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Sleep Apnea for Dentists: An Overview of Signs, Symptoms, Consequences, and Treatment
A Peer-Reviewed Publication Written by Dr. Robb Heinrich

Abstract
Obstructive Sleep Apnea (OSA), a stoppage or partial stoppage of air flow during sleep has been gaining more attention as a Sleep Related Breathing Disorder. This treatable syndrome has many associated co-morbidities that can have severely detrimental effects on a person’s well being and general health. It has been estimated that 20% or more of the population suffers from some level of OSA, many of those are undiagnosed or unaware of their issue. Though a diagnosis must be made by a medical doctor, the dental community can be a first line of defense to screen and even treat many of these people with Oral Appliance Therapy (OAT). OAT has become a popular alternative for treating OSA for those intolerant to the traditional Continuous Positive Airway Pressure (CPAP) machine. This course will give dental professionals a high level overview of obstructive sleep apnea, consequences and treatment options including oral appliance therapy and how the clinicians can become involved in the treatment of OSA.

Educational Objectives:
At the end of this self-instructional education activity the participant will be able to:
1. Recognize the signs and symptoms of Obstructive Sleep Apnea.
2. Describe the risk factors and consequences associated with OSA.
3. Screen for OSA in the dental office.
4. Describe the options to treat OSA.
5. Describe how Oral Appliance Therapy works and recognize the limitations and complications that can occur with the use of oral appliances.
6. Describe the relationship protocol with Sleep Disorder Centers and Sleep Physicians.

Author Profile
Dr. Robb Heinrich graduated from the University of Washington School of Dentistry in 1993 and practices general dentistry in Spokane Washington with an emphasis on restorative and sleep dentistry. Dr. Heinrich maintains a special interest in Dental Sleep Medicine as it aligns with his office philosophy of treating the patient beyond their dental needs. He can be reached at robb@heinrichdds.com

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Abstract
Obstructive Sleep Apnea (OSA), a stoppage or partial stoppage of air flow during sleep has been gaining more attention as a Sleep Related Breathing Disorder. This treatable syndrome has many associated co-morbidities that can have severely detrimental effects on a person’s well being and general health. It has been estimated that 20% or more of the population suffers from some level of OSA, many of those are undiagnosed or unaware of their issue. Though a diagnosis must be made by a medical doctor, the dental community can be a first line of defense to screen and even treat many of these people with Oral Appliance Therapy (OAT). OAT has become a popular alternative for treating OSA for those intolerant to the traditional Continuous Positive Airway Pressure (CPAP) machine. This course will give dental professionals a high level overview of obstructive sleep apnea, consequences and treatment options including oral appliance therapy and how the clinicians can become involved in the treatment of OSA.

As dentists, we are trained to exam patients’ teeth, gums, and other surrounding oral structures. What if, as a dental professional, you could be instrumental in helping in the detection of a potentially life threatening disorder known as Obstructive Sleep Apnea (OSA)? This treatable disorder affects as many as 20% of the population and the majority of those affected are undiagnosed. These people are in our practices every day and we have the ability to quickly detect and screen them for OSA during a regular recall exam. In an average dental practice with 2000 patients there could be as many as 400 patients that have some form of OSA. As with oral cancer and hypertension, we can be a first line of defense to help these patients seek a diagnosis and even collaborate with their physician in their treatment.

So what exactly is sleep apnea? Apnea is a term that means “without breath.” OSA can be characterized by an actual stopping of breathing during sleep due to the complete (apnea) or partial (hypopnea) collapse of the upper airway. This obstruction and collapse can occur somewhere between the nasal turbinates down to the level of the vocal cords; however, the most common area for the collapse to occur is the oropharynx, which runs from the hard palate to the tip of the uvula\(^1\) (Figures 1a - 1c).

There are 3 kinds of sleep apnea: 1.) Obstructive Sleep Apnea (OSA) is characterized by repetitive episodes of complete (apnea) or partial (hypopnea) upper airway obstruction during sleep. OSA is the most common form of sleep apnea. 2.) Central Sleep Apnea is not obstructive in nature,
instead caused when respiratory control centers in the brain temporarily pause the activation of the muscles involved in breathing. 3.) Mixed is a combination of OSA and Central Sleep Apnea.

