CE 2.5 HOURS Continuing Education

Bronchopulmonary Dysplasia Within and Beyond the Neonatal Unit

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ABSTRACT

Background: Bronchopulmonary dysplasia (BPD), also known as chronic lung disease of prematurity or chronic neonatal lung disease, is a major cause of respiratory illness in premature babies. Newborn babies survive at gestational ages of 23 to 26 weeks, earlier than when BPD was first described. New mechanisms of lung injury have therefore emerged and the clinical and pathological characteristics of pulmonary involvement have changed.

Purpose: Improved neonatal intensive care unit modalities have increased survival rates; the overall prevalence of the condition, however, has not changed. Management of evolving BPD aims at minimizing lung injury. Management of established, especially severe BPD, still poses significant clinical challenge as these babies need long-term oxygen therapy (LTOT) for variable length of time. We aim to give an overview of management of established BPD with particular focus on weaning home oxygen therapy at our local center in the United Kingdom.

Search and Results: On the basis of most recent evidence, we concluded that an integrated pathway for managing babies on LTOT is very important after discharge from neonatal unit.

Implications for Practice: A structured weaning pathway for premature babies on home oxygen improves outcome.

Implications for Research: The management of severe BPD and related complications, particularly during the first 2 years of life, remains a continuing challenge for parents and healthcare providers. The most beneficial respiratory support strategy to minimize lung injury and/or promote lung healing remains unclear and requires further investigation.

Key Words: bronchopulmonary dysplasia, chronic lung disease of prematurity, chronic neonatal lung disease, long-term oxygen therapy, prematurity

Bronchopulmonary dysplasia (BPD) is an important cause of respiratory illness in preterm newborns that results in significant morbidity and mortality. There has been a striking lack of uniformity in the diagnostic criteria for BPD, which explains variations in reported incidence of BPD in various centers.

DEFINITION

In 1967, Northway first described BPD as a result of high oxygen and aggressive mechanical ventilation in premature infants with respiratory distress syndrome.[1] This was termed "classical" BPD on the basis of oxygen requirement at 28 days. It did not account for extreme prematurity, that is, birth weight less than 1000 g or gestational age less than 30 weeks.[2,3]

The definition of BPD has continued to evolve since first described. Changes in neonatal management (ie, use of surfactant, antenatal glucocorticoid therapy, and less aggressive mechanical ventilation) and increased survival of more premature infants have resulted in a new form of BPD. Despite being delivered several weeks before alveolarization begins, infants at risk for new BPD often have only mild respiratory distress syndrome at birth. The overall prevalence of the condition, however, has not changed, because with the improved survival rate among infants born at earlier gestational ages, a new pattern of lung injury has emerged.[4] National Institute of Child Health and Human Development modified the preexisting definition by adding criteria that included gestational age and severity of disease.[5] This is termed as "new" BPD and it is characterized by disruption of lung development that results in large alveoli and dysregulation of vasculature development. In contrast, the "old" BPD was characterized by airway injury, inflammation, and parenchymal fibrosis, which were primarily due to injury from mechanical ventilation and oxygen. This definition includes infants born at less than 32 weeks of gestation and needing more than 21% oxygen for at least 28 days. Assigning severity requires a second assessment of the infant at 36 weeks of

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postmenstrual age (PMA). They are further classified as follows[5]:

Mild BPD: Supplemental oxygen for at least 28 days but no oxygen requirement by 36 weeks corrected for gestation or discharge (for infants <32 weeks at birth) or at 56 days or discharge (for infants >32 weeks at birth).

Moderate BPD: Supplemental oxygen for at least 28 days and a need for supplemental oxygen less than 30% at 36 weeks PMA/discharge (for <32 weeks) or at 56 days per discharge (for infants >32 weeks).

Severe BPD: Supplemental oxygen for at least 28 days and a need for greater than 30% oxygen or on nasal CPAP or mechanical ventilation at 36 weeks PMA/discharge (<32 weeks) or at 56 days/discharge (>32 weeks).

