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Clinical guidelines for oral appliance therapy in the treatment of snoring and obstructive sleep apnoea

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ABSTRACT

The purpose of this review is to provide guidelines for the use of oral appliances (OAs) for the treatment of snoring and obstructive sleep apnoea (OSA) in Australia. A review of the scientific literature up to June 2012 regarding the clinical use of OAs in the treatment of snoring and OSA was undertaken by a dental and medical sleep specialists team consisting of respiratory sleep physicians, an otolaryngologist, orthodontist, oral and maxillofacial surgeon and an oral medicine specialist. The recommendations are based on the most recent evidence from studies obtained from peer reviewed literature. Oral appliances can be an effective therapeutic option for the treatment of snoring and OSA across a broad range of disease severity. However, the response to therapy is variable. While a significant proportion of subjects have a near complete control of the apnoea and snoring when using an OA, a significant proportion do not respond, and others show a partial response. Measurements of baseline and treatment success should ideally be undertaken. A coordinated team approach between medical practitioner and dentist should be fostered to enhance treatment outcomes. Ongoing patient follow-up to monitor treatment efficacy, OA comfort and side effects are cardinal to long-term treatment success and OA compliance.

Keywords: Mandibular advancement splint, obstructive sleep apnoea, oral appliance, review, snoring.

Abbreviations and acronyms: AASM = American Academy of Sleep Medicine; ADA = Australian Dental Association; AHI = apnoea–hypopnoea index; ASA = Australasian Sleep Association; BMI = body mass index; CBCT = cone beam computed tomography; CT = computed tomography; ESS = Epworth Sleepiness Scale; MAS = mandibular advancement splints; OA = oral appliances; OSA = obstructive sleep apnoea; PSG = polysomnogram; SDB = sleep disordered breathing; TMD = temporomandibular disorder; TMJ = temporomandibular joint; TRD = tongue retaining device; TSD = tongue stabilizing device; UARS = upper airway resistance syndrome; UPPP = uvulopalatopharyngoplasty.

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INTRODUCTION

Obstructive sleep apnoea (OSA) is a breathing disorder during sleep that is characterized by snoring and recurrent collapse of the pharyngeal airway during sleep, resulting in a partial reduction (hypopnoea) or complete cessation (apnoea) of airflow despite ongoing breathing effort.[1] OSA is at one end of a spectrum of disorders encompassed in the term 'sleep disordered breathing' (SDB). There is a continuum from mild intermittent snoring at one end of the spectrum, through heavy obstructed snoring and high upper airway resistance, heavy snoring and runs of partial and complete obstruction, through to repetitive obstructive apnoea occurring throughout the entirety of sleep. Chronic snoring occurs in up to 30% of adult subjects, and some degree of obstructive apnoea and hypopnoea occurs in 9% of males and around 5% of females. More strictly defined OSA (which is the combination of confirmed apnoea on sleep study and symptoms) has been found to affect 2% to 4% in males and approximately half that in females.[2,3] While the current accepted measure of severity is based on the apnoea–hypopnoea index (AHI), or the respiratory disturbance index (RDI), and or the number and severity of oxyhaemoglobin desaturations per hour of sleep, none of these indices have a clear relationship to symptoms. As an approximate guide, an AHI between 5 and 15 per hour is mild SDB, between 15 and 30 per hour is 'moderate' and above

30 is 'severe'. There are many associated symptoms, the most common of which is excessive daytime sleepiness. Until recently, the main indication for treatment was in response to the various symptoms. However, with the numerous publications that demonstrate a range of adverse outcomes, especially chronic vascular complications and increased cardiovascular mortality, there has been a shift towards treating SDB to prevent long-term adverse outcomes.

SDB represents a significant public health burden in Australia with the prevalence of OSA in men found to range from 10% to 26%.[4] These disruptions to breathing are commonly associated with intermittent blood gas disturbances (hypoxaemia and hypercapnia), sleep fragmentation and surges in sympathetic activation.[5,6] Mounting epidemiological and scientific evidence have associated OSA, and to a lesser degree snoring, to a wide variety of adverse health outcomes.[7] OSA has been linked to systemic hypertension, myocardial infarction, stroke, congestive heart failure, atrial fibrillation, carotid artery atherosclerosis, diabetes, excessive daytime sleepiness, impaired quality of life, and increased mortality.[8–10] Snoring is a major symptom of OSA and affects 40% of males and 20% of females.[4] It is often the chief reason why patients seek treatment and occurs as a result of vibration of the pharyngeal airway structures. It has been proposed as an independent contributor to the development of carotid atherosclerosis.[10,11]

