

# The incidence and prevalence of temporomandibular disorders and posterior open bite in patients receiving mandibular advancement device therapy for obstructive sleep apnea

Cristina V. Perez · Reny de Leeuw · Jeffrey P. Okeson · Charles R. Carlson · Hsin-Fang Li · Heather M. Bush · Donald A. Falace

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## Abstract

**Purpose** This study aims to evaluate the incidence and prevalence of temporomandibular disorders (TMD) in patients receiving a mandibular advancement device (MAD) to treat obstructive sleep apnea using the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD). In addition, it also aims to assess the development of posterior open bite (POB).

**Materials and methods** Data from 167 patients were evaluated at baseline, from 159 patients after 118 days (visit II), from 129 patients after 208 days (visit III), and from 85 patients after 413 days (visit IV). The presence of TMD symptoms was evaluated through a questionnaire. TMD signs were assessed using the RDC/TMD. Clinical evaluation assessed for the presence of POB.

**Results** The prevalence of TMD was 33/167 (19.8 %) at baseline. After an initial decrease to 14.5 % on visit II, the prevalence increased to 19.4 % on visit III and finally demonstrated a decrease to 8.2 % on visit IV. The incidence of TMD was 10.6 % on visit II. This decreased on further visits and only two (1.9 %) patients developed TMD from visit III to visit IV. POB was found to develop with an average incidence of 6.1 % per visit. The prevalence of POB was 5.8 % on visit II, 9.4 % on visit III, and 17.9 % on visit IV.

**Conclusion** The use of MADs may lead to the development of TMD in a small number of patients. Nevertheless, these signs are most likely transient. Patients with pre-existing signs and symptoms of TMD do not experience significant exacerbation of those signs and symptoms with MAD use. Furthermore, these may actually decrease over time. POB was found to develop in 17.9 % of patients; however, only 28.6 % of these patients were aware of any bite changes.

C. V. Perez (✉) · R. de Leeuw · J. P. Okeson  
Orofacial Pain Center, University of Kentucky,  
800 Rose St. D-530,  
Lexington, KY 40536, USA  
e-mail: cristina.perez@uky.edu

C. R. Carlson  
Orofacial Pain Center, Department of Psychology,  
University of Kentucky,  
800 Rose St. D-530,  
Lexington, KY 40536, USA

H.-F. Li · H. M. Bush  
Biostatistics, University of Kentucky,  
725 Rose St.,  
Lexington, KY 40536, USA

D. A. Falace  
Orofacial Pain Center, University of Kentucky, Retired,  
800 Rose St. D-530,  
Lexington, KY 40536, USA

**Keywords** Mandibular advancement device · Oral appliance · Temporomandibular disorders · Obstructive sleep apnea · Side effects

## Introduction

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder affecting approximately 12 % of the population of the USA [1]. During the sleep cycle of an OSA patient, there are episodes of little or no airflow to the lungs, despite continuing respiratory efforts. The restriction of airflow is due to intermittent obstruction of the airway caused by excessive collapsibility of a small airway during sleep. These repetitive lapses in respiration and the resulting lack of oxygen are associated with an increase in morbidity and mortality and contribute to the development of

numerous medical problems including stroke, cardiovascular disease [2, 3], hypertension [4], and type 2 diabetes [5].

Positive airway pressure (PAP) is the gold standard for treatment of OSA, providing a successful outcome in over 95 % of users [6]. However, as many as 50 % of patients cannot tolerate the use of PAP [12]. For many of those patients, mandibular advancement devices (MADs) are a valuable treatment alternative. MADs work by mechanically protruding the mandible, thereby lifting the tongue off the posterior pharyngeal wall. This is similar to the effect of the head tilt–chin lift maneuver used in cardiopulmonary resuscitation and results in an increased airway space with reduced resistance to airflow. Studies have demonstrated that MAD therapy can be used for patients with all degrees of OSA [8]. However, it is most predictably effective in mild to moderate OSA [9–11]. When compared to PAP, MADs are not as effective in reducing the apnea–hypopnea index (AHI) [7]. However, patients tend to favor the use of MADs over PAP [12]. Studies also show a normalization of oxygen desaturation as well as a decrease in the AHI with MAD use [13, 14]. In addition, MADs result in an improved quality of sleep with decreased subjective complaints of sleepiness and snoring [15]. They also provide improvement in blood pressure similar to PAP [16–19].

As with other forms of treatment, the use of MADs is not exempt from side effects [20, 21]. While relatively common, most of the side effects associated with MADs tend to be relatively minor and transient and do not usually prohibit the use of the appliances. Side effects include jaw discomfort, increased or decreased salivation, sore teeth, loose teeth, minor tooth movement, and changes in the bite or occlusion [22, 23].

The side effect of jaw discomfort may be due to temporomandibular disorders (TMD). TMD are defined as a cluster of signs and symptoms affecting the masticatory musculature, temporomandibular (TM) joints, and associated structures [24]. MADs protrude and maintain the mandible in a nonhabitual position during sleep, potentially affecting the harmony of the stomatognathic system with the possible development of signs and symptoms of TMD. Studies of the incidence/prevalence of TMD associated with the use of MADs are few [25–31]; therefore, additional data are needed.

Several studies have described occlusal changes when using MADs including palatal inclination of the maxillary incisors, labial inclination of the mandibular incisors, slight mesial drift of the mandibular molars, and loss of contact between the posterior teeth or posterior open bite (POB) [22, 32–38]. Studies indicate an incidence of occlusal changes ranging from 10 to 12 % in the MAD-wearing population [22, 36, 38–41]. Most studies have not specifically focused on POB, and therefore relatively little is known about its incidence and long-term effects. POB is thought to result

from the persistent anterior positioning of the mandible, even after the MAD has been removed, presumably due to persistent shortening of the lateral pterygoid muscles. Thus, when the patient attempts to bite in maximum intercuspation, the anterior teeth contact and the posterior teeth do not. It is not uncommon for patients to routinely experience this effect for a short time in the morning upon removal of the appliance; however, the bite typically returns to normal quickly [41]. In some instances, however, the effect may persist for hours or days and in some patients may become continuous [22, 37]. Interestingly, POB is often not noticed by the patient; rather, the patient may complain of cheek or lip biting or of heavy contact of the front teeth. If the condition occurs, there are simple jaw exercises that can often improve or reverse these effects [42].

