Comparison of Three Mandibular Advancement Device Designs in the Management of Obstructive Sleep Apnea: A Retrospective Study.

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Abstract

Study objectives: To evaluate the efficacy of three mandibular advancement device (MAD) designs in terms of apnea-hypopnea index (AHI), minimum oxygen saturation (min SpO_2), Epworth Sleepiness Scale (ESS) between pre- and post-treatment sleep studies for the management of obstructive sleep apnea (OSA). The protrusive range at the time of the second sleep study was correlated with the effectiveness of MAD designs.

Methods: Retrospective data of 49 OSA patients referred to Orofacial Pain Clinic at University of Kentucky (March 2016-March 2021) treated with MAD, with a post-treatment sleep study, were included. Treatment success was defined as 50% improvement in AHI, or as 50% improvement with residual AHI < 10/h.

Results: Post-treatment AHI improved with Herbst and SomnoDent-Classic (*p*=.003 and =.000). Post-treatment ESS improved with Herbst, SomnoDent-Classic and D-SAD (*p*=.004, .000, .018). No differences were found between the three MADs in terms of change in AHI, min SpO_2 and ESS (all *p's* >.050). Treatment success was achieved in 59.18% and 48.97%, according to the criteria, with no difference between the appliances and no correlation with the range of protrusion.

Conclusions: The three MADs were efficacious in improving AHI and ESS, but not min SpO_2, in OSA patients, with no differences between MADs designs. As some patients showed a worsening in AHI, a post-treatment sleep study is of utterly importance.

Clinical Implications: MAD should be selected on an individual basis, according to patient preference and trained dentist recommendation, as the design did not affect the effectiveness in terms of AHI improvement.

Keywords: Obstructive Sleep Apnea, Mandibular Advancement Device, effectiveness, design.

Introduction

Obstructive sleep apnea (OSA) is the most common sleep-related breathing disorder, with a prevalence varying between 10-17% in male and between 3-9% in female adults.[1] Treatment options for OSA include behavioral modification, such as weight loss, alcohol avoidance, positional therapy;[2] surgical intervention, hypoglossal nerve stimulation;[3] pharmacological regimen;[4] continuous positive airway pressure (CPAP) and oral appliances (OAs). CPAP therapy is considered the gold standard for the management of OSA, particularly in severe OSA disease.[5,6] Routine CPAP use remains problematic for some patients, due to mask discomfort, claustrophobia, pressure intolerance, noise, nasal congestion, rhinorrhea, eye irritation, sense of suffocation, and lifestyle or social considerations.[7] Unfortunately, its effectiveness is highly dependent on patient adherence, which accounts for 4 hours/night on average.[8]

OAs are recognized as an effective alternative treatment in the management of mild-moderate OSA, primary snoring or in case of lack of tolerance to CPAP machine.[9] OAs intent to protrude and stabilize the mandible to maintain a patent airway during sleep.[9]

Among OAs, mandibular advancement devices (MADs) are the most commonly prescribed in the management of OSA. Several studies have shown that despite the inferior efficacy of MAD compared to CPAP in reduction of apnea-hypopnea index (AHI), both treatments are similar in terms of effectiveness, because the suboptimal efficacy of MAD therapy is counterbalanced by the higher compliance of the patients.[10] Likewise, MADs are more effective than other types of OAs in treating OSA.[11] A systematic review by Ahrens et al. found that all MADs improved polysomnographic indices when compared with inactive appliances,[12] and custom-made monobloc MADs were found to be more effective than thermoplastic monobloc MADs.[13] This suggests that the characteristics of MAD design can have an impact on effectiveness.

