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 5 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.[1] 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 transport- or work-related accidents.

Prosthodontists play an important role in preserving patients' general health by restoring and maintaining physiological oral function and esthetic appearance. Sleep bruxism (SB) and oro-facial 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

Most airway obstructions occur behind the maxilla and mandible at the level of the soft palate, tongue, and lateral fat pads. The size and shape of the upper airway are among factors to consider in 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 size and fat in the posterior area) in patients affected by OSA. Many OSA patients tend to present with a compromised upper airway resulting from skeletal and/or soft tissue abnormalities. [2] An evaluation of the size of the tongue, the presence and size of the tonsils, the opening of the oral and nasal airways, and the size of the neck can raise concerns on the patency of the airway.[3]

The presence of tooth wear and TMJ symptoms is important, because these may play a role in OSA. The terminology "night bruxism" should be replaced by sleep bruxism (SB), because 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, a sub-group of bruxers do appear to have clinical commonalties[4] in which

the activation of the masseter muscle is thought to stabilize the mandible, enabling the genioglossus to dilate the upper airway more efficiently.[5-7] SB can occur before a breathing event, after a breathing event, or unrelated to a breathing event.[8,9] A significant number of bruxism patients may have OSA, with bruxism increasing muscle tone and possibly dilating the airway.[10,11]

Associated comorbidities

Obesity or increased body mass index (BMI) are associated with OSA. The presence of hypertension, cardiovascular disease, stroke, diabetes, and thyroid disease have been identified as aggravating factors or the results of OSA.[12]

OSA in children

OSA in children is often due to enlarged tonsils and adenoids peaking at 5-6 years of age.[13] Craniofacial morphological characteristics often present in children with airway problems are narrow maxillae, anterior open-bite, mouth breathing, and dolichocephalic profile.[14]

Daytime sleepiness and snoring

Snoring occurs in a high percentage of patients with OSA. Witnessed apneas and excessive daytime sleepiness are also important symptoms, but only witnessed apneic events are pathognomonic for OSA. The Epworth Sleepiness Scale (ESS) is a simple questionnaire commonly used in the assessment of daytime sleepiness and screening for potential OSA. When patients with SB and/or temporomandibular disorder (TMD) complain about insomnia, snoring, and/or cessation of breathing during sleep, sleepiness of unidentified causes, or uncontrolled hypertension or hypertension requiring multiple medications, it is prudent to screen for the presence of SDB (OSA).[4]

Diagnosis

The diagnosis must be made by a sleep physician who will prescribe a polysomnography (PSG) test and suggest the best modality of treatment depending on the severity of OSA. A PSG study performed in a sleep laboratory is the gold standard for the diagnosis of OSA. The role of the prosthodontist is to screen patients using the ESS, Stopbang, and Berlin assessment tools, and an oral examination and refer the patient to a sleep physician for diagnostic and treatment prescription when OSA is suspected. The final management of OSA may require input from ear-nose-throat (ENT) specialists, pneumologists, oral and maxillofacial surgeons (OMFS), prosthodontists or dentists trained in dental sleep medicine, and other professionals as required.

Management of OSA

Treatment modalities

Treatment modalities for OSA include a combination of sleep modification oral appliance (OA), positive airway pressure (PAP), myofunctional therapies, and surgery. Behavioral modifications such as weight control or weight loss, sleep position, sleep hygiene, and alcohol intake modification can assist in the management of OSA.

The American Academy of Sleep Medicine (AASM) guidelines indicate that although OA is not as effective as CPAP, OA may be indicated for mild to moderate OSA when patients cannot tolerate CPAP or do not respond to CPAP.[15] Studies on the effectiveness of OA have shown no more than 56% improvement to normal AHI, with compliance as one factor contributing to improved outcomes.[16] An investigation comparing the effect of CPAP to OA on blood pressure (BP) in patients with OSA found that both CPAP and OA were associated with reductions in BP.[17] Approximately 25% of patients receiving an OA may have no improvements and may aggravate the severity of their sleep apnea.[18] PAP 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 OAs 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. TRDs are poorly tolerated and are not often recommended, but may be considered in patients with TMD who cannot tolerate any jaw advancement.[19] For the MRD, the patient's full range of protrusive movement is measured, and a 75% movement has been shown to be more effective than 50% advancement.[20] However, a systematic review and meta-regression analysis on the effectiveness of different mandibular advancements suggests that it would be prudent to begin therapy at minimum effective advancement, since no significant improvements were found at increased movements.[21] Patient compliance plays an important role in the long-term success of the treatment. The use of an OA concomitant with a PAP machine may reduce the pressure required to treat severe OSA, possibly increasing the acceptability of treatment and patient comfort. Thirty-nine to forty-seven percent of SB patients demonstrate greater reduction of motor activity with mandibular advancement splints than conventional occlusal splints.[22]

