10 Complications of Positive Pressure Ventilation

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The adaptation of mechanical ventilators for use in the neonatal population brought about a dramatic breakthrough in the care of premature infants. Further refinements and the development of technologies such as high-frequency ventilation combined with exogenous surfactant have further pushed back the boundaries of survival. Despite these advances, mechanical ventilation is not without risk. Barotrauma and volutrauma, resulting from the mechanical effects of positive pressure, and oxygen toxicity have harmful effects on many neonatal organs, including the lungs, heart, kidneys, eyes, and brain. Of special importance to all neonatal nurses is the risk for infection and airway trauma resulting from placement and use of the endotracheal tube. This chapter begins with a general discussion of lung trauma associated with volume, pressure, atelectasis, and oxygen. A review of some of the most common complications of mechanical ventilation—air leak syndromes, airway injury, pulmonary hemorrhage, and bronchopulmonary dysplasia (BPD)—follows. Patent ductus arteriosus (PDA) and retinopathy of prematurity (ROP) and their relationship to oxygen therapy are also discussed.

Air Leak Syndromes

Air leaks are produced by a rupture in the alveolus that allows air to escape into tissue where it is not normally found.[1] A review of the anatomy and physiology of the thorax and lungs will help the nurse understand why neonates are at especially high risk for developing air leak syndromes. The chest wall, or thoracic cage, consists of 12 thoracic vertebrae, 12 pairs of ribs, the sternum and diaphragm, and intercostal muscles. The cone-shaped thoracic skeleton is quite flexible because of the presence of cartilage. The major respiratory muscle, the diaphragm, stretches across the bottom of the thorax, separating the thorax from the abdomen. Within the thorax are three subdivisions: the two lungs and the mediastinum. The mediastinum contains the thymus gland, the great vessels, the thoracic duct and small lymph nodes, the heart, a branch of the phrenic nerve, and parts of the trachea and esophagus.

The lungs and the thoracic cavity are lined by a double-layer membrane, or pleura: The parietal pleura lines the chest wall, diaphragm, and mediastinum; the visceral pleura covers each lung. These membranes lie in continuous contact with each other and form a potential space, called the pleural space, that contains a thin layer of serous fluid for lubrication and cohesion.

The elastic tissues of the lung and chest wall pull in opposite directions, creating a negative, or subatmospheric, pressure in the pleural space. These pressures are approximately –2.5 to –10 cmH_2O from base to apex during respiration.[2] In situations where air enters the pleural space, it interferes with the negative pressure, resulting in partial or total collapse of the lung.

Neonatal air leaks occur when large transpulmonary pressure swings, uneven alveolar ventilation, and air trapping result in alveolar overdistention and rupture. Uneven ventilation occurs, not only in neonates with immature lungs, but also in those with meconium, blood, or amniotic fluid aspiration or hypoplastic lungs. The air ruptures occur at the alveolar bases, and the air tracks along the perivascular sheaths of the pulmonary blood vessels or peribronchial tissues to the roots of the lung.

TABLE 10-1
Sites of Air Leak Syndromes

Site of Extraneous Air	Syndrome
Pulmonary interstitium (perivascular sheaths)	Interstitial emphysema
Alveoli trabeculae-visceral pleura	Pseudocysts
Pleural space	Pneumothorax
Mediastinum	Pneumomediastinum
Pericardial space	Pneumopericardium
Perivascular sheaths (peripheral vessels)	Perivascular emphysema
Vascular lumina (blood)	Air embolus
Subcutaneous tissue	Subcutaneous emphysema
Retroperitoneal connective tissue	Retroperitoneal emphysema
Peritoneal space	Pneumoperitoneum
Intestinal wall	Pneumatosis intestinalis
Scrotum	Pneumoscrotum

From: Korones SB. 2011. Complications. In *Assisted Ventilation of the Neonate,* 5th ed., Goldsmith JP, and Karotkin EM, eds. Philadelphia: Saunders, 407. Reprinted by permission.

Air may then rupture into the pleura, mediastinum, pericardium, or extrathoracic areas (Table 10-1).

Air leaks occur in 1–2 percent of all newborns; however, only a small percentage of these infants (0.05–0.07 percent) are thought to demonstrate symptoms.[3] Since the advent of surfactant therapy and improvements in neonatal ventilator technology, the incidence of air leaks has declined significantly. In a study done before surfactant use, Yu and associates reported that among 230 infants weighing 500–999 g, 35 percent had pulmonary interstitial emphysema (PIE), 20 percent had pneumothorax, 3 percent had pneumomediastinum, and 2 percent had pneumopericardium.[4] Post surfactant reports for infants 24–32 weeks gestation found that the incidence of pneumothoraces ranged from 3.7 to 10 percent.[5–7] The use of synchronized modes of ventilation has also been reported to result in lower rates of air leaks.[8]

In addition to decreased lung compliance resulting from inadequate surfactant production, several structural differences contribute to the premature infant's increased risk of developing an air leak. In a seminal work published in 1935, Macklin identified the presence of alveolar pores (pores of Kohn), which allow gases to move between ventilated and nonventilated alveoli. Because these pores increase in size and number with increasing lung maturity, premature infants may lack sufficient communication between adjacent lung units to prevent asymmetric ventilation.[9]

