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A Randomized Crossover Study of an Oral Appliance vs Nasal-Continuous Positive Airway Pressure in the Treatment of Mild-Moderate Obstructive Sleep Apnea*

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Study objective: **To compare efficacy, side effects, patient compliance, and preference between oral appliance (OA) therapy and nasal-continuous positive airway pressure (N-CPAP) therapy.**
Design: **Randomized, prospective, crossover study.**
Setting: **University hospital and tertiary sleep referral center.**
Patients: **Twenty-seven unselected patients with mild-moderate obstructive sleep apnea (OSA).**
Interventions: **There was a 2-week wash-in and a 2-week wash-out period, and 2×4-month treatment periods (OA and N-CPAP). Efficacy, side effects, compliance, and preference were evaluated by a questionnaire and home sleep monitoring.**
Measurements and results: **Two patients dropped out early in the study and treatment results are presented on the remaining 25 patients. The apnea/hypopnea index was lower with N-CPAP (3.5±1.6) (mean±SD) than with the OA (9.7±7.3) ($p<0.05$). Twelve of the 25 patients who used the OA (48%) were treatment successes (reduction of apnea/hypopnea to <10/h and relief of symptoms), 6 (24%) were compliance failures (unable or unwilling to use the treatment), and 7 (28%) were treatment failures (failure to reduce apnea/hypopnea index to <10/h and/or failure to relieve symptoms). Four people refused to use N-CPAP after using the OA. Thirteen of the 21 patients who used N-CPAP were overall treatment successes (62%), 8 were compliance failures (38%), and there were no treatment failures. Side effects were more common and the patients were less satisfied with N-CPAP ($p<0.005$). Seven patients were treatment successes with both treatments, six of these patients preferred OA, and one preferred N-CPAP as a long-term treatment.**
Conclusions: **We conclude that OA is an effective treatment in some patients with mild-moderate OSA and is associated with fewer side effects and greater patient satisfaction than N-CPAP.**
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Key words: nasal CPAP; obstructive sleep apnea; oral appliances

Abbreviations: AHI=apnea/hypopnea index; EMG=electromyogram; N-CPAP=nasal-continuous positive airway pressure; OA=oral appliance; OSA=obstructive sleep apnea; SaO_2=arterial oxygen saturation

Obstructive sleep apnea (OSA) is a common disorder that may affect at least 2 to 4% of the adult population.[1] The treatment of OSA depends on the severity of symptoms, magnitude of clinical complications, and etiology of upper airway obstruction. Weight reduction, avoidance of alcohol, and relief of nasal obstruction are conservative measures that should be addressed in every patient. However, in most symptomatic patients, additional treatment is usually required. Nasal-continuous positive airway pressure (N-CPAP) is a highly effective and safe treatment for OSA and is generally considered to be the current primary treatment for OSA. Reported long-term use of N-CPAP in patients with OSA is 50 to 80%, and less symptomatic patients are more likely to discontinue treatment. However, even among patients who report regular use of the treatment, covert monitoring has

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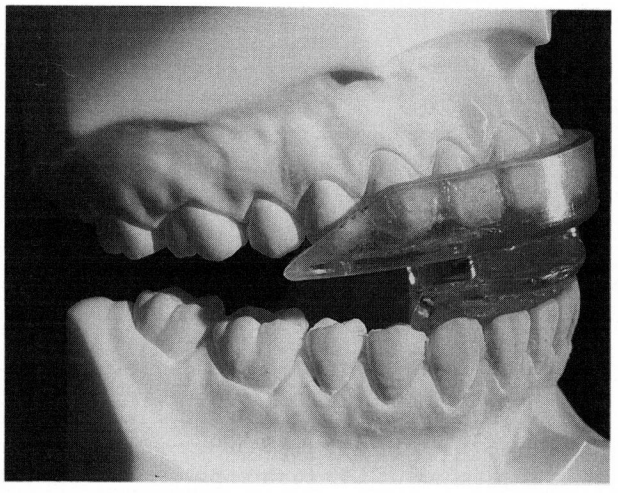

FIGURE 1. Anterior mandibular positioner (Snore-Guard).

shown that average usage is less than 50% of the night.[2] Thus, alternative treatments that are safe, effective, and acceptable are needed. A variety of other therapeutic approaches have been proposed for the treatment of OSA, including pharmacologic intervention, uvulopalatopharyngoplasty, and maxillomandibular surgery. There is, however, a paucity of controlled clinical studies to determine the efficacy of any of these treatments.

