



ORAL SURGERY

ORAL MEDICINE

ORAL PATHOLOGY

REVIEW ARTICLE

Sleep disorders and the dental patient

An overview

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This article is intended to briefly describe common sleep disorders of interest to the dental profession and to render general management guidelines. Topics include sleep-related bruxism, xerostomia, hypersalivation, gastroesophageal reflux, apnea, and the effect of orofacial pain on sleep quality. The term *sleep-related* is used instead of the term *nocturnal* because some of the activities described can occur with daytime sleep. (Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88:257-72)

In daily practice, dentists are becoming more and more involved with patients requesting information or needing management of oral and dental conditions in relation to sleep-related disorders (Figs 1 and 2). Two of these disorders, sleep apnea and sleep bruxism are present in 2% to 8% of the middle-aged population.¹⁻³ Sleep apnea, the diagnosis of which is frequently masked by the presence of snoring, has major health and social consequences. Sleep bruxism is less deleterious to health, but its consequences, including tooth structure breakdown, exacerbation of facial pain, and a grinding noise that has a disturbing effect on the sleep of bed partners, are major

complaints that stimulate patients to visit the dental clinic. Less frequent consultations are noted for salivary flow problems such as xerostomia and hypersalivation and for gastroesophageal reflux (GR); their impact on health and quality of life is nonetheless important.

Dentistry is at yet another turning point in its history. Given the dramatic reduction in caries and the increase in nonsurgical management of periodontal disease, patients are requesting more global and integrated management for their oral health. This review article was written to help the dentist, generalist or specialist, in the management of oral and dental sleep-related conditions.

SLEEP-RELATED BRUXISM

Traditionally, sleep-related bruxism (SB) has been defined as a parafunctional activity that includes clenching, bracing, gnashing, and grinding of the teeth.¹ More recently, SB has been described in a more restricted way as orofacial motor activity during sleep that is characterized by repetitive (phasic) or sustained (tonic) contractions of the jaw-closing muscles (eg, the masseter and temporalis muscles). These contractions are associated with tooth grinding sounds, but the frequency can be variable in a given subject over time and between subjects over nights. Some patients grind once a month whereas others grind "every" night; with up to 90% of observed SB episodes,²⁻⁶ these subjects are classified as severe sleep bruxers.

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Dyssomnias

Disorders associated with difficulty in initiating or maintaining sleep or with excessive sleepiness

Examples:

Sleep apnea
Insomnia
Narcolepsy
Periodic limb movement or restless legs syndromes
Nocturnal eating
Circadian rhythms-related sleep disorders (jet lag, shift work, etc)

Parasomnias

Disorders of arousal or sleep stage transition; disorders that intrude into the sleep process but are not primarily sleep disorders (activations of the central and autonomic nervous systems are expected)

Examples:

Sleepwalking
Rhythmic movement disorders
Nightmares
REM sleep behavior disorders
Sleep bruxism
Sleep-related abnormal swallowing syndrome
Primary snoring
Infant sleep apnea/sudden death

Sleep disorders associated with psychiatric/medical disorders

Not primarily sleep disorders; manifestations inducing sleep disturbance or excessive sleepiness.

Examples:

Mental disorders (eg, related to anxiety, panic, alcohol overuse, etc)
Neurologic and/or movement disorders-related (eg, Parkinsonism, epilepsy, headaches)
Sleep-related gastroesophageal reflux (Fibrositis) Fibromyalgia
Fragmentary myoclonus (noted in 10% of patients with severe sleep bruxism)
Sleep choking syndrome

Fig 1. International classification of sleep disorders.³

The prevalence of frequent SB reports and awareness in the general population ranges from 6% to 8% and decreases rapidly with age; there is no gender predilection.⁷ Furthermore, it has been estimated that 1 in 5 sleep bruxers report concomitant orofacial pain.^{8,9} Daytime/awake clenching without grinding is reported by 20% of the population⁴; this condition should not be confused with SB and is probably a different entity.^{2,10}

Historically, occlusal discrepancies and emotional stress were seen as major causes of SB. However, their relative contribution has been challenged, to such an extent that the former is no longer considered the principal contributing factor and the latter is thought to be only part of the etiology.^{1,11,12} Current polysomnographic and clinical studies have linked SB to the field of sleep disorders through factors such as lightening of

sleep (ie, microarousals, which may be accompanied by electroencephalographic [EEG] K-complexes, rapid and transient EEG and electromyographic [EMG] activities, short electrocardiographic increases in heart rate, and frequent sleep stage shifts).^{2,4,13,14} In addition, brain chemistry (eg, dopaminergic sensitivity) has been associated with bruxism.¹⁵⁻¹⁷ Further investigations are ongoing within these areas.

To date, there has been little supportive evidence that SB is influenced by hereditary-genetic factors. Tooth grinding is observed with a frequency of approximately 23% in monozygotic twins.¹⁸ A familial pattern is also seen in approximately 20% to 35% of bruxers.^{18,19} Interestingly, SB in childhood persists in 87% of twin subjects when they are adults,²⁰ but this statistic is based on reports of bruxism history. Therefore, the presence of an inheritance pattern or the influences of

