The incidence of bronchopulmonary dysplasia (BPD), defined as oxygen need at 36 weeks of postmenstrual age, is about 30% for infants with birth weights <1000 g. BPD is associated with persistent structural changes in the lung that result in significant effects on lung mechanics, gas exchange, and pulmonary vasculature. Up to 50% of infants with BPD require readmission to the hospital for lower respiratory tract illness in the first year of life. Long-term measurements of lung function in BPD include normalization of pulmonary mechanics and some lung volumes over time as somatic and lung growth occur, whereas abnormality of small airway function persists. The majority of data reveals no long-term decrease in exercise capacity. Mild to moderate radiological abnormalities persist. BPD is a result of dynamic processes involving inflammation, injury, repair, and maturation. Infants with BPD have significant pulmonary sequelae during childhood and adolescence, and continued surveillance of young adults with BPD is critical.

Pulmonary Pathology

Bronchopulmonary dysplasia (BPD) was first described by Northway in 1967 in premature infants with respiratory distress syndrome (RDS) who developed chronic lung disease after being treated with intermittent positive pressure ventilation and oxygen supplementation.1 With the widespread use of antenatal steroids, surfactant replacement therapy and gentler methods of mechanical ventilation, this “classic” form of BPD has been replaced by less severe forms which are primarily observed in very small premature infants.2–5 Although the histopathology of “classic” and “new” BPD differ from one another as discussed below, each results in lasting changes in lung architecture with potential functional ramifications throughout life.

The exact incidence of BPD is difficult to assess since there is no universally accepted definition.6 When the definition is confined to the need for supplemental oxygen at 36 weeks postconceptional age, the incidence of BPD is about 30% of all infants with birth weights <1000 g.7 More recent data8 reveal incidence of BPD to be 52% in infants with birth weights between 501 and 750 g. 34% in infants with birth weights 751 to 1000 g, 15% in infants with birth weights 1001 to 1250 g, and 7% in infants with birth weights of 1251 to 1500 g. Although BPD is now thought to be infrequent in infants with birth weights >1200 g and those >30 weeks gestation,7 it is still the most common cause of chronic lung disease in infants.9,10

Pathology of the BPD lung from the presurfactant era was remarkable for the presence of central and peripheral airway injury, airway inflammation, and parenchymal fibrosis. Rosan described four pathological stages of BPD.11 This most commonly represented the effects of oxygen toxicity and barotrauma from positive pressure ventilation on lungs at the saccular phase of development (28–34 weeks gestation) recovering from RDS.

Pathological findings in the lungs of infants with “new” BPD are strikingly different from those found in “classic” BPD. Histological changes represent injury to the lung at an earlier (canalicular, 16–28 weeks gestation) phase of development. Thus, the hallmark of the pathological changes in the lung in new BPD is an arrest of alveolar development.5,12 Both small and large airways demonstrate considerably less epithelial metaplasia, smooth muscle hypertrophy, fibrosis,
and more uniform inflation as compared with lungs of infants with “classic” BPD. Animal models of experimental BPD demonstrate not only a permanent reduction in the number of alveoli, but also enlargement of the airspaces, resulting in a decreased total internal gas exchange surface area. Among infants dying with BPD, a decrease in the arterial count has also been reported, so that the alveolar/arterial ratio remains normal.14

It is unclear whether abnormalities in angiogenesis result in reduced alveolarization, or if the reduction in alveolar number is the cause for altered angiogenesis. The structural changes described in infants who died with BPD13-17 or who have undergone lung biopsies18,19 represent the most severe end of the spectrum. Nevertheless, animal models that examine the histopathological changes in milder forms of BPD13 suggest that the alterations described persist throughout life. No structure–function comparisons are available to assess how the severity of pathological changes influences the course of subjects with BPD. Nevertheless, it is a recurrent finding that those children whose neonatal course was more severe are also more likely to experience greater respiratory sequelae and demonstrate greater abnormalities on tests of respiratory function as they age.

