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Oral appliance therapy for snoring and obstructive sleep apnoea: a Practical Guide for Clinical Care

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ABSTRACT

Obstructive sleep apnoea (OSA) is a disorder associated with multiple cardiovascular, metabolic and neurocognitive comorbidities which impact the health and quality of life of patients. Dentists trained in dental sleep medicine have a significant role as part of a collaborative multidisciplinary team led by specialist sleep physicians in the overall management of OSA. Based on the recommendations of the medical team and the patient's preference, dentists have a pertinent role in the provision of oral appliance (OA) therapy for OSA. This narrative review provides a practical guide for OA therapy for snoring and OSA.

Keywords: Mandibular advancement appliance, mandibular advancement splint, mandibular advancement device, oral appliance, obstructive sleep apnoea, snoring, dental sleep medicine.

Abbreviations and acronyms: AHI = apnoea hypopnoea index; BMI = body mass index; MDA = mean disease alleviation; NREM = non-rapid eye movement; OA = oral appliance; OSA = obstructive sleep apnoea; PSG = polysomnography; REM = rapid eye movement; T2DM = type 2 diabetes mellitus; TMD = temporomandibular disorders; TMJ = temporomandibular joint.

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CLINICAL RELEVANCE

Provision of an oral appliance (OA) can improve the health and quality of life of patients who suffer from obstructive sleep apnoea (OSA). This narrative review explores the current scientific evidence and best practice principles to establish a practical guide for clinical care for OA therapy for snoring and OSA.

INTRODUCTION

Oral appliance (OA) therapy involves the provision of a removable appliance worn over the maxillary and mandibular teeth to protrude (advance) the mandible during sleep to treat snoring and obstructive sleep apnoea (OSA). These appliances may be referred to as a mandibular advancement appliance, a mandibular advancement splint or device or an oral appliance in the literature and in clinical practice. The use of the term OA may be confused with other types of OAs, such as orthodontic appliances and occlusal splints. However, OA appears to be a generally accepted term internationally as an appliance that advances the mandible for the treatment of snoring and OSA in the practice of dental sleep medicine (DSM).

In Australia, the prevalence of primary snoring is estimated to be 8%.[1] The estimated prevalence of moderate to severe OSA (apnoea-hypopnoea index (AHI) ≥15) is 10% among 30- to 49-year-old men; 17% among 50- to 70-year-old men; 3% among 30- to 49-year-old women; and 9% among 50- to 70-year-old women.[2] Untreated OSA is associated with a range of cardiovascular, metabolic, and neurocognitive comorbidities and with a significantly increased risk for mortality.[3–6] There is a complex interplay between OSA and its comorbidities, which necessitates a comprehensive approach to diagnosis and

treatment, emphasizing the need for integrated care strategies that address both OSA and its associated comorbidities.

METHODS

Experts in DSM co-authored this manuscript, which aims to serve as a practical guide for clinical care for OA therapy for snoring and OSA. A comprehensive review of the scientific literature up until December 2024 on the clinical application of OA therapy for snoring and OSA was conducted. These recommendations are grounded in the current evidence derived from peer-reviewed studies and best practice principles. However, clinical decision-making for OA therapy should be guided by the patient's specialist medical providers, based on an individual patient's needs and preferences, the availability of diagnostic resources, accessibility to treatment, and affordability. All contributors have provided their conflict-of-interest disclosures.

DISCUSSION

The use of an OA to treat OSA is a complex treatment modality which has both dental and medical components and consequences. The provision of OA therapy should be instituted by a dentist trained and competent in the field of DSM as part of a multidisciplinary team. There are multiple pathways for the provision of OA therapy. Once OA therapy has commenced, the providing dentist is responsible for monitoring the adherence and the efficacy of treatment. The dentist is also responsible for referring the patient to the appropriate medical practitioner as required and for communicating the patient's progress. Fig. 1 illustrates the patient-centred treatment flow. Advanced training and/or credentialing to achieve competency in the practice of DSM is necessary to ensure patients receive optimal clinical care. The following sections describe the major clinical considerations and consequences involved in OA therapy.

Clinical assessment and informed consent for oral appliance therapy

OA may be constructed after sleep physician-reported polysomnography (PSG) has diagnosed either primary snoring, with mild to moderate OSA, or severe OSA in a patient who is CPAP intolerant or noncompliant or would prefer this type of therapy.[7,8] Patient selection for the provision of an OA should occur after careful discussion with the medical team, including (but not limited to) their supervising medical practitioner, such as a specialist sleep physician and dentist trained in DSM about:

- Different treatment options available to the patient and the patient's preference
- The dental suitability of the patient for OA therapy
- The likely efficacy of OA therapy and the definition of clinical success in treating OSA
- The effect of OA on the symptoms of OSA
- The effect of OA on medical comorbidities
- OA design
- OA titration procedures
- The possible short-term and long-term side effects of OA therapy
- Future care and maintenance of the OA

Once it has been agreed that OA therapy is an appropriate therapeutic choice, a joint agreement should be reached by the patient and the dentist that outlines the responsibility of the dentist for provision, titration, and maintenance of the appliance, and of the patient with regards to adherence to treatment protocols, acknowledging their understanding of possible treatment sequelae and the need for ongoing monitoring with OA therapy.

Patients should be made aware that there are multiple treatment options for OSA. The choice of therapy is dependent on the wishes of the patient and the realistic consideration of treatment needs versus benefits as discussed with their medical team. Patients may also choose to alternate between two therapies.[32] Table 1 provides a summary of current treatment options for OSA. CPAP remains the gold standard therapy.

Clinical Guide: Oral appliance assessment and informed consent

- The provision of OA therapy must be under the supervision of a dentist trained in DSM.
- OA is one of several non-CPAP therapies available for patients who have a specialist sleep physician-reported diagnosis of OSA.
- Patient's preferred treatment is an indication for provision of OA therapy.
- The patient and the dentist must have an interactive discussion covering OA therapy which should include patient eligibility, treatment efficacy, side effects, appliance design, treatment protocol, and the need for ongoing maintenance and monitoring of the appliance.

Screening for obstructive sleep apnoea

A recent systematic review of the role of the dentist in sleep-disordered breathing concluded that dentists have a role in screening patients for sleep-disordered breathing.[33] Screening for OSA may apply to asymptomatic individuals who may not recognize symptoms or do not report symptoms of medical concern. To date, there is insufficient evidence that screening for OSA has a beneficial effect on long-term health

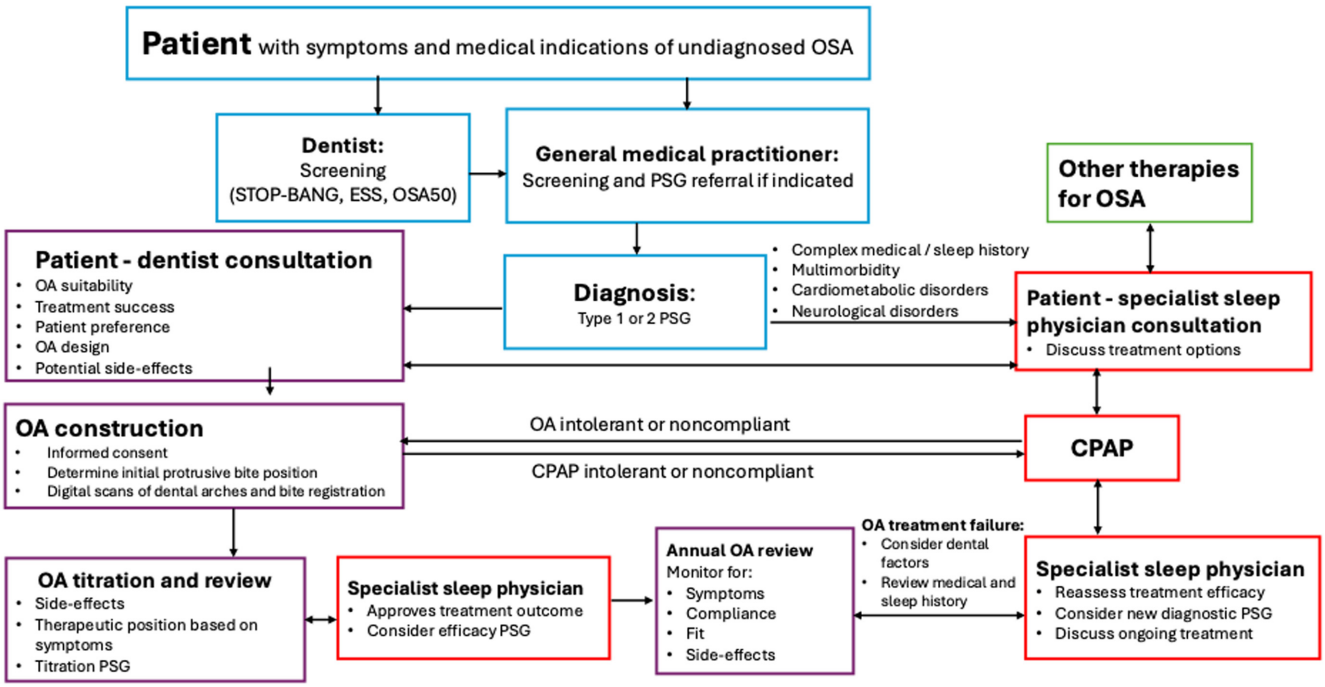

Fig. 1 Patient-centred treatment flow for oral appliance acquisition for obstructive sleep apnoea. OSA = obstructive sleep apnoea, ESS = Epworth Sleepiness Scale, Rx = treatment, OA = oral appliance, CPAP = continuous positive airway pressure.

outcomes, especially in asymptomatic individuals.[34] Patients who complain of signs and symptoms associated with OSA, including snoring, choking, gasping, daytime sleepiness, and changes to mood and cognitive processes, should be screened using validated questionnaires. There are several screening questionnaires which combine self-report of symptoms associated with OSA and clinical findings such as BMI and neck circumference. Potential screening tools include the STOP-BANG, Berlin Questionnaire, and the OSA50, often with the Epworth Sleepiness Scale, which is a subjective measure of daytime sleepiness used in conjunction with screening questionnaires to identify patients with symptomatic OSA. In general, these screening questionnaires tend to have high sensitivity and low specificity.[25,26] Hence, patients who have a positive screening test should be referred for further assessment utilizing PSG to confirm the diagnosis of OSA.

Dentists may refer a patient suspected of OSA for a diagnostic PSG to their general medical practitioner, who may then on-refer the patient to a specialist sleep physician. In Australia, to receive subsidized care based on the Medicare Benefit Schedule rules, there are 2 referral pathways for a sleep study: (i) referral of the patient to sleep or respiratory medicine specialist for a clinical assessment and PSG as deemed necessary by the specialist, or (ii) referral of the symptomatic patient to their general medical practitioner, who may request either an attended or unattended PSG without the patient needing prior assessment by a sleep or respiratory medicine specialist. To qualify for the latter pathway, the patient must have positive screening questionnaires such as a positive Berlin Questionnaire, OSA50 ≥ 5 or STOP-BANG ≥ 3 and an Epworth Sleepiness Scale ≥ 8.[26]

Clinical Guide: Screening for obstructive sleep apnoea

- Patients with symptoms suggestive of OSA such as snoring and waking gasping, mood and cognitive changes, and excessive sleepiness should be assessed with a validated screening test.
- Commonly used screening tests include the Berlin Questionnaire, STOP-BANG, OSA50 and the Epworth Sleepiness Scale.
- Patients with a positive screening test should be referred to their general medical practitioner or to a specialist sleep physician for diagnostic evaluation.

