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Chapter 12

Contemporary Treatment Approaches to Obstructive Sleep Apnea Syndrome

Handan Tuğçe Oğuz

Additional information is available at the end of the chapter

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Abstract

Upper airway diseases decrease sleep time and/or quality, which leads to excessive day time sleepiness, fatigue, and lack of concentration. Upper airway diseases can be classified into two groups: upper airway resistance syndrome and sleep apnea syndrome. Sleep apnea syndrome is a disease, which occurs during sleeping, characterized by apneic and hypopneic events, low oxygen levels in lungs, blood oxygen desaturation, sleep arousal, and/or awakening. It increases morbidity and mortality of the patients. Sleep apnea syndrome has three types: central, mixed, and obstructive. Central and mixed apnea can be treated only by medical practitioners. But in the treatment of obstructive sleep apnea, orthodontists and maxillofacial surgeons have an important role. The aim of this chapter is to present current information about etiology, diagnostic tools, and treatment alternatives of obstructive sleep apnea and to introduce dental sleep medicine to orthodontists.

Keywords: dental sleep medicine, obstructive sleep apnea, dental treatment approaches for obstructive sleep apnea, pediatric obstructive sleep apnea

1. Introduction

Sleep-related breathing disorders are pathological changes in respiratory pattern during sleep. They increase morbidity and mortality, decreasing the life quality of patients. The patients complaining about waking up restless and headache, daytime sleepiness, lack of concentration, emotional changes, and unbalanced blood pressure may probably have a secret sleep-related breathing disorder.

Dental sleep medicine is an area of dental practice that focuses on dental therapy to treat sleep-disordered breathing, including snoring and obstructive sleep apnea. Dentists who are

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specialized in dental sleep medicine can diagnose the early signs and symptoms of potential obstructive sleep apnea.

2. Upper airway diseases

Upper airway diseases decrease sleep time and/or quality, which leads to excessive daytime sleepiness, fatigue, and lack of concentration. Upper airway diseases can be classified into two groups: upper airway resistance syndrome and sleep apnea syndrome.

Snoring is the most evident symptom of these syndromes, but it is not a disease. Snoring is just a sound resulting from soft tissues in the upper airway vibrating during inspiration, due to the increased velocity of air, caused by the decrease in size of airway space. Almost all patients with upper airway resistance syndrome and sleep apnea syndrome snore. However, snoring patients may or may not also have sleep apnea or upper airway resistance syndromes. Prevalence of snoring is estimated in 35–40% of the population. Snoring is a sign that some type of resistance is occurring in the upper respiratory system.

Upper airway resistance syndrome (UARS) occurs when resistance of upper respiratory system increases and breathing effort crosses over from harmless snoring. The transformation of snoring to UARS can be caused by aging (muscle tone in the throat decreases in time) and weight gain. In upper airway resistance syndrome, breathing needs much effort due to the narrow upper airway, and this extra effort leads to sleep arousal, frequent nocturnal awakenings, chronic insomnia, and excessive daytime sleepiness. Apnea and hypopnea are not symptoms of this disease.

UARS and obstructive sleep apnea syndrome (OSAS) have similar etiologies and symptoms, so differential diagnosis is very important. The key differences between UARS and OSAS consist of apnea-hypopnea existence, gender, and weight differences. In UARS apnea and hypopnea are either absent or very low. UARS patients are often in average weight, but OSAS patients are generally overweight or obese. UARS affects two genders equally, but OSAS affects men more than women.

UARS treatment consists of behavior and lifestyle changes, oral-dental appliances, and continuous positive airway pressure (CPAP) therapy. When UARS is untreated or treatment is unsuccessful, it can end up developing sleep apnea syndrome.

Sleep apnea syndrome is a chronic, progressive, and life-threatening disorder which occurs during sleeping and characterized by apneic and hypopneic events, hypoxia, blood oxygen desaturation, sleep arousal, and/or awakening. Sleep apnea has three types: central, obstructive, and mixed. In central apnea respiratory muscles need extra effort because breathing is limited or completely blocked due to the obstruction in the upper airway. Mixed apnea is the combination of central and obstructive apnea. Central and mixed apnea can be treated only by medical practitioners. But in the treatment of obstructive sleep apnea, dentists have an important role [1–4].

