นิพนธ์ต้นฉบับ

The effects of sleep positions and sleep stages in obstructive sleep apnea

ชวนชม บำรุงเสนา[1]
ศรายุทธ ลูเซียน กีเตอร์[2]

The effects of sleep positions and sleep stages in obstructive sleep apnea

Bamrungsena C[1], Geater SL[2].
[1]Sleep Disorder Service and Research Center (SDSRC), Department of Psychiatry,
[2]Department of Internal Medicine, Faculty of Medicine, Prince of Songkla University,
Hat Yai, Songkhla, 90110, Thailand

Songkla Med J 2009;27(6):471-481

Abstract:

Purpose: *To determine the effect of sleep positions and sleep stages on the apnea-hypopnea index (AHI) with oxygen desaturation (OD)* vs. *without OD in obstructive sleep apnea (OSA) patients.*

Materials and methods*: A retrospective analysis was performed on anthropomorphic and nocturnal polysomnographic (PSG) data of a group of 253 OSA patients (AHI $\geq$ 5) who had used both the supine position (SP) and lateral position (LP) between December 2003 and October 2006.*

Results*: The SP had the strongest influence on the AHI at $p < 0.0001$. The rapid eye movement sleep*

[1]Sleep Disorder Service and Research Center (SDSRC), Department of Psychiatry, [2]Department of Internal Medicine, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, 90110, Thailand

รับต้นฉบับวันที่ 3 กรกฎาคม 2552 รับลงตีพิมพ์วันที่ 18 ธันวาคม 2552

(REMs) has influence on the AHI at p = 0.0283. The effect of the SP on AHI in patients with more body mass index (BMI) and neck circumference (NC) had lesser at p = 0.002 when categorized to nonpositional patient (NPP) and positional patient (PP). The patients with more severe apneic events had more oxygen desaturation index (ODI) than those with milder ones at p < 0.001. The patients with higher BMI had more severe apneic events than those with lesser BMI at p < 0.05.

Conclusion*: The SP and REMs had effect on AHI but they had not effect on OD. There was no relationship between the sleep stages or the sleep positions and AHI with OD. The BMI appeared to be the predictor for the severity of apneic events.*

key words: apnea-hypopnea index, obstructive sleep apnea, oxygen desaturation, sleep position, sleep stage,

บทคัดย่อ:

วัตถุประสงค์: *เพื่อศึกษาผลของท่านอนและระยะการนอนต่อการอุดกั้นทางเดินหายใจ ที่มีการลดลงกับไม่มีการลดลงของออกซิเจนในผู้ป่วยโรคหยุดหายใจขณะหลับจากการอุดกั้นทางเดินหายใจ*

วัสดุและวิธีการ: *ศึกษาแบบย้อนหลัง จากข้อมูลทั่วไปและข้อมูลการตรวจสรีรวิทยาระหว่างการนอน ของผู้ป่วยที่เป็นโรคหยุดหายใจขณะหลับ (มีการอุดกั้นโดยรวมอย่างน้อย 5 ครั้ง) จำนวน 253 ราย ที่ทั้งนอนหงายและตะแคง ระหว่างธันวาคม พ.ศ.2546 - ตุลาคม พ.ศ.2549*

ผลการศึกษา: *ท่านอนหงายมีผลต่อการอุดกั้นมากที่สุดที่ p < 0.0001 ระยะการนอนที่มีการกระพริบตาอย่างรวดเร็วมีผลต่อการอุดกั้นที่ p = 0.0283 ท่านอนหงายมีผลต่อการอุดกั้นลดลงที่ p = 0.002 ในผู้ป่วยที่ดัชนีมวลกายและขนาดรอบคอมากขึ้นเมื่อแบ่งกลุ่มเป็นท่านอนหงายมีผลและไม่มีผลต่อการอุดกั้น ผู้ป่วยที่มีการอุดกั้นที่รุนแรงกว่ามีจำนวนครั้งของการลดลงของออกซิเจนมากกว่า ผู้ที่มีการอุดกั้นระดับน้อยลงที่ p < 0.001 ผู้ป่วยที่มีดัชนีมวลกายมากกว่าจะมีการอุดกั้นรุนแรงกว่าที่ p < 0.05*

