Sleep Apnea Management for the Dentist

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- The author reports no conflicts of interest associated with this course.

Introduction – Sleep Apnea

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Course Contents

Overview

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Learning Objectives

Upon completion of this course, the dental professional should be able to:

- Understand the epidemiology and pathophysiology of obstructive sleep apnea (OSA).
- Utilize the STOP BANG mnemonic for assessing OSA risk.
- Stratify patient care and OSA severity based on the apnea hypopnea index (AHI).
- Recall the gold standard in the diagnosis and management of OSA.
- Walk through the process of prescribing and delivering a mandibular advancement device for OSA.

Glossary

apnea – A cessation of breathing attributed to complete airway obstruction during sleep. Specifically, ≥ 90% reduction in airflow for ≥ 10 seconds.

apnea-hypopnea index (AHI) – the combined number of apneic or hypopneic events recorded per hour of sleep during a sleep study.

continuous positive airway pressure (CPAP) – a bedside device that delivers room air to treat a patient with obstructive sleep apnea via a hose-mask assembly to the patient's nose, mouth or both. The stream of air is continuous. This term is often used interchangeably and loosely with PAP (see below).

home sleep apnea testing (HSAT) – a sleep study via a device worn in the patient's own home bedroom.

hypopnea – Involves episodes of labored breathing or a low respiratory rate that does not meet metabolic needs and is attributed to partial obstruction of the airway. Defined as ≥ 30% decrease in airflow for < 10 seconds. Must include a decrease in respiratory effort and at least one of the following: a reduction in oxygen saturation by 3-4% or an arousal.

mandibular advancement device (MAD) – also known as a mandibular advancement splint (MAS), mandibular repositioning device (MRD), oral appliance (OA).

obstructive sleep apnea (OSA) – A result of recurrent episodes of complete or partial blockage of the upper airway despite respiratory effort.

polysomnograph (PSG) – an overnight sleep study, customarily refers to one at a sleep

laboratory facility. Also referred to as a polysomnogram.

positive airway pressure (PAP) – a bedside device that delivers room air to treat a patient with obstructive sleep apnea via a hose-mask assembly to the patient's nose, mouth or both. PAP devices can deliver air in a continuous manner or in an adjustable manner dependent on patient's inhalation/exhalation pattern, e.g. bilevel and adaptive-servo ventilation (ASV).

Introduction

Approximately one-fourth to one-third of life is spent sleeping or in bed. Sleep has been a mysterious black box that has only recently been extensively studied within the past two decades. While intuitive, sleep has been identified as being important for both physical and mental well-being.[1,2] Disrupted sleep has been implicated in pain syndromes in general and even in temporomandibular disorders.[3] The hypothalamus regulates sleep and circadian rhythms with the ventrolateral preoptic (VLPO) nucleus playing a key role.[4] Sleep disorders can be divided into different categories:[5]

- Insomnia
- Sleep breathing disorders
- Sleep movement disorders
- Parasomnias
- Circadian rhythm disorders
- Hypersomnia

Sleep Apnea and Relevant Sleep Disorders

The most common sleep disorder is insomnia. Insomnia represents difficulty in initiating and/or maintaining sleep along with daytime tiredness and impairment. Of note to the dentist, sleep bruxism would fall under the umbrella of a sleep movement disorder.

The topic at hand is sleep apnea. Sleep apnea is classified under sleep breathing disorders (SBD). These disorders range from basic snoring to sleep apnea-hypopnea syndrome (SAHS). While snoring may not always present with adverse effects, SAHS is associated with daytime sleepiness and cognitive issues and an increased risk of developing health issues. Central sleep apnea is a form of SAHS which features a diminished central nervous system drive to breathe. Conversely, obstructive sleep apnea (OSA) implies a mechanical impedance to upper airway airflow. It is in this OSA arena that the dental profession has expanded its service line. While there are several ways to manage OSA, one of the management strategies involves use of a specialized oral appliance.

OSA is a result of recurrent episodes of complete or partial blockage of the upper airway despite respiratory effort. Apnea is a cessation of breathing attributed to complete airway obstruction during sleep. Hypopnea involves episodes of labored breathing or a low respiratory rate that does not meet metabolic needs and is attributed to partial obstruction of the airway.

An obstructive apnea or hypopnea event, by definition, lasts at least 10 seconds and meets one of the two following features (AASM Task Force, 1999):

1. Substantial reduction in airflow (> 50%) relative to a baseline of the preceding two minutes.

2. Moderate reduction in airflow (< 50%) with blood oxygen desaturation (> 3%) relative to a baseline of the preceding two minutes or an electroencephalographic evidence of cortical arousal.

Per the American Academy of Dental Sleep Medicine (*www.aasm.org*), apnea and hypopnea can be further defined:

- Apnea is reported when there is ≥90% reduction in airflow for >10 seconds.
- Hypopnea is scored when all of the following criteria are met:
  1. The peak signal excursions drop by ≥30% of pre-event baseline using nasal pressure (diagnostic study), PAP device flow (titration study), or an alternative hypopnea sensor (diagnostic study).
  2. The duration of the ≥30% drop in signal excursion is ≥10 seconds.
  3. There is a ≥3% oxygen desaturation from pre-event baseline and/or the event is associated with an arousal.

A complete scoring of a polysomnogram is beyond the scope of this presentation. Interested readers are encouraged to review resources available at the AASM website.

MEDICARE scores OSA differently.

The AASM Scoring Manual recommended definition requires that changes in flow be associated with a 3% oxygen desaturation or a cortical arousal, but allows an alternative definition that requires association with a 4% oxygen desaturation without consideration of cortical arousals.

Sleep Architecture

Sleep is managed by homeostatic and circadian rhythmic mechanisms. By homeostatic mechanisms, sleep pressure builds up analogous to an inflating balloon throughout the course of the day. By nightfall, sleep pressure builds (balloon inflated) and there is a physiological urge to sleep. Circadian rhythms are repetitive, cyclical changes occurring over a 24-hour period. Hypothalamic nuclei such as the previously mentioned VLPO along with suprachiasmatic nucleus maintain our "biologic clock." Sunlight and darkness both influence this biologic clock. Typically sleep duration is between 6 to 9 hours for most adults.

The sleep cycle consists of rapid eye movement (REM) sleep and non-rapid eye movement (NREM) sleep. There are 4-6 NREM-REM cycles alternating approximately every 90 to 110 minutes. The first 2-3 cycles feature longer and deeper periods of NREM, while the final 2-3 cycles feature increasing REM periods with shorter and lighter (i.e. less deep) NREM stages. NREM sleep is subdivided into lighter stages (stage 1, stage 2) and a deeper stage 3. It is NREM stage 3 that provides the quality of sleep needed for restoration of sleep-wake physiology and mood (Figure 1).

Epidemiology of Obstructive Sleep Apnea

OSA is a common sleep disorder gaining increased recognition and attention. OSA is prevalent in both children (1-5%) and adults (10-30%). OSA is more prevalent in men (22%) than women (17%) and the risk of developing OSA increases with advancing age and higher body mass index (BMI).[6]

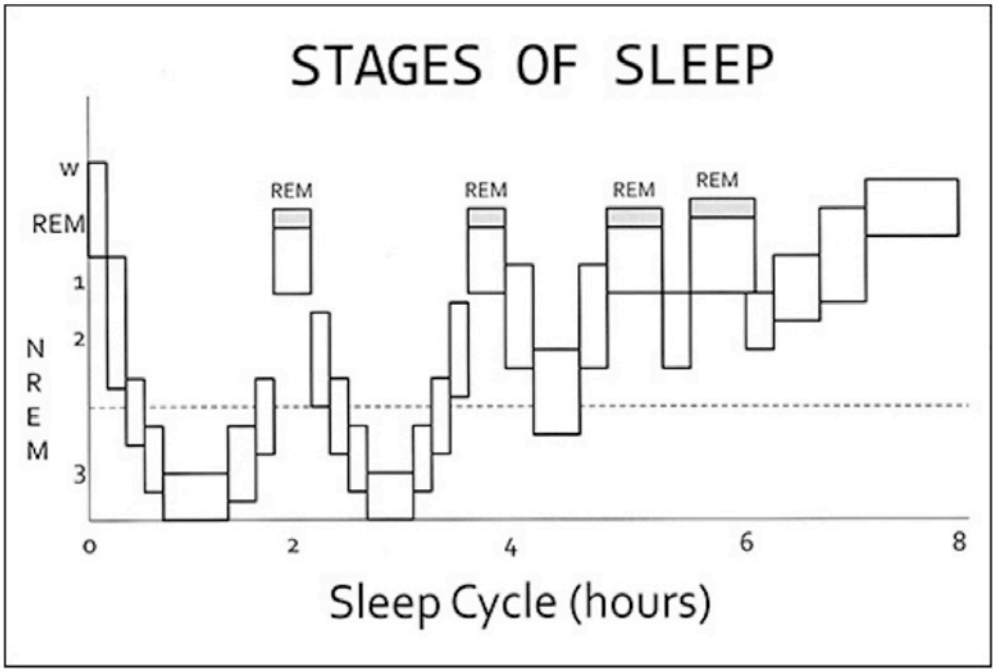


Figure 1. This chart depicts sleep staging on the vertical axis and the sleep cycle in hours on the horizontal axis. Sleep is deeper in the first half of night than the latter half. W = awake. REM = rapid eye movement sleep. NREM = non-rapid eye movement sleep.

