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# Positional obstructive sleep apnoea and supine sleep time in mandibular advancement device treatment

Magnus Ahl<sup>1,2\*</sup> and Ola Sunnergren<sup>3,4</sup>

## Abstract

**Background** Many patients have positional obstructive sleep apnoea (OSA) with an apnoea-hypopnoea index (AHI) that is two-fold higher in supine sleep than in non-supine sleep. In these patients, the proportion of time spent in supine sleep influences the overall AHI. With the example of OSA patients treated with a mandibular advancement device (MAD), the aim of this study was to test the hypothesis that between-measurement differences in the proportion of supine sleep time are a significant factor affecting changes in AHI.

**Methods** One hundred sixty-five adult OSA patients treated with MAD were included in the study. Data on AHI, supine sleep time, age, sex, body mass index (BMI), Epworth Sleepiness Scale (ESS) scores, mandibular protrusion, patient-reported use, and adverse effects of the MAD were retrospectively collected from medical records or the Swedish Sleep Apnoea Register.

**Results** Among included patients, 27.3% (45/165) had both positional OSA at baseline and a  $\geq 50\%$  difference in the proportions of supine sleep time between baseline and follow-up. A generalized linear model showed that changes in the proportion of supine sleep time had a statistically significant impact on the change in overall AHI from baseline to follow-up of similar size as the effect the MAD.

**Conclusions** Changes in the proportion of supine sleep time are an important contributor to between-measurements changes in overall AHI in many patients treated with MAD. Positional OSA must be acknowledged not only in OSA diagnostics but also in MAD treatment follow-up.

**Keywords** Obstructive sleep apnoea, Mandibular advancement device, Supine position, Follow-up studies, Positional obstructive sleep apnoea

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## Introduction

Obstructive sleep apnoea (OSA) is a highly prevalent, chronic sleep-related breathing disorder (De Araujo Dantas et al. 2023) characterised by upper airway collapse during sleep, resulting in episodic total (apnoea) or partial (hypopnoea) reductions of airflow, blood oxygen desaturation, and disturbed sleep. The condition may lead to daytime sleepiness with increased risk of traffic and work accidents, loss of productivity, and a negative impact on social life. In the long run, OSA may result in hypertension and an increased risk of metabolic disease, cardiovascular events, and premature death (Ralls and Cutchen 2019). In clinical practice as well as in research, the apnoea-hypopnoea index (AHI) is the most used measure of OSA severity. Several studies have shown that many patients with OSA have more apnoea and hypopnoea episodes in the supine sleeping position, i.e. positional OSA. The phenomenon is explained by the collapse of the mandible, tongue, and other soft tissues of the upper airway in the supine position due to gravity, dynamic change in airway collapsibility, and effects on arousal threshold with positional changes in patients with positional OSA (Joosten et al. 2015; Landry et al. 2023). In these patients, there is a risk that sleep time in the supine position is a key contributor of the overall AHI. Previous publications have pointed out the risk of diagnostic misclassification of OSA severity if specific AHI values are not acknowledged for both the supine and non-supine sleep positions as well as the amount of sleep in the supine position (Landry et al. 2023; Yalciner et al. 2017; Cartwright 1984; Sunnergren et al. 2013). Overreliance on overall AHI may lead to misclassification that not only influences treatment decisions but also risks inaccurate assessment of treatment efficacy—an issue that has received limited, if any, attention in existing guidelines.

Mandibular advancement devices (MAD) are commonly used to treat OSA (Nationell vårdprogram för behandling av OSA hos vuxna 2021). It has been suggested that MAD treatment is more efficient in patients with positional OSA than in patients with non-positional OSA (Pintucci et al. 2024). In clinical practice, some MAD treatments, judged by the overall AHI, seem to be unsuccessful. However, based on a closer examination of the data, the unchanged overall AHI value may reflect an increase in the proportion of supine sleep despite decreases in both supine- and non-supine AHI values. The main aim of this study was to test the hypothesis that differences in the proportion of supine sleep time between diagnostic and follow-up full-night cardiorespiratory recordings (polygraphy—PG) influence changes in the overall AHI. A secondary aim was to evaluate the outcome of MAD treatment in a clinical cohort of OSA patients.

## Materials and methods

The study population was consecutively recruited at the Department of Otorhinolaryngology in Jönköping County, Sweden. Adult ( $\geq 18$  years) patients diagnosed with OSA based on clinical history (e.g. snoring, witnessed apnoeas, excessive daytime sleepiness, daytime fatigue, or non-restorative sleep) and PG results (overall  $AHI \geq 5$ ) were eligible for the study. All patients who initiated MAD treatment and underwent follow-up PG with the MAD in situ between August 2022 and September 2024 were included in the study. No specific exclusion criteria were applied. None of the patients had undergone previous OSA surgery, and none received any other treatment for OSA during the study period.

