The high dependency of supine position in obstructive sleep apnea

Selma F. Guven, MD, *Bulent Ciftci,* MD, *Huseyin Lakadamyali,* MD, *Tansu U. Ciftci,* MD.

ABSTRACT

الأهداف: تحديد تردد حالات مرض توقف التنفس الانسدادي عند النوم في موضع النوم المستلق على الظهر و تحديد العلاقات الديموغرافية لهذا المرض.

الطريقة: أجريت دراسة رجعية على 3813 مريض خضعوا لجهاز اختبارات النوم المتعددة لمدة ليلة واحدة في مركز اضطرابات النوم، مستشفى اتاتورك للأبحاث في أمراض الصدر و جراحته، أنقره، تركيا خلال الفترة من يونيو 2007 م حتى يونيو 2010م. شملت الدراسة على 3214 مريض تم تشخيصهم بمرض توقف التنفس الانسدادي عند النوم. مرض توقف التنفس الانسدادي عند النوم المتعلق بوضع النوم المستلق على الظهر هو يُعَرّف طبياً بمؤشّر توقف أو ضعف النفس المعروف بأكثر من 5≥ و نفس المؤشر هذا في وضع النوم المستلق علي الظهر/ وضع النوم الغير مستلق على الظهر بأكثر من 2. تم مقارنة الخصائص بشكل احصائي لهذه الحالتين.

النتائج: أظهرت الدراسة أن 39.9% من جميع المرضى لديهم علاقة بين موضع النوم و توقف التنفس الانسدادي خلال النوم. كانت هذه العلاقة بارزة أكثر عند المرضى الأصغر سناً والأخف وزناً و كان خطورة المرض عندهم أقل.

خاتمة: مرض توقف التنفس الانسدادي عند النوم المتعلق بوضعية النوم المستلقي على الظهر قد يحتاج إلى طريقة علاج مختلفة وهو غير مألوف ويجب أن يعتبر ككيان سريري مختلف.

Objectives: To define the frequency of supine positional obstructive sleep apnea (OSA) in patients diagnosed with OSA and to describe the demographic associations with positional OSA (PO).

Methods: A retrospective study was performed in a total of 3813 patients who underwent full-night polysomnography at the Sleep Disorders Center of the Atatürk Chest Diseases, Thoracic Surgery Education and Research Hospital, Ankara, Turkey between June 2007 and June 2010. A total of 3214 patients diagnosed with OSA were included in the study. Positional OSA was defined as a total AHI ≥5, and supine AHI/non-supine AHI ≥2. Characteristics of positional OSA and non-positional OSA groups were compared statistically.

Results: Patients classified as positional OSA composed 39.9% of all OSA patients. Positional OSA patients were younger with lower body mass index and their OSA was less severe.

Conclusion: Positional OSA, which may require different treatment approaches, is not uncommon among OSA patients and should be understood as a different clinical entity.

Saudi Med J 2013; Vol. 34 (2): 147-152

From the Sleep Disorders Center (Guven, Ciftci B), Ataturk Chest Diseases, Thoracic Surgery Training and Research Hospital, Kecioren, and Sleep Disorders Center (Ciftci T), Faculty of Medicine, Gazi University, Beşevler, Ankara, and the Department of Pulmonary Diseases (Lakadamyali), Baskent University Medical School, Alanya, Antalya, Turkey.

Received 9th October 2012. Accepted 23rd December 2012.

Address correspondence and reprint request to: Dr. Selma F. Guven, Sleep Disorders Center, Ataturk Chest Diseases, Thoracic Surgery Training and Research Hospital, Sancak Mah. 548. sok. 3/12 Cankaya, Sanatoryum Caddesi, Kecioren, Ankara, Turkey. Tel. +90 (532) 6960557. Fax. +90 (312) 3552135. E-mail: selmafiratguven@yahoo.com

