Role of oral health professional in pediatric obstructive sleep apnea

Abu-Hussein Muhamad1*, Abdulgani Azzaldeen2, Watted Nezar3, Kassem Firas4

1Department of Pediatric Dentistry, Athens, Greece
2Department of Conservative Dentistry, Al-Quds University, Jerusalem, Palestine
3Center of Dentistry & Esthetics, Jatt, Israel
4Department of Otolaryngology, Head and Neck Surgery, Meir Medical Center, Sackler and Faculty of Medicine, Tel Aviv University, Israel

ABSTRACT

Obstructive sleep apnea is a sleep disorder of airflow at the nose and mouth during sleep. Patients with undiagnosed sleep apnea represent a major public health problem. Dental professionals have a unique doctor patient relationship that can help them in recognizing the sleep disorder and co-managing the patients along with a physician or a sleep specialist. Oral appliance therapy is an important treatment modality for sleep apnea patients. This article discusses the etiology, clinical features, diagnosis and various treatment options with special reference to oral appliances used for obstructive sleep apnea.

Key words: Snoring, Obstructive Sleep Apnea (OSA), Oral appliance therapy

Introduction

The dentist plays an important role in sleep medicine by examining patients during their annual or biannual dental checkup for the risk of sleep-disordered breathing. Patients reporting snoring, sleepiness, and morning headaches in the presence of obesity, large tonsils, and/or dental malformation (eg, retrognathia, deep palate, large tongue) need to be guided by dentists to see their otorhinolaryngologist, respiratory-pulmonologist, or physician, as well as a sleep medicine expert. To manage the sound and tooth damage or pain generated by bruxism, oral appliances can be used, but the dentist needs to understand when such an appliance is indicated and the risks associated with its use. In cases where surgery is indicated, maxillofacial surgeons or otorhinolaryngologists collaborate closely with dentists to provide treatment.[1-4]

*Correspondence
Abu-Hussein Muhamad
Department of Pediatric Dentistry, Athens, Greece
E-mail: abuhusseinnmuhamad@gmail.com

Dentists caring for patients with chronic orofacial pain conditions (such as TMDs) also need to understand basic sleep hygiene principles and to know when to refer patients with chronic or intractable insomnia for behavioral sleep medicine evaluation. Behavioral treatments for chronic insomnia are considered first-line interventions over pharmacologic treatment options. A subset of chronic orofacial pain patients presents with a complex psychologic overlay that contributes to their ongoing pain and disability, a combination that can be managed by sleep psychologists working in conjunction with the interdisciplinary team[5-7].

Sleep apnoea in infants was first described in 1975 in relation to sudden infant death syndrome and obstructive sleep apnoea (OSA) was described in 1976 in school children[1]. Since then there has been a significant increase in the recognition of sleep disorders in children. Despite this, there is still a paucity of data on this problem in children and also a lack of training of medical professionals to address the issue. The long term neurocognitive, metabolic and cardiovascular complications and ability to prevent these with early treatment warrant early diagnosis [8].

Sleep disordered breathing (SDB) encompasses OSA and upper airway resistance syndrome (UARS). OSA is defined as a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction, intermittent complete or partial obstruction (obstructive apnoea or hypopnoea) or both prolonged
and intermittent obstruction that disrupt normal ventilation during sleep, normal sleep patterns or both. It is agreed that an apnoea-hypopnoea index greater than 1 is abnormal in a child. The International Classification of Sleep Disorders 2nd edition (ICSD 2) defines apnoea as a cessation of airflow over two or more respiratory cycles[Table-1]. A specific time in seconds is not applicable to children as normal respirations vary from 12 breaths per minute in an adolescent up to 60 breaths per minute in a newborn. The definition of hypopnoea is more variable across sleep centers; however most agree that a reduction in airflow of at least 30 per cent is required with or without an arousal and/or oxygen desaturation of 3-4 per cent. UARS has more subtle indications on polysomnography, with increased effort of breathing (measured by increased negative intrathoracic pressure) often leading to an arousal being more prominent than apnoeas/hypopnoeas. This article reviews, SDB in children with common symptomatic presentations, and currently accepted treatment options[9-12].