Patient selection for oral appliance therapy

Patient selection for OA therapy is a critical aspect of OA therapy. There are many considerations that will ultimately influence patient compliance and treatment effectiveness.

Dental suitability

There must be sufficient teeth or implant-retained restorations present in the maxillary and mandibular arches to provide adequate retention and comfort

Table 1. Treatment options for obstructive sleep apnoea

Modality	Indications	Poor responders
CPAP[9]	• Adults with OSA • Excessive sleepiness • Hypertension • Sleep physician recommendation	• Low arousal threshold • Comorbid insomnia
Oral appliance therapy[8, 10, 11]	• Mild–moderate OSA • Snoring • Anatomic upper airway constriction • Severe OSA with high REM AHI, high hypoxic burden or multiple comorbidities when CPAP noncompliant or intolerant	• Obesity class 2 or 3 (BMI ≥35 mg/kg^2) • REM predominant OSA • High hypoxic burden • Insufficient teeth for retention • Central apnoea • Systolic hypertension with moderate–severe OSA
Bilateral hypoglossal nerve stimulation[12, 13]	• BMI≤32 kg/m^2 • Age 21–75 years • No positional OSA (AHI <10) • Obstructive AHI: 20–60 • Combined mixed+ central AHI <10 • No positional OSA (defined as non-supine AHI < 10 + supine AHI ≥ non-supine AHI × 2)	• Concentric collapse of airway during apnoea using DISE (1 in 4 patients) • Mean difference in AHI was 18 at 5 years in 129 patients
Maxillomandibular advancement[14, 15]	• Clinically significant OSA with skeletal discrepancy (retrognathia, midface deficiency) • Failed CPAP • Surgical success defined as >50% reduction and AHI <20 and surgical cure as AHI <5	• Medically complex • Temporomandibular disorders
Positional devices[16, 17]	• Reduce time in supine position	• Low arousal threshold • Higher NREM AHI
Weight loss[18] • Dietary modifications[19] • Surgical[20] • Pharmacological[21]	• Obesity and OSA • Other medical disorders that are impacted by obesity	• 20%↓ in BMI associated with 57% ↓ in AHI • Poor response associated with fat distribution, ethnicity, supine OSA, REM OSA, anatomy, and Type 2 diabetes mellitus • Risk for some relapse of weight loss with time
Oral/nasal expiratory positive airway pressure[22, 23]	• Increase supine NREM AHI	• Increases poor sleep quality (leads to lower sleep efficiency)
Pharmacotherapy for OSA (acetazolamide and atomoxetine-plus-oxybutynin)[24]	• To increase muscle responsiveness • Reduce arousability • Change chemosensitivity	• Intolerance to side effects such as drowsiness and dry mouth
Aerobic exercise, 45 min of walking twice daily,[25, 26] resistance training,[27] and high-intensity training[28]	• Improve sleep quality • Improve health • Reduce rostral fluid shift	• Non-compliant • Health issues prevent compliance
Combination therapies[22, 23, 29–31]	• Insufficient clinical response to one therapy • Increased side effects from one therapy at a therapeutic dose • OA + CPAP = 35–40% reduction in CPAP pressure[29] • OA + positioner device reduces time spent in supine sleep	• Increased cost • Increased complexity of treatment and monitoring

AHI, apnoea-hypopnoea index; BMI, body mass index (kg/m^2); CPAP, continuous positive airway pressure; DISE, drug-induced sleep endoscopy; OA, oral appliance; OSA, obstructive sleep apnoea; RDI, respiratory disturbance index; REM, rapid eye movement.

while wearing the OA. A dental examination and the establishment of healthy periodontal and restorative status must precede appliance construction. Temporomandibular disorders should be assessed and managed in highly symptomatic patients prior to or during OA therapy, as oral and jaw pain and dysfunction are the most common causes of discontinuation of OA therapy[35] and may limit the mandibular range of motion and the protrusive titration of the appliance.[36] Any decision to obtain imaging of the temporomandibular joints (TMJs) should follow evidence-based guidelines after completion of a thorough history,

examination, and the provision of preliminary diagnoses of temporomandibular disorders.[37] The routine prescription of radiological studies such as cone-beam computed tomography (CBCT), computed tomography (CT) and magnetic resonance imaging (MRI) is not recommended prior to the provision of OA therapy, unless otherwise necessary for the assessment of oral and maxillofacial pathology. If the dentist is not knowledgeable in the diagnosis and management of TMD, referral to an appropriate dental specialist should be undertaken prior to the construction of an OA.

Clinical Guide: Oral appliance therapy selection criteria
The provision of OA therapy should only be considered if the patient meets the selection criteria:

- An adequately restored dentition with sufficient dental or implant-borne support for appliance retention.
- The health of the periodontium and the TMJs and supporting structures should be stable.
- Imaging should be based on the standard required for dental examination only (x-rays) and prescription of advanced imaging such as CBCT, CT and MRI should be limited for the assessment of oral and maxillofacial pathology.

Prediction of oral appliance therapy response

Multiple techniques including overnight OA titration studies,[38,39] nasendoscopy[40,41] and the measurement of base-of-tongue (but not concentric) airway collapse during drug-induced sleep endoscopy[40,42,43] may predict OA response, but the extra procedures increase the financial and clinical burden of OA use. In the past, a CPAP pressure of >12–13 cm H_20,[44,45] a higher BMI,[46,47] high AHI,[11,47,48] advancing age,[11] REM-dependent OSA,[11] and an increased hypoxic burden[49,50] have been indicators of potential poor response to OA therapy. Conversely, supine predominant OSA is associated with a stronger response to OA therapy.[51,52] Sleep endotypes in OSA describe the pathophysiological mechanisms behind the clinical presentation of OSA. Endotyping is not routinely available during PSG, and its role in patient selection for OA is currently unclear.[53,54] Prediction models of OA success involving CBCT and other awake measurements are not yet better than clinical assessments using criteria such as BMI, neck circumference, and AHI.

Definitions and determinants of success in oral appliance therapy

Multiple definitions of success have been used to describe the effects of OA therapy for OSA. This section describes these definitions and investigates the interactions between OA therapy and clinical outcomes in DSM.

Definition of oral appliance therapy success

OA therapy is less successful at reducing the AHI than CPAP.[55,56] Treatment success in OSA is traditionally measured by the reduction of the AHI and the subjective symptoms of sleep loss. In OA therapy, treatment efficacy has been defined in several different ways including (i) a reduction in the AHI to AHI < 5, (ii) a reduction to AHI < 10, (iii) a reduction in the AHI of >50% with an AHI < 15 and finally, (iv) a >50% reduction of the AHI. Generally, an AHI of >15 is considered to be clinically significant in the absence of comorbidities such as sleepiness or cardiovascular disease.[57,58] The ORCADES prospective cohort study investigated the efficacy of bi-block OA therapy with bilateral posterior connectors for the treatment of OSA for 337 CPAP-intolerant and non-compliant patients. At 2–3 months post-insertion, 76.2% of patients had a >50% reduction in the AHI, 63.5% had an AHI < 10 and 36% had a complete response with an AHI < 5.[48] The success rate at all cut-offs was highest for mild and lowest for severe OSA. Another study reported that at 5-year follow-up, OA efficacy had decreased significantly with 52% of the cohort maintaining a 50% reduction in AHI across all severity categories, while self-reported compliance (>4 h wear/night) remained high at 93.3%.[10] 89% of women and 76% of men maintained a 50% reduction in AHI.[35]

Factors affecting OSA severity in oral appliance therapy

The results of the ORCADES study highlight the underlying heterogeneity in OSA and the difficulty this poses for the prediction of OA therapy outcomes. Females have a lower OSA prevalence, a higher prevalence of REM-predominant apnoea[59–62] and less severe respiratory and hypoxic events than males in NREM.[63] The onset of menopause coincides with increased obesity and the central redistribution of body fat. The NREM AHI and the overall OSA severity are noted to increase.[64–66] Obesity is a major driver of OSA development and may induce upper airway stiffness and circulatory delay in males and females, while in males it is also associated with an increase in the arousal threshold.[67]

OA therapy produces a dose-dependent reduction in airway collapsibility,[68,69] with no effect on muscle responsiveness.[68] In 53 subjects, OA therapy success was predicted by pharyngeal collapsibility, lower loop

gain,[70] higher arousal threshold, and lower arousability.[71,72] In a small clinical trial (n = 20) patients with severe OSA were treated with OA therapy, and non-responders (AHI > 10) were sequentially provided adjunct therapies (supine avoidance therapy, expiratory positive airway pressure) and pharmacotherapeutics (atomoxetine-oxybutynin and 4 L/min of O_2) targeting loop gain and pharyngeal muscle activity. Successful treatment was achieved in all but one participant (AHI < 10).[22] The clinical applicability of this approach remains to be validated in larger clinical trials.

Oral appliance therapy and medical comorbidities

OA therapy may be used to treat OSA in patients who prefer OA therapy, or who are CPAP intolerant or non-compliant.[7,22,73] Termination of CPAP therapy is highest in the youngest and oldest patient groups with at least one major medical comorbidity.[74] The presence of medical comorbidities may seriously impact future health outcomes in patients with OSA over 50 years of age[75] and should be considered when deciding if OA therapy is appropriate. Both regular snoring[76] and OSA[77–79] are associated with an increased risk for adverse cardiovascular events, especially in younger patients and those with worse oxygen desaturation indices.[79] Meta-analyses comparing CPAP and OA confirm that CPAP performs significantly better than OA for the reduction of the AHI and oxygen desaturation indices, but is equivalent to OA for the reduction of sleepiness[80,81] and quality of life measures such as cognition.[82–84] CPAP and OA may have equivalent effects on systolic blood pressure over 1–6 months of use[82,85,86] including cases with moderate to severe OSA,[85] but the improvement of hypoxia with OA does not always parallel the reduction in the AHI.[50]

It is plausible that the superior adherence achieved with OA therapy may improve the long-term effectiveness of OA therapy in chronic disorders.[87] In a small study (n = 24) using OA to treat OSA in patients with T2DM, 3 months of OA therapy at 50% of maximum protrusion effectively reduced HbA1c levels in groups with mild and moderate OSA, but was unsuccessful in those with severe OSA and higher levels of obesity.[88] Six months use of an OA at 70% of maximum protrusion in patients with treatment-resistant T2DM effectively reduced the HbA1c levels in those with mild OSA.[89] There is a disparity in the available evidence for CPAP and OA for prevention and maintenance of long-term health outcomes, and more research is required in this area.

Clinical Guide: Treatment success with oral appliance therapy
Successful OA therapy requires a combination of:

- Regular use for at least 4 h/night.
- Improvement in the quality-of-life outcomes relevant to each patient such as excessive daytime sleepiness, mood and cognitive change.
- Clinically relevant reductions in the AHI and oxygen desaturation and improvements in sleep quality.
- OA efficacy may be altered by advancing age and lifestyle dependent changes which may influence the severity of OSA.
- As patients with OSA are at increased risk for the onset and exacerbation of cardiometabolic, cognitive and other medical disorders, they should be routinely monitored after OA provision so that changes in the effectiveness of OA therapy are detected early and dealt with by further titration of the appliance or referral to their supervising medical practitioner for review.

