Meconium Aspiration Syndrome

The passage of meconium by the fetus *in utero* is estimated to occur in 8–29 percent of all deliveries.[57] However, meconium passage is seen primarily in fetuses born at or beyond term and among those who are small for gestational age or have umbilical cord complications and compromised uteroplacental circulation. During breech deliveries, meconium passage is common and is often ignored.

When meconium-stained amniotic fluid is detected, careful and continuous monitoring of fetal well-being is required during labor. The passage of meconium into the amniotic fluid is considered a sign of fetal distress when accompanied by fetal heart rate abnormalities.[58] Increased stillbirth and neonatal mortality rates have been associated with meconium staining. In the U.S., approximately 520,000 infants are born meconium stained annually. Five percent of these (about 26,000) develop meconium aspiration syndrome, and more than 4 percent (about 1,000) die from the disease. Approximately 30 percent of infants with meconium aspiration syndrome (about 7,800) require mechanical ventilation. Pneumothoraces occur in at least 2,900 of those infants requiring mechanical ventilation.[59] A decline in the number of postterm births has been identified as the most important factor in reducing the incidence of meconium aspiration syndrome by one-third.[60]

Etiology and Pathophysiology

Meconium is first produced during the fifth month of gestation. It is free of bacteria and contains residuals of gastrointestinal secretions. The pathophysiologic stimuli that trigger the fetal passage of meconium are not clearly understood.

The following theories have been proposed to explain the relationship between fetal hypoxia and the passage of meconium *in utero*.[57]

- Fetal gut ischemia resulting from decreased perfusion during the "diving reflex"
- Hyperperistalsis following an episode of intestinal ischemia
- Vagal stimulation elicited by umbilical cord compression, resulting in increased peristalsis and anal sphincter dilation

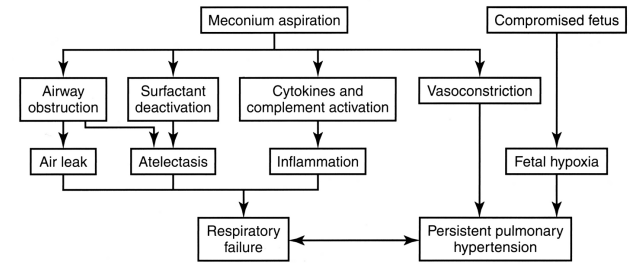
Meconium passage *in utero* is considered by some to be a normal physiologic function of term and postterm fetuses, indicating fetal maturity.[58] It is rarely observed in fetuses of less than 37 weeks gestation.

Fetal breathing movements occur in the healthy fetus at a rate of 30–70 times per minute. Normally, fluid from the airways moves out into the amniotic fluid with fetal respiratory movements. During an episode of fetal asphyxia, these movements cease, and apnea occurs. As the asphyxial episode continues, apnea is replaced by deep gasping. Amniotic fluid containing particulate material may be inhaled into the trachea and large bronchi, and the infant may demonstrate airway obstruction at birth. After the onset of air breathing, meconium migrates rapidly to the distal airways.

The amount of meconium passed into the amniotic fluid affects the appearance and viscosity of the fluid. Amniotic fluid containing meconium may have a light green tinge or the consistency and appearance of thick pea soup. Yellow, or "old," meconium-stained fluid indicates prolonged fetal hypoxia and is an ominous sign.[61]

Mechanical obstruction of the airways with meconium particles results in a ball-valve phenomenon. Complete obstruction of the smaller airways results in atelectasis of alveoli distal to the obstruction. Partial airway obstruction results in areas of overexpansion as air passes around the obstruction to inflate the

FIGURE 2-8
Pathophysiology of meconium aspiration syndrome.



From: Abu-Shaweesh JM. 2011. Respiratory disorders in preterm and term infants. In *Fanaroff & Martin's Neonatal-Perinatal Medicine,* 9th ed, Martin RJ, Fanaroff AA, and Walsh MC, eds. St. Louis: Elsevier Mosby, 1158. Reprinted by permission.

alveoli. As the airway collapses around the obstruction during expiration, residual air becomes trapped distally. Pneumothorax occurs when the overdistended alveoli rupture and air leaks into the pleural space. Pneumomediastinum results when extra-alveolar air moves through interstitial tissue to the mediastinum.

