Received: 3 March 2022 | Revised: 8 September 2022 | Accepted: 3 November 2022

DOI: 10.1111/joor.13392

ORIGINAL ARTICLE

JOURNAL OF ORAL REHABILITATION WILEY

Do dental parameters predict severity of obstructive sleep apnea and mandibular advancement device therapy outcomes? A pilot study

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Funding information
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Abstract

Background: Mandibular Advancement Devices (MAD's) are oral appliances commonly used in treatment of Obstructive Sleep Apnea (OSA). OSA severity and certain other factors, such as BMI and neck circumference, correlate with MAD therapy success. So far, the predictive value of dental parameters, such as dental profile, molar-classification, overjet, overbite, maximal retrusion, maximal protrusion and protrusive range, has not been fully investigated.

Objectives: We aimed to investigate whether dental parameters influence OSA severity and MAD therapy outcome and could therefore be helpful in phenotyping OSA patients. Furthermore, we studied the predictive power of dental parameters for OSA severity and successful MAD therapy. We hypothesise that specific dental parameters correlate with more severe OSA and with more successful MAD treatment.

Methods: We performed a cohort study, including OSA patients diagnosed by polysomnography (PSG). Dental parameters were collected. Objective treatment outcome was collected by performing a PSG with MAD after three months of therapy. Differences between OSA severity groups and MAD treatment outcomes were analysed and dental parameters were correlated between groups.

Results: The relation between dental parameters and OSA severity was analysed in 143 patients, fifty patients had a PSG with MAD in situ after a 3-month therapy. The median baseline Apnea Hypopnea Index (AHI) significantly reduced from 17.6 (8.7–29.3) to 11.1 (5.5–17.5). Overbite and maximal retrusion differed significantly between mild, moderate and severe OSA. Other dental parameters did not differ significantly between the groups, nor correlated with OSA severity or MAD treatment outcome.

Conclusion: In this study, no correlation between dental parameters and OSA severity or MAD treatment outcomes was found. Therefore, screening patients for OSA and MAD treatment outcome based on dental parameters is currently not possible.

KEYWORDS
mandibular advancement device (MAD), obstructive sleep apnea (OSA), polysomnography (PSG), treatment outcome

1 | INTRODUCTION

Obstructive sleep apnea (OSA) is a chronic disease. Treatment consists, amongst others, of oral appliance therapy, which protrudes the mandibula during sleep. Since dentists are in frequent contact with their patients, they may play a role in both screening and treatment of OSA patients.

Risk factors to develop OSA and predictors influencing successful treatment using mandibular advancement device (MAD) have been investigated. Morphological risk factors for OSA include anterior lower facial height, inferior position of the hyoid bone, narrow pharyngeal airway space and a convex or class II retrusive position of the mandible.[1,2] Predictors of MAD treatment outcome include Apnea Hypopnea Index (AHI), age, Body Mass Index (BMI), neck circumference, waist circumference, sleep stage and body position. Various factors have been suggested to contribute to MAD treatment outcome. Possible contributing factors are an increased collapsibility of the upper airway and minimal or no impairment in other non-anatomical variables such as poor upper airway muscle functioning, low respiratory arousal threshold or high loop gain.[3,4] A MAD is more successful among patients with mild OSA and a patent airway than in patients with more severe OSA. However, there is no consensus (yet) on predictors of MAD treatment outcome.[5,6] In addition, cranial base angle and the distance between the sella turcica and the deepest point in the posterior cranial fossa are suggested as potential predictors.[6] Furthermore, a pre-treatment anteriorly located soft palate or tongue base could possibly favour MAD treatment outcome.[7] Finally, large palatine tonsils or pronounced pharyngeal pillars leading to partial obscuration or compression of the tongue base could worsen MAD treatment outcome.[7]

Dental sleep medicine is an emerging discipline in dentistry, which includes the early detection and treatment of sleep-related breathing disorders (SDBs), such as snoring and OSA.[8] The role of the dentist could be twofold.

