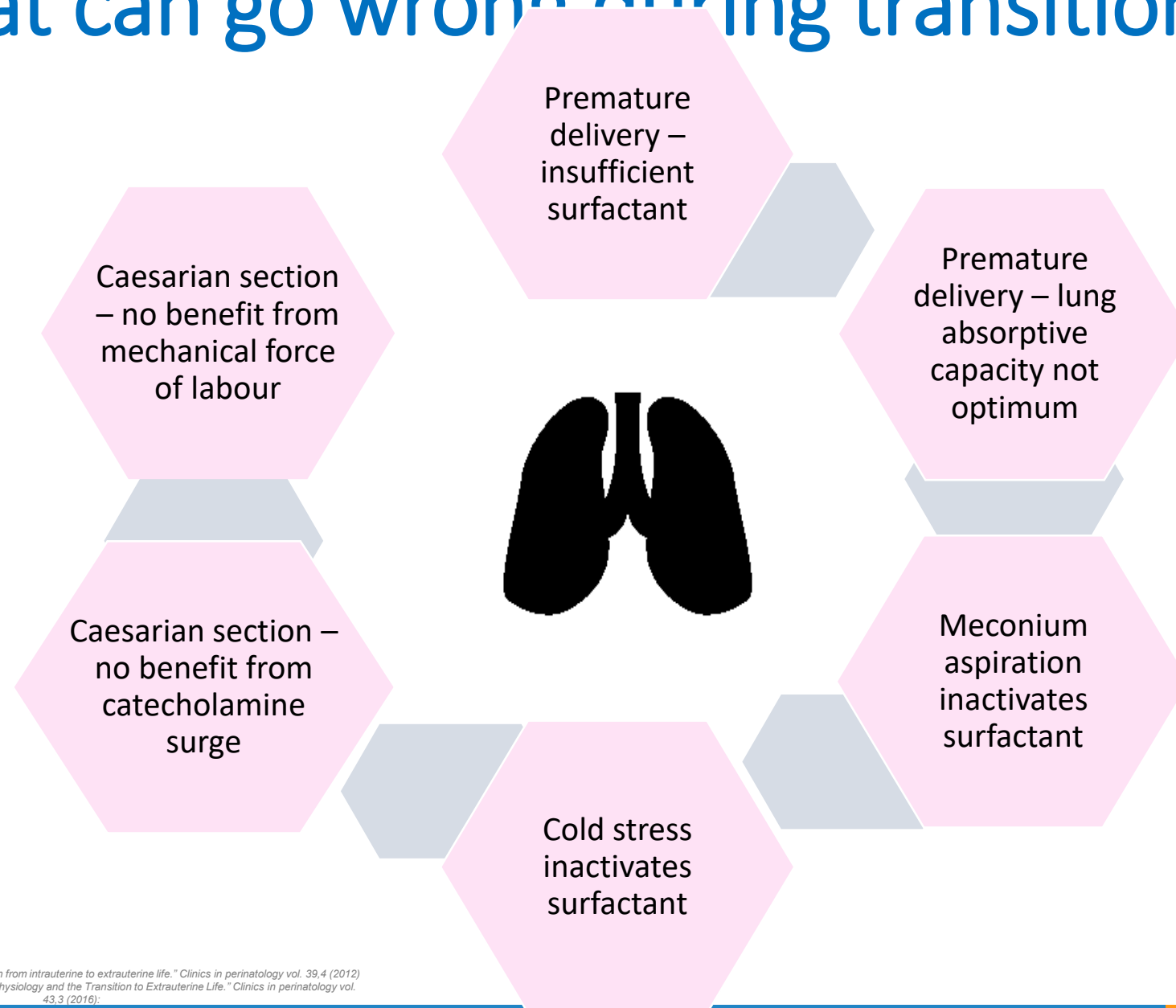
# Surfactant



- 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?



