Analysis of the Influence of a Mandibular Advancement Device on Sleep and Sleep Bruxism Scores by Means of the BiteStrip and the Sleep Assessment Questionnaire

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Purpose: A before-and-after experimental clinical study was carried out with the objective of evaluating the effect of a mandibular advancement device (MAD; 75% advancement), made of a thermoplastic material, on sleep bruxism (SB) and sleep scores. ***Materials and Methods:*** After a habituation period of 1 week, SB scores were taken at baseline and after use of the MAD for 30 days. Scores were compared using the newly developed BiteStrip, which registers the number of contractions of the unilateral masseter muscle after a 5-hour period, giving a severity score from 0 to 3 after the registrations. To assess sleep, the Sleep Assessment Questionnaire (SAQ), a screening tool with scores ranging from 0 to 68, was used before and after use of the MAD. Twenty-eight subjects (13 women, 15 men; mean age: 42.9 ± 12.0 years) with a clinical history of SB and no spontaneous temporomandibular disorder (TMD) pain were selected. The clinical diagnosis of either moderate or severe SB was further confirmed through use of the BiteStrip (scores 2 or 3) at baseline. A 30-day follow-up period was used for evaluation. Both methods were validated against polysomnography. In addition, common signs and symptoms of TMD based on the Research Diagnostic Criteria for Temporomandibular Disorders were also evaluated before and after use to assess the side effects of the MAD. ***Results:*** There was a statistically significant improvement in both SB and sleep scores based on the BiteStrip and the SAQ (Wilcoxon signed rank and Student paired *t* test, $P < .05$). In the signs and symptoms of TMD, there was a significant reduction in temporomandibular joint sounds as well as in masseter and temporalis tenderness to palpation. None of the SB subjects experienced any breakage of the MAD. ***Conclusion:*** The MAD had a positive effect on SB and sleep scores, measured by the BiteStrip and the SAQ, respectively, and did not increase any traditional signs and symptoms of TMD in a 30-day evaluation period. *Int J Prosthodont 2010;23:204–213.*

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Sleep bruxism (SB) is an oral parafunction characterized by tooth clenching or grinding.[1,2] Regarding its epidemiology, there is controversy regarding the different methodologies used, but reports of SB have ranged from 5% to 90% of the adult population.[1] Further, there is no difference in SB prevalence between the sexes, but a greater prevalence is found in younger populations when compared to older ones.[3] Bruxism has been associated with exogenous (peripheral) factors, such as occlusal interferences, stress, and anxiety, as well as with endogenous (central) causes involving brain neurotransmitters of the basal ganglia; however, greater importance has been placed on the central factors.[3–5]

Research has been done to test the hypothesis of whether there is an association between breathing and sleep disorders, such as snoring, sleep apnea/hypopnea syndrome, and SB.[6,7] The number of episodes of masseter contractions and the rhythmic masticatory muscle activity were measured in patients with mild and moderate obstructive sleep apnea (OSA). It was concluded that SB is rarely associated with apnea events, but it is related to the sleep disturbances of patients with OSA.[6] On the other hand, in a clinical case report, an association between SB and OSA was suggested.[8] One patient sought treatment of snoring and SB, which he had suffered from for many years. It was demonstrated that the majority of tooth-grinding events were an awakening response at the end of apnea/hypopnea events. The results suggested, although the evidence was weak, that when bruxism is related to sleep apnea/hypopnea, the treatment of respiratory abnormalities may treat SB.[8]

There are several established methods for diagnosing SB, but the gold standard is polysomnography.[9] Polysomnography can monitor all the events that take place during sleep, including muscle activity by means of electromyography (EMG). There are also other methods for SB diagnosis, such as clinical examination, a patient's self-report of tooth grinding, a portable EMG device used in the patient's actual bedroom,[10–12] and the BiteStrip (S.L.P. Scientific Laboratory Products), which evaluates masseter muscle activity.[13] Additionally, the Sleep Assessment Questionnaire (SAQ) is also validated against the polysomnographic method to screen for sleep disorders.[14]

