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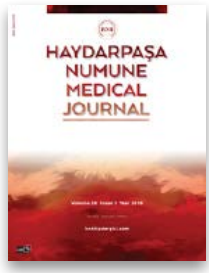

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ORIGINAL ARTICLE

Evaluation of Auditory Function in Patients with Obstructive Sleep Apnea Syndrome

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Abstract

Introduction: To investigate the effect of obstructive sleep apnea syndrome (OSAS) on auditory function.

Methods: We included 120 patients with OSAS [Apnea–hypopnea index (AHI) >5]. We excluded 35 patients with 35 mild OSAS (AHI: 5–15) and 20 with moderate OSAS (AHI: 15–30). Five patients with severe OSAS along with acute and chronic otitis, systemic disease, and medical treatment history that may affect past ear operation and central and peripheral auditory system were also excluded. Finally, we included 60 patients with severe OSAS and 60 healthy controls. Both patients and controls were divided into two groups based on age: 30–40-years group and 40–60-years group. A total of 120 patients were included . Pure-tone audiometry (PTA) and distortion product otoacoustic emission (DPOAE) tests were performed at a frequency range of 250–8000 Hz for both controls and patients, and their values were compared between all the groups.

Results: In the evaluation of auditory functions, there was a difference between the 30–40-years and 40–60-years control groups. There was a difference between the patient age group of 30–40 years and the patient age group of 40–60 years. There was no significant difference between other groups.

Discussion and Conclusion: Hearing loss, which is not influenced by auditory functions in patients with OSAS, arises due to advanced age. Since there is uncertainty about the effect of auditory functions on patients with OSAS in the literature, further studies may be needed.

Keywords: Audiometry; obstructive sleep apnea syndrome; otoacoustic emission.

OSAS is associated with recurrent complete or partial upper respiratory tract obstruction episodes during sleep, often accompanied by reductions in blood oxygen saturation [1]. Reactive oxygen products resulting from repeated attacks of hypoxia and normoxia cause endothelial dysregulation and inflammatory cascade activation, leading to vascular dysfunction and resulting in peripheral perfusion deterioration [2]. Ischemic damage may occur in the cochlea due to decreased cerebral blood flow caused by recurrent episodes of apnea [3].

The transduction mechanism of the inner ear and the conduction of nerve stimuli along the hearing pathway are highly dependent on oxygen support [4]. The auditory function can be negatively affected due to hypoxemia caused by OSAS [5]. In a study conducted by Casale et al. [6], pure-tone audiometry (PTA) test was evaluated in patients with OSAS, and it was determined that only high-frequency threshold elevation was observed. In a study by Hwang et al.[7] including 34 patients, PTA values were found to be at normal levels in all frequencies in patients with mild and moderate

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OSAS. Despite the decrease in tissue oxygenation due to OSAS, the role of OSAS in auditory function is still controversial. In our study, we aimed to determine whether auditory functions were affected in patients with OSAS.

Materials and Methods

Subjects

In total, 165 patients who were admitted to our clinic with complaints of snoring while sleeping were subjected to polysomnography test in the sleep laboratory of our hospital. After polysomnography examination, 120 patients diagnosed with OSAS were included in the study. Patients with anamnesis and a history of neurological and otologic illness, acute or chronic otitis media, familial hearing loss, abnormal tympanogram, head and neck trauma, chronic systemic disease, and drug use history that could affect the auditory system were excluded. Also, patients with simple snoring and mild and moderate OSAS (n=60) were excluded. Finally, 60 patients diagnosed with severe OSAS and 60 healthy controls were included. Both patients and controls were divided into two groups based on age: 30–40-years group and 40–60-years group. PTA and distortion product otoacoustic emission (DPOAE) tests were performed at a frequency range of 250–8000 Hz for both patients and controls, and their values were compared between all the groups. This study was approved by our hospital ethics committee. (Protocol number: B.10.1TKH.4.34.H.GP.0.01/66).

