Chapter 5
Dental Sleep Medicine and the Use of Oral Devices

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Introduction

Approximately, one-third of life is spent sleeping. Sleep is very important to humans; disruption and/or deprivation of sleep typically results in adverse physiological effects. The overall prevalence of sleep problems may be as high as 30 % in children and adults and even higher in elderly people. Obstructive sleep apnea (OSA) is associated with higher risks for HT, coronary heart disease, stroke, congestive heart failure, atrial fibrillation, impotence, mortality, and behavior and cognitive problems [1]. Sleep apnea [2] leading to excessive daytime hypersomnolence may be responsible for many job-related injuries, and it is estimated that people with sleep apnea are ten times more likely to die in a car accident than someone without sleep apnea.

It is also known that adequate sleep is needed to maintain alertness, heal the body, and assist with memory and learning. The physiologic and neurochemical activities of the sleep and awake states need to be better understood. However, the complexity and study of sleep require a comprehensive understanding of the physiology, neuroanatomy, neurochemistry, and associated mechanisms by which these areas interact.

The social and economic impact of sleep disorders is estimated at $16 billion annually for health care expenses and $50 billion annually regarding lost productivity. Sleep disorders are considered one of the most common health problems, and yet it has been demonstrated that between 82 and 98 % of adults with sleep-related breathing disorders (SRBD) are undiagnosed.

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N. Vadivelu et al. (eds.), *Orofacial Pain: A Clinician's Guide*,
DOI 10.1007/978-3-319-01875-1_5,

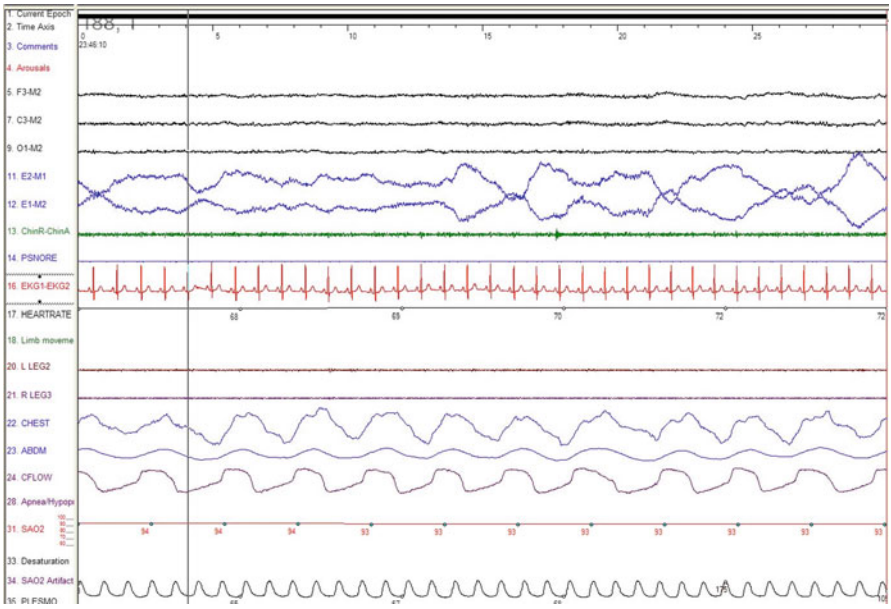

Fig. 5.1 Stage (N1) (courtesy of George Zureikat, MD)

Sleep disturbances can be classified into one of the four groups: insomnia, hypersomnia, parasomnia, and sleep–wake schedule disorders. Insomnia [3] describes a condition in which a patient is tired and desires to sleep, yet suffers from a combination of difficulties involving falling asleep, maintaining sleep, and awakening too early. Hypersomnia, on the other hand, describes a condition in which a patient remains sleepy, usually despite adequate sleep time. Parasomnias are described events occurring during sleep, and sleep–wake schedule disorders (known also as circadian rhythm abnormalities) reflect a situation in which a patient sleeps at undesired times.

Sleep Architect

The discovery of electroencephalography in 1928 by Berger provided a quantum leap for sleep research. In applying the new methods to measure EEG activities in sleeping people, or animals, it was revealed that the transition from wakefulness [4] to sleep is accompanied by specific and well-characterized changes in brain wave activities (Fig. 5.1). EEG has allowed widespread investigations of the brain mechanism controlling sleep and wakefulness [5] by several investigators [4–6].

During sleep, there are periods of physiological and autonomic activation reaching waking levels. EEG and other physiological recordings during sleep define two distinct states of sleep: the time of rapid eye movement (REM) [7] and that of non-REM

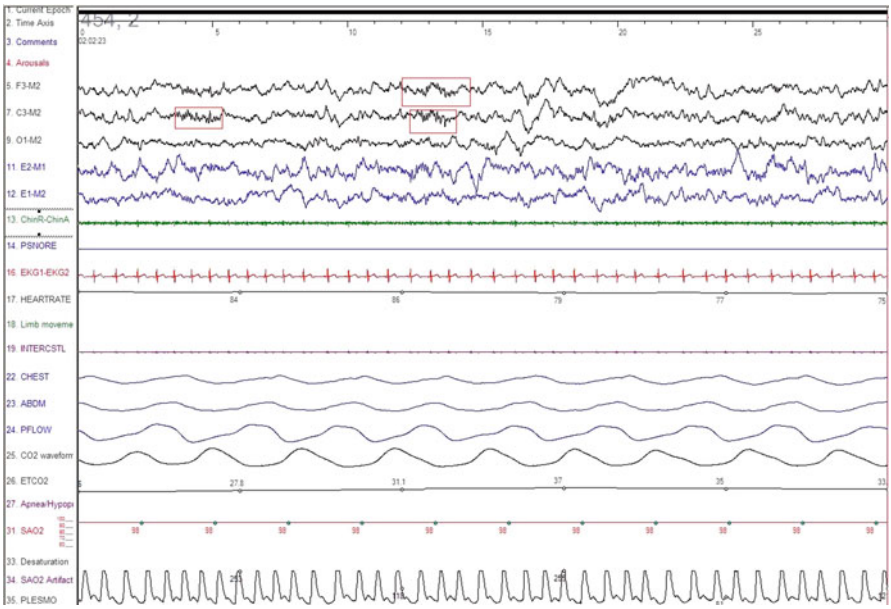

Fig. 5.2 Stage (N2) spindles (courtesy of George Zureikat, MD)

(NREM). The second is divided into four stages: stage (N1) also called light sleep, stage (N2) also called consolidated sleep, and stages (N3) and (N4) also called deep or slow-wave sleep. Division of sleep into these stages relies on electroencephalogram (EEG), electromyography (EMG), and electrooculography (EOG).

The different EEG patterns that are characteristic of NREM sleep stages are shown in Figs. 5.1, 5.2, 5.3, and 5.4. Stage (N1) is characterized by relatively low-amplitude alpha waves and activity intermixed with episodes of alpha activity. In stage (N2) there are K-complex wave forms and sleep spindles, whereas stages (N3) and (N4) are dominated by an increased amount of slow waves and high amplitude of delta activity.

NREM: Historically, this sleep pattern is divided into four distinct stages on the bases of characteristic of brain waves and physiologic activities.

Stage (N1): This stage reflects a change in brain wave activity from rhythmic alpha waves to mixed-frequency waves as the individual passes from wakefulness to the initiation of sleep. Stage (N1) comprises about 2–5 % of the total sleep time, and it is considered to be a drowsy or a light sleep stage from which one can usually be awakened easily. Sudden muscle contractions can occur in this stage, and the individual may also experience a sensation of falling.

Stage (N2): Although this begins to be a deeper stage of sleep with a reduction of heart rate and body temperature, it is still regarded as being light with mixed-frequency EEG activity. He or she can again be easily aroused or awakened, although an additional amount of stimulus is needed as compared to stage (N1). This stage

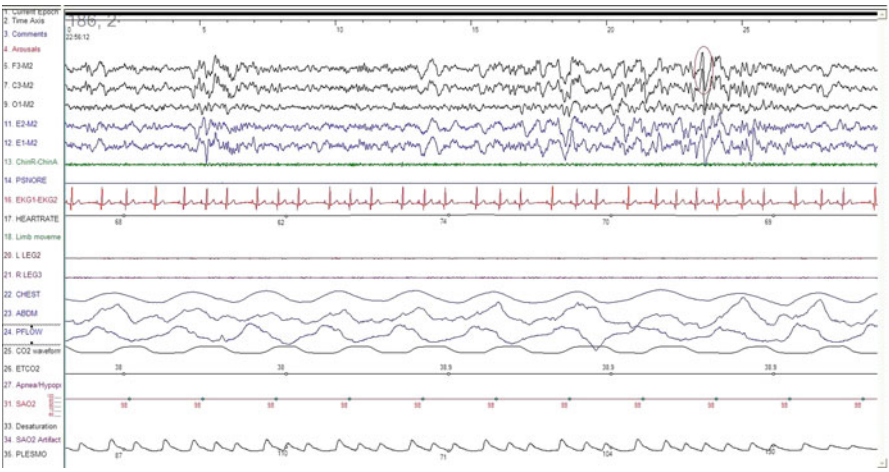

Fig. 5.3 Stage N2 K-complex (courtesy of George Zureikat, MD)

