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The effect of obstructive sleep apnea syndrome on the central auditory system

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Background/aim: Obstructive sleep apnea syndrome (OSAS) is a disease characterized by repeated hypoxia attacks during sleep. The effect of hypoxia on the central nervous system is a well-known entity. In this study we aimed to investigate the effect of OSAS on the central auditory system.

Materials and methods: Twenty-one OSAS patients diagnosed by polysomnography (PSG) and 10 control subjects were included in the study. After a thorough otorhinolaryngology examination, all subjects underwent pure tone audiometry (250 to 8000 Hz frequency). The subjects with normal otoscopic examination and hearing threshold were included in the study. All participants underwent speech discrimination analyses and auditory time processing and sequencing tests, i.e. frequency pattern test (FPT) and duration pattern test (DPT).

Results: Although hearing was normal in the OSAS patients, significant loss was observed in the speech discrimination rates compared to the control group ($P < 0.05$). Significant disruption was also detected in the FPT and SPT in the OSAS patients ($P < 0.05$).

Conclusion: Repeated hypoxic episodes in OSAS resulted in statistically significant impairments in the central auditory pathways, even if the hearing threshold was within normal limits.

Key words: OSAS, central auditory system, hypoxia

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is a disease characterized by repeated apnea and hypoxia attacks occurring during sleep. The prevalence of OSAS ranges between 2% and 4% in the general population. Recurrent sleep disturbances cause increased daytime sleepiness, concentration difficulty, and impaired attention and learning (1). Hypoxic attacks have been shown to cause reduction in neurocognitive functions secondary to neuronal damage and atrophy at the brain cortex and hippocampus (2). Moderate-to-severe OSAS is known to have negative effects particularly on alertness, attention, and concentration (3,4).

The apnea attacks in OSAS can lead to acute ischemic changes in cerebral blood circulation and the cochlea (5,6). The inner ear, cochlea, and acoustic nerve are very sensitive to hypoxia since they receive blood supply from terminal arteries without collateral circulation (7–9).

There are several papers in the literature investigating the relationship between OSAS and hearing impairment (10). The level of pathology in the central auditory pathways has not yet been fully elucidated. In particular, studies addressing central processing and discrimination are insufficient.

Pure tone audiometry and speech audiometry are routine audiological evaluations used to identify the hearing status of an individual. These tests measure the sensitivity of the central and peripheral auditory system. Auditory temporal processing and sequencing tests are used to evaluate temporal analysis, temporal order, and frequency and duration of hearing, which are related to the central auditory system. Of these tests, the frequency pattern test (FPT) and duration pattern test (SPT) are sensitive to central cortex and corpus callosum dysfunction (10).

In the present study we aimed to investigate the effect of OSAS on the central auditory system with routine pure tone and speech audiometry tests and auditory temporal processing and sequencing tests.

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2. Materials and methods

The study was approved by the local ethics committee. All the participants filled out and agreed on the informed consent form. Twenty-one OSAS patients who underwent a full-night polysomnography (PSG) test in the sleep laboratory of our hospital were included in the study. The control group consisted of 10 healthy subjects without OSAS symptoms such as snoring, witnessed apnea, and daytime extreme sleepiness. Any individual suspected of having OSAS was excluded from the control group. Subjects suffering hearing loss or any otorhinolaryngologic disease (otitis media, eustachian tube dysfunction, nasal obstruction, etc.) that may cause hearing loss were excluded from the study. Likewise, the participants with other accompanying diseases like hypertension, diabetes mellitus (DM), and chronic obstructive pulmonary disease, which may cause hypoxia and microvascular circulation problems in the central auditory centers, were excluded from the study.

2.1. Audiological evaluation

All the subjects were subjected to pure tone audiometry (250 to 8000 Hz frequencies) and speech audiometry tests by the same staff. Hearing thresholds were determined as being pure tone averages of 500, 1000, 2000, and 4000 Hz frequencies. The speech discrimination rates were calculated as well. The subjects with normal hearing with pure tone averages of less than 25 were included the study. Speech discrimination rate was determined from all patients and controls by the same staff in 100 words. Then FPT and DPT from nonword auditory tests were performed.

The FPT measures frequency sense and the ability to sort it. For this reason, it has an important role in the ability to understand and produce speech. According to Musiek, two pure sounds with frequencies of 880 Hz and 1122 Hz are used in this test. It is requested from the participant to sort the sounds according to the order of arrival (such as thin–thick–thick) by distinguishing the difference between the frequencies. In our study, 30 consecutive sounds were presented to the participants. We wanted them to sort those sounds by frequency and we recorded their correct answers (11).

The DPT measures the ability to distinguish the time difference between sounds and to sort it. In this test, 2 sounds with a frequency of 1000 Hz and durations of 250 ms and 500 ms are used. Participants are asked to distinguish the duration of the stimulus and to sort by arrival sequence (such as long–long–short). In our study, we presented 30 consecutive sounds to the patients by 300 ms intervals. We wanted them to sort those sounds by arrival sequence and we recorded their correct answers (12).

