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CLINICAL RESEARCH

Effect of mandibular advancement device on sleep bruxism score and sleep quality

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ABSTRACT

Statement of problem. The use of mandibular advancement devices (MADs) in the treatment of sleep bruxism is gaining widespread importance. However, the effects of MADs on sleep bruxism scores, sleep quality, and occlusal force are not clear.

Purpose. The purpose of this clinical study was to analyze the effect of MADs on sleep bruxism scores, sleep quality, and occlusal force.

Material and methods. This uncontrolled before and after study enrolled 30 participants with sleep bruxism. Outcomes assessed were sleep quality, sleep bruxism scores (sleep bruxism bursts and sleep bruxism episodes/hour), and occlusal force before and after 15 and 30 days of using a MAD. Sleep bruxism scores were assessed by ambulatory polysomnography and sleep quality by using the Pittsburgh sleep quality index (PSQI). Occlusal force was recorded by using a digital gnathodynamometer in the first molar region on both sides. Statistical analysis was done by 1-factor repeated measures ANOVA (α=.05).

Results. Statistically significant reductions in sleep bruxism bursts/h, sleep bruxism episodes/h, and PSQI scores were found after 15 and 30 days of using a MAD (P<.001). Statistically significant reduction in occlusal force on both sides was found only after 15 days (P<.001) but not after 30 days of using a MAD (P=.292 on left side, and P=.575 on the right side).

Conclusions. The study showed a short-term improvement in sleep bruxism scores, sleep quality, and reduction in occlusal force in sleep bruxism participants after using MADs. (J Prosthet Dent 2017;117:67-72)

Bruxism is a sleep-related movement disorder characterized by the clenching and grinding of teeth and is of particular importance to restorative dentistry because of the fracture of dental restorations, attrition of teeth, and initiation of temporomandibular disorders.[1] Bruxism has 2 distinct circadian manifestations: during sleep (sleep bruxism) or during wakefulness (awake bruxism).[2] The pathophysiology of sleep bruxism (SB) is still unclear,[3] and the proposed cause of sleep bruxism includes sleep arousal, autonomic sympathetic–cardiac activation, genetic predisposition, and exogenous factors.[4-6] Standard polysomnography (PSG) in a sleep laboratory with research diagnostic criteria is the gold standard for diagnosis of sleep bruxism.[7-11] Criteria are based on a typical electromyographic pattern known as rhythmic masticatory muscle activity in the masseter and temporalis muscles. Ambulatory PSG may be used as an alternative to the standard PSG and offers a more practical approach.[6,12-17]

The effect of sleep bruxism on sleep quality and occlusal force has been reported but is unclear.[18-22] Therefore, evidence is needed to assess any change

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Clinical Implications

Mandibular advancement devices could be an effective management option for sleep bruxism.

brought about by the use of a mandibular advancement device (MAD).[20-25] The most common management strategy for SB includes occlusal splint therapy, which significantly reduces SB motor activity index (episodes/hours of sleep).[25-30] However, recent evidence has confirmed an association between sleep bruxism and obstructive sleep apnea (OSA),[31-34] and 1 study has found a complete absence of tooth grinding sounds in confirmed sleep bruxers during the use of continuous positive airway pressure.[34] This observation led to the assumption that the MAD used in the treatment of OSA can be assessed for their effect on SB,[35-37] although studies are limited.[18,19,38-40] The present study was conducted to analyze the effect of an MAD on sleep bruxism scores, sleep quality, and occlusal force.

MATERIAL AND METHODS

This uncontrolled before and after study was conducted from April 2013 to November 2014 at a tertiary care hospital in India, after approval from the institutional ethical committee (2787 Ethics/R. Cell-14). Sample size was calculated to be 28 according to the formula

$$N=\frac{Z^2_{1-\alpha}P(1-P)}{d^2}$$

where Z is the standard normal distribution of 1.96 for a 2-sided test at the significance level (a) of .05, N is the required sample size, P is the expected prevalence (5%),[19] and d denotes absolute precision (8%).

Inclusion criteria were a history of tooth grinding sounds reported by a family member at least 3 times a week in the previous 6 months, either sex, and between 18 and 40 years of age, with natural dentition having no more than 2 missing teeth except for the third molars. Exclusion criteria were other known sleep disorders (besides OSA), restricted mouth opening, history of orthodontic treatment, current tobacco or alcohol consumption, type 2 diabetes mellitus, severe periodontitis or mobile teeth, and pharmacotherapy that might have affected the central nervous system.

Thirty-seven participants were recruited on the basis of these criteria from a general dental clinic, and informed consent was received. Participants underwent ambulatory PSG to find the actual participants (n=30) who fulfilled the diagnostic cutoff value[12] of either more than 4 SB episodes per hour of sleep or more than 25 SB bursts per hour of sleep. Participant selection and clinical examination were performed by 2 separate trained examiners (N.S., B.P.S.) to facilitate blinding.

