Role of Oral Devices in Managing Sleep-disordered Breathing Patients

Sleep-disordered breathing (SDB) includes mouth breathing, snoring, upper airway resistance syndrome (UARS), and obstructive sleep apnea (OSA). OSA is defined as five or more episodes of complete (apnea) or partial (hypopnea) upper airway obstruction per hour of sleep. It is estimated to involve 24% of middle-aged men and 9% of middle-aged women. Two to three percent of children have OSA, increasing to 30-40% in obese children. Patients suffer from daytime drowsiness, cognitive impairment, and increased risk of heart attacks, strokes, uncontrolled hypertension, and diabetes. Untreated OSA can significantly impair a patient’s quality of life, and increase morbidity due to medical complications or motor vehicle accidents.

Prosthodontists have a responsibility to restore and maintain the oral health, function, comfort, and appearance of patients. Sleep bruxism and orofacial pain are conditions for which the dentist has diagnostic and management skills. With their additional training and expertise in oral anatomy, occlusion, and temporomandibular joint (TMJ) function, prosthodontists should recognize the signs and symptoms of OSA, refer to the physician for diagnosis, and collaborate with the health team surrounding the patient in providing care that will improve the patient’s oral and general health.

Diagnosis

Oral examination

Ninety percent of airway obstructions occur behind the maxilla and mandible in the soft palate, tongue, and lateral fat pads. The size and shape of the upper airway influences the likelihood of upper airway collapse. A smaller airway (obesity or small maxilla and/or mandible) is more susceptible to collapsing than a larger airway. Imaging studies of the upper airway have demonstrated a larger volume of soft tissue structures (tongue, soft palate, and lateral pharyngeal walls) in patients affected by OSA. OSA patients tend to present with compromised upper airway resulting from skeletal and/or soft tissue abnormalities.

The level of obstruction of the oropharynx can be assessed by using the modified Mallampati classification. The evaluation of the size of the tongue, the presence and size of the tonsils, the opening of the oral and nasal airway can raise concerns on the patency of the airway.

Evaluation of tooth wear and TMJ symptoms is important, as these may play a role in OSA. The terminology “night bruxism” should be replaced by sleep bruxism (SB), as it occurs during sleep periods, which are not necessarily at night. No evidence supports association or causality of SB and
OSA at this time. However, there do appear to be clinical commonalities between SB and OSA. Activation of the masseter muscle is thought to stabilize the mandible, enabling the genioglossus to dilate the upper airway more efficiently. SB has been associated with the end of an apnea event and is thought to be related to the vagal regulatory response to tachycardia. Thirty percent of bruxism patients have OSA, with bruxism increasing muscle tone and possibly dilating the airway. The association between SB, gastroesophageal reflux disorder (GERD), and sleep disturbance has been described as the ‘Bruxism Triad.’

Associated comorbidities

Obesity or increased body mass index (BMI) are associated with OSA. Hypertension, cardiovascular disease, stroke, diabetes, or thyroid disease have been identified as aggravating factors or the results of OSA.

OSA in children

OSA in children is often due to enlarged tonsils and adenoids peaking at 5-6 years of age. Craniofacial morphological characteristics often present in children with airway problems are narrow maxillae, anterior open-bite, mouth breathing, and dolichocephalic profile.

Daytime sleepiness and snoring

Snoring occurs in 95% of patients with OSA. Witnessed apneas and excessive daytime sleepiness are important symptoms. The Epworth Sleepiness Scale (ESS) is a simple questionnaire commonly used in the assessment of daytime sleepiness and the screening for potential OSA. When patients with SB and/or TMD complain about insomnia, snoring, and/or cessation of breathing during sleep, sleepiness of unidentified causes, or uncontrolled hypertension, it is prudent to screen for the presence of SDB (OSA).

Diagnosis

Polysomnography (PSG) performed in a sleep laboratory is the gold standard for the diagnosis of OSA. The diagnosis must be made by a sleep physician, who will prescribe the treatment of choice depending on the severity of OSA. The final management of OSA may require input from the prosthodontist/sleep dentist, ENT, and oral and maxillofacial surgeon (OMFS).

Management of OSA

Treatment modalities

Patients who have been diagnosed with OSA need to be informed of all treatment options, such as sleep
behavioral therapy, oral appliance (OA), positive airway pressure (PAP) therapy, and surgery. Behavioral modifications such as weight control, sleep position, sleep hygiene, and alcohol intake modification can assist in the management of OSA.

