

SCIENTIFIC INVESTIGATIONS

Unanticipated Nocturnal Oxygen Requirement during Positive Pressure Therapy for Sleep Apnea and Medical Comorbidities

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Study Objectives: Home-based management of sleep-disordered breathing (SDB) generally excludes patients with significant medical comorbidities, but such an approach lacks scientific evidence. The current study examined whether significant medical comorbidities are associated with persistent hypoxia that requires unanticipated nocturnal O₂ supplementation to positive airway pressure (PAP) therapy. Conceivably, in such patients, home-based management of SDB may not detect or therefore adequately treat persistent hypoxia.

Methods: In this retrospective study of 200 patients undergoing laboratory-based polysomnography, we ascertained significant medical comorbidities (chronic obstructive pulmonary disease, congestive heart failure, and morbid obesity) and their association with the need for unanticipated O₂ supplementation to PAP therapy. Postural oxygen (SpO₂) desaturations between upright and reclining positions were determined during calm wakefulness.

Results: Postural change in SpO₂ during calm wakefulness was greater in patients who eventually needed nocturnal O₂ supplementation to PAP therapy than those needing PAP therapy alone ($p < 0.0001$). The presence of chronic obstructive pulmonary disease (odds ratio [OR] 6.0; 95% confidence interval [CI] 2.1, 17.5; $p = 0.001$), morbid obesity (OR 3.6; 95% CI 1.9, 7.0; $p < 0.0001$), and age older than 50 y (OR 2.8; 95% CI 1.3, 5.9; $p = 0.007$) but not heart failure were associated with unanticipated need for nocturnal O₂ supplementation. A clinical prediction rule of less than two determinants (age older than 50 y, morbid obesity, chronic obstructive pulmonary disease, and postural SpO₂ desaturation greater than 5%) had excellent negative predictive value (0.92; 95% CI 0.85, 0.96) and likelihood ratio of negative test (0.08; 95% CI 0.04, 0.16).

Conclusions: Medical comorbidities can predict persistent hypoxia that requires unanticipated O₂ supplementation to PAP therapy. Such findings justify the use of medical comorbidities to exclude home management of SDB.

Commentary: A commentary on this article appears in this issue on page 7.

Keywords: clinical prediction rule, home sleep apnea testing, obstructive sleep apnea, oxygen, portable testing, positive airway pressure therapy

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INTRODUCTION

Home-based management of sleep-disordered breathing (SDB) utilizing home sleep apnea testing (HSAT) and automatic positive airway pressure therapy (autoPAP) is increasingly used as a management pathway for SDB.^{1–10} Such home-based management of SDB generally excludes patients with significant medical comorbidities because most research studies on which recommendations were based excluded patients with such comorbidities.¹¹ Specifically, significant comorbid medical conditions are thought to degrade the accuracy of HSAT.¹¹ Such comorbid medical conditions include chronic obstructive pulmonary disease (COPD), morbid obesity, and heart failure.¹¹ Whether the existence of such significant comorbid medical conditions contributes to substandard diagnostic or therapeutic management of patients with SDB through home-based management is unclear, and data to support appropriate patient selection for home-based management were recently identified as a research priority.^{1,12} Moreover, a recent meta-analysis of 59 studies involving 5,026 patients found that none of the studies included patients with unstable medical comorbidities and a

BRIEF SUMMARY

Current Knowledge/Study Rationale: Home-based management of sleep-disordered breathing (SDB) generally excludes patients with significant medical comorbidities (such as severe chronic obstructive pulmonary disease, congestive heart failure, and morbid obesity), but such an approach lacks scientific evidence. Moreover, patients prescribed home sleep studies and automatic positive airway pressure (PAP) therapies may not be completely assessed for supplemental oxygen therapy needs in addition to PAP therapy.

