

Vestibular Assessment of Obstructive Sleep Apnea Patients

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Abstract

Background: Obstructive sleep apnea (OSA) is a common disorder. Over recent years, there appears to be a significant number of the idiopathic dizzy patients who simultaneously suffer from some sort of sleep disturbance, either subjectively or objectively. Only one retrospective review has investigated the potential association between sleep disturbance and symptoms of dizziness, but it was done in a cursory manner.

Aim of Study: The aim of the study was to assess vestibular function in obstructive sleep apnea patients using computerized dynamic posturography, to study the relationship between oxygen saturation and vestibular function in obstructive sleep apnea patients and to study if there is correlation between clinical findings and polysomnography results.

Patients and Methods: A prospective interventional study carried out on a group of 30 patients with OSA confirmed diagnosis, according to the American Academy of Sleep Medicine classification as a study group that were selected from patients (age range 20-60 years) attending the ENT outpatient clinic at Al-Hussein University Hospitals with clinically suspected OSA after being investigated with polysomnography. Control group consisted of 30 normal adult volunteers, age, sex and body weight matched with the study group, and without any history of sleep-disordered breathing, otologic, neurologic or vestibular disorders, and sleep apnea was excluded by polysomnography. All the patients included in this study were subjected to thorough history taking, general examination, complete otorhinolaryngologic examination, overnight polysomnography and vestibular assessment in the form of sensory organization test (SOT) using computerized dynamic posturography (CDP).

Results: The results show that there was a statistically significant positive correlation between RDI and desaturation index (Pearson's $r=0.639$ with p -value <0.001). In addition, results also show that there was a positive correlation between body weight and both RDI & average low O_2 during all desaturations but not statistically significant (Pearson's $r=0.379$ & 0.223 with p -value 0.39 & 0.236 respectively). Moreover, as expected RDI is positively correlated to desaturation index and negatively correlated to the average low O_2 during all desaturations, with statistical significance (p -value <0.001 & <0.01 respectively). Similar findings were found in the control group. No statistically significant correlation between the study and control groups as regards SOT results of CDP.

Conclusion: This study is one of the first studies to evaluate OSA patients with computerized dynamic posturography, and with control group.

No statistically significant correlation between BMI, neck circumference and RDI, and the SOT results of CDP could be found. To rationale this result, central vestibular system can compensate for possible unilateral or single factorial affection. Also, CDP is a functional test as opposed to the pathological nature of caloric testing.

Obesity remains one of the main risk factors for obstructive sleep apnea (OSA), and OSA is strongly associated with obesity. Therefore, it seems clear that induction of OSA by obesity involves a mechanical factor.

Neck circumference is strongly correlated with BMI. Both BMI and neck circumference influence the severity of OSA (RDI).

Increase in neck circumference directly aggravates the severity of OSA, and is consequently reflected on oxygen concentration in during sleep apnea. Similar correlation regarding BMI, but not statistically significant.

Key Words: Vestibular – Obstructive sleep apnea – Posturography.

Introduction

OBSTRUCTIVE sleep apnea (OSA) is a common disorder. Healthcare providers are becoming increasingly aware of the entity and its health impact. Charles Dickens, in his Posthumous Papers of the Pickwick club (1837), gave a vibrantly descriptive picture of Joe, a boy so obese that he has difficulty breathing, sounds as if he is snoring even when awake and frequently falls asleep while standing. Besides the classical Pickwickian syndrome, OSA may afflict both obese and non-obese individuals.

In addition, there is evidence to suggest that some of our most noted leaders and dictators suffered from this problem. The 20th president of the United States, William Howard Taft, had a body mass index of $42\text{kg}/\text{m}^2$ while in office and was reported to snore, to fall asleep during the day frequently and to have hypertension. Chouard and colleagues described several reasons to suspect

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Napoleon I (1769-1821) had OSA in the last decade of his life: He was obese and retrognathic, his neck was short and thick, he had nasal obstruction, he frequently slept during the day, he complained of declining energy and intellect and he was quite tired looking and somewhat disheveled.

