European Archives of Oto-Rhino-Laryngology (2019) 276:2215–2221
https://doi.org/10.1007/s00405-019-05468-8

OTOLOGY

The association between obstructive sleep apnea and hearing loss: a cross-sectional analysis

Serkan Kayabasi[1] · **Omer Hizli**[2] · **Guven Yildirim**[3]

Received: 14 March 2019 / Accepted: 8 May 2019 / Published online: 16 May 2019

Abstract

Purpose To determine if sleep apnea had significant effects on hearing functions and to investigate the polysomnography parameters that might be associated with hearing impairment in sleep apnea patients.

Methods We included 120 patients who were admitted to sleep disorders outpatient clinic. We constituted four groups by reference to the apnea–hypopnea index (including control group), and compared the audiometric parameters of the groups. Additionally, we investigated the correlation of apnea–hypopnea index, desaturation index and min. oxygen saturations with pure-tone thresholds, speech recognition thresholds and speech discrimination scores.

Results The median pure-tone thresholds at 250, 500, 1000, 2000, 4000 and 8000 mHz, the median speech recognition thresholds and the median speech discrimination scores on both ears did significantly differ among four groups ($p < 0.001$). Moderate sleep apnea affected high-frequency hearing functions and speech discrimination scores, and severe sleep apnea had significant effects on all hearing functions. Pure-tone thresholds and speech recognition thresholds on the both ears were positively correlated with apnea–hypopnea index and desaturation index, and negatively correlated with min. oxygen saturation ($p < 0.001$). Speech discrimination scores on the both ears were negatively correlated with apnea–hypopnea index and desaturation index, and positively correlated with min. oxygen saturation ($p < 0.001$).

Conclusion Obstructive sleep apnea syndrome (OSA) had several effects on hearing, and hearing impairment might be associated with the severity of OSA. Moderate OSA affected high-frequency hearing functions and severe OSA affected all hearing functions negatively.

Keywords Sleep apnea · Hearing · Polysomnography · Audiometry

Introduction

Obstructive sleep apnea syndrome (OSA) is a multisystem disease characterized by repetitive episodes of complete or partial upper airway obstruction during the sleep, resulting in reduced blood oxygen saturation [1]. It affects many systems in human body including cardiovascular, endocrine, neuro-psychiatric, cognitive and vestibular systems [2, 3]. The main mechanism of effects of OSA on multiple systems is still unclear but clinicians usually think of OSA as leading an inflammatory process in whole body. In OSA patients, elevated levels of circulating inflammatory mediators related to intermittent hypoxemia were reported [4]. In addition to the systems above, cochlear and vestibular functions might be affected in OSA patients due to intermittent hypoxemia and decreased blood oxygen saturations. In the prior literature, studies supporting this contention reported that OSA had significant effects on hearing functions [5, 6]. Additionally, severity of OSA was reported to be associated with the level of hearing impairment [7]. In this study, we investigated the polysomnography (PSG) parameters that might potentially be associated with hearing functions in OSA patients.

The goal of this cross-sectional clinical study was to determine if OSA had significant effects on hearing functions by performing a comprehensive audiometric evaluation in OSA patients, and to investigate the PSG parameters

✉ Omer Hizli
hizliomer@gmail.com

[1] ENT Department, Faculty of Medicine, Aksaray University, 68100 Aksaray, Turkey

[2] ENT Department, A. Ilhan Ozdemir Education and Research Hospital, Giresun University, 28100 Giresun, Turkey

[3] ENT Department, Faculty of Medicine, Giresun University, 28100 Giresun, Turkey

that might be associated with hearing impairment in OSA patients.