This study aims to further understand the incidence and prevalence of TMD and POB during MAD therapy for treatment of snoring and OSA. The objectives of the study were:

1. To assess the prevalence of TMD at baseline and on each visit in patients undergoing MAD therapy using the RDC/TMD
2. To evaluate the incidence of TMD in patients who did not present TMD at baseline
3. To assess the prevalence of TMD in patients who presented with TMD at baseline as well as at any given time point and to evaluate the response of patients with TMD to MAD therapy
4. To assess the incidence and prevalence of POB associated to the use of MADs

## Materials and methods

**Patients** A retrospective analysis of data from 167 consecutive patients with a diagnosis of OSA was obtained from the Orofacial Pain Center of the University of Kentucky from 2003 to 2009. Patient demographics can be viewed in Table 1. Subjects were  $\geq 18$  years of age and presented with an initial AHI of  $\geq 5$  as determined by polysomnographic studies (PSGs). Standard PSGs were performed as requested by the referring physician. These evaluated sleep stages through electroencephalogram, electrooculogram, and submental electromyogram. The respiratory parameters studied were nasal airflow and pressure, oxygen saturation, and chest wall abdominal movement. Snoring was also recorded. An apnea event was defined as the cessation of airflow for  $\geq 10$  s. A hypopnea was defined as a reduction in amplitude of airflow or thoracoabdominal movement to  $\leq 50$  % of the baseline for more than 10 s. The AHI was defined as the number of apneas and hypopneas per hour of sleep. Patients were treated with either a hard Modified Herbst (Great Lakes Orthodontics) or Klearway appliance

**Table 1** Baseline demographic characteristics of patients (full sample, dropouts, and no dropouts) at baseline. Values are means  $\pm$  standard deviation

	Baseline values		
	Full baseline sample	Patients who did not dropout	Dropout patients
Number of patients, <i>N</i> (%)	167 (100 %)	85 (50.9 %)	82 (49.1 %)
Age (years)	54 $\pm$ 12.9	57 $\pm$ 13	51 $\pm$ 11.7*
Male/female, <i>N</i>	91/76	46/37	43/39
Apnea hypopnea index, ( <i>N</i> /h)	18.4 $\pm$ 18.6	18.5 $\pm$ 19	18.2 $\pm$ 18.9
Min O <sub>2</sub> saturation (%)	85.2 $\pm$ 5.8	85 $\pm$ 5.6	85 $\pm$ 6.1
Body max. index (kg/m <sup>2</sup> )	27.8 $\pm$ 4.4	26.9 $\pm$ 3.6	28.8 $\pm$ 5.0*
Tried C-PAP	91 (54.5 %)	50 (58.8 %)	41 (50.0 %)
Symptoms of TMD	54 (32.3 %)	24 (28.2 %)	29 (35.4 %)
Report of pTMD	30 (17.9 %)	9 (10.6 %)	20 (24.4 %)*
Signs of TMD	44 (26.4 %)	20 (23.5 %)	24 (29.3 %)
Signs of pTMD	22 (13.1 %)	9 (10.6 %)	13 (15.9 %)

Min O<sub>2</sub> saturation lowest oxygen hemoglobin saturation during sleep, pTMD pain-related TMD

\* $p \leq 0.05$  between dropout and no dropout patients

(Great Lakes Orthodontics). They reportedly wore the appliance for at least 5 nights a week. The MAD was initially delivered (baseline) at 60 to 70 % of the patient's protrusive capacity measured by a George gauge [43] (Peter T. George, Honolulu, Hawaii). Subsequent advancements were made as needed and were determined by changes in the patients' symptoms and the Epworth Sleepiness Scale. The decrease in symptoms and in the Epworth Sleepiness Scale defined the length of the titration period. Measures of successful treatment included the report of elimination or significant reduction of snoring, waking more rested in the morning, and less sleepiness during the day. Patients were evaluated on a weekly or every-other-week basis until they reported maximum benefit from the appliance. During each visit, patients were evaluated through clinical examination and standardized questionnaire. The information gathered during these visits was used to assess for signs and symptoms of TMD using the RDC/TMD criteria [44] as well as for other complaints, including POB and awareness of bite changes. Once patients attained the maximum benefits of the appliance, they were sent back to the referring sleep physician for a follow-up evaluation and/or polysomnography while wearing the appliance. They were then placed on 6-month recall. The present study was approved by the University of Kentucky Internal Review Board, Office of Research Integrity 10-0847-P3H.

The data used for this observational study was obtained from four time points during the patients' treatment. The cutoff for length of time using the MAD for this study was 3 years. The average times from the delivery of the appliance to visits II, III, and IV are listed on Table 2. Patients whose average times deviated largely from baseline to the last recall visit were considered outliers and were eliminated from the final analyses ( $n=7$ ). In addition, not all patients returned for their follow-up visits. Appropriate analyses

were performed to elucidate the impact of dropouts on the prevalence and incidence of TMD signs and symptoms.

**Variables** The presence of signs and symptoms of TMD and POB were evaluated at baseline, visit II, visit III, and visit IV.

#### Symptoms of TMD and awareness of bite changes

Patients were asked to fill out a standardized questionnaire before starting MAD therapy and on every subsequent visit after its use was initiated. The questionnaire was used to assess the report of TMD symptoms (joint pain, muscle pain, and joint clicks) and patient report of bite changes. The report of pain-related symptoms is noted as a prerequisite for groups I and III of the RDC/TMD criteria. For bite changes, "0" on the numerical rating scale indicated no bite changes and "10" indicated very severe changes in the bite. The longevity of bite changes was assessed on a five-point checklist: "less than 1 h", "1–6 h", "7–12 h", "13–24 h", and "never goes away". A reported bite change lasting less than 12 h was considered as transient.

#### Signs of TMD

The presence of signs of TMD was evaluated by examination using the RDC/TMD Axis I. Only the patients with report of pain were considered for group I and III of the

**Table 2** Time of each follow-up visit in days ( $\pm$  standard deviation) following delivery of MAD

	Mean	SD
Delivery—visit II	118.35	134.83
Delivery—visit III	208.68	152.25
Delivery—visit IV	413.78	208.38

RDC/TMD. The patients without pain-related symptoms were further analyzed for joint clicks. Examinations were standardized and performed by calibrated examiners. Approximately 2 lb of force was used when palpating masticatory muscles and 1 lb was used when assessing the TM joint. Joint clicks were assessed through palpation, feeling the transmission of these sounds on repetitive opening and closing. Pain-related TMD (pTMD) was defined as pain, as indicated by the patient, in the masticatory musculature or the temporomandibular joints when palpating these structures. Joint clicks were not included in this category considering that they do not necessarily represent a pathologic state of the TM joint.

