Obstructive sleep apnea and orthodontics: An American Association of Orthodontists White Paper

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The Board of Trustees of the American Association of Orthodontists asked 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 obstructive sleep apnea. This White Paper presents a summary of the Task Force's findings and recommendations. (Am J Orthod Dentofacial Orthop 2019;156:13-28)

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.

Although OSA can be definitively diagnosed only 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.

Patients with suspected OSA may come to the orthodontist in 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 or suggests orthodontic or orthopedic therapy to assist in the management of the OSA. Other patients or caregivers may present to the orthodontist with concerns about breathing during sleep. In addition, patients may present to the orthodontist unaware of their OSA, and orthodontic screening may reveal the need for further evaluation by a physician.

In November 2017, the Board of Trustees of the American Association of Orthodontists (AAO) tasked a panel of medical and dental experts in sleep medicine and dental sleep medicine to create a document

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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.

This document was subject to editorial changes prior to publication.

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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 around the world and involving 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 U.S. Preventative Respiratory Society, among others.

However, the Task Force could not identify any formal OSA guidance for orthodontists. This was surprising because 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 (eg, cleft lip and palate, craniofacial syndromes, complex restorative cases, orthognathic surgery).

Given that OSA can be a serious, even life-threatening, disorder and 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.

ADULT OSA

Sleep-related breathing disorders (SRBDs) constitute a diagnostic category of disease that encompasses obstructive phenomena, including primary snoring, upper airway resistance syndrome, and 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 (ie, 18 years of age or older). Clinical concerns for other forms of SRBD and additional types of sleep disorders (eg, 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 (Pcrit) 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 OSA 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 (eg, 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 OSA in adults vary in the literature; OSA is commonly 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. Underrecognition of OSA likely leads to underdiagnosis 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 (body mass index [BMI] $\geq$30 kg/m^2), menopause, male

sex, and increasing age. 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 OSA often have a history of snoring, gasping respiration or choking, and witnessed pauses in breathing (apneas) during sleep. Common clinical symptoms of untreated OSA include frequent nocturnal awakenings, nonrestorative 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 OSA is performed by a sleep medicine specialist with the use of the gold standard of an in-center overnight sleep study (polysomnography [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), electrocardiography, and monitoring of sleep, airflow through the nose and mouth, pulse oximetry, respiratory effort, 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 Disorders,[1] OSA can be diagnosed by either of 2 sets of criteria. The first set of diagnostic criteria for OSA includes the presence of at least 1 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, and (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 at least 5 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.

In the second criteria, OSA can be diagnosed if PSG or OCST shows 15 or more predominantly obstructive events (obstructive or mixed apneas, hypopneas, or RERAs per hour of sleep during a PSG or per hour of monitoring on OCST). Examples of apnea and hypopnea are presented in Appendix 1.

A few different terms are used in the classification of OSA. 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 refer to AHI and others RDI, so it is important for clinicians and researchers to understand the difference between these 2 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 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 AHI or RDI per hour; categories are mild (AHI or RDI $\geq$5 and $<$15), moderate (AHI or RDI $\geq$15 and $<$30), and severe (AHI or RDI $\geq$30). The minimum oxygen saturation also should be considered when making clinical assessment of the magnitude of OSA, 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 OSA increases risk of insulin resistance, coronary artery disease, congestive heart failure, myocardial infarction, hypertension, stroke, cardiac arrhythmia, and sudden cardiac death.

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 to establish the presence of preexisting 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 previous diagnosis of OSA, excessive daytime sleepiness,* a previous diagnosis of other forms of SRBDs, fatigue during the day, height,* choking or gasping respirations during sleep, weight,* habitual or loud snoring,* sex,* 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, and neck circumference (*component of the STOP-Bang questionnaire; see next section).