Respiratory Morbidity

It is difficult to find uniform agreement from published studies about the clinical outcomes of children with BPD because different definitions of BPD are used, reported patient populations and their comparison groups are disparate, and there is patient attrition from original cohorts in most studies (Table 1). Many studies do not distinguish those children who developed BPD from others who were very low birth weight (VLBW) but who did not develop chronic lung disease in infancy. Only one study to date has examined the outcomes of children with respect to the use of surfactant in neonatal practice.20

Rehospitalization for respiratory illness is common among infants with BPD younger than 2 years of age.21-24 Hospitalization rates for these BPD infants were not only higher than for the general population, but were also shown to be higher when compared with rates for VLBW infants without lung disease.21,22 Furman and coworkers reviewed the courses of 98 of the 124 VLBW infants from their nursery with BPD who survived to 2 years of age. Fifty percent were re-hospitalized in the first year of life and 37% were hospitalized during the second year of life. The most common causes for re-hospitalization in this population were respiratory: reactive airway disease, pneumonia, and respiratory syncytial virus infection were responsible for 65% of re-hospitalizations in the first year and 81% in the second. Those infants with more severe disease also experienced more sequelae: severity of BPD was significantly associated with the duration of neonatal hospital stay and the total hospital stay over the 2-year period. The duration of re-hospitalization was associated with total duration of oxygen dependence.

After 4 to 5 years of life, hospitalizations for respiratory problems decrease.23-26 Chronic respiratory symptoms, however, continue to be reported in greater frequency among children with a history of BPD than those born at term without lung disease in most studies. At a mean age of 5.4 years, a greater proportion of BPD children had a diagnosis of asthma compared with the general population.27 Although no children required hospitalization after 2 years of age, 8 of 10 of the children with a history of BPD studied by Bader and coworkers had persistent respiratory symptoms at 10.4 ± 0.6 years.28 Kitchen and coworkers compared a cohort of VLBW children, some of whom also had BPD, to age-matched controls born at term.24 Wheezing occurred more frequently in the groups of children who were <1501 g at birth and the age of 2 compared with the term group. At 5 and 8 years, however, although those groups continued to have frequent respiratory symptoms, the frequency of wheezing was not statistically different from the group born at term. The same cohort of children was again studied at 14 years of age.29 The frequency of respiratory symptoms was not different between those children with a history of BPD and those who were VLBW without BPD: 19% of children with BPD had asthma as compared with 18% without BPD. The group of children born at term had an unusually high prevalence of asthma at age 14 (21%), so that no differences existed between any of the groups.

Palta and coworkers20 reviewed the outcomes of VLBW children at 8 years of age, and divided them into three groups based on the practice of surfactant administration at the time of their births: group 1 infants were born when surfactant was used only sporadically in randomized trials; during the group 2 epoch, surfactant became an investigational new drug; and in the group 3 period, surfactant was generally available. Both VLBW infants with and without BPD had a greater frequency of “asthma ever” and wheezing in the previous 12 months compared with control populations born at term. A history of BPD heavily predicted “asthma ever.” There was, however, a significant decrease in the prevalence of wheezing in the past 12 months for children with a history of BPD across time periods. In contrast, those VLBW children without BPD who had milder pulmonary disease during their neonatal course demonstrated an increase in the prevalence of wheezing in the prior 12 months across the time periods. The authors speculated that one possible explanation for these contradictory findings is that those infants diagnosed with BPD were more ill as neonates and therefore received more intensive therapies (ie, doses of surfactant, postnatal steroids) than those with milder disease.

Northway and coworkers followed 26 survivors of BPD (mean age 18.3 years) and reported that 25% of adolescents with BPD had significant pulmonary symptoms (higher number of wheezing episodes, pneumonias, and use of long-term medications), compared with age-matched controls born prematurely with no need for mechanical ventilation and a group born full term.30 A recent population-based study showed that adolescents who were VLBW, with or without BPD, had more respiratory symptoms than age-matched controls who were born at term.31 In contrast, de Kleine and coworkers found no differences in respiratory symptoms in children aged 8 to 18 years who had BPD compared with
age-matched normal controls, children with a history of RDS but no BPD, and those who were preterm infants with no lung disease, even though the BPD group had lower lung function. Finally, a recent study from a Danish collaborative group reported that of 508 19-year-old adults born prematurely, those who had BPD had a higher prevalence of respiratory symptoms, and females were more likely to be affected than males.

