Respiratory Complications in the Newborn
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Objectives
- Describe components of the respiratory assessment of the newborn recognizing the importance of accurate and timely assessment.
- Describe the pathophysiology and clinical presentation of selected respiratory complications in the neonate.
- Discuss strategies and nursing care to support respiratory function for infants experiencing compromised respiratory function.

Fetal to Neonatal Respiratory Transition
- 10% of fetal blood volume perfuses the lungs, rest is shunted through the patent ductus arteriosus and patent foramen ovale.
- Fetal lungs are filled with fetal lung fluid.
- Composition is different than amniotic fluid—probably more chloride, though varies by species.
- Production is an intrinsic metabolic function of developing lung epithelium—comes up the trachea where some is then swallowed and the rest mixed with amniotic fluid.

Fetal to Neonatal Respiratory Transition, cont.
- Based on research in sheep, it is thought that there is an epinephrine mediated clearance of fetal lung fluid initiated by the onset of labor—results in ~65% clearance by delivery.
- With the first breath the alveoli expand, fill with air and the lung fluid is absorbed into the lung tissue allowing oxygen to diffuse into blood vessels surrounding the alveoli.
- Increased systemic BP when the UAs and UV are clamped.
- Vessels in the lung tissue relax, decrease resistance to blood flow secondary to increased O2 and distension of the alveoli.
- Results in lower PA than systemic pressure and decreased flow through the ductus and foramen ovale.

Physical Assessment
- History—maternal risk factors
  - Pregnancy related illnesses (diabetes, PIH, STDs, infection)
  - Duration of labor and timing of ROM
  - Description of amniotic fluid (bloody, foul, meconium)
  - Labor meds
  - Route of delivery
- History—fetal/infant risk factors
  - Resuscitation required at birth/ADGARs
  - Gestational age
  - Since delivery: feedings, VS, hypoglycemia
Physical Assessment—Inspection

- Color
  - Pink and well perfused
  - Pale
  - Ruddy
  - Cyanotic
  - Acrocyanosis is normal for up to 48hrs after delivery—should not return after resolution.

- Tone & Activity
  - Should be well flexed
  - Alert and active if awake

- Respirations
  - Rate: normal = 40-60/minute
  - Quality
    - Diaphragm is the primary muscle of respiration in the neonate
    - Nasal flaring (increase diameter of nares)
    - Retractions: “drawing in” of the skin under the ribs
      - subcostal (below), intercostal (between), suprasternal/clavicular
      - to inflate the lungs
    - Grunting: exhaling against a partially closed glottis
      - whine, grunt, singing with each exhalation
  - Pattern
    - Irregular and brief pauses are normal
    - Periodic (vigorous breaths followed by up to a 20 second pause) common in preterm, can be normal for term

Physical Assessment—Inspection, cont.

- Secretions
  - Attention to quantity and quality
  - Usually increased after delivery, but shouldn’t persist
  - Excessive or frothy can be seen in esophageal atresia

- Chest shape
  - Symmetric, round shape with AP diameter approx that of the transverse diameter.
  - Barrel chest (increased AP diameter) can be seen in TTN, MAS: hyperinflation of the lungs.

Physical Assessment—Auscultation

- Breathsounds
  - Should be clear and symmetrical bilaterally, front and back
  - Coarse—not uncommon as fluid is cleared shortly after birth
  - Non-symmetrical: concerning for pneumothorax or atelectasis
  - Diminished: often due to immaturity/inadequate effort or respiratory disease

- Transillumination
  - Helpful to diagnose pneumothorax
  - In a darkened room air pockets will present with hyperlucency or a lantern-like glow.