It is important to understand what happens when an apnea or hypopnea event occurs during sleep. Apnea is a complete cessation of airflow for 10 seconds or longer. Hypopnea is a 50% reduction in oronasal airflow for 10 seconds or longer accompanied with an oxygen desaturation of >4% with associated arousals.

During the course of a night’s sleep a person with OSA will have multiple apnea and/or hypopnea events, as many as 100 per night, which will trigger signals to the brain to arouse the person from their sleep state to regain a normal course of breathing. The person struggles to breathe, the diaphragm and chest muscles work harder to open the obstructed airway and pull air into their lungs. This is then followed by a loud gasp, snort or body jerk. Consequently this creates very fragmented sleep structure thereby yielding poor sleep quality. Obtaining a deep level of sleep each night is vitally important for sustaining immune function, memory, psychologic wellbeing, biochemical refreshment and decreasing fatigue. With the decrease in oronasal airflow, arterial blood oxygen levels are also decreased (known as hypoxemia) thereby increasing the risk of organ tissue damage due to oxygen/carbon dioxide imbalances.

Sleep apnea is diagnosed using polysomnography (PSG). A PSG, or sleep study, is performed in a sleep laboratory and involves continuous overnight recordings measuring the patient’s breathing and respiratory efforts as well as blood oxygen saturation, body position, heart monitoring and brain wave function. A PSG will be scored by a sleep technician and yield what is called an AHI (apnea-hypopnea index) for the patient. An AHI (average number of apneas and hypopneas per hour of sleep) greater than 5 defines OSA (See Figure 2).

Symptoms of OSA
The cardinal symptom of OSA is loud snoring with subsequent cessation of breathing and gasping attempts for air. It is often the bed partner who will complain first about the loud snoring and perhaps witnessed apnea events. Those with OSA likely are not aware of the events that occur during their sleep. It should be noted the absence of snoring can make OSA less likely but does not exclude it and should not be used as the sole predictor of OSA. Other clinical features must be evaluated. Witnessed apneas are another hallmark symptom of OSA but could be difficult to report if the patient sleeps alone. Often times the patient may report waking themselves during an episode of nocturnal choking.

A third and very important symptom of those affected by OSA is excessive daytime sleepiness (hypersomnia) and fatigue due to the fragmented sleep and multiple nocturnal arousals. Besides the subjective reports from the patient, the Epworth Sleepiness Scale is the most accepted and commonly used report to assess daytime sleepiness (see Figure 3). A score of 10 or greater out of 24 is a good indicator of excessive daytime sleepiness. This screening tool can easily be incorporated into your health history. Other symptoms include, insomnia, esophageal reflux, morning headaches, impaired memory and concentration and possible anxiety and depression.

Figure 3.

<table>
<thead>
<tr>
<th>Severity of OSA</th>
<th>AHI</th>
</tr>
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<tbody>
<tr>
<td>Normal</td>
<td>&lt; 5</td>
</tr>
<tr>
<td>Mild</td>
<td>5 to 15</td>
</tr>
<tr>
<td>Moderate</td>
<td>15 to 30</td>
</tr>
<tr>
<td>Severe AHI</td>
<td>&gt;30</td>
</tr>
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</table>

Signs of OSA
Fortunately many of the signs of OSA can be seen intraorally during your routine recall exam. This is a time when the possibility of your patient having OSA can be assessed during a screening process similar to oral cancer screening. This screening process can be quickly and easily done by your hygienists and assistants. Does your patient have marked anterior wear due to posturing of the mandible forward in an attempt to open their airway? Is their tongue enlarged, filling up much of their oral cavity and scalloped on its lateral border from pressing against the lingual surfaces of the teeth? Many patients with OSA will have marked acid erosion on the lingual surfaces of their teeth due to acid reflux which occurs when the patient struggles
to breathe during an apnea or hypopnea event. As negative pressure builds in the thoracic area during the struggle, a positive pressure builds in the gastric area thereby pushing those acidic fluids back up into the oral cavity. Sleep bruxism is often seen in patients with OSA which will manifest as worn teeth, gingival recession, and abfractions. (see Figures 4a - 4d)

Figure 4a.

