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REVIEW

Clinical handbook for oral, facial, and head pain; part II: sleep & pain

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Abstract

There is a complex relationship between sleep and oral, face, and head pain. In 2023, the Clinical Handbook of Oral, Face, and Head Pain was published in JOFPH, which explored the most common conditions in the field of orofacial pain, including the diagnostic criteria for each and the preliminary treatment options for each in a quick-reference format. However, sleep and its relationship to orofacial pain was intentionally omitted in anticipation of a second document solely dedicated to this topic. This article is the continuation of that work that explores the complex unequal bi-directional relationship between sleep and oral, face, and head pain from an evidence-based perspective. Sleep medicine as a discipline is within the scope of care of orofacial pain specialists, but it is not an officially recognized specialty within dentistry. However, sleep disorders are often associated with the pain experience for conditions like headaches, movement disorders, temporomandibular disorders, myalgia, and other painful disorders of the head and neck. Understanding the complexities of sleep and their relationship to orofacial pain are essential for collaboration with medical professionals who are best equipped to manage complex sleep disorders. Through a bullet-point format along with tables and example screening documents, the authors provide a quick-reference handbook for the clinician that may be used regularly in clinical practice, based on the current available published evidence in similar fashion to the original Clinical Handbook of Oral, Face, and Head Pain.

Keywords

Facial pain; Sleep wake disorders; Sleep apnea, obstructive; Mandibular advancement; Occlusal splints; Temporomandibular joint disorders; Sleep bruxism; Sleep initiation and maintenance disorders; Sleep disorders, circadian rhythm; Parasomnias

1. Introductory comments from the authors

The first publication in this series, the Clinical Handbook for Oral, Facial, and Head Pain [1], serves as a concise summary of common painful and non-painful temporomandibular joint (TMJ) disorders and painful disorders in the face, head, and neck [1]. While the authors recognized the importance of sleep related to pain; it was not included because it was determined that it was a topic too vast to address in the context of that article. Therefore, in collaboration with an author who has dedicated his career to the understanding of sleep and its relationship to pain, this addition to that text endeavors to explore the current knowledge base regarding sleep, specifically as it relates to pain and the painful experience.

This handbook, like the first, is not intended to be comprehensive, nor is it intended to be used as a stand-alone reference. In fact, the reader is encouraged to study the references listed at the end. As in the first Clinical Handbook of Oral, Face, and Head Pain, the goal is for this document to be used as a study reference tool and as a quick-reference guide in the clinical setting. Therapeutic options offered are backed by evidence when possible but may reflect the authors' personal opinions based on clinical experience when published evidence is lacking. As such, therapeutic modalities offered are not intended to be all-inclusive but represent current best practices. While diagnoses are discussed, readers are cautioned that in many locations, particularly in the United States, diagnosis of a sleep disorder must be made by licensed physicians based on face-to-face clinical evaluation and examination. In some instances, state laws may permit the diagnosis based on the outcome of home sleep testing (HST) or what is now more commonly referred to as home sleep apnea testing (HSAT) that is reviewed by a licensed physician. This matter should be reviewed on a case-by-case basis and on current practice guidelines [2, 3]. This area needs to be reviewed individually and is outside the scope of this publication. Where possible, appropriate coding based on the

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International Classification of Sleep Disorders, Third Edition (ICSD-3) [4] as well as on the International Classification of Diseases, Tenth Edition (ICD-10) codes (https://www.cdc.gov/nchs/icd/icd-10-cm.htm) have been included for clinical convenience; where terms vary from the ICSD-3 or the ICD-10 terminology, those terms are included within the description of the condition.

Screening for and providing assessment of the entities discussed in this handbook are, in the authors' opinion, within the scope of care of the Orofacial Pain (OFP) specialist and appropriately trained dentists, with the focus being on the adult population. Screening and assessment for sleep disorders and providing appropriate oral appliance therapy is part of the practice of dentistry, as documented by the American Dental Association (ADA), and particularly all accredited OFP residency programs, as is established by the Council on Dental Accreditation (CODA) in the United States. (https://coda.ada.org/-/media/project/ada-organization/ada/coda/files/orofacial_pain_standards.pdf?rev=58f801e1c8204893919a30bb8187f13d&hash=505A3259E98105802D69894A67174FD8). This is intended to provide screening/assessment for sleep disorders and for appropriate management strategies. Since OFP is now a recognized dental specialty in the United States as well as some other countries, sleep medicine as a discipline is well within the scope of practice of OFP specialists and practitioners.

Sleep medicine is a component of the OFP specialty practice but is not an officially recognized specialty of dentistry. Sleep medicine is closely related and may contribute to orofacial pain like headaches, movement disorders, temporomandibular disorders, myalgia, and other painful disorders of the head and neck. Management of sleep disorders by the dentist is a discipline in OFP that often necessitates collaboration with medical practitioners.

As the science of sleep medicine in dentistry evolves, ongoing education in this field is essential. It is the individual professional's responsibility to remain knowledgeable and updated regarding sleep disorders, testing that may be indicated, and its relationship to OFP. Even though the focus for the dentist as it relates to patient management is primarily focused on sleep related breathing disorders (SRBD), clinicians need to be aware that many other sleep disorders exist that may contribute to the pain experience. This is becoming increasingly more prevalent in the literature for the need to address sleep as it relates to conditions such as nociplastic pain, fibromyalgia, temporomandibular disorders (TMD), and headache. It is important, especially in the growing field of OFP, to remember that the concept of evidence-based practice includes a dynamic interaction between the following: the available scientific literature, patient factors (autonomy based on informed consent, physical and psychological health, *etc.*), and individual clinician experience. On the medical side, this philosophy of care has become known as shared decision making or personalized medicine [5].

Throughout this document, abbreviations and terms have been used that are commonly used in the field of sleep. Therefore, a glossary of relevant terms has been included at the end of the document in section 18. Additionally, the following terms have been abbreviated as identified in parentheses below:

- American Academy of Dental Sleep Medicine (AADSM);
- American Academy of Orofacial Pain (AAOP);
- American Academy of Sleep Medicine (AASM);
- Attention deficit disorder (ADD);
- Attention deficit hyperactivity disorder (ADHD);
- Adverse drug events (ADE);
- Apnea hypopnea index (AHI);
- Blood pressure (BP);
- Body mass index (BMI);
- Cognitive behavioral therapy, usually referred to a psychologist or psychological counselor (CBT);
- Centrally mediated (CM);
- Co-morbid insomnia and sleep apnea (COMISA);
- Cardiovascular disease (CVD);
- Decrease (↓);
- Diagnosis (Dx);
- Discontinue (D/C);
- Gastroesophageal reflux disease (GERD);
- Headache (HA);
- Health-related quality of life (HRQOL);
- Home sleep apnea test (HST);
- History (Hx);
- Intermittent hypoxia (IH);
- Increase (↑);
- Mandibular repositioning appliance (MRA);
- Mandibular repositioning device, commonly used synonymously with MRA (MRD);
- Multiple Sleep Latency Test (MSLT);
- Maintenance of Wakefulness Test (MWT);
- Non-rapid eye movement sleep (NREM);
- Non-steroidal anti-inflammatory drugs (NSAIDs);
- Oral appliance therapy (OAT);
- Oxygen desaturation index (ODI);
- Obstructive sleep apnea (OSA);
- Over-the-counter (OTC);
- Periodic limb movement disorder (PLMD);
- Polysomnogram (PSG);
- Quality of life (QOL);
- Respiratory distress/disturbance index (RDI);
- Rapid eye movement sleep (REM);
- Restless Leg Syndrome (RLS);
- Selective norepinephrine reuptake inhibitor (SNRI);
- Selective serotonin reuptake inhibitor (SSRI);
- Sleep bruxism (SB);
- Sleep efficiency (SE);
- Sleep latency (SL);
- Sleep Quality (SQ);
- Sleep-Related Breathing Disorder (SRBD);
- Slow wave sleep (SWS);
- Tricyclic antidepressant (TCA);
- Temporomandibular disorders (TMD);
- Total sleep time (TST);
- Treatment (Tx);
- Vertical dimension of occlusion (VDO);
- Wake after sleep onset (WASO);
- Within normal limits (WNL).

2. Sleep and pain: an unequal bidirectional relationship

Sleep quality, how sleep is perceived, is inherently intertwined with the pain experience through a bi-directional relationship [6–8]. This relationship is not equal. Sleep has a greater impact on pain than pain does on sleep [8] (Fig. 1). When a person is prone to chronic pain, sleep deprivation increases their pain sensitivity and lowers their pain thresholds [8, 9]; likewise, increased pain may fragment sleep and decrease the quality of sleep. Furthermore, poor sleep increases inflammation, which exacerbates pain [6, 7]; chronic pain elevates inflammation, which in turn reduces sleep efficiency. The relationship between sleep and oral, face, head, and neck pain, as well as pain in general, is a vicious cycle that affects one's quality of life [10].

As stated, the bidirectional relationship is not equal. When pain is present prior to initiation of sleep, it is not predictive of sleep quality. However, sleep quality may be associated with pain levels the following day. This applies to pain upon awakening and into the first part of the day and may not be applicable to the latter part of the day when pain levels may be increased. This is particularly the case for musculoskeletal pain [8].

Disrupted sleep alters pain modulation pathways, reducing pain tolerance, and increasing sensitivity to painful stimuli (Table 1) [9, 11]. In patients with one or more of the myriads of diagnoses of painful TMDs, poor sleep quality may be associated with increased muscle tension and jaw pain [6, 7, 12]. Sleep deprivation enhances central sensitization of pain—a phenomenon where the nervous system becomes hyperreactive to stimuli [11, 13]. Interestingly, REM sleep deprivation is known to be associated with hyperalgesia [9].

3. Classifying sleep disorders

The ICSD-3 is an internationally recognized classification system for sleep disorders. It is based on the original ICSD that evolved over time, currently now in the third edition. A few minor text revisions (TR) resulted in the (ICSD-3-TR) based on emerging evidence that impacts a few of the categories in the ICSD-3. Table 2 highlights the major classifications of sleep disorders [4].

4. Screening in the OFP practice

It has become the standard expectation since 2017 that dentists screen for sleep disorders on some level [14, 15]. Some clinicians choose to pursue advanced training in sleep, but sleep dentistry is not a recognized specialty. However, a large majority may simply ask the patient about their sleep and get the answer, "I sleep fine". This is often taken at face value that results in no further investigation. A better option for basic screening is to include a very simple questionnaire in the medical history review, the Pittsburgh Sleep Quality Index (PSQI) that could indicate one or more sleep disorders [16]. The PSQI can be found here: www.sleep.pitt.edu. Incidentally, this index is the only one currently available that is congruent with the biopsychosocial concept associated with OFP. Using the PSQI initially with new patients may uncover a sleep disorder and lead to a more definitive plan that can contribute to an improved overall outcome for pain management. While calculation of the PSQI is laborious, there are a variety of online calculators available free of charge that greatly simplify scoring, such as: https://calculatorlib.com/pittsburgh-sleep-quality-index-calculator.

