

An Oral Elastic Mandibular Advancement Device for Obstructive Sleep Apnea

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Oral mandibular advancement devices are becoming an increasingly important treatment alternative for obstructive sleep apnea (OSA). The first aim of the study was to determine whether a new oral elastic mandibular advancement device (EMA) prevents pharyngeal airway closure during sleep in patients with OSA. The second aim of the study was to determine if the polysomnographic response to the oral mandibular advancement device was dependent on the site of airway closure. Overnight polysomnograms were performed in 28 untreated OSA subjects with and without EMA. A third polysomnogram was performed in 12 of the subjects to determine the site of airway closure without the device. Site of airway closure above or below the oropharynx was determined by measuring the respective presence or absence of respiratory fluctuations in oropharyngeal pressure during induced occlusions in non-rapid eye movement (NREM) sleep. Mean apnea-hypopnea index (AHI) was 52.6 ± 28.2 (SD) events/h without the device and 21.2 ± 19.3 events/h with the device. Nineteen subjects (68%) had at least a 50% reduction in AHI with the device. The change in AHI with the device (AHI without device – AHI with device) was directly related to the AHI without the device. All three subjects with airway closure in the lower pharyngeal airway had a greater than 80% reduction in AHI with the device. Two of the nine subjects with airway closure in the velopharynx had a similar therapeutic response. The results show the effectiveness of EMA in the treatment of OSA. The results also indicate that polysomnographic severity of OSA and the site of airway closure should not be used to exclude patients from this oral device treatment. Henke KG, Frantz DE, Kuna ST. An oral elastic mandibular advancement device for obstructive sleep apnea.

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Oral mandibular advancement devices are becoming an increasingly important treatment alternative for obstructive sleep apnea (OSA). A variety of appliances are commercially available, differing widely in design and the manner in which they alter the oral cavity. Previous studies by other investigators have tested the ability of these devices to prevent pharyngeal airway closure during sleep in patients with OSA, and report that the apnea-hypopnea index (AHI) decreases by 40 to 75% with the device (1–15). An oral mandibular advancement device has also been reported to be effective in treating upper airway resistance syndrome (16).

Recently, a new oral mandibular advancement appliance (EMA) has been developed by one of the authors (D. Frantz) for the treatment of OSA. The device consists of two plastic trays custom molded to the patient's maxillary and mandibular teeth. Elastic straps attached to the upper and lower trays

pull the mandible forward. The amount of advancement can be adjusted by altering the length and elasticity of the straps. The device allows lateral, vertical, and anteroposterior movement of the mandible while advancing the mandible in a ventral and caudal direction. The first aim of the current study was to determine whether this new appliance prevents pharyngeal airway closure during sleep in patients with OSA.

While the effectiveness of nasal continuous positive airway pressure (CPAP) treatment of OSA is independent of the site of pharyngeal airway closure, studies indicate that the effectiveness of uvulopalatopharyngoplasty, a resection of soft tissue in the velo- and oropharynx, is dependent on the site of closure. Launois and coworkers (17) and Hudgel and coworkers (18) have shown that OSA patients with airway closure in the velopharynx are more likely to have an initial beneficial outcome. A recent study by Millman and coworkers (19) reported that patients with continued OSA after uvulopalatopharyngoplasty had a very favorable polysomnographic response to oral mandibular advancement device treatment. The aforementioned studies raise the possibility that the ability of an oral mandibular advancement device to prevent pharyngeal airway closure during sleep is also dependent on the site of airway closure. The second aim of the study was to determine if the polysomnographic response to EMA was dependent on the site of airway closure. We hypothesized that OSA subjects with airway closure in the lower pharyngeal airway would be more likely to respond to an oral device owing to the ventral

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displacement of the tongue and hyoid apparatus with mandibular advancement.

