

American Association of Orthodontists

White Paper: Obstructive Sleep Apnea and Orthodontics

I. Introduction

The specialty of orthodontics involves much more than just moving teeth, and the management of sleep apnea bears witness to this. As such, there is increasing interest in the role of the orthodontist both in screening for obstructive sleep apnea (OSA) and as a practitioner who may be valuable in the multidisciplinary management of OSA in both children and adults. As experts in the science of facial growth and development, combined with our knowledge of oral devices, orthodontists are well suited to collaborate with physicians and other allied health providers in the treatment of OSA.

While OSA can only be definitively diagnosed by a physician, the orthodontist may be called on to screen for OSA, contribute to the identification of underlying dentofacial components, and assist the physician in managing the disease. As such, the orthodontist is not able to manage this care alone, and a cooperative, shared effort between the orthodontist and other medical professionals is preferred to optimize care of patients with OSA.

A patient with suspected OSA may come to the orthodontist several different ways. A patient who has been medically diagnosed with OSA may be referred to the orthodontist by a physician who prescribes an oral appliance and/or suggests orthodontic/orthopedic therapy to assist in the management of the OSA. At other times, a patient or caregiver may present to the orthodontist with concerns about breathing during sleep. Additionally, patients may present to the orthodontist unaware of their obstructive sleep apnea, and orthodontic screening may unveil the need for further evaluation by a physician.

In November 2017, the Board of Trustees of the American Association of Orthodontists tasked a panel of medical and dental experts in sleep medicine and dental sleep medicine to create a document designed to offer guidance to practicing orthodontists on the suggested role of the specialty of orthodontics in the management of OSA. The panel completed an exhaustive review of the available literature as well as contributed their own personal expertise gleaned from managing these patients in both academic centers and within private practice settings. In considering the literature, it was obvious that there is broad interest in OSA, as evidenced by the development of guidelines for the consideration and treatment of OSA that spans the world and involves many different communities. The topic has been covered by physicians, dentists, and scientists from a variety of organizations including the American Dental Association, American Academy of Dental Sleep Medicine, American Academy of Sleep Medicine, European Respiratory Society, Australian Dental Association, American Association of Oral and Maxillofacial Surgeons, American College of Prosthodontists, American Academy of Pediatric

Dentistry, Canadian Dental Sleep Medicine, Canadian Thoracic Society, American Academy of Pediatrics, and the U.S. Preventative Respiratory Society, among others.

However, the task force could not identify any formal OSA guidance for orthodontists. This was surprising in that orthodontists have specialized knowledge, skill, and experience that would be beneficial in the management and care of patients with OSA. In addition, orthodontists typically have a broad patient population (children, adolescents, and adults), with contact maintained over a long period of time. Moreover, orthodontists have a long and productive history of working with others in medicine and dentistry to provide collaborative care for patients with special needs (e.g., cleft lip and palate, craniofacial syndromes, complex restorative cases, and orthognathic surgery).

Given that OSA can be a serious, even life-threatening disorder and given the quality of patient management and care that can be provided by orthodontists, the task force determined that it was very important to develop specific recommendations that would be useful to an orthodontist in practice. The following represents a summary of their findings and recommendations.

II. Adult OSA

Sleep-related breathing disorders (SRBD) is a diagnostic category of disease that encompasses obstructive phenomena including primary snoring, upper airway resistance syndrome and obstructive sleep apnea (OSA), along with the related entities of central sleep apnea and sleep-related hypoventilation. This document focuses on OSA, beginning with this section on the adult patient (i.e., greater than 18 years of age). Clinical concerns for other forms of SRBD and additional types of sleep disorders (e.g., insomnia, central disorders of hypersomnolence, circadian rhythm sleep-wake disorders, sleep-related movement disorders, and parasomnias), if identified, should be referred to a physician for evaluation and treatment; a sleep medicine physician is preferred.

Etiology

Obstructive sleep apnea occurs as a function of increased collapsibility of the upper airway. The pharyngeal critical closing pressure (P_{crit}) is the pressure at which the upper airway collapses. This collapsibility is influenced further by impaired neuromuscular tone. Respiratory effort increases to maintain airflow through a constricted airway, accompanied by relative increase in serum carbon dioxide (hypercarbia) and decrease in serum oxygen (hypoxemia). The increased work of breathing causes a cortical arousal from sleep, which in turn raises sympathetic neural activity leading to increased heart rate and blood pressure and a tendency for cardiac arrhythmia. With the cortical arousal from sleep comes an increase in airway patency and resumption of normal airflow, with subsequent return to sleep and recurrence of sleep-related upper airway collapsibility. This disruption in breathing may occur multiple times per hour for the entire duration of the patient's sleep.

The complexity of obstructive sleep apnea is exemplified by its multifactorial etiology. Such etiologies involve the craniofacial structures, neuromuscular tone, and other related factors. Collapsibility of the upper airway is influenced further by hormonal fluctuation (e.g., pregnancy or menopause), obesity, rostral fluid shifts, and genetic predisposition that influences craniofacial anatomy. OSA severity is heterogeneous among patients with the disorder. This wide range of presentation leads to variations in management approach and differences in treatment response.

Prevalence

Estimates of the prevalence of obstructive sleep apnea in adults vary in the literature; OSA commonly is thought to involve 14% of men and 5% of women. Prevalence rates are higher in certain populations, such as obese patients considered for bariatric surgery and post-stroke patients. Under-recognition of OSA likely leads to under-diagnosis and a false reduction of the true prevalence of disease.

Risk Factors

Individuals with certain characteristics appear to be predisposed to OSA. Conditions that may be risk factors for the development of OSA in adults include obesity, menopause, gender (male), and increasing age. [Obesity is considered to exist if the BMI is equal to or greater than 30] Genetic influences on craniofacial structure leads to higher OSA prevalence in certain ethnic groups that have been studied. Some genetic syndromes, particularly those with associated craniofacial anomalies, also are associated with an increased risk of OSA.

Craniofacial morphologies that may predispose to OSA include retrognathia, long and narrow faces, dolichocephalic facial type, narrow and deep palate, steep mandibular plane angle, anterior open bite, midface deficiency, and lower hyoid position. It should be noted, however, that the strength of the relationship between these craniofacial morphologies and the development of OSA is not well established.

Symptoms

Patients with obstructive sleep apnea often have a history of snoring, gasping respiration or choking, and witnessed pauses in breathing (apneas) during sleep. Common clinical symptoms of untreated obstructive sleep apnea include frequent nocturnal awakenings, non-restorative sleep, morning headaches, and excessive daytime sleepiness. Patients with OSA often describe difficulty with attention and concentration, mood disturbance, and difficulty controlling other medical comorbidities such as diabetes mellitus, hypertension, and obesity.

Diagnosis

Diagnostic confirmation of obstructive sleep apnea is performed by a sleep medicine specialist using the gold standard of an in-center overnight sleep study (polysomnography or PSG) or out-of-center sleep testing (OCST) for appropriately selected patients. Home sleep apnea testing (HSAT) is a type of OCST. Attended PSG includes at least 7 channels of recording, including electroencephalography (EEG), monitoring of sleep, airflow through the nose and mouth, pulse oximetry, respiratory effort, electrocardiography, and leg movement. HSAT includes 4-7 channels. It is important to note that HSAT typically does not include EEG monitoring of sleep.

According to the International Classification of Sleep Disorders¹ obstructive sleep apnea can be diagnosed by either of two sets of criteria. The first set of diagnostic criteria for OSA includes the presence of at least one of the following: (1) the patient has sleepiness, nonrestorative sleep, fatigue or insomnia symptoms, (2) the patient wakes with breath holding, gasping or choking, (3) a bed partner or other observer reports habitual snoring, breathing interruptions, or both, during the patient's sleep, (4) the patient has been diagnosed with hypertension, a mood disorder, cognitive dysfunction, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, or type 2 diabetes mellitus AND polysomnography or OCST shows 5 or more predominantly obstructive events (obstructive or mixed apneas, hypopneas, or respiratory effort related arousals (RERAs) per hour of sleep during a PSG or per hour of monitoring on OCST.

Secondly, obstructive sleep apnea can be diagnosed if PSG or OCST shows 15 or more predominantly obstructive events (obstructive or mixed apneas, hypopneas, or respiratory effort related arousals (RERAs) per hour of sleep during a PSG or per hour of monitoring on OCST). Examples of apnea and hypopnea are shown in Appendix 1.

A few different terms are used in the classification of obstructive sleep apnea. The respiratory disturbance index (RDI) includes the number of apneas, hypopneas, and RERAs per hour of sleep. The apnea-hypopnea index (AHI) includes the number of apneas and hypopneas per hour of sleep. Thus, a patient's RDI may be higher than the AHI. Some publications reference AHI while others reference RDI, so it is important for clinicians and researchers to understand the difference between these two measurements. Compared with PSG, OCST often underestimates the frequency of obstructive events per hour because OCST typically does not measure total sleep time as determined by EEG. The respiratory event index (REI) can be used to indicate the frequency of respiratory events based on total recording time (rather than total sleep time).

Severity

Severity of obstructive sleep apnea is classified based on the RDI; categories are mild (RDI ≥ 5 and < 15), moderate (RDI ≥ 15 and ≤ 30), and severe (RDI > 30). The minimum oxygen saturation also should be considered when making clinical assessment of the magnitude of obstructive sleep apnea, although there are no consensus classifications for the severity of oxygen desaturation.

Significance

Untreated OSA can lead to many serious consequences. Excessive daytime sleepiness increases the risk of motor vehicle accidents and diminishes quality-of-life. Neurocognitive impairment leads to decreased scholastic and occupational performance. Chronic intermittent hypoxemia and heightened sympathetic neural activity, endothelial damage and heightened inflammation are related to metabolic dysfunction and end-organ sequelae. Untreated obstructive sleep apnea increases risk of insulin resistance, coronary artery disease, congestive heart failure, myocardial infarction, hypertension, stroke, cardiac arrhythmia, and sudden cardiac death.

III. The Role of Orthodontics in Adult OSA

The orthodontist is well positioned to perform an OSA screening assessment and refer at-risk patients for diagnostic evaluation. Once the diagnosis of OSA is confirmed, physicians (and advanced practice providers supervised by physicians) may prescribe orthodontic appliances or procedures in appropriately-selected adult patients as part of OSA management.

Medical and Dental History

Orthodontists should be familiar with the signs and symptoms of OSA in adult patients. Thorough history-taking is critically important in this regard for this establishes the presence of pre-existing conditions, a basis for a diagnosis, the need for referral, and a baseline for evaluating the effects of treatment. Orthodontists also should include assessment of a patient's height, weight, and neck size to screen adult patients for OSA.

The following items should be considered when constructing a health history that is sensitive to OSA:

A prior diagnosis of Obstructive Sleep Apnea (OSA)	Excessive daytime sleepiness*
A prior diagnosis of other forms of Sleep Related Breathing Disorders (SRBD)	Fatigue during the day
Height*	Choking or gasping respirations during sleep
Weight*	Habitual or loud snoring*
Gender*	Observed episodes of pauses in breathing*
Age*	Abrupt awakening and shortness of breath
High blood pressure*	Awakening with dry mouth or sore throat
Mouth breathing	Morning headaches
Menopause	Difficulty staying asleep
Alterations in performance	Enuresis or unexplained Nocturia
Disordered mood	Attention or memory problems

Restlessness during sleep	Sweating
Nasal obstruction	Bruxism
Type 2 Diabetes	Neck Circumference

*Component of the STOP-Bang questionnaire

Screening Tools

In adults, a validated tool for OSA risk assessment is the STOP-Bang questionnaire (Appendix 2),²⁻³ which asks yes or no questions based on its acronym: snoring (S), tiredness (T), observed pauses in breathing (O), high blood pressure (P), BMI higher than 35 kg/m² (B), age older than 50 years (A), neck circumference of 17 inches or larger in males, or 16 inches or larger in females (N), and if patient's gender is male (G). A patient is considered to be at low risk for OSA if the questionnaire has 2 or less "yes" answers, at intermediate risk if there are 3 to 4 "yes" answers, and at high risk if there are more than 5 "yes" answers.

