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Ten Misconceptions That Dentists Have About Treating Obstructive Sleep Apnea

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Evidence-based learning has only recently been incorporated into the dental sleep medicine curriculum. As an increasing number of dentists screen for and help treat obstructive sleep apnea (OSA), research-based learning must guide dental sleep medicine practice parameters. Dentists must understand the evidence-based medicine behind OSA and oral appliance therapy (OAT) and partner with a physician who is well versed in the treatment of sleep disorders. This article addresses the 10 misconceptions that many dentists have come to believe in the field of dental sleep medicine, and it uses published data to explain that many of these concepts are not supported by science. Evidence-based dental sleep medicine knowledge and protocols are necessary in order to provide optimal patient care, and dentists must understand that dental sleep medicine is an evolving field with a growing body of information that will continue to challenge previously accepted concepts.

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INTRODUCTION

Dental sleep medicine (DSM) and the treatment of obstructive sleep apnea (OSA) and snoring with oral appliances is a relatively new field of medicine that lies within the purview of dental therapy. Unlike much of dentistry where concepts were often created based on clinical experience of the lecturer, DSM is a medical field in which dentists interact closely with physicians; these physicians expect concepts based on published literature and strong research. Evidence-based dentistry is also a burgeoning concept. Currently, there is no standardized curriculum or academic programs that are comparable from one educational venue to another. Competencies are just beginning to be defined.[1] With newer research, more information is added to the lexicon and ideas perforce change, which leads to changes in techniques and education. Currently, what is taught in a university program may not be mirrored in a for-profit lecture series presented by a dental laboratory. Because the field of DSM is undertaught in dental schools[2-4] and many well-known advanced education venues for dentistry are managed by educators who may know very little about DSM, these venues are at the mercy of their lecturers who may be biased in the presentation of information possibly based on commercial interests or outdated information. Using a private archive of 2,500 articles gathered over 30 years in the field of DSM, this paper undertakes the presentation of 10 areas of possible misinformation that are not supported by newer research data despite possible underlying intuitive concepts.

Sleep-disordered breathing (SDB) is a chronic disorder that manifests by repeated upper airway collapse during sleep. This creates recurrent sleep hypoxia, increased sympathetic activity, and interrupted sleep[5,6] Over time, these physiologic changes can induce severe health problems and cognitive dysfunction.

SDB is a continuum with disorders of varying levels of airway closure encompassing a range of severity from primary snoring to obesity hypoventilation syndrome. OSA represents the more significant level of SDB, and untreated patients with moderate to severe OSA are at increased risk of several health issues such as diabetes,[7] hypertension,[8] stroke,[9,10] depression, and vehicular accidents.[11] In 1993, a large demographic study, the Wisconsin Sleep Cohort, determined that at least 4% of all men and 2% of all women in the United States had OSA syndrome that included significant daytime sleepiness.[12] Almost 20 years later, with an increasing incidence of obesity, improved diagnostic equipment, and a change in the definition of OSA, data from the Wisconsin Sleep Cohort shows that, by 2013, 13% of men and 6% of women had moderate-severe OSA.[13] Swiss researchers report that as many as 23% of women and 50% of men may have OSA.[14] In 1997 moderate to severe OSA was thought to be undiagnosed in more than 80% of middle-aged patients,[15] but updated literature indicates that number is closer to 75%.[16] OSA is an epidemic with significant medical and

financial consequences. OSA is increasingly recognized as an important public health issue, and dentists, working with physicians, play an important role in screening and offering a treatment option that may be optimal for many patients.

OSA is commonly treated with continuous positive airway pressure (CPAP), which requires a mask over the nose and/or mouth and a blower that creates enough pressure to keep the airway pneumatically stented open. CPAP is recognized as the gold-standard therapy and is the most powerful tool in the physician's armamentarium for treating OSA.[17] Multiple studies show that oral appliance therapy (OAT), which uses a custom-fabricated oral appliance (OA) to advance the mandible, may be as effective as CPAP in the health outcomes of patients with of mild to moderate OSA.[18-21] As an example, one long-term study on cardiovascular mortality in patients with severe OSA treated either with CPAP or OAT found that both therapeutic options appeared equally effective in reducing the risk of fatal cardiovascular events in patients with severe OSA.[19] Adherence to therapy significantly affects the positive outcomes, and actual health improvements may vary.[22]

Although many dentists believe that OSA is a purely anatomic disease caused by a narrow upper airway (UA), anatomy is only one of many factors that influences the presence and severity of OSA. The pathophysiology of OSA is extremely complex and incompletely understood. It is affected by age, body mass index (BMI), race, sex, pharyngeal muscle responsiveness, perturbations in ventilatory control stability, low arousal threshold, sleep-related decrease in lung volume, fluid redistribution, and UA surface tension.[23] Most patients exhibit multiple factors that lead to OSA.[24] There is not a single pathway for all patients, and the role of personalized medicine will hopefully allow better phenotyping of patients and selection of the best management strategies, be it CPAP or OAT with an exercise program, weight loss, or pharmaceutical adjuncts.[25]