Screening tools

In adults, a validated tool for OSA risk assessment is the STOP-Bang questionnaire (Appendix II),[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 >35 kg/m^2 (B), age >50 years (A), neck circumference of ≥ 17 inches in men, or ≥ 16 inches in women (N), and male gender (G). A patient is considered to be at low risk for OSA if the questionnaire has no more than 2 "yes" answers, at intermediate risk if here are 3 or 4 "yes" answers, and at high risk if there are 5 or more "yes" answers.

The patient is considered at high risk also if there are 2 "yes" answers from the STOP section, combined with either male gender, high BMI, or large neck size. Using a cutoff score of ≥ 3 to detect any OSA (AHI >5), moderate to severe OSA (AHI >15), and severe OSA (AHI >30), the sensitivities were 84%, 93%, and 100% and specificities 56%, 43%, and 37%, respectively.[4] 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 as part of 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 (MM) classification to describe the patency of the oral airway (Appendix III).[5–11] 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; and 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; and 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 MM 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 IV)[12] asks patients to self-rate their level of sleepiness in 8 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, because it detects abnormalities in level of daytime sleepiness regardless of the cause of sleepiness.

Practitioners also may find the Friedman tongue classification system (Appendix V),[13] the Kushida index,[14] and the Berlin Questionnaire for Sleep Apnea[15] 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. Therefore, airway imaging with the use of 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. In addition, there is currently 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 SRBDs, because such imaging currently does not represent a proper risk assessment technique or screening method. On the other hand, 3-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.

DIAGNOSIS AND TREATMENT PLANNING IN ADULT OSA

Obstructive sleep apnea and other SRBDs can be definitively diagnosed only by a physician. It is not in the scope of the orthodontist or any other dentist to definitively diagnose OSA or any other SRBD. 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 and when 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 management of OSA not take place without a referral from a physician (or provider supervised by a physician).

TREATMENT OF OSA IN ADULTS BY PHYSICIANS AND SURGEONS

Positive airway pressure (PAP) therapy is the gold standard treatment for OSA 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), bilevel positive airway pressure (BPAP), or autotitrating positive airway pressure (APAP). Of note, CPAP and BPAP devices are available in conventional and autotitrating 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/h).[16] BPAP may be used for patients with OSA who are intolerant of CPAP or those who have other forms of SRBDs (eg, sleep-related hypoventilation). APAP may be considered for patients with OSA who do not have contraindications to APAP use (eg, congestive heart failure, lung disease such as chronic obstructive pulmonary disease, obesity hypoventilation syndrome, or central sleep apnea).

Studies on PAP nonadherence report wide-ranging results. Although definitions of nonadherence vary across studies, a common definition of PAP nonadherence is mean use $\leq$4 hours per night. Estimates of PAP nonadherence range from 29% to 83%.[17,18] 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.[19] Factors that affect PAP adherence include OSA severity, ability to tolerate the prescribed pressure setting, mask fit, spousal support, and other psychologic and social influences.[17]

Other treatment options include positional therapy (avoidance of sleeping on back) and long-term weight reduction as indicated. Nasal congestion and allergic rhinitis may be managed with the use of 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 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, frena, and tongue. Hypoglossal nerve stimulation addresses the impaired neuromuscular tone in OSA and may be considered in certain patients with OSA.

ORTHODONTIC MANAGEMENT IN ADULT OSA

After 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

Before 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 (OA) 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, which include both mandibular advancing oral appliances (OAms) and tongue-retaining devices, are usually effective options for OSA management in appropriately selected patients. OAms are intended to hold the mandible 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 OAs 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 OAs fit into the OSA management paradigm.[20,21]

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

Many types of OAs 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 two-thirds 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 used 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 subtherapeutic, the physician and orthodontist should discuss the possibility of further titration or alternate 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 bed partner 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 sensor[22] 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, or changes to overall health, a consultation with the physician is strongly recommended.

Goals of treatment

The end points of treatment include reduced or eliminated snoring, resolution of the patient's initial symptoms of OSA, normalization of the AHI, and normalization of oxyhemoglobin saturation. No pretreatment risk factors have been consistently shown to predict success for OAs in reaching treatment goals.

