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BRIEF REPORT

# Positive Airway Pressure Compliance in Patients with Epilepsy and Obstructive Sleep Apnea

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## Abstract

Obstructive sleep apnea (OSA) affects approximately one third of patients with epilepsy. Treatment with Continuous Positive Airway pressure (CPAP) is associated with improved seizure control, but is difficult for patients to tolerate. Patients with epilepsy and co morbid OSA may be at higher risk of recurrent seizures if they are noncompliant with CPAP. This study investigates short-term compliance with CPAP therapy in patients with OSA and epilepsy, as this can predict long-term adherence. Identifying factors influencing compliance rates in this population may help improve compliance and reduce seizure recurrence rate in this population. We also investigated the impact of epilepsy on sleep, and the possible effects this has on CPAP compliance.

## Keywords

Epilepsy, Obstructive Sleep Apnea

## Introduction

Obstructive sleep apnea (OSA) is a common disorder resulting in excess daytime sleepiness and decreased health related quality of life, and affects nearly one third of patients with epilepsy [1-3]. Proper and effective use of Continuous Positive Airway pressure (CPAP) can dramatically improve the health-related quality of life of patients with OSA, including boosting energy and productivity and decreasing systemic blood pressure [4].

However, CPAP therapy is often difficult for patients to tolerate, leading to poor compliance with treatment. Patients discontinue CPAP for a variety of reasons including discomfort, pressure sores, persistent air leakage, claustrophobia, and nasal congestion [5]. When non-compliance is defined as a mean of less than or equal to 4 hours of use per night, it is estimated that 29 to 83 percent of patients with OSA are non-compliant with CPAP [6]. This is particularly important to consider in patients with epilepsy and comorbid OSA, because it has been shown that treatment with CPAP has been associated with an improvement in seizure control in patients with epilepsy, and also that CPAP non-compliance in patients with epilepsy portends a higher risk of developing recurrent seizures [7-9]. In other words, in patients with epilepsy, CPAP noncompliance is a modifiable risk factor for seizure recurrence, and is therefore an important aspect of care for this patient population.

Short-term compliance with CPAP during the first few weeks of therapy has been strongly associated with better long-term compliance with CPAP therapy [6]. For this reason, short-term compliance with CPAP is a reasonable measure to use for comparison between groups of patients with and without epilepsy. Given the relatively small amount of data related to CPAP compliance in epilepsy patients, in this study we (1) Compared short-term CPAP compliance rates in patients with OSA and epilepsy to those with only OSA, and (2) Investigated the impact of epilepsy on sleep.

## Subjects and Methods

We retrospectively identified patients with moderate to severe OSA (AHI ≥ 15), who were started on nasal

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CPAP between 2012-2013 at the New York University Comprehensive Epilepsy Center, Sleep Center. We divided them into OSA-only (control) and epilepsy-OSA groups. Patients were excluded if they had a history of non-epileptic seizures, poor compliance with anti-epileptic drugs, greater than ten seizures a day, a diagnosis of epilepsy within the past six months, uncontrolled medical comorbidities and/or psychiatric disease, or a history of substance abuse, and if, for both groups, CPAP compliance rates were found to be less than ten percent. These criteria were chosen to minimize potential confounding variables. This retrospective study was approved by the New York University institutional research ethics board.

We analyzed demographic and clinical information including data from polysomnography (PSG) studies. Demographic variables included age, sex, body mass index (BMI), and Epworth Sleepiness Scale (a standardized self-rated scale used to measure daytime sleepiness in eight specific circumstances, with scores of 0-3 indicating the chances of dozing to sleep for each item, and a total score of 10 or more being suggesting of excessive daytime sleepiness). Clinical data from sleep studies were also evaluated, including variables assessing sleep architecture: Sleep efficiency (the percentage of total time spent in the sleep state while in bed), percent slow wave sleep (percent of total time during the sleep state spent in stage 3 sleep), percent of sleep spent in rapid eye movement (REM) sleep, spontaneous arousal index (the number of spontaneous arousals per hour, not attributable to respiratory events or periodic limb movements), optimal CPAP pressure (determined by in-lab titration), and Apnea Hypopnea Index (the mean number of apnea and hypopnea events per hour during sleep, or AHI). CPAP compliance, defined as percent of days with greater than 4 hours usage, was obtained via a monitoring SD card within the CPAP system. All patients with epilepsy that met inclusion criteria and comorbid OSA had one- and two-month follow up compliance data for analysis, and in the OSA-only group there were 26 patients with data for one- and two-month compliance rates for comparison out of 32 total who met criteria for inclusion. In comparing demographic and clinical variables, parametric tests (Chi-squared, *t*-tests) were used and reported as means with standard errors (SEs), and *p*-values with confidence intervals (CIs). *P*-values < 0.05 were considered significant.

