


The effect of continuous positive airway pressure and mandibular advancement device on sleep bruxism intensity in obstructive sleep apnea patients

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Abstract

We aimed to evaluate and compare the effects of continuous positive airway pressure (CPAP) and mandibular advancement device (MAD) in reducing the intensity of sleep bruxism (SB) in patients with obstructive sleep apnea (OSA). Forty-eight adults with OSA were subjected to single-night full polysomnography (PSG) in the Sleep Laboratory of the Wrocław Medical University. The respiratory events and bruxism episodes were scored according to the standards of the American Academy of Sleep Medicine. The patients were assigned to the CPAP treatment or the MAD treatment in accordance to apnea–hypopnea index (AHI). The second PSG examination was conducted during the MAD or CPAP treatment to assess the effect of treatment on bruxism episode index (BEI) and AHI. The mean AHI and mean BEI in the study material were estimated to be 30.05 ± 15.39 and 5.10 ± 5.31 , respectively. The bruxism parameters were significantly decreased in both the CPAP and MAD groups. Compared to the MAD, the CPAP treatment was more effective in reducing AHI; however, there was no significant difference in effectiveness of CPAP and MAD treatment in BEI reduction. Both CPAP and MAD treatments were effective against SB coexisting with OSA. Due to the application of these treatment options, the risk of OSA should be estimated in patients with SB.

Keywords

sleep bruxism, obstructive sleep apnea, sleep-related breathing disorders, mandibular advancement device, continuous positive airway pressure

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Introduction

Obstructive sleep apnea (OSA) is a common respiratory sleep disorder characterized by recurrent partial (hypopnea) or complete (apnea) obstruction of the upper airway during sleep, which results in intermittent hypoxia, arousals, and sleep fragmentation.¹ Recently, it has been estimated that globally 936 million adults, aged 30–69 years, have mild-to-severe OSA.¹ OSA may coexist with other sleep disorders such as insomnia, periodic limb movement disorder,² parasomias,^{3,4} and sleep bruxism (SB).⁵ According to the

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American Academy of Sleep Medicine (AASM), SB can be defined as a “repetitive jaw muscle activity characterized by the clenching or grinding of teeth and/or bracing or thrusting of the mandible.”⁶ Furthermore, the international consensus on the assessment of bruxism has defined SB as a “masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals.”⁷ The exact prevalence of SB is difficult to determine due to the fact that most population studies use the data collected from self-reported questionnaires, while most bruxers are unaware of their condition.⁸ However, it is assumed that the prevalence of SB is 13%.⁹

A study reported that 33–50% of subjects with OSA also suffer from SB.⁵ A considerable number of risk factors have been identified for SB, which include stress, smoking, caffeine and alcohol consumption, anxiety, use of drugs,^{10,11} and OSA.¹² However, the effectiveness of the treatments used for the management of SB is still limited.¹³ SB treatment is usually symptomatic and is directed toward teeth protection and pain relief.^{14,15} Continuous positive airway pressure (CPAP) and mandibular advancement device (MAD) are two effective treatment options available for OSA;^{16,17} however, CPAP is more effective than MAD in reducing the respiratory events.^{18–20} Recently, our research group identified that diabetes,¹² hypertension, and hypoxia²¹ act as independent risk factors for SB. Among them, hypoxia may be effectively treated using the CPAP or MAD treatment. Therefore, in the present study, we aimed to assess the reduction in SB frequency with the CPAP and MAD treatment in patients with coexisting OSA and SB.

Material and methods

This prospective outcome study was conducted in accordance with the amended Declaration of Helsinki. The study was approved by the Ethical Committee of Wroclaw Medical University (ID KB-195/2017). Written informed consent was obtained from all the patients before inclusion in the study.

A total of 48 adults hospitalized in the Department and Clinic of Internal Medicine, Occupational Diseases, Hypertension and Clinical Oncology of Wroclaw Medical University were included in the study. All these subjects were clinically suspected to have OSA.

The general inclusion criteria were as follows: age between 18 and 90 years, clinical suspicion of OSA, and willingness to participate in the study. The inclusion criteria that were specific to the CPAP treatment were as follows: apnea–hypopnea index (AHI) >15 and consent to undergo this form of treatment. On the other hand, the inclusion criteria that were specific to the MAD treatment were as follows: AHI <15 or >15 and not willing to undergo the CPAP treatment.