Look for enlarged tonsils, edema and erythema of the soft palate and surrounding structures that can be indicative of snoring and OSA. The gingival tissues, usually around the anterior teeth, may appear red and swollen, a sign of mouth breathing which again can be related to snoring, a sleep related breathing disorder. Constricted maxillary and mandibular arches also crowd the tongue and therefore push it back to further block the airway when the patient is relaxed during sleep. Blockage of the upper airway due to tonsillar hypertrophy should also be evaluated (Figure 5). Abnormal enlargement of the tonsils along with constricted arches are frequently major contributors to a sleep related breathing disorder in children. In a study aiming to determine the extent to which tonsil size may contribute to OSA in children, a significant correlation was noted between tonsillar enlargement and severity of OSA. 3

Figure 5.

Extra-oral signs of OSA are also easily observed during a dental exam. Patients with OSA often exhibit retrognathia and/or maxillary deficiency, both of which work against the patient to further constrict the airway during sleep. A large and/or short neck, chronic chapped lips and excessive loose skin below the chin are also often seen.

Risk factors of OSA

One of the most important risk factors for patients having OSA is obesity. In discussions relating to patients’ weight and severity of OSA we speak in terms of their Body Mass Index (BMI) (See Figure 6). The connection between BMI and OSA has been well documented. It was found in a sleep clinic population that 28% of patients had a BMI greater than 30 and 47% had a BMI between 26 and 30. 2 Specifically, the fat around the neck and waist are of particular importance. Though there is no definitive threshold value of neck size where OSA occurs, it is generally accepted that a neck size in men greater than 17 inches and 16 inches in women, places those patients at a higher risk level. The prevalence of OSA among the severely obese has been reported to range from 50% to 90%, leading to the assumption that effective treatment of obesity could have superlative effects on OSA.4 It must be noted that many patients with lower BMIs may also be at risk if they are showing signs and have symptoms of OSA due to the other factors aforementioned.
Figure 6.

\[
\text{BMI} = \left( \frac{\text{weight in pounds}}{(\text{height in inches}) \times (\text{height in inches})} \right) \times 703
\]

<table>
<thead>
<tr>
<th>BMI</th>
<th>Weight Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 18.5</td>
<td>Underweight</td>
</tr>
<tr>
<td>18.5-24.9</td>
<td>Normal</td>
</tr>
<tr>
<td>25.0-29.9</td>
<td>Overweight</td>
</tr>
<tr>
<td>30 and Above</td>
<td>Obese</td>
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Epidemiologic risk factors include: increase in age, male gender, smoking and alcohol use. There also seems to be a genetic and ethnic component to having OSA. OSA prevalence increases 2 to 3 times in people older than 65 when compared to ages 30 to 64. A higher deposition of fat around the upper airway along with an increase in length of the upper airway, therefore increasing collapsibility, seems to contribute to the male predisposition to OSA. Craniofacial abnormalities and enlargement of the soft tissue structures around the upper airway may account for the higher risk of OSA in African-Americans, Pacific Islanders, Mexicans and Asians.

**Consequences of OSA**

Clinically, patients with OSA present with high BMIs and complain of daytime sleepiness. A more in depth investigation of these patients often reveals what can be classified as cardio-metabolic and neurobehavioral dysfunctions.

A number of studies have shown a strong association between having OSA and developing coronary artery disease (CAD) along with increase risk of stroke. A study from the Journal of Oral and Maxillofacial Surgery showed that of those subjects with OSA, 21% had calcified carotid atheromas (fatty plaques) where as in the control group (without OSA) only 2% had calcified carotid atheromas.\(^5\) In addition, a Wisconsin study showed, over an average 13.8 year follow up, subjects with severe OSA (AHI greater than 30) had a greater risk of cardiovascular mortality than did subjects without OSA.\(^3\)

Many studies have shown OSA having a strong link to hypertension. A 2000 study published in the Journal of the American Medical Association of 6000 men and women age 40 and older, showed that people with more than 30 breathing pauses per hour during sleep were more than twice as likely to suffer from high blood pressure than those without breathing pauses.\(^6\) Other studies have shown that treatment of OSA causes blood pressures to fall, again making the correlation between OSA and hypertension strong. It is also worth noting that along with hypertension, there may be an independent association between having OSA and the other components of Metabolic Syndrome (glucose intolerance, dislipidemia, and central obesity). It follows then that OSA may increase the patient’s risk for type 2 Diabetes.