**Table-1: Schematic diagram underlying the sympathetic nerve system activation associated with obstructive sleep apnea syndrome that may lead to increased cardiovascular disease risk**

![Diagram of sympathetic nerve system activation associated with obstructive sleep apnea syndrome]

**Epidemiology**

No definitive population-based study has evaluated the presence of OSAS in children. Previous studies were performed in different settings and implemented a variety of tools. Some considered regular nocturnal snoring a marker of chronic obstructive breathing during sleep. The percentage of individuals younger than 18 years who have been reported with regular heavy snoring oscillated between 8% and 12%. Subjects in other studies underwent polygraphic monitoring, but these studies were limited in terms of sample size and testing difficulties; initial studies estimated OSAS prevalence to be between 1% and 3%. More recently, many specialists have estimated OSAS prevalence to be between 5% and 6%. Although better monitoring techniques during polysomnography (PSG) have shown that more abnormal breathing events are present, the definitive data are still lacking.[1,5,6,7]

**Pathophysiology of OSA**

The pathophysiology of OSA includes factors related to upper airway anatomy, upper airway resistance and upper airway muscle function during sleep. Upper airway anatomy varies considerably among patients, so that no single finding is pathognomonic of obstructive apnea. However, narrowing of the upper airway is commonly observed, especially at the level of the soft palate and the base of the tongue [3]. Cephalometric variants of the facial skeleton have been described, including a relative retrognathia and a low position of the
hyoid bone. Soft tissue changes include a decrease in the posterior airway space, an increase in tongue volume and, in some cases, pathologic enlargement of the palatine or adenoidal tonsils [Fig.1-2].

Upper airway resistance is relatively increased in sleep apnea patients. The resulting more negative inspiratory pressure is thought to be an important factor in airway collapse and obstruction. Increased airway compliance may also contribute to airway collapse in apnea patients. Inspiratory excitation of upper airway muscles maintains patency when awake. Excessive relaxation or loss of compensatory excitation of upper airway muscles explains the propensity to collapse during sleep[1,5,9].

![Figure 1: Massive tonsils could obstruct airway](image1)

![Figure 2: Blocked airway in OSA](image2)

**Diagnosis of OSA**

Diagnosis of snoring and OSA should be based on subjective and objective evaluations. Subjective evaluation primarily includes snoring during sleep and sleepiness during the day in adults. Confirmation of existence and further monitoring of these symptoms can
be achieved by using different questionnaires such as the Epworth Sleepiness Scale (ESS). In addition, the presence of obesity, decreased ability in mental concentration, headaches, and morning mouth dryness are also important parameters that help to establish the correct diagnosis. Polysomnography (PSG) is the best method to monitor and diagnose sleep apnea and other sleep-disordered breathing problems[6-8]. PSG is a comprehensive recording of the biophysiologic changes that occur during sleep. It is usually performed in a sleep laboratory or the patient’s home using a portable device. PSGs are usually performed using four to eight channels measuring the nasal airflow, thoracic eff ort channels, electrocardiography (ECG), pulse oximetry,electroencephalography(EEG),electrooculography (EOG), electromyography (EMG), sleep positioning, and leg movements. The main outcome of a PSG test is the AHI, which represents the sleep apnea severity and reflects the average number of apnea (complete cessation in air flow) and hypopnea (partial cessation in air flow) per hour of sleep. Newer tests provide similar information by measuring alterations in autonomic nervous system using a hand glove [9,11,12].

The severity of OSA is classified by the American Sleep Disorder Association (1999) 24 on the basis of patient’s AHI into the following categories. These criteria are different for children:
  * Mild OSA, 1 to 5 AHI
  * Moderate OSA, 5 to 10 AHI
  * Severe OSA, more than 10 AHI.

