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An Evaluation of a Titration Strategy for Prescription of Oral Appliances for Obstructive Sleep Apnea*

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Background: **Oral appliances (OAs) are first-line therapy for mild-to-moderate obstructive sleep apnea (OSA) and are being used with increasing frequency. Additionally, best practice of OA titration is unknown. We describe the experience of patients treated with an OA, identify factors that predict treatment success with an OA, and offer a protocol for OA titration.**

Methods: **We retrospectively studied patients seen in a dental sleep clinic between 2002 and 2006. Patients selected for OA treatment underwent baseline polysomnography, were individually fit with an OA, and were instructed to titrate it at home until symptom resolution or discomfort. During follow-up polysomnography, additional titration was performed as needed. Primary outcome was successful treatment, defined as apnea-hypopnea index (AHI) < 10 events per hour and AHI decrease at least 50% from baseline. Logistic regression models were created to identify associations between patient characteristics and successful treatment. Overall differences in AHI at baseline, after home titration, and after final titration were compared using Kruskal-Wallis test, and *post hoc* comparisons were performed with sign tests, with Bonferroni corrections.**

Results: **Of 57 subjects treated with an OA, 37 subjects (64.9%) were successfully treated with OA therapy. Of the 49 subjects for whom data were available for AHI after home titration, 27 subjects (55%) achieved successful treatment of OSA by self-titration, without need for further titration during follow-up polysomnography.**

Conclusions: **A majority of subjects, regardless of OSA severity, are successfully treated with an OA. Men and younger patients were found to be the best responders. The titration protocol for an OA offers a beneficial initial step in the treatment of OSA.**

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Key words: mandibular advancement devices; obstructive sleep apnea; oral appliance therapy

Abbreviations: AHI = apnea-hypopnea index; BMI = body mass index; OA = oral appliance; OR = odds ratio; OSA = obstructive sleep apnea

The role of oral appliances (OAs) in the treatment of patients with obstructive sleep apnea (OSA) has gained prominence since their introduction in 1982.[1] Recently, the American Academy of Sleep Medicine published practice parameters for the treatment of snoring and OSA with OA, establishing a first-line role for OA in the treatment of patients with mild-to-moderate OSA, and second-line role for patients with severe OSA.[2] While these practice parameters recommend that an OA should be prescribed by experienced dental personnel knowledgeable in sleep medicine and/or sleep-related breathing disorders, relatively few dentists have the necessary training to accurately identify OSA or treat the entity with an OA. In a survey of dental practitioners in one area, 40% know little or nothing about OA therapy for OSA.[3] Additionally, the best practice for titration of mandibular position of OA devices has not been adequately studied. The efficacy of patient self-titration and timing of a repeat polysomnography for OA titration are factors in OA therapy that remain uncertain. We sought to report the experi-

ence of one sleep-trained dentist in a large metropolitan area, and identify patient demographic and disease characteristics among OSA patients treated with OA therapy that may predict success of therapy.

Materials and Methods

A retrospective study was performed to compare two groups. Data were provided by the clinical provider as de-identified information for the purpose of research, and this project was deemed exempt from continuing review by the Johns Hopkins Institutional Review Board. Patients referred to a dental sleep clinic in the greater Baltimore area and treated with OA therapy for the spectrum of OSA syndromes, from snoring to OSA, between 2002 and 2006 were eligible for the study if they had undergone baseline diagnostic polysomnography and, if treated with an OA, follow-up titration polysomnography. Subjects without baseline age, gender, or body mass index (BMI) data were excluded. The comparison group entailed consecutive patients who were referred to the same dental sleep clinic for treatment of an OSA disorder but were not treated with OA therapy.

The titration protocol is depicted in Figure 1. Subjects selected for OA treatment were individually fitted for an OA. Choice of OA was made by the sleep-trained dentist (S.S.), and factors that determined OA choice included anatomy, dental health, temporomandibular stability, gag reflex sensitivity, oral habits, reported sleep position, nasal patency, and patient preference. Examples of reasons for not prescribing OA for patients referred for OSA treatment include too few healthy teeth and/or dental implants, and active temporomandibular joint capsulitis. Patients were counseled on the lower treatment success rates associated with a long lower facial profile, excessive neck circumference (men $>$ 20 inches, women $>$ 17 inches), and obesity, but these were not contraindications to OA prescription. Patients were instructed to self-titrate the mandibular position of the OA until resolution of sleep symptoms (*eg*, snoring, nocturnal awakenings, unrefreshing sleep) or limitations due to discomfort.

