Chronic Lung Disease of Infancy:

Oxygen Delivery Devices and Airway Management

by Steven E. Sittig, RRT

Chronic lung disease (CLD) in newborns is most commonly referred to as bronchopulmonary dysplasia (BPD), and it is a major contributor to the morbidity and mortality of infants born prematurely. Northway was the first to describe BPD in an article in a 1967 issue of the *New England Journal of Medicine*. It was Northway and his colleagues who introduced the term bronchopulmonary dysplasia.

Bronchopulmonary dysplasia is the term for the condition seen in primarily premature newborns who have respiratory distress syndrome requiring high levels of mechanical ventilatory support with high inspiratory pressures and high-inspired levels of oxygen. BPD ranks with cystic fibrosis and asthma among the most common chronic lung diseases in infants in the United States.

The risk of BPD increases with decreased birthweight and gestational age. It occurs in 5 percent of infants whose birthweight is more than 1,500 grams, but the incidence rises to 85 percent in surviving newborns weighing between 500 to 700 grams. With the advent of more intensive neonatal critical care, these infants of very low birthweight are now surviving with a higher likelihood of developing BPD.

BPD is a disease in which early lung damage is incompletely and inadequately repaired. Physiologic changes occur at all levels in the lung with the development of BPD in the airways, alveoli, and interstitium. With lung injury, there is an influx of inflammatory cells and mediators into the airways. The alveolar-capillary membrane then becomes more permeable, leading to the development of interstitial edema and epithelial swelling. There is also an increased chance of developing hyperreactive airways.

Following the development of the interstitial edema and epithelial swelling, the alveolar walls become thickened and fibrotic, leading to impaired gas exchange. With higher levels of inspired oxygen, ciliary action is inhibited leading to retained secretions causing atelectasis, mucus plugging, and pneumonia.

Bancalari proposed the initial definition of BPD. His definition included such factors as respiratory failure that required at least three days of ventilatory support, continuing respiratory symptoms, and a dependence on supplemental oxygen at 28 days postnatal age, along with radiographic changes such as a mixture of areas of atelectasis and areas of hyper-

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lucent cystic areas.[1] A newer definition is now used in studies investigating ways to reduce BPD. This definition includes the radiographic findings and history of mechanical ventilation, but it quantifies the need for supplemental oxygen at 36 weeks post-conceptual age.[2,3] It was felt this new definition would better identify the infant who would have a higher incidence of ongoing respiratory problems.

The rationale for the use of supplemental oxygen therapy in the treatment of BPD is to treat hypoxemia and potential pulmonary hypertension secondary to hypoxic pulmonary vasoconstriction. Alveolar hypoxia is an important cause of elevated pulmonary artery pressure and, if chronic, may lead to alteration of the structure in the pulmonary vascular bed. Autopsy studies of children with BPD reveal abnormal muscularization of the small pulmonary arteries and overgrowth of the inner layer of blood vessels.

Pulmonary hypertension and the attendant risk of development of irreversible cor pulmonale are serious complications of BPD. The therapeutic goal for long-term oxygen therapy is to maintain the PaO_2 (arterial oxygen tension) level within normal physiologic range. Therefore, alleviating hypoxia is important in reducing pulmonary artery pressure and may also reduce subsequent damage to the pulmonary vascular bed.

Oxygen therapy and airway management

There are many different approaches for the treatment of the BPD child, and approaches must often be tailored to each child. Oxygen therapy is one of the most important therapies for the clinician to address when considering how to help the child survive and thrive.

The most common way to deliver this supplemental oxygen is by low-flow nasal cannula. Although this is a popular method to deliver oxygen to this patient population, it is very difficult to determine the approximate percentage of oxygen delivered to the patient. In the neonatal intensive care unit setting, some overcome this issue by using blenders to adjust inspired FIO_2 (fraction of inspired oxygen) through the cannula. Still, one must be concerned with flow rates through the cannula because higher flows can sometimes produce varying levels of continuous positive airway pressure to the child.[4] In some practices, the oxygen is also humidified to reduce nasal mucosal drying from the cannula flow. Therefore, it is important to titrate the cannula flow to keep the currently recommended saturation levels around 93 to 95 percent. This supplemental oxygen has an additional benefit for these infants, as they generally experience increased growth and development, along with increased weight gain.[5]

Occasionally more severely affected newborns with BPD may fail extubation and require a tracheostomy, particularly those who acquire lesions of the glottis and trachea. There are actually several conditions that would indicate the need for this longer-term artificial airway. The most common reasons would be in cases of severe tracheal subglottic stenosis or vocal cord immobility from endotracheal tube irritation causing fixation or paralysis. This vocal cord dysfunction may be either unilateral or bilateral.

Acquired subglottic stenosis is related to an inconsistent cascade of events that causes 1 to 8 percent of infants to develop this condition after prolonged intubation.

