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INVITED MEDICAL REVIEW

Interactions between sleep disorders and oral diseases

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Dental sleep medicine is a rapidly growing field that is in close and direct interaction with sleep medicine and comprises many aspects of human health. As a result, dentists who encounter sleep health and sleep disorders may work with clinicians from many other disciplines and specialties. The main sleep and oral health issues that are covered in this review are obstructive sleep apnea, chronic mouth breathing, sleep-related gastroesophageal reflux, and sleep bruxism. In addition, edentulism and its impact on sleep disorders are discussed. Improving sleep quality and sleep characteristics, oral health, and oral function involves both pathophysiology and disease management. The multiple interactions between oral health and sleep underscore the need for an interdisciplinary clinical team to manage oral health-related sleep disorders that are commonly seen in dental practice.

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Introduction

Dental sleep medicine is a rapidly growing field that is in close and direct interaction with sleep medicine and comprises many aspects of human health. As a result, dentists who encounter sleep health and sleep disorders may work with clinicians from many other disciplines and specialties, including oral and maxillofacial surgery, prosthodontics, orthodontics, family and internal medicine, pediatrics, pulmonology, otorhinolaryngology, neurology, psychiatry, psychology, and anesthesiology. Many dental school faculties now emphasize the need to include sleep courses in their curriculum and continuing dental education programs, and increasing numbers of professional sleep dental medicine organizations are providing sleep knowledge transfer activities to community dental practitioners. Dental sleep medicine is a broad topic, and it would exceed the scope of this review to cover all its domains. This review therefore highlights some key aspects of the interactions between oral health and sleep, sleep disorders, and related sleep health management issues that are commonly seen in dental practice.

Sleep and wakefulness are vigilance states of the circadian sleep–wake cycle. In the general population, abnormal sleep adversely affects quality of life, with impacts on general health status (Briones *et al*, 1996), satisfaction with life, mood, and work performance (Ulfberg *et al*, 1996; Foley *et al*, 2004; Ohayon *et al*, 2004; Dijk *et al*, 2010; Roepke and Ancoli-Israel, 2010). Sleep disorders can have a wide range of symptoms, including excessive daytime sleepiness (EDS), fatigue, morning headaches, and impaired cognition and attentiveness (Philip *et al*, 2005; Blackwell *et al*, 2006). These symptoms can impair performance at work and lead to motor vehicle accidents. Conversely, poor health, low quality of life, and low life satisfaction can influence sleep patterns (Guilleminault and Brooks, 2001; Crowley, 2011).

Sleep disorders, oral health, and oral function

Improving sleep quality and sleep characteristics, oral health, and oral function involves both pathophysiology and disease management. Sleep disorders can be influenced by craniofacial morphology and can in turn affect oral health. Thus, modifying the maxillofacial structure and oral function can help in the management of sleep disorders. The main sleep and oral health issues that are covered in this review are obstructive sleep apnea, chronic mouth breathing, and sleep bruxism. In addition, edentulism and its impact on sleep disorders are discussed.

Obstructive sleep apnea

Obstructive sleep apnea often includes complete cessation of respiration (i.e., breathing) due to upper airway obstruction for repeated periods lasting at least 10 s (apneas), periods of reduced respiration (hypopneas) for the same length of time during sleep (Ivanhoe *et al*, 1999; Roepke and Ancoli-Israel, 2010), and periods of less pronounced reduction in airflow that result in arousals from sleep,

