ORIGINAL ARTICLES

http://dx.doi.org/10.15331/jdsm.4630

The Effects of an Oral Appliance in Obstructive Sleep Apnea Patients with Prehypertension

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Study Objective: The present study was aimed at estimating the effect of oral appliance treatment on the blood pressure of a cohort of obstructive sleep apnea (OSA) patients on a short-term (3 months) and long-term (1 year) basis and also to evaluate changes in the apnea-hypopnea index (AHI), snoring index (SI) and overall impact on the sleep apnea quality of life (SAQOL).

Methods: 37 patients who fulfilled the study criteria, underwent polysomnography before treatment with oral appliance, and then at 3 months and at 1 year after insertion of the oral appliance. Both systolic and diastolic blood pressure was recorded before treatment, at 3 months, and 1 year after insertion of oral appliance.

Results: A decrease of 9.35% in systolic blood pressure (SBP) and 11.04% in diastolic blood pressure (DBP) of the patients was recorded after 3 months of continuous use of the oral appliance. Snoring index (SI) and AHI decreased by 80.31% and 83.93%, respectively, at the end of this period. A phenomenal increase in sleep apnea quality of life scores (183.9%) was observed in this time interval. The long-term efficacy of oral appliance use was evaluated at 1 year. There was a decrease of 12.16% in SBP and 14.01% in DBP and a decrease of 82.52% in AHI and 89.77% in SI scores. The level of improvement in SAQOL was 240% at the end of a year.

Conclusion: There was a marked improvement in all symptoms observed, i.e., blood pressure, SI, and AHI indices and sleep apnea quality of life (after 3 months), which continued even on a long-term basis (at 1 year).

Keywords: obstructive sleep apnea, mandibular advancement device, apnea-hypopnea index, snoring index, sleep apnea quality of life

Citation: Bhushan A, Tripathi A, Gupta A, Tripathi S. The effects of an oral appliance in obstructive sleep apnea patients with prehypertension. *Journal of Dental Sleep Medicine* 2015;2(2):37–43.

Obstructive sleep apnea (OSA) syndrome is a serious condition that afflicts a substantial number of individuals. It is characterized by disruption of normal sleep architecture due to complete or partial obstruction of respiratory airflow. Airflow obstruction results in reduction of blood oxygen saturation also known as hypoxemia. It produces arousal in an attempt to reopen the airway. These recurring arousals disrupt the sleep architecture, leading to excessive daytime somnolence and poor quality of life.[1] The worldwide OSA prevalence rates in adults ranges between 3.5% and 27%.[2–3] In India, its prevalence ranges between 3% and 28% in men and 2.2% and 16% in women.[4–5]

It is well documented that OSA is associated with systemic hypertension, pulmonary hypertension, cardiac arrhythmia, ischemic heart disease, and stroke.[6–11] Hypertension has been shown to occur in 28% to 57% of OSA patients, and there is a positive correlation between blood pressure and severity of apnea.[12–15] Systemic blood pressure may increase by 20% immediately after an apneic episode and then rapidly fall to normal values after onset of respiration.[16,17] Changes in blood pressure during the night may also influence morning blood pressure, which is usually higher in patients with OSA than normal subjects.[18]

During the last two decades there has been an increased interest in oral appliance as a treatment modality for OSA.[19] The oral appliance is used during night; it protrudes the mandible and thereby opens the airway. The oral appliance is easy to use as it does not require electrical power, does not make any noise, and crossover studies have shown that patients prefer oral appliance therapy to CPAP, which leads to good compliance.[20]

The aim of this study was to estimate the effect of oral appliance treatment of OSA on blood pressure for short-term (3 months) and long-term (1 year) follow-up and to evaluate whether oral appliance treatment effected a decrease in apnea-hypopnea index (AHI) and snoring index (SI) and brought about an improvement in the sleep apnea quality of life (SAQOL).

METHODS

The present study was conducted in the Department of Prosthodontics, Saraswati Dental College & Hospital, Lucknow, in collaboration with Department of Pulmonary Medicine, King George's Medical University, Lucknow. Prior approval of the institutional ethical committee (SDC-IHEC approval no. 018) had been obtained.