Change in occlusion

Oral appliances used in sleep apnea treatment move teeth. In the field of dentistry, orthodontists are generally considered to be the experts in the management of malocclusion owing to their education and clinical experience. Improved awareness of both OSA and the effectiveness of OAs has resulted in increased numbers of OSA patients being treated with the use of OAs by nonorthodontists. Although successful OSA treatment may be evident over the short term in many of these patients, nonorthodontic providers may be unaware of the unwanted effects that 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 OA wear.

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

Orthodontists may be asked to assess and treat OA-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 the OA during treatment; therefore, the patient may need to use PAP therapy during the period of orthodontic care. Communication with the physician helps to ensure that the patient's OSA is still being managed appropriately.

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

Maxillomandibular advancement and surgically assisted rapid maxillary expansion

Patients who are unable to tolerate or adhere to PAP or OA therapy with an underlying sagittal skeletal discrepancy may be candidates for maxillomandibular advancement (MMA) or telegnathic (>10 mm) jaw advancement surgery. MMA is generally reserved for patients with severe OSA who are unable to tolerate PAP therapy and 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 presurgical preparation and that the surgery performed will not adversely affect facial esthetics. Orthodontic care is usually a beneficial adjunct for patients to facilitate obtaining optimal occlusion while simultaneously reducing the risk of postoperative malocclusion. Patients with ideal or minimal Class I malocclusion may not require extensive presurgical orthodontics in that the 2 jaws may have a similar interdigitation after 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 alternate 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 exist 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 the use of SARME and developing a functional and esthetic occlusion with comprehensive orthodontic treatment afterward has been suggested to improve PSG parameters.[23]

Possible treatments on the horizon

New treatment modalities, such as mini-implant (miniscrew or temporary anchorage device)–supported rapid maxillary expansion, are appearing as possible alternatives for SARME. 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.

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. Owing to the airflow restriction, there is a relative increase in serum carbon dioxide (CO_2; 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 to sleep, 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

Because the obesity epidemic also affects children, obesity is becoming a greater factor for childhood OSA. However, because untreated OSA may contribute to growth restriction, some children with OSA paradoxically may be underweight. Therefore, it is recommended that a clinical risk assessment for OSA be performed even in normal-weight and underweight children.

In addition, it is thought that certain craniofacial morphologies can increase a child's risk for having OSA. For example, 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 developing 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 sequence[24] and syndromic craniosynostosis[25] have a high prevalence of OSA. Children with Down syndrome[26] 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. Whereas 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 CO_2 monitoring. Measurement with either end-tidal CO_2 (the partial pressure of CO_2 present at the end of exhalation) or transcutaneous CO_2 monitoring is acceptable.

According to the International Classification of Sleep Disorders,[1] OSA can be diagnosed by either of 2 sets of diagnostic criteria. The first set of criteria for OSA includes the presence of at least 1 of the following: (1) snoring, (2) labored, paradoxic, 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, OSA 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 ($PaCO_2$ >50 mm Hg) associated with at least 1 of the following: (1) snoring, (2) flattening of the inspiratory nasal pressure waveform, or (3) paradoxic thoracoabdominal motion. These OSA diagnostic criteria are for children under the age of 18 years, although adult OSA diagnostic criteria may be used for children of ages 13-18 years, according to the American Academy of Sleep Medicine Manual for the Scoring of Sleep and

Associated Events.[27] HSAT is not indicated in patients under 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/h. Other studies define childhood OSA as mild (AHI or RDI ≥1 and <5/h), moderate (AHI ≥5 <10/h) and severe (AHI ≥10/h). Of note, scoring of obstructive apneas and hypopneas on PSG differs slightly for children than for adults. For adults event duration is defined as 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%-6%, prevalence of parent-reported apneic events during sleep to be 0.2%-4%, and OSA diagnosed by varying criteria to be 1%-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.