The development of oral appliances (OAs) represents an interesting new approach for the management of OSA.[3] This treatment approach is simple, reversible, and cost-effective. There are limited data concerning the efficacy, side effects, and compliance of OA treatment. Furthermore, to our knowledge, there are no comparative data between N-CPAP and OA in patients with OSA. The primary objective of this study was to compare the subjective and objective efficacy, side effects, patient compliance, and preference between OA and N-CPAP in the treatment of patients with mild to moderate OSA.

MATERIALS AND METHODS

Subjects

Twenty-seven patients with symptomatic mild to moderate OSA (apnea and hypopnea index [AHI], 15 to 50/h of sleep during diagnostic laboratory polysomnography) were recruited for this study. Patients were unselected apart from a requirement that they have at least ten teeth in each of the maxillary and mandibular arches, and reside in the metropolitan Vancouver area. All patients were seen in the Sleep Disorders Clinic at the Vancouver Hospital and Health Sciences Center between November 1991 and April 1994. Each patient gave informed written consent and the study protocol was approved by the Clinical Screening Committee for Research and other Studies Involving Human Subjects, at the University of British Columbia. The patients did not have to pay for either treatment.

Oral Appliance

A specific type of OA was used during this study (Snore-Guard; Hays & Meade Inc; Albuquerque, NM).[4] This appliance is constructed of an acrylic polymer. It attaches securely to the anterior upper teeth and advances the mandible by means of a projection that engages the mandibular incisors when the teeth are in contact (Fig 1). The OA was constructed to position the mandible 3 mm posterior to the position of maximal acceptable advance and with a 7-mm opening between the upper and lower incisors. The position of the mandibular projection relative to the maxilla was chosen so that the mandible would be advanced as far as possible without stressing the temporomandibular joints. The appliance was adjusted to maximize comfort by relieving pressure points on the teeth and gums. In a few patients, material was added to increase the vertical dimension of the appliance to improve retention.

N-CPAP

N-CPAP therapy was undertaken with one of two machines (either a REMstar Choice Machine; Respironics Inc; Murrysville, Pa; or a Tranquillity Plus Machine; Healthdyne Technologies; Marietta, Ga), which were the most advanced N-CPAP units available at the time of the study. Patients used a variety of different airway access devices based on their own preference. The use of a humidifier was optional although encouraged. Intranasal corticosteroids and/or anticholinergic medications were used to relieve any nasal symptoms caused by the N-CPAP. All patients underwent an in-hospital overnight polysomnogram at the beginning of the N-CPAP treatment period to determine the optimal pressure necessary to completely relieve the OSA.

Questionnaire and Physical Examination

All patients underwent a complete history and physical examination prior to recruitment to the study. A detailed questionnaire that included questions about symptoms, treatment efficacy, side effects, and patient satisfaction was administered during the treatment and wash-out periods. Side effects were rated both in terms of frequency (never, rarely, sometimes, often) and severity (absent, mild, moderate, severe). Patient satisfaction was rated very satisfied, moderately satisfied, moderately dissatisfied, or very dissatisfied. At the end of the study, all patients were asked whether they would prefer to use OA or N-CPAP as a long-term treatment.

Diagnostic Polysomnography

Each patient had a diagnostic overnight polysomnogram performed prior to recruitment. Sleep and its various stages were documented by standard EEG, electro-oculographic, and electromyographic (EMG) criteria.[5] EEG was recorded with electrodes applied at C_3-A_2 and C_4-A_1 (according to the International 10-20 system) and EMG activity was recorded from the submental muscles. Oronasal airflow was recorded by an infrared CO_2 analyzer (model 1260; Novamatrix Medical Systems Inc; Wallingford, Conn). A single-ECG lead (modified V_2) was monitored to detect cardiac arrhythmias. Arterial oxygen saturation (SaO_2) was monitored continuously with a pulse oximeter (model N-100; Nellcor Inc; Hayward, Calif) attached to the index finger. Chest wall movement was monitored by a respiratory inductive plethysmograph (Respitrace; Ambulatory Monitoring Equipment; Ardsley, NY). The data were recorded on a 15-channel polygraph (model 78; Grass Instruments Co; Quincy, Mass) into a system (CNS Sleep Lab System model 200; CNS Inc; Chanhassen, Minn). All of these studies were manually scored for sleep stage and apnea type and duration. Obstructive apneas were defined as the cessation of airflow for at least 10 s accompanied by ongoing respiratory effort. Central apneas were defined as the cessation of airflow and respiratory effort for at least 10 s. Mixed apneas were defined as a combination of an obstructive and central apnea lasting for at least 10 s. Hypopneas were defined as a greater than 50% decrease in thoracoabdominal amplitude (Respitrace sum) for at least 10 s.[6] Severity of OSA was assessed in terms of the AHI.