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3. Obstructive Sleep Apnea Syndrome (OSAS)

Obstructive sleep apnea syndrome is a sleep-related breathing disorder that increases morbidity and mortality in patients, which is characterized by recurrent episodes of partial or complete upper airway obstructions and blood oxygen desaturation during sleep. This syndrome leads to severe long-term health problems as a result of the decrease in blood pressure during apnea and hypopnea episodes. Increased risk of high blood pressure, heart disease, heart arrhythmias, heart failure, and stroke can be seen in OSA patients.

OSAS prevalence is estimated at 3–7% in men and 2–5% in women. People from all ages, even pediatric and adolescent ages, can be affected from OSAS, but middle ages have the highest prevalence.

There are some definitions and indexes in order to define and classify the syndrome:

Apnea is the arousal of breathing for 10 seconds or more. It is a full obstruction of the airway when a person is asleep.

Hypopnea is a reduction in ventilation of at least 50% that results in a decrease in arterial saturation of 4% or more. It occurs due to partial airway obstruction.

Apnea index (*AI*) shows the average number of apneas and hypopneas per hour of sleep. The term respiratory disturbance index (RDI) can be used instead of AHI.

Arousal is a sudden change from a deeper sleep stage to a superficial sleep stage. It ends up in apnea and hypopnea.

Arousal index shows the average number of arousals per hour of sleep.

Minimum oxygen saturation is the minimum oxygen saturation level recorded in the whole sleep time [5–9].

4. Pathogenesis of obstructive sleep apnea

Upper airway has a great tendency to obstruction. Any pathological change that narrows the airway leads to OSAS. The pharynx is the site of upper airway obstruction during sleep. The size of the pharyngeal lumen depends on the balance between the forces that narrows and dilates the airway. The force that narrows the upper airway is the suction type, negative air pressure occurring during inspiration, and personal anatomical factors. The force that dilates the upper airway is the tension of the genioglossus and tensor veli palatini muscles surrounding the airway and tension of other small muscles attached to the pharynx. The difference between the narrowing and dilating forces are called transmural pressure. During sleep the tension of the muscles surrounding the airway decreases. The tongue and soft palate displace to the posterior wall of the oropharynx and narrows the upper airway. The airflow rate increases due to the narrowing airway dimension. The increased airflow applies more negative, suction-type

pressure to the airway lumen. The increased negative airway pressure, decreased muscle activity, and airway lumen diameter lead to upper airway obstruction and apnea. During apnea the blood oxygen desaturation decreases, carbon dioxide saturation increases, and pH decreases. This change leads to the stimulation of central nervous system chemoreceptors, and awakening occurs to end up in apneic process. When the patient awakens, the tension of the muscles increases, which ends up in the obstruction of the upper airway. Blood oxygen saturation increases, carbon dioxide saturation decreases, pH increases, and the patient falls asleep again. This sleeping, apnea, awakening cycle continues during total sleep time [3, 5, 6, 8].

5. Etiology of obstructive sleep apnea

The etiology of OSAS is multifaceted. The factors that lead to OSA can change in adults and pediatric and adolescent patients.

5.1. Adult OSAS etiology

General factors: Age, gender, obesity, alcohol consumption, sedative medicine consumption.

Anatomical factors: Lesions and anatomical variations, nasal obstruction, neck circumference, head and neck position, airway shape, and dimension.

Mechanical factors: Supine position, upper airway resistance, upper airway compliance, intraluminal pressure, extraluminal pressure, thoracic caudal traction, mucosal adhesive affects, vascular factors.

Neuromuscular factors: Upper airway dilator muscles, upper airway reflexes, dilator muscle, and diaphragm relations.

Central factors: Hypocapnic apneic threshold, periodical breathing, arousal, and cytokines.

5.2. General factors

1. Age: OSA can be seen in all ages but mostly seen in middle ages. The tissue elasticity, ventilation on central, and pulmonary and cardiovascular functions decrease with age, and those factors lead to OSAS.
2. Gender: Men are affected more than women because of testosterone hormone inhibits respiration and men have more pharyngeal and supraglottic airway resistance than women.
3. Obesity: There is a close correlation between obesity and apnea. Obese patients have more fat deposits around soft palate. The fat infiltration from lateral to upper airway decreases the airway area and leads to apnea.
4. Alcohol and sedative medicine consumption: Investigations indicate that the activity of genioglossus and tensor veli palatini muscles decreases when the patient is asleep in a supine position. Alcohol and sedative medicine consumption can exaggerate this condition; the airway area decreases leading to apnea.

