CASE REPORT

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Cessation of Snoring Without Apnea-Hypopnea Improvement During Oral Appliance Therapy for Obstructive Sleep Apnea

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We encountered the case of a 41-year-old male patient with mild obstructive sleep apnea (OSA) treated with a mandibular advancement oral appliance (OA_m) that highlights the fact that cessation of snoring is not always associated with an improvement of the apnea-hypopnea index (AHI). Follow-up polysomnography revealed that snoring improved from 14.8% to 0.4% of total sleep time, whereas AHI slightly increased from 8.5 to 11.6/h and the nadir percutaneous oxygen level fell from 93% to 87%. This case emphasizes the following three points: (1) cessation of snoring does not always indicate a reduction in AHI during OA_m therapy for OSA; (2) objective follow-up evaluation of the efficacy of OA_m treatment using sleep testing is necessary to avoid suboptimal outcomes; and (3) a follow-up sleep evaluation provides dentists and physicians with proper information for discussing not only treatment success but also the modification and/or replanning of OA_m treatment.

Keywords: follow-up polysomnography; obstructive sleep apnea; oral appliance; snoring

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INTRODUCTION

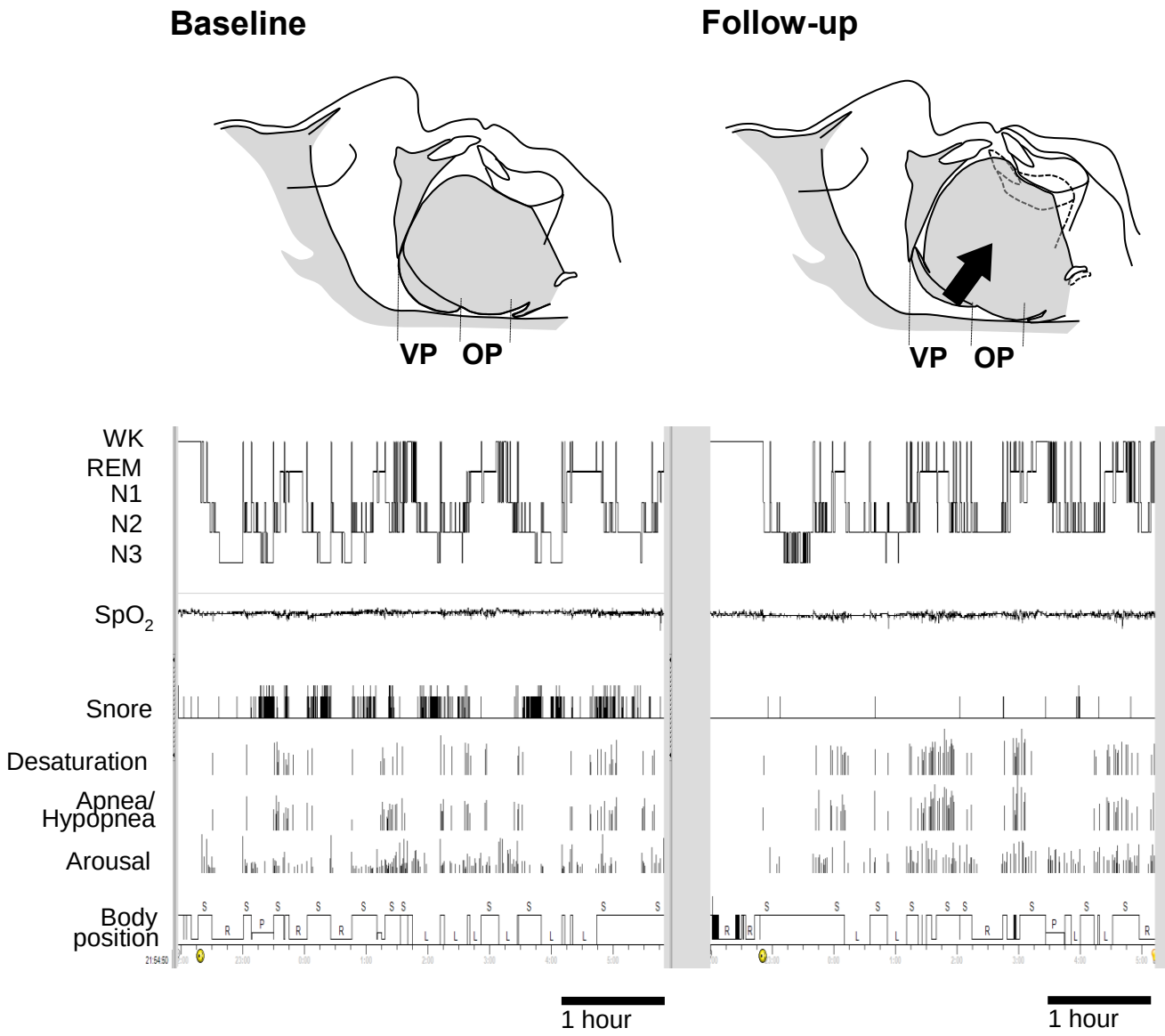
Snoring is a typical chief complaint in patients with obstructive sleep apnea (OSA). The cessation of snoring after the prescription of a mandibular advancement oral appliance (OA_m) can often be assumed to reflect a patent upper airway following mandibular advancement, which would simultaneously support the effectiveness of the OA_m. However, we recently experienced a case of mild OSA in which snoring disappeared with the use of an OA_m, with little change in the apnea-hypopnea index (AHI). This case highlights the necessity of objective follow-up to evaluate the efficacy of OA_m treatment.[1]

