Evaluating Efficacy of Mandibular Advancement Device in Patients With Essential Hypertension and Obstructive Sleep Apnea

Manan Shah, MDS; Somil Mathur, MDS; Snehal Upadhyay, MDS; Rakesh Makwana, MDS

Department of Prosthodontics, Crown and Bridgework and Oral Implantology, Faculty of Dental Science, Dharmsinh Desai University, Nadiad, Gujarat, India

INTRODUCTION

Obstructive sleep apnea (OSA) is a common disease that usually occurs because of narrowing in the size of the upper airway during sleep and is believed to be a leading cause of transient asphyxia. This event leads to intermittent hypoxemia, sleep disturbances, daytime somnolence, brain arousals, and poor quality of life. As a result of the aforementioned events, the cardiovascular system may already be adversely affected by metabolic and neurohormonal disturbances. Epidemiologic data suggest that there is a strong association between OSA and systemic hypertension and that this association affects cardiovascular outcomes. The upper airway muscular activity is inhibited by an acute increase in blood pressure. This occurrence, together with volume overburden and its relocation to the upper body during rest, which can be experienced by individuals with hypertension, leads to pharyngeal edema and may clarify the connection among hypertension and OSA.

It is reported that continuous positive airway pressure therapy for OSA improves blood pressure of patients with hypertension. A few studies have depicted the antihypertensive effect resulting from mandibular advancement device (MAD) therapy on mild to moderate OSA.

Based on the apnea-hypopnea index (AHI) calculated from polysomnographic analysis, OSA can be categorized as mild (AHI = 5 to 15 events/hour), moderate (AHI = 15 to 30 events/hour), and severe (AHI = more than 30 events/hour). Many studies have revealed that use of MAD therapy is an effective treatment to reduce severity of mild to moderate OSA by 50%. MADs relieve snoring and sleep apnea by widening or opening the airway through anterior positioning of the mandible during sleep.

Many subjective and objective means of diagnosis of OSA have been reported to date. The STOP-Bang questionnaire includes both subjective and objective parameters and is used for screening of patients with significant risk of OSA.

Subjective parameters are used to assess the history of snoring, observed apnea, and tiredness whereas objective parameters includes body mass index, blood pressure, neck circumference, age, and sex of the participants. The Epworth Sleepiness Scale (ESS) is a subjective questionnaire to assess daytime sleepiness in participants with OSA. Polysomnography is the most common and gold standard objective analysis for diagnosis and monitoring of OSA that is noninvasive and does not require any radiation. It is possible to obtain reports of different parameters such as electrocardiography, electroencephalography, electromyography, electro-oculography, respiratory events, oxygen saturation, body...
movements, and snoring loudness, depending on level of polysomnography. Polysomnography can be done in the laboratory as well as at home. Polysomnography performed at home is also called a level 3 home sleep apnea test, which includes electrocardiography or heart rate monitoring, oxygen saturation, and at least two channels of respiratory movement.7,15,16

The current study was designed to evaluate the effect of MAD therapy on OSA severity in patients with essential hypertension. The null hypothesis was that the MAD would not have any effect on blood pressure or subjective and objective symptoms of the participants with OSA.

MATERIALS AND METHODS

The current prospective cohort study was conducted in the Department of Prosthodontics, Crown and Bridgework and Oral Implantology, Faculty of Dental Science, Dharmsinh Desai University, Nadiad. Twenty-five participants were screened for OSA with the help of a STOP- Bang Questionnaire. All participants underwent a level 3 home sleep test (ApneaLink Air, Martinsried, Germany) which uses portable monitors that allow sleep studies to be done at home (Figure 1). Participants who were not willing to undergo nasal continuous positive airway pressure therapy were selected for the study, and written informed consent was obtained from each participant. All participants filled out a demographic questionnaire which included age, sex, alcohol ingestion, smoking habits, and history of hypertension. Neck circumference (measured just below the prominence of the thyroid cartilage), body mass index and Mallampati scores were also recorded. Daytime sleepiness was assessed using the ESS. Participants with inadequate nasal airflow capacity, less than 7 mm protrusive jaw movement from maximum intercuspation, temporomandibular joint disorder, fewer than three healthy teeth per quadrant, severe periodontitis, and high caries index were excluded from the study.18