General sleep questions	YES	NO	Sleep/Snoring questions	YES	NO
Do you have difficulty falling asleep?	<input type="checkbox"/>	<input type="checkbox"/>	Are you told that you snore?	<input type="checkbox"/>	<input type="checkbox"/>
Do you have difficulty staying asleep?	<input type="checkbox"/>	<input type="checkbox"/>	Are you told that you stop breathing while asleep?	<input type="checkbox"/>	<input type="checkbox"/>
Is your sleep unrefreshing or of poor quality? (frequent if pain, periodic leg movement)	<input type="checkbox"/>	<input type="checkbox"/>	Do you choke, gasp or snort during sleep?(Hypersalivation must also be considered.)	<input type="checkbox"/>	<input type="checkbox"/>
Do you experience excessive sleepiness during the day? (Apnea must be ruled out if "yes.")	<input type="checkbox"/>	<input type="checkbox"/>	Do you have headaches or a sore throat in the morning?	<input type="checkbox"/>	<input type="checkbox"/>
Do you nap in the daytime? (Apnea must be ruled out if "yes.")	<input type="checkbox"/>	<input type="checkbox"/>	Are you a restless sleeper?	<input type="checkbox"/>	<input type="checkbox"/>
Do you sleep less than 5 hours per night? (exception: mother of infant)	<input type="checkbox"/>	<input type="checkbox"/>	Do you have high blood pressure?	<input type="checkbox"/>	<input type="checkbox"/>
Do you sleep more than 9 hours per night? (Narcolepsy and depression must be considered.)	<input type="checkbox"/>	<input type="checkbox"/>			
Do you grind your teeth during sleep? (This is most frequently reported by bed partner.)	<input type="checkbox"/>	<input type="checkbox"/>	Specific sleep questions to other sleep disorders	YES	NO
Do you clench your teeth during sleep? (There may be jaw lock-stiffness in the morning.)	<input type="checkbox"/>	<input type="checkbox"/>	Do you ever walk or talk in your sleep?	<input type="checkbox"/>	<input type="checkbox"/>
Do you have jaw lock or difficulty in opening your mouth in the morning?	<input type="checkbox"/>	<input type="checkbox"/>	Do your legs jump or twitch prior to or during sleep? (Suggest periodic limb movement.)	<input type="checkbox"/>	<input type="checkbox"/>
Are your teeth sensitive to cold/hot liquid or air? (This suggests clenching/grinding.)	<input type="checkbox"/>	<input type="checkbox"/>	Do you feel sad or anxious?	<input type="checkbox"/>	<input type="checkbox"/>
Do you have pain in the jaw muscles or joints	<input type="checkbox"/>	<input type="checkbox"/>	Do you have unusual dreams or nightmares?	<input type="checkbox"/>	<input type="checkbox"/>

NB: Comments in parentheses are for the practitioner's information; they should be removed if the questionnaire is to be completed by the patient.

Fig 2. Sleep questions that may help to identify (not diagnose) sleep-related problems in adults; modified from the Canadian Sleep Society's VitalAire questionnaire. (These questions are intended for interview support only; full validation for research purposes has not been completed.)

environmental factors on the onset of SB in childhood requires further prospective investigation.

Clinical recognition

From a clinical perspective, the recognition of SB^{2,3} by the dentist (Figs 2 and 3) is based on the combined presence of 2 major observations:

1. A current history of tooth grinding or tapping sounds (not snoring), as confirmed by a bed partner, and
2. The detection of abnormal tooth wear facets that are not compatible with a history of normal (functional) wear (eg, chewing, effects of a coarse diet), occlusal adjustment, dental treatment, GR, or occupational wear (eg, working in a dusty environment).

Because tooth wear may indicate a history of bruxism rather than a current disorder, the latter of these clinical

signs has been shown to be unreliable with respect to monitoring the condition.

In addition, one or more of the following signs and symptoms may concurrently be reported:

1. Temporalis headache
2. Jaw muscle stiffness or fatigue on awakening during the night or in the morning
3. A history of clicking or locking of the temporomandibular joint (TMJ) on awakening
4. Hypersensitive tooth
5. Masseter muscle hypertrophy.

Because SB is characterized by a cyclic remittent course,² these concomitant manifestations are not always present.

3.1 Recognition

Clinical

Complaint of tooth grinding sounds (confirmation by an observer or sleep partner)
Tooth wear (recently aggravated)
Jaw muscle discomfort or pain (headache)
Hypersensitive teeth
Masseter muscle hypertrophy

Sleep laboratory (audio-video with EEG or EMG)*

Muscle activity > 20% of awake maximum voluntary clenching level
Episodes of phasic EMG bursts ($n > 3$) and/or tonic contraction (> 2 seconds)
Grinding sounds

3.2 Management

Behavioral

Auto control
Biofeedback
Sleep hygiene
Assessment of concomitant use of medication and/or caffeine, nicotine, alcohol, cocaine, etc

Occlusal appliances†

Full arch hard acrylic splint
Soft mouth guard

Medications

Commonly used but risk of somnolence:
benzodiazepine (eg, Valium, Ativan, Rivotril)
muscle relaxant (eg, Flexiril, Robaxacet)
Avoid:
Selective serotonin re-uptake inhibitors (eg, Prozac or Zoloft: risk of increase in bruxism)
Unknown safety/efficacy:
Dopaminergic (eg, Sinemet/Prolopa, Parlodel)
Beta-adrenergic (eg, Inderal)
Botulinum toxin (eg, Botox, Synasport)

Other

Gel Kam for thermally sensitive tooth

*A sleep laboratory diagnostic (research criteria) is positive IF more than 4 EMG bruxism episodes/hour sleep are noted with at least 2 positive grinding (sounds) events/night.
†Be aware of possible increase in motor activity in some subjects.

Fig 3. Sleep-related bruxism.

Monitoring

Several methods are available for recording and documenting the motor activity associated with SB. Electronics such as home portable (ambulatory) recording devices allow monitoring of SB in the patient's natural environment, whereas access to sleep laboratory facilities (polysomnography) allows a better discrimination between bruxism events and other orofacial motor activities (eg, myoclonus, tics, rumination, somniloquy).²¹ Sleep laboratory evaluation offers the possibility of using audio/video signals coupled with EEG, EMG, electrocardiographic, and thermally sensitive resistor (airflow) signals to rule out the presence of sleep apnea, periodic arousal, epilepsy, and other orofacial activities.⁴ The cost of such evaluations remains a major drawback.

Polysomnographic scoring

New research diagnostic criteria⁶ have been proposed for the study of SB. According to these criteria, which are based on polysomnographic recordings, SB events are considered present on recognition of the following recording observations:

- EMG level of at least 20% of a maximum voluntary clench while awake
- Presence of phasic bursts if 3 bursts of >0.5 second each are noted, of tonic bursts if a sustained contraction lasts >2.0 seconds, or of a mixture of phasic and tonic bursts
- Presence of grinding-like sounds.

Through use of the following polysomnographic research criteria (sensitivity, >72%; specificity, >94%), SB could be predicted more than 80% of the time if:

1. More than 4 episodes of SB events (containing phasic, tonic, or mixed bursts) occur per hour of sleep or
2. (a) More than 25 EMG bruxismlike bursts per hour of sleep and (b) at least 2 grinding events (sounds) per night occur.