Disorders causing respiratory distress

- Disease
  - Transient tachypnea of the newborn
  - Respiratory distress syndrome
  - Meconium aspiration syndrome
  - Persistent pulmonary hypertension of the newborn
  - Pneumonia

- Structural
  - Congenital diaphragmatic hernia
  - Tracheal-esophageal fistula
  - Congenital cystic adenomatoid malformation
  - Obstructed atresia
  - Pierre Robin Syndrome
  - Cleft lip and palate

- Mechanical
  - Pneumothorax
  - Pulmonary hypoplasia

Standard Respiratory Distress Work Up

- Review history
- Chest xray
- Blood gas
- CBC with differential
- Blood culture
- CRP (?)
**Transient Tachypnea of the Newborn**

- **Definition**
  - Tachypnea (RR 70-120) in near term or term infants secondary to delayed clearance of fetal lung fluid
  - Occurs in 1-2% of all newborns

- **At risk infants**
  - Caesarian births without labor
  - Breech delivery
  - Second twin
  - Precipitous delivery
  - Maternal sedation
  - Delayed clamping of the cord
  - Male gender
  - Macromomia

**TTNB—Pathophysiology**

- Reduced lung compliance due to delayed absorption of lung fluid at the time of birth and/or the distention of the interstitial spaces of the lung with fluid leading to alveolar air trapping and decreased lung compliance.

- The retained fluid accumulates in the peribronchial lymphatics and bronchovascular spaces interfering with the forces that promote bronchiolar patency and results in bronchiolar collapse with air trapping and hyperinflation.

**TTNB—PE findings**

- Tachypnea, often rapid and shallow breathing
- Immediate onset
- May see some grunting, flaring and retracting
- Can be difficult to distinguish clinically from other respiratory pathology, but symptoms are generally milder
- Generally little to no oxygen requirement
- Crackles may be heard as air passes over the retained lung fluid but breathsounds are often clear.

**TTNB—X-ray findings**

- Hyperexpansion of the lungs—hallmark finding
- Prominent perihilar streaking (engorgement of the peribronchial lymphatics)
- Fluid in the fissure and possibly mild pleural effusions
- Depression or flattening of the diaphragm

**Normal CXR** (www.indyrad.iupui.edu)

- Thymus: "mass" looking in superior mediastinum
- Heart: apex to the left, ~60% of diameter at widest point
- Pulmonary venous return to mid-lung, taper gradually
- Lungs: uniform aeration, appear dark
- Bony structures: see 10-12 ribs easily, vertebral bodies, scapulae, clavicles are easily seen
- Diaphragms: right and left are equal, generally expanded 6-9 ribs

**Rotated films**

- Baby is not flat on the table when the film is taken
- Can not adequately evaluate the cardiac silhouette—position or size
TTNB

- Lab findings
  - CBC with diff should be normal
  - ABG may show mild hypoxia. Usually hypocarbia is also seen, if hypercarbia it should be mild (Pco2 <55mm Hg).

TTNB—Supportive Care

- Cardiorespiratory and oximetry monitoring
- Oxygen as needed to maintain saturations >95%
  - Consider NCPAP or vapotherm (high flow nasal cannula) if oxygen needs are >60% or if infant is tiring
- NPO if RR >80, gavage support if RR 70-80, IV fluid administration/antibiotics
- Comfort measures: nestling, pacifier, swaddle if stable/mild distress
- Avoid excessive handling, minimize stress and limit tasks to those necessary

TTNB—Outcomes

- Self limiting over hours to 2-5 days
- No risk of further pulmonary dysfunction

Respiratory Distress Syndrome

- Also called Hyaline Membrane Disease. Causes progressive respiratory distress in primarily preterm infants secondary to inadequate gas exchange resulting from inadequate pulmonary surfactant.
- Risk factors:
  - Prematurity
  - Male sex
  - White race
  - Caesarean section delivery
  - Gestational diabetes
  - Second born twin
  - Family history of HMD
  - No antenatal corticosteroids prior to premature birth

RDS—Pathophysiology

- Lack of surfactant: Surfactant, composed primarily of phospholipid (75%) and protein (10%), is secreted by type II pneumocytes from 24-28 wk GA. Often inadequate until 35-37 wk GA. Decreased surf results in the small air spaces collapsing and progressive atelectasis. Exudative material from cellular damage also accumulates.
- Overly compliant chest wall: secondary to prematurity. Difficult to generate large enough negative pressures to expand.
- Decreased intrathoracic pressure: infants <30wk GA may not be able to generate intrathoracic pressure adequate to inflate the lungs in the absence of surfactant.
- Hyperinsulinemia can impair surfactant production.
RDS—PE Findings

- Tachypnea
- Retractions
- Grunting and flaring
- Cyanosis in room air
- Persists or progresses over the first 48-96hrs of life
- X-ray findings are characteristic: uniform reticulogranular pattern to white out and peripheral air bronchograms. Often low lung volumes.