Obstructive sleep apnea (OSA) syndrome is a highly prevalent disease, affecting 4% of men and 2% of women.[1] It is characterized by recurring episodes of upper airway obstruction during sleep, leading to reduced (hypopnea) or absent (apnea) airflow at the nose/mouth, and resulting in recurrent arousals and episodic oxyhemoglobin desaturations during sleep.[2] In some OSA patients, the level of respiratory disturbance as judged by apnea-hypopnea index (AHI) is approximately 50-60% higher when they lie on their backs than when they lie on their sides.[3-7] A Starling Resistor Model for upper airway is a useful model of upper airway pressure-flow relationships that contains a collapsible tube with a sealed box interposed between

2 rigid segments: the intranasal cavity, and the trachea. The critical closing pressure (Pcrit) of the airway (at which the airway collapses) is the pressure inside the airway. The pressure gradient during airflow through the system is defined by Pupstream (Pup)-Pcrit. An increase in Pcrit, causes inspiratory airflow limitation, and falling of Pus below Pcrit causes total airway collapse.[8,9] The upper airway patency during sleep is determined by the balance of abductor muscles and intraluminal negative pressure while lying on the back; upper airway cross-sectional area narrows because soft tissues of the upper airway (primarily tongue) shift due to gravity, and the balance deteriorates.[5,8-11] Despite the negative effects of sleeping on the back on upper airway patency, people prefer to sleep in this position.[9] Positional OSA (PO) is said to be present when there is a 50% increase in the AHI while lying in the supine position.[4,6] In PO patients, the severity and frequency of respiratory events depend on how long the patient lies supine.[5] Patients with OSA spend 46-51% of their total sleep time on their backs during polysomnography (PSG).[11] The positive airway pressure (PAP) treatment is undisputedly the first-line therapy option in OSA, although the effectiveness of PAP is often limited by suboptimal acceptance and compliance.[12-15] In patients with PO, positional therapy, which aims to prevent the patient from sleeping in the supine position, represents an alternative to PAP treatment.[16,17] Although many patients have more respiratory events in supine position, only a few studies have been reported regarding positional OSA. Various studies of small groups have analyzed the effect of sleeping position among OSA patients; the prevalence of PO is reported to range from 9-74.5%.[5,16,18,19] In this study, we aimed to investigate in a relatively large series of patients, the frequency of supine positional OSA in patients diagnosed with OSA, and to describe the demographic associations with PO. We also present our analysis of the role of sleep position in OSA.

Methods. A retrospective study was performed in a total of 3813 patients who underwent overnight PSG at the Sleep Disorders Center of the Atatürk Chest Diseases, Thoracic Surgery Education and Research Hospital, Ankara, Turkey between June 2007 and June 2010. A total of 3214 patients diagnosed with OSA are included in the study, and 599 patients are excluded according to the following criteria: sleep disorders other than OSA, such as central sleep apnea, periodic limb movement syndrome, or narcolepsy and patients with previous upper-airway surgery, and patients who slept less than 30 minutes in each position were not included in the study. Our sleep center is located in the training and research hospital of Turkey's capital city, Ankara. Most of our patients are from Ankara and its neighboring surroundings. The study group is composed of regional and ethnical groups in Turkey. This study was performed in accordance with the guidelines of Declaration of Helsinki. The Institutional Review Board of the hospital approved the study. Before enrollment, all subjects gave their written informed consent. Our method for finding the prior related studies was by searching the keywords of obstructive apnea, sleep and supine position in the electronic PubMed media (US National Library of Medicine National Institutes of Health (PubMed).

Polysomnography. All participants underwent overnight polysomnography, using the Compumedics Voyager Digital Imaging E-series system (Compumedics Ltd, Melbourne, Victoria, Australia) or Alice 5 system (Respironics,® PA, USA). The polysomnography recordings included electroencephalography (4 channels), electro-oculography (2 channels), submental electromyography, electrocardiography, and tibial electromyography. Airflow was measured by a nasal pressure transducer (Alice 5 [Pro-Tech Inc, Murrysville, PA, USA], and Compumedics [Southern Lab, CA, USA]), and by a thermistor (Alice 5 [Pro-Tech Inc, Murrysville, PA, USA], and E-Series [Southern Lab, CA, USA]), and respiratory effort by thoracoabdominal piezoelectric belts (Alice 5 [Pro-Tech Inc, Murrysville, PA, USA], and Compumedics [Compumedics Ltd, Melbourne, Victoria, Australia]). Measurement of arterial oxyhemoglobin saturation was performed with a pulse oximeter probe (Alice 5 [Masimo Corp. CA, USA], and Compumedics [Nonin, Nonin Medical, Plymouth, MA, USA). A body position sensor (Alice 5 [Pro-Tech Inc, Murrysville, PA, USA], and Compumedics [Compumedics Ltd, Melbourne, Victoria, Australia]) attached to a thoracic belt was used to monitor body position. Body position of the patients was directly observed by the technician using a low-light camera during the recording of PSG. Sleep stages and respiratory parameters were scored manually according to the standard criteria of the American Academy of Sleep Medicine (AASM), published in 2007.[20] Based on the guidelines of AASM, apnea was