METHODS

Subject Selection

The protocol was performed on 28 untreated subjects with OSA (24 males and 4 females): age 49.1 ± 10.1 yr (mean $\pm$ SD), mean body mass index 34.2 ± 6.1 kg/m², mean neck circumference 43.7 ± 3.8 cm. During the subject recruitment phase of the study, consecutive patients evaluated in the sleep laboratory with untreated OSA were asked to participate. The criterion for inclusion in the study was an AHI > 10 events/h on an overnight polysomnogram. Exclusion criteria included edentulous patients and patients who had an oxygen saturation $< 85\%$ for more than 20% of the total sleep time. Patients with a previous history of temporomandibular joint pain ($n = 4$) were not excluded. All subjects presented with complaints of excessive daytime hypersomnolence but no measures were used to quantify this symptom. None of the subjects had evidence on physical examination of right-sided congestive heart failure. The protocol was approved by the institutional review board of The University of Texas Medical Branch at Galveston and written informed consent was obtained from all subjects.

Nighttime Polysomnograms

All subjects were asked to perform three nighttime polysomnograms in the following order: polysomnogram 1 established the diagnosis of OSA and led to subject recruitment into the study, polysomnogram 2 ($n = 28$) was performed with the subjects using EMA, and polysomnogram 3 ($n = 12$) determined the site of pharyngeal airway closure. Body position was not controlled in the first two polysomnograms, but polysomnogram 3 was performed with the subjects in the supine position.

Using standard techniques, the following signals were recorded during the polysomnograms with a computer data acquisition and analysis system (Mallinckrodt, Plymouth, MN): C3A2 and O2A1 electroencephalogram (EEG), bilateral electro-oculograms, chin muscle activity, impedance plethysmography of the rib cage and abdomen (Respirace; Ambulatory Monitoring, Ardsley, NY), airflow at the nose and mouth (Neurosupplies, Waterford, CT), body position, oxygen saturation by pulse oximetry (Ohmeda, Louisville, CO), and presence or absence of tracheal breath sounds. The time interval between polysomnograms 1 and 2 was 112.3 ± 85.2 days (range 49 to 262). The time interval between polysomnograms 2 and 3 was 42.3 ± 17.1 d (range 23 to 84). Paired t tests found no statistically significant differ-

ences in body weight between polysomnograms 1 and 2 or between polysomnograms 2 and 3.

Oral Elastic Mandibular Advancement Device

After the diagnostic polysomnogram, the subjects were fitted with EMA (Frantz Design, Austin, TX) (Figures 1 and 2). The oral device consists of two plastic trays custom molded to the patient's maxillary and mandibular teeth. The trays remain securely in place by snapping into undercut areas of the teeth. The trays are made of a hard material which does not allow tooth movement and prevents the device from falling off during sleep. On the buccal side of the device, bilateral plastic button hooks are located on the maxillary tray at the level of the cuspids and on the mandibular tray at the molar region. Bite planes are located bilaterally on the occlusal surface of the mandibular tray at the molar region. The amount of bite opening was just sufficient to allow clearance of the upper and lower incisors during mandibular advancement. The amount of bite opening in the oral devices was not altered during the course of the study.

To activate the appliance, elastic straps are attached to the right and left pairs of button hooks so that the stationary maxilla pulls the mandible forward. The amount of mandibular advancement can be adjusted by varying the length and elasticity of the straps connecting the upper and lower dental trays. Straps of three different lengths (21 mm, 17 mm, and 13 mm) and three different elastic strengths (60, 70, and 80 durometers) were used.

Before performing polysomnogram 2, the subjects were instructed to wear the appliance at home during sleep. Initially they used the longest strap with the lowest elastic strength. The mandible was then progressively advanced by substituting elastics of shorter length and greater strength. Polysomnogram 2 was obtained at a mandibular advancement that eliminated or greatly reduced snoring and/or daytime hypersomnolence based on history from the patient and bed partner ($n = 19$), advancement equal to or greater than maximal voluntary advancement ($n = 5$), or the greatest advancement that could be tolerated by the subject owing to temporomandibular joint pain ($n = 4$). The latter four subjects were those who had a prior history of temporomandibular joint pain. History from the patient and/or bed partner was used to assess snoring and daytime hypersomnolence.

