MultiCare Women & Children's Grand Rounds



Respiratory Distress in the Term and Late Preterm Infant

Melinda Hendrickson, M.D.

MultiCare/Tacoma General Neonatologist

Pediatrix Medical Group



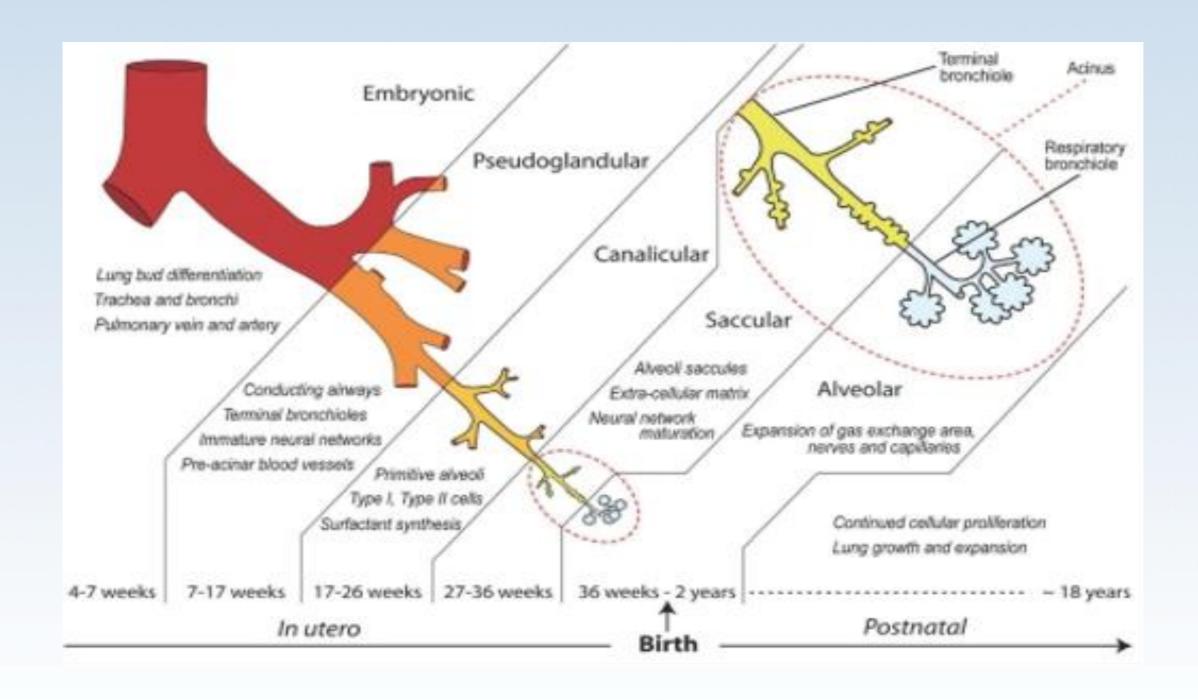


Overview

- Lung development and transition at birth
- Factors affecting lung maturity in newborn
- Case examples of respiratory distress
 - RDS
 - meconium aspiration syndrome
 - pneumonia
- Management of infant with respiratory distress



