Sleep Disorders Affecting Prognosis of Dental Treatment

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KEYWORDS

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KEY POINTS

- The main dental sleep disorders and conditions (sleep bruxism, obstructive sleep apnea, and gastroesophageal reflux disease) have a non-negligible prevalence and may be interconnected.
- Such conditions may have consequences of dental interest, ranging from tooth wear and intraoral complications to orofacial pains.
- Dental practitioners have the twofold role of sentinels and potential caregivers in the diagnosis and management of the main sleep disorders and conditions of dental interest.

INTRODUCTION

Among the main sleep-related disorders and conditions that are of interest for dentists, sleep bruxism (SB), obstructive sleep apnea (OSA), gastroesophageal reflux disease (GERD), xerostomia, hypersalivation, and the effect of orofacial pain (OFP) on sleep quality must be taken into consideration.[1] It is interesting to note that the Australasian Academy of Dental Sleep Medicine also introduced OFP and temporomandibular disorders (TMDs) as dental sleep-related conditions[2]; on the other hand, the American Academy of Orofacial Pain introduced sleep in their mandate.[3]

These changes indicate an ongoing evolution in the dental world, as they highlight that dental sleep medicine (DSM) embraces more than snoring and OSA. For this reason, a group of DSM experts[4] stressed that the management and treatment of

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this type of patients require collaboration between dentist and physicians. In particular, dentists can play a significant role in the prevention and/or assessment and/or management of patients with certain sleep-related disorders by observing the clinical consequences in the mouth and teeth and by identifying possible risk factors; on the other hand, it is fundamental to stress that physicians are responsible for the diagnosis and treatment of sleep-related disorders.

Within this scenario, several studies reported that dental sleep-related conditions frequently coexist and constitute a complex multimorbidity network. For instance, some studies discussed the existence of a possible correlation between SB and OSA,[5–7] while others suggest that patients with OSA experience more temporomandibular pain than otherwise healthy individuals.[8] In addition, a recent study shows that effective mandibular advancement device (MAD) therapy significantly reduces jaw-closing muscle activities that are time related to respiratory arousals in patients with OSA.[9] Concerning SB and GERD, it was found that they are temporally associated with each other, with bruxism episodes often occurring after reflux events.[10,11]

This study discusses the main sleep disorders and conditions (ie, SB, OSA, and GERD) affecting prognosis of dental treatment and examines the role that dentist can play in the assessment and management of these conditions.

Sleep Bruxism

Bruxism is a much debated oromandibular condition that interests several disciplines, such as dentistry, neurology, psychology, and sleep medicine, in both clinical and research settings. This condition is characterized by different activities of the jaw muscles (ie, grinding or clenching of the teeth and/or thrusting or bracing of the mandible) and 2 distinct circadian manifestations, viz., SB and awake bruxism.[12,13]

In 2018, an expert consensus provided the following definition for SB: "*SB is a masticatory muscle activity (MMA) during sleep that is characterized as rhythmic (phasic) or nonrhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals.*"[13] It is noteworthy that the definition begins with MMA, to emphasize the concept that bruxism would not be considered a disorder per se in otherwise healthy individuals, but it might be viewed as a protective and/or a risk factor for some clinical consequences independently on any specific neurologic correlates.[12–17]

The potential negative clinical consequences in the dental field include intrinsic mechanical tooth wear (attrition),[18,19] repeated fractures of teeth, dental restorations and implants,[20] masticatory muscle and temporomandibular joint pain,[21,22] and finally temporomandibular disc displacements.[23] On the other hand, some potential protective effects have also been considered, such as an increased upper airway patency that would aid in the prevention of the airway collapse leading to OSA[5,6] or a reduced risk of detrimental chemical tooth wear by increasing salivation in case of GERD.[11] These specific aspects will be discussed in the following paragraphs.

Current knowledge on the epidemiology of SB reflects the adoption of different evaluation strategies, since the literature reports wide ranges of prevalence for both adults and children/adolescents. The prevalence rates for SB in adults range from 8% to 16%, even if a comprehensive scoping review cautioned about the interpretation of results due to the poor methodological quality and inconsistency of the reviewed studies.[24]

