International Journal of Community Medicine and Public Health
Salamah FSB et al. Int J Community Med Public Health. 2022 Feb;9(2):987-994
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20220016

Review Article

Diagnosis and treatment of obstructive sleep apnea

Fahad Saleh Bin Salamah[1], Hiba Mohammed Al Fayez[2]*, Raghad Taha Melibary[2]

[1]Department of Orthodontics, King Saud Medical City, Riyadh, Saudi Arabia
[2]General Dentist, Ministry of Health, Riyadh, Saudi Arabia

Received: 12 December 2021
Accepted: 27 December 2021

***Correspondence:**
Dr. Hiba Mohammed Al Fayez,
E-mail: Heba95.AlFayez@gmail.com

ABSTRACT

Obstructive sleep apnea (OSA) has become a health issue of high prevalence. The prevalence is rising between 1990-2010 by approximately 30%, with absolute increases of 4.2% in women and 7.5% in men. This condition is characterized by a breathing disorder of partial or complete upper airway obstruction leading to increased resistance to airflow and potential cessation of breathing during sleep. Its multifactorial etiology Such etiologies involve obesity, craniofacial anatomy, and the use of stimulants and medications to reduce muscle tension. This review aims to summarize the recent diagnosis and treatment modalities of OSA. The database PubMed and google scholar were searched for relevant published records. A total of 39 articles were collected randomly limited to the English language. We placed no restrictions on the date of publication. Evidence from methodological studies indicate that undiagnosed OSA is associated with hypertension, cardiovascular disease, stroke, and daytime sleepiness. Thorough clinical and instrumental examinations should precede a proper diagnosis selection. The primary goal of treatment is reducing the Patient's symptoms, depending on the severity of the patient's condition. OSA can be treated in different ways, including behavioral therapy, medical device, surgery, and pharmacological therapies. There are limitations in dental school about education and training of OSA and oral appliances (OA). OSA teamwork must include both qualified sleep physicians and dentists. The Orthodontist plays a critical role in evaluating and treating OSA patients, choosing the right oral appliance, and assessing and adjusting the appliance.

Keywords: Orthodontics, OSA, Diagnosis, Appliance, Treatment

INTRODUCTION

Understanding the complex association between healthy sleep, brain and body functions is relatively new. The knowledge base on sleep disorders has grown exponentially in the last 20 years. Since the publication of the first book on sleep medicine in 1989, sleep as a specialty was introduced in its own right.[1] Dental sleep medicine is also a rapidly emerging discipline that supports sleep specialists, pulmonologists, otolaryngologists, neurologists, and psychiatrists in detecting and treating patients with sleep-disordered breathing.

OSA is a disease characterized by repetitive, complete, or partial collapse of the pharyngeal during sleep, resulting in sleep discontinuity and decreased oxygen saturation.[2] OSA is ubiquitous in the community and often undiagnosed, decreasing sleep quality by interrupting the continuity of sleep and bringing the individual into a short-lived state of arousal.[1] According to the American national sleep foundation, based on scores from the Berlin questionnaire, the prevalence of OSA in adulthood is between 16%-37%. Among them, the 50-64 age group have the highest chance of being diagnosed with OSA in both gender groups (37% in males and 29% in females).[3]

The adverse outcome associated with OSA includes myocardial infarction, coronary artery disease, cognitive

impairment, depression, excessive daytime sleepiness, and increased risk of motor vehicle accidents.[1-3] Few other evidence has linked OSA with memory deficits, irritability, erectile dysfunction, frequent nocturnal waking due to choking or gasping, nocturia, morning headaches, poor concentration.[4,5]

The effective treatment of OSA should significantly benefit patients by stabilizing breathing during sleep to eliminate excessive daytime sleepiness, cardiovascular diseases, and neuropsychiatric.[6] The gold standard treatment of the patient with OSA is continuous positive airway pressure (CPAP) remains the most common and most efficacious treatment, this device function as a pneumatic splint that stabilizes and opens the upper airway and prevents its repeated collapse during sleep. Device acceptance, adherence, and tolerance by a patient are often poor, which reduces the effectiveness of (CPAP).[7] Therefore, several alternative treatment strategies have been used to improve adherence, including surgical intervention, positional therapy, pharmacologic treatment, and weight-loss interventions for obese patients. This review aims to summarize diagnosis protocol and recent treatment modalities for OSAS from a dental perspective.

LITERATURE REVIEW

This literature review is based on an extensive literature search in Medline, Cochrane, and EMBASE databases which was performed on 27th November 2021 using the medical subject headings (MeSH) or a combination of all possible related terms, according to the database. To avoid missing potential studies, a further manual search for papers was done through Google Scholar while the reference lists of the initially included papers. Papers discussing diagnosis and treatment of OSA were screened for useful information. No limitations were posed on date, language, age of participants, or publication type.

