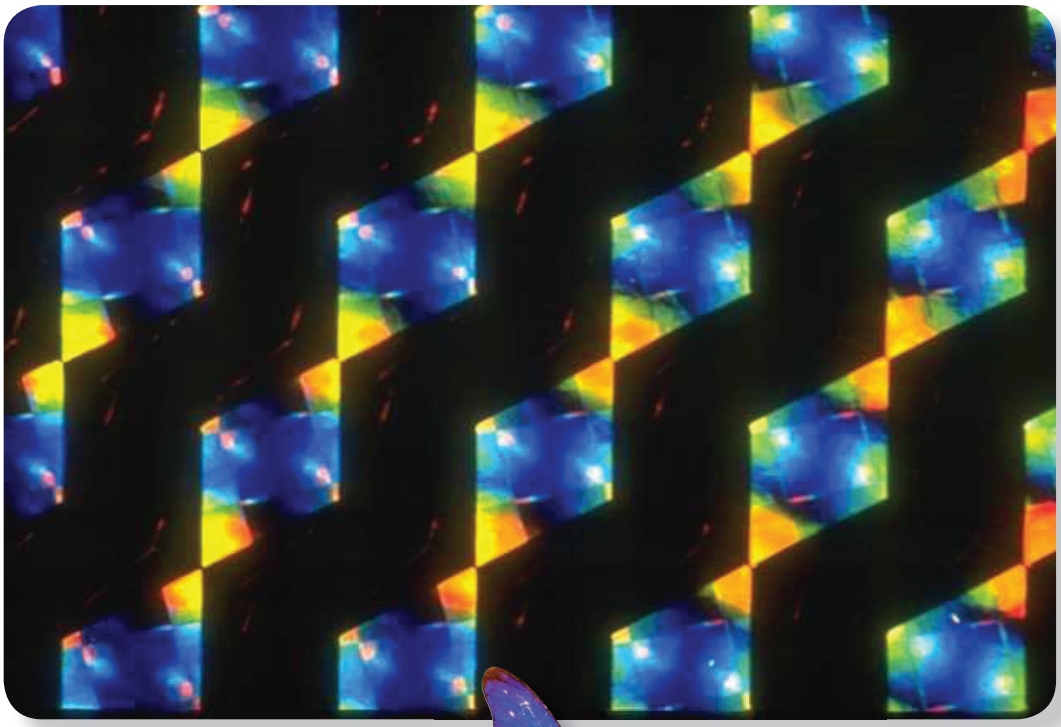
Australian Society of Orthodontists

University of Sydney



Obstructive Sleep Apnea

Creating Brighter Futures

Obstructive Sleep Apnea (OSA)

Introduction

Obstructive Sleep Apnea (OSA) effects 24% of adult males[1], 9% of adult females[1] and 24% of children[2] . OSA is characterised by intermittent, cessations or reductions of airflow, with or without obstructions of the upper airway[3], associated with oxyhaemoglobin desaturation[4], nocturnal fragmentation[5] and arousals[6], all of which can lead to excessive daytime sleepiness (EDS) in adults and the more serious Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS). OSAHS effects 4% of adult males, 2% of adult females[7] and 2% of all children[8].

Airflow obstructions are known as apneas and hypopneas. Both apneas and hypopneas last for 10 seconds or longer. An apnea is characterised by a complete cessation of airflow. Hypopnea, in contrast, is characterised by a 50% reduction in airflow accompanied by at least 4% reduction in airflow saturation. The Apnea Hypopnea Index (AHI) is the total number of apneas and/or hypopneas per hour slept, whilst the Respiratory Disturbance Index (RDI) includes apneas, hypopneas and other respiratory disturbances and so may appear higher than the AHI.

Predisposing Factors

The predominant risk factors for OSA include the following[10]:

1. Obesity, particularly upper body adiposity;
2. Increased pharyngeal soft or lymphoid tissue including tonsillar hypertrophy especially in children;
3. Male gender in adults;
4. Aging[11];
5. Craniofacial abnormalities including mandibular/ maxillary hypoplasia;
6. Nasal obstruction;
7. Endocrine abnormalities: hypothyroidism, acromegaly; and
8. Family history.

Does snoring indicate OSA?

Although snoring is a cardinal symptom of OSA, healthy adults also snore[12].

Is there a link between bruxism and OSA?

There have been suggestions that there is an association between sleep bruxism and breathing disorders, however their relationship is still controversial. A recent epidemiological study showed OSAHS was the highest risk factor for bruxism during sleep (odds ratio of 1.8)[13]. However other studies did not find marked breathing disorders or pathological levels of oxygen desaturation in bruxing patients[14].

