Sleep Apnea:
What Every Orthodontist Needs to Know

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6 Important Airway Issues

1. Current Sleep Apnea Protocols Manage Rather than Prevent

Sleep Dentistry - study of a mandibular advancement appliance's impact on the airway

Sleep Prosthodontics - study of the airway's impact on the stomatognathic system

AAP guidelines omit orthodontics
Focus should be the 4 year old; AOO guidelines omit the child

2. Problem is Bigger than Apnea

Sleep Apnea Facts - see Terminology Handout for definitions

Children OSA 2-3%, increase with obesity, UARS most common

Men OSA middle age 22%, elderly 42% - increase weight and lost muscle tone

Women OSA levels low until after menopause - progesterone key

UARS - <5 AHI, >5 RERA; young fit females, children - impact from SNS activity

3. Airway must be Managed Awake and Asleep

Airway Prosthodontics - must maintain the airway 24/7 including during eating

Airway Occlusion - chewing patterns loose harmony when stressed to breath

Nocturnal - wear

Diurnal bruxism and function - wear, fracture
4. Age and Sex Creates Different Issues and Significance

**Growth Phases** (Rouse, JS Inside Dentistry 2013;7:60-80)

**Early**- birth to 4 (tonsil and adenoid hypertrophy)
SDB due to premature birth and incomplete development or low tongue tone and weak orofacial hypotonia.

**Late**- 4-12, tonsils and adenoid growth created airway obstruction, adenoids are major source of abnormal breathing (mouthbreathing), tonsils lesser role

**Mixed**- early growth cluster that is further impacted when lymphoid tissue increases

**Young, fit females**- UARS, sleep onset insomnia, sleep maintenance insomnia, TMD/myofascial pain

**Old, fat men**- Bruxism TRAID- apnea, bruxism, reflux

5. Impact of Sleep Disordered Breathing on Children

**Impact**-

**Craniofacial**- adenoid enlargement increases risk of mouth breathing, tonsils additive. Point of airway obstruction dictates adaptation which can alter craniofacial growth

**Systemic**- cardiovascular morbidity and endothelial dysfunction in children. Obesity magnifies impact

**Neurocognitive**- (Bonuck, K, Freeman, K Chervin, R, Xu, L. Pediatrics 2012)
Increased significant effect in children with significant SDB, Symptoms at 6-18 months= 40-50% increased risk at 7 years, "Worst case" SDB until 2.5 yr then resolve predictive of hyperactivity and conduct/peer issues, "Late" no problem until 4 yo= hyperactivity and anxiety/depression. Altered ENT and Pediatrician protocols for snoring children. Damage is not reversible.
6. Interdisciplinary "Multilevel" Treatment Key

Treatment-Varies based on Age and Cooperation (See RSL Treatment Algorithm)

Main treatments in children are T&A and Orthodontics. Key is reducing AHI<1 and getting the child to close their mouth and become a nasal breather. May require re-training of old habits

Typical referrals:
1. Sleep MD and sleep laboratory- must be focused on children. Facility, monitoring and reading study different than with adults
2. ENT- dedicated to pediatric airway issues, additional training in sleep issues may prove valuable
3. Orthodontist- must be willing to intervene in early craniofacial issues, understanding of airway-based care

Timing of Care- begin process the moment the problem is discovered
Terminology

1. **Snoring**: sound typically created from the vibration of the soft palate due to a narrowing of the airway between the tongue and soft palate.

2. **Upper Airway Resistance Syndrome (UARS)**: repetitive increases in resistance to airflow in the upper airway leading to RERAs and daytime fatigue. SO2 levels remain normal.

3. **Respiratory Effort Related Arousal (RERA)**: arousal caused by respiratory effort not including apnea or hypopnea.

4. **Apnea**: breathing stops for at least 10 seconds during sleep or a 4% drop in blood oxygen saturation. **Hypopnea**: partial airway blockage or decrease in breathing; reduction in airflow 50% or greater, 3% or greater SO2 drop.

5. **Apnea-Hypopnea Index (AHI)**: average number of apneas and hypopneas per hour of sleep; mild 5-15, moderate 15-30, severe >30.

6. **Respiratory Disturbance Index (RDI)**: measure of severity of sleep apnea, including number of sleep disruptions and desaturations. Counts the number of arousals caused by respiratory effort (RERA).

7. **Polysomnography (PSG)**: A test that records multiple physiological variables during sleep (including brain waves, electrical activity of muscles, eye movement, breathing rate, blood pressure, blood oxygen saturation, and heart rhythm). The test is usually conducted in a sleep lab and involves direct observation of the person during sleep.

8. **Pulse Oximetry**: a non-invasive method allowing the monitoring of the oxygenation of a patient's hemoglobin and pulse rate.

9. **Continuous Positive Airway Pressure (CPAP)**: A machine supplies positive air pressure to inflate the airway like a balloon which eliminates blockages and prevents the collapse of the upper airway during sleep. Air pressure is delivered through a hose to a mask that fits over the nose, or both nose and mouth.

10. **Mandibular Advancement Appliance (MAA), Mandibular Advancement Device (MAD)**: oral appliance used to protrude the mandible creating a tightening of the oral airway and advancement of the tongue in an effort to open the airway.
Airway Algorithm

Private Practice Patient
Signs and Symptoms (Fatigue vs. Sleepiness)
Refer to Medical Specialist or Pulse Oximetry Screening

YES

- AHI Low, Pulse Rate Variability
- UARS Signs/Symptoms

FATIGUE
- TMD
  - Splint Design
  - Provisional Appliance
  - Ortho, Orthognathics,
  - Oral Myologist
  - Eval Results with HRPO
  - ENT Consult-Nasal

SLEEPY
- High Berlin
  - ESS, Stop Bang Screening Exam

CHILD
- Snore
  - Habitual Mouth Breather
  - Witnessed Apnea

CONFIRMED DIAGNOSIS
- PSG, HST, PULSE OX

MAINTAIN/IMPROVE

- UARS
- MILD OSA
- MODERATE OSA
- SEVERE OSA

RESOLVE

- ENT-Surgery
  - Weight Loss
  - Orthognathic Surgery
  - Weight - Diet/Bariatric
  - Genioglossal Stimulation
  - Tracheotomy

NO

1. Re-test 2-5 years, Weight or Medical Change
2. Re-test Immediately - Results Did Not Match Expectations

NOT CONFIRM
The Bruxism Triad
Sleep bruxism, sleep disturbance, and sleep-related GERD.
By Jeffrey S. Rouse, DDS

ABSTRACT
Sleep bruxers are a difficult subset of patients to manage predictably. They damage teeth and restorations at a higher rate than normal stress-related bruxers. The adverse effect of their sleep bruxism does not stop just with tooth damage. These patients are more prone to sleep disturbances including apnea and gastric reflux symptoms. It appears that these three sleep issues are interwoven in a triad of factors that create a uniquely detrimental environment for teeth.

The damage from bruxism is a reality in the everyday practice of dentistry and yet there is a great deal of confusion and controversy. Dental professionals do not even agree on the relative number of people who brux. The estimates range from 5% to 95% of the population.2-4 Many dentists focus on the patients who present with wear to determine rates of bruxism. Tooth wear, however, is a poor indicator of bruxism since attrition may play only a small role in tooth destruction and may not be indicative of an ongoing problem.2 The smaller estimates limit the patients to sleep bruxers. Sleep bruxism (SB) is the grinding or clenching of the teeth during sleep, usually associated with sleep arousals.7 To date, the pathophysiology for SB has not been definitively determined. Research points to neurochemistry, autonomic system, and sleep arousals as possible triggers.2 This article will restrict its focus to a unique subset of the bruxing population: the sleep bruxer. This group is extremely destructive to their teeth and systemic health. Additionally, this article will discuss the significance of the bruxism triad: sleep bruxism, sleep disturbance, and sleep-related gastroesophageal reflux.

Tooth Wear
Tooth wear is described in the literature as the loss of the constitution of the tooth and has been classified as being caused by attrition, abrasion, erosion, or a combination of these factors.6 As it relates to SB, tooth wear is reported to additionally cause tooth mobility, temperature hypersensitivity, and tooth fracture.6 While sleep bruxers and non-bruxers displayed significantly different amounts of wear over time, the contribution of tooth-on-tooth attrition to this wear is still controversial.8 It has been postulated that much of the wear could be erosion rather than attrition. Interestingly, those two factors are interwoven in the bruxism triad patient, magnifying the wear in this patient population (Figure 1).