5. Insomnia disorder

Insomnia is the most common sleep disorder and is highly prevalent [6, 12], both short-term (acute) and long-term (chronic). Most importantly, this disorder is most often associated with orofacial pain [6, 12, 17]. Because it is mostly uncovered by assessment of signs or symptoms, it is typically not subject to any form of testing such as the PSG or HSAT. It is important to understand that insomnia may be responsible for health and medical consequences often associated with sleep related breathing disorders, now known as COMISA (Co-Morbid Insomnia and Sleep Apnea) [18].

Testing (PSG or HSAT) is not indicated for insomnia, and

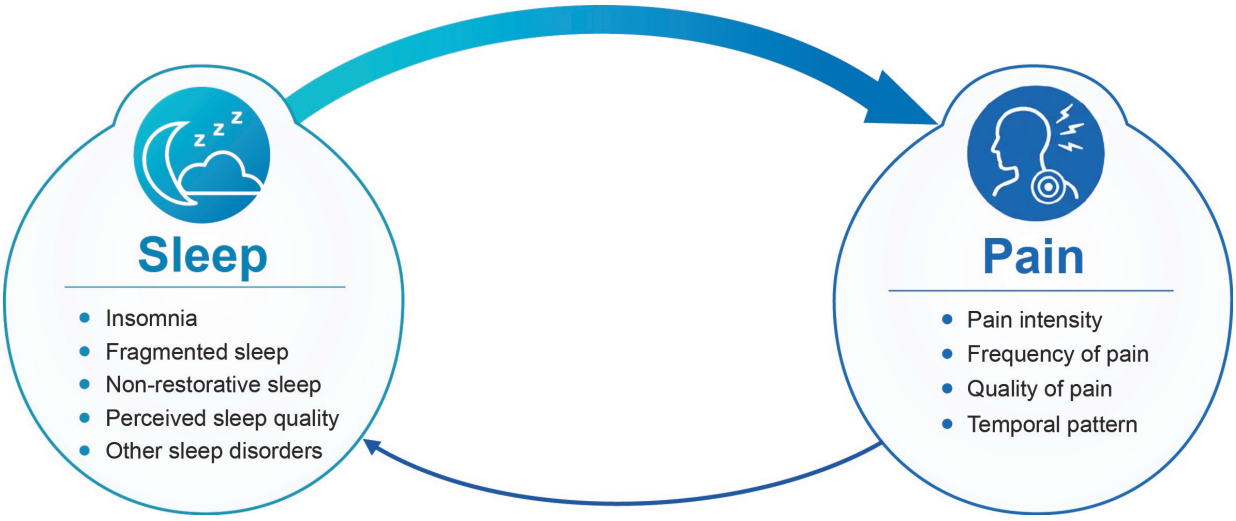


FIGURE 1. Relationship between sleep and pain.

TABLE 1. Relationship of sleep and pain.

Aspect of Sleep	Sleep Effect on Pain	Pain Effect on Sleep
Sleep Deprivation	Increases pain sensitivity and lowers pain threshold	Causes fragmented and poor-quality sleep
Sleep Duration	Short sleep duration correlates with increased pain perception	Chronic pain leads to reduced sleep duration
Sleep Quality	Poor sleep quality leads to increased pain intensity	Pre-sleep pain does not predict sleep quality
Inflammatory Response	Sleep loss increases pro-inflammatory markers, worsening pain	Chronic pain conditions elevate inflammation, reducing sleep efficiency
Central Sensitization	Sleep deprivation enhances central sensitization, amplifying pain	Chronic pain maintains a hyperactive nervous system, disrupting sleep

Sleep and pain share an interactive relationship with each impacting the other.

TABLE 2. Summary of sleep disorders.

Category	Subtypes & Examples	Description	Comment
Insomnia Disorders	● Chronic Insomnia ● Short-Term Insomnia ● Other Insomnia Disorder	Persistent difficulty with sleep initiation, maintenance, or early awakening.	Most often associated with pain.
Sleep-Related Breathing Disorders	● Obstructive Sleep Apnea (OSA) ● Central Sleep Apnea	Disorders causing abnormal breathing patterns during sleep associated with oxygen desaturation and fragmented sleep.	OSA can be treated with oral appliance therapy for mild and moderate cases. Oral appliance therapy is contraindicated for central sleep apnea.
Central Disorders of Hypersomnolence	● Narcolepsy Type 1 ● Narcolepsy Type 2 ● Idiopathic Hypersomnia ● Kleine-Levin Syndrome ● Insufficient Sleep Syndrome	Disorders characterized by excessive daytime sleepiness not due to disturbed sleep.	May have normal sleep efficiency. Need to rule out sleep apnea.
Circadian Rhythm Sleep-Wake Disorders	● Delayed Sleep-Wake Phase Disorder ● Advanced Sleep-Wake Phase Disorder ● Irregular Sleep-Wake Rhythm Disorder ● Non-24-Hour Sleep-Wake Disorder ● Shift Work Disorder ● Jet Lag Disorder	Disruptions in the body's internal clock leading to sleep timing problems, affecting sleep quality and daytime alertness.	Most appropriately managed by sleep physicians.
Parasomnias	● Non-REM Parasomnias (Sleepwalking, Night Terrors, Confusional Arousals) ● REM Sleep Behavior Disorder (RBD) ● Nightmare Disorder ● Sleep-Related Hallucinations ● Sleep Enuresis (Bedwetting) ● Exploding Head Syndrome	Abnormal behaviors, perceptions, or experiences occurring during sleep or sleep transitions, such as acting out dreams or sleepwalking.	Most appropriately managed by sleep physicians.
Sleep-Related Movement Disorders	● Restless Leg Syndrome ● Periodic Limb Movement Disorder ● Sleep-Related Bruxism ● Sleep-Related Rhythmic Movement Disorder	Disorders causing involuntary movements during sleep may disturb sleep continuity.	In medicine, viewed as a movement disorder. In dentistry, viewed as a behavioral disorder.

A summary of the six sleep disorders specific to the OFP practitioner. REM: Rapid eye movement sleep.

diagnosis is based on history. If co-morbid sleep disorder(s) are suspected, testing may be relevant for assessing OSA and/or PLMD. Insomnia is often associated with fatigue rather than feeling tired and being sleepy. Fatigue is infrequently associated with falling asleep in inappropriate environments or situations, especially during the day, whereas being sleepy and tired will often result in falling asleep in those same situations. Generally, someone who experiences fatigue related to insomnia does not easily fall asleep intentionally.

5.1 Diagnostic criteria for insomnia

5.1.1 Chronic disorder (F51.01)

Difficulty with the sleep-wake cycle not due to another/other sleep disorder(s) that are found to be present as well as a medical condition or medication/ substance use.

5.1.2 Short-term disorder (F51.02)

Also known as Acute Insomnia (short-term difficulty with symptoms).

5.2 Clinical features/symptoms of insomnia (main clinical features apply to chronic and short-term phenotypes)

(A) Difficulty with initiating sleep as well as maintaining adequate sleep that impacts quality or duration of sleep.
(B) Related to obstructive sleep apnea, and possibly other sleep disorders.
(C) May observe.
  (a) Mood disorders (anxiety/depression).
  (b) Cognitive difficulty.
  (c) Numerous health related concerns often associated with sleep apnea may be present.
    - Cardiovascular Disease (CVD).
    - Diabetes type 2.
    - Hypertension.
  (d) Fatigue with low energy and malaise.
  (e) Risk for motor vehicle and work-related accidents.

5.3 Association between insomnia and pain

(A) Prevalence: May be between 10 and 30% generally and 50% in those with chronic pain [8, 12, 17].
(B) There is a strong correlation between Insomnia and OFP [6, 8, 17], which is the rationale for strongly recommending screening and co-management as needed.
(C) Always consider that COMISA–OSA may be a contributing factor [18].

5.4 Screening and testing

(A) Questions.
  (a) Do you have difficulty initiating sleep?
    - Sleep-onset insomnia = more than 20 mins.
  (b) Do you have difficulty staying asleep?
    - Sleep-maintenance insomnia = frequent awakenings with difficulty going back to sleep.
  (c) Do you feel fatigued and tired, but you're not able to sleep or nap?
    - Co-morbid sleep disorder = unable to fall asleep involuntarily.
(B) Questionnaire: ISI (Insomnia Severity Index) [19].

5.5 Management—role of the dentist

(A) Insomnia is not typically a disorder directly managed by OFP specialist.
(B) When recognized, CBT-I (cognitive behavioral therapy for insomnia) may be delivered by a qualified practitioner [20], usually a psychologist (www.behavioralsleep.org).
(C) If associated with OFP or headache, may recommend reputable CBT-I self-care.
  (a) SleepioRx®.
  (b) Somryst® (a prescription program based on SHUTi).
  (c) CBT-I Coach® from app store.
  (d) Night Owl Sleep Coach® from app store.
(D) Offer BBTi (Brief Behavioral Treatment for insomnia) [20].
  (a) Derived from CBT-I, shorter in duration.
  (b) May be delivered by clinician involved with pain management.
(E) Manage any orofacial pain/headache related complaints.
  (a) Medications for OFP that may address insomnia while managing the OFP.
    - Anxiolytics (Benzodiazepines).
    - TCAs (Tricyclic antidepressants).
    - SNRIs (Serotonin-Norepinephrine reuptake inhibitors).
    - Melatonin.
    - Gabapentin.
  (b) Be aware of the impact of medications on sleep [21].
(F) Manage the OFP and assess the impact on insomnia symptoms.

6. Sleep related breathing disorders (SRBD)

This is the one disorder where dentistry has the most critical and important role associated with the use of oral appliance therapy (OAT). As such it may impact sleep in a way that contributes to the presence of orofacial pain [10]. In addition, this may impact the patient's health, overall well-being, and their quality of life (QOL), or what has been recognized as their health related QOL (HRQOL) [22].

6.1 SRBD criteria

(A) Obstructive Sleep Apnea (OSA) (G47.33) is the most frequently diagnosed SRBD [23].
  (a) Accompanied by snoring >90% of the time.
  (b) Prevalence is estimated at: 37% of the population.
(B) Central Sleep Apnea (CSA) (G47.31) commonly is associated with [23].
  (a) Opioid medication use.
  (b) High altitude.
(C) Mixed Apnea: combination of both OSA and CSA that occur together during the apneic episode.
(D) Hypoxemia: blood oxygen levels <88%.

(E) Intermittent Hypoxia (IH) or Oxygen Desaturation Index (ODI).
  (a) Frequency of drops in oxygen saturation levels of 4% or more.
  (b) Index often close to the AHI.
  (c) IH is critical to risk for health-related conditions to the SRBD.
(F) Hypoxic Burden.
  (a) A developing metric for a SRBD.
  (b) Referred to as the SASHB (Sleep Apnea Specific Hypoxic Burden).
  (c) Describes frequency of obstructive event in upper airway.
  (d) Quantifies the duration of the event and the depth of the oxygen desaturation.
  (e) Is measured in % min/hr.
  (f) Is associated with risk for related cardiovascular disease.
(G) Apnea-Hypopnea index (Indices) [23].
  (a) Index/indices = events per hour.
  (b) The AHI (apnea-hypopnea index).
    ● Mild = 5–14 events/hr.
    ● Moderate = 15 to 29 events/hr.
    ● Severe = 30 + events per hour − most consequential to health-related issues [10].
  (c) The RDI (respiratory disturbance/distress index).
    ● Indices the same as for the AHI.
    ● Incorporates recognition of arousals related to an apneic event: (RERAs or Respiratory Effort Related Arousal).
    ● Often will impact level of severity.

