Neonatal Respiratory System

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Objectives

Upon completion of this course, the participant will be able to:

- Describe the transition from intrauterine to extrauterine life.
- Identify the equipment that should be readily available for a neonatal resuscitation.
- List the medications recommended in the AAP/AHA Neonatal Resuscitation Program.
- Describe assessment and diagnostic strategies of common neonatal respiratory disorders.
- Define nursing interventions for managing the infant with respiratory distress.

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Contents

Embryology and System Development

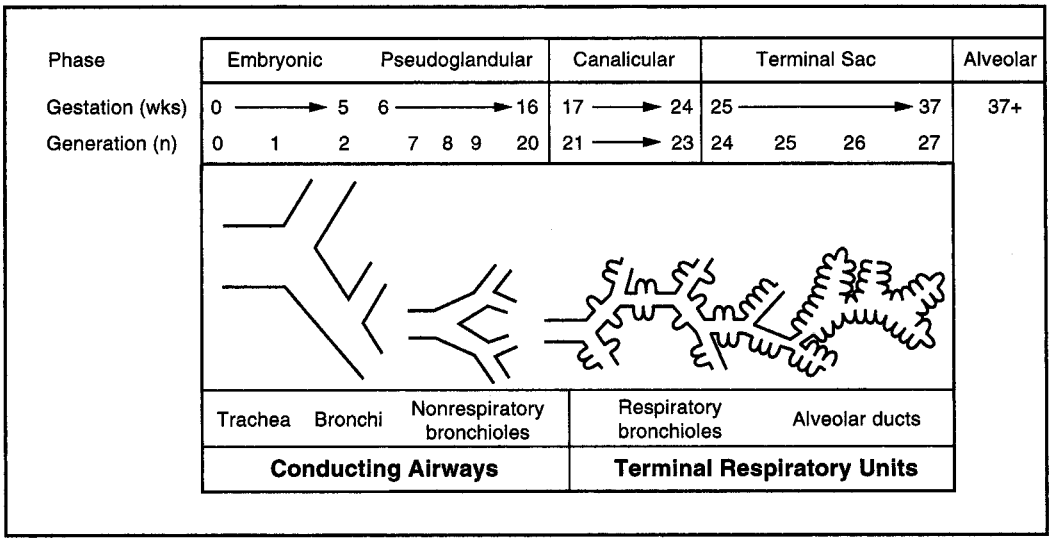
There are five stages in the embryonic development of normal lung growth: embryonic, pseudoglandular, canalicular, terminal sac, and alveolar. As shown in Figure 1, the embryonic stage occurs from conception to week 5, with the major event being the formation of the proximal airways. The lung bud appears and begins to divide, the pulmonary vein develops and extends to join the lung bud, and the trachea develops. In stage 2, the pseudoglandular stage, formation of the conducting airways occurs (weeks 6–16). Cartilage appears and the main bronchi form. Formation of new bronchi is complete and the capillary bed is formed. During the canalicular stage (weeks 17–24), the major feature is formation of acini (gas-exchanging sites). There is an appearance of cuboidal cells, the capillaries invade the terminal air sac walls, type II alveolar cells appear, and the airway changes from glandular to tubular and increases in length and diameter. The alveolar sacs are formed and there is development of gas-exchange sites in the terminal sac stage (weeks 25–37). In the alveolar, or final, stage (week 37–postnatal), expansion of the surface area occurs. This stage continues up to 8 years after birth.

The surface of the lung is lined by a layer of fluid that creates an air-liquid interface. Surface-tension forces act on air-liquid interfaces, causing a water droplet to bead up. A surface-active compound called surfactant reduces the surface tension and allows the droplet to spread out into a thin layer. In the lungs, the surface-tension forces tend to cause the alveoli to collapse. Surfactant is needed to lower the surface tension within these alveoli to prevent their collapse at the end of expiration. Surfactant is a surface-active agent composed of phospholipids (including lecithin and sphingomyelin), cholesterol, lipids, and proteins and is synthesized in type II alveolar epithelial cells. These cells begin to appear in the lung between 20 and 24 weeks of gestation.

KEY LEARNINGS

- There are five stages in the embryonic development of normal lung growth.
- The final stage continues up to 8 years after birth.
- The surface of the lung is lined by a layer of fluid that creates an air-liquid interface.
- Surfactant is needed to lower the surface tension within the alveoli to prevent their collapse.

Figure 1. The five phases of the process of tracheobronchial airway development.



n = number of branches.

Reproduced, with permission, from Leuthner SR: Anatomy and development of the lung. In Weisman LE, Hansen TN, eds: *Contemporary Diagnosis and Management of Neonatal Respiratory Diseases*, 3rd ed. © 2003, Handbooks in Health Care Co, p 2.

Transition from Intrauterine to Extrauterine Life

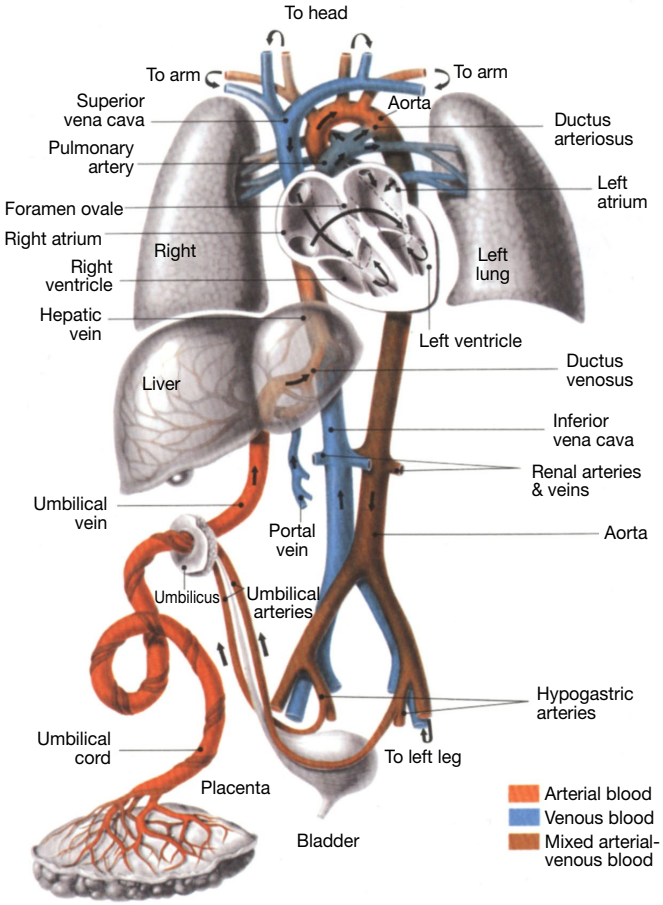
The transition from intrauterine to extrauterine environment and from fetal to postnatal life begins with the clamping of the umbilical cord and the infant's first breath.

In utero, fetal circulation (Figure 2) depends on the placenta and three fetal ducts: the ductus venosus, the foramen ovale, and the ductus arteriosus. The placenta allows for the exchange of gases, nutrients, and metabolic waste products. It is a low-resistance circuit that maintains a low fetal systemic vascular resistance, while the pulmonary fetal circuit maintains a high pulmonary vascular resistance. Subsequently, the increased pulmonary vascular resistance and low systemic vascular resistance promote right-to-left shunting through the fetal ducts. The ductus venosus allows part of the oxygenated blood carried by the umbilical vein to bypass the liver. Oxygenated blood entering the heart flows through the foramen ovale into the left atrium, then perfuses the brain and the heart via the carotid, subclavian, and coronary arteries. The ductus arteriosus directs blood from the main pulmonary artery to the descending aorta. Fetal admixture at the foramen ovale and ductus arteriosus lowers fetal arterial oxygen tension to ~ 25–35 mm Hg. The low fetal oxygen tension helps to maintain pulmonary artery vasoconstriction, allowing blood to bypass the lung and flow instead through the foramen ovale and ductus arteriosus.

Once the infant is delivered and the transition to extrauterine life begins, respiratory and cardiovascular changes occur independently but simultaneously.

In summary, fetal blood flows from the placenta via the umbilical vein, bypasses the liver via the ductus venosus, and enters the inferior vena cava. From the inferior vena cava, blood enters the right atrium, where the majority of it is shunted through the foramen ovale into the left atrium. Blood continues into the left ventricle, where it mixes with blood returning from the pulmonary veins, and is then injected into the ascending aorta. From the ascending aorta, it supplies the carotid, subclavian, and coronary arteries before mixing with blood shunted across the ductus arteriosus. The remainder of the blood entering the right atrium mixes with blood from the superior vena cava and continues into the right ventricle and pulmonary arteries. Most of this blood shunts across the ductus arteriosus into the descending aorta.

Figure 2. Fetal circulation.



Once the infant is delivered and the transition to extrauterine life begins, respiratory and cardiovascular changes occur independently but simultaneously. Fetal lung fluid is replaced by air, so the liquid-liquid interface of alveoli becomes an air-liquid interface and surface tension forces begin. Surfactant decreases the surface tension with the first breath and arterial oxygen tension rises, resulting in reversal of hypoxemia-induced pulmonary vasoconstriction. The pulmonary vascular resistance begins to decline as a result of increasing oxygen saturations and decreasing carbon dioxide levels, resulting in an increase in

pulmonary blood flow. Decreasing prostaglandin levels also will facilitate the reversal of the pulmonary vasoconstriction. Removal of the placental circuit by the clamping of the umbilical cord results in increasing systemic vascular resistance. Simultaneous cardiovascular changes include the closing of fetal shunts. The ductus venosus functionally closes as the umbilical cord is clamped. Functional closure of the foramen ovale occurs at birth from the changing atrial pressures and increasing systemic vascular resistance. The left atrial pressure is now greater than the right atrial pressure. With increasing arterial oxygen tension and decreasing levels of prostaglandin E, the ductus arteriosus closes functionally at 15–24 hours of age but does not close anatomically for 3–4 weeks (Tables 1 and 2).

Table 1. Summary of Main Transitional Events from Fetal to Neonatal Circulation

Loss of fetal lung fluid
Secretion of surfactant
Establishment of functional residual capacity
Fall in pulmonary vascular resistance
Rise in systemic vascular resistance
Closing of fetal shunts
Increasing pulmonary blood flow

KEY LEARNINGS

- In utero, fetal circulation depends on the placenta and fetal ducts.
- Low fetal oxygen tension allows blood to bypass the lung.
- At delivery, respiratory and cardiovascular changes occur independently but simultaneously.

