Peertechz

CLINICAL GROUP

ARCHIVES OF Otolaryngology and Rhinology OPEN ACCESS JOURNAL

ISSN: 2455-1759 DOI: https://dx.doi.org/10.17352/aor

Research Article

Sleep parameters during polysomnography and continuous positive airway pressure titration in severe obstructive sleep apnea adult patients

Utku Kubilay*

Department of Otorhinolaryngology, Ekol Hospital, Istanbul, Turkey

Received: 30 June, 2021
Accepted: 24 July, 2021
Published: 26 July, 2021

***Corresponding author:** Utku Kubilay, MD, Department of Otorhinolaryngology, Ekol Hospital, Istanbul, 8211/11 Sk.No:4 Daire 8, Izmir/Cigli, Turkey, Tel: +90 536 844 86 52;
E- mail: drutkukubilay@hotmail.com

Keywords: Obstructive sleep apnea syndrome; Continuous positive airway pressure; REM sleep; REM density

https://www.peertechzpublications.com

Abstract

Objectives: The aim of the present study was to demonstrate the effect of continuous positive airway pressure treatment on sleep parameters in severe obstructive sleep apnea patients.

Patients/methods: Data regarding apnea-hypopnea index, total sleep time, sleep efficiency, rapid-eye-movement (REM) density, REM latency, total REM episodes during polysomnography and continuous positive airway pressure titration according to the obstructive sleep apnea severity were compared.

Results: Of the 51 patients whose charts were reviewed, the average age was 46.47±10.62 years and the mean body mass index was 31.71±4.97 kg/m^2. Thirty-two patients who had an apnea-hypopnea index between 30 and 60/h included to the Group 1 and 19 patients who had an apnea-hypopnea index ≥60/h included to the Group 2. Among all studied parameters, only rapid-eye-movement latency showed statistical significance between the studied groups. Changes in rapid-eye-movement latency differed significantly among patients during polysomnography and continuous positive airway pressure titration in Group 2 (p=0.003). Indeed, these changes were higher in Group 2 patients with more severe obstructive sleep apnea compared to those with Group1 severe obstructive sleep apnea (p=0.003).

Conclusion: In conclusion, our investigations show that continuous positive airway pressure treatment results in a significant decrease in rapid-eye-movement latency among patients with more severe obstructive sleep apnea.

Introduction

Obstructive Sleep Apnea (OSA) is a serious condition characterized by loud snoring and frequent partial or complete blockage of the upper airway during sleep [1]. The Wisconsin Sleep Cohort Study showed the estimated prevalence of OSA to be 4-9% for women and 9-24% for men [2]. OSA is usually associated with daytime hypersomnolence, neurocognitive impairment, and development of cardiovascular disease [3].

First-line therapy for severe OSA is Continuous Positive Airway Pressure (CPAP) therapy, which was developed with the principle of preventing upper airway collapse using positive pressure as a splint [4]. Multiple effects of positive airway pressure treatment have been documented. One can classify possible benefits into four categories: (1) improvement of symptoms such as daytime sleepiness, disturbed sleep, impaired quality of life, or cognition; (2) reduced bed partner sleep disturbance or quality of life; (3) reduction of risk for cardiovascular disease, neurocognitive degeneration, or increased mortality associated with sleep apnea; and (4) reduction in the risk for motor vehicle accidents [5].

Following therapy with CPAP, most commonly reported changes are reduction of sleep latency, percentage of stage 1 sleep, arousal index and respiratory disturbance index, and

Citation: Kubilay U (2021) Sleep parameters during polysomnography and continuous positive airway pressure titration in severe obstructive sleep apnea adult patients. Arch Otolaryngol Rhinol 7(3): 019-022. DOI: https://dx.doi.org/10.17352/2455-1759.000141

increase in percentages of stage 2 sleep, Rapid-Eye-Movement (REM) sleep and an increase in frequency of eye movements during REM [6,7]. However, despite the high efficacy of CPAP in reversing upper airway obstruction in sleep apnea, treatment effectiveness on sleep characteristics is controversial. The aim of the present study was to demonstrate the effect of CPAP treatment on sleep parameters in severe OSA adult patients.