MISCONCEPTION #1: WE NEED TO SCREEN EVERY PATIENT FOR OSA

There is a lack of knowledge about whether general screening is effective. Many studies have looked at screening by a variety of professional providers: internal medicine physicians,[26] pharmacists,[27,28] nurse practitioners, physician assistants, and dentists. One study looked at a large group of general dental patients who were screened using either a questionnaire, pulse oximetry, or both. Of the 1,800 patients who were notified about the study by e-mail, fewer than 240 consented to be screened, all of whom received financial incentive to participate.[29] Fewer than one-half of the patients found to be at high risk of OSA actually followed through with a physician consultation. Those most likely to seek medical evaluation had a high probability of OSA based on both screening tools and pulse oximetry. In this study, it appears that patients who self-elect for screening may already be concerned about symptoms or have a high positive interest in their own health status.

The American Dental Association (ADA) developed a policy statement that was posted online in 2017 (www.ADA.org/sleepapnea). The policy states, "Dentists are encouraged to screen patients for SRBD as part of a comprehensive medical and dental history to recognize symptoms such as sleepiness, choking, snoring or witnessed apneas and an evaluation for risk factors such as obesity, retrognathia, or hypertension. These patients should be referred, as needed, to the appropriate physicians for proper diagnosis.[30]"

A possible downside to general population screening could include over-diagnosis and overtreatment of patients with mild OSA who may require only education about factors that could exacerbate their OSA. These patients could overburden a medical system already working hard to treat patients with more severe diseases. Screening the general population might be a double-edged sword, leading insurance companies to increase premiums for health, life, and disability insurance for all of those in whom OSA has been diagnosed.

It would seem imprudent to encourage screening of asymptomatic patients in dental practices, primary care health practices, and pharmacies. The US Preventative Services task force (USPSTF) states that "evidence on the use of screening questionnaires in asymptomatic adults to correctly identify who will benefit from further testing for OSA is inadequate." The task force did conclude that screening symptomatic patients (those with snoring, witnessed apneas, excessive daytime sleepiness [EDS], or acute medical conditions that could bring on OSA) are the proper target population.[31]

Multiple studies show that mortality increases with moderate to severe OSA.[32-34] In contrast, mild OSA has not yet been proven to lead to adverse clinical outcomes,[35,36] and it is associated with similar outcomes compared to those who do not have OSA.[29] However, there are some people with mild OSA who are highly symptomatic, which could lead to an increased number of auto accidents, industrial accidents, and difficulty maintaining interpersonal relationships. Generally, untreated OSA resulted in a loss of productivity estimated at $86.9 billion in 2015.14 and treatment of symptomatic patients with mild OSA may be justified.[37]

Large demographic studies looking at improved work productivity, improved health outcomes, and the effectiveness of high-risk screening in the dental setting are lacking. Groups such as the American Thoracic Society published a research statement in 2016 stating that there is inadequate information about treatment of mild OSA as effective at preventing or reducing "long-term adverse neurocognitive and cardiovascular outcomes."[38]

Until studies show that screening the general public

leads to an improvement in the health of the population involved, data are inadequate to justify general population screening.[31] Screening in a dental office includes the use of various questionnaires such as the Berlin questionnaire,[39] Epworth Sleepiness Scale, and the STOP-BANG questionnaire.[40] If a patient has a high probability of having OSA based on a validated questionnaire and a contributory medical history, the dentist's responsibility is to refer that patient to a physician for evaluation and testing.

MISCONCEPTION #2: DENTAL SCREENING FOR OSA REQUIRES EQUIPMENT

Screening patients at high risk for OSA includes assessment of symptomatic patients (e.g. sleepy, history of snoring, witnessed breathing pauses, falling asleep in the dental chair, etc.), combined with the knowledge that the incidence of OSA is higher in obese patients, men, post-menopausal women, African Americans, and those with craniofacial abnormalities.[41] Screening can include questionnaires and clinical prediction tools that combine subjective and objective findings.

Companies that market home sleep apnea tests (HSATs) have targeted dentists as an audience for sales expansion, and sell these devices to dentists while asserting that these monitors are a 'screening tool.' Use of HSATs for screening in a dental environment is inappropriate and, in some states, illegal. These are legal diagnostic tests covered by most medical insurance plans and, therefore, fall within the purview of a medical provider. Many state dental boards have directly addressed a dentist's role in screening patients who may have OSA. The state of Oregon policy states, "The ordering, interpreting and managing of tests for sleep apnea is outside the scope of dentistry, whereas the making of the appliances is well within the scope of dentistry."[42] The Georgia Board of Dentistry adopted the policy stating "…a dentist may not order a sleep study. Home sleep [apnea] studies should only be ordered and interpreted by a licensed physician."[43] The New Jersey Board of Dentistry noted on March 4, 2015, "A dentist cannot order or interpret the home sleep test or screen, treatment plan or diagnose sleep apnea patients."[44] Therefore, any dentist who intends to fabricate OAs for patients with SDB must first know the law in the state where he or she practices and understand the ramifications of providing HSATs.