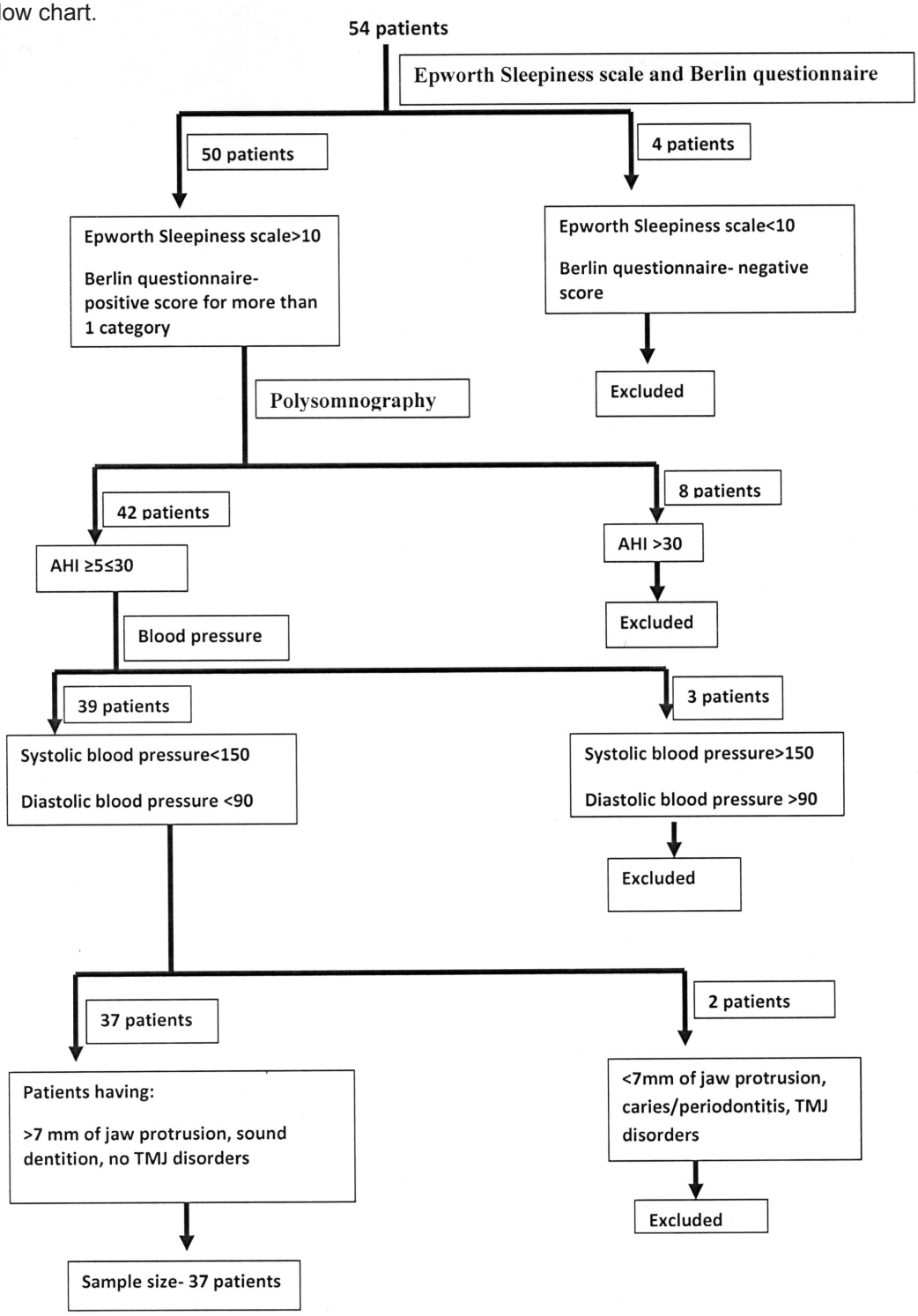
The inclusion criteria were:

1. OSA verified by somnographic evaluation (defined as apnea-hypopnea index > 5 < 30 per hour) and ≥ 2 of the following symptoms: daytime sleepiness, snoring, witnessed apneas, and fragmented sleep.
2. At least 7 mm of sustainable protrusive jaw movement from the position of maximum intercuspation.[21]

The exclusion criteria were:

1. More than 1 missing tooth per quadrant (excluding the third molar) that could minimize retention for the mandibular protruding device.

