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VESTIBOLOGY

Obstructive sleep apnoea syndrome (OSAS): effects on the vestibular system

Obstructive sleep apnoea syndrome (OSAS): effetti sul sistema vestibolare

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SUMMARY

Aim of the present study was to evaluate the effects of obstructive sleep apnoea syndrome (OSAS) on the peripheral and central vestibular system, by means of a case series prospective study at the University referral centre of Otolaryngology Head and Neck Surgery; 45 consecutive patients suffering from OSAS were compared with a control group of 30 volunteer subjects selected from among the department employees. Severity of the disease was evaluated by means of cardio-respiratory function monitoring during sleep; the apnoea-hypopnoea index was calculated. Both groups underwent: 1) head and neck examination; 2) fibre-optic examination; 3) pure tone audiometry; 4) evaluation of eye movement disorders using oculomotility tests recorded with the help of video-nystagmography; 5) caloric vestibular responses recorded with video-nystagmography; 6) auditory brainstem response. Results, when evaluating our data, showed that the peripheral vestibular system may become asymmetric due to hypoxic damage while the central vestibular system corrects this disequilibrium.

KEY WORDS: OSAS • Dizziness • Vertigo • Imbalance • Disequilibrium

RIASSUNTO

Obiettivo del presente studio è la valutazione degli effetti dell'OSAS sul sistema vestibolare periferico e centrale. A questo scopo è stato realizzato uno studio prospettico presso un centro di riferimento Universitario di Otorinolaringoiatria e Chirurgia della Testa e del Collo, basato sullo studio di un gruppo di 45 pazienti consecutive affetti da OSAS confrontati con un gruppo di controllo di 30 soggetti normali. La severità della malattia è stata valutata con monitoraggio cardio-respiratorio nel sonno con calcolo dell'indice apnea-ipopnea. Entrambi i gruppi sono stati esaminati con: visita completa ORL, esame endoscopico, audiometria, videonistagmografia, prove caloriche, ABR. I risultati dello studio hanno evidenziato che il sistema vestibolare periferico può diventare asimmetrico o iporeflessico a causa del danno ipossico, mentre il sistema vestibolare centrale ripristina il corretto equilibrio.

PAROLE CHIAVE: *OSAS • Vertigine • Disequilibrio*

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Introduction

Several Authors in the last decade [1-3] have focused attention on research into brainstem neurophysiology in respiratory tract regulation and related disturbances during sleep. They used auditory brainstem response (ABR) as a means of measuring the effect of brainstem on the pathophysiology of obstructive sleep apnoea syndrome (OSAS). Various branches of medicine such as Neurology, Pulmonology, Otorhinolaryngology, and Cardiology have made efforts to find diagnostic procedures and treatment for OSAS. The role of the ear-nose-throat (ENT) specialist became fundamental in the diagnostic pathway, morphological observations and classification of the anatomical anomalies of the upper respiratory tract, which are often associated with OSAS. The ENT surgeon also plays an important role in the surgical correction of such problems.

The cardiovascular problems and neurological disturbances due to hypoxia of the OSAS are well known. But, the neurotological consequences are not so clearly identified and, therefore, are subject of debate. There are no convincing studies yet, in the English literature, regarding this topic. We could only find some publications on the effect of OSAS on hearing function. Even these do not show any uniform results [1 3].

The aim of this investigation is to verify the functional neurotological alterations in OSAS patients focusing in particular on the peripheral and central vestibular system. Based on the evidence that the vestibular nuclei are more sensitive to hypoxia than other cerebral nuclei, as demonstrated in animal experiments [4], our hypothesis is that the functional alteration of the vestibular nuclei may be an index of abnormal activity of the respiratory nuclei considering the anatomical contiguity of this centre. The

hypoxic damage could also be the cause of a deficit of the peripheral vestibular system.