Home Sleep Monitoring

Home sleep monitoring was performed with a combined system (Poly-G Portable Apnea Recorder and Sleep I/T-8; CNS Inc).[7] This combined system recorded EEG (C_3-A_1), limb EMG, electro-oculogram, ECG, oronasal airflow (thermistor), respiratory effort (chest and abdomen), SaO_2, body position, and limb activity. The data were stored in solid-state memory and transferred into a personal computer. The real-time respiratory data were generated for analysis following the monitoring. This allowed full manual analysis of the respiratory sleep data (airflow, respiratory effort, oxygen desaturation). Sleep staging was done by recently validated automated analysis[8] as the raw data are not available with this monitoring system.

Study Design

The study consisted of a 2-week wash-in period following the initial diagnostic polysomnogram. During this time, the patients were not treated and baseline home sleep monitoring was performed. The questionnaire was administered during the wash-in period. The patients were also weighed at this time and subsequently on each occasion the questionnaire was administered. The patients were then randomized to treatment with either the OA or N-CPAP. The questionnaire and home sleep monitoring were repeated at the end of the 4-month treatment period. The patients then had a 2-week wash-out period during which they were not treated. At the end of this wash-out period, the questionnaire was administered and home sleep monitoring was performed. The patients then underwent a second 4-month treatment period with the other treatment and the questionnaire and home sleep monitoring were performed at the end of this treatment period. Patients were seen monthly during each treatment period. When patients had side effects or were unable to comply with OA treatment, the OA was readjusted to maximize comfort. When patients had side effects or were unable to comply with N-CPAP, we encouraged the use of a humidifier and often changed the type of airway access device. Treatment success was defined as a resolution of symptoms and a reduction in AHI to less than 10/h. Treatment failure was defined as ongoing clinical symptoms and/or a reduction in AHI to more than 10/h. Compliance failure was defined as an inability or unwillingness of the patient to continue to use the treatment.

Statistical Analysis

The data were analyzed according to the method of Jones and Kenward.[9] Each patient received the treatments in one of two possible orders: sequence A (OA followed by N-CPAP) or sequence B (N-CPAP followed by OA). The treatment effect was estimated using the differences of the pairs of observations from each patient (pretreatment and posttreatment). To rule out a treatment-by-period interaction, tests for carryover effect and period effect were performed.[10] The questionnaire and home sleep monitoring data were analyzed by groups such that the results from N-CPAP in sequence A were compared with the results of N-CPAP in sequence B. This was also done for the OA treatment from sequence A and sequence B. Comparisons were also made between the different treatments in each sequence (OA vs N-CPAP for sequence A and B). The results were pooled if there was no difference between the same treatment from the different sequences and if the differences between the two treatments within each sequence were the same for sequence A and B. The data obtained during the wash-in and wash-out periods prior to the OA and prior to N-CPAP were compared to ensure that the baseline values preceding each treatment were the same.

The questionnaire symptom data were grouped into three possible responses—improved, unchanged, or worse. The data were then compared in the same manner as described above before pooling. The symptom, side effect, and compliance data were compared by χ^2 analysis.

The data from OA sequence A were compared to OA sequence B and from N-CPAP sequence A to N-CPAP sequence B by an unpaired *t* test. The results from OA sequence A were compared to N-CPAP sequence A and from OA sequence B to N-CPAP sequence B with a paired *t* test. The final comparisons were made with a paired *t* test on the pooled OA and N-CPAP data from both treatment sequences.

Table 1—*Demographic and Polysomnographic Data*

Data	No.
No.	27
Sex, M/F	24/3
Age, yr	46.2±10.9 (25-72)*
Body mass index, kg/m^2	30.4±4.8 (21.1-42.6)*
Neck circumference, cm	42.2±3.1 (35-49)*
AHI, No./h	24.5±8.8 (15-50)*

*Mean±SD (range).