Gradual advancement of the mandible appeared to be important in preventing temporomandibular joint pain. When unilateral temporomandibular joint pain developed in a subject, it could usually be alleviated by removing the ipsilateral elastic strap for several days. When the joint pain was bilateral, the straps were changed to decrease

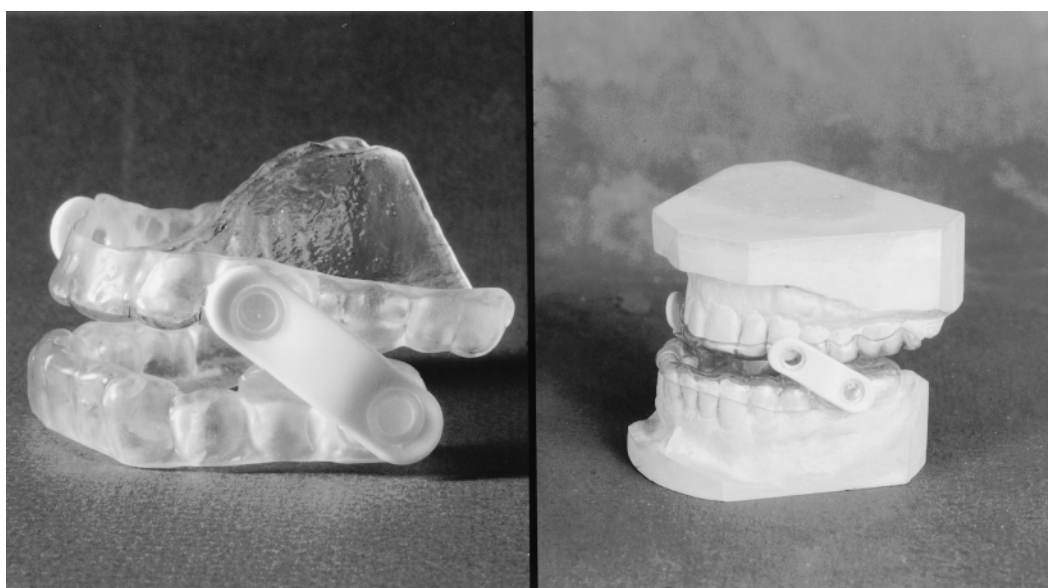


Figure 1. EMA shown off (left panel) and on (right panel) a cast of one subject's upper and lower teeth. Elastic straps attach the upper and lower trays. The maxilla pulls the mandible forward. Different degrees of advancement are achieved by using straps of different elasticity and length.

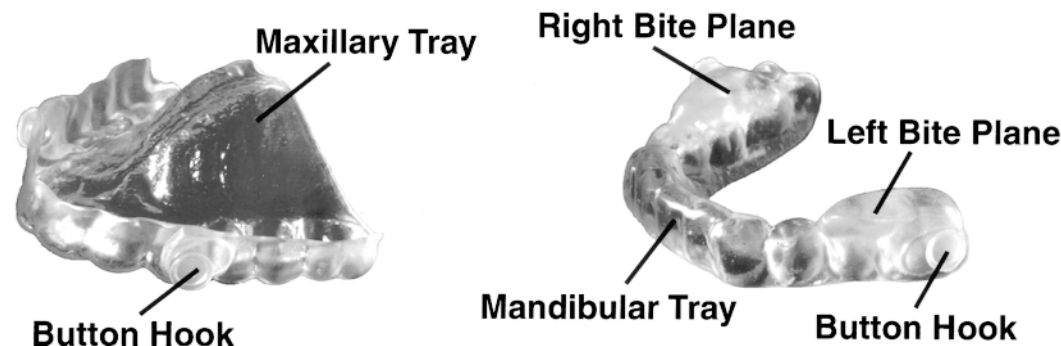


Figure 2. The maxillary (left panel) and mandibular (right panel) trays of EMA.

the amount of advancement. None of the subjects had to discontinue wearing the device because of temporomandibular joint pain. The subjects were instructed to replace the elastic straps every week to avoid stretching. The elastic straps were also replaced by the laboratory technician just prior to polysomnograms 2 and 3. Both the subjects and technicians were shown how to change the straps when the device was out of the subject's mouth. This was easily accomplished manually and required no special instruments. The amount of advancement and bite opening with the device using the elastics worn during polysomnograms 2 and 3 and maximal voluntary mandibular advancement were measured during wakefulness with a caliper and ruler.