สรุป: *ท่านอนหงายและระยะการนอนที่มีการกระพริบตาอย่างรวดเร็วมีผลต่อการอุดกั้น แต่ไม่มีผลต่อการลดลงของออกซิเจนในกระแสเลือด ไม่มีความสัมพันธ์ระหว่างท่านอนหรือระยะการนอนกับการอุดกั้นที่มีการลดลงของออกซิเจน ดัชนีมวลกายเป็นตัวทำนายความรุนแรงของการอุดกั้นทางเดินหายใจ*

คำสำคัญ: *การลดลงของค่าความอิ่มตัวของออกซิเจนในกระแสเลือด, การหยุดหายใจขณะหลับเนื่องจากการอุดกั้นทางเดินหายใจ, จำนวนครั้งของการอุดกั้นทางเดินหายใจส่วนบนใน 1 ชั่วโมงของการหลับ, ท่านอน, ระยะการนอน,*

Introduction

In 1966, Gastaut and colleagues[1] first reported that sleeping in a supine position (SP) was more associated with oxyhaemoglobin desaturation in patients with Pickwickian Syndrome than sleeping in a lateral position (LP). Cartwright in 1984 also found that the apnea-hypopnea index (AHI) decreased significantly by changing from the SP to the LP.[2] None of these studies however, examined the relationship between the sleeping position and the sleeping stage within rapid eye movement sleep (REMs) or non rapid eye movement sleep (NREMs). George and colleagues[3] reported in 1988 that AHI in a SP was higher than in a LP only in NREMs. Moreover, in 1991, Cartwright and colleagues[4] reported a higher AHI in REMs than in NREMs regardless of the sleep position and the differences in apnea severity by sleep position in REMs were found to be equal to those in NREMs.

Obstructive sleep apnea (OSA) patients can be classified into two groups; 1) positional patients (PP), *i.e.* individuals whose supine respiratory disturbance index (RDI) or supine AHI is at least two times higher than their lateral RDI or lateral AHI, and 2) nonpositional patients (NPP), *i.e.* individuals whose supine RDI or supine AHI is less than two times higher than the lateral RDI or lateral AHI.[2, 5-6] Oksenberg and colleagues[5] compared the severity of sleep apneic events occurring in the SP with those in the LP in NPP with severe OSA. They found that the apneic events occurring in the SP were more severe than those occurring while sleeping in the LP.

OSA has been found to be one of the risk factors for stroke, cardiovascular disease (i.e. hypertension (HT) and heart disease (HD)), Diabetes Mellitus (DM) and early mortality.[7-10] Suzuki and colleagues[11] demonstrated that the severity of OSA was independently related to atherosclerosis and that the severity of OSA-related hypoxemia (the duration of an oxygen saturation below 90%) and the mean nadir oxygen saturation were significantly associated with the carotid artery intima-media thickness (IMT), which is regarded as an indicator of atherosclerosis. They also found that the severity of OSA was more important than the frequency of obstructive events. Similarly, Baguet and colleagues[12] found that the severity of oxygen desaturation appeared to be one of the best predictors for carotid IMT and plaque occurrence in OSA patients without cardiovascular disease.

The determining effect of the body position, stage of sleep, and oxygen desaturation on the severity of AHI have been recognized.[1-5] No studies however, have examined the differences between the effects of SP and LP in REMs and NREMs on the AHI with oxygen desaturation (OD) compared with the AHI without OD in OSA patients. Thus current study was undertaken to examine this relationship among OSA patients.

Materials and methods

The polysomnograms of 253 OSA patients (AHI $\geq$ 5) who had undergone a complete polysomnographic (PSG) evaluation both in the SP and LP in the Sleep Disorder Service and Research Center at Songklanagarind Hospital between December 2003 and October 2006 were reviewed.

