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RESEARCH ARTICLE

Study of the Effects of Sleep Apnea Syndrome on the Paraclinical Parameters of Balance Exploration

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ABSTRACT

Aim: To evaluate the impact of Obstructive Sleep Apnea Hypopnea Syndrome (OSAHS) on posturographic parameters.

Objective: The primary objective was to evaluate whether the mean gain of the Visually Vestibulo-Ocular Reflex (VVOR), the parameter most likely to be modified in case of central damage, decreased in patients diagnosed with OSAHS. The secondary objective was to verify if the other parameters of videonystagmography and posturography varied according to the presence or not of OSAHS.

Materials and Methods: Retrospective analysis of posturographic parameters of patients previously tested by polygraphy (whether or not they had OSAHS).

Results: In the 66 patients included, the mean VVOR gains did not differ significantly between the "no OSAHS" vs. "OSAHS" groups (respectively 0.88 vs. 0.71, $p = 0.1224$).

None of the other parameters measured, such as the mean caloric deficits, the mean weights of vestibular, visual and somesthetic afferents measured by posturography, the mean gains of the vestibulo-ocular reflex, and the frequency of falls on the posturograph, had a statistically different distribution according to the presence or absence of OSAHS.

Conclusion: We did not show any significant difference in posturographic parameters according to the presence or absence of OSAHS, in particular the absence of vestibular and/or central involvement.

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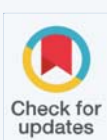
INTRODUCTION

Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS) is characterized by upper airway obstruction during sleep [1]. Clinically, it can be suspected in front of diurnal and nocturnal clinical signs (headaches, fatigue, drowsiness, nocturia, asphyxic awakening) [2]. It must be confirmed by a polygraphy or a polysomnography which finds an Apnea-Hypopnea Index (AHI) >5/h [2]. OSAHS is said to be mild if the AHI is between 5 and 15/h, moderate between 15 and 30/h and severe beyond 30/h. In its severe form, its medium and long term consequences are of several kinds: metabolic, cerebral, behavioral, accidental [2]. Among these risks, the vascular risk is one of the greatest: cardiological (increased risk of heart attack, heart failure, rhythm disorders, hypertension, etc.), neurological (increased risk of stroke, cognitive and memory disorders) and ophthalmological (anterior ischemic optic neuropathy, occlusion of the central artery or vein of the retina) [3]. Because of this central impact (by desaturations and orthosympathetic hyperactivity), it can also cause other symptoms such as vertigo and instability [4-10]. However, this causal link between instability - vertigo and OSA is not clearly established. Indeed, by definition, at least 9 to 17% of the vertiginous and/or unstable patients would suffer from an OSA (as much as the general population [1]). Other possible

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confounding factors such as altered circadian rhythm [11-13], a more obese population [7] or the presence of other chronic diseases have been discussed [14-17].

For the last twenty years, the examination of unstable patients has been made easier by the use of posturography, a series of tests allowing the objective evaluation of vestibular, somesthetic and visual afferents. This instrumental examination of a very subjective symptom often makes it possible to make a diagnosis and to guide the global rehabilitation of balance by physical therapy. Among the different paraclinical explorations of VideoNystagmography (VNG) and posturography, the gains of the vestibulo-ocular reflex after visual fixation (Visually Enhanced Vestibulo-Ocular Reflex VVOR), one of the damped rotatory pendular tests of the VNG, make it possible to search for a mixed central and vestibular impairment.

The primary objective of this study was to determine whether there was an association between vestibular and/or central balance impairment and the presence of OSAHS. The secondary objective was to verify that vestibular, visual, and/or somesthetic afferents decreased in the presence of OSAHS and that the frequency of falls increased in the presence of OSAHS.

METHODS

All patients who underwent both polygraphy and VNG - posturography between 01/01/2011 and 01/01/2020, were retrospectively included thanks to cross-tabulations from the diaries of the Cristal-link® patient software.