Results

Twenty-seven patients were recruited, including 24 men and 3 women. These patients were, in general, middle aged, overweight, and had mild to moderate OSA (Table 1). One patient dropped out during the wash-in period after the N-CPAP titration night and another patient dropped out early in the first treatment period with the OA when he moved out of town. All subsequent results are presented on the remaining 25 patients.

There was no carryover effect between the treatment periods and no period effect on the home respiratory sleep data so these data from the OA and N-CPAP were pooled between the two different treatment sequences. There were differences in the sleep architecture between the OA treatments from sequence A and sequence B. The patients who used the OA after N-CPAP (sequence B) had a greater change in stage 1 sleep and slow-wave sleep between the baseline study and the treatment study. These patients

Table 2—*Home Sleep Monitoring Data With and Without the OA (n=19)**

	Pre-OA	OA
AHI, No./h†	19.7±13.8	9.7±7.3
Apnea index, No./h†	6.2±8.1	1.9±3.3
Total sleep time supine, %	39.8±27.4	43.6±24.2
Desaturations <90%, No.	56.2±75.6	35.6±73.8
Minimum SaO_2, %	83.0±7.4	83.8±7.3
Total sleep time, min	400.3±56.5	376.7±69.9
Sleep efficiency, %	88.0±5.4	86.5±10.6
Awakenings, No.	24.6±13.4	22.1±14.8
NREM, %	80.3±14.7	77.7±12.1
REM, %	14.3±6.5	20.0±12.3

*Values are mean±SD. NREM=nonrapid eye movement sleep; REM=rapid eye movement sleep.
†$p<0.005$.

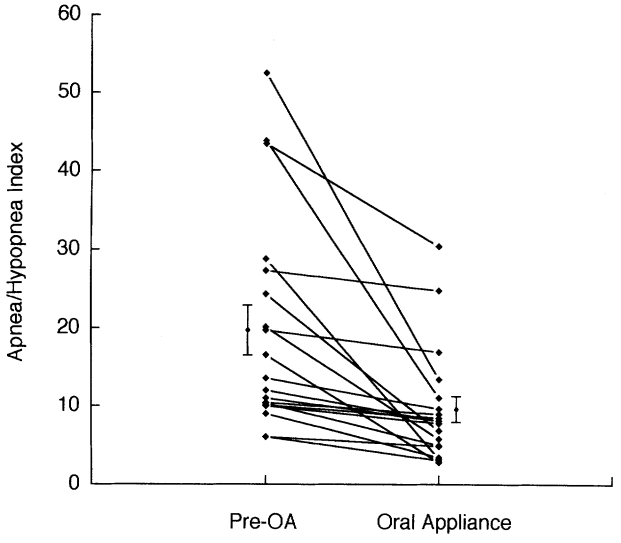

FIGURE 2. AHI with and without the OA (mean±SE).

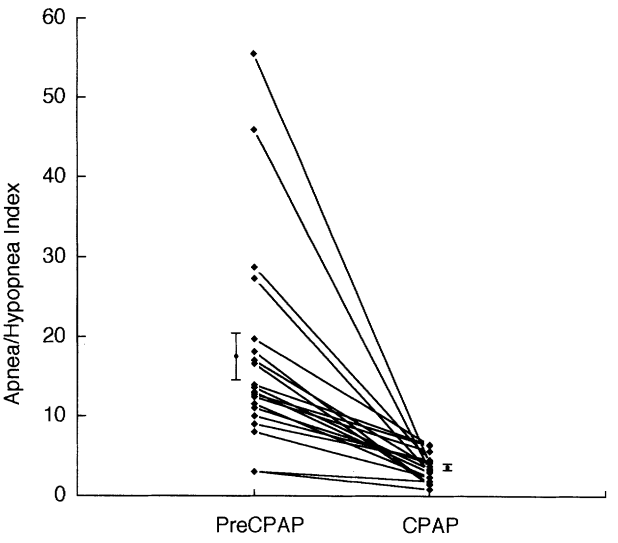

FIGURE 3. AHI with and without N-CPAP (mean±SE).

had less stage 1 sleep and more slow-wave sleep than the patients who used the OA before N-CPAP (sequence A). This was the only period effect found. There were no differences in body weight or in the home sleep monitoring data measured pretreatment with the OA from that measured pretreatment with N-CPAP. There was no significant change in body weight during either the OA or N-CPAP treatment periods.