Sleep Test

All patients had six-channels EEG signals (C4-A1, C3-A2, 02-A1, 01-A2, F4-A1, F3-A2), two-channel EOG, chin, right and left anterior tibial EMG, body position, oral, nasal thermal sensor, nasal pressure sensor, thoracic and abdominal respiratory movements, ECG, breath sounds recording, 02 saturation. Sleep testing was performed using the Neuro-Soft analysis program. Sleep-related abnormal respiratory events were scored according to the American Sleep Medical Academy (AASM, 2014) criteria. According to the AASM criteria, those with apnea–hypopnea index (AHI) <5 were normal, and those with AHI ≥5 and <15 had mild, those with AHI ≥15 and ≤30 had moderate, and those with AHI >30 had severe OSAS. Only patients with severe OSAS were included. Both mean and lowest oxygen saturation levels during sleep were calculated; however, only mean oxygen saturation levels were used.

Audiological Evaluation

All patients were tested using PTA for both ears at a frequency ranging from 250 KHz to 8000 KHz by international standards.

Otoacoustic Emission Tests

The DPOAE measurements were recorded as DP-grams (dB SPL) using the autodynamic ILO-292 USB2 Version 6.0 (Denmark).

Statistical Analysis

IBM SPSS Statistics 22 for statistical analysis (SPSS IBM, Turkey) program was used. The normal distribution suitability of the parameters was evaluated by the Shapiro–Wilk test. Kruskal–Wallis test was used for the comparison of descriptive statistical methods (mean and standard deviation) and quantitative data, and comparison of non-normal distribution parameters between the groups when study data were evaluated. Mann–Whitney U test was used to determine the group causing the difference and to compare the two groups of parameters with non-normal distribution. Significance was assessed at $p<0.05$.

Results

The mean ages of the 30–40-years and 40–60-years patient groups were 35.07±2.50 and 53.37±5.25 years, respectively. The mean ages of the 30–40-years and 40–60-years control groups were 35.07±2.49 and 53.33±5.22 years, respectively. The BMI values of the patient group ranged from 27.8 to 33.9 (mean, 30.58±1.31). The BMI value of the 30–40-years patient group was 30.37±1.34. The BMI value of the 40–60-year patient group was 30.8±1.28 (Table 1). There was no statistically significant difference between the 30–40-years and 40–60-years patient groups with respect to AHI and O_2 values ($p>0.05$).

There were no statistically significant differences on the right side between the control and patient groups of all ages at 250 Hz, 2 kHz, 4 kHz, and 8 kHz ($p>0.05$); however, the-

Table 1. Assessment of BMI and age parameters in patient and control groups

Group	n	Min-Max	Ort±SS
30–40-years control			
Age	30	30-39	35.07±2.49
BMI	30	17.2-24.8	20.41± 2.01
30–40-years patient			
Age	30	30-39	35.07±2.50
BMI	30	27.8-32.9	30.37±1.34
40–60-years control			
Age	30	45-60	53.33±5.22
BMI	30	19.2-24.9	22.11±1.6
40–60-years patient			
Age	30	45-60	53.37±5.25
BMI	30	28.4-33.9	30.8±1.28

BMI: Body mass index.

re was a statistically significant difference in the age range between 30–40-years and 40–60-years control groups (p<0.05) (Table 2). There was no statistically significant difference between the groups at right 500 and 1000 Hz PTA values (p>0.05) (Table 2).

There were no statistically significant differences on the left side between control and patient groups of all ages at 250 Hz, 2 kHz, 4 kHz, and 8 kHz (p>0.05); however, there was a statistically significant difference in the age range between 30–40-years and 40–60-years control groups (p<0.05) (Table 3). There was no statistically significant difference between the groups at left 500 and 1000 Hz PTA values (p>0.05) (Table 3).

Otoacoustic Emission

There was no statistically significant difference between right and left 1.0 kHz, 1.4 kHz, 2.0 kHz, 2.8 kHz and 4.0 kHz DPOAE values between the groups (p>0.05). However, there was 6.0 and 8.0 kHz DPOAE values of the 30–40-years control group were found to be statistically significantly lower than the DPOAE values of the 30–40-years patient group (p<0.05) (Table 4).