Polygraphy was acquired on a MiniScreen plus® polygraph (Löwenstein, Germany). The interpretation of the polygraphy was done according to the criteria of the American Association of Sleep Medicine (available on <https://learn.aasm.org/Public/Catalog/Details.aspx?id=FqkJmktft02E8UCYx20Q3w%3d%3d&returnurl=%2fUsers%2fUserOnlineCourse.aspx%3fLearningActivityID%3dFqkJmktft02E8UCYx20Q3w%253d%253d>) in force at the time of the interpretation. The diagnosis of OSA was made if the apnea-hypopnea index was greater than 5/h.

Dynamic posturography was performed with a Multitest (Framiral®, Grasse, France). Movement directions, areas and velocities were recorded in all the following situations: open and closed eyes, stable and unstable platform, optokinetic and non-optokinetic. Overall stability results were analyzed (see below).

The contraindications of the optokinetic test were:

- Epilepsy.
- Recent ocular surgery (less than 1 month).
- An uncompensated unilateral peripheral vestibular syndrome with directional preponderance of the vestibulo-ocular reflex > 2°/s contralaterally.

- A significant visual alteration (strabismus, exophoria).
- Intolerance to the test.

In this case, the optokinetic test was not performed and only the stability test with eyes closed was performed.

The primary objective of our study was to verify that there was an association between the presence of OSA and vestibular and/or central balance impairment.

The secondary objective was to verify that the weight of vestibular, visual and/or somesthetic afferents decreased in case of OSA and that the frequency of falls and visual dependence increased in case of OSA.

The primary endpoint was the mean VVOR gain according to OSAS status.

Secondary end points were, according to SAHOS status:

- Caloric deficit.
- The mean Vestibulo-Ocular Reflex (VOR) gain.
- The average stability according to platform (eyes open/closed, stable/unstable, optokinetic/no optokinetic).
- The weight of vestibular afferents.
- The rate of fall during posturography.
- The rate of visual dependence.

A difference in VVOR gain of 20% with a risk α at 0.05 and a risk β at 0.2 leads to the recruitment calculation of 393 patients (library pwr of R software).

The values presented correspond to the numbers (proportions) for categorical variables and to the means (\pm standard deviation) for quantitative variables. Univariate analyses were performed with the Student test and the Chi-2 test.

A significance level $\alpha < 0.05$ was used for the primary criterion. A Bonferroni correction was applied to the eight categories of secondary criteria (see above). A significance level of $\alpha < 0.05/8$ or 0.00625 was used.

All tests were performed with R software (v. 3.5.3, www.r-project.org).

The study was declared to the ethics committee of our institution (#20210420_4).

RESULTS

Sixty-six patients met the inclusion requirements

The population included 31 men (47%) and 35 women (53%). The mean age was 60.5 ± 12 years. Thirty-seven patients had OSA (56%) and 29 did not (44%). The mean time spent between the performance of VNG posturography and polygraphy, was 0.2 ± 2.5 years, and highly variable. Other

characteristics of the population with respect to OSAHS and VNG posturography are described in table 1.

Table 2 shows the results of VNG and posturography parameters and their comparisons between the "no OSAHS" vs. "OSAHS" groups.

The mean VVOR gains did not differ significantly between the "no OSAHS" vs. "OSAHS" groups (0.88 vs. 0.71, respectively, $p = 0.122$). In each group, the mean VVOR was normal (> 0.7).

The mean vestibular afference was not significantly different between the "no OSAHS" vs. "OSAHS" group (respectively 50.6% vs. 45%, $p = 0.651$). None of the other measured parameters were significantly different between the "no OSAHS" vs. "OSAHS" groups, using the Bonferroni correction thresholds of significance ($p < 0.00625$).

Table 1: Characteristics of the population.