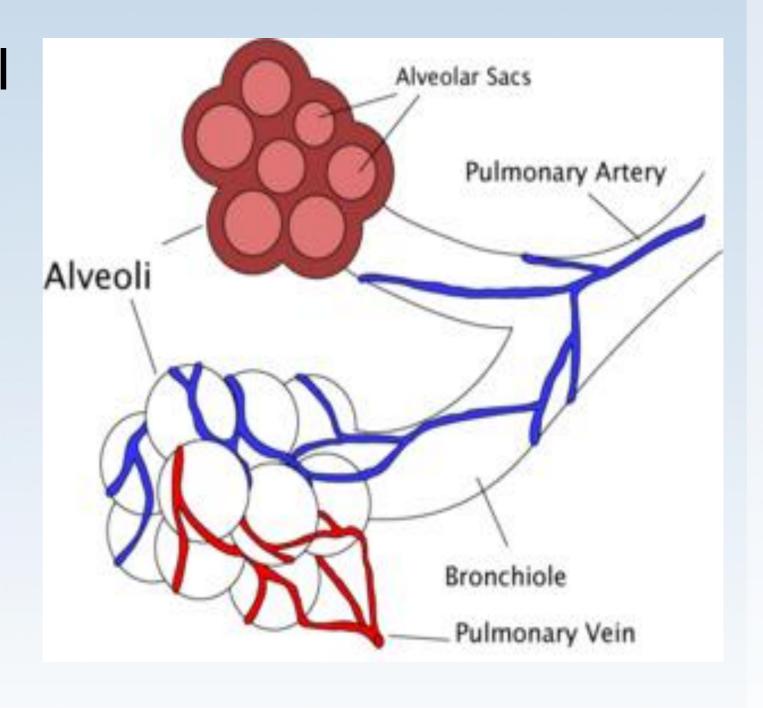
Development of Human Lung



Lung Development

Lung Volume 7	7 (36)	113 (52)	217 (100)
			211 (100)
Surface area (m²) 1.	.2 (27)	2.0 (44)	4.5 (100)
Alveolar wall thickness (μm)	.7 (161)	16.4 (134)	12.2 (100)

Fetal lung fluid critical for lung development Fetal lung fluid cleared at birth-prenatal, labor and postnatal Lung distention with air recruits alveoli/air sacs promoting oxygenation





Lung Development

- Alveolarization in the 3rd trimester
- Type 2 cells appear in pseudoglandular phase
- Type 2 cells are progenitor for Type 1 cells
- Type 2 cells important in surfactant metabolism and secretion
- Surfactant production in cannalicular phase but deficient prior to 32-34wks



Pulmonary Surfactant

- Composed of mostly lipid with majority dipalmitoyl phosphatidycholine (DPPC)
- Less than 10% surfactant proteins SP-A, SP-B, SP-C, SP-D
- Surfactant coats the lung surface after secretion by Type 2 cell and remnants are recycled by Type 2 cells (decreases de novo synthesis)



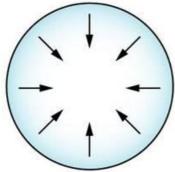
Laplace's Law: pressure required to keep alveoli distended in directly proportional to the surface tension and indirectly proportional to the radius

Surfactant decreases surface tension decreasing the pressure needed to keep the alveoli open increases lung compliance increases FRC stabilizes alveoli reduces V/Q mismatch

According to the law of LaRlace, if two bubbles have the same surface tension, the smaller bubble will have higher pressure.

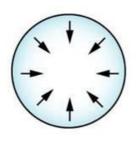
(a) Pressure is greater in the smaller bubble.

(b) Surfactant reduces surface tension (T). Pressure is equalized in the large and small bubbles.

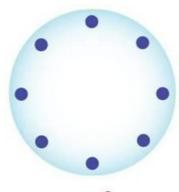


Larger bubble r = 2

T = 3 P = (2 × 3)/2 P = 3



Smaller bubble





Surfactant Production

- Accelerated production
 - chronic HTN, PIH
 - PPROM, chorio
 - corticosteroids
- Delayed
 - diabetes
 - Rh isoimmunization
 - twin, male
- Inactivation
 - meconium, pulmonary edema, infection

Late Preterm Infant

- Defined as infants born at 34 0/7 wks through 36 6/7 wks gestation
- Proportion of US births increased from 7.3% (1990) to 9.1% (2005)
- 70% all preterm births (2005)
- Increased risk of morbidity and mortality
- Respiratory distress, apnea, jaundice, hypoglycemia, temp issues

Respiratory Distress in the LPI

- 36% admitted to level 2+ nurseries and 10% admitted for resp. issues
 - RDS
 - TTN
 - Apnea/bradycardia
 - Pneumonia
- odds of developing RDS decreased with each week of maturity until 38 wks



RDS in LPI

- 34 wks: 7% treated with surfactant, 6% ventilator support
- 38 wks: 0.2% treated with surfactant, 0.5% ventilator support
- no difference in type of ventilator support

- 33 wks 2800 gm male to G8P6 mom with PIH, steroids x 2, mag, SVD
- DR: mask CPAP for low tone
- NICU: nCPAP/25% O2
 - CBG pH 7.28 pCO2 58 (1 hr of age)
- 29 hr of age: nCPAP/42% O2
 - VBG pH 7.26 pCO2 67
 - surfactant given (intubated/extubated)

- Surfactant given and nCPAP resumed
 - 1 hr nCPAP/28%, pH 7.26 pCO2 57
 - 6 hr nCPAP/RA, pH 7.28 pCO2 54
- CPAP stopped dol 5
- Baby remained in RA without resp. issues
- Discharged to home 3 wks of age

Key Points

- Risks for resp. distress: prematurity (33 wks), male, magnesium
- Management in DR: mask CPAP
- Management in NICU: CPAP continued, surfactant when O2 needs increased, CPAP continued until resp. distress/O2 need resolved.