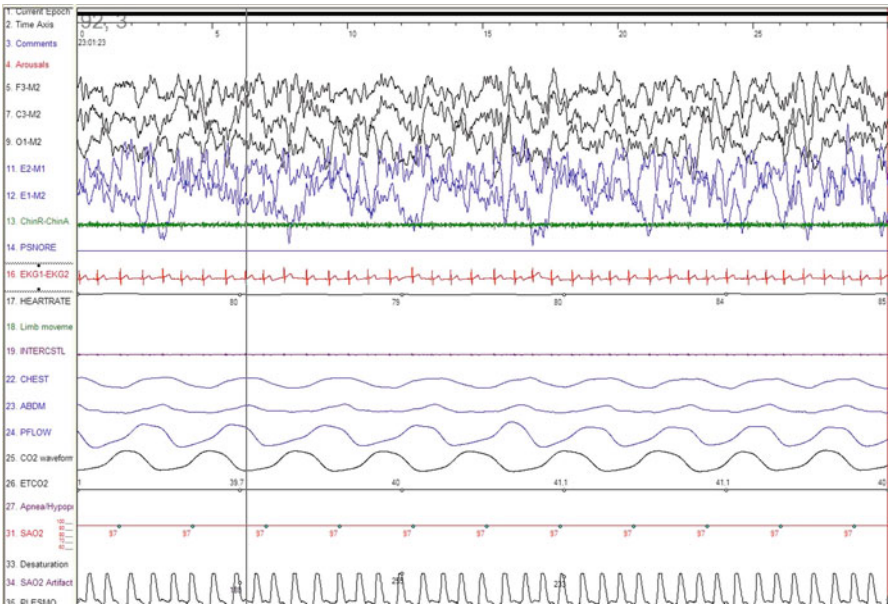

Fig. 5.4 Stage (N3) (courtesy of George Zureikat, MD)

comprises about 45–55 % of the total sleep time. Unique and significant features in the EEG activity of this stage are the presence of K-complex wave forms and sleep spindles, the latter of which have been postulated as being associated with memory consolidation. The K-complex may appear as a result of some type of stimulation, such as noise, or it may appear spontaneously.

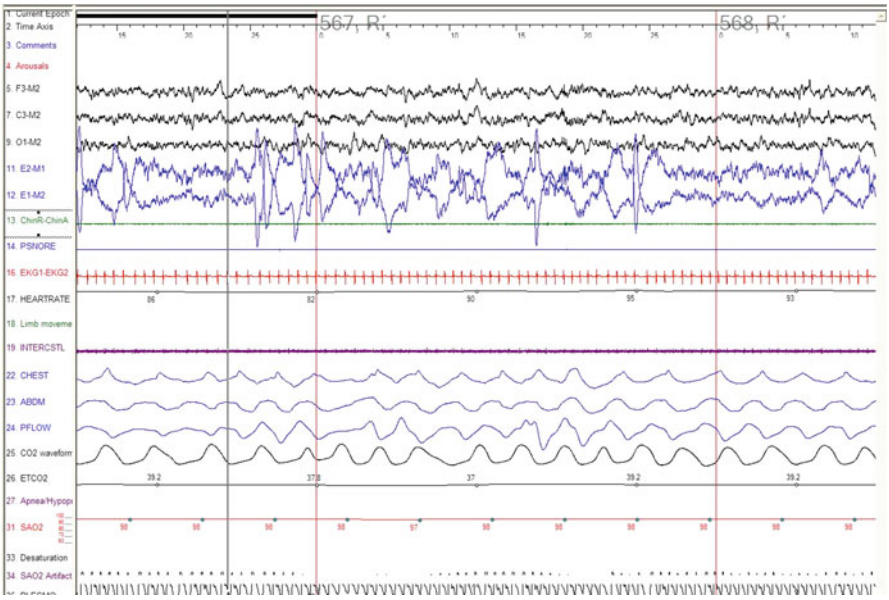

Fig. 5.5 Stage R (courtesy of George Zureikat, MD)

Stages (N3) and (N4): These two NREM stages have their own unique brain wave forms, but they are usually viewed as one stage of sleep, being referred to as slow-wave sleep, deep sleep, or restorative sleep. Because of their unique EEG wave forms, they are also known as delta sleep. Together, they comprise about 13–23 % of the total sleep time. Stage (N4) reflects the highest threshold for awakening from sleep relative to the other NREM stages.

REM Sleep

REM sleep is also referred to as dream sleep. Although it comprises only about 20–25 % of the total sleep time, this state recurs several times throughout the overall cyclical activity of NREM and REM states during a sleep period. In normal sleep, each subsequent recurring REM period is longer than the prior REM period.

EEG activity is increased with a characteristic "sawtooth" wave form and can also appear similar to wakefulness relative to mixed frequency (Fig. 5.5). There is a concomitant increase in heart rate, respiration, blood pressure, and jerky eye movements. During this state of increased cerebral activity, there may be a near immobility or paralysis of the muscles in the limbs, which has been thought to be a preventive mechanism of the individual to not physically act out his or her dreams during sleep. REM sleep may also be contributory to memory consolidation.

Table 5.1 New sleep scoring table

Stage W (wakefulness)
Stage Nl (NREM1)
Stage N2 (NREM2)
Stage N3 (NREM3; this replaces NREM stages 3 and 4)
Stage R (REM)

REM sleep may be further regarded as two phases: tonic and phasic. However, a typical sleep study report will not make a distinction between these two phases.

Tonic REM is a unique phase by virtue of the following characteristics: hypotonia (partial loss of muscle tone) of the skeletal muscles that approaches near paralysis and desynchronized EEG activity with widespread neural activation or wake-like EEG activity.

Phasic REM is also unique because it occurs sporadically instead of continuously, and it reflects the following characteristics: bursts of REMs in all directions, transient swings in blood pressure and heart rate along with tongue movement and irregular respiration, myoclonus (muscular jerks), twitching of the chin, and limb movements.

Alternative Sleep Scoring/Staging

In 2007, a new method of scoring sleep studies led to a revision in sleep staging; the new scoring is explained in the new sleep scoring table (Table 5.1) comparing to the old score in parenthesis.

Cycles and Hours of Sleep

Sleep patterns and architecture change for individuals throughout life. From infancy to old age, these changes are dynamic and distinct, relative to sleep initiation and maintenance and the amount of time for each sleep stage.

During normal sleep, man typically cycles through the NREM and REM sleep stages four to six times per sleep period. In children, these stages are shorter and occur at about 50–60-min intervals. In the adult, regardless of the age, these stages occur at about 90-min intervals. In addition, children have different proportions of REM and NREM sleep as well as different numbers of hours of sleep. A newborn typically sleeps 16–18 h, and 50 % of this sleep time is REM sleep. Slow-wave sleep (NREM stages 3 and 4) is at its maximum in young children since this is when growth hormone is secreted.

With age, slow-wave sleep decreases, which appears to begin after adolescence. After the age of 70, slow-wave sleep is minimal or, in some cases, nonexistent. In addition, the elderly spend more time in bed and less time actively sleeping [8].

Also as people age, they often begin to adjust the time that they go to bed to an earlier and earlier hour. The result is that they have an increased potential to wake up earlier in the morning, which is a condition termed advanced sleep phase syndrome. In this syndrome, the individual purposely adjusts the sleep–wake schedule by attempting to initiate sleep in advance of the circadian rhythm. As such, the intentional adjustment is not synchronized with this internal biological rhythm.

With the aging process, the typical sleep architecture becomes more fragmented with increased awakenings or arousals during the sleep period and people tend to subsequently have an increased risk for sleep disorders, including SRBD and insomnia. With SRBD, the musculature that supports the airway becomes more relaxed during sleep, and this lends itself to increased collapsibility. Therefore, as one's age increases, the potential risk for SRBD is increased. The role of the dentist is significant for the recognition of OSA and for the management of this sleep disorder with oral appliance (OA) therapy.

Sleep–Wake Cycle

Circadian Rhythm

Humans tend to alternate between a period of wakefulness [9] lasting approximately 16 h and a continuous block of 8 h of sleep. Most mammals sleep around a 24-h cycle that is driven by clock genes that control the circadian rhythm [10] (known as process C). Light helps humans synchronize their rhythm with the cycles of the sun and moon by sending a retinal signal (melanopsin) to the hypothalamic suprachiasmatic nucleus. The suprachiasmatic nucleus that acts as a pacemaker to control the circadian timing function [11].