Epidemiology: Because of differences in local practices, there is a wide variability in rates of BPD between European regions. This may reflect neonatal risk factors, care practices, for example, target levels for acceptable oxygen saturation and differences in the clinical definitions of BPD. The incidence of BPD increases with decreasing gestational age, the overall incidence of BPD defined as requiring supplemental oxygen at 36 weeks PMA was 42%, the incidence using the physiologic definition was 40%.[6]

In a large US nationwide epidemiological study, the absolute incidence of diagnosis of BPD was 3.3% annually between 1993 and 2006.[7] The rate when the data were collected for demographic factors was 4.3%.[7] The strongest association of BPD is with degree of immaturity.[8] In the Epicure cohort, 308 babies born at less than 25 weeks of gestation in 1995 were followed up at 30 months and 6 years of age. Of this population, 74% received supplemental oxygen at 36 weeks PMA and 36% were discharged with supplemental oxygen, which continued for a median of 2.5 months.[8]

NORMAL LUNG DEVELOPMENT

It is pertinent to briefly review normal lung development as insults to the lung at the critical stages of development play a major role in the development of BPD. The prenatal lung development occurs in stages[9] and it enables the lung to be able to work immediately after birth. During the 7th to 16th weeks of gestation (embryonic and pseudoglandular stage), the lung appears as an outpouching of the primitive foregut on day 28. This bud bifurcates into the two main stem bronchi. Fifteen to 20 generations of airway branching occur from main segmental bronchi and ending in terminal bronchioles. They are lined by columnar epithelium surrounded by mesenchymal tissue. During 16th to 25th weeks of gestation (canalicular stage), there is further bifurcation of the last generations of distal bronchi as respiratory bronchioles and alveolar ducts. In this stage there is also capillary invasion and differentiation of the air space epithelium into alveolar type II cells and type I cells. At 24 weeks (saccular stage), the peripheral air spaces enlarge at the expense of the intervening mesenchyme, forming saccules. This is followed by the final alveolar stage when alveoli are generated via subdivision (septation) of the distal lung saccules. This process of alveologenesis begins at 36 weeks of gestation and extends into the first few years of life.

PATHOGENESIS AND RISK FACTORS

Our understanding of the pathways by which BPD results remains incomplete. The picture is further complicated by a change in the characteristics of the disease process with an increase in the survival of smaller, more extremely premature babies. The evolution of BPD is multifactorial with contributory factors, in addition to prematurity, likely to include ventilation (barotrauma and/or volutrauma) and oxygen, infection, and the presence of patent ductus arteriosus and possible vascular changes. Many studies have identified more than 50 biomarkers of disease that may be used to further advance our understanding of how BPD develops. In a review of these studies, Bhandari and Bhandari[10] describe correlation between changes in the level of biomarkers and inflammation, infection and response, fibrosis, angiogenesis, and many other disease states. However, the inconsistent presentation of babies who do and do not develop BPD suggest other factors, including a genetic predisposition, are involved.

Until recently, the conventional use of rodent models of BPD has only been able to investigate the relative contributions of inflammation, infection, ventilation, and oxygen toxicity. The development of transgenic models (mice that are bred to possess specific gene mutations) has enabled the investigation of the relative contributions of specific gene mutations.[11] Such mechanistic approaches have been used not only to examine the effects of individual genes, but also to investigate the interplay between environmental (hyperoxia, infection) and genetic factors suggesting a "multiple hit" hypothesis.[12]

In terms of susceptibility to BPD, a large genome-wide study examining more than 600 neonates, backed up by animal work, has implicated the *SPOCK2* gene in the susceptibility to BPD.[13] Other genes implicated in the pathogenesis of BPD have been identified through studies of smaller series of babies with the condition. These studies improve our understanding of the molecular pathogenesis of BPD. Examination of the genome of cells isolated from lung tissue of babies with severe BPD suggests that an accumulation of connective tissue mast cells

is involved.[14] If mast cells participate in BPD pathogenesis and symptomatology, inhibition of mast cells secretion might be expected to be therapeutic in the disease. Methods of targeting mast cells, and mast cell-derived mediators, for intervention of BPD may be warranted. These observations have significant clinical and mechanistic implications.