Figure 1. Mandibular advancement device.

After personal history and sociodemographic data were acquired, sleep quality was assessed subjectively by using the Pittsburgh Sleep Assessment questionnaire.[41] This questionnaire contains 19 questions, of which the last 5 questions are rated by a bed partner or roommate. Only the initial 14 questions were used in this study, as the remaining 5 are more of clinical significance and less of research significance. In addition, the unavailability of a bed partner in some participants could have led to bias. These questions were combined to form 7 "component" scores, ranging from 0 to 3 points, where a higher score indicated greater difficulty. The component scores were added to make up a global score ranging from 0 to 21 points. The global Pittsburgh sleep quality index (PSQI) of 0 to 4 represented good sleep quality, and a score of more than 4 indicated poor sleep quality.

Ambulatory overnight polysomnography (Alice PDx; Philips Respironics) was performed for electromyographic (EMG) recording of the masseter muscle in the participants' bedrooms during their usual sleep time to calculate the number of sleep bruxism bursts per hour and the number of sleep bruxism episodes per hour.

Occlusal force was measured unilaterally with a digital gnathodynamometer (BT 100; Load Master and Digital Indicator LI450; Load Master) in the first molar region. Recordings were made by placing the transducer in the first molar region, and then participants were instructed to occlude as forcefully as possible for about 3 seconds. Three recordings were made with a rest period of 5 minutes for each side for each participant, and the mean of the 3 recordings was used.

Intervention was provided by using a custom-fabricated, hard acrylic resin twin block MAD, fabricated at 50% of maximal protrusion with an interincisal opening of 6 mm (Fig. 1). Any required intraoral adjustments were done by remounting to achieve a proper

Table 1. Effect of mandibular advancement device on outcome measures (N=25), mean ±SD (range)

Outcome Measure	Baseline	Day 15	Day 30	F	P
PSQI (score)	12.00 ±1.41 (9-14)	7.87 ±1.16 (5-10)	4.11 ±1.09 (2-7)	340.47	<.001
Sleep bruxism bursts/h	54.60 ±11.17 (30.32-74.93)	30.21 ±10.29 (12.52-53.95)	26.70 ±8.03 (10.21-38.69)	403.65	<.001
Sleep bruxism episodes/h	7.75 ±1.39 (5.39-10.20)	4.45 ±1.19 (2.55-7.51)	3.10 ±0.75 (1.61-4.76)	234.71	<.001
Occlusal force right side (n)	57.04 ±4.31 (48.41-65.83)	38.16 ±4.60 (26.85-48.95)	36.70 ±4.68 (26.00-47.00)	279.29	<.001
Occlusal force left side (n)	55.05 ±4.99 (42.31-65.79)	38.34 ±4.60 (28.28-48.01)	37.50 ±4.74 (27.74-47.96)	281.32	<.001

PSQI, Pittsburgh Sleep Quality Index. N=25.

Table 2. Comparison of mean differences between periods by using Tukey test

Comparison	PSQI	Number of Sleep Bruxism Bursts/h	Number of Sleep Bruxism Episodes/h	Occlusal Force Right Side	Occlusal Force Left Side
Baseline versus day 15	<.001	<.001	<.001	<.001	<.001
Baseline versus day 30	<.001	<.001	<.001	<.001	<.001
Day 15 versus day 30	<.001	<.001	.006	.292	.575

PSQI, Pittsburgh Sleep Quality Index.

fit on the teeth. Participants were instructed about the use and maintenance of their MAD.

Data were reacquired in the same way as described earlier at 15 days and 30 days after the start of using an MAD. Groups were compared by 1-factor repeated measures ANOVA, and the significance of mean differences between the groups (pairwise comparisons) was found by using the Tukey honest significant differences post hoc test, considering participants as a random effect and time as a fixed effect (α=.05). Analyses were made with software (Statistca v7.1; StatSoft Inc).

RESULTS

Of thirty participants, 2 did not report for follow-up, and 3 exhibited temporomandibular joint pain and withdrew from the study, leaving a final sample size of 25. The global PSQI score was significantly affected by the use of an MAD (F=340.47; P<.001) (Table 1). A statistically significant reduction (P<.001) was observed in the mean global PSQI score at 15 days (34.4%) and at 30 days (65.8%), compared with baseline (Table 2, Fig. 2). A statistically significant reduction was found in the number of sleep bruxism bursts per hour at 15 days and 30 days compared with baseline (P<.001) (Table 2, Fig. 2). A significant effect of an MAD on the number of sleep bruxism episodes/h (P<.001) was found (Table 1). Furthermore, the Tukey test showed a significant (P<.001) decrease in the mean number of sleep bruxism episodes/h at 15 days (42.5%) and at 30 days (59.9%) compared with baseline (Table 2, Fig. 2).

