Nasal Obstruction and Palate-Tongue Position on Sleep-Disordered Breathing

Hyo Yeo Kim · Jong In Jeong · Hun-Jong Dhong · Jung Heob Sohn · Sang Duk Hong · Joon Ho Kim · Seong Yun Jang · Yong Gi Jung · Seung-Kyu Chung

Department of Otorhinolaryngology-Head and Neck Surgery, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

INTRODUCTION

Sleep-disordered breathing (SDB) is characterized by periodic reductions or cessation of the airflow during sleep and this leads to hypoventilation, apnea and sleep fragmentation. SDB is mostly caused by narrowing of the upper airway tract. collapsibility of the upper airway is one of the mechanisms involved in the pathogenesis of SDB, and a large tongue base could be associated with a higher risk of SDB because it can make the airway narrower [1].

Nasal breathing is the preferred route of breathing when we are awake and sleep. The relationship between the nasal airway and the collapse of the upper airways is complex, and the precise role played by the nasal airway in SDB is as yet unknown. But if a given patients has nasal obstruction, then they must breathe through the oral cavity and this mouth breathing lengthens and narrows the upper airway and makes it more collapsible to inspiratory negative pressure [2]. It may cause breathing through the upper airway more difficult to perform and sleep becomes more fragmented.

So in this study, the authors tried to evaluate whether the presence of nasal obstruction makes a change on the association between the modified Mallampati score (MMS) and the severity of SDB and sleep quality.

Objectives. We wanted to evaluate whether the presence of nasal obstruction makes a change on the association between the modified Mallampati score and the severity of sleep-disordered breathing (SDB) and the sleep quality.

Methods. Polysomnography (PSG), the modified Mallampati score (MMS), the body-mass index, and a questionnaire about nasal obstruction were acquired from 275 suspected SDB patients. The subjects were divided into two groups according to the presence of nasal obstruction. The clinical differences between the two groups were evaluated and the associations between the MMS and PSG variables in each group were also assessed.

Results. Significant correlations were found between the MMS and many PSG variables, including the apnea-hypopnea index, the arousal index and the proportion of deep sleep, for the patients with nasal obstruction, although this was not valid for the total patients or the patients without nasal obstruction.

Conclusion. The severity of SDB and the quality of sleep are well correlated with the MMS, and especially for the patients with nasal obstruction. The MMS can give more valuable information about the severity of SDB when combined with simple questions about nasal obstruction.

Keywords. Sleep apnea syndrome, Nasal obstruction, Mouth breathing, Modified Mallampati score, Sleep disordered breathing
MATERIALS AND METHODS

We enrolled the consecutive adult patients who were more than 18 years old with suspected SDB and who underwent polysomnography (PSG) from January 2009 to May 2010. The study was approved by the institutional ethics committee. They had been referred to our department with complaints of snoring and sleep apnea. The patients we excluded from this study had already undergone an operation for obstructive sleep apnea syndrome (OSAS) or they had shown evidence of reduced cardiac function and/or chronic obstructive pulmonary disease at the time of the study. The patients with incomplete data were also excluded. The patients were interviewed and given a questionnaire on whether they usually have nasal obstruction when they are in the supine position and the patients were assigned to the ‘without-nasal-obstruction’ group and the ‘with-nasal-obstruction’ group.

All the patients received a full otolaryngologic evaluation, including the modified MMS, and they had undergone diagnostic PSG. The MMS is often used to assess the tongue base and its relation to the soft palate [3]. All the subjects had their height and body weight recorded and the body-mass index (BMI) was calculated. The MMS was evaluated according to the Friedman’s classification described in our earlier study [4,5].