RDS—Lab Findings

- CBC is generally normal, though WBC count can be slightly elevated due to stress
- BC will remain negative
- CRPs generally not elevated
- ABG: hypercarbia, hypoxemia, acidosis (respiratory usually predominates unless there are other issues) with severity based on severity of disease.

RDS—Supportive Care

- Surfactant replacement
- Respiratory support (hood/cannula, VT, NCPAP, ventilator, HFOV)
- Antibiotics for rule out sepsis
- IVF support/TPN then gavage feedings as stable
- Monitor glucose, blood gases, electrolytes
- Constant CRM and oximetry monitoring
- Reduce stimulation, unnecessary procedures, sedation can be especially helpful for the bigger kids
- Developmental care: nest/pacifier, quiet, involve parents with careful education, coaching and encouragement

RDS—Outcomes

- Highly dependent on birth weight and gestational age at birth—inversely proportional: younger and lower BW = higher incidence of mortality and morbidity.
- <26wk: 100%
- 32wk: 57%
- 36wk: 3.7%
- Survival and outcomes have improved greatly since surfactant.

Meconium Aspiration Syndrome

- Lung injury resulting from aspirated meconium in utero and/or before or during delivery/first breath.
- Incidence of MSAF varies from 10-15% of term deliveries, 1.6% for infants between 34-36wk GA to as many as 30% of infants at 42+ wk GA.
- Significant aspiration of meconium is only a small portion of the total infants with MSAF: 3-10%.
- Evidence of pulmonary vascular remodeling in severe cases
- Risk factors:
  - Maternal pre-eclampsia/hypertension
  - Post term pregnancy
  - Maternal diabetes mellitus
  - Abnormal fetal heart rate/poor BPP
  - Oligohydramnios
  - IUGR
Intrauterine stress may cause the release of meconium into the amniotic fluid. It is aspirated during deep fetal breathing/gasping or at the time of delivery and lung fluid reabsorption.

May occur well prior to delivery and result in chronic changes.

As it moves distally the particles result in ball-valve trapping, hyperinflation and atelectasis.

Chemical pneumonitis from the bile salts in the meconium–bronchiolar edema and narrowing of the small airways.

Surfactant inactivation by the meconium

Increased PVR results from alveolar hypoxia, acidosis and hyperinflation of the lungs–concomitant PPHN

Progressive respiratory distress and hypoxia–may seem normal immediately after birth and develop worsening symptoms in the hours after birth.

Breath sounds with rales from narrowed air passages and air moving through fluid.

As pulmonary pressures rise above systemic blood will bypass the lungs via the PDA and acidosis (resp and metabolic) will worsen. Increased pulse pressures

Barrel chest (increased AP diameter)

Very sensitive to environmental stimuli

Progression from agitation (air hunger) to an obtunded state.

**MAS–Pathophysiology**

- Intrauterine stress may cause the release of meconium into the amniotic fluid. It is aspirated during deep fetal breathing/gasping or at the time of delivery and lung fluid reabsorption.
- May occur well prior to delivery and result in chronic changes.
- As it moves distally the particles result in ball-valve trapping, hyperinflation and atelectasis.
- Chemical pneumonitis from the bile salts in the meconium–bronchiolar edema and narrowing of the small airways.
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**Ball Valve Trapping**

**MAS–PE findings**

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- Breath sounds with rales from narrowed air passages and air moving through fluid.
- As pulmonary pressures rise above systemic blood will bypass the lungs via the PDA and acidosis (resp and metabolic) will worsen. Increased pulse pressures
- Barrel chest (increased AP diameter)
- Very sensitive to environmental stimuli
- Progression from agitation (air hunger) to an obtunded state.