Determination of Site of Airway Closure

Polysomnogram 3 was performed to determine the site of airway closure without the oral device. During the polysomnogram, the subjects were placed on nasal CPAP (Respironics, Murrysville, PA) to eliminate snoring and apneas and hypopneas. In addition to the standard polysomnographic parameters recorded during polysomnograms 1 and 2, nose mask pressure, nasal air flow, and oropharyngeal pressure were recorded during polysomnogram 3. Nose mask pressure was measured with a pressure transducer (Spectramed, Oxnard, CA) attached to a port in the nose mask. Nasal air flow was measured with a pneumotachograph (Hans Rudolph, Kansas City, MO) and symmetrical differential pressure transducer (Validyne, Northridge, CA) interposed between the nose mask and the expiratory port of the nasal CPAP circuit.

Oropharyngeal pressure, i.e., airway pressure below the level of the soft palate, was measured with a saline filled 8-Fr catheter attached to a pressure transducer (Spectramed). After topical anesthesia of one nasal passage with 1 ml of 1% lidocaine spray, the catheter was advanced transnasally into the oropharynx and secured to the nose. Correct position of the catheter below the level of the soft palate and above the tip of the epiglottis was confirmed at the beginning and end of the study by direct visualization of the oropharynx. The pharyngeal catheter was not bothering once in place. Pressure was measured at the side holes near the sealed distal tip of the catheter. Pressure was calibrated in cm H₂O with a water manometer and flow was calibrated in L/min with a rotameter (Fischer & Porter, Warminster, PA).

To determine if the site of pharyngeal airway closure was above or below the tip of the oropharyngeal catheter, airway closure was induced during stable periods of stages 2–4 non-rapid eye movement (NREM) sleep by abruptly lowering nose mask pressure to atmospheric pressure. To achieve this abrupt decrease in pressure, the tube connecting the nose mask to the machine was disconnected at the machine end. Airway closure was determined by the absence of fluctuations in nose mask pressure and absence of nasal air flow. The absence of respiratory-related fluctuations in oropharyngeal pressure during the induced apnea indicated that the site of closure was below the catheter tip, i.e., below the velopharynx. The presence of respiratory-related fluctuations in the pressure signal during the induced apnea indicated that the site of closure was above the catheter tip, i.e., at the velopharynx. Mask pressure was restored to control pressure after 3 to 5 occluded efforts. Approximately 5 min separated three consecutive trials. Trials associated with an arousal before restoration of nasal CPAP were eliminated.

Data Analysis

In a given subject, the polysomnograms were analyzed manually with the aid of computer software by the same polysomnographic technologist. The technologist scoring the polysomnograms was not informed whether or not the subject was wearing the oral device during the recording. Data in polysomnograms 1 and 2 from the entire sleep period and from the NREM and rapid eye movement (REM) sleep periods were used for statistical analysis. Change in a polysomnographic outcome parameter was calculated as (parameter value without device – parameter value with device). Percent change in a polysomnographic parameter was calculated as the change in the parameter divided by the parameter's value without the device. Depending on whether or not the data were normally distributed, the paired *t* test was used for within-group comparisons of the following polysomnographic outcome parameters with and without the oral device: AHI, apnea index (AI), hypopnea index (HI), desaturation index, minimal oxygen saturation, and the amount of sleep time spent below 90% oxygen saturation. Nonparametric tests were used to validate the paired *t* test results in cases where the normality test failed. The results were the same in both cases. A desaturation event was defined as a > 4% drop in oxygen saturation. Comparisons with *p* < 0.05 were considered statistically significant.