In summary, it appears that respiratory symptoms necessitating hospitalization are common in infants and toddlers with a history of BPD. Recurrent cough and wheezing improve over time, but tend to occur more commonly in this

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Study Population</th>
<th>Birth year</th>
<th>Patient Age</th>
<th>Findings</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bader et al</td>
<td>1987</td>
<td>10 children with BPD; 8 FT controls</td>
<td>1973-1979</td>
<td>10.4 ± 0.6 years</td>
<td>No BPD subject hospitalized after 2 years of age, 80% with persistent respiratory symptoms</td>
<td>No mention of gestational ages. No comparison with respiratory course of controls.</td>
</tr>
<tr>
<td>Northway et al</td>
<td>1990</td>
<td>26 PT with BPD, 26 PT with no BPD, 53 FT</td>
<td>1964-1973</td>
<td>18.3 years</td>
<td>6/26 PT with BPD had more current respiratory symptoms including wheezing, pneumonia, limitation of exercise</td>
<td>Patients studied 33-34 weeker preemies. No comment on rate of hospitalization</td>
</tr>
<tr>
<td>Kitchen et al</td>
<td>1992</td>
<td>85 PT 500-999 grams (51.4% with BPD), 124 PT 1000-1500 grams (6.3% with BPD), 60 FT &gt; 2499 grams. No surfactant or postnatal steroids</td>
<td>1977 to 1982</td>
<td>2, 5 and 8 years</td>
<td>More hospitalizations in Groups 1 and 2 vs Group 3; More frequent wheezing in Groups 1 and 2 vs Group 3; not statistically significant at 5 and 8 years</td>
<td>Did not separate those with BPD from those without</td>
</tr>
<tr>
<td>Furman et al</td>
<td>1996</td>
<td>98 children with BPD</td>
<td>1988 to 1990</td>
<td>2 years</td>
<td>50% hospitalized in 1st year; 37% hospitalized in 2nd year</td>
<td>Reactive airway disease and pneumonia most common causes of hospitalization</td>
</tr>
<tr>
<td>Gross et al</td>
<td>1998</td>
<td>96 children 24-31 wk gestation (43 with BPD); FT controls; No surfactant</td>
<td>1985 to 1986</td>
<td>6 months, 15 months, 2, 4 and 7 years</td>
<td>53% of those PT with BPD vs 26% without required rehospitalization during 1st 2 years, vs 3% FT. At age 7, both PT groups more likely had cough or wheeze than FT, at rates similar to each other</td>
<td>Respiratory morbidity at 7 years determined by history of respiratory symptoms/illnesses in the previous year</td>
</tr>
<tr>
<td>Ng et al</td>
<td>2000</td>
<td>55 PT infants with BPD, 28 treated with surfactant</td>
<td>1987 to 1995</td>
<td>5.4 ± 2.3 years</td>
<td>24 (44%) with current asthma; 30 (55%) with past asthma defined by questionnaire, examination, or positive challenge test</td>
<td>Only 34% of eligible children enrolled</td>
</tr>
<tr>
<td>Palta et al</td>
<td>2001</td>
<td>384 PT infants over 3 time periods: 33% with infrequent surfactant use; 36% with surfactant use as investigational new drug; 31% with surfactant widely available</td>
<td>8/1/88 to 7/31/89; 8/1/89 to 7/31/90; 6/30/91</td>
<td>8 years; subsequent control population</td>
<td>Higher prevalence of respiratory symptoms among VLBW children at 8 years. Significant trend of decreasing symptom prevalence across the 3 time periods for children with BPD.</td>
<td>33% of subjects had BPD. Questionnaire used for respiratory symptoms</td>
</tr>
<tr>
<td>Doyle et al</td>
<td>2001</td>
<td>86 PT 500-999 grams, 124 PT 1000-1500 grams, 60 FT &gt; 2499 grams. No surfactant or postnatal steroids</td>
<td>1977 to 1982.</td>
<td>14 years</td>
<td>PT with BPD had similar rates of asthma compared with PT without BPD (19 vs 18%), and similar to FT (21%).</td>
<td>Respiratory health determined by history (presence of cough, wheeze, use of asthma medications, hospitalization). Same study group as in24</td>
</tr>
<tr>
<td>Halvorsen et al</td>
<td>2004</td>
<td>46 PT ≤ 28 wks or birth weight ≤ 1000 grams: 24 with mild BPD, 12 with moderate-severe BPD, 35 FT controls</td>
<td>1982 to 1985</td>
<td>17.7 ± 1.2 years</td>
<td>Previous pneumonia, respiratory hospitalization, current doctor’s diagnosis of asthma greater in PT than controls. Current respiratory symptoms greater in PT than controls (p = 0.065)</td>
<td>Population-based retrospective paired controlled cohorts. Respiratory status by questionnaires and interview. No distinction between BPD and PT for clinical outcomes</td>
</tr>
<tr>
<td>Vrijlandt et al</td>
<td>2005</td>
<td>508 PT ≤ 32 wks (55 with BPD); 182 32-41 weeks (3 with BPD)</td>
<td>19 years</td>
<td>19 years</td>
<td>Prevalence of exercise-related shortness of breath, wheezing and physician-diagnosed asthma greater in PT and in females with BPD than general population</td>
<td>Questionnaire used to assess prevalence of respiratory symptoms and asthma</td>
</tr>
</tbody>
</table>