Data on age, sex, height, weight, BMI, mandibular protrusion at therapy start (i.e. mandibular protrusion with MAD and maximal possible mandibular protrusion from the retruded contact position), and adverse effects of the MAD (dental problems, temporomandibular joint (TMJ) problems, occlusal changes, other problems) were collected from the Swedish Sleep Apnoea Register (SESAR). All MADs were bi-block, titratable and custom-made by trained dentists. The treating dentist monitored each patient until optimal titration of the MAD was achieved, based on anticipated treatment efficacy and tolerability. Once optimal titration had been achieved, the patient was referred for follow-up PG to objectively evaluate treatment response. The duration of dental monitoring differed between patients owing to interindividual variation in titration requirements and clinical logistics.

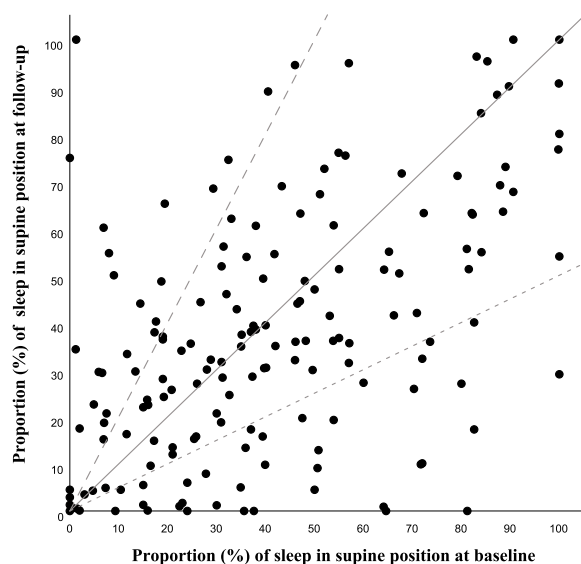
Patient adherence and compliance were assessed using standardized questions and reported to SESAR at the time of referral for follow-up PG. The following items were recorded: “On how many nights per week is the MAD used?” and “For what proportion of the night is the MAD used?” Response options for frequency were:  $\geq 5$  nights/week, 1–4 nights/week,  $< 1$  night/week, or not at all. Response options for nightly duration were: entire night (80–100% of total sleep time) or part of the night ( $< 80\%$  of total sleep time).

Sleepiness was measured by the Epworth Sleepiness Scale (ESS) (Johns 1991), with results collected from hospital electronic medical records (EMR). Data on overall AHI, supine AHI, and non-supine AHI from the diagnostic (baseline) and follow-up PGs, including the proportion of sleep in the supine position, were collected from EMR. All PGs were performed with the Nox T3 recording system (Nox Medical, Iceland). Respiratory events were manually scored according to Swedish national guidelines (SESAR Svenska Sömapnèregistret 2018) and in accordance with the recommendations of the American Academy of Sleep Medicine (Kapur et al. 2017). An apnoea episode was defined as a  $\geq 90\%$  reduction in the airflow amplitude from the nasal cannula for at least 10 s

with no desaturation required. A hypopnoea episode was defined as a  $\geq 30\%$  reduction of airflow for at least 10 s in combination with a desaturation of 3%. Sleep time was estimated based on the stabilisation of breathing patterns (nasal airflow and thoracic-abdominal movements), absence of body movements, and self-reported sleep onset and wake-up time (Cairns et al. 2014). Body position was registered by the built-in body position measurer in the Nox T3 device.

As sleep time cannot be directly determined without polysomnography (PSG), sleep time was estimated by the PG scorer based on patient-reported sleep onset and wake-up time, together with stabilization (for sleep onset) and destabilization (for wake-up) of respiratory and movement patterns in the PG signals, including body position and activity. This approach is supported by previous studies indicating that respiratory signal patterns and subjective sleep time may provide a reasonable approximation of PSG-derived sleep time (Svanborg et al. 1990; Franklin and Svanborg 2000). Thus, the term *sleep time* in this study refers to estimated sleep time.

Positional OSA was defined as a supine AHI at least twice the non-supine AHI (Cartwright 1984). Per the Swedish guidelines for follow-up of MAD treatment, treatment success was defined as a 50% reduction in overall AHI or a residual overall AHI  $< 10$  at follow-up PG (Nationellt vårdprogram för behandling av OSA hos vuxna 2021).