To validate the BiteStrip, research was carried out comparing its results with polysomnography. The SB events detected by the polysomnography EMG were ranked, with identical criteria, and compared with those of the BiteStrip. The sensitivity of the BiteStrip was 0.72, while the specificity was 0.75. The correlations between the BiteStrip and EMG were 0.81 for the total score, 0.79 for the total registration time, and 0.79 for the total sleep time (awakening periods removed). It was concluded that the BiteStrip was a viable instrument with an acceptable accuracy for the identification of SB, considering that it has a strong correlation with EMG readings.[15]

The SAQ is validated against polysomnography results with the objective of screening for primary and secondary sleep disorders in epidemiologic studies. The four factors identified in the SAQ were: nonrestorative sleep, sleep disorder, sleep apnea, and hypersomnolence.[14] Sleep is divided into stages, such as awakening, non–rapid eye movement (NREM), and REM sleep. NREM sleep is further divided into stages 1 and 2 (light sleep) and stage 3 (deep sleep). Stage 2 is linked to K complexes and microarousals besides involuntary limb movements.[11] SB is frequently associated with stages 1 and 2 of NREM sleep, an increase in cardiac frequency, the appearance of K complexes and microarousals, periodic involuntary leg movements and respiratory changes, and an increase in peripheral vasoconstriction.[6,16–18]

Many treatments have been proposed for the reduction of SB, such as occlusal treatment by means of occlusal adjustment, Michigan-type occlusal splints, medication, and psychologic treatments. Nevertheless, none of these has proven to be superior to the others or guarantee total remission of SB signs and symptoms. The objective of clinical interventions related to SB are related directly to tooth protective measures against grinding, relief of facial and head pains, and short-term muscle relaxation.[3] The use of a mandibular advancement device (MAD) is an alternative for the treatment of sleep apnea/hypopnea due to its effect on the oropharynx (an increase in the airway pathway); however, its effect on SB is still relatively unknown. Indeed, only one study reported a reduction in SB patients without sleep disorders using a soft thermoplastic MAD.[19] Tooth and temporomandibular joint (TMJ) sensitivity have been reported by patients after use of the MAD. However, pain and dysfunction of the TMJ seem unlikely to be the consequence of the temporary advancement of the mandible provided by these devices, even when patients report some initial discomfort. This issue is still debatable.[20,21]

A literature review consisting of 10 crossover randomized clinical trial studies compared the different treatments for SB. Among them, 5 studies carried out in the sleep laboratory of the Hospital du Sacré-Coeur, Montreal, employed some SB treatments, of which only 2 used intraoral appliances. There was a decrease in SB in patients who used the MAD; however, this does not mean that this was the best treatment. Patients who used the MAD for only 1 night reported discomfort.[22]

Therefore, the primary objective of this before-and-after study was to evaluate the effect of an MAD on SB scores measured using the BiteStrip. The secondary objective was to evaluate the effect of the MAD on sleep scores by means of the SAQ. A third objective was to assess the effects of the MAD on the traditional signs and symptoms of temporomandibular disorders (TMDs).

Materials and Methods

Sample Size and Study Design

The formula for the sample size calculation for a single sample is:

$$n = Z^2_{1-\alpha/2}\, P(1-P)d^2$$

Fig 1 Placement of the BiteStrip over the masseter muscle.

in which n = the estimated sample size, P = the anticipated proportion in the population, $Z_{1-\alpha/2}$ = the value of the standard normal distribution corresponding to a significance level of α (eg, 1.96 for a two-sided test at the .05 significance level), and d = the absolute precision required on either side of the proportion. Using an anticipated SB proportion of 5% with a confidence level of 95% and absolute precision of 8%, a sample size of 28 individuals was reached.[23]

A before-and-after study design was selected because there was no need of controlling for the sex, age, and craniometric measures of the patients. In this study design, the patient was the control of him- or herself.[24]