BPD, bronchopulmonary dysplasia; FT, full term; PT, preterms; VLBW, very low birth weight.
population than in those children born at term. Children born prematurely without BPD also are more likely to have recurrent respiratory symptoms. It is difficult to ascertain, based on study designs, whether or not a history of BPD influences the frequency and severity of respiratory symptoms over time more than prematurity alone.

**Pulmonary Function**

Measurement of lung mechanics, flows, and volumes from birth through adulthood in subjects born prematurely with and without BPD can lend insight into the normal growth process of the lung, and also help to distinguish the effects of early injury on subsequent airway and parenchymal repair from the effects of prematurity alone. Furthermore, longitudinal studies can help determine if and when in the repair process normalization of function occurs. Comparison of lung function measurements between those born prematurely without lung disease, those who developed RDS but no BPD, and those with BPD can also address whether or not severity of lung disease in the neonatal period correlates with abnormalities of lung function later in life.

Airway function in infants and toddlers has been measured over the tidal range by esophageal manometry (lung resistance) or the single-breath occlusion technique (respiratory system resistance and time constant). Forced expiratory flows have also been measured, both over the tidal range of breathing by the rapid thoracic compression (RTC) technique, and over the vital capacity by either the raised volume (RV), and RV/TLC are elevated by 1 year of age.41

**Plethysmographic techniques**.43 Plethysmographic measurements in young infants with BPD when compared with plethysmographic techniques.43 Plethysmographic measurements of lung volumes in BPD infants demonstrate that, although total lung capacity (TLC) is normal, FRC, residual volume (RV), and RV/TLC are elevated by 1 year of age.41 These findings suggest that significant obstructive airway disease with air trapping occurs within the first year of life in BPD infants.

There are over 20 studies evaluating the effects of prematurity and neonatal lung disease on lung function in school-aged children.25,24,28,44-64 As with studies of clinical outcomes, it is difficult to compare them because control groups differed between studies, neonatal care was not standardized between centers, definitions of BPD varied between studies, neonatal care was not standardized between centers, definitions of BPD varied between studies, and often the studies involved small numbers of patients. Nevertheless, certain broad themes become clear:65 school-aged children with a history of BPD have lower forced expiratory volume in the first second (FEV1) than children born at term or those born prematurely without lung disease. They also have a lower forced vital capacity (FVC) and FEV1/FVC ratio than children born at term, although in most studies, those born prematurely with or without a history of RDS do not differ significantly from the full term group in those measurements. There is no difference in TLC or FRC at school age between children born prematurely or without BPD or history of RDS compared with those born at term, but the RV/TLC ratio, reflecting persistent air trapping, remains significantly higher in BPD children studied at school age.

As in toddlers, several studies have attempted to predict subsequent lung function at school age from neonatal data. In a follow-up of the study that demonstrated a relationship between respiratory system compliance in the first month of
life and small airway function at 24 months. Fillipone and coworkers measured lung function in the same group of children at school age. For the group, the mean FEV₁ was 76% of predicted, mid-expiratory flows (FEF₂₅₋₇₅%) were 63% of predicted, and the FEV₁/FVC ratio was 79% of predicted. There was a significant correlation between the V’maxFRC measured at 24 months and both FEV₁ and FEF₂₅₋₇₅% (r = 0.68 and 0.85, respectively). The authors speculated that the obstruction noted in infancy in this group of children with moderate to severe BPD represented airway remodeling, and that such changes in more severely affected children persisted through school age.