The general exclusion criteria of the study were as follows: neurological disorders and/or neuropathic pain, treatment with or addiction to analgesics and/or drugs that affect the muscle functions and breathing, active inflammation, history of pharyngeal surgery for the treatment of OSA, active malignancy and severe mental disorders, and cognitive disability. The exclusion criteria that were specific to the CPAP treatment were as follows: central sleep apnea, obesity hypoventilation syndrome, chronic obstructive pulmonary disease, and congestive heart failure. The exclusion criteria that were specific to the MAD treatment were as follows: severe temporomandibular disorders, limited range of the lower jaw motions, less than eight natural stable teeth in one dental arch, severe gingivitis and/or periodontitis, increased teeth mobility, tooth decays, poor oral hygiene, massive malocclusions, and need for acute dental treatment.

All the included patients underwent a single-night full polysomnography (PSG; first examination) with NoxA1 (NOX Medical, Iceland) in the Sleep Laboratory of the Wroclaw Medical University. Scoring and analysis of the tests were carried out by a qualified, certified physician, following the AASM guidelines, using a Noxturnal system. Polysomnograms were assessed in 30 s epochs in accordance with the 2013 AASM standard criteria for sleep scoring. The PSG outcome variables included sleep latency (SL), total sleep time (TST), sleep efficiency (SE, %), and the percentages of N1 (sleep stage 1), N2 (sleep stage 2), N3 (sleep stage 3), and rapid eye movement (REM) sleep. Abnormal respiratory events were scored based on the pressure airflow signal evaluated in accordance with the standard criteria of the AASM Task Force.²² The arterial oxygen saturation (SpO₂) was measured using finger pulse oximetry. Apnea was defined as the absence of airflow for ≥10 s, while hypopnea was defined as a reduction in the amplitude of breathing by ≥30% for ≥10 s with a ≥3% decline in blood oxygen saturation or arousal.

SB was assessed using bilateral masseter electromyography (EMG) and by audio–video evaluation. Bruxism episode index (BEI) was scored according to the AASM standards.⁶ In addition, the three forms of EMG pathways—phasic, tonic, and mixed—were scored. The new SB episodes were scored after at least 3 seconds of stable EMG and when the activity was at least twice the amplitude of the background EMG.^{22,23} The electrodes were placed on the right and left masseter muscles to measure electromyographic activity. EMF bursts within 3 s were considered part of the same episode. Persistent episodes lasting > 2 s were rated as tonic bruxism and more than three cyclic EMG increases lasting 0.25–2 s as phase bruxism. Episodes combining the features of a tonic episode and a phase episode were assessed as mixed bruxism. Events that mimic sleep bruxism in EMG (e.g., coughing, swallowing saliva, or yawning) after video evaluation were excluded from BEI.

BEI is the number of episodes of bruxism (tonic, phasic, and mixed) recorded during the study per hour of sleep. The events during the awake state were not included in the BEI. Based on the BEI, the severity of SB is classified as follows: BEI <2 not significant, BEI = 2–4 mild, or moderate or BEI > 4 severe.

Participants with an AHI of >15 per hour were assigned to the CPAP treatment and were connected with an auto-adjusting CPAP machine (Autoset S8, ResMed, Abingdon, UK). The second PSG examination was conducted during the CPAP treatment in the Sleep Laboratory of the Wrocław Medical University. Automatic titration was performed to eliminate respiratory events.

Participants with an AHI of <15 per hour were assigned to the MAD treatment. For these subjects, protrusion was individually adjusted up to 65% of the possible maximum. The custom, titratable MAD was manufactured by a qualified dentist. The second PSG examination was conducted using MAD in the Sleep Laboratory of the Wrocław Medical University.

Participants who had an AHI of >15 and were not willing to undergo the CPAP treatment were also assigned to the MAD treatment. One patient, who was initially assigned to the CPAP treatment, withdrew after the first night of the treatment and was then assigned to the MAD treatment. The recruitment process is shown in Figure 1.

The second polysomnography with CPAP or MAD treatment was conducted 10–12 weeks after baseline polysomnography. In MAD group, the PSG was conducted a week after completed MAD adjustment. In CPAP group, the effect of CPAP was assessed during first night of device treatment.

Statistical analysis was performed using the Statistica 12 software developed by StatSoft (Dell Inc., USA). The normality of distribution of all the parameters was analyzed using the Shapiro–Wilk test. The significance of mean differences between two groups (intergroup differences) was determined using the standard Student *t*-test for the parametric data and the Mann–Whitney *U* test for the nonparametric data. The significance of mean differences for the dependent variables (measured before and during treatment) was determined using the Student *t*-test for the parametric data and the Wilcoxon signed-rank test for the nonparametric data. Correlation analysis was performed with Spearman's rank correlation test. The relationships between the analyzed variables were determined also by multivariable stepwise regression analysis. Statistical significance was set at $p < 0.05$ for all the tests.

