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Bronchopulmonary Dysplasia

Michael A. Matthay*

Division of Pulmonary Medicine, Golden Gate University, San Francisco, CA, USA

Editorial

Bronchopulmonary Dysplasia (BPD), often known as chronic lung disease, is a disorder in the development of a baby's lung tissue. Bronchopulmonary dysplasia is a condition that affects babies who are born prematurely or who have breathing problems after birth (brahn-ko-PUL-moh-nair-ee dis-PLAY-zhee-uh). Most infants improve and have minimal long-term health issues, while some require extensive medical attention. It's more common in low-weight babies who were born more than two months early.

Bronchopulmonary dysplasia is also known by the following terms:

- Premature babies' chronic lung disease
- Infancy-related chronic lung disease
- Chronic pulmonary illness in newborns
- Insufficient breathing

Description

The severity of bronchopulmonary dysplasia can range from minor to severe. Many infants recover completely from this condition. Others may have breathing problems throughout their first two years of life, as well as in their teen and adult years. Babies with this disease are frequently admitted to the hospital and require extensive care. In newborns with respiratory distress syndrome, bronchopulmonary dysplasia is common (RDS). Because their lungs have not fully developed, this respiratory issue is frequent in premature babies. This illness could affect up to 10,000 infants in the United States each year.

This disease usually occurs after a premature baby is given extra oxygen or has been on a breathing machine (mechanical ventilator). A baby's lungs have not fully developed when he is delivered prematurely, and oxygen is required. This makes it easier for the baby to breathe. However, delivering oxygen under pressure, such as through a ventilator, might harm the lungs' air sacs. Bronchopulmonary dysplasia can result from this. Infants who had an infection before or shortly after birth can also develop the disease [1].

The condition is not present at birth. When a newborn has been on oxygen or a breathing machine for a long time, this can happen. This can cause inflammation (swelling and irritation) and scarring in the lungs. As a result, the lungs fail to grow properly. Premature newborns are more susceptible.

For babies whose lungs are too immature to breathe on their own, mechanical ventilators are used. A tube put into the baby's trachea delivers oxygen to their lungs (windpipe). The gadget employs pressure to push air into the baby's immature lungs, which are inflexible. Although many babies do not require a breathing tube, they still require additional oxygen and pressure. The oxygen and pressure are delivered to the patient through nasal prongs [2].

These newborns require a higher concentration of oxygen than that found in the air we breathe. The additional oxygen intake and pressure from ventilation might harm a newborn's sensitive lungs over time. Long-term lung damage limits normal lung development. As a result, these babies' breathing issues persist, necessitating the delivery of more oxygen to their lungs. Bronchopulmonary dysplasia is a condition that affects premature babies who require oxygen therapy for more than 28 days [3].

Bronchopulmonary dysplasia can occur when a newborn's lungs are affected by another condition, such as birth abnormalities, heart problems, pneumonia, or other diseases. Even full-term neonates might develop BPD due to inflammation and scarring.

Pathophysiology of BPD

The BPD phenotype is the outcome of a multifaceted process in which numerous prenatal and postnatal variables interfere with normal lung development in the immature lung. The pattern of lung damage that may occur is influenced by the timing and length of exposures [4]. Notably, the prevalence of BPD in mechanically ventilated babies is negatively related to gestational age and birth weight, implying that defective lungs development or injury during a critical window of lung development influence BPD development. Mechanical ventilation, oxygen toxicity, pre- and postnatal infection, inflammation, and growth restriction or nutritional inadequacies are just a few of the conditions that can affect alveolar growth and pulmonary vascular development in addition to prematurity. Genetic predisposition is well understood.

Bronchopulmonary dysplasia prevalence

Over the previous few decades, the incidence of BPD among surviving newborns less than or equal to 28 weeks gestational age has remained reasonably consistent at around 40%. This currently results in 10,000–15,000 new cases per year in the United States alone. While some studies show an increase in the number of newborns surviving with BPD, differences in definitions and approaches to oxygen therapy use both influence and confound interpretation of historical data. Although frequent use of prenatal steroids in cases of imminent preterm delivery and surfactant treatment in cases of respiratory distress syndrome have improved premature infant mortality, these gains do not appear to have translated into lower rates of BPD. The lack of effective medicines to prevent newborn lung damage and chronic disease is impeding progress. Volume-targeted as well as non-invasive ventilation, permissive hypercapnia, and targeted use of steroids, as well as adjunct medicinal therapy such as coffee and vitamin A, are currently being promoted as lung-protective techniques [5].

Trauma caused by mechanical forces

BPD is almost exclusively seen in premature infants who have been given positive pressure ventilation, implying that mechanical lung over-distension and alveolar stretch are important factors in the development of BPD. The necessity for ventilatory assistance at birth is caused by ineffective pulmonary mechanics. Surfactant deficit makes it difficult to ventilate the premature lung, leading in diminished compliance and difficulties maintaining functional residual capacity (FRC). Surfactant insufficiency also causes non-uniform lung expansion, with areas of localised over-distension and atelectasis. Positive pressure and excess volume delivered via assisted ventilation can injure the immature lung by causing further alveolar over-inflation, resulting in cellular

***Address for Correspondence:** Michael A. Matthay, Division of Pulmonary Medicine, Golden Gate University, San Francisco, CA, USA, E-mail: michaelmatthay@yahoo.com*

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injury, inflammation, and the generation of reactive oxygen species (ROS), potentially amplifying preexisting injury associated with prenatal inflammation.

Oxygen toxicity

Numerous animal models show that simply breathing supraphysiologic oxygen causes a phenotype similar to BPD, such as impaired alveolar formation and pulmonary vascular remodelling. Clinical investigations have found that restricting oxygen use or setting lower saturation objectives reduces lung inflammation and the incidence of BPD. Furthermore, whereas endotracheal injection of recombinant superoxide dismutase to premature infants did not lower BPD rates, long-term pulmonary outcomes were improved, indicating that oxidative damage plays an important role. Supraphysiologic oxygen causes an increase in mitochondrial ROS production, as well as a specific vulnerability to oxidative stress and alveolar cell damage in the developing lung, due to antioxidant deficiencies and immature defences.

Conclusion

Since Northway originally described bronchopulmonary dysplasia (BPD) about 50 years ago, the description, pathogenesis, and therapy of BPD have changed dramatically. Advances in neonatal care have resulted in higher survival rates for extremely preterm newborns, posing new treatment issues as well as the emergence of a new cohort of long-term BPD survivors. Interdisciplinary treatment to address these patients' complex respiratory, nutritional, and developmental needs is crucial, and it may influence severe BPD outcomes. Randomized treatment trials, as well as long-term monitoring of these patients, are still needed to improve care and better understand morbidity risk factors. While these trials are costly, they are critical because there is a paucity of data for the best treatment. Furthermore, little is known about BPD's pulmonary consequences after the second decade of life.

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