NATURAL HISTORY AND OUTCOMES OF BPD

The "new BPD" is believed to be more due to disruption or arrest in development of lungs and less due to the effects of severe lung injury as discussed in the earlier sections. It was initially thought that human pulmonary alveolarization is complete by 3 years. However, recent evidence suggests that lungs grow partly by neoalveolarization throughout childhood and adolescence.[15,16] This has important implications: developing lungs have the potential to recover from early life insults and respond to emerging alveolar therapies.[16]

Mortality

Deaths from pulmonary causes in extremely premature infants have decreased from 2000 through 2011.[17] Death usually is caused by respiratory failure, unremitting pulmonary hypertension with cor pulmonale,[18] or sepsis. Although the exact prevalence of PAH in infants with CLD is unknown, infants with CLD and severe PAH have a high mortality rate.[19-21]

Morbidity

Infants have higher rates of rehospitalization (up to 50%) in the first year of life. Respiratory symptoms in the patients with BPD persist beyond the first 2 years into preschool years, adolescence, and early adulthood.[22-24] It is unclear whether BPD severity or prematurity per se influences the persistence and severity of symptoms.[25]

Respiratory Infection

Infants with BPD are at increased risk for respiratory tract infections,[25-28] including respiratory syncytial virus (RSV), which may be life-threatening.[27] There is increased respiratory morbidity in children with BPD who attend day care.[28,29]

Asthma-Like Disease

Episodes of wheezing suggesting bronchiolitis or asthma are common in preterm survivors with BPD before 2 years of age. Recurrent wheezing episodes are common in children and adolescents with a history of BPD,[22-24,30,31] but the underlying pathophysiology differs from asthma. Persistent abnormalities in pulmonary function are common in patients who had BPD as infants and depend upon the severity of BPD.[31,32]

Pulmonary Hypertension

Pulmonary hypertension and the resultant right-sided heart failure (cor pulmonale) associated with severe BPD are caused by both structural and functional changes within the lung. It is one of the most severe complications of BPD and is an important risk factor for mortality as discussed earlier.

Neurodevelopmental Outcome

Preterm survivors who had BPD compared with those without BPD are at increased risk for neurodevelopmental impairment. The severity of BPD increases the risk of neurodevelopmental impairment.[33] The effect of BPD on neurodevelopmental outcome persists through school age.[34,35] It is important to recognize that many confounding factors influence the neurodevelopmental outcome.

Growth

It is clear that poor growth is observed in infants with BPD during their neonatal intensive care unit hospitalization and after discharge. This is due to the increased energy expenditure associated with respiratory disease, and difficulty of maintaining full nutrient and mineral intake in patients requiring severe fluid restriction and diuretics. It remains uncertain whether BPD has a direct impact on long-term growth.[33] After adjusting for confounding factors, no significant differences were detected in growth at school age in children who had BPD compared with those who did not have BPD.[36,37]

Prevention and Management of Evolving/Established BPD

The combination of improved antenatal and perinatal care, fluid restriction,[38,39] use of diuretics[40] and caffeine improve outcome and/or reduce the risk of BPD in premature infants.

Nutrition is the mainstay of encouraging healing and lung growth. As fluid intake is often restricted, the desired nutrients may need to be contained in a reduced volume of feed. Additional calories and/or protein may be added as needed.[41]

Corticosteroids

Corticosteroids have been extensively studied in management of BPD for its anti-inflammatory effects. Despite a series of sequentially refined Cochrane reviews covering the immediate postnatal (up to 7 days of life), moderately early (7-14 days) and late (after 7 days) periods,[42-44] the corticosteroid treatment of chronic lung disease remains controversial. While it is largely accepted that corticosteroids are effective in reducing the pulmonary effects of chronic lung disease, the concern that this may be accompanied by adverse neurodevelopmental sequelae has limited their use. Corticosteroid