In the attempt to provide homogeneity to the evaluation, a Standardized Tool for the Assessment of Bruxism has been recently proposed by an international expert panel.[25,26] The document highlights the need to evaluate not only the presence or absence of MMA (ie, bruxism status), but also the potential risk factors and comorbid

conditions as well as the potential clinical consequences.[25,26] The assessment relies on a combination of self-reported, clinical, and instrumental strategies. As for self-reported information, it can be obtained from questionnaires and history taking, and in the case of SB, also multiple informants can be interviewed (ie, bed partner or, in the case of children, their parents).[12–14] This approach can be useful to recruit large samples and to screen for the possible presence of bruxism at the individual level, without prejudice to the well-known limitations. The instrumental measurement of electromyographic (EMG) activity in the natural environment during sleep is the most appropriate approach available to collect information on SB behaviors.[27] In recent years, several home EMG recording devices have been introduced to detect SB episodes[28–30] as alternatives to the more technically demanding polysomnography (PSG). On the other hand, it is necessary to underline how these devices, in order to be used routinely in a clinical setting, must necessarily be improved both in terms of software and hardware.

As regards management, in the clinical setting it is necessary to underline that the association of bruxism with occlusal features is negligible, if at all present[14,31–34] and for this reason, performing irreversible occlusal changes with the aim to decrease pain symptoms in the jaw muscles and/or the temporomandibular joints (TMJ) or to reduce bruxism activities is not recommendable. Clinicians must keep in mind that since bruxism should not be considered a disorder but rather a muscle behavior that can be harmless, harmful, or even protective with respect to several health outcomes the treatment, where applicable, must be conservative referring to the so-called "Multiple-P" approach as the standard of reference[34]:

- Pep talk (counseling)
- Psychology (cognitive behavioral strategies)
- Physiotherapy (exercises of the jaw muscles)
- Plates (oral appliances)
- Pills (drugs)

In this scenario, the dentist represents the main figure for diagnosing and managing bruxism, but he/she should treat the disorder only if it results in negative clinical consequences for the patient (ie, TMD pain or severe tooth wear) and, importantly, by trying to address the causes of SB, which is often just an epiphenomenon of some underlying conditions that disrupt sleep. In addition, it must be pointed out that since SB appears to be associated with other sleep-related disorders (ie, periodic leg movements in sleep,[35] rapid eye movement sleep behavior disorder,[36] and insomnia,[37]), in more severe cases the diagnosis and treatment can be carried out together with medical specialists.[38]

Obstructive Sleep Apnea

OSA is a sleep-related breathing disorder characterized by repetitive episodes of a complete (apnea) or partial collapse (hypopnea) of the upper airway with an associated decrease in blood oxygen saturation, with oxygen levels falling as much as 40% or more in severe cases, or arousal from sleep; this pattern can occur hundreds of times in one night.[39]

The obstructive events (apneas or hypopneas) cause a progressive asphyxia that increasingly stimulates breathing efforts against the collapsed airway, typically until the person is awakened. It occurs when the muscles relax during sleep, causing soft tissue in the back of the throat to collapse and block the upper airway, leading to partial reductions (hypopneas) and complete interruption (apneas) in breathing that lasts at least 10 seconds during sleep. For this reason, this disturbance results

in fragmented, nonrestorative sleep that often produces an excessive level of daytime sleepiness.[40]

A common measurement of sleep apnea is the apnea–hypopnea index. This index is the combined average number of apneas and hypopneas that occur per hour of sleep. According to the American Academy of Sleep Medicine, it is categorized into mild (5–15 events/hour), moderate (15–30 events/hr), and severe (>30 events/hr).[40] Other indices, such as the oxygen desaturation index, are also emerging as important predictors for clinical impact.

It is interesting to note that from an epidemiologic point of view, OSA is a common condition that affects almost 1 billion people globally,[41] with 425 million adults aged 30 to 69 years having moderate-to-severe OSA (15 or more events/h).[42] Prevalence increases with age and is more frequent in men than in women, with a 2:1 ratio that tends to equalize over the age of 50 years.[43] Prevalence rates seem to increase following the rise in obesity rates; this could be explained by the fact that obesity appears to be part of the genetically (and phenotypically) determined characteristics of the apneic patients along with upper airway soft tissue structure.[44] As an important remark, it must be borne in mind that despite the potential severe clinical consequences, approximately 80% to 90% of adults with OSA remain undiagnosed.[45]

The pathophysiology of OSA is complex and involves an interaction between unfavorable pharyngeal anatomy and ventilatory control instability[46]; for this reason, anatomic features associated with a small upper airway volume (eg, retrognathia, maxillary hypoplasia, tonsillar and lingual hypertrophy, excess adipose tissue around the airway lumen) may facilitate collapse during sleep when muscle compensation is absent.[47,48] Large neck circumference, cervical soft tissue, vessels, and bony structures are among the anatomic factors that promote pharyngeal narrowing. Many of these factors promote pharyngeal collapsibility by decreasing the caliber of the upper airway or by increasing the upper airway surrounding pressure.[48]

