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Sleep apnea and oxygen saturation in adults at 2640 m above sea level ☆

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ABSTRACT

Purpose: To describe the SpO_2 in wakefulness, sleep and during the apnea–hypopnea in adults living in Bogotá, located at 2640 m above sea level.

Methods: Descriptive observational study in adults referred for polysomnogram (PSG). A normal Apnea hypopnea index (AHI) was defined as ≤ 5 and obstructive sleep apnea (OSA) was classified as mild (AHI 5–15), moderate (AHI 15–30), and severe (AHI >30). *T*-test or ANOVA test for SpO_2 differences between groups was used.

Results: 1799 patients, 33% women. 222 (12.8%) did not have OSA (normal IAH), 268 (14.9%) mild OSA, 315 (17.5%) moderate ,and 993 (55.2%) severe. In all cases a low SpO_2 ($SpO_2<90\%$) was found.

The SpO_2 was lower when the AHI was higher, in wakefulness, in non-REM and in REM ($p<0.001$). For all grades of severity, SpO_2 decreased significantly from wakefulness to non-REM sleep and to REM sleep ($p<0.001$). Patients with severe OSA had higher desaturation during wakefulness (85.2±6.6%), non-REM sleep (83.1±7.7%), REM sleep (78.8±10.2), and during events (75.1±9.1%).

Conclusions: Patients with OSA at 2640 m have nocturnal desaturation lower than 88%, which decreases with higher severity of OSA. The clinical impact of sleep disorders at this point may be greater than at sea level and should be studied.

1. Introduction

Obstructive sleep apnea (OSA) is a frequent condition [1], characterized by recurrent respiratory pauses which lead to sleep fragmentation and intermittent desaturation.

For the definition of hypopnea and consequently, for the diagnosis of OSA, oxygen desaturation is involved as one of its components [2,3]. Specifically, it is a necessary flow reduction (ventilation) associated with a decrease of more than 3–4% of oxygen saturation, (SpO_2).

Even though persistent and intermittent nocturnal desaturation had been related with the presence of some consequences of OSA, such as pulmonary hypertension and atherosclerosis [4], the role that desaturation has on the pathogenesis, development

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and treatment of pulmonary hypertension in patients with SAHS, is still obscure [5–7].

In Bogotá, a city situated at a high altitude (2640 m above sea level), the normal SpO_2 during wakefulness is found between 90 and 92% [8–12]. Unlike the observed sea level, a decrease of 3% or higher from this point, which is the criterion to define the presence of hypopnea, necessarily implicates the appearance of desaturation and its possible consequences. It is, therefore, very important to know the SpO_2 behavior during sleep at Bogota's altitude as an initial step to investigate the role of altitude and the possible clinical repercussions of oxygen desaturation in patients with SAHS and if these findings justify a redefinition of this syndrome at this altitude.

The objective of this study was to describe the oxygen saturation during wakefulness sleep and apneas–hypopneas in adults at an altitude of 2640 m.

2. Materials and methods

Patients of 18 years or more, sent for basal polysomnogram at the Laboratory of Sleep of Fundación Neumológica Colombiana in Bogota, city situated 2640 m above sea level, between the years of January 2004 and December 2005, were included.

The study was approved by the Ethics Committee of the institution in 2006 and the patients approved the realization of the polysomnogram.

Nocturnal polysomnogram were performed for a minimum of 4 h, with Alice 3 and 4 from Respironics®, following international recommendations: 3 electroencephalogram channels (C3M2, C4M1, O1M2), 2 electro-oculography channels and electromyography (mandible and legs), electrocardiogram, air flow by pressure cannula and thermistor, respiratory effort with thoracic and abdominal belts, finger pulse oxymetry, snoring and position. The SpO_2 was measured by MARS Model 2001 oxymeters with the following technical specifications: Measurement amplitude of 0–100%, precision: 70–100%±25 and a resolution of 1%. The value of oxygen saturation and the pulse frequency were determined by an average of a 2 s sample.

Manual scoring of the records was performed. The sleep stages were classified according to international criteria during the years of study (Reschtchaffen and Kales) [13]. Apnea was defined as the complete cessation of air flow during 10 s or more and hypopnea as the reduction of, at least, 30% of the flow for 10 s, associated to a arousal or a oxygen desaturation of 3% or more. The apnea–hypopnea index (AHI) was defined as the total number of apneas and hypopneas per hour of sleep. The severity of OSA was determined according to AHI: Mild: 5–15/h; moderate: 16 to 30/h; and severe: more than 30/h [14]. Oxygen desaturation was defined by a SpO_2 lower than 90%.

