An Investigation of Upper Airway Changes Associated With Mandibular Advancement Device Using Sleep Videofluoroscopy in Patients With Obstructive Sleep Apnea

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Objective: To quantitatively evaluate the effects of the mandibular advancement device (MAD) on changes in the upper respiratory tract during sleep using sleep videofluoroscopy (SVF) in patients with obstructive sleep apnea (OSA).

Design: Retrospective analysis.

Setting: Academic tertiary referral center.

Patients: Seventy-six patients (68 men and 8 women) who were treated with the MAD for OSA were included from September 1, 2005, through August 31, 2008.

Intervention: All patients underwent nocturnal polysomnography and SVF before and at least 3 months after receipt of the custom-made MAD. Sleep videofluoroscopy was performed before and after sleep induction and was analyzed during 3 states of awakeness, normoxegenation sleep, and desaturation sleep.

Main Outcome Measures: Changes in the length of the soft palate, retropalatal space, retrolingual space, and angle of mouth opening were evaluated during sleep events with or without the MAD.

Results: Without the MAD, the length of the soft palate and the angle of mouth opening increased during sleep events, especially in desaturation sleep, compared with the awake state. The retropalatal space and retrolingual space became much narrower during sleep compared with the awake state. The MAD had marked effects on the length of the soft palate, retropalatal space, retrolingual space, and angle of mouth opening. The retropalatal and retrolingual spaces were widened, and the length of the soft palate was decreased. The MAD kept the mouth closed.

Conclusions: Sleep videofluoroscopy showed dynamic upper airway changes in patients with OSA, and the MAD exerted multiple effects on the size and configuration of the airway. Sleep videofluoroscopy demonstrated the mechanism of action of the MAD in patients with OSA. The MAD increased the retropalatal and retrolingual spaces and decreased the length of the soft palate and the angle of mouth opening, resulting in improvement of OSA.

Arch Otolaryngol Head Neck Surg. 2009;135(9):910-914
Eighty-six patients (68 male, with a mean [SD] age of 51.7 [10.3] years [age range, 21-69 years]) who visited the Sleep Center at Seoul National University Bundang Hospital, Seongnam, Korea, from September 1, 2005, through August 31, 2008, were retrospectively included in this study. All patients underwent full-night nocturnal polysomnography and were diagnosed as having OSA. They were referred to a single dentist (P.-Y.Y.), and a custom-made MAD was fabricated for each patient. The MAD was designed as a monobloc that holds the mandible fixed at 60% of maximal protrusion without open bites. Patients underwent a second full-night nocturnal polysomnography at least 3 months after receipt of the custom-made MAD. The mean (SD) apnea-hypopnea index of patients was 38.9 (19.7) without the MAD and 12.3 (11.4) with the MAD. (Patients with an apnea-hypopnea index of \( \geq 5 \) are considered to have OSA: 5-20 indicates mild OSA; 21-40, moderate OSA; and \( \geq 41 \), severe OSA.) Their mean (SD) body mass index (calculated as weight in kilograms divided by height in meters squared) was 25.6 (2.6), and the lowest mean (SD) oxygen saturation was 79.2% (7.8%). This study was approved by the Institutional Review Board of Seoul National University Bundang Hospital.

SLEEP VIDEOFLUOROSCOPY

All patients underwent SVF with or without the MAD as previously described. In brief, patients were in the supine position on a C-arm table with their head on a pillow. They were instructed to breathe in and out naturally. Oxygen saturation was monitored throughout the examination. During normal respiration before sedation, an awake event was recorded for 15 seconds. Thereafter, sleep was induced by intravenous administration of midazolam (2 mg). After the patient fell asleep, the sleep event examinations started. Although oxygen saturation does not decrease, a 15-second respiratory state was recorded as a normoxegenation sleep event. When oxygen saturation dropped by 4% or more, two 15-second desaturation sleep events were recorded.

EVALUATION OF SVF VARIABLES

The length of the soft palate, retropalatal space, retrolingual space, and angle of mouth opening were measured and evaluated during 3 different states (awakeness, normoxegenation sleep, and desaturation sleep). The length of the soft palate was defined as the distance from the posteronasal spine to the uvula tip, the retropalatal space as the narrowest posterior airway space at the level of the soft palate, the retrolingual space as the narrowest posterior airway space at the level of the tongue base, and the angle of mouth opening as the angle formed by the intersection of lines drawn from the maxillary incisor to the glenoid fossa and from the glenoid fossa to the mandibular incisor (Figure 1).

STATISTICAL ANALYSIS

The t test was used to analyze differences among awakeness and sleep events. Paired t test was used to analyze differences between variables with or without the MAD. All results were expressed as the mean (SD). Statistical significance was assumed at \( P < .05 \) for all variables.

RESULTS

EFFECTS ON THE LENGTH OF THE SOFT PALATE

When the length of the soft palate was compared among events without the MAD, it was longer during sleep events than during awakeness. It was longest in desaturation
sleep, although no significant difference was noted between the 2 sleep events. The MAD decreased the length of the soft palate from 46.5 (6.3) mm to 42.0 (5.7) mm in normoxygenation sleep and from 48.3 (6.3) mm to 43.2 (6.2) mm in desaturation sleep (P < .001 for both) (Table). The MAD had no significant effect on the length of the soft palate in awake events (P = .06).