5.3. Anatomical factors

1. Lesions and anatomical variations: Changes in craniofacial form, inadequate muscle activity, soft tissue anomalies, micrognathia, retrognathia, macroglossia, enlarged soft palate, steep mandibular plane angle, nasal obstruction, adenotonsillar hypertrophy, reduced airway lumen, vocal cord dysfunction, edema of the epiglottis, polyps, tumors, acromegaly, nasal obstruction, juvenile temporomandibular ankylosis, and syndromes (Franceschetti-Treacher Collins, Apert, Crouzon, Pierre Robin, and Down syndromes) may cause OSA.
2. Neck circumference: Neck circumference is a significant predictor of OSA. Men and women have a greater risk for OSA if they have neck circumferences of 17 and 16 inch or greater, respectively.
3. Head and neck position: When a patient changes from upright to supine position, the thickness of the soft palate increases, and the anteroposterior oropharyngeal cross-sectional area decreases. The decrease in cross-sectional area is most evident in posterior of the soft palate.
4. Airway lumen diameter and shape: The investigations have shown that the OSAS patients have narrower airway area even they are awake and upright position. The airway has an anteroposterior configuration in normal people, but OSAS patients' airway has horizontal configuration [1–12].

6. Symptoms of obstructive sleep apnea

Major symptoms: Snoring, apnea, excessive daytime sleepiness.

Cardiopulmonary symptoms: Systemic hypertension, pulmonary hypertension, nocturnal arrhythmia, atypical breath ache, acute pulmonary edema, reversible proteinuria.

Neurobehavioral and social: Restlessness and headache in the morning, insomnia, depression, anxiety, mood disturbances, tendency to accidents, nervousness, forgetfulness.

Other symptoms: Dry mouth, night transpiration, nocturnal cough, hearing loss, gastroesophageal reflux [1–21].

7. Classification of OSAS

The severity of OSAS can be classified into three groups according to the apnea-hypopnea index (AHI). An AHI score smaller than 5 is considered normal. An AHI score between 10 and 20 represents mild obstructive sleep apnea, AHI score of 21–40 represents moderate obstructive sleep apnea, and AHI score greater than 40 represents severe obstructive sleep apnea [10–12, 22].

8. Diagnostic methods of OSAS

OSAS is a life-threatening syndrome which affects cardiovascular, pulmonary, psychological, and neurological systems. Because of this reason, the diagnosis and treatment plan of this syndrome should be done by a multidisciplinary team. The team should consist of medical practitioners, sleep specialists, psychiatrists, and dentists. The diagnosis and treatment plan should be done after a comprehensive medical and dental history and examination [10, 12, 13, 22].

The diagnostic methods of OSAS can be done in steps:

1. Clinical examination
2. Endoscopical examination
3. Polysomnography (PSG)
4. Radiological examination

8.1. Clinical examination

a. Nose-ear-throat and head-neck inspection: Inspection of the tonsil, soft palate, and tongue; inspection of maxillofacial characteristics; inspection of craniofacial characteristics, neck circumference, and natural head position.

b. Dental inspection: Malocclusion, maxillar and mandibular deficiency, narrow maxilla and maxillar segment, and dry mouth.

c. Systemic examination: Weight and inspection of pulmonary, cardiac, and neurobehavioral functions.

8.2. Endoscopic examination

Endoscopic examination is frequently used for the inspection of upper airway from the nose to the glottis. The dynamic upper airway changes and the level of collapse in upper airway can be seen in this method. Especially before surgical treatments, the level and severity of collapse can be seen, and the operation can be planned according to these data.

8.3. Polysomnography (PSG)

Nocturnal, laboratory-based polysomnography is the gold standard diagnostic tool for obstructive sleep apnea syndrome. It is used for diagnosing, determining the severity of disease, and evaluating various other sleep disorders that can exist with or without OSAS. It is also used after OSAS treatment for the evaluation of the treatment effectiveness. A classical PSG consists of electroencephalogram (EEG), electromyogram (EMG), and electrooculogram (EOG) records. PSG measures sleep cycles and stages by recording airflow in and out of lungs during breathing, the level of oxygen in blood, breathing effort and rate, brain waves, electrical activity of muscles, eye movements, and heart rate. The most important parameters measured by PSG are apnea index (AI), hypopnea index (HI), apnea-hypopnea index (AHI), and minimum

oxygen desaturation. PSG can be done either at a sleep center or in the patients at home. But home-based PSG records do not have the same reliability as sleep center-based PSG records.

8.4. Radiological inspection

Upper airway imaging should be performed in OSAS patients in order to examine the anatomy of the pharynx, surrounding the craniofacial and soft tissue structures. Lateral cephalometry, computerized tomography (CT), and MR imaging (MRI) are the most commonly used radiological inspection methods of OSAS.

