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ORIGINAL ARTICLE

The impact of sleep duration in obstructive sleep apnea patients

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Abstract
Purpose Obstructive sleep apnea (OSA) is a risk factor for cardiovascular disease. Strong associations have been reported among sleep duration, hypertension, obesity, and cardiovascular mortality. The authors hypothesize that sleep duration may play a role in OSA severity. The aim of this study is to analyze sleep duration in OSA patients.
Methods Patients who underwent overnight polysomnography were consecutively selected from the Sleep Clinic of Universidade Federal de São Paulo database between March 2009 and December 2010. All subjects were asked to come to the Sleep Clinic at 8:00 a.m. for a clinical evaluation and actigraphy. Anthropometric parameters such as weight, height, hip circumference, abdominal circumference, and neck circumference were also measured.
Results One hundred thirty-three patients were divided into four groups based on total sleep time, sleep efficiency, sleep latency, and wake after sleep onset: very short sleepers (n=11), short sleepers (n=21), intermediate sleepers (n=56), and sufficient sleepers (n=45). Apnea–hypopnea index (AHI) was higher in very short sleepers (50.18±30.86 events/h) compared with intermediate sleepers (20.36±14.68 events/h; p=0.007) and sufficient sleepers (23.21±20.45 events/h; p=0.02). Minimal and mean arterial oxygen saturation and time spent below 90 % oxygen saturation exhibited worse values in very short sleepers. After adjustment for gender, age, AHI, and body mass index, mean oxygen saturation was significantly associated to total sleep time (p=0.01).
Conclusions In conclusion, the present study suggests that sleep duration may be associated to low mean oxygen saturation in OSA patients.

Keywords Obstructive sleep apnea · Sleep duration · Cardiovascular diseases · Polysomnography · Actigraphy

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Introduction

Obstructive sleep apnea (OSA) is characterized by repetitive episodes of partial or complete airway obstruction that result in fragmented sleep, hypersomnolence, and often hypoxemia and hypercapnia [1]. Hypoxia and sleep fragmentation trigger a variety of pathophysiologic mechanisms, such as sympathetic activation [2], endothelial dysfunction [3], and inflammation [4], that could lead to arterial hypertension [5], dyslipidemia [6], obesity [7], and increased cardiovascular mortality [8], among other consequences.

Recently, short sleep duration has also been associated with an increased prevalence of arterial hypertension [9], obesity [10], decreased high-density lipoprotein, increased low-density lipoprotein [11], and cardiovascular mortality [12], suggesting that both OSA and short sleep duration share similar consequences. These authors hypothesize that sleep duration is associated with OSA severity. The aim of this study is to investigate the sleep duration of OSA patients and its correlating significance.

Methods

Study population

Patients who underwent overnight polysomnography (PSG) were consecutively selected from the Sleep Clinic of Universidade Federal de São Paulo (UNIFESP) database between March 2009 and December 2010. Subjects were included if they were over 30 years of age, reported no recent hospitalization or change in medication, and presented an apnea–hypopnea index (AHI) of ≥5 events/h in PSG. Exclusion criteria included a body mass index (BMI) of ≥40 kg/m^2, chronic obstructive pulmonary disease, severe systemic disease, pregnancy, and shift work. One hundred fifty subjects were selected. Seventeen patients were excluded (thirteen with actigraphic technical problems, two with BMI of ≥40 kg/m^2, one shift worker, and one pregnant woman). A total of 133 patients (AHI of ≥5 events/h) were studied.

All subjects were asked to come to the Sleep Clinic at 8:00 a.m. for a clinical evaluation and actigraphy. Anthropometric parameters such as weight, height, hip circumference, abdominal circumference, and neck circumference were also measured. The study was approved by UNIFESP's Ethics Committee, and all subjects signed an informed consent form. All patients were referred to CPAP treatment after completing the protocol.