Risk factors for OSA are well known and include high body mass index (BMI), age, male gender,smoking,craniofacial anomalies like micrognathia and retrognathia[1,3,7,10]. Alcohol consumption, enlarged palatine tonsils, enlarged uvula, high-arched palate, nasal septum deviation, inferiorly displaced hyoid bone, disproportionately large tongue, a long soft palate, and general decreased posterior airway space[11-12]. During the daytime, neurobehavioural symptoms such as irritability, behavioural problems, and poor concentration predominate. These result not only from hypoxia but also from sleep fragmentation[18]. In experimental models hypoxia has resulted in neuronal cell loss, which has detrimental effects on memory and cognition. A review identified studies that compared children with obstructive sleep apnoea with controls using tools such as the child behaviour checklist, Conners’ rating scale, and the child health questionnaire[19]. Although the studies provided a low standard of evidence, they showed almost universally poorer concentration, attention, behaviour, and quality of life in the group with obstructive sleep apnoea[1,6,13].

In the most severe cases the child may fail to thrive. Pulmonary complications arise from complex mechanisms that increase pulmonary vessel resistance. When this becomes persistent pulmonary hypertension and cor pulmonale can occur. Serious complications leading to death are rare[9].

**Laboratory investigation**

Polysomnogram (PSG) is considered the gold standard for the diagnosis of sleep apnea and other sleep disorders. It involves an overnight sleep in the laboratory with multichannel monitoring of multiple physiologic variables with the presence of a technician throughout the study. During the study, sleep stages and sleep continuity, respiratory effort, airflow, oxygen saturation, body position, electrocardiogram, and movements are recorded. In the analysis of the study, the number of apneas per hour is expressed as AI, which determines the severity of the OSA [1,7,8,9,11].

**Management protocol for OSA**

Dentists have recently become one of the team players in the field of sleep medicine. Oral appliances for the treatment of snoring and OSA fall into two main categories – those that hold the tongue forward and those that reposition the mandible (and the attached tongue) forward during sleep[12,13]. Having concluded that treatment with an oral appliance is indicated, the physician provides the dentist who has the skill and the experience in oral appliance therapy with a written referral or prescription and a copy of the diagnostic report[14],[Table-2]

Oral appliances have been considered as treatment option for upper airway obstruction caused by mandibular deficiency since the early part of this century. In the 1980’s dentists and orthodontics teamed up with pulmonologists to explore the use of oral appliances to treat patients with OSA[15].

Conservative therapies include weight loss, changes in sleep posture, placement of introradial devices, nasal continuous positive airway pressure (CPAP), and drug therapy. Surgically methods include tracheostomy, uvulopalatopharyngoplasty (UP PP), nasal septoplasty and surgical mandibular advancement[14,15].
Table 2: Treatment Protocol for Oral Appliance Therapy

![Diagram of Snoring and Sleep Apnea]

**Treatment**

**Non specific therapy**
Behavior modification include changing the sleep position from the supine position to the side position; this can be accomplished by placing a tennis ball in the centre of the back of their dress or by positioning a pillow such that they cannot roll on to their back (positional training). The avoidance of alcohol and sedatives; they may also act as muscle relaxants, reducing airway patency. In obese patients, weight loss should be recommended. Even a 10% weight loss can reduce the number of apneic events for most patients[16].

**Specific therapy**

**Oxygen administration**

Oxygen is sometimes used in patients with central apnea caused by heart failure. Oxygen at the correct flow rate when used in conjunction with nasal continuous positive airway pressure (CPAP), however, in many cases corrects this problem[18,19].

**Continuous positive airway pressure appliance (CPAP)**

Continuous positive airway pressure is the gold standard in treatment. CPAP treats patient by pumping room air under pressure through a sealed face mask or a nose mask through the upper airway to lung, however the treatment is associated with poor patient compliance due to lack of portability, pump noise, dryness of the airway passage, and mask discomfort. CPAP is administered at bedtime through a nasal or facial mask held in place by velcro straps around the patient’s head. The mask is connected by a tube to a small air compressor. The CPAP machine sends air under pressure through the tube into the mask, where it imparts positive pressure to the upper airway. This essentially “splints” the upper airway open and keeps it from collapsing in the deeper stages of Rapid eye movement sleep. The pressure acts much in the same way as a splint, holding the airway open[20,21].
Pharmacological agent

Thyroid hormone supplementation might lead to significant correction of the apnea if this is the sole problem. Control of blood sugar levels has a moderate effect in controlling the diagnosed obstructive sleep apnea. Certain medications which increase respiratory drive help some patients[20,22,23].