PEDIATRIC OSA: SKELETAL AND SOFT TISSUE GROWTH

Orthodontists are aware of the impact that 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, tongue, soft palate, turbinates, and 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 sphenoethmoidal synchondrosis up to the age of 7 years. Increases in posterior cranial base length are similarly related to growth at the sphenooccipital synchondrosis up to the age of 13 years. The anterior cranial base carries the nasomaxillary complex forward at the same time that 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 is displaced anteriorly and inferiorly. Thus, the normal facial growth process results in dramatic increases in all 3 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, and adolescence. These changes in airway due to growth far exceed any orthodontic or orthopedic effects on airway shape or size. Knowledge of these changes is important to understanding the dynamics of OSA in children.[31]

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 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]

Thorough history and examination are critically important in that they establish the presence of preexisting 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: previous diagnosis of OSA, loud snoring, previous diagnosis of other forms of SRBDs, 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, and attention deficit–hyperactivity disorder.

Screening tools

One potential screening tool that has been validated and used in orthodontic offices is the Pediatric Sleep Questionnaire (PSQ; Appendix VI).[33–35] This questionnaire has a positive predictive value of 0.4 (ie, 40% of patients with a positive PSQ score will be diagnosed with OSA) and a negative predictive value of 0.99, (ie, only 1% of patients with a negative PSQ score will be 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 VII)[36] may be helpful to assess for problematic sleepiness, but it cannot identify a specific cause of daytime sleepiness. The Epworth scale has been validated only for children 12-18 years of age.[12]

Clinical examination

In addition to the usual orthodontic clinical examination that evaluates dental occlusion, range of mandibular motion, soft tissue frenum attachments, gingival health, and temporomandibular disorder, the orthodontist should also note the degree to which the tonsils impinge on the pharyngeal airway. A commonly accepted tonsil classification system, 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 2 tonsils (Appendix VIII).[37] The Friedman tonsil grading system (Appendix IX)[38] may also be a useful tool to evaluate the size of the tonsils. Because tonsil size does not correlate with OSA severity, there is no set cutoff point for tonsillar hypertrophy necessitating a referral to an otolaryngologist for further evaluation[39]; therefore, this decision is best made in the patient-specific context of symptoms and physical examination 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 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 2-dimensional imaging for assessment of airway volume and area of maximum constriction. Airway imaging with the use of 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, although 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 2-dimensional and 3-dimensional imaging of the airway are helpful, they cannot be used to diagnose sleep apnea or any other SRBDs 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, may also 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.

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 OSA.

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.

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 adenoids are the most common risk factors for OSA in the pediatric population, with tonsillectomy and adenoidectomy typically considered as 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. PAP may be used 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 example, RME is a well known orthodontic treatment option for patients with a narrow maxilla. There is growing evidence, though low level, that in mixed-dentition patients who are properly diagnosed with OSA, RME can decrease AHI in the short and long terms.[40] Unfortunately, untreated control groups generally were not used in the studies considered. Regardless of the presence of OSA, it is recommended that 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 can produce a decrease in AHI. Long-term stability of these changes has not been studied; untreated control groups generally were not used in those studies as well. Regardless of the presence of OSA, it is recommended that the orthodontist use these devices only when there is an indication that a related retrognathic condition exists. As with RME, there is no clear indication in the literature, however, that prophylactic use of mandibular anterior repositioning appliances prevents later development of OSA.

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 the Angle classification of occlusion. In this case, treatment with the use of an oral device is directed primarily toward airway maintenance and less toward 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 protraction on AHI. Only an 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. Again, regardless of the presence of OSA, it is recommended that 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, whereas information on the treatment of OSA in pediatric patients is much more limited. Therefore, 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.

FALLACIES ABOUT ORTHODONTICS IN RELATION TO OSA

Conventional orthodontic treatment has never been proven to be an etiologic factor in the development of OSA. 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 the use of dental extractions or orthopedic headgear (HG).

