Newly Identified Obstructive Sleep Apnea in Hospitalized Patients: Analysis of an Evaluation and Treatment Strategy

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Study Objectives: Obstructive sleep apnea (OSA) case finding in hospitalized patients occurs commonly. In some institutions, the wait time to polysomnography (PSG) may be long. We investigated the impact of a protocol utilizing autoadjusting continuous positive airway pressure (CPAP) for early initiation of therapy in hospitalized patients suspected of having OSA.

Methods: A retrospective chart review of patients with likely OSA (oxygen desaturation index ≥ 10 desaturations per hour with symptoms of OSA) hospitalized between 1999 and 2004 was performed. Subjects were split into intervention and control groups. The intervention group (n = 62) underwent autoadjusting CPAP titration while hospitalized and were discharged on fixed CPAP. The control group subjects (n = 62) had no autoadjusting CPAP titration and were discharged without CPAP (90% on nocturnal oxygen). Both groups then had a follow-up PSG.

Results: The 2 groups were matched for baseline characteristics, admission diagnoses, and oximetry parameters. There were no significant differences in time to PSG or apnea-hypopnea index at PSG. In the intervention group, autoadjusting CPAP improved, but did not normalize, oximetry parameters. No significant differences were found in length of hospital stay or in number of urgent care visits, emergency department visits, or hospital readmissions pending PSG. Compared with PSG-determined CPAP pressures, autoadjusting CPAP underestimated the pressure in 60% of the intervention group, whereas 21% required bilevel positive airway pressure for optimal control.

Conclusions: Compared with oxygen support or no therapy, an autoadjusting CPAP-titration protocol did not improve short-term outcomes in hospitalized patients with symptoms suggestive of OSA. Autoadjusting CPAP may underestimate optimal treatment settings.

Keywords: Continuous positive airway pressure, auto-adjusting CPAP, obstructive sleep apnea, polysomnography


Obstructive sleep apnea (OSA) is a common disorder affecting up to 5% of the adult population in Western countries. Attended laboratory polysomnography (PSG) is recommended as the standard approach for establishing the diagnosis, as well as for initiating treatment with continuous positive airway pressure (CPAP), the first-line treatment for patients with OSA.

OSA case finding in hospitalized patients, based on clinical history and other screening techniques, occurs frequently. At our institution, utilizing history, physical examination, and overnight pulse oximetry, we identified nearly 400 cases of suspected OSA in the year 2004. Unfortunately, clinical instability, limited resources, and variable reimbursement policies often make inpatient PSG impractical. However, the wait time to outpatient PSG may be long, ranging from less than a couple of weeks to more than a year.

Disclosure Statement
Drs. Nader, Steinel, and Auckley have indicated no financial conflicts of interest.

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the efficacy of these strategies in the inpatient setting. There are no data available on how such an approach might affect short-term outcomes in these patients.

Autoadjusting CPAP is a device that automatically adjusts positive pressure, without attendant technician intervention, to maintain airway patency. Autoadjusting CPAP titration can provide an estimated optimal pressure for subsequent fixed-pressure CPAP treatment at home.26-28 We hypothesized that a protocol utilizing autoadjusting CPAP titration for early initiation of CPAP therapy would improve short-term outcomes in newly identified cases of OSA in hospitalized patients.

METHODS

Study Design

A retrospective chart review of consecutive hospitalized adult patients suspected of having OSA was performed. Patients admitted to MetroHealth Medical Center (MHMC), an urban academic center, between January 1999 and August 2004 were included. The study was approved by the institutional review board at MHMC.

Subjects were identified as likely to have OSA by clinical history (loud snoring, excessive daytime sleepiness, witnessed apneas), physical examination (obesity, crowded oropharyngeal or nasopharyngeal airway), and overnight oximetry performed on room air with an oxygen desaturation index (ODI) of 10 or greater. ODI was defined as the number of oxygen desaturations divided by the duration of the study in hours. Oxygen desaturations were defined as an oxygen saturation (So2) less than 90%, a drop of 3% or greater in the SaO2 from baseline for at least 10 seconds, or both.

