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SCIENTIFIC INVESTIGATIONS

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

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Obstructive sleep apnea (OSA) is a common disorder affecting up to 5% of the adult population in Western countries.[1] 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),[2,3] the first-line treatment for patients with OSA.[3-6]

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.[7,8] However, the wait time to outpatient PSG may be long, ranging from less than a couple of weeks to more than a year.[8]

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

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Untreated, OSA is associated with multiple adverse health outcomes. It is an independent risk factor for the development of systemic hypertension[9-11] and has been associated with an increased risk of coronary artery disease,[12,13] congestive heart failure,[13,17,18] and ischemic cerebrovascular disease.[14-16] OSA is also a risk factor for motor vehicle accidents.[19,20] Mounting data suggest that the use of CPAP for the treatment of OSA can improve a number of these conditions. Lowering of blood pressure can occur as soon as within 24 hours of initiating therapy.[11] One- and 3- month trials of CPAP for patients with OSA and congestive heart failure have found improvements in ejection fraction, catecholamine levels, and quality of life.[17,18] Because the hospitalized population is enriched for cardiovascular illnesses, it is reasonable to expect that early identification and treatment of modifiable cardiac risk factors might result in better short-term health outcomes. In addition, 1 month placebo-controlled trials of CPAP have demonstrated improvements in sleepiness and vigilance, factors felt to be responsible for the increased accident rate in patients with untreated OSA.[22,23]

Alternative strategies for the early initiation of CPAP therapy pending PSG-CPAP titration have been proposed for use in the outpatient setting. In some reports, algorithm-derived CPAP pressure[24,25] or autoadjusting CPAP titration[26-28] have been utilized to determine a fixed optimal pressure. In others, oxygen supplementation has been used to eliminate apnea-related hypoxemia and associated cardiac arrhythmias.[29,30] Little research has addressed 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 (SaO_2) less than 90%, a drop of 3% or greater in the SaO_2 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/NPB-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. SaO_2 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 H_2O 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 SaO_2. 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 H_2O 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.

Table 1—General Characteristics of the Study Population

Demographics	Intervention Group (n = 62)	Control Group (n = 62)	p Value
Age, y	55.1 ± 1.7	52.4 ± 1.7	NS
Men	50	55	NS
Race			
Caucasian	55	52	NS
African American	30	35	NS
Hispanic	15	13	NS
BMI, kg/m²	44.6 ± 1.6	43.3 ± 1.6	NS
Married	34	32	NS
Actively employed	23	26	NS
Insured	97	92	NS
Smoker (>20 pack-years)	27	23	NS
Active alcohol drinker	11	8	NS
Baseline comorbidities			
COPD	22	38	< .04
Asthma	10	15	NS
Chronic sinusitis	10	34	< .001
Hypertension	74	89	< .04
Diabetes mellitus	36	41	NS
CAD	18	21	NS
CHF	24	23	NS
Cardiac arrhythmias	10	13	NS
Stroke	7	11	NS
Depression	40	36	NS

Values are presented as percentages, except age and body mass index (BMI), which are presented as mean ± SEM. COPD refers to chronic obstructive pulmonary disease; CAD, coronary artery disease; CHF, congestive heart failure; NS, not significant.

Table 2—Admission Diagnoses

Admission Diagnosis	Intervention Group (n = 62)	Control Group (n = 62)	p Value
Chest pain NOS	30	26	NS
Cardiac arrhythmias	2	1	NS
CHF	11	10	NS
Shortness of breath NOS	13	15	NS
COPD	10	12	NS
Asthma	5	6	NS
Pneumonia	2	4	NS
Stroke	11	13	NS
Seizure	2	2	NS
MVA	2	0	NS
PVD/Cellulitis	4	5	NS
Renal Failure	3	2	NS
Miscellaneous	5	4	NS

Data are presented as percentages of the total number of patients in the group. CHF refers to congestive heart failure; COPD, chronic obstructive pulmonary disease; MVA, motor vehicle accident; PVD, peripheral vascular disease; NOS, not otherwise specified; NS, not significant.

