
Patients with positional versus nonpositional obstructive sleep apnea: a retrospective study of risk factors associated with apnea-hypopnea severity

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Objective. The aim of this study was to investigate the differences in and risk factors for positional and nonpositional obstructive sleep apnea (OSA).

Study design. One hundred twenty-three nonpositional (supine apnea-hypopnea index [AHI] < 2 times the lateral AHI), 218 positional (supine AHI \geq 2 times the lateral AHI), and 109 age-, gender-, and BMI-matched patients with positional OSA performed 2 nights of sleep study. Gender, age, BMI, and percentage of time in supine position, and percentage of time snoring louder than 40 dB were evaluated as risk factors.

Results. Both unmatched positional and matched positional patients had less severe overall AHI values, higher mean SpO₂, lower percentage time SpO₂ less than 90%, and lower percentage of time snoring when compared with the nonpositional group. Overall AHI scores were associated with increasing age and percentage of time snoring for positional and nonpositional groups. However, BMIs were associated with the overall AHI only in the nonpositional group.

Conclusion. The influence of position on OSA severity may contribute to the choice and prognosis of treatment and may represent 2 distinct groups with probable anatomic differences. (*Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2010;110:605-610)

Obstructive sleep apnea (OSA) is a common disorder characterized by recurrent episodes of complete or partial obstruction in the upper airway during sleep.¹ It has been associated with serious sequelae such as daytime somnolence, systemic hypertension, and cardiovascular diseases.²⁻⁴ A number of risk factors for OSA have been identified, including aging, gender, obesity, neck circumference, and facial anatomical abnormalities.⁵⁻⁹

Patients with OSA have been reported to have more obstructive events in the supine position than in the

lateral position, and when this is the case, forcing a change to the nonsupine position during sleep can be an effective treatment.¹⁰⁻¹⁴ Identifying patients with OSA as being either positional or nonpositional thus has important therapeutic implications. Positional OSA patients are defined as those who have a supine respiratory disturbance index (RDI) or apnea-hypopnea index (AHI) at least 2 times higher than their lateral RDI or AHI, and nonpositional patients are those in whom the supine RDI or AHI is less than 2 times higher than the lateral RDI or AHI.¹⁰ Obviously, position restriction therapy is not appropriate for patients with nonpositional OSA.

The anthropomorphic studies that have been reported regarding patient differences between, and risk factors for, positional and nonpositional OSA are few and conflicting. Some investigators have reported that positional OSA patients are younger and less obese; therefore, they have less severe respiratory disturbance than nonpositional patients.^{10,15} Others have found no differences in body mass index (BMI) between positional and nonpositional patients.¹² Although several studies have shown less severe respiratory disturbance in positional OSA patients, the anatomical and physiological mechanisms for this phenomenon have not been clarified. We hypothesized that factors other than age and BMI may contribute to differences in sleep-disordered breathing between positional and nonpositional OSA patients.

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The aim of this study was twofold. First, we wanted to confirm previous reports as to differences in positional and nonpositional OSA patients related to demographics, comorbidities, and AHI values. Second, we wished to investigate risk factors associated with positional and nonpositional OSA. We investigated and compared all positional and nonpositional OSA patients, and also those 2 groups that were matched for age, gender, and BMI. After matching the 2 groups in this fashion, we expected the same risk factors to be associated with AHI in both groups.

METHODS

Subjects

In this retrospective study, we examined 476 records of all the patients who successfully completed a sleep study over 2 nights with a minimum of 4 hours of valid recording time each night. This subset of data was extracted from a database of sleep studies acquired in 2005 through spring 2007 for 1 sleep laboratory (41%), 4 clinical studies (43%), and a number of community-based physician referrals, including dentists; ear, nose, and, throat specialists; primary care physicians; and pulmonologists (16%).¹⁶ Of the 476 records, 341 patients who were categorized as having OSA based on an AHI ≥ 5 were included in this study.