Pathophysiology of Adult OSA

The pathophysiology of OSA is complex. Generally, in the presence of an anatomically compromised, collapsible airway, the sleep-induced loss of compensatory tonic input to the upper airway dilator muscle motor neurons leads to collapse of the pharyngeal airway[3]. Hypopneas and apneas increase in duration and are associated with more pronounced hypoxemia during rapid eye movement (REM) compared with non-REM sleep in OSA[11]. The apneas are terminated by an arousal, with upper airway muscle activation and restoration of upper airway patency.

You may wish to share this issue of Brighter Futures with your hygienists and other staff members.

Diagnostic criteria for OSAHS and OSA

For a diagnosis of OSAHS the individual must fulfill either criterion A or B, plus criterion C[10].

A. Excessive Daytime Sleepiness that is not better explained by other factors;

B. Two or more of the following that are not better explained by other factors:

-choking or gasping during sleep,

-recurrent awakenings from sleep,

-unrefreshing sleep,

-daytime fatigue,

-impaired concentration; and/or

C. Overnight sleep study with a polysomnography that demonstrates five or more obstructed breathing events per hour during sleep

OSA only requires fulfillment of criterion C.

Typically the severity of adult OSA can be defined below[10]

CATEGORY	AHI
Mild	5-15
Moderate	15-30
Severe	>30

In contrast, children with an AHI of 1-5 is indicative of OSA[15].

Consequences of OSA

The morbidity and mortality costs of untreated OSA are significant. The morbidity of OSA relates principally to the cardiovascular system. OSA patients have significantly higher risk of diabetes, hypertension, and left ventricular hypertrophy. There also has been an increased incidence of self-reported cardiovascular disease (coronary heart disease, heart failure, and stroke)[16]. Other consequences of sleep apnea include EDS, cognitive impairment, and an increased automobile accident rate. The patients' relative risk to have an accident lies between 2.3 and 7.3 times that of nonapneic individuals[4]. OSA associated vascular mortality rates have been shown to be 6.3 per 100 patients over a five year period[17]. OSA affects mainly middle-aged individuals, resulting in increasing health costs and loss of working days. It has been estimated that the additional health care costs of OSA in the USA are $3.5 billion per year.

Paediatric Aspects of OSA

There does not seem to be a sexual predilection of OSA in children[18]. Snoring, the hallmark symptom of OSA in the paediatric population has been reported to range from 8-27%[19]. However to date, studies of children, would suggest that benign snoring does not progress to OSA with increasing age[20]. In the paediatric patient, the major morbidity involves neurobehavioral aspects, cardiovascular aspects and somatic growth. Unlike adults with OSA, the majority of children with OSA do not report EDS as a major symptom. Up to 25% of parents of children with OSA describe hyperactivity and behavioural problems[8].

Children with adenotonsillar hypertrophy form the largest group of children with OSA[21]. However, the increasing prevalence of obesity may be leading to the emergence of a new at risk population in middle childhood and adolescence[22]. Typically the first line of treatment is the surgical removal of the enlarged tonsils and /or adenoids with reported success rates of 80%[19]. In 31 children, Rapid Maxillary Expansion (RME) in children without adenotonsillar hypertrophy was successful in achieving AHI of less than 1[23]. RME may also be successful in treating nocturnal enuresis in children, however sample sizes in these studies were small[24, 25].

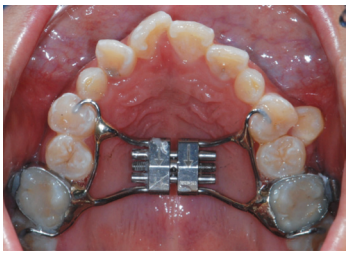


Figure 1: Rapid Maxillary Expander

Treatment of OSA

Although there are medical, dental and surgical treatment options, lifestyle modification, including weight loss in all obese patients, is highly essential for the long term success of treating OSA[26], as obesity is one of the predisposing factors. Additionally, avoidance of alcohol or sedatives and avoidance of supine sleep position in supine related OSA could be helpful in OSA management.

Currently the first line therapy in OSA is Continuous Positive Airway Pressure (CPAP). CPAP produces similar results to those of a tracheostomy in reversing excessive daytime somnolence and the cardiopulmonary sequelae of OSA[27, 28]. The first reported use of CPAP for OSA in adults was by an Australian, Colin Sullivan, in 1981[29]. The success rates of CPAP are reported to be 95%[27]. CPAP rarely results in serious side effects, however about 25% of patients may develop nasal congestion with chronic use[30]. The more common complaints about the device pertain to the noise and bulk of the machine, and also adverse effects of the nasal mask such as air leaks or ulceration of the bridge of the nose. Snoring and daytime sleepiness were relieved in more than 65% of the subjects and sleep quality improved in 75%[30]. However, compliance rates with CPAP range from 65 to 80%[30] and objective compliance monitoring has shown that average usage is less than 50% of the night[31].