There are predisposing risk factors for OSA which include maxillary and/or mandibular hypoplasia, macroglossia, tonsillar hypertrophy, Mallampati classification, a narrow oropharyngeal airway and endocrine disorders such as hypothyroidism. A commonly used screening tool for assessing OSA risk involves use of the STOP-BANG mnemonic.

S – Snoring
T – Tiredness
O – Observed apnea
P – Pressure; blood pressure elevated
B – BMI > 35 kg/m
A – Age > 50 years
N – Neck circumference; > 17" for males or 16" for females
G – Gender, male

- **Snoring**: does the patient snore loudly enough to be heard through a closed door or loud enough requiring a bed partner to poke or elbow the patient? Note that snoring is not synonymous with OSA.
- **Tiredness**: does the patient report excessive daytime sleepiness? Excessive sleepiness can be measured by a questionnaire tool called the Epworth Sleepiness Scale (ESS). A score of ten or greater may warrant a physician to evaluate for a sleep disorder. A positive ESS is not synonymous with OSA as other sleep disorders may also yield a positive ESS such as primary insomnia.
- **Observed apnea**: has a bed partner or roommate witnessed the patient struggling to breathe while sleeping, such as choking or gasping sounds or long periods of time between breaths?
- **Pressure**, elevated blood pressure is both a risk factor of, and consequence of OSA.
- **Body Mass Index (BMI):** increased weight is a well-known risk factor for OSA. Online or smartphone BMI calculators are available to assess this metric by entering patient height and weight.
- **Age**: individuals over the age of 50 years old have higher OSA prevalence than younger people. The soft tissue laxity of aging may contribute to oropharyngeal airway collapsibility.
- **Neck circumference**: another correlate with obesity. Neck size of 17" or more in males or 16" or more in females puts a check in this box.
- **Gender**: males have higher OSA risk than females.

The Score
OSA - Low Risk : Yes on 0 - 2 questions
OSA - High Risk : Yes on 3 or more questions

A recent study found that patients with a STOP-BANG score of 3 had a 25% probability of a severe OSA. For each additional STOP-BANG point there was another 10% increase in severe OSA probability.[7] STOP-BANG does not diagnose patients with OSA, however it should alert the health care professional to make a sleep referral and an eventual sleep study, which is the gold standard in diagnosing the presence of OSA and other sleep disorders.

Pathophysiology of OSA

The essential culprit behind OSA is that oral and/or nasal airway flow is mechanically obstructed (Figure 2). The obstruction lies somewhere at the nexus between collapsibility of the tongue posteriorly or the soft palate and upper airway muscles losing tone during sleep resulting in an obstructed airway. A frequent site of obstruction is the oropharynx, the pharyngeal space posterior to the tongue. OSA pathophysiology includes anatomic and non-anatomic (NA) factors. NA factors include impaired pharyngeal dilator muscle function, low respiratory arousal threshold (patient will be apneic or hypopneic for longer time periods), and high loop gain (unstable control of breathing). Anatomically, narrow pharynx, longer airway lengths and lumen shape are associated with collapse while asleep. There are over 20 airway muscles. The upper airway lacks bony support. The genioglossus is the largest pharyngeal dilator. As all airway muscles do, it receives central respiratory motor output along with reflex influences via chemoreceptors and mechanoreceptors. The genioglossus exhibits a phasic pattern of activation being more active during inspiration and less active during expiration. Whereas the tensor palatini muscle exhibits tonic activity constantly throughout the breath cycle. In OSA, upper airway collapsibility (Pcrit) ranges from -5 to +5 cm H_2O. Pcrit near +5 indicates a high chance of airway collapsibility.[8]

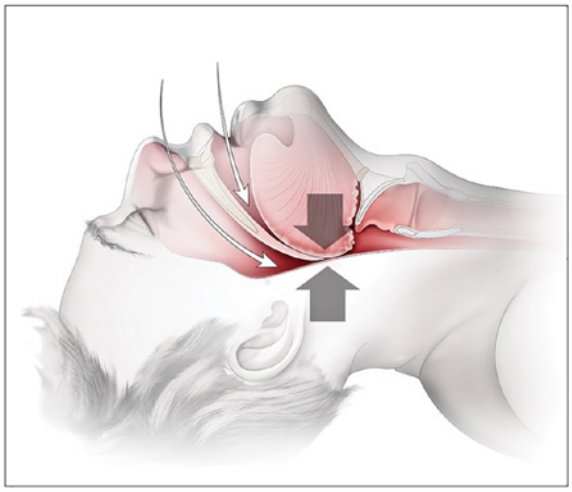


Figure 2. Depiction of the essential pathophysiology in obstructive sleep apnea. The airflow through the oral and nasal cavities (white arrows) are inhibited from reaching the lungs due to mechanical obstruction of the upper airway at the base of the tongue and posterior soft palate (gray arrows).

When airflow is prevented from reaching the lungs, there is a corresponding drop in oxygen saturation within the blood. This can be measurable by a pulse oximeter and is one component of an overnight sleep study. Repeated airway obstruction and the ensuing oxygen desaturation forces the central nervous system to take action. A patient with OSA will be displaced from deeper stages of sleep in order to breathe. These are not full awakenings but have been termed microarousals. A repetitive train of microarousals prevents the patient from enjoying the deeper, more restorative stages of sleep. As sleep architectures deteriorates, OSA patients will report poor sleep quality despite sleeping a lot (sleep quantity).

OSA Consequences and Pathophysiology

Unfortunately the consequences of OSA go beyond just a feeling of tiredness and the annoyance of snoring. One well-studied consequence of OSA, which is also related to aging and obesity, is cardiovascular disease (CVD). CVD may manifest as hypertension, atrial fibrillation or other arrhythmias, cerebrovascular events (stroke), transient ischemic attacks, coronary artery disease (CAD), pulmonary hypertension and even heart failure.

The hypoxia associated with OSA stimulates carotid chemoreceptors resulting in activation of the sympathetic nervous system with downstream elevations in blood pressure. Furthermore, hypoxia is followed by re-oxygenation and the development of reactive oxidative species that can lead to endothelial lesion formation inside the lumen of blood vessels leading to thrombotic lesion formation in CAD. The risk of atrial fibrillation is 5% in OSA while 1% in controls without OSA. Also, OSA leads to glucose intolerance and decreases insulin release and this may lead to the development of diabetes.[9] The OSA patient may also report diminished cognitive skills if the condition is not well managed. Also of note to men, as OSA is higher amongst them, serum testosterone is lower in OSA men.[10]

In more tangible every-day terms, drowsy driving can be as risky as alcohol impaired driving. The undiagnosed, under treated, or non-treated OSA driver presents risks when driving trucks or automobiles. Motor vehicle accidents (MVAs) attributed to drowsy driving are a common occurrence. They pose a risk of injury or death to the drivers themselves, other passengers, pedestrians, or passengers from other vehicles. Injuries and damages related to MVAs have financial and legal consequences. This information sometimes provides the extra impetus to patients who are hesitant to pursue OSA diagnostic work-ups or management.

To further illustrate the point, in 2016 the train crash at the Hoboken New Jersey train terminal was, at least in part believed to be a result of the engineer on board suffering from untreated OSA.