Table 3—Effect of Autoadjusting CPAP on Oximetry Parameters in the Intervention Group

Oximetry Parameters	Oximetry on Room Air	Oximetry on Autoadjusting CPAP	p Value
Study duration, h	6.7 ± 0.2	6.3 ± 0.2	NS
Desaturations, no.	203.4 ± 12.4	74.8 ± 6.7	<0.001
ODI, no./h	30.7 ± 1.8	12.5 ± 1.2	<0.001
SaO_2 < 90%, min	70.8 ± 10.0	19.8 ± 4.7	<0.001
Lowest SaO_2, %	73.8 ± 1.3	80.7 ± 1.2	NS

Data are presented as mean ± SEM. CPAP refers to continuous positive airway pressure; ODI, oxygen desaturation index; SaO_2, oxygen saturation; NS, not significant; SS, statistically significant.

Data Collection

Medical records were abstracted for baseline demographics, admission diagnoses, comorbid illnesses, and autoadjusting CPAP and overnight oximetry data. Data on the following outcomes were also abstracted: length of hospital stay, time to PSG, results of PSG, CPAP titration results, and urgent medical visits (urgent care appointments, emergency department visits, readmissions to the hospital, accidents) between the time of discharge and the time of the PSG.

Statistical Analysis

Statistical analyses were performed on the software package SPSS version 11.0 (SPSS Inc., Chicago, IL). All data were normally distributed and are reported as means ± SEM. Between-group comparisons were performed by χ^2 tests for difference of proportions for nominal variables and 2-sample unpaired t tests for continuous numeric variables. P values < .05 were considered to be statistically significant.

RESULTS

Approximately 180 patients who had autoadjusting CPAP titrations in the hospital were excluded from the study. More than 70% of these patients were excluded due to failure to follow-up for outpatient PSG. The remainder refused therapy, already had a known diagnosis of OSA, or did not have diagnostic oximetry. One hundred twenty-four patients satisfied the inclusion criteria for the study—62 patients in each of the intervention and control groups. No statistically significant differences were observed between the 2 study groups in terms of demographics (Table 1) or admission 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 6.8 ± 0.2 hours), ODI (30.7 ± 1.8 vs 26.3 ± 1.9), number of desaturations (203.4 ± 12.4 vs 76.3 ± 12.8), time with an SaO_2 < 90% (70.8 ± 10.0 minutes vs 59.0 ± 10.0 minutes), and lowest SaO_2 (73.8% ± 1.3% vs 74.75 ± 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 H_2O). 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,

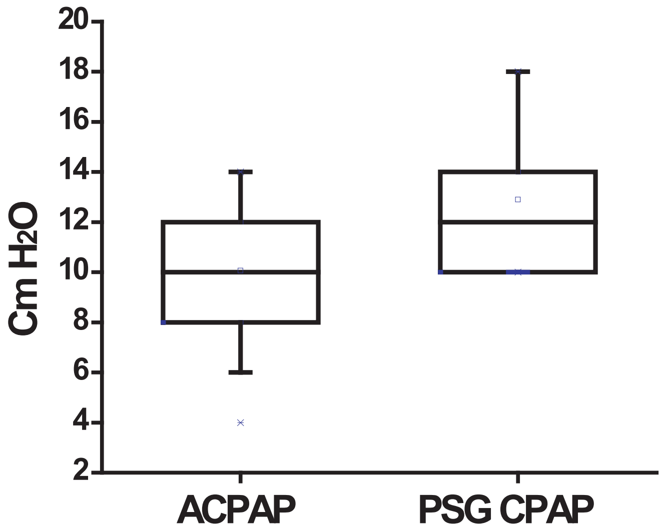

Figure 1—Box Plot of autoadjusting continuous positive airway pressure (ACPAP) vs polysomnography (PSG) CPAP pressures, in cm H_2O, of the 49 subjects (79%) in the intervention group who were titrated to CPAP therapy at PSG. The remaining 13 subjects (21%) who required bilevel pressure support at PSG for optimal treatment are not included.

