Sleep-disordered breathing and orthodontics: An American Association of Orthodontists white paper update

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The board of trustees of the American Association of Orthodontists requested that a panel of orthodontic experts in dental sleep medicine update the 2019 American Association of Orthodontists white paper and create a document to guide practicing orthodontists on the role of orthodontics in managing obstructive sleep apnea. The present updated white paper summarizes the task force's findings and recommendations. (Am J Orthod Dentofacial Orthop 2026;■:■-■)

The 2019 American Association of Orthodontists (AAO) white paper on obstructive sleep apnea (OSA) and orthodontics provided guidance and clinical suggestions on best managing patients with OSA in an orthodontic environment.[1] The manuscript has become a reference on the subject; however, as indicated in the document, there is a need for periodic updates as new evidence arises. In 2024, the AAO board of trustees established a new task force comprising AAO members, academicians, and clinicians actively involved in dental sleep medicine to evaluate and update the content of the AAO 2019 white paper. Upon reviewing both previous and current literature, the task force found that although the information presented in the 2019 document remains relevant, certain areas would benefit from an update.

Although the AAO 2019 white paper addresses OSA in particular, a broader term—sleep-disordered breathing (SDB)—was used in this update. OSA is one of many SDBs, and the American Academy of Sleep Medicine identifies numerous other conditions that fall into this category, including a continuum ranging from habitual snoring to OSA.[2] Moving forward with the intent to be thorough, we will refer to the larger condition of SDB throughout this paper unless the available evidence is only sourced from OSA patients.

SDB may affect neurocognitive functions in children; however, its management and treatment approach are medical decisions that are only appropriate after a proper diagnosis by a physician.[3-7] Early SDB detection is beneficial—a role in which orthodontists should play a key part—for performing risk assessments and referring patients for proper diagnosis by physicians when the presence of SDB is suspected.[3-7] The AAO would like to remind orthodontists that a diagnosis is necessary before any SDB-related intervention is undertaken and that, at the time this document is published, only a physician can make an SDB diagnosis in the United States and Canada.[6,7] This requirement is based not on capabilities or training, but on state medical and dental practice acts. As SDBs are a medical condition, their diagnosis would fall outside an orthodontist's scope of practice.[6,7]

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Submitted and accepted, January 2026.
0889-5406

https://doi.org/10.1016/j.ajodo.2026.01.014

Upon reviewing the literature, there is still no evidence of any orthodontic intervention capable of preventing the development of SDB. Intervention (specialty-specific or interdisciplinary) is only recommended when the condition is present and properly diagnosed.

Orthodontics occupies a significant, albeit nuanced, position in the interdisciplinary management of SDB, particularly in pediatric populations. The role of orthodontists extends beyond traditional malocclusion correction, encompassing screening, referral, and potential interventional strategies within the broader context of sleep medicine.

The areas of the 2019 manuscript that benefit from current knowledge and practice updates are emboldened below.

ENDOTYPES, PHENOTYPES, AND NATURAL HISTORY OF SLEEP-DISORDERED BREATHING

In medicine, an endotype refers to a subtype of a disease defined by a distinct underlying biological or pathophysiological mechanism, which differs from the outward symptoms or clinical presentation (phenotype). SDB represents a heterogeneous disease with variable endotypes and phenotypes, as well as heterogeneous responses to treatment.[8] There are distinct underlying mechanisms (endotypes) behind the repeated collapse of the upper airway during sleep and their associated biological disturbances, influenced by anatomic and nonanatomic factors.[8,9]

Anatomic factors include increased soft tissue volume in the pharynx, such as enlarged adenoids or palatal tonsils, fat infiltration in the pharyngeal walls or tongue, and a reduced volumetric skeletal framework (among others). Nonanatomic factors involve neuromuscular function (the ability of pharyngeal dilator muscles to resist collapse), impaired neurologic control of breathing (high loop gain), and heightened sensitivity to sleep fragmentation (low respiratory arousal threshold).