Fortunately, OSA is a modifiable risk factor of CVD. Treating OSA with positive airway pressure (PAP) decreases systolic BP by 2.8 mmHg. PAP is the first course of therapy in managing these health risks and mandibular advancement devices can be used in those patients who cannot tolerate PAP or have less severe forms of OSA.

The Diagnostic Process in OSA

The gold standard in diagnosis of sleep disorders including OSA is an overnight sleep study called a polysomnograph (PSG).

PSG components include, but are not limited to, an electroencephalograph (EEG), electromyography (EMG), electrocardiogram (ECG), plethysmography, capnometry and pulse oximetry. The EEG will capture brain activity to determine sleep stage. EMG will capture movement of the eyes and facial muscles such as the chin, to assess REM activity and bruxing events. ECG will check for the presence of arrhythmias. The set up for PSG involves attaching numerous electrodes, sensors and wire leads (as many as 30) to the patient (Figure 3).

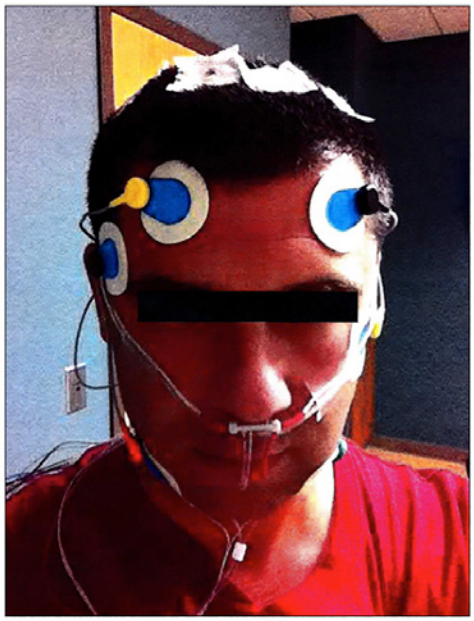


Figure 3. A patient preparing for a sleep study at a sleep laboratory.

Freedom of airflow, respiratory effort and oxygen saturation monitoring determine whether the patient is experiencing apneic or hypopneic events. Apnea is the absence of breathing in a period of time while hypopnea is diminished breathing capability within a given time. Apneic and hypopneic episodes both reduce oxygen saturation (hypoxemia), increase carbon dioxide levels (hypercapnia), induce arousals from sleep, and lead to abnormal pauses in breathing. A sleep lab will utilize PSG data to determine the average number of apneas and hypopneas per hour of sleep. This is called the apnea-hypopnea index, or AHI. An AHI of greater than or equal to 5 events per hour would be needed in order to make a diagnosis of OSA.

The severity of OSA is measured by AHI values. The International Classification of Sleep Disorders 3rd Edition (ICSD-3) in 2014 defined this as the number of obstructive apnea and/or hypopnea events per hour of sleep.[5]

Mild OSA	5 to 15 events per hour
Moderate OSA	> 15 to 30 events per hour
Severe OSA	> 30 events per hour

An AHI of 4.9 or less does not meet the threshold for diagnosis of OSA. The respiratory disturbance index (RDI) is also used in sleep medicine however its definition has varied over the years. RDI uses the same numeric range as AHI to define mild, moderate, and severe OSA; but, in addition to measuring apnea and hypopnea events per hour, RDI also includes less severe respiratory effort-related arousals (RERAs) as part of its scoring.

The Sleep Study Results

At the initial appointment with a sleep physician, he or she will conduct an interview and physical examination as needed. The PSG is usually performed on a later date. The PSG can determine if the patient is asleep, the time it takes to fall asleep (sleep latency), stage of sleep, total sleep time, sleep efficiency (total sleep time/ total time in bed), AHI score in addition to other variables. It may take several business days to receive the PSG results. A typical sleep study report may contain the following data:

Polysomnogram Report

Study date: 1/2/2019

History: The patient is a 44 year old male with a complaint of snoring, nasal congestion, witnessed apnea and daytime sleepiness (ESS=16/24). Past medical history significant for hypertension and retrognathia. Medications: lisinopril, multivitamin. Height: 70.00 inches; Weight: 185 lbs.

Technique: PSG recorded EEG, eye movement, chin EMG, airflow, microphone, chest & diaphragm excursion, leg EMG, ECG, body position and oxygen saturation. Lo-lux CCTV input was recorded digitally for later review.

Interpretation: The patient had a sleep efficiency of 72.0% and a sleep latency of 98 minutes. The overall AHI was 31.1 per hour. AHI was 37.5 in the supine position and 20.6 non-supine. The longest duration of a respiratory event was 47.0 seconds with an average of 13.9 seconds. Baseline oxygen saturation was 96% and reached a low of 88%. Tracheal microphone revealed frequent, moderate snoring. Frequent periodic leg movements were absent. ECG unremarkable.

Diagnosis: Severe obstructive sleep apnea

Home Sleep Testing

PSG remains the gold standard in the diagnosis of OSA as it is accurate and has a low failure rate. However PSG is technically complex and expensive and requires overnight laboratory staffing. Sleep labs may have variable availability in markets outside metropolitan areas. Home sleep apnea testing (HSAT) via portable monitoring (PM) units have become an attractive alternative to lab-based PSG (Figure 4). Clinical judgement is required to determine if PM would be appropriate in a given patient with suspected OSA symptoms. PM may be considered if there is suspected, high pre-test probability that the patient would likely have moderate to severe OSA and the patient is free from medical comorbid health conditions.[11] Or it may be employed in situations where PSG is not possible as in cases with patient immobility, critical illness or facility access issues. PM would not be suited for those with comorbid medical conditions and/ or in the face of other sleep disorders, and thus PSG would then be preferable

HSAT requires at least 6 hours' worth of data. Sleep testing has been classified into 4 levels based on the number of parameters it measures.[12]

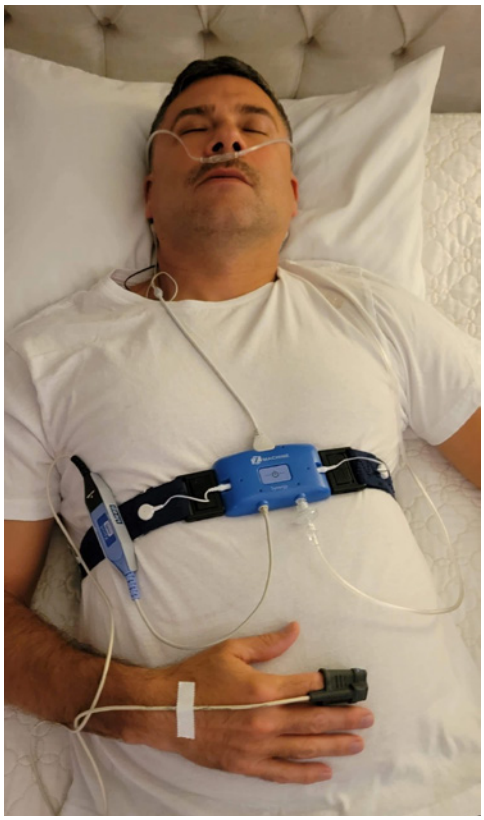


Figure 4. A patient undergoing home sleep apnea testing (HSAT).

Level 1 – Standard PSG: minimum of 7 parameters: EEG, EOG (electrooculogram), chin EMG, airflow, respiratory effort, oximetry with a technician in attendance.

Level 2 – Comprehensive portable PSG: minimum of 7 parameters: EEG, EOG, chin EMG, airflow, respiratory effort, oximetry. No personnel present. No intervention possible.

Level 3 – Modified portable apnea testing: minimum of 4 parameters from the following: airflow, ventilation, respiratory movement, oximetry and heart rate or ECG. Body position can be objectively measured. No personnel present. No intervention possible.

Level 4 – Continuous single or dual parameter recording: minimum of 1 parameter; usually either airflow or pulse oximetry which can measure oxygen saturation and heart rate. Body position cannot be measured. No personnel present. No intervention possible.

Note that level 3 and 4 PM devices cannot determine sleep staging or sleep disruption. Of interest to the dentist who provides dental sleep medicine services, according to the American Academy of Dental Sleep Medicine Portable Monitoring Task Force, PM may be used to monitor treatment response to non-PAP treatments such as oral mandibular advancement devices, upper airway surgery and weight loss.[13] Such patients must have a diagnosis of OSA already made via PSG and the raw data and report signed by a certified sleep physician. HSAT is not meant to screen asymptomatic populations and PM must be ordered by a sleep physician.[11]

Dependent on region-to-region practices, insurance reimbursement of PM devices may depend on who orders the test, i.e. sleep physician or dentist. A dentist may play a role in identifying patients with subjective symptoms and objective risk factors of OSA and make a referral to a sleep physician.