PSG was performed using the Alice3 (Healthdyne Technologies, Marietta, GA, USA) or Somnologica system (Embla, Broomfield, CO, USA). The day before the sleep studies, subjects were asked not to drink alcohol or caffeinated beverages. Overnight polysomnography was performed using a 4-channel electroen-cephalogram (EEG; C3/A2, C4/A1, O1/A2, O2/A1), a 4-channel electrooculogram, an electromyogram (the submental, inter-costal and anterior tibialis muscles, and an electrocardiogram with surface electrodes. A thermistor (for monitoring nasal airflow), a nasal air pressure monitor, an oximeter (for measuring oxygen saturation), piezoelectric bands (for determining thoracic and abdominal wall motion), and a body position sensor were also attached to the patients. The subjects were recorded on videotape using an infrared video camera and they were continuously observed by a PSG technician. The sleep architecture was scored in 30 second epochs, and the sleep staging was interpreted according to the standard criteria of Rechtschaffen and Kales. Apneas and hypopneas were defined by previously reported criteria [6]. Obstructive apnea was defined as a reduction in the airflow >90% lasting ≥10 seconds during which time there was evidence of persistent respiratory effort. Hypopnea was defined as reduction in airflow by 50% with a duration ≥10 seconds or a reduction of airflow by 30% for more than 10, and this was accompanied by EEG arousal and/or 3% or greater oxygen desaturation.

According to the American Sleep Disorders Association Task Force criteria [6], arousals were classified as breathing-related arousals (occurring within 3 seconds following apnea, hypopnea or snoring) and other type of arousals (spontaneous arousal, periodic limb movement-associated arousals). The percentage of time spent in the non-rapid eye movement state versus the time spent in the rapid eye movement (REM) state and the central apnea were also recorded.

Statistical analysis was performed with SPSS ver. 17.0 (SPSS Inc., Chicago, IL, USA). Spearman correlation analysis was performed to evaluate the associations between the MMS and PSG variables, including apnea-hypopnea index (AHI), arousal index and the proportion of deep sleep (deep sleep%) and REM sleep (REM sleep%). Partial Spearman correlation analysis was performed with adjustment for possible covariates. This statistical evaluation was also separately performed in the patient groups with and without nasal obstruction.

Chi-square tests were performed to verify the differences of gender, the SDB severity and the MMS according to the presence of nasal obstruction. Student t-test was used to evaluate the difference of BMI and age between the patients with and without nasal obstruction. The significance level was set at P<0.05 for all the analyses.

RESULTS

Two hundred ninety eight patients visited our clinic and underwent PSG during the recruitment period. Twenty three patients were excluded from the study because of incomplete PSG data (n=9), data loss about nasal obstruction (n=8), known bronchial asthma (n=2), and previous OSAS surgery (n=4).

So, 275 patients were finally included in this study. They were composed of 217 males and 58 females. The female to male ratio was significantly higher in the without-nasal-obstruction group.

| Table 1. Anthropometric and clinical data of the present study |
|-------------------|-------------------|-----------------|-----------------|
|                  | Total            | (+)             | (-)             |
| Sex (male/female)| 217:58 (76.9%/21.1%) | 125:23 (84.5%/15.5%) | 92:35 (72.4%/27.6%) |
| Age (year)       | 45.9±12.8 | 41.8±12.9 | 50.7±11.0 |
| Body mass index (kg/m²) | 23.9±4.2 | 23.6±4.1 | 24.3±4.3 |
| Tonsilar grade   | 1.5±0.8 | 1.6±0.8 | 1.5±0.7 |
| Apnea-hypopnea index | 25.9±23.6 | 25.0±23.9 | 27.0±23.4 |
| P-value          | 0.015 | <0.001 | 0.165 | 0.256 | 0.478 |
The mean age was 45.9 ± 12.8 and this was higher in the without-nasal-obstruction group (P < 0.001). The mean BMI was 23.9 ± 4.2 and it showed no statistical difference between the two groups (P = 0.165). Mean tonsillar grades didn’t show any statistical difference between the two groups either (P = 0.256). All the anthropometric data is summarized in Table 1.

The mean AHI was 25.9 ± 23.6 and the subjects were composed of 55 simple snorers (20.0%), 74 mild OSAS patients (26.9%), 48 moderate OSAS patients (17.5%), and 98 severe OSAS patients (35.6%) (Fig. 1). There was no statistical difference of the AHI between the two groups with/without nasal obstruction (P = 0.478) (Table 1). The distribution of the MMS in the total patients was as follows: 23 patients (8.4%) were MMS I, 79 patients (28.7%) were MMS II, 70 patients (25.5%) were MMS III and 103 patients (37.5%) were MMS IV. The distribution of the MMS showed no statistical difference between the patients with and without nasal obstruction (P = 0.178) (Fig. 2).