Study Protocol

Patients having bruxism as their chief complaint who sought treatment at the Faculty of Dentistry Orofacial Pain Clinic, Catholic University of Rio Grande do Sul, Porto Alegre, Brazil, were included. The preliminary diagnosis of SB was established using a standard questionnaire; in addition, patients also underwent a clinical examination based on the guidelines of the Research Diagnostic Criteria for Temporomandibular Disoders.[25,26] This standard questionnaire was also used to select the sample according to the inclusion criteria: patients with a history of bruxism (at least 3 episodes/week), an absence of no more than one tooth per quadrant, and an absence of any severe limitation of maximum mouth opening (< 35 mm). Exclusion criteria were as follows: pregnancy, severe skeletal alterations, orthodontic treatment in the last 2 years, active periodontal disease, mobile teeth, and use of systemic medication that might interfere with sleep (eg, sedatives).[26] All selected patients signed a consent form approved by the ethics committee at the São Lucas Hospital, Catholic University Faculty of Medicine. All selected procedures were carried out by a single clinical specialist trained in prosthodontics to avoid any major individual variations.

To confirm the initial diagnosis, patients used a new device for measuring the degree of bruxism in each patient, the BiteStrip. This device is similar to surface EMG and has a computer chip that registers the number of contractions of the masseter muscle (SB) during 5 hours of sleep. The computer chip is embeded in a chemical solution and a display is taped over the masseter unilaterally. After the test is completed, the display shows a categorical score representing the number of masseter contractions (SB episodes per minute): 0 = no bruxism (up to 39 episodes), 1 = mild bruxism (40 to 74 episodes), 2 = moderate bruxism (75 to 124 episodes), 3 = severe bruxism ($\mu \geq 125$ episodes), and E = error. The BiteStrip has a simple design and the instructions are understood easily. This helps the professional in the patient's diagnosis, clinical evaluation, and subjective report.[13] The device was employed right before bedtime, according to the steps suggested by the manufacturer: the masseter region and the electrochemical visor were cleaned with alcohol, the visor was covered with the green self-adhesive label, the patient clenched down with maximum force on a wood stick three times within a 20-second period, the patient used the BiteStrip for at least 5 hours during sleep, the label was removed by the researcher, and the score in the visor was registered (Fig1).

Some authors distinguish bruxism episodes from other orofacial movements based on the EMG intensity in each episode. One author stated that bruxism takes place when the force exceeds 40% of the maximum occlusal force during clenching.[27] On the other hand, another affirms that reaching 20% to 25% of the maximum occlusal force is enough to characterize a bruxism episode.[28] The contractions must also last at least 2 seconds to differentiate SB from myoclonic contractions (ie, isolated contractions that last 0.25 seconds). Furthermore, one investigation using audio and visual aids, as well as polysomnography, showed that the functional orofacial and labial movements detected in the masseter EMG hardly exceed 20% of the maximum occlusal force and last longer than 2 seconds.[26] The BiteStrip detects a bruxism episode when the EMG masseter contraction exceeds 30% of the maximum occlusal force.[13]

Patients also responded to the validated SAQ, which contains 17 items with answers that are scored in the following manner: never = 0 points, rarely = 1 point, sometimes = 2 points, frequently = 3 points, and always = 4 points. The sum of these points indicates the quality of sleep of each patient (range: 0 to 68). The higher the score, the worse the sleep quality.[14] In addition, the results of both methods were correlated with each other (BiteStrip and SAQ).

Patients with the diagnosis of SB used the custom-made MAD for 30 days to reduce bruxism activity[22] after a habituation period of 1 week. This habituation period was necessary to make adjustments and exclude those patients who did not tolerate use of the MAD.

To manufacture the MAD, two alginate impressions (JelTrate, Dentsply) were taken of the mandible and maxilla. The impressions were poured with Type IV gypsum (Durone, Dentsply). The casts were then mounted on an adjustable articulator (Whip-Mix Corporation Dental Products) already in the protrusive position (50% to 75% of the maximum protrusive position, depending on patient's tolerance) with approximately 6 mm of interincisal opening, according to the bite registration performed with a silicon-based material (3M Express, Putty and Wash) (Figs 2 and 3). Afterwards, two soft thermoplastic material bite splints, maxillary and mandibular, were fabricated in a thermovacuum device (Plastvac P7, Bio-art Equipamentos Odontológicos). The soft bite splints were 3-mm thick and translucent (Bio-Art Dental Equipments).[21] They were fused on the articulator in the preregistered protrusive position using a microtorch (Piezo Electronic Micro Torch-GB 2001, Micro Torch-Blazer Products). When the separation between the two bite splints was too large, small pieces of the thermoplastic material, which were left from the manufacturing of the soft bite splints, were used to fill the space. The appliance was checked manually for rigidity to prevent fracture and aspiration of the appliance parts. The material type and mounting technique were chosen due to their low cost and the simplicity of building the MAD.