Study Impact: We tested a clinical prediction rule of less than two determinants (age older than 50 y, morbid obesity, chronic obstructive pulmonary disease, and postural SpO₂ desaturation higher than 5%) that had excellent negative predictive value for need for unanticipated oxygen supplementation that could assist in appropriate triage of patients through home-based (portable) over laboratory-based management pathways for their SDB.

small number included mild comorbidities such as hypertension and asthma.¹³

Patients with comorbid medical conditions such as morbid obesity or COPD, by virtue of sleep-related nocturnal

hypoventilation, may require unanticipated additional treatment for SDB. For example, nocturnal oxygen (O₂) supplementation may be required in addition to positive airway pressure (PAP) therapy, due to persistent hypoxemia despite PAP therapy alone. For the purposes of this study, unanticipated O₂ supplementation was defined as occurring in patients who were not originally hypoxic or requiring daytime O₂ supplementation prior to sleep diagnostic testing, who then required O₂ supplementation in addition to PAP therapy after sleep laboratory-based testing. If these individuals had undergone HSAT, such a need for O₂ supplementation would not have been discovered, because overnight laboratory-based testing is generally not performed in patients who are initiated on auto-PAP therapy following HSAT. We aimed to determine whether significant medical comorbidities are associated with persistent nocturnal hypoxia that requires unanticipated O₂ supplementation to PAP therapy in the setting of a retrospective cohort of patients undergoing laboratory-based polysomnography. This would provide evidence for the recommendation that individuals with these comorbidities should be evaluated in the laboratory, because home testing would not have discovered a need for supplementary O₂. To address this aim, we performed a retrospective chart review of patients who underwent laboratory-based sleep testing to determine whether the presence of medical comorbidities (COPD, morbid obesity, and/or heart failure) led to unanticipated O₂ supplementation. The hypothesis for the study was that those with medical comorbidities were more likely to require unanticipated oxygen supplementation. If this is the case, then that would support the idea that home-based management of SDB would not detect, or therefore adequately treat, persistent hypoxia in patients with significant comorbid medical conditions who are issued an autoPAP device. The rationale for performing a retrospective chart review of laboratory-based polysomnography patients, as opposed to a prospective study of home-sleep study patients, was to prevent possible and posited harm that is inherent to a prospective study design in at-risk participants. The time spent below oxygen saturation of 90% has been associated with increased risk for all-cause mortality.¹⁴ Therefore, knowingly allowing patients to remain hypoxic under the auspices of a prospective research study with informed consent would be opposed to the principle of beneficence.¹⁵ Allowing persistent hypoxemia may place research participants under risk, however small, when participating in such a research study. Recently, there have been controversies pertaining to allowing hypoxemia under the auspices of research, even though there may have been perceived clinical equipoise or acceptance at the time of commencing such a study.¹⁶

METHODS

We performed a retrospective, case-control study at a single center by reviewing consecutive electronic medical records between January 2012 and December 2014 after receiving appropriate approval from the Institutional Review Board at the University of Arizona (Protocol# 1411574542). Patients who were prescribed PAP therapy with unanticipated entrained O₂

were identified (n = 50) and patients who were prescribed PAP therapy alone (n = 150) were identified from an electronic database that was maintained at the sleep center.

Inclusion criteria were age older than 18 y and completion of a laboratory-based overnight polysomnogram. Exclusion criteria included patients who were already on O₂ prior to undergoing polysomnography; hypoxemia during wakefulness defined as having oxygen saturation (SpO₂) ≤ 88%; lacking SpO₂ measurements while in the upright position in the clinic visits within 3 mo prior to the polysomnography; and patients with the head-end of the bed elevated on the night of the sleep study (as documented by the sleep technician and video recordings on the night of the sleep study). Patients without baseline SpO₂ measurement in room air within 3 mo of the polysomnography were excluded (n = 28). Titration of PAP therapy was performed according to clinical guidelines for the manual titration of PAP in patients with obstructive sleep apnea (OSA).¹⁷ Initiation of entrained oxygen during the manual titration required that OSA be treated adequately (apnea-hypopnea index < 5 events/h) with oxygen saturation ≤ 88% for greater than 5 min.¹⁷