A position statement published by the American Academy of Sleep Medicine (AASM) clearly states that, "An HSAT is a medical assessment that must be ordered by a physician to diagnose OSA or evaluate treatment efficacy" and "An HSAT should not be used for general screening of asymptomatic populations."[45]

A question as simple as "Do you snore?" along with a review of the patient's medical history, may be all that is necessary to identify patients at high risk for OSA. No equipment, imaging, or airway studies are required or supported scientifically to screen for OSA in a general dental practice.

MISCONCEPTION #3: DENTISTS CAN TREAT A PATIENT FOR SNORING WITHOUT INVOLVING A PHYSICIAN

In the past, many dentists have treated their patients who complained of disruptive snoring with an "anti-snoring appliance." Many dentists assumed that, if the patient has no other comorbidities, such as obesity, EDS, or cardiovascular disease, that treatment with OAT required no medical diagnosis**.** Updated diagnostic criteria from the AASM no longer consider EDS to be a necessary finding in the diagnosis of OSA.[46] Many patients with severe OSA have no daytime sleepiness and actually claim to have good and restful sleep.

Fabricating an OA for a snoring patient may actually mask symptoms that would lead this patient to a physician for proper medical diagnosis. After the snoring stops, and the patient's spouse no longer complains about the noise, the patient's OSA may remain untreated with significant medical morbidity. *Presuming* that the patient has primary snoring is making a de facto diagnosis that the patient does *not* have OSA. This determination is beyond the purview of a dentist. Most dental professional liability insurers will cover a dentist in a malpractice suit involving a patient with snoring or OSA *only* if a physician has written a prescription for that appliance. Definitive diagnosis by a physician with a medical evaluation and appropriate testing is required before a dentist can treat any form of SDB.

MISCONCEPTION #4: IMAGING CAN DETERMINE WHO HAS OSA AND WHO WILL BE SUCESSFUL WITH OAT

Cephalometry

OAT for OSA in the 1980s was created by alteration of functional orthodontic appliances used to treat adolescents with class II malocclusions.[47] Cephalometrics are part of every orthodontist's armamentarium, and imaging of patients in whom OSA has been diagnosed was very common. Throughout the years, variable cephalometric findings were correlated with the presence of OSA and success with OAT. Retrognathia, narrower airway, shorter and thicker soft palate, or lower facial height were among those findings cited; these findings were inconsistent and none were predictive of the presence of OSA with a high sensitivity and specificity.[48] Early studies evaluating cephalometric variables and SDB concluded that " the lack of association between cephalometric variables and mild sleep apnoea suggests that the differences in these variables (soft tissue measures) may be the consequence of habitual snoring and the obstructive sleep apnoea syndrome.[49] Consistent with

previous studies, only 52% of patients with OSA have a reduced posterior airway space on imaging.[50]

Although the presence of craniofacial abnormalities has a strong relationship with OSA, these abnormalities are not diagnostic of OSA.[51] Decreased upper airway size has also been postulated to correlate with the presence of OSA, but men have a higher preponderance of SDB than women, whereas women have smaller airways than men.[52] Therefore, size of the airway does not necessarily predict the presence or absence of SDB. Another consideration is the fact that airway closure often occurs in a lateral dimension, not anteroposterior, and narrowing of this dimension would be missed by conventional lateral cephalometry. Those practitioners hoping to screen for OSA using cephalometry would have to strictly control for head position, breathing phase, and body position because the natural airway is much smaller when a patient is prone than when the patient is upright.[53] Other concerns include the effect of various cephalometric analyses used, which alters correlates with OSA.[54]

Gulati et.al. looked at the single determinant of sella-hyoid (S-H) distance, which was thought to predict OSA severity, and found, in their study, there was no correlation between patient's OSA severity and S-H distance, The authors concluded that use of the S-H as a screening test cannot be recommended as a substitute for existing diagnostic tests.[55] The relatively weak and somewhat inconsistent cephalometric data suggest that decisions based solely on cephalometric factors cannot be recommended, especially because an integrated analysis of other risk factors (eg, age, sex, BMI) should also be taken into account."[56]

Although many authors have found correlates between various cephalometric findings and the presence of OSA, there have been no prospective studies that correlate cephalometric measurements and the success of OAT. In the 2015 Guarda-Nardini et al. review "the mandibular plane angle and the distance between the hyoid bone and the mandibular plane were found to have a reasonable predictive value for MAD [OAT] success in OSA patients but did not accurately identify those who would fail OAT."[56] Ng et.al. found that the definition of success (50% reduction in AHI, a reduction in AHI of 50% with a residual AHI $\leq$ 10 or residual AHI $\leq$ 5) did not correlate consistently with any single cephalometric finding[57] and others found no correlation between cephalometric findings and OAT success.[58,59] Currently, cephalometry cannot identify who patients are at high risk for OSA nor who will find OAT an effective treatment.