#### Prevalence of posterior open bite

The presence of POB was determined by using 12- $\mu$ m shim stock foil (Artus Corp., Englewood, NJ, USA). Open bite was defined as “no occlusal contact” when the shim stock slid through the interocclusal area of the molars and premolars when the patient was biting in maximum intercuspation. If POB was observed, the patient was given a brief information session and taught a simple corrective jaw exercise. The exercise was to be done in the morning following removal of the MAD. The exercise consisted of placing a plastic 2-mm bite tab between the maxillary and mandibular incisors [42]. The patient was then instructed to protrude the mandible as far as possible and then to retrude the mandible as far as possible and, while in this maximally retruded position, to try to clench his/her back teeth for 5 s. This cycle was to be repeated six times. These instructions were repeated at each follow-up appointment as needed. Patients who presented POB at baseline were eliminated from the analysis as this was not a consequence of wearing the MAD.

#### Statistical methods

Archival data were used to evaluate the incidence and prevalence of signs and symptoms of TMD in 167 OSA patients using a MAD. Data obtained at baseline were compared with that of different time intervals up to maximum follow-up time. Continuous variables were summarized using descriptive statistics ( $n$ , mean  $\pm$  SD). Categorical variables were summarized using frequencies and percentages. Paired  $t$ -tests were used to test the differences between the baseline visit and the subsequent visits in continuous data. The McNemar test was used to test the differences between the baseline visit and the subsequent visits in categorical data. To evaluate the statistical significance of the incidence of TMD, pairwise comparisons were performed between the visits. Significance was set at  $\alpha=0.05$ . All analyses were conducted using SAS version 9.2 (SAS Institute Inc) and SPSS version 19.

## Results

The aim of the study was to present the incidence and prevalence of TMD using the RDC/TMD. The incidence and prevalence of POB during MAD treatment for OSA were also studied. At visit IV, 78.2 % of the patients reported that they were wearing their appliance more than 8 h a night, 7 nights a week. One hundred thirty-five of the 167 (80.8 %) patients used a hard Modified Herbst appliance.

The patients evaluated in this study presented variations in the amount of individual questions answered and examinations executed. Therefore, missing data will be reflected in an uneven amount of information for each visit. Consequently, not all denominators represent the total amount of patients on a particular visit.

One hundred sixty-seven patients were evaluated at baseline. Due to dropouts, this number decreased on every visit as follows: visit II, 159 patients; visit III, 129 patients; and visit IV, 85 patients. There were 82 patients (49.1 % of the original sample) that dropped out of the study by visit IV. These patients were demographically and clinically similar to the remainder of the study population at baseline except for age, BMI, and the report of pTMD (Table 2).

#### Dropout patient analysis

Further analyses were performed on the dropout population. These indicated that the highest number of findings were in the patients who came to visits I and II but did not return to visit III ( $N=31$ ) (early dropouts). The late dropout group was constituted by those who came to visits I, II, and III but did not return to visit IV ( $N=43$ ). The development and persistence of signs and symptoms of TMD were compared with the population that continued treatment. The results of the analysis demonstrated differences only in the early dropout group. Patients in the early dropout group reported the development and persistence of jaw joint pain as well as muscle pain. The analysis demonstrated that six of 31 (19.4 %) patients reported jaw joint pain present at baseline that persisted and increased to nine of 31 (29.0 %) on visit II. In this same group, five of 31 (16.1 %) patients reported muscle pain at baseline that persisted and increased to nine of 31 (29.0 %) on visit II. Neither of the two groups of dropout patients demonstrated persistence or development of signs of TMD from their clinical evaluations.

#### Symptoms of TMD by the RDC/TMD

At baseline, the prevalence of TMD symptoms was present in 54/167 (32.3 %) patients. One hundred five of 167 (62.8 %) patients did not report any symptoms of TMD at baseline. Only the patients with report of pain were

considered for groups I and III of the RDC/TMD. The patients without pain-related symptoms were further analyzed for joint clicks.

#### TMD by the RDC

At baseline, the prevalence of TMD using the RDC/TMD was 19.8 % (33/167 patients). One hundred thirty-two patients (79.0 %) did not present signs at baseline and were classified in the “no TMD” group. Within the TMD group, the most prevalent diagnosis was group II, disc displacement with 18 (10.8 %) patients (Table 3).

On visit II, 14/132 (10.6 %) patients developed TMD (incidence) while using the MAD. The incidence on visit III was similar to that of visit II with 14/130 (10.7 %) patients developing TMD. It then decreased on visit IV with only two of 104 (1.9 %) patients going from the “no TMD” group to the TMD group (Fig. 1). Pairwise comparisons demonstrated statistically significant differences in the decrease of incidence from visit III to IV. Figure 1 demonstrates the fluctuating nature of TMD in patients wearing a MAD. After an initial prevalence of 19.8 %, a decrease was observed in this diagnosis on visit II, an increase on visit III, and a final decrease to 8.2 % (seven of 85 patients) on visit IV. This demonstrates a decrease of 11.2 % compared to the previous visit. Sixteen of the 33 patients with TMD at baseline were present at the end of the study period.

#### Pain-related TMD

At baseline, the prevalence of pTMD (group I, III, and their combinations by the RDC/TMD) was 15/167 (8.9 %) (Table 3). Eight of the 15 patients (53 %) with pain-related pTMD were in the I+III group (muscle + joint pain). Seven of the 151 (4.6 %) patients developed pTMD (incidence) at

visit II. The incidence increased on visit III to 12/141 (8.5 %) and later decreased on visit IV to one of 113 (0.8 %) (Fig. 2). Pairwise comparisons demonstrate a statistically significant decrease in the incidence observed from visit III to IV.

At baseline, 15/167 (8.9 %) patients were diagnosed with pTMD. After a decrease on visit II and an increase on visit III, there was a final prevalence of two of 85 (2.4 %) on visit IV (Fig. 2). This demonstrates a 10 % decrease in the occurrence of pTMD from visit III to IV (Table 3). The 15 patients diagnosed with pTMD prior to treatment were followed. Figure 2 demonstrates that their signs fluctuate within the study period moving from the pTMD group to the “no pTMD” group. At visit IV, two of 15 initial pTMD patients continued using the MAD while remaining in the pTMD group. Three of the 15 patients who started in the pTMD group were still wearing the MAD and were then in the “no pTMD” group.