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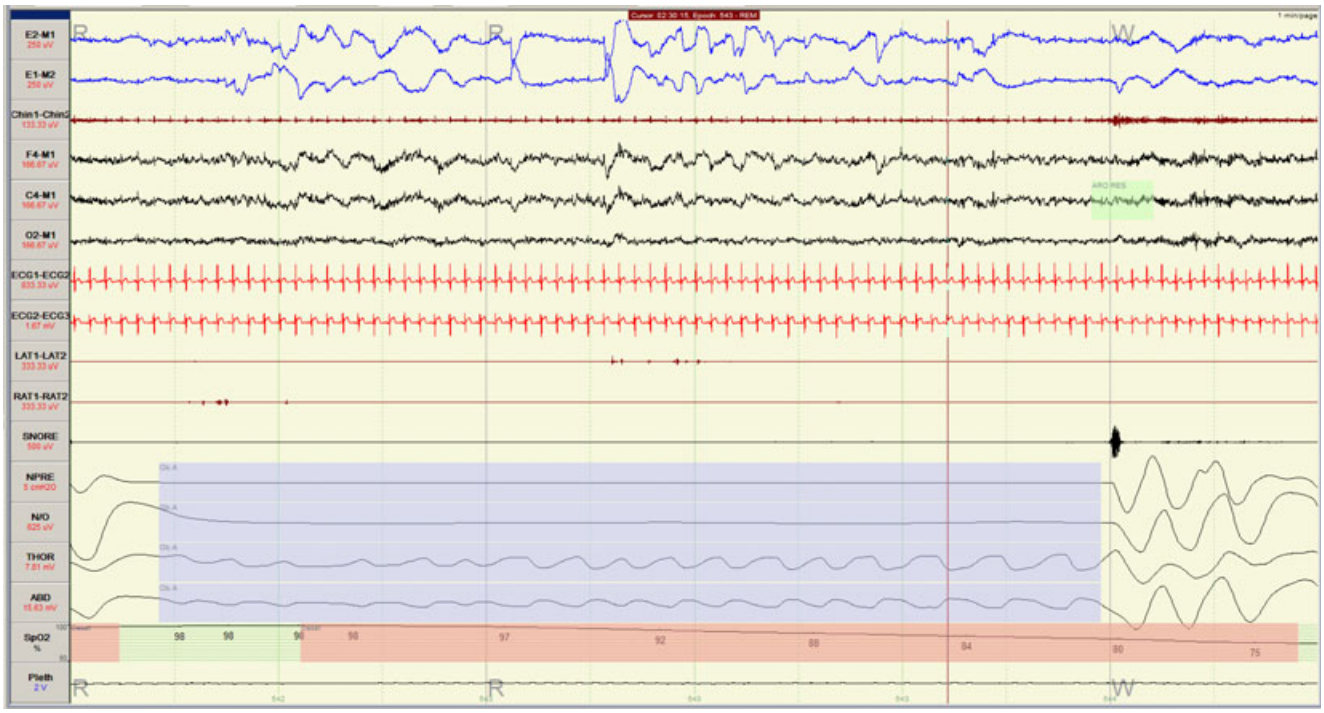

Figure 1 One-min recording from a polysomnogram, showing rapid eye movement sleep and a long obstructive apnea, with absence of nasal airflow despite maintained thoracic and abdominal respiratory effort. E2-M1, E1-M2: right and left electrooculograms (EOG); Chin1-Chin2: chin surface electromyogram (EMG); F4-M1, C4-M1, O2-M1: right frontal, central, and occipital electroencephalograms (EEG); ECG1-ECG2, ECG2-ECG3: electrocardiograms (ECG); LAT1-LAT2, RAT1-RAT2: left and right anterior tibialis surface EMG; SNORE, snoring sound; NRPE, nasal pressure; N/O, nasal/oral airflow (thermocouple); THOR, ABD, thoracic and abdominal excursion (inductance plethysmography); SpO2, oxygen saturation (finger pulse oximetry); Pleth, plethysmogram from oximeter

called respiratory event-related arousals (RERAs). A diagnosis of obstructive sleep apnea is obtained from relevant symptoms along with a sleep study that shows an apnea–hypopnea index (AHI or number of events per hour of sleep) or a respiratory disturbance index (RDI, including RERAs) greater than five respiratory events per hour of sleep (see Figure 1). In adults, the estimated prevalence is at least 2% for women and 4% for men (Young *et al*, 1993). In addition, obstructive sleep apnea is estimated to affect from 1 to 4% of children (Lumeng and Chervin, 2008). With aging, obstructive sleep apnea increases in prevalence to 20–50% among older adults (Ancoli-Israel, 2005). The etiology of obstructive sleep apnea is not completely understood, but likely involves a combination of reduced anatomical cross-section area of the upper airway, changes in upper airway wall compliance, and changes in neurophysiological control of the upper airway musculature. Repeated collapse of the upper airway and choking during sleeping result in repeated arousals and awakenings from sleep, intermittent hypoxia, and multiple downstream effects on the cardiovascular, autonomic, endocrine, and central nervous systems (Guilleminault and Brooks, 2001).

Obstructive sleep apnea is often exacerbated by age, obesity, neurological impairment, abnormal respiratory reflexes, alcohol, and smoking (Crowley, 2011). The consequences can include decreased quality of life, cognitive impairment, and greater risk of nocturia, hypertension, and cardiovascular diseases (Wolkove *et al*, 2007; Neikrug and Ancoli-Israel, 2010). Moreover, obstructive sleep apnea is often accompanied by EDS, most likely caused by both sleep fragmentation and intermittent hypoxia. Sleepiness can impede daily social and work activities. Reduced vigilance, concentration, attention, memory, and health-related quality of life may result at least in part from sleepiness secondary to obstructive sleep apnea (Ancoli-Israel and Ayalon, 2006; Zisberg *et al*, 2010). Obstructive sleep apnea syndrome is one of the most serious sleep disorders in terms of morbidity and mortality (Riley *et al*, 1995).