Oral appliance design

OA therapy stabilizes and improves the oropharyngeal airway during sleep by protruding the mandible relative to the maxilla, primarily increasing the lateral volume of the velopharynx[90–92] and altering flow characteristics in the upper airway.[93] OA can be fabricated from various materials including metal, silicone, acrylic, and nylon, and may be custom-fabricated or non-custom in a mono- or bi-bloc design. The bi-bloc appliances have separate maxillary and mandibular components, while the mono-bloc appliances are fabricated in one piece, usually at 75% of the maximum mandibular protrusion.[94] While all appliances may improve OSA,[95,96] the custom-fabricated OA has the advantages of superior retention and comfort,[97] and the bi-bloc appliances allow for titration, which can be maximised to adequately treat patients with moderate to severe OSA[98] or minimised to balance efficacy and side effects.[99] The use of temporary or non-titratable appliances is not recommended as they add to treatment cost and may prolong the time until effective reduction of the AHI. Custom appliances reduce the AHI and have been reported to have greater comfort than temporary appliances.[100]

A mono-bloc appliance has a large surface area of attachment between the maxillary and mandibular components, which distributes forces and prevents mandibular movements. In bi-bloc appliances, the attachment provides the method for protrusion of the mandible forwards against the maxilla and may limit vertical or lateral mandibular movement. Four bi-bloc coupling designs were identified in a recent network meta-analysis[101] and were classified according to their position on the appliance and whether the forces against the mandible are applied as a compressive force (the mandible is pushed forwards from the

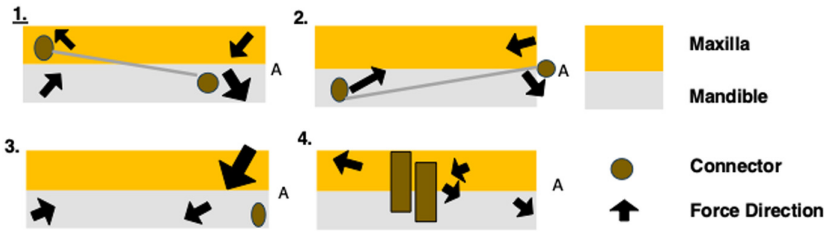

Fig. 2 Connector types for oral appliances and proposed resultant force vectors on the teeth. (1: posterior compression 2: mixed anterior and posterior compression 3: anterior traction 4: posterior traction, A = anterior).

posterior of the maxilla to the anterior of the mandible) or traction (the mandible is pulled forward, the attachment is from the anterior of the maxilla to the posterior of the mandible) (Fig. 2).[102] To date, these design differences have not been associated with clinically significant differences in either dental side effects (tooth movement)[103] or clinical outcomes (AHI reduction).[104,105] The location of the maxillo-mandibular coupling could be considered as a method for mitigation of forces applied to vulnerable teeth by titration.[94,106,107]

Clinical Guide: Oral appliance design

- The design of the OA should be decided after consideration that patient preference may affect treatment compliance while dentists need to balance the retention requirements of each design with the health and characteristics of the patient's dentition.
- A custom-fabricated, titratable, bi-bloc design is recommended for optimal appliance fit, retention, stability, comfort and efficacy.
- The use of a temporary or non-titratable OA is not recommended.

Titration of the oral appliance

Titration of the OA is the process used to establish the optimal subjective (using self-report) and objective (PSG-based) treatment outcomes for the patient. This primarily involves making gradual advancements in the anteroposterior position of the mandible. The vertical maxillo-mandibular relationships can also be changed if required. It may be necessary for the dentist to work as part of a multidisciplinary team to ensure that medical comorbidities and lifestyle factors which may also affect sleepiness and quality of life outcomes are treated and implemented, respectively.[108]

There is a dose-dependent relationship between gradually increased mandibular protrusion and a reduction in the AHI[49,109] which is non-linear.[110] This dose-dependent relationship has a higher impact for patients with more severe OSA[110] and possibly is less predictable for those with more vertical craniofacial phenotypes.[111] The range of maximum mandibular protrusion may increase during therapy[112] and may explain some of the variation between protrusive position and AHI reduction. The amount of vertical should be kept to the minimal level to improve patient comfort and treatment efficacy,[99,113] and anterior mandibular movement should be minimized, if possible based on efficacy, to decrease undesirable craniofacial changes and tooth movements.[114,115] A level of 50% of maximal protrusion appears to be effective in many cases,[116] while cases with moderate OSA experience less improvement after 70% of maximal protrusion, and higher than 70–75% of maximal protrusion may be required for patients with severe OSA.[117] It is noted that the amount of mandibular protrusion necessary for OA efficacy is based on OSA severity, whereby the target protrusion for mild, moderate, and severe OSA was noted to be 3.5 ± 1.8 mm, 5.8 ± 1.9 mm and 5.9 ± 2.2 mm, respectively.[110]

Positional devices limit the time spent sleeping in the supine position, a time when the AHI is often increased due to posture-mediated effects on lung function and airway collapsibility.[118,119] The unfavourable effect of the supine position on the airway may be further increased by mouth opening during sleep, and as such, elastics applied to the OA to maintain mouth closure[120] may be useful adjunctive treatments in patients with severe supine-based OSA. The supine AHI also needs to be considered post-OA therapy, as one-third of patients tend to shift from non-positional to supine-dependent positional OSA when using an OA.[121] Incomplete or non-responders to OA therapy may benefit from combination therapy from expiratory positive airway pressure,[22,23] CPAP,[29] and multilevel soft tissue surgery.[122] These possibilities should be discussed during the informed consent process. Once the final titration position of the OA has been achieved, the dentist may elect to undertake a type 3 home sleep study to determine if the final optimal end-point of titration has been achieved prior to finalizing titration and referring the patient back to the supervising medical practitioner to determine the efficacy of OA therapy.[123] Future developments in artificial intelligence are likely to simplify the ability of all clinicians involved in the patient's care to

monitor compliance and efficacy of OA therapy.[124] OA therapy is typically a life-long commitment. A 12-monthly follow-up appointment with the dentist is necessary to ensure ongoing effectiveness of OA therapy and to assess possible side effects. Table 2 outlines a checklist for the follow-up appointments.

Clinical Guide: Oral appliance titration

- OA titration should include gradual anteroposterior and when necessary vertical changes in the position of the mandible aimed at reducing symptoms and the AHI.
- The amount of protrusion should be kept to the minimum required for therapeutic efficacy and is commonly around 50% of the maximum horizontal protrusion (but may be as high as 70%–75% in severe OSA).
- Once the therapeutic position has been reached, a PSG under the supervision of the medical practitioner to assess the efficacy of OA therapy may be necessary especially for cases of moderate to severe OSA.
- Adjunctive therapies such as vertical elastics, positional devices, weight loss, CPAP and multilevel surgery should be considered if necessary to achieve clinical success.

Adherence and compliance to oral appliance therapy

Based on self-reported use, OA use among patients was 96% of nights per week and 80% of their sleep period.[125] Similarly, a self-reported adherence rate of 75% among 630 patients who continued to use their OA after 12 months of therapy was noted.[126] A scoping review of factors that positively influenced treatment adherence to OA therapy included: the effectiveness of therapy, the appliance being custom fabricated, the communication skills of the clinician, early recognition of side effects, stepwise titration of the OA, and a positive initial experience with using the OA. Factors that negatively impact treatment adherence included: type D personality, diminishing effectiveness of OA therapy, side effects with OA use, thermoplastic appliance, undergoing dental treatment during OA therapy, and poor initial experience with OA or CPAP therapy with inadequate guidance by professionals.[127] The first 20 days of OA use appear to be predictive of long-term use of the appliance.[128]

Over the last decade, microsensors to objectively measure compliance with OA therapy have been readily available and are particularly useful in situations where evidence of treatment adherence is critical as part of occupational health and safety requirements. These microsensors can be embedded into the appliance to provide reliable wear-time data. A prospective study of 51 patients over 3 months found using an embedded microsensor thermometer with on-chip integrated readout to be safe and feasible for objective

Table 2. Oral appliance therapy follow-up appointment checklist

Check item	Action required
Update medical history	• Review for conditions associated with worse sleep outcomes, for example, cardiometabolic disorders, cognitive dysfunction, onset of menopause, increase in BMI and continuous sleep deprivation (short sleep).
Subjective report of sleep quality: STOP-BANG, OSA50, Insomnia Severity Index, Berlin questionnaire, Epworth Sleepiness Scale	• Assess maintenance of sleep quality and for symptoms of residual OSA, any history of cognitive issues or other sleep disorders. • A written referral to the patient's medical team for further assessment may be necessary.
Subjective report of appliance efficacy and adherence	• Snoring, gasping or snorting for breaths, morning tiredness, daytime lethargy, drowsiness during sedentary circumstances, short-term memory loss, reduced concentration, low motivation or mood may indicate incomplete OA titration or persistent symptoms despite maximum titration. • Assess for side effects related to OA therapy. • Assess compliance to OA therapy (the number of days used per week, hours used per night, and correlation between OA use and sleep cycles associated with greater apnoeic events, e.g. REM sleep), self-perceived sleep quality, and the signs and symptoms of poor sleep and other sleep disorders.
Evaluate mandibular range of motion, temporomandibular health and occlusion	• Measure maximum mouth opening and protrusive range of movement. • Assess for tenderness or pain related to the TMJs and muscles of mastication, and for new TMJ sounds. • Assess for changes in occlusion and obtain clinical images where necessary to facilitate long-term monitoring.
Dental health	• Ensure patient undergoes regular dental check-ups and cleans. • Assess need for any restorative or periodontal care.

(continued)

Table 2 (continued)

Check item	Action required
Appliance fit	
	• Assess fit of both the maxillary and mandibular portions of the appliance (loose or tight). • Consider adjustments to relieve pressure or tighten the appliance to improve the fit.
Appliance condition	
	• Assess the overall integrity of the appliance. • Assess for stains and the need for a professional clean. • Assess for any cracks or breakages. • Assess the working order of the titration apparatus.

BMI, body mass index kg/m^2; OA, oral appliance; OSA, obstructive sleep apnoea; REM, rapid eye movement; TMJ, temporomandibular joint.

compliance monitoring whereby regular use of a custom fabricated OA was noted to be 82%–86% at an average of 6.7 h per night, which is higher than compliance with CPAP using compliance criteria of >4 h/night on 70% across all days. Careful assessment of compliance criteria is important when comparing treatments.[128,129]

Another study compared objectively measured adherence with OA therapy to self-reported use over 1 year and found that patients used their appliance on average 6.1 h per night and for 83% of nights per week, with a discontinuation rate of 9.8%. Of interest, self-reported compliance was noted to be overestimated by a mean of 30 min when compared to objective compliance monitoring.[130] In a recent large study, long-term adherence to OA therapy, when defined as >4 h per night for >4 days per week, was 93.3%, and adherence was 91.3% when defined as >6 h per night at the 5-year follow-up. 96.5% of patients continued to adhere to OA therapy at the 5-year follow-up, although the findings of this study were limited by the lack of objective adherence data and nearly half of the recruited patients withdrew from the study prior to the 5-year follow-up period.[10,131]

The use of objective measures of adherence with OA therapy has facilitated the comparison of their clinical effectiveness and health outcomes in comparison to that of CPAP. In spite of the lower efficacy of OA, a study comparing OA to CPAP based on mean disease alleviation (MDA), which is a measure of therapeutic effectiveness based on the combined function of efficacy and compliance, reported a 3-month MDA of 51.1% for OA therapy in patients with predominantly mild to moderate OSA (AHI 18 ± 11.9). This outcome was comparable to an MDA of 50% for CPAP. In essence, the higher compliance to OA therapy offsets its inferior efficacy when compared to CPAP and explains the similar MDA measures.[129] A recent randomized cross-over trial using objectively measured compliance found that 35 of 64 participants (55%) alternated CPAP and OA use, blending efficacy and adherence with reductions in important patient-centred outcomes.[118] Future research should investigate the relevance of MDA to long-term health outcomes. Of note, beyond objective compliance monitoring, microsensors are being developed to record multi-night snoring and breathing sounds, sleep position and cardiorespiratory parameters.[132] With ongoing research and development of microsensors, a true assessment of 'real world' clinical effectiveness will be the standard of care in the near future.