No significant correlation was found between the MMS and the PSG parameters such as total AHI, arousal index, REM sleep% (REM sleep time/total sleep time × 100), the deep sleep% (deep

**Table 2. Polysomnographic data of the subjects**

| Modified Mallampati score | Total AHI | Arousal index | REM sleep% | Deep sleep% | Lowest SaO₂ |
|---------------------------|-----------|---------------|-------------|-------------|-------------|
| Total I                   | 17.6 ± 21.4 | 25.8 ± 16.5   | 19.7 ± 5.7  | 9.3 ± 10.2  | 84.9 ± 6.9  |
| II                        | 22.9 ± 21.8 | 26.7 ± 15.3   | 19.3 ± 5.4  | 6.6 ± 7.9   | 83.6 ± 8.7  |
| III                       | 28.7 ± 24.2 | 31.5 ± 18.2   | 19.0 ± 6.5  | 4.6 ± 5.3   | 82.5 ± 7.8  |
| IV                        | 27.5 ± 24.8 | 30.2 ± 16.9   | 18.6 ± 5.6  | 5.3 ± 7.0   | 82.0 ± 6.5  |

Spearman’s ρ | P-value | Spearman’s ρ | P-value

**With nasal obstruction**

| I             | 12.7 ± 12.4 | 21.0 ± 9.0   | 19.4 ± 6.8  | 13.2 ± 10.5 | 85.9 ± 5.3  |
| II            | 19.1 ± 18.7 | 23.9 ± 13.3  | 18.9 ± 4.4  | 7.4 ± 8.2   | 85.4 ± 6.4  |
| III           | 31.5 ± 27.5 | 33.8 ± 20.1  | 18.6 ± 6.9  | 5.0 ± 5.7   | 82.4 ± 8.1  |
| IV            | 28.6 ± 26.2 | 30.2 ± 17.9  | 19.1 ± 6.2  | 5.8 ± 8.0   | 82.8 ± 5.5  |

Spearman’s ρ | P-value | Spearman’s ρ | P-value

**Without nasal obstruction**

| I             | 26.9 ± 31.3 | 34.8 ± 23.6  | 20.2 ± 2.9  | 1.9 ± 2.9   | 83.0 ± 9.4  |
| II            | 28.7 ± 25.2 | 31.1 ± 17.4  | 19.9 ± 6.6  | 5.4 ± 7.5   | 80.8 ± 10.8 |
| III           | 25.6 ± 19.7 | 29.0 ± 15.7  | 19.4 ± 6.0  | 4.1 ± 4.9   | 82.6 ± 7.5  |
| IV            | 26.5 ± 23.7 | 29.9 ± 16.2  | 18.1 ± 5.0  | 5.0 ± 5.9   | 81.7 ± 8.6  |

Spearman’s ρ | P-value | Spearman’s ρ | P-value

Variable are presented as mean ± SD.
AHI, apnea-hypopnea index; REM, rapid eye movement.

*Analysis using partial spearman correlation with adjustment for age, sex, tonsillar grade, and presence of nasal obstruction. †Analysis using partial spearman correlation with adjustment for age, sex, and tonsillar grade.
sleep time/total sleep time×100) and the lowest $\text{SaO}_2$ for the total subjects, although there was some tendency for a correlation between the MMS and the total AHI ($P=0.058$). Analysis using partial Spearman correlation controlling for age, sex, tonsillar grade, and presence of nasal obstruction also revealed similar results (Table 2).