The PSG recordings were performed using a Biologic polysomnograph (model sleepscan vision: USA), and included the following variables: a two-channel electro-oculogram (EOG); central and occipital electroencephalograms (EEG : C3/A2, C4/A1, O2/A1, O1/A2 of the international 10-20 electrode placement system); a one-channel electrocardiogram (ECG); a two-channel electromyogram (EMG) of the submental muscles; two-channel electromyogram (EMG) of the anterior tibialis muscle of both legs; a one-channel arterial oxyhemoglobin saturation (SaO_2); and body position. The oronasal airflow was monitored by thermister; chest and abdominal efforts were monitored with electric stain gauges with two channels and snoring sounds with one channel, by a microphone located above the patient's trachea. The recordings were conducted at a speed of 10 mm/s, and the sleep stages were scored according to the standard criteria of Rechtschaffen and Kales.[13] Respiratory events were scored as follows: obstructive apnea was defined as an episode of a complete breathing cessation of ≥10 seconds with ongoing respiratory effort; central apnea was defined as complete cessation of airflow lasting at least 10 seconds without concurrent respiratory effort; and hypopnea was defined as any decrease in the amplitude airflow (>50%) from the baseline, or a clear decrease (<50%) in the amplitude followed by either oxygen desaturation (≥4%) or an arousal. Arousals were defined in accordance with the preliminary report from the American Academy of Sleep Medicine (AASM) Task Force.[14] The AHI was calculated as the average number of episodes of apnea and hypopnea per hour of sleep. In this study, a diagnosis of OSA was defined as an AHI of more than five events per hour.[14-15] The severity criteria was based on the AASM Task Force[14], however we modified the criteria defined for mild degree of severity from 5-15/h to 5-14.9/h. According to the AASM Task Force the end value 15/h overlaps with the starting point of the moderate criteria. The severity criteria was defined as mild: AHI 5-14.9/h, moderate: AHI 15-30/h, severe: AHI > 30/h. The oxygen desaturation index (ODI) was calculated by the number of OD (≥4%) per hour of sleep.[14-16] An AHI with OD was calculated by the number of apnea and hypopnea episodes with OD per hour of sleep.[14-16] An AHI without OD was calculated by the number of apnea episodes without OD and hypopnea with arousal per hour of sleep.[14-16] PP had a supine AHI at least two times higher than their lateral AHI, and NPP had a supine AHI less than two times higher than the lateral AHI.[2, 5-6] The Epworth sleepiness scale (ESS), a validated instrument that measures daytime sleepiness, was completed.[17-18] The normal mean score is 4.0±3 (±SD), with excessive daytime sleepiness defined as an ESS score > 10.[17-18] The project was approved by the Ethics Committee for Research in Human Subjects of the Faculty of Medicine, Prince of Songkla University.

Statistical analysis

The results are presented as mean and standard deviation or median with minimum and maximum value. The differences between AHI in SP and LP, REMs and NREMs, with OD were tested using Analysis of Variance (ANOVA, 3-way factorial design). To compare differences in age, body mass index (BMI), neck circumference (NC), Epworth Sleepiness Scale (ESS), oxygen desaturation index (ODI) and sleep efficiency among severity of AHI (mild: AHI 5-14.9/h, moderate: AHI 15-30/h, severe: AHI > 30/h) the ANOVA and Kruskal-Wallis test were used. Comparison of all aforementioned variables between NPP and PP was performed using the Wilcox rank sum test. A p-value < 0.05 was considered statistically significant.

Results

Altogether, 182 males (71.9%) and 71 females (28.1%) were included in the study. Table 1 showed sample characteristics 47% of the subjects were government officers, and mean age was 46.3±14.4 years in men and 51.1±18.5 years in women. The