How does CPAP work?

- Decreases upper airway occlusion (decreases airway resistance)
- Decreases obstructive apnea
- Increases functional residual capacity of the lung (FRC)
- Promotes surfactant conservation
- Prevent/treat microatelectasis and improve V/Q mismatch



CPAP

- delivered via T-piece or anesthesia bag with mask or nasal prongs
- OG tube placed and vented
- PEEP goal 4-6 cm
- patent airway critical (positioning, secretions)
- risk of pneumothorax, nasal septal trauma, skin breakdown

- 37 4/7 wk 4255 gm BG born SVD to G4P1 IDDM,
 +GBS, ROM x 20 hr
- DR: shoulder dystocia, Apgars 5 and 8
- Nursery: grunting/cyanosis, O2 via NC
- 30hr of age: O2 increased to >50%. CXR showed PTX.
 CBG pH 7.3 pCO2 63
- Transfer to NICU for CPAP
- nCPAP/25% with CBG pH 7.32 pCO2 56
- dol 5 CPAP stopped
- dol 6 normal CXR

- 39 wk 3800 gm male IDM repeat c/s
- DR: Apgars 4/6/8, PPV x 3 min
- Nursery: NC O2 for low sats/distress, hypoglycemia treated, abx
- NICU Transport Team: nCPAP/40% with CBG pH 7.3 pCO2 50
- 12 hr of age: nCPAP/RA
- 30 hr of age: room air, no support

Key Points

- Risks for respiratory distress: maternal diabetes
- Management:
 - CPAP not continued after initial resuscitation
 - supplemental O2 given for distress which progressed over few hours
 - CPAP decreased O2 need/work of breathing and improved blood gases
 - CPAP continued until resp. distress resolved



- 36 wks 2945 gm BG to G2P1 +GBS, PROM x 9 hr, abx given
- DR: SVD with nuchal cord, Apgars 2/7, PPVx few minutes then mask CPAP
- Nursery: continued mask CPAP (T-piece) 50% O2 until NICU Transport Team arrived
- Transport Team: ET, surfactant, ventilator
- dol 5 extubated to CPAP/room air
- dol 6 CPAP stopped
- WBC 4.8 (ANC 624), blood culture neg, LP tried x 2, CXR patchy infiltrates

Key Points

- Infection/pneumonia can inactivate surfactant in term and preterm infants
- CPAP started in DR for distress and continued (T-piece CPAP)
- CPAP provides transition from ventilator to RA following extubation



- Meconium stained amniotic fluid occurs in 8-20% of all deliveries
- Rare before 34 wk GA, incidence increases with gestation
- MSAF is NOT indicative of fetal distress
- Meconium Aspiration Syndrome may occur in 4% of deliveries with meconium stained fluid

- Meconium is a viscous, green liquid in the fetal GI tract
- Passage of meconium may be maturational physiologic event or response to hypoxemia
- Fetal distress causes fetal gasping and in utero aspiration of MSAF
- Meconium in airways may be aspirated with first few breaths after birth

- Pulmonary problems caused by airway obstruction and ball-valve effect
- Areas of atelectasis and overexpansion (air trapping)
- Chemical inflammation leads to pneumonitis causing interstitial edema, alveolar epithelial necrosis, surfactant inactivation



- Respiratory distress develops near birth and progresses, appear barrel chested, rales and ronchi
- CXR diffuse patchy infiltrates, areas of hyperinflation, air leaks (20-50%)
- Management may include oxygen, CPAP, ventilation, antibiotics, surfactant, ECMO
- Symptoms may persist for days/weeks
- Mortality decreased from 30% to <10%, infants at risk of developing chronic lung disease & neurodevelopmental sequelae

- 39 wk 2890 gm AGA BG born SVD to G7P1 mom with MSAF
- Cried spontaneously, Apgars 9 and 9
- Retractions/cyanosis, RA sat <80%
- 100% O2/NC then intubated/PPV
- NICU transport team started iNO for sats less than 80% on 100%O2
- CXR: meconium aspiration

- TG NICU started HFOV/surfactant
 - pre-surf ABG pH 7.36 pCO2 33 pO2 112 (FiO2 0.9)
 - post-surf ABG pH 7.32 pOC2 35 pO2 134 (FiO2 0.45)
- iNO dc 20 hr, 30 hr HOFV stopped and baby on SIMV/RA
- extubated dol 3 to NC/30% O2
- dol 4 RA, discharged dol 18

