Oral Appliances in the Management of Obstructive Sleep Apnea

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KEYWORDS

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KEY POINTS

- The concept in oral appliances for obstructive sleep apnea (OSA) management is protrusion of the mandible and/or tongue for structural effects on the upper airway.
- The upper airway is a muscular tube and its dimensions are enlarged with mandibular and tongue advancement.
- Protrusion of the mandible and tongue stretches the muscles, thereby reducing upper airway collapsibility with airway shape change and increase in muscle tone.
- Oral appliances are effective and evidence-based options in managing OSA.

TYPES OF ORAL APPLIANCES

The primary oral appliance (OA) used in obstructive sleep apnea (OSA) treatment is the mandibular advancement device (MAD). MADs may be either an over-the-counter stock device or customized for individual patients. MADs come in various designs and materials, but most comprise upper and lower splints mounted over the dentition as either a 1-piece monoblock (**Fig. 1**) or a 2-piece biblock (**Fig. 2**). Connectors or blocks relate the upper and lower splints in a biblock to protrude the mandible in a forward position during sleep.¹

Tongue-retaining devices, or tongue-stabilizing devices (TSDs) (**Fig. 3**), are a second type of OA, which displace the tongue anteriorly and may be customized or come in different stock sizes. TSDs use negative pressure and salivary adhesion to hold onto the tongue and anterior lip shields to elongate and reposition the tongue in a more forward position independent of the mandible during

sleep, thereby opening the oropharyngeal airway.^{1,2}

TSDs have similar efficacy as MADs but poorer compliance. More than 90% of patients preferred MADs over TSDs for OA therapy.³ The evidence base is stronger for MADs and considerably lower for TSDs.^{2,4,5}

ORAL APPLIANCE EFFECTS ON AIRWAY *Cross-Sectional Area*

Airway imaging with cone-beam computed tomography, magnetic resonance imaging and nasal endoscopy showed anteroposterior (AP) mandibular protrusion predominantly increases the caliber of the airway at the retropalatal area via lateral expansion and displacement of parapharyngeal fat pads^{2,6–13} while the tongue and tongue-base muscles shift forward.^{2,9,13}

The lateral widening from AP movement is attributed to stretching of soft tissue connections

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Fig. 1. Monoblock of 1-piece MAD. (Courtesy of the Orthodontic Laboratory in the National Dental Centre of Singapore, Singapore.)

between the tongue, soft palate, and lateral pharyngeal walls.^{11,14,15} Dynamic MRI suggests a direct connection between lateral pharyngeal walls and the ramus, postulated to be the pterygo-mandibular raphe.¹³

TSDs increase airway AP diameter to a greater degree than MADs, and traction on intrapharyngeal connections through the tongue base additionally increases the lateral dimension of the airway. Compared with MADs, TSDs produce greater increases in retropalatal and retroglossal cross-sectional area (CSA). This is attributed to greater anterior tongue movement with TSDs.²

Other Effects

Lateral expansion with both MADs and TSDs promotes an elliptical cross-sectional shape with a

transverse long axis.² A small but significant decrease in upper airway length has been found with MAD use,² which may counteract the airway length increase from lying supine demonstrated in OSA patients.¹⁶

Electromyography shows that MADs increase activation of masseter, lateral pterygoid, genio-glossus, and geniohyoid muscles. It is postulated that on top of the purely structural anatomic effects of MADs, increased neuromuscular activation contributes to upper airway patency.^{17–19}

Collapsibility

Morphologic increase in CSA, change in cross-sectional shape, decrease in airway length, and neuromuscular activation with MAD may contribute to reduced collapsibility.^{2,12,20} Empirically, a significant reduction in upper airway collapsibility in stage 2 and stage 3 sleep was observed with MAD use. Improvement in collapsibility was significantly greater in complete responders than in partial responders or nonresponders.²¹ Airway collapsibility may have a dose-dependent relationship with mandibular advancement.¹⁰

ORAL APPLIANCE EFFECTS ON OBSTRUCTIVE SLEEP APNEA COMPARED WITH CONTINUOUS POSITIVE AIRWAY PRESSURE

Continuous positive airway pressure (CPAP) and MADs are chronic, noninvasive, symptomatic treatments for OSA that do not treat the underlying

Fig. 2. Biblock or 2-piece MADs with different connectors. (Courtesy of [upper left] SomnoMed, Sydney, Australia; and [upper right, lower left, and lower right] Orthodontic Master, Singapore.)