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_2O versus 13.7 ± 0.3 cm H_2O 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_2O ($p < .05$ 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

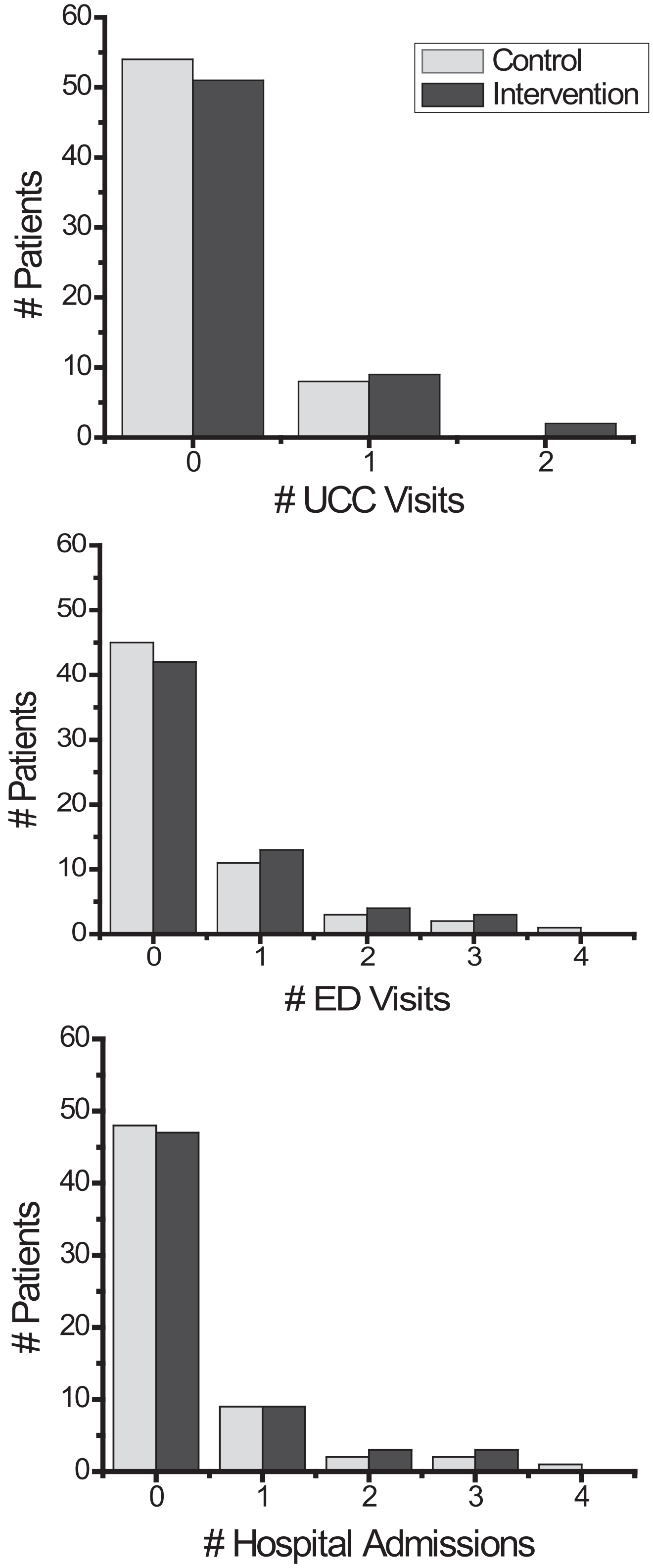

Figure 2—Comparison of outcomes in the intervention and control groups. No statistically significant differences were present between the intervention and control groups in the number of urgent care clinic (UCC) visits, emergency department (ED) visits, and hospital admissions.