**Fig. 1** Individual proportions of sleep time in the supine position at baseline and follow-up. Dots below the solid diagonal line represent patients with a lower proportion of supine sleep time at follow-up. Dots over the dashed diagonal line represent patients who have more than doubled the proportion of supine time, and dots below the dotted diagonal line represent patients who more than halved the proportion of supine time ( $n = 164$ )

### Statistical analysis

SPSS software 29.0.2.0 (SPSS Inc.; Chicago, IL, USA) was used for statistical analysis.

Descriptive statistics, mean, and standard deviations were used for the presentation of data. Pre- and post-treatment differences were evaluated using a paired-samples t-test, with statistical significance defined as  $p < 0.05$  (two-tailed).

A generalized linear model with a log link function was used to examine the influence of supine and non-supine sleep time, age, sex, BMI, percentage protrusion, baseline overall AHI, and time from baseline to follow-up.

Missing values were excluded pairwise.

### Ethics

This study was approved by the Swedish Ethical Review Authority (Dnr 2024–01580-01). Written consent for this study was not required by Swedish law.

### Results

A total of 165 patients (64.2% men) met the inclusion criteria and were included in the study. Detailed information regarding the number and proportion of missing data points for each variable is provided in Appendix 1. The mean age at inclusion was 58.0 years (SD 11.24, min–max 24–79). The mean time from diagnostic PG to follow-up PG was 481 days (SD 325.5, min–max 145–2708 days). The mean time between the start of MAD treatment to the follow-up PG was 198 days (SD 102.1, min–max 19–550 days). All the MAD were biblock-type. Most patients were compliant with their MAD treatment, with 98.8% (162/164) reporting use of  $\geq 5$  days/week, and 94.8% (128/135) using their MAD for  $\geq 80\%$  of the night. The mean mandibular protrusion at referral for follow-up PG was 71.7% (SD 9.5, min–max 50–100%).

#### Differences in the proportion of supine sleep between diagnostic and follow-up measurements

As shown in Fig. 1, the majority of patients spent different proportions of total sleep time in the supine position at baseline and follow-up.

A significant proportion of the patients, 39.6% (65/164), had a  $\geq 50\%$  difference in the proportion of supine sleep time between the baseline and follow-up measurements. Of these, 16.5% (27/164) had doubled and 23.2% (38/164) had halved the proportion of supine sleep time.

#### Factors affecting the change in overall AHI from baseline to follow-up

AHI and sleep time were summarized separately for each subject in the supine position at baseline, non-supine position at baseline, supine position at follow-up and non-supine position at follow-up. For each of these four combinations of sleep-position and occasion, the number

of apnoea/hypopnea events per subject were initially assumed to follow a Poisson distribution with expected value equal to sleep time × intensity, where intensity was modeled as a function of age, sex, BMI, sleeping position, percentage protrusion, baseline overall AHI, and time from baseline to follow-up. Intensity was expressed as the number of apnoea/hypopnea events per hour of sleep.

The data were initially analyzed using a generalized linear model with log link function, log (sleep time) included as offset and only main effects specified. Observations with zero sleep time in supine or non-supine position were excluded.

The analysis was further developed. Overdispersion was clearly present in the data and was accounted for in the model. Time between baseline and follow-up was not statistically significant and was removed from the model. Baseline overall AHI showed a significant association and was retained in the model. The interaction between sleep position sleep position and percentage protrusion was tested but was not statistically significant and was therefore excluded. Although the main effect of sex was not significant, it was retained to satisfy the hierarchical principle, as the sex-by-sleep-position interaction was statistically significant. The results of the final model are presented in Table 1.

After adjustment for sex, age, BMI, baseline AHI, body position, and mandibular protrusion, non-supine sleep was associated with a 38% lower event intensity in women and a 57% lower intensity in men compared with supine sleep. A mandibular protrusion of 100% was associated with an approximately 60% reduction in intensity compared with 0% protrusion. Men exhibited a 26% higher event intensity than women. Event intensity increased by 3% per BMI unit, 1% per year of age, and 2% per unit increase in baseline AHI.

**Treatment effects**

At the individual level, 92.7% (153/165) of patients showed an improvement in overall AHI from baseline to follow-up (Fig. 2). The corresponding proportions for supine AHI and non-supine AHI were 85.4% (134/157)

and 84.2% (133/158), respectively (Figs. 3 and 4) supine OSA. Six patients, 3.6% (6/165) had exclusively positional OSA at both baseline and follow-up. At the group level, the mean supine AHI was more than twice the non-supine AHI both at baseline (44.0 vs 16.6) and follow-up (20.7 vs 9.1). Statistically significant decreases were seen in the mean overall AHI, supine AHI, non-supine AHI, estimated total sleep time and ESS score, while proportion of supine sleep time as a percentage of total sleep time and BMI did not change significantly (Table 2).