These mechanisms explain why some individuals with morbid obesity and visibly narrow airways might not have SDB when assessed clinically, if their muscle responsiveness can compensate.[9] Conversely, some patients have airway collapse because of weak muscles, whereas others may have problems with how their central neural system controls breathing.

In the field of sleep medicine, the phenotype does not only refer to the craniofacial anatomic aspects, but also to the symptoms (daytime fatigue or sleepiness and sometimes associated insomnia), comorbidities (cardiovascular risk, elevated blood pressure, and failure to thrive in children), age, sex, or the response to treatment.[10,11] Different phenotypes have been described in adults and children, with the idea of tailored treatment implementation. Still, no clear guidelines and definitions are available, as no validated consensus has been adopted.

It is also essential to consider SDB over time, as the disease's evolution is likely to change significantly throughout a patient's life, which is referred to as its natural history (ie, its progression in the absence of treatment). For adult SDB, the general trend is a worsening of the condition after the age of 50, with women, who were relatively protected up until that point, eventually reaching prevalence progression rates similar to those of their male counterparts.[12-15] The literature suggests a relationship between reduced neuromuscular tone and the age-related increase in SDB prevalence for normal-weight adults.[16]

In contrast, the trajectory of SDB in children is entirely different, as prepubertal OSA tends to resolve naturally during the transition to adolescence. Primary snoring and mild SDB do not appear to be strongly associated with progression to more severe SDB; in other words, there is a tendency towards spontaneous remission of SDB from preschool years to adolescence.[17,18] However, specific phenotypes, particularly male children and those who are overweight, seem to be exceptions to this trend. An interdisciplinary intervention may still be indicated in select patients in consultation with a prescribing physician. As previously mentioned, SDB is a medical condition with potentially deleterious and irreversible effects on developing children, with medical and clinical guidelines implementing treatments notably to neurocognitive morbidity, associated conditions, and abnormalities of sleep studies.[3-7]

CRANIOFACIAL PHENOTYPES IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

There have been conflicting findings regarding the maxillofacial skeletal characteristics associated with OSA in both nonsyndromic children and adults. Some studies identify mandibular retrusion, facial hyperdivergence, and increased facial height as risk factors for developing OSA. In contrast, others suggest that a larger mandibular width or facial width may also contribute to the condition.[19-21] Recent meta-analyses investigating the relationship between craniofacial characteristics and SDB either found no direct causal relationship or remained inconclusive about such a relationship.[22,23]

A recent study compared 3 clusters of children presenting with OSA symptoms, divided by phenotypes, and found that it could not identify a distinct pediatric OSA phenotype based solely on soft tissue facial features or craniofacial abnormalities.[24]

Similarly, a multicenter Canadian study involving 315 children found no significant differences in facial or dental morphology between those with and without polysomnography-confirmed OSA, suggesting a limited role of craniofacial structure in pediatric OSA pathophysiology.[25]

Future studies may enable a more precise individualization of craniofacial phenotypes, taking into account interethnic comparisons. Although future studies may reveal associations between craniofacial phenotypes and the risk of SDB in a yet-to-be-identified subset of pediatric patients with SDB, current evidence does not support this concept.

RISK ASSESSMENT AND CLINICAL EVALUATION

A proper SDB risk assessment in an orthodontic office involves a combination of medical and dental history reviews, clinical and radiographic evaluations, and an objective risk assessment questionnaire, specifically tailored for children (under 18 years of age) and adults. As stated in the AAO 2019 manuscript, the clinical evaluation of SDB in children should include the assessment of tonsils, tongue size, and position, the presence of overweight or obesity, and the patient's overall growth and development.[1] Snoring, as well as mouth breathing or chronic nasal obstruction, should be considered, particularly in children. Currently, there is no standardized, universally accepted method for assessing mouth breathing. Although some mouth breathing is normal, the proportion of mouth vs nasal breathing that triggers a physiological response remains unknown.[26] A referral to a physician is appropriate for any concerns related to nasal obstruction.