8.4.1. Lateral cephalometry

It is the most commonly used radiological inspection method of OSAS. It allows to investigate the sagittal and vertical positions of the maxilla and mandible according to the cranial base, maxillomandibular relationships, the sagittal and vertical position of hyoid bone, soft palate anatomy, and head posture. It is cheap and noninvasive but documentation can be done in two dimensions.

Cephalometric analysis alone is insufficient to diagnose OSAS. But some cephalometric parameters can accept a signal for OSAS risk. Increased mandibular plane angle, steep occlusal plane, over-erupted posterior dentition, large gonial angle, anterior open bite, adipose tissue placed in the submental and parapharyngeal region, larger and wider soft palate, and increased linear distance from the mandibular plane to the hyoid bone (a distance bigger than 15.4 mm) create OSA risk [10, 13].

8.4.2. Computed tomography (CT)

The high-resolution and three-dimensional evaluation of the skeletal system and soft tissues in maxillofacial complex can be performed by computed tomography. CT imaging is done in supine position and can give us the opportunity of recording the changes in upper airway cross-sectional area in different phases of respiration and predicting the collapse area in OSAS patients. The disadvantages of this technique are as follows: being expensive, high radiation level, and the low-resolution images of fat tissues around upper airway according to MR imaging [14].

8.4.3. Magnetic resonance imaging (MRI)

Magnetic resonance imaging allows 3D imaging and accurate measurement of the upper airway, soft tissues, and skeletal structures. MRI is performed in supine position, it allows high-resolution visualization of soft tissues and fat deposits around pharyngeal airway, and the volume and area of the airway can be measured accurately. With ultrafast mode dynamic observation can be done. But it is expensive and cannot be used in patients who have claustrophobia, ferromagnetic clips, or pacemaker [15].

9. Treatment of adult obstructive sleep apnea syndrome

OSAS is a life-threatening disease. Because of this reason, early, accurate, and individualized treatment should be performed by a multidisciplinary team. The aim of OSAS treatment is

to decrease the number of obstructive episodes and severity and the collapse tendency and increase the airway area, blood oxygen saturation, and life quality.

Treatment options in OSAS are:

1. Behavioral modification
2. Surgical treatments
3. Use of nasal continuous positive airway pressure device (nCPAP)
4. Use of oral appliances

9.1. Behavioral modification

Weight loss, quitting smoking and alcohol, changing sleep positions and head posture, and avoidance of central nervous system depressors may be beneficial for some patients. Weight loss may decrease OSA symptoms by reducing the size of the tongue and soft palate. Quitting alcohol and depressor drugs prevents the relaxation of upper airway muscles. Alteration of sleeping position can prevent the tongue and mandible moving backward and narrowing the airway. The patients are instructed to sleep on their sides rather than their backs.

9.2. Surgical treatment

OSA resulted from anatomic obstruction of the upper airway, unsuccessful behavioral therapies, and inability to tolerate CPAP or oral appliances, which are inclusion criteria for surgical treatment. Tracheostomy, tonsillectomy and adenoidectomy, genial advancement with or without hyoid myotomy, uvulopalatopharyngoplasty, laser glossectomy and lingualplasty, maxillomandibular advancement, and epiglottoplasty are the surgical techniques used for the treatment of OSAS.

9.3. Nasal continuous positive airway pressure (nCPAP) device treatment

It is a device which has a small air pump connected to either a sealed face or nose mask. The device opens the pharyngeal airway and prevents the soft tissues from collapsing and blocking the airway. It is the gold standard treatment option for moderate to severe OSAS cases, and the success rate is about 75%. However, patient compliance is poor because of the pump noise, the irritation of nasal mucosa because of the airflow to the nose, xerostomia, and poor retention. The patient non-compliance ratio was reported to range from 46 to 83%. The ordinary usage of nCPAP device for 4–6 weeks decreases the volume of the tongue and increases the pharyngeal volume. Inclusion criteria for nCPAP therapy are moderate and severe OSAS patients with AHI score greater than 20, mild OSAS patients with AHI scores between 10 and 20 but has excessive daytime sleepiness and cardiopulmonary or cerebrovascular risks, anatomical-based OSAS patients whose medical health condition is inappropriate to surgical treatment, and mild-to-moderate OSA patients who have failed behavioral modification therapy and unable to tolerate oral appliances [1–10, 16, 17].