Obesity, increased neck circumference, male gender, and abnormal facial anatomy are risk factors for obstructive sleep apnea (Bliwise *et al*, 1987; Tishler *et al*, 2003). Malformations of the maxilla, mandible, and associated structures can contribute to upper airway obstruction during sleep. Recently, studies using cephalometry and dental casts of patients with obstructive sleep apnea have shown an association with craniofacial morphological features such as a long and narrow face, transverse facial deficiency (high and narrow palatal arch), and retrognathia, in both adults (Hoekema *et al*, 2003; Johal and Conaghan, 2004; Riha *et al*, 2005; Nuckton *et al*, 2006; Okubo *et al*, 2006; Johal *et al*, 2007; Ishiguro *et al*, 2009; Lee *et al*, 2009; Gulati *et al*, 2010) and children (Marino *et al*, 2009; Pirila-Parkkinen *et al*, 2009, 2010; Tsuda *et al*, 2011; Ikavalko *et al*, 2012). In 6- to 8-year-old children, a recent study suggests that abnormal craniofacial morphology, more than excess body fat, increases the risk of obstructive sleep apnea (Ikavalko *et al*, 2012).

Nightly use of continuous positive airway pressure (CPAP) to physically retain the upper airway open is the

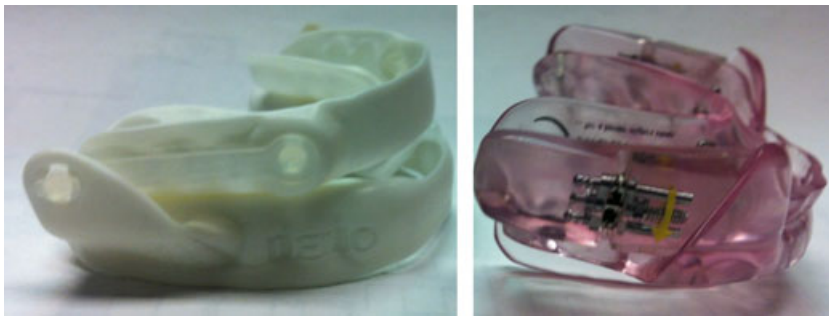

Figure 2 Examples of mandibular advancement devices

usual first-line therapy for obstructive sleep apnea. Although this approach can be highly effective, adherence is often suboptimal. In addition, long-term use of CPAP can have negative consequences for oral health. Patients may complain of dry mouth, or teeth may gradually move (i.e., incisor retroclination). An alternative to CPAP, or occasionally an adjunct therapy, is a mandibular advancement device (see Figure 2). Sleep physicians can prescribe such devices for patients with primary snoring, patients with mild-to-moderate obstructive sleep apnea, patients who have failed CPAP therapy, and patients with contraindications for surgery. However, this therapy is not recommended for patients with temporomandibular joint disorders, inability to sufficiently protrude the mandible (<10 mm), or nasal obstruction, nor for patients with severe or central sleep apnea. Poor oral health, such as dental caries, abscesses, or periodontal diseases, should be managed before starting mandibular advancement therapy.

Device design and material improvements have gradually improved the efficacy of mandibular advancement devices. A recent study in 497 patients with obstructive sleep apnea demonstrated that AHI improved from 30 ± 24.8 to 8.4 ± 11.4 with a mandibular advancement device. A post-treatment AHI < 5 was obtained in 70.3%, 47.6%, and 41.4% of patients with mild, moderate, and severe obstructive sleep apnea, respectively. The study concluded that oral appliances and CPAP were comparable in patients with mild obstructive sleep apnea, but that AHI improvement was superior with CPAP in patients with moderate-to-severe obstructive sleep apnea (Holley *et al*, 2011). A randomized crossover controlled trial that compared CPAP to mandibular advancement therapies in patients with moderate-to-severe obstructive sleep apnea found similar significant health outcomes after 1 month for both therapies (Phillips *et al*, 2013). Although CPAP shows greater efficacy, mandibular advancement devices can lead to higher compliance with therapy (Phillips *et al*, 2013). In one study, both treatments improved subjective and objective EDS, cognitive performance, and quality of life (Gagnadoux *et al*, 2009). However, more than 70% of patients reported that they preferred the oral appliance. In another study, objective compliance measurement allowed calculating the mean disease alleviation (MDA) as the product of objective compliance and therapeutic efficacy. As a measure of overall therapeutic efficacy, MDA was determined as 51.1% (Vanderveken *et al*, 2013).