We categorized our patients as having positional or nonpositional OSA following the criteria suggested by Cartwright.¹⁰ Specifically, these criteria state that patients with positional OSA have a supine AHI at least 2 times higher than their lateral AHI, and nonpositional patients will have their supine AHI less than 2 times higher than their lateral AHI. We revised these criteria to include 2 additional rules to improve sampling: (1) all the patients should have at least 30 minutes of supine- and 30 minutes of nonsupine-positioned sleep, and (2) they should have supine AHI ≥ 5 . From the 341 subjects with OSA, 123 patients were categorized as nonpositional OSA patients and 218 as positional patients. A subset of 109 of the 218 positional OSA patients was selected randomly by stratified method to group match the nonpositional OSA group (age, gender, and BMI). In the statistical analyses, we compared all nonpositional patients ($n = 123$) with the unmatched positional patients ($n = 218$) and the group-matched positional patients ($n = 109$). Demographic and comorbidity data of the subjects, including age, gender, height, weight, and neck circumference, were obtained. Descriptive statistics for the 2 groups are presented in the results section. The study was approved by the Institutional Review Board (BioMed, San Diego, CA) and informed consent was obtained from each patient.

Sleep study data

Two-night in-home studies were performed with the Apnea Risk Evaluation System (ARES) Unicorder model 500 (Advanced Brain Monitoring, Carlsbad, CA). The ARES Unicorder measures oxygen saturation, pulse rate, airflow, respiratory effort, snoring levels, head movement, and head position from a wireless recorder self-applied with a single strap to the forehead. Reflectance oximetry was used to obtain the SpO₂ and pulse rate signals. Respiratory effort was derived from the measurement of changes in forehead venous pressure acquired using a combination of photoplethysmography and changes in surface pressure of the reflectance oximetry sensor, and head movement. Airflow was obtained via a nasal cannula and a pressure transducer. A calibrated acoustic microphone was used to acquire quantified snoring levels (dB). Accelerometers were used to measure head movement and derive head position. The recorder was designed to be easily affixed by the patient, and provide alerts during the study if poor-quality airflow or SpO₂ was detected so the device could be adjusted. This device had been evaluated in 2 separate studies against traditional laboratory-based polysomnography and found to have good to excellent sensitivity and specificity.^{17,18}

Automated scoring algorithms were applied off-line to detect sleep-disordered breathing. The AHI was computed using a time-in-bed measure based on recording time with acceptable signal quality minus periods when the patient was upright or presumed to be awake, based on actigraphy. Apneas, based on a 10-second cessation of airflow detected by the automated algorithms, were included in the apnea index (AI) and the apnea-hypopnea index (AHI). Hypopnea events required a 50% reduction and recovery in airflow, and a minimum 3.5% reduction in SpO₂ and at least a 1.0% recovery. After the automated scoring was applied, the full disclosure recordings were visually inspected by a sleep medicine physician to confirm the accuracy of the automated scoring, and to reclassify as central and/or exclude auto-detected events if necessary. The physiological data, including AHI values, percentage time with SpO₂ below 90%, and percentage of time snoring greater than 40 dB were then calculated. The average of data from 2-night recordings were used for the analysis. Finally, the Epworth Sleepiness Scale (ESS) for daytime sleepiness was obtained using a questionnaire at the onset of the sleep recordings.

Statistical analysis

Baseline demographics (age, BMI, and neck circumference), and measures of apnea severity were analyzed for normality with the Kolmogorov-Smirnov test. Differences between positional and nonpositional OSA

Table I. Comparison of demographic data across groups

	A	B	C	P value	
	Nonpositional OSA (n = 123)	All positional OSA (n = 218)	Matched positional OSA (n = 109)	A vs B	A vs C
Age, y	53.4 ± 13.1	52.6 ± 12.0	52.8 ± 11.6	.576*	.734*
Gender, % male	75.6%	83.9%	79.8%	.042†	.272†
BMI, kg/m ²	34.5 ± 7.1	30.2 ± 4.9	33.6 ± 4.3	<.001‡	.291‡
Neck circumference, inch	17.5 ± 1.8	16.6 ± 1.5	17.4 ± 1.4	<.001*	.415‡
AHI severity					
Mild (5-14)	38 (30.9%)	106 (48.6%)	51 (46.8%)	<.001†	<.001†
Moderate (15-30)	21 (17.1%)	70 (32.1%)	30 (27.5%)	<.001†	<.001†
Severe (>30)	64 (52.0%)	42 (19.3%)	28 (25.7%)	<.001†	<.001†

OSA, obstructive sleep apnea; BMI, body mass index; AHI, apnea-hypopnea index.