Occlusal changes observed in patients after long-term use of OAs are relatively small and include decreased overbite/overjet, and posterior open-bite in the premolar region.[23,24] 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 OAs. Occlusal changes will continue as long as an OA is used. Occlusal correction necessitates discontinuation of OA and alternative therapy such as CPAP.[25]

A surgical approach is most effective in children with hypertrophied tonsils and adenoids. Concurrent maxillary expansion and orthodontic/surgical correction of malocclusions will improve OSA in

children.[26] 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.[15]

Prosthodontists who provide OAs to patients with OSA should have ongoing training in dental sleep medicine (DSM). Understanding the symptoms of SDB, knowing when to refer to a certified physician, 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)[27] and the Canadian Sleep Society (CSS).[28]

Liability

The effect of the use of a night guard for SB on OSA remains uncertain; however, increasing the occlusal vertical dimension (OVD) with a maxillary night guard without mandibular protrusion has been found to aggravate OSA in some patients. It was suggested that the mechanism for increasing the severity of OSA could be related to a reduced upper airway size due to the restriction of the tongue space and the rotation/anterior translation of the condyles.[29] Practitioners should screen patients for OSA prior to fabricating a maxillary night guard that increases the OVD without mandibular protrusion.[30,31] Prosthodontists must screen patients for OSA during the treatment planning stages and be aware that treatment modalities for OSA may influence the final dental treatment plan.[4]

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 through questionnaires for their patients. The simple question, “Do you snore?,” in the medical questionnaire is an excellent marker that can be complemented by OSA questionnaires and specific oral examination. It is important to have the diagnosis of OSA from a sleep physician in the treatment planning stages, because the definitive treatment plan may impact the retention of a future OA required for the OSA patient. Furthermore, increasing the OVD, restricting the tongue space, or prescribing a night guard may have a negative impact on an OSA patient.

When prescribed by the sleep physician, prosthodontists are encouraged to fabricate the OA; however, they should acquire adequate training in dental sleep medicine. OAs have been shown to be effective in the treatment of patients with mild and moderate OSA. Patients with severe OSA or those who cannot tolerate or are not compliant with PAP therapy can also benefit from the use of an OA.

Most recent estimates suggest that a majority of patients remain undiagnosed and untreated. The field of

sleep medicine as it is, will not have sufficient professional and financial resources to sustain the potential demands for treatment. Increasing training of non-physicians in the treatment of uncomplicated cases could be an avenue to meet this demand.[32] Prosthodontists receive advanced training in the oro-facial complex that may benefit the field and should seriously consider investing time in expanding their knowledge in dental sleep medicine.

References

1. Bhattacharee R, Kim J, Kheirandish-Gozal L: Obesity and obstructive sleep apnea syndrome in children: a tale of inflammatory cascades. Pediatr Pulmonol 2001;46:313-323.

2. Lavigne GJ, Cistulli PA, Smith MT: Anatomic predisposing factors in OSA. In Lavigne GJ, Cistulli PA, Smith MT (eds): Sleep Medicine for Dentists: A Practical Overview. Hanover Park, IL, Quintessence, 2009, p. 43.

3. Chi L: Identification of craniofacial risk factors for obstructive sleep apnea using three-dimensional MRI. Eur Respire J 2011;38:348-358.

4. Balasubramaniam R, Klasser G, Cistulli P, et al: The link between sleep bruxism, sleep disordered breathing and temporomandibular disorders: An evidence-based review. J Dent Sleep Med 2014;1:27-37.

5. Yoshida K: A polysomnographic study on masticatory and tongue muscle activity during obstructive and central sleep apnea. J Oral Rehabil 1998;25:603-609.

6. Hollowell DE, Suratt PM: Activation of masseter muscles with inspiratory resistance loading. J Appl Physio 1989;67:270-275.

7. Hollowell DE, Surrat PM: Mandible position and activation of submental and masseter muscles during sleep. J Appl Physio 1991;71:2267-2273.

8. Carra MC, Huynh N, Lavigne G: Sleep bruxism: A comprehensive overview for the dental clinician interested in sleep medicine. Dent Clin North Am 2012;56:387-413.

9. Holley AB, Letieri CJ, Shah AA: Efficacy of an adjustable oral appliance and comparison with continuous positive airway pressure for the treatment of obstructive sleep apnea syndrome. Chest 2011;140:1511-1516.

10. Schames SE, Schames JM, Schames M, et al: Sleep bruxism, an autonomic self-regulatory response by triggering the trigeminal cardiac reflex. J Calif Dent Assoc 2012;40:670-676.