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10. Oral appliance therapy

Oral appliance treatment was first introduced in the 1980s and is a very effective treatment option for mild-to-moderate OSAS and seven patients with severe OSA who cannot tolerate CPAP or refuse surgical therapy. The American Sleep Disorders Association reported that oral appliance therapy is the primary treatment for patients with mild OSA and a secondary treatment option for moderate to severe OSA. For severe OSA patients, reduction in AHI score occurs, but it cannot turn into the normal range. If the AHI score cannot be decreased to 20, long-term health risks will continue. Oral appliances can be successful if only they are used after the etiological factors are eliminated. Only obstructive sleep apnea can be treated with oral devices; they are not indicated for central and mixed apneas [10–12, 16–21, 23–32].

10.1. Oral appliance therapy indications

1. Snoring patients
2. Upper airway resistance syndrome
3. Mild OSAS patients who have failed behavioral modification therapy
4. Moderate to severe OSAS patients who refused or failed CPAP therapy or surgery

10.2. Oral appliance therapy contraindications

1. Central or mixed obstructive sleep apnea
2. TMJ diseases
3. Periodontal diseases
4. Insufficient oral hygiene
5. Anatomical based OSAS

11. Types of oral appliances

Oral appliances can be classified into four groups according to their affect mechanisms:

1. Mandibular advancement devices (MADs)
2. Tongue retaining devices (TRDs)
3. Palate lifting appliances (PLA)
4. OPAP appliances (oral appliances + CPAP device)

12. Mandibular advancement devices (MADs)

MADs were first described by Pierre Robin in 1934 in the treatment of a patient with micrognathia as a modified monobloc in order to reposition the mandible in a more forwarded position and open the airway. This advancement makes the attached soft tissues and tongue stretch and stabilize; by this way, oro- and hypopharyngeal airways enlarge.

MAD appliances can be divided into several groups: monobloc-style one-piece or twin block-style two-piece, available to activation or unavailable to activation, teeth-supported or teeth- and tissue-supported, and soft- or hard-materialed. All of these appliances increase the distance between soft palate and posterior wall of the pharynx and enlarge the space between tongue root and posterior region of the oropharynx.

Tooth- and tissue-supported, soft-materialed, activation-optioned MAD appliances are reported to be more successful in OSAS treatment. One-pieced or two-pieced appliance design does not affect the treatment success.

MAD-type oral appliances are found the most effective type of oral appliances in OSAS treatment [10–12, 17, 19, 23, 25–30].

12.1. MAD indications

Normal or reduced facial height, patients have at least eight teeth per arch, patients who have normal soft palate thickness and normal positioned soft palate, and obese patients.

12.2. MAD contraindications

The patients with thick and enlarged soft palate, periodontal disease, and TMJ problems.

MAD appliance construction:

a. Upper and lower dental impressions are taken.

b. Maximum opening, left and right lateral excursion, and maximum protrusion are measured.

c. The appliance is constructed using a position 75% of the patients' maximum protrusion. Vertical opening amount changes individually. But it is recommended to open the bite at least 5 or 6 millimeters.

d. Bite registrations in centric occlusion and advanced position are obtained. A George gauge and light-body impression material can be helpful in stabilizing the construction bite position.

e. The MAD appliance fabricated from soft acrylic, hard acrylic, silicone-based, or Essix-based materials according to the type of the chosen appliance.

12.3. MAD treatment mechanism

MADs displace the mandible, suprahyoid, and genioglossus muscles anteriorly. This anterior movement prevents oropharyngeal airway obstruction. The forward and downward

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displacement of the mandible also decreases the gravitational effect of the tongue in supine position and enlarges the velopharynx by stretching the palatoglossal and palatopharyngeal arches (**Figures 1** and **2**).



Figure 1. One-piece, non-activation MAD appliance.

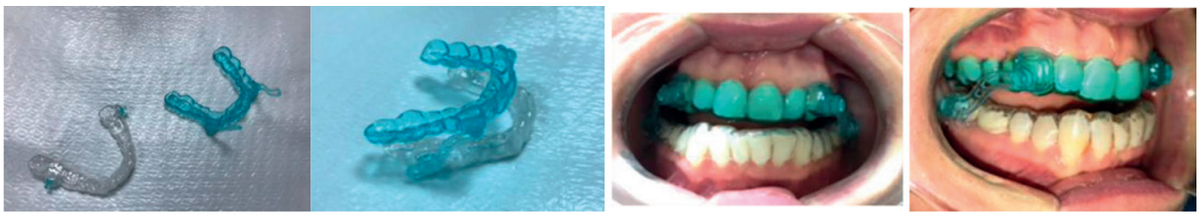


Figure 2. Two-piece, activation-optioned MAD appliance.