6.2 Clinical features of SRBD

(A) Symptoms.
  (a) Unrefreshing sleep.
  (b) Frequent snoring.
  (c) Witnessed/experience pauses in breathing.
  (d) Tired/sleepy during the day.
  (e) Low energy.
  (f) Memory issues/failing memory.
  (g) Mood disorders: anxiety/depression.
  (h) Gastroesophageal reflux disease (GERD) [24].
  (i) Hyperactivity or diagnosis of ADD/ADHD.
  (j) Headaches (especially in the AM).
  (k) Chronic orofacial pain that is difficult to manage (especially myofascial pain).
(B) Health and medically related conditions [7, 10].
  (a) Hypertension (particularly when poorly controlled).
  (b) CVD (especially A-fib).
  (c) Type 2 Diabetes.
  (d) Anxiety, depression, or other mood disorders or using medications for these.
  (e) Headaches (Table 3).
  (f) Overweight/obesity (BMI >30)—presence of increased abdominal girth.
  (g) Fibromyalgia.
(C) Clinical findings [23].
  (a) Snoring–gasping/choking during sleep.
  (b) Frequent awakenings from sleep.
  (c) Tooth wear with or without sleep bruxism.
  (d) Periodontal disease.
  (e) Large neck (≥17″ in males–≥14.5″ in females).
  (f) Waist size (≥40″ in males–≥35″ in females)—↑ abdominal girth.
  (g) Mallampati or Friedman Tongue Position (FTP) score of III or IV.
  (h) Tongue scalloping (crenations).
  (i) Narrowed oropharyngeal arch.
  (j) Long slopping soft palate.
  (k) Swollen/enlarged uvula.

7. Establishing risk for sleep disorders

Historically, screening establishes the risk for a SRBD. The ADA has stated that dentists should be screening for patients who may be at risk for obstructive sleep apnea [14, 15]. At a minimum, this involves the use of one or more questionnaires and recognition of anatomical risk factors. However, dentists may choose to utilize additional screening methods to identify risk (Fig. 2).

Once a dentist has identified that a patient is at risk for a SRBD, they may choose to further assess the condition, proceed directly to definitive testing, or make an appropriate referral to a sleep medicine specialist.

(A) Screening I and II: Investigation (subjective) as a means of establishing risk for a SRBD to determine if further action(s) is needed.
  (a) Screening I.
    ● Starts with the recognition of the presence of various health and medically related features and use of associated pharmacologic agents.
    ● Health related features: if any of these are present, suspicion of OSA should be considered: hypertension, CVD especially A-fib, type 2 diabetes, morning headaches, and orofacial pain that is not well managed.
    ● Interviewing the patient with questions similar to the following.
      ■ Does snoring occur frequently (3 + nights per week)?
      ■ When snoring, is there recognized (experienced) or witnessed pauses in breathing that result in gasping or choking and possible awakenings (arousals)?
      ■ Is the patient tired or sleepy during the day?
      ■ Does the patient awake feeling rested or not?
      ■ Does the patient have difficulty concentrating?
      ■ Are there challenges with memory?
      ■ Does the patient experience anxiety or depression?
  (b) Screening II: If positive responses are identified in Screening I, utilize a standardized and validated questionnaire, of which there are many options. The most utilized screening questionnaires are [25–28].
    ● GOAL 4-question brief screener (2 or more positive responses significantly increase the risk of OSA).
      ■ Gender (males are at higher risk than females).
      ■ Obesity (BMI >30).

TABLE 3. Headaches and sleep disorders.

Headache Type	Related Facts & Possible Associated Sleep Disorder
Tension type headache	
	Insomnia
	SRBD/OSA
	Movement disorder—especially SB
	Circadian rhythm disorder
Migraine headache	
	SRBD/OSA
	Insomnia
	Reduced deep sleep (N3)
	Circadian misalignment when going to Daylight Savings time
	Impairs Glymphatic system
Cluster headache	
	SRBD/OSA
	Related to Hypoxemia
	Related to REM sleep
	Impacted by circadian function—have seasonal predilection
Hypnic headache	
	Occurs after 2–6 hours of sleep
	Lasts 10–180 minutes
	Caffeine is advocated for relief
Cervicogenic headache	
	Impacted by postural changes especially during sleep
	SRBD/OSA (↓ N3 sleep)
	Insomnia and Circadian rhythm disturbances due to pain

Summarizes the potential sleep disorders that may be co-morbid to commonly observed headaches.

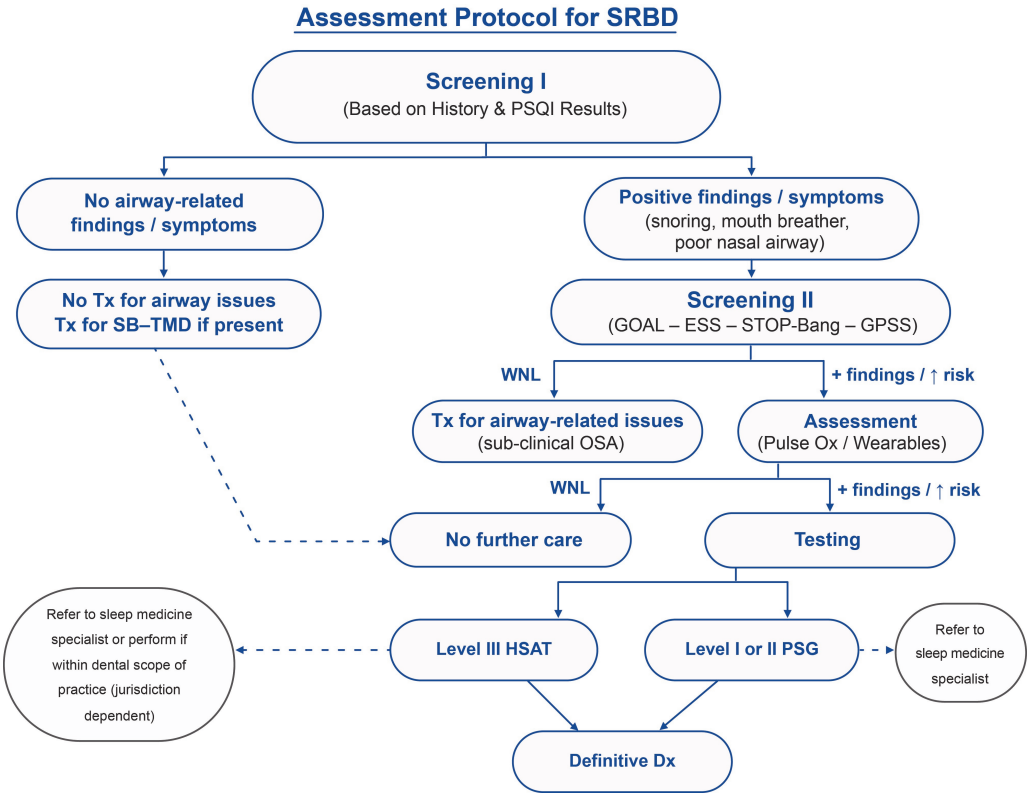


FIGURE 2. Flow chart of diagnostic process for sleep disordered breathing in a dental setting. SRBD: sleep related breathing disorders; PSQI: Pittsburgh Sleep Quality Index; Tx: Treatment; SB: Sleep bruxism; TMD: Temporomandibular disorders; GPSS: General Practice Sleep Scale; OSA: Obstructive sleep apnea; HSAT: home sleep apnea testing; PSG: Polysomnogram; Dx: Diagnosis; ESS: Epworth Sleepiness Scale.

- Age (50 years old or older).
- Loud snoring.

- Epworth Sleepiness Scale (ESS), which can be downloaded from here: https://www.cdc.gov/niosh/work-hour-training-for-nurses/longhours/mod2/epworth-p.pdf.
- STOP-Bang Questionnaire, which can be downloaded from here: http://stopbang.ca/osa/screening.php.
- General Practice Sleep Scale (GPSS).

(B) Assessment: Objective investigation to further establish risk. These assessment modalities rely on the use of photoplethysmography (PPG), a non-invasive optical method used to track pulse, heart rate variability, and oxygen saturation.

(a) Overnight Pulse Oximetry.
- Easy to use.
- Signal based on transmissive PPG signal.
- ODI closely aligns with AHI.

(b) Cardiopulmonary Coupling (CPC): (Uses the Sleep Image® technology).
- Relies on Peripheral Arterial Tonometry (PAT) signal.
- Employed more as Level II testing.
- Associated with autonomic nervous system activity.
- Related to Heart Rate Variability (HRV).
- Not a stand-alone assessment process.

(c) Other methods referred to as wearables.
- Smart watch (uses reflective PPG signal).
- Smart rings (*e.g.*, Oura, Belun (also uses CPC), Circul Plus, *etc.*).

(C) Testing: Of the four levels as defined by the American Academy of Sleep Medicine (AASM), levels I to III result in a definitive diagnosis [23].

(a) Level I: PSG monitored by a trained technician.

(b) Level II: PSG unattended by a technician.

(c) Level III: Home Sleep Apnea Testing (HSAT).
- Most prevalent method used currently.
- Used when pretest probability for OSA exists.
- Most provide PSG quality data.
- Devices are rapidly developing and changing, more compact, and simplistic.
- Evolving into PSG quality (Level II) testing with the addition of Electroencephalography (EEG) and Electromyography (EMG) (example is NoxA1).

(d) Level IV: utilizes 2 channels, one of which is oximetry.
- Does not result in a formal diagnosis.
- Further determines risk for a SRBD.
- Often leads to use of level III study.

(D) EXPERT COMMENTARY: Using level IV testing in the dental practice to assess for risk is equivalent to assessing blood pressure; it does not lead to a diagnosis by the dentist. Positive findings indicate the need for additional evaluation and potential management by a physician.

8. Medical management of SRBD

(A) Positive Airway Pressure (PAP) therapy.

(a) Continuous PAP (CPAP): considered the gold standard [23].

(b) Alternatives.
- Bi-level PAP therapy (*e.g.*, BiPAP®).
- On-demand–at critical time (Kairos) PAP (*e.g.*, KPAP®).
- Expiratory pressure devices (*e.g.*, Bongo®).

(c) Two components of PAP therapy.
- IPAP (Inspiratory PAP) is pressure from a machine–pneumatically splints the airway.
- EPAP (Expiratory PAP) is pressure produced by the patient during expiration that splints airway opening.

(B) Surgical Intervention [29].

(a) Focuses is on anatomic factors.

(b) Bariatric surgery.

(c) MMA (Maxillomandibular Advancement).

(d) Hypoglossal Nerve Stimulation.

(e) Soft tissue surgery (*e.g.*, UPPP (uvulopalatopharyngoplasty) and LAUP (laser-assisted uvulopalatoplasty), hyoid advancement, *etc.*) are not as prevalent in modern practice.

(C) Oral Appliance Therapy (OAT), also known as.

(a) Mandibular Repositioning Device/Appliance (MRD or MRA).

