

# A Preliminary Report on the Effect of a Mandibular Advancement Device on Obstructive Sleep Apnea Using Magnetic Resonance Imaging and Polysomnography

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This preliminary study investigated the effect of a mandibular advancement device on upper airway collapsibility in seven patients with obstructive sleep apnea. Overnight polysomnography and dynamic magnetic resonance imaging were performed at the retropalatal and retroglottal levels, and the apnea-hypopnea Index (AHI), anteroposterior and lateral distances, and airway volumes were recorded. The tests were repeated following a 3-month period of wearing a customized mandibular advancement device. A significant reduction in AHI (from 31 events per hour to 18.2 events per hour) and improvement in airway dimension at both the retropalatal and retroglottal levels were recorded, suggesting a baseline record for future studies with a larger patient sample. *Int J Prosthodont* 2012;25:613–618.

**O**bststructive sleep apnea (OSA) is a sleep disorder characterized by repetitive episodes of upper airway obstruction (apneas) and reduced airflow (hypopneas). It can be diagnosed using an overnight polysomnograph. The severity of OSA is expressed by the apnea-hypopnea index (AHI; total number of apnea and hypopnea events per hour of sleep) and categorized as follows: mild = 5 to 15, moderate = 16 to 30, and severe = > 30.

Continuous positive airway pressure therapy is the effective yet cumbersome treatment of choice for OSA. A simple, noninvasive alternative is oral appliance therapy, with mandibular advancement devices (MADs) cited as the most effective. These devices appear to increase upper airway caliber only at the retroglottal level; however, evidence of an increased retropalatal airway also exists. The MAD is expected to improve the anteroposterior dimension of the upper airway, but an increase in the lateral dimension has also been seen. Due to the intricate linkages between upper airway structures, the anatomical changes induced by an MAD are complex and require sophisticated recording technology. The aim of this study was to evaluate the effect of an MAD on upper airway collapsibility in OSA patients using both magnetic resonance imaging (MRI) and polysomnography.

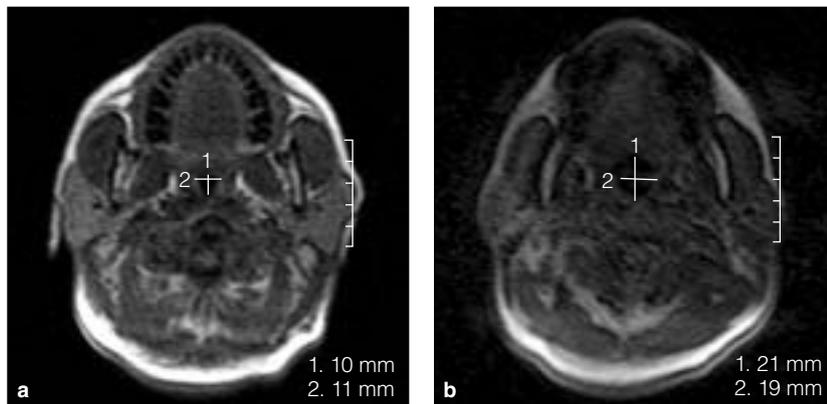
## Materials and Methods

In this preliminary study, a convenience sample of seven male OSA patients (age range: 25 to 65 years) was selected. A matched control group was not obtained due to the low number of patients. Likewise, the AHI classifications of mild, moderate, and severe were not followed because of the small sample size. Inclusion criteria were an AHI > 5 and absence of other local or systemic diseases. In accordance with American Academy of Sleep Medicine recommendations, the criterion for treatment success was an AHI < 5. All patients were recruited from Sri Ramachandra Medical Centre, Chennai, India, and provided informed consent following a thorough explanation of the proposed treatment and monitoring protocol. The Epworth sleepiness scale was used to subjectively diagnose OSA. This is a simple method for measuring daytime sleepiness in adults using a self-administered eight-item questionnaire that asks subjects to describe the chance that they will doze off in common, everyday situations (three-point scale: 0 = would never doze, 3 = high chance of dozing). The total score is calculated and interpreted as follows: normal = 0 to 10, borderline = 10 to 12, and abnormal = 12 to 24. This measurement was used for preliminary diagnosis of OSA, after which each subject underwent diagnostic polysomnography. Dynamic MRI of the upper airway was performed with a 1.5-T MR Imager (General Electric) during sleep to identify the site of obstruction. A localizer was taken in T1 sequence to select the sites. One midsagittal and two axial sections were taken. The two axial sections were at the C<sub>2</sub> vertebra (retropalatal) and between the C<sub>3</sub> and C<sub>4</sub> vertebra (retroglottal). Each image