Methods

Study design

This study was approved by the local Institutional Review Board. Written informed consent was obtained from all subjects. Patients admitted to the Department of Otorhinolaryngology of our hospital, between September 2011 and July 2012 constituted the study group. Clinical examination included standardized scales for the assessment of respiratory symptoms and subjective daytime sleepiness. Body Mass Index (BMI) was calculated from body weight in kilograms divided by the square of height in metres. Patients with incomplete data, split night studies, failed pressure titration studies, those with a short Total Sleep Time (TST<200 min), the ones with underlying neurologic diseases, psychiatric pathology, and the ones taking psychoactive drugs were excluded.

Objective testing

The following physiologic variables were monitored simultaneously and continuously using Grass Comet instrument (Warwick, RI, USA): six channels for the electroencephalogram; two channels for the electro-oculogram; two channels for the surface electromyogram (submentonian region and anterior tibialis muscle); one channel for an electrocardiogram; airflow detection via two channels through a thermocouple (one channel) and nasal pressure (one channel); respiratory effort of the thorax (one channel) and of the abdomen (one channel) using plethysmography; snoring (one channel) and body position (one channel); oxyhemoglobin saturation; and pulse rate.

As for the overnight titration, starting with the lowest tolerated pressure (4 cmH_2O), increments of 1 cmH_2O were added for obstructive apnoea events or hypopnoea preceding EEG arousals. An effective CPAP was reached when respiratory events, desaturation and respiratory event-related arousals were eliminated. The therapeutic CPAP was determined by a sleep physician based on the pressure that controlled obstructive events during supine REM sleep. If supine REM sleep was not sampled, the pressure required to control respiratory events during non-supine REM sleep was determined. In instances where the CPAP that controlled respiratory events during REM sleep did not exceed that for non-REM sleep, then the therapeutic non-REM CPAP level was recommended.

Two trained technicians visually scored all Polysomnography (PSG) recordings according to standardized criteria for investigating sleep [8]. Apnea was defined as complete cessation of airflow for at least 10 seconds; hypopnea was defined as a ≥50% reduction in airflow that was associated with at least a ≥3% drop in O_2 saturation or arousal. The Apnea-Hypopnea Index (AHI) was calculated as the total number of apneas and hypopneas per hour of sleep. OSA was defined by AHI ≥ 5/h. The subjects were categorized into Group 1 (30<AHI<60) and Group 2 (60≤AHI) subgroups according to the AHI severity.

Outcome parameters

AHI, total sleep time, sleep efficiency, REM density, REM latency, total REM episodes during PSG and CPAP titration according to the OSA severity were recorded for each patient.

Statistical analysis

NCSS (Number Cruncher Statistical System), 2007&PASS (Power Analysis and Sample Size), 2008 Statistical Software (Utah, USA) were used for statistical analysis. During the assessment of the study data, we investigated the distribution of categorical measurements according to the frequency and percentages, while we described our numerical parameters with mean and standard deviations. Student t test was applied to data of normal distribution and Mann Whitney U test was applied to data of questionably normal distribution. For within-group analysis, the paired t-test was used for normal data and the Wilcoxon signed-ranks for non-normal data. All differences associated with a chance probability of .05 or less were considered statistically significant.

Results

Fifty-one patients met the eligibility criteria for the study. Of the 51 patients (37 males, 14 females) whose charts were reviewed, the average age was 46.47±10.62 (range 25 to 67) years, and the mean BMI was 31.71±4.97 (range 22.75 to 47.50) kg/m^2. Concerning all patients in the study group, nocturnal PSG disclosed a mean AHI of 53.42±18.69 episodes (range 30 to 101) per hour.

Thirty-two patients had an AHI between 30 and 60/h, and 19 patients had an AHI ≥60/h. Demographic characteristics of the study group according to the severity of the OSA were shown in Table 1. Age, gender, and BMI did not differ between the groups (p=0.549, p=0.173, and p=0.343, respectively).

Data regarding AHI, total sleep time, sleep efficiency, REM density, REM latency, total REM episodes during PSG and CPAP titration according to the OSA severity were summarized in Table 2. Among all studied parameters, only REM latency showed statistical significance between the studied groups. Changes in REM latency differed significantly among patients during PSG and CPAP titration in Group 2 patients (p=0.003). Indeed, Δ REM latency levels (changes in REM latency) were higher in Group 2 patients compared to Group 1 patients (p=0.003).