Table 1 Sample characteristics

Variable	Median (min, max)	
	Male n = 182 (71.9%)	Female n = 71 (28.1%)
Age (years)	48.0 (3.0, 81.0)	52.0 (5.0, 83.0)
BMI (kg/m^2)	26.1 (11.6, 55.5)	28.9 (17.9, 60.5)
Neck circumference (cm)	39.0 (14.5, 57.0)	36.0 (28.0, 46.0)
ESS (unit)	10.5 (0.0, 22.0)	9.0 (1.0, 21.0)
Variable	**n (%)**	**n (%)**
Smoking habit		
No	68 (37.4)	32 (45.1)
Yes	50 (27.5)	4 (5.6)
Not answered	64 (35.2)	35 (49.3)
Alcohol consumption		
No	76 (41.8)	34 (47.9)
Yes	42 (23.1)	3 (4.2)
Not answered	64 (35.2)	34 (47.9)
Hypertension		
No	146 (80.2)	56 (78.9)
Yes	36 (19.8)	15 (21.1)
Heart disease		
No	169 (92.9)	66 (93.0)
Yes	13 (7.1)	5 (7.0)
Diabetes Mellitus		
No	175 (96.2)	68 (95.8)
Yes	7 (3.8)	3 (4.2)

median BMI was slightly lower in men than in women while the neck circumference and ESS were slightly higher in men than in women. The percentages of smoking and drinking were higher in men than in women while the percentages of hypertension, heart diseases and DM were slightly higher in men.

The total AHI was highest in the supine position of the REM sleep stage. The AHI without OD was higher than AHI with OD, regardless of the sleep position and sleep stage (Table 2). The lowest AHI was found while subjects were sleeping in the lateral position of the NREM sleep stage with OD.

No significant interactions between the sleep stages and OD, sleep stage and sleep position, OD and sleep position, and sleep stages, OD and sleep position were found in the analysis using ANOVA,

Table 2 Comparison of AHI with and without oxygen desaturation when sleeping in supine and lateral positions during REM and NREM sleep stages

AHI	REM (n = 253)		NREM (n = 253)	
	Supine (n = 194)	Lateral (n = 232)	Supine (n = 253)	Lateral (n = 253)
Total AHI :				
Mean (SD)	41.5 (36.9)	22.9 (12.6)	35.2 (28.5)	19.8 (6.1)
Median (min, max)	36.9 (0, 209.3)	12.6 (0, 137.5)	26.7 (0, 144.0)	6.1 (0, 184.4)
AHI with OD :				
Mean (SD)	18.2 (24.2)	7.8 (15.8)	15.4 (20.2)	8.0 (16.8)
Median (min, max)	8.6 (0, 141.2)	0 (0, 114.3)	6.9 (0, 96.8)	0.9 (0, 131.3)
AHI without OD :				
Mean (SD)	23.3 (20.9)	15.1 (18.5)	19.8 (18.9)	11.8 (15.5)
Median (min, max)	17.6 (0, 110.2)	7.9 (0, 102.9)	16.5 (0, 144.0)	4.6 (0, 77.2)

3-way factorial design. However, the ANOVA results suggested that all three main effects were significant. The sleep position had the strongest influence on the AHI ($p \leq 0.0001$), followed by OD ($p < 0.0001$) and to a lesser extent the sleep stage ($P = 0.0283$).

We stratified the AHI results on the basis of apnea-hypopnea severity. The median age was highest in the moderate OSA group, compared to the mild and severe groups. There was a dose-response relationship between the BMI, ODI and the severity of OSA. Patients with a higher BMI were significantly more likely to have a higher AHI than those with a lower BMI (Table 3). Patients with severe OSA were significantly more likely to have a higher ODI than those with mild OSA.

Results from categorization by the effect of supine position on AHI

Of the 253 OSA patients, 147 (58.1%) were positional patients. The PP patients tended to be older than the NPP ones. The BMI, neck circumference and oxygen desaturation index were significantly higher in the NPP group than in the PP group (Table 4). AHI in the NPP group tended to be higher than the AHI in the PP group (Table 5).