Clinical Guide: Adherence and compliance to oral appliance therapy

- Adherence to OA therapy may be impacted by ongoing treatment efficacy, custom fabricated appliance design and fit, and adverse side effects as well as personality, and communication features of the patient, the dentist and the entire interdisciplinary team.
- OA therapy may be equally effective as CPAP for treatment of OSA given the superior compliance to OA therapy which offsets its inferior efficacy (AHI reduction) compared to CPAP.

Side effects with oral appliance therapy

Side effects during the initial phase of OA therapy are common; however, they are typically mild and transient. Of note, early recognition and management of side effects are critical to treatment adherence. OA therapy requires the advancement of the mandible in an anterior direction, which produces backward-directed forces on the maxilla and maxillary teeth, and forward-directed forces on the mandible and mandibular teeth. In the morning after overnight OA use, a reduction in biting force may be noted upon removal of the appliance, and an abnormal occlusion (altered bite) can be present for up to 75 min. It is postulated that the abnormal occlusion is related to non-physiological displacement of the TMJs, non-physiological extension of the masticatory muscles and continuous compression of the periodontal ligaments with hours of OA use.[133] Other short-term side effects of OA therapy include general discomfort with appliance use, excessive salivation, dry mouth, difficulty swallowing with the appliance in situ, discomfort in the buccal mucosa, tongue, gingiva and teeth, tenderness and pain of the masticatory muscles and/or TMJs, jaw stiffness, and dental restoration damage.[113,134–136] These side effects, especially excessive salivation and oral tissue discomfort, are typically

transient and resolve with minor OA adjustments or with time.[137,138]

Long-term OA therapy is associated with craniofacial and dentoalveolar changes that can be progressive and irreversible.[139,140] Within a period of 1–2 years of continual OA therapy, patients may present occlusal changes regardless of the patient's age.[138,141–144] Study model analysis after 5 years of OA therapy noted that occlusal changes increase with usage over time. As many as 85% of patients show some occlusal changes, but few are aware of occlusal changes in the long term.[125,143] Dentoalveolar changes occur with all OA design types and are correlated with the duration of therapy.[115] In a study with a mean treatment duration of 11 years, 100% of patients showed some occlusal changes. A mean reduction in overjet of 1.9 mm and in overbite of 2.3 mm was observed related to retroclination of the maxillary incisors and proclination of the mandibular incisors, but these changes were not generally associated with periodontal disease or bone loss.[115,140,145] Other dentoalveolar changes associated with OA use include mesial tipping of the mandibular molars, distal tipping of maxillary molars, and the development of posterior open bites and anterior cross-bites, resulting in fewer occlusal contacts in the premolar and molar region.[142,146] The number of tooth contacts fluctuates over time and may improve.[125,147,148] The use of morning occlusal guides (morning repositioners) has gained popularity over the past decade, with some OA manufacturers recommending their use to prevent dentoskeletal side effects. A cephalometric study involving >1 year follow-up of patients undergoing OA therapy along with a morning occlusal guide and a randomized controlled trial revealed significant upper incisor retroclination and lower incisor proclination despite morning occlusal guide use. In essence, the use of morning occlusal guides to prevent dentoskeletal side effects related to OA therapy is not recommended.[149,150]

Craniofacial changes with OA therapy include downward rotation of the mandible with an increased mandibular plane angle and an increase in the anterior lower facial height, increased total anterior facial height, flattening of the curve of spee in the premolar region, and an increase in the arch length and width as the mandibular canine to second molar segment moves forward in relation to the maxillary arch, resulting in reduction of crowding of teeth and the development of interproximal spaces which may be subject to food traps.[125,137,141,151–156]

Although most patients are not concerned about the craniofacial and dentoalveolar changes that occur with long-term OA therapy, this possibility should be discussed during the informed consent process prior to the fabrication and fit of the OA. In addition, regular follow-up of patients undergoing OA therapy should be facilitated to enable early recognition and treatment where necessary. The decision to terminate OA therapy because of side effects should only be prescribed once an alternative successful treatment is available. Termination of OA therapy must take into account the patient's preference, symptoms, OSA severity and associated medical comorbidity. Given the treatment of OSA is often medically necessary, written communication to the patient's medical team outlining the decision to terminate OA therapy and the need to review the patient to facilitate discussion on alternative treatment options is required. Table 3 outlines the clinical considerations related to potential side effects during OA therapy.[157]

Clinical Guide: Management of potential side effects of oral appliance therapy

- Dentists must discuss potential short-term and long-term side effects prior to the fabrication and provision of OA therapy.
- There is a need for ongoing regular follow-up appointments to monitor the development and persistence of any side effects.
- Early identification of side effects related to OA therapy, and where necessary reassurance and treatment is essential.
- Side effects during the initial stages of OA therapy are common and typically transient.
- Significant long-term side effects of OA therapy include painful temporomandibular disorders, changes in occlusion, and changes in craniofacial morphology.
- Changes in the patient's occlusion and/or craniofacial morphology related to OA therapy does not warrant termination of treatment.
- Use of morning occlusal guides to prevent dentoskeletal side effects related to OA therapy is not recommended.
- Symptomatic temporomandibular disorders in the long term are experienced by a small percentage of patients undergoing OA therapy and require diagnosis and management.
- Termination of OA therapy may be considered, however, must take into account the patient's treatment preference, symptoms, OSA severity and associated medical comorbidity.
- Upon termination of OA therapy for OSA, written communication to the medical team is necessary to facilitate alternative treatment options.

Multidisciplinary team approach for oral appliance therapy

Sleep cuts across all body systems and healthcare disciplines and, as such, sleep medicine is a multidisciplinary field in which dentists have an adjunctive but significant collaborative role. The role of dentists includes screening symptomatic patients for sleep-disordered breathing and the provision of OA therapy for snoring and OSA. Dentists should not directly assess patients and request a PSG with the intent to gather sleep and respiratory parameters to ultimately diagnose sleep disordered breathing and prescribe OA therapy or other treatments. Training in sleep medicine at dental schools remains grossly

Table 3. Clinical considerations of potential side effects related to oral appliance therapy

Clinical Considerations	Clinical Presentations of Side Effects
Temporomandibular function	• Transient morning jaw soreness • Persistent temporomandibular joint (TMJ) tenderness or pain • Persistent tenderness or pain of the muscles of mastication • New onset TMJ sounds, catching or locking • Morning temporal headache
Intraoral tissues	• Soft tissue and tongue irritation • Gingival irritation • Excessive salivation and drooling • Dry mouth
Occlusion	• Altered occlusal contacts / bite changes • Incisor position changes • Decreased overjet and overbite • Alterations in the position of the mandibular canines and molars • Development of interproximal gaps (food traps)
Teeth and restorations	• Tooth mobility • Tooth fractures related to changes in occlusion • Damage to dental restorations caused by appliance retention
Appliance	• Breakage related to sleep bruxism • Allergies to appliance material • Gagging • Anxiety related to fear of choking or difficulty swallowing

inadequate, and dentists are not qualified to treat sleep disorders. Even if a dentist is trained in DSM, it is the role of the patient's medical team, including the supervising medical practitioner, such as their general medical practitioner or specialist sleep physician, to diagnose OSA.[158] Dentists choosing to independently diagnose and manage sleep disorders would be appropriately viewed as practising outside the scope of practice of dentistry as regulated by the Dental Board of Australia.

Table 4 details the major procedural standards developed by this article and the actions that should

Table 4. Practical guide for clinical care for oral appliance therapy for snoring and obstructive sleep apnoea

Practical guide	Clinical care required by dentists
1 The provision of oral appliance (OA) therapy should be instituted by a dentist trained and competent in the field of dental sleep medicine.	• Dentists must engage in education and training in the field of dental sleep medicine. • Appropriate qualification and/or certification to demonstrate competency in the practice of dental sleep medicine is recommended.
2 Screening questionnaires may be administered to symptomatic individuals.	• Validated questionnaires including the OSA50, Berlin questionnaire, STOP-BANG and Epworth Sleepiness Scale should be used.
3 Dentists must obtain the diagnosis for the sleep disorder using the accepted pathway involving the patient's supervising medical practitioner.	• Dentists may not provide polysomnography (PSG) to patients with the intent to diagnose and treat obstructive sleep apnoea (OSA) or other sleep disorders without collaboration with a multidisciplinary team including the patient's supervising medical practitioner such as the general medical practitioner or specialist sleep physician.
4 OA therapy may be provided to patients with snoring and OSA based on recommendations from the medical team.	• Dentists should involve patients in the decision-making process regarding the design and use of OA.
5 The patient's supervising medical practitioner has clinical oversight about the final efficacy of the OA including the need for post-treatment PSG.	• The dentist should provide written communication to the patient's medical team on the completion of the OA titration process and as necessary throughout the patient's treatment journey.
6 Dentists providing OA therapy must assess the patient prior to the provision of the appliance, continue to assess and monitor the patient until such time as therapeutic efficacy with OA therapy has been achieved and thereafter continue to monitor the patient on an annual basis.	• The OA fit, comfort, side effects and the patient's self-reported compliance and symptom level should be assessed and recorded. • Non-compliance, worsening symptoms and side effects should be addressed including by referral back to the patient's supervising medical practitioner where appropriate.

be undertaken by OA dental providers to ensure that they are achieved. They cover the nature of the interactions that are required between the dentist, the patient, and the supervising medical practitioner, and they are intended to provide safety and efficacy in the provision of OA therapy.

Clinical Guide: Multidisciplinary teamwork for oral appliance therapy

- Dentists must work as part of a collaborative, multidisciplinary medical team which is led by the supervising medical practitioner.
- Dentists have a role in screening symptomatic patients for sleep-disordered breathing and in the provision of OA therapy for snoring and OSA.
- Dentists must not provide diagnostic PSG for patients with the intent to diagnose and prescribe OA therapy without the input of the supervising medical team.
- Dentists must follow-up patients undergoing OA therapy on an annual basis.

CONCLUSIONS

Dental sleep medicine is a rapidly evolving field, and it is the responsibility of each dentist to ensure that they are well educated and trained in the field so that they can assess new developments and provide evidence-based care for patients. This work provides a practical clinical guide for OA therapy for snoring and OSA. It summarizes the current state of the field of OA therapy, including the patient's treatment journey, consent process, appliance design, titration, side effects, treatment success, and OA compliance, adherence, and monitoring. It sets out the roles and responsibilities of the dentist and the patient in a multidisciplinary, collaborative setting under the supervision of the lead medical practitioner and outlines a practical guide for clinical care through a series of 'action items' designed to improve the initiation, provision, monitoring, and ongoing outcomes of OA therapy.

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R Balasubramaniam: Conceptualization; methodology; writing – original draft; writing – review and editing. **K McCloy:** Conceptualization; methodology; writing – review and editing. **FR Almeida:** Writing – review and editing. **PA Cistulli:** Conceptualization; writing – review and editing.