OSA is a condition characterized by repetitive upper airway obstruction leading to sleep fragmentation, cardiovascular stimulation and oxygen desaturation during sleep. Together, these lead to symptoms such as snoring, unrefreshing sleep, excessive daytime sleepiness (EDS), and the increased risk of cardiovascular disease, hypertension, cerebrovascular disease and road traffic accidents.

Large cohort studies have demonstrated that OSA is common: Almost 25% of adult men 20-60 years old and 9% of adult women 20-60 years have a respiratory distress index (RDI) >5 events/h.

Attended laboratory-based polysomnography has been and remains a de facto gold standard for diagnosis of sleep-disordered breathing. It allows qualitative and quantitative documentation of abnormalities of sleep and wakefulness, sleep-wake transition, and of physiological function of other organ systems that are influenced by sleep. Many of these, such as sleep apnea, may not be present during wakefulness.

Over recent years, there appears to be a significant number of the idiopathic dizzy patients who simultaneously suffer from some sort of sleep disturbance, either subjectively or objectively. Only one retrospective review has investigated the potential association between sleep disturbance and symptoms of dizziness, but it was done in a cursory manner.

Subjects and Methods

A prospective interventional study carried out at Al-Hussein University Hospitals between Apr. 2014 and Jan. 2018.

Subjects:

Study group (30 patients): Total of 30 patients with OSA confirmed diagnosis, according to the American Academy of Sleep Medicine Classification, were included in the study group. They were selected from patients (age range 20-60 years) attending the ENT outpatient clinic at Al-Hussein University Hospitals with clinically suspected OSA, and confirmed OSA by overnight polysomnography.

Control group: 30 normal adult volunteers, age, sex and body weight matched with the study group, and without any history of sleep-disordered breathing, otologic, neurologic or vestibular disorders, and were investigated by overnight polysomnography to exclude any sleep-breathing disorders.

Exclusion criteria: For both groups, subjects with current or past history of general medical, neurological, musculoskeletal or otologic diseases or findings known to affect hearing or balance, or interfere with test requirements, uncorrected visual problems and postural hypotension were excluded. Those taking medications such as sedatives, hypnotics, anxiolytics, and antidepressants were also excluded.

Methods: All subjects in both groups were submitted for the following protocol:

1- Thorough general history taking: Including personal history, complaints, history of present illness, especially symptoms of habitual snoring, witnessed apneas, nocturnal awakening, gasping or choking episodes during sleep, nocturia, unrefreshing sleep, morning headaches, excessive daytime sleepiness.

2- General examination: Each patient was evaluated clinically by recording of blood pressure, weight and height.

3- Complete otorhinolaryngologic, head and neck examination:

- a- Patients were examined in the Frankfurt plane for craniofacial abnormalities.
- b- Complete neck examination for exclusion of any neck mass or swelling, any scars for previous neck surgery. Thyroid gland examination to exclude any thyroid pathology. Neck circumference was measured at the level of cricothyroid membrane in centimeters.
- c- Nose and nasopharyngeal examination which may reveal septal deviation, hypertrophied turbinate, nasal deformities secondary to trauma or craniofacial anomalies, large adenoid and nasal or nasopharyngeal tumors.
- d- Oropharyngeal and hypopharyngeal examination which include:
 - Tongue examination as regard of its size, length and base position in relation to mouth.
 - Soft palate examination as regard of its length, size and position. The modified Mallampati classification is used to describe the relationship of the palate and to tongue and the rest of the oral cavity.

- Palatine tonsils can be graded according to its size into Grade 0-5.

4- Polysomnography including the following variables: Electroencephalogram (4 channels: C3/A2, C4/A1, O1/A2, and O2/A1); electrooculogram (2 channels: right, left); electromyogram of submental muscles (3 channels); electrocardiogram; and airflow (assessed with an oronasal cannula and a thermistor), chest and abdominal efforts (2 channels) were measured using thoracic and abdominal strain gauges, and arterial oxyhemoglobin saturation (SaO₂: 1 channel) was measured with a finger probe by pulse oximetry.