The study of tooth wear is appropriately described as tribology: the science of interacting surfaces in relative motion and associated issues of lubrication, friction, and wear. Teeth sliding over each other are affected by a complex tribological interaction. Friction is encountered whenever there is relative motion between contacting surfaces, and it always opposes the motion. As no surface is perfectly smooth, when the teeth make contact, even under light load, it may produce the loss of tooth structure. When non-roughened surfaces contact, their coefficient of friction decreases dramatically if a lubricant is introduced. Tribology would suggest that a decrease in oral lubrication coupled with tooth-on-tooth contact would introduce friction, thus, wear.12 In addition, elements that increase the surface roughness (eg, erosion) would unavoidably increase wear (Figure 2).

Intraoral lubrication is provided primarily by saliva. It also lubricates and buffers the esophagus and decreases the risk of aspiration.12 Salivary flow follows a daily circadian variation and is significantly lower at night. During sleep, deglutination is episodic, with long periods without swallowing. Daytime swallowing averages 25 to 60 times per hour and only two to nine times at night.13 Salivary flow and buffering capacity vary between individuals and may be insufficient to prevent frictional tooth damage.13 Slowness is almost exclusively associated with microarousals (MA) during light sleep.12 These MAs play an important role in sleep bruxism.

Reduced lubrication, erosive friction, and contact time play significant roles in tribologic wear of teeth in sleep bruxers. Bruxing force is not as important as previously thought. For years the profession has accepted that SB patients can exert up to six times as much force on their teeth at night than normal subjects.35 It has provided dentists a simple explanation to our patients as to why their teeth wear and restorations break. Interestingly, the facts simply do not support that conclusion. Gibbs et al wrote that the bite strength in some bruxer-clenchers can be as much as six times that of the non-bruxer.36 The study evaluated daytime bite strength, not nocturnal bruxing force. It also did not provide groups for bruxing subjects and a non-bruxing control. In fact, the conclusion of the study should have read that only one patient in the study during the day could produce six times greater force on biting than the average dentate patient. Studies monitoring muscle activity during sleep have confirmed that the elevator muscles are rarely, if ever, contracted to their maximum. In fact, when the EMG levels are evaluated during nocturnal bruxing activity, the muscle response is only half of the maximum voluntary contraction.36,37 Only 66% of the bruxism episodes are at a force level equal to or greater than the force generated during chewing. While it is true that patients with greater jaw muscle size may generate more total force with the same EMG activity, the amount of force is well within daily norms and definitely not six times greater.

Sleep Stages and Arousal Response
Good-quality sleep is important for physical recovery, biochemical refreshment, memory consolidation, and emotional regulation.16 A typical sleep cycle is 90 to 110 minutes of sleep with three to five cycles per night. Sleep is
composed of two distinct states: non-REM (quiet sleep) and REM (active sleep). There are four stages of non-REM sleep. Stages 1-2 are light sleep and Stages 3-4 are deep sleep. In the first third of total sleep, non-REM Stage 3-4 is the longest stage and decreases or disappears in the last third. REM sleep increases in the last third. While dreams may occur in any stage of sleep, they are more common and more vivid in REM sleep.20

A micro-arousal (MA) is a shift in sleep occurring during deeper sleep. These 3- to 10-second rises in EEG activity create an increase in heart rate and muscle tone. MAs occur 8 to 15 times in an hour. Bruxism is an oromotor manifestation secondary to MA. Eighty-six percent of bruxism occurs in non-REM Stage 2 sleep and MA.21 A smaller percentage of bruxism events occur in REM sleep.20

More destructive bruxers, however, have a greater number of episodes and time of bruxism in REM sleep than controls.21

Sleep Disturbances

Respiratory disturbances during sleep fall into three categories: snoring, upper airway resistance syndrome (UARS), and sleep apnea-hypopnea syndrome. Snoring is defined as sounds produced in the upper airway from soft tissue vibration induced by air turbulence. It is reported in 40% of men and 24% of women. The incidence is 10% in children.22 Snoring is a risk factor for obstructive sleep apnea (OSA). A medical consultation is appropriate before making an oral appliance for snoring and is mandatory when snoring is accompanied by daytime sleepiness, insomnia, sleep disruption, or hypertension.

UARS is an increased inspiratory effort creating increased MAs and narrowing of the pharynx without oxygen desaturation below 4%.23 It is characterized by repetitive partial collapse of the upper airway during sleep. UARS have been linked with increased bruxism, headaches, and TMD, and are thought to be an intermediate form of disorder between snoring and OSA.20

Apnea is the repetitive absence of ventilation with cessation of breathing for 10 seconds and oxygen desaturation exceeding 4%. Sleep apnea may be obstructive sleep apnea (most common) resulting from a blockage of the upper airway or central sleep apnea demonstrated by no chest movements resulting from a lack of neural drive.24 Patients may have both types simultaneously. Hypopnea is a decrease in airflow of more than 50% or a decrease of airflow by 30% with an oxygen desaturation of more than 3%. The level of OSA is related to the number of apnea-hypopnea events per hour of sleep. The apnea-hypopnea index (AHI) considers persons with 5 to 15 events per hour of sleep as mild, 15 to 30 as moderate, and > 30 as severe. The severity of sleep apnea is judged by a composite of the symptoms including AHI, daytime sleepiness, and % desaturation. Risk factors for OSA include being male, overweight, over 40, large neck size, large tonsils, and family history (Figure 3).24 It is estimated that 1 in 5 adults has at least mild obstructive sleep apnea (OSA) and 1 in 15 has at least moderate.25 Unlike the bruxism prevalence, OSA increases with age and can affect 70% of men and 56% of women over the age of 65, a three-fold increase from middle age.26 While beyond the scope of this article, OSA is a risk factor for hypertension, cardiovascular morbidity, and daytime sleepiness to name just a few.23

Sleep Bruxism

A sleep bruxer is different than a patient who occasionally bruxes during sleep. By definition, a sleep bruxer must have > 4 episodes of bruxing per hour of sleep, > 25 bruxing bursts per hour, and at least one episode per night must make noise.27 SB is higher in children and decreases with age. SB occurs in up to 30% of children from 3 to 6 years old, 13% in respondents 18 to 29 years of age, and decreasing to 3% in patients over 60 years old.28 Unlike stress-triggered bruxing subjects, sleep bruxing episodes are unrelated to experienced and anticipated stress.29 In addition, SB has little variability in the bruxing episodes and bursts per hour of sleep over months and years.29 In moderate to severe SB, grinding was present every night.30

Sleep bruxism episodes are related to disturbances in sleep. Kato and colleagues induced MAs during the sleep of sleep bruxers and controls.22 Tooth grinding followed the experimentally induced MA in more than 71% of the trials. Interestingly, this reaction was only produced in sleep bruxism patients and never in controls, indicating a heightened responsiveness to sleep arousals. Therefore, anything that induces a greater number of MAs has the ability to increase the amount of tooth grinding in these subjects.

Airway

Researchers observe that the frequency of sleep apnea increased as the frequency of bruxism increased. Given the link between MA and SB, it may be more correctly stated that the frequency of SB

**FIG. 1**

**FIG. 2**

**CLINICAL EXAMPLES (1.)** Classic presentation of the bruxism triad. Lateral wear pattern, generalized buccal tooth loss from erosion and abrasion, and history of sleep disruption. (2.) Asymmetric tooth wear in a bruxism triad patient as a result of friction from bruxing, poor salivary lubrication as a byproduct of medication, and roughened surfaces created as a result of erosive reflux. (3.) In addition to the traditional sleep apnea risk factors (over 40 years old, male, overweight, >17 inch neck size), practitioners should add the tooth wear and erosion components of the bruxism triad.
Bruxism is greatest in 5- to 6-year-olds and slowly declines with age. Adenoid tonsil hypertrophy in the 5- to 6-year-old patient may account for the airway obstruction and, thus, a greater incidence of bruxism. As the airway improves with age, the bruxism decreases in the general population but in the triad group it continues. If bruxism is a reflexive mechanism to improve or protect an airway, then greater bruxism could lower the apnea. Sjöholm concluded that there is limited correlation between bruxing and apnea because mild apnea patients had more bruxing events than moderate apnea patients. However, if bruxism is a protective reflex, clinicians might be able to predict the possibility in a young patient population that bruxism would be linked to less severe apnea. Aggressive bruxism in 20- to 40-year-old subjects may simply be an attempt to open their airway. As the sleep bruxer ages, their neurochemical ability to brux decreases, or it cannot overcome the additional airway obstructions due to a loss of muscle tone, weight gain, etc.