administration through all periods are associated with reductions in the risk of failure to extubate, death, or chronic lung disease at 28 days' or 36 weeks' corrected gestational age with a cost of increased risk of hyperglycemia and hypertension.[42,43] Findings related to the early administration versus late administration of corticosteroids have implications for this population. The late-administered review reported a reduction in risk of clinically important impairments of forced expired volume in 1 second (FEV_1) of children undertaking spirometry later in childhood.[43] The early administered review, however, reported a greater burden of adverse effects, including gastrointestinal bleeding and perforation, hyperglycemia, hypertension, hypertrophic cardiomyopathy, and growth failure.[42] It also documented increased risks of developmental delay (one study), cerebral palsy, and abnormal neurological examination in the limited number of included trials that reported late outcomes.[44] However, the reporting of these adverse neurological outcomes is largely dependent upon the results of one trial.[45] The heterogeneity of corticosteroids involved in the trials involved in the meta-analysis presents difficulty with the interpretation of these Cochrane reviews. Included in the meta-analysis were studies that involved dexamethasone and hydrocortisone, with regimens of varying doses and differing usage of late rescue steroids. Commentators have recommended against the use of early corticosteroids because of the higher risk of adverse events and reserving the use of late corticosteroids to those babies who cannot be weaned from ventilation.[43] As both early and late dexamethasone use has raised concerns about neurodevelopmental outcomes, other safe and efficacious alternative corticosteroid preparation need to be evaluated as the issue of postnatal steroid use in preterm infants for management of BPD remains an unresolved dilemma for the clinician. Oral betamethasone for BPD was shown to allow more extubations and reduction of oxygen requirement.[46]

There is very little available evidence for management of established very severe BPD. In the absence of existing consensus and sufficient evidence from clinical trials, a Delphi methodology involving 144 clinicians was used to establish a consensus for the use of corticosteroids in diffuse lung disease of childhood.[47] Consensus was reached for the use of corticosteroids involving methylprednisolone pulses of 3 days at 500 mg/m^2 every 4 weeks in very sick child who is ventilator dependent or close to ventilation. Anecdotally, this regimen had led to survival and freedom from ventilator dependency in a small number of very severe BPD cases.

Supplemental Oxygen

Long-term oxygen therapy (LTOT) is described as the provision of oxygen therapy for continuous use at home for patients with chronic hypoxia (due to any cause) to maintain oxygen saturations greater than 92%.[48,49] Advanced neonatal nursing has resulted in a greater proportion of infants with neonatal chronic lung disease being discharged home on LTOT.[50]

In the United Kingdom, oxygen is available on prescription as part of an integral service delivered directly to patients in their homes. In a study done to determine the incidence and patterns of home oxygen prescribing in England and Wales, once data collection was done, 68% of the total children who were receiving home oxygen had a diagnosis of chronic neonatal lung disease.[51]

Oxygen equipment and servicing are provided and supplied by the oxygen provider. Once home oxygen order form completed by a healthcare professional is submitted to the oxygen provider, the equipment is installed at home within 24 hours. In an emergency situation, installation can be reduced to within 4 hours.[52] Figure 1 outlines this referral process.

Once it is anticipated that the infant will need oxygen beyond 36 weeks' PMA, that is, has BPD, a referral is made to the respiratory pediatrician who assesses the infant's suitability for LTOT in the community. Once it is confirmed by clinical assessment and overnight oxygen saturation study ("sleep study") that the infant requires continuous oxygen, a correct oxygen flow rate is determined. Continuous monitoring is discontinued prior to discharge, as the family will not have access to monitoring equipment for their own use at home. The child therefore needs to be safe in a stable amount of oxygen. In infants with BPD, oxygen saturation levels of less than 93% should be avoided. A target range of 94% to 95% in a consistent flow rate of supplementary oxygen when the child is awake, during feeding and while asleep should be aimed for.[49] If the child continues to require continuous monitoring due to excessive desaturations, then they are not safe for discharge.[49] Once the infant has not had apnea for at least 2 weeks, a multiagency discharge-planning meeting is held.[53] The meeting ensures that all equipment and services are in place and parents are trained to identify and respond appropriately when their baby is unwell and are able to deliver basic life support. The risks associated with secondary smoke exposure are highlighted. It ensures that the arrangements are made for immunizations, including influenza (if >6 months chronological age) and passive immunization for RSV.[54] Through multiprofessional planning, it is aimed to promote a seamless transition from hospital to home for the infant and their family. This pathway of care[55] has led to a greater number of infants being discharged in oxygen, shorter duration of LTOT, and trend toward fewer hospital readmissions and reduced length of stay on readmission.