#### Joint clicks

At baseline, the prevalence of joint clicks (group II RDC/TMD) was 18/167 (10.8 %). Joint clicks decreased in prevalence from baseline as follows: 11/159 (6.9 %) on visit II, nine of 129 (7.0 %) on visit III, and five of 85 (5.9 %) on visit IV (Fig. 3).

#### Posterior open bite

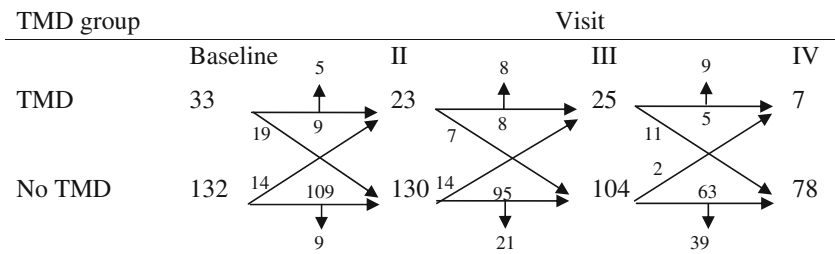
Patients with POB at baseline were eliminated from this analysis ( $N=12$ ). POB developed in eight of 149 (5.4 % incidence) patients on visit II. On visit III, eight of 130 (6.2 % incidence) patients had developed POB, and seven of 106 (6.6 % incidence) had developed POB by visit IV. On visit II, POB was found to occur in eight of 138 (5.8 %)

**Table 3** TMD signs assessed by RDC/TMD

RDC/TMD	Baseline Total, $n=167$	Visit II Total, $n=159$	Visit III Total, $n=129$	Visit IV Total, $n=85$
Muscle pain on palpation (group I myofascial pain)	4 (2.4 %)	8 (5.0 %) ↑ 2.6	11 (8.5 %) ↑ 3.5	2 (2.4 %) ↓ 6.1
Joint click (group II disc displacement)	18 (10.8 %)	11 (6.9 %) ↓ 3.8	9 (7.0 %) ↑ 0.1	5 (5.9 %) ↓ 1.1
TM joint pain on palpation (group III arthralgia)	1 (0.6 %)	0 ↓ 0.6	0 ↓	0
Group I+II	0	0	2 (1.6 %) ↑ 1.6	0 ↓ 1.6
Group I+III	8 (4.8 %)	1 (0.6 %) ↓ 3.2	2 (1.6 %) ↑ 1.0	0 ↓ 1.6
Group II+III	1 (0.5 %)	1 (0.6 %) ↑ 0.1	0 ↓ 0.6	0
Group I+II+III	1 (0.6 %)	2 (1.3 %) ↑ 0.7	1 (0.8 %) ↓ 0.5	0 ↓ 0.8
TMD	33 (19.8 %)	23 (14.5 %) ↓ 5.3	25 (19.4 %) ↑ 4.9	7 (8.2 %) ↓ 11.2
No TMD	132 (79.0 %)	130 (81.8 %) ↓ 2.8	104 (80.6 %) ↓ 1.2	78 (91.8 %) ↑ 11.2
Missing (dropout or did not answer)	2	8	0	0
Pain-related TMD	15 (8.9 %)	12 (7.5 %) ↓ 1.4	16 (12.4 %) ↑ 4.9	2 (2.4 %) ↓ 10

Arrows indicate % of increase (↑) or decrease (↓) when comparing to previous visit

**Fig. 1** Incidence and prevalence of TMD signs at baseline through visit IV. Dropouts are shown with the vertical arrows exiting the figure. Number of responses for individual questions may vary due to missing data



patients. The prevalence of POB increased in the next two visits, with 9.4 % on visit III and 17.9 % on visit IV (Fig. 4). Of the 14 patients with POB at visit IV, ten presented with bilateral POB. Only four of 14 (28.6 %) patients with POB were aware of any bite changes and reported them in the questionnaire. These four patients indicated that the severity of the change was mild, with  $x=2.0$  (1–10 scale), and that the longevity of the bite change was transient, with  $x=1.7$  (on five-point checklist).

**Discussion**

Although several studies have reported on the side effects of MADs for the treatment of OSA [25–31], few have explored the role that MADs play in the development or aggravation of TMD. Furthermore, few studies in this area have used the RDC/TMD to evaluate TMD in a standardized manner [29, 45]. To our knowledge, this is the only study to evaluate and report on the incidence of TMD in patients treated with a MAD.

This study suggests that the use of a MAD may cause the development of TMD in a small number of patients who were pain-free before treatment; however, the incidence was greater at the beginning of treatment and decreased on each successive visit. The prevalence of TMD increased at the beginning of treatment especially for pTMD. This later decreased to values lower than at baseline. Finally, the patients who presented TMD at baseline did not suffer an increase of their signs or symptoms during MAD treatment. Some of these patients actually demonstrated no TMD signs or symptoms as the treatment progressed.

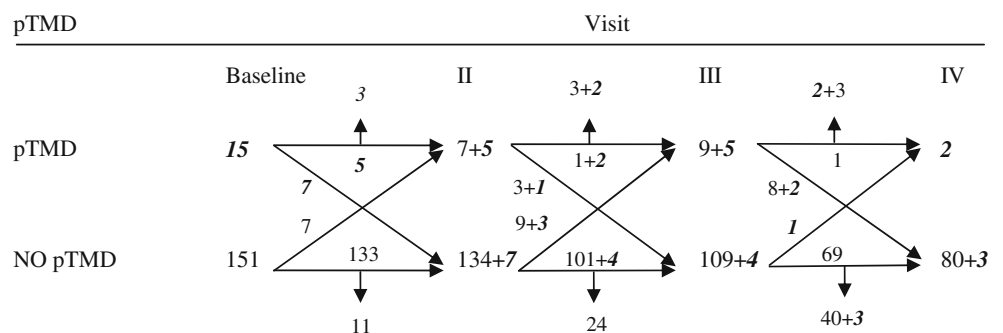
Two studies that used the RDC/TMD to evaluate patients with long-term use of a MAD [29, 45] were found. Doff et

al. [45] demonstrated an increase of TMD, especially pTMD, at 2–3 months of wearing the MAD. By the end of his study period, this variable had decreased substantially. As in the study of Doff et al., the present study further analyzed specifically pTMD, which considers pain on palpation of the masticatory musculature and TM joints, excluding joint noises. We also found a considerable increase of pTMD during the initial treatment period with MAD use and a subsequent decrease of this variable during the last visit.