GERD
Gastroesophageal reflux disorder (GERD) is a medical condition where the stomach contents leak to the esophagus. It affects approximately 40% of Americans. GERD is commonly associated with heartburn or indigestion, although > 50% of people complaining of frequently clearing their throat, hoarseness, or trouble swallowing were found to have silent GERD. Poor quality of sleep may be the sole presentation of silent GERD in asymptomatic subjects. Medical consultation is recommended. Differential diagnosis would include peptic ulcer, angina, and Barrett esophagus (possible precursor to adenocarcinoma).

Some of the acid content of the stomach may reach the oral cavity. This is an extremely destructive acid with a pH of 1 to 2. In comparison, dietary acids are greater than pH 3. The most common site for damage is the palatal surface of the maxillary molars (Figure 5 and Figure 6). Reflux symptoms present mostly in a supine position. The dorsum of the tongue pushes the acid to the maxillary molar palatal surface when swallowing to buffer the acid. While the palatal surface is the most common site of destruction, the pattern of damage will be dictated by the sleep position of the patient during the episodes. Tongue activity associated with airway patency coupled with regurgitation may also create wear on the lingual surfaces of teeth resembling bulimia but are not limited to the maxillary anteriors (Figure 7). Gastroesophageal reflux disorder (GERD) is a medical condition where the stomach contents leak to the esophagus. It affects approximately 40% of Americans. GERD is commonly associated with heartburn or indigestion, although > 50% of people complaining of frequently clearing their throat, hoarseness, or trouble swallowing were found to have silent GERD. Poor quality of sleep may be the sole presentation of silent GERD in asymptomatic subjects. Medical consultation is recommended. Differential diagnosis would include peptic ulcer, angina, and Barrett esophagus (possible precursor to adenocarcinoma).

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grinding, airway-associated sleep disorders, and sleep-related GERD (Figure 8 and Figure 9). While a causal relationship has not been established, significant correlation makes it important for dentists to evaluate their patient population.

Sleep bruxism is concomitant with sleep apnea. Therefore, if the level of apnea can be artificially reduced, a resultant decrease in bruxism would be anticipated. Olsenberg and Arons found that during continuous positive airway pressure (CPAP) treatment, apneas were eliminated and only a few hypopneas were seen. A complete disappearance of all bruxism events occurred. Mandibular advancement appliances (MAA) have reported to reduce bruxism events 50% to 83%. The variability appears to be related to the appliances’ ability to reduce desaturation episodes, which is more inconsistent than CPAP therapy. The MAA may also be used in SB patients without respiratory disturbances. A statistically significant reduction (39% and 47%) of SB episodes per hour was recorded with the MAA at protrusion of 25% and 75%, respectively, providing further evidence that improving airway patency is an important treatment strategy for bruxism even in a normal patient population. One important note is that traditional occlusal stabilizing splints may have a deleterious effect on SB-OSA patients. A prospective study of 10 mild to moderate OSA patients was conducted with multiple nights recorded with and without a stabilizing splint. Results indicated that six of the 10 subjects had a significant increase in their AHI with splint therapy. Further, OSA patients with complete dentures have significantly improved airways when sleeping with their dentures than without them. Both studies highlight that OSA patients have an improved airway when the mandible is placed and held in a protruded position. Efforts which prevent that ability may worsen the airway and increase sleep bruxism. This could account for the severity of wear found on some occlusal splints. The author has noted that patients with airway and GERD issues have notably more wear on their orthosis regardless of design (Figure 10 and 11).

GERD can be linked to the other members of the triad. Patients with GERD had higher AHI scores and shorter periods in restful Stage 2 sleep. Research has also demonstrated that more severe OSA was accompanied by more severe GERD. While the link is still controversial, one explanation is that during bruxism episodes, there is an increase in negative intrathoracic pressure. This increased negative pressure could cause the gastric acids to be expelled from the stomach and into the esophagus. As the esophageal pH decreases, patients’ bruxing episodes were significantly higher. Miyawaki studied 10 SB and 10 controls presenting without GERD symptoms. Esophageal pH was monitored during an evening of sleep. Results showed that when the esophageal pH reached 4 or lower there was a simultaneous bruxism episode ending in a tonic burst representing a swallow. This is apparently an attempt to buffer the esophageal acid content. Interestingly, the pH 4 threshold was reached exclusively by SB patients. No control patient had an esophageal pH low enough to trigger a bruxism episode.

Proton-pump inhibitors are a group of drugs that reduce gastric-acid production. Administration of a proton-pump inhibitor to GERD-SB patients demonstrated a commensurate 40% reduction of SB episodes. Another family of GERD medication, the H2 blocker anti-acids, showed a reduction in MAs, respiratory disturbances, and daytime somnolence. It did not, however, reduce the AHI. Reducing the intrathoracic pressure with CPAP reduces GERD parameters in patients with and without OSA. Studies with MAA have not been conducted but similar results would be anticipated given their impact on the airway.

Recognizing the Triad Patient

Dentists should to be able to recognize the bruxism triad patient at all stages of life. Early diagnosis can alter poor growth, improve physical well-being, and reduce tooth wear. The following are some of the distinguishing features of each of the stages of the triad.

Childhood 3–12

Adenoid and tonsil hypertrophy produces sleep disturbances including OSA. The magnitude of hypertrophy required for obstruction is smaller in obese children compared to non-obese children. GERD will be found frequently in children with adenotonsil hypertrophy and OSA and should be evaluated. Five-year-old children with OSA will commonly display differences in growth when compared to matched controls. This can include a mandibular posterior inclination, greater anterior face height, and retroclined incisors (Figure 12).

Adolescent 13–19

Dentists commonly tell parents that most children “grow out of bruxism” when they reach puberty. More accurately, many children grow enough to overcome the impact of the adenoids and tonsils, or orthodontic intervention expands the palate enough to create an adequate airway. Any signs of pathologic wear due to attrition or erosion at this stage should elicit questions about sleep disturbances, sleep bruxing, and reflux symptoms (Figure 13).

Young Adult 20–40

Pathologic tooth wear or significant relapse of orthodontic correction is usually addressed by fabrication of an occlusal orthosis. Continued grinding on the appliance is a hallmark for the bruxism triad. Self-reports of high stress may be due to repeated sleep disturbance and should not be disregarded. Evidence of GERD may be present on the teeth or maxillary palatal tissue. Bruxism may be acting as a protective reflex for the airway (Figure 14). There is a statistically significant association between childhood wear of the deciduous mandibular molars and canine teeth and the degree of whole-mouth wear observed 20 years later (Figure 15). For sleep bruxers with episodic sleep disturbances and reflux symptoms, this author has been using an anterior repositioning splint with success. The appliance is fabricated at < 40% of the patient’s maximum protrusion, minimizing the occlusal risk associated with advancement appliances (Figure 16 and Figure 17).

FIG. 13

FIG. 14

FIG. 15

FIG. 16

FIG. 17

EARLY SIGNS AND TREATMENT (12) Adolescent triad: A 16-year-old with pathologic wear on incisors and a loss of tooth texture from erosion and abrasion. Medical history was indicative of the triad. Bruxism triad in a young adult. The pathologic wear, once limited to the anterior teeth, is beginning to appear on the posteriors as guidance is lost. Patient reported sleep issues that became exacerbated with pregnancy. GERD was intermittent until pregnancy then it increased. Patient demonstrates wear on deciduous molars increasing the risk of bruxing as an adult. Another explanation may be the triad. Constricted dental arch, crowded lower anteriors, and a deep bite with a lifetime of airway-related issues. GERD history coupled with erosive wear on teeth. Ramp of the anterior repositioning appliance demonstrating the path of the incisors from centric closure to anterior closure. Patient positioned at 40% of voluntary protrusion.
Bruxism will begin to wane as the risk lessen G reduce sleep bruxism, and potentially erature elucidated, management of the complications of O causes of middle age (Figure 18). As the lit-increased. airway during sleep improves all factors and restoration damage may be ap-

unwilling or unable to use CPAP, MAAs parent. Airway management becomes a unique subset.

also be caused by any disruption of air-
tooth wear. However, sleep bruxers are also a history of GERD treated with a proton-pump inhibitor medication. Patient reported a decrease in bruxism but bed partner noticed an increase in snor-
ing and apnea events. Polysomnography revealed an AH1 of 34. (19) Thornton Adjustable Positioner 3 (TAP). Custom made MAAs for the treatment of severe snorers. Action connecter allows night's treatment for titration of the airway. (20) TAP 3 engaged. (21) Mature patient with the bruxism triad. Lifetime history of bruxism, snoring, intermittent poor sleep, and GERD symptoms. Sleep study results indicate severe apnea. (22) Incisal view demon-strates the damage from sleep bruxism and the erosive wear associated with GERD and tongue position. (23) Retracted tongue position in edentulous patients limits the available airway. Wearing their dentures at night may allow for a more favorable airway. (24) Many dentures worn at night demon-strate the same lateral wear facets indicative of the bruxism triad patient. A complete history of bruxism, sleep, and GERD should be obtained.