Once the patient accommodated to the OA at home and it was optimally adjusted relative to symptoms, the patient was referred back to the sleep laboratory for a follow-up overnight polysomnography while wearing the OA. Titration of the OA was done as follows: after sleep onset, if the obstructive apneas and/or hypopneas observed at a rate greater or equal than one event per 20 epochs, then the patient was awakened and instructed to advance the mandible 1 mm by rotating the OA adjustment mechanism. If pain or discomfort was experienced, the patient was instructed to retract the mandible by adjusting the OA as necessary. Titration for nonapneic snoring was performed only if specifically requested by the patient or physician. Twenty minutes of sleep after a titration or sleep position change, and if not in rapid eye movement sleep, the titration process was repeated as necessary. Subsequent iterations of the titration entailed advancement of the OA by 1-mm increments, or less if not tolerated. Mandibular position was recorded as millimeters of mandibular advancement, regardless of type of OA used (reported as posture change). The optimal mandibular position of the OA was determined after the polysomnography was reviewed by the reviewing sleep physician and sleep-trained dentist. Pretreatment apnea-hypopnea index (AHI) was determined from baseline polysomnography. Home titration AHI was determined by the AHI during the first interval of sleep during follow-up titration polysomnography. Final treatment AHI was determined by the AHI of the final titration interval of follow-up titration polysomnography.

The main outcome variable was success of OA therapy, defined as posttreatment AHI $<$ 10 events per hour and reduction of AHI by at least 50%. Secondary outcome measures included success of OA therapy with home titration only, posttreatment AHI, and subjective symptoms. Other data available for analysis included age, gender, body anthropomorphic measurements (BMI, neck circumference, canine classification, facial skeletal classification), baseline sleep characteristics (AHI, minimum low nocturnal oxyhemoglobin saturation), and intervention (type of OA, length of mandibular advancement at home and during polysomnography). All variables were modeled as continuous variables, except for the following categorical variables: success of therapy, gender, symptoms (resolved, improved, unimproved, worsened), canine classification (I: the lower canine is positioned ½ tooth forward of the upper canine; II D1: the lower canine is positioned posterior to the class I position; II D2: the lower canine is positioned posterior to the class I position with deep anterior overbite; III: the lower canine is positioned anterior to the class I position), facial skeletal classification (I, normal; II, relative retrognathia; III, relative mandibular protrusion); type of OA (Thornton adjustable positioner 1 and 2; Airway Labs; Dallas, TX; Herbst, Klearway, Snore-Aid Plus; Great Lakes Orthodontics; Tonawanda, NY; and Elastic Mandibular Advancement; Johns Dental Laboratories; Terre Haute, IN), and subjective symptoms (resolved, improved, unchanged, worsened). Overbite is a measurement of vertical overlap between the upper and lower central incisor teeth when the mouth is closed. Overjet is a measurement of anteroposterior horizontal overlap between the central incisor edges when the mouth is closed. Apneas and hypopneas were defined by Medicare criteria, with some laboratories also reporting hypopneas with arousals independently, although these were not included in the analysis.[4]

Observations across individual subjects were assumed to be independent of each other. Baseline characteristics of patients treated and not treated with OA were compared using Pearson χ^2 tests or Mann Whitney *U* tests,[5] as appropriate. In bivariate analyses, we employed simple logistic regression models. Analyses of success of OA therapy by degree of baseline OSA were stratified into categories of mild ($\leq$ 20 events/h), moderate (21 to 40 events/h), and severe ($>$ 40 events/h) OSA. To determine the significance of multiple independent characteristics on the success of OA therapy, multivariable logistic regression models were developed, and the appropriateness of these models were evaluated using Hosmer-Lemeshow goodness-of-fit tests.[6] In the subset of subjects in whom interval data from the titration polysomnography study was available, Kruskal-Wallis one-way analysis of variance[7] was used to test the equality of medians between baseline AHI, AHI after home titration, and AHI after laboratory titration. For globally significant p value, *post hoc*