Table 1: Demographic characteristics of recruited subjects by OSA severity.

	30<AHI<60 (n=32)	AHI≥60 (n=19)	p Value
Age (yr)	46.53±10.06	46.37±11.78	0.549
Gender (F:M)	10:22	4:15	0.173
BMI (kg/m^2)	30.89±4.40	33.16±6.48	0.343

AHI = Apnea-Hypopnea Index defined as the number of apneas and hypopneas per hour of sleep; BMI = Body Mass Index (kg/m2); OSA = Obstructive Sleep Apnea. Data are reported as mean±SD or as number and percentage

Citation: Kubilay U (2021) Sleep parameters during polysomnography and continuous positive airway pressure titration in severe obstructive sleep apnea adult patients. Arch Otolaryngol Rhinol 7(3): 019-022. DOI: https://dx.doi.org/10.17352/2455-1759.000141

Table 2: Sleep characteristics of recruited subjects by OSA severity.

		30<AHI<60 (n=32)	AHI≥60 (n=19)	*p* Value
AHI		40.15±8.61	75.16±11.15	0.001
Total sleep time	PSG	348.73±45.44	362.10±49.64	*0.331*
	CPAP titration	328.28±42.65	338.07±49.71	*0.460*
	p Value	*0.051*	*0.126*	
Δ total sleep time		20.03±57.04	24.03±65.28	*0.755*
Sleep efficiency	PSG	82.69±10.09	84.24±10.63	*0.606*
	CPAP titration	80.88±11.15	82.29±10.92	*0.663*
	p Value	*0.353*	*0.488*	
Δ sleep efficiency		1.81±10.86	1.95±12.01	*0.740*
REM density	PSG	3.77±1.97	3.48±2.16	*0.386*
	CPAP titration	3.92±1.61	5.11±4.41	*0.961*
	p Value	*0.724*	*0.107*	
Δ REM density		0.15±1.98	1.63±3.45	*0.199*
REM latency	PSG	115.06±77.12	169.84±90.71	*0.026*
	CPAP titration	125.75±99.00	88.47±43.73	*0.071*
	p Value	*0.528*	*0.003*	
Δ REM latency		10.70±94.87	81.38±101.31	*0.003*
Total REM episodes	PSG	3.06±1.21	2.73±1.28	0.441
	CPAP titration	2.78±1.01	3.15±0.69	0.216
	p Value	0.163	0.301	
Δ total REM episodes		0.28±1,27	0.42±1.57	0.066

AHI = Apnea-Hypopnea Index defined as the number of apneas and hypopneas per hour of sleep; ESS = Epworth Sleepiness Scale; PSG = Polysomnography; CPAP = Continuous Positive Airway Pressure. Data are reported as mean±SD or as number and percentage

Discussion

The purpose of this study was to evaluate the performance of CPAP treatment on sleep parameters in severe OSA adult patients. Among the studied parameters, REM latency showed statistical significance between the studied groups.

The positive effects of CPAP on sleep are well-known in the literature [7]. However, despite the dramatic changes that occur before and after CPAP in sleep-disordered breathing, several reports have ascertained the lack of strong relationships between conventional sleep measures and daytime function in patients with OSA [9].

When one considers the symptomatic consequences of sleep disruption, sleepiness is the most obvious, but not the only, outcome. In seminal studies carried out by Clemes and Dement, subjects deprived selectively of REM sleep for six nights showed changes in psychological testing suggesting a higher intensity of feeling and need [10]. That REM sleep plays a role in mood and its dysfunction is supported by the association between depression and a reduced REM sleep latency [11]. Furthermore, depression is seen with increasing frequency in persons suffering from OSA [12]. In cases of OSA where REM is decreased and subsequently rebounds with CPAP exposure, it could be an elevation in mood.