Subjects

Inclusion criteria included (1) a history and examination suggestive of OSA, as detailed above; (2) an ODI of 10 or greater on room air during overnight oximetry; (3) no inpatient PSG; and (4) follow-up PSG as outpatient. Subjects were categorized into 2 groups; intervention and control. The intervention group was titrated to a fixed CPAP pressure using autoadjusting CPAP and discharged home on a fixed CPAP pressure. The control group had no autoadjusting CPAP titration and was discharged home without CPAP (90% discharged on oxygen). Because this is a retrospective study, a protocol was not in place to determine treatment strategy at the time of hospital discharge. The decision of whether or not to perform autoadjusting CPAP titration before hospital discharge was at the discretion of the attending physician on the medical ward, the attendant pulmonary consult physician, or both. Factored into the decision-making process were patient’s willingness to accept CPAP therapy, comorbid conditions, and the morphometry and degree of desaturations on overnight oximetry. Minors, pregnant women, and those failing to meet the inclusion criteria were excluded.

Screening Overnight Oximetry

Portable pulse oximeters (Nellcor® NPB-290/295, Pleasanton, CA) with Score Analysis software version 1.1a (Mallincrodt 1999) were utilized. Equipped with Oxismart® Advanced Signal Processing and Alarm Management Technology, the pulse oximeters identify electronic and optical noise, as well as motion artifact. SaO2 and pulse rate are monitored continuously, with measurements updated at each pulse beat. The pulse oximeters were operated in the default mode that uses a 5- to 7-second averaging time with electrocardiogram synchronization to decrease motion effect. Oxygen desaturations, as defined above, were counted by computer. Oximetry curves and morphometry of desaturations were also manually examined and determined to be suggestive of periodic breathing. Desaturations due to probe displacement or motion artifact were edited out of the final oximetry study report.

Autoadjusting CPAP Titration

All autoadjusting CPAP titrations in the intervention group were single-night studies, performed the night prior to hospital discharge. A portable automatically adjusting CPAP device (AutoSet™, ResMed, Sydney, Australia) was utilized. The device increases pressure at 1-cm H2O increments in response to apneas of 10 seconds or longer, snoring, or changes in the inspiratory flow-time curve suggestive of inspiratory airflow limitation. If there are no abnormalities, the pressure slowly reduces. An overnight oximetry was performed with each autoadjusting CPAP titration. Assessment of the autoadjusting CPAP titration and the accompanying oximetry were made by the pulmonary consultation team upon completion of these studies. The fixed pressure for home CPAP treatment was determined as the AutoSet pressure exceeded for only 5% of the mask-on time (95th percentile), after excluding periods during which the mask leak exceeded 0.4 L per second. If the autoadjusting CPAP titration did not completely correct the hypoxemia, supplemental oxygen was added to correct for this.

Polysomnography

Standard 15-channel PSG was performed on each subject. Sandman™ software was utilized to collect data. Sleep was staged according to Rechtschaffen and Kales.31 Sleep studies performed before the year 2002 (n = 4 in each of the intervention and control groups) used nasal thermistors to measure flow, whereas those performed after that time, used pressure cannulas. Respiratory events were scored as follows: apnea was defined as the cessation of airflow for 10 seconds or longer with continued effort (obstructive) or lack of effort (central) to breath; hypopnea was defined as a 10-second or longer reduction (≥ 30%) in airflow accompanied by either an arousal or a 3% or greater reduction in SaO2. The apnea-hypopnea index (AHI) was calculated by dividing the number of respiratory events by the duration of sleep in hours. CPAP was titrated to eliminate respiratory disturbances, attempting to achieve an AHI < 5. Once this was accomplished, pressure adjustments could be made to eliminate snoring and reduce arousals. Bilevel pressure support was instituted when control of the respiratory events could not be achieved with CPAP (continued obstructive events despite reaching a CPAP pressure setting of 20 cm H2O or the induction of central events at CPAP pressures necessary to eliminate obstructive events) or when the patient did not tolerate the maximal CPAP pressure necessary for optimal control of the respiratory disturbances. All studies were interpreted by 1 of 2 board-certified sleep physicians, who made determinations as to the optimal pressure setting for each patient.