Patients underwent a second evaluation using the BiteStrip and the SAQ to compare the results with the MAD to those at baseline and to assess the bruxism behavior and sleep quality.

Statistical Analysis

The Kolmogorov-Smirnov single-sample test was used for the normality distribution of continuous variables. The Student paired *t* test was used for the improvement evaluation after use of the MAD in bruxers for the continuous variables. The Wilcoxon ranked sum test was used for the ordinal variables, and the McNemar test was used for the dichotomous variables. The statistical levels used were 5% (type I error) and 20% (type II error).[29]

Results

Population

The descriptive characteristics of the sample studied (n = 28) are presented in Table 1. Initially, there were 30 participants; however, 2 individuals withdrew from the study due to tooth sensitivity, uncomfortable sensation, and drooling with use of the MAD. The mandibular advancement was reduced to 50% instead of 75% in 1 patient due to appliance discomfort (ie, excessive drooling and tooth sensitivity), but the patient was included in the final analysis.

Normality

All continuous variables showed nonsignificant results for the Kolmogorov-Smirnov single-sample test, meaning that parametric tests could be used. The continuous variables included the SAQ, maximum mandibular opening, maximum protrusive mandibular movement, maximum right and left lateral mandibular movements, overbite and overjet, weight, height, body mass index, and age.

Results Before and After Using the MAD

According to Table 2, there was an improvement in sleep quality analyzed by the SAQ, and there was no statistically significant difference in the other continuous variables analyzed before and after use of the MAD (maximum protrusive movement, right and left mandibular maximum lateral movements, overbite, and overjet), with the exception of mandibular opening.

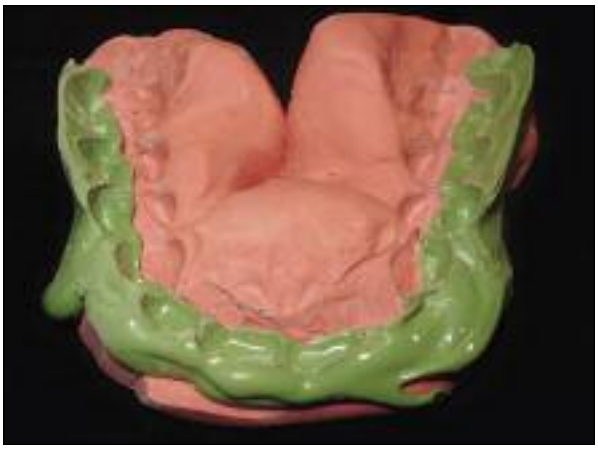

Fig 2 Bite registration with 3M Express (putty and wash) in the final protrusive position (75% of the maximum protrusive position with a 6-mm interincisal distance).