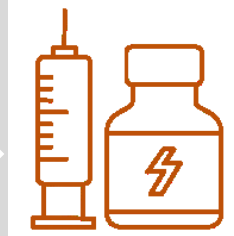
# Antenatal steroids and the fetal lung

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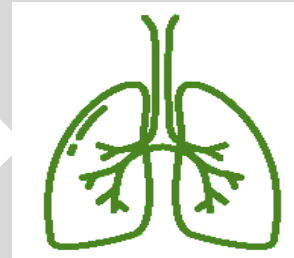
Improved antenatal care – to reduce risk of premature deliveries

2

Premature labour 24 - 34 weeks



Administration of antenatal corticosteroids (ACS)

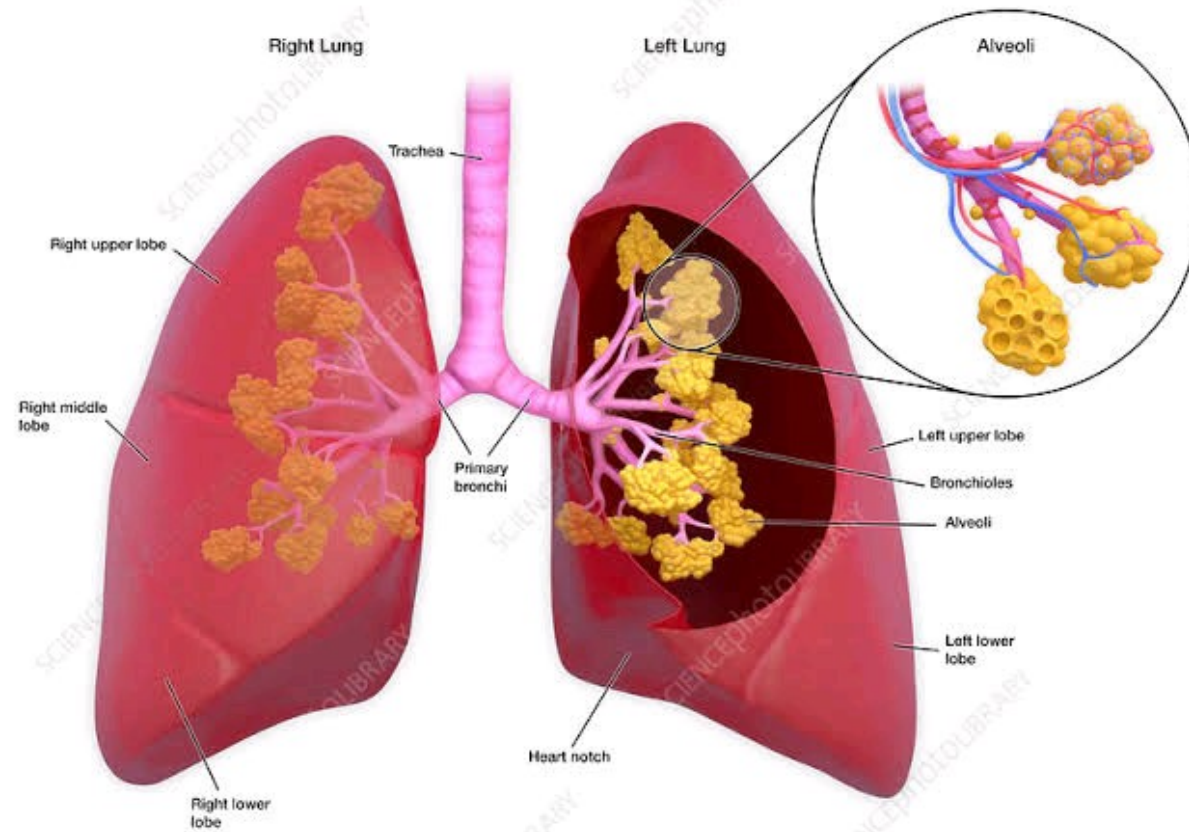


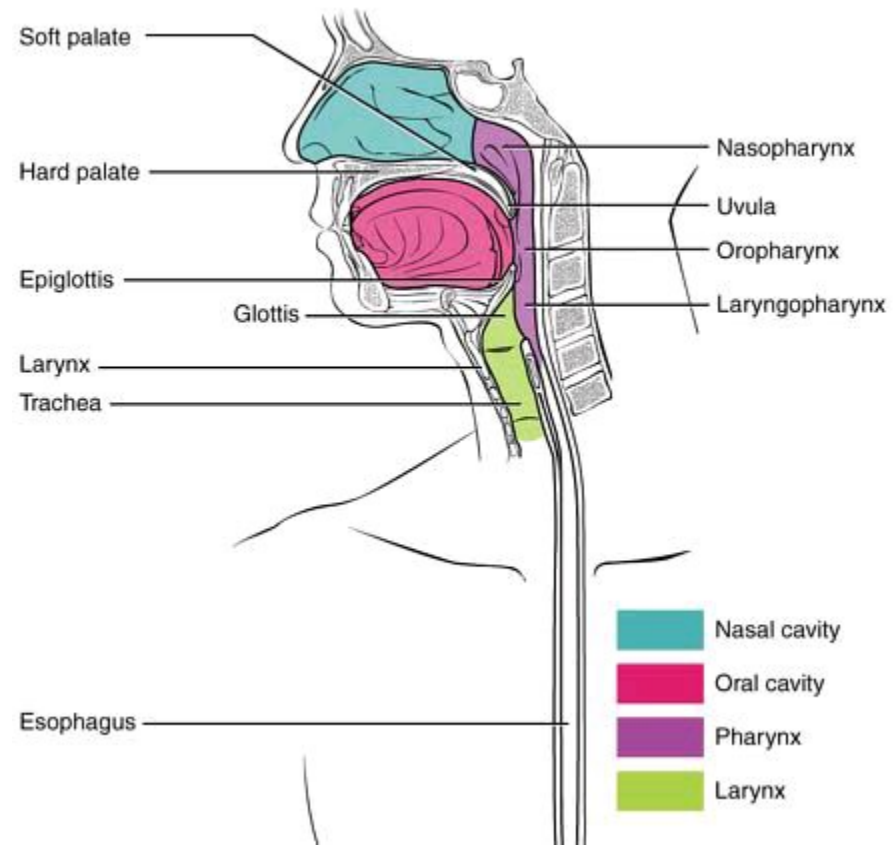
Fetal lung maturation

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

# ANATOMY

Nose/mouth, laryngopharynx (Epiglottis) Trachea, Bronchus, bronchioles, 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)

