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Original Scientific Paper

INFLUENCE OF CONTINUOUS POSITIVE AIRWAY PRESSURE TREATMENT ON AUDITORY EVENT-RELATED POTENTIALS P300

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SUMMARY – Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by obstructions of the upper airway during sleep, resulting in repetitive breathing pauses accompanied by oxygen desaturation and arousal from sleep. OSA can be successfully treated with continuous positive airway pressure (CPAP), weight loss, positional therapy, oral appliances, hypoglossal nerve stimulation, and surgical procedures. It has been observed that untreated OSA is related to chronic disorders including hypertension, arrhythmias, congestive heart failure, coronary heart disease, diabetes mellitus, hyperlipidemia, stroke, depression, and cognitive decline. Event-related potentials (ERPs) is the procedure that has been widely used for evaluating cognitive brain functions. Using auditory event-related potentials (P300), this study aimed to examine the effect of CPAP therapy on cognitive functions in patients with moderate and severe OSA. The results of the study showed improvement in P300 latency and amplitude after 3 months of CPAP therapy, indicating a positive effect of CPAP therapy in the prevention of cognitive decline in patients with OSA.

Key words: *obstructive sleep apnea; continuous positive airway pressure; auditory event-related potentials; P300*

Introduction

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by obstructions of the upper airway during sleep, resulting in repetitive breathing pauses accompanied by oxygen desaturation and arousal from sleep[1]. Clinical features of OSA include snoring, witnessed apnea, excessive daytime sleepiness, morning headache, and fatigue. Treatment of OSA includes continuous positive airway pressure (CPAP), weight loss, positional therapy, oral appliances, hypoglossal nerve stimulation, and surgical procedures[2]. Untreated OSA is related to hypertension, arrhythmias, congestive heart failure, coronary heart disease, diabetes mellitus, hyperlipidemia, stroke, depression, and cognitive decline[3-6]. OSA and cognitive functions have been investigated in multiple studies which have reported a certain level of cognitive dysfunction in patients with OSA[7-14].

Event-related potentials (ERPs) have been used for studying cognitive brain functions. Sutton et al. first reported that the P300 component of ERPs is re-

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lated to the reaction of the subject to the stimulus and found that ERPs are associated with cognitive functions[15]. The P300 wave is a late component of ERP which occurs approximately 300 milliseconds (ms) after the stimulus. The P300 wave is characterized by its latency and amplitude. P300 latency represents the time needed to process information, and P300 amplitude is related to the allocation of attention by the subject[16]. The oddball paradigm is used to record the wave. The paradigm can be administered in visual or auditory form, but the auditory form is more commonly used than the visual one. It involves two different stimuli: frequent and less-frequent presentation of the target stimuli and frequent presentation of the non-target stimuli[17]. P300 is widely used in studies as an electrophysiological correlation to the cognitive deficit as well as to assess cognitive functions such as memory, attention, concentration, and mental processing. ERP findings in patients with OSA are controversial. Some studies showed no change in P300 latency and amplitude while others found prolonged latency and reduced amplitude in patients with OSA in comparison with healthy controls[8,10,11,13,18]. It has been observed that CPAP therapy may improve cognitive functions, but not all studies reported this benefit[19-27].

In the present study, we evaluated cognitive functions by using ERPs P300 in patients with moderate and severe OSA before and 3 months after continuous CPAP therapy.

Methods

In this single-center prospective study, we enrolled 54 adult patients (age range 26-65 years) with a diagnosis of moderate to severe OSA (defined as an apnea-hypopnea index; AHI ≥15). In all patients, OSA was diagnosed according to the standard polysomnography (PSG) criteria. All of the enrolled subjects received CPAP therapy and agreed to participate in a 3-months follow-up.

Inclusion criteria comprised age 18-65 years, newly diagnosed OSA, and good CPAP compliance after 3 months (defined as usage of CPAP device for >4 hours per night on at least 70% of nights). Exclusion criteria comprised previously diagnosed neurological or psychiatric disease, use of drugs known to affect sleep or daytime sleepiness, uncontrolled high blood pressure, any type of hearing loss, previous treatment of OSA with CPAP, oral appliances or surgery, and poor CPAP compliance after 3 months.

All patients underwent full-night PSG in the Sleep Laboratory at the Clinic of Neurology (University Hospital Center Osijek). PSG was performed using the Alice 6 polysomnograph (Philips Respironics, Murrysville, PA, USA). Standard surface electrodes were used to record electroencephalographic, electrooculographic, electromyographic, and electrocardiographic data. Airflow was monitored using an oronasal thermistor. Thoracic and abdominal movements were monitored using plethysmography belts. Arterial oxyhemoglobin saturation was measured with a finger pulse oximeter. The studies were manually scored according to the recommendations of the latest version of the American Academy of Sleep Medicine scoring manual[28]. Excessive daytime sleepiness was evaluated using the Epworth Sleeping Scale (ESS). To exclude the presence of any type or degree of hearing loss, the audiological evaluation was performed at the Department of Otorhinolaryngology and Head and Neck Surgery (University Hospital Center Osijek). Standard pure tone audiometry was performed using an audiometer GSI AudioStar Pro (7625 Golden Triangle Drive, Suite F, Eden Prairie MN 55344, USA). Acoustic reflex thresholds were tested with contralateral stapedius reflexes for frequencies of 500, 1000, 2000, and 4000 Hz and considered to be normal when the thresholds were between 70 and 90 dB above the pure tone threshold.