Cone Beam Computed Tomography

As more and more dentists incorporate cone beam computed tomography (CBCT) into their practices, it presents a new modality for evaluating patient airways. Statistically, the UA of patients with patients with OSA is smaller than that of controls without the disease. CBCT can view the UA volumetrically when compared to cephalometry. Three-dimensional evaluation of the cross-section of the UA has been correlated with the outcomes on the Berlin Questionnaire, Epworth Sleepiness Scale, neck circumference, and patient BMI. A systematic review of three-dimensional imaging of the UA anatomy and OSA concluded that a minimal cross-sectional area is the most relevant anatomical characteristic of the UA related to the pathogenesis of OSA.[60] Although one study showed no significant difference in CBCT findings between moderate to severe OSA and mild to normal subjects,[61] there is a difference when using dynamic CT when imaging was done at end expiration,[62] a technique not often available in a dental office. In a recent study that controlled for BMI, age, and sex, the various subregions of the pharyngeal volume *did not* correlate with AHI. This points to the screening with standard questionnaires and standard anthropomorphic measurements, which allow the patient to avoid unnecessary radiation exposure with the same intended outcome.[63]

The same can be said about imaging performed with the OA in place. It has been hypothesized that CBCT evaluation of the UA structures may be helpful in determining treatment modality and monitoring the effectiveness of the OA.[64] To date, no study has shown a strong model for changes in UA size with or without an OA in awake patients as a predictor of successful therapy for OSA with OAT, possibly because biomechanical observations cannot be directly tied to clinical outcome.[65] Studies of healthy patients without OSA also show an increase in UA space with mandibular advancement.[66] Use of CBCT is also confounded by the lack of standardized scanning protocols and varying nomenclature used from one study to another.[67] With the current published data, there is insufficient evidence that CBCT airway dimensional changes are suitable for assessment of treatment outcomes,[68] and there are no prospective data on predication of success with OAT.

Acoustic Reflection

Developed more than 35 years ago, acoustic reflection (AR) is a noninvasive technique that uses sound reflection to infer the cross-sectional area of the UA. Studies have developed standard UA cross-sectional areas and verified that airway area inferred by AR correlates well with UA area determined with computed tomography (CT) and magnetic resonance imaging (MRI).[69,70] Snorers were found to have a smaller mean pharyngeal cross-section, after breathing out normally, than nonsnorers. Moreover, after breathing out maximally, snorers with OSA and non-snorers had a further reduction in UA area, whereas snorers without OSA had no such decrease.[71] Mandibular advancement has shown to have its major effect in increasing UA dimensions behind the soft palate and the

tongue. Researchers hypothesized that AR could identify the narrowest area of the UA, and those patients shown to have the narrowest airway area behind the palate or tongue would be successful with OAT. Results show that there was no correlation between the narrowest part of the UA as determined by AR and success with OAT.[72] The only article that directly addressed using AR to predict the appropriate position of the mandible for optimal OA effect was a single case study.[73] There is no literature that shows AR to be predictive of the presence of OSA or to determine the mandibular position that would lead to success with an OA.

Although various forms of imaging may help identify the narrowest location of the UA, they have not been shown to predict the location of airway collapse, who may or may not have OSA, or who will positively respond to OAT.

Given all the options for imaging patients, *required* imaging appears to be simply a panoramic or full-mouth series to establish the health of the supporting dentition prior to initiating OAT.

MISCONCEPTION #5: OAT IS AS UNIVERSALLY EFFECTIVE AS CPAP

There is a general misconception among dentists that OAT is as effective as CPAP. The collapsible airway in men is approximately 5 inches long, and in women it is approximately 4 inches long.[74] Collapse can occur behind the soft palate, the tongue, and/or hypopharyngeally; the collapse can be anterior/posterior, lateral, or concentric.[75] CPAP is the only treatment that is not site specific and effectively pneumatically stents open the entire collapsible airway. OAT has its most significant effect behind the tongue, less so behind the soft palate, with decreasing effect closer to the epiglottis; in those patients with supine positional sleep apnea who have a significant component of epiglottic blockage, the positive effect of OAT is minimal.[76] In summary, CPAP is not site specific, whereas OAT is site specific, making it less likely to adequately treat unselected patients.

What makes OAT appealing is that it has a much higher rate of patient adherence than CPAP.[77] The average use of CPAP is less than 4.6 hours per night,[78] whereas the average use of an OA is 6.8 hours per night.[79] A patient with mild-moderate OSA may not have his or her OSA fully controlled by the OA, but uses the appliance all night and is expected to achieve equivalent medical outcomes whether using CPAP or OAT. For both of these major treatments, there is a huge dropout rate within the first few months, and more than 30% of patients discontinue using either OAT or CPAP fairly quickly.[80] Adherence to OAT appears to be higher in those who have a successful outcome and resolution of their symptoms.[81] Favorable outcomes with OAT are more common in thin patients younger than 55 years, those who require CPAP <13 cm, or those who have mild-to-moderate OSA with oropharyngeal collapse,[82] which is expected to improve adherence to therapy.