The patient also is considered at high risk if there are 2 "yes" answers from the STOP section, combined with either male gender, high BMI, or large neck size. For AHI ≤5, AHI between 5 and 15, and AHI ≥30, the sensitivities were 84%, 93%, and 100% and the specificities were 56%, 43%, and 37%, respectively. The STOP-Bang Questionnaire has a high sensitivity for identifying patients with moderate-to-severe OSA. This sensitivity gives the practitioner an excellent tool for identifying patients who have the condition. This questionnaire can be completed in a few minutes when incorporated into an orthodontist's workflow.

Clinical Examination

The clinical examination is an important part of the screening process. In addition to regular orthodontic screening, the orthodontist can use the Modified Mallampati Classification to describe the patency of the oral airway (Appendix 3).⁴⁻¹⁰ Three steps are followed to determine the MM Class: Step 1. Patients are asked to take a seated or supine position. Step 2. Patients are asked to protrude their tongue as far forward as they can without emitting a sound. Step 3. The examiner observes the relationship between the palate, tongue base and other soft tissue structures to determine the MM Classification defined as Class I: Soft palate, fauces (the arched opening at the back of the mouth leading to the pharynx), uvula, and tonsillar pillars are visible; Class II: Soft palate, fauces, and uvula are visible; Class III: Soft palate and base of uvula are visible; Class IV: Soft palate is not visible.

This clinical assessment framework can help orthodontists identify patients who may be at risk for upper airway obstruction during sleep. It should be noted that the MM Class may vary over the course of a pregnancy, so the MM Class may need to be reassessed at various times during pregnancy. The Modified Mallampati Classification is a helpful part of the OSA screening process; it should not, however, be used in isolation to predict OSA presence or severity.

Many other OSA screening questionnaires have been developed and studied in various populations, with wide ranging specificities and sensitivities. The Epworth Sleepiness Scale (Appendix 4)¹¹ asks patients to self-rate their level of sleepiness in eight different sedentary

situations. The Epworth Sleepiness Scale may be used to gauge or track symptomatic impairment (or response to treatment). However, it is not a screening tool for OSA, as it detects abnormalities in level of daytime sleepiness regardless of the cause of sleepiness.

Practitioners also may find the Friedman Tongue Classification System (Appendix 5),¹² the Kushida Index,¹³ and the Berlin Questionnaire for Sleep Apnea¹⁴ useful.

Orthodontic Radiographs

The use of imaging in the assessment of OSA is often limited in a typical orthodontic setting. Conventional cephalometric images are dimensionally limited. Thus, airway imaging using a lateral cephalogram does not portray mediolateral information in the oropharyngeal airway and may give misleading information as to the volume and minimal cross-sectional area.

Cone Beam Computed Tomographic (CBCT) images have been shown to be useful in diagnostic and morphometric analysis of the hard and soft tissues in routine orthodontic treatment, but they have certain limitations regarding the diagnosis of OSA. CBCT provides no information on neuromuscular tone, susceptibility to collapse, or actual function of the airway. There are significant positional and functional differences when the patient is asleep versus awake. It is a snapshot of a specific moment of the breathing cycle. Additionally, there currently is no minimal cross-sectional area or volume of the airway that has been validated as a minimal threshold level at which an individual is at higher risk of having OSA. Thus, orthodontic records may be taken by the orthodontist, but currently no radiographic methods have been reported to have high enough sensitivity or specificity to serve as a risk assessment tool for OSA.

Three-dimensional imaging of the airway should not be used to diagnose sleep apnea or any other sleep-related breathing disorders because such imaging currently does not represent a proper risk assessment technique or screening method. On the other hand, three-dimensional imaging of the airway, when available, may be used for monitoring or treatment considerations. If radiographic records are taken as part of orthodontic diagnosis and treatment planning, the airway and surrounding structure should be analyzed comprehensively.

IV. Diagnosis and Treatment Planning in Adult OSA

Obstructive sleep apnea and other sleep-related breathing disorders can only be definitively diagnosed by a physician. It is not in the scope of the orthodontist or any other dentist to definitively diagnose obstructive sleep apnea or any other sleep-related breathing disorder. If the patient is found to have OSA, the physician will prescribe the appropriate course of action; the orthodontist should consider working in a collaborative way with the physician, providing related orthodontic treatment when necessary, as long as it does not interfere with medical treatment.

The OSA treatment plan should be based on careful consideration of the patient's individual needs and treatment goals. If the treatment plan involves orthodontics, a plan for treatment, monitoring, and long-term follow up care should be developed by all practitioners involved.

Care should be coordinated via communication between the orthodontist and any other practitioners participating in the treatment of the patient. It is recommended that treatment and/or management of obstructive sleep apnea not take place without a referral from a physician (or provider supervised by a physician).

V. Treatment of OSA in Adults by Physicians and Surgeons

Positive airway pressure (PAP) therapy is the gold standard treatment method for obstructive sleep apnea in adults. PAP acts as a pneumatic splint that maintains patency of the upper airway. PAP is delivered through a mask interface as either continuous positive airway pressure (CPAP), bi-level positive airway pressure (BPAP) or auto-titrating positive airway pressure (APAP). Of note, CPAP and BPAP devices are available in conventional and auto-titrating modes. CPAP use can decrease OSA-related cognitive impairment along with improving objective and subjective measures of sleepiness, particularly in patients with severe OSA (AHI $\geq$ 30/hour).¹⁵ BPAP may be used for patients with OSA who are intolerant to CPAP or those who have other forms of sleep-related breathing disorders (e.g., sleep-related hypoventilation). APAP may be considered for patients with OSA patients who do not have contraindications to APAP use (e.g., congestive heart failure, lung disease such as chronic obstructive pulmonary disease, obesity hypoventilation syndrome, or central sleep apnea).

Studies on PAP non-adherence report wide ranging results. While definitions of non-adherence vary across studies, a common definition of PAP non-adherence is mean use $\leq$ 4 hours per night. Estimates of PAP non-adherence range from 29% to 83%.¹⁶⁻¹⁷ Early adherence to PAP use predicts longer-term PAP use; a study of 100 patients started on CPAP showed that CPAP use for at least 4 hours per night 3 days after starting therapy was predictive of CPAP adherence 30 days after treatment initiation.¹⁸ Factors that affect PAP adherence include OSA severity, ability to tolerate the prescribed pressure setting, mask fit, spousal support, and other psychological and social influences.¹⁹

Other treatment options include positional therapy (avoidance of back-sleeping) and long-term weight reduction as indicated. Nasal congestion and allergic rhinitis may be managed with nasal steroids and other oral medications as indicated. For some patients, nasal surgery may be performed as adjunctive therapy to decrease intranasal resistance and facilitate better adherence to PAP therapy. For selected patients, multilevel surgery including nasal and/or palatal surgery with or without mandibular surgery, genioglossus advancement, and hyoid suspension may be considered. Other soft tissue surgeries might be indicated that involve the tonsils, adenoids, frenula, and tongue. Hypoglossal nerve stimulation addresses the impaired neuromuscular tone in obstructive sleep apnea and may be considered in certain patients with OSA.

VI. Orthodontic Management in Adult OSA

Following diagnosis of OSA by a physician, a patient may be referred to (or back to) an orthodontist for one or more types of care.

Informed Consent

Prior to initiating care, informed consent appropriate to OSA must be obtained before any treatment is provided. The proposed treatment plan should be described in detail, and treatment alternatives also should be discussed. The orthodontist should describe the benefits, risks, short and long-term side effects, and complications that might arise. The need for compliance, long-term monitoring, and follow-up care should be discussed. An estimate of the nightly duration of oral appliance therapy use should be provided, and a realistic estimate of the probability of success with the treatment protocol should be presented. Given the serious nature of untreated OSA, it is recommended that the orthodontist carefully document the informed consent process.

Oral Appliance Therapy

Oral appliances (OA), which include both mandibular advancing oral appliances (OAm) and tongue retaining devices, are usually effective options for OSA management in appropriately selected patients. OAm are intended to hold the mandible and/or the associated soft tissues forward, resulting in an increased caliber of the upper airway at the oropharyngeal level. A substantial body of research supports the use of oral appliances for patients with OSA. Specifically, OAs may be used for treatment of mild to moderate OSA and for treatment of patients with severe OSA who are unwilling or unable to use PAP therapy. Published guidelines (American Academy of Sleep Medicine/American Academy of Dental Sleep Medicine) describe how oral appliances fit into the OSA management paradigm.²⁰⁻²¹

Functional appliances and OAm are considered the first line of treatment for patients with OSA that prefer OA over PAP and for those patients that do not respond to PAP therapy. While typically well tolerated, it should also be noted that not all patients with OSA will respond to OAm treatment, with this form of therapy reported to be completely effective in 36% to 70% of OSA cases.

Many types of oral appliances are used in the treatment of OSA in adults. The appliances vary based on the coupling design, mode of fabrication and activation, titration capability, degree of vertical opening, lateral jaw movement, and whether they are custom made or prefabricated. Proper indications for each design should be considered.

Oral Appliance Titration

Oral appliances initially are delivered with the mandible advanced to a position approximating 2/3 of maximum protrusion. After a period of accommodation, based on subjective feedback from the patient regarding their OSA symptoms and sleep quality, the amount of protrusion can be titrated or increased until optimum symptom relief is obtained. Unattended (type 3 or 4)

portable monitors may be employed by the orthodontist to help define the optimal target position of the mandible. Then typically the physician involved will request a sleep study with the OAm in place. Should the physician deem the calibrated position to be sub-therapeutic, the physician and orthodontist should discuss the possibility of further titration or alternative treatment.

Monitoring

During treatment for OSA, the patient should be monitored, which may involve subjective reports as well as objective observations. Reports on usage of the OA may be obtained from the patient and bedpartner or caregiver. Compliance should be evaluated, and the appliance should be checked for fit and comfort, the need for titration, and the development of undesirable side effects. At present, most data on adherence to OA therapy rely on subjective reports. Use of a thermal sensor²³ has been studied in an effort to have objective measurement of OA adherence, although such measures currently are not part of routine clinical care.

It has been suggested that monitoring be conducted at least once every 6 months during the first year and then annually. Routine monitoring should result in regular communications between the physician and orthodontist. If the patient has worsening of OSA-related symptoms, and/or changes to overall health, a consultation with the physician is strongly recommended.

The Goals of Treatment

The endpoints of treatment include:

- Reduced or eliminated snoring
- Resolution of the patient's initial symptoms of OSA
- Normalization of the AHI
- Normalization of oxyhemoglobin saturation

No pretreatment risk factors have been consistently shown to predict success for oral appliances in reaching treatment goals.

Change in Occlusion

Oral appliances used in sleep apnea treatment move teeth. In the global field of dentistry, orthodontists generally are considered the experts in the management of malocclusion because of their education and clinical experience. Improved awareness of both OSA and the effectiveness of oral appliances has resulted in increased numbers of OSA patients being treated with oral appliances by non-orthodontists. While successful OSA treatment may be evident over the short term in many of these patients, non-orthodontic providers may be unaware of the unwanted effects of OAs can have on their patient's occlusion over the long term. Orthodontists can be helpful in providing our medical and dental colleagues valued oversight, and sometimes treatment, of unexpected and unwanted occlusal changes occurring with long-term oral appliance wear.