Several systematic reviews investigating questionnaires for SDB risk assessment published after 2019 confirm the value of the Pediatric Sleep Questionnaire (children and adolescents) and the STOP-Bang Questionnaire (adults) tools as the most efficient and practical ways to incorporate objective risk assessment in an orthodontic office easily. It is recommended that every patient identified as high-risk be referred to a physician for proper diagnosis before the orthodontist provides any intervention linked to SDB management.[27-33] Although questionnaires are useful for screening sleep apnea risk, only physicians can formally diagnose OSA, and home sleep tests should not be treated as substitutes when ordered by nonphysicians.

USE OF IMAGING IN DIAGNOSING OR EVALUATING CHANGES IN SLEEP-DISORDERED BREATHING

OSA is a severe type of SDB, and polysomnography, combined with clinical symptoms, remains the gold standard for diagnosing OSA.[34] Imaging protocols may facilitate anatomic visualization of the airway collapse site during sleep, thereby contributing to an understanding of the patient's presentation, albeit without functional assessment. A patient's presentation refers to the signs and symptoms that lead to a medical evaluation and includes the patient's history, physical examination findings, and diagnostic results, which are used to convey relevant details leading to treatment planning.[35] Examples of such imaging techniques include cephalometric radiographs, cone-beam computed tomography (CBCT), magnetic resonance imaging (static and dynamic), and drug-induced sleep endoscopy. Recent research culminated in an international consensus challenging the notion that upper airway volume is correlated with SDB pathogenesis.[34] The risk assessment of SDB is complex and multifactorial, necessitating a more nuanced understanding of upper airway physiology and neuromuscular control.

A growing trend in upper airway analysis using CBCT or lateral cephalometric radiographs is emerging within the dental community, with some professionals using these analyses to imply diagnosis of the condition. Although CBCT and cephalometric radiographs are valuable for assessing dental and skeletal structures, they are not recommended for diagnosing OSA because of fundamental limitations.[36] The 5 main limitations of these 2-dimensional and 3-dimensional imaging techniques are as follows: (i) patient positioning during image acquisition; (ii) the nonanatomic etiology of OSA in a significant number of patients; (iii) inconsistencies in upper airway analysis methods; (iv) the static nature of these images, whereas OSA is a dynamic condition within the respiratory cycle; and (v) the fact that the imaging is acquired in awake state, whereas the tone of muscles is significantly reduced during sleep stages.[37-41] Most studies on upper airway dimensions in patients with OSA have collected data from patients in an upright position, even though they are typically in a supine position while asleep.[39] This discrepancy affects the reliability of upper airway dimension assessments, as head positioning during image acquisition significantly impacts airway analysis.[37]

In summary, imaging of the upper airway using CBCT or lateral cephalograms has no diagnostic value for SDB risk assessment or diagnosis.[34] The same can be articulated for upper airway measurements through rhinometry or pharyngometry.[34] Such measurements can help screen for anatomic obstacles involving the nasal and pharyngeal passages, which are of interest to physicians, notably ENTs engaged in patients' medical or surgical management.

Using changes in upper airway dimensions to suggest the efficacy of orthodontic or dental treatment is

scientifically flawed. Increasing upper airway volume or dimensions does not necessarily signify functional improvement or effective management of OSA.[34] Although airway dimensional changes may seem visually appealing to patients, the multifactorial nature of OSA limits their clinical significance.

FRENECTOMY AND FRENOTOMY

Ankyloglossia (tongue-tie) remains a topic of debate in pediatric sleep medicine, with conflicting evidence regarding its role in OSA. Systematic reviews and meta-analyses published since the AAO 2019 white paper have highlighted heterogeneity in diagnostic criteria, treatment outcomes, and long-term impacts related to OSA.