Fig. 3. TSD. (Courtesy of Innovative Health Technologies Ltd, Dunedin, New Zealand.)

anatomic basis of the condition. Pneumatic splinting of the airway with CPAP is acknowledged as the gold standard for treatment of OSA, although MAD has been progressively recommended with each iteration of the American Academy of Sleep Medicine (AASM) guidelines.^{4,22,23} The latest AASM guidelines do not specify a particular disease severity for MAD use due to lack of evidence relating MAD efficacy to disease severity.⁴

Apnea-Hypopnea Index

MADs are efficacious in reducing apnea-hypopnea index (AHI) although there is individual variability.²⁴ A nonlinear dose-dependent relationship with degree of advancement is reported.^{25–30}

The efficacy of CPAP over MAD in reducing AHI is well established and has been consistently reported by meta-analyses comparing the 2,^{4,31–34} even with increasing numbers of primary studies. According to the AASM/American Academy of Dental Sleep Medicine (AADSM) task force, CPAP is significantly better than MAD at reducing AHI scores (difference of 6.24 events per hour).⁴ A recent meta-analysis of 14 randomized controlled trials reported that CPAP reduced AHI by an additional 8.43 events per hour over MAD.³⁴

Oxygenation

MADs are efficacious in improving minimum arterial oxygen saturation (SaO_2), and the improvement has been reported to have a nonlinear dose-dependent relationship with degree of advancement.¹⁰

Similar to AHI, the finding of better improvement in SaO_2 with CPAP over MAD has been consistently reported.^{4,31,32,34} The AASM/AADSM task force reported that CPAP is slightly better at improving oxygenation (SaO_2 difference of 3.11%) compared with MADs.⁴ More recently, a significant effect on SaO_2 in favor of CPAP was reported.³⁴

Compliance

Comparing subjective self-reported MAD compliance with objective recorded CPAP compliance, MAD compliance is better. The AASM/AADSM task force reported that MADs were used 0.70 more hours per night than CPAP.⁴ Objective measurements of OA usage after 3 months of OA treatment ranged from a mean of 6.4 hours to 6.6 hours per night.^{35,36} A meta-analysis of 6 studies encompassing both subjective and objective MAD compliance measures reported an additional 1.1 hours per night use of MAD over CPAP.³³

A majority of crossover trials report that MADs are preferred to CPAP, which may imply better patient compliance with MADs.^{4,37,38} Depending on OSA severity and total sleep time, less than ideal compliance with CPAP can substantially decrease its effectiveness. Better MAD compliance over CPAP could be why health outcomes of the 2 treatment modalities are similar despite better CPAP efficacy at reducing AHI and increasing oxygenation.³⁷

Subjective Sleepiness

MADs improve subjective daytime sleepiness measured by Epworth Sleepiness Scale (ESS) scores significantly.³⁹

Despite better efficacy with CPAP than MADs in reducing AHI and improving SaO_2 , differences in ESS scores are more ambiguous. A network meta-analysis of 67 studies found that CPAP and MADs are both effective in reducing excessive daytime sleepiness as assessed by the ESS, but CPAP is likely to be more effective than MADs, with a greater reduction in ESS by an average of 0.8 points.⁴⁰ Other reviews have reported either similar outcomes^{4,32} or slightly better outcomes with CPAP.^{33,34}

Function and Quality of Life

Aside from long-term health effects, untreated OSA also affects health-related quality of life, with hypersomnolence impacting ability to function.⁴¹ Function and quality of life are commonly measured using both the sleep-specific Functional Outcomes of Sleep Questionnaire (FOSQ) and generic Medical Outcomes Study 36-Item Short Form Health Survey (SF-36). MADs are associated with improvements in FOSQ subscale and total scores as well as SF-36 scores.^{4,42,43}