Table 2. Transition from Fetal to Neonatal Circulation

	First Period of Reactivity (early reactivity)	Period of Relative Inactivity (deep sleep)	Second Period of Reactivity (secondary reactivity)
Time Course	Birth to 30–45 minutes	45 minutes to 2–4 hours	3–4 hours thereafter
CNS	Alert, eyes open; vigorous, active crying; increased tone and highly responsive to stimuli	Somnolent, eyes closed; difficult to arouse or interest; decreased tone and general responsiveness; can be awakened only briefly	Hungry; progresses normally through wake, feed, quiet alert, drowsy, and deep sleep in cyclic fashion
Color	Ruddy with acrocyanosis	Pale, no cyanosis	Pink, no cyanosis
Heart Rate	High (140–160 BPM) and very reactive	Low (90–120 BPM) and briefly reactive	Varies with wake/sleep cycle
Respiratory Rate	High (40–60 BPM), mild retractions, moist rales	Low (20–40 BPM), no retractions, no rales; occasional periodic breathing	Varies with wake/sleep cycle
Blood Pressure	Should rise slowly but steadily through all stages		
Bowel Sounds	Active bowel sounds; belly distended; may pass meconium and urine	Inactive bowel sounds; less distention; belly easily palpated	Active bowel sounds; air swallowing and distention with crying

Adapted from Molteni RA: *Neonatal Respiratory Distress* Clinical Education Aid. , p 3.

Resuscitation of the Infant with Respiratory Distress

Anticipation is a key component of the successful resuscitation of a distressed newborn. Maternal or fetal conditions that place a newborn at risk for respiratory depression/distress at birth must be recognized. According to the American Academy of Pediatrics (AAP) and the American Heart Association (AHA), "Every newborn has a right to a resuscitation performed at a high level of competence. The proper equipment must be immediately available at delivery, and healthcare professionals must be skilled in resuscitating a newborn and capable of working smoothly as a team" (Kattwinkel 2011).

Resuscitation Equipment

Resuscitation equipment should be available, ready to use, and functional at all times, and should include:

- Radiant warmer, towels, and blankets (prewarmed)
- Stethoscope
- Bulb syringe
- Mechanical suction and tubing
- Meconium aspirator
- Suction catheters (5F or 6F, 8F, 10F, 12F or 14F)
- Laryngeal airway mask
- Resuscitation bag with manometer or pressure-release valve (capable of delivering 90%–100% oxygen)
- Laryngoscope with extra batteries
- Straight blades: sizes 0 and 1 with extra bulbs
- Endotracheal tubes (2.5, 3.0, 3.5, 4.0 mm internal diameter)
- Stylet
- CO_2 detector or capnograph
- Alcohol sponges
- Scissors
- Tape or securing device for endotracheal tube
- 8F feeding tube; 20-mL syringe
- Face masks (newborn, premature sizes)
- Oxygen source with flowmeter and tubing; oxygen blender for delivery of oxygen from 21% to 100%)
- Compressed air source
- Pulse oximeter; oximeter probe
- Thermoregulation supplies: plastic bag or plastic wrap; chemically activated warming pad; transport incubator (prewarmed)

Table 3. Medications for Neonatal Resuscitation

Medication*	Concentration to Administer	Preparation	Dosage/Route†	Total Dose/Infant: Weight/ Total mg/Total mL	Rate/Precautions
Epinephrine	1:10,000	3-mL or 10-mL ampules	0.1–0.3 mL/kg IV or ET	**Weight** **Total mL** 1 kg 0.1–0.3 mL 2 kg 0.2–0.6 mL 3 kg 0.3–0.9 mL 4 kg 0.4–1.2 mL	Give rapidly May dilute with normal saline to 1–2 mL if giving ET Use two different-sized syringes—one size for IV dosage and one size for ETT dosage (Niermeyer and Clarke 2011)
Volume Expanders	Lactated Ringer's solution or normal saline	100–250 mL	10 mL/kg IV per umbilical vein	**Weight** **Total mL** 1 kg 10 mL 2 kg 20 mL 3 kg 30 mL 4 kg 40 mL	Give over 5–10 minutes

* Sodium bicarbonate is not a recommended therapy in neonatal resuscitation guidelines. Use only after adequate ventilation is established and there is no response to other therapies *(NeoFax 2011)*.
† IV = intravenous, ET = endotracheal
Adapted from Kattwinkel J, ed. *Neonatal Resuscitation Textbook*. © 2011 American Academy of Pediatrics and American Heart Association.

Medications recommended in the AAP/AHA Neonatal Resuscitation Program (NRP) (Kattwinkel 2011) should also be readily available, including intravenous fluids (dextrose 10%; normal saline for flush) and umbilical vessel cannulation materials (Table 3).

Resuscitation Procedure

Mastery of neonatal resuscitation skills is necessary for performing successful resuscitation. The AAP/AHA NRP is a national program that provides health care professionals with the knowledge and skills to resuscitate newborn infants by using a standardized approach (Kattwinkel 2011).

Because the clinical assessment of cyanosis is not reliable, and newborns during transition may take several minutes to increase their blood oxygen saturations, using a pulse oximeter assists in determining the need for supplemental oxygen. Place the pulse oximeter probe preductally (on the right arm), and, after the pulse oximeter is reading reliably, adjust the percentage of supplemental oxygen concentration to achieve target saturations in Table 4.

Table 4: Targeted Preductal Pulse Oximetry Ranges in the First 10 Minutes of Life in Normal, Term Newborns

Time	S_pO_2 Values
1 minute	60%–65%
2 minutes	65%–70%
3 minutes	70%–75%
4 minutes	75%–80%
5 minutes	80%–85%
10 minutes	85%–95%

Adapted from: Kattwinkel et al. 2010.

KEY LEARNINGS

- Anticipation is a key component of the successful resuscitation of a distressed newborn.
- Resuscitation equipment and medications should be available, ready to use, and functional at all times.

Differential Diagnosis

The differential diagnosis for neonatal respiratory distress is very broad (see Tables 5 and 6). The practitioner must carefully review the maternal history, observe the infant's disease course, and assess the results of the physical examination. Laboratory tests and radiologic findings are adjuncts to the differential diagnosis.

Table 5. Pulmonary Disorders

Common	Less Common
Respiratory distress syndrome	Pulmonary hypoplasia
Transient tachypnea	Upper airway obstruction
Meconium aspiration syndrome	Rib cage abnormalities
Pneumonia	Space-occupying lesions
Air-leak syndrome	Pulmonary hemorrhage

Table 6. Extrapulmonary Disorders

Vascular	Metabolic	Neuromuscular
Persistent pulmonary hypertension Congenital heart disease Hypovolemia, anemia Polycythemia	Acidosis Hypoglycemia Hypothermia	Cerebral edema or hemorrhage Drugs Muscle disorders Spinal cord problems Phrenic nerve damage

KEY LEARNINGS

- The differential diagnosis for neonatal respiratory distress is very broad, and may include pulmonary, vascular, metabolic, or neuromuscular disorders.

History and Respiratory System Assessment

Table 7. History	
Mother	Prenatal history and care Family illnesses Age Pregnancy-related complications Medications Substance abuse Gravida, para, abortions, living children Blood type, antibody screening
Intrapartum	Intrapartal complications Time of rupture of membranes (spontaneous or artificial) Description of amniotic fluid (clear, foul-smelling, meconium-stained) Onset and duration of labor (spontaneous or induced) Medications Evidence of fetal distress Method of delivery Vaginal (especially forceps, vacuum extraction) Cesarean
Infant	Apgar scores Resuscitative interventions Gestational-age examination General physical examination

Figure 3. The normal lung.

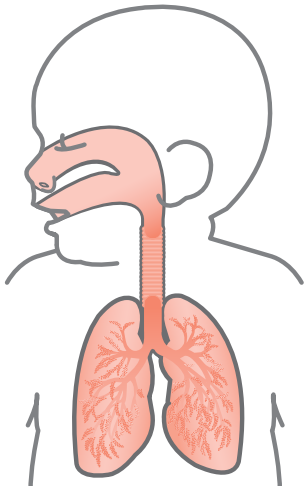


Table 8. Respiratory System Assessment	
Color	
Pink	Reddish-pink hue of skin, nailbeds, and mucous membranes
Cyanosis	Blue discoloration of skin, nailbeds, and mucous membranes
Acrocyanosis	Peripheral cyanosis of hands and feet
Plethora	Ruddy color
Pallor	Pale, white skin
Type of Breathing	
Apnea	Cessation of breathing for >20 seconds, usually with color changes and bradycardia
Periodic respirations	Intermittent cessation of respiration; usually pauses between breaths <15 seconds
Dyspnea	Labored or difficult breathing
Bradypnea	Abnormally slow respiratory pattern; 20–30 BPM of slower, deeper respirations
Tachypnea	Respiratory rate >60 BPM
Respiratory Effort	
Chest movement	Depth of respiration, symmetry, synchrony
Paradoxical respiratory effort	Inward pull of lower thorax and bulging of abdomen with each breath
Retractions	Inward pull of chest wall on inspiration
Other Respiratory Findings	
Expiratory grunt	Audible, forced expiration through a partially closed glottis Delays expiration and increases gas exchange by increasing end-expiratory pressure
Nasal flaring	Increased size of nares with respiration to decrease airway resistance
Stridor	High-pitched crowing sound caused by narrowing of glottis or trachea

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Table 8. Respiratory System Assessment	
Wheeze	High-pitched, continuous lung sounds similar to dry whistling sound produced by air passing through a narrowed lumen
Chest Shape/Symmetry	
Barrel-shaped chest	Suggests increased chest volume, eg, transient tachypnea of the newborn, meconium aspiration syndrome, persistent pulmonary hypertension
Bell-shaped chest	Suggests decreased chest volume, eg, respiratory distress syndrome, pulmonary hypoplasia
Chest wall asymmetry	Results from volume differences between two sides of thoracic cavity, eg, atelectasis, pneumothorax, unilateral pulmonary emphysema, cystic lung disease
Auscultation of Breath Sounds	
Assess air movement and quality of breath sounds: Normal breath sounds Adventitious breath sounds Bronchovesicular (expiration equals inspiration) Rales Rhonchi Wheeze Pleural friction rub	

KEY LEARNINGS

- Assessment should include a history of the mother, infant, and intrapartum issues.
- A respiratory system assessment should include the infant's color, type of breathing, respiratory effort, chest shape/symmetry, and auscultation of breath sounds.

Common Neonatal Respiratory Disorders

Respiratory Distress Syndrome

Respiratory distress syndrome (RDS) is caused by a primary absence or deficiency of surfactant. Endogenous surfactant prevents increased surface tension, which can lead to alveolar collapse.

Incidence

(Moïse and Hansen 2003; Cifuents et al. 2002)

- 40,000 infants per year in the US
- 14% of low-birth-weight infants
- 60%–80% of infants <28 weeks' gestational age
- Inversely proportional to gestational age

Infants at Risk/Predisposing Factors

- Premature infants
- Male infants
- Infants of diabetic mothers—surfactant production can be inhibited due to the infant's hyperinsulinemic state
- Perinatal asphyxia—surfactant production can be decreased due to transient fetal distress

Pathophysiology

The absence or deficiency of surfactant results in increased alveolar surface tension, leading to alveolar collapse and decreased lung compliance (stiff lungs). With decreased lung compliance, greater and greater negative pressure must be generated to inflate the lung with each succeeding breath. Widespread alveolar collapse, or atelectasis, results in mismatches of ventilation and perfusion (V/Q ratio) and hypoventilation. Collapsed areas of the lung may continue to receive capillary blood flow, but gas exchange does not occur. Intrapulmonary shunting causes further hypoxemia. Hypercarbia also develops, which leads to respiratory acidosis. Hypoxia at the cellular level results in anaerobic metabolism and, subsequently, metabolic acidosis. With the hypoxemia and resulting acidosis, there is increased pulmonary vascular resistance and vasoconstriction, leading to pulmonary hypoperfusion and additional hypoxemia.

