Respiratory Disorders in the Newborn: Identification and Diagnosis

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Objectives
After reading this article, readers should be able to:

1. Describe the specific radiologic findings in neonatal respiratory distress syndrome.
2. Differentiate between the normal results of a newborn chest radiograph and the radiographic patterns that reflect meconium aspiration.
3. Differentiate between the normal results of a newborn chest radiograph and the radiographic patterns that reflect pneumonia.
4. Distinguish between pulmonary disease and cyanotic congenital heart disease as a cause of hypoxemia and acidosis in a neonate.

Background
Respiratory distress is encountered frequently in newborns and represents the most frequent indication for re-evaluation of the young infant. Because respiratory distress in the newborn may be a potentially life-threatening condition, physicians are expected to assess and manage affected infants promptly. The key to successful management of the infant who has respiratory distress is based on the ability to obtain a complete maternal and newborn history, perform a thorough physical examination, recognize the common respiratory disorders, differentiate among various diagnostic entities, and identify those that are life-threatening.

Definition
Respiratory distress in the newborn is characterized by one or more of the following: nasal flaring, chest retractions, tachypnea, and grunting. Nasal flaring is a relatively frequent finding in an infant attempting to decrease airway resistance. Suprasternal retraction indicates upper airway obstruction. Subcostal retraction, on the other hand, is a less specific sign that may be associated with either pulmonary or cardiac diseases. Normally, the neonate takes 30 to 60 breaths/min. The infant breathes at a faster rate to maintain ventilation in the face of decreased tidal volume. An infant in respiratory distress may try to maintain lung volume with adequate gas exchange by partially closing the glottis during expiration. This is the mechanism responsible for the audible grunting in these infants. An infant who has an advanced degree of respiratory distress may exhibit additional signs, such as cyanosis, gasping, choking, apnea, and stridor. The managing physician should consider these additional signs to be “alarming.”

Initial Assessment
The aim of the initial assessment of the infant in respiratory distress is to identify life-threatening conditions that require prompt support, such as inadequate or obstructed airway (gagging, choking, stridor), apnea or poor respiratory efforts, cyanosis, and circulatory collapse (bradycardia, hypotension, poor perfusion) (Table 1). The physician should manage these infants promptly with immediate oxygen support and possibly bag-and-mask ventilation or even intubation and mechanical ventilation. Therefore, resuscitation equipment and supplies always should be available for immediate use in the delivery room. Senior physicians and other health care team personnel should be prepared to perform resuscitation. Neonatal resuscitation guidelines should be followed in a stepwise manner.

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History

After the initial assessment and cardiorespiratory management, a history should be obtained. Maternal and obstetrical histories can provide invaluable information. Results of fetal assessment during pregnancy (triple screen, ultrasonography, biophysical profile) and fetal monitoring during labor and delivery (fetal heart rate, beat-to-beat variability, presence of decelerations) may provide useful information about a neonate in respiratory distress. Complications at delivery (eg, birth trauma, presence of meconium, perinatal depression) may be informative or play an important role in the development of respiratory distress. Figure 1 illustrates maternal and fetal conditions commonly associated with respiratory diseases.

Details of the presenting respiratory symptoms may be helpful. A history of coughing and choking in relation to feeding should be clarified. If these symptoms occur during the feeding, both functional (eg, oropharyngeal aspiration occasionally seen in preterm infants) and structural (eg, tracheoesophageal fistula) should be considered. A maternal history of polyhydramnios is consistent with the latter diagnosis when esophageal atresia is also present. If these symptoms follow the feeding, gastroesophageal reflux with aspiration should be suspected. These infants may have a history of recurrent emesis. Gradually improving symptoms are consistent with transient tachypnea of the newborn (TTN); gradual deterioration may be consistent with pneumonia or sepsis. Many nonrespiratory conditions can present with respiratory symptoms, including hypoglycemia, metabolic acidosis, anemia, heart failure, and sepsis. Information that may help to ascertain these conditions should be obtained.

Physical Examination

Inspection is the first and most important tool in a physical examination. Infants who exhibit apnea, poor respiratory effort, marked retractions, cyanosis, or poor perfusion require urgent attention.

Inspiratory stridor is consistent with upper airway obstruction. Stridor in an infant who has a previous history of endotracheal intubation may indicate airway injury, such as subglottic stenosis. Asymmetric chest movement combined with severe distress can be an alarming sign for tension pneumothorax. A scaphoid abdomen is the hallmark for congenital diaphragmatic hernia.