Figure 1—Flow chart.



2. Substantial evidence of TMJ disorders including pain, significant joint crepitation, restricted mouth opening, or sites of muscle tenderness in the masseter or temporalis region.[22]
3. Severe caries and/or compromised periodontal status, which would not allow prolonged use of mandibular protruding device.
4. Systolic blood pressure > 150 mm Hg or diastolic blood pressure > 90 mm Hg.

Thirty-seven dentulous OSA patients (25 males; 12 females; age 41 ± 4 years; BMI 22 ± 5; AHI 5–30), who volunteered and provided written informed consent were included in the study. The initial selection of the patients was based on subjective evaluation of the symptoms according the Epworth Sleepiness scale and Berlin questionnaire. Patients underwent overnight polysomnography to confirm their actual status before being included as study subjects (Figure 1). Five variables were assessed before treatment, at 3 months and at 1 year after

wearing mandibular advancement device: systolic blood pressure (SBP), diastolic blood pressure (DBP), SI, AHI, SAQOL.

All patients were given custom-made mandibular advancement devices (MAD) as described by Napankangas et al.[22] The range of mandibular advancement was set to 50% of maximum protrusive movement (mean 7 mm) since it was the maximum comfortable protrusion that was achieved with this mandibular advancement device.[22]

Blood pressure was measured between 09:00 and 11:00 and was measured before treatment, at 3 months, and at 1 year. All measurements (SBP and DBP) were done by the same operator using sphygmomanometer. During the clinical blood pressure measurement, the patients were reclined in a supine position with the arm at heart level. Three readings were taken at each sitting and averaged to obtain a single value for SBP and DBP, respectively.

Polysomnography

Overnight polysomnography (S-7000, Cogent technologies, EMBLA System Inc) included electroencephalograms (EEG: C3-A2, C4-A1, O2-A1, O3-A2), bilateral electro-oculogram (ROC, LOC), chin and leg electromyogram (EMG), nasal airflow, thoracic and abdominal movements, electrocardiogram (ECG), and body position recorders. AHI was calculated with the help of Somnologica studio software. The apnea episodes were defined as complete cessation of airflow for ≥ 10 s; hypopnea was defined as ≥ 50% reduction in oronasal airflow accompanied by a reduction ≥ 4% oxygen saturation calculated by pulse oximetry.[23] AHI was determined by the frequency of these events per hour during sleep time based on the results of the overnight polysomnography. Recorded polysomnographic data were cross-checked manually for apneas and hypopnea events.

For grading of snoring intensity, the bed partner snoring evaluation index tool[24,25] was used (Figure 2). Sleep apnea quality of life index (SAQLI)[26] was used to assess the SAQOL of the patients (Figure 3).

To determine the course of analysis, all data obtained were subjected to test of normality using Kolmogorov-Smirnov test. The analytical plan was dependent on the symmetry of data

Figure 2—Snoring index (SI).

Evaluation of snoring as reported by bed partner (circle one):

0 1 2 3 4 5 6 7 8 9 10

0-3 : Occasional soft snoring - not bothersome to bed partner

4-6 : Persistent snoring - bothersome to bed partner

7-9 : Persistent loud snoring - frequently annoying bed partner

10 : Heroic snoring - continuous, loud snoring not tolerated by bed partner

Figure 3—Sleep apnea quality of life index (SQQLI).

Before and after therapy

Please score the following questions according to the following:

Very large amount = 1, Large amount = 2, Moderate to large amount = 3, Moderate amount = 4, Small to moderate amount = 5, A little = 6, Not at all = 7

S. No.	Questions	Before	After
1.	How much have you had to push yourself to remain alert during a typical day (e.g., work, school, childcare, housework)?		
2.	How often have you had to use all your energy to accomplish your most important activity (e.g., work, school, childcare, housework)?		
3.	How much difficulty have you had finding the energy to do other activities (e.g., exercise, relaxing activities)?		
4.	How much difficulty have you had fighting to stay awake?		
5.	How much of a problem has it been to be told that your snoring is irritating?		
6.	How much of a problem have frequent conflicts or arguments been?		
7.	How often have you looked for excuses for being tired?		
8.	How often have you not wanted to do things with your family and/or friends?		
9.	How often have you felt depressed, down, or hopeless?		
10.	How often have you been impatient?		
11.	How much of a problem has it been to cope with everyday issues?		
12.	How much of a problem have you had with decreased energy?		
13.	How much of a problem have you had with fatigue?		
14.	How much of a problem have you had waking up feeling unrefreshed?		