The most commonly affected area in these children is the cricoid. Proposed theories regarding the etiology and pathogenesis agree on many important factors, such as small cricoid, reflux, infection, endotracheal tube size, type, movement, and replacement among others. The respiratory epithelium is very susceptible to irritation; and if it persists, the original edema and hyperemia progress to ulceration and local infection with growth of granulation tissue. When the source of the irritation is removed, healing can begin.

Scar tissue usually forms circumferentially, which is caused by the cricoid cartilage forcing the scar tissue to grow inward and decrease the airway lumen. Subglottic stenosis is graded according to the Cotton classification scale of 1 to 4. Most infants who need a tracheostomy have either a severe grade 2 (approximately 70 percent airway occlusion with some lumen) to grade 4, which is a 100 percent occlusion of the airway.[6]

Since most of the infants who develop BPD are quite premature, they are also at increased risk for developing intracranial hemorrhage. These hemorrhages can lead to severe neurologic impairment and leave the newborn unable to protect his own airway.

The addition of a tracheostomy would require additional training for the parents in preparation for discharge from the hospital. Tracheostomies are generally well tolerated by infants but require diligent care and observation by the caregivers. It is also important that a pediatric ear, nose, and throat specialist follow the infant as he grows and his airways change. While the sight of a tracheostomy may seem imposing to most parents, with proper education and medical management, the patient and family will do well.

Pharmacologic management

Since BPD also has an inflammatory component and occasionally a component of bronchospasm, much attention has been focused on the use of steroids and bronchodilator therapy in managing BPD. The use of systemic steroids has been shown to decrease the concentration of inflammatory markers in tracheobronchial secretions and can rapidly improve pulmonary compliance, resistance, and an improvement of gas exchange in ventilator-dependent children.[7]

Systemic administration of dexamethasone within 14 days after birth reduces CLD.[8]

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additional reading

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Steroids can be given via the inhalation route, but the clinician must make sure an appropriate dose from the metered dose inhaler (MDI) is being delivered. The number of accentuations of an MDI must be adjusted to deliver the needed dosage. In two randomized trials, the inhalation route was shown to be associated with a slower onset and slower effect. Ten days of 1,500 micrograms/day of inhaled beclomethasone was less effective than three days of systemic steroids in reducing levels of inflammatory mediators and inspired oxygen requirement.[9]

Bronchodilator therapy is also a common therapeutic modality applied to infants with CLD. As previously mentioned, sometimes there is an element of bronchospasm that needs to be addressed. Once again, giving an adequate dosage is very important. Due to the short inspiratory times and small tidal volumes, deposition of bronchodilator can vary.

The Clark rule of calculating the bronchodilator dosage (0.01–0.03 mg/kg) can at times lead to a subtheraputic dosage of delivered bronchodilator. Since such a potentially small amount of drug reaches the patient's airways, it would be easy to assume that frequent and routine aerosols are needed due to lack of response. This continuous use of bronchodilator can lead to the development of tolerance to bronchodilator as seen in many adult asthmatics. Conversely, several studies have noted a paradoxical effect with bronchodilator therapy in infants with undetected tracheomalacia in BPD patients. The increased airway smooth muscle tone may play a protective role in maintaining airway wall rigidity.[10]

Using bronchodilators may affect the airway muscle tone and cause further airway instability and airflow limitation. Therefore, the utilization of bronchodilators needs to be specifically tailored to each infant with BPD. If wheezing is still present, despite the addition of a bronchodilator, it needs to be determined whether the wheezing is due to bronchospasm or possibly from airway edema and inflammation that might require diuretics.

The basis of diuretic therapy in BPD is due to the early clinical picture that these patients present with, including clinical, radiographic, and histologic evidence of both interstitial and peribronchiolar pulmonary edema.[11] Infants with BPD have abnormal endogenous regulation of water balance and are predisposed to hypervolemia and the development of pulmonary edema. This concentrates near the airways and causes small airway narrowing, then reduction of dynamic compliance and increased airway resistance. Administration of Lasix (furosemide) by either systemic or aerosolized routes appears to help return this fluid balance to normal and improve pulmonary mechanics.

Because more newborns are surviving extreme prematurity and are more likely to develop CLD, a multidisciplinary approach to their care plan is needed. Any pharmacologic approach must weigh proposed benefits against risks of long-term therapy. Neonatologists, nurses, and respiratory therapists need to work together to determine what therapy is needed to assure the infant will be discharged to home with a tailor-made care plan.

In addition, it is crucial for experienced home care personnel to educate the infant's parents on how to administer the best possible care, along with the setup of any required home care equipment, such as oxygen and/or apnea monitoring. Routine follow-up is also important to ensure the child is doing well and progressing to where he will eventually outgrow his CLD.

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