(b) Mandibular Advancement Devices (MAD).

(c) Mandibular Anterior Splint (MAS).

(d) Oral Sleep Appliance (OSA).

(e) Oral Airway Dilator.

(f) EXPERT COMMENTARY: These terms are not the same as OA used for bruxism, TMD, or other dental conditions. A large number have been cleared by the United States Food and Drug Administration (FDA). On the FDA site use LRK (anti-snoring) or LQZ (jaw repositioning devices) in the product code section. To obtain a full listing of oral appliances go to: https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMN/pmn.cfm.

9. Oral appliance therapy (OAT)

OAT, herein referred to as mandibular repositioning devices, MRDs, for clarity, and the management of OSA is the area where dentistry's role in sleep medicine can be most impactful. MRDs are devices designed to treat SRBDs (OSA and snoring) by maintaining an open airway during sleep, by repositioning of the mandible. MRDs are commonly secured to both the maxillary and mandibular dental arches. These appliances function to treat a SRBD by repositioning the mandible protrusively and vertically downward, thereby increasing the area of the oropharyngeal airway. The improvement may be in the anterior-posterior dimension, but improved lateral dimension is equally if not more critical. Of greater importance is the ability of MRDs to prevent the mandible and tongue from collapsing into the oropharyngeal airway space. The repositioning of the mandible should be viewed as both vertical and horizontal (in most cases mandibular repositioning cannot be done without some vertical opening first). MRDs not only reposition the mandible, but they also impact the base of the tongue, which opens the oropharyngeal airway

three-dimensionally. MRDs are of main types: traction-based that pull the mandible forward and compression-based that feature either flanges or hinges (rods). Both limit retrusion of the mandible and seating of the condyles. One important element to these devices is that the occluding surfaces of these appliances must be flat and balanced throughout the protrusive and lateral movements of the mandible to avoid injuring forces being applied to the condyles. MRDs have been proven to be successful in managing mild and moderate OSA [30, 31]. A contraindication that has been identified to MRD therapy for OSA is that they are not indicated for patients with "active TMD" [32]; however, TMD in this statement is poorly described, and the argument may be made that some clinicians treat painful TMDs with anteriorly repositioning OAs that may be either single or dual-arch designs. In fact, an anterior repositioning appliance is an active appliance like the MRD, but it is only indicated for short-term use to manage certain acute painful TMD conditions like acute painful disc dislocation without reduction (closed lock). The argument for using a MRD designed for a SRBD to treat an active TMD is flawed because they are active, restrict the range of motion needed to improve painful TMD, and carry the published risks of adding injury to the temporomandibular joints.

It is important for the clinician to understand that airway is affected by oral appliances, including MRD and OAT, in three dimensions. It has been reported that the vertical opening component is equally as important to affecting airway dimension as the horizontal component [33]. An alternate approach when an MRD is contraindicated clinically is the use of the dual-arch flat plane individual stabilization appliances, where the total vertical dimension inter-incisally is between 5 and 8 mm, depending on the size of the patient. Bailey and colleagues proposed 5–6 mm for females and 6–8 mm for males [33]. In this manner, the TMD is managed while addressing the SRBD. It is important to understand that the literature remains generally inconclusive regarding the optimal vertical dimension for OAT and for MRD. Generally, the literature warns against exceeding 8 mm of vertical dimension increase at the incisors as this may restrict the airway via clockwise autorotation of the mandible and retrusive movement of the base of the tongue [34]. However, the literature generally supports the concept that if the vertical dimension of occlusion (VDO) is increased by 6 mm at the incisors through either one or two appliances, there appears to be no negative impact on the airway and that VDO increases at or below 6 mm at the incisors likely do not have a net positive effect on the airway. Any negative airway effect due to increasing the VDO appears to be primarily by concurrent mandibular rotation and resultant posterior tongue position rather than the vertical increase itself, and the effects are highly variable between individuals. Furthermore, there are inconsistent approaches to measuring airway in research since the research has typically been done when the subjects are alert and awake, not asleep. Therefore, the clinician must rely on both the art of interpreting symptoms and the science of understanding that there are nuances of airway during sleep that currently are not fully understood.

(A) Proposed criteria for MRD therapy [33].

(a) Adjustable.

- Easily adjusted to accommodate changes in the dental status.
- Can be modified to address a new restoration(s).
- Ability to reline or modify OA to improve retention or the fit.

(b) Provide Posterior Support.

- Addresses Sleep Bruxism (doubles as a bite splint/night guard) as well the SRBD.
- Adequately support the TMJs.

(c) Titratable, able to be calibrated.

- Clinical consideration: Titration beyond 50% may not significantly increase success rate [35, 36], but 70% of the protrusive range of motion likely contributes to tooth root resorption [37].
- Modification of the appliance that is designed to improve effect by modifying the degree of advancement and/or by opening increasing the vertical dimension [30, 38].

(d) Provides Full Tooth Coverage.

- All the teeth need to be covered to prevent movement or unwanted super-eruption.
- Cover the distal of the most posterior tooth to prevent contacts from opening.
- Maintain the teeth in the pre-treatment position within each arch.

(e) Enables Mandibular (jaw) Mobility.

- To allow freedom of jaw movement during sleep–termed rhythmic masticatory muscle activity (RMMA) that is commonly found in all patients.
- Improves comfort of the OA during sleep–especially with sleep bruxism.
- Allows for normal function such as swallowing and licking the lips.

(f) Permits Lip Seal and Patent Nasal Airway.

- Comfortable lip seal to limit mouth breathing during sleep.
- Open nasal airway to improve oxygen level, may require adjunctive treatment of the nasal airway.

(g) Enables Maximum Tongue Space.

- Allow for maximum space for the tongue to further prevent the collapse of the tongue into the oropharynx.
- Decrease the encroachment of the OA into the tongue space that may cause the tongue to be in a more posterior position—especially lingual to the anterior teeth.

(B) Combination Therapy: PAP therapy and OAT may be used together [39].

(a) Reduce AHI along with the ODI and improve oxygen levels.

(b) Reduce PAP pressure, thus improved adherence and/or compliance.

(c) If PAP is not tolerated throughout the full night, OAT may continue to be utilized for the remainder of the night.

(d) Provides the overall best outcome, Improved Mean Disease Alleviation (overall effectiveness of OSA treatment).

- Results in improved levels of treatment efficacy (reduced AHI).

- Greater adherence to prescribed therapy.

(C) OAT for Sub-clinical OSA.
  (a) May be viewed synonymously as Upper Airway Resistance Syndrome (UARS).
  (b) Situation where symptoms are present, but AHI/ODI is <5.
  (c) Utilizes OAT—same as for OSA and snoring.
  (d) Goal: reduce/eliminate symptoms and have improved QOL.

SRBD are becoming more common as obesity also becomes more prevalent. Like other sleep disorders a SRBD may not be a singular disorder, thus the recognition of others is common. This disorder may be comorbid to OFP complaints and to SB as well. In fact, anecdotally approximately 50% of OFP patients had a concomitant SRBD, thus the use of the correct appliance is paramount. When it comes to the use of OAT, it must also be recognized that this is infrequently a stand-alone therapy. Many times, other related conditions, such as nasal airway restriction, mode of breathing (mouth *vs.* nose), and tongue posture, need to be managed as well.

Since it has been reported that the vertical opening component is more important than the protrusive positioning for MRD therapy in managing sleep disorders, the authors have anecdotally observed reduction in HSAT AHI values of approximately 5 in more than 50 patients treated with OA therapy for TMDs using mandibular flat plane stabilization appliances of 5–7 mm thickness inter-incisally (6–8 mm opening from maximal intercuspal position on the incisal guide pin of a semi-adjustable articulator) for women and men, respectively, by conducting HSATs with and without the appliance in place. Therefore, a reasonable approach to OA therapy with a patient who has sub-clinical, mild, or low moderate SRBD or in combination therapy with PAP for moderate or severe OSA is the use of the dual-arch flat plane stabilization appliance in a patient who has an active temporomandibular joint disorder. In this manner, the TMD may be managed as well as the sleep disorder as part of a comprehensive OFP management plan. With vertical opening alone and in the absence of purposeful mandibular advancement, the space for the tongue is increased, and a muscular hydrostat will occupy more of the oral cavity and potentially less of the oropharyngeal airway.

One of the challenges with OAT for both the management of TMDs and SRBDs is that occlusal changes may occur. This may either occur from stabilization and remodeling of injured or disease-altered temporomandibular joints or from soft tissue changes related to intentional repositioning of the mandible to alter the airway. In both scenarios, the potential benefits of the appliance must be weighed against the risks of transient and sometimes permanent occlusal change. This is why developing a basic understanding of pre-treatment occlusion is important for both the clinician and the patient and why it is a necessary element of the informed consent process. OAT is one element that may be considered in an overall treatment plan for the treatment of several TMDs, but where the clinician must weigh the outcomes with the risks of therapy and discuss their use with the patient. MRD for SRBDs is an option that may be considered and weighed against the risks of other forms of therapy for SRBDs, such as CPAP. Informed consent demands that a patient understands the reasonable risks and benefits involved with each option.

10. Sleep-related movement disorders

These disorders, historically related to dopamine deficiency [40], can disrupt or fragment sleep in a way that leads to symptoms often associated with insomnia and SRBD; hence, these disorders may be comorbid with them. They may be present but are often not recognized by the patient; therefore, they may not be reported to the patient's physician or dentist. Sleep bruxism (SB) is the most common of this group of disorders. Because these disorders disrupt, or fragment sleep, OFP may occur or if present may be difficult to manage. SB is the one disorder that is managed by the dentist, most often involving the use of an oral appliance. The specific oral appliance may be impacted by the presence of a SRBD [4, 40].

(A) Clinical Features/Symptoms.
  (a) Restless Leg Syndrome.
    - A clinical diagnosis based on anamnestic reporting by the patient.
    - Difficulty keeping the legs calm with the feeling of the need to move them especially prior to or at the time of initiating sleep.
    - Symptoms reported: crawling sensation—achiness.
  (b) Periodic Limb Movement Disorder.
    - Diagnosed during a sleep study (PSG).
    - May cause disrupted (disturbed/fragmented) sleep.
    - Patient may or may not be aware of this condition.
    - May be reported by the bed partner.
  (c) Sleep Bruxism (SB) (G47.63): Defined in two ways according to dentistry or in medicine [31, 40].
    - Dentistry defines this as behavioral disorder.
    - In sleep medicine (ICSD-3), it is a movement disorder–akin to RLS and PLMD.

(B) Screening and Testing.
  (a) Screening: based initially on reported symptoms.
    - RLS: purely clinical diagnosis based on patient reports/symptoms.
    - PLMD: Symptoms of restless sleep or leg activity from bed partner reports.
    - SB: Based on patient or bed partner reports—clinical findings (worn teeth) or masticatory muscle pain/tenderness.
  (b) Testing.
    - RLS: no definitive testing–symptom/history based.
    - PLMD: found on PSG—part of the overall recording—not determined or diagnosed with HSAT.
    - SB: Definitive diagnosis based on PSG with audio/visual recording—three levels of defining SB [40].
      - Possible diagnosis: based on self-reports, history, and questionnaires.
      - Probable diagnosis: combines possible diagnosis with clinical findings (tooth wear, dental findings, and reports of SB by patient/bedpartner).
      - Definite Diagnosis: based on objective testing (PSG).