A 2023 scoping review of 26 studies (>1228 patients) identified associations between untreated ankyloglossia and OSA risk factors, including reduced maxillary width, elongated soft palate, and retroglossal collapse.[41] However, causal relationships remain unproven, as only 1 randomized trial suggested attenuated apnea severity postfrenectomy.[42]

The American Academy of Otolaryngology-Head and Neck Surgery's 2020 clinical consensus on ankyloglossia in children concluded that ankyloglossia does not cause OSA.[43] The consensus states that "surgery to release a buccal tie should not be performed and that the anterior tethering of the tongue serves to prevent the posterior collapse of the tongue, so if the frenulum is released, it could lead to OSA worsening."[42]

The American Academy of Pediatrics' 2024 revised breastfeeding guidelines note the benefits of frenectomies for latch difficulties, but caution against extrapolating these outcomes to OSA prevention, stating that performing frenectomies to prevent issues, such as speech articulation or OSA, is not evidence-based.[44]

Persistent limitations in the literature include diagnostic variability, a lack of standardization across studies, and a lack of a uniform definition of the degree of restriction that contributes to altered form or function, except in the most severe forms of ankyloglossia. Also, numerous studies combine adenotonsillectomy, rapid palatal expansion (RPE), and frenectomy, conflating the benefits of any given single targeted therapy.

Although limited studies suggest ankyloglossia may contribute to SDB risk in select patients, current data do not support routine frenectomy for SDB prevention or treatment. Health care professionals are encouraged to educate their patients about the misinformation surrounding this topic.[45]

PALATAL EXPANSION

Palatal expansion is an intervention in which the palatal suture is separated, also known as skeletal palatal expansion. The current evidence from systematic reviews with meta-analyses suggests a more nuanced perspective on the relationship between RPE and pediatric OSA. Although RPE remains a well-established orthodontic treatment for palatal constriction, its efficacy in managing SDB requires careful consideration. A 2023 meta-analysis by Yu et al found that RPE alone did not show a statistically significant reduction in the apnea-hypopnea index (AHI) compared with the control.[46] This review also found that RPE + adenotonsillectomy showed the most substantial improvement in AHI.[46] Yet, a 2024 randomized controlled crossover trial of RPE and adenotonsillectomy suggested that the sequence of interventions significantly impacts treatment outcomes, with adenotonsillectomy showing a more significant reduction in the AHI than RPE.[47]

Although RPE may benefit patients with palatal constriction, it may be most effective as part of an interdisciplinary approach rather than a stand-alone SDB treatment.[48] Short-term benefits of RPE may include increased nasal cavity and nasopharynx volumes, and though they may contribute to reduced nasal resistance, the overall effect on functional sleep-breathing is variable.[48]

Although using skeletal-anchored palatal expanders is expected to increase the magnitude of skeletal expansion, the impact of that additional skeletal expansion rate on breathing function has not been objectively assessed.

The orthodontic community should reserve the application of palatal expansion for SDB management for patients in whom a clear orthodontic indication exists alongside a confirmed SDB diagnosis. Currently, there is no evidence to support the prophylactic use of pediatric palatal expansion as a preventive measure against SDB over the lifespan. As such, orthodontists should continue to base their treatment decisions primarily on the presence of underlying skeletal discrepancies, collaborating closely with physicians when SDB is a concern.

FUNCTIONAL APPLIANCES

Orthodontic interventions targeting jaw growth and development have been proposed as potential adjunctive therapies for pediatric SDB. Althoug some studies suggest these approaches may increase upper airway dimensions and potentially improve airflow dynamics during sleep, dental professionals must maintain a critical perspective on the efficacy and limitations of such interventions.[49,50]

Currently, there is no evidence to support the prophylactic use of functional appliances as a preventive measure for SDB over the lifespan. As in the previous section, orthodontists should also continue to base their treatment decisions primarily on the presence of underlying skeletal discrepancies, collaborating closely with physicians when SDB is a concern.