Clinical Presentation

The usual clinical presentation is seen within 6 hours after birth and includes the following:

Nasal flaring	An attempt to decrease airway resistance and take in more oxygen
Grunting	An attempt to maintain functional residual capacity
Retractions	A reflection of noncompliant or stiff lungs and compliant chest wall
Tachypnea	An attempt to maintain minute ventilation and prevent lung collapse
Hypoventilation	A result of muscle fatigue
Diminished breath sounds	A reflection of decreased air entry
Edematous extremities	A result of altered vascular permeability
Cyanosis	A result of increasing hypoxemia

Arterial blood gases demonstrate hypoxemia in room air, hypercarbia, and mixed acidemia. With mild RDS, the chest radiograph shows a ground-glass, reticulogranular appearance (diffuse alveolar atelectasis surrounding open bronchi), air bronchograms (aerated bronchioles), and decreased lung volumes (diffuse atelectasis). With severe RDS, a "whiteout" pattern is seen on the chest film, with little aeration and the heart border obscured or fuzzy.

Figure 4.
Lungs with mild RDS.

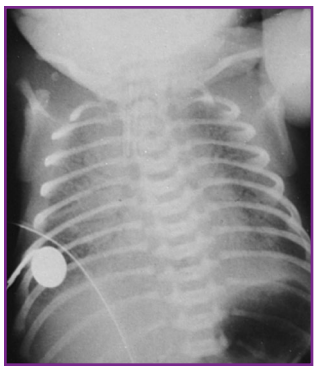
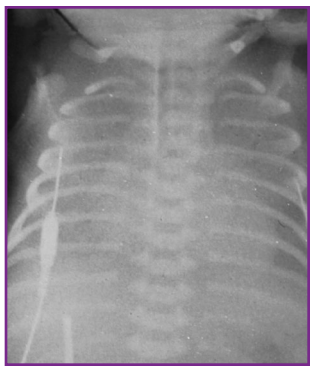


Figure 5.
Lungs with severe RDS.



Used with permission from Gardner SL, et al. *Merenstein and Gardner's Handbook of Neonatal Intensive Care*, 7th ed. © 2011 Mosby.

The clinical course of RDS is variable but self-limiting. There is progressive worsening over the first 2–3 days, as evidenced by increasing oxygen requirements and poor lung performance. Postnatal surfactant production begins at ~ 48–72 hours of age and results in improved lung compliance and decreasing respiratory distress. This recovery phase is usually preceded by a period of spontaneous diuresis.

Management

Management begins with preventive measures. Administration of antenatal steroids results in accelerated maturity of the fetal lungs. The incidence and severity of RDS are decreased in infants whose mothers received corticosteroids 24–48 hours before delivery. Corticosteroids are most effective when infants are less than 34 weeks gestational age and the drug is administered for at least 24 hours but no longer than 7 days before delivery. There appears to be an additive effect in the improvement of lung function with the combined use of antenatal steroids and postnatal surfactant.

Management of RDS begins with preventive measures.

The goals of treating RDS are to prevent alveolar collapse, optimize tissue oxygenation and carbon dioxide elimination, minimize oxygen consumption, and provide supportive care. Administration of oxygen, continuous positive airway pressure, and positive-pressure ventilation may be needed to provide adequate tissue oxygenation, relieve hypoxic vasoconstriction, and reduce right-to-left shunting. Arterial blood gases, pulse oximetry, and transcutaneous oxygen monitors provide information needed to maintain the arterial oxygen tension within an acceptable range. Positive end expiratory pressure, provided by either nasal prongs or tracheal intubation, can be used to prevent atelectasis by maintaining alveolar distention throughout the respiratory cycle. Sedatives and analgesics may be given if the infant's respiratory efforts interfere with effective positive-pressure ventilation (Table 9). Supportive care includes maintaining a neutral thermal environment, hydration, and circulatory support; antibiotics; and standard neonatal care.

Table 9. Sedatives, Analgesics, and Muscle Relaxants for Neonates (*NeoFax 2011*)

	Dose	Side Effects
Sedatives		
Lorazepam	0.05–0.1 mg/kg/dose IV slow push Usual dosing interval: Q 4–8 hr	Respiratory depression Hypotension
Midazolam*	0.05–0.15 mg/kg/dose over at least 5 minutes IV, IM Q 2–4 hr	Respiratory depression and respiratory arrest Hypotension Seizure, seizure-like activity following rapid bolus administration **Due to side effects and insufficient evidence, IV midazolam is not recommended as a sedative for neonates**
Chloral hydrate	25–75 mg/kg/dose PO or PR Usual dosing interval: Q 6 hr Dilute oral preparation or give after a feeding	CNS, respiratory, myocardial depression Ileus and bladder atony Indirect hyperbilirubinemia Cardiac arrhythmias Do not use in patients with significant liver or kidney disease
Analgesics		
Morphine	0.05–0.2 mg/kg/dose over at least 5 minutes IV, IM, SQ Usual dosing interval: Q 4 hr Continuous IV infusion: Loading dose first—100–150 mcg/kg over 1 hour followed by 10–20 mcg/kg/hr	Respiratory depression Hypotension Bradycardia Urine retention Decreased gut motility Tolerance and withdrawal with prolonged administration (weaning regimen needed) Reversed with naloxone
Fentanyl	0.5–4 mcg/kg/dose IV Usual dosing interval: Q 2–4 hr Continuous IV infusion: 1–5 mcg/kg/hr	Fewer respiratory and cardiovascular effects than with morphine With large, rapid boluses: Muscle rigidity Hypotension Seizure activity Bradycardia Tolerance and significant withdrawal with continuous infusion ≥5 days (weaning regimen needed) Reversed with naloxone
Muscle Relaxants		
Pancuronium bromide*	0.1 mg/kg/dose IV (0.04–0.15 mg/kg/dose), as needed for paralysis Usual dosing interval: 1–2 hours Continuous IV infusion: 0.05–0.2 mg/kg/hr	Tachycardia Hypotension/Hypertension Hypoxemia Peripheral edema Increased salivation Reversed with atropine or glycopyrrolate followed by neostigmine
Vecuronium bromide*	0.1 mg/kg/dose IV (0.03–0.15 mg/kg/hr), as needed for paralysis Usual dosing interval: 1–2 hours	Minimal cardiovascular effects when used alone Bradycardia, hypotension when used concurrently with narcotics Hypoxemia

IV = intravenous, IM = intramuscular, PO = oral, PR = rectal, SQ = subcutaneous.
* Should be administered by adequately trained individuals familiar with its actions, characteristics, and hazards.

Other treatments for RDS include surfactant replacement therapy, high-frequency ventilation, continuous positive airway pressure (CPAP), and patient-triggered ventilation.

Surfactant replacement therapy has become a standard treatment. It improves oxygenation and stabilizes alveoli with a resultant reduction in the severity of RDS. Commercial preparations used in surfactant replacement therapy are usually given as a liquid bolus into the endotracheal tube, with the dose divided into aliquots and administered with the infant in different positions.

High-frequency ventilation (HFV), including high-frequency oscillation and high-frequency jet ventilation, appears to produce adequate gas exchange at lower peak airway pressures while potentially reducing barotrauma and the development of chronic lung disease. It uses small tidal volumes at near or less than anatomic dead space at rapid rates.

Bubble CPAP is achieved by submerging tubing underwater at a predetermined depth, which allows the air flow to bubble out. Bubble CPAP has chest wall vibrations similar to HFV and, compared with conventional ventilation, reduced minute volume and respiratory rate.

Synchronized intermittent mandatory ventilation and assist/control mode ventilation are referred to as patient-triggered ventilation. Synchronized intermittent mandatory ventilation uses airway flow, airway pressure, changes in chest wall impedance, or abdominal movements to detect the onset of inspiratory efforts. Spontaneous breaths trigger the ventilator to maintain the rate. During episodes of apnea, controlled breaths occur at the preset rate. In the assist/control mode, a spontaneous breath triggers a mechanical breath. This mode also delivers controlled breaths at the preset rate during apnea. Patient-triggered ventilation has been shown to improve gas exchange and reduce asynchrony between infant-generated and ventilator-generated breaths.

Prognosis

(Moïse and Hansen 2003; Bhutani 1996)

Most infants with RDS recover without further problems, usually within 3–5 days. With severe RDS, the requirement for assisted ventilation, the development of complications such as air leaks, patent ductus arteriosus, or the beginnings of chronic lung disease/bronchopulmonary dysplasia (CLD/BPD) may delay recovery for days, weeks, or even months. Mortality is inversely proportional to gestational age. The incidence of chronic lung disease is <10% in infants with a birth weight >1000 g to ~ 50% in infants with a birth weight <1000 g.

Transient Tachypnea of the Newborn

The most commonly cited cause of transient tachypnea of the newborn (TTN) is delayed absorption of fetal lung fluid.

Infants at Risk/Predisposing Factors

- Term and late-preterm (“near-term”) infants
- Precipitous delivery
- Cesarean delivery, especially in the absence of labor
- Male infants
- Prenatal exposure to methamphetamine

Pathophysiology

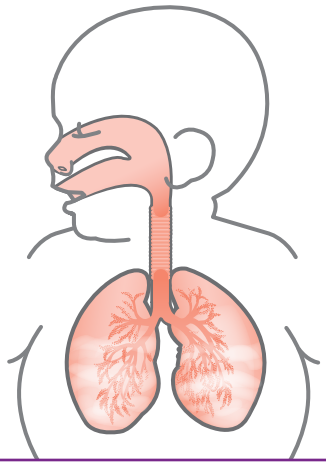
In utero, the fetal lungs are filled with fluid. During normal vaginal delivery, the fluid is usually forced out by the thoracic squeeze. The remainder of the fluid in the lungs is cleared by the pulmonary veins and lymphatic system. With a precipitous or cesarean delivery, absence of the gradual chest compression that occurs during normal vaginal birth causes fluid to be retained. Accumulation of this interstitial fluid interferes with forces that tend to keep the bronchioles open and eventually causes the bronchioles to collapse (air trapping). Air trapping and hyperinflation can increase pulmonary vascular resistance and lead to potential persistent pulmonary hypertension.

Clinical Presentation

The clinical presentation can be difficult to distinguish from that of other neonatal disorders such as bacterial pneumonia, sepsis, and RDS. The onset of symptoms is usually 0.5–6 hours after birth; respiratory rates up to 120–140 BPM are the most common symptom. Grunting, nasal flaring, and retractions may occur with varying severity.

