

Effects of Hypoxia on the Vestibular System in Obstructive Sleep Apnea Syndrome Observed on a Video Head Impulse Test

ABSTRACT

Objective: The aim of this research was to investigate the effects of hypoxia, observed in patients with obstructive sleep apnea (OSA) syndrome, on vestibular functions through the application of video head impulse test (v-HIT).

Methods: Patients with sleep apnea/snoring complaints and who had undergone polysomnography (PSG) between January 10, 2019, and January 10, 2020, received a full otorhinolaryngological examination and v-HIT in this prospective cohort study. Polysomnography findings of 83 patients with appropriate criteria for the study (age, sex, body mass index (BMI), apnea-hypopnea index (AHI), and mean oxygen saturation) and v-HIT findings were compared.

Results: Patients included in the study were divided into 4 groups according to the AHI scores. When age, gender, BMI, AHI, and mean oxygen saturation data were examined, only AHI scores showed statistically significant differences ($P = .000$). There was no statistically significant difference present between the groups in terms of mean vestibulo-ocular reflex (VOR) gains, mean asymmetry, overt and covert saccades in the right ear, and overt saccades in the left ear. There was a statistically significant difference between the groups in the covert saccade in the left ear ($P = .014$). Of all patients included in the study, 72 patients (86.5%) had pathological VOR gains and/or at least 1 overt/covert saccade.

Conclusion: Our study with v-HIT concluded that the vestibular system is affected just as other systems in patients diagnosed with OSA syndrome. The literature review has proved that similar results were achieved in studies evaluating the vestibular system.

Keywords: Obstructive sleep apnea syndrome, snoring, vestibular system, video head impulse test, polysomnography

INTRODUCTION

Obstructive sleep apnea syndrome (OSA) is a syndrome characterized by recurrent episodes of complete (apnea) or partial (hypopnea) upper airway obstruction during sleep, decreased blood oxygen saturation (SpO_2), and excessive daytime sleepiness. It is seen in 2-4% of middle-aged people in the community.¹

Oxygen desaturation due to apnea-hypopnea attacks in OSA is observed. Although vasa nervorum damage in the early stages of hypoxemia is compensated, long-term development of peripheral neuropathy is inevitable.²⁻⁴ Ischemia-reperfusion occurs in the tissues as a result of repeated rise and fall attacks in oxygen saturation. These attacks initiate oxidative stress, leading to loss of endothelial function, particularly impaired vasodilation ability due to nitric oxide loss in vascular walls.⁵

There are studies in the literature on cardiovascular, neurological, pulmonary, metabolic, and hematological diseases that may occur due to hypoxia caused by OSA.⁶ However, there is limited information about neurotological disorders. One of the most comprehensive studies to evaluate the vestibular system is conducted by Galliani et al. This study also used caloric stimulation test, videonystagmography (VNG), pure tone audiometry, and auditory brainstem response (ABR) audiometry to evaluate the audiovestibular system in OSA patients, and resulted in hyporeflexia and asymmetry in caloric test applied



Muhammet Fatih Topuz¹
Fatih Oğhan¹
Gönül Akdağ²
Pınar Yıldız Gülhan³
Onur Erdoğan⁴
Özlem Arık⁵
Aykut Ceyhan⁶
Nurullah Türe¹
Ali Güvey¹

¹Department of Otorhinolaryngology, Kutahya University of Health Sciences School of Medicine, Kütahya, Turkey

²Department of Neurology, Kutahya University of Health Sciences School of Medicine, Kütahya, Turkey

³Department of Chest Medicine, Duzce University School of Medicine, Konuralp, Duzce, Turkey

⁴Department of Otorhinolaryngology, Private Olbamed Hospital, Silifke, Mersin, Turkey

⁵Department of Biostatistics, Kutahya University of Health Sciences School of Medicine, Kütahya, Turkey

⁶Department of ORL Kutahya, Kutahya Evliya Celebi Research Hospital, Kütahya, Turkey

Cite this article as: Topuz MF, Oğhan F, Akdağ G, et al. Effects of hypoxia on the vestibular system in obstructive sleep apnea syndrome observed on a video head impulse test. *ENT Updates*. 2021;11(3):148-152.