RESULTS

Amount of Mandibular Advancement and Bite Opening

The elastic straps used by the subjects during polysomnograms 2 and 3 advanced the mandible by 9.2 ± 3.3 mm (range 3.3 to 17.0) during wakefulness. This amount of mandibular advancement was $88.5 \pm 9.5\%$ (range 72 to 109) of maximal voluntary advancement. In three subjects, the device advanced the mandible further forward than maximal voluntary advancement. The devices increased bite opening by 11.5 ± 1.8 mm (range 8.8 to 16.5).

Polysomnographic Results with and without the Oral Device

No significant differences were present between polysomnograms 1 and 2 with regard to total sleep time, time in NREM sleep, time during sleep in a particular body position, or body weight. There was a significant difference (*p* = 0.03) in the amount of time in REM sleep between polysomnograms 1 and 2 (62.9 ± 29.1 min and 83.1 ± 33.6 min, respectively). In the analysis of the effect of the oral device on the polysomnographic data, the statistical results presented subsequently for the entire sleep period were the same as those obtained from NREM sleep or REM sleep, unless otherwise specified.

The effect of the oral device on AHI and other polysomnographic parameters is shown in Figures 3 and 4 and Table 1. For the entire group, EMA reduced the AHI from 52.6 ± 28.2 events/h (range 10.2 to 112.2) to 21.2 ± 19.3 events/h (range 0

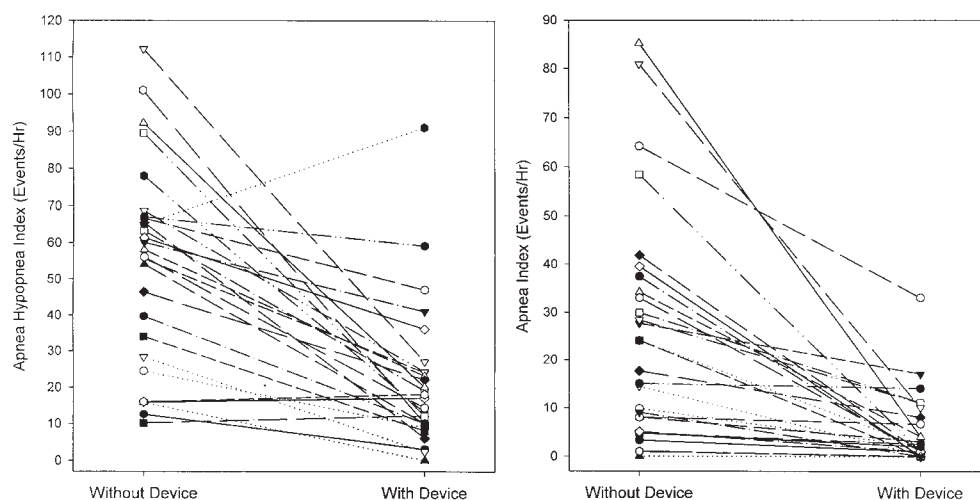


Figure 3. AHI and AI without and with EMA. The largest reductions in AHI and AI with the device occurred in subjects who had an AHI > 40 events/h without the device.

to 91), a $52.8 \pm 39.1\%$ improvement (range -40 to 100%) ($p < 0.001$). Similar statistically significant differences were present for AI, HI, and desaturation index. Nineteen subjects (68%) had at least a 50% reduction in AHI with the device. AHI with the oral device was < 15 events/h in 12 (43%) subjects and < 10 events/h in nine (32%) subjects. The AHI without the device in these subjects was 49.9 ± 28.1 and 41.7 ± 28.1 events/h respectively. Subjects with an AHI > 40 events/h on polysomnogram 1 had a $58.7 \pm 32.6\%$ improvement in AHI with the device, whereas subjects with an AHI < 40 events/h on polysomnogram 1 had a $40.3 \pm 50.1\%$ improvement. Five of the seven subjects who had greater than 80% improvement in AHI with the device had an AHI greater than 40 events/h without the device. Four subjects had a higher AHI with the device. Careful review of the data from these subjects found no explanation for this finding.