To the best of our knowledge, only few studies in the literature assessed the difference in effectiveness between the MAD designs, with conflicting outcomes: Verburg et al. found no significant difference between Somnodent-Flex and Herbst;[14] two studies found no difference between Herbst and Twin Block.[15,16] Interestingly, Gauthier et al. found that Silencer was more effective than Klearway,[17] and Rose et al. found that Karwetzky activator produced a higher reduction in respiratory disturbance index (RDI) compared to Silencor®.[18]

The aim of the study was to evaluate the efficacy of three different MAD designs, by comparing pre- and post-treatment sleep study, in terms of AHI, minimum blood oxygen saturation (min SpO_2), and Epworth Sleepiness Scale (ESS). We hypothesized that the MAD design would not have significant impact on the efficacy of OSA management. A secondary aim was to evaluate if the protrusive range at which MAD was set at the time of the second sleep study was correlated with the efficacy of the different MAD designs.

Materials and methods

Subjects

In this retrospective study, data were analyzed for all consecutive patients, between March 2016 and March 2021, referred to the Orofacial Pain Clinic at the University of Kentucky by a sleep physician for the management of OSA with a MAD. The included patients presented with a diagnosis of OSA confirmed by a sleep study (polysomnography, PSG, or home sleep apnea test, HSAT) (mild 5/h ≤ AHI < 15/h, moderate 15/h ≤ AHI < 30/h, severe AHI ≥ 30/h), and had undergone a second post-treatment sleep study (PSG or HSAT prescribed, interpreted and approved by a certified sleep physician) performed with MAD in situ, after obtaining a maximum subjective improvement or the maximum range of protrusion.

Data collection

Patients' demographic data, medical records and sleep parameters were collected.

Primary outcome measures:

- *Efficacy of MADs*, assessed by comparing AHI and min SpO_2 values at pre- and at post-sleep study with MAD.
- *ESS*, recorded at baseline before MAD delivery and on the follow-up visit before the post-sleep study with MAD.

Secondary outcome measures:

- *Treatment success*, established using two different criteria: first criterion was established as 50% reduction in AHI, with residual AHI < 10/h; second criterion was established as 50% reduction in AHI.
- *Treatment failure*, defined as an increase of AHI after MAD therapy.
- *Range of protrusion*, intended as percentage of appliance advancement after reaching the maximum subjective improvement, or the maximum range of protrusion.
- *Subjective improvement*, measured via questionnaire with visual analogue scale (VAS), and expressed as a percentage ranging from "no improvement at all" (0%) to "maximum possible improvement" (100%); it was recorded during the follow-up appointment prior to the post-sleep study.

Appliances

Three FDA-approved commercially produced MADs were used: Herbst (Great Lakes, Tonawanda, NY, USA), SomnoDent-Classic (SomnoMed, Sydney, Australia) and D-SAD (Panthera Dental, Quebec, QC Canada) appliances (Figure 1). All three designs are duo bloc, customized, titratable oral appliances, that fulfil the criteria of adjustability of mandibular protrusion, and limited lateral and vertical mandibular movement during sleep.[19] Each design presents a different attachment and propulsion mechanism. Herbst is characterized by an attached bilateral compression (bilateral push), SomnoDent-Classic by an unattached bilateral interlocking, D-SAD by an attached bilateral traction (bilateral pull). Appliance selection was determined based on the preference of the clinician and the patient. The initial protrusion was established between 60 and 70% of the maximum protrusion, and subsequent advancements were performed progressively every two weeks based on patient's tolerance. Once the maximum anatomical protrusion or the maximum therapeutic benefit at 100% subjective improvement were attained, the patient was referred to the sleep physician for re-evaluation of the MAD effectiveness.

Statistical analysis

Normality of distribution was tested with Shapiro-Wilk test. Assumption of homogeneity of variances was verified with Levene's test. Normally distributed continuous variables are presented as mean ± standard deviation (SD).

Herbst, SomnoDent-Classic and D-SAD appliances were categorized as independent variables; AHI, min SpO_2, ESS values as dependent variables. Paired samples *t*-test was used to compare pre- (T_0) and post-treatment (T_1) dependent variables for each appliance.

Pearson correlation examined the correlation between reported subjective improvement and range of titration; reported subjective improvement and post-treatment AHI; post-treatment ESS and post-treatment AHI; treatment success and severity of pre-treatment AHI.