**MAS–X-ray/lab findings**

- CBC, blood culture, ABG
- ABG will show worsening hypoxemia, acidosis
- Chest X-ray
  - Hyperinflation of the lungs, flattened diaphragms, and coarse, irregular patchy infiltrates
  - High risk of air leaks–monitor for pneumothorax and pneumomediastinum
- Echocardiogram
  - Evaluate for evidence of PPHN and ductal shunting
MAS—Treatment/Supportive care

- Antibiotic coverage
  - Broad-spectrum, can be hard to differentiate pneumonia from MAS on x-ray

- Supplemental oxygen/respiratory support
  - Hood, cannula, rebreather, NCPAP, conventional vent, HFOV if not responding to conventional
  - Goal: normal pH and normal to slightly elevated alveolar oxygen levels

- Surfactant—best with HFOV

- Inhaled nitric oxide if PPHN is present

- Extracorporeal membrane oxygenation

- Cardiovascular management
  - May require dopamine to maintain systemic blood pressure, especially with rising pulmonary pressures
  - Consider dobutamine to improve cardiac function if concerns on echo

MAS—Treatment/Supportive care

- Monitoring—CRM, oximetry, BP, glucose, ABGs

- Reduce environmental stimuli

- Provide sedation as necessary

- Careful observation for and reporting of clinical changes
  - Pneumothorax development, worsening PPHN

- Developmental care
  - Nesting, positioning, comfort measures

- Parental support
  - Rarely expected

ECMO
Persistent Pulmonary Hypertension of the Newborn

- Definition: Marked pulmonary hypertension resulting from elevated pulmonary vascular resistance and altered pulmonary vasoreactivity leading to right-to-left extrapulmonary shunting of blood across the foramen ovale and the PDA. (Gomella, p364)
- Basically: pulmonary vascular resistance is significantly greater than peripheral vascular resistance

Risk factors
- Postterm delivery
- Placental insufficiency
- Meconium stained amniotic fluid
- Postnatal hypoxia
- Asphyxia
- Pneumonia/sepsis
- Diaphragmatic hernia
- Hypoglycemia
- Hypothermia

Three causes
- Underdevelopment of the lung and vascular bed (hypoplastic lungs)
- Inappropriate development of the pulmonary vascular bed in utero
- Failure of the pulmonary vascular bed to transition appropriately around the time of birth (caused by various conditions of perinatal stress)

Pathophysiology—PPHN
- Pulmonary arteries remain actively vasoconstricted until the latter part of gestation
- Hypoxia is a potent vasoconstrictor, especially below 40-45 torr. pO2 in utero is 20-25 torr so pulmonary vasoconstriction is facilitated.
- Lungs of infants with PPHN contain undilated precapillary arteries and the pulmonary arterial medial thickness is increased.
- Also extension of muscle in the small and peripheral arteries that are normally non-muscular.
- Pulmonary vasculature is highly sensitive to changes in PaO2, pH, ever during stress: including hypothermia, hypoxemia and hypoglycemia.

Pathophysiology of PPHN, cont
- With PVR > SVR the heart’s (esp. R side) workload is greatly increased.
- As PVR increases the heart is unable to force blood into the pulmonary bed and it is shunted through the PFO and PDA.
- Infant becomes increasingly hypoxemic and acidemic.
- Myocardial performance is compromised resulting in right heart dilation, tricuspid insufficiency and CHF.

Diagnosis
- Essentially a diagnosis of exclusion
- Physical findings
  - Respiratory distress with cyanosis
  - Poor O2 saturations, possible pre-post ductal difference (possibly not if shunting at the FO)
  - Very stimulation/noise sensitive
  - Hypertension
  - S/S heart failure are late findings
- Chest x-ray
  - May demonstrate cardiomegaly
  - Decreased pulmonary vasculature (or findings consistent with associated pulmonary disease, if present)
Diagnosis, cont.

- Lab work up should include CBC, blood culture and serial blood gases—hypoxemia in the presence of adequate ventilation
- Echocardiogram: necessary to rule out congenital heart disease
  - Measures pulmonary artery pressure
  - Provides details about shunting at the atrial and ductal levels
  - Assesses ventricular output and contractility

Treatments/Goals—PPHN

- Decrease pulmonary vasoconstriction and improve alveolar oxygenation
- Maintain systemic BP (>50mmHg in term infants), perfusion and cardiac function
- Maintain PaO2 >80-90mmHg
- Pressor support—dopamine, dobutamine, epinephrine if cardiac function concerns
- Sedate and reduce stimulation
- Trial hyperventilation/alkalotic state (pH~7.5 or greater)—can help decrease PVR
- Mechanical ventilation—trial conventional, consider HFOV quickly

Treatment—PPHN, cont.