The chemical composition of meconium causes local toxic effects. Bile salts, pancreatic enzymes, desquamated intestinal epithelium, and biliverdin in meconium initiate a chemical pneumonitis that further compromises pulmonary function (Figure 2-8).[62] Surfactant function is disrupted by serum and nonserum proteins and fatty acids, leading to atelectasis, decreased lung compliance, and hypoxia.[63]

Clinical Presentation

Typically, an infant with meconium aspiration syndrome has a history of fetal distress and meconium-stained amniotic fluid. The classic postmature infant shows signs of weight loss with little subcutaneous fat remaining. The umbilical cord may be thin, with minimal Wharton's jelly. The nails, umbilical cord, and skin may be meconium stained. Respiratory distress at birth may be mild, moderate, or severe.

Tracheal occlusion by a meconium plug causes severe, gasping respirations; marked retractions; and poor air exchange. The severity of meconium aspiration syndrome is related to the amount of aspirated meconium. In mild cases, hypoxemia is present but easily corrected with minimal oxygen therapy; tachypnea is present but usually resolves within 72 hours. A low partial pressure of carbon dioxide in arterial blood ($PaCO_2$) and normal pH may be seen. Infants with moderate disease gradually worsen during the first 24 hours.

Severely affected infants have neurologic and respiratory depression at birth resulting from the hypoxic insult that precipitated the passage of meconium. They develop respiratory distress with cyanosis, nasal flaring, grunting, retracting, and tachypnea. The chest appears overinflated. Coarse crackles are common. Diminished breath sounds or heart tones may indicate a pulmonary air leak. Arterial blood gases typically show hypoxemia and acidosis. These infants have combined respiratory and metabolic acidosis secondary to respiratory failure and asphyxia. Because of large intrapulmonary shunts and persistence of fetal circulation patterns, hypoxemia is often profound despite administration of 100 percent oxygen.

Diagnosis: Radiographic Findings

Chest radiographs should be obtained to confirm the diagnosis of meconium aspiration and to rule out pulmonary air leaks. The classic radiographic picture of meconium aspiration syndrome includes coarse, patchy, irregular pulmonary infiltrates. Areas of irregular aeration are common, with some appearing atelectatic and others appearing emphysematous. Hyperaeration of the chest with flattening of the diaphragm is frequently seen. Pneumothorax and pneumomediastinum are common. Chemical pneumonitis may be apparent after 48 hours.[11,12] Massive aspiration is characterized by a "snowstorm" appearance. The extent of clinical and radiographic findings depends on the amount of meconium aspirated into the lungs (Figure 2-9).

Treatment and Nursing Care

Intrapartum and Immediate Postpartum Interventions

Prevention is the key to managing the infant at risk for meconium aspiration. Continuous electronic fetal monitoring is an essential tool for identifying the fetus in distress following passage of meconium *in utero*. Amnioinfusion (infusion of normal saline into the amniotic sac during labor) is used to correct oligohydramnios and decrease vagal stimulation caused by cord compression.[64] In prospective randomized studies, infants identified with thick meconium who received amnioinfusion had significantly fewer low one-minute Apgar scores, less meconium below the cords, and a significantly lower incidence of operative delivery.[65] However, other studies report continued occurrence of meconium aspiration syndrome and no improvement in neonatal outcome following prophylactic amnioinfusion for thick meconium.[66] Current evidence does not support

routine use of amnioinfusion to dilute meconium stained amniotic fluid. Furthermore, the intervention requires systematic study in clinical trials.[67] Several studies have demonstrated decreased mortality and morbidity when meconium is removed from the mouth, pharynx, and trachea before the onset of breathing.[68–70] More recent evidence from a multicenter trial failed to show a positive effect from oropharyngeal and nasopharyngeal suctioning before the delivery of the shoulders in meconium-stained infants.[71] Current recommended practice does not include routine intrapartum suctioning of infants delivered through meconium-stained amniotic fluid.[72,73]