Materials and methods

Subjects and study design

In this cross-sectional clinical study, we included 120 patients from two different institutions who were admitted to sleep disorders outpatient clinic. We took a detailed medical history of all patients, performed a comprehensive otolaryngologic and rhinologic examination, a pure-tone audiometry and a whole-night PSG (type 1). Excluded from the study were the patients elder than 65 years (to avoid presbycusis) and the patients with following conditions that might be the cause of hearing loss: perforated tympanic membrane, otitis media, otosclerosis, Meniere's disease, history of otologic surgery, ototoxicity, and noise-induced hearing loss and acoustic trauma. We noted the PSG parameters like AHI, desaturation index (DI) and min. oxygen saturation ($MinO_2$) that might be in association with hearing loss in patients with OSA. To facilitate the inter-study comparability, we utilized American Academy of Otolaryngology—Head and Neck Surgery minimal reporting standard for reporting audiometric data and constituted scatter diagrams of pretreatment hearing results of our study population [8]. For statistical analysis, we noted the ages, genders, pure-tone thresholds (PTT) (bone conduction) at 250, 500, 1000, 2000, 4000 MHz, and PTTs (air conduction) at 8000 MHz, speech discrimination score (SDS) and speech recognition threshold (SRT) of all patients as well. For pure-tone audiometry, we used fully calibrated and well-maintained Orbiter 922-version 2 clinical audiometer (Madsen Electronics, Copenhagen, Denmark). We used the Alice® 5 Diagnostic Sleep System (Respironics, Murrysville, PA, USA) linked to the Alice® Sleepware™ software for whole-night PSG (type 1). We recruited the data of the patients who were admitted to outpatient clinic of sleep disorders with any compliant related to sleep disorders including snoring, witnessed apnea, daytime sleepiness, insomnia and periodic leg movements, etc., and the patients undergone PSG for the anxiety of having OSA (without any symptom of OSA). Among these patients, the patients of whom we were sure that they did not have OSA [those with apnea–hypopnea index (AHI) under 5] were included in the control group. Given the aim of this study was to investigate the association of hearing impairment with OSA, we included only the individuals who undergone PSG and had the evidence that they had AHI less than 5, in our control group. Thus, we ensured that our control group did not contain any patient with OSA. Additionally, study groups consisted of the patients with the diagnosis of OSA (those with AHI over 5). To avoid the confounding effects of the age (presbycusis, e.g.), we matched the groups regarding the age by excluding elderly patients (≥ 65 years), without any additional effort. Gender matching of the groups was incidental. To investigate the effects of OSA on hearing and the association of the severity of OSA with hearing impairment, we constituted four groups by reference to the AHIs of the patients: The control group (AHI ≤ 5), the mild OSA group (AHI = 6–15), the moderate OSA group (AHI = 16–30) and the severe OSA group (AHI > 30). Then, we statistically compared the PTTs at 250, 500, 1000, 2000, 4000 and 8000 MHz, SDSs and SRTs of the groups. Additionally, we investigated the association of DI and $MinO_2$ with PTTs at 250, 500, 1000, 2000, 4000 and 8000 MHz, SDSs and SRTs. The study was conducted in line with the dictates of the World Medical Association Declaration of Helsinki and approved by the local ethic committee (IRB number: 01–07).

Statistical Analysis

We presented the results as median (min–max). We confirmed the abnormal distribution of all data using Kolmogorov–Smirnov normality test ($p < 0.001$). We performed the comparisons of PTTs at 250, 500, 1000, 2000, 4000 and 8000 MHz, the SRT values and the median SDS values among the groups using Kruskal–Wallis analysis. We used Mann–Whitney *U* test as the post hoc test for advanced comparisons of the values of mild OSA, moderate OSA and severe OSA group with the control group. We performed the correlation analysis between the audiometric parameters and PSG parameters using Spearmen correlation test. A *p* value less than 0.05 was considered as statistically significant. Additionally, for post hoc comparison tests, we used Bonferroni correction of four groups (hexed combination) and a *p* value less than 0.008 (0.05/6) was considered as statistically significant.