Although most patients report subjective improvement in sleep quality after the CPAP titration study, many do not. In untreated OSA, cortical arousal is often necessary to reopen the obstructed airway and to restore breathing. By restoring normal breathing, CPAP reduces arousal and permits normal sleep cycling. On initial exposure, CPAP can precipitate an increase or rebound in either REM or slow wave sleep and a decrease in stage N1 sleep [6]. This rebounding of REM and slow wave sleep during an initial night of in-laboratory CPAP titration has been shown to correlate with improvement in subjective sleep quality [13]. Some studies have shown that REM increases to a greater extent than slow wave sleep in response to first CPAP exposure and, suggesting that REM restoration may play a more significant role in sleep quality improvement [6,14]. Issa, et al. investigated conventional sleep patterns in the first and third night of CPAP treatment. During the first night, there was a rebound of slow wave sleep and REM sleep, but treatment had no effect on the time spent in wakefulness [7]. One study stated that REM latency decreased on second and third nights after CPAP, possibly contributing to the earlier results for REM latency [15]. In some severely sleepy OSA patients, there may be a higher-than-normal amount of stage 3,4 and/or stage REM sleep (rebound) on the initial night of CPAP titration. In less sleepy patients, this may not occur possibly due to difficulties adapting to monitoring equipment or the first exposure to CPAP. McArdle, et al. demonstrated an increase in stage 3,4 sleep and a decrease in stage 1 sleep after 1 month of CPAP treatment; in this study, the amount of REM sleep did not increase [16]. In the present study, a significant decrease was shown in REM latency among patients with more severe OSA. However, total REM episodes did not show any statistical difference neither between PSG and CPAP parameters, nor between groups.

Three experimental studies obtained results consistent with the existence of an inverse relationship between sleep need and REM density. A significant reduction of REM density was found by Feinberg, et al. and by Travis, et al. [17,18] In both studies the reduction of REM density was to a lesser degree than total sleep deprivation. Barbato, *et al.* reported that, in extended sleep, the REM periods terminating in wakefulness showed higher REM densities than those which did not terminate in wakefulness and concluded that REM density is indicative of lesser sleep depth [19]. In the present study, an increase in REM density, which did not reach a significant level, was detected especially in Group 2 patients with more severe OSA.

Arzt, *et al.* revealed that all sleep parameters including total sleep time, sleep efficiency, percentage of slow-wave sleep, and percentage of rapid eye movement sleep obtained by PSG increased after initiation of CPAP but this increment did not reach a significant level [20]. In the present study, total sleep time and sleep efficiency decreased with CPAP titration; however similar with Arzst's study, this decrease did not show any statistical significance.

Our study has several limitations. The main limitations are the retrospective design and relatively small number of our series recruited from a single sleep center. Some details of history and factors that may influence the outcome may not be completely documented. The possible contribution of alcohol and hypnotic drugs to REM sleep was not specifically analyzed. The patients were awakened at the end of the allotted time as the PSG studies are performed during fixed hours in our sleep lab. Finally, the "first night effect" for diagnostic PSG due to the patient sleeping in an unfamiliar environment with sleep study equipment, and application of a mask for the pressure

Citation: Kubilay U (2021) Sleep parameters during polysomnography and continuous positive airway pressure titration in severe obstructive sleep apnea adult patients. Arch Otolaryngol Rhinol 7(3): 019-022. DOI: https://dx.doi.org/10.17352/2455-1759.000141

titration on the CPAP titration night would have affected the sleep quality. Due to these restrictions, associations should be interpreted with caution.

Conclusion

In conclusion, our investigations show that CPAP treatment results in a significant decrease in REM latency among patients with more severe OSA.