2.2. Polysomnography

A complete PSG test was performed in the OSAS patients. Eight-channel EEG, EMG, EOG, ECG, pulse oximeter, flowmeter, etc. records were taken through the all night polysomnographic examination with the PSG device (Grass Technologies Twin Version 4.5-3.25, USA). Patients with moderate-to-severe OSAS (apnea hypopnea index (AHI) ^{3} 15) were included in the study. The average oxygen saturation scores and desaturation percentages were recorded in the PSG test along with the AHI.

2.3. Statistical analysis

Statistical analysis was performed using SPSS 20.0 for Windows (SPSS Inc., Chicago, IL, USA). The descriptive data were presented as mean ± standard deviation. The independent samples t test was used to compare means between the groups. A P value less than 0.05 was considered statistically significant.

3. Results

There were 12 male and 9 female patients in the OSAS group. The mean age was 47.1 ± 8.2 years. The control group consisted of 5 male and 5 female subjects. The mean age in the control group was 43.6 ± 3.8 years. The mean BMI was 30.9 ± 2.9 and 26.9 ± 5.5 in the OSAS group and control group, respectively (Table 1). The median AHI of the OSAS patients was 28.5 (IQR = 14.1). The mean O_2

Table 1. Demographic data found in the study and statistical differences between groups.

	OSAS		Control		P
	Mean	±SD	Mean	±SD	
Age (years)	43.3	±8.2	43.6	±3.8	ns*
BMI	30.1	±0.6	26.4	±1.7	ns*
Sex	Man	Woman	Man	Woman	ns*
	12	9	5	5	

ns, nonsignificant, P > 0.05; *Student's t test and chi-square test

saturation was 91 ± 2.9 and the median desaturation rate was 17.2% (IQR = 10.8).

Mean hearing thresholds with pure tone audiometry (PTA) were 25 dB or better for all participants in the study and control groups. The mean speech discrimination rates of the OSAS patients revealed 96.1 ± 3.5 for the right ear and 95 ± 4.0 for the left. In the control group, the mean speech discrimination rates were 99.2 ± 1.3 and 99.4 ± 1.3 for the right and left ears, respectively (Figures 1 and 2).

The OSAS patients correctly responded with a mean of 21.4 ± 7.3 for 30 different sounds in DPT test. They responded correctly with a mean of 16.4 ± 8.9 in the FPT. In the control group, the mean correct answers for the DPT and FPT were 25.7 ± 3.4 and 22.8 ± 3.5, respectively.

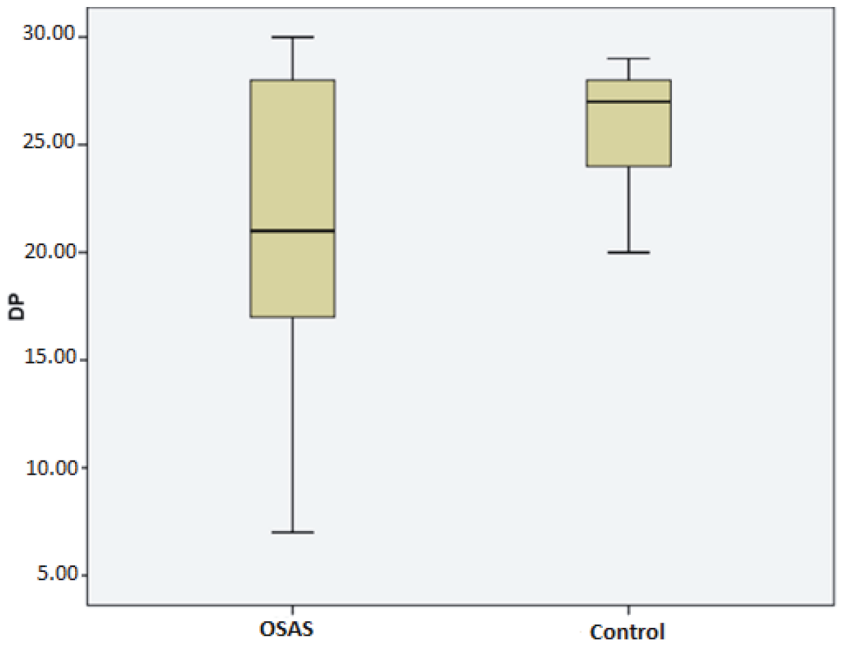


Figure 1. Distribution of DPT results between groups.