However, significant correlations were found between many of the PSG variables and the MMS when the subjects were divided according to the presence of nasal obstruction. In the patient group with nasal obstruction, a significant association was found between the MMS and sleep disordered breathing. The total AHI and arousal index became higher as the MMS was increased (Table 2). Sleep quality was also influenced by the MMS in the patients with nasal obstruction. Deep sleep showed a significant decrease according to the increase of the MMS. The lowest $\text{SaO}_2$ showed a decreasing tendency as the MMS increased even though it didn’t reach statistical significance. Analysis using partial Spearman correlation controlling for age, sex, and tonsillar grade also revealed similar results (Table 2).

There was little significant correlation found for the patients without nasal obstruction. Only the REM sleep% showed significant correlation with the MMS (Table 2). However, after controlling for possible covariates including age, sex, and tonsillar grade, the REM sleep% didn’t show significant correlation with the MMS any more.

**DISCUSSION**

The present study showed that the MMS might be associated with SDB and the sleep quality in patients with nasal obstruction. An elevated AHI and arousal index and a decreased % of deep sleep were noted in the patients with higher MMS and nasal obstruction even though the association is low (Spearman’s $\rho$ for AHI = 0.210). These results were confirmed with partial Spearman correlation analysis controlling for possible covariates.

Nasal breathing is the preferred route of breathing when we are awake and sleep, and the nasal airway is responsible for ap-
cal studies have shown that allergic rhinitis affects 9%-42% of the general population [24]. Therefore, we think the subjective method might have merits for evaluating nasal obstruction in these patients.

The present study showed the MMS is well correlated with the severity of SDB as well as the sleep quality, especially in patients with nasal obstruction. The MMS can give more valuable information about the severity of SDB to the sleep physician when combined with simple questions about nasal obstruction.

CONFLICT OF INTEREST
No potential conflict of interest relevant to this article was reported.

ACKNOWLEDGMENTS
We are grateful to Sook-young Woo in Biostatistics team for her expert consultation with statistical analysis.