**Treatment success**

Treatment success, defined as a 50% reduction in overall AHI, was achieved in 61.8% (102/165) of the patients. Treatment success, defined as an overall AHI <10 (in patients with an overall AHI ≥ 10 at baseline, n = 164) at follow-up, was achieved in 51.8% (85/164) of the patients. If the criteria for success were applied to supine AHI and non-supine AHI, a 50% reduction from baseline was achieved in 61.7% (95/154) and 54.8% (86/157), respectively. If the AHI <10 criterion for treatment success was applied on supine AHI and non-supine AHI, the success rates were 34.0% (53/156) and 69.6% (110/158), respectively.

**BMI, ESS and adverse effects**

There were no marked individual changes in BMI from baseline to follow-up (Fig. 5). Regarding the ESS, 77.1% (118/153) had a lower ESS score, while 22.9% (35/153) had an unchanged or increased ESS score at follow-up (Fig. 6).

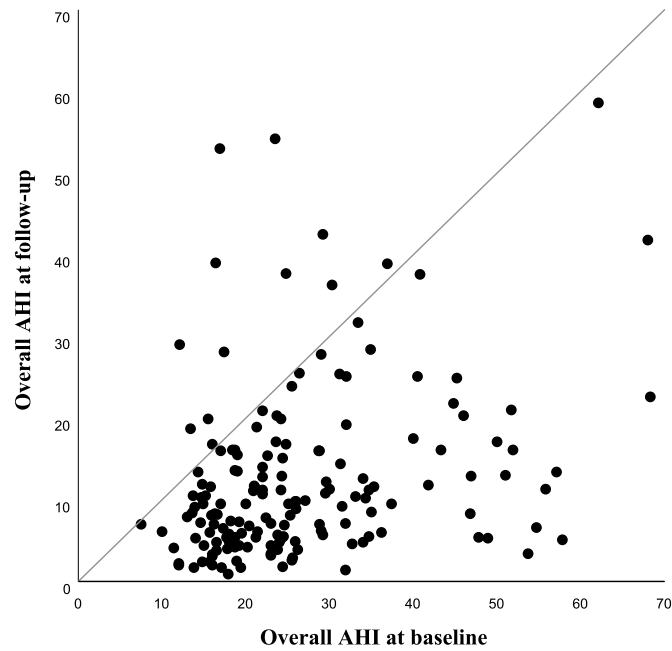
Adverse effects were reported by 21.7% (35/161) of the patients. TMJ problem was the most reported problem, 13.3% (22/165) followed by problems with teeth 6.7% (11/165), other adverse effect 3% (5/165) and occlusal change 0.6% (1/165).

**Discussion**

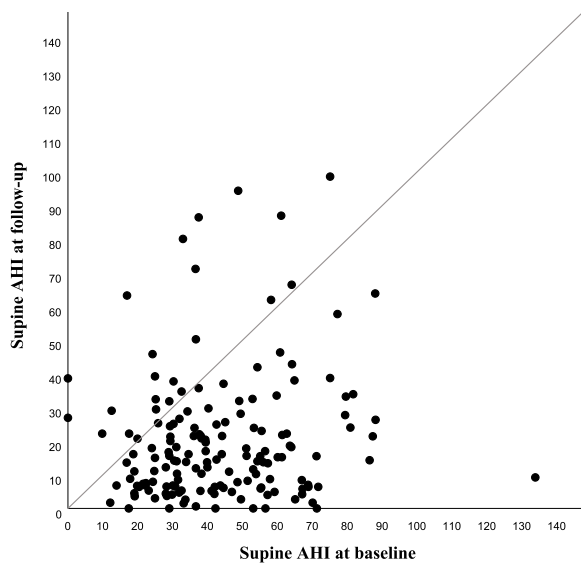
The main finding of this study was that we were able to confirm our hypothesis that intra-individual variability in the proportion of supine sleep time has a statistically

**Table 1** Results of the generalized linear model for overall AHI at follow-up. Coefficients are presented on the log scale; exp(B) represents multiplicative effect estimates