Auditory event-related potentials (aERP) recordings were performed before and 3-months after CPAP therapy in a silent room in a sitting position using the 5-channel device Medelec Synergy EMG/EP, VIASYS Healthcare Inc. NeuroCare Group, 5225-2 (Verona Rd., Madison, WI 53711, USA). The international 10-20 system was used for electrode placement with impedance less than 5 KΩ. Stimuli were delivered via headphones binaurally with 65-dB sound pressure level intensity. Electrical signals were filtered with 40 Hz high-pass and 0.1 Hz low-pass filters. A 700 ms time window was used. Recordings were performed using frontal (Fz), parietal (Pz), and vertex (Cz) Ag/AgCl electrodes. The referral electrode was the right mastoid (M2). The non-target (regular) tone (1000 Hz) was present during 80% of the total stimulation time, while the target (irregular) tone (2000 Hz) occured 20% of the total stimulation time. Patients were instructed to discriminate between target and non-target stimuli, count irregular tones, and report their results to a trained technician. Two parameters of P300

were measured: P300 amplitude (in microvolts; μV) which was defined as the potential difference between the baseline and the peak of the positive wave, and P300 latency (in milliseconds; ms) which was defined as the period between the stimulus onset and the wave apex.

Ethics

The study was approved by the institutional ethics committee, and all of the participants provided informed consent.

Statistics

Descriptive statistics were calculated to examine the levels of demographic variables, body mass index, sleep, and AHI. All values are expressed as mean ± standard deviation (SD). Frequency and percentage of gender and levels of sleep were calculated. The normality of the distribution of the observed numerical variables was tested by the Kolmogorov-Smirnov test. The t-test was used to examine whether the amplitude and latency of the P300 wave were different before and after the application of the CPAP therapy. The level of significance was 0.05. The statistical program SPSS (21.0, SPSS Inc, Chicago, IL, USA) was used for statistical analysis.

Results

The study included 54 patients with newly diagnosed OSA (42 men, 12 women). The mean age of the patients was 50.46±10.30 years. The mean body mass index was 36.42±8.44 kg/m^2, while the mean ESS was

Table 1. Demographic data and clinical characteristics

Variable	**Result (mean, SD)**
Patients/sex, n	54 (42 men, 12 women)
Age, years	50.46±10.30
BMI, kg/m^2	36.42±8.44
ESS	10.89±4.80
AHI events/h	57.21±17.82

Data are expressed as the mean ± standard deviation (SD);
BMI = body mass index
AHI = apnea/hypopnea index
ESS = Epworth Sleepiness Scale score

Table 2. P300 latency before and after 3 months of CPAP use

P300 latency (ms)	Mean ± SD	Standard error	t-test	df	p-value
Before CPAP	341.31± 25.199	3.429			
After CPAP	315.28±20.466	2.785	7.415	53	0.001

ms = milliseconds
SD = standard deviation
df = degrees of freedom

Table 3. P300 amplitude before and after 3 months of CPAP use

P300 amplitude (μV)	Mean ± SD	Standard error	t-test	df	p-value
Before CPAP	10.206± 5.500	.7485			
After CPAP	11.043±4.395	.5981	-1.232	53	0.224

μv = microvolts
SD = standard deviation
df = degrees of freedom

10.89±4.80. The PSG of all patients showed OSA with a mean AHI of 57.21±17.82/h (Table 1.) Results of the P300 latency before and after 3 months of CPAP use are shown in Table 2. The mean latency of P300 before CPAP therapy was 341.31±25.199 ms, and the standard error of this mean was 3.429. The mean latency of P300 after 3 months of CPAP therapy was 315.28±20.466 ms, and the standard error of this mean was 2.785. The difference was statistically significant (t=7.415, df=53. p<0.001) (Table 2.) The mean amplitude of P300 before CPAP therapy was 10.206±5.500 μV, and the standard error of this mean was 0.7485. The mean amplitude after 3 months of CPAP therapy was 11.043±4.395 μV, and the standard error of this mean was 0.5981. The difference in the P300 amplitude before and 3 months after CPAP therapy was not statistically significant (t= -1.232, df=53, p>0.05) (Table 3).