The investigation of process C uses biologic markers to assess a given individual's rhythm. A slight drop (hundredths of a degree centigrade) in body temperature and a rise in salivary and blood melatonin and growth hormone release peaking around midnight in the 24-h cycle are key indications of the acrophase (high peak) of process C. Interestingly, corticotropins (adrenocorticotropic hormone and cortisol) reach a nadir (lowest level) during the first hour of sleep. They then reach an acrophase in the second half of the sleep cycle process C that can also be studied using temperature recordings in relation to hormone release and polygraphy to measure brain, muscle, and heart activities.

Ultradian Rhythm

Under the 24-h process C of sleep and wakefulness, sleep onset and maintenance are governed by an ultradian cycle of three to five periods in which the brain, muscles, and autonomic cardiac and respiratory activities fluctuate. These cycles consist

of REM, sleep (active stage), and NREM sleep (light and deep stages). The REM stage is known as paradoxical sleep in Europe.

In humans, a clear decline in electrical brain and muscle activities as well as heart rhythm is observed from wakefulness to sleep onset. This decline is associated with a synchronization of brain waves toward stage I of sleep. Stage I is a transitional period between wakefulness and sleep. Stage 2 sleep then begins, accounting for about 50–60 % of total sleep duration. Stage 2 sleep is characterized by two EEG signals, K-complexes (brief, high-amplitude brain waves) and spindles (rapid, springlike EEG waves), both of which are described as sleep-promoting and sleep-preserving factors. Sleep stages I and 2 are categorized as light sleep.

Next, sleep enters a quiet period known as deep sleep or stages 3 and 4. These stages are characterized by slow, high-amplitude brain wave activities. Stages 3 and 4 are usually scored together and are characterized by a dominance of slow-wave activity (delta sleep = 0.5–4.5 Hz). This sleep period is associated with the so-called sleep recovery process.

Finally, sleep enters an ascension period and rapidly turns into either light sleep or REM sleep. REM sleep is associated with a reduction in the tone of postural muscles (which is poorly described as "atonia" in literature but is in fact hypotonia because muscle tone is never zero) and a rise in heart rate and brain activity to levels that frequently surpass the rates observed during wakefulness. Humans can dream in all stages of sleep, but REM dreams may involve intensely vivid imagery with fantastic and creative content. During REM sleep, the body is typically in a paralyzed-like state (muscle hypotonia). Otherwise, dreams with intense emotional content and motor activity might cause body movements that could injure individuals and their sleep partners.

An understanding of the presence of ultradian sleep cycles is relevant because certain pathologic events occur during sleep, including the following sleep disorders:

Most periodic body movements (leg or arm) and jaw movements, such as sleep bruxism, are observed in stage 2 sleep and with less frequency in REM sleep.

Sleep-related breathing events, such as apnea and hypopnea (cessation or reduction of breathing), are observed in stage 2 and REM sleep. Acted dreams with risk of body injury, diagnosed as the sleep movement disorder and REM behavior disorder, occur during REM sleep.

Sleep Disorders and Their Assessment

The second edition of the International Classification of Sleep Disorders (ICSD) lists 85 sleep disorders in eight major categories:

- Insomnias
- Sleep-related breathing disorders
- Hypersomnia

- Circadian rhythm disorders
- Parasomnias
- Sleep-related movement disorders
- Isolated symptoms as a category
- Other sleep disorders

Insomnias

Insomnia is defined by a difficulty in sleep initiation, duration, consolidation, or quality that occurs despite adequate time and opportunity for sleep and results in some form of daytime impairment.

Insomnia is the most commonly reported sleep disorder. It is subjectively reported by the patient and can typically include difficulty getting to sleep, awakening from sleep earlier than the desired wake-up time, and then getting back to sleep as well as the feeling of a poor quality of sleep. These subjective complaints are reported even though the individual has had ample time for sleep, and the end result is typically excessive daytime sleepness (EDS) or fatigue along with its subsequent adverse impact on daytime function and quality of wakefulness[7]. These subjective complaints may be generally confirmed through the objective findings of a polysomnography (PSG) sleep study, but PSG is not the standard of care for the definitive diagnosis of insomnia.

Although the etiology of insomnia is not completely understood, it appears to involve biological, psychological, and social elements, and it can be regarded as a condition of hyperarousal.

Sleep-Related Breathing Disorders

Abnormal breathing during sleep is increasingly recognized as a clinical problem. Patients with lung disease, such as asthma and chronic obstructive pulmonary disease (COPD), may develop periods of even worse oxygen desaturations of the body's tissues during sleep. However, decreased frequency and depth of breathing during sleep-related breathing disorder are mostly applied to sleep apnea and hypopnea syndrome.

Snoring

Snoring is an acoustic phenomenon. Usually it happens on inhalation and is caused by fluttering and vibration of the soft tissues of the upper airway; the snoring described here occurs without episodes of apnea or hypoventilation.

Snoring is very common with an occurrence in 24 % of adult females and 40 % of adult males. Because snoring is usually more of a complaint for the bed partner than it is for the snorer, the noise is typically the primary reason for seeking a medical

consultation. Not all individuals who snore have OSA, but those individuals with OSA generally demonstrate a snoring component during their sleep period. Thus, it is possible for snoring to be a precursor to OSA.

When snoring occurs without any sleep pattern fragmentation or respiratory apneic or hypopneic episodes, it is often referred to as primary (benign) snoring. Whereas individuals with OSA will report the classic descriptions of EDS or insomnia, individuals with primary or benign snoring will not have these experiences. However, snoring may also be associated with many of the same symptoms as OSA.

Unfortunately, the individual who snores may not be aware of the association of OSA symptoms and snoring, and thus some snorers may also experience EDS along with associated but underlying subclinical health issues that have not been brought to the attention of their primary care physician. The patient has no complaints of insomnia, EDS, or sleep disruption that are attributable to snoring or airflow limitation.

Obstructive Sleep Apnea Syndrome

Apnea is a Greek word that means "without breath." The sleep apnea syndrome is characterized by frequent cessations of breathing during sleep. Physiologically, apnea can be classified into three types: central, obstructive, and mixed [12].

When central sleep apnea (CSA) is diagnosed, it must be pure CSA or at least 80 % of mixed sleep apnea. When most of the events are obstructive or mixed, OSA [13] is diagnosed. The severity of the syndrome, in both OSA and CSA, is primarily determined by the rate of sleep-disordered breathing (SDB) events per hour of sleep (respiratory disturbance index or RDI; apnea–hypopnea index or AHI) and the magnitude of associated oxygen desaturations. As both hypopneas and complete apneas result in arousals from sleep, the distinction between them is not considered important from a severity point of view.

OSA is the most prevalent sleep disorder seen in diagnostic sleep laboratories worldwide, accounting for some 75–80 % of the diagnoses. CSA is considerably less prevalent, except for specific patient populations, such as patients with chronic heart failure or patients with neurological disorders.

Central Sleep Apnea Syndrome

CSA is the cessation of an airflow without any respiratory effort to move air into or out of the lungs. Although the etiology is unknown, there are investigations suggesting that this disorder is related to cardiac problems or central nervous system dysfunction associated with a ventilatory controller mechanism. A PSG in the sleep laboratory similar to the evaluation for OSA is necessary for diagnosis. However, different than the PSG demonstrating OSA, there is an absence of any respiratory effort throughout the duration of the apneic episode for CSA.

The CSA patient suffers from EDS, frequent arousals, and awakenings during sleep or insomnia complaints and/or awakening by shortness of breath.

Obstructive Sleep Apnea Syndromes

OSA is characterized by repetitive episodes of partial (hypopnea) or complete (apnea) upper airway obstruction occurring during sleep.

Sleep apnea literally involves the cessation of breathing on a repeated basis during sleep. This can occur for a brief period of time for a few seconds to longer than a minute, and the frequency can be as much as several hundreds of times during a sleep period.

OSA typically involves an airway obstruction that results in an increased respiratory effort and insufficient ventilation. OSA can involve complete blockage of the upper airway resulting in an apneic episode or partial blockage of the airway resulting in a hypopneic episode. Whereas apnea is complete cessation of airflow, hypopnea is characterized by a 70 % reduction of airflow for >10 s or any reduction in airflow that is associated with either an arousal from sleep or a >3 % arterial oxygen desaturation. Apneas and hypopneas as a result of these varying degrees and locations of upper airway obstructions are regarded as the most common SRBD.

Hypersomnias

Daytime sleepiness, or hypersomnia, is the inability to stay awake and alert during the major waking episodes of the day, resulting in unintended lapses into drowsiness or sleep.

Hypersomnia is a group of disorders in which the primary complaint is daytime sleepiness and in which the cause of the primary symptom is not disturbed nocturnal sleep or misaligned circadian rhythms.

EDS is sleepiness that interferes with activities and quality of life during the waking hours. Typically the individual is unable to remain alert and awake during the hours that are normally regarded as the waking hours for that individual. EDS may be an indication that the individual is suffering from an inadequate amount of sleep, a fragmented or disrupted sleep.