DISCUSSION

Etiology

OSA occurs due to increases in collapsibility of the upper airway during sleep at the pharyngeal level, which increases respiratory effort to maintain airflow of the constricted airway accompanied by an increase in hypercarbia and decrease in hypoxemia.[8] Genioglossus muscle is considered as major upper airway dilator muscle, which contracts with each inspiration to prevent a subsequent collapse of the tongue, assisted by the levator veli palatini and tensor veli palatini muscles (Advancing and elevating the soft palate) and the geniohyoid and stylopharyngeus muscles (opposing medial collapse of the lateral pharyngeal walls).[9] The muscular tension of soft palate muscles and the tongue, posterior pharyngeal wall, uvula, and respiratory rhythm decrease during sleep, leading to retraction of the tongue to approach pharyngeal walls.[10] Concerning sleeping position, OSA patients tend to use the supine position more frequently during sleep which can aggravate "AHI" up to 75% higher versus 50% in the general population using the same position.[11] Sleep posture has a significant influence on the dimensions and collapsibility of the upper airway. To illustrate, during sleeping in a supine position, gravitational forces can cause a decrease in pharyngeal space due to changing the mandible's position backward, especially in obese patients due to accumulated adipose tissues in the submandibular region.[10]

The etiology is multifactorial, including adenotonsillar hypertrophy, neuromuscular disorders, and craniofacial anomalies even through hormonal changes (e.g., pregnancy or menopause) that can cause collapsibility of the upper airway.[8,10] Heinzer et al found that postmenopausal women present 3-to 6-times higher OSA prevalence.[11] An exposure-response pattern is believed to exist between the time of menopause and the severity of AHI. Interestingly, the prevalence of OSA in menopausal women receiving hormone replacement therapy (HRT) is reduced, suggesting a hormonal effect on the risk of sleep apnea regardless of age and body mass index (BMI).[12] However, the potential benefit of using HRT must be weighed against the risk factors. In addition, dental malocclusion such as micrognathia /retrognathism, bilateral/unilateral crossbite, Macroglossia, long soft palate, and steep mandibular plane angle is associated with OSA (Figure 1).[8,9,11,13]

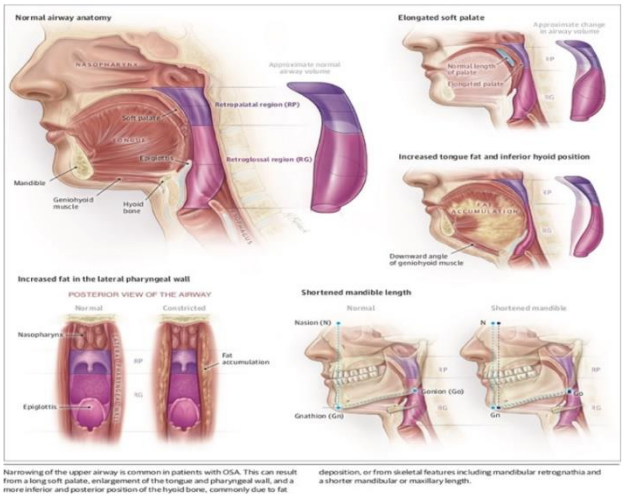

Figure 1: Anatomic features contributing to OSA.[9]

Several conditions considered as risk factors and have been identified and linked to OSA in adults include obesity, alcohol, sedentary lifestyle, sleep position, late-evening eating habits, palatine adenoid tonsils, smoking, nasal polyp and central nervous system depressant substance.[10,11] Research has shown that a 10% reduction in body weight can lead to a 50% decrease in apneas and an increase in arterial oxygen saturation; this also improves the sleeping pattern.[14] Moreover, the genetic influence on the craniofacial structure has led to a higher prevalence of OSA in certain ethnic groups that have

been studied.[8] A study of 5 years follow-up shows that gender plays an essential role in the occurrence of OSA symptoms in middle-aged patients of (11.1% in men and 5.9% in women). Men with a 10 kg increase in body weight show a 5.2-fold risk of higher apnea/hypopnea index (AHI), over 15 events per hour, while in women, this risk is a 2.5-fold higher.[10]

Clinical feature

Detailed clinical evaluation is an integral part of evaluating patients with suspected OSAS, although most reports indicate that predicting disease via clinical features alone has limited value.[15] The clinical feature often has a history of chocking, snoring, and apnea during sleep; the consequences of untreated OSA are wide-ranging and postulated to result from frequent nocturnal awakenings, fatigue, headaches, insomnia, and excessive daytime sleepiness.[8,16] Witnessed apnea consider as an excellent diagnostic predictor of OSAS, but they cannot predict the severity of the disease. Bed partner's concern of witnessing apnea during sleep is a common reason for referral to a sleep clinic. Among female patients with OSAS, witnessed apneas are less common and reported in up to 6% of the normal population.[15]