Corresponding author:
Fatih Oğhan
Email: fatihoghan@hotmail.com
Received: June 2, 2021
Accepted: July 30, 2021



to patients with severe OSA conditions.⁷ The caloric stimulation test is used to evaluate the vestibular system and has been used and trusted for many years, in which a very low frequency (0.003 Hz) is utilized to inspect lateral semicircular canal function. However, in daily life, all components of the vestibular system are used altogether. Therefore, the caloric test alone is insufficient because it cannot evaluate the high frequency and cannot test all semicircular channels. Furthermore, the caloric stimulation test is inconvenient as it inflicts nausea and dizziness in the patient. The video head impulse test (v-HIT), which is achieved by a sudden head impulse on the horizontal plane and tests the lateral canal induced vestibulo-ocular reflex (VOR), is used to evaluate high frequencies. Video head impulse test is a more efficient physiological test since it can evaluate high frequencies.⁸ The purpose of this study was to evaluate the effects of hypoxia on the vestibular system using v-HIT in patients diagnosed with OSA syndrome.

METHODS

Patients

This prospective cohort study was conducted between January 10, 2019, and January 10, 2020, in the Otorhinolaryngology Clinic of Kutahya Health Sciences University Evliya Celebi Training and Research Hospital with the permission of Kutahya Health Sciences University Clinical Research Ethics Committee dated January 9, 2019, and numbered 2019/01-2. All patients included in the study signed an informed consent form. The study was conducted as per the Declaration of Helsinki.

Our study consisted of 83 patients, between 18 and 65 years of age with sleep apnea/snoring complaints, who consulted our neurology/otorhinolaryngology clinics, underwent polysomnography (PSG), gave anamnesis, and received full otorhinolaryngologic examination, followed by v-HIT.

The following patients were excluded from the study: patients with chronic diseases (diabetes, hypertension, hypercholesterolemia, chronic renal failure, cardiovascular diseases, chronic airway diseases, and chronic parenchymal lung disease), alcohol addicts, patients with tinnitus, conductive/sensorineural hearing loss, otitis media, neurological diseases (multiple sclerosis and poliomyelitis), intracranial tumor and vestibular schwannoma, vertigo, previous ear surgery, patients with ototoxic drug usage history, and patients currently receiving sleep apnea treatment. Patients who refused to participate in the study and patients refusing to undergo PSG and/or v-HIT were also excluded.

Polysomnography

Polysomnography results were evaluated by the same researcher following the Clinical Practice Guideline by the American Academy of Sleep Medicine. Polysomnography recordings were performed under the supervision of a sleep technologist, using a PSG device with at least 16 channels (Respironics Inc, Germany). The PSG device had 6 electroencephalography (EEG) (F4-M1, C4-M-1, O2-M1, F3-M2, C3-M2, O2-M2), 2 electro-oculography (EOG), 3 mentalis/submentalis electromyography, oximeter, snoring signal, body position sensor, nasal pressure/current signal (thermistor and nasal cannula), respiratory effort (thorax-abdomen bands), 2 EMG (tibialis anterior), and electrocardiography (single-derivation) recording features. Apnea is the cessation of airflow through mouth and nose for 10 seconds

or more. Hypopnea is the reduced depth of breathing at a rate above 30% longer than 10 seconds, accompanied by desaturation and/or awakening. Apnea-Hypopnea Index (AHI) (I) was defined as the average number of apnea and hypopnea during sleep per hour. The patients were divided into 4 groups based on AHI according to PSG results (simple snoring/normal (AHI <5), mild (AHI = 5-15), moderate (AHI = 16-30), and severe (AHI = 30).⁹

Head Impulse Test

The video head impulse test (Interacoustics A/S, Denmark) was performed with the EyeSeeCam system, a lightweight goggle attached to a video-oculography camera with a sampling rate of 250 Hz. The patients were instructed to fix their gaze at a small target object at 1.2 m distance. Whilst the patient gazed at the target object, the patient's head was rotated on the horizontal plane for 150-200 ms, 10-20° at low amplitude, 2000-6000°/s² head acceleration, and 200°/s head rotation speed.¹⁰ A total of 10 head impulses were done in both directions. Impulses were performed in such a way that the patient was not able to anticipate the time and the direction of the impulse. The patient's VOR gain was calculated by the device. Vestibulo-ocular reflex gain normal ranges for the lateral canal were assumed between 0.8 and 1.2.¹¹ Gains outside of this range were marked pathological. The saccades emerged during the head impulse were considered "covert" saccades; the saccades after the head impulse was considered "overt" saccades. The presence of any covert and/or overt saccades was marked as a pathological v-HIT response.¹¹

Statistical Analysis

The application results were achieved using the Statistical Package for the Social Sciences (SPSS) version 20.0 (IBM SPSS Corp.; Armonk, NY, USA), independent sample t-test, Levene's test, and one-way analysis of variance (ANOVA) test. *P* less than .05 were considered statistically significant.