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

Table 4—Diagnosis-Specific Comparison of Outcomes

Diagnosis	Intervention Group			Control Group			p Value
	UCC n=11	ED n=20	HA n=15	UCC n=8	ED n=17	HA n=14	
Chest pain NOS	9	25	25	13	22	30	NS
Cardiac arrhythmias	0	5	7	0	6	7	NS
Congestive heart failure	18	15	20	13	12	21	NS
Shortness of breath NOS	27	20	20	25	18	14	NS
COPD/Asthma	18	10	7	13	12	7	NS
Hypertension	9	10	0	13	12	0	NS
Stroke	0	5	7	0	6	7	NS
Seizure	0	5	7	0	6	7	NS
MVA	0	0	0	0	0	0	NS
Miscellaneous	19	5	7	23	6	7	NS

Data are presented as percentages. UCC refers to urgent care clinic visit; ED, emergency department visit; HA, hospital admission; NOS, not otherwise specified; COPD, chronic obstructive pulmonary disease; MVA, motor vehicle accident; NS, not significant.

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 urgent care clinic visits, emergency department visits, and hospital readmissions (Table 4). No motor vehicle accidents were noted in either group.

DISCUSSION

The optimal strategy for the diagnosis and management of suspected new cases of OSA in the inpatient setting has not been defined. Whereas attended PSG is considered the procedure of choice for diagnosing OSA and initiating treatment with CPAP therapy, this approach has not been studied in this patient population. In addition, this strategy requires the inefficient use of a limited resource and therefore may not be considered an option for many institutions. Our study was designed to examine a potential protocol for the diagnosis and management of suspected cases of OSA in hospitalized patients. Similar approaches have been utilized in the outpatient setting with a fairly high degree of success.[25,32-34] Studies suggest that protocols utilizing auto-CPAP–derived fixed pressure settings can reasonably approximate PSG-determined CPAP settings and result in earlier initiation of therapy without decreasing compliance.[33,34] In contrast, our study suggests that this approach in hospitalized patients may lead to a significant underestimation of pressure requirements and does not appear to alter short-term outcomes, though several aspects of the study warrant further discussion.

The use of overnight oximetry recordings as a method for detecting OSA has remained controversial because of the variable sensitivity (31%-98%) and specificity (41%-100%) of oximetry, though some authors have embraced it as a reasonable tool for risk stratification of suspected cases.[35] In studies reporting a positive predictive value, the presence of recurrent oscillations in the oximetry tracing suggest periodic breathing in more than 87% of cases.[36-38] Utilizing overnight oximetry data in conjunction with symptoms consistent with OSA may help to improve accuracy.[39] In our study, 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.[34] 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.[43] Low levels of CPAP (usually 1-4 cm H_2O) 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.[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.[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.[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.[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.[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.[49] Despite this, a recent evidence-based review did not cite oxygen as a treatment modality for OSA.[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.