It is still unclear whether hypoxemia or sleep fragmentation have the greatest influence on cardiometabolic disease.\(^3\) A Cleveland Family Study found a stronger association between sleep fragmentation and incident of hypertension, while glucose intolerance was more closely associated to hypoxemia.\(^3\)

**Neurocognitive consequences** of OSA are thought to be due to a combination of sleep fragmentation and hypoxemia from the repetitive respiratory events during sleep.\(^7\) Those with OSA often report memory loss, loss of ability to concentrate and problem solve, depression, and mood changes. An increase in motor vehicle and occupational accidents have also been reported. It is also important to note the neurocognitive effects of a sleep related breathing disorder in children. A sleep related disorder such as OSA can lead to behavioral issues such as hyperactivity and inability to pay attention which then can lead to a decrease in academic performance. Many of these kids have been diagnosed with ADHD (attention-deficit hyperactivity disorder) without any regard to their quality of sleep. One study found that 13.8% of preschool age children, 29.4% of preadolescent kids, and 40.9% of adolescent aged kids, all with a sleep related breathing disorder, had an associated condition of ADHD.\(^7\) Signs of a sleep related breathing disorder in children are similar to those of adults and may also include occasional bed wetting, mouth breathing during the day, difficulty waking up in the morning, night time bruxism, poor or inadequate lip seal, and dark circles under their eyes, known as allergic shiners.

**Treatment options for OSA**

**CPAP**

It is generally accepted that those with OSA first be treated with Continuous Positive Airway Pressure (CPAP) therapy and CPAP therapy is considered the gold standard for treatment of OSA (See Figure 7). The CPAP mechanism most often works to direct the flow of pressurized air through the nasal passages and upper airway and then into the lungs. The pressurized airflow creates airway patency in the oropharyngeal area, anteriorly displaces the tongue, and distends the lateral pharyngeal walls thereby creating patency of the entire airway, keeping it from collapsing\(^7\) (see figure 8). Not all patients with OSA will accept or be able to tolerate CPAP. Compliance rates vary widely with the definition of compliance and are also dependant upon the severity of the patient’s OSA. When compliance is defined as 4 or more hours of nightly use, non compliance can range between 46% and 83%.\(^2\) A higher compliance rate is also seen in those patients exhibiting a more severe level of OSA.
Surgical options

It should be noted that prior to commencing any surgical procedure to treat OSA, a pre-treatment evaluation be conducted by a qualified physician to identify specifically the areas of obstruction in the upper airway. In addition, success rates differ widely depending a more liberal or rigorous definition of success.

Tonsillectomy and/or adenoidectomy has been shown to be 80% successful in adults with severe OSA and 100% successful in adults with mild OSA when surgical success is defined as a > 50% reduction in AHI and a less than 20 AHI score. These patients typically will present with tonsillar and/or adenoid hypertrophy. This procedure can also be very effective in children.

Uvulopalatopharyngoplasty (UPPP) is a common surgical procedure involving the reduction of the tissues in the soft palate area, the uvula and the lateral and posterior walls of the pharynx. It is often performed in conjunction with a tonsillectomy/adenoidectomy. Using the same definition of surgical success, the UPPP can yield a 25 to 75% success rate. The success rate of the UPPP is higher when the obstruction is limited to the retropalatal area.

A Maxillo-mandibular advancement (MMA) involves the forward repositioning and fixation of the maxilla along with the mandible. It is considered an effective procedure because it involves the retropalatal and retroglossal areas and also increases the tension on the genioglossal muscle, improving the airway space. The success of the MMA varies between 75% and 100%, again depending on the definition of surgical success. The MMA procedure is often times performed along with a (GA), genioglossus advancement (chin advancement). This GA creates tension on the tongue, pulling it forward, preventing collapsing of the tongue into the retroglossal area during sleep. When the GA is performed alone it can have a success rate of 42% to 75% (see Figure 9).