RESULTS

Of the 83 patients included in the study, 56 (67%) were male and 27 (33%) were female. The mean age was 52.49 (-130.1) and mean O₂ saturation was 89.85 (range: 77-98). The demographic distribution of patients is summarized in Table 1, as indicated in the American Academy of Sleep Medicine Scoring Manual. When the intergroup age, sex, body mass index (BMI), AHI, and mean oxygen saturation data were analyzed, only AHI scores had statistically significant differences (*P* = .000); age, BMI, and mean oxygen saturation had no statistically significant differences.

Video head impulse test results of the patients were evaluated; the mean right ear gain was calculated as 1.05 (range: 0.54-1.3) and the mean left ear gain was calculated as 0.99 (range: 0.58-1.36). There was no statistically significant difference between the groups in terms of mean VOR gains in right and left ears (*P* > .05). There was no statistically significant difference between the 2 groups in terms of gain averages (*P* > .05). The mean asymmetry between the 2 ears was found to be 4.71 (range: 0-21). When the mean asymmetry values of both ears were compared, no statistically significant difference was observed (*P* > .05).

The mean overt saccade was 2.80 (range: 0-12) and the mean covert saccade was 1.12 (range: 0-9) in the right ear. There was no statistically significant difference in mean overt saccade and

Table 1. The Demographic Distribution of Patients

OSA Groups	AHI	Gender		Age	BMI	Mean O ₂ Saturation
		Male	Female			
Simple snoring/normal (AHI: <5)	2.67±1.46 (range: 0.8-4.6)	6	1	50.7±13.1 (range: 41-78)	28±3.94 (range: 22.9-32.4)	93.4±1.51 (range: 91-97)
Mild OSA (AHI: 5-15)	9.36±3.03 (range: 5.6-14.4)	6	5	48±12.2 (range: 33-70)	26.2±3.33 (range: 19.8-31.7)	92.45±1.13 (range: 91-95)
Moderate OSA (AHI: 15-30)	22.14±3.86 (15.6-29.6)	5	5	50.2±6.4 (range: 37-58)	31.6±8.25 (range: 51.4-22.5)	91.9±1.91 (range: 90-98)
Severe OSA (AHI: >30)	64.05±26.1 (range: 22.1-130.1)	39	16	54±8.43 (36-73)	30.7±4.62 (range: 42.2-21.6)	88.8±3.01 (77-94)
<i>P</i>	.000			.842	.113	.435

OSA, obstructive sleep apnea syndrome; AHI, apnea-hypopnea Index; BMI, body mass index.

covert saccade values in the right ear in groups separated according to AHI scores ($P = .186$; $P = .561$). The mean overt saccade was 3.35 (range: 0-10) and the mean covert saccade was 1.24 (range: 0-8) in the left ear. There was no statistically significant difference in mean overt saccade values between the groups in the left ear, but there was a statistically significant difference in covert saccade values ($P = .875$; $P = .014$). The v-HIT results of the patients according to AHI scores are summarized in Table 2.

While 12 (14.5%) of the patients included in the study had pathological VOR gains, 69 (83%) had at least 1 overt/covert saccade. A total of 9 patients (10.8%) had both pathological VOR gains and at least 1 overt/covert saccade. Only 11 (13.5%) patients had normal VOR gains in both ears and no overt/covert saccades. Of these patients, 2 had simple snoring complaints, 3 were moderate, and 6 were severe OSA patients (Table 3).