Minimal difference was found between CPAP and MAD with respect to improvements in daytime functional outcomes measured by SF-36 and FOSQ scores.⁴ This finding was corroborated in a meta-analysis,⁴⁴ with direct comparisons as well

as network meta-analysis showing no significant difference in treatment outcomes between CPAP and MAD in the mental and physical components of SF-36. Other meta-analyses have also reported no difference in FOSQ or SF-36 scores.^{31–34}

Cardiovascular Effects

Meta-analyses by multiple groups have found MADs to be the equivalent of CPAP at reducing blood pressure in adults with OSA.^{4,34,45–47} A network meta-analysis reported that CPAP was associated with a reduction in systolic blood pressure of 2.5 mm Hg and diastolic blood pressure reduction of 2.0 mm Hg. MADs were associated with reduction in systolic blood pressure of 2.1 mm Hg and diastolic blood pressure of 1.9 mm Hg. There was no significant difference between CPAP and MADs.⁴⁰

Studies on other cardiovascular markers (heart rate variability, circulating cardiovascular biomarkers, endothelial function, and arterial stiffness) were deemed heterogeneous and inconclusive.⁴⁶

Mortality

A prospective cohort study on long-term cardiovascular mortality in 208 subjects found CPAP and MAD equally effective in reducing the risk of fatal cardiovascular events in patients with severe OSA, despite higher residual AHI for MAD compared with CPAP users (16.3 vs 4.5 events per hour). There was no difference in the cumulative cardiovascular mortality between OSA patients treated with CPAP or MAD and that of the nonapneic controls, suggesting effective symptomatic treatment with either modality despite differences in residual AHI.⁴⁸ This outcome, however, is from a single observational study. More longitudinal studies with better methodology are needed to answer key questions on cardiovascular morbidity and mortality outcomes.^{46,48}

ORAL APPLIANCE ADVERSE EFFECTS

Similar to efficacy, adverse side effects have a dose-dependent relationship with protrusion.²⁹ A balance must be struck between efficacy and side effects because more adverse effects reduce long-term compliance,^{35,49} resulting in patients terminating MAD therapy.^{50,51} Most adverse effects caused by MADs are mild and transient, occur during the initial phase of therapy, and tend to resolve with time.^{51,52}

Dental Effects

Long-term use of OAs leads to occlusal changes. A meta-analysis reported a significant increase in

lower incisor inclination by 2.07°, resulting in 0.99-mm decrease in overjet (OJ) and 1.0-mm decrease in overbite (OB). A greater decrease in OJ and OB was associated with longer treatment. There was no significant change in the upper incisor inclination or interincisal angle.⁵³

Due to the lower incisor inclination change and attendant decrease in OJ and OB, there was significant increase in anterior crossbites,⁵⁴ increase in mandibular arch width, and reduction in lower arch crowding.^{54,55} Maxillary arch width increase and upper arch decrowding also have been reported.⁵⁵ A decrease in posterior occlusal contacts^{54,56–58} may result in a transient difficulty with chewing.⁵¹ Although the magnitude of dental changes are small, they may be significant to patient perception of developing dental malocclusion.⁵⁹

Craniofacial Changes

A meta-analysis of skeletal changes from MAD use reported no significant skeletal changes.⁵³ Other investigators, however, report significant increase in lower and total anterior facial height from long-term MAD use of more than 2 years.^{60,61}

Temporomandibular Joint Disorders

Transient muscle soreness and temporomandibular joint (TMJ) discomfort have been reported after MAD use, especially during the initial titration period.^{3,51} A 5-year follow-up study of MAD patients found mild, temporary subjective side effects, such as muscular or TMJ discomfort, but no changes in temporomandibular disorder prevalence using the Research Diagnostic Criteria for Temporomandibular Disorders.⁵⁷