Narcolepsy with Cataplexy

Narcolepsy with cataplexy is primarily characterized by excessive daytime sleepiness. Many of its symptoms are due to an unusual proclivity to transition rapidly from wakefulness into REM sleep and to experience dissociated REM sleep events. A definite history of cataplexy, defined as sudden and transient episodes of loss of muscle tone triggered by emotions, is present.

Narcolepsy Without Cataplexy

Excessive daytime sleepiness in narcolepsy without cataplexy is most typically associated with naps that are refreshing in nature, while nocturnal sleep is normal or moderately disturbed without excessive amounts of sleep.

The two most common types of narcolepsy are that with cataplexy and that without cataplexy. Some individuals who initially do not exhibit cataplexy will subsequently develop such episodes with their narcolepsy.

The multiple sleep latency test (MSLT) is used to assess daytime sleepiness and diagnose narcolepsy.

Circadian Rhythm Sleep Disorders

For optimal sleep, the desired sleep time should match the timing of the circadian rhythm of sleep and wake propensity. Therefore, a recurrent or a chronic pattern of sleep disturbance may result from alterations of the circadian timing system or a misalignment between the timing of the individual's circadian rhythm of sleep propensity and the 24-h social and physical environments.

Circadian rhythm sleep disorders (CRSD) [14] should be included as a possibility when considering a differential diagnosis of individuals who report EDS, insomnia, and impairment of daily functional activities.

Circadian in Latin means "about a day." The human body has an internal timing that demonstrates a circadian rhythm, and one of the more powerful external stimuli for indicating time is the light–dark cycle. Another time indicator for the body is melatonin [15], which is low during the day since light suppresses the secretion of melatonin and increases as the body prepares for the onset of sleep.

The common chronophysiologic characteristic of CRSD is the recurrent asynchrony between the individual's pattern of sleep and what is regarded as society's norm for sleep. With most CRSD cases, the individual has difficulty sleeping at the desired sleep time or when it is required. When there is the desynchronization of the individual's circadian clock relative to the light–dark cycle, CRSD can occur.

Parasomnias

Parasomnias are undesirable physical events or experiences that occur during all sleep stages from entry into sleep to arousals from sleep.

Parasomnias are undesirable and unintended physical and/or subjective experiences that occur as the individual begins to enter into sleep, during sleep, or during arousals from sleep. Included in these disorders are sleep-related movements, emotions, behaviors, dreaming, and functioning of the autonomic nervous system. Parasomnias often take place during arousal and transitions between sleep states when there is reorganization of brain activity, which lends to the belief that the sleep

and waking states are not mutually exclusive. The result, therefore, of the overlap of one state with the other is these episodes.

Of the parasomnias, those considered to be disorders of arousal are the most common and can manifest in 4 % of the adult population. Examples of such arousals can include sleepwalking, mumbling, shrieking, disorientation upon awakening, limb paralysis, and uncontrollable eating.

Sleep Movement Disorders

Restless Leg Syndrome

Restless legs syndrome (RLS) [16] is a sensorimotor disorder characterized by a complaint of a strong, nearly irresistible, urge to move the legs. This urge to move is often but not always accompanied by other uncomfortable paresthesias felt deep inside the legs or is a feeling that is simply difficult or impossible to describe.

Episodes of RLS are present mainly when the individual is at rest or during periods of inactivity, and they occur later in the day/evening or as the individual is attempting to initiate sleep. Generally, the experience can have a duration of a few minutes to several hours.

Individuals often describe associated paresthesias or uncomfortable sensations such as jittery or itchy feelings being associated with RLS [17]. The urge to move the legs and the paresthesias can be so unpleasant as to preclude the individual from initiating sleep. It has also been reported that individuals can awaken from sleep because of the RLS episode. Often the individual will relieve these sensations by getting up and walking. It is not uncommon to associate RLS in individuals who demonstrate reduced iron levels along with renal failure.

Periodic Limb Movement Disorder

Periodic limb movement disorder (PLMD) is characterized by periodic episodes of repetitive, highly stereotyped, limb movements that occur during sleep (PLMS) and by clinical sleep disturbance that cannot be accounted for by another primary sleep disorder.

PLMD is characterized by repetitive limb movements that occur during sleep. PLMD can be associated with RLS [17], although PLMD can stand alone as an episode independent of RLS. These periodic episodes of limb movement can result in sleep disturbances[7], although the individual is usually unaware of such partial arousal or awakenings. Even though the disorder is unrecognized by the individual, it is not uncommon for the individual to report a history of EDS and/or insomnia.

PLMD usually displays as extensions of the big toe or flexions of the ankle, knee, or hip, but it can also involve the upper limbs.

Note: The PLMS index must be interpreted in the context of a patient's sleep-related complaint. In adults, normative values higher than the previously accepted

value of 5 per hour have been found in studies that did not exclude respiratory event-related arousals (using sensitive respiratory monitoring) and other causes for PLMS. New data suggest a partial overlap of PLMS index values between symptomatic and asymptomatic individuals, emphasizing the importance of clinical context over an absolute cutoff value.

Sleep-Related Leg Cramps

Sleep-related leg cramps are painful sensations caused by sudden and intense involuntary contractions of muscles or muscle groups, usually in the calf of small muscles of the foot, occurring during sleep.

Sleep-related leg cramps are characterized by sudden intense muscle contractions that occur during sleep. Typically, the muscles of the calves or the feet are affected. This disorder has also been known as "charley horse."

These cramps usually occur during sleep, which then result in a disruption of sleep such as an arousal or even an awakening with severe pain. Because of these disruptions to sleep, the individual may report EDS and/or insomnia.

Sleep-Related Bruxism

Sleep bruxism is an oral activity characterized by a repetitive activity (repeated at least three times per episode) in the jaw muscles that generates tooth-grinding sounds and occasionally jaw clenching. Grinding or clenching of the teeth during sleep is usually associated with sleep arousals.

Sleep-related bruxism (SRB) is an oromotor activity characterized by clenching and/or grinding of the teeth during sleep, and it is regarded as a separate entity than bruxism that occurs during the waking hours.

The etiology and pathophysiology of the disorder are unknown. However anxiety and stressful life situations have both been suggested to be risk factors, but more studies are needed in general population to confirm this association. Most SRB events tend to occur in clusters in relation to recurrent arousals (7–14 times per hour of sleep) with transient (3.0 to 10.0 s) reactivation of muscle tone, brain, and heart activities during sleep. According to the reports of children's parents, awareness of tooth-grinding sounds in infants stands at 14–18 %. Findings based on the reports of sleep partners show that 8 % of adults make tooth-grinding sounds, a level that drops to 3 % in older individuals, although this estimate is less precise because of the presence of dentures and habits of sleeping alone.

In dentistry, bruxism is regarded as a mandibular parafunctional activity, whereas in sleep medicine, SRB is considered to be a movement disorder. The patient reports or is aware of tooth-grinding sounds or tooth clenching during sleep. One or more of the following is present: abnormal wear of the teeth, headaches on awakening, jaw muscle discomfort, fatigue, limited mouth opening in the morning, meniscus displacement, jaw pain upon awakening, and masseter muscle hypertrophy.

A dentist's decision to request a sleep laboratory examination may be based on frequent tooth grinding as reported by parents or sleep partners, tooth damage, and orofacial pain (OFP) [18] or headache in relation to sleep [19]. The diagnosis is confirmed by polygraphic recordings of masseter muscle activity and audio–video recordings. Patients with mild sleep bruxism will exhibit more than two jaw muscle contractions per hour of sleep, and patients with moderate-to-severe SRB will exhibit more than four such events per hour of sleep. The differential diagnosis of SRB must exclude the tooth tapping activity and sounds associated with faciomandibular myoclonus. This disorder causes rapid jaw muscle contractions (of less than 0.25-s duration) and is found in 10 % of tooth grinding events. Faciomandibular myoclonus is dominant in REM sleep, and, because it may be associated with sleep-related epilepsy or RBD, a full electroencephalographic examination is recommended.

Children may exhibit various tics during sleep, including throat grunting, enuresis, and sleep talking, and these also have to be excluded in the diagnostic process. SDB such as sleep apnea in children or in older individuals also must be verified in the sleep laboratory. The persistence of wakeful dyskinetic movement (dystonia, tremor, chorea, and dyskinesia) is also possible, but it is rarely concomitant with sleep bruxism.

Sleep Disorders Associated with Other Medical Conditions

Numerous medical conditions affect sleep or are affected by sleep. The list is a small number of medical disorders that may be of particular importance to sleep specialists.

Pain and Sleep

Insomnia, lack of sleep, and other sleep disorders intensify the pain conditions. Also pain can cause poor sleep, loss of sleep, and a reduction in an adequate number of hours of sleep that only continues to perpetuate the vicious pain cycle. Therefore, improving sleep can cause pain relief or reduction.

Studies have demonstrated that chronic pain can be present in 11–29 % of the adult population and that 50–90 % of these individuals can indicate that their sleep is adversely affected by their pain [20].

Sleep loss, specifically 4-h and REM-type sleep, is associated with hyperalgesia the following day. There is a bidirectional relationship between the loss of sleep and pain; that is, the loss of sleep impacts pain levels, and pain levels can reduce the amount of sleep [21].