*P value was obtained from independent *t* test.

†P value was obtained from chi-square test.

‡P value was obtained from Mann-Whitney test.

Table II. Comparison of comorbidity data across groups

Conditions	A	B	C	P value	
	Nonpositional OSA (n = 123)	All positional OSA (n = 218)	Matched positional OSA (n = 109)	A vs B	A vs C
Hypertension, %	58.3	40.2	50.9	.001*	.161*
Heart disease, %	9.3	8.4	8.3	.462*	.490*
Diabetes, %	23.7	10.7	14.8	.002*	.063*
Stroke, %	4.3	1.9	1.9	.175*	.257*
ESS, mean ± SD	11.0 ± 5.6	9.5 ± 4.8	10.0 ± 4.7	.017‡	.149†

OSA, obstructive sleep apnea; ESS, Epworth Sleepiness Scale.

*P values were obtained from chi-square test.

†P value was obtained from independent *t*-test.

‡P value was obtained from Mann-Whitney test.

groups were analyzed with the independent *t* test for normally distributed variables and Mann-Whitney test for nonparametric variables.

The associations between overall AHI values and risk factors including gender, age, BMI, percentage time in supine position, and percentage time of snoring were estimated using multiple linear regression analysis in both the nonpositional group and the matched positional group. Because overall AHI values failed to pass the Kolmogorov-Smirnov test for normality, the AHI values were log-transformed. The transformed data were found to be normally distributed, and then used for multiple linear regression analyses. The linear regression assumptions of linearity, homoscedasticity, and normality of the residuals were successfully evaluated.

RESULTS

Demographics

Age, gender, BMI, neck circumference, and comorbidity data are shown in Tables I and II. There were significant differences in gender, BMI, and neck circumference; prevalence of hypertension and diabetes; and daytime sleepiness by the ESS score between nonpositional OSA patients and unmatched positional OSA

patients. Once we compared the subset of positional patients who were age, gender, and BMI group matched to the nonpositional group, these differences were no longer significant.

Comparison of patients with positional and nonpositional OSA

The sleep data were compared between the first night and second night, and no statistical differences were found. Data from the 2 nights were pooled for the analyses. Table III shows the descriptive data of apnea severity in nonpositional and positional OSA groups (all subjects and matched group). Total percentage of time in supine position during sleep and supine AI and AHI values showed no statistically significant differences between the groups. The data set with all positional OSA patients as well as the matched group had less severe overall AI and AHI values (*P* < .001), less severe nonsupine AI and AHI values (*P* < .001), higher mean SpO₂ (*P* < .001), lower percentage time with SpO₂ less than 90% (*P* < .001), and lower percentage time of snoring louder than 40 dB (*P* < .001) when compared with the nonpositional OSA group.

Table III. Comparison of sleep study data across groups

Variables	A	B	C	P value	
	Nonpositional OSA (n = 123)	All positional OSA (n = 218)	Matched positional OSA (n = 109)	A vs B	A vs C
% time in supine	36.7 ± 27.5	40.1 ± 25.6	38.2 ± 25.0	.168†	.657*
Mean SpO ₂ , %	93.7 ± 3.2	95.7 ± 1.4	95.5 ± 1.4	<.001†‡	<.001†‡
% time SpO ₂ below 90%	13.1 ± 18.5	2.8 ± 5.7	3.5 ± 6.7	<.001†‡	<.001†‡
% time of snoring >40 dB	35.1 ± 16.8	24.0 ± 17.0	26.5 ± 17.3	<.001*	<.001†‡
AI (events/h)	21.3 ± 22.8	10.9 ± 10.6	10.8 ± 10.2	.001†‡	.006†
AHI (events/h)	35.3 ± 26.0	18.9 ± 12.8	20.3 ± 13.6	<.001†‡	<.001†‡
Supine AI (events/h)	26.4 ± 27.1	26.4 ± 36.1	28.7 ± 46.3	.342†	.381†
Supine AHI (events/h)	37.4 ± 29.5	38.6 ± 35.8	42.2 ± 46.0	.345†	.237†
Nonsupine AI (events/h)	18.2 ± 21.3	3.3 ± 5.0	3.3 ± 4.6	<.001†‡	<.001†‡
Nonsupine AHI (events/h)	33.7 ± 25.3	8.8 ± 8.5	10.1 ± 9.6	<.001†‡	<.001†‡

OSA, obstructive sleep apnea; AI, apnea index; AHI, apnea-hypopnea index.