Table 2. Video Head Impulse Test Results of the Patients According to Apnea-Hypopnea Index Scores

OSA Groups	Right v-HIT Gain	Left v-HIT Gain	Asymmetry	Right Overt Saccade	Right Covert Saccade	Left Covert Saccade	Left Covert Saccade
Simple snoring/normal (AHI: <5)	0.878±0.17 (range: 0.54-1.09)	0.867±0.179 (range: 0.58-1.10)	3.57±1.81 (range: 1-7)	0.71±1.1 (range: 0-3)	0.42±0.79 (range: 0-2)	2.71±2.98 (range: 0-7)	0.57±0.79 (range: 0-2)
Mild OSA (AHI: 5-15)	0.958±0.12 (range: 0.78-1.19)	0.988±0.15 (range: 0.65-1.2)	6.09±4.99 (range: 0-16)	2.8±2.3 (range: 0-6)	0.7±1.49 (range: 0-4)	3.3±3.8 (range: 0-10)	2.1±2.37 (range: 0-5)
Moderate OSA (AHI: 15-30)	0.99±0.787 (range: 0.84-1.08)	1.04±0.5 (range: 0.97-1.13)	3.4±3.06 (range: 0-10)	2.1±2.68 (range: 0-7)	0.2±0.42 (range: 0-1)	3.43±3.6 (range: 0-9)	0 (range: 0-0)
Severe OSA (AHI: >30)	0.958±0.15 (range: 0.61-1.3)	0.997±0.15 (range: 0.58-1.36)	4.82±4.77 (range: 0-21)	3.2±3.16 (range: 0-12)	1.46±2.2 (range: 0-9)	3.4±3.08 (range: 0-10)	1.4±2.17 (range: 0-8)
<i>P</i>	.436	.102	.499	.186	.561	.875	.014

OSA, obstructive sleep apnea syndrome; AHI, apnea-hypopnea index; v-HIT, video head impulse test.

Table 3. Distribution of Pathological Video Head Impulse Test Findings According to OSA Groups

OSA Groups	Number of Patients Without Pathological v-HIT Findings	Number of Patients with Pathological v-HIT Findings			Total Number of Patients
		Pathological v-HIT Gain	Pathological Saccade	Pathological v-HIT and Saccade	
Simple snoring/normal (AHI <5)	2	-	4	1	7
Mild OSA (AHI: 5-15)	-	1	9	1	11
Moderate OSA (AHI: 15-30)	3	-	7	-	10
Severe OSA (AHI: >30)	6	2	40	7	55
Total	11 (13%)	3 (4%)	60 (72%)	9 (11%)	83

OSA, obstructive sleep apnea syndrome; AHI, apnea-hypopnea index; v-HIT, video head impulse test.

DISCUSSION

The basic principle of vestibular tests performed to evaluate the vestibular system is to compare the functions of both vestibular organs. The caloric test, vestibular evoked myogenic potentials (VEMP), and v-HIT are the main tests performed for this purpose.

The caloric test, which is used for many years to evaluate VOR, evaluates the lateral canal and tests VOR especially at low frequencies.¹² Vestibular evoked myogenic potentials is a newer vestibular test compared to the caloric test. This test evaluates the translational VOR system via the otolith-ocular reflex.¹³

Video head impulse test was first described by Halmagyi and Curthoys in the evaluation of VOR.¹⁴ Video head impulse test is a relatively easier method to evaluate the vestibular system disorders that can be applied at bedside and has been widely used.⁸ One of the major advantages of v-HIT is its ability to be performed in a short period, its ability to evaluate all 3 semicircular canals, and its high tolerability by the patients. Video head impulse test is also useful for detecting saccades that cannot be detected with the naked eye. It tests the VOR by recording eye movements during high-speed head movements. It is a physiologically more suitable high-frequency test compared to the caloric test. Although there are claims that it may replace the caloric test, there are no conclusive results to confirm these claims.

Patients with OSA have symptoms of a decrease in oxygen saturation in the blood due to repetitive apnea-hypopnea attacks, and even hypoxia in severe cases. Hypoxia causes serious damage to all organs by affecting their physiology through various mechanisms. The oxidative stress caused by apnea attacks leads to loss of endothelial function and impaired vasodilation due to nitric oxide loss in vascular walls.⁵ This leads to deterioration of the blood-brain barrier.¹⁵ Furthermore, triggered by inflammatory processes, the vasa nervorum in the central and peripheral nervous system is damaged.