Results

The mean age of all the participants was 56.42 ± 12.11 years, while the mean age of the participants in the CPAP and MAD group was 56.13 ± 12.26 and 56.89 ± 12.12 ,

respectively. Women constituted 34.04% ($n = 16$) of the entire sample. Hypertension was diagnosed in 61.70% ($n = 29$) of the participants, while diabetes and ischemic heart disease were diagnosed in 14.89% ($n = 7$) and 2.12% ($n = 1$), respectively. The mean AHI and mean BEI in the entire study group were 30.05 ± 15.39 and 5.10 ± 5.31 , respectively. Baseline AHI, oxygen desaturation index (ODI), and phasic bruxism were found to be increased in the CPAP group compared to the MAD group, while baseline BEI was similar in both groups. The baseline PSG parameters in the studied group are presented in Table 1, and the PSG parameters measured during the CPAP and MAD treatment are presented in Supplementary Table A. The bruxism parameters were found to be significantly decreased in both the CPAP and MAD group (Table 2). The effects of CPAP and MAD treatments on sleep and respiratory parameters are presented in Table 3.

Due to the baseline differences in the MAD and CPAP groups, the effects on sleep and respiratory parameters were compared in only patients with an AHI of >15 (Supplementary Table B). The CPAP treatment was found to be more effective in decreasing AHI, ODI, snoring, and mean O₂ saturation (Supplementary Table B, Figure 2), whereas the MAD treatment was more effective in decreasing BEI and phasic BEI (Table 4, Figure 3).

Furthermore, we studied the correlations between the differences in BEI and sleep and respiratory parameters in the entire studied group. We observed a negative correlation between the reduction in BEI (Δ BEI) and AHI (Δ AHI) ($r = -0.28$, $p = 0.04$), Δ BEI and Δ snoring ($r = -0.29$, $p = 0.04$), and Δ BEI and Δ SE ($r = 0.37$, $p = 0.01$). In addition, a positive correlation was observed between Δ BEI and Δ SL ($r = 0.30$, $p = 0.04$).

In the next step, we examined the correlation in the group with AHI >15. We observed a positive correlation between Δ BEI and an increase in minimal oxygen saturation ($r = 0.30$, $p = 0.05$), whereas a negative correlation between Δ BEI and an increase in average desaturation ($r = -0.31$, $p = 0.04$).

Then we conducted multivariable stepwise regression analysis and determined factors independent of the BEI and AHI decrease after treatment.

For the “BEI decrease after treatment” variable the following relationship model was obtained: BEI decrease after treatment = $0.041 \text{ age} - 1.207 \text{ diabetes} + 0.919 \text{ BEI before treatment} - 0.088 \text{ mean SpO}_2 \text{ before treatment} + 0.068 \text{ mean heart rate (HR) before treatment}$. Multivariable analysis showed that older age, higher BEI before treatment, and higher average pre-treatment HR are independently associated with greater BEI decrease after treatment. Diabetes and higher mean SpO₂ before treatment are independent factors associated with lower BEI decrease after treatment. The method of treatment (CPAP vs MAD) does