Auscultation is helpful in determining the symmetry and adequacy of air exchange. Abnormal breath sounds also should be identified. The presence of a heart murmur deserves special attention, although a murmur alone is not diagnostic of congenital heart disease. Neonates frequently present with heart murmurs that are “innocent,” and some serious congenital heart diseases may present without any murmur. Transillumination of the chest is useful to detect a pneumothorax on an emergent basis.

Differential Diagnosis of Respiratory Distress in the Newborn

Respiratory distress of the newborn is a common presentation for a wide variety of other diseases (Fig. 2). Parenchymal lung diseases, such as pneumonia, surfactant deficiency, and meconium aspiration, are the most common causes of respiratory distress in the neonate. However, airway and chest wall deformities as well as abnormalities of the diaphragm and mediastinal structures also can cause respiratory distress. Additionally, neurologic, cardiac, and metabolic abnormalities must be considered. Neonates who have congenital heart diseases commonly present with respiratory distress. Seizures, asphyxia, and meningitis are examples of neurologic disorders associated with respiratory signs. Common metabolic causes of respiratory distress include hypoglycemia and acidosis. Respiratory distress can be a presentation of hypothermia, sepsis, acidosis, polycythemia, or anemia.

Case Scenarios

A few common scenarios of infants presenting with respiratory distress follow.
**Case #1**
A 3.5-kg newborn male has tachypnea and acrocyanosis. Delivery was via an elective cesarean section at 41 weeks of gestation. Apgar scores were 7 and 8 at 1 and 5 minutes, respectively. The infant’s temperature is 96.4°F (35.8°C), heart rate is 153 beats/min, and respiratory rate is 82 breaths/min. He has slight substernal retractions. The remainder of the examination is normal. The chest radiograph is shown in Figure 3. At 18 hours after birth, the infant’s color is pink and respiratory rate is decreased to 48 breaths/min.

**Case #2**
A 3.9-kg female infant appears blue and tachypneic immediately after vaginal delivery. Amniotic fluid was stained with thick meconium. Apgar scores were 4 and 6 at 1 and 5 minutes, respectively. The infant’s temperature is 98.4°F (36.9°C), heart rate is 173 beats/min, respiratory rate is 110 breaths/min, and mean blood pressure is 61 mm Hg. She is barrel-chested and has retractions with poor-to-fair air entry. Coarse rales are audible bilaterally. The chest radiograph is shown in Figure 4.

**Case #3**
A 1.2-kg male infant is born at 32 weeks’ gestation. Apgar scores are 7 and 8 at 1 and 5 minutes, respectively. The infant is acrocyanotic and has grunting and marked retractions with breathing. The infant’s temperature is 96.8°F (36.0°C), heart rate is 170 beats/min, respiratory rate is 75 breaths/min, and mean blood pressure is 39 mm Hg. The chest radiograph is shown in Figure 5.

![Figure 1. Maternal and obstetric conditions associated with respiratory distress in neonates.](image-url)
Figure 2. Differential diagnosis for infants in respiratory distress.
TTN

TTN represents transient pulmonary edema resulting from delayed clearance of fetal lung liquid. It can occur in both term and preterm neonates. Lung liquid is produced actively in utero by a chloride pump that causes influx of chloride and water from the interstitium into the alveolar space. Approximately 2 to 3 days before delivery, lung liquid starts to clear due to an interesting transformation process in the pulmonary epithelium. The fetal pulmonary epithelium changes from a chloride-secreting membrane to a sodium-absorbing membrane, with reversal of the flow of lung liquid away from the airless alveolar spaces. Because this liquid contains very little protein, low oncotic pressure also favors the movement of water from the alveolar spaces into the interstitium. At this time, prostaglandin secretion by the fetoplacental unit increases to trigger labor. Prostaglandins are responsible for the lymphatic dilatation that accelerates clearance of liquid from the interstitium. After birth, when the lungs expand with air, water moves rapidly from air spaces to connective tissue, to be removed gradually from the lungs by the lymphatic system and pulmonary blood vessels.