Arterial blood gases reveal hypoxemia in room air, mild hypercarbia, and mild to moderate acidosis. Chest radiographs show hyperinflation (from air trapping) and streaky infiltrates (interstitial fluid along the bronchovascular space) from the hilum.

Figure 6. Transient tachypnea of the newborn.



Management

Treatment of TTN consists of supplemental oxygen (usually <40% fractional inspiratory oxygen [FiO_2]), pulse oximetry and/or transcutaneous monitoring, antibiotics (if infection is suspected), a neutral thermal environment, and general supportive neonatal care. Oral feedings should be delayed to prevent aspiration from high respiratory rates.

Prognosis

Although TTN is self-limiting and usually clears within 1–3 days, it is a diagnosis of exclusion made after the infant has recovered. Infants generally recover completely without any residual respiratory problems.

Meconium Aspiration Syndrome

Meconium aspiration syndrome (MAS) is the most common aspiration syndrome causing repiratory distress in newborns.

Meconium aspiration syndrome is the most common aspiration syndrome causing respiratory distress in newborns.

Incidence

(Clark and Clark 2012; Yoder et al. 2002)

- 8% – 25% of all US births >34 weeks gestation have meconium in amniotic fluid
- ~10% of those infants develop MAS
- changes in obstetrical practice appear to be decreasing the incidence

Infants at Risk/Predisposing Factors

- Term or postterm infants
- Term or postterm, small-for-gestational-age infants
- Any event causing fetal distress, such as:
  - Reduced placental or uterine blood flow
  - Maternal hypoxia and/or anemia
  - Placental or umbilical cord accidents
- African-American race
- Chorioamnionitis/infection

Pathophysiology

Meconium is normally retained in the fetal gut until postnatal life, but passage of meconium occurs in response to fetal distress (hypoxic bowel stimulation). The rectal sphincter tone or muscle may relax after vagal reflex stimulation and release meconium into the amniotic fluid. The fetus begins gasping in response to asphyxia and may inhale meconium into the airway. With the infant's first breath, meconium can be aspirated into the lungs. This aspirated thick meconium can result in:

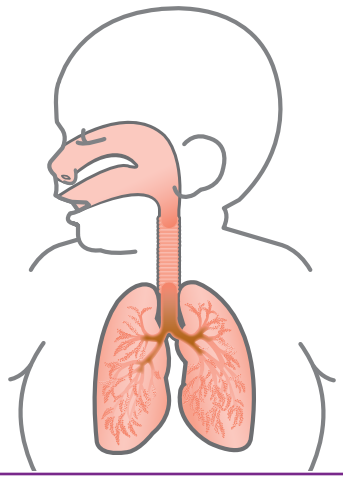
- Partial airway obstruction (a ball-valve obstruction), leading to air trapping and overdistention of the airways, with alveolar rupture and air leaks
- Complete airway obstruction, leading to small airway atelectasis
- Inflammatory response of the tracheobronchial epithelium to meconium, leading to chemical pneumonitis
- Possible surfactant displacement or inactivation of endogenous surfactant

Uneven pulmonary ventilation with hyperinflation of some areas and atelectasis of others leads to ventilation-perfusion mismatches and, subsequently, hypercarbia and hypoxemia. Hypoxemia may worsen pulmonary vasoconstriction, resulting in further hypoxemia and acidemia, and set up a vicious cycle.

Clinical Presentation

Usually infants with MAS have a history of fetal distress and meconium-stained fluid. Respiratory distress can range from mild to severe, with varying degrees of cyanosis, tachypnea, retractions, grunting, nasal flaring, and coarse rales and rhonchi. The chest appears barrel-shaped (increased anteroposterior diameter) from gas trapping. Arterial blood gases may reflect varying degrees of hypoxemia, hypercarbia, and acidosis. The chest radiograph shows coarse, patchy areas of decreased aeration (atelectasis) and areas of hyperaeration (air trapping). Later, chemical pneumonitis can become apparent on the chest film.

Figure 7. Meconium aspiration syndrome.



Management

Prior to birth, early recognition of the compromised fetus, appropriate intervention, and prevention of cesarean delivery are preventive strategies for meconium aspiration (Meydanli et al. 2001; Sheiner et al. 2002; Yoder et al. 2002). Infusion of a sterile isotonic solution into the amniotic sac (amnioinfusion) is used to dilute meconium, correct oligohydramnios often associated with meconium-stained amniotic fluid, and decrease cord compression and acidemia. However, amnioinfusion does not reduce the risk for moderate to severe MAS or perinatal death and is not recommended for the prevention of MAS (Fraser et al. 2005).

Prior to recent research, nasopharyngeal and oropharyngeal suction were advocated as a preventive strategy for MAS. Results of a 12-center randomized controlled study found no significant difference in the need for mechanical ventilation, mortality, duration of oxygen use, number of ventilator days, and length of stay between suctioned and nonsuctioned groups of term infants born through meconium-stained amniotic fluid (Gardner et al. 2011; Vain et al. 2004). Since routine suctioning does not prevent MAS, suction is only recommended for depressed infants (nonvigorous neonates with depressed tone and respirations and/or a heart rate <100 BPM and neonates with respiratory symptoms (Velaphi and Vidyasagar 2006).

Additional treatment is required for infants who develop MAS. Because meconium in the alveoli can injure type II alveolar epithelial cells and interfere with endogenous surfactant production, surfactant replacement therapy may improve oxygenation. Respiratory status should be constantly monitored with pulse oximetry or transcutaneous monitoring, frequent blood gases, and clinical assessment to determine the need for oxygen and positive-pressure ventilation. Supportive care includes, but is not limited to, broad-spectrum antibiotics for suspected infection, correction of metabolic abnormalities, maintenance of fluid balance, a neutral thermal environment, and minimal stimulation. Sedatives and analgesics may be given if the infant's respiratory efforts interfere with effective positive-pressure ventilation (Table 9). Potential complications associated with MAS include air-leak syndrome, chemical pneumonitis, persistent pulmonary hypertension, and end-organ damage.

Other treatment interventions for MAS depend on the disease progression and may include high-frequency ventilation (discussed in the RDS section), nitric oxide, and extracorporeal membrane oxygenation (ECMO).

Inhaled nitric oxide (iNO), a potent and selective pulmonary vasodilator, is effective therapy for persistent pulmonary hypertension and reduces the need for ECMO. Inhaled nitric oxide selectively lowers pulmonary artery pressure and improves oxygenation without causing adverse effects on cardiac performance or systemic blood pressure.

It enhances gaseous exchange by improving ventilation-perfusion mismatching and decreasing intrapulmonary shunting. However, progressive atelectasis and decreased cardiac performance limit the effectiveness of iNO. Potential toxicities include both methemoglobinemia and direct lung injury from nitric dioxide.

ECMO is a process of prolonged cardiopulmonary bypass that provides cardiorespiratory support until the lungs recover. It is used with infants who have reversible lung disease and who have not responded to maximal medical therapy. ECMO is often used to treat infants with predictably fatal pulmonary failure from diseases such as MAS, and infants with a birth weight >2 kg with RDS, pneumonia and sepsis, and persistent pulmonary hypertension. ECMO is performed by either venoarterial or venovenous techniques. Significant complications accompany ECMO use so that specific selection criteria have been established to determine candidates for this highly invasive therapy.

Prognosis

(Orlando 1997)

Meconium aspiration syndrome is usually resolved by 1 week of life for infants who do not require assisted ventilation, but may persist in infants requiring prolonged assisted ventilation. The outcome depends on the severity of the asphyxial insult and the extent of lung damage caused by the disease and its potential complications.

Pneumonia

Neonatal pneumonia can be caused by bacterial, viral, protozoan, fungal, or other pathogens such as *Treponema pallidum* or *Chlamydia trachomatis*. It can occur as a primary infection or as part of a generalized infection.

Incidence

(Carey and Trotter 1997)

- 1% of term neonates
- 10% of preterm neonates

Infants at Risk/Predisposing Factors

- Premature infants
- Prolonged rupture of membranes >24 hours
- Excessive intrapartum manipulation
- Maternal fever
- Maternal viral, bacterial, or other infection
- Prolonged labor
- Maternal urinary tract infection
- Amnionitis
- Immature immune system
- Invasive procedures such as intubation and assisted ventilation
- Nosocomial infections acquired in the NICU

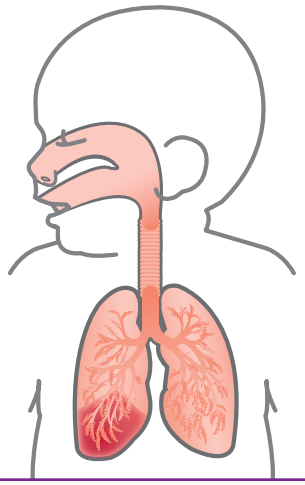
Pathophysiology

Transmission occurs transplacentally, intrapartally, or postnatally. Pathologic organisms include, but are not limited to, those listed in Table 10. Transplacental pneumonia can develop from aspiration or ingestion of infected amniotic fluid or from transmission of organisms from an infected mother across the placenta to the fetus. Intrapartum pneumonia results from colonization of the neonate by ascension of organisms after rupture of membranes, passage through an infected birth canal, or aspiration of infected amniotic fluid at birth or with resuscitative efforts. Postnatal pneumonia usually develops from hospital-acquired or nosocomial sources such as unwashed hands and open skin lesions, as well as contaminated equipment, nutritional products, or blood products.

Table 10. Pneumonia: Pathologic Organisms

Transplacental	Intrapartum	Postnatal Nosocomial
Cytomegalovirus	Herpes simplex virus	*Staphylococcus aureus*
Rubella	*C trachomatis*	*Staph. epidermidis*
T pallidum	Group B streptococci	Herpes simplex virus
Toxoplasma gondii	*Escherichia coli*	*Candida* sp
Varicella	*Klebsiella* sp	Cytomegalovirus
Enterovirus		Group B streptococci
Listeria monocytogenes		Enteroviruses
		Respiratory syncytial virus

Figure 8. Pneumonia.



Clinical Presentation and Diagnostic Workup

A high index of suspicion of pneumonia is the key to early diagnosis. The clinical presentation is often nonspecific and includes temperature instability, apnea, tachycardia, tachypnea, grunting, nasal flaring, retractions, lethargy, poor peripheral perfusion, and poor feeding. Skin lesions may be found in infants with congenital pneumonia caused by herpes simplex virus, *Candida* sp, or *T pallidum*. A shocklike syndrome is often seen in the first 7 days of life with early-onset group B ß-hemolytic streptococcus (GBS) sepsis.

Bacterial and viral cultures, rapid viral screening tests, and antigen tests (latex agglutination, counterimmunoelectrophoresis) should be performed on infants with suspected pneumonia. A Gram stain of tracheal aspirate may be useful if done during the first 8 hours of life, but it may not differentiate overt pulmonary infection from early colonization. A complete blood count with differential and platelets as well as a C-reactive protein (CRP) are useful diagnostic tests. The chest radiograph may show patchy opacifications, unilateral or bilateral alveolar infiltrates, pleural effusions, and/or changes in lung volume. GBS pneumonia is difficult to differentiate from RDS on a chest radiograph.