The American Academy of Sleep Medicine (AASM) indicates that an OA is the first line of therapy for mild to moderate sleep apnea, and for severe sleep apnea patients who cannot tolerate or are non-compliant with their PAP machine. The PAP machine pneumatically opens the airway using continuous or on-demand positive air pressure, and is considered the gold standard of therapy for OSA.

There are two categories of OA for OSA: mandibular repositioning devices (MRD) and tongue-retaining devices (TRD). The MRD’s objective is to reposition the mandible forward, enough to enlarge the upper airway and prevent it from collapsing. The TRD’s objective is to maintain the tongue in a forward position, preventing it from falling back and obstructing the airway during sleep. TRD’s are poorly tolerated and are not often recommended, but may be considered in patients with TMD who cannot tolerate any jaw advancement.15 For the MRD, the patient’s full range of protrusive movement is measured, and a 75% advancement has been shown to be more effective than 50% advancement.16 Increasing vertical dimension shows promise, but requires further clinical studies. Use of an oral appliance and concomitant use of PAP machines may reduce the pressure required to treat severe OSA, which can increase the acceptability of treatment and comfort for the patient. Thirty-nine to forty-seven percent of sleep bruxism patients demonstrate greater reduction of motor activity with mandibular advancement splints than conventional occlusal splints.17

Occlusal changes observed in patients after long-term use of oral appliances are relatively small and include decreased overbite/overjet and posterior open-bite in the premolar region.18,19 Prosthodontists have an in-depth knowledge of occlusion and TMJ function and are the most qualified to monitor and manage any occlusal changes that can occur with oral devices.

A surgical approach is most effective in children with hypertrophied tonsils and adenoids. Concurrent maxillary expansion and orthodontic/surgical correction of malocclusions will potentially avoid OSA in adulthood.20 Surgical procedures such as uvulopalatopharyngoplasty (UPPP) in adults are considered secondary to non-surgical therapy when the patient is non-responsive. There are concerns regarding the predictability and stability of surgery in the adult OSA patient, but when there is a clear maxillomandibular discrepancy, orthognathic surgery is most effective.21

Prosthodontists who treat patients with OSA should have ongoing training in the diagnosis and management of sleep-related breathing disorders. Understanding the symptoms of sleep-disordered breathing, knowing when to refer to a physician certified in sleep medicine, assessing the TMJ, occlusion, oropharyngeal structures, orofacial pain, and headaches requires additional training and certification, as recommended by the American Academy of Dental Sleep Medicine (AADSM)22 and the
Canadian Sleep Society (CSS).23

Liability

The effect of the use of an OA for SB on OSA remains uncertain.24 Increasing the occlusal vertical dimension (OVD) without mandibular protrusion might aggravate OSA in some patients. It was suggested that the mechanism for increasing AHI could be related to reduced tongue space and rotation and anterior translation of the condyles, which consequently reduces the upper airway size.25 Practitioners should screen patients for OSA prior to fabricating an OA that increases the OVD without mandibular protrusion.26 It would be judicious to assess patients for OSA during the treatment planning stage for a full mouth rehabilitation.5 Prosthodontists must be aware that treatment modalities for OSA may influence the final dental treatment plan.

Conclusion

Due to the complexity and extensive amount of time and financial expenses involved in a prosthodontic rehabilitation as well as the serious health risks of untreated OSA, prosthodontists should include a mandatory screening for OSA for their patients. It is important to have the diagnosis of OSA in the treatment planning stages as it may impact the retention of a future OA required for the OSA patient. Furthermore, increasing the OVD, restricting the tongue space, or prescribing an occlusal splint may have a negative impact on an OSA patient. When prescribed, prosthodontists are encouraged to fabricate the OA; however, they should acquire adequate training in dental sleep medicine. OA’s have been shown to be effective in the treatment of patients with mild to moderate OSA. Patients with severe OSA or who cannot tolerate or are not compliant with PAP therapy can also benefit from the use of an OA.

References


References cont.


References cont.


Authors

Jean C. Wu, DDS
Nancy M.G. Dubois, DMD, Cert Prosth, MDSc, FRCD (C), FACP

Date

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