

COMMENTARY

Who Needs Oxygen with Positive Airway Pressure Therapy?

Commentary on Shetty et al. Unanticipated nocturnal oxygen requirement during positive pressure therapy for sleep apnea and medical comorbidities. *J Clin Sleep Med.* 2017;13(1):73–79.

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The diagnosis and management of obstructive sleep apnea (OSA) are increasingly being performed in the home. In the home management pathway, patients with high pretest probability for OSA are first tested using home sleep apnea testing and if positive for OSA, are treated with automatic positive airway pressure (APAP). The home management pathway has been validated by multiple investigators¹⁻³ and is considered an alternative pathway in guidelines for the evaluation and management of OSA.⁴ However, there are limitations to the routine use of this pathway. First, studies on home sleep apnea testing have generally excluded patients with significant comorbidities such as morbid obesity (usually body mass index greater than 50 kg/m²), chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), and disorders associated with hypoventilation. Second, if the patient has been prescribed autoPAP, the sleep physician will not know if the patient has residual hypoxia while on treatment with autoPAP because these devices measure a residual apnea-hypopnea index but not residual low oxyhemoglobin saturation. This could result in undertreatment of the patient's sleep-disordered breathing. However, there have been no previous studies that have examined which patients require oxygen in addition to positive airway pressure (PAP) therapy and how to predict who those patients are pretitration.

In this issue of the Journal of Clinical Sleep Medicine, Shetty and colleagues⁵ assess the relationship between medical comorbidities (COPD, CHF, and/or obesity), positional change in pulse oximetry, and need for nocturnal oxygen therapy added to PAP therapy during in-laboratory overnight PAP titration. The study analyzed data obtained retrospectively from 200 patients with OSA, of whom 50 required oxygen supplement in addition to PAP therapy during sleep. Positional change in oxyhemoglobin saturation was measured as the difference between sitting and supine positions using pulse oximetry data during the clinic visit and the sleep study (performed within 3 mo), respectively. The presence of the following predictors: COPD, body mass index greater than 35 kg/m², age older than 50 y, and a more than 5% drop in oxyhemoglobin saturation between upright and supine positions are associated with increased odds of needing oxygen added to PAP therapy. However, having less than two of these predictors provided a negative predictive value of 0.92 for needing oxygen supplementation in addition to PAP therapy.

This study adds important assessment tools to current practice to guide clinicians when ordering PAP titration for patients with OSA. However, there are several remaining questions to be answered. The need for oxygen added to PAP therapy in OSA patients with comorbidities such as COPD and obesity could be related to either hypoventilation during sleep, blunted ventilatory chemoresponses or worsened ventilation-perfusion mismatch without hypoventilation;^{6,7} the data presented by Shetty et al. does not distinguish between these potential etiologies. It is unclear from the data in this study whether there is a linear relationship between severity of airflow obstruction and the need for oxygen supplement added to PAP therapy. Another issue not addressed by this study is how many of the COPD patients used a long-acting inhaled anticholinergic that has been known to improve oxygen saturation during sleep in COPD.⁸ In addition, it is not clear why smoking history did not predict the need for nocturnal oxygen saturation, given its association with COPD, snoring, and OSA. In fact, in another study, it was found that smoking history, mean sleeping oxyhemoglobin saturation, and expiratory snoring were associated with the presence of airflow obstruction on pulmonary function testing and predicted the degree of decline in forced expiratory volume in 1 sec.9

The authors should be commended for carefully analyzing the effect of multiple factors that may contribute to hypoxia during sleep, for example, increasing degrees of obesity and the presence of smoking and COPD, and included these in the prediction model. However, the severity of COPD was not taken into consideration in the analysis and, in the absence of pulmonary function testing findings, it is not clear whether the mere presence of COPD predicted the requirement for supplemental oxygen or whether the severity of lung disease and/or associated daytime hypoventilation contributed to this risk. The information on the severity of obstructive lung disease could be easily obtained using spirometry and would provide a greater degree of certainty for the clinician triaging patients to home versus in-laboratory sleep studies. Moreover, as noted by the authors, the severity or the presence of systolic versus diastolic heart failure could not be assessed adequately in this

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study because of the small sample size. Finally, the authors did not account for the use of prescription opioid drugs, which have also been associated with both daytime and nocturnal hypoxemia independent of the presence of OSA,¹⁰ and thus might be a predictor of supplemental oxygen during PAP treatment. Therefore, it is recommended that future studies evaluate the role of spirometry as a screening measure and consider other potential sources of nocturnal hypoxia when evaluating the appropriateness of home management of OSA based on medical comorbidities.

Finally, given the results from the recent large randomized controlled trial that failed to find any mortality, health or quality-of-life benefits following the use of long-term 24-h oxygen in patients with stable COPD with moderate resting or exercise induced desaturation,¹¹ one needs to investigate whether the addition of the nocturnal oxygen affects cardiovascular and other outcomes in the OSA population. Future prospective studies should also investigate whether supplemental nocturnal oxygen has added health benefits particularly in OSA patients with underlying moderate to severe lung disease.

CITATION

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