FIGURE 1

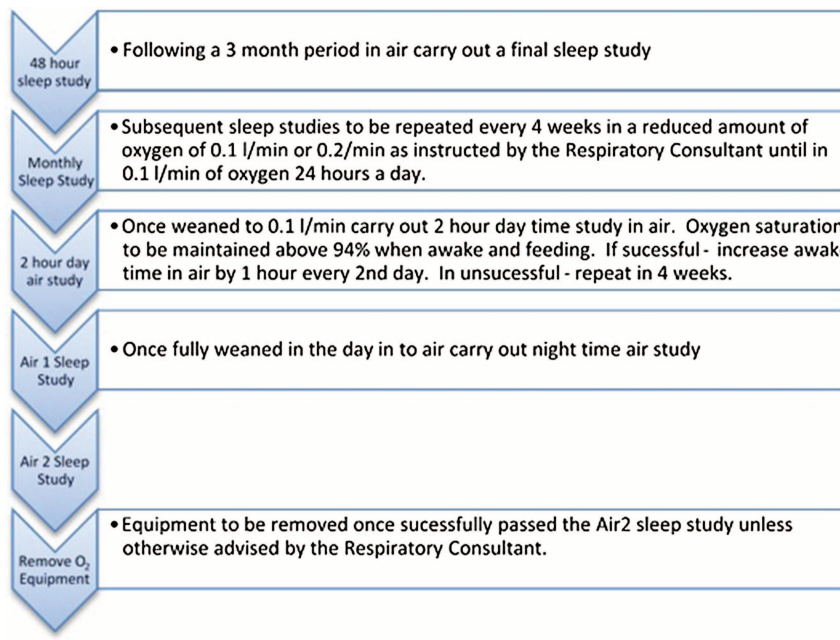
Nottingham chronic neonatal lung disease care pathway.

Figure 2 outlines the care pathway for weaning oxygen in BPD infants in Nottinghamshire.

Sleep studies are carried out using saturation monitors with data storage facilities to record oxygen saturation level and heart rate overnight. The data are downloaded on to a computer and analyzed. The results from the sleep studies can be used to successfully wean the infant off supplementary oxygen without putting the onus on parents. However, it is noted that those who receive home oxygen treatment show high rates of utilization of health service resources after discharge from the neonatal care unit.[50]

A repeat sleep study is carried out at home within 48 hours postdischarge. This is to ensure that the home oxygen supply is working effectively in administering an optimal amount of oxygen. Careful follow-up sleep studies should then take place in the family home with the support of the community nurse to minimize the chance of nosocomial infection.[49] This practice ensures that the child continues

FIGURE 2



Nottingham care pathway for weaning home oxygen.

to receive the optimal amount of oxygen and that it is not discontinued too soon. Reassessment through sleep studies is carried out every 4 weeks if the infant is stable at night. The oxygen is reduced by 0.1 l/min at every study if the target saturations are met (mean saturation >93% and <5% of total time less than 90%). Once stable in 0.1 l/min day and night, a 2-hour oximetry study is carried out in at home during the day. If this is successful with no desaturations lower than 94%, including periods of feeding, the infant can start increasing the time spent in air until fully weaned in to air for all waking time. See Figure 3 for daytime weaning regimen in Nottinghamshire.

Once weaned in the day, a repeat study in air is carried out at night aiming for the same target range as in the day. Once successfully weaned in room air, a repeat study is carried out 3 months later to confirm ongoing normal saturations, especially if the infant has suffered from an intercurrent respiratory illness and may have briefly required supplemental oxygen again. After the second air study, the oxygen equipment will be removed from the family home especially if this happens over the summer months.