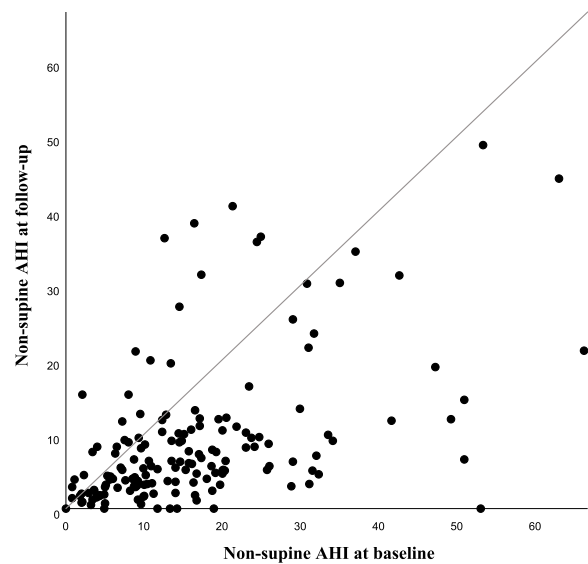
Parameter	B	Std Error	95% Confidence Interval		Exp(B)	Hypothesis Test
			Lower	Upper		Sig
(Intercept)	1.579	0.2835	1.024	2.135		< 0.001
Non-supine position	-0.474	0.0782	-0.627	-0.321	0.62	< 0.001
Mandibular protrusion 100%	-0.908	0.0896	-1.084	-0.733	0.40	< 0.001
BMI	0.025	0.0050	0.015	0.035	1.03	< 0.001
Age	0.008	0.0026	0.003	0.013	1.01	0.002
Sex	0.231	0.0666	0.100	0.361	1.26	< 0.001
Baseline AHI <sub>tot1</sub>	0.023	0.0018	0.019	0.026	1.02	< 0.001
Non supine position*sex	0.363	0.1000	-0.559	-0.167	0.70	< 0.001



**Fig. 2** Individual overall AHI at baseline and follow-up. Dots below the diagonal line represent patients with an improved overall AHI at follow-up ( $n = 165$ )



**Fig. 3** Individual supine AHI at baseline and follow-up. Dots below the diagonal represent patients with an improved supine AHI at follow-up ( $n = 157$ )



**Fig. 4** Individual non-supine AHI at baseline and follow-up. Dots below the diagonal represent patients with an improved non-supine AHI at follow-up ( $n = 158$ )

significant impact on changes in overall AHI from diagnosis to follow-up in OSA patients undergoing MAD treatment. Interestingly, and as shown by the linear model, the contribution of time spent sleeping in the supine position to the change in overall AHI was about the same as the effect of the MAD.

The clinical implication of these findings is that the overall AHI must be questioned as the main measure in OSA diagnostics and MAD treatment follow-up, especially as the majority of patients in clinical practice have

positional OSA. If changes in supine sleep time, supine AHI, and non-supine AHI are not taken into account, a patient with positional OSA who spent most of the night sleeping in the supine position at baseline and non-supine at follow-up may be considered a successful treatment even if both supine AHI and non-supine AHI are unchanged. This may mean that the patient is not offered another more effective treatment. Conversely, in a patient with positional OSA who spent most of the night sleeping in a non-supine position at baseline and most of the

**Table 2** Baseline and follow-up data on overall AHI, OSA severity, supine AHI, non-supine AHI, total sleep time, proportion of supine sleep time, ESS scores and BMI ( $n = 165$ )

	Baseline	Follow-up	Difference	p-value
No OSA (AHI < 5), n (%)	0 (0%)	35 (21.2%)	na	
Mild OSA (AHI 5–14), n (%)	19 (11.5%)	83 (50.3%)	na	
Moderate OSA (AHI 15–29), n (%)	99 (60.0%)	36 (21.8%)	na	
Severe OSA (AHI ≥ 30), n (%)	47 (28.5%)	11 (6.7%)	na	
Overall AHI mean, SD, (min–max)	26.4 ± 12.2 (7.5–68.3)	12.5 ± 10.6 (0.9–58.6)	-13.9 ± 13.5	< 0.001
Supine AHI mean, SD, (min–max)*	44.0 ± 20.3 (0.0–133.7)	20.7 ± 19.54 (0.0–98.5)	-23.3 ± 26.0	< 0.001
Non-supine AHI mean, SD, (min–max)*	16.6 ± 12.6 (0.8–66.2)	9.1 ± 9.5 (0.0–48.8)	-7.5 ± 11.4	< 0.001
Total sleep time, (minutes) mean, SD, (min–max)	417.8 ± 64.2 (240–600)	395 ± 70.8 (145–550)	-22.3 ± 83.9	< 0.001
Proportion of supine sleep time (%), mean, SD, (min–max)*	41.3 ± 27.7 (0.0–100.0)	37.3 ± 26.7 (0.0–100.0)	-4.0 ± 26.9	0.057
ESS score, mean, SD, (min–max)**	9.4 ± 4.7 (0–20)	7.2 ± 4.2 (0–20)	-2.2 ± 3.9	< 0.001
BMI, mean, SD, (min–max)***	30.6 ± 5.2 (21.4–51.4)	30.5 ± 5.1 (21.2–49.5)	0.06 ± 1.5	0.62