5- Vestibular assessment in the form of sensory organization test (SOT) using computerized dynamic posturography (CDP) model: Neurocom Smart Equites, International. Inc., Clackamas, Oregon, USA (Fig. 1). The subject standing on a force plate, enclosed by a visual surround, estimating the position of the body sway. Subjects are exposed to six sensory conditions, SOT 1: Eyes open, steady surface and visual surround. SOT 2: Eyes closed, steady surface and visual surround. SOT 3: visual surround rotated, eyes open and platform steady. SOT 4: visual surround steady, platform rotated and eyes open. SOT 5: visual surround steady, platform rotated and eyes closed. SOT 6: platform and visual surround rotated with eyes open. A fall under any of the conditions = a score of 0% and a score of 100% when no sway at all [1].



Fig. (1): Computerized dynamic posturography (CDP) model: Neurocom Smart Equites, International. Inc., Clackamas, Oregon, USA.

6- *Statistical analysis:* Data were statistically described in terms of mean \pm standard deviation (\pm SD), median and range, or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between cases and control groups was done using Student *t*-test for

independent samples for normally distributed data and Mann Whitney U-test for independent samples for not-normal data. For comparing gender, Fisher exact test was used. Correlation between various variables was done using Pearson moment correlation equation for linear relation in normally distributed variables and Spearman rank correlation equation for non-normal variables/non-linear monotonic relation. *p*-values less than 0.05 was considered statistically significant. All statistical calculations were done using computer program IBM SPSS (Statistical Package for the Social Science; IBM corp, USA) release 22 for Microsoft Windows.

Results

60 subjects included in the study, age ranged from 20 to 60 years. Subjects were divided into two equal groups and matched regarding gender distribution (Table 1).

Table (1): Shows gender distribution in the study and control groups.

	Study	Control
Males	21 (70%)	21 (70%)
Females	9 (30%)	9 (30%)

In the present study group, the age mean is 46.3 ± 6.417 , while mean of control group was 42.6 ± 8.315 . There was no statistically significant difference between mean age values in the two groups (*p*-value=0.586) (Fig. 2) (Significant at $p \leq 0.05$).

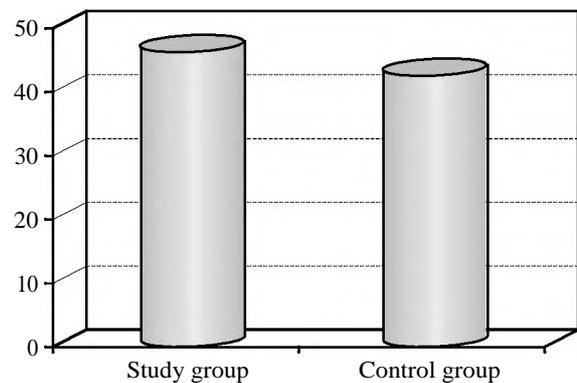


Fig. (2): Mean age in the two groups.

In the present study, the mean body weight for study group was 97.27 ± 9.23 Kg and 97.20 ± 8.36 Kg for the control group. There was no statistically significant difference between mean body weight in the two groups (*p*-value=0.977) (Significant at $p \leq 0.05$). Also the mean neck circumference for

study group was $36.2 \pm 2.89\text{cm}$ and $35.1 \pm 2.41\text{cm}$ for the control group. There was no statistically significant difference between mean neck circumferences in the two groups ($p\text{-value}=0.541$) (Significant at $p \leq 0.05$).

There was a statistically significant difference between the two groups in regards of RDI, desaturation index and average low O_2 during all desaturations, and with $p\text{-value}$ less than 0.001, 0.001 & 0.02 respectively) (Significant at $p \leq 0.05$).

The results show that there was a statistically significant positive correlation between RDI and desaturation index (Pearson's $r=0.639$ with $p\text{-value}$

<0.001). In addition, results also show that there was a positive correlation between body weight and both RDI & average low O_2 during all desaturations but not statistically significant (Pearson's $r=0.379$ & 0.223 with $p\text{-value}$ 0.39 & 0.236 respectively). Moreover, as expected RDI is positively correlated to desaturation index and negatively correlated to the average low O_2 during all desaturations, with statistical significance ($p\text{-value}$ <0.001 & <0.01 respectively). Similar findings were found in the control group (Table 4).