Over the last decade, oral appliances (OA) have gained increasing acceptance as a viable treatment alternative to CPAP therapy for the treatment of OSA. Mandibular advancement splints (MAS) are the predominant type of OA used in clinical practice. They attach to both the upper and lower dental arches to advance and retain the mandible in a forward position, aiming to stretch the soft tissues and to prevent airway obstruction. Although the precise mechanism of airway stabilization through mandibular advancement is not clearly understood, the predominant effect of MAS is the enlargement of velopharyngeal airway calibre in the lateral dimension.[12,13] Increased upper airway patency and reduced collapsibility of airway structure may also be mediated by increasing upper airway neuromuscular tone. The stimulation of upper airway dilator muscles, in particular increased genioglossus muscle activity with MAS therapy has been demonstrated and has been proposed as an additional mechanism of upper airway stabilization.[14,15]

There have been numerous randomized controlled trials evaluating OAs to either placebo or CPAP treatment.[16,17] Collectively, they have established the objective efficacy of these devices for the treatment of OSA across a broad range of OSA severity.[18] In Australia, there exists a brief scope of practice definition by the Dental Board of Australia[19] and OA protocols were established by the Australasian Sleep Association (ASA) in 2010.[20] Very recently, the Australian Dental Association (ADA) issued a policy statement 6.7 regarding the use of OA therapy for SDB.[21]

In recognition of the important collaborative role between dentists and physicians in the use of OAs and the emerging need to regulate these practices, this position paper provides recommendations for the use of OAs in adults for the treatment of snoring and OSA based on the published literature up to June 2012. Although there exists some studies on the use of OAs[22,23] and rapid maxillary expanders[24–26] in the orthodontic management of paediatric SDB, the limited scientific evidence does not facilitate the establishment of guidelines at this stage.

METHODS

A dental and medical sleep specialist team consisting of respiratory sleep physicians, an otolaryngologist, oral and maxillofacial surgeon, orthodontist and an oral medicine specialist developed these guidelines. A review of the scientific literature regarding the clinical use of OAs in the treatment of snoring and OSA was undertaken. The detailed recommendations are based on the current evidence from studies obtained from peer-reviewed literature. All members of the team completed conflict of interest statements.

The recommendations of this position paper define the standards of clinical practice in the use of OAs for the treatment of snoring and OSA in Australia. They should address the needs of the majority of patients undergoing OA therapy. Ultimately, the decision to peruse OA therapy should be guided by the physician, and should take into account the specific and individualized needs of each patient, the availability of diagnostic tools, accessibility and affordability of the treatment options proposed.

It is anticipated that these recommendations will impact on the standards of care, clinical protocols, treatment outcomes and health care costs of OA therapy. These current recommendations reflect the most recent publications up to June 2012 and will be reviewed and revised as the state of understanding and knowledge of OA therapy advances.

DISCUSSION

OSA is a sleep disorder characterized by snoring and recurrent partial or complete collapse of the pharyngeal airway, resulting in nocturnal oxygen desaturation, sleep fragmentation and increased sympathetic nerve activation during sleep. This common condition may result in excessive daytime sleepiness, neurocognitive impairment with long-term adverse

cardiovascular outcomes.[27] As body weight is a key factor in the development of snoring and SDB in many subjects, a first approach to treatment is weight reduction supervised by a sleep physician/medical practitioner. However, this typically takes considerable time, and is often very difficult to achieve.

It is generally agreed that OSA is part of a broad spectrum of SDB, with the same pathophysiological disorder.[28] The treatment required to treat this condition requires an appreciation of the topodiagnosis of the collapse site(s) in the upper airway. Fujita initially classified the patient pool into three types – retrovelar, retrolingual or combined collapsed sites.[29] However, research with sleep endoscopy, CT and cine MRI have demonstrated that this classification is over simplified, and the site of collapse changes between wakefulness and sleep, at different sleep stages, in different body positions, after surgical intervention and at different ages of the one individual. It is increasingly recognized that correcting obstruction at any area in the upper airway will positively contribute to the better management of the patient with SDB, allowing both apparative devices such as CPAP and OAs to be delivered more successfully.