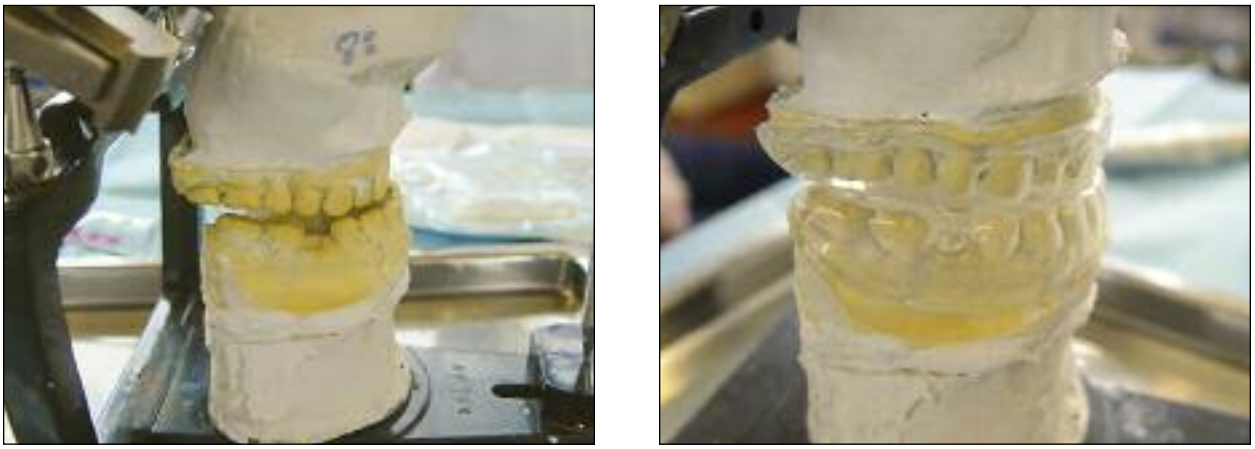

Fig 3 Casts mounted in the 75% maximum protrusive position with a 6-mm interincisal distance via a maxillary and mandibular soft thermoplastic MAD fused using a Bunsen burner.

Table 1 Social and Demographic Description of Bruxers (n = 28)

	Bruxers
Orofacial pain (%):	
Absent = 0	0.0
Present = 1	100.0
Education level (%):	
Incomplete elementary school = 1	0.0
Complete elementary school = 2	3.6
Incomplete high school = 3	3.6
Complete high school = 4	14.3
Incomplete undergraduate degree = 5	14.3
Complete undergraduate degree = 6	32.1
Postgraduate education = 7	32.1
Sex (%):	
Female = 0	46.4
Male = 1	53.6
Mean age (y)	42.9 (SD: 12.0)
Mean weight (kg)	72.7 (SD: 14.4)
Mean height (cm)	168.8 (SD: 8.2)
Mean body mass index	24.8 (SD: 3.9)

SD = standard deviation.

Table 2 Before-and-After Evaluation of the Continuous Variables in Bruxers

	Baseline mean (SD)	Day 30 mean (SD)	Student paired *t* test *(P)*
Sleep Assessment Questionnaire	30.1 (6.1)	23.3 (5.8)	.000***
Maximum mouth opening (mm)	55.6 (3.8)	54.7 (4.0)	.007**
Maximum protrusive movement (mm)	5.4 (2.4)	5.1 (2.1)	NS
Maximum right lateral excursive movement (mm)	4.2 (3.3)	4.2 (3.3)	NS
Maximum left lateral excursive movement (mm)	4.7 (3.8)	4.5 (3.6)	NS
Overbite (mm)	1.9 (1.4)	1.8 (1.5)	NS
Overjet (mm)	2.3 (1.4)	2.3 (1.5)	NS

NS = not significant.
**$P < .01$; ***$P < .001$.

Table 3a Description of Scores for Ordinal Variables in Bruxers

BiteStrip	
0	No bruxism: 0–39
1	Light bruxism: 40–74
2	Moderate bruxism: 75–124
3	Severe bruxism: ≥ 125
E	Error: no reading
Occlusal wear	
1	No wear or light tooth wear
2	Wear in enamel
3	Wear in dentin spots
4	Exposure of dentin in the area > 2 mm^2
5	Wear greater than one third of the clinical crown
TMJ sounds	
1	Crepitation
2	Click

Table 3b Before-and-After Evaluation of the Ordinal Variables in Bruxers

	No. of patients with higher, lower, and equal scores	Wilcoxon signed rank test *(P)*
BiteStrip		
Reduced	27	.000***
Increased	0	
Equal	1	
Occlusal wear		
Reduced	0	NS
Increased	2	
Equal	26	
TMJ sounds		
Reduced	13	.005**
Increased	3	
Equal	12	
Grinding or clenching of teeth		
Reduced	23	.000***
Increased	0	
Equal	5	

NS = not significant.
**$P < .01$; ***$P < .001$.

