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Original Scientific Paper

AUDIOLOGICAL PROFILE OF OSA PATIENTS: THE EFFECT OF CHRONIC NOCTURNAL INTERMITTENT HYPOXIA ON AUDITORY FUNCTION; A PILOT STUDY

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SUMMARY – Objective: The objective of this study was to investigate whether there is any positive or negative correlation between inner ear function and obstructive sleep apnea (OSA).

Methods: The study included 35 patients diagnosed with moderate or severe OSA and a control group consisting of 25 healthy individuals. Pure-tone audiometry, tympanometry, transient evoked otoacoustic emissions (TEOAE), distortion product otoacoustic emissions (DPOAE) and auditory brainstem response (ABR) tests were performed, evaluated and compared between the two groups.

Results: The air-conduction thresholds at 1000, 4000 and 8000 Hz were higher in OSA patients compared to the controls ($P < 0.05$). Biauricular wave I and wave V latencies in the OSA group were longer than in the control group, but were still within reference values (1.60 ± 0.45 vs 1.43 ± 0.16 ms, $P < 0.001$; 5.52 ± 0.70 vs 5.47 ± 0.19 ms, $P < 0.001$). There was a significant difference in the wave I–V interval between the two groups (4.17 ± 0.36 vs $4.03 \pm 4.03 \pm 0.16$ ms, $P < 0.006$). An analysis of TEOAE signal-to-noise ratio (SNR) mean values revealed a significant difference between the target OSA group and the control group at 1.00 kHz, 1.42 kHz, 2.00 kHz, 2.83 kHz and 4.00 kHz ($P < 0.001$). An analysis of DPOAE SNR mean values revealed a significant difference between OSA patients and the control group at all tested frequencies ($P < 0.027$ for 500 Hz; $P < 0.001$ for 1000 to 8000 Hz).

Conclusion: There is a positive correlation between inner ear function and OSA. High-frequency hearing loss was detected in adults with OSA. The results of otoacoustic emissions showed damage of the cochlear receptor cells.

Keywords: *OSA*; *Inner ear*; *Pure-tone audiometry*; *DPOAE*; *TEOAE*

Introduction

Obstructive sleep apnea (OSA) is the most common sleep-related breathing disorder. It is characterized by obstructions of the upper airway during sleep, resulting in repetitive breathing pauses accompanied

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by oxygen desaturation and arousal from sleep[1,2]. Clinical features of OSA can be divided in daytime symptoms (excessive daytime sleepiness, irritability, morning headache, fatigue) and nighttime symptoms (snoring and apnea, nocturia, gastroesophageal reflux, restless sleep)[2,3]. Patients often experience morning headaches, decreased concentration and an inability to remember and recall information[3,4]. The male sex is an independent risk factor for OSA. Diagnosis is made by anamnesis and clinical examination, but the gold standard for the diagnosis of OSA is polysomnography (PSG) (level I study). The severity of OSA is determined by an index — Apnea-Hypopnea index (AHI) or Respiratory Disturbance Index (RDI) — if PSG is performed. AHI indicates the total number of episodes of sleep apnea and hypopnea divided by the number of hours of sleep. There are four categories: < 5 normal, 5–15 mild, 15–29 moderate, > 30 severe[2].

OSA is now considered a systemic disease. It is known that it leads to arterial hypertension, has an effect on the elasticity of arteries with a consequent risk of heart attack and stroke, and causes chronic renal disease and metabolic disorders[2,3,4]. However, its impact on hearing is still unclear.

The inner ear is very sensitive to hypoxia due to its high metabolic activity and low resistance to changes in oxygen partial pressure[5]. According to the available literature, it has been observed that delayed recurrent hypoxia in OSA patients may cause some damage to cochlear receptor cells[8]. Since cochlear cells are surrounded by the lowest partial pressure of oxygen in the body, any additional change or disturbance in the pressure's balance can further reduce it and lead to damage with a consequent drop in the threshold of auditory sensitivity and potential hearing loss[5,6].