(C) Management—Role of the Dentist.

(a) RLS and PLMD.
- Look for other comorbid sleep disorders.
  - Insomnia.
  - Sleep Apnea.
  - SB.
- May be related to low blood iron and ferritin levels [40].

The fields of dentistry and sleep medicine view the etiology of SB differently. Complicating this matter, it has been acknowledged that OSA, SB, and GERD mutually interact, but presently this relationship is not a cause/effect relationship [24]. Currently, there are no best practices for managing SB. Dentistry views SB as a behavioral disorder, and medicine views it as a movement disorder. Therefore, management approaches are vastly different [31, 40]. In dentistry, nightguard therapy is advocated to minimize dental and periodontal trauma; occlusal orthotic therapy is utilized in OFP management; OAT is used for SRBD; and referrals are made for evaluation and management of psychological comorbidity. Medicine focuses on sleep apnea management, medication use, management of GERD, and psychological therapy.

For those clinicians who diagnose and manage OFP conditions, it is important to be aware of and screen for the presence of these disorders as they may impact OFP by virtue of sleep disruption, poor sleep quality, or lack of adequate sleep. Also, beware that SSRIs have been reported to increase muscle hyperactivity [41, 42].

11. Central disorders of hypersomnolence

These disorders are not typically ones that the dentist will manage. In the presence of OFP, these disorders may impact sleep and, therefore, impact the outcome of the OFP management plan. Being aware of these disorders, recognizing their signs and symptoms, and being able to assist with the appropriate care is important in a variety of ways. Symptoms of hypersomnolence consist of daytime sleepiness, non-refreshing sleep, the need for naps, inability to stay awake or alert during the day, urge to sleep, and periods of drowsiness.

(A) Types of hypersomnolence disorders [4].
(a) Narcolepsy Type 1 (with cataplexy) and Type 2 (without cataplexy).
(b) Idiopathic Hypersomnia.
(c) Kleine-Levin syndrome.
(d) Hypersomnia related to Medical Condition.
(e) Hypersomnia related to Medication or Substance.
(f) Hypersomnia related to Psychiatric Disorder.
(g) Insufficient Sleep Syndrome.

(B) Clinical Features/Symptoms: The focus here is on the relationship between each condition and pain.
(a) Narcolepsy Type 1.
- Sudden sleep attacks associated with loss of muscle tone with weakness (cataplexy).
- Associated with emotional situation (laughter/excitement).
- Symptoms of sleepiness may suggest a SRBD.
- Headaches common: Migraine and Tension-Type [43].
- Obesity.
- Deficiency of the neurotransmitter Orexin (Hypocretin) [4, 43].

(b) Narcolepsy Type 2.
- Daytime sleepiness and increased REM sleep on PSG/MSLT.
- Seen at earlier age (pediatric)—may be confused with OSA.
- No cataplexy or loss of muscle tone.
- Obesity.
- May have history of head trauma or unspecified viral illness.
- Deficiency of the neurotransmitter Orexin (Hypocretin) [4, 43].
- May progress to Type 1.

(c) Idiopathic Hypersomnia.
- Excessive daytime sleepiness (EDS) and no cataplexy reported.
- Rule out a SRBD/OSA.
- Symptoms suggest dysfunction with autonomic nervous system associated with headache.
- Symptoms of Raynaud's phenomenon and vasculitis.
- Need to rule out other conditions in this category.

(d) Kleine-Levin syndrome.
- Recurring episodes of severe hypersomnolence.
- Associated with behavioral issues: cognitive and psychiatric.
- No known association to pain.

(e) Hypersomnia related to Medical Condition.
- May be related to sub-optimal management of OSA.
- May be associated with fatigue (Insomnia) and depression.

(f) Hypersomnia related to Medication or Substance [21].
- Typically related to sedating medications—as it relates to pain consider benzodiazepines, opioids, anticonvulsants, anticholinergics, antidepressants, antihistamines [21].
- Withdrawal from stimulant medications increase symptoms.

(g) Hypersomnia related to Psychiatric Disorder.
- May spend excessive time in bed.
- Are subject to mood disorders.

(h) Insufficient Sleep Syndrome.
- Inadequate amount of sleep by report.
- Compromised executive function (lack of alertness).

These disorders may impact sleep quality and/or sleep efficiency as well as total sleep time, all of which can impact OFP, whether by the presence of these conditions or with effective management. Therefore, the role of the dentist is to be aware of the existence of these conditions, to screen for possible sleep disorders, and to be willing to question clinical symptoms or complaints that vary from reported testing results. If any of these conditions are believed to be present, referral to a sleep medicine physician is indicated.

12. Circadian rhythm sleep-wake disorders

Circadian rhythm sleep-wake disorders are disorders that do not conform to the 24-hour biological rhythm that exists and are related to the light-dark cycle. In general, these disorders impact the ability to control pain and lead to hyperalgesia that is associated with alterations in melatonin and cortisol levels associated with inflammation [6, 7, 9]. Shift Work disorder and Jet Lag disorder may impact pain thresholds, like insomnia.

These disorders are becoming more common because of modern lifestyles and increased electronic screen time. Blue light, associated with these devices, impacts normal sleep cycles because it alters the timing release of melatonin from the pineal gland, much like occurs with seasonal affective disorder. However, the effect of electronics is not well recognized because the use of cell phones, televisions, and portable electronic devices is so prevalent. With their impact on sleep, the risk for painful conditions, especially head pain, may be more common.

Generally, the role of the dentist involves patient education about common problems with sleep hygiene that may contribute to these disorder, screening for comorbid sleep disorders, and making appropriate referrals to the primary medical provider or to a sleep medicine physician for appropriate management. One example of an intervention that the dentist may be able to assist patients suffering from these disorders is providing education about "social" jet lag—a condition where there is misalignment of the biological clock and self-imposed schedule (*e.g.*, variation of sleep pattern on the weekend compared to the weekday) [44].

(A) Circadian rhythm disorders of most importance to the OFP practitioner include [4].
  (a) Delayed Sleep-Wake Phase Disorder.
    - Onset of sleep is delayed beyond what might be viewed as appropriate (usually by 2 hours).
    - Difficulty falling asleep—resulting in difficulty to awaken as may be desired or necessary, such as early school start times.
    - Is typical in adolescence and younger age group.
    - May be associated with stimulation prior to initiation of sleep, such as bright light and use of technology.
  (b) Advanced Sleep-Wake Phase Disorder.
    - Sleep onset occurs earlier than might be appropriate (usually by 2 hours or more).
    - Most common in older individuals.
    - Often results in early awakenings (morningness) and may be associated with attempts to continue to sleep, thus resulting in insomnia.
    - May be prone to use of alcohol, sedatives, or stimulants.
  (c) Irregular Sleep-Wake Rhythm Disorder.
    - Irregular sleep-wake cycle.
    - May be associated with neurodegenerative disorders (dementia).
    - Consider time in bed (abnormal is greater than 8 hours) or chronic fatigue.
  (d) Shift Work Disorder.
    - May be associated with symptoms of excess sleepiness or insomnia occurring during work that is during the normal time for sleep.
    - Total sleep time may be reduced by up to 4 hours.
    - History of fatigue is possible.
    - Reports exist on the impact of this on health-related consequences especially some cancers.
  (e) Jet Lag Disorder.
    - Associated with travel across 2 or more time zones.
    - May have reduced total sleep time.
    - Typically, worse when traveling east.
    - Typical symptoms.
      - Insomnia.
      - Reduced alertness.
      - Gastrointestinal (GI) symptoms.

(B) Clinical Features/Symptoms.
  (a) Headaches (See Table 3) [21, 43].
    - Specifically: Hypnic headaches (aka "alarm clock headaches").
    - Migraine headache more prevalent when going from Standard time to Daylight Savings time.
    - Tension-type headaches.
  (b) Musculoskeletal pain: more prevalent with altered circadian rhythm.
  (c) More risk for SB.

(C) Screening/Testing.
  (a) Screening: primarily by history.
    - Especially present in shift work and jet lag.
    - Look for symptoms of other sleep disorders (Insomnia, OSA, Narcolepsy).
  (b) Testing: no specific testing.

13. Parasomnia

Parasomnia events occur at sleep onset, during sleep, or upon awakening. These may impact the individual, the bed partner, or both. They occur during REM or NREM (Non-rapid eye movement) and there are events specific to each stage. The main impact is disrupted/fragmented sleep as well as health issues and psychosocial effects. There can be effects on the autonomic nervous system that can impact orofacial pain. The patient may present with a pain or headache complaints that impact their quality of life. Being able to uncover a parasomnia that they or the bed partner has can be impactful in managing their pain complaint. Therefore, the role of the dentist is to be aware of the potential for these disorders and to screen for sleep disorders in general, to provide appropriate patient education about the existence of these disorders, and to make appropriate referral to a sleep physician or to the primary medical provider for appropriate diagnosis and management.

(A) Specific parasomnia disorders contributing to OFP are [4].
  (a) NREM-Related.
    - Sleepwalking.
    - Sleep Terrors.
    - Sleep Related Eating Disorder.
  (b) REM-Related.
    - REM Sleep Behavior Disorder (RBD).
      - Dream enactment.

- Loss of muscle atonia.
- Risk for injury (self or bed partner).
- May be associated with anti-depressants, SSRIs, Beta-blockers [4, 21].
- Typically, in older people (males >50).
- May signal risk for future neurodegenerative disease [4, 21].

- Nightmares.

(c) Others.

- Exploding Head Syndrome.
  - Occurs at initiation of sleep or when awakening during the night.
  - Accompanied by drowsiness at sleep onset.
  - Accompanied by abrupt arousal.
  - Need to rule out Hypnic headache, which is relieved by caffeine [43].
- Sleep Related Hallucinations.
- Due to Medical Disorder: RBD is most common.
- Due to Medication/Substance.

(B) Clinical considerations.

(a) Chronic pain, sleep apnea, or movement disorders that fragment sleep can trigger.

(b) Associated with high stress, post-traumatic stress disorder (PTSD), anxiety, and depression.

(c) Potential for secondary harm (sleepwalking and RBD).

(C) Screening/Testing.

(a) Screening is by history.

(b) Testing.

- HST: inadequate for identifying parasomnias.
- PSG.
  - RBD—also may see PLMD.
  - Eating disorder during sleep.
  - If other sleep disorder suspected (*e.g.*, OSA, COMISA, PLMD).

14. Medications and sleep

OFP practitioners often will encounter patients who are on medications or for whom prescribing a medication is indicated. Accordingly, it is necessary to understand the impact the specific medication may have on sleep as well as the sleep architecture. The following is a summary from a wide variety of sources, that is by no means all-inclusive, that describes these effects on the most common medications in OFP and other medications commonly encountered (Table 4) [21, 41, 42].

(A) Miscellaneous Comments about Medications and Sleep [21].

(a) Triptans: Somnolence but no universal impact on sleep architecture. May impact Circadian Rhythm. Improved headache status may improve sleep and when headache present sleep may be impacted.