## Results

There were 65 patients identified with epilepsy who underwent polysomnography evaluation for OSA between 2012-2013, of whom 41 were found to have concomitant OSA, and 14 who met pre-specified inclusion criteria. During the same time period, there were a total of 32 patients without epilepsy who also met the specific inclusion criteria, and of these, 26 had one- and two-month follow up data and were used as controls.

Patients who had epilepsy with comorbid OSA and patients with only OSA had comparable baseline characteristics, with the exception of age, as the group of patients with epilepsy were slightly younger on average (Table 1). In assessing the impact of epilepsy on sleep, sleep characteristics were similar between groups, although there was increased slow wave sleep in the epilepsy group compared to the OSA-only group. In comparing CPAP compliance rates between groups, there were no statistically significant differences in one- or two-month compliance rates (Table 2).

## Discussion

A number of factors influence CPAP compliance, including patient-related (i.e. patient education, social support system, etc.), therapy-related (i.e. side effects to device use, degree of efficacy), and health profes-

**Table 1:** Comparison of patient characteristics between patients with epilepsy-OSA and patients with OSA-only.

| | Epilepsy-OSA | OSA-only | *p*-value |
|---|---|---|---|
| Mean Age (SE) | 50.7 (4.2) | 59.9 (2.2) | 0.04 |
| Sex (% male) | 78.6 | 75 | 0.79 |
| Mean Body Mass Index (SE) | 32.3 (1.5) | 32.2 (1.1) | 0.96 |
| Mean Epworth Sleepiness Scale (SE) | 8.4 (1.5) | 9.8 (1) | 0.44 |
| Mean Spontaneous Arousal Index (SE) | 10 (2.3) | 7.7 (1.1) | 0.35 |
| Mean Sleep efficiency (SE) | 80.7 (4.4) | 79.2 (2.9) | 0.7 |
| Mean % Slow wave sleep (SE) | 15.7 (2.8) | 9.2 (2.2) | 0.04 |
| Mean % REM sleep (SE) | 9 (3.1) | 11.4 (2) | 0.55 |
| Mean Optimal CPAP pressure in cm $H_2O$ (SE) | 11.4 (0.6) | 10.7 (0.6) | 0.45 |
| Mean Apnea Hypopnea Index (SE) | 30.2 (6) | 41 (4.1) | 0.15 |

**Table 2:** One- and Two-month CPAP compliance rates between patients with epilepsy-OSA and OSA-only.

| | Epilepsy-OSA (n = 14) | OSA-only (n = 26) | *p*-value s(95% CI) |
|---|---|---|---|
| One month mean % compliance (n, SE) | 65.7 (14, 7.3) | 78.3 (26, 3.2) | 0.07 |
| Two months mean % compliance (n, SE) | 68.3 (14, 7.4) | 71.5 (26, 3.8) | 0.67 |

sional related factors (quality of physician-patient relationship). Additionally, comorbid medical conditions can contribute to noncompliance, and in this study we investigated the impact of comorbid epilepsy on CPAP compliance due to the fact that this non-compliance increases the risk of recurrent seizures [7-9].

Another important consideration is the impact of epilepsy on sleep, and possible consequences on CPAP compliance. OSA is prevalent in nearly a third of patients with epilepsy, and the relationship between OSA and epilepsy can be reciprocal. Sleep disorders can contribute to difficulty in managing seizures, while patients with epilepsy commonly report poor sleep quality, increased nocturnal awakenings, early morning awakenings, challenges with sleep initiation, and excessive daytime sleepiness. Through the use of combined electroencephalography and polysomnography (EEG-PSG), there has been support from studies indicating that both clinical seizures and interictal discharges produce sleep fragmentation, suppression of REM, and increases spontaneous arousals [10-12]. It is possible that undertreated epilepsy leads to a self-reinforcing cycle of sleep fragmentation, further predisposition to seizures, and further sleep loss. Besides the obvious sleep disruption from clinical seizures, sleep is also disrupted in patients with epilepsy on seizure-free nights as compared to nonepileptic controls. These patients were found to have lower sleep efficiencies, increase in sleep stage shifts, and periods of wakefulness when compared to normal controls. There were also more stage shifts, arousals, and less deep sleep in individuals with focal onset seizures than those with primary generalized epilepsies [10]. The pathophysiologic cause of epilepsy associated sleep disruption remains unclear, though multiple awakenings at night can potentially affect CPAP compliance.