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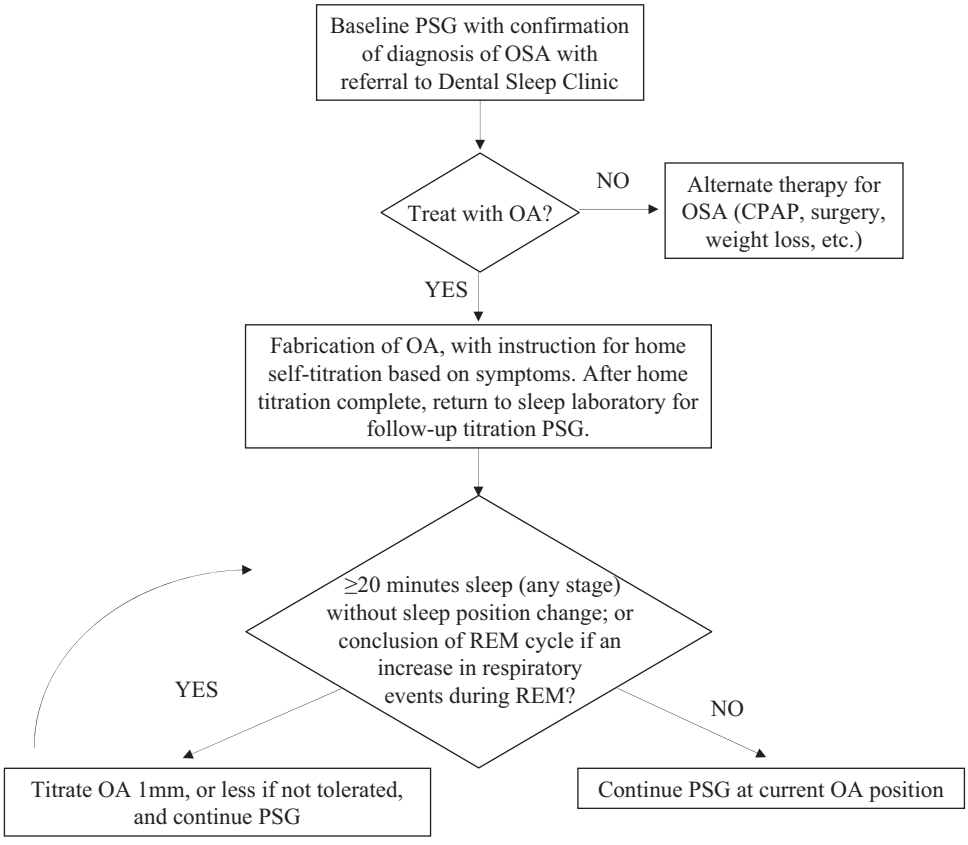

FIGURE 1. Flowchart for OA titration protocol. PSG = polysomnography; CPAP = continuous positive airway pressure.

testing for pairwise comparisons was performed using a one-way sign test, and the Bonferroni correction[8] was used to account for multiple comparisons.

A two-tailed p value ≤ 0.05 was used to detect statistically significant differences. Analyses and graph generation were performed using software (STATA version 9.0; StataCorp; College Station, TX; and Microsoft Excel 2000; Microsoft Corporation; Seattle, WA).

RESULTS

Baseline Characteristics and Outcomes

A total of 57 subjects were identified who were treated with OA and for whom there were pretreatment and posttreatment polysomnogram data, and all were included in the analysis. Data for the first 79

Table 1—*Demographics of Subjects

Variables	Treated (n = 57)	Not Treated (n = 68)	p Value
Age, yr	56.0 (48.0–60.0)	54.0 (48.0–64.5)	0.83
Male gender	38 (55.9)	38 (66.7)	0.28
BMI, kg/m^2	27.4 (24.3–29.8)	28.9 (26.6–33.5)	0.01
Neck circumference, cm	16.0 (15.0–16.5)	16.0 (15.0–17.0)	0.64
Pretreatment overbite, mm	2.8 (2.0–4.5)		N/A
Pretreatment overjet, mm	5.0 (3.0–5.0)		N/A
Pretreatment AHI, events/h†	24.8 (19.5–39.2)	27.8 (14.8–43.2)	0.66
Pretreatment oxyhemoglobin saturation, %	83.0 (78.0–87.5)	85.0 (77.0–89.0)	0.32
Time from insertion to titration polysomnography, d	111 (87–183)		N/A
Home posture change, mm‡	4.9 (3.0–6.0)		N/A
Total posture change, mm‡	6.0 (4.2–7.0)		N/A

*Data are presented as median (range) or No. (%). N/A = not applicable.
†Minimum AHI among treated group is 5.9.
‡Data available for 20 patients treated with OA only.