One-way ANOVA (normally distributed variables) was performed to assess the correlation between appliance design and reduction in min SpO_2, treatment success and treatment worsening. Kruskal Wallis test (skewed variables) was used to assess the difference between appliance design and efficacy, and change in post-treatment ESS. Significance level was set at $\alpha = 0.05$. Data were analyzed with SPSS software (IBM SPSS Statistics for Macintosh, Version 27.000, IBM Corp, Armonk, NY).

Results

49 patients met the inclusion criteria and were included in the study. Demographic data and clinical features are presented in Table 1. 13 patients (26.53%) received Herbst, 30 (61.22%) SomnoDent-Classic, 6 (12.24%) D-SAD appliance. Patients treated with MAD Herbst were older compared to the other two groups, and all of them could not tolerate a previous CPAP therapy ($p < 0.05$) (Table 1).

Patients' BMI decreased by 0.30 kg/m^2 ± 6.11 during the observation period, although it was not statistically significant ($p = .727$, 95% CI -1.45 - 2.06) (Table 2).

Treatment efficacy

Mean baseline AHI was 23.44 ± 17.47 (range 6-88 events/h) (Table 2). An overall decrease in AHI by 13.24 ± 17.36 was noted, with a residual AHI with MAD of 10.20 ± 9.02 (95% CI 8.25 - 18.22). The difference between AHI at baseline and residual AHI with MAD was statistically significant ($p = .000$).

Kruskal Wallis test revealed no statistically significant difference between the three appliance designs (p = .753) (Table 2). AHI improvement was statistically significant for Herbst and SomnoDent-Classic (p = .003, = .000, respectively), and not statistically significant for D-SAD (p = .106) (Table 3).

Treatment success, defined as 50% improvement with residual AHI < 10, was achieved in 24 patients (48.97%): 7 with Herbst (53.84%), 15 with SomnoDent-Classic (50.00%), 2 with D-SAD (33.33%). Treatment success, defined as 50% improvement in AHI, was achieved in 29 patients (59.18%): 8 with Herbst (61.53%), 18 with SomnoDent (60.00%), 3 with D-SAD (50.00%). No statistically significant difference was found between the three appliance designs, according to both criteria (F(2,46), = .117, p = .890 and F(2,46), = .345, p = .710, respectively). Treatment success was not correlated with baseline severity of OSA (r(48) = -.128, p = .427).

The mean percentage of protrusion obtained at the end of titration was 91.77% of the maximum anatomical protrusion: 91.65% with Herbst, 90.74% with SomnoDent-Classic, 93.87% with D-SAD. There was no statistically significant correlation between range of protrusion and treatment success (r(45) = -.049, p = .747), post-treatment ESS (r(45) = .175, p = .250), and subjective improvement (r(42) = .092, p = .564).

Treatment worsening was noted in 6 subjects (12.24%), with no statistically significant difference between the appliances (F(2,46) = .186, p = .831), and no correlation with BMI change (r(49) = -.009, p = .952) (Table 4).

Minimum oxygen saturation

Change in mean min SpO_2 was not statistically significant, from a baseline of 84.02 ± 5.94 to a post-treatment of 84.98 ± 4.83 (p = .316) (Table 2). No significant difference in change of min SpO_2 at T_1 was found between the three appliances (F(2, 64) = .808, p = .219). Min SpO_2 changed by 2.14 ± 6.21 (p = .067) with SomnoDent-Classic, by 1.38 ± 5.95 (p = .594) with D-SAD, and by -1.94 ± 7.61 with Herbst (p = .375) (Table 3).

Epworth Sleepiness Scale

Overall, ESS statistically reduced in all three groups by an average of 4.36 ± 4.48, to a mean value of 5.72 ± 4.48 secondary to MAD therapy (p = .000). No significant difference was found between the different designs (p = .185) (Table 2 and 3).