Lifestyle change and positional therapy

A very important conservative treatment option for OSA is a reduction in BMI. As it has been shown, there is a direct correlation to obesity and severity of OSA and reduction in central obesity can lower a patient’s AHI. A 10% weight gain can increase a person’s risk of developing OSA by 6 times. If applicable, weight loss should always be considered in conjunction with other treatments for OSA. Smoking cessation and limited alcohol use are also very important. Good sleep hygiene should also be practiced which includes going to bed and waking at the same time each day, limiting the intake of caffeine, alcohol, and food before bedtime, not exercising before bed and always sleeping in a fully darkened environment. Positional therapy may include bed wedges or body pillows to keep a person from turning onto their back in the supine position where OSA is usually most severe. A t-shirt with tennis balls sewn into the back is another option, in hopes of keeping the patient from sleeping on their back.

Oral Appliance Therapy (OAT)

Oral appliances (OA) are becoming a more recognized and accepted treatment option for OSA because of their non invasive nature, high patient acceptance, and better portability over a CPAP machine. The most widely used OA is the mandibular repositioning device (MAD). These appliances move the mandible forward creating patency of the upper airway during sleep (See figures 10, 11). Studies
have shown that anterior repositioning of the mandible allows the tongue to move forward away from the upper airway and that the volume of the upper airway is increased. Repositioning the mandible forward also has an effect on the muscles that support the pharynx. It seems that there is a stabilization and increase in tone of the muscles that support the upper airway which in turn helps to decrease the collapsibility of the airway during sleep. Studies have shown that not only is the area of the velopharynx increased in the lateral and anteroposterior dimensions but the oropharynx is increased in the lateral dimension during mandibular advancement.

Figure 10.
Oral appliance pulls the lower jaw forward opening the airway

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Figure 11.

Success rates using OAs (defined as AHI <5 and resolution of symptoms) have varied between 19% and 75% for patients with mild to moderate OSA. When a more liberal definition of success is used success rates increase. Factors contributing to higher success rates include the patient having a lower BMI, having a lower baseline AHI (OSA in the mild to moderate range), and having a higher mandibular range of motion. Anatomical factors of the upper airway will also affect efficacy of oral appliance therapy (OAT). Studies comparing OAT to CPAP have shown CPAP to be more efficacious in reducing the AHI to normal levels (<5) whereas OAT was able to do the same in two-thirds of patients; however, compliance was higher with OAT. Indications for OAT include patients with mild to moderate OSA who prefer an oral appliance over CPAP or for those who failed CPAP. For those with severe OSA, OAT is indicated only if CPAP therapy has been attempted and failed or if the patient is found to be CPAP intolerant. Contraindications for OAT include poor dental health including advanced periodontal disease, inadequate number of teeth to support the appliance (consensus is at least 6 to 10 teeth per arch), severe TMJ disorders, and inadequate mandibular function or limited range of motion. It is extremely important for the treating dentist to assess these conditions prior to fabrication of an OA.

It is important to discuss the possible side effects with those interested in OAT. Dry mouth, excessive saliva, gingival irritation, along with tooth discomfort, morning bite changes and TMJ pain can be reported and are usually transient in nature. Long term effects include more prominent occlusal changes, tooth movement, increased facial height, and changes in the inclination of the incisors. Many of these side effects are subclinical and are generally considered negligible to the patient.

It is important that long term follow up be conducted to adequately assess these possible changes.

There are over 40 FDA approved oral appliances. The most important feature of any OA in treating OSA is that it be adjustable or titratable. The adjustable appliances allow the dentist to incrementally adjust the anterior-posterior mandibular position of the appliance over time and it is thought that the more protrusive the mandible can be positioned (and tolerated by the patient) the better the chance of lowering the AHI. In a review of the literature by Ferguson et al, an AHI of <10 was obtained in 31% of patients when the oral appliance was set at 50% of the patient’s maximum protrusion. At 75% of the patients’ maximum protrusion an AHI of <10 was obtained in 52% of patients. The final or most effective mandibular position of the oral appliance is somewhat subjective and based on patient report. New technology is available that may be able to help pinpoint a more objective mandibular position for greater OAT success.