Results

One-hundred and twenty patients (68 males and 52 females) were eligible for this study. The mild OSA group consisted of 33 patients (19 males and 14 females, mean age 42 ± 8 years), the moderate OSA group consisted of 24 patients (14 males and 10 females, mean age 43 ± 6 years), the severe OSA group consisted of 36 patients (21 males and 15 females, mean age 42 ± 8 years) and the control group consisted of 27 patients (14 males and 13 females, mean age 43 ± 7 years). The groups were age- and gender matched ($p = 0.98$ and $p = 0.95$, respectively). Fig. 1 represents the scatter diagram of the patient numbers by reference to pure-tone averages (500–1000–2000–3000 MHz) and SDSs of the right ears [8]. Figure 2 represents the scatter diagram

of the patient numbers by reference to pure-tone averages (500–1000–2000–3000 MHz) and SDSs of the left ears [8]. In the scatter diagram of the right ears, we detected one patient (from severe OSA group, AHI: 90.5) with unaffected pure-tone average (10 dB) and impaired SDS (68%). Additionally, we detected another patient (from severe OSA group, AHI: 50.1) in the scatter diagram of the left ears with unaffected pure-tone average (10 dB) and impaired SDS (76%). Kruskal–Wallis analysis revealed that the median PTTs at 250, 500, 1000, 2000, 4000 and 8000 mHz, the median SRT values (Fig. 3) and the median SDS values (Fig. 5) on the right ears did significantly differ among four groups ($p < 0.001$) (Table 1). Additionally, the median PTTs at 250, 500, 1000, 2000, 4000 and 8000 mHz, the median SRT values (Fig. 4) and the median SDS (Fig. 5) values on the left ears did significantly differ among four groups ($p < 0.001$) (Table 2).

In post hoc comparison test between mild OSA group and control group, we found that the median PTTs at 250, 500, 1000, 2000, 4000 and 8000 mHz, the median SRT values and the median SDS values on the right ears of mild OSA group did not significantly differ from the control group ($p = 0.54$, $p = 0.39$, $p = 0.3$, $p = 0.24$, $p = 0.11$, $p = 0.14$, $p = 0.93$ and $p = 0.16$, respectively), as did not the values on the left ears ($p = 0.1$, $p = 0.28$, $p = 0.73$, $p = 0.04$, $p = 0.16$,

Fig. 1 The scatter diagram of the patient numbers by reference to pure-tone averages and SDSs of the right ears

Speech Discrimination Score (%)

Pure Tone Average (dB)	100-90	89-80	79-70	69-60	59-50	49-40	39-30	29-20	19-10	9-0
0-10	42	10		1						
11-20	26	16	2	1						
21-30	4	13	2							
31-40		1	2							
41-50										
51-60										
61-70										
71-80										
81-90										
>91										

Fig. 2 The scatter diagram of the patient numbers by reference to pure-tone averages and SDSs of the left ears

Speech Discrimination Score (%)

Pure Tone Average (dB)	100-90	89-80	79-70	69-60	59-50	49-40	39-30	29-20	19-10	9-0
0-10	43	7	1							
11-20	24	17	4	2						
21-30	4	11	3	1						
31-40			2							
41-50			1							
51-60										
61-70										
71-80										
81-90										
>91										