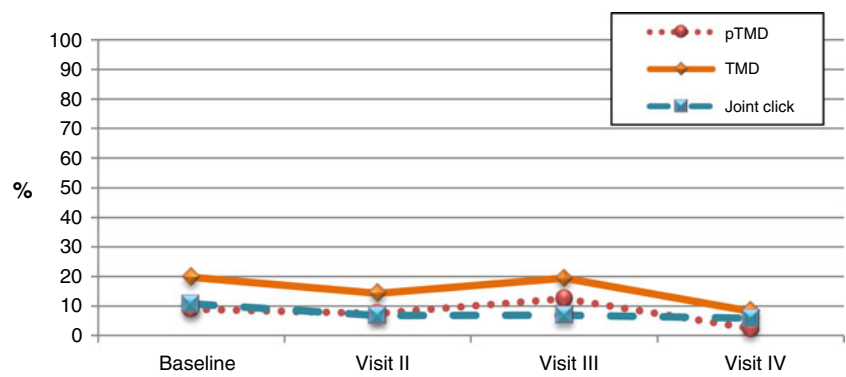
Martinez-Gomis and coworkers [29] studied 40 patients also using the RDC/TMD. They determined no significant differences in the prevalence of any TMD diagnosis at any of the three checkups lasting up to 58 months. In contrast to the finding of Martinez-Gomis, the present study demonstrated an initial increase in the prevalence of TMD with a subsequent decrease in this variable at the end of the study.

Notwithstanding the fact that, overall, the prevalence of TMD decreased, in a few cases TMD developed over the course of treatment with a MAD. The development of TMD as well as the increase in its occurrence during the initial phases of treatment is likely due to the non-habitual protruded position in which the mandible is placed during the hours of sleep. Muscles and ligaments are made to function within certain limits and for certain periods of time. If any of these factors are exceeded, these structures are likely to respond to this insult with pain. The incidence as well as the prevalence of TMD continuously decreased as treatment progressed. This could possibly reflect the adaptive capacities of the population studied. The present results could therefore indicate that MADs are likely to cause pain-related signs and symptoms of TMD to a small number of patients. Nevertheless, these are most probably transient.

**Fig. 2** Incidence and prevalence of pain-related TMD signs at baseline through visit IV. Dropouts are shown with the vertical arrows exiting the figure. Number of responses for individual questions may vary due to missing data. *Italic numbers* represent the initial pain patients as they fluctuate throughout visits

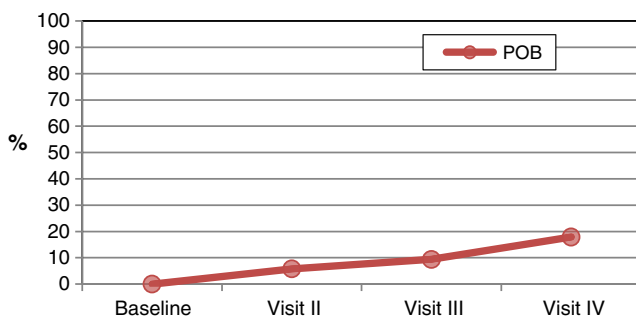


**Fig. 3** Variation of occurrence of TMD, pain-related TMD, and joint clicks on each visit



Two studies were found that suggest that the presence of TMD is a contraindication for MAD treatment and excluded patients with preexisting TMD from their studies [27, 28]. The present study did not exclude the patients diagnosed with TMD at the beginning of treatment. These patients were followed throughout the visits, finding only two patients who had persistent pain on palpation throughout the study. It is important to mention that these patients did not drop out of the study in spite of this diagnosis. It is interesting to note that some of these patients moved to the “no pTMD” group as treatment progressed, and three patients initially diagnosed with pTMD finished the study in the “no pTMD” group. Thus, the present study suggests that TMD is not necessarily a contraindication for the use of a MAD as a form of treatment for OSA. This is in accordance with longitudinal studies on MAD as a treatment for OSA which found that little to no patients dropped out of the study because of TMD pain [29, 45, 46]. As Doff and co-workers [45] have suggested, MADs may actually have a therapeutic effect on the masticatory system. Al-Ani et al. [47], speculated that the therapeutic effect of MADs may be much like an occlusal appliance in reducing the deleterious effects of nocturnal parafunctional activity.

Joint clicks are considered part of the TMD diagnosis by the RDC. The present study demonstrated that the percentage of joint clicks decreased as MAD therapy progressed. Other authors have found similar results [27, 30]. This decrease may be due to an adaptive response to the protrusive jaw position dictated by the use of the MAD. A



**Fig. 4** Incidence and prevalence of POB

reduction in joint clicks as well as pain has also been observed as a result of anterior repositioning appliance therapy [48]. This accepted therapy for painful anterior disc displacement with reduction functions by transiently protruding the jaw away from the inflamed retrodiscal tissues. This therapy sometimes results in a recapture of the TM joint disc with the subsequent elimination of the joint click [49]. The position in which the mandible is placed in the anterior repositioning appliance is similar to how the MAD positions the mandible; therefore, a reduction in joint clicks may be observed.

Finally, POB developed with the use of the MAD. Although the initial incidence was only 5.4 %, this number increased on every visit thereafter. In spite of presenting POB, only a small number of these patients reported feeling changes in their bite. The reason postulated for the development of POB is a transient shortening or contracture of the inferior lateral pterygoid muscle. When this muscle is not stimulated to return to its normal length, it may develop a myofibrotic contracture [50], decreasing its ability to return to its habitual length. Other authors have suggested that bony adaptations in the functional surface of the condyle or temporal bone may occur when loaded in a forward and downward position for long periods of time. However, the scientific evidence does not seem to support this theory [51]. The present results are in accordance with studies reporting on occlusion and bite changes when using MADs [36, 40, 41]. Ueda et al. [22] found greater changes studying 45 patients using cast study models and an occlusal diagnostic system. He determined that the occlusal contact area changed in 39/45 patients, decreasing in 66.7 % of them. This decrease of contact area was mostly in the molar and premolar regions. Pantin et al. [36] found that 14 % of his study population presented with clinical POB; in his population, none of the 15 patients were aware of this change. Because of the number of hours the MAD is used, a transient bite change each morning upon its removal is expected. The present study asked the patients who reported awareness of bite changes how long these lasted. More than half indicated that they were transient, lasting less than 12 h. Martinez-Gomis [29] stated that the tendency of reduction in occlusal contacts reversed after 2 to 5 years of treatment.