Medical issues related to overall health may be linked to OSA. Bruxism may be present but will be limited in its ability to clear the airway and reduce GERD. Patients may need to address tooth wear issues that have been compounded through years of neglect (Figure 21 and Figure 22). Comprehensive evaluation for signs and symptoms of OSA and referral for polysomnographic study is recommended. Patients with dentures should be encouraged to wear their dentures at night if sleep disruption is present (Figure 23 and Figure 24).

Conclusion
It has been demonstrated that a majority of dental patients present with tooth wear. However, sleep bruxers are a unique subset, Sleep bruxism is reflectively triggered by sleep microarousals. These disturbances in the sleep pat-
terns are a natural occurrence but can also be caused by any disruption of air-
way patency or a significant reduction of esophageal pH. The bruxism triad is an attempt to explain the interlocking nature of bruxism, breathing, and ero-
sion. These patients suffer a significant loss of tooth structure and restoration damage due to the increase in friction due to poor lubrication and roughened surfaces. Medical evaluation is key. CPAP, MAA, and GERD medications were proposed as possible treatment options for the bruxism triad patient.

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Continuing education efforts. We are pleased to offer you another avenue for obtaining credits for your Continuing education lessons. In addition to our free course on our web site, www.insidedentistryce.com, we are now offering a mail-in option to our readers who prefer to send in their tests for scoring. For a nominal fee of $28 ($14 per credit) to cover administrative and handling costs, your test will be graded and your certificate will be sent to you in the mail.

Sleep Prosthodontics: A New Vision for Dentistry
Part 1 of this 2-part series focuses on the snoring child
Jeffrey S. Rouse, DDS

ABSTRACT
The purpose of this article is to introduce a new term, sleep prosthodontics, to the dental community and to differentiate airway analytics from sleep dentistry. Sleep dentistry may be thought of as the study of an oral appliance and its impact on the airway. Sleep prosthodontics is the study of airway and its impact on the stomatognathic system. In other words, sleep dentistry addresses the how (ie, how does an appliance assist nocturnal breathing?), whereas sleep prosthodontics addresses the why (ie, why are patients developing malocclusions, creating myofacial pain symptoms, and wearing their dentition?) This article will focus on the impact of sleep disordered breathing on the growing child and the unique role dentistry plays in screening and treating these patients.

Learning Objectives
• Describe the difference between sleep prosthodontics and sleep dentistry.
• Explain the unique impact of sleep fragmentation and obstructive apnea on children.
• Correlate dental abnormalities to airway deficiencies in pediatric patients.

To receive up to 2 credits for this article, log on to www.insidedentistryCE.com to take the quiz.

UARS and OSA: A Comparison
Although many clinicians describe UARS and OSA as the same disease with a slight variance in severity, their pathophysologies appear to be different.4 OSA is characterized by complete upper airway obstructions lasting longer than 10 seconds with an associated 4% oxygen desaturation. It is most commonly attributed to a hypotonia of the soft palate or base of tongue. Partial airway obstructions that lead to desaturation or brief awakenings from sleep are classified as hypopneas. Continued desaturations over time may cause excessive daytime sleepiness and hypertension. They have been correlated to endothelial dysfunction, myocardial infarction, and cerebrovascular accidents. The level of severity of OSA has been associated with an increased mortality.5,6

Anatomic irregularities or minor breathing impairments can create UARS.7 Patients with UARS may have a more collapsible airway because of abnormal inspiratory flow dynamics8 or increased collapsibility on expiration due to atypical anatomy.9 UARS patients have more sensitivity to restricted breathing or negative oropharyngeal pressure. The airway constriction is recognized...
and responded to more quickly, preventing obstruction. These respiratory effort–related arousals (RERAs) and sleep fragmentations lead to activation of the autonomic nervous system—in particular, increased sympathetic nerve activity.10

**Sleep Dentistry**
Continuous positive airway pressure (CPAP) was introduced in 1981.11 CPAP is still the standard of care today for OSA.12 Even with significant improvements in CPAP technology, it is unpopular with patients, rarely worn throughout an entire night,13 and has less than optimal long-term compliance.14

Oral appliances (OA) were introduced in the 1980s in an attempt to provide an alternative to the unpopular CPAP. OAs act by protruding the mandible and attempting to position the tongue out of the oropharyngeal region. OAs are currently divided by their manner of therapy. Tongue-retaining devices utilize negative pressure from a bulb attached to the tip of the tongue to reposition the tongue. Mandibular advancing appliances (MAAs) are attached to the dental arch (Figure 1). The mandible is held in a protruded position. The protrusion is either fixed or titratable.

Although the quest to create an alternative to CPAP is understandable, sleep dentistry has become single-minded in its treatment of adult apnea with an appliance. If dentistry compartmentalizes itself on OA fabrication for OSA, women with UARS and the majority of children are eliminated from the purview of the dental practitioner.

**Sleep Prosthodontics**
Sleep dentistry can be thought of as the study of an OA and its impact on the airway. Sleep prosthodontics is the study of the airway and its impact on the stomatognathic system. The stomatognathic system encompasses the mouth, jaws, and the closely related structures of the oro-pharynx and fauces. Dentists deal with this system during its development and maintain it throughout a lifetime.

Although a physician must make the diagnosis of SDB, the dentist plays a critical “diagnostic” role. Many times the lack of witnessed apneic episodes or the lack of particularly egregious daytime symptoms may lead to a delay in care by the medical community. The impact of a poor airway can many times be detected in the patient’s craniofacial development, oral impairment, and occlusal dysfunction well before the clinical presentation of systemic disease.15

Sleep prosthodontics is not restricted to an appliance, but instead has a single-minded focus on the patient’s health. It also encourages a patient-centered, interdisciplinary solution that includes a wide range of options, including orthodontics, oral mycology, nutrition/diet counseling, orthognathics, CPAP, MAA, and otolaryngologic surgeries.

**The Snoring Child**
In the general population, 2% to 3% of children have apnea.16 That proportion is growing given the increase in childhood obesity.17 Apneas and hypopneas are defined in children as events lasting longer than two missed breaths and most commonly associated with some change in oxygen saturation or end tidal CO2 increase. UARS presents in children during polysomnography (PSG) as an increased respiratory effort with no apnea and little oxygen saturation change (Figure 2). The characteristic signs and symptoms of UARS vary with the age of the child, as will treatment options.18

The prevalence of snoring in children ranges from 10% to 21% from 6 to 81 months. Habitual snoring has been reported in 9% of infants aged 0 to 3 months. In a general pediatric clinic, habitual snoring was documented in 17% of patients, with that rising to 29% of the children reporting for neurologic indications such as headaches and 56% of the children diagnosed with psychiatric disorders (half with anxiety/mood disorders). In a 2-year follow-up on habitually snoring children, 30% of subjects had worsened from baseline. OSA developed more often in boys, especially if adenotonsillar hypertrophy or an increase in waist circumference was present.