REPORT OF CASE

A 41-year-old male patient visited the Yoyogi Sleep Disorder Center, Tokyo, Japan. He complained of mild insomnia and shallow sleep, and his wife reported loud snoring with episodes of apnea. The patient did not complain of dozing off during work. Mild OSA was diagnosed in this patient based on a nocturnal polysomnographic study (AHI = 8.5/h), and the patient was referred to the Sleep Apnea Dental Clinic in the same center for treatment with an OA_m[2] (Table 1). A custom-made monobloc OA_m was fabricated at 50% of the maximum mandibular protrusion (3 mm advancement). After 3 months of acclimatization to the OA_m and treatment with additional advancement of the mandible (1.5 mm) to stop his snoring, the patient used the OA_m 6 to 7 nights a week. Consequently, the final amount of mandibular advancement was 4.5 mm. Although the patient's body weight increased by 2.6 kg, his wife no longer reported that he was snoring. When we proposed that the patient undergo follow-up sleep testing with the OA_m in place, he was initially opposed; he thought that he had not made a remarkable subjective improvement in the quality of his sleep.

A follow-up polysomnographic evaluation revealed that OA_m treatment was effective with respect to snoring; the patient's snoring had indeed improved from 67.4 to 1.6 minutes (from 14.8% to 0.4% of the total sleep time) (Figure 1). Nevertheless, there was no significant reduction in apneic and hypopneic episodes. The apnea index changed from 0.3 to 0.7/h and the hypopnea index slightly increased from 8.2 to 10.6/h. Consequently, the AHI increased from 8.5 to 11.4/h, whereas the nadir percutaneous oxygen (SpO_2) fell from 93% to 87%. There was no regular intake of hypnotics nor alcohol throughout the treatment period or during the polysomnographic studies.

Figure 1



All-night hypnogram consisting of sleep stages, body position, presence of apneas and hypopneas, SpO_2 desaturation, and snoring at baseline (without oral appliance) and at follow-up (with oral appliance).

A microphone was used for monitoring snoring in accordance with the American Academy of Sleep Medicine manual.[2] Schematic illustrations show changes in the upper airway following the application of an oral appliance in patients with OSA are also shown. Part of this illustration was based on results from Tsuiki et al..[4] The arrow (top right) indicates the possible direction of the change in upper airway soft tissue caused by mandibular advancement in this patient. Note that the numerous snoring events at baseline almost disappeared at follow-up, whereas there was no positive change in apneas or hypopneas. VP = velopharynx, OP = oropharynx.

Table 1

Effects of an oral appliance on respiratory and sleep variables.

	Baseline	Follow-up
Height (cm)	175.0	175.0
Weight (kg)	68.1	70.7
Body mass index (kg/m^2)	22.2	23.1
Epworth Sleepiness Scale	7	10
TST (min)	423.6	333.0
Stage REM %TST	21.0	20.0
Stage N1 %TST	17.9	17.1
Stage N2 %TST	44.1	58.9
Stage N3 %TST	17.0	4.1
Snoring (min)	67.4	1.6
Snoring %TST	14.8	0.4
Apnea Index /h	0.3	0.7
Hypopnea Index /h	8.2	10.6
AHI /h	8.5	11.4
Supine AHI /h	13.7	13.5
Nonsupine AHI /h	6.2	9.3
Arousal Index /h	23.7	20.5
Respiratory arousal index /h	5.0	4.7
Nadir SaO_2 (%)	93.0	87.0
SpO_2 90%> min	0.1	0.2

AHI = apnea-hypopnea index, SaO_2 = arterial oxygen saturation, REM = rapid eye movement, TST = total sleep time.

DISCUSSION

The current case is probably typical of cases that are often encountered by sleep dentists and physicians. Users of an OA_m sometimes do not consent to follow-up sleep testing if, for example, the cessation of snoring is witnessed by a bed partner and/or the resolution of subjective OSA symptoms is confirmed. However, the recent clinical practice guidelines from the American Academy of Sleep Medicine suggest that "sleep physicians conduct follow-up sleep testing to improve or confirm the treatment efficacy, rather than conduct follow-up without sleep testing, for patients" fitted with an OA_m.[1] Physicians should also consider follow-up testing for OA_m users "who develop recurrent symptoms, show substantial weight changes, or receive diagnoses of comorbidities relevant to OSA".[1] Although barriers to polysomnographic sleep studies, including cost, waiting time, accessibility to the sleep clinic, and disease severity, have always been argued, the follow-up sleep study in this patient played a role in the avoidance of suboptimal treatment by further adjustment of OA_m while encouraging weight control by explaining for an interaction between consumption of nighttime snack and likely increase in body weight.[3]

The OA_m predominantly enlarges the velopharynx, which is both where snoring originates and the primary site of upper airway occlusion.[4,5] However, it is clinically crucial to speculate why apnea and hypopnea remained despite the cessation of snoring. First, the favorable effects on the velopharynx due to mandibular advancement were maintained with OA_m because the ventral displacement of the tongue associated with advancement of the mandible could stiffen the velopharynx through the palatoglossal arch[6] more than the oropharynx because snoring was well controlled throughout the follow-up sleep study. In addition, the sleep-related atonia of the genioglossus muscle that maintains the tongue in position may offset the effect of mandibular protrusion on the oropharynx.[7] Second, fat deposits in the tongue *per se* (a heavier tongue weight), despite representing only a slight change, may be a factor in increasing the vulnerability of the oropharyngeal space, because body weight was the only parameter that changed during the treatment period (2.6 kg). These speculations can be more easily tested without laborious imaging and/or endoscopic approaches by further refinements of a method for determining the site of upper-airway occlusion by focusing solely on the shape of inspiratory flow limitations.[8]Finally, the reduction in stage N3 sleep along with an increase in stage N2 sleep as shown in Table 1 would result in the absence of a remarkable subjective improvement in the quality of patient's sleep, although it should be investigated in additional studies.

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DISCLOSURE STATEMENT

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