Discussion

This study found similar results to previous studies that apneic events tend to occur more frequently in patients in the supine position than in the lateral position.[2,3,4] We questioned what could be the mechanism for the greater likelihood of apneic events in the SP compared with the LP. The physiologic mechanism responsible for the effect is most

Table 3 Demographic characteristics categorized by severity groups

Variable	Median (min, max)			P-value
	Mild OSA n = 108 (42.7%)	Moderate OSA n = 71 (28.1%)	Severe OSA n = 74 (29.2%)	
Age (years)	48.0 (3.0, 81.0)	53.0 (3.0, 83.0)	50.0 (9.0, 83.0)	0.024*
BMI (kg/m^2)	26.0 (11.6, 52.1)	26.6 (19.0, 60.5)	29.0 (16.3, 53.1)	0.035*
Neck circumference (cm)	38.0 (14.5, 47.0)	38.5 (32.0, 57.0)	38.5 (30.0, 53.0)	0.191
ODI (time/h)	5.0 (0.0, 27.4)	14.3 (0.5, 47.5)	39.1 (0.2, 93.4)	< 0.001*
Sleep efficiency (%)	84.8 (45.1, 96.5)	85.6 (41.2, 98.6)	82.0 (37.8, 95.8)	0.061
ESS (unit) (mean (SD))	10.1 (4.8)	10.5 (4.8)	11.0 (4.7)	0.518

Table 4 Demographic characteristic comparisons between non-positional and positional patients

Variable	Median (min, max)		P-value
	NPP n = 106 (42%)	PP n = 147 (58%)	
Age (years)	48.0 (3.0, 83.0)	50.0 (8.0, 81.0)	0.034*
BMI (kg/m^2)	29.2 (11.6, 60.5)	25.9 (16.3, 52.1)	< 0.001*
Neck circumference (cm)	39.5 (29.0, 57.0)	38.0 (14.5, 49.5)	0.002*
ESS (unit)	10.0 (0.0, 20.0)	10.0 (1.0, 22.0)	0.871
ODI (time/h)	16.5 (0.0, 93.4)	8.2 (0.4, 51.4)	< 0.001*
Sleep efficiency (%)	82.2 (37.8, 98.6)	84.8 (41.2, 98.5)	0.431

probably related to the effect of gravity on the upper airway (UA). It is known that in the SP, the gravitational forces increase the tendency of the tongue and soft palate to fall back into the throat, causing a smaller caliber of the UA. Thus, in this sleep position, the likelihood for an obstruction in the airway is higher, which would lead to the occurrence of a larger number of breathing abnormalities.[19]

One interesting finding was that apneic events with and without OD were more likely to occur in REMs than in NREMs, which was in agreement with a previous study which found that total REM AHI was higher than NREM AHI.[20] It has also been shown that during REMs there is a reduced tone in the UA muscles, which increases resistance, and therefore the tendency of the UA to collapse is greater.[21] When

Table 5 AHI comparisons between non-positional patients and positional patients

AHI	Median (min, max)					
	REM		P-value	NREM		P-value
	NPP n = 106 (42%)	PP n = 147 (58%)		NPP n = 106 (42%)	PP n = 147 (58%)	
Total AHI	35.0 (0, 160.0)	15.1 (0, 142.0)	< 0.001*	29.3 (1.6, 157.4)	13.4 (2.5, 70.5)	< 0.001*
Total AHI with OD	7.3 (0, 124.4)	2.5 (0, 60.0)	< 0.001*	7.1 (0, 114.5)	3.1 (0, 45.1)	< 0.001*
Total AHI without OD	18.6 (0, 108.2)	8.6 (0, 82.0)	< 0.001*	14.1 (1.3, 102.9)	9.0 (1.3, 61.9)	0.029*
Supine AHI	36.0 (0, 209.3)	37.9 (0, 192.0)	0.989	30.2 (0, 144.0)	25.4 (3.0, 94.9)	0.704
Supine AHI with OD	9.0 (0, 141.2)	7.7 (0, 110.0)	0.710	8.3 (0, 96.8)	6.6 (0, 74.9)	0.463
Supine AHI without OD	16.0 (0, 108.2)	18.3 (0, 110.2)	0.690	13.8 (0, 144.0)	17.2 (1.1, 79.2)	0.056
Lateral AHI	39.0 (0, 137.5)	4.3 (0, 72.0)	< 0.001*	26.8 (0, 184.4)	3.0 (0, 45.0)	< 0.001*
Lateral AHI with OD	7.2 (0, 114.3)	0 (0, 51.0)	< 0.001*	5.3 (0, 131.3)	0 (0, 32.8)	< 0.001*
Lateral AHI without OD	19.2 (0, 102.9)	3.6 (0, 60.0)	< 0.001*	16.2 (0, 77.2)	2.6 (0, 30.3)	< 0.001*