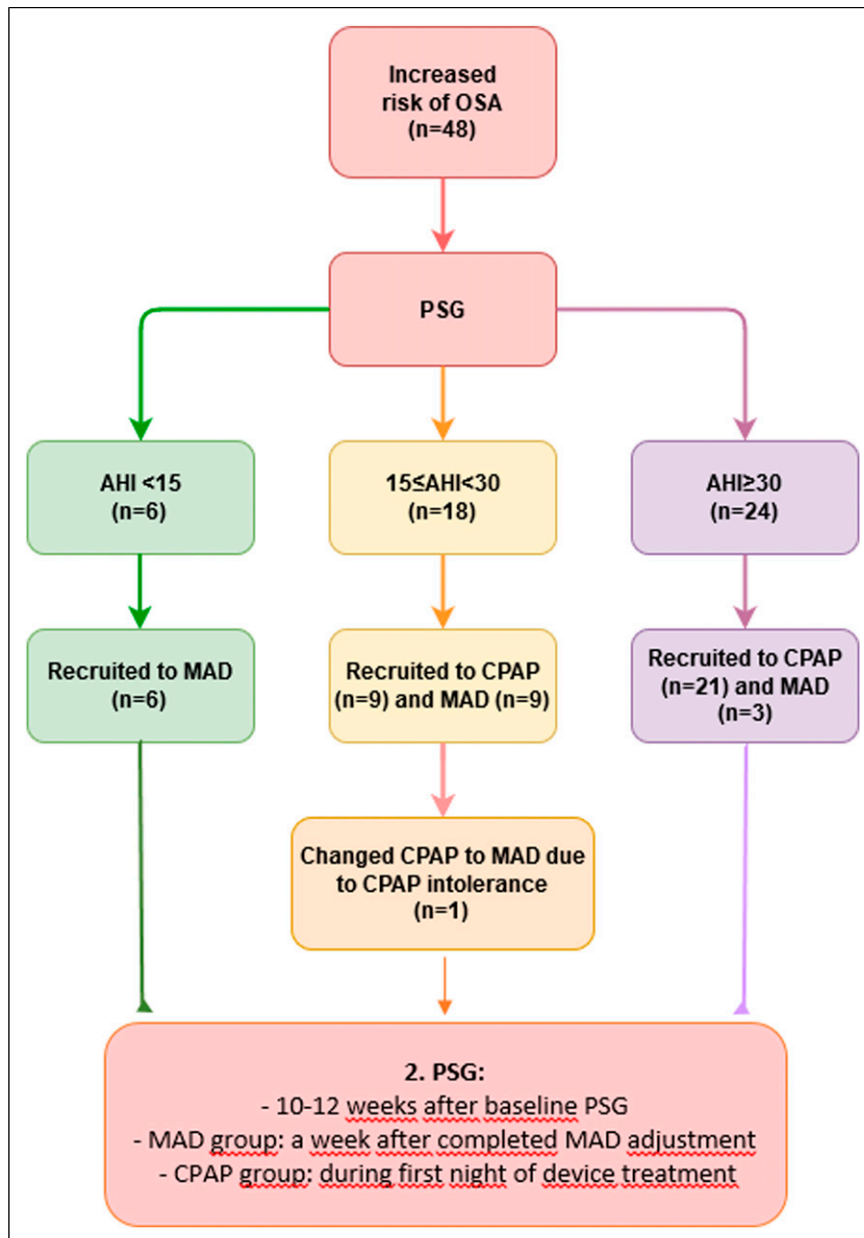


Figure 1. Scheme of subjects' recruitment to the CPAP and MAD groups. CPAP: continuous positive airway pressure, MAD: mandibular advancement device, AHI: apnea–hypopnea index, PSG: polysomnography, OSA: obstructive sleep apnea.

not affect BEI decrease after treatment independently of other factors.

For the “AHI decrease after treatment” variable the following relationship model was obtained: AHI decrease after treatment = 5.213 CPAP treatment (yes)/MAD treatment (no) + 8.482 male sex + 0.606 AHI before treatment—0.521 BEI before treatment—0.367 minimum SpO₂ before treatment. Multivariable analysis showed that for CPAP treatment (compared to MAD treatment) male gender and higher AHI before treatment are independently associated

with greater drops in AHI after treatment. Higher pre-treatment BEI and higher pre-treatment minimum SpO₂ are independent factors associated with smaller post-treatment AHI decreases.

Summarizing, based on multivariable stepwise regression analysis, it can be stated that the choice of treatment method (CPAP vs MAD) is significant in the context of the greater impact of CPAP on AHI decrease, while it is not significant in the context of BEI decrease ([Supplementary Table C](#)).

Table 1. Baseline polysomnographic parameters evaluated in the entire group, CPAP group, and MAD group.