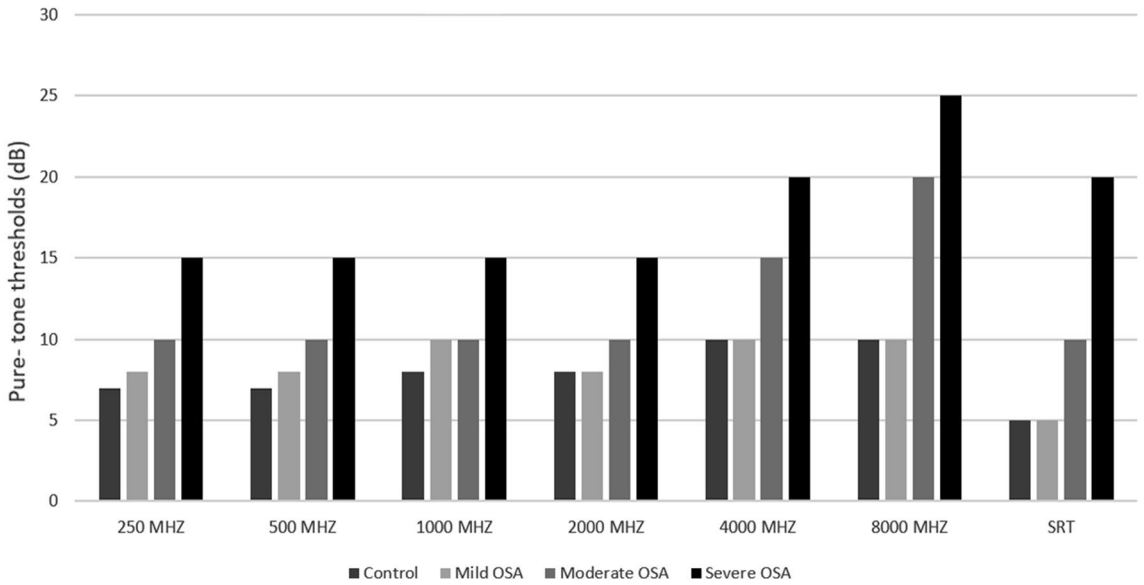

Fig. 3 Comparative graph of PTTs and SRTs of the right ears

Table 1 Median values of audiometric parameters of the groups (Right ears)

	Control group	Mild OSAS	Moderate OSAS	Severe OSAS	*P* value[#]
PTT at 250 MHz (dB)	7 (5–13)	8 (3–20)	10 (5–20)	15 (5–35)	< 0.001
PTT at 500 MHz (dB)	7 (5–15)	8 (3–25)	10 (5–20)	15 (5–25)	< 0.001
PTT at 1000 MHz (dB)	8 (2–20)	10 (3–25)	10 (5–25)	15 (5–25)	< 0.001
PTT at 2000 MHz (dB)	8 (3–15)	8 (5–20)	10 (3–30)	15 (5–28)	< 0.001
PTT at 4000 MHz (dB)	10 (3–22)	10 (5–45)	15 (5–45)	20 (10–45)	< 0.001
PTT at 8000 MHz (dB)[a]	10 (5–25)	10 (5–45)	20 (5–50)	25 (10–65)	< 0.001
SRT (dB)	5 (0–15)	5 (0–15)	10 (0–20)	20 (5–40)	< 0.001
SDS (%)	95 (90–100)	92 (86–100)	88 (76–96)	84 (68–100)	< 0.001

PTT pure-tone threshold, *SRT* speech reception threshold, *SDS* speech discrimination score

[#]Of Kruskal–Wallis analysis

[a]At 8000 MHz, we noted air-conduction thresholds

$p = 0.27$, $p = 0.43$ and $p = 0.32$, respectively). Thus, we found that mild OSA had no effect on hearing functions.

In post hoc comparison test between moderate OSA group and control group, we found that the median PTTs at 4000 and 8000 mHz were significantly higher ($p < 0.001$), and the median SDS values were significantly lower ($p < 0.001$) on the right ears of the moderate OSA group, compared to the control group. Additionally, the median PTTs at 2000, 4000 and 8000 MHz were significantly higher ($p = 0.001$) and the median SDS values were significantly lower ($p < 0.001$) on the left ears of the moderate OSA group, compared to the control group. Thus, we found that moderate OSA affected high-frequency hearing functions (over 4000 MHz, particularly) and SDSs.

In post hoc comparison test between severe OSA group and control group, we found that the median PTT at 250, 500, 1000, 2000, 4000 and 8000 mHz and the median SRT values on the right ears were significantly higher ($p < 0.001$) in severe OSA group compared to the control group as were the values on the left ears ($p < 0.001$). Additionally, the median SDS values on the right ears were significantly lower ($p < 0.001$) in severe OSA group compared to the control group as were the values on the left ears ($p < 0.001$). Thus, we found that severe OSA had significant effects on all hearing functions.

Spearman correlation test revealed that PTT at 250, 500, 1000, 2000, 4000 and 8000 MHz, and SRT on the right ears were positively correlated with AHI ($p < 0.001$, rho = 0.48, 0.42, 0.45, 0.51, 0.58, 0.63, 0.57, respectively) and DI ($p < 0.001$, rho = 0.43, 0.38, 0.43, 0.51, 0.58, 0.59, 0.53, respectively), and negatively correlated with $MinO_2$ ($p < 0.001$, rho = − 0.41, − 0.37, − 0.39, − 0.45, − 0.55, − 0.58, − 0.41, respectively). Also, SDSs on the right ears were negatively correlated with AHI ($p < 0.001$,