Pre treatment assessment
The American Academy of Dental Sleep Medicine has outlined specific protocols for OAT in treating OSA. Before treating OSA with OAT the dentist must obtain a referral from a qualified sleep physician, usually after the patient has undergone a sleep study and has been diagnosed with OSA by the physician. It is important
to note that the diagnosis of OSA cannot be made by a dentist and must come from a qualified physician. The dentist will then assess the patient’s suitability for OAT (see indications and contraindications above) and fabricate the appliance after reviewing the risks and benefits along with an informed consent for treatment. Follow up usually begins at 2 weeks post delivery of the appliance. At this time titration (very small, usually .1mm/day, forward advancements of the appliance’s mandibular piece) usually begins. The patient will be seen 4 to 6 times over the next 6 months to assess the initial signs and symptoms (ie snoring and daytime sleepiness) as well as determine if any dental changes have occurred. Once proper fit, final mandibular position, and positive subjective results have been obtained with the OA, the patient should be referred back to their sleep physician to undergo objective testing usually with a follow up sleep study. The patient should then be seen annually by the dentist to again assess signs, symptoms and any dental changes.

Conclusion
It should now be clear that as dentists and hygienists we can easily screen our patients for Obstructive Sleep Apnea during a regular recall exam and because OSA has been associated with long term deleterious health consequences it is a major public health issue. If OSA is suspected, a conversation should occur with the patient alerting them to your findings and a referral made either to their primary care physician or to a sleep doctor asking for further examination in regards to possible OSA. This is an excellent way to merge the efforts of physicians and dentists in creating long term health for our patients. Oral Appliance Therapy is one mode of treatment that a dentist can use to treat those that are found to be CPAP intolerant. It’s one more way that we can go beyond the teeth and treat the patient as a whole being, potentially adding years to their lives.

Disclaimer
This article is intended to give the dentist a high level overview of Obstructive Sleep Apnea and treatment options including Oral Appliance Therapy. It is not intended to teach the dentist the technical aspects of treating OSA with Oral Appliance Therapy. The author highly recommends instruction from a qualified educator in the field of dental sleep medicine before treating sleep apnoea on any level.

References
2. A M Li, E Wong, J Kew, S Hui, T F Fok. Use of tonsil size in the evaluation of obstructive sleep apnoea. Archives of Disease in Childhood. 2002: 87: 156-159

Author Profile
Dr. Robb Heinrich graduated from the University of Washington School of Dentistry in 1993 and practices general dentistry in Spokane Washington with an emphasis on restorative and sleep dentistry. Dr. Heinrich maintains a special interest in Dental Sleep Medicine as it aligns with his office philosophy of treating the patient beyond their dental needs. He can be reached at robb@heinrichdds.com

Author Disclosure
Dr. Robb Heinrich maintains a partnership with Sleep Better Northwest, www.sleepbetternw.com, where he and his partner help those with Obstructive Sleep Apnea from the Eastern Washington and North Idaho areas.
1. One of the most important risk factors in children what physical characteristic was found to be highly correlated with obstructive sleep apnea?  
   a. smoking  
   b. being female  
   c. parents having OSA  
   d. having a high BMI (obesity)

2. An apnea event is characterized by:  
   a. A complete stopping of breathing during sleep for 10 seconds or longer  
   b. A desaturation of blood oxygen only  
   c. A partial stopping of breathing during sleep  
   d. A complete stopping of breathing during sleep for 5 seconds or longer

3. Which of the following is NOT a type of sleep apnea?  
   a. Hypopnea Sleep Apnea  
   b. Central Sleep Apnea  
   c. Mixed Sleep Apnea  
   d. Obstructive Sleep Apnea

4. Which best describes the obstruction that occurs during an apnea/hypopnea event?  
   a. nasal turbinates  
   b. the uvula  
   c. the oropharnyx  
   d. below the tongue

5. How is obstructive sleep apnea diagnosed?  
   a. by a dentist using polysomnography (sleep test)  
   b. by a sleep lab using polysomnography  
   c. by a certified sleep doctor using polysomnography  
   d. by a dentist or certified sleep doctor using polysomnography

6. A hypopnea is best defined as:  
   a. 50% reduction in oronasal airflow for 10 seconds or longer accompanied by an oxygen desaturation of >4%  
   b. 50% reduction in oronasal airflow for 5 seconds or longer accompanied by an oxygen desaturation of >2%  
   c. 50% reduction in oronasal airflow for any amount of time accompanied by an oxygen desaturation of >4%  
   d. 50% reduction in oronasal airflow for 10 seconds or longer

7. Which of the following is not a major symptom of OSA?  
   a. acid erosion of the teeth  
   b. snoring  
   c. daytime sleepiness  
   d. witnessed apnea events of stoppage of breathing during sleep