The assessment of the specificity of SB motor activity over other orofacial motor activities is needed because approximately 40% of reported instances could be related to swallowing, sighing, sleep talking, etc.^{6,21} Moreover, 10% of our clinically diagnosed SB patients mainly exhibited very rapid shocklike jaw muscle contraction while asleep.²² These activities are designated myoclonus (<0.25 seconds) and must be differentiated from sleep epileptic spikes.^{3,23}

Ambulatory recordings scoring

More recently, the following criteria for SB “event” detection by means of portable devices were suggested:^{24,25}

- EMG threshold >10% of the maximal “awake” voluntary contraction, with an event duration of >3 seconds and with <5 seconds between events.
- Concomitant to SB, a heart rate beat increase of at least 5% per minute.
- Acquisition at a minimum frequency of 16.7 Hz.

Both the diagnosis and scoring criteria for polysomnographic and portable devices now need full validation over larger population.

Management

Up to now, there has been no specific cure for SB. The role of the health care clinician is to correctly manage these patients. The main goal remains the prevention of damage to the orofacial structures. The different types of intervention can be divided into 3 groups: behavioral, orthopedic, and pharmacologic. Thus far, controlled studies confirming the efficacy of behavioral and pharmacologic strategies have been lacking. Some of the following recommendations directly address the patient’s awareness of the disorder and his or her control of behaviors that are related to stress and anxiety (more specifically, to the psychosocial reactive mode of the patient).

Behavioral strategies (derived from Zarcone et al²⁶)

1. Wind down during the second half of the evening. Rest for 60-90 minutes before bedtime; avoid intense thinking, discussions, and actions; separate body and mind from the day’s activities.
2. Learn a relaxation technique (eg, “conscious” respiration) and practice it during the day and before

bedtime. (Psychologists and physical therapists can help patients master such strategies.)

3. Maintain a good physical condition to feel healthier. (However, extensive exercise after 6:00 PM should be avoided.)
4. Avoid copious meals and beverages such as coffee (which should not be consumed after noon), tea, soft drinks, and alcohol (intake should be limited to the period around dinnertime; alcohol should not be consumed in the evening during the 3 hours or so preceding bedtime).
5. Stop smoking after 7:00 PM, because it has been suggested that nicotine increases muscle tone and arousal. Moreover, smoking habit is an SB-grinding risk factor.^{27,28}
6. Create a good sleeping environment, with a comfortable bed and mattress in a quiet room (hamper external sounds; use ear plugs when the bed partner snores or makes disrupting sounds while sleeping; adjust the temperature to approximately 65°F [18°C]; allow incoming fresh air).
7. Make arrangements, if an infant is in the house, to get at least one night of undisturbed sleep per week.

These behavioral strategies need validation so that their efficacy in SB management can be assessed.

Other techniques include biofeedback and hypnosis.^{29,30} Biofeedback appliances that use an audio signal have been applied in short-term management of SB, but they may cause sleep disruption for the bed partner. Hypnosis, considered as a form of relaxation therapy, reported as to aid certain patients; however, controlled trials to support its efficacy are lacking.

An occlusal appliance,^{1,12} such as a mouth guard or stabilization splint, “protects” the orofacial structures from damage (eg, bumpers action). The soft mouth guard is usually recommended for use on a short-term basis because its degradation can occur rapidly. The hard occlusal stabilization splint is particularly useful in patients who are frequent and severe grinders or clencher. However, not every bruxer finds relief with orthopedic treatment. It has been shown that approximately 20% of people with the condition have an increase in EMG activity when they wear an occlusal appliance.^{31,32} A recent critical review was published on the use of and mechanisms related to splints in cases of temporomandibular disorder (TMD) and bruxism.³³ More studies are needed for the sake of improvement in the understanding of splint efficacy, because this therapy did not reduce the occurrence (frequency) of SB-related motor activity^{34,35}; conversely, it has been reported to reduce the level of muscle activity.³⁶ Moreover, it remains to be assessed whether the use of

bite splints in sleep apneic patients with bruxism helps them, has no influence, or has a deleterious effect on respiratory sleep parameters.

Pharmacologic treatment is indicated for use on a short-term basis only. Centrally acting drugs in the benzodiazepine group (eg, diazepam [Valium]) and muscle relaxants (eg, methocarbamol-acetylsalicylic acid [Robaxisal], cyclobenzaprine [Flexeril]) are "known" to reduce bruxism-related motor activity. However, in the absence of data from controlled studies, their long-term use is contraindicated because of diurnal somnolence.⁴

Tricyclic antidepressants (TCAs), such as amitriptyline, have also been recommended for the management of SB.^{37,38} Unfortunately, a recent controlled study in which a low dose (25 mg) was used failed to support this recommendation.³⁹ In our opinion, this class of drugs should be used with caution; TCAs tend to reduce the duration of REM (rapid eye movement) sleep and to increase that of non-REM sleep stages 1 and 2, a period during which more than 80% of bruxism events occur in most patients with SB without a concomitant medical condition (eg, depression). In addition, it has been shown that with TCAs the tone of the jaw-opening muscles is increased during sleep.⁴⁰ Fluoxetine (eg, Prozac) was also reported to induce "complex movement disorders."⁴¹ SB, either clenching or grinding, was also reported in association with fluoxetine and sertraline (Zoloft), both of which are selective serotonin re-uptake inhibitors (SSRIs)^{42,43}; thus far, these observations have not been confirmed with objective sleep recording. Again, caution is suggested with serotonin-related medications in SB management.

The safe use of dopamine-related medications has not yet been demonstrated. As in a recent review, grinding bruxism can be noted secondary to chronic antidopaminergic drug exposure¹⁵; this could be considered iatrogenic bruxism, and it is sometimes noted with oral tardive dyskinesia.^{4,44} Two doses (100 mg L-dopa/25 mg benserazide $\frac{1}{m}$, one at bedtime and another 4 hours later) of acutely administered L-dopa (Prolopa) reduce bruxism activity in otherwise healthy SB patients.¹⁷ At the present time, however, too few controlled studies have been performed for a dopaminergic bruxism regimen to be considered a therapeutic modality for the general population. Furthermore, these drugs have the potential to provoke severe adverse effects and should only be used under medical supervision/collaboration.