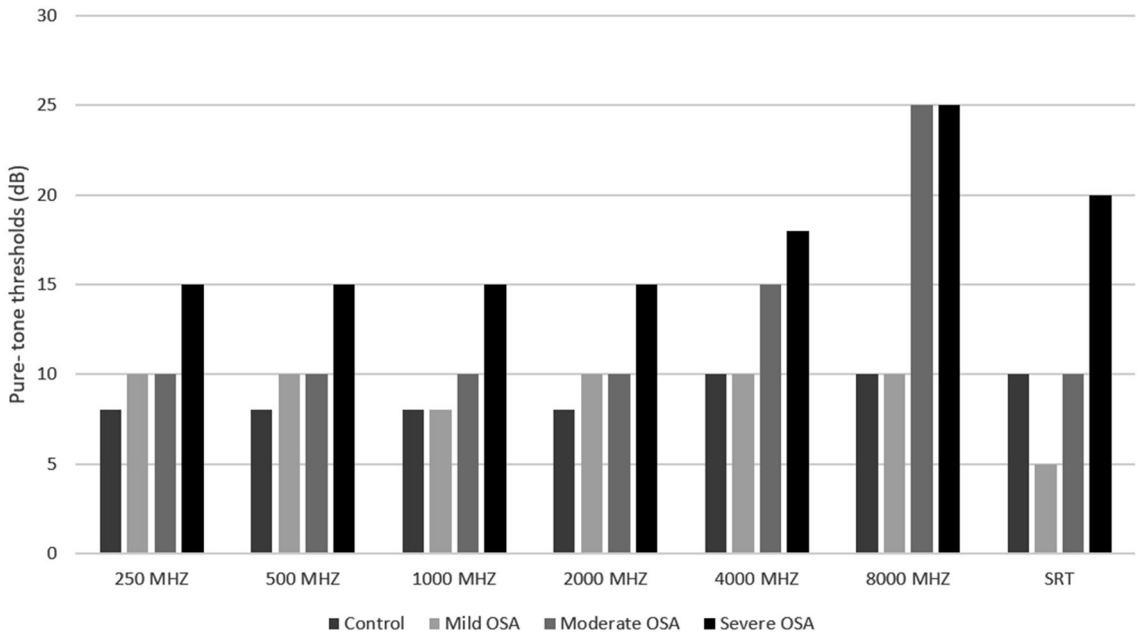

Fig. 4 Comparative graph of PTTs and SRTs of the left ears

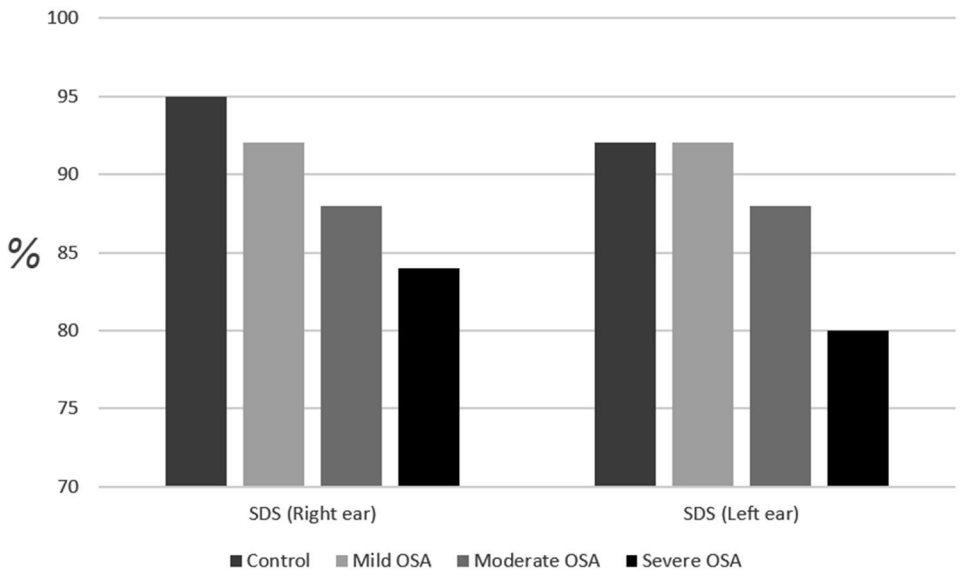

Fig. 5 Comparative graph of SDSs

rho = − 0.63) and DI ($p < 0.001$, rho = − 0.53), and positively correlated with $MinO_2$ ($p < 0.001$, rho = 0.55). Moreover, PTT at 250, 500, 1000, 2000, 4000 and 8000 MHz, and SRT on the left ears were positively correlated with AHI ($p < 0.001$, rho = 0.51, 0.47, 0.49, 0.51, 0.59, 0.60, 0.54, respectively) and DI ($p < 0.001$, rho = 0.47, 0.42, 0.45, 0.55, 0.55, 0.52, 0.47, respectively), and negatively correlated with $MinO_2$ ($p < 0.001$, rho = − 0.44, − 0.41, − 0.44, − 0.51, − 0.56, − 0.57, − 0.41, respectively). In addition, SDSs on the left ears were negatively correlated with AHI ($p < 0.001$, rho = − 0.61) and DI ($p < 0.001$, rho = − 0.53), and positively correlated with $MinO_2$ ($p < 0.001$, rho = 0.56). Thus, as AHI and DI increased, PTTs and SRT increased, and SDS decreased. Moreover, as $MinO_2$ decreased, PTTs and SRT increased, and SDS decreased.

Discussion

OSA has well-known negative effects on multiple systems of human body and might also affect cochlear and vestibular systems; but the characteristics and the type of effects of

Table 2 Median values of audiometric parameters of the groups (Left ears)