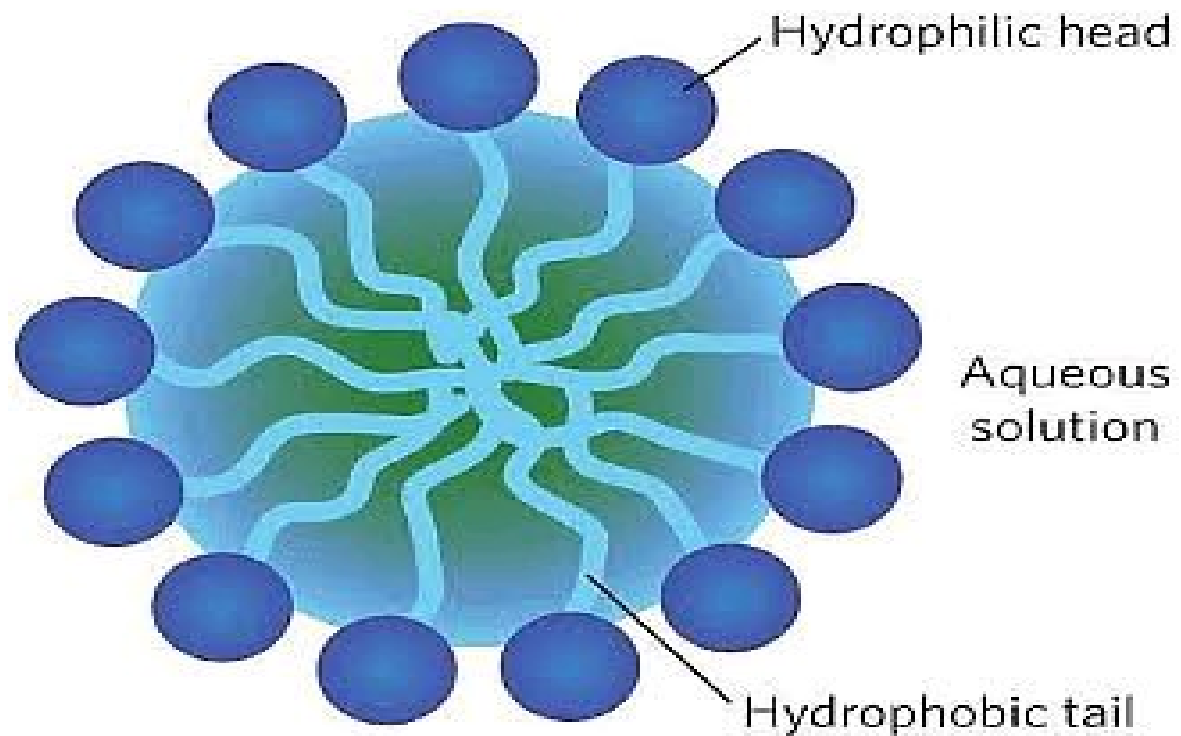
Hydrophilic head (phosphate and protein AD) faces alveolar epithelium and hydrophobic tail (lipid and proteins BC) tail faces the air forming micelle.

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

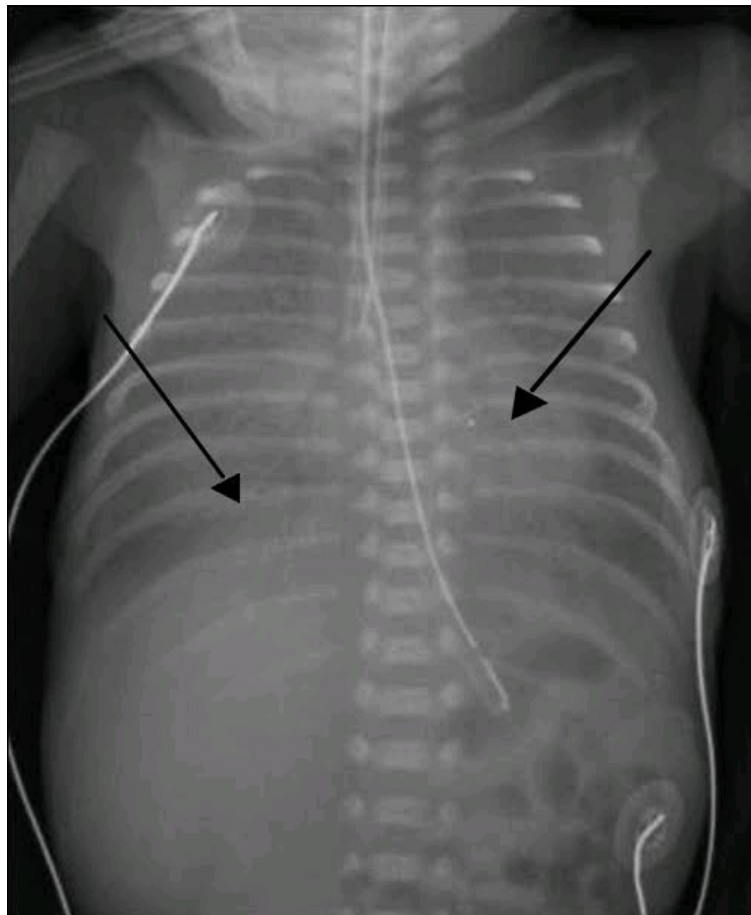
7-10; Severe distress

Feature observed	Score		
	0	1	2
Chest movement	 Synchronized respirations	 Lag on respirations	 Seesaw respirations
Intercostal retraction	 None	 Just visible	 Marked
Xiphoid retraction	 None	 Just visible	 Marked
Nares dilation	 None	 Minimal	 Marked
Expiratory grunt	 None	 Audible by stethoscope	 Audible by unaided ear