The soft tissue that is greatly assisted in using OAs is the tongue base. Surgical techniques preventing the posterior collapse of the tongue musculature started with the hyoid suspension myotomy procedure by Riley and colleagues in 1986.[30] These investigators recognized that in sleep apnoeics, the hyoid bone was positioned lower than in healthy subjects on cephalometric studies. Many modifications have occurred since, resulting in the hyoid suspension procedure which in essence is a hyoidthyroidpexia. Intrinsic reduction of the tongue size is also available through the use of lingual tonsil surgery, radiofrequency or plasma frequency ablation[31] or partial glossectomy.[32] A systematic review of several studies found 139 cases with varying success rates between 20% to 80%, with an overall success rate of 52%.[33] However, these procedures bring with the surgery significant morbidities such as discomfort and haemorrhage, and are generally reserved for very severe patients. The concept of multilevel procedure for OSA was presented by Waite *et al.* in 1989.[34] They combined surgery at the level of the nose, the palate, transoral tongue surgery, genioglossus advancement and a maxillomandibular advancement osteotomy. The multilevel surgical approach has evolved as experience by multiple institutions realized the overall success rates of their philosophy. Currently, a staged approach is now being recognized for the severe OSA patient. Refinements in the type of soft palate surgery being performed, the use of intrinsic tongue base surgery rather than external approach tongue suspension, along with hyoid suspension have achieved a success rate of at least 60%, whilst minimizing patient discomfort and complication rates.[35]

In contrast to the wide variability in treatment responses achieved with surgical techniques described above, CPAP is highly efficacious and currently the reference standard of treatment in preventing airway collapse. Although early CPAP systems and masks were cumbersome and intrusive, newer systems are light weight, less noisy and easier to use. Nonetheless many subjects find the system difficult to tolerate.[36,37] A passive wearable device such as an OA has obvious advantages.

Although CPAP is almost completely effective in controlling apnoea and snoring, long-term compliance can be a major issue. Objective monitoring of CPAP usage in one study found that 46% to 83% of patients with OSA were non-adherent to treatment, defined as greater than four hours of nightly use.[37] In contrast, covert compliance with a thermo-sensitive monitor embedded in an OA demonstrated a mean of 6.8 hours nightly use with a range of 5.6 to 7.5 hours per night.[38] It can be argued that more regular use of an OA that is less effective might be preferable to CPAP used for shorter times.

It is not surprising that many patients prefer OAs instead of CPAP due to their portability, ease of use and comfort. OAs are recognized as a viable and effective treatment alternative with robust validation in randomized controlled trials.[16,17,39] Although they are currently indicated for mild and moderate OSA and in severe OSA adult patients who are intolerant or fail a trial of CPAP therapy,[40] there is increasing evidence of the potential role of OAs in severe OSA patients.[17,41–44]

However, in children there are still limited studies for the use of removable OAs[22,23] and orthodontic rapid maxillary expanders for the treatment of snoring and OSA.[24–26,45] Although remarkable reductions in AHI[22,25,26] and nasal resistance[24] have been reported, collectively the level of evidence is still weak due to small sample sizes and the lack of randomized controlled trials. As such, guidelines for the use of OAs in the paediatric field are not within the scope of this publication.

Currently, an in-laboratory polysomnogram (PSG) is the standard method used to diagnose OSA, although portable at-home sleep studies are gaining widespread use. Differentiation of the severity of OSA is based on the AHI, which is the average number of apnoeic and hypopneic events per hour of sleep. Using a robust definition of an AHI <5 events/hour as a measure of treatment success, approximately 35–40% of patients are treated successfully with a further 25% having partial response to OA therapy.[17,41] Using a more liberal criteria of AHI <10 events/hour, 54% of subjects can be defined as successfully treated.[46]

However, some 35–40% of treated subjects may not respond favourably (defined as less than 50% reduction in AHI) with some individuals worsening in OSA symptoms despite OA therapy.[47,48] Overall, two-thirds of patients experience clinical improvement in symptoms with OA therapy.[46,49]

It is very clear from the many studies that the response to OA use is non-uniform, i.e. there are responders and non-responders, so it is misleading to pool the data. In contrast, CPAP virtually attains a 100% success rate during any one night of testing. Typically, CPAP failure only occurs in those with completely blocked nasal airways. However, in practice, nasal obstruction on long-term CPAP and factors such as mouth leaks can make CPAP use difficult for many patients.