Polysomnography

PSG was performed using an EMBLA digital system® (17 channels, EMBLA Medicare Medical Devices, Broomfield, CO). The following variables were monitored: electroencephalogram (four channels: C3-A2, C4-A1, O1-A2, and O2-A1), electrooculogram (two channels: LOC-A2 and ROC-A1), electromyogram (two channels: submental and anterior tibialis muscles), ECG (one channel), snoring, and body position. Airflow was monitored using a thermocouple and pressure transducer. Chest and abdominal piezoelectric sensors monitored respiratory effort. Arterial oxygen saturation (SaO_2) and pulse were recorded with a pulse oximeter (model 9500, Nonin®, Nonin Medical, Plymouth, MN). All polysomnograms were performed and scored by an experienced sleep technician following guidelines for sleep studies [13] and reviewed by a sleep physician. Arousals were defined using criteria from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association [14], and respiratory events were rated using the American Academy of Sleep Medicine Task Force criteria [15]. Apnea was defined as a decrease in airflow of at least 80 % for 10 s or more, and hypopnea was defined as a decrease in airflow of at least 50 % for 10 s or more.

Actigraphy

The rest–activity cycle was studied using a piezoelectric sensor placed on the nondominant wrist [16] that generates a signal based on movement (Octagonal Sleep watch 2.01®, Ambulatory Monitoring Inc, Ardsley, NY). Data were collected using the "zero-crossing mode," in 1-min epochs [16]. Computerized analyses were performed according to the algorithm proposed by Cole et al. [17] (Action W 2—version 2.5, Ambulatory Monitoring Inc) for the estimation of actigraphic sleep parameters. The following sleep variables were analyzed: total time in bed (TTB) was defined in minutes from start to end of the bedtime interval, according to event button pressing. Total wake time was defined in minutes of total time scored as awake. Total sleep time (TST) was defined as the actual time spent asleep. Sleep efficiency (SE) was defined as the percentage of time calculated as the TST divided by TTB times 100. Sleep latency (SL) was defined as the time elapsed from pushing the event button and the occurrence of five consecutive sleep epochs. Wake after sleep onset (WASO) was defined as total awakening time after sleep onset.

Recordings were conducted for seven consecutive days under typical life conditions. Sleep logs were completed each day in order to ensure actigraphic data with regard to bedtime and wake time.

Statistical analyses

Subjects' characteristics are presented as means and standard deviations. Kolmogorov–Smirnov normality tests were performed for demographics and polysomnographic

variables. Actigraphy variables were analyzed during week and weekend days.

t and Wilcoxon tests were used to determine differences in actigraphy parameters between week and weekend days. The sample was divided using a similarity test (cluster analysis) taking into consideration the following parameters: TST, SL, SE, and WASO. Sleep duration groups were compared using ANOVA, Chi-square, and Kruskal–Wallis tests. A Bonferroni post hoc test was applied when necessary. *p* values of 0.05 or less were considered statistically significant. A multiple regression model was built considering TST a dependent variable. The statistical analysis was performed using Statistic 8.0 software, (StatSoft Inc, Tulsa, OK).

Results

There were no differences between week and weekend days in actigraphic parameters for the entire sample with the exception of TTBs which were longer in the weekend days compared with week days (8.07±1.35 and 7.65±1.10 h, respectively; $p=0.006$).

According to cluster analysis, the sample was divided into four groups: very short sleepers ($n=11$), short sleepers ($n=21$), intermediate sleepers ($n=56$), and sufficient sleepers ($n=45$). Figure 1 shows the sample distribution of the cluster test. The actigraphic characteristics for all groups are demonstrated in Table 1. Very short sleepers were characterized by a TST of 3.11±0.75 h, SE of 43.17±10.83 %, SL of 1.28±0.80 h, and 2.88±1.10 h of WASO.

Demographic characteristics are presented in Table 2. Neck circumference was larger in short sleepers than intermediate sleepers (37.99±3.40 and 35.88±2.87 cm, respectively; $p=0.04$). Abdominal circumference also demonstrated a significant difference between very short and intermediate sleepers (103.20±8.64 and 91.74±10.28 cm, respectively; $p=0.02$).

Polysomnographic parameters are shown in Table 3. AHI was significantly higher in very short sleepers (50.18±30.86 events/h) than intermediate sleepers (20.36±14.68 events/h; $p=0.007$) and sufficient sleepers (23.21±20.45 events/h; $p=0.02$). Minimal and mean oxygen saturation and the time spent below 90 % saturation were worse in very short sleepers compared with other groups.