The aim of this study was to examine the differences in auditory function between OSA patients and healthy individuals, and to determine whether there is any positive or negative correlation between inner ear function and OSA.

Patients and methods

The study was designed as original scientific research, i.e. a prospective cohort study in the Department of Otorhinolaryngology and Head and Neck Surgery of University Hospital Center Osijek.

The study included participants of both sexes divided into two groups: a target group of patients newly diagnosed with moderate or severe OSA (N = 35) and a control group consisting of healthy individuals (N = 25), in the period from November 2021 to December 2022. The patients were matched according to age, sex and BMI in both groups to control for sampling bias. The target group consisted of OSA patients who had been previously examined for sleep disorders by a neurologist, filled out STOP-BANG questionnaires and the Epworth sleepiness scale, underwent polysomnography and were diagnosed with moderate or severe OSA according to the AHI index. The STOP-BANG questionnaires and Epworth sleepiness scale were used to establish the control group. Eligibility criteria for the control group were STOP-BANG < 4 and Epworth sleepiness scale < 4.

Exclusion criteria were age under 18 years and over 69 years, presbyacusis, conductive hearing loss, previous neurootological diseases and patients who deviate from the research protocol. The purpose of the study was explained to the participants during the audiological examination, and an informed consent was obtained from all participants.

Audiological diagnostics was performed on all patients using the same devices at the Department of Audiology and Phoniatrics, and it included pure-tone audiometry (PTA), tympanometry, an Auditory Brainstem Response (ABR) and Otoacoustic Emission (OAE). PTA is a subjective method of hearing testing using a device called an audiometer. The test is performed at frequencies of 500–8000 Hz, with earphones placed on the ear for air conduction and vibration on the mastoid, which records bone conduction. The sound is emitted from quieter to louder and the lowest value the respondent hears is considered the hearing threshold[7]. Tympanometry is a method of testing the pressure of the middle ear that is performed by placing a three-channel probe, speaker, microphone and pressure-changing channel in the subject's external ear canal. The method is objective, but movement

and swallowing can affect the results. The test result is a curve which shows how much of the sound released into the external ear canal bounced off the eardrum and returned to the microphone[7]. ABR is a neurootological method for examining the response of cranial nerve VIII and the auditory pathway in the brainstem to acoustic stimulation, i.e. audiometry using evoked brainstem responses. It is performed using a headset which gives a large number (N = 2000) of short (< 1 ms) click stimuli at a speed of 20/s. The electrical activity of cranial nerve VIII and auditory structures in the brainstem is registered; it occurs in the interval of 1.5–10 ms after a repeated stimulus of a certain intensity[7]. Otoacoustic Emission (TEOAE and DPOAE) is an objective method. It is performed by placing a probe with a plastic insertion into the subject's external ear canal and recording a returning sound (an echo) with a sensitive microphone. The echo occurs in cases where the middle ear and cochlea, i.e. cochlear receptor cells, are healthy. TEOAE shows the status of the whole cochlea, while DPOAE examines more specific frequency regions[7].

Ethics

The Ethics Committee of University Hospital Center Osijek (Number: R1/6414/2021, in Osijek, May 11, 2021) and the Ethics Committee of the Faculty of Medicine, J. J. Strossmayer University of Osijek (class: 602-04/2021-08/07, Osijek, July 14, 2021) accepted and approved this research.

Statistics

Data analysis was performed with the SPSS statistical program (Version 22.0 © 2013 IBM SPSS Statistic for Windows, Armok, NY: IBM Corp). Descriptive and inferential statistic methods were used in data analysis. Depending on the satisfaction of the assumptions, the hypotheses were tested using the non-parametric Mann-Whitney test and the parametric t-test. The normality of data distribution was verified by the Shapiro-Wilk test. Before conducting the t-test, the homogeneity of variances was tested using Levene's test. Statistical significance was set at a P value of < 0.05.