Sleep exam parameters	
OSAHS n(%) :	37 (56.0)
Severe OSAHS n(%)	10 (15.2)
Moderate OSAHS n(%)	9 (13.6)
Mild OSAHS n(%)	18 (27.3)
No OSAHS n(%)	29 (44.0)
Mean Apnea Hypopnea index \pm SD (/h)	13.1 \pm 16.1
Mean Apnea index \pm SD (/h)	7.7 \pm 14.3
Mean Hypopnea index \pm SD (/h)	5.5 \pm 5.4
Mean percutaneous oxygen saturation \pm SD (%)	93.6 \pm 1.8
Mean Time percutaneous oxygen saturation $< 90\% \pm$ SD (min)	25.4 \pm 46.8
Posturographic parameters	
Mean caloric deficit \pm SD (%)	24.4 \pm 20.5
Patients with caloric deficit (%)*	45
Mean Vestibulo-Ocular Reflex \pm SD	0.52 \pm 0.26
Mean Visual enhanced Vestibulo-Ocular Reflex \pm SD	0.80 \pm 0.28
Mean stability EO/SP \pm SD (%)	92.1 \pm 3.5
Mean stability EO/UP \pm SD (%)	82.6 \pm 8.5
Mean stability EC/SP \pm SD (%)	89.1 \pm 6.1
Mean stability EC/UP \pm SD (%)	67.6 \pm 14.0
Mean stability OK/SP \pm SD (%)	90.6 \pm 4.6
Mean stability OK/UP \pm SD (%)	72.7 \pm 13.2
Falling during posturography (%)	40.9
Mean weight of vestibular afferents \pm SD (%)	47.6 \pm 46.1
Mean visual dependency \pm SD (%)	8.3 \pm 23.7
SD: Standard Deviation; OSAHS: Obstructive Sleep Apnea-Hypopnea Syndrome	
EO/SP: Eyes Open / Stable platform: Somesthetic, visual and vestibular afferences	
EO/UP: Eyes Open / Unstable platform: Visual and vestibular afferences	
EC/SP: Eyes Closed / Stable platform: Somesthetic and vestibular afferences	
EC/UP: Eyes Closed / Unstable platform: Vestibular afferences	
OK/SP: Optokinetic / Stable platform: Somesthetic and vestibular afferences	
OK/UP: Optokinetic / Unstable platform: Vestibular effects	
*Caloric deficit carried if inter-ear difference was $\geq 20\%$.	

Table 2: Mean by sleep status and comparisons.

	No OSAHS (n = 29)	OSAHS (n = 37)	p
Mean caloric deficit (%)	25.9	23.2	0.612
Patients with caloric deficit (%)*	51.9	39.4	0.481
Mean Vestibulo-Ocular Reflex	0.57	0.45	0.215
Mean Visual enhanced Vestibulo-Ocular Reflex	0.88	0.71	0.122
Mean stability EO/SP (%)	92.3	92.0	0.796
Mean stability EO/UP (%)	81.8	83.3	0.526
Mean stability EC/SP (%)	91.0	87.6	0.027
Mean stability EC/UP (%)	70.2	65.3	0.313
Mean stability OK/SP (%)	91.0	90.2	0.506
Mean stability OK/UP (%)	73.5	71.7	0.711
Falling during posturography (%)	59.3	50	0.636
Mean weight of vestibular afferents (%)	50.6	45.0	0.651
Mean visual dependency (%)	7.7	8.8	0.857

OSAHS: Obstructive Sleep Apnea-Hypopnea Syndrome
EO/SP: Eyes Open / Stable platform: Somesthetic, visual and vestibular afferences
EO/UP: Eyes Open / Unstable platform: Visual and vestibular afferences
EC/SP: Eyes Closed / Stable platform: Somesthetic and vestibular afferences
EC/UP: Eyes Closed / Unstable platform: Vestibular afferences
OK/SP: Optokinetic / Stable platform: Somesthetic and vestibular afferences
OK/UP: Optokinetic / Unstable platform: Vestibular effects
*Caloric deficit carried if inter-ear difference was $\geq 20\%$.