subjects referred to dental sleep clinic during the study time period who were not treated with OA were collected, 11 of whom were excluded due to insufficient baseline sleep data. A total of 15 sleep laboratories were involved in the study of the patients. A comparison of baseline characteristics of subjects chosen for OA therapy vs those who were not is presented in Table 1. Among subjects referred for OA therapy, subjects selected for OA therapy were significantly less obese than those who were not (BMI median, 27.8 kg/m^2 vs 28.9 kg/m^2; p = 0.013). Other anthropomorphic measurements, age, gender, and baseline sleep characteristics did not differ between the two groups. Analyses did not differ significantly when the 11 subjects without baseline sleep data were included (data not shown). Reasons for not treating subjects with OA are given in Table 2. Treatment with OA was not pursued due to medical or dental concerns in 41.2% of the cases, and due to the nonmedical or dental concerns in 34% of cases. Potential risks and complications of concern to subjects included compliance to titration protocol, jaw pain or stiffness, teeth sensitivity, bite changes, and damage to teeth and dental work.

Overall, 37 subjects (64.9%) were successfully treated with OA therapy. Of the 49 subjects for whom data were available for AHI after home titration, 27 subjects (55%) achieved successful treatment of OSA by self-titration, without need for further titration during follow-up polysomnography.

Demographic and Body Anthropomorphic Factors

Odds ratios (ORs) for variate and multivariable analyses are presented in Table 3. Women were significantly less likely to have successful OA treatment than men (OR, 0.32; p = 0.05). With increasing age, subjects were less likely to have successful OA treatment, with a trend toward statistical significance (OR, 0.96; p = 0.08). Anthropomorphic characteristics, including BMI, neck circumference, canine classification, and facial skeletal classification, were not associated with likelihood of successful OA treatment. When simultaneously accounting for clinically significant patient and treatment characteristics, women were still significantly less likely to have successful OA treatment than men (OR, 0.25; p = 0.04). The multivariable logistic model demonstrated a good fit of the data (p = 0.18).

Severity of OSA and Success of OA Treatment

Subjects were compared in groups based on degree of baseline OSA (Fig 2). Of the 15 subjects with mild OSA, 10 subjects (67%) had successful treatment with OSA. Subjects with moderate OSA had a 68% (21 of 31 subjects) success rate for OSA treatment with OA. Subjects with severe OSA had a 54% treatment success rate (6 of 11 subjects). There was

Table 2—*Reasons for Not Treating Referred Patients (n = 68)**

Reasons	Primary	All†
Medical/dental		
Concerns about risks/complications	11 (16.2)	11 (16.2)
Not a dental candidate	17 (25.0)	19 (27.9)
Nonmedical/nondental reasons		
Insurance/finance	15 (22.1)	18 (26.5)
Prefers continuous positive airway pressure	7 (10.3)	8 (11.8)
Not convinced treatment is needed	1 (1.5)	2 (2.9)
Prefers surgery	0 (0)	2 (2.9)
Miscellaneous		
No polysomnography	1 (1.5)	2 (2.9)
Other/unknown	16 (23.5)	17 (25)

*Data are presented No. (%). The primary reason for not treating the patient with OA. All refers to all reasons given for not treating a given patient with OA.
†Does not add up to 100% due to multiple reasons in some patients.

Table 3—*ORs of Patient Characteristics Associated With Successful Treatment With OA (n = 57)*

	Bivariate Analysis		Multivariable Analysis†	
Patient Characteristics	OR	p Value	OR	p Value
Age	0.96	0.08	0.96	0.14
Gender (reference = male)	0.32	0.05	0.25	0.04
BMI	0.94	0.24	1.01	0.84
Neck circumference	1.02	0.93		
Right canine classification*				
I	Reference			
II D	2.28	0.18		
III	0.57	0.72		
Left canine classification*				
I	Reference			
II D	1	1.00		
III	1	1.00		
Skeletal classification (mandibular protrusion vs normal)*	3.33	0.29		
Pretreatment overbite*	1.67	0.14		
Pretreatment overjet*	1.40	0.24		
Pretreatment AHI	0.98	0.12		
Pretreatment oxyhemoglobin saturation	1.04	0.13	1.05	0.16
Time from initial treatment to titration polysomnography	1.00	0.71		

*Available from 20 subjects.
†Independent variables included in the multivariable analysis included age, gender, BMI, and pretreatment oxyhemoglobin saturation.