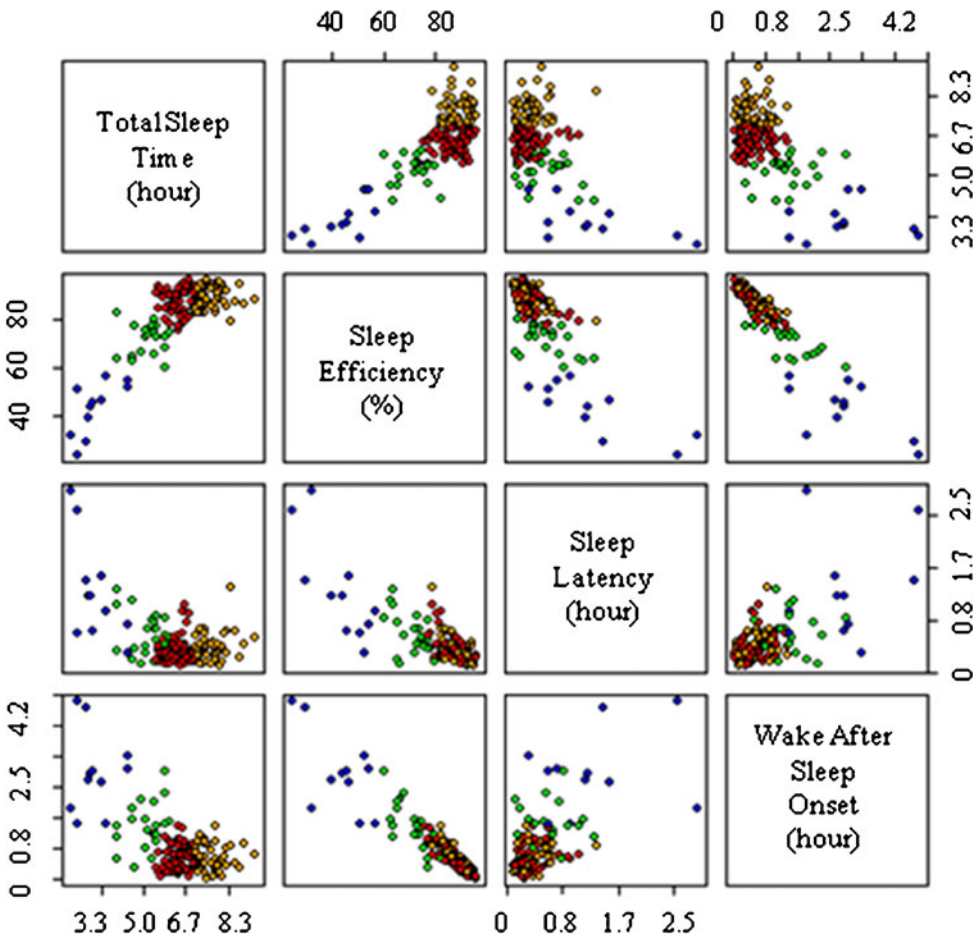

Fig. 1 Sample distribution of the similarity test, taking into consideration the following parameters: total sleep time (in hours), sleep efficiency (in percent), sleep latency (in hours), and wake after sleep onset (in hours). *Yellow cluster* represents sufficient sleepers; *red cluster* represents intermediate sleepers; and *green and blue clusters* represent short and very short sleepers, respectively

Table 1 Sleep characteristics according to actigraphic data among four cluster groups

Actigraphic characteristics	Very short sleepers (n=11)	Short sleepers (n=21)	Intermediate sleepers (n=56)	Sufficient sleepers (n=45)	p
Total time in bed (h)	7.39±1.57	7.24±1.04	7.29±0.61	8.70±0.74	<0.001
Total wake time (h)	4.28±1.62	2.11±0.68	0.92±0.44	0.97±0.45	<0.001
Total sleep time (h)	3.11±0.75	5.14±0.63	6.37±0.40	7.72±0.56	<0.001
Sleep efficiency (%)	43.17±10.83	71.55±6.33	87.63±5.09	89.00±4.53	<0.001
Sleep latency (h)	1.28±0.80	0.61±0.33	0.35±0.20	0.39±0.22	<0.001
Wake after sleep onset (h)	2.88±1.10	1.43±0.63	0.54±0.33	0.56±0.33	<0.001

After adjustment for gender, age, AHI, and BMI, mean oxygen saturation was significantly associated with TST (p=0.01).