Disclosure. The authors have no conflict of interests, and the work was not supported or funded by any drug company.

defined as ≥90% decrease in airflow relative to the basal amplitude, persisting for at least 10 seconds. Hypopnea was defined as ≥50% decrease in air-flow amplitude relative to the basal amplitude with associated ≥3% oxygen desaturation or arousal, all sustained for at least 10 seconds.[20] The OSA was defined with an overall AHI≥5. Patients were categorized according to the overall AHI; patients were considered as mild OSA with an AHI equal to 5-15, and as moderate-severe OSA with an AHI≥15.[17,21] The PO was defined as supine AHI/non-supine AHI≥2, and non-positional OSA (NPO) was defined as supine AHI/non-supine AHI<2 in OSA patients.[4] The body mass index (BMI) was calculated by dividing body weight in kilograms by the square of the height in meters, and subjects with BMI ≥30 kg/m^2 were classified as obese; BMI - 25.0-29.9 kg/m^2 as overweight; and BMI - 18.5 to 24.9 kg/m^2 as normal.[22] An Epworth Sleepiness Scale (ESS) was completed by each subject on the night of polysomnography.[23] It is a brief, simple, self-administered scale that asks the subject to give a rate on a scale of 0-3, the chances in 8 different situations in daily life (0=would never doze; 3=high chance of dozing). The ESS has a total score range of 0-24, and scores ≥10 are associated with daytime sleepiness.[23]

Statistical analysis. The mean and standard deviations (SD) were determined for continuous variables and percentages were determined for categorical variables. The significance of differences between the PO and the NPO group, and between the mild OSA and the moderate-severe OSA group was analyzed with student's t-test. Categorical data were analyzed by chi-squared and Fisher's exact probability test. All statistical analyses were performed using the Statistical Package for Social Sciences version 11 for Windows (SPSS Inc, Chicago, IL, USA). Differences were considered significant at $p<0.05$.

Results. In the study group, 39.4% were women and 60.6% were men. The characteristics of the patients are demonstrated in **Table 1**. Out of 3214 patients, 838 (26.1%) had mild OSA, and 2376 (73.9%) had moderate-severe OSA. The characteristics of the study group according to severity are shown in **Table 2**. In the study group, 1283 (39.9%) were considered to have PO, and 1931 (60.1%) were NPO. The characteristics of the study group according to position are seen in **Table 3**. Patients grouped as PO compared to NPO were found to be younger with lower BMI and less severe OSA; these trends were significant (p=0.000). Patients in NPO group had higher ESS scores compared to

Table 1 - Characteristics of the patients diagnosed with OSA included in a study at the Sleep Disorders Center of the Atatürk Chest Diseases, Thoracic Surgery Education and Research Hospital, Ankara, Turkey.

Variables	Mean ± SD
Age, years	47.7 ± 11.0
Male/female	1948/1266
Body mass index, kg/ m^2	32.21 ± 5.9
Epworth sleepiness scale	8.0 ± 4.6
AHI	33.8 ± 24.9
AHI supine	43.8 ± 29.5
AHI nonsupine	29.0 ± 25.6
AHI supine/nonsupine	2.9 ± 6.1
Awake SpO_2	92.5 ± 3.7
Mean SpO_2	89.5 ± 5.8
Lowest SpO_2	77.6 ± 14.4

OSA - obstructive sleep apnea, SD - standard deviation, AHI - apnea-hypopnea index, SpO_2 - oxygen saturation by pulse oximeter

Table 2 - Characteristics of the patients included in a study according to severity of OSA at the Sleep Disorders Center of the Atatürk Chest Diseases, Thoracic Surgery Education and Research Hospital, Ankara, Turkey.