Management of obstructive sleep apnea sometimes requires surgery and sometimes teamwork between oral and maxillofacial surgeons and otolaryngologists. Many factors can influence the odds of successful surgery, including the patient's age and health, the surgeon's skill and experience, and the type of surgery (i.e., intrapharyngeal or skeletal) (Jacobson and Schendel, 2012). Although surgeries performed on soft tissue do not always resolve obstructive sleep apnea, they can improve symptoms. Maxillomandibular advancement reduces upper airway collapsibility and soft tissue obstruction (see Figure 3). An advancement of 10 mm is associated with a 90–100% success rate (Hochban *et al*, 1997; Prinsell, 1999; Li *et al*, 2000; Riley *et al*, 2000). A recent meta-analysis showed that maxillomandibular advancement has a good success rate for obstructive sleep apnea when performed in adults, but with better results in younger patients (Holty and Guilleminault, 2010). However, following maxillary and mandibular advancement, postoperative recovery and orthodontic treatment lasting for 12–18 months are often required.

Another suggested surgical option for the management of obstructive sleep apnea involves genial advancement, hyoid suspension, and uvulopalatopharyngoplasty. However, a retrospective outcome assessment demonstrated only a 26% cure rate (using a <20 postoperative RDI and a >50% reduction in postoperative RDI as cure parameters). Outcomes of a 10-mm maxillomandibular advancement showed lower success rates (~60%) in patients with BMI > 32 and RDI > 70. In contrast, the cure rate of the subset of subjects with BMI < 32 and RDI < 70 by the same procedure exceeded 90%. Bimaxillary distraction osteogenesis was suggested for the subgroup of patients with very severe obstructive sleep apnea and significant obesity. In 9 patients with an average BMI of 34.33 and RDI > 70 treated with bimaxillary distraction osteogenesis of 25 mm, the postoperative cure rate was 100% (Magliocca and Helman, 2006).

In children with obstructive sleep apnea, a meta-analysis showed a 60–66% success rate with adenotonsillectomy. Thus, the obstructive sleep apnea was unresolved in 34–40% of the children. A multicenter study to assess the efficacy of adenotonsillectomy found complete resolution of obstructive sleep apnea in only 157 of 578 children, or 27.2% (Bhattacharjee *et al*, 2010). Furthermore, long-term obstructive sleep apnea recurrence was reported in adolescents more than 10 years after tonsillectomy and adenoidectomy (Guilleminault *et al*, 1989; Tasker *et al*, 2002).

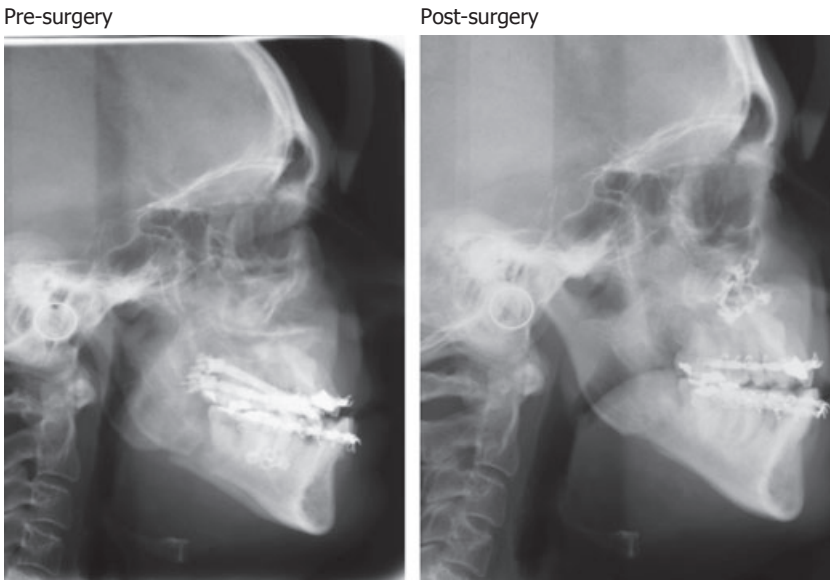

Figure 3 Patient at the beginning and after distraction osteogenesis. Evidence of improve posterior space in the upper airway after bimaxillary distraction osteogenesis