In general, most studies of lung function at school age have not assessed outcomes as a function of specific neonatal therapies. Bertrand and coworkers, however, showed that V’maxFRC, a measure of flow through small airways, was reduced at school age in inverse proportion to an oxygen score that quantified neonatal supplemental oxygen exposure. Jacob and coworkers found a significant inverse relationship between FEV₁ at school age and duration of neonatal oxygen therapy in BPD survivors. Kennedy and coworkers also recently demonstrated that duration and amount of supplemental oxygen correlated with the reduction in FEV₁ measured at 11 years of age in children who were VLBW, with or without BPD. Another recent study compared lung function of preterm infants with a history of RDS or BPD at 8 to 9 years of age treated with conventional mechanical ventilation to similar groups that received high frequency ventilation during the stabilization period and were then treated with HFOV. This may be another reason why this study found no effect of mode of ventilation on subsequent function, in contrast to the findings in infants. Young adults who developed RDS as neonates but who were not born prematurely had normal lung function when studied at 18 to 22 years of age. Young adults with a history of BPD, however, had significantly lower FVC, FEV₁, FEF₂₅, 7₅%, and RV/TLC compared with those who were normal full term infants and preterm controls without BPD.

In summary, a number of changes in pulmonary function occur with growth in infants with BPD: normalization of pulmonary mechanics and some lung volumes occur over time, whereas abnormalities of small airway function and air trapping persist. These findings suggest that the processes for lung parenchymal repair may differ from those affecting the conducting airways, and thus magnify the effects of dysynchrony on lung growth. Severity of lung disease in the neonatal period is reflected in abnormalities of lung function, even through school age.

Airway Disease

Although pulmonary function measurements in BPD survivors reflect abnormalities primarily of the small airways, large airway disease occurs commonly in this patient population during infancy. Tracheomalacia and bronchomalacia secondary to endotracheal intubation and prolonged mechanical ventilation are well known. An increase in central airway compliance can result in “BPD spells” which are acute cyanotic events most commonly seen in older BPD infants. The natural history of abnormal central airway collapsibility in BPD infants has not been reported, but in our experience, this problem resolves by the second or third year of life. Other airway problems include inssipated secretions, and formation of granulation tissue and pseudopolyps related to trauma from endotracheal tubes and aggressive “deep” airway suctioning practices. These problems often require surgical intervention.

Airway hyperreactiveness has been demonstrated in infants, children and long-term survivors of BPD. Evidence of airway reactivity in premature infants destined to develop BPD has been demonstrated as early as 3 days of age. Furthermore, a highly significant correlation between the degree of airway reactivity and the severity of respiratory disease as determined by the duration of ventilator dependence was demonstrated, suggesting that airway reactivity may play an important role in the development and severity of BPD. Unlike children with asthma, no increase in prevalence of atopy has been found in children with BPD and bronchial hyperresponsiveness was reported to be unrelated to atopic status.

Exercise Testing

Although the majority of survivors of BPD participate in play, exercise, and other physical activities without symptoms, there is concern over their respiratory reserve given their often stormy, perinatal course. Most studies, however, show no reduction in exercise capacity in children with BPD when compared with children who were healthy term infants or preterm babies without lung disease. Only one study of 12 children between the ages of 6 to 12 years with mild BPD found a decreased maximal exercise capacity compared with 16 healthy controls. In 2 reports from the same institution, children with a history of severe BPD demonstrated no decrease in maximum work load or maximal oxygen consumption compared with former preterm or term controls. In contrast, the BPD group displayed an increase in respiratory rate and decrease in tidal volume resulting in a higher ratio of dead space to tidal volume. The authors speculated that their finding of a reduced maximal minute ventilation/maximal mandatory ventilation ratio suggested a decreased respiratory reserve. In contrast, Pianosi
and Fisk found that both BPD and former preterm subjects without lung disease responded to submaximal exercise by increasing respiratory rate while maintaining tidal volume. They speculated that regulation of breathing during exercise is different in preterm children compared with those born at term.

In most studies, oxyhemoglobin desaturation during exercise was more likely to occur among subjects with a history of BPD than in other groups. Mitchell and Teague found decreases in soluble gas transfer at rest and during exercise in school aged children (6-9 years) with a past history of BPD compared with normal children born at full term or preterm children without BPD. The authors concluded that this finding might be related to a long-term lung structural abnormality resulting in a reduced total alveolar capillary surface area in BPD survivors. This reduction could also account for the observed exertional oxyhemoglobin desaturation.