Patients identified by screening of the general population reported excessive daytime sleepiness (EDS) by 15%-20% of people with OSA.[9] Furthermore, several studies reported that the severity of EDS and sleep apnea do not correlate.[15] although patients without accompanying symptoms of gasping or choking are more common than patients who experience symptoms, A systematic review concluded that nocturnal gasping or choking is the most reliable indicator of OSA on history and physical examination. At the same time, snoring is not specific.[9] A large number of studies have shown that, over time, untreated OSA has a negative impact on and related to cardiovascular and metabolic health and is associated with hypertension, type II diabetes, myocardial infarction, coronary artery disease, stroke, Heart failure, pulmonary hypertension, and cancer.[9,12,17,18] Multiple studies have consistently demonstrated the link between sleep apnea and hypertension. The finding of hypertension in patients with symptoms suggestive of OSA increases the likelihood of developing the disease. In addition, Patients with drug-resistant hypertension show be an exceptionally high incidence of OSA.[15] Patients with untreated OSA increase the risk of ischemic stroke, particularly in men patients with an incidence of AHI more than 19 per hour or women with an AHI of more than 25 incidences per hour.[19]

At present, OSA is recognized as a growing concern and a significant public health problem worldwide. Due to the increase of the obesity pandemic, aging of our society, and improvements in detection and testing methods. In the United States alone, the number of people affected with untreated sleep-disordered breathing is approximately 12 to 18 million adults, and this number is increasing.[12] Identifying and treating OSA is essential for several reasons. Treatment for OSA has been shown to improve quality of life, reduce the rate of motor vehicle accidents, and decrease healthcare usage. However, there are challenges and uncertainties in making a diagnosis, and several questions remain unanswered.[20]

Assessment and diagnosis

The diagnosis of OSA requires a combined assessment of the relevant clinical symptoms and the objective demonstration of abnormal breathing during sleep. Examination for OSA is recommended in any patient complaining of excessive daytime sleepiness, unexplained nocturia, headache, witnessed nocturnal apneas, nocturnal gastroesophageal reflux, and overweight. One of the most efficient screening methods for OSA in a dental setting is a questionnaire composed of commonly asked questions about excessive fatigue, sleepiness during the day, snoring, and breathing pauses at night.[9] Two of the best questionnaires are the stop-bang questionnaire and the Berlin questionnaire. The stop-bang questionnaire consist of four yes/no questions and four clinical attributes to assess the high-risk patients of OSA if three or more symptoms of snoring, tiredness, observed apneas, hypertension, BMI >30, age >50 years old, neck<40 cm and male gender are present.[1,12] To confirm the appropriate diagnosis, the diagnostic strategy is composed of collecting data via questionnaire, physical examination, objective testing (in-laboratory polysomnography (PSG) and home sleep apnea test (HSAT)), and education of the patient.[21] Physical examination of the upper airway may discover anatomic abnormalities, such as macroglossia, tonsillar hypertrophy, or retrognathia, but normal findings of upper airway examination do not rule out OSA. If the clinical evaluation identifies OSA, diagnostic confirmation of the diagnosis requires overnight testing made by a sleep physician.[9] In addition, it is highly desirable to interview the bed partner to obtain additional information based on direct observation of the patient while asleep.[21]

Polysomnography (PSG) is a gold standard sleep test used to identify the presence and severity of OSA by measuring neurologic electroencephalogram and cardio-respiratory parameters during sleep. Respiratory sensors detect a reduction in ventilation, which is classified as apnea, almost complete cessation of airflow for 10 seconds, hypopnea as partial decrease in the airflow by 10 seconds, or respiratory-effort-related arousals (subtle changes in airflow due to increased upper airway resistance that result in arousals).[22] Apnea hypopnea index AHI is defined as the total number of apneas, hypopnea events divided by total sleep time in hours derived from scoring data obtained by multichannel polysomnography (PSG) in specialized sleep laboratories.[23] The American Academy of sleep medicine (AASM) categorized OSA severity according to AHI as mild (AHI 5-15 per hour of sleep), moderate (AHI >15-30 events per hour of sleep), or severe (AHI >30 events

per hour of sleep).[19] Notably, other measures must be considered during patient assessment. The search for a global predictive index that allows a more meaningful assessment of the effectiveness of treatment and the risk of morbidity and mortality compared to AHI remains an open research question.[11]