When OA devices are effective, there are improvements of polysomnographic indices such as AHI and oxygen desaturation,[50,51] as well as improvements in daytime sleepiness as measured with the Epworth Sleepiness Scale (ESS) and modest reductions in blood pressure (2–4 mm Hg).[16,47,52,53] Limited studies have also reported improvements in some neurocognitive assessments[16,54] with one showing comparable results between OAs and CPAP in simulated driving performance.[55] There are also few studies that compare OAs to upper airway surgical intervention. However, Walker-Engstrom *et al*. compared MAS to uvulopalatopharyngoplasty (UPPP) in a randomized four-year study[56] and found greater success (AHI <10 events per hour) with MAS therapy than UPPP (63% vs 33%, $p < 0.05$).

In support of these findings, Millman and colleagues treated 18 patients who had failed UPPP with an adjustable OA and found the OA to be effective in the control OSA after unsuccessful UPPP.[57]

Oral appliance candidates

To be suitable for OA therapy, candidates generally require sufficient healthy teeth and alveolar ridge to retain the device, the absence of temporomandibular disorders (TMD), and adequate protrusive jaw function. Nevertheless, the lack of sufficient teeth may not be a contraindication as the use of a dental implant-retained MAS[58] and mini-implants has been reported in edentulous and partially dentate patients.[59,60] Moreover, tongue retaining devices (TRDs) and tongue stabilizing devices (TSDs) which protrude and hold the tongue forward using suction have also been proposed as a treatment alternative for edentulous patients. When compared to MAS, a similar efficacy in reducing the AHI was found with a TSD.[61] Furthermore, different patterns and magnitude of change were found to occur with MRI investigation with a TSD.[62]

The periodontal status of OSA patients warrants consideration. Periodontal disease may pose as a contraindication to OA therapy.[63] A pilot study found an association between OSA and periodontitis with significant correlations between periodontal clinical attachment and total sleep time.[64] Likewise, a comprehensive temporomandibular joint (TMJ) assessment is a prerequisite. The prevalence of TMD in OSA subjects have been reported to range from 2% to 52%,[63,65] with 50% of subjects complaining of myofascial pain associated with and without limited mouth opening and arthralgia.[65] In another study, Smith and colleagues found a 28.4% OSA prevalence rate in 53 TMD patients and highlighted the need for PSG evaluation in TMD patients complaining of sleep disturbances.[66]

Overall, some 16% of patients require dental or periodontal care before the use of MAS and the rate of contraindications for OA use has been found to be 34% of OSA subjects.[63]

Patient specific factors

The predictive factors for success with OA therapy is the focus of ongoing research. Better treatment responses have been found in younger patients,[46,67,68] patients with smaller neck circumferences,[41] females[67] and supine-dependent OSA subjects.[67,69] Poorer responses have been found with increases in body mass index (BMI); however, a 10-year study did not find lower BMI to be related to long-term OA success.[70] Although it is generally believed that less severe OSA subjects respond better to OA therapy,[46,67] this has not been systematically evaluated, for as a general rule subjects with severe OSA are not treated with OAs. It is notable that in the few reports published, remarkable improvements in severe OSA subjects have been documented.[17,41–44] A reason why OAs are not recommended in patients with severe OSA is the concern that failed treatment, or partial treatment, may lead to respiratory failure. However, there could be a place for using an OA in such patients as an initial treatment provided they are fully monitored and supervised by attending clinical staff.

Craniofacial morphology appears to play an influencing role in OA treatment outcomes. Cephalometric variables associated with better treatment responses have included a longer maxilla, shorter facial heights and soft palate, reduced overjet and shorter distances between mandibular plane and hyoid bone.[41,71,72] Ethnicity may also contribute to treatment outcomes.[73] Likewise, upper airway physiology may be a key determinant of OA success as OAs predominantly enlarge the velopharyngeal airway with relatively minor changes in the oropharynx and hypopharynx.[12]

Nevertheless, patients who have upper airway collapse primarily in the oropharyngeal region have been found to respond better to OA therapy.[74] Moreover, lower nasal resistance has also been shown to be a predictor of OA success.[75] Despite these various findings, there is still no easy way to predict those individuals who will have a good response, and perhaps more importantly, to predict those in whom the OA device will fail.