Typical changes include a reduction in overjet and overbite, changes in facial height, development of anterior crossbites, and posterior openbite. Changes are progressive with ongoing oral appliance use. In that many patients ultimately will be treated for a protracted period of their lifetime, appliance-generated malocclusions often become significant over the long term and may require treatment to reverse the dentoskeletal adaptations that may occur.

Orthodontists may be asked to assess and treat oral appliance-related malocclusions, a condition that has become a more frequent occurrence in recent years. When considering treatment of these malocclusions, orthodontists need to be aware that the patient will not be able to wear their oral appliance during treatment; therefore, the patient may need to use PAP therapy during the period of orthodontic care. Communication with the physician helps ensure the patient's OSA is still being managed appropriately.

Should the patient return to using an oral appliance for OSA following orthodontic treatment, then the malocclusion may also return. Consequently, such patients often switch to PAP therapy or can be evaluated for surgical treatment options.

MMA and SARME

Patients who are unable to tolerate or adhere to PAP and/or oral appliance therapy with an underlying sagittal skeletal discrepancy may be candidates for maxillomandibular advancement (MMA) or telegnathic (>10mm) jaw advancement surgery. MMA generally is reserved for patients with severe OSA who are unable to tolerate PAP therapy, and for those patients who also have an orthodontic indication for the procedure. The severity of OSA is not the only determinant of candidacy for MMA; these patients often require detailed evaluation and counseling before MMA is selected as a treatment option.

Such patients typically should proceed with routine orthodontic diagnosis and treatment planning, including comprehensive soft tissue facial evaluation to assure optimal pre-surgical preparation and that the surgery performed will not affect facial esthetics adversely. Orthodontic care is usually a beneficial adjunct for patients to facilitate obtaining optimal occlusion while simultaneously reducing the risk of post-operative malocclusion. Patients with ideal or minimal Class I malocclusion may not require extensive pre-surgical orthodontics in that the two jaws may have a similar interdigitation following symmetric maxillary and mandibular advancement. Telegnathic surgery is not recommended for patients who are already bimaxillary protrusive; such patients should usually be reevaluated by the team to explore alternative treatment options. One of the concerns of telegnathic surgery in this situation involves esthetics. As such, each practitioner and patient should decide for themselves if the benefits of the surgery outweigh the risks involved.

Significantly less data exists for surgically assisted rapid maxillary expansion (SARME), which aims to correct a maxillary transverse deficiency. In OSA patients with maxillary transverse deficiency, normalizing the width of the maxilla with SARME and developing a functional and esthetic occlusion with comprehensive orthodontic treatment afterward has been suggested to improve PSG parameters.²²

Possible Treatments on the Horizon

New treatment modalities such as mini-implant (aka, miniscrew or temporary anchorage device) supported rapid maxillary expansion (MARME) are appearing as possible alternatives for surgical assisted rapid maxillary expansion. However, to date there is very limited PSG evidence for its use in the management of OSA patients. Future studies are needed, and with time mini-implant supported expansion may become a viable adjunctive form of treatment for OSA management in adult patients.

VII. Pediatric OSA (Under 18 Years of Age)

Etiology

As with adult OSA, impaired neuromuscular tone underlies upper airway collapsibility in children. In addition to etiologic factors similar to those in adults, exacerbating factors for pediatric OSA often include lymphoid hyperplasia and growth-related changes in the size of the upper airway.

As the upper airway is narrowed or completely occluded, the patient's effort during breathing progressively increases. Due to the airflow restriction, there is a relative increase in serum carbon dioxide (hypercarbia) and decrease in serum oxygen (hypoxemia). The escalating respiratory effort causes a cortical arousal from sleep, which results in the upper airway opening so that normal airflow is reestablished. Once the patient falls back asleep, the upper airway may collapse again with recurrence of the above-noted process. This breathing sequence may have significant consequences for the child.

Risk Factors

As the obesity epidemic also affects children, obesity (BMI equal to or greater than 30) is becoming a greater factor for childhood OSA. However, as untreated OSA may contribute to growth restriction, some children with OSA paradoxically may be underweight. Thus, it is recommended that a clinical risk assessment for OSA be performed even in normal weight or underweight children.

In addition, it is thought that certain craniofacial morphologies can increase a child's risk for having OSA. For instance, mandibular retrognathia, long and narrow faces, narrow and deep palate, steep mandibular plane angle, anterior open bite, and midface deficiency may predispose a child to develop OSA. However, the presence of OSA cannot be determined by craniofacial morphology alone; these physical findings should be interpreted in the context of the clinical history.

Genetic syndromes that are associated with craniofacial anomalies can confer an increased risk of OSA. For example, patients with Pierre Robin sequence²⁴ and syndromic craniosynostosis²⁵ have a high prevalence of OSA. Children with Down syndrome²⁶ also have an increased OSA prevalence. Orthodontists who care for children with these and other genetic syndromes that

affect craniofacial morphology should pay attention to clinical features that may suggest the presence of untreated OSA.

Symptoms

Children with OSA may present with snoring, witnessed apneas, and choking or gasping during sleep. Parents or caregivers may describe that the child sleeps in unusual positions, such as having the neck hyperextended or with the head hanging off the side of the bed, as well as appearing very restless with frequent position changes during sleep.

Some children with OSA may present with sleepiness; those who previously had discontinued daytime napping may resume daily or near-daily naps. In other children, untreated OSA may manifest as hyperactivity rather than excessive sleepiness. While obesity may be a contributor to the pathogenesis of OSA in some children, others may present with failure to thrive. As such, it is recommended that the evaluation for OSA in every child should be part of an orthodontist's comprehensive clinical assessment.

Diagnosis

Diagnosis of OSA in children is confirmed only by the gold standard PSG. Diagnostic evaluation of childhood OSA has evolved in recent years. In addition to standard recording channels, all pediatric PSG is now conducted with carbon dioxide (CO₂) monitoring. Measurement with either end-tidal CO₂ (the partial pressure of CO₂ present at the end of exhalation) or transcutaneous CO₂ monitoring also is acceptable.

According to the International Classification of Sleep Disorders,¹ obstructive sleep apnea can be diagnosed by either of two sets of diagnostic criteria. The first set of criteria for obstructive sleep apnea includes the presence of at least one of the following: (1) snoring, (2) labored, paradoxical, or obstructed breathing during the child's sleep, or (3) sleepiness, hyperactivity, behavioral problems, or learning problems AND polysomnography shows one or more obstructive apneas, mixed apneas, or hypopneas per hour of sleep.

Alternatively, obstructive sleep apnea can be diagnosed if the PSG shows a pattern of obstructive hypoventilation, which is defined as at least 25% of total sleep time with hypercapnia (PaCO₂ > 50 mm Hg) associated with at least one of the following: (1) snoring, (2) flattening of the inspiratory nasal pressure waveform, or (3) paradoxical thoracoabdominal motion. These OSA diagnostic criteria are for children below age 18 years, though adult OSA diagnostic criteria may be used for children ages 13-18 years per the American Academy of Sleep Medicine Manual for the Scoring of Sleep and Associated Events.²⁷

HSAT is not indicated in patients less than 18 years of age.²⁸⁻²⁹

Severity

Published studies on childhood OSA have included various diagnostic criteria; some studies use the adult criteria of AHI ≥5/hour. Other studies define childhood OSA as mild (AHI 1-5/hour), moderate (AHI 5-10/hour) and severe (AHI ≥10/hour). Of note, scoring of obstructive apneas

and hypopneas on PSG differs slightly for children compared with adults. For adults, event duration is at least 10 seconds whereas for children obstructive event duration is defined as at least 2 breaths.

Prevalence

Prevalence of childhood OSA is obscured by different diagnostic criteria used in published studies. Epidemiologic data from 2008 indicate prevalence of parent-reported “always” snoring to be 1.5% to 6%, prevalence of parent-reported apneic events during sleep to be 0.2% to 4%, and OSA diagnosed by varying criteria to be 1% to 4%. Multiple studies have shown that during certain phases of growth, childhood OSA remits without any intervention. These data indicate that prevalence of childhood OSA changes across periods of growth and development. Specific populations, such as children with certain craniofacial or other genetic syndromes and those who are obese, have a higher prevalence of OSA compared with the general population.

Significance

Consequences of OSA in children include impaired growth and cardiovascular dysfunction. The impaired neurocognitive function seen in children with untreated OSA can have an effect on academic performance. Behavioral problems also can result. Persistent snoring and nocturnal enuresis (bedwetting), which can result from untreated OSA, can be embarrassing for children in social settings and thus affect interpersonal interactions.

VIII. Pediatric OSA: Skeletal and Soft Tissue Growth

Orthodontists are aware of the impact facial growth has on orthodontic treatment outcome. Facial growth also influences the size and shape of the upper airway in the pediatric population. One approach to understanding the interaction of hard and soft tissue growth on upper airway morphology can be described as follows. The hard tissue boundaries of the upper airway include the upper and lower incisors and the piriform rim in the anterior, the cranial base superiorly, the cervical vertebrae posteriorly and the hyoid bone inferiorly. Laterally, the size of the airway is related to the width of the palate, the middle cranial fossa, and the distance between the ascending rami. Together these structures define the bony skeletal boundaries of the airway. Soft tissues then line this hard tissue framework. These tissues include the pharyngeal muscles, the tongue, the soft palate, the turbinates, as well as the pharyngeal tonsils, adenoids, and nares.

Importantly, growth of the bony components effectively increases the size of the skeletal boundaries in the following ways. The anterior cranial base increases in length via growth at the spheno-ethmoidal synchondrosis up to age 7 years. Increases in posterior cranial base length are similarly related to growth at the spheno-occipital synchondrosis up to age 13 years. The anterior cranial base carries the nasomaxillary complex forward at the same time as the individual bones of the midface are displaced in an anterior and inferior direction. Simultaneously, the mandible elongates and is displaced downward and forward with

deposition of bone on the posterior and superior borders of the ramus, increasing the height of the rami (bony pharyngeal height) and increasing the distance between the ascending rami (bony pharyngeal width). Concurrently, resorption on the anterior border of the ramus increases corpus length (oropharyngeal length). While all these bony changes are occurring, the hyoid bone also is displaced anteriorly and inferiorly. Thus, the normal facial growth process results in dramatic increases in all three dimensions of the skeletal framework.³⁰

While the skeletal boundaries of the airway are increasing, the major lymphatic tissues of the upper airway (tonsils and adenoids) are shrinking. This combination of increases in skeletal dimensions along with decreases in soft tissue mass results in enormous increases in the size of the upper airway over infancy, childhood, adolescence, and teenage years. These changes in airway due to growth far exceed any orthodontic or orthopedic effects on airway shape and/or size. Knowledge of these changes is important in the understanding of the dynamics of OSA in children.³¹

IX. The Role of Orthodontics in Pediatric OSA

It is strongly recommended that the orthodontist perform a clinical risk assessment for OSA and refer at-risk patients to the appropriate physician for definitive diagnosis of OSA. Subsequently, orthodontists may be involved in treatment of pediatric OSA if the treating physician refers the patient back to the orthodontist to address an underlying skeletal discrepancy thought to contribute to the child's OSA.

Medical and Dental History

Orthodontists should be familiar with the signs and symptoms of OSA in pediatric patients. Questions concerning the health history of a pediatric patient should solicit information on snoring, sleep-related behaviors, daytime sleepiness, difficulty concentrating, and/or formal diagnosis of attention deficit hyperactivity disorder. The American Academy of Pediatric Sleep Physicians recommends that if a patient reports snoring, more thorough questioning is warranted; the guidelines state, "If they snore, you must do more."³²

A thorough history and examination are critically important in that they establish the presence of pre-existing conditions, a basis for a diagnosis, the need for referral, and a baseline for evaluating the effects of treatment. Orthodontists also should include assessment of a patient's height, weight, and neck size to screen pediatric patients for OSA.