Firstly, there could be a role for the dentist in the early detection of patients with OSA since a patient generally visits the dentist once or twice a year. During the visits, intraoral and extraoral features could be measured and a history of severe snoring, and other complaints such as hypersomnolence could be mapped. Intraoral and extraoral features may be facial profile, molar occlusion, overbite, overjet, maximal retrusion, maximal protrusion, protrusive range and maximal mouth opening. During specific intraoral examination, a dentist may focus on: enlarged tonsils, large tongue, webbing of the soft palate (i.e. a flap of mucosa), a long and wide uvula, and palatal stenosis after palate surgery. In addition, general risk factors, such as a large neck and waist circumference and a high BMI, can be identified by the dentist.

Secondly, there may be a role for the dentist in treatment of OSA patients with MAD therapy. In this regard, it is remarkable that relatively little is known about the potential dental predictors for MAD treatment outcome, such as overjet, overbite, maximal retrusion, maximal protrusion, protrusive range and maximal mouth opening.

The aim of this study is to investigate whether clinical dental parameters such as profile, molar classification, overjet, overbite, maximal retrusion, maximal protrusion, protrusive range and maximal mouth opening in patients with OSA influence or predict OSA severity and MAD treatment outcome. Intuitively, dental overjet and overbite or maximal retrusion might also be prognostic for MAD treatment outcome. We therefore hypothesise that dental parameters, such as a more convex profile (Angle class II), more retrusive molar classification, and larger maximal retrusion, protrusive range, or overjet correlate with a higher OSA severity and favour MAD treatment outcome. The outcomes of this study may be helpful in early detection of OSA patients and responders to MAD therapy.

2 | MATERIALS AND METHODS

2.1 | Patient selection

We performed a cohort study, with inclusion of patients between January 2019 and July 2020. Patients who underwent Drug-Induced Sleep Endoscopy (DISE) at the Department of Otolaryngology, Head and Neck surgery of the OLVG (Amsterdam, the Netherlands) were selected for inclusion. Patients had to be 18 years or older, give written informed consent and be diagnosed with OSA confirmed by a polysomnography (PSG) (AHI≥5 events/h). Patients were included independently of OSA severity or obstruction level. Patients were excluded when diagnosed with Central Sleep Apnea (CSA), in case of earlier (failed) MAD therapy, or when their dental condition was insufficient to fit a MAD. Insufficient dental condition was defined by having less than eight teeth per jaw, having extensive periodontal disease or having extensive tooth decay. Patients with active temporomandibular disorders (TMD) were also excluded.

This study was performed in accordance with the ethical standards of the responsible committee on human experimentation and with the Declaration of Helsinki of 1975 and approved by the Medical research Ethics Committees United (MEC-U nr. NL66070.100.18). A written informed consent was obtained from each patient before enrolment.

2.2 | Interventions

2.2.1 | Polysomnography

All included patients had a full-night PSG at baseline, patients who were advised to use a MAD had another PSG with their MAD in situ at 3-month follow-up (EMBLA A10/Titanium, Medcare Flaga; and

SomnocreenTM, SOMNOmedics GmbH). Obstructive respiratory events were analysed according to the 2017 AASM criteria.[9] Total AHI, supine AHI, non-supine AHI, total sleep time (TST) in supine position and Oxygen Desaturation Index (ODI) were used for further calculations. Patients were divided into subgroups based on OSA severity; mild (AHI 5–15 events/h), moderate (AHI 15–30 events/h) or severe (AHI > 30 events/h) OSA.

2.2.2 | Orofacial examination

All patients underwent a clinical physical examination by the same dentist specialised in Dental Sleep Medicine before making a MAD. The facial profile classification, molar classification, overjet, overbite, maximal retrusion, maximal protrusion, protrusive range and maximal mouth opening, were scored. Facial profile classification is extra orally observed from a lateral view and defined as neutral (I), retrusive—convex (II) or protrusive—concave (III). Molar occlusion is intra orally observed as the contact of the upper and lower molars and defined as a neutral (I) distal (II) or mesial (III) occlusion. The maximal retrusion is defined as the most retrusive position of the mandible, independent of tooth contact, in which the condyles articulate in the anterior-superior position against the posterior slopes of the articular eminences. The maximal protrusion is defined as the most protrusive position of the mandible, independent of tooth contact. Both are measured using a George Gauge and scored as distance from end to end at incisal position in mm. The distance between the most protrusive and most retrusive position of the mandible, measured by the George Gauge, is defined as the protrusive range. Overjet is scored as horizontal overlap of teeth, overbite is scored as vertical overlap of teeth, also quantified in mm.[10]

2.2.3 | Treatment

A selection of patients was advised to start with MAD treatment. Patients received a TAP appliance (Thornton Adjustable Positioner, Airway Management Inc.) or a Somnodent appliance (Somnodent, Somnomed AG). Patients who were willing to participate on a further crossover trial with regard to different MADs were selected for the TAP appliance and also joined another study protocol. Patients who were not willing to join further research were selected for the Somnodent appliance. Treatment outcome was based on the outcomes of follow-up PSG, with MAD in situ, after three months of MAD therapy.