We screened patient data of consecutive patients between January 2012 and December 2014 to yield 50 cases and 150 controls. The cases were patients who were not on oxygen supplementation at the time of referral to the sleep laboratory but then were prescribed such therapy in addition to positive airway pressure therapy after the sleep study was performed. Over this period of time, 452 of the 1,536 consecutive sleep studies were duplicates of patients returning for PAP titration. These and other studies yielded the required 150 consecutive controls that met the selection criteria as per a 1:3 case-to-control ratio that was determined *a priori*. Four hundred eighty-two sleep studies were excluded because those patients met the exclusion criteria.

Determination of Postural Oxygen Desaturation

The rationale for assessing postural oxygen desaturation measured by SpO₂ was to quantify the respiratory status that can be compromised in the supine position and was obtained in the following manner. Recumbent SpO₂ was derived from the polysomnography data during a period of calm wakefulness in the supine posture (confirmed by electroencephalography and digital time-synchronized video). Such information was available in the time period soon after the performance of bio-calibrations while the patient was in the supine position and breathing at a steady state. In contrast to such measurements obtained in the supine position during the polysomnography, during the clinic visit, patients had their oxygen saturation checked by finger pulse oximetry in sitting posture soon after undergoing measurement of other vital signs such as pulse rate and blood pressure. The postural change in SpO₂ was assessed as SpO₂ in the sitting position (obtained from electronic medical record chart review that was performed during the clinic visit) minus SpO₂ measured in the recumbent calm wakefulness position (derived from polysomnography data).

Patient's age, sex, and medical history for significant comorbid conditions (history of heart failure, COPD, neuromuscular disease) as well other patient characteristics (smoking history)

Table 1—Patient characteristics.

	PAP Alone	PAP + O ₂	p value
Age (y)	51 ± 14	59 ± 13	0.001
Sex	87 (58%)	25 (50%)	0.33
COPD	6 (4%)	10 (20%)	0.001
BMI (kg/m ²)	33.8 ± 8.8	38.1 ± 8.5	0.003
Heart failure	8 (5%)	5 (10%)	0.25
Smoker			0.23
Active	13 (9%)	7 (14%)	
Former	51 (34%)	21 (42%)	
Never	86 (57%)	22 (44%)	
Sitting SpO ₂	95% IQR 93, 97	96% IQR 95, 97	0.3
NREM SpO ₂ nadir	88% (83, 90)	83% (79, 85)	< 0.0001
REM SpO ₂ nadir	89% (86, 91)	85% (79, 89)	0.001
AHI (events/h)	15 (6, 27)	21 (7, 40)	0.15
Sleep efficiency	80 ± 13%	77 ± 15%	0.2
Postural (SpO ₂) desaturation	3% (1, 4)	5% (2, 7)	< 0.0001

AHI = apnea-hypopnea index, BMI = body mass index, COPD = chronic obstructive pulmonary disease, NREM = nonrapid eye movement, PAP = positive airway pressure, REM = rapid eye movement, SpO₂ = O₂ saturation by pulse oximetry.

and polysomnography variables were collected by reviewing the medical records. Physician diagnosis of comorbidities that was documented in the electronic medical record was required for such comorbidities to be extracted as being present. Additionally, apnea-hypopnea index, SpO₂ during rapid eye movement and nonrapid eye movement sleep were also extracted from the polysomnography reports.