	Total		CPAP, n = 30		MAD, n = 19		p value
	Mean	SD.	Mean	SD.	Mean	SD.	
BEI	5.10	5.31	4.16	4.73	6.60	5.95	0.087
Phasic BEI	2.77	4.22	1.82	3.73	4.23	4.61	0.003*
Tonic BEI	1.47	1.21	1.53	1.26	1.37	1.16	0.658
Mixed BEI	0.86	0.95	0.7345	0.86	1.06	1.07	0.246
AHI	30.05	15.39	36.5667	14.66	19.77	10.14	0.00008*
ODI	27.79	27.79	34.2345	14.38	17.97	10.53	0.0002*
Snoring (%)	37.72	37.72	42.1379	20.53	30.97	22.22	0.081
TST (min)	423.24	57.85	419.3286	65.59	429.01	45.16	0.579
SL (min)	23.06	24.11	17.6714	18.37	31.01	29.46	0.051
REML (min)	84.84	49.99	81.3893	53.63	89.94	45.01	0.404
WASO (min)	49.80	36.58	52.7893	37.64	45.39	35.49	0.448
SE (%)	83.34	8.29	82.7679	8.63	84.18	7.93	0.570
N1 (%TST)	6.28	5.76	7.1321	6.14	5.03	5.06	0.233
N2 (%TST)	48.86	11.44	50.0679	11.83	47.10	10.91	0.389
N3 (%TST)	23.54	10.04	22.3107	10.12	25.35	9.92	0.313
R (%TST)	21.30	6.78	20.4857	7.13	22.52	6.23	0.319
Ari	9.91	13.43	10.4714	15.01	9.08	10.85	0.879
Mean SpO ₂ (%)	92.34	1.70	92.2931	1.71	92.42	1.72	0.794
Min. SpO ₂ (%)	79.91	5.96	79.0690	5.92	81.21	5.94	0.228
SpO ₂ <90% (%)	12.17	16.69	12.4103	16.21	11.82	17.84	0.213
Mean desat. (%)	4.84	1.067	4.9690	0.86	4.64	1.31	0.176

CPAP: continuous positive airway pressure, MAD: mandibular advancement device, BEI: bruxism episode index, AHI: apnea-hypopnea index, ODI: oxygen desaturation index, TST: total sleep time, SL: sleep latency, REML: REM latency, WASO: wake after sleep onset, SE: sleep efficiency, N1: sleep stage 1, N2: sleep stage 2, N3: sleep stage 3, R: rapid eye movement sleep stage, Ari: arousal index, mean SpO₂: mean oxygen saturation, minute. SpO₂: minimal oxygen saturation, mean desat, mean desaturation; *p statistically significant.

Table 2. Effect of the MAD and CPAP treatment on sleep bruxism parameters.

	Baseline		On treatment		p value
	Mean	SD.	Mean	SD.	
CPAP n = 30					
BEI	4.16	4.73	1.71	1.54	0.0003*
Phasic BEI	1.82	3.73	0.62	0.86	0.056
Tonic BEI	1.53	1.26	0.76	0.68	0.0007*
Mixed BEI	0.73	0.86	0.34	0.36	0.001*
MAD n = 19					
BEI	6.60	5.95	1.06	1.24	0.0002*
Phasic BEI	4.23	4.61	0.47	1.04	0.0002*
Tonic BEI	1.37	1.16	0.45	0.47	0.0015*
Mixed BEI	1.06	1.079	0.12	0.12	0.0004*

CPAP: continuous positive airway pressure, MAD: mandibular advancement device, BEI: bruxism episode index; *p statistically significant.

Discussion

The most important observation of our study was the reduction of BEI in both the CPAP and MAD group. This confirmed that both CPAP and MAD might be effective in the management of SB. However, we have assessed the CPAP effectiveness in the

first single night, therefore, long-term effects can be different. There were no significant differences in effectiveness between these two treatment methods. Oksenberg and Arons as well as Martinot et al. described that severe OSA cases with bruxism were successfully treated with CPAP.^{24,25} Furthermore, different types of oral devices have been shown to reduce bruxism episodes.^{26,27} Some studies have confirmed the higher efficacy of MAD in reducing SB episodes compared to occlusal splints.²⁸⁻³⁰ However, the groups were commonly not large, CPAP was not studied, intraoral appliances were used because of occurrence other medical conditions, or study was not conducted in a sleep laboratory. Therefore, we cannot compare these results with those of the present study. Moreover, no large, reliable clinical trials have been carried out on bruxism management with CPAP and MAD thus far. In the present study, we have evaluated and compared the reduction in SB frequency with the CPAP and MAD treatment in patients with coexisting OSA and SB.

SB is a common sleep phenomenon, frequently co-occurring with other sleep disorders such as periodic limb movement disorder,³¹ catathrenia,³² sexsomnia,³³ and sleep-related breathing disorders.^{34,35} Although the relationship between OSA and SB remains controversial, the hypothesis that SB can act as a protective factor of OSA is fairly

Table 3. Sleep and respiratory parameters in the CPAP group ($n = 30$) and MAD group ($n = 19$).