Table 4 Before-and-After Evaluation of the Dichotomous (Binary) Variables in Bruxers

	Baseline	Day 30	McNemar test *(P)*
TMJ sounds (right):			
Absent = 0	12	23	.001**
Present = 1	16	5	
TMJ sounds (left):			
Absent = 0	15	20	NS
Present = 1	13	8	
Masseter (sensitivity to palpation):			
Absent = 0	6	15	.02*
Present = 1	22	13	
Temporalis (sensitivity to palpation):			
Absent = 0	8	17	.04*
Present = 1	20	11	
Sternocleidomastoid (sensitivity to palpation):			
Absent = 0	14	19	NS
Present = 1	14	9	
Trapezius (sensitivity to palpation):			
Absent = 0	10	13	NS
Present = 1	17	14	

NS = not significant.
*$P < .05$; **$P < .01$.

However, the decrease in opening was less than 2%, which was not considered clinically relevant. In relation to the results of the SAQ, only one patient did not have an improvement in sleep quality; however, this patient had only a 50% mandibular advancement of the mandible. Weight, height, body mass index, and age were not analyzed due to the unlikely changes in these variables during the short span of the study (30 days).

According to Table 3, there was a significant improvement in bruxism scores using the BiteStrip in almost all patients after use of the MAD. Of the 28 patients, 27 saw an improvement in SB scores, 1 remained the same, and none got worse. In addition, there was a reduction in the self-perception of articular sounds (clicking and crepitation), as well as the marked individual perception of grinding/clenching of the teeth. There was no statistically significant difference in the occlusal grinding parameters.

Table 4 shows a significant improvement in sounds of the right TMJ and in tenderness to palpation of both masseter and temporalis muscles. A small but nonsignificant reduction in joint sounds was noticed in the left TMJ, as well as in the muscle palpation sensitivity of the sternocleidomastoid and trapezius muscles.

Discussion

Sample Size and Study Design

The prevalence of bruxism has shown wide variation across studies from 5% to 90%, which makes it virtually impossible to calculate a representative sample with 100% precision. Nevertheless, the current sample size is similar (20 to 30 subjects) to other recent studies in the literature on bruxism, which confirms its external validity.[22,30,31] Indeed, the proportion of men to women was close to 50%, confirming that no difference among the sexes was reported for bruxism. In addition, the average age of patients (42.9 years) also confirms that the prevalence is higher in young adults.[30,31]

A before-and-after study design was chosen because it is a longitudinal type of study that provides simultaneous control groups, randomized distribution, and prognostic stratification. In addition, patients act as their own controls within the sample, it has a low cost, it shows low individual variation, and it can be done in a short period of time. This type of study is indicated for new therapies and diagnostic methods, which is the case for the BiteStrip and the MAD.[24] However, this study design has a major shortcoming: it does not control for the placebo effect. Nevertheless, the average influence of the placebo effect in treatment outcome studies for most diseases has been reported to be around 15%.[32] This study design is not indicated for orofacial pain, where the placebo effect has been reported to be around 60%.[24] The influence of the placebo effect in bruxism treatment outcome studies is not yet known, but it is unlikely that a marked reduction in 27 of 28 patients in BiteStrip readings might be due solely to placebo.

Clinical Examination, BiteStrip, and SAQ

In the clinical history, all patients in this sample reported headaches, tooth sensitivity, and bruxism. In addition, despite not being TMD patients, clinical examination of the masseter, temporalis, sternocleidomastoid, and trapezius muscles, as well as both TMJs, revealed sensitivity to palpation in the majority of them. The report of muscle tension is not reliable for the diagnosis of SB, even though this is a common report in bruxers.[2] This happens when the motor activity becomes physiopathologic with an increase in masticatory muscle activity.[5] In the current examination, the authors dichotomized the scores (0 = absent, 1 = present) to increase the agreement level when compared to the 0 to 3 scoring system of the Research Diagnostic Criteria for Temporomandibular Disorders.[25]

In one study,[22] the MAD showed the best results in the treatment of SB in relation to other types of intraoral appliances (occlusal and palatal). Similarly, the majority of individuals reported a reduction in grinding and clenching frequency during the course of this study (30 days), which indicated a reduction of SB and relaxation of the masticatory muscles.