8. On the Epworth Sleep Scale a score of what or greater usually is a good indicator of daytime sleepiness?  
   a. 5  
   b. 8  
   c. 10  
   d. 12

9. Which of the following are intraoral signs a patient may have OSA?  
   a. marked anterior wear on  
   b. scalloped lateral border of the tongue  
   c. gingival recession along with abfractions and chemical erosion  
   d. all of the above

10. In children what physical characteristic was found to be highly correlated with OSA?  
    a. daily hyperactivity  
    b. enlarged tonsils  
    c. daytime sleepiness  
    d. night time snoring

11. One of the most important risk factors contributing to patients having OSA is:  
    a. smoking  
    b. being female  
    c. parents having OSA  
    d. having a high BMI (obesity)

12. Which statement is not true:  
    a. increase in age correlates to a higher risk of having OSA  
    b. being of the male gender correlates to a higher risk of having OSA  
    c. smoking and alcohol use increase risk of having OSA  
    d. genetics does not seem to be a risk factor in having OSA

13. The score associated with the diagnosis of OSA is the:  
    a. BMI  
    b. PSG  
    c. AHI  
    d. CAD

14. The AHI between 15 and 30 indicates what level of OSA?  
    a. Mild  
    b. Moderate  
    c. Severe  
    d. None of the above

15. The AHI is defined as:  
    a. the average number of apneas and hypopneas per hour during sleep  
    b. the total number of apnea and hypopneas for the entire time the patient was asleep  
    c. the number of apneas minus the number of hypopneas per hour during sleep  
    d. the average number of hypopneas minus apneas per hour during sleep

16. Which of the following have been shown to be associated with OSA?  
    a. coronary artery disease  
    b. hypertension  
    c. a and b  
    d. a but not b

17. Signs of obstructive sleep apnea in children are similar to those in adults along with:  
    a. night time bruxism  
    b. mouth breathing during the day  
    c. dark circles under their eyes  
    d. all of the above

18. CPAP therapy works to do all of the following except:  
    a. create upper airway patency by distending the pharyngeal walls  
    b. anteriorly displace the tongue  
    c. keep the patient on his or her side  
    d. allow the tongue to move forward

19. The following is true about tonsillectomy/adenoidecetomy when it relates to the treatment of OSA:  
    a. success rates vary between 80% and 100% depending on the definition of success  
    b. success rates vary between 30% and 50% depending on the definition of success  
    c. can be very effective in children  
    d. a and c are both true

20. The Uvulopalatopharyngoplasty (UPPP) involves the reduction of the following soft tissues except:  
    a. the tongue  
    b. soft palate  
    c. the uvula  
    d. lateral and posterior walls of the pharynx

21. Which of the following is not true regarding the Maxillo-mandibular advancement surgical option in treating OSA:  
    a. Success rates vary between 75% and 100% depending on the definition of success  
    b. cannot be performed with a genioglossus (chin) advancement  
    c. involves the retropalatal and retroglossal areas  
    d. provides anterior tension on the genioglossal muscle

22. A 10% weight gain can increase a persons’ risk of developing OSA by how many times:  
    a. 4  
    b. 5  
    c. 6  
    d. 10

23. Positional therapy to treat OSA involves which of the following:  
    a. something the keep the person from sleeping on their backs  
    b. sleeping in a darkened room  
    c. not exercising before bed  
    d. going to bed at the same time each night

24. The mandibular advancement device works to do all of the following except:  
    a. move the mandible forward  
    b. open the mandible as far as the patient can without creating TMJ pain  
    c. stabilize the muscles that support the pharynx  
    d. allow the tongue to move forward

25. Three main factors that increase the success of the mandibular advancement device include all of the below except:  
    a. a lower BMI  
    b. having at least 6 to 10 teeth in each arch  
    c. having a lower baseline AHI  
    d. having a larger mandibular range of motion

26. Which of the following is not true regarding Oral Appliance Therapy:  
    a. OAT is indicated for any patient diagnosed with OAT  
    b. OAT is indicated for those with mild to moderate OAT if an appliance is preferred  
    c. OAT is indicated for those who fail CPAP  
    d. OAT is indicated only for those with severe sleep apnea only after CPAP therapy has been attempted