Recent literature shows that there is a high likelihood that OAT will become less effective with time despite control of subjective symptoms. Although many studies show an improvement in the effectiveness of OAT over 5 years, beyond 15 years this improvement seems to wane,[83] but part of this may be the known increase in severity of the disease with aging.[84] No one knows what other factors may play into this loss of positive effect, and it remains unclear why patients continue to feel well rested and report control of snoring despite exacerbation of disease with the OA in place. It is unknown if further mandibular advancement will improve outcome sleep parameters in long-term OA users. Given that there are multiple causes for OSA, that OAT may decrease in efficacy with time, and there are multiple treatments available, every dentist must understand that treating OSA and managing a patient's disease requires a multidisciplinary approach.

MISCONCEPTION #6: OAT HAS NO APPRECIABLE SIDE EFFECTS

OAT is based on an orthodontic device that was invented by Emil Herbst in 1909.[85] It was designed to permanently advance the mandible in children with retrognathia. Therefore, it would seem intuitive that the major side effects could be orthodontic in nature. Multiple studies show that this is indeed what happens with a tendency for the entire mandibular dentition to drift forward, accompanied by significant labial tipping of the mandibular incisors.[86] Because of opposing forces on the maxilla, the maxillary incisors tend to tip lingually; there is also a change in intermolar width. This reduces overjet and overbite, which can lead to incisor prematurity and a posterior open bite. These orthodontic changes in patients using OAT are permanent and continue to progress with prolonged OA use.[87,88] Long-term (> 7 years), dental changes occur in more than 80% of OA users, but the patient is rarely aware of these changes and alteration in occlusion is an unusual reason for discontinuing OA therapy.[89] Morning occlusal guides have been introduced in an attempt to mitigate tooth movement, but there are no studies, to date, on the effectiveness of this intervention. Dentists seem to be more concerned than patients about alterations in occlusion, and adequate treatment of a serious form of a medical disease is more important than occlusal concerns as long as there is no significant alteration in function.

There has been concern on the part of sleep physicians that OAT can cause significant temporomandibular joint pain and dysfunction. Research studies show that while the OA is being titrated to an effective position, there may be a transient increase in temporomandibular joint signs and symptoms, but these resolve over the long term.[90] Interestingly, one study shows that over 50% of patients

with OSA present with symptoms of temporomandibular joint dysfunction pretreatment.[91] Careful titration and follow-up support from the dental provider are often all the care that is required.[92]

Other side effects are often self-limiting and may or may not lead to discontinuation of OAT. These include tooth discomfort, gingival discomfort, dry mouth, poor fit of the OA, mouthpiece breakage, muscle discomfort, and increased salivary flow. The major reason patients discontinue OAT is lack of effectiveness and continuing symptoms.[93] As many as 45% of patients are inadequately treated,[94] and need to return to the sleep physician to discuss alternative treatment options.

Side effects are extremely common and continue through the duration of OA use. Temporomandibular symptoms decrease with continued use of an OA, whereas occlusal changes and tooth movement increase. Restoration of open contacts, dental rehabilitation to re-create posterior support, occlusal adjustment, or orthodontic care are contraindicated unless there is a significant functional impairment[92] and the patient is willing to discontinue OAT. Patients may be willing to return to CPAP part time if this will minimize alterations in the dentition. Alternating one therapy with another, including expiratory positive airway pressure, positional therapy, weight loss, and consideration of surgical interventions, are options that may be required.

MISCONCEPTION #7: THE MORE THE MANDIBLE IS ADVANCED THE MORE EFFECTIVE THE OA

Studies attempting to determine the effect of mandibular advancement on airway size and shape or to pinpoint the optimal mandibular advancement to control the patient's SDB have been ongoing since the 1990s. The early studies were done on patients under general anesthesia,[95,96] on nonapneic patients,[97] and awake patients.[66] Research by Kato et al. in 2000 looked at the number of oxygen desaturations when patients under general anesthesia had their mandibles advanced 2 mm, 4 mm, and 6 mm. The authors found a 50% reduction in oxygen desaturation index in 25% of patients at 2 mm advancement, 48% at 4 mm, and 65% at 6 mm. The requirement for further advancement was significantly dependent on body size and the severity of nocturnal desaturation.[98] Many dentists extrapolated the findings of these studies to naturally sleeping patients with OSA and arbitrarily decided to start mandibular advancement at more than 65% of the patient's protrusive range, disregarding that more than 30% of patients were effectively treated at a lesser advancement.[99] Researchers hypothesized that lesser mandibular advancement might lead to adequate treatment of the OSA with fewer side effects.[100] Four-year follow-up of patients wearing an OA set at 50% advancement showed virtually no tooth movement and acceptable control of OSA.[101] Studies done on naturally sleeping patients wearing appliances set at very low levels of mandibular advancement are more recent. Mandibular advancement as low as 1 mm is effective in treating some patients with OSA.[100,102] Statistically, further advancement does correlate with further decrease in AHI[99,101] which is found necessary in heavier patients and those with severe OSA.[103,104] If a lesser advancement is effective in adequately controlling the SDB, and side effects are minimal, then long-term use of an OA is expected to be less deleterious to the dentition and allow for more years of OA use.[101]