Management

For an infant with suspected bacterial pneumonia, broad-spectrum antibiotics, such as ampicillin and an aminoglycoside, should be started immediately and adjusted, if necessary, once the organism has been identified. Some viral pneumonias can be treated with pharmacologic agents such as acyclovir or vidarabine for herpes simplex virus, and ribavirin for respiratory syncytial virus (RSV). Supportive treatment is needed for respiratory problems, hematologic instability, and acid-base imbalance. Oxygen and positive-pressure ventilation may be required in addition to volume expanders, blood products, and vasopressors if the infant is in shock. With continued deterioration, the infant may require newer treatment options, including granulocyte transfusion, intravenous immunoglobulins, colony-stimulating factors, high-frequency ventilation, inhaled nitric oxide, and extracorporeal membrane oxygenation.

Prognosis

(Speer and Weisman 2003)

Overall mortality from sepsis, both related and unrelated to pneumonia, ranges from 5% to 10% in term infants and is as high as 67% in infants with a birth weight <1500 g.

Persistent Pulmonary Hypertension of the Newborn

Persistent pulmonary hypertension of the newborn (PPHN) has been described as the persistence of the cardiopulmonary pathway seen in the fetus, but without the passage of blood through the placenta. It is characterized by high resistance in the pulmonary arteries, which produces an obstruction of blood flow through the lungs and right-to-left shunting through the ductus arteriosus and/or foramen ovale. PPHN may be idiopathic or secondary to another disorder such as MAS or sepsis.

Incidence

(Wearden and Hansen 2003)

- 1.9:1000 live births

Infants at Risk/Predisposing Factors

- Late-preterm (“near-term”), term, or postterm neonates
- Underdevelopment of the pulmonary vascular bed—decreased cross-sectional area of pulmonary vascular bed secondary to hypoplasia (eg, Potter’s syndrome, diaphragmatic hernia)

- Maldevelopment of the pulmonary vascular bed—abnormal pulmonary vascular structure resulting in excessive muscularization (eg, fetal ductal closure, congenital heart disease)
- Maladaptation of the pulmonary vascular bed—functional pulmonary vasoconstriction with normal structural development and anatomy (eg, MAS, cold stress, asphyxia, sepsis)
- Maternal factors (tobacco use, obesity, diabetes, asthma, use of public insurance/lack of private insurance, black or Asian race, premature rupture of membranes)
- Cesarean delivery
- Large-for-gestational-age (LGA) infant

Pathophysiology

The neonatal pulmonary vasculature is sensitive to changes in arterial oxygen tension (PaO_2) and pH. With hypoxemia and acidemia, the pulmonary vasculature constricts, resulting in increased pulmonary vascular resistance and a ventilation/perfusion (V/Q) ratio mismatch. This leads to an impaired release of endogenous nitric oxide. High pulmonary vascular resistance promotes blood flow away from the lungs through the ductus arteriosus into the systemic system and results in right-to-left shunting. It also maintains higher right-sided pressures in the heart. When right atrial pressure is greater than left atrial pressure and pulmonary artery pressure is greater than systemic pressure, blood flow follows the path of least resistance through the foramen ovale and ductus arteriosus, again bypassing the lungs. This promotion of right-to-left shunting results in hypoxemia due to venous admixture. The cycle repeats as hypoxemia increases pulmonary vascular resistance, resulting in further intrapulmonary shunting, hypoxemia, and pulmonary vasoconstriction.

Clinical Presentation

Clinical presentation is variable due to the different etiologies of PPHN. Respiratory distress and cyanosis worsen despite high concentrations of inspired oxygen. Arterial blood gases demonstrate severe hypoxemia, normal or mildly elevated arterial carbon dioxide tension ($PaCO_2$), and metabolic acidosis. There is no classic chest radiograph finding for PPHN; rather, the x-ray reflects the underlying lung disease. It may show a prominent main pulmonary artery segment, mild to moderate cardiomegaly, and variable prominence of the pulmonary vasculature (normal or decreased vascular markings).

There is no classic chest radiograph finding for PPHN; rather, the x-ray reflects the underlying lung disease.

The diagnostic work-up for PPHN may include a hyperoxia/hyperventilation test and/or preductal and postductal PaO_2 tests. With the hyperoxia/hyperventilation test, the infant is placed in 100% FiO_2 and hyperventilated at rates >100 BPM. An increase in PaO_2 from <50 mm Hg before the test to >100 mm Hg after the test is indicative of PPHN. Preductal and postductal blood is sampled to demonstrate a right-to-left shunt through the ductus arteriosus. Blood is drawn simultaneously from a preductal site (right radial or either temporal artery) and a postductal site (umbilical, femoral, or posterior tibial artery). In the hypoxemic infant, ductal shunting is demonstrated with a PaO_2 difference >15–20 mm Hg between the preductal and postductal sites. Pulse oximetry also demonstrates an arterial oxygen percent saturation (SaO_2) difference between the right arm and the rest of the body and supports the diagnosis of PPHN.

Management

The goal of treatment is to correct hypoxemia and acidosis and promote pulmonary vascular dilation. Treatment consists of positive-pressure ventilation, pharmacologic support, supportive care, and perhaps the use of high-frequency ventilation, nitric oxide, and ECMO. Use of mechanical ventilation to produce alkalosis results in pulmonary vasodilation, which decreases pulmonary venous return and improves pulmonary perfusion and oxygenation. This approach is not without risks, as mechanical hyperventilation can impede venous blood return and reduce cardiac output, which further reduces

oxygenation. Induced hypocarbia can also diminish cerebral blood flow.

A more conservative approach attempts to minimize barotrauma while maintaining PaO_2 between 50 and 70 mm Hg and $PaCO_2$ between 40 and 60 mm Hg. The appropriate peak inspiratory pressure for either ventilatory approach is then determined by the infant's chest excursion.

Inhaled nitric oxide or ECMO may be needed if an infant does not respond to maximal medical treatment (see MAS Management section describing ECMO and nitric oxide).

Pharmacologic management includes a variety of agents. Vasopressors, which increase systemic vascular resistance, and volume expanders can be used to keep the systemic pressure normal or above normal in an attempt to reduce the pulmonary and systemic pressure gradient, thereby decreasing right-to-left shunting. Systemic vasodilators (such as tolazoline and sodium nitroprusside) have been used in the past to treat PPHN with variable and unpredictable results and are associated with systemic hypotension and other serious side effects. Because of their adverse effects, use of systemic vasodilators is no longer recommended, especially with the advent of selective pulmonary vasodilation obtained with iNO therapy. Use of phosphodiesterase inhibitors (such as milirone and sildenafil) improves vasodilation and increases cardiac output but has not been studied in large, randomized trials and is considered experimental (Gardner et al. 2011). Sedatives, analgesics, and muscle relaxants are used when the infant's respiratory efforts interfere with positive-pressure ventilation (Table 9). Supportive care includes continuous monitoring of arterial blood pressure, pulse oximetry, maintenance of fluid and electrolyte balance, and provision of a neutral thermal environment, hematologic support, and minimal stimulation.

Prognosis

(Hoskote et al. 2008; Gardner et al. 2011)

The prognosis varies according to disease etiology, severity, and mode of treatment. Treatment with iNO results in no increase in adverse pulmonary outcomes or neurodevelopmental/behavioral abnormality. Outcomes at 1 year in children treated with iNO show a lower incidence of respiratory morbidity than those treated with conventional mechanical ventilation or ECMO. Regardless of conventional therapy or treatment with ECMO, survivors of PPHN may have significant pulmonary and neurodevelopmental impairment.

Air-Leak Syndrome

Air leaks develop from alveolar rupture and the escape of air into tissue in which air is not normally present (pleura, mediastinum, pericardium, or extrathoracic areas).

Incidence

(Gardner et al. 2011)

The generation of increased lung pressure necessary for the first breath of life results in spontaneous air leaks in 2%–10% of healthy term neonates. The incidence of air-leak syndrome in the sick or preterm newborn varies with the underlying lung disease as well as with resuscitation and ventilation methods:

- 16%–36% of neonates resuscitated with bag/mask or bag/ETT or who are ventilated with CPAP or IMV
- 15%–33% incidence in term infants with MAS

Use of surfactant for RDS enables lower levels of ventilatory support, especially pressure, to adequately ventilate the immature lung and thus results in a lower incidence of air leaks.

Infants at Risk/Predisposing Factors

These factors include hypoplastic lungs, RDS, MAS, or congenital malformations; the use of ventilatory assitance or vigorous resuscitative efforts; and post-thoracic surgery.

Pathophysiology

Air leaks develop from abnormal distribution of gas and subsequent alveolar overdistention and rupture. Air ruptures out of the alveoli and moves along the pulmonary blood vessels or peribronchial tissues. The escaping air flows toward the point of least resistance. The location of the air leak determines which air-leak syndrome develops:

Pulmonary interstitial emphysema (PIE)	Air that is trapped in interstitial space
Pneumo-mediastinum	Air that has traveled along the pulmonary blood vessels and entered the mediastinum
Pneumothorax	Air that has escaped directly into the pleural space
Tension pneumothorax	Free pleural air that compresses the lung
Pneumo-pericardium	Air that has entered the space between the heart and the pericardial sac
Pneumo-peritoneum	Air that has traveled downward into the abdominal cavity and entered the peritoneal space via the postmediastinal openings in the diaphragm
Air embolism	Thought to arise when air ruptures out of alveoli into small pulmonary veins

Clinical Presentation

The clinical presentation of air-leak syndrome is outlined in Table 11.

Transillumination provides a preliminary diagnosis for pneumothorax. It works by placing a high-intensity, fiber optic light source over the chest wall and comparing the ring of the light bilaterally. Normal lung and pleura are dense, so light is absorbed. The presence of air pockets produces light around the fiber optic light. However, negative transillumination does not rule out pneumothorax. The *definitive* diagnosis for air leaks is a chest radiograph, either an anteroposterior (A-P) view or an A-P and a lateral view. The chest radiograph will identify the location and extent of air outside the tracheobronchial tree.

The definitive diagnosis for air leaks is a chest radiograph.

Management

A nitrogen washout can be used to treat pneumo-mediastinum or nontension pneumothorax. The infant is placed in 100% oxygen for 6–12 hours to establish a diffusion gradient between the pleural air and the pleural capillaries so that air is more rapidly absorbed by the capillaries. Nitrogen washout is not recommended for preterm infants because of the relationship between high oxygen concentrations in the blood and retinopathy of prematurity. Even use of 100% oxygen for “nitrogen washout” in term infants should be used with caution due to new understanding about the toxic effects of oxygen (oxidative stress) (Gardner et al. 2011). Tension pneumothoraces require emergency treatment. Needle aspiration can be used to remove air quickly and the catheter can be left in place until a chest tube is inserted. A chest (thoracostomy) tube and chest drainage system will restore negative pressure and expand the lung. Local anesthesia with 1% xylocaine and an analgesic should be given for pain relief.