(b) Botox: No reported impact on sleep architecture. May help sleep by ↓ sleep bruxism, muscle pain and nocturia. Actigraphy shows ↓ TST possible.

(c) Migraine medications that target CGRP (Calcitonin gene-related peptide).

- No studies addressing sleep architecture.
- Do not cause Insomnia.
- ↑ SQ and ↑ SE directly relates to ↓ headaches.
- Possible side effects;
  - Sleepiness.
  - Fatigue.

(d) Diphenhydramine (Benadryl).

- ↓ SL.
- ↑ SE.
- No effect on.
  - N3 sleep.
  - REM.
  - REM latency.

(e) Hydrochlorothiazide (HCTZ).

- Improves overall sleep.
- ↓ OSA incidence.

(f) PPIs for GERD: reduced reflux leads to improved sleep and ↓ WASO.

(g) Savella (for Fibromyalgia).

- ↓ SL.
- ↑ Wake (Insomnia).
- ↓ NREM.
- ↓ REM.

(h) Levothyroxine.

- No effect on sleep architecture.
- People with hypothyroidism → resolves sleep disturbance.
- Improves daytime sleepiness.

(i) Statins: ↓ WASO.

(j) Melatonin: As an OTC product that is the only hormone sold in the USA, it does not require a prescription. Melatonin is primarily secreted by the pineal gland in the brain and is stimulated by the absence of natural light via the retinal-hypothalamic tract. It is also found in ovaries, testes, the gut, and bone marrow. It is synthesized from tryptophan, which is why eating a large portion of turkey commonly leads to drowsiness. Other facts related to Melatonin are.

- Dosing is variable: The typical dose is between 2 and 10 mg but may be recommended up to 50 mg.
- It does not induce sleep, but it impacts the brain by increasing the impact of neurotransmitters, in particular GABA (Gamma-aminobutyric acid).
- It helps to stabilize sleep architecture, stabilizes REM, and improves N3 sleep.
- By user report, it has a positive impact on sleep quality.
- Purity is variable and dependent on the manufacturer.
- The route of administration may be via tablet, chewable gummies, or by sublingual liquid.
- There is no currently established dosing schedule, but based on its mechanism of action, it is most logical to advocate dosing at dusk to mimic and enhance its natural release in the body.
- It is advocated as an adjunctive therapy for improving symptoms of disorders like insomnia, RBD, and circadian rhythm disorders.
- It has anti-inflammatory and antioxidant properties.

TABLE 4. Common OFP medications and their effect on sleep.

Medication for OFP and Headache	Sleep Latency	Sleep Efficiency	Effect on N3 sleep	Effect on REM	Comments
NSAIDs, aspirin, and ibuprofen in healthy subjects	↑	↓ More awakenings	Delays onset		If pain present ↑ perceived sleep quality ↓ Melatonin levels indomethacin may ↑ SRBD
Opioids (for pain)		None or may ↑	↓	↓ (dose dependent)	↑ sleep disordered breathing ↑ N2 sleep No effect on TST ↓ WASO Respiratory depression—↑ risk for CSA
TCAs	↓	↑	↑	↓ ↑ REM latency	↑ N2 sleep ↓ arousals ↓ WASO amitriptyline ↑ TST nortriptyline ↓ TST
Benzodiazepines	↓	↑	↓	↓ or none	↑ stages N1 and N2 ↑ TST ↑ sedation clonazepam: ↓ SWS ↓ REM
Antiepileptics (in general) Carbamazepine/ oxcarbazepine	None	None ↓	↑	↓ More with Tegretol	pregabalin ↓ SL ↑ SWS and may ↓ REM ↑ WASO ↑ TST with oxcarbazepine ↓ N1 sleep
Gabapentin	↓	↑	Big ↑	↑	Daytime sleepiness
Muscle Relaxers	↓ usually	↑	↑	↑	↑ TST ↓ WASO Above mainly for cyclobenzaprine & baclofen ↑ Drowsiness D/C use ↑ Insomnia
Corticosteroids	↑	Disrupts Sleep	↑	↓	↑ Insomnia ↑ WASO

This table summarizes common medications and how they impact sleep.

15. Sleep disorders–GERD–periodontal disease–TMD–sleep bruxism: the multi-disorder relationship

A relationship has been recognized between wear patterns on teeth and what may happen during sleep that contributes to this. These are:

(A) Attrition is the wear of occlusal tooth surface(s) by parafunctional activity resulting in repetitive eccentric movements of the tongue or teeth. Attrition results in smooth, flat, wear facets.

(B) Erosion is the dissolution of tooth structure and restorative material by acids from either diet or from gastroesophageal reflux disease (GERD); erosion results in negative cup-shaped lesions, which may occur within flat wear facets or on the axial surface of teeth.

(C) Abfraction is the flexing of teeth that occurs by parafunctional activity resulting in repetitive eccentric movements of the teeth; it results in characteristic chevron-shaped cervical lesions, facial bone dehiscence, and widened radiographic periodontal ligament space.

It has generally been observed that these phenomena seem to appear in people who have been diagnosed with OSA, hence the assumption is easy to make that obstructive sleep apnea is a likely cause of GERD and bruxism. While the available data does not support a causative relationship but rather a co-morbid relationship, recognizing the signs of erosion, attrition, abfraction, and risk factors for obstructive sleep disorders is an essential role of the orofacial pain specialist so that timely referrals to the appropriate medical professionals may be made. GERD can be both symptomatic and asymptomatic (silent) which requires vigilance for identifying related findings such as sore throat, dry cough, and even nasal airway restriction that leads to mouth breathing.

One of the often-overlooked related aspects of sleep disorders is the nasal airway. Nasal breathing is critical to

good sleep and overall health. Nasal airway restriction or obstruction leads to poor oxygen levels, to compensatory mouth breathing and poor sleep. The nose is the carburetor of the body. With nasal breathing, nitric oxide levels are enhanced, which facilitates absorption of the inspired oxygen in the lungs. It is critical that nasal breathing and airway be evaluated as part of the clinical evaluation.

Periodontal disease may be a significant recognizable factor that is related to GERD and SB, both of which may impact sleep quality. This has been shown to be more prevalent in people with a SRBD and especially sleep apnea (OSA). Periodontal disease is also related to a variety of health issues that are related the oral and gut microbiome and are also associated with chronic inflammation [45]. Sleep duration may be one of the many related factors. It is generally accepted that optimum sleep is between 7 and 8 hours, but when sleep is less than 7 hours and greater than 8–9 hours, there is a risk for increased morbidity and mortality [46]. There is now evidence that a gut-brain-jaw axis exists that contributes to OFP and TMD, which is mediated by the vagus nerve and affects the trigeminal nerve [45]. More recently, there has been an increased interest in the microbiome and its impact on health and disease related to sleep medicine; however, it is beyond the scope of this document to go into detail regarding this because it is a developing topic deserving of much more detail and understanding than is currently available for publication as a guideline.

Table 5 identifies several of the common clinical findings within the stomatognathic system that have been associated with sleep disorders.

16. Appliances and TMD

Oral appliances (OAs) are commonly used to treat temporomandibular disorders (TMDs). However, the quality of published evidence on the efficacy of OAs to treat TMDs is weak [47]. Anecdotally, there is robust clinical support for their use because many patients seem to have reduced symptoms while wearing OAs. Many studies have been published over the past thirty years, but these clinical studies, systematic reviews, and even meta-analyses have spawned controversy within the dental profession about the efficacy of OAs as a primary treatment modality for TMDs. Favorable outcomes of decreased pain and improved mandibular range of motion are the most published results, primarily when worn during sleep. While the mode of action of OAs in treating TMDs remains unclear, Menchel and colleagues proposed that OAs reduce internal joint pressures for approximately 6–8 weeks to allow for other modalities of a comprehensive treatment plan to take place (self-care, medications, physiotherapy, *etc.*), enable faster return to the mean, disrupt muscle engrams to reduce stress in the system for 6–8 weeks, and serve as a valuable and effective placebo for psychosocial aspects of clinical care. However, historical research on OA therapy is inadequate because most of the studies regarding OA therapy have been done for the treatment of a generalized diagnosis of "TMD" or "jaw pain", which are not adequate terms for direct targeted therapy [48]. "TMD" is not a diagnosis but a category of clinical problems affecting the mandible, temporal fossa, and supporting soft tissues. In recent years, the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) is now considered to be the standard for diagnosis of TMDs, albeit not an all-inclusive categorization of all diagnoses affecting the temporomandibular joints; however, it is a standardized framework that can be used effectively for communicating diagnoses within the research and clinical realms of practice that takes into account the biopsychosocial model of pain management, where Axis I addresses biomechanical diagnoses and Axis II provides consideration for psychosocial factors in the pain experience [49].

The long-term impact of continuous OA wear remains a subject of controversy because it has the potential to change occlusal and craniomandibular relationships, depending on the physical features of the OA utilized. Because there is variation in the designs of OAs and because specific design features have not been adequately researched relative to a common categorization of TMD conditions, it is unclear which kind of OA is most appropriate for treating any particular TMD (as defined within the DC/TMD framework), which leads to the conclusion in Cochrane-level reviews that there is "weak evidence" for the efficacy of OA therapy. Many years of anecdotal reports and experience of skilled clinicians, however, support the judicious use of OA therapy, despite the lack of published evidence. The available literature supports the use of OAs in the management of three common TMD conditions: myalgia of the muscles of mastication, TMJ arthralgia, and degenerative joint disease (DJD) [48].

OAs provide a conservative and relatively reversible and non-pharmacological treatment approach with minimal adverse effects when used with reasonable appliance designs and protocols. Generally, passive appliances (without directive ramps and features intended to direct specific forces) do not produce permanent anatomic changes in the stomatognathic system. As part of a comprehensive treatment plan that may include other therapeutic modalities such as physiotherapy, cognitive behavioral therapy, patient education, and self-care, OAs are a pragmatic first line therapeutic option for TMDs that may be prescribed by orofacial pain specialists as well as by appropriately trained dentists [50].

OA designs are described as active or passive [33, 48]. Active OAs have features that are designed to direct forces in a certain manner to reposition the mandible or to direct functional movements in some restricted movement. One common example of active appliances is the mandibular repositioning appliances used to treat obstructive sleep apnea. With these appliances, however, the mandible is restricted to an open and protrusive posture that usually forces the condyle downward and forward along the articular eminence with mechanical elements and is stabilized by both maxillary and mandibular arches. Another active appliance is the so-called "neuromuscular orthotic" that is designed to train the mandible to function in a specific position based on measured musculature positions, and this appliance is typically retained on one of the dental arches. While other types of appliances that are intended to be passive in nature with flat platforms that allow for passive and even contacts in a reproducible mandibular closing position (sometimes referred to as the "tap-tap" position because of the instructions given to the patient

TABLE 5. Stomatognathic observations correlated with sleep disorders.