Table 1—Test for normality (Kolmogorov-Smirnov Test).

	SDB	DBP	AHI	SI	SAQOL
Pretreatment Normality Test	0.119	0.168	0.160	0.218	0.276
Posttreatment Normality Test (3 months)	0.103	0.163	0.154	0.194	0.272
Posttreatment Normality Test (1 year)	0.144	0.118	0.141	0.255	0.403

SDB, systolic blood pressure; DBP, diastolic blood pressure; AHI, apnea-hypopnea index; SI, snoring index; SAQOL, sleep apnea quality of life.

Table 2—Before treatment correlation between blood pressure, AHI, SI, and SAQOL (Kendall's tau b "τ-b").

	SBP	DBP	AHI	SI	SAQOL
SBP	1.00	0.62**	0.72**	0.62**	−0.36
DBP	–	1.00	0.73**	0.62**	−0.47*
AHI	–	–	1.00	0.79**	−0.31
SI	–	–	–	1.00	−0.26
SAQOL	–	–	–	–	1.00

**p < 0.01; *p < 0.05. SDB, systolic blood pressure; DBP, diastolic blood pressure; AHI, apnea-hypopnea index; SI, snoring index; SAQOL, sleep apnea quality of life.

obtained. It was planned that if all the parameters recorded had a normal distribution, a parametric plan would be followed; otherwise a nonparametric plan was the choice. Data were analyzed using Statistical Package for Social Sciences, version 15.0. Wilcoxon signed rank test was used to evaluate before-after changes. A P value < 0.05 indicated statistically significant difference.

RESULTS

On evaluating the distribution for normality (before treatment), only blood pressure, AHI, SI were found to be normal and hence a nonparametric analysis plan was adopted (Table 1).

Table 2 shows the correlation between blood pressure, AHI, SI and SAQOL. This correlation was explored using the Kendall's tau-b (nonparametric variant of Pearson correlation coefficient). SBP had a moderate positive correlation with DBP (τ = 0.62) and SI (τ = 0.62) whereas the correlation with SBP and AHI was positive and strong (τ = 0.72). However, the correlation between SBP and SAQOL was inverse and mild (τ = −0.36). DBP had moderate positive correlation with SBP (τ = 0.62) and snoring index (τ = 0.62) and a strong correlation with AHI (τ = 0.73). The correlation between DBP and SAQOL was inverse and mild (τ = −0.47).

AHI had a strong positive correlation with SBP (τ = 0.72), DBP (τ = 0.73), and snoring index (τ = 0.79), and a mild inverse correlation (τ = −0.31) with SAQOL scores. Snoring index had a moderate positive correlation with SBP and DBP (τ = 0.62) and a strong correlation with AHI (τ = 0.79). The correlation between snoring index and SAQOL was inverse and weak in nature (τ = −0.26).SAQOL had mild inverse correlation with SBP, DBP, and AHI (τ = −0.3 to −0.5) and a weak inverse correlation with snoring index (τ = −0.26) (Table 2).

A significant reduction in mean SBP, DBP, AHI, and SI was observed after 3 months of wearing oral appliance (P ≤ 0.001), whereas a significant increase in sleep apnea quality of life score was observed (P = 0.001). Among all variables, maximum proportional change was observed in SAQOL scores where an increase of 139.1% ± 113.32% (mean ± standard deviation [SD]) (from 2.3 preoperative to 5.5 after 3 months) was observed, whereas minimum change was observed in DBP (mean ± SD decrease of 3.27% ± 3.86%; Table 3).

A significant reduction in mean SBP, DBP, AHI, and SI was observed at 1 year treatment interval as compared to pretreatment values (P = 0.001), whereas a significant increase in SAQOL scores was observed (P = 0.001) after 1 year of wearing oral appliance. Among all variables, maximum proportional change was observed in SAQOL scores, where an increase of 186.9% ± 134.45% (from 2.3 preoperative to 6.6 after 1 year) was observed; minimum change was observed in SBP (Table 4).