11. Saito M, Yagamaguchi T, Mikami S, et al: Temporal association between sleep apnea-hyponea and sleep bruxism events. J Sleep Res 2013 Nov 4. Doi: 10.1111/jsr.12099. [Epub ahead of print].

12. Lavigne GJ, Cistulli PA, Smith MT: Clinical approach to diagnosis of obstructive sleep apnea. In Lavigne GJ, Cistulli PA, Smith MT (eds): Sleep Medicine for Dentists: A Practical Overview. Hanover Park, IL, Quintessence, 2009, p. 56.

References cont

13. Gozal D, Pope DW Jr: Snoring during early childhood and academic performance at ages thirteen to fourteen years. Pediatr 2001;107:1394-1399.

14. Flores-Mir C, Korayem M, Heo G, et al: Craniofacial morphological characteristics in children with obstructive sleep apnea syndrome: a systemic review and meta-analysis. J Am Dent Assoc 2013;144:269-277.

15. Walker-Engstrom ML, Tegelberg A, Wihelmsson B, et al: 4-year follow-up of obstructive sleep apnea: a randomized study. Chest 2002;121:739-746.

16. Almeida FR, Parker JA, Hodges JS, et al: Effect of a titration polysomnogram on treatment success with a mandibular repositioning appliance. J Clin Sleep Med 2009;15:198-204.

17. Bratton DJ, Gaisi T, Wons AM, et al: CPAP vs Mandibular Advancement Devices and Blood Pressure in Patients with Obstructive Sleep Apnea. JAMA 2015;314:2280-2293.

18. 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;11:773-827.

19. Brown EC, Cheng S, McKenzie DK, et al: Tongue and lateral upper airway movement with mandibular advancement. Sleep 2013;36:397-404.

20. Walker-Engstrom ML, Ringqvist I, Vestling O, et al: A prospective randomized study comparing two different degrees of mandibular advancement with a dental appliance in treatment of severe obstructive sleep apnea. Sleep Breath 2003;7:119-130.

21. Bartolucci ML, Bortolotti F, Raffaelli E, et al: The effectiveness of different mandibular advancement amounts in OSA patients: a systematic review and meta-regression analysis. Sleep Breath 2016 Jan 15. [Epub ahead of print].

22. Landry-Schonbeck A, de Grandmont P, Rompre PH, et al: Effect of an adjustable mandibular advancement splint on sleep bruxism: A crossover sleep laboratory study. Int J Prosthodont 2009;22:251-259.

23. Almeida FR, Lowe AA, Otsuka R, et al: Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: Part 2. Study-model analysis. Am J Orthod Dentofacial Orthop 2006;129:205-213.

24. Doff MHJ, Finnema KJ, Hoekema A, et al: Long-term oral appliance therapy in obstructive sleep apnea syndrome: a controlled study on dental side effects. Clin Oral Investig 2013;17:475-482.

25. Pliska BT, Chen HNH, Lowe AA, et al: Obstructive sleep apnea and mandibular advancement splints: Occlusal effects and progression of changes associated with a decade of treatment. J Clin Sleep Med 2014;10:1285-1291.

26. Guilleminault C, Huang YS, Glamann C, et al: Adenotonsillectomy and obstructive sleep apnea in children: a : Part 2 prospective survey. Otolaryngol Head Neck Surg 2007;136:169-175.

27. American Academy of Dental Sleep Medicine: Policy Statement on the Diagnosis and Treatment of OSA. Available online at http://www.aadsm.org/osapolicystatement.aspx. Accessed February 11, 2015.

References cont.

28. Gauthier L, Almeida F, Arcache JP, et al: Position paper by Canadian Academy of Sleep Medicine. Can Respir J 2012;19:307-309.

29. Nikolopoulou M, Ahlberg J, Visscher CM, et al: Effects of occlusal stabilization splints on obstructive sleep apnea: a randomized controlled trial. J Orofac Pain 2013;27:199-205.

30. Nikolopoulou M, Visscher CM, Aarab G, et al: The effect of raising the bite without mandibular protrusion on obstructive sleep apnea. J Oral Rehabil 2011;38:643-647.

31. Gagnon Y, Mayer P, Morisson F, et al: Aggravation of respiratory disturbances by the use of an occlusal splint in apneic patients: a pilot study. Int J Prosthodont 2004;17:447-453.

32. Phillips B, Gozal D, Malhotra A: What is the future of sleep medicine in the US? Am J Respir Crit Care Med 2015;192:915-917.

Authors

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

Date

Original source document dated Feb. 4, 2005
Revised and approved ACP Board of Directors: June 13, 2015
Revised and approved ACP Board of Directors: June 3, 2016