The following items should be considered when performing a pediatric evaluation that is sensitive to OSA:

A prior diagnosis of Obstructive Sleep Apnea (OSA)	Loud snoring
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A prior diagnosis of other forms of Sleep Related Breathing Disorders (SRBD)	Mouth breathing during sleep
Height	Poor school performance
Weight	Aggressive behavior
Medications	Developmental delays
Age	Bed wetting that is not age-appropriate
Attention problems	Hard to wake up in the morning
Trouble breathing during sleep	Morning headaches
Pauses in breathing during sleep	Fall asleep quickly
Nasal obstruction	ADHD

Screening Tools

One potential screening tool that has been validated and used in orthodontic offices is the Pediatric Sleep Questionnaire (PSQ)(Appendix 6).³³⁻³⁵ This questionnaire has a positive predictive value of 0.4 (i.e., patients with a positive PSQ score will be diagnosed with OSA 40% of the time) and a negative predictive value of 0.99, (i.e., patients with a negative PSQ score will only have a 1% chance of being diagnosed with OSA). The PSQ often is a valuable first step in screening patients presenting to the orthodontic office without a history of OSA. The Epworth Sleepiness Scale for Children and Adolescents (Appendix 7)³⁶ may be helpful to assess for problematic sleepiness, but this tool cannot identify a specific cause of daytime sleepiness. The Epworth scale has been validated only for children ages 12-18 years.¹¹

Clinical Examination

In addition to the usual orthodontic clinical examination that evaluates the dental occlusion, range of mandibular motion, soft tissue frenum attachments, gingival health, and TMD, the orthodontist should also note the degree to which the tonsils impinge on the pharyngeal airway. A commonly accepted tonsil classification system called the Brodsky scale grades the clinical manifestation of tonsil hypertrophy from 1 to 5 based on the percentage of the oropharyngeal airway taken up by the two tonsils (Appendix 8).³⁷ The Friedman Tonsil Grading System (Appendix 9)³⁸ also may be a useful tool to evaluate the size of the tonsils. As tonsil size does not correlate to OSA severity, there is no set cut-point about which level of tonsillar hypertrophy necessitates a referral to an otolaryngologist (ENT physician) for further evaluation;³⁹ thus, this decision is best made in the patient-specific context of symptoms and physical exam findings. The clinical evaluation of OSA in children should include evaluation of

tongue size and position, the presence of obesity, and the patient's overall growth and development.

Orthodontic Records

The typical orthodontic record set captures some important information that can be useful for further evaluation of the upper airway. For example, the adenoid mass and the hyoid bone can be seen on both the lateral cephalogram and the cone beam computed tomographic (CBCT) image. A low position of the hyoid bone when measured from the inferior border of the mandible has been shown to be an indicator of low muscle tonicity and has been linked with OSA.

Three-dimensional imaging is more accurate than two-dimensional imaging for assessment of airway volume and area of maximum constriction. Airway imaging using a cephalogram does not portray mediolateral changes in the oropharyngeal airway and may give misleading information as to the volume and minimal cross-sectional area. As in adult patients, while CBCT images have been shown to be useful in diagnostic and morphometric analysis of the hard and soft tissues in routine orthodontic treatment, there are limitations regarding the screening of OSA. CBCT provides no information on neuromuscular tone, susceptibility to collapse, or actual function of the airway. Although both two dimensional and three-dimensional imaging of the airway are helpful, they cannot be used to diagnose sleep apnea or any other sleep-related breathing disorders alone, and they do not provide a proper risk assessment technique or screening method.

Importantly, there is no direct link between any radiographic measures of airway size or shape and PSG results. Therefore, imaging values should be interpreted cautiously and in conjunction with other clinical signs and symptoms. Three-dimensional imaging of the airway, when available, also may be used for monitoring or treatment planning. If radiographic records are taken for orthodontic purposes, the airway and surrounding structures, specifically the adenoids in children, should be evaluated.

X. Diagnosis and Treatment Planning in Pediatric OSA

As mentioned previously, orthodontists should not assume the responsibility for the definitive diagnosis of OSA. The definitive diagnosis is appropriately made by a physician. If the patient is found to have OSA, the physician should decide on an appropriate course of action for the treatment of OSA. The orthodontist may choose to work in a collaborative way with the physician, providing orthodontic treatment when necessary and when it does not interfere with ongoing medical treatment.

The plan for treating pediatric OSA should be based on consideration of the patient's individual needs and treatment goals. If the OSA treatment regimen involves orthodontics, a plan for treatment, monitoring, and long-term follow up care should be considered by all medical and dental practitioners involved. Care should be coordinated via communication between the

orthodontist and all other practitioners who are working to treat the patient's obstructive sleep apnea.

The orthodontic treatment plan for patients with OSA should follow the same orthodontic principles for correction of dental and skeletal deformities. Two orthodontic procedures that may change upper airway physiology are rapid maxillary expansion (RME) and mandibular advancement appliances for Class II correction. With both types of interventions, the primary objective of the orthodontic appliance should be to improve the occlusion and address the underlying skeletal discrepancy.

It would be appropriate, for example, to recommend rapid maxillary expansion (RME) for patients diagnosed with maxillary transverse deficiency. In this situation, the primary treatment goals would be to normalize the transverse width of the maxilla and establish a normal occlusion. Secondary effects of this treatment may result in reduction of nasal airway resistance and increase in the volume of the nasopharynx and nasal cavity. Both secondary effects of RME have the potential to improve OSA.

In the case of mandibular advancement devices for mandibular retrognathia, the primary goals should be to correct the skeletal discrepancy and the Class II molar relationship. A secondary effect of mandibular advancement devices may be the increase in the caliber of the oropharyngeal airway. The same applies to maxillary advancement appliances used in the treatment of Class III malocclusions.

It is possible that an OSA patient might be referred for expansion but does not have a transverse discrepancy. Likewise, it is possible a patient with OSA might be referred for mandibular advancement (or maxillary advancement) where no sagittal discrepancy exists. In such situations, the treatment alternatives should be considered on a case-by-case basis by the medical and dental practitioners involved. In such situations, it is appropriate to prioritize the treatments to serve the best interests of the patient.

XI. Treatment of Pediatric OSA

In the growing child, OSA management is dramatically different than for the adult. It is recommended that orthodontists become aware of the vast array of potential treatment modalities that are available and that they work in unison with medical and dental practitioners when managing pediatric OSA. Hypertrophic tonsils and/or adenoids are the most common risk factor for OSA in the pediatric population, with tonsillectomy and/or adenoidectomy typically considered the first-line of treatment.

Various forms of pharmacologic agents may be prescribed by the attending physician to reduce the size of the nasal soft tissues if there is suspicion of these tissues being a potential cause of OSA. Nasal surgery, including turbinate reduction and deviated septum correction, also may be considered in selected cases. For the obese child, weight reduction management should be considered as part of the treatment plan. Positive airway pressure (PAP) also may be utilized in

severe cases. Possible negative craniofacial consequences of longitudinal usage of PAP on the developing facial structures should be considered.

Dentofacial orthopedic management, which is within the scope of the orthodontic specialist, also may be considered. For instance, rapid maxillary expansion is a well-known orthodontic treatment option for patients with a narrow maxilla. There is growing evidence, although of low-level, that in mixed-dentition patients who are properly diagnosed with OSA, RME can decrease AHI in the short and long term.⁴⁰ Unfortunately untreated control groups generally were not used in the studies considered. Regardless of the presence of OSA, it is recommended the orthodontist use these devices only when there is an appropriate underlying skeletal condition. There is no indication in the literature that prophylactic application of maxillary expansion prevents the future development of OSA.

Based on a few studies that were performed on mixed dentition samples, mandibular anterior repositioning appliances also can produce a decrease in AHI. Long-term stability of these changes has not been studied. Untreated control groups generally were not used in these studies as well. Regardless of the presence of OSA, it is recommended the orthodontist use these devices only when there is an indication that a related retrognathic condition exists. There is no clear indication in the literature, however, that prophylactic use of mandibular anterior repositioning appliances prevents later development of obstructive sleep apnea.

In addition, the orthodontist should be aware that some children who remain PAP intolerant may require airway support while sleeping. The use of mandibular advancing devices may be prescribed by the physician, and this prescription is not predicated solely on Angle's classification of occlusion. In this instance, treatment with an oral device is directed primarily toward airway maintenance and less so with dentofacial orthopedic management. Careful monitoring of facial growth and development is important during this time.

For Class III patients, there are no studies that have assessed the impact of maxillary protrusion on AHI. Only assessment of pharyngeal dimensions has been published so far. It appears inappropriate for the clinician to make the jump from enlarged airway dimensions to improvement in airway function or sleep-related breathing parameters. Regardless of the presence of OSA, it is recommended the orthodontist use these devices when there is an underlying skeletal issue.

Orthognathic surgery usually is not indicated until craniofacial growth is completed. As a result, the pediatric patient that presents with clear skeletal issues should typically be managed to adulthood in the normal fashion with corrective jaw surgery planned later when the timing of the surgery is appropriate. An exception might be considered in a case where the patient has OSA and a severe skeletal discrepancy. After considering the potential benefits and risks involved (including the need for later surgical revision), orthognathic or telegnathic surgery could be considered.

In summary, much is known regarding treatment for OSA in adults. In contrast, information on the treatment of OSA in pediatric patients is much more limited. Thus, care should be taken regarding the indications for orthodontic and orthopedic treatment intended to treat OSA in the young patient. Clearly defined treatment goals, focusing on the orthodontic and orthopedic

components, should be articulated to the responsible parties involved. Improvement of the OSA should be highlighted as a “possible,” or some studies say “anticipated,” outcome of treatment. But, no guarantees of OSA resolution can be implied or stated emphatically by the treating orthodontist.

XII. Fallacies About Orthodontics in Relation to OSA

Conventional orthodontic treatment never has been proven to be an etiologic factor in the development of obstructive sleep apnea. When one considers the complex multifactorial nature of the disease, assigning cause to any one minor change in dentofacial morphology is not possible. However, misinformation exists regarding the potential airway related sequelae of orthodontic treatment performed with dental extractions or orthopedic headgear.

The specific effects on the dental arches and the muscles and soft tissues of the oral cavity following orthodontic extractions can differ significantly, depending on the severity of dental crowding, the amount of protrusion of the anterior teeth and the specific mechanics used to close the extraction spaces. The indication for extractions varies from patient to patient, as does the resulting change to the width, length and arch perimeter of the dentition—all may increase, decrease, or stay the same following treatment. The impact that orthodontic treatment with or without dental extractions may have on the dimensions of the upper airway also has been examined directly, first with two-dimensional cephalograms and more recently with three-dimensional CBCT imaging.⁴¹

In certain instances, namely in patients with significant protrusion of both upper and lower anterior teeth where skeletal anchorage or extractions are used to retract the anterior teeth as much as possible to reduce lip protrusion in profile, reductions in the cross-sectional area of the oropharynx have been reported. More frequently, as in patients where extractions are performed to help address dental crowding or improve the occlusion, there is no discernible change in airway dimensions when extractions are employed.⁴²⁻⁴³ The studies examining these effects in children and adolescents have reported increases in airway volumes and cross-sectional areas in patients both with and without extractions performed as part of their orthodontic treatment.⁴⁴⁻⁴⁶ These effects may likely be related to normal growth changes.