Various types of home sleep apnea testing (HAST) are available in clinical use, (HSAT) records at least three channels of data while the patient sleeps at home, usually monitoring airflow, snoring, respiratory effort, heart rate, and oximetry.[19] There are several advantages of HAST, which include lower cost, greater convenience compared with PSG; it appears evident that patients usually prefer HAST to the sleep laboratory environment since patients reported to sleep better and they have a better overall test experience, this comes in disagreement with what Fry study reported that patients preferred the lab study.[15] There are also disadvantages, as many as 17% the possibility of false-negative results found, and up to 18% have a technical failure due to self-applied sensor by the patient without supervision by technologist.[19] therefore, for patients with unexplained sleepiness and suspicion for OSA, a negative result with HAST should be followed by laboratory-based polysomnography to confirm the diagnosis; this approach is accurate and cost-effective.

Treatment

The treatment of OSA needs a topodiagosis of the collapse site of the upper airway.[24] There are different aspects of management of OSA, including behavioral management, medical devices, oral appliance, pharmacological treatment, and surgery. Behavioral measures include abstinence from alcohol, preventing supine sleep position, exercising, encouraging weight loss, avoiding fatty meals, and late evening eating habits.[9,11] There is no clear definition of the ideal sleeping positional for OSA; some studies state that non-supine position lower AHI by 50% than supine position during sleep. There is usually lesser soft tissue in the lateral sleep position that exerts obstructive forces on the upper airway.[25] Furthermore, for all over-weight patients with sleep-disordered breathing, it is recommended to reduce their weight, the most improvement in OSA in the following six months. OSA may predispose patients to gain weight because of low activity levels due to sleep deprivation, insulin resistance, and/or resistance to weight loss due to the effect of leptin. However, this strategy needs time and is often very difficult to achieve.[16,24]

Continuous positive airway pressure (CPAP) is the most effective method to manage OSA, and it considers the first line of therapy for most patients. The mechanism of action improves the quality of sleepiness by acting as a pneumatic splint to elevate and maintain constant pressure along the upper airway during inspiration and expiration. It improves sleep quality and daytime sleepiness following one month of using CPAP.[12,16] The indirect effect of using CPAP include increased vagal tone, increased cardiac output, increased stroke volume, decreased systemic vascular resistance, and reduced risk of cardiovascular mortality.[16]

On the other side, noncompliance patients have been categorized by Zozula et al due to tolerance problems, psychological problems, and lack of instruction, support, or follow-up. Dry mouth, conjunctivitis, rhinorrhea, skin irritation, pressure sores, swelling of the mucous membrane, nasal septum bending, nasal congestion and epistaxis, mask leaks, difficulty exhaling, aerophagia, chest discomfort, and bed partner intolerance are considered as tolerance problems. They can result as a side effect of using CPAP appliances. Additionally, psychological issues include lack of motivation, claustrophobia, and anxiety.[10,16,26] Different Types of CPAP are categorized as standard CPAP, bi-level CPAP, and automated CPAP. Standard type has a potential disadvantage to conventional CPAP in the inability to compromise between the side effects of high pressures and adequate airway pressures, which may vary based on body position, sleep stage, and nasal patency.[16] Bi-level CPAP means bilevel positive airway pressure therapy permits independent adjustment of inspiratory and expiratory pressures. While automated CPAP can continuously adjust airway pressures, its role is to provide greater efficacy and/or compliance than standard CPAP.[16]

Oral appliances (OA)

The rationale for OA is to increase the posterior oropharyngeal airway space, reducing upper airway collapsibility during sleep. In 2006, the American Academy of Sleep Medicine (AASM) updated its practice parameters to treat OSA with OA. In this update, the AASM stated that OA are indicated for use in patients who prefer OA over CPAP with mild-to-moderate severity, patients who do not respond or are not appropriate to CPAP, or who fail treatment attempts either with CPAP or behavioral measures. As CPAP is a more efficacious treatment, it is recommended that CPAP should be considered before OA for patients with severe OS.[16,27]