MISCONCEPTION #8: HSATs CAN DETERMINE THE OPTIMAL POSITION FOR AN OA

Although HSAT is the best empiric test that a dentist may have to evaluate the effectiveness of mandibular advancement OAs, it may not truly assess exact outcomes. Guidelines for the use of HSATs say that it is effective in determining the presence of OSA in uncomplicated adult patients presenting with signs and symptoms that indicate an increased risk of *moderate to severe* OSA and is less accurate in those with mild OSA.[105] After treatment with OAT, the expectation is that the patient will have no or only residual mild SDB, which no longer meets criteria for expectation of accurate HSAT results. Use of pulse oximetry, high-resolution pulse oximetry, and the plethora of HSAT devices tends to underscore those patients who suffer from a low arousal threshold and whose symptoms are related to frequent shifts in sleep level. Although many of the equipment manufacturers state that their equipment is comparable to overnight in-laboratory polysomnography (PSG), that is only true in specific situations.[106] Because HSATs often underscore the number of breathing events, the patient's symptomatic relief and bed partner's report are often required to determine if treatment is effective. OSA has a bipartite effect on the patient: frequent sleep arousals caused by breathing events or other sleep disturbances, which often lead to EDS, fatigue, and difficulty functioning during the day.[107] Oxygen drops or intermittent hypoxia related to breathing events trigger the overt medical problems related to endothelial dysfunction,[108] heart attack, stroke, and alterations in endocrine function such as insulin insensitivity.[109,110] HSAT is thought to be more accurate in measuring oxygen level and hypoxia.

There is much controversy over the use of HSATs in a dental setting. The AASM considers an HSAT to be a medical assessment that must be ordered by a physician. With state dental boards having minimal guidelines to follow, the ADA developed a policy in 2017 that a dentist may use HSAT, pulse oximetry, and high-resolution pulse oximetry to aid in the titration of an OA, but it is up to the *physician* to actually determine whether the patient is adequately treated. Knowing that OAT can completely

control OSA in only a portion of patients, there will be many patients who continue to use their appliance as a "better than nothing" treatment. The ADA strongly advises a close relationship between the dentist and the treating physician when using HSAT to determine the improvement in hypoxia when a patient with OSA is treated with OAT.

Sleep physicians expect CPAP to reduce the number of breathing events to fewer than five per hour. They have subsequently expected the same results from OAT. Ear, nose, and throat surgeons define success of UA surgery as a decrease in breathing events by 50% with a final AHI <20. However, other criteria must also be used to determine whether the patient is successful with therapy, including compliance with the OA, improvement in physiologic parameters (such as blood pressure), and neurobehavioral findings (often addressed with questionnaires), as well as symptomatic improvement. Medicine and dentistry will eventually settle on a definition of success that relates to more than just improvements in PSG outcomes. The basis of success should be the medical outcomes, reduction in related disease states, and improved daytime functioning of patients with OSA.[111] Optimal mandibular positioning must be determined by physician input about the sleep study parameters as well as patient symptoms and side effects.

MISCONCEPTION #9: ORTHODONTIC THERAPY CAN CAUSE OR PREVENT OSA

There has been significant interest in the role of orthodontics adding to the burden of OSA, or having the ability to mitigate OSA, in both children and adults. It was believed by many dentists that four-bicuspid extraction orthodontics could aggravate OSA; it was thought that retraction of the incisors would contribute by crowding the tongue and decreasing UA space. Orthodontists started correlating changes in UA dimensions after orthodontic therapy with the probability of an OSA diagnosis. In 2010 a study was published showing there was no statistical change in the volume of the UA between those patients who underwent either extraction or nonextraction orthodontics.[112] In 2015, Larsen et al. reviewed the medical records of 5585 adult patients at the HealthPartners of Minnesota. Half of the patients had one bicuspid missing in each quadrant and were assumed to have had orthodontic treatment earlier in life. The subjects were case controlled, matched for age, sex, BMI, and the diagnosis of OSA confirmed by PSG. This record review determined that 267 of those without missing bicuspids had received a diagnosis of OSA and 299 subjects with missing bicuspids had received a diagnosis of OSA. The prevalence of OSA was therefore not significantly different between the two groups.[113]

Childhood OSA is most common between the ages of 2 and 7 years, when lymphoid tissue (tonsils, adenoids) is largest. For this reason, the major treatment for childhood OSA is adenotonsillectomy or surgical removal of the enlarged lymphoid tissue. Although this corrects the problem for many patients, there is a large subset of children with residual OSA.[114] It has been found that a small maxilla or mandible may predispose children to SDB mediated by high nasal resistance and mouth breathing, which then alters tongue position and oropharyngeal volume. Rapid maxillary expansion (RME) decreases nasal resistance and allows the tongue to rise toward the palate, improving muscle tone and aiding nasal breathing. Studies of children with craniofacial abnormalities and OSA treated with RME show a good response with a decrease in AHI and long-term resolution of their SDB.[115,116] When comparing the two options (adenotonsillectomy versus RME) in children with both enlarged lymphoid tissue and/or malocclusion, those children with an AHI > 5 automatically went through surgical intervention first, despite the fact that 84% of these children had dental malocclusions and a narrow palate.