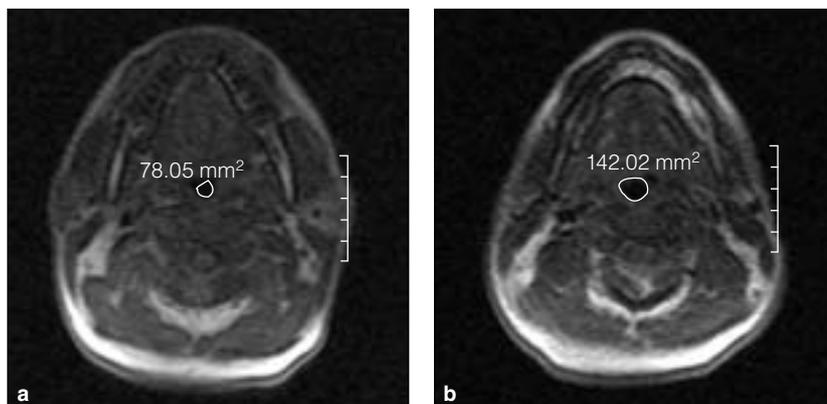
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**Fig 1** (a) Pretreatment and (b) posttreatment MRIs of the anteroposterior and lateral dimensions at the retropalatal level.



**Fig 2** (a) Pretreatment and (b) posttreatment MRIs of the cross-sectional area at the retroglottal level.

was repeated 45 times, each lasting 1 second. The anteroposterior and lateral diameter and area (Figs 1 and 2) were measured at inspiration and expiration at both levels with the aid of General Electric Advantage Workstation software (General Electric) in DICOM format. The MAD was customized for each patient at 75% of his maximum advancement (achieved via a Gothic arch tracing). After a 3-month wearing period during sleep, the tests were repeated with the appliance in place.

### Results

The mean AHI decreased from 31 events per hour before treatment to 18.2 per hour after treatment. An AHI < 10 per hour was achieved in 71.5% of subjects, while an AHI < 5 per hour was achieved in 57% of subjects. Dynamic MRI showed an increase in lateral dimension at the retropalatal and retroglottal levels at inspiration and expiration. The mean lateral increase was 30.8% retropalatally and 33.9% retroglottally at inspiration and 12.7% retropalatally and 26.9% retroglottally at expiration. The anteroposterior dimension showed no significant changes but did show more

improvement at the retroglottal region. Mean cross-sectional area improved at both regions (Table 1).

Analysis of variance was conducted with AHI as the independent variable and all other measurements as predictors (Table 2). A backward stepwise multiple linear regression was used to progressively exclude the least important variable. The final model included  $AP_{E,RP}$ ,  $Area_{E,RP}$ ,  $AP_{E,RG}$ , and  $LAT_{I,RG}$  ( $AP$  = anteroposterior;  $E$  = expiration;  $RP$  = retropalatal;  $RG$  = retroglottal;  $LAT$  = lateral; and  $I$  = inspiration), which accounted for 85% of the variability ( $R^2 = 0.851$ ,  $P < .001$ ) (Tables 2 and 3). Given the number of patients, a nonparametric correlation on pretreatment and posttreatment variables was executed, and the Spearman correlation coefficient indicated some significant correlated variables (Table 4).

### Discussion

A reduction of AHI from 31 events per hour to 18.2 per hour was achieved, which is comparable to a recent study by Lee et al,<sup>1</sup> who used a monoblock device with 60% advancement. In the present study, 71.5% of subjects achieved an AHI < 10 per hour, while 57%