There are differences in the design features of commercially available OA. Differences predominantly relate to the degree of customization to the patient's dentition. Custom oral appliance shows improved minimum oxygen saturation by 3.22% in adult patients with OSA, reduced AHI and respiratory disturbance index (RDI), and lower arousal index. In contrast, non-custom OA do not significantly improve minimum oxygen saturation in adults, reduce AHI and respiratory disturbance index (RDI) in adults, and have insufficient evidence for improvement in arousal index in adults with OSA.[7,28] Bailey established six criteria to maximize the outcomes of an oral appliance and reduce side effects. First criteria, the adjustability, the OA can be modified as needed over time. Titratability, with this criterion, jaw position is easily adjusted without needing to significantly

alter or remake the OA. Posterior support, the oral appliance has some type of support in the posterior aspect, which allows for contact between the upper and lower arches. This criterion is essential to provide support for TMJs during use. Full tooth coverage, the teeth must be covered to prevent the potential for tooth movement or eruption, even though it is happening either slight movement or eruption but is often not significant. Jaw mobility, normal mandibular movement will occur during sleeping, like swallowing, licking the lip, or altering jaw position during positional changes. This criterion will allow the mandible for some degree of movement during sleeping. Lastly, patent nasal passages, one of the successful primary outcomes of wearing OA, the ability of patients to breath from his/her nose; this plays an essential role in improving the oxygen level and reducing mouth breathing.[29]

Types of OA

First, tongue-retaining devices (TRD) or tongue-stabilizing devices (TSDs), consider the second type of treatment. Use a suction pressure to maintain the tongue in a protruded position during sleep, which position the tongue anteriorly utilizing negative pressure, indicated in patients who have few teeth or edentulous patients, macroglossia patients, or cannot adequately advance their mandible, mechanism of action is enlarge the volume of the upper airway (Figure 2).[5,16,27,30]

Figure 2: Tongue stabilizing device.

Secondly, mandibular advancement devices (MAD), also known as mandibular advancement splints, mandibular-repositioning devices (Figure 3).[27,31] It can increase the anteroposterior and lateral dimensions of the upper airway, especially at the level of the velopharynx by moving the mandible and associated structures, i.e., tongue and hyoid bone in anterior position.[16] MADs are indicated in patients with mild to moderate OSA who prefer OA over CPAP therapy. However, patients with moderate to severe temporomandibular disorders (TMD), inadequate protrusive ability, and multiple missing teeth are considered a contraindication to use MAD.[21]

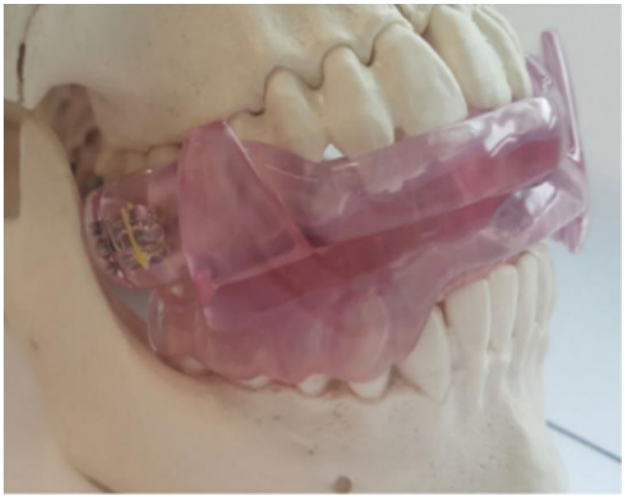

Figure 3: Mandibular advancement device.

Accordingly, the patients should be examined to determine if there is any clicking or crepitation in the TMJ with jaw opening and to assess how far the patient can protrude his/her mandible; 6 mm is required for an adequate response.[21] There are specific requirements must-have in the patient like good stable dentition, typically 6 to 8 teeth per arch; if fewer than these present in any arch, the appliance can cause significant movement or loosening of existing teeth. However, the case must be free from caries, gingivitis, periodontitis or loose teeth, and significant bruxis.[1,21,32] A prospective longitudinal study conducted by Teixeira et al show a modification done in a twin block (TB) appliance, fabricated with self-curing acrylic resin with occlusal coverage and consists of two parts for the upper arch and the other for the lower arch. The use of a modified TB was based on cost, ease of fabrication, and greater mandibular motion. The advantage of the TB appliance is that many laboratories can simply fabricate, requiring no training or specific materials, leading to increased costs and access difficulties.[6] A greater advancement of the mandible is required in patients with severe OSA. Therefore, temporomandibular joint symptoms should be closely examined and monitored by cephalometric radiographs. A study stated that each 2-mm mandibular advancement coincides with approximately 20% improvement of the number and severity of nocturnal desaturations. However, in case of urgent treatment, it is advisable to manage with CPAP over MAD.[16,29]

Complete mouth examination should be done before placing the appliances, all the implants, and crown if there should be assessed for stability; as mentioned above, the success of oral appliance depends on dentition's health. However, force is applied on the teeth during appliance use leads to an increased risk of bite change and can be noticed during the first years of treatment. Bite change is the most common side effect of

MAD; more severe changes are expected in patients who have edentulous or fewer teeth present. The initial type of bite is associated with the degree of bite change; patients with normal bites or class III malocclusion seem to be at high risk for undesirable bite change. Unlike class II malocclusion patients might receive a favorable orthodontic result of oral appliance treatment (Figure 4).[17,32]