Those children, older than 4 years, with an AHI < 5 and a narrow maxilla underwent RME as a first-line therapy. Subjects who underwent RME as their first line of therapy were found to have higher posttreatment AHI than those who underwent surgical intervention, even though they initially had a milder form of the disease. Regardless of first-line therapy, approximately 60% of the children displayed residual disease after singular therapy, which underlines the high possibility of long-lasting disease despite therapy and the fact that OSA remains a complex disease with multiple interconnected causative factors.[117] A significant number of children who underwent bimaxillary expansion had worsening of their SDB.[117] Obese children are often incompletely treated by either therapeutic approach, so weight loss is also an important part of therapy.[118] This also points to the need to consider multiple therapies and the need for cooperation among treating specialists.

Kikuchi hypothesized that orthodontic treatment of children with OSA would prevent SDB as they got older. This was based on the functional matrix growth theory.[119] There are no studies that support this theory, and prognosis and positive effects of long-term intervention for OSA remains uncertain, requiring periodic reevaluation and, if necessary, instituting new interventions to control malocclusions and possible incidence of OSA.

To date, all studies that evaluated RME or arch expansion for the treatment of OSA in children were done on small groups of patients, all of whom had a craniofacial need for RME. Orthopedic mandibular advancement or RME may be useful to correct craniofacial morphology. In the Cleveland Children's Sleep and Health Study, a racially mixed urban community-based cohort and not a group chosen for preexisting craniofacial abnormalities, researchers found that there was a high probability of remission of childhood OSA, with minimal data showing a

continuation of disease from middle childhood to late adolescence.[120]

In adults as with children, studies have found that surgical maxillary expansion helps to reduce AHI in those with transverse deficiencies.[121-123] There are no data that would support orthodontic therapy of patients with OSA who do not have an underlying craniofacial abnormality.[124]

MISCONCEPTION #10: THERE IS A CAUSE AND EFFECT TRIAD OF OSA, SLEEP BRUXISM, AND GASTROESOPHAGEAL REFLUX DISEASE

There is intense interest in the field of dental sleep medicine looking at the relationship between OSA and other disease entities that affect the dental condition. Although the coexistence of two disease states does not imply cause and effect, there is continued speculation on the relationship between OSA, sleep bruxism (SB), and gastroesophageal reflux disease (GERD), which is found in 20% to 35% of the general population.[125]

GERD and OSA

The relationship between GERD and OSA is hypothesized to be related to generation of negative intrathoracic pressure during obstructive apneas, which is expected to more easily move stomach contents into the esophagus. Ing et al. found a significant increase in GERD events in patients with OSA but that fewer than half of apneic events were related to acid reflux and 28% of reflux events preceded the breathing event; only 43.8% of arousals were related to reflux events. CPAP reduced reflux events in both those with OSA and in controls without OSA.[126] A large demographic study showed only a slight difference in the prevalence of GERD in those who had OSA versus those without OSA (p=0.064). Although there appears to be an association between the severity of GERD, the severity of OSA did not influence the GERD prevalence.[125] There is also a physiologic compensatory change that actually protects against reflux during breathing events.[127]

Although the incidence of OSA increases with age, there does not appear to be an age-related increase in GERD. The correlation with increased incidence of GERD in patients with OSA may reside in the common risk factors such as obesity, alcohol intake, female sex, hip circumference,[125] daytime sleepiness, race, and family history.[128]

OSA and SB

Historically, SB was associated with occlusal discrepancies and stressful events, but these have not been shown to be causative. More recently, dental sleep medicine practitioners hypothesized a correlation between a breathing event and subsequent SB, as a physiologic reaction to a breathing event, which assists in reestablishment of an open airway.[129] The relationship between SB and SDB has yet to be elucidated.[130]

The gold standard for SB diagnosis is PSG with electromyography (EMG) leads over the major muscles of mastication to identify rhythmic masticatory muscle activity (RMMA). For a dental clinician, obtaining PSG data is not realistic. Without access to sophisticated diagnostic tools, practitioners often use anmnestic patient report or evidence of tooth wear to identify the presence of SB.