REFERENCES

1. Appleton SL, Gill TK, Lang CJ, *et al*. Prevalence and comorbidity of sleep conditions in Australian adults: 2016 Sleep Health Foundation national survey. Sleep Health 2018;4(1):13–19. https://doi.org/10.1016/j.sleh.2017.10.006.
2. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. Am J Epidemiol 2013;177(9):1006–1014.
3. Uyar M, Davutoglu V. An update on cardiovascular effects of obstructive sleep apnoea syndrome. Postgrad Med J 2016;92(1091):540–544.
4. Heffernan A, Duplancic D, Kumric M, Ticinovic Kurir T, Bozic J. Metabolic crossroads: unveiling the complex interactions between obstructive sleep apnoea and metabolic syndrome. Int J Mol Sci 2024;25(6):3243. https://doi.org/10.3390/ijms25063243.
5. Kim HC, Young T, Matthews CG, Weber SM, Woodward AR, Palta M. Sleep-disordered breathing and neuropsychological deficits. A population-based study. Am J Respir Crit Care Med 1997;156(6):1813–1819.
6. Kim JY, Ko I, Kim DK. Association of obstructive sleep apnea with the risk of affective disorders. JAMA Otolaryngol Head Neck Surg 2019;145(11):1020–1026.
7. Jacobowitz O, Afifi L, Alkan U, *et al*. Endorsement of "European Respiratory Society guideline on non-CPAP therapies for obstructive sleep apnoea" by World Sleep Society. Sleep Med 2024;113:293–298.
8. Randerath W, Verbraecken J, de Raaff CAL, *et al*. European Respiratory Society guideline on non-CPAP therapies for obstructive sleep apnoea. Eur Respir Rev 2021;30(162):210200.
9. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: an American academy of sleep medicine clinical practice guideline. J Clin Sleep Med 2019;15(2):335–343.
10. Vecchierini MF, Attali V, Collet JM, *et al*. Mandibular advancement device use in obstructive sleep apnea: ORCADES study 5-year follow-up data. J Clin Sleep Med 2021;17(8):1695–1705.
11. Sutherland K, Takaya H, Qian J, Petocz P, Ng AT, Cistulli PA. Oral appliance treatment response and polysomnographic phenotypes of obstructive sleep apnea. J Clin Sleep Med 2015;11(8):861–868.
12. Eastwood PR, Barnes M, MacKay SG, *et al*. Bilateral hypoglossal nerve stimulation for treatment of adult obstructive sleep apnoea. Eur Respir J 2020;55(1):1901320. https://doi.org/10.1183/13993003.01320-2019.
13. Costantino A, Rinaldi V, Moffa A, *et al*. Hypoglossal nerve stimulation long-term clinical outcomes: a systematic review and meta-analysis. Sleep Breath 2020;24(2):399–411.
14. Zaghi S, Holty JE, Certal V, *et al*. Maxillomandibular advancement for treatment of obstructive sleep apnea: a meta-analysis. JAMA Otolaryngol Head Neck Surg 2016;142(1):58–66. https://doi.org/10.1001/jamaoto.2015.2678.
15. Prinsell JR. Maxillomandibular advancement (MMA) in a site-specific treatment approach for obstructive sleep apnea: a surgical algorithm. Sleep Breath 2000;4(4):147–154.
16. Mok Y, Tan A, Hsu PP, *et al*. Comparing treatment effects of a convenient vibratory positional device to CPAP in positional OSA: a crossover randomised controlled trial. Thorax 2020;75(4):331–337. https://doi.org/10.1136/thoraxjnl-2019-213547.
17. Ravesloot MJL, White D, Heinzer R, Oksenberg A, Pepin JL. Efficacy of the new generation of devices for positional therapy for patients with positional obstructive sleep apnea: a systematic review of the literature and meta-analysis. J Clin Sleep Med 2017;13(6):813–824.

18. Malhotra A, Heilmann CR, Banerjee KK, Dunn JP, Bunck MC, Bednarik J. Weight reduction and the impact on apnea-hypopnea index: a systematic meta-analysis. Sleep Med 2024;121:26–31.
19. Kuna ST, Reboussin DM, Strotmeyer ES, *et al*. Effects of weight loss on obstructive sleep apnea severity. Ten-year results of the sleep AHEAD study. Am J Respir Crit Care Med 2021;203(2):221–229. https://doi.org/10.1164/rccm.201912-2511OC.
20. Demaeyer N, Bruyneel M. Factors associated with persistent obstructive sleep apnea after bariatric surgery: a narrative review. Nat Sci Sleep 2024;16:111–123.
21. Malhotra A, Grunstein RR, Fietze I, *et al*. Tirzepatide for the treatment of obstructive sleep apnea and obesity. N Engl J Med 2024;391(13):1193–1205. https://doi.org/10.1056/NEJMoa2404881.
22. Aishah A, Tong BKY, Osman AM, *et al*. Stepwise add-on and endotype-informed targeted combination therapy to treat obstructive sleep apnea: a proof-of-concept study. Ann Am Thorac Soc 2023;20(9):1316–1325. https://doi.org/10.1513/AnnalsATS.202210-892OC.
23. Lai V, Tong BK, Tran C, *et al*. Combination therapy with mandibular advancement and expiratory positive airway pressure valves reduces obstructive sleep apnea severity. Sleep 2019;42(8):zsz119. https://doi.org/10.1093/sleep/zsz119.
24. Sands SA, Collet J, Gell LK, *et al*. Combination pharmacological therapy targeting multiple mechanisms of sleep apnoea: a randomised controlled cross-over trial. Thorax 2024;79(3):259–268.
25. Mangione CM, Barry MJ, Nicholson WK, *et al*. Screening for obstructive sleep apnea in adults: us preventive services task force recommendation statement. JAMA 2022;328(19):1945–1950. https://doi.org/10.1001/jama.2022.20304.
26. Hamilton GS, Chai-Coetzer CL. Update on the assessment and investigation of adult obstructive sleep apnoea. Aust J Gen Pract 2019;48(4):176–181.
27. Lin CF, Ho NH, Hsu WL, Lin CH, Wang YH, Wang YP. Effects of aerobic exercise and resistance training on obstructive sleep apnea: a systematic review and meta-analysis. J Clin Sleep Med 2024;20(11):1839–1849.
28. Lins-Filho O, Germano-Soares AH, Aguiar JLP, *et al*. Effect of high-intensity interval training on obstructive sleep apnea severity: a randomized controlled trial. Sleep Med 2023;112:316–321.
29. Tong BK, Tran C, Ricciardiello A, *et al*. CPAP combined with oral appliance therapy reduces CPAP requirements and pharyngeal pressure swings in obstructive sleep apnea. J Appl Physiol 2020;129(5):1085–1091.
30. Alshhrani WM, Hamoda MM, Okuno K, *et al*. The efficacy of a titrated tongue-stabilizing device on obstructive sleep apnea: a quasi-experimental study. J Clin Sleep Med 2021;17(8):1607–1618.
31. Dieltjens M, Vroegop AV, Verbruggen AE, *et al*. A promising concept of combination therapy for positional obstructive sleep apnea. Sleep Breath 2015;19(2):637–644.
32. Hamoda MM, Hyunh N, Ayas NT, *et al*. Continuous positive airway pressure and mandibular advancement splints: the CHOICE multi-center open-label randomized clinical trial. Eur Respir J 2024 in press. https://doi.org/10.1183/13993003.01100-2024.
33. Gianoni-Capenakas S, Gomes AC, Mayoral P, Miguez M, Pliska B, Lagravere M. Sleep-disordered breathing: the dentists' role-a systematic review. J Dent Sleep Med 2020;7(1):1–15.
34. Jin J. Screening for obstructive sleep apnea. JAMA 2022;328(19):1988.
35. Vecchierini MF, Attali V, Collet JM, *et al*. Sex differences in mandibular repositioning device therapy effectiveness in patients with obstructive sleep apnea syndrome. Sleep Breath 2019;23(3):837–848. https://doi.org/10.1007/s11325-018-1766-8.
36. Dinsdale A, Liang Z, Thomas L, Treleaven J. Are jaw range of motion, muscle function and proprioception impaired in adults with persistent temporomandibular disorders? A systematic review and meta-analysis. J Oral Rehabil 2020;47(11):1448–1478.
37. Schiffman E, Ohrbach R, Truelove E, *et al*. Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Group†. J Oral Facial Pain Headache 2014;28(1):6–27. https://doi.org/10.11607/jop.1151.
38. Remmers JE, Topor Z, Grosse J, *et al*. A feedback-controlled mandibular positioner identifies individuals with sleep apnea who will respond to oral appliance therapy. J Clin Sleep Med 2017;13(7):871–880. https://doi.org/10.5664/jcsm.6656.
39. Mosca EV, Bruehlmann S, Zouboules SM, *et al*. In-home mandibular repositioning during sleep using MATRx plus predicts outcome and efficacious positioning for oral appliance treatment of obstructive sleep apnea. J Clin Sleep Med 2022;18(3):911–919.
40. Van den Bossche K, Op de Beeck S, Dieltjens M, *et al*. Multimodal phenotypic labelling using drug-induced sleep endoscopy, awake nasendoscopy and computational fluid dynamics for the prediction of mandibular advancement device treatment outcome: a prospective study. J Sleep Res 2022;31(6):e13673.
41. Sutherland K, Chan ASL, Ngiam J, Dalci O, Darendeliler MA, Cistulli PA. Awake multimodal phenotyping for prediction of oral appliance treatment outcome. J Clin Sleep Med 2018;14(11):1879–1887.
42. Op de Beeck S, Vena D, Mann D, *et al*. Polysomnographic airflow shapes and site of collapse during drug-induced sleep endoscopy. Eur Respir J 2024;63(6):2400261.
43. Kastoer C, Op de Beeck S, Dom M, *et al*. Drug-induced sleep endoscopy upper airway collapse patterns and maxillomandibular advancement. Laryngoscope 2020;130(4):E268–E274.
44. Sutherland K, Phillips CL, Davies A, *et al*. CPAP pressure for prediction of oral appliance treatment response in obstructive sleep apnea. J Clin Sleep Med 2014;10(9):943–949. https://doi.org/10.5664/jcsm.4020.
45. Storesund A, Johansson A, Bjorvatn B, Lehmann S. Oral appliance treatment outcome can be predicted by continuous positive airway pressure in moderate to severe obstructive sleep apnea. Sleep Breath 2018;22(2):385–392.
46. Suzuki K, Nakata S, Tagaya M, *et al*. Prediction of oral appliance treatment outcome in obstructive sleep apnoea syndrome: a preliminary study. B-ENT 2014;10(3):185–191.
47. Chen H, Eckert DJ, van der Stelt PF, *et al*. Phenotypes of responders to mandibular advancement device therapy in obstructive sleep apnea patients: a systematic review and meta-analysis. Sleep Med Rev 2020;49:101229.
48. Vecchierini MF, Attali V, Collet JM, *et al*. A custom-made mandibular repositioning device for obstructive sleep apnoea-hypopnoea syndrome: the ORCADES study. Sleep Med 2016;19:131–140.
49. Kato J, Isono S, Tanaka A, *et al*. Dose-dependent effects of mandibular advancement on pharyngeal mechanics and nocturnal oxygenation in patients with sleep-disordered breathing. Chest 2000;117(4):1065–1072.
50. Park JW, Almeida FR. Disparities in oxygen saturation and hypoxic burden levels in obstructive sleep apnoea patient's response to oral appliance treatment. J Oral Rehabil 2022;49(6):633–643.
51. Pu L, Long H. Is the response rate of oral appliance therapy for subjects with and without position-dependent

obstructive sleep apnoea different? Evid Based Dent 2022;23 (3):124–125.