Table 11. Air-Leak Syndrome: Types, Symptoms, Diagnoses, Management

Air Leak	Clinical Presentation	Chest Radiograph Findings	General Management
Pulmonary interstitial emphysema (PIE)	Increased oxygen requirements CO_2 retention Increased noncompliant lung	Small dark bubbles of air outside the tracheobronchial tree but trapped within the lung tissue	Positive-pressure ventilation High-frequency ventilation Optional: selective main stem bronchus intubation
Pneumomediastinum	Generally asymptomatic Tachypnea Bulging sternum	"Spinnaker sail sign": thymus gland lifted by the mediastinal air "Angel wing sign": both lobes of thymus lifted	Mediastinal tube placement
Pneumothorax	If symptomatic, see: Tachypnea Grunting Retractions Cyanosis	May not show any changes, or may show air in pleural space outlining the visceral pleura	Usually no specific management if air leak is small Needle aspiration and/or chest tube if air leak is large and neonate is compromised Optional: nitrogen washout
Tension pneumothorax	Tachypnea Grunting/retractions Cyanosis Hypotension Decreased breath sounds Chest asymmetry Shift in point of maximal impulse Distended abdomen	Pocket of air impinging on the lung Mediastinal shift may/may not be evident	Needle aspiration Chest tube placement
Pneumopericardium	Distant/absent heart sounds Bradycardia Diminished/absent pulses Marked hypotension Cyanosis and/or pallor Reduced EKG voltage	Dark circle surrounding the heart Decreased heart size	Needle aspiration Pericardial tube placement
Pneumoperitoneum	Distended abdomen Pulmonary function may be compromised	Dark layer over the abdomen Blurring or obscuring of normal bowel pattern	Usually none required Optional: insertion of soft catheter into the peritoneum
Air embolism	Catastrophic Sudden cyanosis Circulatory collapse Air-blood mixture crackles and pops with each heartbeat Air-blood mixture aspirated from the umbilical artery catheter	Bizarre picture of intracardiac and intravascular air	No effective treatment

Congenital Diaphragmatic Hernia

A congenital diaphragmatic hernia (CDH) is the herniation of the abdominal contents into the chest through a defect in the diaphragm. Ninety percent of these hernias occur on the left side and in the posterolateral portion of the diaphragm.

Incidence

(Lovvorn et al. 2011)

- 1:4000 live births
- More common in male than female infants

Infants at Risk/Predisposing Factors

- None

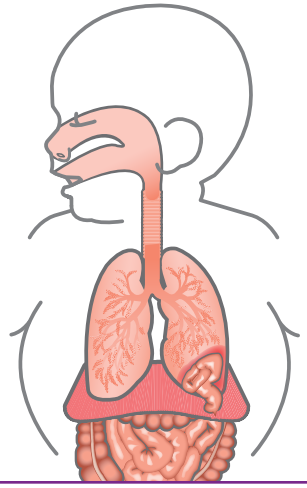
Pathophysiology

Closure of the diaphragm occurs at 8–10 weeks' gestational age. If closure is delayed, the bowel can move into the thoracic cavity and result in a diaphragmatic hernia. The stomach as well as the small and large bowel, spleen, and liver can also herniate into the chest. The presence of the abdominal contents in the thorax does more than just cause lung hypoplasia by compression. Decreased numbers of bronchial generations and alveoli are seen, and the pulmonary artery is small. Increased muscularization of the pulmonary arteries is also present. Both the bronchial and vascular changes restrict pulmonary blood flow, which can result in persistent pulmonary hypertension.

Clinical Presentation

A history of polyhydramnios is frequently associated with CDH, because the thoracic location of the intestine interferes with the intrauterine flow of amniotic fluid. Severity of signs and symptoms and age at onset depend on the extent of lung hypoplasia and the degree of interference with ventilation. Clinical presentation includes a scaphoid abdomen, barrel-shaped chest, cyanosis, dyspnea, retractions, shifted heart sounds, and decreased or absent breath sounds on the affected side. Chest and abdominal radiographs show loops of bowel in the chest (although these may not be evident until the infant has swallowed adequate air), sparse or absent abdominal bowel gas, a mediastinal shift, and a markedly elevated or indistinct diaphragm.

Figure 9. Congenital diaphragmatic hernia.



Management

Immediate recognition of the defect in the delivery room is a key component in management of an infant with a congenital diaphragmatic hernia. Bag-and-mask ventilation should be avoided to prevent the accumulation of air in the stomach and bowel, which will compromise respiratory expansion and worsen respiratory function. Instead, immediate intubation and ventilation, using the lowest possible pressure, should be instituted. A large double-lumen orogastric tube placed to low, intermittent suction prevents stomach and bowel distention. Other interventions include establishing intravenous access and providing a neutral thermal environment. Elevating the head of the bed and positioning the infant so that the affected side is down allows for maximal expansion of the unaffected lung. Sedatives, analgesics, and muscle relaxants are used for pain relief and asynchrony of infant- and ventilator-generated breaths (Table 9). The definitive treatment is surgical correction of the hernia. The development of pulmonary hypertension is frequently seen postoperatively (see Persistent Pulmonary Hypertension of the Newborn).

The definitive treatment is surgical correction of the hernia.

Prognosis

(Lovvorn et al. 2011; Miller et al. 2005)

Neonates with CDH who do not present with respiratory distress in the first 24 hours of life have a survival rate approaching 100%. For newborns with CDH who require mechanical ventilation in the first 18–24 hours of life, the survival rate is about 64%. Pulmonary hypoperfusion on the affected side may persist for years as the number of bronchi and alveoli remains reduced and increased muscularization of the pulmonary blood vessels continues. For survivors, gastroesophageal reflux can be a long-term problem after surgical repair.

Apnea of Prematurity

Apnea is a cessation of respiration lasting ≥20 seconds and associated with physiologic alterations such as bradycardia and/or color change. It can be obstructive, central, or mixed. With obstructive apnea, respiratory efforts are observed, but there is blocked air flow from collapse of the upper airway. Central apnea involves the cessation of both respiratory efforts and air flow, with no airway obstruction. The most common classification in preterm infants is mixed apnea, which involves a pause in respiratory effort preceded or followed by airway obstruction at the upper airway level.

Incidence

(Schmidt et al. 2007; Gardner et al. 2011)

Incidence of apnea is related to gestational age, so that the younger the preterm infant the greater the incidence of apnea: 85% of preterms <34 weeks gestation have apnea of prematurity.

Infants at Risk/Predisposing Factors

Primary apnea is a developmental phenomenon of preterm infants that must be distinguished from secondary apnea, which is apnea caused from another, treatable cause. Factors contributing to apnea in the preterm are listed in Table 12.

Pathophysiology

Apnea of prematurity is a diagnosis of exclusion when other underlying causes of apnea have been ruled out for infants of <37 weeks gestational age. It has been related to neuronal immaturity of brain stem function, which controls respirations. In addition, the central responsiveness to carbon dioxide is blunted in preterm infants. A diminished response to peripheral chemoreceptors located in both the aortic arch and the carotid arteries has

Table 12. Factors Contributing to Apnea

Infection	Septicemia, meningitis, pneumonia
Respiratory distress	Airway obstruction, CPAP application, congenital anomalies of the upper airway (eg, choanal atresia, laryngotracheal malacia), RDS, air leaks
Cardiovascular disorders	Congestive heart failure, patent ductus arteriosus, prostaglandin E infusion
Gastrointestinal disorders	Vomiting, necrotizing enterocolitis Gastroesophageal reflux as a factor contributing to apnea is not supported by research
Central Nervous System disorders	Analgesics/anesthetics (either intrapartal/postnatal), intraventicular hemorrhage, seizure, infection, bilirubin encephalopathy, tumors/ischemia
Metabolic disorders	Electrolyte imbalance (hypo/hypernatremia, hypocalcemia), hypoglycemia, inborn errors of metabolism
Hematopoietic	Anemia, polycythemia
Environmental	Hypothermia, rapid increase in environmental temperature, feeding, vigorous suctioning, stooling, stretching/movement, fatigue/stress, positioning, sleep state (increased apnea in active vs quiet sleep)

Modified from Gardner SL, Hines ME, Dickey LA: Respiratory diseases. In Gardner SL, Carter BS, Hines ME, Hernandez JA, eds: *Merenstein and Gardner's Handbook of Neonatal Intensive Care*, 7th ed. St Louis, Mosby; 2011.

also been noted. These receptors sense changes in PaO_2, pH, and $PaCO_2$ that affect the regulation of respirations and relay them to the respiratory center in the brain. Upper airway obstruction contributes to apnea because the negative pressure generated during inspiration may result in pharyngeal and laryngeal collapse.

Clinical Presentation

Cessation of respiratory effort with cyanosis, pallor, hypotonia, or bradycardia is noted. Frequent swallowing-like movements in the pharynx during apnea can be a problem, because swallowing directly inhibits the respiratory drive.

Management

Treatment of the infant with apnea of prematurity begins with assessment and monitoring (Table 13). Tactile stimulation, oxygen administration, and/or bag-and-mask ventilation can be used to stimulate an infant who is experiencing an apneic episode.

The standard long-term treatment for apnea of prematurity is the use of methylxanthines, specifically caffeine, theophylline, and aminophylline, which act on the brain stem respiratory neurons to exert a central stimulatory effect. Other effects include improved sensitivity to carbon dioxide response, increased diaphragmatic contractions, increased catecholamine activity, enhanced resting pharyngeal muscle tone, and decreased diaphragm fatigue. Doxapram, a peripheral chemoreceptor stimulator, has also been used for infants with apnea; it increases both minute ventilation and tidal volume. However, doxapram contains benzyl alcohol and requires continuous infusion, so it is not recommended for newborns.

The standard long-term treatment for apnea of prematurity is the use of methylxanthines.

Table 13. Apnea Monitoring

Type of Monitor	Characteristics/Capabilities	Problems
Impedance monitoring with EKG electrodes	Detects changes in electrical impedance as size of thorax increases and decreases during respiration	Unable to detect obstructive apnea Obstruction may not trigger a respiratory alarm Heart rate may not decrease with episode of apnea Sensitivity level usually set so monitor will sound in presence of shallow respirations (false alarm)
Pulse oximetry	Continuous measurement of hemoglobin saturation with oxygen	Decreased accuracy during hypoperfusion, hypothermia, and active movement
Three-channel pneumocardiogram	Assesses respiratory effort, heart rate, pulse oximetry Overnight or 24-hour recording	Motion and cardiogenic artifacts can interfere with respiratory signals
Two-channel pneumocardiogram	Assesses respiratory effort, heart rate Continuous recording on memory chip x 2–3 weeks Allows analysis of apnea, bradycardia, or both	Motion and cardiogenic artifacts can interfere with respiratory signals
Polysomnography	Expanded sleep study EEG, ECG, EMG, chest wall, and abdominal movement analysis, end-tidal CO_2, oxygen saturation, continuous esophageal pH determination, nasal air flow	Involves use of transducers and electrodes
Inductance plethysmography	Uses chest and abdominal belts to monitor respirations Detects obstructive apnea and decreases false alarms	Belt slippage Changes in body position

Infants who fail to respond to methylxanthines or who continue to have apnea while on these medications may respond to continuous positive airway pressure (CPAP), which increases functional residual capacity and stabilizes the chest wall. CPAP is beneficial to infants with mixed and obstructive apnea, but not central apnea. Infants who fail to respond to medications and CPAP require positive-pressure ventilation. Supportive care includes provision of a neutral thermal environment, use of pulse oximetry and/or transcutaneous monitoring, and positioning to prevent flexing of the neck.