Because of the shorter length of the present study, this could not be confirmed. Exercises appear to be a helpful approach to avoid or correct this condition [42]. Although the present study population was taught jaw exercises, they still developed POB. This may be due to the lack of close follow-up to assure compliance. Patients should be advised that POB is one of the most prevalent side effects of this therapy. They should be taught to check that their occlusion returns to normal each morning. Health professionals treating patients with MADs should monitor continuously for its appearance. If POB is found, exercises are important to try to re-lengthen the muscles before it advances to a persistent state. It may also be necessary for the patient to stop wearing the appliance for short periods of time to allow for a return to normal occlusion.

The nature of this study was observational. It was complicated by a significant number of dropouts, limiting the strength of the conclusions. Nevertheless, the number of dropouts in the present study is comparable to other studies which have found that between 18 and 45 % [46, 52] of the population treated with MAD therapy discontinue treatment. McGown and coworkers [52] studied the factors affecting the continued usage of MADs in 166 patients after more than 1 year of treatment. Similarly to the present study, McGown and coworkers discovered that only 55 % of the study population continued using the MAD after more than 1 year of treatment. The patients' lack of compliance was mainly associated to discomfort and to ineffectiveness of the MAD. The mentioned discomfort included symptoms such as altered bite, excessive salivation, sleep disturbance, and temporomandibular joint pain. The cited study determined that discomfort associated to TM joint pain was present in only about half of the non-user population. It is worthy to note that 40 % of the population that continued using the MAD also reported TM joint discomfort. This leads us to believe that there may be a certain percentage of the population who could possibly be more sensitive to the perception of TM joint pain, and this may be enough to stop wearing the MAD while others, in spite of feeling TM joint pain, continue wearing the MAD. McGown's odds ratio analysis found the strongest association between lack of improvement according to the bed partner and discontinuing MAD use.

Although the studies found indicate that the number of dropouts are similar to other studies and that little to no patients have dropped out because of perceived TM joint pain, the present study performed additional analyses to minimize the possible effect of dropouts. The baseline data of the dropout patients were compared to the data of the patients who returned on every visit. The analysis demonstrated that these two groups were demographically and clinically similar at baseline. Demographically, the only differences were that the dropout patients were younger and had higher BMI scores. Marklund et al. [46] and McGown et al. [52] did not find that age was a predictor

for lack of tolerability and, therefore, discontinuation of use of the MAD. Marklund et al. found that MAD therapy was less effective in patients whose BMI increased during therapy. In our study, the dropout population with higher BMIs could have found the MAD therapy less effective as the airway obstruction may have been greater to overcome in patients with higher BMIs. Clinically, the dropouts reported more often pTMD at baseline, although this could not be verified on clinical examination. Thus, the patients who dropped out may have been more sensitive to changes in their TM joints and related structures to begin with, and the MAD may have led to TMD, causing the patients to drop out. On the other hand, if they were more sensitive in general they may have not been able to tolerate the MAD, causing them to drop out as well.

Further statistical analysis of the development and persistence of signs and symptoms of TMD in the dropout population confirmed that the early dropout population differed from the rest of the population presenting higher reports of muscle as well as jaw joint pain. This information supports the above-mentioned speculation. The dropout population reported more symptoms which may have been the reason for their lack of follow-up. However, it is important to mention that these same patients did not present differences compared to the rest of the population in their signs upon examination.

Even though many speculations have been made on the reasons for the dropout population not to return for treatment, this could be considered a limitation of this study as it would have been ideal to know exactly why these patients did not return.

Furthermore to better understand the prevalence/incidence of MAD-associated TMD, more prospective studies are needed.

Among other limitations, follow-up polysomnography data to confirm the end-point of titration were not available for most patients. Associated cost and lack of referring physician request prohibited this procedure. This information would have been ideal to objectively assess the adequate end-point of titration and overall success of MAD treatment. This is most important considering that some authors have found residual OSA in patients whose symptoms had reportedly decreased, representing a placebo effect of the MAD [13, 60].

Compliance with treatment was assessed through self-report. This may also be considered a limitation of this study as recall bias is a possibility. A potential solution for this problem would be to use a home monitoring device. These devices have been studied and are considered a feasible and accurate way of evaluating the success of OSA treatment [53–56].

A hard Herbst appliance was used in more than 80 % of the patients in this study. Studies comparing the effectiveness of different types of oral appliances to treat OSA indicate significant differences between fixed and adjustable appliances [57, 58]. These studies demonstrated that an adjustable

appliance is more effective in reducing patient symptoms as well as AHI. Nevertheless, studies comparing different types of adjustable appliances are few. Recently, Ahrens, through a systematic review on the efficacy of different oral appliances, found that all mandibular advancement appliances, independent of their design, decreased objective measures for OSA. This author suggested that the efficacy of an oral appliance depends more on other important factors, such as the severity of OSA, material used to fabricate the MAD, and the degree of protrusion [59]. This information leads us to believe that the results found in this study using mostly the Herbst appliance may be generalizable to other MAD devices.

## Conclusions

- The use of MADs may lead to the development of TMD in a small number of patients; nevertheless, these signs are most likely transient.
- Patients with pre-existing signs and symptoms of TMD do not experience significant exacerbation of those signs and symptoms during MAD use. These signs and symptoms may actually decrease over time.
- The presence of signs and symptoms of TMD is not necessarily a contraindication for treatment with a MAD for OSA.
- Persistent POB was found in 17.9 % of patients but only one out of every four of these patients was aware of any bite changes. Thus, it is important for the clinician to increase awareness of this side effect so that measures can be installed to counteract it.