Overall, despite the recent technological advances in diagnostic imaging[62,72] and techniques such as nasopharyngoscopy,[12] spirometry[76] and craniofacial photographic analysis[77,78] for the prediction of treatment outcomes, a reliable validated method of predicting MAS responders in the clinical field is yet to be established. In addition, OA type and titration protocols are thought to impact on treatment outcomes.[49]

Oral appliance device type

Presently, there exist a multitude of OA designs available on the market. These OAs vary in coupling design, mode of fabrication and activation, titration capability, degree of vertical opening and lateral jaw movement. These devices can be one-piece (monobloc) or two-pieces (bi-bloc) in design and either custom-made or prefabricated. However, two recent reviews could not identify a specific OA design that was most effective in improving polysomnographic indices due to the absence of a universal definition of treatment success.[79,80] More recently, a retrospective review of 805 consecutive patients treated with either a titratable or non-titratable OA reported greater reductions in obstructive events with titratable OAs, especially with moderate-severe OSA subjects.[68] Obstructive events were reduced to <5/h in 56.8% with the titratable OA compared to 47.0% with the non-titratable OA ($p = 0.02$). Non-titratable OAs were found to be effective in mild OSA cases only and less successful in patients with higher AHI scores.

Thermoplastic (boil and bite) OAs have been proposed as a temporary device to be used when the regular device is being repaired.[81] Initially it was proposed as a feasible, low cost, screening device for the treatment of snoring and OSA.[82,83] A later randomized controlled cross-over study found that AHI was only reduced with the customized device which was preferred by 82% of subjects.[84] In this study, 33% of patients cited poor overnight retention as the main reason for compliance failure whereas a later study by Tsuda *et al*. recorded 77.8% of non-users of thermoplastic OAs were non-compliant in the first three months complaining of poor comfort and fit of the device.[85] It is now generally accepted that custom-fabricated OAs are better tolerated and, in particular, remain in place during sleep and are more efficacious compared to a thermoplastic OA.[84,86]

Oral appliance titration

Titratable OAs are usually activated by means of a screw, elastomeric chain or hook attachment that incrementally advances the mandible. The degree of mandibular advancement proposed is highly variable among clinicians; however, it usually ranges from 50% to 80% of maximum protrusion.[43,87] On average, a baseline advancement of 78% of maximum protrusion has been shown to be a therapeutically efficient and comfortable mandibular position,[41] which should be followed by an acclimatization period. Typically, following an acclimatization phase of 2–4 weeks, the device is gradually advanced until the patient or bed partner subjectively reports resolution of OSA symptoms. Occasionally jaw and teeth discomfort with mandibular protrusion may restrict further titration despite residual symptoms.

The use of subjective feedback such as a cessation of snoring or perceived resolution of OSA symptoms is typically employed to finalize titration. However, this can lead to residual OSA.[88] A major problem in the use of OAs is that alarmingly, objective quantification of the AHI, and in particular snoring, is not routinely performed in clinical practice. This fact is probably one of the reasons that OAs are often not considered by many physicians and is in contrast to CPAP therapy where nightly measurements and ready availability of CPAP downloads allow a clinician to verify the effectiveness of treatment. There is currently no such feedback either to physician or patients apart from spouse/partner reporting, which is notoriously inadequate.

Although there does not appear to be a direct correlation between the amount of mandibular advancement and therapeutic response,[41,89] sequential PSG studies have been shown to facilitate OA titration, and there appears to be a dose-dependent improvement of the AHI.[87,90] Similar dose-dependent improvements in cross-sectional airway volume[91] and oxygen desaturation events have also been reported.[92] Nonetheless, the degree of mandibular advancement appears to be one of many key variables affecting OA treatment success, and it relies on the amount of change in the upper airway patency in response to mandibular advancement.[93] More recently, remarkable treatment success (defined as AHI less or equal to 10), from 65% to 95%, highlighted the benefit of further mandibular advancement during a titration PSG.[88]

Studies evaluating the use of an OA with a hydraulic or motorized advancement mechanism have been reported.[54,94–96] In these scenarios, the patient uses the OA during sleep and the degree of mandibular

protrusion is remotely controlled such that the target amount of protrusion can be determined without waking the patient. This single night titration protocol has the potential to distinguish MAS responders from non-responders whilst determining the optimal level of protrusion required.[95,96] More recently, the use of ambulatory monitoring devices with limited channels has been proposed as an alternative method to determine the therapeutic titration dose.[50,97] However, in moderate and severe OSA, a follow-up PSG after OA titration has been recommended as the desired standard of care by the American Academy of Sleep Medicine (AASM).[40] In practice, this protocol is rarely adhered to due to financial constraints and/or sleep laboratory availability.