Discussion

Previous studies have shown that OSA is associated with cognitive deficit and that it causes impairments in attention, vigilance, memory, executive functions, visuospatial functions, and psychomotor performance[7,29-35]. Cognitive impairment in patients with OSA is a consequence of neurophysiological changes and is characterized by prolonged P300 latency and reduced amplitude. P300 amplitude is related to the allocation of attention by the subject, and changes in amplitude correlate negatively with target stimulus frequency and positively with the difficulty of the task[36]. P300 latency is related to stimulus classification speed, and P300 changes correlate positively with age and negatively with attention[36].

Studies that evaluated cognitive functions in patients with OSA through ERPs reported prolonged P300 latency and lower amplitudes in patients with OSA in comparison with healthy controls[8-10,13,37,38]. Sangal et al. found that patients with OSA had prolonged P300 latency in comparison with healthy controls[11,12]. Gelir et al. investigated the effects of OSA on cognitive functions such as learning, attention, and memory in 15 patients with OSA and compared them with 15 healthy controls. Results showed prolonged P300 latency and reduced P300 amplitude in the OSA group compared with the control group[10]. Similar results were found by Vakulin et al., who found a significant difference in the P300 latency between patients with OSA and healthy controls. In the same study, the difference in P300 amplitude was not statistically significant, but P300 amplitude was reduced in patients with OSA in comparison with the control group[39]. Yerlikaya et al. investigated cognitive impairment in 54 patients with severe OSA in comparison with 34 healthy controls matched for age, gender, and education. Their results showed lower P300 amplitudes in patients with OSA compared with the control group[13].

Previous studies have shown the benefits of CPAP treatment on cognitive functions in patients with OSA[22,24,25,27,40-43]. Ak et al., in their study on 54 patients with OSA, reported significant improvement in P300 latency and amplitude after 3 months of CPAP therapy[44]. In a study on 20 patients with OSA, El Gharib et al. investigated the influence of CPAP on cognitive functions. The results of that study showed a significant difference in latency and amplitude before and after CPAP use[14].

In the present study, initial results of ERPs/P300 in all patients revealed prolonged latency and lower amplitude. After 3 months of continuous CPAP therapy, all patients showed improvement in both P300 components. P300 latency was statistically significantly shorter after 3 months of CPAP therapy, and P300 amplitude was increased but without statistical difference in the results. It has been observed that P300 amplitude is reduced with increasing age[45]. In their study on patients with OSA, Akcali et al. investigated differences in P300 components according to age (<45 years vs. ≥45 years)[8]. P300 latencies were significantly longer in both younger and older OSA patients compared with their age-matched control groups. However, a P300 amplitude decrease was observed in younger OSA patients, but there was no observed difference in older OSA patients compared with healthy age-matched controls. The mean age of subjects included in this study was 50.46±10.30 years. Thus, the lack of statistical difference in P300 amplitude in our study could be partially explained by the previously observed impact of age on this P300 component.

The main limitation of our study was the small sample size. Nonetheless, our results are similar to the results of Akcali et al., who performed a similar study on a larger number of participants[8].

In conclusion, our study showed that the regular use of CPAP in patients with OSA reduces P300 latency and increases P300 amplitude, indicating the positive impact of this type of OSA therapy on cognitive impairment.

Conflict of interest:

The authors declare no conflict of interest.

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Sažetak

UTJECAJ TERAPIJE KONTINUIRANIM POZITIVNIM TLAKOM U DIŠNIM PUTEVIMA NA KOGITIVNE EVOCIRANE POTENCIJALE P300

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Opstruktivna apneja u spavanju (OSA) je poremećaj disanja u spavanju karakteriziran opstrukcijom gornjih dišnih puteva tijekom spavanja koji dovodi do prestanka disanja u spavanju, desaturacije krvi kisikom i buđenja. OSA se uspješno liječi uređajima za potpomognuto disanje s neprekinutim pozitivnim tlakom (CPAP), gubitkom tjelesne težine, pozicijskom terapijom, oralnim udlagama, stimulacijom živca hipoglosusa i operativnim liječenjem. Opaženo je da je neliječena OSA povezana s kroničnim poremećajima, uključujući arterijsku hiperteziju, aritmije, kongestivno zatajenje srca, koronarnu srčanu bolest, šećernu bolest, hiperlipidemiju, moždani udar, depresiju i kognitivno propadanje. Kognitivni evocirani potencijali se koriste za proučavanje kognitivnih funkcija. Cilj ovog rada je ispitati učinak terapije CPAP-om na kognitivne funkcije bolesnika s umjerenom i teškom OSA – om koristeći slušne kognitivne evocirane potencijale. Rezultati ove studije su pokazali poboljšanje P300 latencija i amplituda, ukazujući na pozitivni učinak terapije CPAP – om u prevenciji kognitivnog poremećaja u bolesnika s OSA – om.

Ključne riječi: *opstruktivna apneja u spavanju; disanje s neprekinuitim pozitivnim tlakom; slušni kognitivni evocirani potencijali; P300*