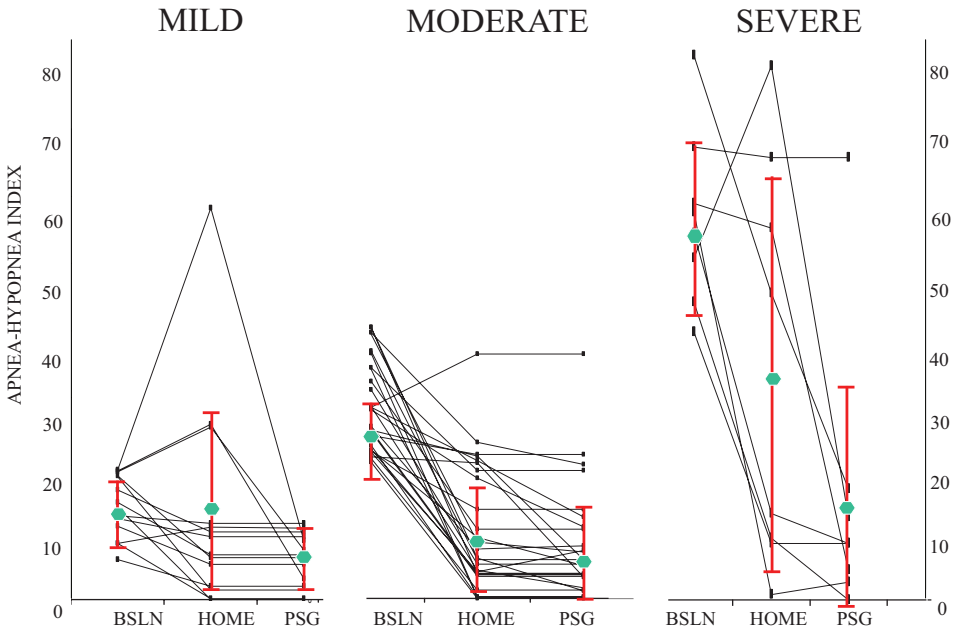

FIGURE 2. Treatment response to OAs by degree of OSA severity. Individual responses are tracked from baseline AHI, to AHI after home titration of OA, to AHI after polysomnography-guided titration for subjects with complete available data. Subjects are separated into subsets of mild (n = 14), moderate (n = 27), and severe (n = 8). BSLN = baseline AHI; HOME = AHI after home titration; PSG = AHI after follow-up titration polysomnography. Data are presented by individual patient (black lines) and aggregate data (mean, blue dots; SD, red bars).

no significant difference found in treatment success rates among subjects by degree of OSA severity (Pearson χ^2, $p = 0.72$).

Titration Process

Data were available for the AHI after home titration and prior to final OA titration, for 49 of 57 subjects treated with OA, 34 of whom (69%) had eventual successful treatment of OSA with OA. Of the subjects successfully treated, 27 subjects (79%) were treated successfully at the home titration stage, without need for further titration during follow-up polysomnography. Seven of the 22 subjects (32%) who did not achieve successful OA titration at home benefited from follow-up titration study and achieved successful treatment. Overall for the 49 subjects who were treated with OA for whom full follow-up titration data are available, there was significant reduction in AHI from baseline to home titration of OA ($p < 0.001$), and overall laboratory titration reduced the AHI more than home titration ($p < 0.001$), even after Bonferroni correction.

DISCUSSION

Our study has several interesting findings. First, patients referred for OA therapy are more likely to be treated if they are less obese. An earlier review[9] of OA therapy suggests more successful in patients who are less obese, and the difference in BMI in patients treated vs not treated with OA may be influenced by this knowledge. Second, women selected for OA therapy were less likely to have successful treatment of their OSA than men, even after accounting for differences in age, BMI, and baseline oxyhemoglobin saturation. Third, differences in severity of baseline OSA did not impact on efficacy of OA treatment. While current treatment standards indicate OA therapy is first-line therapy for mild-to-moderate OSA, our findings support the thinking that OA therapy can also be considered for patients with severe OSA. Finally, we show that while a majority of patients (55%) treated for OSA with OA achieve successful self-titration at home, without the need for further polysomnography-guided titration, a significant number of patients who do not achieve successful home titration benefit from follow-up titration by polysomnography, supporting the need for follow-up polysomnography for patients treated with OA.