The results show that there was no statistically significant difference between study and control group regarding SOT results of CDP.

Table (2): Shows the polysomnography results for the study and control groups statistically described in terms of mean, number, standard deviation (SD) and median.

	Study group			Control group		
	RDI	Desaturation index	Average low O_2 during all desaturations	RDI	Desaturation index	Average low O_2 during all desaturations
Mean	38.34	45.35	86.55	2.649	7.644	91.09
Number	30	30	30	30	30	30
Standard Deviation	25.75	27.69	9.946	1.652	5.782	3.524
Median	33.8	52.6	91.1	2.9	5	90.5

Table (3): Correlation matrix showing the correlations among body weight, RDI, desaturation index and Average low O_2 during all desaturations in the study group.

	Body weight	RDI	Desaturation index	Average low O_2 during all desaturations
<i>Body weight:</i>				
Pearson's r		0.313	0.379	0.223
$p\text{-value}$		0.925	0.39	0.236
<i>RDI:</i>				
Pearson's r	0.313		0.639	-0.554
$p\text{-value}$	0.925		<0.001	<0.01
<i>Desaturation index:</i>				
Pearson's r	0.379	0.639		-0.346
$p\text{-value}$	0.39	<0.001		0.061
<i>Average low O_2 during all desaturations:</i>				
Pearson's r	0.223	-0.554	-0.346	
$p\text{-value}$	0.236	<0.01	0.061	

Significant at $p \leq 0.05$.

Table (4): Correlation matrix showing the correlations among body weight, RDI, desaturation index & Average low O_2 during all desaturations in the control group.

	Body weight	RDI	Desaturation index	Average low O_2 during all desaturations
<i>Body weight:</i>				
Pearson's r		0.125	-0.120	0.013
$p\text{-value}$		0.510	0.528	0.946
<i>RDI:</i>				
Pearson's r	0.125		0.544	-0.347
$p\text{-value}$	0.510		<0.01	0.060
<i>Desaturation index:</i>				
Pearson's r	-0.120	0.544		-0.481
$p\text{-value}$	0.528	<0.01		0.007
<i>Average low O_2 during all desaturations:</i>				
Pearson's r	0.013	-0.347	-0.481	
$p\text{-value}$	0.946	0.060	0.007	

Significant at $p \leq 0.05$.

Table (5): Shows comparison of numerical variables between SOT results of CDP for the study and control groups using Student *t*-test for independent samples for normally distributed data.

Item	Cases			Controls			df	Mean difference	Std. Error diff	<i>t</i>	<i>p</i> -value
	Mean	SD	n	Mean	SD	n					
SOT composite	81.40	3.971	30	82.10	4.245	30	58	0.700	1.061	0.660	0.510
Condition 1	94.22	2.10	30	94.63	1.640	30	58	0.417	0.486	0.857	0.055
Condition 2	92.05	2.869	30	92.43	2.307	30	58	0.383	0.672	0.570	0.893
Condition 3	92.38	3.240	30	92.22	3.117	30	58	0.162	0.821	0.203	0.771
Condition 4	85.53	5.096	30	85.30	5.715	30	58	0.233	1.398	0.167	0.527
Condition 5	68.95	6.364	30	69.78	7.008	30	58	0.833	1.728	0.482	0.529
Condition 6	68.52	7.390	30	70.47	8.668	30	58	1.950	2.080	0.983	0.169

Significant at $p \leq 0.05$.

Discussion

Obstructive sleep apnea (OSA) is a chronic disorder of the upper respiratory system characterized by repetitive partial or complete of the upper airway during sleep. This results in hypoxemia, increased sympathetic overdrive, increased blood pressure, and increased carbon dioxide levels in the blood; the resulting hypoxia is linked to a wide range of problems stemming from oxidative stress and inflammation and is associated with multiple cardiovascular morbidities.

As OSA worsens in severity, it has been shown to be associated with the development of significant medical co-morbidities, including hypertension, cardiovascular disease, stroke, obesity, and insulin resistance. Furthermore, the presence of OSA has been linked to an increased risk of motor vehicle accidents, impaired daytime performance and quality of life and increased mortality independent of co-morbidities [2].