**Table 1** Pre- and Posttreatment Polysomnography and Dynamic MRI Results

	Patient*					
	1	2	3	4	6	7
<b>Pretreatment</b>						
AHI (events/h)	7.7	49.2	5.6	25.0	18.9	36.0
AP <sub>I,RP</sub> (mm)	5.2	7.0	5.0	7.2	6.4	5.8
LAT <sub>I,RP</sub> (mm)	6.9	4.0	5.0	9.4	6.3	5.7
Area <sub>I,RP</sub> (mm <sup>2</sup> )	26.6	25.0	15.2	58.1	32.1	26.3
AP <sub>E,RP</sub> (mm)	11.3	10.0	6.0	7.5	9.1	8.3
LAT <sub>E,RP</sub> (mm)	15.3	11.0	9.0	11.7	13.7	12.4
Area <sub>E,RP</sub> (mm <sup>2</sup> )	148.0	65.6	47.1	75.2	104.8	85.8
AP <sub>I,RG</sub> (mm)	5.0	10.0	11.0	12.9	12.0	10.9
LAT <sub>I,RG</sub> (mm)	6.9	11.0	12.0	15.3	12.2	11.0
Area <sub>I,RG</sub> (mm <sup>2</sup> )	23.1	78.1	98.2	164.9	112.0	91.7
AP <sub>E,RG</sub> (mm)	8.0	13.0	13.0	13.0	14.7	13.3
LAT <sub>E,RG</sub> (mm)	31.0	15.0	18.0	27.7	19.9	18.0
Area <sub>E,RG</sub> (mm <sup>2</sup> )	172.9	164.8	178.8	243.8	228.6	187.1
<b>Posttreatment</b>						
AHI (events/h)	0.0	22.3	3.6	5.0	7.6	5.5
AP <sub>I,RP</sub> (mm)	6.0	9.0	4.0	10.3	6.0	5.5
LAT <sub>I,RP</sub> (mm)	9.0	11.0	5.0	10.1	10.5	9.5
Area <sub>I,RP</sub> (mm <sup>2</sup> )	30.6	73.7	16.7	100.0	52.9	43.3
AP <sub>E,RP</sub> (mm)	9.0	21.0	7.0	9.0	8.7	7.8
LAT <sub>E,RP</sub> (mm)	21.0	19.0	9.0	12.0	12.6	16.5
Area <sub>E,RP</sub> (mm <sup>2</sup> )	169.7	334.8	60.2	116.2	104.3	123.4
AP <sub>I,RG</sub> (mm)	10.0	13.0	8.0	13.0	12.6	11.4
LAT <sub>I,RG</sub> (mm)	13.0	15.0	14.0	15.5	15.6	14.1
Area <sub>I,RG</sub> (mm <sup>2</sup> )	117.2	142.0	81.4	167.0	155.9	127.6
AP <sub>E,RG</sub> (mm)	13.0	18.0	14.0	13.7	15.4	13.9
LAT <sub>E,RG</sub> (mm)	25.0	22.0	25.0	28.3	26.3	23.8
Area <sub>E,RG</sub> (mm <sup>2</sup> )	236.5	252.7	218.9	251.0	267.6	219.1

AP = anteroposterior; LAT = lateral; RP = retropalatal; RG = retroglossal; E = expiration; I = inspiration.

\*Patient 5 removed from table for comparison purposes.

achieved an AHI < 5 per hour. The effect of an adjustable device on severe apneic patients was not tested; however, the authors believe that in such patients, an adjustable device may be more effective.<sup>2</sup> One of the seven patients showed increased posttreatment AHI, which may be attributed to his high body mass index (32.23 kg/m<sup>2</sup>), severe pretreatment AHI (74.3 per hour), and significant collapse of the lateral pharyngeal walls at the retropalatal and retroglossal regions, with thickening of the parapharyngeal fat pads (Fig 3). Recent studies have found that obesity and central adiposity can lead to crowding of pharyngeal lumen and increased surrounding tissue pressure, thus increasing airway collapsibility. These studies have

also demonstrated that mandibular advancement can decrease collapsibility in lean subjects but not in obese subjects, possibly due to the adiposity in parapharyngeal fat pads, which leads to collapse of lateral pharyngeal structures (as seen in this patient).<sup>3</sup> Rodenstein et al<sup>4</sup> found that the pharynx in OSA subjects was circular or elliptical, with the long axis oriented in the sagittal plane as opposed to normally in the coronal plane. It is possible that the pharyngeal walls were pulled closer together with the MAD, thus adding to the obstruction and negating the effects of the appliance. Additionally, subjects who showed improvement with the device had reduced transverse dimension of the upper airway at the retropalatal level,