For the entire group, the change in AHI with the device was linearly related to the AHI without the device (Figure 5). A straight line fit to the data using least squares linear regression explained 60% of the variance (coefficient of determination [R^2] = 0.60). Similar statistically significant relationships

were present for other polysomnographic outcome parameters: AI ($R^2 = 0.90$), HI ($R^2 = 0.43$), minimal oxygen saturation ($R^2 = 0.41$), and amount of time spent below 90% oxygen saturation ($R^2 = 0.93$). All the p values of these linear regressions were < 0.001 . The change in a given polysomnographic outcome parameter (AHI, HI, AI, minimal oxygen saturation, time below 90% oxygen saturation) with and without the device was not related to the amount of mandibular advancement, amount of bite opening, neck circumference, body weight, and body mass index. The amounts of variance explained by these relationships were all less than 15% and the p values were > 0.05 . The amount of polysomnographic improvement with the device could not be predicted on the basis of whether the amount of advancement had been determined by symptomatic improvement or limited by maximal anatomic advancement or temporomandibular joint pain.

Effect of Site of Airway Closure on Efficacy of the Oral Appliance

The AHI of the 12 subjects who agreed to perform the third polysomnogram was 70.2 ± 24.7 events/h (range 28.3 to

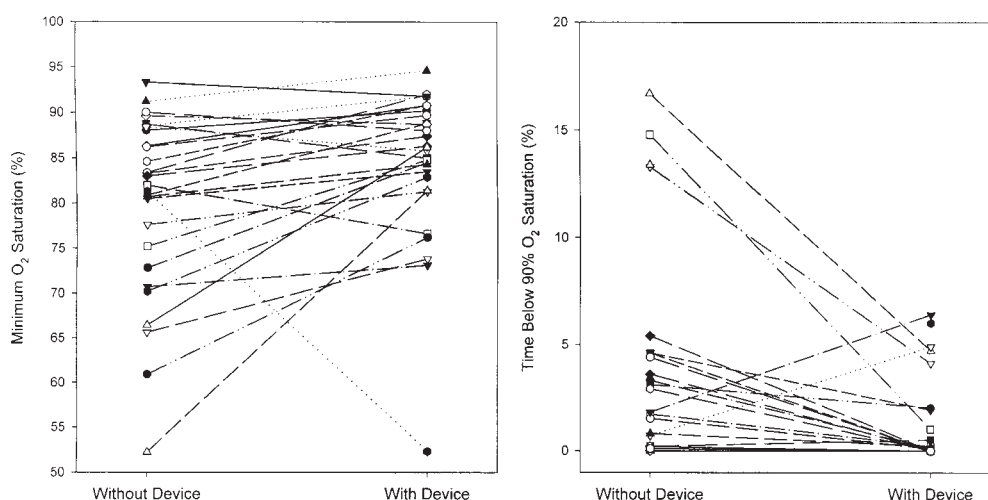


Figure 4. Minimal oxygen saturation and percent sleep time spent below 90% oxygen saturation without and with EMA.

TABLE 1
EFFECT OF EMA ON POLYSOMNOGRAPHIC PARAMETERS*

	Without Device	With Device
NREM and REM sleep		
AI, events/h	25.4 ± 23.6	4.8 ± 7.3 [†]
HI, events/h	27.3 ± 19.7	16.5 ± 17.8 [‡]
AHI, events/h	52.6 ± 28.2	21.2 ± 19.3 [†]
Minimal oxygen saturation, %	80.1 ± 10.0	84.4 ± 8.4 [‡]
Sleep time below 90% oxygen saturation, %	5.4 ± 8.9	2.3 ± 4.4 [§]
NREM sleep		
AI, events/h	22.5 ± 21.8	4.5 ± 7.8 [†]
HI, events/h	27.5 ± 20.4	14.0 ± 18.1 [§]
AHI, events/h	50.4 ± 29.5	18.5 ± 21.0 [†]
REM sleep		
AI, events/h	28.6 ± 26.5	10.4 ± 16.5 [†]
HI, events/h	24.5 ± 19.4	25.9 ± 18.5
AHI, events/h	53.1 ± 33.2	36.4 ± 24.1 [§]

* Values are expressed as mean ± SD.