In the present study, a strong positive correlation was found between BMI and the severity of OSA (Pearson's $r=0.760$ with p -value <0.001) and a statistically significant difference in BMI between the study and control groups (p -value <0.001). A large number of different authors have found similar results. Plywaczewski et al., [3] found similar result in 133 confirmed OAS patients, BMI = 35.8 ± 6.1 kg/m²; RDI = 45.3 ± 23.6 with a p -value <0.0001 . Also, Young et al., [4] after studying a random sample of 602 with PSG found that obesity is a significant risk factor p -value <0.001 .

The role of obesity as a risk factor for OSA was also shown in two other large epidemiological studies, the Sleep Heart Health Study by Newman et al., [5] and the Cleveland Family Study by Tishler et al., [6], with 2968 and 1149 participants respectively. It has subsequently been estimated that 58% of moderate-severe OSA can be attributed to obes-

ity, with an effect greatest in males and females aged 30-49 years by Joosten et al., [7].

To further consolidate these results, Peppard et al., [8] evaluated twice 690 subjects for SDB and found that a 10% weight gain predicted an approximate 32% (95% confidence interval [CI], 20%-45%) increase in the AHI. A 10% weight loss predicted a 26% (95% CI, 18%-34%) decrease in the AHI. Moreover, Quintas-Neves et al., [9] found after conducting a meta-analysis of 22 studies of bariatric surgery for OSA patients, a postoperative reduction of the AHI to $<20/h$ and/or a $>50\%$ postoperative reduction of AHI.

Our results also showed that RDI tends to increase with the increase in neck circumference, with a high statistical significance correlation (Pearson's $r=0.585$ with p -value=0.003). Subsequently this was reflected on the decrease of values of average low arterial oxygen concentration during all desaturations encountered in PSG (Pearson's $r=0.383$ with p -value=0.065).

Most of the other studies that studied similar relation supported this fact. Borges et al., [10] found a statistically significant Pearson's correlation between neck circumference and RDI in a study of 102 OSA patients with p -value <0.01 . Similarly, Santaolalla Montoya et al., [11] further proven this relation statistically with logistic regression analysis of 433 OSA patients with a very high statistical significance (p -value <0.0001), and additionally with multivariate logistic regression (p -value=0.018).

Same correlation was proven by, Pinto et al., [12] in 82 OSA patients with (Pearson's $r=+0.389$ and $p=0.001$), Yucel et al., [13] in 47 OSA patients with (Pearson's $r=+0.411$ and $p<0.05$) and Hoffstein & Mateika [14] in 670 OSA patients with (multiple $R^2=0.27$, $p<0.001$).

In the present study, there was a statistically significant difference between neck circumference values in the two groups (p -value <0.001), with a mean neck circumference for study group was 40.06 ± 2.66 cm and 36.5 ± 3.4 cm for the control group. Similarly, Julia-Serda et al., [15] proven a statistically (p -value <0.001) between OSA patient group ($n=115$) and a non-OSA group ($n=92$) with means of 43.5 ± 3.5 and 40.6 ± 3.9 respectively.

Form previous results, BMI and neck circumference are statistically positively correlated to RDI, and as expected this study showed a statistically significant positive correlation between BMI and neck circumference (Pearson's $r=0.685$ with p -value <0.001).

An explanation of the correlation between BMI and neck circumference and its effect on RDI may be through deposition of fat in the neck, leading to subsequent narrowing of the pharyngeal airway. The total amount of fatty tissue surrounding the upper airways may predispose to OSA when the force of extraluminal adipose becomes greater than the contracting forces of the dilator muscles responsible for maintaining pharyngeal patency [16].

As regards the correlation between neck circumference and average low arterial oxygen concentration during desaturations encountered in PSG we noticed in this study (Pearson's $r=-0.383$ with p -value $=0.065$), was further noticed by Tangerina Rde et al., [17] and Rey de Castro et al., [18] with p -value $=0.004$ and <0.001 respectively.

Over recent years, there appears to be a significant number of the idiopathic dizzy patients who simultaneously suffer from some sort of sleep disturbance, either subjectively or objectively. Furthermore, for patients with sleep disturbances in general, it is the morbidity associated with excessive daytime somnolence that interferes with a patient's daily life in a number of different ways the most [19].