Post-treatment ESS was not statistically significantly correlated with residual AHI (r(48) = -.146, p = .322), nor with subjective improvement (r(45) = -.183, p = .228).

Subjective improvement

Subjective improvement (81.25% with Herbst, 83.08% with SomnoDent-Classic, and 90.00% with D-SAD) was not statistically significantly correlated with range of protrusion (r(42) = .092, p = .564), nor with residual AHI (r(45) = .083, p = .590).

Discussion

The results of this study revealed no difference between the three MAD designs in terms of AHI, min SpO_2 and ESS values. All of them were efficacious in improving AHI and ESS at post-treatment, whereas the change in min SpO_2 was negligible and not statistically significant.

Treatment efficacy

This study analyzed three custom titratable OAs, reported in the literature to obtain better outcomes in AHI compared to non-custom OAs,[20] with a mean reduction of 13.89 events/h compared to a mean reduction of 6.28 events/h.[21]

In accordance with the findings of thirty-four RCTs,[10] our study revealed a statistically significant difference between pre- and post-treatment AHI, with a mean AHI reduction by 13.24 ± 17.36 events/h, comparable with studies in the literature (mean reduction by 13.60 events/h).[19]

Interestingly, the decrease in post-treatment AHI was significant for patients treated with SomnoDent-Classic and Herbst appliances but not with D-SAD. We attribute this lack of significant difference to the small number of patients treated with D-SAD.

No significant difference was found between the three appliances in respect to improvement in AHI. This result is in accordance with a wide body of the literature, which supported the findings that MAD

effectiveness is generally independent on design features.[11] Verburg et al. found no difference between 67 patients treated with Somnodent-Flex and 70 patients treated with Herbst (p = .608).[14] Similarly, Bloch et al. compared Monobloc, Herbst and controls, revealing that treatment with MAD was significantly more effective than controls, and that Monobloc was more effective than Herbst, although the difference was not significant.[15] Likewise, Lawton et al. found no significant difference between Herbst and Twin Block (p = .071), both improving AHI.[16] Conversely, a study by Rose et al. reported a higher effectiveness of Karwetzky activator compared to Silencor® (p < .010), although the two appliances differed not only on design but also on vertical and sagittal dimension.[18] This confirmation has a direct influence on our patient management in every day clinical setting. Indeed, if a precise MAD design was found to be more effective than another, the clinical decision would have been driven by this evidence. Instead, the results of the study suggest that the treatment should be tailored based on individual needs and personal preference, also considering the crucial role of the compliance of the patient in success rate, and the adoption of a precise design can take into consideration other clinical conditions, such as the presence of parafunctional activities (grinding), absent or tilted posterior teeth, and crowded anterior teeth, among others.[22]

Minimum oxygen saturation

The literature reported a modest improvement in min SpO_2 in OSA patients treated with OAs, with a mean value of 3.09% in a weighted analysis of 22 RCTs.[19] The values vary across the studies, with the greatest improvement reported by Hoekema et al., with min SpO_2 increasing by 13%.[11]

On the contrary, in accordance with some other reports,[7,8] the present study did not show a statistically significant change in min SpO_2 with MAD. Interestingly, Herbst population revealed a decreased by 1.94 ± 7.61 in the post-treatment, although this difference was not significant. Similarly, the change achieved with SomnoDent-Classic and D-SAD (2.14 ± 6.16 and 1.38 ± 5.95, respectively) did not reach the significance level. Concordant with the literature,[17,23] no significant difference was found by comparing appliance design and min SpO_2. We agreed with the explanation of Lawton et al., suggesting that the arterial blood SpO_2 level may be influenced by other factors, including supine sleeping position, individual low hematocrit, and heart failure.[23]

Epworth Sleepiness Scale

The ESS, an 8-item questionnaire assessing daytime sleepiness,[24] has been shown to positively correlated with OSA severity.[24] The present work found a statistically significant improvement in daytime sleepiness between pre- and post-treatment values, with the greatest change in the group treated with D-SAD. The reduction in ESS by 4.36 was slightly higher than what reported by a meta-analysis, where the mean reduction was 3.81.[19] However, the effect on daytime sleepiness is uncertain, particularly in subjects within the mildest spectrum of disease severity and in snorers, as some studies did not observe any significant improvements in daytime sleepiness when compared to placebo.[25]

Our study corroborates the findings of other reports[17,23] in that the improvement in ESS is not influenced by MAD design. This suggests that the crucial factor may be the mandible protrusion, rather than the distinctive propulsion mechanism of the different designs.