Some investigators have questioned the need for routine tracheal suctioning at the birth of meconium-stained infants who are delivered vaginally and have a one-minute Apgar score of more than 8. In a prospective study, meconium-stained but vigorous infants who made their first inspiratory effort before being handed to the pediatrician did not benefit from immediate tracheal suctioning.[74] Furthermore, case reports have demonstrated that aggressive airway management during and immediately after birth does not always prevent aspiration of meconium.[75] The Neonatal Resuscitation Program guidelines recommend no tracheal suctioning for infants with strong respiratory efforts, good muscle tone, and a heart rate greater than 100 beats per minute. Direct tracheal suctioning is recommended for the meconium-stained infant with depressed respiratory effort, poor muscle tone, and a heart rate less than 100 beats per minute. This procedure should be accomplished before the infant makes repeated inspiratory efforts.

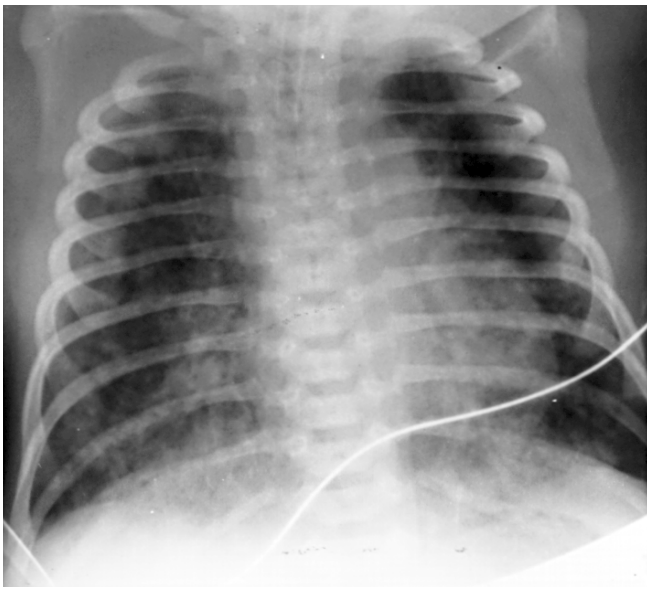
Universal precautions should be taken. Suctioning should always precede positive-pressure ventilation. Meconium aspirator devices and regulated wall suction should be utilized to effectively clear meconium from the airway. The urgent need for oxygenation and ventilation in these infants should not be ignored.[76,77]

Nursery Management

Supportive respiratory therapy is required for infants who develop meconium aspiration syndrome. The infant should be monitored continuously for tachypnea. Frequent assessment of blood gases is essential. The need for oxygen and assisted ventilation is dictated by arterial blood gas values. Continuous monitoring of oxygenation by pulse oximetry will alert the nurse to early deterioration. Ventilatory assistance is indicated when adequate oxygenation cannot be achieved or maintained in a high concentration of oxygen. Respiratory failure commonly occurs in severe cases of meconium aspiration and may necessitate prolonged assisted ventilation. Once the infant requires assisted ventilation, morbidity and mortality increase. Sedatives and neuromuscular blocking agents may be added to the therapeutic regime when the infant's own ventilatory efforts interfere with the effectiveness of mechanical ventilation.

FIGURE 2-9
AP view of the chest in an infant with meconium aspiration syndrome.

There are areas of patchy, asymmetric alveolar consolidation and volume loss in addition to areas of overexpansion resulting from obstruction (ball-valve effect). The lung fields are hyperexpanded.



Gastric lavage is used to remove meconium-stained fluid from the stomach and reduce the chance of further aspiration with vomiting. There is no evidence from studies to support this practice.[78] As noted under **Diagnosis: Radiographic Findings,** chest radiographs should be obtained to confirm the diagnosis of meconium aspiration and rule out pulmonary air leaks.