Variables	Mild OSA (n=838)*	Moderate-severe (n=2376)*
	Mean ± SD	
Age,* years	43.5 ± 10.5	49.2 ± 10.7
Body mass index,* kg/m^2	29.1 ± 5.1	33.0 ± 6.0
Epworth sleepiness scale*	6.1 ± 4.1	8.7 ± 4.6
AHI*	9.7 ± 3.0	42.3 ± 23.7
AHI supine*	18.7 ± 16.5	52.7 ± 27.9
AHI nonsupine*	7.3 ± 5.3	36.6 ± 25.5
AHI supine/nonsupine*	4.4 ± 7.3	2.4 ± 5.6
Mean SpO_2* (%)	92.2 ± 3.4	88.6 ± 6.2
Lowest SpO_2* (%)	85.4 ± 7.6	74.8 ± 15.1

OSA - obstructive sleep apnea,*all p=0.000, SD - standard deviation, AHI - apnea-hypopnea index, SpO_2 - oxygen saturation by pulse oximeter

Table 3 - Characteristics of patients included in a study according to position dependency at the Sleep Disorders Center of the Atatürk Chest Diseases, Thoracic Surgery Education and Research Hospital, Ankara, Turkey.

Variables	Positional OSA (n=1283)	Non-positional OSA (n=1931)
	Mean ± SD	
Age, years*	44.7 ± 10.5	49.7 ± 10.8
Body mass index,* kg/m^2	30.5 ± 5.1	33.4 ± 6.1
Epworth sleepiness scale*	6.3 ± 4.2	9.2 ± 4.5
AHI*	24.1 ± 18.1	40.2 ± 26.7
AHI supine†	45.1 ± 29.9	43.1 ± 29.2
AHI nonsupine*	13.0 ± 11.3	1.2 ± 0.4
AHI supine/nonsupine*	5.7 ± 9.0	2.4 ± 5.6
Mean SpO_2* (%)	91.4 ± 3.9	88.2 ± 6.5
Lowest SpO_2* (%)	82.5 ± 11.0	74.3 ± 15.4

OSA - obstructive sleep apnea,*p=0.000, †p=0.061, SD - standard deviation, AHI - apnea-hypopnea index, SpO_2 - oxygen saturation by pulse oximeter

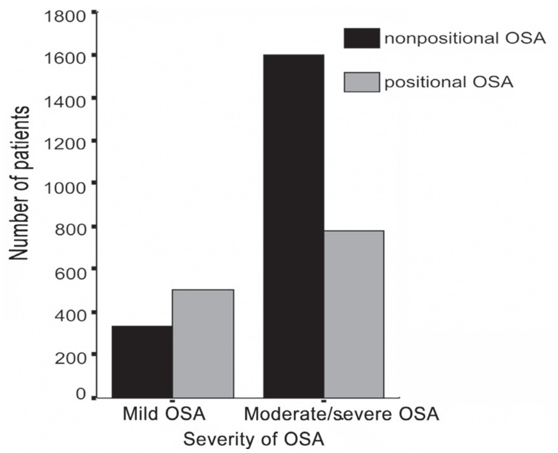

Figure 1 - The relationship between the severity of obstructive sleep apnea (OSA) and position dependency.

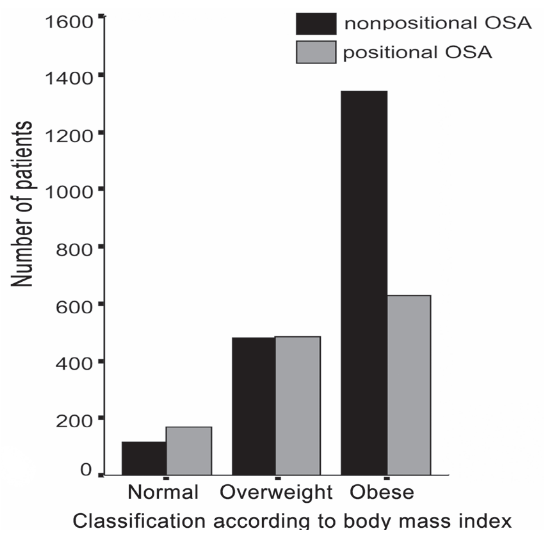

Figure 2 - The relationship between body mass index and position dependency. OSA - obstructive sleep apnea