Results

The study included 60 participants divided into two groups: 35 patients (70 ears) newly diagnosed with moderate or severe OSA and 25 healthy individuals (50 ears). The median age was 57 years in the OSA group and 58 years in the control group. There were 27 male and 8 female participants in the OSA group, and 20 male and 5 female participants in the control group. The median BMI was 32.8 kg/m^2 in the OSA group and 32.7 kg/m^2 in the control group (Table 1). All 60 respondents completed the STOP-BANG questionnaire and the Epworth sleepiness scale. The median result for the STOP-BANG questionnaire in the OSA group was 6, with a range between 4 and 8. The median result in the

Table 1. Sample characteristics

Characteristics	Descriptive statistics	OSA patients (n = 35)	Control Group (n = 25)
Age	Mean ± SD	55.20 ± 10.74	55.36 ± 10.69
	Median	57.00	58.00
	Range	29.00–69.00	31.00–69.00
Sex	Male (%)	77.1	80.0
	Female (%)	22.9	20.0
BMI (kg/m^2)	Mean ± SD	33.52 ± 6.50	33.14 ± 4.00
	Median	32.80	32.70
	Range	22.20–50.90	26.10–40.10

control group was 2, with a range between 1 and 4. The median result for the Epworth sleepiness scale in the OSA group was 12, with a range between 5 and 24, while in the control group it was 3, with a range between 1 and 4. Higher average scores in both cases were found in the target OSA group. According to the Mann-Whitney test, the differences in the results of the STOP-BANG questionnaire and Epworth sleepiness scale for the target and control group were statistically significant (Table 2). According to the AHI index, 11 patients had moderate OSA and 24 had severe OSA (Table 3).

The air-conduction thresholds at 1000, 4000 and 8000 Hz were higher in OSA patients compared to

Table 2. The significance of the differences in the results of the STOP-BANG Questionnaire and the Epworth Sleepiness scale in the OSA group and control group

Variable	**Descriptive statistics and test results**	**OSA patients (n = 35)**	**Control group (n = 25)**
STOP-BANG Questionnaire	Mean ± SD	5.86 ± 1.48	2.40 ± 0.87
	Median	6.00	2.00
	Range	4.00–8.00	1.00–4.00
	Mean rank	42.71	13.40
	Mann-Whitney test	$Z = 6.472$, $P < 0.001$*	
Epworth sleepiness scale	Mean ± SD	11.60 ± 4.62	2.80 ± 1.00
	Median	12.00	3.00
	Range	5.00–24.00	1.00–4.00
	Mean rank	43.00	13.00
	Mann-Whitney test	$Z = 6.581$, $P < 0.001$*	

* Statistically significant at $P < 0.05$

Table 3. Number and percentage of respondents with moderate and severe apnea

Apnea	**AHI**	**Number**	**%**
Moderate	AHI from 15 to 30	11	31.4
Severe	AHI > 30	24	68.6

Table 4. The significance of the differences in audiogram results (ADG) for both ears in the target OSA group and control group

Variable	**Descriptive statistics and test results**	**OSA patients (n = 70)**	**Control group (n = 50)**
ADG 500 Hz	Mean ± SD (dB)	14.14 ± 9.25	12.20 ± 3.37
	Median (dB)	10.00	10.00
	Range (dB)	10.00–65.00	10.00–25.00
	t-test	$t = 1.614$, $P = 0.110$	
ADG 1000 Hz	Mean ± SD (dB)	18.79 ± 13.20	14.70 ± 4.78
	Median (dB)	15.00	15.00
	Range (dB)	10.00–75.00	10.00–25.00
	t-test	$t = 2.380$, $P = 0.019$*	

Variable	Descriptive statistics and test results	OSA patients (n = 70)	Control group (n = 50)
ADG 2000 Hz	Mean ± SD (dB)		19.90 ± 14.30
	Median (dB)	10.00	15.00
	Range (dB)	10.00–85.00	10.00–75.00
	t-test	$t = 0.087$, $P = 0.931$	
ADG 4000 Hz	Mean ± SD (dB)	34.50 ± 19.77	27.60 ± 14.26
	Median (dB)	35.00	27.50
	Range (dB)	10.00–75.00	10.00–75.00
	t-test	$t = 2.221$, $P = 0.028$*	
ADG 8000 Hz	Mean± SD (dB)	41.20 ± 24.21	33.10 ± 15.45
	Median (dB)	40.00	32.50
	Range (dB)	10.00–105.00	10.00–75.00
	t-test	$t = 2.234$, $P = 0.027$*	