52. Marklund M, Persson M, Franklin KA. Treatment success with a mandibular advancement device is related to supine-dependent sleep apnea. Chest 1998;114(6):1630–1635.
53. Tolbert TM, Schoenholz RL, Parekh A, *et al*. Night-to-night reliability and agreement of obstructive sleep apnea pathophysiologic mechanisms estimated with phenotyping using polysomnography in cognitively normal elderly participants. Sleep 2023;46(8):zsad058. https://doi.org/10.1093/sleep/zsad058.
54. Strassberger C, Hedner J, Sands SA, *et al*. Night-to-night variability of polysomnography-derived physiologic endotypic traits in patients with moderate to severe OSA. Chest 2023;163(5):1266–1278. https://doi.org/10.1016/j.chest.2022.12.029.
55. Chang JL, Goldberg AN, Alt JA, *et al*. International consensus statement on obstructive sleep apnea. Int Forum Allergy Rhinol 2023;13(7):1061–1482.
56. Agarwal S, Agarwal S, Garg Y, *et al*. Efficacy of titratable mandibular advancement device versus continuous positive airway pressure therapy in the treatment of obstructive sleep apnea: a clinical crossover trial. Med J Armed Forces India 2023;79(Suppl 1):S84–S93. https://doi.org/10.1016/j.mjafi.2022.01.002.
57. Hirotsu C, Haba-Rubio J, Andries D, *et al*. Effect of three hypopnea scoring criteria on OSA prevalence and associated comorbidities in the general population. J Clin Sleep Med 2019;15(2):183–194. https://doi.org/10.5664/jcsm.7612.
58. Sateia MJ. International classification of sleep disorders-third edition: highlights and modifications. Chest 2014;146 (5):1387–1394.
59. Sasai-Sakuma T, Kayaba M, Inoue Y, Nakayama H. Prevalence, clinical symptoms and polysomnographic findings of REM-related sleep disordered breathing in Japanese population. Sleep Med 2021;80:52–56.
60. Qanash S, Mufti H, Alhejaili F, *et al*. The prevalence of rapid eye movement-related obstructive sleep apnea in a sample of Saudi population. Ann Thorac Med 2023;18(2):90–97.
61. Bahammam RA, Al-Qahtani KM, Aleissi SA, Olaish AH, Almeneessier AS, Bahammam AS. The associations of gender, menopause, age, and asthma with REM-predominant obstructive sleep apnea: a prospective observational study. Nat Sci Sleep 2020;12:721–735.
62. Koo BB, Dostal J, Ioachimescu O, Budur K. The effects of gender and age on REM-related sleep-disordered breathing. Sleep Breath 2008;12(3):259–264.
63. Won CHJ, Reid M, Sofer T, *et al*. Sex differences in obstructive sleep apnea phenotypes, the multi-ethnic study of atherosclerosis. Sleep 2020;43(5):zsz274. https://doi.org/10.1093/sleep/zsz274.
64. Matthews KA, Lee L, Kravitz HM, *et al*. Influence of the menopausal transition on polysomnographic sleep characteristics: a longitudinal analysis. Sleep 2021;44(11):zsab139. https://doi.org/10.1093/sleep/zsab139.
65. Dugral E, Ordu G. Differences in polysomnography parameters of women in the post and transitional phases of menopause. Cureus 2021;13(12):e20570.
66. Zhou Y, Liu F, Li C, *et al*. Association of snoring and body composition in (peri-post) menopausal women. BMC Womens Health 2020;20(1):175.
67. Nokes B, Orr JE, White S, *et al*. Effect of obesity on sleep apnea pathogenesis differs in women versus men: multiple mediation analyses in the retrospective SNOOzzzE cohort. J Appl Physiol 2024;136(6):1516–1525.
68. Bamagoos AA, Cistulli PA, Sutherland K, *et al*. Dose-dependent effects of mandibular advancement on upper airway collapsibility and muscle function in obstructive sleep apnea. Sleep 2019;42(6):zsz049. https://doi.org/10.1093/sleep/zsz049.
69. Ng AT, Gotsopoulos H, Qian J, Cistulli PA. Effect of oral appliance therapy on upper airway collapsibility in obstructive sleep apnea. Am J Respir Crit Care Med 2003;168(2):238–241.
70. Op de Beeck S, Dieltjens M, Azarbarzin A, *et al*. Mandibular advancement device treatment efficacy is associated with polysomnographic endotypes. Ann Am Thorac Soc 2021;18 (3):511–518.
71. Bamagoos AA, Cistulli PA, Sutherland K, *et al*. Polysomnographic endotyping to select patients with obstructive sleep apnea for oral appliances. Ann Am Thorac Soc 2019;16 (11):1422–1431. https://doi.org/10.1513/AnnalsATS.201903-190OC.
72. Antonaglia C, Vidoni G, Contardo L, *et al*. Low arousal threshold estimation predicts failure of mandibular advancement devices in obstructive sleep apnea syndrome. Diagnostics Basel 2022;12(10):2548. https://doi.org/10.3390/diagnostics12102548.
73. Ramar K, Dort LC, Katz SG, *et al*. Clinical practice guideline for the treatment of obstructive sleep apnea and snoring with oral appliance therapy: an update for 2015. J Clin Sleep Med 2015;11(7):773–827.
74. Pepin JL, Bailly S, Rinder P, *et al*. CPAP therapy termination rates by OSA phenotype: a French nationwide database analysis. J Clin Med 2021;10(5).
75. Marrone O, Lo Bue A, Salvaggio A, Dardanoni G, Insalaco G. Comorbidities and survival in obstructive sleep apnoea beyond the age of 50. Eur J Clin Investig 2013;43(1):27–33.
76. Lechat B, Naik G, Appleton S, *et al*. Regular snoring is associated with uncontrolled hypertension. NPJ Digit Med 2024;7 (1):38.
77. Yao X, Li N, Heizhati M, *et al*. Obstructive sleep apnea remains a risk factor for major adverse cardiovascular and cerebrovascular events even in hypertensive patients under treatment: the Urumqi Research on Sleep Apnea and Hypertension (UROSAH) data. Cardiovasc Diagn Ther 2023;13 (6):968–978. https://doi.org/10.21037/cdt-23-284.
78. Zhang Y, Hao W, Fan J, *et al*. Association between obstructive sleep apnea and cardiovascular events in acute coronary syndrome patients with or without revascularization—a prospective cohort study. Circ J 2023;87(10):1369–1379.
79. Trzepizur W, Blanchard M, Ganem T, *et al*. Sleep apnea-specific hypoxic burden, symptom subtypes, and risk of cardiovascular events and all-cause mortality. Am J Respir Crit Care Med 2022;205(1):108–117. https://doi.org/10.1164/rccm.202105-1274OC.
80. Trzepizur W, Cistulli PA, Glos M, *et al*. Health outcomes of continuous positive airway pressure versus mandibular advancement device for the treatment of severe obstructive sleep apnea: an individual participant data meta-analysis. Sleep 2021;44(7):zsab015. https://doi.org/10.1093/sleep/zsab015.
81. Schwartz M, Acosta L, Hung YL, Padilla M, Enciso R. Effects of CPAP and mandibular advancement device treatment in obstructive sleep apnea patients: a systematic review and meta-analysis. Sleep Breath 2018;22(3):555–568.
82. Phillips CL, Grunstein RR, Darendeliler MA, *et al*. Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea: a randomized controlled trial. Am J Respir Crit Care Med 2013;187(8):879–887. https://doi.org/10.1164/rccm.201212-2223OC.
83. Gagnadoux F, Fleury B, Vielle B, *et al*. Titrated mandibular advancement versus positive airway pressure for sleep apnoea. Eur Respir J 2009;34(4):914–920. https://doi.org/10.1183/09031936.00148208.
84. Barnes M, McEvoy RD, Banks S, *et al*. Efficacy of positive airway pressure and oral appliance in mild to moderate

obstructive sleep apnea. Am J Respir Crit Care Med 2004;170 (6):656–664. https://doi.org/10.1164/rccm.200311-1571OC.