DISCUSSION

The present study was aimed at estimating the effect of oral appliance treatment on the blood pressure on a cohort of OSA patients on a short-term (3 months) and long-term (1 year) basis and also to evaluate changes in the AHI index, snoring index, and the overall impact on the sleep apnea quality of life. For this, 37 prehypertensive dentulous patients falling into the category of mild-to-moderate OSA with sound dentition (caries/periodontitis free) and without any sign or symptoms of temporomandibular disorder were selected. Prehypertension is a systolic pressure from 120 to 139 mm Hg or a diastolic pressure from 80 to 89 mm Hg.[27] Prehypertension can be corrected with lifestyle modification and does not require hypertensive drug therapy; to eliminate the bias of hypertensive drug, only those patients that were not taking any hypertensive medication and falling into the prehypertension category were selected.[27] All these patients were given custom-made mandibular advancement devices.[22] Oral appliances are successful in individuals with mild-to-moderate OSA but have been proven less effective for severe cases.[28] In one study comparing CPAP to dental appliance in mild-to-moderate OSA, dental appliances decreased AHI from 21 to 14, compared to a decreased AHI of 5 in patients using CPAP.[29] Oral appliances serve to advance the mandible within a range of physiological limits along the path of condylar guidance and bring about an increase in the volume of the hypopharynx. Any temporomandibular disorder (including pain, significant joint crepitation, restricted mouth opening, or sites of muscle tenderness in the masseter or temporalis region) would prevent smooth gliding of the mandible; hence patients having TMDs and AHI > 30 were excluded.[22]

It was interesting to note that the profound improvement was observed in SI, AHI, and blood pressure after 3 months of oral appliance use, which became less marked in profile in the next 9 months. However SAQOL scores showed a steady upward trend (Figure 4).

Table 3—Comparison of before treatment and 3 months after treatment mean SBP, DBP, AHI, SI, and SAQOL scores.

	Before Treatment (n = 37)		3 Months after Wearing Oral Appliance (n = 15)		% Change		Significance of Difference (Wilcoxon Signed Rank Test)	
Parameters	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Z**	**P**
SBP (mm Hg)	137.6	2.4	132.9	8.8	−3.41	2.50	3.440	0.001
DBP (mm Hg)	88.5	1.8	85.6	4.9	−3.27	3.86	3.421	0.001
AHI	22.7	6.6	4.7	2.5	−79.29	6.80	3.415	0.001
SI	8.2	1.3	1.5	1.4	−81.70	13.84	3.508	< 0.001
SAQOL	2.3	1.0	5.5	0.8	139.1	113.32	3.449	0.001

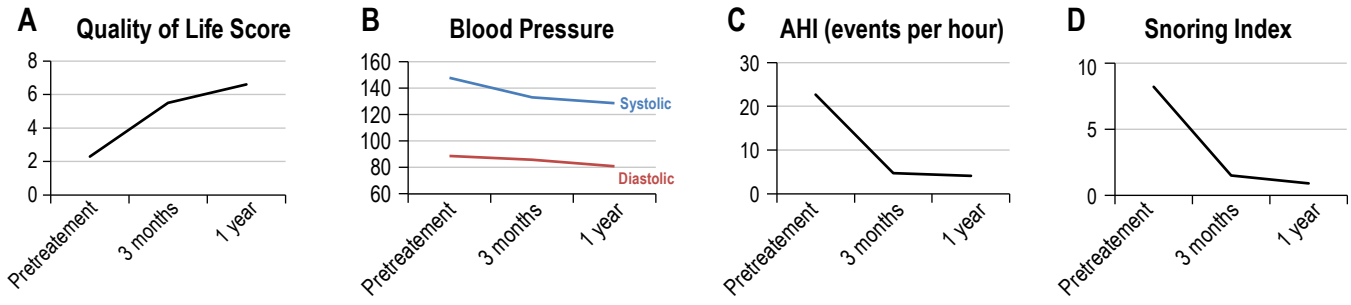
SD, standard deviation; SDB, systolic blood pressure; DBP, diastolic blood pressure; AHI, apnea-hypopnea index; SI, snoring index; SAQOL, sleep apnea quality of life.

Table 4—Comparison of before treatment and 1 year after treatment mean SBP, DBP, AHI, SI, and SAQOL scores.