The transduction mechanism of the inner ear and the conduction of nerve impulses depend on high oxygen supply; various studies in the literature have suggested that resulting apnea-hypopnea may affect the formation and conduction of nerve impulses at the hearing system level.^{16,17} Hildesheimerve et al.¹⁸ applied hypoxic blood transfusion to rats, measured cochlear action potential threshold and action potential amplitude values before and after hypoxia, and noted a statistically significant difference. In their study on 39 patients with OSA and 21 patients with simple snoring complaints, Casale et al.¹⁹ found that their pure tone audiometry mean values are higher than the control group. The reason for this result was hypoxia due to OSA. Nevertheless, Munchnick et al.²⁰ compared the ABR results of patients with mild, moderate, and severe OSA and found that first, third and fourth wave latency values were significantly prolonged compared to the control group. When we examined studies on the vestibular system, which is another important function, we found similar findings. Gallani et al.⁷ found hyporeflexia and asymmetry in the caloric stimulation tests on severe OSA patients and attributed this to the sensitivity of the posterior labyrinth system to hypoxia. The study of Mutlu et al.²¹ demonstrated that

severe OSA patients had brainstem damage caused by hypoxia and VEMP responses were affected due to sacculocolic reflex.

As it is seen, various studies are investigating the relationship between OSA and neurotological diseases in terms of audiological and vestibular systems. However, there are no studies in the literature investigating this relationship using v-HIT.

In our study, we examined the relationship between OSA and neurotological diseases using relatively newer v-HIT and have attained results consistent with the literature. The incidence of pathological v-HIT findings in OSA patients was 83% in our study. Although there was no statistically significant difference between the severity of OSA and the incidence of pathological v-HIT, a significant difference was found compared to the healthy population.

We also discovered that the presence of left covert saccade in the left ear was significantly lower in patients with OSA compared to the right. Unfortunately, the clinical significance and the causing factors are yet to be found. A similar finding is also observed in a study with VEMP by Mutlu et al.²¹ In patients with OSA, VEMP wave (p1n1, p2n2) amplitude was reduced, but no significant change was observed in wave latency; however, no comments were made on clinical importance.

Gallani et al.⁷ commented that the disequilibrium caused by hypoxia and the peripheral vestibular system in OSA patients is very well balanced by the central vestibular system. Although our patients manifested such high rates of pathological findings, the rate of symptomatic complaints was not so high. Our study shows that the vestibular system is affected due to hypoxia in OSA patients, and other studies in the literature and Gallani et al. support the above comments.

Legal boundaries limited our study, and therefore we were not able to include healthy volunteers without any snoring/sleep apnea symptoms. Another limitation was that the number of patients with simple snoring complaints was low. The main reason for this low number of patients is that simple snoring is not perceived as a serious health condition; these patients were also reluctant to undergo PSG. Therefore we only had a limited number of patients with simple snoring complaints that underwent PSG. Furthermore, there is no clear data on how long OSA patients had sleep apnea (the patients were not able to answer these questions clearly); hence no clear data was present on how long the vestibular system had been subjected to hypoxic damage. Undoubtedly, future studies, with larger sample groups, including healthy volunteers and OSA patients with different levels of severity, using batteries testing the whole vestibular system (the caloric test, VEMP, VNG, and v-HIT) will unravel the unknown.

CONCLUSION

This is the first study to evaluate the vestibular system in patients with OSA using v-HIT. As a result, this study demonstrated that the vestibular system is affected by sleep apnea. The literature review has proved that similar results were achieved in studies evaluating the vestibular system. However, no correlation was observed between the increase in pathological v-HIT findings

and the increase in the severity of OSA. Again, we think further research is required to explain the significantly lower presence of saccades in the left peripheral vestibular system compared to the right peripheral vestibular system.

Ethics Committee Approval: Ethics committee approval was received from the Kutahya Health Sciences University Clinical Research Ethics Committee (January 9, 2019, and numbered 2019/01-2).

Informed Consent: Written informed consent was obtained from all participants who participated in this study.

Peer Review: Externally peer-reviewed.