Infants who have TTN present clinically with tachypnea and occasionally grunting and nasal flaring immediately after birth (Case #1). The incidence of TTN may be increased in infants born via elective cesarean section, especially before the onset of labor. Typically, arterial blood gases reveal respiratory acidosis and mild-to-moderate hypoxemia. Chest radiography reveals increased interstitial markings and occasionally fluid in the interlobar fissures (Fig. 3). Occasionally, pleural effusion or signs of alveolar edema may be seen. TTN is generally a benign, self-limited disease that usually responds well.
to oxygen therapy. Mechanical ventilation seldom is needed, although many infants require nasal continuous positive airway pressure support. The additional distending airway pressure may assist in maintaining alveolar surface area as well as absorbing the retained intralveolar fluid. Infants whose disease is uncomplicated usually recover without long-term pulmonary sequelae. Full recovery is expected within 2 to 5 days.

**Meconium Aspiration Syndrome (MAS)**

Meconium staining of amniotic fluid occurs in approximately 10% to 26% of all deliveries and almost exclusively in term and postterm deliveries. Meconium consists of water, desquamated cells from the alimentary tract, skin, lanugo hair, vernix, bile pigments, lipid, and mucopolysaccharides. Passage of meconium in utero may represent fetal hypoxemia. Meconium can be aspirated before, during, or after delivery. Once aspirated, meconium can cause obstruction of the air passages, chemical pneumonitis with activation of several inflammatory mediators, and inactivation of lung surfactant.

The infant who has MAS may present with varying degrees of respiratory distress and is likely to have a “barrel chest” with audible rales or rhonchi on auscultation (Case #2). The chest radiograph usually shows patchy areas of atelectasis alternating with areas of overinflation (Fig. 4). Pneumothorax is seen in 10% to 20% of infants who have MAS. It is recommended that obstetricians suction the infant’s mouth, pharynx, and nose as soon as the head is delivered, before delivery of the body, to prevent MAS. Once the infant is delivered and placed under the radiant warmer, the hypopharynx should be suctioned to clear any residual meconium. If the newborn has absent or depressed respiration, a heart rate of less than 100 beats/min, or poor muscle tone, direct tracheal visualization and suctioning should be performed to remove meconium from the airway.

**Hyaline Membrane Disease (HMD)**

HMD is a common condition in preterm infants. The cause is insufficient pulmonary surfactant, a mixture of lipids and proteins produced by type II pneumocytes. It serves to create a thin alveolar-lining layer that reduces the surface tension inside the alveoli, preventing them from collapsing.

The preterm infant who has HMD usually presents with respiratory distress beginning at or immediately after birth (Case #3). The blood gas typically reveals hypoxemia and respiratory acidosis. Radiographically, lungs demonstrate the typical “ground glass” appearance that represents diffuse atelectasis and “air bron-
iod are shown in Figure 2. Differentiation between CHD and pulmonary disease can be challenging, and the two can coexist. It is important to recognize that points of differentiation between heart and lung disease are cumulative rather than exclusive. A history of meconium staining of the amniotic fluid does not exclude the diagnosis of transposition of the great vessels. Alternatively, a heart murmur heard on physical examination does not exclude the possibility of pneumonia. Signs that generally are consistent with CHD include: visibly hyperactive precordial impulse, gallop rhythm, poor capillary refill, weak pulses, decreased or delayed pulses in lower extremities, hepatomegaly, and abnormal vascularity or cardiomegaly on chest radiography (Fig. 6). A single second heart sound without “split” also may be indicative of CHD. Recognition of this finding in infants requires considerable experience, and appreciating this can be challenging, especially in the presence of tachycardia. Infants who have isolated CHD usually do not have hypercapnia. The $P_{CO_2}$ may be decreased unless CHD is associated with lung disease. Metabolic acidosis is slightly more common in patients who have low cardiac output as a consequence of CHD, but it can occur in newborns who have infections or inborn errors of metabolism. Finally, it should be emphasized that complete echocardiography can detect most congenital heart defects.

Infants who have cyanotic heart diseases typically do not present with severe chest retraction. However, tachypnea is common. Oxygen saturation usually is decreased. Increasing the concentration of inspired oxygen to 100% for 20 minutes (“hyperoxygenation test”) will not produce a significant increase in $P_{AO_2}$ in most infants who have cyanotic CHD. Table 2 describes steps to help differentiate cyanotic heart diseases from pulmonary diseases.