Snoring and mouth breathing in children were initially thought of as unreliable markers for OSA and not as potential problems in their own right. More recently, it is believed that snoring independent of OSA may cause neurocognitive dysfunction and impaired daytime performance. Habitually snoring children are at higher risk for social problems, poor academic performance, decreased attention, and anxiety/depression issues. Children who are chronic snorers have abnormal slow-wave sleep patterns and experience more fragmentation. This sleep instability may explain the detrimental effects of non-apneic snoring. Studies of occasionally snoring children who otherwise have normal sleep demonstrated altered brain function and more delayed and effortful processing. These children also experienced more behavioral problems than non-snoring children. Children who snore are not likely to “grow out of it” without experiencing cognitive impairment.
The neurocognitive and behavioral damage from snoring in children appears to be related to the fact that their brains are still developing. A confounding issue is that the impact of the snoring may not be detected for years, even after the snoring has resolved. The genesis of the long-term neurocognitive effect in snoring children may be during a critical developmental period—at or before 3 years of age.

Bonuck and colleagues examined 7 years of epidemiologic data from more than 11,000 children followed from birth. Cognitive and behavioral assessments were conducted when the children reached 4 and 7 years of age. By 4 years old, children who had a history of snoring were 20% to 60% more likely to exhibit behavioral difficulties; by 7 years, they were 40% to 100% more likely. The more severe symptoms were linked to the poorest behavioral outcomes. The “Worst Case” cluster had a peak of SDB symptoms at 30 months that abated. Nonetheless, at 7 years the cluster displayed hyperactivity and conduct and peer difficulties. Inclusion in the “Later SDB Symptom” cluster, with a peak at 42 through 69 months, was predictive of emotional difficulties and hyperactivity at both 4 and 7 years.

Bonuck’s work underscores that the presence of irregular sleep breathing may not be directly linked to the academic and behavioral symptoms. Instead, SDB during periods of brain development is very predictive of later damage. The neurocognitive damage in areas such as academic performance and executive functioning is not reversible, so early identification and treatment are paramount.

Metabolic Consequences
Because of the close link between sleep, the immune system, and inflammation, children with SDB are prone to many of the same systemic inflammatory conditions that as adults lead to high blood pressure, arrhythmias, and congestive heart failure. Sleep disturbances in children lead to aberrant sympathetic nervous activation that creates cardiovascular and metabolic injury. Pediatric apnea is connected with endothelial microvascular dysfunction: a marker of subclinical cardiovascular disease, systemic hypertension, pulmonary hypertension, and myocardial left ventricular remodeling. In the presence of obesity, the metabolic consequences are exacerbated. Treatment of the SDB is mandatory to prevent complications. If the child’s SDB is resolved, the systemic inflammation in non-obese subjects appears to be reversible. The unanswered question is whether the childhood autonomic disturbance promotes metabolic morbidity later in life even after SDB resolution.
**Abnormal Craniofacial Growth**

SDB and abnormal craniofacial development are bidirectional. SDB may create craniofacial changes. These skeletal alterations can further exacerbate the SDB difficulty. Treating the breathing issue as early as possible prevents the continuation or the worsening of the craniofacial problems. It may also lead to an improvement in growth and development if addressed soon enough in the process. Before the age of 4 years, 60% of craniofacial maturity is completed. Approximately 90% is finished by the age of 12 years. Tonsils and adenoids begin hypertrophying at 2.5 years, reaching their greatest dimensions around 5 or 6 years. Early and late growth clusters exist in children with SDB. The early cluster will have SDB without tissue hypertrophy. The late cluster will react to the additional airway blockage.

**Early Growth Cluster**

The pattern for bone growth resides not in the bone itself but rather in the soft tissue and muscle that encase the bone. Oral-facial muscle tone and tongue tonicity create a framework for normal development of the nasomaxillary complex and mandible. SDB is noted in children with pathologic hypotonia of facial and tongue muscles. Children born with a normal palate and oral-facial hypotonia will develop a high, narrow palate over the first year of life (Figure 3 and Figure 4). Children born with a high, narrow palate have hypotonia at birth. These myofunctional changes may be detected in utero.

Premature children suffer from SDB and OSA due to the lack of completion of craniofacial development in utero. They typically have a narrow, hard palate, abnormal nasal resistance, and mouth breathing. These changes promote the development of an abnormally long lower third of the face. Kin and colleagues concluded that if nothing is done in these premature infants, SDB and OSA will develop.

Instinctively, clinicians have concentrated on the mandible when discussing airway dimensions. However, the maxilla appears to be the more important arch in determining upper airway dimensions in OSA patients. The distance from A point (most posterior point in the concavity of the anterior maxilla) to

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**Sleep Prosthodontics and the Snoring Child**

The American Academy of Pediatrics (AAP) guidelines for the diagnosis and management of childhood OSA syndrome call for every child/adolescent to be screened for snoring at each office visit. A PSG should be ordered on children who snore and have neurocognitive, behavioral, or medical issues indicative of OSA. The goal of the guidelines is to screen for more SDB, but there are two diagnostic problems. Although PSG is the gold standard for diagnosis of OSAs, there is a shortage of sleep laboratories with pediatric expertise and equipment. In addition, the worst-case children have no behavioral issues when the snoring is occurring. It is not until years later that the hyperactivity and emotional issues arise, after the snoring has abated. Physicians following the AAP guidelines will not discover the worst SDB children. Dentists and dental hygienists have a unique role in early identification of SDB in children. Beginning sleep prosthodontic indicators of SDB are craniofacial anomalies resulting in malocclusions and sleep-related bruxing.
Porion vertical (vertical line drawn from the most superior part of the external auditory meatus) was the most contributory cephalometric marker for airway patency. Appropriate positioning of the maxilla opens the velopharyngeal and oropharyngeal airways. Additionally, proper maxillary positioning enhances mandibular growth. Thus, the lack of facial muscle activity and ideal tongue tone constrains the premaxilla, producing an abnormal airway dimension and amplifying the threat of SDB.

**Late and Mixed Growth Cluster**

Tonsils and adenoids occupy space, increase airway resistance, and create turbulent nasal airflow. The period of tissue hypertrophy is especially damaging to mixed cluster patients. These children are undiagnosed early growth cluster children who become further compromised due to enlargement of oral and nasal tissues.

Tonsils and adenoids should be judged against the relative size of the airway rather than the absolute size of the lymphoid tissue (Figure 5 and Figure 6). Adenoids are located at the posterior of the nasal cavity on the roof of the nasopharynx (Figure 7). The normal distance from the adenoids to the soft palate for an acceptable airway should be at least 12 mm. For each millimeter decrease, the odds of the child snoring increase 1.61 times. Mouth breathers typically show a smaller upper airway dimension as well.47 The adenoid and tonsillar obstruction creates the trigger, but the deviate facial and neck muscle recruitment and tongue hypotonia cause the maldevelopment.48

The point of obstruction tends to determine the type of skeletal impact. Nasal obstruction from enlarged turbinates, blocked ostium maxillare, deviated septum, or nasal valve stenosis creates Angle occlusions of Class I, II, and III equally (Figure 8). The maxilla in these cases is positioned posteriorly and the mandible is posterior-inferior. The facial type is most commonly dolicocephalic. Blockage of the airway predominately by the adenoids will create growth patterns that yield mostly Class II occlusions and anterior open bite with both jaws located posterior-inferiorly. Facial type is again dolicocephalic with the typical long-thin “adenoidal” face (Figure 9 through Figure 12). If the tonsillar tissue is responsible for the airway obstruction, the tongue will have an abnormal resting posture. Class III occlusions will be more common with the maxilla normal or posterior placed (Figure 13 through 15). The tongue may direct the mandible anteriorly or, because the tongue is not in the roof of the mouth driving A point anterior, the maxilla will become bimaxillary retrusive.50 In some cases, the anterior posture of the tongue will create an open bite. This is incorrectly referred to as a tongue thrust. The impact from a thrust does not alter the tooth position. Long-term, low forces cause tooth movement. The posture of the tongue against or between the anterior teeth due to the excessive tonsillar size creates the open bite (Figure 16 and Figure 17). Facial types in this group are more brachyfacial. Lastly, if the airway is blocked through a combination of factors, the Angle classification will be either Class II or III. The maxilla will be in a normal location and the mandible will be the affected arch (Figure 18). These craniofacial changes are not restricted to OSA; all SDB will create unique alterations depending on the patient.