Prognosis

(Pillekamp et al. 2007; Gardner et al. 2011)

Apnea of prematurity usually begins in the first week of life and spontaneously resolves at 36 weeks' postmenstrual age (PMA). Some infants may require home monitoring after discharge from the hospital.

Chronic Lung Disease (CLD)/ Bronchopulmonary Dysplasia (BPD)

Infants requiring supplemental oxygen at 28–30 days of life or at 36 weeks' PMA have CLD/BPD (Walsh et al. 2004). The "new BPD/CLD" of today's more preterm, lower-birth-weight infants may not have the characteristic x-ray changes of cystic lung disease. Arrested lung development resulting in interference with alveolarization and vascularization is characteristic of the "new BPD/CLD" (Clark et al. 2001).

Incidence

(Gardner et al. 2011)

CLD, a significant clinical problem, has an incidence of 23%–85% in very low-birth-weight preterm infants. This wide variation in incidence is due to variations in respiratory care practices that expose the immature lung to oxygen toxicity and barotraumas and volutrauma. Therefore, the incidence of CLD varies among NICUs; increased use of intubation, mechanical ventilation, and higher pressures is associated with more CLD/BPD, while use of noninvasive respiratory strategies, such as nasal continuous positive airway pressure (NCPAP), is associated with lower rates.

Infants at Risk/Predisposing Factors

Lung injury is iatrogenic with multifactorial predisposing factors interacting to result in CLD/BPD (Gardner et al. 2011).

- Preterm infants: inversely proportional to early gestation and low birth weight
- Male gender
- Respiratory support:
  - Oxygen toxicity from supplemental oxygen
  - Barotrauma/volutrauma from mechanical ventilation
- Patent ductus arteriosus
- Nurtitional deficiency: small-for-gestational-age (SGA) infants; poor postnatal nutrition; antioxidant deficiency of preterm infants
- Fluid overload
- Family history of asthma
- Infection and/or inflammation

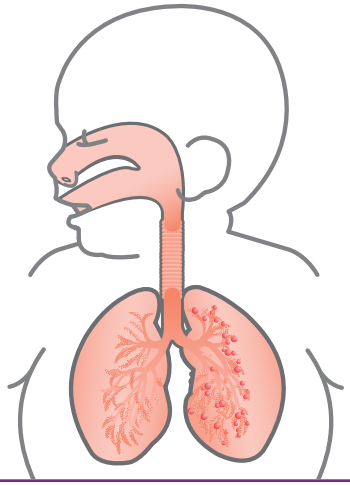
Pathophysiology

Oxygen toxicity, excessive tidal volume, and other contributory factors cause lung injury, which in turn produces an inflammatory reaction, capillary leak, abnormal lung mechanics/surfactant function, and airway obstruction. A pattern of constant and recurring lung injury, repair, and scarring occurs. This produces cellular, airway, and interstitial changes, including inflammation, atelectasis, emphysema, inactivation of surfactant, pulmonary edema, decreased lung compliance, increased airway resistance, ventilation/perfusion mismatch, overdistention, air trapping, and increased production of mucus. These pulmonary function disturbances lead to hypoxemia, hypercarbia, and some degree of bronchial hyperactivity. Bronchial hyperactivity and airway smooth-muscle hypertrophy (which decreases lumen size) cause bronchospasms or constrictions. The hypoxemia or ongoing marginal oxygenation induces pulmonary artery vasoconstriction, vascular muscular hypertrophy, and hypertension, resulting in pulmonary hypertension and subsequently increasing stress of the right-sided cardiac function.

Clinical Presentation

The most common alteration of pulmonary function in infants with CLD/BPD is increased airway resistance. In addition to low pulmonary compliance, this resistance results in increased work of breathing, hypoventilation, and retention of carbon dioxide. In infants with mild CLD, there is an initial need for positive-pressure ventilation, which must be maintained longer than was anticipated, followed by days or weeks of oxygen supplementation. Retractions, crepitant rales, and

Figure 10. Chronic lung disease.



diminished breath sounds occur. In the early phase of moderate to severe BPD, oxygen and ventilatory pressure requirements increase relentlessly. Chest radiographs show progressive overdistention of the lungs. Clinically, a barrel-shaped chest is noted, and the infant demonstrates lability with handling and acute episodes of bronchospasms. Generally, if respiratory support can be decreased during the first month of life, the subsequent course of BPD is relatively benign. But if increased support is needed at this time, a severe, protracted course is usual. BPD often becomes a progressive disease if it persists beyond 1 month of age. Growth failure is prominent and osteopenia is common. Right-sided cardiac failure, bronchospasms, inspiratory stridor, overproduction of airway secretions, and systemic hypertension are common in infants with progressive CLD/BPD (Table 14).

With mild BPD, chest radiograph findings are identical to those for RDS. As BPD progresses, coarse, irregular-shaped densities and air cysts start to develop. With advanced BPD, the lungs

Table 14. Overall Signs and Symptoms of CLD/BPD

Rapid and shallow respirations	Crackles
Increased work of breathing	Decreased air entry
Hyperinflated chest	Atelectasis
Hypoxemia	Hypercarbia
Pulmonary hypertension with right-sided cardiac failure	Intercostal/substernal retractions

appear bubbly (air cysts continue to enlarge) and are extensively hyperinflated, emphysema has progressed considerably, and cardiomegaly (indicating right-sided heart failure) is present.

Management

The treatment goals for CLD/BPD are to promote growth and to heal the infant's lungs. Oxygen administration and positive-pressure ventilation are both the cause of and the treatment for BPD. Adequate oxygenation is required to prevent recurrent hypoxemia and reduce pulmonary hypertension. This applies whether the infant is awake or asleep, crying or feeding. The lowest oxygen concentration and ventilator settings to produce acceptable ventilation and oxygenation should be used. Infants with CLD/BPD are often able to ventilate themselves before they are able to oxygenate, so that weaning the ventilator occurs before weaning from supplemental oxygen. Weaning from respiratory support should be done slowly to avoid the effects of hypoxemia (such as pulmonary hypertension) and hyperoxia (increasing lung injury and incidence and severity of retinopathy of prematurity [ROP]). Maintaining adequate hemoglobin concentration is necessary to maximize oxygen delivery to the tissues.

Pharmacologic management is critical for infants with BPD. Excessive interstitial fluid accumulates in the lung and can result in deterioration of pulmonary function, adding to the existing hypoxemia and hypercarbia. Pharmacologic management includes diuretics, bronchodilators, and steroids (Table 15).

Pharmacologic management is critical for infants with BPD.

Table 15. Pharmacologic Management of Infants with CLD/BPD		
Medication	**Action**	**Side Effects**
Diuretics	**Decreases interstitial fluid and pulmonary edema**	
Furosemide	Decreases interstitial pulmonary edema Lowers pulmonary vascular resistance and improves ventilation-perfusion ratios Improves lung compliance and lung mechanics Improves oxygenation Routine/sustained use in infants with or developing CLD cannot be recommended based on current evidence (Brion and Primhak 2002)	Electrolyte imbalance (hypokalemia, hypocalcemia, hypochloremia, hyponatremia) Metabolic acidosis Renal and gallstone formation Dehydration Ototoxicity Requires KCL supplementation
Thiazide Diuretics: Chlorothiazide Hydrochlorothiazide Used in combination with spironolactone, a potassium-sparing drug	Decreases interstitial pulmonary edema Improves pulmonary function Decreases airway resistance Increases pulmonary compliance Improves pulmonary function and lung mechanics Further study required to assess whether thiazides reduce mortality, decrease duration of supplemental oxygen, ventilator use, and length of stay and improve long-term outcomes (Brion et al. 2002)	Electrolyte imbalance (hypercalcemia, hypomagnesemia, hyponatremia, hypokalemia, hypophosphatemia) Hyperglycemia/glycosuria Metabolic alkalosis GI upset/vomiting, diarrhea Irritability/lethargy Rash **Do not use in patients with significant impairment of renal or hepatic function**
Bronchodilators	**Improves pulmonary mechanics**	
Inhaled: Albuterol Terbutaline sulfate Isoproterenol Ipratropium bromide	Increases surfactant production Decreases pulmonary edema Enhances mucociliary transport Increases lung compliance and decreases airway resistance	Tachycardia Tremors Hypertension Irritability Gastrointestinal disturbances (nausea, vomiting) Paradoxical bronchoconstriction
Systemic: Methylxanthines Caffeine citrate Aminophylline Theophylline	Decreases pulmonary resistance Stimulates central nervous system Increases inspiratory drive Improves skeletal muscle and diaphragm contractility and increases lung compliance Actions as noted for caffeine citrate Increases surfactant production	Mild Tachycardia, diuresis, dysrhythmias, hyperglycemia/glucosuria, ketonuria, vomiting/hemorrhagic gastritis, irritability, seizures Vomiting, tachycardia, tremors, gastroesophageal reflux, electrolyte abnormalities, agitation, GI irritation, hyperglycemia, CNS irritability, sleeplessness
Albuterol Terbutaline sulfate	Decreases pulmonary resistance/adjunct to methylxanthine	Tachycardia, tremors, hypertension, irritability, gastrointestinal disturbances, hypokalemia (albuterol)

table continued on next page

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Table 15. Pharmacologic Management of Infants with CLD/BPD

Medication	Action	Side Effects
Corticosteroids	**Promotes weaning from ventilator and decreases inflammatory response**	
Dexamethasone	Use of postnatal steroids is associated with both short and long-term adverse effects **Routine use of postnatal steroids to prevent/treat CLD is not recommended and should be limited to exceptional clinical circumstances, under carefully designed study protocols, and with written informed parental consent** (American Academy of Pediatrics 2006) Improves pulmonary status, probably by decreasing tracheobronchial and alveolar inflammation and decreasing pulmonary edema Facilitates gas exchange Increases lung compliance Diminishes airway resistance	Short-term adverse effects (Gardner et al. 2011) Hyperglycemia Hypothalamic-pituitary-adrenal axis suppression Renal calcification Protein depletion/tissue catabolism (failure to gain weight/increase BUN) Gastric irritation/perforation/bleeding Restlessness/irritability Myocardial hypertrophy Hypertension Increased risk for infection Long-term adverse effects (Gardner et al. 2011) Slower growth (somatic, head growth) Arrested lung development (alveolarization, vascularization) Neurotoxic: reduced brain size/growth; cerebral palsy; cognitive deficits Increased severity of retinopathy of prematurity Contributes to long-term conditions/diseases: renal, cardiovascular, immune system, neurologic, behavioral deficits

Diuretic therapy decreases excessive lung fluid. Bronchodilator and systemic methylxanthines have been used for both reactive airway disease and airway hyperreactivity. Corticosteroids promote weaning from the ventilator and decrease the inflammatory response, thereby improving pulmonary function.