Statistical Analysis

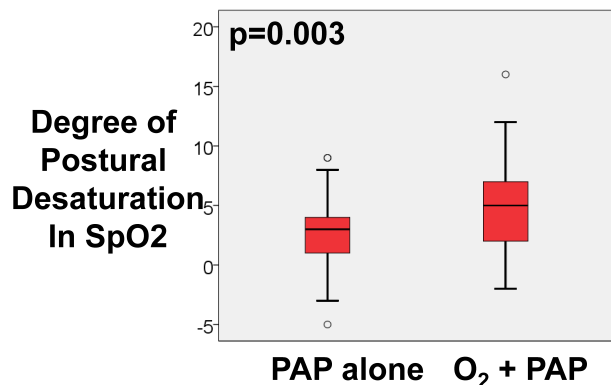
Data were assessed for normality of distribution using one-sample Kolmogorov–Smirnov test. Receiver operating characteristic (ROC) curves were generated for identifying a threshold level with best discriminant function for postural SpO₂ desaturation. Sensitivity, specificity, negative predictive value, positive predictive value, and likelihood ratios were calculated by generating 2 × 2 contingency tables. Student *t*-test or Mann-Whitney *U* tests were used for making comparisons as appropriate for parametric and nonparametric data. Simple and multiple logistic regressions were performed with dependent variable being the prescription for unanticipated O₂ supplementation to PAP therapy. From the list of predictors and potential confounders, simple logistic regression analysis was performed to identify significant factors (independent variables) that influenced the prescription for unanticipated O₂ supplementation. Subsequently, we built multivariate logistic regression models with prescription for unanticipated O₂ supplementation as the dependent variable using significant independent variables identified by univariate logistic regression analysis (*p* ≤ 0.10). Multicollinearity between independent variables was verified, and in the event of collinearity, the strongest predictor variable alone was included. Results are presented as mean and standard deviation unless otherwise specified. Values of *p* < 0.05 were considered significant. All analysis was performed using SPSS version 20.0 (IBM SPSS Inc., Armonk, NY). Unadjusted proportions were compared using Pearson χ^2 test.

RESULTS

Patient characteristics by group (PAP with and without unanticipated entrained O₂) are provided in **Table 1**. Patients who eventually required PAP with unanticipated entrained O₂ were older, had greater body mass index (BMI), with lower nadirs during polysomnography, and greater postural SpO₂ desaturation. Notably, the presence of congestive heart failure was not associated with increased risk for needing unanticipated O₂ supplementation (*p* = 0.25). Degree of postural oxygen desaturation measured by finger pulse oximetry (SpO₂) was greater in patients who eventually needed PAP with unanticipated entrained O₂ than those needed PAP therapy alone (**Figure 1**). ROC curve for degree of postural desaturation in SpO₂ as a predictor of need for unanticipated O₂ supplementation to PAP therapy in patients with SDB is shown in **Figure 2** (ROC area under the curve [AUC] 0.73; 95% confidence interval [CI] 0.65, 0.82). A threshold value of 5% SpO₂ desaturation was selected with the best test characteristics. Similarly, a threshold of 50 y for age was identified. For BMI the threshold for conventional definition for morbid obesity (35 kg/m²) was used. Analysis with a threshold value for morbid obesity as 40 or 45 kg/m² yielded weaker associations with unanticipated oxygen prescription (odds ratio [OR] 3.0; 95% CI 1.5, 6.1; *p* = 0.002 and OR 1.8; 95% CI 0.8, 4.3; *p* = 0.16) than that for 35 kg/m² (OR 3.6; 95% CI 1.9, 7.0; *p* < 0.0001; **Table 2**).

Simple logistic regression identified age older than 50 y, BMI > 35 kg/m², COPD, and postural SpO₂ desaturation > 5% were independently associated with the need to prescribe unanticipated O₂ supplementation to PAP therapy (**Table 2**). A clinical prediction rule of fewer than two of the significant determinants (such as age older than 50 y, morbid obesity, COPD, and postural oxygen desaturation > 5%) had excellent negative predictive value (0.92; 95% CI 0.85, 0.96) and likelihood

Figure 1—Degree of postural oxygen desaturation in finger pulse oximetry (SpO₂) was greater in patients who eventually needed positive airway pressure (PAP) with O₂ supplementation than PAP therapy alone.



PAP alone = patients who needed PAP therapy alone, PAP + O₂ = patients who needed PAP therapy with unanticipated O₂ supplementation for their sleep-disordered breathing (Mann-Whitney *U* test).