we stratified the data on the basis of apnea-hypopnea severity, we found that patients who had a higher BMI had more incidences of AHI than those with a lower BMI. This can be explained by the fact that the accumulation of fat in the tongue and soft tissues surrounding the pharynx may increase with an increasing BMI causing a smaller caliber of the UA.[22] When we stratified the data to NPP and PP, the PP group were found to be younger and weigh less than the NPP group. In addition, they had fewer and less severe breathing abnormalities than the NPP group, which again is in accordance with the findings of a previous study.[19]

One limitation of our study is that we studied the sleep stage and sleeping position effect without taking into account the effect of sex, age, BMI, and neck circumference on the occurrence and severity of breathing abnormalities during sleep. Future studies should consider these aspects, which might also be of significance as current data on these points have indicated.[23-26]

This study found that all OSA patients, both PP and NPP, had breathing abnormalities, and mainly in the SP. Positional therapy, notably the avoidance of the SP during sleep, would therefore benefit all patients, not mainly for PP as suggested in previous studies.[19, 27] It is, of course, difficult for a patient to control his/her body posture all night long, thus at least in a hospital setting, nurses can play an important role in seeking ways to help patients suffering from this type of problem. In our setting, we found that patients accepted the use of two side-pillows connected together with pieces of cloth wide enough to lock the patient in the lateral position more readily than wearing a shirt with a bag of tennis balls attached to the back of it, as suggested in a previous study.[19] Our study also found that a higher BMI tended to be associated with more severe OSA, thus the role of nurses or other caregivers in providing weight loss educational programs is also important. One study found a reduction of body weight by 10% to be related to a 26% reduction in the AHI.[28] Nevertheless, in spite of the fact that, at present, nasal continuous positive airway pressure (nCPAP) is the best solution for these patients, not all of the patients accept this form of therapy, and many do not comply well with it,[29, 30] and the therapeutic success of nCPAP relies on patient acceptance.[31] The role of the nurse in promoting greater acceptance of therapeutic CPAP education programs, which have shown good results, should also be encouraged.[31]

Conclusions

This paper showed the differences in AHI with and without oxygen desaturation in OSA patients. Apneas and hypopneas with oxygen desaturation occurred more frequently when patients sleeping in a supine position than in a lateral position. Apneas or hypopneas without oxygen desaturation occurred more frequently than events with oxygen desaturation. There was no association between the sleep stages or the sleeping positions and apneas and hypopneas with oxygen desaturation.

References

1. Gastaut H, Tassinari CA, Duron B. Polygraphic study of the episodic diurnal and nocturnal (hypnic and respiratory) manifestations of the Pickwickian syndrome. Brain Res 1966;2:167-86.
2. Cartwright RD. Effect of sleep position on sleep apnea severity. Sleep 1984;7:110-4.
3. George CF, Millar TW, Kryger MH. Sleep apnea and body position during sleep. Sleep 1988; 11:90-9.
4. Cartwright RD, Diaz F, Lioyd S. The effects of sleep posture and sleep stage on apnea frequency. Sleep 1991;14:351-3.
5. Oksenberg A, Khamaysi I, Silverberg DS, et al. Association of body position with severity of apneic events in patients with severe nonpositional obstructive sleep apnea. Chest 2000;118:1018-24.
6. Mador MJ, Kufel TJ, Magalang UJ, et al. Prevalence of positional sleep apnea in patients undergoing polysomnography. Chest 2005; 128:2130-7.
7. Mohsenin V. Sleep-related breathing disorderes and risk of stroke. Stroke 2001;32:1271-8.
8. Shahar E, Whitney CW, Redline S, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. Am J Respir Crit Care Med 2001;163:19-25.
9. Peker Y, Hedner J, Norum J, et al. Increased incidence of cardiovascular disease in middle-aged men with obstructive sleep apnea: a 7-year follow up. Am J Respir Crit Care Med 2002;166:159-65.
10. Marti S, Sampol G, Munoz X, et al. Mortality in severe sleep apnoea/hypopnoea syndrome patients: impact of treatment. Eur Respir J 2002;20:1511-8.
11. Suzuki T, Nakano H, Maekawa J, et al. Obstructive sleep apnea and carotid-artery