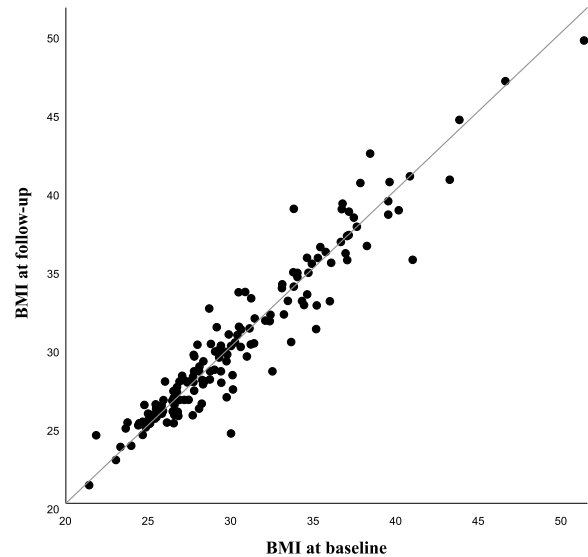
AHI apnoea-hypopnoea index, BMI body mass index, ESS Epworth Sleepiness Scale, na not applicable, OSA obstructive sleep apnoea, SD standard deviation  
 \*Patients with exclusively supine or non-supine sleep at either baseline or follow-up were excluded,  $n = 12$

\*\*ESS missing data  $n = 12$

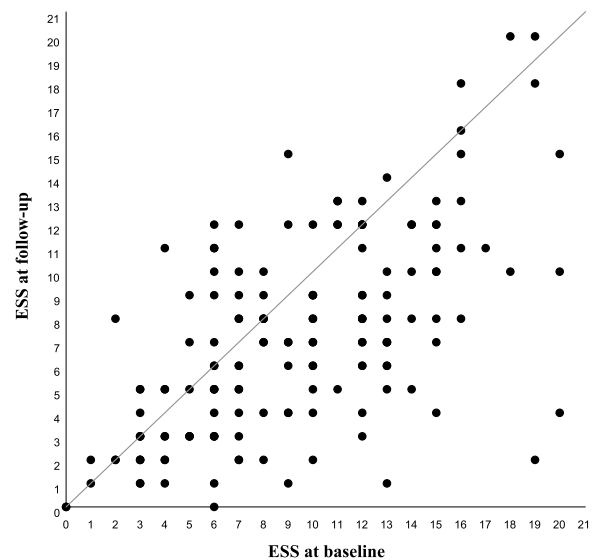
\*\*\*BMI missing data  $n = 14$

night supine at follow-up, the clinician may consider the MAD treatment unsuccessful and opt to switch to positive airway pressure (PAP) therapy or attempt to increase mandibular protrusion, resulting in reduced patient comfort and lower compliance, although adjunctive positional therapy may be a better option (Joosten et al. 2015; Landry et al. 2023). We believe that existing methodological approaches for the diagnosis and follow-up of OSA treatment, if fully applied, are sufficient to address the concerns raised in this study, namely the consistent use of position sensors and consideration of separate AHI values for different sleep positions.

The large intra-individual variability in the proportion of supine sleep time between baseline and follow-up,



**Fig. 5** BMI at baseline and follow-up. Dots below the diagonal line represent patients with a lower BMI at follow-up ( $n = 157$ )



**Fig. 6** Individual ESS scores at baseline and follow-up. Dots below the diagonal line represent patients with a lower ESS score at follow-up. One dot may represent more than one patient as some patients had the same scores ( $n = 153$ )

with 39.6% of the patients having a difference of  $\geq 50\%$  in our study (Fig. 1), corroborates previous reports. Yalciner et al. observed night-to-night variability in AHI in most patients and found that this variability was strongly associated with the amount of sleep in the supine position (Yalciner et al. 2017). In a study using peripheral arterial tonometry over three nights, Tcshopp et al. reported misclassification of OSA severity in 24% of the measurements compared with the average of all three nights and that the night-to-night variability could be partly explained by the time spent in the supine position. It was

also noted that the positional effect appeared to be more pronounced in mild-to-moderate OSA, whereas sleeping position appeared to have less of an impact on severe OSA (Tschopp et al. 2021). Contrary, Yo et al. reported stable night-to-night body position in 113 patients across three consecutive nights measured by a neck position therapy device at home without PSG apparatus (Yo et al. 2022).