*P values were obtained from independent t-test.

†P values were obtained from Mann-Whitney test.

‡Significant differences after Bonferroni correction.

Table IV. Multiple linear regression analysis of the risk factors on the log overall AHI of the nonpositional OSA group

Risk factors	β	Coefficient	95% CI	P value
Gender (male)	0.290	0.582	0.260, 0.903	<.000
Age	0.180	0.012	0.001, 0.023	.033
BMI	0.207	0.026	0.005, 0.046	.016
% time in supine	0.130	0.004	-0.001, 0.009	.126
% time of snoring >40 dB	0.299	0.016	0.007, 0.024	<.001

Multivariate analysis of variance F-test $P < .001$, adjusted R square = 0.262.

OSA, obstructive sleep apnea; AHI, apnea-hypopnea index; BMI, body mass index; CI, confidence interval.

Table V. Multiple linear regression analysis of the risk factors on the log overall AHI of the unmatched positional OSA group

Risk factors	β	Coefficient	95% CI	P value
Gender (male)	0.132	0.235	0.115, 0.454	.035
Age	0.334	0.018	0.011, 0.025	<.001
BMI	0.015	0.002	-0.015, 0.019	.813
% time in supine	0.232	0.006	0.003, 0.009	<.001
% time of snoring >40 dB	0.391	0.015	0.010, 0.020	<.001

Multivariate analysis of variance F-test $P < .001$, adjusted R square = 0.229.

OSA, obstructive sleep apnea; AHI, apnea-hypopnea index; BMI, body mass index; CI, confidence interval.

Impacts of risk factors on the severity of apnea-hypopnea index

In Tables IV, V, and VI, we present the results of the multiple regression analyses for nonpositional, unmatched positional, and matched positional OSA groups. Both unmatched and matched positional groups showed similar results. Age (nonpositional: $\beta = 0.180$,

Table VI. Multiple linear regression analysis of the risk factors on the log overall AHI of the matched positional OSA group

Risk factors	β	Coefficient	95% CI	P value
Gender, male	0.089	0.150	-0.148, 0.449	.320
Age	0.351	0.020	0.010, 0.030	<.001
BMI	-0.038	-0.006	-0.033, 0.022	.678
% time in supine	0.253	0.007	0.002, 0.011	.004
% time of snoring >40 dB	0.447	0.017	0.010, 0.024	<.001

Multivariate analysis of variance F-test $P < .001$, adjusted R square = 0.254.

OSA, obstructive sleep apnea; AHI, apnea-hypopnea index; BMI, body mass index; CI, confidence interval.

$P = .033$; unmatched positional: $\beta = 0.334$, $P < .001$; matched positional: $\beta = 0.351$, $P < .001$) and percentage time snoring (nonpositional: $\beta = 0.299$, $P < .001$; unmatched positional: $\beta = 0.391$, $P < .001$; matched positional: $\beta = 0.447$, $P < .001$) were associated with the log AHI scores for nonpositional and both unmatched and matched positional OSA patients. The percentage of time in supine was associated only with the log AHI of both unmatched and matched positional patients (unmatched positional: $\beta = 0.232$, $P < .001$; matched positional: $\beta = 0.253$, $P < .001$). BMIs ($\beta = 0.207$, $P = .016$) were associated only with log AHI values of nonpositional patients.

DISCUSSION

As was expected, we found several factors that are important in the progression of apnea severity. Although our data are cross-sectional in nature, our linear regression analysis showed that the AHI severity of both unmatched and matched positional OSA patients appeared to be primarily affected by age (and percent-

age of time spent on supine position), but surprisingly, we did not find a strong influence on severity from BMI. When we put the neck size instead of BMI into our regression analysis we found that neck size was not associated with increased OSA severity in the positional patients either. The AHI severity appeared to be strongly influenced by BMI only in the nonpositional OSA group. These findings need to be confirmed with longitudinal data.