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**FIG. 8**

**FIG. 9**

**FIG. 10**

**FIG. 11**

**FIG. 12**

**ADENOIDAL OBSTRUCTION (8.)** Exam should include nasal anatomy and function. Inferior nasal turbinates occupy the lateral wall of the nose and are easily visualized. When swollen, as in this 12-year-old child, the available airway is reduced and may lead to altered function. (9) An “adenoidal” face is long, thin, and dolicocephalic (Case courtesy of James Awbrey, DMD). (10) Narrow maxilla with tooth wear and bilateral crossbite. (11) Adenoidal tissue blocking the velopharyngeal airway. (12) Class II Angle classification with worn dentition and anterior open bite.
compensation. Children with UARS have been reported to display high, narrow palates, dolicofacial form, and a Class II malocclusion, indicative of largely adenoidal blockage.21

Dentists identifying craniofacial changes early in development may resolve the malocclusion by simply referring for adenotonsillectomy (T&A). The impact of T&A on the pediatric immune system is controversial. A recent 5-year longitudinal, prospective study demonstrated that adenotonsillectomy does not pose adverse short- or long-term impact on the cellular or humoral immunity.52 Cephalometric changes (eg, posterior incline to the mandible, anterior incline to the maxilla, longer anterior and shorter posterior face height, and upper and lower teeth more retroclined than a normal matched control) were detected in 5-year-old subjects with adenoid-induced OSA.53 T&A resolved the OSA in all subjects. At the 5-year recall, cephalometric evaluation demonstrated that the mandibular plane angle and incisor relationship was similar to the control. Early resolution of the SDB allowed time for the proper use of the oral-facial and tongue muscles. Closed mouth breathing with the tongue in the roof of the mouth directed ideal growth.

In some children, T&A alone may not completely resolve the OSA (Figure 19 and Figure 20). The longer the airway dysfunction, the greater the structural impact on the airway. An interdisciplinary clinical study54 was conducted on children approximately 6.5 years old with inclusion criteria of OSA, large tonsils, visually constricted airway, and high and narrow palate. Group 1 was treated with rapid maxillary expansion (RME) and Group 2 with T&A. Maxillary expansion has been shown to create improved nasal resistance and an increase nasal cavity volume. In cases without excessive lymphoid hypertrophy, RME can resolve significant levels of OSA.55,56 After the original therapy, only one child had been completely resolved (apnea-hypopnea index (AHI) <1). The remaining subjects switched groups and received the opposite treatment. After receiving both treatments, 29 of the 31 children were cured. It can be concluded that many children must be treated with multiple therapies before resolution, especially if the SDB has previously altered the airway to a significant degree.

Sleep Bruxism

Not only are dentists in the best position to detect and intercede in cases of abnormal craniofacial development, but they are also the best judges of aberrant tooth wear. Bruxism occurs in up to 30% of children, often around 5 and 6 years during late cluster adenoid and tonsilar hypertrophy.57 Carlsson and colleagues58 determined in a 20-year prospective study that bruxism in childhood may be a persistent trait. Early tooth wear was predictive of increased tooth wear 20 years later. The results emphasize that the triggering mechanism for sleep bruxism is present as a child and does not develop over time.

For restorative dentists, it is significantly more important to locate what elicits the action than the “genetic code” that produces a bruxer. Historically, popular theories have postulated that the generator for bruxism was stress, neurochemical, or occlusion. PSG-based research has disproven these theories. Stress leads to awake bruxism, not sleep bruxism.59,60 Most chemical irregularities in bruxers are linked to sleep fragmentation.61,62 Finally, bruxism is a centrally, not...
peripherally, mediated event.\textsuperscript{63} Idealizing occlusions may control the impact of bruxism and improve chewing function, but it will not resolve sleep bruxism.\textsuperscript{64}

Bruxism occurs during microarousals from regular sleep patterns.\textsuperscript{64,65} Many factors may introduce microarousals, including reflux and tactile and auditory stimuli.\textsuperscript{66,67} The most common reason for these bruxism-related microarousals appears to be respiratory effort. It is the author’s assertion that sleep bruxism serves a functional role in protecting and improving the airway during episodes of inspiratory flow limitation and obstruction. The activity of increasing genioglossal and infrahyoid muscle tone along with the lateral movement of the mandible dilates the upper airway, raises inspiratory flow, and reduces upper airway resistance.\textsuperscript{68}

Sleep bruxism is classified as a sleep-related movement disorder similar to restless leg syndrome and is routinely referred to as a possible indicator of SDB. The majority of bruxism occurs during light non–rapid eye movement sleep.\textsuperscript{69} With traditional PSG, 80\% or more of the bruxing episodes have related respiratory events.\textsuperscript{68,70} Linking bruxism and airway resistance causally is difficult, given that many abnormal breathing patterns are not necessarily conspicuous on PSG. Sleep apneas are more easily recognized, but the RERAs can be a challenge to identify, especially in children who do not desaturate like adults. Additionally, bruxism minimizes the degree of obstruction and flow restriction.\textsuperscript{71,72} A healthy autonomic nervous system of a bruxing child can fix the airway before it can be detected within the framework of a normal PSG.

Without esophageal pressure monitoring to demonstrate the increase respiratory effort, bruxism activity may not be recognized as being associated with a respiratory event.\textsuperscript{71,72} This RERA-related phenomenon was verified in a study of 50 pediatric subjects with an inclusion criteria of sleep-related tooth wear.\textsuperscript{73} No significant statistical association was found between AHI and the severity of bruxism. However, when respiratory effort–related arousals were added to the AHI, a statistically significant association was found. The bruxing events acted to protect the airway rather than

\textbf{FIG. 18} Tonsils and adenoids impeding breathing. \textbf{(19.)} Five-year-old child with significant attrition. Mother reports a history of sleep bruxism (Case provided by Kathy French, DDS). \textbf{(20.)} Maxillary anterior tooth wear. Adenotonsillectomy (T&A) performed and postoperative apnea-hypopnea index (AHI) was 6.1. Continued therapy recommended for resolution. \textbf{(21.)} Six-year-old male presents nocturnal bruxism, habitual snoring, and behavioral issues. PSG reports an AHI of 9.6/hr and respiratory effort–related arousal (RERA) of 14.2/hr (Case provided by Kathy French, DDS). \textbf{(22.)} Radiograph highlights the obstructed nasal airway preoperatively. Three months after T&A, another PSG demonstrated and AHI of 0.3 and RERA of 0.0/hr.
to resolve an obstruction. Bruxism should rise with UARS, habitual snoring, and occasional snoring, because negative pressure and respiratory effort drives the action. The researchers concluded that pediatric sleep-related tooth wear could be used as a marker for SDB. Currently, no other healthcare provider is more equipped to evaluate and monitor pediatric nocturnal tooth wear than the dental practitioner (Figure 21 and 22).

**Conclusion**

The impact of SDB on the growing, snoring child can be serious, and sleep prosthodontics plays a unique role in screening and treating these patients. Dentists should screen children based on history and pediatric sleep questionnaire, as well as physical, intraoral, airway, and radiographic examination. It is important to note that dentists should not consider treatment in these children without a medical evaluation and possibly a sleep study. Interdisciplinary treatment options must be reviewed within the context of the particular sleep breathing disorder, age of the patient, and level of cooperation of the child and the parents. Given its focus on MAAs, sleep dentistry is limited in treating the snoring child. Sleep prosthodontics screens possible OSA/UARS in children and acts as a conduit of care, placing the patient in the proper medical, orthodontic, orthognathic, nutritionist/dietician, and/or oral mycology care for the best possible results. Dentistry has a bigger role to play in SDB, and sleep prosthodontics encapsulates that role.

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Sleep Prosthodontics: A New Vision for Dentistry

Jeffrey S. Rouse, DDS

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1. Sleep disordered breathing (SDB) encompasses a spectrum of dysfunctional sleep breathing, including:
   A. habitual snoring.
   B. upper airway resistance syndrome (UARS).
   C. sleep apnea.
   D. all of the above.

2. OSA is characterized by complete upper airway obstructions:
   A. lasting longer than 4 seconds with 10% oxygen desaturation.
   B. lasting longer than 10 seconds with 4% oxygen desaturation.
   C. lasting longer than 6 seconds with 6% oxygen desaturation.
   D. lasting less than 15 seconds with 5% oxygen desaturation.

3. According to the author, sleep dentistry can be thought of as the study of:
   A. an oral appliance (OA) and its impact on the airway.
   B. polysomnography (PSG).
   C. the way occlusion affects sleep.
   D. continuous positive air pressure (CPAP).

4. Sleep prosthodontics is the study of:
   A. UARS, but not OSA.
   B. occlusion and centric relation.
   C. the airway and its impact on the stomatognathic system.
   D. SDB in women.