**Table 2** Backward Linear Regression Models

Model	R	R <sup>2</sup>	Adjusted R <sup>2</sup>	Standard error
1	0.99	0.98	0.79	12.36
2	0.99	0.98	0.85	10.47
3	0.98	0.96	0.81	11.65
4	0.98	0.96	0.85	10.31
5	0.98	0.95	0.88	9.35
6	0.97	0.94	0.87	9.77
7	0.95	0.91	0.83	10.97
8	0.94	0.89	0.82	11.41
9*	0.92	0.85	0.79	12.48

\*Predictors: AP<sub>E,RP</sub>, Area<sub>E,RP</sub>, LAT<sub>I,RG</sub>, AP<sub>E,RG</sub>.

**Table 3** AHI Predictor Model Based on Backward Regression

	Unstandardized coefficients		β	t	P
	B	Standard error			
Constant	-28.62	24.02	-	-1.19	.26
AP <sub>E,RP</sub>	5.41	2.80	0.83	1.93	.09
Area <sub>E,RP</sub>	-0.34	0.14	-0.94	-2.47	.04
LAT <sub>I,RG</sub>	-4.05	1.94	-0.42	-2.09	.07
AP <sub>E,RG</sub>	6.33	1.65	0.74	3.83	.00

AP = anteroposterior; LAT = lateral; RP = retropalatal; RG = retroglossal; E = expiration; I = inspiration.

**Table 4** Nonparametric Correlation Analysis (Spearman)

	AHI	AP <sub>I,RP</sub>	LAT <sub>I,RP</sub>	Area <sub>I,RP</sub>	AP <sub>E,RP</sub>	LAT <sub>E,RP</sub>	Area <sub>E,RP</sub>	AP <sub>I,RG</sub>	LAT <sub>I,RG</sub>
<b>Pretreatment</b>									
AHI	1	0.8	0.1	0.3	0.5	0.1	0.3	0.1	-0.2
AP <sub>I,RP</sub>		1	0.5	0.7	0.4	0.2	0.4	0.6	0.1
LAT <sub>I,RP</sub>			1	0.9*	0.2	0.6	0.6	0.6	0.1
Area <sub>I,RP</sub>				1	0.3	0.5	0.6	0.7	0.2
AP <sub>E,RP</sub>					1	0.8	0.8	-0.3	-0.8
LAT <sub>E,RP</sub>						1	1*	-0.1	-0.6
Area <sub>E,RP</sub>							1	0	-0.5
AP <sub>I,RG</sub>								1	0.7
LAT <sub>I,RG</sub>									1
Area <sub>I,RG</sub>									
AP <sub>E,RG</sub>									
LAT <sub>E,RG</sub>									
Area <sub>E,RG</sub>									
<b>Posttreatment</b>									
AHI	1	-0.1	0.2	-	0.1	-0.3	-0.2	0.6	0.6
AP <sub>I,RP</sub>		1	0.8	0.9*	0.9*	0.5	0.7	0.8	0.4
LAT <sub>I,RP</sub>			1	0.9*	0.7	0.5	0.6	0.7	0.5
Area <sub>I,RP</sub>				1	0.7	0.4	0.6	0.8	0.6
AP <sub>E,RP</sub>					1	0.6	0.7	0.7	0.3
LAT <sub>E,RP</sub>						1	0.9*	0.1	-0.3
Area <sub>E,RP</sub>							1	0.3	-0.2
AP <sub>I,RG</sub>								1	0.8
LAT <sub>I,RG</sub>									1
Area <sub>I,RG</sub>									
AP <sub>E,RG</sub>									
LAT <sub>E,RG</sub>									
Area <sub>E,RG</sub>									

AP = anteroposterior; LAT = lateral; RP = retropalatal; RG = retroglossal; E = expiration; I = inspiration.

\*Significant correlation.

**Fig 3** Sagittal view of the pharynx showing obstruction sites in two different patients. **(a)** Negative response to MAD; **(b)** positive response to MAD.