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No statistically significant differences were observed between the study—62 patients in each of the intervention and control groups. One hundred twenty-four patients satisfied the inclusion criteria for the known diagnosis of OSA, or did not have diagnostic oximetry. One for outpatient PSG. The remainder refused therapy, already had a diagnosis (Table 2). The intervention group had a significantly lower rate of obstructive lung disease, sinusitis, and hypertension. The groups were comparable for other comorbidities.

No statistically significant differences were found in comparing the baseline oximetry parameters between the intervention and control groups: study duration (6.7 ± 0.2 hours vs 6.3 ± 0.2 hours), ODI (30.7 ± 1.8 vs 26.3 ± 1.9), number of desaturations (203.4 ± 12.4 vs 74.8 ± 6.7), time with an oxygen saturation < 90% (70.8 ± 10.0 vs 19.8 ± 4.7), and lowest SaO₂ (73.8 ± 1.3 vs 80.7 ± 1.2). Autoadjusting CPAP improved oximetry parameters of patients in the intervention group but did not completely normalize them (Table 3).

All subjects in the intervention group were discharged home on a fixed CPAP pressure (mean 10.3 ± 0.3 cm H₂O). Fifteen of them (24%) were also discharged home with supplemental nocturnal oxygen. In the control group, 56 subjects (90%) were discharged home on supplemental nocturnal oxygen pending outpatient PSG. Fifty subjects in the intervention group (81%) had their outpatient PSG within 90 days of hospital discharge, compared with 38 (61%) of the controls (p < .05). However, no statistically significant difference was found in mean time to outpatient PSG between the intervention and control groups (77.2 ± 14.4 vs 80.9 ± 10.5 days, autoCPAP in hospitalized patients.)
respectively). All study subjects suspected of having OSA had the diagnosis confirmed on PSG. The mean AHI at outpatient PSG was almost identical between the intervention and control groups (59.4 ± 4.7 and 60.0 ± 4.5, respectively). The intervention group AHI ranged from 7.0 to 145.0; 51 subjects (82%) had an AHI > 30. The control group AHI ranged from 12.0 to 162.8; 51 subjects (82%) had an AHI > 30.

The fixed CPAP setting determined by autoadjusting CPAP titration agreed with the PSG-determined CPAP setting in only 12 subjects (19%) in the intervention group. Autoadjusting CPAP titration underestimated the optimal pressure in 37 (60%) of these subjects. For this subset of subjects in the intervention group, the mean pressure determined by autoadjusting CPAP was 9.7 ± 0.3 cm H\textsubscript{2}O versus 13.7 ± 0.3 cm H\textsubscript{2}O at PSG (p < .0001) (Figure 1). This observation remained valid after adjusting data for subjects admitted to the hospital with respiratory distress due to chronic obstructive pulmonary disease (COPD) or congestive heart failure. The remaining 13 subjects in the intervention group (21%) required bilevel positive airway pressure at PSG for optimal control of the sleep-disordered breathing. Ten of these 13 subjects in the intervention group failed CPAP therapy due to persistent respiratory events on maximal CPAP pressure, and the remaining 3 subjects could not tolerate the maximal CPAP pressure necessary for optimal control. Three of these 13 subjects in the intervention group who failed CPAP (23%) had COPD. Likewise, of the 14 subjects in the intervention group with a history of COPD, 3 (21%) required bilevel positive airway pressure; the remaining 11 subjects (79%) with COPD tolerated CPAP well.