	Control group	Mild OSAS	Moderate OSAS	Severe OSAS	*P* value[#]
PTT at 250 MHz (dB)	8 (0–15)	10 (5–20)	10 (5–20)	15 (5–25)	< 0.001
PTT at 500 MHz (dB)	8 (0–15)	10 (5–25)	10 (5–20)	15 (5–25)	< 0.001
PTT at 1000 MHz (dB)	8 (5–15)	8 (5–25)	10 (5–25)	15 (5–25)	< 0.001
PTT at 2000 MHz (dB)	8 (5–15)	10 (5–25)	10 (5–30)	15 (5–38)	< 0.001
PTT at 4000 MHz (dB)	10 (5–25)	10 (5–40)	15 (5–30)	18 (10–45)	< 0.001
PTT at 8000 MHz (dB)[a]	10 (5–25)	10 (5–50)	25 (5–40)	25 (12–60)	< 0.001
SRT (dB)	10 (0–15)	5 (0–15)	10 (0–20)	20 (5–40)	< 0.001
SDS (%)	92 (90–100)	92 (82–100)	88 (76–100)	80 (68–100)	< 0.001

PTT pure-tone threshold, *SRT* speech reception threshold, *SDS* speech discrimination score

[#]Of Kruskal–Wallis analysis

[a]At 8000 MHZ, we noted air-conduction thresholds

OSA on the cochlear elements are not clear. Additionally, the data about association between the OSA and the features of hearing loss is insufficient in the prior literature. Based on the hypothesis of that the severity of OSA might be a determining factor of the type of hearing impairment, we performed a comprehensive audiologic evaluation in OSA patients to assess the results of the negative effects of OSA on cochlear elements. The cross-sectional association of the severity of OSA with increased PTTs, SRTs and decreased SDSs was evident in this study.