Our finding that women selected for OA therapy were less likely to be successfully treated for OSA than men is a novel finding. Most previous studies have found no gender difference. Marklund and Franklin[10] found female gender to be a positive predictor of OA success. Established causes of OSA

include anatomic obstruction as well as local neuromuscular responses and ventilatory responses to upper airway obstruction to explain sleep-disordered breathing. Our study suggests that mandibular advancement is more effective in men for OSA treatment. Women may have either less of a response in terms of upper airway caliber enlargement to mandibular advancement, or a greater perturbation in local neuromuscular responses or ventilatory responses to explain the etiology of their OSA.

An optimal titration strategy for OA devices has not yet been established. The treatment success of OA depends on both the degree of titration of the OA device and the individual response in upper airway caliber and compliance to mandibular advancement.[11] Several studies have examined the feasibility of a single-night pretreatment oral appliance titration as a predictor of long-term OA efficacy with mixed results. In each case, a trial manual or automated mandibular advancement device was used during the test night. One possible complication of this approach is that a different OA than the trial device was subsequently fabricated for treatment.[12] The efficacy of titration with one device, and long-term treatment with a different device, is not comparable to titration and long-term treatment with the same device, as is proposed in our study. Suggested titration strategies employing temporary remote-controlled OA devices during titration polysomnography require expensive additional equipment, as well as introduce a difference in treatment effect between the titration study device and actual long-term therapy OA device.[13–15] Fleury et al[16] utilized home titration of an adjustable OA before performing a follow-up polysomnography. The home titration was guided by patient symptoms and oximetry. A fourth of the home titration adjustments were made in asymptomatic subjects due to abnormal oxygen desaturation index, suggesting that a more aggressive titration protocol may increase treatment success. Parker et al[17] reported an overall OA success rate of 92% after combined home and polysomnography OA titration in a retrospective study. As in our study, subjects who failed to return for follow-up polysomnography were excluded from the study. The success rate is reported to have increased by 40% due to the polysomnography titration. While we did not study long-term treatment effect of our OA devices, the current study suggests an effective short-term strategy for prescribing OA.

We acknowledge limitations to our study. First, while all study subjects were evaluated at the same dental sleep clinic, polysomnography was performed and analyzed at different sleep laboratories. While this may introduce inconsistencies in intersubject interpretation, all efforts were made, as part of routine clinical care, to have follow-up studies done at the same laboratory as baseline studies, and we demonstrated the feasibility of carrying out this titration protocol successfully in a variety of hospital and private sleep laboratory settings. Also, we would have ideally wanted a full baseline polysomnography with OA therapy after home titration, and full-night polysomnography of the final OA titration effect, but this was out of the scope of our study. While no minimum standard was established for the duration of intervals, data were provided for intervals of sleep that included uninterrupted rapid eye movement (where available) and were of sufficient length (usually >1 hour) to merit confidence. The titration protocol used in this study, while limited by the necessity to awaken the subject to adjust the device, was a reasonable method that can be simply executed in an American Academy of Sleep Medicine-accredited sleep laboratory without additional equipment besides the OA. Subsequent studies are required to determine the long-term effect of final titration of OA therapy using this method. Next, because this was not a prospective study, the data available for analysis was limited to the de-identified data available. A selection bias may be introduced due to exclusion of patients treated with an OA who did not return for follow-up polysomnography titration. These patients were presumably unavailable for follow-up for treatment satisfaction, treatment failure, noncompliance to medical recommendations, or issues with the sleep study, but these data and these patient baseline characteristics were not available to assess the impact of this subset of patients on our findings. Variables such as percentage of apneas vs hypopneas at baseline and after treatment would have been insightful in our analysis. Finally, our study explores the effects of multiple different types of mandibular advancement devices. However, we feel that the general effect of mandibular advancement, regardless of the particular OA used, is what is evaluated in our data.

Our findings support the role for OA therapy for treatment of OSA. While currently OAs are recommended for only mild-to-moderate OSA as first-line therapy, our evidence suggests OA treatment of patients with severe OSA may also be beneficial. Further studies are certainly needed to determine whether the final OA mandibular position determined by the current titration protocol is the optimal OA position for long-term therapy. The findings that the proposed titration protocol offers symptom relief in a majority of patients, and results in treatment success in 64% of patients suggest that our proposed titration protocol is a reasonable initial method to titrate OA position for treatment of OSA.

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