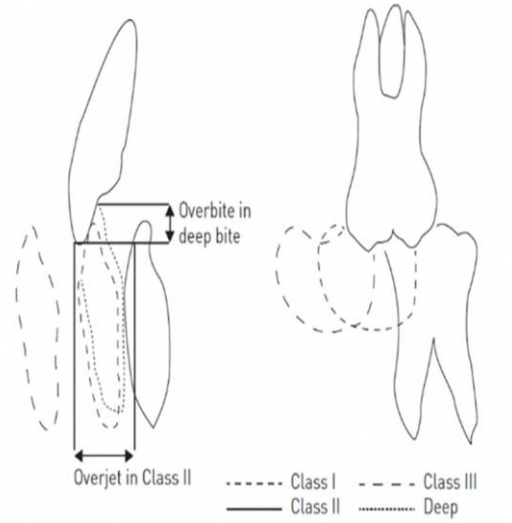

Figure 4: Different dental occlusion types that are useful in mandibular advancement device.

The success measurement of OA includes a decrease in AHI, improvement of oxygen desaturation events, and decrease in symptoms. Many studies have shown the benefits of the OA versus placebo in decreasing AHI. Even though there is still uncertainty as to what kind of MAD has desired outcome for patients with OSA, especially in severe cases, further research needs to be done to compare different appliances and designs to shed light on this issue.[12,29]

Generally, clinical improvement in symptoms has been shown in two-thirds of patients experience with OA therapy. Many studies state that the response to OA use is non-uniform. That is means there are responders and non-responders to the OA treatment; thus, it is misleading to merge the data. In contrast, CPAP practically 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, CPAP can cause trouble for many patients due to nasal obstruction on long-term use of CPAP and other factors such as mouth leaks.[24] At ten years follow-up, both the MAD and CPAP groups showed a significant reduction in AHI. Whereas Michiel et al conducted a study of two years follow up, showed no significant difference between oral appliance therapy and CPAP in treating mild to severe OSA. However, dental follow-up for OA is initially at 2-4 weeks, then at 6 and 12 months subsequently annually.[11,33,34]

Medications

Medication can be used adjunctively to improve residual daytime sleepiness in OSA patients even through regular use of CPAP at effective pressures. Such medication modafinil (Provigil; Cephalon, Frazer, PA) is wake promoting that helps wakefulness and alertness. The most common adverse effects of this medication are headache and nervousness. Antidepressants such as protriptyline and selective serotonin receptors inhibitors were used in the past; nowadays, there is very little evidence to recommend protriotyline for treating OSA.[16]

Surgical intervention

It is indicated for severe OSA patients. One of the most effective surgical interventions for correcting OSA is maxillomandibular advancement, which involves the maxilla and mandible being simultaneously advanced to enlarge the retropalatal and retro-lingual airway.[35] The patient may seek this operation because they are unsatisfied with wearing OA and not tolerate them. Another alternative option is uvulo-palatopharyngoplasty (UPPP) which involves excision of tonsils, uvula, and posterior velum. The American Academy of sleep medicine does not recommend UPPP for treating moderate to severe OSA patients since UPPP does not frequently achieve normalization of AHI.[22,24] There are different surgical techniques to prevent the posterior collapse of the tongue musculature: the hyoid suspension myotomy procedure and lingual tonsil surgery; in this case, the tongue size will be altered by intrinsic reduction, radiofrequency, or plasma frequency ablation or partial glossectomy. Significant side effects such as discomfort and hemorrhage may occur. Waite et al state a concept of multilevel procedure for OSA. It is combined surgery at different levels, nose levels, palate levels, transoral tongue surgery, genioglossus advancement, and a maxillomandibular advancement osteotomy.[24]

Treatment modalities for pediatric OSA patients

OSA affects 2-3% of children, with the age peak occurring between 2-8 years old. Untreated OSA increases the risk of growth failure, cognitive impairment, and endothelial dysfunction. In a classic way, the treatment of choice for pediatric is adenotonsillectomy (AT) procedure to remove both the adenoids and tonsils. Some studies reported AT not always being the first line of treatment, especially with mild severity cases. At the same time, the most common non-surgical therapy is continuous positive airway pressure (CPAP). The newer treatment modalities include weight loss, anti-inflammatory drug, dental treatment and high flow nasal cannula. [36,37]