In discussing orthodontic treatment to changes in the dimensions of the upper airway, it also is helpful to understand that an initial small or subsequently reduced or increased size does not necessarily result in a change in airway function. Reflecting the higher significance of neuromuscular control on airway function during sleep, it has been demonstrated that a narrow airway does not result in OSA, but rather it is an inability for a patient’s airway muscles to compensate adequately that leads to obstruction and sleep-disordered breathing.⁴⁷

As such, future investigations should aim to place greater emphasis on the effects of airway function following orthodontic treatment, as opposed to focusing solely on quantifying airway dimensions. One such study assessed dental extractions as a cause of OSA later in life with a large retrospective examination of dental and medical records.⁴⁸ Researchers reviewed the health records of more than 2700 adults with four missing premolars and evaluated whether

this group had a higher prevalence of OSA compared to an equally sized group of patients with no missing teeth who also were matched for the most significant confounding variables of OSA in adults, namely age, BMI and gender. The study concluded that the prevalence of OSA was essentially the same in both groups, and that dental extractions were not a causative factor in OSA.

Overall it can be stated that existing evidence in the literature does not support the notion that arch constriction or retraction of the anterior teeth facilitated by dental extractions, and which may (or may not) be the objective of orthodontic treatment, has a detrimental effect on respiratory function.

Headgear Therapy

Growth modification, including orthopedic headgear (HG), which alters the direction of growth of the maxilla, has long been a staple of certain orthodontic treatments. While dentoalveolar movement can be significant, the absolute skeletal change to the position of the maxilla elicited by headgear is relatively small. Consequently, meaningful effects on volume or morphology of the upper airway should not be expected. A few studies with small sample sizes or methodological limitations have examined this relationship directly. The best evidence available at this time, however, indicates that headgear does not pose an increased risk to the airway in that the airway remains the same or increases over the study periods reported.

Anecdotal concern exists about whether headgear used during adolescence could contribute to the future development of OSA as an adult. To date, no studies have been performed using objective PSG to demonstrate an elevated risk of OSA in HG patients. Studies have investigated this concern indirectly by evaluating the radiographic airway in two dimensions with lateral cephalograms on HG patients. One study⁴⁹ concluded that the absolute value of the airway dimension was smaller in HG patients than activator patients, but these differences were both small and not statistically significant. A longitudinal study, examined patients over a twelve-year period reported that the radiographic dimension of the airway decreased during the treatment phase but increased to the level of controls during follow-up.⁵⁰ A prospective, randomized, blinded study,⁵¹ demonstrated an increase in airway during the six-year study period. In summary, the best evidence available at this time indicates that HG does not pose an increased risk to the airway in that the airway dimension remains the same or increases over the study periods reported.

Frenectomy

Functional deficits regarding suction, swallowing, masticatory and speech difficulties are known consequences of ankyloglossia or tongue-tie. However, uncertainty remains as to what degree of frenum attachment will contribute to a deviation of normal form or function in all but the most severe forms of ankyloglossia. More recently a four-point severity scale of tongue mobility was reported, with the most severely restricted tongues graded as 4.⁵² These investigators have reported a reduced maxillary intercanine width and a longer soft palate in patients with more severe levels of tongue restriction, compared to patients with no such restriction. However, the relationship between tongue mobility and function of the airway indeed is complex. Future research efforts should aim to assess airway function during sleep as

it relates to tongue mobility. At this time, frenectomy remains an appropriate treatment for speech and mastication deficiencies, but such procedures are not supported as a treatment to prevent future development of OSA.

XIII. Legal Issues

Obstructive sleep apnea is a medical disorder that can have serious consequences on overall health. Given some of the possible medical conditions associated with OSA, it is strongly recommended that orthodontists work with qualified and appropriately trained physicians in addressing obstructive sleep apnea.

With that in mind, it is strongly recommended that orthodontists screen orthodontic patients for known OSA risk factors. Should the screening indicate an elevated risk for having OSA, it is strongly recommended that the patient be referred to an appropriate physician for a definitive OSA diagnosis and treatment. Depending on the physician's diagnosis and plan for treatment, the orthodontist may be involved in the treatment by proper referral by the physician.

Any orthodontist involved in the treatment of adult and/or pediatric obstructive sleep apnea should confirm they are legally permitted to do so under the dental laws and standards of care in their jurisdiction. That is, orthodontists must not perform out of their scope of practice or involve themselves in any treatment that would be non-compliant with applicable laws or outside the standards of care.

An orthodontist who provides prescribed treatment of obstructive sleep apnea needs to have the appropriate training, qualifications, and must operate within the laws and standards of care. Failure to do so may subject that orthodontist to civil and criminal penalties. In situations in which a qualified and appropriately-trained orthodontist has confirmed his or her ability to treat obstructive sleep apnea, the orthodontist also should consult with his or her insurance carrier to guarantee insurance coverage in this domain.

XIV. Executive Summary

Obstructive sleep apnea is a medical disorder that can have many serious consequences if left untreated. OSA can affect adults and children and can present at any point in the lifespan. All orthodontists should consider incorporating OSA screening into their history-taking and examination of patients. When an orthodontist has a clinical suspicion that a patient may have OSA, it is strongly recommended that referral to a physician be made; a sleep medicine physician is preferred. The definitive diagnosis of OSA should be made by a physician. Individual orthodontists may elect to participate in the treatment and monitoring of OSA patients as appropriate and permissible under applicable laws, standards of care, and insurance coverages.

1. It is strongly recommended that orthodontists be familiar with the signs and symptoms of OSA.
2. It is strongly recommended that orthodontists screen patients with regard to the signs and symptoms of OSA. A thorough history and clinical examination are critically important in that they establish the presence of pre-existing conditions, a basis for diagnosis, the need for referral, and a baseline for evaluating the effects of treatment.
3. It is strongly recommended that the orthodontist refer patients with risk factors for OSA to a physician for further evaluation and a definitive diagnosis. A sleep medicine physician is preferred.
4. It is recommended that the orthodontist refer pediatric patients with nasal obstruction or adenotonsillar hypertrophy to an otolaryngologist.
5. It is recommended that the orthodontist refer adult patients to an otolaryngologist when nasal obstruction or adenotonsillar hypertrophy is present.
6. The decision for an orthodontist to participate in the treatment of OSA is a choice that should be made based on interest as well as training, skills, experience, laws, standards of care, and insurance coverages applicable to the orthodontist.
7. If involved in the treatment of OSA, an orthodontist should monitor oral appliance treatment efficacy.
8. An orthodontist may elect to manage adverse side effects of oral appliance therapy.
9. No orthodontic treatments have been shown to cause or increase the likelihood of OSA. Rather, some forms of orthodontic treatment have been shown to be important in the treatment of OSA.
10. Interdisciplinary treatment of OSA helps to serve the best interests of patients with OSA.

XV. Action Plan

Future Research

Meaningful research concerning OSA can be enhanced dramatically by using the sleep study polysomnogram (PSG), which objectively assesses airway function, to measure outcomes of the long list of treatment possibilities, especially in growing children. There is a substantial leap of faith when researchers make the jump from “enlarged airway” to “OSA cure” or even “OSA improvement.”

Areas of study worthwhile of future research include:

- Which craniofacial variables contribute to the pathogenesis of OSA?
- How is airway function affected by various orthodontic treatments?
- At what age can OSA be detected?
- Does OSA progress from childhood into adulthood?
- Does OSA treatment in childhood prevent OSA in adulthood?
- What are the endpoints expected for OSA therapy?

Education

At this time, the subject of obstructive sleep apnea (OSA) in pediatric and adult populations is not included in the curricula of most dental school predoctoral and postdoctoral programs. Prior to the introduction of OSA as a curriculum subject, it is paramount for the American Dental Education Association (along with the American Dental Association (ADA) and Commission on Dental Accreditation (CODA)) to adopt educational standards for this subject, so OSA subject matter is taught with the proper endorsements and qualifications. A standardized curriculum should be developed and incorporated into all predoctoral and postdoctoral programs.

Additional Recommendations

It is recommended that the AAO consider developing a health history form for OSA for children and adults or include OSA questions in current health history forms. When screening for possible OSA in their patients, practitioners should consider recording their patient's height, weight, and neck size. They should also consider calculating the patient's body mass index (BMI) (Appendix 10). An Informed consent document for OSA might also be useful. The use of validated tools for risk-assessment of OSA is recommended to develop more efficient and standardized screening methods. The AAO might also consider whether the definition of orthodontics needs modification relative to obstructive sleep apnea.

XVI. Literature Resource for AAO Members

A Literature Resource for Orthodontics and OSA is being developed by Jackie Hittner, AAO Librarian. It will be available via the AAO Library webpage.

The Resource now contains over 4,000 article citations. It is estimated that this Resource eventually will contain approximately 5,000 article citations. If AAO members want to access the collection, they may access the searchable file and select articles. Initially, they will only see the abstract. If they want to view the entire article, they may then request the article from the AAO Library using the [journal request form](#). It is intended that this resource will be updated periodically.

XVII. Appendices

Appendix material will be available on the AAO Library Website. The appendices include the following:

Appendix 1	Examples of apnea and hypopnea
Appendix 2	STOP-Bang Questionnaire
Appendix 3	Modified Mallampati Score
Appendix 4	Epworth Sleepiness Scale
Appendix 5	Friedman Tongue Position
Appendix 6	Pediatric Sleep Questionnaire
Appendix 7	Epworth Sleepiness Scale for Children and Adolescents
Appendix 8	Brodsky Tonsil Grades
Appendix 9	Friedman Tonsil Grading System.
Appendix 10	BMI Tables 1 and 2

XVIII. Abbreviations Used in this Document

Apnea-Hypopnea Index (AHI)
Auto-titrating Positive Airway Pressure (APAP)
Bi-level Positive Airway Pressure (BPAP)
Body Mass Index (BMI)
Cone Beam Computed Tomographic (CBCT)
Continuous Positive Airway Pressure (CPAP)
Electroencephalography (EEG)
Headgear (HG)
Home Sleep Apnea Testing (HSAT)
Mandibular Advancing Appliances (OAm)
Maxillomandibular Advancement (MMA)
Modified Mallampati (MM)
Obstructive Sleep Apnea (OSA)
Oral Appliances (OA)
Otolaryngologist (ENT)
Out-of-Center Sleep Testing (OCST)
Pediatric Sleep Questionnaire (PSQ)
Pharyngeal Critical Closing Pressure (Pcrit)
Polysomnography (PSG)
Positive Airway Pressure (PAP)
Rapid Maxillary Expansion (RME)
Rapid Palatal Expansion (RPE)
Respiratory Disturbance Index (RDI)
Respiratory Effort Related Arousals [RERAs)
Respiratory Event Index (REI)
Sleep Related Breathing Disorders (SRDB)
Surgically Assisted Rapid Maxillary Expansion (SARME)
Temporomandibular Disorders (TMD)

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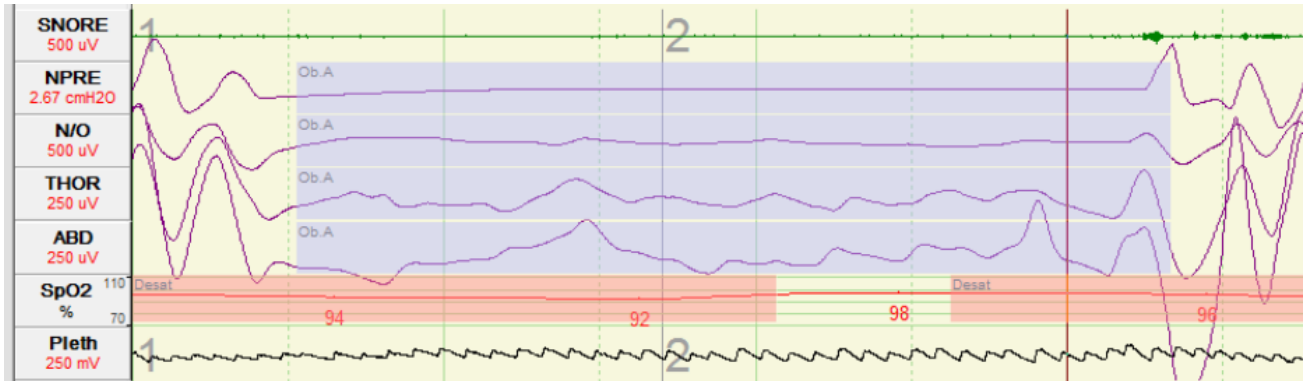
Footnote: A White Paper is an authoritative report or guide that informs readers concisely about a complex issue, presents the issuing body's philosophy, and offers proposals on the matter.