These data underscore the potential significance of craniofacial morphology as a risk factor for pediatric obstructive sleep apnea. Orthodontic treatments such as mandibular advancement and rapid maxillary expansion guide craniofacial growth to the correct occlusal relationship between the maxilla and mandible and help reposition the tongue and improve swallowing (Schutz *et al*, 2011). Some studies that assessed the effects of rapid maxillary expansion on pediatric obstructive sleep apnea showed improvement on sleep studies (Pirelli *et al*, 2004, 2005; Villa *et al*, 2007, 2011; Miano *et al*, 2009; Guilleminault *et al*, 2011; Schutz *et al*, 2011). The only long-term follow-up study, in 10 children, showed that the positive effects of treatment persisted after 2 years (Villa *et al*, 2011). In addition, maxillary expansion can improve nasal breathing by widening the nasal floor and increasing nasal volume (Villa *et al*, 2011). In a study on the effect of a functional mandibular advancement device (orthodontic appliance) on obstructive sleep apnea, half the treated children experienced resolution of their obstructive sleep apnea and the other half had fewer respiratory events during sleep (Villa *et al*, 2002). Cephalometric studies indicated that mandibular advancement with functional appliances widens the posterior upper airway, for a potential positive impact on obstructive sleep apnea in children (Singh *et al*, 2007; Hanggi *et al*, 2008). Another study suggested that some patients may need more than one therapy to resolve childhood obstructive sleep apnea. Patients underwent both rapid maxillary expansion and adenotonsillectomy in random order. After the first treatment (expansion or adenotonsillectomy), symptoms improved but obstructive sleep apnea persisted. The second treatment led to obstructive sleep apnea resolution, regardless of treatment sequence (Guilleminault *et al*, 2011).

Chronic mouth breathing

'Normal' respiration is mainly nasal breathing. Mouth breathing can arise, however, in the setting of nasal obstruction, for example, from hypertrophy of the turbinates, deviated septum, seasonal allergies, chronic rhinitis, or enlarged tonsils and adenoids. Mouth breathing leads to altered muscle recruitment in the nasal and oral cavities. This can impact craniofacial growth in a developing child (Darendeliler *et al*, 2009). In an experiment in which nasal breathing was chronically obstructed to force mouth breathing, young primates developed a transverse deficiency and increased height of the last third of the face (Harvold *et al*, 1972). The topic of mouth breathing is covered here because many of the anatomic consequences of chronic mouth breathing are the same that are risk factors for sleep-disordered breathing. Clinicians who specialize in oral health should be aware that chronic mouth breathing may promote concurrent as well as future risk for sleep-disordered breathing.

Predominant mouth breathing is associated with altered craniofacial growth (Linder-Aronson, 1970), following the absence of active nasal breathing (Schlenker *et al*, 2000) and the change in associated posture (Josell, 1995). In children, mouth breathing is associated with hyperextension of the head, mandibular retrognathia, increased height of the last third of the face, lower position of the hyoid, and antero-inferior position of the tongue (Woodside *et al*, 1991). A lower mandible position, which lowers and pushes the tongue forward, causes decreased orofacial muscle tone (Valera *et al*, 2003; Sousa *et al*, 2005). The influences on craniofacial skeletal growth include high arch palate, long and narrow face, increased overjet, anterior open bite, and Class II malocclusion (retrognathia). Evaluation of craniofacial growth in children with nasal obstruction due to a deviated septum showed retrognathia of the maxilla and mandible compared with control subjects (D'Ascanio *et al*, 2010). Of the children with nasal obstruction, 66.3% had Class II skeletal malocclusion (mandibular retrognathia), 24.5% Class III (mandibular prognathism), and only 9.2% normal occlusion (Class I) (D'Ascanio *et al*, 2010). In contrast, only 17.3% and

4.1% of controls had Class II and Class III skeletal malocclusion, respectively, and the vast majority (78.6%) had normal occlusion (Class I) (D'Ascanio *et al*, 2010).

Nasal breathing can be re-established by various surgical or orthodontic treatments, depending on the sites of reduced upper airway volume. A follow-up study 5 years postadenotonsillectomy suggested slight improvement in the angle of the mandible, high arch palate, and a transition to nasal breathing (Linder-Aronson, 1970). Another 5-year follow-up study post-tonsillectomy indicated that mandible growth was re-established during the 5 years after surgery (Woodside *et al*, 1991). Orthodontic rapid maxillary expansion can improve nasal breathing by reducing nasal resistance and increasing nasal volume (Oliveira De Felippe *et al*, 2008; Gorgulu *et al*, 2011).

Sleep-related gastroesophageal reflux

Nighttime heartburn, or gastroesophageal reflux, commonly occurs with obstructive sleep apnea. Approximately 60% of the adult population report symptoms of gastroesophageal reflux at least once a year and up to 20% report them once a week (Locke *et al*, 1997). Some studies found that gastroesophageal reflux specifically night is also commonly reported in adults and is associated with a particularly severe form of the disorder (Farup *et al*, 2001; Shaker *et al*, 2003). Potential mechanisms of gastroesophageal reflux include low esophageal sphincter pressure (<10 mmHg), which allows reflux in reaction to negative pressure in the esophagus and positive pressure in the gastric lumen. Reflux may also be caused by intragastric stress, which can elevate the intragastric pressure to exceed the lower esophageal sphincter pressure. Nighttime heartburn occurs mainly in sleep stage 2 of non-REM sleep and less in REM sleep or deep sleep stages of non-REM (Dent *et al*, 1980; Freidin *et al*, 1991; Penzel *et al*, 1999). Reflux episodes are associated with electroencephalographic arousals. Compared to awake gastroesophageal reflux, acid clearance during sleep takes longer. Some studies suggested decreased frequency of swallowing and less efficient acid clearance (Orr *et al*, 1981, 1984). Thus, sleep-related compared to awake gastroesophageal reflux may be a more severe form of the condition, with longer-lasting reflux episodes, slower acid clearance, and more frequent or more intense patient complaints (Farup *et al*, 2001; Shaker *et al*, 2003).