	Area <sub>I, RG</sub>	AP <sub>E, RG</sub>	LAT <sub>E, RG</sub>	Area <sub>E, RG</sub>
	-0.3	0.6	-0.7	0.1
	0.1	0.5	-0.4	0.5
	0.1	0.2	0.5	0.7
	0.2	0.3	0.3	0.8
	-0.8	0.2	-0.2	-0.3
	-0.5	0.2	0.4	0.1
	-0.5	0.4	0.2	0.3
	0.7	0.5	0	0.9*
	1*	0.1	0.2	0.6
	1	0.2	0.2	0.7
		1	-0.5	0.5
			1	0.3
				1
	0.6	0.9*	0.2	0.8
	0.4	-0.4	-0.2	0.2
	0.3	-	-0.5	0.2
	0.4	-0.3	-0.2	0.1
	0.4	-0.1	-0.2	0.4
	-0.4	-0.5	-0.7	-0.2
	-0.2	-0.4	-0.8	-0.2
	0.8	0.3	0.1	0.6
	0.8	0.4	0.4	0.7
	1	0.3	0.6	0.8
		1	0.1	0.6
			1	0.5
				1

with mild changes at the retroglottal level (Fig 3). This stands in contrast to the patient without improvement, who had significant airway collapse at both levels. A recent study by Chan et al<sup>5</sup> showed that the MAD increases airway volume predominantly at the retropalatal level. These factors may explain the failure of the MAD in one patient but its success in the others. Johal and Battage<sup>6</sup> suggested that mandibular advancement is of little use with retropalatal obstruction; however, 85.7% of subjects with retropalatal obstruction in this study showed marked improvement with the MAD. Further, as reported by previous studies, significant airway increase in the lateral dimension was seen, possibly due to the connection between the tongue and soft palate through the palatopharyngeal and palatoglossus arch.<sup>7</sup> The nonparametric analysis suggested that the MAD can act simultaneously on the lateral dimensions of both the retropalatal and retroglottal regions, with a significant effect on the anteroposterior dimension in the retroglottal region only (Table 3).

The limitations of the study were as follows: (1) the device was nontitrable; (2) the MAD was designed with a fixed vertical opening of 2 mm anteriorly, whereas a higher vertical opening may have been more effective; and (3) AHI classifications of mild, moderate, and severe were not followed because of the small number of subjects. The authors acknowledge that this should be considered a preliminary study due to the small sample size and lack of a control group.

## Conclusion

The results suggest that an MAD appears to be effective in both the retropalatal and retroglottal regions. This preliminary study could provide baseline information for future comparative studies.

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### Literature Abstract

#### Surgical management of bisphosphonate-related osteonecrosis of the jaw in oncologic patients: A challenging problem

This study analyzed the type of surgical management and its outcome in a patient cohort suffering from bisphosphonate-related osteonecrosis of the jaw (BRONJ). One hundred forty-two patients (95 females, 47 males) with BRONJ were included in the study. Patients had previous or ongoing intravenous bisphosphonate treatment for various oncologic diseases with a mean treatment duration of 37.1 months. The mandible was affected by BRONJ for 58% of patients, while the maxilla was affected in 27% of patients, and both jaws were affected in 15% of patients. All except 2 patients had minor dental surgical procedures prior to the onset of BRONJ: 86% of patients had surgical treatment comprising transoral sequestrectomy in the exposed bone area and tension-free soft tissue closure, 64% of patients had continuity mandibular resection with immediate rigid fixation using titanium reconstruction plates employing a submandibular approach, and 14% of patients had soft tissue reconstruction with a myofascial flap from the mylohyoid muscle. One patient received a fasciocutaneous vascularized graft to close a soft tissue defect. No patient underwent vascularized bone reconstruction. The authors noted that 40% of treated patients required additional surgical interventions due to refractory BRONJ. Conservative treatment, which includes intensive oral irrigation with chlorhexidine and antibiotic medications, was effective in 14% of patients. The authors concluded that presently, there is no consensus on the extent of surgical resection and reconstruction in cases of BRONJ, and there is limited evidence for vascularized bone reconstruction in BRONJ patients.

**Eckardt AM, Lemound J, Lindhorst D, Rana M, Gellrich NC.** *Anticancer Res* 2011;31:2313–2318. **References:** 52. **Reprints:** Dr André M. Eckardt, Department of Cranio-Maxillofacial Surgery, Hannover Medical School, Carl-Neuberg-Strasses 1, 30625 Hannover, Germany. Fax: +49 5115328879. Email: eckart.andre@mh-hannover.de—Simon Ng, Singapore

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