Principles of OSA Management

The degree of intervention in OSA management is based on OSA disease severity (see AHI) and mitigation of risk factors involved. Patient education is pivotal in the treatment for all forms of OSA. Health care providers should advocate for and extoll the benefits of lifestyle changes such as weight loss, maintenance of nasal patency, avoidance of respiratory depressants such as alcohol intake close to bedtime, and adjustments to sleep habits. For some patients, OSA severity is dictated by their sleep posture.

For instance, in "positional OSA," defined as an AHI two times higher in the supine position when compared to lying on one's side, could be managed with a number of simple, non-invasive treatments that encourage the patient to sleep more on their side. Sleeping in the supine position allows the tongue to fall backwards and can aggravate airway obstruction. Any change in sleep position away from the supine position should lead to improvements in AHI.

Weight loss strategies alone in OSA treatment have mixed results and may be better used as a stand-alone treatment in mild OSA without other comorbidities.[14] Combining weight loss with non-supine sleep positioning improved non-supine AHI to normal in 22% of obese OSA patients.[15]

Table 1. Treatment Options for Obstructive Sleep Apnea.

Positive airway pressure – continuous vs. automatically adjustable

Mandibular advancement device – titratable vs. fixed

Lifestyle modification – weight loss, sleep position changes

Targeted hypoglossal nerve stimulation

Surgery – orthognathic, oropharyngea

The definition of success in OSA management varies somewhat. Goalposts for success vary by different study paradigms. An example of several success goalposts include:

- Reduction of AHI to less than 5
- Reduction of AHI to less than 10
- Reduction of AHI by 50% or better

Besides just treating the numbers, improvements in sleep quality, daytime function and blood pressure would represent notable achievements.

Surgical options aim to enhance airflow through the upper airway, thereby alleviating the symptoms of OSA. A UPPP (uvulopalatopharyngoplasty) is one such procedure. Laser-assisted uvuloplasty (LAUP) and bipolar radiofrequency volumetric reduction are two others. These surgeries have yielded mixed results.[16] Maxillomandibular advancement surgeries have been shown to reduce AHI and decrease OSA severity.[17]

Principles of OSA Management, Other Therapies

A relatively new mode of OSA management under recent study is hypoglossal

neurostimulation (HS). With this therapy the hypoglossal nerve receives electrical stimulation to cause contraction of the tongue muscles, including the genioglossus, a major dilator of the pharynx. The increased activity is proposed to improve airway resistance thereby alleviating OSA. A recent study of a more targeted version of HS showed that this emerging therapy may be feasible and safe.[18] An implantable pulse generator is surgically placed unilaterally below the surface of the skin of the chest inferior to the clavicle.

The orexinergic agents modafinil and armodafinil, which are commonly used for narcolepsy, may also be used to treat residual excessive daytime sleepiness in patients already on appropriate levels of positive airway pressure treatment to promote wakefulness.[19] The benefits of adding such a medication should be balanced with its side effects.

A tongue-retained device (TRD) may be used for primary snoring or be considered in mild cases of OSA if other therapies are deemed intolerable. The tongue is inserted into the bulb which is squeezed to express air out. Negative pressure holds the tongue in a forward position thereby increasing space in the oropharynx. The TRD has not gained wide acceptance as a modality in OSA due to poor patient acceptance (Figures 5A-B).

Positive Airway Pressure for OSA Treatment

The gold standard for the management of OSA is positive airway pressure (PAP). PAP therapy involves the delivery of room air by way of a bedside machine that delivers a column of air to the patient's airway via some combination of oral or nasal mask and hood. This stream of air prevents airway collapse. The now patent airway allows for delivery of air to the patient which alleviates the symptoms and detrimental adverse effects linked to OSA.

The PAP can be delivered as a continuous air stream (CPAP) or it can be automatically adjustable (APAP). APAP devices sense when the patient exhales and decreases delivery pressure. Upon inspiration it will automatically increase pressure. The sleep physician will initially set the PAP to deliver air at a lower rate such as 6L/minute during the acclimation phase of wearing a PAP device. It may take several weeks of wearing PAP during sleep for patient's to become accustomed to it. It is not uncommon for the patient to be prescribed a hypnotic to help fall asleep during this period.

Soon thereafter the sleep physician may increase air flow rate to 9L/ minute or higher and titrate to effect. New PAP devices come with a card reader, which can send information to a secure internet cloud for the sleep center

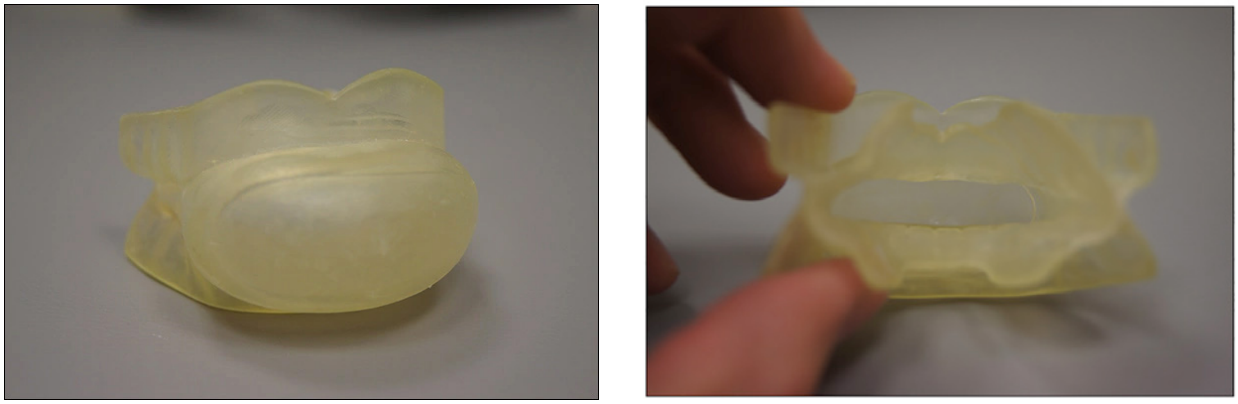


Figure 5 (A&B). Examples of tongue retained devices.

staff to monitor compliance, hours of usage and estimate a general AHI. Some companies offer a smart phone or tablet application which can download information via Bluetooth™ to the patient's device so they can monitor their own progress.

The goal of treatment is to reduce AHI to acceptable levels. As mentioned earlier, reducing the AHI 50% or more (significant improvement) or reducing AHI to under 5 constitute success. In order to show compliance, it is expected that the patient wear the PAP device for at least 4 hours a night; 21 days per a 30-day period (70% of a 30-day period). If the patient is non-compliant, the insurer may take away this service. The above compliance metric is a minimal standard. Providers should aim higher though, such as encouraging about 6 hours of nightly use. One to two months after initiation of PAP, ideally there would be a follow up with the sleep physician to check for improvements in daytime sleepiness, cognitive and other factors.

Figure 6. A typical set up of a bedside, home-use positive airway pressure (PAP) unit.

PAP Tolerance and Side Effects

Even when PAP is beneficial and successfully lowers the patient's AHI to targeted therapeutic levels, some patient's will simply not tolerate the mask and straps associated with this therapy. This intolerance can result in taking a very long time to fall asleep (long sleep latency).

To overcome this, it is suggested that the patient try wearing PAP during the daytime even if awake and just reading for a half an hour, in order to become acclimated to the PAP mask. Furthermore, a hypnotic such as zolpidem or eszopiclone may be prescribed within the first month to help facilitate the process of falling asleep while wearing the mask.

There is regular home maintenance required for PAP users. At weekly intervals, the tubes and mask need to be washed and hung to dry. At monthly or bimonthly intervals the filters need to be cleaned out or replaced. There is also an ongoing cost in that replacement tubing, filters, headgear and masks will be mailed to the patient to periodically replace used parts.

Side effects of PAP include dry mouth. Humidification and a chinstrap to help keep the mouth closed assist to a degree. Naturally, in the presence of dry mouth caries risk increases. Research on the effects of PAP on plaque, calculus build-up and periodontal disease is minimal but mixed.[20-22] Some studies have suggested there can be changes in facial morphology and maxillary incisal flaring with continued use.[23]

PAP requires a power source. Thus, there are limitations to PAP use amongst campers, deployed military personnel in austere environments, and a need for power adapters when traveling abroad.

