

predicting respiratory outcomes (1, 6). Evaluation of the data from Jensen and colleagues may provide supporting or refuting evidence for this maturational cutoff for defining BPD.

We agree that our goal should be to adopt an evidence-informed or data-driven approach to identify neonates of extremely low gestational age with a future risk of developing pulmonary and neurodevelopmental issues. The critical task for members of the neonatal community is to decide what we aim to achieve in defining BPD. Do we want to identify nearly all children who may develop adverse outcomes (minimum false negatives), do we want to rule out all who may not develop adverse outcomes (minimum false positives), or do we want to settle for a compromise and accept a middle ground? The answer may require careful thinking. We may want to use criteria with minimum false negatives when identifying children for closer surveillance during childhood, to predict and manage respiratory adverse outcomes; use criteria with minimum false positives when testing experimental therapies, to rationalize exposure for many children; and use “compromise” criteria (with acceptable sensitivity and specificity cutoffs) for quality improvement initiatives, benchmarking, and assessing trends. Discussions about these issues need to happen through an international forum and consensus process, as is currently underway via the International Neonatal Consortium (1). Purpose-defined BPD criteria derived from an international consensus process and supported by data are essential for avoiding ongoing confusion and inconsistency. ■

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Reply to Bancalari *et al.* and to Isayama and Shah

From the Authors:

Much of the recent debate on how to define bronchopulmonary dysplasia (BPD) has focused on identifying diagnostic criteria that adequately predict meaningful childhood outcomes (1). The goal of our study group was to help inform this debate by providing an evidence-based definition of BPD that was chosen according to the ability to predict respiratory and neurodevelopmental outcomes at 18–26 months corrected age (2). In a contemporary, multicenter cohort of very preterm infants, we found that stratification of disease severity based on the mode of respiratory support administered at 36 weeks postmenstrual age (PMA), irrespective of current or prior exposure to supplemental oxygen, discriminated best between infants with and without adverse early childhood outcomes (2).

In separate letters, Bancalari and colleagues and Isayama and Shah stress that choosing contemporary diagnostic criteria for BPD will require compromise. We agree that a single definition of BPD is unlikely to serve all purposes. For example, the clinical and diagnostic information required to establish the underlying respiratory pathophysiology may differ from that used to predict the presence or absence of future respiratory morbidity. Nevertheless, we believe that our study provides valuable information on how best to define BPD in the current era.

To our knowledge, there are no widely available, uniformly applied, validated diagnostic tests that can precisely characterize the etiology of respiratory failure in preterm infants. Therefore, all commonly used definitions of BPD invoke clinical respiratory support—treatment with supplemental oxygen and, in some cases, the mode of respiratory support—as proxy measures of the underlying respiratory illness (3). Bancalari and colleagues write that “inspired oxygen is the simplest and most sensitive indicator of the severity of respiratory failure and parenchymal lung disease.” However, heterogeneity in oxygen administration is a noted limitation of the existing diagnostic criteria for BPD (1). Moreover, supplemental oxygen is used to treat multiple cardiopulmonary diseases in preterm infants, including apnea, pneumonia, pulmonary hypoplasia, and pulmonary arterial hypertension.

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Certain characteristics of the administered respiratory support, such as the mean airway pressure, can also affect the level of supplemental oxygen required to achieve a target SaO_2 (4). Our analysis considered several potential definitions of BPD that stratified infants based on treatment with supplemental oxygen at levels of $<30\%$ versus $\geq 30\%$. We found that this additional information did not improve our ability to predict respiratory or neurologic outcomes, once we accounted for an infant's mode of respiratory support at 36 weeks PMA.

Bancalari and colleagues also suggest that our proposed definition would be improved by including an indicator of disease chronicity. Notably, we found that requiring exposure to supplemental oxygen for at least 28 days before 36 weeks PMA to establish a diagnosis of BPD did not improve the prognostic accuracy of the definition (2). Whether diagnosing BPD using data collected over several days immediately before or after 36 weeks PMA would improve prediction of childhood outcomes is uncertain. Any benefit conferred by such data must be weighed against the burden of further data collection and possible variability in the application of more complex diagnostic criteria.

Isayama and Shah raise another key question, namely, at what PMA should BPD be diagnosed for the purpose of predicting future morbidity? The literature suggests that the optimal time point remains uncertain. Studies are inconsistent as to when, between 36 and 40 weeks PMA, a diagnosis of BPD best predicts early childhood outcomes (5–7). There is even variability within individual studies depending on which outcome is selected (5, 6). The data do consistently show, however, that diagnosing BPD at later PMAs results in an increase in specificity but a decrease in sensitivity for predicting future morbidity (5–7). This means that moving the diagnosis of BPD beyond 36 weeks PMA will make us more confident that infants with BPD will experience childhood respiratory morbidity. Conversely, this change will make us less certain that infants “without BPD” will survive and be free of respiratory illness. Isayama and Shah suggest that a compromise might be to *a priori* select the diagnostic criteria and assessment time point that best serve a project's stated goals. We agree that such decisions will require careful thinking. As part of this debate, we must consider the pros and cons of using one (albeit imperfect) definition across all research and clinical endeavors versus using multiple definitions that serve select diagnostic purposes.

We look forward to these important discussions and learning about new research that improves our understanding of the pathophysiology and heterogeneity of BPD. In the meantime, we believe that the evidence-based definition of BPD we identified in our analysis will aid in the care of contemporary very preterm infants and support trials investigating new therapies aimed at reducing pulmonary morbidity and preventing adverse long-term outcomes in this vulnerable population. ■

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Erratum: Evaluation and Management of Obesity Hypoventilation Syndrome. An Official American Thoracic Society Clinical Practice Guideline



There is an error in the ATS clinical practice guidelines published in the August 1, 2019, issue of the *Journal* (1). The ATS recommendations in response to Question 4 (“Should hospitalized adults suspected of having OHS, in whom the diagnosis has not yet been made, be discharged from the hospital with or without PAP treatment until the diagnosis of OHS is either confirmed or ruled out?”) should begin with the words “We suggest. . .” This correction should have been made to page e17 of the full document and page 287 of the Executive Summary. Table 1 in both documents does include the correct wording.

The full recommendation should read:

We suggest that hospitalized patients suspected of having OHS be started on NIV therapy before being discharged from the hospital and continued on NIV therapy until they undergo outpatient workup and titration of PAP therapy in the sleep laboratory,

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