Another pharmacologic avenue for SB management is use of the β -adrenergic antagonist, propranolol (Inderal). Two recent case studies reported a reduction of SB in one bruxer without any medical history⁴⁵ and

in 2 patients in whom iatrogenic SB was noted under antipsychotic (dopaminergic antagonist) medication.⁴⁶ Again, before adrenergic medication becomes a choice for SB management, controlled and double-blind sleep studies are needed to assess its efficacy and safety, because β -blockers have been reported to exacerbate sleep disorders such as REM behavior disorder, sleep apnea, and insomnia.⁴⁷⁻⁴⁹

Finally, botulinum toxin was used to reduce masseter muscle hypertrophy secondary to daytime clenching habits⁵⁰; however, both the efficacy and the safety of this toxin in SB management are unknown.

SLEEP-RELATED XEROSTOMIA AND HYPERSALIVATION

Occasionally, dentists have patients who complain of dry mouth or excessive salivation during sleep. Mouth wetness is closely related to the rate of swallowing and esophageal clearance. There are numerous reports indicating that the salivary flow comes to a complete stop with sleep onset and that swallowing is less frequent during sleep.⁵¹⁻⁵⁴ On the other hand, several other studies indicate that the salivary flow rate is only partially reduced during sleep.⁵⁵⁻⁵⁷ Hence, more research is needed on salivary function in relation to sleep, especially in patients diagnosed with Sjögren's syndrome who have concomitant poor sleep.⁵⁸

Sleep-related xerostomia can be defined as a sensation of dry mouth and is associated with a report of mouth or throat discomfort that may induce awakenings for water intake.³ The prevalence of self-reported dry mouth complaint during sleep (associated with awakening and water intake) is 23%; this compares with 8% for hypersalivation.⁵⁹

Sleep-related hypersalivation should be suspected when a patient reports excessive dribble or drooling of saliva that wets the pillow. Sometimes this induces awakenings because of associated discomfort. In extreme cases, this condition can be described as "sleep-related abnormal-swallowing syndrome,"³ which is mainly characterized by saliva aspiration, leading to coughing, choking, and brief sleep arousals. Hypersalivation may coexist with reduced swallowing activity. In the presence of abnormal swallowing, accumulation of saliva in the mouth and oropharynx and aspiration may take place.

Etiology

Whenever sleep-related xerostomia or hypersalivation is suspected, one must carefully assess drug intake (eg, central nervous system [CNS] depressants, hypnotics, cardiovascular drugs, caffeine) to rule out the presence of psychiatric disorders, to evaluate the

4.1 Recognition

Clinical

Complaint of sleep-related dry mouth or excessive dribble/drooling of saliva with wet pillow
Reported awakenings to sip water or related to hypersalivation that may induce coughing/choking

Sleep laboratory

Low swallowing frequency (extremely difficult to monitor accurately with EMG or strain gage; possible with ultrasound Doppler)
Several awakenings (eg, for water intake or to replace wet pillow)
Frequent EEG/EMG arousals with sleep fragmentation (eg, frequent awakenings, sleep stage shifts)

4.2 Management

Review of underlying disease

Assessment of medication side effect and/or caffeine, alcohol use

Gum/periodontium evaluation with assessment of tongue-related tics (eg, unstable denture)

Medical evaluation for upper airway infection/pathosis, gastroesophageal reflux, or Sjögren's syndrome

Planned schedule for sleep water intake and/or control of bedroom humidity

Prescription of sialogue* (eg, Sialor, Salagen) if xerostomia

Prescription of benzodiazepine or TCA* (eg, Elavil) if hypersalivation

*Suggested, not recommended in the absence of research evidence base.

Fig 4. Sleep-related xerostomia and hypersalivation.

condition of the teeth and periodontium, and to estimate the quality of the sleeping environment.

The medical conditions known to be involved in sleep-related xerostomia are upper airway infection or inflammation, diabetes, and Sjögren's syndrome. Such underlying medical conditions as sleep-related hypersalivation, obstructive sleep apnea (OSA), sleep-related GR, and upper airway pathosis should be ruled in or out through ear, nose, and throat, sleep laboratory, and neurologic evaluations as necessary.⁶⁰

CLINICAL AND POLYSOMNOGRAPHIC RECOGNITION

Clinical recognition

The clinical recognition of sleep-related xerostomia (Fig 4) is mainly based on the patient's report of a dry mouth. It is further suggested by a history of awakenings for water intake during the night. Reports such as "My mouth feels dry while I am eating," "I find it difficult to swallow solid food," and "I can facilitate the process of swallowing with the intake of fluids" are suggestive of salivary hypofunction during the day.⁶¹ However, there is no direct relationship between the levels of daytime xerostomia and sleep-related xeros-

tomia. When assessing salivary function, one should take into consideration that the flow rate is lower in the morning than during the day, probably worsens with anxiety-stress, and may be reduced as a result of clenching activity.⁶² At the other extreme, mastication and speech (or sleep talking) both increase the flow rate. We recently hypothesized that frequent chewing-like jaw activity or rhythmic masticatory muscle activity (1.7 episodes per hour of sleep) observed during the sleep of asymptomatic subjects (up to 58% of the population), may be related to oral lubrication and swallowing.⁶³

Sleep laboratory monitoring, such as noting the frequency of swallowing and the occurrence of multiple awakenings (EEG and motor activation) for water intake, can be considered, although it is extremely difficult to distinguish swallowing activity from nonspecific head-neck movements. Moreover, very little objective documentation on decreased salivary flow rate during sleep is available,⁵⁷ because the direct monitoring of the continuous flow rate of the major salivary glands in a freely moving patient is quite difficult. Indirect measures, such as recording the

5.1 Recognition

Clinical

Auto-awakening with complaint of sour taste and/or heartburn with supine position
Complaint of dysphagia
Tooth erosion and/or hypersensitivity to heat or cold
Damage to dental restoration