Material and methods

This prospective study was performed on 45 consecutive patients suffering from OSAS in the period 2004-2005. They were compared with a control group of 30 volunteers selected from among the department employees. Case history and presenting symptoms were collected. The severity of the disease was evaluated by monitoring cardio-respiratory function during sleep in both the groups. The apnoea-hypopnoea index (AHI) (event per hour of sleep) was calculated according to the American Academy recommendations [5].

All of the patients in the OSAS group and subjects in the control group were evaluated as follows: 1) A detailed head and neck examination; 2) fibre-optic nasal, pharyngeal and laryngeal examination; 3) pure tone audiometry (PTA) (recording the average of 0.5, 1, 2, 4 kHz); 4) evaluation of eye movement disorders using oculomotility tests recorded by videonystagmography (VNG); 5) caloric vestibular responses with cold water stimulation and recorded by VNG; 6) auditory brainstem response (ABR).

The oculomotility test evaluation included: a) voluntary saccadic eye movements: latency, maximal velocity, accuracy; b) smooth-pursuit movements: latency, gain, morphology; c) optokinetic nystagmus: slow-phase angular velocity, gain, symmetry; d) nystagmus during caloric vestibular tests: duration, frequency, slow-phase angular velocity with the classic Jongkees' formula [6].

Patients with previous diagnosis of neurotological disease (i.e., labyrinthine diseases, benign paroxysmal positional vertigo, etc.), cardiovascular diseases, pulmonary disorders or metabolic alterations were excluded from the study.

Informed consent was obtained, in each case, and our Institutional Review Board approved the present study.

Results

The OSAS group included 31 males and 14 females. Mean age of the patients was 43.3 years (range 24-56 years). Mean age of the Control group was 41 years (range 27-54 years) and the male/female ratio was 2:1. No differences in obesity grade, cardiovascular disease, diabetes or other cardiovascular diseases were observed between the two groups.

Among the 45 OSAS patients evaluated, 29 patients suffered from low to moderate grade OSAS (AHI 10-30) and 16 had severe grade OSAS (AHI > 30) (Table I). None of the individuals in the control group suffered from OSAS (AHI ≤ 4).

Four patients (8.8%) in the OSAS group complained of one episode of dizziness or imbalance during the last year and were excluded from the evaluation. No patients in either group complained of any hearing loss.

Table I. Apnoea Hypopnoea Index (AHI) of the two groups.

AHI	OSAS Group	Control Group
0-5	0	30
10-30	29	–
> 30	16	–

All patients in the OSAS group were classified as moderate to severe disease. In the control group, no patients presented sleep snoring.

Audiological assessment of the OSAS patients revealed: normal hearing in 36 patients and high frequencies of bilateral sensorineural hearing loss in 5 patients with a down-sloping audiometric curve (mean PTA for 4 and 8 KHz of 45 dB).

Auditory brainstem response examination showed an increased I-V wave interval in 36.5% of OSAS cases and in 4 subjects in the control group. All subjects with increased latency had normal hearing.

In the control group, we discovered 2 patients with a bilateral sensorineural hearing loss at 4 KHz and 8 kHz frequencies, with mean PTA of 35 dB, but the ABR in these 2 individuals was normal.

Caloric vestibular tests in the OSAS group demonstrated abnormal findings in 27 patients and normal vestibular functions in the remaining 8 patients.

Of the 27 patients with an abnormal test result, 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 OSAS did not show any significant difference between the incidence of bilateral or unilateral hyporeflexia ($p > 0.23$). All data are shown in Tables II, III.

Discussion

The recurrent apnoic episode, which is so characteristic of OSAS, results in a continuous reduction in the haemoglobin 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.

Regardless of the cause of the OSAS, the central causative pathology lies in the upper respiratory tract. Although there has been much research in the field of ENT in treating this condition, it is surprising to find that very little research has been performed looking into the effect that OSAS may have on the neighbouring anatomical structures that are regularly dealt with on a day-to-day basis.