Chest physiotherapy (CPT) is used in many neonatal units to assist in mobilization of secretions and prevent accumulation of debris in the airway of neonates with respiratory distress. Percussion, vibration, and tracheal instillation of saline followed by suctioning are commonly performed in the delivery room and nursery following aspiration of meconium-stained amniotic fluid. There are no randomized controlled trials demonstrating positive short- or long-term effects of this therapy in neonates. Some infants may show signs of acute clinical deterioration with further hypoxemia and the need for increased oxygen following chest physiotherapy. There

is insufficient evidence to support the use of chest physiotherapy for meconium aspiration syndrome.[79]

Broad-spectrum antibiotic therapy is indicated when infection is suspected. Appropriate cultures should be obtained before starting therapy. Prophylactic use of antibiotics is a common practice in infants with meconium aspiration syndrome because it is difficult to distinguish on the chest radiograph from superimposed bacterial pneumonia. However, there is no evidence to suggest that prophylactic antibiotic therapy improves outcomes in nonventilated infants with meconium aspiration syndrome. No difference in duration of tachypnea, oxygen requirement, or need for NCPAP has been reported in a group of untreated, nonventilated infants with meconium aspiration syndrome. In the absence of perinatal risk factors for infection, these infants did not receive antibiotic therapy and had no evidence of bacteremia, pneumonia, or meningitis.[80]

There is no reported increase in bacteremia among meconium-stained infants when compared to nonstained infants. The decision to use antibiotic therapy for these infants is based on each infant's course.[81]

Surfactant replacement therapy early in the course of respiratory failure may reduce the severity of the disease in some infants. Surfactant therapy has been shown to reduce pulmonary air leaks, duration of mechanical ventilation and oxygen therapy, as well as length of hospital stay.[82] Further research is needed to determine the optimal timing, preparation, and method of surfactant administration for infants with meconium aspiration syndrome.

The infant should be carefully monitored for signs of seizure activity reflecting anoxic cerebral injury. Anticonvulsant therapy may be required. Metabolic derangements such as hypoglycemia and hypocalcemia require appropriate therapy and monitoring. Fluid balance is critical in these infants because cerebral edema and inappropriate secretion of antidiuretic hormone often occur following an asphyxial insult. Fluid restriction may be initiated early in the course of the disease. Careful monitoring of urine output is essential in the postasphyxial stage. Hematuria, oliguria, and anuria may indicate anoxic renal damage.

Recovery from meconium aspiration syndrome usually occurs within three to seven days in infants who do not require assisted ventilation. Those requiring assisted ventilation are usually ventilator dependent for three to seven days. Although the infant may be weaned successfully from assisted ventilation, tachypnea may persist for weeks. Pulmonary air leaks, persistent pulmonary hypertension, and pulmonary barotrauma often complicate the course of the disease. Prolonged ventilator therapy predisposes these infants to bronchopulmonary dysplasia with resulting oxygen dependency. More long-term deficits may be seen as sequelae of asphyxia.

The major cause of death in infants with meconium aspiration syndrome is respiratory failure. As noted earlier, surfactant replacement therapy may improve oxygenation and reduce the incidence of pulmonary air leaks. In some cases, however, the infant cannot be adequately oxygenated and ventilated with conventional respiratory support. Timely transfer to a tertiary level neonatal intensive care unit is essential. High-frequency ventilation and inhaled nitric oxide have been used for infants with respiratory failure and severe hypoxemia unresponsive to conventional mechanical ventilation. The combined use of surfactant, inhaled nitric oxide, and high-frequency oscillatory ventilation has resulted in a significant decrease in the need for the most invasive therapies such as ECMO.[83,84]

Careful consideration should be given before initiating treatment with high-frequency oscillatory ventilation and inhaled nitric oxide in facilities where ECMO is not available. Collaborative agreements with an ECMO center and a mechanism for timely transport of the infant are recommended.[85] Once nitric oxide therapy is initiated, transfer should take place without interruption of the treatment. A transport incubator equipped with a nitric oxide delivery system is required for these infants. Abrupt discontinuation of therapy can cause acute deterioration, with severe hypoxemia and possible death.[86,87] When all other treatment options fail to reverse respiratory failure, ECMO has been used in many of these infants to improve survival.[88]