This investigation shows that compliance rates were not significantly different in epilepsy patients with concomitant OSA compared with patients with only OSA in the first two months after beginning CPAP. Although there seemed to be slightly increased spontaneous arousals and decreased REM sleep in the epilepsy patients, the only significant difference in sleep was increased slow wave sleep compared to control patients. However, there was no difference in sleep efficiency. Possible confounders that may have influenced these findings included a somewhat decreased AHI in the epilepsy group, though this was not statistically significant, and potential effects of anti-epileptic medications on sleep architecture. Another limitation of this study is the wide range in CPAP compliance rates in general, which can be influenced by external variables such as level of patient education, counseling, and motivation, which could not be accounted for in this study.

The findings in this study show that in patients with OSA, there are similar compliance rates in patients who have epilepsy and those who do not. Subsequent studies with enrollment of more subjects and superior use of matched controls would need to be undertaken for further evaluation and to increase statistical power, though the implication of these findings is that sleep disruption in epilepsy patients is not related to CPAP compliance. Another consideration for further investigation would be to further stratify epilepsy and OSA patients by the frequency of interictal discharges or average number of nightly clinical events via combined electroencephalography- polysomnography. Given the effect sleep disruption can have on patients with epilepsy, providing early and aggressive support to ensure proper compliance to CPAP therapy remains an important aspect of care for patients with epilepsy.

## Author Contributions

Jacob Pellinen, MD – drafting and review of the manuscript, drafting of tables and figures Christopher K. Cheng, MD – acquisition and analysis of data, drafting and review of the manuscript.

Alcibiades J. Rodriguez, MD – conception and design of the study, drafting and review of the manuscript.

## Disclosure

The authors of this study have nothing to report. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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## References

1. Malow BA, Fromes GA, Aldrich MS (1997) Usefulness of polysomnography in epilepsy patients. Neurology 48: 1389-1394.

2. Malow BA, Levy K, Maturen K, Bowes R (2000) Obstructive sleep apnea is common in medically refractory epilepsy patients. Neurology 55: 1002-1007.

3. Manni R, Terzaghi M, Arbasino C, Sartori I, Galimberti CA, et al. (2003) Obstructive sleep apnea in a clinical series of adult epilepsy patients: frequency and features of the comorbidity. Epilepsia 44: 836-840.

4. Chokroverty S (2000) Editor's corner. Sleep Med 1: 257.

5. Uyar M, Elbek O, Mete A, Bayram N, Çifçi N, et al. (2012) Factors influencing compliance with continuous positive airway pressure ventilation in obstructive sleep apnea syndrome. Tuberk Toraks 60: 47-51.

6. Weaver TE, Kribbs NB, Pack AI, Kline LR, Chugh DK, et al. (1997) Night-to-night variability in CPAP use over the first three months of treatment. Sleep 20: 278-283.

7. Beran RG, Plunkett MJ, Holland GJ (1999) Interface of epilepsy and sleep disorders. Seizure 8: 97-102.

8. Malow BA, Foldvary-Schaefer N, Vaughn BV, Selwa LM, Chervin RD, et al. (2008) Treating obstructive sleep apnea in adults with epilepsy: a randomized pilot trial. Neurology 71: 572-577.

9. Vendrame M, Auerbach S, Loddenkemper T, Kothare S, Montouris G (2011) Effect of continuous positive airway pressure treatment on seizure control in patients with obstructive sleep apnea and epilepsy. Epilepsia 52: 168-171.

10. Culebras A (2010) Interface of sleep and neurologic disease. Rev Neurol Dis 7: 76-81.

11. Touchon J, Baldy-Moulinier M, Billiard M, Besset A, Cadilhac J (1991) Sleep organization and epilepsy. Epilepsy Res Suppl 2: 73-81.

12. Latreille V, Bubrick EJ, Pavlova M (2018) Positive airway pressure therapy is challenging for patients with epilepsy. J Clin Sleep Med 14: 1153-1159.