In patients with osteoarthritis, improvement of sleep latency and sleep efficiency was analgesic when compared to control subjects.

Dentists encounter a number of painful conditions, and it is imperative that the loss or the lack of sleep be considered in the overall management plan for the

painful condition. In addition, it is essential that an understanding of the relationship between pain and its relationship to the sleep state be considered when planning for the management of each situation and condition.

Orofacial Pain

Dentists deal with dental and oral pain, so they have more interest in the head and neck pain, including headaches. As with pain in general, these conditions are frequently associated with some type of sleep disruption. It is difficult to determine which came first, the pain or the sleep problem.

Temporomandibular Disorders

The occurrence of temporomandibular disorders (TMDs) in conjunction with poor sleep may be associated with MFP and/or bruxism that occurs during sleep and/or waking hours.

Myofacial Pain

Muscle pain is a common finding among patients with poor sleep. The patients with muscle pain are most likely to have insomnia. However, not all patients with poor sleep will have muscle pain or muscle tenderness when palpated. Sleep disturbance is a common finding in myofacial pain (MFP) patients.

Patients with lack of sleep and sleep breathing disorders may suffer from fibromyalgia (FM). One study demonstrated that greater than 50 % of subjects diagnosed with FM also experienced chronic fatigue. The FM patient will have the presence of multiple tender points that have been anatomically mapped, and the presence of these is a factor in determining the risk for this condition. MFP and FM are related, and their coexistence as well as the relationship to sleep disorders are important to recognize.

Trigeminal and Glossopharyngeal Neuralgia

This particular facial neuralgia is paroxysmal in nature and is precipitated by function or touch. The attacks are often unilateral, and they are described as sharp or electric-like and usually brief and unpredictable. The interesting fact related to this pain and sleep relationship is that the attacks do not occur during sleep.

Temporal Arteritis (TA)

Also known as giant cell arteritis, this is a painful headache-like condition with throbbing around the area of the temporal artery. The patient may have pain in the masticatory [22] muscles associated with chewing. The pain will frequently be worse at night and may be exaggerated by resting the head on a pillow. Because of the serious nature of this type of pain, immediate attention to the treatment should be initiated.

Toothache

Anecdotally, toothache is one of the OFP conditions that can interfere significantly with sleep. Patients with acute pulpitis or apical periodontitis often report awakenings and lack of sleep due to pain. Epidemiologic studies have, indeed, substantiated the influence of toothaches on sleep. Periodontal pain after adjustment of orthodontic archwires is reported to have little influence on sleep.

Idiopathic Atypical Odontalgia

This type of OFP is what appears to the patient as being an odontogenic pain, but it is without any distinct or obvious dental pathology. The pain is typically more prevalent in the maxillary posterior teeth, and it does not resolve with the use of local anesthesia. Because the pain is continuous and a cause is frequently elusive, the patient may become depressed or have increased stress, which in turn may lead to insomnia.

These patients will usually report that they will awaken with this pain, but they do not awaken because of the pain. The treatment of this condition is generally responsive to a low-dose regimen of tricyclic antidepressant medication.

Headache Disorders

Headache and sleep disorders are the most prevalent conditions seen in clinical practice [23]. As with other painful conditions, headaches can be related to sleep disturbances. Also similar to other pain, the headaches frequently will not resolve unless the sleep disorder is also addressed. As such, sleep may both provoke as well as relieve headaches.

In particular, chronic daily and morning headaches are indicators of a probable sleep disorder. These encompass SRBD, insomnia, circadian rhythm disorders, and

parasomnias. The most frequently reported headaches that are related to a sleep disorder are migraine, cluster, and muscle tension type.

Headache seems to be more common in snorers as compared to non-snorers. Habitual snoring is also more prevalent in chronic daily headache patients than in those with episodic headache. Insomnia can lead to headache, and the severity of headache is related directly to the degree of insomnia. Because insomnia is the most common sleep complaint relative to sleep disorders, it is found to occur in one-half to two-thirds of headache patients.

In cluster headache, the presence of OSA is 8.4 times that of the normal population. When the patient is over 40 years of age and has an increased body mass index (BMI), the odds ratio increases. Accordingly, the risk decreases with a lower BMI and when the patient is less than 40 years old.

In the ICSD-2, the most common headaches that are sleep related are the following: cluster, migraine, tension type, and paroxysmal hemicrania. Of the patients who had headache, 53 % were diagnosed with OSA.

When a patient reports temporal or tension-type headaches on awakening, the dentist must assess for SDB or sleep bruxism [24] because these are frequently related complaints. Dentists should gather the patient's and the sleep partner's reports of snoring, cessation of breathing, and sleepiness by using the Epworth Sleepiness Scale (ESS) questionnaire.

Sleep-related headaches are a group of unilateral or bilateral headaches of varying severity and duration that occur during sleep or upon awakening from sleep. Sleep medicine recognizes the association that may exist for some individuals relative to sleep disorders and various headache disorders, including migraine, cluster, chronic daily, awakening or morning, and tension-type headaches. As compared to the general population, individuals with headaches demonstrate a two- to eightfold greater risk for sleep disorders, and the most common sleep disorder associated with headache subjects is insomnia.

It has been suggested that the neuroanatomy of the hypothalamus and the neurophysiological mechanisms involving the secretions of serotonin and melatonin may be contributory to the comorbidity of sleep disorders and headaches. Relative to cluster headache, melatonin secretion may be impaired in those individuals.

Modifications of sleep hygiene such as sleep loss, sleep disturbance, and even oversleeping have been identified as the most common precipitating factors of migraine and tension-type headaches. Studies demonstrated a significant increase in SRBD with cluster headache subjects.

Migraine attacks can also be reported during the sleep period because about half of such attacks are reported to occur between 4 and 9 AM. Migraine attacks mainly occur in relation to REM sleep, although they sometimes occur during deep sleep (stages (N3) and (N4)). Patients may also report the occurrence of cluster headaches during REM sleep; such headaches are unilaterally periocular or temporal in nature and accompanied by autonomic reaction. A rare form of sleep-related headache is the hypnic headache, which occurs at sleep onset. The hypnic headache is mainly found in older patients and tends to be bilateral.

The review suggests that (1) chronic daily headache, and especially "morning headache," is a particular, though nonspecific, indicator for sleep disorders; (2) the identification and management of a primary sleep disorder in the presence of headache may improve or resolve the headache (headache secondary to primary sleep disorder); (3) headache patients exhibit a high incidence of sleep disturbance which might trigger or exacerbate headache; and (4) such primary headache may improve with regulation of sleep. These findings argue for screening and management of sleep disturbance among headache patients.

Fibromyalgia (FM)

According to the 1990 American College of Rheumatology consensus criteria, FM is characterized by widespread pain of at least 3-month duration and muscle tenderness, lack of sleep, headaches, anxiety, and mood alteration [25].

FM is a syndrome described by multiple tender point sites and long-standing musculoskeletal pain that is usually diffuse. The criteria for FM established by the American College of Rheumatology states that there must be a widespread distribution above and below the waist of musculoskeletal pain occurring for a minimum of 3 months along with 11 or more of the 18 recognized tender points. The prevalence of FM is the second most common rheumatological disorder after osteoarthritis. It is reported that more than 80–96 % of patients with FM may also suffer from poor sleep quality (also reported as a sensation of unrefreshing sleep) and TMDs or pain.

The sleep-related brain activity termed alpha–delta sleep is no longer considered a pathognomonic finding in these patients. Clinicians making a differential diagnosis in these patients must exclude periodic limb movement during sleep and SDB.

Many individuals with the FM syndrome also experience sleep disturbances that can result in feeling tired, unrefreshed sleep, reduced cognitive function, and early awakening from sleep. It has been found that most subjects with FM experienced microarousals and an electroencephalographic alpha–delta or alpha–NREM anomaly that interrupts the deep stage (N4) restorative level of sleep. This EEG anomaly, though, may not be specific to FM as it has also been found in individuals who did not have FM complaints.

Gastroesophageal Reflux

Sleep-related gastroesophageal reflux also known as heartburn is characterized by regurgitation of stomach contents into the esophagus during sleep. This sleep disorder may result in a pain that is usually located substernal, but it may also manifest in the area of the throat.

Subjective Self-Assessment

Although this type of self-assessment methodology does not provide objective physiologic data that measure wake/sleep periods of the patient, it does allow for a lower cost method to easily acquire at least some patient-based information that can be correlated with the patient's history and clinical examination.

Epworth Sleepiness Scale

The most frequently used instrument is ESS, which was developed in 1991. It has been demonstrated to identify degrees of sleepiness, and the results are considered to be within acceptable limits for test–retest reliability. The eight questions of the ESS query the individual about his or her subjective reporting of sleepiness relative to his or her expectation of dozing in eight different situations.