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## References

1. Yaggi HK, Strohl KP (2010) Adult obstructive sleep apnea/hypopnea syndrome: definitions, risk factors, and pathogenesis. *Clin Chest Med* 31(2):179–186
2. Shahar E, Whitney CW, Redline S, Lee ET, Newman AB, Javier Nieto F, O'Connor GT, Boland LL, Schwartz JE, Samet JM (2001) Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. *Am J Respir Crit Care Med* 163(1):19–25
3. Eskafi M, Ekberg E, Cline C, Israelsson B, Nilner M (2004) Use of a mandibular advancement device in patients with congestive heart failure and sleep apnoea. *Gerodontology* 21(2):100–107
4. Sjoström C, Lindberg E, Elmasry A, Hagg A, Svardsudd K, Janson C (2002) Prevalence of sleep apnoea and snoring in hypertensive men: a population based study. *Thorax* 57(7):602–607
5. Resnick HE, Redline S, Shahar E, Gilpin A, Newman A, Walter R, Ewy GA, Howard BV, Punjabi NM (2003) Diabetes and sleep disturbances: findings from the Sleep Heart Health Study. *Diabetes Care* 26(3):702–709
6. Kushida CA, Littner MR, Hirshkowitz M, Morgenthaler TI, Alessi CA, Bailey D, Boehlecke B, Brown TM, Coleman J Jr, Friedman L, Kapen S, Kapur VK, Kramer M, Lee-Chiong T, Owens J, Pancer JP, Swick TJ, Wise MS (2006) Practice parameters for the use of continuous and bilevel positive airway pressure devices to treat adult patients with sleep-related breathing disorders. *Sleep* 29(3):375–380
7. Engleman HM, Wild MR (2003) Improving CPAP use by patients with the sleep apnoea/hypopnoea syndrome (SAHS). *Sleep Med Rev* 7(1):81–99
8. Rogers RR (2000) Oral appliance therapy for the management of sleep disordered breathing: an overview. *Sleep Breath* 4(2):79–84
9. Kushida CA, Morgenthaler TI, Littner MR, Alessi CA, Bailey D, Coleman J Jr, Friedman L, Hirshkowitz M, Kapen S, Kramer M, Lee-Chiong T, Owens J, Pancer JP (2006) Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: an update for 2005. *Sleep* 29(2):240–243
10. Tan YK, L'Estrange PR, Luo YM, Smith C, Grant HR, Simonds AK, Spiro SG, Battagel JM (2002) Mandibular advancement splints and continuous positive airway pressure in patients with obstructive sleep apnoea: a randomized cross-over trial. *Eur J Orthod* 24(3):239–249
11. Walker-Engstrom ML, Ringqvist I, Vestling O, Wilhelmsson B, Tegelberg A (2003) A prospective randomized study comparing two different degrees of mandibular advancement with a dental appliance in treatment of severe obstructive sleep apnea. *Sleep Breath* 7(3):119–130
12. Marklund M, Sahlin C, Stenlund H, Persson M, Franklin KA (2001) Mandibular advancement device in patients with obstructive sleep apnea: long-term effects on apnea and sleep. *Chest* 120(1):162–169
13. Gotsopoulos H, Chen C, Qian J, Cistulli PA (2002) Oral appliance therapy improves symptoms in obstructive sleep apnea: a randomized, controlled trial. *Am J Respir Crit Care Med* 166(5):743–748
14. Blanco J, Zamarron C, Abeleira Pazos MT, Lamela C, Suarez Quintanilla D (2005) Prospective evaluation of an oral appliance in the treatment of obstructive sleep apnea syndrome. *Sleep Breath* 9(1):20–25
15. Ferguson KA, Cartwright R, Rogers R, Schmidt-Nowara W (2006) Oral appliances for snoring and obstructive sleep apnea: a review. *Sleep* 29(2):244–262
16. Yoshida K (2006) Effect on blood pressure of oral appliance therapy for sleep apnea syndrome. *Int J Prosthodont* 19(1):61–66
17. Otsuka R, Ribeiro de Almeida F, Lowe AA, Linden W, Ryan F (2006) The effect of oral appliance therapy on blood pressure in patients with obstructive sleep apnea. *Sleep Breath* 10(1):29–36
18. Gotsopoulos H, Kelly JJ, Cistulli PA (2004) Oral appliance therapy reduces blood pressure in obstructive sleep apnea: a randomized, controlled trial. *Sleep* 27(5):934–941
19. Andren A, Sjoquist M, Tegelberg A (2009) Effects on blood pressure after treatment of obstructive sleep apnoea with a mandibular advancement appliance—a three-year follow-up. *J Oral Rehabil* 36(10):719–725
20. Jonsson T, Arnlaugsson S, Saemundsson SR, Magnusson TE (2009) Development of occlusal traits and dental arch space from adolescence to adulthood: a 25-year follow-up study of 245 untreated subjects. *Am J Orthod Dentofacial Orthop* 135(4):456–462
21. Almeida FR, Lowe AA, Sung JO, Tsuiji S, Otsuka R (2006) Long-term sequelae of oral appliance therapy in obstructive sleep apnea patients: part 1. Cephalometric analysis. *Am J Orthod Dentofacial Orthop* 129(2):195–204
22. Ueda H, Almeida FR, Lowe AA, Ruse ND (2008) Changes in occlusal contact area during oral appliance therapy assessed on study models. *Angle Orthod* 78(5):866–872
23. Dube C, Rompre PH, Manzini C, Guitard F, de Grandmont P, Lavigne GJ (2004) Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. *J Dent Res* 83(5):398–403