Knowledge of OSA in dental professionals

During routine dental examination and using a simple screening questionnaire, the dentist can play a critical role

in recognizing the symptoms of OSA, screen risk factors of a small upper airway, or other anatomical deformities. This can help reduce the problem of undiagnosed OSA.[38] It would be imprudent to provide treatment in the absence of sleep medicine expertise. In 2012, Canadian dental sleep medicine professionals ruled out the responsibilities of a dentist in the diagnosis and treatment of OSA. A Dentist must identify and evaluate the symptoms of OSA then refer the patient to a qualified sleep physician. The dentist should check oral health in general and bruxism as specific; as a result of poor sleep quality, also assess orofacial pain and temporal headache. Subsequently, any oral effects should be managed well with suitable treatment options and any side effects with OAT. At the same time, sleep physicians should notice any changes physically and mentally in OSA patients. As well as monitor the efficacy and safety of the treatment and accurately assess the result of the home sleep apnea test (HAST). It is important to illustrate dentist will mainly observe treatment but will not diagnose the patient as a final follow-up.[21] The essential education and training of OSA and oral appliance in dental school are limited; for this reason, several cases cannot be identified easily by the dentist.[3]

CONCLUSION

Sleep-related breathing disorder is considered one of the significant public concerns. Interdisciplinary teams, including sleep medicine physicians and dentists, are part of diagnosing, treating, and following up with OSA patients. Relevant clinical symptoms and physical examination are required to diagnose OSA. In addition, different treatment strategies are delivered depending on the severity of the patient.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Barewal RM, Hagen CC. Management of snoring and obstructive sleep apnea with mandibular repositioning appliances: a prosthodontic approach. Dent Clin North Am. 2014;58(1):159-80.
2. Ng A, Gotsopoulos H, Darendeliler AM, Cistulli PA. Oral appliance therapy for obstructive sleep apnea. Treat Respir Med. 2005;4(6):409-22.
3. Chen H, Lowe AA. Updates in oral appliance therapy for snoring and obstructive sleep apnea. Sleep Breath. 2013;17(2):473-86.
4. Johal A, Fleming PS, Manek S, Marinho VC. Mandibular advancement splint (MAS) therapy for obstructive sleep apnoea--an overview and quality assessment of systematic reviews. Sleep Breath. 2015;19(3):1101-8.
5. Ng JH, Yow M. Oral Appliances in the Management of Obstructive Sleep Apnea. Sleep Med Clin. 2019;14(1):109-18.
6. De Britto Teixeira AO, Abi-Ramia LB, De Oliveira Almeida MA. Treatment of obstructive sleep apnea with oral appliances. Prog Orthod. 2013;14:10.
7. Sutherland K, Vanderveken OM, Tsuda H, Marklund M, Gagnadoux F, Kushida CA, Cistulli PA. Oral Appliance Treatment for Obstructive Sleep Apnea: An Update. J Clin Sleep Med. 2014;10(2):2014.
8. Behrents RG, Shelgikar AV, Conley RS, Flores-Mir C, Hans M, Levine M et al. Obstructive sleep apnea and orthodontics: An American Association of Orthodontists White Paper. Am J Orthod Dentofacial Orthop. 2019;156(1):13-28.
9. Gottlieb DJ, Punjabi NM. Diagnosis and Management of Obstructive Sleep Apnea: A Review. JAMA. 2020;323(14):1389-400.
10. Kostrzewa-Janicka J, Śliwiński P, Wojda M, Rolski D, Mierzwińska-Nastalska E. Mandibular Advancement Appliance for Obstructive Sleep Apnea Treatment. Adv Exp Med Biol. 2017;944:63-71.
11. Lavigne GJ, Herrero Babiloni A, Beetz G, Dal Fabbro C, Sutherland K, Huynh N, Cistulli PA. Critical Issues in Dental and Medical Management of Obstructive Sleep Apnea. J Dent Res. 2020;99(1):26-35.
12. Barewal RM. Obstructive Sleep Apnea: The Role of Gender in Prevalence, Symptoms, and Treatment Success. Dent Clin North Am. 2019;63(2):297-308
13. Carvalho FR, Lentini-Oliveira DA, Prado LBF, Prado GF, Carvalho LBC. Oral appliances and functional orthopaedic appliances for obstructive sleep apnoea in children. Cochrane Database of Systematic Reviews 2016;10.
14. Wojda M, Jurkowski P, Lewandowska A, Mierzwińska-Nastalska E, Kostrzewa–Janicka J. Mandibular Advancement Devices in Patients with Symptoms of Obstructive Sleep Apnea: A Review. In: Pokorski M. (eds) Medical Science and Research. Adv Exp med Biol. 2014;1153.
15. Walter M. Diagnosis of Obstructive Sleep Apnea in Adults. Proceedings Am Thoracic Society. 2008;5:154-60.
16. Fleisher KE, Krieger AC. Current trends in the treatment of obstructive sleep apnea. J Oral Maxillofac Surg. 2007;65(10):2056-68.
17. Marklund M, Braem MJA, Verbraecken J. Update on oral appliance therapy. Eur Respir Rev. 2019;28(153):190083.
18. Ahrens A, McGrath C, Hägg U. A systematic review of the efficacy of oral appliance design in the management of obstructive sleep apnoea. Eur J Orthod. 2011;33(3):318-24.
19. Laratta CR, Ayas NT, Povitz M, Pendharkar SR. Diagnosis and treatment of obstructive sleep apnea in adults. CMAJ. 2017;189(48):E1481-8.
20. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K et al. Clinical Practice Guideline for Diagnostic Testing for Adult Obstructive Sleep Apnea: An American Academy of