The study of Seo et al. focusing on the auditory dysfunction aggravated with the severity of OSA suggested the potential mechanism of hearing loss as the damage of sensory epithelia of inner ear in OSA patients [9]. In this study, authors used mice as a model for OSA and performed a histopathologic assessment to figure out the mechanism of hearing impairment in OSA. Unlike the report by Seo et al., our study focused on the cross-sectional association between polysomnography parameters and audiology parameters. Given that the chronic intermittent hypoxemia affects vestibular system in OSA patients [3, 10], a cochlear damage is also expected. However, the causative mechanism of hearing loss in OSA patients remains a matter of debate. Some previous publications suggested that negative effects of OSA on central auditory pathways might be the main mechanism of hearing impairment in OSA patients [6]. In contrast, most of the publications focusing on hearing functions in OSA patients reported that a cochlear ischemia due to chronic intermittent hypoxemia and cochlear inflammation due to pro-inflammatory base of OSA might cause hearing impairment in OSA patients [5, 9]. According to the investigation by Chopra et al. [5], increased OSA severity (defined by AHI) was associated with hearing loss both in high and low frequencies in the Hispanic population. We found similar results in Anatolian population; however, our study had the data of SDSs unlike this publication, since we found decreased SDSs in moderate and severe OSA patients. Additionally, studies reporting a hearing loss only at higher frequencies and reporting an elevated tinnitus risk in OSA patients are also available in English-language literature [11, 12]. Previous archival studies conducted using human temporal bones from deceased patients with different types of diseases were capable of demonstrating the damage in vestibular and cochlear sensory neuro-epithelia [13, 14]. Hence, future comparative studies using human temporal bones of deceased patients with different levels of OSA might be needed to find out the main mechanism of hearing loss in OSA patients. However, in this cross-sectional clinical study, we investigated the audiometric parameters of OSA patients in association with PSG parameters. It is not surprising that severe OSA group had significantly higher PTTs and SRTs, and significantly lower SDSs. Although the PTTs of patients with OSA were found within normal limits in the report by Iriz et al. [6], we instead hypothesize that severe OSA had negative effects on all hearing functions, based on our results. In addition, we found decreased SDSs that might be associated with affected central auditory pathways in patients with OSA, and this result was consistent with the report by Iriz et al. [6]. Furthermore, our study could compare the hearing levels of the patients with three different levels of OSA (mild–moderate–severe), including a greater number of the patients with OSA. In moderate OSA group, we found higher PTTs only in higher frequencies. In this study, we matched the mean ages and gender distribution of all groups to control the potential confounding effects of age like presbycusis. For this reason, this study represents a clinical implication that patients with moderate to severe OSA might be under risk of hearing impairment regardless aging.

In this study, we found that severe OSA had significant effects on all hearing functions. Regarding severity of OSA, there might be several potential mechanisms for hearing loss in OSA patients. Many factors having a role in OSA etiopathogenesis could be a leading potential factor of hearing loss in severe OSA patients, this is not clear. Given the higher AHI was a risk factor for lower SDSs,

severity of OSA might be related to central integration of hearing pathways, affecting SDSs. Another potential effect of sever OSA on hearing could be the acoustic trauma of snoring sound. Maimon et al. reported that severity of OSA is significantly related to increased snoring sound intensity [15]. Hence, higher levels of snoring sound could contribute to hearing loss in moderate to severe OSA patients.

PSG is well known to be gold standard for the diagnosis of OSA. The main parameter of PSG to diagnose and classify an OSA patient is AHI. However, DI, $MinO_2$ are implicated for clinical practice, giving insight about the status of OSA patient concerning the blood oxygen saturation and hypoxemia. In our correlation, PTTs were positively correlated with DI and negatively correlated with $MinO_2$. Additionally, SDSs were negatively correlated with AHI and DI, and positively correlated with $MinO_2$. As a result, we can hypothesize that reduced blood oxygen concentrations and chronic intermittent hypoxemia might have negative effects on both cochlear sensory epithelia and auditory pathways.

Conclusion

In conclusion, OSA had several effects on hearing, and hearing impairment might be associated with the severity of OSA. Moderate OSA affected high-frequency hearing functions and severe OSA affected all hearing functions negatively. Increased DI and decreased $MinO_2$ were related to increased PTTs and decreased SDSs.

Author contribution SK: designed study, collected and analyzed data, supervised study. ÖH: collected and analyzed data, made statistics, wrote article. GY: analyzed data, supervised study, revised article.

Funding None.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest.

Ethical statement All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors. This study was approved by the local ethic committee (IRB number: 01-07) and informed consent was obtained from all individual participants included in the study.