Discussion

The main finding of this study is an association of sleep duration and OSA severity.

There are several factors associated with severe OSA including hypertension [5], obesity [7], and metabolic syndrome [18], among others. According to our results, short sleep duration may be another potential variable adding to this process.

Gangwisch et al. [19], in an epidemiologic study, reported a significant relationship between short sleep duration and BMI. Increasing sleep duration from 2–4 to 6 h resulted in a reduction of BMI. Kohatsu et al. [20] also showed an inverse linear relationship between sleep duration and BMI. In a recent study, López-García et al. [21] analyzed 3,576 persons whose habitual sleep duration was reported. Subjects who slept under 5 h had a greater frequency of obesity when compared with subjects who slept 7 h.

In the present study, we did not find a significant difference in BMI between very short, short, intermediate, and sufficient sleep duration. One possible explanation is the presence of obesity and overweight patients in all study groups. Even those who slept about 7 h/night had a BMI of 28.30±4.48 kg/m^2. We found that neck and abdominal circumferences were higher in those who slept 3 to 5 h/night, suggesting that not only obesity, but also central fat distribution, could be associated with sleep duration and OSA [22].

In our study, the group classified as very short sleepers had an AHI of 50.18±30.86 events/h compared with those who slept about 7 h (AHI of 23.21±20.45 events/h; p=0.02). The mechanisms involved in the association of OSA and short sleep duration are unknown but there are three possible explanations: may represent consequences of underlying poor health, short sleep duration may in fact worsen OSA severity, and that shortening of sleep duration may represent a brain defense mechanism. It is well known that an apnea event triggers hypoxia [1], sinus tachycardia [23], and hypertension [5]. Thus, short sleep duration may represent a marker for OSA severity rather than a causal risk factor for disease. Stranges et al. [24], after analyzing 6,472 UK adults and 3,027 US adults, demonstrated that short sleep may represent a risk marker for poorer health outcomes rather than a causal risk factor for disease. Zee and Turek [25] reported that sleep habits may represent a marker of health status and quality of life rather than a causal factor for hypertension and other health outcomes. In contrast,

Table 2 Demographics characteristics in OSA patients according to their sleep duration

Characteristics	Very short sleepers (n=11)	Short sleepers (n=21)	Intermediate sleepers (n=56)	Sufficient sleepers (n=45)	p
Age (years)	55.91±8.86	50.67±9.76	51.23±9.08	54.27±8.38	0.15
Gender (male (n))	8	13	29	21	0.40
BMI (kg/m^2)	30.80±4.82	29.71±4.63	27.74±5.14	28.30±4.48	0.16
Neck circumference (cm)	38.80±3.66	37.99±3.40*	35.88±2.87*	35.84±4.84	0.03
Abdominal circumference (cm)	103.20±8.64**	99.41±10.83	91.74±10.28**	94.61±12.19	0.01
Hip circumference (cm)	107.85±11.06	104.93±9.45	102.36±9.07	101.13±9.93	0.23
Systolic blood pressure (mmHg)	139.55±13.57	135.38±16.93	129.64±17.71	136.48±20.25	0.17
Diastolic blood pressure (mmHg)	88.36±7.68	90.19±13.01	83.66±11.69	87.57±10.88	0.05