Table 2. Evaluation of right PTA values among patient and control groups

Right PTA	30–40-years control group Ort±SS (median)	30–40-years patient group Ort±SS (median)	40–60-years control group Ort±SS (median)	40–60-years patient group Ort±SS (median)	p
250 Hz	13.5±4.18 (15)	14.17±4.37 (15)	17.67±3.88 (20)	18.83±4.09 (20)	0.000*
500 Hz	12.5±3.66 (12.5)	13.33±3.56 (15)	14.33±3.88 (15)	13.17±3.82 (10)	0.375
1000 Hz	10.67±4.69 (10)	10.83±4.17 (10)	11.83±3.07 (10)	11.83±2.45 (10)	0.665
2000 Hz	9.33±5.04 (10)	10.5±5.62 (10)	11.83±5 (10)	13.33±6.34 (10)	0.004*
4000 Hz	14.33±5.68 (12.5)	13.5±4.94 (10)	45.17±18.68 (40)	42±24.83 (40)	0.000*
8000 Hz	17.83±7.15 (15)	18.5±9.84 (15)	58.67±18.89 (55)	57.83±21.48 (55)	0.000*

PTA: Pure-tone audiometry; Hz: Hertz; Kruskal–Wallis Test; *p<0.05.

Table 3. Evaluation of left PTA values among patient and control groups

Left PTA	30–40-years control group Ort±SS (median)	30–40-years patient group Ort±SS (median)	40–60-years control group Ort±SS (median)	40–60-years patient group Ort±SS (median)	p
250 Hz	14.83±5.17 (15)	14.67±4.34 (15)	18±4.28 (20)	18.5±3.75 (20)	0.000*
500 Hz	14.33±3.65 (15)	13±3.62 (15)	14.33±3.88 (15)	13±3.37 (12.5)	0.302
1000 Hz	10.83±4.17 (10)	11.17±4.09 (10)	11.83±3.07 (10)	11.67±2.4 (10)	0.862
2000 Hz	11.5±6.84 (10)	10.5±5.47 (10)	12.5±5.98 (10)	12.17±4.49 (10)	0.149
4000 Hz	13.83±5.2 (10)	13.67±4.9 (10)	43.83±18.27 (40)	44±24.61 (40)	0.000*
8000 Hz	19±7.12 (15)	18±7.94 (15)	57.33±18.56 (55)	60.83±19.35 (57.5)	0.000*

PTA: Pure-tone audiometry; Hz: Hertz; Kruskal–Wallis Test; *p<0.05.

Table 4. Evaluation of right DPOAE values among patient and control groups

Right DPOAE	30–40-years control group Ort±SS (median)	30–40-years patient group Ort±SS (median)	40–60-years control group Ort±SS (median)	40–60-years patient group Ort±SS (median)	p
1.0 kHz	12.14±6.27 (11.6)	13.98±7.25 (14)	11.36±6.04 (10.7)	13.43±6.5 (12.2)	[1]0.445
1.4 kHz	12.76±6.75 (12.4)	13.42±5.53 (14)	17.29±6.74 (18.9)	14.12±6.03 (13.7)	[1]0.079
2.0 kHz	15.26±7.8 (15.2)	15.77±11.22 (14.5)	15.79±6.87 (14.5)	15.06±7.86 (15.1)	[1]0.978
2.8 kHz	12.14±6.84 (12.3)	13.27±6.74 (12.3)	12.49±6.18 (12.8)	13.52±7.15 (14.2)	[1]0.846
4.0 kHz	10.88±5.83 (9.9)	12.75±6.91 (11.9)	11.58±6.37 (11.5)	12.98±6.89 (12.1)	[1]0.588
6.0 kHz	10.23±6.9 (7.6)	14.21±5.98 (15.3)	-	-	[2]0.011*
8.0 kHz	9.81±5.1 (8.1)	7.21±4.54 (5)	-	-	[2]0.012*

DPOAE: Distortion product otoacoustic emission; [1]Kruskal–Wallis Test; [2]Mann–Whitney U Test; *p<0.05.