Author Contributions: Concept - M.F.T.; Design - M.F.T.; Supervision - F.O.; Resource - O.E., A.C., N.T., A.G.; Materials - G.A., A.C., M.F.T.; Data Collection and/or Processing - O.E., O.A., A.C.; Analysis and/or Interpretation - O.E., A.C., A.G.; Literature Search - P.Y.G., N.T.; Writing - G.A., P.Y.G., N.T.; Critical Reviews - F.O.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993;328(17):1230-1235. [\[CrossRef\]](#)
2. Fanfulla F, Grassi M, Taurino AE, D'Artavilla Lupo N, Trentin R. The relationship of daytime hypoxemia and nocturnal hypoxia in obstructive sleep apnea syndrome. *Sleep*. 2008;31(2):249-255. [\[CrossRef\]](#)
3. Bradley TD, Floras JS. Obstructive sleep apnea and its cardiovascular consequences. *Lancet*. 2009;373(9657):82-93. [\[CrossRef\]](#)
4. Redline S, Tishler PV. The genetics of sleep apnea. *Sleep Med Rev*. 2000;4(6):583-602. [\[CrossRef\]](#)
5. Cofta S, Wysocka E, Piorunek T, et al. Oxidative stress markers in the blood of persons with different stages of obstructive sleep apnea syndrome. *J Physiol Pharmacol*. 2008;59(suppl 6):183-190.
6. Tapan ÖO, Tapan U, Kılıç ÜS. Effects of hypoxia on the eye in patients with obstructive sleep apnea syndrome. *J Turk Sleep Med*. 2016;2:43-47.
7. Gallina S, Dispenza F, Kulamarva G, Riggio F, Speciale R. Obstructive sleep apnea syndrome (OSAS): Effects on the vestibular system. *Acta Otorhinolaryngol Ital*. 2010;30(6):281-284.
8. Blödow A, Pannasch S, Walther LE. Detection of isolated covert saccades with the video head impulse test in peripheral vestibular disorders. *Auris Nasus Larynx*. 2013;40(4):348-351. [\[CrossRef\]](#)
9. Kapur VK, Auckley DH, Chowdhuri S, et al. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: An American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med*. 2017 March 15;13(3):479-504. [\[CrossRef\]](#)
10. Alhabib SF, Saliba I. Video head impulse test: A review of the literature. *Eur Arch Otorhinolaryngol*. 2017 March;274(3):1215-1222. [\[CrossRef\]](#)
11. Angov G, Mihaylova-Angelova E, Petrova D, Stambolieva K. Vestibular function in panic disorder patients: A vestibular-evoked myogenic potentials and video head impulse test study. *Eur Arch Otorhinolaryngol*. 2019 June;276(6):1607-1616. [\[CrossRef\]](#)
12. Premachandra P. Special investigations used in the assessment of the dizzy patient. *Otorhinolaryngologist*. 2013;6:6-14.
13. Young YH. Potential application of ocular and cervical vestibular-evoked myogenic potentials in Meniere's disease: A review. *Laryngoscope*. 2013;123(2):484-491. [\[CrossRef\]](#)
14. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol*. 1988;45(7):737-739. [\[CrossRef\]](#)
15. Somers VK, Dyken ME, Clary MP, Abboud FM. Sympathetic neural mechanisms in obstructive sleep apnea. *J Clin Invest*. 1995;96(4):1897-1904. [\[CrossRef\]](#)
16. Colrain IM, Campbell KB. The use of evoked potentials in sleep research. *Sleep Med Rev*. 2007;11(4):277-293. [\[CrossRef\]](#)
17. Ni D. Auditory brain-stem response in obstructive sleep apnea syndrome. *Zhonghua Er Bi Yan Hou Ke Za Zhi*. 1991;26(5):284-6, 317, 317.
18. Hildesheimer M, Muchnik H, Rubinstein M. Cochlear hypoxia and the compound action potentials. *Laryngoscope*. 1988;98(5):557-560. [\[CrossRef\]](#)
19. Casale M, Vesperini E, Potena M, et al. Is obstructive sleep apnea syndrome a risk factor for auditory pathway? *Sleep Breath*. 2012;16(2):413-417. [\[CrossRef\]](#)
20. Muchnik C, Rubel Y, Zohar Y, Hildesheimer M. Auditory brainstem response in obstructive sleep apnea patients. *J Basic Clin Physiol Pharmacol*. 1995;6(2):139-148. [\[CrossRef\]](#)
21. Mutlu M, Bayır Ö, Yücege MB, et al. Vestibular evoked myogenic potential responses in obstructive sleep apnea syndrome. *Eur Arch Otorhinolaryngol*. 2015;272(11):3137-3141. [\[CrossRef\]](#)