Side effects

OAs rely on teeth and alveolar ridges for retention to advance the mandible forward. The dynamics of mandibular repositioning invariably exerts reciprocal forces that are distributed throughout the dentoalveolar and skeletal structures. As the degree of mandibular advancement has been shown to improve the AHI in a dose-dependent manner,[88,90] some authors have proposed that greater titration may lead to greater side effects[50] and hence advocate lesser mandibular advancement.[43] Short-term side effects are generally considered to be minor and transient in nature. These include excessive salivation, dry mouth, teeth discomfort, gingiva irritation, masticatory muscle tenderness and TMJ discomfort. These symptoms generally occur in the first few days or weeks during adaption to the OA.[46,49] More severe and persistent side effects during the initial and later treatment stages include arthralgia, myofascial pain, teeth pain, and occlusal changes. These adverse effects impact on OA compliance and should be addressed immediately.

Dental and skeletal changes

As early as six months of OA use, changes in facial height, teeth and jaw positions have been noted to occur.[98] Longer term five-year studies reveal increases in facial height, occlusal changes, incisor inclination and molar positional changes.[99–101] In a recent study, Perez and co-workers found an increase in the incidence of posterior open bite in 167 patients by an average of 6.1% per visit while 5.8% of patients demonstrated a posterior open bite after 118 days, increasing to 17.9% after 413 days.[102] Nevertheless, the degree of detrimental impact to the dentition and facial aesthetics may be based on the initial skeletal and dental morphology. Skeletal types with high angled vertical growth tendencies appear to be more at risk whereas individuals with greater overjet/overbite may display more favourable changes.[99,101] Interestingly, a recent pilot study evaluating an orthodontic oral appliance specifically designed to counteract the reciprocating forces generated by OA wear reported modest increases in overjet (+0.4 mm).[103] The amount of bite change may also depend on the importance of the intercuspidation, the amount of overbite locking the bite and duration and the frequency of use. Overall, longer term changes in occlusion,[104] overbite/overjet and mandibular length may occur but the effects appear to be negligible.[105] Even when unfavourable, these effects are well tolerated and accepted by patients[102] and do not typically require the cessation of OA therapy.

Temporomandibular disorders

There have been numerous studies that have collectively reported temporomandibular disorders (TMD) as a result of OA use.[46,91,103,107] However, in an MRI study, translation of the condyle with OA therapy was equal or less than that observed with maximum jaw opening with no significant TMJ morphological alterations observed.[91] Nonetheless, when compared with CPAP therapy, more subjects were found to experience TMD related pain in the initial stages of treatment.[106] This finding was transient in nature and was not found to be a contraindication to OA use. In support of this finding, a recent questionnaire based evaluation of TMD in 167 patients over 413 days found that although OAs may lead to the development of TMD in a small number of patients, the effects are transient and appear to decrease with time.[102] Overall, there appears to be a decrease in intensity of TMD pain with continued OA use.[90,100,102,106,107] To date, only two studies have addressed the issue of TMD related pain and occlusal function associated with OA use.[108,109] Both these studies advocated the use of mandibular jaw exercises to manage side effects. Ueda and colleagues described the effect on two jaw exercises (jig exercise and stretching exercises) and found significant increases in bite force and occlusal contacts.[108] Cunali and colleagues found significant improvements in sleep quality and life quality with significant reductions in jaw pain in subjects performing jaw exercises compared to neck exercises, thereby increasing OA compliance.[109]

Oral appliance compliance

In contrast to CPAP adherence monitoring, there are currently no commercially available monitoring devices to objectively measure MAS adherence. At 30 months, 56–68% of subjects were still OA compliant[46] whereas a 10-year questionnaire survey of OSA patients found a 68% compliance rate with 47% of subjects wearing the OA every night.[70] In support of

these findings, an 11-year follow-up of 630 OSA patients found 76% compliance with OA use.[67] Older data from Lowe *et al.* utilized a thermo-sensor embedded in a thermoplastic OA and found a mean of 6.8 hours nightly use with a range of 5.6 to 7.5 hours per night.[38]

The extent of compliance is a delicate balance between perceived benefit and the side effects encountered. The lack of subjective perceived benefit and OSA symptoms recurrence[46] together with side effects such as jaw pain and dysfunction with OA therapy may ultimately result in poor OA adherence or discontinuation.[63,87,90] The erosion of OA compliance has also been linked to OA wear and tear[110] and weight gain.[67] Although current studies available have failed to identify one OA design that is most effective in reducing polysomnographic indices,[79,80] it has been suggested that OAs which allow lateral jaw movement with reduced vertical opening[48] are more comfortable and hence may increase OA compliance. However, as there still exists considerable controversy regarding OA design on outcomes and adherence, the final choice of OA design requires careful consideration based on a patient's individualized needs and treatments goals.