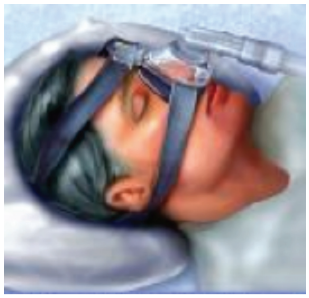


Figure 2: Continuous Positive Airway Pressure

There is insufficient evidence to recommend the use of drug therapy in the treatment of OSA[32].

Various forms of surgery including uvulopalatopharyngo-plasty (UPPP), maxillomandibular advancement (MMA), hyoid separation, bariatric surgery, and tracheostomy have been used to assist in the management of OSA. Substantial and consistent reduction in the AHI have been found following MMA, in cases where the surgeries are indicated, however other pharyngeal surgeries were less consistent[33]. The American Academy of Sleep Medicine have published surgical guidelines for surgery and OSA[33].

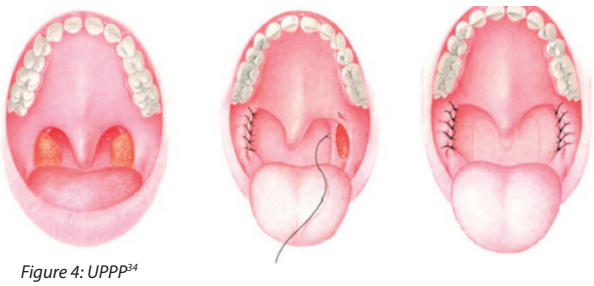


Figure 4: UPPP[34]

Brighter Futures is published by the Australian Society of Orthodontists (NSW Branch) Inc. in conjunction with the Orthodontic Discipline at the University of Sydney.

The newsletter is intended to help keep the dental profession updated about contemporary orthodontics, and also to help foster co-operation within the dental team.

Without the generous support of Henry Schein Halas and Colgate, who are an integral part of the dental team, this publication would not be possible.

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Dental appliances have played a significant role in patients who refuse or are unable to tolerate CPAP. The most widely used appliances include a mandibular advancement splint (MAS) and a tongue stabilizing device (TSD). MAS success rates vary between 50-80%[35]. Generally MAS holds the mandible at 75% of the patient's maximal protrusion[36], however greater protrusion may achieve higher success rates. Predictors of responsiveness to MAS still require further research, however remote controlled titrations of mandibular advancement measured with respiratory events during sleep studies show promise[37, 38]. Ninety percent of the patients reported subjective reductions of snoring and apnea (reduced 50% or more from baseline values)[39].

Figure 5:
Somnomed Mandibular Advancement Splint

Side effects of MAS are mild but well tolerated by most patients. Side effects after 1 to 2 years include mucosal dryness (80%), tooth discomfort (59%), hypersalivation (55%), a slight mean reduction of overbite of less than 1mm and a significant reduction of the overjet.[40] There are no recorded adverse effects on the TMJ after 5 years of nocturnal use of the MAS[39,41] and a significant decrease in headaches was reported[39]. Objective testing showed the MAS and TSD had similar efficacy in terms of AHI reduction[42]. However better compliance and a clear preference for MAS over TSD has been seen[42]. The TSD has been proposed as an option for patients with a reduced number (less than 10 teeth per jaw) or absence of teeth, compromised dental health (periodontal disease), gag reflex or those not wanting the dental side effects of a MAS.

Figure 6:
Tongue Stabilising Devices

Treatment Outcome

Any treatment of OSA should be followed up with a polysomnography to confirm that the OSA has been adequately managed. In the scientific literature, the definition of a successful treatment of OSA can be variable. Generally, complete success can be defined as a resolution of symptoms plus reduction of the AHI to less than 5 per hour. Partial success is defined as improved symptoms plus a greater than 50% reduction of the AHI, but the AHI still remaining greater than 5 per hour. Treatment failure is defined as ongoing clinical symptoms and 50% or less reduction of the AHI.[36]

Conclusion

OSA is a medical condition which may be associated with severe morbidity and increased mortality. The dental profession has a responsibility to ask appropriate questions as part of their routine check up, however the ultimate management of OSA needs to be directed and coordinated by the Sleep Physician. Dentists and Orthodontists have a role in the management of OSA with the increasing use of dental devices especially in patients who fail a trial of CPAP.

REFERENCES AVAILABLE UPON REQUEST