13. Tongue-retaining devices (TRDs)

Tongue-retaining devices were first described by Cartwright and Samelson in 1982 [28]. During sleeping and in supine position, the tongue and all gravity-dependent tissues tend to fall posteriorly. With TRDs, the tongue is prevented from dropping posteriorly by suction created when the patient forces the tongue into a hollow bulb built into the device. The forward position of the tongue increases the volume and decreases the resistance of upper airway. The superiority of TRDs over MADs is that they can be used for edentulous patients [10–12, 16, 17, 19, 20, 23, 25, 31].

13.1. TRD indications

Edentulous OSA patients, OSA patients who have TMJ disorders, OSA patients with big tonsils and large tongues, OSA patients who have less than six teeth per arch, hypothyroidism, and sleep position–related apnea-hypopnea occurrence.

13.2. TRD contraindications

Severe periodontal diseases, bruxism, chronic nasal obstruction, patients who are unable to move their tongue anteriorly, and obese OSA patients.

13.3. TRD appliance construction

The TRD appliance can be fabricated from soft copolymer materials by the technician to the OSA patient individually, or standard fabricated appliances can be used to the patient.

13.3.1. Individualized TRD appliance construction

a. Upper and lower dental impressions are taken.

b. Maximum opening, left and right lateral excursion, and maximum protrusion measured.

c. The appliance is constructed using a position 75% of the patients' maximum protrusion. Vertical opening amount differs patient to patient, but it is more than MAD vertical opening amount, in order to place the hollow bulb part.

d. The appliance is fabricated from soft copolymer material like a monobloc with a hollow bulb part to place the tongue.

13.3.2. Fabricated TRD appliance construction

a. The tongue is measured by wrapping a piece of dental floss around the tongue. Then, the floss is removed and measured.

b. The fabrication appliances have two types, edentulous and dentulous, and three sizes, small, medium, and large.

c. The correct type and size are chosen for the patient.

d. The bulb appliance is moistened and compressed and the tongue is inserted.

TRD-type appliances are found to be effective in mild to moderate OSA treatment. They decrease AI, HI, AHI, oxygen desaturation, and excessive daytime sleepiness and increase minimum oxygen desaturation and genioglossus muscle activity. But the patient compliance is low with TRDs because of the irritation in the tongue, hypersalivation, uncomfortable feeling, and non-esthetic appearance.

14. Palate lifting appliances (PLA)

They are used in patients who have a thick and enlarged soft palate. The appliance supports and stabilizes the soft palate, prevents soft palate blocking the airway, and reduces the vibration of soft palate leading to snoring. But they are uncomfortable appliances and the patient compliance rates are very low. Because of this reason, they are not frequently used in OSA treatment [10, 11].

15. OPAP appliances (oral appliance + CPAP device)

nCPAP device applies very high pressure and high airflow rate which leads to irritation of nasal, oral, and throat mucosa and decreases patient compliance. When MAD-type oral appliances are combined with CPAP device, the pressure given by nCPAP diminishes; irritation

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of nasal, oral, and throat mucosa decreases; and patient compliance and treatment success increase. The adaptation of CPAP and MAD appliance should be done by a dental sleep specialist [10, 18].

16. Side effects of oral appliance therapy

There are very few side effects of oral appliances when compared to other treatment alternatives. Researches have reported that patients prefer treatment with an OA over surgery or CPAP appliance, and compliance with OA has been reported to be 40–80%.

The side effects of OA therapy can be divided into two groups:

1. Short-term side effects of OA therapy: Excessive salivation or dry mouth, TMJ sounds and/or TMJ pain, tooth pain, odd bite feeling in the morning, gum irritation, tenderness in masticatory muscles, or myofascial pain. These complications are generally mild and transient [10, 11, 19, 20].
2. Long-term side effects of OA therapy: OA therapy for OSAS patients is a lifelong treatment. Skeletal, dental, and occlusal changes can occur from OA therapy in 2 or more years' time. Craniofacial changes related to long-term oral appliance use can be determined with cephalometric investigations. Skeletal changes generally occur after average treatment duration of 5 years. The changes can be summarized as more downward and forward position of the mandible, increased lower facial height, decreased overbite and overjet, retroclination of the maxillary incisors, proclination of the mandibular incisors, changes in molar relationship, and curve of Spee flattening. Long-term use of OA also makes differences in upper airway configuration. Palatal length decreases and pharyngeal area increases in time. These changes can be due to the loss of edema caused by snoring and repetitive apneas [10, 32–38].