Recommendations

The AASM first published recommendations for the use of OAs in the treatment of snoring and OSA in 1995. A decade later, new practice parameters were introduced and recommendations were updated.[40] Published in 2006, these clinical parameters of the AASM state that MAS devices are indicated as the first line treatment for patients with mild to moderate OSA who prefer an OA over CPAP, who do not respond to CPAP or are inappropriate candidates for CPAP. In 2007, the German Society of Dental Sleep Medicine issued a position paper on the use of MAS.[111] In Australia, a brief regulatory definition by the Dental Board[19] and protocols for the dental management of SDB were established by the ASA in July 2010.[20] Very recently in April 2012, the ADA issued a policy statement 6.7 regarding the use of dental appliances to treat SDB.[21] Based on scientific appraisal of the current literature, international and national policies, the following are current recommendations for the clinical use of OAs in the treatment of snoring and OSA.

Diagnosis

The diagnosis of SDB must be established prior to the commencement of treatment with OAs so as to identify those patients who may be at risk of the complications of OSA and to provide a baseline to assess the efficacy of OA therapy.

- The diagnosis of SDB can only be made by a physician with training in sleep medicine with the aid of a sleep study. The diagnostic criteria of OSA have been described in detail and include the clinical signs and symptoms of OSA and findings identified by PSG.[40] The degree of SDB severity and site(s) of obstruction should be established to enable appropriate treatment proposal. A multidisciplinary approach should be encouraged so that treatment options are made available to patients in a timely manner.
- The use of cephalometric evaluation, cone beam computed tomography (CBCT) and computed tomography (CT) diagnostic imaging, although not routinely indicated have been demonstrated to be useful in diagnostic and morphometric analysis of hard and soft tissue structures. If necessary, advanced imaging may be ordered in select cases for further evaluation of certain structures such as the TMJs prior to OA therapy. These examinations should be interpreted by a radiologist when required.

The role of the dentist

The principal role of the dentist in the management of SDB is to screen for potential SDB and provide OA therapy where indicated but not to diagnose SDB. This key role may include the following:

- Recognizing the signs and symptoms of SDB.
- Referral to a physician with training in sleep medicine for assessment, where appropriate.
- Referral to an ENT surgeon, if suspecting the need for assessing other airway lesions which would contribute to SDB, e.g. chronic nasal obstruction, adenotonsillar hypertrophy.
- Referral to an orthodontist, oral and maxillofacial surgeon or oral medicine specialist, when appropriate for specialist assessment.
- Delivery of OAs where appropriate (refer to section 'treatment indications').
- Monitor OA treatment efficacy (refer to section 'treatment aims/objectives').
- Manage adverse side effects of OA therapy as they develop.

Appliance type/fitting

Oral appliance therapy should only be provided by the dentist. Ongoing care of patients treated with OAs should be managed by dentists with training and experience in the field of dental sleep medicine. The key focus of this care includes coordination and written correspondence with the referring physician with regards to the treatment plan and long-term follow-up.

The use of non-customized, prefabricated 'boil and bite' type devices as a screening tool to identify potential candidates for OA therapy cannot be recommended. As the lack of retention in these devices may lead to poor compliance and treatment efficacy, they are not indicated as a therapeutic option for the treatment of snoring and SDB.

The decision on the appropriate type of OA design should be made by the dentist, and should take into account the specific and individualized needs of each patient, the degree of OSA severity, the availability of diagnostic tools, accessibility and affordability of the treatment options proposed. Although a titratable OA is preferred, non-titratable OA may be fitted by the dentist based on the above-mentioned factors.

Informed consent must be obtained prior to the delivery of OAs. The benefits, risks, short and long-term side effects and complications associated with OA therapy should be explained verbally and followed up in writing. The consent form should clearly indicate the potential and probable side effects of using OAs, the appliance longevity and treatment follow-up procedures.

Treatment indications

OAs are indicated as treatment for the following clinical presentations:

- Primary snoring.
- Upper airway resistance syndrome (UARS).
- Mild to moderate OSA (AHI less than 30 events/hr) patients who prefer OAs to CPAP, who are not appropriate CPAP candidates, or who fail attempts with CPAP and other alternative treatment modalities such as weight loss or positional therapy.
- In cases of severe OSA (AHI >30 events/hr), an initial trial of nasal CPAP should be undertaken prior to OA therapy as greater effectiveness has been demonstrated with CPAP therapy in comparison to OA therapy.
- In cases of obesity (BMI >30 kg/m^2), an initial trial of behavioural modification therapy including weight loss, dietary modification and exercise should be undertaken prior to OA therapy as greater effectiveness has been demonstrated in non-obese patients.