The specific effects on the dental arches and the muscles and soft tissues of the oral cavity after 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 after 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 the use of 2-dimensional cephalography and more recently with 3-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 used.[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 and changes in the dimensions of the upper airway, it is helpful also 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 after orthodontic treatment, instead of focusing solely on quantifying airway dimensions. One such study assessed dental extractions as a cause of OSA later in life by means of a large retrospective examination of dental and medical records.[48] Researchers reviewed the health records of more than 2700 adults with 4 missing premolars and evaluated whether this group had a higher prevalence of OSA compared with an equal-size group of patients with no missing teeth who were matched for the most significant confounding variables of OSA in adults, namely age, BMI, and sex. 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 HG, which alters the direction of growth of the maxilla, has long been a staple of certain orthodontic treatments. Although dentoalveolar movement can be significant, the absolute skeletal change to the position of the maxilla elicited by HG 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 methodologic limitations have examined this relationship directly. The best evidence available at this time indicates that HG 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 HG 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 2 dimensions with the use of lateral cephalograms of HG patients. One study concluded that the absolute value of the airway dimension was smaller in HG patients than in activator patients, but the differences were both small and not statistically significant.[49] A longitudinal study examined patients over a 12-year period and reported that the radiographic dimension of the airway decreased during the treatment phase but increased to the level of control subjects during follow-up.[50] A prospective, randomized, blinded study demonstrated an increase in the airway during the 6-year study period.[51] 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 would contribute to a deviation of normal form or function in all but the most severe forms of ankyloglossia. More recently a 4-point severity scale of tongue mobility was reported, with the most severely restricted tongues graded as 4.[52] The investigators reported a reduced maxillary intercanine width and a longer soft palate in patients with more severe levels of tongue restriction compared with patients with no such restriction. However, the relationship between tongue mobility and function of the airway 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.

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 OSA.

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 definitive OSA diagnosis and treatment. Depending on the physician's diagnosis and plan for treatment, the orthodontist may be involved in the treatment after proper referral by the physician.

Any orthodontist involved in the treatment of adult or pediatric OSA should confirm that 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 noncompliant with applicable laws or outside the standards of care.

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

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 preexisting 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 coverage applicable to the orthodontist.
7. If involved in the treatment of OSA, an orthodontist should monitor OA treatment efficacy.
8. An orthodontist may elect to manage adverse side effects of OA 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.

ACTION PLAN

Future research

Meaningful research concerning OSA can be enhanced dramatically with the use of the 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 the following. 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 end points expected for OSA therapy?

Education

At this time, the subject of OSA in pediatric and adult populations is not included in the curricula of most dental school predoctoral and postdoctoral programs. Before the introduction of OSA as a curriculum subject, it is paramount for the American Dental Education Association (along with the American Dental Association and Commission on Dental Accreditation) to adopt educational standards for this subject, so that 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 BMI (Appendix X). 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 OSA.

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 Web page.

The Literature Resource now contains more than 4,000 article citations. It is estimated that eventually it will contain around 5,000 article citations. If AAO members want to access the collection, they may access the searchable file and select articles. Initially, they will see only the abstract. If they want to view the entire article, they may then request the article from the AAO Library by means of the journal request form. It is intended that this resource will be updated periodically.

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APPENDICES

Appendix material will be available on the AAO Library Web site. They include the following:
Appendix I: Examples of apnea and hypopnea
Appendix II: STOP-Bang questionnaire
Appendix III: Modified Mallampati score
Appendix IV: Epworth Sleepiness Scale
Appendix V: Friedman tongue position
Appendix VI: Pediatric Sleep Questionnaire
Appendix VII: Epworth Sleepiness Scale for Children and Adolescents
Appendix VIII: Brodsky tonsil grades
Appendix IX: Friedman tonsil grading system
Appendix X: Body mass index tables 1 and 2