ORTHODONTIC EXTRACTIONS

The role of dental extractions in orthodontic treatment and their potential impact on SDB has been a subject of considerable debate within the dental community. Dental professionals must approach this topic with a critical, evidence-based perspective. Dental extractions may be indicated in patients with severe crowding or to address significant dental protrusion. A decision to extract is based on comprehensive diagnostic criteria, including the amount of crowding, alveolar bone and periodontal support, facial esthetics, and occlusal relationships. Extractions continue to be an orthodontic option that should be used when indicated.[51]

Evidence on the effect of extractions on oral cavity dimensions is mixed, with no evidence to support a causal relationship between extractions and the development of SDB.[52]

After a review of extensive epidemiologic assessments, it is concluded that no substantive evidence supports a causal relationship between orthodontic extractions and airway obstruction.[53]

DISTALIZING MECHANICS

Distalization mechanics is a standard procedure used to generate space for crowding relief and alignment. Distalization can be accomplished using various appliances, including headgear, temporary anchorage-supported devices, brackets with compressed coils, and clear aligners.[54,55]

The use of distalizing mechanics on SDB patients may raise concerns regarding the possibility of anteroposterior narrowing; however, current evidence shows that distalizing teeth does not inherently constrict the airway.[56,57]

ORAL APPLIANCE THERAPY FOR OBSTRUCTIVE SLEEP APNEA

There are 2 very different types of oral appliances used for SDB management: mandibular advancement and myofunctional appliances. Mandibular advancement devices (MADs) are oral appliances worn during sleep by adults with OSA. Their primary function is to prevent airway collapse mechanically by posturing the mandible and tongue forward, thereby stretching the pharyngeal walls and maintaining airway patency during sleep.[58,59] The efficacy of MADs must be titrated, meaning the optimal degree of mandibular advancement is individually determined through symptom resolution and sleep study data (with the appliance in the mouth).[60] In contrast, myofunctional or orthopedic functional appliances, typically worn by growing children and fabricated following the same principles (anterior mandibular positioning), are designed to influence craniofacial development by redirecting condylar and dentoalveolar growth.[61] Although they may incidentally reduce upper airway obstruction during sleep in children during treatment, their impact on OSA is assessed after treatment, with a sleep study without the appliance in situ, since the goal is long-term anatomic change rather than mechanical airway support. Some clinicians report positive outcomes with MADs in children, particularly in carefully selected patients (mean reduction of AHI by approximately 2 units, along with other markers of apnea and hypopnea events in children with OSA compared with controls). Still, the current scientific literature remains limited in both quantity and quality, as well as the stability of any portrayed improvements. As a result, there is insufficient evidence to support the routine use of these devices for pediatric OSA.

Oral appliance therapy is an effective method for managing upper airway collapse in adult patients with OSA. A recent meta-analysis of 33 studies involving 1883 patients reported that MADs improved the AHI by 48%-67% across different OSA severity levels.[62]

Despite their effectiveness, MADs have both short and long-term side effects. Short-term effects are generally manageable, including dry mouth, excessive salivation, morning bite changes, and soreness in the gingiva, teeth, or jaw. However, long-term effects, including irreversible tooth movement and occlusal changes, require careful consideration.[63-65]

For adults, MADs can be a viable long-term strategy for patients who consistently wear their device.[66] Innovations, such as smart wear monitoring, optimized titration based on real-time usage data, and interdisciplinary follow-up care, enhance treatment effectiveness. Tailoring therapy to individual patient needs can further improve the efficiency of MADs in managing OSA.

COLLABORATIVE APPROACH TO SLEEP-DISORDERED BREATHING MANAGEMENT

Effective management of SDB necessitates an interdisciplinary approach, integrating various medical specialties and dental disciplines. As orthodontists, we occupy a unique position in this collaborative effort, particularly in the early detection and treatment of

SDBs in both children and adults. Our expertise in craniofacial development enables us to make significant contributions to the comprehensive care of patients with SDB.

