

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. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

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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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ideally during the first 3 months after hospital discharge (conditional recommendation, very low level of certainty in the evidence). ■

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Erratum: Microbiological Laboratory Testing in the Diagnosis of Fungal Infections in Pulmonary and Critical Care Practice. An Official American Thoracic Society Clinical Practice Guideline

There are errors in the ATS clinical practice guidelines (1) published in the September 1, 2019, issue of the *Journal*. Because of a problem with the processing of an Excel file, some incorrect values were included in the publication.

On page 540, third column, the negative predictive value for a single positive blood *Aspergillus* PCR test should read 98%, and the negative predictive value for two consecutive positive blood *Aspergillus* PCR tests should read 96%; the negative predictive value for a BAL *Aspergillus* PCR test should read 98%. Reflecting the corrected BAL test performance, the document should read that the test would be expected to yield 87 true positives, 847 true negatives, 13 false negatives, and 53 false positives.

On page 542, first column, the positive predictive value and negative predictive value for a (1→3)-β-D-glucan test with a positivity threshold of 80 pg/ml should read 19% and 97%, respectively. On page 543, third column, the negative predictive value for a *Histoplasma* antigen test should read 98% when assuming a prevalence of 10%, and should read 94% when assuming a prevalence of 25%. The yield would be 204 true positives and 46 false negative (instead of 46 false positives).

The authors of the document stress that none of these corrections alter any of the recommendations included in this ATS clinical practice guideline. ■

Reference

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