References

1. Ioachimescu OC, Collop NA (2012) Sleep-disordered breathing. Neurol Clin 30: 1095-1136. Link: https://bit.ly/3y5jlPz
2. Young T, Palta M, Dempsey J, Skatrud J, Weber S, et al. (1993) The occurrence of sleep disordered breathing among middle-aged adults. N Engl J Med 328: 1230-1235. Link: https://bit.ly/3x9z8Rz
3. Zhang W, Si LY (2012) Obstructive sleep apnea syndrome (OSAS) and hypertension: pathogenic mechanisms and possible therapeutic approaches. Ups J Med Sci 117: 370-382. Link: https://bit.ly/3j3pXNV
4. Sullivan CE, Issa FG, Berthon-Jones M, Eves L (1981) Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1: 862-865. Link: https://bit.ly/3zxljgs
5. Simon S, Collop N (2012) Latest advances in sleep medicine: obstructive sleep apnea. Chest 142: 1645-1651. Link: https://bit.ly/3eYp7jY
6. Aldrich M, Eiser A, Lee M, Shipley J (1989) Effects of continuous positive airway pressure on phasic events of REM sleep in patients with obstructive sleep apnea. Sleep 12: 413-419. Link: https://bit.ly/3kV6UaN
7. Issa FG, Sullivan CE (1986) The immediate effects of nasal continuous positive airway pressure treatment on sleep pattern in patients with obstructive sleep apnea. Electroenceph Clin Neurophysiol 63: 10-17. Link: https://bit.ly/3kVZSmc
8. Rechtschaffen A, Kales A (1968) A manual of standardized terminology: techniques and scoring system for sleep stages of human subjects. Los Angeles: Brain Information Service/Brain Research Institute. Link: https://bit.ly/2Vcu55C
9. McNicholas WT (1998) Arousal in the sleep apnoea syndrome: a mixed blessing? Eur Respir J 12: 1239-1241. Link: https://bit.ly/3i4dtpJ
10. Clemes SR, Dement WC (1967) Effect of REM sleep deprivation on psychological functioning. J Nerv Ment Dis 144: 485-491. Link: https://bit.ly/3rATu5p
11. Kupfer DJ (1976) REM latency: a psychobiologic marker for primary depressive disease. Biol Psychiatry 11: 159-174. Link: https://bit.ly/3kWfl62
12. Reynolds CF, Coble PA, Spiker DG, Neil JF, Holzer BC, et al. (1982) Prevalence of sleep apnea and nocturnal myoclonus in major affective disorders: clinical and polysomnographic findings. J Nerv Ment Dis 170: 565-567. Link: https://bit.ly/37122ZI
13. Verma A, Radtke RA, VanLandingham KE, King JH, Husain AM (2001) Slow wave sleep rebound and REM rebound following the first night of treatment with CPAP for sleep apnea: correlation with subjective improvement in sleep quality. Sleep Med 2: 215-223. Link: https://bit.ly/3eVuOPO
14. Espinar Sierra J (1989) Immediate effect of nasal continuous positive pressure in the treatment of obstructive sleep apnea. Rev Clin Esp 184: 69-74. Link: https://bit.ly/3BlaafS
15. Altman DG (1991) Clinical trials. Practical statistics for medical research. London: Chapman & Hall 467-471.
16. McArdle N, Douglas NJ (2001) Effect of continuous positive airway pressure on sleep architecture in the sleep apnea-hypopnea syndrome. Am J Respir Crit Care Med 164: 1459-1463. Link: https://bit.ly/3iOuC5T
17. Feinberg I, Baker T, Leder R, March JD (1988) Response of delta (0-3 Hz) EEG and eye movement density to a night with 100 minutes of sleep. Sleep 11: 473-487. Link: https://bit.ly/3x5W18s
18. Travis F, Maloney T, Means M, March JD, Feinberg I (1991) Acute deprivation of the terminal four hours of sleep does not increase delta (0-3-Hz) electroencephalograms: a replication. Sleep 14: 320-324. Link: https://bit.ly/3x7RYIZ
19. Barbato G, Barker C, Bender C, Giesen HA, Wehr TA (1994) Extended sleep in humans in 14 hour nights (LD 10:14): relationship between REM density and spontaneous awakening. Electroencephalogr Clin Neurophysiol 90: 291-297. Link: https://bit.ly/3x6WQxQ
20. Arzt M, Wensel R, Montalvan S, Schichtl T, Schroll S, et al. (2008) Effects of dynamic bilevel positive airway pressure support on central sleep apnea in men with heart failure. Chest 134: 61-66. Link: https://bit.ly/3eQS1Th

Citation: Kubilay U (2021) Sleep parameters during polysomnography and continuous positive airway pressure titration in severe obstructive sleep apnea adult patients. Arch Otolaryngol Rhinol 7(3): 019-022. DOI: https://dx.doi.org/10.17352/2455-1759.000141