Optimal nutrition is required for growth and development. Meeting the nutrient needs of a preterm infant may be limited by fluid restriction and feeding intolerance, therefore a high-calorie, nutrient-dense feeding would be advisable. Adequate vitamin A is critical for normal growth and differentiation of epithelial cells, and appropriate intake of minerals and vitamin D is necessary to prevent the development of osteopenia of prematurity.

The environment surrounding the infant is important for recovery from CLD/BPD. Minimizing agitation to prevent the hypoxemia and bronchospasms that often accompany agitation is essential. Sedation may be needed in addition to evaluation of noise, light, and touch to avoid overstimulation, which has a negative effect on weight gain, respiratory function, and development.

Prognosis

(Adams 2003; Barrington and Finer 1998)

Survival to discharge is inversely related to duration of ventilation. Improvement in pulmonary function occurs slowly over 1–3 years. Morbidities are most common in the smallest, sickest preterms and include: chronic respiratory difficulties, rehospitalizations in the first 2 years of life for respiratory infections, right-sided heart failure and cor pulmonale, growth restriction, osteopenia/fractures, cognitive impairment, cerebral palsy, behavior and attention problems, hearing loss, retinopathy of prematurity, and death. Overall mortality ranges from 25% to 40%, with most deaths related to infection or cardiopulmonary failure associated with pulmonary hypertension or cor pulmonale.

KEY LEARNINGS

- Respiratory distress syndrome (RDS) is caused by a primary absence or deficiency of surfactant.
- The goals of treating RDS are to prevent alveolar collapse, optimize tissue oxygenation and CO_2 elimination, minimize O_2 consumption, and provide supportive care.
- The most commonly cited cause of transient tachypnea of the newborn is delayed absorption of fetal lung fluid.
- Meconium aspiration syndrome (MAS) is the most common aspiration syndrome causing respiratory distress in newborns.
- Prior to birth, early recognition of the compromised fetus, appropriate intervention, and prevention of cesarean delivery are preventive strategies for MAS.
- Neonatal pneumonia can be caused by bacterial, viral, protozoan, fungal, or other pathogens.
- Persistent pulmonary hypertension of the newborn is characterized by high resistance in the pulmonary arteries, which produces an obstruction of blood flow.
- Air leaks develop from alveolar rupture and the escape of air into tissue.
- A congenital diaphragmatic hernia (CDH) is the herniation of the abdominal contents into the chest through a defect in the diaphragm.
- Recognition of CDH in the delivery room is a key component in management.
- Apnea is a cessation of respiration lasting ≥20 seconds and associated with physiologic alterations such as bradycardia and/or color change.
- The most common alteration of pulmonary function in infants with chronic lung disease (CLD)/bronchopulmonary dysplasia (BPD) is increased airway resistance.
- BPD often becomes a progressive disease if it persists beyond 1 month of age.
- Treatment goals for CLD/BPD are to promote growth and heal the lungs, pharmacologic management, optimal nutrition, and a proper environment.

Special Respiratory Concerns with the Premature Infant

The clinician or bedside caregiver should be alert to special respiratory concerns with the premature infant, as outlined in Table 16 (Gardner et al. 2011).

Table 16. Premature Infants: Special Respiratory Considerations

Concern/Condition	Impact/Result
Brain respiratory control center	May lack sufficient maturity to consistently regulate respirations; therefore, may experience periodic breathing and apnea
Compliant (immature) chest wall	Insufficient breathing and retractions
Noncompliant lungs	Increased work for respiratory muscles, leading to increased work of breathing and retractions
Surfactant deficiency	Collapsed alveoli plus intrapulmonary shunting, resulting in hypoxemia
Pulmonary vascular smooth muscle	Not as well developed as in term infants, so fall in pulmonary vascular resistance occurs more rapidly
Immaturity of terminal air sacs and associated vasculature	Poor gas exchange
Immaturity of diaphragm and other muscles of respiration	Inspiratory difficulty
Peripheral chemoreceptors (in aortic arch and carotid arteries)	Blunted response; therefore, can experience apnea
Muscle fiber type distribution	Muscles may be more susceptible to fatigue
Ductus arteriosus	Ductal smooth muscle does not have a fully developed constrictor response to oxygen Ductal tissue exhibits increased dilatory response to prostaglandins Persistently high circulating levels of prostaglandins May remain patent, shunting blood away from systemic organs
Lower hemoglobin	Limited oxygen-carrying capacity

KEY LEARNINGS

» Be alert for special respiratory concerns with the premature infant.

Related Nursing Care

Nursing Diagnosis:
Impaired Gas Exchange

Patient Outcome

- Infant will maintain adequate gas exchange and effective breathing pattern, as evidenced by:
  - — Respiratory rate 40–60 BPM
  - — Heart rate 110–160 BPM
  - — Clear and equal breath sounds
  - — Mild to no retractions
  - — Lack of nasal flaring and grunting
  - — Pink color
  - — Pulse oximetry values 92%–94% or as prescribed for gestational and chronological age and disease process
  - — Blood gases within normal limits

Interventions

- Assess for signs of impaired gas exchange/ respiratory distress every hour and as necessary (PRN)
  - — Nasal flaring
  - — Expiratory grunt
  - — Tachypnea
  - — Cyanosis
  - — Retractions—note type and degree
    - ▪ Type: suprasternal, substernal, intercostal, subcostal
    - ▪ Degree: mild, moderate, severe

- Auscultate breath sounds and note adventitious sounds every 1–2 hours and PRN
  - — Air movement
  - — Equality—compare and contrast each side of chest
  - — Clarity—clear, rales, rhonchi

- Maintain a patent airway:
  - — Small roll under shoulders (if needed)
  - — With endotracheal tube (ETT):
    - ▪ Suction PRN
    - ▪ Assess and document ETT size and position—note insertion depth (mark located at infant's lips)

- Use ETT adaptor or closed suction system to allow suctioning without removing infant from ventilator

— With nasal continuous positive airway pressure (NCPAP) or bubble CPAP:

- Keep infant calm; swaddle if necessary (crying releases pressure through mouth)
- Maintain patency of nares and nasal prongs
- Guard against pressure necrosis

- Administer oxygen in correct amount and by correct route of delivery

— Analyze and document inspired oxygen percentage every hour and with changes

— Document oxygen administration temperature

— Assess and document pulse oximetry every hour, changes in pulse oximetry values, interventions, and response to interventions

- Assess and document alarm limits every shift; assess and document ventilator settings every hour and with changes

- Assess, document, and notify physician/practitioner of blood gas results

- Maintain pulse oximetry (oxygen saturation) or transcutaneous monitor (transcutaneous and partial pressure of oxygen [$TcPO_2$] and carbon dioxide pressure [$TcPCO_2$])

— Pulse oximetry:

- Note probe site and change PRN
- Place probe so light source and photodetector are opposite one another
- Shield probe from ambient light, especially if phototherapy is in use
- Set monitor alarms according to unit policy
- Document readings every hour and PRN

— Transcutaneous monitor:

- Position probe on a flat, well-perfused area
- Change probe position every 4 hours and PRN
- Preferred temperature range: 43° C for preterm infants, 44° C for term infants
- Set monitor alarms according to unit policy
- Document readings every hour and PRN

- Maintain end-tidal CO_2 monitoring if ordered

- If chest tube required:

— Assist with transillumination process

— Assist with needle aspiration of chest

— Assist with chest tube placement:

- Administer analgesic
- Monitor vital signs during procedure
- Place chest tube to chest drainage system at 15–20 cm H_2O pressure
- Note bubbling activity

— Document tolerance to procedure

- If chest tube in place:

— Maintain tube stability:

- Tape all connections securely
- Secure tubing from infant to bed to relieve tension at insertion site
- Assess for kinks in tubing

— Do not strip/milk chest tube; this generates extremely high pressures

— Bubbling activity slows several hours after chest tube placement and usually stops after 72 hours

— If no bubbling noted for 24 hours, place chest tube to underwater seal (provides an outlet for any reaccumulated air after suction is discontinued); do not clamp tube

— After discontinuing chest tube, use an occlusive dressing (such as petrolatum gauze) for 48 hours

— Keep occlusive dressing at bedside for application at insertion site if chest tube becomes dislodged

— Assess and document amount of chest tube drainage every hour or every shift

— Assess and document bubbling activity every hour and PRN

— Reposition infant every 2–4 hours to facilitate removal of air

— Elevate head of bed

- Reposition infant every 2–4 hours
- Provide cluster care with minimal handling
- Assess infant's response to and tolerance of handling and procedures to determine appropriate nursing care
- Administer sedatives, analgesics, and muscle relaxants as ordered
  - — Assess response to medications
- Maintain neutral thermal environment
- Provide support to family

Nursing Diagnosis:

Ineffective Airway Clearance

Patient Outcome

- Infant will have an adequately clear airway, as evidenced by:
  - — Clear and equal breath sounds
  - — Respiratory rate 40–60 BPM
  - — Pink color
  - — Unlabored respirations

Interventions

- Assess respiratory status with continuous cardiorespiratory monitoring, with vital signs (every 2–4 hours) and PRN
- Administer aerosol medications
- Assess need for suctioning on the basis of:
  - — Quality of breath sounds
  - — Current condition
  - — Blood gas results
  - — Oxygen saturation readings
  - — General clinical appearance:
    - ▪ Chest movement
    - ▪ Color
- Suction PRN
  - — Use closed suctioning system with appropriate-sized suction catheters
  - — Assess patient tolerance of suctioning procedure
  - — Document amount, characteristics, and color of secretions
  - — Assess and document auscultatory findings of breath sounds before and after suctioning
- Initiate appropriate interventions to minimize hypoxia (bag ventilation presuctioning or postsuctioning)
  - — Determine degree of hypoxia by pulse oximeter or transcutaneous monitor readings and time to return to baseline
  - — Note degree of bradycardia, if any, and time to return to baseline
  - — Note any other physiologic changes
- Allow infant to rest after suctioning procedure and before other major stress activities
- Reposition infant every 2–4 hours and PRN
- Maintain adequate hydration

Additional Nursing Diagnoses:

These include, but are not limited to, the following:

High risk for injury: intraventricular hemorrhage, air leaks, or other related to treatment for respiratory disorders

Alteration in comfort: pain, related to chest tube placement and other procedures

High risk for fluid volume deficit: related to disease process, fluid loss, and IV administration

High risk for fluid volume excess: related to renal inability to excrete any volume overload and to iatrogenic fluid volume excess

Inadequate nutrition: less than body requirements, related to increased caloric expenditures and decreased nutritional intake

Knowledge deficit: related to lack of parental understanding of the disease process

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