Other Effects

Other MAD side effects include increased salivation, more frequent and excessive dry mouth, tongue discomfort, and a sense of suffocation.^{3,35,51} TSD side effects include excess salivation, drooling, dry mouth, and soft tissue irritation.³

PREDICTING AND IMPROVING ORAL APPLIANCE TREATMENT RESPONSE

Although most patients show an increased airway CSA with mandibular advancement, a minority of patients show no change or even a decreased CSA.^{9,12} MAD is a primarily structural treatment⁹ for a heterogeneous condition with nonanatomic etiology in up to 56% of patients.^{62,63} Consequently, MAD treatment completely resolves AHI to fewer than 5 events per hour in only 36% to 70% of OSA patients.^{24,38} Patients of female gender, younger age, smaller neck circumference, lower body mass

index, lower AHI, and supine-dependent OSA are predicted to have better treatment success with MADs.^{38,50,64,65} For TSDs, the best predictor of success is believed to be the presence of a single-site airway obstruction in supine-dependent OSA.¹

There is currently no validated clinical method to reliably differentiate responders from nonresponders.²⁴ Uncertainty of MAD treatment response is exacerbated by cost of treatment.⁶⁶ The literature lacks consensus in successful treatment outcome, with a majority of success criteria used in research not meshing with clinical definitions of OSA severity. Discordant success criteria makes comparing results of different studies difficult.⁶⁷ Prospective trials are under way to determine predictive potential of wakeful nasoendoscopy with Müller maneuver, drug-induced sleep endoscopy (DISE), and computational fluid dynamics.^{68,69} In the meantime, careful diagnosis and patient selection can increase the chances of success.

Cephalometry

Classically, shorter soft palate length, larger retro-palatal airway space, lower hyoid bone position, and a smaller mandible are associated with favorable MAD treatment response.^{1,65} Two recent systematic reviews,^{70,71} however, exploring cephalometric predictors for MAD response found a majority of observational studies on cephalometric predictors had flawed designs and failed to control for known confounding factors such as age, gender, body mass index, and baseline AHI. Definitions of treatment success were inconsistent, and heterogeneity in design prevented data synthesis and meta-analysis. Cephalometric parameters warranting further study included mandibular plane angle, hyoid to mandibular plane distance, and soft palate length.

Mandibular Advancement Device Design

Customized versus noncustomized

Both customized and noncustomized MADs reduce AHI in adult patients with OSA, but meta-analysis by the AASM/AADSM task force shows the improvements to be far greater in customized than noncustomized, with an AHI reduction of 13.89 events compared with 6.28 events per hour.⁴ Noncustomized MADs do not improve minimum Sao_2 , whereas customized MADs increase Sao_2 by 3.22%.⁴ They also do not reduce ESS scores to any significant level,⁷² whereas modest improvements in ESS scores can be expected from customized MADs.⁴

Titratable versus nontitratable

Both custom titratable and custom nontitratable MADs reduce AHI, improve Sao_2 and reduce

ESS scores, with meta-analyses by the AASM/AADSM task force showing the improvements to be approximately equivalent. Titratable MADs are recommended, however, over nontitratable MADs because the confidence interval for the effect of custom, titratable MADs is considerably smaller than for custom, nontitratable MADs.⁴

Due to dose-dependent effects, a clinical titration can improve MAD response and increase the amount of achievable protrusion. This is only possible with titratable MADs.²⁷

Therapeutic Diagnosis

Visualizing airway response

Treatment response to MAD can be estimated by visualizing airway response to mandibular protrusion or tongue thrust with nasoendoscopy or MRI in awake supine patients, or in patients using DISE. An increase in velopharyngeal CSA with mandibular advancement was significantly associated with greater AHI reduction with MADs.^{8,9,73,74} This was not fully predictive, however, with some patients without velopharyngeal widening showing high AHI reductions and vice versa.^{2,73}

Müller maneuver during nasoendoscopy induced significantly greater collapse in velopharyngeal and oropharyngeal CSA in MAD nonresponders than responders. With mandibular advancement, Müller maneuver induced a significantly greater collapse at all airway levels in nonresponders.⁸