85. Ou YH, Colpani JT, Cheong CS, *et al*. Mandibular advancement vs CPAP for blood pressure reduction in patients with obstructive sleep apnea. J Am Coll Cardiol 2024;83(18):1760–1772. https://doi.org/10.1016/j.jacc.2024.03.359.
86. Belanche Monterde A, Zubizarreta-Macho A, Lobo Galindo AB, Albaladejo Martinez A, Montiel-Company JM. Mandibular advancement devices decrease systolic pressure during the day and night in patients with obstructive sleep apnea: a systematic review and meta-analysis. Sleep Breath 2024;28 (3):1037–1049.
87. Sutherland K, Cistulli PA. Oral appliance therapy for obstructive sleep apnoea: state of the art. J Clin Med 2019;8 (12):2121. https://doi.org/10.3390/jcm8122121.
88. Baslas V, Chand P, Jurel SK, *et al*. A pilot study to determine the effect of three months of oral appliance therapy using a mandibular advancement device on HbA1c in subjects with type 2 diabetes mellitus and obstructive sleep apnea. J Prosthodont 2019;28(3):271–275.
89. Gupta A, Tripathi A, Rai P, Sharma P, Yadav V, Kumar D. Effect of mandibular advancement splint on obstructive sleep apnea with insulin resistant diabetes. J Sleep Med 2020;30:171448. https://doi.org/10.13078/jsm.200001.
90. Chan AS, Lee RW, Srinivasan VK, Darendeliler MA, Grunstein RR, Cistulli PA. Nasopharyngoscopic evaluation of oral appliance therapy for obstructive sleep apnoea. Eur Respir J 2010;35(4):836–842.
91. Isono S, Tanaka A, Sho Y, Konno A, Nishino T. Advancement of the mandible improves velopharyngeal airway patency. J Appl Physiol 1995;79(6):2132–2138.
92. Van Gaver H, Op de Beeck S, Dieltjens M, *et al*. Functional imaging improves patient selection for mandibular advancement device treatment outcome in sleep-disordered breathing: a prospective study. J Clin Sleep Med 2022;18 (3):739–750.
93. Zhao M, Barber T, Cistulli P, Sutherland K, Rosengarten G. Computational fluid dynamics for the assessment of upper airway response to oral appliance treatment in obstructive sleep apnea. J Biomech 2013;46(1):142–150.
94. Caragiuli M, Mandolini M, Landi D, *et al*. A finite element analysis for evaluating mandibular advancement devices. J Biomech 2021;119:110298.
95. Gagnadoux F, Nguyen XL, Le Vaillant M, *et al*. Comparison of titrable thermoplastic versus custom-made mandibular advancement device for the treatment of obstructive sleep apnoea. Respir Med 2017;131:35–42.
96. Bosschieter PFN, Uniken Venema JAM, Vonk PE, *et al*. Equal effect of a noncustom vs a custom mandibular advancement device in treatment of obstructive sleep apnea. J Clin Sleep Med 2022;18(9):2155–2165.
97. Vanderveken OM, Devolder A, Marklund M, *et al*. Comparison of a custom-made and a thermoplastic oral appliance for the treatment of mild sleep apnea. Am J Respir Crit Care Med 2008;178(2):197–202.
98. Lettieri CJ, Paolino N, Eliasson AH, Shah AA, Holley AB. Comparison of adjustable and fixed oral appliances for the treatment of obstructive sleep apnea. J Clin Sleep Med 2011;7 (5):439–445.
99. Anitua E, Duran-Cantolla J, Almeida GZ, Alkhraisat MH. Minimizing the mandibular advancement in an oral appliance for the treatment of obstructive sleep apnea. Sleep Med 2017;34:226–231.
100. Levendowski DJ, Morgan T, Westbrook P. Initial evaluation of a titration appliance for temporary treatment of obstructive sleep apnea. J Sleep Disord Ther 2012;1(1):101. https://doi.org/10.4172/2167-0277.1000101.
101. Iftikhar IH, Cistulli PA, Jahrami H, *et al*. Comparative efficacy of mandibular advancement devices in obstructive sleep apnea: a network meta-analysis. Sleep Breath 2023;27 (4):1365–1381.
102. Mayoral Sanz P, Lagravere Vich M. Oral appliances for obstructive sleep apnea. Dent Clin N Am 2024;68(3):495–515.
103. Vezina JP, Blumen MB, Buchet I, Hausser-Hauw C, Chabolle F. Does propulsion mechanism influence the long-term side effects of oral appliances in the treatment of sleep-disordered breathing? Chest 2011;140(5):1184–1191.
104. Venema JA, Rosenmöller BR, De Vries N, *et al*. Mandibular advancement device design: a systematic review on outcomes in obstructive sleep apnea treatment. Sleep Med Rev 2021;60:101557.
105. Sangalli L, Yanez-Regonesi F, Fernandez-Vial D, Martinez-Porras A, Moreno-Hay I. Comparison of three mandibular advancement device designs in the management of obstructive sleep apnea: A retrospective study. J Dent Sleep Med 2021;9(3).
106. Caragiuli M, Candelari M, Zalunardo F, *et al*. Effects of oral appliances for obstructive sleep apnoea in reduced periodontium: a finite element analysis. Int Dent J 2024;74:1306–1316.
107. Zalunardo F, Bruno G, Caragiuli M, *et al*. Periodontal effects of two Somnodent oral devices for the treatment of OSA: a finite element study. Cranio 2024;42(4):400–410.
108. Barateau L, Baillieul S, Andrejak C, *et al*. Guidelines for the assessment and management of residual sleepiness in obstructive apnea-hypopnea syndrome: endorsed by the French Sleep Research and Medicine Society (SFRMS) and the French Speaking Society of Respiratory Diseases (SPLF). Respir Med Res 2024;86:101105. https://doi.org/10.1016/j.resmer.2024.101105.
109. Bamagoos AA, Eckert DJ, Sutherland K, Ngiam J, Cistulli PA. Dose-dependent effects of mandibular advancement on optimal positive airway pressure requirements in obstructive sleep apnoea. Sleep Breath 2020;24(3):961–969.
110. Ma Y, Yu M, Gao X. The effect of gradually increased mandibular advancement on the efficacy of an oral appliance in the treatment of obstructive sleep apnea. J Clin Sleep Med 2020;16(8):1369–1376.
111. Ma Y, Yu M, Gao X. Role of craniofacial phenotypes in the response to oral appliance therapy for obstructive sleep apnea. J Oral Rehabil 2023;50(4):308–317.
112. Stipa C, Incerti-Parenti S, Cameli M, Ippolito DR, Gracco A, Alessandri-Bonetti G. Antero-posterior mandibular excursion in obstructive sleep apnea patients treated with mandibular advancement device: a retrospective cohort study. Int J Environ Res Public Health 2023;20(4):3561. https://doi.org/10.3390/ijerph20043561.
113. Pitsis AJ, Darendeliler MA, Gotsopoulos H, Petocz P, Cistulli PA. Effect of vertical dimension on efficacy of oral appliance therapy in obstructive sleep apnea. Am J Respir Crit Care Med 2002;166(6):860–864.
114. Uniken Venema JAM, Doff MHJ, Joffe-Sokolova D, *et al*. Long-term obstructive sleep apnea therapy: a 10-year follow-up of mandibular advancement device and continuous positive airway pressure. J Clin Sleep Med 2020;16(3):353–359.
115. Pliska BT, Nam H, Chen H, Lowe AA, Almeida FR. Obstructive sleep apnea and mandibular advancement splints: occlusal effects and progression of changes associated with a decade of treatment. J Clin Sleep Med 2014;10(12):1285–1291.
116. Bartolucci ML, Bortolotti F, Raffaelli E, D'Anto V, Michelotti A, Alessandri Bonetti G. The effectiveness of different mandibular advancement amounts in OSA patients: a systematic review and meta-regression analysis. Sleep Breath 2016;20 (3):911–919.
117. Sakamoto Y, Furuhashi A, Komori E, *et al*. The most effective amount of forward movement for oral appliances for

obstructive sleep apnea: a systematic review. Int J Environ Res Public Health 2019;16(18):3248. https://doi.org/10.3390/ijerph16183248.

118. Huang W, Li C, Zou J, *et al*. Effects of the combination of novel eye mask sleep position therapy device and oral appliance on positional OSA: a multi-arm, parallel-group randomized controlled trial. Sleep Med 2023;102:52–63.
119. Joosten SA, Edwards BA, Wellman A, *et al*. The effect of body position on physiological factors that contribute to obstructive sleep apnea. Sleep 2015;38(9):1469–1478.
120. Milano FMS, Sutherland K, Milioli G, *et al*. Influence of vertical mouth opening on oral appliance treatment outcome in positional obstructive sleep apnea. J Dent Sleep Med 2018;5(1):17–23.
121. Dieltjens M, Braem MJ, Van de Heyning PH, Wouters K, Vanderveken OM. Prevalence and clinical significance of supine-dependent obstructive sleep apnea in patients using oral appliance therapy. J Clin Sleep Med 2014;10(9):959–964.
122. MacKay S, Carney AS, Catcheside PG, *et al*. Effect of multilevel upper airway surgery vs medical management on the apnea-hypopnea index and patient-reported daytime sleepiness among patients with moderate or severe obstructive sleep apnea: the SAMS randomized clinical trial. JAMA 2020;324(12):1168–1179. https://doi.org/10.1001/jama.2020.14265.
123. Almeida FR, Parker JA, Hodges JS, Lowe AA, Ferguson KA. Effect of a titration polysomnogram on treatment success with a mandibular repositioning appliance. J Clin Sleep Med 2009;5(3):198–204.
124. Pepin JL, Cistulli PA, Crespeigne E, *et al*. Mandibular jaw movement automated analysis for oral appliance monitoring in obstructive sleep apnea: a prospective cohort study. Ann Am Thorac Soc 2024;21(5):814–822.
125. Almeida FR, Lowe AA, Otsuka R, Fastlicht S, Farbood M, Tsuiki S. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: part 2. Study-model analysis. Am J Orthod Dentofacial Orthop 2006;129(2):205–213.
126. Marklund M, Stenlund H, Franklin KA. Mandibular advancement devices in 630 men and women with obstructive sleep apnea and snoring: tolerability and predictors of treatment success. Chest 2004;125(4):1270–1278.
127. van der Hoek LH, Rosenmöller B, van de Rijt LJM, de Vries R, Aarab G, Lobbezoo F. Factors associated with treatment adherence to mandibular advancement devices: a scoping review. Sleep Breath 2023;27(6):2527–2544. https://doi.org/10.1007/s11325-023-02862-9.
128. Sutherland K, Almeida FR, Kim T, *et al*. Treatment usage patterns of oral appliances for obstructive sleep apnea over the first 60 days: a cluster analysis. J Clin Sleep Med 2021;17(9):1785–1792.
129. Vanderveken OM, Dieltjens M, Wouters K, De Backer WA, Van de Heyning PH, Braem MJ. Objective measurement of compliance during oral appliance therapy for sleep-disordered breathing. Thorax 2013;68(1):91–96.
130. Dieltjens M, Braem MJ, Vroegop A, *et al*. Objectively measured vs self-reported compliance during oral appliance therapy for sleep-disordered breathing. Chest 2013;144(5):1495–1502. https://doi.org/10.1378/chest.13-0613.
131. Johal A, Hamoda MM, Almeida FR, Marklund M, Tallamraju H. The role of oral appliance therapy in obstructive sleep apnoea. Eur Respir Rev 2023;32(168):220257.
132. Yang J, Rosenmöller B, van Riet TCT, *et al*. Smart mandibular advancement devices for obstructive sleep apnea: a systematic literature review. Sleep Breath 2024;28(5):1879–1887. https://doi.org/10.1007/s11325-024-03068-3.
133. Nakamura S, Sato M, Mataki S, Kurosaki N, Hasegawa M. Subjective and objective assessments of short-term adverse effects induced by oral appliance therapy in obstructive sleep apnea: a preliminary study. J Med Dent Sci 2009;56(1):37–48.
134. Marklund M, Carlberg B, Forsgren L, Olsson T, Stenlund H, Franklin KA. Oral appliance therapy in patients with daytime sleepiness and snoring or mild to moderate sleep apnea: a randomized clinical trial. JAMA Intern Med 2015;175(8):1278–1285.
135. Aarab G, Lobbezoo F, Hamburger HL, Naeije M. Oral appliance therapy versus nasal continuous positive airway pressure in obstructive sleep apnea: a randomized, placebo-controlled trial. Respiration 2011;81(5):411–419.
136. Engleman HM, McDonald JP, Graham D, *et al*. Randomized crossover trial of two treatments for sleep apnea/hypopnea syndrome: continuous positive airway pressure and mandibular repositioning splint. Am J Respir Crit Care Med 2002;166(6):855–859. https://doi.org/10.1164/rccm.2109023.
137. Wang X, Gong X, Yu Z, Gao X, Zhao Y. Follow-up study of dental and skeletal changes in patients with obstructive sleep apnea and hypopnea syndrome with long-term treatment with the Silensor appliance. Am J Orthod Dentofacial Orthop 2015;147(5):559–565.
138. de Almeida FR, Lowe AA, Tsuiki S, *et al*. Long-term compliance and side effects of oral appliances used for the treatment of snoring and obstructive sleep apnea syndrome. J Clin Sleep Med 2005;1(2):143–152.
139. Araie T, Okuno K, Ono Minagi H, Sakai T. Dental and skeletal changes associated with long-term oral appliance use for obstructive sleep apnea: a systematic review and meta-analysis. Sleep Med Rev 2018;41:161–172.
140. Karadeniz C, Lee KWC, Lindsay D, Karadeniz EI, Flores-Mir C. Oral appliance-generated malocclusion traits during the long-term management of obstructive sleep apnea in adults. Angle Orthod 2022;92(2):255–264.
141. Doff MH, Finnema KJ, Hoekema A, Wijkstra PJ, de Bont LG, Stegenga B. Long-term oral appliance therapy in obstructive sleep apnea syndrome: a controlled study on dental side effects. Clin Oral Investig 2013;17(2):475–482.
142. Marklund M. Predictors of long-term orthodontic side effects from mandibular advancement devices in patients with snoring and obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2006;129(2):214–221.
143. Hammond RJ, Gotsopoulos H, Shen G, Petocz P, Cistulli PA, Darendeliler MA. A follow-up study of dental and skeletal changes associated with mandibular advancement splint use in obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2007;132(6):806–814.
144. Marklund M, Franklin KA. Treatment of elderly patients with snoring and obstructive sleep apnea using a mandibular advancement device. Sleep Breath 2015;19(1):403–405.
145. Heda P, Alalola B, Almeida FR, Kim H, Peres BU, Pliska BT. Long-term periodontal changes associated with oral appliance treatment of obstructive sleep apnea. J Clin Sleep Med 2021;17(10):2067–2074.
146. Minagi HO, Okuno K, Nohara K, Sakai T. Predictors of side effects with long-term oral appliance therapy for obstructive sleep apnea. J Clin Sleep Med 2018;14(1):119–125.
147. Martínez-Gomis J, Willaert E, Nogues L, Pascual M, Somoza M, Monasterio C. Five years of sleep apnea treatment with a mandibular advancement device. Side effects and technical complications. Angle Orthod 2010;80(1):30–36.
148. Robertson C, Herbison P, Harkness M. Dental and occlusal changes during mandibular advancement splint therapy in sleep disordered patients. Eur J Orthod 2003;25(4):371–376.
149. Zheng P, Chalidapongse P, Changsiripun C. Mandibular advancement devices used with morning occlusal guides for treating obstructive sleep apnea-changed incisor inclination and its associated factors. Sleep Breath 2023;27(5):2059–2067.
150. Alhozgi AI, Almeida FR. Dentoskeletal changes of long-term oral appliance treatment in patients with obstructive sleep

apnea: a systematic review and meta-analysis. J Prosthodont 2024. https://doi.org/10.1111/jopr.13946. Epub ahead of print.