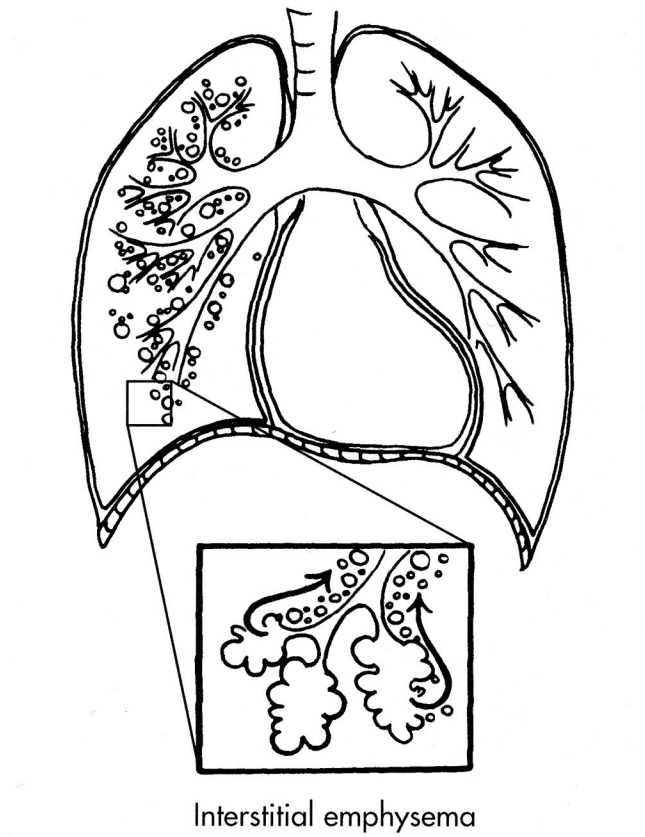
Risk factors for air leak syndromes include respiratory distress syndrome (RDS), meconium aspiration syndrome (MAS), hypoplastic lungs, congenital malformation, prematurity, endotracheal tube malposition, and overzealous resuscitation and suctioning.[10] Neonates on mechanical ventilation or continuous positive airway pressure (CPAP) are at much higher risk for air leaks, as are low birth weight (LBW) infants.[3] Sepsis and pneumonia caused by Pseudomonas or Candida have also been identified as potential risk factors.[11]

Mechanical ventilator factors that may increase the incidence of air leaks include positive end-expiratory pressure (PEEP), prolonged inspiratory time, high peak pressure, and breathing out of phase with the ventilator. An early study showed a 34 percent incidence of air leaks in infants receiving 3–8 cmH_2O of PEEP versus a 21 percent incidence in those not receiving PEEP.[12] One study reported a 50 percent incidence of air leaks when the inspiratory-to-expiratory (I:E) ratio was 1:1 or higher.[13] This finding was confirmed by a Cochrane review that identified a higher incidence of air leaks in infants ventilated with a long inspiratory time.[14] Prolonged inspiratory time can cause the infant to breathe against the ventilator, which can produce larger pressure and volume swings and lead to the rupture of alveoli. Studies have reported a higher incidence of air leaks with high peak inspiratory pressures (PIP) and mean airway pressures (Paw) >12 cmH_2O.[15,16] In a meta-analysis, patient-triggered ventilation was shown to decrease the risk of air leak compared with conventional ventilation.[17]

Pulmonary Interstitial Emphysema

PIE, a collection of gases in the connective tissue of the peribronchovascular sheaths, is a frequent complication in premature neonates with RDS who require mechanical ventilation.[10] Neonates with meconium or amniotic fluid aspiration or infection may also develop PIE, but premature infants are more prone to develop this condition because of their increased pulmonary connective tissue, which traps extra-alveolar air. Barotrauma, usually resulting from mechanical ventilation, combined with reduced lung compliance, causes rupture of small airways and alveoli, resulting in air in the interstitial spaces along the peribronchovascular, pleural, and interlobar passages.[10] This free air compromises lung ventilation

FIGURE 10-1
Pulmonary interstitial emphysema.

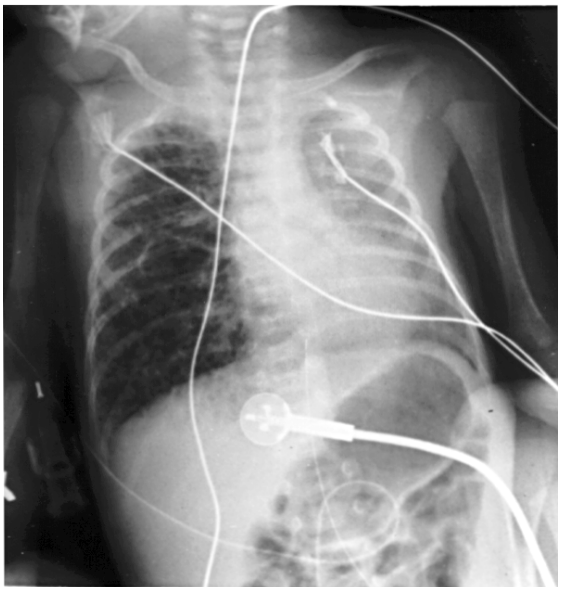


Adapted from: Korones S. 1986. Diseases of the lungs. In *High Risk Newborn Infants: The Basis for Intensive Nursing Care,* 4th ed. Philadelphia: Mosby, 252. Reprinted by permission.