A statistically significant reduction (P<.001) in occlusal force on the right side was noted at 15 days (33.1%) and 30 days (35.7%) (Table 2, Fig. 2). The occlusal force on the left side showed a trend similar to the occlusal force on the right side, where a significant (P<.001) reduction was noted at 15 days (30.4%) and 30 days (31.9%) (Table 2, Fig. 2). No significant effect was found on the occlusal force from 15 to 30 days of using an MAD (P=.292 on left side, and P=.575 on right side).

DISCUSSION

The study found that the use of an MAD significantly improved sleep quality, sleep bruxism scores, and occlusal force on both sides at 15 and 30 days (P<.05). However, when the change in occlusal force on the right and left sides at 15 days was compared with 30 days, it was not significantly different (P>.05).

The clinical findings of bruxism can be confirmed with help of electromyography of the masseter muscles by using an ambulatory PSG (BiteStrip; Up2dent.com) or standard PSG in a sleep laboratory.[5] An ambulatory PSG was selected for this study. It measures only the EMG activity of the masseter muscles and cannot differentiate between bruxism and other orofacial activities.[19,42] Standard PSG is the gold standard for the assessment of bruxism,[7-11] but it is expensive and records the parameters when participants are sleeping in an artificial environment,[13,14] which may lead to false positive or false negative findings.[6,15] Ambulatory PSG is readily available, inexpensive, records the parameters while the participants is sleeping in his natural milieu,[6,12-14] and is highly sensitive to EMG activities.[6,12,30] Ambulatory PSG criteria by Doering et al[12] were used in this study to detect SB.[12]

The number of sleep bruxism bursts per hour and number of sleep bruxism episodes per hour showed clinically significant reduction following the use of an MAD. The decrease in these indices was high in the first 15 days of using an MAD, and there was further significant reduction in the next 15 days. A similar observation was made in other studies. Landry et al[39] showed a

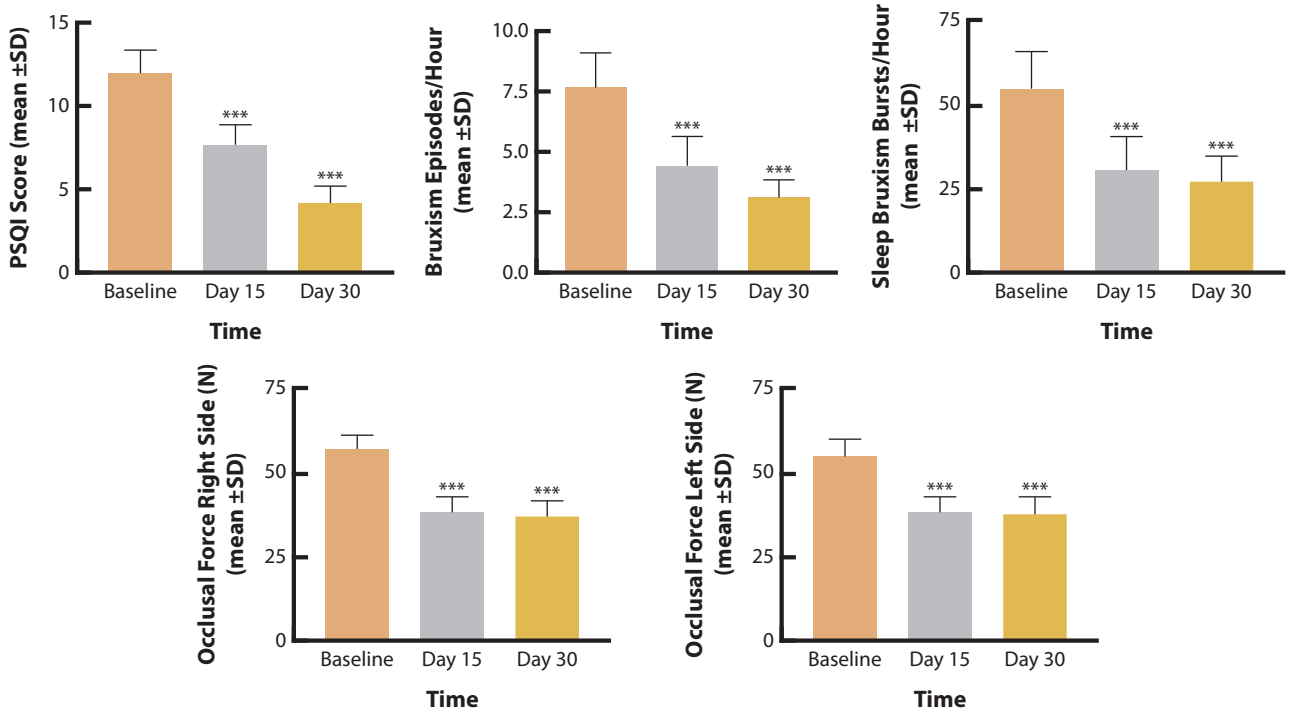

Figure 2. Mean outcome measures over periods.