5. In the general population, what percentage of children has apnea?
   A. 2% to 3%
   B. 4% to 5%
   C. 6% to 7%
   D. 8% to 10%

6. Habitually snoring children are at higher risk for:
   A. social problems.
   B. poor academic performance.
   C. decreased attention.
   D. all of the above.

7. Sleep disturbances in children lead to sympathetic nervous activation that creates:
   A. cardiovascular and pulmonary problems.
   B. cardiovascular and metabolic injury.
   C. metabolic and pulmonary developmental delays.
   D. growth problems and social-emotional issues.

8. Beginning sleep prosthodontics indicators of SDB are craniofacial anomalies resulting in:
   A. a low, narrow palate.
   B. congenital edentulism.
   C. malocclusions.
   D. ankyloglossia.

9. The normal distance from the adenoids to the soft palate for an acceptable airway should be:
   A. at least 15 mm.
   B. between 8 and 10 mm.
   C. no more than 10 mm.
   D. at least 12 mm.

10. With traditional PSG, what percentage of bruxing episodes is related to respiratory events?
    A. 80%
    B. 65%
    C. 50%
    D. 35%
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Sleep Prosthodontics: Understanding Myofascial Pain
A new paradigm for examining temporomandibular disorder
Jeffrey S. Rouse, DDS

ABSTRACT
Epistemic uncertainties are those that result from a lack of knowledge. Often resolution is achieved for these types of uncertainties by testing alternative explanations that lie outside current unsatisfactory paradigms. Sleep prosthodontics, the study of the airway and its impact on the stomatognathic system, is a new concept that may help to explain many uncertainties that remain within the field of dentistry in terms of myofascial pain. Dentistry has historically concluded that myofascial discomfort has a mechanical explanation, with the most predominant areas of interest being malocclusion and bruxism. This article presents the airway as an alternative source for problems with myofascial pain.

In dentistry, it is often said, “You cannot diagnose what you cannot see”; in statistics, this concept is referred to as epistemic uncertainty. Epistemic uncertainty results from a lack of knowledge, and can occur when available information is incomplete or imprecisely evaluated. Improving knowledge acquisition is one way to avoid epistemic uncertainties. As part of this process, alternative explanations for a problem can be envisioned and experimental data can be gathered to support or refute them.

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Prevalence and Symptoms
TMDs are a varied group of conditions that may affect the TMJ, the masticatory muscles, or both, and are the leading cause of non-dental pain in the orofacial region. A classic presentation includes muscle or joint pain, joint sounds, and restricted or altered motion. In an attempt to create a standard for comparing findings from different TMD studies, the Research Diagnostic Criteria for Temporomandibular Disorders was established in 1992. Patients with known TMD are grouped by diagnostic criteria: muscle disorders (myofascial pain and myofascial pain with limited opening), disc displacements (with reduction, without reduction with limited opening, and without reduction without limited opening), or arthralgia/arthritis/arthrosis (arthralgia, osteoarthritis, and osteoarthritis). In a systematic review, the prevalence of each group was as follows: muscle disorders, 45.3%; disc displacements, 41.1%; and arthralgia/arthritis/arthrosis, 30.1%. The most common of the subgroups was myofascial pain without limited opening and with more than three sites of pain on palpation.
As expected, prevalence of TMD was found to be lower in the general population, with 10.5% having myofascial pain subtype of TMD (M-TMD). Other studies have showed that the risk for TMDs is significantly elevated among women, especially black women. This article’s focus is limited to M-TMD, given that it is a common subtype of myofascial pain seen in patients presenting to restorative practices.

M-TMD is characterized by a dull, aching pain that worsens with palpation and function. Physical examination reveals hypersensitive regions or “trigger points” of taut skeletal muscle fiber. Cairns identified patients with M-TMD who complained of pain as exhibiting a localized hyperexcitability of the central nervous system when challenged with painful stimuli. The population seeking treatment for M-TMD comprises mostly women (3.8:1 ratio of women to men). The age of those diagnosed with M-TMD has been shown to range from 25 to 40 years, with prevalence decreasing with advancing age. This subtype of TMD has been shown to have variable pain intensity, as well as self-limiting and vacillating characteristics, making it difficult to create a framework for the natural progression of the disorder.

A study was conducted by van Selms and colleagues to determine whether predictive factors for M-TMD pain could be identified over time. Patients were assessed before TMD treatment was initiated and were followed up for 12 months after resolution of pain symptoms. The pain complaints of patients with low baseline somatization scores continued to diminish over the 12-month period. Those with higher somatization scores at baseline had a measured relapse of complaints. It appears that somatization plays a role in the etiology and recurrence of M-TMD. This is interesting, given that many patients with TMD have functional somatic syndromes such as fibromyalgia, irritable bowel syndrome, and interstitial cystitis. These conditions are more associated with a generalized central nervous system hyperexcitability.

Etiologic Theories for Myofascial Pain
Mechanical Theories
The mechanical explanations for myofascial pain that predominate the dental literature include malocclusion and bruxism. A prevailing etiologic theory is the connection between occlusion and TMD. The concept is that if the teeth are not in harmony with a centered condylar position in the fossa and/or if the guidance in function to a maximum intercuspal position has interferences, the muscle of mastication must reposition the mandible to allow proper function. This constant effort will create muscle dysfunction and M-TMD. Treatment options include occlusal adjustment, orthodontics, and orthognathic surgery.

Although 90% of the population does not have harmonious centric relation/maximum intercuspation positioning, and the majority have functional interferences, very few people have TMD. In addition, no systematic review has found evidence to indicate that occlusal adjustment leads to a greater degree of TMD resolution than placebo adjustment. Orthodontic interventions performed to improve occlusal relationships have also proved to not routinely resolve M-TMD and should not be considered a preventive measure or treatment option.

Although there are many malocclusions that do not lead to TMD symptoms, those such as unilateral cross bite, deep bite, increased overjet, and anterior open bite have been correlated with an increased risk of TMD. The underlying mechanism connecting these malocclusions with TMD may be the airway, given the bidirectional nature of the disorder. Many dentofacial physical risk indicators for malocclusion and TMD are also identified as indicators of increased risk for sleep-disordered breathing (Figure 1 through Figure 3). Measures aimed at prevention, reversal, and/or adequate treatment of malocclusion early in development might also help preclude negative health outcomes often associated with sleep–airway issues.

Another common mechanical theory is that force from bruxism is the pathogenic factor in myofascial pain. It seems that some patients also hold this belief. A recent study showed the self-reported rate of nighttime bruxism in patients with M-TMD (55.3%) to be higher than that of the general population (15.2%). However, polysomnography...
data failed to confirm this higher rate, and higher rates of bruxism were actually found in patients with less myofascial pain and in control subjects (Figure 4).

**Biologic Theory: UARS**

A biologic theory that serves as an alternative to the traditional mechanical theories is that hyperresponsive management of a more collapsible airway creates spontaneous pain and hyperalgesia. Within the fields of sleep medicine and sleep dentistry, recent focus has been placed on obstructive sleep apnea (OSA), which is characterized by complete upper airway obstruction lasting longer than 10 seconds with an associated 4% oxygen desaturation. It is most commonly attributed to a hypotonia of the soft palate or base of the tongue. Partial airway obstructions that lead to desaturation or brief awakenings from sleep are classified as hypopneas. Continued desaturation over time may cause excessive daytime sleepiness and medical comorbidities.

Upper airway resistance syndrome (UARS) was first described in the literature in 1993. Although many clinicians describe UARS and OSA as being the same disease with a slight variance in severity, their pathophysiology in fact appear to differ. Anatomic irregularities or minor breathing impairments can cause UARS. Patients with UARS may have a more collapsible airway because of abnormal inspiratory flow dynamics or increased collapsibility on expiration due to atypical anatomy.

Patients with UARS also differ from those with OSA in their responsiveness to the induction of an airway event. In patients with OSA, repetitive closures of the upper airway appear to dull the sensory receptors, causing an absence of activation of the dilator muscles in the airway. Therefore, patients with OSA may exhibit hyperresponsiveness or nonresponsiveness to upper airway collapse. Patients with UARS have more sensitivity to restricted breathing or negative oropharyngeal pressure, however, and airway constriction is recognized and responded to more quickly, preventing obstruction.