Some have argued that negative intrathoracic pressure generated by the effort to breathe, following an apnea event, will help pull fluid up from the stomach, thus creating gastroesophageal reflux. However, existing evidence to support this pathophysiology appears to be limited to uncontrolled studies and case reports (Karkos *et al*, 2009). Whether obstructive sleep apnea causes or contributes to gastroesophageal reflux remains uncertain, although these two conditions share some common risk factors, such as obesity and supine sleep. Moreover, treatment of obstructive sleep apnea with a CPAP device may improve gastroesophageal reflux (Tawk *et al*, 2006). Gastroesophageal reflux has been associated with oral health problems such as dental erosion—because acid wears away tooth enamel—and chronic throat conditions such as hoarseness, sore throat, chronic laryngitis, difficulty speaking, cough, and granulomas on the vocal cords. Untreated chronic acid exposure due to gastroesophageal reflux can cause Barrett's esophagus, which is strongly associated with esophageal adenocarcinoma. Patients with suspected gastroesophageal reflux should be referred to an otorhinolaryngologist. Potential treatments include lifestyle changes, medication, and in some cases, surgery.

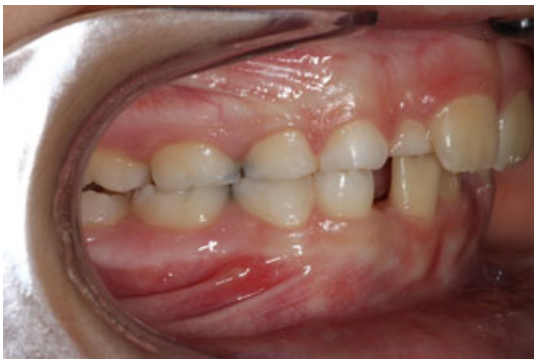

Figure 4 Teeth wear due to sleep bruxism (SB). (Source: A. Bellerive, Université de Montréal)

Sleep bruxism

Sleep bruxism (SB) is categorized as a sleep-related movement disorder in the International Classification of Sleep Disorders and is defined as 'a repetitive jaw muscle activity characterized by clenching or grinding of the teeth and/or bracing or thrusting of the mandible' (Lobbezoo *et al*, 2013). Rhythmic masticatory muscle activity (RMMA) during sleep is recorded in 60% of normal sleepers (Lavigne *et al*, 2001). SB is an exacerbated presentation of normal RMMA during sleep, with higher activity frequency, stronger electromyography (EMG) contractions, and accompanying tooth grinding noises (Lavigne and Kato, 2005). RMMA episodes are often preceded by and then associated with a sequence of physiological events: (i) preceded by about 4 min of increased sympathetic and decreased parasympathetic activity, (ii) preceded by 4 s of electroencephalograms (EEG) arousal, (iii) preceded by 1 s of increased respiratory amplitude and faster heart rate, (iv) onset of jaw-opening and jaw-closing muscle activity, and (v) swallowing of saliva (Kato *et al*, 2001; Miyawaki *et al*, 2003; Huynh *et al*, 2006; Khoury *et al*, 2008).

The consequences of SB can include abnormal tooth wear (see Figure 4), dental pain, temporomandibular pain, and headaches. Laterotrusive grinding, specifically incisor–canine–premolar–molar, and mediotrusive grinding patterns appear to have more deteriorative consequences on clinical gingival attachment level, tooth mobility, non-carious cervical lesions, and hypersensitivity (Tokiwa *et al*, 2008). SB is a problem not only for the patient, but also for bed partners, as the tooth grinding noise disrupts their sleep. Generally, patients with SB have normal sleep architecture compared to age-matched controls (Sjoholm *et al*, 1995; Lavigne *et al*, 1996; Macaluso *et al*, 1998).