# 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

Ventilation  
should be started  
within 60 secs  
(Golden Minute)

### Initial steps of stabilization:

- Dry & stimulate baby with warm towel
- Observe for Crying/breathing (not laboured?) Good muscle tone?
- Cover with warm dry towel

Is baby crying/breathing (not laboured?) Good muscle tone?

No to any one

Immediate cord cutting &  
transfer to radiant warmer

Yes

Place on mother's abdomen for skin-to-  
skin care, delayed cord cutting, initiate  
breastfeeding & essential newborn care

- Check if airway is clear  
*If secretions are visible, suction to clear*
- Put head in **sniffing** position

Is baby breathing?

Yes

Laboured  
breathing?

Yes

Oxygen/ CPAP

No

- Keep warm
- Monitor rate of breathing  
and heart rate

Poor or No  
breathing/gasping

### SHOUT FOR HELP!

Start ventilation with **room air** ensuring the  
chest rises at **40 - 60 breaths/min**  
**After 1 min**, check heart rate for 5-10 secs

Is heart rate > 60bpm?

Yes

No

Continue ventilations for  
1 min at **40 - 60 breaths**

- Connect 100% oxygen
- Give 1 **EFFECTIVE**  
breath for every 3 chest  
compressions for 1 min

Reassess ABC  
after 1 minute

### Regular breathing (RR >30bpm) + HR >100bpm:

- Give oxygen & titrate against SpO<sub>2</sub> (90-95%)
- IVF/EBM
- Blood sugars 2hrs after birth
- Keep warm & maintain at 36.5 - 37.5°C
- Essential Newborn Care - Vit K, cord care, eye care
- Treat infections if indicated
- Family centered care
- Ensure HIV risk is known

Newborn Care Management guidelines

# 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  $O_2$  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

**A**irway patency should be ensured

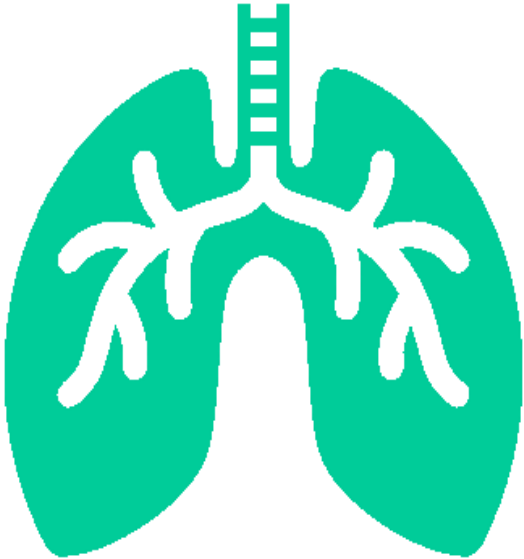
**B**reathing - Specific management; Surfactant use and Respiratory Support (CPAP)

**C**irculation- 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)

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- Non invasive method of **oxygen delivery**
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Provides **continuous distending pressure** that's keeps alveoli open during expiration

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Reduces work of breathing therefore improves oxygenation

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Decreases atelectasis and respiratory fatigue

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# Why use CPAP?