In the control group, 37 subjects (60%) were titrated to CPAP at PSG with a mean pressure of 12.4 ± 0.3 cm H\textsubscript{2}O (p < .05 when compared with mean CPAP pressure in the intervention group). The remaining 25 subjects in the control group (40%) required bilevel positive airway pressure at PSG for optimal control of the sleep-disordered breathing (p < .05 when compared with the intervention group). Eighteen of these 25 subjects in the control group failed CPAP therapy due to persistent respiratory events on maximal CPAP pressure, and the remaining 7 subjects could not tolerate the maximal CPAP pressure necessary for optimal control. Eight of the 25 subjects (32%) in the control group who failed CPAP had COPD (p < .05 when compared with the intervention group).
group). Of the 24 subjects in the control group with a history of COPD, 8 (33%) required bilevel positive airway pressure; the remaining 16 subjects (67%) with COPD tolerated CPAP well.

No significant differences were observed in length of hospital stay between the intervention and control groups (7.8 ± 1.1 vs 7.6 ± 1.1 days, respectively). In the time period patients were awaiting PSG (mean 79 days), there were no significant differences between the 2 groups in the number of urgent care clinic visits, emergency department visits, and hospital readmissions (Figure 2). This observation remained valid after adjusting data for diagnoses at the time of the autoadjusting CPAP titration or an unattended autoadjusting CPAP titration followed by treatment with a fixed CPAP setting. In addition, the combination of symptoms and an abnormal nocturnal oximetry recording accurately predicted the presence of OSA in all cases from both groups. Furthermore, 81% of the patients studied were classified as having severe OSA by their PSG evaluation.40

This is in contrast with a recent report that in-patient level 3 portable sleep studies overestimated the presence of OSA in an at-risk population when the patients were studied while hospitalized.41 The discrepancy between this finding and the highly predictive value of screening by symptoms and oximetry utilized in our study may best be explained by the requirement of oxygen desaturations (and not flow abnormalities) to screen for OSA, thus improving the positive predictive value of an abnormal test. In addition, there appeared to be an overrepresentation of severe OSA in our population, which would enhance the predictive value of overnight oximetry as the screening tool.

CPAP therapy for OSA has been shown to improve both blood pressure and cardiac ejection fraction following as short as 4 to 12 weeks of treatment.11,17,18,42 This might be expected to translate into improved short-term outcomes in a hospitalized patient population enriched with cardiovascular diseases (most of whom were admitted for cardiovascular illnesses). Why, then, did this study not find a difference in outcomes? The patient populations were comparable in terms of demographics, admission diagnoses, comorbidities, and presence and severity of OSA. Other factors may provide an explanation for the apparent lack of improvement.

In the hospitalized patients examined in this study, autoadjusting CPAP titration to a fixed pressure setting appeared to yield an effective therapeutic pressure setting in only 19% of the patients. The remaining patients required either substantially higher pressures (Figure 1) or bilevel pressure support for optimal control of their sleep-disordered breathing. This was independent of patient’s diagnosis at the time of the autoadjusting CPAP titration and occurred despite choosing a fixed CPAP setting from the autoadjusting CPAP titration using an algorithm found to be effective in an outpatient setting.44

This discrepancy may be related to the unstable nature of hospitalized patients, who might be subject to interventions or illnesses that worsen their OSA or nocturnal hypoxemia while admitted. A recent review of autoadjusting CPAP for treatment of OSA by the American Academy of Sleep Medicine cautioned against the use of autoadjusting CPAP in patients with significant lung disease or congestive heart failure, though this recommendation was not evidence based.45 Low levels of CPAP (usually 1-4 cm H2O) have been used as “sham” or placebo CPAP in some studies and have generally failed to improve quality-of-life measures or blood pressure.11,41,44 Whereas a suboptimal titration via autoadjusting CPAP does not equate to “sham” CPAP, it is possible that ineffective pressure settings may have contributed to the lack of improvement in the intervention group.