Table II. Clinical and instrumental findings in OSAS group.

Clinical and Instrumental data		Patients	Controls
Symptoms	Dizziness	4*	0
	Subjective Hearing Loss	0	0
Sensorineural Hearing Loss at Pure Tone Audiometry		5 (12.2%)	2 (6.6%)
ABR (increased I-V wave interval)		15 (36.5%)	4 (13%)
Caloric Test	Hyporeflexia Bilateral	20 (48.7%)	0
	Hyporeflexia Unilateral	7 (17%)	1 (3.3%)
SEM (latency > 200 msec.)		4 (9.7%)	0
SPM (morphology alterations)		5 (12.2%)	0

*SEM: saccadic eye movements; SPM: smooth-pursuit movements; * patients excluded from final analysis.*

Table III. Stratification of Vestibular tests with Apnoea Hypopnoea Index (AHI) score.

	Bilateral hyporeflexia	Unilateral hyporeflexia	Morphological alteration of smooth pursuit
AHI 10-30 (29 cases)	11 (37.9%)	4 (13.7%)	2 (6.8%)
AHI > 30 (16 cases)	9 (56.2%)	3 (18.7%)	3 (18.7%)

The percentage was calculated for each subgroup of AHI score. The χ^2 test did not show a significant difference between grade of OSAS and incidence of hyporeflexia ($p > 0.23$).

As already mentioned, there are no studies in the English literature mentioning the effect of OSAS on the vestibular apparatus. Urban et al. [1] did not find vestibular alterations in 83% of the series reported.

However, there is some mention in two studies, one conducted by an Italian group and the other by a Chinese group on a similar topic, although both were focused on the effect of OSAS on hearing function. Only abstracts are available for these studies in the English literature [7,8].

We conducted the present study in order to find out whether there were any possible effects on the vestibular labyrinth secondary to OSAS.

The functional alteration of the vestibular nuclei may be an indication of abnormal activity of the respiratory nuclei considering the anatomical contiguity of this centre. The vestibular nuclei damage could be revealed by an alteration of the slow and rapid eye movements.

The quantitative and qualitative evaluation of peripheral and central vestibular function, by computed VNG, allowed us to acquire precise data about the alterations found in OSAS. The susceptibility of the posterior labyrinth to a hypoxic state was proven by the high percentage of vestibular reflex alterations observed during caloric stimulation. The test results were found to be worse in patients with a severe grade of OSAS.

Sleep deprivation due to OSAS might affect postural stability on account of a reduced adaptation ability and lapses in attention [9]. The right temporo-parietal cortex activity, an area also involved in VOR control, may be altered by sleep deprivation [10]. A slight compromise of the central vestibular system is demonstrated by the low percentage of oculomotility test alterations in our series. These structures, located in the brainstem and cerebellum, are thought to regulate the slow eye movements and ocular reflex.

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 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 OSAS with consequent suppression of the occurrence of caloric nystagmus, resulting in bilateral hyporeflexia.

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 apnoea [11].

However, it is tempting to hypothesize that the chronic hypoxic state seen in OSAS results in the development of a progressive reduction in vestibular function. If the peripheral vestibular system becomes asymmetric due to hypoxic damage, the central vestibular system corrects this disequilibrium between the two sides. Our results can also indicate that the central vestibular system is more resistant to the hypoxic state. Because of this, a unilateral peripheral vestibular deficit may be well compensated by the central vestibular system and the equilibrium may be restored.

Conclusions

Evaluation of vestibular function, in OSAS patients, shows the effects of the sleep apnoea and its associated hypoxia on the peripheral, principally, and central vestibular systems. The peripheral vestibular system may become asymmetric or hypo-reflexic due to hypoxic damage while the central vestibular system corrects this disequilibrium. These findings may help to better identify some of the vertiginous symptoms which OSAS patients complain of and which are classified as idiopathic dizziness [11].

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