## In-utero



- Fetal lungs in utero remain distended due to the 2-4 cm H<sub>2</sub>O maintained by the fluid in the fetal lungs

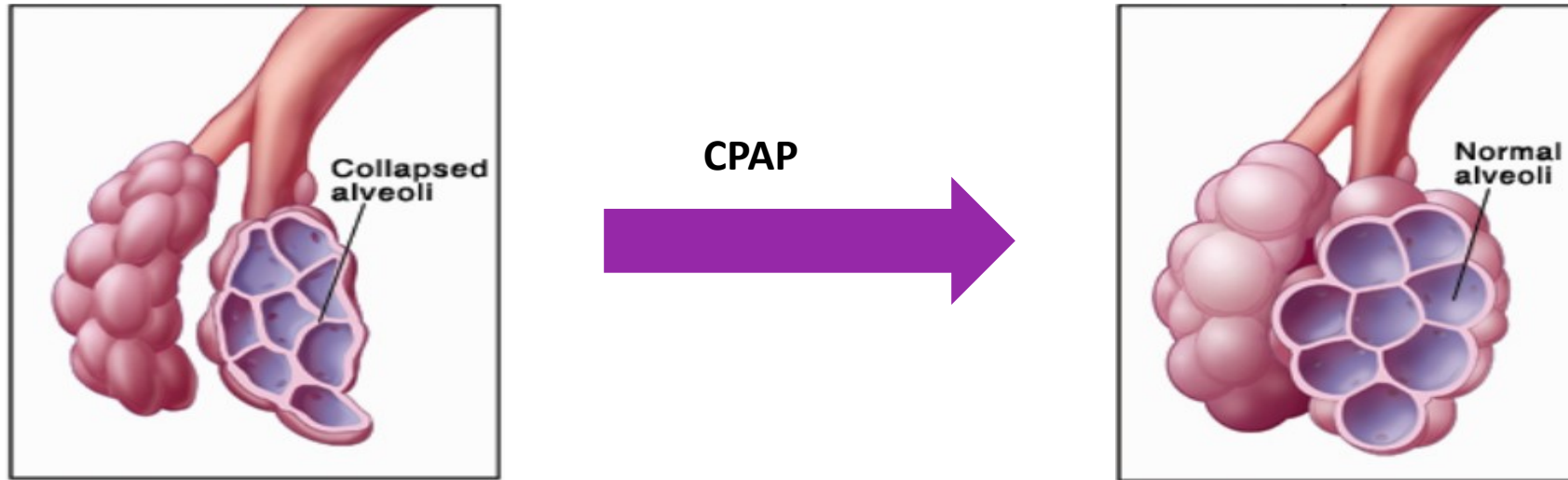
## CPAP



- CPAP mimics normal physiology.
- Constant distending pressure at 2-3cm H<sub>2</sub>O.