17. Efficacy and success of oral appliance therapy

OA therapy is an effective and safe long-term therapy for patients with snoring, mild to moderate OSAS. However, the efficiency varies on many factors including the type of OA, materials used for fabrication, piece number of appliance (monobloc or bibloc), titration ability (titrable or untitrable), and degree of sagittal and vertical mandibular displacement.

The efficacy criteria of oral appliance therapy in OSA are:

a. Positive changes in PSG test: Decrease in AHI, AI, HI, arousal score, increase in blood oxygen saturation, sleep time, and efficiency
b. Positive subjective feedback of patients: Decrease or quitting snoring and decrease in daytime sleepiness
c. Enlargement of pharyngeal airway area [10–12, 25, 28, 31, 32]

17.1. To summarize

1. MADs are reported more effective than other types of OAs.
2. Monobloc-type one-piece and soft-materialed OAs are found to be more efficient in reducing AHI, AI, and snoring.
3. Forward and downward displacement of the mandible is recommended for enlargement in pharyngeal airway area. But this displacement amount should be assessed individually.
4. No definite conclusions can be drawn regarding which type or design of OA has a beneficial influence on subjective treatment efficacy. Individually determination of appliance is very important in OA treatment success [10–12, 16, 17, 19, 20, 23, 25–28, 30–32].

18. Intraoral appliance treatment guideline for OSAS patients

The American Sleep Disorders Association (ASDA) has prepared a protocol for OA treatment in OSAS patients. A dental sleep specialist should follow up the stages below in the treatment of an OSAS patient [21, 24]:

1. Detailed medical and dental history.
2. PSG and medical specialist's report investigation.
3. Dental investigation: Soft tissue and intraoral inspection, periodontal-occlusal-TMJ inspection, intraoral habits and parafunction inspection, teeth and restoration inspection, and radiological inspection.
4. Upper-lower dental impressions and bite registration are obtained.
5. Selection of OA type for the patient individually. (The success and compliance rate varies case to case, and choosing the right type of OA is the most important stage of OA treatment protocol.)
6. OA fabrication.
7. Adjustment of the OA. Appliance fit and comfort are controlled, and usage instructions are given to patient.
8. Follow-up PSG and lateral cephalogram are taken after appropriate appliance adjustment/titration and patient adaptation period. Objective determination of the treatment efficiency can only be observed by PSG test with the appliance in place. Nearly all patients report positive change subjectively; however, they should be proven with PSG reports.
9. If the treatment is found to be effective in the first year of the therapy, four follow-up appointments are recommended. After 1 year, two follow-up appointments per year are enough. At the follow-up appointments, appliance fit, comfort, effectiveness, and patient

compliance are investigated. If the OA is found to be less effective, the titration of OA or fabrication of a new OA should be done.

19. Pediatric and adolescent obstructive sleep apnea

Obstructive sleep apnea in pediatric and adolescent patients is characterized by episodic partial or complete upper airway obstruction during sleep. All children with OSAS snore. It has been estimated to occur in 5–6% of children. It is most seen in preschool children; the peak age is 3–6 years, which coincides with the growth of adenoids and tonsils. Pediatric OSAS is similar to adult OSAS, but there are differences. Sleep disruption occurred by respiratory pauses less than ten seconds. Hypopneic episodes can be seen, usually more than five to ten episodes per night with oxygen saturations less than 85%. Pediatric OSAS effects both gender at the same ratios, different from adult OSA. Seven percent to nine percent of children snore every night; 18% of them snore in nasal, ear, or throat infection periods. The prevalence of pediatric OSAS is estimated about 0.5–3%. AHI scores of pediatric sleep apnea is controversial. Some protocols of AHI score greater than 1 should be accepted as a pediatric OSA predictor; some protocols accepting AHI score greater than 5 is pathognomonic.

19.1. Pediatric and adolescent OSA etiology

Hypertrophic adenoids, hypertrophic tonsils, maxillary transverse deficiency, class 3 maxillary skeletal deficiency, class 2 mandibular skeletal deficiency, overweight and obesity, and craniofacial anomalies. (Pierre Robin Sequence, Goldenhar syndrome, Crouzon syndrome, Apert syndrome, cleft lip and/or palate, vertical face anomalies, Marfan syndrome, and associated 22q deletion syndromes).