Surgical correction

Uvulopalatopharyngoplasty, partial tongue resection tracheostomy, lingual plasty, genioglossal advancement with hyoid myotomy and suspension nasal septal surgery hyoid bone suspension, and mandibular advancement osteotomy are the various surgical modalities suggested for obstructive sleep apnea. In nasal, septal and adenoid surgery weak or malpositioned cartilages around the nostrils, droopy nasal tip or excessively narrow nostrils, nasal septal deviation and enlarged adenoids are all indicated for surgical interventions[24,25].

Tonsillectomy allows the removal of redundant tissue and hence increases the caliber of the throat thereby reducing blockage to breathing. Genioglossus tongue advancement procedure produces a larger space between the back of the tongue and the throat thereby creating a wider airway. Uvulopalatopharyngoplasty (UPPP) involves the removal of part of the soft palate, uvula and redundant peripharyngeal tissues, sometimes including the tonsils. Laser- assisted uvulopalatoplasty like UPPP may decrease or eliminate snoring but not eliminate sleep apnea itself. Maxillomandibular advancement or double jaw advancement is a procedure where the upper and lower jaws are surgically moved forward. Radiofrequency tissue volume reduction (RFTVR) is a surgical method which uses radiofrequency heating to create targeted coagulative submucosal lesions resulting in shrinkage of the inner tissues leaving the outer tissues intact[26,27]. Hyoid suspension is a procedure which was developed specifically for the treatment of OSA. The operation advances the tongue base and epiglottis forward, thereby opening the breathing passage at this level.

In Tracheostomy any area of blockage to breathing, from the nose to the voice-box, is bypassed by a hole placed into the windpipe[28,29].

Oral appliance therapy

Orthodontic appliances are made in such a manner that it can be worn permanently or removeably depending upon the condition. Appliance are designed to bring the mandible and tongue forward, opening up the lower pharynx to allow unrestricted breathing [1,3,30,31].[Fig.3] Oral appliance are indicated for use in patients with primary snoring or mild OSA who do not respond or are not appropriate Candidates for treatment with behavioral measures such as weigh loss or sleep position change; Patients with moderate to severe OSA should have an initial trial of nasal CPAP because greater effectiveness has been shown with this intervention that with the use of oral appliances; Oral appliances are indicated for patients with moderate to severe OSA who are intolerant of, or refuse treatment with, nasal CPAP, oral appliances are also indicated for patients who refuse or who are not candidates for tonsillectomy and adenoidectomy, cranial facial operations or tracheostomy[32-34]. [Fig.4-5]

![Figure 3: Obstructive Sleep Apnea osa appliances](image-url)
Dental devices include tongue retaining devices (TRD) and mandibular advancement appliances (MAA). Tongue retaining device is a splint that holds the tongue in place to keep the airway as open as possible [Fig.6]. Mandibular advancement device (MAA) is by far the most common type of dental appliance in use today. It protrudes the mandible forward, thus preventing or minimizing upper airway collapse during sleep. Mechanism of action: Oral appliances are worn only during sleep and they help to maintain an open and unobstructed airway by repositioning or stabilizing the lower jaw, tongue, soft palate, or uvula. Mandibular advancement devices – as mentioned earlier – are the first use of mandibular advancement devices was suggested by Pierre Robin in 1903. It protrudes the mandible forward, thus preventing or minimizing upper airway collapse during sleep [Fig.7]. Currently available appliances: First category: one piece appliance with no ability to advance the mandible incrementally. Second category: Appliance are principally two piece in design and offer the potential for incremental advancement. Third category: They permit incremental advancement and lateral movement of mandible. Tongue retaining devices: Tongue retaining device is a splint that holds the tongue in place to keep the airway as open as possible. They are excellent devices for patients with Tranemomandibular sensitivity. There are several advantages of tongue retaining devices, they do not require retention from dentition, minimal adjustments are required and cause minimal sensitivity to teeth and temperomandibular joint [1, 9, 30, 32] [Fig.8].
Significant reduction in apneas for those with mild-to-moderate apnea, they may also improve airflow for some patient with severe apnea, improvement and reduction in the frequency of snoring and loudness of snoring in most patients and higher compliance rates than with CPAP[1-9].