At baseline, mandibular protrusion was set at 50% of maximum protrusion. Patients were subsequently, instructed to advance their mandible until symptoms abated or until maximum comfortable protrusion was achieved. Maximum comfortable protrusion was determined at baseline by advancement of the mandible with MAD in situ until pain or discomfort in teeth, jaw or muscles occurred. From there, the device was retruded for one millimetre.

Complete MAD treatment success was defined as an AHI < 5. Partial success was defined as a reduction in the AHI of more than 50% and an AHI < 15. Patients were considered non-responders if they did not meet the criteria for complete or partial treatment success.

2.3 | Statistical analysis

Statistical analysis was performed using SPSS (SPSS Inc., version 22).

This is a secondary analysis of another prospective study, therefore no power analysis for this analysis was performed.[11]

Patients were divided into subgroups based on OSA severity and analysed differences and potential correlations in dental parameters between the subgroups. Furthermore, we divided patients into subgroups based on MAD treatment outcome and analysed differences and potential correlations in dental parameters between the subgroups.

Continuous variables were presented as mean and standard deviation (SD) if normally distributed, or as median and interquartile range (Q1–Q3) if the distribution was skewed. Categorical variables were presented in terms of proportions.

Descriptive statistics were used for continuous variables. Student t-test, in case of normal distributions, and Mann–Whitney U test, in case of skewed distributions, were used to calculate the difference between dental parameters in OSA severity groups and treatment outcome groups. A p-value of <.05 was considered to indicate statistical significance. For categorical variables, Chi-square test was used. For numeric variables, ANOVA test was used; in case of not normal distribution, Kruskal–Wallis test was used. To calculate correlations, Spearman correlation coefficient was used.

3 | RESULTS

3.1 | Baseline characteristics

In total, 143 patients were analysed. The majority of the patients were male (83.9%); the median (interquartile range) age was 50 (39–55) years and their mean (±SD) BMI was 27.2 ± 3.1 kg/m^2 (BMI > 30 kg/m^2 in 19.6%). Sixty-four patients (44.8%) were diagnosed with mild OSA, 56 patients (39.2%) with moderate OSA and 23 patients (16.1%) with severe OSA. The mean age was significantly lower in mild OSA (44.0 ± 11.8) compared to moderate OSA (51.3 ± 11.4) (p = .001). The mean BMI was significantly higher in severe OSA (29.3 ± 3.1) compared to mild OSA (26.7 ± 3.2) (p = .001) and in severe OSA compared to moderate OSA (27.0 ± 2.9) (p = .002). The mean neck circumference (cm) was significantly higher in moderate OSA (40.8 ± 2.9) compared to mild OSA (39.6 ± 3.0) (p = .029) and in severe 42.0 (41.0–44.0) compared to mild OSA (p = .001). The overall mean AHI at baseline was 16.0 events/h (10.4–25.0); the mean ODI at baseline was 18.8 events/h (13.2–29.2). The mean AHI,

AHI supine, AHI non-supine and ODI differed significantly between all the groups of OSA severity ($p < .00$). These values significantly increased with higher OSA severity. Minimum SpO2 was significantly lower in the subgroups with more severe OSA.

3.2 | Dental parameters

The overall mean overbite was 3.0 (1.0–5.0) mm; the mean overjet was 3.0 (2.0–4.0) mm. Maximal retrusion was −6.0 (−8.0 to −5.0) mm, and maximal protrusion was 6.0 ± 2.3 mm. There was a significantly larger overbite in the moderate OSA group than in the subgroups with mild OSA ($p = .03$) and severe OSA ($p = .002$). In addition, there was a significantly smaller maximal retrusion in the mild OSA group compared to the subgroup with moderate OSA ($p = .027$). The other dental parameters did not differ significantly between the subgroups (Table 1). There was no correlation between the dental parameters and OSA severity at baseline.