	Baseline		CPAP		p value	Baseline		MAD		p value
	Mean	SD.	Mean	SD.		Mean	SD.	Mean	SD.	
AHI	35.95	14.53	6.22	9.01	<0.001*	24.12	7.78	10.00	5.81	<0.001*
ODI	33.65	14.30	9.82	10.77	<0.001*	22.27	8.64	10.92	6.53	<0.001*
Snoring	42.00	20.89	11.5071	10.77	<0.001*	37.54	22.21	25.47	19.18	0.006*
TST (min)	415.91	64.25	377.38	86.29	0.034*	427.23	46.79	402.84	33.14	0.102
SL (min)	17.67	18.37	40.13	47.45	0.004*	33.62	30.59	28.98	22.96	0.470
REML (min)	81.38	53.63	69.00	51.78	0.264	88.35	44.50	72.10	31.49	0.198
WASO(min)	52.78	37.64	68.01	62.85	0.613	55.32	36.38	55.54	41.48	0.9
SE (%)	82.37	8.54	71.81	18.06	0.002*	82.91	7.35	81.97	8.73	0.737
N1 (%TST)	7.13	6.14	4.00	4.97	0.014*	6.22	5.43	3.01	2.84	0.008*
N2 (%TST)	50.50	11.82	43.79	13.81	0.059	46.25	11.55	40.42	9.35	0.078
N3 (%TST)	21.99	10.17	28.62	11.69	0.015*	25.29	9.26	33.71	9.55	0.010*
R (%TST)	20.21	7.12	23.14	7.86	0.136	22.24	6.13	24.27	5.37	0.258
Arl	10.47	15.09	4.35	7.39	0.016*	8.55	7.70	3.09	2.68	0.003*
Mean SpO ₂ (%)	92.32	1.77	93.81	1.88	<0.001*	92.22	1.64	92.79	1.68	0.059
Min. SpO ₂ (%)	78.66	5.92	85.33	5.36	<0.001*	78.85	4.97	82.71	7.66	0.085
SpO ₂ <90% (%)	12.41	16.21	4.75	15.87	<0.001*	13.49	18.80	7.79	13.86	0.011*
Mean desat. (%)	5.02	0.86	3.66	0.45	<0.001*	5.10	1.23	3.97	0.72	0.003*

CPAP: continuous positive airway pressure, MAD: mandibular advancement device, AHI: apnea-hypopnea index, ODI: oxygen desaturation index, TST: total sleep time, SL: sleep latency, REML: REM latency, WASO: wake after sleep onset, SE: sleep efficiency, N1: sleep stage 1, N2: sleep stage 2, N3: sleep stage 3, R: rapid eye movement sleep stage, ArI: arousal index, mean SpO₂: mean oxygen saturation, minute SpO₂: minimal oxygen saturation, mean desat, mean desaturation; * p statistically significant

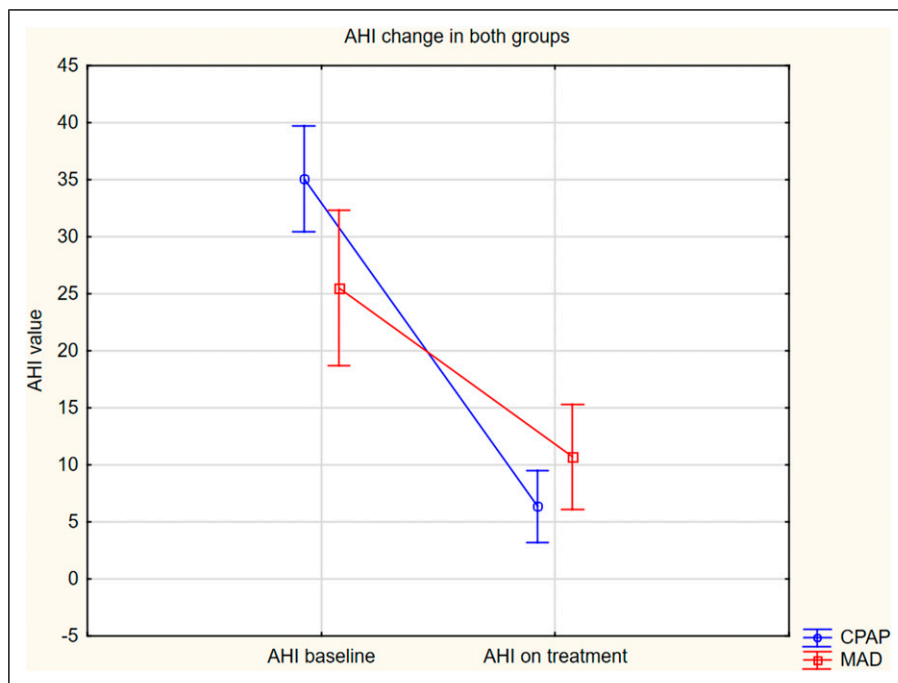


Figure 2. AHI reduction in the study groups. The graph present data from entire study group: CPAP group ($n = 30$) and MAD group ($n = 19$). CPAP: continuous positive airway pressure, MAD: mandibular advancement device, AHI: apnea-hypopnea index.

accepted.³⁶ Currently, there are ineffective treatments available for SB, and certain pharmacological, psychological, and dental strategies and rehabilitation techniques are used to

manage the condition. Because of its complex underlying mechanism, treatment schemes based on a single method are usually not sufficient to treat SB.