REFERENCES

1. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. Am J Respir Crit Care Med 2002;165:1217-39.
2. Kushida CA, Littner MR, Morgenthaler T, et al. Practice parameters for the indications for polysomnography and related procedures: An update for 2005. Sleep 2005;28:499-521.
3. American Thoracic Society. Indications and Standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. Am J Respir Crit Care Med 1994;150:1738-45.
4. Loube DI, Gay PC, Strohl KP, Pack AI, White DP, Collop NA. Indications for positive airway pressure treatment for adult obstructive sleep apnea patients: a consensus statement. Chest 1999;115:863-6.
5. Ballester E, Badia JR, Hernandez L, et al..Evidence of the effectiveness of continuous positive airway pressure in the treatment of sleep apnea/hypopnea syndrome. Am J Respir Crit Care Med 1999;159:495-501.
6. Monasterio C, Vidal S, Duran J, et al. Effectiveness of continuous positive airway pressure in mild sleep apnea-hypopnea syndrome. Am J Respir Crit Care Med 2001;164:939-43.
7. Newman SA. Inpatient sleep testing. Sleep Review 2002;3:16-7.
8. Flemons WW, Douglas NJ, Kuna ST, Rodenstein DO, Wheatley J. Access to diagnosis and treatment of patients with suspected sleep apnea. Am J Respir Crit Care Med 2004;169:668-72.
9. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med 2000;342:1378-84.
10. Brooks D, Horner RL, Kozar LF, Render-Teixeira CL, Phillipson EA Obstructive sleep apnea as a cause of systemic hypertension: evidence from a canine model. J Clin Invest 1997;99:106-9.
11. Becker HF, Jerrentrup A, Ploch T, et al. Effect of nasal continuous positive airway pressure treatment on blood pressure in patients with obstructive sleep apnea. Circulation 2003;107:68-73.
12. Leung RS, Bradley TD. Sleep apnea and cardiovascular disease. Am J Respir Crit Care Med 2001;164:2147-65.
13. Shahar E, Whitney CW, Redline S, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. Am J Respir Crit Care Med 2001;163:19-25.
14. Hui DSC, Choy DKL, Wong LKS, et al. Prevalence of sleep-disordered breathing and continuous positive airway pressure compliance: results in Chinese patients with first-ever ischemic stroke. Chest 2002;122:852-60.
15. Bassetti C, Aldrich MS, Chervin RD, Quint D. Sleep apnea in patients with transient ischemic attack and stroke: a prospective study of 59 patients. Neurology 1996;47:1167-73.
16. Dyken ME, Somers VK, Yamada T, Ren ZY, Zimmerman MB. Investigating the relationship between stroke and obstructive sleep apnea. Stroke 1996;27:401-7.
17. Mansfield DR, Gollogly NC, Kaye DM, Richardson M, Bergin P, Naughton MT. Controlled trial of continuous positive airway pressure in obstructive sleep apnea and heart failure. Am J Respir Crit Care Med 2004;169:361-6.
18. Kaneko Y, Floras JS, Usui K, et al. Cardiovascular effects of continuous positive airway pressure in patients with heart failure and obstructive sleep apnea. N Engl J Med 2003;348:1233-41.
19. McNicholas WT. Sleep apnea and driving risk. Eur Respir J 1999;13:1225-7.