REFERENCES
1. Kim HY, Bok KH, Dhong HJ, Chung SK. The correlation between pharyngeal narrowing and the severity of sleep-disordered breathing. Otolaryngol Head Neck Surg. 2008 Mar;138(3):289-93.
2. Lee SH, Choi JH, Shin C, Lee HM, Kwon SY, Lee SH. How does open-mouth breathing influence upper airway anatomy? Laryngoscope. 2007 Jan;117(6):1102-6.
3. Friedman M, Tanyeri H, La Rosa M, Landsberg R, Vaidyanathan K, Pieri S, et al. Clinical predictors of obstructive sleep apnea. Laryngoscope. 1999 Dec;109(12):1901-7.
4. Friedman M, Ibrahim H, Joseph NJ. Staging of obstructive sleep apnea/hypopnea syndrome: a guide to appropriate treatment. Laryngoscope. 2004 Mar;114(3):454-9.
5. Kim HY, Min JY, Cho DY, Chung SK, Dhong HJ. Influence of upper airway narrowing on the effective continuous positive airway pressure level. Laryngoscope. 2007 Jan;117(1):82-5.
6. EEG arousals: scoring rules and examples: a preliminary report from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. Sleep. 1992 Apr;15(2):173-84.
7. Ferris BG Jr, Mead J, Optie LH. Partitioning of respiratory flow resistance in man. J Appl Physiol. 1964 Jul;19:653-8.
8. Armengot M, Henríquez R, Miguel P, Navarro R, Basterra J. Effect of total nasal obstruction on nocturnal oxygen saturation. Am J Respir Crit Care Med. 2008 May;177(10):1325-8.
9. Miljeteig H, Hoffstein V, Cole P. The effect of unilateral and bilateral nasal obstruction on snoring and sleep apnea. Laryngoscope. 1992 Oct;102(10):1150-2.
10. Miljeteig H, Savard P, Mateika S, Cole P, Haight JS, Hoffstein V. Snoring and nasal resistance during sleep. Laryngoscope. 1993 Aug;103(8):918-23.
11. DeVito A, Berrettini S, Carabelli A, Sellari-Franceschini S, Bonanni E, Gori S, et al. The importance of nasal resistance in obstructive sleep apnea syndrome: a study with positional rhinomanometry. Sleep Breath. 2001 Jan;5(1):3-11.
12. Lofaso F, Coste A, d’Ortho MP, Zerah-Lancner F, Delclaux C, Goldberg F, et al. Nasal obstruction as a risk factor for sleep apnoea syndrome. Eur Respir J. 2000 Oct;16(4):639-43.
13. Craig TJ, Mende C, Hughes K, Kaku-munna S, Lehman EB, Chinchielli V. The effect of topical nasal fluticasone on objective sleep testing and the symptoms of rhinitis, sleep, and daytime somnolence in perennial allergic rhinitis. Allergy Asthma Proc. 2003 Jan-Feb;24(1):53-8.
14. Craig TJ, Teets S, Lehman EB, Chinchielli VM, Zwillich C. Nasal congestion secondary to allergic rhinitis as a cause of sleep disturbance and daytime fatigue and the response to topical nasal corticosteroids. J Allergy Clin Immunol. 1998 May;101(3):633-7.
15. Hughes K, Glass C, Ricipichini M, Gurevich F, Weaver TE, Lehman E, et al. Efficiency of the topical nasal steroid budesonide on improving sleep and daytime somnolence in patients with perennial allergic rhinitis. Allergy. 2003 May;58(5):380-5.
16. Kiey JL, Nolan P, McNicholas WT. Intranasal corticosteroid therapy for obstructive sleep apnoea in patients with co-existing rhinitis. Thorax. 2004 Jan;59(1):50-5.
17. Rombaux P, Liistro G, Hamoir M, Bertrand B, Aubert G, Verses T, et al. Nasal obstruction and its impact on sleep-related breathing disorders. Rhinology. 2005 Dec;43(4):242-50.
18. Vural C, Gungor A. The effect of topical fluticasone on nasal nitric oxide levels in a patient with allergic rhinitis. Ear Nose Throat J. 2003 Aug;82(8):592-7.
19. Series S, St Pierre S, Carrier G. Surgical correction of nasal obstruction in the treatment of mild sleep apnoea: importance of cephalometry in predicting outcome. Thorax. 1993 Apr;48(4):360-3.
20. Fitzpatrick MF, McLean H, Urton AM, Tan A, O’Donnell D, Driver HS. Effect of nasal or oral breathing route on upper airway resistance during sleep. Eur Respir J. 2003 Nov;22(5):827-32.
21. Koutsourelakis I, Georgoulakou C, Pieri S, et al. Obstructive apneas during sleep in patients with seasonal allergic rhinitis. Sleep Res Rev. 2008 Jul;1365-73.
22. Park SS. Flow-regulatory function of upper airway during sleep and daytime somnolence in patients with sleep-disordered breathing. Sleep. 2004 Mar;27(3):521-7.
23. Park SS. Flow-regulatory function of upper airway in health and disease: a unified pathogenetic view of sleep-disordered breathing. Lung. 1993 Nov;171(6):311-33.
24. Georgalas C. The role of the nose in snoring and obstructive sleep apnoea: an update. Eur Arch Otorhinolaryngol. 2011 Sep;268(9):1365-73.
25. Mirza N, Lanza DC. The nasal airway and obstructed breathing during sleep. Otolaryngol Clin North Am. 1999 Apr;32(2):243-62.
26. Andre RF, Vuyk HD, Ahmed A, Graamans K, Nolst Trenite GJ. Correlation between subjective and objective evaluation of the nasal airway: a systematic review of the highest level of evidence. Clin Otolaryngol. 2009 Dec;34(6):518-25.
27. Kim CS, Moon BK, Jung DH, Min YG. Correlation between nasal obstruction symptoms and objective parameters of acoustic rhinometry. Auris Nasus Larynx. 1998 Jan;25(1):45-8.
28. McNicholas WT. The nose and OSA: variable nasal obstruction may be a risk factor for obstructive sleep apnoea. Eur Respir J. 2000 Nov;16(5):1365-73.
29. McNicholas WT, Tarlo S, Cole P, Zamel N, Rutherford R, Griffin D, et al. Obstructive apneas during sleep in patients with seasonal allergic rhinitis. Am Rev Respir Dis. 1982 Oct;126(4):625-8.