3.3 | Treatment

Out of the 143 patients, 85 patients were treated with a MAD in our hospital (Figure 1): Fifty-eight patients with a TAP appliance and 27 with a Somnodent appliance. Thirty-five of the 85 patients treated with a MAD did not have a PSG at three months: Twenty-one patients did not schedule the PSG while still wearing the MAD, 14 patients did not schedule a PSG due to MAD failure. Out of these 14 patients, four patients preferred another treatment, six patients were intolerant to wearing a MAD, one patient did not experience any effect, and four patients experienced too much discomfort and pain. Fifty patients completed follow-up at three months with MAD in situ; their mean MAD protrusion was 83% ± 17% of maximum protrusion.

After three months of MAD treatment, respiratory parameters, such as AHI, AI, AHI supine and ODI were significantly reduced (Table 2).

To investigate the influence of the dental parameters on MAD treatment outcome, patients were divided into three subgroups based on treatment outcome. No significant differences were found in dental parameters between complete success, partial success and non-responders groups (Table 3). Dental parameters also did not differ between the two types of MADs. In addition, the dental parameters did not correlate with treatment outcome.

3.4 | Correlations

No correlations were found between each of the dental parameters, OSA severity and MAD treatment outcomes, neither in the total baseline group of patients ($n = 143$), nor in the follow-up group with patients treated for three months with a MAD ($n = 50$). Furthermore, no correlation was found between the extent of protrusion and the different dental parameters. A linear regression model could not be calculated based on the included dental parameters.

TABLE 1 Dental parameters per severity subgroup

Baseline	Total ($n = 143$)	Mild OSA ($n = 64$)	Moderate OSA ($n = 56$)	Severe OSA ($n = 23$)
Profile (angle I, II, III) *n* (%)	I *n* = 80 (56%) II *n* = 57 (40%) III *n* = 5 (3%)	I *n* = 39 (61%) II *n* = 23 (36%) III *n* = 2 (3%)	I *n* = 28 (50%) II *n* = 26 (46%) III *n* = 2 (4%)	I *n* = 14 (61%) II *n* = 8 (35%) III *n* = 1 (4%)
Molar occlusion left side (I, II, III) *n* (%)	I *n* = 76 (47%) II *n* = 27 (19%) III *n* = 30 (21%) Missing *n* = 10 (7%)	I *n* = 33 (52%) II *n* = 11 (17%) III *n* = 16 (25%) Missing *n* = 4 (6%)	I *n* = 33 (59%) II *n* = 11 (20%) III *n* = 7 (13%) Missing *n* = 5 (9%)	I *n* = 10 (43%) II *n* = 5 (22%) III *n* = 7 (30%) Missing *n* = 1 (4%)
Overbite (mm)	3.0 (1.0–5.0)	2.0 (1.0–4.0)[a]	3.4 ± 1.8[a,b]	1.9 ± 1.8[b]
Overjet (mm)	3.0 (2.0–4.0)	2.6 ± 1.7	3.0 (2.0–4.0)	2.6 ± 1.7
Maximal retrusion (mm)	−6.0 (−8.0 to −5.0)	−6.0 ± 1.8[a]	−6.9 ± 2.4[a]	−6.1 ± 2.0
Maximal protrusion (mm)	6.0 ± 2.3	6.1 ± 2.3	5.7 ± 2.3	6.4 ± 2.1
Protrusive range	12.3 ± 2.4	12.1 ± 2.6	12.6 ± 2.2	12.5 ± 2.2
Maximal mouth opening (mm)	53.1 ± 7.2	53.0 ± 7.8	53.0 ± 6.9	53.4 ± 6.8

Note: Data presented as mean ± standard deviation, median (interquartile range), or number.

Molar occlusion was measured at the first molar on the left side. If absent, it was measured at the first molar on the right side. If absent, data were defined as missing.

For categorical variables, Chi-square test was used. For numeric variables, ANOVA test was used; in case of not normal distribution, Kruskal–Wallis test was used.

[a]Significant difference calculated between mild and moderate OSA.