In using a scale of 0–3, where 0 indicates no chance of dozing, 1 indicates a slight chance, 2 indicates a moderate chance, and 3 indicates a high chance, a total maximum score of 24 is possible. Investigations have shown that a score of 10 or 11 is considered to be the upper parameter for normal. While higher scores correlate with sleep disorders, it has also been shown that scores also improve relative to the efficacy of management of SRBD.

Although the ESS is attractive via its simplicity and ease of administration, the instrument does have its limitations, including not taking into account the individual's age, acuteness of sleep pathology, medical conditions, or use of pharmaceutics. Thus, the ESS is best when it is employed to be an adjunct to the patient history and clinical examination.

Clinical Assessment

As defined by the American Academy of Sleep Medicine (AASM), there are four levels (I–IV) of sleep studies from which an objective-based assessment is made. A level I study is regarded as the most accepted study for the assessment and treatment of OSA. The four levels are differentiated as per the number of simultaneously recorded physiological signals as well as whether or not the sleep study was attended by a sleep technologist.

Polysomnography

A polysomnography (PSG) is an overnight sleep study attended by a sleep technologist during which at least seven different physiological signals are measured, and it is a level I study. The PSG is considered to be the "gold standard" in sleep medicine

relative to objective-based sleep studies. The study is usually conducted in a sleep laboratory/center type of facility with trained staffs to ensure proper placement of the necessary sensors as well as recognize and address any displacement of sensors as needed during the study.

The physiologic parameters measured during a PSG include simultaneous and continuous monitoring of at least brain wave activity, eye movements, muscle activity of the legs and mandible, body position, heart rate and rhythm, blood pressure, snoring, and respiratory activity that includes breathing patterns and oxygen saturation. Analysis of the data from these various measurements can reveal sleep disorder activities such as apnea. A summary of the entire PSG data can be reflected in a graphic form called a hypnogram which provides a comprehensive glimpse of sleep architecture relative to stages of sleep.

The raw data are scored into different sleep stages and physiologic activities in accordance with standardized criteria, and a sleep physician interprets the study results, reviews the patient history and clinical examination data, and subsequently prepares a sleep study report of outcomes and recommendations. Most common sleep disorders can be assessed with a PSG, including SRBD. Effectiveness of treatment methods can also be achieved with a PSG.

PSG performed in a sleep laboratory involves continuous overnight recording of a minimum of 12 channels of sleep- and breathing-related measurements, such as EEG, electrooculogram, electromyogram, nasal airflow (preferably measured by nasal pressure cannula), oral airflow (thermistor), respiratory effort, oxygen saturation, body position, and electrocardiogram. Recordings require manual scoring of the events by trained sleep technologists and interpretation of the results by sleep medicine physicians, taking into account the clinical context. The examinations monitor the occurrence of apneas (complete cessation of airflow for 10 s or more) and hypopneas (reduction in amplitude of airflow or thoracoabdominal wall movement for 10 s or more with an accompanying oxygen desaturation of at least 3 % and/or associated arousals). OSA is defined as a total of more than five events per hour of sleep. Notably, variations exist in scoring definitions, especially for hypopneas.

The severity of sleep apnea is assessed with the AHI, although other factors such as the degree of oxygen desaturation and the extent of sleep fragmentation are important for the clinical interpretation of OSA severity. Some laboratories report an RDI, which often incorporates all respiratory events (beyond apneas and hypopneas), although the definition for this score may vary.

Generally, diagnosis of OSA can be based on a single night of testing, although night-to-night variability in results should be considered, especially if test results are negative for a patient with high clinical risk of OSA. Apparent variability in the severity of OSA may result from a number of factors, including differences in sleeping position, alcohol use, prior sleep debt, sleep efficiency, and sleep stage distribution. Furthermore, variation in the definitions and scoring of the respiratory events can also significantly alter the AHI. The major limitations of PSG are that it is expensive and labor intensive, and thus waiting lists for the procedure tend to be very long.

Multiple Sleep Latency Test

An MSLT is commonly used to measure daytime sleepiness. The MSLT consists of a series of four to five 20-min daytime naps during 2-h intervals at a sleep laboratory/center, and the test often lasts 7–8 h. In addition, the test is to begin 1.5–3 h after awakening from an overnight PSG. Similar to the PSG, sensors are placed by a trained sleep technologist in order to measure brain wave activity, eye movements, muscle activity of the mandible, and cardiac activity. There are standardized conditions under which the MSLT is performed, including the instruction to the patient, such as "Please lie quietly, assume a comfortable position, keep your eyes closed, and try to fall asleep."

The physiological data outcomes demonstrate the amount of time it takes for the individual to initiate sleep (i.e., sleep latency) and to attain the different stages of sleep during each nap. The final scoring for the MSLT is the averaged times of sleep latency for the series of naps. An MSLT score of greater than 10 min is considered normal and less than 5 min is regarded as generally indicating the presence of a sleep disorder. Individuals who demonstrate a quick onset of REM sleep are also more likely to have a sleep disorder. Often the MSLT is the instrument of choice to definitively diagnose narcolepsy [26] or idiopathic hypersomnia. The MSLT can also be used to document outcomes of treatment. Residual sleepiness can also be demonstrated for those individuals who do not report sleepiness after undergoing treatment.

In and of itself, the MSLT is not regarded as an accurate means to differentiate pathologic sleep disorders. Practice parameters for the clinical application of the MSLT have been published by the AASM.

Maintenance of Wakefulness Test

A maintenance of wakefulness test (MWT) is commonly used to measure the alertness of an individual during the waking hours. The MWT can also be employed to assess a sleep disorder patient's response to treatment. Whereas the MSLT determines whether or not an individual can initiate sleep sooner than what is regarded as a normal amount of time, the MWT determines whether or not an individual can stay awake for what is regarded as a normal amount of time.

Similar to the MSLT, sensors are placed by a trained sleep technologist in order to measure brain wave activity, eye movements, muscle activity of the mandible, and cardiac activity, and subsequently there is a series of four to five 20-min daytime naps during 2-h intervals at a sleep laboratory/center. Although there are also similar standardized conditions under which the MWT is performed, the significant procedural difference of the MWT from the MSLT is with the instruction to the patient, "Please sit still and remain awake for as long as possible." Look directly ahead of you, and do not look directly at the light.

The physiological data outcomes demonstrate the ability of an individual to stay awake. The final scoring for the MWT is the averaged times of wakefulness for

the series. An MWT score of 8 min or more is considered normal, whereas abnormal would be an average of initiating sleep in less than 8 min. If the individual does not fall asleep in 40 min, then the test is terminated.

Practice parameters for the clinical application of the MSLT have been published by the AASM.

With a heightened awareness in sleep disorders relative to public safety, the MWT is acknowledged by the Federal Aviation Administration as a method of assessing effectiveness of treatment for EDS, thereby affording the ability to the aviation medical examiners to reissue an airman medical certificate for commercial airline pilots.

As with the other assessment instruments, test outcomes must be viewed in light of the patient history and clinical examination. Also, it has been shown that there is poor correlation between the ESS, MSLT, and MWT.

Sleep Apnea Management/Treatment

Behavioral Management

Behavioral treatment involves the abstinence from alcohol and sedatives in the early evening. The intake of alcohol selectively reduces the muscle tone of the upper airway and increases the frequency of abnormal breathing during sleep. Alcohol also prolongs apnea by delaying arousal. Behavioral methods also include training patients to sleep in a lateral position if upper airway obstruction is present only during sleep in the supine position. Weight reduction in a patient without anatomical risk factors can often eliminate OSA.

Medical Treatment

Patients with OSA, snoring, or upper airway resistance syndrome (UARS) will benefit primarily from positive airway pressure (PAP) [27–30]. However, the patient's compliance is a major issue in this therapy, which frustrates the sleep specialists and patient's bed partner.

The PAP device produces a positive pressurized airflow that is delivered to the patient via a mask over the face.

There are three primary types of PAP modes: (1) continuous positive airway pressure (CPAP), (2) bilevel positive airway pressure (BiPAP) [31], and (3) autoadjusting positive airway pressure (APAP). A fourth mode receiving some attention is the expiratory pressure relief mode (flexible CPAP).

There is a significant amount of published literature about PAP therapy. The purpose of this chapter is to present an emphasis on PAP therapy as it relates to the adult population with SRBD, in particular OSA.

Medical treatment involves the use of CPAP [32]; it is a device that controls apnea by providing a stream of air, under slight pressure, through a tube into the nasal passage. This positive air pressure acts as a splint holding the tissues in the back of the throat open to prevent collapse. Use of this device requires a sleep study to determine the proper pressure to use.

This treatment is regarded by most as the first fine of treatment in patients with moderate to severe OSA. This generally means patients with more than 20 episodes of apnea or hypopnea per hour with associated oxygen desaturations. However, patient's compliance with CPAP is approximately 50 %. Many patients therefore go untreated. Side effects reported by patients include irritation related to the nasal mask, nasal congestion [33], occasional rhinorrhea, and feelings of claustrophobia.