This practice takes away a degree of responsibility and control from parents, which inevitably places a lot of stress on them; the risk of harm is reduced as weaning becomes medically determined. This ensures that oxygen saturation levels remain in the safe range and removes the possibility of excessive adjustment of the flow rate by parents.[54]

When weaning home oxygen therapy, the following factors should be borne in mind as presence of any of these is likely to increase the duration of home oxygen therapy:

- Associated medical problems (pulmonary hypertension): If present, will require that the target saturations are maintained 95% or more (as discussed previously).
- Poor growth: Suboptimal saturation has been associated with poor growth and weight gain normalizes with low flow oxygen and average weight gain falters on discontinuation.[56] Considerable catch-up growth occurs when saturation is maintained at 95% or more,[57] and infants have better growth velocity[58] and can achieve comparable growth patterns to those of birthweight-matched preterm infants without BPD.[59]
- Weather: Infants are more likely to encounter respiratory viruses during the winter months and may require increased oxygen from the stable basal rate.
- Restricted versus liberal oxygen exposure: A policy of unrestricted, unmonitored oxygen therapy has potential harms, without clear benefits. However, the question of what is the optimal target ranges for maintaining blood oxygen levels in preterm/LBW infants was not answered by the data available for inclusion in a systematic review.[60]
- Whether oxygen is weaned early or late after discharge, home is not as important as weaning while maintaining target oxygen saturation levels.[61]
- Gradual weaning does seem preferable to abrupt weaning, independent of the duration of oxygen therapy.[62]

FIGURE 3

Day	Morning	Afternoon
1	1hour	0hours
2	1hour	1hour
3	1hour	1hour
4	2hours	1hours
5	2hours	1hours
6	2hours	2hours
7	2hours	2hours

Nottingham daytime oxygen weaning regimen.

The specialist nurse performs regular sleep studies on premature infants discharged home on oxygen. This allows not only carrying out the study but also additional visit at home by a healthcare professional. This is an opportunity for holistic assessment of home environment, prompting preventive care and promoting parental education. Feeding is an important aspect of home care and the team ensures that infants gain weight appropriately through the use of calorie-condensed formula or breast milk fortifiers. Feeding could be particularly difficult if infants have gastroesophageal reflux and/or oral aversion related to their stay in the neonatal intensive care unit.

When parents have concerns about their child's respiratory effort, oxygen requirement, or feeding regimen, it is recommended that an assessment by the children's community nurse be carried out. Care will aim to manage at home with the support of continuing care nurse. Backup from the general practitioner (primary care physician) and respiratory consultant is provided for unwell babies.

Healthcare professionals need to recognize that caring for an infant on LTOT also brings new strains to parents in both economic and human terms. An infant on home oxygen brings an element of social isolation due to the risk of exposure to respiratory tract infections to the already-vulnerable infant. The need for respite care to reduce the feeling of isolation is common among these families,[52] especially if the infant has complex health needs in addition to the supplementary oxygen due to their prematurity. Parents need to qualify for these specialist services and rely on their key professional to submit a health

needs assessment to the local commissioning board to have somebody who will care for their infant in their home. Often parents rely on family members for their respite care if unsuccessful in qualifying for specialist services. For the parents who had not budgeted for any extended time off work caring for an infant with BPD can cause financial anxiety. Government benefits are available such as Disability Living Allowance and carers allowance, but the processing of these applications can be lengthy, leaving families struggling in the initial months at home.

The issue of whether parents should be provided with their own saturation monitor has been an area of debate. There is no evidence on whether the routine use of a saturation monitor at home is of benefit or harm, and it cannot be recommended. Nevertheless, some clinicians and parents may find it helpful in certain circumstances.[48] Table 1 highlights pros and cons in relation to this issue.

Follow-up After Discharge

After discharge the neonatal continuing care team follows until there is sustained growth and development in the baby, family is settled in home environment, and neonatal needs are met. Community pediatric team and health visitor are integrated in the care in a planned manner. A pediatric respiratory consultant follows infants with BPD until they no longer require oxygen and have had 2 winters without very severe or frequent or persistent respiratory symptoms. Babies who meet the Joint Committee on Vaccination and Immunization criteria[63] receive monthly intramuscular injections of palivizumab[63] (15 mg/kg) during RSV season (October to February).