Clinically observable signs and possible sleep correlation			
Observation	Etiology	Clinical Sign	Potential Sleep Relationship
Abfraction	Dynamic Bruxism	chevron-shaped lesions at the cemento-enamel or crown margins that are wide vertically at the external aspect and taper to an internal point; widened periodontal ligament space; facial dehiscence of bone	Bruxism increases muscle support of airway and moves mandible to minimize constriction; occurs after apneic or hypopneic event (timing is not consistent and reproducible)
Attrition	Dynamic Bruxism	flat, shiny wear facets	Bruxism increases muscle support of airway and moves mandible to minimize constriction; occurs after apneic or hypopneic event (timing is not consistent and reproducible)
Erosion	GERD	cupped lesions that are broad and rounded at the base that may be present on the occlusal, facial, or lingual surfaces that have a matte appearance; alternative appearance of lesion may make restorations appear to be "erupting" above their cavosurface margins	Hydrochloric acid is released in GERD prominently during sleep
Vertical Dental Fractures	Static Bruxism	Vertical cracks	Buttressing of the teeth during sleep may provide airway stability
Myalgia	Static or Dynamic Bruxism	Jaw pain and/or mild-moderate pressure-type headache upon waking	Supporting the airway during sleep
Chronic Pharyngitis	GERD	Dry cough, halitosis, elongated and/or inflamed uvula—coated tongue	Tissue response (inflammation) to GERD
Hyperactive Gag Reflex or loss of the gag reflex	GERD	Hypersensitivity to anything being placed in contact with the palate except for food; no difficulty swallowing Loss of gag reflex found on examination	Hypersensitized pharyngeal tissues due to chronic inflammation Loss of gag reflex associated with neurologic ability to control soft palate and uvula

An overview of possible observed conditions and their clinical presentation that may indicate related risk for a sleep disorder.

when marking the appliance) can inadvertently be made to be active in excursive movements. For example, if there are anterior ramps built to provide immediate disocclusion of the posterior teeth in excursive movements ("canine guidance" or "anterior guidance") and the ramps are steeper than the natural envelope of function for the patient, then this appliance becomes an active appliance. Another example may be the soft or soft/hard laminated varieties of OAs that are commonly made today under the guise of improved comfort. While the occluding surface of these appliances may be flat or adjusted to be flat, when the patient clenches, they may distort and cause directive, uncontrolled micromovement of the mandible; therefore, these types of OAs are more active than passive. Any active appliance can increase muscle activity and bruxism [48].

Predictably passive OAs are designed so the biting surface is relatively flat relative to the occlusal plane to allow freedom of excursive movement without eccentric interference. Depending on the individual anatomy of the patient, this may result in balanced excursive contacts in the anterior only or with some posterior eccentric contact that transitions through lateral excursion into anterior crossover contact.

In general, the passive appliance with the most reported success, anecdotally and academically, is a hard, rigid flat plane appliance characterized by a flat occluding surface with equal bilateral contacts adjusted to provide shallow anterior guidance with smooth incisor crossover contact or group function posterior excursive contacts. This type of appliance is typically fabricated on one arch, but some clinicians choose to use a dual-arch design especially if the occlusal plane of the opposing arch is irregular. Appliances designed in this fashion have been proven to be safe and effective in managing many TMDs and are appropriate for short and/or long-term wear, providing mechanical protection to the joints while avoiding contribution to permanent occlusal changes that occur with the natural processes of adaptation and remodeling of the temporomandibular joints during the progress of degenerative processes. Appliances with these design features are best called stabilization appliances [48].

17. Concluding commentary

Oral, face, and head pain are intimately related with sleep quality and quantity, which is why the recognition of sleep disorders and dental options for management of SRBD is a recognized component of the specialty of OFP. Therefore, competence in dental aspects of sleep medicine is a required component for graduation from OFP residency programs. In fact, it is the only recognized dental specialty within the United States that includes detailed training in SRBD diagnosis and management in its curricula for accreditation by the Committee on Dental Accreditation (CODA) of the ADA [31, 51]. Other organizations may provide training in dental sleep medicine in various depths and may offer accolades documenting that various levels of training have been completed. However, there is no officially recognized dental specialty, in the United States at least, for dental sleep medicine (DSM). Legitimate OFP specialists—those who have met the criteria for specialty by either having graduated from an accredited orofacial pain residency program and/or have passed the rigorous certification process to become a Diplomate of the American Board of Orofacial Pain—are wholly qualified to practice dental sleep medicine within their scope of care as OFP specialists.

DSM, as defined by the American Academy of Dental Sleep Medicine (AADSM), has primarily been associated with the recognition and management of SRBD. The definition for this discipline needs to be expanded and updated to include more than just fabrication of a mandibular reposition device for a SRBD. There is need to consider sleep health overall as was discussed in a recent article in the Journal of the American Dental Association (JADA) and is the intent of this manuscript. DSM should include all aspects of sleep medicine that fall within the scope of practice for dentistry, which involves other disorders that may contribute to poor sleep. As was discussed by Lobbezoo *et al*. [31], DSM in the 21st century involves OFP, TMD, SB, GERD, and related insomnia. DSM as practiced by the well-trained dentist needs to move beyond only SRBD and oral appliances [24, 52].

The authors have attempted to share the best available evidence-based information at the time of this publication so that the OFP clinician may have a dependable quick-reference guide to consult as the field rapidly changes. However, other modalities of care are currently being marketed and promoted by various clinicians and for-profit entities that do not have adequate validated evidence currently to rise to the level of best practices for the management of sleep disorders that may show promise in the future but must at this time be viewed as experimental. Most are promoted as stand-alone treatment for obstructive breathing disorders, but this is problematic because of the multifactorial etiology of sleep disorders that have been discussed in this text. Perhaps, these additional therapies may be appropriate as part of a comprehensive sleep disorder management treatment plan involving behavioral management for improved sleep hygiene, weight control, diet, exercise, pain management, *etc.* At the time of publication of this text, a summary of these potential therapies follows:

17.1 Tongue retaining devices (TRD)

TRDs are oral devices that may be considered as an option for managing a SRBD. Currently the original TRD is no longer available and has been replaced with tongue stabilizer devices that are readily available and are not typically fabricated by the dentist. To some degree, they also reposition the mandible because the patient needs to open (vertical repositioning) and will advance the mandible so that the tongue can be inserted into the bulb-like part of the device that holds the tongue forward. Their overall efficacy is variable due to comfort.

17.2 GLP-1 (glucagon-like-peptide-1) receptor agonist therapy

This has become a popular therapy designed for weight loss, particularly when the BMI >30, which in turn may be helpful in the management of type 2 diabetes. The medication enhances appetite control and food intake by slowing gastric mobility. The reported effect is reduction in initial body weight by 15 to 20%, and reduction of the A1C has been observed with some of these medications. One brand-name drug (Zepbound®) has obtained FDA approval in the United States for weight loss and managing OSA. FDA approval means simply that a drug or device is safe for the purposes for which it is being marketed. The generic form of this drug is tirzepatide, which is also marketed under the brand name Mounjaro®, currently FDA-approved for type 2 diabetes. These medications should not be used as a stand-alone therapy for obstructive sleep disorder management but are most effective when combined with exercise, nutritional counseling, and behavioral changes. Added benefits may be to lower cholesterol, to reduce risk for CVD, and to reduce the risk of other health concerns associated with obesity [52].

17.3 Myofunctional therapy

This therapy focuses on proper tongue posture and positioning. This is best provided by a therapist trained in its use and who understands the relationship of tongue function to speech, breathing and swallowing. This is often best utilized in combination with other management strategies especially as it relates to OSA.

17.4 Tongue-tie surgery (frenectomy)

This is utilized in instances where tongue movement and posture are restricted and may be helpful in some cases. However, the research is inadequate, and outcomes are mostly anecdotal. This procedure should only be performed after a clinical evaluation is completed that leads to a medical sleep diagnosis unless there are other reasons to increase tongue mobility (*e.g.*, inadequate ability to latch when nursing, some speech impediments, significant risk of trauma, *etc.*). Currently, the technique is commonly done with a laser of various wavelengths, and laser manufacturers often tout one wavelength over another as a sales technique. Once a frenectomy is completed, tongue exercises or myofunctional therapy are important to prevent relapse and re-tethering of the tongue. If any speech issues are detected, a consult with a speech therapist is indicated.

17.5 Mouth taping

This has been advocated as a means of reducing mouth breathing and to enhance nasal breathing. Recent studies show that this is not the panacea it once was. Better methods to improve nasal breathing and reduce or eliminate mouth breathing are available [52].

17.6 Hypoglossal nerve stimulation (HGNS)

Currently, Inspire® is the most recognized device in this category. It is designed to activate the Hypoglossal nerve (CN XII) to move the tongue forward and reduce the risk for airway restriction. The device itself is placed in the anterior chest region like a cardiac pacemaker with a stimulating lead wire attached to the hypoglossal nerve in the floor of the mouth. The protocol indicates that DISE (drug induced sleep endoscopy) needs to be done prior to the surgery to determine that this procedure has a favorable prognosis. This is typically indicated for moderate to severe sleep apnea. Unfortunately, OAT (MRD) therapy, a much less invasive option, is usually not attempted prior to this surgery but may be just as effective without associated surgical risks. In fact, there are no long-term (>10 years) studies on this therapeutic option [52].

17.7 eXciteOSA® tongue therapy

This is a device that uses NMES (neuromuscular electrical stimulation) and is intended to improve tongue strength to prevent the tongue base from collapsing and, thus, obstructing the oropharyngeal airway. It is indicated for snoring and mild OSA. It is used for 20 minutes daily for 6 weeks and then twice a week. Limited studies indicate a 50% reduction in the AHI and ODI as well as reduced ESS scores [52].

17.8 Upper airway resistance syndrome (UARS)

This no longer exists as a diagnostic classification in the ICSD-3.3 UARS is unique as a SRBD in that the signs and symptoms are often like OSA, often with the lack of good nasal breathing, with one exception; there is no significant oxygen desaturation. This may be recognized in children and in pre-menopausal females. Since anecdotally patients are known to present with radiographic, physical, and audible signs and symptoms that mimic those diagnosed with OSA, currently this phenomenon might best be described as sub-clinical OSA.