and pulmonary vascular circulation because it compresses alveoli and blood vessels (Figure 10-1). As a result, lung compliance decreases and pulmonary vascular resistance increases. There are case reports of PIE occurring in LBW infants receiving CPAP and in premature infants before CPAP or mechanical ventilation is initiated.[18–20] A study by Verma and colleagues also noted an independent relationship between antenatal magnesium sulfate exposure and PIE in extremely low birth weight (ELBW) infants.[21]

There are two varieties of PIE: a localized form and a diffuse form. The localized, unilateral form may involve one or more lobes of the lung and may be accompanied by mediastinal shift. Diffuse PIE occurs more often in premature infants on mechanical ventilation, because of barotrauma. Morbidity and mortality are highest in low birth weight and lower gestational age infants who develop PIE in the first 48 hours of life.[10] Premature infants with PIE are at great risk for developing BPD

FIGURE 10-2
Pulmonary interstitial emphysema.



and other air leak syndromes. In a study by Greenough and colleagues, 31 of 41 infants with PIE developed a pneumothorax, and 21 of these babies also developed an intraventricular hemorrhage (IVH).[22]

Clinically, neonates with PIE often exhibit deterioration in respiratory and cardiac status, necessitating additional ventilatory support. This can lead to a vicious cycle of increasing pressure causing more PIE.

The diagnosis of PIE is made radiographically. The classic picture is a "salt and pepper" pattern in which cyst-like radiolucent air pockets are visible against the dark background of lung parenchyma. Overinflation may be noted on the affected side (Figure 10-2). In some cases, the overinflated cysts characteristic of PIE can further enlarge to form pneumatoceles, which are visible on x-ray as cystic blebs.

When the diagnosis is in doubt, CT scanning has been shown to be of value in confirming the presence of PIE.[19]

Treatment

Several medical regimens—from conservative to surgical interventions—have been recommended for infants with PIE. In some infants, unilateral PIE can be managed by placing the neonate with the affected side down. This position improves oxygenation in the unaffected lung and may allow a reduction in PIP, which will help to resolve the PIE. If this approach is unsuccessful, selective mainstem bronchus intubation

and bronchial occlusion are recommended.[23] The bronchus of the unaffected side is intubated for preferential ventilation while the affected lung resorbs interstitial air and becomes atelectatic. Improvement is generally seen in 3–72 hours.[24,25] Complications of this treatment include difficulty in left-side intubation, bronchial mucosal damage, infection, excessive secretions, hyperinflation of the intubated lung, and further air trapping.[10]

High-frequency ventilation—including high-frequency positive pressure, jet, and oscillatory ventilation—has been used effectively to treat diffuse PIE. In one study of 18 premature infants, high-frequency oscillatory ventilation was effective in improving oxygenation, CO_2 elimination, and circulation in infants with RDS and PIE.[26] High-frequency ventilation allows for adequate minute ventilation using lower airway pressures, which may reduce the amount of air leaking into the interstitial space.

A variety of other strategies, such as percutaneous evacuation of enlarged pneumatoceles, has been described in case reports.[27,28] Surgical intervention—including pleurotomy, pneumonotomy, pneumonectomy, and lobectomy—has been utilized when the neonate does not respond to medical management.

Nursing Care

BPD is a frequent sequela in neonates surviving PIE. Nursing care of the neonate with PIE begins with close monitoring of all neonates who are intubated and mechanically ventilated. Initially, the nurse will note increasing oxygen and pressure requirements based on falling oxygen saturations and poor blood gas readings. Hypotension may also be noted.

Ventilatory management is crucial in preventing the development of further PIE. The endotracheal tube should be maintained in the proper position, above the level of the carina. Although the goal is to decrease Paw, thereby preventing further air leaks, neonates with lung disease often require higher levels of PIP and PEEP. Barotrauma can be reduced by using a synchronized mode of ventilation.[17]

The nurse should closely monitor oxygen saturations and blood gas levels so that ventilator changes can be made promptly. If the treatment of PIE necessitates the use of high-frequency ventilation, the nurse must be familiar with the equipment and maintain a high level of vigilance.

When treating PIE conservatively, the nurse should position the neonate on the affected side, using oxygen saturation levels and vital signs to monitor tolerance of the change in position. Follow-up x-ray examinations will determine if more aggressive therapy is needed.

Neonates who are treated with selective mainstem bronchus intubation should be monitored continuously. Adequate humidification and appropriate suctioning are vital to prevent plugging of the endotracheal tube and further development of PIE.