DISCUSSION

We showed that the presence of OSAHS did not significantly modify the results of the VNG-posturography tests.

Comparison with other scientific data was difficult because of the lack of available literature. First of all, we did not find any study that included the VVOR as a primary endpoint even though it is the vestibulometric parameter that, physiologically, should be the most modified by a mixed vestibular and central impairment as it is supposed to be in case of OSAHS. Two publications showed a link between posturographic impairment and OSAHS. Degache, et al. [5], in 158 patients (41% without OSAHS and 59% with OSAHS) showed a significant increase in stability areas and velocities with correlations between stability and nocturnal mean percutaneous saturation, AHI, desaturation index, time spent with percutaneous saturation $< 90\%$. Sowerby, et al. [10], on the other hand, looked for OSAHS in idiopathically unstable patients with benign paroxysmal positional vertigo. They showed that OSAHS was statistically more frequent in patients with chronic instability. However, other authors did not find such results. Alessandrini, et al. [4], after treatment of OSAHS with continuous positive airway pressure in 32 patients with severe OSAHS, showed a significant improvement of the instability questionnaires without finding a significant improvement of the posturography parameters. Micarelli, et al. [8] compared the posturography results of 32 healthy volunteers with 32

patients with moderate to severe OSAHS. The only significant difference was an increase in VOR gain. Nakayama, et al. [9] showed no difference in calorimetric tests before and after treatment with continuous positive airway pressure in 20 patients with severe OSA and Menière's disease. The rate of patients with vestibular hyporeflexia in our patients (45%) was consistent with the literature where 44.4 to 65% of patients had unilateral or bilateral vestibular hyporeflexia [6,7,9]. It is important to note that no α -risk inflation control procedure was performed for all of these studies. The retained significances are probably not a reflection of a real difference between the "OSAHS" and "no OSAHS" groups.

Another possible explanation for the difficulties in comparing with other cohorts would be the major demographic differences in our study vis-à-vis other studies:

- Sex ratio (53% women in our study). Men were more numerous in the other studies [5-9,12,13] except for a study [10].
- The mean age of our population, which was comparable to that of Nakayama et al. (60 years-old) [9] but younger than from Gallina, et al. [6] and Kayabasi, et al. [7] (43.5 and 45 years respectively). An age difference could explain a decrease in VOR gain [8]. In our study, it could perhaps explain in part the increase in vestibular dysfunction in the "no OSAHS or mild OSAHS" group, which has already been suggested by other teams [6,7].

Other authors have shown alterations of results on dynamic posturography platform on patients suffering from sleep deprivation [18,19]. They first showed that sleep deprivation affects the nervous system by altering neural connections at multiple levels [20]. A disruption of the VVOR would cause a disruption of the three compensatory pathways of the oculomotor reflexes (ocular pursuit, optokinetic nystagmus and VOR) [21]. However the situation of sleep deprivation is very different from the OSAHS whose evolution is old with repetition of nocturnal hypoxias, and orthosympathetic discharges causing rather chronic central attacks [4-10]. Sleep deprivation is an acute phenomenon, without associated desaturations.

If the size of our cohort is one of the largest ever published (only that of Degache, et al. [5] surpasses it), the lack of power of our study could be criticized. However, it is obvious, given the number of patients to be included as calculated, that it is impossible for a single center to recruit all the necessary patients. A complementary prospective multicenter study would be necessary to complete these descriptive results.

CONCLUSION

With a patient population comparable to or larger than those of other equivalent studies and taking into account

the risk of α -risk inflation by Bonferroni correction, we did not show a significant difference in VNG and posturography parameters according to OSAHS status, especially the absence of vestibular and/or central involvement. A complementary prospective multicenter study would be necessary to complete these descriptive results.

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