At this time, the majority of the data suggests that there is no decrease in exercise capacity in survivors of BPD. There remains a concern about decreased respiratory reserve in this population, resulting either from a reduced alveolar-capillary surface area, or other alterations in pulmonary blood flow.

**Radiological Findings**

There have been few follow-up studies that looked at the long-term effects of BPD radiologically. In 10 patients with BPD studied at 6 to 9 years, Hakulinen and coworkers found only minor fibrotic changes on chest radiographs in 40% of the BPD children. No hyperinflation was evident, even though the RV/TLC ratio was elevated. Andreasson and coworkers found chest radiographic abnormalities in 8 of 10 children with BPD studied at 8 to 10 years. All demonstrated generalized hyperinflation, and 4 had localized areas of hyperinflation as well. Five demonstrated mild perihilar fibrosis. Children who had abnormal radiographs immediately after being ventilated had a greater incidence of having abnormal findings on follow-up, but radiological findings improved in children who had also been studied 4 years before. In older subjects, Northway and coworkers found generally subtle findings on chest radiographs in survivors of BPD (mean age 18.3 years), including hyperinflation, interstitial and pleural thickening, blebs, and peribronchial cuffing.

The chest radiograph is relatively insensitive to the structural changes present in the BPD lung. In contrast, high resolution computed tomography (HRCT) is much more sensitive at detecting such abnormalities. In 23 children, ages 2 months to 13 years (mean: 4 years) with BPD who had signs of chronic pulmonary dysfunction (recurrent episodes of coughing, wheezing, dyspnea, pneumonia, or respiratory insufficiency), chest radiographs showed hyperexpansion in 17, hyperlucent areas in 11, and linear opacities in 10 subjects. Pleural thickening was not observed, and four children had normal chest radiographs. All 23 HRCT scans showed abnormalities, however, including multifocal areas of hyperaeration, well-defined linear opacities, and triangular subpleural opacities. In 20 of 23 children, all three abnormalities were present and in 3 other children, 2 of these 3 abnormalities were found. The authors concluded that lesions in survivors of BPD with chronic pulmonary dysfunction are visualized better by CT scans than by chest radiographs. In 26 older children with BPD studied by HRCT, 92% had abnormal findings. Reticular opacities were found in 85%, 69% had areas of architectural distortion, and 92% had gas trapping. There was a positive correlation between abnormal radiological findings and increased air trapping and obstructive lung disease by pulmonary function testing. HRCT scan features in adult survivors of BPD included multifocal areas of reduced lung attenuation and perfusion, bronchial wall thickening, and decreased bronchus-to-pulmonary artery diameter ratios. These findings reflect areas of air trapping, and perhaps the result of airway damage and remodeling.

Advances in diagnostic imaging have uncovered findings in the BPD lung that were either unsuspected or appeared as only mild changes on the chest radiograph. As techniques like controlled ventilation HRCT and protocols that reduce radiation exposure become more widely available, noninvasive imaging will become an important part of the armamentarium to unravel the changes in structure of the BPD lung that occur with growth and maturation.

**Conclusions**

BPD is the result of dynamic processes involving inflammation, injury, repair, and maturation. Outcomes of BPD are difficult to assess given the lack of a uniform definition, and changing modalities of management. Infants with BPD continue to have significant pulmonary sequelae during childhood and adolescence. Neonates with chronic lung disease are more immature today than those studied in the past, and so the prognosis for this population may be different from that reported thus far. There is a progressive decrease in FEV1 with aging in normal adults. Hjalomsen and coworkers estimated that the deficit in lung function seen in the BPD subjects of his study represented more than a decade of decline in lung function. Whether the pulmonary dysfunction in BPD patients will predispose them to obstructive lung disease earlier in adulthood remains to be seen.

Prevention of BPD starts at successful prevention of preterm deliveries; failing this, a better understanding of the effects of prenatal and postnatal factors that affect the immature lung is essential to decrease the severity of BPD. Continued surveillance of young adults with a history of BPD will be critical to understand the long-term impact of neonatal lung injury on pulmonary maturation and aging.

**Acknowledgments**

The authors would like to thank D.J. Weiner, MD, and J.L. Allen, MD, for their critical review of the manuscript.

**References**

3. Parker RA, Lindstrom DP, Cotton RB: Improved survival accounts for most, but not all, of the increase in bronchopulmonary dysplasia. Pediatr Pulmonol 30:663-668, 2000