Besides effectiveness for management of moderate to severe OSA cases, PAP therapy may be effective for the management of mild OSA. However, there is a greater propensity for those with mild OSA to demonstrate less adherence (compliance) to PAP therapy. It is important for even those individuals with mild OSA to know that there exists the increased risk for cardiovascular issues, such as HT.

The individuals with mild OSA or even primary (benign) snoring may not find appealing the alternative treatment options that include either surgical intervention of the upper airway tissues or use of an OA [34]. The remaining possible conservative options include weight loss, sleep positional changes, sleep hygiene modifications, or other alternative therapies.

As with other areas of medical care, there are Medicare guidelines for PAP therapy reimbursement, which can serve as references for the indications of PAP therapy. If there is an AHI of 5–14 episodes/h that represents mild OSA, then there must also be symptoms or signs of significant impairment. Impairments that qualify include EDS, HT, insomnia, mood disorders, and cardiovascular issues. These impairments are not required for individuals with an AHI of 15 or more episodes per hour that represent moderate to severe OSA.

The effectiveness of PAP therapy for each individual diagnosed with OSA is assessed during an attended PSG or sleep study. During the PSG visit, the type of PAP mask interface and the level of airflow pressure effective for managing the OSA are established. This determination may be done during a second sleep study, also known as a two-night study, or it may be performed during the latter portion of a single-sleep study, also known as a split-night study.

Dental Appliances

OA therapy has been used for the management of sleep apnea, snoring, and upper airway resistance since the early 1930s [35].

Dental appliances in the treatment of OSA are of three classes. One type attempts to push the soft palate with a distal extension from a palate plate. This neither has nor found wide acceptance due to gagging and the uncertainty of maintaining hypopharyngeal width during sleep. The second class of appliances is designed to act directly on the tongue by holding it forward by means of negative pressure from an

anterior suction bulb or proprioceptive reminder. The tongue-retaining device (TRD) is the most successful of these appliances, especially in the elimination of snoring. The third group of appliances repositions the mandible in a more protrusive position and has general acceptance as being the most effective device in the elimination of both snoring and OSA. Both these TRD and MAD are designed to reduce the upper airway obstruction.

Standards of practice guidelines relative to the use and effectiveness of OA therapy for OSA and snoring were first published in 1995 by the AASM case studies comprising the evidence on which those initial clinical guidelines were based. The AASM guidelines document noted that OA therapy can be considered as a first-line treatment option for the management of mild OSA and simple snoring and also as a second-line treatment option for moderate OSA after unsuccessful attempts with other treatment options. Following the publication of these guidelines, significant research-based findings pertaining to OA therapy have been published [36, 37].

In 2006, the AASM published two documents that further recognized OA therapy as a medical device option for the management of OSA and snoring: (1) an evidence-based review of literature regarding OA therapy in sleep medicine and (2) a practice parameter update. Scientific literature published since 1995 comprised the evidence on which the current practice parameters were based. The updated practice parameters indicate that OA therapy is now an option for patients with mild to moderate sleep apnea and who prefer this method of treatment as opposed to using CPAP. In addition, OA therapy may be utilized in patients with severe sleep apnea who (1) are unable to tolerate CPAP, (2) have failed surgery, or (3) are primary (benign) snorers (i.e., snoring without apnea).

OAs have also been reviewed by the US Food and Drug Administration (FDA), and OAs are regarded as class II medical devices. The FDA document states that special controls apply to these devices, and they are deemed to be medical devices appropriate for the treatment of OSA. As such, OAs marketed to the public for the treatment/management of OSA and snoring are required to have a 510 k or premarket notification clearance in order to be commercially available.

The mechanism by which these appliances work appears simple. Mandibular advancement splints prevent the tongue from collapsing against the posterior pharyngeal wall nocturnally. This is achieved by mechanical means in that the origin and insertion of genioglossus are at the hyoid bone and mandibular symphyseal region, respectively. Thus, by advancing the mandible, the tongue is held in a more anterior position at night. Elevation of the hyoid bone in an anterosuperior direction is therefore the desired radiographic modification. A second consideration given by Lowe et al. is that, in man, voluntary passive opening of the mandible produces definite enhancement of genioglossus EMG through activation of receptors located in the temporomandibular joint. Because the contraction of the genioglossus opens the airway, airway obstruction may be prevented.

Despite considerable variation in the design of these appliances, the desired effects are remarkably consistent. Snoring is reduced and often eliminated in almost all patients who use OAs. Theoretical complications of long-term use of mandibular advancement splints include temporomandibular joint dysfunction.

However, intermittent forward positions of the mandible have yet to be shown to produce irreversible TMJ dysfunction as a consequence. Limited follow-up data indicate that oral discomfort is a common but tolerable side effect and that dental and mandibular complications appear to be uncommon.

Surgical Treatment

Surgical Therapy for OSA

A surgical solution to OSA, if successful, eliminates any question of compliance.

The surgical approach to SDB continues to evolve. Many surgical approaches have emerged, including tracheostomy, uvulopalatopharyngoplasty (UPPP), laser-assisted UPPP (LAUP), septoplasty, orthognathic surgery, and radiofrequency volumetric tissue reduction (RFVTR) ("somnoplasty" or "coblation"). Most recently, palatal implants have been developed and marketed as treatments for sleep apnea. The effects of bariatric surgery on adult sleep apnea have also been investigated. However, outside of tracheostomy and jaw advancement, telegnathic surgery remains the most accepted successful surgical therapy for OSA at this time [38].

In an extensive search for relevant studies of randomized trials of UPPP treatment for sleep apnea, the Cochrane investigators found eight studies, totaling 412 participants. Surgical treatment of SDB has been so poorly evaluated in the literature that it cannot be recommended to patients as a first-line treatment option. A common problem is that the surgical literature continues to define "success" on the basis of reduction in RDI by 50 % and not on a more strict definition.

UPPP and LAUP

UPPP is the most commonly performed and best studied of the surgical procedures used to treat sleep apnea. In this procedure, redundant soft palate and pharyngeal tissues, uvula, and tonsils are removed. Potential complications include velopharyngeal insufficiency, stenosis, and dysphagia. Because of problems with UPPP, several variations on this theme have emerged, including the uvulopalatal flap and laser-assisted uvula palatoplasty (LAUP for the uninitiated). Unfortunately, there simply is no sufficient evidence that these procedures are beneficial for patients with sleep apnea.

Radiofrequency Volumetric Tissue Reduction

RFVTR applied to the palate ("somnoplasty") was originally investigated as a less painful treatment option for SDB than UPPP. This procedure has largely been abandoned as treatment for sleep apnea, though it is occasionally used primarily for snoring.

Oral and Maxillofacial Surgery (Telegnathic Surgery)

When applying the basic orthognathic surgical technique for the purpose of effecting airway expansion in patients with OSA, the correct terminology is "telegnathic surgery." This term differentiates this procedure from that performed for correction of dentofacial deformities, as the purpose of this surgery is instead focused on airway expansion.

In a recent review of oral and maxillofacial surgery, Prinsell et al. concluded, "MMA" (maxillomandibular advancement surgery) for OSA surgery is a highly successful and potentially definitive primary single-staged surgery that may result in a significant reduction in OSA syndrome-related health risks, as well as financial savings for the health care system." Li reaches the same conclusion: MMA surgery is the most effective procedure for OSA. In four studies including a total of 214 patients who had MMA the (surgically defined) success rate was 96–100 %. MMA results in enlargement of the entire upper airway.

Jaw advancement surgery is better tolerated and has a far more significant and beneficial effect than most airway soft tissue surgical procedures and may produce beneficial effects on nocturnal breathing. Excellent long-term (5-year) results have been noted in patients who do not gain weight postoperatively, and favorable effects on quality of life following telegnathic surgery have been documented.

Application of this procedure to the pediatric population using distraction osteogenesis techniques continues to advance, as new and modified distraction devices continue to evolve. Distraction osteogenesis offers the treating surgeon the ability to "titrate" the degree of advancement with the amount of airway expansion needed to resolve the airway problem. Rapid maxillary expansion has been shown to have very favorable effects in the growing child on improvement (and often elimination) of OSA.

Telegnathic surgery is appropriate when there are ill effects from the use of MRAs on the temporomandibular joints, occlusion, lack of compliance to an OA, failure of previous soft tissue surgical procedures, and other structural anomalies present, such as in the hypoplastic or constricted maxilla.

The surgical methods to eliminate OSA include reduction of the inferior turbinate bones, adenoidectomy (nasopharyngeal involvement), UPPP, tonsillectomy (oropharyngeal involvement), genioglossal and hyoid advancement (hypopharyngeal involvement), bimaxillary advancement, and tracheostomy. UPPP is curative in less than 50 % of patients. LAUP has recently been introduced as an outpatient treatment for snoring.

UPPP enlarges the oropharynx and reduces the collapsibility of the upper airway. Patients with a narrow oropharynx relative to tongue size have a good response to UPPP.