Treatment contraindications

OAs are contraindicated as treatment for the following clinical presentations:

- In cases of multiple comorbidities such as heart failure, respiratory failure, where there is the possibility of central apnoea and/or central hypoventilation.
- In cases of severe periodontal disease, whereby there is significant risk for teeth mobility and loss.
- In cases of severe TMD, especially when the pain and dysfunction is aggravated with mandibular protrusion.
- In cases of inadequate retention for the OAs such as the lack of sound teeth, loose denture or where the augmentation of retention/anchorage of OAs by dental implants, mini-implants or the splinting of teeth is not suitable.
- In cases of severe gag reflex.
- In cases of poor coordination or dexterity as required for placement and removal of OAs.

Treatment aims/objectives

For patients with primary snoring or UARS, the treatment objective is to reduce or eliminate snoring to a subjectively acceptable level. For patients with OSA, the treatment goal should include the resolution of the clinical signs and symptoms of OSA with normalization of the AHI and oxyhaemoglobin saturation.

Follow-up

Although a follow-up PSG is not indicated in primary snorers, a clinical review to assess the subjective efficacy of OA therapy should be undertaken. Assessment by the physician with training in sleep medicine with an unattended cardiorespiratory (Type 3) or Type 4 diagnostic device may be used to help assess the effect of OAs on snoring.

To monitor the therapeutic efficacy of OA therapy and to distinguish the placebo effect, patients with OSA should undergo an overnight PSG or an unattended cardiorespiratory (Type 3) sleep study with the OA *in situ* with the physician. As the degree of mandibular advancement has been shown to affect the final treatment outcome and overall OA success, the use of unattended cardiorespiratory (Type 3) or Type 4 diagnostic device may be used to help define the optimal target protrusion of the mandible. A dentist trained in the use of portable monitoring devices may accurately assess but not diagnose the results of OA titration. A final follow-up polysomnographic or an unattended cardiorespiratory (Type 3) sleep study with the OA *in situ* should be performed with the physician once final adjustments and optimal target advancement has been determined based on the improvement of signs and symptoms.

Patients with OSA treated with OAs should be reviewed routinely by the treating dentist (refer to section 1.3.1). Once a comfortable fit and optimal OA efficacy has been attained, patients should return for follow-up once every six months in the first year and at least annually thereafter. Judicious monitoring of

the patient's OA compliance, jaw function, functional occlusion, dental side effects and state of associated oral structures should be performed. Side effects both in the short and long-term have been shown to affect OA compliance. The evaluation of OA deterioration, OA retention, bite alterations and overall comfort should be thoroughly assessed and managed. In addition, given that inadvertent or inappropriate device modification by the patient may compromise the OA efficacy, assessment of OA integrity and the degree of mandibular advancement should be carried out.

Patients with OSA undergoing OA therapy should be periodically reviewed by the referring physician. The dentist must correspond with the referring medical practitioner with regards to the treatment plan and long-term follow-up. Follow-up allows the assessment of clinical signs and symptoms or worsening OSA. A multi-disciplinary approach and good communication between practitioners is paramount. If the signs and symptoms of OSA worsen or reoccur, an objective review with an overnight sleep test is indicated.

CONCLUSIONS

Oral appliances are an effective therapeutic option for the treatment of snoring and SDB across a broad range of disease severity. However, the response to therapy is non-uniform with some subjects having a very good response, and others having no response. Currently, there are no satisfactory clinical predictors of good and poor responders, and ideally patients should have objective assessment both before and after therapy has been established. There should be careful evaluation of patients' weight when reviewing therapy as weight gain will lead to recurrence of snoring and OSA despite the use of OAs.[67]

Strong evidence support their key role in improving polysomnographic indices and reducing the medical risks associated with OSA. OA therapy for the treatment of snoring and SDB should be carried out by dentists with training and experience in dental sleep medicine. Coordination and correspondence between physician and dentist should be encouraged to enhance treatment outcomes and optimise patient care. Ongoing patient follow-up during OA therapy to monitor treatment efficacy, OA comfort and fit in addition to titration and side effect management are cardinal to long-term treatment success and patient compliance.

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