Other PAP Considerations

There are a variety of mask options with PAP:

- Nasal mask
- Nasal pillows
- Full face mask
- Total face mask
- Oral mask
- Hybrid mask (nasal cushion with oral mask combination)

Personal preference and facial anatomy dictate which mask might be chosen. The mask should meet comfort requirements and provide a good seal to prevent air from escaping (Figure 7).

Figure 7. This patient is using PAP via a nasal mask. In order to prevent air escaping through the mouth, the mouth needs to be closed or at least nearly so. A chin strap can aid in keeping the mouth closed if the patient has a propensity to sleep with it open.

A PAP unit is considered a medical device by the Transportation Security Administration (TSA). As of this writing, a PAP device does not count against your carry-on baggage. The device may need to be removed from its carrying case prior to x-ray scanning at security check points. The PAP device however, may be kept in a clear plastic bag to prevent it from becoming dirty. Masks and tubing may remain in the case. Please check the TSA website at www.tsa.gov for updates prior to air travel.

Oral Appliances and Mandibular Advancement Devices for OSA Treatment

If PAP is the gold standard for the management of OSA, then mandibular advancement devices (MAD) can be considered the silver standard. MAD may be a first line option in those with mild to moderate OSA. MAD may even be considered in severe OSA in those who fail PAP therapy due to a compliance issue or to PAP intolerance.

The rationale behind the therapeutic effect of MAD is that the mandible is advanced (positioned anteriorly or protruded). This advancement brings the major muscles of the tongue forward, as these are attached to the mandible thereby relieving the mechanical obstruction posteriorly in the oropharynx. With improvements in airway shape, air can now flow more readily to the lungs. This mechanism alleviates OSA and snoring with concomitant decreases in the number of apneas and hypopneas.

While PAP can be used for all severities of OSA, MAD is typically used in mild to moderate cases. However, in severe OSA cases where the patient is intolerant of PAP, MAD is recommended as a back-up option.[24-26]

Oral Appliance Patient Evaluation

When considering MAD for OSA, several criteria must be considered:

- The patient is interested in MAD as an alternative to PAP
- There should be 5-6 healthy teeth per quadrant
- All necessary dental work should be treated prior to fabrication of a MAD

Initial evaluation should include an assessment of oral health to include soft tissues, the periodontium, the tongue, uvula and soft palate. Endodontic sensibility testing should be performed on any tooth with a questionable pulpal status. The patient's periodontal condition should include checking for attachment loss and any excessive mobility. Select intraoral radiographs if not current, should be obtained to rule out any dental, pulpal, or periodontal pathosis. Tonsil grade and Mallampati score should be annotated, but their severity is not strongly correlated with OSA presence or severity.

Additionally, an examination of the temporomandibular joint (TMJ) should be performed. The TMJ and its associated musculature such as the masseter and temporalis should be free of pain and should be non-tender to palpation. It is recommended to palpate with a force strong enough to make the fingernail blanch. The temporal tendon should be palpated intraorally from the retromolar pad and then proceed superiorly along the anterior ramus of the mandible. The lateral pterygoid (LP) cannot be palpated directly. Thus a LP provocation test should be performed. The dentist would place their thumb on the patient's chin and ask the patient to protrude against the thumb while the dentist provides some moderate resistance. If the patient meets the criteria for a temporomandibular disorder, then

MAD therapy may exacerbate this condition and might preclude the patient from this form of treatment.

Assessing jaw range of motion (ROM): measuring from the incisal edge of the mandibular incisors to the incisal edge of the maxillary incisors on maximum opening will give you the patient's active range of motion. A normal TMJ ROM is 35-50 mm. Some providers add the amount of overbite in mm (if present) to this number. Lateral ROM is roughly 7-12 mm (Figure 8).

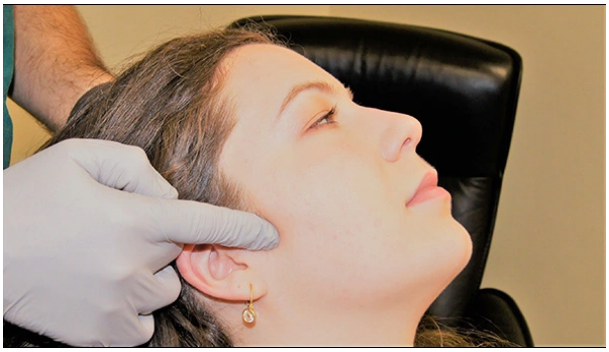


Figure 8. Digital palpation of the lateral pole of the condyle of the right temporomandibular joint to rule out the presence of pre-existing jaw pain.

If not performed at an earlier appointment, a STOP-BANG score should be obtained. The ESS should also be recorded. After MAD delivery, these inventories can be repeated to assess quality of life (QOL) changes. QOL improvements can be pointed out after MAD treatment of the patient's OSA to highlight their improvement in health. This yields a great deal of satisfaction from the patient.

Recording Maximum Mandibular Protrusion

The most important measurement is the patient's ability to protrude the jaw. The range of normal protrusive ROM is 5 to 12 mm. For MAD therapy to be successful, the patient must be able to demonstrate the ability to protrude the jaw and hold it there tolerably. For the purpose of MAD fabrication, a minimum protrusion of 5 mm is required. The measurement can be made with commercially available paper rulers, metal autoclavable rulers or any number of gauges designed for measuring protrusive capacity such as the George gauge.

Begin by measuring the degree of overjet present in millimeters. Next, ask the patient to protrude maximally and record the distance between the incisal edge of the maxillary central incisor to the incisal edge of the mandibular central incisor. Add this measurement to the amount of overjet to arrive at the maximum protrusion as measured in millimeters. Annotate this in the dental chart (Figure 9 A,B).

Figure 9A. Measuring protrusive capability by a disposable paper ruler.

Figure 9B. A commercially available protrusion gauge, such as a George gauge.

Figures 9. The process of recording maximum protrusion is crucial as this measurement will be referenced during the bite registration, laboratory fabrication and titration of the delivered mandibular advancement device (MAD).

Dental Impressions/Scanning and Bite Registration

Dental impressions with alginate or other desired medium are taken of both the maxillary and mandibular full arches. The impression should adequately capture the dentition and a good deal of the periodontium both facially and lingually. Alginate impressions should be poured immediately for optimal accuracy.

Digital workflow: Among dental laboratories which accept digital workflow, the maxillary and mandibular dental arches may be scanned in.

Next, a bite registration needs to be taken so the dental laboratory technician can relate the maxillary arch to the mandibular one. This registration should be taken with the patient at the 50-60% protrusive level. Some of the previously mentioned protrusion gauges have attachments that serve as bite forks to facilitate the bite registration process. Whether one uses blue mousse or thermacryl as their bite registration of choice, either one can flow into these attachments providing a degree of rigidity. This makes the registration more durable during packaging and handling at the lab. It is best to add the Blue Mousse® onto the bite fork extraorally with a thin layer on either side. Return the bite fork still attached to the gauge to the mouth (gauge set at 50-60% maximum protrusive). Have the patient's incisors seat back into the gauge. The mousse will intermingle with the teeth. Additional mousse can be added with a syringe tip to fill in voids, gaps or bulk up the registration in select areas (Figure 10).

Figure 10. George gauge with bite fork and bite registration capturing the patient at 50% protrusive.

As an alternative if no gauge is available, a light curable Triad™ ball can be formed and can serve as a bite jig. The patient can be guided to bite into the jig at 50-60% maximum protrusive. Instruct the patient to bite only partially through it, thus leaving 1-2 mm between the upper and lower molars. This space will be filled in by the registration paste later. Light cure with a hand held curing device. Remove the acrylic jig and continue to light cure the lingual aspect until fully cured. Replace the jig back into the mouth and inject blue mousse bite registration paste covering the exposed occlusal surfaces of both arches. Once set, remove and disinfect. Both the jig and the bite registration will be sent to the dental laboratory (Figure 11).

Figure 11. Bite registration taken with the patient's jaw in the 50% protruded position by way of an acrylic jig.