Mandibular advancement splints generate reciprocal forces on the teeth and jaw that can result in acute symptoms, as well as long-term dental and skeletal changes. While mandibular advancement splints are primarily attached to the dental arches, most extend beyond these and thus apply pressure to the gums and oral mucosa. The incidence of reported side effects and complications vary significantly between studies. This is probably due to difference in the type of oral appliance used, the design of the oral appliance, the degree of mandibular advancement, as well as the frequency and duration of follow-up. During the acclimatization period, it is common for adverse effects to develop, which are usually minor and self-limiting. These include excessive salivation, mouth dryness, tooth pain, gum irritation, headaches, and temporomandibular joint discomfort. Patients should have regular visits with a health professional to check the devices and make adjustments[27-29].

Future randomized controlled trials are needed to compare the effectiveness of different types of appliances and different design features (eg, the amount of vertical opening). The effect of oral appliances on excessive daytime sleepiness and performance must be determined with objective and validated tools. The precise indications, complication rates, and reasons for treatment failure must be determined for each oral appliance if it is going to be used in clinical practice[18]. Ongoing refinements of appliance design eventually may lead to improved treatment outcomes. Only when the mechanisms of action of oral appliance therapy are fully understood can more effective appliances be developed.
On the horizon for the field of oral appliance therapy is the introduction of a compliance monitor that will allow an objective determination of appliance usage. Several investigators also are developing systems that would allow overnight titration of oral appliances in the sleep laboratory. This might ultimately shorten the time from initiation of oral appliance therapy to optimization of the appliance[21-25].

According to Brouillette and colleagues,[25] increasing awareness of OSA and examination of sleeping patients should result in earlier treatment and less morbidity for infants and children with OSA. As adenotonsillar hypertrophy is one of the main causes of OSA among children, investigating the prevalence of OSA among children with adenotonsillar hypertrophy is an important research task. There is evidence that physicians may not always recognize childhood OSA.

According to Konno and colleagues an average delay of 23 months occurred between identification of pediatric patients with large tonsils and their referral to a sleep clinic.[Table.3]

Among the physicians treating children, dentists are most likely to identify adenotonsillar hypertrophy; thus, it may be in the patient’s best interests if dentists act as “gatekeepers” in identifying children with adenotonsillar hypertrophy. As discussed above, dentists are becoming increasingly aware of sleep apnea in adults, as some are involved in using OAs to treat this disorder. Once dentists identify children with adenotonsillar hypertrophy, they should inform the parents about the risk of OSA and further inform their family physician about the importance of sleep assessment in children with enlarged tonsils. Involvement of dentists in this process can contribute significantly to the health of patients, as OSA, with such significant developmental consequences, can be diagnosed and treated at an early stage, preventing later problems and complications [19, 23, 25,35].

Table-3: Management of Obstructive Sleep Apnea
Conclusion

The dentist has the benefit of an optimum view of the throat of the patient and can therefore determine if it is anatomically blocked and classify according to the Mallampatic classification. The dentist can observe if the patient displays any of the signs that produce snoring, can determine via clinical history (20) or in and oral anamnesis whether the patient snores and often the dentist can ask the patients partner or spouse, which will leave little doubt as to the presence of snoring. Often the patient or his/her partner will inform the doctor without prompting. How many times in social situations do we hear complaints or comments related to snoring? There exists a demand for the services of the dental surgeon who can treat snoring and mild to moderate apneas. In the general public, there is increasing awareness of the field of sleep medicine and particularly as related to obstructive respiratory problems. This growing awareness is particularly true among medical surgeons. Unfortunately, there are few dentists who are appropriately trained to respond efficiently and ethically to this need and it is therefore, important to increase their numbers and distribution throughout the country. What is also needed is that dentist play a greater role in the field of sleep medicine.

References


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