- 40 wk 3676 gm AGA BG born c/s for chorio, FTP with MSAF
- DR: ET with mec below cords, Apgars 2/5/6, PPV x 2 min then CPAP x 7 min
- NC started in nursery
- CXR: aspiration

- NICU Transport Team started CPAP and O2 weaned from 100% to 40%
- dol 3 CPAP/room air
- dol 5 CPAP stopped
- dol 8 discharged to home

Key Points

- Meconium aspiration may inactivate normal surfactant stores in term infants
- Surfactant may be considered in term infants with MAS requiring O2 with or without ventilation
- CPAP may reduce work of breathing and atelectasis leading to decreased O2 exposure, ventilator support and lung injury

Infant presents with respiratory distress

- Temperature
- Blood sugar
- History/infection risks
- Maternal medications including magnesium, narcotic, anesthesia
- Exam: cardiac, lungs
- Pulse ox



Infant presents with respiratory distress

- Mild (tachypnea): pulse oximetry, supplemental O2 per NRP guidelines
 - improvement/resolution: continued monitoring, routine care
 - persistent/recurrent: CPAP, CXR, blood work, consult NICU



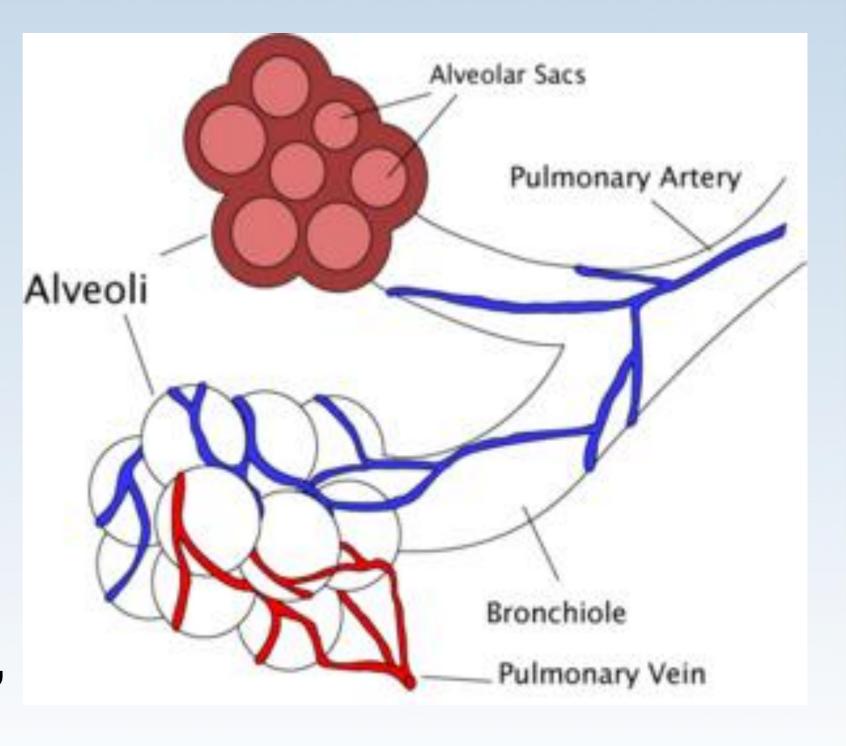
Infant presents with respiratory distress

- Severe (grunting, cyanosis, retractions): pulse ox, supplemental O2 per NRP guidelines, CPAP
- improvement/resolution: monitor
- persistent: NICU consult, CPAP, CXR, blood gas, consider abx/intubation/surfactant



Lung distention with

air recruits alveoli/air sacs promoting oxygenation CPAP, PPV, intubation Maintenance of lung volume essential CPAP, intubation, surfactant, ventilation



Take Home Points

- Late preterm infants remain at risk of significant respiratory issues leading to nursery admissions or transfers
- Respiratory distress in the newborn often results from suboptimal lung expansion following birth
- PEEP/CPAP recruits and maintains lung volume



Questions?

CME credit

 Physicians must successfully complete the post test (80% correct answers) to obtain CME credit. Non physicians may obtain a participation certificate, if they successfully complete the post test.

To access the post test, please cut and paste the following link into your browser:

http://www.surveygizmo.com/s3/1241995/WCGR0513