24. De Leeuw R, American Academy of Orofacial P (2008) Orofacial pain: guidelines for assessment, diagnosis, and management. Quintessence, Chicago
25. Cunali PA, Almeida FR, Santos CD, Valdrighi NY, Nascimento LS, Dal'Fabbro C, Tufik S, Bittencourt LR (2009) Prevalence of temporomandibular disorders in obstructive sleep apnea patients referred for oral appliance therapy. *J Orofac Pain* 23(4):339–344
26. Doff MH, Hoekema A, Pruim GJ, Huddleston Slater JJ, Stegenga B (2010) Long-term oral-appliance therapy in obstructive sleep apnea: a cephalometric study of craniofacial changes. *J Dent* 38(12):1010–1018
27. Giannasi LC, Almeida FR, Magini M, Costa MS, de Oliveira CS, de Oliveira JC, Kalil Bussadori S, de Oliveira LV (2009) Systematic assessment of the impact of oral appliance therapy on the temporomandibular joint during treatment of obstructive sleep apnea: long-term evaluation. *Sleep Breath* 13(4):375–381
28. Bondemark L, Lindman R (2000) Craniomandibular status and function in patients with habitual snoring and obstructive sleep apnoea after nocturnal treatment with a mandibular advancement splint: a 2-year follow-up. *Eur J Orthod* 22(1):53–60
29. Martinez-Gomis J, Willaert E, Noguez L, Pascual M, Somoza M, Monasterio C (2010) Five years of sleep apnea treatment with a mandibular advancement device. Side effects and technical complications. *Angle Orthod* 80(1):30–36
30. Fransson AM, Tegelberg A, Johansson A, Wenneberg B (2004) Influence on the masticatory system in treatment of obstructive sleep apnea and snoring with a mandibular protruding device: a 2-year follow-up. *Am J Orthod Dentofacial Orthop* 126(6):687–693
31. de Almeida FR, Bittencourt LR, de Almeida CI, Tsuiki S, Lowe AA, Tufik S (2002) Effects of mandibular posture on obstructive sleep apnea severity and the temporomandibular joint in patients fitted with an oral appliance. *Sleep* 25(5):507–513
32. Chen H, Lowe AA, de Almeida FR, Fleetham JA, Wang B (2008) Three-dimensional computer-assisted study model analysis of long-term oral-appliance wear. Part 2. Side effects of oral appliances in obstructive sleep apnea patients. *Am J Orthod Dentofacial Orthop* 134(3):408–417
33. Fritsch KM, Iseli A, Russi EW, Bloch KE (2001) Side effects of mandibular advancement devices for sleep apnea treatment. *Am J Respir Crit Care Med* 164(5):813–818
34. Ghazal A, Jonas IE, Rose EC (2008) Dental side effects of mandibular advancement appliances—a 2-year follow-up. *J Orofac Orthop* 69(6):437–447
35. Marklund M, Franklin KA, Persson M (2001) Orthodontic side-effects of mandibular advancement devices during treatment of snoring and sleep apnoea. *Eur J Orthod* 23(2):135–144
36. Pantin CC, Hillman DR, Tennant M (1999) Dental side effects of an oral device to treat snoring and obstructive sleep apnea. *Sleep* 22(2):237–240
37. Robertson C, Herbison P, Harkness M (2003) Dental and occlusal changes during mandibular advancement splint therapy in sleep disordered patients. *Eur J Orthod* 25(4):371–376
38. Rose EC, Staats R, Virchow C Jr, Jonas IE (2002) Occlusal and skeletal effects of an oral appliance in the treatment of obstructive sleep apnea. *Chest* 122(3):871–877
39. Almeida FR, Lowe AA, Otsuka R, Fastlicht S, Farbood M, Tsuiki S (2006) Long-term sequelae of oral appliance therapy in obstructive sleep apnea patients: part 2. Study-model analysis. *Am J Orthod Dentofacial Orthop* 129(2):205–213
40. Monteith BD (2004) Altered jaw posture and occlusal disruption patterns following mandibular advancement therapy for sleep apnea: a preliminary study of cephalometric predictors. *Int J Prosthodont* 17(3):274–280
41. Otsuka R, Almeida FR, Lowe AA (2007) The effects of oral appliance therapy on occlusal function in patients with obstructive sleep apnea: a short-term prospective study. *Am J Orthod Dentofacial Orthop* 131(2):176–183
42. Ueda H, Almeida FR, Chen H, Lowe AA (2009) Effect of 2 jaw exercises on occlusal function in patients with obstructive sleep apnea during oral appliance therapy: a randomized controlled trial. *Am J Orthod Dentofacial Orthop* 135(4):430 e431–437; discussion 430–431
43. George PT (1992) A new instrument for functional appliance bite registration. *J Clin Orthod* 26(11):721–723
44. Dworkin SF, LeResche L (1992) Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord* 6(4):301–355
45. Doff MH, Veldhuis SK, Hoekema A, Slater JJ, Wijkstra PJ, de Bont LG, Stegenga B (2011) Long-term oral appliance therapy in obstructive sleep apnea syndrome: a controlled study on temporomandibular side effects. *Clin Oral Investig*. doi:10.1007/s00784-011-0555-6
46. Marklund M, Stenlund H, Franklin KA (2004) Mandibular advancement devices in 630 men and women with obstructive sleep apnea and snoring: tolerability and predictors of treatment success. *Chest* 125(4):1270–1278
47. Al-Ani MZ, Davies SJ, Gray RJ, Sloan P, Glenney AM (2004) Stabilisation splint therapy for temporomandibular pain dysfunction syndrome. *Cochrane Database Syst Rev* (1):CD002778
48. Garcia AR, Folli S, Zuim PR, de Sousa V (2008) Mandible protrusion and decrease of TMJ sounds: an electrovibratographic examination. *Braz Dent J* 19(1):77–82
49. Kurita H, Ohtsuka A, Kurashina K, Kopp S (2001) A study of factors for successful splint capture of anteriorly displaced temporomandibular joint disc with disc repositioning appliance. *J Oral Rehabil* 28(7):651–657
50. Okeson JP (2008) Management of temporomandibular disorders and occlusion. Mosby, St. Louis
51. Bondemark L (1999) Does 2 years' nocturnal treatment with a mandibular advancement splint in adult patients with snoring and OSAS cause a change in the posture of the mandible? *Am J Orthod Dentofacial Orthop* 116(6):621–628
52. McGown AD, Makker HK, Battagel JM, L'Estrange PR, Grant HR, Spiro SG (2001) Long-term use of mandibular advancement splints for snoring and obstructive sleep apnoea: a questionnaire survey. *Eur Respir J* 17(3):462–466
53. Levendowski DJ, Morgan TD, Patrickus JE, Westbrook PR, Berka C, Zavora T, Popovic D (2007) In-home evaluation of efficacy and titration of a mandibular advancement device for obstructive sleep apnea. *Sleep Breath* 11(3):139–147
54. Westbrook PR, Levendowski DJ, Cvetinovic M, Zavora T, Velimirovic V, Henninger D, Nicholson D (2005) Description and validation of the apnea risk evaluation system: a novel method to diagnose sleep apnea-hypopnea in the home. *Chest* 128(4):2166–2175
55. Ayappa I, Norman RG, Seelall V, Rapoport DM (2008) Validation of a self-applied unattended monitor for sleep disordered breathing. *J Clin Sleep Med* 4(1):26–37
56. Chen H, Lowe AA, Bai Y, Hamilton P, Fleetham JA, Almeida FR (2009) Evaluation of a portable recording device (ApneaLink) for case selection of obstructive sleep apnea. *Sleep Breath* 13(3):213–219
57. Lettieri CJ, Paolino N, Eliasson AH, Shah AA, Holley AB (2011) Comparison of adjustable and fixed oral appliances for the treatment of obstructive sleep apnea. *J Clin Sleep Med* 7(5):439–445
58. Sari E, Menillo S (2011) Comparison of titratable oral appliance and mandibular advancement splint in the treatment of patients with obstructive sleep apnea. doi:10.5402/2011/581692
59. Ahrens A, McGrath C, Hagg U (2011) A systematic review of the efficacy of oral appliance design in the management of obstructive sleep apnoea. *Eur J Orthod*. 33(3):318–324
60. Mehta A, Qian J, Petocz P, Darendeliler MA, Cistulli PA (2001) A randomized, controlled study of a mandibular advancement splint for obstructive sleep apnea. *Am J Respir Crit Care Med* 163(6):1457–1461