[†] p < 0.001.

[‡] p < 0.05.

[§] p < 0.01.

112.2). The percent change in AHI with the device in these subjects was $69.9 \pm 25.2\%$ (range 11.9 to 91.9). Airway closure during the induced apnea was above the oropharynx, i.e., in the velopharynx, in nine of the subjects and below the oropharynx, i.e., in the hypopharynx, in three subjects. The percent change in AHI in subjects with pharyngeal airway closure at or below the velopharynx is shown in Figure 6. All three subjects with airway closure in the lower pharyngeal airway had a greater than 80% reduction in AHI with the device with the AHI decreasing to less than 6 events/h. Two of the 9 subjects with airway closure in the velopharynx had a similar therapeutic response, and seven of these subjects had at least a 58% reduction in AHI.

DISCUSSION

The results indicate that our new EMA significantly improves the polysomnographic severity of OSA and compares favorably with results reported in the literature for other mandibular advancement devices (1–15). A recent review by Strollo and Rogers (20) concludes that patients with mild OSA who do not tolerate therapy with nasal CPAP are good candidates for oral device treatment. This recommendation is supported by several previous reports that treatment success is inversely

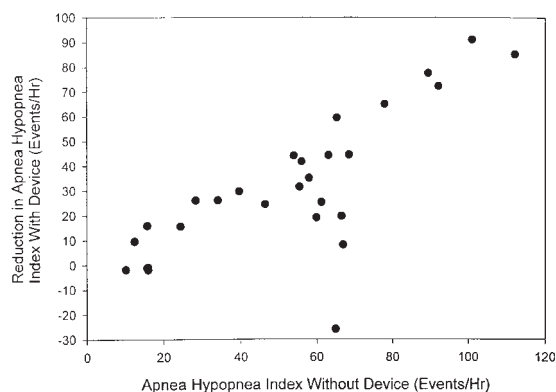


Figure 5. AHI without EMA versus change in AHI with the device, i.e., AHI without device – AHI with device. The change in AHI with the device was linearly related to the AHI without the device ($R^2 = 0.60$).

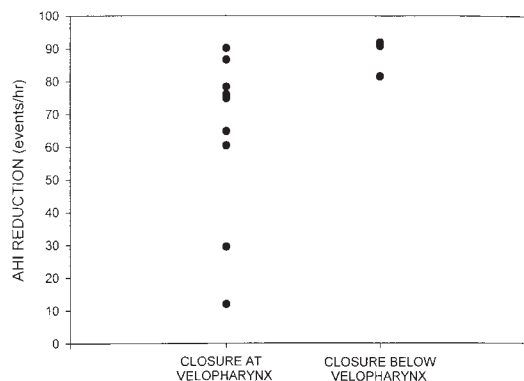


Figure 6. Percent change in AHI with EMA in subjects closing at or below the velopharynx. All three OSA subjects with airway closure below the velopharynx had a reduction in AHI to less than 6 events/h; however, many subjects with velopharyngeal closure also had a very favorable improvement in AHI.

related to the AHI without the device (3, 8, 11, 13). In contrast, we found a strong positive correlation between polysomnographic parameters of OSA severity and the amount of improvement in those polysomnographic parameters with the device. Our results indicate that subjects with moderate to severe OSA should not necessarily be excluded from oral device treatment.

As reported with other oral devices, the degree of polysomnographic improvement with EMA varied greatly among our OSA subjects. These different responses could not be explained on the basis of body weight, body height, neck circumference, or amount of mandibular advancement or bite opening with the device. It is possible that the amount of mandibular advancement and bite opening were not optimal at the time of polysomnographic testing with the device. Further advancement in the subjects whose AHI remained greater than 15 events/h with the device may have resulted in even greater reductions in AHI.