Regardless of method used, return the jig or bite fork registration to the mouth. Have the patient sit in the chair for 5-10 minutes. If the patient can tolerate this protruded position, continue with the MAD fabrication process. If the patient cannot tolerate this position at all, MAD may not be the right treatment for their OSA. But note that some forms of MAD allow the dentist to decrease the degree of protrusion so this from the original 50% protrusive captured at the first appointment.

Digital workflow: Bite registration with the mandibular teeth at 50-60% protrusive and slightly apart may also be scanned in. Protrusion can be maintained by various means such as a tongue blade or an acrylic jig to hold the position while the dental team scans the protrusive bite. With most digital scanner

systems a buccal scan on just one side at the premolar level is sufficient for the lab to piece together the bite (Figures 12-14).Retraction of the cheeks and tongue during image capture is recommended so those structures do not interfere with capture of the dentition and gingiva. Any excess saliva on the teeth or in the mouth should be suctioned out and the teeth gently dried with the air-water syringe.

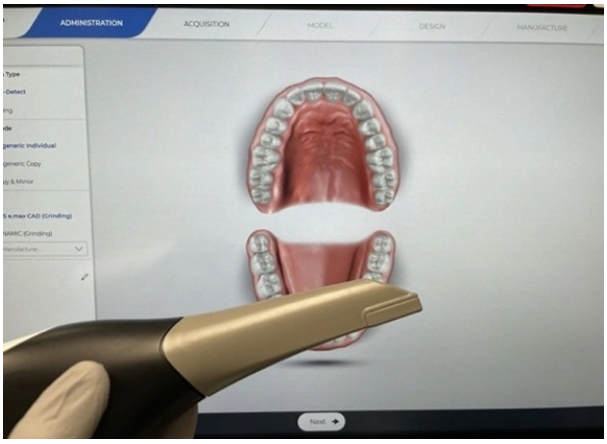


Figure 12. Preparing for digital scanning of the dental arches and bite registration in lieu of traditional impression taking with alginate and trays.

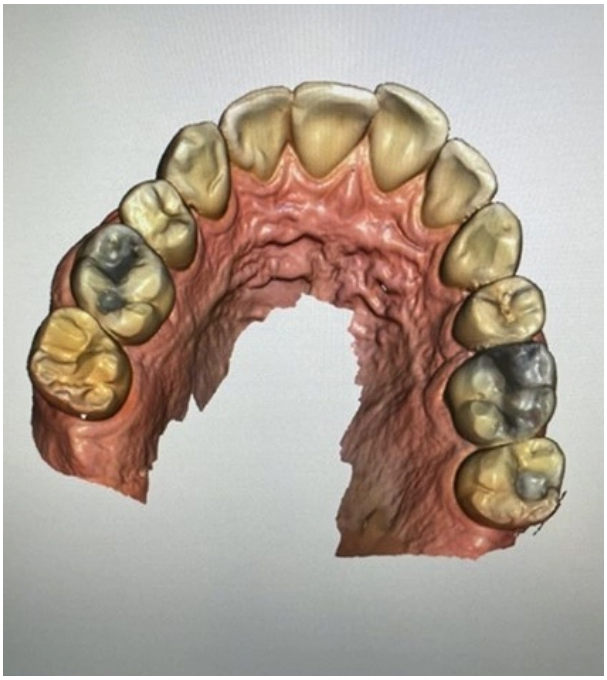


Figure 13. The maxillary arch has been digitally scanned. Repeat with the mandibular arch.

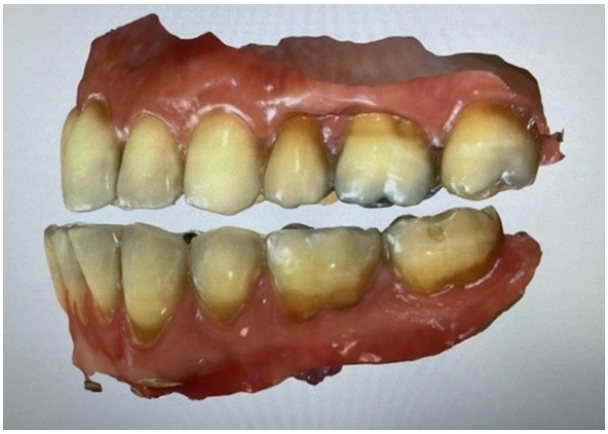


Figure 14. After capturing the protrusive bite at 50-60% of maximum protrusive ability, the software builds a model relating the mandibular arch to the maxillary one. Based on the scans, the dental laboratory can fabricate a mandibular advancement device.

Side Effects of MAD Therapy

MAD therapy may have a number of side effects that some patients may experience. Excess salivation is often reported early after delivery of a MAD while the appliance is in place. With time this subsides. Some patients report that areas where the appliance contacts the tissues causes some discomfort, for instance the teeth or the gingiva. Sore jaws are a side effect, mostly occurring upon awakening, but is most often transient.[27]

Additionally, the MAD can be reported as being over-retentitve, not retentitve enough, causing sore cheeks internally, or causing teeth to feel loose. Most of these effects are minor and can be readily resolved with minor adjustment of the MAD.

The most common side effect reported are bite changes. This can include posterior open bite (POB) changes or, due to advancement forces, mandibular incisors may be directed labially while the maxillary incisors can be pushed palatally.[27] The bite changes can be short-term or long term. After a night of wearing a MAD, it is normal for the patient to feel that their teeth do not interdigitate just right when they bite together. Sustained contraction of the lateral pterygoid muscles is the mechanism

behind this. This may resolve on its own in twenty minutes after removal of the MAD in the morning. Alternatively, some MAD products come with a bite tab or an acrylic bite pad (sometimes referred to as morning repositioner) that can be worn for 5-10 minutes upon wakening to facilitate in "resetting the bite."

Long-term occlusal changes can occur and it is best to expect them and inform the patient that this will happen. These bite changes may be permanent even if the MAD is discontinued. An 11 year follow up study using the Klearway MAD in 77 patients determined that the mandibular arch expands significantly and both overbite (2.3 ± 1.6 mm) and overjet (1.9 ± 1.9 mm) decreased.[28] The dental changes seemed to progress over time. However, in this study there was no realignment tab used in the morning.

A retrospective study of 167 OSA patients treated with either a Klearway MAD or a Herbst appliance showed that 7.1% had POB at baseline and eventually 17.9% developed a POB, however only about 1 in 4 patients noticed this change.[29] Additionally, 10.8% of the cohort met criteria for TMD before MAD delivery. These symptoms increased but by the fourth follow up diminished down to 2.4%.

Both the dental professional and the patient should recognize that bite changes can happen. Due to the side effect of our training, there is a tendency to overreact to bite changes as the end of the world. However, returning to the topic of OSA, we should realize that if left untreated, OSA will cause significant morbidity and mortality to the patient with a significant negative impact on their quality of life. Let us therefore weigh the pros and cons: bite change vs. morbidity/ mortality.

Hence a good written consent form should be developed in the dental office indicating the reason for the treatment, risks of MAD treatment, and risk of not treating OSA. The need for a follow up sleep study after MAD delivery should be emphasized. The consent should be signed. The form should also include limitations in MAD treatment in improving OSA. The definition of success was defined earlier.

Selection of the MAD Device

The number of appliances available to treat OSA has exploded in the past decade or so. There are numerous dental laboratories that have developed a product line to serve the dental professional's needs when considering a MAD. The common denominator is that they all advance the mandible in some manner using the maxilla as a push off point. Most rely on some form of hard acrylic or a bilaminar hard/ soft acrylic combination.

The two basic type of MAD are the fixed and the adjustable. The fixed MAD implies that the MAD is a monobloc appliance fabricated at a pre-set protrusive record of 50-60% and cannot be changed. The fixed MAD is generally less expensive. It may help the patient's OSA symptoms, or it may not. The downside of the fixed MAD is a patient may need a greater degree of advancement than this appliance allows. At the least, this may help reduce or eliminate snoring, a welcome change for the bed partner.

More commonly used now by far are the adjustable MAD. As mentioned earlier, the bite registration sent to the lab is most commonly fabricated at the 50% protrusion level. However, if 50% is not quite enough to alleviate their OSA symptoms per the ESS or a sleep study, then the device allows for further protrusion. A 0.25mm advancement every other night and titrated to effect can usually achieve the desired outcome. Some adjustable MAD are adjusted via a keyhole at either the front or side of the appliance (SomnoDent, dreamTAP). Other models have several arches that interface differently allowing for several preset advancements (MicrO2).