* Statistically significant at $P < 0.05$

Table 5. The significance of differences in wave I and V latency values and the I-V interval for both ears of the target OSA group and control group

Variable	Descriptive statistics and test results	Patients with OSA (n = 70)	Control group (n = 50)
Wave I latency (ms)	Mean ± SD	1.60 ± 0.45	1.43 ± 0.16
	Median	1.53	1.39
	Range	1.20–4.17	1.20–1.90
	Mean rank	69.91	47.32
	Mann-Whitney test	$Z = 3.512$, $P < 0.001$*	
Wave V latency (ms)	Mean ± SD	5.52 ± 0.70	5.47 ± 0.19
	Median	5.70	5.47
	Range	1.80–6.20	5.10–5.87
	Mean rank	70.62	46.33
	Mann-Whitney test	$Z = 3.772$, $P < 0.001$*	
Wave I–V interval (ms)	Mean ± SD	4.17 ± 0.36	4.03 ± 0.16
	Median	4.16	4.07
	Range	3.57–5.97	3.50–4.30
	Mean rank	67.83	50.24
	Mann-Whitney test	$Z = 2.732$, $P = 0.006$*	

* Statistically significant at $P < 0.05$

controls. According to the t-test, the differences in the results for the target and control group were statistically significant (Table 4).

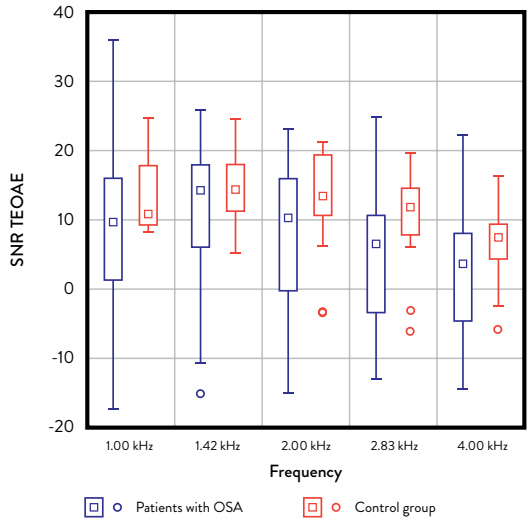
Biauricular wave I and wave V latencies in the OSA group were longer than in the control group, but were still within reference values. There was a significant difference in the wave I–V interval between the two

groups. Higher average scores for wave I latency, wave V latency and the wave I–V interval were found in the target OSA group. According to the Mann-Whitney test, these differences were statistically significant (Table 5).

An analysis of TEOAE SNR mean values showed a significant difference between the target OSA group and the control group at 1.00 kHz, 1.42 kHz, 2.00 kHz, 2.83 kHz and 4.00 kHz.

According to the t-test, the differences in the results for the target and control group were statistically significant (Table 6, Picture 1).

An analysis of DPOAE SNR mean values showed a significant difference between OSA patients and the control group at all tested frequencies. According to the t-test, these differences were statistically significant (Table 7).



Picture 1. Box-plot diagrams of the distribution of TEOAE scores for both ears in the OSA group and control group

Table 6. The significance of the differences in TEOAE results for both ears in the target OSA group and control group