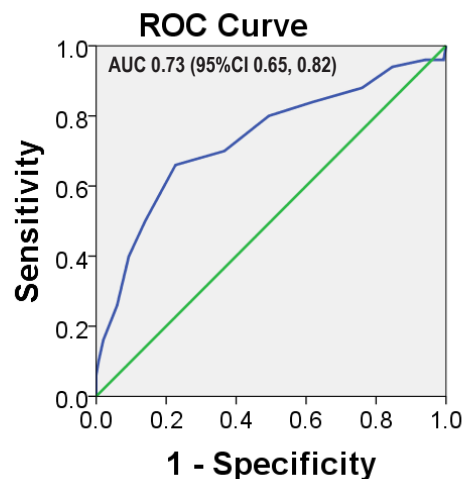
ratio of negative test (0.08; 95% CI 0.04, 0.16) for unanticipated need for O₂ supplementation in addition to PAP therapy.

DISCUSSION

Some general observations can be made about our study. First, a clinical prediction rule of fewer than two of the following determinants—age older than 50 y, morbid obesity, COPD, and postural oxygen desaturation greater than 5%—had excellent negative predictive value and likelihood ratio of negative test for unanticipated need for O₂ supplementation in addition to PAP therapy in patients with SDB. Second, our study provides scientific data that support current consensus guidelines that exclude patients with significant comorbid medical conditions from undergoing home-based management of SDB. Last, heart failure was not a determinant that was associated with increased risk for requiring O₂ supplementation in addition to PAP therapy in patients with SDB.

HSAT was previously recommended as not appropriate for the diagnosis of OSA in patients with significant comorbid medical conditions such as COPD, morbid obesity, neuromuscular disorders, and heart failure. The stated reason for such exclusion was based primarily on the fact that most studies testing such pathways excluded patients with significant comorbid medical conditions.^{11,13,18,19} The cited reason was that such comorbid medical conditions may degrade the accuracy of HSAT. In our study, instead of the accuracy of HSAT as an outcome measure, we chose the requirement for unanticipated need for nocturnal O₂ supplementation to PAP therapy as an outcome measure of importance. Such an outcome measure was chosen based on the rationale that patients with comorbid medical conditions such as morbid obesity or COPD, by virtue of sleep-related nocturnal hypoventilation, may require additional treatment for SDB with nocturnal O₂ supplementation

Figure 2—Receiver operating characteristic (ROC) curve for degree of postural desaturation in O₂ saturation by pulse oximetry as a predictor of need for O₂ supplementation in addition to PAP therapy in patients with sleep-disordered breathing.



AUC = area under the curve, CI = confidence interval.

due to persistent nocturnal hypoxia despite PAP therapy. Such additional need for O₂ supplementation would not be apparent immediately because oximetry testing on autoPAP therapy is usually not performed when patients are initiated on autoPAP therapy following HSAT. Our finding that significant medical comorbidities are associated with persistent hypoxia that requires unanticipated nocturnal O₂ supplementation to PAP therapy justifies current consensus guidelines for medical comorbidities to remain as exclusionary criteria for home management of SDB.¹¹ Such a finding is responsive to calls for research to support appropriate patient selection for home-based management.¹

The time spent below oxygen saturation of 90% has been associated with increased risk for all-cause mortality.¹⁴ Therefore, knowingly allowing patients to remain hypoxic under the auspices of a prospective research study with informed consent would be opposed to the concept of beneficence.¹⁵ Allowing persistent hypoxemia may place research participants under risk, however small, when participating in such a research study. Therefore, we chose to perform a retrospective chart review of laboratory-based polysomnography patients as opposed to a prospective study of home-sleep study patients in order to prevent possible and posited harm that is inherent to a prospective study design. We know that ventilatory response and the arousal stimulus to hypoxia are blunted in both nonrapid eye movement and rapid eye movement sleep.^{20,21} Sustained nocturnal hypoxia may predispose such susceptible patients to cardiac arrhythmias and hence increase morbidity and mortality.