The primary role of a dental clinician as far as OSA is concerned is to act as a sentinel.[49] The suspicion of OSA is based on the presence of clinical symptoms, such as daytime sleepiness, excessive tiredness, witnessed apneas, choking or gasping at night, nocturia, and loud snoring with periods of silence when airflow is reduced or blocked.[50] The majority of these signs and symptoms are easy to intercept through the use of simple and reliable self-report questionnaires (ie, the Epworth Sleepiness Scale[51] and the STOP-Bang questionnaire[52]) and clinical examination. Regarding the latter, the dental practitioner has the role of identifying physical characteristics that potentially predispose to OSA, such as mandibular retrognathia, maxillary micrognathia, arched palate and signs of nasal obstruction (ie, polyps, septal deviation, turbinate hypertrophy, significant congestion), and soft tissue hypertrophy (ie, tonsillar hypertrophy, macroglossia, enlarged or elongated uvula).[53] The early recognition of predisposing factors has a considerable importance, as it reduces the risk of long-term pathologic consequences of OSA (ie, systemic hypertension, cardiovascular, neuropsychological, and metabolic consequences).[54]

The definite diagnosis is made with PSG, which allows for measurement of the number of apnea/hypopnea as well as oxygen desaturation events to rate the condition's severity.[55] PSG is always recommended when clinical examination and history taking suggest the possible presence of OSA.[56] On the other hand, it is important to underline that PSG does not reveal the specific obstruction site, nor does it provide any differential diagnostic information as far as the potential relationship between the site of obstruction and OSA severity is concerned.[57] For this reason, drug-induced sleep endoscopy (DISE) is the standard of reference to evaluate the anatomic features of the upper airway, the number and location of the sites of collapse and the degree

and configuration of collapse. In addition, from a clinical point of view, this type of evaluation can guide the treatment choice, also including the predictability of MADs.[58]

Treatment of OSA patients' needs a multidisciplinary approach and it is classified into behavioral, medical, and surgical. The aims are to relieve symptoms such as loud snoring and daytime sleepiness and to restore normal breathing during sleep.

Behavioral therapy is always recommended when needed, but in the majority of cases, a combination with surgical medical or medical treatment is necessary. Continuous positive airway pressure (PAP) is considered the gold standard treatment for any symptomatic individual with OSA. In the cases of primary snoring, mild-moderate OSA, and those cases where PAP is not tolerated, a therapy with MAD is recommended and dentist can play a crucial role.[59] MADs are oral appliances that maintain the mandible in a protruded position with respect the normal relationship, resulting in an enlarged upper airway volume and, consequently, reduced obstruction.[60] Dental clinicians must take into consideration that patients with high loop gain and/or with multiple obstruction sites identified at DISE show poor response to MAD; on the contrary, young age, female gender, retrognathia, short soft palate, absence of severe obesity, tongue base collapse, and high collapsibility are predictors of positive response to MAD.[61,62] Concerning the surgical approach, the aim is to prevent collapse by modifying upper airway anatomic abnormalities. Multiple interventions have been described, involving different nose and throat specialist (ENT) and maxillofacial surgery techniques, which must be considered mainly in selected patients after noninvasive treatments have been unsuccessful.

As highlighted in the previous paragraphs, the role of the dental specialist is 2 fold, possibly acting both as a sentinel in identifying the pathology and a caregiver/management provider with the use of MADs.

Gastroesophageal Reflux Disease

GERD is a chronic gastrointestinal disorder characterized by the regurgitation of gastric contents into the esophagus.[63] It affects approximately 20% of the adult population in high-income countries, and in the United States, the prevalence of GERD is between 18.1% and 27.8%.[64]

The etiology of GERD is caused by multiple mechanisms leading to the disruption of the esophagogastric junction barrier, resulting in exposure of the esophagus to acidic gastric contents.