### Table 2. Differentiation of Cyanotic Heart Diseases From Pulmonary Diseases Among Infants in Respiratory Distress

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Cyanotic Heart Diseases</th>
<th>Pulmonary Diseases</th>
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<tbody>
<tr>
<td><strong>History</strong></td>
<td>—Previous sibling who has CHD</td>
<td>—Maternal fever</td>
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<td></td>
<td>—Diagnosis of CHD by prenatal ultrasonography</td>
<td>—Meconium-stained amniotic fluid</td>
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<tr>
<td></td>
<td>—Preterm delivery</td>
<td>—Cyanosis</td>
</tr>
<tr>
<td><strong>Physical Findings</strong></td>
<td>—Cyanosis</td>
<td>—Severe retraction</td>
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<td></td>
<td>—Gallop rhythm</td>
<td>—Split second heart sound</td>
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<td></td>
<td>—Large liver</td>
<td>—Fever</td>
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<td></td>
<td>—Mild respiratory distress</td>
<td></td>
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<tr>
<td><strong>Chest Radiograph</strong></td>
<td>—Increased heart size</td>
<td>—Normal heart size</td>
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<td></td>
<td>—Decreased pulmonary vascularity (except in transposition of the great vessels and total anomalous pulmonary venous return)</td>
<td>—Abnormal pulmonary parenchyma, such as a) total whiteout or patches of consolidation in pneumonia, b) fluid in the fissures in TTN, or c) ground glass appearance in HMD</td>
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<tr>
<td><strong>Arterial Blood Gases</strong></td>
<td>—Normal or decreased $P_{CO_2}$</td>
<td>—Increased $P_{CO_2}$</td>
</tr>
<tr>
<td></td>
<td>—Decreased $P_{O_2}$</td>
<td>—Decreased $P_{O_2}$</td>
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<tr>
<td><strong>Hyperoxygenation test</strong></td>
<td>$P_{AO_2} &lt; 150$ mm Hg</td>
<td>$P_{AO_2} &gt; 150$ mm Hg (except in severe PPHN)</td>
</tr>
<tr>
<td><strong>Echocardiography</strong></td>
<td>Abnormal heart or vessels</td>
<td>Normal heart and vessels</td>
</tr>
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</table>

CHD = congenital heart diseases; HMD = hyaline membrane disease; TTN = transient tachpnea of the newborn; PPHN = persistent pulmonary hypertension of the newborn

### Suggested Reading


Peterson HG, Pendleton ME. Contrasting roentgenographic pulmonary patterns of the hyaline membrane and fetal-aspiration syndromes. AJR. 1989;73:800


PIR Quiz
Quiz also available online at www.pedsinreview.org.

10. A radiograph that reveals increased interstitial markings with fluid in the interlobar fissures in an infant who has respiratory distress is most likely to represent:
   A. Hyaline membrane disease.
   B. Lobar pneumonia.
   C. Meconium aspiration syndrome.
   D. Transient tachypnea of the newborn.
   E. Transposition of the great vessels.

11. The most likely diagnosis associated with the radiographic findings of a diffuse ground glass appearance to the parenchyma in an infant who has respiratory distress is:
   A. Hyaline membrane disease.
   B. Lobar pneumonia.
   C. Meconium aspiration syndrome.
   D. Transient tachypnea of the newborn.
   E. Transposition of the great vessels.

12. The radiographic finding of patchy atelectasis with areas of hyperinflation in an infant who has respiratory distress syndrome is most indicative of:
   A. Hyaline membrane disease.
   B. Lobar pneumonia.
   C. Meconium aspiration syndrome.
   D. Transient tachypnea of the newborn.
   E. Transposition of the great vessels.

13. For an infant who has respiratory distress syndrome, increased pulmonary vascularity on a radiograph most likely represents:
   A. Hyaline membrane disease.
   B. Lobar pneumonia.
   C. Meconium aspiration syndrome.
   D. Transient tachypnea of the newborn.
   E. Transposition of the great vessels.

14. Among the following, the information that is most helpful in distinguishing cyanotic heart disease from pulmonary parenchymal disease in a newborn who has respiratory distress is:
   A. Decreased Po₂ in blood gas analysis.
   B. Gestational age less than 32 weeks.
   C. Maternal infection during the third trimester.
   D. Respiratory rate of 70 breaths/min.
   E. Results of a hyperoxygenation test.