Figure 15. Example of an adjustable MAD by Respire Medical.

Delivery of the MAD

Inspect the MAD prior to patient arrival to ensure that the case was designed and fabricated as prescribed. Once the patient is in the chair, try the two arches in one at a time to ensure there is the right amount of retention. The patient should not be able to easily dislodge the appliance with their tongue. If the device seems over retentive or is impinging too much of the soft tissues then trim back with an appropriate lab bur to desired effect.

Now try and place the maxillary arch together with the mandibular arch and have the patient try to insert it into their mouth. Remind the patient that they will need to protrude their lower jaw forward to so their lower teeth can engage their appropriate place in the MAD. Start off at the 50% protrusion setting. Depending on MAD brand used, some patients can disengage if not advanced far enough forward. If this is the case, titrate forward another 1 mm and try again. Most MAD are delivered initially in the 50-70% maximum protrusion range.

You may consider starting at 50% with the notion that this would be most tolerable to the patient. It is advisable to do no further protrusions the first week to allow the patient to acclimate to the device. Develop an advancement plan with the patient. Depending on device type, this can be done every other night or once a week. Have the patient monitor their sleep quality.

Make sure the patient understands how to use the device and how to titrate it. If the device comes with a key, instruct them how to use it and how far each turn of the key advances the mandible. Give the patient limitations on how far to go. There is no need to go to 100% of maximum protrusion. Give the patient a sheet on what side effects to expect. This should be a refresher of what was given to them when the informed consent was signed. **Note: It is important to wear the MAD ≥ 4 hours per night!** Failure to do so results in incomplete management with continued sleep dysfunction along with continued risk for all the negative health consequences associated with OSA (Figure 16).

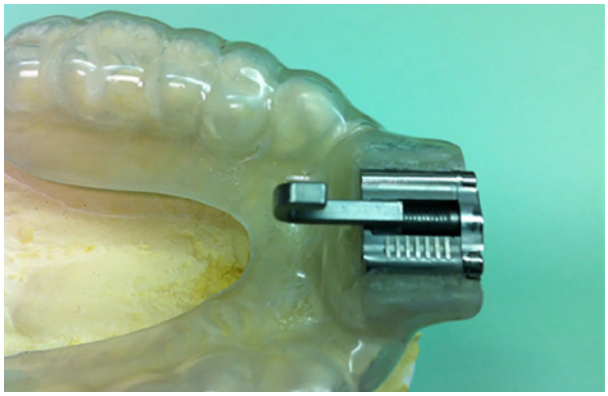


Figure 16. Close up of the upper arch of a TAP appliance, an adjustable form of MAD. It is easy for patients to "get lost" when moving the hook back and forth. Thus, they need clear instructions on finding the proper setting. Note: the keyhole is on the anterior face of the metal advancement apparatus (at the far right).

Once fit and initial settings have been determined, have the patient wear the device in the chair for 5-10 minutes to check for tolerability. This will also serve as an introduction to the patient on how the device is to be worn.

Fabricate the morning repositioner at this appointment. Many MAD products include some type of repositioner or bite tab included with the kit. If there is none, instruct the patient to put steady pressure on their chin and push backwards on it for 1-2 minutes each morning. The purpose of this is to help reset the patient's occlusion in the morning (Figures 17 and Figures 18A-B).

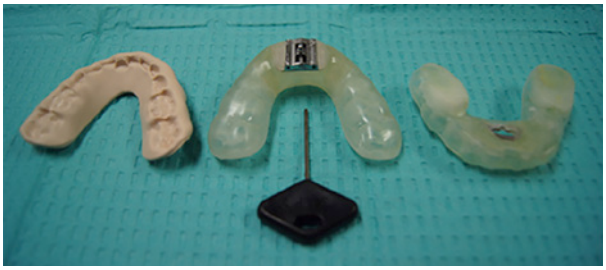
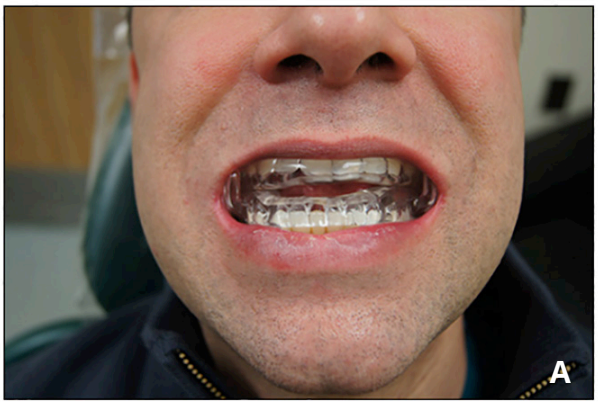


Figure 17. The TAP3 MAD system. The appliance on the left is A.M. Aligner worn for 8 minutes to help restore the bite in the morning. The middle arch is the maxillary component that houses the advancement hook. The arch on the right is the mandibular component that houses a receptacle or sometimes a bar which engages the hook. The key is shown in the center as well. Each half turn towards the patient's left ear advances the mandible 0.25 mm.



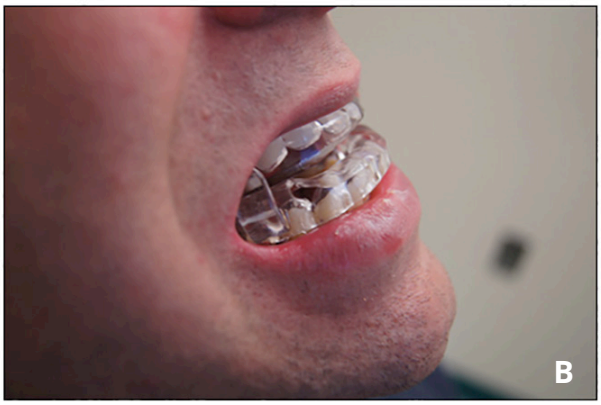


Figure 18A-B. Anterior (A) and sagittal (B) views of an adjustable MAD (MicrO2) set at 60% of patient's maximum protrusive range of motion.

Follow-up

A follow-up appointment 1-3 weeks status post MAD insertion should be undertaken to assess progress. The following check list can aid the practitioner:

- Is the MAD appliance tolerated?
- Quantify patient adherence to the MAD. Is it worn nightly? If not, how often is it worn? How many hours per night is the MAD being used?
- Is the titration plan on track?
- Any side effects or problems of fit with the appliance?
- Check the dental and oral tissues for any signs of irritation
- Check to make sure the patient can insert and remove the MAD on their own
- Have the patient demonstrate that they know how to protrude/retrude the mandibular component of the MAD
- Does the bed partner still report snoring
- Any QOL improvements in daytime sleepiness? In addition to the patient's subjective QOL reporting, a repeat ESS or STOP-BANG can provide additional data in this regard. The STOP-BANG includes blood pressure which should also be re-checked.

In regard to titrating the MAD, i.e. progressively protruding the mandible, track progress since time of delivery. If there was jaw discomfort at a particular protrusive setting, consider retruding the device by 0.25 mm or the next smallest increment particular to the MAD system you are using. These first few weeks are critical to finding a balance between tolerability and improvement in sleep. Sleep quality and device tolerability determine the titration pattern of the adjustable MAD. Do not exceed 100% of patient's maximum protrusive ability as the TMJ retrodiscal laminae may elongate.

The ESS should be repeated at each patient visit and can even be done over the phone or via email at periodic desired time points. If the ESS is less than 10, the appliance is tolerable and the patient is compliant with its use then no further titration is necessary for the time being.

Once the desired protrusive setting achieved, the patient should continue wearing the MAD for another month or longer to acclimate to the appliance to ensure complete adherence. If this is demonstrated, then it is crucial to conduct a follow up PSG with the MAD inserted into place. A good sleep technician would be familiar with advancing the MAD during the sleep study if they want to further optimize AHI. The patient will now have a pre-treatment AHI value and a post-treatment AHI value. Success can be defined by reducing the AHI to less than 5, or in more severe cases a reduction of AHI by 50% or greater is also considered a good outcome.