Blakley had reported the potential link between sleep disturbance and dizziness for the first time in the literature in 1999. The primary focus of that retrospective review, however, was the association of dizziness with depression and the subsequent response to treatment with a tricyclic antidepressant. Sleep disturbance was only screened for with a single question as to whether the patient had difficulty initiating and maintaining sleep, and none of the patients were subjectively diagnosed with sleep apnea.

Sowerby et al., [19] tried to further study this relation, where 46 subjects with idiopathic dizziness (ID), 20 with benign paroxysmal positional vertigo (BPPV), and 69 negative controls with hearing loss but no dizziness were enrolled. Participants were screened using three different methods to identify patients at high risk for sleep disorders, the Berlin Questionnaire and the Multivariable Apnea Risk (MAP) Index to assess for sleep-disordered breathing, and the Epworth Sleepiness Scale (ESS) to assess for daytime somnolence in general. Dizziness group was subjected to videonystagmography, then two groups of patients from the dizziness group were selected for evaluation: those with BPPV, to act as a positive dizzy control, and those with ID as the group of interest.

Results showed that the ID were more likely to have significant daytime somnolence than the hearing loss group, with a likelihood ratio (LR) of 7.8 for the ESS 12 score ($p=0.021$) and 7.1 for the ESS 10 score ($p=0.029$). Using the MAP score, a statistically significant difference between the ID group and both the BPPV group (LR 3.99, $p=0.046$) and the hearing loss group (LR 5.46, $p=0.019$) was found. However, this study had the same drawback as Blakley's study, [20] this study consisted only of a questionnaire and physical exam; and no formal sleep studies were included in patient work-up to confirm or refute the findings of a patient being at high risk for sleep disturbance.

Gallina et al., [21] study was to evaluate the effects of OSA on the peripheral and central vestibular system using videonystagmography and caloric test, in 45 OSA patients and 30 volunteers as a control group. None of the individuals in the control group suffered from OSA ($AHI \leq 4$). Four patients (8.8%) in the OSA group complained of one episode of dizziness or imbalance during the last year.

Caloric vestibular tests in the OSA group demonstrated abnormal findings in 27 patients: 20 had bilateral vestibular hyporeflexia and 7 revealed a unilateral vestibular hyporeflexia. The saccadic eye movement study showed an increased latency (>200 msec) in 4 cases. Five patients had a morphological alteration of smooth-pursuit movements. The optokinetic nystagmus was normal in all cases. The control group presented normal instrumental vestibular tests in all cases but one that revealed an asymptomatic unilateral hyporeflexia. Stratification of the data according to the grade of OSA did not show any significant difference between the incidence of bilateral or unilateral hyporeflexia ($p>0.23$).

The results observed show an apparent incongruence between the occurrence of subjective symptoms of vestibular hypofunction such as giddiness or vertigo, that was seen in just four patients (4/35), as opposed to the objective findings of VNG which showed variations in 27/35 patients. A possible explanation by Gallina et al., is that the caloric test, in such patients, could be influenced by a different factor such as mental alertness, drowsiness or lapse in attention due to OSA with consequent suppression of the occurrence of caloric nystagmus, resulting in bilateral hyporeflexia.

Urban et al., [22], found that 15 of 18 patients in their study showed no reproducible electrophysiological brainstem abnormalities during waking. In one patient a left pontine and in two patients a bilateral pontomesencephalic lesion in combination with OSA was observed, although a causal connection was not conclusively confirmed. Thus, their results did not support the assumption of a relevant structural brainstem lesion in OSA patients with normal neurological condition. However, the possibility of functional brainstem changes occurring during sleep or outside the tested regions could not be excluded.

Kayabasi et al., [23], selected a group of 25 mild OSA and another group of 25 moderate-severe OSA patients, and were evaluated videonystagmography and caloric testing. Results also confirmed significant and positive correlation between BMI and RDI. VNG results for moderate-severe OSA group, showed that 14 patients (56%) experienced unilateral canal paresis, four patients (16%) experienced spontaneous nystagmus, and 10 patients (40%) experienced post-head shake nystagmus. One patient (4%) in this group was diagnosed with pursuit abnormality and demonstrated bilateral low gain.