Both medical professionals and dentists share the responsibility of screening for SDBs, whereas physicians remain responsible for the definitive diagnosis. It is strongly recommended that comprehensive SDB risk assessments be incorporated into routine orthodontic examinations. The management of accompanying comorbid presentations often requires referral to appropriately positioned physicians for further disposition. When there is suspicion of possible SDB, the orthodontist should refer the patient to a physician for a definitive diagnosis before any intervention. After a physician makes a proper diagnosis, orthodontists can offer valuable interventions, such as rapid maxillary expansion, maxillary and MADs, and surgical preparation for dentofacial abnormalities coincident with SDBs. These treatments should be implemented in close consultation with sleep physicians and other specialists to ensure that they complement the overall treatment strategy. Collaborative treatment planning between medical specialties and dental disciplines frequently establishes a bidirectional pattern of referrals. Such collaboration ensures that patients receive comprehensive care, often involving the combination of multiple therapeutic approaches for optimal outcomes, and may include coordinating orthodontic interventions with specialties, such as sleep medicine, otolaryngology, and oral surgery, among others.

The ongoing relationship between orthodontists and their patients may better facilitate long-term monitoring of SDB conditions. Patients being treated for SDBs should be assessed yearly for maintenance of their condition and possible signs of deterioration. The combined efforts of the orthodontist and other dental disciplines, along with medical professionals, enhance the continuity of care provided by the team.

EXECUTIVE SUMMARY

As concluded in the 2019 AAO white paper, SDB is a medical disorder that can have many serious consequences if left untreated. SDBs can affect adults and children and can develop at any point in the lifespan. All orthodontists should consider incorporating SDB objective risk-assessment methods. Upon suspicion of the disorder, it is strongly recommended that referral to a physician be made for a definitive diagnosis before any intervention.

The revision of current literature suggested that:

1. SDB encompasses a spectrum of breathing abnormalities, ranging from habitual snoring to OSA. SDBs can significantly impact the developing child and adults, and early detection is beneficial. Only a physician can diagnose SDB, and no SDB-related intervention is appropriate before proper diagnosis. Patients with SDB are ideally cared for with an interdisciplinary team.
2. Current evidence does not support that any orthodontic intervention, such as maxillary expansion or functional appliances, can prevent the development of SDB. There is no evidence to support the prophylactic use of pediatric palatal expansion as a preventive measure for SDB at any age. As such, orthodontists should continue to base their treatment decisions primarily on underlying skeletal discrepancies, collaborating closely with physicians when SDBs are present.
3. Current evidence does not support that orthodontic treatment is a stand-alone therapy for managing SDB.
4. No currently known craniofacial phenotypes can identify the presence of SDB.
5. Imaging analysis of the upper airway, such as widths, volumes, and areas, using cephalograms or CBCTs, is not suitable for diagnosis, risk assessment, or outcome assessment of intervention in SDB. This finding differs from the 2019 white paper.
6. The use of validated questionnaires is an effective method for assessing SDB risk. Once a risk is identified, a referral for a definite diagnosis by a physician is strongly recommended before any intervention.
7. Current evidence does not support ankyloglossia as an SDB etiology. Routine frenectomy is not recommended for SDB prevention or treatment.
8. Current evidence does not support that traditional orthodontic procedures and mechanics, such as extractions and distalizing mechanics, affect the etiology or increase the likelihood of any SDB.
9. When an orthodontist is involved, it is strongly recommended that the management and treatment of SDBs be an interdisciplinary collaboration between dental and medical health care professionals.

CONCLUSIONS

A review of the 2019 AAO white paper on the orthodontist's role in managing OSA reinforced some previously shared evidence and added new insights that are now shifting from anatomic phenotypes to functional analysis.

As a health care professional, the orthodontist can play an essential role in screening for SDB conditions.

Ideally, the orthodontist would be part of an interdisciplinary medical team when any SDB management is taking place.

AUTHOR CREDIT STATEMENT

Juan Martin Palomo, Julia Cohen-Levy, Carlos Flores-Mir, Rooz Khosravi, Mitchell Levine, Michael Pickard, John Callahan, and Steven M. Siegel contributed to conceptualization, investigation, and original draft – review & editing. Jackie Hittner contributed to supervision and literature search support.

SUPPLEMENTARY DATA

Supplementary data associated with this article can be found on the American Association of Orthodontists' library Web site (https://www2.aaoinfo.org/library/), in the online version, at https://doi.org/10.1016/j.ajodo.2026.01.014.

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