The risk factors for SB range from psychological factors, oral habits, temporomandibular pain, medications,

and recreational drugs to medical conditions such as mental disabilities and other sleep-related disorders. Medications that may initiate or exacerbate SB include amphetamines, anti-dopaminergic drugs, anti-psychotic drugs, selective serotonin reuptake inhibitors, calcium blockers, and anti-arrhythmic drugs. Anxiety and stress are psychological factors that exacerbate SB (Pierce *et al*, 1995; Major *et al*, 1999). SB has also been associated with obstructive sleep apnea in some patients (Bader *et al*, 1997; Ohayon *et al*, 2001; Gold *et al*, 2003). Patients with SB reported a 2–3 times higher prevalence of obstructive sleep apnea (Ohayon *et al*, 2001). Recently, RMMA episodes were associated with an increase in respiration amplitude (Khoury *et al*, 2008). It was hypothesized that RMMA may serve to re-establish upper airway patency, which was decreased during an obstructive apnea or hypopnea, by repositioning the retruded mandible and re-establishing muscle tone in the tongue during swallowing (Lavigne *et al*, 2003).

The diagnosis of SB requires a positive history of tooth grinding noise reported by a bed partner with clinical observation of abnormal tooth wear and the patient's complaint of jaw muscle tenderness or fatigue (Lavigne and Kato, 2005). SB should be differentiated from other sleep-related orofacial movements that are associated with various parasomnias or sleep disorders. These include faciomandibular myoclonus (found in 10% of SB patients), obstructive sleep apnea, REM sleep behavior disorder, sleep-related abnormal swallowing, night terrors, confusional arousals, daytime dyskinetic movements, and sleep epilepsy (AASM, 2005). Moreover, rhythmic jaw muscle contractions (i.e., RMMA) without tooth grinding can be present in patients with partial complex or general seizures (AASM, 2005).

For SB, reports of tooth grinding noises by the sleep partner or a family member are often the main complaint and are considered a reliable indicator. Orofacial evaluation should include assessments of tooth wear, tongue indentation, ridge-like bite marks inside the cheek, temporomandibular joint sound, and masseter muscle hypertrophy during clenching (Lavigne and Kato, 2005). SB should be differentiated from tooth clenching while awake, as these have different pathophysiology and management requirements. As a cure for SB has yet to be found, the clinician can manage SB-related symptoms and exclude concomitant neurological or sleep disorders. Lifestyle and sleep hygiene, such as behavioral changes and relaxation exercises, can partially improve SB. To prevent orodental damage, occlusal appliances can be used (see Figure 5). However, they are not recommended for SB patients with concomitant obstructive sleep apnea, as a study showed that a maxillary occlusal appliance actually exacerbated obstructive sleep apnea (Gagnon *et al*, 2004). Mandibular advancement devices have been shown to reduce SB (Landry *et al*, 2006; Landry-Schonbeck *et al*, 2009) and could be considered as an alternative for SB patients who have concomitant sleep apnea. It has been hypothesized that improved breathing during sleep may decrease secondary SB. Some medications, such as benzodiazepines, muscle relaxants, or clonidine, have been suggested to decrease SB, but should be recommended only as short-term treatment.

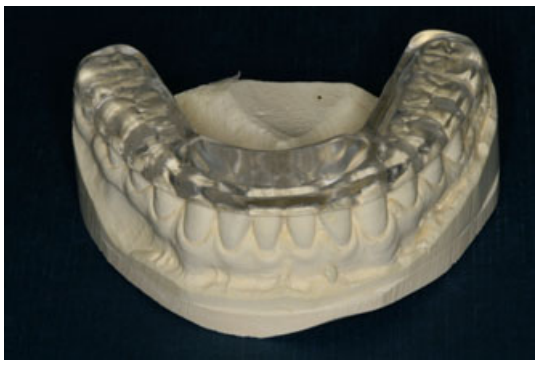

Figure 5 Occlusal splint used to protect from tooth wear and reduce jaw muscle pain (Source: P.-L. Michaud, Université de Montréal)

Edentulism

Edentulism is a debilitating illness defined as the absence or complete loss of all natural dentition. Aging substantially increases the risk of tooth loss and sleep disturbances. Sleep disturbances are reported to affect more than 50% of individuals aged 65 years and older (Foley *et al*, 1995; Neikrug and Ancoli-Israel, 2010). Anatomical changes associated with edentulism may predispose patients to obstructive sleep apnea. These changes include (i) decrease in the occlusal vertical dimension, (ii) change in the position of the mandible and the hyoid bone, and (iii) impaired oropharyngeal muscle function (Tallgren *et al*, 1983; Unger, 1990). Reduction in the retropharyngeal space associated with impaired function of the genioglossus and other upper airway dilator muscles increases upper airway resistance, which in turn increases the risk of apneic events.