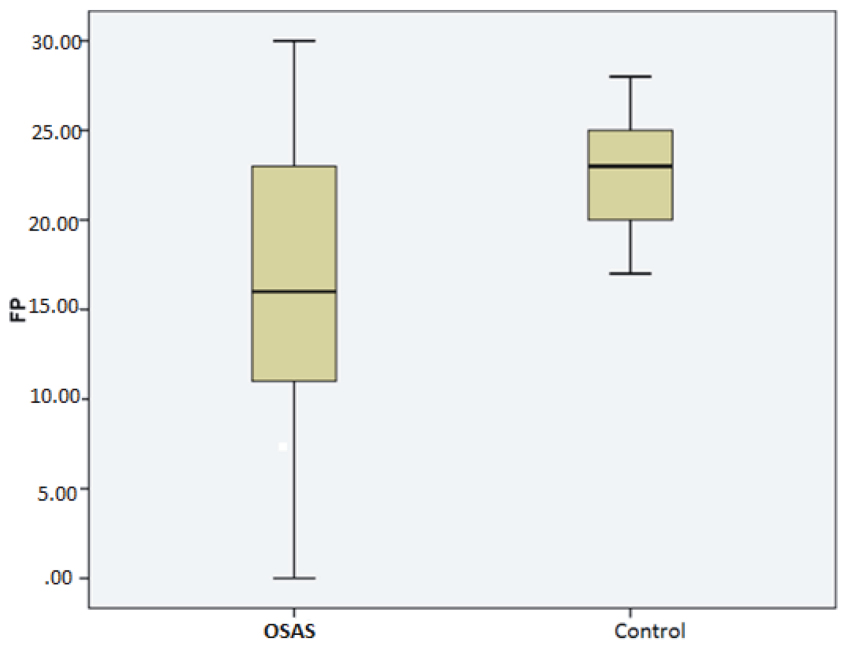


Figure 2. Distribution of FPT results between groups.

A significant decrease was observed in speech discrimination scores in the OSAS patients compared to the control group (P = 0.01). Moreover, in the central nonverbal detection tests, a statistically significant deterioration was found in the OSAS patients compared to the control group (P < 0.05) (Table 2).

4. Discussion

Alongside the clinical symptoms such as daytime sleepiness, morning headaches, and memory and concentration loss, OSAS increases the risk of cardiovascular system diseases such as coronary artery disease and hypertension and the risk of CNS disorders such as stroke as a result of deterioration of endocrine and metabolic systems (6,13,14).

Previously, different researchers conducted studies to investigate the effects of OSAS on the hearing system. In these studies ABR, OAE, and audiometry were used and it was shown that hearing loss can be especially profound in moderate to severe OSAS (6,15).

However, the location and cause of the auditory pathology in OSAS has not yet been fully elucidated. It is thought that hypoxia/reoxidation episodes that occur in the cochlea following hypoxic periods, which are seen in OSAS, and oxidative stress, endothelial dysfunction, and inflammatory events that are secondary to it may be damaging auditory pathways (6,16). Several studies have suggested that this oxidative stress may cause nerve damage in the hearing system (16–18).

In some of the studies investigators have suggested that sudden hearing loss seen in OSAS develops due to increased risk factors such as DM, hypertension (HT), thyroid diseases, and hyperlipidemia (19,20). In our study, the subjects with systemic diseases that may affect hearing negatively such as DM, HT, and thyroid disease and the subjects with apparent hearing loss were excluded. These comorbid chronic systemic conditions are well known to be associated with hypoxemia and disturbances in microvascular circulation. These diseases are common with OSAS and they can be a cause of hearing loss on their own (20–22).

There are studies in the literature specifically addressing the central auditory system in OSAS (6,16). In our study, the auditory pathways were tested via speech discrimination rates, central processing, and sequencing tests in moderate-to-severe OSAS patients and control subjects with normal hearing thresholds. A significant loss in speech discrimination was found in the OSAS patients compared to the control group. Likewise, significant deterioration was found in both auditory processing and sequencing tests (DPT, FPT) in OSAS patients compared to the control group. As a result, it was found that cochlear processing and central auditory damage could occur before the presentation of apparent hearing loss in OSAS patients.

This work differs from studies in the literature in that it specifically tests auditory processing, discrimination, and sequencing (i.e. auditory perception) in OSAS patients, even if the hearing is normal.

Table 2. Comparison of right and left speech discrimination tests and DPT and FPT in OSAS and control groups.

	Group	N	Mean	Std. deviation	P(*)
DPT	OSAS	21	21.47	7.35	0.038
	Control	10	25.70	3.43	
FPT	OSAS	21	16.04	8.98	0.006
	Control	10	22.80	3.55	
RSD	OSAS	21	96.19	3.51	0.002
	Control	10	99.20	1.39	
LSD	OSAS	21	95.04	4.03	0.000
	Control	10	99.40	1.34	

*Independent samples t test
DPT: Duration pattern test
FPT: Frequency pattern test
RSD: Right ear speech discrimination score
LSD: Left ear speech discrimination score

There are some limitations of our study. The healthy control group consisted of subjects without any OSAS symptoms (snoring, increased daytime sleepiness, etc.). However, we did not perform a PSG study in the control group. Subjects with normal hearing threshold were included in this study to rule out any other diseases (presbiacusis, otitis media, etc.) that may already cause damage to central auditory pathways. However, this exclusion criterion may also result in failure to include some OSAS patients with damage to both central and peripheral auditory pathways.

In conclusion, central auditory pathways were significantly impaired in the normal hearing OSAS patients compared to the control group in our study. Thus, we have shown that repeated hypoxic episodes that result in multisystemic disorders in OSAS could cause damage also to central auditory pathways before the presentation of apparent hearing loss. However, further studies with larger sample sizes are needed to verify our findings.

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