According to the portable monitoring task force of the American Academy of Sleep Medicine, HSAT is not recommended for the diagnosis of OSA in patients with significant comorbid medical conditions, including, but not limited to, moderate to

Table 2—Determinants associated with need for unanticipated O₂ supplementation to positive airway pressure therapy for sleep-disordered breathing.

	Odds Ratio	95% CI	p value
Age	1.04	1.02, 1.07	0.002
Sex	1.38	0.7, 2.6	0.33
COPD	6.0	2.1, 17.5	0.001
BMI	1.05	1.02, 1.09	0.004
Heart Failure	1.97	0.6, 6.3	0.25
Smoker	1.7	0.9, 3.1	0.13
Postural desaturation	5.6	2.8, 11.1	< 0.0001
Age older than 50 y	2.8	1.3, 5.9	0.007
BMI ≥ 35 kg/m ²	3.6	1.9, 7.0	< 0.0001
BMI ≥ 40 kg/m ²	3.0	1.5, 6.1	0.002
BMI ≥ 45 kg/m ²	1.8	0.8, 4.3	0.16
ABCD	12.7	5.3, 30.7	< 0.0001

ABCD = combination of age older than 50 y + BMI ≥ 35 kg/m² + presence of COPD and postural SpO₂ desaturation > 5%, BMI = body mass index, CI = confidence interval, COPD = chronic obstructive pulmonary disease.

severe pulmonary disease, neuromuscular disease, or congestive heart failure and other sleep disorders.²² Although such guidelines exist, health insurance companies modify such criteria to impose restrictions on the use of laboratory-based polysomnography. For example, some insurance companies require BMI > 50 kg/m² as a threshold above which laboratory-based polysomnography would be allowable. Based upon our data, BMI > 35 kg/m² was associated with odds ratio of 3.6 (95% CI; 1.9, 7.0; *p* < 0.0001). We originally used a threshold of 35 kg/m² because it is the accepted threshold for defining morbid obesity; however, we realize that there are some who have variably applied other higher thresholds such as 40 or 45 kg/m². When compared with a threshold of 35 kg/m², the application of higher thresholds weakened the association of such definitions of morbid obesity with the need for unanticipated oxygen supplementation (**Table 2**). Application of such higher BMI thresholds as a cost-saving measure to triage patients away from laboratory sleep studies could potentially decrease our ability to identify patients who may need unanticipated O₂ supplementation following HSAT.

Additionally, currently, there is no age limit for HSAT and home-based initiation of autoPAP therapy. Our study suggests that older individuals may be more likely to require nocturnal O₂ supplementation to PAP therapy was not anticipated prior to testing. Conceivably, such a finding may be due to the fact that many elderly patients may suffer from as yet undiagnosed COPD or other medical conditions that may increase the likelihood for unanticipated O₂ supplementation.

Most studies that validated the use of HSAT excluded patients with comorbid medical problems.¹¹ Two studies, however, included patients with comorbid medical conditions but in neither of these studies was autoPAP therapy initiated following the diagnosis of SDB.^{23,24} Home-based initiation of autoPAP therapy would have been unable to detect concurrent persistent hypoxia and the need for unanticipated O₂ supplementation in addition to PAP therapy in patients with significant medical comorbidities.

There was no association between heart failure and unanticipated need for nocturnal O₂ supplementation to PAP therapy in our study. Such results are in line with recent findings of utility of HSAT in two cohort studies of patients following myocardial infarction or patients with heart failure.^{25,26} There are no studies, however, that test the performance of autoPAP in the presence of significant medical co-morbidities including heart failure as outlined by the American Academy of Sleep Medicine.^{3,11} Most studies of autoPAP therapy excluded participants with significant comorbidities.^{18,27–29} Our prediction tool of positional change in oxygen saturation in combination with age and medical comorbidities could help clinicians identify patients who are most likely to need unanticipated O₂ entrainment to PAP therapy and hence were not ideal candidates for home-based management of SDB.