Several studies have noted associations between edentulism and obstructive sleep apnea (Meyer and Knudson, 1990; Ancoli-Israel *et al*, 1991; Strohl and Redline, 1996; Bucca *et al*, 1999; Blanchet *et al*, 2005). Accordingly, ten percent of elderly people may show obstructive sleep apnea as a result of edentulism (Strohl and Redline, 1996; Bucca *et al*, 1999). In a cross-sectional study, Endeshaw *et al* found a statistically significant association between being completely edentulous and AHI $\geq$ 15 per hour of sleep (Endeshaw *et al*, 2004). Among 403 older individuals, 71% of those who did not wear their prostheses at night were at high risk for obstructive sleep apnea (Gassino *et al*, 2005). Sleeping without prostheses is associated with a significant increase in the AHI (Buysse *et al*, 1989; Bucca *et al*, 1999, 2006; Erovigni *et al*, 2005; Gassino *et al*, 2005; Gupta *et al*, 2011; Emami *et al*, 2012).

Despite these results and certain plausible anatomical explanations for the association of edentulism with obstructive sleep apnea, not all the research has confirmed this relationship. Results of a longitudinal cohort study indicated that edentate older adults are good sleepers, regardless of whether they wore their prostheses at night (Emami *et al*, 2012). In that study, poor oral health, quality of life, and general health were predictors of sleep disturbances (Emami *et al*, 2012). Furthermore, edentate

elders who wore prostheses at night had higher daytime sleepiness scores than those who did not. According to a recent study, the use of prostheses during sleep substantially increased the risk of apneic events in seniors affected by mild obstructive sleep apnea, but the magnitude of the 'denture detrimental effect' varied according to the severity of obstructive sleep apnea as moderate-to-severe patients' AHI was not significantly different when wearing dentures overnight (Almeida *et al*, 2012). However, in this study, patients with mild obstructive sleep apnea had higher body mass indices.

This paradoxical and controversial evidence precludes clinicians from making evidence-based clinical decisions on the issue. However, because nocturnal use of prostheses can increase the risk for certain oral diseases, such as candidiasis and denture stomatitis, most patients should be advised to remove their dentures at night unless they have or are at high risk for obstructive sleep apnea.

Management of patients with sleep and oral health issues

The multiple interactions between oral health and sleep underscore the need for an interdisciplinary clinical team to manage oral health-related sleep disorders. The team usually includes one or more sleep specialists and oral health specialists, such as a dentist, orthodontist, or oral and maxillofacial surgeon. The role of the dentist or orthodontist is to screen, assess, refer to a sleep specialist, and possibly refer to an otolaryngologist. The dentist or orthodontist also plays an important role in short- and long-term follow-up. Craniofacial morphology risk factors, in both adults and children, can be screened in patients with obstructive sleep apnea during their annual or biannual visit to the dentist. Patients who report EDS, snoring, or witnessed apneas, often with concomitant risk factors such as obesity, large tonsils, or specific craniofacial morphologies (narrow jaw, deep palate, retrognathia, macroglossia), should be referred to a sleep medicine physician. Obstructive sleep apnea and sleep-related gastroesophageal reflux are medical diagnoses.

Furthermore, general dentists and prosthodontists can provide obstructive sleep apnea therapy to suitable candidates, on the recommendation of the treating sleep physician, using various oral appliances such as a mandibular advancement device. Patients should be advised to maintain good oral health and healthy sleep hygiene. When using an oral appliance to prevent tooth damage or jaw muscle pain due to SB, dentists should choose the appliance according to the potential presence or absence of snoring and sleep apnea. Maxillomandibular surgery by either an oral and maxillofacial surgeon or otolaryngologist to treat adult obstructive sleep apnea is usually preceded and followed by orthodontic treatment to help ensure successful and stable repositioning. Orthodontists can also treat and prevent exacerbation of obstructive sleep apnea in children through guided craniofacial growth, such as rapid maxillary expansion and mandibular advancement with a functional appliance. Management of oral cavity and upper airway health can improve sleep by treating or decreasing the severity of obstructive sleep apnea or snoring. Successful management of obstructive sleep apnea can subsequently have positive consequences for many other sleep disorders, such as insomnia and parasomnias, which in some patients are secondary to the sleep apnea. Successful control of sleep apnea may also reduce subsequent risks for diabetes, hypertension, heart attack, stroke, arrhythmia, and premature death. In short, a surprisingly broad range of health benefits may be obtained when patient care is managed by a multidisciplinary clinical team, which can include various dental specialists, surgeons, and sleep medicine physicians.

Author contributions

Dr E. Emami contributed to the overall review and to the section on edentulism. Dr R.D. Chervin contributed to the overall review. Dr J.I. Helman contributed to the overall review and to the section on surgery. Dr N.T. Huynh contributed to the overall review.

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