- INO—inhaled nitric oxide
  - Originally considered an atmospheric pollutant
  - Studied and approved for treatment of PPHN in term infants in the 1990’s
  - Potent pulmonary vasodilator
  - Can also improve oxygenation by redirecting blood from poorly aerated/diseased lung areas to better aerated distal spaces
  - Start with 20ppm, wean slowly as oxygenation improves
  - Consider referral for ECMO

Pneumonia

- Respiratory infection, presents with increased work of breathing
- At risk infants
  - Maternal history: chorioamnionitis, premature/prolonged ROM, premature labor, maternal fever
- Work up
  - CBC, BC, CRPs
  - CBC symptomatic of infection, BC definitive but may be unreliable if maternal treatment
  - Chest x-ray: patchy infiltrates
Pneumonia: supportive care

- Antibiotics
- CRM, oximetry and blood gas monitoring
- Ventilatory support
- Nutritional support
- Comfort care and family support
  - Sedation, decreased stimulation
  - Education

Mechanical disorders

- Airleaks
- Pulmonary hypoplasia

Pulmonary Airleaks

- Pneumothorax: collection of air in the pleural space resulting in collapse of the lung on the affected side.
- Pneumomediastinum: air in the mediastinum from ruptured alveolar, has traversed fascial planes. No treatment is indicated, will resolve spontaneously.
- Pneumopericardium: air in the pericardial sac, usually secondary to passage of air along the vascular sheaths. Rare, generally occurring in vent dependent CLD kids.
- Pneumoperitoneum: air in the peritoneal cavity, generally caused by intestinal perforation, but may be caused by air that has ruptured from the mediastinum into the peritoneum.

Pneumothorax—pathophysiology

- Pleura: lungs are surrounded by two layers, visceral and parietal pleura. Surface tension of fluid between them keeps them together. Pressure is negative in relation to atmospheric pressure.
- Alveolar Rupture: Damaged alveoli rupture and air rushes into the pleural space until it reaches atmospheric pressure. Air is trapped outside the lung.
- Tension Pneumo: Pressure from free air in the pleural space displaces mediastinal structures and compromises cardio-pulmonary function. Opening to pleural space acts as a one way valve. Hypoxia and hypotension due to decreased cardiac output are progressive.

Incidence

- Healthy term: 0.5-2% Oligos
- TTN: 5-10%
- RDS: up to 20%
- MAS: up to 50%

Critical Care Nurse Snapshot
### Airleak Risk Factors

- Resuscitation requiring PPV
- MAS
- TTNB
- RDS
- Infection
- Intubation
- Mechanical ventilation
- High inflating pressures at birth
- Genetic abnormalities

### Pneumo– signs and symptoms

- Acute desaturation with cyanosis and increased WOB with poor response to increased oxygen
- Decreased breath sounds on affected side
- Poor perfusion and hypotension
- PMI shift
- Muffled heart tones
- Poor peripheral pulses
- Increased AP diameter
- ABG: hypoxia, acidosis, hypercarbia
- Agitation/irritability
- Asynchrony of the chest

### Pneumo– diagnosis

- PA: inspection of chest and auscultation
- Transillumination
  - Quick and cost effective
  - Affected area with larger area of illumination than normal area
  - Can be difficult to see if skin is very darkly pigmented or if room is too light
- Chest X-ray
  - Diagnostic
  - 2-view often helpful: AP and lateral decubitus or cross table

### Transillumination

- Tension Pneumo

### 2-view films
Treatment Options

- Nitrogen washout
  - Suitable for small pneumos
  - 100% FiO2 results in differing gas tensions between blood and accumulated air and rapid absorption of the oxygen in the pleural space.