151. Hou HM, Sam K, Hägg U, *et al.* Long-term dentofacial changes in Chinese obstructive sleep apnea patients after treatment with a mandibular advancement device. Angle Orthod 2006;76(3):432–440.
152. Ueda H, Almeida FR, Lowe AA, Ruse ND. Changes in occlusal contact area during oral appliance therapy assessed on study models. Angle Orthod 2008;78(5):866–872.
153. Gong X, Zhang J, Zhao Y, Gao X. Long-term therapeutic efficacy of oral appliances in treatment of obstructive sleep apnea-hypopnea syndrome. Angle Orthod 2013;83(4):653–658.
154. Uniken Venema JAM, Doff MHJ, Joffe-Sokolova DS, *et al.* Dental side effects of long-term obstructive sleep apnea therapy: a 10-year follow-up study. Clin Oral Investig 2020;24(9):3069–3076.
155. Rose EC, Staats R, Virchow C Jr, Jonas IE. Occlusal and skeletal effects of an oral appliance in the treatment of obstructive sleep apnea. Chest 2002;122(3):871–877.
156. Chen H, Lowe AA, de Almeida FR, Fleetham JA, Wang B. 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 2008;134(3):408–417.
157. Sheats RD, Schell TG, Blanton AO, *et al.* Management of side effects of oral appliance therapy for sleep-disordered breathing. J Dent Sleep Med 2017;4(4):111–125.
158. Balasubramaniam R, Pullinger A, Simmons M. Sleep medicine education at dental schools in Australia and New Zealand. J Dent Sleep Med 2014;1(1):9–16.

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QUESTIONNAIRE
B3(26) Part 1 of 2
Oral appliance therapy for snoring and obstructive sleep apnoea: a practical guide for clinical care

INSTRUCTIONS

- Read through the article and answer the multiple-choice questions provided below.
- **Select ONE correct answer unless otherwise indicated.**

Introduction and Methods

Question 1: What is the primary mechanism of oral appliance (OA) therapy as described in the introduction?

- **A:** It uses positive airway pressure to keep the airway open
- **B:** It stabilizes the tongue to prevent it from collapsing backward
- **C:** It is a removable appliance worn over the teeth to protrude (advance) the mandible during sleep
- **D:** It is a surgical implant that stimulates the hypoglossal nerve

Question 2: Untreated obstructive sleep apnoea (OSA) is associated with which range of comorbidities?

- **A:** Primarily dental and periodontal disorders
- **B:** Only psychological and mood-related disorders
- **C:** Cardiovascular, metabolic and neurocognitive comorbidities
- **D:** Musculoskeletal and autoimmune conditions

Question 3: Clinical decision-making for OA therapy should be guided by all the following, **except** ……..?

- **A:** The patient's specialist medical providers
- **B:** An individual patient's needs and preferences
- **C:** Affordability and accessibility to treatment
- **D:** A dentist's independent diagnosis without medical consultation

Discussion: Clinical Assessment and Screening

Question 4: What is the responsibility of the providing dentist after OA therapy has commenced?

- **A:** To conduct a new polysomnography (PSG) to confirm the medical diagnosis
- **B:** To independently manage all the patient's medical comorbidities
- **C:** Monitoring adherence to and efficacy of treatment
- **D:** To switch the patient to CPAP if the OA fails

Question 5: An OA may be constructed after a sleep physician-reported polysomnography (PSG) ……? [Choose both correct answers]

- **A:** Has diagnosed secondary snoring with mild to moderate OSA
- **B:** Has diagnosed severe OSA in a patient who is CPAP intolerant
- **C:** Has diagnosed severe OSA in a patient who is not compliant with CPAP
- **D:** None of the above

Question 6: Despite the availability of multiple treatment options for OSA, what remains the gold standard therapy?

- **A:** CPAP
- **B:** Oral appliance (OA) therapy
- **C:** Maxillomandibular advancement
- **D:** Bilateral hypoglossal nerve stimulation

Question 7: Which of the following is described as a subjective measure of daytime sleepiness often used in conjunction with screening questionnaires?

- **A:** The STOP-BANG questionnaire
- **B:** The Berlin Questionnaire
- **C:** The OSA50
- **D:** The Epworth Sleepiness Scale

Question 8: In general, what characteristic do screening questionnaires for OSA tend to have?

- **A:** Low sensitivity and low specificity
- **B:** Low sensitivity and high specificity
- **C:** High sensitivity and low specificity
- **D:** High sensitivity and high specificity

Patient Selection and Prediction of Response

Question 9: What is a critical requirement for dental suitability before constructing an OA?

- **A:** The patient must have a full dentition with no restorations
- **B:** There must be sufficient teeth or implant-retained restorations present in the maxillary and mandibular arches to provide adequate retention and comfort
- **C:** The patient must first undergo routine CBCT and MRI imaging
- **D:** The patient must have no history of temporomandibular disorders (TMD)

Question 10: Why is it important to assess and manage temporomandibular disorders (TMDs) in highly symptomatic patients before or during OA therapy?

- **A:** Because OA therapy is the primary treatment for TMDs
- **B:** Because TMDs are a known contraindication and prevent OA construction
- **C:** Because advanced imaging like MRI is always required for these patients
- **D:** Because oral and jaw pain and dysfunction are the most common causes of discontinuation of OA therapy

Question 11: Which of the following patient characteristics is associated with a stronger response to OA therapy?

- **A:** A high apnoea-hypopnoea index (AHI)
- **B:** Advancing age
- **C:** Supine predominant OSA
- **D:** REM-dependent OSA

Definitions of Success and Medical Comorbidities

Question 12: In the ORCADES prospective cohort study, what was the success rate for achieving a complete response, defined as an AHI < 5?

- **A:** 76.2%
- **B:** 63.5%
- **C:** 36%
- **D:** 93.3%

Question 13: TRUE or **FALSE:** According to one study mentioned, at 5-year follow-up, OA efficacy had decreased significantly, but self-reported compliance remained high at 93.3%.

- **A:** TRUE
- **B:** FALSE

Question 14: Meta-analyses comparing CPAP and OA confirm that CPAP performs significantly better than OA in which measures?

- **A:** Reduction of sleepiness and quality of life measures
- **B:** The reduction of the AHI and oxygen desaturation indices
- **C:** Patient adherence and long-term effectiveness
- **D:** Reduction of systolic blood pressure and cognitive improvement

Question 15: For which outcomes are CPAP and OA considered equivalent?

- **A:** The reduction of AHI and hypoxic burden
- **B:** The reduction of AHI and oxygen desaturation indices
- **C:** The reduction of sleepiness and quality of life measures such as cognition
- **D:** Overall treatment adherence and patient preference

THE END

QUESTIONNAIRE
B3(26) Part 2 of 2
Oral appliance therapy for snoring and obstructive sleep apnoea: a practical guide for clinical care

INSTRUCTIONS

- Read through the article and answer the multiple-choice questions provided below.
- **Some questions may have more than one correct answer;** in which case you must mark **all** the correct answers.

Appliance Design and Titration

Question 1: What type of OA design is recommended for optimal fit, retention, stability, comfort and efficacy?

- **A:** A temporary, non-titratable, mono-bloc design
- **B:** A custom-fabricated, titratable, bi-bloc design
- **C:** A non-custom, mono-bloc design set at 75% protrusion
- **D:** A bi-bloc design with an anterior traction connector

Question 2: What one of the following is **TRUE** regarding the use of temporary or non-titratable appliances?

- **A:** They are recommended as a low-cost alternative to CPAP
- **B:** They are recommended for predicting long-term OA success
- **C:** Their use is not recommended
- **D:** They are recommended for patients with mild OSA only

Question 3: Titration of the OA primarily involves making gradual advancements in which direction?

- **A:** The vertical position of the maxilla
- **B:** The lateral position of the mandible
- **C:** The vertical position of the mandible
- **D:** The anteroposterior position of the mandible

Question 4: What level of mandibular protrusion is noted to be effective in many cases, although higher levels may be needed for severe OSA?

- **A:** A level of 50% of maximal protrusion
- **B:** A level of 25% of maximal protrusion
- **C:** A level of 100% of maximal protrusion
- **D:** A minimal level of 10% of maximal protrusion

Adherence, Compliance and Side Effects

Question 5: Which one of the following is listed as a factor that **negatively** impacts treatment adherence to OA therapy?

- **A:** The duration of therapy
- **B:** Cost related to a custom fabricated appliance
- **C:** Diminishing effectiveness
- **D:** Stepwise titration of the OA

Question 6: Why are OA therapy and CPAP considered to have similar mean disease alleviation (MDA) measures?

- **A:** Because OA therapy has superior efficacy which offsets its inferior compliance
- **B:** The higher compliance to OA therapy offsets its inferior efficacy when compared to CPAP
- **C:** Because both treatments have similar efficacy and compliance rates
- **D:** Because CPAP has higher compliance which offsets its inferior efficacy

Question 7: Which of the following is a common, short-term side effect noted in the morning after overnight OA use?

- **A:** Permanent changes to the mandibular plane angle
- **B:** An abnormal occlusion (altered bite) can be present for up to 75 minutes
- **C:** The development of posterior open bites
- **D:** Proclination of the mandibular incisors

Question 8: Long-term OA therapy is associated with what specific dentoalveolar change?

- **A:** Proclination of maxillary incisors and retroclination of mandibular incisors
- **B:** An increase in overjet and in overbite
- **C:** Retroclination of the maxillary incisors and proclination of the mandibular incisors
- **D:** The development of anterior open bites and distal tipping of mandibular molars

Question 9: TRUE or FALSE: The article recommends the use of morning occlusal guides (morning repositioners) to effectively prevent long-term dentoskeletal side effects.

A: TRUE
B: FALSE

Multidisciplinary Team Approach and Conclusion

Question 10: Which of the following statements are **TRUE**?

A: Dentists trained in DSM are qualified to diagnose and treat sleep disorders independently
B: Dentists should request a PSG and prescribe OA therapy based on their own assessment
C: Dentists should not directly assess patients and request a PSG with the intent to diagnose sleep disordered breathing
D: Dentists have the primary role in diagnosing OSA, with physicians managing comorbidities

THE END

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ANSWER SHEET

B3(26)

Oral appliance therapy for snoring and obstructive sleep apnoea: a Practical Guide for Clinical Care

***Time spent on activity hour min**

PART 1												PART 2											
	A	B	C	D	E		A	B	C	D	E		A	B	C	D	E		A	B	C	D	E
1						8						1						6					
2						9						2						7					
3						10						3						8					
4						11						4						9					
5						12						5						10					
6						13																	
7						14																	
						15																	

How relevant was the content of this activity to your scope of practice?			
Not at all	Somewhat	Mostly	Extremely

RETURN ANSWER SHEET TO

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You will receive a confirmation of receipt SMS within 12-24hours, if not received please send again.

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