In the pattern of occlusal grinding, a frequent sign in bruxers, there was no statistically significant difference observed after use of the MAD. This might be attributed to the short period of time between the initial and the final examination of patients, as well as the lack of dental contact during the MAD time of use (30 days). On the other hand, all patients showed some type of occlusal grinding, particularly on the enamel, due to the young age of this sample. The amount of grinding also depends on how long the patient has been bruxing, the frequency of grinding, the force applied to the teeth during parafunction, and the presence or absence of awakened bruxism in combination with SB, among others.[2]

The results showed that no visible changes in the stomatognathic system were noticed after use of the MAD, which is in agreement with the literature.[33] Actually, in relation to joint sounds, there was a significant improvement in the right TMJ. In addition, there was also a significant reduction in muscle palpation sensitivity in the masseter and temporalis muscles. These findings are in agreement with the results observed in another study in which the authors reported that TMD signs and symptoms are unlikely to be the consequence of the temporary advancement of the mandible caused by the MAD.[20]

The adaptation period of 2 weeks is also in agreement with the literature and probably had no effect on the results.[34] In addition, the increase in vertical dimension used in this study (6 mm) is in agreement with previous studies[35,36] and has been shown not to cause major EMG changes in the masticatory muscles or in apnea events.[37,38] In addition, the choice of the monoblock-type over the split-type, which has been shown to be more comfortable for patients,[39] had a possible positive impact in the stomatognathic system. Finally, the choice of a soft silicon-based thermoplastic 3-mm-thick material, besides being less costly, has been shown to be as effective as the rigid ones.[40,41] However, the rigidity of this soft appliance must be assured to prevent the aspiration of broken parts.[42] Despite the reasons behind this choice, two individuals were unable to use the MAD for 30 days due to tooth sensitivity, uncomfortable sensation, and drooling. This is also in agreement with previous studies that reported symptoms of discomfort in some patients after use of the MAD.[22]

After use of the MAD, there was an improvement in sleep scores measured by the SAQ. The underlying mechanism might be related to microarousals, which

prevent deep sleep, leaving individuals in a light stage of sleep or in deep sleep with random awakenings.[18] As a consequence, there is an increase in blood circulation and heart rate, respiratory changes, peripheral vasoconstriction, muscle activity, presence of rhythmic masticatory muscle activity, and bruxism activity.[11] Periodic limb movement is also present in SB episodes, confirming the association of SB with the awakening process.[4,8,11] Additionally, the rhythmic masticatory muscle activity and bruxism episodes take place during light sleep,[11] ie, stages 1 and 2 of NREM sleep.[16,17] It must be pointed out that the SAQ is a screening questionnaire and only the global score was assessed. A more detailed assessment in a sleep laboratory would have been more conclusive. However, the SAQ has been shown to have a good correlation with polysomnographic results.[14]

According to the BiteStrip, there was an improvement in SB scores after use of the MAD. It must be taken into consideration that the BiteStrip was used only twice, before and after use of the MAD. A variation in episodes of SB might have influenced the results.[43] It is important that future long-term studies use an average of the measurements to reduce possible fluctuation in the readings. Nevertheless, the majority of patients (27 of 28) had an improvement in the final score, which seems to reduce the possibility of incidental findings. Finally, the BiteStrip detects a bruxism episode when the EMG masseter contraction exceeds 30% of the maximum occlusal force. Therefore, if the recordings started after 20% or 40% of the maximum occlusal force, the results would have been different.[26] Regardless of the percentage of maximum occlusal force used, orofacial activities, such as lip sucking, head movements, chewing-like movements, swallowing, head rubbing and scratching, eye opening and blinking, might be taken erroneously as bruxism episodes and might have influenced the results.[3,13,26] However, a recent study in the literature found a good correlation between orofacial activities recorded with the BiteStrip and the polysomnographic recordings.[15] It must be pointed out that orofacial activities are different from SB events and only audio-video recordings are capable of discriminating between the two. A study comparing the results of the BiteStrip with EMG readings in a sleep laboratory is currently under investigation by this research group. On the other hand, it might be suggested that the MAD has a positive effect not only on light and mild OSA, but also on SB patients, which is in agreement with the recent literature.[19]