Variable	Descriptive statistics and test results	Patients with OSA (n = 70)	Control group (n = 50)
SNR 1.00 kHz TEOAE	Mean ± SD	8.20 ± 10.49	13.25 ± 5.44
	Median	9.75	10.85
	Range	-17.30–36.20	8.00–25.00
	t-test	t = -3.439, P = 0.001*	
SNR 1.42 kHz TEOAE	Mean ± SD	11.71 ± 9.23	15.05 ± 4.60
	Median	14.25	14.35
	Range	-15.00–25.90	5.10–24.70
	t-test	t = -2.610, P = 0.010*	
SNR 2.00 kHz TEOAE	Mean ± SD	7.75 ± 9.81	13.84 ± 5.77
	Median	10.30	13.30
	Range	-15.00–23.30	-3.40–21.30
	t-test	t = -4.262, P < 0.001*	
SNR 2.83 kHz TEOAE	Mean ± SD	4.45 ± 9.06	11.69 ± 5.51
	Median	6.35	11.80
	Range	-12.90–24.80	-6.00–19.80
	t-test	t = -5.422, P < 0.001*	
SNR 4.00 kHz TEOAE	Mean ± SD	2.14 ± 8.24	6.75 ± 5.31
	Median	3.65	7.40
	Range	-14.50–22.40	-5.80–16.60
	t-test	t = -3.729, P < 0.001*	

* Statistically significant at P < 0.05

Table 7. The significance of the differences in DPOAE results for both ears in the target OSA group and control group

Variable	Descriptive statistics and test results	Patients with OSA (n = 72)	Control group (n = 50)
SNR 500 Hz	Mean ± SD	-0.15 ± 6.42	2.41 ± 5.82
	Median	0.40	3.75
	Range	-20.20–11.40	-9.60–13.70
	t-test	$t = -2.238$, $P = 0.027$*	
SNR 1000 Hz	Mean ± SD	3.61 ± 8.04	11.08 ± 6.02
	Median	3.10	11.15
	Range	-10.00–20.20	-7.00–22.30
	t-test	$t = -5.812$, $P < 0.001$*	
SNR 1500 Hz	Mean ± SD	9.04 ± 8.02	15.31 ± 6.53
	Median	10.30	15.05
	Range	-11.10–40.70	0.60–28.90
	t-test	$t = -4.553$, $P < 0.001$*	
SNR 2000 Hz	Mean ± SD	6.78 ± 10.51	14.44 ± 5.92
	Median	10.10	13.25
	Range	-18.10–32.60	-7.90–24.80
	t-test	$t = -5.073$, $P < 0.001$*	
SNR 3000 Hz	Mean ± SD	6.27 ± 8.93	15.07 ± 6.98
	Median	9.15	14.45
	Range	-18.80–23.80	-5.00–30.10
	t-test	$t = -6.056$, $P < 0.001$*	
SNR 4000 Hz	Mean ± SD	5.57 ± 11.13	14.08 ± 8.64
	Median	5.05	11.50
	Range	-17.60–26.90	-1.60–34.40
	t-test	$t = -4.706$, $P < 0.001$*	
SNR 5000 Hz	Mean ± SD	4.09 ± 10.61	13.01 ± 11.63
	Median	3.15	11.95
	Range	-17.80–29.30	-24.40–37.30
	t-test	$t = -4.363$, $P < 0.001$*	
SNR 6000 Hz	Mean ± SD	2.47 ± 9.88	12.28 ± 12.05
	Median	1.20	11.50
	Range	-14.10–24.10	-7.90–60.00
	t-test	$t = -4.890$, $P < 0.001$*	
SNR 7000 Hz	Mean ± SD	0.65 ± 8.82	6.41 ± 8.48
	Median	0.30	6.15
	Range	-18.10–20.50	-24.40–23.40
	t-test	$t = -3.587$, $P < 0.001$*	
SNR 8000 Hz	Mean ± SD	0.15 ± 7.31	4.75 ± 6.61
	Median	-0.10	4.75
	Range	-23.20–12.70	-7.80–17.20
	t-test	$t = -3.535$, $P < 0.001$*	

* Statistically significant at $P < 0.05$

Discussion

OSA has well-known negative effects on multiple systems of the human body and might also affect the cochlear system, but the characteristics of OSA and the type of its effects on cochlear elements are not clear[8]. Many studies investigated the functional state of the auditory nervous systems in OSA patients[1,8,9,13,14,15].