Sleep laboratory

Endoscopic evaluation
Low oral pH (< 4) measured with intubated pH probe
Low swallowing frequency
Awakening/sleep fragmentation
Insomnia or late sleep onset

5.2 Management

Medical evaluation (ENT, gastroenterology)

Mouth guard with fluoride to protect teeth from erosion

Meal-sleep schedule planning

Sleep water intake (or concomitant use of Salagen to increase salivary flow; however, efficacy must be demonstrated during sleep)

Medication: antacid, cholinergic, or proton pump (risk of xerostomia)

Other: CPAP (continuous positive airway pressure device, as in apnea management)

Fig 5. Sleep-related gastroesophageal reflux.

frequency of swallowing by means of ultrasound Doppler, have been used.^{51,52}

The diagnosis of sleep-related hypersalivation is clinically based on the patient's report of gurgling sounds that are sometimes noted by a bed partner.³ Sleep laboratory recordings will show a higher frequency of swallowing, although it should be noted that a diagnostic criterion for this finding has not yet been established. Again, elevated phasic-rhythmic jaw motor activity during sleep may contribute to the complaint of hypersalivation in patients with reduced swallowing. Future research is necessary to delineate the accuracy of this hypothesis.

Management

Because it has been suggested that these conditions could exacerbate burning mouth/dry mouth-related complaints, one should first screen for drug intake (as noted in the subsection on etiology), correct any abnormal orofacial habits (eg, mouthing, rumination, tics), and check on the fitting, stability, and retention of dentures.⁶⁴ It is also recommended that dental and periodontal infections be ruled out.

In cases of sleep-related xerostomia, consumption of coffee, alcohol, tea, and soft drinks should be avoided in the evening because these beverages enhance the reduction of the salivary flow rate. Water vaporization should be used to control the humidity of the bedroom.

The use of salivary stimulants at bedtime (eg, anetholetrithione [Sialor]) or pilocarpine-related drugs (eg, Salagene) may be beneficial, but no controlled sleep-related study has demonstrated their clinical efficiency to date. Hence, it could be suggested that a glass of water be kept at the bedside if awakenings with dry mouth discomfort occur.

For patients with xerostomia and abnormal swallowing, an awakening schedule for water intake may promote overall better sleep and reduce stress and anxiety related to the condition. For patients with oral breathing during sleep, a water vaporizer may help by increasing room humidity.

Hypersalivation without abnormal swallowing can be attenuated by drugs known to reduce the salivary flow rate (eg, benzodiazepines or low doses of TCAs). However, such medications may not be a good choice in a patient with a normal flow rate and a reduced swallowing rate, because these drugs are known to depress the motor system.

SLEEP-RELATED GASTROESOPHAGEAL REFLUX

In general, a patient with sleep-related GR will consult his or her physician with a complaint of being awakened during sleep because of heartburn and a sour taste in the mouth. In addition, such a patient visits the dentist because of enamel erosion, hypersensitive teeth,

failure of composite restorations, or a complaint of a sour taste. GR consists of regurgitation of the stomach contents, which are typically very caustic ($\text{pH} < 4.0$), into the esophagus during sleep.³ Our review of this condition can be concise, although GR has major consequences for the patient's quality of life and health. Because the condition is chronic for most patients, dysphagia frequently occurs and development of a Barrett's esophagus may be present, the latter being a possibly premalignant condition. Moreover, pulmonary aspiration with lung abscess and exacerbation of bronchial asthma has been reported.

Etiology

The pain associated with GR is associated with the regurgitation of gastric fluid or contents to the esophagus and pharynx. It seems that a lower esophageal sphincter pressure (< 10 mm Hg) or a decrease in peristaltic tone (abnormal clearance) predisposes a patient to GR or delayed gastric emptying (gastroparesis or obstruction). Although no gender difference has been noted, age (> 40 years), obesity, and pregnancy are predisposing factors. The hypothesis that acid clearance is low in patients with GR is controversial and needs further investigation. Similarly, the associated risk between sudden infant death syndrome and sleep-related GR is a topic of current interest.⁵⁴

Clinical and polysomnographic recognition

Any complaint of sour taste on awakening during sleep, with or without coughing, choking, or heartburn, and any report of dysphagia or epigastric burning pain should be considered clues suggesting the presence of GR (Fig 5). When the condition is suspected, the dentist should request further medical (otolaryngologic or gastroenterologic) evaluation to rule out the possibility of an associated condition such as peptic ulcer or angina. Endoscopic, histologic, and manometric esophageal examinations are currently performed to evaluate sphincter function, peristaltic efficiency, and swallowing function. Erosive esophagitis, Barrett's esophagus, laryngopharyngitis, and lung abscess are possible complications of GR.

A sleep laboratory evaluation with polysomnographic recordings will reveal frequent arousals from sleep, an esophageal or oral pH value below 4 (normal values range from 5.5 to 5.6), and an abnormal swallowing frequency.^{65,66} Ambulatory home monitoring with an intubated pH probe may also be performed (under medical supervision only) to further document the duration and frequency of GR episodes and concomitant problems (eg, chest pain, asthma); unfortunately, this technique is uncomfortable.⁶⁵ Interestingly, continuous oral pH

monitoring during sleep⁶⁶ did not reveal any fluctuation overnight or during the day (ie, no circadian rhythm was found). Through use of sleep laboratory recordings, sleep-related breathing problems and syndromes such as abnormal swallowing, choking, laryngospasm, and paroxysmal nocturnal dyspnea should be ruled out.