Treatment success

The definition of treatment success is variable in the literature and the success rate of OSA treatment with MAD can vary remarkably according to success criteria.[26] Hence, two sets of criteria, normally accepted as cut-off across studies,[27] were applied to effectively differentiate between success and failure.

When treatment success was defined as 50% improvement with residual AHI < 10, MAD successfully treated 24 patients (48.97%), which is in line with the range of 30-94% reported by a systematic review.[28] When treatment success was defined as 50% reduction in AHI, it was achieved in 29 patients (59.18%), in accordance with a meta-analysis which reported greater than 50% AHI reduction in 23 of the 25 RTCs included.[19] However, the broad variability of effectiveness suggests the lack of good and consistent predictors of successful MAD treatments. In the literature, treatment success has been correlated with degree of protrusion,[29] and inversely related to disease severity.[26] However, the present study did not find any correlation between range of protrusion and treatment success. Interestingly, the most protruded position was achieved in D-SAD group (*i.e.*, protrusion of 96.83%), which actually

coincided with a non-statistically improvement in post-treatment AHI. However, a larger sample size is warranted to replicate these results.

The sample of this retrospective analysis showed a heterogeneity disease severity at baseline: most of the patients exhibited a moderate OSA (46.93%), for which MAD is recognized as an effective treatment. Nevertheless, MAD therapy obtained promising results also in the group of patients diagnosed with severe OSA (22.44%), considering that only 2 patients (4.08%) had residual severe AHI in the post sleep study. Surprisingly, the most relevant improvements in AHI were found in 4 severe OSA patients. The most impressing case was a patient with a baseline AHI of 88 events/h, which reduced to 5 events/h after treatment.

This study confirmed the unpredictability of treatment success secondary to MAD;[30] indeed, nor appliance design, nor gender, nor range of protrusion, nor pre-treatment OSA severity could predict treatment success.

Moreover, for those patients who cannot tolerate CPAP therapy (73.46% in this study), MAD may constitute a valuable and encouraging option, besides surgical and pharmacological treatments. Besides the objective reduction in AHI with MAD, ultimately the goal of OSA therapy is to improve daytime somnolence, sleep quality and overall quality of life,[30] and at the same time to reduce health risks, including cardiovascular function and neurocognitive behavior.[9]

Importance of second sleep study

This study revealed some cases of AHI worsening following MAD therapy, even if the patients reported a subjective improvement. The percentage of treatment worsening was 12.24%, in line with the 14% reported in the literature.[30] A 12.24% of treatment worsening is a noteworthy finding. However, our analysis did not reveal any significant confounding factors, such as appliance design, BMI or OSA severity. Use of drug induced sleep endoscopy (DISE) before embarking in a specific OSA treatment might help identifying good responders to OA therapy.[31]

These results suggest that prescribing a second sleep study is therefore essential, as PSG-based definitions of success and subjective improvement reported by the patient do not always coincide. Thus,

if the end of titration is exclusively driven by subjective feedback, the patient may remain sub-optimally treated.

Strengths and limitations of the study

Our study is the first to compare three commercially available appliances, which differ for propulsion mechanism, design and attachment, by comparing polysomnographic parameters at baseline and with MAD in situ.