**EFFECTS ON THE RETROPALATAL SPACE**

Sleep videofluoroscopy performed without the MAD showed drastic changes in the retropalatal space associated with different sleep events. The retropalatal space in awake events was 4.7 (1.4) mm and decreased to 2.1 (1.5) mm during normoxygenation sleep and to 1.2 (1.4) mm during desaturation sleep, demonstrating narrowing of the upper airway during sleep in patients with OSA (P < .001) (Table). The MAD increased the retropalatal space significantly during different sleep events (P < .001 for both). It increased from 4.7 (1.4) mm to 5.5 (2.0) mm in awake events, from 2.1 (1.5) mm to 3.2 (2.3) mm in normoxygenation sleep, and from 1.2 (1.4) mm to 2.6 (2.7) mm in desaturation sleep.

**EFFECTS ON THE RETROLINGUAL SPACE**

The retrolingual space during SVF without the MAD also showed dramatic changes during different sleep events. It was 10.2 (2.4) mm in awake events and decreased to 5.1 (3.7) mm in desaturation sleep (Table). With the MAD, the retrolingual space widened significantly in awake and sleep events (P < .001). The MAD increased the retrolingual space from 10.2 (2.4) mm to 11.8 (2.8) mm in awake events, from 7.5 (3.4) mm to 10.1 (4.0) mm in normoxygenation sleep, and from 5.1 (3.7) mm to 10.0 (4.1) mm in desaturation sleep. However, the retrolingual space with the MAD did not change significantly during different sleep events (P > .05), showing relative constant preservation of the retrolingual space.

**EFFECTS ON THE ANGLE OF MOUTH OPENING**

In awake events, the angle of mouth opening was 0.58° (1.19°) without the MAD, showing that the mouth was almost closed during awakeness (Table). During sleep, it increased to 3.77° (1.79°) in normoxygenation sleep and to 4.94° (2.00°) in desaturation sleep. With the MAD, the angle of mouth openings were 1.22° (0.73°) during awakeness, 1.42° (0.96°) in normoxygenation sleep, and 1.56° (1.28°) in desaturation sleep. The angle of mouth opening did not differ significantly with various awake or sleep events, indicating that the MAD prevented mouth opening during sleep and maintained a constant angle of opening (Figure 2).

**COMMENT**

In this study, SVF showed dynamic upper airway changes during different sleep events and demonstrated the mechanism of action of the MAD on dynamics of the upper airway in patients with OSA. The upper airway size changed dramatically during sleep, especially in desaturation sleep, compared with awakeness. During desaturation sleep, the length of the soft palate increased, the retropalatal and retrolingual spaces narrowed, and the angle of mouth opening increased in patients with OSA. The MAD had an important role in the dynamics of the upper airway and counteracted the aforementioned changes during sleep. In other words, it decreased the length of the soft palate,
widened the retropalatal and retrolingual spaces, and decreased the angle of mouth opening during sleep.

The mechanism of action of the MAD on the upper airway size has been studied using various methods, including cephalometry, computed tomography, and magnetic resonance imaging, and results vary slightly among studies. The velopharynx (retropalatal space) or the hypopharynx was claimed to be widened in those studies. Variable findings may be attributed to different study methods, body position, or sleep status among patients.

Although several studies have described the effects of the MAD on upper airway changes, most have limitations in that the data were obtained while patients were awake, or the studies were based on static images of patients in the supine position. To overcome these limitations, we used SVF to evaluate the mechanism of action of the MAD. Sleep videofluoroscopy has several advantages. First, it provides dynamic images while patients are asleep in the supine position for a short period. Therefore, it can be used to detect dynamic airway changes during sleep. Second, it easily shows the mechanism of action of the anatomical structures outside of the pharyngeal airway. For instance, movements of the cervical spine, mouth opening, tongue, hyoid bone, and jaw can be directly observed. Third, SVF detects upper airway changes not only in awake events but also in sleep events (normoxegenation sleep and desaturation sleep). Because desaturation sleep corresponds to the period of sleep apnea when the upper airway changes most severely and dynamically, analysis of desaturation sleep events can elucidate the exact mechanism of action of the MAD during apneic periods, which cannot be evaluated by other methods.

Indeed, our results provide useful information about the mechanism of action of the MAD. In addition to preventing mouth opening and widening the retropalatal and retrolingual spaces, the MAD decreases the length of the soft palate. In general, the MAD has been thought to be mainly effective in widening the retropalatal and retrolingual spaces. However, the present study shows that the MAD applies tension to the soft palate, preventing collapse of the retropalatal space.

This study has some limitations, which are the same as those discussed in a previous study. Briefly, SVF is a superimposed 2-dimensional image of 3-dimensional structures. Therefore, it cannot explain lateral movement of the upper airway. In addition, full-night sleep was not included for SVF, and sleep was induced by drug administration, although another study proved the validity of drug-induced sleep as representative of normal sleep. Despite those limitations, our study provides novel information about the mechanism of action of the MAD on the upper airway in patients with OSA.

In conclusion, the upper airway changes dynamically during awake and sleep events. Sleep videofluoroscopy showed dynamic upper airway changes in patients with OSA, and the MAD exerted multiple effects on the size and configuration of the upper airway. The mechanism of action of the MAD in patients with OSA includes widening the retrolingual space, decreasing the length of the soft palate, and narrowing the angle of mouth opening.

Submitted for Publication: February 16, 2009; final revision received April 10, 2009; accepted April 23, 2009.

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Financial Disclosure: None reported.

Funding/Support: This study was supported by grant 11-2008-011 from the Seoul National University Bundang Hospital Research Fund.

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