MAD Effectiveness

Both CPAP and MAD are effective in treating all severities of OSA. There is a trend for better success with CPAP amongst severe OSA patients.[30] MAD can reduce AHI in mild-moderate OSA cases 76%; and in severe cases 79%.[27] The surgical cure rate (AHI <5) is 43%[27] Even in severe OSA cases with the AHI in the 80s, MAD treatment significantly reduced AHI to the 1.7 – 11 range.[24]

Adherence to nightly use of 4 hours or greater is an issue with both MAD and CPAP therapies. MAD adherence ranges from 76-98% while it is 30-80% amongst CPAP users.[27] In a crossover study of 108 OSA patients, CPAP users used their machines 5.3 hours per night while MAD users wore their appliances for 6.5 hours per night.[31] Research has shown that adherence is better with MAD than CPAP.

A reasonable question to ask is what is the overall morbidity and mortality of OSA between CPAP vs. MAD treatment when adherence is taken into consideration? In a one month study of with respect to outcome measures of mean arterial pressure, blood pressure and sleep quality, MAD was not inferior to CPAP.[32]

In regards to effectiveness between titratable (adjustable) and non-titratable (fixed) MAD appliances, the titratable appliances are superior. Non-titratable appliances have a fixed mandibular position, thus protrusion cannot be further increased for therapeutic effect, nor can it be decreased to relieve TMJ discomfort or other side effects should they occur. In a study of 180 OSA patients treated with 2 titratable proprietary MAD appliances and a fixed appliance, the titratable appliances significantly improved OSA (AHI <5) in 72% of cases compared to 52% in the fixed group.[33]

A final thought in regard to MAD appliance design: early appliances assumed that increasing the vertical dimension (VD) in addition to protrusion would add further benefit. While some increase in VD is inherent in these appliances, excessive VD can actually have adverse airway effects.[34]

Other Considerations

Morning headaches can be a symptom of OSA. Morning headaches are reported in 33.6% of OSA patients compared to 8.9% in a control group, and treating the OSA resolves nearly 90% of these headaches.[35]

Some medications dentists prescribe may aggravate OSA. Drugs to avoid in untreated or non-adherent OSA patients include the benzodiazepines and the opioid class of medications, as both of these aggravate OSA symptoms.[36] However the non-benzodiazepine hypnotics such as zolpidem or eszopiclone are able to be used in OSA.

And finally on the dental side, a relationship exists between the presence of tooth wear with OSA and sleep bruxism (SB).[37] While tooth wear may or may not be indicative of current SB, both OSA and SB exhibit periods of cortical hyperarousal and micro-arousals during sleep.[39]

Summary

Patients who present with symptoms suggestive of OSA should be identified and referred to appropriate providers in the health care system. Figure 19 shows, as an example, an entry point in health care where a patient such as this may be screened. The dentist may be involved in two different points in this flow diagram. First, the dentist who is aware of OSA as a condition will be able to ask the appropriate screening questions that may increase his or her index of suspicion that there may be sleep apnea at play. Secondly, the dentist may be tasked to help manage OSA by way of a mandibular advancement device.

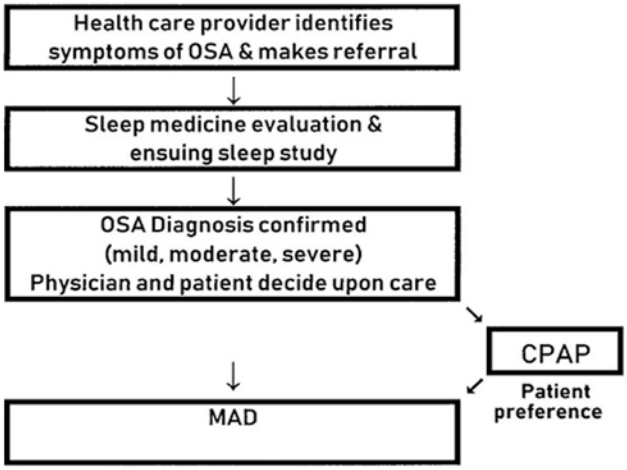


Figure 19. Patient Tracer Flow Diagram for Sleep Apnea.

CPAP = continuous positive airway pressure; **MAD** = mandibular advancement device, **OSA** = obstructive sleep apnea

Dental office staff should be aware of opportunities to intervene in patients who are suffering from OSA and through their efforts can reduce the negative health consequences of sleep apnea.

 # Clinicians interested in pursuing further education are encouraged to explore the resources available through the American Academy of Sleep Medicine and the American Academy of Dental Sleep Medicine.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: *www.dentalcare.com/en-us/ce-courses/ce578/test*

1. Sleep apnea is classified under _____.
A. sleep movement disorders
B. sleep breathing disorders
C. insomnia
D. circadian rhythm disorder

2. Stage 3 NREM sleep is the most restorative stage of sleep.
A. True
B. False

3. Obstructive sleep apnea is most prevalent in which of the following groups?
A. Children
B. Females
C. Males
D. Older, obese males

4. The letter "O" in the STOP-BANG mnemonic screening tool for OSA stands for _____.
A. observed apnea
B. onset sudden
C. older age
D. overweight

5. Which of the following are medical consequences of untreated OSA?
A. Glucose intolerance
B. Hypertension
C. Coronary artery disease
D. All of the above.

6. A diagnosis of OSA can be made by _____.
A. an Epworth sleepiness scale questionnaire
B. the STOP-Bang tool
C. a polysomnograph
D. a pulse oximeter

7. A patient returning from a sleep study has a report indicating that his mean AHI is 20 per hour. This patient would be categorized as having _____.
A. mild OSA
B. moderate OSA
C. severe OSA
D. does not meet the criteria for OSA

8. The preferred, first-line treatment of severe OSA is _____.
A. orthognathic surgery
B. continuous positive airway pressure
C. hypoglossal nerve neurostimulation
D. None of the above.

9. Reasonable goals of both PAP and MAD therapy in OSA patients is to _____.

A. lower AHI by 50% or greater
B. improve daytime wakefulness and cognition
C. eliminate periodic limb movements
D. A and B

10. Prior to fabricating a MAD for an OSA patient, all needed dental work should be completed after delivery of the MAD.

A. True
B. False

11. The most important mandibular range of motion (ROM) data to record as part of a MAD fabrication work up is _____.

A. maximum incisal opening
B. maximum lateral excursion
C. maximum retrusion
D. maximum protrusion

12. Some common side effects of mandibular advancement device treatments include _____.

A. transient TMJ soreness
B. occlusion changes
C. increase salivation
D. All of the above.

13. Bite registration for a mandibular advancement device should be captured at ____% of maximum protrusive.

A. 0%
B. 50%
C. 100%
D. 110%

14. Whether a patient treats their apnea with positive airway pressure or a mandibular advancement device, the minimal adherence required is _____.

A. 2 hours per night 20 days per month
B. at least 4 hours or more per night on 70% of nights per month (21 days)
C. at least 6 hour per night 3 days per week
D. until the patient subjectively feels less sleepy

15. A crucial step in finishing a sleep apnea case with a mandibular advancement device (MAD) is _____.

A. a follow-up sleep study once the MAD is titrated and adherence is assured
B. discontinuing treatment after 6 months
C. prescribing a benzodiazepine to help facilitate sleep
D. All of the above.

16. Continuous positive airway pressure (CPAP) is preferred in severe OSA cases but MAD is acceptable in those who prefer oral appliances or cannot tolerate or adhere CPAP.

A. True
B. False

17. All of the following statements are true EXCEPT:

A. Adherence to treatment is better with MAD than CPAP.
B. MAD treatment is only effective in mild OSA.
C. OSA treatment can improve morning headaches.
D. Excessive vertical dimension built in to a MAD can have negative effects on OSA treatment.

18. Which of the following medications can aggravate sleep apnea in the untreated, undertreated or undiagnosed OSA patient?

A. Opioids and zolpidem
B. Eszopiclone and benzodiazepines
C. Eszopiclone and zolpidem
D. Opioids and benzodiazepines

19. Other treatment modalities for OSA management include _____.

A. weight loss if body mass index is increased
B. orthognathic surgery if retrognathic
C. sleeping more on the side in positional OSA
D. All of the above.

20. Which of the following is true when delivering a mandibular advancement device?

A. First follow-up should occur a month or two after appliance delivery.
B. No follow up polysomograph is required if the Epworth sleepiness scale score is under 15.
C. Sleep quality and device tolerability determine the titration sequence of the adjustable MAD.
D. Fixed or prefabricated advancement devices are more effective in reducing the apnea hypopnea index than titratable ones.

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Additional Resources

- No Additional Resources Available.

About the Author

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