patients in PO group (p=0.000). According to OSA severity, the frequency of PO was 60.1% in the mild OSA group, and 32.8% in the moderate-severe OSA group (p=0.000) (**Figure 1**). In the study group, 283 patients (8.8%) were of normal weight, 964 (30.0%) were overweight, and 1967 (61.2%) were obese. The PO frequency was 60.1% in the group with normal BMI, 50.3% in the overweight group, and 31.9% in the obese group (p=0.000) (**Figure 2**).

Discussion. In our study, 39.9% of the OSA patients were categorized as PO. In the literature, the prevalence of PO is reported to range from 9-74.5%.[5,18,19] The research presented here represents the largest series yet reported, where the other study groups comprised 248-2077 patients.[3,5,6,16,18,19] In this study, position dependency was more prevalent in patients who were younger, had lower BMI and AHI levels. An increase in the Pcrit of the airway, which causes inspiratory airflow limitation, may be affected by some epidemiological risk factors of OSA such as, male gender, age, and obesity.[2] Although the frequency and the severity of OSA increase with age, there is conflicting data on the relationship between age and PO prevalence. Some authors reported the lack of any age difference between PO and NPO patients, while some reported that PO increases with age.[16,19,24,25] The PO prevalence is generally reported to decrease with age.[5,26] In our study, the PO patients were significantly younger than the NPO patients. With age, fat deposition in the parapharyngeal area increases, the soft palate lengthens, and body structures surrounding the pharynx change.[27-29] All these changes cause a circular narrowing around the pharynx and contribute to the occurrence of respiratory events in non-supine sleep positions. We believe that these age-related changes in the upper airway exacerbate OSA and reduce the prevalence of PO.

Although the prevalence of PO is variable, it has been reported that PO prevalence is higher in mild OSA patients.[5,6,19,25] Oksenberg et al found that PO was more severe and that minimal oxygen saturation values during sleep were significantly lower in NPO patients, in 2 different studies.[5,25] The authors reported that PO prevalence was 66.6% in patients with respiratory disturbance index (RDI) ≤40, and 32.4% in patients with RDI>40.5,25 Tanaka et al[19] found that AHI levels were significantly lower in the PO group than in the NPO group. Mador et al[6] reported the prevalence of OSA was 27.4% on a series of 248 OSA patients in the supine position, furthermore, PO rates were higher in patients with mild OSA. Pevernagie and Shepard[16] studied 81 OSA patients, demonstrating a PO prevalence of 60.5%. The AHI differed only between PO and NPO patients, with higher AHI levels among NPO patients.[16]

In our study, similar to the literature, overall AHI was significantly lower in the PO group, and the PO frequency was higher in the mild OSA group than in the moderate-severe OSA group. The OSA severity has a strong relationship with position dependency and PO is seen more frequently in mild OSA groups. In our opinion, in the natural development of sleep-related breathing disorders, as the severity of disease increases, patients with positional apnea switch to non-positional apnea. Even though obesity is an important risk factor for OSA, the frequency of PO is reduced with increasing BMI.[5,25-27] Itasaka et al[30] used daytime polysomnography in 257 male OSA patients, and they found a reduction of AHI percentage >50% in the lateral position; in patients with normal weight, this reduction was 90.9%; in mildly obese patients, it was 74%; and in the obese group, this reduction was 57.4%.[30] Oksenberg et al[5,25] found that PO prevalence was significantly higher in obese patients than in non-obese counterparts, in 2 different studies in 1997 and 2009.