Management

It is the dentist's responsibility to recommend measures to prevent tooth erosion, degradation of fillings, and xerostomia discomfort. Referral to a family physician or gastroenterologist for management of the medical aspects of the symptoms is mandatory. To protect the teeth from erosion, patients with GR can wear mouth guards over both dental arches (the maxillary being the more important); the mouth guards can also serve as fluoride gel carriers. These patients should also develop a sleep hygiene strategy (as noted in the section on bruxism). For example, they should avoid certain medications (eg, benzodiazepine, hypnotics), foods, and beverages (eg, alcohol) for at least 3 hours before bedtime. Some patients experience relief when the head of the bed is slightly raised or when they use two pillows (control of the supine position). Regular water intake (a few sips before sleep and at each awakening during sleep time) may also help to reduce the "acid" reflux. Medical management includes the use of antacid medications (eg, Maalox or an H₂ receptor antagonist such as cimetidine or famotidine), cholinergic drugs (eg, bethanechol, a parasympathomimetic drug that increases esophageal sphincter tonus/peristaltic efficacy), and, in severe and/or complicated cases, proton pump inhibitors (eg, omeprazole, lansoprazole); surgery may also be indicated.⁶⁷ Moreover, use of a continuous positive airway pressure (CPAP) device in a study of patients with apnea was reported to improve GR both in patients with apnea and in subjects without apnea, as reported by Orr.⁵⁴ More collaborative studies are needed to further support the efficacy of GR management strategies.

SLEEP APNEA AND SNORING

Because the condition known as sleep apnea was recently well reviewed in this journal,⁶⁸ we will here merely summarize the essential information that dentists should have about sleep apnea and snoring. The conditions have similarities, but the diagnoses are different for snoring, OSA, and central sleep apnea (Fig 6).

Sleep apnea syndrome (SAS) is a medical condition in which dentists have become more and more involved during the last 10 years, ever since it was shown that some patients with SAS or snoring problems could be helped with oral appliances or by means of oral surgery.⁶⁸⁻⁷¹ Dentists should be aware that the oral

6.1 Recognition

SNORING (with and without apnea)

Clinical

Complaint of snoring by an observer or sleep partner
No excessive daytime sleepiness or insomnia complaint
Mouth dryness complaint at morning waketime or with awakening during sleep time
Overweight plus some of the features noted in sleep apnea

Sleep laboratory

Loud snoring sounds confirmed with neck microphone recording
Non-cyclical loud inspiratory/expiratory sounds
No associated arousals
Normal sleep and respiratory patterns
No cardiac arrhythmic or O₂ desaturation
Exacerbation in supine position

OBSTRUCTIVE SLEEP APNEA*

Clinical

Complaint of excessive sleepiness (sometimes observed by close observer)
Daytime hypersomnolence
Loud snoring
Obesity/bulk neck
Morning headache
Dry mouth
Intellectual performance deterioration
Sexual dysfunction

Sleep laboratory†

More than 5 obstructive apneas, > 10 seconds in duration/hour of sleep
Frequent EEG-EMG arousals with respiratory events
Bradycardia
Arterial O₂ desaturation
Short sleep latency (< 10 min; normal, 10-20 min)
Could be repeated during daytime as the MSLT (multiple sleep latency test) to assess daytime sleepiness
Rule out sleep gastroesophageal reflux, choking, periodic limb movement, asthma, etc

CENTRAL SLEEP APNEA‡

Clinical

Complaints of insomnia and/or excessive sleepiness with daytime nap or driving somnolence
Reports of memory impairments or other cognitive problems
Reports of headache at wake time
Report of loss of libido/erection problems
Report of depressive reactions
Reports of hypertension in some patients

Sleep laboratory

Apneic pauses > 10 seconds
Apnea/hypoapneas 10-30 seconds with return of respiratory effort
10-60 second hypoventilation after apnea
To discriminate from obstructive apnea, esophageal manometry is very helpful
Frequent arousals related to apnea
Bradycardia
O₂ desaturation
MSLT < 10 min

Fig 6.1. Snoring and sleep apnea.

appliances that are currently used to control snoring can mask the presence of SAS, which may be a life-threatening condition that needs medical monitoring. Hence, an oral appliance used because of snoring could easily harm a patient, acting as a “silent killer” in the absence of follow-up to reassess respiratory function during sleep. The presence or absence of OSA, central sleep apnea, or apnea of the mixed type (a combination of OSA and central sleep apnea) must be determined before initiating treatment with an oral appliance for snoring. Patients at risk as a result of complications associated with sleep apnea can then be identified.⁷²

Snoring is characterized by loud upper airway

breathing sounds during sleep.^{3,73} It may be associated with apnea and hypoventilation. Its prevalence markedly increases with age, reaching 40% to 50% of the general population in both men and women more than 65 years of age.

SAS is characterized by repetitive episodes of upper airway obstruction or by a cessation or decrease of ventilatory effort during sleep.^{3,73} The estimated prevalence in the general population is estimated at 2% to 6%. The disorder is encountered predominantly in middle-aged, overweight men; women (predominantly after menopause) and infants may also have SAS. Signs and symptoms secondary

6.2 Management

Medical evaluation (nasopharyngeal abnormality or pathosis, cerebral/ cardiovascular diseases, etc)

Review weight control strategy, alcohol use, drug regimen

CPAP (continuous positive airway pressure) device after controlling the pressure titration in sleep laboratory (pressure between 5-15 cm H₂O) for efficacy

Oral appliances (snoring and mild to moderate apnea)

Soft palate-uvuloplasty surgery

Orthognathic surgery

Pharmacologic therapy (eg, protriptyline) has thus far **not** been shown to be effective (One exception: use of paroxetine [a serotonin uptake inhibitor] improved non-REM breathing without any reversal of poor sleep architecture and daytime somnolence.⁹⁹ This new avenue of treatment needs further assessment.)

*Repetitive, sleep-related "upper" airway obstruction (nasopharyngeal abnormality, pathosis, or infection, obesity, hypothyroidism, acromegaly, etc).³

†Chest belt for respiratory movement, nose themistance for air flow, and finger O₂ saturation are used with EEG, EMG, and ECG signals.

‡Cessation or decrease of ventilatory effort during sleep, usually associated with O₂ desaturation. No anatomical abnormality noted, but nervous system or cerebrovascular lesion or cardiac disease must be suspected.³

Fig 6.2. Snoring and sleep apnea.

to the sleep disturbance and hypoxemia (deficient oxygenation of the blood) include, but are not limited to, the following: excessive daytime sleepiness, fatigue, memory impairment, mood disturbance, decreased libido, cardiovascular disease (eg, hypertension). Moreover, a higher risk of vehicle accident has been reported in these patients.⁷⁴

Etiology and predisposing factors

Snoring sounds are related to vibrations of the pharyngeal tissues (base tongue, soft palate, and uvula) as a result of air turbulence at inspiration. Obesity, nasal obstruction, large tonsils, retrognathia, depressant medications, and alcohol are predisposing factors.