Appendices

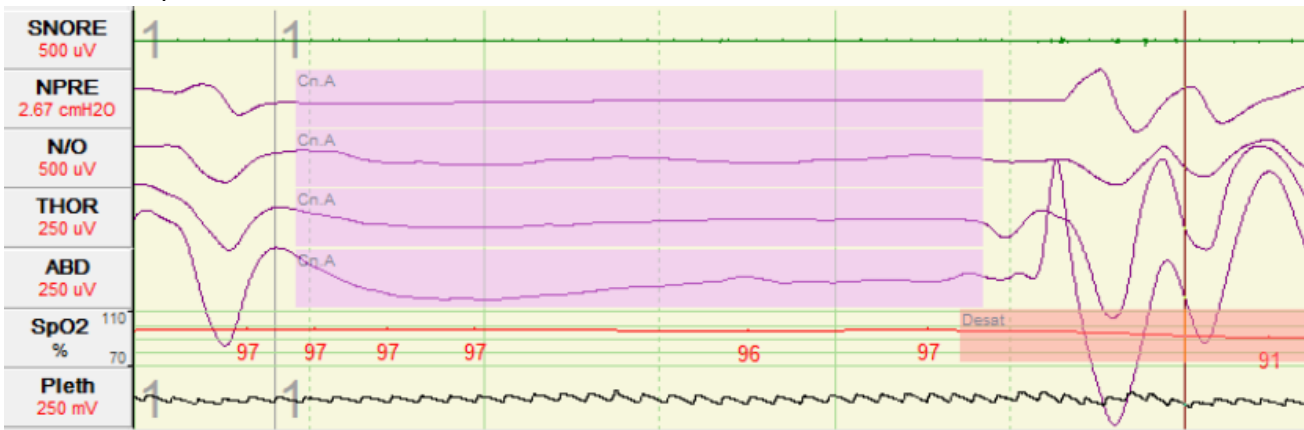
(Permission to use these illustrations must be obtained, or new illustrations prepared, if the illustrations are to be disseminated)

Appendix 1

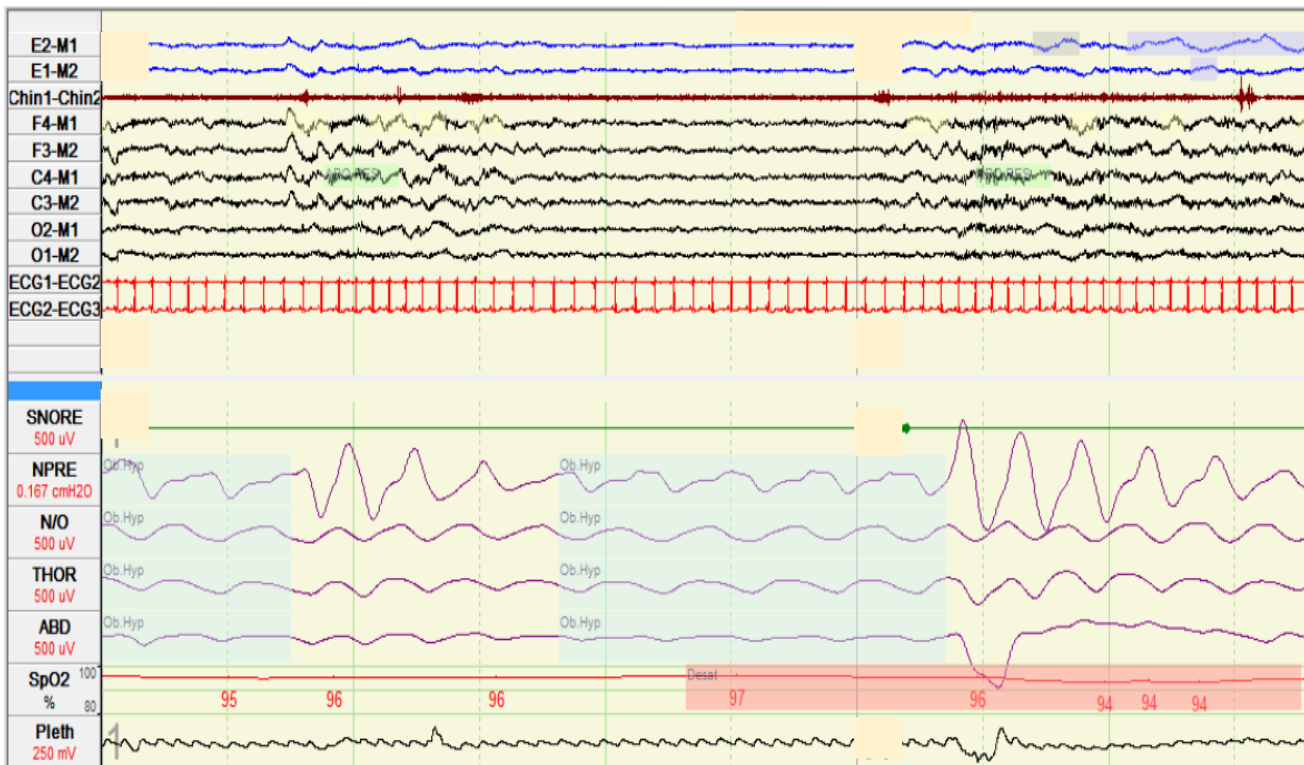
A. Obstructive apnea.



B. Central apnea.



C. Obstructive hypopnea.



Appendix 1

Examples of Apnea and Hypopnea

Panel A shows an obstructive apnea (at least 90% decrement in the N/O flow signal along with persistent thoracic and abdominal respiratory effort). Panel B shows a central apnea (at least 90% decrement in the N/O flow signal along with absent thoracic and abdominal respiratory effort). Panel C shows two obstructive hypopneas (at least 30% decrement in the NPRES flow signal followed by either a cortical arousal or a 3% oxygen desaturation). SNORE = snore channel; NPRES = nasal pressure; N/O = nasal-oral; THOR = thoracic; ABD = Abdomen; SpO2 = blood oxygen saturation; Pleth = plethysmography; E = eye; M =mastoid; F = frontal; C = central; O = occipital; ECG = electrocardiogram.

Appendix 2

Name _____
 Height _____ Weight _____
 Age _____ Male / Female _____

STOP-BANG Sleep Apnea Questionnaire

Chung F et al Anesthesiology 2008 and BJA 2012

STOP		
Do you SNORE loudly (louder than talking or loud enough to be heard through closed doors)?	Yes	No
Do you often feel TIRED , fatigued, or sleepy during daytime?	Yes	No
Has anyone OBSERVED you stop breathing during your sleep?	Yes	No
Do you have or are you being treated for high blood PRESSURE ?	Yes	No

BANG		
BMI more than 35kg/m ² ?	Yes	No
AGE over 50 years old?	Yes	No
NECK circumference > 16 inches (40cm)?	Yes	No
GENDER : Male?	Yes	No

TOTAL SCORE		
--------------------	--	--

High risk of OSA: Yes 5 - 8

Intermediate risk of OSA: Yes 3 - 4

Low risk of OSA: Yes 0 - 2

Appendix 2

STOP-Bang Questionnaire

In adults, a validated tool for OSA risk assessment is the STOP-Bang questionnaire which asks yes or no questions based on its acronym: snoring (S), tiredness (T), observed pauses in breathing (O), high blood pressure (P), BMI higher than 35 kg/m² (B), age older than 50 years (A), neck circumference of 17 inches or larger in males, or 16 inches or larger in females (N), and if patient's gender is male (G).

See Appendix 10 for BMI calculations involving inches and pounds.

A patient is considered to be at low risk for OSA if the questionnaire has 2 or less "yes" answers, at intermediate risk if there are 3 to 4 "yes" answers, and at high risk if there are more than 5 "yes" answers.

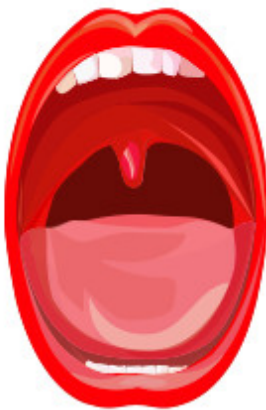
The patient also is considered at high risk if there are 2 "yes" answers from the STOP section, combined with either male gender, high BMI, or large neck size.

Adapted from:

Chung F, Yegneswaran B, Liao P, Chung SA, Vairavanathan S, Islam S, Khajehdehi A, Shapiro CM. STOP Questionnaire: A Tool to Screen Patients for Obstructive Sleep Apnea Anesthesiology 2008; 108:812–21. Copyright © 2008, the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Appendix 3

The Modified Mallampati Score



CLASS I
Complete
visualization of
the soft palate



CLASS II
Complete
visualization
of the uvula



CLASS III
Visualization
of only the
base of the uvula



CLASS IV
Soft palate
is not
visible at all

Appendix 3

Modified Mallampati Score

Three steps are followed to determine the MM Class: Step 1. Patients are asked to take a seated or supine position. Step 2. Patients are asked to protrude their tongue as far forward as they can without emitting a sound. Step 3. The examiner observes the relationship between the palate, tongue base and other soft tissue structures to determine the MM Classification.

The Modified Mallampati Scores are defined as Class I: Soft palate, fauces (the arched opening at the back of the mouth leading to the pharynx), uvula, and tonsillar pillars are visible; Class II: Soft palate, fauces, and uvula are visible; Class III: Soft palate and base of uvula are visible; Class IV: Soft palate is not visible.

A high Modified Mallampati score (Class 3 or 4) is associated with more difficult intubation as well as a higher incidence of [sleep apnea](#).^[2]

Adapted from:

Mallampati, SR; Gatt, SP; Gugino, LD; Desai, SP; Waraksa, B; Freiburger, D; Liu, PL (Jul 1985). A clinical sign to predict difficult tracheal intubation: a prospective study. *Canadian Anaesthetists' Society Journal*. 1995;**32** (4): 429–34. [doi:10.1007/BF03011357](#). [PMID 4027773](#).

Nuckton TJ, Glidden DV, Browner WS, Claman DM. Physical examination: Mallampati score as an independent predictor of obstructive sleep apnea. *Sleep*. 2006;29(7):903-8.

Samsoon, GL; Young, JR (May 1987). Difficult tracheal intubation: a retrospective study. *Anaesthesia*. 1987;**42** (5): 487–90. [doi:10.1111/j.1365-2044.1987.tb04039.x](#). [PMID 3592174](#).

Lee, A; Fan, LT; Gin, T; Karmakar, MK; Ngan Kee, WD (Jun 2006). A systematic review (meta-analysis) of the accuracy of the Mallampati tests to predict the difficult airway. *Anesthesia and Analgesia*. 2006;**102** (6): 1867–78. [doi:10.1213/01.ane.0000217211.12232.55](#). [PMID 16717341](#).

Appendix 4

Epworth Sleepiness Scale

Name: _____ Today's date: _____

Your age (Yrs): _____ Your sex (Male = M, Female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the **most appropriate number** for each situation:

- 0 = would never doze
- 1 = slight chance of dozing
- 2 = moderate chance of dozing
- 3 = high chance of dozing

It is important that you answer each question as best you can.