19.2. OSA symptoms in children

Abnormal breathing during sleeping, frequent awakenings or restlessness, frequent nightmares, enuresis, difficult awakening, excessive daytime sleepiness, hyperactivity-behavior problems, daytime mouth breathing, poor or irregular sleep patterns, early recognition of mouth breathing and airway obstruction; symptoms of recurrent blocked nose; recurrent nasal, ear, and throat infections; parents concerned about snoring should alert the dental professional for definitive diagnosis for pediatric OSA.

19.3. Differences between pediatric and adult OSA

a. Pediatric OSAS effects both genders equally.

b. Snoring is irregular and interrupted with apneic-hypopneic events in adult OSAS. In pediatric OSAS snoring can be continuous.

c. Daytime sleepiness is mostly seen in adult OSA. In pediatric OSA behavioral changes and growth retardation are seen more than daytime sleepiness.

d. Mouth breathing is mostly seen in pediatric OSA [39–42].

e. Obesity is mostly seen in adult OSA; in pediatric OSA, the patient can be of normal weight or thin.

f. The etiology of pediatric OSA is mostly hypertrophic adenoids and tonsils. In adult OSA retropharyngeal and retrolingual pathologies are more effective.

g. The gold standard treatment protocol for adult OSA is CPAP appliance. In pediatric OSA adenotonsillectomy is advised rather than CPAP therapy.

19.4. Clinical inspection in pediatric sleep apnea syndrome

a. Nose-ear-throat and head-neck inspection: Craniofacial, orofacial, and maxillofacial characteristics are investigated. In craniofacial inspection symptoms of various syndromes are investigated. In orofacial inspection features of the tongue, soft palate, and tonsils are investigated. In maxillofacial inspection maxillofacial characteristics like facies adenoidalis, rhinolalia clausa, long-thin face, narrow maxilla and maxillary segment, overlenghtened teeth, hypoplastic mandible, and septum deviation are investigated.

b. Systemic inspection: Includes inspection of pulmonary, cardiac, physical, and mental condition functions and weight.

20. Pediatric OSA treatment protocol

1. Surgical approaches: In pediatric patients dentists should examine oropharynx carefully. Both the lingual and pharyngeal tonsils can be visible intraorally, but the adenoids will not. When hypertrophic tonsils are observed clinically or radiographically, referral for endoscopic evaluation and possible surgical removal by a pediatric otolaryngologist should be made. Early removal of these tissues can prevent the long-face growth pattern with narrow upper and lower dental arches and anterior open bite.
2. Weight loss and behavioral changes
3. Rapid maxillary expansion (RME): Cephalometric and acoustic rhinometry studies report that with RME maxilla, the palate and floor of nasal cavity expand, which leads to increase the volume and decrease the airflow resistance in nasal cavity. It also makes statistically significant changes in tongue size position and hyoid position. The expansion in maxillary dentition gives the tongue a greater space and more forward positioning to the tongue. The widened maxillary basal bone on the velum, the superior pharyngeal constrictor muscles, and the surrounding orofacial musculature can increase the muscle tonus.
4. Class 2 growth modification therapy: When the pediatric and adolescent patients have both mandibular deficiency and obstructive sleep apnea, mandibular advancement devices like Herbst, twin block, monobloc, bionator, Frankel 2, etc. can be used for the treatment.

5. Class 3 growth modification therapy: When the pediatric or adolescent patients have both class 3 maxillary deficiency and OSA, class 3 growth modification therapy is recommended. In this treatment maxilla is widened first with RPE, followed by orthopedic traction with protraction face mask (reverse pull headgear).
6. CPAP therapy: This therapy can be advised to severely affected adolescent OSAS patients. In pediatric and adolescent population, the long-term use of CPAP can cause craniofacial side effects. The force applied from the elastic strap of CPAP mask applies a restraining force on the maxilla. So, CPAP therapy can lead a class 3 skeletal deficiency in pediatric and adolescent OSA patients. But it is unclear how much of the malocclusion results from CPAP and how much results from an underlying adverse growth pattern.

21. Conclusion

Sleep-related breathing disorders are complicated problems, which decrease life quality and increase morbidity and mortality in patients.

Dentists, who are specialized in dental sleep medicine, can see the early symptoms of these diseases and can be frontline screeners for potential OSAS diagnosis. In the treatment of snoring and mild-to-moderate obstructive sleep apnea, the oral appliance therapy was found to be a very effective treatment option.

Author details

Handan Tuğçe Oğuz

Address all correspondence to: htugce@gmail.com

Private Orthodontics Clinic, Ankara, Turkey

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