Surgical treatment of OSA by MMA should be restricted to patients with a retrognathic dolichofacial type combined with pharyngeal narrowing. The maxilla and mandible must both be advanced at least 10 mm to ensure success. In severe instances of OSA that are life threatening, the ultimate treatment is tracheostomy. This procedure completely bypasses the upper airway and thus all upper airway obstruction. The procedure is, however, usually used as the last option in the treatment of OSA.

Conclusion

Understanding sleep and the processes related to it is important because it helps in understanding the dynamics of sleep disorders; since the use of OA for management of OSA is recognized as an acceptable treatment option, the dentist now has an increasing role in the recognition of a patient who may be at risk for a sleep disorder. The dentist does not have to understand the complex neurologic and neurochemical relationships that present with sleep, but it is prudent to have an understanding of the basics, the knowledge of the relevant aspects of sleep, and the ability to recognize how normal sleep may become altered. This understanding allows the dentist to have the foundational knowledge to explain certain aspects of sleep to patients who wish to have a better appreciation of their sleep and why disruption of it is occurring.

In addition, understanding sleep and sleep disorders will enable the dentist to better communicate with physicians regarding a patient who may have sleep disorders. For the sake of the patient, the dentist should have an ever-increasing awareness of medical issues relative to their patients.

It is not necessary for the dentist to determine the specific sleep disorder. More importantly, it is helpful if the dentist can recognize symptoms that may indicate the need for a more appropriate referral.

Acknowledgement The author would like to thank Dr. George Zureikat MD for his recordings of sleep patterns to be used in this chapter.

Conflict of Interest None declared.

References

1. Khan F, Hazin R, Han Y. Apneic disorders associated with heart failure: pathophysiology and clinical management. South Med J. 2010;103(1):44–50.
2. Ross SD, Sheinhait IA, Harrison KJ, Kvasz M, Connelly JE, Shea SA, et al. Systematic review and meta-analysis of the literature regarding the diagnosis of sleep apnea. Sleep. 2000;23(4): 519–32.
3. Morin CM, Rodrigue S, Ivers H. Role of stress, arousal, and coping skills in primary insomnia. Psychosom Med. 2003;65(2):259–67.
4. Mitler MM, Gujavarty KS, Browman CP. Maintenance of wakefulness test: a polysomnographic technique for evaluation treatment efficacy in patients with excessive somnolence. Electroencephalogr Clin Neurophysiol. 1982;53(6):658–61.
5. Johns MW. Sensitivity and specificity of the multiple sleep latency test (MSLT), the maintenance of wakefulness test and the Epworth sleepiness scale: failure of the MSLT as a gold standard. J Sleep Res. 2000;9(1):5–11.
6. Sangal RB, Mitler MM, Sangal JM. Subjective sleepiness ratings (Epworth sleepiness scale) do not reflect the same parameter of sleepiness as objective sleepiness (maintenance of wakefulness test) in patients with narcolepsy. Clin Neurophysiol. 1999;110(12):2131–5.
7. Loureiro CC, Drummond M, Winck JC, Almeida J. Clinical and polysomnographic characteristics of patients with REM sleep disordered breathing. Rev Port Pneumol. 2009;15(5):847–57.

8. Roepke SK, Ancoli-Israel S. Sleep disorders in the elderly. Indian J Med Res. 2010; 131:302–10.
9. Wise MS. Objective measures of sleepiness and wakefulness: application to the real world? J Clin Neurophysiol. 2006;23(1):39–49.
10. Kanathur N, Harrington J, Lee-Chiong T. Circadian rhythm sleep disorders. Clin Chest Med. 2010;31(2):319–25.
11. Castriotta RJ, Atanasov S, Wilde MC, Masel BE, Lai JM, Kuna ST. Treatment of sleep disorders after traumatic brain injury. J Clin Sleep Med. 2009;5(2):137–44.
12. White DP. Sleep apnea. Proc Am Thorac Soc. 2006;3(1):124–8.
13. McNicholas WT, Ryan S. Obstructive sleep apnoea syndrome: translating science to clinical practice. Respirology. 2006;11(2):136–44.
14. Silva EJ, Wang W, Ronda JM, Wyatt JK, Duffy JF. Circadian and wake-dependent influences on subjective sleepiness, cognitive throughput, and reaction time performance in older and young adults. Sleep. 2010;33(4):481–90.
15. Cajochen C, Kräuchi K, Wirz-Justice A. Role of melatonin in the regulation of human circadian rhythms and sleep. J Neuroendocrinol. 2003;15(4):432–7.
16. Chahine LM, Chemali ZN. Restless legs syndrome: a review. CNS Spectr. 2006;11(7): 511–20.
17. Michaud M, Chabli A, Lavigne G, Montplaisir J. Arm restlessness in patients with restless legs syndrome. Mov Disord. 2000;15(2):289–93.
18. Wong MC, McMillan AS, Zheng J, Lam CL. The consequences of orofacial pain symptoms: a population-based study in Hong Kong. Community Dent Oral Epidemiol. 2008;36(5): 417–24.
19. Dodick DW, Eross EJ, Parish JM, Silber M. Clinical, anatomical, and physiologic relationship between sleep and headache. Headache. 2003;43(3):282–92.
20. Smith MT, Perlis ML, Smith MS, Giles DE, Carmody TP. Sleep quality and presleep arousal in chronic pain. J Behav Med. 2000;23(1):1–13.
21. Pilowsky I, Crettenden I, Townley M. Sleep disturbance in pain clinic patients. Pain. 1985; 23(1):27–33.
22. Bertoli E, de Leeuw R, Schmidt JE, Okeson JP, Carlson CR. Prevalence and impact of posttraumatic stress disorder symptoms in patients with masticatory muscle or temporomandibular joint pain: differences and similarities. J Orofac Pain. 2007;21(2):107–19.
23. Rains JC, Poceta JS. Headache and sleep disorders: review and clinical implications for headache management. Headache. 2006;46(9):1344–63.
24. Bader GG, Kampe T, Tagdae T, Karlsson S, Blomqvist M. Descriptive physiological data on a sleep bruxism population. Sleep. 1997;20(11):982–90.
25. Morin CM, Gibson D, Wade J. Self-reported sleep and mood disturbance in chronic pain patients. Clin J Pain. 1998;14(4):311–4.
26. Parkes JD, Chen SY, Clift SJ, Dahlitz MJ, Dunn G. The clinical diagnosis of the narcoleptic syndrome. J Sleep Res. 1998;7(1):41–52.
27. Loube DI, Gay PC, Strohl KP, Pack AI, White DP, Collop NA. Indications for positive airway pressure treatment of adult obstructive sleep apnea patients: a consensus statement. Chest. 1999;115(3):863–6.
28. Sanders MH, Montserrat JM, Farré R, Givelber RJ. Positive pressure therapy: a perspective on evidence-based outcomes and methods of application. Proc Am Thorac Soc. 2008;5(2): 161–72.
29. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. Proc Am Thorac Soc. 2008;5(2):173–8.
30. Barnes M, McEvoy RD, Banks S, Tarquinio N, Murray CG, Vowles N, et al. Efficacy of positive airway pressure and oral appliance in mild to moderate obstructive sleep apnea. Am J Respir Crit Care Med. 2004;170(6):656–64.
31. Kushida CA, Littner MR, Hirshkowitz M, Morgenthaler TI, Alessi CA, Bailey D, et al. Practice parameters for the use of continuous and bilevel positive airway pressure devices to treat adult patients with sleep-related breathing disorders. Sleep. 2006;29(3):375–80.

32. Redline S, Adams N, Strauss ME, Roebuck T, Winters M, Rosenberg C. Improvement of mild sleep-disordered breathing with CPAP compared with conservative therapy. Am J Respir Crit Care Med. 1998;157(3 Pt 1):858–65.
33. Sugiura T, Noda A, Nakata S, Yasuda Y, Soga T, Miyata S, et al. Influence of nasal resistance on initial acceptance of continuous positive airway pressure in treatment for obstructive sleep apnea syndrome. Respiration. 2007;74(1):56–60.
34. Ferguson KA, Cartwright R, Rogers R, Schmidt-Nowara W. Oral appliances for snoring and obstructive sleep apnea: a review. Sleep. 2006;29(2):244–62.
35. Hoekema A, Stegenga B, De Bont LG. Efficacy and co-morbidity of oral appliances in the treatment of obstructive sleep apnea-hypopnea: a systematic review. Crit Rev Oral Biol Med. 2004;15(3):137–55.
36. Kushida CA, Morgenthaler TI, Littner MR, Alessi CA, Bailey D, Coleman J, et al. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: an update for 2005. Sleep. 2006;29(2):240–3.
37. Lim J, Lasserson TJ, Fleetham J, Wright J. Oral appliances for obstructive sleep apnoea. Cochrane Database Syst Rev. 2006;1, CD004435.
38. Sundaram S, Bridgman SA, Lim J, Lasserson TJ. Surgery for obstructive sleep apnoea. Cochrane Database Syst Rev. 2005;4, CD001004.