# Benefits of using 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

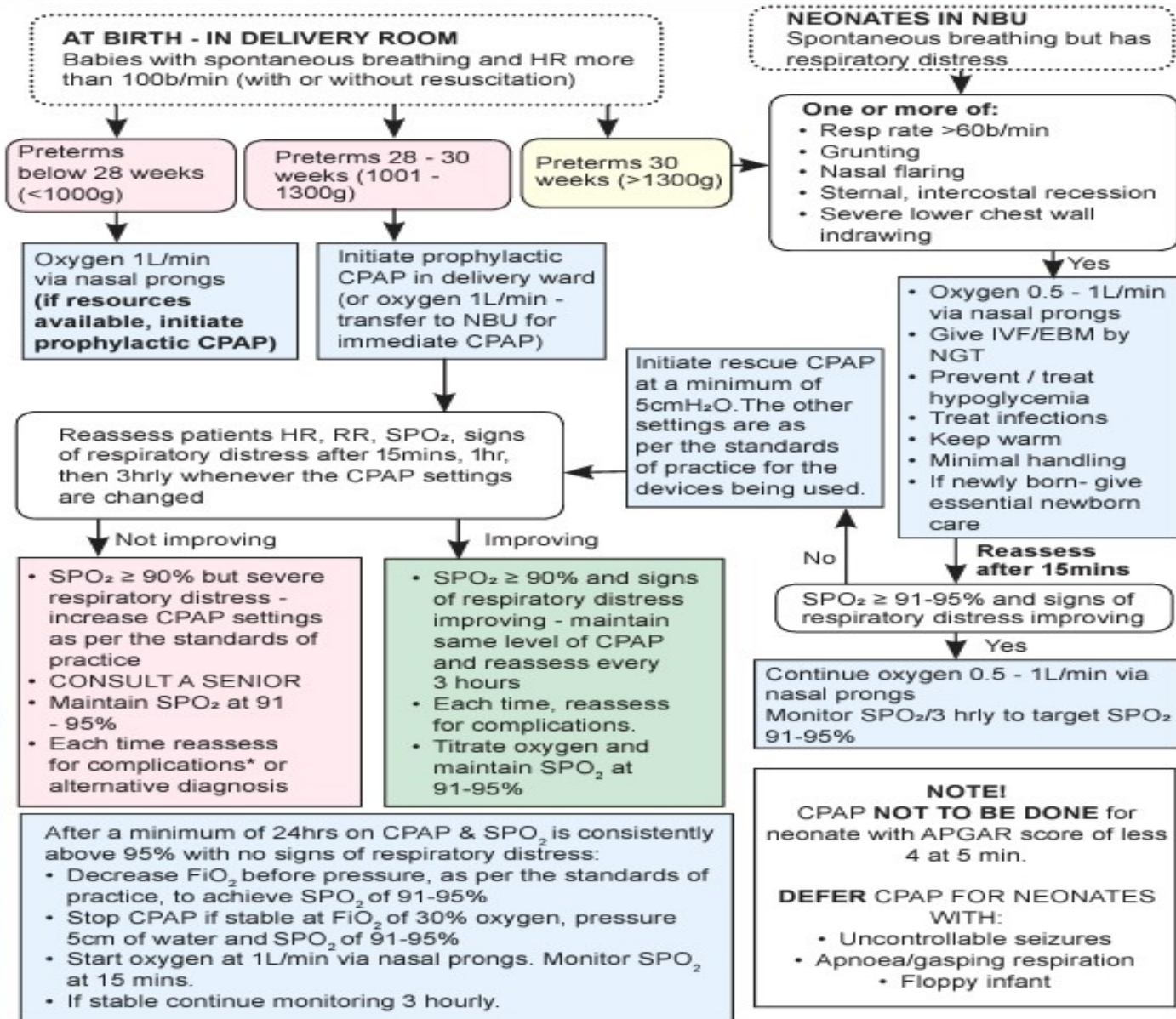


## Rescue CPAP

- **Above 30 weeks (>1.3kgs)**
- Initiated after trial of oxygen therapy
- Neonate with increased work of breathing and  $SpO_2 < 90\%$  on nasal prongs at 1L/min

# Continuous Positive Airway Pressure (CPAP)

## Newborn Care Management guidelines



\* Complications of CPAP include air leaks syndromes e.g. pneumothorax, abdominal distention, pressure injury e.g. nasal septum necrosis/distortion of nares, hypoxia or oxygen toxicity.

# 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 H<sub>2</sub>O.
- 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 H<sub>2</sub>O.



# 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

$\text{SpO}_2$  - 85 - 89%

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

## Hyperoxia

$\text{SpO}_2$  - > 95%

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

## Prevention

- a) Monitor  $\text{SpO}_2$
- b) Aim for  $\text{O}_2$  saturation of 90-95%
- c) Titrate the  $\text{FiO}_2$  based on  $\text{SpO}_2$

# CPAP Failure

CPAP leads to a 35% reduction in death and use of assisted ventilation<sup>1</sup>.

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 recessions and grunting
2.  $\text{SpO}_2 < 90\%$
3. Recurrent apneas even on a maximum CPAP pressure of 8cm  $\text{H}_2\text{O}$

# Risk factors for CPAP failure



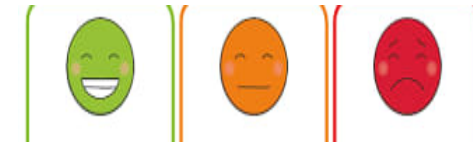
## Newborn Characteristics

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



## Maternal Factors

- Poor antenatal steroid coverage
- PPRROM



## 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 FiO<sub>2</sub> 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