Patients who received oxygen during the study, the studies for CPAP titration, and those with less than 4 h, were excluded.

We used averages and standard deviations for the quantitative variables and the proportions for qualitative variables. To establish differences between the SpO_2 during the different stages of sleep for each group according to AHI, we used the ***t*** test for independent samples and the ANOVA test. To establish the degree of statistical significance of the different proportions, we used the ***chi*** square test. We used Pearson's coefficient to establish the correlation between AHI with age and IMC. We considered it significant when $p<0.05$. We used the statistical programs SPSS 10.0.

3. Results

We included 1799 patients sent to the Laboratory of Sleep for the realization of nocturnal basal polysomnogram, 33% were women and 222 (12.8%) had normal AHI. In the group of sleep apnea, 268 (14.9%) patients presented mild OSA, 315 (17.5%) moderate OSA, and 993 (55.2%) severe OSA. Age and body mass index increased significantly with the grade of severity for OSA ($p<0.001$) (Table 1). There was a significant correlation, even weak, between AHI and BMI ($r=0.325$, $p<0.001$) and between AHI and age ($r=0.167$, $p<0.001$) (Table 1).

The average SpO_2 was inferior to 90% (oxygen desaturation) in all cases, during wakefulness or sleep. All subjects with normal AHI as well as all grades of severity of OSA, were found with a progressive and significant decrease in the average SpO_2, when going from wakefulness to non-REM sleep and from non-REM sleep to REM sleep ($p<0.001$). The higher AHI and OSA severity were found with less SpO_2, during vigil as well as during non-REM and REM sleep ($p<0.001$). Desaturation was larger (lower SpO_2) during apnea or hypopnea events for all the severity groups of OSA. Average SpO_2 during the events were equally lower with the increased severity of OSA ($p<0.001$) (Table 2 and Fig. 1).

Compared with the patients with mild SAHS, the patients with severe OSA had higher desaturation during wakefulness (87.5±5.0 vs. 85.2±6.6), non-REM sleeps (86.6±5.4 vs. 83.1±7.7), REM sleep (84.4±8.7 vs. 78.8±10.2) and during apneas and hypopneas (82±6.9 vs. 75.1±9.1) ($p<0.001$).

4. Discussion

The main finding of this study was that patients with OSA at an altitude of 2640 m above sea level showed a significant oxygen desaturation during sleep, which decreases even more with the increased severity of OSA especially during REM sleep and during the events of apnea and hypopnea, reaching values as low as 78% during REM sleep and 75% during the above mentioned events.

The worst desaturation during REM sleep in patients with sleep apnea has been described and has been related with the duration of apneas during this sleep stage [15].

Table 1 – Characteristics of the patients and distribution of the severity of SAHS.

	N (%)	Age, years	BMI
Normal AHI	222 (12.8)	43.4±16.0	26.0±5.5
Mild OSA	268 (14.9)	50.2±13.3	28.7±5.6
Moderate OSA	315 (17.5)	53.1±13.4	28.0±5.7
Severe OSA	993 (55.2)	54.7±12.8	31.1±6.2

AHI: Apnea hypopnea Index (apnea hypopnea per hour of sleep).
OSA: obstructive sleep apnea.
BMI: body mass index.
Values are averages±SD or N(%).

Table 2 – Average oxygen saturation during wakefulness, sleep stages and respiratory events.

IAH	SpO_2 average wakefulness	SpO_2 average, during sleep			p^*
		Non-REM	REM	Events	
Normal	88.7±6.2	88.1±5.7	87.0±7.3	–	<0.001
Mild OSA	87.5±5.0	86.6±5.4	84.3±8.7	82±6.9	<0.001
Moderate OSA	86.4±6.3	85.2±6.4	83±8.0	80±8.0	<0.001
Severe OSA	85.2±6.6	83.1±7.7	78.8±10.2	75.1±9.1	<0.001
p^{**}	<0.001	<0.001	<0.001	<0.001	

AHI: apnea hypopnea index (apneas-hypopneas per hour of sleep).
OSA: obstructive sleep apnea.
Values as average±SD.
* Value of p when comparing SpO_2 during the different stages of sleep in each group according to IAH.
** Value of p when comparing SpO_2 of the groups according to IAH in each of the stages of sleep.