While the mild OSA group, six patients (24%) experienced unilateral canal paresis, one patient (4%) experienced spontaneous nystagmus, and two patients (8%) experienced post-head shake nystagmus. Abnormality in oculomotor tests was not observed in patients belonging to this group. Optokinetic and saccadic tests of all patients did not demonstrate any abnormalities. Nystagmus and canal paresis rates were significantly higher in moderate-severe OSA group compared to mild OSA group. Results of the Romberg test, tandem Romberg test, cerebellar examinations, and positional tests were normal in both groups.

However, this study had a drawback of absence of a control group that consisted of participants without OSA to statistically validate the results.

There is some mention about effect of OSA on the vestibular apparatus in two studies, one conducted by an Italian group and the other by a Chinese group on a similar topic, however only abstracts are available for these studies in the English literature and not the main article. In addition, both studies were focused on the effect of OSA on hearing function [21].

After reviewing all published studies to date, this current study is the only one evaluating OSA patients with computerized dynamic posturography, and with control group. The results showed that there was no statistically significant difference between study and control group regarding SOT results of CDP, (79.67 ± 3.76) and (81.1 ± 3.5) respectively with p -value=0.201.

To rationale this result, we must consider the fact that the recurrent apneic episode, which is so characteristic of OSA, results in a continuous reduction in the hemoglobin oxygen saturation. This causes, especially in the severe cases, a hypoxic condition during sleep. The effects of this chronic hypoxia, during sleep, result in alterations in the normal physiology of several organ systems in the body. A possible explanation is that the caloric test, in such patients, could be influenced by a different factor such as mental alertness, drowsiness or lapse in attention due to OSA with consequent suppression of the occurrence of caloric nystagmus, resulting in bilateral hyporeflexia [21].

A potential link between sleep disturbance and dizziness could possibly explain the difference between the incidence of symptoms and the otoneurological findings. The chronic idiopathic dizziness present in some OSAS patients may be related to sleep deprivation related to sleep apnea [19].

It is tempting to hypothesize that the chronic hypoxic state seen in OSA results in the development of a progressive reduction in vestibular function. However, if the peripheral vestibular system becomes asymmetric due to hypoxic damage, the central vestibular system corrects this disequilibrium between the two sides [21].

Our results can also indicate that the central vestibular system can compensate for possible unilateral or single factorial affection. Especially that CDP is a functional test as opposed to the pathological nature of caloric testing. Because of this, a unilateral peripheral vestibular deficit may be well compensated by the central vestibular system and the equilibrium may be restored.

Conclusion: This study is one of the first studies to evaluate OSA patients with computerized dynamic posturography, and with a control group.

No statistically significant correlation between BMI, neck circumference and RDI, and the SOT results of CDP could be found. To rationale this result, central vestibular system can compensate for possible unilateral or single factorial affection. Also, CDP is a functional test as opposed to the pathological nature of caloric testing.

Obesity remains one of the main risk factors for obstructive sleep apnea (OSA), and OSA is strongly associated with obesity. Therefore, it seems clear that induction of OSA by obesity involves a mechanical factor.

Neck circumference is strongly correlated with BMI. Both BMI and neck circumference influence the severity of OSA (RDI).

Increase in neck circumference directly aggravates the severity of OSA, and is consequently reflected on oxygen concentration in during sleep apnea. Similar correlation regarding BMI, but not statistically significant.

Conflict of interest: None declared.