[b]Significant difference calculated between moderate and severe OSA.

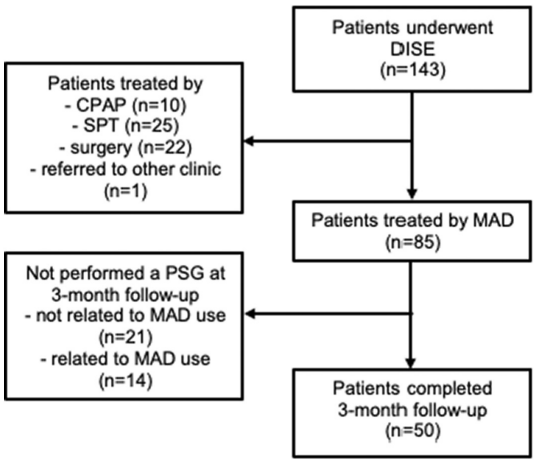

FIGURE 1 Flowchart of study. CPAP, Continuous Positive Airway Pressure; DISE, Drug Induced Sleep Endoscopy; MAD, Mandibular Advancement Device; PSG, Polysomnography; SPT, Sleep Positioning Trainer

TABLE 2 Treatment outcome with MAD at 3-month follow-up PSG

	Baseline (n = 50)	Follow-up (*n* = 50)
AHI (events/h)	17.6 (8.7–29.3)	11.1 (5.5–17.5)[a]
AI (events/h)	6.8 (2.5–14.7)	1.9 (0.4–6.0)[a]
AHI-supine (events/h)	33.5 (12.4–55.0)	14.7 (7.5–31.0)[a]
% TST supine (%)	38.9 (25.3–50.6)	41.3 (24.2–59.7)
AHI non supine (events/h)	8.5 (2.4–19.1)	5.4 (2.0–10.4)[a]
ODI (3%) (events/h)	24.0 (10.3–31.0)	10.0 (4.7–16.5)[a]
Minimum SpO2 (%)	85.5 ± 4.4	85.2 ± 10.1[a]

Note: Data presented as mean ± standard deviation, median (interquartile range).

Complete success (AHI < 5), partial success (AHI 5–15 with >50% reduction in AHI) and not successful (AHI > 15 or AHI < 50% reduction).

Student *t*-test was performed in case of normal distributions, Mann–Whitney U test was performed in case of skewed distributions.

Abbreviations: AHI, Apnea Hypopnea Index; ODI, Oxygen Desaturation Index; PSG, Polysomnography; SpO2, Oxygen Saturation Level; TST, Total Sleep Time.

aSignificant difference calculated between baseline and follow-up.

4 | DISCUSSION

The aim of this study was to investigate whether dental parameters influence or predict OSA severity and MAD therapy outcomes. No correlations between the included variables were found. However, the dental parameters overbite and maximal retrusion significantly differed between mild and moderate OSA (overbite and maximal retrusion) and between moderate and severe (overbite) OSA. Finally, no dental parameters were significantly different between no, partial or complete MAD treatment success. Other dental parameters such as profile classification, molar classification, overjet, protrusive range and maximal mouth opening did not differ significantly between the subgroups for OSA severity or MAD treatment outcome.

The fact that we found only a few dental parameters with significant differences between the groups could be related to the small treatment groups. We used data from another prospective study for which the power was calculated based on the difference between two measure instruments during DISE.[11] When building a linear regression model, at least 10 participants must be included per variable and correlations must be present.[12] After dropouts, this was unfortunately not the case in our study.

Based on literature, one would have expected that a larger maximal retrusion, protrusive range, or a class II profile or molar occlusion is predictive of OSA severity or MAD treatment outcome.[13] Unfortunately, we were unable to confirm this in our study. Differences in outcome could also be explained by the observation by visual inspection of the facial profile, overjet and overbite in our study as opposed to X-rays or photographs taken in other studies. In addition, a larger maximal retrusion, protrusive range or overjet have no predictive value about the retrolingual airway dimension. Possibly, our results may be influenced by to the generally increased use of orthodontic braces in patients in the Netherlands. Following orthodontics, many dental parameters, such as overjet and overbite, change. This might influence our observations. Unfortunately, we do not know if our participants had a history of orthodontic treatment.