SAS can have a central (depression of the motor respiratory system), peripheral (obstructive), or mixed (both central and peripheral) etiology. Again, obesity, drugs inducing CNS depression, alcohol, cigarette smoking, respiratory tract pathosis (eg, adenoid or tonsilar enlargement), craniofacial abnormalities (eg, micrognathia, retrognathia), and hypothyroidism or acromegalia are conditions that have been associated with SAS. It should be stressed that obesity is not an essential factor; thin people too may have SAS. When children with craniofacial modifications (eg, high angle face, adenoids) were compared with age-matched controls, a higher prevalence of upper airway obstruction-related symptoms was noted in the former.⁷⁵ Consequently, a good clinical

examination is recommended in younger snorers to rule out any physical abnormalities and prevent the consequences of SAS. Abnormal tonus of the pharyngeal muscles is reported to predispose a person to pharynx collapses during inspiration; this could be idiopathic, iatrogenic (eg, related to CNS-depressive medications), or specific to a neurologic lesion related to motor control of the pharyngeal muscles.

Clinical and polysomnographic recognition

A snoring patient can be recognized clinically by a history of reported snoring sounds in the supine sleep position and sometimes by excessive weight (body mass index), large tongue, retrognathia, large tonsil and/or soft palate, chronic use of a depressant medication or alcohol, and age (ie, being middle-aged).

For SAS, clinical recognition of a patient is again a matter of large body mass index, retrognathia, drug or alcohol use with narrow oropharynx, excessive soft tissue, large tonsil and soft palate, large tongue, and nasal obstruction. A "bulky neck" appearance is also common.

During clinical interview, patients with SAS may complain (or report from observation by sleep partner) about loud snoring, daytime sleepiness, unrefreshing sleep, morning headaches, choking during sleep, reduced libido or erection problems, morning drunkenness, memory impairments, depressive reactions, and enuresis.^{3,67} It should be noted that some orthognathic

class III surgical corrections may be associated with the appearance of snoring and/or apnea, possibly because of modifications in upper airway resistance/flow.⁷⁶ Medical examination for nasal obstruction, anatomical abnormality, or pharyngeal tumor is important.

In the sleep laboratory, polysomnographic recordings (Fig 6) will confirm a diagnosis of SAS if more than 5 apneic events, each with a minimum duration of 10 seconds, are present per hour of sleep. In addition, frequent arousals, bradycardia, and arterial oxygen desaturation may be noted. Ambulant monitoring is also possible with specially designed devices and software. Although such devices are useful for monitoring apneic activity in the home environment, the final diagnosis of SAS should be based on a complete medical evaluation by a physician trained in sleep medicine or respiratory disorders (pneumology).

Management

Dentists should not initiate treatment of SAS with an oral appliance without a prior medical evaluation of the patient. It is important to rule out nasopharyngeal obstruction, cerebrovascular and cardiac diseases, CNS lesions, the influence of a developmental anomaly (eg, retrognathia), and other conditions. The medical program of surveillance usually includes weight reduction and the use of nasal CPAP, but moderate long-term compliance is a limitation in this approach.⁶⁷ At this writing, sales of oral appliances are booming; more than 40 different types have been offered during the last 10 years. As recently reviewed by Lowe,⁶⁹ Schmidt-Nowara et al,⁷¹ and the American Sleep Disorders Association,⁷⁷ oral appliances should only be used in primary snoring (ie, without any concomitant medical condition) and in mild to moderate obstructive SAS. Use of an oral appliance could be suggested to a patient with moderate to severe SAS who is intolerant of or refuses treatment with CPAP; again, a written medical referral is strongly recommended.

The dentist should also evaluate the dental, periodontal, and temporomandibular conditions; the last of these could be aggravated by an oral appliance, as recently reported in 12.5% of patients.⁷⁸ Moreover, approximately 10% of patients using mandibular advancement devices for sleep breathing disorders over long periods showed changes in dental occlusion.^{78,79} Recalls are mandatory for the sake of monitoring and managing the efficacy of respiratory parameters and oral changes. In addition, nearly 10% of patients are not using these oral devices because of adverse effects (eg, excessive salivation, xerostomia, pain discomfort).⁷⁹ A recent short report stated that more than 25% of apneic patients had concomitant SB;⁸⁰ the design of an oral

appliance is therefore critical in patients with TMD or bruxism. Long-term compliance is similar to that associated with CPAP, varying from 50% to 86%⁷⁸; discomfort or unpleasantness is reported. Because so many different types of appliances are currently available and not all have been tested for efficacy and safety under controlled conditions, readers are referred to the specific papers^{69,71,77-80} for further information concerning appliance designs and limitations.

At the other extreme, a decision for major mandibular surgery has to be balanced with health risks, surgical complications, and expectations regarding long-term efficacy in subjects who are poor responders/compliers with respect to CPAP or oral appliances. Another alternative is laser reduction of the soft palate to reduce snoring sounds. Again, this must be performed with caution; patients may report pain, voice alteration, or effects on taste as a result of post-nasal drip.⁸¹⁻⁸³ Before and after oral treatment (oral appliance or surgery), polygraphic evaluation and reevaluation of sleep variables are mandatory.

OROFACIAL PAIN AND SLEEP PROBLEMS

It is not uncommon for patients with chronic muscle pain, including jaw muscle pain, to report poor sleep quality.^{44,84-86} Some patients with myofascial TMD/pain also report waking up during their sleep. Other facial pain associated with tooth pulpitis or conditions such as osteoarthritis of the TMJ and trigeminal neuralgia can also delay sleep onset or interrupt ongoing sleep.