References

1. AASM (2014) International classification of sleep disorders, 3rd edn. American Academy of Sleep Medicine, Darien
2. Hizli O, Ozcan M, Unal A (2013) Evaluation of comorbidities in patients with OSAS and simple snoring. Sci World J. https://doi.org/10.1155/2013/709292
3. Kayabasi S, Iriz A, Cayonu M, Cengiz B, Acar A, Boynuegri S, Mujdeci B, Eryilmaz A (2015) Vestibular functions were found to be impaired in patients with moderate-to-severe obstructive sleep apnea. Laryngoscope 125(5):1244–1248. https://doi.org/10.1002/lary.25021
4. Sozer V, Kutnu M, Atahan E, Caliskaner Ozturk B, Hysi E, Cabuk C, Musellim B, Simsek G, Uzun H (2018) Changes in inflammatory mediators as a result of intermittent hypoxia in obstructive sleep apnea syndrome. Clin Respir J 12(4):1615–1622. https://doi.org/10.1111/crj.12718
5. Chopra A, Jung M, Kaplan RC, Appel DW, Dinces EA, Dhar S, Zee PC, Gonzalez F 2nd, Lee DJ, Ramos AR, Hoffman HJ, Redline S, Cruickshanks KJ, Shah NA (2016) Sleep apnea is associated with hearing impairment: the hispanic community health study/study of Latinos. J Clin Sleep Med 12(5):719–726. https://doi.org/10.5664/jcsm.5804
6. Iriz A, Duzlu M, Kokturk O, Kemaloglu YK, Eravci FC, Kuukunal IS, Karamert R (2018) The effect of obstructive sleep apnea syndrome on the central auditory system. Turk J Med Sci 48(1):5–9. https://doi.org/10.3906/sag-1705-66
7. Matsumura E, Matas CG, Sanches SGG, Magliaro FCL, Pedreno RM, Genta PR, Lorenzi-Filho G, Carvallo RMM (2018) Severe obstructive sleep apnea is associated with cochlear function impairment. Sleep Breath 22(1):71–77. https://doi.org/10.1007/s11325-017-1530-5
8. Gurgel RK, Jackler RK, Dobie RA, Popelka GR (2012) A new standardized format for reporting hearing outcome in clinical trials. Otolaryngol Head Neck Surg 147(5):803–807. https://doi.org/10.1177/0194599812458401
9. Seo YJ, Ju HM, Lee SH, Kwak SH, Kang MJ, Yoon JH, Kim CH, Cho HJ (2017) Damage of inner ear sensory hair cells via mitochondrial loss in a murine model of sleep apnea with chronic intermittent hypoxia. Sleep 40(9):1–7. https://doi.org/10.1093/sleep/zsx106
10. Sowerby LJ, Rotenberg B, Brine M, George CF, Parnes LS (2010) Sleep apnea, daytime somnolence, and idiopathic dizziness—a novel association. Laryngoscope 120(6):1274–1278. https://doi.org/10.1002/lary.20899
11. Ekin S, Turan M, Arısoy A, Gunbatar H, Sunnetcioglu A, Asker S, Yıldız H (2016) Is there a relationship between obstructive sleep apnea (OSA) and hearing loss? Med sci Monitor 22:3124–3128. https://doi.org/10.12659/MSM.897347
12. Koo M, Hwang JH (2017) Risk of tinnitus in patients with sleep apnea: a nationwide, population-based, case-control study. Laryngoscope 127(9):2171–2175. https://doi.org/10.1002/lary.26323
13. Hizli O, Hizli P, Kaya S, Monsanto Rda C, Paparella MM, Cureoglu S (2016) Histopathologic ear findings of syphilis: a temporal bone study. Eur Arch Otorhinolaryngol 273(9):2443–2449. https://doi.org/10.1007/s00405-015-3834-z
14. Kariya S, Kaya S, Hizli O, Hizli P, Nishizaki K, Paparella MM, Cureoglu S (2016) Cochlear histopathologic findings in patients with systemic lupus erythematosus: a human temporal bone study. Otol Neurotol 37(5):593–597. https://doi.org/10.1097/mao.0000000000001017
15. Maimon N, Hanly PJ (2010) Does snoring intensity correlate with the severity of obstructive sleep apnea? J Clin Sleep Med 6(5):475–478