Recently, the relationship between SB and hypoxia has been receiving much attention. Hypoxia, which is considered as a new risk factor for SB, may be a promising treatment goal in patients with OSA and SB. Dumais et al. demonstrated that minor transient hypoxia is potentially associated with the onset of bruxism episode, independent of concomitant sleep arousal or body movements.³⁷ Interestingly, mild transient hypoxia observed before the onset of bruxism episode was not found to be linked to changes in the end-tidal CO₂.³⁸ Recently, our research team showed that AHI, lower mean SpO₂, and higher SpO₂ percentage (<90%) constituted independent risk factors for an increase in BEI.²¹ Considering these data, we looked for a relationship between the reduction of BEI and changes in the PSG parameters and observed that there were correlations between the reduction in BEI and AHI, SL, and SE in the entire study group. In the group with

AHI>15, we observed a positive linear correlation between BEI reduction and an increase in minimal oxygen saturation, whereas a negative linear correlation between BEI reduction and an increase in average desaturation. These observations suggest that improvement of oxygen saturation might be responsible for the reduction in BEI during the treatment of OSA; however, causality cannot be assumed and further studies are needed to clear the effect of oxygen saturation variability on bruxism intensity.

In the present study, we have observed reduction of frequency of AHI, ODI, snoring, arousal index (ArI), N1 duration (% of TST), percentage of sleep with oxygen saturation <90%, and mean desaturation value during the first night of CPAP treatment. CPAP also increased mean oxygen saturation, minimal oxygen saturation, and N3 duration (% of TST). However, SL was found to be increased and SE and TST were decreased compared to the first diagnostic PSG. This adverse effect of CPAP on sleep can be related to the fact that the assessment was made during the first night of the CPAP treatment. It is well known that CPAP tolerance may be poor on the first night of treatment. In the case of the MAD treatment, a reduction in AHI, ODI, snoring, N1 duration, ArI, percentage of sleep with oxygen saturation <90%, and an increase in mean desaturation and N3 duration were noted. However, no effect was observed on mean oxygen saturation. In addition, there was no adverse effect on SL, SE, and TST, which confirms the better tolerance of the MAD treatment, compared to CPAP.³⁹ Furthermore, the CPAP treatment was more effective in reducing AHI, while both treatment methods

Table 4. Comparison of the effectiveness (Δ of bruxism parameters) of the CPAP ($n = 30$) and MAD ($n = 19$) treatment.

	Total		CPAP group		MAD group		p value
	Mean	SD.	Mean	SD.	Mean	SD.	
BEI	-3.64	5.16	-2.44	4.549	-5.53	5.63	0.009*
Phasic BEI	-2.22	4.17	-1.22	3.69	-3.75	4.50	0.0004*
Tonic BEI	-0.83	1.03	-0.77	0.99	-0.92	1.10	0.80
Mixed BEI	-0.65	0.93	-0.45	0.80	-0.94	1.04	0.101

CPAP: continuous positive airway pressure, MAD: mandibular advancement device, BEI: bruxism episode index.