In both cases, the mechanism by which this improvement takes place is the forward movement of the mandible with a consequent increase in the airway space; however, further studies are needed to clarify this.[20,21] Therefore, there might be an apparent relationship between SB and OSA. In one study, it was observed that SB is rarely related to apnea events but rather to sleep disturbances in patients with OSA.[6] Another study suggested that the indicated treatment for OSA might also be favorable for SB, once it becomes clear that it is correlated to the awakening process caused by apnea.[8,18] Finally, the long-term benefits and side effects of the MAD, besides tooth and TMJ discomfort, must be assessed further.[44]

Comparing the results of the SAQ before and after use of the MAD, there was an improvement in sleep scores concomitant with the improvement in bruxism scores, as measured by the BiteStrip. It might be suggested that there must be an improvement in sleep quality and bruxism due to the reduction of microarousals.[18] In addition, it might also be suggested that there is a direct relationship between the SAQ and BiteStrip scores, agreeing with a previous study that compared these two screening tests to polysomnography.[14,15] However, this study could not find a direct relationship between the SAQ (global score) and the BiteStrip. This might be due to the different scales used in the two instruments. The SAQ is a continuous scale (scores: 0 to 68), while the scores of the BiteStrip are categorical (scores: 0 to 4). Finally, the scores of the SAQ, different than the BiteStrip, are subjective and depend on the patient's self-assessment.

Further research must be carried out comparing these research instruments with polysomnography (gold standard), which is in progress by this research group, to confirm these initial findings of MAD effectiveness.[10–12] In addition, the effect of the MAD in patients with both SB and TMDs must be further investigated.

Conclusions

Taking into consideration the limitations of this study, the following conclusions can be drawn:

- The soft thermoplastic MAD did not cause any increase in the existing signs and symptoms of TMD in non-TMD subjects.
- In general, there was a significant reduction in the SB scores after use of the MAD, as measured by the BiteStrip.
- There was a reduction in the patient's perception of clenching and grinding of the teeth after use of the MAD.
- There was a significant improvement in sleep scores, as measured by the global score of the SAQ.
- A relationship between the sleep scores of the SAQ and the BiteStrip could not be demonstrated in this study.

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Literature Abstract

Is the presence of *Helicobacter pylori* in the dental plaque of patients with chronic periodontitis a risk factor for gastric infection?

The aim of this prospective clinical study was to evaluate the prevalence of *Helicobacter pylori* in both dental plaque and the gastric mucosa of patients with gastric symptoms who were admitted to a gastroenterology department. A total of 101 patients (45 women, 56 men; mean age: 40.77 ± 14.15 years) were enrolled in the study. All patients were nonsmokers. The following patient exclusion criteria were applied: diabetes, pregnancy, human immunodeficiency virus–positive, previous treatment of peptic ulcer, cancer, antibiotic therapy during previous 2 months, signs of severe periodontal and caries infections, and recent use of nonsteriodal anti-inflammatory drugs. All patients complained of dyspepsia and underwent gastroscopy and antral biopsies. Biopsy samples were analyzed immediately with a rapid urease test kit. One investigator performed the periodontal examination for all patients, comprised of probing depth, Plaque Index, bleeding on probing, and clinical attachment levels. Patients exhibiting bleeding on probing and presenting with at least four teeth with 3 mm or more of probing depth were designated as having chronic periodontitis (n = 62). The remainder were considered healthy oral cavity subjects. Dental plaque samples were then collected from two teeth and immediately inoculated into the rapid urease test gel. The χ^2 test was used to analyze the association between the presence or absence of *H pylori* in the stomach and the detection frequencies of *H pylori* in the dental plaque of patients with and without periodontitis. Seventy-nine percent of patients with periodontitis had a significantly higher rate of *H pylori*-positive test results compared with 43% of patients without periodontitis ($P < .05$). Fifty patients harbored *H pylori* in the stomach. Within these 50 patients, those with periodontitis had a higher prevalence of *H pylori* in the stomach (n = 37) than those without periodontitis. Conversely, 29 of the same 37 patients with periodontitis harbored *H pylori* in their plaque samples, whereas only 4 of the 13 subjects without periodontitis harbored *H pylori* in their dental plaque ($P < .05$). The authors suggest that the oral cavity and dental plaque could act as reservoirs for *H pylori* and therefore act as a potential source for reinfection after eradication treatment. As such, they propose that a combination of professional medical and dental treatment modalities be instituted for patients with *H pylori* infection.

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