A particularly interesting subgroup is patients with exclusively positional OSA (i.e. no OSA in non-supine positions). In our cohort, 14% (23/165) of the patients had exclusively positional OSA at baseline and 17.0% (28/165) at follow-up. Of these, 3.6% (6/165) had exclusively positional OSA at both PGs. It may be so that these patients could be offered positional therapy instead of MAD or PAP therapy (Richard et al. 2006) or supplementary treatment with a combination of MAD and a sleep position trainer device as proposed by Dieltjens et al. (Dieltjens et al. 2015).

The success rates in the present study, with 61.8% of patients reaching a  $\geq 50\%$  reduction in overall AHI and 51.8% reaching a residual overall AHI of  $\leq 10$ , were slightly lower than the success rates reported by Anitua (Anitua et al. 2023), where 69% had a  $\geq 50\%$  reduction in overall AHI and 75% had a residual overall AHI of  $\leq 10$ . However, the mean overall baseline AHI was higher in our cohort compared to Anitua et al. (26.4 vs 20.6), and 88% of patients in our study had moderate to severe OSA (AHI  $\geq 15$ ) at baseline, compared to 32% in the study by Anitua et al. These differences in baseline severity may partly explain the variation in reported success rates. Notably, previously published systematic reviews and meta-analyses using similar success criteria ( $\geq 50\%$  AHI reduction and/or residual AHI  $< 10$ ) have reported responder rates in the range of approximately 40–95% (JaM et al. 2021; Liptak et al. 2025). The success rates observed in the present study therefore fall well within the range reported in the literature.

Although 38.2% and 48.2% of patients did not meet the predefined success criteria of  $\geq 50\%$  reduction in overall AHI or residual AHI  $\leq 10$ , respectively, the vast majority demonstrated objective improvement: 92.7% improved their overall AHI, 85.4% their supine AHI, 84.2% their non-supine AHI, and 77.1% their ESS score. The substantial interindividual variability in treatment response (Figs. 2, 3, 4 and 6) underscores the importance of individualized follow-up during MAD therapy.

The effect on daytime sleepiness measured by the change in ESS score from baseline to follow-up in the present study is equal ( $-2.2$ ) to the mean reduction in ESS score in 3650 MAD-treated patients in the SESAR cohort (2015–2025) (SESAR Svenska Sömapneregistret 2025), but less than the mean ESS score reduction ( $-4.3$ ) in 13,732 CPAP-treated patients from the same register.

The ESS improvement in our study is slightly better than the mean improvement ( $-1.7$ ) in patients receiving MAD treatment in a review by Bratton (Bratton et al. 2015). The observed difference may reflect variations in patient populations across studies. Baseline characteristics, including BMI, OSA phenotype, and disease severity, may influence the magnitude of symptomatic improvement. Differences in clinical titration procedures may also have contributed. In our setting, MAD therapy was individually titrated and monitored until satisfactory efficacy and tolerability were achieved prior to follow-up PG. Given heterogeneity in study design and patient populations, direct comparisons should therefore be interpreted with caution.

The patients in our study reported limited adverse effects and had good compliance, with 98.8% of the patients reporting use of the MAD for  $\geq 5$  nights/week and 94.8% using the device for  $\geq 80\%$  of the night. These compliance rates are consistent with the long-term compliance rates reported by Vecchierini et al. (Vecchierini et al. 2021), where 91.3% of the patients used the device  $\geq 6$  h/night for  $\geq 4$  days/week after 5 years of treatment. In the present study, 21.7% of the patients reported overall adverse effects while using the device, and 13.3% reported TMJ problems. This is considerably lower than in a report by Chen et al., where 54.4% of patients had overall adverse events and 27.0% had TMJ dysfunction (Chen et al. 2022). One possible explanation for the relatively low rate of reported adverse events may be that all dentists providing MAD treatment had completed a certified course in OSA and MAD therapy approved by the regional healthcare authority (Region Jönköping County). Such training may contribute to careful patient selection and titration. However, as adverse events were not systematically evaluated in this study, this interpretation should be considered exploratory.

The generalized linear model in the present study indicated that a mandibular protrusion of 100% was associated with an approximately 60% reduction in AHI compared with 0% protrusion. However, the average effect in our cohort was likely smaller, given that the mean protrusion was 71.7%. However, Ma et al. (Ma et al. 2020) reported a non-linear, dose-dependent relationship between the reduction in overall AHI and the degree of mandibular protrusion. The dose-dependent relationship was more pronounced in patients with more severe OSA, with a plateau at approximately 70% of maximal mandibular protrusion. Papenfuss et al. reported that an increase from 60 to 80% of mandibular maximal protrusion even had a slightly counterproductive effect resulting in a slightly increased AHI (Papenfuss et al. 2024). Anitua et al. (Anitua et al. 2023) reported that effective treatment of OSA with MAD may be possible even with a limited mandibular protrusion by controlling vertical

mouth opening. Since the optimal mandibular protrusion giving a maximal therapeutic effect with minimal adverse effects seems to be highly individual, the 2015 AASM Clinical Practice Guideline for Treatment of Obstructive Sleep Apnoea and Snoring with Oral Appliance Therapy (Ramar et al. 2015) recommends that a qualified dentist use a customised, titratable device rather than a non-customised oral device. In the present study, the degree of mandibular protrusion was titrated by specially trained dentists in all cases.