Twenty-five patients were treated with the OA and there were 6 patients (24%) who were compliance failures. These six patients were unable to wear the OA even after adjustments were made. In one patient, there was moderate to severe jaw discomfort in addition to poor retention of the OA. The remaining 19 patients had follow-up studies performed with the OA. The pretreatment AHI measured at home in the 19 patients was 19.7±13.8/h (mean±SD) and the mean AHI decreased to 9.7±7.3/h with the OA ($p<0.005$) (Table 2 and Fig 2). This represents a greater than 50% reduction in mean AHI with the OA. There were 7 treatment failures in whom the AHI was not reduced to the normal range (<10/h) and/or they had ongoing clinical symptoms.

The average prescribed pressure with N-CPAP was 9.3±1.5 cm H_2O (range, 7.0 to 12.0). Twenty-one of the 25 patients used N-CPAP and 20 of these patients had follow-up home sleep monitoring. Four patients refused to use N-CPAP after using the OA because they were satisfied with this treatment or preferred to use another OA instead of using N-CPAP. One of the compliance failures with N-CPAP did not have follow-up home sleep monitoring. The pretreatment AHI was 17.5±13.2/h and with N-CPAP decreased to 3.5±1.6/h ($p<0.005$) (Table 3 and Fig 3). AHI was lower with N-CPAP than OA ($p<0.05$). There was also

Table 3—*Home Sleep Monitoring Data With and Without N-CPAP (n=20)

	Pre-N-CPAP	N-CPAP
AHI, No./h†	17.6±13.2	3.6±1.7
Apnea index, No./h†	5.7±6.2	0.4±0.6
Total sleep time supine, %	40.5±26.2	48.4±33.1
Desaturations <90%, No.†	53.5±76.4	3.5±6.4
Minimum SaO_2, %†	82.6±6.0	88.7±2.5
Total sleep time, min	416.8±60.0	402.7±93.6
Sleep efficiency, %	87.8±7.7	88.1±7.3
Awakenings, No.	28.9±16.6	22.0±11.5
NREM, %	79.9±11.7	80.8±7.8
REM, %	16.5±8.2	16.1±6.1

*Values are mean±SD. NREM=nonrapid eye movement sleep; REM=rapid eye movement sleep.
†$p<0.005$.
‡$p<0.05$.

Table 4—*Symptoms With and Without the OA and N-CPAP*

	Pre-OA, No. (%)	OA, No. (%)
Moderate to loud snoring*	25/25 (100)	6/25 (24)
Witnessed apneas*	21/25 (84)	2/25 (8)
Unrefreshing sleep†	20/25 (80)	12/25 (48)
Excessive daytime sleepiness/ fatigue*	21/25 (84)	10/25 (40)
Sleepiness while driving†	11/25 (44)	4/25 (16)
	Pre-N-CPAP, No. (%)	N-CPAP, No. (%)
Moderate to loud snoring*	21/21 (100)	0/21 (0)
Witnessed apneas*	17/21 (81)	1/21 (5)
Unrefreshing sleep†	21/21 (100)	6/21 (29)
Excessive daytime sleepiness/ fatigue*	18/21 (86)	5/21 (24)
Sleepiness while driving†	6/21 (29)	1/21 (5)

*$p<0.005$.
†$p<0.05$.

an improvement in oxygenation with N-CPAP ($p<0.005$) but not with the OA. There was no difference in sleep architecture, awakenings, or sleep efficiency with the OA or with N-CPAP compared to pretreatment values.

Both the OA and N-CPAP were effective in reducing symptoms associated with OSA (Table 4). Snoring was still present in six patients with the OA and all of these were treatment or compliance failures. Snoring was eliminated in all patients with N-CPAP. The OA was not as effective as N-CPAP in relieving symptoms of excessive daytime sleepiness ($p<0.05$).

Minor side effects were common with the OA, particularly in the first month of treatment. These side effects included sore teeth, a sore jaw, and excessive salivation. In most patients, the side effects were mild and improved with time. By the end of the 4-month treatment period, 9 patients (36%) had persistent mild side effects, 5 patients (20%) had moderate side effects, 1 patient (4%) had severe side effects, and the remaining 10 patients (40%) had no side effects (Table 5). No patient developed any symptoms of temporomandibular joint dysfunction. Seventeen patients (68%) were moderately or very satisfied with the treatment. One patient who was a treatment failure discontinued OA treatment early.