Since we believe that our research questions are clinically relevant, and we believe our research is underpowered, we encourage further research in this field. For future research, questionnaires about orthodontic treatment are advised. In addition, additional dental findings could be investigated in further research. For example dental findings such as tooth-wear, abfraction, wear-facets, restorative and implant failures but also oral dryness, dental pain or sensitivity and Gastroesophageal reflux disease (GERD) which are associated with sleep bruxism.[14] Sleep bruxism is in turn associated with especially mild and moderate OSA.[15]

Significant differences between mild, moderate and severe OSA and dental parameters are found for overbite when comparing mild to moderate, and moderate to severe OSA. Also, in maximal retrusion significant differences are found comparing mild to moderate OSA. In literature, overjet, a large interdental width and mandibular retrognathia sometimes emerge as having a prognostic value to OSA severity.[5] However, similar to our group, in literature, mostly no correlation coefficients are mentioned.[6,16,17] Therefore, we could conclude that phenotyping OSA patients based on dental parameters such as overjet, overbite, maximal retrusion, protrusive range and maximal mouth opening is not yet possible.

It is important to elaborate on the included patients. All included patients were selected following DISE. DISE is not a standard procedure for patients diagnosed with OSA. DISE is only performed in case of CPAP or MAD failure or intolerance, or when upper-airway surgery or upper-airway stimulation is considered. Furthermore, previous MAD use was an exclusion criterion in the study. Therefore, the included study population is slightly different from the general OSA population. Secondly, the population in our study had a BMI < 32, which is lower than in the general OSA population. In our institution, a BMI > 32 is an exclusion criterion for DISE. This might

TABLE 3 Dental parameters per treatment responder group

Treatment outcome	Total ($n = 50$)	Non-responder (AHI > 15 and <50% reduction) ($n = 34$) (68%)	Partial success (AHI < 10 and >50% reduction) ($n = 5$) (10%)	Complete success (AHI < 5) ($n = 11$) (22%)
Profile (angle class I, II, III) *n* (%)	I $n = 29$ (58%) II $n = 20$ (40%) III $n = 1$ (2%)	I $n = 19$ (56%) II $n = 14$ (41%) III $n = 1$ (3%)	I $n = 4$ (80%) II $n = 1$ (20%) III $n = 0$ (0%)	I $n = 6$ (55%) II $n = 5$ (45%) III $n = 0$ (0%)
Molar occlusion (I, II, III) *n* (%)	I $n = 22$ (44%) II $n = 14$ (28%) III $n = 11$ (22%) Missing $n = 3$ (6%)	I $n = 15$ (44%) II $n = 11$ (32%) III $n = 6$ (18%) Missing $n = 2$ (6%)	I $n = 4$ (80%) II $n = 1$ (20%) III $n = 0$ (0%) Missing $n = 0$ (0%)	I $n = 3$ (27%) II $n = 2$ (18%) III $n = 5$ (45%) Missing $n = 1$ (9%)
Overbite (mm)	3.0 (1.8–5.0)	2.5 (1.8–4.3)	4.0 ± 2.0	2.7 ± 1.8
Overjet (mm)	3.0 (2.0–4.3)	3.0 (2.0–4.3)	2.0 (2.0–4.5)	2.8 (2.0–5.0)
Maximal retrusion (mm)	−6.0 (−8.0 to −5.0)	−6.0 (−8.0 to −5.0)	−7.2 ± 1.1	−6.1 ± 1.9
Maximal protrusion (mm)	5.8 ± 2.5	5.7 ± 2.7	5.6 ± 2.1	6.5 ± 1.9
Protrusive range	12.5 ± 2.4	12.4 ± 2.4	12.8 ± 1.5	12.6 ± 2.7
Maximal mouth opening (mm)	53.0 ± 7.3	53.0 (49.3–58.0)	49.0 ± 5.3	53.2 ± 7.9

Note: Data presented as mean ± standard deviation, median (interquartile range).

No significant differences were calculated between the different treatment success rates.

Molar occlusion was measured at the first molar on the left side. If absent, it was measured at the first molar on the right side. If absent, data were defined as missing.