*p=0.04; **p=0.02

Table 3 Polysomnographic parameters in OSA patients according to sleep duration

Polysomnographic characteristics	Very short sleepers ($n=11$)	Short sleepers ($n=21$)	Intermediate sleepers ($n=56$)	Sufficient sleepers ($n=45$)	p
Apnea hypopnea index (events/h)	50.18±30.86*· **	32.47±24.15	20.36±14.68*	23.21±20.45**	0.01
Arousal index (events/h)	35.18±28.83***	23.08±16.98	16.63±12.42***	25.26±18.58	0.005
Total sleep time (min)	330.16±113.63	349.38±91.89	354.76±68.71	355.86±60.98	0.96
Sleep stage 1 (%)	3.74±1.73	4.33±2.49	5.22±4.29	6.13±4.52	0.19
Sleep stage 2 (%)	61.13±13.03	56.23±11.76	56.78±13.24	59.68±9.38	0.43
Sleep stages 3 and 4 (%)	15.94±13.08	20.33±10.38	17.61±8.84	15.95±7.93	0.32
REM sleep (%)	19.19±4.61	19.10±5.40	20.25±9.54	18.25±6.69	0.64
Sleep latency (min)	13.87±11.14	15.85±12.49	17.69±16.31	20.86±24.09	0.93
REM sleep latency (min)	98.21±27.85	94.71±63.83	114.91±65.21	133.95±76.28	0.07
Sleep efficiency (%)	75.54±21.82	82.15±11.68	81.26±13.45	79.80±13.24	0.70
Minimal oxygen saturation (%)	71.13±13.32*****, ******, ******	81.14±10.48*****	85.66±5.84******	85.01±6.25******	<0.001
Mean oxygen saturation (%)	91.31±3.66********, ********, **********	94.27±1.58*********	94.69±1.79**********	94.16±1.97**********	<0.001
Time spent below 90 % (min)	60.61±75.91**********	5.05±9.53	3.35±4.14***********	9.28±21.58	0.03

*$p=0.007$; **$p=0.02$; ***$p=0.01$; ****$p=0.04$; *****$p<0.001$; ******$p=0.01$; *******$p=0.01$; ********$p=0.01$; *********$p=0.01$; **********$p=0.02$

several studies suggest that sleep deprivation may worsen OSA. Laudencka et al. [26] showed that acute sleep deprivation may worse OSA index. OSA severity has been classified according to an AHI, but some studies have addressed oxygen saturation [27] and/or presence of cardiovascular comorbidities [28] as factors to be considered. In fact, hypoxia is a strong stimulus to elicit arousal throughout the chemo reflex [27]. We found that mean oxygen saturation is strongly associated with sleep duration.

There is no clear cut-off of sleep duration in the literature, to our knowledge. Fichten et al. [29] considered good sleepers to be those who failed to meet the diagnostic criteria of difficulty initiating or maintaining sleep and reported sleeping well. Hall et al. [30] considered very short sleep as <6 h, short sleep as 6 to 6.99 h, a reference group as 7 to 8 h, and long sleep as >8 h. Gangwisch et al. [9] observed that sleep duration under 5 h/night was associated with a significant increase in the risk of hypertension (hazard ratio, 2.10; 95 % CI, 1.58 to 2.79). Heslop et al. [31] demonstrated that habitual sleep of less than 7 h was associated with an increased mortality.

The lack of a standard classification of sleep duration could be partially explained by the heterogeneity of sleep behavior among different study populations, e.g., age, gender, environment, culture, or socioeconomical status. Moreover, sleep duration has been assessed in the literature using different techniques that could under- or overestimate the TST. Thus, these authors chose to perform a similarity test (cluster analyses) in order to find a suitable classification of sleep duration, based not only on the duration per se using exclusively TST but also considering other parameters, such as SE, SL, and WASO.

The majority of studies assessing sleep duration have used questionnaires which have been considered a controversial measurement tool. Lauderdale et al. [32] found that actigraph-measured duration was shorter than self-reported sleep duration. Mean self-reported sleep duration reflected similar race-gender differences but was, on average, almost 1 h longer than measured duration. This same conclusion was also reached in the Sleep Heart Study [33] in which healthy participants reported sleeping, on average, about 1 h longer than objectively measured sleep duration. Thus, the authors decided to use an objective measurement, actigraphy, to minimize the possibility of sleep misperception.

There are some limitations of this study that need to be considered. First, the cross-sectional design did not allow us to establish causality or temporality. Second, our population was limited to obese or overweight subjects. Third, despite the objective profile of actigraphy, it appears to be less accurate in populations showing fragmented sleep compared with healthy subjects, which is the case for the OSA population [34]. This belief is not shared by all authors, which in the case of some studies [35], reported a high agreement for TST between actigraphy and PSG in patients with sleep-

related breathing disorders. Moreover, this study was not designed to rule out insomnia, thus an overlap between these conditions was not excluded in all participants.

In conclusion, the present study suggests that sleep duration may be associated to low mean oxygen saturation in OSA patients.

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Conflict of interest None

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