### Strengths and limitations

A strength of this study is the analysis of real-world data from a consecutively recruited clinical cohort. Another strength is that all dentists involved in MAD treatment were specially trained and approved by the Unit for Dental Care Subsidy in Jönköping County, Sweden. The limitations of the study include the long period from diagnosis to follow up, in some patients up to 2708 days. However, the time between baseline and follow-up was not significant in the generalized linear model and the vast majority (90.3%) of the patients were followed up within two years of diagnosis.

The variability in follow-up time entails an increased risk that factors other than the treatment itself, i.e. the MAD device, such as the natural progression of OSA with age, may influence the observed change in AHI. In addition, inter-night variability in OSA severity represents another potential source of temporal confounding, as night-to-night fluctuations in respiratory events may affect AHI independent of treatment effects. However, since the main finding of the present study is that differences in supine sleep time between baseline and follow-up have a substantial impact on overall AHI in patients with supine OSA and that the treatment results are consistent with previous reports, we consider the long follow-up period to be of limited importance and we therefore choose not to exclude patients with long follow-up.

Another limitation is the use of PG instead of PSG, which is considered the gold standard for diagnosing sleep-related breathing disorders. Unlike PSG, PG does not include electroencephalography (EEG) and therefore cannot directly determine sleep time. In the present study, sleep time was estimated rather than directly measured using EEG. While this reflects established clinical practice and is supported by validation studies, some degree of uncertainty in sleep time estimation cannot be excluded.

Although PG tends to underestimate AHI compared to PSG, previous studies have demonstrated good agreement between PG and PSG in predicting the need for OSA treatment (Cairns et al. 2014; Xu et al. 2017; Pordzik et al. 2024; Ng et al. 2010). Moreover, PG remains the

recommended standard clinical procedure to evaluate OSA in all Scandinavian countries (SESAR Svenska Sömapnèregistret 2018). The use of PG in our study may be considered a strength as previous reports have shown that PSG increases the amount of supine sleep time and that the amount of supine sleep time may be even greater for laboratory PSG than at-home PG (Yo et al. 2022; Kukwa et al. 2022). Thus, using PSG would risk overestimating the overall AHI in positional OSA patients who are undergoing MAD treatment. It seems reasonable that PG performed at home, in the patient's own bed, gives a better reflection of their normal sleep.

Another limitation of this study is the absence of REM sleep data. This precludes examination of potential interactions between sleep stage and sleep position. However, as the primary aim of the study was to emphasize the importance of systematically considering sleep position, we consider the available data sufficient to support this objective. Future studies incorporating REM sleep analyses may further clarify stage-specific positional effects.

Swedish national guidelines recommend MAD therapy as a first-line treatment for patients with mild to moderate OSA, as well as for those who prefer it over PAP therapy. We acknowledge that this difference in treatment recommendations may influence the generalizability of our findings to settings where MAD is used primarily as a secondary treatment option (Nationellt vårdprogram för behandling av OSA hos vuxna 2021).

### Conclusion

Changes in the proportion of supine sleep, supine AHI, and non-supine AHI between diagnostic and follow-up PGs are all contributors to the change in overall AHI in patients undergoing MAD treatment. Positional OSA, with separate AHIs for each sleeping position and the proportion of supine sleep must be acknowledged both in OSA diagnostics and MAD treatment follow-up. All patients must be followed up individually during MAD treatment due to the large inter-individual variations in all AHI indices, and especially due to the between-measurements differences in the proportion of supine sleep time.

### Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s41606-026-00185-8>.

Supplementary Material 1.

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**Authors' contribution**

MA: Design of the study, project planning, ethical application, analysis of data, drafting, revising and final approval of the manuscript. OS: Conceptualization, design of study, project planning, ethical application, data collection, supervision, reviewing, editing and final approval of the manuscript.

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**Data availability**

Data cannot be shared directly by the authors due to Swedish law. However, data may be available after due submitted application to the entity responsible for the research (<https://www.rjl.se>).

**Declarations****Ethics approval and consent to participate**

The study was approved by the Swedish Ethical Review Authority, Dnr 2024-01580-01.

**Consent for publication**

Not applicable.

**Competing interests**

The authors declare no competing interests.

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