Our results show that patients with nonpositional OSA have more severe overall AI and AHI values, and more severe nonsupine AI and AHI values compared with the unmatched positional OSA patients. And also, they showed lower mean oxygen saturation, and higher percentage snoring time compared with the unmatched positional OSA patients. These results generally agree with a prior study by Oksenberg et al.¹⁵ However, the differences in sleep-disordered breathing severity between these 2 groups are not fully explained by time spent in the supine position or by supine AI and AHI values. We found that the nonsupine AHI values remain significantly higher in nonpositional patients even after controlling for age, gender, BMI, and neck size. These results argue against the hypothesis that positional OSA patients are simply younger and less obese than nonpositional OSA patients.

Although we are not yet ready to offer a specific hypothesis that might explain what factors push someone to become a nonpositional OSA patient above and beyond age and BMI, we did find that after controlling for age, gender, BMI, and neck size, diabetes was the only OSA-associated comorbidity with increased prevalence in the nonpositional group ($P = .06$). This observation provides further evidence of the inverse relationship between glucose intolerance and OSA severity.¹⁹

Based on results from this study, we suggest that patients with positional and nonpositional OSA form 2 distinct but overlapping etiologies in which airway length²⁰ and craniofacial features²¹ influence genioglossal responsiveness to negative-pressure pulses in the lateral position.²² Our conclusions are supported by previous findings that neck size and BMI do not influence the critical pressure in which the pharynx collapses (Pcrit).²³ This interpretation would help to explain why patients with positional OSA are more efficaciously treated with an intraoral mandibular advancement device compared with nonpositional patients.²⁴ Although based on different grouping criteria from our study, the report of Yoshida et al.²⁵ also confirms that effectiveness of oral appliance therapy is greatly influenced by sleep posture. Group distinctions might also explain differences in continuous airway pressure (CPAP) needed to maintain airway patency in

the lateral position. Unfortunately, we did not directly measure pharyngeal collapsibility in our subjects, nor did we gather 3-dimensional images of the airway to see if any substantive anatomic differences existed. Further studies of dynamic pharyngeal airway collapsibility should be performed to identify anatomical factors that provide the greatest contribution to defining positional and nonpositional OSA. Assuming individuals can be fitted into 2 distinct etiological groups, one could then predict the benefit of different OSA treatment options based on each treatment's mechanism of action. The categorization of individuals by positional phenotype is also needed to adequately assess the risk of complications in patients with severe positional and mild overall OSA (because of limited supine sleep time). It would be of clinical benefit for the clinician to be able to assess the likelihood of increased symptomatology in patients requiring postoperative nursing care and confined to a supine position as a result of a temporary injury. Results from this study argue in favor of the mandatory measurement and reporting of supine and nonsupine OSA severity in the diagnosis of OSA by laboratory polysomnography and home sleep testing.^{26,27}

Two limitations of this study should be mentioned. First, because the ARES measured head position instead of body position, the amount of prone sleep is very limited. Virtually all nonsupine sleep is in the lateral left or right position. It would be interesting to evaluate differences between sleep-disordered breathing on the left or right side but we would need additional information that might allow us to interpret differences (handedness, separate measures of flow by nostril, as changes in flow over the course of the night may be influenced by sleep position). Second, this study was limited by its retrospective design. Retrospective studies may result in underestimation of problems residing in the studied population owing to bias in subject selection and interpretation of the data. However, in this study we analyzed all the obtained data that fitted in the generally accepted selection criteria. So we believe that the data and following analysis in this study are free of the selection bias that commonly occurs in retrospective studies.

In conclusion, the influence of position on OSA severity may contribute to the choice and prognosis of treatment with a lower prognosis existing for nonpositional OSA patients and it may represent 2 distinct groups with probable anatomic differences.