Sleep Medicine Clinical Practice Guideline. J Clin Sleep Med. 2017;13(3):479-504.
21. Shigemoto S, Shigeta Y, Nejima J, Ogawa T, Matsuka Y, Clark GT. Diagnosis and treatment for obstructive sleep apnea: Fundamental and clinical knowledge in obstructive sleep apnea. J Prosthodont Res. 2015;59(3):161-71.
22. Chang HP, Chen YF, Du JK. Obstructive sleep apnea treatment in adults. Kaohsiung J Med Sci. 2020;36(1):7-12.
23. Escourrou P, Grote L, Penzel T. The diagnostic method has a strong influence on classification of obstructive sleep apnea. J Sleep Res. 2015;24:730-8
24. Ngiam J, Balasubramaniam R, Darendeliler MA, Cheng AT, Waters K, Sullivan CE. Clinical guidelines for oral appliance therapy in the treatment of snoring and obstructive sleep apnoea. Aust Dent J. 2013;58(4):408-19.
25. Cistulli PA, Gotsopoulos H, Marklund M, Lowe AA. Treatment of snoring and obstructive sleep apnea with mandibular repositioning appliances. Sleep Med Rev. 2004;8(6):443-57.
26. Martha S, Luis A, Yuan-Lung H, Guevara P, Enciso M. Effects of CPAP and mandibular advancement device treatment in obstructive sleep apnea patients: a systematic review and meta-analysis. Sleep Breathing. 2018;22.
27. Chan AS, Cistulli PA. Oral appliance treatment of obstructive sleep apnea: an update. Curr Opin Pulm Med. 2009;15(6):591-6.
28. Ramar K, Dort LC, Katz SG, Lettieri CJ, Harrod CG, Thomas SM 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.
29. Bailey DR. Dental therapy for obstructive sleep apnea. Semin Respir Crit Care Med. 2005;26(1):89-95.
30. Chan AS, Lee RW, Cistulli PA. Dental appliance treatment for obstructive sleep apnea. Chest. 2007;132(2):693-9.
31. Kim JW, Kwon SO, Lee WH. Nocturnal heart rate variability may be useful for determining the efficacy of mandibular advancement devices for obstructive sleep apnea. Sci Rep. 2020;10(1):1030.
32. Mickelson SA. Oral Appliances for Snoring and Obstructive Sleep Apnea. Otolaryngol Clin North Am. 2020;53(3):397-407.
33. Uniken Venema JAM, Doff MHJ, Joffe-Sokolova D, Wijkstra PJ, van der Hoeven JH, Stegenga B 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 Mar 15;16(3):353-9.
34. Doff MH, Hoekema A, Wijkstra PJ, van der Hoeven JH, Huddleston Slater JJ, de Bont LG, Stegenga B. Oral appliance versus continuous positive airway pressure in obstructive sleep apnea syndrome: a 2-year follow-up. Sleep. 2013 Sep 1;36(9):1289-96.
35. Garg RK, Afifi AM, Sanchez R, King TW. Obstructive Sleep Apnea in Adults: The Role of Upper Airway and Facial Skeletal Surgery. Plast Reconstr Surg. 2016;138(4):889-98.
36. Tapia IE, Marcus CL. Newer treatment modalities for pediatric obstructive sleep apnea. Paediatr Respir Rev. 2013;14(3):199-203.
37. Rauf KK, Abdul, Anil S. High-flow Nasal Cannula in Children: A Concise Review and Update. 2019.
38. Quan SF, Schmidt-Nowara W. The Role of Dentists in the Diagnosis and Treatment of Obstructive Sleep Apnea: Consensus and Controversy. J Clin Sleep Med. 2017;13(10):1117-9.

Cite this article as: Salamah FSB, Al Fayez HM, Melibary RT. Diagnosis and treatment of obstructive sleep apnea. Int J Community Med Public Health 2022;9:987-94.