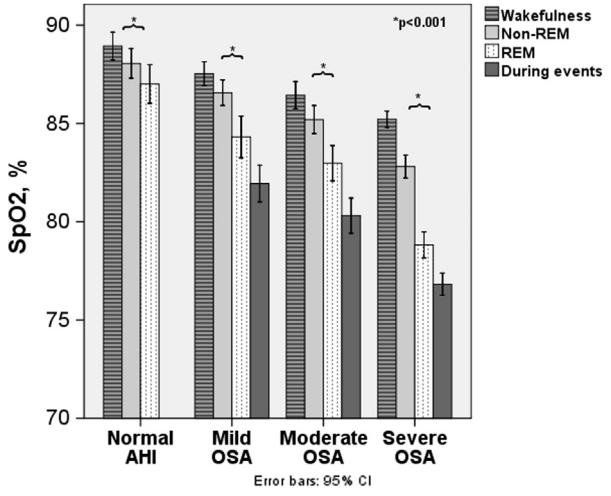

Fig. 1 – SpO_2 (%) in wakefulness, during sleep and the respiratory events according to AHI. SpO_2: oxygen saturation per oxymetry; AHI: Apnea hypopnea index (apneas–hypopneas per hour of sleep).

Patz et al., have studied the effect of altitude upon AHI and did not find important differences between the minimal saturation in three different altitudes (0 m, 1370 m and 2423 m) [16].

Some studies have described the behavior of oxygen saturation during sleep. Gries et al. found that the average saturation in 350 normal subjects during sleep, in Cleveland, Ohio (173 m above sea level), was of 96.5%. The saturation decreased mildly with age, ranging from 96.8% in the group of 1–10 years of age, to 95.1 in the group of 60 years of age [17].

With altitude, the saturation values differ, however, the majority of the studies has been performed at more than 3000 m. In six normal subjects, the average SaO_2 during sleep were of 97.3%, 83.0% and 71.0% measured, respectively at 500 m, 4200 m, and 6400 m [18]. In another study, the saturation at extreme altitude (8400 m), was of 59% [19]. The studies during sleep in intermediate altitudes are few and were performed at simulated altitudes (normobaric hypoxia) [20,21].

Findings of the majority of studies performed at altitude are not comparable with the ones of our study, since they were performed after acute exposition to altitude, which can induce the appearance of central apneas and reflect the changes that are detected with an early adaptation [22]. Rey de Castro et al., did respiratory polygraphy to truck drivers who worked at 2020 m and found out that 12% had central apnea and 10% had OSA. In the study group, the majority had intermittent exposition to the altitude. All desaturation indexes measured on the study were lower in central apnea when compared with OSA [23].

In Bogota (2640 m), the studies of normal values of PaO_2 have been conducted during wakefulness and involves normal and young subjects [8–12]. At this altitude, the average normal values of PaO_2, for the range of age of our patients, are of around 65 mmHg, between 25 and 30% lower than at sea level.

However, at this altitude, PaO_2 is 60 mmHg, from which the curve of hemoglobin dissociation inclines and small changes of PaO_2 have significant repercussions on SpO_2. All the patients with OSA had an average SpO_2 at desaturation levels (SpO_2<90%) with significant decreases according to the increased severity of OSA and going from wakefulness to non-REM sleep, from non-REM sleep to REM sleep and during events, reaching levels of SpO_2 inferior to 80% in patients with severe OSA during REM sleep and during events.

The repercussion of intermittent and persistent hypoxia, reflected upon the levels of desaturation in the patients of the study, might be very significant.

There are several ways to express the severity of desaturation during sleep such as average desaturation, the minimum, and the percentage of sleep time with saturation lower than 90%, all of these are comparable [19]. In this study, we have described the average oxygen saturation.

The strengths of this study were the significant number of patients included and that this is as our best knowledge, the first article describing the behavior of oxygen saturation during sleep in individuals with OSA, living at high altitude (above 2500 m), contributing for better understanding of this pathology among our population and in populations living in similar altitudes.

This study has some limitations: firstly, the population of the study was sent for a polysomnogram evaluation with a suspicion of a sleep disorder, for which there is no well defined normal group. From the total patients, only 12% had a normal AHI(<5/h) with saturation during wakefulness of less than 90%, which suggests that some of these patients might have some pulmonary pathology. Additionally, we have not analyzed separately the population with diagnostic of hypoventilation, obesity, obstructive pulmonary disease, nor cardiovascular comorbities.

Due to the fact that patients with more severe AHI were older and had higher BMI, additional future studies are dully required to evaluate the impact of these variables upon SpO_2 at Bogota's altitude, as well as the possible differences that might exist between genders.

In conclusion, in this group of patients, the oxygen saturation during sleep in patients with OSA at Bogota's altitude, is lower than 88%, decreasing progressively with the increased severity of OSA, especially during REM sleep. The impact of this upon the development of complications such as pulmonary hypertension in patients with SAHS at this time, is still undetermined.

Conflict of interest

There are no conflicts of interests for any of the authors.

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