REFERENCES

1. McNicholas WT. Diagnostic criteria for the sleep apnoea syndrome: time for consensus. *Eur Respir J* 1996;9:634-5.

2. Seneviratne U, Puvanendran K. Excessive daytime sleepiness in obstructive sleep apnea: prevalence, severity, and predictors. *Sleep Med* 2004;5:339-43.
3. Narkiewicz K, Wolf J, Lopez-Jimenez F, Somers VK. Obstructive sleep apnea and hypertension. *Curr Cardiol Rep* 2005;7:435-40.
4. Arias MA, Sánchez AM. Obstructive sleep apnea and its relationship to cardiac arrhythmias. *J Cardiovasc Electrophysiol* 2007;18:1006-14.
5. Block AJ, Boysen PG, Wynne JW, Hunt LA. Sleep apnea, hypopnea and oxygen desaturation in normal subjects: a strong male predominance. *N Engl J Med* 1979;300:513-7.
6. Davies RJ, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnoea syndrome. *Eur Respir J* 1990;3:509-14.
7. 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;328:1230-5.
8. Richman RM, Elliott LM, Burns CM, Bearpark HM, Steinbeck KS, Catterton ID. The prevalence of obstructive sleep apnoea in an obese female population. *Int J Obes Relat Metab Disord* 1994;18:173-7.
9. Deegan PC, McNicholas WT. Predictive value of clinical features for the obstructive sleep apnoea syndrome. *Eur Respir J* 1996;9:117-24.
10. Cartwright RD. Effect of sleep position on sleep apnea severity. *Sleep* 1984;7:110-4.
11. Cartwright RD, Diaz F, Lloyd S. The effects of sleep posture and sleep stage on apnea frequency. *Sleep* 1991;14:351-3.
12. Pevernagie DA, Shepard JW. Relations between sleep stage, posture and effective nasal CPAP levels in OSA. *Sleep* 1992;15:162-7.
13. Nakano H, Ikeda T, Hayashi M, Oshima E, Onizuka A. Effects of body position on snoring in apneic and nonapneic snorers. *Sleep* 2003;26:169-72.
14. Oksenberg A, Silverberg D, Offenbach D, Arons E. Positional therapy for obstructive sleep apnea patients: a 6-month follow-up study. *Laryngoscope* 2006;116:1995-2000.
15. 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;112:629-39.
16. Westbrook PR, Levendowski DJ, Zavora T, Scarfeo D, Berka C, Popovic D. Night to night variability of in-home sleep studies—Is one night enough? *Sleep* 2007;30(Abstr Suppl):A188.
17. Westbrook PR, Levendowski DJ, Cvetinovic M, Zavora T, Velimirovic V, Henninger D, et al. Description and validation of the Apnea Risk Evaluation System: a novel method to diagnose sleep apnea-hypopnea in the home. *Chest* 2005;128:2166-75.
18. Ayappa I, Norman RG, Seelall V, Rapoport DM. Validation of a self-applied unattended monitor for sleep disordered breathing. *J Clin Sleep Med* 2008;4:26-37.
19. Aronsohn RS, Whitmore H, Van Cauter E, Tasali E. Impact of untreated obstructive sleep apnea on glucose control in Type 2 diabetes. *Am J Respir Crit Care Med* 2010;181:507-13.
20. Segal Y, Malhotra A, Pillar G. Upper airway length may be associated with the severity of obstructive sleep apnea syndrome. *Sleep Breath* 2008;12:311-6.
21. Hsu PP, Tan AKL, Chan YH, Lu PKS, Blair RL. Clinical predictors in obstructive sleep apnoea patients with calibrated cephalometric analysis—a new approach. *Clin Otolaryngol* 2005;30:234-41.
22. Malhotra A, Trinder J, Fogel R, Stanchina M, Patel SR, Schory K, et al. Postural effects on pharyngeal protective reflex mechanisms. *Sleep* 2004;27:1105-12.
23. Sforza E, Petiau C, Weiss T, Thibault A, Krieger J. Pharyngeal critical pressure in patients with obstructive sleep apnea syndrome. Clinical implications. *Am J Respir Crit Care Med* 1999;159:149-57.
24. Chung JW, Enciso R, Levendowski DJ, Morgan TD, Westbrook PR, Clark GT. Treatment outcomes of mandibular advancement devices in positional and nonpositional OSA patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2010;109:724-31.
25. Yoshida K. Influence of sleep posture on response to oral appliance therapy for sleep apnea syndrome. *Sleep* 2001;24: 538-44.
26. Kushida CA, Littner MR, Morgenthaler T, Alessi CA, Bailey D, Coleman J Jr, et al. Practice parameters for the indications of polysomnography and related procedures: an update for 2005. *Sleep* 2005;28:499-521.
27. Collop NA, Anderson WM, Boehlecke B, Clarman D, Goldberg R, Gottlieb DJ, et al. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. *J Clin Sleep Med* 2007;3:737-47.

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