Obesity was an important determining factor in PO in our study as well. The highest percentage of PO was 60.1% in the normal weight group. The PO rates were 50.3% in the overweight group, and 31.9% in the obese group. The BMI was significantly lower in the PO (30.5 kg/m^2) group compared to the NPO (33.4 kg/m^2 (p=0.000). Obesity is an important risk factor for OSA.[29] Central obesity has been associated with an increase in pharyngeal collapsibility and loss of caudal traction on the upper airway.[31] In OSA related to obesity, fat deposition on the lateral pharyngeal walls and circular narrowing of the upper airway may have reduced position dependence. Position dependency may be less common in obesity-related OSA, because fat deposits around lateral pharyngeal walls and circular narrowing in the upper airway are common. Finally, to quantify daytime sleepiness, we used the ESS, a self-completion questionnaire that measures the subjective perception of sleepiness on a 0-24 point scale. The ESS is positively correlated with the severity of OSA.[32] Here in this study, we found higher ESS scores in the NPO group compared to the PO group. A possible explanation for this finding could be due to the patients in NPO group had more severe disease.

The limitation of our study is its being in retrospective design. If it was planned prospectively, we might have evaluated the spending time and sleep stages on all positions, besides the AHI on supine REM and nonsupine REM could be compared. One might expect that the AHI on the supine positional REM would be higher than the nonsupine positional AHI. Although the prevalence of PO in OSA patients is high, there are limited studies regarding positional treatment's effectiveness in the literature, and we believe that there is a need of further investigations on this issue.

In conclusion, positional OSA is not uncommon among OSA patients and should be understood as a different clinical entity. Evaluation of OSA patients with AHI is not sufficient, OSA patients must be evaluated regarding severity of OSA according to body position.

References

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. ***N Engl J Med*** 1993; 328: 1230-1235.
2. Gaudette E, Kimoff RJ. Pathophysiology of OSA. ***Eur Respir Mon*** 2010; 50: 31-50.
3. Gharibeh T, Mehra R. Obstructive sleep apnea syndrome: natural history, diagnosis, and emerging treatment options. ***Nature and Science of Sleep*** 2010; 2: 233-255.
4. Richard W, Kox D, den Herder C, Laman M, van Tinteren H, de Vries N. The role of sleep position in obstructive sleep apnea syndrome. ***Eur Arch Otorhinolaryngol*** 2006; 263: 946-950.
5. Oksenberg A, Silverberg DS, Arons E, Radwan H. Positional vs nonpositional obstructive sleep apnea patients: anthropomorphic, nocturnal polysomnographic, and multiple sleep latency test data. ***Chest*** 1997; 112: 629-639.
6. Mador MJ, Kufel TJ, Magalang UJ, Rajesh SK, Watwe V, Grant BJ. Prevalence of positional sleep apnea in patients undergoing polysomnography. ***Chest*** 2005; 128: 2130-2137.
7. Akita Y, Kawakatsu K, Hattori C, Hattori H, Suzuki K, Nishimura T. Posture of patients with sleep apnea during sleep. ***Acta Otolaryngol Suppl*** 2003; 550: 41-45.
8. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. ***Chest*** 2007; 132: 325-337.
9. Dempsey JA, Veasey SC, Morgan BJ, O'Donnell CP. Pathophysiology of sleep apnea. ***Physiol Rev*** 2010; 90: 47-112.
10. Eckert DJ, Malhotra A. Pathophysiology of adult obstructive sleep apnea. ***Proc Am Thorac Soc*** 2008; 5: 144-153.
11. Metersky ML, Castriotta RJ. The effect of polysomnography on sleep position: possible implications on the diagnosis of positional obstructive sleep apnea. ***Respiration*** 1996; 63: 283-287.
12. Gay P, Weaver T, Loube D, Iber C, Positive Airway Pressure Task Force; Standards of Practice Committee, American Academy of Sleep Medicine. Evaluation of positive airway pressure treatment for sleep related breathing disorders in adults. ***Sleep*** 2006; 29: 381-401.
13. Bakker J, Campbell A, Neill A. Randomized controlled trial comparing flexible and continuous positive airway pressure delivery: effects on compliance, objective and subjective sleepiness and vigilance. ***Sleep*** 2010; 33: 523-529.
14. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. ***Proc Am Thorac Soc*** 2008; 5: 173-178.
15. Morgenthaler TI, Kapen S, Lee-Chiong T, Alessi C, Boehlecke B, Brown T, et al. Standards of Practice Committee; American Academy of Sleep Medicine: Practice parameters for the medical therapy of obstructive sleep apnea. ***Sleep*** 2006; 29: 1031-1035.
16. Pevernagie DA, Shepard JW Jr. Relations between sleep stage, posture and effective nasal CPAP levels in OSA. ***Sleep*** 1992; 15: 162-167.
17. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Patil SP, et al. Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. ***J Clin Sleep Med*** 2009; 5: 263-276.
18. George CF, Millar TW, Kryger MH. Sleep apnea and body position during sleep. ***Sleep*** 1988; 11: 90-99.
19. Tanaka F, Nakano H, Sudo N, Kubo C. Relationship between the body position-specific apnea-hypopnea index and subjective sleepiness. ***Respiration*** 2009; 78: 185-190.
20. Iber C, Ancoli-Israel S, Chesson AL Jr, Quan SF. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester (IL): American Academy of Sleep Medicine; 2007.

21. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. ***Sleep*** 1999; 22: 667-689.
22. National Institutes of Health Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report. ***Obes Res*** 1998; 6 (Suppl 2): S51-S209.
23. Johns MW. Reliability and factor analysis of the Epworth Sleepiness Scale. ***Sleep*** 1992; 15: 376-381.
24. Young T, Skatrud J, Peppard PE. Risk factors for obstructive sleep apnea in adults. ***JAMA*** 2004; 291: 2013-2016.
25. Oksenberg A, Arons E, Greenberg-Dotan S, Nasser K, Radwan H. [The significance of body posture on breathing abnormalities during sleep: data analysis of 2077 obstructive sleep apnea patients]. ***Harefuah*** 2009; 148: 304-309, 350-351. Hebrew
26. Richard W, Kox D, den Herder C, Laman M, van Tinteren H, de Vries N. The role of sleep position in obstructive sleep apnea syndrome. ***Eur Arch Otorhinolaryngol*** 2006; 263: 946-950.
27. Eikermann M, Jordan AS, Chamberlin NL, G autam S, Wellman A, Lo YL, White DP, Malhotra A. The influence of aging on pharyngeal collapsibility during sleep. ***Chest*** 2007; 131: 1702-1709.
28. Schwab RJ, Remmers JE, Kuna ST. Chapter 101 - Anatomy and Physiology of Upper Airway Obstruction. In: Kryger MH, Roth T, Dement WC, editors. Principles and Practice of Sleep Medicine. 5th ed. Missouri (MO): Elsevier Saunders; 2011. p. 1153-1171.
29. Lam JC, Sharma SK, Lam B. Obstructive sleep apnoea: definitions, epidemiology and natural history. ***Indian J Med Res*** 2010; 131: 165-170.
30. Itasaka Y, Miyazaki S, Ishikawa K, Togawa K. The influence of sleep position and obesity on sleep apnea. ***Psychiatry Clin Neurosci*** 2009; 54: 340-341.
31. Schwartz AR, Patil SP, Laffan AM, Polotsky V, Schneider H, Smith PL. Obesity and obstructive sleep apnea - pathogenic mechanisms and therapeutic approaches. ***Proc Am Thorac Soc*** 2008; 5: 185-192.
32. Guilleminault C, editor. Excessive daytime sleepiness and obstructive sleep apnea syndrome. Excessive sleepiness. Sleep Medicine Clinics. Philadelphia (PA): Elsevier-Saunders; 2006. p. 64-78.

Authorship Entitlement

Excerpts from the Uniform Requirements for Manuscripts Submitted to Biomedical Journals updated November 2003.
Available from www.icmje.org

The international Committee of Medical Journal Editors has recommended the following criteria for authorship; these criteria are still appropriate for those journals that distinguish authors from other contributors.

Authorship credit should be based on 1) substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data; 2) intellectual content; and 3) final approval of the version to be published. Authors should meet conditions 1, 2, and 3.

Acquisition of funding, collection of data, or general supervision of the research group, alone, does not justify authorship.

Author should be prepared to explain the order in which authors are listed.