The hypothesis of this study was that OSA patients have damaged cochlear receptor cells when compared to the control group. It was also considered that hypoxia, which is present in moderate and severe OSA, may be a risk-factor for auditory system dysfunction[9].

The cochlea derives its blood supply from a single terminal artery source and, having inadequate collateral circulation, is highly reliant on the amount of oxygen it receives[10,11]. Hence, recurrent episodes of apnea cause damage to cochlear cells. Over the years, several studies have been conducted that studied the pathogenesis of damage to the cochlea. Most support the fact that oxidative stress is responsible for the damage to hearing cells[12]. The outer hair cells of the basal turn of the cochlea are more vulnerable to free radical injury due to their substantially lower activity of glutathione-related antioxidant enzyme[12].

A study by Seo et al. focused on auditory dysfunction aggravated by the severity of OSA. It suggested that damage to the sensory epithelium of the inner ear is a potential mechanism leading to hearing loss. The authors used mice as a model for OSA and performed a histopathological assessment to figure out the mechanism of hearing impairment in OSA[13].

Human cochlear tissue is not available for direct analysis. We can get information about its functioning using OAE; a type of sound energy produced in the cochlea. It can be recorded in the external auditory canal and can also reflect the functional status of outer hair cells in the cochlea. OAE can be categorized as spontaneous and evoked, depending on the presence or absence of external stimulation signal induction. The most common OAEs are TEOAE and DPOAE[14].

Some previous publications suggested that the negative effects of OSA on central auditory pathways might be the main mechanism of hearing impairment in OSA patients[15]. By contrast, most publications focusing on the hearing system in OSA patients reported that cochlear ischemia caused by chronic intermittent hypoxemia and cochlear inflammation caused by the pro-inflammatory base of OSA might precipitate hearing impairment in OSA patients[5,13].

A study of OSA patients conducted by Casale et al. found that air-conduction thresholds were elevated in the OSA group when compared to controls; in the TEOAE test, the authors observed significantly higher values than in the control group[16]. Iriz et al. reported that the presence of OSA does not affect hearing thresholds[15]. A study conducted by Kayabasi et al. found that moderate OSA affects high-frequency hearing functions and severe OSA has significant effects on all hearing functions[8].

In this study, we determined sensorineural hearing loss in patients with OSA. When the different frequencies were evaluated separately, hearing threshold values in OSA patients were significantly higher compared to controls at 1000, 4000 and 8000 Hz.

ABR reflects the bioelectrical potential arising from the auditory nerve and brainstem in response to acoustic sounds: the click[9,14]. It consists of a series of peaks within the first 10 ms after the onset of the stimulus, labeled with numerals I-V. Wave V is the most stable component. The latency and amplitude of wave V and wave I, and interpeak latencies between wave I and V are often used to localize the lesions of auditory nerve pathways.

Li et al. found that high-frequency hearing loss was detected in adults with severe OSA, and the latencies of wave I and wave V of ABR were prolonged[14]. The results of a study by Fu et al. found no significant differences between the OSA group and the control group in terms of the peak latencies (I-III-V) and interpeak latencies of waves I-V[9]. According to the authors, the reasons for this may be ascribed to a small sample size (31 patients), or, more likely, to the fact that auditory impairment caused by hypoxemia is a long, persistent process[9].

In our study, we found that biauricular wave I and wave V latencies in the OSA group were longer than in the control group, but still within reference values (up to 2 ms for wave I and up to 6.2 ms for wave V). There was also a significant difference in the wave I–V intervals between the two groups.

Matsmura et al.[17] showed that inner ear damage caused by severe OSA manifests as a decrease of DPOAE amplitude. A study conducted by Li et al.[14]

revealed that DPOAE amplitude is significantly reduced in OSA patients, although no hearing loss was observed. A possible reason for this phenomenon could be the fact that chronic cochlear hypoxia in OSA patients may damage the outer hair cells in the cochlea. It has also been observed that the change in DPOAE amplitude manifests earlier than changes in hearing thresholds[18]. In our study, we analyzed SNR in TEOAE and DPOAE for different frequencies (500–8000 Hz in DPOAE and 1.0–4.0 kHz in TEOAE) and found a significant difference between the target OSA group and the control group in all frequencies.