	Before Treatment (n = 37)		1 Year after Wearing Oral Appliance (n = 15)		% Change		Significance of Difference (Wilcoxon Signed Rank Test)	
Parameters	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Z**	**P**
SBP (mm Hg)	137.6	2.4	128.5	9.5	−6.6	3.02	3.431	0.001
DBP (mm Hg)	88.5	1.8	80.7	4.0	−8.81	3.35	3.429	0.001
AHI	22.7	6.6	4.1	1.9	−81.9	4.73	3.413	0.001
SI	8.2	1.3	0.9	0.9	−89.02	9.35	3.473	0.001
SAQOL	2.3	1.0	6.6	0.6	186.9	134.45	3.473	0.001

SD, standard deviation; SDB, systolic blood pressure; DBP, diastolic blood pressure; AHI, apnea-hypopnea index; SI, snoring index; SAQOL, sleep apnea quality of life.

Figure 4—Graph showing quality of life, blood pressure, AHI, and snoring index variation at 3 months and at 1 year.



AHI, apnea-hypopnea index.

Otsuka et al. showed a significant reduction in SBP (from 118.4 to 113.7) and DBP (from 71.6 to 67.2) following the use of oral appliance.[30] The study suggested that successful OSA treatment with an oral appliance may also be beneficial to lower blood pressure in OSA patients, as previously suggested for nasal continuous positive airway pressure therapy.

Becker et al. showed that active CPAP treatment resulted in a pronounced reduction in daytime and nighttime blood pressure (blood pressure reduction of 8.1 and 11.4 mm Hg for SBP and DBP), a result comparable to those of the present study.[31]

There are conflicting data in the literature on the impact of CPAP on blood pressure, with some studies reporting limited or no effect while others have found benefit.[32–38] This may be because compliance with oral appliance is much higher than compliance with CPAP.

Zang et al. showed that after a 12-week treatment, an oral appliance group showed significant reduction in nocturnal mean SBP and DBP, and 24-h and diurnal SBP ([121.3 ± 7.0] vs [125.3 ± 9.3], [76.1 ± 6.1] vs [78.8 ± 6.8], [127.2 ± 7.5] vs [129.4 ± 8.8], and [131.5 ± 6.9] vs [133.6 ± 8.1], respectively, all $P < 0.01$).[38]

A systematic review incorporating seven studies on effect of oral appliance on blood pressure in patients with OSA by Iftikhar et al.[39] showed that the pooled mean change in the SBP, DBP, and the MAP (mean arterial blood pressure) were −2.7 mm Hg, −2.7 mm Hg, and −2.40 mm Hg, respectively, showing

that effective oral appliance therapy can bring about reduction in SBP, DBP, and MAP.

Sleep apnea quality of life is an objective measurement of a combined state of well-being in an individual and is dependent on a multitude of factors. There is evidence that altered health is often accompanied by psychological distress that hampers the quality of life.[40] A minor elevation in health status might influence the quality of life tremendously. The findings in the present study also support this assumption, as we observed almost 240% rise in sleep apnea quality of life of patients with a change of 12.16% and 14.01% in SBP and DBP and a change of 82.52% and 89.77% in AHI and SI scores.

Limitations of this study include the absence of any controls, as it was unethical to leave hypertensive patients untreated for 1 year. Another limitation was the effect of lifestyle factors, intake of caffeine and alcohol, and presence of obesity, which were not part of the study. All the patients were regularly recalled, and most of them did not report any discomfort with mandibular advancement device. However, in a few patients, altered maxillomandibular relationships (due to mandibular advancement) could cause impingement of the dental prosthesis in newer areas. In this event, such areas were identified and the MAD was suitably modified to prevent further impingement.

CONCLUSION

The result of the present study suggests that oral appliance can reduce blood pressure and can bring a radical change in blood pressure, apnea-hypopnea index, snoring index, and sleep apnea quality of life within a short period (3 months) and has the ability to sustain this trend in the long run (1 year).

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SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication December, 2014
Submitted in final revised form February, 2015
Accepted for publication February, 2015

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DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest. The work was performed at the Department of Prosthodontics, Saraswati Dental College & Hospital, Lucknow and Department of Pulmonary Medicine, King George's Medical University, Lucknow, India.