Maxillary and mandibular diagnostic impressions were made with irreversible hydrocolloid impression material (Imprint, DPI, India) and casts were poured using type 3 dental stone (Kalabhai, Mumbai, India) (Figure 3). In maximum intercuspal position (P1), two vertical lines were marked on casts: one from the buccal surface of the first premolar and molar to the buccal surface of the maxillary premolar and molar, respectively (Figure 3). A protrusive interocclusal record was made using aluminum-reinforced wax (Morsa, Bavaria, Germany) at 75% of the maximum mandibular protrusion (P2) (Figure 4). Seventy-five percent of maximum mandibular protrusion (P2) was recorded by measuring the distance between the labial surface of the maxillary incisor to the labial surface of the mandibular incisor with a vernier caliper. This record was transferred to casts and 75% of maximum mandibular
protrusion was verified by the distance between the two previously drawn vertical lines. Both casts were articulated on a mean value articulator using the interocclusal record. Wax for the MAD was made using modeling wax (Y-Dent, India) and processing was done using heat-polymerized acrylic resin (Trevlon, DENTSPLY, India) as per manufacturer instructions (Figures 5 and 6). Minor adjustments were carried out to eliminate processing errors, and verification of 75% of maximum mandibular protrusion was done. Following delivery of the MAD, participants were recalled after 24 hours for postinsertion adjustments. After 1 month of MAD therapy, all participants were again subjected to a level 3 home sleep apnea test with the MAD in position. Twenty-four-hour ambulatory blood pressure analysis and assessment of daytime sleepiness with ESS were performed. All the data were plotted and compared statistically using paired t test.

**RESULTS**

Demographic characteristics of patients with OSA are outlined in Table 1.

**Subjective assessment**

The ESS after MAD therapy (10.15 ± 2.06) was significantly low when compared to ESS before MAD therapy (ESS = 15.68 ± 2.80) (P < 0.05) Subjective assessment is outlined in Table 2.

**Objective assessment**

AHI (27.25 ± 16.67 events per hour) was significantly reduced after MAD therapy in comparison with AHI before MAD therapy (46.66 ± 15.02 events per hour) (Figure 7). Twenty-four-hour ambulatory mean systolic blood pressure (140.28 ± 15.8 mmHg) showed significant reduction following MAD therapy (130.24 ± 14.1 mmHg). Similarly, 24-hour ambulatory mean diastolic blood pressure was also reduced significantly, from 89.64 ± 10.4 mmHg to 85.29 ± 11 mmHg (Figures 8 and 9).

**DISCUSSION**

The goal of the current study was to evaluate the effect of MAD therapy in patients with essential hypertension and OSA. Use of the MAD reduces sleep apnea severity to half and is most effective in mild to moderate cases. The MAD dilates or opens the airway by repositioning the mandible and tongue during sleep, which is helpful in relieving snoring and OSA. OSA is closely associated with high blood pressure.19,20 This research was intended to determine whether the use of the MAD in affected individuals had an influence on blood pressure values. The pathogenesis of hypertension in OSA is affected by various factors, including load of non-positive intrathoracic pressure as a result of upper airway occlusion, recurrent arousals, reduction in stretch receptor stimulation of the lung, and temporary hypoxemia.21 In addition, multifaceted relationships with the boost of sympathetic nervous activity, inflammation, active enzyme, oxidative stress, and endothelial dysfunction are responsible for an increase in and variation of blood pressure.22,23 The antihypertensive effect of MAD therapy is not only because of the
Figure 7. Apnoea-Hypopnoea index (Before and after MAD therapy)

Figure 8. Systolic blood pressure (Before and after MAD therapy)