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تقييم وظائف الاتزان لمرضى توقف التنفس الانسدادي النومي

يتضمن التصنيف الدولي لاضطرابات النوم، ثمانون تشخيصاً مختلفاً، ومن أكثرها شيوعاً تلك المصحوبة بأضطرابات في التنفس أثناء النوم، وتنقسم هذه المجموعة إلى نوعين، تبعاً إذا كانت مصحوبة بانسداد تنفسي أثناء النوم أم لا. ويتصف مرض الانسداد التنفسي أثناء النوم بوجود نوبات متكررة من إنسداد التنفس بالممرات الهوائية العليا أثناء النوم والذي يكون في الغالب مصحوباً بالشخير، وبناءً على ذلك فإن تلك الاضطرابات التي تحدث تودي إلى عدم النوم بصورة صحية وكافية مما ينتج عن ذلك شعور المريض بأعراض نقص النوم مثل الميل إلى النوم أثناء ساعات العمل وعدم القدرة على التركيز وحوادث الطرق، بالإضافة إلى الأعراض الجانبية الناتجة عن إنسداد التنفس أثناء النوم والمصحوب بالضرورة بنقص كمية الأكسجين في الدم والذي يؤدي إلى زيادة خطر الإصابة بأمراض القلب والأوعية الدموية وارتفاع ضغط الدم والأمراض الدماغية الوعائية.

كان ولا يزال المعيار الذهبي لتشخيص اضطرابات التنفس أثناء النوم هو اختيار معمل النوم، حيث يوفر الأختبار المعلومات الكافية عن التغيرات الفسيولوجية التي تحدث في مختلف أعضاء جسم الأنسان وعلاقتها بمراحل النوم واليقظة المتعددة، كما إنه يسمح بتوثيق اضطرابات النوم نوعاً وعدداً، والانتقال من مرحلة اليقظة إلى مرحلة النوم العكس، وتأثير مراحل النوم المختلفة على الوظائف الفسيولوجية لأعضاء الجسم المختلفة.

على مدى السنوات الأخيرة، تم تسجيل أصابة أعداد كبيرة من مرضى اضطرابات النوم بالدوار مجهول السبب، إما بشكل موضوعي أو غير موضوعي، تم إجراء الدراسة الحالية على مجموعتين، تضمنت المجموعة الأولى (الدراسة) ٣٠ مريضاً ممن يعانون من الأنسداد التنفسي أثناء النوم بعد تأكيد التشخيص بواسطة أختبار معمل لنوم ووفقاً لمعايير تصنيف أكاديمية الأمريكية لطب النوم، وتم أختبار هذه المجموعة من المرضى من المترددين على عيادة الأنف والحنجرة الخارجية في مستشفى الحسين الجامعي، ضمن الفئة العمرية من إلى عاماً، وبأعراض الأنسداد التنفسي النومي، أما المجموعة الثانية (المرجعية) فتتألف من ٣٠ متطوعاً من نفس الفئة العمرية ومتوازنة نوعياً مع مجموعة الدراسة، مع عدم وجود أي تاريخ مرضي لأضطرابات النوم أو أمراض الأذن أو الجهاز الدهليزي أو الجهاز العصبي وبعد الفحص بمعمل النوم.

وكان الهدف من هذه الدراسة هو تقييم وظيفة الجهاز الدهليزي في مرضى أنقطاع التنفس الأنسدادي النومي، بإستخدام الجهاز الحركي لفحص التوازن بالكمبيوتر، ودراسة العلاقة بين تشبع الدم بالأكسجين والوظيفة الدهليزية لمرضى أنقطاع التنفس الأنسدادي النومي، ودراسة ما إذا كان هناك ارتباط بين الخصائص الإكلينيكية ونتائج أختبار معمل النوم.

المرضى الذين شملتهم الدراسة خضعوا لفحص شامل للتاريخ المرضي، والفحص الإكلينيكي العام وفحص الأذن والأنف والحنجرة، وتقييم لوظيفة الجهاز الدهليزي بإستخدام الجهاز الحركي لفحص التوازن بالكمبيوتر.

تبقى السمعة واحدة من أهم المسببات الرئيسية لمرض أنقطاع التنفس الأنسدادي النومي، حيث تؤثر زيادة الوزن على شدة المرض (مؤشر اضطراب التنفس)، والذي ينعكس بالتالي على تشبع الدم بالأكسجين في أثناء أنقطاع التنفس النومي، ولم يتم التوصل إلى علاقة ذات دلالة إحصائية بين وزن الجسم أو مؤشرات إضطراب التنفس، وبين نتائج أختبارات الجهاز الحركي لفحص التوازن بالكمبيوتر.