- Needle thoracentesis
  - Needle is inserted at 2nd, 3rd ICS at midclavicular line
  - Air gently aspirated
  - Use three-way stopcock

- Chest tube
  - Drain is inserted into chest and connected to collection chamber below chest level
  - Water seal or suction

Nitrogen washout is suitable for small pneumos as it results in differing gas tensions between blood and accumulated air, leading to rapid absorption of oxygen in the pleural space. Needle thoracentesis involves inserting a needle at the 2nd or 3rd intercostal space at the midclavicular line, gently aspirating the air, and using a three-way stopcock. Chest tube drainage involves inserting a drain into the chest and connecting it to a collection chamber below the chest level, either using a water seal or suction.
Supportive Care
- Careful monitoring
- Reduce stimulation
- Sedation as necessary
- Close attention to change in clinical condition
- Attention to chest tube sight, maintaining closed system
- Pain control
- Developmental support
- Attention to pressures used during bagging

Pulmonary hypoplasia
- Lack of pulmonary tissue
- Difficult conclusive diagnosis: must rely on maternal history
  - Severe oligohydramnios, prolonged early ROM
  - Conclusive diagnosis post-mortem
- CXR findings
  - Bell shaped chest, poor lung expansion
- Treatment is supportive
  - Mechanical ventilation
  - Minimize MAP to reduce risk of pneumo

Structural causes of respiratory distress
- Congenital diaphragmatic hernia
  - Mostly left sided, concave abdominal
  - Immediate goal: GI decompression
- Tracheo-esophageal fistula
  - Frothy secretions
  - Pouch or gastric suction to prevent aspiration
- Pierre Robin sequence: hypoplasia of the lower jaw
  - Often prone positioning is adequate, tracheotomy if severe

Congenital diaphragmatic hernia

Choanal Atresia
- Definition: congenital blockage of the posterior nares by a persistent bony septum (90%) or membrane (10%).
- Epidemiology
  - 2-4/10,000 births
  - Familial tendency
  - Female to male ratio of 2:1
  - 2/3 are unilateral
  - 50% association with other often significant anomalies
  - 50-70% have congenital heart disease

Choanal Atresia – CT
Choanal Atresia—Clinical Presentation

- If unilateral, may escape diagnosis for years
- Bilateral—respiratory distress and cyanosis which is relieved by crying. Infants are obligate nose breathers unless crying.
- Initial diagnosis is based on the inability to pass a 3–5 fr catheter via either side of the nose
- Diagnosis is confirmed and location and thickness of the obstruction are defined by CT
- Intubation or oral airway is used for initial management
- Surgery is corrective

Apnea

- Definition: absence of breathing for >20 seconds or shorter pause in breathing if it was associated with oxygen desaturation or bradycardia.
- Apnea of prematurity is common in infants <36wks, especially <34wks and will resolve as their brains mature.
- Apnea in a term infant is NOT normal and requires a full work up.
- A few possible causes: seizures, thermal instability, GI reflux, metabolic derangement, impaired oxygenation, sepsis, drugs
- Clinical findings other than the apnea will depend on the underlying cause as will supportive care and management.

Case Study #1

- 28 wk gestation, uncomplicated pregnancy.
  Infants delivered emergently by C/S. Mother did not receive betamethasone.
  - What is the respiratory complication for which this infant is at high risk?
  - To get this infant off to the best start, what are a couple respiratory factors/strategies you will want to keep in mind in the delivery room?

Case Study #1, cont.

- As the nurse caring for this infant, what care do you anticipate providing from a respiratory perspective?
- What nursing interventions will help optimize this infant’s outcome?
- What are some of the things you will anticipate educating the parents about as this infant recovers and progresses toward discharge?

Case Study #2

- You are the nursery admit nurse. L&D calls you right after change of shift to assess a baby in respiratory distress. You arrive in the mother’s room and find the baby under the warming lights grunting, flaring and retracting in 100% blow-by oxygen.
  - What do you need to find out from the L&D staff?
  - What respiratory ailments come to mind as you look at this infant?

Case Study #2, cont.

- You find out this is a term infant who had meconium stained amniotic fluid. You contact the infant’s provider and transport the infant to the nursery.
  - What are a couple important things to ensure as you are transporting and admitting this infant?
  - Depending on the progression of this infant’s respiratory disease, what are some of the respiratory interventions you are anticipating?
Case Study #2, cont.

- What are some of the potential complications for which you will be monitoring this MAS baby?
- What are some of the nursing interventions you will use to facilitate the best outcome for this infant?

Selected References