Situation	Chance of Dozing (0-3)
Sitting and reading _____	_____
Watching TV _____	_____
Sitting, inactive in a public place (e.g. a theatre or a meeting) _____	_____
As a passenger in a car for an hour without a break _____	_____
Lying down to rest in the afternoon when circumstances permit _____	_____
Sitting and talking to someone _____	_____
Sitting quietly after a lunch without alcohol _____	_____
In a car, while stopped for a few minutes in the traffic _____	_____

THANK YOU FOR YOUR COOPERATION

Appendix 4

Epworth Sleepiness Scale

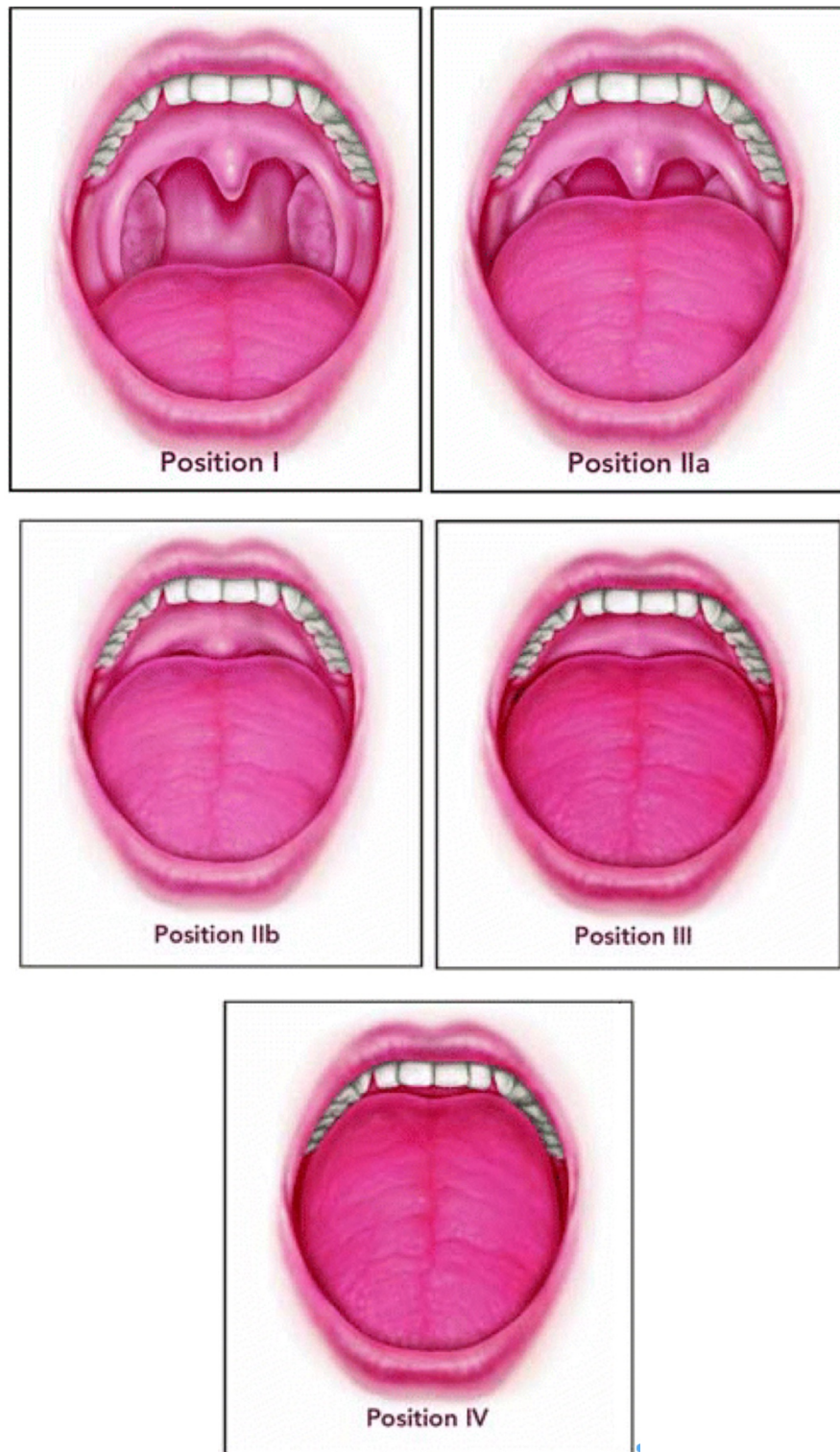
The EES is a subjective measure of a patient's sleepiness. The test is a list of eight situations where you rate your tendency to become sleepy on a scale of 0, no chance of dozing, to 3, high chance of dozing.

Interpretation:

- 0-7 It is unlikely that the patient is abnormally sleepy.
- 8-9 Average amount of daytime sleepiness.
- 10-15 The patient may be excessively sleepy depending on the situation. The patient may want to consider seeking medical attention.
- 16-24 The patient is excessively sleepy and should consider seeking medical attention.

Adapted from:

Reference: Johns MW. A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep* 1991; 14(6):540-5.

Appendix 5

Appendix 5

Friedman Tongue Position (FTP).

The Friedman Tongue Position (FTP) is a grading system used to assess the relationship of the palate to the tongue and is frequently utilized in the preoperative evaluation of patients with OSA. The tongue is evaluated in a neutral position within the oral cavity. The procedure for assigning an FTP grade involves having a patient open their mouth widely without tongue protrusion or phonation. The tongue position is then graded; Friedman suggests repeating the process five times and the most consistent grade be assigned as the FTP.

- FTP I visualizes the uvula and tonsils/pillar.
- FTP IIa visualizes most of the uvula but not the tonsils/pillar.
- FTP IIb visualizes the entire soft palate to the uvular base.
- FTP III shows some of the soft palate with the distal end absent.
- FTP IV visualizes only the hard palate.

FTP has also been found to correlate strongly with OSA severity.

Adapted from:

Friedman M: Friedman tongue position and the staging of obstructive sleep apnea/hypopnea syndrome; in: Scheidt S, Clansey N, eds. *Sleep Apnea and Snoring: Surgical and Non-Surgical Therapy*. Philadelphia, W.B.Saunders; 2009: 204-110.

Friedman M, Ibrahim H, Joseph NJ. Staging of obstructive sleep apnea/hypopnea syndrome: a guide to appropriate treatment. *Laryngoscope* 2004;114:454–459.

Friedman M, Salapatras AM, Sonzelaar LB. Updated Friedman staging system for obstructive sleep Apnea. Lin H-C (ed): *Sleep-Related Breathing Disorders*. Adv Otorhinolaryngol. Basel, Karger, 2017, vol 80, pp 41-48.

Appendix 6

PEDIATRIC SLEEP QUESTIONNAIRE PATIENTS UNDER 18 YEARS OF AGE

Last Name First Name Age Date

Please answer on behalf of your child for the past month.

If you don't know, circle "?"

While sleeping, does your child . . .

1. snore more than half the time? Yes / No / ?
2. always snore? Yes / No / ?
3. snore loudly? Yes / No / ?
4. have trouble breathing, or struggle to breathe? Yes / No / ?
5. have "heavy" or loud breathing? Yes / No / ?
6. have you ever seen your child stop breathing during the night? Yes / No / ?

Does your child . . .

7. tend to breathe through the mouth during the day? Yes / No / ?
8. have a dry mouth on waking up in the morning? Yes / No / ?
9. occasionally wet the bed? Yes / No / ?
10. wake up feeling unrefreshed in the morning? Yes / No / ?
11. have a problem with sleepiness during the day? Yes / No / ?
12. has a teacher commented that your child appears sleepy during the day? Yes / No / ?
13. is it hard to wake your child up in the morning? Yes / No / ?
14. does your child wake up with headaches in the morning? Yes / No / ?
15. did your child stop growing at a normal rate at any time since birth? Yes / No / ?
16. is your child overweight? Yes / No / ?

My child often . . .

17. does not seem to listen when spoken to directly. Yes / No / ?
18. has difficulty organizing task and activities. Yes / No / ?
19. is easily distracted by extraneous stimuli. Yes / No / ?
20. fidgets with hands or feet or squirms in seat. Yes / No / ?
21. is 'on the go' or often acts as if 'driven by a motor'. Yes / No / ?
22. interrupts or intrudes on others (e.g. butts into conversations or games) Yes / No / ?

Appendix 6

Pediatric Sleep Questionnaire

On the PSQ, the response categories for all questions are “yes” or “no” or “don't know,” indicated by “?”. The cutoff value to identify patients at high risk for SDB is 0.33, meaning that 33% of the responses were “yes.” A proportion of “yes” responses greater than or equal to 0.33 indicated a high risk of SDB; a lower percentage indicated a low risk. Missing and “don't know” responses were excluded from the denominator when estimating the percentage for risk status. When compared with polysomnographic data, the PSQ has previously demonstrated sensitivity of 0.85 and specificity of 0.87.²

Adapted from:

Rohra AK, Demko CA, Hans MG, Rosen C, Palomo JM. Sleep disordered breathing in children seeking orthodontic care. *Am J Orthod Dentofacial Orthop*. 2018;154:65-71.

Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. *Sleep Med*. 2000 Feb 1;1(1):21-32.

Appendix 7

Epworth Sleepiness Scale for Children and Adolescents

ESS – CHAD

(parent/carer to answer)

Your child's name: _____ Today's date: _____

How old is your child? _____ (years) Boy? (____) or Girl? (____) check one space

Over the past month, how likely has your child been to fall asleep while doing the things that are described below (activities)?

Even if your child hasn't done some of these things in the past month, try to imagine how they would have affected him/her.

Use the following scale to choose one number that best describes what has been happening to your child during each activity over the past month. Write that number in the box below.

0 = would never fall asleep

1 = slight chance of falling asleep

2 = moderate chance of falling asleep

3 = high chance of falling asleep

It is important that you answer each question as best you can

Activities	Chance of falling asleep (0 – 3)
Sitting and reading _____	
Sitting and watching TV or a video _____	
Sitting in a classroom at school during the morning _____	
Sitting and riding in a car or bus for about half an hour _____	
Lying down to rest or nap in the afternoon _____	
Sitting and talking to someone _____	
Sitting quietly by himself/herself after lunch _____	
Sitting and eating a meal _____	

Thank you

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Appendix 7

Epworth Sleepiness Scale for Children and Adolescents

The Epworth Sleepiness Scale for Children and Adolescents asks parents/patients to rate their level of sleepiness in several different sedentary situations. The ESS-CHAD may be helpful to assess for problematic sleepiness, but this tool cannot identify a specific cause of daytime sleepiness. The Epworth scale has been validated only for children ages 12-18 years.¹¹

Scoring is based on the following:

0-10 Normal levels of sleepiness

10-16 Excessive daytime sleepiness

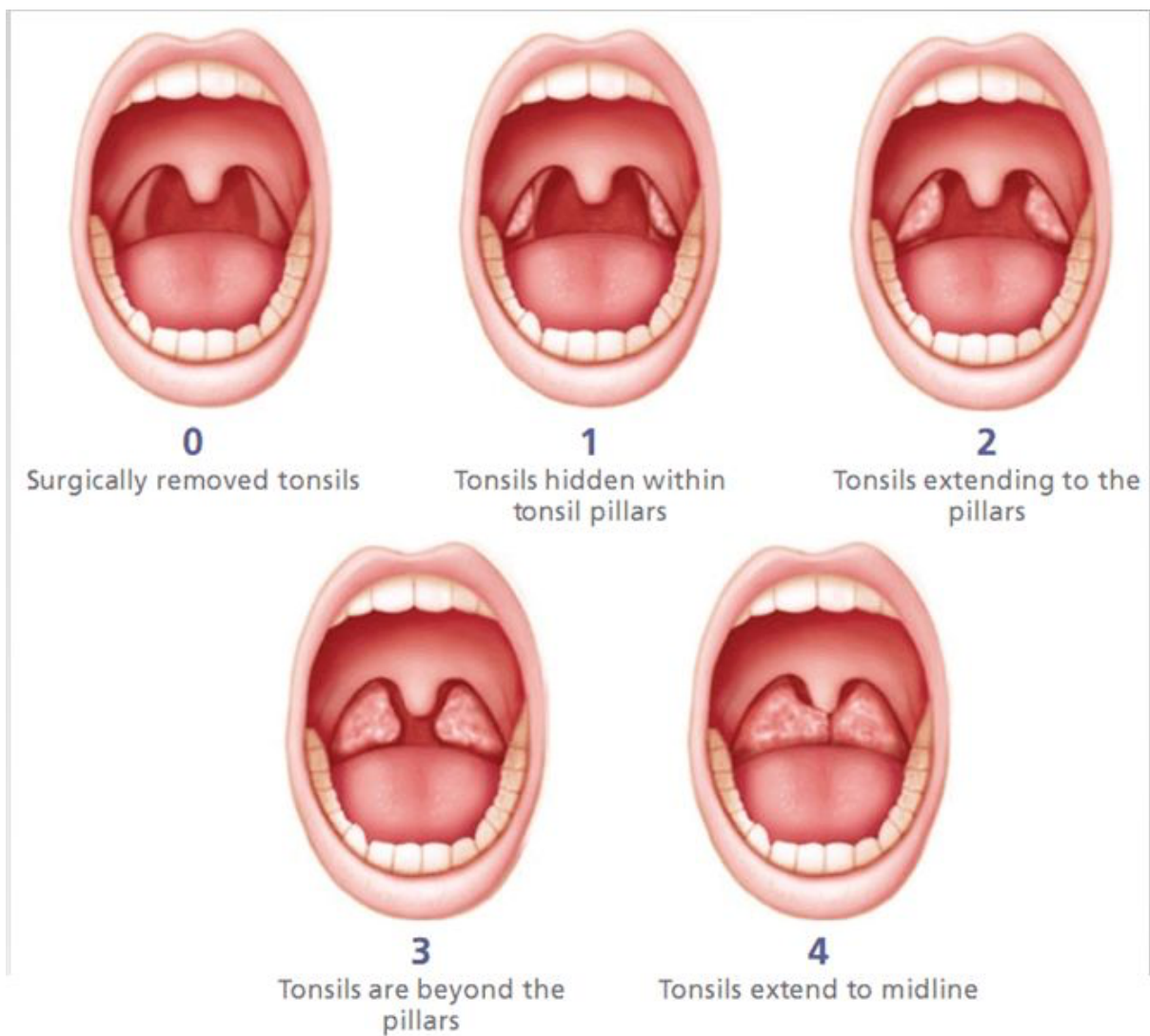
16-24..High level of sleepiness suggestive of significance sleep disorder

A score above 10 suggests excessive daytime sleepiness and should be discussed with a sleep specialist.

Adapted from:

Johns MW. The assessment of sleepiness in children and adolescents. Sleep Biol Rhythm 2015;13(Suppl 1):97

Appendix 8



Appendix 8

Brodsky Tonsil Grades

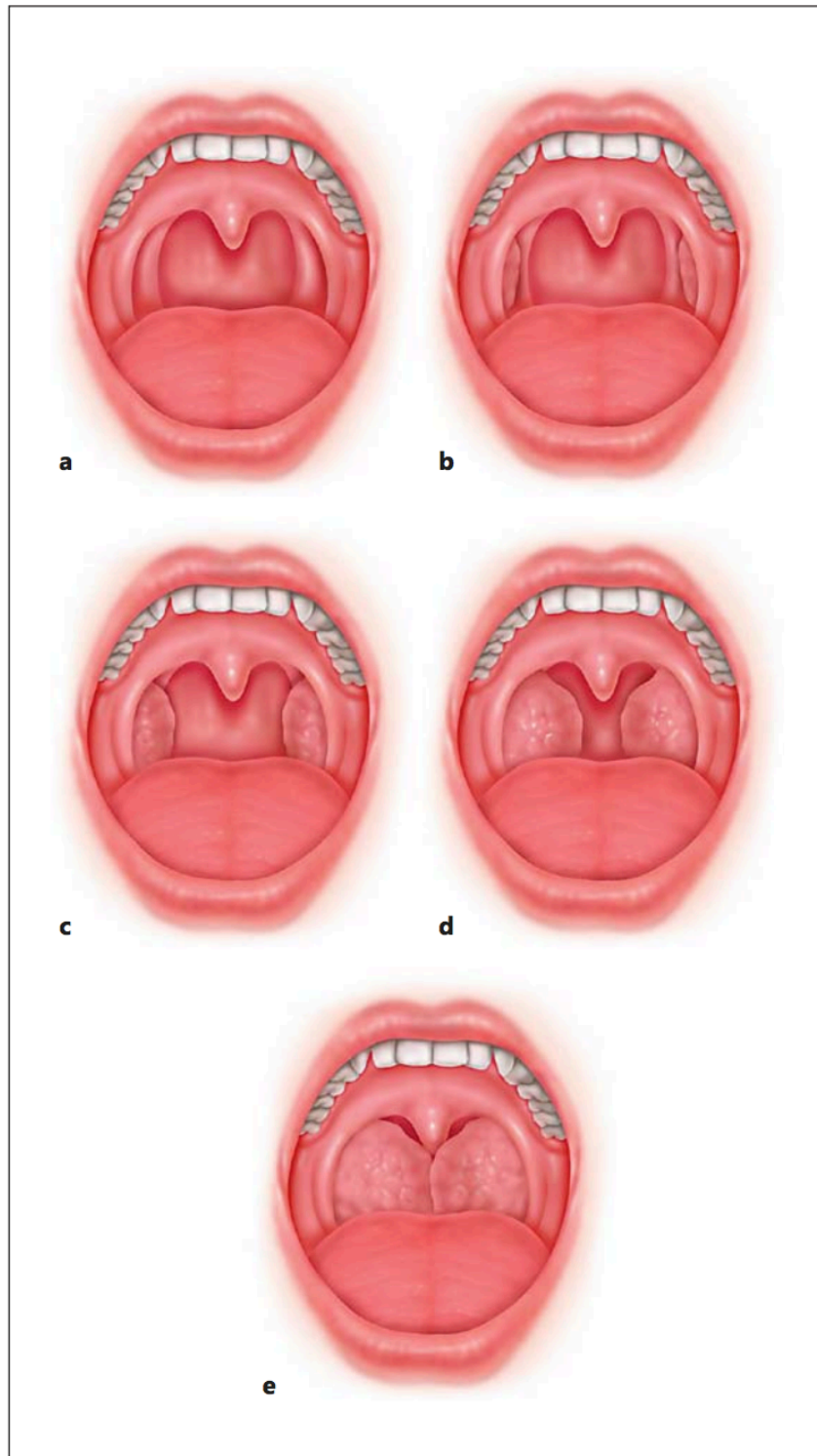
The tonsils are classified into five grades as follows:

- grade 0 indicated the previous tonsillectomy;
- grade 1 indicated that the tonsils were hidden in the pillars;
- grade 2 indicated that the tonsils were beyond the anterior pillar and between 25 and 50% of the pharyngeal space;
- grade 3 indicated that the tonsils were beyond the pillars but not to the middle and occupied >50% and up to 75% of the pharyngeal space;
- grade 4 indicated that the tonsils occupied >75% of the pharyngeal space.

Tonsil grades 1 and 2 comprise the small tonsil group, while grades 3 and 4 comprised the big tonsil group.

Adapted from:

Brodsky L. Modern assessment of tonsils and adenoids. *Pediatr Clin North Am* 1989;36(6):1551-69

Appendix 9

Appendix 9

Friedman Tonsil Grading System.

In the Friedman grading scale, the tonsil size is classified on the basis of the location of the tonsil in relation to its surrounding structures in the oral cavity.

- **a** Size 0, absence of tonsillar tissue.
- **b** Size 1, within the pillars.
- **c** Size 2, extended to the pillars.
- **d** Size 3, extended past the pillars.
- **e** Size 4, extended to the midline.

Adapted from:

Friedman M: Friedman tongue position and the staging of obstructive sleep apnea/hypopnea syndrome; in Friedman M (Ed.): Sleep Apnea and Snoring: Surgical and Non-Surgical Therapy. Philadelphia, Saunders Elsevier, 2009, Chapter 16.

Appendix 10

Body Mass Index Table 1

for BMI greater than 35, go to Table 2

To use the table, find the appropriate height in the left-hand column labeled Height. Move across to a given weight (in pounds). The number at the top of the column is the BMI at that height and weight.
Pounds have been rounded off.

[Select the PDF version for better printing](#)

BMI	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
Height (inches)	Body Weight (pounds)																
58	91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	163	167
59	94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168	173
60	97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174	179
61	100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180	185
62	104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186	191
63	107	113	118	124	130	135	141	146	152	158	163	169	175	180	186	191	197
64	110	116	122	128	134	140	145	151	157	163	169	174	180	186	192	197	204
65	114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204	210
66	118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210	216
67	121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217	223
68	125	131	138	144	151	158	164	171	177	184	190	197	203	210	216	223	230
69	128	135	142	149	155	162	169	176	182	189	196	203	209	216	223	230	236
70	132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236	243
71	136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243	250
72	140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250	258
73	144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257	265
74	148	155	163	171	179	186	194	202	210	218	225	233	241	249	256	264	272
75	152	160	168	176	184	192	200	208	216	224	232	240	248	256	264	272	279
76	156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279	287

Body Mass Index Table 2

To use the table, find the appropriate height in the left-hand column labeled Height. Move across to a given weight. The number at the top of the column is the BMI at that height and weight. Pounds have been rounded off.

[Select the PDF version for better printing](#)

BMI	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54
Height (inches)	Body Weight (pounds)																		
58	172	177	181	186	191	196	201	205	210	215	220	224	229	234	239	244	248	253	258
59	178	183	188	193	198	203	208	212	217	222	227	232	237	242	247	252	257	262	267
60	184	189	194	199	204	209	215	220	225	230	235	240	245	250	255	261	266	271	276
61	190	195	201	206	211	217	222	227	232	238	243	248	254	259	264	269	275	280	285
62	196	202	207	213	218	224	229	235	240	246	251	256	262	267	273	278	284	289	295
63	203	208	214	220	225	231	237	242	248	254	259	265	270	278	282	287	293	299	304
64	209	215	221	227	232	238	244	250	256	262	267	273	279	285	291	296	302	308	314
65	216	222	228	234	240	246	252	258	264	270	276	282	288	294	300	306	312	318	324
66	223	229	235	241	247	253	260	266	272	278	284	291	297	303	309	315	322	328	334
67	230	236	242	249	255	261	268	274	280	287	293	299	306	312	319	325	331	338	344
68	236	243	249	256	262	269	276	282	289	295	302	308	315	322	328	335	341	348	354
69	243	250	257	263	270	277	284	291	297	304	311	318	324	331	338	345	351	358	365
70	250	257	264	271	278	285	292	299	306	313	320	327	334	341	348	355	362	369	376
71	257	265	272	279	286	293	301	308	315	322	329	338	343	351	358	365	372	379	386
72	265	272	279	287	294	302	309	316	324	331	338	346	353	361	368	375	383	390	397
73	272	280	288	295	302	310	318	325	333	340	348	355	363	371	378	386	393	401	408
74	280	287	295	303	311	319	326	334	342	350	358	365	373	381	389	396	404	412	420
75	287	295	303	311	319	327	335	343	351	359	367	375	383	391	399	407	415	423	431
76	295	304	312	320	328	336	344	353	361	369	377	385	394	402	410	418	426	435	443

Appendix 10

BMI Tables

The Body Mass Index in inches and pounds.

Adapted from:

U.S. Department of Health and Human Services