20. George CFP. Reduction in motor vehicle collisions following treatment of sleep apnea with nasal CPAP. Thorax 2001;56:508-12.
21. Faccenda JF, Mackay TW, Boon NA, Douglas NJ. Randomized placebo-controlled trial of continuous positive airway pressure on blood pressure in the sleep apnea-hypopnea syndrome. Am J Respir Crit Care Med. 2001;163:344-8.
22. Engleman HM, Martin SE, Deary IJ, Douglas NJ. Effect of continuous positive airway pressure treatment on daytime function in sleep apnoea/hypopnoea syndrome. Lancet 1994;343:572-5.
23. Hack M, Davies RJO, Mullins R, et al. Randomized prospective parallel trial of therapeutic versus subtherapeutic nasal continuous positive airway pressure on simulated steering performance in patients with obstructive sleep apnoea. Thorax 2000;55:224-31.
24. Stradling JR, Hardinge M, Smith DM. A novel, simplified approach to starting CPAP therapy in OSA. Respir Med 2004;98:155-8.
25. Stradling JR, Hardinge M, Paxton J, Smith DM. Relative accuracy of algorithm-based prescription of nasal CPAP in OSA. Respir Med 2004;98:152-4.
26. Masa JF, Jimenez A, Duran J, et al. Alternative methods of titrating continuous positive airway pressure. Am J Respir Crit Care Med 2004;170:1218-24.
27. Teschler H, Berthon-Jones M, Thompson AB, Henkel A, Henry J, Konietzko N. Automated continuous positive airway pressure titration for obstructive sleep apnea syndrome. Am J Respir Crit Care Med 1996;154:734-40.
28. Littner M, Hirshkowitz M, Davila D, et al. Practice parameters for the use of auto-titrating continuous positive airway pressure devices for titrating pressure and treating adult patients with obstructive sleep apnea syndrome. Sleep 2002;25:143-7.
29. Phillips BA, Schmitt FA, Berry DT, Lamb DG, Amin M, Cook YR. Treatment of obstructive sleep apnea. A preliminary report comparing nasal CPAP to nasal oxygen in patients with mild OSA. Chest 1990;98:325-30.
30. Fletcher EC, Munafo DA. Role of nocturnal oxygen therapy in obstructive sleep apnea. When should it be used? Chest 1990;98:1497-504.
31. Rechtschaffen A, Kales A, eds. A Manual of Standardized Terminology: Techniques and Scoring Systems for Sleep Stages of Human Subjects. Los Angeles: UCLA Brain Information Service/Brain Research Institute; 1968.
32. Berkani M, Lofaso F, Chouaid C, et al. CPAP titration by an auto-CPAP device based on snoring detection: a clinical trial and economic considerations. Eur Respir J 1998;12:759-63.
33. Series F. Accuracy of an unattended home CPAP titration in the treatment of obstructive sleep apnea. Am J Respir Crit Care Med 2000;162:94-7.
34. Gagnadoux F, Rakotonanahary D, Martins de Araujo MT, Barros-Vieira S, Fleury B. Long-term efficacy of fixed CPAP recommended by autoset for OSAS. Sleep 1999;22:1095-9.
35. Netzer N, Eliasson AH, Netzer C, Kristo DA. Overnight pulse oximetry for sleep-disordered breathing in adults. Chest 2001;120:625-33.
36. Nuber R, Vavrina J, Karrer W. Predictive value of nocturnal pulse oximetry in sleep apnea screening. Schweiz Med Wochenschr Suppl 2000;116:120S-22S.
37. Ryan PJ, Hilton MF, Boldy DAR, et al. Validation of British Thoracic Society guidelines for the diagnosis of the sleep apnoea/hypopnoea syndrome: can polysomnography be avoided? Thorax 1995;50:972-5.
38. Levy P, Pepin JL, Deschaux-Blanc C, Paramelle B, Brambilla C. Accuracy of oximetry for detection of respiratory disturbances in sleep apnea syndrome. Chest 1996;109:395-9.
39. Gurubhagavatula I, Maislin G, Pack AI. An algorithm to stratify sleep apnea risk in a sleep disorders clinic population. Am J Respir Crit Care Med 2001;164:1904-9.
40. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. Sleep 1999;22:667-89.
41. Skinner MA, Choudhury MS, Homan SDR, Cowan JO, Wilkins GT, Taylor DR. Accuracy of monitoring for sleep-related breathing disorders in the coronary care unit. Chest 2005;127:66-71.
42. Dimsdale JE, Loredo JS, Profant J. Effect of continuous positive airway pressure on blood pressure; a placebo trial. Hypertension 2000;35:144-7.
43. Berry RB, Parish JM, Hartse KM. The use of auto-titrating continuous positive airway pressure for treatment of adult obstructive sleep apnea. Sleep 2002;25:148-73.
44. Jenkinson C, Davies RJO, Mullins R, Stradling JR. Comparison of therapeutic and subtherapeutic nasal continuous positive airway pressure for obstructive sleep apnoea: a randomized prospective parallel trial. Lancet 1999;353:2100-5.
45. Stradling JR, Barbour C, Pitson DJ, Davies RJO. Automatic nasal continuous positive airway pressure titration in the laboratory: patient outcomes. Thorax 1997;52:72-5.
46. Means MK, Edinger JD, Husain AM. CPAP compliance in sleep apnea patients with and without laboratory CPAP titration. Sleep Breathing 2004;8:8-14.
47. Hers V, Liistro G, Dury M, Collard PH, Aubert G, Rodenstein DO. Residual effect of nCPAP applied for part of the night in patients with obstructive sleep apnoea. Eur Respir J 1997;10:973-6.
48. Ali NJ, Davies RJO, Fleetham JA, Stradling JR. The acute effects of continuous positive airway pressure and oxygen administration on blood pressure during obstructive sleep apnea. Chest 1992;101:1526-32.
49. Teramoto S, Kume H, Yamamoto H, et al. Effects of oxygen administration on the circulating vascular endothelial growth factor (VEGF) levels in patients with obstructive sleep apnea syndrome. Internal Medicine 2003;42:681-5.
50. Flemons WW. Obstructive sleep apnea. N Engl J Med 2002;347:498-504.