Compliance with CPAP therapy is suboptimal for many patients with OSA. In a randomized, controlled, parallel-group study in which patients underwent either an attended conventional CPAP titration or an unattended autoadjusting CPAP titration followed by treatment with a fixed CPAP setting, there was no difference in acceptance of CPAP therapy and an actual improvement in plans...
to continue CPAP therapy at 6 weeks in the group who had the autoadjusting CPAP titration.\textsuperscript{45} In contrast, a retrospective review found that patients titrated to a fixed CPAP setting by autoadjusting CPAP were less likely to be compliant with CPAP at their 4 to 6 week follow-up appointment, as compared with those who underwent in-lab CPAP titration.\textsuperscript{46} Of interest, data suggest that as little as 4 hours of use of CPAP per night may be enough to improve oxygenation for the duration of the night in patients with severe OSA.\textsuperscript{47} However, even with this in mind, it is quite possible that the time on CPAP therapy prior to outpatient PSG (mean 79 days) was too short to significantly impact short-term cardiovascular outcomes. Inadequate treatment with CPAP, either due to lack of compliance (which we were unable to verify) or too short of a treatment time, could have minimized any potential differences in outcomes between the groups.

The majority of the patients in our control group were placed on supplemental nocturnal oxygen between the time of their hospitalization and their outpatient PSG. Whereas nocturnal oxygen has been utilized as a treatment for OSA, there are few data examining its effectiveness. Oxygen therapy, when compared with CPAP, was found to improve nocturnal oxygenation and decrease hypopneas, but not apneas or daytime symptoms related to OSA in a small controlled trial.\textsuperscript{29} Another small single-night study failed to find improvement in blood pressure with the use of supplemental oxygen in 8 patients with severe OSA.\textsuperscript{48} In contrast, circulating vascular endothelial growth factor, a potential mediator of cardiovascular disease that is found to be elevated in patients with OSA when compared with controls, reduces to levels of those of the control population following amelioration of nocturnal hypoxia with oxygen therapy.\textsuperscript{49} Despite this, a recent evidence-based review did not cite oxygen as a treatment modality for OSA.\textsuperscript{50} The role of supplemental oxygen therapy in our study is unclear; based upon the above information, it is conceivable that its use in the control population may have attenuated differences in outcomes.

Finally, because this study was performed as a retrospective review, we were limited in the outcomes that could be assessed. Recurrent urgent care clinic visits, emergency department visits, and readmission rates are important outcomes to be considered in this patient population. However, we only had data for our own institution, and it is possible that patients could have sought healthcare elsewhere following discharge from MHMC, though one might expect this to occur randomly and equally between groups. The same reasoning could be applied to accident rates. It should be noted that MHMC is the only Level I trauma center for the region, and most serious trauma is brought to the emergency department at MHMC.

Our study lacks data on other key outcomes, including blood pressure control, heart function, functional recovery from acute illness (especially stroke), and quality-of-life measures (such as alertness, vitality, sense of well-being). Significant differences in any of these outcomes would have been missed by this study. Furthermore, the retrospective nature and the inclusion and exclusion criteria may have introduced selection biases in the patients included in the study. This could have attenuated potential differences in some of the outcomes measured.

The diverse patient population and retrospective nature of our study introduces limitations and potential biases that should be considered in the interpretation of our findings. Given the importance of this issue and the limited resources available at many institutions, protocols for efficient and economical evaluation of these patients need to be developed. Randomized, controlled, prospective studies should be performed to better clarify the role of autoadjusting CPAP titration in the acute inpatient setting, as well as its effect on health outcomes. Alternative strategies, such as algorithm-derived CPAP settings or oxygen therapy, should also be further investigated in this setting.

\textbf{REFERENCES}

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