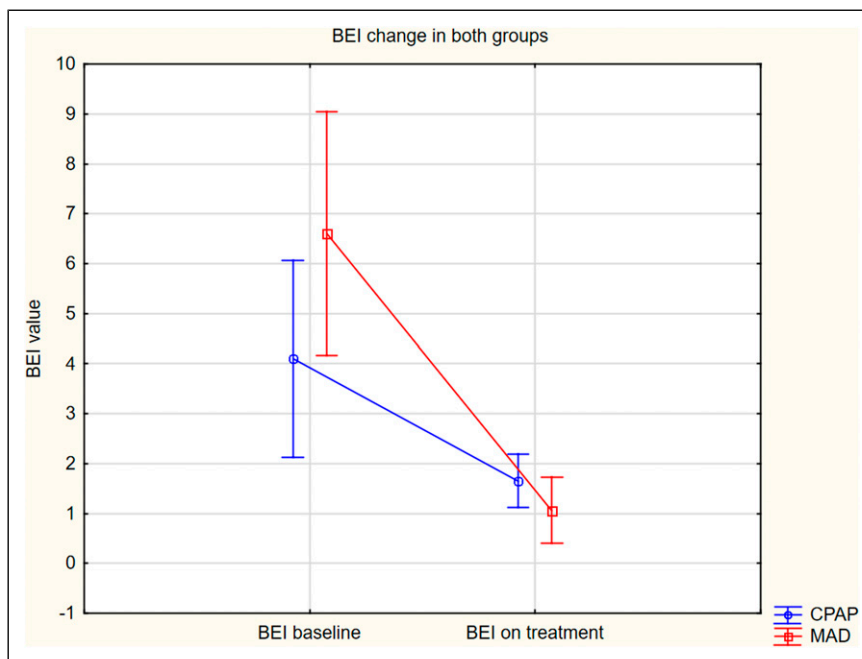


Figure 3. BEI reduction in the study groups The graph present data from entire study group: CPAP group ($n = 30$) and MAD group ($n = 19$). Caption: CPAP: continuous positive airway pressure, MAD: mandibular advancement device, BEI: bruxism episode index.

were similarly effective in reducing BEI. Both CPAP and MAD were effective in bruxism episodes count reduction; however, some residual SB episodes were observed. The clinical significance of residual SB episodes remains unclear and further studies are needed.

Although MAD is considered better tolerated treatment than CPAP, it may result in excessive salivation. On the other hand, sleep bruxism is concerned as protective mechanism of mouth dryness,⁴⁰ thus this side-effect of MADs might result in effective bruxism reduction. However, salivation was not investigated in this study and the exact mechanism supporting bruxism reduction remains to be explained.

The importance of the bruxism phenotypes is not well understood. Episodes of bruxism in SB patients are more often phased or mixed, while in healthy subjects tonic is more common.⁴¹ Increased severity of phasic bruxism was observed in hypertensives, while the tonic activity was similar in hypertensives and normotensives.²¹ The SB-type phase frequency correlated positively with the frequency of obstructive apnea and oxygen desaturation.³⁴ Therefore, phasic episodes may be associated with OSA and hypertension. In the present study, MAD was more effective than CPAP in total reduction of BEI and phasic BEI; however, multivariable stepwise regression analysis showed that both treatments were similarly effective in reducing bruxism episodes.

To our best knowledge, this is the first clinical study on the treatment of SB using CPAP, which demonstrated the effectiveness of CPAP and MAD in the management of this condition. However, the study has some limitations. First, the MAD group was relatively small and consisted mostly of patients with moderate OSA. Second, the efficacy of the CPAP treatment was measured on a single initial overnight study on the first night of treatment and we do not know if benefit would be sustained and therefore, the later outcomes might differ.

We cannot also exclude selection biases for patients opting for CPAP versus MAD treatment. Another serious limitation was lack of randomization. The patients in the CPAP and MAD groups were not matched for OSA severity.

Conclusion

Both CPAP and MAD might be effective in the reducing of SB events coexisting with OSA at the initial stage of using the device. CPAP is more effective in improving sleep and respiratory parameters. There is no statistically significant difference in effectiveness of MAD and CPAP in reducing the number of bruxism episodes. However, future studies are needed to assess the effectiveness of CPAP and MAD after following months of treatment.

Author Contributions

HM and MW contributed to the study conception; HM and MW designed the study; PM, TW, AW, JS, RP, PG, and HM were

responsible for data acquisition and analysis; HM, MW, RP, and TW interpreted the data; HM, MW, TW, and JS prepared the manuscript draft; MW, GM, and RS revised the draft; MW and HM supervised the study. All authors read and approved the final manuscript.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Supplemental material

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Trial registration

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Appendix

List of abbreviations

AASM	American Academy of Sleep Medicine
SB	sleep bruxism
OSA	obstructive sleep apnea
CPAP	continuous positive airway pressure
HR	heart rate
MAD	mandibular advancement device
PSG	polysomnography
AHI	apnea–hypopnea index

ODI	oxygen desaturation index
BEI	bruxism episode index
EMG	electromyography
ArI	arousal index
N1	sleep stage 1
N2	sleep stage 2
N3	sleep stage 3
REM	rapid eye movement
SE	sleep efficiency
SL	sleep latency
WASO	wake after sleep onset
REML	rapid eye movement latency