27. Side effects of Oral Appliance Therapy include all of the following except:  
    a. morning bite changes  
    b. excessive salivation  
    c. dry mouth  
    d. loss of hearing

28. It is generally accepted that the most important aspect of any mandibular advancement device is that it be:  
    a. all plastic  
    b. titratable or adjustable  
    c. be of two separate pieces, upper and lower  
    d. allow the patient to speak and drink

29. Which of the following is not true regarding the final mandibular position of the oral appliance:  
    a. it is usually based on subjective reports from the patient  
    b. it must be tolerated by the patient in regards to TMJ and muscle pain  
    c. the more anteriorly the mandible can be positioned the better chance of lowering the patients’ AHI  
    d. the appliance should also open the patients’ bite as far as the patient can tolerate.

30. Follow up protocol after the insertion of an oral appliance includes all of the following except:  
    a. patient is to be seen 4 to 6 times over the course of 6 months post insertion and then at one year  
    b. referral back to the patient’s sleep doctor to assess any bite or tooth changes  
    c. referral back to the patient’s sleep doctor for follow up sleep study  
    d. patient should be assessed for changes in snoring and daytime sleepiness
**Educational Objectives**

1. Recognize the signs and symptoms of Obstructive Sleep Apnea.
2. Describe the risk factors and consequences associated with OSA.
3. Screen for OSA in the dental office.
4. Describe the options to treat OSA.
5. Describe how Oral Appliance Therapy works and recognize the limitations and complications that can occur with the use of oral appliances.
6. Describe the relationship protocol with Sleep Disorder Centers and Sleep Physicians.

**Course Evaluation**

1. Were the individual course objectives met?
   - Objective #1: Yes No
   - Objective #2: Yes No
   - Objective #3: Yes No

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

2. To what extent were the course objectives accomplished overall? S 4 3 2 1 0
3. Please rate your personal mastery of the course objectives S 4 3 2 1 0
4. How would you rate the objectives and educational methods? S 4 3 2 1 0
5. How do you rate the author’s grasp of the topic? S 4 3 2 1 0
6. Please rate the instructor’s effectiveness. S 4 3 2 1 0
7. Was the overall administration of the course effective? S 4 3 2 1 0
8. Please rate the usefulness and clinical applicability of this course. S 4 3 2 1 0
9. Please rate the usefulness of the supplemental webography. S 4 3 2 1 0
10. Do you feel that the references were adequate? Yes No
11. Would you participate in a similar program on a different topic? Yes No
12. If any of the continuing education questions were unclear or ambiguous, please list them.
13. Was there any subject matter you found confusing? Please describe.
14. How long did it take you to complete this course?
15. What additional continuing dental education topics would you like to see?

**PLEASE PHOTOCOPY ANSWER SHEET FOR ADDITIONAL PARTICIPANTS.**

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**CANCELLATION/REFUND POLICY**

Any participant who is not 100% satisfied with this course can request a full refund by contacting PennWell in writing.

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**ADDITIONAL INFORMATION**

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** 있게 되어서, 자두나무와 고양이의 경우를 상황을 설명한 후, 해당문장의 중요한 내용을 3차례에 걸쳐 추출하고 싶습니다.**

1. **계속적 교육의 목표를 정리**
   - 개인의 교육목표를 달성하였는지:
     - Objective #1: Yes No
     - Objective #2: Yes No
     - Objective #3: Yes No
   - 개별 교육목표의 성과를 평가:
     - 성과 = 5, 미흡 = 0

2. 개별 교육목표의 효과성은有多大?: S 4 3 2 1 0
3. 개인이 개인적으로 할 수 있는 주요 교육목표를 평가:
4. 교육목표 및 교육방법의 중요성과 효용성을 평가:
5. 교육자료의 악수와 전문적 창의성:
6. 총괄적으로 관리 및 교육의 관리:
7. 교육장치의 기능성과 효용성:
8. 교육장치의 유용성과 전문적 적용성:
9. 교육장치의 전체적인 관리 및 교육의 관리의 적합성:
10. 기술적 및 임상적 적용성:
11. 학습자 없었던 주제에 대해 설명:
12. 교육 자료에서 미분명한 부분을リスト:
13. 교육 자료에서 미분명한 부분을 리스트:
14. 총 학습이 너무 오래 걸렸는지:
15. 추가 교육 교육주제에 대해 생각:

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