For categorical variables, Chi-square test was used. For numeric variables, ANOVA test was used; in case of not normal distribution, Kruskal–Wallis test was used.

have affected the outcome of our study due to the larger tongue size and more lateral pharyngeal wall fat tissue in patients with a higher BMI.[18] In general, patients are eligible for primary MAD treatment with an AHI between five and 30 events/h.[19] In our study, the AHI varied from 5.1 to 85.6 events/h with a mean of 16.0 events/h. This might also have affected our study outcomes because MAD's generally perform better in mild to moderate OSA.

This study is a secondary analysis of a large prospective randomised controlled clinical trial comparing two measure instruments during DISE to predict MAD treatment outcome.[20] This means that the data used for this paper was originally collected for answering other questions. Therefore, the validity of the results might be questioned. Due to this secondary analysis, two different MAD brands were incorporated in the analysis. This might also influence validity. However, dental parameters between the two different MAD groups did not differ at baseline. Furthermore, the advantages of doing a secondary analysis are that it is timesaving, cost-efficient and patient friendly. In this paper, this might have resulted in a non-representative study population compared to the general OSA population. Nevertheless, the outcomes of this study help us to elaborate more on phenotyping OSA patients and develop further research.

The position of the MAD during the follow-up of this study was calculated being 83% of maximum protrusion. In literature, the most beneficial percentage of protrusion is 70–75% for severe OSA, and 50% for mild to moderate OSA.[21,22] The mean percentage of protrusion in our study was higher, however, still not all patients experienced beneficial effects with the MAD. This could be explained by the fact that patients were selected for MAD therapy independently of baseline physical characteristics or DISE outcome due to the initial study purposes. In the Netherlands, a follow-up PSG at three months is not standardly performed if the baseline AHI is <15 events/h. Therefore, not all of the 85 patients treated with a MAD had a follow-up PSG. Another limitation of this secondary analysis was that no information on adherence, snoring and self-reported outcomes were collected. In addition, unfortunately the COVID-19 pandemic could also have influenced the study results. Due to the pandemic, many clinic visits were postponed or done by telephone calls. Therefore, patient care and support were declined and patients could more easily dropout.

5 | CONCLUSION

In this study, we found no correlation between dental parameters and OSA severity or MAD treatment outcomes. Therefore, screening patients for success of OSA and MAD treatment based on dental parameters is currently not possible.

AUTHOR CONTRIBUTIONS

Study design: JUV, PB, AH, FL, NV. Data collection: JUV, PB. Data analysis: JUV, PB. Interpretation of results: JUV, PB, AH, FL, NV. Preparation of the manuscript: JUV, PB, AH, JP, FL, NV.

ACKNOWLEDGEMENTS

All authors have read and approved the manuscript. The authors affirm that this is an original manuscript, is unpublished work and is not under consideration elsewhere. This research was financially supported by Airway Management Inc. (no grand number given). This research was performed at the Onze Lieve Vrouwe Gasthuis (OLVG),

Amsterdam, the Netherlands. Data are available upon reasonable request to the corresponding author.

CONFLICT OF INTEREST

AH is a medical advisor for Airway Management Inc, SomnoMed and Zephyr Sleep Technologies. FL receives research grants from Sunstar Suisse SA, SomnoMed and Vivisol-ResMed and is an unpaid member of the academic advisory boards for GrindCare and for Oral Function (Sunstar Suisse SA). NV is a member of the Medical Advisory Board of NightBalance, consultant of Philips Healthcare, Inspire Medical Systems and Nyxoah. All other authors certify that they have no affiliations with or involvement in any organisation or entity with any financial interest, or non-financial interest in the subject matter or materials discussed in this manuscript.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/joor.13392.

DATA AVAILABILITY STATEMENT

Data is available upon reasonable request to the corresponding author.

INFORMED CONSENT

Research involving human participants. All procedures performed in this study involving human participants were in accordance with the ethical standards of the MEC-U nr. NL66070.100.18, and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. A written informed consent was given by all participants.

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How to cite this article: Uniken Venema JAM, Bosschieter PFN, Hoekema A, Plooij JM, Lobbezoo F, de Vries N. Do dental parameters predict severity of obstructive sleep apnea and mandibular advancement device therapy outcomes? A pilot study. *J Oral Rehabil*. 2023;50:203-209. doi:10.1111/joor.13392