Clinical and polysomnographic recognition

The most common sleep-related complaints of patients with trigeminal myofascial pain are restlessness, unrefreshing sleep, insufficient sleep, light sleep, disrupted sleep, and unrestorative sleep (Fig 7). Concomitantly, patients with orofacial pain may report excessive daytime fatigue, irritable bowels, and poor resistance to stress.^{86,87} The use of a diary for rating pain in relation to sleep habits (eg, bedtime, sleep latency, sleep duration) and diurnal activities (eg, times and types of meals, stress factors) is sometimes helpful in characterizing ongoing patterns and contributing factors (eg, anxiety). Frequently, it is the perception of sleep quality rather than real sleep performance that is impaired. Much more work is needed to understand the modifications in sleep microstructure of chronic pain and/or poor sleep complainers. A linear cause-and-effect relationship is plausible because pain is reported more frequently than sleep problems, but other variables, such as concomitant fatigue, mood disturbance or depression, and the presence of chronic poor health

7.1 Recognition*

Clinical

Jaw muscle pain or TMJ pain
Complaint of unrefreshing sleep
Complaint of insomnia (long sleep onset latency, several awakenings)
Fatigue, depression, and low fitness are frequently noted and are confounders
Poor resistance to stress, irritable bowels, etc are sometimes present

Sleep laboratory

Low % of sleep efficiency (awake intrusion into total time in bed/normal > 90%)
Reduce duration of sleep stages 3 & 4 (restorative sleep)
EEG alpha wave intrusion in sleep stage 3 & 4 (not specific to pain)
Frequent sleep stage shifts, EEG arousals, awakenings, body movement

7.2 Management

Behavioral approaches (see sleep bruxism)

Physical therapy

Oral splint

Medications such as hypnotics (eg, triazolam/Halcion), muscle relaxants (eg, cyclobenzaprine/Flexeril), TCA, (eg, amitriptyline/Elavil), and common analgesics (eg, ibuprofen, aspirin) could be used, but with caution because of adverse effects (eg, daytime somnolence) and lack of controlled study (except for triazolam) for orofacial pain-related sleep problems

*Normative sleep data for comparison between controls and chronic orofacial pain patients are missing in the literature.

Fig 7. Orofacial pain and sleep problems.

could be important when specificity assessments are considered in multivariate analysis.

The polysomnographic observation of a reduced sleep duration, an increase in the number of awakenings and body movements, and a reduced duration of sleep stages 3 and 4 (also known as restorative sleep) are also common in the “poor” sleeper, both with and without pain. The presence of cortical α EEG activity (waves between 8.0 and 12.0 Hz) is described in the literature as a sleep intrusion that reduces the quality of restorative sleep, and it has been noted in fibromyalgia and arthritis patients.⁸⁴ However, the specificity of this phenomenon in relation to pain remains to be assessed, because it has also been observed in several other medical (eg, depression, sleep apnea, periodic myoclonus) and environmental (eg, noise, excessive heat in the bedroom) conditions.^{44,84} Alpha EEG activity is usually scored manually after intense training or, more rarely, by means of computer-aided spectral analysis.⁸⁸ Although α EEG scores are useful to quantify sleep quality, they are not a specific marker of pain during sleep. For example, α EEG is noted in fewer than 36% of patients with fibromyalgia.⁸⁹

Management

In patients with chronic orofacial pain and sleep

complaints, both presleep rituals to promote good sleep and regular use of behavioral techniques (as described for sleep bruxism) are interesting and low-cost alternatives. The addition of physical therapy and/or relaxation techniques can also be useful. However, further validation of efficacy is needed for such approaches. A recent US National Institutes of Health panel on treatment for chronic pain and insomnia reached the following conclusions:

1. Strong evidence supports the use of relaxation and hypnosis in chronic pain management; a moderate effectiveness is seen with cognitive-behavioral techniques and biofeedback.
2. Use of cognitive-behavioral techniques and biofeedback produce “some improvements” in sleep, with a major residual question: What are the best outcome measures by which to assess such improvements—sleep onset latency or total sleep time?⁹⁰

Interestingly, it was noted that poor sleep is a secondary complaint to pain in most patients.⁹¹ Consequently, improvement in sleep quality does not completely relieve pain but does restore some quality of life.

Because some patients may present concomitant TMD problems and clenching and/or grinding related to bruxism,⁹ another approach used in patients with

chronic orofacial pain (TMJ and/or muscles) is the oral splint (described in the section on bruxism). Again, this type of oral device must be used with caution because efficacy is not observed in every patient.³³

Several medications have also been used to treat chronic musculoskeletal pain in patients with sleep complaints. It is interesting to note that the use of hypnotics such as triazolam (Halcion) in patients with chronic orofacial TMD has improved sleep without relieving pain.⁹² This is similar to the use of zolpiden (Imovan) in patients with fibromyalgia.⁹³ Muscle relaxants such as cyclobenzaprine (Flexeril) are frequently prescribed, but adverse effects such as daytime somnolence and dizziness limit their use to short periods.^{94,95} A common belief in the clinic is that amitriptyline (Elavil), a serotonin-related medication, improves sleep and should help in reducing pain by "resetting restorative sleep" in patients with fibromyalgia.⁹⁶ This has not been fully supported by recent findings, however, because amitriptyline does not reverse "alpha EEG intrusions," nor does it improve the pain conditions of fibromyalgia subjects.⁸⁹ The SSRIs, such as fluoxetine and sertraline, must be avoided for orofacial pain if clenching is reported, because this type of medication may exacerbate bruxism activity.^{42,43}

Finally, it should be mentioned that not every muscle pain condition induces poor sleep quality. For example, muscle pain associated with idiopathic cervical dystonia (spasmodic torticollis) is relieved by sleep. In such patients, both lying down without the intention to go to sleep and sleeping itself reduce the abnormal cervical muscle activities.⁹⁷ Moreover, masticatory muscle pain in sleep bruxers is not associated with major sleep disruption or complaints of poor sleep quality.⁹⁸ Further research is ongoing with respect to these interesting questions.

CONCLUSION

In conclusion, the role of dentists in the management of some sleep-related disorders is highly relevant. It is a unique chance to fill some of the gaps between medicine and dentistry, although dental practitioners must be aware of the potential risks of some treatments. Controlled studies are needed to assess not only the efficacy of medications and oral devices but also their possible beneficial or harmful effects in sleep-related orofacial conditions (eg, bruxism or apnea by SSRIs,^{42,43,99,100} the unknown effects of oral splinting on upper airway resistance, and the risk of apnea).

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