Figure 9. Diastolic blood pressure (Before and after MAD therapy)
Table 1. Demographic characteristic (mean ± standard deviation); (n=21) of patients with obstructive sleep apnea.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Statistics</th>
</tr>
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<tbody>
<tr>
<td>Gender : Male</td>
<td>15 (71.42%)</td>
</tr>
<tr>
<td>Male</td>
<td>6 (28.57%)</td>
</tr>
<tr>
<td>Age (Years)</td>
<td>50.04 ± 10.05 (27-65)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>160.64 ± 10.16 (149-170)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.05 ± 11.43 (60-90)</td>
</tr>
<tr>
<td>Smoking: Yes</td>
<td>15 (71.42%)</td>
</tr>
<tr>
<td>No</td>
<td>6 (28.57%)</td>
</tr>
<tr>
<td>Diabetes Mellitus: Yes</td>
<td>11 (52.38%)</td>
</tr>
<tr>
<td>No</td>
<td>10 (47.61%)</td>
</tr>
<tr>
<td>Hypertension: Yes</td>
<td>21 (100%)</td>
</tr>
<tr>
<td>No</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Snoring: Yes</td>
<td>21 (100%)</td>
</tr>
<tr>
<td>No</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Mallampati Score: 1</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Neck Circumference (inch)</td>
<td>16.05 ± 0.81 (15-18)</td>
</tr>
<tr>
<td>Apnoea Hyponea Index (AHI) (events/hour)</td>
<td>27.25 ± 16.67 (6-56)</td>
</tr>
</tbody>
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enhancement of negative intrathoracic pressure responsible for an effect on hemodynamics, but also because of improvement of arousal reaction by suppression of sympathetic activity.24,25 Significant reduction in blood pressure values in known participants was observed after 1 month of MAD therapy. MADs have the ability to alter dimensions of the upper airway by carrying out an anterior and inferior movement of the jaw that facilitates an increase in the dimension of the airway in the pharyngeal region.26 As a result of this movement, stabilization of the jaw and the hyoid bone takes place, which opens the airway and prevents the posterior rotation of these structures during the decubitus. It shows consequences for all the pharyngeal segments, though its main effect appears in the velopharyngeal area. Rigidity and swelling occurs between the anterior and posterior pharyngeal pillars. Ventral
displacement of the soft palate, and thereby an increase in the caliber of the lateral walls of the velopharyngeal area, are the main factors for reduction in snoring. Functional MADs enable the hyoid bone to occupy a more forward position. As a result of this, a new position of balance of the suprahyoid musculature is created, which in turn increases the permeability and volume of the upper airway. Because of attachment of palatoglossi muscles, lateral relocation of the pharyngeal fat pads from the airway occurs and the base muscles of the tongue move anteriorly, which reduces pharyngeal collapsibility in lateral dimension. In the current study, after 1 month of MAD usage the AHI was significantly reduced. These values are in correlation with the other studies. The ESS is a scale intended to measure daytime sleepiness. Tendency to doze off or fall asleep unintentionally during the day is termed excessive daytime sleepiness. Induction of sleepiness/fatigue retards performance capability due to increased phases of non-responding or delayed response time, increased reaction times, slow information processing, reduced vigilance, reduced precision about short-term memory, and accelerated decrements in performance with time on task. This leads to an increase in human error and risk for accidents. Cumulative evidence indicates that the probability of motor vehicle crashes and sleepiness are directly proportional. The current study used the ESS to subjectively assess participants’ daytime sleepiness for this type of treatment and it was found that the results were in agreement with other similar studies. Thus, the null hypothesis that the MAD would not have any effect on subjective and objective symptoms of the participants was rejected. Because the current study has limited sample size and shows short-term results, further studies with large sample size and long-term follow-up would be more helpful in understanding the complex relationship between OSA and essential hypertension.

**CONCLUSION**

On the basis of the findings and within the limitations of the current study, it can be concluded that use of MAD therapy improves objective and subjective symptoms at 75% of maximum intercuspation position. MAD usage in patients with OSA and hypertension can lead to reduction of blood pressure and AHI. This reduction may attribute to improvement of sleep quality and overall health of an individual.

**ABBREVIATIONS**

AHI, apnea-hypopnea index  
ESS, Epworth Sleepiness Scale  
MAD, mandibular advancement device  
OSA, obstructive sleep apnea

**REFERENCES**


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Address correspondence to: Manan Shah, MDS, shahman973@gmail.com

DISCLOSURE STATEMENT
The authors have no conflicts of interest to disclose.