Several physiological changes occur with normal aging and may have contributed to age older than 50 y being associated with unanticipated need for nocturnal O₂ supplementation to PAP therapy. Various physiological changes in the elderly occur, such as increased chest wall compliance, reduction in lung elastic recoil, and reduction in respiratory muscle strength.³⁰ Such changes may contribute to alveolar hypoventilation and an increase in closing volume of the lungs leading to nocturnal hypoxemia. Additionally, ventilatory response to hypercapnia and hypoxia are reduced in the elderly.³¹ Previously, investigators have demonstrated that in patients with obesity, standing posture improves oxygenation when compared to the supine position.³² Moreover, effect of body position on respiratory physiology of patients with COPD has been studied in great detail by others, suggesting that the supine position places the respiratory system at a disadvantage for performing gas exchange function that would require compensatory changes.^{33–36} Such a “stress test” of the respiratory system could identify individuals who are more likely to be hypoxemic.

In a recent study, Polese and colleagues validated HSAT in the elderly (age older than 65 y) and reported that HSAT could be extended to elderly patients with high probability of SDB, but they too excluded patients with significant comorbidities.³⁷

Conceivably, the age-based effect observed in our study may have been due to as-yet undiagnosed medical comorbidities (**Table 2**). Moreover, in obese patients, Steier and colleagues found that supine posture resulted in elevated gastric pressure and end-expiratory esophageal pressures in obese individuals when compared to control patients, and that such mechanisms may contribute to nocturnal hypoventilation and hypoxemia.³⁸ Others have reported that a restrictive defect noted on pulmonary function testing in morbidly obese individuals was associated with failure of continuous PAP therapy when compared to bilevel PAP therapy in treating obesity hypoventilation syndrome. Such findings suggest that such underlying mechanisms may play a role in the inadequate treatment of SDB in morbidly obese patients.³⁹

Patients with COPD experience reduction in minute ventilation by nearly 25% and elevated upper airway resistance during sleep when compared to wakefulness.⁴⁰ In addition to hypoventilation, ventilation perfusion mismatch, reduced functional residual capacity, and diminished responsiveness to hypoxia and CO₂ retention can occur and contribute to nocturnal hypoxia.^{41,42} Such mechanisms may have played a role in the observed association between COPD and need for unanticipated nocturnal O₂ supplementation to PAP therapy in our study (**Table 2**).

Limitations

There are several limitations to our study. First, this is a retrospective case-control study and such analyses are subject to bias that is inherent to such a retrospective analysis. However, we selected continuous patients in order to prevent such a bias. Second, the initiation of oxygen therapy on the night of the study is subject to the technician's measurement of apnea-hypopnea index on the night of the study to ensure that such patients' sleep-disordered breathing is adequately controlled. To account for such an operator error, we included the cases based on whether the interpreting sleep physician agreed with the sleep technician's decision and issued a prescription for oxygen upon review of the scored polysomnography. Third, the sample size of patients with heart failure was too small for us to understand the effects of types of heart failure on the need for unanticipated oxygen therapy.

CONCLUSIONS

Management pathways guided by simple prediction tools including advanced age, BMI, history of comorbid COPD, and postural desaturation of oxygen may help clinicians predict patients who may need unanticipated nocturnal O₂ supplementation in addition to PAP therapy. Such findings provide scientific evidence to support the current consensus-derived practice guidelines that exclude such patients from undergoing HSAT and home-based autoPAP therapy.

ABBREVIATIONS

autoPAP, automatic positive airway pressure therapy
BMI, body mass index

COPD, chronic obstructive pulmonary disease
CI, confidence interval
HSAT, home sleep apnea testing
OR, odds ratio
OSA, obstructive sleep apnea
PAP, positive airway pressure
ROC, receiver operating characteristics
SDB, sleep-disordered breathing
SpO₂, oxygen saturation

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