Questionnaires alone may not be adequate; in a population of patients who both answered questionnaires about SB positively and subsequently underwent PSG, of the 12.5% of patients who thought they had SB only 7.5% of the population were found to have SB by PSG.[131] Determination of SB by the presence of tooth wear may also present inaccuracies.[132, 130]

A recent study found an increase in evidence of tooth wear as patients' severity of OSA increased, but more severe wear was predominantly found in older patients and male patients, which could be confounding factors.[133] Questionnaires to screen for the risk of OSA, insomnia, or other sleep disorders could be combined with single-channel EMG channel recordings done at home over a series of nights to establish a diagnosis.[134]

Other diagnostic markers are a bed partner's report of witnessed bruxism and a patient's complaint of sore muscles of mastication after waking. Persistence of morning headaches may also indicate a low level of sleep-related breathing events. Although there is often an intersection in the prevalence of OSA and SB, clinicians must understand that the prevalence of OSA increases with age, whereas the incidence of SB declines with age; SB is most common in children (14% to 20%), stabilizes to approximately 8% to 12% in teenagers and adults, and decreases thereafter to 3%.[135] Thus middle aged, at-risk patients have the highest probability of an intersection of both disorders.

There is no single explanation that accounts for the SB mechanism[136] and the association between SB and OSA exhibits a complex relationship with many clinical commonalities such as an alteration in muscle tone, obesity, sex, race and BMI.[137] Currently, causality of SB with OSA has not been established and interindividual relationships may explain the variable temporal findings between the two entities.[138]

GERD and SB

Nocturnal GERD is often associated with nocturnal bruxism. It is unclear if these are intersecting diseases as are OSA and GERD, or if there is a stronger relationship. One study evaluated the reaction of normal subjects without SDB, SB, or GERD when either a weak acid solution or saline was instilled into the esophagus just

above the lower esophageal sphincter. After infusion of the acidic solution, there was an increase in RMMA episodes, tooth-grinding noises, and swallowing compared to saline infusion. An increase in intraesophageal pH was observed after swallowing that occurred in conjunction with RMMA episodes.[139] Looking at patients who present with GERD and SB, pharmacologic therapy with a proton pump inhibitor did decrease EMG bursts, RMMA episodes, and grinding noises, but did not have a significant effect on swallowing episodes or sleep variables.[140] Studies that actually measure GERD severity, SB, and the presence of OSA are important because patients often report the presence of a disorder that may be historically or incorrectly identified. Clinicians should keep in mind that the SB-OSA relationship is complex, and that inter-individual differences may explain the possible different SB-OSA relationships.[138]

There do appear to be common features in adult patients with SB and SDB related to sleep position, oropharyngeal muscle activity, sleep arousal, headache, and GERD. They often have common risk factors including obesity, age, sex, alcohol consumption, and smoking. These three disorders often coexist with intersecting prevalence across the lifespan and clinical features that influence their clinical presentation; however, a causality among these findings cannot be assumed.[141]

CONCLUSIONS

Dentists can play an important role in screening for and treatment of OSA and are expected to play a much more important role in years to come. Screening for OSA in the dental setting should include those patients who are, a priori, at high risk for OSA and should include validated sleep questionnaires and review of medical data. Treatment is expected to include multiple options because as many as half of the patients who receive an OA will be inadequately treated, and 37% may discontinue therapy within the first year.[142]

Currently, there is little research to show that imaging available in the dental office will accurately identify patients with OSA or predict outcome of OAT, but screening of high-risk populations with questionnaires and medical history review is warranted. OAT is only one of many therapies for OSA and must be seen as part of a much larger treatment scheme.

Although OAT has been shown to improve health outcomes as well as CPAP related to high adherence to treatment, dental side effects steadily continue with OA use. Titration of the OA should be minimized to the smallest advancement required to adequately control the SDB, balancing improved health outcomes with possible occlusal changes. Though HSAT is often used to determine the optimal mandibular position, current technology is inaccurate in determining residual disease in those with a low arousal threshold and mild OSA. Optimizing therapeutic outcomes with OAT encompasses more than sleep parameters and may in the future include as-yet-undefined criteria.

Orthodontic therapy for patients with OSA with craniofacial abnormalities such as transverse deficiency can have a positive effect on sleep parameters; this cannot be extrapolated to patients without such abnormalities.

SDB is a very complex disease with multiple factors that interact to create a disease state. Knowledge of the literature is important to understand the difference between comorbid diseases that have strong clinical commonality but not a causal relationship.

Future research may again change the previous parameters and improve the diagnosis and management of patients with OSA.

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ABBREVIATIONS

AASM – American Academy of Sleep Medicine
ADA – American Dental Association
AHI – apnea-hypopnea index
AR – acoustic reflection
CBCT – cone beam computed tomography
CPAP – continuous positive airway pressure
EDS – excessive daytime sleepiness
EMG – electromyography
GERD – gastroesophageal reflux disease
HSAT – home sleep apnea test
MRI – magnetic resonance imaging
OA – oral appliance
OAT – oral appliance therapy
OSA – obstructive sleep apnea
RME – rapid maxillary expansion
RMMA – rhythmic masticatory muscle activity
SB – sleep bruxism
SDB – sleep-disordered breathing
UA – upper airway

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