Side effects were more common and the patients were less satisfied with N-CPAP ($p<0.005$), and six patients finished N-CPAP treatment early because of side effects that included persistent nasal symptoms and a sense of suffocation. Four patients used nasal medications to help improve nasal symptoms. Twenty-one patients used N-CPAP and after the 4-month treatment period, 1 patient (5%) had mild side effects, 5 (24%) had moderate side effects, and 4 (19%) had severe side effects (Table 5). The patient who did not have follow-up home sleep monitoring with N-CPAP had stopped using it because of severe nasal symptoms. Thirteen patients (62%) were moderately or very satisfied with N-CPAP. Eleven of the 12 patients who were treatment successes with the OA preferred it to N-CPAP. Two patients who were treatment successes with N-CPAP preferred N-CPAP to the OA. Seven patients were treatment successes with both treatments; six of these patients preferred OA and one preferred N-CPAP as a long-term treatment. Compliance was assessed subjectively by patient questionnaire data (Table 5). There was no difference in the reported percent of the night or nights/week treatment was used between the OA and N-CPAP groups. Based on our prestudy outcome definitions in the OA group, 12 of 25 (48%) were treatment successes, 6 of 25 (24%) were compliance failures, and 7 of 25 (28%) were treatment failures. In the N-CPAP group, 13 of 21 (62%) were treatment successes, 8 of 21 (38%) were compliance failures, and there were no treatment failures.

Table 5—*Side Effects and Patient Satisfaction With the OA and N-CPAP*

	OA, No. (%) (n=25)	N-CPAP, No. (%) (n=21)
Side Effects*		
None	10 (40)	11 (52)
Mild	9 (36)	1 (5)
Moderate	5 (20)	5 (24)
Severe	1 (4)	4 (19)
Satisfaction*		
Very satisfied	14 (56)	4 (19)
Moderately satisfied	3 (12)	9 (43)
Moderately dissatisfied	3 (12)	1 (5)
Very dissatisfied	5 (20)	7 (33)
Percent of nights treatment used†		
100	15 (60)	10 (48)
>75	9 (36)	4 (19)
25-75	1 (4)	3 (14)
<25	0	4 (19)
Percent of the night treatment used†		
100	12 (48)	9 (43)
>75	8 (32)	5 (24)
25-75	4 (16)	3 (14)
<25	1 (4)	4 (19)

*Significant difference, $p<0.005$.
†No difference.

Discussion

This study is one of the first randomized, prospective crossover studies comparing an OA to N-CPAP in the treatment of an unselected group of patients with OSA. We have shown that OAs are an effective treatment in some patients with mild to moderate OSA. Forty-eight percent of the OA group were treatment successes compared to 62% of the N-CPAP group. The 3 patients in the OA group with an AHI greater than 40/h were all treatment failures. However, 2 of these 3 patients had a 75% reduction in their AHI associated with relief of symptoms, but they were considered treatment failures because their treatment AHI (11/h and 13/h) were above our prestudy outcome definitions of 10/h. N-CPAP was more effective at improving sleep arterial oxygen desaturation and daytime sleepiness. There was no difference in reported compliance, but OA was associated with fewer side effects and greater patient satisfaction than N-CPAP. The long-term preference was overwhelmingly in favor of OA therapy. Eleven of the 12 patients with a treatment success with OA chose it as a long-term treatment option after completion of the study. Only two patients preferred N-CPAP as their long-term treatment option.

OAs represent an interesting new approach to the treatment of OSA and this has recently been the subject of a detailed review.[3] Mandibular advancement by