Another purpose of the current study was to determine whether the polysomnographic improvement in OSA subjects with the oral device was related to the site of pharyngeal airway closure. Previous investigators have shown that airway closure in OSA can occur in one or more pharyngeal segments: velopharynx, oropharynx, and/or hypopharynx (21, 22). We hypothesized that OSA subjects with closure of the pharyngeal airway below the level of the soft palate would have the greatest improvement in polysomnographic parameters with the oral device. Although all of the OSA patients with airway closure below the velopharynx had a reduction in AHI to less than 6 events/h, many subjects with velopharyngeal closure also had a very favorable improvement in AHI. Therefore, while airway closure in the hypopharynx predicts a very favorable outcome, airway closure in the velopharynx should not be used to exclude patients with OSA from this treatment alternative. These results are supported by the work of Isono and coworkers (23) reporting that manual mandibular advancement in anesthetized paralyzed subjects with OSA enlarges both the oropharynx and the velopharynx.

In the current study, pharyngeal pressure measurements during an induced occlusion were used to determine whether the site of closure occurred in the upper or lower pharyngeal airway. Pharyngeal airway closure during sleep is a complex phenomenon with primary and secondary sites of airway closure (21). The technique used in the current study was only able to distinguish airway closure above or below the tip of the

pressure catheter and therefore separated velopharyngeal closure from airway closure in lower pharyngeal segments. In addition, this technique detects the lowest site of airway closure, but, in the presence of lower pharyngeal airway closure, does not reveal what is happening at sites above the catheter tip. For example, subjects with lower pharyngeal airway closure may also have closure in the upper pharyngeal airway that would not be detected by the pressure measurement technique. Despite its acknowledged limitations, the pharyngeal pressure measurement used in this study to detect site of airway closure allowed us to determine that the oral device can be effective in OSA subjects with airway closure in the upper or lower pharyngeal airway.

It is important to note that the current results were obtained using one particular mandibular advancement device. Different results may have been obtained with use of other commercially available oral devices, given their great variability in design. By using elastics to pull the mandible forward, EMA allows lateral, vertical and anteroposterior movement of the mandible while advancing the mandible in a ventral and caudal direction. Allowing movement of the advanced mandible reduces the risk of temporomandibular joint pain and should improve patient compliance with chronic treatment. EMA with its elastic straps attached has an average volume displacement of 11 ml. This reduced bulk and the absence of projections or screws in the palate area decreases the amount of distortion of the oral cavity and optimizes the volume available for the tongue in the oral cavity. The comfort of EMA should enhance the compliance of patients in using the device on a nightly basis.

Like other oral devices, EMA has the ability to adjust the amount of mandibular advancement (3, 4, 7, 12). Whereas most other adjustable devices advance the mandible using a screw mechanism, EMA adjusts the amount of advancement by changing the elastic straps. As indicated in the current results, the amount of advancement differed among the subjects. Therefore, the ability to adjust the amount of mandibular advancement is an important advantage. The ability to vary the amount of advancement was also important when initiating the mandibular advancement treatment. It is our experience that many subjects, particularly those with a history of temporomandibular joint problems, are unable to tolerate immediate advancement to their "optimal" level. Subjects tolerate the device better if the mandible is progressively advanced over a several week period.

The results may have been influenced by the relatively long time interval between polysomnograms 1 and 2 that was needed to manufacture the custom fitted device and progressively advance the mandible to avoid temporomandibular joint pain. However, at the time of polysomnograms 1 and 2, no differences were noted in two factors that are known to cause night-to-night variability in the results: body weight and amount of time in a particular body position during the study. There was a significant increase in the amount of time spent in REM sleep stage during polysomnogram 2 which may have been caused by a REM rebound in noncompliant subjects. However, statistical analysis of the data with and without the device either over the entire sleep period or confined to NREM or REM sleep yielded similar results.

In summary, the current study shows the effectiveness of EMA in the treatment of OSA subjects. The device was well tolerated by the subjects and resulted in a mean reduction in AHI of $52.8 \pm 39.1\%$. Clinically acceptable polysomnographic improvements with the oral device were observed in subjects with mild to severe OSA and in subjects with pharyngeal closure in the hypopharynx or velopharynx. These results indicate that polysomnographic severity of the OSA and the site

of airway closure should not be used to exclude patients from this oral device treatment.

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