18. Glossary of terms

The following terms are commonly used in the literature related to sleep and in discussions related to sleep and orofacial pain:

- Advanced Sleep Phase Syndrome: The sleep-wake times are earlier than the accepted times thus resulting in earlier bedtimes and earlier awakenings.
- Alpha Intrusion: The appearance of alpha waves in a stage of sleep where they are not normally seen. Typically seen during N3 sleep and referred to as alpha-delta sleep. Typically associated with pain.
- Alpha Waves: Typical EEG waveform associated with the wake state.
- Anticholinergic: Term refers to substances that block the action of acetylcholine (Ach). Blocks the action of the parasympathetic nervous system, often associated with reduced saliva production, and may cause sedation.
- Apnea: Historically defined as the total cessation of breathing for 10 seconds or longer. In sleep medicine it is a reduction or cessation of 70% or greater in airflow for 10 seconds or more.
- Apnea Hypopnea Index (AHI): The average number of apneas and hypopneas per hour of sleep.
- Arousal: An abrupt or sudden change from a deep stage of sleep to a lighter plane of sleep or from REM sleep to wakefulness.
- Atonia: Loss of muscle tone–usually associated with REM sleep.
- Cataplexy: A finding often found in Narcolepsy. A sudden loss of muscle tone associated with an emotional event such as laughter, surprise, excitement or even anger.
- Cognitive Behavioral Therapy (CBT): A form of psychotherapy designed to reduce negative thoughts and behaviors to improve quality of life and sleep quality.
- Circadian Rhythm: Often times referred to as one's internal clock. Associated with the light dark cycle and occurs over about a 24-hour period.
- Co-Morbid Insomnia and Sleep Apnea (COMISA): The co-occurrence of insomnia and a SRBD, typically sleep apnea, on a nightly basis.
- Deep Sleep: Synonymous term to as Delta Sleep or Restorative Sleep, this is associated with delta waves on an electroencephalogram (EEG). In a sleep study, it is known as stage N3 (previously termed NREM stage 3 and 4). In some situations, deep sleep is associated with or thought to be REM sleep because the ability to be awakened is more difficult.
- Delta Sleep: Synonymous term to as Deep Sleep or Restorative Sleep, this is associated with delta waves on an electroencephalogram (EEG). In a sleep study, it is known as stage N3 (previously termed NREM stage 3 and 4). In some situations, deep sleep is associated with or thought to be REM sleep because the ability to be awakened is more difficult.
- Delayed Sleep Phase Syndrome: The sleep-wake times are delayed making it difficult to fall asleep at the normal times and awakenings are later.
- Drowsiness: A state of quiet wakefulness that is seen or experienced prior to sleep onset.
- Entrainment: This is how environmental variables influence the intrinsic pacemaker (circadian rhythm). It is the optimal synchronization of the sleep-wake cycle by light.
- Epoch: Refers to the amount of time that sleep is being recorded during a sleep study, typically a 30 second duration.
- Excessive daytime sleepiness (EDS): Reported difficulty staying awake during times of wakefulness.
- Fatigue: The feeling of low energy levels, listlessness (feeling weary), or low motivation to be active and may impact performance; the ability to fall asleep when inactive or resting may be difficult.

- Fragmentation: The presence of an interruption of any stage of sleep by the appearance or presentation of a different sleep stage or by the onset of wakefulness.
- Free Running Rhythm: The loss of control and synchronization of the sleep-wake (circadian) cycle where the sleep- wake cycle continues to be regular but is greater than 24 hours.
- Gamma-Amniobutyric Acid (GABA): The main brain neurotransmitter associated with sleep.
- Hypercapnia: An elevation of CO_2 levels in the blood.
- Hypersomnia or Hypersomnolence: Excessive sleepiness or Excessive Daytime Sleepiness (EDS) where the feeling of sleepiness exists at times when an individual should not be sleepy and can also be associated with prolonged sleep.
- Hypnagogic Hallucinations: Images that may be visual, auditory or tactile that occur at the onset of sleep and may be related to the onset of REM.
- Hypnopompic Hallucinations: These are events that occur at a time when there is a transition from sleep to wakefulness.
- Hypocretin (also known as Orexin): A neurochemical associated with the control of sleep that is often associated with narcolepsy, more commonly when cataplexy is present.
- Hypoxemia: Low blood oxygen that is measured by oximetry.
- Hypoxia: Reduced blood oxygen's impact on the body and organs of the body.
- Hypopnea: A reduction in airflow by 30% or greater associated with a similar reduction in thoracoabdominal movement and a 4% or greater fall in the blood oxygen level as measured by oximetry.
- Infradian rhythm: An intrinsic (endogenous) rhythm that is longer than the circadian rhythm, such as the menstrual cycle.
- Insomnia: The inability to initiate or maintain sleep and associated with sleep loss.
- Intermittent Hypoxia (IH): Brief period of hypoxia or drops in the blood oxygen level.
- K-Complex: A distinct sharp EEG waveform specific to stage N2 (NREM stage 2) sleep.
- Light Sleep: Often associated with stage N1 (NREM stage 1) sleep and at times with stage N2 (NREM stage 2) sleep.
- Maintenance of Wakefulness Test (MWT): A daytime test that assesses the ability of an individual to remain awake when reclined in a darkened room. This test is oftentimes used to determine the success of therapy for sleep apnea and the resolution of daytime sleepiness by the ability to remain awake.
- Micro-arousal: Partial arousal from sleep.
- Micro-sleep: An episode that lasts up to 30 seconds associated with the loss of perception of external stimuli; may be associated with excessive sleepiness.
- Montage: This refers to the arrangement of EEG leads on the head for a sleep study.
- Multiple Sleep Latency Test (MSLT): A form of daytime testing consisting of a series of 4 or 5 times (naps) that determine the propensity to fall asleep. This is a test used to test for the risk of narcolepsy and the onset of REM (stag R) within a 15 minute period.
- Nadir: Refers to the minimum value or the lowest point.
- Nightmare: An unpleasant/frightening dream that occurs with REM sleep and may be recalled.
- Night Terror: An unpleasant/frightening dream that is associated with deep sleep (stage N3 or NREM 3 and 4) and are typically not recalled.
- NonREM (NREM) Sleep: Non-Rapid Eye Movement sleep that is comprised of three stages (N1, N2, N3). These stages occur 4 to 6 times during a normal night's sleep.
- Non-restorative Sleep: Perceived as poor sleep–poor quality sleep that results in feeling that sleep is unrefreshing in the morning.
- Noradrenergic: Refers to areas that produce or are affected by norepinephrine (NE), also referred to as noradrenaline.
- Paradoxical Sleep: Synonymous with Rapid Eye Movement (REM) sleep.
- Parasomnia: A disorder of sleep associated with arousal(s) or the transition between different sleep stages. Event that disrupts sleep (*i.e.*, sleepwalking, sleeptalking).
- Periodic Breathing (Cheyne-Stokes Respiration): A type of breathing pattern that has a crescendo-decrescendo type of respiration.
- Periodic Limb Movement Disorder (PLMD): An involuntary movement, usually of the legs, flexion of the foot, or extension of the large toe, that occurs during sleep.
- Pickwickian Syndrome: Descriptive term derived from a character described by Charles Dickens who was obese, snored, was sleepy, and had hypoventilation that applies to a number of different disorders, especially severe sleep apnea.
- Polysomnogram (PSG): A sleep study performed during sleep (overnight) that records multiple physiologic events; most commonly is performed in a specialized facility under direct supervision.
- Rapid Eye Movement (REM): A period of sleep when the eyes typically move rapidly and the body is in a paralyzed state.
- REM Latency: Period of time between sleep onset and the first REM period.
- REM Rebound: Associated with an increased amount or duration of REM sleep. Often seen when the influence to suppress REM sleep is removed such as treatment for sleep apnea or with the discontinued use of an antidepressant.
- Respiratory Disturbance Index (RDI): The Apnea-Hypopnea index (AHI) that also includes the RERAs.
- Respiratory Effort Related Arousal (RERA): A respiratory event that is associated with an arousal (change in sleep state) but cannot be scored on the sleep study as an apnea or hypopnea.
- Restorative Sleep: Synonymous term to as Delta Sleep or Restorative Sleep, this is associated with delta waves on an electroencephalogram (EEG). In a sleep study, it is known as stage N3 (previously termed NREM stage 3 and 4). In some situations, deep sleep is associated

with or thought to be REM sleep because the ability to be awakened is more difficult.
- Seasonal Affective Disorder (SAD): A condition associated with an increased period of darkness (winter) with symptoms of daytime fatigue, mood swings, depression, increased appetite especially for carbohydrates and weight gain.
- Sensitivity: The ability and reliability of a test to identify the presence of a condition or disease.
- Sleep Breathing Disorder (SBD): Generic description of breathing disorders during sleep that usually refers to obstructive sleep apnea (OSA) and snoring.
- Sleep Disordered Breathing (SDB): Generic description of breathing disorders during sleep that usually refers to obstructive sleep apnea (OSA) and snoring.
- Sleep Related Breathing Disorder (SRBD): Preferred generic description of breathing disorders during sleep that usually refers to obstructive sleep apnea (OSA) and snoring that is synonymous with SBD and SDB.
- Sleep Debt: Term used to describe the accumulated effects of recurrent sleep deprivation that leads to feeling sleepy and not rested after sleep.
- Sleep Efficiency (SE): Usually measured in percent and is the amount of recorded time asleep to the amount of time in bed. The normal is 85%.
- Sleep Hygiene: The habits and lifestyle that contribute to favorable sleep, such as room temperature, noise, and the amount of light.
- Sleep Latency: The time between the time a patient attempts to fall to sleep and the presentation of sleep or a given sleep stage such as REM sleep (REM sleep latency).
- Sleep Onset: The amount of time observed on a sleep study that it typically takes to go from wakefulness to N1 (NREM stage 1) sleep.
- Sleep Paralysis: A stage observed between sleep and wakefulness associated with the inability to move (atonia) or to speak that is associated with REM sleep; may be frightening, especially if breathing is also affected.
- Sleep Quality (SQ): The perceived quality of sleep reported by the individual based on hours of sleep, sleep efficiency, and low Wake After Sleep Onset (WASO).
- Sleep Spindles: EEG finding that is associated with N2 sleep and is associated with preventing noxious stimuli from disrupting sleep.
- Sleep-Wake Cycle: The internal clock control of the sleep and wakefulness cycle (Circadian Rhythm).
- Slow Wave Sleep (SWS): Similar term to Delta Sleep, Stage N3 (NREM stage 3 and 4) or Restorative Sleep, that is often used synonymously with those terms but is actually more descriptive because it is associated with slower EEG waves as seen on the sleep study.
- Snoring: The noise associated with respiration during inspiration that is caused by vibration of the unsupported soft tissues in the upper airway (soft palate, uvula and oropharyngeal tissues). This is often associated with incomplete obstruction but may also be a symptom of sleep apnea.
- Somnabulism: Term for walking during sleep, also called sleep walking.
- Specificity: The ability or reliability of a test to determine the absence of a condition or disease.
- Time in Bed (TIB): The actual time a person is recorded as being in bed during a sleep study.
- Total Sleep Time (TST): The total amount of time actually spent in the sleep state (total of REM and NREM sleep).
- Ultradian rhythm: The intrinsic (endogenous) rhythm that is shorter than 24 hours and may be repeated within this time period, such as heart rate, nostril dilation, appetite, body temperature and hormonal release.
- Upper Airway Resistance Syndrome (UARS): Subclinical condition characterized by events that do not strictly meet the criteria for apnea and hypopnea but appear as RERAs on a sleep study that appears similar to sleep apnea, but oftentimes the blood oxygen level does not drop.
- WASO (Wake After Sleep Onset): The amount of time that exhibits the wake state (time awake) after initiating sleep, which optimally would be 30 minutes or less.
- Zeitgeber: German term for "time givers" that is a cue found in the environment (sunlight, social interaction, noise or an alarm clock) that allows an individual to entrain to their circadian (24 hour clock) rhythm.

AVAILABILITY OF DATA AND MATERIALS

Support for the information contained in this handbook can be found within the references provided. Upon reasonable written request from the corresponding author that does not violate applicable copyright laws, supporting literature is available.

AUTHOR CONTRIBUTIONS

DRB—wrote the primary manuscript and conducted the initial literature review. KDH—provided the organizational design of the manuscript, conducted literature review, and provided editorial support. RB—reviewed the manuscript for accuracy, provided editorial support, and provided assistance with project design. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

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The authors declare no conflict of interest. Rafael Benoliel is serving as Editor-in-Chief of this journal. We declare that

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