The present study is not exempt from some limitations:

- Small and heterogeneous sample size, especially in the group treated with D-SAD appliance due to the recent commercialization of the design.
- No power analysis nor randomization, due to the retrospective design of the study, which resulted in unequal distribution of subjects to different appliance designs.
- Heterogeneous disease severity at baseline.
- Heterogeneity in age distribution among the appliance designs due to insurance coverage. Patients over 65 years were usually limited to the Herbst design.
- No placebo group; however, MAD effectiveness in reducing apneic events and improving subjective daytime sleepiness is well established in the literature compared to placebo.[7]
- Due to the retrospective nature of this study, the device used to assess sleep parameters at baseline and with the MAD in situ varied among patients. All sleep studies were prescribed and interpreted by the treating certified sleep physician, as recommended by AASM.[32] However, it should be taken into consideration that HSAT and PSG have different specificity and sensitivity.

Conclusions

The three MAD designs demonstrated to be similarly efficacious in the improvement of AHI and ESS scores in the management of OSA, and to not differ in terms of change in min SpO_2. Based on the results on this study, some patients showed a worsening in AHI with MAD; hence, a post-treatment sleep study is of utterly importance to assess MAD efficacy.

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Ethical approval: the study was approved by the Institutional Review Board of the Office Research Integrity of the University of Kentucky (Lexington KY, USA), and therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

Abbreviations

OSA: obstructive sleep apnea

MAD: mandibular advancement device

AHI: apnea-hypopnea index

ESS: Epworth Sleepiness Scale

Min SpO_2: minimum oxygen saturation

CPAP: continuous positive airway pressure

OAs: oral appliances

RDI: respiratory disturbance index

PSG: polysomnography

HSAT: home sleep apnea test

VAS: visual analogue scale

SD: standard deviation

FDA: food and drug administration

BMI: body mass index

RCTs: randomized controlled trials

DISE: drug induced sleep endoscopy

AASM: American Academy of Sleep Medicine

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Figure Titles

Figure 1- Different designs of MAD: A) SomnoDent-Classic B) Herbst C) D-SAD.

Tables

Table 1- Demographic data and clinical features of patients treated with MADs (Herbst, SomnoDent-Classic and D-SAD).

	Baseline	MAD Herbst	MAD SomnoDent -Classic	MAD D-SAD	*p* value
Total (%)	49 (100)	13 (26.53)	30 (61.22)	6 (12.24)	
Male (%)	23 (46.93)	7 (53.84)	14 (46.66)	2 (33.33)	.720
Age (mean ± SD)	61.16 ± 9.60	69.54 ± 6.07	58.27 ± 9.43	57.50 ± 5.64	.001
BMI (mean ± SD)	31.75 ± 7.60	32.38 ± 7.60	30.65 ± 5.57	34.38 ± 4.63	.033
Neck circumference (mean ± SD)	15.38 ± 1.54	15.31 ± 1.93	15.30 ± 1.42	15.87 ± 1.66	.758
Previous CPAP (%)	36 (73.46)	13 (100)	19 (63.33)	4 (66.66)	.040
Severity of OSA (%)					.568
Mild	15 (22.44)	4 (30.76)	10 (33.33)	1 (16.67)	
Moderate	23 (46.93)	7 (53.84)	12 (40.00)	4 (66.66)	
Severe	11 (22.44)	2 (15.38)	8 (26.67)	1 (16.67)	

One-way ANOVA was used to compare the three groups.

BMI: body mass index; MAD: Mandibular Advancement Device; SD: standard deviation; CPAP: Continuous Positive Airway Pressure; OSA: Obstructive Sleep Apnea.

Table 2- Intra- and inter-group difference between pre- and post-treatment dependent variables in total patient population treated with MADs.