considerable reduction in the sleep bruxism motor activity of the 12 participants in their study after the use of a MAD. Landry et al[43] also showed a significant reduction ($P<.04$) of 39% and 47% in the number of episodes per hour after using a mandibular advancement appliance at slight and advanced protrusive levels, respectively. Saueressing et al[19] reported a positive effect from a mandibular advancement device by means of the sleep bruxism scores obtained with the help of the BiteStrip.[19]

The mechanism of action of a MAD is still unclear. Landry et al[39] hypothesized that pain associated with the use of a MAD might be responsible for reducing sleep bruxism activity in their participants, as shown by a high number of participants complaining of pain after the use of a MAD. This claim was supported by an earlier study, indicating decreased jaw muscle activity concomitant with pain.[42] However, a recent study found no significant difference in electromyographic jaw muscle activity, as measured with a single channel portable EMG device, between pain-free participants and participants with painful conditions.[16] The action of a MAD is associated with the forward placement of the mandible and the maintenance of a more patent airway. Several studies indicate the relationship of OSA and SB.[31-33] A relationship might exist between the awakening process associated with OSA and sleep bruxism episodes, which would further clarify the effects of treatments indicated for OSA on SB.

Occlusal force was reduced considerably on the right and left sides (33.1% and 30.4%, respectively) after 15 days of using a MAD. Reduction in occlusal force values at 30 days from baseline was also significant ($P<.05$) on both sides, exhibiting a reduction of 45.4% and 42.9%, respectively. The reduction in occlusal force at 30 days after the insertion of MAD was not statistically significant compared with the reduction observed at 15 days. Mainieri et al[44] compared the occlusal force in individuals with bruxism before and after the use of a MAD and obtained results similar to those in this study. Some studies support the hypothesis that occlusal force increases in individuals with bruxism because of hypertrophy of the masseter muscles caused by overtraining.[22] However, earlier studies found no clinically significant influence of bruxism on occlusal force.[23,24] This variation can be explained by the multiple methodologies used to measure occlusal force and the variation in occlusal force from individual to individual based on the sex and general build of the participant. Reduction in occlusal force after the use of a MAD could be due to the relaxation of strained and hypertrophied muscles resulting from passive stretching of masticatory muscles while the jaw is held in a forward position.[18,19]

A significant reduction in the global sleep score was evident at the end of 30 days of using MAD. Similar to the change in other parameters in this study, a significant change was observed in the global sleep score when comparing the change at 15 days and 30 days. A dramatic improvement in sleep quality resulted after the use of a MAD, and none of the participants reported any discomfort that might worsen sleep quality. The results of

this study are in agreement with those of previous studies.[44] The improvement in sleep quality can be attributed to a reduction in rhythmic masticatory muscle activity episodes, leading to a concomitant reduction in microarousals.[9] Moreover, sleep quality might also have improved as a result of maintaining a more patent airway.

An interesting observation in this study was that global PSQI score, SB bursts/h, SB episodes/h, and occlusal force on both sides decreased significantly after 15 days of using a MAD, however occlusal force did not change significantly after 30 days of using a MAD. This could be interpreted as continued improvement of sleep quality and sleep bruxism indices, except that the reduction in occlusal force did not occur significantly after the initial 15 days of use of a MAD. This effect could not be clearly explained; however, one explanation is that relaxation of the masticatory musculature was achieved in the initial phase of therapy. Previous studies have not observed this effect, perhaps because observations at such short intervals have not been attempted. The implications of this observation are still unclear, and further study in this direction is warranted.

The sample size was selected from a tertiary care institution (incidental sample), so this result should be generalized with caution. A false negative effect could result from decreased activity, specifically on the night of PSG. Polysomnography could have been performed for 1 additional night before the night of evaluation so that the participant could become habituated. This limitation could be somewhat overcome by selecting participants with a high frequency of reported bruxism activity. This study did not involve a control group wearing a sham appliance and so the observed effect cannot be definitively attributed to MAD therapy. This type of study design could not explain the mechanism of action. Further long-term randomized controlled trials will be able to clarify the mechanism of action of a MAD in sleep bruxism and its possible side effects.

CONCLUSIONS

Based on the findings of this clinical study, it was concluded that a mandibular advancement device improved sleep quality and significantly reduced sleep bruxism scores and occlusal force.

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