intima-media thickness. Sleep 2004;27:129-33.
12. Baguet JP, Hammer L, Levy P, et al. The severity of oxygen desaturation is predictive of carotid wall thickening and plaque occurence. Chest 2005;128:3407-12.
13. Rechtschaffen F, Kales A. A manual of standardization terminology, techniques and scoring systems for sleep stages of human subjects. Los Angeles: BIS/BRI, UCLA; 1968.
14. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. Sleep 1999; 22:667-89.
15. Kushida CA, Littner MR, Morgenthaler T, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. Sleep 2005;28:499-521.
16. Torre-Bouscoulet L, Castorena-Maldonado A, Banos-Flores R, et al. Agreement between oxygen desaturation index and apnea-hypopnea index in adults with suspected obstructive sleep apnea at an altitude of 2240 m. Arch Bronconeumol 2007;43:649-54.
17. Johns M. A new method of measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14:540-5.
18. Johns M. Reliability and factor analysis of the Epworth sleepiness scale. Sleep 1992;15:376-81.
19. Oksenberg A, Silverberg DS, Arons E, et al. Positional vs nonpositional obstructive sleep apnea patients: anthropomorphic, nocturnal polysomnographic and multiple sleep latency test data. Chest 1997;112:629-39.
20. Findley LJ, Withoit SC, Suratt PM. Apnea duration and hypoxemia during REM sleep in patients with obstructive sleep apnea. Chest 1985; 87:432-6.
21. Issa FG, Sullivan CE. Upper airway closing pressure in obstructive sleep apnea. J Appl Physiol 1984;57:520-7.
22. Dixon JB, Schachter LM, O'Brien PE. Predicting sleep apnea and excessive day sleepiness in the severely obese: indicators for polysomnography. Chest 2003;123:1134-41.
23. Dancey DR, Hanly PJ, Soong C, et al. Gender difference in sleep apnea: the role of neck circumference. Chest 2003;123:1544-50.
24. Bixler EO, Vgontzas AN, Ten Have T, et al. Effects of age on sleep apnea in men: I. prevalence and severity. Am J Respir Crit Care Med 1998; 157:144-8.
25. Deegan PC, McNicholas WT. Predictive value of clinical feature for the obstructive sleep apnoea syndrome. Eur Respir J 1996;9:117-24.
26. Davies RJ, Ali NJ, Stradling JR. Neck circumference and other clinical features in the diagnosis of the obstructive sleep apnoea syndrome. Thorax 1992;47:101-5.
27. Berger M, Oksenberg A, Silverberg DS, et al. Avoiding the supine posture during sleep lowers 24 hr blood pressure in obstructive sleep apnea (OSA) patients. J Hum Hypertens 1997;11:657-64.
28. Smith PL, Gold AR, Meyers DA, et al. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. Ann Intern Med 1985; 103:850-5.
29. Kribbs NB, Pack AI, Kline LR, et al. Objective

measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. Am Rev Respir Dis 1993;147:887-95.

30. Fleury B, Rakotonanahary D, Hausser-Hauw C, et al. Objective patient compliance in long term use of nCPAP. Eur Respir J 1996;9:2356-9.

31. Hui DS, Choy DK, Li TS, et al. Determinants of continuous positive airway pressure compliance in a group of Chinese patients with obstructive sleep apnea. Chest 2001;120:170-6.