These respiratory effort–related arousals (RERAs) and sleep fragmentations lead to activation of the autonomic nervous system, particularly increased sympathetic nerve activity. This, in turn, causes a release of catecholamine (epinephrine and norepinephrine) into the bloodstream, creating a transient increase in pulse rate, blood pressure, and respiration. Catecholamine levels remain abnormally high for 24 hours after the event. This “fight-or-flight” response may be responsible for many of the damaging aspect of UARS.

Polysomnography indicators for UARS include an apnea-hypopnea index score of 5 or less, five or more RERAs, and a minimum oxygen saturation level greater than 92%. Although some symptoms of UARS overlap with those of OSA, there are important distinctions between the two disorders. The typical patient with UARS is not overweight, for example. Women (typically aged 25-50 years) are three times more likely to be affected than men. It appears that women are more hyperresponsive to airway challenges because of the hormones progesterone and, to a slight degree, estrogen. Progesterone is a pharyngeal dilator and respiratory stimulator and also generates tongue muscle tone. Menstrual cycle phases alter hormone levels; one study found that more women complained of fatigue during the follicular phase, whereas there was a higher level of apnea (as measured by the apnea-hypopnea index) in women during the luteal phase. The authors found that hormonal contraceptive use appeared to be associated with a reduced risk of airway dysfunction, improved sleep efficiency, and increased sleep duration. Most likely because of hormonal changes, postmenopausal women are more likely to snore and experience witnessed apneas, gasping, and frequent awakenings. In many cases, UARS develops into OSA in postmenopausal women.

Chronic sleep-onset insomnia and sleep-maintenance insomnia are more common in patients with UARS. In a recent pilot study, 90% of the nocturnal awakenings experienced by patients with chronic insomnia were sleep breathing events, the majority of which were RERAs. All of the awakenings that lasted 5 minutes or longer, a duration that predisposes toward an insomnia episode, were preceded by a sleep-disordered breathing event. Adults with UARS may complain of fatigue rather than sleepiness. Higher incidence of sleepwalking, sleep terrors, myalgia, depression, and anxiety have been seen in patients with this disorder.

![FIG. 4](image_url)

(4.) Higher rates of bruxism are not seen in patients with myofascial pain.
Patients with UARS often present with a functional somatic syndrome misdiagnosis, including migraine/tension headaches, irritable bowel syndrome, chronic fatigue syndrome, TMD, and fibromyalgia. Many young women with sleep-disordered breathing are mistakenly treated with hypnotics, antidepressants, pain medication, attention-deficit/hyperactivity disorder medication, eugeroics, and muscle relaxants.

The Link Between Pain and Sleep-Disordered Breathing

Sleep fragmentation may be culpable in cases of both allodynia, in which a normally inoffensive stimulus causes pain, and hyperalgesia, in which an increased response to a painful stimulus is experienced. Fragmented sleep profiles increase spontaneous pain or sensitize mechano-insensitive nociceptors with catecholamine, contributing to sympathetically maintained pain, and further impair natural pain-control mechanisms that are thought to play a key role in the development, maintenance, and exacerbation of chronic pain. Polysomnography reports on 25 consecutive patients who reported headache or palpable muscle pain revealed that 100% were diagnosed with UARS (J. Metz, personal communication, 2011).

The phase of sleep that is reduced or eliminated by sleep fragmentation may also be important for maintaining homeostasis. Rapid eye movement (REM) sleep is a normal stage of sleep that usually accounts for 20% to 25% of total sleep time. It is considered the lightest level of sleep and is responsible for nondeclarative memory consolidation, anxiety/depression regulation, and pain control. Sleep deprivation, especially deprivation of REM sleep, induces spontaneous pain and hyperalgesia. During REM sleep, the only muscles that maintain normal tone are the diaphragm and those of the eyes. The remaining muscles are hypotonic, including the muscles that protect the airway. This is the first time during the evening that a hyperresponsive patient with UARS cannot adequately protect his or her airway. Many patients wake with a large release of adrenaline and cannot return to sleep.

In this case, a patient may lose the majority of REM sleep for the evening.

Women are more likely to experience sleep-disordered breathing during REM sleep (40.8%) than men (21.0%). As women age, the number of REM sleep disturbances they experience decreases, falling by 26.7% per decade; TMD proclivity data mimic REM sleep disturbance data. Postmenopausal women have been shown to experience more apnea and awakenings than premenopausal women.

Traditional stabilization splint designs are routinely used in restorative dental practices. Despite their reparative advantages, they do not appear to be helpful in cases of sleep-disordered breathing. In fact, many patients with airway difficulties may experience an exacerbation of their condition when using a splint. Future studies are needed, but this may explain why many patients experience unsuccessful treatment or increased bruxing with their use, as well as why many remove the splint during the night.

Conclusion

A lack of knowledge, or the inaccurate interpretation of available data, can create epistemic uncertainty, a state of understanding that invites new approaches and additional research. The objective of this article was to explore sleep-disordered breathing as an alternative explanation for M-TMD, which had not been completely understood and addressed through current models in dentistry.

Data indicate that there is considerable overlap between patients with M-TMD and patients with UARS in terms of both demographics and signs and symptoms. Further research in the area of sleep prosthodontics represents an opportunity for innovative thinking. If dentistry begins to recognize that M-TMD could be a biologic problem rather than, or in addition to, a mechanical one, treatments could be designed to equilibrate to a systemic balance instead of an occlusal one.

References

Sleep Prosthodontics: Understanding Myofascial Pain

Jeffrey S. Rouse, DDS

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Please complete the Answer Form on page 42, including your name and payment information.

1. Sleep prosthodontics is the study of:
   A. oral appliance therapy for the treatment of apnea.
   B. sleep disorders related to malocclusion.
   C. bruxism and its impact on systemic health.
   D. the airway and its impact on the stomatognathic system.

2. The myofascial pain subtype of temporomandibular disorder (M-TMD) comprises what percentage of the general population?
   A. 10.5%
   B. 30%
   C. 45.5%
   D. 75%

3. M-TMD is characterized by:
   A. reduced response of the central nervous system.
   B. dull, aching pain that worsens with palpation and function.
   C. higher prevalence in men.
   D. increased prevalence in older age.

4. The population seeking treatment for M-TMD comprises what ratio of women to men?
   A. 3.8:1
   B. 5:1
   C. 7.2:1
   D. 10:1

5. Which of the following malocclusions have been correlated with an increased risk of TMD?
   A. Unilateral cross bite
   B. Increased overjet
   C. Anterior open bite.
   D. All of the above.

6. Obstructive sleep apnea is characterized by an upper airway obstruction:
   A. lasting longer than 5 seconds with no change in oxygenation.
   B. lasting longer than 10 seconds with 10% oxygen desaturation.
   C. lasting longer than 10 seconds with 4% oxygen desaturation.
   D. lasting longer than 20 seconds with no change in oxygenation.

7. In patients with upper airway resistance syndrome (UARS):
   A. obesity is a recognized cause.
   B. airway constriction is responded to more quickly, preventing obstruction.
   C. there is less sensitivity to negative oropharyngeal pressure.
   D. sensory receptors in the airway are dulled.

8. Women are more hyperresponsive to airway challenges because:
   A. of the hormone progesterone.
   B. of the hormone testosterone.
   C. they tend to have high-stress lifestyles.
   D. of the use of oral contraceptives.

9. As women age, the number of sleep disturbances during rapid eye movement sleep:
   A. remains the same.
   B. decreases by 26.7% per decade.
   C. increases by 26.7% per decade.
   D. increases by 40.8%.

10. Which of the following treatments have been shown to be effective in the treatment of M-TMD?
    A. Continuous positive airway pressure
    B. Neuromuscular onlays
    C. Anterior repositioning splints
    D. All of the above.
To use our mail-in option, please completely fill out the Answer Form and mail it along with your payment of $32 to the address provided below. **NOTE: THIS FORM MUST BE COMPLETELY FILLED OUT AND INCLUDE YOUR NAME AND PAYMENT INFORMATION IN ORDER TO BE PROCESSED AND CREDIT AWARDED.** Your test will be graded and your certificate will be sent to you in the mail; please allow approximately 2 to 3 weeks for processing. Course valid from 11/27/13 to 12/31/16.

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**December 2013**

**Sleep Prosthodontics: Understanding Myofascial Pain**

Please circle your level of agreement with the following statements.

- **(4 = Strongly Agree; 0 = Strongly Disagree)**

**PROGRAM EVALUATION**