On MRI, TSD responders showed a greater increase with TSD in AP diameter, minimum and mean CSA, and volume compared with nonresponders.²

Mandibular advancement device titration using polysomnography

Due to the dose-dependent relationship between efficacy and protrusion, increasing protrusion can increase treatment response in OSA patients. Conventional titration protocols use subjective symptoms to adjust mandibular advancement over several months and an outcome polysomnography (PSG) to confirm efficacy. There is no consensus, however, on an optimal titration protocol.⁶⁷

Manually increasing MAD advancement during overnight PSG can increase therapeutic efficacy of mandibular protrusion while reducing the number of titration visits required by most therapeutic protocols. After a clinical titration period, overnight MAD titration with PSG using the final MAD increased treatment response by an additional 9.9% to 30.4% of study subjects.^{28,75} Disappointing outcomes were reported, however, using an interim MAD without a period of clinical titration

because efficacy of the interim MAD was not translated to the final MAD.⁷⁶

Overnight MAD titration with PSG may require large single-night mandibular advancements to relieve respiratory events, in contrast to slow titration with conventional protocols. Significant jaw discomfort was noted with PSG titration without prior MAD use⁷⁶ but was not reported by patients who had an adaptation period with conventional clinical MAD titration.²⁸

Remote-controlled mandibular positioners

Using remote-controlled mandibular positioners (RCMPs) for single-night titration of MADs is similar to overnight MAD titration with PSG, with the advantage of adjustment done remotely without waking the patient or removing the appliance. Sleep architecture is maintained while protruding the mandible progressively until respiratory events are eliminated.^{26,77–80}

RCMPs can determine an effective target protrusion that is correlated with successful MAD treatment, may be able to identify nonresponders early, and reduce the number of titration reviews needed compared with conventional titration protocols.⁸¹ RCMP titration, however, necessitates large single-night mandibular advancements, which can cause significant jaw discomfort.²⁶

Combination Therapy

Mandibular advancement device and tongue-stabilizing devices

Because TSDs have somewhat different anatomic effects on the airway compared with MADs,² combining the therapies using a novel hybrid appliance resulted in augmentative treatment effects. Tongue suction with 6 mm mandibular protrusion produced better treatment response than 8 mm mandibular protrusion alone.³⁰ Combination therapy might improve OA treatment response and prove useful in patients with limited mandibular protrusion.

Mandibular advancement device and continuous positive airway pressure

Combining MAD and CPAP lowered the therapeutic CPAP pressure.^{82–85} An augmentative treatment effect on reducing AHI was found in patients not responding to CPAP or MAD use alone.^{82–84}

PRACTICE RECOMMENDATIONS

Indications

OAs in the form of MADs can be used as an alternative for patients of all OSA severities who are intolerant of CPAP or prefer an alternative therapy.⁴

Contraindications

Patients who are generally unsuitable for MAD treatment include edentulous patients and patients with inadequate number of sound teeth, severe periodontitis, and/or history of TMJ disease. There are exceptions, and MADs can sometimes be worn successfully by edentulous patients with good dentoalveolar ridges.¹ TSDs are not dependent on the dentition for retention and can be prescribed for edentulous or insufficiently dentate patients.

Appliance Selection

A plethora of designs is currently in use.⁸⁶ Based on current evidence, customized, titratable MADs are the preferred form of OAs.⁴ TSDs can be used in MAD nonresponders who want OA therapy.

Clinical Follow-up

Trained dentists in sleep practice should follow-up patients using MADs to reduce dental side effects and occlusal changes.⁴ Initial side effects are transient and reversible.^{51,52} Close monitoring and coaching through the initial period may improve compliance.^{35,50,51}

For initial comfort, mandibular advancement is 75% of maximum protrusion.¹ Clinical titration with an adaptation period increases the achievable protrusion and improves treatment response.²⁷

Clinical titration should be followed by overnight PSG. Subjective feedback is not sufficient to determine the optimal setting of MAD. Post-PSG titration has been shown to improve MAD efficacy significantly.^{4,28,75}

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