MAD therapy (Herbst + SomnoDent-Classic + D-SAD)	T_0 mean (SD)	T_1 mean (SD)	ΔT_0-T_1 mean (SD)	*p* value (paired *t*-test)	95% CI	*p* value (Kruskal Wallis test) between three MADs
AHI (/hour)	23.44 (17.47)	10.20 (9.02)	13.24 (17.36)	.000*	8.25 - 18.22	.753
Min SpO_2 (%)	84.02 (5.94)	84.98 (4.83)	0.96 (6.65)	.316	-2.87 - 0.94	.219
ESS	10.09 (5.81)	5.72 (4.48)	4.36 (4.30)	.000*	3.11 - 5.61	.185
BMI	31.75 (7.60)	31.44 (7.06)	-0.30 (6.11)	.727	-1.45 - 2.06	.416

Intra-group differences were analyzed with paired sample *t*-test; inter-group differences were assessed with Kruskal Wallis test. Dependent variables: AHI, min SpO_2, ESS.

MAD: Mandibular Advancement Device; T_0: pre-treatment; T_1: post-treatment; ΔT_0-T_1: pre- and post-treatment difference; SD: standard deviation; AHI: apnea-hypopnea index; min SpO_2: minimum oxygen saturation; ESS: Epworth Sleepiness Scale; BMI: body mass index; 95% CI: confidence interval of the difference; *$p < .05$.

Table 3- Paired sample *t*-test to compare dependent variables (AHI, min SpO_2, ESS) for Herbst, SomnoDent-Classic, D-SAD at T_0, T_1 and ΔT_0-T_1.

MAD design	N	Dependent variables	T_0 mean (SD)	T_1 mean (SD)	ΔT_0-T_1 mean (SD)	*p* value	95% CI
MAD Herbst	13	AHI (/hour)	18.97 (8.40)	8.92 (7.87)	10.04 (9.71)	.003*	4.17 - 15.91
		Min SpO_2 (%)	87.46 (5.60)	85.51 (6.53)	1.94 (7.61)	.375	-2.65 - 6.55
		ESS	11.11 (6.44)	7.84 (4.98)	3.26 (3.29)	.004*	1.27 - 5.26
MAD SomnoDent-Classic	30	AHI (/hour)	24.96 (20.04)	9.98 (9.21)	14.98 (20.40)	.000*	7.36 - 22.59
		Min SpO_2 (%)	83.07 (5.61)	85.21 (3.93)	-2.14 (6.16)	.067	-4.44 - 0.16
		ESS	9.03 (5.55)	5.00 (4.26)	4.03 (4.01)	.000*	2.50 - 5.56
MAD D-SAD	6	AHI (/hour)	25.59 (18.95)	14.11 (14.30)	11.47 (14.30)	.106	-3.53 - 26.48
		Min SpO_2 (%)	81.33 (4.45)	82.71 (5.02)	-1.38 (5.95)	.594	-7.63 - 4.86
		ESS	13.00 (5.17)	4.6 (3.38)	8.33 (5.88)	.018*	2.15 - 14.51

MAD: Mandibular Advancement Device; N: number of patients; T_0: pre-treatment; T_1: post-treatment; ΔT_0-T_1: pre- and post-treatment difference; SD: standard deviation; AHI: apnea-hypopnea index; min. SpO_2: minimum oxygen saturation; ESS: Epworth Sleepiness Scale; BMI: body mass index; 95% CI: confidence interval of the difference; *$p < .05$.

Table 4- Treatment success, treatment failure and number of patients with residual AHI > 30 at post-treatment for each appliance.

	N	Success (1^{st} criterion)	Success (2^{nd} criterion)	Failure	AHI > 30
MAD Herbst (%)	13	7 (53.84)	8 (61.53)	1 (7.69)	0 (0.00)
MAD SomnoDent-Classic (%)	30	15 (50.00)	18 (60.00)	4 (13.33)	1 (3.33)
MAD D-SAD (%)	6	2 (33.33)	3 (50.00)	1 (16.67)	1 (2.04)
Total (%)	49	24 (48.97)	29 (59.18)	6 (12.24)	2 (4.08)
p value		.710	.890	.831	

Inter-group differences were assessed with one-way ANOVA.

N: number of patients; AHI: apnea-hypopnea index; MAD: Mandibular Advancement Device.