an OA was first reported by Robin[11] in 1934 as a treatment for upper airway obstruction in infants with mandibular hypoplasia. More recently, a variety of OAs have been proposed for the treatment of OSA.[3] The tongue-retaining device reduces the number and duration of apneas and improves sleep quality in patients with OSA.[12] The tongue-retaining device is believed to be most successful in patients who are less than 50% above ideal weight and in whom their OSA is worse when they sleep in the supine position.[13] The proposed mechanism of action of the tongue-retaining device is that it holds the tongue in a forward position preventing it from being drawn downward by the negative pressure of inspiration. The other common form of OA treatment is a mandibular repositioning appliance which is believed to act by advancing the mandible and increasing upper airway size. Schmidt-Nowara and coworkers[4] have reported their experience with the same mandibular repositioning appliance as was used in our study. They evaluated 68 patients with snoring or OSA. In the 20 patients with follow-up polysomnography, the OA reduced the AHI by more than 50% and also significantly improved SaO_2 and sleep quality. Clark and coworkers[14] have reported their experience with an anterior mandibular positioning device in 24 patients with OSA. In the 15 patients who had polysomnography before and after treatment, 12 had a reduction in AHI to less than 15/h. Several of the patients with a poor response to treatment did not have follow-up polysomnography, so the exact success rate is not known. O'Sullivan and coworkers[15] have recently shown that a mandibular advancement splint decreases AHI to less than 20/h in 12 of 17 patients in whom untreated AHI was between 20 to 60/h, and in 2 of 9 patients in whom untreated AHI was more than 60/h. Eveloff and colleagues[16] reported on the results of an anterior mandibular positioning appliance in 19 patients with OSA. Their success rate was 53% when they defined treatment responders as having an AHI less than 10/h with the OA. They also performed lateral cephalometry in patients with and without the OA and compared the responders and nonresponders. They found that two cephalometric indexes were associated with treatment success. The distance from the hyoid bone to the mandibular plane was smaller and the soft palate length was shorter in the responder group. Certain orthodontic characteristics may also be important in selecting patients for OA therapy. The one patient in our study who had a skeletal anterior open bite was unable to keep the OA in his mouth at night. Anterior mandibular positioning devices require adequate dentition, particularly anterior teeth, in order to anchor the appliance and hold the mandible forward. The tongue-retaining device can be used in edentulous patients.

N-CPAP is generally established as the primary treatment for symptomatic patients with OSA who do not respond to conservative measures.[17] N-CPAP is not a realistic long-term treatment option in some patients because of side effects, discomfort, or inconvenience. Success and compliance rates vary considerably depending on the criteria of compliance, experience in initiation of treatment, adequacy of follow-up, type of CPAP machine and mask used, and the other treatment modalities available. Rauscher and colleagues[18] reported that the acceptance of N-CPAP for long-term use was 72% after a first night of treatment. Long-term acceptance of N-CPAP has been found to be best in patients with excessive daytime sleepiness and more severe OSA.[19,20] It has been found that patients overestimate their use of N-CPAP considerably when compliance is measured objectively.[21] In one study, 66% of patients claimed to use it nightly but covert monitoring revealed that only 46% of patients used it at least 4 h per night more than 70% of nights.[2] Therefore, although N-CPAP is highly effective in treating OSA, there can be substantial problems with patient acceptance and long-term compliance. As such, there is a need for alternative treatments for patients with mild to moderate OSA that are both effective and acceptable.

Our study has certain limitations. Our sample size was relatively small and not all patients completed the study. Two patients dropped out of the study and another four patients refused to cross over to N-CPAP after using the OA. Five compliance failures with the OA and one compliance failure with N-CPAP did not have follow-up home sleep monitoring. Some of these problems are inherent in any clinical trial. Our crossover design has excellent power characteristics for detecting small treatment effects and any potentially confounding carryover or period effects.[9] We used home sleep monitoring to establish treatment efficacy. This was necessary because of the expense associated with the large number of follow-up studies. We believe home sleep monitoring is appropriate for treatment follow-up once the diagnosis of OSA has been established. All treatment comparisons were made based on data from the home sleep monitoring and no comparisons were made with the initial diagnostic polysomnography. Two recent studies have validated our home sleep monitoring system and have shown excellent reliability between laboratory and home sleep data.[7,8] We also recognize the limitations of subjective reports of treatment compliance, but at the time of the study, the technology was not available for covert monitoring of either OA and N-CPAP compliance. Finally, we present only subjective data on excessive daytime sleepiness and recognize that if the resources had been available, objective data would have been preferable.

N-CPAP remains the primary treatment for patients with severe OSA and associated arterial oxygen desaturation and daytime sleepiness. We have shown that OA therapy is effective in some patients with mild to moderate OSA and is associated with fewer side effects and greater patient satisfaction than N-CPAP. OA should be considered for the treatment of patients with mild to moderate OSA as most patients prefer this treatment to N-CPAP when both treatments have been shown to be effective. OA can be used as an adjunct to N-CPAP therapy when the patient is away from home and is unable or unwilling to use N-CPAP. Larger randomized clinical trials are necessary to further determine the precise indications, benefits, and risks of each of these OAs in the treatment of OSA. These studies should include objective assessment of daytime performance, covert compliance monitoring, and long-term follow-up.

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