Although this sample may be representative of the normal population, the study still had some limitations. For example, only 35 OSA patients and 25 healthy individuals (and of those only 8 and 5 female participants, respectively) were included. More subjects with confirmed OSA should be studied to reinforce our results. Based on the results, we recommend that all OSA patients pay more attention to their auditory function or dysfunction and seek a professional consultation. Also, clinicians should be aware of this risk factor for hearing dysfunction and treat OSA more actively to prevent hearing degeneration.

Conclusion

The hearing system is affected to varying degrees in OSA patients. A high-frequency hearing loss was detected in adults with OSA. OAE results showed damage of the cochlear receptor cells. The present study showed that abnormalities of auditory pathways in patients with moderate or severe OSA can be electrophysiologically observed in their early stages. The combined use of pure-tone audiometry and OAE may help recognize subclinical hearing loss.

Conflict of interest

The authors decline no conflict of interest.

Abbreviations

OSA – Obstructive Sleep apnea
AHI – apnea/hypopnea index
PTA – pure-tone audiometry
ABR – auditory brainstem response
OAE – otoacoustic emission
TEOAE – transient evoked otoacoustic emission
DPOAE – distortion product otoacoustic emission
SNR – signal to noise ratio

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SAŽETAK

AUDIOLOŠKI PROFIL BOLESNIKA OBOLJELIH OD OSA-E: UČINAK KRONIČNE INTERMITENTE HIPOKSIJE TIJEKOM NOĆI NA SLUŠNU FUNKCIJU: PILOT STUDIJA

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Cilj: Cilj istraživanja bio je ispitati postoji li pozitivan ili negativan odnos između funkcije unutarnjeg uha i opstrukcijske apneje tijekom spavanja (OSA).

Metode: U studiju je uključeno 35 pacijenata oboljelih od umjerene ili teške OSA i 25 zdravih ispitanika koji su činili kontrolnu skupinu. Svim je ispitanicima učinjena tonska audiometrija, timpanometrija, prolazno izazvana otoakustička emisija (TEOAE), otoakustička emisija koja je produkt distorzije (DPOAE) i slušni evocirani potencijali.

Rezultati: Prag zračne vodljivosti na 1000, 4000 i 8000 Hz bio je veći u pacijenata s OSA u usporedbi s kontrolnom skupinom ($P < 0.05$). Latencije I i V vala oba uha u OSA skupini bile su dulje nego u kontrolnoj skupini, no i dalje su bile u referentnim vrijednostima (1,60 ± 0,45 vs 1,43 ± 0,16 ms, $P < 0,001$; 5,52 ± 0,70 vs 5,47 ± 0,19 ms, $P < 0,001$). Razlika između intervalnih latencija I do V vala OSA skupine i kontrolne skupine bila je statistički značajna (4,17 ± 0,36 vs 4,03 ± 4,03 ± 0,16 ms, $P < 0,006$). Rezultati analize omjera signala i buke (eng. *signal-to-noise ratio*, SNR) kod TEOAE pokazali su statistički značajnu razliku između ciljne OSA skupine i kontrolne skupine na 1,00 kHz, 1,42 kHz, 2,00 kHz, 2,83 kHz i 4,00 kHz ($P < 0,001$). Rezultati analize SNR kod DPOAE pokazali su statistički značajnu razliku između pacijenata s OSA i kontrolne skupine na svim ispitivanim frekvencijama ($P < 0,027$ za 500 Hz; $P < 0,001$ za 1000 do 8000 Hz).

Zaključak: Utvrđena je pozitivna korelacija između OSA i funkcije unutarnjeg uha. Kod pacijenata s OSA postoji oštećenje sluha u visokim frekvencijama. Rezultati otoakustičke emisije pokazali su oštećenje receptorskih stanica pužnice.

Ključne riječi: *OSA*; *Unutarnje uho*; *Tonska audiometrija*; *DPOAE*; *TEOAE*