Further Research

Recent insights into stem cell biology have allowed a better understanding of the role of resident and exogenous stem/progenitor cells in lung health and disease with the ultimate hope of offering new therapeutic options for life-threatening and debilitating lung diseases. On the basis of the promising studies in various animal models, stem cells may provide an opportunity of providing a therapy that may promote lung repair.[64]

An ongoing study assesses the safety and efficacy of inhaled nitric oxide to reduce the risk of chronic lung disease in preterm infants with respiratory distress and to assess the long-term effects of the therapy on the development of these children over 7 years of clinical follow-up.[65]

Macrolide antibiotics (especially azithromycin) have been demonstrated to exert anti-inflammatory and immunomodulatory activity in addition to being antimicrobial and the potential benefit of macrolide antibiotics has been evaluated in a variety of chronic respiratory diseases.[66] Infection/inflammation plays a major role in the pathogenesis of BPD and a recent meta-analysis concluded that azithromycin, when given prophylactically, is associated with reduction in BPD and BPD/death in preterm infants.[67] The authors, however, caution that high-quality pharmacokinetic studies are needed before routine use of azithromycin the neonatal intensive care. A randomized, placebo-controlled trial of azithromycin for the prevention of chronic lung disease of prematurity in preterm infants is planned.[68] The aim of the TINN2 study is to evaluate the efficacy of azithromycin in prevention of BPD in preterm neonates. The role of azithromycin in established very severe BPD also needs to be determined.

Natural human Clara cell protein (CC10) regulates inflammatory responses and protects the structural integrity of pulmonary tissue while preserving pulmonary mechanical function during various insults, for example, viral infection, bacterial endotoxin, ozone, allergens, hyperoxia. Administration of CC10 may help facilitate development of normal airway epithelia and prevent the inflammation that leads to chronic respiratory morbidity in these infants. This study aims to evaluate the pharmacokinetics, safety, tolerability, and anti-inflammatory effects of a single intratracheal dose of recombinant rhCC10 to intubated premature infants.[69]

TABLE 1. Pros and Cons of Home Saturation Monitors

Pros	Cons
Reduces anxiety	Increase anxiety
Detects O_2 disconnection or cannula displacement	Undue reliance on monitor with difficulty for parents weaning off it
May provide early indication of worsening respiratory status, eg, with intercurrent infection	Unnecessary minor adjustments of flow rates
May provide warning of sudden severe hypoxemia	False reassurance of respiratory status
Can assess response to URTI in the home after O_2 therapy has been stopped	False alarms (less with newer motion resistant technology)
Cost	Cost

Abbreviation: URTI, upper respiratory tract infection.

Summary of Recommendations	
What we know:	• Management of evolving BPD requires optimal nutrition, judicious fluid management, effective and safe pharmacotherapy, and respiratory support aiming at minimal lung injury
What needs to be studied:	• Better understanding of pathophysiology
What we can do today:	• Structured weaning pathway for babies on home oxygen • Weaning of home oxygen based on physiological targets • Nursing support invaluable • Role of pulsed steroids and anti-inflammatory/antibiotics in established BPD

SUMMARY

Bronchopulmonary dysplasia is associated with increased mortality and significant long-term cardiorespiratory and neurodevelopmental sequelae. Management of evolving BPD requires optimal nutrition, judicious fluid management, effective and safe pharmacotherapy, and respiratory support aiming at minimal lung injury. Systemic postnatal corticosteroids should be reserved to ventilated infants at highest risk of BPD who cannot be weaned from the ventilator. There may be a role of systemic corticosteroids and/or other anti-inflammatory strategies like Azithromycin in infants with established very severe BPD. This review focused on respiratory pediatric and community nursing team management of long-term home oxygen therapy in stable premature babies with BPD. The most beneficial respiratory support strategy to minimize lung injury remains unclear and requires further investigation.

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