The pathophysiology of GERD is multifactorial, including the presence of a hiatal hernia, the influence of the tone of the lower esophageal sphincter, esophageal motility, and esophageal mucosal defense against the refluxate.[65]

Clinically, GERD typically manifests with symptoms of heartburn and regurgitation. It can also be present in an atypical fashion with extra-esophageal symptoms such as chest pain, dental erosions, chronic cough, laryngitis, or asthma.[66] Concerning dental erosion, several papers described a strong association between GERD and intrinsic chemical tooth wear. In addition, the severity of GERD symptoms seems to be associated with the severity of the tooth wear.[18,67] In addition, oral lesions, mucositis,[68] and burning mouth syndrome[69–71] were associated with GERD. Furthermore, symptomatic GERD is associated with chronic, painful TMD[72] and the increased TMD in patients with GERD.[73] There was an increased risk of postoperative complications of dentoalveolar treatment in patients with GERD.[74]

The diagnosis of GERD is made solely based on symptoms or in combination with other factors such as responsiveness to antisecretory therapy, esophagogastroduodenoscopy, and ambulatory reflux monitoring.[63] From a therapeutic point of view, the treatment options from the least invasive to the most invasive include lifestyle

modifications, medical management with antacids and antisecretory agents, surgical therapies, and endoluminal therapies, with the aims to address the resolution of symptoms and prevent complications such as esophageal adenocarcinoma and esophagitis.[75]

Since the complications of GERD should be promptly recognized, also dentists can play a determinant role in the diagnosis by recognizing complications at the dental level (eg, dental erosion), also because it can cause erosive (chemical) tooth wear, resulting in sensitive teeth. Consequently, the restorative dentist has a major role in this field.

INTERRELATION OF SLEEP BRUXISM, GASTROESOPHAGEAL REFLUX DISEASE, AND OBSTRUCTIVE SLEEP APNEA

The interrelation between SB, OSA, and GERD has raised a lot of interest in the fields of dental and medical research.[1,76] More than 30 years ago, Lavigne and colleagues defined DSM as a new discipline that focuses on several sleep-related disorders (viz., SB, OSA, xerostomia, hypersalivation, OFP, and GERD) that are of interest to dental clinicians.[77] Successive definitions have followed thereafter, in parallel with the increasing attention.

Concerning the correlation between SB and GERD, some investigations suggested that the 2 phenomena are temporally associated[10,11,78,79]: importantly, when the onset of a GERD event precedes the onset of an SB event, more tooth wear can be expected due to the fact that grinding on teeth covered by acid saliva may accelerate the amount of hard tissue loss by the SB activities.[10] On the other hand, to date, several studies investigated the existence of association between SB and OSA reported inconsistent and often conflicting results.[5–7,80] Thus, despite the many speculations, currently there is not enough scientific evidence yet to define a clear temporal relationship, if any, between SB and respiratory events during sleep.[5–7,80] Finally, GERD is also considered a factor that is associated with potential worsening of snoring and OSA, even if the evidence of a clear pathophysiology is still lacking.[81]

It is important to underline that most of the phenomena that fall into the category of DSM are disorders (ie, OSA, GERD) that have adverse consequences on the patient and should therefore be diagnosed and, if necessary, treated with immediacy. The only exception is SB, which is considered a muscular activity and therefore does not always occur to harm the patient; for this reason, a cautious approach to its pathogenesis is recommended before jumping to speculations on treatment, as is often typical of dentists.

In light of the available data, it can be suggested that dental sleep-related conditions are rarely found in isolation, while they frequently coexist and constitute a complex network.[82] In addition, these phenomena (ie, SB, OSA, GERD) have in common the capacity of potentially affecting the dental practice: SB may cause intrinsic mechanical tooth wear,[18,19] fractures of teeth, restorations, and implants[20]; GERD may cause dental erosion; and finally, the use of oral appliances in case of SB must be carefully valued to avoid possible worsening of respiratory pathology. For these reasons, dental practitioners play a relevant role in the prevention, assessment, and management of OSA, SB, and sleep-related gastroesophageal reflux condition.

SUMMARY

Sleep-related disorders have attracted a growing interest among dental practitioners for a twofold reason: (1) for the possible clinical consequences to which they can lead

and (2) for the potential role of the dentist in the screening and early recognition of individuals with sleep-related conditions.

The knowledge and the ability to identify signs and symptoms, risk factors and consequences of SB, OSA, GERD as well as their mutual relationships can be helpful to recognize other conditions and avoid their possible negative consequences at a systemic and oral level.

CLINICS CARE POINTS

- Sleep bruxism, obstructive sleep apnea and gastroesophageal reflux disease, also defined as sleep-related disorders and conditions, have a non negligible prevalence in the population, can often be found in association and, finally, may have consequences of dental interest.
- Clinicians must thus be aware of the fact that management of patients with these conditions may require a thorough evaluation and in this scenario they can play a key role in the diagnosis and treatment of these pathologies.

DISCLOSURE

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this article.

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