Table 5. Evaluation of Left DPOAE values among patient and control groups

Left DPOAE	30–40-years control group Ort±SS (median)	30–40-years patient group Ort±SS (median)	40–60-years control group Ort±SS (median)	40–60-years patient group Ort±SS (median)	p
1.0 kHz	11.76±5.63 (11.2)	15.1±8.41 (19.4)	15.06±4.8 (14.6)	13.79±6.1 (13.9)	[1]0.166
1.4 kHz	13.51±7.51 (13.7)	10.77±4.72 (10.4)	14.05±6.07 (14.7)	13.57±8.42 (11.8)	[1]0.157
2.0 kHz	12.67±6.63 (11.3)	19.53±19.26 (15.9)	18.55±23.33 (14)	13.22±7.62 (12.8)	[1]0.112
2.8 kHz	14.14±6.96 (14.5)	12.39±7.7 (12.4)	14.24±6.69 (14.2)	12.27±5.67 (12.8)	[1]0.541
4.0 kHz	12.13±7.4 (11.9)	13.4±7.52 (12.8)	14.67±8.05 (14)	10.03±7.51 (9.1)	[1]0.074
6.0 kHz	9.32±5.97 (7.9)	13.61±8.05 (13)	-	-	[2]0.042*
8.0 kHz	9.72±5.21 (8.8)	11.59±11.71 (4.6)	-	-	[2]0.201

DPOAE: Distortion product otoacoustic emission; [1]Kruskal–Wallis Test; [2]Mann–Whitney U Test; *p<0.05.

The left 6.0 kHz DPOAE values of the 30–40-years control group were found to be statistically significantly lower than the DPOAE values of the 30–40-years patient group (p<0.05). However, there was no statistically significant difference between the groups for left DPOAE values of 8.0 kHz (p>0.05) (Table 5).

Discussion

The auditory system may be affected by reduced oxygen support resulting from vascular damage [5]. In PTA tests of patients with OSAS, it was shown that they were elevated at the hearing thresholds at high frequencies and that this was due to OSAS severity [5]. Transient otoacoustic emission (TEOAE) test and auditory brainstem (ABR) test responses in patients with OSAS have not been shown to be altered [5].

In our study, there was no difference in the auditory function between the patients in the same age range. The 30–40-years patient group had better hearing levels than the 40–60-years control group. This suggests that impairment in auditory function may be age-related rather than OSAS-related. The age-related hearing loss is due to the functional loss of sensorial and neuronal components [8]. Age-related presbycusis may be due to various physiological degenerations as well as noise pollution, cumulative effects of the drugs used, and genetic predisposition [9]. Presbycusis has shown that the vast majority of the affected auditory functions are extremely high frequency than in low frequency [10]. In daily life, hearing loss often occurs at high frequency because noise pollution is an important factor and the use of medical treatment increases in later ages. In our study, it was observed that there was hearing loss particularly in the high-frequency group in older age group. In a study comparing the control group with patients with moderate and severe OSAS, a significant difference regarding amplitudes was detected in the DPOAE test at 6–8 KHz [11]. In our study, there was no difference in the comparison between the patient and control groups of the same age. In our study, it was found that the comparison of age groups in the elderly was influenced by auditory functions independently of OSAS in the older age group. In another study, patients with severe OSAS showed a decrease in high frequencies such as 4000 and 8000 Hz in the PTA test, but no effect on otoacoustic emission and ABR tests [12]. In our study, it was found that DPOAE and PTA values were not affected in patients with severe OSAS. The role of OSAS in auditory functions is still controversial, depending on the outcome of the work being done. In our study, it was determined that OSAS did not affect the auditory function. It was determined that the effect on auditory functions was not related to OSAS but to progressive age. The fact that there are different results from studies investigating the effect of OSAS on auditory functions suggest that this field requires more extensive studies.

Conclusions

Hearing loss, which is not influenced by auditory functions in patients with OSAS, emerges due to advanced age. Since there is uncertainty about the effect of auditory functions on patients with OSAS in the literature, further studies may be needed.

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Conflict of Interest: None declared.

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